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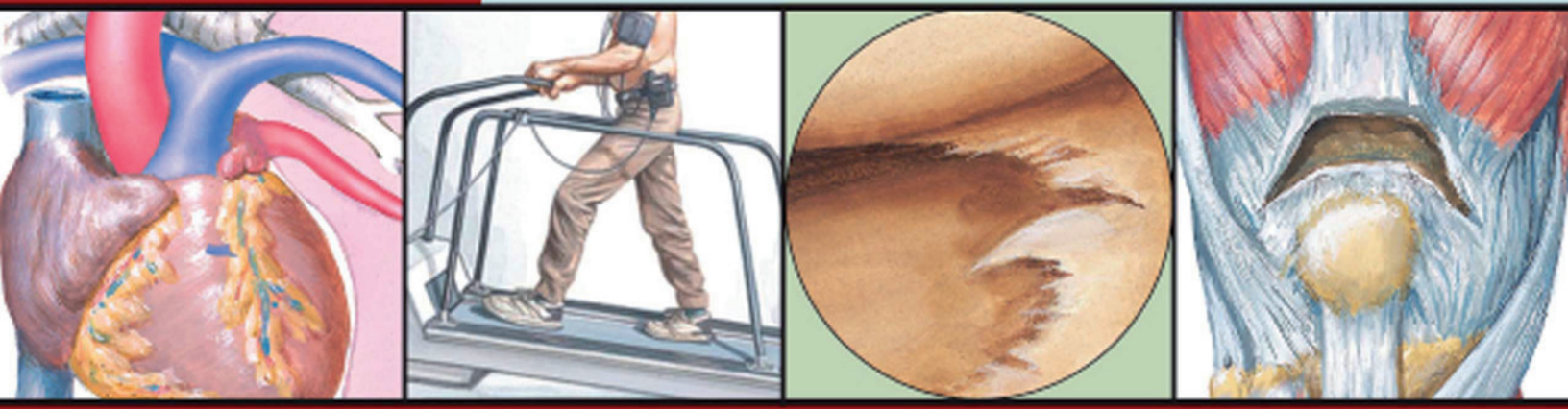

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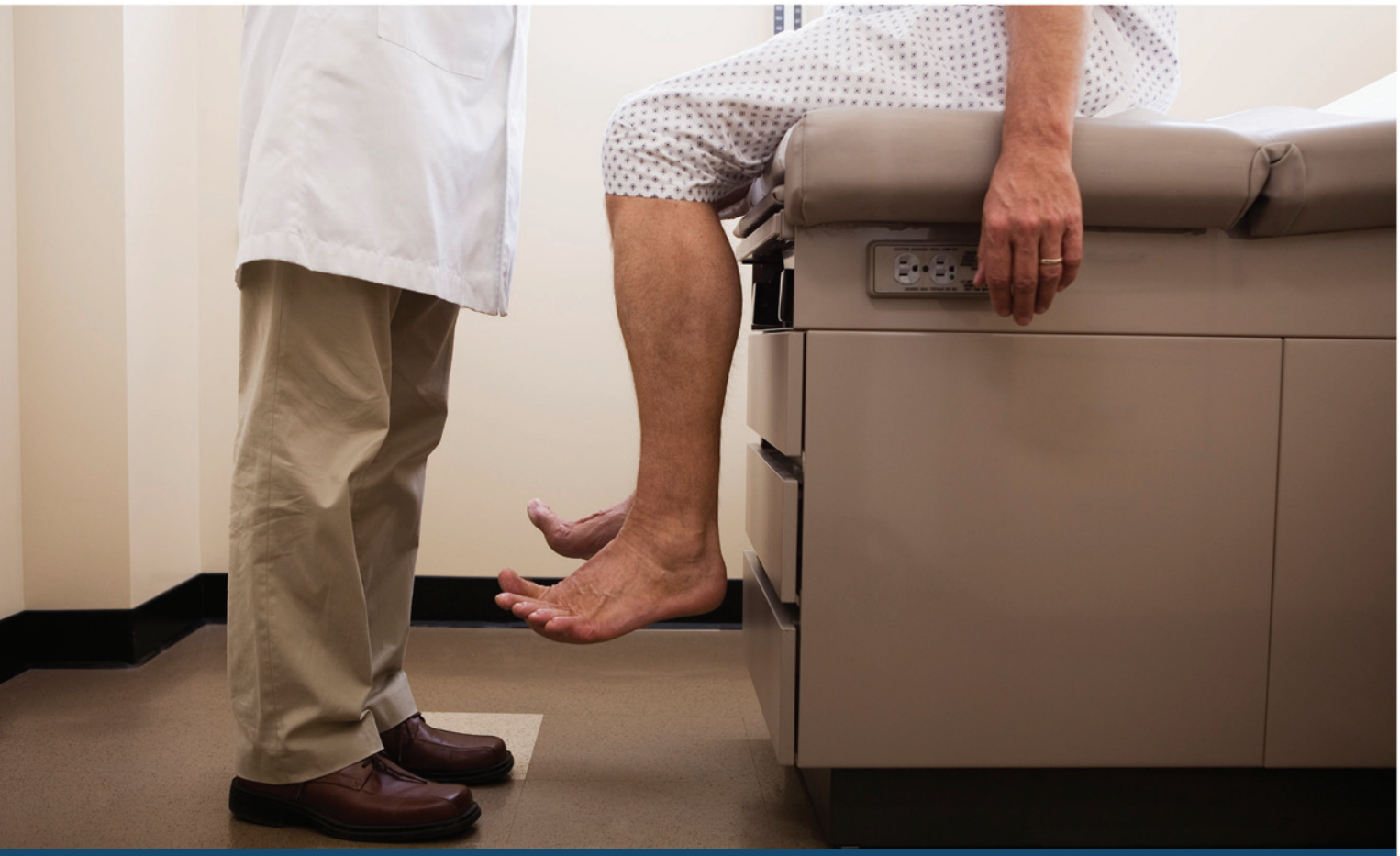

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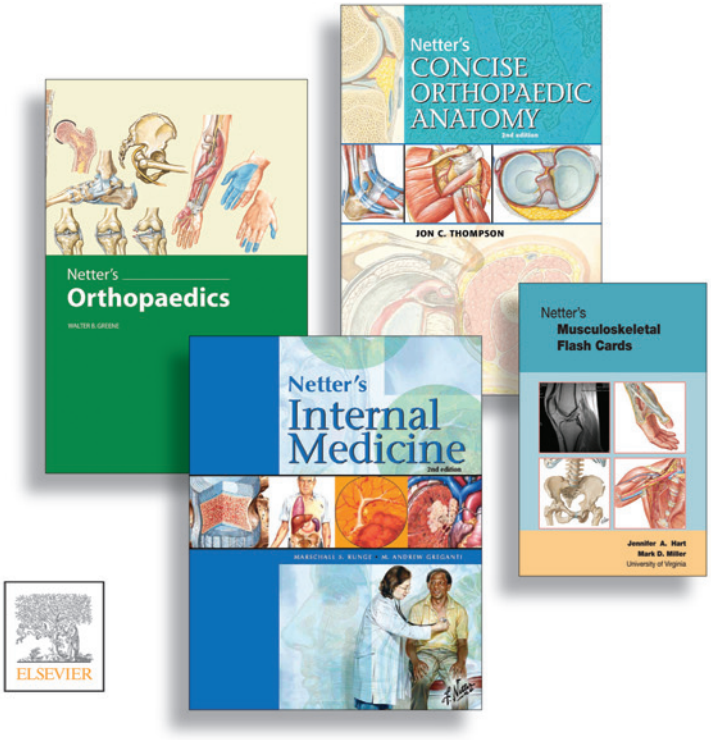

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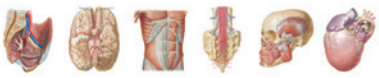

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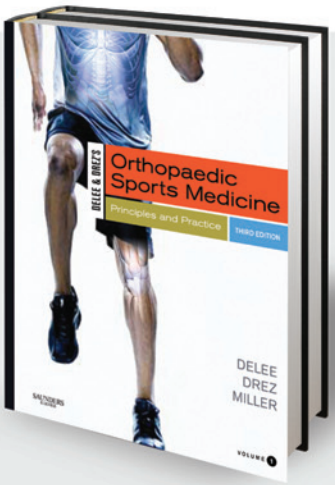

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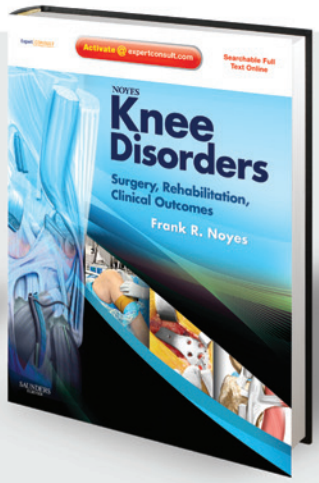

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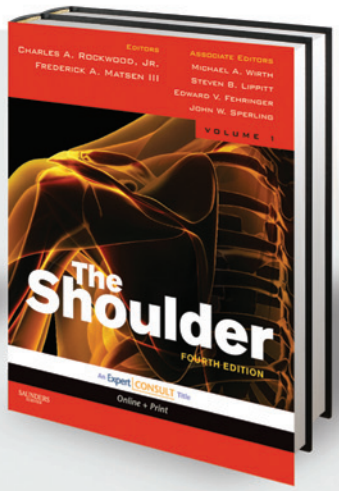

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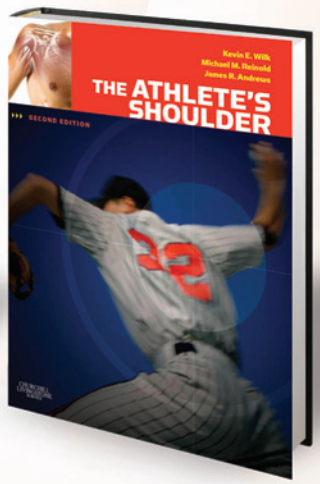

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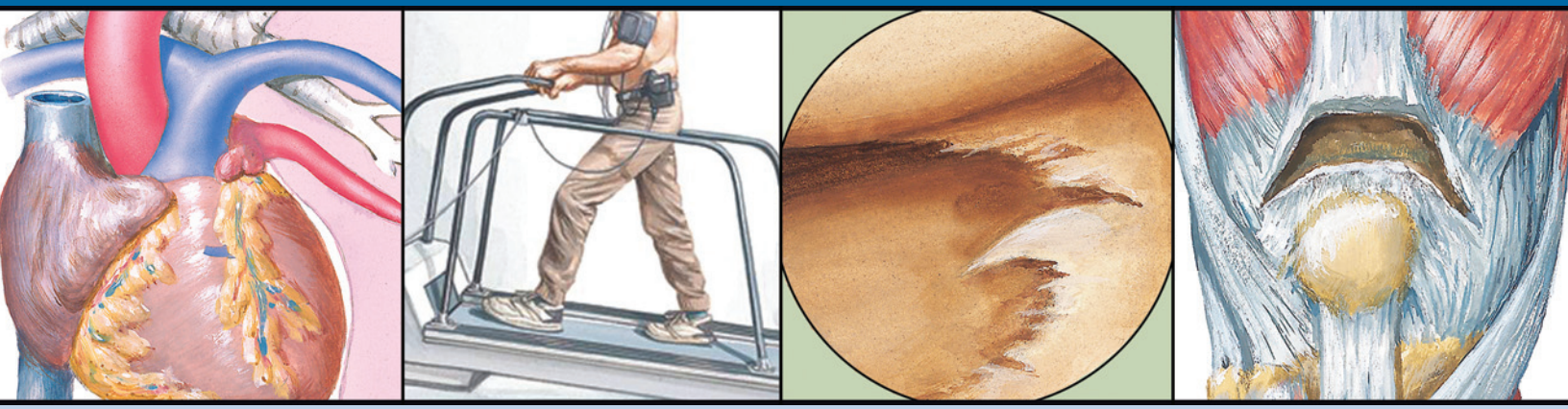

CHRISTOPHER C. MADDEN, MD
MARGOT PUTUKIAN, MD, FACSM
CRAIG C. YOUNG, MD
ERIC C. McCARTY, MD

Illustrations by Frank H. Netter, MD

CONTRIBUTING ILLUSTRATORS
Carlos A. G. Machado, MD
John A. Craig, MD
James A. Perkins, MS, MFA
Kristen Wienandt Marzejon, MS, MFA

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1600 John F. Kennedy Blvd.
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Editorial Assistant: Julie Goolsby
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Dedication

Netter's Sports Medicine is dedicated to the *Team Physician's Handbook* and to the many thousands of sports medicine physicians and health professionals who have loyally followed and evolved with its content over three spectacular editions. *Netter's Sports Medicine* is the formal heir to the *Team Physician's Handbook* and all that the text has embodied for sports professionals through the years. The new title brings with it topic re-organization, the addition of many new chapters and authors, cutting-edge Netter and other graphics, and a new, colorful, user-friendly format. *Netter's Sports Medicine* invites a much broader audience of sports medicine professionals and promises to be a premier-ready reference and a detailed resource for the sports medicine field as a whole. We thank everyone who has followed and supported the past editions of the *Team Physician's Handbook*, and we look forward to sharing many more years and sports medicine experiences with all who endeavor to use this new text to transcend to a new level of quality sports medicine care.

This book is dedicated with respect and honor to the original creators of the *Team Physician's Handbook*, W. Michael Walsh, MD, and Morris B. Mellion, MD, whose vision and wisdom made this work possible. May their love of sports medicine, conduct in the field, and integrity as human beings set an example for us all to follow.

And, to the most wonderful, loving family I can imagine: Jessica, my beautiful wife, and Sage, my energetic and inquisitive daughter. They are both my best teachers, and they bring me infinite happiness.

—Christopher C. Madden, MD

This book is dedicated to my parents, John and Elissa, who taught me to work hard and respect other people; to my brother, Peter, and sister, Lisa; and to my husband and best friend, Joe Hindelang. I treasure the time and memories we're able to spend together. I have learned so much in my career through my mentors, including my fellowship directors Dave Hough and Doug McKeag, as well as so many colleagues (many of whom are contributors). I feel fortunate that I've had an opportunity to participate in this book with such great contributors and editors.

—Margot Putukian, MD, FACSM

This is dedicated to my parents, Margaret and Jimmie, who instilled in me the thirst for knowledge, and to my teachers who had the patience to give me that knowledge, especially to Drs. Jim Puffer, Bob Dimeff, and John Bergfeld. And finally, and most importantly, to my beautiful and wonderful wife, Sharon Busey, who has given me the love, support, and time to complete this project—I couldn't have done this without you.

—Craig C. Young, MD

My dedication of this book goes to my best friend and loving wife, Miriam, who is ever supportive, patient, and enduring in her love, especially in an effort such as this book, with the hours it takes and late nights to make it happen. To my dear children, Madeleine, Cleveland, Shannon, and Torrance, who are the light and diversion to the busy life as an academician, surgeon, and team physician. To my parents, Cleve and Jackie, who gave me great inspiration in pursuing excellence. To my many teachers, mentors, coaches, and orthopedic partners who along the way have taught me so much and continue to live within me. Finally, to my ultimate mentor and the one who gave so much, my savior, Jesus Christ.

—Eric C. McCarty, MD

Editors

Christopher C. Madden, MD
Assistant Clinical Professor
University of Colorado Health Sciences Center
Department of Family Medicine
Sports and Family Medicine Physician, Private Practice
Longs Peak Family Practice
Longmont, Colorado
Team Physician
Niwot High School
Niwot, Colorado

Margot Putukian, MD, FACSM
Director of Athletic Medicine
Head Team Physician
Princeton University
Associate Clinical Professor
Robert Wood Johnson, UMDNJ
University Health Services
Princeton, New Jersey
Team Physician, U.S. Soccer
President 2004-2005
American Medical Society for Sports Medicine

Craig C. Young, MD
Professor of Orthopaedic Surgery and Community and Family Medicine
Medical Director of Sports Medicine
Medical College of Wisconsin
Team Physician
Milwaukee Ballet, Milwaukee Brewers, U.S. National Snowboard Team
President 2007-2008
American Medical Society for Sports Medicine
Milwaukee, Wisconsin

Eric C. McCarty, MD
Associate Professor
Chief of Sports Medicine and Shoulder Surgery
Department of Orthopaedics
University of Colorado School of Medicine
Fellowship Director
Colorado Sports Medicine and Shoulder Fellowship
Director of Sports Medicine
University of Colorado Department of Athletics
Head Team Physician
University of Colorado
Head Team Physician
University of Denver
Denver, Colorado

About the Editors

Christopher C. Madden, MD, is in private practice on the Front Range of the Rocky Mountains in Colorado. His sports medicine practice is broad, and he has special interests in head injuries, biomechanics, environmental and altitude medicine, backcountry sports, endurance and ultra-endurance medicine, snowboarding injuries, and cycling and mountain biking injuries. Chris edits and authors a variety of topics in sports publications, ranging from previsit patient education to professional textbooks. He also teaches sports medicine to primary care residents from Rose and the University of Colorado Family Medicine Residency Programs, in addition to out-of-state residents. He is active in the American Medical Society for Sports Medicine, where he is on the Board of Directors, is past annual meeting Program Chair, has served on the Program Planning and Public Relations Committees, and currently chairs the Economics Committee, where he has developed various sports medicine economics and business practice tools, workshops, and national presentations for the membership. Chris is also involved with the American College of Sports Medicine, where he has served on the Education Committee and has served on and chaired panels at various annual meetings. An avid backcountry enthusiast, Chris loves to tackle epic rides and ultra-endurance, cross-country, and Super D races on his mountain bikes, snowboard and telemark in pristine, off-piste, and backcountry powder, trail run, hike, mountaineer and rock climb, ride motocross, practice yoga, and spend time with his family.

Margot Putukian, MD, FACSM, received her BS degree in Biology from Yale University, where she participated in soccer and lacrosse. She received her MD from Boston University School of Medicine and then did both her internship and residency at the Primary Care Internal Medicine Program at Strong Memorial Hospital in Rochester, New York. She completed her fellowship in sports medicine at Michigan State University in East Lansing, Michigan. Margot worked as the Director of Primary Care Sports Medicine at Penn State University before starting her current position in January 2004 as the Director of Athletic Medicine and Head Team Physician for Princeton University. Margot also serves as a team physician for U.S. Soccer. Margot is a charter member of the American Medical Society for Sports Medicine, where she served as President from 2004 to 2005, and is currently on the Board of Trustees for the American College of Sports Medicine (ACSM). She served previously on the NCAA Competitive Safeguards and Medical Aspects of Sport Committee and is currently serving as the chair of the Sports Science and Safety Committee for U.S. Lacrosse. Margot has participated in the Position Statement on Concussion by the NATA, several Team Physician Consensus Statements, and the Third International Concussion Conference in Zurich, Switzerland. She serves on the editorial board for both Medicine and Science in Sports & Exercise, the Journal of Athletic Training, the Physician and Sports Medicine, and Athletic Training and Sports Health Care. She is the proud recipient of the 2007 Dr. David Moyer Team Physician's award presented by the Eastern Athletic Trainer's Association.

Craig C. Young, MD, is a professor and the Medical Director of Sports Medicine at the Medical College of Wisconsin. He received a BS degree (cum laude) in Biological Sciences from the University of California, Irvine. He is a graduate of the University of California, San Diego School of Medicine. He completed a residency in family medicine at UCLA and a sports medicine fellowship at the Cleveland Clinic Foundation. Dr. Young has served as a team physician for the Milwaukee Brewers since 1994 and company physician for the Milwaukee Ballet since 1992. He has also served as a team physician at the U.S. Olympic Training Center (Chula Vista) and is a team physician for the U.S. National Snowboard Team. In 2007, he was appointed by the U.S. Olympic Committee as a team physician for the 23rd World Winter University Games in Torino, Italy. Dr. Young is board certified in both family practice and sports medicine. He was the President of the American Society for Sports Medicine (AMSSM) from 2007 to 2008. His clinical interests include dance medicine, wilderness medicine, female athletes, adolescent athletes, and endurance athletes. His research interests include dance medicine, in-line skating, and injury prevention.

Eric C. McCarty, MD, is a board-certified and fellowship-trained orthopaedic surgeon with a longtime interest in sports medicine and athletics. He attended college at the University of Colorado, where he excelled and received numerous honors for his exploits in the classroom, as well as on the football field, where he was an All Big-Eight linebacker and also was an Academic All-American. After medical school at the University of Colorado, he completed his training in orthopaedic surgery at Vanderbilt University in Nashville, Tennessee. From there he completed an intensive year of fellowship training in sports medicine and shoulder surgery at the internationally renowned Hospital for Special Surgery in New York City. He subsequently returned to Vanderbilt as a faculty member in the department of orthopaedics. In 2003, Dr. McCarty was recruited from Vanderbilt University to take over the sports medicine and shoulder program and to serve as the head team physician for the University of Colorado and University of Denver athletic programs. His specialized practice involves the care of these collegiate athletes, as well as recreational and highly competitive athletes from the community. In addition to his busy clinical practice, Dr. McCarty is very active in research, teaching, and writing articles in the field of sports medicine and knee and shoulder surgery. He has received grants for his research and frequently gives talks at both the national and international level. Since his playing days, Dr. McCarty continues to maintain a very active lifestyle with his family. He enjoys the activities he grew up with in Colorado, including hiking, cycling, climbing, and skiing. This carries over into his unbridled dedication to returning his patients to their desired activity/sport.

About the Artists

Frank H. Netter, MD, was born in 1906, in New York City. He studied art at the Art Student's League and the National Academy of Design before entering medical school at New York University, where he received his MD degree in 1931. During his student years, Dr. Netter's notebook sketches attracted the attention of the medical faculty and other physicians, allowing him to augment his income by illustrating articles and textbooks. He continued illustrating as a sideline after establishing a surgical practice in 1933, but he ultimately opted to give up his practice in favor of a full-time commitment to art. After service in the United States Army during World War II, Dr. Netter began his long collaboration with the CIBA Pharmaceutical Company (now Novartis Pharmaceuticals). This 45-year partnership resulted in the production of the extraordinary collection of medical art so familiar to physicians and other medical professionals worldwide.

In 2005, Elsevier Inc. purchased the Netter Collection and all publications from Icon Learning Systems. There are now over 50 publications featuring the art of Dr. Netter available through Elsevier Inc.

Dr. Netter's works are among the finest examples of the use of illustration in the teaching of medical concepts. The 13-book *Netter Collection of Medical Illustrations*, which includes the greater part of the more than 21,000 paintings created by Dr. Netter, became and remains one of the most famous medical works ever published. *The Netter Atlas of Human Anatomy*, first published in 1989, presents the anatomical paintings from the Netter Collection. Now translated into 18 languages, it is the anatomy atlas of choice among medical and health professions students the world over.

The Netter illustrations are appreciated not only for their aesthetic qualities, but more importantly, for their intellectual content. As Dr. Netter wrote in 1949 "… clarification of a subject is the aim and goal of illustration. No matter how beautifully painted, how delicately and subtly rendered a subject may be, it is of little value as a medical illustration if it does not serve to make clear some medical point." Dr. Netter's planning, conception, point of view, and approach are what inform his paintings and what make them so intellectually valuable.

Frank H. Netter, MD, physician and artist, died in 1991. Learn more about the Netter Collection of Medical Illustrations at www.netterimages.com.

Carlos A. G. Machado, MD, was chosen by Novartis to be Dr. Netter's successor. He continues to be the main artist who contributes to the Netter collection of medical illustrations.

Self-taught in medical illustration, cardiologist Carlos Machado has contributed meticulous updates to some of Dr. Netter's original plates and has created many paintings of his own in the style of Netter as an extension of the Netter collection. Dr. Machado's photorealistic expertise and his keen insight into the physician/patient relationship inform his vivid and unforgettable visual style. His dedication to researching each topic and subject he paints places him among the premier medical illustrators at work today.

Learn more about his background and see more of his art at http://www.netterimages.com/artist/machado.htm.

Contributors

Joanne B. "Anne" Allen, MD, FAAPMR, FACSM
Orthopedic Specialist
Clinical Adjunct Faculty
University of North Carolina at Wilmington
Wilmington, North Carolina

Annunziato Amendola, MD
Director UI Sports Medicine
Department of Orthopaedics and Rehabilitation
University of Iowa Health Care
Iowa City, Iowa

Jeffrey M. Anderson, MD
Director of Sports Medicine
University of Connecticut Student Health Services
Head Team Physician
Division of Athletics
Medical Director for Research
Human Performance Laboratory
Storrs, Connecticut

Eric J. Anish, MD, FACP, FACSM
Assistant Professor of Medicine
Division of General Internal Medicine
University of Pittsburgh School of Medicine
Pittsburgh, Pennsylvania

Brian Aros, MD
Sports Medicine Fellow
The Ohio State University
Columbus, Ohio

Holly J. Benjamin, MD, FAAP, FACSM
Associate Professor of Pediatrics and Surgery
Section of Orthopedic Surgery and Rehabilitation Medicine
Director of Primary Care Sports Medicine
University of Chicago
Chicago, Illinois

Kris Berg, EdD
Professor
School of Health, Physical Education, and Recreation
University of Nebraska at Omaha
Omaha, Nebraska

O. Josh Bloom, MD
Carolina Family Practice & Sports Medicine
Cary, North Carolina

Michael Broton, MD
Department of Family Med and Community Health
Minneapolis, Minnesota

Kevin E. Burroughs, MD, ABFP, CAQSM
Assistant Professor
Department of Family Medicine
UNC Chapel Hill
Director
Cabarrus Sports Medicine Fellowship
Director of Sports Medicine
Cabarrus Family Medicine Residency
Team Physician
Catawba College
JM Robinson High School
Concord, North Carolina

J. W. Thomas Byrd, MD
Nashville Sports Medicine Foundation
Nashville, Tennessee

Lisa R. Callahan, MD
Associate Attending Physician
Hospital for Special Surgery
Associate Professor of Clinical Medicine
Weill Medical College of Cornell University
New York, New York

William G. Callahan, MD
Clinical Fellow
UC Davis/UC Berkeley Sports Medicine Fellowship Program
Sacramento, California

Dennis Cardone, MD

John C. Carlisle, MD
Chief Resident
Department of Orthopaedic Surgery
Washington University School of Medicine
St. Louis, Missouri

Michael Case, MD
Department of Otolaryngology
Gundersen Lutheran Medical Center
LaCrosse, Wisconsin

Cindy J. Chang, MD, CAQSM, FACSM
Staff Physician
Musculoskeletal and Sports Medicine Specialist
Former Head Team Physician
University of California, Berkeley
Assistant Clinical Professor
Department of Family and Community Medicine
University of California, San Francisco, and University of California, Davis

Leon Y. Cheng, MD
Mercy Medical Group
Department of Internal Medicine
Folsom, California

Stephanie M. Chu, DO

Thomas O. Clanton, MD
University of Texas Medical School–Houston
Department of Orthopaedic Surgery
Team Physician
Houston Rockets and Rice University
Orthopaedic Consultant
Houston Texans
Houston, Texas

Steven J. Collina, MD
Division Chief of Sports Medicine
Director
Crozer-Keystone Sports Medicine Fellowship
Assistant Director
Crozer-Keystone Family Medicine Residency

David B. Coppel, PhD
Private Practice
Kirkland, Washington
Department of Psychiatry & Behavioral Sciences
Department of Psychology
University of Washington
Seattle, Washington

David D. Cosca, MD
Assistant Clinical Professor
UC Davis Sports Medicine
Director
UC Davis/UC Berkeley Sports Medicine Fellowship Program
Sacramento, California

Lauren Costello, MD
Assistant Professor
Princeton University
University of Medicine and Dentistry
Robert Wood Johnson Medical
Princeton, New Jersey

Jonathan C. Crist, MD
Sports Medicine Fellow
Department of Family Medicine
University of Utah
Salt Lake City, Utah

Katherine L. Dec, MD
Medical Director
Women's Sports Medicine
CJW Sports Medicine, LLC
Richmond, Virginia

Michael R. Derosier, MS, ATC
Dartmouth College
Hanover, New Hampshire

William W. Dexter, MD, FACSM
Director
Sports Medicine Program
Main Medical Center
Professor
University of Vermont
College of Medicine
Portland, Maine

Jon Divine, MD, MS
Associate Professor of Pediatrics
Sports Medicine Biodynamics Center
Cincinnati Children's Hospital
University of Cincinnati Medical Center
Cincinnati, Ohio

Sameer Dixit, MD
Interim Head Team Physician
University of California, Berkeley
Berkeley, California

Jesse J. Donnenwerth, PhD, LAT
Staff Athletic Trainer
Department of Intercollegiate Athletics
University of Wisconsin
Madison, Wisconsin

Jonathan A. Drezner, MD
Associate Professor
Department of Family Medicine
University of Washington
Seattle, Washington

Kevin Eerkes, MD
Clinical Assistant Professor
Department of Medicine
New York University School of Medicine
New York, New York

Todd Ellenbecker, DPT, MS, SCS, OCS, CSCS
Clinic Director
Physiotherapy Associates Scottsdale Sports Clinic
National Director of Clinical Research
Chairman
Sports Science Committee, USTA

Jessica Ellis, MD
Primary Care Sports Medicine
Orthopaedic & Rheumatology Associates, P.C.
Bettendorf, Iowa

Steven Erickson, MD
Head Team Physician
Arizona State University
Program Director
Primary Care Sports Medicine Fellowship
ASU/St. Joseph's Hospital and Medical Center
Phoenix, Arizona

Scott Escher, MD
Department of Family Medicine
Division of Sports Medicine
Gundersen Lutheran Medical Center
LaCrosse, Wisconsin

John E. Femino, MD
Chief of Foot and Ankle Service
Department of Orthopaedics and Rehabilitation
University of Iowa Health Care
Iowa City, Iowa

Karl B. Fields, MD
Professor of Family Medicine
Moses Cone Family Medicine Residency Program
Greensboro, North Carolina

David Z. Frankel, MD
Booth Bartolozzi Balderston Orthopaedics
Philadelphia, Pennsylvania

Renata J. Frankovich, BMath, MD, CCFP, DipSportMed
Medical Director
PSI Rehab Group
Palladium Sport Medicine and Physiotherapy Centre
Kanata, Ontario, Canada

Thomas A. Frette, MA, ATC
Senior Associate Athletic Director
University of Nebraska at Omaha
Omaha, Nebraska

Matthew R. Gammons, MD
Assistant Clinical Professor
Department of Family and Community Medicine
Medical College of Wisconsin
Vermont Orthopaedic Clinic
Rutland, Vermont

David J. Gerlach, MD
Chief Resident
Department of Orthopaedic Surgery
Washington University School of Medicine
St. Louis, Missouri

Brett W. Gibson, MD
Department of Sports Medicine
University of Colorado Health Sciences Center
Aurora, Colorado

Danica N. Giugliano, BA
Ludwig Research Fellow
Women's Sports Medicine Center
Hospital for Special Surgery
New York, New York

Laura M. Gottschlich, DO
Assistant Director of Sports Medicine
St. Joseph Family Resident Program
Department of Family and Community Medicine
Department of Orthopedics
Medical College of Wisconsin
Milwaukee, Wisconsin

Gary A. Green, MD
UCLA Division of Sports Medicine
UCLA Olympic Analytical Laboratory
Pacific Palisades Medical Group
Pacific Palisades, California

Scott H. Grindel, MD
Ferris State University
Big Rapids, Michigan
Spectrum Health Reed City Campus
Reed City, Michigan

Mandy Gruner, MS, RD, CSSD
Clinical and Sport Dietitian
Michigan State University
East Lansing, Michigan

Kimberly G. Harmon, MD
Clinical Associate Professor
Department of Family Medicine
Department of Orthopaedics and Sports Medicine
University of Washington
Seattle, Washington

Jennifer A. Hart, MPAS, PA-C
Physician's Assistant
Sports Medicine
Department of Orthopaedic Surgery
University of Virginia
Charlottesville, Virginia

Disa L. Hatfield, MA
Human Performance Laboratory
Department of Kinesiology
University of Connecticut, Storrs
Storrs, Connecticut

Suzanne Hecht, MD, CAQ
Assistant Professor
Department of Family Medicine and Community Health
Division of Sports Medicine Team
University of Minnesota
Physician
University of Minnesota Athletic Department
Minneapolis, Minnesota

John C. Hill, DO, FACSM
Associate Professor
Department of Family Medicine
Director
Primary Care Sports Medicine Fellowship
University of Colorado Health Sciences Center
Denver, Colorado

Kevin M. Honig, MD
Orthopedic Sports Medicine and Shoulder Fellow
Department of Orthopaedics
University of Colorado School of Medicine
Denver, Colorado

Thomas Howard, MD
Director
Sports Medicine Fellowship Program
Fairfax Family Medicine
Fairfax, Virginia

Brian A. Jacobs, MD
Team Physician
Marian High School
Volunteer Physician
Bengal Bouts
University of Notre Dame
Family Medicine of South Bend
South Bend, Indiana

Robert Johnson, MD
Associate Professor
Department of Family Medicine and Community Health
Team Physician
University of Minnesota
Primary Care Sports Medicine
Hennepin County Medical Center
Minneapolis, Minnesota

Christopher C. Kaeding, MD
Professor of Orthopaedics
Director of Sports Medicine
The Ohio State University
Columbus, Ohio

Lawrence Kent, DDS
Gadient, Andreasen, and Associates
Roseville, Minnesota

Morteza Khodaee, MD, MPH
Assistant Professor
Department of Family Medicine
University of Colorado Denver School of Medicine
Denver, Colorado

Melissa D. Koenig, MD
Colorado Permanente Medical Group
Department of Orthopedics
Denver, Colorado

William J. Kraemer, PhD, FACSM, FNSCA, CSCS
Human Performance Laboratory
Department of Kinesiology
University of Connecticut, Storrs
Storrs, Connecticut

Erica L. Kroncke, MD
ThedaCare Orthopedics Plus
Team Physician
Neenah High School
Event Physician
Fox Cities Marathon
Oshkosh, Wisconsin

Alicia Lacovara, BS
University of New Mexico
Albuquerque, New Mexico

Scott R. Laker, MD
Staff Physician
University of Colorado Denver
Department of Physical Medicine and Rehabilitation
Aurora, Colorado

Gregory L. Landry, MD
Professor
Department of Pediatrics
Division of Sports Medicine
University of Wisconsin School of Medicine and Public Health
Madison, Wisconsin

Mark E. Lavallee, MD, CSCS, FACSM
Director
Sports Medicine
Memorial Hospital of South Bend
Co-Director
South Bend Sports Medicine Fellowship Program
Assistant Clinical Professor
Indiana University School of Medicine
Head Team Physician
Indiana University South Bend
Team Physician
Men's Soccer, The University of Notre Dame
Co-Chairman
USA Weightlifting, Sports Medicine Committee
Director
Ehlers-Danlos Clinic
Ehlers-Danlos National Foundation
Professional Advisory Council
South Bend, Indiana

Constance M. Lebrun, MD
Director
Glen Sather Sports Medicine Clinic
University of Alberta
Edmonton, Alberta, Canada

Benjamin D. Levine, MD
Director
Institute for Exercise and Environmental Medicine
S. Finley Ewing Jr. Chair for Wellness
Presbyterian Hospital of Dallas
Harry S. Moss Heart Chair for Cardiovascular Research
Professor of Medicine
University of Texas Southwestern Medical Center at Dallas
Dallas, Texas

Edward Josiah Lewis, MD
Sports Medicine Fellow
Fairfax Family Medicine
Fairfax, Virginia

Cheryl Lindly, MA, ATC, PA-C
Alegen Health Family Care Clinic
Omaha, Nebraska

Colin G. Looney, MD
Orthopaedic Surgery/Sports Medicine
The Bone and Joint Clinic
Franklin, Tennessee

Robert E. Mayle, Jr., MD
Resident
Orthopaedic Surgery
Stanford University
Stanford, California

Teri Metcalf McCambridge, MD
Assistant Professor
Department of Pediatrics
Johns Hopkins School of Medicine
Towson, Maryland

D. Thompson McGuire, MD
Down East Orthopedics
Bangor, Maine

Morris B. Mellion, MD
Past President
American Academy of Family Physicians
Teton Village, Wyoming

Deana Mercer, MD
Resident Physician
University of New Mexico
Department of Orthopaedics and Rehabilitation
Albuquerque, New Mexico

Mark D. Miller, MD
Professor of Orthopaedic Surgery
Sports Medicine Division Head
University of Virginia
Team Physician James Madison University
Charlottesville, Virginia

Cherie B. Miner, MD
Sports Medicine & Orthopedic Specialists, P.C.
Birmingham, Alabama

Marc A. Molis, MD
American Board of Family Medicine
CAQ in Sports Medicine
Sports Medicine of Iowa
Urbandale, Iowa

Whitney E. Molis, MD, FAAP
American Board of Allergy and Immunology
American Board of Pediatrics
Pediatric and Adult Allergy, P.C.
Des Moines, Iowa

George A. Morris, MD
Physician
Chain of Lakes Medical Clinic
Cold Spring, Minnesota

Kinshasa C. Morton, MD
Clinical Instructor
Family and Sports Medicine
UMDNJ–RWJ Family Medicine Residency
New Brunswick, New Jersey

Balakrishnan "Balu" Natarajan, MD
Faculty
Macneal Hospital
Primary Care Sports Medicine Fellowship
Berwyn, Illinois
Private Practice
Chicago, Illinois

Sarah Newman, MS, ATC
Head Athletic Trainer
US Speed Skating, Short Track
Kearns, Utah

Mark W. Niedfeldt, MD
Associate Professor
Departments of Family and Community Medicine, Orthopaedic Surgery, and Cell Biology, Neurobiology, and Anatomy
Milwaukee, Wisconsin

Francis G. O'Connor, MD, MPH
Associate Professor
Military and Emergency Medicine
Medical Director
USUHS Consortium for Health and Military Performance
Department of Military and Emergency Medicine
Uniformed Services University of the Health Sciences
Bethesda, Maryland

David E. Olson, MD
Assistant Professor U of MN
Department of Family Medicine and Community Health
CAQ Sports Medicine
Minneapolis, Minnesota

Amy Jo F. Overlin, MD
ASU Team Physician
Arizona State University
Tempe, Arizona

Lucien Parrillo, MD, MPH
Sports Medicine Fellow
Maine Medical Center
Portland, Maine

Kirtida Patel, MD, CAQSM
Assistant Professor
Department of Family Medicine
East Carolina University
Brody School of Medicine
Greenville, North Carolina

Charles S. Peterson, MD
Instructor of Family Medicine
Mayo College of Medicine
Arizona Sports Medicine Center
Gilbert, Arizona

David J. Petron, MD
Assistant Professor
Department of Orthopaedics
Director
Primary Care Sports Medicine,
Associate Director
Primary Care Fellowship
University of Utah
Salt Lake City, Utah

Sourav K. Poddar, MD
Director
Primary Care Sports Medicine
Assistant Professor
University of Colorado Denver School of Medicine
Team Physician
University of Colorado, Buffaloes
Denver, Colorado

Emily B. Porter, MD
University of Wisconsin
Department of Family Medicine
Madison Residency Program
Verona, Wisconsin

James C. Puffer, MD
President and Chief Executive Officer
American Board of Family Medicine
Lexington, Kentucky

Thomas Pulling, MD
Department of Family Medicine and Community Health
Minneapolis, Minnesota

Martha I. Pyron, MD
Adjunct Professor in Kinesiology
Team Physician for Texas Cheer/Pom/Mascot
Team Physician for UT Club Sport Soccer, Lacrosse, and Rugby
University of Texas at Austin
Sports Medicine Clinic
University Health Services
Austin, Texas

William G. Raasch, MD
Professor and Director of Sports Medicine
Department of Orthopaedic Surgery
Medical College of Wisconsin
Team Physician—Milwaukee Brewers, Milwaukee Ballet, Wisconsin Lutheran College, Wisconsin Lutheran High School
Milwaukee, Wisconsin

Tracy R. Ray, MD
Fellowship Director
Primary Care Sports Medicine
Andrews Sports Medicine and Orthopaedic Center
American Sports Medicine Institute
Birmingham, Alabama

Stephen G. Rice, MD, PhD, MPH, FACSM, FAAP
Director
Jersey Shore Sports Medicine Center
Program Director
Pediatric Sports Medicine Fellowship
Jersey Shore University Medical Center
Neptune, New Jersey
Clinical Associate Professor of Pediatrics
UMDNJ–Robert Wood Johnson Medical School
Piscataway, New Jersey

Michael E. Roberts, MA, ATC
Assistant Athletic Trainer
University of Nebraska at Omaha
Omaha, Nebraska

William O. Roberts, MD, MS, FACSM
Professor
Department of Family Medicine and Community Health
University of Minnesota Medical School
Medical Director, Twin Cities Marathon, Inc.
Minneapolis, Minnesota

Jason A. Robertson, MD
Chattanooga Orthopaedic Group
Center for Sports Medicine and Orthopaedics
Chattanooga, Tennessee

Aaron Rubin, MD, FAAFP, FACSM
Program Director
Kaiser Permanente Sports Medicine Fellowship Program
Academic Faculty
Kaiser Permanente Family Medicine Residency Program
Fontana, California
Team Physician
University of California, Riverside
Patriot High School
Riverside, California

Jane S. Rumball, PhD
Glen Sather Sport Medicine Clinic
Edmonton, Alberta, Canada

Thomas R. Sachtleben, MD, MS, FAAFP
Colorado State University
Department of Sports Medicine
Fort Collins, Colorado

Marc Safran, MD
Professor
Orthopaedic Surgery
Associate Director
Sports Medicine
Stanford University
Stanford, California

Deborah Saint-Phard, MD
Director
Active Women's Health
CU Sports Medicine Program
Associate Professor
Department of Physical Medicine and Rehabilitation
University of Colorado Denver School of Medicine
Aurora, Colorado

Robert C. Schenck, Jr., MD
Professor and Chairman
Division Chief
Sports Medicine Section
Head Team Physician
Department of Orthopaedics and Rehabilitation
Albuquerque, New Mexico

Brock Schnebel, MD
Orthopedic Surgery and Sports Medicine
McBride Clinic, Inc
Oklahoma City, Oklahoma

Wayne Sebastianelli, MD
Director of Athletic Medicine
Assistant Professor
Orthopedic Surgery and Rehabilitation
Penn State University
State College, Pennsylvania

Selina Shah, MD
Center for Sports Medicine
St. Francis Hospital
Walnut Creek, California

Paul S. Sherbondy, MD
Associate Professor
Department of Orthopaedics and Rehabilitation
Milton S. Hershey Medical Center
Hershey, Pennsylvania
Team Physician
Penn State University
Penn State Orthopaedics
University Park, Pennsylvania

Ian Shrier, MD, PhD
Centre for Clinical Epidemiology and Community Studies
SMBD–Jewish General Hospital
Montreal, Quebec, Canada

Robby S. Sikka, MD
TRIA Orthopaedic Center
Minneapolis, Minnesota

Marc R. Silberman, MD
New Jersey Sports Medicine and Performance Center
Warren, New Jersey

Charles D. Simpson II, DPT, CSCS
Minor League Physical Therapist
Boston Red Sox

R. Lance Snyder, MD
Assistant Team Physician
Kansas City Royals Baseball Organization
Clinical Professor
University of Missouri–Kansas City
Department of Orthopedics
Kansas City, Missouri

Kurt Spindler, MD
Professor of Orthopaedics
Director of Sports Medicine
Vanderbilt University
Nashville, Tennessee

Russell G. Steves, MEd, ATC, PT
Coordinator of Physical Therapy
Princeton University
Princeton, New Jersey

James Stray-Gundersen, MD
Cooper Clinic
Dallas, Texas

Josh Takagishi, MD
Clinical Instructor
Michigan State University
Private Practice
Lansing Pediatric Associates
Lansing, Michigan

Gwendolyn A. Thomas, MA, CSCS
Human Performance Laboratory
Department of Kinesiology
University of Connecticut, Storrs
Storrs, Connecticut

Thomas H. Trojian, MD
Sports Medicine Fellowship Director
Associate Professor
Department of Family Medicine
Team Physician
University of Connecticut
Saint Francis Hospital and Medical Center
Storrs, Connecticut

Kimberly A. Turman, MD
Fellow Sports Medicine
Department of Orthopaedic Surgery
University of Virginia
Charlottesville, Virginia

Amy Elizabeth Valasek, MD
Assistant Pediatric Resident
Johns Hopkins School of Medicine
Baltimore, Maryland

Jeremy J. Vanicek, MPAS, PAC
OrthoWest, P.C.,
Omaha, Nebraska

Nathan van Zeeland, MD
Chief Resident
Vanderbilt University
Department of Orthopaedics and Rehabilitation
Vanderbilt Orthopaedics Institute
Nashville, Tennessee

Carole S. Vetter, MD
Associate Professor
Orthopaedic Surgery
Associate Program Director
Orthopaedic Surgery Residency
Division of Sports Medicine
Medical College of Wisconsin
Milwaukee, Wisconsin

Armando F. Vidal, MD
Department of Sports Medicine
University of Colorado Health Sciences Center
Aurora, Colorado

Bryant Walrod, MD, CAQ
Sports Medicine
Team Physician
Carthage College
Kenosha, Wisconsin
Team Physician
University of Wisconsin–Parkside
Kenosha, Wisconsin
Assistant Clinical Professor
Medical College of Wisconsin
Milwaukee, Wisconsin
Comprehensive Orthopaedics
Kenosha, Wisconsin

W. Michael Walsh, MD
Orthopaedic Surgeon
OrthoWest, P.C.
Clinical Associate Professor
Department of Orthopaedic Surgery
University of Nebraska Medical Center
Adjunct Graduate Associate Professor
School of Health, Physical Education, and Recreation
University of Nebraska at Omaha
Team Orthopaedic Surgeon
University of Nebraska at Omaha
Omaha, Nebraska

Jeffry T. Watson, MD
Assistant Professor
Vanderbilt University
Department of Orthopaedics and Rehabilitation
Vanderbilt Orthopaedics Institute
Nashville, Tennessee

Douglas R. Weikert, MD
Associate Professor
Vanderbilt University
Department of Orthopaedics and Rehabilitation
Vanderbilt Orthopaedics Institute
Nashville, Tennessee

Russell D. White, MD
Professor of Medicine
Director, Sports Medicine Fellowship Program
Medical Director, Sports Medicine Center
University of Missouri–Kansas City
Kansas City, Missouri
Fellow of the American College of Sports Medicine
Fellow of the American Academy of Family Medicine
Diplomate of the American Board of Family Medicine
Certificate of Added Qualifications in Sports Medicine
Certificate of Added Qualifications in Geriatric Medicine
Certificate of Added Qualifications in Adolescent Medicine
American Association of Clinical Endocrinologists

Kevin E. Wilk, PT, DPT
Associate Clinical Director
Champion Sports Medicine
A Physiotherapy Associates Clinic
Birmingham, Alabama
Rehabilitation Consultant
Tampa Bay Rays Baseball Organization
St. Petersburg, Florida
Associate Clinical Professor
Programs in Physical Therapy
Marquette University
Milwaukee, Wisconsin

John J. Wilson, MD
Primary Care Sports Medicine Fellow
Division of Sports Medicine
University of Wisconsin School of Medicine and Public Health
Madison, Wisconsin

Michelle Wolcott, MD
Department of Sports Medicine
University of Colorado Health Sciences Center
Aurora, Colorado

Rick W. Wright, MD
Associate Professor
Residency Director
Co-Chief Sports Medicine
Washington University School of Medicine
St. Louis, Missouri

Preface

It is an honor to combine the widespread popularity of the contents of *Team Physician's Handbook* with the revered anatomical graphic works of Frank Netter, MD. The new book comes to life with a re-invigorated, colorful, bulleted outline format combined with Netter graphics, helpful tables, figures, pictures, diagnostic imaging, and other medical artwork.

Carrying forward the momentum of the original *Team Physician's Handbook*, we have asked two new enthusiastic editors to join us. Craig Young is a professor in the Departments of Orthopedic Surgery and Community and Family Medicine, Medical Director of Sports Medicine, and Sports Medicine Fellowship Director at the Medical College of Wisconsin. He is the past president of the American Medical Society of Sports Medicine, and he maintains strong interests in dance medicine, wilderness medicine, and endurance athletes. Eric McCarty is the Chief of Sports Medicine and Shoulder Surgery in the Department of Orthopaedics at the University of Colorado Denver School of Medicine. He serves as Director of Sports Medicine and Head Team Physician for the University of Colorado athletic department, and he maintains strong interests in collegiate sports injuries, shoulder instability, degeneration and rotator cuff injuries, and knee cartilage restoration in the athletic individual. Chris Madden and Margot Putukian have combined forces with the two new energetic editors to recruit a national and international author base that represents the best in sports medicine today.

Serving as a Team Physician is a unique privilege and an awesome challenge. *Netter's Sports Medicine* is written for the multitude of physicians and other health care professionals who are fortunate enough to provide care to a variety of athletes and active individuals in almost any athletic setting imaginable, from pediatric to senior athletics, Little League to professional sports, weekend warrior to Olympic champion, and backcountry mountainside to Super Bowl field. The book is designed to serve as a comprehensive sports medicine resource and a ready reference in the busy outpatient office, in the training room, on the sideline, and in the long, quiet hours of preparation for sports medicine board certification. Insightful, expert, anecdotal experience fills the void where the most current evidence in sports medicine falls short, and careful considerations of controversies are mindfully presented. The sports medicine literature has grown exponentially since the first three editions of *Team Physician's Handbook*, and many new chapters and chapter sections were added and revised to reflect the depth and breadth of our exciting field. The text is divided into user-friendly sections for quick reference, and each chapter hosts a Recommended Readings section limited to only the best sources. We welcome many new, respected authors who joined us to produce this book, and we are fortunate to continue our lasting relationship with numerous previous authors, who are leaders in their respective areas of emphasis. We thank all the authors who contributed chapters to previous texts, and whose chapter templates provided a strong foundation to build upon.

Whether you are a primary care physician attempting to manage a common or unique musculoskeletal injury in an efficient ambulatory setting, an orthopaedic surgeon trying to gain insight about a medical or psychological problem foreign to the cast or operating room, an athletic trainer trying to figure out a diagnosis in the training room, or a physical therapist pursuing further in-depth sports medicine knowledge, we sincerely hope you find this reference all it is meant to be and more, and we thank you for opening the cover and sharing with us what we feel is one of the highest quality sports medicine works produced to date. Please enjoy.

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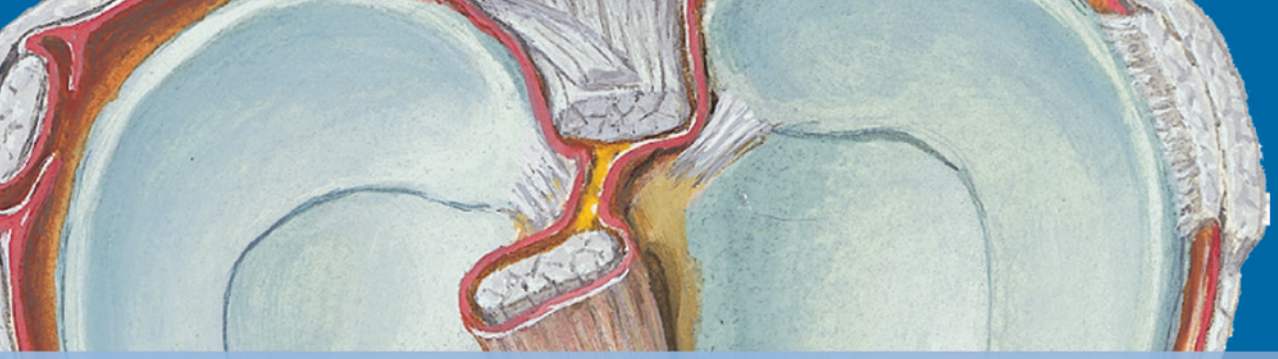

SECTION

I

Medical Care and Supervision of the Athlete

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The Team Physician

Christopher C. Madden, Margot Putukian, Craig C. Young,
Eric C. McCarty, Morris B. Mellion, and W. Michael Walsh

BEING A TEAM PHYSICIAN: A SPECIAL PRIVILEGE, AN AWESOME CHALLENGE

Special Role

Team physicians have a unique responsibility for important decisions. They are expected by athletes, parents, and school, community league, or professional team administrators to make major decisions about athletes' health, qualifications to join the team, and ability to participate safely. These decisions are often made in a setting of intense time pressure. Team physicians may affect the competitive success of the team, as well as the athlete. They often influence the athlete's mental, economic, and physical well-being. Level of performance, scholarships, and professional opportunities may depend on timely, high-quality medical care.

Addressing the Needs of the Athlete

The team physician addresses the physical, emotional, and spiritual needs of the athlete in the context of the sport and the needs of the team. The success of team physicians depends on their ability and training to meet the athletes' broad range of medical and psychosocial needs. Family physicians, pediatricians, general internists, and other generalists are potentially well-suited by training to function as team physicians. This is particularly true when they have training and experience in sports medicine. Other specialists, such as orthopedists, general surgeons, and emergency physicians, have unique skill sets that when combined with training and experience in sports medicine that make them able to function well as team physicians. Six major professional associations concerned about clinical sports medicine issues collaborated to develop two important documents for team physicians: **Team Physician Consensus Statement** (see Appendix A) and **Sideline Preparedness for the Team Physician: Consensus Statement** (see Appendix B). All other Team Physician Consensus statements are referenced in "Recommended Readings."

To perform effectively, the team physician must maintain a broad, up-to-date knowledge base that addresses athletics, as well as medicine. All team physicians should feel comfortable providing emergency care at sporting events. Training in CPR is essential, and additional knowledge of advanced cardiac life support (ACLS) and advanced trauma life support (ATLS) is useful. In addition, a team physician should have knowledge in the following areas:

Medicine: musculoskeletal system, growth and development, cardiorespiratory function, gynecology, neurology, nephrology, gastroenterology

Psychology and behavior: mental health issues such as depression, anxiety, eating disorders, alcohol and other drug use/abuse, response to injury

Pharmacology: therapeutics, performance aids, recreational drugs, interactions of these agents, and their effects on performance

Exercise science: exercise physiology, biomechanics, specific sports

Responsibilities of the Team Physician

The team physician has a range of ethical responsibilities that reflect the many relationships involved in the care of the team. Responsibilities to the athlete, the team, and the institution and its representatives must be balanced.

Responsibilities to Athletes

To allow participation: The team physician should not arbitrarily disqualify athletes from participation for reasons that are insignificant or out of line with current thinking. Especially in school-based programs, athletes have a right to participate if there is no valid medical contraindication.

To protect: Athletes must be protected from injury, reinjury, permanent disability, and themselves. When there is valid medical contraindication to participation or resumption of participation, athletes must be counseled and thoroughly informed. It may be especially difficult to reason with an athlete who has a "participate at any cost" attitude.

To educate: It is the responsibility of the team physician to educate the athlete as to the nature of the injury and the potential treatment options. This also includes educating the athlete on potential risks and benefits that may be associated with the treatment options. Additionally, the team physician educates the athlete about injury prevention.

To provide optimal health care: Physicians must strive for quality health care, while being mindful of the costs of diagnostic tests and treatment and of the athlete's competitive schedule.

To ensure confidentiality: Physicians must balance the demands of the Health Insurance Portability and Accountability Act of 1996 (HIPPA), which limit the release of information, with the rights of the team under worker's compensation law and the need for others (e.g., trainers and coaches) to plan appropriate activities for the athlete's medical condition. Confidentiality is often challenged by the relationship to the school or professional club. Seldom may information be held in strict physician-patient confidentiality. However, the team physician must be sensitive to how widely information is disseminated. For example, if an athlete wishes to resign from the team without the physician telling others the medical reasons for this decision, that wish should be honored.

It is a cardinal rule that the team physician may not sacrifice the welfare of the athlete to the welfare of the team or the institution. Rob Johnson, in commentary on ethics and legal issues in *Clinics in Sports Medicine*, appropriately states: "Hand in hand with altruism is an ethical sense that is both accepted by the physician and expected by those who seek that physician's guidance and counsel."

Responsibility to the Team

To facilitate success of the group: Team members have all dedicated time and effort to the sport. Team physicians will be constantly challenged by tensions that arise from balancing individual athlete care against nonmedical ends such as winning an event.

Responsibilities to the Coach

To facilitate success: The coach should not view the team physician as an impediment to success but rather as a part of the team striving for success. Thus, good communication among the athlete, physician, and coaching staff is vital.

To educate: Continuing education about improvements in medical and preventive care is important to eliminate archaic and possibly harmful techniques. Sometimes agendas of coaches and team physicians differ. "Doctors should doctor, and coaches should coach." The health of the athlete is the ultimate responsibility of the team physician, and a skilled team physician needs to be mindful of what information a coach wants and needs, and how to deliver it effectively (Boyd).

To protect from possible future liability: Team physicians need to be ready to serve as mindful guides when the agenda of a coach or team is not supportive of appropriate health care decisions for an injured athlete.

Responsibilities to the Institution

To facilitate success in light of financial commitment:

- To provide optimal health care for the athletes
- To prescreen scholarship and professional athletes. In this regard, the team physician does not have a doctor-patient relationship and may disqualify athletes who fail to meet physical standards.
- For professional athletes, team physicians should be aware of worker's compensation laws, disability insurance, and union rehabilitation rules.

To protect from liability: Physicians should incorporate a "broad, institutional" level of thinking when they are addressing individual injuries.

Balancing Life

Balancing personal life and team physician duties is both incredibly demanding and rewarding. Mary Lloyd Ireland eloquently lays out ten commandments that help achieve this balance:

1. Do the right thing, always, no exceptions.
2. It is better to be an advocate than curse your competition.
3. Don't demand respect from players and coaches, earn it.
4. Loyalty is the weakest of human values.
5. Communicate: team physicians must always be available to athletic training staff.
6. In order to hit the mark, one must aim a little higher.
7. Enjoy your role as a team physician.
8. Remember the five A's: **a**vailability, **a**bility, **a**ffability, **a**dvocacy, **a**ffiliation.
9. Dare to care.
10. Don't forget your family and friends.

Availability

Availability is a cornerstone for success as a team physician. Personal availability and a well-organized coverage system are essential.

On the sidelines: The sidelines are the front lines of sports medicine, especially for contact sports. A physician who covers a team solely from the stands or the office does not truly deserve the title "team physician."

In the training room: It is important to demonstrate interest in the team by seeing athletes in their own environment, rather than only in the physician's environment.

In the office: Team physicians may wish to make special accommodations in their office schedules for athletes with urgent problems.

Nights and weekends: Most athletic activity goes on outside the normal work day. Team physicians should always be available to coaches or trainers.

Unstructured time with the athletic trainer and/or coach: Responsibilities that may influence **time demands** include the following:

- Preseason and exit physicals
- Coverage expectations
- Travel expectations
- VIP event expectations
- Number of total teams/athletes
- Length of seasons
- Sole or shared team physician responsibilities

Who Serves as Team Physician?

A broad spectrum of generalists and specialists serve as team physicians (Table 1-1).

Physicians may share responsibility, such as a generalist with a specialist. For example, a primary care physician may pair with an orthopedic surgeon. This situation may be ideal, because the majority of injury problems are musculoskeletal. A primary care physician (family physician, pediatrician, internist) and an orthopedist should be able to deal effectively with most problems that arise. They can then call on other specialists as necessary.

Table 1-1 *THE PHYSICIAN AND SPORTS MEDICINE* TEAM PHYSICIAN LIST, JUNE 1995

	Number	Percentage
Family practice	7788	25.5
Orthopedic surgery	4972	16.2
Osteopathic medicine	3347	10.9
Internal medicine	3078	10.1
General practice	1925	6.3
Pediatrics	1643	5.4
Emergency medicine	1499	4.9
General surgery	1382	4.5
Obstetrics/gynecology	847	2.8
Cardiology	608	2.0
All other specialties	3508	11.5
Total	**30,597**	**100.0**

Data from *The Physician and Sportsmedicine* circulation department.

Rewards from Service

Team physicians derive a variety of rewards from service in this capacity.

Satisfaction: Immense personal satisfaction can be derived from providing a service to the community and working with young, motivated patients.

Credibility: Undoubtedly, affiliation with teams from high school to professional level enhances a physician's prestige in the community and may contribute to practice building.

Remuneration: Serving as a team physician should be a labor of love. Time may be the greatest negative financial burden you accept as a team physician. At anything less than a professional team level, most of the time spent is as a volunteer. Above the high school level, some compensation may be a part of the agreement. However, remuneration is extremely variable, ranging from none to a considerable retainer with a professional club. Some colleges and universities provide team physician services through Student Health or an affiliated medical school, but most of these arrangements include large amounts of volunteer time as well. Surgeons may receive surgical fees for procedures performed, but often these services are provided on a discount basis. Four general contractual arrangements to serve as a team physician for an institution, all of which can directly or indirectly have financial impact, include: volunteer, soft-money arrangement, fee for service, and paying to serve as a team physician. Note: In 2005 the American Orthopaedic Society for Sports Medicine (AOSSM) passed a resolution that discourages paying to serve as a team physician. Two professional leagues, the National Football League and Major League Baseball, have also passed rules against linking team health care coverage to sponsorship contracts.

Relationship of Team Physician to Institution

It is important for both physician and institution to **establish an explicit formal relationship between the physician and the school, league, or team.** It should include job description, any fiscal arrangements, and a statement of expectations. The contract should address all parties discussed, team responsibilities, physician responsibilities, and specific terms of contract. Whenever possible, especially if monetary arrangements are involved, it should be in writing.

Employer of Team Physicians

The athletic director, athletic trainer, business manager or other officer of the professional team hires or obtains the services of the team physician. Standard of quality guidelines for selecting a team physician are suggested in a 2005 AOSSM publication:

- The selection of a team physician should be based fundamentally on the physician's credentials and ability to provide the highest level of care available.
- The process of selecting the team physician should include input from multiple parties that have an interest in the well-being of the players.
- The selection of team medical staff should not be based on financial incentives offered by the physician and/or his or her institution.
- The team should fully disclose any sponsorship, advertising, or financial arrangement the medical staff (or their institution) have made with the team.
- The team and medical staff should ensure appropriate communication (with legal limitations) to players, other medical providers, and management to provide for a more open understanding regarding the health care environment.

Job Description of Team Physician

The description of a team physician's position should include the following:

- Person to whom team physician reports
- Services provided by team physician at home and away (reimbursement for travel expenses, if any)
- Remuneration and benefits
- Any other expectations of the institution or the physician

Principles and Guidelines for Care

The team physician must have professional autonomy over all medical decisions and be protected against coercive pressures.

Team physicians should realistically assess time commitment and ability to meet demands of position.

THE SPORTS MEDICINE TEAM

The team physician cares for an athletic team and also serves as a key player on the sports medicine team, which consists of the athlete, the team physician, the coach, and the athletic trainer, when one is available. Each of these individuals has a support system to draw upon. The care of the athlete is a team effort in which the members of the sports medicine team support each other for the benefit of the athlete and the athletic team. Like the athletic team itself, sports medicine services are best provided following a team concept of this kind (Fig. 1-1).

The Athlete's Support System

- Teammates
- Family and significant others
- Friends
- Teachers
- Athletic trainers
- Coaching staff

The Team Physician's Support Systems

Clinical support: medical specialists, physical therapists, sports psychologists/psychiatrists, nutritionists, dentists, podiatrists, equipment managers, health educators

Research support: medical researchers, exercise physiologists, sports psychologists, kinesiologists, nutritionists, physical educators, sociologists, equipment industry

Team and school support: Athletic trainer, coaching staff, strength and conditioning staff, administrative staff

The Coach's Support System

- School and league administration
- Athletic director
- Coaching staff
- Athletic trainer
- Equipment manager
- Medical staff

The Athletic Trainer

The athletic trainer occupies a unique position at the center of the athletic health care triangle:

- Eyes and ears for the team physician (triage or screening; supervision of conditioning, care, and rehabilitation; continuous functional evaluation of the athlete)
- Works under the auspices of the team physician for evaluating injuries and medical problems in the athlete
- Therapist and counselor for the athlete
- Advisor and friend to the coach

Ideally every high school and college athletic program should have a certified athletic trainer.

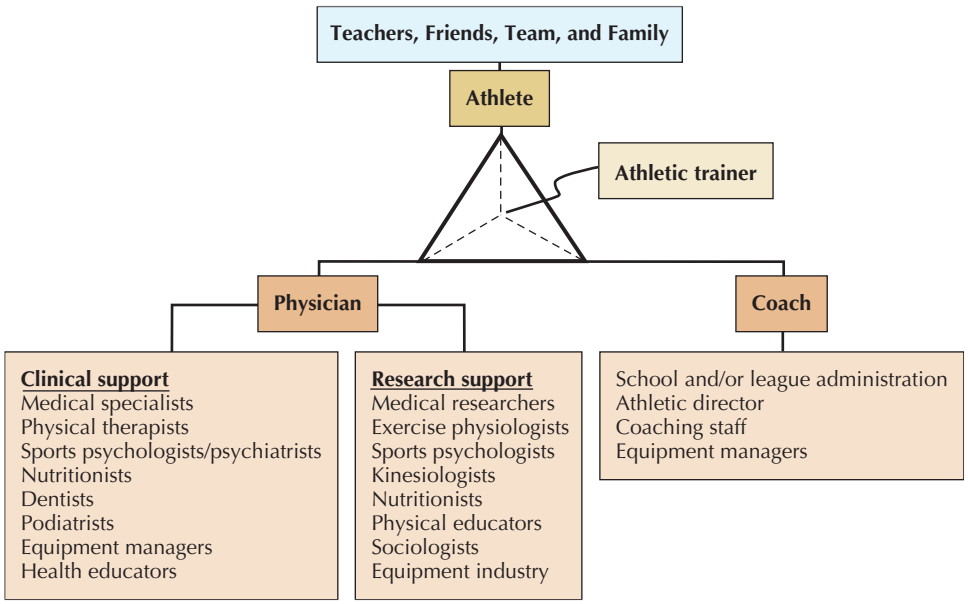

Figure 1-1 The sports medicine team. *(Modified from Mellion MB: Office Sports Medicine. Philadelphia: Hanley & Belfus, 1996.)*

ROLES AND FUNCTIONS OF THE TEAM PHYSICIAN

Medical Supervision of Athletes

The traditional function of a team physician, which has now been greatly expanded, includes the following roles and functions:

Prevention

- The team physician is responsible for the **preparticipation evaluation:** qualification of athletes (general and sport-specific), counseling on appropriate sports, treatment and rehabilitation of deficits.
- The team physician is often asked for advice on **proper conditioning techniques** to prevent or rehabilitate injuries (preseason and in-season, general and sport-specific).
- The team physician may be asked for advice on **protective equipment** (selection, fit, injury, and reinjury prevention).
- The team physician may be asked for advice on **nutrition, hydration,** and **supplementation** (caloric requirements, event and nonevent fluids, ergogenic aids).

Planning

Team physicians, athletic trainers, and other medical personnel should develop a detailed plan for the evaluation, treatment, and disposition of injuries on the field or in other venues. Knowledge of sport-specific risks and injuries and familiarity with the physical environment will help team physicians anticipate potential problems. Plans and protocols should be in writing when possible (see Appendix B) and should cover preseason, game day/event, and postseason problems.

Supervision

Supervision should include on-site coverage at field, gym, arena, or pool, as well as coverage of tournaments, training rooms, and mass events. A physician should be present for high-risk situations and high-risk sports. When a physician is not available for coverage, an athletic trainer or other personnel trained in prevention, recognition, and initial evaluation and care of injured athletes should be present.

Preparticipation Evaluation

The team physician conducts the preparticipation evaluation and evaluates illness and injury on the field, in the training room, in the team physician's office, in the student health clinic, and/or in the emergency department.

Management

Management includes treatment (Is the athlete at risk for further injury or can the athlete safely return to play? Is the athlete comfortable with the decision after reviewing risks and benefits?), consultation and referral (when appropriate), and documentation.

Administration

The team physician may be expected to perform a variety of administrative functions, including the following:

- **Develop a general system of care for the team.**
- Establish a **chain of command** with a **team leader.**
- **Establish guidelines for consultation** with the team physician or referral to a consultant.
- **Plan and organize the preparticipation evaluation:**
  - Determine content and standards of the evaluation
  - Establish guidelines for participation
  - Arrange location
  - Secure and coordinate personnel
- **Prearrange a system of emergency care, and plan and rehearse** important emergency procedures and protocols. Examples include lightning policy, natural disaster response, and terrorist threats, etc. Most important is the development of emergency action plans (EAPs) that are site and sport specific. Characteristics of a successful **EAP,** modified from the Inter-Association's Task Force Recommendations on Emergency Preparedness and Management of Sudden Cardiac Arrest in High School Athletic Programs Consensus Statement, may include:
  - Sponsoring schools or institutions should have a structured and written EAP.
  - EAPs should be developed and coordinated in consultation with local EMS, school public safety officials, on-site first responders, and school administrators.
  - EAPs should be specific to venue and encompass emergency communication, equipment, personnel, and transportation to emergency facility.
  - EAPs should be reviewed and practiced annually by the sports medicine team.
  - Targeted first responders should ideally have training in CPR and automated external defibrillator (AED) use when possible.
  - In cases of sudden cardiac death, access to early defibrillation within 3 to 5 minutes is ideal.
  - Review of the EAP and available equipment by on-site personnel for each athletic event is desirable.

Availability of General Medical Equipment and Supplies

The team physician should **ensure the availability** of general medical equipment and supplies (as influenced by length of journey, destination, age and condition of participants, and number of participants and spectators [see Appendix B]), emergency equipment, emergency transport (ambulance present at high-risk events), referral to a hospital that is equipped to care for anticipated injuries, and communication facilities (e.g., telephone on sidelines, radio).

Coordination and Medical Supervision

- Medical personnel
- Athletic trainers
- Paramedical personnel

Legal and Medicolegal Issues

- Contract with school, league, or team
- Permission to treat minors
- Liability (institutional and professional)
- Athlete's right to participate
- Treatment of athletes on out-of-state trips
  - Legality: For *major* athletic events, the host state or country generally passes legislation granting visiting team physicians temporary licenses. For routine competitions and tournaments, it is recommended that the traveling team physician work through the host team or tournament physician or local physician in the host town.
  - "Good Samaritan" laws: No suit has yet been brought against a team physician traveling with a team to another state over the issue of practicing without a license. "Good Samaritan" laws focused on the care of athletes have been passed in many states. These laws vary from one jurisdiction to another. They generally do not protect team physicians who receive compensation for their services.
  - Professional liability insurance coverage: Physicians must ensure that their medical malpractice policies cover their duties as a team physician, especially when traveling nationally and sometimes internationally.

Insurance Coverage

- Insurance may be provided by the school, league, or team.
- Various insurance coverage problems will be encountered (e.g., capitated medical care systems such as **HMOs; PPOs;** military dependents; coverage for preventive services; coverage for cognitive versus procedural services).

- Regarding **malpractice insurance,** make sure physician coverage extends to the sideline, training room, and team setting. Coverage is frequently more expensive with professional and high-level sports.

Communication/Liaison

The ideal team physician is a skilled communicator who can often resolve conflicts or enhance cooperation among members of the athletic and sports medicine teams. A good team physician establishes and reasonably enforces an appropriate chain of command and precise, well-rehearsed protocols, and expertly manages the general flow of information.

- Certain relationships often require special attention: athlete–coach, athlete–parents, team physician–parents, team physician–athlete's family doctor, athlete–medical colleagues and consultants, athletic trainer–coach, injured athlete–press.
- In many settings, it may be beneficial to send a preseason letter to athletes and/or their parents describing the role of the team physician and what services are provided. Below the college level, such letters should emphasize the importance of the student athlete's own family physician or pediatrician in the student's care.
- Athlete confidentiality is frequently challenged by **demands of the public arena,** especially with higher-level and spotlight sports. **Team physicians should be well versed in careful and responsible media communication. Team physicians and the athletes should discuss what information may be relayed to the media, and team physicians should obtain permission to release information prior to the media conference.**

Education

The team physician can serve as an educator at many levels and to many audiences in sports medicine.

Audiences include athletes, coaches, athletic trainers, administration (especially athletic directors), medical personnel (medical students, house officers, colleagues, and consultants), paramedical personnel, parents, and the general public.

Methods include one-on-one instruction and preceptorship; in-service training; lectures, workshops, and seminars; formal instructional courses; newsletters; audio and video instructional tapes; books and articles in sports and professional journals; and computerized training modules.

Continuing medical education: In addition to educating others, the team physician should regularly attend educational events that continue to develop his or her skills in sports medicine.

Effective team physician educators are always prepared to teach, lead by example, understand their audience, encourage dialogue among members of the sports medicine team, and use a multidisciplinary approach when needed.

Research

Sports medicine is a relatively young discipline with a growing scientific literature. The team physician can improve the health care and safety of athletes by promoting and performing sports medicine research.

Student

The team physician is responsible for remaining current with a body of knowledge that changes rapidly and has the opportunity to learn about life, sport, and medicine from highly motivated athletes, coaches, and other members of the sports medicine team.

Healer

Even in this highly technical era of medicine, the intangible effect of the "laying on of hands" and the supportive care of the team physician are often the keys to recovery and participation for the athlete.

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The Certified Athletic Trainer and the Athletic Training Room

2

Michael R. Derosier and Cheryl Lindly

HOW THE CERTIFIED ATHLETIC TRAINER AND PHYSICIAN FUNCTION AS A TEAM

Standing Orders

- **By law, ATCs function under the auspices of a physician. As such, the physician under whom an ATC is practicing should have standing orders to help guide the ATC.** The National Athletic Trainers' Association website (www.nata.org) has several guidelines and position statements posted as a reference for ATCs and physicians to use when making policy and procedure decisions.
- **Standard operating procedures should be written whenever possible** for emergency care, transportation, modalities, use of nonprescription medication, and other treatments and procedures.
- Written procedures regarding when and how quickly an athlete should be seen by the physician are useful. In addition, communication between the athletic trainer and the physician they are working under is essential in providing ongoing athlete care.
- The ATC **coordinates referrals** among physicians and other health care providers. For an example, see the "When to Refer an Athlete to a Physician After Concussion" section of a paper published by Guskiewicz and colleagues in the *Journal of Athletic Training* in 2004 (see "Recommended Readings"). That document now serves as the NATA's Position Statement on Management of Sport-Related Concussion.
- The ATC develops and implements **game/practice coverage procedures,** including the following:
  - Establishes roles in emergency situations for on-field and off-field assessment. All members of an organization who are responsible for the medical care of athletes should work together to develop an emergency action plan (EAP). Andersen and colleagues published a paper in the *Journal of Athletic Training* in 2002 that now serves as the NATA's Position Statement on Emergency Planning in Athletics (see "Recommended Readings").
  - Provides emergency equipment (see Chapter 4). The advent and popularity of automated external defibrillators (AED) over the past decade and a half may be the most significant change to what is now considered "standard emergency equipment." The NATA has issued a position statement regarding the need for an AED program as part of an athletic trainer's EAP. The need for such programs is also heralded in a paper by Drezner and colleagues, who spearheaded a multiassociation task force that authored a Consensus Statement regarding sudden cardiac arrest (SCA) in high school and college athletics (see "Recommended Readings").
- The ATC, together with the physician they are working under make **return-to-play decisions**—defines parameters (range of motion, strength, functional ability, functional taping/bracing).
- The ATC maintains **open lines of communication** on treatment and rehabilitation, return to competition, and emergency care.

ROLES OF THE CERTIFIED ATHLETIC TRAINER (ATC)

- **Responsible** for prevention, emergency care, first aid, evaluation, and rehabilitation of injuries to athletes under his or her care
- **Liaison** between the team physician, the athlete, the athlete's parents, and coaching staff
- **Consultant** to the coaching staff on conditioning, nutrition, and protective equipment

EDUCATION AND PROFESSIONAL CREDENTIALING OF THE ATC

Achieving Certification by the National Athletic Trainers' Association Board of Certification (NATABOC)

- An individual becomes a certified athletic trainer (ATC) by passing a national certification examination that is administered by the National Athletic Trainers' Association Board of Certification (NATABOC). In order to sit for this exam, candidates must graduate from an accredited entry-level program (bachelor's or master's) in no less than 2 years. As of July 1, 2006, there were approximately 352 athletic training education programs (ATEP) accredited by the Commission on Accreditation of Athletic Training Education (CAATE).
- All accredited programs consist of curricula that include, but are not limited to, the following:
  - Human anatomy and physiology
  - Exercise physiology
  - Basic and advanced athletic training
  - Therapeutic exercise and therapeutic modalities
  - Acute care
  - Assessment and evaluation (general medical and injury)
  - Nutrition
  - Psychology
  - Pharmacology
  - Athletic training administration
- An alternate route to certification, previously referred to as the Internship Pathway, was abolished as of December 31, 2003.

State Certification, Licensure, and Registration

- Currently, 44 states certify, license, register, or otherwise regulate athletic trainers practicing in their states.
- State certification may or may not reflect the same competencies measured in the NATABOC certification exam.

ROLES AND RESPONSIBILITIES OF THE CERTIFIED ATHLETIC TRAINER

Prevention of Injuries

- Education of athletes and student athletic trainers
- Conditioning—development of conditioning programs
- Preseason musculoskeletal screening
  - Identify factors that put athlete at risk
  - Correct deficiencies:
    - "Prehabilitation"—begin work on deficits before injuries occur or preoperatively for scheduled surgeries
    - Make a referral for further workup
- Prescribe taping and bracing when needed.

Injury Evaluation

Acute injuries, chronic injuries, referral to team physician or specialist when appropriate

Immediate Care

- Necessary equipment available
- Communication procedures for emergency situations—scenarios and procedures rehearsed by entire staff
- Prompt, accurate triage

Treatment, Rehabilitation, and Reconditioning

- Protect (i.e., crutches, immobilization, etc.), Rest, Ice, Compression, Elevation, Support (PRICES) (see Chapter 36)
- Qualified use of modalities: cold or heat, ultrasound, electrical stimulation
- Development and supervision of individual therapeutic exercise programs
- Evaluation and modification in program as progress deems necessary
- Return to competition—functional testing

Organization and Administration

- Development of policies and procedures for personnel and facility
- Oversight of athletic training room personnel
- Administration of budget for athletic training room
- Oversight of insurance reimbursement and billing for student athletes
- Maintenance of medical records

Professional Responsibility

- Adhere to statutory, regulatory, and case law relating to the practice of athletic training.
- Maintain status of good standing through NATABOC by fulfilling continuing education requirements.

Training Room Management

Record keeping: injury reports, home care instructions, referrals, treatment records, rehabilitation progress notes, insurance
Budgeting: consult with team physician regarding specific needs
Equipment and supplies:

- **Basic athletic training room supplies and equipment**
  - Taping
  - Wound care
  - Rehabilitation devices—e.g., weights, surgical tubing, exercise equipment, proprioception boards
  - Reconditioning equipment—e.g., stationary bicycle, upper body ergometer, and other cardiovascular equipment
  - Modalities—e.g., whirlpool, ultrasound, ice machine or freezer, hydrocollator, electrical stimulator
- **Emergency equipment and supplies**
  - Automated external defibrillator (AED)
  - CPR mask
  - Spine boards, cervical immobilizer, stretchers
  - Splints, extremity immobilizers, slings
  - Crutches
- **Equipment available for team physician use only** (see Chapter 1)
  - prescription medications
  - suture kit cardiorespiratory emergency medications
  - oxygen

Student athletic trainers:

- Education
  - hands-on experience
  - didactic methodology
  - preparation for certification examination
- Supervision
- Establishment of competencies before taping, evaluations, or application of modalities

Professional Relationships with Support Personnel

- Coaches, including strength and conditioning coaches
- School administrators
  - Nutritionists/dieticians
  - School nurses
  - Physical therapists
  - Emergency services

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The Preparticipation Physical Evaluation

3

Morteza Khodaee, Christopher C. Madden, and Margot Putukian

PREPARTICIPATION PHYSICAL EVALUATION (PPPE)

- History and physical examination performed before participation in sport that meets several objectives and is one of the most important functions provided by the sports medicine physician.
- Often the first interaction between the physician and the athlete; for many young adults, it is the first exposure to the health care system.
- Does not take the place of regular physical examinations, although many athletes think that it covers all health care needs.
- Encompasses clearance for participation in sport, and provides education and information to athletes about issues such as nutrition, supplementation, training and conditioning, injury prevention, and rehabilitation.
- Special considerations of PPPE include age specificity, gender specificity (special concerns for female vs. male athletes), and sport specificity (specific demands of each sport should be considered).

OBJECTIVES OF THE PPPE

- Emphasize cardiovascular, neurologic, and musculoskeletal issues.
- Identify any life-threatening or disabling conditions (e.g., underlying cardiovascular or neurologic abnormalities).
- Identify any conditions that may place athlete at risk for injury or illness (e.g., underlying ligamentous instability, musculoskeletal abnormalities, organomegaly, or acute medical illness).
- Assess for an injury that has not been properly rehabilitated.
- Assess for medical conditions and strength and flexibility deficits that place athlete at risk for injury.
- Assess general health status (e.g., immunizations), fitness, and maturity.
- Meet insurance or legal requirements.
- Screen for menstrual dysfunction, stress fractures, eating disorders (female athlete triad).
- Introduce athletes to health care system and preventive medicine concepts.
- Offer an opportunity to address issues such as recreational and performance-enhancing substance use and abuse, sexuality issues, depression and emotional issues, and health promotional activities (seat belts, helmets, self breast or testicular examination).

TIMING

- The PPPE should be performed at least 6 weeks before the beginning of the sport season to allow adequate time for further evaluation of identified problems and treatment or rehabilitation of any conditions or injuries.
- If athletes are unavailable 4 to 6 weeks before the start of an early fall season, examinations performed at the end of the previous school year may be considered. Athletes should report any interval injuries or illnesses between their exam and the start of the fall season.
- A detailed medical history may be completed by athletes and/or parents in advance, which may improve the accuracy of the information (e.g., immunization record) and exam efficiency. Internet resources can facilitate the history and interval injury reporting process.

FREQUENCY

- Variable recommendations depend on individual athlete (i.e., age, gender, sport [single or multiple], health [underlying medical conditions, injury history], cost); availability of records from past PPPEs (continuity of care); and requirements of state, city, or athletic governing body.
- **General guidelines** (no consensus about optimal frequency)
  - Comprehensive baseline PPPE before initiating a new sport or attaining a new level (e.g., entry into high school, college, or professional level), every 2 years in younger athletes (e.g., middle school and high school students), and every 2 to 3 years in older athletes.
  - Subsequent annual PPPEs may be limited to injuries or illnesses disclosed by an interim health questionnaire; yearly evaluation of cardiopulmonary system may be appropriate.
  - If in multiple sports during the year, consider more frequent evaluations.
  - Many **states** require a full screening exam annually.
  - The **NCAA** requires an initial comprehensive PPPE on entrance followed by interim history in intervening years; limited additional exams focusing only on new problems.
  - The **American Heart Association** (AHA) recommends initial comprehensive PPPE on entrance for high school and college athletes. The AHA recommends another comprehensive PPPE after 2 years for high school athletes and follow-up interim history and blood pressure measurements annually and focused additional exams for new problems for college student athletes.

METHODOLOGY

Office-Based

Potential advantages: physician–patient familiarity, privacy, and continuity of care.

Potential disadvantages: greater cost, limited appointment time, limited physician interest/experience, and lack of communication of pertinent information to school athletic staff.

Coordinated Medical Team-Based (Table 3-1)

Potential advantages: specialized personnel, time and cost efficiency, and good communication with school athletic staff.

Potential disadvantages: rushed examinations, lack of privacy, and inadequate follow-up of identified problems.

Two types of group PPPEs: multistation (multiple physicians, each at specialized station) and "locker room" (single or multiple physicians performing complete exams individually, each in own area [e.g., locker room]).

Recommendations

- At the final station of a station-based exam, an experienced team physician should be available to review all of the data and to determine clearance or provide appropriate recommendations.
- Communication between other primary or consulting physicians, athletic trainers, coaches, and parents may be enhanced by carefully documenting problems and specific recommendations in the clearance section of the PPPE form.
- Cases of special concern may warrant a phone conversation between the team physician and other involved health care providers.

Table 3-1 REQUIRED AND OPTIONAL STATIONS AND PERSONNEL FOR COORDINATED PREPARTICIPATION PHYSICAL EVALUATION

Required stations	Personnel
Sign-in, height and weight (BMI*), blood pressure, vision	Ancillary personnel (coach, nurse, community volunteer)
History review, physical examination†, clearance	Physician
Optional stations	**Personnel**
Nutrition	Dietitian
Dental	Dentist
Injury evaluation‡	Physician
Flexibility	Trainer or therapist
Body composition	Physiologist
Strength	Trainer, coach, therapist, physiologist
Speed, agility, power, balance, endurance	Trainer, coach, physiologist

*Body mass index can be calculated from height and weight (for specific age- and gender-adjusted categories see www.cdc.gov/growthcharts).
†The physical examination can be subdivided if more than one physician is present.
‡A musculoskeletal injury evaluation station may be used to provide a more complete evaluation when a musculoskeletal injury is detected during the required musculoskeletal screening examination.

- The 2005 *Preparticipation Physical Evaluation* monograph (see "Recommended Readings") considers the "gymnasium examination" to be inadequate to achieve the goals and objectives of the PPPE process.

Personnel

Physicians

- The PPPE should be performed by an MD or DO physician.
- Regulations by some states at the high school level allow other practitioners (e.g., chiropractors or naturopathic clinicians) to perform PPPEs.
- Primary care physicians perform the majority of PPPEs because of their ability to evaluate all organ systems (i.e., cardiopulmonary, musculoskeletal, neurologic, ophthalmologic, gastrointestinal, genitourinary, dermatologic).
- Specialists such as orthopedic surgeons, cardiologists, and ophthalmologists or optometrists are key consultants and may be on site during the screening-station format examination.

Ancillary

- Medical staff, including athletic trainers, physical therapists, nurses, exercise scientists, dietitians, and sports psychologists, may be involved, especially in the screening-station format PPPE.
- Nonmedical staff, including coaches, school administrators, and community volunteers, are especially helpful in the screening-station format PPPE.

MEDICAL HISTORY

- There is emphasis on screening for cardiovascular and musculoskeletal problems, prior head injuries and other neurologic problems, and significant recent illnesses. In addition, prior heat illness, pulmonary problems, medication problems, inadequate immunizations, allergic reactions, skin problems, and, in female athletes, menstruation abnormalities and disordered eating should be addressed (Fig. 3-1). The medical history is an essential component of the PPPE that detects abnormalities in the majority of athletes.
- Joint completion of history forms by athletes and parents/guardians is recommended when possible, especially if the athlete is unclear about family or personal history. In addition, parent/guardian should be present or available during PPPE for additional questions that may arise.
- **Cardiovascular history:** (Box 3-1)
  - Screen for causes of sudden cardiac death (SCD; Fig. 3-2). The most common cause in people younger than 35 years is hypertrophic cardiomyopathy (HCM; Fig. 3-3); in people older than 35 years, the most common cause is coronary artery disease (CAD). The PPPE is scrutinized by some physicians for its ability to detect underlying causes of SCD, especially at younger ages. Yet the American Heart Association states that some form of preparticipation screening for high school and college athletes is justifiable and compelling based on ethical, legal, and medical grounds.
  - Determine past history of invasive or noninvasive cardiac tests.
  - **Family history is important.** Twenty-five percent of first-degree relatives of patients with HCM have morphologic evidence in echocardiography.
  - **Prior history of hypertension or prehypertension noted during exams, or family history for hypertension important.**

Neurologic Concerns

- It is important to ask questions about previous head or neck injury, concussion, neurologic symptoms, stingers/burners, and seizure disorder.
- Any positive response mandates more thorough history, physical exam, and evaluation.

Musculoskeletal Concerns

- Complete history is essential.
- Ask about previous ligamentous injuries, documentation of surgery, rehabilitation, time out of play.
- Any positive response mandates careful attention during physical exam: assessment of ligamentous instability, strength and flexibility deficits/mismatches, and completeness of rehabilitation.
- If athlete has had prior surgery, obtain documentation that the operating surgeon has cleared the athlete to return to competition and/or determine the athlete's rehabilitation status.

Previous Medical Illnesses

- Heat exhaustion/illness
- Infectious mononucleosis
- Hepatitis
- HIV disease
- Diabetes
- Sickle cell disease
- Asthma
- Allergic reactions

Female Athlete Triad

Screening questions:

- Age of menarche, history of amenorrhea or oligomenorrhea
- History of stress fractures, bone injury, risk factors for osteoporosis
- History and risk factors for disordered eating patterns: questions that ascertain ideal versus current body weight, body image concerns, pathogenic eating behaviors

Additional Concerns

Additional concerns not always included on the PPPE form may be addressed on an individual basis. If you don't ask, you might not find out.

Preparticipation Physical Evaluation

Medical History Form

Date of exam_____

Name_____ **Birth date** ____/_____/____ **Age** _____ **Gender** _____
Grade _____ **School** _____ **Sport (Primary)** _____ **Sport (Other)** _____
Address _____ **Phone** _____
E-mail: _____ **Primary care physician** _____
Emergency contact:
Name _____ **Relationship** _____ **Phone (H)** _____ **(W)** _____

Explain "Yes" answers below.
Circle question number of questions for which the answer is unknown.

	Yes	No
1. Have you ever been denied or restricted to participate in sports for any reason by a physician?	☐	☐
2. Do you have any current or chronic medical condition (like diabetes or asthma) for which you have seen a physician on a regular basis?	☐	☐
3. Are you presently taking any prescription or non-prescription (over-the-counter) medicines or pills?	☐	☐
4. Do you have allergies to medications, foods, pollens, stinging insects, or any products?	☐	☐
5. Have you ever had chest tightness, cough, wheeze, and/or shortness of breath during or after exercise?	☐	☐
6. Have you ever felt dizzy, lightheaded, and/or passed out during or after exercise?	☐	☐
7. Have you ever had the feeling of your heart racing or skipping beats during or after exercise?	☐	☐
8. Do you get tired more quickly than your teammates or friends during exercise?	☐	☐
9. Have you ever been told by a physician that you have a heart murmur, high blood pressure, high cholesterol, or a heart infection?	☐	☐
10. Have you ever had an electrocardiogram (EKG) and/or echocardiogram (ECHO) of your heart?	☐	☐
11. Has anyone in your family died of heart problems or sudden death before age 50?	☐	☐
12. Has anyone in your family less than 50 years old had unexplained drowning while swimming or an unexplained car accident?	☐	☐
13. Does anyone in your family have Marfan syndrome?	☐	☐
14. Have you ever been admitted overnight to a hospital?	☐	☐
15. Have you ever had surgery?	☐	☐
16. Have you ever had an injury, like a sprain, muscle or ligament tear that caused you to miss a game or practice?	☐	☐
17. Have you ever had any broken or fractured bones, or dislocated joints?	☐	☐
18. Have you ever had a bone/joint injury that required x-rays, MRI, CT, surgery, injections, rehabilitation, physical therapy, a brace, a cast, or crutches? If yes, circle below: Head Neck Shoulder Chest Upper arm Elbow Forearm Hand/Fingers Upper back Lower back Hip Thigh Knee Calf/Shin Ankle Foot/Toes	☐	☐
19. Have you ever had a stress fracture?	☐	☐
20. Have you ever been told that you have or have you had an x-ray for atlantoaxial (neck) instability?	☐	☐
21. Do you regularly use corrective braces or assistive devices?	☐	☐
22. Have you ever been diagnosed with asthma, exercised-induced asthma, and/or allergies?	☐	☐
23. Does anyone in your family have asthma?	☐	☐
24. Do you or have you ever used an inhaler?	☐	☐
25. Do you develop a rash or hives when you exercise?	☐	☐
26. Do you have any rash or other skin problems?	☐	☐
27. Have you had a herpes skin infection?	☐	☐
28. Have you had mono (infectious mononucleosis) within the last month?	☐	☐
29. Were you born without or are you missing a kidney, an eye, a testicle, or any other organ?	☐	☐
30. Have you ever had a head injury/concussion?	☐	☐
31. Have you ever been knocked out, become unconscious, and/or lost your memory due to a head injury/concussion?	☐	☐
32. Have you ever had a seizure?	☐	☐
33. Do you have headache with exercise?	☐	☐
34. Have you ever had numbness, tingling, or weakness in your arms or legs after being hit or falling?	☐	☐
35. Have you ever had a sudden burning pain, numbness, and/or inability to move your arms or legs after being hit or falling?	☐	☐
36. Have you ever suffered from a heat-related injury (like heat muscle cramps or heat exhaustion)?	☐	☐
37. Do you or any member of your family have sickle cell trait or sickle cell disease?	☐	☐
38. Have you had any problems with your eyes or vision?	☐	☐
39. Do you wear glasses or contact lenses?	☐	☐
40. Do you wear protective eyewear, such as goggles or a face shield?	☐	☐
41. Are you unhappy with your weight?	☐	☐
42. Has anyone recommended you change your weight or eating habits?	☐	☐
43. Do you limit or carefully control what you eat?	☐	☐
44. Do you have any concerns that you would like to discuss with a doctor?	☐	☐

Females only
Have you ever had a menstrual period?_____
How old were you when you had your first period? _____
How many periods have you had in the last 12 months? _____

Explain "Yes" answers here: _____
_____
_____
_____
_____

I do not know of any existing physical or additional health reason that would preclude participation in sports. I certify that the answers to the above questions are true and accurate and I approve participation in athletic activities.
Student-Athlete's signature _____ Parent's/Guardian's signature_____ Date_____

Figure 3-1 Preparticipation physical examination history form. *(Adapted from Minnesota State High School League, 2009-2010 sports qualifying physical forms. Available at http://www.mshsl.org/mshsl/publications/code/forms/PhysicalExam.pdf. Accessed on April 26, 2009; and from Preparticipation Physical Evaluation, 3rd ed. Minneapolis: McGraw-Hill/Phys Sportsmed, 2005.)*

- Nutritional issues: fluids, game-day nutrition, general nutrition
- Supplements and performance-enhancing agents
- Sexuality concerns: pregnancy, sexually transmitted diseases, sexual orientation (best addressed in private setting)
- Recreational drugs and alcohol use
- Preventive medicine: seat belts, helmets, self breast or testicular exam, cholesterol screening, gynecologic exams/Pap smear
- Psychosocial issues: stress management, anxiety, depression, suicide

PHYSICAL EXAMINATION (Box 3-2)

- The physical exam should be comprehensive. It should focus on areas of greatest importance in sports participation and address any problems uncovered by the history.
- Adequate exposure during the exam is important.
- The **physical examination form** (Fig. 3-4) is general in scope but should not limit the clinician if additional examination is deemed pertinent.

BOX 3-1 *The 12-Element American Heart Association Recommendations for Preparticipation Cardiovascular Screening of Competitive Athletes*

Rights were not granted to include this textbox in electronic media. Please refer to the printed publication.

BOX 3-2 *Standard Components of the Preparticipation Physical Evaluation*

- Height
- Weight
- Eyes: visual acuity, pupil size
- Oral cavity
- Ears
- Nose
- Lungs
- Cardiovascular system: blood pressure, pulses, (femoral, radial), heart (rate, rhythm, murmurs)
- Abdomen: masses, tenderness, organomegaly
- Genitalia (males only): single or undescended testicle, testicular mass, hernia
- Skin: rashes, lesions (infectious)
- Musculoskeletal system: contour, range of motion, and symmetry of neck, back, shoulder/arm, elbow/forearm, wrist/hand, hip/thigh, knee, leg/ankle, foot

From Preparticipation Physical Evaluation, 3rd ed. Minneapolis: McGraw-Hill/Phys Sportsmed, 2005, p 23.

Height and Weight

- In athletes with excessive weight change, explore possibility of eating disorders or steroid abuse.
- Body mass index should be calculated (gender and age specific; see www.cdc.gov/growthcharts).
  - Underweight (<5th percentile)
  - At risk of overweight (85th-95th percentile)
  - Overweight (≥95th percentile)

Head, Eyes, Ears, Nose, and Throat (HEENT)

- Eye exam is important: check visual acuity in all athletes, pupils for anisocoria, conjunctiva for anemia.
- Swimmers (otitis externa), scuba divers (otic barotrauma), and wrestlers (auricular hematoma) need ear exams.
- Allergy sufferers and athletes with history of nose trauma need nasopharynx exams.
- Smokeless tobacco users need oropharynx exams.

Cardiovascular Assessment

Cardiovascular assessment is essential for both initial PPPE and annual reevaluations (see Box 3-2).

Blood pressure measurement (with appropriate cuff size): if elevated, recheck after the athlete rests quietly for 15 minutes and later if needed (see Table 3-2). The following are classification categories of hypertension in children and adolescents (see www.nhlbi.nih.gov/guidelines/hypertension/hbp_ped.htm):
- Normal (<90th percentile for age, sex, and height)
- High-normal (90th-95th percentile for age, sex, and height)
- Hypertension (95th-99th percentile for age, sex, and height)
- Severe hypertension (>99th percentile for age, sex, and height)

Palpate radial and femoral pulses:
- Decreased or nonpalpable femoral pulses raise suspicion for coarctation of aorta.
- Irregular pulse raises suspicion for arrhythmia and requires ECG evaluation.

Heart auscultation in supine and standing positions: note presence and character of any murmurs.
- **HCM murmur:** systolic murmur heard best at lower left sternal border with standing; increases with maneuvers that decrease venous return to the heart.
- Provocative maneuvers help to differentiate functional murmurs from pathologic murmurs.
  - To decrease venous return: Valsalva maneuver, squat-to-stand.
  - To increase venous return: deep inspiration, stand-to-squat, isometric hand grip.
- General recommendations for murmurs requiring further evaluation before athlete can participate:
  - Any systolic murmur grade ≥ III/VI in severity
  - Any diastolic murmur
  - Any murmur that gets louder with Valsalva maneuver

Marfan syndrome stigmata (Fig. 3-5): Tall stature, arachnodactyly, kyphoscoliosis, anterior chest deformity, arm span greater than height, decreased upper body length to lower body length ratio, heart murmur or midsystolic click, ectopic lens, thumb sign (the thumbs protrude from the clenched fists), wrist sign (the distal phalanges of the first and fifth digits of one hand overlap when wrapped around the opposite wrist), and family history of Marfan syndrome.

36th Bethesda Conference Guidelines (Maron et al.): excellent resource for physicians with concerns about evaluation and clearance of most cardiovascular problems.

Pulmonary Assessment

- Focus on detecting abnormal breath sounds: wheezes, crackles, rubs, abnormal inspiratory-to-expiratory ratio

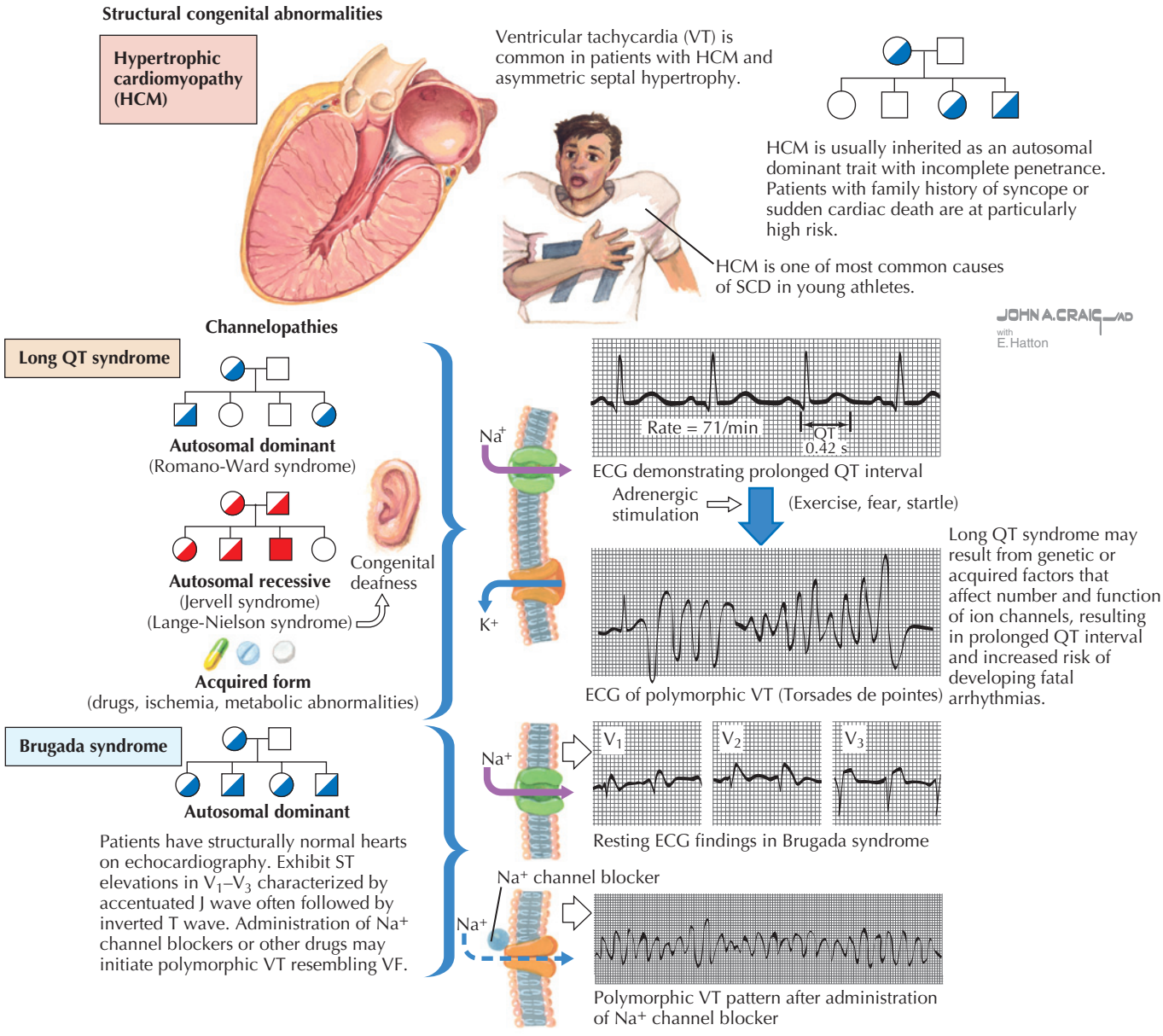


Figure 3-2 Sudden Cardiac Death.

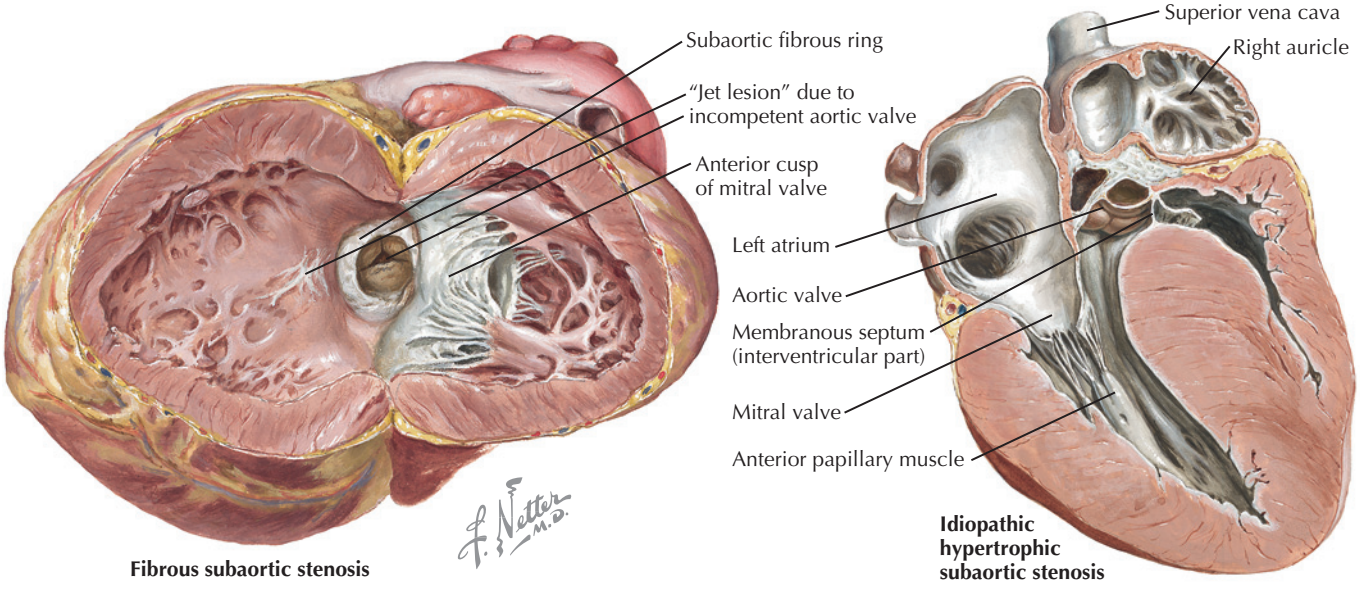


Figure 3-3 Hypertrophic Cardiomyopathy.

Preparticipation Physical Evaluation

Physical Examination Form

Name _____ **Birth date** _____ /_____/_____
Height _____ **Weight** _____ **BMI** _____ **% Body fat (optional)** _____
Pulse _____ **BP** ____ /____ **(repeat** _____ /_____ ,_____ /_____ **) Pupils: Equal** _____ **Unequal** _____
Vision R 20/_____ **L 20/**_____ **Corrected: Y N**

Follow-Up Questions About More Sensitive Issues: **Yes No**

1. Do you feel stressed out or under a lot of pressure? ☐ ☐
2. Do you ever feel so sad or hopeless that you stop doing some of your usual activities for more than a few days? ☐ ☐
3. Do you feel safe? ☐ ☐
4. Do you smoke cigarettes, use smokeless tobacco, or tobacco in any form? ☐ ☐
5. During the past 30 days, have you had at least 1 drink of alcohol? ☐ ☐
6. Have you ever taken steroid shots without a doctor's prescription? ☐ ☐
7. Have you ever taken any supplements to help you gain or lose weight or improve your performance? ☐ ☐
8. Question "Risk Behaviors" like guns, seatbelts, unprotected sex, domestic violence, drugs, and others. ☐ ☐

Notes About Follow-Up Questions: _____

	Normal	Abnormal findings	Initials*
Medical			
Appearance			
Eyes/ears/nose/throat			
Hearing			
Lymph nodes			
Heart			
Pulses			
Lungs			
Abdomen			
Genitalia (males only)			
Skin			
Musculoskeletal			
Neck			
Back			
Shoulder/arm			
Elbow/forearm			
Wrist/hand			
Hip/thigh			
Knee			
Leg/ankle			
Foot			

*Required only if multiple examiners

Physician name (print/type/stamp) _____
Address _____ **Phone:** _____
Physician's signature (MD or DO) _____ **Date:** _____ /_____ /_____ /_____

Figure 3-4 Preparticipation physical examination form. *(Adapted from Minnesota State High School League, 2009-2010 sports qualifying physical forms. Available at http://www.mshsl.org/mshsl/publications/code/forms/PhysicalExam.pdf. Accessed on April 26, 2009; and from Preparticipation Physical Evaluation, 3rd ed. Minneapolis: McGraw-Hill/Phys Sportsmed, 2005.)*

- Asthma screening (e.g., exercise challenge test) has been suggested but is impractical in most preparticipation settings.

Abdominal/Gastrointestinal Assessment

Should be performed with athlete in supine position. Problems requiring further evaluation before participation include organomegaly (liver and spleen), masses, tenderness and/or rigidity, and, in females, possible pregnancy.

Genitourinary Assessment

- Male: undescended testes, absence of testicle, hernia, mass; counsel about cancer screening with self-exam.

Table 3-2 CLASSIFICATION OF HYPERTENSION (HTN)

Age and phase	90th-95th percentile† High normal* Prehypertensive‡	95th-99th percentile† Significant HTN* Stage 1 HTN‡	>99th percentile† Severe HTN* Stage 2 HTN‡
6-9 yr			
Systolic†	104-121	108-129	>115-129
Diastolic†	68-81	72-89	>83-89
10-12 yr			
Systolic†	112-127	116-135	>123-135
Diastolic†	73-83	77-91	>84-91
13-15 yr			
Systolic†	117-135	121-142	>128-142
Diastolic†	76-85	80-93	>87-93
16-17 yr			
Systolic†	121-140	125-147	>132-147
Diastolic†	78-89	82-97	>90-97
≥ 18 yr			
Systolic‡	120-139	140-159	≥160
Diastolic‡	80-89	90-99	≥100

†The BP range is from the Fourth Report on the Diagnosis, Evaluation, and Treatment of High Blood Pressure in Children and Adolescents. For specific BP levels by age, gender, and height see http://www.nhlbi.nih.gov/guidelines/hypertension/child_tbl.pdf.

*American Academy of Pediatrics, Task Force on Blood Pressure in Children: Report of the Second Task Force Control on Blood Pressure in Children—1987. Pediatrics 79:1-25, 1987. Updated in 98(4 Pt 1):649-658, 1996.

‡From the Seventh Report of the Joint National Committee on Detection, Evaluation, and Treatment of High Blood Pressure, 2003.

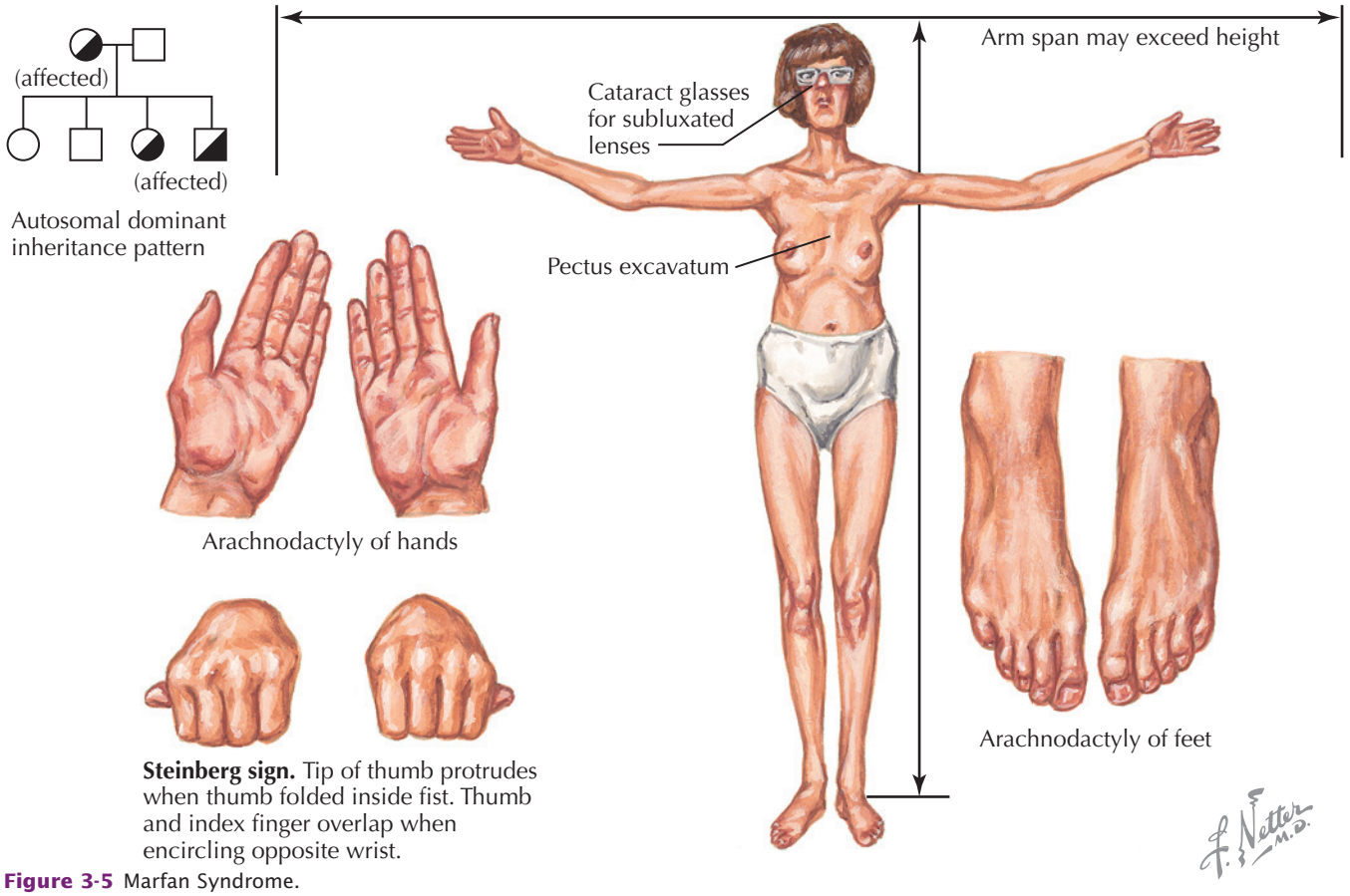


Figure 3-5 Marfan Syndrome.

- Female: routine exam not recommended; if warranted by history or other findings, examine in private setting.
- Tanner staging no longer recommended in PPPE monograph because of controversy over using it for injury prevention and psychological benefits; may be useful for counseling on growth and development in boys 11 to 17 years old.

Musculoskeletal Exam

- Musculoskeletal exam is important to identify musculotendinous, bone, or joint problems that may limit athletic participation or predispose to acute injury or long-term complications (e.g., shoulder instability, anterior cruciate-deficient knee, unrehabilitated ankle sprain, juvenile rheumatoid arthritis).
- General screening exam is most efficient for asymptomatic athletes with no prior musculoskeletal injuries (Fig. 3-6).
- Joint-specific exam: recommended for problematic areas; most accurate and most time-consuming.
- Back flexion is recommended to screen for thoracolumbar deformities like scoliosis (Fig. 3-7).
- Sport-specific exam: some advocate need for focusing on commonly injured or stressed areas in particular sports (e.g., shoulder exam for throwers, tennis players, swimmers; knee exam for basketball, football, and soccer players); adds measures of endurance, strength, and flexibility to orthopedic screen but time-consuming and requires in-depth knowledge of particular sports.
- Consider screening for flexibility (e.g., back, hamstrings, Achilles tendon) because clinical anecdotal evidence suggests that increasing flexibility reduces risk of overuse problems (e.g., mechanical back pain, patellofemoral pain, medial tibial stress syndrome). Studies, however, do not support decreased risk of acute injury (e.g., sprains, strains, dislocations).

Neurologic Assessment

Neurologic assessment (gross motor) is generally screened through musculoskeletal evaluation. Perform a more comprehensive neurologic exam in athletes with unexplained strength deficits, paresthesias, history of burners/stingers, history of head injury/concussion, or any focal or generalized neurologic deficit.

Other Assessment

Assessment for other problems, such as lymphadenopathy, thyromegaly, physical findings of eating disorders, and skin conditions, should be considered on an individual basis.

Fitness and Performance Evaluation

- Secondary (ideal) objective of PPPE
- More often performed in group screening-station format
- Measures any or all of the following parameters:
  - body composition (skinfold, underwater weighing, circumferences)
  - flexibility (sit-and-reach, goniometry)
  - strength (manual muscle testing, hand or leg dynamometer, bench press or leg press, push-ups, pull-ups, or sit-ups)
  - endurance (12-minute run, 1.5-mile run)
  - power (vertical jump, standing broad jump)
  - speed (40-yard dash), agility (agility run)

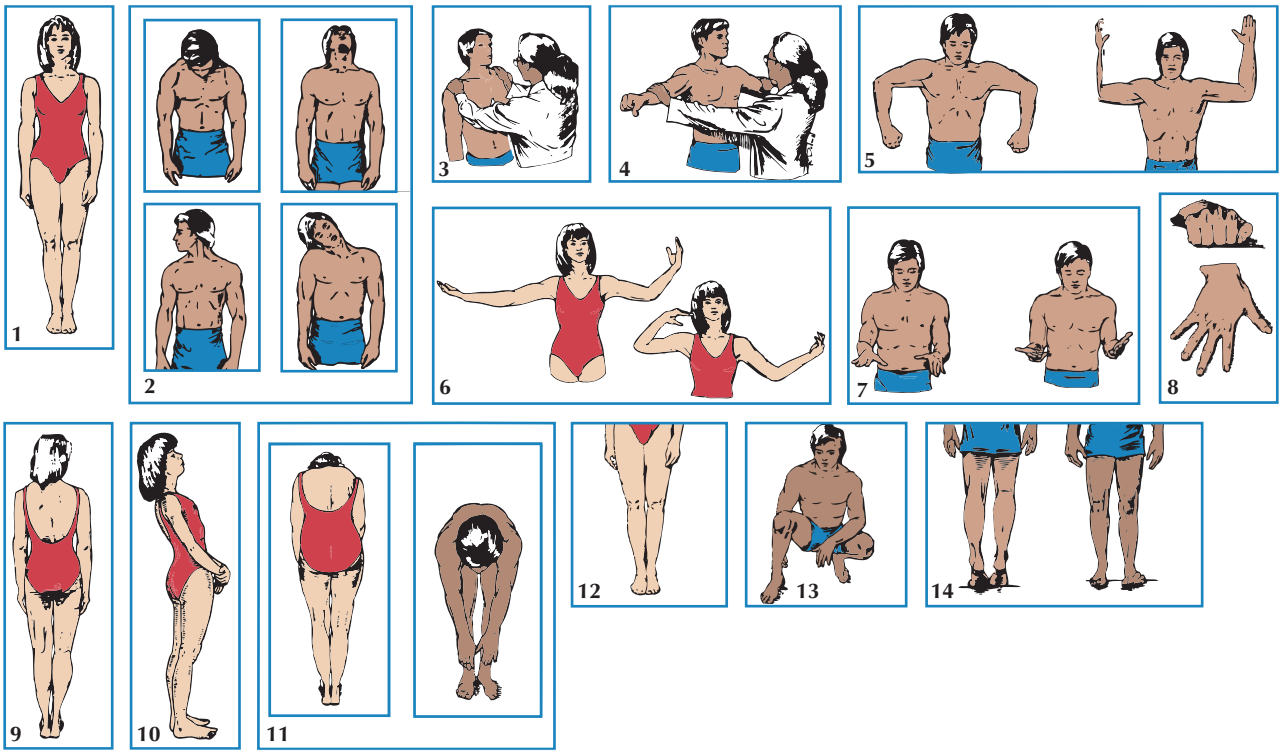


Figure 3-6 General musculoskeletal screening examination. Examination consists of the following: (1) inspection, athlete standing, facing examiner (symmetry of trunk, upper extremities); (2) forward flexion, extension, rotation, lateral flexion of neck (range of motion, cervical spine); (3) resisted shoulder shrug (strength, trapezius); (4) resisted shoulder abduction (strength, deltoid); (5) internal and external rotation of shoulder (range of motion, glenohumeral joint); (6) extension and flexion of elbow (range of motion, elbow); (7) pronation and supination of elbow (range of motion, elbow and wrist); (8) clench fist, then spread fingers (range of motion, hand and fingers); (9) inspection, athlete facing away from examiner (symmetry of trunk, upper extremities); (10) back extension, knees straight (spondylolysis/spondylolisthesis); (11) back flexion with knees straight, facing toward and away from examiner (range of motion, thoracic and lumbosacral spine; spine curvature; hamstring flexibility); (12) inspection of lower extremities, contraction of quadriceps muscles (alignment, symmetry); (13) "duck walk" four steps (motion of hip, knee, and ankle; strength, balance); (14) standing on toes, then on heels (symmetry, calf; strength; balance). *(© Rebekah Dodson.)*

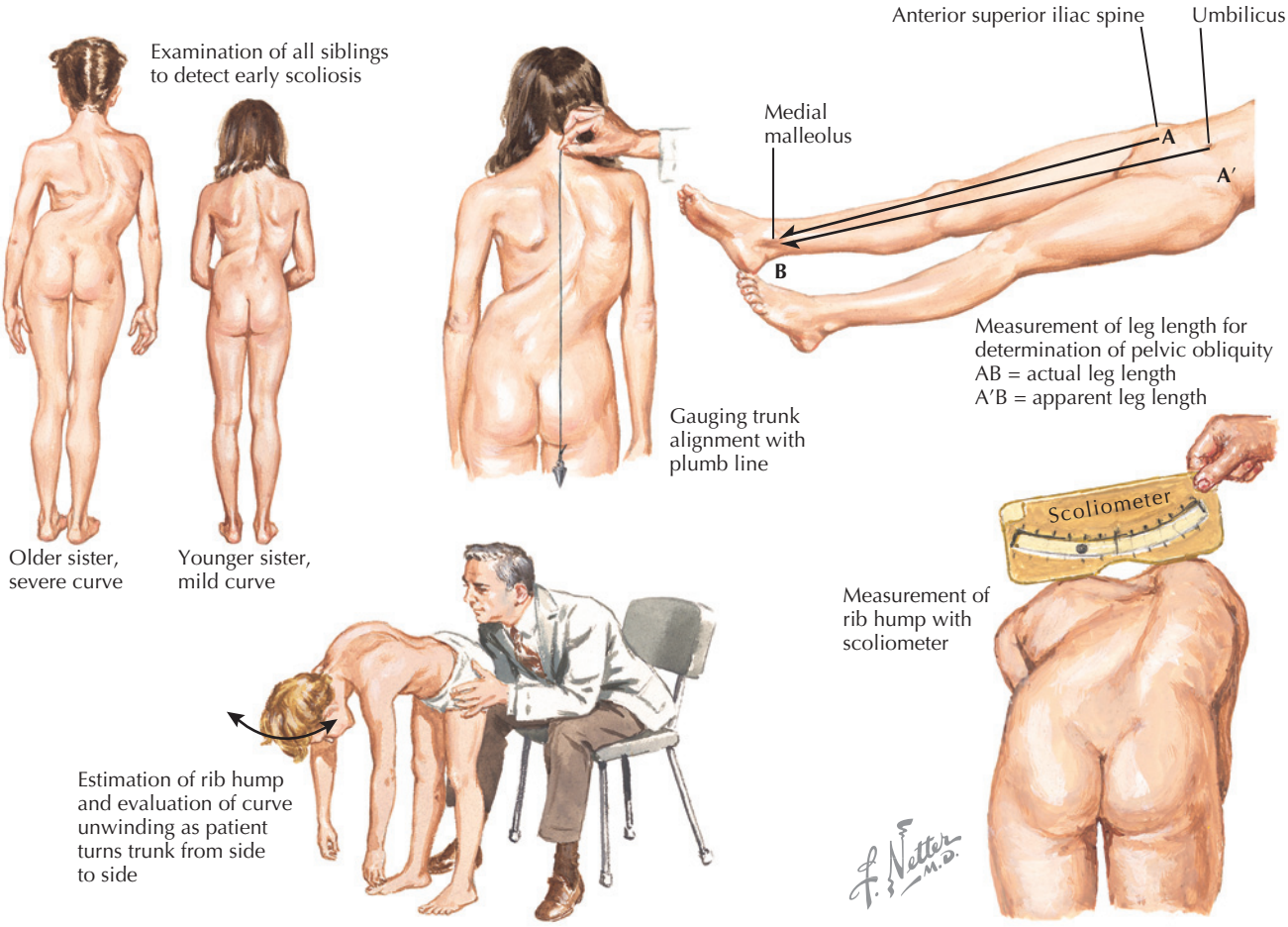


Figure 3-7 Clinical Evaluation of Scoliosis.

- balance (stork stand, balance beam walking)
- Vision performance testing has been added in some settings:
  - dynamic visual acuity
  - depth perception
  - visual tracking or pursuit
  - eye-hand and eye-body coordination

SCREENING TESTS

Differentiate Between Routine Screening and Diagnostic Testing

A complete blood count in an asymptomatic female athlete is a screening test, whereas a complete blood count in a female athlete with poor eating habits, heavy menstrual periods, fatigue, and pale conjunctiva becomes a diagnostic test.

- Does the burden of suffering resulting from the condition warrant screening?
- If the answer is "yes," ask the following:
  - What is the sensitivity of the proposed screening test?
  - Are the potential risks and cost of the test acceptable?
  - If the screening test identifies the condition, are proven and acceptable treatments available? Is there a clear advantage to initiating such treatment during the asymptomatic phase of the condition?

Routine Screening Tests

Routine screening not recommended, but many screening tests have been proposed and used in various settings, including the following:

Laboratory tests: urinalysis, complete blood count, chemistry profile, lipid profile, ferritin, sickle cell trait, HIV, infectious hepatitis, urine drug screening.

Cardiopulmonary tests: ECG, echocardiogram, exercise stress test, spirometry, exercise spirometry, other exercise challenge tests. (The American Heart Association and PPPE panel do not recommend ECG or echocardiogram for screening purposes.)

Radiographs: chest, cervical spine, and joint x-rays.

The "Italian Experience"

In Italy, a systematic, state-subsidized national program for mandatory annual PPPE of all athletes 12 to 35 years of age has been in place for about 30 years. Minimum annual tests include a general exam, and a 12-lead ECG. Elite competitive athletes receive a more comprehensive medical and physiologic evaluation that includes routine echocardiography; findings are as follows:

- Detection of few definitive examples of potentially lethal cardiovascular abnormalities remains the norm.
- 2.2% to 2.5% of athletes are disqualified (about 51% because of cardiovascular abnormalities).
- A study by Basso and colleagues suggests that the Italian national screening program can decrease the incidence of SCD among young athletes. During the study period, SCD occurred in 55 screened athletes versus 265 in nonscreened nonathletes.
- Right ventricular dysplasia causes more athletic deaths than HCM. Reasons for the discrepancy with North American data (where HCM causes more deaths in those less than 30 years old) are unresolved. Possible disqualification of athletes with screening may contribute to discrepancy.

With the rarity of potentially lethal cardiovascular abnormalities in young athletes and the overwhelming number of sports and athletic participants in the United States, screening of the Italian magnitude would be impractical in most settings.

ESC and IOC

In 2004-2005, the European Society of Cardiology (ESC) and International Olympic Committee (IOC) recommended combining noninvasive testing (e.g., a 12-lead ECG) with the standard history taking and physical examination for cardiovascular screening in large populations of young trained athletes.

ECG and/or Echocardiogram

ECG and/or echocardiogram should be considered in athletes with any significant cardiac symptoms or abnormal findings on exam or with a family history of sudden death (unknown cause), SCD, or other cardiac condition known to predispose to SCD (e.g., right ventricular dysplasia, HCM, long QT syndrome, Marfan syndrome) in a family member less than 50 years old, especially a first-degree relative. Ninety percent of people with HCM have abnormal ECG.

Baseline Neuropsychological Testing

Baseline neuropsychological testing may be considered and recommended, if available, for athletes in sports considered to present risk for head injury (see Chapter 39, Head Injuries). If a neuropsychological test battery is used in head injury evaluation, baseline data should be obtained before the season starts.

Down Syndrome

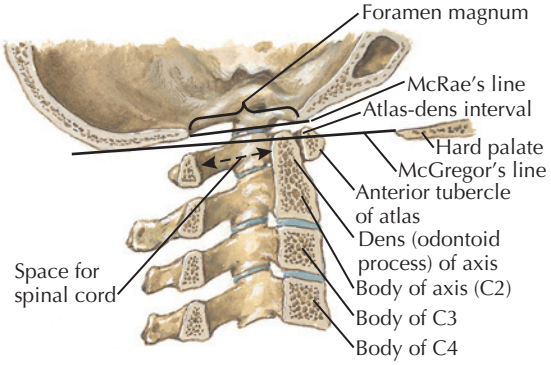
Patients with Down syndrome and their parents should be questioned closely about signs or symptoms of atlantoaxial instability. Cervical radiographs, including flexion and extension views, may be considered (Fig. 3-8).

- In asymptomatic patients, neurologic signs or symptoms may be more predictive of risk for injury progression than radiographic abnormalities.
- The American Academy of Pediatrics acknowledges potential but unproven value of lateral plain films of cervical spine but does not recommend routine screening x-rays.
- Special Olympics requires cervical spine radiographs before athletic participation in all patients with Down syndrome participating in judo, equestrian sports, gymnastics, diving, pentathlon, butterfly stroke, and diving starts in swimming, high jump, Alpine skiing, snowboarding, squat lift, and soccer.
- General agreement on criteria for exclusion from sport is lacking.
- For more information related to the athlete with disability, see Chapter 12, The Physically Challenged Athlete.

CLEARANCE FOR PARTICIPATION

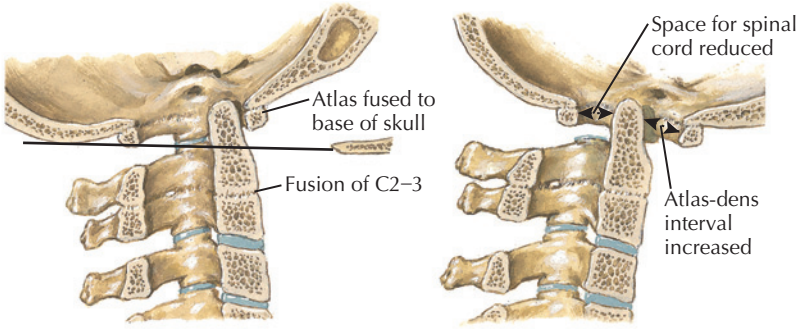
- Clearance falls into **four categories:**
  1. Full participation without restrictions
  2. Participation pending further testing/evaluation
  3. Participation just in certain sports
  4. Disqualification

Normal relationships of occipitocervical junction



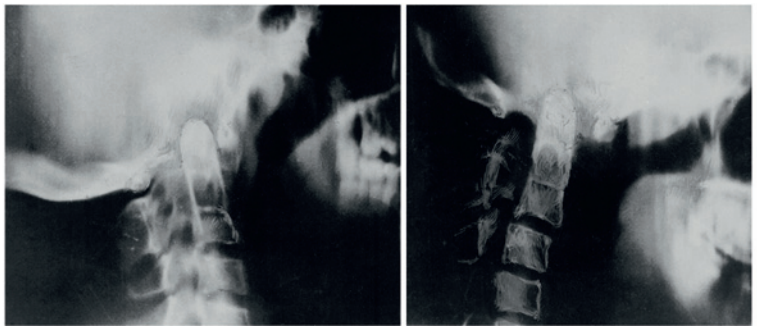
Normal position of tip of dens of axis above McGregor's line: mean = 1.32 mm, SD ± 2.6 mm. Atlas-dens interval is small, leaving adequate space posteriorly for spinal cord.

Occipitalization of atlas with instability of C1–2 (atlantooccipital fusion)



Atlas fused to base of skull. Dens projects into foramen magnum well above McGregor's line. 70% of patients with occipitalization of atlas and fusion of C2–3 develop C1–2 instability.

When neck is flexed, space available for spinal cord may be considerably reduced as atlas-dens interval increases. Fusion of C2–3 accentuates instability.



Lateral radiographs in extension (left) and flexion (right) of patient with occipitalization of atlas and hypermobile dens extending well into foramen magnum (basilar impression).

Figure 3-8 Congenital Anomalies of Occipitocervical Junction.

- Differentiation of categories is important.
- Familiarity with demands of specific sport is essential; use of classification system for sports by contact and strenuousness is helpful in this regard (Tables 3-3 and 3-4).
- **Published guidelines for medical conditions and sports participation** are helpful, but clinical judgment should be used in applying general guidelines to individual athletes (Table 3-5).
- Additional considerations:
  - How does condition/illness affect the athlete's risk of morbidity or mortality?
  - How will condition/illness affect other participants?
  - Are there limitations or modifications within the sport that allow the athlete to continue participation despite injury or illness? If so, is it reasonable to allow participation with limitations until condition resolves?
- Most athletes are cleared for full participation without restriction or with minimal additional evaluation (e.g., reassessment of visual acuity, blood pressure, or ligamentous instability; correction of improper rehabilitation; further musculoskeletal consultation).
- Explain reasons for further evaluation to athlete and parent/guardian (if athlete is less than 18 years old).
- Extensive updated information about medical condition and risk for participation often requires attention to specifics of the medical problem and the individual athlete as well as a breakdown of sport-specific requirements.
- Decisions to disqualify may require additional specialist consultations, as well as one or two "second expert opinions." A total of three opinions are suggested.

AREAS OF CONCERN

Medicolegal Issues

Right to participate:

- Under enactments such as the Americans with Disabilities Act and the Federal Rehabilitation Act, the athlete may have a legal right to participate against medical advice.
- If athletes choose to participate against medical advice, **an exculpatory waiver** or **prospective release** is highly recommended. Despite questions of validity, these forms of written contracts are intended to demonstrate that the athlete was fully informed of his or her condition and potential risk of participation, thus releasing the physician from liability in the event of morbidity or mortality caused by participation against medical advice.

"Good Samaritan" statutes: some states have made an effort to protect volunteer examiners (PPPE) under "Good Samaritan" statutes.

Sexual harassment:

- Athlete expectations, lack of privacy during exams, and inappropriate examinations (e.g., breast or gynecologic exam in nonprivate setting) may contribute to allegations.
- Clear communication with athletes, respect for their privacy, and common sense during examinations minimize potential problems.

Confidentiality

- Free flow of information occurs in most sports settings.
- Good communication with other health care providers, parents, athletic trainers, and coaches is important, but it must take place with respect for the athlete's confidentiality.

Table 3-3 CLASSIFICATION OF SPORTS BY CONTACT

Contact/Collision	Limited contact	Noncontact
Basketball	Baseball	Archery
Boxing*	Bicycling	Badminton
Diving	Cheerleading	Body building
Field hockey	Canoeing/kayaking (white water)	Canoeing/kayaking (flat water)
Football, tackle	Fencing	Crew/rowing
Ice hockey	Field events (high jump and pole vault)	Curling
Lacrosse	Floor hockey	Dancing (ballet, modern, and jazz)
Martial arts	Gymnastics	Field events (discus, javelin, shot put)
Rodeo	Handball	Golf
Rugby	Horseback riding	Orienteering
Ski jumping	Racquetball	Power lifting
Soccer	Skating (ice, in-line, and roller)	Race walking
Team handball	Skiing (cross-country, downhill, and water)	Riflery
Water polo	Skateboarding	Rope jumping
Wrestling	Snowboarding	Running
	Softball	Sailing
	Squash	Scuba diving
	Ultimate Frisbee	Swimming
	Volleyball	Table tennis
	Windsurfing/surfing	Tennis
		Track
		Weight lifting

*Participation not recommended by the AAP. The AAFP, AMSSM, AOASM, and AOSSM have no stand against boxing.

From American Academy of Pediatrics Committee on Sports Medicine and Fitness: Medical conditions affecting sports participation. Pediatrics 107(5):1205-1209, 2001.

Table 3-4 CLASSIFICATION OF SPORTS BY STRENUOUSNESS

High-to-moderate dynamic and static demands	High-to-moderate dynamic and low static demands	High-to-moderate static and low dynamic demands
Boxing*	Badminton	Archery
Crew/rowing	Baseball	Auto racing
Cross-country skiing	Basketball	Diving
Cycling	Field hockey	Equestrian
Downhill skiing	Lacrosse	Field events (jumping)
Fencing	Orienteering	Field events (throwing)
Football	Ping-pong	Gymnastics
Ice hockey	Race walking	Karate or judo
Rugby	Racquetball	Motorcycling
Running (sprint)	Soccer	Rodeoing
Speed skating	Squash	Sailing
Water polo	Swimming	Ski jumping
Wrestling	Tennis	Water skiing
	Volleyball	Weight-lifting
Low Intensity (Low Dynamic and Low Static Demands)		
Bowling	Curling	Riflery
Cricket	Golf	

*Participation not recommended by the AAP. The AAFP, AMSSM, AOASM, and AOSSM have no stand against boxing.

From American Academy of Pediatrics Committee on Sports Medicine and Fitness: Medical conditions affecting sports participation. Pediatrics 107(5):1205-1209, 2001.

Table 3-5 MEDICAL CONDITIONS AND SPORTS PARTICIPATION

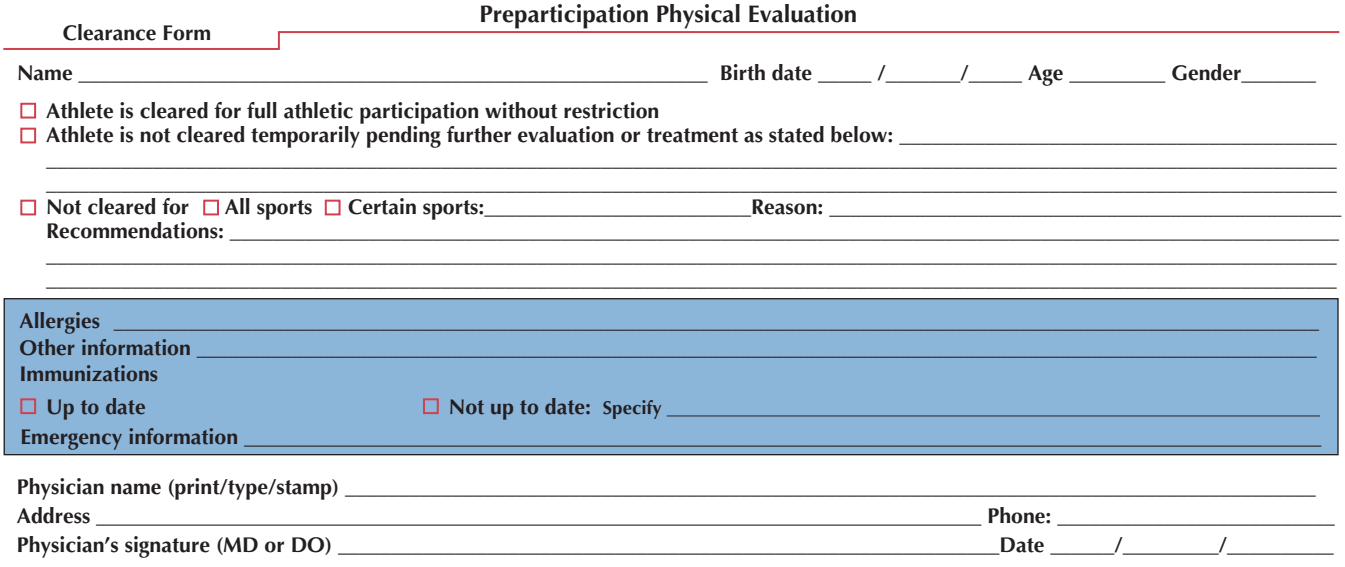
Condition	May participate
Atlantoaxial instability (instability of joint between cervical vertebrae 1 and 2) *Explanation:* Athlete needs evaluation to assess risk of spinal cord injury during sports participation.	Qualified yes
Bleeding disorder *Explanation:* Athlete needs evaluation.	Qualified yes
Cardiovascular diseases	No
Carditis (inflammation of the heart) *Explanation:* Carditis may result in sudden death with exertion.	
Hypertension (high blood pressure) *Explanation:* Those with significant essential (unexplained) hypertension should avoid weight and power lifting, body building, and strength training. Those with secondary hypertension (hypertension caused by a previously identified disease), or severe essential hypertension, need evaluation.	Qualified yes
Congenital heart disease (structural heart defects present at birth) *Explanation:* Those with mild forms may participate fully; those with moderate or severe forms, or who have undergone surgery, need evaluation.	Qualified yes
Dysrhythmia (irregular heart rhythm) *Explanation:* Athlete needs evaluation because some types require therapy or make certain sports dangerous, or both.	Qualified yes
Mitral valve prolapse (abnormal heart valve) *Explanation:* Those with symptoms (chest pain, symptoms of possible dysrhythmia) or evidence of mitral regurgitation (leaking) on physical examination need evaluation. All others may participate fully.	Qualified yes
Heart murmur *Explanation:* If the murmur is innocent (does not indicate heart disease), full participation is permitted. Otherwise the athlete needs evaluation (see congenital heart disease and mitral valve prolapse above).	Qualified yes
Cerebral palsy *Explanation:* Athlete needs evaluation.	Qualified yes
Diabetes mellitus *Explanation:* All sports can be played with proper attention to diet, blood glucose concentration, hydration, and insulin therapy. Blood glucose concentration should be monitored every 30 minutes during continuous exercise and 15 minutes after completion of exercise.	Yes
Diarrhea *Explanation:* Unless disease is mild, no participation is permitted, because diarrhea may increase the risk of dehydration and heat illness. See fever below.	Qualified no
Eating disorders Anorexia nervosa Bulimia nervosa *Explanation:* These patients need both medical and psychiatric assessment before participation.	Qualified yes
Eyes Functionally one-eyed athlete, loss of an eye, detached retina, previous eye surgery, or serious eye injury *Explanation:* A functionally one-eyed athlete has a best corrected visual acuity of <20/40 in the worse eye. These athletes would suffer significant disability if the better eye was seriously injured as would those with loss of an eye. Some athletes who have previously undergone eye surgery or had a serious eye injury may have an increased risk of injury because of weakened eye tissue. Availability of eye guards approved by the American Society for Testing Materials (ASTM) and other protective equipment may allow participation in most sports, but this must be judged on an individual basis.	Qualified yes
Fever *Explanation:* Fever can increase cardiopulmonary effort, reduce maximum exercise capacity, make heat illness more likely, and increase orthostatic hypotension during exercise. Fever may rarely accompany myocarditis or other infections that may make exercise dangerous.	No
Heat illness, history of *Explanation:* Because of the increased likelihood of recurrence, the athlete needs individual assessment to determine the presence of predisposing conditions and to arrange a prevention strategy.	Qualified yes
Hepatitis and HIV infection *Explanation:* Because of the apparent minimal risk to others, all sports may be played that the state of health allows. In all athletes, skin lesions should be properly covered, and athletic personnel should use universal precautions when handling blood or body fluids with visible blood.	Yes
Kidney: absence of one *Explanation:* Athlete needs individual assessment for contact/collision and limited contact sports.	Qualified yes
Liver: enlarged *Explanation:* If the liver is acutely enlarged, participation should be avoided because of risk of rupture. If the liver is chronically enlarged, individual assessment is needed before collision/contact or limited contact sports are played.	Qualified yes
Malignancy *Explanation:* Athlete needs individual assessment.	Qualified yes
Musculoskeletal disorders *Explanation:* Athlete needs individual assessment.	Qualified yes
Neurologic ***History of serious head or spine trauma,*** severe or repeated concussions, or craniotomy *Explanation:* Athlete needs individual assessment for collision/contact or limited contact sports, and also for noncontact sports if there are deficits in judgment or cognition. Research supports a conservative approach to management of concussion.	Qualified yes

From American Academy of Pediatrics Committee on Sports Medicine and Fitness: Medical conditions affecting sports participation. Pediatrics 107(5):1205-1209, 2001, with permission.

Table continued on following page

Table 3-5 MEDICAL CONDITIONS AND SPORTS PARTICIPATION—cont'd

Condition	May participate
Convulsive disorder, well controlled *Explanation:* Risk of convulsion during participation is minimal.	Yes
Convulsive disorder, poorly controlled *Explanation:* Athlete needs individual assessment for collision/contact or limited contact sports. Avoid the following noncontact sports: archery, riflery, swimming, weight or power lifting, strength training, or sports involving heights. In these sports, occurrence of a convulsion may be a risk to self or others.	Qualified yes
Obesity *Explanation:* Because of the risk of heat illness, obese persons need careful acclimatization and hydration.	Qualified yes
Organ transplant recipient *Explanation:* Athlete needs individual assessment.	Qualified yes
Ovary: absence of one *Explanation:* Risk of severe injury to the remaining ovary is minimal.	Yes
Respiratory	Qualified yes
Pulmonary compromise including cystic fibrosis *Explanation:* Athlete needs individual assessment, but generally all sports may be played if oxygenation remains satisfactory during a graded exercise test. Patients with cystic fibrosis need acclimatization and good hydration to reduce the risk of heat illness.	
Asthma *Explanation:* With proper medication and education, only athletes with the most severe asthma have to modify their participation.	Yes
Acute upper respiratory infection *Explanation:* Upper respiratory obstruction may affect pulmonary function. Athlete needs individual assessment for all but mild disease. See fever above.	Qualified yes
Sickle cell disease *Explanation:* Athlete needs individual assessment. In general, if status of the illness permits, all but high exertion, collision/contact sports may be played. Overheating, dehydration, and chilling must be avoided.	Qualified yes
Sickle cell trait *Explanation:* It is unlikely that individuals with sickle cell trait (AS) have an increased risk of sudden death or other medical problems during athletic participation except under the most extreme conditions of heat, humidity, and possibly increased altitude. These individuals, like all athletes, should be carefully conditioned, acclimatized, and hydrated to reduce any possible risk.	Yes
Skin: boils, herpes simplex, impetigo, scabies, molluscum contagiosum *Explanation:* While the patient is contagious, participation in gymnastics with mats, martial arts, wrestling, or other contact/collision or limited contact sports is not allowed.	Qualified yes
Spleen, enlarged *Explanation:* Patients with acutely enlarged spleens should avoid all sports because of risk of rupture. Those with chronically enlarged spleens need individual assessment before playing contact/collision or limited contact sports.	Qualified yes
Testicle: absent or undescended *Explanation:* Certain sports may require a protective cup.	Yes



Preparticipation Physical Evaluation

Clearance Form

Name _____ **Birth date** ____ /_____/____ **Age** _____ **Gender**_____

☐ **Athlete is cleared for full athletic participation without restriction**
☐ **Athlete is not cleared temporarily pending further evaluation or treatment as stated below:** _____

☐ **Not cleared for** ☐ **All sports** ☐ **Certain sports:**_____**Reason:** _____
Recommendations: _____

Allergies _____
Other information _____
Immunizations
☐ **Up to date** ☐ **Not up to date: Specify** _____
Emergency information _____

Physician name (print/type/stamp) _____
Address _____ **Phone:** _____
Physician's signature (MD or DO) _____ **Date** _____/_____/_____

Figure 3-9 Preparticipation physical evaluation clearance form. *(Adapted from Minnesota State High School League, 2009-2010 sports qualifying physical forms. Available at http://www.mshsl.org/mshsl/publications/code/forms/PhysicalExam.pdf. Accessed on April 26, 2009; and from Preparticipation Physical Evaluation, 3rd ed. Minneapolis: McGraw-Hill/Phys Sportsmed, 2005.)*

- The newest PPPE form includes multiple pages—clearance considerations and recommendations are available as pages separate from the detailed history and examination (Fig. 3-9).

Team Physician versus Personal Physician

- Team physicians may perform the PPPE but have minimal control over evaluation of specific problems uncovered in the PPPE, especially in high schools, smaller colleges, and managed care settings.
- Space is provided near end of the recommended PPPE form to write specific recommendations for further evaluation before clearance (see Fig. 3-9). Telephone communication is sometimes helpful. Clear communication between physicians is essential.
- Team physicians must keep the "big picture" in mind, regardless of level of participation.

Communication

- The team physician's **primary responsibility is to the athlete and his or her parent/guardian if the athlete is younger than 18 years.** Secondary responsibility is to the university, school, or organization. This distinction is often critical at the professional level. It is important at times to explain primary and secondary responsibility to coaching staff and administration in terms of sharing information. Emphasis must be placed on concern for long-term safety and health.
- The Team physician must respect the athlete's confidentiality amid free flow of information in many sport settings. Explain to the athlete the need to inform the coach if the athlete is unable to participate in practice or play. Communication with athletic trainers, administrators, parents, and, at times, the press is important. First discuss with the athlete what information needs to be disclosed.
- The team physician can arrange for follow-up communication. Have a plan for follow-up care, and establish it in writing, if appropriate. Arrange for additional evaluation and final clearance once additional testing requirements are met. Arrange for further rehabilitation, functional testing, return to play with modifications, and progression as necessary. Inform the athlete of risks and concerns for continued participation, and do not assume that the athlete will choose to participate. Follow-up care is essential.

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Sideline Preparedness and Emergencies on the Field

4

Aaron Rubin

PREPARATION

Training

- Many different health care professionals may cover sporting events. This chapter is directed to physician coverage of events.
- Physicians of any specialty may cover events ranging from high level (professional, Olympic, college) to informal games played by their children or in the neighborhood.
- The training for covering any event should be similar, but from a practical point, physicians with more experience cover the higher-level events.
- Nothing substitutes for getting sideline experience with an experienced sports medicine physician.
- There are few evaluations or procedures that will occur in the training room or on the sideline that are not done in the office setting. The difference is often the comfort level of the physician outside of his or her normal working environment in the office or hospital.

Pre-game

- Discuss the potential problems in advance with the athletic trainer.
- Consider weather, location, access to the venue (by you and emergency personnel), and where the athletes will be evaluated.
- Have a clear understanding as to your responsibilities. If there is no plan to care for spectators, the responsibility will likely fall on you. It would be difficult to ethically and legally defend a failure to act in case of a life-threatening injury or illness in the stands, at least until emergency personnel arrive at the scene.

Game Time

- Arrive to the game at least 30 minutes before game time.
- Meet the other members of the sports medicine team and discuss communications and response issues.
- Meet members of the opposing team. If you are the home team, make sure the opponent's team members know how to activate your emergency action plan. If you are the visiting team, make sure you know the same.
- Meet the paramedics and ambulance personnel. It's always easier to meet and discuss approach to response on the field before the problem occurs.
- Meet the officials for the game. The on-field officials are crucial in observing injuries and stopping the contest to allow the medical team to assess and treat injured athletes. The health and welfare of the athletes should always be of utmost importance, and the officials need to know who to turn to in an emergency.
- The sports medicine team should put even the most serious rivalries aside and assist in care to any injured athlete.
- Tradition holds that the home team offers care to the officials and other neutral attendees at the event. Discuss this prior to the event with appropriate medical staff.

THE SPORTS MEDICINE TEAM

Team Physician

The team physician should generally be the leader of the sports medicine team. At times, this leadership role is assigned to or shared with the athletic trainer (see Chapter 1).

Athletic Trainer

- The athletic trainer should be certified by the Board of Certification (BOC) for athletic trainers. For more information check the National Athletic Trainers Association (NATA) website. A certified athletic trainer is designated by the title ATC. Most, but not all, states have licensure or accreditation requirements for athletic trainers (see Chapter 2).
- The athletic trainer is the member of the sports medicine team that generally has the most contact with the athletes and will provide preventive services and rehabilitation of injury in addition to sideline management.

Emergency Medical Personnel

- Some events will have paramedics (PM) or emergency medical technicians (EMT) present. They are an essential part of the medical team, especially in times of a serious or catastrophic injury.
- These personnel specialize in stabilization and transportation of the ill and injured. They may not have as much experience with other treatments.
- Paramedics will frequently have supplies and training to carry out advanced cardiac life support. Some EMTs will have specialized training that allows them to perform some advanced procedures. Not all ambulance personnel have this equipment or expertise, and the team physician should know what is available at the venue.
- The emergency medical personnel will likely have advanced communication with the local communication center and hospital emergency base stations.
- In general, the emergency medical personnel should not intervene unless asked by the sports medicine team. The procedures for emergency response should be discussed and planned in advance of the event.
- Check your local regulations. Once the emergency medical system (EMS) is activated, they may have the legal obligation to carry out care according to their protocols. They may ask the physician to prove that he or she is licensed and have the physician sign a document stating that the physician will take medical control of the patient until the patient reaches a higher level (emergency department) of care.

Security/Police/Sheriff

- Security staff may be school employees, work for the venue or private security services, or be sworn peace officers (sheriff or police).
- They will be expected to provide crowd control and assure the safety of the medical providers.
- Sheriff or police officers may have the ability to communicate with dispatchers for EMS.

School Administration

Coach

- The coaching staff usually has the most contact with the athletes and their families.
- The coaching staff can be extremely helpful in calming family members, friends, and teammates in the case of a serious injury.
- The coach needs to know whether or not the athlete can continue in the competition, but specific medical information should be kept private unless permission from the athlete and

parents (if athlete is a minor) is obtained to maintain confidentiality.

Athletic Director

- The athletic director or venue director needs to be involved in the emergency action plan to assure that adequate medical care can be provided to the athlete.
- The athletic director must also consider the needs of the officials and spectators.

EQUIPMENT

Team Physician's Bag

Equipment carried by the team physician is dependent on many factors, including the following:

- Number of athletes
- Sporting event
- Equipment at the venue (athletic trainer, paramedics)
- Distance and transportation time from definitive medical care
- Expertise in using the equipment (don't carry equipment you are not trained to use)

Pocket Gear

The following ready-to-use supplies should be available:

- Gloves
- Gauze pads
- Airway and pocket barrier airway device
- Trauma scissors
- Flashlight
- Cell phone
- Pocket multi-tool

Emergency Equipment

Ambulance

An ambulance may or may not be present. If not, consideration must be given to whether or not there is a need at the event for the following equipment:

- Spine immobilization equipment, including spine board, rigid collars, sand bags, head bed, tape
- Traction splint for femur fracture
- Advanced cardiac life support equipment
  - Defibrillator, monitor
  - Advanced airway and oxygen delivery supplies
  - Medications
  - IV access, fluids
- Communication equipment

Athletic Trainer

- Preventive braces, tape, pads
- Splints
- Wound care
- Gloves
- Facemask removal equipment
- Basic life support supplies

Team physician

- Wound care (suture, suture kit, skin staples, wound adhesive)
- Medication (consider local medical and pharmacy laws regarding dispensing of medications, especially to minors)
  - Over-the-counter medications (acetaminophen, ibuprofen)
  - Asthma/anaphylaxis medications (albuterol inhaler with AeroChamber, epinephrine or EpiPen, antihistamines)
  - Wound care (wound cleaning, lidocaine 1%)
  - Gastrointestinal (antacid, loperamide, antinausea)
  - Antibiotics (more a consideration for traveling with a team to get treatment started early, not much indication for sideline management)
- Diagnostic equipment
  - Stethoscope
  - Blood pressure cuff
  - Oto-ophthalmoscope and eye tray
  - Glucose monitor, oxygen saturation monitor
- Head lamp (helpful in evaluating facial wounds and in suturing)

Dress Code

- Team shirt, jacket, or some other identification as member of the sports medicine team
- Comfortable appropriate shoes for the venue
- Appropriate clothing for the weather (cold, rain, etc.)

PRIORITIZATION OF ACUTE INJURY (ABCDEF)

Airway

- Obtain an airway with care to maintain spine precautions.
- Must be able to gain access to the player's face.
  - Must be able to remove facemask while maintaining spine in neutral position.
- Advanced airway devices may be considered if the user has been properly trained.
- Do not let insertion of an advanced device interfere with CPR (Fig. 4-1).

Breathing

- Mouth to mouth only if barrier device not available
- Bag-valve mask
- Demand valve

Circulation

- Start CPR if indicated.
- Remove the athlete's clothing and protective equipment if necessary to enable access for evaluation and CPR.

Disability and Defibrillation

- Always assume that the unconscious athlete also has a cervical spine injury (Fig. 4-2).
- Maintain control of the spine throughout evaluation and treatment.
- Early defibrillation of cardiac arrest using an automated defibrillator or fully functioning defibrillator is essential in treating ventricular fibrillation.

Exposure and Evaluation

- Once immediate life-threatening conditions are managed, a complete evaluation must be performed.
- Expose the injured area to fully evaluate the site of injury. Pads, braces, tape, and interfering clothing must be removed with care to maintain the privacy of the athlete.

Final Disposition

- Appropriate follow-up care instructions must be provided.
- Discuss the care of injuries and warnings about late effect of injury with parents or other appropriate adults. This is especially true for any athlete released with a head injury.

DECISION PROCESS

Emergency

- The first decision is whether or not the injury requires activation of the emergency action plan for the venue:
  - Is this a life- or limb-threatening emergency to an individual?
  - Is this a situation with multiple individuals injured or potential for multiple casualties?

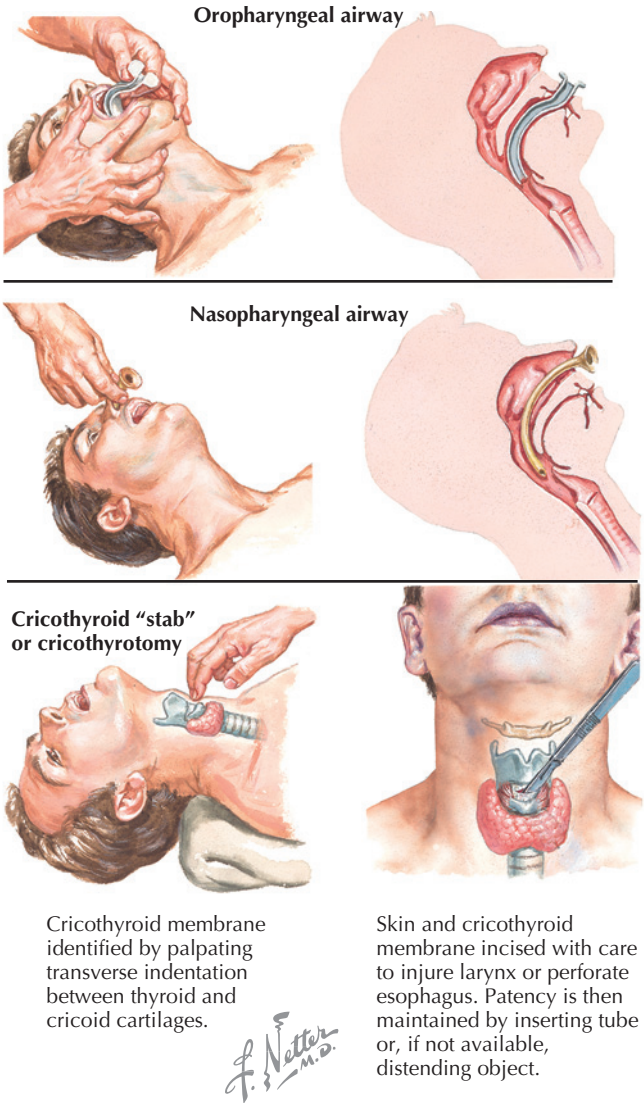

Figure 4-1 Patent Airways.

- There are often inherent delays in getting emergency response to sporting venues (traffic patterns, security issues, venue access), and an early activation means an early response.
- Keep in mind, however, that it is difficult to cancel an EMS response once 911 has been called.

Urgency

Is this an urgent issue that requires immediate care but does not demand immediate outside resources?

Transportation

- Will the injured athlete require transportation to the hospital?
- Can the injured athlete safely go by private vehicle or is an ambulance needed?
- Does the injured athlete need transportation back to the school or to home? Is it safe for him or her to drive?

Return to Play

Often the most difficult decision for the team physician is whether or not the athlete can return to play. Consider the following:

Figure 4-2 Skull traction. In-line traction to maintain head and neck in neutral position.

- Is there risk to worsen the injury? Is this a reasonable risk for a worsened minor injury or is there concern for a major injury?
- Is there risk for other injury?
- Is there risk to other players if the athlete continues?

SPECIFIC CONDITIONS AND INJURIES

Cardiopulmonary Arrest

The sports medicine team should be trained in CPR.

- Check responsiveness. If unresponsive, activate emergency action plan (EAP) for the venue.
- See Airway, Breathing, Circulation, and Defibrillation on p. 25.
- Check **airway.** If the patient is not breathing, open airway while maintaining spine precautions by chin-lift or jaw-thrust maneuvers. Remove any foreign bodies (check for mouth guard, missing teeth, vomitus).
  - Consider inserting airway, airway device (laryngeal-mask airway [LMA] or Combitube), or tracheal intubation. Do not delay resuscitation for insertion of airway device if adequate air exchange is occurring.
  - If there is laryngeal damage, you may need to provide a surgical airway by inserting a needle cricothyroidotomy through the cricothyroid membrane. The device should be at least 14-gauge size.
- Look, listen, and feel for **breathing.** If the patient is not breathing once the airway is open, breathe for the patient using an appropriate barrier device.
- Check pulse for **circulation.** If there is no pulse, begin chest compressions.
- If cardiac arrest has occurred, early **defibrillation** is necessary. Retrieve and apply automated external defibrillator (AED). Follow directions of the AED. Continue CPR while awaiting EMS and transportation to appropriate medical facility.

Anaphylaxis

- Check **airway.** Administer oxygen.
- For adults, administer epinephrine 0.3 to 0.5 mg (0.3 to 0.5mL of 1:1000 dilution) subcutaneous (SC) or intramuscular (IM) or via adult EpiPen (provides 0.3 mg). Repeat every 15 minutes.
- For children, administer epinephrine 0.01 mg/kg (up to 30 kg) IM or SC or via EpiPen Jr. (provides 0.15 mg).

- Transport to appropriate medical facility for definitive care and observation.

Asthma

- Check **airway.** Administer oxygen.
- Administer albuterol via inhaler (two puffs via AeroChamber).
- Monitor for improvement. Transport if not improving rapidly.

Head

- Much has been written about evaluation and treatment of concussion (see Chapter 39).
- Athletes do not die from concussions; they die as a result of other head injuries, and the goal on the sidelines is to rapidly exclude the possibility of these other injuries.
- The severity of any closed head injury is often determined in retrospect. It is difficult, if not impossible, to determine the disability caused by head trauma until sufficient time has passed.
- The primary evaluation of head injury during a sporting event is to determine if a potentially more serious (subdural hematoma, epidural hematoma, intracranial contusion, elevated intracranial pressure) head injury has occurred and begin treatment.
- Any loss of consciousness, confusion, neurologic abnormalities on physical examination, vomiting, emotional changes, or other abnormalities should warrant close evaluation and observation. Any deterioration of mental status discovered on neurologic exam warrants transport to appropriate facility for definitive evaluation and management.
- In general, athletes who have sustained a head injury should not return to play in the same contest. This is controversial and is further discussed in Chapter 39.
- Grading of head injury (not concussion) may be determined by use of the Glasgow Coma Scale (Fig. 4-3). An easier scale is the **AVPU** scale (**a**wake, responds to **v**erbal, responds to **p**ain, **u**nresponsive).

Facial Injuries

- Assess for associated injuries. Facial injuries are often bloody, and the team physician should not be distracted from evaluation for airway, head, and cervical spine injury.
- Inspect the area of injury, and control bleeding with direct pressure.
- Palpate bony prominences for crepitus or step-off.
- Check range of motion of mandible and extraocular movements. Ask the athlete about diplopia, which could indicate entrapment of extraocular muscles and an orbital fracture.
- If there is a nose bleed, palpate bony area for crepitus. Look at alignment of nose. Check for septal hematoma. If a septal hematoma is present, arrange for definitive treatment.
  - To stop bleeding, apply direct pressure by squeezing nostrils together for 5 minutes. Placing an ice bag on the bridge of the nose may help decrease swelling. Placing an ice bag on the back of the neck may decrease bleeding by triggering a reflex mechanism.
  - If bleeding continues, consider transport to appropriate facility for further evaluation and possible nasal packing.
- Check for cerebrospinal fluid (CSF) mixed in blood by putting a drop of the blood on a gauze pad. If a ring of clear fluid is seen around the blood, consider the possibility of a CSF leak and a cribriform plate fracture.

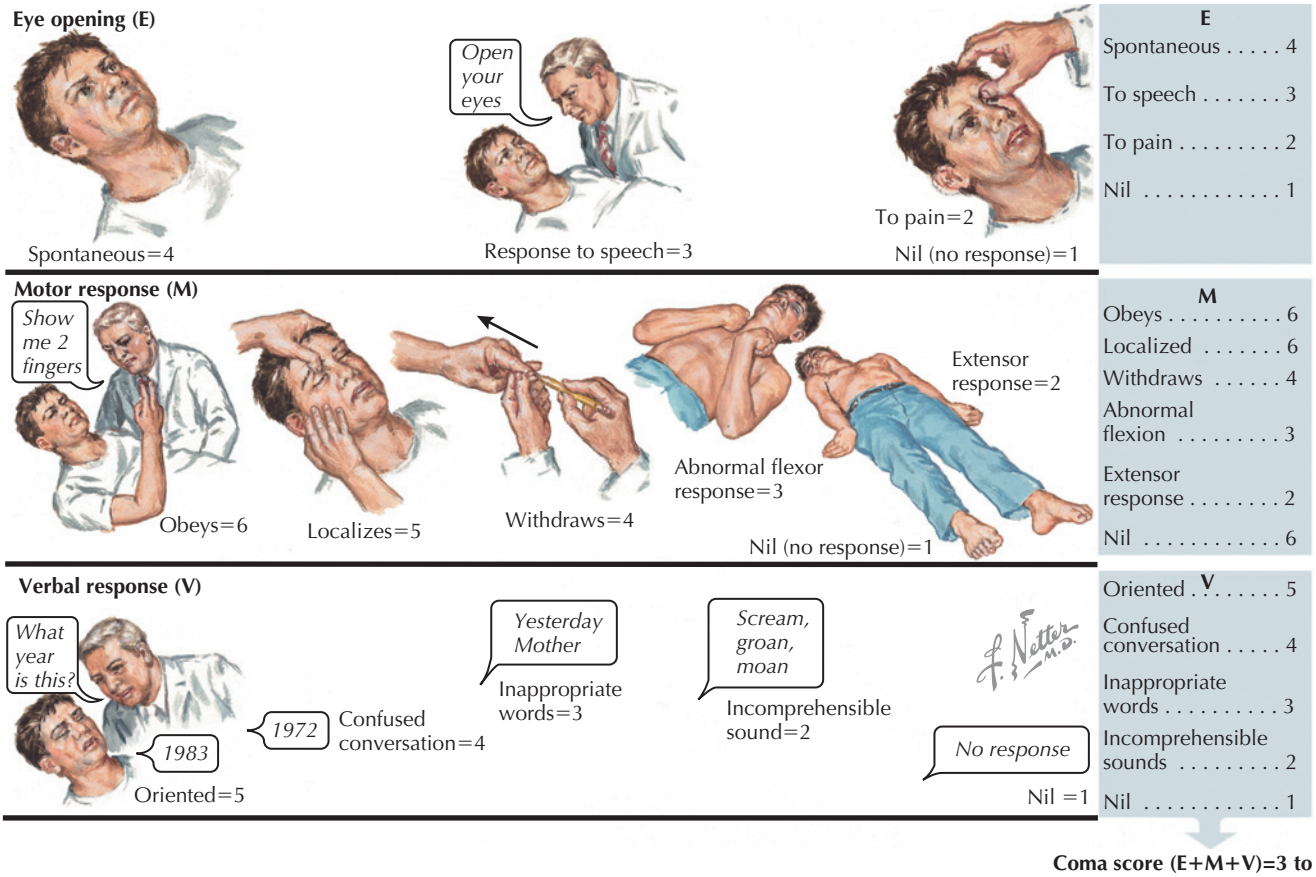

Figure 4-3 Glasgow Coma Scale.

Dental

- Many dental injuries can be prevented by proper use of well-fitted mouth guards. The team physician should make certain that the players are wearing their mouth guards. To keep them functional and prevent them from obstructing the airway if an athlete were to lose consciousness, the mouth guards should not be modified by cutting them down.
- A fractured tooth can involve just the enamel (Type I), be down to the dentin (Type II), or go all the way down to the pulp (Type III).
  - Type I fractures will generally not result in loss of the tooth; the major result would be cosmetic.
  - Type II fractures are generally uncomplicated by tooth loss, but they are likely to be more painful and may be difficult to distinguish from a Type III.
  - Type III fractures are usually very painful and the athlete should not continue participation. The athlete should be seen by a dentist within 3 hours to have best chance of saving the tooth.
- There are three basic types of tooth luxation:
  - Extruded, where the tooth appears longer than the adjacent teeth. May reposition by gently pushing the tooth back into place.
  - Laterally displaced, where the tooth is anterior or posterior to the rest of the teeth. May also reposition by gently aligning the tooth with the rest of the teeth. Both extruded and displaced teeth need to be braced to allow healing and should be seen by a dentist as soon as possible. If unable to reposition or if severe pain is present, a dentist should be seen soon because there is likely root damage.
  - Intruded tooth, where the tooth appears shorter than the rest of the row. Do not reposition; have the athlete see a dentist within 48 hours.
- A missing tooth, or avulsion, needs to be found immediately. If not found on the ground or on the athlete's clothing, one must consider the possibility that the tooth has been swallowed or aspirated. An avulsed tooth should be carefully handled, by the crown only. The crown should be gently cleaned with saline or clean water (not scrubbed), and replaced in the socket if possible.
  - If unable to replace immediately, store the tooth in saline, milk, or specially made "tooth saver" solutions.
  - Poor outcomes result if the tooth is not replaced and stabilized within 2 hours.

Eye

- Eye injuries can range from minor irritation from dirt to a ruptured globe and permanent loss of vision.
- Accurate history regarding mechanism of injury is very helpful when determining treatment.
- Loss of vision, loss of visual field, persistent blurring of vision, and double vision are all signs of a serious eye injury. It is, therefore, important to check visual acuity on any eye-injured athlete. Check by having the athlete read a program or lettering on an item in your medical bag if a standard visual acuity card is not available. Check extraocular movements and papillary response (Fig. 4-4).
- Other signs of serious injury include a deep throbbing or stabbing pain, abnormal protrusion, abnormal pupil shape, poor reactivity of pupil, laceration of the globe. These should prompt immediate transport to appropriate medical facility with the athlete sitting upright and protective shielding of the affected eye.
- Corneal irritation from a foreign body can be rinsed out and carefully evaluated for other injury. If no signs of a more serious injury, the athlete can return to the contest. The athlete should be re-evaluated at the contest's end.

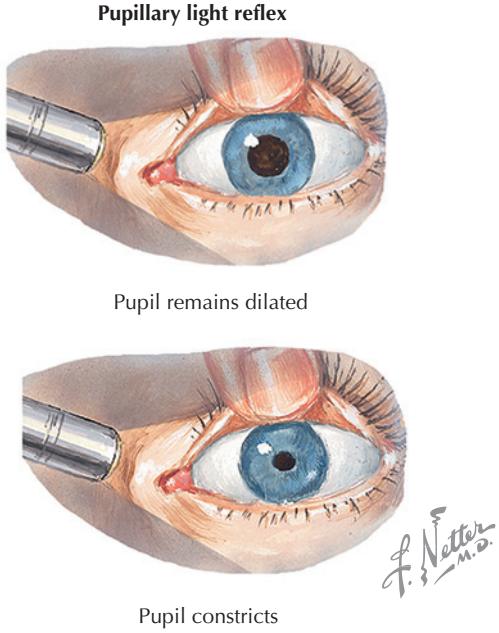

Figure 4-4 Assessing Pupillary Size and Reaction.

Neck

- Cervical spine injury is a major cause of disability in the athlete.
- Careful management of a suspected cervical spine injury can lessen this disability in some cases.
- The sports medicine team should practice management of the suspected cervical-spine injured athlete and have appropriate supplies to access the airway while maintaining cervical spine stability.
- The decision to remove the athlete from the field of play after a neck injury may be difficult. Any neurologic symptoms, bony tenderness, or distracting injury should prompt the medical team to put the athlete into full spinal precautions before moving him or her. Distracting injuries include head injury, which may make the athlete unaware of neurologic symptoms, or extremity injury, which may "distract" the athlete and medical team from doing a thorough evaluation.

Chest

- Chest trauma may result from a blunt or direct blow, a crush or squeeze injury, or rapid deceleration trauma. In athletics, chest trauma is rarely caused by penetrating trauma, although there have been some cases of javelin injuries recorded at track and field events.
- The injuries may involve the chest wall (muscle, rib cage, sternum), the lung, or the heart and great vessels.
- Rib cage injuries may be difficult to assess at the venue. Any crepitus, pain with compression of the chest, or obvious deformity of the rib cage should prompt a more thorough evaluation before allowing an athlete to return to play.
- Upper rib injuries are associated with vascular and brachial plexus injury, middle ribs with lung injury, and lower rib injury with renal and hepatic injury.
- Pulmonary injury includes pulmonary contusion and pneumothorax. Shortness of breath, hemoptysis, or decreased breath sounds should lead to further evaluation. If a tension pneumothorax is suspected, 100% oxygen should be administered and needle decompression should be performed immediately.

- Needle decompression is performed by inserting a large bore needle (14 gauge or 16 gauge) into the second intercostals space on the affected side. The needle should be inserted just above the third rib at the midclavicular line. There should be a release of air once the area is entered.
- The athlete then needs to be transported to an emergency department for definitive treatment.

- Cardiac muscle injury during sporting events is rare. High impact cardiac injuries behave much like a myocardial infarction.
- Commotio cordis may lead to ventricular fibrillation and sudden cardiac death. Treatment involves recognition, CPR, and early defibrillation.
- Great vessel injuries require rapid recognition and transportation to a medical center with the capacity to perform thoracic surgical intervention.

Abdomen

- Severe abdominal trauma, especially penetrating injury, is rare in sporting events.
- Hepatic and splenic trauma account for most internal injuries.
- Abdominal wall contusions may be difficult to distinguish from internal injury. Repeat examination is helpful.
- Athletes with abdominal injuries should be given nothing orally, in case a surgical intervention becomes necessary.
- The athlete should be transported to an emergency department with IV access.

Genitourinary

- Renal trauma, while uncommon, can occur from direct blows to the flank or back.
  - Most cases will be minor renal injury such as a contusion.
  - Major renal trauma includes kidney laceration, fracture, or damage to the renal vessels or proximal ureter.
  - Hematuria, lower rib fracture, and Grey-Turners sign (flank ecchymosis) should raise suspicion of renal injury.
  - All suspected renal trauma deserves immediate evaluation in the emergency department.
- Scrotal trauma needs to be carefully, privately evaluated.
  - Scrotal lacerations are rare as a sports injury.
  - Careful evaluation needs to occur to rule out testicular dislocation, torsion, or rupture.
  - Testicular trauma must be evaluated soon to save the testicle.
- Penile injuries are rare in athletic events.
- "Straddle" injuries can occur in male and female athletes. Blood at the meatus suggests injury and should be followed up with urologic evaluation.

Skin

- Acute skin injuries are common in sports and include contusions, lacerations, abrasions, burns, and cold injury. Infection is a later complication and can be prevented by good care of broken skin.
- Always examine the structure beneath the skin. Make sure lacerations do not extend to bone, tendon, or into joints.
- Abrasions should be cleaned and covered, with bleeding controlled, before the athlete is allowed to return to play. More thorough evaluation and treatment should be carried out at contest end.
- Lacerations should be carefully evaluated, cleaned, and covered. Bleeding should be controlled. Closure of wound may be considered between periods or at end of contest. Closure of wound may be deferred until later, but wounds should be closed within 6 hours whenever possible.
  - Wound may be closed with suture, staples, surgical adhesive, or Steri-strips.
  - Proper wound cleaning and anesthesia are required.
  - Laceration closure should be done only by qualified, trained personnel.
- Tetanus status should be checked and updated if required in any athlete with a skin injury.

Musculoskeletal

- Orthopedic injuries are common in sports activities and include fractures, dislocations, and ligament and tendon injuries.
- Return-to-play issues are prevalent in musculoskeletal injuries.

Fracture

- Fractures range from obvious to subtle and difficult to diagnose.
- Suspect fractures if there is tenderness over the bone, crepitus, deformity, or ecchymosis.
- It is rare to have x-ray facilities at most sporting venues. Unless the deformity is obvious or the fracture is open, the definitive diagnosis of fractures may have to be delayed until radiographic studies are done.
- Sideline management is aimed at preventing further injury and providing comfort until the definitive diagnosis and subsequent treatment can be started.
- When evaluating a suspected fracture, always check neurologic and vascular integrity of the extremity before and after manipulating the injury.
- An open fracture, whether bone is exposed or a laceration is evident over a suspected fracture site, is an orthopedic emergency and should be splinted and sent for emergent evaluation.
- Closed fractures may be splinted and sent for routine evaluation if distal neurologic and vascular status is normal. Urgent evaluation and appropriate radiographic studies should be sought if there are any questions about the stability or neurovascular status of the fracture site.
- Reduction may be needed if there are neurovascular compromise and an anticipated delay in obtaining definitive care. Reduction may also help with pain reduction, or if the deformity makes it difficult to transport the athlete.
- Reduce the fracture only if there is the ability to maintain the reduction.

Dislocation

- As with fractures, it may be difficult to make the definitive diagnosis without radiographs.
- Reductions should be attempted only by properly trained physicians after neurologic and vascular evaluation.
- Multiple attempts at reduction are not recommended. If the reduction is not going easily, there may be something blocking the reduction, such as an undiagnosed fracture.
- Finger (interphalangeal joints), shoulder, and elbow dislocations are the most common reductions.
- Ankle dislocations may be difficult to reduce and are usually associated with fractures.
- Treatment of hip dislocation generally should be deferred until x-ray and definitive care is available.
- Knee dislocations should be suspected if three ligaments are injured. It is important to notify the emergency department so proper evaluation of vascular integrity can be performed. Knee joint dislocation should not be confused with patella dislocation.

Splinting

- The general rule for splinting is to splint the joint proximal and distal to a suspected fracture and the bone proximal and distal to a joint injury.

- The splint should be securely applied, but with caution not to put it on too tightly. Extremities will swell and a tight wrap could increase the risk of compartment syndrome developing.
- Always check neurovascular status before and after application of splint. Monitor neurovascular status.
- If pain is increasing, remove the splint and re-evaluate the injury. If the athlete is sent home with a splint, the athlete should be given the same advice regarding worsening pain.
- There are many types of splinting materials and the team physician should be familiar with materials available. All have advantages and disadvantages.
  - Malleable aluminum splint (e.g., SAM splint)—lightweight, x-ray lucent, relatively expensive
  - Padded wooden splints—lightweight, inexpensive, difficult to store, not malleable
  - Cardboard—inexpensive, difficult to store, weaken if they get wet
  - Prepackaged padded fiberglass splints—conformable, customizable, more expensive, require some additional training to apply
  - Air splints—conformable, relatively expensive, monitor air pressure, need pump
  - Vacuum splints—conformable, expensive, need pump

SUMMARY

- Sideline management skills are an essential role for the team physician.
- There are few procedures or assessments performed on the sidelines or in the training room that the team physician would not do in the office setting.
- Comfort on the sidelines can be attained only by spending time on the sidelines.
- Preplanning, proper equipment, mental preparation, and communication are the keys to sideline management.
- One of the hardest jobs of the team physician is determining the ability of an athlete to return to play with the limited information available at the sporting venue.

RECOMMENDED READINGS

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Sports Nutrition

5

Mandy Gruner

GOALS OF A NUTRITION AND PERFORMANCE PLAN

Both team and individualized nutrition strategies should strive to promote performance and well-being (Fig. 5-1).

Individualize! Certainly generalizations can be made about the diets of athletes, but it is paramount to gain an understanding of an individual athlete's eating behaviors, food preferences, current nutrition knowledge, availability to food, and motivation for change when advising an athlete about nutrition.

Promote a state of energy flux: The goal is to achieve the appropriate nutrient timing and overall caloric intake to best support the daily metabolic, physical, and mental demands of an athlete's training and competition.

Optimize body composition: Strive to sustain an optimal balance of fat-free mass and fat mass that best promotes a state of health and performance for the individual athlete.

Instill responsibility and empowerment: Certainly it is impossible for a clinician to eat *for* an athlete; therefore, it is important that the athlete take ownership of his/her own health and eating habits to promote positive change. It is important to emphasize that lifestyle habits off the field (i.e., eating properly, getting adequate sleep) are just as influential to training as training itself. Assessing an athlete's importance and confidence to make health behavior change can help facilitate successful eating strategies.

Think long-term and proactively: Younger athletes often think that they can "get away" with less than optimal eating behaviors and food choices. However, poor eating habits established in youth may affect health later in life—even young athletes should know how nutrition effects growth, health, and physical and mental performance.

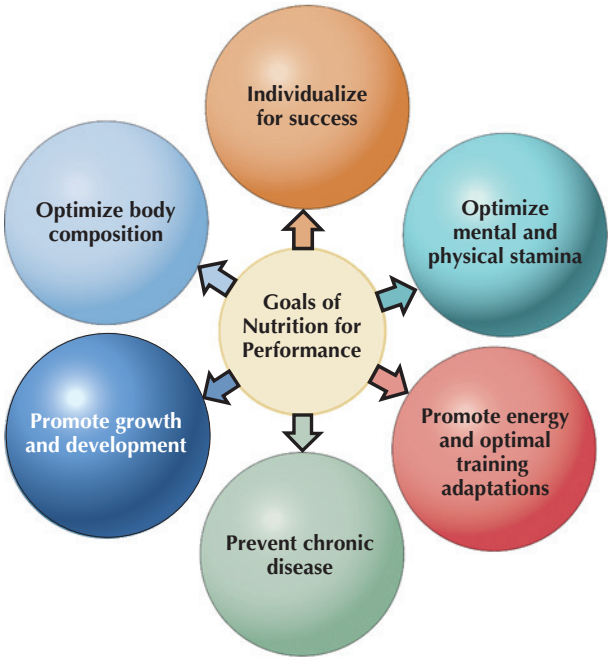

Figure 5-1 Goals of Nutrition for Performance.

THE PHYSIOLOGY OF EXERCISE

Energy Transfer

- Adenosine triphosphate (ATP) is the high-energy compound that is harvested from the oxidation of the macronutrients (i.e., carbohydrate, protein, and fat) in food, and allows cells to do biologic work (Fig. 5-2).
- Cells store a limited quantity of ATP and therefore ATP must be resynthesized.
  - ATP concentration changes rapidly in response to cellular metabolism; thus the imbalance stimulates the breakdown of other stored energy-containing compounds.
  - This change in ATP concentration depends largely on the intensity of the activity performed, thus determining which of the three energy systems becomes the major contributing source of ATP for the working muscles.
- Energy is primarily transferred either anaerobically or aerobically, but no form of exercise is exclusively one or the other (Fig. 5-3).
  - **The immediate system:** The ATP/phosphocreatine system fuels high-intensity or high-power bursts of activity

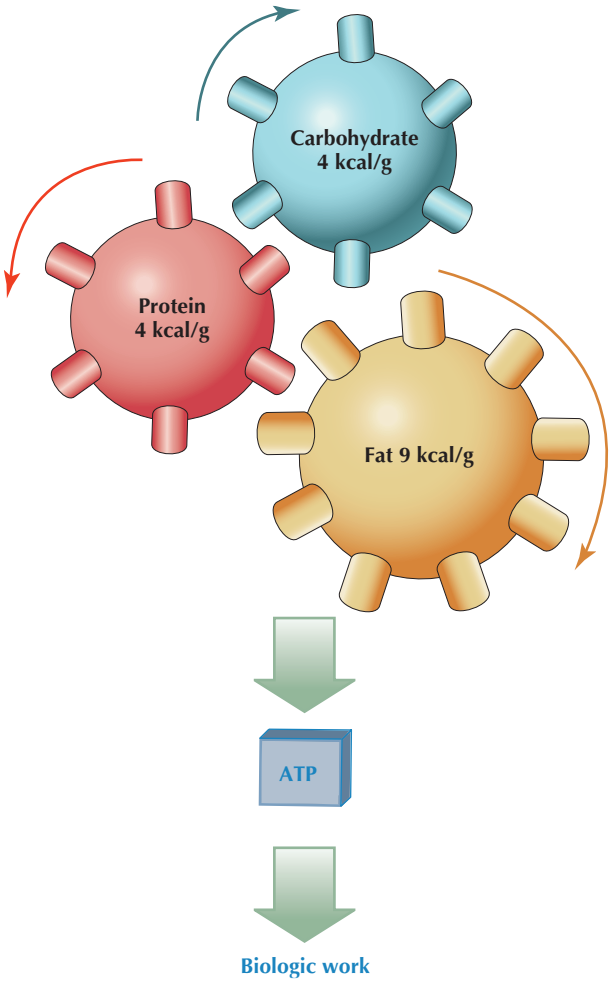

Figure 5-2 Sources of ATP for biologic work. ATP is harvested from the macronutrients, carbohydrate, protein, and fat to synthesize ATP for biologic work.

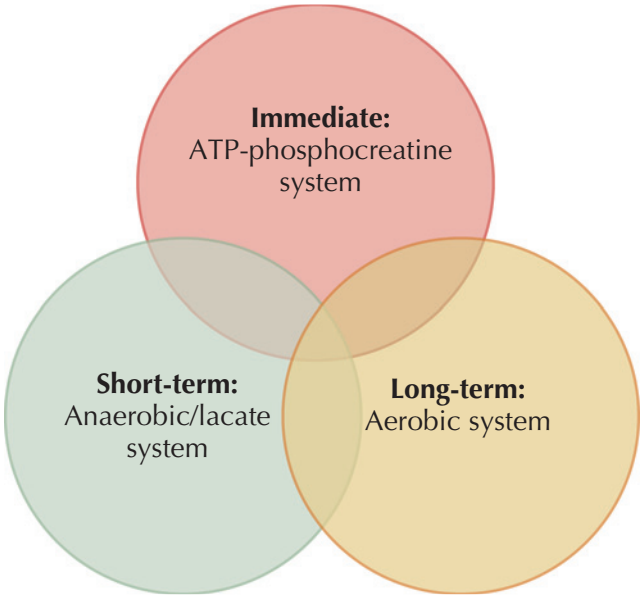

Figure 5-3 The three energy systems. All of the energy systems work to supply ATP to working muscles during exercise, but the intensity and duration of the exercise performed will dictate which system is more predominant.

lasting 5 to 6 seconds (e.g., power lifting, short all-out sprinting such as 50 or 100 m). Creatine is derived from animal food sources such as meat, fish, and poultry.

- **The short-term system:** Human cells' capacity for glycolysis remains crucial during physical activities that require maximal effort for up to 90 seconds whenever adequate oxygen for aerobic activity is not available. Lactate accumulation results when a lactate threshold is reached; then work intensity decreases because of increased muscle acidity and the inhibition of fatty acid breakdown. Carbohydrate is the only macronutrient that can act as a substrate to supply ATP anaerobically.
- **The long-term system:** The aerobic energy system takes place in the mitochondria of cells and may use carbohydrates (glucose and glycogen), fats (fatty acids from adipose tissue and intramuscular triglyceride), or protein (amino acids) as substrate for ATP in the presence of oxygen. The two parts of this energy system include the Kreb cycle and the electron transport chain.

Energy Storage

- Energy is stored in the chemical bonds of dietary carbohydrates, fats, and proteins.
- **Glucose and fatty acids** are the primary sources of energy for skeletal muscle. **Amino acids** are not a preferred energy source for working muscles but serve more regulatory, structure, and function purposes.
- **Muscle and liver glycogen** are the storage reservoirs for dietary carbohydrate. An adult liver can store approximately 100 g of glycogen and the muscles can store 400 g of glycogen (Fig. 5-4). Muscle glycogen levels can be further optimized through aerobic training and a high-carbohydrate diet.
- **Adipose tissue and intramuscular triglycerides** represent storage reservoirs for triglycerides; they contain 60,000 to 110,000 kcal of triglycerides and 3000 kcal of stored energy (see Fig. 5-4). Endurance training increases the muscles' capacity to oxidize fat as a fuel source; thus, muscle glycogen is spared. The body's capacity to store fat far exceeds its capacity to store carbohydrates. However, at higher exercise intensities, the body is unable to oxidize fat as a fuel source; thus, the availability of muscle glycogen stores is crucial to exercise performance.

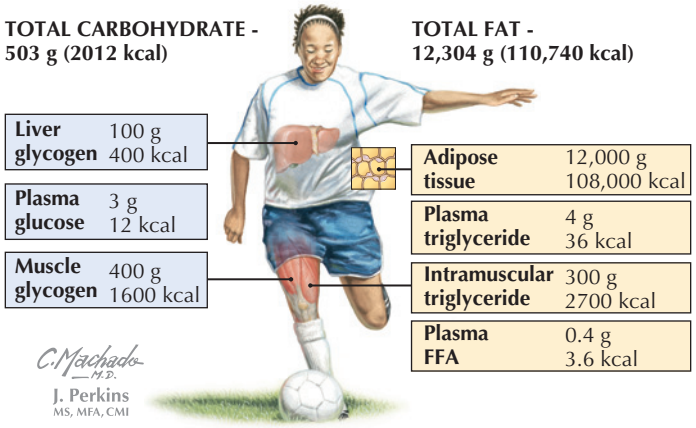

Figure 5-4 Energy stores in humans. Distribution and quantity of energy stored as fat and carbohydrate in an average 80-kg person.

NUTRIENT REQUIREMENTS

Energy (Daily Caloric Requirements)

Energy intakes of athletes: Energy intake is highly variable depending on the age, gender, and body size of the athlete, as well as the frequency, duration, and intensity of training. Male athletes may consume on average 4000 to 6000 kcal per day, and female athletes may consume anywhere between 1600 and 3000 kcal per day. A state of "energy drain"—a condition in which energy demands far exceed energy intake—is commonly observed in female athletes.

Energy balance equation: The energy ingested and the energy expended must be accounted for; thus, the first law of thermodynamics serves as the basis for the energy balance equation. Balancing energy intake and expenditure represents a primary goal of physically active individuals who desire body mass maintenance (Fig. 5-5).

Importance of energy balance: A balance between ingested and expended energy optimizes physical performance, maintains

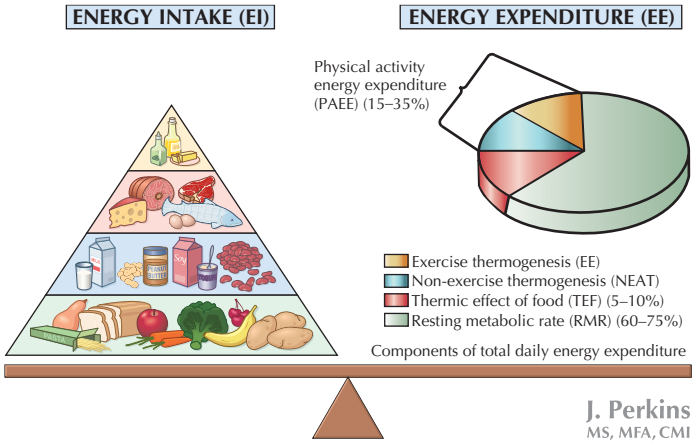

Figure 5-5 The energy balance equation. Intake of adequate calories in addition to an optimal nutrient composition helps support the daily energy demands of training and competition. When an athlete desires to manipulate body mass, the state of energy balance needs to be manipulated. For weight loss: EI < EE; for weight gain: EI > EE.

lean body mass, maximizes positive training adaptations, and protects immune and reproductive function.

Regulation of human energy intake: The factors that regulate human energy intake and expenditure are complex, and include both physiological and psychosocial factors.

- **Physiological signals:** Intake is regulated by the hypothalamus, which integrates gastrointestinal-related signals, signals perpetuated by macronutrient metabolism, and chemical signals from the central and peripheral nervous systems that are either anabolic (hunger-stimulating, such as neuropeptide Y [NPY]) or catabolic (hunger-suppressing, such as leptin) to determine the biological drive to eat or not eat.
- **Psychosocial signals:** Team or sport culture (e.g., a wrestler restricting food intake to make weight), behavioral factors (e.g., eating before bed or skipping breakfast), and environmental factors (e.g., food availability, portion sizes, sensory qualities of food) may greatly influence an athlete's eating behaviors and beliefs about food.

Total daily energy expenditure (TDEE): Daily caloric needs are influenced by many factors including age, gender, genetics, total body weight, and amount of lean body mass. The amount of energy expended varies widely among individuals and even within the same individual on different days (Fig. 5-6).

- **Resting energy expenditure (REE):** The largest component, accounting for 60% to 75% of TDEE. Its biggest determinant is body size, specifically lean mass, including skeletal muscle and internal organs.
- **Exercise thermogenesis (ET)** has two components:
  - **Nonexercise activity thermogenesis (NEAT):** This includes energy expenditure from maintenance of posture, the activities of daily living, and even fidgeting.
  - **Physical activity energy expenditure (PAEE):** For athletes this includes energy expenditure from daily training and competition, in addition to recreational exercise.
- **Thermic effect of food (TEF):** This accounts for about 5% to 10% of TDEE and represents the increase in energy expenditure for digestion, absorption, and assimilation of macronutrients.

Carbohydrate

Fuel for training: Dietary carbohydrate, a macronutrient that supplies 4 kcal/g, is the preferred energy source for the working muscles. Carbohydrate is the predominant fuel for exercise performed at an intensity of 65% of maximal oxygen consumption ($\dot{V}O_{2max}$) or more—the level at which most athletes train and compete. Therefore, consuming adequate daily carbohydrates is imperative to maintain and replenish muscle and liver glycogen stores between bouts of training and competition.

Recommendation: Nutrition recommendations for the general public are commonly expressed as a percentage of total energy intake—approximately 45% to 65%. For athletes it is best to express recommendations in an absolute quantity or relative to body weight (Fig. 5-7).

Application: Athletes speak more in the language of food versus grams and percentages, so it is important to educate them about (1) the preferred food source of carbohydrates and (2) carbohydrate goals per eating occasion. Generally, two thirds of an athlete's plate should be composed of foods that supply nutrient-dense, carbohydrate-rich foods (more whole foods than processed foods) (see Fig. 5-7). Often lack of cooking skills or lack of time to prepare meals causes many athletes to derive a majority of their dietary carbohydrate from more convenience foods (e.g., chips, crackers, candy bars, macaroni and cheese, pizza), which are often higher in fat and lower in fiber, magnesium, selenium, and other micronutrients and health-benefiting phytochemicals.

- **Complex carbohydrates:** Balancing the diet with carbohydrate sources that are rich in vitamins, minerals, phytochemicals, and fiber is an important strategy to ensure optimal metabolic processes. A variety of fruits, vegetables, and whole grains would be optimal at each of an athlete's primary daily meals (see Fig. 5-7).
- **Simple carbohydrates:** Because of an athlete's typically high-energy expenditure, foods that contain simple sugars (honey, maple syrup, fruit preserves, juices, sport drinks) can easily fit into a sound training diet; this way, foods high in processed sugars (e.g., high-fructose corn syrup) are not displacing more nutrient dense complex carbohydrate sources.
- **Carbohydrate replacement supplements:** Athletes who have difficulty eating enough food to consume adequate overall energy and carbohydrate to meet training demands may consider incorporating commercially available nutritional supplementation that helps boost overall energy and carbohydrate intake. Overall, these products (e.g., sport drinks, recovery beverages, bars, and gels) can be more practical and convenient pre-, during, and post-training or competition, but should be used only to supplement a well-balanced diet that contains plenty of whole foods.

Protein

Fuel for training: If carbohydrate and overall energy intake are adequate to support training, amino acids should account for less than 5% of total energy expenditure. Unlike carbohydrate and fat, no "reservoir" for protein exists—protein contributes to tissue repair and acts as a component of metabolic, transport, and hormonal systems. Essentially, amino acids are the building blocks for synthesizing tissue. However, protein does supply

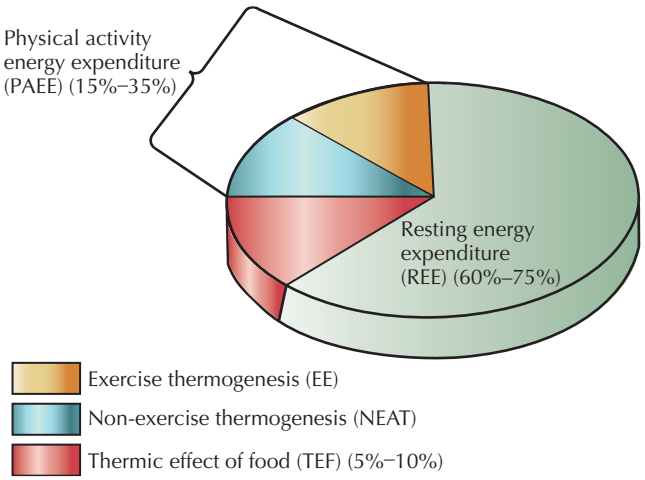

Figure 5-6 Components of Total Daily Energy Expenditure.

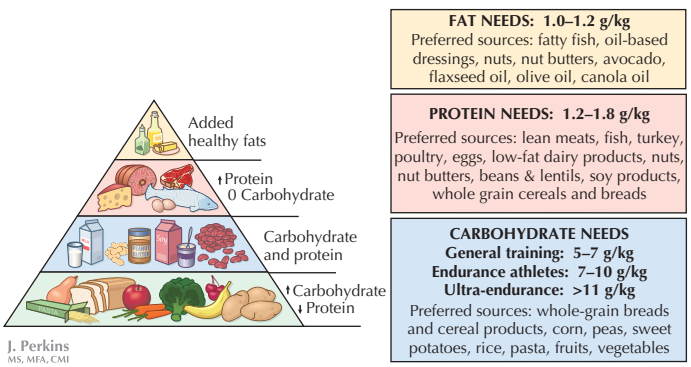

Figure 5-7 Daily Macronutrient Recommendations for Athletes.

4 kcal/g and can become a significant energy source under certain circumstances, such as in a low-carbohydrate condition or during prolonged exercise.

Recommendation: In general, athletes' protein requirements are greater than those of their sedentary counterparts and are most accurately determined relative to body weight rather than as a percentage of total energy intake. Daily protein requirements range from 1.2 to 1.7 g/kg for strength-trained (1.6 to 1.7 g/kg) and endurance-trained athletes (1.2 to 1.4 g/kg), with no benefit beyond 2.0 g/kg of body weight (Table 5-1). Very few athletes are at risk of protein deficiency provided that energy intake is sufficient to maintain body weight and sound nutrition principles are followed.

Application: Athletes are encouraged to obtain protein in their diets from a wide variety of plant and animal sources in order to obtain all of the essential amino acids along with essential vitamins, minerals, and antioxidants (see Fig. 5-7). Athletes are advised to choose leaner animal sources to avoid a high intake of saturated fat. Preferably one third of an athlete's plate should provide protein-rich foods**.** There is no benefit to "bolusing" or eating large quantities of protein per meal—generally 20 to 40 grams per meal spread over three meals and a couple protein-containing snacks will easily meet an athlete's daily protein needs.

Considerations for vegetarian and vegan athletes: Whether vegetarian diets can adequately meet the nutritional needs of athletes and support athletic performance continues to be questioned. Although long-term studies are lacking, some observations can be made:

- Well-planned vegetarian diets can provide sufficient energy and an appropriate range of the macronutrients to support performance and health.
- Vegetarian athletes can meet protein needs from plant sources with both plant and animal protein sources appearing to provide equivalent support for athletic performance. Vegetarian athletes should consume at the higher end of current recommendation ranges because of the lower digestibility and essential amino acid profile of plant protein (see Table 5-1).
- Female vegetarians are particularly at risk for nonanemic iron deficiency, given the lower bioavailability of iron from plant foods.
- Health professionals should be aware that disordered eating can be masked or perpetuated by the adoption of a vegetarian diet because it gives a "reason" to be restrictive and eliminate food groups. Especially if unwarranted weight loss results, the athlete should be assessed for a potential eating disorder.
- Other nutrients of concern depending on the degree of vegetarianism may include zinc, vitamin B12, vitamin D, and calcium.

Protein supplements and timing of intake: In general, there is no compelling scientific evidence to suggest that athletes need to supplement their habitual diets with protein powders or amino acid supplements. However, there is benefit to consuming a protein-containing snack or beverage after a muscle-damaging workout. Because commercially available "protein shakes" are convenient, palatable, and portable, many athletes may prefer a drink right after a workout; in these circumstances, a drink that provides "supplemental protein" may be appropriate and beneficial. However, athletes should be advised of what would be a proper amount of protein in a recovery beverage (Fig. 5-8).

Table 5-1 PROTEIN RECOMMENDATIONS FOR ATHLETES

Specific athlete group	Recommendation in g/kg of body weight
Endurance athletes	1.2-1.4
Strength athletes	1.6-1.8
Vegetarian athletes	1.3-1.8
Energy restricted athletes	1.5-1.7

Fat

Fuel for training: Dietary fat plays several important physiologic roles including protection of vital organs, thermal insulation, and acting as a vitamin carrier and hunger suppressor. Like carbohydrate, fatty acids provide an alternate energy source and reserve during lower-intensity exercise.

- Free fatty acids (FFAs), intramuscular triglycerides (IMTGs), and circulating plasma triglycerides can supply 30% to 80% of the energy for physical activity, depending on the training and nutritional status of the individual as well as the intensity and duration of exercise.
- Depletion of muscle glycogen stores may limit an individual's ability to maintain exercise at higher training intensities. As a result, the use of fuel will shift to the mobilization and oxidation of free fatty acids as the primary fuel source.
- Aerobic training enhances the ability of muscle mitochondria to oxidize fatty acids as fuel. In addition, the enhanced responsiveness of adipocytes to lipolysis allows endurance athletes to exercise at higher absolute submaximal exercise levels before experiencing the fatigue that results from glycogen depletion.

Recommendation: According to the Dietary Guidelines for Americans, the recommendation for fat is in the range of 20% to 35% of total calorie intake. Usually after carbohydrate and protein needs are determined relative to an athlete's body weight, it is generally recommended that the remainder of calories come from dietary fat—usually about 1.0 to 1.2 g/kg of body weight—and should be individualized based on the athlete's physical activity level, energy expenditure, growth stage, nutritional needs, and food preferences. In general, athletes are encouraged to choose foods wisely in order to obtain "heart-healthy fats"—the essential fatty acids (omega-3 fatty acids) and unsaturated fats rather than foods that are high in saturated and trans fatty acids that contribute to the onset of atherosclerosis and heart disease. However, very low fat diets (<15%) may harm both performance and health and are discouraged.

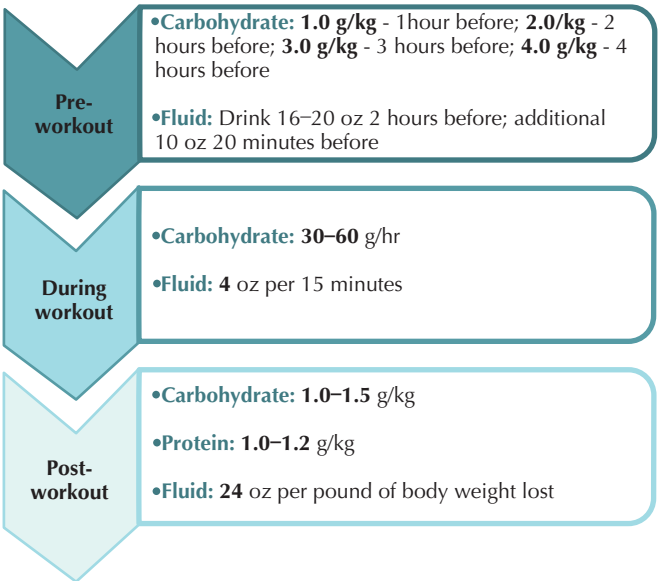

Figure 5-8 Pre-, During, and Post-Workout Carbohydrate, Protein, and Fluid Recommendations.

Micronutrients

- Micronutrients play a specific role in facilitating energy transfer and tissue synthesis—two functions very important to the overall performance and recovery of athletes.
- Generally, if an athlete meets overall energy needs and incorporates a wide variety of nutrient-dense foods, vitamin and mineral recommendations can be met through the diet.
- Additionally, athletes tend to eat many highly fortified "sport" foods and drinks. However, athletes competing in lean (e.g., distance running, gymnastics, diving) or weight-restricted sports (e.g., wrestling, lightweight crew) are often at risk for low or marginal intakes of the micronutrients because of low overall food intakes.

Iron

Functions: Iron is a component of hemoglobin, myoglobin, cytochromes of the electron transport chain, and some iron-dependent enzymes; therefore, it greatly influences oxygen transport and energy metabolism.

Effects on performance: Iron deficiency anemia decreases the capacity of skeletal muscle to consume oxygen and produce ATP. Low iron stores are a frequent finding in elite endurance athletes.

Important interventions:

- **Screening:** Endurance athletes and all female athletes should have their serum ferritin, hemoglobin, and hematocrit monitored to treat both iron depletion and iron-deficiency anemia.
- **Education about iron-rich foods:** Athletes should be informed about types and sources of dietary iron to prevent or augment depletion or deficiency (Table 5-2).
- **Supplementation:** Currently, decisions regarding iron supplementation are best made on an individual basis. The treatment for adults with known iron deficiency anemia often involves 3 months or less of 100 mg/day of elemental iron taken on an empty stomach. In addition, supplementation may also be warranted for athletes with low serum ferritin levels.

Table 5-2 COMMON FOOD SOURCES OF IRON

Food	Serving size	Iron (mg)
Fortified ready-to-eat cereals	1 oz	23.8
Fortified instant cooked cereals	1 packet	4.9-8.1
Soybeans, mature, cooked	½ cup	4.4
Pumpkin and squash seed kernels, roasted	1 oz	4.2
Lentils, cooked	½ cup	3.3
Spinach, cooked from fresh	½ cup	3.2
Beef, cooked	3 oz	3.0
Kidney beans	½ cup	2.6
Chickpeas, cooked	½ cup	2.4
Lamb	3 oz	2.3
Prune juice	¾ cup	2.3
Refried beans	½ cup	2.1

Source: Nutrient Values from Agricultural Research Service (ARS) Nutrient Database for Standard Reference, Release 17.

Calcium

Function: Calcium deserves attention because of the incidence of amenorrhea in female athletes, the role of calcium in bone density, and the often low to marginal dietary intake of calcium seen in some athletes. Altogether, these factors place athletes at risk for compromised bone health and/or increased risk for injury. The DRI for calcium is 1000 mg/day for female athletes ages 19 to 50 and 1300 mg for athletes 9 to 18 years old.

Important interventions:

- **Screening:** Female athletes should be asked about current and past intake of dairy products and calcium supplements, as well as menstrual history.
- **Education:** Female athletes should be educated about the highly bioavailable dietary sources in addition to the role that menstrual function and overall diet adequacy has on bone health (Table 5-3).
- **Supplementation:** For athletes with known compromised bone density, 1500 mg of calcium and 800 mg vitamin D are recommended. Calcium supplementation with calcium carbonate or citrate in doses of 500 mg or less taken between meals is recommended to optimize absorption.

HYDRATION: FLUIDS AND ELECTROLYTES

Dehydration is the most common hindrance to performance, but it is also the most preventable.

- **Fluid loss** as little as 1% to 2% of total body weight can begin to elevate core temperature during exercise, resulting in a negative impact on performance. Dehydration greater than 3% of body weight further disturbs physiologic function and can increase risk for exertional heat illness.
- **Involuntary dehydration:** Humans do not maintain fluid balance during prolonged periods of physiologic or thermal stress when fluid is consumed as desired. Thirst is not an adequate indicator of fluid needs during exercise.
- **Acute and chronic dehydration:** Acute dehydration can occur in a matter of 2 to 3 hours; this is most commonly seen in endurance athletes such as marathon runners and triathletes. However, chronic dehydration is less visible and potentially more dangerous, usually resulting from successive days of inadequate fluid replacement; this is often seen during early preseason training in soccer or football.

Fluid type: Fluids from all foods and beverages contribute to one's overall hydration status. Water can be an appropriate choice for some athletes, but others can greatly benefit from rehydration fluids that contain carbohydrate (6% to 8% concentration) and electrolytes. Carbohydrate-electrolyte replacements ("sport

Table 5-3 CALCIUM CONTENTS OF DAIRY AND NON-DAIRY FOOD SOURCES

Dairy Food Sources			*Non-Dairy Sources*		
Food	**Serving size**	**Calcium (mg)**	**Food**	**Serving size**	**Calcium (mg)**
Plain yogurt	8 oz	452	Fortified cereals	1 oz	236-1043
Romano cheese	1.5 oz	452	Soy beverage	1 cup	368
Fruit yogurt	8 oz	345	Sardines	3 oz	325
Swiss cheese	1.5 oz	336	Tofu, firm	½ cup	253
Ricotta cheese, part skim	½ cup	335	Spinach	½ cup	146
American cheese, processed	2 oz	323	Soybeans, cooked	½ cup	130
Cheddar cheese	1.5 oz	307	Oatmeal, instant, fortified	1 packet	99-110
Skim milk	1 cup	306	Cowpeas	½ cup	106
2% reduced-fat milk	1 cup	285	White beans, canned	½ cup	96
Chocolate milk	1 cup	280	Rainbow trout, cooked	3 oz	73

Source: Nutrient Values from Agricultural Research Service (ARS) Nutrient Database for Standard Reference, Release 17.

drinks") can help prompt better overall fluid intake as well as help maintain blood glucose and delay fatigue during intense activities lasting more than 45 to 60 minutes as well as longer endurance events lasting several hours.

Practical means to maintain and monitor hydration status:

- The preferred strategy for maintaining fluid balance at all times during training is to consume fluid before, during, and after training (Fig. 5-8).
  - Athletes should obtain a pre- and post-exercise body weight and consume 24 ounces of fluid for each pound lost.
  - Fluid replacement beverages should be easily accessible in individual containers.
- **Check for signs of dehydration:** Infrequent urination, dark yellow urine, headache, and weakness should be clear signs and symptoms of dehydration; the athlete should stop and drink adequate fluids.

NUTRIENT TIMING

Precompetition or Workout Nutrition

Purpose: Pre-exercise meals or snacks are geared to "top-off" muscle glycogen stores prior to activity, to prevent onset of hunger during exercise, and to optimize hydration status. Because dehydration and energy depletion are results of a cumulative effect of not properly recovering from training, athletes should be conscious of optimal food and fluid choices every day, not only during the few hours before a training session or competition.

Timing: Athletes may choose to eat a larger meal 3 to 4 hours prior to activity because this typically gives plenty of time for digestion. Some may "graze" throughout the time prior to activity and some may fear eating altogether. Overall, carbohydrates and fluids are critical in the time before exercise (see Fig. 5-8).

Physiologic considerations:

- Having an "empty stomach" prior to competition may be desirable for athletes in contact and high-intensity sports for fear of vomiting or discomfort.
- Athletes should know that the protein and fat content of a meal slows gastric emptying; therefore, higher carbohydrate foods at meals and snacks on the day of competition with more moderate amounts of fat and protein are warranted.

Practical considerations:

- Liquid meals can replace conventional foods for athletes who may report having trouble with solid foods prior to training or competition.
- Athletes should be encouraged to try new or unfamiliar foods in a practice situation prior to using them on a competition day.
- Personal preference and toleration need to be considered—athletes should consume foods and fluids that are well-liked, well-tolerated, usually eaten, and believed to result in a winning performance.

Recovery nutrition: Recovery from strenuous exercise or competition is optimal when athletes are following a high-carbohydrate diet because glycogen stores are maximized.

- Recovery of muscle glycogen can occur in a 24-hour period given that adequate dietary carbohydrate is consumed.
- Carbohydrate (1.0 to 1.5 g/kg) should be consumed immediately following exercise, with an additional 1.0 to 1.5 g/kg to be consumed every 2 hours thereafter (see Fig. 5-8).
- For muscle-damaging workouts, the addition of protein to a recovery snack or beverage has been shown to be beneficial to stimulate protein synthesis.

ISSUES OF WEIGHT AND BODY COMPOSITION

Sports medicine physicians are likely to receive questions regarding both weight gain and weight loss. Because fad dieting, "fat-burners," and muscle-building supplements are mainstream, athletes may have skewed and unrealistic expectations about the amount and rate of body-fat loss and/or muscle gain that is both healthy, genetically feasible, and that may actually deliver an ergogenic effect. Athletes who desire to lose weight fall into three categories:

Relatively Lean Group

- Members of this group may desire weight loss for performance or appearance.
- If the athlete is female and amenorrheic, she may already be restricting intake to a point beyond which further restriction is physiologically inappropriate. It is important to discuss with the athlete why chronically maintaining a lower than natural body weight is detrimental to both health and performance. Achieving a state of energy balance and a level of body fat that allows normal menstrual function is warranted.
- Body-fat and lean body mass levels are largely ruled by genetics. An attempt to maintain a lower than natural level of body fat can harm both health and performance (e.g., recurrent injury or illness, amenorrhea, diminished strength and endurance). Clinicians should be aware of pathogenic weight control behaviors (e.g., overexercise, purging, use of diuretics or laxatives) that athletes may engage in to achieve a lower weight or body-fat level.

Athletes Who Carry Excess Body Fat

- Members of this group may need to lose weight for both health and performance. If training is already maximal or further aerobic exercise would compromise strength or anaerobic training, a modest (300 to 500 kcal) daily calorie reduction may be warranted.

The Rapid Weight Loss Group

- This group includes athletes who have little or no excess body fat but wish to lose weight to compete in a weight-restricted sport (e.g., lightweight crew, wrestling).
- The process of rapid weight loss is usually called "cutting" and is typically accomplished by restricting food and fluids and increasing fluid loss (e.g., sweating, spitting) for 3 to 10 days prior to competition. Precompetition dieting and dehydration are followed by refeeding and rehydrating after weigh-in. This practice is repeated with each competition of the season.
- **Consequences:** From a performance standpoint, even an acute negative energy balance and slight dehydration (1% to 2%) can negatively impact physiologic function and body composition; this can include decreased lean mass and decreased muscle and liver glycogen stores.

Guidelines for the Reduction of Excess Body Fat

Clinicians should review the following information with the athlete who needs or desires to lose body fat:

Basic bioenergetics: Discuss the creation of an energy deficit.

Timing: Weight loss is best accomplished in the off-season.

Rate: For lean and normal weight athletes, a loss of 0.5 to 1.0 pound per week is optimal; for an athlete with excess body fat, a loss of up to 2 pounds per week may be attempted.

Realistic schedule: Based on the above rates of weight loss, a significant loss of fat takes weeks to months to achieve.

Two methods to achieving a negative calorie balance:

- **Food choices and/or eating behaviors:** Based on current intake, consider reducing foods that are not nutrient dense and high in saturated fat and processed sugars; reduce meals eaten out; establish an eating schedule; increase fiber intake; and decrease use of caloric beverages. Often look to episodes of "mindless" or emotional eating when an athlete may be eating for reasons other than physical hunger.
- **Exercise:** Based on current training, determine whether aerobic activity can be increased: Is the athlete already at a maximum level of daily training? Will increased aerobic activity impair strength or sprint training? If not, recommend increased aerobic activity.

Guidelines for Lean Tissue Gain

- Progressive resistance training and adequate calories are critical for weight gain. However, genetic predisposition, somatotype, maturity level, and compliance determine progress.
- **Initially increase caloric intake** by 500 to 700 kcal per day:
  - **Meal and snack frequency** usually must increase to five to nine eating occasions per day. Continuous availability of food allows an athlete to eat whenever hunger occurs or on a predetermined schedule.
  - **Diet composition:** If food volume is maximal, the next option is to concentrate calories without adding bulk. Strategies might include adding nut butters, nuts, dried fruit, chocolate milk, 100% fruit juices, cheese, and sport drinks. Athletes should still adhere to a high-carbohydrate, low-saturated-fat diet.
  - **High calorie supplements:** If an athlete is unable to increase calories to the needed level with traditional foods alone, supplements are an option. High-calorie shakes, meal replacement beverages, smoothies, or bars are useful to increase frequency of eating as well as calories. Exact supplement can be individualized according to the athlete's personal preference.
  - **Evaluate NEAT:** Sometimes athletes who have trouble gaining weight may have a greater NEAT, such as fidgeting where hundreds of calories are being burned just during everyday activity.
- **Increase protein intake:** A positive nitrogen balance is necessary to achieve hypertrophy. See previous section for protein requirements.

DIETARY SUPPLEMENTS

Vitamins and minerals: Many athletes believe that vitamins and minerals are actual sources of "energy." Because "more energy" is a highly sought after commodity, the "more is better" mentality can often lead an athlete to seek out and take supplements that promise enhanced energy and other misleading performance claims. Education regarding the significant health and safety risks regarding consuming "megadoses" of individual vitamins and/or minerals is important and athletes should be encouraged to consult with a health professional (e.g., team physician, sport dietitian, athletic trainer) before taking any supplement.

Other supplements: See Chapter 6, Sports Supplements.

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Sports Supplements

6

Thomas H. Trojian, Stephanie M. Chu, and Jeffrey M. Anderson

PRODUCT OVERSIGHT AND MARKETING

Dietary Supplement Health and Education Act of 1994 (DSHEA)

Food and Drug Administration (FDA): The FDA regulates dietary supplements. This is done under separate regulations from those that cover "conventional" foods and drug products (prescription and over-the-counter). Under DSHEA, the dietary supplement manufacturer is responsible for ensuring that the product is safe before it is marketed. The FDA is responsible for taking action against any unsafe product after it reaches the market. Unlike other drugs, manufacturers need not register or get approval from the FDA before producing or selling dietary supplements. It is the manufacturer's responsibility to make sure that the product label information is truthful and not misleading; the manufacturer also dictates product purity. The FDA established a Dietary Supplements Final Rule to require current goods manufacturing practices (CGMP) for dietary supplements. The rule requires that dietary supplements be produced in a quality manner, not contain contaminants or impurities, and have accurate labeling. Postmarketing responsibilities of the FDA include monitoring safety (voluntary dietary supplement adverse event reporting) and inspecting product information (claims, labeling, package inserts, and accompanying literature).

Federal Trade Commission (FTC): The FTC is responsible for overseeing truth in dietary supplement advertising. The FTC requires that claims on products be symptom-specific, not disease-oriented. For example, statements such as "supplement X can stimulate immune system" are acceptable, but statements such as "supplement X can treat, cure, or resolve infections" are not. Despite the FTC requirement that claims on products be symptom-specific, not disease-oriented, one study that analyzed Internet websites to assess the nature of marketing claims for the eight best-selling herbal products found that this rule is not always followed. The study revealed that most available information derives from vendor sites and half of these sites claim that these products can treat, prevent, diagnose, or cure specific diseases. Physicians should be aware that these claims were on the first page of the most commonly used Internet search engines.

Supplements: Dietary supplements are not evaluated for safety, and manufacturers are not required to prove safety. It is the FDA's responsibility to prove harmful consequences. Therefore, all supplement use should be closely scrutinized in patients who are pregnant or breastfeeding, and in children, unless specifically noted.

Other Oversight

U.S. Pharmacopoeia (USP) has set standards for natural product potency ranges. Some companies obtain USP guarantee for their products. ConsumerLab (www.consumerlab.com) is a helpful site that tests the supplements of various companies and reports their potencies.

Marketing

- Dietary supplements are estimated to be a $60 billion industry worldwide ($20 billion in the United States). The supplement industry has assumed a substantial portion of the health market, grossing nearly $18 billion in 2001.
- The supplements are marketed to athletes' fears. Many athletes believe that they have to use supplements to stay equal to competitors or to gain "competitive edge." They frequently fear that competitors are using supplements. An example of the athletes' commitment to gold: More than 50% of Olympic-caliber athletes stated that they would take a banned substance if it meant they would win every competition for the next 5 years, even if they would then die from adverse effects of the substance. Athletes are aware that small enhancement in sports may lead to large potential gains. Among elite athletes, performance differences are minuscule between first-place and fourth-place winners; even minor enhancements may mean the difference between victory and defeat.
- Marketing companies rely heavily on testimonials of personal experiences, especially from famous people and athletes. As a result, many companies successfully sell unproven products. Supplement manufacturers often sponsor supplement studies, and negative findings may not be published. Word of mouth and hopes to gain "competitive edge" help fuel many sales.
- Sports supplements frequently have no "instant" effects, so to promote sales, companies often add stimulants to give an "energy boost." Despite a new FDA label law to ensure accuracy in labeling of dietary supplements, there continue to be cases of inaccurate labeling. In one FDA analysis of ephedra supplements, between 6 and 20 other ingredients were found. Cases have been reported of legal supplements containing trace amounts of illegal supplements. It is truly a "buyer beware" market (Box 6-1).

COMMONLY USED ATHLETIC PERFORMANCE SUPPLEMENTS

The optimal dose and long-term side effects of most supplements are not known. Manufacturers recommend doses and durations that have been tested, and claimed side effects apply to these instructions. Many athletes may use higher doses than recommended and/or use them for longer periods of time, which raises concern for unknown effects. Most supplements try to enhance the normal effects of exercise on the body.

Arginine

Claims: Arginine is said to acutely improve exercise capacity; its chronic effect results from the stimulation of muscle protein synthesis and thus anabolism of muscle protein. Soy protein is an excellent source of arginine.

Mechanism: Arginine may promote secretion of endogenous growth hormone (GH). It is a precursor in the synthesis of creatine. It augments the production of nitric oxide.

Efficacy: There is little scientific evidence available to support claims of promoting and increasing functional capacity in healthy, athletic participants. Intravenous arginine does increase GH but oral has not shown the same effect. It may increase ni-

BOX 6-1 *"Buyer-Beware" Information about Supplementation*

Most ergogenic aids lack scientific proof.
Most supplements have not been tested adequately for efficacy, purity, or safety.
Be careful of misleading product information.
"Natural" does not mean safe.
"More" is seldom better.
Nothing replaces a well-balanced diet that includes a variety of high-quality foods.
Athletes use supplements at their own risk.

tric oxide production but definitive studies need to be completed. Studies have shown that arginine's effects on muscle protein synthesis are likely a net effect in combination with nitric oxide as well as concurrent elevation of other amino acids.
Side effects: None reported in short-duration studies.
Dosage: 3 to 9 g daily is used in studies, or 250 mg/kg/day.

Bovine Colostrum (BC)

Claims: Some claim that BC supplementation increases insulin-like growth factor-1 (IGF-1) levels. BC supplementation appears to positively influence exercise performance characterized by short bursts of activity. In elite-level athletes, supplementation has not been found to improve body composition. In non-elite athletes, body composition improves.
Mechanism: BC stimulates growth factors, including structurally identical IGF-I. IGF-I has an anabolic effect and is involved in the regulatory feedback of growth hormone. GH stimulates hepatic production of IGF-1. As IGF-1 levels rise, they provide negative feedback to inhibit the pituitary's production of GH.
Efficacy: Limited studies show consistent beneficial effects on recovery and exercise performance.
Side effects: Occasional, minor gastrointestinal complaints, including flatulence and nausea. A high percentage of participants complained about the "unattractive" taste of the beverage.
Dosage: 60 g daily.

Branched-Chain Amino Acids (BCAAs)

Claims: BCAAs are an important source of energy in prolonged endurance exercise. Supplementation is proposed to increase endurance in long tennis matches, soccer, marathons, long-distance swimming, and cycling activities. BCAA supplementation may contribute to increased body fat loss and maintenance of a high level of exercise performance. BCAAs are also claimed to decrease chronic fatigue/overtraining symptoms.
Mechanism: Supplementation replenishes loss of BCAAs used as fuel, increases protein synthesis and growth hormone secretion, shifts leucine metabolism to fat metabolism, stimulates fat metabolism over glycogen in hypocaloric diets, and prevents decrease in plasma glutamine. BCAAs inhibit dietary tryptophan transport across blood brain barrier leading to decreased brain serotonin (associated with several brain regions that control central fatigue).
Efficacy: Most studies show neither beneficial nor detrimental effect of BCAA. Studies of effect of BCAA in hypocaloric state are limited. BCAAs have not been shown to reduce chronic fatigue/overtraining symptoms.
Side effects: Fatigue and ergolytic effects have been reported.
Dosage: Usually combined with other amino acids. Range: 5 to 10 g daily or before exercise.

Carbohydrate Supplements

Claims: Carbohydrate supplements are used to restore muscle glycogen after exercise, maintain plasma glucose during endurance events (especially those lasting more than 90 minutes), and to maximize muscle glycogen before significant glycogen-depleting exercise (e.g., marathon, long-course triathlon). Various sugars are used, including sucrose, glucose, fructose, and maltodextrin (popular among ultra-endurance athletes).
Mechanism: Increased blood glucose stimulates insulin production and GLUT-4 translocation in muscle, which results in increased glucose uptake and glycogen storage in muscle. Carbohydrates with a high glycemic index increase plasma glucose quickly and serve as a fuel source in sustained exercise.
Efficacy: Reviews are mixed for pre-exercise supplementation and carbohydrate loading. Benefits of supplementation after exercise and during long events (more than 90 minutes) are well supported.
Side effects: None have been noted.
Dosage: 0.7 to 1.0 g/kg every 2 hours for the first 4 hours after exercise (first 90 minutes post-exercise is most important). Best if started within 30 minutes of stopping exercise. Use a food source with a high glycemic index. Addition of protein to carbohydrate supplement increases glycogen production. During exercise: 0.7 to 1.0 g per hour for events lasting more than 1 hour. Sources include sports drink (5 to 10 ounces every 15 minutes), sports gels or candies (2 gels and water), or gummy candy (a handful per hour and water). Before exercise (benefits of carbohydrate loading controversial): 4 g/kg within 3 hours before and 1.1 g/kg 1 hour before; 10 g/kg/day of carbohydrates 3 to 7 days before the event.

Chromium

Claims: Chromium is a trace mineral that is used for weight loss and for enhancement of glycemic control in the treatment of diabetes; proposed for the treatment of hyperlipidemia and hypercholesterolemia. Used by athletes in attempts to gain muscle and lose fat.
Mechanism: Chromium functions in carbohydrate, protein, and fat metabolism as a cofactor that enhances action of insulin and uptake of amino acids into muscles. It improves lipid profile and is theorized to sensitize insulin receptors in the brain, resulting in appetite suppression and downregulation of insulin secretion. Glycogen synthesis increases in chromium-deficient individuals. Exercise may result in loss of chromium, but athletes conserve chromium and probably do not become deficient.
Efficacy: Chromium is possibly effective when used to reduce cholesterol, but probably ineffective for weight loss. Mild hypoglycemic effect is caused by a mechanism similar to that of metformin. A large body of scientific evidence indicates that chromium has no effect on body composition when taken in supplement form, and there are serious concerns for the potential adverse effects of chromium accumulation within the body.
Side effects: Chromium interferes with iron metabolism and zinc absorption. Prolonged use and abuse are linked with serious side effects, including anemia, chromosomal damage, cognitive impairment, interstitial nephritis, and gastrointestinal (GI) intolerance. Commercial preparations containing ephedrine recently have been restricted; low doses of the combined preparation have been found to cause hypertension, stroke, and even death.
Dosage: Chromium picolonate is more easily absorbed than other forms of chromium; chromium is complexed to picolonate to facilitate absorption. Common dose is 50 to 200 μg/day, mean dose is 120 μg/day; similar dosing often found in multivitamins; lower doses may be safer than higher doses.

Creatine

Creatine is probably the most often used and the most researched supplement taken by athletes.
Claims: Creatine is said to increase exercise performance in short repetitive bouts of high-intensity exercise offset by brief rest periods (30 to 120 seconds). This increase in exercise performance and work capacity probably leads to increased muscle mass in some athletes.
Postulated mechanism: Creatine is a low-molecular-weight, complex amino acid produced endogenously primarily in the liver and stored primarily in skeletal muscle. Hydrolysis of creatine phosphate results in rapid production of adenosine triphosphate (ATP), which is needed for muscle contraction. Maximal muscle stores of total creatine may enhance ATP turnover rate and increase phosphocreatine resynthesis, resulting in shorter recovery periods and overall increased training load (volume/intensity). Creatine depletion is the limiting factor in anaerobic exercise. Free creatine may stimulate protein synthesis and cause muscle hydration, which results in increased muscle mass and strength.

Efficacy: Numerous studies have examined the effect of creatine supplementation on athletic performance. Despite some disagreement, general consensus is that creatine supplementation has a small, but real, beneficial effect on anaerobic activity, specifically short-duration, repetitive, high-intensity exercise. It does not benefit aerobic training or performance. Data about chronic creatine supplementation, high-dose supplementation, and supplementation in young athletes are lacking. There appear to be responders and nonresponders to creatine. Specifically, vegetarians who do not ingest primary exogenous sources of creatine (meat and fish) may benefit more from creatine supplementation.

Side effects: No serious side effects have been consistently documented from creatine supplementation. Weight gain is a proven side effect. Areas of theoretical concern and anecdotal reports include the following:

- **Renal:** Creatine is spontaneously degraded to creatinine, and increases in both urine and serum creatinine levels have been reported. These elevations are likely brief and clinically insignificant. An important consideration: Does serum creatinine lose its power as a marker for renal function? People with a history of renal dysfunction or disease that may lead to renal dysfunction (e.g., diabetes) should use creatine with caution. Additionally, athletes using potentially nephrotoxic drugs (e.g., nonsteroidal anti-inflammatory drugs [NSAIDs]) may be at higher risk of renal dysfunction. Close monitoring of renal function should be considered. Long-term effects of creatine on the kidney are unknown.
- **Gastrointestinal:** Nausea, bloating, cramping, and diarrhea have been described in creatine users, but such effects are not supported by studies. It appears not to cause hepatic dysfunction.
- **Cardiovascular:** It is unknown how much creatine is taken up by myocardium. Animal studies that report no substantial myocardial uptake also report no skeletal muscle uptake. It remains unknown whether this is detrimental or possibly even beneficial.
- **Dehydration:** Because of anecdotal reports of dehydration, especially in hot, humid conditions, encourage adequate hydration in creatine users. The patient is not truly "dehydrated." Total body water is actually increased, but the osmotic properties of creatine can increase third spacing, depleting intravascular fluid volume.
- **Muscular:** Anecdotal reports have led to widespread belief that creatine can induce muscle cramping. This effect remains unproven. Muscle strains also have been anecdotally reported. Fluid retention that may accompany creatine ingestion, especially with loading doses, may theoretically increase compartment pressures and predispose an athlete to exertional compartment syndrome.

Dosage: Some recommend loading doses of 20 to 30 g/day (5 to 7 g four times daily) for 5 to 7 days (an amount of creatine equal to the amount in 5 to 6 pounds of beef), followed by doses of 2 to 4 g/day to maintain intramuscular creatine stores. Other investigators have shown that intramuscular creatine stores reach the same level with a dose of 3 g/day and that stores are maintained with as little as 2 g/day. This approach eliminates the loading phase. Lower doses take longer to attain the desired intramuscular creatine levels. Dosing may be based on body weight: loading dose of 0.3 g/kg/day with maintenance dose of 0.03 g/kg/day. "More is better" philosophy held by many athletes remains a concern because of the "ceiling" for muscle storage (5 g creatine/kg muscle mass). Excess creatine is not used by the muscles.

Fluid Replacement Beverages

Claims: Fluid replacement beverages are used to prevent and treat dehydration. Dehydration greater than 3% decreases maximal aerobic power by 5%.

Mechanism: These beverages prevent decreases in tolerance of exercise in heat, and help to sustain stroke volume, cognitive functioning, and work capacity.

Efficacy: Numerous studies have shown decreased performance in "hypohydrated" athletes. Various methodological flaws have prevented the documentation of strength in hypohydrated individuals consistently. Recent studies have demonstrated that hypohydration does decrease strength.

Side effects: Overhydration may cause hyponatremia, but the amount of hydration that causes hyponatremia is usually quite excessive and frequently occurs over long periods (e.g., endurance or ultra-endurance events longer than 4 hours). GI upset may occur, especially with fructose-containing fluid replacement drinks.

Dosage: The American College of Sports Medicine recommends consumption of 400 to 600 mL of water 2 hours before exercise and 150 to 300 mL every 15 to 20 minutes of exercise; more is needed in climates associated with high sweat rates. Addition of carbohydrates is recommended for activities greater than 90 minutes. Hydration after exercise is also important.

HMB (Beta-Hydroxy Beta-Methylbutyrate)

Claims: HMB supplementation is said to regulate protein metabolism and is theorized to decrease catabolism, thus increasing lean muscle mass and strength; it is often used to enhance aesthetic physical appearance in bodybuilding.

Mechanism: How HMB works is not fully known. HMB is a metabolite of leucine, a branched-chain amino acid, and may regulate enzymes responsible for protein breakdown, inhibiting breakdown of muscle during and after vigorous activity. HMB in liver and muscle cells is metabolized to HMG-CoA, which is then used in the synthesis of cholesterol. This increases availability of cholesterol for cell wall synthesis. A localized deficiency of cholesterol for cell wall synthesis is postulated as one restriction to muscle hypertrophy, and increased local cholesterol stores could theoretically relieve this restriction.

Efficacy: Few scientific investigations have been published. HMB may have additive effects when combined with creatine.

Side effects: No reported side effects, but studies are inadequate.

Dosage: Dose of HMB used in clinical trials is 1.5 g once or twice daily (2 to 3 g/day).

L-Carnitine

Claims: L-carnitine is said to increase aerobic and anaerobic capacity and promote fat loss.

Mechanism: L-carnitine increases long-chain fatty acid oxidation in skeletal muscle during exercise.

Efficacy: Clinical trials of carnitine supplementation are inconclusive and suffer from a number of design limitations.

Side effects: Nausea, vomiting, abdominal cramps, diarrhea.

Dosage: 2 to 6 g/day consumed in two to three doses with meals.

L-Glutamine

Claims: L-glutamine is the most abundant amino acid in the body. It is used for treatment of wound healing, immune function, and chemotherapy-induced mucositis. Athletes use L-glutamine in attempts to prevent impaired immune response following prolonged exercise.

Mechanism: Originally classified as a nonessential amino acid, glutamine is now considered essential for maintaining intestinal function, immune response, and amino acid homeostasis during times of stress. It is an important fuel for cells of the immune system (lymphocytes and macrophages). During prolonged exercise, as in other forms of chronic stress, plasma glutamine may decrease. Muscle glutamine may drop in efforts to sustain an anabolic state; if glutamine drops below critical levels, athletes may revert to a catabolic state.

Efficacy: Human and animal studies have shown conflicting data. Preliminary data suggest that glutamine supplementation may

enhance immune function. Glutamine has been shown to reduce upper respiratory infections in athletes after vigorous exercise, but more data are needed to confirm this finding. It may be effective in treating chemotherapy-induced stomatitis. In general, reliable data are insufficient to support effectiveness of glutamine for most of its proposed uses.

Side effects: No adverse reactions reported; may be safe at appropriate doses.

Dosage: Glutamine has been tolerated without side effects in doses up to 40 g/day. Typical dose: 20 to 30 g/day.

Nitric Oxide (NO)

Claims: Nitric oxide is thought to increase muscular strength and endurance. It has been shown to be beneficial in patients with cardiac disease and endothelial dysfunction. NO is marketed to athletes with the claim that the vasodilation associated with NO improves muscular vascular perfusion, and that the increase in blood flow improves muscular gains with resistance training.

Mechanism: Nitric oxide (NO) is produced in the body by an enzyme called nitric oxide synthase, which converts the amino acid L-arginine to nitric oxide and L-citrulline. NO works by vasodilatation, thereby facilitating blood flow to muscle cells. NO is bactericidal; it is released by macrophages. The combination of these two effects is seen in septic shock.

Efficacy: Small subject numbers and lack of standardization of previous activity level make current studies inconclusive.

Side effects: None identified in short-term studies.

Dosage: Supplements rely on the conversion of an intermediate to NO, most often arginine. Dosages of 3 to 6 g of arginine daily are used.

Protein Supplements

Claims: Protein supplementation above American Dietetic Association (ADA) recommendations (0.8 g/kg/day) is used to prevent negative nitrogen balance and to aid protein synthesis, especially during high-intensity exercise. Many athletes, especially weight lifters, use protein supplements to "bulk up" or to add muscle mass. Most frequently used varieties include whey, soy, or egg whites.

Mechanism: Protein supplements aid synthesis of new muscle proteins. Whey protein is a good source of branched-chain amino acids, which were discussed previously.

Efficacy: Whey protein is a soluble, easy-to-digest protein. Most studies show change in muscle synthesis with increased protein intake, but subjects were not tested for increased strength. Athletes require more protein then nonathletes (see Chapter 5, Sports Nutrition), and protein supplements may be used as adjuncts to diet.

Side effects: None documented at doses up to 2 g/kg/day in healthy individuals, but sustained use at this level is concerning. Caution is recommended in athletes with renal insufficiency or failure as well as in individuals with lactose or dairy protein allergies. Excessive protein intake is stored as fat.

Dosage: Recommended dose for endurance athletes is 1.2 to 1.4 g/kg/day, and for strength-trained individuals, 1.6 to 1.7 g/kg/day is recommended.

Tribulus Terrestris

Claim: Tribulus terrestris is an herb that is said to "naturally" increase testosterone levels.

Mechanism: It is postulated that tribulus terrestris increases the release of luteinizing hormone (LH), indirectly stimulating testosterone release.

Efficacy: Studies do not support claims that tribulus terrestris improves body composition or athletic performance. Elevation of serum testosterone may have anabolic effects.

Side effects: No reported side effects in humans. Photosensitivity has been reported in animals grazing on tribulus terrestris. If testosterone is elevated, there are side effects similar to those of anabolic steroids.

Dosage: Tribulus terrestris is commonly sold in tablets of 500 to 650 mg; once-daily dosing is frequently recommended. It is often combined with other "prohormones."

LESS COMMONLY USED ATHLETIC SUPPLEMENTS

Ginseng

Claims: Ginseng has been used by the Chinese for thousands of years as a stimulant, a diuretic, to promote menstruation, and to fight infection. Other claims concern treatment of adrenal and thyroid dysfunction and aphrodisiac qualities. Athletes use ginseng to enhance aerobic performance and energy level and to help the body deal with stress.

Mechanism: It is hypothesized that ginseng stimulates the hypothalamic-pituitary-adrenal axis, which may result in increased resistance to various types of stress. It may enhance myocardial metabolism, increase oxygen extraction by muscles, and optimize mitochondrial metabolism in muscle. During strenuous exercise, ginseng may increase cortisol response and enhance the body's ability to sustain muscle creatine phosphate levels thus decreasing lactic acid production.

Efficacy: No quality evidence supports the claim that ginseng supplementation enhances physical performance.

Side effects: The most common side effects are nervousness and excitability that decrease after the first few days. Many people find the taste unpleasant. Hypoglycemia and difficulty concentrating have been reported. Because of ginseng's estrogen-like effect, women who are pregnant or breastfeeding should not take it. Occasionally, there have been reports of more serious side effects, such as asthma attacks, increased blood pressure, palpitations, and in postmenopausal women, uterine bleeding.

Dosage: Doses of 100 to 200 mg three times a day have been studied. A study of 50 commercial products revealed a level of active ingredient ranging from 1.9% to 9%, with 6 showing no evidence of ginseng.

Glutathione

Claims: During exercise free radicals are produced, and the stimulation effects of glutathione may counteract the results of extreme physical training.

Mechanism: Hepatic glutathione production increases as exercise duration increases. The liver uses glutathione to remove vitamin E radicals and replenish vitamin C from radicals.

Efficacy: The effectiveness of aerosolized, intramuscular, or intravenous glutathione has not been well established. Glutamine and N-acetylcysteine are under study to determine if they increase glutathione levels.

Side effects: No known side effects; not well studied.

Dosage: Oral glutathione is not bioavailable; standard dosages have not been established.

Glycerol

Claims: Oral ingestion of glycerol may induce a state of "hyperhydration," which may result in superior athletic performance.

Mechanism: Oral glycerol acts as an osmotic agent and increases water retention.

Efficacy: Results are equivocal regarding benefits of glycerol-induced hyperhydration on core temperature, plasma volume, and exercise tolerance. Hyperhydration does not appear to offer benefit compared with euhydration. Hyperhydrated athletes may be less likely to become dehydrated, especially during exercise in extreme environmental conditions of high heat and humidity. Adverse effects of hyperhydration remain concerning (e.g., electrolyte shifts), and practice should be to never replace

proper oral hydration, proper acclimatization, and good sense during exercise in extreme conditions.

Side effects: Isolated reports of headache, bloating, and nausea after oral ingestion. Otherwise data about side effects are limited.

Dosage: Typically 1 to 1.2 g/kg mixed with 1.5 L of fluid 1 to 2 hours before competition.

Lysine

Claims: Lysine is used for prevention and treatment of recurrent herpes simplex labialis and improved athletic performance.

Mechanism: Lysine inhibits the growth of herpes simplex virus (HSV) in vitro; it is important in collagen synthesis and bone formation. It has been proposed that lysine, arginine, and ornithine increase human growth hormone (HGH).

Efficacy: Lysine reduces healing time, severity, and recurrence of HSV labialis. Lysine, arginine, and ornithine have shown no ergogenic benefit in resistance or aerobic exercise. Intravenous arginine before endurance activity has been insufficiently studied.

Side effects: Lysine is contraindicated in patients with renal disease or hepatic impairment, as they may not be able to eliminate large amounts of nitrogen produced from supplemented amino acid breakdown. Hypercalcemia may result from increased gastric absorption and decreased excretion. No data supports the use in children or pregnant or breastfeeding women.

Dosage: For recurrent herpes: 1000 mg/day for 1 year or 1000 mg three times daily for 6 months. No ergogenic dose has been established.

Pyruvate

Claims: Pyruvate is said to increase exercise endurance, help with weight loss, and reduce body fat content.

Mechanism: BCAAs transfer an amine group to pyruvate to form alanine. Alanine has the role of increasing lipid oxidation and decreasing carbohydrate oxidation. Pyruvate also may reduce free-radical production. It enhances leg exercise endurance capacity by increasing glucose extraction by muscle.

Efficacy: Studies are limited. Reports of oral pyruvate supplementation used for weight loss are promising. Increases in time to exhaustion with endurance exercise have been noted in untrained participants. Further studies are needed before a recommendation can be made.

Side effects: GI upset including gas, bloating, and diarrhea. One death reported after intravenous administration.

Dosage: Typically 22 to 44 g/day.

Sodium Bicarbonate

Claims: It is said that sodium bicarbonate increases time to exhaustion, and decreases time in sprinting for races of 400 to 1500 meters.

Mechanism: Sodium bicarbonate causes buffering of blood, which leads to metabolic alkalosis, thereby decreasing the effects of lactic acid.

Efficacy: Mixed reviews have been published. Although sodium bicarbonate has been found to be effective in horses, it is illegal for horse racing. The positive effects are seen for events lasting more than 1 minute and less than 7 minutes.

Side effects: GI symptoms of belching, bloating, and flatulence.

Dosage: Dosage used in studies is 300 mg/kg before exercise.

GENERAL HEALTH SUPPLEMENTS

Black Cohosh

Claims: Black cohosh is used for the treatment of menopausal symptoms, dysmenorrhea, and premenstrual discomfort.

Mechanism: Unknown. Evidence of estrogenic effects is conflicting. There is some evidence of competitive inhibition of estradiol binding to estrogen receptors.

Efficacy: Black cohosh may be helpful in treatment of menopausal symptoms, dysmenorrhea, and premenstrual discomfort.

Side effects: Contraindicated in pregnancy; GI upset (diarrhea, vomiting, nausea).

Dosage: 20 mg black cohosh and rhizome extract twice daily.

Coenzyme Q-10 (Ubiquinone)

Claims: Coenzyme Q-10 (CoQ-10) is used to treat congestive heart failure (CHF), angina, diabetes, metabolic myopathies, and hypertension; to prevent drug toxicity; to increase exercise tolerance; and to stimulate the immune system in AIDS patients. There are many other uses.

Mechanism: CoQ-10 is an essential cofactor in ATP production. It has antioxidant effects and prolongs antioxidant effects of vitamin E. Some patients with AIDS, cardiovascular disease, and periodontal disease are deficient in CoQ-10.

Efficacy: CoQ-10 does not improve aerobic exercise performance. It may reduce symptoms and increase quality-of-life scores in patients with CHF (mechanism unknown) and be minimally effective in treatment of hypertension, diabetes, muscular dystrophy, and immunosuppression associated with AIDS.

Side effects: Therapeutic doses of CoQ-10 may cause GI upset, headache, dizziness, and allergic skin reactions. It may increase risk of bleeding with warfarin and other anticoagulant or antiplatelet drugs.

Dosages: 100 to 225 mg/d, depending on use.

Chondroitin Sulfate

Claims: Chondroitin sulfate is used as a chondroprotective against progressive osteoarthritis, a non-cyclooxygenase inhibitor, and an anti-inflammatory agent.

Mechanism: Unknown. In animal models chondroitin sulfate reduces inflammatory response and experimental cartilage destruction. In vitro studies show stimulation of chondrocytes to replace or repair damaged proteoglycans in the joint.

Efficacy: Chondroitin sulfate has not been as well studied as glucosamine; a few randomized, controlled trials have shown a decrease in NSAID use for pain relief in patients with osteoarthritis. There is no evidence that the natural process of osteoarthritis was altered or that surgical intervention was ultimately prevented. The large chondroitin sulfate molecules are poorly absorbed with approximately 10% bioavailability.

Side effects: Theoretically, people with shellfish allergy can have an allergic reaction because chondroitin is made from marine exoskeleton.

Dosage: 1200 mg/day in divided dosages.

Echinacea

Claims: Echinacea is thought to prevent and shorten duration of upper respiratory infections and flulike illnesses.

Mechanism: Three species of medicinal interest (*Echinacea purpurea*, *E. augustifolia*, and *E. pallida*) act as immunostimulants and help immune defenses combat infection. It may have indirect antiviral capabilities, possibly by stimulating interferon response.

Efficacy: A recent meta-analysis indicates that Echinacea is probably effective in shortening the course of viral upper respiratory infections if started within first 48 hours of illness. The same meta-analysis failed to document benefits in prevention of viral illness.

Side effects: Echinacea is probably safe when used orally for short periods; no long-term data is available. It may case allergic reaction, especially in people with atopy or allergies to ragweed, daisies, and chrysanthemums. Several minor side effects reported, including nausea, unpleasant taste, dizziness, and abdominal pain.

Dosage: Varying doses have been studied, including *E. purpurea* herb juice, 6 to 9 mL for up to 8 weeks and 20 drops every

2 hours on the first day of illness, followed by three times a day for the remainder of the illness; *E. purpurea* extract tablets, two tablets three times daily; and herbal compound tea, 5 to 6 cups on first day of illness followed by titration to 1 cup a day.

Evening Primrose

Claims: Evening primrose is used for the treatment of premenstrual dysphoric disorder; cyclic and noncyclical mastalgia; and the reduction of perimenopausal symptoms (e.g., hot flashes) and pain of rheumatoid arthritis. There are many others uses.

Mechanism: Gamma-linolenic acid (GLA) found in evening primrose oil is precursor to prostaglandins E_2 and E_1. GLA also is converted to 15-hydroxy-dihomo-GLA, which may competitively inhibit production of prostaglandins and leukotrienes, thereby reducing inflammation. Premenstrual dysphoric disorder may be associated with lower GLA levels.

Efficacy: Good evidence for use in mastalgia; evening primrose is less effective but safer than danazol. One study shows usefulness in rheumatoid arthritis. It may be beneficial in premenstrual dysphoric disorder. The use of evening primrose in pregnancy is associated with complications during delivery. Other uses not adequately evaluated.

Side effects: Nausea, soft stools, indigestion, and headache; seizures when used with phenothiazines.

Dosage: Formulations are standardized to 9% gamma linolenic acid. Normal dosages are 2 to 4 g/day. In the United Kingdom, evening primrose is used as a prescription medication, in doses of 6 to 8 g/day, to treat atopic eczema.

Garlic

Claims: Garlic is used frequently to reduce high blood pressure and prevent coronary artery disease by improving lipid profiles. Other uses include stimulation of immune system and treatment of diabetes, reduction and prevention of colds and flu, and reduction of stress and fatigue.

Mechanism: In hypertension patients, garlic may cause smooth muscle relaxation and vasodilation by stimulating production of endothelium-derived relaxation factor. Lipid profiles improve secondary to HMG-CoA reductase inhibitor activity. Garlic may stimulate humoral and cellular immunity.

Efficacy: Garlic is possibly effective in the treatment of hyperlipidemia and hypertension, but evidence is inconclusive. There is insufficient reliable data to support effectiveness of other uses.

Side effects: Dose-related, including unpleasant breath odor and GI upset; more common with raw garlic.

Dosage: In clinical trials for hyperlipidemia and hypertension, 600 to 1200 mg/day divided in three doses. Fresh garlic, 4 g/day (about 1 clove) may be used.

Ginkgo biloba

Claims: There are many uses associated with *Ginkgo biloba:* to improve cognitive function in people with dementia, including Alzheimer's disease; to treat vascular insufficiency (central and peripheral), dysmenorrhea, and acute mountain sickness; to improve sleep in patients with depression.

Mechanism: *Ginkgo biloba* is a free radical scavenger and inhibitor of monoamine oxidase; it is thought to stimulate populations of nerve cells that have remained functional as well as to protect nerve cells from pathologic influences.

Efficacy: *Ginkgo biloba* may improve cognitive function with dementia. Clinical trials and meta-analyses have demonstrated a delay in the progression of Alzheimer's. It possibly is effective in treatment of claudication and acute mountain sickness.

Side effects: Therapeutic doses of *Ginkgo biloba* may cause GI upset, headache, dizziness, and allergic skin reactions. It may increase risk of bleeding with warfarin and other anticoagulant or antiplatelet drugs.

Dosage: 120 to 240 mg/day, depending on use.

Glucosamine

Claims: Glucosamine is used as a chondroprotective against progression of osteoarthritis (OA); it is said to have anti-inflammatory effects.

Mechanism: In vitro glucosamine stimulates cartilage cells to produce proteoglycans and glycosaminoglycans on a dose-dependent basis; it also increases mRNA production. Weak anti-inflammatory effect is not produced by inhibition of cyclooxygenase and is unrelated to prostaglandins. Anti-inflammatory effect is poorly understood. Glucosamine is produced in the body by attachment of an amino group to glucose. Glucosamine is acetylated to acetyl glucosamine.

Efficacy: Glucosamine sulfate is best. Numerous controlled trials in Europe and the United States show positive effects. Glucosamine often is combined with chondroitin sulfate, but the effects of the combination have been inadequately studied. Glucosamine is available in many forms. Two bioavailable forms that have been studied are glucosamine sulfate and glucosamine hydrochloride (HCL). Absorption rate approaches 80% to 90%. Some studies show decreased pain and stiffness associated with OA, but studies are conflicting. In vitro studies show reduction in NSAID requirements by one-half after addition of glucosamine; combination may yield similar anti-inflammatory effects to NSAIDs alone. Glucosamine may slow progressive osteoarthritis at knee, as evidenced by decreased rate of radiographic joint space loss. A Cochrane review from 2005 found that the Rotta preparation of glucosamine was superior to placebo in alleviation of pain and functional deficits in OA patients. Non-Rotta preparations were not different than placebo. Neither Rotta nor non-Rotta preparations were different than placebo when Western Ontario and McMasters Universities (WOMAC) osteoarthritis index scores were assessed. Using the WOMAC scale as a measure of effectiveness, neither glucosamine nor chondroitin sulfate, either alone or in combination, is better than placebo in decreasing knee pain in OA patients by 20%. Those with moderate to severe knee pain responded more favorably to glucosamine.

Side effects: None reported for glucosamine HCL. Glucosamine sulfate may cause mild GI problems, including nausea, heartburn, diarrhea, and constipation. Supplements are made from bovine and calf cartilage; hence, theoretical but unlikely risk of exposure to bovine spongiform encephalopathy. Glucosamine does increase serum glucose levels in some diabetics with osteoarthritis.

Dosage: 1500 mg/day in single or divided doses. Intramuscular dosages of 400 mg twice weekly have been used experimentally. Dose may vary with product; dose equivalences have not been established.

Melatonin

Claims: Melatonin is said to have sedative/hypnotic effects; to assist in recovery from jet lag; to improve sleep in blind patents and patients with other disorders that alter the sleep-wake cycle (e.g., shift workers). It is an adjunct to chemotherapy and in treatment of depression. Other claims include use as antioxidant, prevention of aging effects, increases in energy, boosting of immune system, adjunct in treatment of epilepsy, contraception, and prevention of cancer.

Mechanism: Melatonin is naturally produced in the pineal gland and promotes sleep; it is secreted in diurnal fashion and its release is inhibited by light and stimulated by darkness. It lowers alertness and body temperature. Low levels of melatonin are reported in people with insomnia.

Efficacy: Melatonin may reduce jet lag, aid sleep, and improve circadian rhythm disturbances in blind, mentally retarded, and autistic patients. There is some in vitro support for antioxidant effects. There is insufficient data to support other claims.

Side effects: Drowsiness; users should not operate motor vehicles or heavy machinery within 4 hours of oral ingestion. There is

theoretical concern about interference with gonadal development in children and adolescents. Melatonin can also cause irritability, dysphoria, dizziness, and abdominal cramping. The data concerning long-term use is insufficient.

Dosage: Typical dosing for jet lag: 5 mg at nighttime for 3 days before flight; typical dosing for sleep disturbance is 0.5 to 6 mg at bedtime.

Niacin

Claims: Niacin is used with diet therapy to treat dyslipidemia; it is also used as an adjunct in treatment of peripheral vascular disease/coronary artery disease (PVD/CAD). Niacin is said to augment energy during exercise. It is used in the prevention and treatment of niacin deficiency. Used by bodybuilders to increase superficial vascularity (causes "flush") before bodybuilding contests. It may decrease joint pain in osteoarthritis. It is used to decrease side effects of isoniazid therapy.

Mechanism: Niacin may increase homocysteine levels and modify abnormal coagulation factors that accompany PVD/CAD. B-complex vitamins such as niacin are involved in energy production during exercise via oxidative phosphorylation and the Krebs cycle.

Efficacy: Niacin is effective in lowering triglycerides and raising high-density lipoproteins; it may be effective as a secondary means of preventing heart attack and in symptomatic treatments of osteoarthritis.

Side effects: Low-dose dietary supplementation with niacin can cause minor effects such as flushing, which may be accompanied by pruritus, rash, headache, and occasionally muscle pain. Higher doses have been associated with headache, dizziness, nausea, and vomiting. Interactions may occur when niacin is combined with carbamazepine and HMG-CoA reductase inhibitors.

Dosage: Limited data suggest the need for B-complex vitamins may be twice the daily recommended amount. Dietary supplement dosage is 10 to 20 mg/day, as an adjunct to isoniazid, 20 mg/day; for hyperlipidemia, 1000 to 2000 mg nightly; for osteoarthritis, 3 g/day in divided doses of niacinamide. RDA is 16 mg/day; 18 mg/day for pregnant women.

Saw Palmetto

Claims: Saw palmetto is used to reduce symptoms associated with benign prostatic hyperplasia (BPH), such as urinary frequency, dysuria, urgency, and hesitancy; it is an adjunct, with other herbs, in treatment of prostate cancer. Other claims include treatment of male alopecia, "strengthening" of thyroid gland function, reduction of testicular inflammation, expectorant, digestive aid, female breast enlargement, and aphrodisiac qualities.

Mechanism: Saw palmetto has antiandrogenic, antiproliferative, and anti-inflammatory capabilities. In vitro, it inhibits 5-alpha reductase, diminishing conversion of testosterone to dihydrotestosterone. The same effects have not been demonstrated in vivo.

Efficacy: Saw palmetto has been shown to be more effective than placebo, equally effective as finasteride, and less effective than alpha-adrenergic blockers in treating BPH; it does not decrease total prostate size or prostate-specific antigen. It requires 1 to 2 months of use for beneficial effects.

Side effects: Similar to placebo in studies; unlikely to be associated with increased impotence, as is finasteride.

Dosage: Clinical studies have used 320 mg/day, either as one dose or in two divided doses. Tablets are made of extract containing 80% to 90% fatty acids from saw palmetto berry.

Selenium

Claims: Selenium is used by people hoping to prevent cancer, heart disease, osteoarthritis, and rheumatoid arthritis. Athletes use antioxidants such as selenium to combat the potentially damaging free radicals that are produced from lipid peroxidation associated with the stress of exercise.

Mechanism: Selenium is a trace mineral that plays an important role as a cofactor for glutathione perioxidase, which helps reduce oxidative tissue damage from free radicals.

Efficacy: The efficacy of selenium is unknown. Recommendations are for athletes to eat foods rich in selenium. Selenium supplementation may be beneficial in athletes who abstain from meat, in athletes who restrict calories in weight-control sports, and in persons who live in areas of natural selenium deficiency (e.g., China).

Side effects: Side effects are dose-dependent, and usually seen in doses greater than 100 μg/day; they include nausea, vomiting, abdominal pain, and fatigue.

Dosage: RDA is 70 μg/day for males and 50 μg/day for females.

Soy Isoflavonoids

Claims: Soy isoflavonoids are used for menopausal symptoms and to prevent osteoporosis and heart disease in postmenopausal women. They are also used for hypertension and hyperlipidemia.

Mechanism: Isoflavones are hydrolyzed by enzymes in jejunum to phytoestrogens, which may stimulate estrogen receptors in heart, vasculature, bone, and bladder.

Efficacy: Soy isoflavonoids may be effective for hyperlipidemia in combination with low-fat diet and for treatment of perimenopausal symptoms such as hot flashes.

Side effects: GI complaints are common, including nausea, constipation, and bloating; allergy in susceptible people.

Dosage: Typically 25 to 30 mg/day.

St. John's Wort

Claims: St. John's wort is used in the treatment of mild to moderate depression and obsessive-compulsive disorder. Other claims include use for muscle spasms, ulcer, menstrual cramps, and as an expectorant.

Mechanism: Unproven. Some data indicate that hyperforin, a potentially active component, may elevate serotonin levels through reuptake inhibition or by 5-HT3 and 5-HT4 receptor antagonism. Hypericin, another potentially active component, may act as a receptor antagonist at GABA-A, GABA-B, benzodiazepine, and adenosine receptor.

Efficacy: St. John's wort is effective compared with placebo in short-term treatment of mild to moderate depression; it is possibly as effective as low-dose tricyclics and some selective serotonin reuptake inhibitors (SSRIs; sertraline and fluoxetine). It may be effective in treatment of secondary symptoms associated with depression. The data is not sufficient to comment on ability to treat obsessive-compulsive disorder. Other claims lack sufficient supporting evidence.

Side effects: St. John's wort is well-tolerated and probably safe when used appropriately for short periods. Most common side effect is insomnia, which can be alleviated by taking it in the morning or by decreasing dose. Other minor side effects include dry mouth, restlessness, agitation, headache, vivid dreams, dizziness, and paresthesias. Important concerns arise from drug-drug interactions, especially involving other antidepressants. Use with SSRIs may be synergistic and increase risk of serotonin syndrome. Addictive effects with monoamine oxidase inhibitors (MAOIs) include hypertension, hyperthermia, confusion, and coma. It should not be used by anyone who has used an MAO inhibitor in the past 14 days.

Dosage: Most trials have used St. John's wort with hypericin content of 0.3% at dose of 300 mg three times daily. Doses up to 1200 mg/day have been reported.

Valerian

Claims: Valerian is said to have sedative and anxiolytic effects, to elevate mood, and to improve concentration. Other claims (from various websites) include beneficial use for treatment of

tremors, epilepsy, attention deficit hyperactivity disorder, rheumatic pain, muscle spasm, menstrual cramps, ulcers, and hypertension.

Mechanism: Precise mechanism by which valerian root may cause sedation is not known. Some data suggest that interference with catabolism of gamma aminobutyric acid (GABA) concentrations may result in its elevation.

Efficacy: Small studies indicate that valerian may be beneficial in elevating mood and improving concentration and as a sedative. Sedative effects are less rapid than with benzodiazepines; may need to use consistently for up to 4 weeks to achieve effects. There is insufficient data to support other claims.

Side effects: Valerian is probably safe if used for short periods. Safe use has been documented in trials lasting up to 28 days. Longer use is associated with benzodiazepine-like withdrawal response. There have been four reports of hepatotoxicity with longer-term use. Potential side effects include drowsiness, headache, excitability, and cardiac disturbances. Patients should be warned not to operate motor vehicles or heavy machinery.

Dosage: Numerous doses and formulations are studied and marketed. Maximum dose should be 15 g/day. Valerian can be taken as tea, tincture, or extract in pill form.

Vanadyl Sulfate

Claims: Vanadyl sulfate is used for diabetes, hypoglycemia, and heart disease and to increase strength with weight training.

Mechanism: Vanadyl sulfate is a cofactor for various essential enzymatic reactions; it may mimic the effects of insulin or potentiate its actions.

Efficacy: Vanadyl sulfate is possibly effective in treatment of diabetes; it acts like metformin. There is no ergogenic effect in weightlifters compared with placebo. There is not enough scientific evidence to support other claims.

Side effects: It is possibly safe in small doses for short-term use. Minor side effects include green discoloration of tongue and GI upset. Serious effects have been reported with high doses; these include leukocytosis and manic-depressive disorder. It potentiates warfarin and digoxin.

Dosage: 10 to 60 μg/day with food. Average diet provides 15 to 30 μg/day.

Vitamin C

Claims: Vitamin C is said to prevent or reduce the duration of upper respiratory infections. Antioxidant properties are claimed to prevent cardiovascular disease and cancer. Vitamin C is used to treat hypertension, exercise-induced asthma, and osteoporosis. Claims also include that it speeds recovery from injury and improves absorption of iron from the GI tract.

Mechanism: Vitamin C is a water-soluble vitamin, naturally found in citrus fruits and vegetables, that plays a role in collagen formation and bone health. Its antioxident properties led to speculation about its role in treating diseases in which oxidative stress may play a part. Vitamin C increases glucose and free fatty acid mobilization through epinephrine synthesis, enhanced iron absorption, and antioxidant benefits, therefore enhancing immune resistance.

Efficacy: Vitamin C has been shown to promote iron absorption from the GI tract, and it may have a role in preventing cancer when obtained directly from fruit and vegetable sources. The same benefit has not been shown with supplement use. It may modestly decrease duration of common cold symptoms, aid conventional antihypertensives in lowering systolic blood pressure, and decrease exacerbations of exercise-induced asthma. It is effective in the treatment and prevention of osteoporosis and may slow progression of osteoarthritis (only when obtained directly from fruit and vegetable sources). Vitamin C decreases the risk of complex regional pain syndrome after wrist fracture. It does not appear to prevent the common cold or aid treatment of cancer.

Side effects: Side effects are uncommon and dose-related. Hyperoxaluria, hematuria, crystalluria, hyperuricosuria, and predisposition to urinary stone formation may be related to intake greater than 1 g/day. Other side effects include intestinal obstruction, other GI distress, headache, insomnia, fatigue, and flushing.

Dosage: Recommended daily allowance (RDA) ranges from 65 to 120 mg/day. Tobacco use increases daily requirements. As a dietary supplement, vitamin C is typically taken at doses of 75 to 90 mg/day. Some common foods (guava, kiwifruit, broccoli, loganberry, brussels sprouts) can supply 80 to 100 mg of vitamin C/100 g of food, and they may be beneficial. Food sources that supply 100 to 200 mg/day may be beneficial. Studies have shown that up to a quarter of athletes take less than 70% of the recommended daily dose. Doses of 1 to 3 grams have been described for prevention and treatment of the common cold. Vitamin C side effects are more common at higher doses.

Vitamin E

Claims: Vitamin E is also an antioxidant, and similar claims to vitamin C are made concerning prevention of cardiovascular disease and cancer. Vitamin E may be useful in treating diabetes and its complications, and Alzheimer's disease and other dementias. Numerous other claims include treatment of asthma and various neuromuscular disorders, prevention of allergies, negative side effects of air pollution, signs of aging, and cataracts. Also, vitamin E is said to reduce delayed onset of muscle soreness in extreme exercise.

Mechanism: This is a fat-soluble vitamin found naturally in grains, meats, poultry, eggs, fruits, and vegetables. Because of its wide distribution through different foods, true vitamin E deficiency is rare. Deficiency may occur with fat malabsorption syndromes or eating disorders. Primary function is as antioxidant, preventing the formation of free radicals; proposed benefits are mostly related to this function.

Efficacy: Vitamin E's role in the prevention of cardiovascular disease is controversial. Controlled, blind, multicenter trials have shown no benefit, whereas other studies have shown benefit from vitamin E supplementation. It may be efficacious for a large number of different problems, including treatment of dementia and normalizing retinal blood flow in diabetics.

Side effects: Side effects are dose-dependent; vitamin E is generally safe, even at doses exceeding RDA. Upper limit of dosage is 1000 mg/day. Reported side effects include GI distress, fatigue, weakness, rash, gonadal dysfunction, and creatinuria.

Dosage: Recommendations can be confusing; they are expressed in mg, but most products are labeled in IUs. To convert to IU of natural vitamin E, multiply the number of mg by 0.67. To convert to IU of synthetic vitamin E, multiply the number of mg by 0.45. RDA for older children and adults is 15 mg/day; with lactation, 19 mg/day. Doses vary for other claimed uses.

Yohimbine

Claims: Yohimbine is use in the treatment of impotence, diabetic neuropathy, postural hypotension, and sexual dysfunction caused by SSRIs.

Mechanism: Yohimbine increases blood flow to the penis, nerve impulses to the genitals, and reflex excitability in the spinal cord; it penetrates the central nervous system and works primarily as an alpha$_2$-adrenergic receptor blocker. Effects are similar to those of monoamine inhibitor, calcium channel blocker, and peripheral serotonin receptor.

Efficacy: Yohimbine is probably effective in the treatment of impotence; use is limited by numerous side effects. Other uses not adequately studied.

Side effects: Tremors, hallucinations, excitability, anxiety, hypertension, and psychosis. It interacts with many drugs and supplements, including clonidine, MAO inhibitors, phenothiazines

(promethazine), tricyclic antidepressants, caffeine, ephedra, *Ginkgo*, and St. John's wort. It is contraindicated in people with angina, hypertension, bipolar disease, depression, anxiety disorder, prostate disease, and schizophrenia.

Dosage: 5.4 mg tablet three times daily. No approved FDA indication, but it is listed as a drug, not a supplement. It can be purchased over the counter as bark extract.

RECOMMENDED READINGS

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Sports Pharmacology of Pain and Inflammation Control in Athletes

7

Sourav K. Poddar

OVERVIEW

The pharmacology of pain management in the athletic arena can be a critical component in returning an athlete to play. Several options exist and choosing an appropriate medication should involve careful consideration of the goals of treatment and potential adverse reactions. This chapter will review some commonly used formulations in greater detail.

NONSTEROIDAL ANTI-INFLAMMATORY DRUGS (NSAIDs)

Nonsteroidal anti-inflammatory drugs (NSAIDs) are frequently prescribed to athletes by sports medicine providers as a way to limit inflammation and pain and to subsequently facilitate return to play. Research into the effects of these widely used medications has brought into question the role of these drugs in treating athletic injuries.

Prevalence

- NSAIDs are one of the most commonly used medications. Estimates put the number of prescriptions at more than 85 million annually. This amounts to more than $3 billion dollars spent annually on these medications.
- About one out of seven Americans use NSAIDs with some regularity. There are 17 million regular users; according to some estimates, as many as 50% of those users are over the age of 65.
- An estimated 13% of the U.S. population over the age of 65 uses an NSAID.

Mechanism of Action

- **NSAIDs work by inhibiting mainly the cyclooxygenase (COX) pathway** and, to a lesser extent, the lipoxygenase pathway, thereby blocking the conversion of arachadonic acid to prostacyclins, prostaglandins, and thromboxanes.
- Through this mechanism, NSAIDs possess antipyretic, analgesic, and anti-inflammatory actions.
- Blocking the production of certain prostaglandins also causes NSAIDs to exert an inhibitory effect on neutrophil aggregation and lysosomal enzyme release.
- NSAIDs are thought to also have nonprostaglandin effects on limiting leukotriene synthesis via inhibition of membrane-related processes.
- **The two main forms of cyclooxygenase, COX-1 and COX-2,** are thought to have different functions (Fig. 7-1).
  - COX-1 is thought to be more of a constitutive enzyme, involved in the synthesis of prostaglandins that regulate physiologic processes. It plays an important part in the function of the gastric mucosa, kidneys, vascular endothelium, and platelets.
  - COX-2, on the other hand, is thought to be mainly an inducible isoform (although recent data show that it may have a role in some constitutive processes). It is involved in the synthesis of prostaglandins that mediate inflammation, pain, and fever in response to tissue injury.
  - The concept behind the development of COX-2 specific inhibitors was to preserve the physiologic function of COX-1 while limiting the effects of COX-2 on tissue injury. The nonselective NSAIDs block both isoforms and subsequently have a significant side effect profile (Fig. 7-2).

Alternate Mode of Delivery

Topical

- Pharmaceutical compounding of NSAIDs has been commonplace in Europe for some time and compounded NSAIDs have become readily available in the United States as well.
- The purported benefit of topical delivery of NSAIDs lies in the decrease in adverse systemic effects on the gastric mucosa, kidneys, and vascular endothelium.
- Serum concentrations of topical NSAIDs appear to be significantly lower than levels measured after oral intake or intramuscular administration. This may result in fewer drug-drug interactions as well.
- Efficacy has been shown in several trials with improvement in subjective pain symptoms. In one such study, topical ketoprofen has been shown to reduce the incidence of delayed-onset muscle soreness with minimal systemic absorption.
- The most common side effect is local irritation at the site of application, although this is uncommon.
- Topical NSAID compounding appears to provide an interesting alternative mode of use in the setting of acute superficial soft tissue injury with limited side effects.

Types

Within the family of NSAIDs are several subclasses that may provide subtle differences in metabolism and therapeutic effect (Box 7-1).

Adverse Reactions

- The significant side effect profile of NSAIDs should lead physicians to use caution when prescribing them. Long-term use of NSAIDs may increase the risk of stroke, myocardial infarction, and thrombotic events.
  - One study showed that short-term (3-day) use of the intravenous COX-2 inhibitor parecoxib followed by 1 week of oral valdecoxib for perioperative pain after coronary artery bypass increased cardiac morbidity.
  - Though the majority of those in the athletic setting are young and healthy, care should nonetheless be taken when considering COX-2 inhibitors at high doses for a prolonged period.
  - NSAIDs in general have also shown to increase the incidence of hypertension.
- The most common side effect associated with NSAID use is gastrointestinal (GI) bleeding.
  - Although the development of the COX-2 inhibitors was supposed to ameliorate this problem, the data is conflicting regarding an overall decrease in GI side effects.
  - The therapeutic doses of nonselective NSAIDs increase the risk of bleeding problems.
  - The increased risk of GI bleed, ulceration, and stomach or intestinal perforation may occur at any time without warning symptoms with prolonged use.
  - Using medications that decrease gastric acid production, such as histamine-2 blockers or proton pump inhibitors, in conjunction with NSAIDs has been recommended to decrease risk of GI bleed, ulceration, and stomach or intestinal preformation.
  - Dosing for shorter intervals and taking the medication with food has also been used as a potential preventive measure.

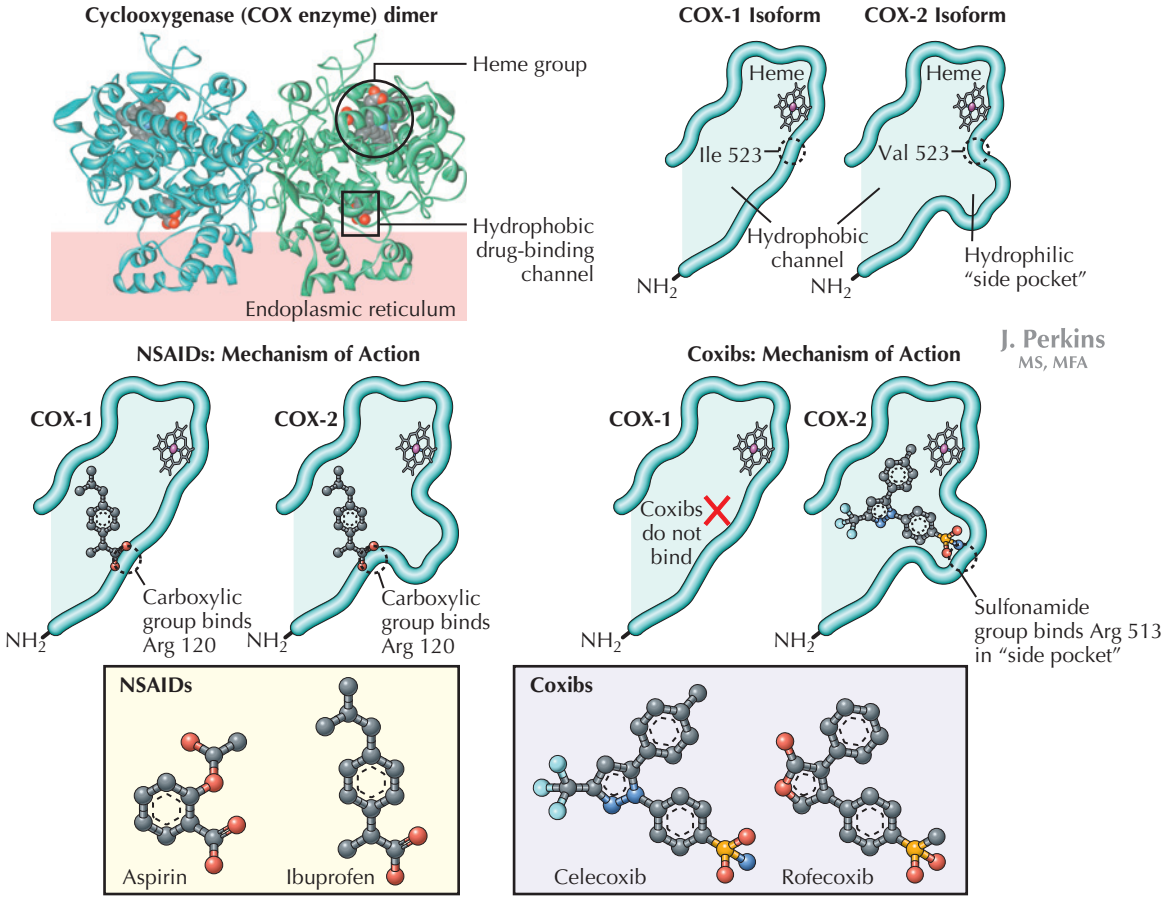
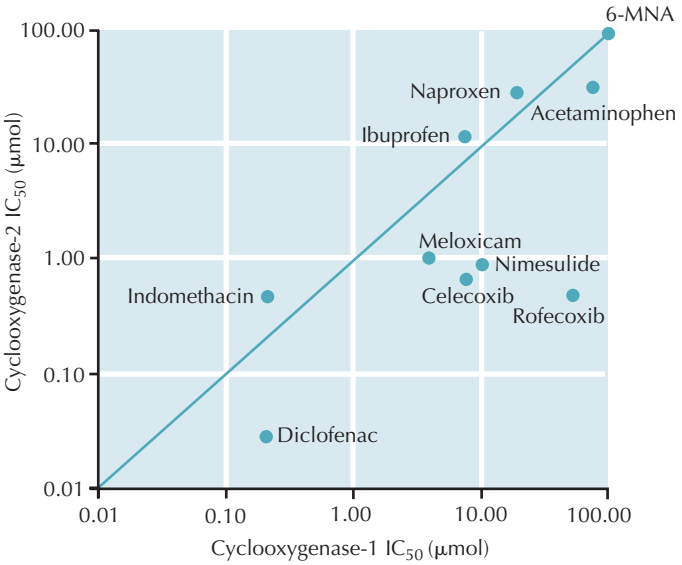


Figure 7-1 Nonopioids: NSAIDs, Selective Cyclooxygenase-2 Inhibitors, and Acetaminophen.



Relative selective of NSAIDs Concentrations of various nonsteroidal antiinflammatory drugs required to inhibit the activity of cyclooxygenase-1 and cyclooxygenase-2 by 50 percent (IC_{50}) in assays of whole blood. The diagonal line indicates equivalence. Drugs plotted below the diagonal line are more potent inhibitors of cyclooxygenase-2 than of cyclooxygenase-1. 6-MNA denotes 6-methoxy-2-naphthylacetic acid. (Adapted from FitzGerald GA, Patrono CN: Engl J Med 345:433, 2001.)

Figure 7-2 Various NSAIDs have different degrees of inhibition of COX-1 and COX-2.

BOX 7-1 *Subclasses of NSAIDs*

Salicylic acid derivatives ASA Salicylsalicylate Diflunisal	**Indole acetic acids** Indomethacin Sulindac Etodolac
Heteroaryl acetic acids Diclofenac Ketorolac Tolmetin	**Arylpropionic acids** Ibuprofen Naproxen Ketoprofen
Fenamates Mefenamic acid Meclofenamic acid	**Enolic acids** Piroxicam Phenylbutazone
Alkanones Nabumetone	**COX-2 inhibitors** Celecoxib

- The impact of NSAID use on various musculoskeletal factors has been studied.
  - Animal studies show that use slows fracture healing and may contribute to malunion and/or nonunion. It has been suggested that use should be curtailed in stress fractures at high risk for nonunion.
  - Although studies of NSAID use in the setting of ligament sprains and muscle strains show an earlier improvement in symptoms and subsequent return to activity, the effect of long-term use on soft tissue healing is unknown.

- Finally, NSAIDs block constitutive prostaglandins necessary for proper kidney function.
  - NSAIDs decrease sodium excretion and increase free water retention. This is more of an issue with long-term use, although care should be taken regarding use during endurance events.
  - NSAIDs may also cause interstitial nephritis, regardless of length of use.

Pharmacokinetics

- NSAIDs are rapidly and completely absorbed in the GI tract.
- Although most NSAIDs are metabolized via the cytochrome P450 system through the enterohepatic circulation, excretion occurs through the kidneys.
- The half-lives of various NSAIDs vary considerably, ranging from a few to several hours.

Therapeutic Recommendations

- Despite their significant side-effect profile, judicious short-term use of NSAIDs in the athletic arena is justifiable. In the setting of sprains, acute muscle strains, eccentric load injury to muscle, and acute tenosynovitis or tendonitis, a 3- to 5-day course may help improve pain and facilitate quicker return to activity.
- Caution should be exercised regarding use of NSAIDs with acute fractures or stress fractures of areas at high risk for nonunion.

OTHER ANALGESICS

Opioids

- Narcotic analgesics should be used judiciously. Significant side effects, such as sedation and subsequent deficits in coordination, cognition, and reaction time, make these poor choices for pain control before or during competition and use during a sporting event cannot be justified (Fig. 7-3).

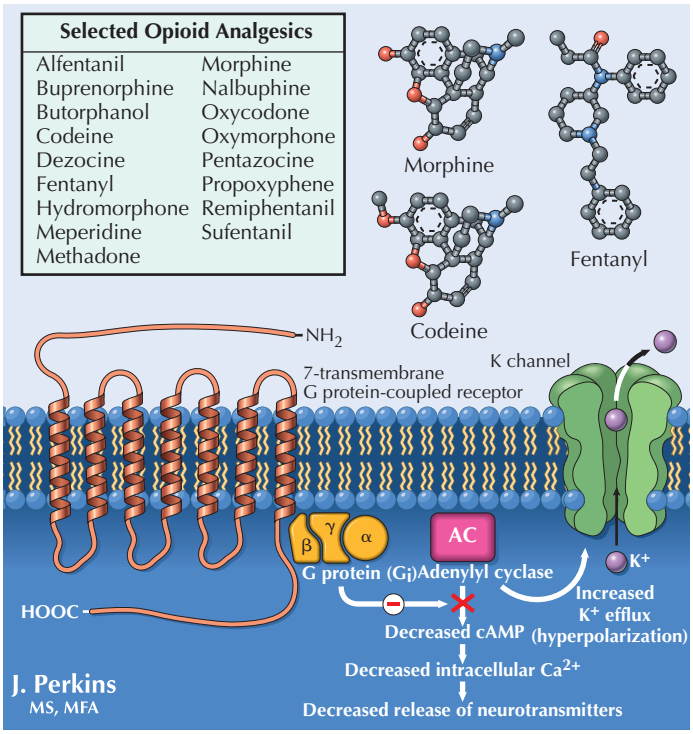


Figure 7-3 Opioids: Receptor-Transduction Mechanisms.

- Most opioids are banned by major sports governing bodies unless prescribed for use by a physician with appropriate cause.
- Certain situations, such as a broken bone or severe acute trauma, may warrant the use of opioids. However, care must be taken to avoid extended use because physical dependency may readily develop.

Acetaminophen

- One of the most common over-the-counter medications recommended by physicians, acetaminophen is considered a first-line pain reliever according to many current guidelines.
  - Although the exact analgesic mechanism is not known, acetaminophen does have inhibitory effects on the cyclooxygenase-1 and -2 pathways in the body.
  - In addition, acetaminophen also exerts an antipyretic effect via direct action on the hypothalamic heat-regulating center.
- Metabolism occurs via the cytochrome P450 system in the liver and the potentially toxic metabolites are then excreted in the urine.
- Athletes with renal impairment should have maximal dosages of acetaminophen reduced because of the potential accumulation of these toxic metabolites. Similarly those with impaired liver function should have liver enzymes monitored with prolonged use.
- The maximal recommended dose in healthy adolescents and adults is 4 grams a day.

Tramadol

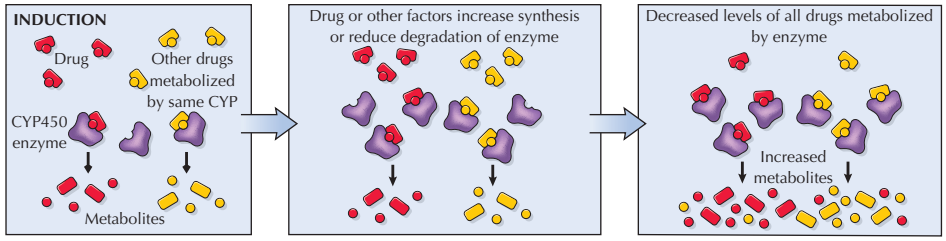
- A central opioid agonist, tramadol is commonly listed in the subclass of "other analgesics." In the athletic setting it may be used over a short course for moderate to severe pain associated with injury.
- Tramadol produces its analgesic effect by binding to μ-opioid receptors and also by weakly inhibiting norepinephrine and serotonin reuptake.
- Tramadol is metabolized in the liver extensively by the cytochrome P450 system, has the potential for many drug interactions, and has an active metabolite that increases the half-life of the drug in the body (Fig. 7-4).
- Because of its mode of action, there is possibility of dependency and abuse if prescribed long term. Tramadol remains a viable alternative for short-term use for pain management.

CORTICOSTEROIDS

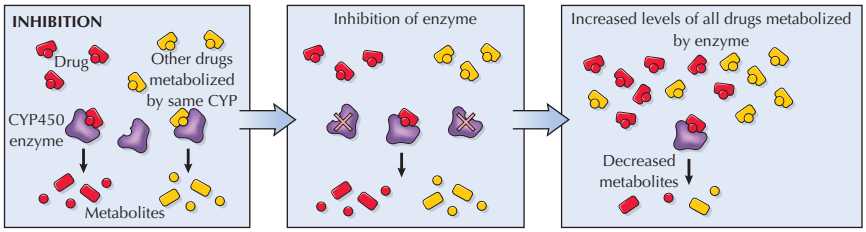
- Corticosteroids are potent anti-inflammatory agents that produce multiple glucocorticoid and mineralocorticoid effects. In the athletic setting their use should be judicious, given the numerous systemic effects and drug interactions.
- Corticosteroids are available in oral formulations or are administered via intra-articular injection for inflammation. Consider tapering oral doses if using for more than 5 to 7 days.
- Use of corticosteroids during the athletic season should be managed with subsequent modification of activities.

Mechanism of Action

- Corticosteroids are lipid-soluble molecules that work by binding to intranuclear receptors. They inhibit the chemotaxis of inflammatory cells and decrease lysosomal enzyme release and production of inflammatory mediators.
- Intra-articularly, corticosteroids decrease neutrophil migration into arthritic joints that are inflamed. They also reduce prostaglandin synthesis and decrease interleukin-1 secretion and subsequent leukocyte aggregation by the synovium.
- Studies have demonstrated the indirect increase of synovial fluid viscosity in a joint injected with corticosteroid prepara-



CYP	Inducers	Inhibitors
1A2	Smoking, charbroiled foods, cruciferous vegetables, insulin, modafinil, nafcillin, omeprazole, phenobarbital, primidone, rifampin	Amiodarone, anastrozole, cimetidine, ciprofloxacin, diltiazem, enoxacin, erythromycin, fluoroquinolones, fluvoxamine, grapefruit (juice), mexiletine, norfloxacin, ritonavir, tacrine, ticlopidine
2A6	Dexamethasone, phenobarbital	Methoxsalen, ritonavir, tranylcypromine
2B6	Cyclophosphamide, dexamethasone, phenobarbitol, phenytoin, primidone, rifampin	Efavirenz, nelfinavir, orphenadrine, ritonavir, thiotepa, ticlopidine
2C8/9	Dexamethasone, primidone, rifampin, secobarbital	Anastrozole, amiodarone, cimetidine, diclofenac, disulfiram, fluconazole, fluvoxamine, flurbiprofen, fluvastatin, isoniazid, ketoprofen, lovastatin, metronidazole, omeprazole, paroxetine, phenylbutazone, ritonavir, sertraline, sulfinpyrazone, sulfonamides, sulfamethoxazole, trimethoprim, troglitazone, zafirlukast
2C19	Barbituates, rifampin	Cimetidine, ketonconazole, modafinil, omeprazole, oxcarbazepine, ticlopidine
2D6	Dexamethasone, quinidine, rifampin	Amiodarone, buproprion, celecoxib, chlorpromazine, chlorpheniramine, cimetidine, clomipramine, cocaine, doxorubicin, fluoxetine, fluphenazine, fluvoxamine, haloperidol, lomustine, metoclopramide, methadone, norfluoxetine, paroxetine, perphenazine, propafenone, quinidine, ranitidine, ritonavir, sertindole, sertraine, terbinafine, thioridazine, venlafaxine, vinblastine, vinorelbine
2E1	Acetone, ethanol, isoniazid	Disulfiram, ritonavir
3A4	Barbituates, carbamazepine, dexamethasone, efavirenz, macrolides, glucocorticoids, modafinil, nevirapine, oxcarbazepine, phenobarbital, phenylbutazone, pioglitazone, phenytoin, primidone, rifabutin, rifampin, St. John's wort, sulfinpyrazone, troglitazone	Amiodarone, anastrozole, chloramphenicol, cimetidine, ciprofloxacin, clarithromycin, clotrimazole, danazol, delavirdine, diltiazem, erythromycin, fluconazole, fluoxetine, fluvoxamine, grapefruit juice, indinavir, itraconazole, ketoconazole, metronidazole, mibefradil, miconazole, nefazodone, nelfinavir, nevirapine, norfloxacin, norfluoxetine, omeprazole, paroxetine, propoxyphene, quinidine, ranitidine, ritonavir, saquinavir, sertindole, troglitazone, troleandomycin, verapamil, zafirlukast, zileuton



J. Perkins
MS, MFA

Figure 7-4 Metabolic Enzyme Induction and Inhibition.

tion via an increased concentration of hyaluronic acid in the joint.

Types

The original corticosteroid used for intra-articular injection was hydrocortisone (thus the familiarity with the term "cortisone" injection by many patients). Subsequent research helped develop formulations with longer durations of effect, mediated primarily by solubility (Table 7-1).

Uses

- The use of corticosteroids in athletes includes intra-articular injections for chronic and acute inflammation. It has been noted that pain relief in small joints is greater than that in larger joints.
- The lack of inflammatory markers in tendinopathy make the use of corticosteroids of controversial value in treatment. Tendon sheath injection in the treatment of tenosynovitis of the wrist and proximal biceps have been shown to have benefit. Some studies have also shown a role in the treatment of inflamed bursae.
- There may also be indications for use in improving motion in the treatment of adhesive capsulitis of the shoulder joint.

Table 7-1 DURATION OF EFFECT OF COMMONLY USED CORTICOSTEROIDS

Generic name	Average duration of effect (days)
Methylprednisolone sodium succinate	4
Dexamethasone sodium phosphate	6
Triamcinolone diacetate	7
Methylprednisolone acetate	8
Dexamethasone acetate	8
Hydrocortisone acetate	8
Betamethasone acetate	9
Prednisolone tebutate	10-14
Triamcinolone acetonide	14
Triamcinolone hexacetonide	21

Adapted from Snibbe JC, Gambardella RA: Clin Sports Med 24:1-3, 2005.

Adverse Reactions

- The potential side effects of corticosteroids can be significant. Local effects include increased risk of tendon and ligament rupture secondary to inhibition of collagen synthesis. Subsequently, injection into sites such as the Achilles or patellar tendons is contraindicated.
- Other potential adverse effects associated with injection of corticosteroids include skin atrophy at the site of injection, infection of the joint injected or injection site, and the potential for calcification of the synovium.
- Systemic effects of both oral and intra-articular administration of corticosteroids include inhibition of the hypothalamic-pituitary-adrenal axis at higher doses. A taper is recommended to minimize the risk of adrenal insufficiency with prolonged or repetitive use.
- In diabetics, transient worsening of glycemic control may result.
- Interestingly, at low doses, intra-articular injection of corticosteroid may have beneficial anti-inflammatory effects on distant joints.

VISCOSUPPLEMENTATION

Background

- The use of intra-articular viscosupplementation, or hyaluronic acid (HA), injections in pain management in lower extremity osteoarthritis (specifically knee and more recently ankle) has been established.
- A normal human knee contains about 5 to 8 mg of hyaluronic acid in 2 mL of synovial fluid.
- In an arthritic knee the amount of HA is diminished, reducing the viscoelastic property of the synovial fluid. This in turn increases the stress and shear forces experienced by the articular surface and may lead to further damage.

Mechanism of Action

- Although the exact mechanism of action of intra-articular viscosupplementation is not known, studying the characteristics of HA helps to shed light on an answer to this question.
- At low shear forces in the joint, HA exhibits high viscosity and low elastic properties. Conversely, at high shear forces, the properties of HA are the opposite, demonstrating low viscosity and high elasticity.
- HA has both anti-inflammatory and analgesic properties.
  - It has been shown to inhibit macrophage phagocytosis and neutrophil adherence.
  - It also reduces release of arachidonic acid (a precursor of inflammatory mediators) from fibroblasts in the synovium.
  - The analgesic effects of HA include possible direct inhibition of pain receptors.
  - HA purportedly also indirectly binds substance P, thereby decreasing pain signals.

Properties

- HA demonstrates several properties that lead to it effectiveness and tolerability.
- HA displays a remarkable lack of immunogenicity, helping to limit the chance of a local reaction after intra-articular injection.
- In the joint, HA exhibits passive diffusion in synovial fluid and a prolonged half-life within the synovium.

Types

- At present there are five different brands of viscosupplementation available for injection in the United States (Table 7-2).
- Each has a differing profile of molecular weight and preparation of purified sodium hyaluronate, and maintains a neutral pH.
- All have relative contraindications to use in patients with avian or avian-derived product allergies, except Euflexxa, which is a bioengineered, fermentation-derived product.

Adverse Reactions

- Side effects of intra-articular viscosupplementation with HA occur at a rate of about 1% per injection.
- Local reactions such as warmth, swelling, and pain can last 1 to 2 days.
- Granulomatous inflammation arising within 48 hours after injection has occurred with Synvisc. This adverse effect typically has been shown to resolve in 1 to 2 weeks.

Clinical Results

- The clinical efficacy of intra-articular viscosupplementation has been well studied.
- Most studies have been done in the treatment of knee arthritis and the results have been variable. Improvement in pain and functionality versus placebo has been demonstrated in several studies.

Table 7-2 BRANDS OF HYALURONIC ACID (HA) VISCOSUPPLEMENTS

Brand name	Molecular weight (kDA)	Comments
Hyalgan	500-730	Studies with 5 weekly injections show decrease in visual analog scale pain scores at 26 weeks Additional studies with 3 weekly injections show benefit over placebo at 60 days
Synvisc	6000	Cross-linked mixture of gel and fluid formulation of HA Studies with 3 weekly injections show benefit versus placebo out to 26 weeks
Supartz	620-1170	Improvement in Lequesne index and VAS pain scale with 5 weekly injections followed out to 13 weeks
Euflexxa	2400-3600	Bioengineered hyaluronic acid that is not derived from purified rooster comb Series of 3 weekly injections showed improvement of VAS WOMAC index similar to that of Synvisc at 12 weeks
Orthovisc	1000-2900	Improvement in WOMAC pain scores seen up to 22 weeks after 3 weekly injections

- The salutary effects may be similar to or better than intra-articular corticosteroid injection in the intermediate term.
- Recently, smaller studies have shown benefit in the treatment of ankle arthritis. Larger scale studies involving this and other joints, including the shoulder, are currently under way.
- There is plenty of scope for further investigation of HA viscosupplementation.
  - Though most of the current literature focuses on studies in older adults, future studies should look into the question of a potential of a chondroprotective effect in younger athletes with early arthritis.
  - In vitro studies suggest that early use HA viscosupplementation in low-grade osteoarthritis may slow progression of disease.

SUMMARY

The management of pain and inflammation in the athlete can be a challenging task. Careful consideration of side effects, both adverse and in some cases beneficial, drug interactions, and possible contraindications should be taken into account when making a choice. Although NSAIDs have long been a favorite in the armamentarium of the sports medicine practitioner, limiting use to appropriate situations and shorter durations of therapy is prudent. Other options for pain management and different preparations and modes of administration should be remembered, which will give athletes the chance for maximal therapeutic effect and a quick and safe return to sport.

RECOMMENDED READINGS

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SECTION

II

Conditioning

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The Pediatric Athlete

8

Holly J. Benjamin

GENERAL PRINCIPLES

- The pediatric athlete can be any child of any age (although usually younger than 18 years old) who participates regularly in sports activities.
  - Type of activity, skill level, and motivation for sport participation varies greatly at different ages and different levels of maturity. Therefore, it is best to understand young athletes in the context of their chronologic age, developmental stage, and physical maturity, coupled with an assessment of the nature and level of sports participation.
  - Health care professionals face challenges at both ends of the physical activity spectrum: the sedentary obese child who faces a lifetime of morbidity versus the highly competitive, overzealous, potentially undernourished youth athlete who is at risk for a myriad of overuse injuries associated with excessive exercise.
- The key for all youth of today is to find balance.
  - A successful athlete will lead a healthy lifestyle and can successfully integrate exercise, nutrition, and recreational pursuits.
  - The ultimate goal of youth sports participation should be the promotion of lifelong physical activity, pursuit of recreation, and enjoyment of the challenge of competition.
  - The physician's role is to understand both the child's and the parent's motivation for sport participation, to treat injuries early and comprehensively, and to capitalize on opportunities to educate parents, athletes, and coaches on healthy athletic participation and sports safety.

BENEFITS OF PHYSICAL ACTIVITY

- What are the positive effects of exercise in children?
  - Helps control weight
  - Lowers blood pressure
  - Raises HDL "good" cholesterol
  - Reduces risk of diabetes
  - Improves self-esteem
- How does one encourage physical activity?
  - Limit or reduce sedentary time (television, computer, video games, telephone) to 30 minutes per day.
  - Find fun activities that children enjoy.
  - Encourage parent role models.
- The following are the American Heart Association recommendations for physical activity:
  - All children age 2 and older should participate daily in 30 minutes of moderate intensity physical activities that are developmentally appropriate and varied.
  - Multiple small periods of activity, such as two 15-minute periods or three 10-minute periods of exercise, are acceptable alternatives.

CHILDHOOD OBESITY

- Excess weight is the most common health problem facing U.S. children.
- The obesity prevalence rate in children is 11% to 22%.
- The obesity rate has doubled in the last 20 years.
- Obesity in children is increasing exponentially at an epidemic rate, particularly in economically disadvantaged areas and minority populations.

Age Range: Preschool through High School

- Preschoolers spend approximately 11% of their time in vigorous activities, 60% in sedentary activities.
- Preschoolers spend an average of 3 to 5 hours per day watching TV.
- Every hour of television is associated with a 2% increase in obesity risk.

Risks of Adult Obesity

- Fifty percent of children who are obese at age 6 are likely to remain obese into adulthood.
- Eighty percent of children who are obese at age 10 are likely to remain obese into adulthood.
- Additional risk is associated with concurrent parental obesity.

Body Mass Index (BMI)

- Body Mass Index (BMI) = [weight (kg)] ÷ [height $(m)^2$]
- A child with a BMI in the 85th to 95th percentile is considered overweight and at risk for obesity.
- A child with a BMI in the 95th percentile and above is considered obese.
- Annual BMI calculation is recommended for children during routine and sports physicals, thus it can be followed longitudinally. The pediatric growth charts based on age and gender include BMI and are available online (www.cdc.gov/growthcharts).

Causes of Childhood Obesity

- Energy intake is greater than energy expenditure.
- Endocrine, hormonal, and genetic syndromes can each cause or contribute to obesity in children.

Complications of Childhood Obesity

Any and all organ systems in the body can be affected by childhood obesity. Cardiac, orthopedic, endocrine, gastrointestinal, respiratory, and neurologic are among those most often affected.

Treatment Recommendations

- Nutrition interventions include education, changes in advertising, healthy school lunches, and adequate, varied healthy food choices in the home environment.
- Exercise recommendations include increased recreational activities, organized sports participation, preservation of adequate physical education time in school, and decreased sedentary screen time (computer, television).

CHANGING TRENDS IN EXERCISE PATTERNS

- As more children participate in organized athletics at younger ages, the incidence of overuse injuries is increasing. It is estimated that 30 million to 45 million youth ages 6 to 18 participate in athletics (two thirds in organized sports, one third in recreational). Many children participate in year-round same-sport activities or on multiple sports teams simultaneously.
- Overtraining can lead to burnout and/or increased injuries, which can eventually cause a young athlete to stop participating in athletic activities, sometimes permanently. Parental influence is a critical confounding factor that affects youth participation in sports and must be addressed by physicians who care for pediatric athletes.

Risks of Overuse Injuries

- Intrinsic factors include growth, anatomic alignment, muscle-tendon imbalance, flexibility, and conditioning.
- Extrinsic factors include training errors, environment, and equipment.

- Recommendations for overuse injury prevention include the following:
  - Limit one sporting activity to 5 days per week.
  - Provide one day of rest from organized activity.
  - Take 2 to 3 months off per year from a single sport.
  - Vary workout routines to maintain interest and fun.
  - Educate athletes and parents about wellness and nutrition.
  - Monitor for warning signs of injury.
  - Monitor special events such as tournaments and showcases for fatigue, pain, and alterations in sports performance.

Sport-Specific Issues: Endurance Event Competition

- Endurance events such as marathons and triathlons are increasing in popularity among youth athletes.
- The American Academy of Pediatrics has stated that "triathlons for children and adolescents are reasonably safe as long as the events are modified to be age appropriate."
- Modifications include shorter duration of activities and conservative guidelines for safety and environmental conditions such as exercising in heat and humidity.
- Training regimens are often altered from traditional plans; alterations include lower weekly mileage, gradual increases in training, and extra attention to hydration and nutrition.

ORGANIZED SPORTS FOR CHILDREN AND PREADOLESCENTS

- Changing trends include a movement away from spontaneous, unstructured activity that allows imagination, enjoyment, and motor skill development.
- Organized sport participation is heavily influenced by goals and expectations of parents and coaches. More activities are structured that de-emphasize spontaneous interaction of youth with various skill levels and motivation.
  - The reactions and feedback from parents and coaches have a strong influence on the attitudes and confidence in preadolescent athletes.
  - If the focus is placed on skill development, cooperative play, and having fun, athletes seem to have a greater enjoyment of sports participation.
- Advantages of organization include the establishment of rules for participation, equity in matching competitors at similar skill levels, definition of readiness-to-play criteria, and fairness in the establishment of teams to promote safe participation.

ANATOMY AND PHYSIOLOGY ISSUES: GROWTH AND MATURATION

- **Concerns about the potential negative effects of athletic competition on growth and maturation have existed for many years,** especially in light of the increasing trend of intense competition at younger ages.
  - The demands of sports require a certain level of physical and psychological maturity in order to successfully participate. Feelings of insecurity, frustration, and failure may cause young athletes to quit because of burnout or inability to perform up to expectations.
  - While young athletes are struggling to master advanced sport-specific skills, unfortunately their coaches are often less experienced and less educated in appropriate training techniques. These barriers can negatively affect a young athlete's enjoyment and participation in his or her sport(s) of choice.
- **One of the greatest challenges facing health care professionals in sports medicine today** is the consideration of the neurologic, cognitive, somatic, and psychological interdependent processes and the effects of each on the health and well-being of the modern pediatric athlete.
  - An understanding of the fundamental principles of normal child and adolescent growth and development is essential in providing quality health care for young sports participants.
- **Growth and maturation is a natural, fundamental, continuous process.**
  - Growth and maturation occur sequentially in all children, with achievement of the same milestones in the same order.
  - The rate of progression varies greatly and even in the athletic population seems predominantly genetically regulated.
  - Neuropsychological development often does not parallel physical development.
  - Growth refers to an increase in size of the body and its parts, including stature, body systems, and body composition.
  - Maturation refers toward a biologically mature state of skeletal, sexual, and somatic development with varied timing and tempo.
  - Neurodevelopment is culturally mediated and is the acquisition and mastery of behavioral competence.
  - Quantitative milestones are easy to measure by the number of skills performed.
  - Qualitative milestones are harder to measure because they reflect the mastery of specific skills.

Neurodevelopmental Domains

Motor: fine and gross motor, strength, and endurance
Visual: acuity, discrimination, tracking
Cognitive: attention, alertness, memory, comprehension, and solving complex problems or multiple tasks simultaneously
Language: receptive and expressive
Auditory: hearing acuity and processing, sound discrimination, and auditory cues
Emotional and psychological: relationships with teammates and coaches, and regulation of emotions
Motor: fine and gross, visual-spatial discrimination, temporal sequencing, proprioception, sport-specific motor adaptive skills, muscular strength and endurance, reaction time

Motor Developmental Milestones at Various Age Groups

- Understanding sequential developmental milestones from infancy through young adulthood is essential in successfully caring for the pediatric athlete who is constantly in a state of growth and developmental evolution.
- It is difficult if not impossible to "skip" major neuromuscular milestones during periods of growth; however, the rate at which young athletes often progress through developmental milestones can be accelerated in some cases.
- Accelerated motor development can be problematic if the young athlete is not psychologically or emotionally ready to fully function at this new level of expectation and skill.
- Health care professionals should be familiar with the sequence of skill acquisition in a logical pattern that is predictable among various young athletes.
- **Preschoolers (age 4 to 6 years)**
  - Ride bike without training wheels.
  - Hop six times on one foot.
  - Catch a small ball thrown from 10 feet.
  - Run, gallop, and skip using alternating feet.
  - Broad jump up to 3 feet.
  - Throw a ball with a shift of their bodies at a target.
  - Move from parallel play to interactive play with others.
- **Middle Childhood (age 6 to 11 years)**
  - Gender differences can be seen.
  - Girls excel at hopping, skipping, catching, and balance.
  - Boys excel at striking objects, jumping (vertical and long), kicking, and throwing, and can run faster.

Implications for Sport Participation in Youth Athletes

- Coach and parent reaction with appropriate feedback to young athletes is crucial in sport development.
- Confidence, self-esteem, and body awareness are all developing.
- Young athletes should be taught to think along the lines of "I'm learning and improving" rather than "I can or can't."

Gender Differences

- In the preadolescent period, there is little difference in strength, power, and endurance between males and females.
  - Females are consistently more flexible.
  - Males are consistently better throwers.
- Power and maximal oxygen uptake ($\dot{V}O_2$ max) increase linearly with age until adolescence when it accelerates. However, in adolescent males the accelerated rate increases far exceed those seen in adolescent females and are in part a result of increased muscle mass in males.

Neuropsychological and Emotional Readiness

- Peer relationships with teammates involve the ability to take turns, attend to the game, focus, and participate in teamwork.
- The coach-athlete relationship requires the ability to follow rules, understand strategies, and control emotions.

Implications for Organized Sports Participation

- Physical maturation is necessary to master sport-specific skills.
- Neurodevelopmental maturation allows the functional integration of multiple skills simultaneously to meet the demands of competition.
  - **Motor:** for example, a soccer player needs to run and kick simultaneously in a coordinated fashion
  - **Visual:** monitor for position of teammates and defenders
  - **Auditory**: process instructions from coaches
  - **Language**: communicate with teammates and coaches
  - **Cognitive**: problem solve and carry out sport strategies
  - **Emotional**: possess the ability to process a variety of emotions such as excitement, anxiety, elation of winning, and frustration with losing
- Athletes that are competing in sports at levels above their neurodevelopmental abilities will be more likely to experience negative feelings such as frustration, anger, and lower confidence and self-esteem. They will be less likely to have fun and to enjoy the overall sports participation experience. Drop-out as well as injury rates may be higher in these situations.

Psychological Concerns

Common mistakes that parents make that negatively influence youth athletes include the following:

- Vicariously participate in their sport(s) of choice through their children.
- Push their children to "overtrain."
- Criticize youth athletic performance.
- Promote a "win at all costs mentality."
- Allow early sport specialization, often in the parent's sport of choice.
- Serve as parent-coaches who either favor or disfavor their own children.
- Refuse to educate themselves as to warning signs of injury.
- Fail to monitor their children for risks of injury.

Sport Safety

The following modifications are appropriate for youth elementary-school age participants:

- Use smaller fields and courts.
- Use size- and weight-appropriate equipment.
- Shorten duration of games and practices.
- Have a smaller number of participants playing at the same time.
- Make the time during sport participation to teach and enforce rules and safety.
- Promote equal playing time and rotate positions.
- Avoid score keeping and win-loss records; reinforce "fun" as the goal of sport participation.

YOUTH STRENGTH TRAINING

- Muscle strength development has been a topic of debate for several decades.
- Parents are often looking for a competitive edge for their children, or for ways to deal with overweight kids.
- Data is limited that allow evidence-based recommendations for patients; more work is being done in this area and knowledge continues to grow and beliefs continue to change.

Definitions

- *Strength training* is a broad term that is defined as the use of resistance methods to increase one's ability to exert or resist force. Machines, free weights, and/or a patient's body weight can be used.
- *Weightlifting* and *powerlifting* are competitive sports that contest maximum lifting ability.
  - *Weightlifting* involves the clean-and-jerk and the snatch.
  - *Powerlifting* involves the squat, the bench press, and the dead lift.
- *Bodybuilding* is an aesthetic sport that involves weight training but not competitive lifting.

Where Are We Today?

- American Academy of Pediatrics (AAP) 1983: "Pubertal boys (pubic hair stage 1 or 2) do not significantly improve strength or increase muscle mass in a weight training program because of insufficient circulating androgens."
- AAP 2001: "Studies have shown that strength training, when properly structured with regard to frequency, mode, intensity, and duration of program can increase strength in pre-adolescents and adolescents."
- AAP 2008: "Proper resistance techniques and safety precautions should be followed so that strength training programs for preadolescents and adolescents are safe and effective."

How Much, How Soon?

- The topic of weight training for young people remains controversial.
- The AAP and the American Orthopaedic Society for Sports Medicine (AOSSM) recommend that children and adolescents refrain from weightlifting, powerlifting, and bodybuilding until skeletally mature mostly because of the lack of data demonstrating safety.
- The National Strength and Conditioning Association (NSCA) supports the sports of weightlifting, powerlifting, and general strength training for children and adolescents.
- Why the controversy?
  - The NSCA cites recent research done by Hamill and Faigenbaum that demonstrate low injury rates and a progressive learning curve.
  - It appears to be necessary to master basic sport-specific needs and skills before the body can progress to attempt more highly technical maneuvers. In essence, these re-

searchers feel that the skills required for weightlifting and powerlifting make it almost impossible to lift too much weight too soon.
- More research is needed in these areas and is ongoing.
- It is important to note that many sports use intrinsic strength techniques to perform sport-specific exercises, such as the young gymnast who tumbles and vaults bearing her entire body weight on her hands and wrists.

Anatomy and Physiology: Training Effects on Strength

- Strength gains are a result of neuromuscular adaptation in preadolescents. Muscle hypertrophy is not seen in preadolescents but is evident during puberty.
- The effects of strength training on body composition can be evident at all ages and have a favorable effect on improving lean body mass, particularly in obese children.
- Increases in neuronal activation, intrinsic muscular adaptations, and improvements in motor coordination (learning) all seem to play a role in strength development in childhood.
- Faigenbaum and colleagues demonstrated strength gains with as little as twice-a-week training sessions in a group of 9-year-old youths who weighed approximately 40 kg and who lifted one-half their body weight for leg extension and chest press exercises and 1½ times their body weight for leg press exercises.
- No long-term studies exist on the effects of preseason resistance training on improved sports performance in children.
- It appears that strength gains made during training can be lost during periods of rest or "detraining." These must always be evaluated in the context of naturally occurring strength gains that are associated with normal growth and development.
- Good nutrition and age-specific activity guidelines should be followed. Strength training has no known negative effects on growth.

Risk Factors for Injury

- The greatest injury risk to children who perform any type of strength training is a lack of proper supervision.
- Other risks include using improper technique, improper size equipment, or improper weight and repetitions.
- Types of injuries associated with weight training include acute and overuse.
  - Acute injuries include fractures, muscle-tendon injuries, and, rarely, head injuries.
  - Overuse injuries include sprains, strains, and growth plate injuries; overuse injuries particularly involve the back and shoulder.
  - No reported cases of growth plate fractures are reported to have occurred in appropriately supervised settings.

Office Guidelines for Strength-Training Programs

- Program design considerations should include education on proper technique, progression, function, and fun.
- Strength training for kids should always be incorporated into an exercise program with aerobic conditioning, flexibility, and sports participation.
- The following should be kept in mind when creating a strength-training program:
  - Body weight exercises should be done first.
  - Perform 2 to 3 sessions per week, 2 to 3 sets, 10 to 15 repetitions of each exercise.
  - Vary program and add weight gradually in 10% increments.
  - Use child-sized equipment for preadolescents, such as smaller 1-pound plates, small dumbbells, and/or Thera-Bands.
  - Proper supervision at all times is essential.
  - Emphasize correct form and technique.
  - Use single joint and multijoint activities and train antagonistic muscle groups equally.
  - Design an individualized program, vary it regularly, and keep workout logs to monitor progress.

COMMON PEDIATRIC SPORTS INJURIES RELATED TO GROWTH AND DEVELOPMENT

Physeal Fractures

Overview: Acute musculoskeletal injuries unique to a young athlete are growth plate fractures. An injury at the end of a long bone in a skeletally immature athlete is a physeal (growth plate) fracture until proven otherwise. The bone and cartilage that makes up the physis is relatively weaker than the surrounding ligaments, tendons, joint capsule, and other soft tissue structures, thus excessive force applied to the musculoskeletal system will likely result in a physeal fracture.

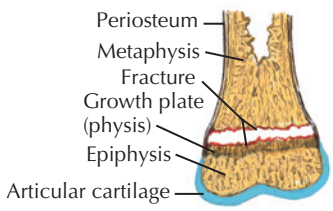
Presentation: The highest rates of physeal fractures are seen during the growth spurt of adolescence and are usually associated with concentric, eccentric, or shear forces placed on the physis at the time of injury. The Salter-Harris (SH) Classification is based on the pathoanatomy of the fracture and the radiographic appearance of the bone itself (Fig. 8-1):
- Type I is a transphyseal injury.
- Type II is a transphyseal injury with metaphyseal extension.
- Type III is a transphyseal injury with epiphyseal extension into the joint space.
- Type IV has extension from the epiphysis through the physis and into the metaphysis.
- Type V fractures are rarely seen and involve a crush injury to the physis itself.

Physical examination: Most common finding on examination is tenderness at the ends of long bones over the physis. Swelling, ecchymosis, bony deformities, inability to bear weight, or decreased resistance on strength testing may be associated.

Diagnosis: Accurate diagnosis requires a high index of suspicion and a basic knowledge of anatomy in skeletally immature individuals.
- Radiographs are required; although most Salter-Harris Type I injuries are diagnosed clinically because radiographs are usually inconclusive.
- Additional imaging studies may be necessary such as bone scan, magnetic resonance imaging (MRI), or computed tomography (CT) scan in certain situations where the diagnosis is in question or the patient is not responding to clinical treatment as expected.

Treatment: Treatment of physeal fractures depends on the location and classification of the injury as well as the degree of displacement of the fracture fragment.
- Most require casting with or without a closed reduction for displaced fractures.
- Other fractures with significant displacement and/or instability of fracture fragments will require an open reduction, internal fixation (ORIF).
- Some SH-I fractures, such as SH-I distal radius and fibular fractures, can be treated with splints.
- Although there is less stiffness postimmobilization, noncompliance with splinting can be problematic.

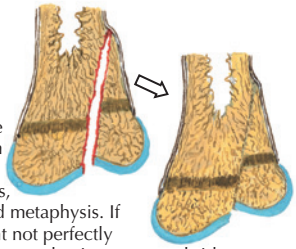
Prognosis and return to play: Prognosis varies but in general is good for SH-I to SH-III's. The biggest risk factor is that of a partial or complete growth arrest with malalignment and/or biomechanical issues post injury.
- **All physeal fractures will require additional monitoring for 6 to 12 months post injury to adequately assess continued long bone growth post injury**.



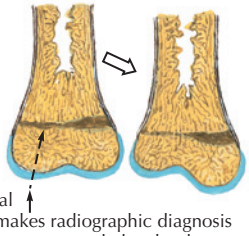
Type I. Complete separation of epiphysis from shaft through calcified cartilage (growth zone) of growth plate. No bone actually fractured; periosteum may remain intact. Most common in newborns and young children.

Type II. Most common. Line of separation extends partially across deep layer of growth plate and extends through metaphysis, leaving triangular portion of metaphysis attached to epiphyseal fragment.

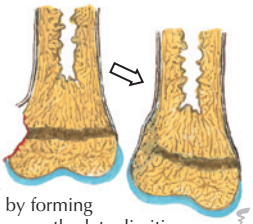
Type III. Uncommon. Intraarticular fracture through epiphysis, across deep zone of growth plate to periphery. Open reduction and fixation often necessary.



Type IV. Fracture line extends from articular surface through epiphysis, growth plate, and metaphysis. If fractured segment not perfectly realigned with open reduction, osseous bridge across growth plate may occur, resulting in partial growth arrest and joint angulation.



Type V. Severe crushing force transmitted across epiphysis to portion of growth plate by abduction or adduction stress or axial load. Minimal or no displacement makes radiographic diagnosis difficult; growth plate may nevertheless be damaged, resulting in partial growth arrest or shortening and angular deformity.



Type VI. Portion of growth plate sheared or cut off. Raw surface heals by forming bone bridge across growth plate, limiting growth on injured side and resulting in angular deformity.

Figure 8-1 Injury to Growth Plate (Salter-Harris Classification, Rang Modification).

- Return to play varies from approximately 3 weeks to 12 weeks or more. In general, immobilization lasts 3 to 6 weeks because physeal injuries heal quickly.
- General return-to-play criteria should apply, including an assessment of range of motion, strength, and ability to perform sport-specific exercises with or without the use of a brace or other assistive device.

Apophyseal Injuries

Overview: Apophyseal injuries include both acute and overuse injuries and represent a noninflammatory, stress injury to growth plates. Acute injuries are avulsion fractures at the apophyseal growth plate where a muscle-tendon unit attaches. Overuse injuries are far more common and present usually in a similar fashion as tendonitis with growth plate involvement.

Presentation: Concentric, eccentric, and shear forces create excessive biomechanical force at the apophysis that can result initially in a painful acute inflammatory condition (apophysitis). Excessive workload of constant repetitions of a sport-specific maneuver can lead to a more chronic overuse tendinopathy presentation. Many injuries are predictable based on the sport performed and the predicted stresses to certain areas of the body. Common types of apophyseal injuries include the following:

- "Little League Elbow" involves the medial epicondyle (flexor-pronator attachment).
- Osgood-Schlatter disease involves the tibial tuberosity (patellar tendon attachment).
- Sinding-Larson-Johannson syndrome (SLJ) involves the inferior pole of the patella (patellar tendon).
- Iselin's disease involves the base of the fifth metatarsal (peroneus brevis attachment).
- Sever's disease ("soccer heel") involves the calcaneus (Achilles tendon attachment).
- Hip/pelvis apophysitis such as anterior superior iliac spine (ASIS-sartorius), anterior inferior iliac spine (AIIS-rectus femoris), ischial tuberosity (hamstrings), iliac crest (obliques and gluteus), and greater/lesser trochanter are most common.

Physical examination: Tenderness to palpation at the tendon and the apophysis is common. Perform a general assessment of biomechanical and genetic predisposition (e.g., check for severe pes planovalgus feet, genu valgum, benign hypermobility, etc.)

Diagnosis: Diagnosis is often clinical, based on a high index of suspicion. Radiographs are helpful to evaluate for acute injuries, malalignment, and growth disturbances but are often normal. Additional imaging studies are useful adjuncts for more chronic, severe, and persistent cases.

Treatment: Acute injuries may require a brief period (2 to 4 weeks) of immobilization but can require ORIF to correct significant displacement (e.g., avulsion of the tibial tuberosity in the knee). Overuse injuries are treated in a similar fashion as tendonitis/tendinosis with relative rest and guided rehabilitation. Rehabilitation focuses on stretching to minimize forces at apophysis. A biomechanical assessment of a sport-specific exercise may aid in the prevention of recurrences.

Return to play: When pain has resolved and the athlete can perform the necessary sport specific maneuvers. A guided gradual resumption of normal sports activities is usually necessary to prevent frequent recurrence of symptoms.

Osteochondritis Dissecans (OCD)

Overview: The term *osteochondritis dissecans* is attributed to Konig in 1883 and is based on the theory that inflammation contributed to subchondral necrosis of bone and cartilage (Fig. 8-2). Trauma, ischemia, ossification defects, and genetic abnormalities all likely contribute to the pathophysiology. There is a juvenile form and an adult form, distinguished by closure of the physes. The role of subchondral bone in providing cellular and humoral factors for healing contributes to the multilayered organization of articular cartilage and affects its ability to heal with conservative management. The three most common areas in pediatric athletes where OCDs occur are the knee (femur), elbow (capitellum), and ankle (talus).

Presentation: Juvenile OCDs (JOCDs) are more common in athletes, and 40% to 60% of JOCDs have a preceding history of trauma. Vague symptoms such as recurrent pain, crepitus, decreased range of motion, joint tenderness, and/or joint effusions may be present. More severe or advanced cases may include mechanical symptoms such as catching or locking.

Physical examination: Tenderness may be reproduced at the site of the OCD lesion so the medial femoral condyle, the capitellum, or the talar dome should be palpated as indicated clinically. The presence of a nontraumatic joint effusion in an athlete is highly suspicious for an OCD lesion as is the presence of me-

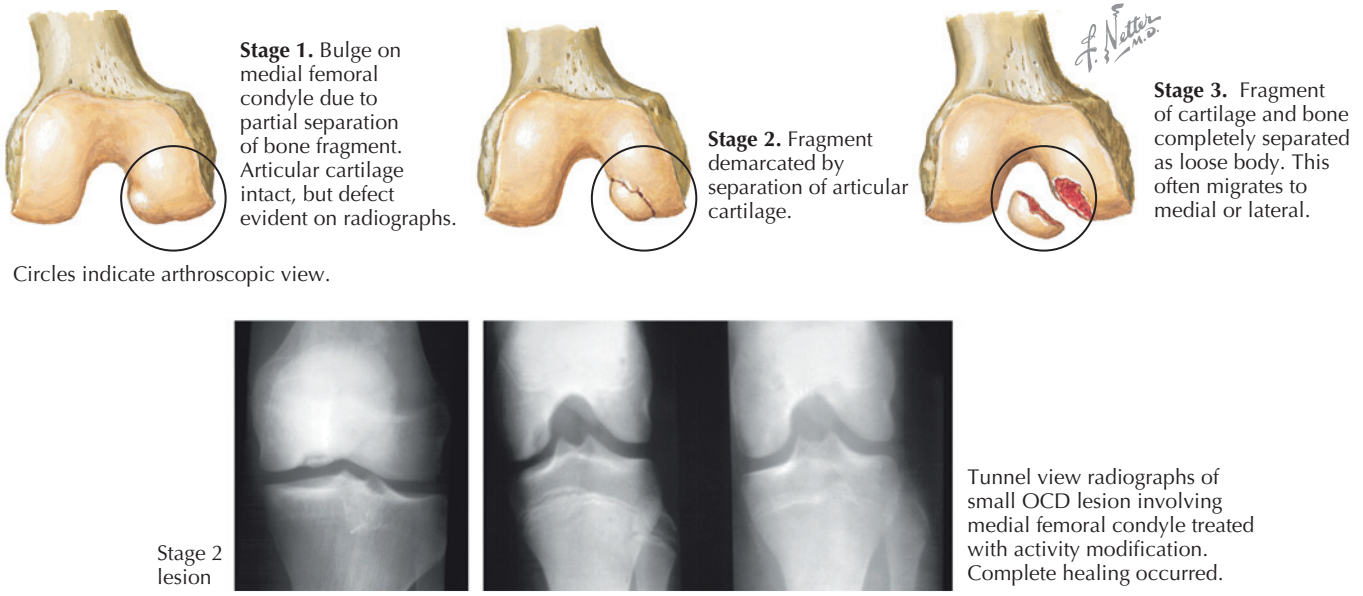


Figure 8-2 Osteochondritis Dissecans.

chanical symptoms. Gait abnormalities may be seen in knee OCD patients and include walking with the tibia externally rotated to decrease pressure on the lesion.

Diagnosis: Often made on plain radiographs.

- **Knee:** AP, notch or skier's, lateral, and sunrise views should be obtained. Often, the lesion in the posterior aspect of the medial femoral condyle is evident only on the notch or skier's view.
- **Elbow:** AP, lateral, and oblique views should be obtained with consideration of comparison views.
- **Ankle:** AP, lateral, and mortise views are necessary. The anterolateral talar dome and the posteromedial talus are the common areas of occurrence in the ankle.
- In general, up to 50% of OCD lesions can be missed on plain radiographs. MRIs substantially improve the ability to image the lesion and also allow for staging of joints with OCDs.
- MRI staging of OCDs
  - Stage I: thickening of articular cartilage and low signal changes.
  - Stage II: articular cartilage is disrupted with low signal rim behind fragment.
  - Stage III: articular cartilage is widened and high signal changes behind fragment indicate instability.
  - Stage IV: loose body (displaced intra-articular fragment).

Treatment: Varies, depending on the age of the patient, skeletal maturity, and location and size of the lesion. Conservative treatment with rest and decreased mechanical forces is often effective in 50% to 91% of stage I to II lesions that are stable in skeletally immature patients. Indications for surgical referral to an orthopedic sports medicine surgeon include persistent symptoms such as recurrent effusions, instability, chronic pain, ongoing mechanical symptoms, concomitant injury, loose bodies, and skeletally mature patients.

Spondylolysis

Overview: Studies indicate that 10% to 30% of adolescent athletes suffer from low back pain. In young athletes, back pain that persists for more than 3 weeks should be evaluated for the presence of spondylolysis (Fig. 8-3). Spondylolysis can be acute (pars stress fracture) or chronic. Spondylolisthesis refers to a subluxation or anterior displacement of a vertebral body. Congenital spondylolysis patients have radiologic evidence by age 5 to 6, but symptoms are often delayed until adolescence with athletes presenting at ages 12 to 18. Both unilateral and bilateral pars defects are seen. In general, bilateral defects have a higher complication rate, slower healing times, and are associated with a risk of slippage resulting in a concurrent spondylolisthesis.

Presentation: Classically, the athlete presents with activity-related back pain that is worse with extension. Neurologic symptoms are rare as are nighttime symptoms.

Physical examination: There may be tenderness to palpation at the level of the fractured pars. Paraspinal muscle tenderness may be present. A "stork" test is a single leg balance spine extension test that reproduces back pain when positive. Neurologic symptoms should not be present.

Diagnosis: Standard radiographs are AP, lateral, and obliques. A "Scotty dog" sign is a fracture of the pars seen best on the oblique views. The lateral view will demonstrate the presence or absence of a "slip" or spondylolisthesis.

- Radiographs have a low sensitivity for detection of spondylolysis.
- SPECT bone scans are highly sensitive and are the gold standard in diagnosis.
- A reverse angle thin-cut CT scan is best at evaluating healing or sclerosis versus nonunion of pars defects. If symptoms are localized, CT may be used in diagnosis.
- MRI is best used in the evaluation of disc pathology. The sensitivity is lower for the detection of pars defects and many false negative results are seen, which can lead to delays in diagnosis and treatment. Specialized institutions are increasingly using MRI to assess pars injuries.

Treatment: Activity modification with decreased sports participation until pain free. Guided rehabilitation that emphasizes core strength, posture, and hamstring/hip flexor flexibility. Sport participation is usually restricted for 6 weeks. Bracing can be an important part of the treatment for pars defects, especially if athletes are not pain free with rest from activities. Bracing type is controversial as is the length of time required (23 hours per day for 6 weeks to 6 months is the most common):

- Boston brace is the gold standard. It is a rigid lumbosacral orthosis (LSO).
- Warm-N-Form orthosis is useful and cheaper.
- Semirigid and soft lumbar orthoses are sometimes useful for less severe unilateral cases and when compliance is poor with the Boston brace or Warm-N-Form.

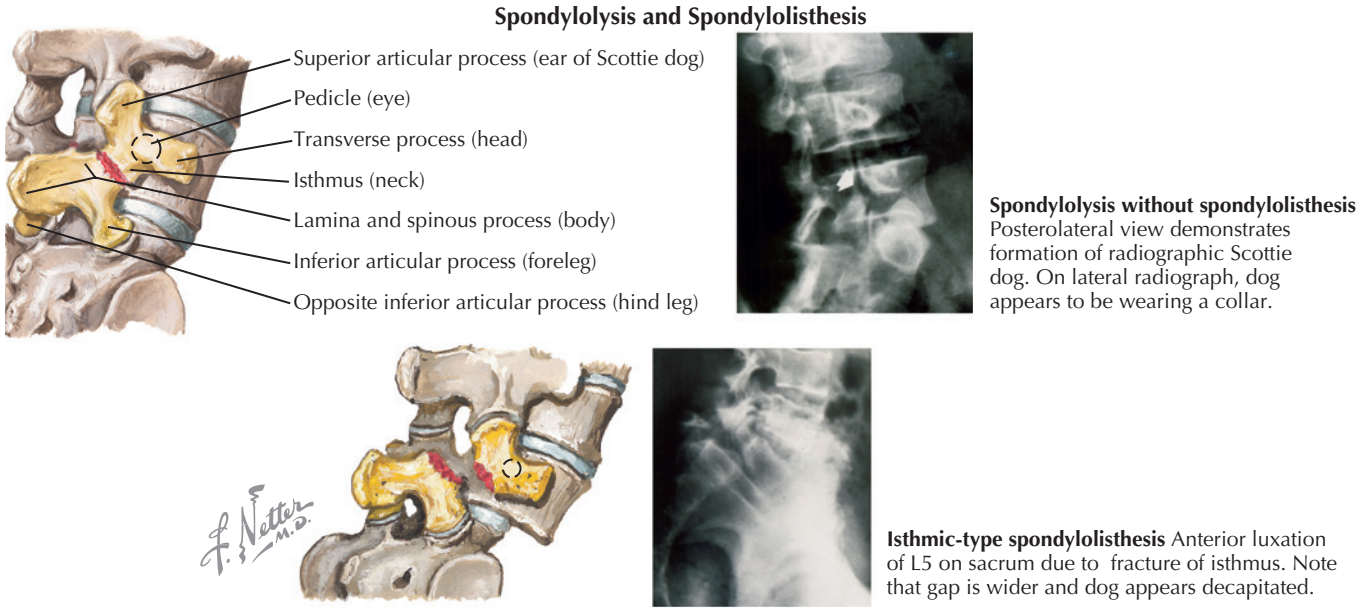


Figure 8-3 Spondylolysis and Spondylolisthesis.

- Braces are unlikely to significantly limit extension; rather, efficacy may be achieved via a proprioceptive mechanism.

Prognosis: Unilateral pars defects have a better prognosis for healing without long-term complications than do bilateral defects. Nevertheless, there is a low rate of bony healing (50%). Twenty-five percent show a fibrous union or partial bony healing and 25% result in nonunions as seen on follow-up CT scans.

Return to play: Most athletes return to their previous level of sport participation. Athletes should be pain free. Some can return to participation in a brace following the initial period of rest (usually 6 weeks). High-risk back extension activities such as gymnastics and weightlifting should not be performed for a minimum of 6 weeks and only then if athletes are pain free during activity. Recurrent symptoms are common. Treatment should include an evaluation of sport biomechanics and workload. Adjustments in amount and frequency of training as well as changes in technique should be made as part of a comprehensive treatment plan.

Hip Disorders in Youth Athletes

Overview: Hip injuries in youth athletes can be among the most serious that occur. Hip trauma may be epiphyseal, apophyseal, or diaphyseal. Many soft tissue injuries are seen, but growth plate injuries are still the most common. Skeletal and vascular growth affects the development of immature bone, the appearance of ossification centers, and blood supply to the femoral head.

Presentation: Acute injuries to the hip are common; they are usually the result of an injury during sport participation and require urgent evaluation. Overuse injuries to the hip may have a more insidious presentation and require a high index of suspicion and a thorough evaluation to accurately diagnose. Athletes with hip disorders may not always have hip pain. They may have referred pain to the thigh or knee only, with or without stiffness, muscle weakness, and/or a limp.

- **Slipped capital femoral epiphysis** (SCFE) presents usually in an overweight or obese, rapidly growing male, age 10 to 14 years. It is bilateral in 50% of the cases (Fig. 8-4).
- **Legg-Calvé-Perthes disease (LCP)** is a condition of unknown etiology that appears as avascular necrosis of the femoral head. It is commonly seen in children age 4 to 10 years, with a 4:1 male predilection and a 20% occurrence of bilateral cases (Fig. 8-5).

Physical examination: A SCFE or LCP patient may present with hip pain or just knee/thigh pain. A limp is often noted. A Trendelenburg gait is characteristic (see Fig. 8-5). Chronic cases may present with a limp or stiffness in the absence of pain. There may or may not be tenderness to palpation over the anterior hip joint. More classically, particularly in a SCFE case, when the hip is flexed the leg rides into external rotation with significant limitation in internal rotation

Diagnosis: Radiographs should always include a lateral and frog-leg anterior-posterior (AP) pelvis. Comparison of both sides allows for easier identification of pediatric hip pathology. In SCFE, there is often subtle blurring or widening of the femoral physis seen in early cases. In more severe cases there is an obvious deformity of the femoral neck. In Perthes cases, the radiographs reveal a characteristic cessation in the growth of the bony epiphysis accompanied by sclerosis, fragmentation, collapse, and/or advanced avascular necrosis. LCP is staged based on radiographic appearance as well as the percent of femoral head involvement and alignment. Once the diagnosis is made, both SCFE and LCP patients should be immediately referred to a pediatric orthopedist for definitive care.

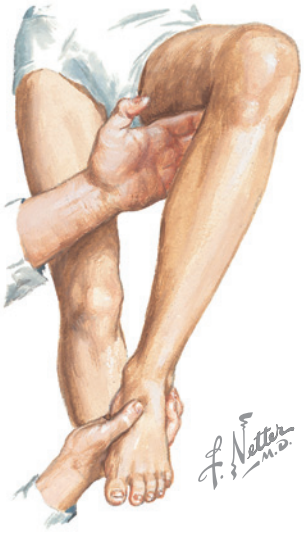
Treatment: Definitive treatment for SCFE is internal fixation or pinning of the epiphysis to stabilize it and prevent further slippage. Realignment is not routinely attempted because of the risk of disruption of the vasculature to the femoral head. The **treatment of LCP** is complicated. Initial treatment involves rest with crutches, and physical therapy to preserve range of motion and joint stability. For advanced cases, a variety of surgical containment procedures may be performed by a pediatric orthopedist.

Blount's Disease (Tibia Vara)

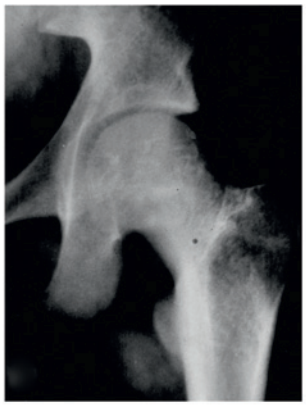
Overview: Tibia vara is a condition that involves a growth disturbance of the medial tibial physis (Fig. 8-6). It is most commonly seen in obese males age 10 to 14 but can been seen in children as young as 2 to 3; more common in African Americans. Blount's disease should be included in the differential diagnosis of any child presenting with a limp or bowlegs (genu varum) at any age.

Presentation: Classic presentation is of an obese child with a chronic progressive limp and either a unilateral or bilateral bowing of the tibia. Knee pain may be present and often localizes to the medial tibia.

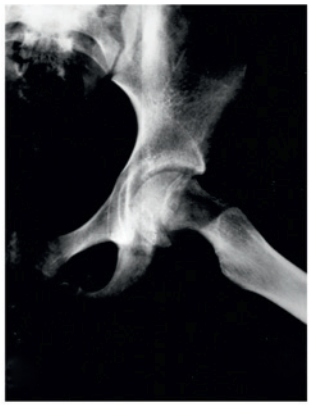
Slipped Capital Femoral Epiphysis



Best diagnostic in physical examination. With patient supine, as thigh is flexed it rolls into external rotation and abduction.



Slipped capital femoral epiphysis not readily apparent on anteroposterior radiograph because slip is usually posterior.



Frog-leg radiograph, which demonstrates slipped epiphysis more clearly, always indicated when disorder is suspected.

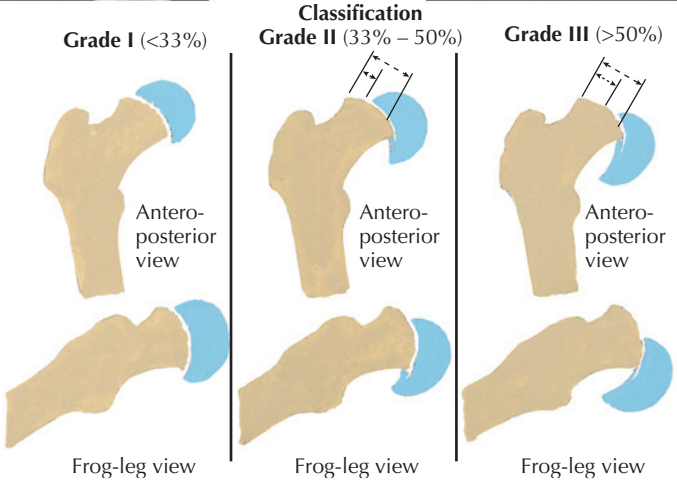
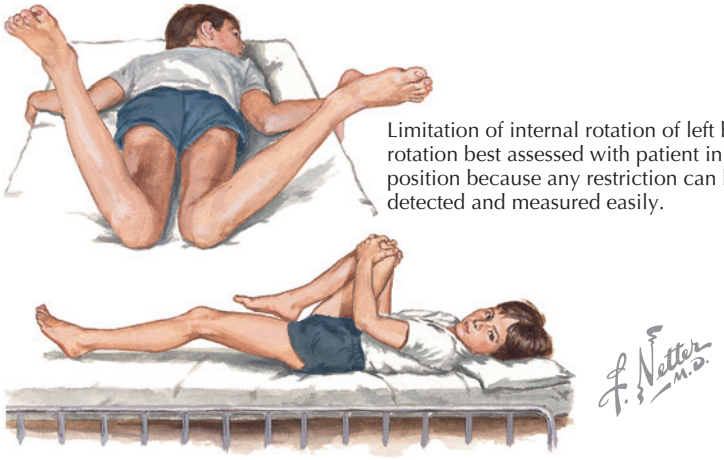
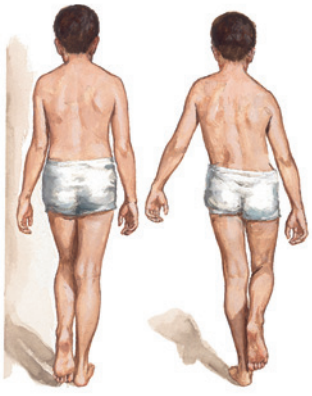


Figure 8-4 Slipped Capital Femoral Epiphysis.



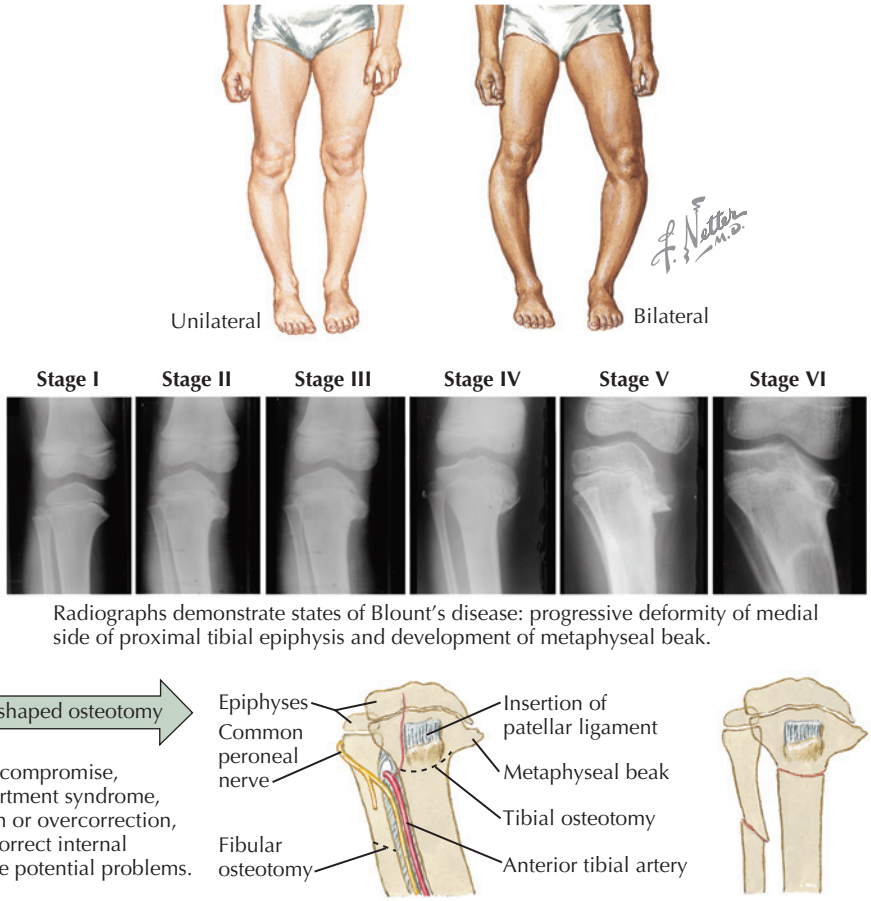
Limitation of internal rotation of left hip. Hip rotation best assessed with patient in prone position because any restriction can be detected and measured easily.

Thomas' sign: Hip flexion contracture determined with patient supine. Unaffected hip flexed only until lumbar spine is flat against examining table. Affected hip cannot be fully extended, and angle of flexion is recorded. 15° flexion contracture of hip is typical of Legg-Calvé-Perthes disease.



Trendelenburg's test
Left: Patient demonstrates negative Trendelenburg's test of normal right hip.
Right: Positive test of involved left hip. When weight is on affected side, normal hip drops, indicating weakness of left gluteus medius muscle. Trunk shifts left as patient attempts to decrease biomechanical stresses across involved hip and thereby maintain balance.

Figure 8-5 Physical Examination in Legg-Calvé-Perthes Disease.



Radiographs demonstrate states of Blount's disease: progressive deformity of medial side of proximal tibial epiphysis and development of metaphyseal beak.

Figure 8-6 Blount's Disease.

Physical examination: Findings often include a leg length discrepancy, tenderness at the medial tibial physis, and tibial torsion associated with the tibia vara. Instability to valgus stress testing may be present. Gait is usually antalgic and is notable for a varus thrust.

Diagnosis: Radiographs of the knee often reveal the tibia vara and demonstrate the growth arrest of the medial tibial physis with acute medial angulation of the proximal tibia. A standing bilateral lower extremity AP view showing pelvis to foot is the standard orthopedic view but it is often not obtained until after the diagnosis is made on standard knee radiographs.

Treatment: Blount's disease patients should be referred to a pediatric orthopedic surgeon for definitive care. Surgical treatment options include an osteotomy to realign the tibia into slight valgus, thus eliminating the leg length discrepancy and bowing. Other options include a stapling procedure of the lateral tibial physis to stop the growth throughout the proximal tibia. This can result in a significant leg length discrepancy in comparison to the contralateral side and may lead to further complications. Weight management as part of the comprehensive treatment for obesity must be emphasized in these patients and primary care providers must be involved.

Tarsal Coalition

Overview: Tarsal coalition involves a congenital bony or fibrocartilaginous fusion of tarsal bones (Fig. 8-7). It is seen in 1% to 3% of the general population and may be autosomal dominant with variable penetrance. Bilateral occurrence is seen in 50% to 60% of cases. Two types are commonly seen: calcaneonavicular and talocalcaneal.

Presentation: Symptoms usually develop in the second decade of life as increased bony ossification occurs. Progressive stiffness and limping may be seen. Pain is initially activity related.

Physical examination: A rigid flatfoot is seen with hindfoot valgus. Decreased subtalar joint motion and limited inversion is noted. Tenderness along the lateral aspect of the subtalar joint and/or sinus tarsi may be present.

Diagnosis: Bilateral weight-bearing AP, lateral, oblique, and Harris radiographs of the feet should be obtained but are often nondiagnostic, particularly in the setting of a fibrocartilaginous coalition. Calcaneonavicular tarsal coalition is more likely to be seen on plain films. Beaking of the talar neck and widening of the talonavicular joint may be secondary findings. A bilateral noncontrast CT of the feet is the gold standard.

Treatment: A referral to a pediatric orthopedic surgeon is recommended. Initial treatment is conservative and includes observation, physical therapy, gait training, and orthotics. Immobilization may be performed for 4 to 6 weeks for a more painful condition. Definitive treatment may include a surgical excision to decrease pain, improve mobility, and limit progressive degenerative changes. Surgical intervention is most effective if performed before age 14 years.

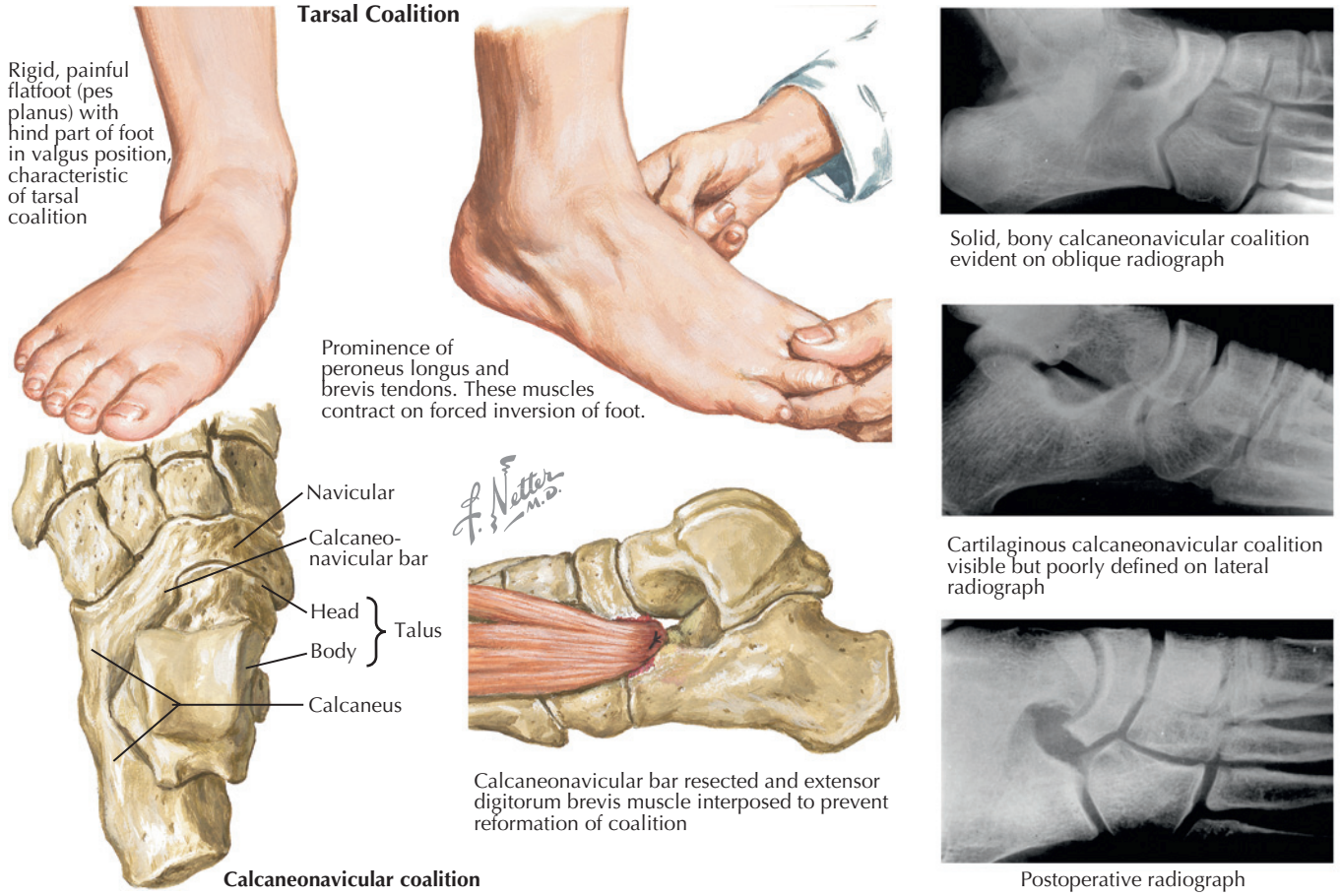


Figure 8-7 Tarsal Coalition.

RECOMMENDED READINGS

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The High School Athlete: Setting Up a High School Sports Medicine Program

9

Stephen G. Rice

GENERAL PRINCIPLES

Reasons Athletic Health Care Is Well Established at College and Professional Levels

- Awareness of needs and obligation to meet responsibilities
- Commitment to solve "problem" by meeting responsibilities
  - Risk management and loss control are central concerns to organization
  - Well-being and health of the athlete optimize on-field performance
- Attention to detail routinely practiced
- Control in all situations is usual operating procedure of the organization
- Adequate financial resources available
- Professional personnel secured in adequate quantity
  - Certified athletic trainers
  - Qualified team physicians
    - Primary care physicians with board-recognized subspecialty in sports medicine (ideal)
    - Orthopedists (ideally with sports orthopedic fellowship)
    - Other specialists with vast experience in sports medicine
    - Compliance with *Team Physician Consensus Statement* (see Appendix A).
  - Other allied health professionals
    - Certified strength and conditioning coaches
    - Nutritionists
    - Psychologists
    - Optometrists
    - Dentists
    - Exercise physiologists
    - Physical therapists
  - Policies delineated and enforced routinely

Reasons Athletic Health Care in Secondary Schools Is Clearly Inferior

- Lack awareness of scope of "problem"
- Assume obligations are met by minimal standards and effort by external agencies
  - Preparticipation physical examinations required
  - Team physician or ambulance present at home varsity football games
- Resist change—do not like to establish new policies
- Lack adequate financial resources and seek assistance at little or no expense (if athletic trainer is employed, salary scale may be at lower end of entry level)
- Unwilling to exert effort routinely, as in daily preparedness and record keeping
- Seek "quick fix," "big splash," easy solutions
- Turnover of personnel (school board, superintendent, principal, athletic director, coaches); lack of continuity leads to lack of policy
- Avoid real, long-range, long-term, permanent change—too hard to commit
- Not a priority—too many other issues

Common Pitfalls for High Schools

- Lack of knowledge among coaches and athletic directors about sports medicine
- Lack of standards—unclear exactly what is expected; seek low standards
- Poor communication with medical community
- No overall single system of care—each coach does own thing
- Lack of leadership—maintain status quo

Solution: Goals and Requirements

- Goals: proper health care for athletes and minimal liability through a risk management (loss control) policy
- Requirements: knowledge, organization, attention to detail (commitment)

Approaches to Improving Current High School Athletic Health Care

Key Elements

Three key elements to the solution (three legs to a stool): school, medical community, certified athletic trainers (ATC)

Commitment of School Is Critical

- School should work as a unit, operating a single interscholastic athletic program and single athletic health care program.
- Solution must be internal as well as external.
  - Obligations and knowledge of athletic directors and coaches
  - Use of student trainers in preparedness and athletic first aid
  - Institution of policies, guidelines, and procedures for daily use
  - Record-keeping system
- Team physicians and outside clinics alone cannot provide the constant daily health care needed for all athletes in all sports at all athletic venues.

The Key Person—Certified Athletic Trainer

- **Not automatically the entire solution**
- Cannot be present at all athletic venues simultaneously (National Athletic Trainers' Association [NATA] statement about ratio of trainers to athletes and sports attempts to ensure appropriate coverage for all athletes and all teams; focused on college programs but may apply to large high schools.)
- Needs support and system to work under
  - Educated coaches, educated corps of student trainers (who can cover all venues), and knowledgeable and supportive administration
  - Policies and procedures, including record-keeping, accountability, and quality assurance systems
- Should insist on medical supervision and quality assurance system
- Should have adequate budget
- Reasonable schedule demands because turnover may be likely after a few years (burnout or starting family); consider second trainer in large high schools
- Needs wireless communication and golf cart to meet obligations of daily coverage and emergency response during fall and spring seasons

School Should Assume Responsibility for Operating Safe Program

- Know obligations and commit to meeting them.
- Develop a system with clear written policies, procedures, and mandates.
- Hire National Athletic Trainers' Association Board of Certification (NATABOC) certified athletic trainer as best person to coordinate and operate the athletic health care program.

- Seek assistance of medical community
  - Know what is desired from physicians, physical therapists, and clinics.
  - Ask your entire medical community to get involved. Designate a head team physician.
  - Seek broad assistance and coverage for all sports: preparticipation physical exams, as well as preseason fitness screening, weekly school visits, event coverage, and therapy treatments.
- Appreciate who is in charge (and who is the consumer).

Role of Head Team Physician (and the Medical Community)

- Written contract—yes or no?
  - Delineates responsibilities and expectations
  - Helps ensure that the school has given careful thought to its obligations
  - Good communication leads to good working relationships
- Monetary compensation—yes or no?
  - If compensated, may nullify "Good Samaritan" immunity
  - Amount of compensation offered frequently meager
  - Compensation recognizes value of assistance provided
- Responsibilities (enumerated in *Team Physician Consensus Statement*)
- Jurisdiction—are your decisions final?
- Primary duty—to athlete or to school?
- Malpractice insurance and liability coverage
  - Incorporated into your personal or clinic policy (already existing or a new rider clause to be added)
  - Through school district insurance policy
- Medical-legal (liability) concerns
  - Good Samaritan Law immunity may cover team physician in some states
    - Team physician not really a Good Samaritan under strict definition— "someone without obligation who steps forward to render emergency care"
    - Has clearly defined responsibility to athletes, school, and athletic program
    - Event coverage is evidence of that responsibility
    - May be covered by "good intent, no compensation" concern
    - Good Samaritan immunity extends only to "emergency care" rendered during event coverage; protection does not extend to preparticipation physical examinations, weekly injury clinics at school, and return-to-play clearance exams
  - Potential responsibility and liability for athletic trainer's actions
    - Need to clarify issue with school district.
    - NATA-certified athletic trainers generally function "under direct medical supervision of a physician."
    - In those states that have not specifically defined the "scope of practice" for athletic trainers through licensure, certification, or registration, the team physician needs to assess implications and responsibilities of "direct medical supervision." An analogous supervisory situation may be the physician–physician's assistant relationship.
    - Written standing orders for the athletic trainer and emergency action plans are essential requisites for limiting liability risks.
  - Medical privacy concerns (HIPAA and FERPA)
    - HIPAA (Health Insurance Portability and Accountability Act) of 1996 and FERPA (Family Education Rights and Privacy Act) in 1974 were developed to regulate "protected health information."
    - Within domain of public schools, FERPA regulations prevail. FERPA governs school nurses, school physicians, coaches, and certified athletic trainers. Athletes seen at medical facilities outside of school will most likely fall under purview of HIPAA.
    - HIPAA Privacy Rule allows release of medical information without authorization for "treatment, consulting with other providers, referring the patient to other providers, and notifying patient's family." Eligibility decisions regarding "cleared" or "not cleared" on preparticipation physical examinations can be provided to coaches and school administrators (without inclusion of other medical information) without signed consent.
    - For group preparticipation physical examinations conducted at the school, must ensure confidential storage of forms, with information pertaining to restrictions shared only with those who have "need to know."
    - Need to know—always includes certified athletic trainer, school nurse, school physician (team physician). Coach and athletic administration "need to know" is variable depending on circumstances.
    - Following injury or illness, need to know often includes coaches, because well-being of athlete requires that the coach have an understanding of athlete's limitations or signs/symptoms to look for.
    - Coaches and administrators, as well as school nurses and athletic trainers, must be made aware of FERPA and HIPAA regulations and constraints regarding privacy of health care information.
- Degree of involvement
  - Set overall medical policy with athletic director and athletic trainer.
  - Strongly consider forming a medical advisory board with school district.
  - Provide medical advice to the interscholastic athletic program.
  - Provide medical coverage at games. Ideal goal—to see every team member at least once during season—may require division of coverage among several physicians, possibly by sport or other rotating basis.
    - Football team: home varsity coverage (mandatory); away varsity and home junior varsity coverage (recommended)
    - Wrestling team: preseason weight class recommendations, midseason weight certification, assessment of skin for communicable diseases, and coverage of home matches (recommended)
    - Coverage of all tournaments at home school
    - Soccer team (boys and girls): coverage of events as schedule permits
    - Basketball team (boys and girls): coverage of events as schedule permits
    - Other sports as schedule permits
  - Develop an emergency contact plan and emergency action plan, including automated external defibrillator (AED) use for sudden cardiac arrest.
  - Conduct preparticipation physical examinations.
  - Visit school/athletes regularly.
  - Educate coaches and athletic trainers.
  - Provide support for athletic trainer's authority
    - Medical-legal supervisor of certified athletic trainer
    - Assess knowledge, skills, and experience of the athletic trainer and mutually develop an appropriate set of standing orders commensurate with your joint working relationship and the legal scope of practice for athletic trainers in your jurisdiction
    - Role in writing job description
    - Role in interviewing and hiring
    - Role in job evaluation
    - Role in quality assurance of care rendered by athletic trainer: frequent regular communication as well as chart review and case studies

- Role of team physician in school without athletic trainer
  - Understand history and culture of school
  - Assess strengths and weaknesses of how athletic care is and was provided
  - Greater challenge to meet responsibilities
  - Possible institution of Athletic Health Care system (see following section)
  - Encourage school to recognize need for athletic trainer
- Role of new team physician in school with established trainer
  - The team physician should understand the methods and culture of the existing system.
  - Trainer may welcome active, involved, "hands-on" team physician or prefer more distant consultant model if he or she is comfortable as central focus of the athletic health care system and confident of abilities and skills. Team physician should develop appropriate relationship with trainer.

Athletic Trainer

- Hiring considerations and working conditions (team physician should help school in hiring process).
- Scenario—getting a job isn't easy (but it's getting easier!). Trainer submits resume and NATA pamphlets as to why certified athletic trainer is necessary.
- Athletic trainer may be perceived as salesman.
  - Identifying "a problem I didn't know existed"
  - Offering solution to problem
- Funding is a factor—athletic trainer is usually a low-paying, entry-level job.
- If athletic trainer is hired, under what conditions?
  - Full-time trainer or part-time?
  - Teacher and trainer? How many classes?
  - How many working hours per week, including games?
  - How many work days per year?
    - Trainer needs more days than regular school calendar.
    - If general contract calls for same number of days as teachers, it must take into account preseason football days, weekends, and holiday tournament days.
    - Possible solution: part-time substitute trainer, who works one day per week throughout school year (40 days); this schedule decreases risk of burnout for full-time trainer (from not having time off) and allows trainer to work same number of days as teachers.
    - Part-time trainer can service several schools each week, if more work is desired.
  - Medical backup and supervision
    - Head team physician should be specifically recognized as medical supervisor for the certified athletic trainer.
    - Degree and frequency of communication should be clearly established.
  - Whose decision is final regarding return to play?
  - Adequate budget for supplies
  - Quality of training room
  - Written job description
  - Job performance (accountability and quality assurance)—evaluated by team physician and others (e.g., athletic director, school nurse, coaches, principal, athletes)
  - Potential for career advancement
  - Budget for professional books and continuing education

ATHLETIC HEALTH CARE SYSTEM

Generic Model System

Developed by examining what makes college and professional sports programs successful in handling athletic health care and adapting to high school level

- An athletic health care system can be installed at any school
  - Large or small; urban, suburban, or rural
  - Especially helpful in remote, poor, rural areas
  - Also effective for large urban districts
- The system can work with or without other health professionals; however, the quality of the system is greatly improved with the addition of a certified athletic trainer and team physician.

Model System

Developed under strict guidelines of the U.S. Department of Education

- Development from 1978-1982; national dissemination from 1983-1995
- Rigorously tested and evaluated with new evaluation methodologies
  - Full evaluation is repeated with each new adopting school
  - Large data bank for injuries from all high school sports
- Validated by U.S. Department of Education review panels in 1982, 1987, and 1995; potential gold standard to adopt or to compare with existing program
- Approved for national dissemination via competitive grants in 1983, 1988, 1990, and 1993 by U.S. Department of Education through the National Diffusion Network (NDN), consortium of "proven programs that work"
  - Don't have to reinvent the wheel
  - Schools know and recognize NDN programs in all areas of education
    - "Proven educational programs that work"
    - Rigorously tested, transportable, cost-effective
    - Have a track record with most school districts nationally
- Orientation is practical; specific forms, protocols, lists

Six Key Elements of the Athletic Health Care System

What would a certified athletic trainer do if newly hired by a high school to start a program? He or she would install the following six key elements, in some fashion.

Assessment

- Thorough evaluation of current program
- Standards for care delineated
- Areas of assessment
  - Staff training
  - Athletic facilities
  - Athletic equipment
  - Emergency preparedness
  - Central training room
  - Provision of athletic health care services
  - Record keeping
- Self-assessment initially followed by an outside evaluation
  - Similar concept and methodology to assessments by Joint Commission on Accreditation of Health Organizations (JCAHO) and educational assessments
  - Formal written report issued
  - Action plan developed based on weaknesses and deficiencies
- Assessment frequency—every 3 to 5 years

Education

EDUCATION OF COACHES

- More states introducing coach education requirements
- National Coaching Standards promulgated in 1995 by National Association for Sport and Physical Education (NASPE)
- Scope of knowledge
  - Cardiopulmonary resuscitation—annual recertification
  - Red Cross First Aid—is it relevant?
  - Sports medicine and athletic training
    - Content (prevention, injury management); organization
    - Length—hours, days (30 to 35 hours)
    - Methods: didactic (lectures, PowerPoint slides, videos, CD-ROM, DVDs), demonstration, laboratories, and supervised practice

- Course for "soldiers on the front lines every day"
- Sensitivity of coaches
  - Toward injuries and injured athletes
    - Old school—"no pain, no gain"; no practice, no play
    - New school—recognize injury, treat, and fully rehabilitate; pain-free participation only
  - Sports psychology
- Receptivity to installing a new system, athletic trainer, or team physician
  - Personal knowledge still required even though more support available
  - Prevention and preparedness
    - Recognition of injuries and applying sports first aid
    - Insisting on full treatment and rehabilitation
  - Share responsibilities—don't dump everything onto athletic trainer
  - Recognize need to defer to knowledge of objective health professionals (certified athletic trainer or team physician) when present

EDUCATION OF STUDENT TRAINERS

- Assist coaches and certified athletic trainers in daily tasks
- Courses in summer and/or during school year
- May be funded through vocational education monies
- Provides career opportunities in health care fields

EDUCATION OF ATHLETIC DIRECTORS AND OTHER KEY ADMINISTRATIVE PERSONNEL

- Athletic administration courses
  - Athletic Health Care System, National Leadership Institute—1-week course
    - Also attended by team physicians, athletic trainers, coaches, physical therapists, school nurses, and risk managers
    - Contact: Stephen G. Rice, MD, Athletic Health Care System, Jersey Shore University Medical Center, Box 397, Neptune, NJ 07754-0397; 732-776-2384; fax 732-776-4403; e-mail: srice@meridianhealth.com.
  - National Certification for Athletic Administrators through National Federation of State High School Associations
- Organizational management of operating safe athletic program
- Safety, health care, and liability issues stressed
- Course for top overseer who must supervise the entire athletic program
- Broader scope than perspective of athletic trainer alone

Central Training Room

- Treatment facility
  - May also be rehabilitation room
  - Not weight room or conditioning center
- Physical parameters of room
  - Size—square footage
  - Location within building—proximity to gymnasium and lockers
  - Access for boy and girl athletes
  - Lighting
  - Heating and ventilation
  - Plumbing and drainage
  - Electricity
- Equipment and supplies
- Layout of room for smooth traffic flow
- Stocking, storage, inventory, and budgeting
- Security
- Daily operations
- Educational resource center—posters and books

Standard Procedures

PRESEASON SCREENING AND PREPARTICIPATION PHYSICAL EXAMINATION

- **Coordinate with medical community, athletic trainer, and coaches**
- Establish fitness expectations or requirements
  - Flexibility
  - Muscle strength in legs and shoulders
  - Muscle endurance
  - Aerobic capacity
  - Body composition
- Know athletes
  - Chronic illnesses (asthma, diabetes, seizures)
  - Differences from other athletes based on screening exam
  - Response to pain
    - High pain threshold—hide injuries; rarely report
    - Low pain threshold—constant injury reporting
  - Plays within capabilities and rules versus takes excessive risks
  - Mental "toughness"—psychological and emotional makeup
- Athlete education
  - Weight gain—anabolic steroids and supplements
  - Weight loss—fat, not water or muscle mass; eating disorders
  - Nutrition
  - Wellness and general health
  - Substance abuse
  - High-risk behaviors
- Guidelines for athletes—in season, off-season, summer fitness

COMMUNICATION

- Location of telephones, wireless (cellular) telephone, pagers
- Lists of key people and telephone numbers widely disseminated
- Whom to notify in case of emergencies
- Emergency information cards for every athlete
- Lists of hospitals, clinics, physicians—addresses and phone numbers

PREPAREDNESS

- Emergency information cards for every athlete
- Checklists to ensure proper materials available daily
  - First-aid kit supply list
  - Sideline equipment list, including AED
  - Communication supplies—sideline notebook

AREA SAFETY

- Safety inspections at start of year and season
- Daily inspections of playing surface and surroundings

EVALUATION PROCEDURE FOR ACUTE "ON THE FIELD" INJURIES

- Triage severity—rule out "worst first"
- Life threatening—unresponsive, unconscious—check airway, bleeding, circulation (ABCs)
- Stable or unstable—if left unattended, would athlete deteriorate?
  - Airway obstruction
  - Bleeding—acute hemorrhage (internally or externally)
  - Circulatory failure (shock)
  - Anaphylaxis
  - Infection
  - Metabolic condition
  - Cardiogenic
  - Neurogenic
  - Heat illness
- Permanent injury
  - Neurological

  - Severe fracture
  - Major muscle contusion
  - Eye trauma
  - Finger tendon rupture
  - Cauliflower ear
- Limb threatening—major dislocation or severe fracture
- Common sports injuries
  - Contusions
  - Strains
  - Sprains—grades I and II
  - Dislocations—patella and fingers
  - Fractures—closed and nondisplaced
- Initiate emergency action plan as indicated; treat bleeding, shock as needed
- Conduct step-by-step evaluation
- Determine if is safe and appropriate to move athlete from field or court

WRITTEN ACTION PLAN FOR EMERGENCIES

- Written policy statement—what to do in case of an emergency with special attention to AED location and use
- Define specific medical emergencies
- Emergency map—prepared in advance
  - How to get emergency response vehicle to site
  - How to drive to appropriate medical facility
  - Share emergency map and directions to fields and gyms with local emergency response personnel—at start of school year or season
- Calling for emergency help: EMT or paramedic?
  - Paramedics: 800 to 1000 hours of training
  - EMTs: 100 hours of training
  - Be sure to ask for most appropriate assistance
- Written note should always accompany injured athlete
- Call ahead to hospital when appropriate—talk directly to emergency room physician
- Notify parents, guardians, and personal physicians

PROTOCOLS AND GUIDELINES

- Preseason fitness and conditioning for athletes
- Stretching
- RICES—rest, ice, compression, elevation, support/splint—injury first aid
- Use of crutches
- Use of nonsteroidal anti-inflammatory drugs (NSAIDs)
- Ice friction massage
- Contrast therapy
- Head injury home instruction sheet for concussion
- Ankle rehabilitation guidelines
- Quadriceps strengthening
- Steps to rehabilitation

STEPS TO RECOVERY AND FULL REHABILITATION—AS POLICY

- Control swelling
- Control pain
  - Healing achieved with elimination of swelling and pain
  - Healed, but not necessarily rehabilitated
- Restore range of motion
- Restore muscle strength
- Restore joint stability—through rehabilitation, taping, bracing, surgery
- Restore general function, including the following:
  - Aerobic capacity
  - Neuromuscular function
  - Core stability
  - Scapulothoracic function
- Restore sport-specific function

CRITERIA FOR RETURN TO PLAY—AS POLICY

- Little or no swelling
- Little or no tenderness (pain) to touch
- Full, pain-free range of motion
- Full, pain-free strength through the full range of motion
- Pain-free joint stability—through rehab, taping, bracing, surgery
- Full, pain-free general functional activities
- Full, pain-free sport-specific functional activities

Record Keeping

PURPOSES

- Injury surveillance
- Document care and treatments given
- Communicate among persons involved in athlete's health care
- Protection regarding medical-legal liability
- Inventory supplies and cost justification
- Quality assurance and accountability

RECORD KEEPING MAY BE ELECTRONIC OR WRITTEN

- Athlete emergency information card
- Attendance and injuries (daily report as part of injury surveillance system)
  - Note all athletes absent, ill, or injured
  - Healthy athletes who are present—leave box blank
  - Completed daily; submitted monthly
- Training room treatment log
  - All treated athletes sign in daily
  - Trainers record (check off) treatments received
- Athlete injury report—two-part, four-copy NCR (no-carbon-required) form
  - From coach, athletic trainer, or team physician to parents, athlete's personal physician, or hospital emergency department
    - One part gives information to health care provider
    - Other part receives findings or advice from provider
  - Copies to coach, athletic trainer or athletic director, team physician, family health care provider
- Sports injury report or individual charts for athletes
  - Can be "charting" for athletic trainer or team physician
  - Uses date of evaluation as the filing point
  - With three-copy NCR form, copies for coach, athletic trainer or athletic director, and team physician
- Master daily injury list—all sports
  - List of all athletes not at full participation
  - All those on limited participation and no participation recorded
  - Includes diagnosis, limitations, treatments, and rehabilitation

Evaluation and Feedback, Technical Assistance

- **Needs assessment**
- **Education—knowledge gain in courses for coaches and student trainers**
  - Pretest/posttest comparison—document significant knowledge gains
  - Compare to established norms among similar trainees
- **Prevention**—emergency preparedness and sideline safety
  - Observation technique
  - Checklist for first-aid kit contents
  - Checks for stretching, area safety, emergency equipment, presence of water, emergency information and written action plan, availability of athletic trainers and coaches to care for athletes
  - Printed report to return to school
  - Compare results to established norms among similar schools

- **Injury data—injury surveillance**
  - Tabulate data, determine injury rates
    - Numerator—number of injuries, body part or injury type
    - Denominator (measure of risk)—number of participants or athletic exposures
  - Follow year-to-year trends within school and sports
  - Compare self to others in data bank—this year and against prior years
  - Actual findings may challenge "conventional wisdom"; nearly 60,000 athletes followed over 13 years in 2.5 million athletic exposures (Table 9-1)
    - Girls cross country has the highest injury rate of all sports.
    - Fall sports have more injuries than spring sports.
    - Girls' sports have more injuries than identical boys sports.
    - Cross country and soccer (boys and girls) are in the highest tier of injuries along with football, wrestling, and gymnastics.
  - Provide basis for epidemiologic studies
- **Case studies**
  - Technique for monitoring quality of care rendered from moment of injury through return to play
  - Questionnaire to gather data from coach, athlete, and athletic trainer
  - Narrative prepared
  - Specific evaluation criteria developed and shared with evaluators
  - Blind review by expert evaluators in sports medicine
  - Areas of evaluation
    - Time frames: moment of injury, later that day (before going home or to hospital), next day until rehabilitation over, day of return to play
    - Categories of activities: injury recognition, transport, examination and assessment, first aid and treatments, communication and advice, documentation (record keeping)
  - Identify strengths and weaknesses in care
  - Overall quality assurance evaluation—process of health care provided

Adoption Process

- **Goal of permanent change and long-term results**
  - Fidelity to installation of all six key elements
  - Local control—identify local resources to maintain implementation and establish "ownership"
  - Self-sufficiency from continued fidelity
- **Adoption begins with awareness:** Parents and boosters, school boards, superintendents, curriculum directors, principals, risk managers/business managers, athletic directors, coaches, physicians, athletic trainers, sports medicine clinics, regional and state organizations, media
- **Networking**
  - Helps to generate a standard for quality of care for athletes
  - Identifies other health professionals interested in bringing program to their area
  - Spreads via the ripple effect through "disciples" with fidelity to concept
- **Adoption specifics**
  - Athletic Health Care System staff travels nationally to install system in individual schools or states.
  - School physicians can attend National Leadership Institute for a week to learn in detail how to implement system in their locale with or without direct help of Athletic Health Care System staff.

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Table 9-1 INJURY DATA ANALYSIS: ALL YEARS, FALL 1979 THROUGH SPRING 1992

Rank*	Sport	Season	Total athletes	Injury rate/100 athletes/ season	Injury rate/1000 athletes/ season	Significant injury rate 1000 athletic exposures (1 wk)	Major injury rate 1000 athletic exposures (3 wk)	Percent of different athletes injured
1	Girls' cross country	Fall	1299	61.4	17.3	3.3	0.9	33.1
2	Football	Fall	8560	58.8	12.7	3.1	0.9	36.7
3	Wrestling	Winter	3624	49.7	11.8	2.6	1.0	32.1
4	Girls' soccer	Fall	3186	43.7	11.6	2.9	0.7	31.6
5	Boys' cross country	Fall	2481	38.7	10.5	2.3	0.5	24.6
6	Girls' gymnastics	Winter	1082	38.9	10.0	2.3	0.7	26.2
7	Boys' soccer	Spring	3848	36.4	9.5	2.1	0.4	25.2
8	Girls' basketball	Winter	3634	34.5	7.1	1.7	0.5	24.2
9	Girls' track	Spring	3543	24.8	6.2	1.6	0.3	18.0
10	Boys' basketball	Winter	3874	29.2	5.5	1.3	0.3	22.9
11	Volleyball	Fall	3444	19.9	5.4	1.1	0.3	16.1
12	Softball	Spring	2957	18.3	4.8	1.2	0.3	14.8
13	Boys' track	Spring	4425	17.3	4.4	1.1	0.3	13.6
14	Baseball	Spring	3397	17.1	4.2	1.0	0.3	14.4
15	Fastpitch softball	Spring	134	11.9	2.4	1.2	0.6	11.9
16	Coed swimming	Winter	4004	8.3	2.2	0.5	0.2	6.4
17	Coed tennis	Fall/Spring	4096	7.0	1.9	0.4	0.1	5.8
18	Coed golf	Fall/Spring	2170	1.4	0.8	0.0	0.0	1.3
Total			59,758	30.6	7.6	1.8	0.5	21.1

*Ranking based on injury rate per 1000 athletic exposures. Data contributed by 21 different high schools.

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The Female Athlete

10

Renata J. Frankovich

GENERAL PRINCIPLES

- Female athlete issues can be divided into health concerns exclusive to women and those more common in women.
- Menstruation and pregnancy are conditions specific to young women and influence exercise performance and sport.
- The female athlete triad is a condition that describes the relationship between **energy availability, menstrual function,** and **bone mineral density.**
- Other health problems, such as osteoporosis and anterior cruciate ligament (ACL) tears are more common in women and have female-specific determinants contributing to their incidence.

ANATOMICAL AND PHYSIOLOGICAL DIFFERENCES BETWEEN FEMALES AND MALES

Growth and Maturation

- Skeletal growth is similar until approximately age 9 or 10. No significant difference in physical capabilities of prepubertal boys and girls exists if body size and composition are accounted for.
- Average age of linear growth spurt for girls is 11 and for boys is 13 years. Rate of growth in girls slows during menarche (ages 12 to 14). Boys continue to grow from ages 12 to 15. Maximum height is attained at age 16 to 17 for girls and 20 to 21 for boys. Body weight pattern of development is similar to that for linear height.
- At maturity, the 11 to 14 kg weight difference between males and females can be accounted for by the additional bone mass and muscle mass in males.

Body Fat Composition and Distribution

- Average body fat is 26% for women and 14% for men. The increase in lean body mass in men is attributed to the androgenic hormones.
- Subcutaneous fat distribution in men is found mainly in the abdomen and upper trunk, whereas in women it is found in the hips and thighs.

Anatomical and Biomechanical Differences

- Typical male body shape pattern is wide shoulders and narrow hips. Female body shape pattern is narrow shoulders and wide hips. Variation of pelvic shapes among women is greater than the difference between sexes. Hip and shoulder widths have been found to be similar in competitive male and female track athletes.
- In *some* women, a wider pelvis contributes to increased Q angle at the knee and narrow shoulders contribute to increased carrying angle at the elbows.
- Women typically have shorter limbs compared to men. This may contribute to decreased power as a result of shorter lever action, but it may be beneficial in activities that require balance because it means women typically have a lower center of gravity than men.

Muscle Strength

- Men are generally stronger than women as a result of overall increased muscle mass. However, no significant difference exists if strength is expressed relative to lean body mass.
- Strength gains are the result of muscle recruitment and muscle hypertrophy. With strength training, males gain more muscle hypertrophy than females because of the effect of testosterone.
- Women have the same capacity for attaining strength gains as men.
- Upper extremity strength in women is approximately 40% to 75% that of men and lower extremity strength is approximately 60% to 80% that of men.

Aerobic Capacity

- The difference in maximum oxygen uptake ($\dot{V}O_2$ max) between men and women is 40% on average, but this difference decreases to 20% if compared per kg body weight and 10% if compared to lean body weight.
- Women have a lower total oxygen-carrying capacity of blood because of lower blood volume, approximately 6% fewer red blood cells, and approximately 15% less hemoglobin.
- Women have a smaller heart, which results in lower stroke volumes and cardiac output. To compensate, heart rates are higher at the same percentage of maximum aerobic capacity.
- Women have a smaller thoracic cage and consequently lower lung volumes, but this is offset by a smaller blood volume and less tissue to oxygenate. Vital capacity is about 10% less in women compared to men, controlling for size and age.

Endurance Performance

- Males outperform females in distance events by about 5% to 15% because of differences in body size, body composition, and $\dot{V}O_2$ max.
- Performance gaps in endurance events like marathons, ultramarathons, and Ironman distance triathlons have narrowed as a result of increased competitive opportunities for women and improved training regimens.

THE MENSTRUAL CYCLE, CONTRACEPTION, AND SPORT PERFORMANCE

Menstrual Cycle

- Average age of menarche is 12.8 years. Average length of the menstrual cycle is 28 days (varies from 20 to 45 days). The cycle involves a well-defined pattern of hormonal changes (Fig. 10-1).
- **Follicular or proliferative phase:** First half of the cycle. Starts with menses. Ends with ovulation. Initially, estrogen and progesterone levels are low. Follicle-stimulating hormone (FSH) is released, causing the ovaries to produce estrogen. The uterine lining proliferates and the primary follicle forms. When this follicle ruptures, the ovum is released, marking the end of the follicular phase.
- **Luteal or secretory phase:** Second half of the cycle. Starts with ovulation. Ends with menstruation. Normal duration is 14 days. Increased levels of estrogen result in a surge in luteinizing hormone (LH) secretion, triggering ovulation. Estrogen and progesterone levels are high. If fertilization of the follicle does not occur, estrogen and progesterone decline rapidly and menstruation occurs.
- Table 10-1 lists the effects of estrogen.
- Table 10-2 lists the effects of progesterone.

Athletic Performance and Contraception

- Female athletes may be using the oral contraceptive pill (OCP) for contraception, cycle control, management of men-

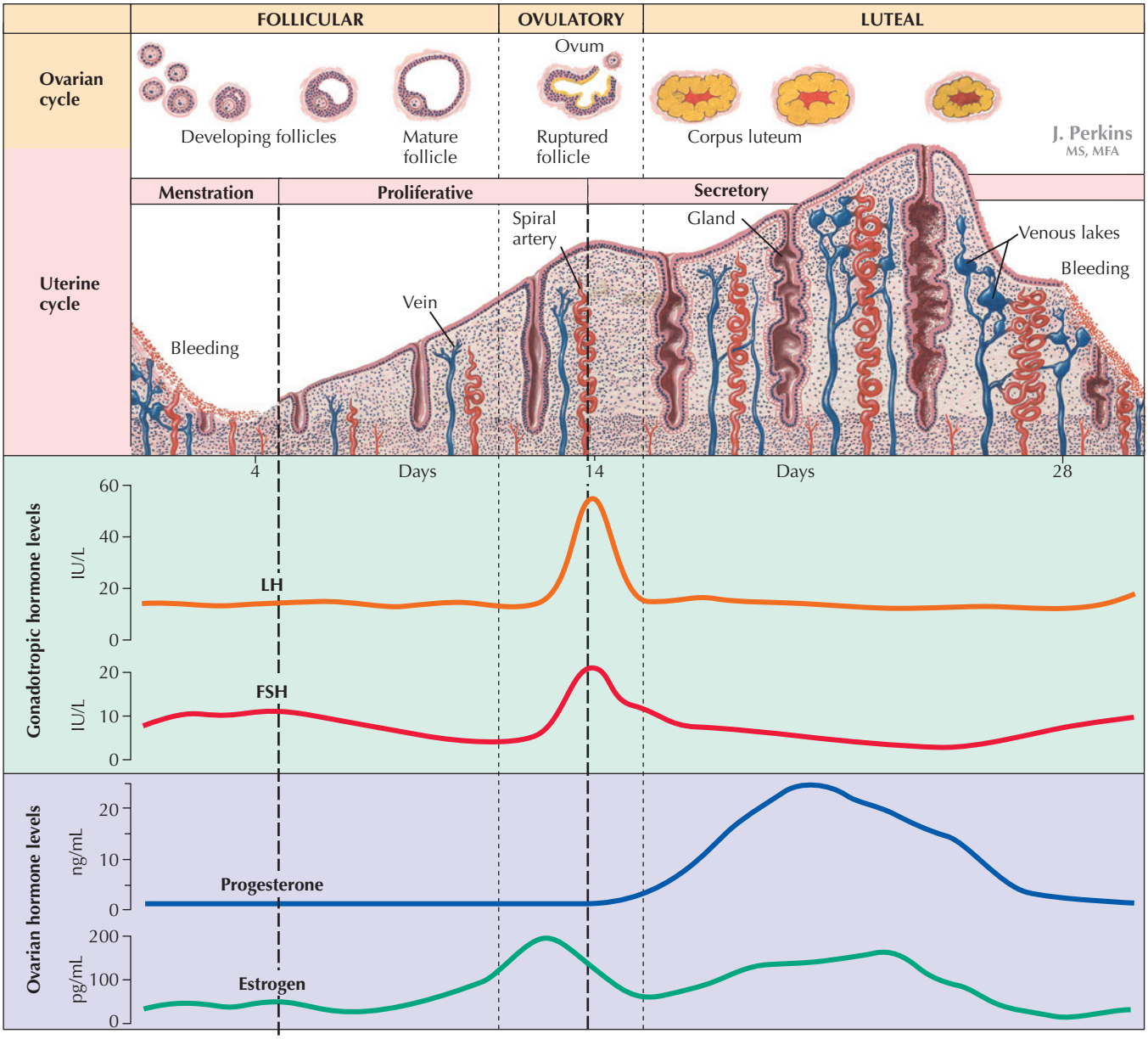

Figure 10-1 Phases of the menstrual cycle with gonadotropic and ovarian hormone levels.

Table 10-1 EFFECTS OF ESTROGEN

System	Effect
Cardiovascular	↑ in thrombosis ↓ total cholesterol ↓ low-density lipoprotein (LDL) levels ↑ high-density lipoprotein (HDL) levels Vasodilates vascular smooth muscle ↑ blood pressure
Endocrine	↑ intramuscular and hepatic glycogen storage and uptake Glycogen sparing (↑ lipid synthesis, ↑ lipolysis in muscle, and ↑ utilization of free fatty acids) ↑ insulin resistance and ↓ glucose tolerance Deposition of fat in the breasts, buttocks, and thighs
Bone metabolism	Facilitates uptake of calcium into bone
Neurologic	Cognitive function and verbal memory in postmenopausal women

Table 10-2 EFFECTS OF PROGESTERONE

System	Effect
Thermoregulation	↑ core body temperature of 0.3-0.5° C
Respiratory	↑ minute ventilation ↑ ventilatory response to hypoxia and hypercapnia
Metabolic	Fluid retention ↑ dependence on fat as a substrate Induce peripheral insulin resistance

strual disorders like oligomenorrhea or amenorrhea, premenstrual symptoms, and time-shifting of the menstrual cycle.

- Despite the widespread use of the OCP, the literature in this area is scarce. Given the proliferation of OCPs into monophasic combination pills consisting of various doses of fixed combinations of estrogen and progesterone, biphasic and triphasic preparations with variable hormone concentrations, and

progestin-only pills, injectables, or implantable forms, generalizations from the limited research cannot be drawn. The effect of OCPs on physical fitness parameters and athletic performance is still unclear.
- Athletic performance may be improved by controlling premenstrual symptoms (fatigue, fluid retention, etc.) with OCPs. Endurance athletes may benefit from a lower incidence of iron deficiency anemia found in OCP users.
- Lower incidence of musculoskeletal injuries has been documented in women taking OCPs, likely because of a reduction of premenstrual symptoms and dysmenorrhea. OCP use reduced the risk of stress fractures in female runners in a study limited by small sample size.

Athletic Performance and the Menstrual Cycle

- Anecdotal evidence suggests that some athletes feel that there is a detrimental effect on performance during the premenstrual or menstrual phases. Studies have failed to support this link but there are numerous methodology challenges: measuring determinants of physical fitness, measuring performance variables, simulating competition conditions, and measuring for and controlling for individual variations in hormone levels and their fluctuations across the menstrual cycle.
- A luteal phase increase of 7 beats per minute in heart rate response to exercise has been documented. However, hormone-mediated changes in cardiovascular variables appear to offset one another, resulting in no significant change in performance.
- Female athletes with asthma may be vulnerable during the premenstrual and perimenstrual phases because peak expiratory flow rates have been reported to be decreased by 30% to 40% and presentation to the emergency department increased fourfold in the general asthmatic population. Performance implications in the female athlete population must be studied further but theoretically the drop in progesterone, which acts as a smooth muscle relaxant, in the late luteal phase, leads to bronchoconstriction of the bronchial smooth muscle.
- Higher core temperature during the luteal phase may be a factor for women exercising under extreme heat and humidity.
- The predominant role of estrogen and progesterone in substrate metabolism is complex given the changing levels of hormones throughout the menstrual cycle and the interaction with other hormones in energy production.
- Recent studies linking estrogen to beneficial effects on cognition, alertness, and cognitive performance may have menstrual cycle phase athletic performance effects. This is a newly developing area of research interest.
- Muscular strength has not been shown to change significantly across the menstrual cycle, despite some studies reporting an inotropic effect of estrogen. Muscle strength loss with the onset of menopause is thought to be related to the drop in estrogen.
- Aerobic and anaerobic capacity, as well as aerobic endurance, have been studied but no consistent menstrual cycle phase effects have been demonstrated in the studies done to date.
- A number of studies have found that ACL injuries have a higher incidence during the ovulatory phase and a decreased incidence during the luteal phase. However, other studies have reported the complete opposite, making the relationship between ACL injuries and menstrual phase unclear. The cellular effects of estrogen and progesterone on ACL laxity is a controversial topic, garnering much interest because estrogen receptors were found on the human ACL. Studies examining ACL laxity across the menstrual cycle report conflicting results. The relationship between knee laxity and injury rates is also unclear.
- Menstrual cycle effects on performance parameters likely vary between individuals and should be of particular concern to women with premenstrual and/or menstrual symptoms. Female athletes, especially elite competitors, should be encouraged to monitor their own individual responses across the menstrual cycle in order to adapt their training programs and maximize performance.

FEMALE ATHLETE TRIAD

General Principles

- The female athlete triad was first described in 1992 by an American College of Sports Medicine (ACSM) Task Force on Women's Issues as **disordered eating, amenorrhea,** and **osteoporosis.**
- Concept of the triad has evolved to the interrelationship between **energy availability, menstrual function,** and **bone mineral density (BMD)** along the continuum from health to disease. Optimal energy availability supports eumenorrhea and bone health. At the other end of the spectrum, low energy availability induces amenorrhea, resulting in a loss of the estrogen protective effect on bone health, and suppresses hormones that promote bone formation, resulting in osteoporosis.
- Energy availability can vary daily from sufficient to inadequate. Menstrual dysfunction can take months to manifest and can vary across a spectrum ranging from subclinical disorders, like luteal deficiency, to functional hypothalamic amenorrhea (FHA). Alterations in BMD range from below average values for age to osteoporosis, and may take a year or more before they become evident.
- Each disorder in itself or in combination can have a negative effect on health and athletic performance. Identification of one component should prompt the physician to evaluate for the presence of the other components of the triad. The triad should be screened for during the preparticipation physical exam or during the periodic health exam.
- The etiology of the triad in the female athlete is thought to start with a belief that lower body weight is necessary for athletic success and/or social acceptance. Restrictive eating can lead to menstrual dysfunction, which subsequently can result in detrimental effects on BMD.
- Disordered eating behavior manifesting as **low energy availability** is the key dysfunction underlying the triad.
- **Prevalence** of the complete triad among female athletes has been debated. Some claim the problem has been overestimated. Only three studies to date have examined prevalence of all components of the triad and have used a direct measure of BMD. These studies have shown that the presence of all three components of the triad simultaneously is small (approximately 1% to 3%). Each component, however, is much more prevalent: disordered eating was found in 18% to 25% of female athletes and menstrual dysfunction was found in 24% to 26%. In a study of Norwegian athletes, the presence of all three components was similar to those in nonathletic controls, but the presence of menstrual dysfunction and disordered eating together was much more common in athletes (11%) compared with nonathletes (3%). In a study published in 2004, prevalence of eating disorders in Norwegian female athletes was 20% higher compared with nonathletic female controls.
- Women participating in sports that emphasize thinness or leanness tend to be at greater risk for developing disordered eating. Female athletes in aesthetic sports, running, and weight-dependent sports have also been shown to be at higher risk. Prevalence rates for disordered eating have been found to be as high as 62% in collegiate gymnasts and 25% to 31% in elite female athletes, compared with 5.5% to 9% in the general population. Prevalence of inadvertent low energy availability is unknown.
- Prevalence of menstrual dysfunction in the general population of premenopausal women is thought to be 2% to 5% and

in female athletes it is 6% to 69%, depending on the type of sport, level of competition, body weight, and age. Sports emphasizing leanness have reported prevalence rates of amenorrhea as high as 69% in dancers and 65% in long-distance runners. Prevalence of amenorrhea in distance runners went from 3% to 60% with mileage increases from less than 13 to more than 113 km per week and body weight declined from more than 60 kg to less than 50 kg. Primary amenorrhea is more prevalent in female cheerleaders, divers, and gymnasts than in the general population (22% compared with 1%).

- Prevalence of low bone mass among female athletes is unclear because of differences in diagnostic criteria used in studies. Using the World Health Organization (WHO) classification, prevalence rates of osteoporosis in female athletes has been found to be 0% to 13% and of osteopenia has been found to be 22% to 50%, compared with 2.3% rate of osteoporosis and 12% rate of osteopenia in the general population (Table 10-3). The concern with bone health is that bone not formed, or bone that is lost, may not be completely regained.
- Prevention of the triad needs to be done through education and implementation of policies and procedures aimed at eliminating harmful weight loss practices. The National Collegiate Athletic Association (NCAA) has a handbook entitled "Managing the Female Athlete Triad" that serves as an excellent resource for coaches to guide their female athletes to good health and peak performance (available at www.ncaa.org/wps/wcm/connect/resources/file/ebad9e4a146e2d0/Handbook.pdf?MOD=AJPERES). Prevention also requires the commitment of the health care team along with cooperation from athletic administrators and international and national sport governing bodies. Promotion of physical activity for physical, psychological, and social benefits should continue to be encouraged in women because the benefits of exercise far outweigh potential risks.

Energy Availability: Definitions and General Concepts

- Energy availability is defined as dietary energy intake minus exercise energy expenditure. Energy availability is the residual dietary energy, after exercise, accessible for physiological functions such as growth and reproduction.
- **Low energy availability** can occur when energy expenditure is increased through exercise or when dietary energy intake is reduced. It is usually associated with disordered eating behaviors directed at weight loss but may be inadvertent.
- There is a continuum of abnormal eating behavior, from failure to meet the energy requirements of exercise to pathological end-stage clinical eating disorders.
- **Subclinical eating disorders** is a term used to describe individuals who do not meet the strict criteria of a clinical eating disorder but manifest many of the features related to pathological eating behavior and body weight concerns.

Table 10-3 WORLD HEALTH ORGANIZATION DIAGNOSTIC CRITERIA FOR OSTEOPOROSIS USING DUAL-ENERGY X-RAY ABSORPTIOMETRY (DXA)

Classification	BMD using T-score
Normal	T-score > -1
Osteopenia	T-score -1 to -2.5
Osteoporosis	T-score < -2.5
Severe or established osteoporosis	T-score < -2.5 plus history of fracture

Adapted from World Health Organization: Assessment of Fracture Risk and Its Application to Screening for Postmenopausal Osteoporosis. Geneva: WHO, 1994.

- **Clinical eating disorders** are psychiatric conditions defined in the *Diagnostic and Statistical Manual of Mental Disorders*, 4th edition (DSM-IV). These disorders include anorexia nervosa, bulimia nervosa, and eating disorders not otherwise specified (Box 10-1). These disorders are often accompanied by other conditions such as depression, anxiety, and obsessive-compulsive disorder.
- There is good quality evidence to support that severe undernutrition impairs reproductive and skeletal health.
- There is good quality evidence that disordered eating and eating disorders occur more frequently in sports that emphasize leanness.
- Low energy availability affects a cascade of metabolic hormones and substrates (e.g., insulin, cortisol, growth hormone, insulin-like growth factor, triiodothyronine, leptin, glucose, fatty acids, and ketones). It has been suggested that the hormone leptin, which regulates basal metabolic rate, reflects nutritional status. A level of 1.85 mg is required for normal menstruation; low levels are found in athletes with disordered eating and amenorrhea. Ghrelin, a peptide hormone linked to the sensation of hunger, luteinizing hormone pulsatility, and energy homeostasis, is found in higher levels in amenorrheic women with energy deficit.

Etiology of Low Energy Availability

- Low energy availability may occur inadvertently because of inadequate nutritional knowledge, lack of availability or insufficient access to appropriate foods, or time constraints preventing the athlete from meeting their energy demands. Hunger was increased with dietary restriction but the same energy deficit induced by exercise did not trigger the sensation of hunger.
- The cause of eating disorders is not clear. A model for the development of eating disorders in female athletes has been proposed by Sundgot-Borgen (Fig. 10-2). Dieting and psychiatric morbidity were the best predictors of clinical eating disorders in teenage boys and girls in a prospective study done by Patton and colleagues.

Risk Factors for Low Energy Availability and Disordered Eating

- Dieting or restrictive eating
- Vegetarianism
- Belief that thinness equates with social success
- Belief that decreasing body weight or body fat will enhance performance
- Behavioral traits characterized by perfectionism and obsessive-compulsive tendencies
- Competitive nature—striving to be the leanest athlete
- Involvement in sports involving judging (diving, gymnastics, figure skating, etc.), revealing uniforms (swimming, beach volleyball, etc.), weight classifications (rowing, martial arts, wrestling, etc.), and thin body types (endurance running)
- Excessive exercisers
- Onset of sport-specific training at an early age
- Injury or illness
- Overtraining or sudden increase in training load
- Coaching behavior emphasizing weight and body type
- Psychological traumatic event (e.g., change in coaching, poor performance)

Medical Evaluation of Energy Availability

Medical History

- Eating behavior
  - Restricting: average daily intake, meals per day, calories per day, elimination of certain foods (e.g., fats, dairy, protein, meat)
  - Bingeing: frequency of binges, foods binged on, binge trigger foods

BOX 10-1 *Clinical Eating Disorders*

DIAGNOSTIC CRITERIA FOR 307.1 ANOREXIA NERVOSA

A. Refusal to maintain body weight at or above a minimally normal weight for age and height (e.g., weight loss leading to maintenance of body weight less than 85% of that expected; or failure to make expected weight gain during period of growth, leading to body weight less than 85% of that expected).
B. Intense fear of gaining weight or becoming fat, even though underweight.
C. Disturbance in the way in which one's body weight or shape is experienced, undue influence of body weight or shape on self-evaluation, or denial of the seriousness of the current low body weight.
D. In postmenarcheal females, amenorrhea, i.e., the absence of at least three consecutive menstrual cycles. (A woman is considered to have amenorrhea if her periods occur only following hormone [e.g., estrogen] administration.)

Specify type:

Restricting Type: during the current episode of Anorexia Nervosa, the person has not regularly engaged in binge-eating or purging behavior (i.e., self-induced vomiting or the misuse of laxatives, diuretics, or enemas)

Binge-Eating/Purging Type: during the current episode of Anorexia Nervosa, the person has regularly engaged in binge-eating or purging behavior (i.e., self-induced vomiting or the misuse of laxatives, diuretics, or enemas)

DIAGNOSTIC CRITERIA FOR 307.51 BULIMIA NERVOSA

A. Recurrent episodes of binge eating. An episode of binge eating characterized by both of the following:
(1) eating, in a discrete period of time (e.g., within any 2-hour period an amount of food that is definitely larger than most people would eat during a similar period of time and under similar circumstances)
(2) a sense of lack of control over eating during the episode (e.g., feeling that one cannot stop eating or control what or how much one is eating)
B. Recurrent inappropriate compensatory behavior in order to prevent weight gain, such as self-induced vomiting; misuse of laxatives, diuretics, enemas, or other medications; fasting; or excessive exercise.
C. The binge eating and inappropriate compensatory behaviors both occur, on average, at least twice a week for 3 months.
D. Self-evaluation is unduly influenced by body shape and weight.
E. The disturbance does not occur exclusively during episodes of Anorexia Nervosa.

Specify type:

Purging Type: during the current episode of Bulimia Nervosa, the person has regularly engaged in self-induced vomiting or the misuse of laxatives, diuretics, or enemas

Nonpurging Type: during the current episode of Bulimia Nervosa, the person has used other inappropriate compensatory behaviors, such as fasting or excessive exercise, but has not regularly engaged in self-induced vomiting or the misuse of laxatives, diuretics, or enemas

307.50 EATING DISORDER NOT OTHERWISE SPECIFIED

The Eating Disorder Not Otherwise Specified category is for disorders of eating that do not meet the criteria for any specific Eating Disorder. Examples include:
1. For females, all of the criteria for Anorexia Nervosa are met except that the individual has regular menses.
2. All of the criteria for Anorexia Nervosa are met except that, despite significant weight loss, the individual's current weight is in normal range.
3. All of the criteria for Bulimia Nervosa are met except that the binge eating and inappropriate compensatory mechanisms occur at a frequency of less than twice a week or for a duration of less than 3 months.
4. The regular use of inappropriate compensatory behavior by an individual of normal body weight after eating small amounts of food (e.g., self-induced vomiting after the consumption of two cookies).
5. Repeatedly chewing and spitting out, but not swallowing, large amounts of food.
6. Binge-eating disorder: recurrent episodes of binge eating in the absence of the regular use of inappropriate compensatory behaviors characteristic of Bulimia Nervosa.

Reprinted with permission from Diagnostic and Statistical Manual of Mental Disorders, 4th ed. Washington, DC: American Psychiatric Association, 1994.

  - Purging activities: self-induced vomiting, laxatives, diuretics, excessive exercise
- **Weight:** current, ideal, most and least in past year; level of satisfaction with current weight
- **Menstrual history:** onset of menarche, last menstrual period, number of menstrual periods in past year
- **Family history:** disordered eating, obesity, depression, anxiety disorders, substance abuse
- **Psychological and behavioral characteristics** (Box 10-2).
- **Review of systems**
  - **Anorexia nervosa:** symptoms caused by starvation and dehydration and include lightheadedness, syncopal episodes, weakness, palpitations, overuse injuries, and decreased school, work, and sport performance
  - **Bulimia nervosa:** symptoms caused by purging activity and include frequent sore throats, dental or periodontal disease, bloating, abdominal pain, diarrhea, constipation, gastrointestinal (GI) bleeding (upper GI—Mallory-Weiss tears resulting from self-induced vomiting; lower GI—rectal bleeding from finger manipulation of the rectum and/or frequent use of laxatives), overuse injuries, and decreased school, work, and sport performance
- **Medication and substance use**
  - Medications: diet pills, diuretics, oral contraceptives
  - Tobacco, alcohol, illicit drugs

Physical Examination

- Pulse, blood pressure (including orthostatic), temperature
- Height, weight, body mass index (BMI)
- Tanner staging to assess growth and development in adolescent

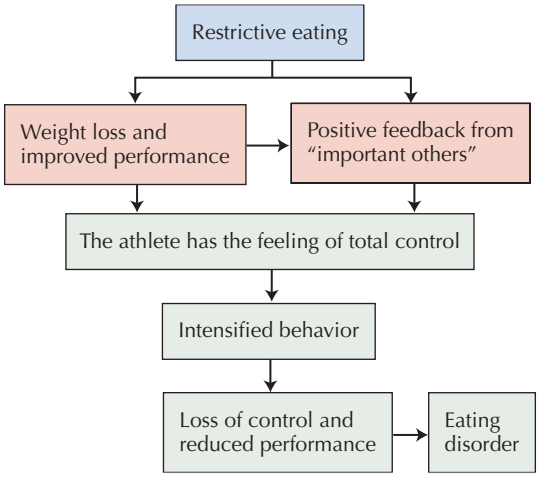

Figure 10-2 Etiologic model for the development of eating disorders in female athletes. *(From Sundgot-Borgen J: Disordered eating. In Ireland ML, Nattiv A (eds): The Female Athlete, Philadelphia: Saunders, 2002, p 242.)*

BOX 10-2 *Psychological and Behavioral Characteristics of Eating-Disordered Athletes*

Anxiety
Dieting (unnecessary for health, sport performance, or appearance)
Avoidance of eating and eating situations
Claims of "feeling fat" despite being thin[a]
Self-critical, especially concerning the body, weight, and sport performance
Resistance to weight gain or maintenance recommended by sport support staff
Unusual weighing behavior (i.e., excessive weighing, refusal to weigh, negative reaction to being weighed)
Compulsiveness and rigidity, especially regarding eating and exercise
Excessive or obligatory exercise beyond that recommended for performance enhancement
Exercising while injured (despite prohibitions by medical training staff)
Restlessness: relaxing is difficult or impossible
Change in behavior from open, positive, and social to suspicious, untruthful, and sad
Social withdrawal
Depression and insomnia
Secretive eating
Binge eating[b]
Agitation when bingeing is interrupted[b]
Evidence of vomiting unrelated to illness[b]
Excessive use of the restroom or "disappearing" after eating[b]
Use of laxatives or diuretics (or both) that is unsanctioned by medical or training staffs[b]
Substance abuse, whether legal, illegal, prescribed, or over-the-counter drugs, medications, or other substances[b]

[a]Especially for anorexia nervosa.
[b]Especially for bulimia nervosa.
Reprinted with permission from Sundgot-Borgen J: Disordered eating. In Ireland ML, Nattiv A (eds): The Female Athlete. Philadelphia: Saunders, 2002, p 243.

- Complete physical examination including pelvic examination in women with menstrual dysfunction
- Physical signs of eating-disordered athletes (Box 10-3)

Laboratory and Diagnostic Tests

- Complete blood count with differential (anemia)
- Electrolytes (abnormalities associated with use of diuretics, laxatives, self-induced vomiting, undernourishment)
- Blood urea nitrogen, creatinine (dehydration)
- Urine pregnancy test (amenorrhea)
- Calcium, ferritin, magnesium, phosphorus, albumin, total protein (nutritional laboratory tests)
- Erythrocyte sedimentation rate (ESR) and C-reactive protein (CRP) if applicable
- Thyroid function tests (amenorrhea)
- Electrocardiogram to rule out prolong QT interval or arrhythmia
- Urinalysis
- Stool guaiac (laxative abuse)

BOX 10-3 *Physical Signs and Symptoms of Eating-Disordered Athletes*

Dermatologic/Dental
Hair loss[a]
Dry skin, brittle hair and nails[a]
Lanugo[a]
Callus or abrasion on back of hand (from inducing vomiting)[a]
Dental and gum problems[a]
Cardiovascular
Bradycardia
Hypotension
Metabolic/Gastrointestinal
Gastrointestinal problems (i.e., constipation, diarrhea, bloating, postprandial distress) Swollen parotid glands[b]
Endocrine
Hypoglycemia
Low female sex hormone levels
Delayed onset of puberty[a]
Amenorrhea or menstrual dysfunction
Reduced bone mineral density
Stress fractures
Thermoregulation
Hypothermia[a]
Hematologic
Anemia
Fluids and electrolytes
Dehydration
Edema
Electrolyte abnormalities
Hypokalemia
Muscle cramps
Metabolic alkalosis
Others
Significant weight loss (beyond that necessary for adequate sport performance)[a]
Frequent and often extreme weight fluctuations[b]
Low weight despite eating large volumes[b]
Fatigue (beyond that normally expected in training or competition)
Muscle weakness
Hyperactivity

[a]Especially for anorexia nervosa.
[b]Especially for bulimia nervosa.
Sundgot-Borgen J: Disordered eating. In Ireland ML, Nattiv A (eds): The Female Athlete. Philadelphia: Saunders, 2002, p 242.

- Hormonal studies—follicle-stimulating hormone (FSH), estradiol (amenorrhea)
- Bone density evaluation—dual-energy X-ray absorptiometry (if more than 6 months of disordered eating, menstrual dysfunction, or stress fracture)
- Nutritional assessment

Treatment

PRINCIPLES

- Consider athlete "injured."
- Establish and maintain a therapeutic relationship by acknowledging the anxiety related to body image.
- Coordinate care with other physicians, dietitians, mental health professionals, dentists, schools, and coaches.
- Determine level of treatment (inpatient versus outpatient).

GOALS OF TREATMENT

- Regain and maintain healthy weight.
- Increase energy availability by increasing energy intake and/or reducing energy expenditure.
- Minimize restrictions on food choices.
- Minimize bingeing and purging behaviors.
- Educate regarding healthy eating and exercising.
- Facilitate engagement in treatment program.
- Treat physical manifestations of disordered eating.
- Treat underlying thoughts, attitudes, and psychiatric conditions that contribute to maladaptive behaviors.
- Provide counseling for family if necessary.
- Monitor for relapse.

INPATIENT TREATMENT

Box 10-4 lists criteria for consideration for inpatient treatment.

OUTPATIENT TREATMENT

- **Set a weight goal:** A general guideline, for a height of 5 feet estimate 100 pounds; add 5 pounds for each inch over 5 feet or subtract 5 pounds for each inch under 5 feet.
- **Behavior contract:** Mutually agreed upon treatment objectives can be determined and used to guide participation in sport. For example, once a body weight of 100 pounds is attained, athlete may start training or may compete if attends all scheduled appointments with all health care providers.

BOX 10-4 *Criteria for Consideration of Inpatient Treatment*

- weight < 85% of healthy body weight
- heart rate < 40 bpm
- blood pressure < 90/60 mm Hg (80/50 mm Hg if child or adolescent)
- glucose < 60 mg/dL
- potassium < 3 mEq/L
- significant arrhythmia
- repeated syncopal episodes
- severe electrolyte disturbances
- temperature < 97° F
- dehydration
- poorly controlled diabetes
- suicidal intent
- severe and disabling purging behaviors
- severe body image disturbance
- failure to respond to outpatient treatment after 3 months
- concurrent alcohol/drug abuse
- poor patient support system
- inadequate outpatient treatment facilities

- **Medications**
  - Multivitamin, calcium, vitamin D, iron
  - Hormone replacement therapy (amenorrhea)
  - Antidepressants
    - Selective serotonin reuptake inhibitors are most efficacious and useful with bulimics and obsessive-compulsive symptoms.
    - Avoid bupropion because of the risk of seizures.
    - Tricyclic antidepressants (TCAs) and monoamine oxidase inhibitors (MAOIs) could be toxic in underweight patients.
- **Referrals**
  - Multidisciplinary team
  - Nutritionist or dietician
  - Mental health practitioner
  - Dentist if required
- **Physician follow-up:** every 1 to 2 weeks, check vital signs, weight; laboratory tests (hemoglobin, electrolytes) if indicated

Exercise-Induced Menstrual Disorders

Definitions and General Concepts

DELAYED MENARCHE OR PRIMARY AMENORRHEA

- American Society of Reproductive Medicine defined delayed menarche as absence of menstruation by age 15 years in girls with secondary sex characteristics.
- The average age of menarche in athletic females is approximately 1 year later than in nonathletic females. This applies to women who commenced high-level training prior to and after menarche.
  - Competitive athletes report delayed onset of menarche even if intense training started after menarche. This suggests late menarche confers an advantage in athletic performance. Late maturing athletes have longer legs, narrower hips, less weight, and less body fat. These physical characteristics are conducive to better athletic performance. As well, there may be sociological factors at play.
  - Consequences of delayed menarche are not clear. Although an association with decreased peak bone mass has been found and greater incidence of stress fractures in this group has been reported, more research is required to clarify if a cause-and-effect relationship exists.

SECONDARY AMENORRHEA

- Secondary amenorrhea is defined as the absence of at least three consecutive menstrual cycles after menarche.
- Often thought to be a normal response of hard physical training and often a welcome break from the inconvenience of menstruation, **amenorrhea is not a normal response to training.**
- Amenorrhea occurs more frequently in sports emphasizing leanness.

LUTEAL PHASE DEFICIENCY OR SHORTENED LUTEAL PHASE

- Luteal phase deficiency is a shortening of the luteal phase to less than 10 days from the normal 14 days, resulting in decreased progesterone levels and anovulatory cycles.
- Menstrual cycle length is normal because of a slightly prolonged follicular phase. As a result the condition often goes unrecognized.
- Incidence in sedentary population was 9% and in recreational runners was 78% in a study of regularly menstruating women by DeSouza and colleagues.
- Luteal phase deficiency appears to be directly related to the amount of exercise. In runners, higher weekly mileage paralleled greater shortening of the luteal phase.

OLIGOMENORRHEA

- Menstrual cycle length greater than 35 days.
- May be anovulatory.

ANOVULATION

- Absence of ovulation.
- Estrogen and progesterone levels are low, therefore follicular development is impaired.
- Often there is sufficient estrogen to stimulate proliferation of the uterine lining for menstrual bleed.

Etiology of Exercise-Induced Menstrual Dysfunction

- Decreased pulsatile release of gonadotropin-releasing hormone (GnRH) from hypothalamus.
- Leads to decreased luteinizing hormone (LH) pulses from anterior pituitary, resulting in anovulation and low circulating estrogens.
- The currently accepted cause of menstrual dysfunction in athletes is the **energy deficit theory,** which states that if there are insufficient calories to meet energy requirements and provide the necessary carbohydrates to the brain, GnRH pulse generation is disrupted. The caloric deficit leads to low leptin levels, weight loss, metabolic hormone changes, and a state of energy conservation that results in reproductive function suppression and hypoestrogenism. Hence this form of amenorrhea is called **functional hypothalamic amenorrhea.** Other causes of amenorrhea must be excluded prior to making the diagnosis.
- Previously held notions of low body weight, low body fat, and stress of exercise have been replaced by the energy deficit concept stated above, which is also known as the **energy availability hypotheses.**
- In a study by Loucks and colleagues, the level of energy availability below which menstrual disorders are likely to occur is approximately 30 kcal/kg lean body mass per day. This equates to the energy expended in resting metabolism in healthy young adults. More than 5 days below this level disrupts LH pulsatility.

Consequences of Exercise-Induced Menstrual Dysfunction

DECREASED BMD

- The beneficial effects of exercise on bone formation can be disrupted by menstrual dysfunction, in particular, if amenorrhea has been present for longer than 6 months or if there has been frequent phases of oligomenorrhea.
- Athletes with menstrual dysfunction have a BMD significantly lower than eumenorrheic controls. There is a 10% to 20% decreased BMD in the lumbar spine of amenorrheic athletes. BMD at other sites has also been found to be significantly lower.
- Drinkwater and colleagues looked at lumbar bone density in amenorrheic and cyclic athletes. Previously amenorrheic athletes who became cyclic increased their bone mass but not to level of the athletes who had never been amenorrheic. Athletes who remained amenorrheic continued to lose bone density over the study period.
- The loss of BMD is proportional to the duration of menstrual dysfunction throughout the athlete's lifetime.
- High-impact exercise (especially basketball, volleyball, and gymnastics) may have a protective effect on bone health of female athletes. In one study, these athletes had higher BMD despite menstrual dysfunction.
- A 6% increase in vertebral bone density was found 15 months after resumption of menses in a study of amenorrheic athletes by Drinkwater and colleagues, but the gain ceased after 2 years. This raises the concern that these athletes may never reach peak bone mass.
- Eumenorrheic premenopausal women lose 0.3% of BMD per year. Amenorrheic, oligomenorrheic, or menopausal women lose up to 2% per year.

INCREASED RISK OF STRESS FRACTURES

- Other risk factors for stress fractures are late menarche, dietary insufficiency, genetic predisposition, biomechanical abnormalities, training errors, and bone geometry (narrow width and/or shorter length of tibia).
- A study of college female distance runners showed 48% amenorrheic athletes suffered stress fractures, compared with only 29% of regularly menstruating runners.
- Female athletes who never used oral contraceptives were twice as likely to suffer stress fracture compared with those who have used oral contraceptives.

INFERTILITY

Menstrual dysfunction can cause infertility as a result of lack of ovarian follicular development, ovulation, and luteal function.

HYPOESTROGENIC EFFECTS

- Impaired endothelium-dependent arterial vasodilation, which decreases muscle perfusion
- Impaired oxidative metabolism of skeletal muscle
- Greater low-density lipoprotein cholesterol levels
- Vaginal dryness

Evaluation of Menstrual Dysfunction in a Female Athlete

MEDICAL HISTORY

- **Menstrual history:** menarche, menstrual periods per year, flow length, last menstrual period, development of secondary sexual characteristics, family history of menstrual disorders
- **Training schedule:** sessions per week, miles per week, intensity, competitive activities
- **Diet:** calories per day, protein intake, calcium intake, disordered eating patterns, vegan diet
- Review of systems to rule out other causes of menstrual dysfunction (Fig. 10-3)
  - Galactorrhea, headaches, visual changes (prolactinoma)
  - Vasomotor symptoms, vaginal atrophy, dyspareunia (estrogen deficiency)
  - Hirsutism, oily skin, acne (androgen excess)
  - Cold intolerance, constipation, dry hair and skin (hypothyroidism)
  - Sexual activity history (pregnancy)
- **Drugs:** oral contraceptive pills, steroids, diuretics, laxatives, diet pills, thyroid medications, vitamins, illicit drugs, alcohol
- **Injury history:** stress fractures, overuse injuries
- **Psychological stress:** disordered eating behavior
- **Family history:** genetic defects, infertility, menstrual history, pubertal growth history

PHYSICAL EXAMINATION

- General appearance: physical manifestations of Turner's syndrome (e.g., short stature, webbed neck) or Cushing's disease (e.g., buffalo hump, central obesity), hirsutism, acne, dry skin
- Height, weight, BMI (growth delay)
- Fundi (papilledema)
- Thyroid (enlarged, nodules)
- Tanner staging
- Breasts (galactorrhea, development)
- Pelvic examination (gonadal dysgenesis, development)

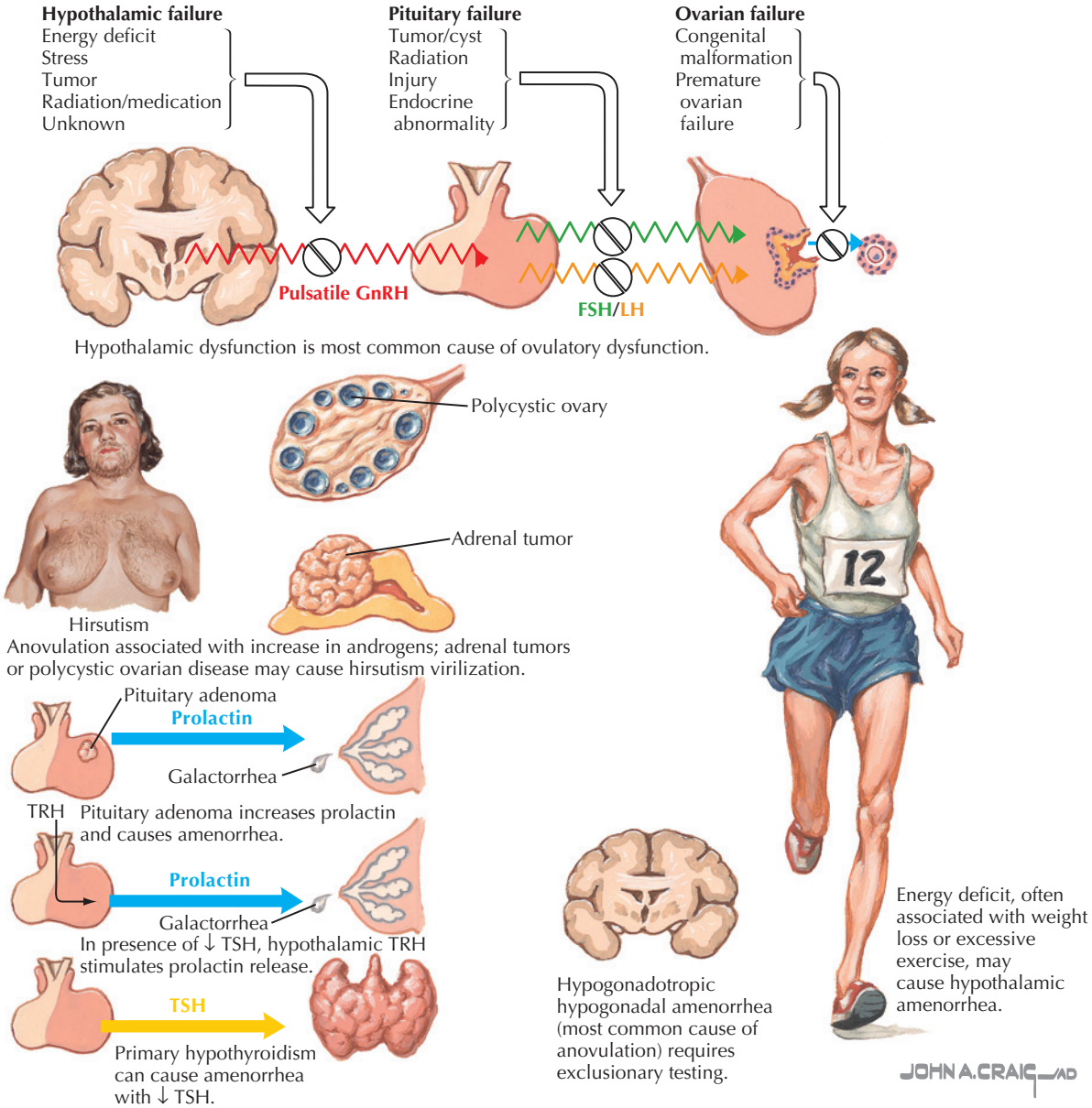

Figure 10-3 Causes of Menstrual Dysfunction.

LABORATORY AND DIAGNOSTIC TESTS

- Urine pregnancy test, thyroid-stimulating hormone (TSH), prolactin, FSH and LH
- Progestin challenge test:
  - Administer medroxyprogesterone acetate (Provera) 10 mg orally daily for 7 to 10 days.
  - Two to 7 days after challenge, if withdrawal menstrual bleed occurs, then test is positive, indicating adequate levels of circulating estrogen and no outflow tract obstruction.
- Estrogen/progesterone challenge test:
  - Administer if progestin challenge test is negative.
  - Administer conjugated estrogens (Premarin) 1.25 mg orally daily for 21 days, followed by Progestin challenge test to induce a withdrawal bleed.
  - If test is negative (no withdrawal bleed), test indicates an outflow tract obstruction. If positive (withdrawal bleed occurs), amenorrhea is caused by hypothalamic-pituitary axis dysfunction or ovarian failure.
- If signs of androgen excess, measure free testosterone and dehydroepiandrosterone sulfate.
- If a patient with a history of regular menstrual periods becomes amenorrheic during period of increased training:
  - Rule out pregnancy.
  - Consider hormone replacement therapy, if amenorrhea persists longer than 6 months or patient develops stress fracture.
  - Consider reduction in training intensity—if spontaneous menstruation returns, no further workup required.

Treatment

INCREASE ENERGY AVAILABILITY

- Increase caloric intake. May require nutritional assessment. In a study by Kopp-Woodroffe, menses were restored in amenorrheic runners when energy availability increased from an average of 25 to 30 kcal/kg of fat free mass.
- Decrease training if necessary.

HORMONE REPLACEMENT TREATMENT

- American Academy of Pediatrics recommends OCP use for treatment of amenorrhea if athlete is over the age of 16 years or if she is 3 years post menarche.
- Additional benefits of OCP use include decreases in dysmenorrhea, premenstrual syndrome, menorrhagia, iron-deficiency anemia, dysfunctional uterine bleeding, ovarian cysts, pelvic inflammatory disease, benign breast disease, ovarian cancers, endometrial cancers, and ectopic pregnancies. Possibly increase risk of breast and cervical cancer but controversial.
- Low-dose pills (20 to 35 μg ethinyl estradiol per day) have shown no association with weight gain or mood changes, and there is no evidence to support a deleterious effect on athletic performance.
- Screening prior to prescribing an OCP should include a personal and family history of cardiovascular risk factors and of breast cancer and a blood pressure measurement. Pelvic exam is not a prerequisite.
- For absolute contraindications for OCP from the World Health Organization (2004), see Box 10-5.
- Continuous-use OCP:
  - No 7-day pill-free interval each cycle, continuous use for several cycles without withdrawal bleed.
  - Option for women with premenstrual and perimenstrual symptoms or those who would prefer the convenience of eliminating cyclic bleeding.
  - Breakthrough bleeding decreases with time.

Bone Mineral Density

Definitions and General Concepts

- Bone health occurs along a spectrum that spans from good bone strength to low bone mass to osteoporosis.
- Implications of poor bone health include increased risk of stress fractures during the athlete's competitive career and fractures from falls in the postmenopausal years (Fig. 10-4).
- **Bone strength** is characterized by bone mineral content, BMD, and the quality of bone.
- Quality of bone is determined by the microarchitecture, geometry, and size of the bone.
- Bone remodeling is a dynamic process involving the breakdown of bone by osteoclasts (bone resorption) and laying down of new bone matrix or osteoid by osteoblasts (bone formation).
- BMD, although only one aspect of bone strength, is the current method being used to evaluate bone health, diagnose osteoporosis, and predict risk of fracture. BMD is assessed by dual-energy x-ray absorptiometry (DXA).
- Low BMD has been associated with eumenorrheic athletes with low energy availability and with amenorrheic athletes.
- Loss of bone mass should be assessed after a 6-month history of amenorrhea, oligomenorrhea, disordered eating or eating disorder, or after a stress or low-impact fracture.
- BMD loss can be irreversible.
- Further research is needed in defining measures of bone quality to be used with BMD to better characterize bone strength.
- Athletes participating in weight-bearing sports will have BMD measurements 12% to 15% greater than sedentary women.
- BMD has a normal distribution, which is divided in standard deviation (SD) units from either the mean of young healthy women (T distribution) or the mean for chronologic age (Z distribution).

BOX 10-5 *Absolute Contraindications for OCP from World Health Organization (2004)*

- < 6 weeks postpartum if breastfeeding
- hypertension (systolic blood pressure > 160 mm Hg or diastolic > 100 mm Hg)
- venous thromboembolism (current or past)
- ischemic heart disease
- history of cerebrovascular accident
- complicated valvular heart disease
- migraine headache with focal neurologic symptoms
- migraine headache without aura in women over age 35
- breast cancer (current)
- diabetes with end-organ involvement
- severe cirrhosis
- liver tumor
- active viral hepatitis
- women over age 35 who smoke (>15 cigarettes/day)
- known thrombogenic mutation

From World Health Organization: Improving Access to Quality Care in Family Planning: Medical Eligibility Criteria for Contraceptive Use, 3rd ed. Geneva: WHO, 2004.

Classification of Low Bone Mass and Osteoporosis

WORLD HEALTH ORGANIZATION

- T-scores (average peak adult BMD) are used to diagnose osteoporosis in postmenopausal women.
- Fracture risk is predicted by T-scores and doubles for every SD below the mean of young healthy women.
- WHO classification of BMD is based on DXA (Table 10-3).

INTERNATIONAL SOCIETY FOR CLINICAL DENSITOMETRY (ISCD) CLASSIFICATION

- Z-scores, based on chronologic age, are used for BMD evaluation in premenopausal women, adolescents, and children.
- Z-scores are better predictors of BMD in women younger than 20 years of age because peak bone mass has not yet been attained.
- Z and T distributions are similar with increasing age of women.
- The following BMD classification terminology is used by the ISCD for premenopausal women and children:
  - For ages older than 20 years, BMD is below the expected range if Z-score is less than or equal to −2.
  - For ages younger than 20 years, BMD is low for chronologic age or below the expected range if Z-score is less than or equal to −2.

AMERICAN COLLEGE OF SPORTS MEDICINE (ACSM) CLASSIFICATION

- ACSM classification is similar to ISCD classification but takes into consideration that athletes have 5% to 15% higher BMD than normal controls.
- The following BMD classification terminology is used by ACSM:
  - BMD is considered low if secondary clinical risk factors for fracture exist and Z-score is between −1 and −2.
  - Osteoporosis risk exists if there are secondary clinical risk factors for fracture and the BMD Z-score is less than or equal to −2.
  - Secondary clinical risk factors for fracture include history of nutritional deficiencies, hypogonadism, glucocorticoid exposure, hypoestrogenism, and stress fractures.

Etiology of Low Bone Mass in the Female Athlete

- Low bone mass in the female athlete may be attributable to failure to attain peak bone mass or to loss of bone mass over

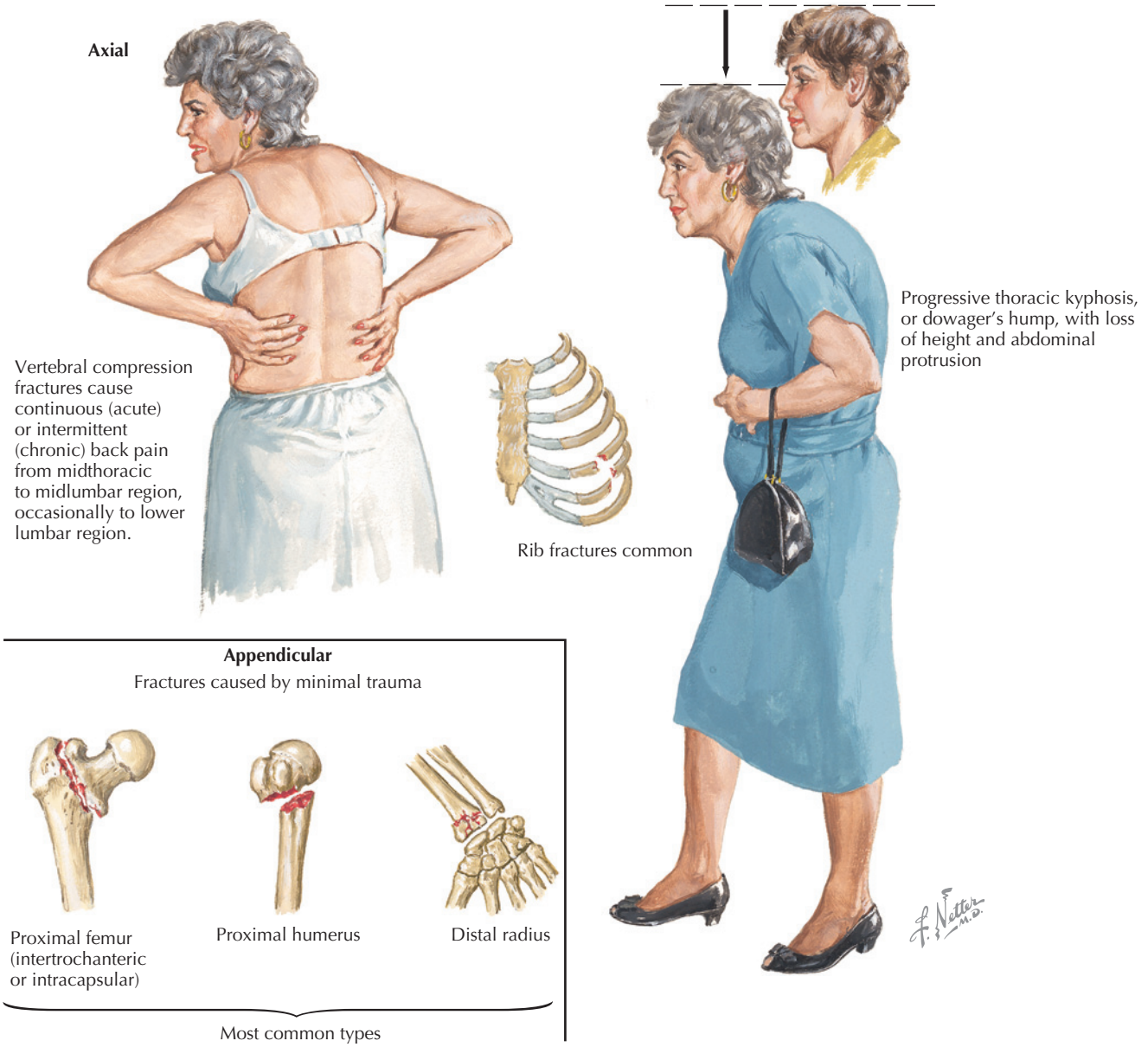

Figure 10-4 Clinical Manifestations of Osteoporosis in Postmenopausal Women.

time. Severe undernutrition and menstrual dysfunction impairs skeletal health.

- Menstrual disorders with a hypoestrogenic state result in accelerated bone resorption, because of the loss of the suppressing effect of estrogen on osteoclast activity.
- Low energy availability may also be a factor, as indicated in studies that found that athletes with disordered eating had low BMD in the absence of menstrual dysfunction. Furthermore, estrogen replacement therapy alone, with no increase in energy intake, did not restore BMD.
- Energy deficit results in metabolic changes mediated by insulin, growth hormone, insulin-like growth factor-1, leptin, cortisol and thyroid hormone, which are all important in bone metabolism and may be factors independent of estrogen status in affecting bone mass.

Risk Factors for Osteoporosis

- Age
- Small, thin frame (body weight less than 127 pounds)
- Smoking
- Use of oral corticosteroid therapy for more than 3 months
- Estrogen deficiency
- Lifelong low calcium intake, lactose intolerance
- Inadequate weight-bearing physical activity
- History of fragility fracture in first-degree relative
- Genetics (Caucasian or Asian)
- Alcohol in amounts greater than two drinks per day

Evaluation of Low Bone Mineral Density

- DXA scan—both spine and hip should be measured. Use the lowest BMD Z-score of either posterior-anterior (PA) spine or the hip (using femoral neck or total hip). In patients younger than 20 years old, recommended sites are PA spine and whole body.
- Rule out other causes of osteoporosis (Fig. 10-5).
- Laboratory testing: serum calcium, phosphorus, and magnesium; thyroid and parathyroid hormone; complete blood cell count; kidney function and liver enzymes; serum 25-

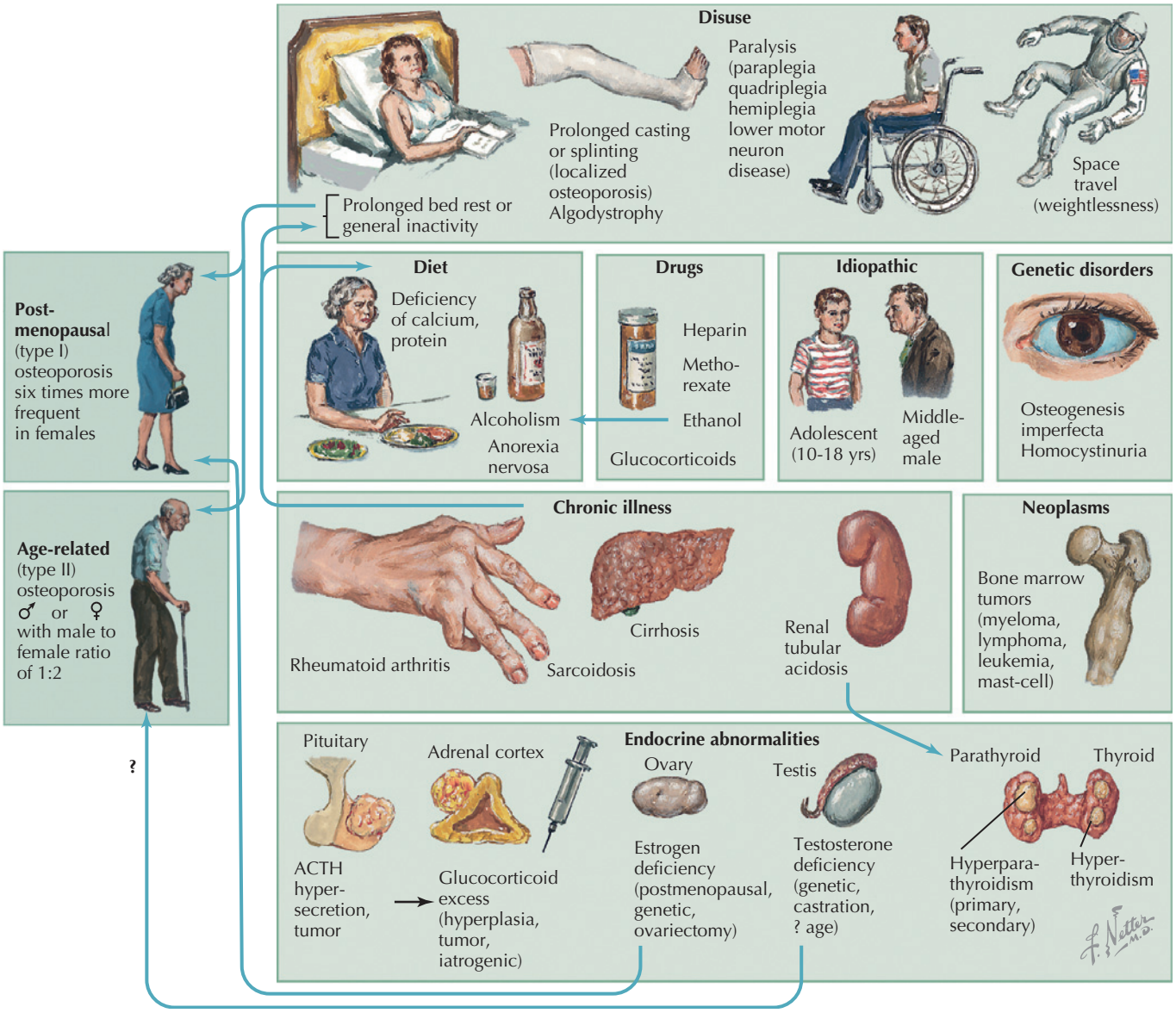

Figure 10-5 Causes of Osteoporosis.

hydroxyvitamin D3; celiac sprue autoantibodies, and 24-hour urinary calcium excretion.

Treatment of Low Bone Mineral Density

- Important to initiate treatment within first year of onset of amenorrhea when rapid bone loss occurs.
- Baseline BMD is useful to measure response to treatment.
- Evaluate nutrition and correct energy deficits by decreasing training or increasing energy intake to allow menses to resume.
- There is an association between increase in body weight and increase in BMD; the extent of this association requires further study.
- Oral contraceptives—evidence for positive effect on BMD is good in perimenopausal women, fair in hypothalamic oligo/amenorrheic premenopausal women, limited in anorexic premenopausal women, and limited in healthy premenopausal women.
- In athletes with functional hypothalamic amenorrhea:
  - If over age 16 and BMD is decreasing despite adequate nutrition and body weight, OCP should be considered for prevention of further bone loss.
  - If less than 16 years of age, there is no evidence to support the use of OCP for restoration and maintenance of bone health and there is concern regarding premature closure of growth plates.
- Nasal spray calcitonin has been shown to be effective in preventing further decrease in BMD in one small study; further studies are required to evaluate efficacy.
- Calcium 1500 mg daily and vitamin D 400 to 1000 IU daily.
- Weight-bearing exercise and resistance training.
- Smoking cessation and reduction of excessive alcohol consumption.
- Bisphosphonates and selective estrogen receptor modulators are contraindicated in premenopausal women because of potential negative effects during pregnancy. Long-term effects of bisphosphonates in women of childbearing age are not known. Adverse fetal effects have been shown in animal studies.
- Synthetic human parathyroid hormone 1-34 leads to skeletal remodeling and an increase in BMD; categorized as class C for pregnant women.

EXERCISE AND PREGNANCY

General Concepts

- Exercise during a **normal pregnancy** should be encouraged as part of a healthy lifestyle during pregnancy. For normal pregnancies, moderate-intensity exercise on a regular basis has no risks to the fetus and has maternal benefits.
- Goals of exercise during pregnancy should be to exercise safely while maintaining maternal fitness levels and minimizing risk to developing fetus.
- Exercise prescription should be individualized and adapted to each phase of pregnancy.
- American College of Obstetricians and Gynecologists (ACOG) has established guidelines for exercise during pregnancy, which were updated in 2002. Evidence for guidelines is based on animal studies and expert consensus opinion given the difficulty of conducting ethical randomized trials on pregnant women. Box 10-6 lists the ACOG's contraindications to aerobic exercise during pregnancy.

Benefits of Exercise during Pregnancy

- Same benefits as in nonpregnant state
- Prevention of gestational diabetes, especially if BMI is greater than 33
- Decrease incidence of pregnancy-induced hypertension (PIH) and physical symptoms of pregnancy such as nausea, vomiting, etc.
- Improve or maintain fitness
- Decrease musculoskeletal complaints
- Decrease depression and anxiety
- Improve self-esteem
- Decrease maternal weight gain
- Decrease postpartum recovery time

BOX 10-6 *Contraindications to Aerobic Exercise during Pregnancy (ACOG 2002)*

Rights were not granted to include this textbox in electronic media. Please refer to the printed publication.

Effect of Exercise on Pregnancy and Fetus

- In well-conditioned women, there is no increased incidence of preterm labor and premature rupture of membranes (PROM). Labor is shorter, with less need for obstetrical intervention and fewer signs of fetal distress. Delivery is 5 to 7 days earlier if the mother is doing weight-bearing exercise at or above training level.
- Epidemiologic studies suggest link between lack of physical activity, deficient diets, and intrauterine growth restriction (IUGR). Other studies demonstrate that mothers who engage in moderate- to high-intensity exercise give birth to healthy infants who weigh less because of decreased body fat in the newborn and the earlier delivery.
- No adverse pregnancy outcomes related to exercise have been reported. Many of the potential risks, for example, reduced transplacental oxygen, are hypothetical. There have been no reported cases of teratogenesis resulting from exercise-induced hyperthermia in pregnancy.

Exercise Prescription during Pregnancy

- In a normal pregnancy, recommend 30 minutes or more of moderate exercise a day on most, if not all, days of the week.
- If medical or obstetric complications present, recommend seeing family doctor or obstetrician for advice first.
- Avoid activities with high risk of falling (e.g., horse riding, downhill skiing).
- Avoid activities with high risk of abdominal trauma (e.g., ice hockey, soccer).
- Avoid scuba diving because of risk of decompression sickness in the fetus.
- Avoid exertion at altitudes greater than 6000 feet and be familiar with the signs and symptoms of altitude sickness.
- Avoid supine position during exercise after the first trimester. The gravid uterus can cause a relative obstruction of venous return resulting in decreased cardiac output and hypotension. Prolonged Valsalva maneuvers can produce a similar situation, so exercise caution with breath holding during weight training.
- Avoid prolonged motionless standing, which can also be associated with a decrease in cardiac output and hypotension.
- Prepregnancy exercise routines may be gradually resumed after delivery. There are no known complications of rapid resumption of exercise, but most women undergo some degree of detraining and may require 4 to 6 weeks during the postpartum period to reestablish their normal physiology.
- The following are warning signs to terminate exercise while pregnant:
  - Vaginal bleeding
  - Dyspnea before exertion
  - Dizziness
  - Headache
  - Chest pain
  - Muscle weakness
  - Calf pain or swelling (rule out thrombophlebitis)
  - Preterm labor
  - Decreased fetal movement
  - Amniotic fluid leakage

RECOMMENDED READINGS

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The Senior Athlete

11

Eric J. Anish

GENERAL CONSIDERATIONS

Demographics

- In Western society, the population of older adults is growing both in size and in proportion to the total population.
- It is estimated that by year 2030, the number of people older than 65 will approach 70 million in the United States.
- People over the age of 85 are expected to comprise the fastest growing segment of the population.

Physical Activity and Health Promotion

- For many adults, aging brings a general decline in health and functional capacity.
- Substantial evidence demonstrates that many health problems faced by older adults are related to some extent to a decline in physical activity.
- Regular physical activity is recommended for older adults, including the very elderly.
- Health care providers are challenged to promote physical and emotional well-being of an aging population.
- Recommendations from the American College of Sports Medicine (ACSM) and the American Heart Association (AHA) encourage older adults to maintain a physically active lifestyle with an emphasis on moderate-intensity aerobic activity and muscle-strengthening activity. Activities that maintain or increase flexibility are also recommended, as well as balance exercises for older adults at risk of falls (Box 11-1).
- Although recent data show that most older adults remain sedentary, increasing numbers are beginning to incorporate regular physical activity into their lives.

Competitive Sports

- Although older Americans are the least physically active of any age group, many older adults do participate in competitive sports.
- Competitive senior athletes are those who participate on an organized team or in an individual sport that requires regular competition against others as a central component, places a high premium on excellence and achievement, and requires some form of systematic (and usually intense) training.
- The number of older athletes participating in competitive sports over the past two decades has increased dramatically.
- An example of this growth can be seen in the number of athletes participating in the **National Senior Games,** which features athletes ages 50 years and above.
  - In 1987, the first National Senior Games, held in St. Louis, Missouri, attracted close to 2500 competitors.
  - The 2007 National Senior Games, held in Louisville, Kentucky, attracted more than 12,000 participants.
- The increasing number of older adults participating in athletic training at a level beyond that which is necessary for basic fitness can present health care providers with the challenge of providing appropriate guidance.

Decline in Athletic Performance with Aging

- Many athletes who continue to train and compete into their older years observe a decline in performance.
- From the mid-30s to approximately 60 years of age, a slow but progressive decline in athletic performance is seen in most athletes.
- An accelerated loss in performance ability is observed in athletes after 60 years of age.
- A larger decline in performance is seen in older female athletes compared to men (Fig. 11-1).
- The decline in athletic performance with aging is related to a multitude of factors including the following:
  - Inevitable physiologic changes that occur with aging, such as decreases in muscle mass and maximal oxygen uptake (see "Effects of Aging on Specific Physiologic Systems")
  - Impact of general health issues, because many medical problems become more prevalent after the fifth decade of life
  - Difficulty maintaining optimal training, which may be influenced by:
    - Underlying medical conditions
    - Musculoskeletal injuries (including the accumulation of residual injuries over many years)
    - Reduced hormonal concentrations, which can influence the physiologic response to exercise
    - Need for longer recovery times from hard training sessions
    - Changes in competitive motivation
    - Lack of available time for training and competition (e.g., job and family commitments)

PHYSIOLOGIC CHANGES ASSOCIATED WITH AGING

General Considerations

- Some controversy surrounds the nature of the physiologic changes associated with aging.
- Some changes result not merely from aging, but from declining levels of physical activity as people grow older.
- Sedentary older adults undergo more significant changes in body composition and declines in functional capacity than adults who remain physically active as they age.
- Many physiologic changes that occur with aging can be limited, prevented, or even reversed through sustained physical activity and exercise training.
- Older athletes maintain the capacity to adapt to exercise training and can improve strength, endurance, flexibility, and athletic performance.

Effects of Aging on Specific Physiologic Systems

Cardiac: decreases in maximal heart rate, stroke volume, maximal cardiac output, maximal arteriovenous oxygen difference, and maximal oxygen uptake ($\dot{V}O_2$ max); increase in systolic and diastolic blood pressure.

Pulmonary: decrease in vital capacity; increases in residual volume, respiratory frequency, and work of breathing (related to increased rib cage rigidity and decreased lung tissue elasticity).

Musculoskeletal: decrease in muscle mass (sarcopenia), including decreases in types I and II muscle fibers (type II decreases more than type I); decreases in bone mass and tensile strength of tendons and ligaments; increases in stiffness of muscles, tendons, and ligaments; articular cartilage weakness secondary to decreased mucopolysaccharides and increased water content.

Hematologic: decreases in plasma volume, red cell mass, and blood volume.

BOX 11-1 *Summary of Physical Activity Recommendations for Older Adults*

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Neurologic: decreases in spinal motor neurons (contributing factor to decreased muscle strength), reaction times, coordination, balance, and proprioception.

Metabolic: decreases in basic metabolic rate and glucose tolerance; increases in plasma triglycerides, total cholesterol, and low-density lipoprotein concentrations.

BENEFITS OF EXERCISE IN OLDER ADULTS

- In addition to slowing the decline in many age-related physiologic changes (such as a decrement in $\dot{V}O_2$ max and loss of lean body mass), physical activity and exercise training can play a role in the prevention of numerous disease states that are common in older individuals including coronary artery disease (CAD), hypertension, dyslipidemia, obesity, cerebrovascular disease, cancer (including colon, breast, prostate, and lung), type 2 diabetes mellitus, osteoporosis, depression, and anxiety (Fig. 11-2).
- Additional exercise-related health benefits include reduced all-cause mortality, improved postural stability, improved cognition, delayed functional disability, enhanced self-image, improved appearance, improved sleep habits, enhanced psychological well-being, reinforcement of other healthy lifestyle changes (e.g., better diet, smoking cessation).

RISKS OF EXERCISE IN OLDER ATHLETES

General Considerations

- Although regular exercise and sports participation can provide numerous health benefits, these activities may be associated with a variety of health risks.
- Although sports-related safety issues exist regardless of age, older adults are faced with certain age-specific challenges.

Orthopedic Injury

- Age-related changes in muscle, bone, tendon, and ligament predispose older athletes to an increased frequency of musculoskeletal injuries (see "Effects of Aging on Specific Physiologic Systems").

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Figure 11-1 Decline in athletic performance with aging. Schematic pattern (based on data from multiple sources) of decreases in best performances by age in running (10-km), swimming (1500-m), and weightlifting. *Closed* symbols represent men, and *open* symbols represent women. With the exception of swimming, the loss of performance is typically somewhat more rapid in women. It is unclear whether this is a primary sex-related difference or related to the smaller relative participation rate by women. After age 60, the rate of deterioration may increase in both men and women. *(From Foster C, Wright G, Battista RA, et al: Training in the aging athlete. Curr Sports Med Rep 6:200-206, 2007.)*

- Previous joint injuries and underlying osteoarthritis, which is more common in older adults, are risk factors for activity-related injury.
- Sensory impairment, such as alterations in vision, hearing, vestibular function, and proprioception, may increase susceptibility to accidents and falls.
- Overuse injuries remain the most common cause of athletic disability in athletes older than 50.
- As in younger athletes, training errors frequently contribute to the development of overuse injuries.

Common Musculoskeletal Problems

MUSCLE STRAINS

- Reported by many researchers to be the most common injury among older athletes.
- A majority of these injuries are acute in nature.
- More common in strength-and-power sports.
- Increased muscle stiffness with aging contributes to risk.

TENDINOPATHIES

- Common sites of involvement include knee (e.g., patellar tendon), shoulder (e.g., rotator cuff), and Achilles tendon.
- Age-associated alterations in biochemical and biomechanical properties of tendons decrease flexibility and tensile strength and increase susceptibility to injury.
- Degenerative changes within substance of tendon can occur with repetitive loading.
- Age-related decreases in blood supply to certain regions may contribute to increased risk of tendon injury (e.g., supraspina-

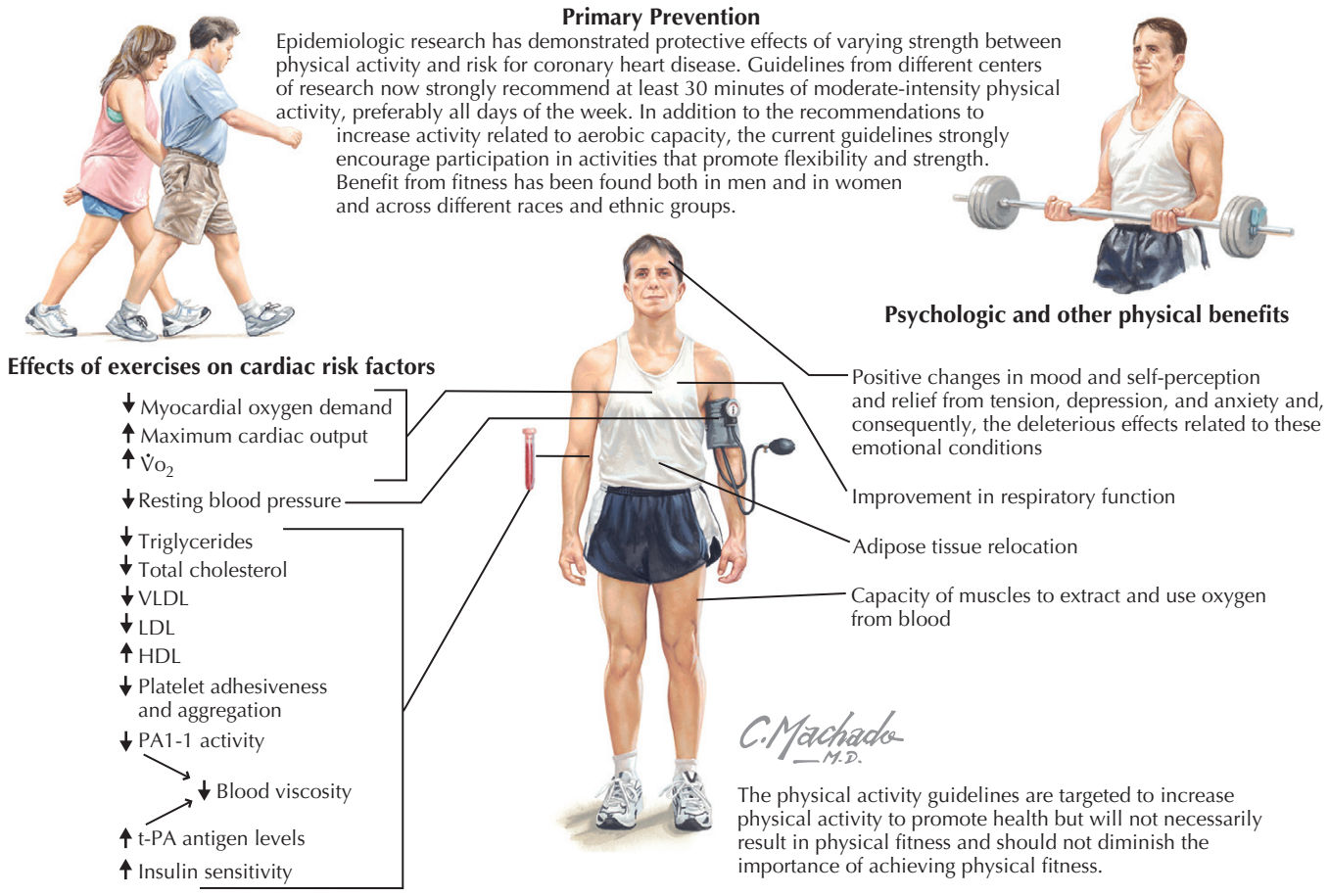


Figure 11-2 Effects of Exercise on Cardiovascular Health.

tus tendon near its insertion; Achilles tendon 4 to 5 cm proximal to calcaneal insertion).

MENISCAL CARTILAGE INJURIES

- Acute meniscal tears often occur as a result of a twisting injury or knee hyperflexion.
- Individuals with degenerative meniscal tears often have a history of minimal or no trauma and commonly have associated osteoarthritis of the knee.
- Degenerative meniscal tears are increasing in frequency as older individuals are becoming or staying active in athletic activities.
- Susceptibility to such injury may be related to changes in collagen proteoglycan matrix that occur with aging.

LUMBAR DISC DISEASE

- Acute lumbar disc herniations are most common in middle age, but may occur in older athletes.
- Lumbar disc degeneration is more common in older athletes.
- With aging, disc become avascular and lose water content; degeneration of viscoelastic and structural properties predisposes to degenerative disc disease.
- Loss of disc height can contribute initially to spine instability and, subsequently, degenerative lumbar stenosis (Fig. 11-3).

OSTEOARTHRITIS

- Osteoarthritis is the most common joint disorder in the United States, affecting more than 60% of adults over 50. This prevalence increases to 85% in the group 75 and older.
- Age is the most consistent risk factor for radiographic and symptomatic osteoarthritis at all articular sites.
- With aging, articular cartilage can weaken as the amount of mucopolysaccharides decreases and water content increases (Fig. 11-4).
- Prior injuries that change the joint architecture (e.g., anterior cruciate ligament tear or meniscal tear) can contribute to the future development of arthritis.

Temperature-Related Injury

- Many physiologic changes that occur with aging influence an older person's ability to adapt to change in temperature, particularly while exercising.
- Older athletes are more susceptible to both heat-related and cold-related injuries.
- Factors affecting thermoregulation that may contribute to a heightened risk of temperature-related injury in older athletes include:

Heat Injury

- Increased propensity for dehydration resulting from impaired thirst mechanism, increased water output by kidneys, and decreased total body water
- Decreased sweat gland function
- Attenuated increases in skin blood flow in response to elevation in core temperature
- Effects of medications
  - Beta-blockers: decreased sweating
  - Diuretics: sodium loss and decreased intravascular volume
  - Antihistamines: decreased sweating
  - Tricyclic antidepressants: decreased sweating
  - Monoamine oxidase inhibitors: increased endogenous heat production
  - Phenothiazines: decreased thirst

Cold Injury

- Impaired ability to perceive ambient air temperature adequately
- Attenuated vasoconstrictor response
- Autonomic dysfunction

Figure 11-3 Degenerative Hypertrophic Spondylitis (Osteoarthritis).

- Decreased capacity for shivering thermogenesis (resulting from decreased muscle mass, decreased basal and resting metabolic rates, and effects of medications such as phenothiazines and benzodiazepines)

Cardiovascular Disease

- Preexisting cardiovascular conditions may pose serious risks for older athletes, particularly in terms of exercise-related mortality.
- Compared with younger population, senior athletes are more likely to be affected by conditions associated with exercise-related cardiovascular complications.
- Atherosclerotic CAD is the most common cause of sudden death during exercise in older adults (Fig. 11-5).

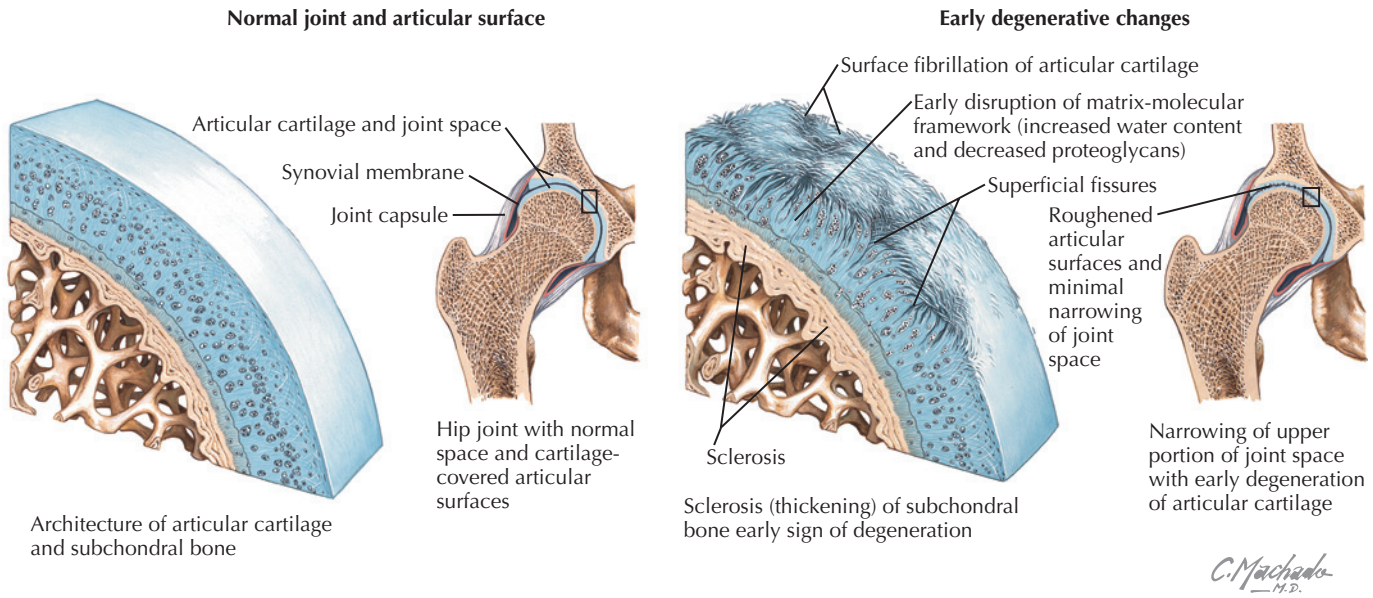
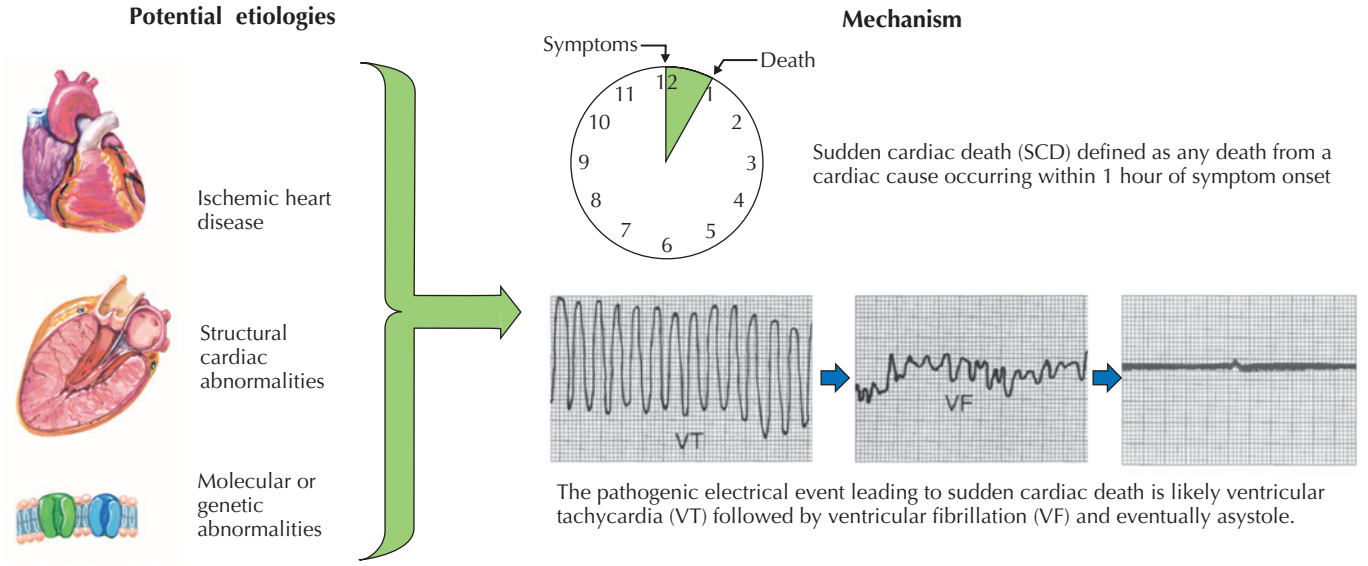


Figure 11-4 Joint and Articular Changes.



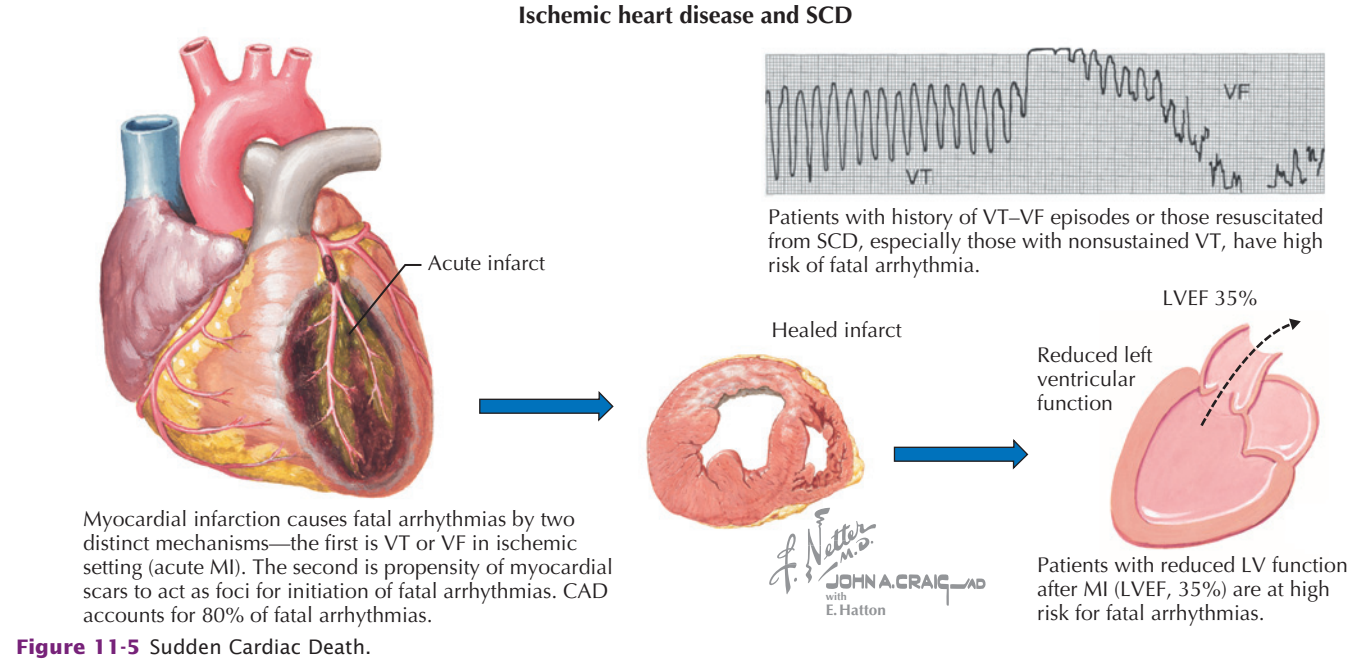


Figure 11-5 Sudden Cardiac Death.

- Other cardiovascular conditions that pose exercise-related risks include valvular heart disease, uncontrolled hypertension, cardiomyopathies, cardiac arrhythmias, and decompensated heart failure.
- Medical problems commonly seen in older adults, such as diabetes mellitus and obesity, may negatively affect cardiovascular function. Comorbid conditions need to be considered in estimating cardiovascular risk with athletic participation (Fig. 11-6).
- Extent of cardiovascular risk also depends on severity of underlying disease and specific type and intensity of athletic participation. In general, the more vigorous the exercise, the greater the cardiovascular risk.
- People who engage in vigorous physical exercise have a slightly increased risk of sudden cardiac death during activity. Risk is greatest among sedentary people performing sudden, unregulated, strenuous exercise. Compared with people with habitual low levels of physical activity, people who exercise regularly have a lower risk of exercise-related sudden death.

Drug–Exercise Interactions

General Considerations

- Prescription and over-the-counter drug use is widespread in older adults. Advancing age is associated with an increased usage of all medications and polypharmacy is quite common.
- Many drugs have physiologic effects that may impair exercise performance and increase the likelihood of certain sports-related health risks. Drugs commonly used by older adults that may pose problems include:
  - **Diuretics** impair thermoregulation, exacerbate exercise-related dehydration, decrease stroke volume, cause reflex increase in total peripheral resistance (which may decrease muscle blood flow), and contribute to electrolyte depletion.
  - **Beta-blockers** decrease cardiac output, reduce maximal heart rate, decrease $\dot{V}O_2$ max, may induce bronchoconstriction, and decrease lipolysis and glycogenolysis.
  - **Calcium channel blockers** increase risks of postexercise orthostatic hypotension and exercise-related gastroesopha-

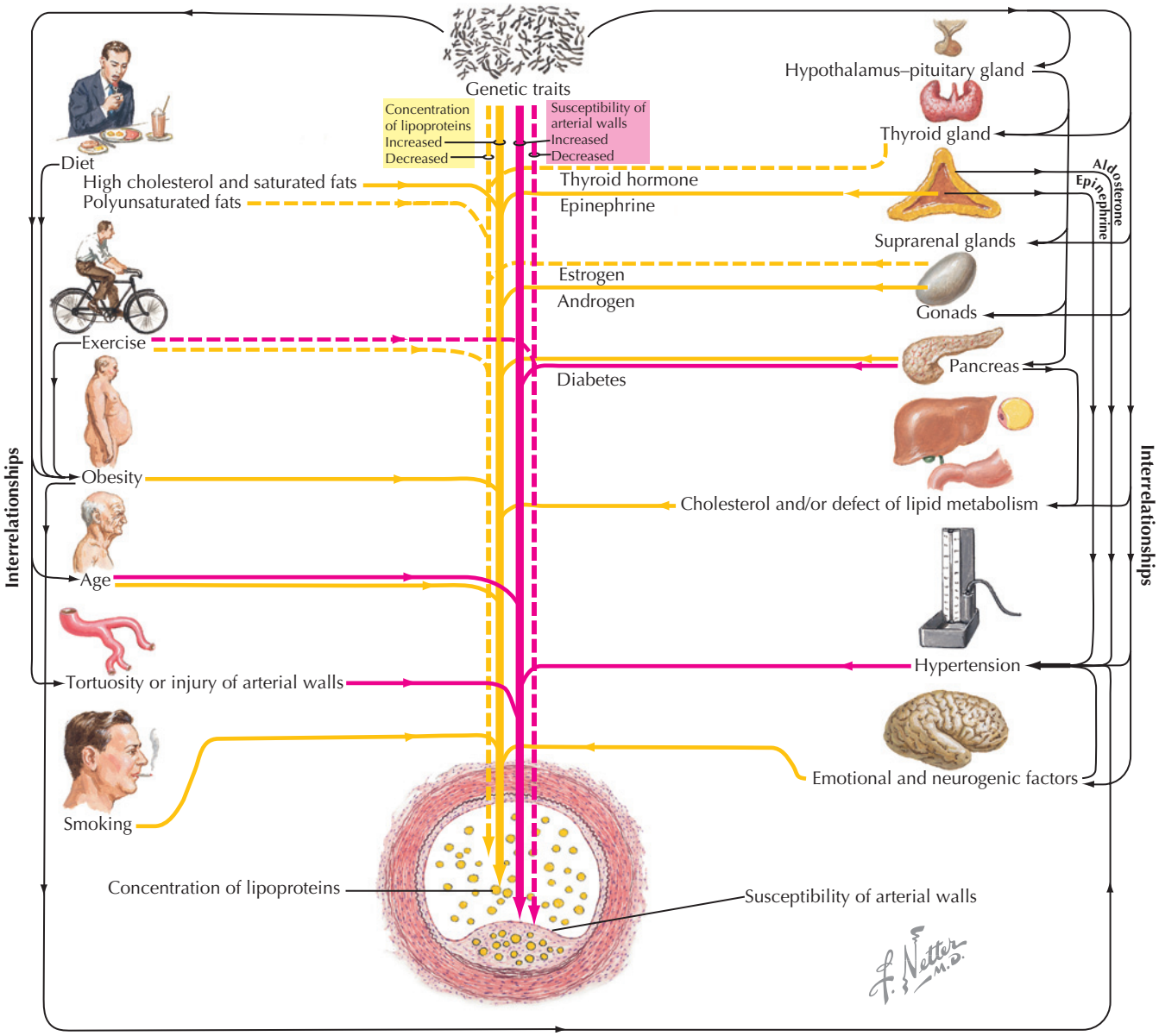


Figure 11-6 Cardiac Risk Factors.

geal reflux; certain agents (e.g., verapamil) may impair heart rate response to exercise.
- **Statins** (3-hydroxy-3-methyl-glutaryl coenzyme A [HMG-CoA] reductase inhibitors) can produce a variety of skeletal muscle disorders including myositis, rhabdomyolysis, mild serum creatinine kinase (CK) elevations, myalgia with and without elevated CK levels, muscle weakness, muscle cramps, and persistent myalgia and CK elevations after statin withdrawal. Statins have been shown to exacerbate exercise-induced skeletal muscle injury. As a result, it may be reasonable to discontinue stain drug use for 48 hours prior to an extremely demanding athletic activity such as participating in marathon running.
- **Insulin** requirements may be altered by exercise as a result of improved insulin sensitivity, enhanced absorption of exogenous insulin, and improved glucose tolerance.
- **Oral hypoglycemic agents** may require dosage adjustments secondary to improved glucose tolerance with exercise.
- **Tricyclic antidepressants** promote orthostatic hypotension, may be sedating, and increase risk of cardiac arrhythmias.
- **Glucocorticoids** cause skin atrophy and fragility, resulting in increased risk of traumatic injury; contribute to osteoporosis, resulting in increased risk of stress fractures; and may result in drug-induced myopathy.

PREPARTICIPATION MEDICAL EVALUATION (PPE)

General Considerations

- Older adults should undergo a medical evaluation appropriate to their circumstances before initiating any exercise program.
- The extent of evaluation depends on various factors, including patient's age, underlying medical problems, and planned exercise intensity.
- Primary goals of the preparticipation medical evaluation (PPE) include identification of underlying medical conditions that may limit ability to exercise or increase risk of participation.
- A major objective of this evaluation is to identify, or raise suspicion of, occult cardiovascular disease, such as CAD, that could result in sudden cardiac death, myocardial infarction, heart failure, or cardiac arrhythmia that could be triggered by intense athletic activity.
- In athletes with known cardiovascular disease, the PPE can be used to determine whether continued athletic participation is prudent and consistent with the extent of their disease.
- The PPE can also be used to assess fitness so that appropriate exercise counseling can be provided, including determination of appropriate physical activity (type, frequency, intensity, duration) and setting of exercise goals.

Preparticipation Medical History

- Active medical conditions
- Previous medical problems
- Medication use that might alter exercise response and require modification of exercise prescription
- Tobacco and alcohol use
- Family history of cardiovascular disease
- Assessment of nutritional status
- Previous exercise program
- Current activity level and functional capacity
- Present exercise regimen, including type, intensity, frequency, and duration
- Review of **signs and symptoms suggestive of underlying cardiovascular disease,** such as chest pain, palpitations, shortness of breath, dyspnea on exertion, lightheadedness or dizziness with exertion, syncope, paroxysmal nocturnal dyspnea, lower limb edema, and claudication
- Review of **signs and symptoms suggestive of underlying musculoskeletal disease,** such as joint pain, joint swelling, "mechanical" joint symptoms (e.g., catching or locking), limited joint range of motion, and weakness

Preparticipation Physical Exam

Preparticipation physical exam should include height and weight, blood pressure, resting pulse, cardiac auscultation, lung auscultation, palpation of peripheral pulses, evaluation of joints (range of motion, flexibility, strength), and assessment of gait, balance, and coordination.

Preparticipation Diagnostic Testing

Preparticipation diagnostic testing is not indicated in all patients but should be based on medical history, physical exam findings, medication use, and the anticipated intensity of the athletic activity in which the individual will be participating.

Examples of Testing

- Electrolytes (with diuretic use)
- Urinalysis (with history of renal disease)
- Lipid analysis (for evaluation of CAD risk factors)
- Body composition analysis (to help establish exercise goals and assess response to training)
- Bone density testing (to assess risk of fracture and provide baseline for measuring improvements, especially in patients with risk factors for osteoporosis, such as estrogen deficiency, Caucasian race, low body weight and body mass index, positive family history, smoking, prior fracture, oral corticosteroid or anticonvulsant use, inadequate calcium and vitamin D intake)
- Electrocardiogram (to identify evidence of an old myocardial infarction and certain cardiac conditions seen less frequently in older athletes such as hypertrophic cardiomyopathy; long-QT, Brugada, and Wolff-Parkinson-White syndromes; and arrhythmogenic right ventricular cardiomyopathy)

Exercise Testing

- Exercise testing is often recommended for older adults to attempt to detect underlying CAD and help assess safety of participation. However, formal exercise testing in older adults remains controversial.
- Exercise testing can identify older adults at increased risk of symptomatic or silent cardiac ischemia or malignant ventricular arrhythmias. Diagnostic yield is greatest in symptomatic adults and in those with intermediate pretest probability (i.e., 10% to 90%) of CAD.
- In asymptomatic adults, exercise stress testing is a poor predictor of major cardiac complications during exercise (e.g., acute myocardial infarction, sudden cardiac death).
- Positive exercise test typically requires hemodynamically significant coronary artery stenosis, whereas acute coronary events often occur at site of previously nonobstructive atherosclerotic plaques. Thus, normal ECG response to exercise can be seen in adults who subsequently experience cardiovascular complications during exercise.
- The American College of Sports Medicine (ACSM) stratifies adults into low-, moderate-, and high-risk groups (Box 11-2). The ACSM recommends exercise testing for adults based on level of risk and intensity of planned exercise activity and encourages special considerations for exercise testing in elderly adults (Table 11-2 and Box 11-4).
- The World Heart Federation, the International Federation of Sports Medicine, and the American Heart Association Committee on Exercise recommend that masters athletes with a moderate-to-high cardiovascular risk profile for coronary artery disease, and who desire to enter vigorous competitive situations, undergo exercise testing. This would

BOX 11-4 *Special Considerations for Exercise Testing in Elderly Adults*

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- Studies have demonstrated that the total amount of coronary calcium predicts coronary disease events beyond standard risk factors.
  - A high calcium score (>100) is associated with a high risk of a cardiac event in the next 2 to 5 years.
  - A negative test is associated with an extremely low risk (0.1% per year) of a cardiovascular event in the next 2 to 5 years.
- It is unknown whether the risk of coronary events during intense exercise is increased in the setting of higher calcium scores.
  - Because a negative test (calcium score = 0) makes the existence of both atherosclerotic plaque and significant luminal obstructive disease highly unlikely (negative predictive value 95% to 99%), an older athlete with a negative EBCT would theoretically appear to be at low risk of suffering an exercise-related coronary event.
  - In contrast, the presence of a high calcium score (calcium score > 100), and its strong association with total coronary atherosclerotic disease burden, would suggest that an older athlete is at an increased risk for an exercise-related cardiac event.
  - It has been recommended that athletes with coronary artery calcification scores greater than 100 should undergo the same evaluation as individuals with more clinically apparent CAD.
- In addition to a lack of prospective data demonstrating the benefits of EBCT as a preparticipation screening test, other concerns regarding the widespread use of this study for screening include more limited accessibility, higher cost compared with exercise testing, and the inability to provide the physiologic information that can be obtained from an exercise test.

Risk Assessment in Athletes with Coronary Artery Disease

- Athletes who have had a prior coronary event or who have been diagnosed with CAD by any diagnostic modality should be further risk stratified prior to participation in competitive sports. This can be accomplished by:
  - Assessment of left ventricular function through imaging such as echocardiography.
  - Maximal exercise testing to evaluate exercise capacity and the presence or absence of inducible ischemia (Fig. 11-7).
- The results of these tests allow each athlete to be categorized as being at *mildly increased risk* or *substantially increased risk* for sustaining an adverse cardiac event during exercise (Table 11-3).
- Athletes in the *mildly increased risk* group may participate in low dynamic and low-moderate static competitive sports (classes IA and IIA), although intensely competitive situations should be avoided (Fig. 11-8).
- Athletes in the *substantially increased risk* group should restrict participation to low-intensity competitive sports (class IA).
- Some older athletes with mild atherosclerotic disease, normal left ventricular systolic (LV) function, and no inducible ischemia or arrhythmia may be cleared to participate in more vigorous competitive sports, but approval should be established on a special case-by-case basis and with the clear understanding by both the athlete and the physician that participation in vigorous, competitive sports increases the risk of an acute cardiac event.
- It is advised that older competitive athletes undergo repeat risk stratification at least annually to allow for appropriate revision of sports participation recommendations.
- The development of new signs or symptoms of myocardial ischemia, including an unanticipated decline in athletic performance, should necessitate an immediate cessation of participation in athletic activity and prompt further cardiac evaluation (Fig. 11-9).

EXERCISE AND OSTEOARTHRITIS

- Participation in intense competitive sports throughout life may be associated with an increased risk of developing osteoarthritis (OA).
  - The nature of the sport is closely related to the degree of risk.
  - The greatest risk is associated with sports that involve repetitive, high-intensity, high-impact forces through the joint.
- Joint trauma, whether sports-related or non-sports-related, is a major risk factor for developing OA.
- Other risk factors for OA include:
  - High bone mass
  - Genetic predisposition
  - Increased body mass index
  - Presence of bony deformities
  - Joint instability
  - Muscle weakness
  - Reduced joint proprioception
  - Participation in occupations requiring strenuous physical activity
- Before participating in sports, individuals with osteoarthritis should undergo a careful examination to evaluate joint alignment and integrity, as well the strength and flexibility of the surrounding musculature.
- Proper activity selection and incorporation of specific exercises addressing joint range of motion, flexibility, and muscle strengthening helps reduce the risk of physical activity–related joint injury and improve pain and disability related to osteoarthritis.

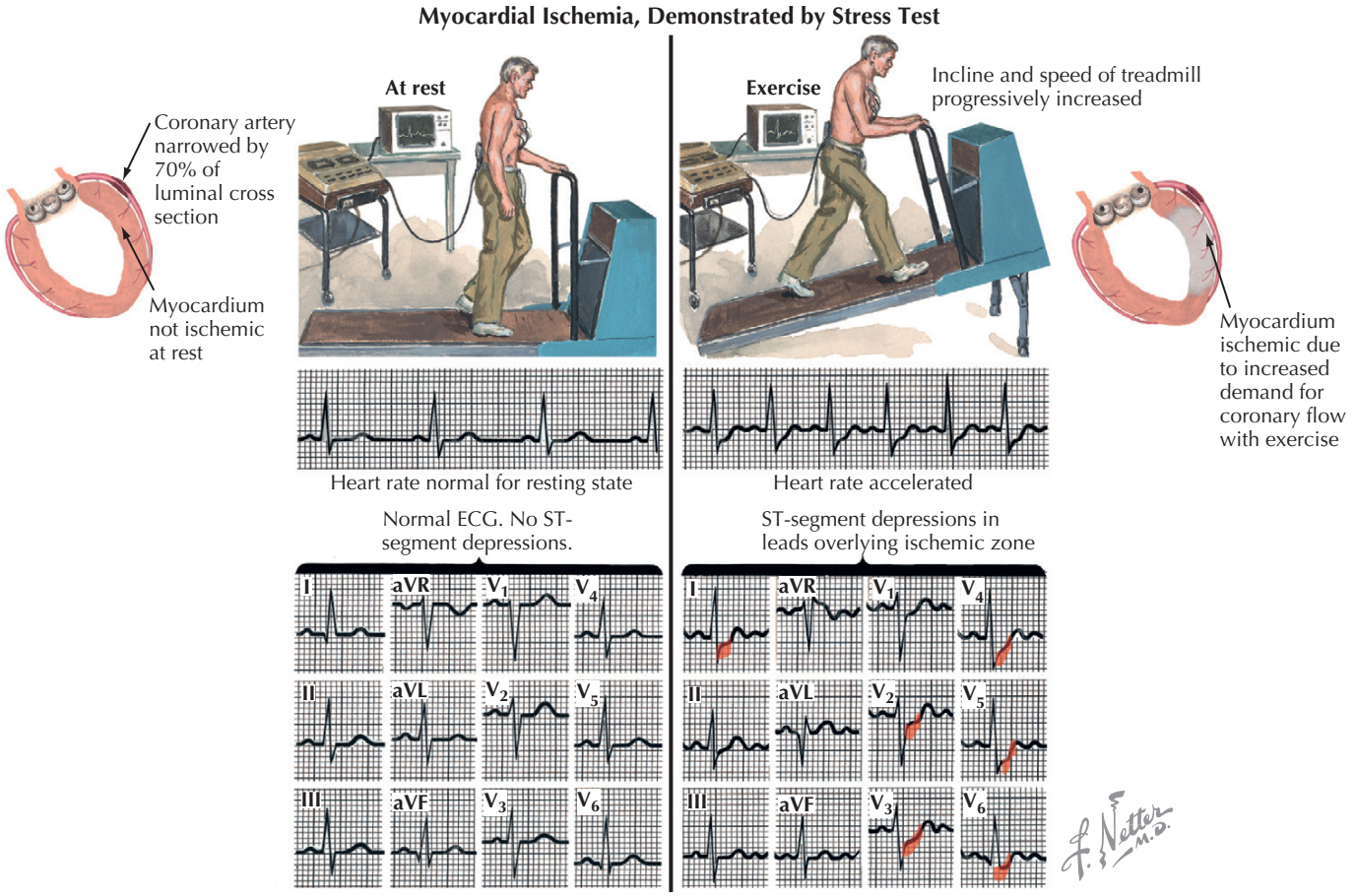

Figure 11-7 Testing to Detect Myocardial Ischemia.

Table 11-3 RISK CATEGORIZATION OF ATHLETES WITH CORONARY ARTERY DISEASE

Test result	Mildly increased risk	Substantially increased risk
Left ventricular systolic function	Normal. EF >50%	Reduced. EF <50%
Exercise tolerance for age*	Normal	Reduced
Normal values: <50 yrs >10 METS 50-59 yrs >9 METS 60-69 yrs >8 METS >70 yrs >7 METS		
Exercise-induced ischemia	Absent	Present
Significant coronary stenosis	Absent	Present
Myocardial revascularization	Successful	Incomplete

*One MET unit implies the consumption of 3.5 mL of $o_2 \cdot kg^{-1} \cdot min^{-1}$.
EF, ejection fraction; METS, metabolic equivalents.
Modified from Whiteson JH, Bartels MN, Kim H, et al: Coronary artery disease in masters-level athletes. Arch Phys Med Rehabil 87:S79-S81, 2006.

- Multiple studies have demonstrated improvements in strength, pain, function, and quality of life with muscle strengthening exercise programs for those individuals who already have OA.
- Aerobic exercises, such as walking and aquatic exercises, have also been shown to help reduce pain and improve function.
- Randomized, controlled trials have demonstrated that regular moderate-intensity exercise, such as walking, aquatic exercise, and resistance training, does not exacerbate OA or accelerate the progression of this disorder.
- Participation in high-intensity, direct joint impact sports, can accelerate the progression of OA.
- Any exercise involving an acutely inflamed joint may be harmful, particularly if the activity involves weight-bearing or significant impact forces.

ATHLETIC ACTIVITY AFTER ARTHROPLASTY

- As the population of older adults has grown in size and the incidence and prevalence of symptomatic arthritis has increased, the number of total joint replacement implants performed in the United States has steadily increased. More than 500,000 hip and knee replacements are performed annually in the United States.
- Health care providers will have increasing opportunities to counsel patients about participation in athletic activity after joint replacement.
- The main goals of joint arthroplasty have traditionally been pain relief and restoration of basic function. However, many patients desire to engage in very high levels of physical activity, including competitive sports.
- The quality of the surgical procedure itself is critical to a successful outcome, including participation in athletics postoperatively. Important surgical factors include:
  - Performing an anatomically and biomechanically accurate joint reconstruction.

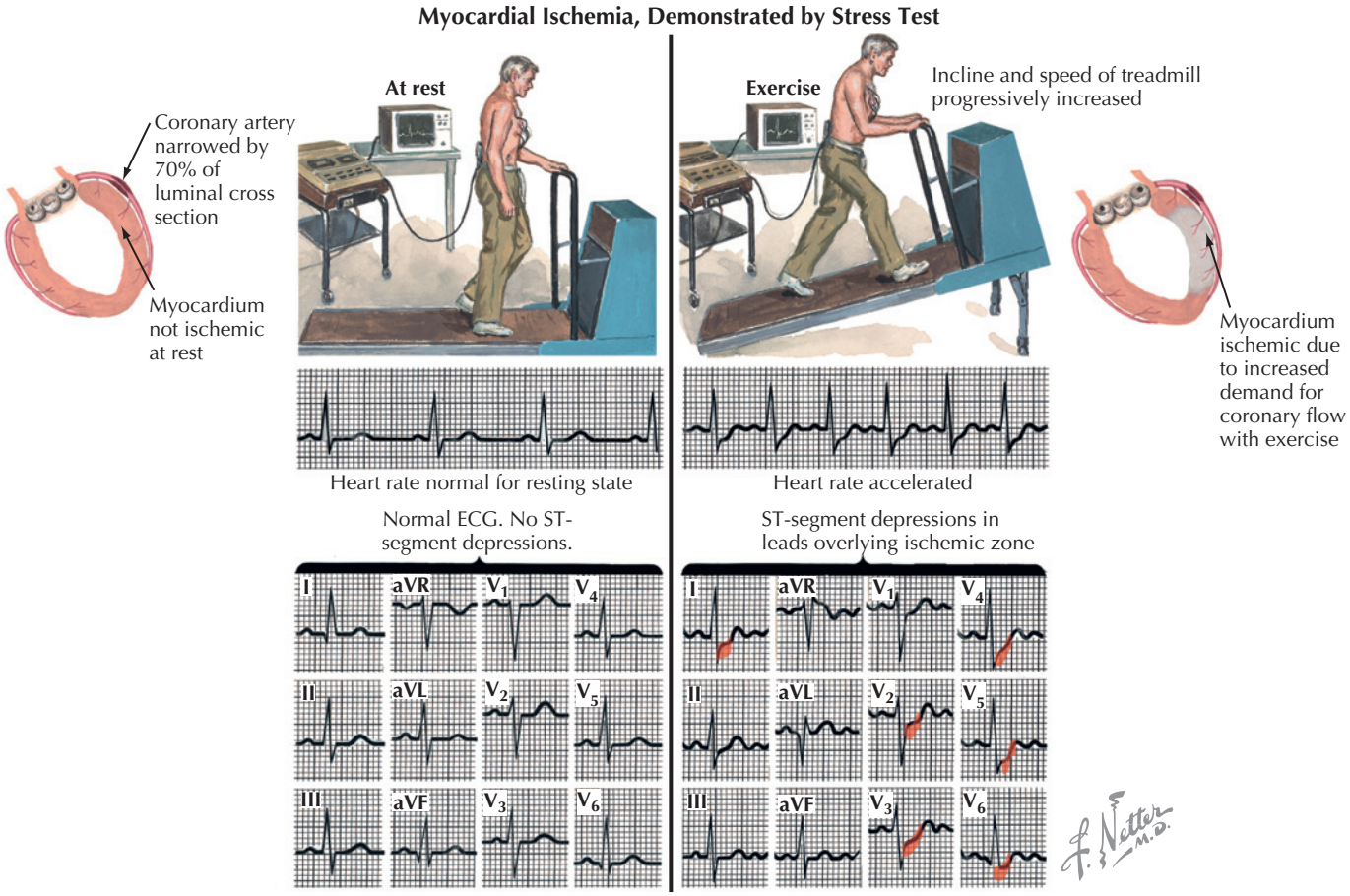

Figure 11-7 Testing to Detect Myocardial Ischemia.

Table 11-3 RISK CATEGORIZATION OF ATHLETES WITH CORONARY ARTERY DISEASE

Test result	Mildly increased risk	Substantially increased risk
Left ventricular systolic function	Normal. EF >50%	Reduced. EF <50%
Exercise tolerance for age*	Normal	Reduced
Normal values: <50 yrs >10 METS 50-59 yrs >9 METS 60-69 yrs >8 METS >70 yrs >7 METS		
Exercise-induced ischemia	Absent	Present
Significant coronary stenosis	Absent	Present
Myocardial revascularization	Successful	Incomplete

*One MET unit implies the consumption of 3.5 mL of $o_2 \cdot kg^{-1} \cdot min^{-1}$.
EF, ejection fraction; METS, metabolic equivalents.
Modified from Whiteson JH, Bartels MN, Kim H, et al: Coronary artery disease in masters-level athletes. Arch Phys Med Rehabil 87:S79-S81, 2006.

- Multiple studies have demonstrated improvements in strength, pain, function, and quality of life with muscle strengthening exercise programs for those individuals who already have OA.
- Aerobic exercises, such as walking and aquatic exercises, have also been shown to help reduce pain and improve function.
- Randomized, controlled trials have demonstrated that regular moderate-intensity exercise, such as walking, aquatic exercise, and resistance training, does not exacerbate OA or accelerate the progression of this disorder.
- Participation in high-intensity, direct joint impact sports, can accelerate the progression of OA.
- Any exercise involving an acutely inflamed joint may be harmful, particularly if the activity involves weight-bearing or significant impact forces.

ATHLETIC ACTIVITY AFTER ARTHROPLASTY

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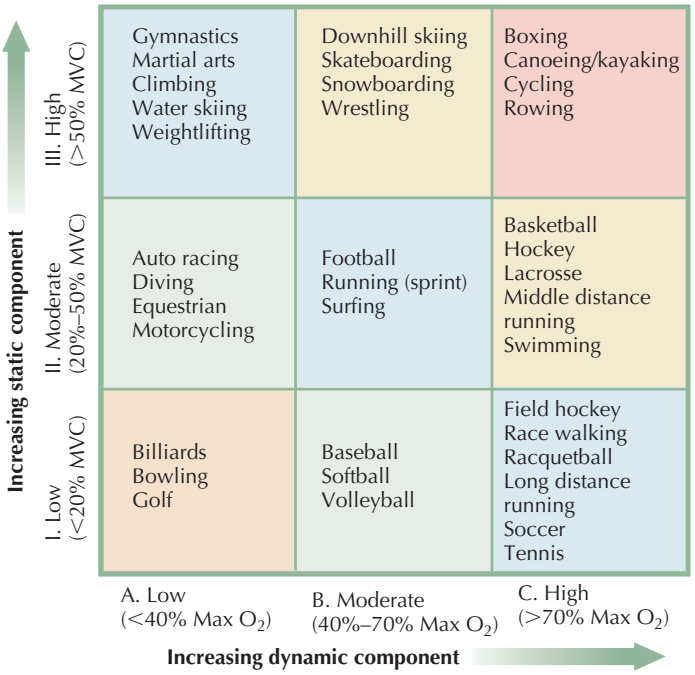
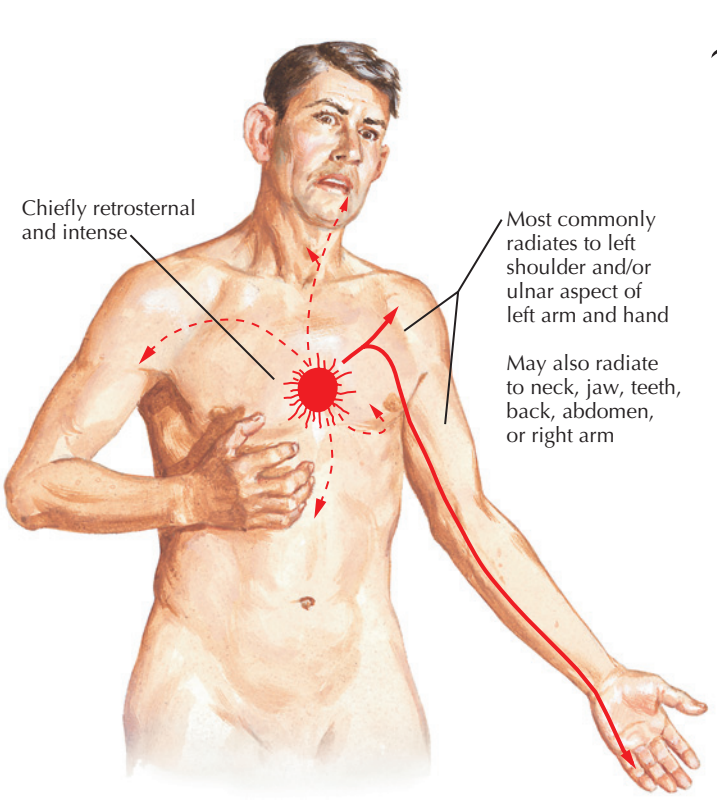


Figure 11-8 Classification of sports. This classification is based on peak static and dynamic components achieved during competitions. Higher values may be achieved during training. The increasing dynamic component is defined in terms of the estimated percent maximal oxygen uptake ($\dot{V}O_2$Max) achieved and results in an increasing cardiac output. The increasing static component is related to the estimated percent of maximal voluntary contraction (MVC) reached and results in an increasing blood pressure load. The lowest total cardiovascular demands (cardiac output and blood pressure) are shown in *green* and the highest in *red*. *Blue*, *yellow*, and *orange* depict the low-moderate, moderate, and high-moderate total cardiovascular demands. *(From Whiteson JH, Bartels MN, Kim H, et al: Coronary artery disease in masters-level athletes. Arch Phys Med Rehabil 87:S79-S81, 2006.)*



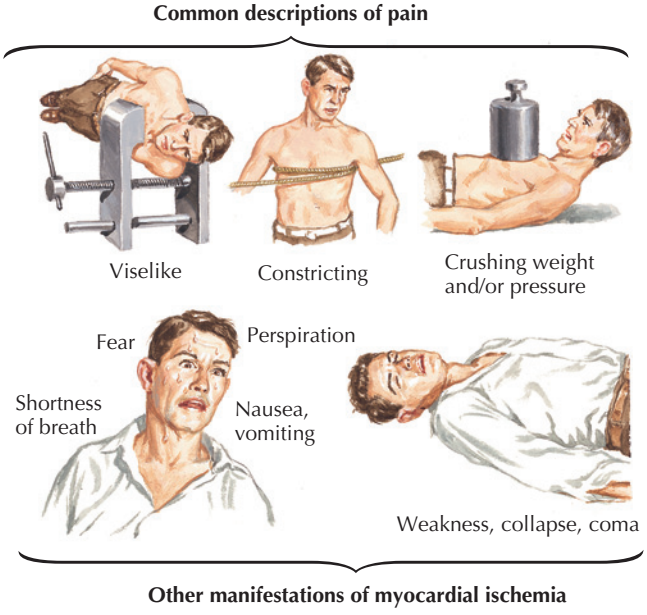


Figure 11-9 Pain of Myocardial Ischemia.

- Using a well-designed implant.
- Creating a properly balanced soft-tissue envelope.

- Other issues to consider when advising patients about participation in sports after joint replacement include:
  - Experience of the participant
  - Preoperative athletic activity is a critical issue to consider when recommending athletic activity after arthroplasty.
  - Those who have achieved proficiency in their sport preoperatively have the greatest likelihood of safely resuming this activity after joint replacement.

  - Attaining high skill levels in a sport after arthroplasty can be quite challenging for patients who have not participated in this activity preoperatively.
  - Participation in a new athletic activity after joint replacement may result in an increased risk of injury.
- The type of athletic activity and the extent of participation
  - Repetitive motion and joint loading can accelerate joint-bearing surface wear.
  - Higher joint loads stress the implant fixation surface, increasing the risk of aseptic loosening.
  - Contact sports and sports with a higher risk of falls increase the likelihood of catastrophic failure, dislocation, and fracture of bone or implants.
- The type of joint being replaced
  - Activities that exhibit high joint loads in knee flexion (e.g., jogging or mountain hiking) will place greater stress on a total knee prosthesis than on a total hip prosthesis.
  - Joint dislocation is more common in the setting of total hip arthroplasty compared with total knee arthroplasty.
- The long-term outcome of joint replacement surgery and return to competitive sports participation has not been well studied and few validated guidelines exist for a return to sports after arthroplasty.
- Most recommendations regarding athletic activity after joint replacement are based on expert opinion and conservatively favor low-impact activities such as walking, cycling, and swimming.
- Contact sports, sports with a high-risk for falls, and high-impact activities are typically discouraged.
- In 1999, members of the Knee Society, the Hip Society, and the American Shoulder and Elbow Society were surveyed regarding their recommendations for athletics and sports participation for their patients who had undergone joint replacement. The responses were statistically analyzed to determine a consensus recommendation for each activity (Tables 11-4 to 11-6).
- Health care providers should be able to counsel patients about the risks and benefits of sports participation in the setting of total joint replacement, but ultimately patients must decide whether or not to participate in certain athletic activities.

TREATMENT OF MUSCULOSKELETAL INJURIES IN OLDER ATHLETES

Initial Management

- Initiation of appropriate treatment for sports-related injuries is often delayed in older athletes because they may wait longer than younger athletes to seek medical advice and because health care professionals may adapt a negative attitude toward older competitive athletes and attribute many injuries to overloading of an aging body.

Table 11-4 ACTIVITY AFTER TOTAL HIP ARTHROPLASTY—1999 HIP SOCIETY SURVEY

Rights were not granted to include this table in electronic media. Please refer to the printed publication.

Table 11-5 ACTIVITY AFTER TOTAL KNEE ARTHROPLASTY—1999 KNEE SOCIETY SURVEY

Rights were not granted to include this table in electronic media. Please refer to the printed publication.

Table 11-6 ACTIVITY AFTER TOTAL SHOULDER ARTHROPLASTY—1999 AMERICAN SHOULDER AND ELBOW SOCIETY SURVEY

Rights were not granted to include this table in electronic media. Please refer to the printed publication.

- Delay in appropriate evaluation may contribute to injury chronicity, complicated rehabilitation, and delayed recovery.
- Initial management of acute musculoskeletal injuries is based on the **PRICE** regimen: **p**rotection, **r**est, **i**ce, **c**ompression, and **e**levation.

Subsequent Treatment Principles

- Some reports suggest that healing of musculoskeletal injuries is slower in older athletes, but results from other studies are inconsistent.
- Physiotherapy, addressing issues such as range of motion, flexibility, strength, and proprioception, plays an important role in treatment process of many injuries.
- Older athletes have the capacity to respond to an active and progressive rehabilitation regimen.
- Principles of relative rest and activity modification should be followed.
- The vast majority of injuries in senior athletes respond to conservative measures.
- Fewer than 5% of musculoskeletal injuries require surgical intervention.
- Because many older adults are retired from the work force, they may have more time to devote to rehabilitation and thus hasten recovery.
- Total inactivity should be avoided during recovery period to minimize potential complications, such as range-of-motion loss, accelerated loss of bone mineral density, and cardiovascular deconditioning.
- Alternative training methods should be recommended.

Pharmacotherapy

- Nonsteroidal anti-inflammatory drugs (NSAIDs) are frequently prescribed for musculoskeletal injuries.
- Potential adverse effects associated with NSAID use include:
  - Gastrointestinal toxicity, including dyspepsia, peptic ulcer disease, and bleeding.
    - Advanced age (>60 years) is associated with increased risk of gastrointestinal (GI) toxicity.
    - Other factors that increase risk of NSAID-related GI toxicity include prolonged use (>1 week), high dosage (>2 times normal), prior GI event, and concurrent use of glucocorticoids or anticoagulants.
    - Selective **cyclooxygenase-2 (COX-2) inhibitors** (e.g., celecoxib) have analgesic and anti-inflammatory properties comparable to nonselective agents but are associated with less GI toxicity (e.g., fewer symptomatic gastroduodenal ulcers, less upper GI bleeding). For athletes at higher risk for NSAID-induced GI toxicity, a COX-2 inhibitor may be a reasonable option.
    - If nonselective NSAIDs are used, effective prophylactic therapy to reduce risk of GI toxicity can be achieved with concurrent use of misoprostol, H_2-blockers, or proton pump inhibitors.
  - Acute renal failure resulting from renal vasoconstriction or interstitial nephritis. NSAIDs are also associated with the development of membranous nephropathy and papillary necrosis.
    - Selective COX-2 inhibitors are associated with the same renal complications as the nonselective NSAIDs.
  - A variety of cardiovascular effects including:
    - Modest worsening of underlying hypertension.
    - Exacerbation of preexisting heart failure.
    - Potential increased risk of ischemic cardiovascular events (risk varies based on specific agent, dose, and duration of use).

MUSCULOSKELETAL INJURY PREVENTION IN OLDER ATHLETES

- Prevention of musculoskeletal injury is a main goal of the PPE and individual exercise prescription.
- Evidence-based recommendations are difficult because few controlled studies have included older adults. It is assumed that prevention guidelines for younger athletes also may be beneficial for older athletes.
- Proper warm-up before activity and adequate cool-down after activity are important.
- Abrupt changes in frequency, duration, or intensity of activity should be avoided.
- Days of intense physical activity should be alternated with less strenuous days to allow adequate recovery time.
- Surface conditions deserve close attention.
  - Soft surfaces reduce impact forces on lower limbs.
  - Uneven surfaces should be avoided in athletes with balance problems.
- Important environmental factors include temperature, humidity, and lighting.
- Activities that may exacerbate an underlying condition should be avoided; for example, athletes with osteoarthritis of the lower limb should restrict high-impact activities.
- Adequate activity-specific strength and neurophysiologic capacities must be ensured before an older adult engages in a particular athletic activity.

- Adequate nutrition must be maintained (see "Nutrition in Older Athletes").

NUTRITION IN OLDER ATHLETES

General Considerations

- Regardless of age, proper nutrition is essential for optimal health and athletic performance.
- All athletes need adequate energy intake to fuel working muscles during physical activity.
  - The key factor for predicting energy needs in older athletes is training volume.
  - In general, older adults require lower caloric intakes to maintain body weight, primarily because of decreased lean body mass, which is a major determinant of daily energy expenditure.
- With fewer overall calories consumed, nutritional quality of the older athlete's diet becomes increasingly important.
- Several factors may influence an older athlete's nutritional needs and intake.
  - Decreased appetite and food intake related to diminished sensitivity to taste and smell and problems with dentition.
  - Decreased sense of thirst.
  - Decreased efficiency of gastrointestinal tract related to reduced production of gastric acid and other digestive enzymes and delayed gastric emptying.
  - Imposed dietary restrictions to help manage chronic medical conditions, such as low-fat, low-cholesterol diets for cardiovascular disease and low-sodium diets for hypertension.
  - Potential for prescription drugs to affect food digestion, nutrient absorption, and nutrient utilization.

Macronutrients

- In older athletes, carbohydrates remain the primary fuel for working muscles and should be in the range of 45% to 65% of total daily caloric intake.
- Fat requirements do not change with aging. Intake should comprise less than 30% of total daily caloric intake and should focus on monounsaturated and long-chain polyunsaturated fats.
- According to most guidelines, protein intake should be 0.8 gm/kg per day, but some studies suggest that protein requirements increase with aging. Prolonged or high-intensity physical activity also increases protein needs. Overall, older athletes may require 1 to 1.7 gm/kg of protein per day to promote positive nitrogen balance, which is important to help maintain lean body mass.

Micronutrients

- The combined effects of exercise and aging may affect certain micronutrient needs, but little research has been conducted examining the micronutrient needs of older athletes.
- Adults over the age of 50 have increased needs for vitamin B6, vitamin D, and calcium, but there is no evidence to suggest higher requirements in older individuals who participate in sports.
  - Men older than 50 should consume at least 1.7 mg of vitamin B6 daily.
  - Women older than 50 should consume at least 1.5 mg of vitamin B6 daily.
  - Men and women between the ages of 51 and 70 should consume at least 10 μg of vitamin D daily.
  - Men and women older than 70 should consume at least 15 μg of vitamin D daily.
  - Men and women older than 50 should consume 1200 mg of calcium daily.
- Iron is the one micronutrient that is needed in smaller amounts for women after menopause. The recommended dietary allowance (RDA) for iron decreases from 15 mg daily for premenopausal women to 8 mg daily after menopause. The RDA does not change for men as they get older. However, it is estimated that athletes, particularly those who engage in regular endurance exercise, require 30% more iron than those individuals who do not exercise, owing to greater loss of iron.
- Micronutrients with antioxidant properties, such as vitamins A, C, and E, may help reduce exercise-related tissue damage and promote repair, but this issue remains unresolved. Deficiency of these micronutrients is rare, particularly if older athletes emphasize fresh fruits and vegetables in their diets.

Fluids

- Adequate fluid intake and hydration are essential for optimal health and athletic performance, but they may be more challenging for older athletes.
- Older adults may be at an increased risk for developing fluid and electrolyte imbalances during and after vigorous physical exertion.
- Age-related blunting of thirst sensitivity and slower renal responses to water and sodium loads can increase risk for dehydration and electrolyte disturbances such as hyponatremia.
- Strategies recommended for younger athletes can be used to help establish hydration guidelines for older individuals, including:
  - Do not rely on thirst to decide when to drink.
  - Consume fluids on a regular schedule during activity.
  - Routine measurement of pre- and postexercise body weights can be used to determine sweat rates and individualized fluid replacement programs.
  - Consuming beverages and foods with sodium after exercise will help stimulate thirst and fluid retention.
  - Fully replace any fluid or electrolyte deficits before the next bout of exercise.

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The Physically Challenged Athlete

12

Katherine L. Dec

GENERAL CONSIDERATIONS

Definitions

Physically challenged: combines all groups of athletes competing in international competition such as Paralympics. Athletes have an impairment that restricts or decreases their ability to participate in athletic arenas within manner considered "normal" for defined sport.

Impairment: any loss or abnormality of psychological, physical, or anatomical structure or function.

Disability: any restriction imposed from an impairment that limits an individual's ability to perform an activity within manner considered normal for an able-bodied individual.

Handicap (as defined by World Health Organization): a disadvantage, resulting from impairment or disability, that interferes with a person's efforts to fulfill a role that is normal for that person. Handicap is a *social concept*, representing social and environmental consequences of a person's impairments or disabilities.

Statistics

- There are more than 8 million disabled people in the United States; many are nonambulatory.
- "Disabled" classification is broad and includes also the nonathletic population.
- There are more than 200,000 people in the United States with a spinal cord injury (SCI).
  - Includes traumatic and nontraumatic
  - 11,000 new injuries per year; average age at time of injury is 32
  - 55% quadriplegia, 45% paraplegia
- There are more than 1,285,000 people in the United States with limb loss.
  - Incidence of congenital limb deficiency is 25.64 per 100,000 live births.
  - People older than 65 account for 19.4 per 1000 of those with limb loss.
  - Comorbidity: diabetes, vascular, malignancy.
  - Incidence rate:
    - Lower extremity amputation (LEA), diabetes, and younger than 30: 7.2%
    - LEA, diabetes, and older than 30: 9.9%
    - Dysvascular disease: 46.2 per 100,000 with limb loss
    - Trauma: more than 5.86 per 100,000 with limb loss (war increases rate)
    - Malignancy: 0.35 per 100,000 with limb loss
- Multiple sclerosis: 400,000 cases are diagnosed in the United States each year.
- Muscular dystrophies: estimates put the number of new cases at 250,000 each year in the United States. Duchenne muscular dystrophy is 1 of 9 types of muscular dystrophy.

History

- First sports event for the physically challenged: 1888, Sport Club for the Deaf in Berlin, Germany.
- First international competition for disabled: International Silent Games in 1924.
- First international sports competition for people with various physical impairments: Stoke Mandeville Games for the Paralyzed, 1948.
- Youth divisions, in addition to adult competitions, for athletes with physical impairment since 1980s.
- Also, adaptive physical education, hippotherapy, and aquatic therapy have been offered since the 1980s.

Competition

- Interscholastic, collegiate, professional sports: physical impairment cannot require rule change of sport, lowering of standards for achievement, or modification of defined sport to accommodate athlete.
- Neither adaptive equipment nor physical impairment can impart danger or advantage to athlete or others competing.
- Wrestling
  - Those with hearing loss have competed successfully with normal hearing athletes. If hearing loss is greater than 55 decibels in the better ear, a wrestler can qualify for physically challenged competition.
  - Limb loss: Wrestlers must weigh in with prosthesis, if used.
- Jim Abbott, professional major league baseball player. Congenital absent right hand
- Archery
- Below knee amputations (BKA)
  - Allowed in high school football after restriction removed in 1978. Check local competition rules.
  - National Federation of State High School Associations rules concerning contact sports:
    - Restricted to BKA. No upper extremity or above knee prosthesis.
    - Metal hinges restricted to lateral and medial; require covering.
    - No metal in front of knee unless properly padded.
    - Prosthesis must be wrapped with minimum of ½-inch foam rubber or appropriate polyurethane.
    - Approval of physician associated with amputee care recommended.
- Paralympics
  - An international competition that follows the Olympics.
  - Traditionally includes athletes with limb deficiency, cerebral palsy, visual impairment, spinal cord injuries, les autres (those not fitting into other groups), intellectual disability.
  - Currently, intellectual disability is not a participant group in International Paralympic Committee (IPC) sanctioned events. The IPC has asked the International Sports Federation for Persons with an Intellectual Disability (INAS-FID) to develop eligibility and verification processes that are commensurate with other IPC divisions to ensure fair competition among these athletes.

Classification Systems

- System used to equalize athletes in sports competition using objective methods:
  - **Medical diagnosis only:** cerebral palsy, limb deficiency, muscular dystrophy, etc.
  - **Functional measurement only:** wheelchair mobility level, above knee or below knee amputation, etc.
  - **Hybrid:** use functional measurements and medical diagnosis; multiple sclerosis with full trunk control and wheelchair mobility.
- May be sport-specific for alpine skiing, cycling, etc.
- Classification systems may be different at international versus local competitions.
- At the 1996 Atlanta Paralympics, of 3500 athletes, the most common impairment was limb deficiency.

GENERAL CONSIDERATIONS FOR TREATMENT OF ATHLETE

- Cognitive age differences: coping with impairment
  - Adult: potential concurrent medical issues, social isolation
    - Management of comorbid diabetes, arthritis, other disease
  - Youth: peer interaction, relationships
    - Missed social/peer opportunities
    - Constant change in size/fit of adaptive equipment
    - Health insurance: have benefit limits, Medicaid limits
- Counsel: assist athlete in redesigning athletic or career goals
- Financial needs: insurance coverage, private funds, home equity loans
  - Paperwork, appeal process, funding for equipment needs
- Physical office facilities: Americans with Disabilities Act (ADA) criteria for accessibility
  - Adjustable height exam table
  - Appointment scheduling adjustment to allow time to address unique mobility, equipment, or comorbid issues
- Establish virtual office with other health professionals to integrate care for athlete
  - Potential team members: neurosurgeon, physiatrist, therapists, vocational rehab, counselor, primary physician, prosthetist, orthotist

ORGANIZATIONS

- Several U.S. and international organizations address the needs of physically challenged athletes (Box 12-1). One example is Disabled Sports USA (DS/USA):
  - Founded in 1967 by disabled Vietnam veterans
  - Provides opportunities for those with disabilities to gain confidence and dignity through sports, recreation, and educational programs
  - Nation's largest multisport, multidisability organization, serving more than 60,000 people
  - Member of U.S. Olympic Committee
  - Sponsors the Wounded Warrior Project

BOX 12-1 *Resource Organizations*

USA
National Disability Sports Alliance (NDSA)
www.ndsaonline.org
United States Les Autres Sports Association (USLASA)
Dwarf Athletic Association of America (DAAA)
www.daaa.org
Amputee Coalition of America (ACA)
www.amputee-coalition.org
National Center on Physical Activity and Disability (NCPAD)
www.ncpad.org
Disabled Sports USA (DS/USA)
www.dsusa.org
Adaptive Sports Foundation
www.adaptivesportsfoundation.org
BlazeSports
www.blazesports.com
INTERNATIONAL
International Paralympic Committee (IPC)
www.paralympic.org
International Wheelchair and Amputee Sports Federation (IWAS) www.iwasf.com

SPINAL CORD INJURY

Physiology Changes in Exercise

- Altered venous return, thus decreased ability to respond to exercise stress
- Depending on level of spinal cord injury level, possible blunting of heart rate response to exercise
- Vagal withdrawal, not sympathetic drive
  - Decreases the reflexive regulation of blood flow
  - Decreased total peripheral resistance (increased vasodilation)
  - Increased peripheral pooling
    - Treatment to minimize: compression garments, abdominal binder
  - Decreased oxygenated blood to exercising muscle
    - Fatigue, limited aerobic endurance
- Cardiac repolarization abnormalities
- Decreased lactate threshold
- Limited pulmonary capacity
- Kinetic chain disruption
  - Loss of ground reactive force from lower extremity
  - Stabilizing muscles become prime movers
- Greater muscular strength improves aerobic power and endurance
- Paraplegics and people with high-level SCI can increase VO_2 max with exercise
  - Dependent on intensity, frequency, and duration

Medical Concerns in Athlete with SCI

History

- SCI level: complete or incomplete, type of injury (Figs. 12-1 and 12-2)
- Surgeries related to injury: past spinal fusion, surgical muscle transfers for functional improvement, surgically implanted medical devices

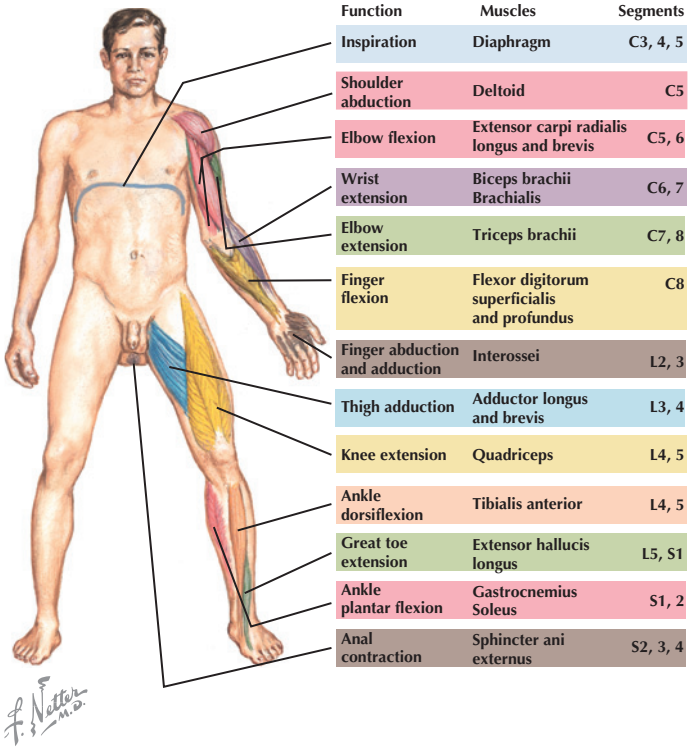


Figure 12-1 Motor Impairment Related to Level of SCI.

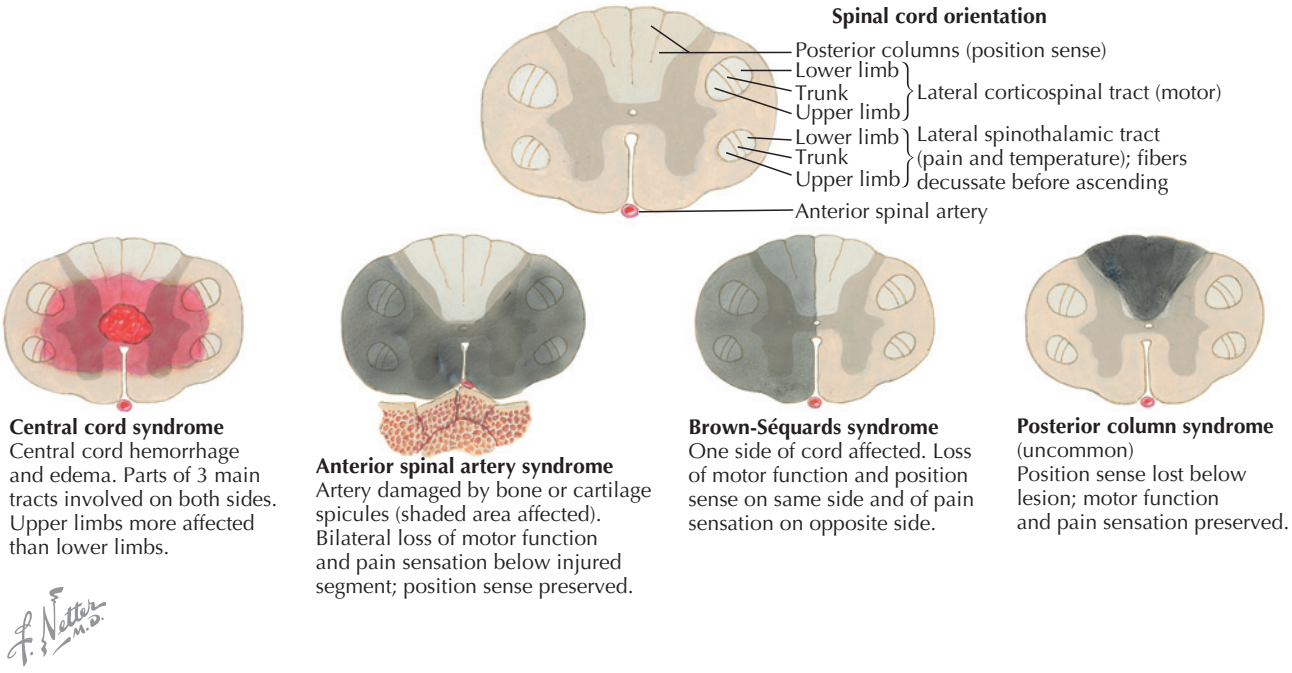


Figure 12-2 Incomplete Spinal Cord Injuries.

- Medications: antiepileptics, antispasmodics, tricyclic antidepressants, anticholinergics, baclofen pumps, pain medicines, other medicines for comorbid conditions
- Comorbid medical issues:
  - Related to impairment: e.g., pressure sore, type and success of bowel/bladder management program; recurring urinary tract infection (UTI)
  - Related to concurrent illness: e.g., traumatic brain injury (TBI), diabetes, visual impairment, amputation, cardiac disease, seizures
- Level of functional independence: independent transfers with wheelchair, self-management of personal hygiene
- Adaptive equipment needs: sports specific or for general mobility
- Prior training: environmental conditions, aerobic and anaerobic conditioning, flexibility

Physical Conditions to Consider in SCI

Deep Venous Thrombosis (DVT)

- Risk greatest within first 2 weeks after injury
- Other risk factors: obesity, trauma to pelvis and lower extremities, congestive heart failure, history of malignancy, tight garments below level of lesion, and previous occurrence of thromboembolism
- Venous pooling occurs in lower limbs
- Prevention: passive stretching of limbs, abdominal binder, functional electrical stimulation (the latter needs further research)

Heterotopic Ossification

- Etiology unclear
- Symptoms: pain, increased warmth, swelling, decreased joint motion or contracture
- Incidence rate: 16% to 53%
- Most common location: hip, followed by knee, shoulder, and elbow
- Prevention: initial treatment after onset of SCI with nonsteroidal anti-inflammatory drugs (NSAIDs)
- Risk decreases two to three times with proper treatment
- Treatment:
  - Stretching, passive range-of-motion exercises.
  - Medication: NSAID, etidronate disodium, and other bisphosphonates.
    - Etidronate disodium: blocks aggregation, growth, and mineralization of calcium hydroxyapatite crystals. Heterotopic ossification can occur in previously etidronate-treated populations.
  - Surgical excision: high reoccurrence rate; if wait until mature, chance of recurrence is less.
- Imaging: three-phase bone scan. X-rays often negative in initial symptom presentation and may take 4 to 5 weeks for findings
- Laboratory findings: significant elevation in fractionated alkaline phosphatase during bone ossification

Autonomic Dysreflexia (AD)

- Medical emergency in T-6 and above SCI level
- "T-6" refers to sixth thoracic level motor and sensory nerve impairment of SCI
- No supraspinal neurological inhibition is present; sympathetic nervous system is left unchecked
- Symptoms: elevated blood pressure, headache, piloerection, profuse sweating, nasal congestion, and/or bradyarrhythmia
- Elevated blood pressure: an increase of 20 to 40 mm Hg in adults, or 15 mm Hg in adolescents; may be the *only* symptom
- Cause: noxious stimulus to spinal cord below level of SCI, such as pressure sores, UTI, fracture, tight clothes, distended bowel or bladder, heterotopic ossification
- Treatment: remove offending stimulus (Box 12-2)
- Performance enhancement: boosting
  - Increases cardiovascular demand. A small study demonstrated that an increased release of catecholamines does occur during exercise; higher peak performances, heart rate, O_2 consumption, and blood pressure noted.

Pulmonary Complications

Atelectasis, pneumonia, and mucous plugging are the most common causes of morbidity in high-thoracic SCI and quadriplegia.

BOX 12-2 *Treatment Steps for Autonomic Dysreflexia (AD)*

Sit person up, lower legs.
Loosen clothing or constrictive devices.
Check blood pressure every few minutes until patient stabilizes.
Remove offending stimulus.
Bladder distension is most common.
Bladder catheterization: 2% topical lidocaine jelly helpful.
Nifedipine 10 mg immediate release, bite and swallow.
Repeat medicine in 15 minutes if no improvement.
Continue to monitor blood pressure and symptoms for 2 hours after episode.
If offending stimulus still present, recurrence of AD possible.

Urinary Tract Infection

- Potential comorbid issue because of bladder management options
- Long-term use of indwelling catheters leads to higher risk for UTI
- Symptoms can be masked because of lack of feeling below SCI level: patient won't feel typical urinary urgency, dysuria, or flank pain
- Initial symptom usually increased spasticity, feeling sick, sweating, or autonomic dysreflexia (AD)
- Treatment: rigorous personal hygiene for prevention is paramount
- High frequency of resistant organisms: obtain urinalysis with Gram stain, urine culture, and sensitivity when possible *before* initiation of antibiotic therapy
- Abnormal bacterial count but no symptoms usually managed without antibiotics

Thermoregulation

- Sweating often impaired below the level of SCI
- Less surface area (e.g., arms and upper trunk) for evaporative cooling
- Check skin under arms of a distressed athlete during competition; if it is hot, athlete is likely not dissipating heat adequately
- Treatment: lighter clothing, more fluids, or dousing the skin with water are necessary

Hypothermia

- Contributing factors: decreased muscle mass below level of lesion, loss of vasomotor and sudomotor neural control, and possible decreased input to the hypothalamic thermoregulatory centers
- Impaired or absent sensation intensifies risk: unaware of clothing dampness, which augments heat loss

Pressure Sores

- Improperly fitting prosthetics or, in wheelchair athletes, poor seat position/posture
- Risks: racing/track wheelchairs put knees higher than hips and increase pressure over sacrum and ischium
- Treatment:
  - Proper positioning in wheelchair, attaining correct fit of prosthetics/orthotics
  - Performing regular pressure reliefs
  - Lifting self off seat for 10 to 20 seconds throughout day
  - Appropriate cushioning, seat system
  - Reducing skin moisture (wearing absorbent fabric)
  - Minimizing potential for skin shear
  - Wound management immediately at first signs of pressure sore
    - Topical skin products before dermis is breached; debridement

Neurogenic Bladder

- Disrupted regulatory control of bladder contraction and voiding
  - Areflexic (decreased tone/increased capacity, with urinary retention)
  - Reflexic (increased tone/decreased capacity, with involuntary voiding)
- Treatment:
  - Medication and/or program to empty bladder
  - Intermittent urinary catheterization: insert catheter into urethra for few seconds to drain urine into disposable bag; typically performed on strict schedule
  - Continuous (indwelling) urinary catheter: long-standing indwelling catheters contribute to recurrent UTIs; condom catheters option in men
  - Suprapubic catheter: least common in athletic population
  - Other option in complete SCI (minimal research in athletes): sacral anterior root stimulation
  - Medications:
    - Anticholinergics (e.g., oxybutynin, tolterodine, hyoscyamine, imipramine, and propantheline) indicated with detrusor hyperreflexia
    - Alpha-adrenergic antagonists (e.g., terazosin, tamsulosin, prazosin, and phenoxybenzamine) reduce internal sphincter tone
    - Antispasticity drugs (e.g., baclofen, diazepam, and dantrolene) might be of value in those with severe spasticity of perineal muscles
    - Medications used for both neurogenic bladders can have side effects such as impaired sweating, drowsiness, hypotension, and gastrointestinal problems

Neurogenic Bowel

Regulation of normal bowel elimination is impaired.

- Medications can include stool softeners, rectal suppository, enema
- Nutritional plan and regular bowel program are key

Spasticity

- See "Spasticity" section in "Cerebral Palsy" section later in chapter.
- Medications can interfere with sports or function, and may have negative effect on alertness and muscle strength. Medication option more successful in SCI than in brain-mediated injury.

Osteoporosis

- Up to 50% decline total bone content 10 years post-SCI
- Causes: immobilization, significant increase in osteoclastic bone activity combined with only slight increase in osteoblastic activity, parathyroid hormone suppression, reduced absorption of calcium from gastrointestinal tract, possible vitamin D deficiency, and loss of active muscle traction effect
- Treatment: limb range of motion, weight-bearing exercises including standing frame, lower extremity orthoses, treadmill walking with partial body weight–support equipment, functional electrical stimulation, bicycle ergometry

LIMB DEFICIENCY

History

- Surgeries related to injury: type of amputation or limb difference (Figs. 12-3 to 12-5), surgical muscle transfers for functional improvement, surgically implanted medical devices

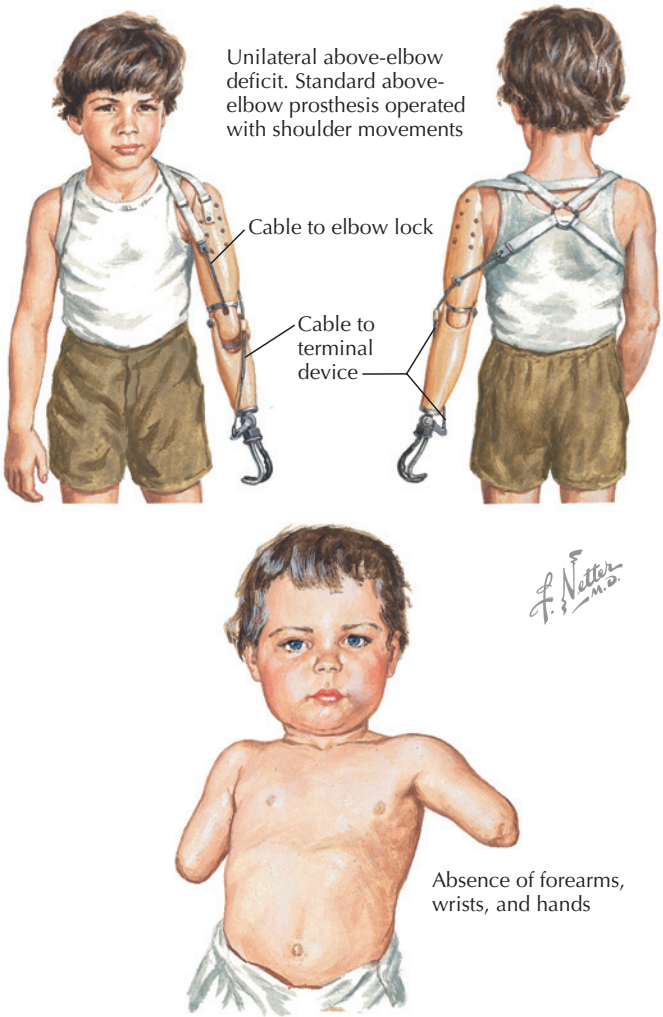


Figure 12-3 Congenital Limb Deficiency: Hemimelia.

- Medications: antispasmodics, tricyclic antidepressants, pain medicines (may affect cognition), other medicines for comorbid conditions
- Comorbid medical issues:
  - Related to impairment: e.g., skin breakdown from prosthesis
  - Related to concurrent illness: e.g., traumatic brain injury, diabetes, visual impairment, low back pain, cardiac disease
- Presence of phantom limb pain or sensation
- Level of functional independence: if multiple limb loss or difference, adaptive equipment needs for mobility
- Independence donning adaptive equipment; prosthesis (see Fig. 12-3)
- Adaptive equipment for sports-specific needs: prosthesis, orthosis, sports equipment
- Prior training: environmental conditions, prior exercise program
- Level of aerobic and anaerobic conditioning, flexibility

Physical Conditions in Athlete

- Skin breakdown: can occur with improper fitting prosthesis, impact type of sport at prosthesis-skin interface
- Treatment: attaining correct fit (see "Pressure Sores")

Phantom Pain

- Phantom limb sensations or pain: occur first few weeks after amputation in approximately 70% of individuals (Fig. 12-6)
- Theory: sudden lack of afferent input and cortical reorganization after amputation
- Medications: antidepressant and anticonvulsant drug classes; opioids such as tramadol. Tricyclic antidepressants continue to be one of initial choices. Acupuncture also found to be effective.

Energy Expenditure

- Energy expenditure with LEA is greater than in the able-bodied:
  - BKA: 16% to 25% more energy expended in ambulation
  - Above knee amputation (AKA): 56% to 65% more energy expended in ambulation

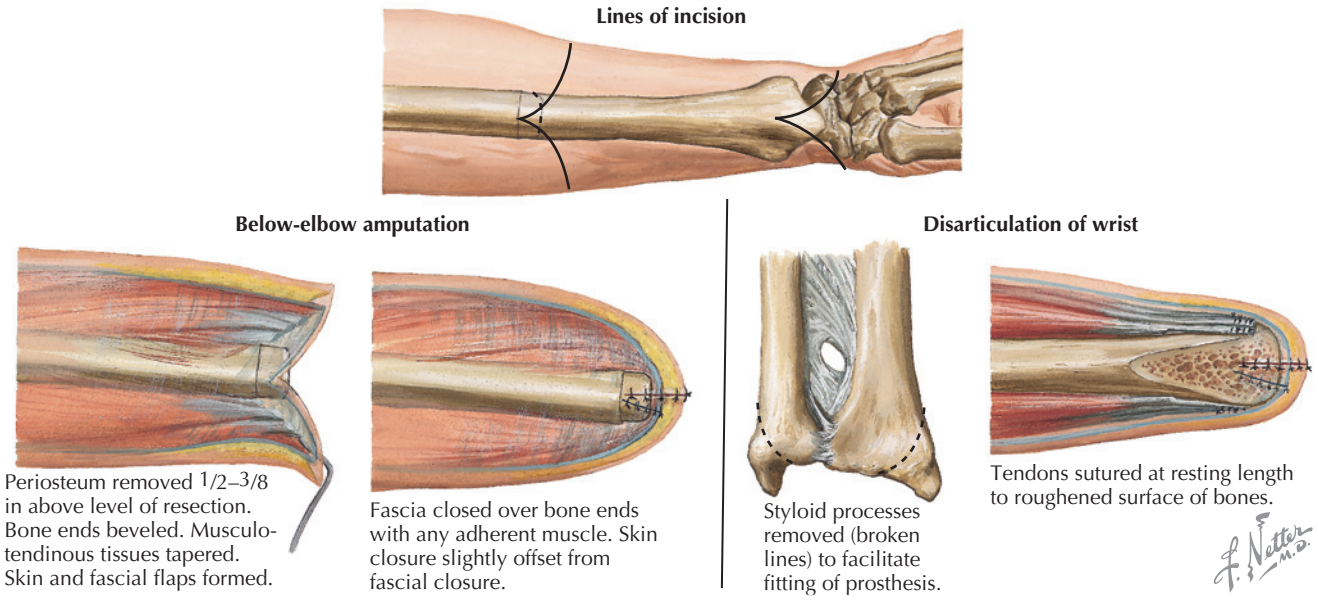


Figure 12-4 Amputation of the Forearm and Hand.

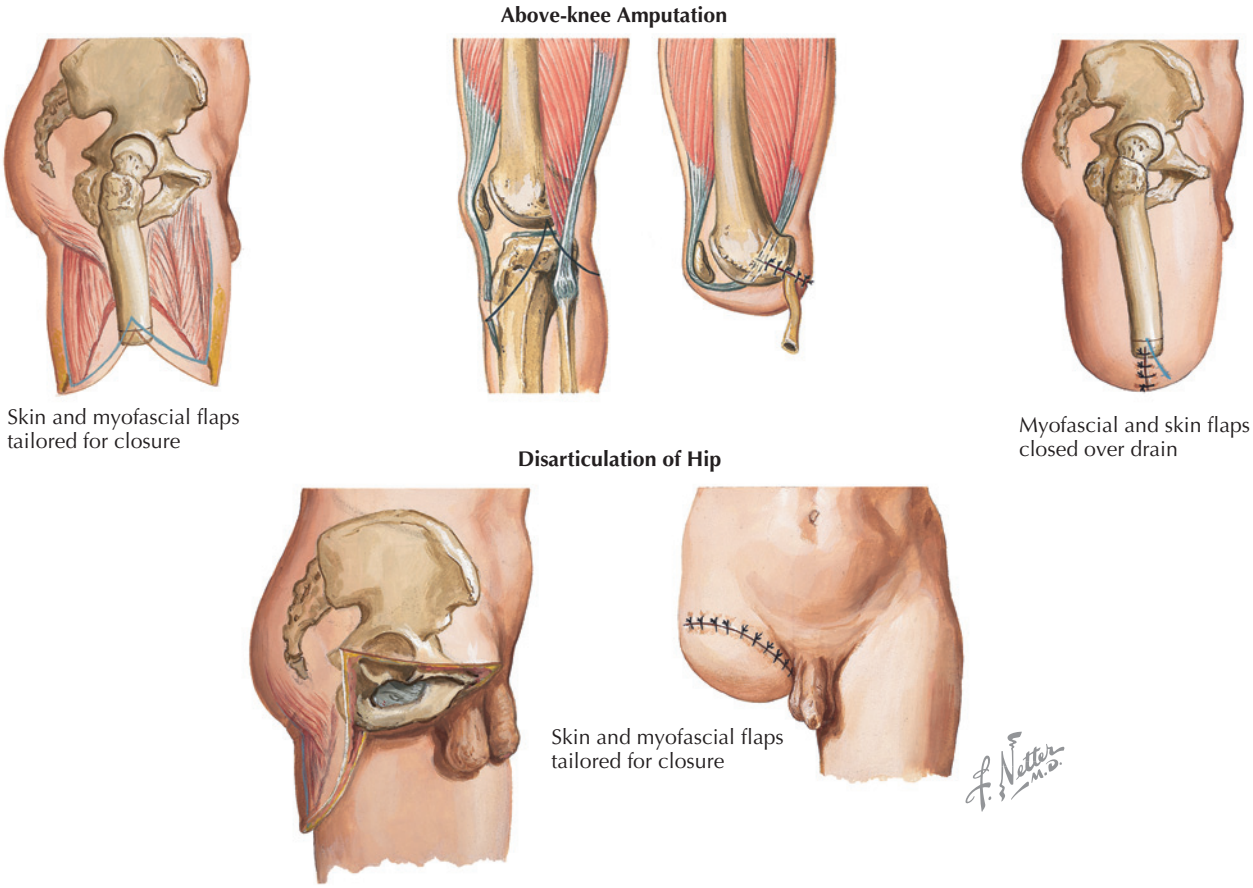


Figure 12-5 Above Knee Amputation.

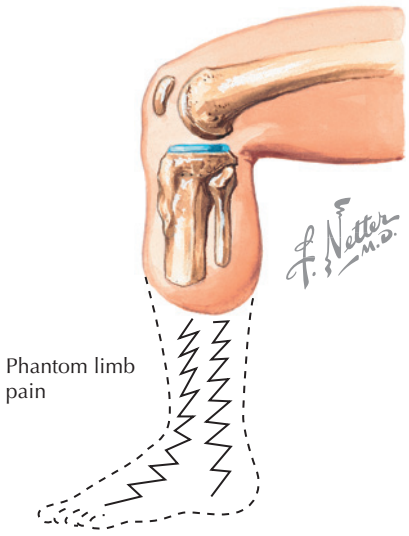


Figure 12-6 Complications of Amputation.

- Require good strength, balance on limb, to better adapt to prosthetic component mismatch
- Quadriceps and hip abductors and adductors important for success with prosthesis

LEA Secondary Issues

- Low back pain: found in greater than 50% of lower extremity amputees
- Knee degenerative arthritis
- Knee pain: three times increased risk in AKA; two times increased risk with intact limb in BKA, five times *reduced* risk in prosthetic limb of BKA

CEREBRAL PALSY (CP)

Neurologic injury may include persistence of primitive reflexes and posture-mediated movement patterns.

History

- Surgeries related to injury: surgical muscle transfers for functional improvement, surgically implanted medical devices, tendon-lengthening procedures
- Medications: antispasmodics, antiepileptics, tricyclic antidepressants, pain medicines, medications for comorbid conditions such as heart disease
- Comorbid medical issues:
  - Related to impairment: spasticity, nutritional support (e.g., gastrostomy tube), seizures
  - Related to concurrent illness: e.g., diabetes, visual impairment, cardiac disease
- Cognitive impairment: such as memory, calculation/organizational aspects
  - Intellectual disability is *atypical for athletes* in international level of competition.
  - Impulsivity or risk taking behavior may be present.
- Adaptive equipment: necessary for mobility, or sports specific
- Level of functional independence: donning adaptive equipment, personal hygiene
- Level of independence in mobility: wheelchair, orthoses
- Prior training: environmental conditions, prior exercise program
- Level of aerobic and anaerobic conditioning, flexibility

Glossary in Cerebral Palsy

Spastic CP: most common form; affects 70% to 80%; increased muscle tone and stiffness. Spasticity increases with excessive fatigue or anxiety.
Athetosis CP: four limbs, trunk, and sometimes face. Athetonia is a slow, writhing involuntary muscle movement. Muscle tone can be mixed: increased or decreased.
Ataxic CP: four limbs and trunk, primarily decreased coordination of movement, also hypotonia. Intention tremor present.
Diplegia: lower limbs involved more than upper limbs.
Hemiplegia: upper and lower limbs on one side more involved.
Triplegia: three limbs, usually both lower extremity limbs and one upper extremity limb involved.
Quadriplegia: upper and lower limbs involved.

Spasticity

- Pathophysiology is unclear. Theory is velocity-dependent increase in tonic stretch reflexes (muscle tone) with exaggerated tendon jerks, resulting from hyperexcitability of stretch reflex. Increased with nociceptive stimulus, such as UTI, distended viscera, bowel obstruction.
- Decision to treat spasticity depends on help (ability to ambulate) or hindrance (impedes independence with mobility and self-care).

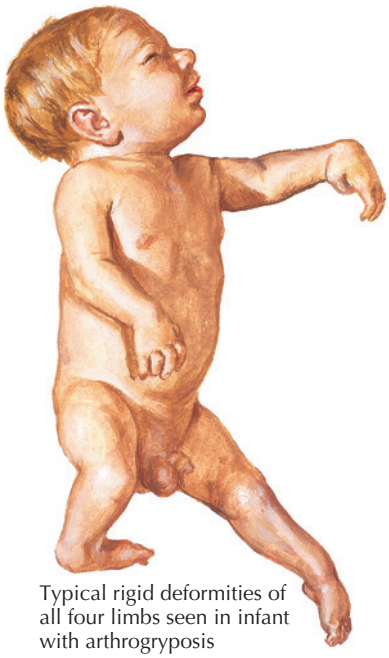
Treatment

- Daily stretching to prevent contractures
- Proper positioning in wheelchair or adaptive equipment helps decrease muscle tone
- Oral medicine:
  - Baclofen: works centrally. High doses of baclofen require weaning-off period to avoid risk of withdrawal seizures and hallucinations.
  - Dantrolene: works peripherally at muscle. Risks: rarely, liver toxicity. Liver function laboratory tests should be given at start of treatment and every 3 to 6 months during treatment.
  - Tizanidine: works centrally. Side effects with medicine initiation may decrease patient compliance: dizziness, dry mouth, and sedation.
  - Gabapentin: works centrally. May require high doses.
- Baclofen infusion via an implanted pump in abdomen
- Injections: phenol or botulinum toxin A into key spastic muscles (e.g., adductors)
- Risks: chronic dysesthesia, pain, skin sloughing, peripheral edema, wound infection
- Surgery: tendon-lengthening, muscle-release procedures for joint position issues
- Intention tremor (subset): medicine options include primidone and benzodiazepines. Also, localized botulinum toxin A injections. Cooling extremity with circulating cold wrap has also reduced tremor for 30 minutes after cooling
- Seizure control with medications
- Heterotopic bone formation: see Physical Conditions to Consider in SCI, Heterotopic Ossification

LOCOMOTOR DISORDERS

History

- Surgeries related to injury: surgical muscle transfers for functional improvement, surgically implanted medical devices
- Medications: antiepileptics, tricyclic antidepressants, antispasmodics, pain medicines; other medicine for comorbid conditions
- Comorbid medical issues:
  - Related to impairment: spasticity, type and success of bowel/bladder management program, restrictive lung disease (muscular dystrophy or neuromuscular condition), nutritional support (e.g., gastrostomy tube), seizures, skeletal deformity (Fig. 12-7)
  - Related to concurrent illness: e.g., traumatic brain injury, diabetes, visual impairment, low back pain, cardiac disease, respiratory illness
- Adaptive equipment: necessary for mobility, or sports specific
- Level of functional independence: donning adaptive equipment, personal hygiene
- Level of independence in mobility: wheelchair, orthoses
- Prior training: environmental conditions, prior exercise program
- Level of aerobic and anaerobic conditioning, flexibility



Typical rigid deformities of all four limbs seen in infant with arthrogryposis

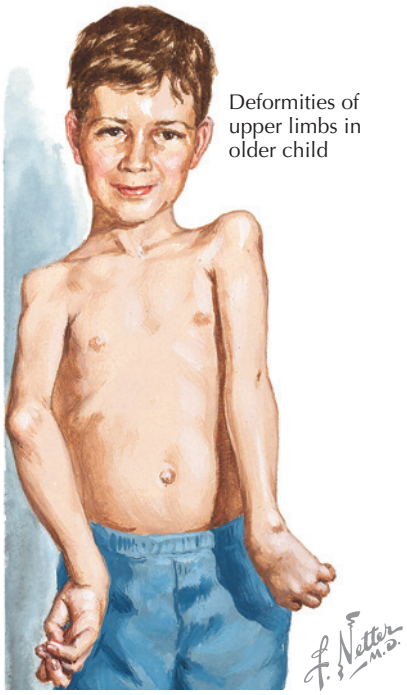


Figure 12-7 Arthrogryposis Multiplex Congenita.

Physical Considerations in Athlete

Spasticity, neurogenic bowel or bladder, seizure, joint contracture, pulmonary issues are caused by muscle weakness.

Short Stature Syndrome

Two general types:

- **Disproportionate**
  - Average-size torsos, unusually short limbs.
  - Causes: skeletal dysplasia or chondrodystrophy, caused by inherited or spontaneous gene mutations.
  - Spondyloepiphyseal dysplasia (SED) and diastrophic dysplasia: typically progressive kyphosis and/or scoliosis (Fig. 12-8). Eye complications can be present in SED.
  - Can involve joint defects, limited ROM, and high incidence of joint dislocation.
- **Proportionate**
  - Overall unusually small size for age.

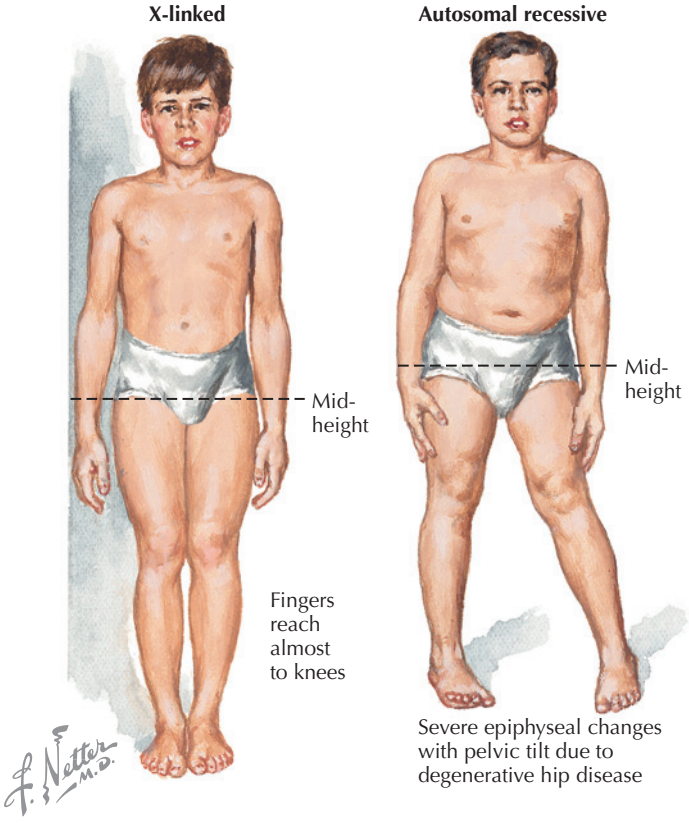


Figure 12-8 Spondyloepiphyseal Dysplasia Tarda.

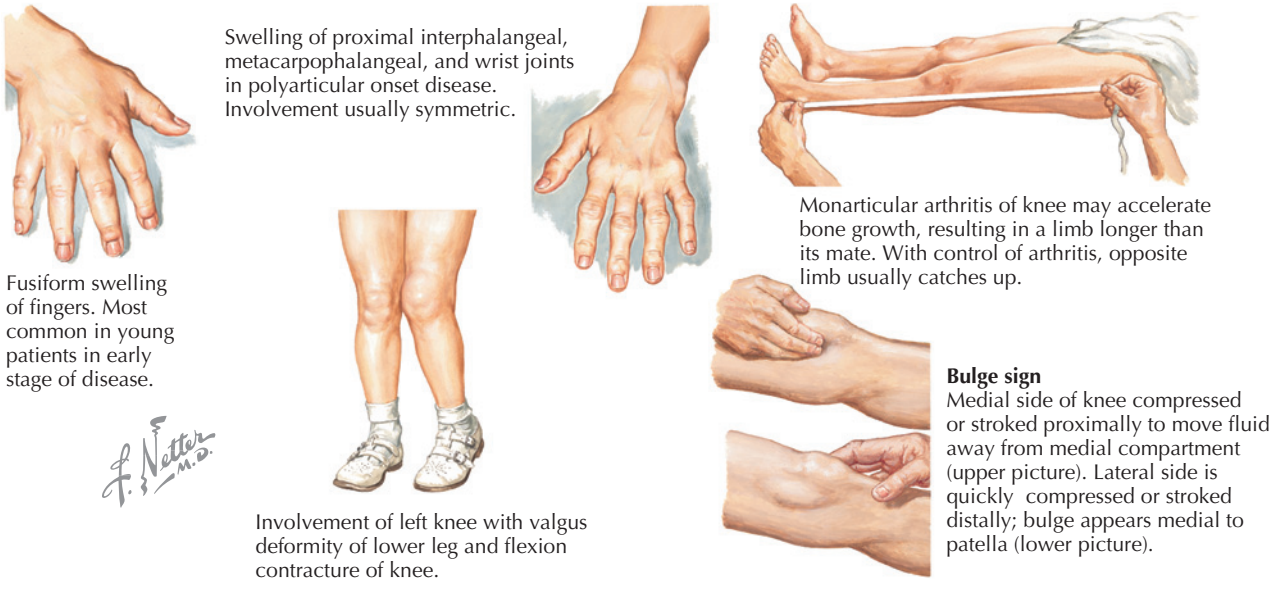


Figure 12-9 Joint Issues in Juvenile Arthritis.

- Causes: probable endocrine or growth hormone deficiency.

Muscular Dystrophy

- Mobility issues.
- Restrictive lung disease: obtain pulmonary function tests before exercise plan. Test during sleep for more accurate vital capacity assessment.
- Muscle weakness: Submaximal isokinetic training, when carefully monitored for fatigue, can increase strength in boys with muscular dystrophy.
- Resistance training should be done only in muscles with at least antigravity strength.

Friedreich's Ataxia

Issues in balance, coordination, and vision can affect function in a competition.

Juvenile Rheumatoid Arthritis (JRA)

Joint impairment: *if* significant, the athlete with juvenile rheumatoid arthritis may qualify for competitions in "les autres" category (Fig. 12-9).

VISUAL IMPAIRMENT

Classification (Classes B1-B4) is a primary sports medicine issue. Differences between classes address issues of light perception, visual acuity, and degree of visual field with best eye correction in place. Check website of the U.S. Association for Blind Athletes, www.usaba.org, for descriptions.

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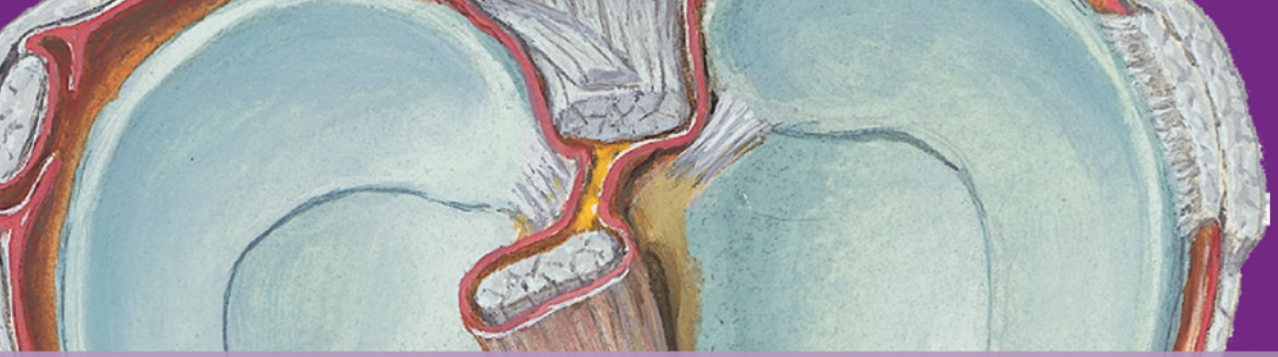

SECTION

III

Special Consideration for Athlete Populations

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Exercise Prescription and Physiology

13

O. Josh Bloom and Karl B. Fields

EXERCISE PHYSIOLOGY

Exercise can be defined as "bodily exertion, especially for the sake of training, recreation, or fitness."

Exercise Physiology

- Exercise physiology is the science of the processes and mechanisms of skeletal muscle contraction and the corresponding interaction of other body systems that facilitate and respond to skeletal muscle contraction.
- For the team physician, understanding the body's physiologic response to repetitive skeletal muscle contraction is imperative.
- Skeletal muscle contraction that exceeds physiologic limits, is inappropriate in duration or intensity, or for which the musculoskeletal system has been inadequately prepared can lead to injury.
- The relevance for clinicians lies in identification of the pathology, what triggered it, and how to correct, alleviate, and/or prevent this in both individuals and populations of active people.

Terminology

- METs (metabolic equivalents): expression of metabolic cost of exercise in terms of oxygen consumption
  - Rest defined as 1 MET = 3.5 mL O_2/kg/min
  - MET chart (Table 13-1)
- $\dot{V}O_2$ max: maximal oxygen utilization
  - Gold standard for evaluating cardiorespiratory fitness
  - Measured in mL O_2/kg/min
- Anaerobic threshold: the point at which oxygen demand exceeds oxygen availability
- Workload: the amount of energy required to complete a specific task (Table 13-2)

Table 13-1 METs IN COMMON COMPETITIVE AND RECREATIONAL SPORTS

Basketball (comp)	7-12
Biking (rec)	3-8
Dancing	3-7
Football	6-10
Golf (cart)	2-3
Jogging (5-6 mph)	7-15
Skiing (downhill)	5-8
Soccer	5-12
Tennis	4-9
Volleyball	3-6

Adapted from Mead WF, Hartwig R: Fitness evaluation and exercise prescription. J Fam Pract 13(7):1039-1050, 1981.

Table 13-2 METs IN WALKING

Workload	Miles	Minutes
5 METs	1	15-18
6 METs	1.5	21-25
8 METs	2	24-29
10 METs	4	50-54
12 METs	5	70-80

Adapted from Mead WF, Hartwig R: Fitness evaluation and exercise prescription. J Fam Pract 13(7):1039-1050, 1981.

Basic Science

- Actin/myosin filaments compose the sarcomere, which is the basic unit of muscle (Fig. 13-1).
- This linkage between actin and myosin is facilitated via acetylcholine release at the motor endplate, which triggers depolarization and subsequent release of calcium from the sarcoplasmic reticulum. This is followed by a series of reactions, causing formation of an adenosine-triphosphate-myosin complex, subsequent change in the conformation of the myosin unit, and ultimately traction on the actin filament. This then pulls on the connective tissue components of the sarcomere, ultimately resulting in contraction of the muscle (Fig. 13-2).
- A coordinated contraction of the muscle leads to movement.
- The motor nerve center directs timing and sequencing of motor unit recruitment and firing, which facilitates coordinated movement.
- Continued muscle contraction relies on an adequate supply of adenosine triphosphate (ATP) in each sarcomere.
- ATP is provided by three interlinked, overlapping energy systems that synthesize ATP for both short periods of intense, vigorous activity and longer periods of lower level, sustained activity.

Energy Systems

PHOSPHAGENS (ATP–CREATINE PHOSPHATE SYSTEM)

- Anaerobic system used in maximum intensity exercise lasting only seconds.
- Composed of the ATP and creatine phosphate stored in the cytoplasm of each sarcomere.
- Phosphagens provide rapid resynthesis of ATP on myosin head, facilitating brief, high-intensity bursts of muscle activity.
- Particularly important during very high intensity exercise and at the beginning of exercise.

GLYCOGEN TO PYRUVATE ("LACTIC ACID SYSTEM")

- Anaerobic process that degrades muscle glycogen to pyruvate.
  - Pyruvate is an essential substrate, which can be oxidized to ATP via aerobic energy system (see "Oxidative System").
- Also directly provides some ATP, which becomes part of the phosphagen pool.
- Energy source in high-intensity, short-burst exercise (typically 3 minutes or less).
- Pyruvate can be reversibly converted to lactate, which can be transported out of the cell for use by other tissues.
  - This also serves as a negative feedback loop, as increased lactate causes metabolic acidosis, which increases the rate of ventilation and causes muscle discomfort. These reactions will eventually encourage an individual to reduce the intensity of exercise.

OXIDATIVE SYSTEM (RESPIRATORY RESPONSE TO EXERCISE)

- The aerobic energy system
- Quantitatively the most important system
- Typically use in activities lasting more than 3 minutes
- Dependent on oxygen availability at the cellular level
  - Accordingly, much of the body's physiology is structured to facilitate transport of O_2 to tissues via the cardiorespiratory system.
- Dependent on oxidation of *pyruvate*, *acetyl-CoA* (formed directly from glucose), or *free fatty acids*
- Low power, but extremely high capacity energy system

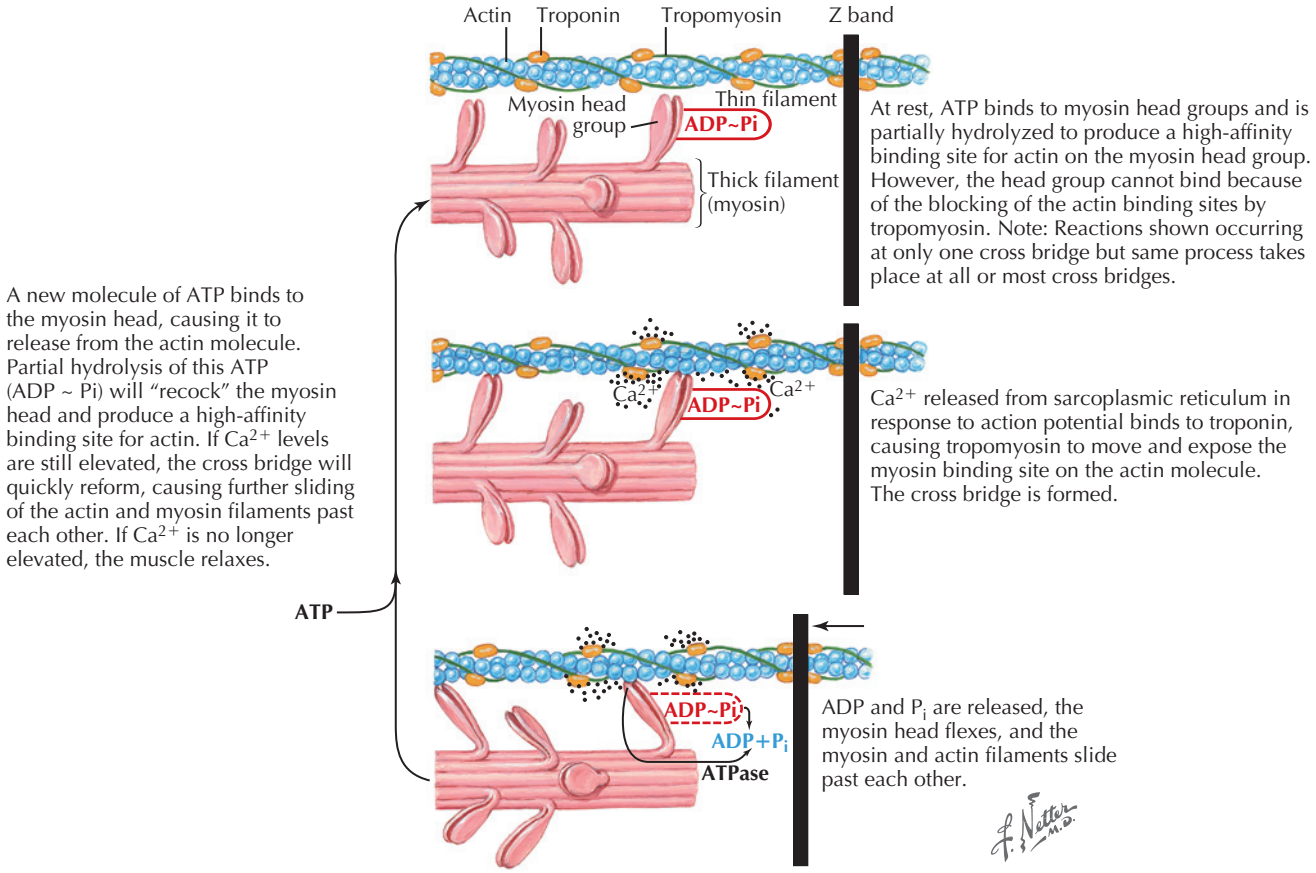


Figure 13-1 Biochemical Mechanics of Muscle Contraction.

Adaptations to Chronic Exercise ("Training")

- Musculoskeletal and cardiorespiratory systems are highly adaptable to regularly performed exercise.
- Adaptation to repetitive exercise can be understood in terms of several factors, which can be represented by the mnemonic P-ROIDS.
  - **P**rogression: This involves a gradual increase in intensity, duration, and difficulty of physical activity to improve strength, endurance, and sport-specific skills.
  - **R**eversibility: Training is generally continuous or cyclic in nature, owing to the rapid loss of benefits from conditioning when people stop exercising.
    - Inactivity for as little as several days can lead to reductions in work capacity.
  - **O**verload: This involves exercising above normal levels via combinations of type of activity, intensity, duration and/or frequency.
  - **I**ndividual **D**ifferences
  - **S**pecificity: Specific training develops specific adaptations beneficial to a particular sport/activity.
- Training should focus on the energy system that is most important to that sport (e.g., distance runners spend more time working on the oxidative system).
- Modern training programs typically incorporate cross-training because training focused on optimizing all energy systems can improve sports-specific performance (e.g., dry-land and weight training can improve performance in skiers).

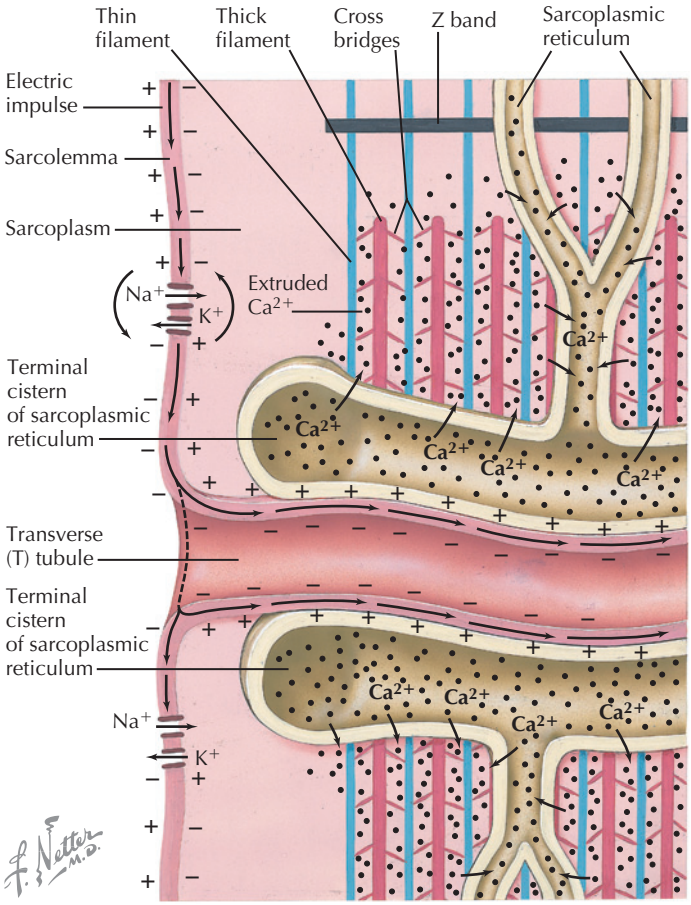
Types of Exercise

- Aerobic exercise: fueled largely by oxidative energy system and in place when oxygen supply/delivery to tissues is adequate for sustained, low- to moderate-intensity exercise.
- Anaerobic exercise: typically high-intensity, shorter duration exercise that exceeds capacity of oxidative system and is fueled largely by phosphagens and glycogen to pyruvate energy systems.
- Resistance exercise.
- Range of motion/stretching/warm-up/cool-down exercise (Fig. 13-3)
- Flexibility exercise: increases the individual's ability to lengthen the muscle unit and improve motion around specific joints.
- "Lifestyle activity": This is the cumulative, nonstructured, moderate-intensity physical activity in which individuals partake throughout a typical day (e.g., taking the stairs, walking in from the parking lot, etc).
  - 10 minutes three times daily of moderate-intensity exercise facilitates changes comparable to the health benefits of 30 minutes of continuous exercise.
  - Increasing lifestyle activity is as effective as structured activity for improving cardiorespiratory fitness and reducing cardiovascular disease risk factors.

EXERCISE PRESCRIPTION

General Considerations

- An exercise prescription is a tool to teach, coach, and educate patients concerning opportunities to improve overall health and well-being, as well as ways to enhance exercise and sport-specific performance; it should also address health risk factors.
- A written exercise prescription has been demonstrated to increase activity level more than verbal advice alone.



Electric impulse traveling along muscle cell membrane (sarcolemma) from motor endplate (neuromuscular junction) and then along transverse tubules affects sarcoplasmic reticulum, causing extrusion of Ca^{2+} to initiate contraction by "rowing" action of cross bridges, sliding filaments past one another.

Figure 13-2 Initiation of Muscle Contraction by Electric Impulse and Calcium Movement.

Implement As Part of Clinical Practice

- Identify individuals who would benefit from exercise prescription.
  - Identify conditions amenable to exercise therapy.
- Assess patient's activity level.
- Educate patient about the benefits of exercise.
- Assess patient interest, motivation, and goals.
  - Strive to find an activity/activities that are sustainable.
- Monitor progress; assess barriers.

Initiation of an Exercise Program

- Start slowly, allow fitness level to improve.
- Goal is long-term lifestyle change.
- Initially, prescribe shorter periods of light- to moderate-intensity exercise and build in duration, frequency, and intensity over time.

Safety

- Data is scarce, but reassuring.
  - Overwhelming majority of medical-related problems are soft tissue and overuse injuries.
  - Encourage warm-up and cool-down.
  - There is little scientific evidence to support that preactivity stretching prevents injuries.
- Sudden death during exercise is rare.
  - Vast majority (>80%) of instances are caused by coronary heart disease.
  - Risk is greater in sedentary people who start vigorous activity.
    - Relative risk of sudden death during vigorous activity is 2.1 times that of risk during less strenuous activity.
  - Regular activity is protective.
  - Encourage slow start, consistency, and progress over time.

Basic Principles of Exercise Prescription

- Tailor prescription to individual patients, based on their goals, physical/financial/time considerations, medical conditions, and motivation.
- Prescreen as indicated:
  - History and physical examination is the most important tool.
    - Exertional chest pain, syncope, near-syncope, disproportionate dyspnea on exertion, and marked decrease in exercise capacity are red flags warranting further evaluation.
  - Conduct functional/orthopedic evaluations as needed.
  - Administer exercise stress testing in adults if moderate or high risk (Table 13-3 and Box 13-1) for occult coronary heart disease; symptomatic individuals; or individuals planning on vigorous exercise (workload greater than 6 METs)
    - Can use exercise stress test as a motivational tool at times.

Elements of Exercise Prescription (FIT'M)

Frequency

Ideally five or more times per week

Intensity

Instruct patient on methods to assess intensity of their exercise:

- Rating of perceived exertion
- Borg scale (Box 13-2)
- $\dot{V}O_2$ max
- Max predicted heart rates can be calculated by subtracting age from 220
- "Talk test"—defined as the ability to talk without significant breathlessness; rough marker of moderate or lower intensity exercise

Time/Duration

- Current guidelines call for 30 to 60 minutes of exercise most, to all, days of the week.
- Obesity data call for 90 minutes of exercise daily.

Mode

- Growing body of evidence supports the benefits of cross-training in regard to injury prevention, improved function, fitness level, and compliance.
- Flexibility exercises and disciplines such as yoga or Pilates may be most useful in those who wish to pursue activities that require excellent flexibility and in older individuals who tend to lose mobility around joints with the aging process (Fig. 13-4).

Screening/Exercise Testing

- Exercise stress testing (ETT) indications
  - Diagnosis of occult/suspected disease
  - Prognosis/management of a previously diagnosed problem (e.g., evaluating antihypertensive therapy, antianginal therapy)
  - Development of an exercise prescription and determining functional capacity
- The American College of Sports Medicine (ACSM) recommends screening exercise stress testing for:
  - Asymptomatic individuals with diabetes who plan to start moderate or vigorous exercise programs

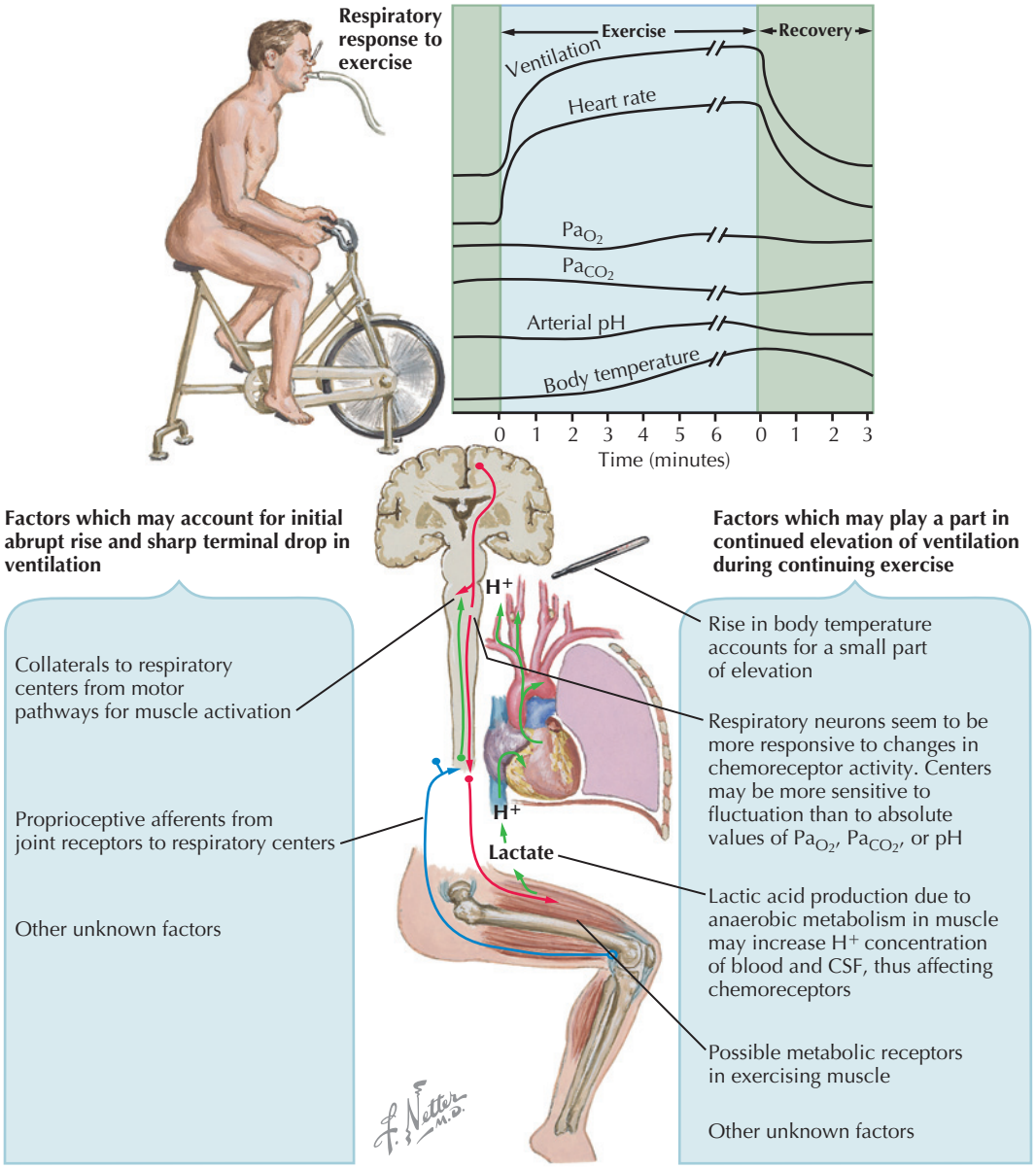


Figure 13-3 Physiologic Response to Exercise and Cool-down.

Table 13-3 RELATIVE RISK FOR EXERCISE PROGRAMS

Moderate risk	Two or more cardiac risk factors for individuals who wish to begin a vigorous exercise program (women >55 years, men >45 years)
	Patients who have positive family histories and have two additional risk factors
	Past history of coronary artery disease or stable heart disease without recent problems
	Chest pain or other symptoms not considered classic angina
High risk	Known coronary artery disease
	Structural heart disease
	Multiple uncontrolled or poorly controlled cardiac risk factors
	Chest pain or other symptoms of unstable angina
	Recent cardiac event

BOX 13-1 *Major Cardiac Risk Factors (American Heart Association)*

Nonmodifiable factors:
Male gender
Increasing age
Heredity
Modifiable factors:
Smoking
Hyperlipidemia
Hypertension
Diabetes mellitus
Physical inactivity
Obesity and overweight

Modified from The American Heart Association: Risk Factors and Coronary Heart Disease, AHA Position Statement.

BOX 13-2 *Linear 6-to-20 Borg Scale of Perceived Exertion of Pain*

6	
7	Very, very light
8	
9	Very light
10	
11	Fairly light
12	
13	Somewhat hard
14	
15	Hard
16	
17	Very hard
18	
19	Extremely hard
20	

From Morrison C, Norenberg R: Using exercise test to create the exercise prescription. Primary Care: Clinics in the Office Practice, 28(1):137-138, 2001.

- Asymptomatic men older than 45 years or women older than 55 years with two or more risk factors who plan to start a vigorous exercise program (see Table 13-3 and Box 13-1)
- American College of Cardiology/American Heart Association (ACC/AHA) recommends screening exercise stress testing for:
  - Asymptomatic persons with diabetes mellitus who plan to start vigorous exercise (Class IIa)
  - Asymptomatic men older than 45 years or women older than 55 years who plan to start vigorous exercise (Class IIb)
- The U.S. Preventive Services Task Force (PSTF):
  - Advises against routine testing of low-risk adults in general and finds insufficient evidence for exercise testing before exercise training

Mortality Data

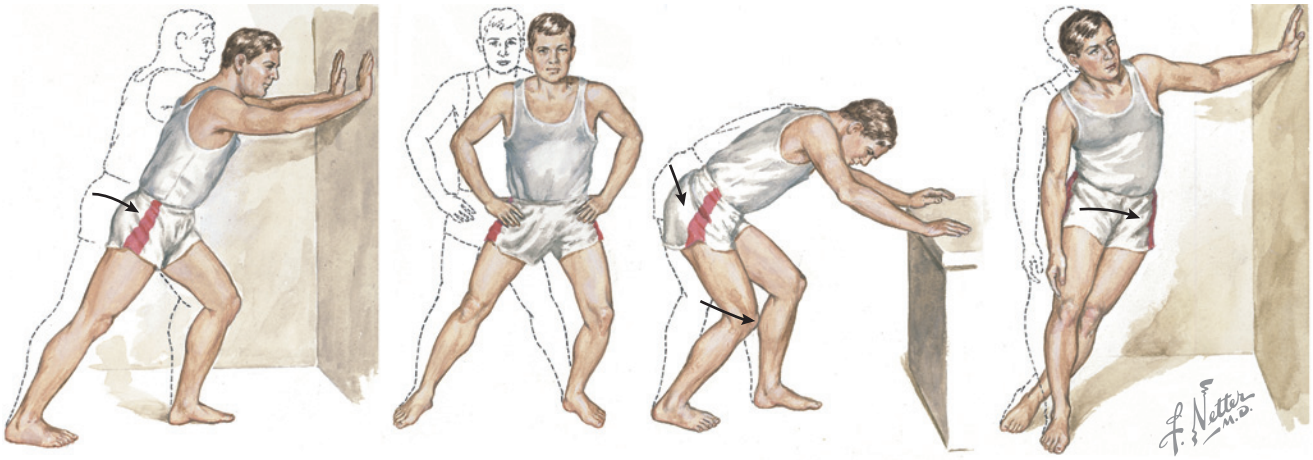
- Exercise capacity (fitness level) is an independent, powerful predictor of clinical outcomes.
- In healthy patients and those with known cardiovascular disease, peak exercise capacity is a stronger predictor for all-cause mortality than hypertension, diabetes, smoking, obesity, or hypercholesterolemia. All-cause mortality in patients who achieve less than 5 METs is double the all-cause mortality of those who achieve greater than 8 METs.
- There is an inverse relationship between fitness level and mortality (i.e., higher fitness level correlates with lower mortality).
- Patients with low fitness levels have a fourfold increased relative risk for cardiovascular death and an eightfold increased relative risk for cancer death, relative to peers with high fitness levels.
  - Those unable to achieve 5 to 6 METs on ETT are at high risk for coronary events and all-cause mortality.
  - Inability to achieve 6 METs on ETT places an individual at statistically significantly increased risk of triple vessel or left main coronary artery disease and at four times the age adjusted risk for a cardiac event.
- Poor fitness level is a modifiable risk factor.
  - Every 1 MET increase in exercise capacity confers increased survival rates of 8% to 18%.
- Doing some exercise is better than being sedentary.
  - The greatest improvements in survival rates are generally between the least and the next-to-least fit quintiles.

Special Populations

Diabetes Mellitus (DM)

EXERCISE PRECAUTIONS

- Strongly consider baseline ETT if:
  - Age older than 35 years
  - Diabetes mellitus (DM) type 2 greater than 10 years duration; type 1 greater than 15 years duration
  - Additional cardiovascular disease risk factors
  - Known microvascular disease (including microalbuminuria)
  - Autonomic neuropathy
  - Peripheral vascular disease
- Retinopathy—avoid strenuous exercise and weight training (increased risk of vitreous hemorrhage, retinal detachment)
- Significant peripheral neuropathy—risk of ulcerations/skin breakdown and fracture
  - Appropriate socks/footwear important
- Caution in extremes of weather
- Ensure adequate hydration
- Skin issues and trauma around insulin pump site
- Caution about hypoglycemia and hyperglycemia
  - Higher intensity aerobic exercise increases risk of hypoglycemia.



Straight-leg stretch for gastrocnemius and plantar fascia

Adductor stretch

Bent-knee stretch for soleus, achilles tendon, posterior tibial tendon, and plantar fascia

Abductor and iliotibial tract stretch

Figure 13-4 Stretching Exercises.

- Exercise induces a spike in endogenous insulin secretion so individuals may develop hypoglycemia relatively early in exercise.
- Delay exercise if glucose is greater than 250 and ketonuria is present.

EXERCISE IMPROVES INSULIN SENSITIVITY

- Improvements decline rapidly (typically within 48 hours after exercise).
- Accordingly, recommend that diabetics exercise daily and not skip more than 1 day between workouts.

IMPROVES GLYCEMIC CONTROL

- Meta-analysis (of 14 studies, 12 addressing aerobic exercise and 2 resistance exercise, at moderate intensity, done only 2.5 to 3.4 times per week) shows a 0.66% drop in hemoglobin A1C (irrespective of weight change or body mass index [BMI] change).

PREVENTS PROGRESSION

- Exercise prevents progression of glucose intolerance from "pre-diabetes" to diabetes mellitus.
  - Inverse relationship between level of physical activity and risk of type 2 DM.
  - Each 500 kcal expended in exercise per week is associated with a 6% decline in risk of diabetes.
  - Improved fitness level decreases mortality at each level of glycemic control.

GENERAL EXERCISE RECOMMENDATIONS

- Regular exercise is roughly comparable to taking one oral hypoglycemic medication.
- Encourage combination of daily aerobic exercise as baseline, with high-volume, moderate-intensity resistance training (twice weekly) to augment.
- Encourage 5 to 6 days per week, 30 to 60 minutes per day (90 minutes per day if obese).
- Avoid vigorous exercise when blood glucose exceeds 250 mg/dL. Check urine for ketones. If ketones present, exercise should be deferred until better glucose control is established.

Hypertension (HTN)

- Acute blood pressure response to exercise varies depending on type and intensity of exercise and severity of hypertension (HTN).
  - Blood pressure (BP) equals cardiac output (CO) multiplied by total peripheral resistance (TPR).
    - With exercise in normotensive patients, CO increases and TPR decreases, maintaining normal BP.
  - High normal blood pressure—typically will get exaggerated BP response for given workload because of only a minimal drop in TPR.
  - Mild HTN—normal CO, but no drop in TPR leads to significantly increased BP.
  - Moderate HTN—lower CO and increased TPR lead to left ventricular hypertrophy (LVH).
  - Severe HTN—diastolic dysfunction and LVH can lead to CHF with vigorous exercise.
- Hypertensive fit men and women had 60% lower mortality rate than unfit normotensive cohorts.
- Multiple studies have shown a wide variety of modes of exercise to be effective in lowering blood pressure. This BP reduction is independent of weight change.
  - It is important to note that great variability in degree of BP reduction is found in these studies (likely related to baseline differences in study populations and characteristics of exercise).
- Aerobic exercise is effective in lowering blood pressure.
  - Increases vagal tone and decreases plasma norepinephrine.
  - Decreases resting sympathetic tone.
  - Consistent aerobic exercise lowers exercise and resting HR and myocardial oxygen demand.
  - Recent meta-analysis of 127 randomized clinical trials (RCTs) irrespective of baseline showed systolic BP reductions of 3.4 to 4.7 and diastolic BP reductions of 2.4 to 3.1 mm Hg.
  - Higher initial BPs have larger reductions (average BP reduction in hypertensive patients is 7.4 to 5.8 mm Hg).
  - Benefit typically achieved by 3 months.
    - BP benefit is lost with cessation of exercise.
  - Additional meta-analysis of 54 RCTs in hypertensive and normotensive patients showed that all frequencies, intensities, and types of aerobic exercise lowered blood pressure (regardless of race, BMI, weight loss, or baseline BP).
    - Moderate intensity exercise done more than 150 minutes per week had the best results.
  - In sedentary adults, "lifestyle activity" was as effective as a structured exercise program for BP reduction.
  - Most studies involve walking, running, or biking.
  - BP has been shown to be reduced up to 22 hours after endurance exercise—this phenomenon is known as *postexercise hypotension* (PEH).
- Resistance training also effectively lowers blood pressure.
  - Regular resistance training lowered BP on average up to 5 mm Hg.
    - Circuit training at 30% to 50% maximum heart rate, with 15 to 30 seconds rest.
  - Regular resistance training decreases BP and HR response to any given workload (may be cardioprotective).
  - High-intensity lifting can dramatically increase BP (SBP > 300 documented in power lifters).
  - Much milder increases in BP with less intensity.
  - No documented clinical evidence of increased risk from increased BPs during static exercise.
  - Consistent, circuit-type low- to moderate-resistance strength training appears most beneficial in hypertensives.
- Inverse relationship between amount of exercise and risk of hypertension.
- Precautions/Recommendations/Medications
  - BP should be below 180/105 prior to initiating exercise program.
  - Exercise is safe in uncomplicated, unmedicated mild HTN.
  - Angiotensin converting enzyme inhibitor (ACE-I) and angiotensin receptor blocker (ARB) are the least likely to impact exercise tolerance.
  - Caution should be exercised with regard to heat and dehydration, especially with patients on diuretics.
  - Beta-blockers will blunt heart rate response and can decrease exercise tolerance.
  - Calcium channel blockers (CCBs) are generally well-tolerated but can cause postexercise orthostasis (cool-down period helps).
  - CCBs may be preferred in African-American patients.

Osteoarthritis

- Aerobic exercise, resistance training, and physical therapy all can reduce pain and disability in people with osteoarthritis (OA).
  - Benefits seen in strength, symptoms, function, and balance
  - Systemic review assessing a wide variety of exercises recently showed benefit on pain, self-reported disability, walking speed, and overall patient global assessment of effect
  - Systemic review of 16 trials of land-based exercise showed benefit in pain and function

- Moderate intensity exercise is safe (including in the elderly).
  - Increases in exercise intensity should be kept to a minimum during flare-ups of OA
- Isometric exercises are particularly helpful in lower extremity OA.
- Walking, biking, swimming, and low-weight/high-repetition resistance training all have demonstrated benefit.
- Range of motion exercises have not been shown to be beneficial.
- Exercise does *not* cause OA in previously normal joints.

Chronic Obstructive Pulmonary Disease

- Although exercise has not been shown to reverse the physiologic changes of chronic obstructive pulmonary disease (COPD), multiple studies confirm that exercise improves dyspnea by reducing rate of ventilation and improves exercise tolerance.
- Regular (daily), moderate-intensity exercise is most beneficial in improving functional capacity.
- Cardiopulmonary testing and periodic pulmonary function tests can be used to assess if the athlete with α-1 antitrypsin, cystic fibrosis, or other chronic pulmonary disease can achieve the MET level required for a particular sports activity without developing acidosis, hypoxia, or other complications that would limit participation.
- Athletes with COPD from sarcoidosis and cystic fibrosis have competed at elite international levels in basketball and other demanding sports, underscoring that mild to moderate pulmonary limitation rarely prevents participation.

Obesity

- Negative caloric balance (less caloric intake than caloric expenditure) is required for weight loss. Increasing physical activity is crucial to establish this negative caloric balance.
- Multiple longitudinal and cross-sectional studies demonstrate that physical activity is inversely proportional to weight.
- Many studies demonstrate that physical activity is critical to maintaining weight loss.
- Regardless of weight loss, improvements in body composition (loss of body fat) can be expected with increased physical activity.
- A large observational study of adult men demonstrated clearly that obese but fit men had significantly lower all-cause mortality than unfit, normal-weight men.
- Current data suggests that longer duration of exercise (60 to 90 minutes per day) is necessary for sustainable weight loss.
- Increased lifestyle activity has been shown to be as efficacious as formal exercise for weight control.
- Increasing lean muscle mass will increase basal metabolic rate, allowing increased baseline calorie utilization. Because resistance training has been demonstrated to substantially improve lean muscle mass, it should be considered as part of an exercise prescription for overweight and obese individuals.
- Specific weight-related considerations for overweight and obese patients:
  - Overweight and obesity are major risk factors for coronary artery disease. Accordingly, appropriate evaluation/screening is warranted prior to initiating an exercise prescription.
  - Low-impact or non–weight-bearing exercise may need to be considered for certain overweight and obese patients, because of the increased risk and prevalence of orthopedic problems.
  - Swimming and water aerobics, biking, and circuit-type weight programs should be considered in these populations.
- Thermoregulation can also be an issue in overweight and obese patients.
  - Exercising at cooler times of day, wearing loose-fitting clothing, and ensuring adequate hydration may reduce the risk of heat-related problems.

KEY CONSIDERATIONS

- A basic understanding of exercise physiology guides the physician in preparing a safe exercise prescription.
- Written exercise prescriptions appear to improve compliance with exercise recommendations.
- The exercise prescription should incorporate frequency, intensity, time, and mode (FIT'M).
- Physicians should decide which patients merit ETT testing prior to beginning an exercise program.
- Patients with chronic disease benefit greatly from exercise, but their exercise prescription and medications must be individualized to maximize their health outcomes.

RECOMMENDED READINGS

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Aerobic Training

14

John C. Hill

INTRODUCTION

- In the early days of sporting events coaches and athletes learned through trial and error that they could not develop maximal endurance and maximal power simultaneously. They found that by first establishing an aerobic endurance base and later by adding faster training they could peak at the proper times.
- It was not until the 1960s that the study of exercise as a science became widespread, and gradual changes in training methods occurred in the 1970s. By the 1980s exercise science grew by quantum leaps and the explosion in scientific information did not slow during the 1990s. Now we can use evidence gleaned from studies using top cyclists, runners, swimmers, rowers, and triathletes to better understand which physiologic components can be pushed to allow performance at a higher, more economic aerobic potential than ever thought possible.
- Aerobic physical fitness is measured in three basic components:
  - Maximal aerobic capacity ($\dot{V}O_2$ max)
  - Lactate threshold
  - Economy
- Elite endurance athletes have excellent values for all three of these physiologic traits; however, this is not true for the recreational athlete. A very high measure of maximal aerobic capacity is primarily a genetic phenomenon that few individuals possess, but lactate threshold and economy can be significantly improved with proper training techniques (Fig. 14-1, see also Fig. 13-3).

AEROBIC CAPACITY ($\dot{V}O_2$)

- **Aerobic capacity is a measurement of oxygen the body can use during all-out endurance exercise.** This is accepted as the absolute measure of cardiovascular fitness. **It is referred to as *$\dot{V}O_2$ max*.** Maximal oxygen uptake is the product of the peak cardiac output (liters [L] of blood per minute) and arterial-venous oxygen difference (mL O_2 ÷ L blood)
- $\dot{V}O_2$ may be mathematically and physiologically defined as:

$$\dot{V}O_2 = HR \times SV \times a - \bar{V}O_2 \text{ difference}$$

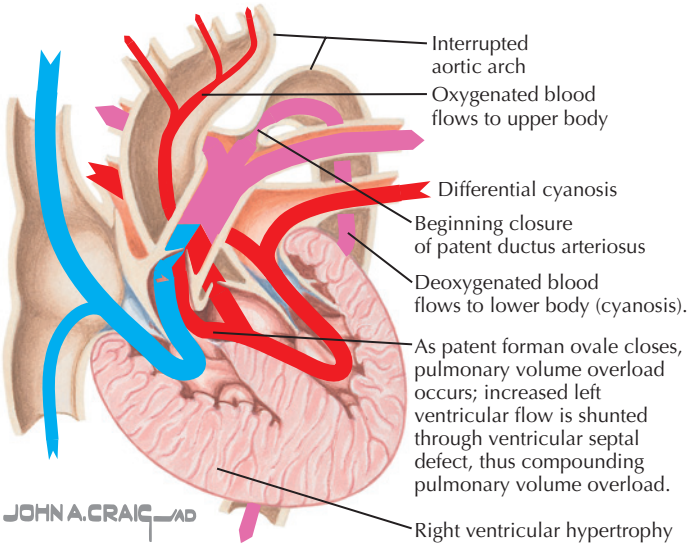


Figure 14-1 Physiologic Adaptation to Exercise.

- The cardiovascular system has central and peripheral components (Fig. 14-2):
  - **Central components** include heart rate (HR), stroke volume (SV), and cardiac output (CO = HR × SV). A primary component of high $\dot{V}O_2$ is the heart's ability to pump a large volume of blood.
  - **Peripheral components**
    - Arterial–mixed venous oxygen difference (a − $\bar{V}O_2$ difference)
    - Ability of tissues to extract and use oxygen for adenosine triphosphate (ATP) resynthesis is another primary component of $\dot{V}O_2$, particularly for muscles recruited during activity.
- Maximum oxygen uptake ($\dot{V}O_2$ max):
  - Measure of $\dot{V}O_2$ max represents maximum capabilities of oxygen transport system.
  - It is generally expressed in milliliters of O_2 consumed per kilogram of body mass per minute (ml/kg/min). It may also be expressed in liters per minute (L/min).
- Aerobic capacity is largely determined by genetics and is limited by heart size, stroke volume, blood hemoglobin content, aerobic enzyme concentrations, mitochondrial density, and muscle fiber type (Fig. 14-3). It is trainable to a certain extent, but usually takes 6 to 8 weeks of high-intensity training to significantly elevate peak $\dot{V}O_2$ max.
- Aerobic capacity usually drops as much as 1% per year after age 25 in sedentary individuals (Table 14-1). Athletes who regularly train using high-intensity workouts will loose smaller percentages of their $\dot{V}O_2$ max and the decline will not begin until their late 30s.
- On average, women's aerobic capacities are about 10% lower than men's.

LACTATE THRESHOLD

- Aerobic capacity alone is not a good predictor of performance. If all the participants in a race such as the Tour de France have similar aerobic capacities, why do only a select few consistently win? It may seem counterintuitive, but it's possible, in fact often the case, that the winners have a lower $\dot{V}O_2$ max than some of the cyclists who finish in the middle of the pack. It is the person who can maintain the highest value of $\dot{V}O_2$ for an extended length of time, riding faster with more power, who will win. **This sustainable high value is the *lactate threshold*, sometimes referred to as the *aerobic threshold*.** It is the point at which lactic acid begins to accumulate in the circulatory system faster than the body can clear it; at this point energy production shifts from a dependence on fat and oxygen metabolism to a dependence on glycogen. The higher this threshold is as a percentage of the $\dot{V}O_2$ max, the faster the athlete can perform for extended periods of time. Individuals with higher lactate thresholds will generate more power and sustain the effort longer. Eventually, as lactate reaches a high enough level in the blood, there is no other option but to slow down and allow the lactic acid to clear. Lactate itself is not detrimental; recent data illustrate that lactate, up to a certain point, may be used for energy production as well—too much is bad, but some may be good.
- Sedentary individuals experience lactate threshold at 40% to 50% of their $\dot{V}O_2$ max. Highly trained aerobic athletes who already have a higher $\dot{V}O_2$ max experience their lactate threshold at 80% to 90% of their $\dot{V}O_2$ max. Clearly, if two runners have identical maximal aerobic capacity, but one runner has a

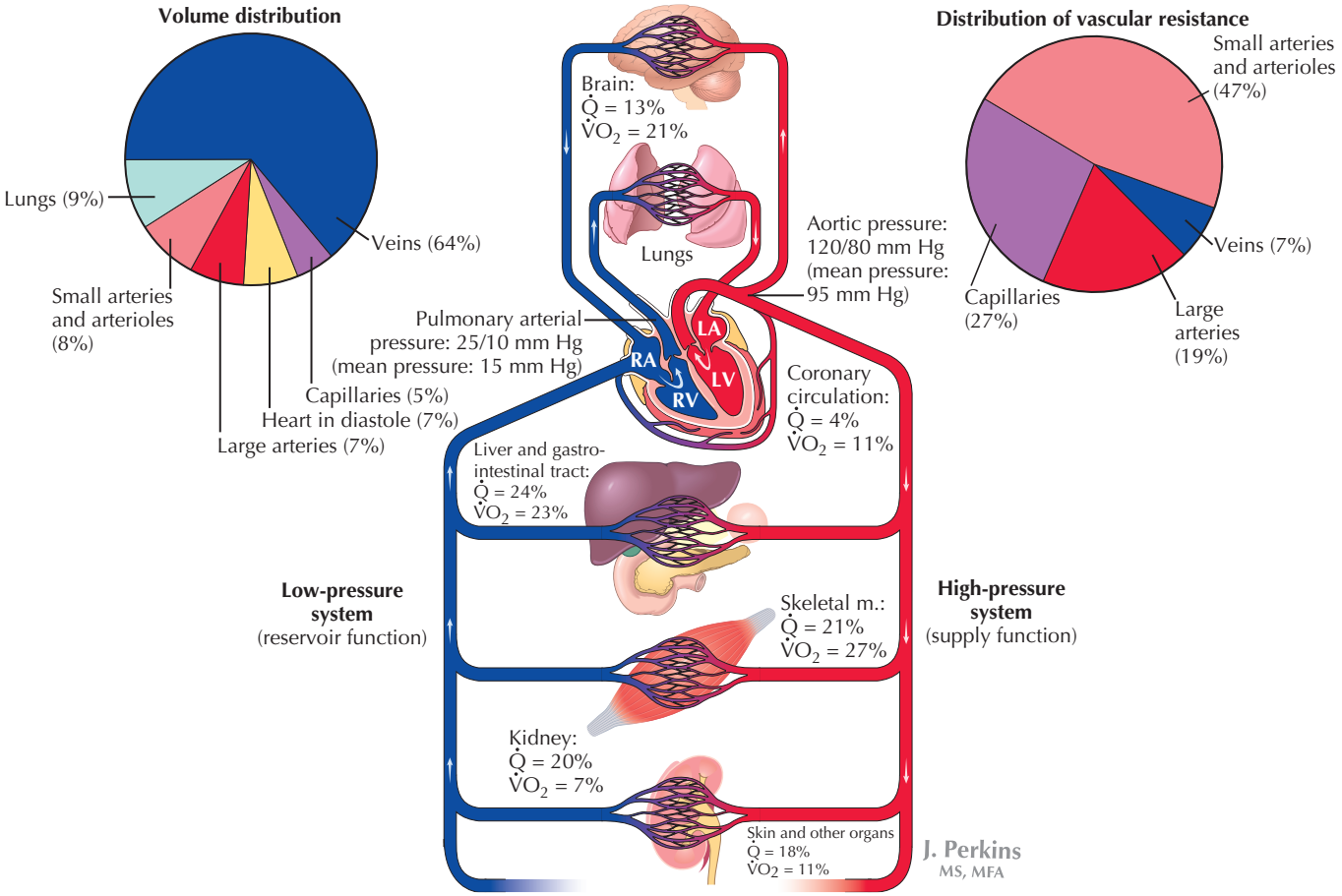


Figure 14-2 Components of the Cardiovascular System.

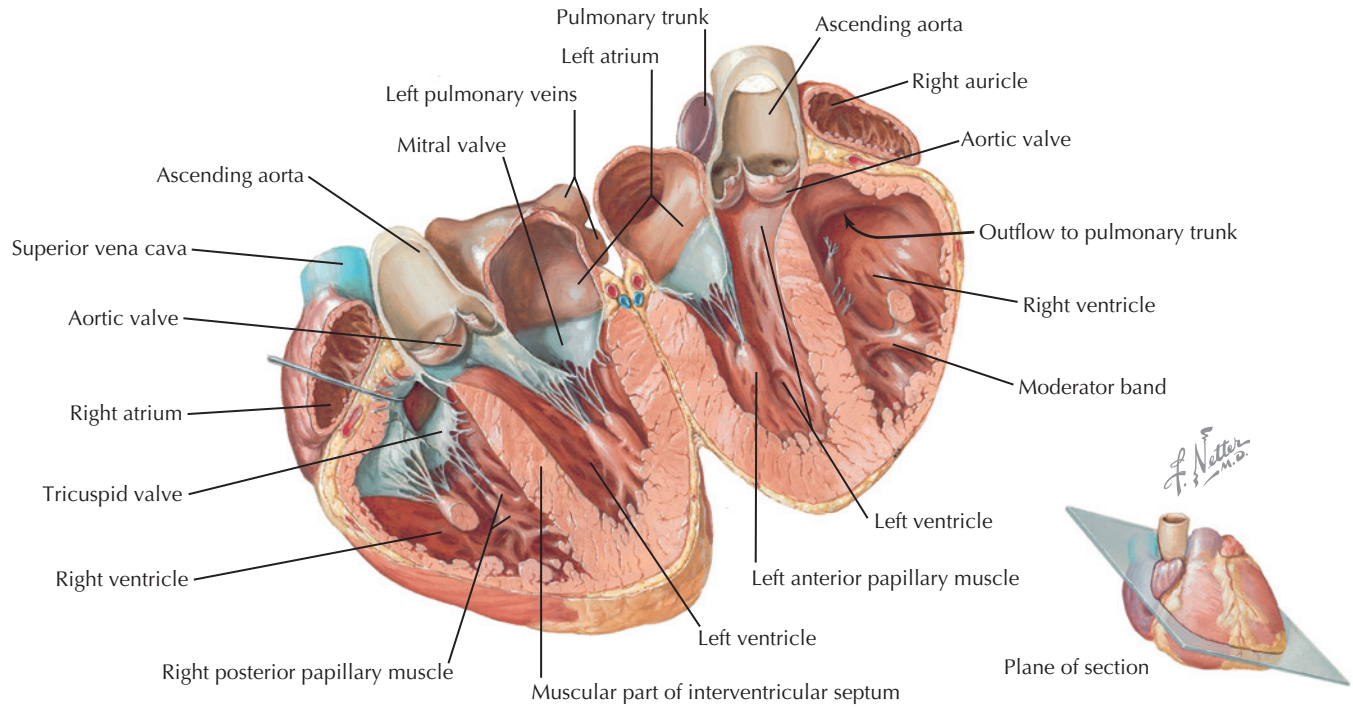


Figure 14-3 Anatomy of Right and Left Sides of the Heart.

Table 14-1 MEN'S AEROBIC FITNESS CATEGORIES ($\dot{V}O_2$ max in mL/kg/min)

	Ages			
Category	**13-19**	**20-29**	**30-39**	**40-49**
Very Low	<35.0	<33.0	<31.5	<30.2
Low	35.0-38.3	33.0-36.4	31.5-35.4	30.2-33.5
Fair	38.4-45.1	36.5-42.4	35.5-40.9	33.6-38.9
Good	45.2-50.9	42.5-46.4	41.0-44.9	39.0-43.7
Excellent	51.0-55.9	46.5-52.4	45.0-49.4	43.8-48.0
Superior	>56.0	>52.5	>49.5	>48.1

lactate threshold at 70% of his or her VO_2 max, and the other's lactate threshold is 90% of his or her VO_2 max, the latter runner has a significant physiologic advantage in a head-to-head endurance race (Table 14-2).

- Compared to aerobic capacity, which is primarily a genetic gift, lactate threshold is highly trainable. **Exercise physiologists have proven in multiple studies and in the Tour de France that programs based on improving lactate thresholds are highly effective.**

ECONOMY

- Compared to recreational runners and cyclists, elite runners and cyclists use less oxygen to hold a steady submaximal pace. When you observe world class runners, it appears that they are moving effortlessly. Elite athletes are using less energy to produce the same power output. Similarly, a large sport utility vehicle and a hybrid car going from St. Louis to Kansas City at exactly 60 m.p.h. arrive in Kansas City at exactly the same time, but the hybrid uses much less fuel. Economy is using less oxygen and calories to perform the same amount of work. This is an obvious advantage in endurance racing. The longer the event, the more significant this factor is (Fig. 14-4).
- Studies have shown that an endurance athlete's economy improves if he/she:
  - Has a high percentage of slow twitch muscle fibers (primarily genetic)
  - Has a low body mass index
  - Uses light and aerodynamic equipment that is well fitting
  - Has low psychological stress
  - Limits body frontal areas that are exposed to wind
  - Eliminates useless and energy-wasting movements
- **Fatigue can also have a negative impact on economy.** When muscles are tired, technique becomes sloppy and economy declines. The longer the race, the more critical economy becomes in determining the eventual winner.
- **Just as with lactate threshold, economy is highly trainable**. Economy will increase with all aspects of endurance training, but sport specific-activity is critical; the only way to train swimming economy is by swimming. The only way to train cycling economy is by riding your bicycle. You cannot improve your cycling economy by rollerblading.

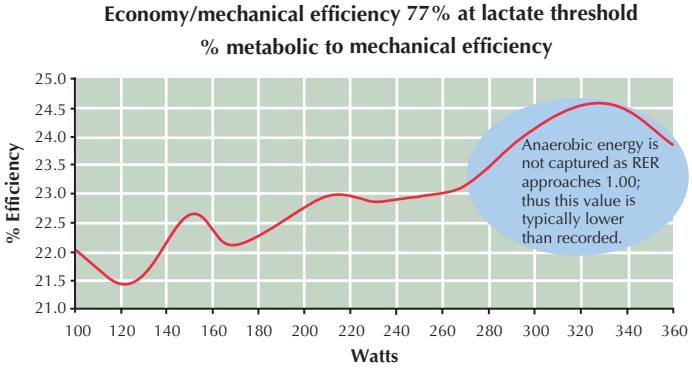


Figure 14-4 Mechanical efficiency. Mechanical efficiency reached a steady state of 23% prior to the lactate threshold. This indicates that overall economy is 77%.

LACTATE THRESHOLD TRAINING

Is Lactic Acid Harmful?

- During anaerobic metabolism, glucose is the primary fuel used in glycolysis. Lactic acid is one of the breakdown products that can be easily detected in the blood. Early research done at the Harvard Fatigue Laboratory by C. A. Knehr, D. B. Dill, and William Neufeld theorized that lactic acid production during intense exercise was caused by a lack of oxygen at the cellular level. The Harvard group felt that lactate was an indicator that the body was in an oxygen deficit and that anaerobic energy production had begun.
- We now know that lactic acid is always being produced, even at rest. A consistently low level of lactic acid in the serum does not mean that during rest and moderate exercise lactic acid is not being produced, only that the body can clear lactic acid from the blood as fast as it is generated. With high-intensity exercise, even when there is an adequate supply of oxygen, glycolysis becomes very active. The rise in lactate does not

Table 14-2 PHYSIOLOGIC PROFILES AND COMPARISONS OF COMPETITIVE CYCLISTS AS THEY PERTAIN TO TRAINING AND RACE STATUS

Category	Trained cyclists	Well-trained	Elite	World class
Training and race status				
Training frequency	2-3 times a week	3-7 times a week	5-8 times a week	5-8 times a week
Training duration	30-60 min	60-240 min	60-360 min	60-360 min
Training background	1 year	3-5 years	5-15 years	5-30 years
Race days per year	0-10	0-20	50-100	90-110
UCI ranking	None	None	First 2000	First 200
Physiologic variables				
Max wattage at LT	250-400	300-450	350-500	400-600
Relative W-max (W/kg)	4.0-5.0	5.0-6.0	6.0-7.0	6.5-8.0
$\dot{V}O_2$ max (L/min)	4.5-5.0	5.0-5.3	5.2-6.0	5.4-7.0
$\dot{V}O_2$ max (mL/kg/min)	64-70	70-75	72-80	75-90
Economy (W/L/min)	72-74	74-75	76-77	>78

From Jeukendrup AE et al: The bioenergetics of world class cycling. J Sci Med Sport 3(4):414-433, 2000.

prove that the body is in an oxygen debt (anaerobic), only that a significant amount of glycolysis is necessary to keep up with the energy needs of the muscle.

- To further compound the problem of rising lactic acid levels, sometimes there is not enough oxygen supplied to the muscles during high-intensity exercise. When glycolysis becomes a significant contributor to energy needs and oxygen concentrations are inadequate, then lactic acid levels in the blood rise at an exponential rate. These high lactate levels in your serum and muscles inhibit the muscles' ability to contract properly. You must either stop completely or at least slow down in order to recover.
- The point at which lactic acid begins to rise in your blood depends on your $\dot{V}O_2$ max and your lactate threshold. This is the point at which lactic acid production is greater than your body's ability to clear it and when effort becomes disabling. At race pace, many competitive athletes will stay just below the lactate threshold, which allows them to use both aerobic and anaerobic energy. **An important aspect of training is to shift the lactate threshold so it corresponds to a higher percentage of your maximal oxygen consumption.**

Determining the Lactate Threshold

- An exercise test can determine an athlete's lactate threshold. Lactic acid accumulates in the blood when the requirements of energy production are so high that the body can no longer effectively clear the lactate from the blood. This acid begins to shut down the muscles' contractile mechanisms and often causes a burning sensation. During exercise a cyclist can perform up to certain intensity without building up much lactic acid in the blood, but when this threshold is crossed lactic acid builds in the muscles and fatigue ensues. **The level of exercise intensity at which contractile forces in the muscle begin to shut down is the lactate threshold** (Fig. 14-5).
- The best way to determine the lactate threshold for a cyclist is to have the cyclist ride a stationary bike at increasing intensities in the laboratory. The same type of test can be done for a runner using a treadmill. A small sample of blood is taken from the finger or earlobe and analyzed for lactic acid. The workload is then slightly increased. The sampling process is repeated every few minutes after the workload is increased (Fig. 14-6). Lactic acid values are plotted against oxygen consumption and heart rate. When the lactic acid level precipitously rises, it is noted as the lactate threshold. Rise in respiratory rate corresponds to increasing lactate levels. Exercise is continued to a point of absolute exhaustion, which indicates $\dot{V}O_2$ max. A range of heart rates or power outputs is then calculated to guide the cyclist in his or her training intensity (Table 14-3).

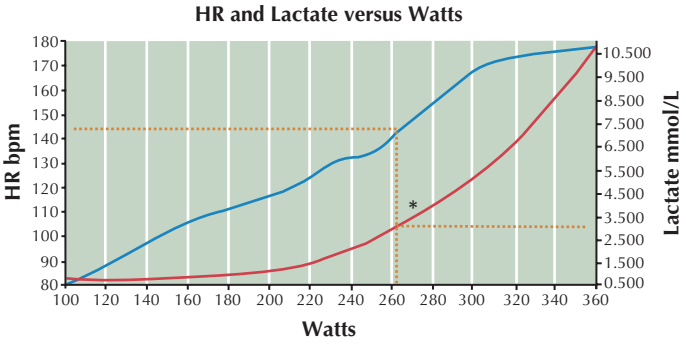


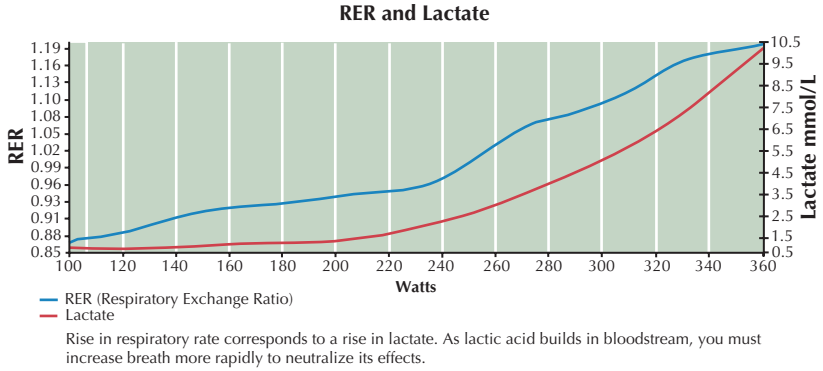
Figure 14-5 Physiologic characteristics and lactate threshold of a road cyclist. As the effort increases, the heart rate rises. But the serum lactate levels remain stable until, at an effort of 264 watts and a heart rate of 145, the lactic acid level begins to exponentially climb. This indicates the lactate threshold.

Training Programs That Increase Lactate Threshold

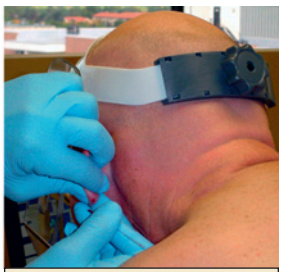
- To shift the point at which lactic acid accumulates in your blood to the right, you must train slightly below the lactate threshold. Eventually, you will be able to perform at a faster pace using a higher percentage of your $\dot{V}O_2$ max (Fig. 14-7).
- The type of detailed assessment of the lactate threshold as previously described is not available to everyone. Although the test is helpful, the physiologist who performs such a test must have extensive skills attained after testing hundreds of athletes. **The key in achieving the benefits of lactate training is the ability to accurately measure lactate in a field setting, rather than in a laboratory.** If it were possible to estimate the lactate threshold without actually measuring serum levels, more athletes could access this training method. Estimates are available, using power meters and/or heart rate and respiratory rate (Fig. 14-8; see also Fig. 14-6). **The ventilatory threshold,** a point at which breathing suddenly becomes nonlinear and more rapid, correlates closely with lactate threshold.

Field Estimate of Lactate Threshold Heart Rate (LTHR)

- To find your lactate threshold heart rate (LTHR) for either bicycling or running, complete a 30-minute time trial as follows. Find a course that is relatively flat. Warm-up as you would before a short race and then begin the time trial. Start your heart rate monitor, preferably with an average heart rate mode, immediately. The effort of this time trial should be



Performing lactate threshold and $\dot{V}O_2$ max on cycle ergometry in the performance lab.



Obtaining lactate blood sample from earlobe.

Figure 14-6 Determining Lactate Threshold for Cyclist.

Table 14-3 EXAMPLE HEART RATE AND POWER TRAINING ZONES

RPE	Zone	Zone description	HR	Power	% of LTHR
<10	1	Active Recovery	<102	<150	65-81%
10-12	2	Endurance	103-125	151-200	82-88%
13-14	3	Tempo	126-141	201-245	89-93%
15-16	4	Lactate Threshold	142-159	246-286	94-100%
17-18	5	$\dot{V}O_2$ max	160-165	287-350	101-105%
19-20	6	Anaerobic Capacity	165+	351+	106%+

Heart rate and power meter zones as % of lactate threshold for an athlete (see Fig. 14-5) whose lactate threshold occurred at HR of 145 and power of 264 watts.
LTHR, lactate threshold heart rate; RPE, Borg Relative Perceived Exertion scale.

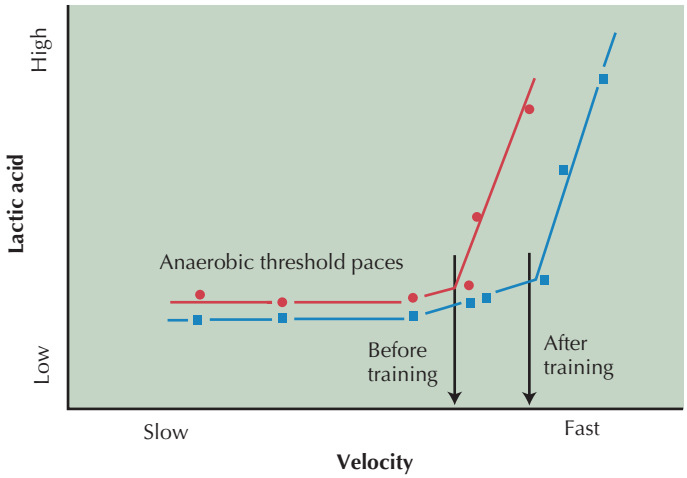


Figure 14-7 Aerobic threshold paces. After training slightly below the lactate threshold you will shift the threshold to the right. You will be able to compete at higher intensity for longer periods of time.

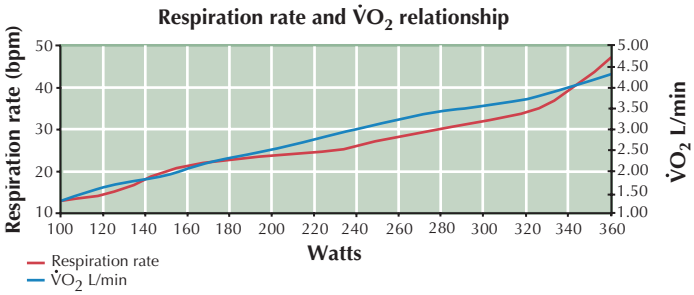


Figure 14-8 Relationship of respiratory rate to $\dot{V}O_2$ max. Hyperventilation is evidence of maximal effort and rising lactate levels.

race-like—maximum intensity. Ten minutes into the time trial (20 minutes to go) press the "lap" button on your heart rate monitor so that when you finish you have your average heart rate for the last 20 minutes. This number is an approximation of your LTHR.

- Pay attention to your heart rate whenever you feel burning sensations in your legs and the onset of heavy breathing. The more times you complete this test and observe your heart rate relative to breathing in workouts, the more refined your LTHR will become.

Perspective

- A word of caution about the previous discussion: it implies that fitness and race results can be easily quantified and predicted. You might think that scientists can take a group of athletes, predict who has elite potential, train them in a state-of-the-art facility, prod, probe, and analyze their performance, and at the end of the day, be able to predict the outcomes of all major sporting events. But this is not the case.
- Although the information about lactate threshold training is effective, it is not the real world of racing.
- Many variables are beyond scientific explanation.
- The incredible drive of certain athletes allows them to perform far beyond their abilities, whereas other genetically gifted individuals never seem to win any races.

TRAINING GUIDELINES AND STRESS

- The *American College of Sports Medicine's Guidelines for Exercise Testing and Prescription* takes the abstract concepts of aerobic training and puts them into practical guidelines that can be easily followed.
- Improvement of fitness never occurs in an unbroken continuum and fatigue is the principle reason why.
- **All successful training programs require a balance in frequency, duration, and intensity, also described as volume or workload.** We will define each of these terms and discuss the interaction each has on the other.

Frequency

- This refers to how often training sessions are done. Studies have found that training three to five times a week brings about the greatest aerobic gains for time invested, but as your overall fitness improves, so must your training frequency. When training begins, three to five workouts per week will produce rapid improvements in fitness. Aerobic conditioning frequently improves 10% to 20% within a few weeks. If the same novice athlete were to train more frequently, instead of more rapid improvement, he or she would see a decrease in fitness because of overtraining. Elite athletes training for a spot on the Olympic may need to schedule 12 to 15 workouts each week and may only improve their fitness by 1% because they are so close to achieving their full potential.
- Ideal frequency of workouts depends on current body adaptations. For example, an experienced cyclist who has not trained for 3 weeks should restart his/her training regimen at a lower frequency than where he/she left off 3 weeks earlier to avoid the risk of overtraining.

Duration

- Individual training sessions may vary considerably in length. Some may last several hours with a goal of improving aerobic fitness; others are short in length and will promote either speed or recovery. As with frequency, the duration of a training session depends on an athlete's conditioning. As fitness improves, the athlete's body will be able to tolerate longer training sessions.
- The appropriate length of a workout is directed by the length of a race. If a runner never competes in a race longer than 5000 meters, there is no real benefit to 4-hour runs. However, if the runner competes in ultra-marathons that last 12 to 30 hours, he/she may train less frequently, but will need to train for a much longer duration.

Intensity

- Frequency and duration are much easier to quantify than intensity. Intensity is often referred to as volume or workload, or simply, "hardness" of a workout. **Poor understanding of intensity is the primary cause of ineffective training programs.** If you are performing your workouts with precise frequency and duration, but you allow them to become too hard, your aerobic fitness will suffer because of overtraining. If the training sessions are too easy, again your aerobic fitness will suffer because of inadequate stress.
- Athletes often describe their training in terms of volume. For example, if they rode 5 days last week for 1 hour each day, then they would relate that they trained for 5 hours. This actually describes the volume of training (frequency × duration), but does not quantify intensity. **A better summary of training is workload, defined as a combination of volume and intensity.** Understanding how much effort or power went into each workout gives you a better understanding of training stress. Table 14-3 provides three ways to assess intensity. For example, if your goal is to ride at the pace described as "Tempo" (Zone 3) in the table, at 90% of your lactate threshold for 30 minutes, then you could quantify this by using a heart rate monitor, a power meter, or the Borg Relative Perceived Exertion scale. Planning your workload around one of these scales is critical.
- Athletes need to determine their systematic training objectives to be able to plan appropriately for intensity training. Optimal endurance adaptations take place with long workouts at lower intensities. Many seasoned athletes use an easy "over-distance" pace ("Active Recovery" in Table 14-3) for endurance training; the majority of less-intense athletes train at a slightly higher intensity ("Endurance" in Table 14-3), which can preclude optimal adaptations that occur at the slightly lower level. At least half, and usually more, of most training regimens should be spent at these lower intensities for most of the training cycle. A smaller percentage of training occurs at higher intensity levels, and amount of time spent training at these higher levels increases as the training cycle moves toward peaking for competition. Tempo pace, hill workouts, and intervals are examples of higher intensity workouts. Race pace and racing occur at and above the lactate threshold, so very little time is spent here until nearing peak performance; 5% to 15% of training may be performed at this level, with adequate recovery.
- Training intensity is the stressor that most athletes get wrong. Many athletes overtrain or train without varying intensity levels appropriately. If you work out too intensively when you should be taking it easy, then you will be tired on days when you should do high-intensity training. All training days begin to look alike and shift toward mediocrity, as easy runs become too hard and hard runs become too easy.

Fatigue

- Fatigue is the reason we are all not world champions. When and to what extent we experience weariness is the great predictor of our fitness level. **Delaying fatigue onset is the primary reason for training.** The fittest athletes are those who can best resist fatigue's crippling effects. As previously discussed, the causes of fatigue are lactate accumulation, glycogen depletion, and muscle failure. By understanding the principles of lactate threshold training, each of these causes of fatigue can be addressed.

TRAINING PRINCIPLES

Overload

- Overload is to impose work stresses that are greater than those normally encountered. The purpose of training is to cause your body to positively change in order to better manage physiologic stresses of competition. **In order to stress your body, you must present a load that challenges its current level of fitness.** This load will initially cause fatigue, followed by recovery and eventually an increased level of fitness or overcompensation. If workouts are of the right magnitude, slightly more than the body can handle, adaptation occurs and fitness steadily progresses (Fig. 14-9).
- **It is important to remember that overload happens during workouts, but adaptation and overcompensation occur at rest. In other words, the potential for fitness is produced during training sessions, but the realization of fitness occurs during rest.** If you repeatedly short-change your body of rest, you will not continually improve, and you will loose fitness. This is called overtraining. The biggest mistake most athletes make is disregarding their need to rest or reduce intensity of training. Smart athletes will know when to back off in training or rest. However, when the load of training is reduced for too long your body will adapt to this as well. It is called being out of shape.

Progression

- **Progression involves gradual, systemic increases in training intensity or volume as improvement occurs.** If you have ever done a workout or a race so hard that for days afterward you were too sore to even walk, much less run, then you have slowed your training progression. Such workouts violate the progression principle. Your body does not get stronger; rather, you loose fitness and waste time and energy.
- **You must gradually increase workloads with intermittent periods of rest and recovery.** The stresses must be greater than your body is accustomed to handling. As the intensity of the workload is increased by small increments, usually 5% to 15% every week to week and a half, then you can avoid overtraining and injury, yet provide enough stress for adaptation to occur. Cumulative training volume should not exceed 5% to 15% per year, and athletes training more than 600 hours per year should probably not increase volume by more than 10% per year. Workload increases are largely individual matters, especially with regard to intensity (Fig. 14-10).

Periodization

- **Periodization** is the structuring of training hours for a given cycle to produce a progressive increase of training stress and performance. Many athletes use training cycles, frequently dividing the competition year into 4-week cycles (meso- or micro-cycles), although experienced athletes may individualize lengths. Each cycle is planned to stimulate the proper training response for that part of the year relative to peak performance and competition. Many athletes will increase intensity in a stepwise manner over the first 3 weeks of a micro-cycle, then scale back hours and intensity during the final week of the cycle. Again, experienced athletes individualize this pattern. The training year may also be broken into different stages or cycles (macro-cycles) such as base, building

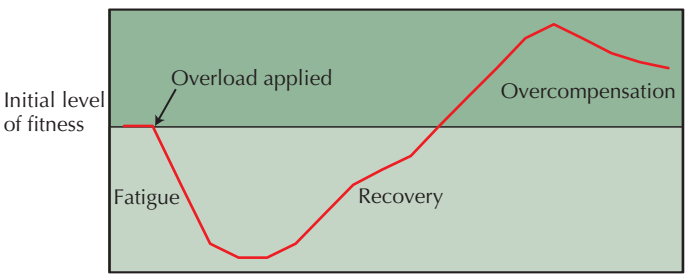


Figure 14-9 Overcompensation and Improved Fitness Resulting from Training Workload.

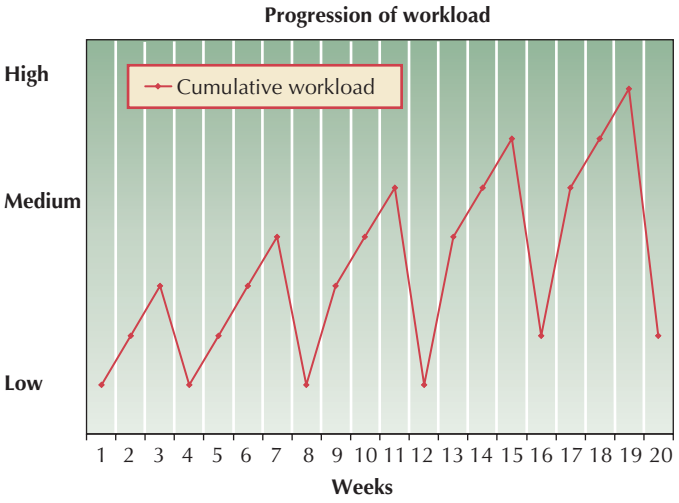


Figure 14-10 Example of Weekly Cumulative Workload Progression.

or intensity, and peak and/or race (Fig. 14-11). Periodization takes place from cycle to cycle (micro and macro).

- The basic premise of all periodization programs is that training should progress from general to specific. Early in the season, training is focused on maintaining weight, strength training, and general aerobic fitness. Later in the season, more time is spent focused on a specific sport. The reason for dividing the year into specific periods is to emphasize specific aspects of fitness while maintaining others developed in earlier periods (see Fig. 14-11). Trying to improve all aspects of training simultaneously is impossible for the athlete to handle. Periodization of training helps to reduce injury, maintain flexibility, and limit burnout. Scientific evidence to support such training programs is scant, however much the system makes logical sense. In addition to the logic, the majority of the world's elite aerobic athletes follow these principles.

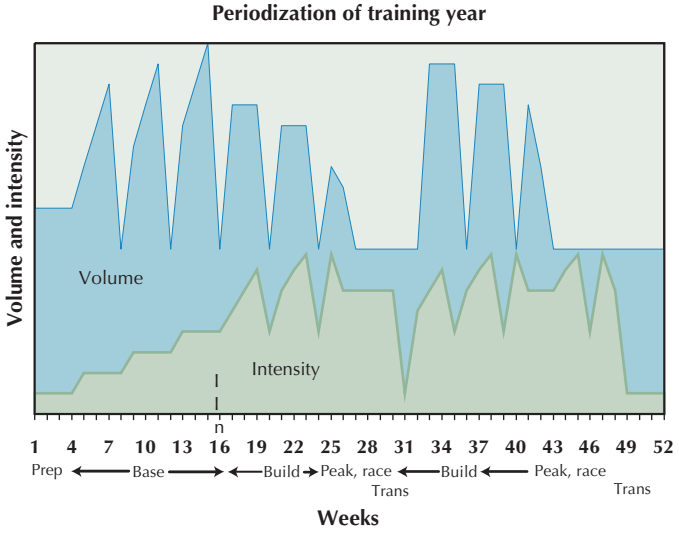


Figure 14-11 Hypothetical training year divided into periods that show the interplay between volume and intensity. Prepare to train: "base" cycle or stage is to establish speed, strength, and endurance; "build" cycle is to increase intensity and improve weaknesses; "peak" cycle is to taper for race readiness; "race" cycle is for priority races; and "transition" cycle is the recovery period.

Individualization

- **Many factors go into an athlete's capacity to handle a given workload. These factors are not purely physiologic, but are psychological, socioeconomic, environmental, and genetic.**
- Each of these factors has the ability to improve or impede fitness. Periodization of training addresses many of these individual variables. Entire books are written on the subject of periodization. Simply put, two athletes should not do exactly the same workouts.
- An athlete should not do the same training each day, or each week, or each month. **Constantly varying workouts during the week and over the year is the key to progressing in your aerobic fitness.**

Principle of Specificity

- Training specificity is best accomplished through a training program that involves movement patterns and speeds similar to a given exercise task or competition goal (e.g., bikers must bike, runner must run).
- Training with sports-specific tasks contributes to exercise economy.
- Training specificity has both neuromuscular and metabolic components, and is one of the most important conditioning principles.

SUMMARY

- Fatigue is the great determiner of fitness level. To delay its onset is the primary reason for training. The fittest athletes are those who can best resist the crippling effects of fatigue. Lactic acid is always being produced, even at rest. A consistently low level of lactic acid in the serum during moderate exercise implies that the body can clear it from the blood as fast as it is generated. The point at which lactic acid begins to rise in serum depends on $\dot{V}O_2$ max and lactate threshold. This is the point at which lactic acid production is greater than the body's ability to clear it and when effort becomes disabling.
- Aerobic capacity alone is not a good predictor of performance. It is the person who can maintain the highest value of $\dot{V}O_2$ for an extended length of time who stands the best chance of winning the race. This sustainable high value is equivalent to lactate threshold. The purpose of aerobic conditioning is to cause your body to positively change in order to better manage physiologic stresses of competition. In order to appropriately stress your body, you must present a load that challenges its current level of fitness. This load will initially cause fatigue, followed by recovery, and will eventually lead to an increased level of fitness or overcompensation. If the workouts are of the right magnitude, slightly more than the body can handle, adaptation occurs and fitness steadily progresses.

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Resistance Training

15

William J. Kraemer, Gwendolyn A. Thomas, and Disa L. Hatfield

GENERAL PRINCIPLES

- As physiologic demands of sports become greater and greater, prevention of injury becomes one of the most important aspects of sports performance. Physical conditioning of athletes plays an important role in preventing injury and improving athletic performance.
- Changes are mediated by increasing the athlete's physical potential to perform through enhancements of physiologic capacities (e.g., improved acid-buffering mechanism) and improved morphologic structures (e.g., increased fiber size, increased tendon thickness).
- Although strength (maximal force production) is a key factor in conditioned athletes, other trainable variables (e.g., power or rate of force development, local muscular endurance, hypertrophy of muscle) are equally important. Power equals force times distance divided by time; this equation illustrates the fundamental components of power development.
- Resistance training is only one part of a total conditioning program for athletes. Other factors complete a total conditioning program; these include cardiovascular conditioning, flexibility, speed and agility, plyometrics, nutritional components and body composition, and psychological aspects.

Needs Analysis

- Because of the paramount importance of "specificity" in training, a needs analysis should be done for each sport. The needs analysis should address the following four basic areas, and results should be reflected in the training program.
  - **Biomechanic analysis** of each sport: biomechanic movements and types of muscle actions that need to be trained (e.g., velocity, concentric and eccentric muscle actions, power, which muscles are activated).
  - **Metabolism** of each sport: primary metabolic demands (e.g., burstlike adenosine triphosphate/phosphocreatine [ATP-PC] metabolism, glycolysis, aerobic metabolism, combinations).
  - **Classic injury sites** related to each sport. Injury prehabilitation is important, as is keeping the focus on continued rehabilitation of past injuries.
  - **Individual needs** of each program. Physical testing and evaluation of the athlete's strength, power, muscular endurance, body composition, speed, and agility must be part of a successful training program. Without this analysis, decisions cannot be made as to the specifics of the exercise prescription, which ultimately must be individualized and be related to the sport demands.

Training Cycles

- Most sport-training programs consist of three basic periods:
  - **Off-season cycle:** longest period that athlete can use to prepare body for rigor of sport.
  - **Preseason cycle:** period of 6 to 8 weeks when peaking occurs and sport-specific skills are stressed to prepare for start of practice.
  - **In-season cycle:** attempt to maintain level of fitness and physiologic advantages achieved in two earlier cycles.
- Some of the major problems in sports are athletes' lack of recovery from sport- and practice-induced trauma during preseason and in-season cycles. The potential exists for overtraining in all cycles, especially when coaches add extra conditioning in sport practices.
- Quality of training and, more importantly, variation of training stimulus over different training cycles are vital to successful physiologic adaptation by athletes. They cannot simply perform exercises of the same intensity (load used) and volume (sets × repetitions) for the entire off-season cycle. By varying the workouts types and demands, the problems associated with overtraining and/or recovery can be avoided.

Periodization of Training

- Periodization is the most important training concept to help athletes achieve their goals.
- Periodization accommodates different workout styles and/or training cycles and makes specific plans for periods of rest and recovery.
- Sport practice schedules and their makeup remain a major problem in terms of an athlete's ability to recover from and repair physiologic and psychological stresses.

Specificity of Training

- Adaptations by the neuromuscular system and thus improvements in performance are specific to the exact type of resistance training performed (e.g., lifting weights intentionally or super slow will not develop power capabilities; the sole use of one-set training will not provide the needed total work to develop muscle strength, hypertrophy, or variation in training).
- Different programs result in very different training adaptations and physiologic responses. Specificity of training applies to all features of each exercise used in the conditioning program (e.g., each specific workout impacts various physiologic systems, signaling molecules, and gene expression differently).
- Although power is associated with all movement as force increases to maximum, velocity and power go to zero. Thus, optimal training for power is at a low to moderate load with high velocity. Velocity specificity of resistance training is one of most contentious issues in the field of muscle strength and power development. If athletes train at a slow velocity, they tend to increase strength only at that velocity. Resistance training also should be performed at high speed with no deceleration in the exercises (e.g., Olympic-type lifts such as hang power cleans, also jump squats, bench throws, etc.) chosen; this will increase power output or rate of force development (i.e., optimal loading for maximal mechanical power is 30% to 50% of the one-repetition maximum [1 RM]).
- Strength increases are specific to the mode of muscle action used during training.
- Most sport skills take place in less than 0.2 msec (rate of force development), which makes the ability to produce force in small amounts of time vital to performance.
- Speed repetitions, in which the athlete holds on to weight (e.g., bench press, military press), should not be used to train for muscular power because the athlete's body tries to decelerate mass by activating antagonist muscles and reducing activation of agonist muscles. Instead, power exercises such as Olympic high pulls, power cleans, isokinetic exercises, pneumatic exercises, and medicine ball exercises should be used to promote power development. When loading cannot reach the 30% to 50% range, such as in medicine ball or many plyometric exercises, velocity is being trained to contribute to the power equation as power output is lower.

- Velocity of movement typically is associated with type of resistance used, choice of exercise, and exercise modality. Terms such as *speed strength* and *power* relate to rapid development of force at high speeds of movement. Continuum of velocities is used in conventional resistance training, from very slow concentric movements (e.g., 1-repetition maximum [RM] lift) to higher-speed power movements (e.g., 30% of 1 RM). Slower, high-resistance training enhances development of high force but has little carry-over to faster velocities; conversely, high-speed training has little carry-over to development of slower-speed force. Periodization spans the entire continuum by using different loads and intensities, performed at different velocities of movement, thereby stimulating different training adaptations.

Physiologic Compatibility of Exercise Training Modes

- Aerobic endurance exercise (e.g., running and cycling), when performed concurrently with resistance training at high enough intensity and volume, has the potential to compromise increases in strength and power. High-volume running and cycling training (>80% of $\dot{V}O_2$ max directed at improvement) has been shown to impact strength, power, and type I muscle fiber size increases.
- For most sports, the phenomenon of interference can be accommodated by **prioritization of goals;** this is made possible by using periodization in the training program. Prioritization involves working on the most important training goals first and maintaining others. For example, one training phase may focus on power development, but high levels of strength also need to be maintained to optimize power.

Variation in Training

- The two most important factors in designing resistance training programs are **specificity** (discussed earlier in the chapter) and **variation.** To provide overload and thus continue to stimulate the body's adaptation, training must be novel and change in character.
- Working to muscular failure or RM at every workout may create overtraining stress and too much compression on joints. Variation also is required in this aspect of training. An RM zone of three repetitions allows an athlete to target the zone without having to struggle to squeeze out a failed repetition.
- The principle of variation relates to changes in program characteristics to match changing program goals as well as to provide changing target for the body's adaptation. For experienced lifters, it is prudent to design resistance training program cycles that vary as often as every 2 to 4 weeks (microcycles).

Task- and Athlete-Specific Strength

- *Task-specific strength* necessitates determination of specific requirements of the target activity. Movement analysis must be completed to determine muscle groups involved, type of contraction, velocity of movement, requirements for stretch-shortening cycle movements, loads lifted or moved, duration of activity, requirements for sustained high-energy output, available rest periods, and risks for injury.
- *Athletic-specific strength* takes into account the abilities of individual athletes and addresses aspects that need to be developed in the training program. The less developed a particular component of performance, the greater the window for adaptation and benefit to performance. This process has been termed *strength diagnosis* and may involve a wide range of tests of neuromuscular performance to assess all aspects of strength and power.
- Task- and athlete-specific characteristics must be combined to determine optimal design of the training program, which should involve general exercises for development of strength and power as well as sport-specific exercise. It is difficult to use only sport-specific exercises in training programs because of significant transfer from general strength power exercise (e.g., normative movements for each major muscle group) to almost every sport. Clearly, if the particular quality of strength is a high priority for a specific task (e.g., rate of force development) and the athlete is weak in this quality, training that enhances the athlete's ability in this area will have the greatest effect on sport performance.

Considerations for Women

- The same general principles apply to the training of women and men. By understanding specific gender differences (e.g., weaker upper body in women), the needed elements can be added to the training program. The average woman's maximal mean total body strength is 63.5% of the average man's; a woman's isometric upper body strength averages 55.8% of the average man's, and a woman's isometric lower body strength averages 71.9% of the average man's.
- Some initial evidence indicates that women's strength gains may plateau after 3 to 5 months of training. This plateau may be more pronounced in the upper body, where absolute muscle mass is less than in men. Thus, emphasis on development of upper lean body mass in women may be warranted for sports in which upper body strength is a limiting factor of performance.
- In some cases, women may need much more maximal strength to optimize power development and to optimize performance in power and speed types of activities. One can use a wider loading range for women than in men to optimize maximal power output in such exercises (30% to 50% of 1 RM in women compared with 30% to 40% of 1 RM for men).

DEVELOPMENT OF WORKOUT

- A basic workout must be developed for every training session in off-season and preseason programs. Acute program variables describe all possible single training sessions. Training sessions are designed by carefully examining each variable in detail and making decisions about its priority. From the time the athlete performs a single training session, training history begins.
- **Acute program variables** describe a single workout session and include the following:

Choice of Exercise

- Exercise choice involves a host of different decisions, which must be based on factors ranging from type of weight-training equipment to type of muscle action used in the workout.
- Exercises can be classified as structural (i.e., involving multiple joints) or body-part (i.e., involving an isolated joint).
- **Structural exercises** include whole-body lifts that require coordinated action of many muscle groups as well as coordination of joint movements (e.g., closed kinetic chain exercises). Most primary or core exercises are structural (e.g., squats, power cleans). These should be in every athlete's training program.
- **Body-part exercises** attempt to isolate a particular muscle group or a single joint (e.g., bicep curl). Most assistance exercises also can be classified as body-part or single-joint.

Order of Exercise

- For many years, resistance training programs have been designed to exercise large muscle groups before smaller muscle groups. Exercising larger muscle groups first results in greater

intensity and more effective training stimulus for all muscles involved.
- Another consideration is to place exercises that are being taught or practiced (especially those involving complex movements) at the beginning of the session.
- Ordering of exercises also involves ordering of various types of circuit weight-training protocols. Certified strength and conditioning specialists (CSCS) and athletes must address the question of whether one lower-body exercise should be followed by another lower-body exercise or whether one should go to an upper-body muscle group. Fitness level and exposure to such training are the major determinants.
- The final consideration is the fitness level of the athlete. Training session design should never place too much stress on athletes, especially if they are just starting to use resistance training as supplemental training for their sport. Nausea and dizziness caused by too much physiologic stress should *not* be equated with a "good workout."

Number of Sets

- The number of sets in a workout is related directly to training goals for a specific joint or musculature and constitutes part of volume calculation in strength training.
- Typically, three to six sets are used for each exercise to achieve optimal adaptation in athletes. However, not every exercise in the workout needs to have the same number of sets.
- To date, no single-set system has been shown to be superior to multiple-set programs, but some coaches still believe in the single-set mythology of the past, which evolved from the marketing wars in the 1970s among various companies.
- For some characteristics of muscle performance, such as power, many sets with few repetitions (three to six) are needed for optimal training stimulus. Therefore, power training requires a multiple-set format to expose the body to an adequate volume of exercise in training sessions.
- During the needs analysis, the practitioner will evaluate the athlete and decide what the primary goal of the resistance training program is. This training goal will be used to determine specific load, rest, and repetition arrangements.

Length of Rest Periods

- Length of rest periods between sets and exercises is an often overlooked variable in an exercise prescription.
- Rest periods determine how much of the ATP-PC energy source is recovered and how high lactate concentrations are in blood.
- It is important to know that we now understand that lactate is a buffer and not responsible for the acid base disruption, or fatigue, nor does it play a role in muscle soreness; lactate is simply a good marker of the metabolic demands of anaerobic exercise.
- Short rest periods can cause greater psychological anxiety and fatigue, perhaps because of greater effort and higher metabolic demands (e.g., high lactate production). As a result, coaches and strength and conditioning specialists may have to spend extra time preparing athletes for such high-intensity metabolic workouts in the weight room. Psychological ramifications of short rest periods must be carefully considered in designing training sessions.
- Typically, rest periods less than 60 seconds are considered very short and usually are associated with the use of lighter weights. Rest periods of 90 to 120 seconds are considered short to moderate and usually are associated with loads in range of 10 to 12 RM. As loads get heavier, more rest is needed for optimal neuromuscular recruitment. Typically rest periods $\geq$ 3 minutes are considered long. Rest periods shorter than 90 seconds should be carefully built up to over a training period of 6 to 8 weeks. Symptoms should be monitored carefully (e.g., nausea, dizziness, faint spells), and rest should be increased if adverse symptoms occur during the workout.

Amount of Resistance

- The amount of resistance used for each specific exercise is probably the most important variable in resistance training.
- Resistance is a major stimulus related to changes in measures of strength, power, and local muscular endurance.
- The amount of resistance must be chosen for each exercise in the resistance training programs.
- **RMs or specific resistance,** which allows only a specified number of repetitions to be performed, are probably the easiest methods of determining resistance.
  - Six or fewer very heavy RM resistances appear to have the greatest effect on strength measures, which contributes to the force component of the power equation for a certain amount of time. It also recruits the most motor units and therefore stimulates hypertrophy of the entire motor unit array of a muscle.
  - Resistances in the 8 to 10 RM range contribute to both strength and hypertrophy, and allow for the heavy to moderate resistance load commonly used in resistance training.
  - Twenty or more RM resistances show the greatest effect on local muscular endurance measures (e.g., number of repetitions at 75% of 1 RM, etc). This continuum makes it possible to develop a specific feature of muscular performance to varying degrees over a range of RM resistances.
  - Typically a three-repetition target zone (e.g., 3 to 5 RM, 8 to 10 RM) is used for training. This range allows athletes to target a specific repetition training zone without having to go to failure or squeeze out the last repetition (both of which increase compression on joints).
- Another method of determining resistance for specific exercises is to use **percentages of 1 RM** (e.g., 70% or 85% of 1 RM).
  - This method requires regular evaluation of maximal strength in various lifts used in the training program.
  - It is vital to the success of power training programs in which loads must be in the range of 30% to 50% of 1 RM to optimize training effects (e.g., high-velocity or explosive power training).
  - Typically this approach has been used for exercises such as the Olympic lifts and exercises in which the 1RM is known because it demands more testing or use of the estimation equation [e.g., Epley Equation 1 Repetition Maximum Prediction = 0.033(reps) $\times$ (repetition weight) + (repetition weight)] to keep up with the improvements over a training program, especially in beginners.
- The relationship between possible RM and percentages of 1 RM varies with the amount of muscle mass needed to perform the exercise. For example, leg presses require more muscle mass than knee extensions.
  - When athletes use machine resistances with 80% of 1 RM (previously thought to be primarily for strength-related prescriptions), the number of repetitions that can be performed is typically more than 10, especially for large muscle group exercises such as leg presses.
  - Larger muscle group exercises using machines appear to require a much higher percentage of 1 RM to keep them within the RM strength zone of repetition continuum. Thus, loading in machines versus free weights is related to the size of the muscle being exercised.
  - Percentages were developed for free weight exercises and do not translate very well to machine exercises, especially as the muscle mass used gets bigger (e.g., leg press, bench press).

Frequency of Training

- Elite athletes may require training frequencies of 4 to 5 days a week to see significant improvement over short periods.

Periodized training cycles use variations in training frequency to alter exercise stimulus, thus allowing recovery and enhancing the effect of exercise stimulus.

- Athletes may train twice daily to reduce volume within a single workout so that quality (intensity) of workout can be maintained at the highest level. Twice-daily regimens usually consist of morning and evening workouts, which allow 4 to 6 hours of rest between sessions.
- Training frequencies greater than twice weekly typically involve different training programs and do not repeat the same program at each workout.
- A minimal frequency of two sessions per week for a given exercise is at the core of every strength/power training program.
- Some type of variation (i.e., periodization) must be employed when consecutive training days are used.
- Progression in frequency is a key component in resistance training. Frequency of training varies, depending on the phase of the training cycle, fitness of the athlete, goals of the program, training history, exercise selection, training volume, intensity, recovery ability, and nutrition.
- Excessive soreness on the morning after a workout may indicate that the exercise stress is too demanding. In such cases, workout loads, sets, and/or rest periods between sets and training frequency need to be evaluated and adjusted downward or complete rest given for few days.
- Periodization of training is vital to address recovery over time.
- Training with heavy loads increases the recovery time needed before subsequent exercise sessions.
- In order to avoid overtraining, the ability of the individual to tolerate the training stress needs to be considered.

Periodization of Resistance Training

There are two basic forms of periodization and both have been shown to be superior to constant loading programs (e.g., the same relative load is used over time, such as 8 to 12 RM or 80% of 1 RM). Both formats can be used to design a program for any phase of the yearly training cycle.

Classic "Linear" Format

- Decreases training volume and increases training intensity as training progresses.
- Used for athletes peaking for a single performance in a strength/power sport (e.g., weightlifting, field events such as shot and discus) in a major competition (e.g., World Championships, Olympic Games).

"Nonlinear" or Undulating Format

- Developed out of need to periodize throughout multiple-competition sports in which one peak does not necessarily produce success. Shown to be very effective in college and high school settings in which schedule influences are dramatic.
- Useful in sports with long seasons in which success during the season and qualification for major tournaments and competitions are important.
- Varies training volumes and intensity so that fitness gains occur over long training periods.
- Training volume and intensity are varied using different workouts for each day to address a specific trainable feature (e.g., strength, power, muscle size, local muscular endurance); this is done by using percentages of 1 RM or near RM training zones, different volumes of work, different rest periods, different orders, and choices of supplemental exercises.

PHASES OF TRAINING

General Preparatory Conditioning

- The general preparatory conditioning phase is typical of linear periodization programs (important at start of a training program for beginners). It can also be helpful for beginners using a nonlinear program.
- Athletes should begin training with a general or "base" program that allows gradual entry into the process of physical conditioning and gives the strength and conditioning specialist the opportunity to learn more about the athlete without excessive physical and psychological stress and teach the exercises to be used in the program.
- Proper progression is one of the fundamental elements of periodized resistance training programs. Proper progression considers progressive overload, specificity, and variation to accompany the dynamic training goals and fitness levels of the athlete.
- Typical mistakes include "overshooting" or "undershooting" an athlete's physical abilities. At start of the program, the biggest problem is "too much, too soon." It is essential not to overshoot the athlete's ability to recover from the workout or to cope mentally with the required consistency of training.
- Duration of this phase is highly dependent on age, health, and fitness of the athlete (typically 2 to 4 weeks). Highly trained athletes need less time.
- The general preparatory conditioning phase allows a coach to prepare an athlete's mind and body for the challenge of the strength-training program. The goal is to improve the basic physical and mental tolerance of resistance-exercise stress.
- Educational aspects of resistance training, proper exercise techniques, aerobics, and nutrition should be stressed. Loads should be light, and the ability to concentrate with little fatigue creates the ideal teaching situation.
- Almost unlimited numbers of programs can be developed for various sports and athletes. The key to choosing the right program lies in scientific and theoretical aspects of the program design. A needs analysis is vital to designing a periodization program for specific sports and specific athletes, but strength is an important base for all programs and power is a major contributor to all sports. Although local muscular endurance may be important for particular sports, strength and power must be developed to enhance the absolute magnitude of local muscular endurance. Certain athletes may also need to concentrate on improving hypertrophy of muscle groups to help protect a joint or because they are underdeveloped (e.g., shoulders in women basketball players or football linemen, or quadriceps in athletes with past knee injuries, etc.).

Linear Periodized Programs

- Classic periodization methods use progressive increases in intensity with small variation in each 4-week **microcycle.** A classic four-cycle linear periodized program (4 weeks for each cycle) is outlined in Box 15-1. Some variation can be observed within each microcycle because of repetition range of each cycle, but the general trend for 16-week programs is steady linear increase in intensity of training program.

BOX 15-1 *General Example of a Linear Periodized Program with 4-Week Microcycles*

Microcycle 1
3-5 sets of 12-15 RM
Microcycle 2
4-5 sets of 8-10 RM
Microcycle 3
3-4 sets of 4-6 RM
Microcycle 4
3-5 sets of 1-3 RM

RM, repetition maximum.

- Each 16-week program is called a **mesocycle** (made up of microcycles), and 1-year training programs typically are composed of several mesocycles. Each mesocycle attempts to increase muscle hypertrophy, strength, local muscular endurance, and/or power toward the athlete's theoretical genetic maximum. Thus, the linear method of periodization is based theoretically on development of muscle hypertrophy and improved nerve function and strength/power. This process is repeated with each mesocycle, and progress is made in training programs.
- Training volume starts with a higher initial volume and, as intensity of the program increases, volume gradually decreases. Drop-off between intensity and volume of exercise can decrease as the training status of the athlete advances. Advanced athletes can tolerate higher starting intensities and volumes of exercise during heavy and very heavy microcycles.
- Increases in the intensity of periodized programs begin the development of needed nervous system adaptations for enhanced motor unit recruitment. Adaptations develop as the program progresses and heavier resistances or velocities of movement are used. Heavier weights demand involvement of high-threshold motor units in the process of force production. Exercises with higher power output and training velocities also use specialized motor unit recruitment patterns.
- Athletes must be careful not to progress too quickly to high volumes using high intensities. Pushing too hard can lead to serious overtraining syndromes. Although it takes a great deal of excessive work to produce overtraining effects, highly motivated athletes can easily make mistakes out of shear desire to make gains and see progress in training and performance. It is important to monitor the stress of workouts. Rest between training cycles (active rest phases) allows time for needed recovery so that overtraining problems are reduced, if not eliminated. One or two days of complete rest in a training week has been shown to reduce the chance of overtraining.
- High-volume exercise in early microcycles is the start of a linear periodized program. Thus, late cycles of training are linked to early cycles by changing the stimulus of the training. Once basic strength has been developed, specialized power training programs allow separate development of power capabilities in muscle. Maintenance or continued improvement in strength is needed for power development as loading is reduced. A major error in some training programs, especially in those for women, is to drop-off the high resistance loading needed for strength development too long while focusing on power using lighter, more ballistic loading schemes. This has led to the use of 2-week microcycles for power development and different linear progression strategies.

Nonlinear or Undulating Periodized Programs

- Nonlinear, or undulating, periodized programs are designed to maintain variation in training stimulus without holding the athlete to the strict phasing of linear periodized programs. Nonlinear programs make program implementation easier when schedule or competitive demands over long periods limit sequential variation in linear manner. They also allow variation in intensity and volume within each 7- to 14-day cycle over the course of the training program (e.g., 16 weeks). Typically, changes in intensity and volume of training vary within each 7- to 14-day cycle. For example, in 16-week programs an active rest phase of 1 to 2 weeks may be followed by another nonlinear cycle or even by a linear program, if desired. Box 15-2 offers an example of loading or intensity variation over a 5-day rotation during a 16-week mesocycle. Important to this concept is that one can also have a different priority for each mesocycle, thereby allowing more workouts to emphasize one style and at the same time keeping a lot of variation in the program (e.g., a power mesocycle or a strength mesocycle in which more workouts will be directed toward these types of workouts over the 16 weeks).
- Variation in training is much greater within each 5-day workout over 10 days. Intensity spans a maximal range of 15 RM (possible 1-RM sets versus 15-RM sets). This span of variation in repetition intensity appears to be as effective as linear programs. Workout cycles also may include other types of training protocols (e.g., plyometric protocols or 6 to 10 sets of 2 to 3 repetitions at 30% of 1 RM for power and velocity training days). The key element is variation during an acute 1- to 2-week cycle within a 12- to 16-week mesocycle.
- The athlete trains different components of muscle within each 7- to 14-day microcycle. Although nonlinear programs attempt to train various components of the neuromuscular system within the same cycle, only one feature is trained during a single workout.
- The mesocycle is completed within a certain number of workouts (e.g., 48) instead of a certain number of training weeks. Workouts rotate between different styles for each training session. If the athlete misses a Monday workout, the rotation order is simply pushed forward (i.e., the athlete performs the scheduled rotation at the next workout). In this way, no workout stimulus is missed in the training program.
- Primary exercises are typically periodized. Supplemental exercise movements (e.g., hamstring curls, abdominal exercises) also can be periodized; careful attention should be given to the type of movement capabilities. In triceps push-downs, for example, the athlete can rotate between moderate (8 to 10 RM) and heavy (4 to 6 RM) cycle intensities. Assistance exercises of supplemental training must be examined carefully to avoid interference with the primary training programs.
- Nonlinear programs accomplish the same effects and are superior to linear programs in certain situations. The key to success in all training programs appears to be variation. Different approaches can be used over the year to reach the training goals of the individual athlete in his or her specific sport.

BOX 15-2 *General Example of a Nonlinear Periodized Program Progression*

This program uses a 5-day rotation:
Monday
2 sets of 12-15 RM
Wednesday
6 sets of 1-3 RM
Friday
3 sets of 4-6 RM
Monday
Power day: 10 sets of 2-3 repetitions at 30% of 1 RM
Wednesday
4 sets of 8-10 RM

RM, repetition maximum.

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Flexibility

16

Ian Shrier

GENERAL PRINCIPLES

- The term *flexibility* is often used clinically as a synonym for range of motion (ROM) around a joint.
- Both muscles and ligaments can limit ROM.
- When ligaments limit ROM, it is referred to as a decrease in mobility; flexibility is usually reserved to refer to limited ROM caused by the muscle-tendon unit.
- Flexibility depends on both muscle stiffness (force required to stretch a muscle) and the stretch tolerance of the individual (the amount of discomfort a person feels when the muscle is stretched).

Definitions of Related Terms

Stretching: an activity in which a person purposefully attempts to increase ROM by applying a longitudinal force to the muscle.

Elastic effects: refers to an increase in tissue length that immediately returns to original length when stress is removed.

Viscous effects: refers to an increase in length that is dependent on time and returns to its original length slowly (i.e., is reversible). Viscous effects occur because molecules move when force is applied over time, and therefore return to original length is not immediate.

Visco-elastic effects: a combination of viscous and elastic effects.

Plastic effects: refers to a permanent change in the molecular structure of a tissue, as occurs when force is applied to a plastic sheet without completely tearing it. Plastic deformation means damage has occurred to a tissue—it does not occur with appropriate stretching (i.e., the ROM returns to normal within a reasonable time frame after stretching).

Flexibility training: program of stretching exercises designed to increase ROM of targeted joints to desired level, or to maintain that level once it is attained.

Specificity of Flexibility Training

- The immediate gain in ROM with stretching is mostly limited to the muscle being stretched, but there is some increase in ROM in the contralateral limb as well. This suggests that a neurologic reflex is one component of the mechanism for the effects of an acute stretch.
- If one stops moving a joint, one loses flexibility. How much movement is necessary to maintain flexibility is unknown, but it would seem logical that any muscle that is not moved through a particular ROM for a long period of time will lose that ROM.

Effects of Temperature on Flexibility Training

- The results of most studies suggest that the **effectiveness of stretching is increased when the tissue is warmer.**
- The most effective way to increase muscle temperature is with muscle activity, although deep heating methods (e.g., ultrasound) can be effective.
- Superficial heat is not an effective method to warm up the deep muscles.

Age and Sex Differences

- Flexibility decreases during the first year of life, and then begins to increase until approximately age 5 to 8 years. This is followed by a steady decline in flexibility in both boys and girls until age 12 to 14 years (near puberty). Flexibility then increases again until approximately age 20 to 24, after which there is a slow but steady decline. These changes appear to coincide with changes in levels of activity but it remains to be determined if the relationship is causal. For example, one begins to walk at about 1 year (flexibility begins to increase), and one starts school around age 5 to 8, which is associated with increased sitting (decreases in flexibility). Flexibility may increase again at age 12 to 14 as general activity increases with walking to secondary school and social relationships, but may decrease by age 20 to 24 as people enter the workforce and again become less active.
- Although females are generally more flexible than males, the difference is not great within the general population. The general perception that females are much more flexible may occur because females participate in activities and sports that include a large amount of flexibility training (e.g., dance, gymnastics). There are two further reasons why the effect may not be caused by hormones: (1) it is seen prepuberty and (2) the increase in flexibility that occurs around the time of adolescence occurs earlier in boys than in girls (but girls enter puberty earlier than boys).

Stretch Reflex

- The stretch reflex is a protective reflex mediated by muscle spindles. It causes the stretched muscle to contract and therefore prevent excessive ROM.
- The stretch reflex is the main reason some people advise against ballistic stretching (bounce stretching). As the stretch is released during the bounce, the muscle contracts and then the subsequent stretch occurs against an eccentrically contracting muscle. This theoretically increases the risk of injury, but there are no studies that actually compare rates of injury with ballistic stretching versus other types of stretching. An alternative view is that the force involved with appropriate ballistic stretching is very small and much less than what occurs during regular sport. Therefore, if an injury occurred during the stretch, some argue that it would likely have occurred during the sport as well.

ROLE OF FLEXIBILITY IN INJURY PREVENTION

Optimal Flexibility

- A graph of the relationship between flexibility and the risk of injury would be U-shaped. Both subjects who are inflexible and subjects who are very flexible have a higher risk of injury than subjects with an intermediate level of flexibility. The subjects with increased flexibility may be representative of a hypermobility group (because of their ligaments) rather than a hyperflexible group (because of their muscle-tendon unit) because these two features are sometimes difficult to differentiate.
- The U-shaped curve is based on cross-sectional data and does not mean that inflexible subjects have a reduced injury risk if they stretch before exercise. First, there may be some other associated factor with inflexibility that is responsible for the risk of injury and this is not affected by stretching. Second, the *immediate effects of stretching are opposite to the long-term effects of stretching* (see following text). The cross-sectional data on flexibility refers to long-term effects and therefore provides no information on the effect of stretching immediately before exercise.
- Stretching immediately before exercise: Immediately after an acute bout of weightlifting, the muscles are fatigued and

weaker. Similarly, but perhaps through a different mechanism, muscles are also weaker after stretching. This would not be expected to reduce injury risk and most studies have shown no change in injury risk when a stretching intervention is started.
- Regular stretching: If a person does weightlifting over weeks to months, the muscles are fatigued and weaker. Similarly, but perhaps through a different mechanism, muscles are also weaker after stretching. This is expected to reduce the injury risk. All three studies examining the effect of regular stretching on injury risk have shown a beneficial effect, but only one was statistically significant. Yoga's beneficial effects may work through this mechanism, although certain types of yoga also include strengthening exercises and balance exercises in addition to stretching exercise. The potential psychological benefits of yoga (and stretching in general) are other possible mechanisms.
- There remains a lot of work to be done in this area. For example, most studies have examined lower intensity activities such jogging. Whether the results can be generalized to higher intensity sports such as basketball remains to be determined.

ROLE OF FLEXIBILITY IN PERFORMANCE ENHANCEMENT

- As one must differentiate the effects of different kinds of stretching on injury risk, one must differentiate the effects of an acute stretch with the effects of regular stretching.
  - An acute stretch weakens the muscle and it cannot produce as much force or contract as rapidly.
  - If one stretches regularly over weeks, both force and velocity of contraction increase.
- Different sports have different requirements and performance does not depend solely on force and velocity of contraction.
  - In the running gait, energy is lost with each step. The energy lost is less when the gastrocnemius complex is stiff.
  - The performance of a ballerina depends much more on aesthetics than on the height of a jump. If stretching improves aesthetics, the performance is improved, even if the jump height is 2 cm lower. Also, if a hurdler cannot get the leg over a hurdle without stretching immediately before the race, then stretching will improve performance, even if "running speed" between the hurdles is less.

ROLE OF STRETCHING IN REHABILITATION FOLLOWING INJURY

- One of the dogmas of sport medicine is that ROM must be restored before strengthening begins. In fact, there is no evidence to support this belief for the vast majority of injuries in which weakness is a significant part of the pathology.
- There are no studies that look at the effectiveness of stretching versus a control group without stretching after an acute injury. Because stretching affects the type III and IV fibers that transmit pain, stretching should have an analgesic effect just as it does for other conditions. Further, if stretch-induced hypertrophy occurs as it does in healthy tissue, stretching should increase the strength of the tissue and improve healing.
- If the purpose of stretching were to increase strength, then it would be more logical to use a strengthening program. Studies are needed in this area for acute injuries. In chronic injuries, the results from the two studies that compare a strengthening program to a stretching program without strengthening suggest strengthening is much more important (see Recommended Readings):
  - For chronic groin pain (Holmich and colleagues), the strengthening group had 23 excellent results, versus 4 excellent results for the static stretching group.
  - For lateral epidondylitis (Svernlov and colleagues), the eccentric strengthening program had a 71% success rate at 1 year, compared to only 39% for the proprio-neurofacilitory (PNF) stretch group.

TECHNIQUES FOR IMPROVING FLEXIBILITY

- Stretching daily improves flexibility; the minimum frequency required remains to be determined.
- The optimal way to stretch is unknown and likely depends on the subject, the muscle (e.g., the angle of the muscle fibers or muscle pennation may influence the optimal duration for a stretch), and the baseline stiffness of the muscle. This hypothesis is supported by studies showing that the optimal duration of a stretch within an individual is different for different muscles, and different for different individuals.
- Because there is variability from one individual to another, one proposed method is to individualize the treatment. The subject begins by stretching the muscle to an acceptable length and holds the stretch. Once the sensation of force decreases, the stretch is increased so that the sensation felt during initial stages of the stretch is resumed. As long as there is an increased ROM over time, the stretching technique is effective. There are no studies that evaluate the relative effectiveness of this method.
- Some examples of types of stretching
  - **Static stretching:** The subject holds the stretch for a given period of time. It is important to feel the sensation of the stretch but not pain.
  - **Active stretching:** The subject performs the stretch by contracting the antagonist muscle. For example, a subject may contract the quadriceps to stretch the hamstring, with no other passive force being applied to the hamstring. Although this technique gained a lot of popularity several years ago, studies suggest it is no more effective at increasing ROM than static stretching.
  - **Proprio-neuro-facilitory (PNF) stretching:** One uses a combination of muscle contraction and passive force to improve the effectiveness of the stretch. Some forms of PNF contract the muscle being stretched, and some forms contract the antagonist muscle. PNF stretching is the most effective type of stretch. The original hypothesis that PNF stretching would decrease muscle activity/tone has been disproved in many studies and the effect is more likely an increase in stretch tolerance (because there is also an effect on the contralateral limb).
  - **Ballistic stretching:** The subject moves to the end ROM, then relaxes, and then returns to the end ROM using a bouncing technique. The amount of force used, and how much movement occurs during the relaxation phase (i.e., how close to the neutral position the subject returns to) vary according to different recommendations. Some authors advocate a full return to the neutral position and some authors advocate small oscillations at the end ROM. In general, traditional ballistic stretching is not superior to static stretching. The sport medicine community often considers ballistic stretching dangerous. There are theoretical reasons why it might be dangerous and theoretical reasons why it would not be more dangerous; there are no published studies on this topic.
  - **Dynamic stretching:** Different authors use this term differently. One common use refers to the subject who moves the joint through a regular ROM without applying force at the end of the motions (i.e., a form of ballistic stretching). Because muscle contractions are involved, and because muscle contractions increase the temperature of the muscle, it would not be surprising if this technique were superior when compared to a stretching technique that does not warm-up the muscles first. Because there are no studies comparing its effectiveness against other forms of stretching for injury risk or performance, all claims should be interpreted with great caution.

PERSONALIZING A STRETCHING PROGRAM

Objectives

- As with any intervention, one must clearly understand the objectives. Is the objective to reduce injury, improve tests of performance, improve performance, or something else? Do not confuse tests of performance (e.g., force achieved on a maximal voluntary contraction test) with performance (e.g., vault in gymnastics). Performance requires a much more sophisticated level of muscle coordination, often has an aesthetic value, and is greatly influenced by the athlete's psychological state of mind. The effect of a stretching program on performance is the combination of multiple factors.
- Different sports require different ROM for different joints. Any stretching program should target the specific muscles that require flexibility for the specific sport.
- Most sports require an increase in ROM with increasing velocity of contraction. For example, jogging requires more ROM than walking, and running requires more ROM than jogging.
- Because different muscles have different optimal stretching times, and different individuals have different optimal stretching times, it is important to evaluate the effectiveness of any program shortly after it is started. Regardless of the program, the subject should improve his or her ROM for the muscles being stretched. If this does not occur within 2 weeks (or sooner), the program should be modified. This may mean changing how long the stretch is held, how many times the stretch is applied in one session, how many sessions per day or week, and how much force is applied with each stretch. As a starting point, I ask patients to stretch the muscle so that they feel a slight pulling or tension. I ask them to hold this position until they no longer feel the stretch, and then to stretch the muscle a little further so that the sensation of tension is felt again. The stretch is held the second time for approximately the same amount of time as the first stretch. In general, most people will end up stretching each muscle for approximately a total of 20 to 40 seconds using this method. If patients want to stretch more than once per day, this is okay as well.
- Several stretch positions have been deemed "dangerous" by the sport medicine community.
- As with any exercise, injury will occur when the stress applied to a tissue exceeds the stress it can withstand.
- A stretch should not place stress on tissues that are not targeted for increased ROM. For example, the target muscle for the hurdler's stretch is the hamstring muscle when leaning forward, and the quadriceps muscle when leaning backward. However, there is also an increased stress to the medial aspect of the knee that appears unnecessary. This stretch should therefore be replaced using positions that do not stress the medial aspect of the knee, unless stressing the medial aspect of the knee is one of the specific objectives of the subject.

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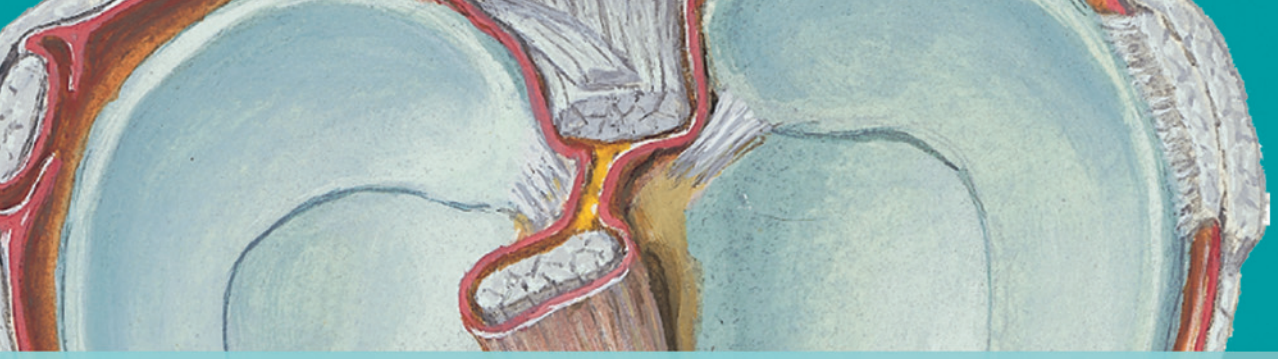

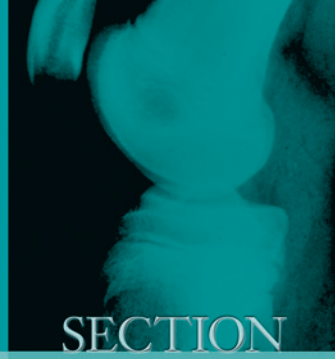

SECTION

IV

Environment

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Exercise in the Heat and Heat Illness

17

Jon Divine and Josh Takagishi

HEAT PRODUCTION

Exercise: The Body's Furnace

- **Thermodynamics Law #1:** energy can neither be created nor destroyed
- Contraction is directly related to and limited to muscle blood flow
- "Incoming" energy is transformed into:
  - Energy-rich chemical compounds: phosphates
  - Mechanical contraction work
  - Heat
  - Resting heat production

Energy Transformed into Heat

- Heat production occurs as a result of muscle work (Fig. 17-1).
  - Muscles that are producing heat are working at 15 to 20 times their rest rate.
  - Muscle contraction is an inefficient machine:
    - At maximum exercise, the human body's work efficiency is 15% to 30%.
    - 70% to 85% of muscle energy consumption is converted to heat and must be dissipated.
- Heat production during the exercise recovery phase:
  - Is exercise volume dependant.
  - Is a result of oxidative metabolism returning muscle back to resting state.
- Energy not used for muscle contraction or restoration of homeostasis must be dissipated or else body core temperature increases.

Energy Transformed into Resting Heat Production

- Resting heat production can be calculated as 21 J of heat per mL of oxygen (O_2).
  - Resting rate is usually 1.3 kJ of heat per minute for a 70-kg man.
- Contractions occurring "in the background" include involuntary smooth muscle, myocardium, joint stabilizing groups, and groups responsible for maintaining posture.
- Other potential sources for nonexercising heat production include digestion, shivering, and actions of hormones and catecholamines on cellular metabolism.

HEAT DISSIPATION AND HEAT TRANSFER

Principles of Heat Regulation

- Because energy is neither created nor lost, the body's **core temperature** is transient, but must be constantly regulated because of heat generated internally and heat gained from the environment.
- Without an "internal regulator," heat generated at rest would increase body temperature 1° C every 5 minutes.

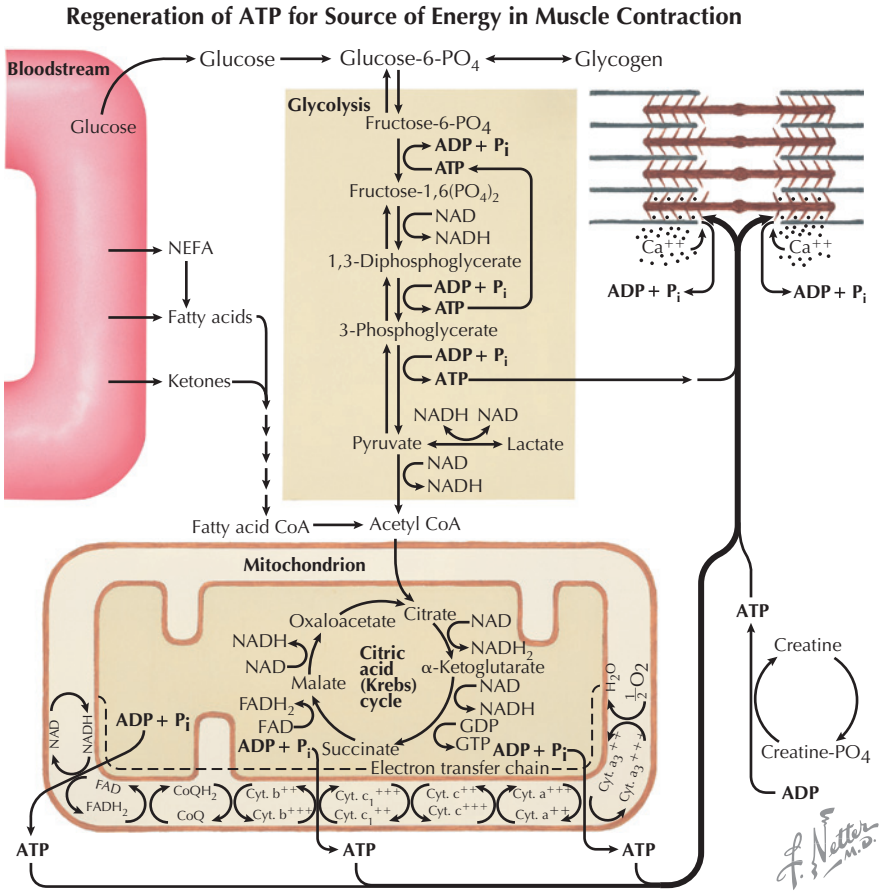

Figure 17-1 Regeneration of ATP for Source of Energy in Muscle Contraction.

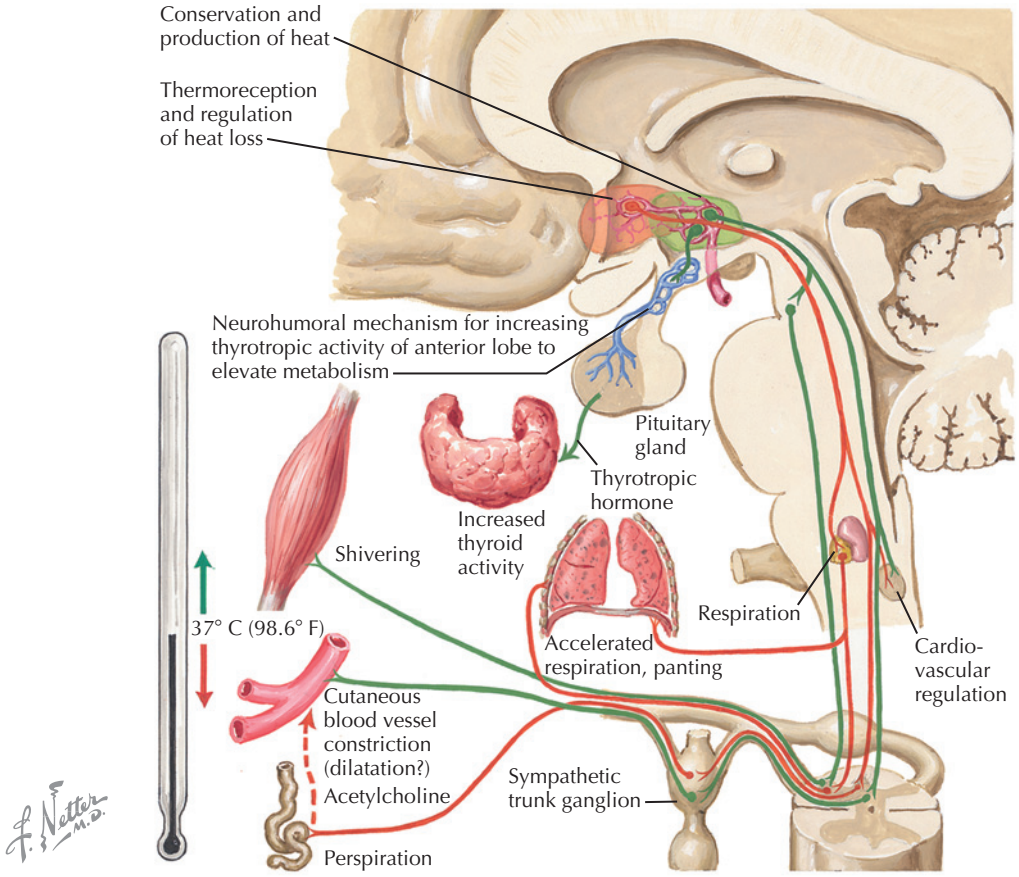

Figure 17-2 Temperature Regulation.

- Early in exercise,
  - Heat production is greater than heat loss, resulting in increased core temperature.
  - The rate of core temperature elevation increases even more in a hot environment.
- Rise in core temperature is sensed and centrally regulated primarily by **thermo detectors** in the hypothalamus, which then provide stimulus via the sympathetic nervous system to initiate sweating and increase skin blood flow (Fig. 17-2).
- Heat loss can be divided simply into:
  - **Nonevaporative (conduction, convection, and radiation) heat loss**
  - **Evaporative heat loss** (Fig. 17-3)

Nonevaporative Heat Dissipation

- **Radiation and convection** dissipate most heat when the ambient temperature is below 68° F (20° C).
- ***Evaporation accounts for most heat loss when temperature is above 68° F (20° C).***

Conductive Heat Loss

- A warmer body that is in direct contact with a colder body will result in heat transfer to the colder body.
- *Fourier's Law* states that the rate of conductive heat loss is directly dependant upon the heat transfer area, the thermal conductivity of the materials, and the temperature difference between the materials and indirectly related to the material thickness.

Figure 17-3 Methods of Heat Dissipation.

Convection Heat Loss

- Convection heat loss is heat transfer as a result of forced fluid flow (usually cooler) across a warmer, relatively stationary surface.
- *Newton's Law of Cooling* states that the rate of heat transferred is directly related to the convective **heat transfer coefficient** of the fluid and the temperature difference between the surface and fluid.
  - The heat transfer coefficient of water is 50 to 100 times greater than air.
- Blood has a high **heat transport coefficient** and can transport a relatively large amount of heat energy with only a moderate increase in blood temperature.
- Cutaneous blood flow also transfers heat by **convection** when dilated peripheral vessels come in contact with circulating air coming in direct contact with the skin surface.
  - Convective heat loss is directly related to:
    - The amount of body surface exposed to circulating air
    - The speed of air circulation
    - Inversely related to skin thickness between peripheral veins and skin surface

Radiation Heat Loss

When heat is lost via radiation, energy (heat) flows from high temperature to low temperature, resulting in heat transfer through electromagnetic waves.

Evaporative Heat Dissipation

- Evaporation occurs when a liquid changes into a gas.
- Heat is transferred by evaporation of sweat and respiratory moisture.
  - Insensate loss of moisture equals 600 grams daily.
- Sweating
  - Sweating usually begins when body temperature is above 98.6° F (37° C).
  - Amount varies by body surface area.
    - Rate depends on acclimatization and level of conditioning.
    - Sweat rates can vary between 600 mL and 3500 mL per hour.
  - **Heat of vaporization** of water governs the cooling effect of perspiration.
    - The normal rate of heat vaporization of water is 540 calories per gram but from the skin surface is 580 calories per gram.
- Cooling as a result of evaporation of perspiration is directly related to:
  - The sweat rate multiplied by 580 calories per gram (the heat of vaporization of water on skin).
  - The skin surface area.
  - The velocity of air crossing the skin surface area.
  - For example: A 70-kg athlete sweats 0.5 to 2.0 L/hour during intense exercise: heat loss by evaporation equals 300 to 1200 kcal of heat generated per hour.
- Problems with heat dissipation when environment is hot/humid:
  - Evaporative cooling is ***indirectly related to humidity.***
  - Evaporation can account for 98% of heat loss in hot, dry conditions.
  - In temperatures above 95° F (35° C), convection and radiation **do not** contribute to heat loss and sun **radiation causes heat gain.**
  - In heat-acclimated athletes, equilibrium between heat production and heat dissipation results in core temperatures during exercise between 98° F and 104° F without diminished performance.
  - If heat-dissipating mechanisms fail or if there is overwhelming heat stress, core temperature will continue to rise, even to dangerous levels.

Cardiac Ouput and Plasma Volume for Heat Dissipation

- **Specific demands for cardiac output and plasma volume are needed for heat dissipation during exercise in heat.**
- 15% of cardiac output is shunted to working muscles early in exercise.
- 15% to 25% of cardiac output is also shunted from central circulation to the skin for cooling, effectively lowering central plasma volume.
- Exercise decreases plasma volume, venous return, and stroke volume.
- An exercising sweat rate results in a 500 to 2000 mL loss of plasma volume per hour.
- To maintain necessary cardiac output required for maintaining exercising intensity (and core temperature), heart rate must increase.
- If plasma volume is not maintained and exercise is maintained at the same intensity in the same environmental conditions, then work and exercise performance are adversely affected, cooling efficiency decreases and could result in hypotension, and shock could result.
- Process can be changed favorably by reducing exercise intensity (slowing down), building plasma volume (drinking), and improving nonevaporative heat loss mechanisms (e.g., improve convection by increasing skin surface exposure or circulating local wind/water speed, reduce radiation exposure).

MINOR HEAT-RELATED ILLNESS

Heat Edema

Signs and symptoms: Edema of hands and feet.
Potential etiology: Marked peripheral vasodilatation and sweating, increased aldosterone production, sodium and water retention, increased plasma volume. May be caused by "gravitational" or centripetal force of arm swing.
Predisposing factors: Unacclimated persons who exercise in hot environments.
Treatment: Rest and/or elevation of the affected extremity. Generally resolves with acclimation.
Complications: None.

Heat "Cramps"

Signs and symptoms: Severe, spreading muscular tightening and spasm seen during or after intense, prolonged exercise in the heat. The large, lower limb muscles are most commonly affected, but any muscle may be affected, including the abdominals and intercostals.
Etiology: Defined as "salt loss, fluid loss, and muscle fatigue."
Predisposing factors: Lack of acclimation and or conditioning, ongoing negative sodium balance ("salty sweaters"); presence of sickle cell trait has also been proposed as a risk factor for frequent exercise-associated heat cramps.
Treatment (Box 17-1)**:**

- Rest and cooling down.
- Massage of affected muscles with ice.
- Oral rehydration with electrolyte solution.
- Oral intake of salty foods.
- Intravenous bolus of normal saline if symptoms not improving over 30 to 45 minutes with oral rehydration or if oral rehydration is not feasible. Recommend checking blood sodium levels prior to administering intravenous (IV) fluid.
- Medications have been used in severe cases, especially when heat cramps accompany heat stroke and a family or individual history of malignant hyperthermia is suspected.
  - Diazepam, 1 to 5 mg IV
  - Midazolam, 1 to 2 mg IV

BOX 17-1 *Suggested Equipment and Supplies for Treatment of Heat-Related Illness*

Stretchers
Cots
Wheelchairs
Bath towels
High-temperature rectal thermometers (43° C, 110° F)
Disposable latex-free gloves
Stethoscopes
Blood pressure cuffs
Intravenous (IV) tubing and cannulation needles
D5% NS and NS IV fluids in 1-L bags
3% saline IV fluid in 250-mL bags
Sharps and biohazard disposal containers
Alcohol wipes, tape, and gauze pads
Tables for medical supplies
Water supply for tubs or ice water buckets
Tub for immersion therapy
Fans for cooling
Oxygen tanks with regulators and masks
Ice, crushed or cubed
Plastic bags
Oral rehydration fluids
Cups for oral fluids
Glucose blood monitoring kits
Sodium analyzer and chemistry chips
Diazepam IV 5 mg or midazolam IV 1 mg vials
Defibrillator (automatic or manual)

Adapted from Adner MM, Scarlet JJ, Robinson W, Jones BH: The Boston Marathon medical care team: Ten years of experience. Phys Sportsmed 16:99-106, 1988, and Noble HB, Bachman, D: Medical aspects of distance race planning. Phys Sportsmed 7:78-84, 1979.

  - Magnesium sulfate
  - Dantrolene (if malignant hyperthermia suspected)
- **Prevention:** Conditioning and heat acclimatization. Recurrent crampers, particularly "salty sweaters," may benefit from liberal use of salt within their diet, especially in hotter climates.
- **Complications:** Rare, but may be a warning sign of impending heat exhaustion. Rhabomyolisis should be suspected in severe, prolonged episodes of muscle cramping of multiple muscle groups.

Heat Syncope/Exercise Associated Collapse (EAC)

Signs and symptoms: Syncope (fainting) or lightheadedness usually seen at the end of an endurance event, usually running.
Etiology: Orthostatic "syncopal" event in which an athlete is peripherally vasodilated during activity. When activity is stopped, much of the blood volume "pools" in the lower limbs, leading to decreased venous return and subsequently to decreased cardiac output and blood flow to the brain.
Predisposing factors: Ending exercise suddenly without lower intensity, "leg-pumping" cool-down, dehydration, lack of acclimation, elderly age.
Evaluation/Treatment:
- Assess ABCs.
- Obtain initial vital signs.
- Lie down and elevate legs (Trendelenburg position).
- Rest in cool, shaded place with air movement.
- Orally rehydrate whenever possible.
- Assess for other injuries or other causes of syncope (e.g., cardiac, pulmonary, neurologic, or metabolic).
- Symptoms should resolve with treatment in less than 30 minutes.
- Follow vital signs closely.

Complications: Rare, but may be a warning sign of impending heat exhaustion or another more concerning cause of syncope.

MODERATE HEAT-RELATED ILLNESS

Heat Exhaustion/Exertional Hyperthermia

Overview: Most common heat-related illness.
Signs:
- Elevated rectal temperature (above 104° F or 40.5° C).
- Decreased cardiac output, more exaggerated if also fluid depleted (tachycardia, orthostatic hypotension, tachypnea, syncope).
- Exaggerated peripheral cooling (profuse sweating, cutaneous flushing).
- Mild mental status changes (mild confusion, mild agitation/irritability, mild emotional liability, mild uncoordination). **If mental status signs are worse than mild, then proceed to heat stroke evaluation and management.**

Symptoms: Fatigue (inability to continue exercise), headaches, nausea, vomiting, heat cramps, chills (particularly of head and neck), and "goose bumps." ***If also dehydrated: thirst may be present but is not a reliable indicator of hydration status.***
Etiology: Failure of the cardiovascular system responses to workload, usually a combination of exertional heat stress and dehydration, which results in the body's inability to adequately dissipate heat. Pure sodium depletion or water depletion forms of heat exhaustion are rare in athletes; most athletes have mixture of both syndromes.
Evaluation/Treatment:
- Assess ABCs.
- Obtain initial vital signs.
- Rest in cool, shaded environment with air circulation. If more severe symptoms are present, then treat with rapid cooling techniques such as ice baths.
- Elevation of lower limbs (Trendelenburg position).
- Oral rehydration with an electrolyte-containing solution.
- If vomiting, diarrhea, or decreased mental status is present, then intravenous fluids may be necessary.
- Recommend checking blood sodium levels before administering IV fluids.
  - 5% dextrose in 0.9% normal saline (D_5NS) is most often used.
  - Start with administering 1 L over 30 to 60 minutes. Individual fluid replacement may vary between 600 to 4000 mL. Watch symptoms closely during IV rehydration.
- Follow temperature and vital signs closely.
- Symptoms usually resolve within 2 to 3 hours. Slower recovery should initiate transfer to a medical facility for further evaluation.

Complications: No long-term sequelae of heat exhaustion have been reported. Anecdotal evidence suggests that one episode of significant heat illness increases the risk of future episodes of heat illness or complications.

SEVERE HEAT-RELATED ILLNESS

Exertional Heat Stroke (EHS)

Overview: A true medical emergency.
Definition: Usually characterized by extreme hyperthermia (rectal temp above 104° F or 40.5° C) with thermoregulatory failure and profound central nervous system dysfunction (differentiates EHS from heat exhaustion).
Signs:
- Rectal temperature above 104° F or 40.5° C (may be as high as 107° F to 108° F).
- Significantly impaired cardiac output (hypotension, tachycardia, tachypnea).

- Pronounced mental status changes (irritability, ataxia, confusion, disorientation, syncope, hysterical or psychotic behavior, seizure, and/or coma).
- Diminished peripheral cooling ability (cessation of sweating, hot skin).
- Signs of life-threatening disseminated intravascular coagulation (DIC), which include epistaxis, bleeding from IV sites, bruising, and pulmonary edema; signs of acute renal failure (ARF), including peripheral edema, may be seen in the emergency department and hopefully not "in the field."

Symptoms: Similar to exertional hyperthermia: fatigue, dizziness, nausea, vomiting, heat cramps, and chills.

Etiology: ***Total thermoregulatory failure that will not spontaneously reverse itself.*** The greatest risk of EHS exists when the wet bulb globe temperature (WBGT) is above 82° F (28° C), during higher intensity exercise (more than 75% of $\dot{V}O_2$ max), and when strenuous exercise lasts more than 1 hour (Table 17-1).

On site WBGT index is calculated with the formula:

WBGT = 0.7 (WB) + 0.2 (BB)+ 0.1 (DB)

WB = wet bulb temperature: humidity indicator

BB = black bulb temperature: provides measure of radiant heat gained.

DB = dry bulb temperature: normal air temperature

Pathophysiology of EHS: There are three distinct mechanisms of injury in which heat stress causes damage to the body:

- Damage to cells via denaturation of proteins and thus interruption of cellular function.
- Release of inflammatory cytokines and other heat shock proteins, which contributes to early circulatory collapse and systemic damage.
- Damage to vascular endothelium, which subsequently activates the coagulation cascade.

Predisposing Factors:

- Genetic predisposition possible.
- Dehydration (acute and chronic).
- Lack of acclimation to heat (often seen on exceptionally hot spring day).
- Negative sodium balance (over time).
- Frequently occurs near race finish line, when an already dehydrated athlete increases speed, causing increased muscle heat production, increased muscle blood flow, secondarily decreased skin blood flow (similar to EAC mechanism), and rise in core temperature.
- Approximately 25% of patients have preexisting gastrointestinal (GI) or respiratory illness, and many more have had warning signs of impending illness.

Evaluation/Treatment:

- Assess ABCs.
- Remove from the hot environment to cool, shaded area with airflow.
- Remove all necessary clothing.
- Obtain vital signs and core temperature.
  - Determine core temperature with rectal thermometer before rapid cooling is commenced.
  - ***Methods of measuring temperature other than true-core (rectal) temperature should not guide diagnosis and therapy.***
  - Oral, tympanic membrane, aural canal, and axillary temperatures do not correlate well with core temperature in heat-injured patients.
  - Peripheral temperatures may be up to 1° C lower than core temperatures.
  - Rapid cooling measures and/or delayed core temperature readings may lead to considerably lower temperature readings.
  - According to the American College of Sports Medicine 2007 Position Statement, ***"Cold water immersion provides the fastest whole body cooling rate and the lowest morbidity and mortality for EHS."*** *(Recommendation based on consistent and good-quality patient- or subject-oriented evidence.)*
- Immediate external cooling (via one of the three following methods) in patients with rectal temperature above 104° F.
  - Wet patient down with tepid or cool spray (or single layer of wet cheesecloth or thin sheeting) and use large fan to speed evaporation.
  - Immerse in ice water bath. Research suggests this is the best modality for rapid cooling.

Table 17-1 WBGT LEVELS FOR MODIFICATION OR CANCELLATION OF WORKOUTS OR ATHLETIC COMPETITION FOR HEALTHY ADULTS

WBGT			Training & noncontinuous activity	
Degree F	**Degree C**	**Continuous activity & competition**	**Nonacclimatized, unfit, high-risk individuals**	**Acclimatized, fit, low-risk individuals**
<50.0	<10.0	Generally safe; EHS can occur; associated with individual factors.	Normal activity.	Normal activity.
50.1-65.0	10.1-18.3	Generally safe; EHS can occur.		
65.1-72.0	18.4-22.2	Risk of EHS and other heat illness begins to rise; high-risk individuals should be monitored or not compete.	Increase rest:work ratio. Monitor fluid intake.	Normal activity. Monitor fluid intake.
72.1-78.0	22.3-25.6	Risk for all competitors is increased.	Increase rest:work ratio and decrease total duration of activity.	Normal activity. Monitor fluid intake.
78.1-82.0	25.7-27.8	Risk for unfit, nonacclimatized individuals is high.	Increase rest:work ratio; decrease intensity and total duration of activity.	Normal activity. Monitor fluid intake.
82.1-86.0	27.9-30.0	**Cancel exercise.**	Increase rest:work ratio to 1:1, decrease intensity and total duration of activity. Limit intense exercise. Watch at-risk individuals carefully.	Plan intense exercise with discretion. Watch at-risk individuals carefully.
86.1-90.0	30.1-32.2		**Cancel exercise.**	Limit intense exercise and total daily exposure to heat and humidity; watch for early signs and symptoms.
>90.1	>32.3			**Cancel exercise.**

WBGT, wet bulb globe temperature.

- Place intravenous line and measure serum sodium.
  - May be difficult because of peripheral vasoconstriction.
  - *Do not delay* efforts to cool down the athlete while waiting to place an IV.
- *Keep in mind:* Cooling techniques cause peripheral vasoconstriction, which in turn increases venous return and cardiac output.
- If patient remains hypotensive after cooling, give 250- to 500-mL boluses of normal saline by rapid infusion and monitor blood pressure.
- Medications:
  - Vasopressors: Occasionally, judicious use of pressor agents may be necessary in order to maintain blood pressure above 90 mm Hg.
  - Dantrolene may be helpful in patients with known malignant hyperthermia and patients on neuroleptic agents with exertional heat stroke.
- Monitor rectal temperatures closely (every 5 to 10 minutes).
  - External cooling may be discontinued when the core temperature reaches 102° F so as to avoid hypothermia.
- Follow all vitals signs closely (every 5 to 10 minutes).
- Transfer to a medical facility may be necessary for airway management, careful fluid and electrolyte administration, circulatory support, and cardiac, hemodynamic, and laboratory monitoring.

Complications: Extend to all major organ systems (Box 17-2):

- Central nervous system injury—may be permanent in up to 20% of cases.
- Rhabdomyolosis—results in myoglobinuria, which may lead to renal damage.
- Acute renal failure—25% of patients develop.
- Disseminated intravascular coagulation—may lead to a hemorrhagic diathesis.
- Liver damage.
- Myocardial injury—may lead to arrhythmias, myocardial infarction, and cardiac arrest.
- Pulmonary injury—may lead to pulmonary edema, pulmonary infarction, or acute respiratory distress syndrome (ARDS).
- Metabolic/Lab abnormalities—hypokalemia (early), hyperkalemia (late), hypernatremia (dehydration), hyponatremia, hypocalcemia, hyperphosphatemia, hypoglycemia, lactic acidosis, uremia, elevated CPK, elevated liver function tests, elevated WBC, thrombocytopenia, elevated coagulation studies, myoglobinuria, anemia, hemoconcentration.

Morbidity and mortality: Permanent damage and mortality rates vary directly with time elapsed between core temperature elevation and initiation of cooling therapy. Mortality can approach 10%.

Return to play: Following an episode of EHS an athlete may temporarily lack heat tolerance and possess residual thermoregulatory compromise that may last up to several months. A graduated return-to-play plan should be followed (Box 17-3).

BOX 17-2 *Severe Complications of Heatstroke*

Cardiovascular
Arrhythmias
Myocardial infarction
Pulmonary edema
Shock
Central Nervous System
Confusion
Coma
Seizures
Cerebral or spinal infarction
Gastrointestinal
Diarrhea and vomiting
Hepatocellular necrosis
Upper gastrointestinal bleeding
Hematologic
Fibrinolysis
Thrombocytopenia
Disseminated intravascular coagulation
Musculoskeletal
Rhabdomyolysis
Myoglobinemia
Pulmonary
Hyperventilation
Respiratory alkalosis
Adult respiratory distress syndrome
Pulmonary infarction
Renal
Acute renal failure

From Mellion MB, Shelton GL: Thermoregulation, heat illness, and safe exercise in the heat. In Mellion MB (ed): Office Sports Medicine, 2nd ed. Philadelphia: Hanley & Belfus, 1996, pp 45-57.

RISK FACTORS AND POPULATIONS AT INCREASED RISK FOR HEAT-RELATED ILLNESS

- **Healthy adults**
  - Poorly acclimated to heat or humidity.
  - Poorly conditioned.
  - Inexperienced in competition (limited judgment about heat risk).
  - Salt or water depleted.
- **Large or obese adults**
  - Generate more heat for the same level of activity; adipose tissue has lower specific heat than lean tissue.
  - Dissipate heat less efficiently. People who are obese have a lower body surface-to-mass ratio, have fewer heat-activated sweat glands in the skin overlying adipose tissue, and are less likely to be conditioned or are at a lower fitness level.
- **Children**
  - Produce more metabolic heat per mass unit than adults. Although the high surface area-to-body mass ratio is typically advantageous in this population, when the sun is hot or ambient temperature is high, children absorb relatively more heat from the environment.
  - Sweat less, require greater core temperature increases to trigger sweating, acclimatize more slowly, cardiac output at given metabolic rate is lower; they may lack adequate blood flow for both muscle and cooling needs. Transition to adult thermoregulation begins after puberty.
- **Elderly individuals**
  - Generally are less efficient at cooling than younger adults because of multiple mechanisms associated with the aging process.

BOX 17-3 *Suggested Guidelines for Return to Play Following Exertional Heat Stroke (EHS)*

Clearance by a physician and at least 7 days of *asymptomatic* rest following release from medical care.

Follow-up for repeat medical evaluation including testing for laboratory or imaging tests of affected organs.

Once cleared, begin exercise in a cool environment and gradually increase the intensity, duration, and level of heat exposure over a 2-week period.

If return to pre-EHS activity levels is difficult, consider a laboratory-based exercise-heat tolerance test 1 month after EHS event.

The athlete may be cleared for full competition if heat tolerance exists after 2 to 4 weeks of training.

- Age-related limitation to full heat acclimation is a result of reduced vasodilator response, which may begin as early as age 50.
- Decreased maximum heart rate with age leads to decreased maximum cardiac output.
- Reduced thirst response after water deprivation, which results in underhydration.
- Frequently have reduced fitness level.

- **History of previous heat injury:** Unknown mechanism: speculation that the central nervous system–controlled cooling mechanism has been irreversibly "injured," resulting in a higher "set point" to begin sweating.
- **Women of reproductive age during the postovulatory phase of menstrual cycle:** Heat dissipation may be reduced because of an increased temperature "set point" to begin sweating or possibly a smaller plasma volume.
- **Acute illnesses:** Increase metabolic demand for blood flow throughout the body. Gastrointestinal illnesses specifically:
  - Increase risk because increased blood flow to GI tract competes with skin blood flow for cardiac output.
  - Are often associated with dehydration and electrolyte disturbances.
- **Chronic illnesses associated with heat illness:** Cystic fibrosis, diabetes (uncontrolled), history of malignant hyperthermia.
- **Presence of sickle-cell trait (SCT):** See Chapter 26, Hematologic Problems in Athletes.
- **Alcohol and other substance abuse**
  - Alcohol has a residual effect on thermoregulation capacity and makes the athlete predisposed to dehydration and reduced cooling efficiency.
  - Acute stimulant intoxication (cocaine, methamphetamine) may be difficult to differentiate from heat stroke because of increased metabolic activity.
- **Use of certain medications:** Anticholinergic agents, antihistamines, beta-blockers, diuretics, tricyclic antidepressants and monoamine oxidase inhibitors, stimulant agents for attention-deficit disorder put people at higher risk for heat-related illness.

EXERCISE-ASSOCIATED HYPONATREMIA (EAH)

Definition: Hyponatremia (serum sodium ≤ 135 mEq/L) observed in athletes is usually associated with endurance/ultra-endurance exercise.

Incidence: Seen in 5% to 13% of marathon participants and 0.3% to 27% of ultra-endurance participants, most of whom are minimally symptomatic or asymptomatic.

Presenting postevent symptoms: Complaints of "not feeling right," nausea, lightheadedness, malaise, lethargy, cramps. Vomiting is a very common, postmarathon symptom of EAH.

Signs of fluid overload: Edema (e.g., ring or wrist band fitting more tightly), weight gain, emesis. **More ominous signs of fluid overload are indicative of early pulmonary and cerebral edema and include tachypnea, tachycardia, mental status change (confusion, seizure, coma, and death).**

Etiology: *Controversial at present*. Thought to be caused by a combination of excessive intake of hypotonic fluids, loss of sodium in sweat, and inappropriate exercise-elevation of ADH (vasopressin).

Predisposing factors:

- Endurance activity lasting more than 4 hours
- Body mass index less than 20
- Weight gain during endurance event. Runners who *fail to lose 0.75 kg of body weight* during a marathon are ***seven times*** more likely to develop hyponatremia than those who lose more then 0.75 kg.
- Female sex (likely secondary to lower BMI). Women in the postluteal phase of their menstrual cycle have also been found to have an increased risk of EAH, because of the influence of progesterone on fluid retention and increased sensitivity to ADH during this phase.
- Inexperience with endurance events.
- Use of nonsteroidal anti-inflammatory medications (NSAIDs). Cause is controversial at present; may result from decreased glomerular filtration rate.
- Prolonged activity in a hot and humid environment.

Evaluation/Treatment (Houston Marathon Protocol):

- **Level 1 prerace assessment:**
  - Prerace education.
  - Stress importance of measuring prerace and postrace weights.
  - Prerace questionnaire to self-assess risk of hyponatremia.
  - List of postrace symptoms associated with hyponatremia.
  - Encourage salty food and fluid intake in a cool environment.
  - Dilute urine production offers a good prognosis.
- **Level 2 assessment (serum sodium below 135 with few or no symptoms):**
  - Participant presents with weight gain or postevent symptoms of hyponatremia and no mental status changes.
  - Obtain initial vital signs including temperature.
  - Obtain weight (compare to prerace weight if one was obtained).
  - Obtain serum electrolytes and glucose.
  - Observe closely while encouraging oral fluid intake.
  - IV fluids are typically not needed unless oral fluids are not tolerated—consider 3% NS (hypertonic saline) if mental status is deteriorating.
  - Production of dilute urine indicates progress toward normal sodium levels.
- **Level 3 assessment (serum sodium below 135 with significant mental status changes—seizures, coma, or increasing delirium):**
  - Usually not seen unless sodium is below 128.
  - Rapid deterioration of respiratory status usually follows worsening mental status.
  - Establish and monitor airway. Be prepared for intubation. Aggressive measures should be taken immediately.
  - Establish IV access and use hypertonic saline: **3% NS, 100 mL over 10 minutes, and follow with 1 mL/min/kg, up to 70 mL/hr, until symptoms improve.**
  - Arrange for immediate transport to a medical facility.
  - In a critical care setting with central pressure monitoring, loop diuretics and mannitol may be needed to reduce pulmonary edema and cerebral edema.

Prevention of EAH: Educate athletes not to drink more than their sweat rate (usually 600 mL to 1200 mL per hour). Athletes can be taught the following method to predetermine their own sweat rate (see **"calculate sweat loss under similar conditions"** in "Prevention of Heat Illness" section). Hypotonic carbohydrate (6% to 8%) fluids may be more beneficial for prevention of EAH than water. Event organizers can lessen distance between drinking stations for endurance events.

PREVENTION OF HEAT-RELATED ILLNESS

Provide an Adequate Preparticipation Medical History and Evaluation

Identify athletes at risk for sustaining heat-related illness.

Ensure Adequate Acclimation and Conditioning

- Physiologic effects of acclimation to exercise in heat require 7 to 10 days of exposure and include:
  - Improved cooling efficiency from sweating: earlier initiation of sweating, increased rate and amount of sweating,

increased maximum sweating capacity, lower sweat sodium concentration.
- Increased cardiovascular efficiency: increased basal plasma volume, decreased heart rate at given workload and heat stress.
- Thermal effects: increased exercise capacity in heat, lower core and skin temperature at given workload and heat stress, reduced perceived intensity of exercise, increased subjective thermal comfort.
- Approximately 75% of effect occurs in first 4 to 6 days.
- 90 to 100 minutes daily of continuous aerobic exercise may be optimal.
- Acclimation effect persists for 1 to 4 weeks.
- Individual variation is significant, and effect may persist longer for dry heat than humid heat.

General Points for Improving Heat Acclimation

- Preseason conditioning should include strength, endurance, and skills acquisition drills in a warm environment.
- Delay full participation until minimum conditioning levels are met.
- Heat acclimation generally requires 10 to 14 days
- Avoid supramaximal efforts, such as performance testing, in the heat—especially involving those with sickle cell trait.
- The "ancient" practice of fluid restriction to improve heat adaptability or conditioning is extremely dangerous and should never be done.
- Provide education regarding heat-related illness to all athletes. This should include:
  - Providing awareness and counseling to at-risk populations and their coaches regarding the signs and symptoms of early heat stress, the dangers and sequelae of heat-related illness, and strategies to minimize the risk of developing these illnesses.
  - To all athletes and coaches emphasize the importance of staying properly hydrated, obtaining adequate sleep, avoiding drugs and alcohol, knowing each athlete's individual limitations, and avoiding exercise in dangerous environmental conditions.

Monitor Atmospheric and Environmental Conditions and Enforce Activity Restriction in Dangerous Situations

- The American College of Sports Medicine (ACSM) and several high school athletic federations have guidelines on when to restrict activity during periods of high temperatures.
- **Monitoring of weather reports**
  - Simple and convenient sources for approximate temperature and humidity readings such as frequently updated Internet sites are helpful to calculate heat stress but may not allow for local variation of sun exposure, wind velocity, or local airflow.
  - The use of color-coded warning flags along the course of an endurance event is endorsed by ACSM and used by many endurance races to alert runners/participants about the relative risks of continuing based on relative heat stress (Fig. 17-4).

Adjust Workout Schedule Based on Environmental Conditions

- Reschedule workouts, practices, and competitions to a cooler time of day or cancel altogether.
- Alter practices and workouts by (Table 17-2):
  - Decreasing their intensity
  - Making them of shorter duration
  - Providing more frequent breaks
  - Modifying practice clothing to increase evaporative cooling
  - Moving to a more shaded or breezy area

Air temperatures (°F)

Relative humidity (%)	70	75	80	85	90	95	100	105	110	115	120
0%	64	69	73	78	83	87	91	95	99	103	107
10%	65	70	75	80	85	90	95	100	105	111	116
20%	66	72	77	82	87	93	99	105	112	120	130
30%	67	73	78	84	90	96	104	113	123	135	148
40%	68	74	79	86	93	101	110	123	137	151	
50%	69	75	81	88	96	107	120	135	150		
60%	70	76	82	90	100	114	132	149			
70%	70	77	85	93	106	124	144				
80%	71	78	86	97	113	136					
90%	71	79	88	102	122						
100%	72	80	91	108							

Figure 17-4 Heat Stress (Apparent Temperatures in °F): Calculation of Heat Stress and Heat Stress Risk. (Adapted from National Weather Service: Heat Wave and U.S. Department of Commerce, National Oceanic and Atmospheric Administration, PA 85001, 1985. Color code adapted from ACSM Position Statement [1975] on prevention of heat injuries during distance running.)

Proper Clothing for Exercise in the Heat

- Short-sleeved, loose fitting, open-weave or mesh jerseys allow better evaporation.
  - Evidence to support or discourage the wearing of newer, sweat-"wicking" shirts (made of fabric that absorbs sweat away from the skin to the outer surface to promote evaporative cooling) to reduce the risk of heat illness is lacking.
  - Wearing no shirt involves both benefit (better heat loss from evaporation and convection) and risk (more radiant heat gain).
- Athletes should practice or play in shorts when possible.
- Light-colored uniforms reflect sunlight, reducing "black box" radiation absorption.
- As they become sweat-soaked, uniforms or clothing should be changed to allow for efficient evaporation.
- Avoid restrictive clothing and bulky protective equipment such as poorly aerated helmets; heavy, long-sleeved uniforms; protective pads; and rubberized workout suits, which block skin surface area and reduce cooling by radiation, convection, and evaporation.
- Keep in mind that the protective benefits of oil- or gel-based sunscreens may block evaporative cooling by also blocking harmful ultraviolet (UV) rays.
  - Using a water-based sunscreen, applied frequently, is the ideal means to provide adequate sun protection while not adversely affecting heat dissipation.

Monitor Athletes Closely

Observe athletes at increased risk for heat illness.

Prevent Athletes with Fevers, Acute Illnesses, and at High Risk of Heat Illness from Participating

- Core temperature elevations caused by fever and illness are additive to those caused by exercise. Therefore, both cardiac output and aerobic capacity are reduced in febrile athletes. *Exercise with fever may be dangerous, especially in heat.*

Table 17-2 MODIFYING PRACTICE SESSIONS FOR EXERCISING CHILDREN

Rights were not granted to include this table in electronic media. Please refer to the printed publication.

- Aspirin, NSAIDs, acetaminophen, or other antipyretics cannot reduce core temperature elevations caused by exercise.
- When in doubt about participation, apply the "neck check" rule: *No participation if symptoms are present below the neck or if fever is present.*

Prevent Dehydration

*"**Dehydration reduces endurance exercise performance, decreases time to exhaustion, increases heat storage**."—recommendation based on consistent and good-quality patient- or subject-oriented evidence—ACSM 2007.*

- In general, when fluid deficits exceed:
  - 2%, work capacity decreases by 15% to 20% and thermoregulatory function is impaired.
  - More than 3% to 5% of body weight, sweat production and blood flow to the skin begin to decrease.
  - More than 6% to 10% of body weight, cardiac output significantly decreases, resulting in lower sweat production and reduced blood flow to both skin and working (heat-producing) muscle.
- Dehydration *plus* heat stress of exercise causes:
  - Greater reduction in central volume, venous return, and, consequently, stroke volume and cardiac output than *either factor alone.*
  - Performance reductions proportional to level of dehydration and intensity of exercise heat stress.
  - Reduced heat dissipation resulting from decreased skin flow and diminished sweating.
- Monitoring body weight helps assess dehydration.
  - Acute weight loss means dehydration has occurred.
  - *Caution is indicated when an athlete has a workout weight loss greater than 3% or fails to regain the previous day's weight loss by the time of the next day's workout.* During summer workouts, dehydration can be cumulative over several days, such as during two-a-day football workouts.
  - Athletes with large or persistent acute weight loss should be restricted from activity until rehydrated.
- For prolonged or repetitive endurance exercise in the heat, carbohydrate-electrolyte solutions (6% to 8% carbohydrate) may retard dehydration, speed rehydration, and have been reported to maintain performance.
- **Thirst is not an adequate guide for fluid consumption in humans:** calculation of individual fluid replacement rates should be encouraged for all athletes.
  - Athletes *may not become thirsty* until they have become more than 5% dehydrated.
  - Inexperienced athletes may automatically associate heat illness symptoms with being "dehydrated" and drink excessively, resulting in overhydration.
  - For optimal individual hydration, athletes should drink at regular intervals (every 15 to 20 minutes) and replace what they sweat. Fluid loss from sweat usually occurs at the rate of 600 mL to 1200 mL per hour, but can be higher or lower in some athletes and will vary based on weather conditions and exercise intensity.
- **Calculating sweat loss under similar conditions is the ideal method for determining fluid replacement.**
- The most accurate way to easily determine fluid needed is to **calculate sweat loss under similar conditions:**
  - Weigh nude before the activity.
  - Perform activity at competition level for one hour. (One hour is recommended to get a reliable representation of sweat rate expected in an endurance event.)
  - Track fluid intake (in ounces) during the activity.
  - Record nude weight after activity. Subtract from starting weight. Convert the difference in body weight to ounces.
  - To determine hourly sweat rate, add the difference in body weight (in ounces) to the volume of fluid consumed.

- To determine how much to drink every 15 minutes, divide the hourly sweat rate by 4. This becomes the guideline for fluid intake every 15 minutes of the activity.
- Note the environmental conditions on this day and repeat the measurements on another day when the environmental conditions are different. This will give you an idea of how different conditions affect your sweat rate.

RECOMMENDED READINGS

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Exercise in the Cold and Cold Injuries

18

Christopher C. Madden

GENERAL PRINCIPALS

Physiology of Cold Exposure

Mechanisms of Heat Loss

RADIATION

- Radiation involves direct emission or absorption of heat energy from the body (mostly infrared radiation).
- Radiation is the largest source of heat loss from the body.
- Clothed, sedentary individuals in a calm, temperate climate lose more body heat (≈60%) by radiation than active individuals (≈45%) in a thermoneutral environment where heat production equals heat loss.
- The human body constantly radiates heat to nearby solid objects with cooler temperature, and rate of heat loss increases with the difference in temperature between body and object.
- Radiant heat loss increases as temperature drops, but only becomes a significant problem in extremely cold environments.
- Clothing does not affect radiant heat loss significantly.

EVAPORATION

- Evaporation occurs when water or moisture is transformed into vapor.
- Evaporation occurs with perspiration on the body's surface, and in the respiratory passages as inspired air is warmed and moistened.
- High altitude increases heat and water losses from the lungs because breathing deepens and respiratory rate increases.
- A small amount of "insensible" perspiration occurs at cold temperatures, but may become significant in active individuals who are "layered" improperly.
- Vigorous exercise with increased sweating leads to greater evaporative heat loss than with normal evaporative loss in a sedentary individual in a temperate climate (a difference of approximately 20% to 30%).
- Vapor barrier systems meant to decrease evaporative water loss are for the most part ineffective in physically active individuals because they trap perspiration between the barrier and the skin and lead to increased heat loss by convection when the sweat rate exceeds permeability capability (Fig. 18-1).
- Wet clothing combined with wind leads to significant evaporative heat losses.

CONVECTION

- Convection involves the transfer of heat from the body to cold air or water in contact with the body surface.
- Ongoing heat loss occurs when warmed air or water is continually displaced from the body surface and is replaced by air and water of colder temperatures.
- The amount of heat loss that occurs by convection is determined by the temperature difference between the air and the body surface, and by the speed of air moving over the body.
- Convective heat loss increases greatly with **wind** moving over the body surface and especially with submersion in cold water.
- In addition to **natural wind, wind moving over the body surface** created by cycling, skiing, running, windsurfing, or other activities that increase air flow over the body can cause significant heat loss, especially at cold temperatures sustained over prolonged periods.
- **"Wind chill"** is wind combined with cold that causes a lower "equivalent" temperature (Table 18-1) than current environmental temperature and greatly accelerates heat loss by convection; wind chill is most significant with the first 20 m.p.h. increase in wind speed, with little additional effect at higher wind speeds.
- Cold-water swimmers lose significant heat by convection because continuous motion prompts ongoing displacement of water away from the body, resulting in constant exposure of a "warm" body to cold water.
- Windproof clothing, especially combined with appropriate insulation layers, can greatly reduce convective heat loss in most environments by trapping warm air and minimizing air displacement.

CONDUCTION

- Conduction involves direct transfer of heat from the body when it is in contact with a surface colder than body temperature (e.g., water, snow, ice, rocks).
- Water is an excellent conductor, and it has an exponentially greater volumetric heat capacity than air, which can lead to significant heat losses for anyone immersed in cold water, especially without protective clothing.
- Wet clothing also increases conductive heat losses.

Mechanisms of Heat Production

- Metabolic and biochemical reactions (small increase)
- Muscular activity
  - Involuntary shivering (increases heat production 2 to 6 times basal level; subsides after several hours when core temperature drops below 30° C)
  - Voluntary exercise (increases heat production up to 10 times basal level)
- Nonshivering thermogenesis
  - Increases in thyroxine, epinephrine, and norepinephrine and a subsequent small increase in overall tissue metabolism
  - Relatively ineffective for preventing cold injury; may increase heat production 10% to 15% in adults)

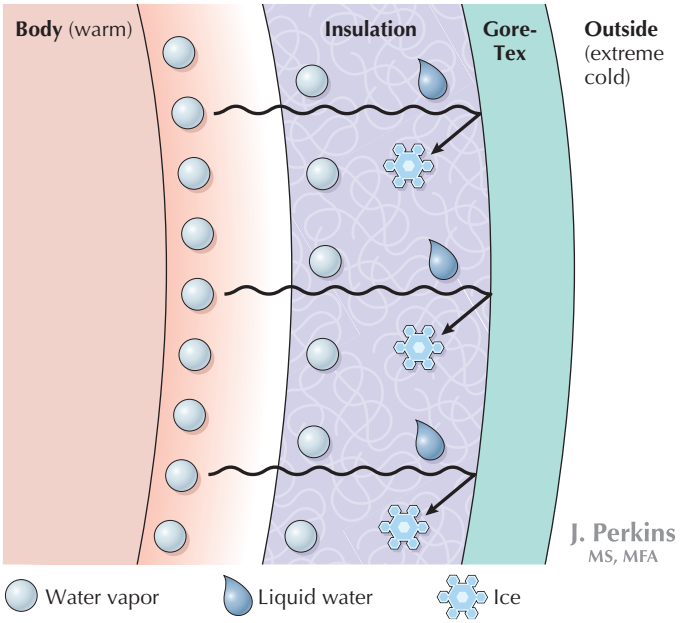

Figure 18-1 Vapor Barrier Systems.

Table 18-1 WIND CHILL CHART

WIND (MPH) Equivalent Temperature (Degrees Fahrenheit) *WIND (KPH) Equivalent Temperature (Degrees Celsius [Centigrade])*														
Calm	**35**	**30**	**25**	**20**	**15**	**10**	**5**	**0**	**−5**	**−10**	**−15**	**−20**	**−25**	**−30**
	2	−1	−4	−7	−9	−12	−15	−18	−21	−23	−26	−29	−32	−34
5 mph	**33**	**27**	**21**	**16**	**12**	**7**	**1**	**−6**	**−11**	**−15**	**−20**	**−26**	**−31**	**−35**
8 kph	1	−3	−6	−9	−11	−14	−17	−21	−24	−26	−29	−32	−35	−37
10 mph	**21**	**16**	**9**	**2**	**−2**	**−9**	**−15**	**−22**	**−27**	**−31**	**−38**	**−45**	**−52**	**−58**
16 kph	−6	−9	−13	−17	−19	−23	−26	−30	−33	−35	−39	−43	−47	−50
15 mph	**16**	**11**	**1**	**−6**	**−11**	**−18**	**−25**	**−33**	**−40**	**−45**	**−51**	**−60**	**−65**	**−70**
23 kph	−9	−12	−17	−21	−24	−28	−32	−36	−40	−43	−46	−51	−54	−57
20 mph	**12**	**3**	**−4**	**−9**	**−17**	**−24**	**−32**	**−40**	**−46**	**−52**	**−60**	**−68**	**−76**	**−81**
32 kph	−11	−16	−20	−23	−27	−31	−36	−40	−43	−47	−51	−56	−60	−63
25 mph	**7**	**0**	**−7**	**−15**	**−22**	**−29**	**−37**	**−45**	**−52**	**−58**	**−67**	**−75**	**−83**	**−89**
40 kph	−14	−18	−22	−26	−30	−34	−38	−43	−47	−50	−55	−59	−64	−67
30 mph	**5**	**−2**	**−11**	**−18**	**−26**	**−33**	**−41**	**−49**	**−56**	**−63**	**−70**	**−78**	**−87**	**−94**
48 kph	−15	−19	−24	−28	−32	−36	−41	−45	−51	−53	−57	−61	−66	−70
35 mph	**3**	**−4**	**−13**	**−20**	**−27**	**−35**	**−43**	**−52**	**−60**	**−67**	**−72**	**−83**	**−90**	**−98**
56 kph	−16	−20	−25	−31	−33	−37	−42	−47	−51	−55	−58	−64	−68	−72
40 mph	**1**	**−4**	**−15**	**−22**	**−29**	**−36**	**−45**	**−54**	**−62**	**−69**	**−76**	**−87**	**−94**	**−101**
64 kph	−17	−16	−26	−30	−34	−38	−43	−48	−52	−56	−60	−66	−70	−74

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Thermoregulation and Physiologic Adaptations

- The hypothalamus is the thermoregulatory center for maintenance of body temperature and physiologic response to cold.
- The body needs to stay between 34° C and 40.5° C (95° F and 105° F) to maintain normal organ function; core temperature normally hovers around 36.5° C to 37.5° C (97.7° F to 99.5° F).
- The body dissipates heat relatively efficiently but is less effective at compensating for cold.
- The body responds initially to cold by regulating **core** (brain and other major organs) and **shell** (skin, muscle, extremities).
  - Exposure to cold stimulates thermoreceptors in the skin that signal hypothalamus to activate shivering and nonshivering thermogenesis, constrict peripheral or "shell" circulation, and decrease sweating in an effort to increase heat production and maintain core temperature.
  - The body shell acts as an interface between the body and environmental stresses—shell temperature may vary widely, in contrast to core temperature.
  - Athletic performance may be decreased with shell cooling as cold weakens and slows muscle contractions, decreases tissue elasticity, and slows nerve conduction.
- Prolonged cooling causes body core temperature to drop and results in many physiologic changes:
  - When core temperature falls below 95° F (35° C), rates of essential biochemical reactions slow.
  - Shivering gradually ceases at approximately 88° F to 90° F (31° C to 32° C) as muscles become cooler and stiffer.
  - Severe cold may affect all organ systems, particularly the central nervous system (CNS) and the cardiovascular system (Table 18-2).
  - As temperature decreases, heart rate and cardiac output fall and arrhythmias may occur secondary to myocardial irritability.
  - Cerebral blood flow decreases, leading to dilated pupils, obtundation, stupor, and eventually coma.
  - Respiratory rate slows and oxygen consumption decreases.
  - Diuresis occurs secondary to shunting of blood volume to the body core from the periphery.
  - Lactic acidosis develops secondary to decreased shell tissue perfusion (decreased tissue oxygenation) and subsequent anaerobic glycolysis combined with carbon dioxide retention that occurs with reduced respiratory rate.
  - Insulin activity decreases and hyperglycemia develops.

Exercise in the Cold

Sports Associated with Cold Injury

- Any cold weather or cold water sport can be associated with cold injury.
- Prerequisites include *cold temperature*, frequently augmented by wind and precipitation, and *human exposure*, where athletes are inadequately prepared or are unable to protect themselves from cold exposure for other reasons.
- Specific sports may include skiing (all types), snowshoeing, mountaineering and rock climbing, snowmobiling, canoeing, kayaking, white-water rafting, open water swimming, and skating. Windsurfing and small boat sailing may result in cold injury after unexpected capsizing. Unsuspecting runners, hikers, backpackers, and cyclists may also experience significant cold injuries, especially with prolonged and inadequate exposure combined with unexpected weather (e.g., blizzard, cold rain, high winds, dropping temperature and wind chill).

Prevention

Almost all cold illness is caused by exposure, often unexpected, to cold without adequate protection. Proper planning is imperative to prevent cold illness and injury.

INCREASE HEAT PRODUCTION

- Increasing heat production is the least effective way to prevent cold illness.
- Exercise: voluntary muscular activity will produce heat.
- Shivering: involuntary muscular activity also will produce heat.
- Eating: frequent meals or snacks are needed to replenish glycogen and fat stores, especially with prolonged exercise in cold environments.
- Hydration: consume adequate fluids to prevent dehydration and subsequent impairment in circulating blood volume.

DECREASE HEAT LOSS

- Decreasing heat loss is the most effective way to prevent cold illness.
- Physiologic mechanisms of increasing heat production are much less effective than strategies applied to decrease heat loss.
- Heat loss is mainly prevented with adequate **"layering"** of clothing.
- Insulation and permeability are important properties of cold weather clothing.

Table 18-2 CLINICAL MANIFESTATIONS OF HYPOTHERMIA

System	Mild hypothermia (35-32° C [95-90° F])	Moderate hypothermia (32.2-28° C [90-82.4° F])	Severe hypothermia (<28° C [82.4° F])
Central nervous system	Confusion, slurred speech, impaired judgment, amnesia	Lethargy, hallucinations, loss of papillary reflex, EEG abnormalities	Loss of cerebrovascular regulation, decline in EEG activity, coma, loss of ocular reflex
Cardiovascular system	Tachycardia, increased cardiac output, and systemic vascular resistance	Progressive bradycardia (unresponsiveness), decreased cardiac output, BP, atrial and ventricular arrhythmias, J (Osborn) wave on EKG	Decline in BP and cardiac output, ventricular fibrillation (<28° C [82.4° F]) and asystole (<20° C [68° F)
Respiratory	Tachypnea, bronchorrhea	Hypoventilation (decreased RR and tidal volume), decreased oxygen consumption and CO_2 production, loss of cough reflex	Pulmonary edema, apnea
Renal	Cold diuresis	Cold diuresis	Decreased renal perfusion and GFR, oliguria
Hematologic	Increase in hematocrit, decreased platelet count and white blood cell count, coagulopathy, and DIC		
Gastrointestinal		Ileus, pancreatitis, gastric stress ulcers, hepatic dysfunction	
Metabolic Endocrine	Increased metabolic rate, hyperglycemia	Decreased metabolic rate, hyper- or hypoglycemia	
Musculoskeletal	Increased shivering	Decreased shivering (<32° C [89.6° F])	Patient appears dead "pseudo-rigor mortis"

DIC, Disseminated intravascular coagulation.
Reprinted with permission from Mellion M: Sports Medicine Secrets, 3rd ed. Philadelphia: Hanley & Belfus, 2002.

- Most versatile cold weather clothing systems are usually composed of three layers:
  - Inner hydrobic polyester fabric (e.g., Capilene, Coolmax, Thermax, Thermolite, Thermostat) that allows wicking of moisture away from the body. Avoid cotton.
  - Middle insulating material can be second light layer (similar to inner layer fabric) or heavier layer (can cause overheating; best used during exercise warm-up, cool-down, or in extreme cold) such as wool and wool/synthetic blends (heavy when wet; excellent insulator, even when wet), pile and fleece (varying weights offer versatility for exercising athlete, fleece replacing pile), synthetic fillers (Primaloft, Liteloft, Dacron, Hollofill, Quallofil, Thinsulate) or down (good in cold, dry conditions; loses insulating properties when wet); middle layer can lead to overheating.
  - Outer protective shell that is windproof and water repellant (e.g., newer treated nylons):
    - Newer treated nylons are best because they offer highest breathability; fibers are tightly woven and sprays are used to increase water repellency.
    - Laminates such as Gore-Tex and other brand-specific materials designed to mimic Gore-Tex are advertised as "waterproof" and breathable, but the amount of sweat produced during exercise can exceed capability of laminate to transmit water vapor.
    - Ideal fabric that allows water vapor to pass outward but not inward has not been developed.
    - Breathability of outer layer is inversely proportional to the degree of water repellency and waterproofing; adequate breathability is important during exercise.
- Middle and outer layers should each be slightly larger than the direct inner layer to allow a small space for warm air trapping; avoid "tight" layering.
- The primary goal with layering is to stay relatively warm without excessively sweating or overheating, and layers need to be shed or added depending on current activity level and environmental conditions.
- Ventilation ports in some outer garments may be opened for adequate breathing, and are especially important when using laminates.
- Excessive sweating causes heat loss through increased evaporation and conduction and may affect insulating properties of certain fabrics.
- Wind chill can cause dangerously large amounts of heat loss through convection without windproof garments and proper insulation adjusted to activity level.

SPECIAL PROTECTION

- Special protection and other measures to minimize heat loss and injury should be applied.
- Prevent significant conductive loss by placing a "barrier" between body and colder objects (e.g., foam or other sleeping pads such as Therma-Rest, wet or dry suits with cold water exposure, leather or plastic boots with an inner insulating layer).
- Maintain adequate trunk warmth to minimize vasoconstriction in hands and feet that may result in subsequent injury.
- Wear mittens instead of gloves, preferably with a protective, windproof outer shell and an inner insulating layer.
- Prevent radiant heat loss from the head (blood supply to the head is maintained in cold) by wearing an insulating cap or balaclava, and by using a hood when needed (high wind, wet weather).
- Protect exposed skin of face and ears by using a balaclava or neck gaiter pulled up over face (neck gaiters also decrease heat loss from relatively superficial, large vessels of the neck).
- Prevent genital injury by wearing undershorts with windproof front panel.
- Protect eyes (corneal freezing) by using ski goggles.
- Avoid excessive wetting at all times, and if immersed in cold water, minimize movement and pull body into a "tight" position (assume the HELP posture—**h**eat **e**scape **l**essening **p**osture—which is similar to an "upright fetal" position) to decrease exposure surface area, unless shore or boat is close.

- Adequately warm-up before exercising in cold conditions (may warm-up indoors, or use insulated, protective clothing if outdoors).

EXTERNAL WARMING SOURCES

See Treatment discussion in the following "Accidental Hypothermia" section.

SPECIFIC INJURIES

Systemic Cold Injury

Accidental Hypothermia

Definition: Unintentional decline in core body temperature below 35° C (95° F) not due to organic disease.

- **Mild** (35° C to 32° C [95° F to 90° F])
- **Moderate** (<32° C to 28° C [90° F to 82° F])
- **Severe** (<28° C [82° F])
- Some authorities define values more conservatively. The advanced cardiac life support (ACLS) algorithm for the treatment of hypothermia classifies severe hypothermia as below 30° C (86° F).

Risk factors: Old age, infancy, alcohol, CNS depressants, hypothyroidism, hypopituitarism, hypoadrenalism, diabetes mellitus, sepsis, central nervous system insult (e.g., cerebral vascular accident, trauma, etc.), malnutrition, and hypoglycemia.

Presentation: Assume hypothermia until proven otherwise when evaluating an athlete with confusion, dysarthria, and ataxia who has been exposed to cold (see Table 18-2). Rectal temperature (estimation of core temperature) and clinical findings establish definitive diagnosis, but low-reading rectal thermometers are frequently not available. **Low-reading thermometers should be available in first-aid kits during cold weather athletic events.** Degree of hypothermia can be estimated using clinical observation: quickly note **level of consciousness** and **presence of shivering.** A conscious individual who is shivering likely has a core temperature above 88° F to 90° F (31° C to 32° C); a severely confused or obtunded individual who is not shivering likely has a core temperature below 88° F to 90° F.

- **Mild hypothermia:** Confusion, poor judgment, dysarthria, amnesia, dizziness, apathy and fatigue, mood lability, ataxia, increased shivering, tachycardia, tachypnea, elevated blood pressure, urinary frequency (cold diuresis), hyperreflexia, and hyperglycemia.
- **Moderate hypothermia:** Lethargy, stupor, occasional unconsciousness, shivering slows, becomes intermittent, and then ceases; hallucinations, loss of pupillary reflex, bradycardia, atrial fibrillation and other arrhythmias, decreased ventricular fibrillation threshold, hypotension, decreased rate and volume of breathing, hyporeflexia or areflexia, muscle rigidity and decreased or absent voluntary motion, paradoxical undressing, and electrocardiogram changes (prolonged P-R, QRS, and Q-Tc intervals, J (Osborn) wave (Fig. 18-2).
- **Severe hypothermia:** May appear dead, profound coma, hypotension, respiratory depression, pupils fixed and dilated, severe muscular rigidity, areflexia (even to painful stimuli), atrial arrhythmias common, ventricular fibrillation easily induced with minimal movement of patient, asystole, pulseless electrical activity, and other arrhythmias, pulmonary edema, oliguria, coagulopathy.
- **Paradoxical undressing:** A conscious, profoundly hypothermic patient undresses, usually when near or just before death. Perception of warmth and cold relies on temperature receptors near the skin surface. When core temperature drops significantly, vasoconstricted vessels in the periphery may suddenly dilate, "fooling" the body into thinking it is warm. Combined with severely impaired consciousness and judgment, the feeling of warmth causes the affected individual to shed clothing. This is a poor prognostic sign.

Treatment:

General Principals (all degrees of hypothermia)

- **Preventing further heat loss** is highest priority.
- Gently *remove* hypothermic individual *from the cold* to a shelter (tent, snow cave, other natural or man-made shelter) and *insulate* against further heat loss.
- Insulate using sleeping bags, or by using a combination of sleeping bags and a vapor barrier such as a tarp, parka, or plastic bag.
- Minimize conductive heat loss may by placing blankets or a sleeping pad between the patient and the ground.
- Affected individual should be kept in a horizontal position to minimize potential orthostatic worsening of hypotension.
- Remove all wet clothes by gently cutting them off.
- Hypothermia should always be treated before frostbite.

Mild Hypothermia

- Patients without impaired consciousness or severe confusion and dysarthria should be encouraged to eat warm food and to drink warm, sugar or calorie-containing noncaffeinated fluids to boost morale, supply calories, and curb dehydration.
- Once adequately protected from the cold and well insulated, the affected individual should be allowed to "shiver back to normal" (passive rewarming).
- Exercise is occasionally recommended in individualized circumstances.

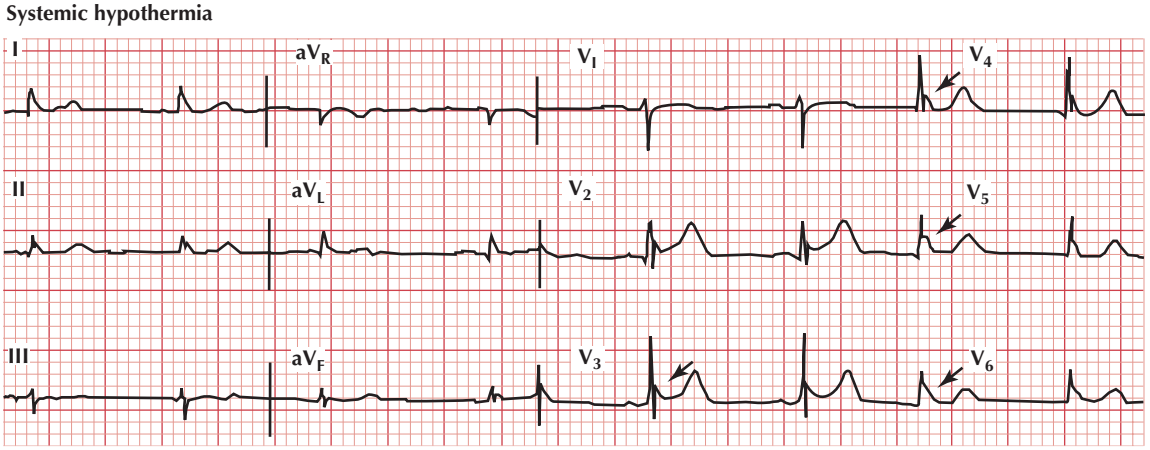

Figure 18-2 Systemic Hypothermia. *(Reprinted with permission from Goldberger A: Clinical Electrocardiography: A Simplified Approach, 7th ed. Philadelphia: Mosby, 2006.*

- Some practitioners advocate the use of active external warming measures (see following discussion) or noninvasive internal rewarming in the field with heated humidified oxygen.
  - These rewarming measures do not likely significantly increase core temperature and are controversial because they may hinder the shivering mechanism, and subsequently the warming process, by warming the body periphery.
  - Exogenous rewarming techniques are typically not needed in conscious, shivering individuals because shivering is more effective than exogenous rewarming, which inhibits shivering.

Moderate and Severe Hypothermia

- **Field Management:**
  - All of these patients are **unable to rewarm themselves.**
  - Field management involves preventing further heat loss, stabilizing the patient, and arranging for emergent transport to a medical facility for definitive care.
  - Basic life support principles should be applied. Cautious endotracheal intubation is indicated in patients with significantly impaired consciousness, coma, or respiratory distress. Moderate and severe hypothermic victims should be handled extremely gently because minimal motion may trigger ventricular fibrillation that is often fatal.
  - After affected patients are adequately insulated, some experts advocate exogenous rewarming in the field, but practice varies, and other experts feel that any rewarming should be attempted in a hospital setting only.
  - Overall, when **shivering is absent** in moderate to severe hypothermic individuals, some form of **active external rewarming,** in addition to **noninvasive active internal rewarming** (e.g., heated humidified oxygen), is probably safe in the field.
  - Humidified warmed oxygen will not likely affect core temperature significantly, but may prevent heat loss through the airways.
  - Slow administration of warmed intravenous D5 normal saline provides only a small amount of heat, but it may slowly correct intravascular volume depletion and may help stabilize the conduction system of the heart.
  - **Active external rewarming** involves the application of exogenous heat sources.
    - Forced heated air devices (e.g., Bair Hugger)
    - Hot packs or bottles placed over high-flow areas such as the neck, axilla, and groin
    - Hot pads or blankets
    - Radiant heat using heat lamps
    - Immersion of hands, forearms, lower legs, and feet in warm water (44° C to 45° C [111° F to 113° F])
    - Body-to-body contact
  - Active external rewarming techniques are relatively ineffective for core rewarming compared to active internal rewarming techniques. If used, care must be taken to avoid thermal burns with certain techniques.
  - Minimal warming of the cardiovascular and respiratory systems may help stabilize cardiorespiratory parameters, even if core temperature is not increased significantly.
  - Because core temperature is essentially unaffected, or at best minimally elevated, complications resulting from rapid rewarming do not usually occur. The amount of core rewarming, if any, achieved using these methods is controversial, but some studies suggest a benefit.
  - A recent survey of Mountain Rescue Association teams to determine common rescue and treatment practices reports the use (by number of teams) of the following rewarming techniques: chemical pads (19); spontaneous or passive rewarming using sleeping bag (16); hot water bottles (13); warm intravenous fluids (7); warm oxygen or air inhalation (3); charcoal Heatpack; water-perfused sarong (1). Active noninvasive and minimally invasive internal rewarming techniques sometimes used in the field (by those who advocate field rewarming) include warmed oxygen and intravenous fluids.
  - In conclusion, exogenous rewarming may be instituted cautiously in moderate to severely hypothermic patients, especially if active internal core rewarming is not immediately available.
  - **Afterdrop** considerations:
    - Central perception of warmth caused by warm objects in contact with the body causes decreased vasconstriction in cold extremities.
    - Blood may be cooled rather then warmed as is flows through the periphery, especially after exercise, and shunting of blood from the core to the cold periphery followed by the return of cool blood may cause a significant **"afterdrop"** in core body temperature in moderate to severe hypothermics.
    - Afterdrop, combined with dehydration, cold diuresis, relative peripheral vasodilation, fluid sequestration in the tissues, and venous pooling may lead to relative core hypovolemia and hypotension (rewarming shock).
    - There is no strong evidence that supports theoretical massive peripheral vasodilation and subsequent hypotension and shock resulting from exogenous rewarming, with maybe the exception of whole body immersion in hot water, which should be avoided in all settings.
- **Cardiopulmonary Resuscitation (CPR)**
  - **Cardiopulmonary resuscitation (CPR) is indicated if there is no sign of pulse or breathing after assessing for 60 seconds.** It may be extremely difficult to detect pulses or respirations in profoundly hypothermic individuals.
  - **Do not initiate CPR in patients with a discernable pulse because a lethal cardiac rhythm may easily be precipitated.** A cardiac monitor should be used if available.
  - If ventricular fibrillation occurs at a core temperature below 30° C (86° F), do not administer medication and limit defibrillation to one shock (200 J biphasic) because defibrillation rarely succeeds at these temperatures. Once temperature is 30° C (86° F) or above, medication may be administered (at longer than standard intervals) and defibrillation is repeated as indicated.
  - The most frequently used antiarrhythmic medication with hypothermia historically was bretylium tosylate. It is no longer included in the ACLS guidelines. Other medications are usually relatively ineffective and excessive pharmacologic intervention should be avoided with a depressed, vasoconstricted cardiovascular system. Magnesium sulfate may help stabilize the ventricles and convert ventricular fibrillation in hypothermic patients.
  - Rate of chest compression should be half normal.
  - No patient should be pronounced dead until he or she is "warm and dead."
  - CPR should not delay transfer to a medical facility for definitive management, and ideally should be performed simultaneously with transfer, even if intermittent.
- **Hospital Management**
  - **Serious cardiovascular, central nervous system, and acid-base complications may occur with rewarming.**
  - All patients should be placed on a cardiac monitor, given warm humidified oxygen, and have intravascular access. Warm D5 normal saline (104° F [40° C]) is administered as indicated to correct dehydration and volume depletion (elevates core temperature minimally, <1° C per hour). Endotracheal tube, nasogastric tube, and bladder catheter are used in appropriate clinical situations.
  - Appropriate labs (complete blood count, comprehensive metabolic profile, coagulation studies, consider uncorrected

arterial blood gas) should be drawn and close serial monitoring is required. Serum potassium greater than 10 mEq/L in the presence of hypothermia is a strong marker for death. ECG and chest x-ray should be performed.
- Some experts believe that limiting the application of external heat to the trunk may minimize the theoretical circulatory problems associated with active external rewarming. Still, many medical facilities use active external rewarming for significant mild and some moderate hypothermia.
- Heated humidified oxygen raises core temperature by 1° C to 2° C per hour, and it may be a useful adjunct to other rewarming methods. Passive external rewarming alone in a warm environment will raise core temperature approximately 1.5° C per hour.
- Active core rewarming techniques deliver direct heat to the body core and are the procedures of choice in severely hypothermic individuals and in individuals with cardiovascular instability or poor perfusion. Temperature monitoring is best accomplished with an esophageal temperature probe.
  - The **gold standard** is **extracorporeal blood rewarming**—it may raise core temperature 1° C to 2° C every 3 to 5 minutes.
  - Other effective methods include peritoneal lavage, and more recently, closed thoracic lavage (both raise temperature 2° C to 3° C per hour).
  - Arteriovenous rewarming using a negative pressure device placed over the forearm shows promise for treating hypothermia. The device works by overcoming peripheral vasoconstriction through application of a significant vacuum pressure that "opens up" arteriovenous anastamoses, and then applies a direct thermal load to the high-flow anastamoses using a chemical heating pad.
  - Less effective methods include gastric, colonic, and bladder irrigation.

COLD WATER IMMERSION

- Drowning or fatal cardiac arrhythmia may cause death earlier than hypothermia after cold water immersion.
- Hypothermia usually requires a significant duration of immersion (30 minutes to 2 hours) to kill a victim. On the other hand, drowning may occur relatively quickly as a result of the cold shock response.
- Sudden exposure to cold water causes an immediate and involuntary gasp that may result in aspiration of water and/or laryngospasm. This is followed by profound hyperventilation that results in respiratory alkalosis and subsequent muscle tetany and cerebral hypoperfusion.
- Breath-holding duration is reduced significantly (kayakers that roll may even have a hard time holding their breath for the duration of the roll).
- Severe tachycardia, increase in blood pressure, and vagal overload may induce lethal cardiac arrhythmias.
- Cold water–induced peripheral vasoconstriction facilitates rapid cooling of the extremities and greatly decreases neuromuscular activity, making it near impossible to tread water (if no flotation device), hold onto a flotation device, signal, or grasp anything (e.g., rescue line or hoist).
- If an arrhythmia does not occur, immersed individuals often drown because of excessive inhalation of water (especially with turbulent conditions) and failure to initiate or maintain survival performance.

Local Injury

Frostbite

Definition: Localized cold injury produced by freezing of tissues. Hands, feet, face (especially nose), and ears are most frequently affected. Frostbite may occur in any environment where the temperature is below freezing (0° C [32° F]), and it is frequently associated with hypothermia.

Risk factors: Environmental risk factors may include cold temperature, wind chill (wind velocity plus temperature), high altitude, high humidity, prolonged exposure, and direct skin contact with conductors such as cold metal, gas, or other liquids. Other risk factors may include previous frostbite, wet or inadequate clothing, dehydration, diminished mental capacity (e.g., drug-induced or other), associated trauma, alcohol use, tobacco use, use of drugs that cause vasoconstriction or alter thermoregulation, and comorbid medical conditions, especially those that compromise circulation (e.g., atherosclerosis, diabetes mellitus).

Athletes at risk: Any athlete exercising or competing in cold weather, especially without adequate protection, may sustain frostbite. Joggers are most frequently affected. Alpine and Nordic skiers, mountaineers and climbers, snowshoers, snowboarders, cold-weather distance runners and cyclists, speed skaters, snowmobilers, and players on almost any outdoor (cold weather) team sport (e.g., football, soccer) may incur frostbite.

Pathophysiology: Frostbite occurs with tissue freezing, subsequent tissue ischemia and release of inflammatory mediators, and eventual healing or tissue necrosis. The net result of frostbite involves varying degrees of direct cell damage and progressive tissue ischemia. Inflammatory mediators such as prostaglandins, thromboxanes, bradykinin, and histamine likely play a significant role in endothelial injury, edema formation, and arrest of dermal blood flow in all phases.

Pathophysiological classification: prefreeze, freeze-thaw, vascular stasis, and **late ischemic phases**
- *Prefreeze:* Tissue chilling, alternating vasoconstriction and vasodilation, capillary membrane instability, plasma leakage, and early edema.
- *Freeze-thaw:* Cyclic vasodilation and vasoconstriction at cold temperatures contributes to tissue freezing and partial thawing (causes significant damage); extracellular ice crystal formation is followed by fluid shifts that cause intracellular dehydration and intracellular ice crystal formation; vascular endothelium may encounter microemboli; and arteriovenous shunting bypasses obstruction (leading to severe hypoxia of affected tissue).
- *Vascular stasis phase:* Continued plasma leakage, formation of ice crystals, vasospasm and vasodilation, and shunting. Stasis coagulation may be more pronounced than at earlier stages.
- *Late ischemic phase:* Characterized by continued tissue ischemia, vascular thrombosis, arteriovenous shunting, autonomic dysfunction, denaturation of tissue proteins, and tissue necrosis and gangrene.

Clinical classification: Historically, frostbite is classified into four categories: first-, second-, third-, and fourth-degree frostbite. A more clinically practical classification of **superficial** (previous first- and second-degree) and **deep** (previous third- and fourth-degree) frostbite is now more frequently used by clinicians.
- *Superficial frostbite* involves only the skin, and permanent tissue loss almost never occurs.
- *Deep frostbite* involves the skin and underlying tissue, which may include muscles, nerves, vessels, bone, and cartilage, and it is almost always associated with permanent tissue loss.
- It is important to realize that most frostbite injuries appear the same at initial evaluation (unless thawing has started), and that the *classification of frostbite is applied when tissue changes become evident after rewarming*.

Presentation:
- **Initial freezing:** coldness, numbness, clumsiness, and pain of affected extremity.
- As freezing progresses, pain disappears and the affected body part turns stiff and hard.
- All degrees of frostbite usually initially present with paleness, coldness, and firmness of affected tissue.

- The affected body part may also appear yellowish, mottled blue, and waxy.
- Initial tissue pliability may indicate superficial frostbite, whereas frozen-solid tissue without pliability usually indicates deep frostbite.
- As tissue thaws with **rewarming,** signs and symptoms follow a relatively predictable pattern that illustrates the severity of tissue injury.
- Numbness and throbbing pain occur with tissue thawing.
- *Superficial frostbite:* hyperemia, increased sensation, and subsequent edema and blisters filled with clear or yellow fluid occur (Fig. 18-3A).
- *Less severe deep frostbite:* hemorrhagic blisters form, eschars form after blisters desquamate (Fig. 18-3B).
- *Severe deep frostbite:* no blebs or blisters form, edema is minimal (especially proximal to injury), the affected body part lacks sensation, skin initially turns dark reddish and purple and eventually black, and mummification and autoamputation may occur weeks after injury (Fig. 18-3C).
- Mummification forms the line of demarcation between viable and necrotic tissue.
- Progression of events: edema is usually present a few hours after thawing, blisters form within 24 hours, eschars may form over affected tissue after 1 to 2 weeks, and mummification may occur between 3 and 6 weeks.

Treatment: General Considerations

Field treatment of frostbite should be balanced against time for evacuation and available treatment facilities. Frostbite is best rewarmed in an appropriate medical facility under tightly controlled conditions. The longer that tissue stays frozen, the greater the tissue damage. Because of this, some experts recommend rapid field rewarming (provided appropriate equipment is available) if evacuation is not imminent (within a few hours) and if the affected body part can absolutely be protected from refreezing (more significant tissue damage may occur with refreezing after thawing). Spontaneous thawing may be allowed in the same circumstances if equipment is not available for rapid rewarming, but morbidity is higher than with rapid rewarming, and this recommendation is highly controversial. If there is no other option, individuals with frostbitten feet may walk to safety, but they must realize that their feet will likely thaw and they may become incapacitated.

Field Treatment

- The *best treatment is prevention*. Adequate protection and proper planning minimizes exposure of skin to cold environmental conditions.
- Continuously monitor tissue status in cold conditions, especially if numbness is present, and seek safe conditions with any warning signs of frostbite.
- Individuals exposed to cold should:
  - Wear mittens instead of gloves.
  - Ensure adequate trunk insulation (prevents peripheral vasoconstriction).
  - Keep head, face, and neck covered.
  - Avoid wetting (via exposure or perspiration).
  - Avoid tight footwear and clothing.
  - Avoid prolonged exposure to cold.
  - Avoid using tobacco, alcohol, and vasoconstrictive drugs.
  - Maintain adequate hydration and caloric intake.
  - Avoid direct skin contact with cold conductive substances such as metal or gasoline.
- Avoid cold-protecting ointments or emollients**.**
  - Application of ointments or emollients, especially to the face and ears, is a traditional technique believed by many to protect against frostbite.
  - Use of ointments is especially common in women and children of Finland, where the incidence of frostbite is high.
  - Use of ointments may actually be a considerable risk factor for developing frostbite, especially of the face and ears. Ointments at best provide a negligible thermal insulation effect, and may even provide a negative effect. However, they create a subjective skin warming perception during acute cold exposure that may contribute to a false sensation of safety. The net result is likely increased incidence of frostbite, probably resulting from a neglect of effective protective measures.
- All frostbitten individuals should be evacuated to an appropriate medical setting as soon as possible.
- Ibuprofen or other antiinflammatory medication should be administered (inhibits inflammatory mediators involved in tissue injury).
- Tight and/or wet garments should be removed and replaced with dry insulating garments that protect the affected body part from further exposure.

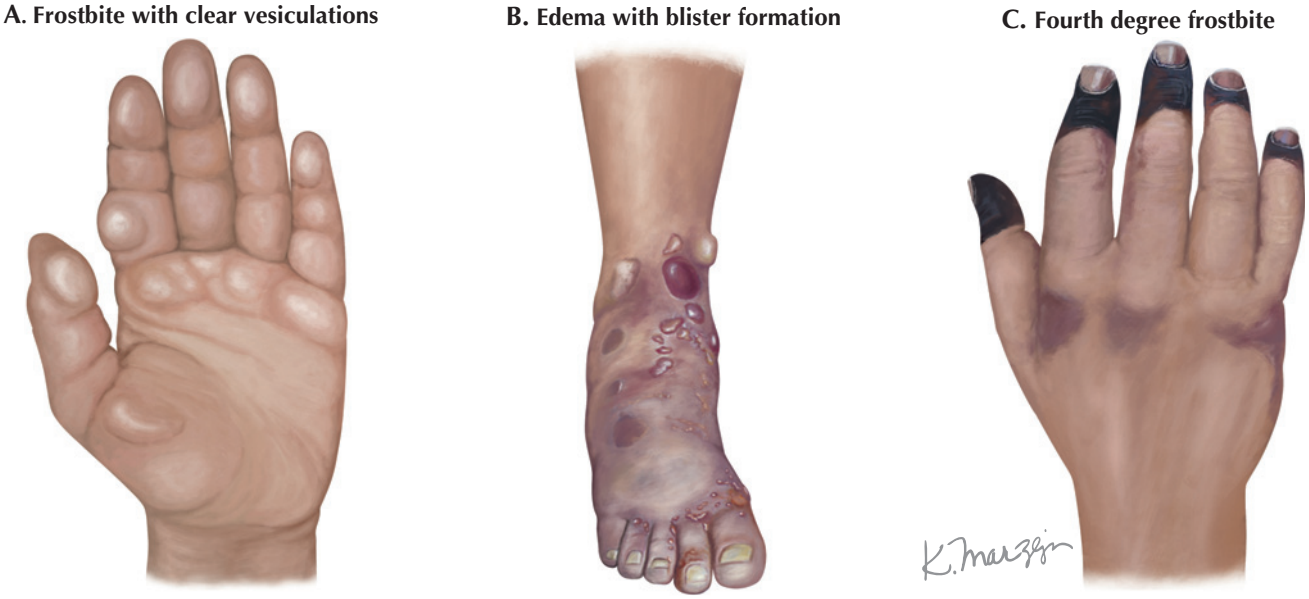

Figure 18-3 Frostbite.

- Affected extremities should be adequately padded.
- Oral hydration should be encouraged.
- If equipment is available, some experts feel that rapid rewarming may be performed during transport if transport time to a definitive treatment facility is minimal.
- Avoid tissue massage and using dry or radiant heat.

Definitive Treatment: Rapid Rewarming of Frozen Tissue

- Rapid rewarming protocols are based on the work of McCauley and colleagues.
- Frostbite patients should be admitted to a specialized unit if available. Affected body parts should be rapidly rewarmed in warm water (40° C to 42° C [104° F to 108° F]) for 15 to 30 minutes, or until thawing is complete.
- Active motion during rewarming is helpful, but massage should be avoided.
- White blisters should be debrided, hemorrhagic blisters should be left intact, and aloe vera should be applied every 6 hours.
- Affected body parts should be elevated and splinting may be used as indicated.
- Antitetanus prophylaxis, ibuprofen or other anti-inflammatory medicines, and narcotic analgesics are administered.
- Some experts advocate the use of prophylactic benzyl penicillin every 6 hours for 2 to 3 days.
- Perform daily hydrotherapy for 30 to 45 minutes with water at 40° C (104° F).
- Adequate intravascular hydration should be maintained. Some experts use dextran (plasma expander) and vasodilators.
- Photographs of frostbitten tissue may be taken at admission, 24 hours, and every few days until discharge.
- Close serial follow-up should be arranged after discharge until all wounds are stable.
- The saying "Frostbite in January, amputate in July" illustrates that surgical debridement of severely frostbitten tissue is normally reserved for late treatment of frostbite, after the demarcation line between viable and necrotic or gangrenous tissue is clear.
- Traditionally, the decision of whether to debride (and sometimes reconstruct) or allow autoamputation of affected tissue must be faced months after initial injury, and early surgical intervention is reserved for escharotomy and fasciotomy as indicated for circulatory compromise and compartment syndrome.
- Early surgical intervention involving free tissue transfer to improve vascularization of potentially viable tissue is currently being studied. However, early identification of viable tissue is impossible clinically. The use of technetium bone scanning (scintography) within the first few days after injury may help predict eventual tissue demarcation by identifying deep tissue and bone infarction, but fails to identify the condition of surrounding, potentially viable tissue. Magnetic resonance imaging (MRI) and magnetic resonance angiography (MRA) show promise because they allow direct visualization of surrounding tissue, and may allow a more accurate early assessment of the line of demarcation between viable and ischemic tissue.

Long-term sequelae: Significant tissue and limb loss may occur with deep frostbite. Other morbidity may include permanent cold sensitivity and susceptibility to cold injury, sensory loss, tingling and paresthesias, hyperhydrosis, chronic pain, growth plate disturbances (phalanges of children most common), osteoarthritis, and heterotopic calcification.

Frostnip

Description: Occurs prior to tissue freezing when cold induces local vessel constriction.

Symptoms: Ice crystals may form on the skin surface and the skin may appear whitish or pale.

Physical examination: Deeper tissues are soft and pliable, and affected skin is easily thawed by covering the face (with mittens, neck gaiter, etc.) or by placing affected hands in armpits or under other garments.

Treatment: Almost instant thawing may be followed by redness, pain and hypersensitivity, and sometimes swelling. Skin often peels a few days later. **Frostnip signals inadequate protection** and **risky environmental conditions,** and attention should be directed toward preventing frostbite and further cold injury.

Chilblains

Description: Also referred to as *pernio*, chilblains ("cold sores") are characterized by tissue injury that may involve localized erythema, cyanosis, plaques, nodules, and occasionally in more severe cases, vesicles, bullae, and ulceration after *pronounced nonfreezing cold-induced vasoconstriction*.

Pathophysiology: May be related to vascular hypersensitivity, sympathetic instability, and subsequent lymphangiocytic vasculitis.

Symptoms: Symptoms of intense pruritis, burning paresthesias, and skin changes may occur 12 to 14 hours after cold exposure.

Treatment: Involves anti-inflammatory medication, gentle rewarming, dry bandaging, and elevation to minimize swelling.

Trench Foot

Description: Otherwise known as *immersion foot*, trench foot involves tissue (e.g., muscles, nerves, vessels) injury secondary to prolonged vasoconstriction and subsequent ischemia from *prolonged exposure to nonfreezing cold* and *wet conditions*.

Pathophysiology: May share a common pathophysiology with childblains. It progresses through prehyperemic, hyperemic, and posthyperemic phases.

Symptoms: Occur after days and include painful paresthesias, numbness, and initial erythema followed by pallor, mottling, and swelling.

Complications: Vesiculation, ulceration, and gangrene may occur.

Treatment: Involves administering anti-inflammatory medication and keeping affected extremity dry, warm, protected, and elevated.

Prognosis: Lifelong cold sensitivity, and sometimes pain, is common after injury.

Miscellaneous Cold Injuries

COLD-INDUCED BRONCHOSPASM

Description: Cold may induce *bronchospasm*, especially in asthmatics.

Risk factors: Most frequently affects athletes who exercise or compete in cold, dry conditions such as ice hockey and winter outdoor aerobic activities (e.g., running, cycling, cross-country skiing).

Treatment: May include pre-exercise warm-up in a warm environment and/or pretreatment with beta-agonists, mast cell stabilizers, or leukotriene modifiers.

COLD-INDUCED URTICARIA AND/OR ANAPHYLAXIS

Description: Affects athletes most frequently during pre-exercise warm-up in the cold.

Presentation: Athlete presents with wheals, hives, or pruritis; angioedema may be present, but true anaphylaxis involving laryngeal edema and hypotension is rare.

Prevention: Achieved with proper clothing and avoidance of cold-water activities.

Treatment: Acute treatment may include antihistamines, and if anaphylaxis is present, may require administration of epinephrine. Prophylaxis using antihistamines (both H1 and H2 blockers may be used), the tricyclic antidepressant doxepin, and/or leukotriene modifiers is sometimes effective.

RAYNAUD'S PHENOMENON

Description: Vasospastic disorder characterized by initial pallor (ischemia secondary to vasoconstriction) followed by hyperemia (rebound vasodilation) of the digits (fingers most common) after cold exposure and/or emotional stress.

Pathophysiology: May be primary idiopathic (Raynaud's disease) or secondary (Raynaud's phenomenon or syndrome) to a variety of disorders.

Presentation: Initial "white" ischemic phase may be followed by a "blue" cyanotic phase before the "red" hyperemic phase begins.

Treatment: Acute treatment involves warming the affected extremity. Underlying disorders should be addressed.

Prevention: May be achieved by avoiding direct cold exposure, ensuring adequate trunk insulation (minimizes peripheral vasoconstriction), wearing mittens instead of gloves, and using appropriate footwear. Prophylaxis using vasodilating medications (e.g., calcium channel blockers, phosphodiesterase inhibitors) may be attempted if other primary preventive measures fail.

COLD AGGLUTININ DISEASE

Pathophysiology: Usually postinfectious and may follow mycoplasma infections or infectious mononucleosis in athletes. May also be idiopathic or associated with a variety of other disorders.

Symptoms: Symptoms of cyanosis, mottling, numbness, and pain of exposed body parts may occur when cold-activated IgM antibodies agglutinate red blood cells.

Treatment: Initially involves warming the affected body part and protecting from further exposure to cold.

COLD-INDUCED RHINITIS

Description: Occurs in athletes exposed to cold air (e.g., skiers). Form of nonallergic, sometimes referred to as vasomotor, rhinitis that has minimal clinical significance and resolves after removal from the cold environment.

Treatment: Ipatropium nasal spray may be administered if an athlete prefers treatment.

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High-Altitude Training and Competition

19

Benjamin D. Levine and James Stray-Gundersen

HIGH-ALTITUDE ENVIRONMENT

- Athletes must cope with hypoxia, cold, and dehydration, yet maintain maximal performance.
  - Timing of altitude exposure and degree of acclimatization are critical to successful outcome.
  - Physiologic adaptation to high altitude may be beneficial; altitude training is frequently used by elite athletes in attempt to improve sea-level performance.
- Barometric pressure is reduced at high altitude, with a parallel decrease in inspired partial pressure of oxygen (P_IO_2); **hypobaric hypoxia is thus the most prominent physiologic manifestation of high altitude.** Range of terrestrial altitudes may be characterized as follows:
  - 0 to 500 m—sea level
  - 500 to 2000 m—low altitude
  - 2000 to 3000 m—moderate altitude
  - 3000 to 5500 m—high altitude
  - 5500 to 8848 m—extreme altitude
- Temperature decreases at a rate of approximately 6.5° C per 1000 m.
- Other features include dry air (increasing risk of dehydration), decrease in air density and therefore air resistance (dramatic effect on flight characteristics of objects), and increase in amount of ultraviolet light (4% per 300 m), which increases risk of sunburn.

EFFECT OF HIGH ALTITUDE ON EXERCISE

- *Oxygen cascade* is the term used to describe the physiologic effects of high altitude on exercise: Oxygen moves from **environment** (determined by altitude achieved) to **alveoli** (function of ventilation and hypoxic ventilatory response), across **pulmonary capillary bed** (limited by diffusion), to be transported by **cardiovascular system** (function of cardiac output and hemoglobin concentration), and diffused into **skeletal muscle** (dependent on muscle capillarity and biochemical state), to be used by muscle **mitochondria** (influenced by oxidative enzyme activity) for aerobic respiration and ATP production.
- **Altitude-induced hypoxia reduces amount of oxygen available to do physical work.**
  - **Maximal aerobic power ($\dot{V}O_2$ max) is reduced by approximately 1% for every 100 m above 1500 m in normal individuals.**
  - **For endurance-trained athletes, this effect is even greater—reductions in $\dot{V}O_2$ max and performance can be identified at altitudes as low as 500 m and are linear (decrease of about 0.5% to 1.5% for every 100 m altitude) at altitudes from 300 to 3000 m.**
    - Occurs because of diffusion limitation in both lung and skeletal muscle exacerbated by high pulmonary and systemic blood flow (cardiac output) of endurance athletes. Severe hypoxemia results even from base training pace (oxyhemoglobin saturation [SaO_2] <80% at 6 min/mile pace at 2700 m).
    - Training velocity, $\dot{V}O_2$, heart rate, and lactate are lower during interval training at altitude.
    - Training velocity and $\dot{V}O_2$ are lower during typical base training at altitude, but heart rate is the same as training at sea level at the same relative effort, and lactate is slightly higher; these differences complicate determination of appropriate training zones at altitude.
  - **During submaximal exercise at high altitude, ventilation, lactate, and heart rate are greater for the same absolute work rate, increasing sensation of dyspnea and fatigue.**
  - **Peak** blood lactate concentration is **lower** in individuals acclimatized to high altitude (termed *lactate paradox*), though this outcome is controversial and depends on the nuance of the workload and training altitude.
- **Altitude affects endurance athletes and sprinters in different ways.**
  - **Endurance events** requiring high levels of aerobic power (>2 minutes): performance is impaired at altitude because of reduction in $\dot{V}O_2$ max.
  - **Mixed events** requiring high sustained power outputs (30 seconds to 2 minutes): performance may or may not be impaired at altitude depending on interplay of oxidative and glycolytic energy pathways.
  - **Sprint and field events** requiring short bursts of high-intensity activity (≤30 seconds): energy sources are not dependent on oxygen transport. Reduced air resistance at altitude thus actually improves sprint performance. For some athletes (e.g., cyclists), reduced air resistance may be the dominant effect, even for events of longer duration and at much higher altitudes.

ACCLIMATIZATION PROCESS

- Chronic exposure to altitude stimulates acclimatization, which includes adaptations that improve **submaximal** work performance at altitude. At high and extreme altitudes (4000 m and above), $\dot{V}O_2$ max never returns to sea-level values despite prolonged acclimatization. At low altitudes (below 2000 m), maximal oxygen uptake may approach sea-level values after 1 to 2 weeks, at least in nonathletic populations.
  - Increases in alveolar ventilation and reductions in mixed venous oxygen content maximize exercise capacity at altitude—**this begins immediately on ascent.**
  - Hyperventilation causes respiratory alkalosis, which stimulates renal excretion of bicarbonate **over the first week** to normalize acid-base balance.
  - Sympathetic activation acutely (minutes to hours) increases heart rate and cardiac output so that tissue oxygen delivery remains close to sea-level values at rest and during submaximal work. By **2 to 3 weeks,** systemic and regional blood flow have returned toward sea-level values as oxygenation improves. However, sympathetic activity continues to increase and may reach extraordinary levels, particularly at higher altitudes (>4000 m).
  - Oxygen-carrying capacity of the blood increases as a result of the increase in hemoglobin and hematocrit: **early** (1 to 2 days) increases result from plasma volume reduction; **later** (weeks to months) increases result from increases in red cell mass. **This critical adaptation offsets reduction in oxygen availability, thereby restoring oxygen transport toward normal sea-level values.**
  - Peripheral uptake of oxygen by skeletal muscle is facilitated by increased capillary density, mitochondrial number, myoglobin concentration, and 2,3-diphosphoglycerate (2,3-DPG), though these local changes are not universal.
  - Buffer capacity of skeletal muscle may be increased.
  - Substrate utilization is altered: at the same absolute workload, fat utilization is decreased acutely, but increases over the course of acclimatization; glucose utilization increases at altitude and there is less carbohydrate oxidation, potentially sparing muscle glycogen. However, this outcome is

different in men than in women, and the final effect of altitude on substrate utilization depends on whether the same absolute or relative workload is being compared to sea level, whether the fuel utilization is corrected for energy expenditure, and whether or not weight loss has occurred.

- **For competitions at altitude**
  - **Acclimatization** is critical and clearly improves performance **at altitude.**
  - If possible, adequate time for acclimatization should be allowed to maximize performance at altitude (2 to 3 weeks).
  - If adequate time for acclimatization is not possible, anecdotal experience among athletes suggests that competing immediately on arrival at altitude may be best.
  - Recent data from authors suggest that for low-altitude competitions (500 to 2000 m), living at altitudes higher than competition may be worse than living at competition altitude, unless at least 3 weeks of acclimatization are possible.
- **For competitions at sea level**
  - Living at altitude and training at altitude has not been shown to improve performance at sea level.
  - **Living at altitude and training as close to sea level as possible (known as *living high–training low*) does improve sea-level performance.**
  - Performance is best immediately on return to sea level and remains high at least up to 3 weeks after return.
- Recreational athletes who hike, climb, or mountain bike but are not interested in athletic competition are also affected by hypoxia of altitude. For recreational athletes, **sea-level training** is effective at increasing ability to perform **at altitude.**

FAILURE OF ACCLIMATIZATION—HIGH-ALTITUDE ILLNESS AND OVERTRAINING

Acute Mountain Sickness

- With moderate or higher altitudes (>2000 m) and rapid ascent rates (>300 m sleeping altitude per day above 3000 m), maladaptive state called acute mountain sickness (AMS) may develop.
- Symptoms include headache, nausea, anorexia, fatigue, and difficulty in sleeping.
- Symptoms are usually mild and self-limited; rest and analgesics are sufficient treatment.
- No evidence that competitive athletes are at any greater risk of developing AMS than nonathletes, although exercise may exacerbate development of AMS, and physical activity should be reduced appropriately in symptomatic individuals.
- For patients who do not improve with rest, supplemental oxygen or descent to lower altitude virtually always results in prompt symptom relief.
- Other effective treatments include acetazolamide, dexamethasone, and simulated descent with portable hyperbaric bag.
- Problem is best prevented by limiting rate of ascent, allowing for rest or acclimatization days, maintaining adequate hydration, avoiding alcohol or sedatives during early acclimatization phase, and limiting training volume and intensity during first few days at altitude.
- Use of drugs to prevent AMS is discouraged in endurance athletes who are going to moderate altitude (below 3000 m) unless clear history of recurrent AMS is obtained.
- Drug most frequently used is acetazolamide, which may be effective at low dose (125 mg at night or twice daily). However, diuretics including acetazolamide are on the World Anti-Doping Agency banned list as masking agents. Dexamethasone is probably more potent, but also is banned as a steroid.

Severe High-Altitude Illness

- In some individuals, AMS may progress to or be associated with more severe and life-threatening forms, including high-altitude pulmonary (HAPE) or high altitude cerebral edemal (HACE).
- **HAPE** is characterized by **dyspnea at rest,** cyanosis, severe hypoxemia, and noncardiogenic pulmonary edema.
- **HACE** is characterized by vomiting, **ataxia,** reduction in level of consciousness, and, in some cases, frank coma.
- Both of these syndromes can result quickly in death. **Immediate descent is mandatory.** High-flow supplemental oxygen or portable hyperbaric bag, if available, may be useful adjunctive therapy while descending or if descent is delayed.
- Both HAPE and HACE are rare at moderate altitudes to which most athletes are exposed (<0.1%), though occurrence in athletes at low-moderate altitude (<2000 m) should initiate search for congenital abnormalities of the pulmonary circulation.
- Medications that lower pulmonary artery pressure may be used for adjunctive treatment of HAPE (this is less effective than descent and oxygen). Nifedipine has been most extensively studied and is effective both for treatment and prophylaxis. Phosphodiesterase inhibitors (e.g., sildenafil, tadalafil) are being investigated, but they may exacerbate AMS. Very recent studies suggest that dexamethasone is effective at preventing both AMS and HAPE, and is the most effective adjunctive therapy for HACE. Drug treatment should be considered in athletes only if oxygen is unavailable or descent/evacuation is delayed. A staged, slow ascent is the most effective preventive strategy and it sidesteps the need to use banned substances for prevention.

Overtraining

- Another potentially serious problem with training at altitude is increased risk of overtraining.
- The comparative relationship between exercise training and administration of medication shown in Figure 19-1 is helpful. Every medication has a specific dose-response relationship, accompanied by a toxic/therapeutic range. These parameters define optimal dose and frequency of administration to maximize benefit but minimize side effects and toxicity. Exercise can be conceived as medication: training response is proportional to volume and intensity (ED 50), but too much exercise results in the clear toxic effects of musculoskeletal injury and systemic effects of overtraining (LD 50).

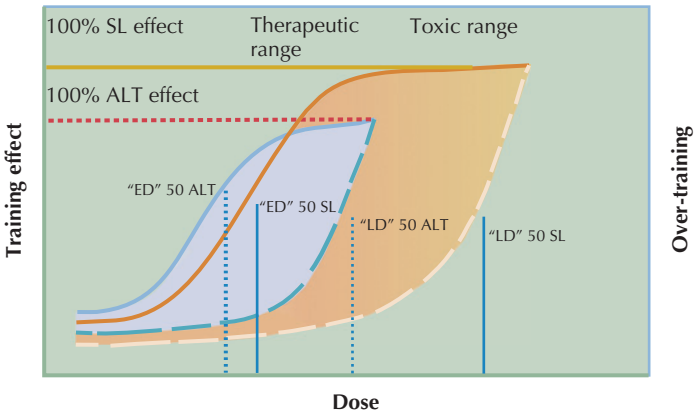

Figure 19-1 Toxic-therapeutic range of exercise training: the effect of high altitude. Although the mechanical stress of exercise is likely to be less at altitude because of the reduction in training speed, the metabolic stress may well be greater, at least with regard to the effect of hypoxia on the central nervous system. Thus, exercise training at altitude narrows the toxic/therapeutic range of exercise, possibly enhancing the training effect, but also increasing the risk of toxicity. SL = sea level, ALT = altitude, ED = effective dose at which 50% of the maximal response is achieved, LD = lethal dose at which 50% of the maximal toxicity is achieved.

- **Overtraining may be precipitated by:**
  - **Inappropriately hard workouts:** Base pace too fast because of narrowed training zones and/or athlete inexperience, intervals too hard—run at maximal speed rather than 105% race pace, recovery exercise too hard (i.e., no recovery pace).
  - **Inadequate recovery:** Dehydration, sleep disturbance, too short recovery times either between workouts or between intervals within hard workout.

PRACTICAL STRATEGIES FOR IMPLEMENTING ALTITUDE TRAINING

From a practical perspective, critical issues for athletes and coaches are **"indications"** for altitude training and, if indicated, for appropriate "dose" of altitude and training.

Indications

Which Athletes Should Use Altitude Training?

- Evidence is best for events in which $\dot{V}O_2$ max is important for performance (i.e., endurance athletes in events lasting longer than 2 to 3 minutes); sprint athletes may benefit from neuromuscular/kinesthetic training because of faster speeds allowed by reduced air resistance.
- Because supervised training camp is powerful stimulus for training, even in elite athletes, athletes who are already well trained with solid base and regular interval work are likely to obtain most specific benefit from altitude per se.
- **Altitude training is no substitute for a focused, well-designed training program with appropriate rest and nutrition.**
- Athletes who respond best to altitude training have robust and sustained increase in erythropoietin concentration at altitude and strong ability to maintain training speed and oxygen flux during interval training. These responses may be genetically determined.

Which Athletes Should Not Use Altitude Training?

- Team sports that depend more on strategy and technique for success (e.g., water polo, baseball, soccer, field hockey, handball) are unlikely to derive much benefit from altitude training for sea-level performance.
- Sports that depend on normal air resistance for fine motor skill (e.g., basketball, archery, tennis, soccer) are likely to be impaired by altitude training; such sports may require adjustment period both at altitude and on return to sea level to compensate for differences in projectile movement through air.
- Swimming is most controversial. Because of extremely low mechanical efficiency, the key to successful performance is **biomechanics** rather than **physiology.** Although many swimmers attempt to use altitude training, no evidence supports its benefit.
- Iron-deficient athletes should not engage in altitude training (see "Nutritional Factors" section).
- Athletes with small and brief increase in erythropoietin at altitude and those who cannot maintain training speeds even at low altitudes are least likely to respond well.

Dose

How high to live: Locations 2200 to 2700 m above sea level will maximize acclimatization and minimize complications (muscle wasting and AMS). Recent explosion of techniques to bring mountain to athlete involves sleeping in nitrogen-enriched environment to simulate hypoxia of high altitude. Molecular concentrators can regulate environments as small as a single bed (nitrogen tents) or as large as an entire dormitory (nitrogen houses).

How long to reside at altitude: Erythropoietin levels increase acutely, are clearly still elevated after 2 weeks, and return to sea-level values by 4 weeks. A minimum of 3 to 4 weeks appears necessary to develop sufficient acclimatization and augmented red cell mass, particularly for competition at sea level. Box 19-1 outlines a sample training camp based on a 4-week mesocycle. Some athletes use multiple shorter cycles, although no evidence indicates that altitude-specific effect persists for more than a few weeks after altitude training camp. For nitrogen house environments, additional questions include how long to spend in environment each night and how many nights per week. Australian researchers found no increase in hemoglobin/myoglobin mass after 10 days to 3 weeks at 8 to 10 hours daily, although they did

BOX 19-1 *Sample Training Camp Based on 4-Week Mesocycle*

Week 1 (acclimation week 2200–2500 m)
Mon. Base training
Tues. Base training
P.M. Base training
Wed. Base training
Thurs. Base training
Fri. Base training
Sat. Base training (or off)
Sun. Long run
0/1/8 (# hard/# long/# total workouts)
Week 2 (medium week)
Mon. Base training
Tues. 5 × 1000 m
105% 5-K race pace
P.M. Recovery run
Wed. Base training
Thurs. Base training
Fri. Easy run
Sat. 5-K road race
P.M. Recovery run
Sun. Base training
2/0/9
Week 3 (hard week)
Mon. Long run
Tues. Base training
Wed. 5 × 1000 m
105% 5-K race pace
P.M. Recovery run
Diet log
Thurs. Base training
P.M. Base training
Fri. Base training
P.M. Hill drills
Plyometrics
Sat. Base training
Sun. Long run
2/2/11
Week 4 (easy week)
Mon. Easy run
Tues. 5 × 1000 m
105% 5-K race pace
P.M. Recovery run
Wed. Base training
Thurs. Base training
Fri. Base training
Sat. Off
Sun. Long run
1/1/7

Mesocycle = period incorporated into athlete's training plan for year (macrocycle), which typically includes increasing volume and intensity, followed by period of rest and recovery.

report improvement in performance. Finnish and French investigators reported results similar to field environments with 14 to 16 hours daily, which include some easy exercise in hypoxic environment. Optimal dose of such environments has yet to be determined, though a minimum of 12 to 16 hours appears necessary. "Intermittent" hypoxic exposures ranging from a few minutes on and off, to up to 3 hours, of severe altitude are of no benefit for sea-level performance.

How high to train: Interval training: Definitely as low as possible, preferably below 1500 m. **Base training:** Possibly can be performed at altitude to minimize travel requirements in living high–training low approach. **Base training must be performed at a relatively easy pace to prevent overtraining. Careful monitoring of heart rate and/or blood lactate during training may help ensure correct training pace.**

When to compete on return to sea level: Immediately on return appears best, particularly if high-intensity training has been done low. Some reacclimatization may be necessary (2 to 3 weeks) to restore sea-level running speed and allow increased ventilation (and therefore respiratory muscle work) to return to baseline, particularly if interval training has been done at altitude. Although athletes often report "feeling better" after such reacclimatization periods, no clear evidence indicates that performance is correspondingly improved.

Nutritional Factors

- **Nutritional factors, particularly iron stores, play a critical role in the ability to respond to altitude training.**
- Many athletes (male and female) have reduced iron stores based on a **low serum ferritin.** Such athletes are unable to increase the volume of red cell mass (blood volume minus plasma volume) and do not increase $\dot{V}O_2$ max; thus, they are unable to obtain the potential benefits of altitude acclimatization.
- Simple measurement of hemoglobin or serum iron is inadequate because they do not reflect bone marrow iron stores. Because iron is also a critical moiety in myoglobin as well as mitochondrial cytochromes, iron deficiency may not only compromise oxygen carrying capacity, but also may inhibit oxygen extraction (arteriovenous O_2 difference) and reduce O_2 flux, thereby limiting $\dot{V}O_2$ max and performance, even in nonanemic athletes. **Iron stores (ferritin) must be normal before undertaking a period of altitude training.**
- High doses of oral iron (150 to 400 mg elemental iron daily in divided doses) are usually required to maintain ferritin levels during altitude training. Even athletes with normal iron stores at the start of an altitude training program experience rapid falls in serum ferritin at altitude and must be supplemented and monitored closely.
- Oral iron doses are best tolerated in liquid, pediatric preparation—Feosol, 15 mL dissolved in orange juice and mixed with 500 mg vitamin C, one to three times daily, ½ hour before or 1 to 2 hours after a meal. May use more frequent administration of smaller doses if gastrointestinal upset develops.

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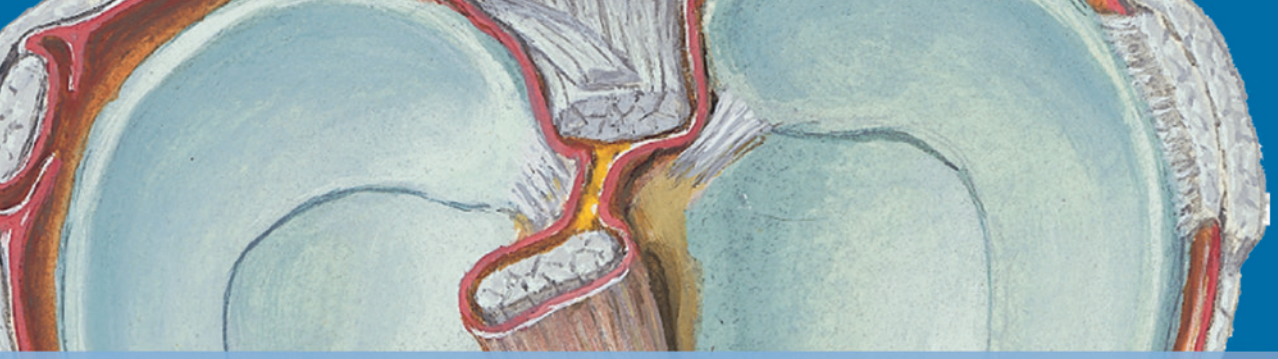

SECTION

V

Behavioral and Psychological Problems

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The Role of Sport Psychology and Psychiatry

20

David B. Coppel

WHAT IS SPORT PSYCHOLOGY (SP)?

Definitions

- The American Psychological Association (APA) Division 47 Exercise and Sport Psychology defines sport psychology as:
  - Helping athletes apply psychological principles to achieve improved or optimal sport performance and mental health.
  - Increasing knowledge regarding the impact of sport/exercise and physical activity on psychological development, health, and well-being over the lifespan.
- APA is expanding the definition of SP, as a result of its Sport Psychology Proficiency certification program, to include psychological skills, optimizing the well-being of athletes, dealing with organizational and systemic issues in sport settings, and understanding the social and developmental issues related to sports participation.
- **In a multidisciplinary context, SP is increasingly seen as a strong component of the sports medicine team.**
- Psychology has played a well-established and important role in physical medicine and rehabilitation programs over many years, so the application to the sports medicine context is a natural extension.
- Further evidence of the SP role in sport medicine is found in the recent Team Physician Consensus Statement published by the American College of Sports Medicine (ACSM): "Psychological Issues Related to Injury in the Athlete and the Team Physician."
- Although it is clear that physical injuries, illness, or disease can directly impact athletic performance and/or participation, psychological factors (broadly defined to include behavioral, emotional, or cognitive patterns, personality variables, developmental or adjustment issues, or diagnosable clinical issues) are seen as critically important to optimal athletic performance. Performance is often described as up to 80% mental, and thus, the acquisition and application of psychological skills relate strongly to a role for SP ("90% of the game is half mental"—Yogi Berra)
- Within the sport medicine team, SP is involved with psychological care and consultation with athletes at all levels of competition including professional, elite amateur, Olympic, collegiate, high school, and youth sports.
- Sport psychologists can use training and experience to provide a unique contribution to the sports medicine team in dealing with athletic injuries. SP can address factors (e.g., stress) that may influence the risk of injuries, as well as the emotional, cognitive, and behavioral factors related to becoming injured and dealing with recovery and rehabilitation (see Appendix A).

WHO IS A SPORT PSYCHOLOGIST?

Qualifications

Influence of history: SP has emerged from its roots in motor learning and kinesiology research and applications of clinical and counseling psychology principles to athletic settings. As a result, a range of sport psychology practitioners were created: from academicians and researchers in exercise and sport science (often housed in physical education departments) to applied/clinical psychologists experienced in athletic settings.

U.S. Olympic Committee: In the early 1980s, the U.S. Olympic Committee (USOC) created a Registry of Sport Psychologists, which was split into three categories: educational, clinical, and research. This three-part approach gave way to current and ongoing efforts by USOC sport psychologists to include only those practitioners who meet standards and certain criteria in their educational training and supervised practice in sport psychology. **USOC Registry** members are often involved in consulting with elite/Olympic athletes or teams or serve as regional referrals for inquiring athletes or teams.

Association for Applied Sport Psychology (AASP): Certified Consultant in Sport Psychology status emerged from this multidisciplinary organization oriented to sport science. AASP reviews and approves individual credentials, coursework, and experience, indicating that these consultants have met the designated standard in their educational training in sport science and in psychology; these consultants agree to support the AASP Ethical Code (which parallels the American Psychological Association's Ethical Code). Certified Consultants can provide information about the role of psychological factors in sport, exercise, and physical activity to individuals, groups, and organizations. Consultants can teach athletes specific mental, behavioral, psychosocial, and emotional control skills for sport, exercise, and physical activity. Consultants with clinical or counseling training are qualified to work with clinical disorders or issues emerging out of injury.

American Psychological Association Division 47 (Exercise and Sport Psychology): This division is involved in ongoing efforts at creating a standard for proficiency in sport psychology. This proficiency "encompasses training in the development and use of psychological skills for optimal performance of athletes, in the well-being of athletes, in the systemic issues associated with sports setting and organizations, and in developmental and social aspects of sports participation." The psychologists who qualify for the proficiency should have experiences dealing with theory and research in aspects of sport psychology, principles and practices of applied sport psychology (including issues and techniques of sport-specific psychological assessment and mental skills training for performance enhancement), clinical and counseling issues with athletes, organizational and systemic aspects of sport consulting, understanding of developmental and social issues related to sport participation, and knowledge of biobehavioral bases of sport and exercise (see www.apa47.org).

Importance of licensure: For clinical issues and/or emotional disorders in athletes, a referral to a licensed mental health provider is essential; a referral to a provider with experience working with athletes is optimal. Other providers/practitioners may offer "sport psychology consultation," focusing on mental skills training or performance enhancement. Qualified clinical psychologists may also provide performance enhancement consultation. The title of "Psychologist" is typically one that requires licensure by states, so those identifying themselves as a "Sport Psychologist," should be licensed **and** have competency (education and training) in the field of sport and performance psychology.

PSYCHOLOGICAL HEALTH ISSUES IN ATHLETES

Clinical Concerns

- Clinical concerns include anxiety, depression, stress reactions, adjustment reactions, phobias, substance abuse, eating disorders, and burnout. All of these problem areas can be associated with performance decrements.

- **Athletes are not immune from mental health problems.** However, it has been shown that physical activity and organized sports participation decreases depression.
- **Psychological treatment from a licensed mental health professional (and possibly psychotropic medication evaluation) is usually indicated for clinical level issues; for these athletes, finding a licensed mental health professional with sport psychology experience is preferable (e.g., clinical psychologist/sport psychologist or sport psychiatrist).**

Mood Disorders

Mood disorders involve significant deviation from normal mood states with significant cognitive, behavioral, and physical symptoms that usually impact daily functioning and performance.

Major Depression

- **Major depression** has diagnostic criteria (*Diagnostic and Statistical Manual of Mental Disorders*, 4th ed.—DSM-IV) that require 2 weeks of psychological or psychosocial symptoms such as the following (Fig. 20-1):
  - Depressed mood/dysphoria
  - Feelings of guilt
  - Thoughts of death or suicidal ideation
  - Social withdrawal
  - Loss of pleasure or interest
  - Feelings of worthlessness
  - Low self-esteem
  - Hopelessness
  - Helplessness
  - Indecisiveness
  - Sleep disturbance (insomnia or hypersomnia)
  - Change in appetite or weight
  - Change in energy level or fatigue
  - Attention/concentration problems
  - Restlessness or psychomotor slowing
- Diagnostically, these symptoms do not emerge within the context of bereavement or grief, diagnosed medical illness, medication reaction or substance abuse; however, it is noted that depression can accompany these issues. Depressive feelings, and even clinical depression, can emerge following injury or when recovery/rehabilitation is slower than expected.

Dysthymia

- Diagnostic criteria for dysthymia require:
  - At least 2 years of symptoms, having more days of depressed mood than not depressed.
  - Symptoms include at least two of the following: hopelessness, helplessness, problems in concentration, indecisiveness, appetite loss or overeating, low energy or fatigue, low self-esteem, sleep disturbance.
- Individuals are often seen as chronically unhappy, irritable, and pessimistic. Athletes with dysthymia may not self-report having depression, but may be ruminative, pessimistic, and not seem to be enjoying their athletic involvement (see Fig. 20-1).

Bipolar Disorder

- Bipolar disorder can involve both depressive episodes and manic or hypomanic episodes, and often extreme affective dysregulation. **Manic symptoms** can include:
  - Abnormal or excessive elation
  - Unusual irritability

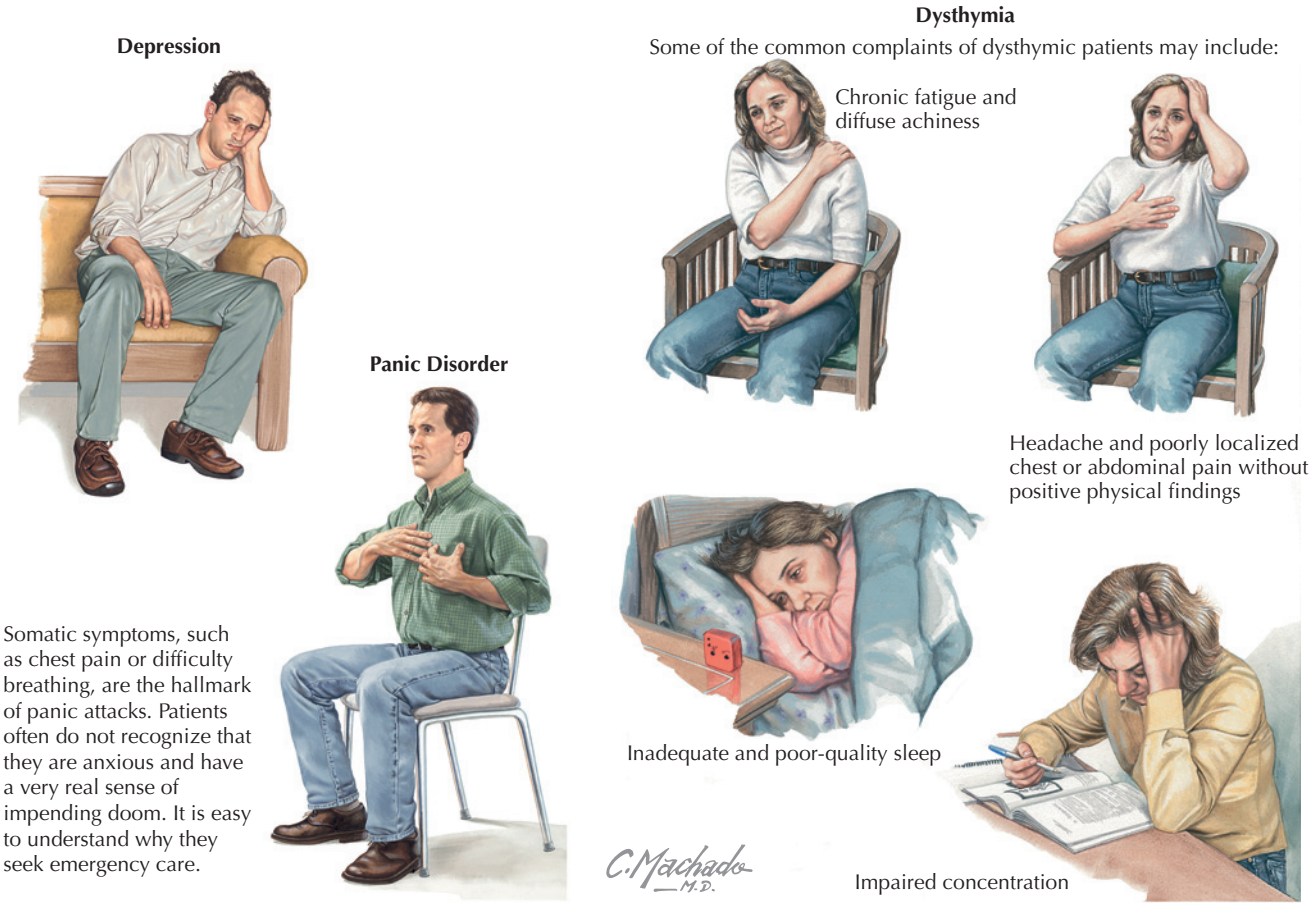

Figure 20-1 Common Psychological Disorders.

  - Decreased need for sleep
  - Grandiose notions
  - Increased talking
  - Racing thoughts
  - Increased sexual desire
  - Greatly increased energy
  - Poor judgment
  - Inappropriate social behavior
  - **Hypomanic episodes** are milder versions of manic episodes.
- Bipolar disorder may present in athletes as an extreme behavioral change (in a previously well-functioning person) or as self-destructive or violent behavior (and may involve substance use/abuse). Psychological treatment with a licensed mental health professional, such as a clinical psychologist, and/or medication evaluation and management by a psychiatrist, are recommended.

Anxiety Disorders

- Anxiety disorders involve intense apprehension, general fearfulness, fear of losing control, associated physical symptoms such as palpitations, tremors, and mental and physical avoidance efforts (worry and avoidance of people, places, or things).
- Efforts at avoidance are initiated to reduce the anxiety, but often create significant life disruption.

Panic Disorder

- Panic disorder involves periods of severe fear or discomfort with significant concomitant physical symptoms such as palpitations, dizziness, nausea, lightheadedness, fear of losing control or going crazy, numbness, or tingling. These experiences often result in emergency room visits for suspected cardiac problems; subsequently, individuals focus on panic sensations, or the possibility of having a panic attack, and become anxious about being anxious ("what if…") (see Fig. 20-1).
- Athletes with panic disorder often have difficulty maintaining focus or concentration because of the greater focus on anxiety/panic symptoms and sensations; they may also display reduced energy and motivation. Sensations and fear of panic attacks are associated with certain conditions, which generalize to an expanding set of conditions.
- Behavioral avoidance may occur as the person tries to prevent further attacks, which, in the extreme, creates only a small "safe zone" (agoraphobia) for the individual.
- It should be noted that a vast majority of individuals with panic disorder report a life stress event as being causative (e.g., death of significant family member, relationship breakup, job loss, illness or injury).

Generalized Anxiety Disorder (GAD)

- Generalized anxiety disorder (GAD) usually involves long-standing anxiety symptoms, such as excessive worry, which can result in sleep disturbance, restlessness, and general muscle tension.
- Typically individuals with GAD ruminate and worry excessively in multiple (if not all) spheres of their life (sports, school, work, health, family, relationships).
- Vigilance to threats in the environment is thought to reduce threat or bad things happening to the individual; the perceived success (no bad things occurring) reinforces the approach.
- For athletes, subclinical worry may also interfere with athletic performance, but it is less severe and interfering and does not extend to other areas.

Social Anxiety Disorder (SAD)

- Social anxiety disorder (SAD) is often called "social phobia" and involves fear of evaluation in social situation.
- Individuals are concerned about, and fear, poor or inadequate performance and being negatively scrutinized, which can include athletic competitive situations, social interactions, public speaking, or other performance/outcome situations. This fear of negative outcome/evaluation can lead to avoidance behavior, which in turn reduces general functioning and reinforces negative self-perceptions.
- Many athletes may describe competitive anxiety, most often precompetitively, but this does not rise to the intensity of symptoms found in SAD; SAD athletes may not be able to refocus after distraction, and have a greater negative response to real or perceived performance failures.
- Athletes with significant anxiety complaints and/or symptoms should have a thorough assessment regarding the degree and pervasiveness of their negative evaluation focus (usually a clinical interview and/or anxiety questionnaires or inventories).

Obsessive-Compulsive Disorder (OCD)

- Obsessive-compulsive disorder (OCD) involves the intrusion of recurrent obsessions (thoughts, ideas, images, or impulses) or compulsions into daily functioning, creating significant difficulties and anxiety; individuals engage in compulsive behaviors (often repetitive) that serve to reduce anxiety and distress.
- The blockage of the compulsive behavior results in increased anxiety and decreased functioning.
- Athletes will often have precompetitive routines or rituals, which generally do not rise to the level of OCD; however, if athletes associate successful performance with only a narrow set of behaviors, it could create a negative impact on general functioning.

Posttraumatic Stress Disorder (PTSD)

- Posttraumatic stress disorder (PTSD) involves a group of symptoms emerging as an intense emotional response to an event that involved actual or possible death or injury (or involved witnessing an event).
- Symptoms include re-experiencing symptoms from the event from flashbacks or trigger cues, avoidance of all cues associated with the trauma and emotional detachment, physiologic hyperarousal (vigilance, extreme startle response, decreased attention and concentration). These symptom clusters often impair interpersonal relationships.

Acute Stress Disorder (ASD)

- For some athletes, a severe or career-ending injury may generate PTSD-like symptoms, but the symptoms may be a better diagnostic fit for acute stress disorder (ASD), which involves exposure to a traumatic event that involves serious injury, actual or threatened death, or threat to physical integrity of self or others.
- Also required are three or more of the symptoms of numbness or detachment or lack of emotion, being dazed or somewhat unaware of current circumstances, derealization, depersonalization, poor recall of the trauma.
- ASD involves recurrent thoughts/re-experiencing of the trauma and some avoidance behaviors.
- Symptoms must emerge within 4 weeks of trauma and must last at least 2 days but no longer than 4 weeks.

Eating Disorders

- There are three diagnostic categories for eating disorders: Bulimia Nervosa, Anorexia Nervosa, and Eating Disorder Not Otherwise Specified (see Chapter 22 for a detailed review of eating disorders).
- Clinical sport psychologists with training and experience in eating disorders may be helpful in assessment, diagnosis, and treatment of athletes with eating disorders or disordered eating.

- Education of athletes regarding eating disorders can be helpful, but understand that athlete culture may help camouflage the eating issues and/or be offering mixed messages.
- In many, if not most cases, depression or anxiety symptoms coexist with eating disorder behavior (e.g., through issues of control and guilt or negative perfectionism).
- It is desirable to identify practitioners with expertise in eating disorders so that referrals can be easily made if indicated. A sport medical physician can be a crucial member of the multidisciplinary team that includes athletic trainer, psychiatrist, and therapist.
- The National Collegiate Athletic Association (NCAA) provides a useful handbook for team physicians, athletes, and coaches *(Managing Student Athletes' Mental Health Issues)*. The Female Athlete Triad position paper (American College of Sports Medicine, 2007) contains important information.

Attention Deficit Hyperactivity Disorder (ADHD)

- Attention Deficit Hyperactivity Disorder (ADHD) involves symptoms related to two clusters: inattention/disorganization and hyperactivity and impulsivity.
- Some degree of impairment must occur before the age of 7, in multiple settings (e.g., school, work, and home).
- It should be noted that ADHD is comorbid with other adolescent disorders (conduct or oppositional-defiant disorders), or incipient mood, anxiety, and substance abuse disorder; therefore, ADHD diagnoses should be made after thorough assessment efforts.
- Common symptoms in childhood include difficulty concentrating, difficulty sitting still, excessive talking, impulsive behavior, general restlessness.
- Although most individuals experience a decline in ADHD symptoms through adulthood, significant number of adults describe ongoing problems with focus, concentration, organization, impulsive and/or experience problems in the workplace or within interpersonal relationships.
- **Assessment** should include a clinical interview and detailed history regarding medical, academic, and family history. Some interview protocols include ratings of behavior from parents and teachers. Psychological and/or neuropsychological testing may provide further data regarding the cognitive status of the individual and help in the differential diagnosis effort.
- **Attentional difficulties, or problems with impulsivity, focus, and follow-through, usually create problems within the competitive sports environment. However, an ADHD diagnosis should not be made on inference only (poor performance or mistakes are attributed to inattention or poor concentration) or self-reported attention problems or impulsive behavior; as noted above, thorough and careful diagnostic evaluation of history and other factors should be completed before an ADHD diagnosis is made.**
- Athletes with ADHD may show inattentiveness, distractibility, or poor focus and referral to a clinical sport psychologist may be an initial response; sometimes, these athletes are thought to have problems in motivation or attitude because of their inattentive behavior or errors in practice or games, or their impulsivity or difficulty following rules. Interpersonal issues may arise because frustration tolerance may be low. These athletes may respond positively to a structured sports environment, which helps to reduce distraction.
- Primary treatment options for ADHD can include behavioral interventions and medications. Psychiatrists, or sports psychiatrists, are able to provide medication evaluation for athletes, and monitor medication effectiveness over time and potential negative side effects.
- Many of the medications used to treat ADHD, such as Adderall, are banned by the NCAA except as medically indicated and their use can result in a positive drug test result. Because of this, new NCAA rules mandate that athletes using stimulant medications such as Adderall for the treatment of ADHD provide documentation regarding the diagnosis (including tests performed to make the diagnosis of ADHD), medication dose, and treatment regimen by the treating physician as part of their records.

Anger Control and Impulse Control Issues

- Anger control and impulse control issues are usually described as aspects of other disorders, such as mood or anxiety disorders or general impulse control problems (e.g., pathological gambling).
- Generally, anger control issues emerge when the aggressive response (verbal or behavioral) is thought to be out of proportion to the nature of the event; some individuals experience guilt or remorse following their behavior and its consequences.
- Anger dyscontrol may be associated with aggressive behavior, history of family violence, substance abuse, and with certain medical conditions.
- Athletes may display problems with frustration and anger control, with a range of frequency observed. If these behavior problems are occurring at significant frequency, evaluating whether it is part of a larger dyscontrol pattern is important. It is crucial that the sport psychologist or other provider take time to distinguish between competitive intensity/anger and anger control problems.
- In some instances, the athletic environment rewards aggressiveness (usually seen as controlled aggression), because it may be associated with athletic success. Some athletes, who may be predisposed to anger, may respond well to these rewards, but have difficulty with controlling or compartmentalizing their impulses to appropriate levels or venues (i.e., they exhibit the behaviors in nonathletic circumstances).

Personality Disorders

- Personality disorders involve personality or behavior traits that endure over time and are maladaptive, causing significant subjective distress and/or observable functional impairment in major areas of life (school, work, or family).
- Individuals are not usually diagnosed with personality disorder until age 18.
- Disorders are grouped in three clusters:
  - **Cluster A** includes schizoid, paranoid, and schizotypal personality disorders, which are characterized by atypical interpersonal behavior, and often odd interaction styles and perceptions of the world.
  - **Cluster B** includes histrionic, borderline, narcissistic, and antisocial personality disorders, which are characterized by affective and behavioral deregulation.
  - **Cluster C** includes avoidant, obsessive-compulsive, and dependent personality disorders, which are characterized by anxiety and avoidant behavior symptoms.
- These disorders or their subclinical variants (traits or features that are present but do not meet diagnostic criteria) may be seen in athletes who have repeated interpersonal difficulties with teammates or staff, problems with authority or complying with rules, disciplinary problems, aggression or emotional dyscontrol, or problems accepting responsibilities. Comorbid problems include substance abuse, anger, aggression, and mood issues.
- Clinical sport psychologists can be helpful in assessment of these disorders or the general traits involved. Cognitive behavioral approaches have been helpful in reducing symptoms

and enhancing functional levels; these chronic disorders may fluctuate but their complexity usually suggests long-term therapeutic interventions.

PSYCHOLOGICAL ASPECTS OF INJURY

- **Psychological aspects of injury can be an important area in which a sport psychologist makes significant contributions to the sports medicine team. A sport psychologist can offer services/consultation to athletes dealing with the cognitive, emotional, and behavioral responses to being injured and to the recovery/rehab process.**
- There may be certain stages for physical recovery that are common from a medical standpoint; **however, psychological stages in coping with injury are often much more individualized and influenced by other factors (e.g., social support, impact on team or career, or general personality traits).**
- **Referral for psychological consultation has been associated with enhancing physical recovery.**
- **Psychological components of injury and recovery can include shock and emotional disorganization immediately postinjury; general anxiety and uncertainty about the future during evaluation and treatment decision making; impatience, anger, frustration, lack of control, and depression during recovery.**
- **Psychological issues can occur in the return-to-play phase, involving fear of reinjury or of not being at preinjury level of performance. Also, self-doubt and rumination can emerge, producing a larger issue of decreased confidence.**
- Psychological factors, such as stressful life events, may contribute to the risk of athletic injuries. Personality factors have not been found to reliably relate to risk of athletic injury. **Athletes with high levels of stress, low or ineffective coping skills, and low social support may be at particular risk for injury; these athletes may also be at risk for a problematic injury response and recovery course.**
- Athletes make efforts to deal with being injured, in individualized ways and time frames. Emotional responses can include sadness, feelings of isolation, irritation, frustration, anger, as well as changes in sleep, appetite, and energy (fatigue). These symptoms can worsen and emerge as problems for the recovery/rehab course; depression is the most common response; it can progress from sadness to more clinical level depression.
- **It should be noted that athletes often use their training/exercise and general physical activity as a coping mechanism and outlet for dealing with stress; injury can reduce access to this coping strategy and thus add to the emotional stress.**
- Signs of poor adjustment to injury include exaggerated or generalized fear of reinjury, general impatience or irritability, mood swings, withdrawal from significant others or social network, rumination about guilt for being injured and letting others down, increased somatic focus, obsession with return to play, general pessimism about the future in sports and other areas (demoralization), or apathy.
- **Watch for athletes sliding (or falling) down the "D Slide."** This involves athletes beginning with feeling some **disappointment** about the injury or being injured; this is a normal range experience and involves the thought that, "I can't do it and I should be able to." If disappointment is not dealt with it can progress to feeling **devastated,** which involves the generalized thought, "I can't do anything." If devastation is not responded to, the slide continues to feeling **defeated** ("I'm helpless to do anything [to change it]"). The low point on the slide is when an athlete feels **defective** and thinks, "There's something wrong with me."
- Injured athletes who are strongly identified with their sport may experience a greater sense of identity loss. Separation and loneliness (from the team) are experiences that can increase the probability of problematic emotional responses to injury. Maintaining contact with teammates reduces the sense of isolation and gives the athlete opportunities for support.
- **Return-to-play issues** include not only physical readiness and clearance, but also **psychological readiness.** Assessment of psychological readiness is crucial to successful return to play. Psychological readiness information can be obtained from a sport psychologist (if involved with player or team), trainers, and other support network members.
- For further discussion of the importance of psychological issues related to injury in athletes, please refer to the ACSM Team Physician Consensus Statement (see Recommended Readings).

SPORT PSYCHOLOGIST ROLES

- **A psychological skills training consultant** typically educates athletes or teams about mental skills that enhance performance. These skills include goal setting, relaxation, imagery or visualization, positive self-talk, arousal regulation, increased concentration/focus, precompetitive routine/mental preparation, adaptability/handling pressure, time management, general communication skills. Team consultation can enhance communication and cohesion among athletes.
- **A clinical sport psychologist** can provide assessment and interventions to athletes dealing with psychological health issues that may have performance implications or clinical level consequences. Sport psychologists may provide services as a team clinician or consultant:
  - Assessment can involve classification or diagnosis of presenting issue, as well as identifying etiology or contributing factors.
  - Assessment can involve acquiring a specific and detailed understanding of personal and sport issues or problems.
  - Assessment techniques can include interview, behavioral observation, psychological testing, neuropsychological testing, and completion of inventories or questionnaires.
  - Assessment involves integration of the athlete's predisposing factors, precipitating factors, and current maintenance factors.
- **A specialized role for a clinical sport psychologist** with training and experience as a neuropsychologist involves evaluation and consultation regarding sport concussion. This role involves the evaluation of neurocognitive, emotional, and reported physical symptoms following concussion. **Sport neuropsychologists will use neuropsychological testing to assess cognitive status and provide input to team physicians and/or the sport medicine team (see Chapter 39, Head Injuries). Sport neuropsychologists can evaluate the often overlooked emotional symptoms of concussion.**

PERFORMANCE ENHANCEMENT

- Athletes can be referred to a sport psychologist for help in developing mental skills to improve performance. **It should be seen as adding or improving skills, not as a need to be fixed.**
- **Areas for sport psychology consultation (7 C's):**
  - **Commitment** issues deal with motivational concerns (e.g., burnout or mental fatigue) and may involve developing goal-setting skills.
  - **Control** skills are usually important for athletes, who must have control over their body and movements and their minds. As athletes progress upward in competitive level, the importance of control over thoughts, emotions, and reactions becomes increasingly important. Athletes must learn

to handle pressure (arousal control) and develop relaxation skills, in order to find the level of arousal associated with optimal performance. Visualization or imagery skills are useful in improving control and focus.
  - **Concentration** is crucial for successful performance. This skill involves not only the initial focus, but also the ability to refocus and deal with inevitable distractions. Focus on task-relevant skills is important for optimal performance.
  - **Confidence** is a factor that athletes (and coaches) describe as primary in successful athletic performance. Confidence is an expectation of success. It has connections to optimism, belief in self, and positive self-talk. Some athletes focus on having confidence regarding outcomes, whereas others focus on having confidence about being able to put forth effort and concentrate on the process (what they need to do). Slumps are examples of a loss of confidence, an issue that can benefit from sport psychology consultation.
  - **Communication** skills can be described as both interpersonal and intrapersonal. Sport psychologists can consult with teams to improve communication among teammates or between athlete and coach (interpersonal). More often, sport psychologists help individual athletes with how they communicate with themselves (intrapersonal). Developing positive and adaptive self-talk is associated with successful performance.
  - **Consistency** skills have to do with developing consistent effort in practice and games, and developing precompetitive routines and mental preparation skills. Precompetitive routines help build confidence in athletes by producing a reliable and predictable response to the behavioral sequence.
  - **Competence** skills have to do with helping athletes see themselves as competent and capable competitors and people. They can identify with the qualities of resiliency and flexibility. Self-statements such as, "I can cope. I can handle it," are part of having competency skills. **Competency overlaps with confidence, but is more reflective of self-efficacy (knowing what you need to do and when to do it and believing you can do it).**
- Athletes also need **courage and coping** skills, especially when dealing with injuries.

SPORTS PSYCHIATRISTS

- Sports psychiatrists provide support to the sports medicine team and team physician for psychiatric consultation.
- Sports psychiatrists may provide medication evaluation and treatment as an adjunct to sport psychology service.
- Sports psychiatry services can be helpful for a wide variety of conditions, including depression, anxiety, eating disorders, substance abuse, and attention deficit disorder.
- **The International Society for Sport Psychiatry (ISSP)** is an organization that applies the practice of psychiatry to the world of sports. ISSP members are described as consulting to high school, college, and professional teams and to individual athletes.
- Sport psychiatrists describe themselves as using the biopsychosocial model, which makes them able to diagnose underlying disorders and treat them with psychotherapy and medication. Some sport psychiatrists may also be trained in cognitive-behavioral techniques.
- Sport psychiatry issues include aggression in sports, "achievement by proxy (Tofler)," depression and suicide in athletes, body dysmorphic disorder, and steroid and/or substance abuse in athletes.
- Sport psychiatry providers advocate for a significant role in sports medicine clinics and describe outpatient consultation and liaison model.

SUMMARY

- Sport psychologists provide a range of educative and consultative services to individual athletes and teams for a variety of psychological health issues; they can also provide psychological skill training related to performance enhancement.
- Inclusion of a clinical sport psychologist on the sports medicine team is thought to provide an excellent opportunity to address the psychological aspects of performance, adjustment, and injuries.
- If a clinical sport psychologist is not a part of the sport medicine team, physicians should use sport psychology (or sport psychiatry) referrals to appropriately trained (and licensed) providers in the community.
- Sport psychology can make a particularly useful contribution to the team physician and sports medicine team in dealing with injured athletes.

RECOMMENDED READINGS

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Drugs and Doping in Athletes

21

Gary A. Green, David Z. Frankel, and James C. Puffer

DEFINITION

- According to the World Anti-Doping Agency (WADA) Code, doping is defined as the occurrence of one or more of the following anti-doping rule violations:
  - The presence of a prohibited substance or its metabolites or markers in an athlete's bodily specimen.
  - Use or attempted use of a prohibited substance or prohibited method.
  - Refusing, or failing without compelling justification, to submit to sample collection after notification as authorized in applicable anti-doping rules or otherwise evading sample collection.
  - Violation of applicable requirements regarding athlete availability regarding out-of-competition testing including failure to provide required whereabouts information and missed tests, which are declared based on reasonable rules.
  - Tampering, or attempting to tamper, with any part of doping control.
  - Possession of prohibited substances and methods.
  - Trafficking in any prohibited substance or prohibited method.
  - Administration or attempted administration of a prohibited substance or prohibited method to any athlete, or assisting, encouraging, aiding, abetting, covering up or any other type of complicity involving an anti-doping rule violation or any attempted violation.

SCOPE OF PROBLEM

- In 1964 Tokyo became the first Olympiad to conduct a trial of nonpunitive drug testing. Foreign substances were found in several athletes, which led to the establishment of a banned substance list, which has been modified and expanded repeatedly (Box 21-1).
  - A substance or method shall be considered for inclusion on the prohibited list if WADA determines that the substance meets two of the following three criteria:
    - Medical or other scientific evidence, pharmacological effect, or experience that the substance or method has the potential to enhance or enhances sport performance.
    - Medical or other scientific evidence, pharmacological effect, or experience that the use of the substance or method represents an actual or potential health risk to the athlete.
    - WADA's determination that the use of the substance or method violates the spirit of sport.
  - A substance or method shall also be included on the prohibited list if WADA determines that there is medical or other scientific evidence, pharmacological effect, or experience that the substance or method has the potential to mask the use of other prohibited substances and prohibited methods.
- In 1968, formal drug testing was adopted for the Summer and Winter Olympics. Drug testing has been used at every Olympiad since.
- In 1985, the NCAA began a series of quadrennial surveys that documented substance use/abuse and abuse patterns by intercollegiate athletes. The methodology changed significantly in 1997 and increased the number of participating schools and subjects. The sixth iteration in 2005 measured the substance-use patterns in 19,676 male and female athletes (Table 21-1). Periodic surveys are necessary to assess patterns of use of recreational and ergogenic substances.
- The International Olympic Committee (IOC) convened the World Conference on Doping and Sport in February 1999, which led to the creation of the World Anti-Doping Agency (WADA). WADA was charged with developing standards and a consistent, worldwide doping-control program. In addition, each country founded its own anti-doping agency (e.g., United

BOX 21-1 *Banned Substance List*

Prohibited substances
Anabolic agents
  Anabolic androgenic steroids (AAS)
    Exogenous
    Endogenous
  Other anabolic agents (e.g., clenbuterol, zeranol, zilpaterol, tibolone)
Hormones and related substances
  Erythropoietin (EPO)
  Growth hormone (hGH), insulin-like growth factors (e.g., IGF-1), mechano growth factors (MGFs)
  Gonadotrophins (LH, hCG) (prohibited in males only)
  Insulin
  Corticotrophins
Beta-2 agonists
Hormone antagonists and modulators
  Aromatase inhibitors
  Selective estrogen receptor modulators
  Other antiestrogenic substances and agents modifying myostatin function(s)
Diuretics and other masking agents
Stimulants
Narcotics
Cannabinoids
Glucocorticosteroids
Prohibited methods
Enhancement of oxygen transfer
Chemical and physical manipulation
Gene doping
Substances prohibited in particular sports
Alcohol
Beta-blockers

Data from The World Anti-Doping Code, The 2009 Prohibited List, International Standard.

Table 21-1 DRUG USE BY INTERCOLLEGIATE ATHLETES IN PAST 12 MONTHS

	Percent of athletes using	
Drug	**2001**	**2005**
Anabolic steroids	1.1	1.2
Spit tobacco	22.5	16.3
Alcohol	80.5	76.9
Ephedrine	3.5	2.5
Amphetamines	3.1	4.1
Marijuana	28.4	20.3
Psychedelics	5.6	2.5
Cocaine	2.1	
Ecstasy	N/A	1.2

Data from Green GA, Uryasz FD, Petr TA, Bray CD: NCAA study of substance use and abuse habits of college student-athletes. Clin J Sports Med 11:51-56, 2001; National Collegiate Athletics Association: NCAA Study of Substance Use of College Student Athletes. Indianapolis, Ind: NCAA, 2006.

States Anti-Doping Agency [USADA]) to ensure compliance. WADA is composed and funded equally by the sports movement and governments of the world.
- Beginning in 2003, the Bay Area Lab Cooperative (BALCO) has been at the center of a scandal in sports involving the company's role in developing tetrahydrogestrinone (THG), or "The Clear," and allegedly supplying it to U.S. professional athletes as well as U.S. and international Olympic athletes.
- Regarding the prevalence of substance abuse by athletes, studies reveal conflicting data as to whether athletes are more or less at risk than their nonathlete peers. These studies are complicated by lack of adequate controls, age groups, and appropriate definition of who is an athlete.
- Several independent studies focus on National Collegiate Athletic Association (NCAA) athletes, but methodologies vary widely, making comparisons difficult.
- **Sports pharmacology**
  - Sports pharmacology classifies drugs according to their reason for use, rather than chemical structure, mechanism of action, or pharmacological effects—a drug is classified as ergogenic, recreational, or therapeutic depending on the main reason why the athlete has chosen to use it.
    - Ergogenic drugs are substances that are taken specifically to increase performance.
    - Recreational drugs are used by athletes for the same reasons as nonathletes and carry the risk for addiction.
    - Therapeutic drugs are taken to treat an underlying condition.
- **The physician's role**
  - The often complicated relationship between physicians and drugs in sport has a long historical legacy.
    - Since the ancient Greek Olympics, athletes have sought out physicians to provide performance aids.
    - A 2005 NCAA study found that 13% of anabolic steroid users named a physician as a source of their drugs.
  - Physicians play a central role in the use and abuse of drugs by athletes.
    - Physicians are asked to provide education, act as medical directors or medical review officers for drug testing, and provide therapeutic use exemptions (TUEs).
    - It is imperative that physicians who serve as team physicians are familiar with performance-enhancing drugs used by athletes and also with the procedures used to determine a positive test result.

ANABOLIC-ANDROGENIC STEROIDS (AAS)

Definition

Anabolic-androgenic steroids (AAS) are synthetic drugs that mimic the effects of testosterone; they have both anabolic effects (e.g., increase protein synthesis) and androgenic effects (e.g., enhance the development of male secondary sexual characteristics).

Prevalence

- The 2005 "Monitoring the Future" study reported that 1.5% of 12th graders had used AAS in the previous 12 months. Since 1989, the rate has been between 1% and 2.5%.
- A 2005 NCAA survey revealed that 1.2% of male and female athletes had used AAS in the past 12 months. (Tables 21-2 and 21-3 present additional data from the NCAA survey.)
- The androgenization program developed by the former German Democratic Republic (GDR) provided anecdotal information that supported the efficacy of AAS and described adverse effects.
- Current estimates indicate that there are as many as 3 million AAS users in the United States and that 2.7% to 2.9% of young American adults have taken AAS at least once in their lives.

Table 21-2 COLLEGE ATHLETES' INITIAL USE OF ANABOLIC STEROIDS

Response category	Percent	
	2001	2005
Junior high or before	27	15
High school	25	40
Freshman year of college	19	15
After freshman year of college	29	30

Data from Green GA, Uryasz FD, Petr TA, Bray CD: NCAA study of substance use and abuse habits of college student-athletes. Clin J Sports Med 11:51-56, 2001; National Collegiate Athletics Association: NCAA Study of Substance Use of College Student Athletes. Indianapolis, Ind: NCAA, 2006.

Table 21-3 MAIN REASONS FOR COLLEGE STUDENT-ATHLETES' USE OF ANABOLIC STEROIDS

Reason	Percent	
	2001	2005
To improve athletic performance	47	44
For sports-related injury	28	19
For non–sports-related injury or illness	23	8
To prevent injury	2	6
To improve physical appearance	N/A	12
For weight loss/weight gain	N/A	11

Data from Green GA, Uryasz FD, Petr TA, Bray CD: NCAA study of substance use and abuse habits of college student-athletes. Clin J Sports Med 11:51-56, 2001; National Collegiate Athletics Association: NCAA Study of Substance Use of College Student Athletes. Indianapolis, Ind: NCAA, 2006.

- Parkinson and Evans surveyed 500 AAS users and found that 78.4% were noncompetitive bodybuilders and nonathletes who use these drugs for cosmetic purposes rather than to enhance sports performance.

Mechanism of Action

- AAS are bound by cytoplasmic proteins and transported to nucleus. Activation of DNA-dependent RNA polymerase results in production of messenger RNA for protein synthesis.
- Muscle size increases in AAS users through hypertrophy and formation of new muscle fibers. As reported by Kadi and colleagues, muscle biopsies suggest that use of AAS enhances activation of satellite cells and contributes to muscle fiber growth.
- AAS also may have an anticatabolic effect by attenuating the effects of cortisol. Haupt proposes that AAS displace cortisol from receptors, allowing an athlete to train at a high level. He also suggests that AAS increase motivation through heightened aggressiveness.

In Vivo Studies of Athletes and Anabolic Steroids

- Numerous studies of anabolic steroid use by male athletes have produced conflicting results. Some support improvement in strength, whereas others found no significant improvement in strength. Review of these studies by the American College of Sports Medicine (ACSM) has led to the following official position on the use of anabolic steroids:
  - Use of steroids by athletes is contrary to the rules and ethical principles of athletic competition.
  - With adequate diet, AAS can contribute to increases in body weight and lean mass.
  - Gains in muscular strength achieved through steroid use at doses beyond those used in clinical medicine improve performance and seem to increase aerobic power or capacity

for muscular exercise, giving an unfair advantage to those who are willing to risk the potential side effects to achieve gains in athletic performance.
  - Steroids have been associated with adverse side effects in therapeutic trials and in limited research on athletes.
- A well-conducted study by Bhasin and colleagues in 1996 demonstrated an increase in fat-free mass and muscle size and strength in weightlifters using weekly injections of 600 mg testosterone enanthate for 10 weeks. This was the first study to demonstrate that supraphysiologic doses of testosterone, combined with resistance training, can increase strength.

Recent Designer Steroids

- Norbolethone
  - Originally documented 30 years ago; isolated in two urine samples from a female athlete in 2002 who later received a lifetime ban.
  - Never commercially marketed because of possible toxic effects in animal studies and/or reports of menstrual irregularities.
  - Anabolic activity 20 times higher than its androgenic activity, thus very attractive to athletes looking to gain a competitive edge.
- Tetrahydrogestrinone (THG)
  - Unlike norbolethone, THG is a new chemical entity and would have remained undetectable if a syringe containing it had not been anonymously sent to USADA in 2003.
  - Closely related to gestrinone, a progestin, and to trenbolone, a veterinary androgen.
  - Very little information about the safety and anabolic effects of THG; has been found to be a potent androgen.
  - Several Olympics labs have reported positive results for THG.
- Madol or desoxymethyltestosterone (DMT)
  - First reported in scientific literature in the 1960s, but never marketed or approved for use in humans.
  - Isolated in 2004 from an oily product allegedly containing an anabolic steroid and confirmed by two independent labs to contain Madol or DMT.
  - No known safety or efficacy data and no reports of positive tests by any testing laboratory.
- It is likely that additional designer AAS will appear as increasing sophistication of drug testing continues.

Over-the-Counter Steroids

- In 1994 the Dietary Supplement and Health Act made supplements available over the counter, including testosterone (androstenedione, androstenediol, dehydroepiandrosterone [DHEA]) and nandrolone precursors (19-norandrostenedione, 19-norandrostenediol).
- The Anabolic Steroid Control Act of 2004 added steroid precursors such as androstenedione to the list of controlled substances. Although banned by many sports organizations, DHEA was not included among the prohibited substances in the 2004 act and is still available as a dietary supplement.
  - Because of the lack of regulation in the dietary supplement industry, many of the substances included in the 2004 act are still available via the Internet (e.g., a 2007 Internet search of the term "buy andro" yielded 343,000 websites).
- Athletes use these short-acting compounds to increase muscle mass, despite the lack of definitive studies of benefits and adverse effects.
- Some over-the-counter supplements been found to increase testosterone and nandrolone metabolites. They are assumed to have a profile similar to that of AAS. The worldwide increase in nandrolone-positive cases may be a result of use of these compounds.

Potential Therapeutic Uses

- True medical indications for AAS probably account for less than 3 million prescriptions per year.
- In 1990, anabolic steroids were added to Schedule III of the Controlled Substances Act. In 2006, sentencing guidelines for illegal AAS trafficking were increased.
- **Indications** include refractory anemias, hereditary angioedema, palliation therapy in advanced breast carcinoma, replacement therapy in hypogonadal males, and muscle-wasting states associated with HIV infection.
- AAS also may be useful in patients with constitutional delay of growth (as adjunct to growth hormone therapy) and osteoporosis, and it has potential use as a male contraceptive.

Dosage

- **Doses taken by athletes may be 10 to 40 times higher than the therapeutic dose.**
- Athletes frequently use combinations of anabolic steroids (known as **stacking**) or cycle them in pyramidal fashion to achieve maximum effect. Table 21-4 describes several regimens used by bodybuilders.

Adverse Reactions

Gastrointestinal: Hepatocellular dysfunction, peliosis hepatis, case reports of hepatocellular carcinoma. Hepatic effects are increased with 17-α-alkylated compounds consumed orally. Serum liver function tests should be checked in athletes with suspected history of AAS use.

Cardiovascular: Increase in total cholesterol and low-density lipoprotein (LDL) cholesterol; decrease in high-density lipoprotein (HDL) cholesterol; hypertension; thrombotic risks; compartment syndromes; reported cases of myocardial infarction

Table 21-4 SAMPLE AAS DRUG REGIMENS

Drug regimen	AAS used	Dosing protocol
Simple cycle (8 weeks)	Methandrostenolone	25 mg/day PO, weeks 1-8
	Testosterone cypionate	500 mg/week IM, weeks 1-8
Moderate cycle (12 weeks)	Sustenon (testosterone)	750 mg/week IM, weeks 1-12
	Nandrolone decanoate	400 mg/week IM, weeks 1-12
	Methandrostenolone (switch to)	40 mg/day PO, weeks 1-4
	Oxymetholone	50 mg/day PO, weeks 5-8
Complex cycle (20 weeks)	Methandrostenolone (switch to)	50 mg/day PO, weeks 1-5
	Oxymetholone	100 mg/day PO, weeks 6-10
	Testosterone cypionate	1500 mg/week IM, weeks 1-20
	Boldenone undecylenate (switch to) Nandrolone decanoate	800 mg/week IM, weeks 1-10 800 mg/week IM, weeks 11-20
	Stanozolol (switch to) Trenbolone acetate	700 mg/week IM, weeks 1-10 700 mg/week IM, weeks 11-20
	Growth hormone	6 IU/day × 20 weeks
	Insulin (Humalog)	15 U/day (post-workout)

Data from Parkinson AB and Evans NA: Anabolic androgenic steroids: A survey of 500 users. Med Sci Sports Exerc 38:644-651, 2006.

and cerebrovascular accident. Testosterone may contribute to myocardial ischemia through imbalance between myocardial oxygen supply and demand.

Psychological effects: Changes in libido, mood swings, aggressive behavior, exacerbation of underlying mental illness, addiction to the appearance on AAS, suicide attempts. Dependence pattern with opioid-type features has been reported. Pope and Katz interviewed 41 bodybuilders and football players who had used AAS and found that 9 (22%) displayed full affective syndrome and 5 (12%) had psychotic symptoms in association with AAS use. However, a well-designed study by Bahrke and colleagues of current AAS users, previous users, and nonusers demonstrated that, although perceived or actual psychological changes may occur, they were not demonstrated on several standardized inventories.

Male reproductive effects: Oligospermia, azoospermia, decreased testicular size, and gynecomastia. A case of adenocarcinoma of prostate also has been reported.

Female effects: Reduced luteinizing hormone (LH), follicle-stimulating hormone (FSH), estrogens, and progesterone; menstrual irregularities; male pattern alopecia; hirsutism; clitoromegaly; and deepening of voice. Last three are probably irreversible.

Youths: Irreversible, premature closure of the epiphyses. Several highly publicized cases of suicide in high school athletes related to steroid use.

Additional drug use: AAS users are likely to use other drugs. A 2007 study by Elliot of female high school students revealed that AAS users were more likely to use alcohol, cigarettes, marijuana, and cocaine.

Miscellaneous: Spontaneous tendon rupture; increase in sebaceous glands and acne; infectious complications including HIV (resulting from sharing of contaminated needles), hepatitis B and C, and intramuscular abscess; worsening of tic symptoms in patients with Tourette's syndrome; suppression of humoral immunity and immunoglobulin levels.

Side effects: Documented in a secret AAS program for elite athletes sponsored by the German Democratic Republic: liver damage, gynecomastia, polycystic ovarian syndrome, arrested body growth; three deaths were reported.

Prevention

Programs designed to reduce AAS use have been developed (e.g., by L. Goldberg and the ATLAS program; see Recommended Readings) for use at the high school level.

Detection

Clinical suspicion should be aroused by presence of the aforementioned adverse effects. Drug testing (discussed later) can detect AAS with a high degree of accuracy.

GROWTH FACTORS

Human Growth Hormone (hGH)

Definition: A polypeptide hormone composed of 191 amino acids with molecular weight of 21,500; contains many different isoforms, the predominant being a 22 kD isomer and about 10% 20 kD. Normally, 5 to 10 mg is stored in anterior pituitary. Men have a production rate of 0.4 to 1.0 mg per day. The production of recombinant human growth hormone (rhGH) in the 1980s dramatically increased the potential supply. As opposed to natural growth hormone, rhGH contains only the 22 kD isomer.

Prevalence: With the growing effectiveness of gas chromatography and mass spectrometry in detecting AAS and testosterone, many athletes have turned to hGH. Prevalence is difficult to estimate because no reliable method is currently available for detecting exogenous hGH. It is thought to be widely used by football players and bodybuilders and other strength athletes.

Mechanism: In man, hGH stimulates the production of various markers, the most prominent being insulin-like growth factor (IGF-1) or somatomedin-C. Although there is some debate about whether substances such as hepatic-produced IGF-1 are markers or mediators, hGH exerts most of its effects through receptors at target cells. Despite a great deal of debate and marketing, there is little evidence that the commercially available IGF-1 products have any ability to increase IGF-1 levels or strength. It is also clear that while some controlled, albeit limited, studies of hGH have revealed increases in IGF-1 and changes in lean body mass, none have definitively demonstrated increases in strength or athletic performance.

Function: Administration of hGH to growth hormone–deficient children results in positive nitrogen balance and stimulation of skeletal and soft tissue growth.

Metabolic effects: Growth hormone reduces glucose and protein metabolism and has a net anti-insulin effect by inhibiting cellular uptake of glucose. It also stimulates mobilization of lipids from adipose tissue, and protein synthesis is greatly increased in hypophysectomized animals.

Effects on muscle: Several studies report conflicting data about effect of hGH on muscle. Animal experiments by A. L. Goldberg concluded that hGH increased basal metabolic rate of protein synthesis, but that this effect was also determined by the amount of muscular work. It is difficult to predict the ability of hGH to increase contractile elements and improve performance of normal muscle in normal humans. Deyssig showed in studies of hGH that while there may be increases in IGF-1 and changes in lean body mass, there is no definitive demonstration of increases in strength or athletic performance. hGH treatment of adults with acquired growth hormone deficiency increased lean body mass, decreased fat mass, and increased basal metabolic rate. The study concluded that hGH can regulate body composition through anabolic and lipolytic actions.

Therapeutic uses:

- hGH increases stature of growth hormone–deficient children and can also increase rate of growth in some short-statured children who are not hGH-deficient.
- hGH is approved for treatment of growth failure in children with chronic renal failure.
- hGH is used in other countries to promote growth in Turner's syndrome.
- hGH may accelerate wound healing in children with large cutaneous burns, in growth hormone–deficient adults, and in the elderly.
- hGH increases lean body mass and decreases fat in patients postoperatively.
- hGH is approved for treatment of wasting syndrome secondary to HIV infection.

Dosage: FDA-approved dose is 0.003 to 0.004 mg/kg daily via subcutaneous injection, although some recommend 0.15 to 0.3 mg daily, regardless of body weight.

Adverse reactions:

- **Acromegaly** is a potentially serious side effect of megadoses of hGH. It is estimated that acromegalic patients with hGH concentrations of 5 to 30 ng/mL have production rates of 1.5 to 9 mg per day. **As little as a twofold increase in the recommended dose may result in acromegaly, leaving a narrow therapeutic window.** With athletes consuming up to 20 mg daily, the risk of acromegaly is significant. Complications of acromegaly include diabetes, arthritis, myopathies, and characteristic coarsening of bones of face, hands, and feet.
- Side effects reported in growth hormone–deficient patients are generally few. Reported adverse reactions include intracranial hypertension, hyperglycemia, and glycosuria.
- Adult patients using hGH have described fluid retention, arthralgias, gynecomastia, and carpal tunnel syndrome (via median nerve edema).

- Use of hGH in adults who are not deficient in growth hormone has not been established.
- **Creutzfeldt-Jakob disease has resulted from use of hGH derived from cadaveric pituitary glands.** Although use of synthetic hGH eliminates this problem, athletes often obtain substances from black market sources, thereby increasing risk for this catastrophic neurologic disorder.

Detection: hGH is banned by WADA. Testing for hGH was focused on two types of serum-based tests.

- Immunoassays to estimate the amounts of the 20 kD and 22 kD isomers in the serum. The amount of 20 kD is suppressed when rhGH (22 kD) is given, thus a high ratio of 22 kD to 20 kD will indicate use of synthetic GH. This testing has been used at the 2004 and 2008 Summer Olympics and 2006 Winter Olympics.
- Measuring a medley of serum markers, such as IGF-1, osteocalcin, and procollagen type III, can discriminate hGH users from nonusers.

IGF-1

- hGH stimulates the production of IGF-1 by the liver; there is some debate as to whether IGF-1 is a marker or mediator.
- Despite a great deal of debate and marketing, there is little evidence that the commercially available IGF-1 products have any ability to increase IGF-1 levels or strength.
- Some limited controlled studies of rhGH have revealed increases in IGF-1 and changes in lean body mass, but none have definitively demonstrated increases in strength or athletic performance.
- Adverse effects related to IGF-1 use include hypoglycemia, decreased hGH secretion, interference in the insulin-glucagon axis, and an association with carcinoma of the prostate, colon, and lung.
- Mechano growth factors are a new form of IGF-1 that are produced in response to mechanical loads and have been described by Goldspink.

Insulin

- Physicians are trained to view insulin as a solely therapeutic drug, but increasing anecdotal information associates AAS use with insulin and a study by Parkinson and Evans found that 25% of AAS users also used insulin.
- It is speculated that insulin might enhance strength through its inhibitory functions (deters lipolysis, glycolysis, gluconeogenesis, proteolysis, and ketogenesis); when combined with an anabolic agent such as hGH or AAS, the protein-sparing effects of insulin produce larger anabolic results and significantly increase lean body mass.
- Risks of insulin use in normoglycemic athletes include lethal hypoglycemia, lipodystrophy, lipoatrophy, insulin allergy/resistance, and the production of insulin auto-antibodies.
- Insulin is prohibited by WADA, although there is no approved test for its use.

AMPHETAMINES

Definition: Stimulants classified as indirect-acting sympathomimetic amines with central and peripheral effects.

Prevalence: Among the entire group of student-athletes in the 2005 NCAA study, the use of amphetamines as ergogenic aids has continually increased since 1993. Additionally, the use of methylene-n-methylamphetamine (Ecstasy) as a recreational stimulant has caused concern. Amphetamines are commonly found on the black market ("greenies") and contain various drugs, such as clobenzorex.

Mechanism of action: Several theories have been proposed to explain the central and peripheral effects of amphetamines: increased liberation of endogenous catecholamines, displacement of bound catecholamines, inhibition of monoamine oxidase, interference with catecholamine reuptake, and production of false neurotransmitters. All probably contribute to observed physiologic responses, including increases in blood pressure and heart rate, bronchodilation, increased metabolic rate, and increased free fatty acid production.

Relationship to athletic performance: Literature contains contradicting data.

- Smith and Beecher reported that 75% of trained swimmers, weight throwers, and runners had improved performance after taking amphetamines.
- Chandler and Blair demonstrated no substantial improvement in athletic performance.
- Explanation may be that amphetamine enhances performance of simple, repetitive tasks but not of more complicated maneuvers.

Therapeutic uses: Amphetamines have been used legitimately to treat many conditions, including refractory obesity, narcolepsy, attention deficit disorder, and severe depression. High abuse potential has limited their utility in these conditions.

Dosage: Taken orally, amphetamines exert effects within 30 minutes of ingestion; their actions can last 12 to 24 hours. Dosages vary, depending on the athlete and the type of preparation.

Adverse reactions: Central nervous system: restlessness, insomnia, psychological addiction, psychosis, tremor, anxiety, dizziness, cerebral hemorrhage. Cardiovascular: lowered threshold for arrhythmias and provocation of angina. Miscellaneous: disruptions in thermoregulation, predisposition to heat illness.

Detection: Amphetamines are readily detected by urine tests because both unchanged amphetamines and metabolites appear in urine.

COCAINE

Definition: A naturally occurring alkaloid derived from the leaves of the *Erythroxylon coca* plant. Although it is a topical anesthetic, it also acts as a central nervous system stimulant. This effect has led to its use by athletes as an ergogenic aid.

Prevalence: According to the 2005 National Survey on Drug Use and Health, approximately 33.7 million Americans ages 12 and older had tried cocaine at least once in their lifetimes, representing 13.8% of the population ages 12 and older. Approximately 5.5 million (2.3%) had used cocaine in the past year and 2.4 million (1.0%) had used cocaine within the past month. The 2005 NCAA study found that 2.1% of athletes had used cocaine in the previous 12 months; this figure has trended upward from 1.1% in 1993 (Table 21-1). Cocaine dependence is typically associated with cyclical patterns of drug use and abstinence. During abstinence, periods of intense cocaine craving and other withdrawal symptoms (anergia, anhedonia, and depression) often lead to relapse.

Mechanism: Cocaine increases the release and blocks the reuptake of norepinephrine from neurons in nervous system. Increased availability of epinephrine causes euphoria, increased blood pressure, tachycardia, lowered threshold for seizures, and ventricular arrhythmias. Cocaine also stabilizes axonal membranes and blocks nerve impulse initiation and conduction. Combined with its properties as a vasoconstrictor, it is an excellent topical anesthetic. Cocaine may cause hyperglycemia, hyperthermia, and increased peripheral reflex speed.

Therapeutic uses: Although scientists of the 19th century hailed cocaine as a cure for a variety of ailments from hemorrhoids to broken bones, its legitimate use is now limited to topical anesthetic.

Dosage: Cocaine is readily absorbed by intravenous, intranasal, and pulmonary routes. Recreational users of intranasal cocaine may use 1 to 3 g per week. To mimic the intense high associated with IV use without the complications of needles, users have turned to smoking. This began with smoking of the free alkaloid form, known as "free base." Availability of ready-to-smoke, low-

priced free base ("crack") cocaine has led to epidemic smoking in urban areas. The effect of crack is rapid and lasts only 5 to 10 minutes. The half-life of cocaine is 2 to 6 hours; it can be detected in urine for 3 to 5 days.

Adverse reactions:

- **Cardiovascular:** Increased levels of catecholamines associated with cocaine use can directly induce ventricular dysrhythmia, coronary vasospasm with thrombosis, and myocardial infarction, all of which can lead to **sudden death, even in patients without underlying heart disease.** Aortic rupture and cerebrovascular accidents also have been reported.
- **Central nervous system:** Chronic use can result in agitation, insomnia, and tremulousness. Toxic psychosis, severe depression, paranoia, and dysphorias have been reported as well as rapid addiction.
- **Respiratory system:** Taken intranasally, cocaine can cause swelling of nasal mucosa, rhinitis, sinusitis, epistaxis, and nasal septal necrosis. Bronchitis and bronchiolitis obliterans with organizing pneumonia have been reported.
- **Considerations in athletes:** Cocaine has direct effects on central thermoregulation, and an athlete exercising in heat is susceptible to hyperthermia. It has been proposed that elite sprint-trained athletes may be at a greater risk for severe lactic acidosis and cocaine-induced seizures because of higher percentage of glycolytic muscle fibers. Studies have demonstrated that chronic cocaine-conditioned animals have exaggerated catecholamine response to combination of cocaine and exercise. A study by Welder and Melchert suggests an increase in myocardial events when cocaine is combined with AAS.

Detection: Cocaine is readily detectable by most drug testing. According to data from Anderson and colleagues, most cocaine users do not participate in drug use with teammates. It may be difficult for coaches, trainers, or team physicians to detect patterns of cocaine usage.

CAFFEINE

Definition: A naturally occurring plant alkaloid derived from aqueous extracts of *Coffea arabica* and *Cola acuminata*. Caffeine is classified as central nervous system stimulant and is found in coffee, tea, and cola drinks. It is a methylxanthine and chemically related to theobromine and theophylline. It has been used by athletes for its stimulant properties and potential for increased work and power.

Prevalence: According to year 2000 statistics, nearly 54% of Americans over age 18 drink coffee every day and another 25% drink it occasionally. The average coffee drinker in the United States consumes 3 cups per day. The 2007 U.S. market for energy drinks was estimated to be $3 billion per year; that includes caffeinated products. Energy drinks are heavily marketed toward young adults, but they contain stimulants that make them unsuitable for young children and pregnant women. Analysis has shown that caffeine levels per 8-ounce serving ranged from 80 mg to 280 mg; a cup of coffee contains about 100 mg of caffeine, and a 12-ounce Coke Classic has about 35 mg (Tables 21-5 and 21-6).

Mechanism of action: Caffeine is rapidly absorbed, and peak levels are achieved in 30 to 60 minutes with half-life of 3.5 hours. Caffeine acts as a competitive antagonist of adenosine and causes vasoconstriction (except in renal afferent artery), increased diuresis and natriuresis, central nervous system stimulation, increased lipolysis in adipocytes, and increased gastric secretion. Caffeine probably potentiates calcium release from skeletal muscle sarcoplasmic reticulum, thus increasing force of muscle contraction at lower frequencies of stimulation with sparing of muscle glycogen.

Performance: Increased work and power probably result from increased mobilization of free fatty acids, increased rate of lipid metabolism, and direct effects on muscle contraction secondary to increases in calcium permeability of sarcoplasmic reticulum. Overall, ability of caffeine to enhance or prolong work output has been controversial, and recent studies cast doubts on its effectiveness. If it has benefit in athletic performance, it is limited to endurance activities.

Table 21-5 CAFFEINE CONTENT OF COMMON DRINKS

Name	Ounces	Caffeine (mg)
Coke Classic	12	34.5
Diet Coke	12	45
Pepsi	12	38
Diet Pepsi	12	36
Jolt Cola	23.5	220
Coffee (Drip)	8	145
Starbucks Grande Coffee	16	372
Cocaine Energy Drink	8.4	280
Full Throttle	16	144
Red Bull	8.3	80
Redline RTD	8	250
Rockstar	16	160

Table 21-6 CAFFEINE CONTENT OF COMMON SUBSTANCES

Substance	Concentration	Level (μg/mL)*
Coffee (mg/mL)	55-85	1.5-3 (1 cup)
Tea (mg/mL)	55-85	1.5-3 (cup)
Cola (mg/mL)	10-15	0.75-1.5 (1 cup)
Medications (mg/tablet)		
Cafergot	100	3-6
NoDoz	100	3-6
Anacin	32	2-3
Midol	32	2-3

*Level depends on size of athlete and rate of metabolism. These figures represent general estimates based on average size and rate of metabolism.

Therapeutic uses: Caffeine has been used as stimulant in fatigue states, in combination with analgesic compounds, and in diet pills. Table 21-5 provides information about caffeine content of certain over-the-counter medications.

Dosage: WADA no longer bans caffeine but maintains monitoring. The NCAA has set maximum urinary concentration of caffeine at 15 μg/mL. One study found that to exceed that threshold, the athlete needed to consume almost 1000 mg of caffeine within 3 hours of testing. This is far greater than the average daily consumption of 200 mg of caffeine. Athletes concerned about testing should be aware that caffeine excretion is variable and can be affected by many factors, including exercise. Caffeine concentration as it relates to urinary levels is provided in Table 21-5.

Adverse reactions: Central nervous system: anxiety, hypochondriasis, insomnia, headache, tremors, depression, scotomata, and addiction with withdrawal states. Cardiovascular: tachyarrhythmias, especially paroxysmal atrial tachycardia. Renal: diuretic effect, which is of significance in athletes at risk of dehydration.

Detection: The NCAA allows a maximum level of 15 μg/mL. Caffeine levels can be increased by concomitant use of selective serotonin reuptake inhibitors (SSRIs, e.g., fluoxetine) and cimetidine.

SYMPATHOMIMETIC AMINES

Definition: Synthetic congeners of naturally occurring catecholamines. In addition to amphetamines, several other weaker sympathomimetic amines have the potential to be abused by athletes, including phenylpropanolamine, phenylephrine, ephedrine, and pseudoephedrine.

Prevalence: Sympathomimetic amines appear in various cold remedies, common nasal and ophthalmologic decongestants, and most asthma preparations. After passage of the Dietary Supplement Health and Education Act in 1994, ephedrine appeared in various over-the-counter dietary supplements for weight loss and energy and was often listed as ephedra and ma huang. However, following at least 150 deaths, including several high-profile athletes purportedly linked to ephedrine use (Steve Bechler of the Baltimore Orioles, Korey Stringer of the Minnesota Vikings, and Rashidi Wheeler of Northwestern University), the Food and Drug Administration in 2004 banned dietary supplements containing ephedrine. Despite the ban, ephedrine-containing products are still available in the black market on the Internet.

Mechanism: Response of sympathomimetic amines depends on relative selectivity of specific drug. Although earlier scientific studies demonstrated that ephedrine improved athletic performance, three studies have shown no significant increases in performance.

- Alpha effects: smooth muscle contraction, primarily vasoconstriction.
- $Beta_1$ effects: production of intracellular cAMP, increased heart rate and strength of contraction.
- $Beta_2$ effects: smooth muscle relaxation, bronchodilation, stimulation of skeletal muscle.

Therapeutic uses: Nonemergent treatment of allergic reactions, asthma, hypotension during spinal anesthesia, atrioventricular block, and nasal congestion.

Dosage: Many types of drugs in this category have varying potencies and durations of action.

Adverse reactions with increasing doses: Anxiety, epigastric distress, palpitations, tremulousness, insomnia, drowsiness, hypertension, stroke, seizures.

Detection: Sympathomimetic amines, such as pseudoephedrine, phenylephrine, phenylpropanolamine, and synephrine, are now being monitored by WADA but are no longer considered prohibited substances. Ephedrine is still banned by WADA. Sympathomimetic amines can be detected by drug testing. WADA allows the use of inhaled selective $beta_2$ agonists, terbutaline, albuterol, bitolterol, orciprenaline, and rimiterol. Because of the changing regulations with sympathomimetic amines, it is always recommended that an athlete check with his or her sports testing authority before consuming these substances.

Clenbuterol

Overview: Clenbuterol has garnered attention because of its potential as an anabolic substance.

Definition: A $beta_2$-agonist, similar to albuterol, used as a bronchodilator. It has recently been considered an anabolic or "repartitioning" agent. Anabolic effects have been purported to occur only with oral forms, not via inhalation route.

Prevalence: Limited information about prevalence is derived mostly from increasing numbers of athletes who are disqualified by positive drug tests. Six Olympians were disqualified in 1992: two Americans, two Germans, and two British. Parkinson and Evans reported clenbuterol use for fat loss by AAS users. A 2007 report found that clenbuterol was illegally present in some dietary supplements available on the Internet.

Mechanism: Most data about anabolic effects of clenbuterol are derived from animal studies. No studies to date have examined effects of oral clenbuterol on nonasthmatic athletes. Livestock treated with clenbuterol demonstrated increased muscle mass and decreased fatty deposits. Denervated rat hind limbs demonstrated reduced muscle wasting when treated with clenbuterol as well as a decreased net bone loss. Clenbuterol produced no changes in muscle cross-sectional area in a randomized study by Maltin and colleagues of patients undergoing medial meniscectomy. Although the finding was not statistically significant, the authors asserted that the clenbuterol group regained strength more rapidly. The exact mechanism of clenbuterol's anabolic effects has not been determined, but it has been previously shown that catecholamines attenuate amino acid release from muscle by beta-mediated depression of protein catabolism. Clenbuterol may also act by increasing contractile tension.

Therapeutic uses: Clenbuterol is used as a bronchodilator for treatment of asthma; however, it is not legally available in the United States.

Dosage: Recommended dosage for asthma is 0.02 to 0.03 mg twice daily. Anabolic dosage is not known, but extrapolation from animal data translates into 0.001 to 0.01 mg/kg or 0.07 to 0.7 mg in a 70-kg person. Maltin and associates used the lower asthma dose, 0.02 mg twice daily, in their study of orthopedic patients.

Adverse reactions (typical $beta_2$ effects): Muscle tremor, palpitations, muscle cramps, tachycardia, tenseness, headaches, peripheral vasodilation. There has also been a case report of clenbuterol potentiating the effects of AAS that resulted in myocardial infarction.

Detection: Clenbuterol is prohibited by WADA and the NCAA. It is also banned by the Association of Official Racing Chemists for its ergogenic effects on thoroughbred racehorses. It can be detected in urine by gas chromatography–mass spectroscopy.

Other $Beta_2$-Agonists

- Debate about clenbuterol has raised questions about its anabolic effects and the possible banning of other $beta_2$-agonists.
- Controversial because most studies of athletes reveal that 10% suffer from exercise-induced asthma and depend on $beta_2$-agonists to compete.
- Studies have been inconclusive about ergogenic effects of other inhaled $beta_2$-agonists, and further research is warranted.
- It would be extremely difficult to deny asthmatics the use of inhaled $beta_2$-agonists.

ALCOHOL

Definition: Ethanol, most abused recreational drug in the United States, is classified as a depressant.

Prevalence: Approximately 70% of adult Americans drink alcohol, and per capita consumption is estimated at 2.23 gallons per year. Perhaps 5% to 10% of drinkers are, or will become, alcoholics. There are 75,000 annual alcohol-related deaths. The 2005 NCAA study revealed that 77% of student-athletes consume alcohol. Nelson and Wechsler compared college-aged athletes with nonathlete college students and found that athletes report more binge drinking, heavier alcohol use, and increased drinking-related harm. They concluded that athletes are a high-risk group for such behaviors.

Mechanism: Physiologic effects of alcohol are well-known and not reviewed here.

Therapeutic uses: None.

Adverse reactions: Physicians are familiar with many adverse consequences of alcohol abuse. With respect to athletes:

- **After literature review, the American College of Sports Medicine issued a statement about the use of alcohol in relation to athletic performance:** It states that acute ingestion of alcohol has a deleterious effect on many psychomotor skills, including reaction times, hand-eye coordination, accuracy, balance, and complex coordination. Alcohol consumption does not substantially influence physiologic functions crucial to physical performance ($\dot{V}O_2$ max), respiratory dynamics, cardiac function). It does not improve muscular work capacity and may decrease performance levels as well as impair temperature regulation during prolonged exercise in cold environment.

- A study by Urbano-Marquez and colleagues concluded that alcohol is toxic to striated muscle in a dose-dependent manner.
- Arrests for driving while impaired substantially increase the risk of eventual death in an alcohol-related crash.

Detection: Except for shooting events (including the modern pentathlon) and bowling, neither the NCAA nor WADA specifically tests for presence of alcohol. Breath or blood levels may be determined at the request of an international federation.

Identification: The American Medical Association defines **alcoholism** as "Illness characterized by significant impairment that is directly associated with persistent and excessive use of alcohol. Impairment may involve physiological, psychological, or social dysfunction." Because the effects of alcoholism may take 5 to 20 years to develop and usually occur after age 30, early identification of athletes with alcohol problems is imperative. Several measures are designed to help assess alcohol problems, including the Perceived Benefit of Drinking Scale (PBDS), the Children of Alcoholics Test (CAST), and the Michigan Alcoholism Screening Test (MAST).

- The easiest test to administer is the **CAGE questionnaire:**
  - Have you ever felt you should *Cut down* on your drinking?
  - Have people *Annoyed* you by criticizing your drinking?
  - Have you ever felt bad or *Guilty* about your drinking?
  - Have you ever had a drink first thing in the morning *(Eye-opener)* to steady your nerves or to get rid of a hangover?
- A CAGE score of 2 or more is associated with alcohol abuse or dependence. Two additional questions that are helpful are:
  - Is there a positive family history of alcoholism?
  - Have you had any arrests for driving while intoxicated?
  - What is your regular consumption? (Three drinks per day for men and two drinks per day for women suggests an alcohol abuse problem).

Prevention: Education and identification are keys to prevention in athletes.

NICOTINE

Definition: Volatile alkaloid derived from tobacco and responsible for many effects of tobacco. It first stimulates (small doses) then depresses (large doses) autonomic ganglia and myoneural junctions. Methods of consumption include cigarettes and smokeless tobacco (loose leaf tobacco [chewing], moist or dry powdered tobacco [snuff or "dipping"], compressed tobacco ["plug"]).

Prevalence: Athletes are aware of the health risks associated with cigarette smoking, and according to the 2005 NCAA study, the number of daily cigarette users has decreased dramatically, with many more student-athletes reporting that they only smoke at social occasions. However, there was an increase among smokers in those who smoke a pack or more a day. Previously, athletes turned to smokeless tobacco as an alternative. However, the 2005 NCAA study showed that smokeless tobacco use was at its lowest level since the study began.

Mechanism: Nicotine has various actions, depending on receptor binding and dose, and readily crosses the blood-brain barrier. It binds to acetylcholine receptors at autonomic ganglia, adrenal medulla, neuromuscular junction, and in the central nervous system. Stimulation of central nicotinic receptors activates central nervous system neurohumoral pathways to release acetylcholine, norepinephrine, dopamine, serotonin, vasopressin, growth hormone, and ACTH. Nicotine affects sympathetic nerves by the release of catecholamines. At low doses, ganglionic stimulation and sympathetic discharge lead to an increase in heart rate and blood pressure mediated through central nervous system. At moderate doses, direct peripheral nervous system effects lead to ganglionic stimulation and adrenal catecholamine release. At high doses, ganglionic blockade leads to hypotension and bradycardia. Pharmacodynamic tolerance develops to subjective and hemodynamic effects.

Therapeutic uses: As an aid to smoking cessation, nicotine gum and transdermal patches have been somewhat effective in reducing withdrawal symptoms of physical nicotine dependence in smokers trying to quit. Bupropion has been used to reduce cravings and varenicline may assist quitting by blocking alpha-4-beta-2 nicotinic acetylcholine receptors.

Dosage: Smokeless tobacco users have blood nicotine levels equivalent to nicotine-dependent smokers. A normal single dose of snuff contains 1.5 to 2.5 g of tobacco; each gram has 14 mg of nicotine. Ten percent of nicotine is absorbed, leading to 2.0 to 3.5 mg of nicotine in the bloodstream (two to three times higher than the dose of a standard 1-mg cigarette). The average single dose of chewing tobacco contains 7 g, with a nicotine content of 7.8 mg per g; 8% is available to be absorbed. Eight to 10 chews per day result in a nicotine dose equivalent to 30 to 40 cigarettes per day.

Adverse reactions: Long-term effects of cigarette smoking are well known and are not reviewed here. Studies on the effects of smokeless tobacco have demonstrated:

- **Oropharynx:** A 50-fold increase in oral carcinomas, a 2.4 times higher incidence of dental caries, increases in gingival disease and leukoplakia.
- **Hemodynamics** (cigarette smoking compared with smokeless tobacco): Similar levels of nicotine throughout day, equivalent increases in heart rate and myocardial oxygen demand. Increased sodium (added for flavoring) absorption from smokeless tobacco may lead to increases in blood pressure.
- **Reaction time and concentration:** Smokeless tobacco has long been touted for its ability to improve reaction time and concentration. A study of athletes and nonathletes by Edwards and colleagues demonstrated no neuromuscular performance enhancement; no changes in reaction time, movement time, or total response time; and a significant elevation in heart rate. A survey by Connolly and colleagues of major league baseball players who used smokeless tobacco found that only 10% believed that it improved concentration, and none felt it sharpened reflexes.
- **Addiction:** Smokeless tobacco can be as addictive as cigarette smoking. The National Cancer Institute, in conjunction with major league baseball, has prepared "Beat the Smokeless Habit: Game Plan for Success" to give readers the facts about smokeless tobacco. It includes a "9-inning game plan" to help athletes quit smokeless tobacco.

Detection: Nicotine is not currently tested for by international organizations or the NCAA, but the NCAA bans use of tobacco products by players, coaches, and officials during competition.

MARIJUANA

- **Definition:** Naturally occurring cannabinoid containing active ingredient δ9-tetrahydrocannabinol (THC). It is currently an illegal drug used recreationally as euphoriant.
- **Prevalence:** It is estimated that more than 94 million Americans age 12 and older have tried marijuana, and at least 14.6 million are regular users. Of 20.3% of athletes who had used marijuana in the past 12 months in the 2005 NCAA study, 63.5% stated that they used marijuana for recreational or social reasons.
- **Mechanism:** THC affects various tissues, with central nervous system and cardiovascular system effects being most prominent. Effects depend on route, dose, setting, and prior experience of user.
  - **Cardiovascular:** Tachycardia is dose-related and can be blocked by propranolol. Systolic blood pressure increases in supine position and decreases in standing position.
  - **Central nervous system:** Impaired motor coordination, decreased short-term memory, difficulty in concentrating, decline in work performance.
  - **Male reproductive system:** Decreased plasma testosterone, gynecomastia, oligospermia.

Therapeutic uses: THC has been used as an antiemetic agent in conjunction with chemotherapy for cancer patients and for lowering intraocular pressure in patients with glaucoma.

Dosage: The THC content of marijuana in the United States ranges from 0.5% to 11%; serum concentration depends on smoking technique.

Adverse reactions: Renaud and Cormier found the following effects on exercise performance: reduction of maximal exercise performance with premature achievement of $\dot{V}O_2$ max; no effects on tidal volume, arterial blood pressure, or carboxyhemoglobin compared to controls. Marijuana causes inhibition of sweating that can lead to increase in core body temperature.

Detection: Marijuana is banned by WADA in competition and is considered a street drug by the NCAA and is prohibited at a level of 15 ng/mL. Because of its high lipid solubility, marijuana can be detected for as long as 2 to 4 weeks by drug testing. Passive inhalation of marijuana smoke would not be expected to exceed levels above 15 ng/mL.

BLOOD DOPING

Definition: Blood is removed from an athlete and stored in a frozen state. The athlete's red cell mass is allowed to reequilibrate; then donated red cells are reinfused with a resultant increase in red cell mass (also known as blood boosting or blood packing).

Prevalence: The actual extent of blood doping is unknown; however, there have been widespread rumors of this practice for the past 20 years. In 1984, U.S. Olympic Committee (USOC) admitted that seven U.S. cyclists had engaged in blood doping at the Summer Olympic Games. In 1990, a study by Scarpino and colleagues of 1018 Italian athletes revealed that 7% had tried blood doping. The advent of better testing for erythropoietin (EPO) has led athletes back to blood doping. Cyclist Tyler Hamilton tested positive for blood transfusions after winning the gold medal at the Athens 2004 Olympics. Operation Puerto is a 2006 Spanish doping case against a doctor accused of administering prohibited doping products to hundreds of professional athletes.

Mechanism: Transfusion increases oxygen delivery to exercising muscle, and studies confirm that red cell mass and $\dot{V}O_2$ max are well correlated. Studies of blood doping in elite runners compared with controls demonstrated improvement in maximal oxygen consumption, increased total exercise time, and increased hemoglobin concentration.

Therapeutic uses: Red cell transfusions are limited to patients with symptomatic anemia.

Dosage: Most studies have used 2000 mL of homologous blood or 900 to 1800 mL of cryopreserved autologous blood.

Adverse reactions: Improperly matched donor blood can result in transfusion reactions that may be fatal. Immune side effects are reported in 3% of all transfusions. Using donor blood has attendant risks of infectious complications. Risk is substantially lower with use of autologous blood.

Detection: WADA bans blood doping, but enforcement is limited by lack of effective technique for its detection. A test for homologous blood transfusions was implemented at the 2004 Summer Olympic Games in Athens. This test can show that doping took place by providing evidence of different cell populations. WADA is funding research projects aimed at developing a test for autologous transfusions and may include measurements of hemoglobin mass.

ERYTHROPOIETIN (EPO) AND RELATED COMPOUNDS

Definition: Sialic acid–containing hormone that enhances erythropoiesis by stimulating formation of proerythroblasts and release of reticulocytes from bone marrow. It is mainly secreted by kidneys, and level of erythropoietin is inversely related to number of circulating red cells. In 1986, a recombinant form of EPO (called r-HuEPO) was discovered that is identical to natural EPO except for the addition of sialic acid residues. In 2002, a long-acting form called darbepoetin was introduced.

Prevalence: With increased attention (and attendant risks) to blood doping, r-HuEPO has become a potential avenue of abuse for athletes. The unexplained deaths of 18 Dutch and Belgian cyclists between 1987 and 1990 raised the specter of r-HuEPO abuse. The Festina cycling team was expelled in the first week of the 1998 Tour de France after a team car was found loaded with performance-enhancing drugs including r-HuEPO.

Mechanism: Subcutaneous injection of r-HuEPO stimulates red cell production within days, and effects can be seen for as long as 3 to 4 weeks. Based on data from red cell reinfusion studies, r-HuEPO can theoretically increase $\dot{V}O_2$ max by 10%. One uncontrolled study demonstrated an increase in mean maximal oxygen uptake and run time to exhaustion using r-HuEPO.

Therapeutic uses: In patients suffering from anemia secondary to end-stage renal disease, studies demonstrate that r-HuEPO eliminates the need for transfusions and restores hematocrit to normal in many patients; it can partially correct renal anemia and results in significant increase of both exercise capacity and maximum work, and can maintain normal hemoglobin concentration in uremic patients over time. r-HuEPO was found to be useful in treatment of anemias secondary to prematurity, multiple myeloma, and cancer, and in patients with AIDS treated with zidovudine. r-HuEPO increases the yield of autologous blood donors safely and effectively over a 21-day period and can reduce the need for transfusions in patients undergoing hip replacement.

Dosage: In a study of autologous blood donors with initially normal hemoglobin levels, 600 U/kg of r-HuEPO was given intravenously 6 times over 21 days. Doses in chronic renal patients have ranged between 15 and 500 U/kg three times weekly.

Adverse reactions:

- Observed side effects in patients with renal anemia
  - Hypertensive patients required additional antihypertensive medication.
  - Serum levels of potassium and bilirubin increased.
  - 20% of patients developed flulike syndrome.
  - 14% of patients developed thrombosis of arteriovenous fistulas and veins.
  - 11 patients reported visual hallucinations.
- Potential risks of r-HuEPO in athletes with normal hemoglobin
  - Increases in hemoglobin and blood viscosity accentuated with dehydration may lead to cerebral or cardiovascular ischemia, vascular thrombosis, hypotension, hyperkalemia, and iron deficiency.
  - Attendant risks of intravenous medication (e.g., infection with hepatitis, human immunodeficiency virus [HIV], endocarditis).

Detection: There is a direct test that looks at the presence of r-HuEPO in the urine. The test shows the isoform patterns of r-HuEPO in urine by separation in an electrical field and detection with a very sensitive and selective method. The isoform pattern of r-HuEPO is distinctively different from EPO. The isoelectric-focusing urine test also yields a pattern for darbepoetin alfa that is distinct from both EPO and r-HuEPO.

GAMMA-HYDROXYBUTYRATE AND DERIVATIVES

Definition: Endogenous neurotransmitter produced through metabolism of gamma-aminobutyric acid (GABA). Gamma-hydroxybutyrate (GHB) increases cerebral dopamine levels and acts on the endogenous opioid system. It is structurally similar to GABA. Following its ban in 1991, manufacturers began producing GHB precursors, including gamma-butyrolactone (GBL), 1,4 butanediol, and gamma-valerolactone (GVL).

Prevalence: There are no surveys to date about prevalence of use; however, anecdotal reports seem to indicate that it is widely used.

Mechanism: GHB crosses the blood-brain barrier and has been shown to double dopamine and dynorphin levels in the brain. Hippocampal receptors for GHB have been isolated, and synap-

tic transmission and regulation have been demonstrated. GHB facilitates slow wave sleep, which is associated with growth hormone release. It is postulated that this may increase muscle mass.

Potential therapeutic uses: GHB is a Schedule I drug and because of its ability to increase cerebral dopamine levels, GHB has been used experimentally to treat myoclonus and cataplexy associated with narcolepsy (the only indication for use in the United States). GHB has been used in other countries as an anesthetic agent (especially in children), for ethanol withdrawal, and as a treatment for ischemic conditions. It has been illegally marketed in the United States as a muscle-building drug and a sleeping aid.

Dosage: The usual directions are 1 tablet or ½ to 3 teaspoons of powder dissolved in water. It is unclear how much athletes are consuming. Ten mg/kg orally can cause amnesia and hypotonia; 20 to 30 mg/kg produces somnolence within 15 minutes; and more than 50 mg/kg can cause unconsciousness and coma.

Adverse reactions: Multiple cases of poisonings secondary to GHB have been reported with patients complaining of gastrointestinal symptoms, central nervous system and respiratory depression, and uncontrolled movements. At least 11 patients have been hospitalized, and 9 required ventilator support. In 1991, the FDA issued a report that GHB was unsafe and illicit and that its use should be discontinued because of potentially dangerous adverse effects. In 2001, there were 119 reported adverse reactions to GBL and 9 toxic events and 2 deaths attributed to 1,4 butanediol.

Detection: These compounds are highly volatile with half-lives of about 1 hour, making detection difficult. Kankaapaa recently reported an experimental method for blood and urine detection.

AGENTS WITH ANTIESTROGENIC ACTIVITY

Definition: These are substances that oppose estrogens and include:

- Aromatase inhibitors such as anastrazole and letrozole.
- Selective estrogen receptor modifiers such as tamoxifen and raloxifene.
- Other antiestrogen substances such as clomiphene.

Prevalence: Unknown, but multiple anecdotal reports among users of anabolic steroids.

Mechanism: High doses of androgens will overwhelm hepatic capacity and some of the excess is aromatized to estrogenic compounds, creating unwanted adverse effects in males. These antiestrogenic drugs are used to counteract the estrogenic effects, although they are not technically performance-enhancing themselves.

Potential therapeutic uses: These drugs are used for a variety of legitimate uses including adjuvant therapy in breast cancer and ovulation induction.

Adverse reactions: These drugs are rarely used therapeutically in males and as such the adverse reactions are limited to women. It is likely that interfering with the hypothalamic-pituitary-gonadal axis will have major adverse effects in males.

Detection: These drugs are banned by WADA and can be detected using liquid chromatography–mass spectrometry (LC-MS) technology.

GENETIC DOPING

Definition: The nontherapeutic use of genes, genetic elements, and/or cells that have the capability to enhance athletic performance.

Mechanism: Genetic doping may be achieved through several methods, including viral vector (such as modified adenovirus) or gene transfer that produces a specific message or "switch" to turn on production of a desired substance.

Potential therapeutic uses: Genetic manipulation has the potential to cure a wide assortment of diseases, such as muscular dystrophy, and has been used in congenital immunodeficiency states.

Adverse reactions: The limited use of gene therapy has resulted in some severe effects, including the development of cancer. Primate studies with the insertion of an EPO-producing gene resulted in the animals requiring regular phlebotomies because of uncontrolled red cell production. Immune reactions are a major concern.

Specific drugs: It has been speculated, but unconfirmed, that the patented drug Repoxygen may have been used by athletes. The drug alters EPO production in response to low oxygen tension.

Detection: Although the practice is banned by WADA, there are no tests for detecting it. Possible deterrents include detection of the viral genome (may be difficult if a common virus is used), detection of the identifying switch, testing serial biomarkers or pharmacodynamic tests, having manufacturers "label" genetic material to allow detection, and control over the production and supply of materials.

Miscellaneous: In addition to genetic doping, the practice of genetically identifying sport-specific genes in children has begun in order to promote athletic success. Some of these markers include creatine kinase, angiotensin-converting enzyme, and mitochondrial enzymes. This practice raises ethical questions and challenges the very definition of "sport." Genetic doping and genetic identification of athletic traits is on the horizon and likely will be very difficult to harness once unleashed.

DRUG TESTING

Purpose: Drug testing has been a major means of attempting to enforce compliance with the banned substance list. Public awareness has been heightened by positive tests in elite athletes. To date, most drug testing has been of the "announced" variety at championship or Olympic events, although there are attempts to increase unannounced testing.

Cost: The 2005 budget for USADA was $11.89 million, with approximately 63% coming from the federal government and 37% from the USOC.

Reliability: Tests vary in specificity, sensitivity, and expense.

- **Thin-layer chromatography:** The least expensive screening test, but it has less specificity and cannot provide positive identification of the substance. It is not used in athletic drug testing.
- **Radioimmunoassay and enzyme-multiplied immunoassay:** The two most commonly used screening methods. During the 1984 Olympic Games, radioimmunoassay was used as a screening test for amphetamines, morphine, and benzoylecgonine. Although manufacturers claim a 97% to 99% accuracy rate, such is not usually the case. To avoid false-positive tests when an athlete's career may be at stake, a second, highly sensitive and specific test is required.
- **Gas chromatography—mass spectroscopy (GC-MS):** The gold standard and the only drug test admissible in court. All state-of-art laboratories must use GC-MS as the confirmatory test because it provides a "fingerprint" of the detected substance. At the 1984 Olympics, GC alone was used to screen for volatile and nonvolatile agents and GC-MS to screen for anabolic steroids. All athletes with positive results underwent confirmatory GC-MS. Unfortunately, the high cost of both the equipment and the test prohibits most smaller laboratories from using GC-MS.
- **Gas chromatography-combustion-IRMS:** A new tool that WADA accepts as a method for determining whether an elevated T/E ratio is the result of exogenous testosterone use. Isotope Ratio Mass Spectrometry (IRMS) is based on the finding that 98.9% of the carbon atoms in nature are ^{12}C,

with 1.1% being ^{13}C (an isotope that contains an additional neutron). The ratio of ^{13}C to ^{12}C can be measured with high accuracy and precision by an isotope ratio mass spectrometer. Very small differences in the abundance of ^{13}C can be detected to allow differentiation of carbon sources. The IRMS values for steroids are expressed as delta values: $\delta^{13}C‰$ percentage. The more negative a delta value, the less ^{13}C the compound contains. Pharmaceutical testosterone is manufactured from a soy compound that contains relatively less ^{12}C content as compared with endogenously produced testosterone, thus yielding significantly different results on IRMS analysis.

Testing for anabolic steroids/testosterone: To combat the use of exogenous testosterone, the ratio of testosterone to its isomer epitestosterone (T:E) has been quantified. A normal T:E ratio is 1:1. When exogenous testosterone is administered, serum testosterone is elevated out of proportion to epitestosterone. A ratio greater than 6:1 is considered a positive test by the NCAA; however, the USADA and the NFL consider a ratio greater than 4:1 to be positive. The test is limited because some men have naturally high T:E ratios (greater than 6:1). Because of the improved accuracy of high-resolution GC-MS in detecting synthetic AAS, use of exogenous testosterone has increased.

- **Carbon isotope ratio** (CIR) helps distinguish a naturally high T:E ratio from use of exogenous testosterone by comparing the ratio of carbon-13 to carbon-12 in a sample with the ratio of pharmaceutical testosterone.
- Athletes have begun taking epitestosterone in conjunction with testosterone ("the cream") to maintain a "normal" T:E ratio. To combat this practice, WADA and the USADA currently ban epistestosterone in concentrations greater than 200 ng/mL.
- Athletes using testosterone also use human chorionic gonadotropin (hCG) to help reduce the T:E ratio. Testing is available for hCG, and hCG is banned by WADA.

Circumvention by athletes: Although GC-MS may approach 100% accuracy, athletes have attempted numerous methods of avoiding detection:

- **Masking agents:** Diuretics and tubular blocking agents such as probenecid have been used to mask presence of banned substances in urine. Most drug tests use minimum urinary specific gravity to combat this practice; probenecid and diuretics have been specifically banned by WADA.
- **Determination of drug half-life:** With announced drug testing, athletes can determine how long a drug can be detected in urine. Table 21-7 lists some elimination times. This problem can be addressed with random, unannounced testing.
- **Substitution of urine:** Athletes have developed numerous methods to substitute "clean" urine, including self-catheterization and innovative "delivery systems." To eliminate this problem, collection is conducted under constant supervision and close observation.

Table 21-7 DRUG CLEARANCE TIMES

Drug	Approximate elimination time
Stimulants (e.g., amphetamines)	1-7 days
Cocaine	
Occasional use	6-12 hr
Repeated use	3-5 days
Codeine and narcotics in cough medicine	24-48 hr
Tranquilizers	4-8 days
Marijuana	3-5 wk
Anabolic steroids	
Fat-soluble injectable	6-12 mo
Water-soluble oral	1-6 wk
Over-the-counter cold preparations containing ephedrine	48-72 hr

Extent of testing: Most athletic organizations, professional and amateur, have developed drug-testing programs. These policies are subject to frequent change depending on law, collective bargaining, and contemporary issues. It is best to check with respective organizations when specific questions arise.

- **Olympic level:** WADA and USADA conduct formal drug testing at sanctioned events, such as the Olympics, the Olympic Trials, and the Pan Am Games. No announced notice (NAN) testing out of competition has been initiated as an increased deterrent. National pool players are required to provide whereabouts to USADA and testing agencies.
- **Collegiate level:** The NCAA began testing in 1986 at postseason football games and championship events at Division I, II, and III levels. The NCAA conducts year-round drug testing on the campuses of NCAA Division I and Division II member schools and on the campuses of Division III schools that sponsor Division I or Division II sports.
- **Major League Baseball:** All players are randomly selected for steroid testing both in and out of season; testing for drugs of abuse is on a basis of reasonable cause. Amphetamines are tested randomly during the season.
- **National Basketball Association:** Amphetamine and its analogs, cocaine, LSD, opiates, PCP, marijuana, and steroids are prohibited. Individual players can be tested with "reasonable cause" for prohibited substances without prior notice.
- **National Football League:** Currently tests both in and out of season in an unannounced fashion for "street drugs," anabolic steroids, amphetamines; may also test with "reasonable cause."
- Beta-blockers are prohibited during competition in specified sports, particularly target-shooting sports.

Effectiveness: The effectiveness of drug testing in preventing drug abuse by athletes is difficult to evaluate. In the 2005 NCAA study, 54.6% of those surveyed agreed that drug testing by individual colleges has deterred college athletes from using drugs, and 60.3% agreed that NCAA drug testing has deterred college athletes from using drugs. It is difficult to reconcile the disparity between the lack of positive drug tests (less than 1% in Olympics testing and less than 2.5% in NCAA testing) and the presumed larger prevalence of drug use by athletes. Many moral and ethical questions remain to be answered about drug testing, especially for college athletes. The courts have upheld the legality of high school testing.

Announced versus NAN testing: The limitations of testing only at competitions are obvious, and sports organization have moved to NAN testing to deter drug use (Fig. 21-1).

Legal issues: This evolving aspect of law varies according to state, and many issues have yet to be fully resolved. It is prudent to consult local legal experts before embarking on a testing program. Several landmark cases have involved drug testing.

- 1987: As a result of a suit by Stanford athletes, a court ruled that drug testing violates student-athletes' right to privacy. The decision was overturned in 1994 by the U.S. Supreme Court.
- 1994: The U.S. Supreme Court rules that random drug tests violate the privacy rights of University of Colorado athletes, trainers, and cheerleaders.
- 1995: The U.S. Supreme Court rules that urine drug screening of junior high and high school student athletes is allowable. Court states that minors are not protected by Fourth Amendment rights to privacy like adults, and "individualized suspicion" is not necessary to conduct drug testing of athletes.
- 2002: The U.S. Supreme Court ruled in the Earls case that students possess limited privacy rights, the intrusion of drug

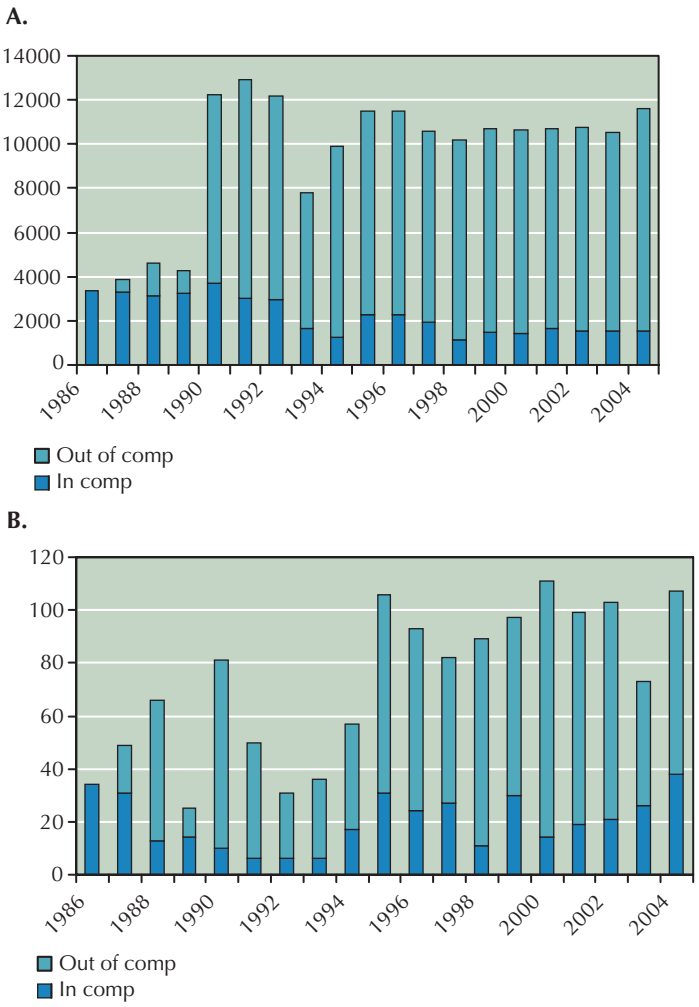

Figure 21-1 NCAA drug testing. **A,** Expansion of NCAA testing program from competition to out-of-competition drug testing program, 1986-2005. **B,** Summary of positive results in competition and out-of-competition testing programs. *(Data from the National Collegiate Athletic Association.)*

testing was not significant, and the government had a legitimate interest in deterring drug use.

Guidelines for drug testing: The NCAA has suggested guidelines for member institutions considering a drug-testing protocol. Although not obligated to institute separate programs, the university must follow its own guidelines once the drug-testing program has begun.

THERAPEUTIC USE EXEMPTIONS (TUEs)

- A therapeutic use exemption (TUE) is an approval given to an athlete to use a medication that is normally prohibited in his or her sport by the restrictions of a doping control program.
  - Must be applied for prior to competition during which it will be needed.
  - Retroactive approval is considered only in case of emergency treatment of an acute condition or exceptional circumstances.
- The athlete must have a documented need for the prohibited substance or method and would experience serious adverse consequences if the use of the medication or method was not allowed.
- There must be no reasonable alternatives to the use of the prohibited substance or method.
- The approval of the TUE would not allow use of a prohibited substance or method to enhance performance above what would be found if the athlete returned to normal health.

DRUG EDUCATION

- **Education of athletes** can be used to deter drug abuse. But to be effective, it must be started at a young age.
  - The 2005 NCAA study showed that the percentage of student-athletes using ergogenic drugs during high school or before increased significantly. Reversing a previous trend, more than half of the cocaine users indicated having first tried cocaine in high school or before. The percentage of student-athletes trying alcohol, marijuana, or cigarettes in junior high or before also increased.
  - For the majority of athletes, education begun at the collegiate level is probably too late for primary prevention.
- Educational intervention is probably best suited for deterring the use of **recreational drugs,** which usually have a negative impact on performance.
- Although education may alert athletes to the risks of **performance-enhancing substances,** the positive benefits of improved performance make drug testing a necessary component of deterrence.
- **Substance abuse patterns of athletes are constantly changing,** depending on current social practices and technologic advances. Sports medicine professionals have the obligation to keep abreast of such changes.

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Eating Disorders in Athletes

22

Lauren Costello and Kirtida Patel

ANOREXIA NERVOSA (AN)

DSM Criteria

- Essential features and diagnostic criteria for anorexia nervosa (Fig. 22-1):
  - Individual refuses to maintain a minimally normal body weight, is intensely afraid of gaining weight, and exhibits a significant disturbance in perception of the shape or size of his or her body.
  - Weight loss leading to body weight less than 85% of expected; or failure to make expected weight gain during period of growth leading to body weight less than 85% of that expected.
  - Primary (no menses by age 16 with normal secondary sex characteristics) or secondary amenorrhea (absence of at least three consecutive menstrual cycles).
- Subtypes:
  - Restrictive type only—no binging or purging.
  - Binge-eating/purging type—during episodes of anorexia nervosa the individual regularly engages in binge eating or purging behaviors.
- **Characteristics:** Strong need to maintain sense of control, concrete thinking, limited social spontaneity, perfectionism, preoccupation with food (discusses food excessively, prepares meals for others yet limits own intake, and restricts certain categories, such as elimination of all fats), excessive or compulsive exercise (exercise is excessive when it interferes with important activities, when it occurs at inappropriate times or in inappropriate settings, or when an individual continues to exercise despite injury or other medical complication and has been advised to refrain) (Fig. 22-2).
- **Comorbidity:** Depressive disorders, obsessive compulsive disorder (OCD), personality disorders.

Medical Complications of Anorexia Nervosa

Cardiovascular: Cardiac complications are the most common cause of death; the mortality rate is about 10%.

- **Hypotension and bradycardia:** Systolic pressures as low as 70 mm Hg and sinus bradycardia with heart rates as low as 30 to 40 beats per minute. These changes are a response to a decrease in basal metabolic rate. These changes are physiologic cardiovascular responses.
- **Arrhythmias:** *EKG shows evidence* of sinus bradycardia, ST-segment elevation, T wave flattening, low voltage, and rightward QRS axis may be present. Of most concern is the frequency of rhythm disturbances, mainly QT interval prolongation that may be an indication for those at risk for cardiac arrhythmias and sudden death.
- **Cardiomyopathy:** Can result from excessive, aggressive refeeding or ipecac use.

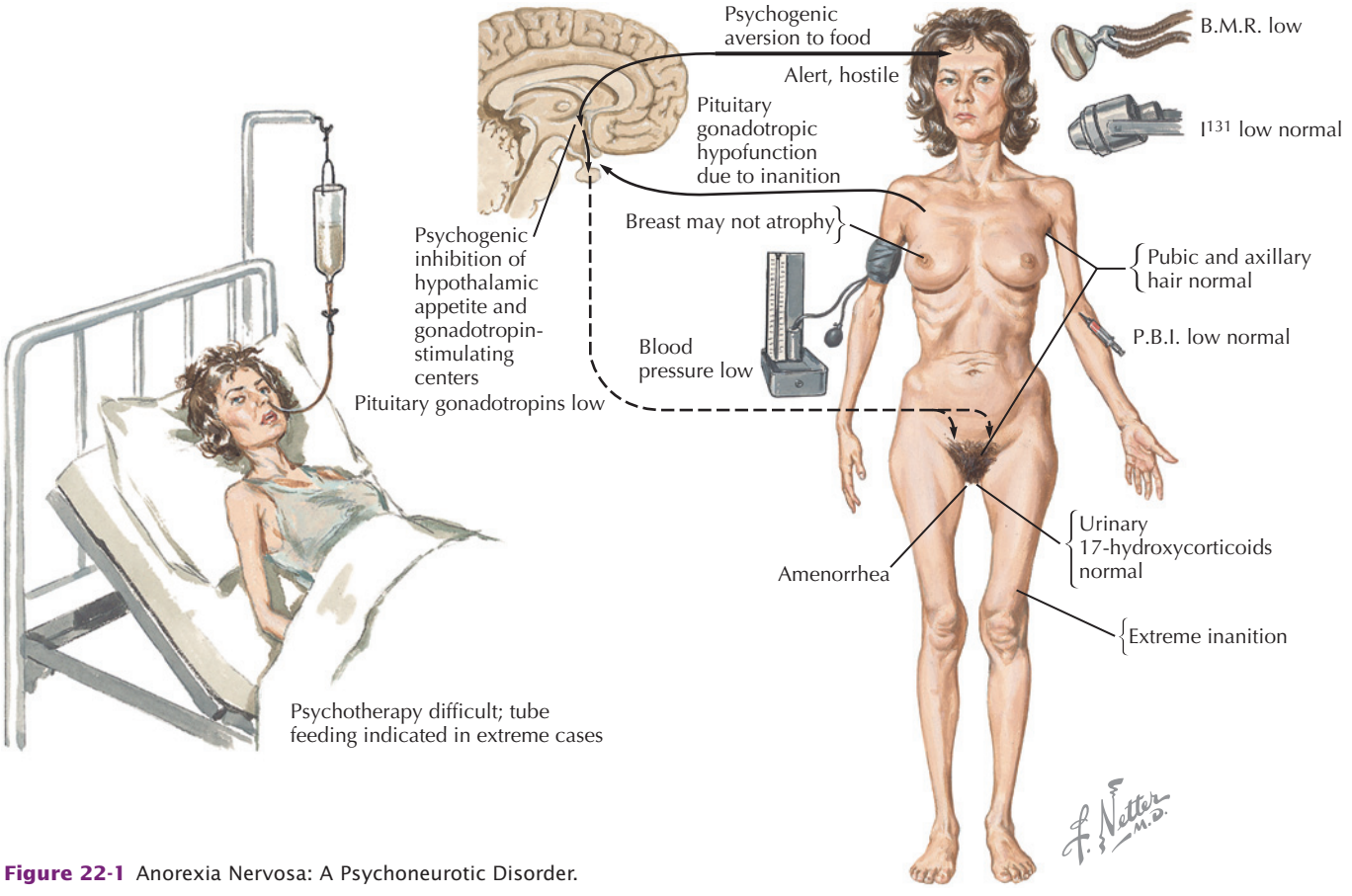

Figure 22-1 Anorexia Nervosa: A Psychoneurotic Disorder.

Figure 22-2 Distorted Body Image.

Endocrine and Metabolic:

- **Amenorrhea:** Results from disorders in the hypothalamic-pituitary-ovarian axis (Fig. 22-3). *Levels* of follicle-stimulating hormone (FSH) and luteinizing hormone (LH) are low despite low levels of estrogen. Amenorrhea persists in 5% to 44% of patients in whom weight gain has been documented. Marginal energy intake coupled with high energy expenditure may result in neuroendocrine adaptations to an overall metabolic deficit. Leptin may be an important indicator of nutritional status and may also mediate reproductive function by responding to an altered metabolic state that is characteristic of individuals who have exercise-associated amenorrhea.
- **Electrolyte imbalance:** Hypokalemia, hyponatremia, hypomagnesemia, hypophosphatemia (especially with refeeding).
- **Euthyroid sick syndrome:** Thyroid function is also affected, revealing a decrease in triiodothyronine (T3), thyroxine (T4), and an increase in reverse T3. These changes are characteristic of the euthyroid sick syndrome. No treatment is required.
- **Osteopenia/Osteoporosis:** Researchers traditionally attributed bone loss to estrogen deficiency in women who have hypothalamic amenorrhea. We now understand that undernutrition and its metabolic consequences appear to directly reduce bone turnover and, more important, bone formation, thereby causing osteopenia. Dysfunctional eating patterns and leptin effects may also have a role in the metabolic signals suppressing GnRH and the pathogenesis of osteopenia despite normal body weight. Also contributing to bone loss are low levels of progesterone (accelerates remodeling) formation and decreased insulin-like growth factor-1 (IGF-1) levels.
- Hypothermia
- Hypoglycemia
- Diabetes insipidus

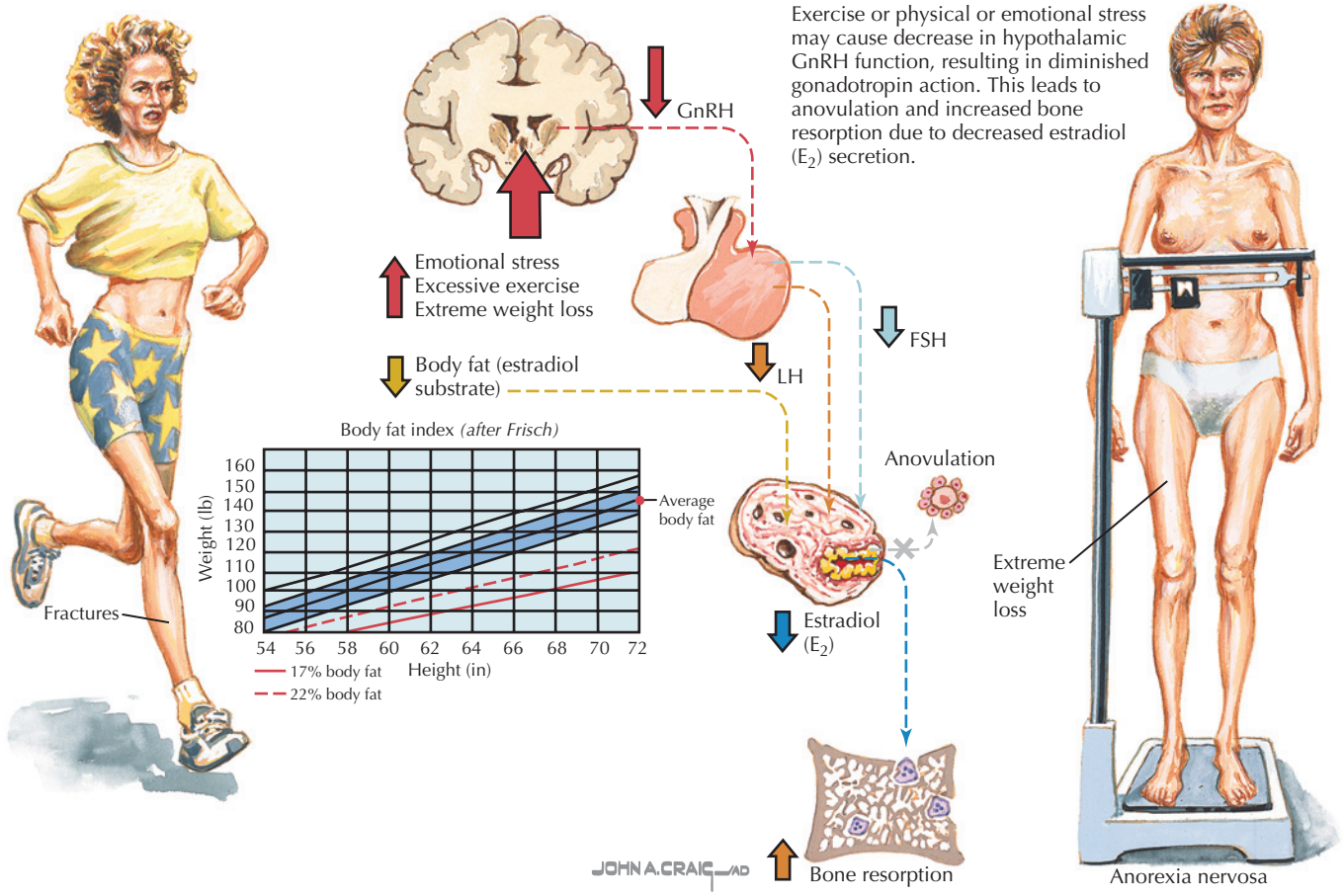

Figure 22-3 Osteoporosis Associated with Amenorrhea.

Gastrointestinal: Constipation, delayed gastric emptying, decreased intestinal motility.
Hematologic: Anemia, leucopenia, thrombocytopenia.
Integumentary: Dry skin and hair, lanugo, nail fragility.
Neurologic: Cerebral atrophy, ventricular enlargement.
Reproductive: Infertility, low birth weight infant.

BULIMIA NERVOSA (BN)

DSM Criteria

- Essential features and diagnostic criteria for bulimia nervosa:
  - Binge eating and inappropriate compensatory methods to prevent weight gain. In addition the individual's self-evaluation is excessively influenced by body shape and weight.
  - The binge eating and inappropriate compensatory behaviors must occur on average at least twice weekly for 3 months.
  - A binge is characterized by eating, in a discrete period of time, an amount of food that is definitely larger than most people would eat.
  - Lack of control over eating during these episodes—cannot stop.
  - Recurrent inappropriate compensatory behaviors in order to prevent weight gain (e.g., self-induced vomiting; misuse of laxatives, diuretics, enemas, or other medications; fasting; or excessive/compulsive exercise).
- Subtypes:
  - Purging type—regularly engaged in self-induced vomiting or misuse of laxatives, diuretics, or enemas.
  - Nonpurging type—use other inappropriate compensatory behaviors such as fasting, excessive exercise, but no purging.
- **Characteristics:** Generally of normal weight, but may be slightly overweight or underweight. Individual may conceal food or hoard food for binges, which usually occur in secret, and may be planned or spontaneous. Low self-esteem, anxiety/depressive symptoms, substance abuse.
- **Comorbidity:** Mood disorders (dysthymic and major depressive disorder); personality disorders; substance abuse/dependence. Among patients exhibiting symptoms of bulimia, substance abuse has been related to an increased incidence of attempted suicide, stealing, and promiscuity. Several studies have reported high rates of kleptomania among patients with eating disorders.

Medical Complications of Bulimia Nervosa

Cardiovascular: Arrhythmias, hypertension (with use of diet pills).
Endocrine and metabolic: Menstrual irregularities, *less common than in patient's with anorexia nervosa*. Electrolyte imbalance—compulsive vomiting can cause hypokalemia, hypomagnesemia, and metabolic alkalosis. Pseudo–Bartter syndrome (i.e., normotensive hypokalemic alkalosis) is common among patients who vomit or use diuretics excessively. Hyperchloremic metabolic alkalosis suggests excessive laxative use.
Gastrointestinal: Enlarged salivary glands, constipation, gastritis, esophageal dysmotility patterns (this includes gastroesophageal reflux and, rarely, Mallory-Weiss or gastric tears), postbinge pancreatitis.
Integumentary: Russell's sign: Scarring and calloused areas on the dorsum of the index and middle finger from self-induced vomiting.
Neurologic: Cerebral hemorrhage (with use of diet pills).
Orofacial: Dental caries, dental erosion, enlarged parotid glands.
Respiratory: Pneumomediastinum.

EATING DISORDER NOT OTHERWISE SPECIFIED (EDNOS)

- Meets some or most criteria for anorexia nervosa or bulimia nervosa, but does not meet the full criteria for either specific disorder. Causes a decrease in level of functioning.
- Subtypes
  - Binge eating disorder—recurrent episodes of binge eating in absence of regular use of inappropriate compensatory behaviors.
  - Repeatedly chewing and spitting out, but not swallowing, large amounts of food.
  - Anorexia athletica—fear of weight gain although lean.
    - Weight is 5% or more below expected weight.
    - Muscular development maintains weight above anorexic threshold.
    - Restricted caloric intake—may be broken by binges.
    - Excessive or compulsive exercise.
    - Menstrual dysfunction—may include delayed puberty.
    - GI complaints.

PREVALENCE/ETIOLOGY

- Anorexia nervosa: Lifetime prevalence in the general population is approximately 0.5% to 1%.
- Bulimia nervosa: Lifetime prevalence in the general population is approximately 1% to 3%.
- A large study of elite athletes, both male and female, found an overall prevalence of eating disorders (AN, BN, and EDNOS) of 13.5% in the elite athletes and 4.6% in a control group of nonathletes.
- Male versus female: More than 90% of cases occur in females. However, it should be emphasized that males represent approximately 10% of anorexia nervosa cases, a fact that often is overlooked.
- Athletes participating in sports that emphasize leanness or sports that have specific weight limits are more likely to develop an eating disorder. This is true for both male and female athletes.
- Female athletes are especially at risk in sports such as gymnastics, ballet, figure skating, and distance running. Males in sports such as bodybuilding, wrestling, and light-weight crew are also at greater risk.
- Twin and family studies suggest a substantial genetic influence for development of eating disorders and disordered eating, specifically through serotonergic pathways.

RISK AND TRIGGER FACTORS IN ATHLETIC POPULATION

- Early dieting, prolonged dieting, weight fluctuations, early sports-specific training, traumatic events (injuries), participating in sports emphasizing leanness or weight-controlled sports.
- The "athletic personality"—characteristics include goal orientation, perfectionism, compulsiveness and ability to block distractions, and rechanneling athletic drive from sport into eating.
- Extreme exercise appears to be a risk factor for developing anorexia nervosa, especially when combined with dieting. There has also been evidence that AN patients with excessive physical activity constitute a subtype of the disorder with strong links to OCD.

TREATMENT

Medical Monitoring/Treatment

- During treatment, it is important to monitor the patient for shifts in weight, blood pressure, pulse, other cardiovascular parameters, electrolytes, and behaviors likely to provoke physiologic decline and collapse (Box 22-1).
- Clinical consensus suggests that for underweight patients, realistic weight gain targets are 2 to 3 pounds a week for hospitalized patients and 0.5 to 1 pound a week for individuals in outpatient programs.
- There is no approved indication, at this time, for the use of bisphosphonates such as alendronate in patients with amenor-

BOX 22-1 *Laboratory Assessments for Patients with Eating Disorders*

Laboratory studies
CBC with differential
Complete metabolic panel
Liver function tests
Thyroid function tests
HCG, FSH, LH, prolactin, estradiol
ESR
Drug screen
Urinalysis
Amylase (if purging)
Other studies
ECG
Bone density test (DEXA) (if indicated)
Echocardiogram (if indicated)
Chest or abdominal x-ray (if indicated)

rhea and low bone density. When dietary intake is inadequate for growth and maintenance, daily supplementation with calcium (1000 to 1500 mg) and vitamin D (400 IU) should be considered.
- Although hormone replacement (HRT) is frequently prescribed to improve bone density, no good supporting evidence exists either in adults or in adolescents to demonstrate its efficacy. Before estrogen is offered, it is recommended that efforts be made to increase weight and achieve resumption of normal menses.
- Weight gain should not be excessive because rapid refeeding can lead to cardiovascular collapse, hypophosphatemia, and dangerous fluctuations in potassium, sodium, and magnesium levels. Attempts should be made to increase daily caloric intake slowly by 200 to 300 kcal every 3 to 5 days until a sustained weight gain of 1 to 2 pounds per week is achieved.
- Promotility agents such as metoclopramide may be useful for bloating and abdominal pains that occur during refeeding in some patients.
- Patients with history of purging behaviors should also be referred for a dental examination.
- Physical activity should be adapted to the food intake and energy expenditure of the patient, taking into account the patient's bone mineral density and cardiac function and ability to gain weight while still exercising. Activity may need to be decreased or stopped all together.

Additional Treatment

- Multidisciplinary team: Psychologist, physician, and a nutritionist preferably trained in subspecialty of eating disorders. Including certified athletic trainer (ATC) as part of the team is also recommended. Coaches, teammates, and family may also need to be involved when appropriate.
- Medications
  - Anorexia nervosa: Selective serotonin reuptake inhibitors (SSRIs) are ineffective in hastening weight gain but may be used to treat comorbidities (e.g., depression/anxiety/OCD). Atypical antipsychotic agents at low dose (e.g., olanzapine, at a dose of 2.5 to 10 mg daily) may improve weight gain, depression, obsessive thoughts, but controlled studies are lacking.
  - Bulimia nervosa: Fluoxetine, at a dose of 60 mg daily, is FDA-approved for the treatment of BN. Patients showed decrease in binge eating/purging frequency, but rarely attain abstinence.
- Psychotherapy:
  - Various techniques are used in the treatment of eating disorders. Cognitive behavioral therapy (CBT) appears to be one of the more effective treatment options for acute episodes of bulimia. This treatment is based on the idea that feelings and behaviors can be changed based on evaluating cognitive distortions and core beliefs and learning new coping mechanisms. Interpersonal psychotherapy (IPT) may be another option in those who do not respond to CBT.
  - Psychodynamic/psychoanalytic approaches may be incorporated once bulimia symptoms improve. These approaches address developmental issues. Family therapy should be considered whenever possible.
  - Treatment of patients with acute anorexia nervosa with low weight should be supportive and aimed at weight restoration. Traditional family therapy may also be useful. Once weight is reasonably restored, more structured psychotherapy is useful, especially CBT and group therapy. Nonverbal interventions such as art therapy, yoga, and meditation are often an adjunct to therapy. Recent approaches in the adolescent patient, such as the Maudsley Method, emphasize separating the patient from the illness and assisting the family in taking on the problems of anorexia nervosa without attacking their child.
- Level of care:
  - Treatment options include outpatient, partial hospitalization (full-day outpatient care), residential, or inpatient hospitalization (Box 22-2). For full criteria see the American Psychiatric Association's *Practice Guidelines for the Treatment of Patients with Eating Disorders* (see Recommended Readings).
  - Sports participation and symptomatic athletes: Decisions regarding sport participation while the student-athlete is symptomatic should be made on a case-by-case basis. Potential guidelines may be found in the National Collegiate Athletic Association handbook "Managing the Female Athlete Triad" and the International Olympic Committee's "Consensus Statement on the Female Athlete Triad."

PREVENTION

- Emphasize the role of overall long-term good nutrition in optimizing athletic training and performance.
- Do not overly focus on the effect of lower body weight on athletic performance.
- Encourage coaches to *not* weigh their athletes.
- Be aware of behaviors associated with eating disorders—constant preoccupation with food/calories/dieting; purposeless, excessive physical activity that is not part of training; extreme weight fluctuations; secretive eating; avoidance of eating with others; vomitus in restroom—and refer athletes for professional help.
- Educate coaches on the role they may play in helping or hindering the development of eating disorders.

BOX 22-2 *Indications for Hospitalization*

Adults: heart rate <40 bpm
Children: heart rate <50 bpm
Adults: blood pressure <90/60 mm Hg
Children: blood pressure <80/50 mm Hg
Temperature <36.1° C (97.0° F)
Potassium <3 mmol per liter
Symptomatic hypoglycemia
Severe dehydration
Weight <75% of expected weight
Rapid weight loss of several kilograms within a week
Lack of improvement or rapid worsening while in outpatient treatment

RECOMMENDED READINGS

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Overtraining

23

Edward Josiah Lewis, Thomas Howard, and Francis G. O'Connor

OVERVIEW

Introduction

- Overtraining syndrome is a medical disorder of athletes that is complicated by many diagnostic and therapeutic challenges. Current research in this area is limited by a small number of studies and inconsistent results. Several researchers have concluded the following: there are poorly established diagnostic criteria; there are significant confounding influences, including illness, injury, menstruation, and unique training methods for different sports; and it is difficult, if not impossible, to establish controls and/or lab models to study the illness.
- This chapter will review the epidemiology and key terminology and will describe the proposed pathophysiology of overtraining syndrome. Most important, the clinical presentation, diagnosis, management, and prevention of this disorder will be discussed.

Epidemiology

- Overtraining syndrome is a problem that has plagued athletes, trainers, coaches, and clinicians for generations. As evidenced by this quote from Dr. D. C. Parmenter, this ailment is not new to the medical literature:
  - "Overtraining or staleness is the bug-a-boo of every experienced trainer…a condition difficult to detect and still more difficult to describe…evaluation should focus on training load, nutrition, sleep and rest, competition stress and psychological state." (Parmenter)
- The current literature on overtraining syndrome has limited insight into the incidence and prevalence of this disorder. There are estimates that 7% to 20% of elite athletes at any one time may be afflicted by overtraining syndrome. It is additionally thought that up to two-thirds of runners during an athletic career will experience the signs and symptoms of overtraining. Overtraining fatigue may be more prevalent in amateur athletes than elite athletes, with participants in endurance events such as running, cycling, and swimming thought to be at greatest risk.
- Overtraining susceptibility is thought to occur in highly motivated, goal-oriented individuals. In addition, those athletes who design their own exercise regimens may be at greater risk. The risks of overtraining syndrome are significant and include prolonged poor performance, injury, illness, and premature retirement.

Terminology

Training: A series of stimuli used to stress or displace homeostasis to provide stimulation for adaptation. Training involves progressive overload in an effort to improve performance in sport or activity. Training for success involves a balance between achieving peak performance and avoiding the negative consequences of overtraining.

Adaptation: Physiologic response to stress that results in an adjustment in function or dimension of an organism.

Recovery: The period of time following a training stimulus when the work capacity returns to prestimulus levels. If recovery time is optimal, supercompensation results, whereas excessive training, without rest, can result in a decreased performance (Fig. 23-1). The literature describes four components of recovery: hydration and nutrition; sleep and rest; relaxation and emotional support; stretching and active rest. Recovery that is inadequate results in fatigue. Fatigue may be classified as either pathologic or physiologic.

- Physiologic fatigue includes categories such as insufficient sleep, nutritional, jet lag, pregnancy, and training-induced (from either excessive competition or overreaching).
- Pathologic fatigue includes the following categories: medical including infectious, neoplastic, hematologic, endocrine, toxic, iatrogenic, and psychiatric; chronic fatigue syndrome; and overtraining syndrome.

Periodization: Planned sequencing of increased training loads and recovery periods within a training program.

Overreaching/Overwork: Acute phase during which training load (intensity or volume) is significantly increased. It is believed that occasional periods of heavy training sufficient to depress performance capacity may result in larger ultimate levels of performance when the training load is reduced. Short-term deterioration in performance generally lasts less than 2 weeks.

Overtraining: Maladaptive response to training from an extended period of overload (usually > 2 weeks); the result of too severe or prolonged a period of overreaching (see Fig. 23-1). Overtraining is manifested by decreased sport-specific performance, enhanced fatigability, pronounced vegetative complaints, sleep disorder, emotional instability, organ-related complaints without organic disease, overuse injuries, blood chemistry changes, and immune dysfunction.

PATHOPHYSIOLOGY

Overview

- The pathophysiology of overtraining syndrome has not been fully elucidated. There are multiple models/hypotheses that attempt to explain the signs and symptoms of this disorder including the following: the autonomic nervous system model; the neuroendocrine model; the glycogen/glutamine/branched-chain amino acid hypotheses; and the cytokine hypothesis.
- Figure 23-2 is one attempt to unite the various hypotheses to explain the complexity of this disorder.

Autonomic Hypothesis

- Autonomic dysfunction, as it relates to overtraining, was classified into two forms by Israel in 1958: a sympathetic, or Basedowian, form, and an Addisonoid, or parasympathetic,

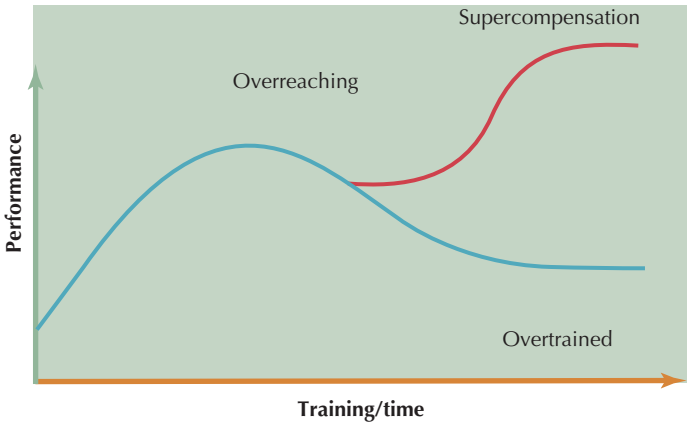

Figure 23-1 Hypothetical Overtraining Syndrome Model.

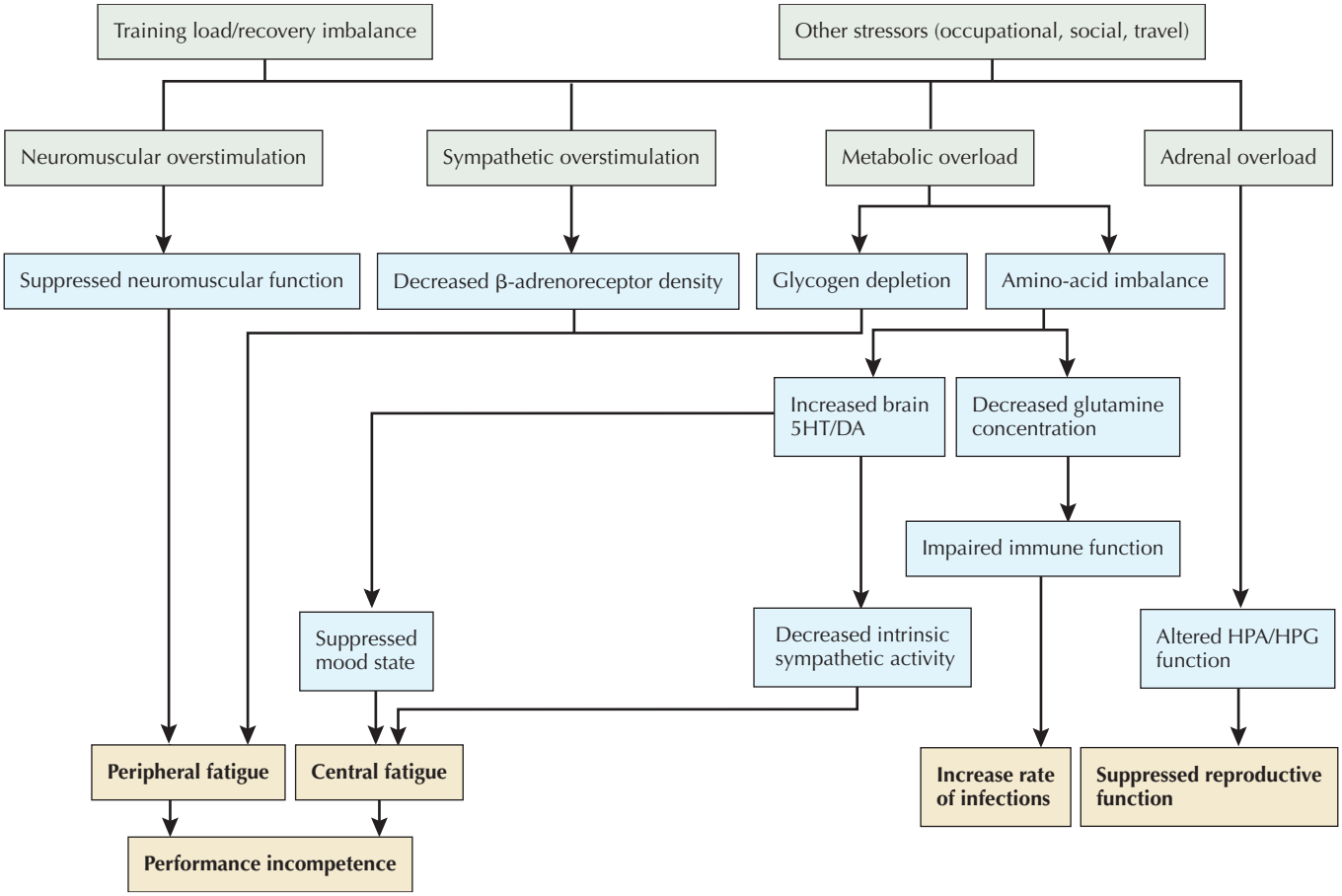

Figure 23-2 Genesis of overtraining syndrome. 5-HT: Serotonin, DA: Dopamine. *(Modified with permission from Foster C, Lehmann M: Overtraining syndrome. In Guten GN (ed): Running Injuries. Philadelphia: W.B. Saunders, 1997.)*

form. The sympathetic form refers to morbus Basedow (hyperthyroidism) and is marked by symptoms consistent with increased adrenergic and noradrenergic stimulation. The Addisonoid form mimics the symptoms of adrenal insufficiency and is marked largely by vegetative symptoms (Box 23-1).

- The two forms of autonomic dysfunction represent different points in the overtraining continuum, with sympathetic predominance occurring early, followed by a prolonged parasympathetic predominance. Practically speaking, the sympathetic form is more rarely seen in the clinical setting and has been less well studied, with most descriptions being anecdotal. Other than an increase in baseline heart rate, symptoms of parasympathetic activation predominate in most patients.
- Objective findings that support the autonomic hypothesis of overtraining include:
  - Decreased adrenal responsiveness to adrenocorticotropic hormone (ACTH) in overtraining syndrome
  - Decreased cortisol levels
  - Early increase in pre-exercise and post-exercise catecholamine levels
  - Late decrease in basal urinary catecholamine excretion
  - Decreased beta-receptor density in endurance athletes
- The decreased sensitivity of the body to sympathetic pathways may be seen as a protective negative feedback mechanism after recurrent exposure to catecholamines released during exercise. Parasympathetic overtraining may thus represent the result of negative feedback in response to sustained or repeated arousal without adequate rest from stimulating stressors. This model also seems to explain why non–exercise-related life stressors make athletes more prone to developing overtraining syndrome.

BOX 23-1 *Autonomic Dysfunction Symptomatology*

Sympathetic/Basedowian symptoms
Agitation or jitteriness
Increased heart rate and BP
Weight loss
Insomnia
Parasympathetic/Addisonoid symptoms
Depression
Fatigue
Decreased libido
Hypersomnolence
Somatic symptoms (myalgias)

Neuroendocrine Hypothesis

- Overtraining syndrome may affect change in any portion of the hypothalamic-pituitary-adrenal (H-P-A) axis. Multiple studies have delineated altered concentrations of anterior pituitary hormones, or their downstream products.
- Hormonal findings include:
  - Suppression of the H-P-A axis with blunted response to ACTH and lower cortisol levels
  - Lower growth hormone peaks
  - Suppressed thyroid-stimulating hormone (TSH) secretion
  - Suppressed luteinizing hormone (LH) secretion

  - Suppressed free testosterone-to-cortisol ratio
  - Decreased prolactin secretion
  - Increased norepinephrine and epinephrine levels
- The multiple endocrine abnormalities associated with overtraining syndrome have yet to be completely elucidated and likely vary along the continuum of overtraining syndrome.
- The neuroendocrine hypothesis and hormonal changes associated with overtraining syndrome are not distinct from the autonomic and cytokine theories of overtraining syndrome, but rather are interrelated and in some cases secondary to the autonomic and inflammatory changes.

Glutamine Hypothesis

- Glutamine is the most abundant amino acid in muscle and plasma and is synthesized in muscle, lungs, liver, brain, and fat tissues. Glutamine is an important nutrient source for monocytes, lymphocytes, and natural killer cells, and has been demonstrated to be found in decreased plasma concentrations in overtrained athletes. Although not classically thought of as immunocompromised, highly stressed and overtrained athletes demonstrate an increased susceptibility to infectious disease. Specifically, there is a demonstrated increase in the rate of upper respiratory infections in endurance athletes during times of intense training not followed by adequate rest.
- It has been demonstrated that secretory immunoglobulin A (IgA) levels are decreased in overtrained athletes and that serum IgA concentrations are decreased in overtrained swimmers. Natural killer cell cytotoxicity has also been demonstrated to be lower in overtrained athletes. The glutamine hypothesis proposes that it is this decreased concentration of glutamine that is therefore responsible for the increased upper respiratory infection rate in endurance athletes.

Branched-Chain Amino Acid (BCAA) Hypothesis

- The branched-chain amino acid (BCAA) hypothesis theorizes that a driving feature in overtraining syndrome is increased central levels of 5-hydroxytryptamine (5-HT), or serotonin, which subsequently leads to central fatigue.
- According to this theory, glycogen depletion after intensive exercise necessitates the utilization of alternative energy sources by the musculature. BCAAs, including leucine, isoleucine, and valine, are oxidized to glucose with a concurrent rise in the level of fatty acids. The fatty acids compete with tryptophan for albumin-binding sites, displacing tryptophan and increasing its plasma concentration. After passing through the blood brain barrier, tryptophan is converted into 5-HT, or serotonin, which may cause centrally modulated fatigue in increased concentrations.

Glycogen Hypothesis

- Glycogen is the predominant energy source for moderate to intense exercise.
- According to the glycogen hypothesis, glycogen depletion may lead to overtraining through three different mechanisms:
  - Glycogen depletion may contribute directly, as low levels of muscle glycogen stores may cause muscular fatigue and poor performance.
  - Glycogen depletion may also cause increased oxidation of BCAA, leading eventually to central fatigue.
  - Glycogen depletion may lead to a net negative caloric state, inducing a catabolic state and multiple neuroendocrine changes.

Cytokine Hypothesis

- The cytokine hypothesis theorizes that through chronic tissue injury without regenerative healing, a systemic inflammatory and immune response occurs. This systemic inflammatory response leads to increased concentration of interleukins, interferons, tumor necrosis factor, and other proinflammatory factors.
- These factors are thought to act centrally, promoting central fatigue, anorexia, depression, a catabolic state, and changes in the hypothalamic-pituitary-adrenal axis and the hypothalamic-pituitary-gonadal axis.

CLINICAL PRESENTATION

Overview

- Although a universal feature of overtraining syndrome is decreased performance, it may include a broad array of psychiatric, musculoskeletal, cardiovascular, and immunologic symptoms (Box 23-2). Because of individual variation, however, few if any athletes exhibit all features of overtraining.
- Training volume, training intensity, or lack of rest, alone, may not explain many or even most cases of overtraining syndrome. Rather, overtraining syndrome commonly develops when the cumulative stress imposed on an organism outstrips its ability to cope with that stress. Contributing factors can include the training regimen, rest status, nutritional state, concurrent infectious disease, allergic reactions, and financial and relationship stress.

Psychological Features

- Fatigue
- Anhedonia, including loss of appetite or libido
- Anxiety, irritability, or anger
- Insomnia or hypersomnolence
- Depression

Cardiovascular Features

- Increased first A.M. resting heart rate (increase of 5 to 10 beats per minute over baseline is suggestive)
- Reduced maximum heart rate and $\dot{V}O_2$ max
- Decreased stroke volume and altered contractility
- Possible relative decrease in plasma volume

BOX 23-2 *Clinical Presentation of Overtraining Syndrome*

Sport-Specific performance complaints
Inability to meet prior performance standards
Prolonged recovery time
Decreased coordination
Decreased muscular strength
Physiologic findings
Blood pressure changes
Increased resting heart rate
Weight loss
Increased incidence of injuries
Increased incidence of infections
Amenorrhea
Subjective complaints
Fatigue
Feelings of depression
Anorexia
Hypersomnia/disturbed sleep
Myalgias
Gastrointestinal disturbances
Headaches
Increased irritability
Concentration difficulties
Apathy

Musculoskeletal Features

- Muscular fatigue at previously tolerated exercise levels
- Decreased performance
- Persistent soreness

Immune Features

- Demonstrated increase in the rate of upper respiratory infections in endurance athletes during times of intense training not followed by adequate rest
- Decreased serum and secretory IgA
- Decreased natural killer cell function

DIAGNOSIS

Differential Diagnosis

- The diagnosis of overtraining syndrome is made clinically—there is no currently available confirmatory laboratory test. Accordingly, **overtraining syndrome is considered a diagnosis of exclusion.**
- The differential diagnosis of overtraining is extremely broad and should be considered before a diagnosis of overtraining syndrome is established. Infectious, inflammatory, malignant, endocrine, cardiopulmonary disease, renal disease, hematologic disease, myopathies, and psychiatric disease should all be considered as should substance abuse (Box 23-3 and Figs. 23-3 and 23-4).
- A broad lab workup may be necessary to exclude many of the above conditions. The authors' suggested evaluation strategy is demonstrated in Figure 23-5.

BOX 23-3 *Differential Diagnosis of Overtraining Syndrome*

Infectious etiologies
Postviral syndrome
Infectious mononucleosis
Lyme disease
Viral hepatitis
Myocarditis
Collagen vascular disorders
Polymyalgia rheumatica
Systemic lupus
Fibromyalgia
Chronic fatigue syndrome
Metabolic
Hypothyroidism or hyperthyroidism
Anemia
Electrolyte disorders
Pharmacologic
Alcohol
Caffeine
Illegal or performance-enhancing drugs
Psychiatric
Depression
Physical abuse
Sexual abuse
Emotional abuse
Posttraumatic stress disorder
Malingering
Other
Cancer
Myopathy
Acquired nutritional problems
Pregnancy
Sleep deprivation

Laboratory Analysis

- Although there have been many elucidated hormonal and hematologic abnormalities in overtraining syndrome, there is no one characteristic set of abnormalities and many changes may represent points along a continuum.
- Clinical and laboratory markers that have been proven to have poor sensitivity and specificity in evaluating overtraining syndrome include body mass, resting heart rate and blood pressure, hematocrit, ferritin, creatinine kinase, and hormonal markers.
- Glutamine has recently been a frequent subject of study as a possible marker of overtraining. Glutamine, as discussed earlier, is an amino acid that is depleted in catabolic states such as overtraining, infection, surgery, and acidosis. Glutamine has been shown in multiple studies to be decreased in the overtrained state; however, there are multiple confounding factors, as discussed earlier, and glutamine testing is not yet widely available for practical use.

Neuropsychiatric Testing

- Many of the earliest symptoms of overtraining are psychiatric, and a number of screening questionnaires have been developed. One commonly used questionnaire that has been validated for overtraining syndrome screening and risk assessment is the Profile of Mood States (POMS).
- The POMS is a survey in which patients rate 65 adjectives on a 5-point scale in accordance with how well the adjective describes their feelings over the past week. The POMS is designed to assess tension/anxiety, depression/dejection, anger/hostility, vigor/activity, fatigue/inertia, and confusion/bewilderment. The test is summarized in one global score, which subtracts the sum of points assessed for positive mood states from that of negative mood states.
- Other commonly used standardized tests include the Total Quality Recovery score by Kentta and Hassmen, the Daily Analyses of Life Demands for Athletes, which is used by Australian Olympic teams, and the Psycho-Behavioral Overtraining Scale, which has been used by some British athletes.

MANAGEMENT

- Because overtraining syndrome should be considered a diagnosis of exclusion, and because assessing response to therapy requires time, the initial workup should be divided into two appointments, separated by 2 to 3 weeks. During the first appointment, the history and physical examination are completed, the diet evaluated, and the training diary reviewed. At this time a screening hematologic panel to evaluate for other causes of decreased performance may be obtained (see Fig. 23-5).
- The mainstay of treatment in overtraining syndrome is rest. The athlete's response to imposed rest is also critical to making an accurate diagnosis. After the initial visit, a period of 2 to 3 weeks of rest from training is recommended. Athletes may remain active, but not in their chosen sport, and training volume should generally be decreased by at least 50%.
- At the 2- to 3-week follow-up visit, the athlete may resume training if symptoms have resolved and the lab workup is within normal limits, but the training regimen should be closely assessed for appropriate periods of recovery. If the athlete's symptoms have not resolved at the follow-up visit, and the lab workup reveals no explanation, longer periods of imposed rest are necessary, while symptoms and mood scores are tracked.
- A multidisciplinary treatment approach is often useful, and may include sports psychology and nutrition consultations. Consideration should be given to all four components of recovery: hydration and nutrition; sleep and rest; relaxation and emotional support; stretching and active rest.

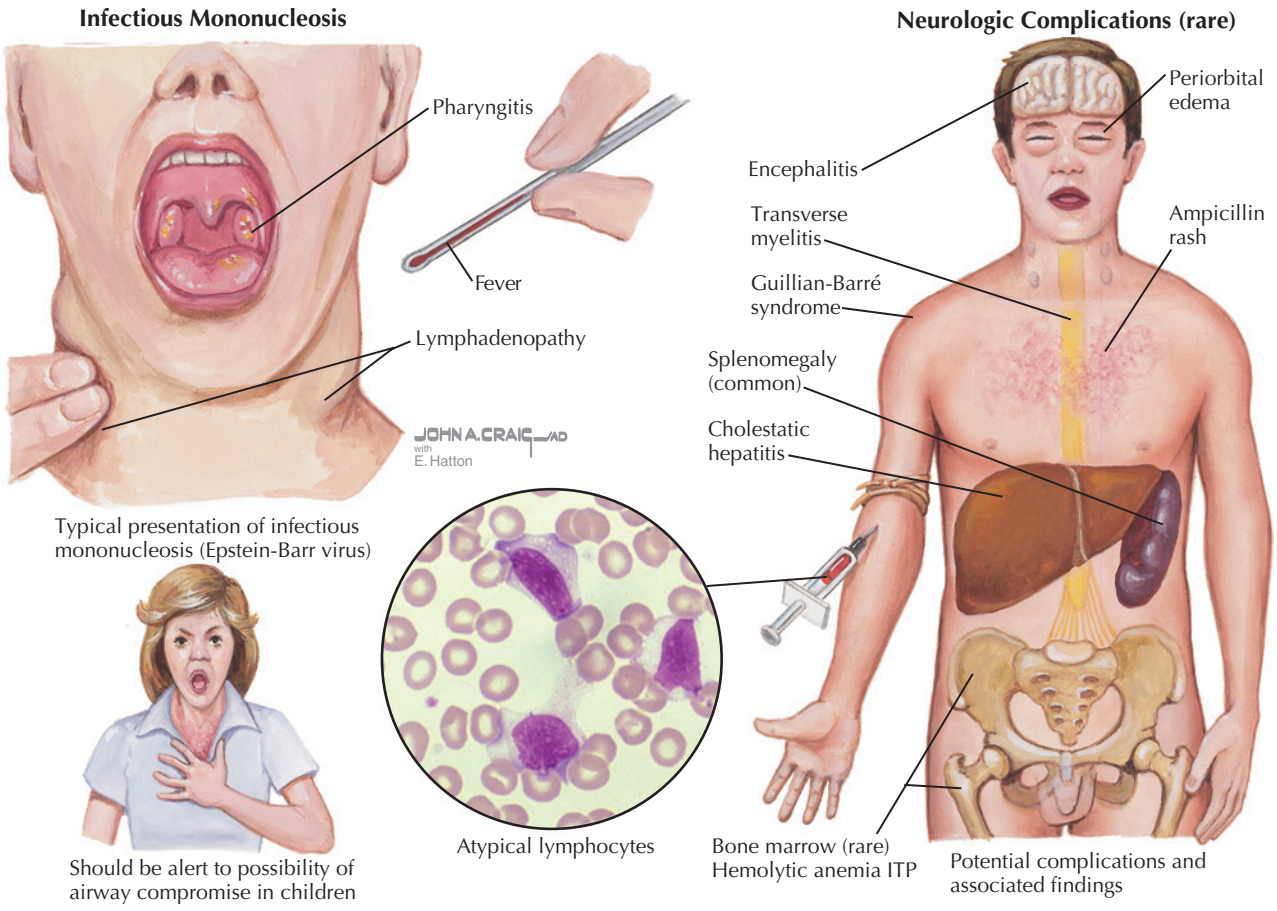

Figure 23-3 Infectious Mononucleosis.

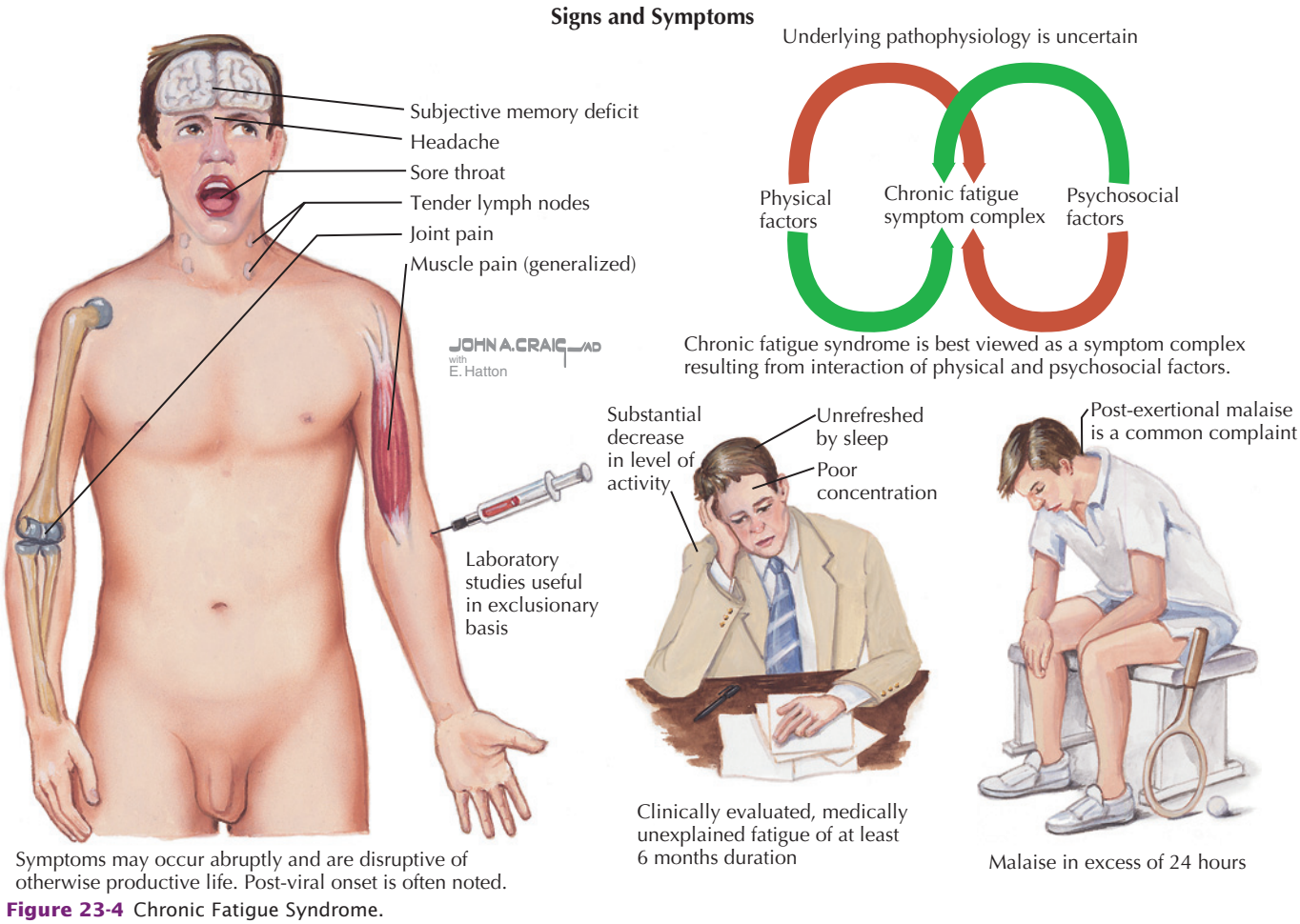

Figure 23-4 Chronic Fatigue Syndrome.

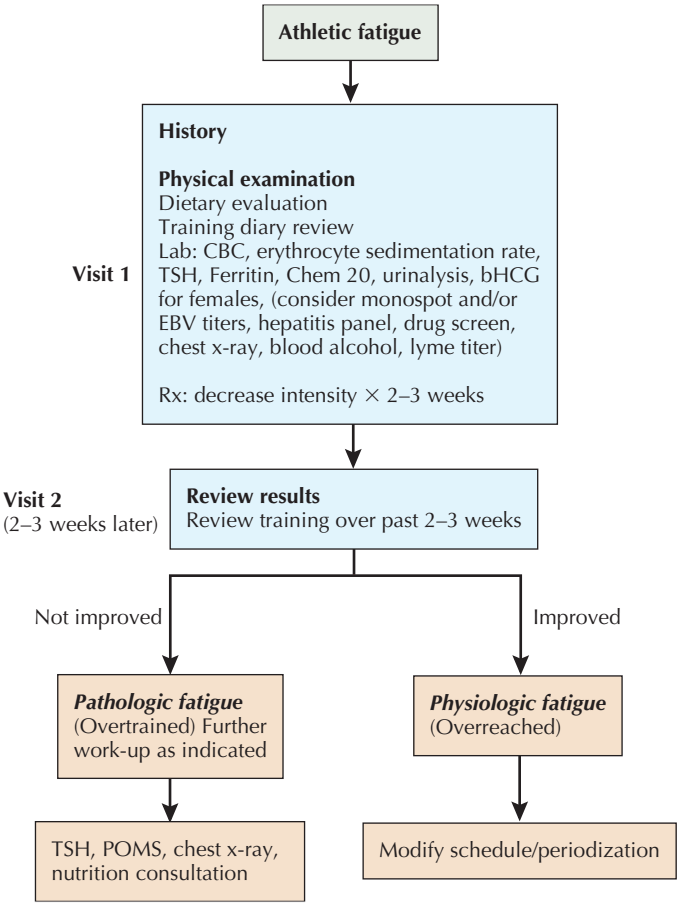

Figure 23-5 Proposed Clinical Evaluation of Athletic Fatigue.

- **Glutamine:** There may be a role in the future for enteral or parenteral glutamine supplementation**.** To date, no study has demonstrated improved immune function or recovery with glutamine supplementation in healthy athletes.
- **Branched-chain amino acids:** BCAA supplementation may improve subjective reporting of mood and energy, as well as positively affecting POMS scores. No definitive improvement in athletic performance has been demonstrated with BCAA supplementation. Supplementation with BCAA is often limited by gastrointestinal side effects, with possible poor tolerance of doses greater than 7 to 10 g daily.
- **Selective serotonin reuptake inhibitors (SSRIs) and tricyclic antidepressants (TCAs):** Numerous studies have demonstrated that dramatic increases in training volume lead to undesirable mood changes. Increases in fatigue, depression, anger, and global mood disturbances are common and may lead to clinical depression. Care should be taken prior to starting an SSRI or TCA if symptoms of fatigue predominate, because increased central serotonin has been hypothesized to cause central fatigue early in overtraining syndrome. As symptoms progress, especially if the patient meets or is close to meeting diagnostic criteria for a mood disorder, a TCA or SSRI may be helpful.

PREVENTION

- Once overtraining syndrome has developed there is no treatment other than prolonged rest, making prevention the best strategy. Coaches, athletes, physicians, and trainers should monitor athletes closely for early signs of overtraining. **Mood and sleep disturbances and resting heart rate elevation may be the earliest signs.** Athletes at high risk for developing overtraining syndrome may be monitored with daily training logs, frequent screening test (POMS) administration, and first A.M. basal heart rate monitoring. The normal resting heart rate of an athlete should be determined when he or she is healthy. Consistent elevation of more than 10 beats per minute above baseline is concerning for overtraining syndrome. Periodic assessment of athletic performance, such as time trials in a runner or swimmer, may also be performed to screen for the decrease in performance associated with overtraining syndrome.
- Care should be taken to maintain adequate sleep, nutrition, and hydration and to minimize outside stressors. Training regimens should be individualized and flexible, with periodization of training, and adequate recovery time. A decrease in training volume at the first sign of overtraining may facilitate a quicker recovery. Counsel athletes to avoid the tendency to *panic train*, or train harder after a decline in their performance. Care must be taken not to allow overtraining syndrome to develop and progress, because the prolonged recovery may interfere significantly in an athlete's preparation and participation.

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SECTION

VI

General Medical Problems in Athletes

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Infections in Athletes

24

Lisa R. Callahan and Danica N. Giugliano

INTRODUCTION

- With millions of people in the United States participating in athletics recreationally or at the high school, college, or professional levels, there is growing interest in infectious disease outbreaks in competitive sports.
- These diseases can easily spread among athletic teams and can result in decrease in performance, morbidity, and mortality.
- Infectious diseases can also pose a public health concern, because of the possibility of their spreading among athletes to coaches, support staff, spectators, and social contacts.
- In contrast to the general public's impression that athletes are "the picture of health" and rarely "get sick," athletes are actually faced with a variety of physiologic, psychological, and environmental stressors, which, in combination with an inadequate diet and an insufficient amount of sleep, can cause immunodepression and increased susceptibility to infections.
- Many athletes are amore apt to be risk-takers, which may increase risk of contracting certain infectious diseases, including sexually transmitted ones.
- Athletes may also find it difficult to take sufficient time off from training to recover from an infectious disease, which increases the challenge of eradicating these diseases in the athletic setting.

EPIDEMIOLOGY

- The first published report of an infectious disease outbreak in competitive sports was *Chlamydia trachomatis* in professional wrestlers, in 1922.
- From 1922 to 2005, there were 59 reported cases of non–food-borne infectious disease outbreaks, which most commonly involved:
  - The skin
  - Sports with direct contact between players, such as wrestling, rugby, and football
  - Herpes simplex virus and the bacterium *Staphylococcus aureus*

EXERCISE IMMUNOLOGY

J-Shaped Curve

- It was first suggested by Nieman in 2000 that a graph of an athlete's susceptibility to infection would appear as a J-shaped curve related to their intensity of training.
- When compared to a sedentary lifestyle, moderate exercise is associated with improved immune function and decreased upper respiratory tract infection (URTI) risk.
  - Studies indicate a 20% to 30% decrease in URTI incidence in adults participating in occupational and leisure time activities.
- By contrast, endurance training (high exercise workload), particularly with inadequate recovery, decreases immune function and increases URTI risk.
  - Studies indicate that marathon runners are two to six times more likely than nonrunners to have a URTI.
  - Faster marathon runners and those who trained for longer distances per week experience more URTI symptoms.
- Salivary immunoglobulin A (s-IgA) increases immune function by inhibiting the colonization of pathogens, binding antigens for transport across the epithelial barrier, and neutralizing viruses.
  - Moderate training of relatively short duration increases s-IgA levels and decreases infection risk.
  - Chronic intense exercise (>6 months) has been shown to decrease s-IgA levels and increase infection risk.

Negative Effects of Intense Exercise on Immune System

- Immune cell function
  - Numbers and effectiveness of white blood cells are decreased
  - Increased production of reactive oxygen species ("free radicals")
- Impact on stress hormone production/circulation
  - Adrenaline, cortisol, growth hormone, and prolactin all increase.
  - Increases in these hormones depress immune cell function.
- Glutamine function
  - Glutamine assists in control of the rates of T and B lymphocyte proliferation and antibody synthesis.
  - Athletes with URTIs or those performing heavy, frequent repetitive exercise have low glutamine levels.
  - Oral supplementation has been shown to increase plasma glutamine levels and improve immune function.
    - A study by Castell found that marathon and ultramarathon runners who took oral glutamine supplementation 2 hours after a race decreased their rate of infection.
    - A study by Moriguchi found that glutamine supplementation in rats after exercise maintained blood lymphocyte proliferation.

Cumulative Effects

- There is a temporary depressive effect on immune cell functions after repetitive bouts of intense exercise.
- If the recovery time between consecutive bouts is insufficient, chronic immunosuppression can occur.
- Immunosuppression is commonly seen in overtrained athletes.

COMMON INFECTIONS: RESPIRATORY INFECTIONS

Upper Respiratory Tract Infection (URTIs)

Etiology: Viral (rhinoviruses, coronaviruses, influenza A and B viruses, parainfluenza viruses, adenoviruses, enteroviruses).
Epidemiology: Average one to six URTIs per year.
Symptoms: Rhinorrhea, fatigue, sore throat, headache, cough, and congestion.
Treatment: Symptomatic.
Return to play: Base decision on "neck check": If symptoms exist above the neck (e.g., runny nose, nasal congestion, sore throat), recommend a trial of exercise at half intensity for 10 minutes and continue as tolerated if symptoms do not worsen. If below the neck symptoms exist (e.g., fever, malaise, severe cough, gastrointestinal symptoms), there should be no exercise. Exercising with fever may increase injury risk; it also impairs concentric muscle strength, mental cognition, and pulmonary perfusion; and increases fluid loss. It may also be associated with more serious illness including myocarditis.
Prevention: Hand hygiene: mucoid secretions on the hands is common means of viral transmission; viruses that cause URTIs are viable on human skin for at least 2 hours.

Infectious Mononucleosis

Etiology: Epstein-Barr virus.
Epidemiology: Most common between the ages of 15 and 24.

Symptoms: Sore throat, fever, lethargy, malaise, lymphadenopathy, and splenomegaly (Fig. 24-1).
Potential complications: Splenic rupture, airway obstruction, Guillain-Barré syndrome, meningitis, DIC, aplastic anemia, hemolytic-uremic syndrome.
Treatment: Symptomatic.
Return to play: It may take several months for a highly trained athlete who has infectious mononucleosis to return to a high fitness level. Exercise during the first 21 days of the illness is not recommended because of splenic rupture risk (1 to 2 cases per 1000; almost all cases reported in males). Recommendations vary after 21 days; if asymptomatic, normal exam and lab test results, trial of exercise at 50% intensity generally is acceptable. There is controversy surrounding ultrasound use to assess spleen size (because of the variation of "normal" spleen size), but ultrasound probably does not accurately predict risk of rupture.

Influenza

Etiology: Influenza A and B virus.
Epidemiology: CDC reports annual infection rate of 5% to 20% in the U.S. population. There is a 2-day incubation period; infected person is contagious 1 day before symptoms develop and for several days after becoming sick.
Symptoms: High fever, headache, fatigue, myalgias, dry cough, sore throat, rhinorrhea, congestion.
Potential complications: Bronchitis, pneumonia; encephalopathy, transverse myelitis, myocarditis, pericarditis can also occur, but are rare.
Treatment: Symptomatic, antiviral medication: It should be begun within 48 hours, and taken for 3 to 5 consecutive days. Amantadine, rimantadine, zanamivir, and oseltamivir are currently available, but amantadine and rimantadine are not recommended to treat influenza A because of resistance. If outbreak is in a closed setting, zanamivir and oseltamivir should be used.
Return to play: Follow URTI guidelines.
Prevention: Vaccination: in October or November most preferred; prevents influenza in 70% to 90% of healthy persons younger than age 65 and decreases morbidity of affected individuals. A nasal-spray flu vaccine is available for people ages 5 to 49 years who are not pregnant.

COMMON INFECTIONS: SKIN AND SOFT TISSUE INFECTIONS

Herpes Simplex Virus (HSV) Infection

Etiology: HSV; on the skin, often called "herpes gladiatorum" in wrestlers and "scrumpox" in rugby players; on the lips, called herpes labialis or "cold sores."
Epidemiology: Most prevalent in sports such as rugby and wrestling because of the frequency of direct contact between players. Most common sites of infection include the head, face, and neck; most common mode of transmission is via direct contact with open wounds and abrasions. Uncommon in the lower extremities.
Symptoms: Mild flulike symptoms, then development of painful, papular rashes or vesicles; fatigue, weight loss, pharyngitis, and lymphadenopathy also reported.
Treatment: Antiviral medications (acyclovir, valacyclovir, famciclovir); coverage of lesions.
Return to play: After lesions resolve.
Prevention: Daily suppressive therapy if recurrent. Thorough cleaning (HSV can survive for several hours on inanimate objects) of shared equipment; use equipment that reduces transmission of the infection, such as nonabrasive shirts, whenever possible. Despite the frequent contact wrestlers have with wrestling mats, mats have not been shown to be a mode of transmission. Screen athletes for active lesions. The United Kingdom has used a vaccine to control an outbreak of HSV in rugby players but vaccination is currently not available in the United States.

Verrucae (Warts)

Etiology: Papillomaviruses.
Epidemiology: Transmitted via direct contact, shared showers, or locker room floors; sports such as pole-vaulting, gymnastics, football, and distance running, which can cause calluses to form on the skin, can also increase transmission between athletes.

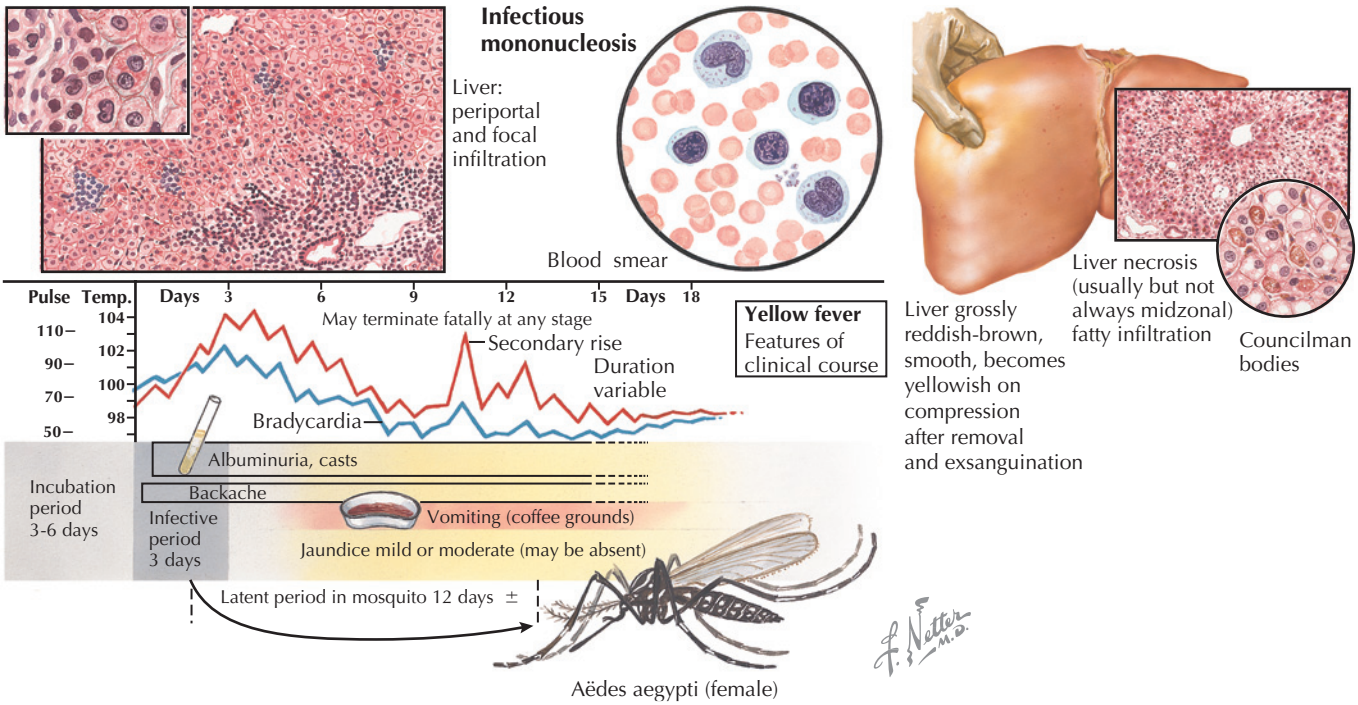


Figure 24-1 Infectious Mononucleosis.

Symptoms: Firm, rough papules or nodules with punctuate black dots within the lesion.
Treatment: Salicylic acid, freezing with liquid nitrogen, or trichloroacetic acid (painless treatment of plantar warts).

Tinea Infections

Etiology: Various fungi.
Epidemiology: Sweat-soaked clothing, infrequently washed/sanitized clothing, occlusive clothing, and local skin trauma associated with form-fitting equipment create common conditions in sports that put athletes at risk for tinea infections (Fig. 24-2).
Specific fungal infections: Tinea pedis ("athlete's foot"), tinea cruris ("jock itch"), tinea corporis ("ringworm").
Treatment: Prolonged applications of topical antifungal medications (e.g., clotrimazole, miconazole, ketoconazole, terbinafine, tolnaftate) until 2 weeks after the disappearance of physical signs. Changing the environmental conditions that facilitate the spread of the infection is necessary. In widespread or resistant cases of tinea infection, oral antifungal treatment such as voriconazole, micafungin, caspofungin can be used.

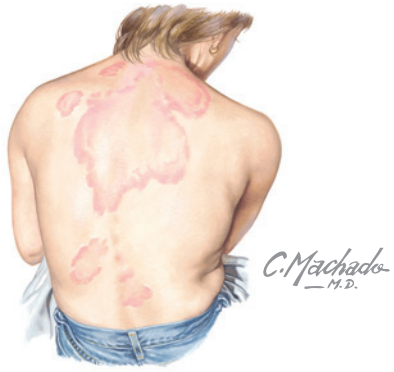
Community-Acquired Methicillin-Resistant *Staphylococcus aureus* (CA-MRSA)

Epidemiology: Originally a hospital-acquired infection, MRSA began to emerge in the wider population during the 1990s. Strains often affect previously healthy people and have been reported in athletic settings. Athletes involved in frequent, repetitive contact are most susceptible. CA-MRSA has been reported in several sports, including college and professional football and basketball, wrestling, rugby, and fencing. The most common mode of MRSA transmission is through person-to-person contact through open skin wounds, but transmission through environmental sources (e.g., towels, shared equipment, and whirlpools) has also been described.
Symptoms: Presentation ranges from localized cellulitis to a soft tissue abscess. The infected area is sometimes described as a skin lesion and is often mistaken by patients and clinicians as a spider bite.
Treatment: Oral antibiotics, including clindamycin, trimethoprim-sulfamethoxazole (TMP/SMX), rifampin (used in combination with other agents), and linezolid. Beta-lactams, fluoroquinolones, and macrolides/azalides are not optimal choices because of their relatively high prevalence of resistance. If an abscess is present, an incision and drainage of the abscess *must* be performed. Intravenous antimicrobial agents can be administered for patients with severe staphylococcal infections or for patients who do not respond to oral antibiotic treatment; vancomycin is first-line therapy intravenously, but clindamycin, daptomycin, linezolid, quinupristin-dalfopristin, tigecycline, and TMP/SMX have also been used
Prevention: Screen athletes regularly and teach them to report all skin injuries. When injuries to the skin occur, they should be kept clean and completely covered with dressings until the lesions heal. Maintaining good hygiene is essential: the Centers for Disease Control and Prevention (CDC) has reported that the main transmission mode of MRSA is via hands that are contaminated; therefore, hand washing or using alcohol-based hand rubs should be emphasized by all athletes and staff. Athletes should also avoid sharing equipment, towels, clothes, and other personal items including razors. Routine showering immediately after activity, cleaning of facilities and showers, and using antibacterial soap and clean towels can help. New MRSA hygiene products (e.g., Sani Sport, SportsAide, TurfAide) have been developed to combat MRSA; they are expensive and their relative efficacy has not been established. Culture all suspicious skin lesions.

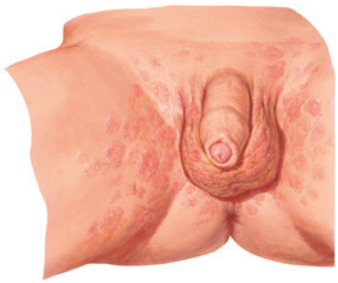
COMMON INFECTIONS: SEXUALLY TRANSMITTED DISEASES

Chlamydia

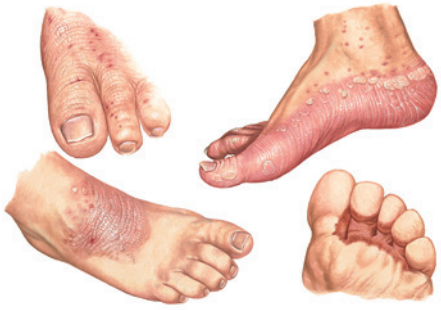
Etiology: The bacterium *Chlamydia trachomatis.*
Epidemiology: The CDC estimates that most cases are not reported. In 2005, there were 332.5 cases of chlamydia per 100,000 population, an increase of 5.1% since 2004. It is three times more common in young women than in young men; seven times more common in African-American women than in white women. The highest rates of chlamydia are reported in the 15- to 24-year-old age group.
Symptoms: Early infection usually asymptomatic; may have urogynecologic symptoms such as discharge, urinary frequency, or dysuria.
Treatment: Very responsive to antibiotics (a single dose of azithromycin or a week of doxycycline, twice daily).
Potential complications: Reproductive health problems. Forty percent of untreated women develop pelvic inflammatory disease and subsequent fertility issues. Rarely, untreated chlamydia can also cause sterility in men. Chlamydia increases the risk of contracting HIV in both males and females.
Screening/Prevention: All sexually active women younger than age 25, all sexually active women over age 25 who have new sexual partners or a history of multiple partners, and women who are pregnant should be tested. Retesting should occur 3 months after initial treatment. There are no recommendations



Tinea corporis ("ringworm") - causes a ring-shaped rash, usually on the legs or trunk of the body.



Tinea cruris ("jock itch") - affects the inner thighs and groin. It causes pain and severe itching and usually a rash of red, ring-like patches that grow outward in the crease of the thighs. The patches usually have bumps and a different color than nearby skin.



Tinea pedis ("athlete's foot") - causes severe itching and a rash on bottoms of the feet and between the toes. Itching is often worst between the toes.

Figure 24-2 Tinea Infections.

for screening in males; however, it is prudent for team physicians to discuss screening with male athletes at risk. Encourage condom use.

Gonorrhea

Etiology: The bacterium *Neisseria gonorrhoeae*.

Epidemiology: There were 115.6 reported cases of gonorrhea per 100,000 population in 2005. It is most often reported in African Americans; infection rate is 18 times higher in blacks than in whites. The CDC estimates that only one third of cases are actually reported.

Symptoms: As with chlamydia, early infection is often asymptomatic. Men may experience dysuria, penile discharge, and/or swollen, painful testicles. Women are usually asymptomatic but can have dysuria and/or vaginal discharge.

Treatment: Many effective antibiotics; first choice should be a single dose of ceftriaxone or spectinomycin. Fluoroquinolones are not recommended because of a recent increase of fluoroquinolones-resistant strains of *N. gonorrhoeae*.

Potential complications: Pelvic inflammatory disease, systemic disease including gonococcal arthritis, infertility, also at a greater risk for contracting HIV.

Screening/Prevention: No recommendations by the CDC for gonorrhea screening. Team physicians should discuss testing with athletes and encourage condom use.

Human Papillomavirus (HPV)

Epidemiology: According to the CDC, more than half of the sexually active men and women in the United States are infected with human papillomavirus (HPV) at some point in their lives. Currently approximately 20 million Americans between the ages of 15 and 49 are infected, half of whom are between 15 and 24 years old. Women who are under the age of 25, have an increased number of sexual partners, had first sexual intercourse before the age of 17, or have a male partner who has or has had multiple sex partners are at increased risk.

Symptoms: Often asymptomatic, may be associated with genital warts, Pap test abnormalities, or, rarely, cervical cancer.

Diagnosis: HPV can be diagnosed in women using a Pap test or a test that detects HPV DNA. No diagnostic HPV tests are currently available for men.

Prevention: A recently developed vaccine, Gardasil, has a three-dose schedule and is recommended for females 9 to 26 years old who are not pregnant; it does not protect against all types of HPV strains that cause cervical cancer.

COMMON INFECTIONS: BLOOD-BORNE PATHOGENS

HIV/AIDS

Epidemiology: Currently more than 1 million Americans are infected with the human immunodeficiency virus (HIV), 25% of whom do not know they are infected. Of the approximately 37,000 Americans diagnosed with the virus each year, approximately 75% are male and 50% are black. The infection rate is almost three times higher in males than in females and almost nine times higher in blacks than in whites. About 80% of HIV cases are contracted through sexual contact. Those infected with STDs such as chlamydia and gonorrhea are at a three- to five-times higher risk of being infected with HIV. Athletes may also be at risk of HIV infection through use of nonsterile needles or syringes during illicit drug use or steroid use; HIV transmission has been reported through the injection of anabolic steroids. According to the CDC, there is also a risk of contracting HIV through nonsterile needles used in tattooing or body piercing. Although there has been much media attention to the possibility of contracting HIV during athletic participation, no such case has actually been documented. In addition, contact with saliva, tears, or sweat has not been shown to result in transmission of HIV.

Screening in athletes: Currently there is no accepted recommendation for screening of athletes for HIV.

Athletic participation for athletes who have HIV infection: There is no evidence that supports that exercise and training of moderate intensity is harmful to an HIV-infected athlete. Exercise may be beneficial to an HIV-infected athlete by reducing anxiety and depression. An HIV-infected athlete should be allowed to participate in sports if he or she is asymptomatic and without evidence of deficiencies in immunologic function.

Hepatitis B Virus (HBV)

Epidemiology: Approximately 60,000 people in the United States acquire the hepatitis B virus (HBV) each year. One in twenty people will be infected sometime in their life. The highest rate of infection occurs in 20- to 49-year-olds. The only well-documented case of HBV transmission in sports participation occurred in Japan among sumo wrestlers. Though HBV infection risk in the sports setting is small, it is thought to be 50 to 100 greater than HIV risk because HBV is more highly concentrated in the blood than HIV. Unlike HIV, HBV can survive outside the body for at least 7 days and is resistant to drying, ambient temperatures, simple detergents, and alcohol; thus, HBV can be transmitted through inanimate objects, such as environmental surfaces or equipment. Of those with chronic HBV, 15% to 25% will die from chronic liver disease.

Symptoms: Jaundice, tiredness, loss of appetite, nausea, abdominal discomfort, dark urine, clay-colored bowel movements, joint pain; late symptoms (ascites, varices, peripheral edema, encephalopathy) are secondary to portal hypertension and hepatic decompensation. There are acute, chronic, and chronic active phases.

Treatment: Antiviral medications.

Prevention: Vaccination is 95% effective; athletes are not required to be immunized in order to participate in sports, but are encouraged to get the vaccine.

Athletic participation for athletes who have HBV infection: Participation should be determined according to signs or symptoms such as fever, fatigue, or hepatomegaly. There is no evidence that intense, competitive training is harmful to an asymptomatic HBV carrier (acute or chronic) without evidence of organ impairment.

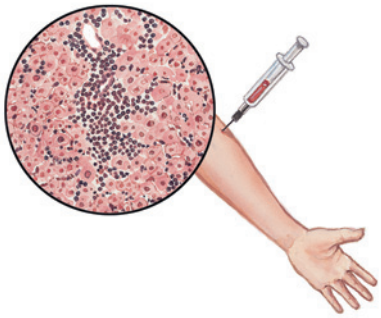
Hepatitis C Virus (HCV)

Epidemiology: One of the most common blood-borne infections in the United States. Approximately 3.5 million Americans are chronically infected with the hepatitis C virus (HCV), almost twice as many as those infected with chronic HBV; 70% of HCV-infected individuals will develop chronic infection. The most common mode of transmission of HCV is injection drug use. There have been no reported cases of HCV transmission during sports activities, but there has been a report of transmission through sharing a bloody cloth during a fistfight, suggesting that in sports where bleeding may occur, there is the possibility of HCV transmission. Athletes contracting HCV through shared needles (injecting anabolic steroids, intravenous vitamin complexes), and tattooing and body piercing have been reported. HCV is the leading cause of chronic liver disease (Fig. 24-3).

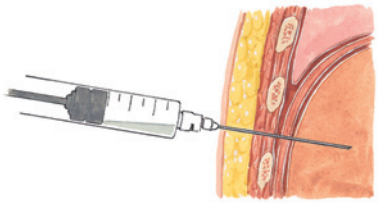
Symptoms: Usually asymptomatic early; symptoms similar to HBV infection late in disease.

Treatment: Antiviral medications are more effective than with hepatitis B; HCV is *possibly* curable with *aggressive medical therapy*.

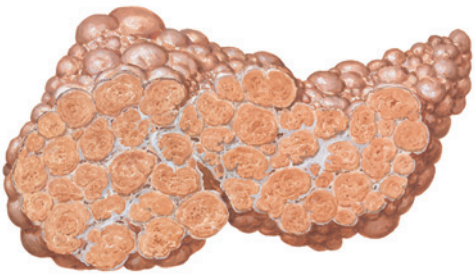
Athletic participation for athletes who have HBV infection: There are no current CDC recommendations for athletic participation for athletes who have HCV infection.



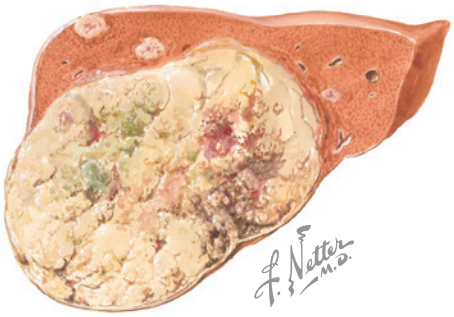
Most patients with HCV have abnormal liver (ALT, AST) findings.



Liver biopsy plays an important role in diagnosis and prognosis.



Twenty percent of patients develop cirrhosis by 20 years of age.



The risk for hepatocellular carcinoma is estimated to be 1% to 5% per year after 20 years of disease or 1% to 4% per year in patients with cirrhosis.

Figure 24-3 Clinical Pictures of Hepatitis C Infection.

COMMON INFECTIONS: MYOCARDITIS

Myocarditis

Etiology: Usually viral: enteroviruses, particularly the Coxsackie B viruses, respiratory viruses, such as influenza A and B viruses and cytomegalovirus, herpes simplex viruses, human herpes virus 6, parvovirus B19, Epstein-Barr viruses, and hepatitis C virus. HIV-induced myocarditis has also been reported. Possible bacterial causes include *Mycoplasma pneumoniae* and *Chlamydia pneumoniae*. Noninfectious etiologies, such as cocaine-induced, have also been reported.

Epidemiology: More common in males than females (males constitute 62% of reported cases). More than half of myocarditis cases occur in the 20- to 39-year-old age range.

Pathology: Myocardial infections result in tissue necrosis, edema, and scar tissue formation (Fig. 24-4). This can lead to a variety of arrhythmias, and can be a cause of sudden cardiac death in athletes. Myocarditis can also increase the risk of heart failure, cardiomyopathy, and pericarditis.

Symptoms: Most commonly, chest pain or shortness of breath with exertion. May present with no cardiac symptoms or with fatigue, fluid retention, fever, palpitations, or syncope.

Diagnosis: Myocarditis can be difficult to diagnose. Diagnostic tools include electrocardiography, serum markers, cardiac magnetic resonance imaging (MRI), and endomyocardial biopsy, with the latter being the gold standard.

Treatment: Stop all exercise. Angiotensin-converting enzyme (ACE) inhibitors, diuretics, and antiarrhythmics are available to treat myocarditis. Immunosuppressive agents remain controversial, but may be considered if clinical deterioration occurs. For acute myocarditis, nonsteroidal anti-inflammatory drugs, intravenous immunoglobulin therapy, methylprednisolone, and azathioprine can be used.

Return to play: Recovery can take several months. Risk for lethal arrhythmias until recovery is complete. Consider evaluating cardiac status at 3 and 6 months (radionuclide angiography and stress echocardiography). Restrict from training and competition for 6 months (current Bethesda Conference recommendations). After 6 months, if an athlete's cardiac status returns to normal, training is allowed, but must be gradual. Consider ambulatory cardiac monitoring to ensure the absence of arrhythmia before an athlete is allowed full sports participation.

INFECTIOUS DISEASE AND INTERNATIONAL PLAY

Special Considerations for International Play

- Athletes should seek medical advice regarding current vaccination and chemoprophylaxis 4 to 6 weeks prior to international travel for competition.
- Current vaccinations recommended by the CDC for travel in different regions of the world can be found on the CDC website (http://cdc.gov/travel) or in the "Yellow Book," which is published every two years by the CDC as a reference for health care providers.

Special Precautions: Blood-Borne Pathogens

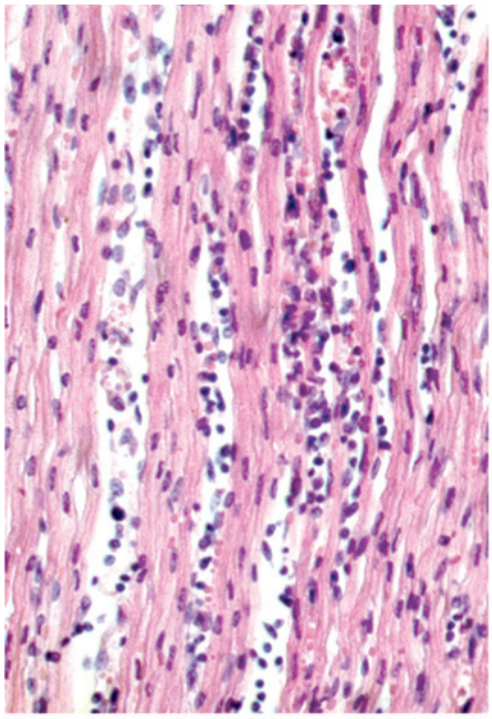
- Athletes should be counseled regarding the higher prevalence of blood-borne pathogens worldwide, and preventive measures should be taken to avoid exposure while traveling.
- Internet references may be useful, including the CDC website mentioned earlier and the World Health Organization (WHO) website (www.who.int/ith/en).

Special Precautions: Tuberculosis

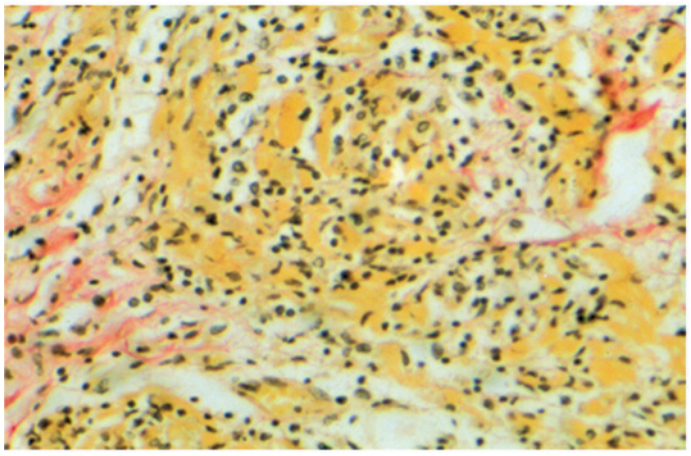
Epidemiology: In 1993 the WHO declared tuberculosis (TB) a global emergency. One third of the world's population is currently infected with the TB bacillus (Fig. 24-5).

Treatment: Several recommended TB treatment regimens address drug-susceptible organisms. Options include an initial 2-month phase of isoniazid, rifampin, pyrazinamide, and ethambutol. The initial phase is followed by a continual phase for either 4 or 7 months using isoniazid and rifampin.

Prevention/Screening: Athletes who are traveling to areas where prolonged exposure to TB is possible should be particularly careful to avoid prolonged close contact in crowded, enclosed



Coxsackie group B virus infection. Diffuse and patchy interstitial edema; cellular infiltration with only moderate muscle fiber destruction (x100)



Diffuse cellular infiltration of bundle of His and right and left bundle branches (x100)

Figure 24-4 Viral Myocarditis.

environments. They should have a follow-up skin test 8 to 10 weeks after returning to the United States. The American College of Health Association recommends screening for college students who have signs or symptoms of active TB, are HIV-positive, inject drugs, have had close contact with another person with TB, have emigrated in the last 5 years from countries where TB is endemic, or have spent significant time in congregate high-risk settings.

SUMMARY

Infectious diseases are common in sports, can affect individual athletes, can be spread to other athletes, and can affect performance and the short-term and long-term health of athletes. Prevention of many of these diseases is possible. Athletes and sports medicine physicians must be properly educated on preventative measures such as accurate immunizations, good hygiene practices, and appropriate screening in order to minimize the risk of infections in athletes.

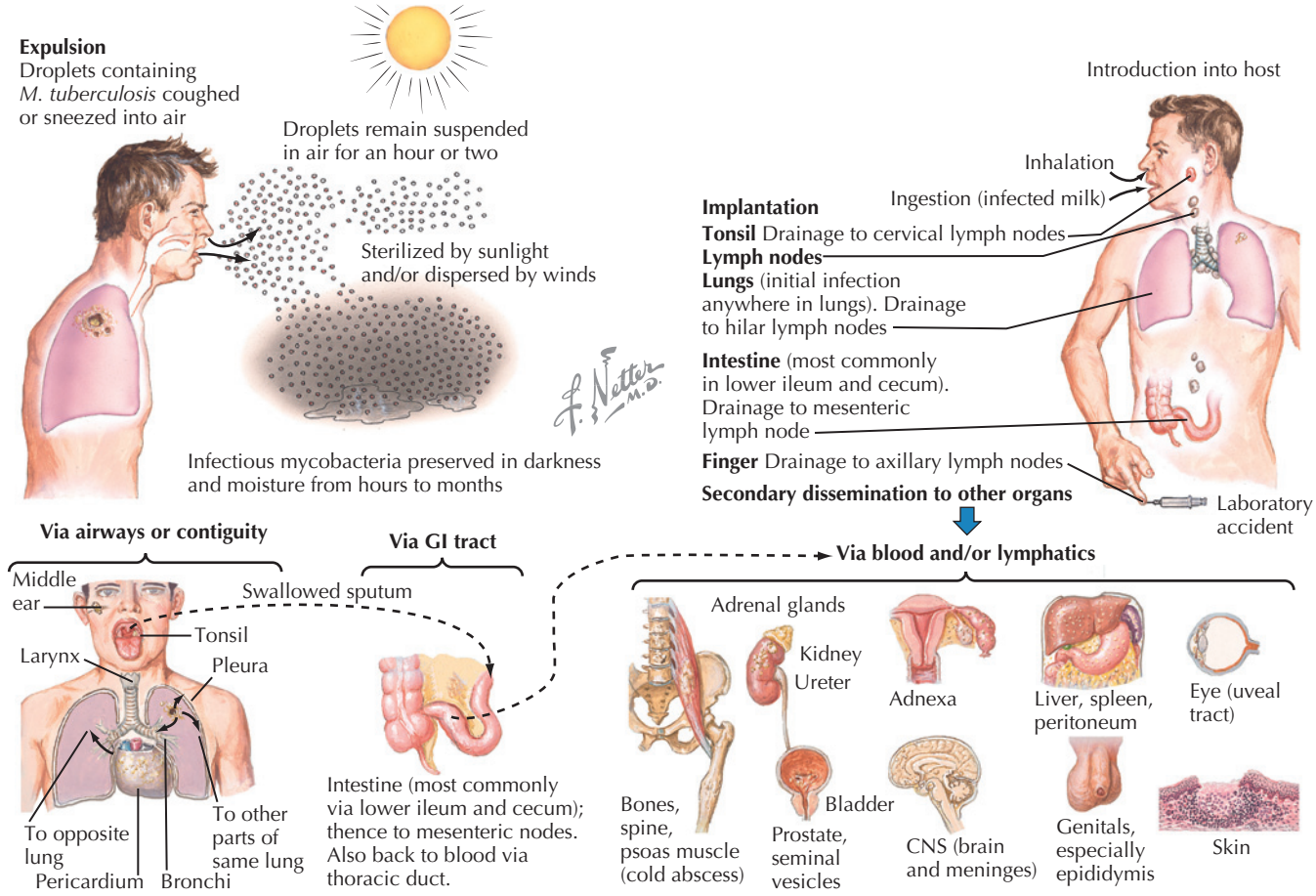


Figure 24-5 Dissemination of Tuberculosis.

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Gastrointestinal Problems

25

Balakrishnan Natarajan

INTRODUCTION

Competitive athletes frequently experience gastrointestinal (GI) problems, many of which are related to training or competition. Surveys of serious runners have shown that up to 80% have experienced GI symptoms, mostly in the lower tract, before, during, or after competition. The problems are more common, and perhaps more severe, at higher levels of training and competition.

PROBLEMS NOT UNIQUE TO ATHLETES

Anxiety and Stress Reaction

- Performance anxiety.
- Inhibitory effect on upper GI function activity: decreased acid secretion in stomach, slowed motor activity, reduced blood flow.
- Continued anxiety may result in acid hypersecretion.
- Stimulant effect on lower GI activity: increased motility, decreased transit time.
- **Symptoms:** dry mouth, dyspepsia ("knot in stomach"), heartburn, reflux, abdominal cramping, diarrhea.
- **Treatment:** reassurance and education, behavior modification, relaxation exercises.

Acute Gastroenteritis

- Incidence second only to upper respiratory infections in adolescents and young adults.
- Etiologic agents: viral (most common, including rotavirus, Norwalk agent), bacterial, protozoan *(Giardia lamblia)*.
- Peak incidence: winter in cities, summer in rural or outdoor sports.
- **Symptoms:** nausea, vomiting, abdominal cramps, diarrhea, fever, myalgia.
- **Treatment:**
  - Usually self-limited (2 to 3 days).
  - Clear fluids, electrolyte-containing fluids (e.g., sport drinks) are cornerstone; replace fluid loss liter for liter.
  - Assess degree of dehydration (body weight, urine output, blood pressure) before strenuous practice or game.
  - Antimotility drugs may be effective for abdominal cramps but also may prolong carrier state of some organisms: loperamide (Imodium), diphenoxylate hydrochloride with atropine (Lomotil).
  - **"Traveler's diarrhea"** may respond to trimethoprim/sulfamethoxazole, double-strength, twice daily; Pepto-Bismol, 1 ounce every hour, until symptoms abate or 8 ounces have been consumed; or ciprofloxacin, 500 mg twice daily.
- Return to competition limited only by hydration status, infective nature of problems, symptom complex (i.e., frequent diarrhea), and reconditioning.

INITIAL APPROACH TO GI PROBLEMS IN ATHLETES

Differential Diagnosis

- Infectious: gastroenteritis, hepatitis.
- Neoplastic: GI tract cancer, lymphoma.
- Endocrine: hyperthyroidism, hypothyroidism, pancreatic disease.
- Autoimmune: Crohn's disease, ulcerative colitis, celiac disease.
- Trauma: GI organs, genitourinary organs.
- Vascular: cardiac ischemia, mesenteric ischemia.
- Other: peptic ulcer disease, irritable bowel syndrome, constipation, medication- or supplement-induced disorder, problems related to food or beverage intake.

History

- Differentiate upper GI from lower GI symptoms: nausea, cramping, bloating, diarrhea.
- Determine severity of disease: hematochezia, melena.
- Consider effects of foods and beverages, anxiety/stress, caffeine, tobacco, alcohol, nonsteroidal antiinflammatory drugs (NSAIDs) or other medications, drugs of abuse or other supplements.
- Assess symptoms of systemic disease.

Physical Exam

- Signs of volume depletion: orthostatic hypotension, tachycardia.
- Signs of inflammatory bowel disease: oral ulcers, dermatologic, ocular, joint manifestations (Fig. 25-1).
- Signs of thyroid disease: thyromegaly, altered reflexes, dermatologic manifestations, ocular manifestations.
- Signs of systemic wasting: temporal wasting, lymphadenopathy, hepatomegaly, splenomegaly.

Laboratory Data

Determine necessity of tests based on severity of symptoms. Tests may include cell blood count, iron studies, hepatic function panel, *Helicobacter pylori* testing, electrolytes, thyroid studies, and **occult blood in stool** and other stool studies.

HYPOPERFUSION OF GI TRACT DURING INTENSE EXERCISE

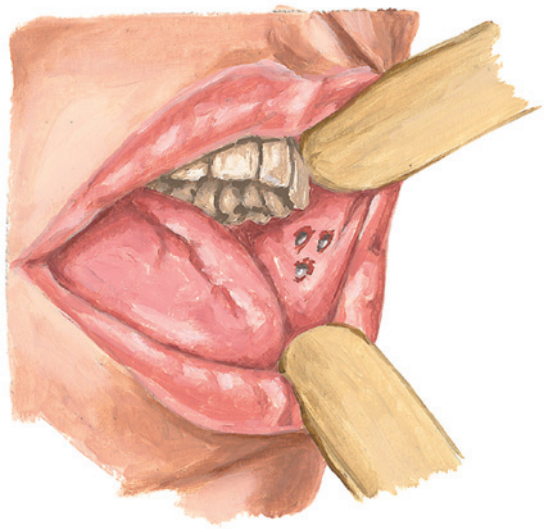
Physiologic arterial shunting during exercise: During the first few minutes of exercise, 15% of central blood volume is shunted to working muscles. As core temperature rises during exercise, 20% of central blood volume is shunted to the skin for cooling. Central blood volume is maintained by redirecting blood away from other organs, especially the splanchnic bed. Studies have revealed that splanchnic blood flow declines from 1.56 L per minute at rest to 0.3 L per minute at maximal exercise.

Possible etiology of exercise-induced shunting: Decreased esophageal motility, erosive hemorrhagic gastritis, delayed gastric emptying, diarrhea, intestinal bleeding.

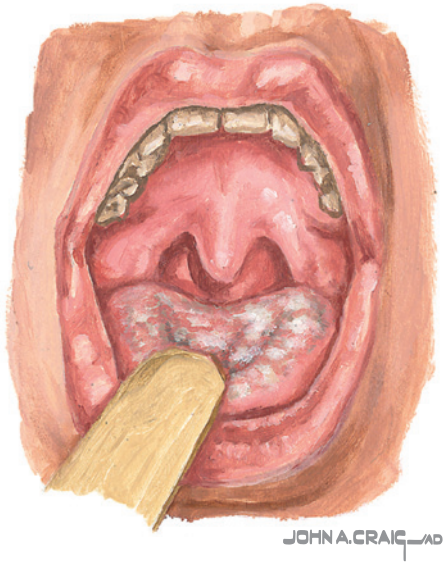
Factors exacerbating hypoperfusion of GI tract: Dehydration, high ambient temperature, lack of acclimation to exercise in heat.

UPPER GI PROBLEMS

- **Pain syndromes:** Upper GI pain related to training and competition often presents a diagnostic dilemma. "Heartburn" may be even more difficult to diagnose in athletes because physical demands of sport add to the list of potential causes.
  - Gastroesophageal reflux (GER)
    - Vigorous exercise causes GER in normal subjects. Effect is notable in runners, bicyclists, and weightlifters.
    - Frequency, amplitude, and duration of esophageal contractions decline with increasing exercise intensity.
    - Exertional symptoms include belching and chest pain.
    - Hypoperfusion resulting from physiologic arterial shunting to muscles and skin may cause reduced esophageal motility.



Aphthous ulcers (occur on buccal mucosa, tongue, and palate)



Oral candidiasis (secondary to chronic illness and use of antibiotics)

Figure 25-1 Oral Ulcers.

- There may be some protective effect from increases in lower esophageal sphincter tone that have been demonstrated with moderate exercise.
- Treatment: H_2-blockers or proton pump inhibitors 4 hours before exercise, standard medical management for reflux, alteration of oral intake (avoid symptom-triggering foods and beverages, no food for 3 hours prior to exercise) (Fig. 25-2).

- **Gastritis:** Erosive gastritis may be induced by exercise-related hypoperfusion, NSAIDs, or anxiety; often hemorrhagic. Treat with proton pump inhibitors, H_2-blockers, antacids (Fig. 25-3).
- **Peptic ulcer disease** is no more or less common in runners than in the general population; use standard medical management (see Fig. 25-3).
- **Delayed gastric emptying** may be related to bloating, reflux, or both; it also may be caused by hypoperfusion resulting from arterial shunting away from the splanchnic bed.
- **Dyspepsia** (upper GI pain with no identified cause) is treated empirically with H_2-blockers or proton pump inhibitors 4 hours before practice or competition.
- **Hypoferritinemia** has been associated with exercise-induced abdominal pain.
- **Angina or cardiac ischemia** must be considered in older athletes.
- **Asthma** symptoms can overlap with GER.

- **Upper GI bleeding:** May be related to hemorrhagic gastritis or peptic ulcer disease (see "Gastritis"). Mechanical cause is proposed in some cases. Shearing forces of diaphragm on gastric fundus may induce bleeding.
  - Evaluate and treat with standard upper GI methodology.
  - Improve hydration before and during performance. Increased plasma volume may not reduce ischemia.

EXERCISE-INDUCED DIARRHEA AND LOWER GI PROBLEMS

Runner's Diarrhea ("Runner's Trots")

- Stimulated by intense endurance running, with or without accompanying GI bleeding.
- **Descriptive data** from a study by Priebe of 425 runners in a 10-K race showed the following:
  - **Incidence:** 30% of runners in race.
  - **Characteristics of syndrome:** 85% passed semiformed or watery stools; 60% had low abdominal pain or rectal urgency, generally relieved by defecation; 15% had multiple stools; 13% had large-volume stools; and **12% had frank blood in stool.**

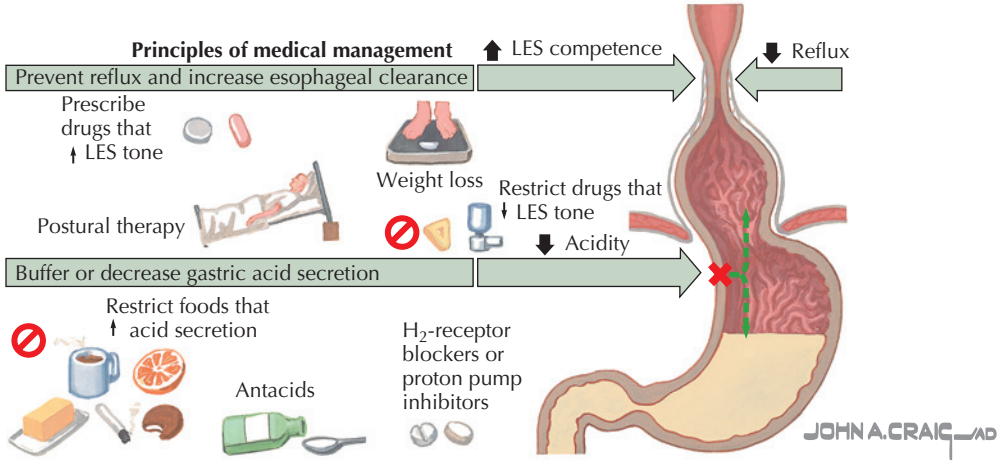


Figure 25-2 Gastroesophageal Reflux.

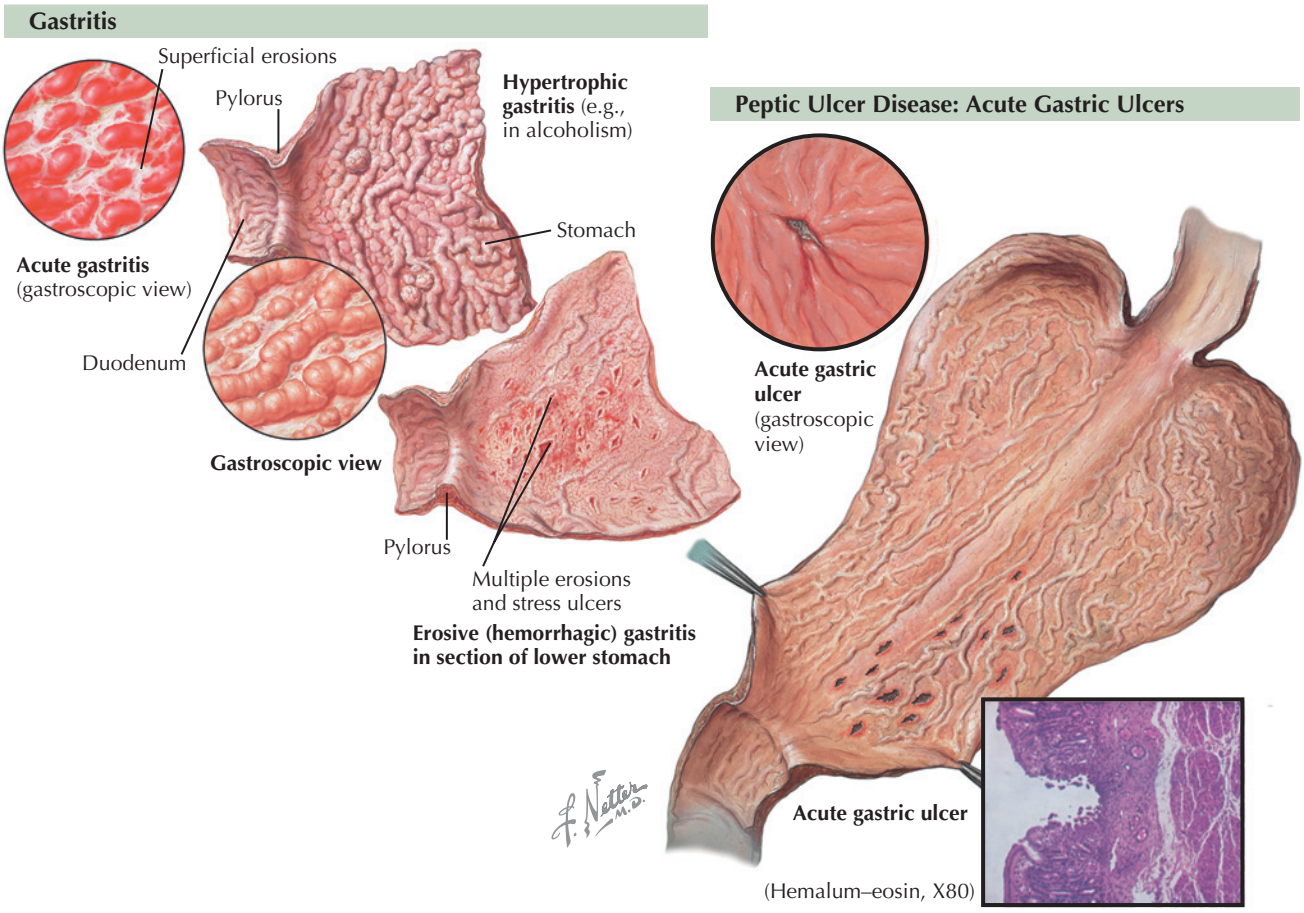


Figure 25-3 Upper GI Problems.

- **Other data:** 30% to 42% of serious runners have urge to defecate; 14% to 30% report running-induced diarrhea; direct relationship between severity of symptoms and level of physical exertion; diarrhea more common in running than in other sports.

Lower GI Bleeding

- **Grossly bloody diarrhea stimulated by intense performance:** large amounts of red, maroon, or clotted blood may be seen; also severe abdominal pain.
- Microscopic increases in fecal hemoglobin after intense running are even more common and also have been noted in endurance bicyclists; may be caused by upper or lower GI bleeding; transient in endoscopic studies.

Proposed Etiologies

Diarrhea Only

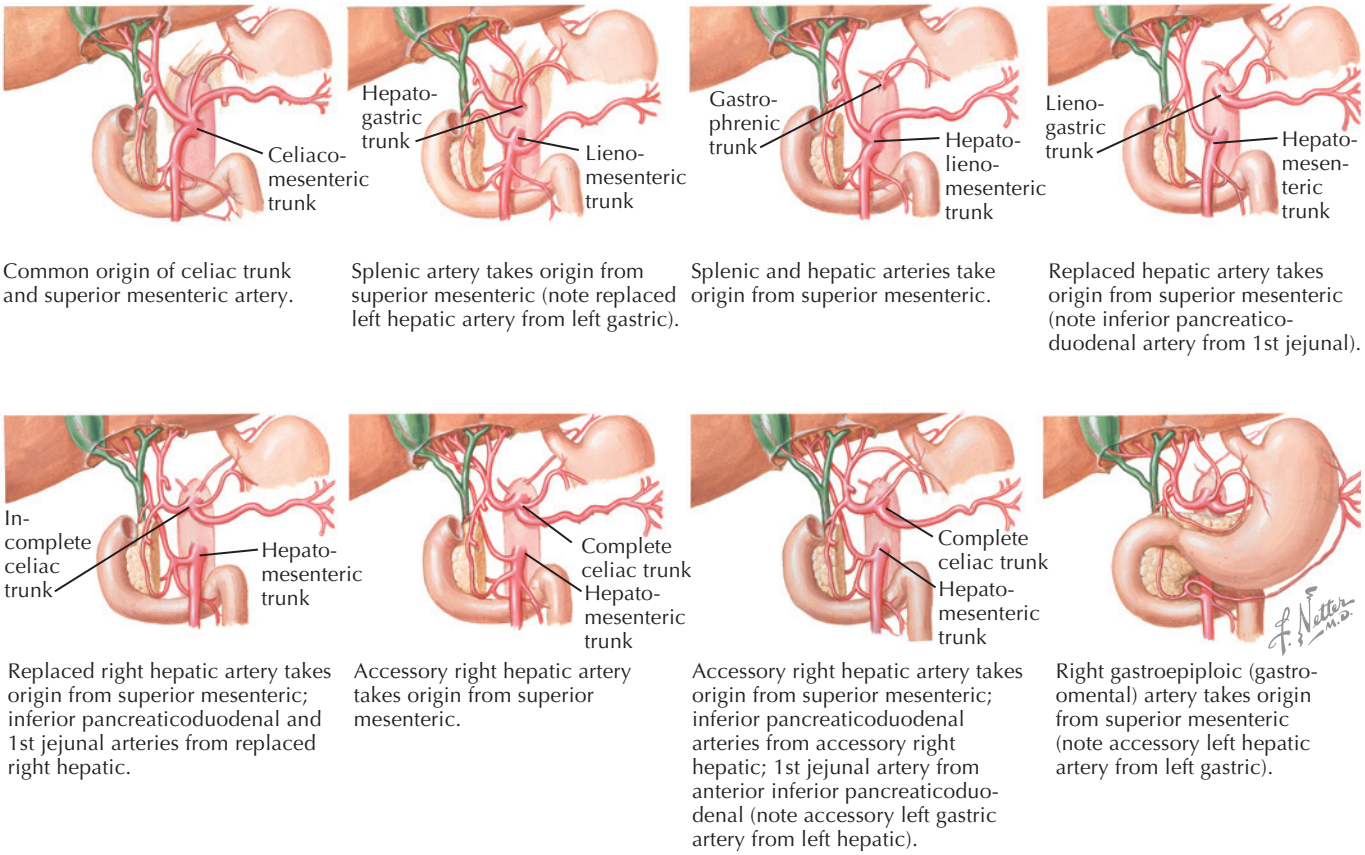
- **Anxiety-induced diarrhea** (see "Anxiety and Stress Reaction").
- **Increased GI motility:** exercise increases secretion of gastrin, motilin, other hormones affecting motility; irritable bowel syndrome.
- **Dietary factors:** high-fiber diet may cause exercise-induced diarrhea in small subset of runners; lactose intolerance has higher incidence in patients with exercise-induced diarrhea than in general population; symptoms may occur only when exercising; sorbitol or fructose intolerance with fruit intensive diets; large doses of caffeine or vitamin C.
- **Possible immune system etiology:** variant of exercise-induced anaphylaxis; generalized urticaria including urticarial lesions in intestines.
- **Endotoxins** have been proposed as a cause of gastrointestinal symptoms.

Intestinal Bleeding with or without Diarrhea

- **Intestinal ischemia:** hypoperfusion caused by shunting blood flow from mesentery to muscles and skin. Mild exercise decreases intestinal perfusion by 40%; strenuous exercise may decrease it by 80% (may be magnified by dehydration). Relative gut ischemia causes focal areas of necrosis and ulceration. Ischemia may also cause intestinal malabsorption and, thereby, diarrhea. Clinically important ischemia should generally also cause abdominal pain.
- **Cecal slap syndrome:** mechanical trauma from running reported to cause hemorrhagic cecal lesions and diarrhea.

Celiac Artery Compression Syndromes

- Celiac artery compression syndromes (median arcuate ligament syndrome): rare and controversial cause of chronic recurrent abdominal pain reported in nonathletic literature.
- External compression of celiac and sometimes superior mesenteric artery by median arcuate ligament of diaphragm (left crus or fibrous band connecting left and right crus), especially during exhalation (Fig. 25-4).
- **Diagnosis of exclusion:** all other GI pathology must be ruled out and high index of clinical suspicion maintained.



Common origin of celiac trunk and superior mesenteric artery.

Splenic artery takes origin from superior mesenteric (note replaced left hepatic artery from left gastric).

Splenic and hepatic arteries take origin from superior mesenteric.

Replaced hepatic artery takes origin from superior mesenteric (note inferior pancreatico-duodenal artery from 1st jejunal).

Replaced right hepatic artery takes origin from superior mesenteric; inferior pancreaticoduodenal and 1st jejunal arteries from replaced right hepatic.

Accessory right hepatic artery takes origin from superior mesenteric.

Accessory right hepatic artery takes origin from superior mesenteric; inferior pancreaticoduodenal arteries from accessory right hepatic; 1st jejunal artery from anterior inferior pancreaticoduodenal (note accessory left gastric artery from left hepatic).

Right gastroepiploic (gastro-omental) artery takes origin from superior mesenteric (note accessory left hepatic artery from left gastric).

Figure 25-4 Celiac Trunk.

- No clear diagnostic criteria and no imaging modality predict response to surgery.
- Most common in young people, people with recent weight loss, and women.
- Pain may be sharp, dull, steady, or crampy and may worsen after meals, weight loss, or position changes.
- **Loud systolic bruit** may be appreciated in epigastrium.
- **Four proposed mechanisms for pain:** compression of celiac and superior mesenteric artery (SMA), resulting in mesenteric ischemia; extensive collateral circulation "steals" from primary distribution of SMA in fed state; inadequate collateral circulation, resulting in nonocclusive ischemia during hypotension or decreased perfusion; irritation of celiac plexus or overstimulation of sympathetic nerve fibers.
- Risk of syndrome in athletes is theoretical. Athletes with anatomic setup for syndrome may become symptomatic with decreased splanchnic/mesenteric blood flow during exercise, dehydration, high ambient temperatures, and poor acclimatization.
- SMA syndrome also described in nonathletic literature as an atypical cause of high intestinal obstruction resulting from compression of third part of diaphragm against posterior structures by narrow-angle SMA origin.

Evaluation

- **Detailed history and physical examination:** standard procedure, with close review of symptoms and family history of GI disease; intensity, type, and duration of exercise; dietary history; travel history focusing on potential food and water pathogens.
- **Laboratory work** based on history and physical examination: stool evaluation (culture, ova and parasites, occult blood, white blood cells), enterotest for giardiasis, complete blood count, erythrocyte sedimentation rate, electrolytes.
- **Endoscopy for frank lower GI bleeding** should be performed within 1 to 2 days of bleeding; otherwise, ischemic lesions may resolve before endoscopy.
- **Arteriography** confirms clinical diagnosis of celiac artery compression syndrome. Duplex ultrasonography recently used to measure flow velocities in SMA and celiac arteries.

Prevention (of Diarrhea) and Therapy

- Encourage bowel movement. Have light meal a few hours before competition and then jog to stimulate gastrocolic reflex.
- **Improve hydration before and during performance:** increased plasma volume may decrease ischemia (with or without diarrhea).
- **Dietary manipulations:**
  - Athletes on low-fiber diet may improve by adding fiber to absorb intraluminal fluid.
  - Athletes on high-fiber diet may improve by reducing fiber to decrease stimulation of intestinal motility. Many athletes prefer high-fiber diet because it stimulates intestinal motility, thus reducing intraluminal contents.
  - Eliminate foods that trigger bowel symptoms in individual athletes—lactose, fructose, sorbitol, caffeine.
- **Antimotility medications** may be helpful in cases of diarrhea that appear to be a form of functional bowel syndrome and in those that remain undiagnosed: Imodium (risk of hyperthermia), Lomotil.
- **Decrease training and competition level 20% to 40% in both mileage and intensity, then build back up slowly.** May be able to cross-train.

- Surgery involving division of obstructing diaphragmatic fibers and denervation of celiac ganglion may benefit patients with celiac compression syndrome but is controversial.

EXERCISE AND THE LIVER

Normal liver: No significant changes in function are associated with strenuous exercise; there are asymptomatic declines in portal pressure.

Acute hepatitis: Although many practitioners advocate strict bed rest, studies have shown no significant difference in outcomes between bed rest and moderate exercise. No strenuous exercise or competition until liver function tests (LFTs) are normal.

Chronic hepatitis: There is no significant effect of exercise on serum aminotransferases; there is a significant positive effect of exercise on oxygen consumption and work capacity. Activity and competition as tolerated, with medical follow-ups.

Chronic cirrhosis: Exercise appropriate for compensated cirrhotics; no hazardous effects with vigorous walking, swimming, ergometer bicycle training.

Hepatomegaly: No contact or semicontact sports.

EXERCISE-RELATED TRANSIENT ABDOMINAL PAIN (SIDE-STITCH)

- Most common in lateral aspect of midabdomen.
- Symptoms are generally worse in postprandial state.
- Exercise-related transient abdominal pain (ETAP) is greatest in sports involving repetitive torso movement (running, swimming, equestrian sports).
- Incidence declines with age.
- Proposed mechanisms are benign and include:
  - Diaphragmatic ischemia/spasm.
  - Stress placed on peritoneal ligaments connecting diaphragm to viscera.
  - Exertional irritation of parietal peritoneum.
  - Mechanical compression of thoracic intercostal nerves.

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Hematologic Problems in Athletes

26

Jason A. Robertson and Tracy R. Ray

SPORTS/DILUTIONAL ANEMIA

Description: Known as dilutional anemia or pseudoanemia.

Epidemiology: The most common cause of anemia found in the athletic population. Dilutional pseudoanemia is not pathologic but rather an adaptation to endurance training and normalizes after training cessation. It is hypothesized that the dilutional anemia enhances the efficiency of oxygen delivery by decreasing blood viscosity and increasing cardiac output.

Etiology: The dilutional anemia occurs because the plasma expansion is greater than the red blood cell (RBC) mass increase. Typically there is no change or an actual increase in RBC mass and total RBCs. Hemoglobin (Hgb) levels average 0.5 g and 1.0 g lower in competitive and elite athletes, respectively, as compared with their sedentary counterparts. With strenuous exercise, there is an initial volume contraction caused by fluid loss from an increase in hydrostatic pressure and an increase in insensible water losses. The volume contraction can cause a decrease in the total volume of 6% to 20%. Within 3 to 5 hours postexercise, plasma volume levels equilibrate, and then volume expansion occurs secondary to an increase in renin, aldosterone, vasopressin, and an increase in albumin production.

Laboratory tests: Lab values reveal a mild anemia (Hgb >13 g/dL in males and Hgb >11.5 g/dL in females). These changes resolve within 3 to 5 days of discontinuing exercise.

Treatment: Dilutional anemia should not negatively affect athletic performance and treatment is not required.

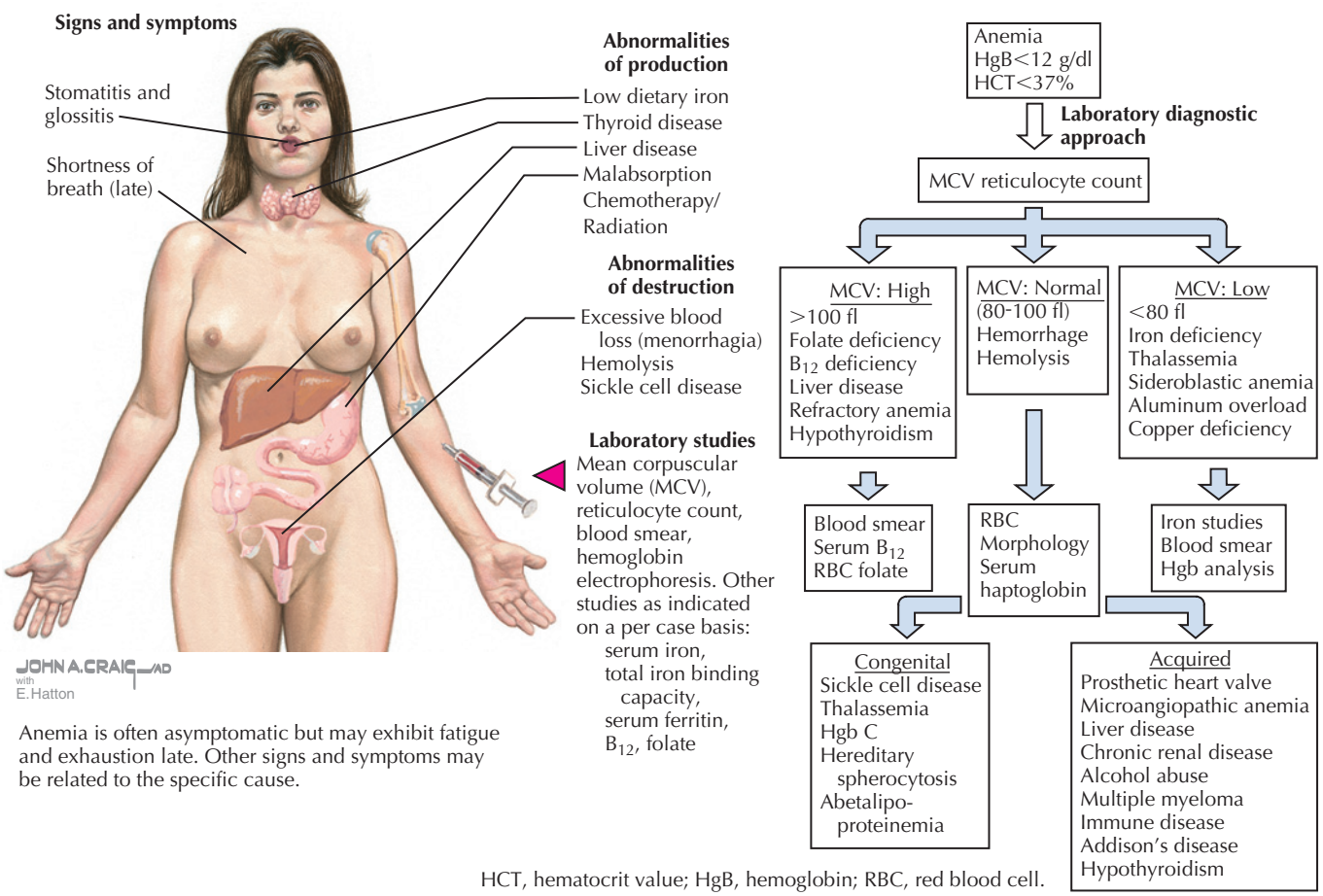
IRON-DEFICIENCY ANEMIA

Epidemiology: Iron-deficiency anemia is the most common true anemia in athletes and the most common nutritional deficiency in the United States. Twenty percent of menstruating women may have low ferritin levels, but only 1% to 3% of the general population will actually be anemic.

Presentation: It may be asymptomatic but oftentimes presents with weakness, lassitude, palpitations, shortness of breath, and pica (craving for starch, ice, or clay). Paleness, glossitis, angular cheilitis, and koilonychias (spoon-shaped nails) may be found on physical exam in severe cases (Fig. 26-1).

Etiology: The underlying etiology is due to either blood loss and/or a nutritional deficit, and it is important to differentiate the cause of the deficiency. Most commonly, the blood loss is from menstruation or the gastrointestinal tract, but it can also be from the genitourinary system. Guaiac-positive stools have been found in 85% of athletes following intense exercise secondary to mucosal ischemia. Nonsteroidal anti-inflammatory (NSAID)–induced gastritis may occur frequently in athletes.

Laboratory testing: Objective findings include a low hemoglobin level in adults (<12 g/dL in women and <14 g/dL in men),



Anemia is often asymptomatic but may exhibit fatigue and exhaustion late. Other signs and symptoms may be related to the specific cause.

HCT, hematocrit value; HgB, hemoglobin; RBC, red blood cell.

Figure 26-1 Anemia.

mean corpuscular volume below 75 fL, a peripheral smear that is hypochromic and microcytic, low serum iron with high total iron-binding capacity, and a serum ferritin below 12 μg/L.

Treatment: Oral iron therapy in the form of elemental iron, 50 mg three times daily as ferrous gluconate, sulfate, or lactate. Absorption is best between meals, and orange juice or ascorbic acid increases absorption. A response should be seen clinically and objectively within the first several weeks of therapy, but should be continued for 6 to 12 months to fully replenish iron stores. Empiric treatment in borderline cases is warranted for 8 weeks, looking for a 1-gram increase in hemoglobin. Women with documented cases of recurrent iron deficiency should be given prophylactic doses of iron.

IRON DEFICIENCY WITHOUT ANEMIA

Overview: Importance in athletic performance is unclear and controversial. There does seem to be agreement in the literature on the similar prevalence of iron deficiency anemia in the athletic population compared to the general population.

Laboratory testing: Lab values will show a low ferritin level with a normal hemoglobin.

Treatment: Most studies show there to be little or no benefit to iron supplementation on exercise performance without anemia. There are conflicting studies on the effects of iron deficiency without anemia on athletic performance. If an athlete presents with iron deficiency and a borderline or low hemoglobin, a trial of oral iron therapy may be considered to determine if there is a rise in hemoglobin.

FOOT STRIKE HEMOLYSIS/EXERCISE-INDUCED HEMOLYSIS/HEMOGLOBINURIA

Overview: Rarely severe enough to cause clinically significant anemia or iron deficiency. Typically, healthy individuals can counteract the degree of hemolysis with reticulocytosis.

Etiology: Mechanical forces from muscle contractions and heel-strike have been shown to be a reproducible cause of RBC hemolysis leading to hemoglobinuria. An elevated body temperature is also thought to play a role in the disruption of the RBC cell membrane leading to an increase in RBC fragility. The increase in RBC fragility leads to what has been described as runner's macrocytosis secondary to the loss of the older microcytes. Not only has the hemolysis associated with exercise been documented in the elite endurance athlete but it has also been documented in swimmers, dancers, rowers, and triathletes. Once hemolysis occurs, hemoglobin and other intracellular contents are released into the intravascular space. Haptoglobin, an intravascular protein, binds to the free hemoglobin, transporting it to the liver for processing and recycling of iron. When haptoglobin becomes saturated, the free hemoglobin spills into the urine, producing hemoglobinuria.

Laboratory studies: A urinalysis will reveal hemoglobinuria, which will resolve within 3 to 5 days with exercise cessation. Lab values reveal a macrocytic anemia with an increased reticulocyte count. Haptoglobin may be low or immeasurable, leading to urinary loss of iron.

Treatment: Treatment is not necessary but one could consider changes in gait, shoes, running surfaces, and intensity of one's training regimen.

SICKLE CELL TRAIT (SCT)

Overview: Sickle cell trait (SCT) is present in 5% to 8% of the African-American population. In SCT, both normal hemoglobin A and abnormal hemoglobin S are produced, which does not seem to affect exercise capacity. With exercise, an acidosis occurs to improve oxygen delivery and leads to an increase in deoxygenated hemoglobin levels.

Laboratory testing: Hemoglobin electrophoresis reveals there to be one gene for normal hemoglobin A and one gene for hemoglobin S.

Risks: Athletes with SCT have been shown to have an increased risk of sudden cardiac death (SCD), splenic infarction, and hematuria. In 1987 the U.S. military found that recruits with SCT had a 28 times increased risk of SCD compared to African-American recruits who tested negative for SCT.

Exercise guideline: Guidelines to decrease the risks associated with SCT seek to prevent overexertion and dehydration while gradually acclimatizing athletes to conditioning in the heat and with altitude changes. Activity and conditioning modifications as described earlier are needed to decrease morbidity and mortality associated with SCT. Recommendations for exercise include encouraging preseason training and conditioning, as well as a gradual progression of exercise and allowing for longer periods of recovery between repetitions and workouts.

EFFORT-INDUCED THROMBOSIS

Overview: Also known as Paget-Schroetter syndrome, effort-induced thrombosis is a rare cause of upper extremity deep venous thrombosis.

Epidemiology: Typically presents in the athlete involved in strenuous upper extremity activity. Theoretically, the upper extremity veins become compressed by hypertrophied anterior scalene muscles or a cervical rib during strenuous upper extremity motion. Typically occurs in the dominant extremity as those muscles become more hypertrophied compared to the nondominant side.

Presentation: Unilateral upper extremity symptoms including pain and swelling, which worsen with activity.

Diagnostics: Ultrasonography is the initial noninvasive test of choice for the diagnosis of effort-induced thrombosis but magnetic resonance imaging and computed tomography may also be used. A hypercoagulability workup should begin to rule out any underlying disease.

Treatment: May involve a combination of surgical decompression, anticoagulation, or thrombolytic therapy. Symptoms should ultimately resolve with treatment.

VENOUS THROMBOEMBOLISM (VTE)

Epidemiology: Venous thromboembolism (VTE) affects 1 in 1000 people per year. Virchow's triad includes stasis, endothelial injury, and hypercoagulability and helps identify those with risk factors for thrombosis.

Risk factors: Risk factors for thrombosis in the athletic population include trauma, immobilization after injury, frequent travel, hemoconcentration after exertion, and oral contraceptives.

Treatment: Some literature recommends the use of low–molecular weight heparin (LMWH) for the prevention of VTE after injury requiring immobilization or prior to air travel in those at risk, but the cost is very high. Aspirin has been shown to be an alternative but it is much less effective. Basic precautions during flights include hourly aisle walks, not crossing legs, wearing loose clothing, adequate hydration with water or juices, low-fat meals, and using aspirin or LMWH if at risk for thrombosis. Travelers with one or more risk factors for VTE should consider compression stockings and/or LMWH for flights over 6 hours. Treatment for a first episode VTE with reversible risk factors should be for at least 3 months, whereas those with idiopathic VTE should be treated for 6 months. Recurrent episodes should be treated for 12 months, and after three episodes, patients should be anticoagulated indefinitely.

Exercise guidelines: Athletes with an acute VTE on anticoagulation should not participate in contact or collision sports, but may participate in noncontact sports and exercise with proper counseling.

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Renal and Genitourinary Problems

27

Kevin E. Burroughs

ANATOMY

Genitourinary system: Composed of internal and external organs of the urinary system and genital organs. Both systems are contained in lower abdomen and pelvic region.

Urinary system: Comprised of kidneys, ureters, urinary bladder, and urethra.

Genital system: Male (penis, testicles), female (ovaries, Fallopian tubes, uterus, vagina, vulva).

Kidneys: Located in retroperitoneal upper lumbar area of abdomen (Fig. 27-1). Upper third of right and upper half of left located under 12th rib. Posteriorly, are protected by psoas, paravertebral, and latissimus dorsi muscles. Kidneys contained in cushion of pericapsular fat. Ureters run along posterior peritoneal wall and are protected by muscles of posterior abdominal wall; are most vulnerable where they cross bony brim of pelvis. Bladder lies within pelvis and is most vulnerable when full.

Female genitourinary system: Situated within the pelvis, except for vulva, which is external (Fig. 27-2).

Male genitourinary system: Prostate and internal portion of male urethra located within pelvis. Penis, scrotum, and testes are located externally and are most vulnerable in men (see Fig. 27-2).

PHYSIOLOGY

- The major function of the kidney is to maintain a constant extracellular environment.
- The kidneys regulate the excretion of fluid and electrolytes.
- Daily urine volume may vary from 500 mL to 15 L.
- Renal blood flow at rest is approximately 1100 mL per minute. Renal blood flow is approximately 20% of cardiac output.
- Oxygen consumption of kidneys at rest is 26 mL per minute. Consumption is 10% of resting metabolism.
- Volume of urine determined primarily by antidiuretic hormone (ADH).
  - ADH regulates water reabsorption by increasing permeability of distal tubule of nephron and collecting duct.
  - ADH is released from posterior lobe of pituitary.
  - ADH is released in response to signals from supraoptic nucleus of hypothalamus.
  - The main stimuli for release of ADH:
    - Increased signals from osmoreceptors in hypothalamus.
    - Decreased blood volume.
    - Increased plasma angiotensin concentration.
- At rest, 15% to 20% of renal plasma flow is continuously filtered by glomeruli.
  - Results in 170 L of filtrate per day.
  - 99% is reabsorbed in tubular system.
- Exercise results in a reduction of renal blood flow proportional to the intensity of the event.
  - Moderate exercise (50% $\dot{V}O_2$ max) results in a 30% reduction; strenuous exercise leads to a 40% to 50% decrease in both renal blood flow and glomerular filtration rate (GFR).
  - During maximal exercise (65% $\dot{V}O_2$ max), there is a 75% reduction in renal blood blow, which is approximately 1% of the cardiac exercise output.
- Renal blood flow decreases as blood is shunted to exercising muscle.
  - Decreases even more if individual is dehydrated.
  - Usually returns to pre-exercise levels within 60 minutes.
- **Mechanism for decreased renal blood flow** is due to increased levels of epinephrine and norepinephrine and constriction of afferent and efferent arterioles.
  - Drop in renal blood flow is proportionate to intensity of exercise.
  - Glomerular filtration is usually maintained but does decrease.
  - Free water clearance decreases even in short exercise periods.
  - Transient proteinuria may develop.
  - There is significant reduction in sodium (Na^+) excretion with increased tubular resorption.
  - ADH release may increase threefold in heavy exercise, helping to prevent free water loss and dehydration.
  - Increased excretion of white blood cells, red blood cells, hyalin, and granular casts occurs.
- Damage to renal parenchyma does not occur during heavy exercise. Renal changes are related to constriction of renal vasculature.

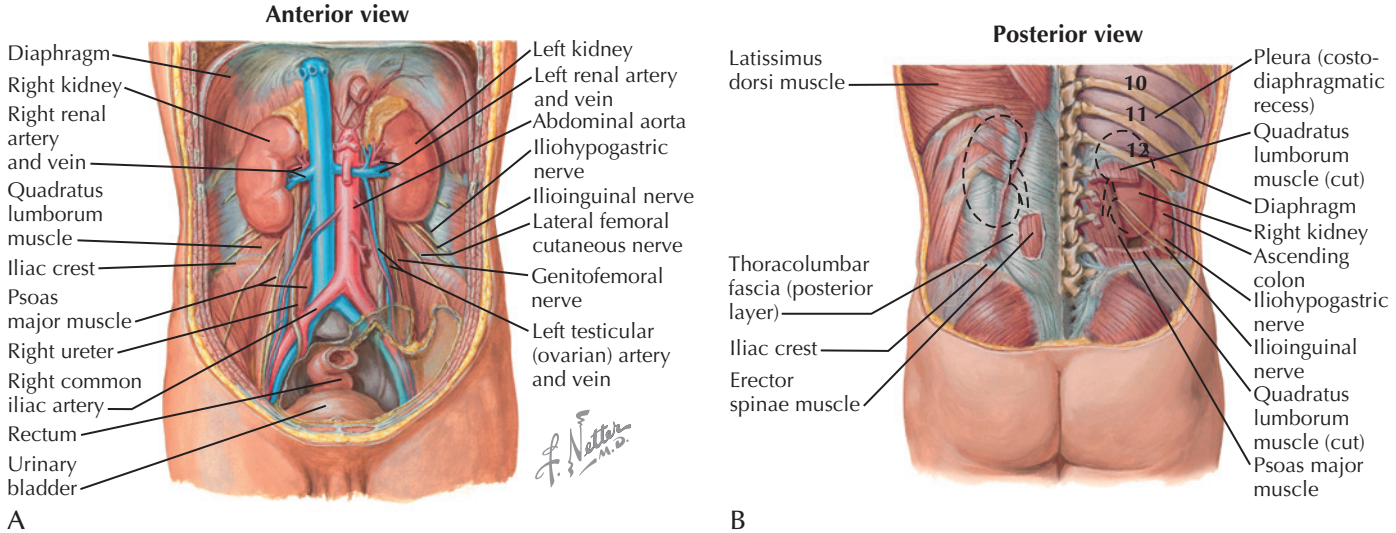


Figure 27-1 Kidneys In Situ.

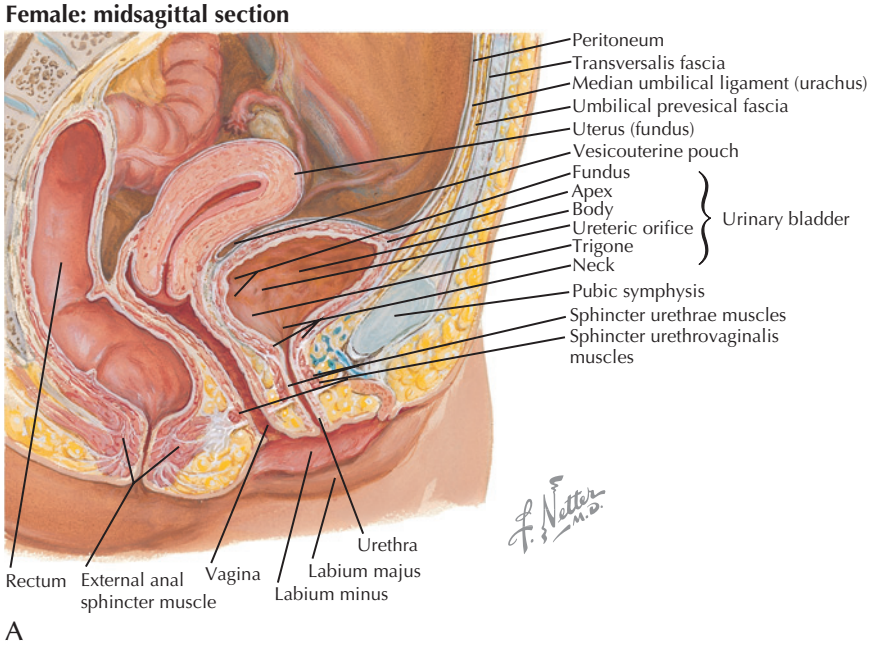


Figure 27-2 Genitourinary System.

HEMATURIA

- Can be macroscopic, gross, or microscopic (Fig. 27-3).
- Microscopic generally defined as more than 3 RBC per high power field.
- In athletes, rates of hematuria can be as high as 75% to 80%; it occurs in both contact and noncontact sports.
- Although hematuria typically resolves within 48 to 72 hours, in ultra-marathoners resolution may take up to 7 days.

Etiology

Overview: Etiologic factors can be categorized by site (kidney vs. bladder).

Nontraumatic renal: Decreased renal blood flow (RBF) (700 mL per minute to 200 mL per minute, with decrease proportional to intensity) leads to ischemia in the nephron, increased permeability, and subsequent passage of RBCs. Also, the increased glomerular filtration pressure, secondary to efferent vasoconstriction, leads to passage of RBCs at the glomerulus. Clots are usually not renal in etiology; dysmorphic cells are suggestive.

Traumatic renal: Obvious direct contact (helmet, ski pole, balance beam, etc.). Indirect trauma from repetitive jarring during running, jumping.

Traumatic bladder: Sports hematuria of bladder origin is almost always traumatic. Thought to occur from repetitive contact of the flaccid posterior vesicle wall against bladder base or trigone (known as "bladder slap"); it has been described in runners and also in snowmobilers. Damage usually a result of repetitive, cumulative effects. Cystoscopy shows damage of the superficial urothelium or contusion. Incidence can be decreased if bladder is partially filled, and adequate hydration maintained.

Prostate/Urethra: Usually traumatic, most often seen in cyclists. Adjusting seat height and pitch will usually alleviate.

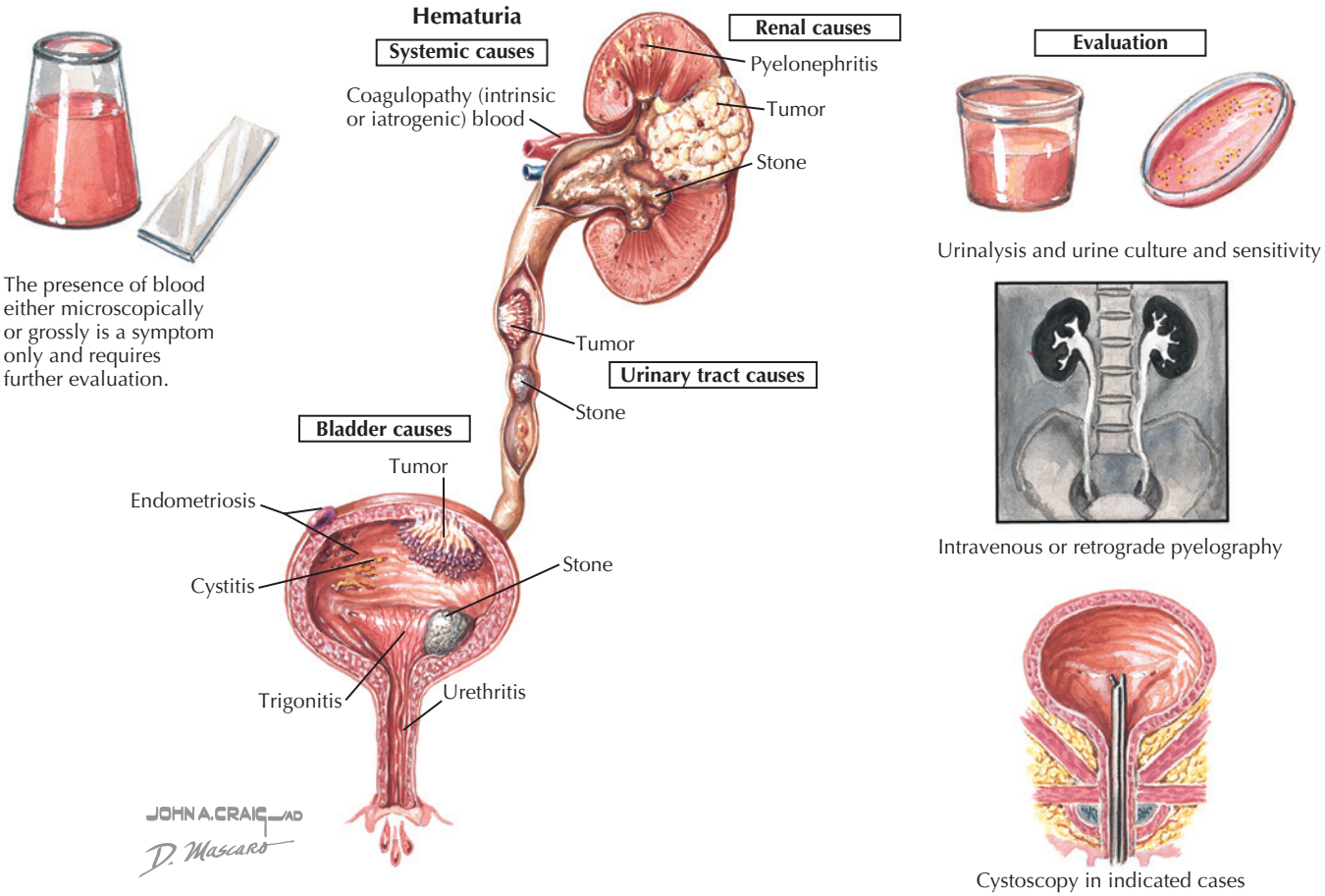


Figure 27-3 Hematuria.

Causes

- Direct kidney injury
- Renal vein kinking
- Bladder contusion
- Preexisting pathology
- Nephrolithiasis
- Urinary tract infection
- Drug or medication use (including penicillin, cephalexin, thiazides, allopurinol, nonsteroidal anti-inflammatory drugs [NSAIDs], aspirin, furosemide, and oral contraceptives)
- Red but nonbloody urine attributed to beets, berries, food coloring, phenazopyridine, phenytoin, ibuprofen, nitrofurantoin, sulfamethoxazole, and rifampin

Diagnostic Considerations

- Timing of hematuria is important to consider:
  - Initial hematuria often urethral in origin
  - Terminal hematuria may originate in bladder or posterior urethral
  - Continuous hematuria may originate in upper urinary tract (kidney, ureter, bladder)
- Some have advocated using mean corpuscular volume (MCV) and red blood cell (RBC) morphology to help establish the origin of hematuria
  - Casts or dysmorphic appearance of cells consistent with glomerular origin
  - MCV less than 72 fl considered to be of glomerular origin
  - MCV more than 72 fl considered to be of nonglomerular origin

Treatment

- If no concerning history, physical exam or diagnosis, stop exercise and/or suspected medications and repeat urinalysis after 24 to 72 hours.
  - If urine clears after 24 to 72 hours, and patient is under 40 years, can diagnose exercise-induced hematuria.
  - If gross or microscopic hematuria persists, or patient is over 40 years, need:
    - Culture, urine cytology, PT/PTT
    - Serum creatinine, blood urea nitrogen (BUN), CBC, sickle cell preparation (in African-Americans)
    - Renal ultrasound or intravenous pyelogram (IVP)
  - If normal, should consider excretory urogram and cystography to exclude bladder lesions, especially if patient is over 40 years old.
  - If testing remains normal and hematuria persists, must consider intrinsic renal disease:
    - Creatinine clearance and protein excretion should be measured.
    - Ultrasound, retrograde pyelography, and computed tomography scan may be useful.
    - Indications for renal angiography and renal biopsy are controversial.
- Athletes with benign hematuria secondary to exercise may continue to be active:
  - Should be encouraged to drink quantities of fluids before exercise
  - Should avoid dehydration

- Exertional hematuria is a diagnosis of exclusion. Must be related temporally to exertion and must resolve within 3 to 5 days of discontinuing or reducing activity.

PROTEINURIA

- Occurs in many sports; present in up to 70% of athletes after exertion and in 5% to 85% of screening urinalysis (UA) on all patients.
- Normal protein excretion is 30 to 45 mg per day.
- Exertional proteinuria usually 2+ to 3+ by dipstick measurement.
- Quantitative measurement shows range of 100 to 300 mg in 24 hours.
- Caused by alterations in renal hemodynamics with vigorous exercise:
  - Have acute decrease in renal blood flow with maintenance of glomerular filtration rate
  - Caused by elevations in renin, angiotensin II, and ADH
- Usually occurs within 30 minutes of exercise and clears in 24 to 48 hours.
- More common following strenuous, prolonged exercise; evidence suggests a direct relationship between intensity of exercise and amount of proteinuria.

Testing

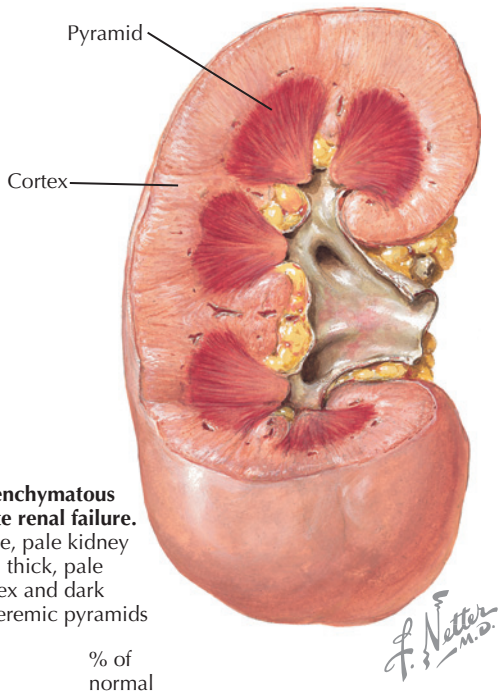
- Urine dipstick is excellent for screening.
- Possibility of false positive reaction should be considered in all positive tests; causes include:
  - Highly concentrated urine (specific gravity > 1.030)
  - Contamination with antiseptics
  - Pyridium (phenazopyridine HCl)
  - Highly alkaline urine (pH > 8)
- Repeat with UA in 24 to 48 hours to assure true positive.
- History should be taken, including personal or family history of:
  - Renal disease
  - Anemia
  - Hypertension
  - Medication use (e.g., protein powder supplements, NSAIDs, antibiotics)
- If repeat positive, must exclude benign orthostatic proteinuria.
  - Patient voids before bedtime; record protein.
  - Go to bed, lying flat. At midnight void, remaining flat. Dipstick protein; save.
  - Void at 6 A.M., again lying flat; dipstick, save.
  - Repeat at 7 to 7:30 A.M., again lying flat; dipstick, save.
  - Arise; collect sample at 9 A.M., standing or sitting.
- If proteinuria persists in supine position, more extensive testing should be undertaken.
  - Serum tests for renal function
  - 24-hour urine for total protein, creatinine, creatinine clearance
  - Fasting blood glucose
  - CBC or other tests as medically dictated

ACUTE RENAL FAILURE

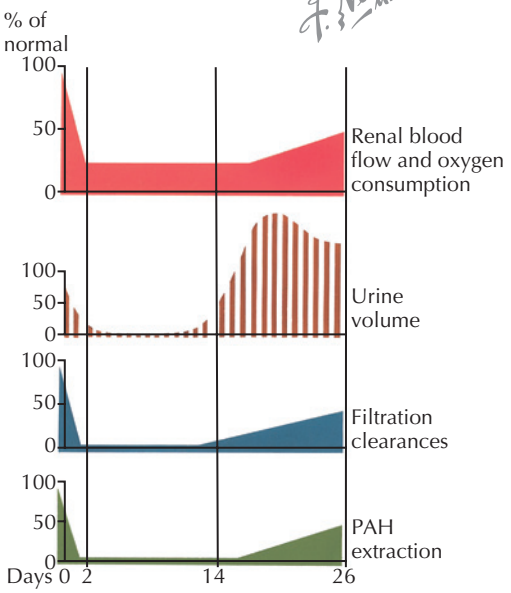
- Uncommon in sports, but occurrence is well documented.
- The combined effects of exercise-to-exhaustion, dehydration, hyperpyrexia, and rhabdomyolysis culminate in renal dysfunction (Fig. 27-4).

Rhabdomyolysis

- Combined effects mentioned above lead to release of muscle enzymes and myoglobin that precipitates in renal tubules.
- Creatine kinase (CK) levels correlate well with myoglobin concentrations in the blood.



Parenchymatous acute renal failure. Large, pale kidney with thick, pale cortex and dark hyperemic pyramids



Circulatory and other selected functional disturbances in the course of acute renal failure. Urine flow, filtration clearances, and paraamino hippurate (PAH) extraction are much more affected than renal blood flow and oxygen consumption.

Figure 27-4 Acute Renal Failure.

- Although 20,000 IU per liter is suggested as a threshold for treatment and hospitalization, levels of up to 20,000 have been reported in sports in asymptomatic individuals.
- Renal failure from exercise-induced rhabdomyolysis and myoglobinuria is not inevitable.
- In certain situations (e.g., underlying disease, dehydration, environmental heat stress, or genetic predisposition [sickle cell trait]), a buildup in myoglobin leads to obstruction of flow and further insult and hypoxemia to the nephron.
- A large number of disorders may lead to rhabdomyolysis:
  - Exercise stress, heat illness
  - Ethanol and many drugs

- McArdle's syndrome—familial lack of muscle phosphorylase
- Sickle cell trait

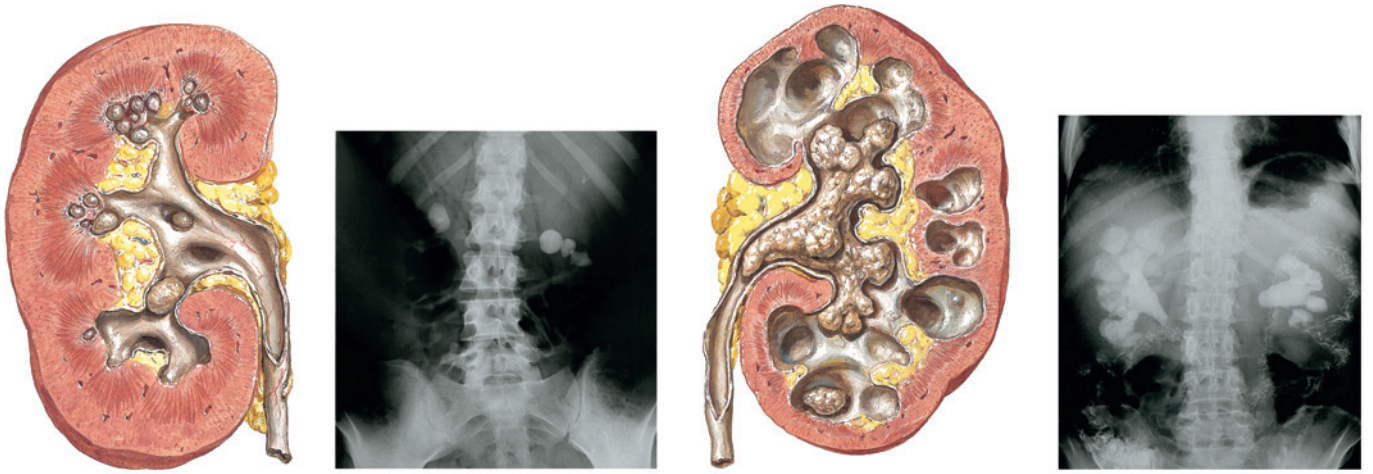
- Acute renal failure with rhabdomyolysis may also be associated with:
  - Disseminated intravascular coagulation
  - Hyperkalemia, hyperphosphatemia, hyperuricemia
  - Hypocalcemia—caused by precipitation of calcium phosphate in muscle from hyperphosphatemia
- A patient with acute renal failure should be treated according to standard treatment guidelines.
- Prevention is primarily related to proper hydration before and during exercise.

NEPHROLITHIASIS

- Occurs in 0.5% of general population (Fig. 27-5).
- Peak incidence of first stone formation is in the 4th and 5th decade of life
- Most stones are calcium: 85% in men, 65% in women.
- Remainder in men are often uric acid stones.
- Remainder in women are often infection-related struvite stones.
- Composition of every stone should be analyzed:
  - Greatly affects treatment against recurrence
  - Recurrence rate about 10% per year
- Renal colic begins suddenly and intensifies over 15 to 30 minutes:
  - Steady, unbearable pain that can cause nausea and vomiting
  - Pain often passes downward from flank anteriorly toward groin
- Stones less than 5 mm usually pass spontaneously; 7 mm or larger have a poor chance of passing spontaneously.
- Stone formation is the result of numerous physiologic causes:
  - Hypercalciuria, primary hyperparathyroidism
  - Hyperuricosuria, cystinuria
  - Deficient urinary inhibitors
  - Urea-splitting organisms in infections

MEDICATION SIDE EFFECTS

- NSAIDs drugs may have deleterious effects because of their ability to inhibit cyclooxygenase within the kidney (Fig. 27-6).
- Cyclooxygenase is the rate-limiting enzyme for synthesis of prostaglandins (PG) from arachidonic acid.
- Two cyclooxygenase enzymes have been determined: COX-1 and COX-2
  - COX-1 regulates PGE_2 synthesis in the inner cortex and outer medullary regions, which modulates tubular Na and chloride (Cl) reabsorption, and attenuates vasopressin-induced reabsorption of water.
  - COX-2 is associated with media smooth muscle and vascular pericytes of the pre- and postglomerular vasculature and regulates arteriolar tone to maintain RBF and GFR.
  - The actual role of COX-2 in exercise-induced changes in renal function is unknown.
- Indomethacin and celecoxib showed no statistically significant decrease in RBF or GFR in healthy individuals undergoing exercise:
  - Under stress conditions (salt restriction, dehydration, and heat), ibuprofen decreased GFR after 45 minutes of exercise at 65% $\dot{V}O_2$ max, compared to placebo or acetaminophen.
  - Both indomethacin and celecoxib showed decreases in free water clearance, which in certain environments or conditions could predispose one to hyponatremia.
- Vasodilatory PGE_2 and PGI_2 play a protective role in the kidney by modulating renal vasoconstriction caused by:
  - Increased renal sympathetic activity
  - Renin-angiotensin II
  - Circulating catecholamines
- In healthy unstressed subjects, renal vasoconstriction is low.
- Renal function can become prostaglandin-dependent in:
  - Hypohydration, sodium depletion
  - Heart failure, hypertension, atherosclerosis
  - Cirrhosis, diabetes, renal disease
  - Aging
- Strenuous exercise also increases renal sympathetic activity, circulating catecholamines, and renin-angiotensin II:
  - Not been shown to affect renal function adversely if the person is healthy.
  - May be problem if coupled with one of the other renal prostaglandin-dependent states (e.g., hypohydrated, or sodium-depleted from heat stress).
- Creatine
  - Creatine monohydrate is widely used as an ergogenic aid.



Multiple small calculi | Plain film: multiple renal calculi | Staghorn calculus plus smaller stone | Bilateral staghorn calculi

F. Netter M.D.

Figure 27-5 Renal Calculi.

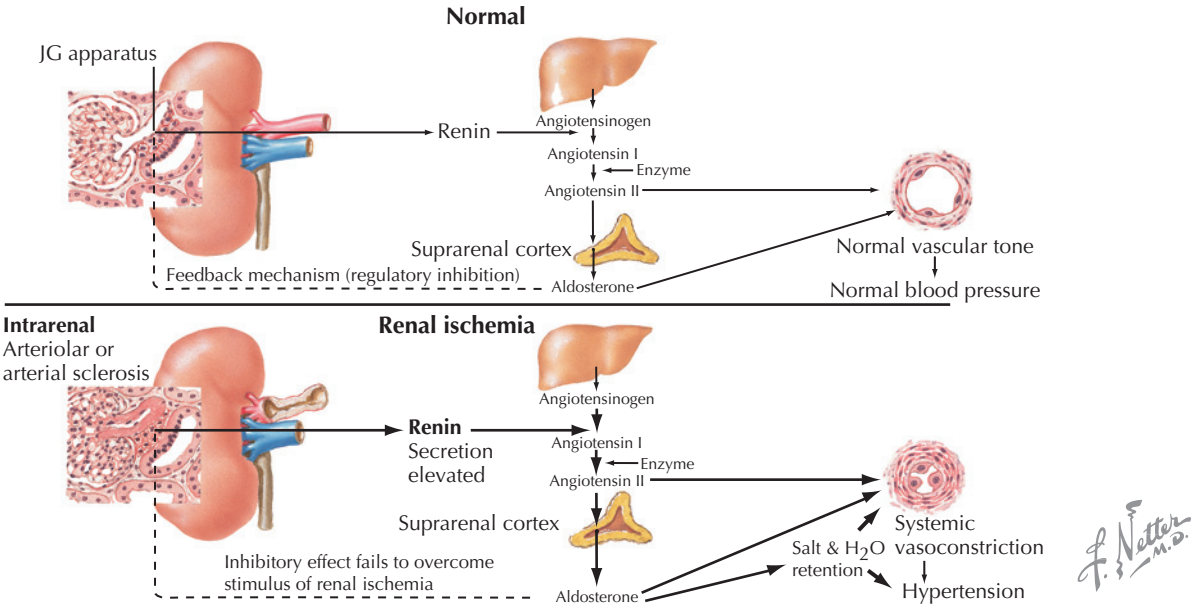


Figure 27-6 Renovascular Hypertension.

- Creatine monohydrate increases muscle stores of creatine, theoretically leading to greater ATP resynthesis.
- End product of creatine is creatinine, which is filtered in glomeruli and excreted.
- In healthy individuals, there is no link between creatine ingestion and renal dysfunction if used in the typical doses, but only short-term studies exist.
- There have been case reports in the literature of acute renal failure in individuals who were long-term, high-dose users.
- Those with established renal disease should avoid supplementation.
- In those using creatine, renal function can be evaluating by measuring, under resting conditions, the albumin excretion rate (normal < 20 micrograms per minute).

UROLOGIC DIRECT TRAUMA

- **Exact incidence is unknown.**
- May be by direct blow or contrecoup injury from high-speed collision.
- May have pain, tenderness, ecchymosis, and hematuria (Fig. 27-7).
- Weak correlation between amount of hematuria and degree of injury
- Hypovolemic shock may result from extensive bleeding
- Athlete who develops hematuria after blow to flank should undergo testing. Computed tomography (CT) scan is the test of choice, intravenous pyelogram (IVP) if CT not available. If IVP shows nonfunctioning kidney or major injury with extravasation, renal arteriogram should be considered.
- Examining physician should check for injury to other abdominal organs.

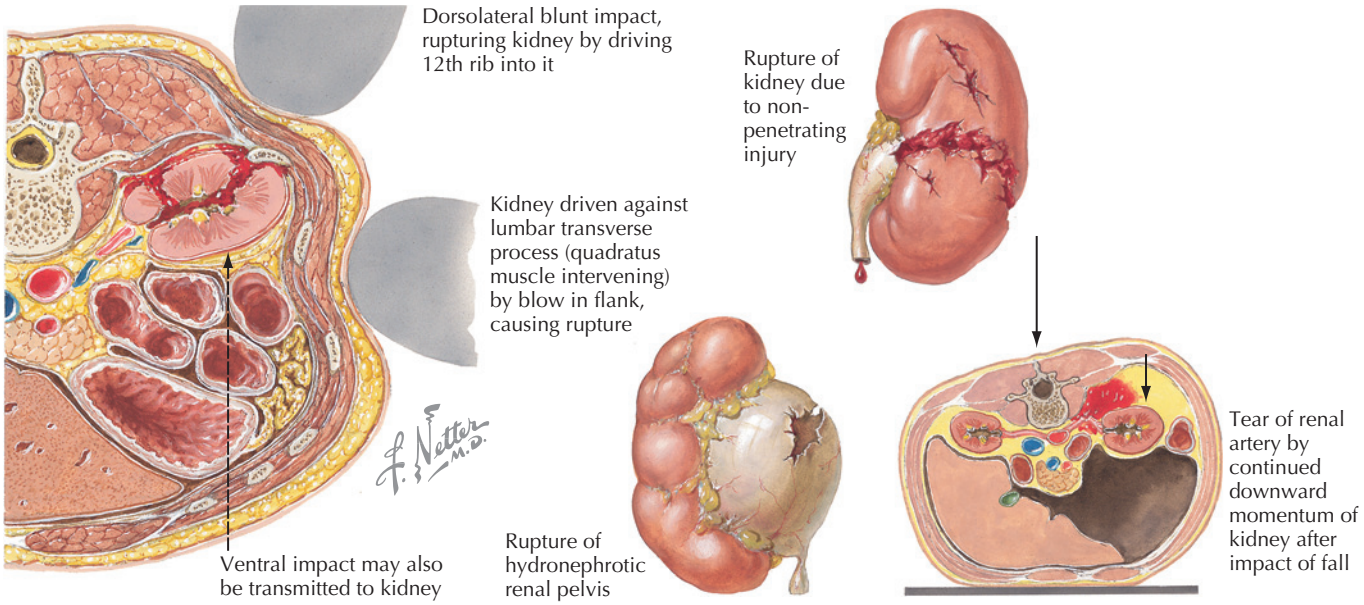


Figure 27-7 Renal Trauma—Nonpenetrating Trauma of Kidney.

- CT scan is the recommended method for initial evaluation of suspected renal trauma, if available.
  - Can establish contusion, which IVP cannot
  - Localization of injury if surgery needed

Five Classes of Renal Injury

See Figure 27-8.

Grade 1: Contusion or subcapsular hematoma without parenchymal laceration. Majority of sports-related renal injuries. Hematuria (microscopic or gross) and flank pain usually present but IVP negative. Treatment usually consists of observation, bed rest, and repeat urinalysis. No sports until hematuria clears, and no contact sports for 6 weeks.

Grade 2: Nonexpanding perirenal hematoma (confined to renal retroperitoneum), or renal or cortical laceration less than 1 cm deep without urinary extravasation. IVP shows extravasation of dye. X-ray may have loss of psoas shadow. Treatment is often observation, bed rest, and repeat urinalysis.

Grade 3: Parenchymal laceration deeper than 1 cm into the cortex without collecting system rupture or urinary extravasation. IVP shows intact capsule with intrarenal extravasation and disruption of pelvicaliceal system. Angiography can be used to further delineate surgical versus nonsurgical cases. Treatment involves observation; surgery in more severe cases.

Grade 4: Major parenchymal laceration extending through the corticomedullary junction and into the collecting system (complete renal fracture), injury to main renal artery or vein with contained hemorrhage. Rare sports injury. IVP shows separation of pelvicaliceal system with intrarenal and extrarenal dye extravasation.

Grade 5: Multiple major lacerations resulting in a shattered kidney or renal pedicle injury. Will present in hypovolemic shock. Rare in sports. Kidneys usually not visualized on IVP. Selective renal arteriogram shows renal vascular damage. Treatment always involves surgery.

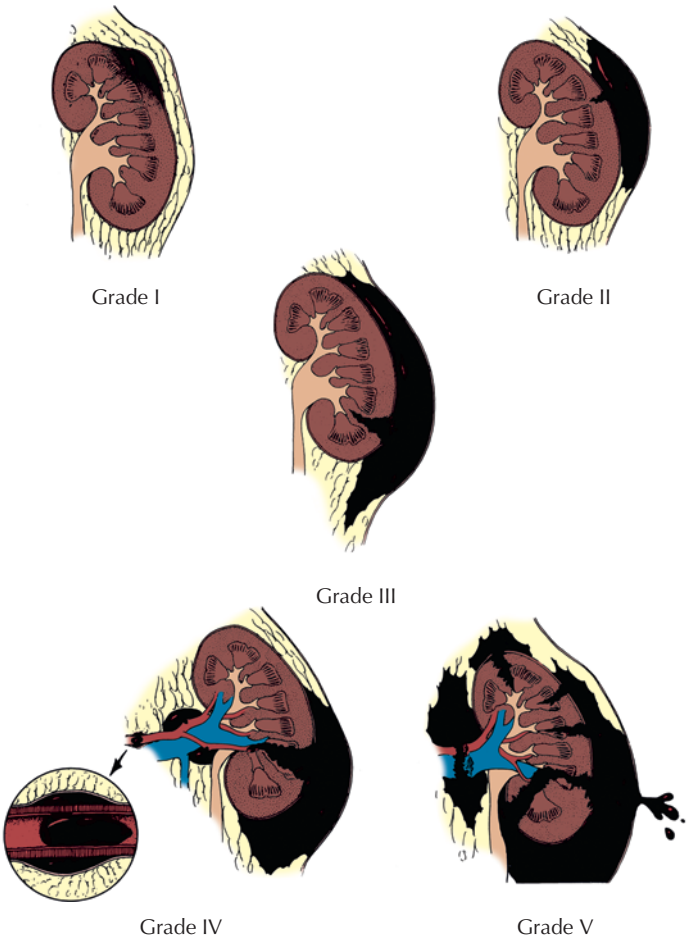


Figure 27-8 Classification of renal injuries by grade. Based on the organ injury scale of the American Association for the Surgery of Trauma. *(Reprinted with permission from McAninch JW, Santucci RA: Genitourinary trauma. In Walsh PC, Retik AB, Vaughan ED Jr, et al [eds]: Campbell's Urology, 8th ed. Philadelphia: Elsevier, 2002.)*

Injuries to Abdominal Areas

- Renal trauma in sport usually involves contusions or intracapsular injuries.
  - Blunt trauma causes more than 90% of pediatric renal injuries and the kidney is injured in pediatric patients more often than the spleen, liver, pancreas, bowel, lung, heart, or great vessels.
  - Participation of athlete with one kidney
    - The incidence of unilateral renal agenesis is 1 in 500 to 1 in 1800, so it is likely that many athletes are unaware of having one kidney; most found incidentally or after nephrectomy.
    - To develop participation recommendations consider age of athlete, type of sport, motivation for participation, as well as position, size, and function of remaining kidney.
    - Most agree the athlete should be excluded from contact/collision sports if the remaining kidney is ectopic, multicystic, or has any degree of obstruction/impairment of function.
    - Only consensus opinion is from the American Academy of Pediatrics, which gives a qualified yes to participation.
- Ureter injury usually associated with major renal damage:
  - Rare in sports.
  - Must consider pelvic and lumbar vertebrae fracture.
- Bladder injuries most often related to blunt trauma on distended bladder.
  - Two types of bladder injury are common: contusion and bladder rupture.
  - May require urinalysis, cystogram, and retrograde pyelogram for diagnosis.
  - Contusion:
    - May have suprapubic pain and guarding.
    - May pass small clots and have dysuria and hematuria.
    - Degree of hematuria does not correlate well with severity of injury.
    - Severe contusions require use of indwelling catheter for 7 to 10 days with antibiotics.
  - Bladder rupture:
    - Rare in sports.
    - Usually associated with pelvic fracture.
    - May be intraperitoneal or extraperitoneal.
    - Requires immediate surgery.
- Bladder injury present in 10% to 15% of pelvic fractures.

STRESS INCONTINENCE

- Involuntary loss of urine during physical exertion:
  - Viewed as bladder outlet incompetence.
  - Women twice as likely to have difficulty as men.
  - Denial is common and the average person waits 7 to 9 years to seek help.
- 28% to 47% of regularly exercising women report some degree of incontinence.
- Sports requiring jumping, running, or prolonged Valsalva maneuver may exacerbate.
- Urethral sphincters:
  - Proximal or internal sphincter at bladder neck composed of smooth muscle and innervated by sympathetic system.

- Intrinsic urethral sphincter composed of type I (slow-twitch) muscle fibers innervated by pudendal and sympathetic nerves.
- External urethral sphincter composed of type II striated muscle innervated by perineal branches of the pudendal nerve.

- Treatment options
  - Behavioral—emphasis on establishing proper pelvic muscle function:
    - Kegel exercises; other pelvic floor exercises
    - Pelvic floor exercises with biofeedback
    - Vaginal cones
  - Pharmacologic:
    - Internal urethral sphincter under alpha-adrenergic control
    - Alpha-adrenergic agents such as phenylpropanolamine are often effective
  - Surgical—effective treatment but should be considered *after* other options

GENITAL INJURIES

- Trauma to the unprotected perineal area of either gender may cause hematoma formation.
- **Testes** are paired organs that descend into scrotum in 8th fetal month.
- Testes subject to contusion, torsion, or epididymitis.

Male Injuries

Direct Testicular Trauma

- Direct trauma to scrotum may cause testicular contusion (Fig. 27-9).
- Can cause pain, pallor, nausea, and anxiety.
- Ice and elevate to control bleeding and swelling for 12 to 24 hours. If pain persists more than 1 hour, must rule out torsion.
- If expanding mass cannot be transilluminated or if epididymis cannot be palpably separated from testicle, consider fracture of testicle or epididymis.
- Testicular ultrasound can help differentiate less serious injuries such as scrotal hematomas, hydroceles, and hematoceles that can be managed conservatively from surgical emergencies such as testicular rupture (see Fig. 27-9).
- There is evidence that significant testicular trauma can lead to subfertility and atrophic testes

Preexisting Scrotal Abnormalities

- May increase risk of testicular injury; therefore, evaluation during the preparticipation exam is important.
- Unilateral anorchia found in 5% of boys presenting for surgical exploration—thought to be most commonly due to torsion and infarct before birth.
- Retractile and undescended testes:
  - Undescended testicles most commonly reside permanently within inguinal canal and cannot be pulled into scrotum.
  - Require surgical repair.
  - At increased risk for testicular cancer and decreased fertility.

Torsion of Spermatic Cord

- Mobility of testis limited by tunica vaginalis, a single ligament that attaches lower end of spermatic cord and epididymis to scrotal lining.

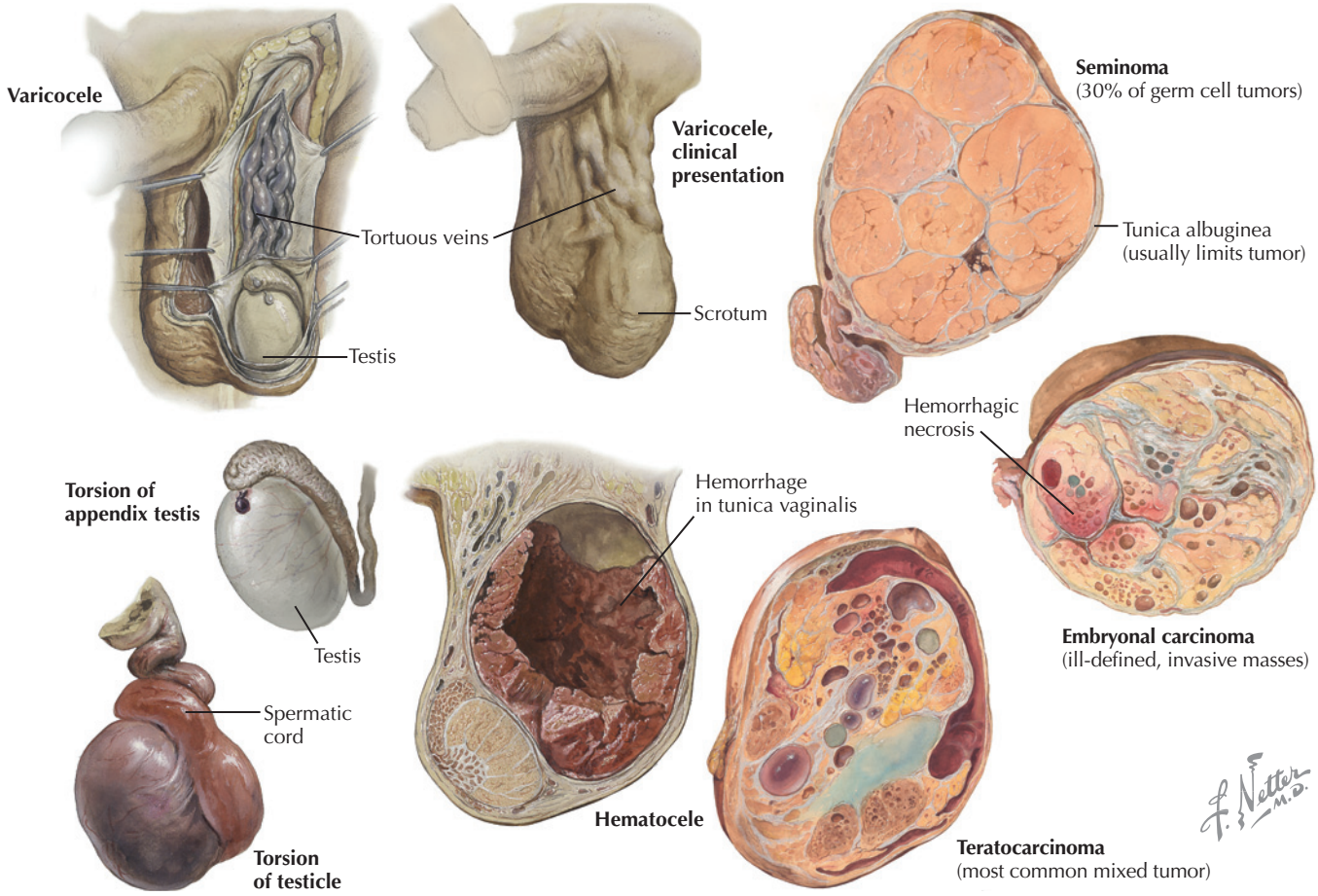


Figure 27-9 Varicocele, Hematocele, Torsion.

- Extravaginal torsion occurs if tunica vaginalis is loosely attached to scrotal lining:
  - Allows spermatic cord to rotate above testis
  - Not common
  - Occurs almost exclusively in neonates
- Intravaginal torsion occurs if tunica vaginalis is attached unusually high on spermatic cord, allowing motion of testis below. More common in pubertal and prepubertal boys but can occur in any age group.
- Torsion should be considered whenever scrotal pain and swelling occur:
  - Is a true emergency warranting prompt urologic evaluation
  - Develops increasing abdominal or groin pain
  - Often develops excruciating testicular pain
  - Should be questioned about history of mobile testis
- Physical examination may reveal:
  - Localized tenderness, edema, and hyperemia of scrotal skin
  - Vas deferens may be inseparable from swollen cord
  - High-riding testicle and abnormal position of epididymis
  - Cremasteric reflex usually absent in testicular torsion
- Epididymitis is other major consideration in differential diagnosis:
  - Elevation of scrotum often relieves pain of epididymitis.
  - Elevation of scrotum often increases pain of torsion.
- If patient presents within 4 to 6 hours after torsion occurs:
  - Cooling of scrotal skin, lidocaine (Xylocaine) cord block, and manual derotation may be attempted. Should not delay surgical exploration and repair.
  - Radionuclide scan may distinguish torsion from epididymitis:
    - Clinical suspicion should override negative scan.
    - Surgical intervention should not be delayed for scan.
  - Torsion can often be reduced through external manipulation.

Epididymitis

- Tender indurated epididymis may be palpable early in course.
- Subsequently may become hard and fixed to skin with swollen spermatic cord.
- May develop fever and elevated white blood cell count.
- Elevation of scrotum may reduce pain—must be distinguished from torsion of spermatic cord.
- Urinalysis usually positive for leukocytes.
- Etiologic agent often chlamydia in men under 35 years and *E. coli* in men over 35. Requires appropriate antibiotic intervention.
- Cultures should be obtained, including gonorrhea.

Scrotal Masses

- Evaluation for gynecomastia should be performed when examining scrotal masses (rule out Leydig cell tumors).
- Varicocele present in 9% to 19% of males, and in 30% of infertile men.
  - Varicosities of internal spermatic veins
  - Surgical correction may be needed for:
    - Pain control
    - Diminished ipsilateral testicular size
    - Infertility
- Spermatoceles are cystic masses:
  - Within epididymis or adjacent to testicle
  - Caused by extravasation of sperm from trauma or infection
  - Require treatment if large or painful
- Hydrocele is cystic mass surrounding testicle and epididymis:
  - A collection of peritoneal fluid between parietal and visceral layers of tunica vaginalis, caused by decreased absorption of tunica vaginalis secretion.
  - Caused by trauma, infection, or tumor.
  - Acute hydrocele may contain a malignancy and should be investigated by ultrasound or surgery.
- Hematocele is blood accumulation in tunica vaginalis
  - Does not transilluminate.
  - Treatment includes ice, elevation, and bed rest.
  - Rapidly expanding hematoma may need surgical exploration.
- Testicular cancer is the most common malignancy in 16- to 35-year-old men (see Fig. 27-9).
- Presence of mass in testicle separate from cord and epididymis demands prompt evaluation.
- A mass separate from testicle should be transilluminated; if cannot be transilluminated, ultrasound should be considered.

Penile Injuries

- Penile injuries are not common in sports (see Fig. 27-9).
- **Direct blow** may cause vascular injuries and potential impotency.
  - Caused by straddle injuries or direct blow to pubis
  - Mechanism could lead to partial or complete urethral rupture:
    - Complete rupture has not been described during sports activities.
    - Involves immediate pain, swelling, and perineal ecchymosis.
    - Diagnostic retrograde urethrogram may be needed.
  - Erect penis susceptible to fracture of tunica albuginea:
    - Area of fracture swollen and ecchymotic
    - Penis bent to affected side
    - Urologic emergency—requires hematoma evacuation and tunica repair
- **Penile frostbite** described in runners wearing inadequate clothing in cold weather.
- **Traumatic irritation** of pudendal nerve is not uncommon:
  - Especially common in bicycle racers or touring cyclists.
  - May cause priapism, impotence, paresthesia, or ischemic neuropathy.

Female Injuries

- Vulva is quite vascular—trauma can result in hematoma formation.
- Fall while water skiing may force water into vagina (water douche).
  - This may lead to laceration, and possibly severe internal bleeding.
  - Has also been related to cases of endometritis or salpingitis.

GENITOURINARY INFECTIONS

Numerous types of genitourinary infections exist, with various causative agents (Fig. 27-10).

Urinary Tract Infection

- Five categories of adult patients
  - Young women with uncomplicated cystitis
  - Young women with recurrent cystitis
  - Young women with acute uncomplicated pyelonephritis
  - All adults with complicated urinary infection
  - All adults with asymptomatic bacteriuria
- Most common is bacterial cystitis, usually with:
  - Pyuria
  - Hematuria
  - Colony count of more than 100,000 per mL
- Numerous treatments

Prostatic Disease

Prostatitis

- Acute bacterial prostatitis uncommon:
  - Sudden onset of chill, fever, and pain in back and perineum

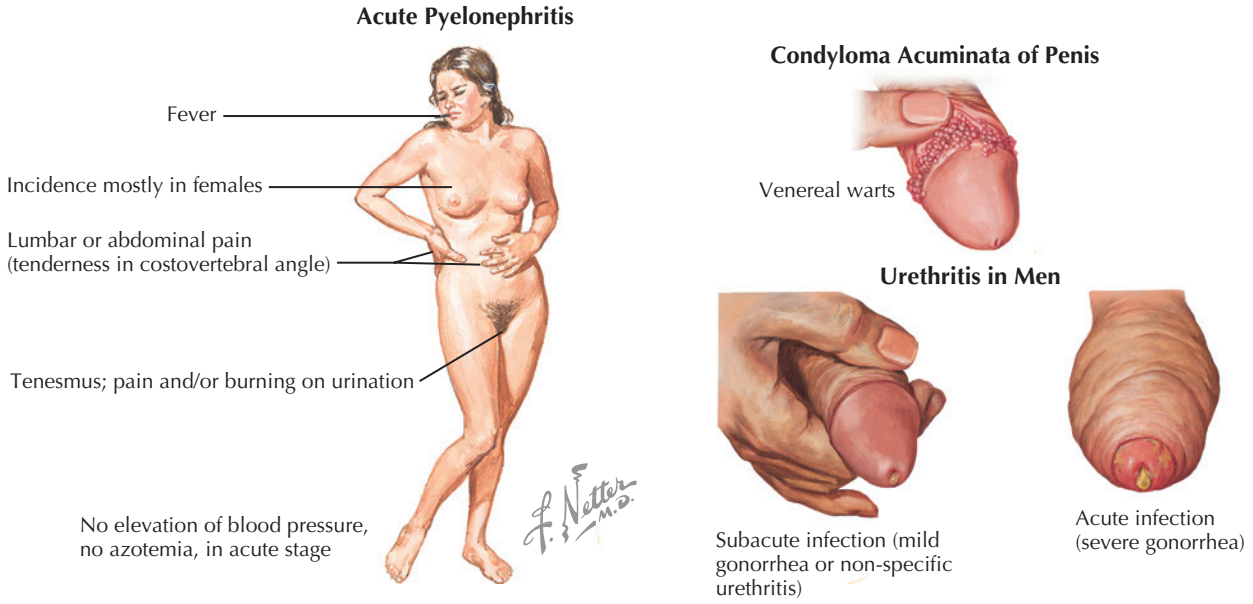


Figure 27-10 Genitourinary Infections.

- Dysuria and obstruction with voiding
- Chronic bacterial prostatitis common cause of recurrent urinary tract infection
- Need to examine both urine and prostatic fluid

Benign Prostatic Hypertrophy

- Symptoms primarily related to bladder outlet obstruction
- Frequent cause of hematuria in men—may have leakage of blood from enlarged veins in benign hypertrophy

Prostatic Cancer

- According to the U.S. Preventive Services Task Force, there is insufficient evidence to recommend for or against routine screening with prostate specific antigen (PSA) or digital rectal exam.
- Those who may benefit are men ages 50 to 70 with average risk, or those over 45 with increased risk.

Sexually Transmitted Diseases

- Diagnosis of sexually transmitted diseases (STDs) should be sentinel event indicating high-risk behavior
- Infections of epithelial surfaces:
  - Chlamydia
  - Gonorrhea
  - Genital warts
- Bacterial syndromes—nonspecific urethritis and epididymitis
- Diseases with genital ulcers or inguinal lymphadenopathy:
  - Syphilis
  - Herpes simplex
  - Chancroid
  - Lymphogranuloma venereum
- Control of STDs based on four major concepts:
  - Education of persons at risk for disease transmission
  - Detection of infection in asymptomatic individuals
  - Effective diagnosis and treatment of current infections
  - Evaluation, counseling, and treatment of sexual contacts
- The Centers for Disease Control Advisory Committee on Immunization Practices recommends the routine use of the human papilloma virus vaccine for girls ages 11 to 12.

WHEELCHAIR ATHLETES

- Neurologic control of urinary tract is often lost after spinal cord injury (Fig. 27-11).
- Paraplegics and quadriplegics at significant risk for:
  - Bladder and kidney infections
  - Kidney stones
  - Bladder distention
  - Urethral fistula
- Kidney damage secondary to infection is the main cause of death in people with spinal cord injury.
- Wheelchair athletes have demonstrated lower incidence and frequency of urinary tract complications when compared with sedentary wheelchair users.
- Autonomic dysreflexia:
  - Athletes with spinal cord injury above T6 level are at risk; autonomic dysreflexia is an uncontrolled sympathetic response from noxious stimuli below the lesion level.
  - Common symptoms include headache, hypertension, flushing, diaphoresis, and bradycardia.
  - Despite the danger associated with autonomic dysreflexia, some athletes attempt to induce it for a competitive advantage referred to as boosting.
  - An athlete may self-induce autonomic dysreflexia by overdistending the bladder, sitting on sharp objects, or by the use of tight leg straps.
  - Theoretically, the elevated blood pressure increases cardiac output and results in improved racing performance.

CYCLISTS

Pudendal neuropathy: Prevalence of perineal and penile/labial numbness is 50% to 91% in competitive cyclists. Typically not pain, only numbness. Risk factors are age above 50, high body weight, cycling time (>10 years, >3 hours per week). Treatment is cessation until numbness resolves; on return to riding, rising from saddle intermittently during rides; wider rear saddle in upright riders or a more narrow rear saddle in riders using the forward racing position.

Erectile dysfunction: Many cyclists report at least transient (1 to 4 weeks) erectile dysfunction. Risk factors are similar to puden-

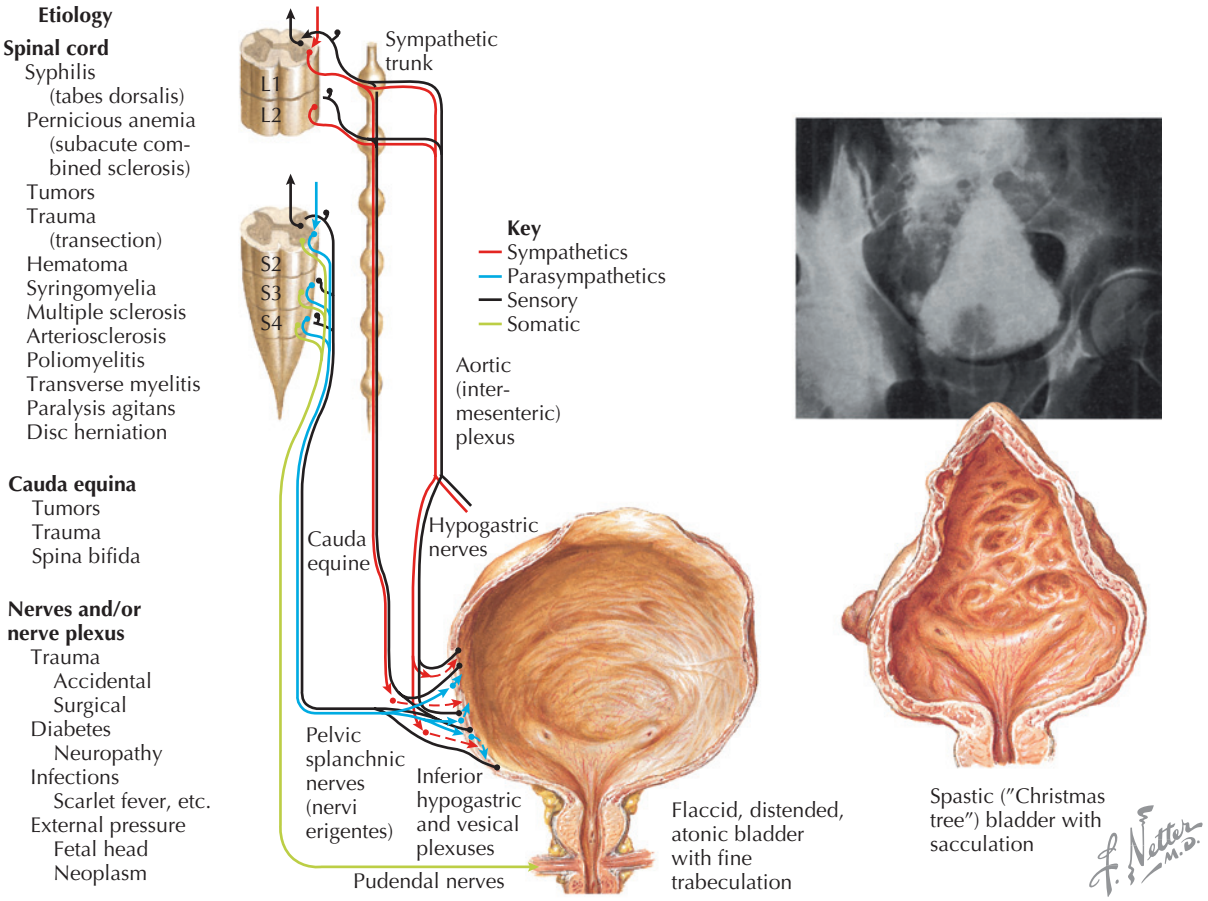


Figure 27-11 Neurogenic Disorders of the Urinary Bladder.

dal neuropathy, and etiology is thought to be neurovascular. Central saddle cutouts have not proven to decrease incidence. Consider adjustments in handlebar height and saddle position

Male infertility: Some evidence of decreased sperm motility during season in elite cyclists. No direct causal evidence exists.

Testicular cancer: Case control study showed increased risk with cycling and horseback riding. Not enough direct evidence to prove link.

UNILATERAL ORGAN

- Great debate in the literature on recommendations for exclusion of sport for those athletes with a solitary kidney, ovary, or testicle.
- In a large trauma registry review of almost 15,000 patients, only 3% had a renal or testis injury, and only 5% of those occurred during team sports.
- Another registry of almost 50,000 patients showed that in patients with a renal injury, no kidneys were lost in a contact sport.
- The only medical guideline on this subject is from the American Academy of Pediatrics Committee on Sports medicine that gives participation with an unpaired testicle, ovary, or kidney a qualified yes.
- This is best decided on a case-by-case basis after discussion with the athlete, the family, the coach and physicians.

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The Athlete with Diabetes

Russell D. White, Dennis Cardone, and Kris Berg

GENERAL CONSIDERATIONS

- Exercise is one component of the treatment triad (medication, medical nutritional therapy, exercise) for diabetes mellitus. Position statements of the American Diabetes Association support exercise for persons with type 1 and type 2 diabetes.
- **Short-term effects of exercise in both types of diabetes are well understood, but long-term effects are less clear with type 1 patients.**
  - Evidence (e.g., studies of diabetic athletes) clearly suggests numerous long-term benefits, including weight loss, reduced risk of cardiovascular disease, increased insulin sensitivity, and improved regulation of blood glucose in type 2 patients; response in type 1 patients is more variable.
  - Long-term exercise does not enhance glucose control (based on A1C determinations) in type 1 patients because of compensation for exercise-induced hypoglycemia.
  - Data show that type 1 patients who were high school athletes have significantly **lower incidence of cardiovascular disease** as adults than type 1 patients who were not.
  - Exercise can help to induce or prolong remission in newly presenting type 1 patients.
- **Persons with diabetes are as trainable as nondiabetic persons if under reasonable metabolic control (i.e., blood glucose).**

KEY TRAITS OF DIABETES PERTINENT TO EXERCISE

- **Type 1 and type 2 diabetic patients using insulin or insulin secretagogues are most susceptible to hypoglycemia.**
  - Available insulin formulations include:
    - Human short-acting (regular) and rapid-acting analogues (insulin lispro, insulin aspart, and insulin glulisine)
    - Human intermediate-acting (NPH) and long-acting analogues (insulin glargine and insulin detemir)
    - Human insulin combinations (NPH/Reg 70/30) (NPH/Reg 50/50) and analogue insulin combinations (insulin aspart protamine/insulin aspart 70/30 and insulin lispro protamine/insulin lipro 75/25
  - Insulin secretagogues include:
    - Sulfonylureas (glimepiride, glipizide, glyburide)—promote second phase insulin secretion
    - Meglitinides (repaglinide, nateglinide)—promote first phase insulin secretion (Fig. 28-1A)
  - Other medications that may contribute to hypoglycemia but rarely cause hypoglycemia alone include:
    - Biguanides (metformin)—may contribute to hypoglycemia when combined with other agents (Fig. 28-1B)
    - Thiazolidinediones (rosiglitazone, pioglitazone)—may contribute to hypoglycemia when combined with other agents (Fig. 28-1C)
    - Alpha-glucosidase inhibitors (acarbose, miglitol)—delays glucose absorption (Fig. 28-1D)
    - Sitagliptin (inhibits dipeptidylpeptidase-4)—slows incretin metabolism
    - Exenatide (incretin mimetic)—may contribute to hypoglycemia when combined with sulfonylureas
    - Pramlintide (analog of human amylin)—may contribute to hypoglycemia when combined with insulin
- **Type 1 patients are prone to ketoacidosis, whereas type 2 patients rarely experience this problem.**
- Hypoglycemia may occur during, immediately after, or several hours following exercise.
- Hypoglycemia and ketoacidosis can be minimized with appropriate monitoring of blood glucose and proper adjustments of diet and medication. Many athletes with diabetes monitor blood glucose six or more times daily.
- Response to exercise depends on several factors:
  - Intensity and duration of exercise
  - Level of metabolic control
  - Type and dose of insulin injected before exercise
  - Site of insulin injection
  - Timing of previous insulin injection
  - Meal relative to exercise
- Competitive athletes may have increased glycemic variability resulting from irregular schedule of intense exercise and travel. They may have to sacrifice some tightness of glycemic control to avoid episodes of hypoglycemia.

BENEFITS OF EXERCISE

- **An athlete's motivation to improve athletic performance may enhance his or her diabetic management** through more frequent blood glucose assessment and closer attention to diet.
- **Consistent, good control of blood glucose reduces microvascular complications in type 1 and type 2 diabetes.** Some degree of reversal of these conditions is possible with prolonged near-normal glucose control.
- **Physical trainability and performance are probably optimized when blood glucose is consistently good** because of more normal substrate utilization, reduced protein degradation (which leads to greater muscle hypertrophy and possibly greater mitochondrial enzymes), greater muscle and liver glycogen, and increased body water with increased heat tolerance.
- **Psychological effects of exercise** include improved self-esteem and self-confidence.
- **Reduction in cardiovascular disease risk factors,** including reduced total cholesterol and low-density lipoprotein cholesterol, increased high-density lipoprotein cholesterol, reduced triglyceride level, reduced blood pressure, increased fibrinolysis, and reduced stress can be attributed to exercise.
- **Increased insulin sensitivity:** Exercise augments the action of glucose transporter 4 (GLUT-4). Reduced insulin or oral hypoglycemic medication doses often result. Medications for some type 2 patients may be discontinued. Moderate-intensity, high-volume resistance training also improves insulin sensitivity in type 2 patients.

CONTRAINDICATIONS FOR EXERCISE

- Exercise electrocardiogram is warranted in diabetic patients with any one of the following:
  - Age older than 35 years
  - Age younger than 25 years and:
    - Type 1 diabetes of more than 15-year duration
    - Type 2 diabetes of more than 10-year duration
  - Presence of any additional risk factors for coronary artery disease
  - Presence of microvascular disease
  - Peripheral arterial disease
  - Autonomic neuropathy
- **Patients with peripheral neuropathy or microangiopathy should avoid exercise that traumatizes feet.**
  - Swimming, cycling, and chair exercises are good alternatives to walking and jogging.

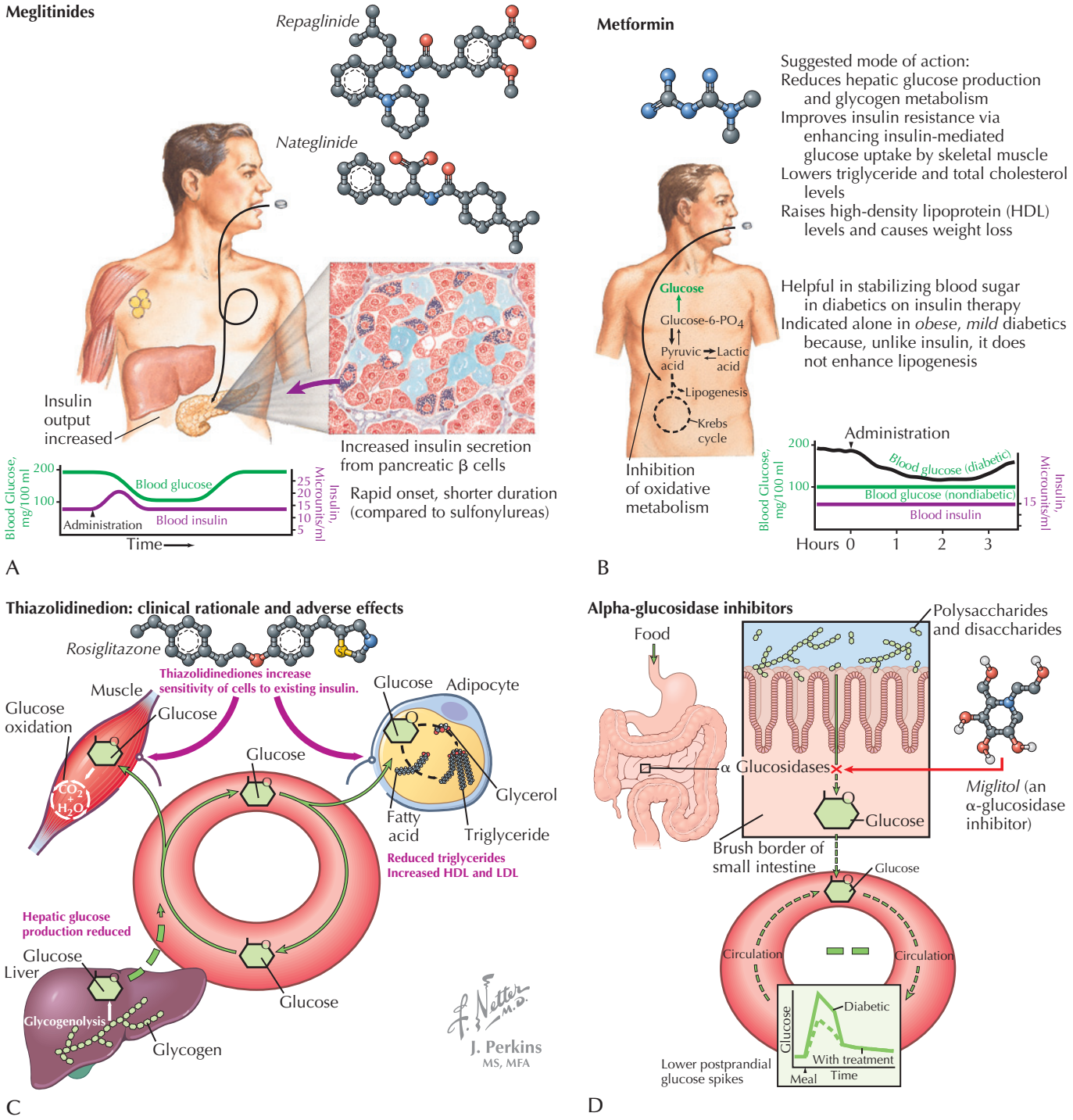

Figure 28-1 Medications That May Contribute to Hypoglycemia.

- **Examine feet daily and keep them well lubricated with an oil, cream, or ointment.** Autonomic neuropathy with decreased sweating predisposes to dry skin. Trim nails carefully; avoid blisters, corns, and calluses; and wear properly fitting shoes and socks.
- **Treat foot injuries immediately to prevent complications.**
- Prevent thick callus formation by periodic filing with a pumice stone.
- Screen for signs/symptoms of early neuropathic joint disease (Fig. 28-2).

- **Proliferative retinopathy precludes:**
  - Anaerobic exercise and exercise involving jarring, straining, or Valsalva-type activity. (e.g., heavy weightlifting training, contact sports, high-impact aerobics, gymnastics, running, boxing, volleyball).
  - Activity that raises heart rate dramatically and systolic blood pressure beyond 170 mm Hg.
  - SCUBA diving because of increased water pressure.
  - Exercise while inverted (some yoga positions, standing on head, hanging upside-down).

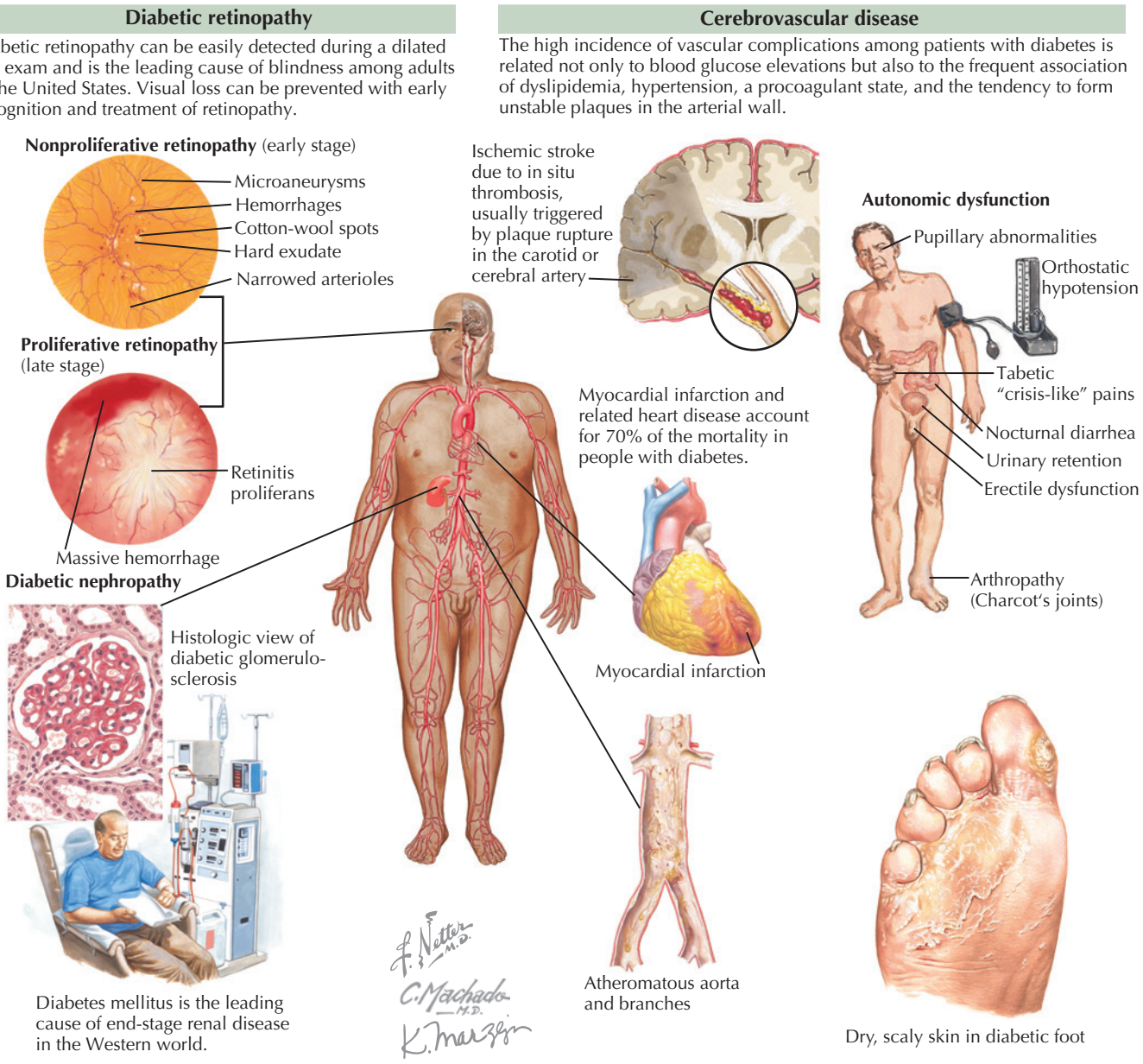

Figure 28-2 Signs of Neuropathic Joint Disease and Peripheral Vascular Disease.

- Patients with macular edema, nonproliferative diabetic retinopathy, and proliferative diabetic retinopathy should be evaluated by an ophthalmologist prior to exercise. Some patients with controlled disease states listed earlier may safely exercise.

EXERCISE GUIDELINES

- **Good blood glucose control should be established before starting an exercise program.**
- **Adjustment of diet and insulin when exercising needs to be *individualized*.**
- **Blood glucose should be measured before, during, and after exercise.** Allows patient and physician to study blood glucose response to various exercise conditions (e.g., consecutive hard days of training, tournaments, reduced training days before competition).
- **If blood glucose exceeds 250 to 300 mg/dL prior to exercise, blood glucose tends to rise, rather than fall, during exercise:**
  - **If ketosis exists before exercise, ketone production rises.**
  - These effects are due to influence of counter-regulatory hormones (catecholamines, cortisol, and growth hormone) and relative insulin deficiency.
  - Check urine for ketones if blood glucose exceeds 250 mg/dL. Do not exercise if ketones are present.
- **Pregame anxiety mimics hypoglycemia, leading many diabetic athletes to overeat before competition.**
  - They may treat what is perceived to be hypoglycemia.
  - They may reduce dosage of insulin or insulin secretagogues peaking during contest to avoid hypoglycemia and possibly become hyperglycemic.
  - These inappropriate steps may lead to accentuated ketoacidosis and poor performance because of limited use of muscle glycogen and reduced blood pH.
- **Endurance training**
  - Increases fat utilization in submaximal endurance exercise because of increase in mitochondrial density and associated enzymes.

- Increases muscle and liver glycogen stores, allowing athlete to be active for much longer time before needing supplemental carbohydrate.

- **Hypoglycemia is more likely to occur during exercise in the evening and least likely to occur in morning exercise because of diurnal variation in cortisol and growth hormone levels.**
  - If exercising in evening, amount of insulin taken that peaks after eating and during the rest of the evening should be reduced or more food should be consumed before and possibly after exercise.
  - Decrease rapid-acting and short-acting insulin pre-exercise as follows:
    - Exercise lasting less than 1 hour—30% reduction.
    - Exercise lasting 1 to 2 hours—40% reduction.
    - Exercise lasting 2 or more hours—50% reduction.
    - Further adjustments are based on historical glucose response to exercise.
  - Emphasizing the use of rapid-acting insulin analogs before eating (insulin lispro, insulin aspart, insulin glulisine) and basal long-acting insulin analogs (insulin glargine, insulin detemir) decreases the incidence of hypoglycemia because of consistent absorption and more predictable pharmacodynamics.
  - Before actual competition in evening, alteration in insulin or food intake should be experimented with several times to mimic conditions (e.g., same time and similar energy expenditure) during evening competition.
  - Because insulin sensitivity is affected for at least 4 hours and as long as 28 hours after exercise, nocturnal hypoglycemia may occur. Blood glucose may have to be assessed during nighttime to prevent hypoglycemia.
  - Increase caloric intake for 12 to 24 hours postexercise.
  - Decrease insulin pump basal rate during and after exercise.
  - Assess trends in blood glucose during training to determine individual response to exercise.
- If possible, avoid the use of short-acting (regular) insulin and intermediate-acting (NPH) insulin because of variable absorption from day to day in the same person. This variability predisposes to unpredictable hypoglycemia.
- The abdomen is the preferred site of insulin injection for athletes because absorption is most consistent and predictable.
- Physiologic factors that predispose to hypoglycemia:
  - Longer exercise time
  - Greater exercise intensity
  - Reduced fitness level
  - Higher insulin dose
  - Insulin injection into or over exercising muscle prior to exercise
  - Massage or heat application to area of insulin injection
- **Consistent daily energy expenditure facilitates blood glucose control.**
  - Extra medication or lower food intake may be required on days of reduced activity, whereas less medication or greater food intake may be needed on days of increased training duration or intensity.
  - **When exercise lasts for several hours** (e.g., triathlons, mountaineering, cycling, tournament play), **basal dose of insulin should be reduced in athletes with type 1 diabetes by as much as 50%.** Additional blood glucose monitoring during events has proved helpful in such sports.
- High-intensity exercise may acutely raise blood glucose rather than lower it secondary to catecholamine release. Several studies suggest an intensity level above lactate threshold (about 80% of $\dot{V}O_2$ max in athletes). Be cautious of "overcorrection" with insulin because insulin sensitivity may be elevated for many hours after vigorous work.
- **Medication requirements are reduced in early months of training** (25% to 40% reduction in dosage is typical) and remain lower as long as training occurs:
  - Athletes with type 1 diabetes require approximately 0.5 unit of insulin per kg body weight or less. This dosage is decreased with regular exercise.
  - **Athletes with type 2 diabetes require 10 to 15 units per day of supplemental insulin therapy and up to 1.0 to 1.2 units of replacement insulin per kg body weight per day, and this dosage decreases with training and exercise. Oral medication dosage also decreases and some patients may eventually require little or no oral hypoglycemic medication** because of the combined effect of fat loss and exercise on insulin sensitivity.
- Achievement of good blood glucose control facilitates development of muscle mass:
  - Adequate insulin enhances normal uptake and utilization of amino acids.
  - Inadequate insulin promotes protein degradation and water loss.
- **Good blood glucose control facilitates glycogen storage in skeletal muscle:**
  - This reduces the likelihood of glycogen depletion occurring after consecutive days of vigorous training as well as during a prolonged endurance event.
  - Evidence suggests that the amount of water stored with skeletal muscle glycogen has a strong effect on exercise capacity in the heat.
  - Many marginally controlled diabetic patients have particular difficulty exercising in warm environments, partly because of reduced muscle glycogen and water.

GUIDELINES FOR AVOIDING HYPOGLYCEMIA ASSOCIATED WITH EXERCISE

- Hypoglycemia is a problem diabetic patients must manage when exercising. They should measure blood glucose (BG) before exercise and ingest carbohydrate using the following criteria if the intensity is low to moderate. Realize that high-intensity exercise leads to increased BG level and so fewer carbohydrates, not more, are needed.
  - If BG is below 120 mg/dL, consume 15 grams of carbohydrate initially and then 30 grams of carbohydrate per 60 minutes of light to moderate exercise (<60% $\dot{V}O_2$ max).
  - If BG is 120 to 180 mg/dL, consume 30 grams of carbohydrate per 60 minutes of light to moderate exercise.
  - If BG is 180 to 250 mg/dL, take no food before exercise. If the exercise is heavy and duration exceeds 30 minutes, take second blood glucose reading and use criteria stated in first two items of this list.
  - If BG is 250 mg/dL or higher, check urine for ketones. Exercise is permissible if no ketones are present, but do not exercise if ketones are present or BG exceeds 300 mg/dL because action of counter-regulatory hormones may cause blood glucose to rise during exercise as well as increase ketone level.
  - These guidelines are only estimates that need to be modified for a given person.
- **Easily digested carbohydrates should be readily available for supplemental feeding** by athletic trainer or coach and on athlete's person (e.g., pocket of running shorts, pack on bicycle). Glucose gel or tablets elicit faster rise in blood glucose than fruit or fruit juices.
- **Decrease dosage of insulin that peaks during exercise.** Rapid-acting analogue insulin normally taken before meals might be decreased or deleted.

- Hypoglycemia occurs during and after exercise more frequently and severely in those who have had type 1 diabetes for 10 years or more. Patients in good glycemic control may suffer more episodes of hypoglycemia if there are no alterations in the management plan. Frequent exposure to hypoglycemia promotes hypoglycemic unawareness resulting from an attenuated epinephrine response secondary to reduced sympathoadrenal responses. The downregulation of the response system promotes a decreased response of counter-regulatory hormones, which predisposes to further recurrent hypoglycemia and requires medical consultation. Raising the mean glucose level for several weeks often resets this defense mechanism. Coaches and trainers need to know symptoms and steps for treatment. Team physicians should carry a glucagon kit to revive the unconscious patient.
- Avoid exercise in the evening or develop a plan to meet the reduced insulin needed if exercise cannot be avoided at this time of day.
- After expending an unusually large amount of energy or if exercise is done in the evening, expect possible nocturnal hypoglycemia and into the next day. Extra blood glucose monitoring is advisable and may be needed during nighttime.
- Choose injections of rapid-acting analogue insulin in the abdomen for consistent absorption and glycemic effect.
- The athlete must adequately replace glycogen stores postexercise ("golden replenishment period"). Endurance athletes often need 1.5 g of carbohydrate per kg of body weight and glycogen-depleting activity may require up to 550 g of carbohydrate for repletion. Insufficient replacement of glycogen stores is the primary cause of delayed hypoglycemia.

Continuous Subcutaneous Insulin Infusion

Continuous subcutaneous insulin infusion (CSII) and continuous glucose monitoring systems can be useful for selected endurance athletes.

General Concepts

- Continuous subcutaneous insulin infusion (CSII) is a device that contains a reservoir of rapid-acting insulin, a screw-drive pump, a non-waterproof computer, and a catheter connected to a quick-release device inserted into the subcutaneous tissue (usually in the abdomen).
- CSII is used in the management of both type 1 and type 2 diabetes mellitus.
- Basal insulin is delivered by the continuous infusion of rapid-acting insulin while a superimposed bolus of the same insulin is administered over a few minutes.
- CSII allows minute-to-minute adjustments of insulin infusion, which is an advantage to the athlete.

Indications for Temporary Interruption (<60 minutes) of CSII

- Showering
- Sex
- Specific sports:
  - SCUBA, swimming, sailing
  - Contact sports—rugby, football, wrestling

To Prevent Hypoglycemia

- Suspend insulin pump infusion for 1 hour or less
  - Administer an insulin bolus (usually <50% of the expected insulin infusion for the exercise time period)
  - Then, abruptly suspend (cease infusion) the pump, ***or***
- Maintain, but lower, basal rate infusion
  - Modify basal rate initially by 50% (when exercising prebreakfast or >4 hours postmeal (e.g., if basal rate is 1.4 units per hour prebreakfast and the athlete exercises during this period, the rate is reduced to 0.7 units per hour).
  - Further modifications are made based on self-monitoring blood glucose (SMBG).
  - Following exercise, decrease the basal rate for 30 to 60 minutes because of improved insulin sensitivity.
  - Reduce the subsequent prandial bolus by 30% to 50%.
- Prolonged endurance exercise (e.g., walking, running distance, cycling, hiking)
  - Athlete develops a metabolic "steady-state."
  - A low-dose temporary basal rate is set (0.1 to 0.2 units per hour) and the ingested calories are metabolized by the activity each hour. Often, the glucose ranges from 100 mg/dL to 140 mg/dL over hours of continuous endurance exercise.
  - Most athletes require 15 to 30 g of carbohydrate each 30 minutes of exercise but this is individualized.
- Weight gain is rarely seen with athletes using CSII because the athlete can adjust the basal infusion or "suspend" (discontinue insulin infusion) instead of eating additional carbohydrates.
- The infusion catheter must be securely anchored to the skin with specific adhesive devices because the combination of exercise and perspiration may dislodge the catheter. With no background long-acting insulin, a potential problem is diabetic ketoacidosis, which can ensue within 3 to 4 hours.

HIGH ALTITUDE

- Glycemic control is decreased in patients with type 1 diabetes at high altitudes.
  - Increased sympathetic tone leads to increased hepatic glucose production
  - Increased insulin resistance
  - Loss of appetite at high altitude
- There can be problems with glucose monitor reliability at high altitude.
- Insulin needs to be adequately protected from extremes in temperature (inactivated by freezing).

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The Athlete's Heart and Sudden Cardiac Death

29

Kimberly G. Harmon and Jonathan A. Drezner

THE ATHLETE'S HEART

Definition

Intense regular physical exercise can induce physiologic and morphologic cardiac changes known as athlete's heart. Theses adaptations are considered a normal response to repetitive exercise training.

Increased Vagal Tone

- As stroke volume increases in response to regular exercise training, the resting heart rate decreases without a change in overall cardiac output (pulse × stroke volume = cardiac output). A lower heart rate is regulated by increased parasympathetic input and may cause some resting electrocardiographic (ECG) changes (see following text) that should be considered normal in a well-trained athlete.

Morphologic Changes

- The myocardial adaptations that occur in athlete's heart depend on the frequency, duration, and intensity of physical conditioning. Morphologic cardiac adaptations include left ventricular (LV) enlargement, increases in LV wall thickness, and increases in LV mass. Myocardial changes occur according to Laplace's law: wall stress = (pressure × radius) / (wall thickness × 2).
- Isotonic (dynamic) exercise such as running, cycling, or swimming presents a volume load to the heart. Isotonic exercise increases venous return and thus LV end-diastolic diameter, allowing for a larger stroke volume and cardiac output. In response to a chronic volume load, the LV wall thickens proportionately in order to normalize wall stress. Thus, the myocardium hypertrophies in an eccentric fashion and the mass-to-volume ratio remains unchanged.
- Isometric (static) exercise, such as weightlifting or shot-putting, places a pressure load to the heart by brief increases in systemic blood pressure during training. In response to chronic pressure demands, the myocardium hypertrophies in a concentric fashion and the mass-to-volume ratio increases.
- Most sports involve a combination of both isotonic and isometric exercise so the resulting structural changes are typically a blend of eccentric and concentric hypertrophy. The result is an overall increase in cardiac mass resulting from an increase in LV diastolic cavity dimension, wall thickness, or both.

Pathologic vs. Physiologic Hypertrophy

- Physiologic changes that occur in response to training can be difficult to differentiate from pathologic processes such as hypertrophic cardiomyopathy (HCM) (Table 29-1).
- In adults, LV wall thickness less than 12 mm is generally considered normal, and LV wall thickness greater than 16 mm is suggestive of pathologic hypertrophy. Wall thickness in the 13 to 15 mm range falls in a morphologic "gray zone" and can represent either pathologic or physiologic hypertrophy. In these cases, looking at other functional cardiac parameters such as LV filling and end-diastolic diameter is helpful. HCM produces a stiff restrictive myocardium with impaired diastolic filling, reduced LV end-diastolic cavity dimension (<45 mm), and left atrial enlargement. In contrast, athlete's heart maintains normal LV filling and larger end-diastolic cavity dimension (>55 mm).
- If the distinction between pathologic and physiologic hypertrophy cannot be established, a period of deconditioning should be considered. Reduced LV wall thickness with deconditioning (3 to 6 weeks of no exercise) assessed with serial echocardiography or cardiac magnetic resonance imaging (MRI) suggests the increases in wall thickness are related to regular physical training and is supportive of athlete's heart. A decrease in wall thickness would not be expected in patients with HCM.

Electrocardiogram (ECG) Changes

- The increased parasympathetic (vagal) tone and increase in cardiac mass from physiologic hypertrophy seen in athlete's heart are associated with several ECG changes that should be considered normal in a well-conditioned athlete. Table 29-2 lists the common ECG changes seen in athlete's heart as well as abnormal ECG changes that suggest underlying pathology.
- Changes related to increased vagal tone, such as resting sinus bradycardia, sinus arrhythmia, and first-degree atrioventricular block, should readily reverse with exercise.

Participation Recommendations

- Athlete's heart consists of normal physiologic adaptations to regular intense exercise and should be expected in well-conditioned athletes. These changes provide a functional advantage for the athlete, result in no adverse consequences, and resolve with the cessation of regular exercise. No treatment and no limits on sports participation are required.

SUDDEN CARDIAC DEATH

Epidemiology

- Sudden cardiac death (SCD) is the leading cause of death in young athletes.
- The true incidence of SCD in athletes is difficult to estimate because of the lack of a mandatory national reporting system.
- Traditional estimates of SCD in high school and college-aged athletes are about 1 in 200,000 per year. However, recent studies show that past estimates are low and likely underrepresent the extent of the problem. The Sudden Death in

Table 29-1 DISTINGUISHING HCM FROM ATHLETE'S HEART

HCM	Athlete's heart
Unusual pattern of LVH, may be heterogeneous	Symmetric LVH or uniform distribution of hypertrophy
LV cavity < 45 mm (small)	LV cavity > 55 mm (not small)
Left atrial enlargement	No left atrial enlargement
Abnormal LV filling	Normal LV filling
EKG abnormalities (see Table 29-2)	EKG with high voltage, but no Q wave changes (see Table 29-2)
Thickness does not decrease with deconditioning	LVH decreases with deconditioning
+ Family history of HCM	No family history of HCM
+ Genetic testing for HCM	Negative genetic testing for HCM

HCM, hypertrophic cardiomyopathy; LVH, left ventricular hypertrophy.

Table 29-2 ELECTROCARDIOGRAM (ECG) INTERPRETATION IN ATHLETES AGES 12-35

ABNORMAL
Findings strongly suggest underlying cardiovascular disease.
ST segment depression in at least 2 leads
Deep negative T waves (≥0.2 mV) in at least 2 leads
Romhilt-Estes voltage criterion for LVH (R or S wave ≥2.0 mV in any limb lead, or S wave in V1 or V2 ≥3.0 mV or R wave in V5 or V6 ≥3.0 mV) *with* ST segment depression or T wave inversion
Pathologic Q wave patterns (≥0.04 sec in duration or depth >25% of the height of the ensuing R wave or QS pattern in ≥2 leads)
Complete right or left bundle branch block (QRS duration ≥0.12 sec)
R or R′ wave in V1 ≥0.5 mV in amplitude and R:S ratio ≥1
ST segment elevation in V1 to V3 with the right bundle branch block pattern (Brugada-like pattern)
Prolonged QT_c interval (≥0.48 sec)
Short QT_c interval (≤0.33 sec)
Ventricular pre-excitation (short PR <0.12 sec) with or without delta wave
Epsilon wave (small negative deflection or terminal notch just beyond the QRS in V1 or V2, found in ARVC)
Left atrial enlargement (negative portion of P wave in V1 ≥0.1 mV in depth and ≥0.04 sec in duration)
Right atrial enlargement (P wave ≥0.25 mV in leads II and III or V1)
Left axis deviation (−30 degrees to −90 degrees)
Right axis deviation (≥120 degrees)
Third-degree atrioventricular block
Second-degree Mobitz II atrioventricular block
Complex ventricular arrhythmias
Atrial tachydysrhythmias (supraventricular tachycardia, atrial flutter, atrial fibrillation)
POSSIBLY ABNORMAL
Findings may suggest underlying cardiovascular disease.
Romhilt-Estes voltage criterion for LVH (R or S wave ≥2.0 mV in any limb lead, or S wave in V1 or V2 ≥3.0 mV or R wave in V5 or V6 ≥3.0 mV) *without* ST segment depression or T wave inversion
QT_c interval in men greater than 0.44 sec and greater than 0.46 sec in women
Second-degree Mobitz I (Wenckebach) atrioventricular block
QRS duration of at least 0.11 sec and less than 0.12 sec
Left anterior fascicular block
Ventricular premature contractions (≥3 PVCs per tracing)
PROBABLY NORMAL
Findings common in well-conditioned athletes.
Sinus bradycardia (<60 beats/min)*
Sinus arrhythmia*
Prolonged PR interval or first-degree atrioventricular block*
Junctional rhythm*
Early repolarization pattern (J-point ST segment elevation)*
Tall T waves
Incomplete right bundle branch block (RSR' pattern in precordial leads with QRS duration <0.11 sec)
Isolated Sokolow voltage criterion for LVH (S wave in V1 + R wave in V5 or V6 ≥3.5 mV) *without* ST depression or T wave inversion

*Changes normalize with exercise.
LVH, left ventricular hypertrophy; ARVC, arrhythmogenic right ventricu-

Young Athletes Registry (a nonmandatory surveillance system) has documented about 120 deaths per year in young competitive athletes in the United States, or approximately 1 in 50,000 per year, or one death every 3 days.

- SCD in athletes occurs more commonly in males, with a male-to-female ratio ranging from 5:1 to 9:1.
- Sports with the highest incidence of SCD are football and basketball, although SCD can occur in any sport.
- A disproportionate amount (40%) of SCD occurs in African-American athletes, above the proportion of African-Americans in athletics.

Presentation

- Healthy appearing athletes may harbor unsuspected cardiovascular disease. In 50% to 80% of SCD, the athlete is previously asymptomatic and SCD is the first presenting event of their cardiovascular disorder.
- Warning symptoms of underlying cardiovascular disease include a history of exertional chest pain, exertional syncope or presyncope, dyspnea or fatigue disproportionate to the degree of exertion, and palpitations or irregular heart beats. Athletes with these symptoms require a careful and comprehensive cardiac workup before returning to exercise.
- A family history of sudden unexplained death or SCD before age 50, or a history of familial cardiac disorders known to cause SCD in young athletes, also warrant further diagnostic investigation before participation in athletics.

Causes

SCD in young athletes is caused by a variety of congenital structural cardiac abnormalities, primary electrical diseases, and acquired or traumatic heart disorders.

Structural

HYPERTROPHIC CARDIOMYOPATHY

Epidemiology: HCM is the most common cause of SCD in young athletes, accounting for 25% to 36% of cases. Its prevalence is 1 per 500 in the general United States population as recognized by echocardiography.

Pathologic features: Characteristic morphologic features of HCM include asymmetric LV hypertrophy (usually involving the ventricular septum), LV wall thickness of 16 mm or more (normal ≤12 mm; borderline 13 to 15 mm), a ratio between the septum and free wall of more than 1.3, and a nondilated LV. Histologic analysis shows disorganized cellular architecture. Intramural tunneling (myocardial bridging), in which a segment of coronary artery is completely surrounded by myocardium, is present in about one third of cases (Fig. 29-1).

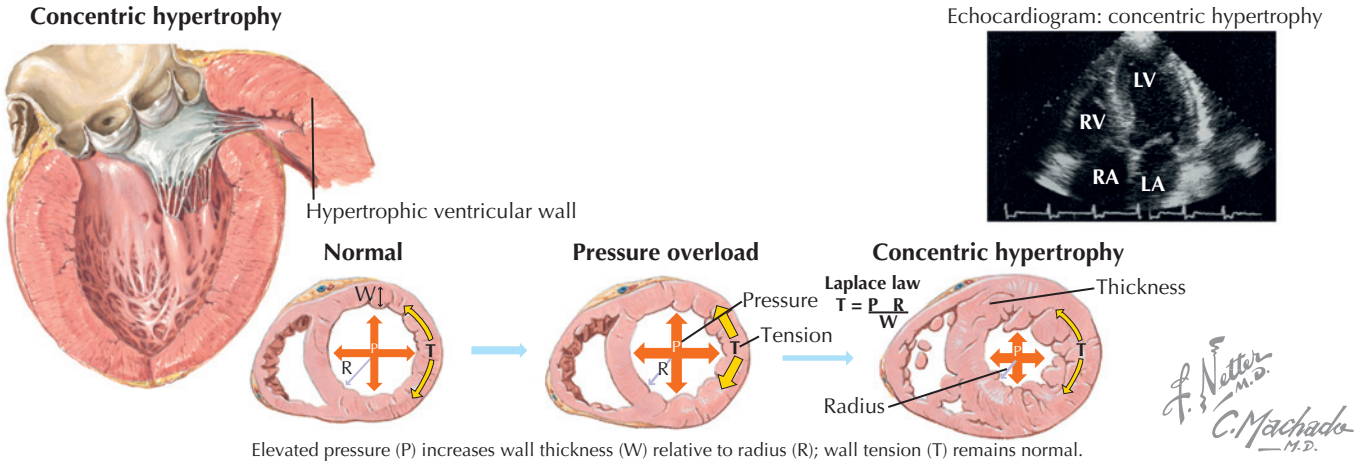
Development: Morphologic features may appear in childhood, but usually develop in early adolescence or young adulthood.

Genetics: Eleven mutated sarcomeric genes have been associated with HCM. The most common mutations are associated with beta-myosin heavy chain and myosin-binding-protein C. The 10 most common HCM-causing mutant genes have been identified and can be tested for in commercial laboratories (see www.hpcgg.org/LMM/tests.html); however, there are many false negatives with genetic testing. HCM is autosomal dominant in 55%, and sporadic in 45%.

Symptoms: Most athletes with HCM remain asymptomatic until the time of death. In one study, only 21% of athletes who died had any signs or symptoms of cardiovascular disease prior to their death. Symptoms may include exertional chest pain, dyspnea, lightheadedness, or syncope.

Physical exam findings: HCM should be suspected in any athlete in whom a harsh systolic ejection murmur is heard on auscultation. The characteristic murmur of HCM increases with any maneuver that decreases venous return (i.e., Valsalva, or moving from squatting to standing) and diminishes with maneuvers that increase venous return (i.e., lying supine, or moving from standing to a squatting). A fourth heart sound may also be present.

Diagnostic tests: ECG is abnormal in up to 95% of athletes with HCM. Specific ECG abnormalities suggestive of HCM include prominent Q waves, deep negative T waves, or dramatic increases in QRS voltage (often associated with ST depression or T wave inversion). Echocardiography remains the standard to confirm the diagnosis of HCM by identifying pathologic LV



Elevated pressure (P) increases wall thickness (W) relative to radius (R); wall tension (T) remains normal.

Figure 29-1 Left Ventricular Hypertrophy.

wall thickness (≥16 mm) and LV diastolic dysfunction. MRI has additional value in identifying segmental hypertrophy in the anterolateral LV free wall or at the apex, and can help differentiate HCM from athlete's heart with serial MRIs after an interval of deconditioning.

Return to play: The 36th Bethesda Conference recommends that athletes with an unequivocal diagnosis of HCM not participate in sports with the possible exception of class IA (low-static, low-dynamic) sports. In athletes older than 30 years, without high risk features (no ventricular tachycardia on ambulatory ECG, no family history of sudden death due to HCM, no syncope), the decision is left to individual judgment. Athletes who are genetically positive (but are phenotype negative) for HCM but also have cardiac symptoms or a family history of SCD should be excluded from participation. If no such history exists, there is no evidence to support exclusion from participation in genetically positive (phenotype negative) individuals.

LEFT VENTRICULAR NONCOMPACTION

Epidemiology: Left ventricular noncompaction (LVNC) is a newly recognized disorder with an unknown incidence but being identified more frequently with higher quality echocardiograms.

Pathologic features: LVNC is characterized by prominent trabeculations and deep intertrabecular recesses in the LV endocardium in association with LV hypertrophy, dilation, or hypertrophy/dilation. Because of thickened myocardium, coronary vessels have little reserve capacity.

Genetics: LVNC can be familial or sporadic. Familial recurrence is high and found in approximately 40% of patients. Some gene mutations have been identified.

Symptoms: Patients may be asymptomatic or present with heart failure, atrial and ventricular arrhythmias, syncope, sudden cardiac arrest (SCA)/SCD, or thromboembolic events.

Physical exam findings: Patients may have a normal cardiac exam, or present with heart failure with third or fourth hearts sounds, edema, and pulmonary congestion.

Diagnostic tests: ECG is often abnormal but no characteristic findings. May present as right bundle branch block, fascicular block, atrial fibrillation, or ventricular tachycardia. Echocardiographic features include:

- Segmental thickening of the left ventricular myocardial wall consisting of two layers: a thin compacted epicardial layer and an extremely thickened endocardial layer with prominent trabeculations and deep recesses. A 2:1 or greater ratio of noncompacted to compacted myocardium at end-systole with thickening of the myocardial wall is considered to be diagnostic of LVNC with high specificity and sensitivity.
- Predominant localization of the pathology in the apical midlateral and midinferior regions of the left ventricle. Most of the noncompacted segments are hypokinetic.
- Color Doppler evidence of flow within the deep intertrabecular recesses.
- Absences of coexisting cardiac abnormalities.

Return to play: Little information on this disease, particularly in asymptomatic athletes, is available. The 36th Bethesda Conference recommends restriction from competition.

CONGENITAL CORONARY ANOMALIES

Epidemiology: Congenital coronary anomalies (CCA) account for approximately 19% of cases of SCD.

Pathologic features: The most common CCA is an abnormal origin of the left coronary artery arising from the right sinus of Valsalva. Other features that may contribute to ischemia during exercise include an acute angled take-off, hypoplastic ostium, or impingement of the anomalous artery as it traverses between the expanding great vessels during exercise. In 3% of cases of sudden death the only anomaly found is a tunneled left coronary artery; however, this finding is present in 2% of the general population without resulting in sudden death (Fig. 29-2).

Genetics: No familial predisposition.

Symptoms: In one study, 12 of 27 athletes who died of CCA had prodromal symptoms such as exertional syncope, chest pain, or palpitations.

Physical exam findings: None.

Diagnostic tests: ECG is typically normal but may show Q waves suggesting prior infarction. Advanced cardiac imaging such as computed tomography (CT) angiography or cardiac MRI is usually needed to identify anomalous origin coronary abnormalities, because transthoracic echocardiography has limited sensitivity. Definitive diagnosis is made with coronary angiography.

Return to play: Detection should result in exclusion from all competitive sports. However, participation may be considered 6 months after correction of the defect.

MYOCARDITIS

Epidemiology: Acute inflammatory process involving the myocardium. Coxsackie B virus is implicated in more than 50% of cases, but echovirus, adenovirus, influenza, and *Chlamydia pneumoniae* have also been associated with myocarditis.

Pathologic features: Lymphocytic infiltrate of myocardium with necrosis or degeneration of adjacent myocytes. Acute phase presents with flulike illness, which may lead to dilated cardiomyopathy. SCD may occur in the presence of either active or healed myocarditis (Fig. 29-3).

Genetics: No familial predisposition.

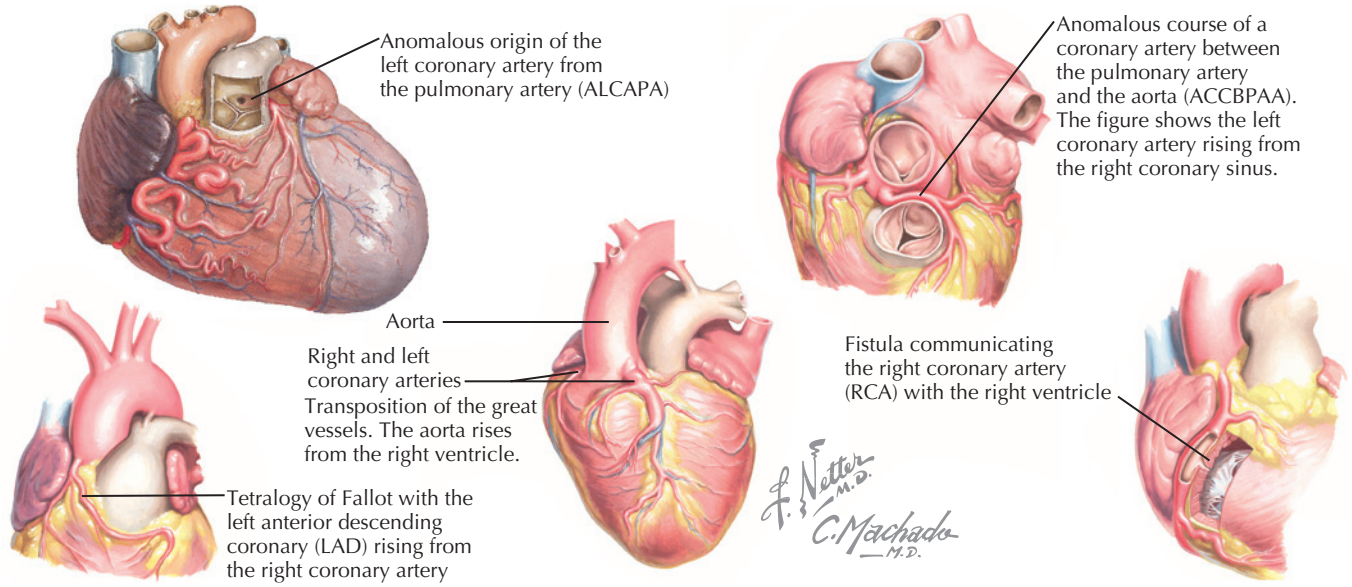


Figure 29-2 Congenital Coronary Artery Anomalies.

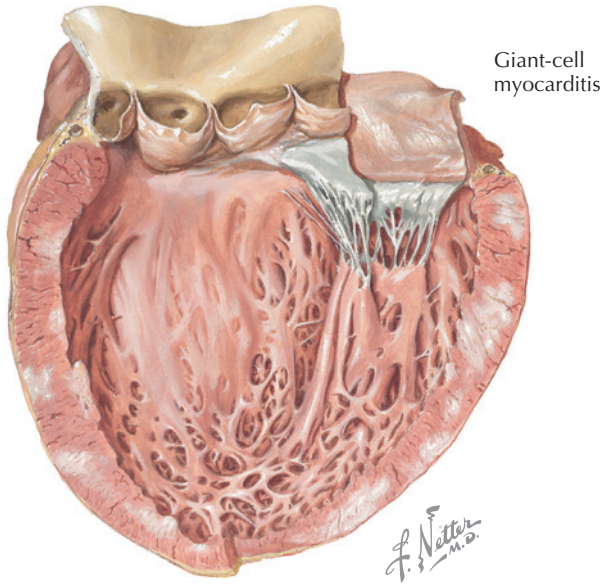


Figure 29-3 Idiopathic Myocarditis.

Symptoms: Characteristic symptoms include a prodromal viral illness followed by progressive exercise intolerance and congestive symptoms of dyspnea, cough, and orthopnea.
Physical exam findings: S3 gallop; signs of heart failure (edema, pulmonary rales).
Diagnostic tests: ECG may show diffuse low voltage, ST and T wave changes, heart block, or ventricular arrhythmias. Laboratory tests include leukocytosis, eosinophilia, elevated sedimentation rate or C-reactive protein, and increased myocardial enzymes. Echocardiography may show dilated LV, global hypokinesis, segmental wall abnormalities, and decreased LV ejection fraction.
Return to play: May return to competitive sports after 6 months with normal ventricular function (on stress echocardiogram), normal cardiac dimensions, and absence of clinically relevant arrhythmias on ambulatory monitoring and stress testing.

ARRHYTHMOGENIC RIGHT VENTRICULAR CARDIOMYOPATHY (ARVC)

Epidemiology: Arrhythmogenic right ventricular cardiomyopathy (ARVC) accounts for 4% of SCD in the United States (but 22% of SCD in the Veneto region of northeastern Italy). Estimated prevalence is 1 case per 5000 population.
Pathologic features: Characterized by myocyte death with replacement by fibrous and/or adipose tissue in the right ventricle. May be segmental or diffusely involve the right ventricle and predisposes the athlete to ventricular tachyarrhythmias.
Genetics: Results from mutations of genes encoding for desmosomal (cell adhesion) proteins. There are many different mutations leading to defective proteins of the desmosomal complex. Only one third of family members with positive genetic tests will fulfill diagnostic criteria for disease.
Symptoms: Of athletes who died from ARVC, 68% had prodromal symptoms such as syncope, chest pain, or palpitations.
Physical exam findings: Normal.
Diagnostic tests: ECG may show right precordial T wave inversion (beyond V1), an epsilon wave (small terminal notch seen just beyond the QRS in V1 or V2), prolongation of QRS duration to more than 110 msec, or right bundle branch block pattern. Echocardiogram, cardiac MRI, or CT may demonstrate right ventricular dilatation and wall thinning, reduced right ventricular ejection fraction, or right ventricular aneurysms. Fibro-fatty infiltration is best seen on cardiac MRI.
Return to play: No competitive athletics.

MITRAL VALVE PROLAPSE (MVP)

Epidemiology: Mitral valve prolapse (MVP) is reported in 5% of the general population; however, studies using more strict criteria for diagnosis suggest it occurs in only 0.5% to 1.3%. MVP accounts for less than 2% of SCD in athletes. MVP can be primary (present in the absence of any recognizable disorder), or secondary related to connective tissue disorders (Marfan syndrome, Ehlers-Danlos syndrome), HCM or dilated cardiomyopathy, or papillary muscle dysfunction after myocardial infarction.
Pathologic features: Characterized by systolic protrusion of the mitral valve leaflet into the left atrium, mitral valve thickening, elongation, and myxomatous degeneration. May lead to mitral regurgitation.

Genetics: Primary mitral valve prolapse can be inherited as an autosomal dominant condition with incomplete penetrance. The prevalence of the disease in first-degree relatives ranges from 30% to 50%.
Symptoms: No reliable indicators of MVP.
Physical exam findings: Midsystolic click and possible late systolic murmur if mitral regurgitation is present.
Diagnostic tests: Echocardiography is diagnostic.
Return to play: An athlete with MVP can participate in all sports unless there is a history of syncope with documented arrhythmia, family history of sudden death, supraventricular tachycardia or complex ventricular arrhythmias, moderate to marked mitral regurgitation, or prior embolic event, in which cases they should be restricted to class IA sports.
2007 American Heart Association guidelines no longer recommend prophylactic antibiotics for mitral valve prolapse.

DILATED CARDIOMYOPATHY (DCM)

Epidemiology: Estimated prevalence of 1 case per 2500 population. Dilated cardiomyopathy (DCM) is a rare cause of SCD in athletes, accounting for 2% of cases.
Pathologic features: Characterized by LV dilatation and systolic dysfunction with normal LV wall thickness. Secondary DCM results from prolonged untreated systemic hypertension, ischemic heart disease, viral myocarditis, infiltrative diseases (sarcoidosis, amyloidosis, hemochromatosis), autoimmune illnesses, or toxins (ethanol).
Genetics: Of primary DCM cases, 20% to 35% have been reported as familial, with incomplete and age-dependent penetrance.
Symptoms: Progressive exertional intolerance, dyspnea, orthopnea, fatigue, and edema.
Physical exam findings: S3 or S4 may occur in DCM; holosystolic murmur to suggest mitral regurgitation.
Diagnostic tests: ECG and Holter monitoring may show supraventricular and ventricular tachyarrhythmias, as well as major conduction delays (bundle branch block and atrioventricular block). Echocardiogram is diagnostic demonstrating a dilated LV with reduced ejection fraction.
Return to play: Excluded from all competitive sports regardless of symptomatology, with the possible exception of low-intensity sports.

AORTIC RUPTURE/MARFAN SYNDROME

Epidemiology: Reported incidence of aortic rupture/Marfan Syndrome is 1 in 10,000 to 20,000 individuals.
Pathologic features: Progressive dilatation and weakness (cystic medial necrosis) of proximal aorta and myxomatous degeneration of mitral and aortic valves leading to valvular dysfunction.
Genetics: Mutations in the FBN-1 gene encoding the glycoprotein fibrillin-1; 85% autosomal-dominant inheritance with variable expression.
Symptoms: Symptoms related to aortic root dissection include chest and thoracic pain. Heart failure may also occur secondary to aortic valve incompetence.
Physical exam findings: Highly variable clinical features usually manifested in adolescence and young adulthood. The diagnosis of Marfan syndrome is based on the Ghent nosology. These criteria rely on the recognition of both "major" and "minor" clinical manifestations involving the skeletal, cardiovascular, and ocular systems, and the dura (Table 29-3). Major criteria include the following: four of eight typical skeletal manifestations (pectus carinatum or excavatum, arm span to height ratio > 1.05, scoliosis > 20 degrees, reduced elbow extension < 170 degrees, pes planus, and protrusio acetabulae by radiograph); ectopia lentis (lens dislocation detected by slit lamp examination); aortic root dilatation or dissection; and lumbosacral dural ectasia (detected by CT or MRI). Minor criteria include joint hypermobility, high arched palate, and typical facial appearance (dolichocephaly, malar hypoplasia, enophthalmos, retrognathia, and down-slating palpebral fissures).

Table 29-3 MAJOR AND MINOR CRITERIA FOR THE DIAGNOSIS OF MARFAN SYNDROME

Major	Minor
Skeletal	
Need 4	Pectus excavatum (moderate)
Reduced upper-to-lower segment ratio (0.85)	Joint hypermobility
Arm span exceeding height	High arched palate
Arachnodactyly of fingers and toes	
Scoliosis > 20 degrees or spondylolisthesis	
Pectus carinatum	
Pectus excavatum	
Reduced extension of elbows	
Pes planus	
Cardiovascular	
Need 1	
Dilatation of the ascending aorta involving the sinuses of Valsalva, with or without aortic regurgitation	Mitral valve prolapse Mitral regurgitation Dilatation of the pulmonary artery
Dissection of the ascending aorta	Calcification of the mitral annulus
Ocular	
Ectopic lens	Flat cornea Increased axial globe length Hypoplastic iris Myopia Retinal detachment
Other findings	
Dural ectasia affecting the lumbosacral spinal canal	Spontaneous pneumothorax Apical blebs Stretch marks

Diagnostic tests: ECG may show changes secondary to chronic aortic regurgitation including LV and left atrial hypertrophy. Echocardiogram is diagnostic for aortic root dilatation and aortic regurgitation. Slit light ophthalmologic exam for ectopia lentis and lumbosacral MRI or CT for dural ectasia can also be diagnostic.
Return to play: If the diagnosis of Marfan syndrome is made, only IA sports are recommended (billiards, bowling, cricket, curling, golf, and riflery) if aortic root diameter is greater than 40 mm and there is family history of sudden death or there is moderate or severe mitral regurgitation. If these conditions are not present, IIA sports (archery, auto racing, motorcycling, diving, equestrian) may also be considered with serial echocardiograms to look at the aortic root every 6 months. No contact sports should be allowed.

AORTIC STENOSIS (AS)

Epidemiology: Bicuspid aortic valve, which is associated with AS, occurs in about 0.5% of school-aged children.
Pathologic features: Congenital narrowing of the aortic valve that causes a pressure gradient (mild <20 mm Hg; moderate 21 to 49 mm Hg; severe >50 mm Hg). In many cases cardiac output and blood pressure remain normal because LV systolic pressure and cardiac work are elevated. This leads to LV hypertrophy without a compensatory increase in coronary blood supply. Ischemia can develop secondary to increased LV cardiac mass, coupled with poor coronary artery supply and decreased diastolic filling time (because of prolonged systolic phase). This is worsened with physical exertion.

Genetics: Patients with congenital AS are slightly more likely to have offspring with congenital heart disease.
Symptoms: Usually asymptomatic; less than 5% develop chest pain, angina, or syncope.
Physical exam findings: Systolic ejection murmur at upper right sternal border and apical ejection click. Murmur diminishes with maneuvers that decrease venous return (i.e., Valsalva).
Diagnostic tests: ECG may show LV hypertrophy and ST and T wave changes of LV strain. Echocardiography shows narrowing of the aortic valve with estimated pressure gradient and LV hypertrophy. Exercise stress testing can show evidence of ischemia, arrhythmia, and BP response.
Return to play: Dependent on severity of stenosis and presence or absence of symptoms. Athletes with mild AS can participate in all competitive sports, but should undergo serial evaluations of AS severity on at least an annual basis. Patients with severe AS or symptomatic patients with moderate AS should not engage in any competitive sports.

CORONARY ARTERY DISEASE

Epidemiology: Atherosclerotic coronary artery disease is the most frequent cause of SCD in athletes over the age of 30. The incidence of exercise-related sudden death in adults is 1 in 15,000 to 18,000.
Pathologic features: Most often caused by atherosclerotic plaque disruption. Exercise may be a stimulus for plaque disruption. Development is progressive and related to coronary risk factors (hypertension, diabetes, dyslipidemia, tobacco use, illicit drug use, and a family history of premature atherosclerotic disease).
Symptoms: Exertional chest pain, angina, lightheadedness, palpitations, or SCD.
Physical exam findings: No specific cardiac findings.
Diagnostic tests: ECG may show evidence of prior ischemia. Exercise stress testing may show ST segment depression or U wave inversion. Cardiac CT, MRI, or angiography will show extent of coronary artery narrowing.
Return to play: Dependent on extent of disease, symptoms, and stability after medical management, percutaneous coronary interventions, or surgery.

Primary Electrical Disease

Channelopathies: Diseases predisposing to potentially lethal ventricular tachyarrhythmias that are characterized by mutations in ion channel proteins leading to dysfunctional sodium, potassium, calcium, and other ion transport across cell membranes. Confirmed channelopathies account for approximately 3% of SCD in athletes. However, nearly 30% of SCDs in young people are autopsy-negative (sudden unexplained death) and most likely secondary to cardiac channelopathies based on postmortem molecular diagnosis.

LONG QT SYNDROME (LQTS)

Epidemiology: Long QT syndrome (LQTS) is the most common ion channelopathy.
Pathologic features: Characterized by prolongation of ventricular repolarization and QT interval corrected for heart rate (QT_c). There are six defined types, with three of them (LQTS-1, LQTS-2, and LQTS-3) occurring more frequently.
Genetics: There are 10 different genes on which hundreds of mutations have been identified. Defects in different ion channels can result in the same clinical syndrome.
Symptoms: Syncope or presyncope related to physical exertion or emotional stress. Family history of sudden unexplained death, unexplained drowning or motor vehicle accident, or sudden infant death.
Diagnostic tests: ECG features: QT_c greater than 460 msec for women and greater than 440 msec for men. Of people with LQTS, 25% will have normal QT_c. Syncope is often due to Torsades de pointes, a specific form of polymorphic ventricular tachycardia.
Common subtypes and features:
- **LQTS-1:** Most common. Arrhythmias are often related to emotional or physical stress, swimming, and diving.
- **LQTS-2:** Arrhythmias are often related to emotional or physical stress or sudden loud noises (alarm clocks).
- **LQTS-3:** Arrhythmias often related to rest or sleep and may account for 5% to 10% of sudden infant deaths.

Return to play and treatment: No competitive athletics and an automated internal cardiac defibrillator (AICD) are recommended in symptomatic patients. Beta-blockers reduce mortality in LQTS-1 and LQTS-2. For asymptomatic individuals, risk stratification is difficult.

SHORT QT SYNDROME (SQTS)

Epidemiology: Short QT syndrome (SQTS) has only recently been described (in 2000).
Pathologic features: Hyperfunctioning of potassium channel.
Genetics: Familial, though sporadic cases do occur. Three genes have been identified that are associated with SQTS
Symptoms: Palpitations, syncope, atrial fibrillation, SCA/SCD.
Diagnostic tests: ECG changes include a QT_c interval less than 340 msec and tall peaked T waves.
Return to play and treatment: No competitive athletics. Therapy is recommended because of high association with SCA. Use of quinidine, which lengthens QT interval, is investigational and recommended for patients who cannot use an AICD or who have received multiple shocks.

BRUGADA SYNDROME

Epidemiology: Brugada syndrome was first described in 1992. It is a rare channelopathy that is more prevalent in males from Southeast Asia.
Pathologic features: Abnormalities of sodium channels.
Genetics: Familial.
Symptoms: Syncope, SCA/SCD, and sudden death while sleeping.
Diagnostic tests: ECG shows a pattern of right bundle branch block and ST-segment elevation in leads V_1 to V_3. The ECG pattern may not be present unless "unmasked" with the administration of sodium channel blockers (flecainide, procainamide, etc.).
Return to play and treatment: Athletes with Brugada syndrome should be restricted from competition with placement of an AICD.

CATECHOLAMINERGIC POLYMORPHIC VENTRICULAR TACHYCARDIA

Epidemiology: Catecholaminergic Polymorphic ventricular tachycardia is a familial disorder characterized by stress-induced ventricular arrhythmias that result in SCD in children and young adults.
Pathologic features: Abnormalities in calcium channel function.
Genetics: Two major genetic types have been identified, with mutations on six different genes.
Symptoms: Syncope, sudden death, and polymorphic ventricular tachycardia triggered by vigorous physical exertion or acute emotion.
Diagnostic tests: Resting ECG is normal although some patients will have prominent U waves. Exertion or epinephrine challenge can induce ventricular tachycardia.
Return to play: No competitive athletics. AICD recommended. Beta-blocker or calcium-channel blocker may improve outcomes.

WOLFF-PARKINSON-WHITE SYNDROME (WPW)

Epidemiology: Around 1% of ECGs will show changes consistent with Wolff-Parkinson-White (WPW) syndrome; however, only a small subset will go on to develop the associated arrhythmias

that define the syndrome. Risk of sudden death in WPW is estimated at 1 per 1000 patient years.

Pathologic features: A tachyarrhythmia caused by an accessory pathway (the Bundle of Kent) which directly connects the atria and ventricles and bypasses the AV node. The arrhythmia can be atrioventricular tachycardia (80%), atrial fibrillation (15% to 20%), or atrial flutter (5%) (Fig. 29-4).

Genetics: Most cases are nonfamilial although there is one type of familial WPW associated with HCM.

Symptoms: Palpitations, syncope, near-syncope.

Diagnostic tests: ECG may show a short PR interval (<0.12 seconds), a delta wave (ventricular pre-excitation represented by a slurred upstroke of QRS complex), and a prolonged QRS (>0.12 seconds). Workup for asymptomatic patients is controversial, although for younger patients electrophysiologic testing should be considered.

Return to play and treatment: In athletes without palpitations or tachycardia who are over 25, return to play without treatment is recommended. For younger patients who are asymptomatic, electrophysiology testing should be considered. Symptomatic athletes with a ventricular rate higher than 240 should be considered for catheter ablation. Athletes who have had successful ablation, have normal AV conduction, and have no inducible arrhythmia on follow-up electrophysiology testing may participate in all competitive sports in several days. Those without an EP study and no spontaneous recurrence of arrhythmia in 2 to 4 weeks after ablation can return to all competitive sports.

Traumatic

COMMOTIO CORDIS

Epidemiology: Commotio cordis involves a direct nonpenetrating blow (blunt trauma) to the chest wall overlying the heart during a vulnerable phase of ventricular repolarization (just prior to peak of T wave), which leads to ventricular fibrillation. Accounts for approximately 20% of SCD in young athletes. Of commotio cordis cases, 96% are in males (mean age 14) with more than 80% under the age of 18. More than 80% of cases involve sports with a firm projectile such as baseball, softball, hockey, and lacrosse. No current chest protector effectively protects against commotio cordis, but the use of safety balls has shown a decrease in commotio cordis in pig models.

Pathologic features: Structurally normal heart.

Genetics: Predisposition not thought to be familial.

Symptoms: Instantaneous collapse following a blow to the chest. In up to half of cases, a brief period (<10 seconds) of consciousness occurs prior to the collapse.

Treatment and return to play: Treatment is expedient defibrillation. Survival is possible with early defibrillation with survival rates of 25% if the resuscitation is started within 3 minutes from collapse, and only a 3% survival if resuscitation is started later than 3 minutes after collapse.

PREVENTION OF SCD IN ATHLETES

Screening Preparticipation Physical Evaluation (PPE)

See Chapter 3.

- Primary objective is to identify athletes with silent cardiovascular abnormalities at risk for sudden death.
- Any PPE should (at a minimum) encompass recommendations for screening set forth by the American Heart Association (AHA). The exam should include a careful family history with special attention to early causes of death and known cardiac disease, questioning about symptoms known to be precursors of SCA (exertional chest pain, presyncope, syncope, excessive dyspnea, or palpitations), and careful physical exam by a provider skilled in cardiac auscultation and the recognition of Marfan physical stigmata.
- The Preparticipation Physical Evaluation Monograph, 3rd Edition (endorsed by the AAP, AAFP, ACSM, AMSSM, AOASM, and AOSSM) is a detailed resource.

AHA Recommendations 2007

- Personal medical history including a history of exertional chest pain or discomfort, unexplained syncope or near-syncope, excessive unexplained exertional dyspnea/fatigue associated with exercise, prior recognition of a heart murmur, elevated systemic blood pressure.
- A family history of premature death (sudden and unexpected, or otherwise) resulting from cardiac causes before age 50, disability from heart disease in a close relative before age 50, or specific knowledge of cardiac conditions in family members: hypertrophic or dilated cardiomyopathy, long QT syndrome

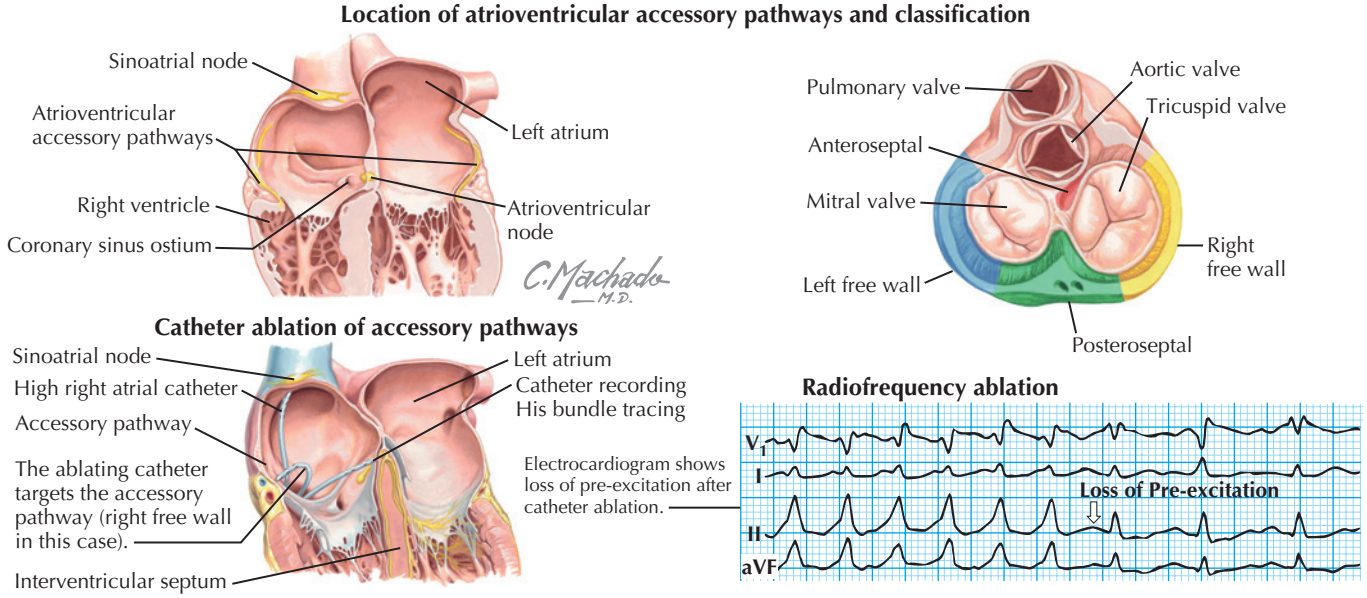


Figure 29-4 Accessory Pathways and the Wolff-Parkinson-White Syndrome.

or other ion channelopathies, Marfan syndrome, or clinically important arrhythmias.
- A physical exam assessing for a heart murmur (auscultation standing, supine, and with Valsalva), femoral pulses to exclude coarctation of the aorta, recognition of the physical stigmata of Marfan syndrome, and measurement of brachial artery blood pressure.

Limitations of the PPE

- Healthy appearing athletes may harbor unsuspected cardiovascular disease.
- 50% to 80% of athletes have no premonitory signs or symptoms prior to their SCA.
- No proven efficacy that the traditional screening PPE reduces SCD.
- Available comprehensive screening questionnaires are underutilized.

Screening ECG

The Italian Experience

- Mandatory annual history and physical and screening ECG required for all competitive athletes beginning in 1982.
- From 1982 to 2004, death in athletes from SCA decreased by 89%.
- A 9% overall positive rate and a 7% false positive rate.
- ECG has a 77% greater specificity for detecting potentially lethal heart disease compared to history and physical alone.
- European Society of Cardiology also supports a common screening protocol based on 12-lead ECG in addition to history and physical exam.

American Heart Association (AHA) Position

- A PPE model with inclusion of ECG is "a benevolent and admirable proposal deserving of serious consideration."
- Considers the European model with inclusion of ECG unrealistic for the United States:
  - Not enough providers adequately trained to do screening.
  - The legal framework for instituting mandatory screening does not exist. A law would be required to mandate screening and make standard disqualification decisions binding.
  - Too expensive. AHA-estimated cost of an annual screening PPE and ECG for all athletes is $2 billion annually at a cost of $330,000 per lethal cardiovascular condition detected.

ATHLETES WITH SUSPECTED OR KNOWN CARDIOVASCULAR DISEASE

Recognition and Evaluation of Worrisome Symptoms

- 20% to 50% of those with SCD had prior symptoms. Many had consulted providers and been reassured.
- Exertional syncope is *always* alarming and should be thoroughly investigated.
  - ECG will detect HCM 95% of time and also screens for LQTS, SQTS, Brugada syndrome, and pre-excitation syndromes. Other diseases that may cause changes in the ECG (myocarditis) can also be detected.
  - Echocardiogram is useful in evaluation for HCM, structural heart disease, and aortic root dilatation.
  - Exercise stress testing may uncover exertional arrhythmias, channelopathies, or ischemia secondary to coronary artery disease or anomaly.
  - Holter monitor or event monitor may also identify arrhythmias.
  - Cardiac MRI or CT scan may show structural abnormalities such as coronary anomalies or ARVC. *It is strongly recommended that these abnormalities be excluded in any athlete with a history of exertional syncope.*
  - Electrophysiology (EP) study may identify inducible arrhythmias or re-entrant pathways.
  - Coronary angiography is the gold standard for identification of coronary artery anomalies and coronary artery disease.
  - Tilt table testing may provide supporting evidence that syncope occurred because of hemodynamic changes (neurocardiogenic syncope).
- Consultation with a specialist is recommended if an abnormality is suspected or identified.
- Athletes should be restricted from sports participation until the diagnostic workup is complete.

Athletes with Cardiovascular Disease

- When athletes are identified with cardiac diseases that place them at risk for SCA or disease progression, the question of advisable athletic activities is very complex. Exercise is a known trigger for SCD and some level of activity modification is recommended.
- The 36th Bethesda Conference provides consensus recommendations for competitive athletes with known cardiovascular abnormalities.
  - A competitive athlete is defined as one who participates on an organized team or individual sport that requires regular competition against others and places a high premium on excellence and requires some form of systematic training. The recommendations are not intended for recreational athletes although many aspects of the guidelines will be applicable.
  - There is established legal precedent that the recommendations of the 36th Bethesda Conference represent competent medical opinion on which to base participation decisions.
- North American Society of Pacing and Electrophysiology Consensus Statement on arrhythmias in the athlete provides recommendations for detecting, evaluating, and treating athletes with arrhythmias.
  - Recommendations on participation are similar to those from the 36th Bethesda Conference.
- AHA Scientific Statement: Recommendations for physical activity and recreational sports participation for young patients with genetic cardiovascular diseases is also a resource for recreational athlete activity recommendations.

Legal Considerations

PPE: A provider is not necessarily liable for missed cardiac conditions that result in sudden death, provided the provider adhered to accepted medical standards. Using a form that includes the AHA-recommended elements should mitigate legal risk.

Screening: A provider is not currently expected to use noninvasive diagnostic testing (ECG or echocardiogram) as part of a routine PPE. Programs that provide ECG and/or echocardiographic screening for unsuspected cardiovascular abnormalities are at unknown legal risk.

Restriction of participation: An institution has a right to restrict an individual from participation as long as that restriction is based on objective medical evidence and supported by the team physician and institutional medical consultants.

EMERGENCY PREPAREDNESS AND MANAGEMENT OF SCA

Emergency Action Plan

- Every school or institution that sponsors athletic activities should have a written and structured emergency action plan (EAP).
- The EAP should be developed in concert with local emergency medical services, school safety officials, likely first responders, and school administrators.

- The EAP should be specific to each individual athletic venue and provide plans for:
  - Communication: a communication system should be in place to activate the emergency medical services (EMS) system and to alert local/school responders and expedite transfer of emergency equipment (e.g., automated external defibrillator [AED]) to the scene.
  - Personnel: an identified team of targeted first responders (e.g., coaches, school health officials) should receive training in the recognition of SCA, cardiopulmonary resuscitation (CPR), and AED use.
  - Equipment: on-site AED programs are strongly encouraged and are likely the only means of achieving early defibrillation in the athletic setting.
  - Transportation: transportation routes for arriving EMS should be defined, as well as transport to an identified hospital with advanced cardiac life support capabilities.
- The EAP should be reviewed and practiced at least annually by all potential responders to an SCA.
- The target time from collapse to first shock should be *less than 3 to 5 minutes*.

Management of SCA

- Initial management of SCA should include early activation of EMS, early CPR, early defibrillation, and early transition to advance cardiac life support.
- Approximately 50% of athletes with SCA have brief myoclonic or seizure-like activity, and SCA should not be mistaken for a seizure.
- Agonal respirations or gasping, and inaccurate rescuer assessment of pulse can also delay recognition of SCA.
- **SCA should be suspected in any collapsed and unresponsive athlete** and an AED applied as soon as possible for rhythm analysis and defibrillation if indicated.
- Commotio cordis should be suspected in any collapsed athlete who has been struck in the chest.
- CPR should be performed while waiting for an AED. The current recommended ratio of chest compressions to ventilation is 30:2. CPR should be restarted immediately after the first shock with repeated rhythm analysis every five cycles (about every 2 minutes) until the victim becomes responsive or advanced life support providers take over.

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The Hypertensive Athlete

30

Mark W. Niedfeldt and Leon Y. Cheng

INTRODUCTION

- Fifty million U.S. adults have hypertension.
- Hypertension (HTN) is the most common cardiovascular condition observed in competitive athletes.
- Athletes are usually considered to be free of cardiovascular disease because of their apparent high level of fitness.
  - Overall incidence of HTN in athletes is approximately 50% less than in the general population, and most cases are in the mild to moderate range.
- Hypertension begins in young adulthood.
  - Incidence increases with age.
    - 5% to 10% of adults 20 to 30 years old
    - 20% to 25% of middle-aged adults
    - 50% over age 60
    - Residual lifetime risk of 90%
  - Almost 80% of adolescents with an elevated blood pressure (>142/92 mm Hg) during preparticipation physical examinations (PPE) do have HTN; therefore, it is important to screen and if diagnosed treat appropriately.

CLASSIFICATION OF HYPERTENSION (JNC-7)

- Progresses through three classifications (Fig. 30-1, Table 30-1)
- Normal is systolic below 120 mm Hg and diastolic below 80 mm Hg
- **Prehypertension is 120 to 139 mm Hg systolic and 80 to 89 mm Hg diastolic**
  - Associated with increased cardiac output (CO) and "normal" vascular total peripheral resistance (TPR).
  - TPR is normal compared with resting levels in normotensives but inappropriately high in face of elevated CO.
    - In a nonhypertensive patient, TPR falls to compensate for rise in CO, thereby maintaining normal BP.
    - Lack of TPR decrease is a result of impaired baroreceptor function.
    - Baroreceptors are "reset" to maintain elevated rather than normal BP over time.
  - People with prehypertension are hypersensitive to catecholamine secretion, mental stress, and have hyperkinetic circulatory state.
- **Stage 1: 140 to 159 mm Hg systolic and 90 to 99 mm Hg diastolic**
  - Earliest stage and most common form detected in the medical setting.
  - Increased heart rate (HR) and CO and decreased TPR.
  - Decreased arterial lumen and disturbed autoregulation of blood flow in the periphery.
- **Stage 2: above 160 mm Hg systolic and above 100 mm Hg diastolic**
  - Normal HR and CO
  - Increased TPR
    - Increased afterload leads to left ventricular hypertrophy (LVH). Severe and/or uncontrolled hypertension may lead to the development of diastolic dysfunction and congestive heart failure.
    - CO can no longer increase in response to exercise or other physiologic demands.
    - Marked LVH exists.
    - Loss of contractility and congestive heart failure may develop. Peripheral edema and pulmonary edema are threats to cardiovascular function and electrolyte balance.
- Most active individuals with HTN will fall into stage 1 or lower stage 2.
- Those with comorbidities such as diabetes or renal disease should be treated at prehypertensive level.
- Values for pediatric athletes are adjusted for age, gender, and height (Table 30-2).
- Higher stages are associated with higher risk of nonfatal and fatal cardiovascular disease as well as progressive renal disease (Fig. 30-2).

CLINICAL PATHOPHYSIOLOGY OF HYPERTENSION

Primary Hypertension

- 95% of cases
- Abnormal neuroreflexes and sympathetic control of peripheral resistance
- Abnormal renal and metabolic control of vascular volume and compliance
- Abnormal local smooth muscle and endothelial control of vascular resistance
- Sustained increases in systemic vascular resistance (SVR)

Secondary Hypertension

- 5% of cases
- Tends to be seen in younger patients or when a rapid onset occurs in adults without a prior history of hypertension
- BP is often poorly responsive to routine therapy

Causes

- Renal (most common)
  - Renal vascular disease
    - Increased renin stimulates conversion of angiotensin I to angiotensin II, which is a vasoconstrictor, and release of aldosterone
    - Renal retention of sodium and water
  - Renal parenchymal disease
    - Inability of damaged kidneys to excrete sodium and water
- Endocrine
  - Adrenal
    - Pheochromocytoma
    - Cushing syndrome
    - Primary aldosteronism
  - Thyroid
    - Hyperthyroidism—increased CO leads to increased systolic and decreased diastolic
    - Hypothyroidism—increased diastolic from peripheral resistance
  - Acromegaly (fluid volume excess)
  - Hyperparathyroidism (increased calcium leads to increased peripheral resistance)
  - Estrogen
    - Oral contraceptive pills (OCP)
    - 5% will develop HTN over 5 years
- Other
  - Coarctation of the aorta

RISK FACTORS FOR HYPERTENSION

- Genetic factors
  - More males than females
  - More Blacks than Whites (2:1) with Asians least
  - Family history

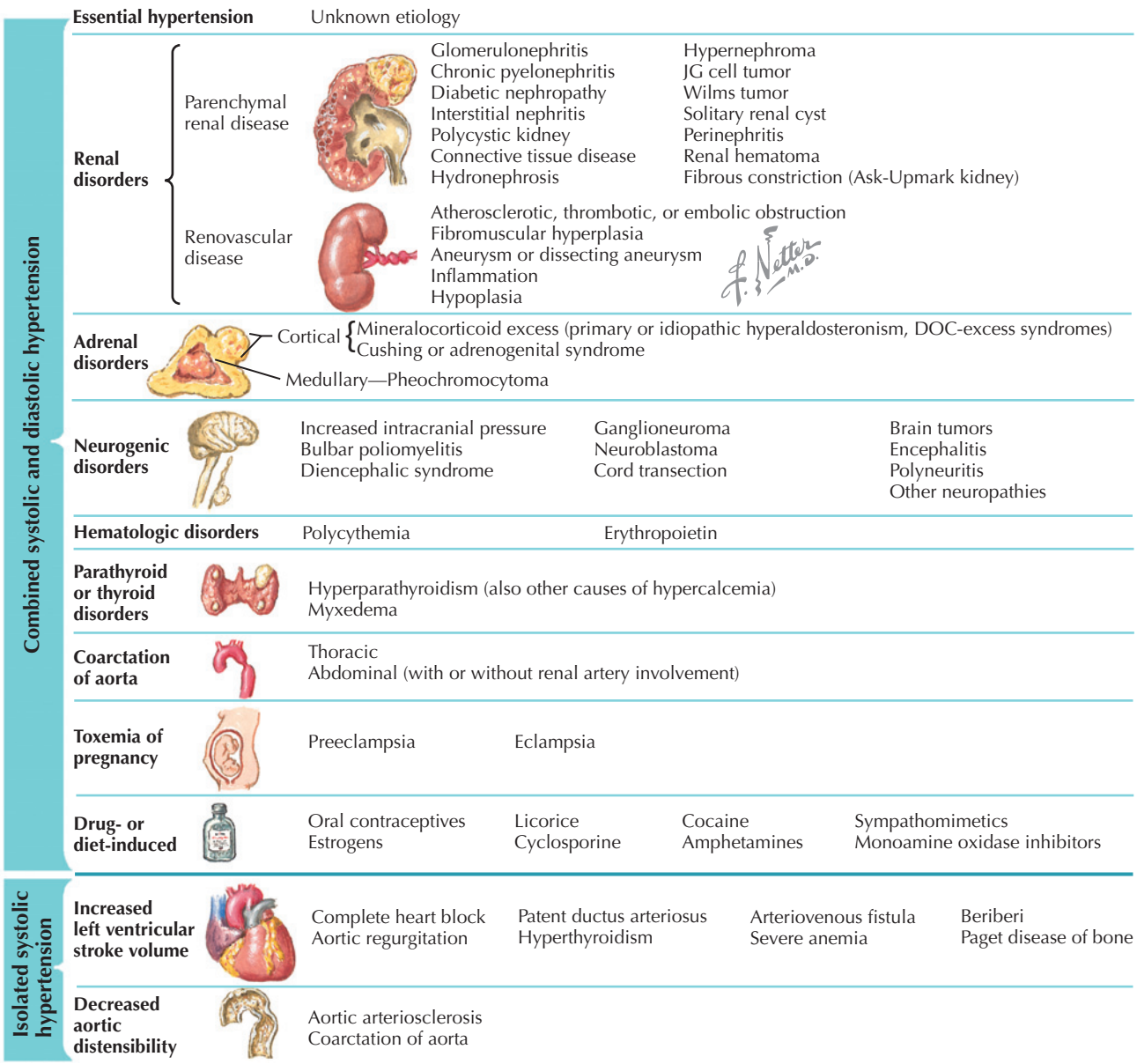


Figure 30-1 Causes of Hypertension.

Table 30-1 CLASSIFICATION OF HYPERTENSION

	Systolic (mm Hg)	Diastolic (mm Hg)
Normal	<120	<80
Prehypertension	120-139	80-89
Stage 1 Hypertension	140-159	90-99
Stage 2 Hypertension	≥160	≥100

Classification of blood pressure (BP) for adults aged 18 years or older. The classification is based on the mean of two or more properly measured seated BP readings on each of two or more office visits. In contrast with the classification provided in the JNC VI report, a new category designated prehypertension has been added, and stages 2 and 3 hypertension have been combined. Patients with prehypertension are at increased risk for progression to hypertension; those in the 130/80 to 139/89 mm Hg BP range are at twice the risk to develop hypertension as those with lower values.

Adapted from the Seventh Report of the Joint National Committee on Detection, Evaluation, and Treatment of High Blood Pressure (JNC-7). JAMA 289(19):2560-2575, 2003.

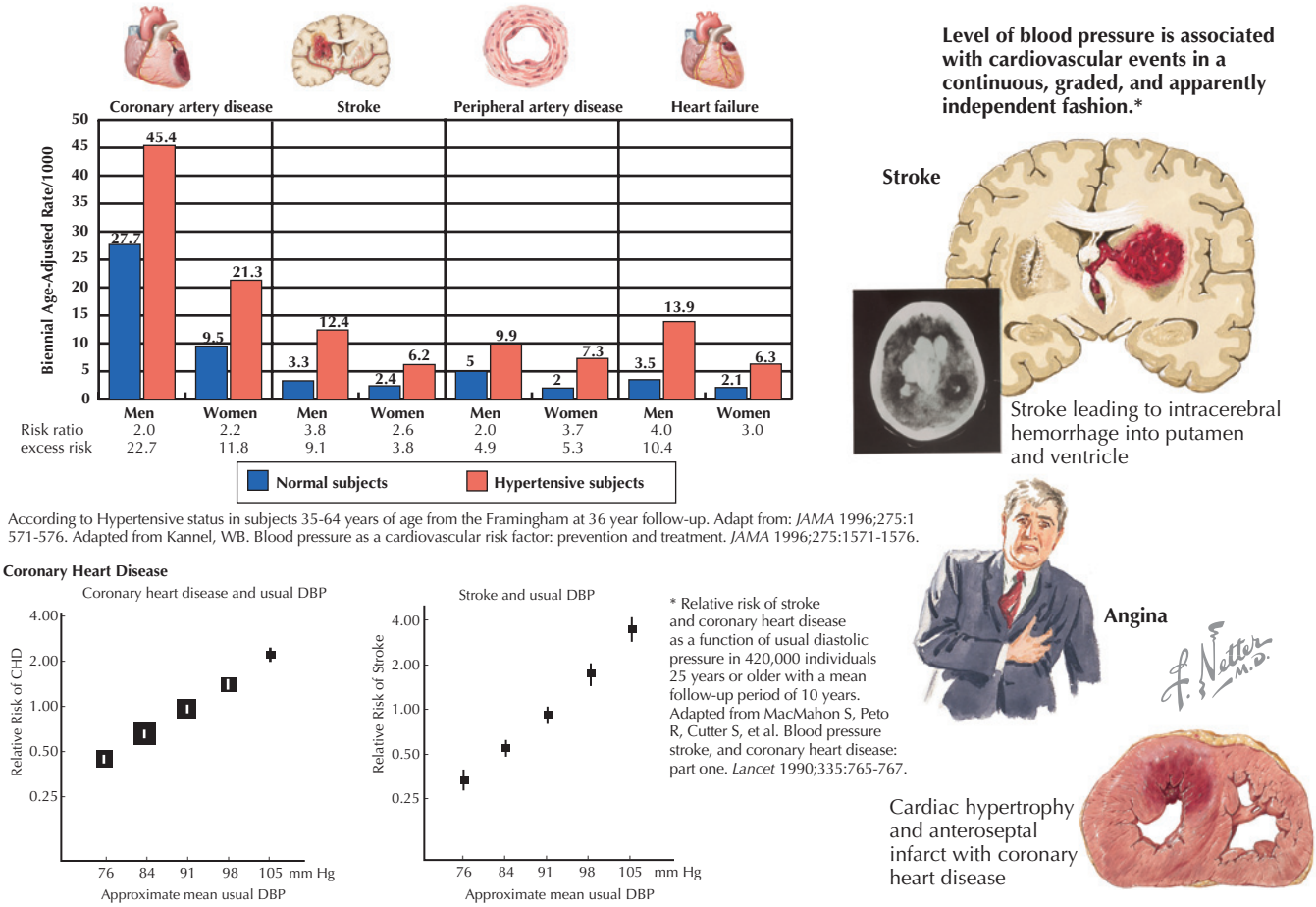
Table 30-2 CLASSIFICATION OF BLOOD PRESSURE FOR PEDIATRIC ATHLETES

Normal	Systolic and diastolic BP <90th percentile
Prehypertension	Systolic and/or diastolic BP ≥90th percentile but <95th percentile, or if BP exceeds 120/80 mm Hg even if <90th percentile.*
Stage I hypertension	Systolic and/or diastolic BP between the 95th percentile and 5 mm Hg above the 99th percentile
Stage 2 hypertension	Systolic and/or diastolic BP ≥99th percentile plus 5 mm Hg.

*A systolic pressure of 120 mm Hg may typically occur at 12 years of age, whereas a diastolic pressure of 80 mm Hg typically occurs at 16 years of age.

In children, the above definitions, based on the 2004 National High Blood Pressure Education Program Working Group (NHBPEP), are used to classify BP measurements. BP percentiles are based on gender, age, and height and on measurements on three separate occasions. The systolic and diastolic BP are of equal importance; if there is a disparity in category, the higher value determines the severity of the HTN.

From The National Heart, Lung, and Blood Institute: The Fourth Report on the Diagnosis, Evaluation, and Treatment of High Blood Pressure in Children and Adolescents. Pediatrics 114(2):555, 2004.



According to Hypertensive status in subjects 35-64 years of age from the Framingham at 36 year follow-up. Adapt from: *JAMA* 1996;275:1 571-576. Adapted from Kannel, WB. Blood pressure as a cardiovascular risk factor: prevention and treatment. *JAMA* 1996;275:1571-1576.

Figure 30-2 Hypertension as Risk Factor for Cardiovascular Disease.

- Metabolic factors
  - Obesity
  - Glucose intolerance
  - Endocrine disorders (see "Causes")
- Stress
  - Environmental
  - Social
  - Leads to chronic neurogenic activation of the sympathetic nervous system
- Behavioral factors
  - High sodium intake
  - Excessive alcohol consumption
  - Drug abuse
    - Recreational: cocaine, tobacco (chew)
    - Ergogenic: stimulants, anabolic steroids

DIAGNOSIS OF HYPERTENSION

Resting Blood Pressure (BP)

- Diagnosis of hypertension is based on an average of two or more BP readings above 140/90 mm Hg at each of two or more subsequent visits (see Table 30-1).
- In children and adolescents, hypertension is defined as average systolic or diastolic BP ≥ 95th percentile for age, gender, and height, measured on three occasions (see Table 30-2).
- Prehypertension is usually marked by increase in systolic BP primarily caused by increased CO.
- As hypertension progresses, the diastolic BP begins to increase as well, primarily caused by increased TPR.

Environment during Measurement

- Measurement of blood pressure should be performed in a standard measurement situation, preferably a quiet area.
- Let the athlete sit for a few minutes if possible.
- Repeat BP measurements if elevated.
- Choose the appropriate size blood pressure cuff. Many athletes will need a large cuff, and a thigh cuff should be available for very large athletes.
  - The inflatable bag of the BP cuff should cover approximately 80% of the arm's circumference.
- Avoid rapid deflation of cuff.

"White Coat" Hypertension and Other Stress Phenomena

- Anxiety provoked by medical examination or other sources of mental stress can lead to artificially elevated BP, known as "white coat" hypertension.
- Average of several readings is better estimate of true BP.
- If initial BP is high, have athlete rest for 5 minutes and repeat BP check.
- If BP remains elevated, check BP at least once per week for at least two additional visits.
- Averaged daily BP is better predictor of later end-organ damage than random office BP.
- Ambulatory 24-hour BP monitoring remains difficult and expensive.
- Athletes/exercisers can take their own BP, but must be well trained in measurement of BP. Emphasize importance of *accurate* readings.

CLINICAL EVALUATION

History

- Cardiovascular risk factors:
  - Smoking
  - Family history of cardiac disease in men younger than 55 and women younger than 65
  - Obesity
  - Physical inactivity
  - Diabetes
  - Dyslipidemia
- Diet and behaviors
  - High sodium and saturated fat intake (fast foods)
  - Alcohol consumption
  - Herbs and supplements (especially those for energy or weight loss)
  - Drug use
    - Over the counter (nonsteroidal anti-inflammatory drugs [NSAIDs], decongestants, caffeine, diet pills)
    - Prescription
    - Illicit (including ergogenic)
- Stress
- Review of systems to rule out secondary causes of HTN

Physical Exam

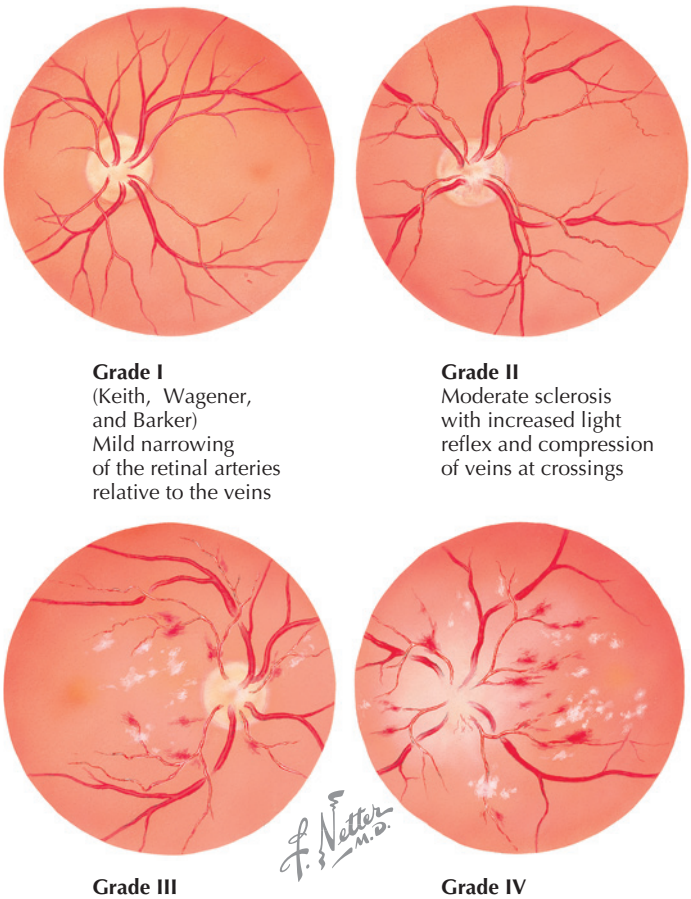
- Focused on ruling out secondary causes of hypertension and looking for end organ damage
- Body mass index (BMI) often not useful in athletes because of higher levels of muscle mass
- Fundi are examined for retinopathy, as indicated by retinal hemorrhages or exudates, with or without papilledema (Fig. 30-3)
- Thyroid
- Cardiovascular (pulses, murmurs, bruits)
- Abdominal masses
- Peripheral edema

Laboratory Studies

- Hct, Na^+, K^+, BUN, creatine, glucose, lipid panel, urinalysis, EKG
- Further workup if suspicious of secondary cause

Exercise Stress Testing

- **Although not routinely done, exercise stress testing can be used to predict and differentiate types of hypertension.**
- Differentiating stages of hypertension
  - People with prehypertension start at higher resting levels than normotensives but do not show abnormally high BP levels during maximal exercise.
  - Some research suggests that prehypertensives show less increase in pulse pressure than normotensives. This may be due to less compliant vascular beds.
  - Rapid elevation in systolic BP indicates established hypertension.
  - Hypertensives tend to have increased diastolic BP during and after exercise.
- Predicting future hypertension
  - Exaggerated systolic BPs (>85th percentile) during treadmill testing are associated with higher risk of developing future hypertension.
  - Exaggerated diastolic BP response to exercise predicts risk for new-onset hypertension (2 to 4 times increased risk).
  - Delayed recovery systolic BP response is also predictive of future hypertension.
  - Exercise BP may be significant predictor of adverse cardiovascular events in high-risk patients.



Grade I
(Keith, Wagener, and Barker)
Mild narrowing of the retinal arteries relative to the veins

Grade II
Moderate sclerosis with increased light reflex and compression of veins at crossings

Grade III
Edema, exudates, and hemorrhages; sclerotic and markedly spastic ("silver-wire") arteries

Grade IV
Papilledema or choked disc, extensive hemorrhages, and exudates

Figure 30-3 Eye Grounds in Hypertension.

APPROACHES TO MANAGEMENT OF HYPERTENSION

- Management of hypertensive athletes (or exercising adults with hypertension) generally involves a combination of nonpharmacologic therapies, often with one or more antihypertensive medications.
- Goals
  - BP below 140/90 mm Hg
  - BP below 130/80 mm Hg if coexisting disease
  - Primary focus on systolic blood pressure
- Special considerations in athletes
  - Potential influence of exercise and training on HTN
  - Potential side effects of antihypertensive medications on athletic performance
  - Some therapies are banned by the U.S. Olympic Committee (USOC) or National Collegiate Athletic Association (NCAA)

Nonpharmacologic Therapy

- Nonpharmacologic strategies provide safe, effective foundation for any good antihypertensive regimen because the risk of side effects is low to nonexistent.
- Depend on long-term compliance and lifestyle changes, which can be difficult because of deeply ingrained lifestyle behaviors.
- Most appropriate for above optimal blood pressure and stage 1 hypertension.

Dietary Interventions

ELECTROLYTES

- **Na^+**
  - A reduction in sodium can result in significant decrease in blood pressure.
  - Fast food and lunch meats provide 75% of sodium in typical American diet.
  - 2.3 g per day (6 g NaCl) is recommended intake.
  - Salt sensitive groups benefit most:
    - Two thirds of African-Americans
    - Diabetics
    - Older people
- **K^+**
  - Adding high potassium foods may protect against developing hypertension and improve blood pressure control.
  - Increasing K^+ decreases BP in hypokalemic patients (endurance athletes) and may protect from ventricular ectopy.
  - Increasing potassium may have some effect in normokalemic.
  - Effect of potassium supplementation greatest in those with higher levels of sodium in diet.
  - Goal is 90 mmol per day.
- **Ca^{++}**
  - 800 to 1200 mg per day of elemental calcium may reduce BP in calcium deficient (especially women), but the overall effect is minimal.
  - Supplementation not routinely recommended for treatment of hypertension, but is recommended as a healthy lifestyle behavior.
- **Mg^{++}**
  - Controversial, possible benefit.
  - Important in patients who are deficient, especially from diuretics.
  - Supplementation not routinely recommended.

WEIGHT REDUCTION

- Obesity is defined as body mass index (BMI = kg/m^2) greater than 30.
- Obesity increases preload and afterload, resulting in hypertensive effect.
- The mechanism by which obesity raises BP is not well understood. Obesity, glucose intolerance, and hypertension are components of the metabolic syndrome, which suggests hyperinsulinemia and insulin resistance may play a central role.
- Weight reduction is the most effective nonpharmacologic measure for BP reduction.
  - For each 1 kg of weight loss, the systolic and diastolic BP each fall by approximately 1 mm Hg.
  - Weight reduction of 10 pounds reduces BP in overweight athletes with HTN and enhances BP-lowering effects of medications, possibly because of reductions in both preload and afterload.
- DASH diet
  - Reduced levels of total and saturated fat and cholesterol.
  - Increased potassium, calcium, magnesium, fiber, and protein.
  - Emphasizes fruits, vegetables, low fat dairy, whole grains, fish, poultry, and nuts.
  - Showed decreases in blood pressure equivalent to medications (11.4/5.5 mm Hg in hypertensive patients and 5.5/3.0 mm Hg decrease in those with prehypertension) and is especially effective in African-Americans.
- Fish oil supplementation
  - High doses may decrease blood pressure by 6/4 mm Hg.
  - Long-term safety of fish oil in doses high enough to lower the blood pressure is unknown.

LIFESTYLE CHANGES

- Alcohol consumption should be limited to 1 ounce (2 beers) of alcohol per day.
- Stimulants—caffeine acutely raises blood pressure, but avoidance is unnecessary because of the rapid development of tolerance.
- Cocaine.
- Anabolic steroids.
- Other medications such as sympathomimetics (decongestants), appetite suppressants, and oral contraceptives may affect blood pressure.

Relaxation Techniques

Relaxation techniques such as biofeedback, stress management, muscle relaxation techniques, meditation, and yoga may be used but their main value is as adjunct therapy.

Exercise

- Vigorous exercise correlates with lower risk of developing hypertension.
- Regular aerobic exercise regimen can within 4 weeks lower the BP by as much as 5 to 15 mm Hg in patients with essential hypertension.
- Some clinical trials show that aerobic exercise conditioning lowers resting systolic BP by an average of 11 mm Hg and diastolic BP by an average of 6 mm Hg in hypertensives.
  - Benefit more marked in prehypertension and stage 1 hypertension.
  - Some are poorly designed, uncontrolled studies.
  - Difficult to tell if benefit is direct effect of exercise or secondary to weight loss.

Pharmacologic Therapy

Basic Concepts

- Must be individualized and carefully monitored for potential side effects.
- Most individuals will require two or more medications to achieve goal blood pressure.
- If initial blood pressure is more than 20/10 mm Hg over goal, consider initiating therapy with two agents.
- Monitor effects of therapy on performance.
- NSAIDs may decrease activity of many antihypertensive medications.
  - Beta-blockers, ACE inhibitors, and diuretics may have their therapeutic activity inhibited.
  - Limited reports suggest possibility of acute increases in BP with uses of cyclooxygenase-2 (COX-2) inhibitors.
- USOC and NCAA regulations ban the use of some medications such as diuretics and beta-blockers (in some events).

Diuretics (Thiazides, Loop Inhibitors)

- Decrease plasma volume, cardiac output, and SVR (Fig. 30-4)
  - Short-term use reduces maximal exercise capacity and submaximal endurance
  - Attenuated increase in BP, decreased SV, but maintenance of normal HR response during exercise
- Decreased mortality and morbidity in elderly and are superior in preventing one or more forms of cardiovascular disease
- Inexpensive
- Side effects
  - Hypovolemia, orthostatic hypotension—Not recommended for athletes prone to dehydration because of further reductions in intravascular volume.
  - Urinary loss of K^+ and Mg^{++}, which can lead to muscle cramps, arrhythmias, and rhabdomyolysis, especially when competing or exercising vigorously in warm

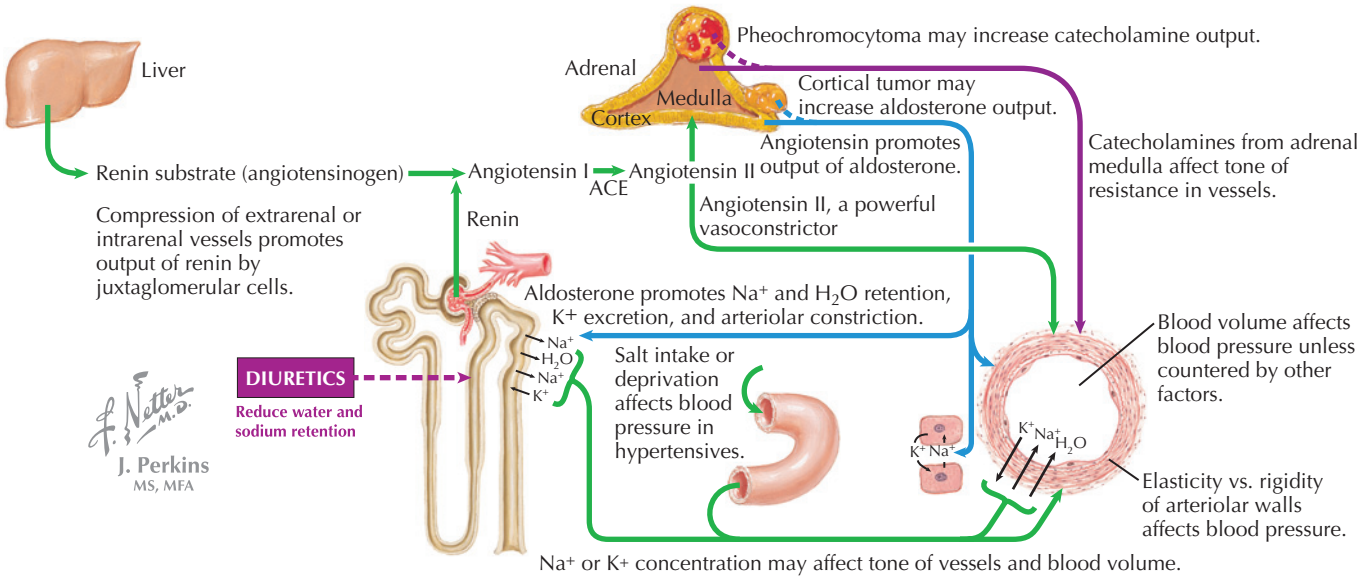


Figure 30-4 Hypertension Treatment: Diuretics.

weather. Cramping may occur despite normal serum potassium.
  - Increases in plasma cholesterol, glucose, uric acid (higher doses).
  - Higher incidence of sexual dysfunction in males.
- Thiazides are the preferred first step in therapy for casual exercisers, active elderly, and African-Americans.
- Loop diuretics are inappropriate for use in athletes.
- All diuretics are athletic association–banned substances and cannot be used by athletes subject to drug testing because they dilute the concentration of steroids and drugs in urine.

Angiotensin-Converting Enzyme (ACE) Inhibitors

- Captopril, enalapril, lisinopril, benazepril, fosinopril, quinapril, and ramipril (Fig. 30-5)
- Competitive inhibition of conversion of angiotensin I to angiotensin II by angiotensin-converting enzyme in plasma and vascular smooth muscle
- Blocks vasoconstriction and Na^+ retention from angiotensin II
- Increases stroke volume, slight decrease in HR, decreases TPR
- Beneficial effects in patients with heart failure, systolic dysfunction, or nephropathy; reverses ventricular hypertrophy and microalbuminuria and preserves renal function
- In exercise
  - No major effect on energy metabolism
  - No impairment of $\dot{V}O_2$ max, training, or competition
  - Decreased "exaggerated" BP effect during exercise, including isometric exercise
  - Major side effect is a dry, nonproductive cough
  - Anecdotal reports of postural hypotension when stopping abruptly after intense exercise (adequate cool down needed)
- Excellent for mild to moderate hypertension, and effectiveness may be improved with addition of low-dose thiazide diuretic

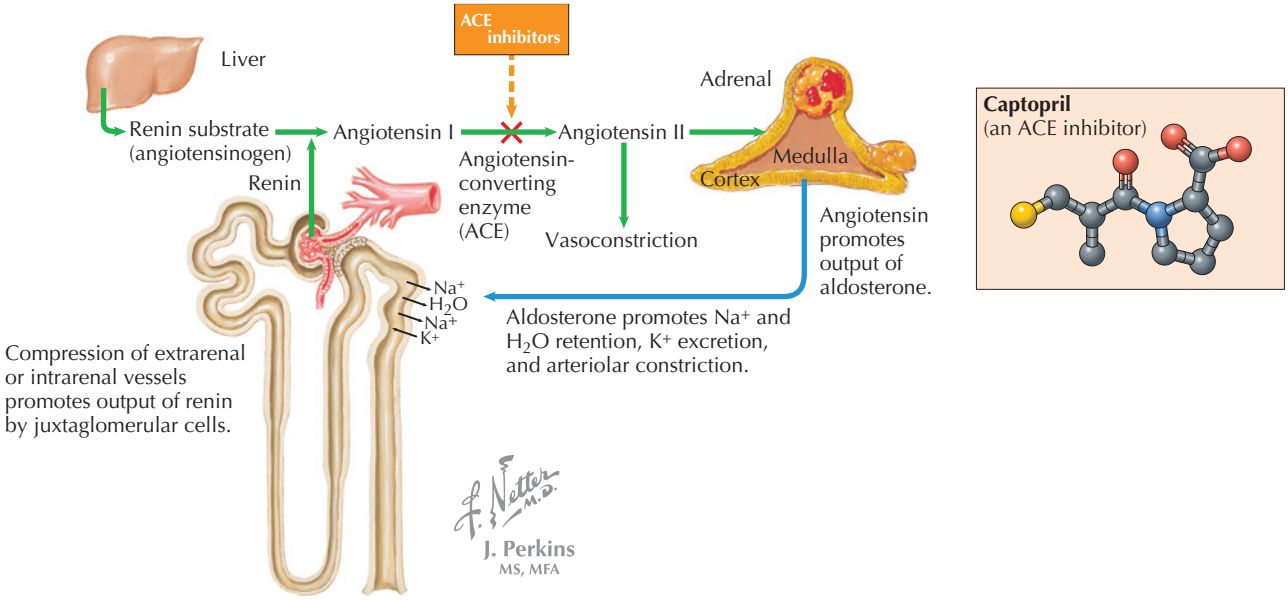


Figure 30-5 Hypertension Treatment: Angiotensin-Converting Enzyme Inhibitors.

- Often first-line agent for hypertension in active athletes
- Concomitant use of NSAIDs may increase potassium-sparing effect and potentially cause hyperkalemia
- Women of childbearing age need contraception because this class is contraindicated in pregnancy

Angiotensin II Receptor Blockers

- Irbesartan, losartan, valsartan, and candesartan
- Block renin-angiotensin system by preventing angiotensin II from binding to its subtype 1 receptor
- Selective blockade prevents vasoconstriction and aldosterone secretion
- Similar effects to ACE inhibitors but avoids the most common side effect of dry cough
- May improve exercise tolerance in hypertensives because angiotensin II levels increase during exercise
- May be beneficial in athletes with early diastolic dysfunction because of blocking effect during LV relaxation
- Generally recommended only for those who cannot tolerate ACE
- Women of childbearing age need contraception because this class is contraindicated in pregnancy

Alpha$_1$ Receptor Blockers

- Prazosin, terazosin, and doxazosin
- Competitively block postsynaptic alpha$_1$ arteriolar smooth muscle receptors
- Decrease SVR (no reflex increase in HR or CO)
- First dose effect of syncope, so give first dose at night
- In exercise
  - No major changes in energy metabolism
  - $\dot{V}O_2$ max preserved
  - No major effect on training or sports performance
- Useful for diabetic athletes with HTN and hypercholesterolemia
- Antihypertensive and Lipid-Lowering Treatment to Prevent Heart Attack Trial (ALLHAT)
  - Discontinued doxazosin arm because of increased congestive heart failure as compared to diuretic
  - Use this class with caution in masters athletes over age 55

Central Alpha Antagonists

- Clonidine, guanabenz, guanfacine, and methyldopa
- Act on alpha$_2$ receptors in brainstem to block central sympathetic stimulation with decreased HR and decreased TPR at rest
- Also block sympathetically mediated sodium retention
- In exercise
  - No major changes in energy metabolism
  - $\dot{V}O_2$ max preserved
  - No major effect on training or sports performance
- Side effects
  - Mild to moderate drowsiness and dry mouth
  - Impotence
  - Rebound hypertension with abrupt discontinuation of oral clonidine
- Clonidine available in transdermal system (patch replaced once per week)
- Rarely used because of side effects

Beta Blockers

See Table 30-3

- Noncardioselective
  - Decreases heart rate 20% to 30%
  - Decreases contractility
  - Increases SVR (muscle, skin)
  - Inhibition of lipolysis, glycogenolysis
  - Increased cholesterol (decreased HDL)

Table 30-3 BETA-BLOCKING AGENTS ARRANGED BY CARDIOSELECTIVITY AND INTRINSIC SYMPATHOMIMETIC ACTIVITY (ISA)

	No ISA	ISA
Cardioselective	Atenolol (Tenormin) Metoprolol (Lopressor) Betaxolol (Kerlone) Bisoprolol (Zebeta)	Acebutolol (Sectral)
Noncardioselective	Nadolol (Corgard) Propranolol (Inderal) Timolol (Biocadren)	Pindolol (Visken) Carteolol (Catrol) Penbutolol (Levatol)

  - Increased perception of exertion
  - Bronchoconstriction in predisposed athletes
- Cardioselective
  - Less effect on beta-2 vasodilatation, lipolysis, glycogenolysis
  - Impairment of CO and $\dot{V}O_2$ max is generally similar but may be less because of compensatory increase in SV
- In exercise
  - Significant loss of $\dot{V}O_2$ max
    - Decreased CO and skeletal muscle flow
    - Well trained have greater drop in $\dot{V}O_2$ max than untrained subjects
  - Beta-blockers increase perceived exertion in working muscles, thus causing reduced endurance, probably as a result of metabolic effects with no increase in perceived cardiovascular exertion
  - Beta-blockers decrease performance more in people with high percentage of slow-twitch muscle fibers. This effect is more pronounced with propranolol (noncardioselective) than atenolol (cardioselective).
  - Impairment of substrate mobilization results in earlier fatigue and lower lactate threshold
    - Blocks lipolysis and glycogenolysis
    - The symptoms of hypoglycemia during or after intense exercise may be masked in diabetics
  - Exercise bronchospasm may be increased, so use with caution in athletes with asthma
  - Side effects less with cardioselective
- Not recommended in athletes unless underlying condition requires their use
  - Intermittent exertion sports
  - Untrained or partially trained athletes
  - Patients with arteriosclerotic heart disease where increased exercise tolerance may be noted
  - Hypertensives with excessive rise in systolic BP during exercise
  - Performance anxiety in minimal-exertion sports
- Banned in precision sports

Combined Alpha- and Beta-Blocker

- Labetalol, carvedilol
- Three effects (Fig. 30-6)
  - Beta-blockade (decreased HR leading to decreased CO, decreased renin)
  - Alpha$_1$-blockade (decreased vasoconstriction leading to decreased TPR)
  - Beta$_2$-agonist (decreased TPR)
- Beta effects greater than alpha (3:1 for oral formulations)
- Decreased SVR
- Less impairment of muscle blood flow and $\dot{V}O_2$ max
- May be best choice if beta-blockade is necessary
  - CO decreased 10% to 14% at rest and during exercise after 1 year of therapy

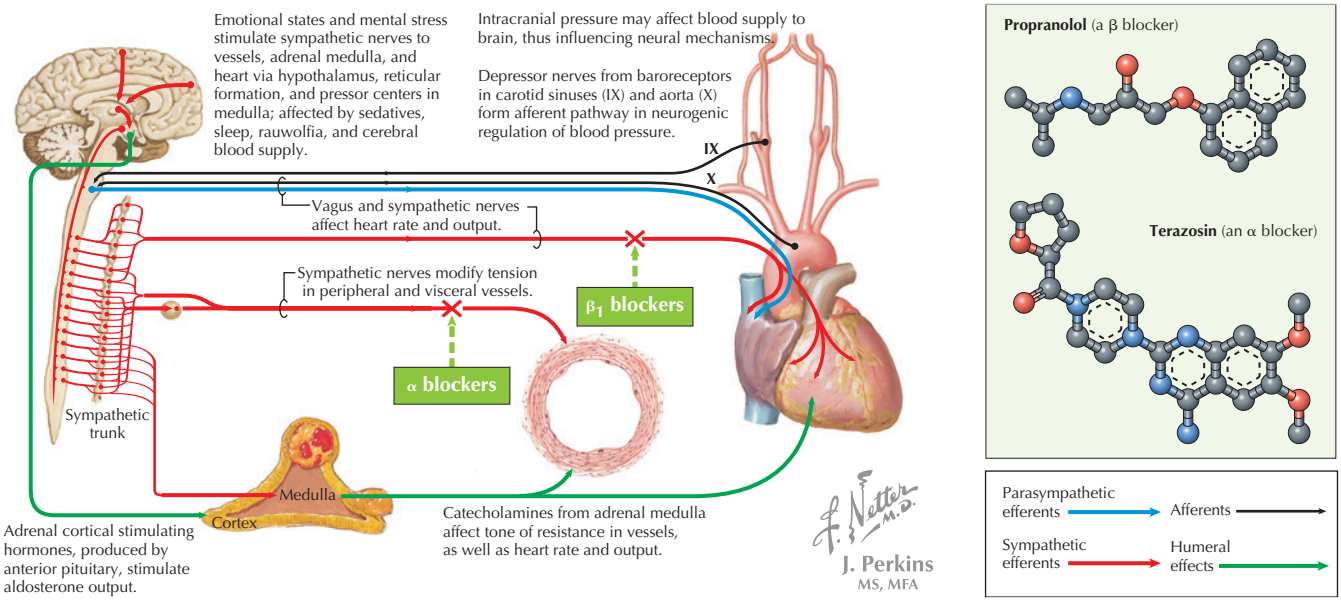


Figure 30-6 Hypertension Treatment: Beta-Blockers and Alpha-Blockers.

- CO gradually returns to baseline over next 5 years because of increased SV
- TPR remains decreased 15% to 20%
- Exercise hemodynamics return to normal

Calcium Channel Blockers

- Include the dihydropyridines (e.g., amlodipine, nifedipine, nicardipine, felodipine, nisoldipine) and nonhydropyridines (e.g., verapamil, diltiazem)
- Inhibit calcium slow-channel conduction, reducing calcium concentration in vascular smooth muscle cells and leading to decreased SVR (generalized vasodilatation)
- Effective in reversing ventricular hypertrophy
- Dihydropyridines (e.g., nifedipine, amlodipine)
  - Reflex tachycardia
  - Fluid retention (pedal edema)
  - Vascular headaches
- Nondihydropyridines (e.g., verapamil, diltiazem)
  - Heart rate suppression
  - Minor impairment of maximal HR
  - Decreased LV contractility
  - Verapamil decreases pressor response during isometric exercise
  - Women show greater BP responsiveness to exercise than men with diltiazem
  - Constipation (verapamil)
- In exercise
  - No major effect on energy metabolism
  - $\dot{V}O_2$ max generally preserved
    - Potential for competitive "steal" of muscle blood flow because of vasodilatation
    - Earlier onset of lactate threshold
  - Verapamil preferred over dihydropyridines for high-intensity exercise because of improved BP control
- Generally well tolerated and effective for active patients, especially young African-Americans
- Many physicians have found this class of drugs useful as first-step therapy in athletes
  - May be particularly effective in hypertensive patients who are noncompliant with dietary salt restriction
  - May also be preferred in patients concurrently taking NSAIDs because antihypertensive efficacy is not blunted

Other

- Direct vasodilators (e.g., hydralazine, minoxidil) directly cause smooth muscle vasodilation in arterioles. They should be used in conjunction with diuretic and beta-blocker because of reflex tachycardia and excess fluid retention.
- Peripheral-acting adrenergic antagonists (e.g., guanadrel, guanethidine, reserpine, and mecamylamine) inhibit catecholamine release and may cause serious orthostatic and exercise-induced hypotension.

APPROACH TO ACTIVE PATIENTS

Prehypertension

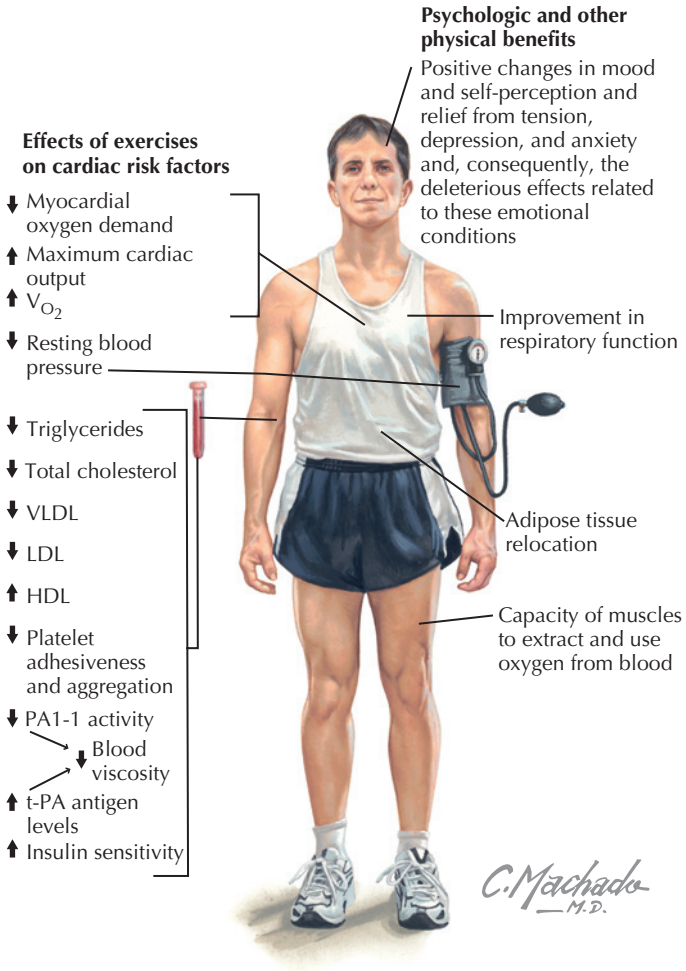
- Modify lifestyle

Stage 1 Hypertension

- Nonpharmacologic intervention
  - 3 to 6 months
  - Monitor frequently
- Pharmacologic therapy
  - Low dose
  - Observe for 6 to 8 weeks
- Initial therapy
  - ACE
  - Thiazide diuretic initially or added if Na^+ sensitive
  - Combined beta/alpha if beta-blockade needed
- Reduce or withdraw in 6 months
  - Small number will remain normotensive after BP is controlled
  - May involve reestablishing baroreceptor control mechanisms
- Emphasize need for long-term follow-up care and management

EXERCISE PRESCRIPTION FOR HYPERTENSIVE PATIENTS

- The recommended mode, frequency, duration, and intensity of exercise is generally the same as those for nonhypertensive individuals (Fig. 30-7).
- Repetitive performance of both aerobic and resistance exercise lowers systolic and diastolic blood pressure.
  - Each 30-minute period of aerobic exercise at 50% of maximal oxygen uptake lowers BP for 24 hours.



The physical activity guidelines are targeted to increase physical activity to promote health but will not necessarily result in physical fitness and should not diminish the importance of achieving physical fitness.

Figure 30-7 Effects of Exercise on Cardiovascular Health.

- An even greater reduction is seen with exercise at 75% of maximal oxygen uptake.
- Monitor blood pressure every 2 to 4 months to monitor impact of exercise.
- Prescribing exercise—"FITT"
  - Frequency: five to six sessions per week.
  - Intensity: 55% to 70% of predicted maximum heart rate (MHR).
    - Moderate-intensity exercise (55% to 70% MHR; defined as 40% to 70% $\dot{V}O_2$ max by some) lowers BP more effectively than higher intensities (80% to 85% MHR). Higher intensity exercise may actually increase resting BPs.
    - Training at 20% and 60% of maximum work capacity results in similar resting BP reductions.
    - In older or less fit patients, start at 55% to 60% MHR; in more fit patients, start at 65% to 70% MHR.
    - Predicted MHR: 220 minus age.
  - Time (duration): initially 15 to 20 minutes per session; eventually 30 to 40 minutes per session
  - Type of exercise: "dynamic isotonic exercise" or moving body through space is most effective. Examples include walking, jogging, swimming, cycling, cross-country skiing, and aerobic dance.

Table 30-4 EXERCISE RESTRICTIONS FOR HYPERTENSIVE ATHLETES

Prehypertensive	No restrictions.
Controlled mild/moderate (<140/90)	No restrictions on dynamic exercise; physicians may choose to limit isometric training or sports in some cases.
Uncontrolled (>140/90)	Limit to low-intensity dynamic exercise. Avoid isometric sports.
Controlled with end organ damage	Limit to low-intensity dynamic exercise. Avoid isometric sports.
Controlled severe hypertension	Low-intensity dynamic sports if the blood pressure is under adequate control.
Secondary hypertension, renal origin	Low-intensity sports are recommended. Avoid "collision" sports that could lead to kidney damage.

Information from 26th Bethesda Conference: Recommendations for determining eligibility for competition in athletes with cardiovascular abnormalities. J Am Coll Cardiol 24(4):845-899, 1994.

- Resistance training
  - Circuit weight training may reduce BP when performed at 30% to 50% maximum resistance with intervals of 15 to 30 seconds between stations.
  - Progressive resistance exercise results in small reductions in resting BP.
  - Regular resistance training leads to attenuated BP and HR responses to any given load.
- Hypertensive athletes should have their blood pressure controlled before returning to participation in vigorous sports.
- Recommendations regarding athletic participation are based on the 36th Bethesda guidelines (Table 30-4)
  - Presence of stage 1 hypertension in the absence of target organ damage including LVH or concomitant heart disease should not limit the eligibility for any competitive sport.
  - Athletes with stage 2 hypertension should be restricted from high-static sports (activities that generate large intramuscular forces with little change in muscle length) until blood pressure is controlled because they do not shunt blood to skin as effectively, resulting in increased core temperatures and increased fluid and K^+ losses.
  - In secondary hypertension of renal origin
    - Low intensity sports are recommended.
    - Should avoid "collision" sports that could lead to kidney damage.

RECOMMENDED READINGS

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Exercise-Induced Bronchospasm, Anaphylaxis, and Urticaria

31

Marc A. Molis and Whitney E. Molis

EXERCISE-INDUCED BRONCHOSPASM

Definitions: Exercise-induced bronchospasm (EIB) is defined as a decline in forced expiratory volume in 1 second (FEV_1) or peak expiratory flow rate (PEFR) shortly after onset or cessation of exercise. The terms *exercise-induced asthma* (EIA) and *exercise-induced bronchospasm* are often used interchangeably; however, *exercise-induced asthma* should be reserved for those athletes who have a known diagnosis of asthma or asthma symptoms at rest. *Exercise-induced bronchospasm* should refer to those athletes that have symptoms only while exercising.

Pulmonary function criteria: A 15% decline in FEV_1 with exercise is the generally accepted value for diagnosing EIB. For patients with a known history of asthma, some people will use a 20% decline in FEV_1 with exercise to establish a diagnosis. Maximal decrease in FEV_1 usually occurs after 5 to 8 minutes of vigorous exercise. Pulmonary function tests (PFTs) usually return to baseline 60 minutes after exercise.

Epidemiology: Approximately 12% of the total population experiences EIB. EIB can be detected in about 41% of those people with a history of allergic rhinitis; 70% to 90% of asthmatics have EIA. Occurs equally between sexes and can occur at any age.

Risk factors: History of asthma is the biggest risk factor for EIA. For EIB, risk factors include family history of asthma, allergic rhinitis, viral bronchitis, and frequent chest symptoms (coughing, congestion).

Mechanism: The exact mechanism of EIB is unknown at this time, but there are two major theories:

- **Thermal theory:** Hyperventilation during exercise causes loss of heat and drying of the airways which in turn causes a transient bronchoconstrictive response.
- **Osmolarity theory:** Heat loss during exercise and rapid rewarming of the airways after exercise causes a reactive hyperemia of the microvasculature and edema of the airways that sets up an osmotic gradient. This stimulates the release of inflammatory mediators that result in bronchospasm.

Triggers: Mouth breathing (unlike nose breathing, which warms the air, mouth breathing does not and is more likely to cause cooling of the airways and trigger EIB), dry air, cold air (essentially, colder, drier air will cause more symptoms), pollution and allergens in the air; intense exercise is more likely to trigger EIB (e.g., cross-country skiing, basketball, running); chemicals such as chlorine in pools, and insecticides, pesticides, herbicides, and fertilizers used to maintain fields can trigger EIB.

Clinical signs and symptoms: Can include wheezing, shortness of breath, coughing, chest tightness, chest pain (usually reported in children), feeling "out of shape," early fatigue, dry cough postexercise, and poor performance. Similar to acute asthma, but of much shorter duration. Can have a late-phase response that occurs 4 to 8 hours after exercise.

Physical exam: May include the following on exam:

- Lungs—wheezing (especially expiratory phase); may hear rales or rhonchi
- Skin—signs of atopic disease
- Nose—enlarged and boggy turbinates
- Throat—cobblestoning, enlarged tonsils
- Sinus—tenderness or pressure

Testing: EIB is generally a clinical diagnosis, and starting patients on a trial of albuterol (or other short-acting beta-2 agonist) based on history and exam is often sufficient. Further testing may be warranted if the patient does not respond to the treatment, has a negligible reduction of symptoms, or has symptoms consistent with underlying asthma.

Medications and testing: When doing PFTs, certain medications need to be avoided or stopped so as to not confound the testing and produce false negative results.

- Inhalers—Albuterol and other beta-agonists should not be used 12 to 24 hours prior to testing.
- Steroids—If a patient has been on inhaled steroids for more than 4 weeks, it may have a protective effect and symptoms of EIB may not be produced during testing.
- Theophylline—avoid 24 hours prior to testing.
- Leukotriene modifiers—avoid at least 24 hours prior to testing.
- Cromolyn sodium/nedocromil sodium—avoid 24 hours prior to testing.
- Antihistamines—do not need to be stopped for PFTs, but need to be stopped for skin testing.

Types of testing:

- Pulmonary function tests (PFTs)
  - PFTs can range from hand-held spirometry, which can be done in the office, to more formal testing with PFTs, including methacholine challenge (used to diagnose asthma, usually when the diagnosis is uncertain such as in chronic cough) and DLCO (diffusing capacity of the lung for carbon monoxide) used to diagnose pulmonary parenchymal disorders such as chronic obstructive pulmonary disease (COPD). Formal PFTs with methacholine need to be done in a hospital setting because of the risk of airway compromise.
  - PFTs can be performed many different ways if testing solely for EIB. The goal is to mimic the activity or sport that causes the patient's symptoms as closely as possible.
- Free running
  - The patient runs either indoors or outdoors. This can also be done in a stairwell if space is limited.
  - Advantages—Most likely to induce symptoms, requires minimal cardiovascular (CV) monitoring.
  - Disadvantages—Depending on the season, difficult to control environmental factors such as temperature and humidity, and may not trigger EIB in all patients.
- Treadmill
  - Advantages—CV and pulmonary monitoring can be done during the workout. Workload can be standardized.
  - Disadvantages—Expensive equipment needed. Less likely to induce EIB because it is indoors and temperature, humidity, pollutants, etc., are more controlled.
- Bicycle ergometer
  - Advantages—Workload can be easily maintained. CV monitoring is the easiest.
  - Disadvantages—Least likely to produce EIB symptoms. Expensive equipment is needed.
- Other testing
  - Generally a clinical diagnosis and PFTs are the preferred way to diagnose EIB. Other testing can be done if there is a concern for an underlying condition.
  - Skin testing—probably the most useful because there is a strong correlation between allergies and EIB/EIA.
  - Computed tomography (CT) of sinuses if there is a concern for chronic sinusitis causing symptoms.
  - Chest x-ray—may show signs of underlying lung disease.
  - Echocardiogram—if a CV abnormality is a possible diagnosis.

- Special testing
  - At present, the International Olympic Committee (IOC) requires prior notification for the use of a beta-2 agonist. The notification must be accompanied by objective evidence that justifies the need for the medication. The IOC accepts the following tests and the associated decrease in FEV_1 for diagnosing EIB and allowing an athlete to use a beta-2 agonist: eucapnic voluntary hyperpnea (EVH) test (10%), exercise challenge test (10%), or hyperosmolar aerosis test (15%). EVH testing is done using dry air containing 5% carbon dioxide. Hyperosmolar aerosis can be done using either hypertonic saline or inhaled powdered mannitol (not available in the United States). These tests are called bronchial provocation tests, meaning a substance is inhaled to try and create bronchospasm.

Differential diagnosis/confounding factors:

- Vocal cord dysfunction (VCD)
  - Vocal cord dysfunction can produce respiratory symptoms whenever the ventilatory rate rises. Usually have inspiratory wheezing and/or stridor as opposed to EIB and asthma, which primarily produces expiratory wheezing. The stridor in VCD occurs because of paradoxical closure of the vocal cords. Patients complain of difficulty "getting air in" and difficulty breathing. VCD is frequently misdiagnosed as asthma and warrants special consideration in the diagnosis of EIB.
  - The diagnosis of vocal cord dysfunction is often made clinically. Flow-volume loop is performed while the patient is symptomatic; a flattening of the inspiratory loop can be seen. Laryngoscopy, which allows visualization of vocal cord adduction on inspiration (classically seen as adduction of the anterior two thirds of the vocal cords with a posterior diamond-shaped chink remaining open), is considered to be the gold standard of diagnostic testing for VCD. Treatment is reassurance, education, and speech therapy.
- Other confounding factors
  - Many other diseases can produce the symptoms that are seen in EIB.
  - These include general deconditioning, cardiac disease that produces shortness of breath, other pulmonary disease, and McArdle syndrome (glycogen storage disease that causes muscle phosphorylase deficiency).

Prevention and treatment:

- Pharmacologic
  - **Inhaled beta-adrenergic agonists:** Prevention and treatment can be achieved by using a medication such as albuterol inspired about 15 to 30 minutes prior to exercise. This can often prevent or reduce the symptoms of EIB. Remember to always dispense a spacer with the albuterol so the patient receives the maximum delivery of medication (in both pediatric and adult populations). Only albuterol in an aerosolized form has been shown to be effective in EIB and asthma (Fig. 31-1).
  - **Leukotriene antagonists:** Can be added as a second-line medication or occasionally as monotherapy. Not as effective in those individuals with isolated EIB, but may aid in those athletes that have underlying asthma.
  - **Inhaled corticosteroids:** Not effective in isolated EIB, but the mainstay of treatment of persistent asthma. All athletes that have persistent asthma and have trouble with exercise should be on an inhaled steroid.
  - **Antihistamines/intranasal steroids:** Both will help those athletes that have underlying allergic rhinitis.
  - **Other medications:** Cromolyn can be used as an adjunctive treatment for asthma. Not used as much because of the effectiveness of inhaled corticosteroids. Theophylline can be used to control asthma; however, because of the side effect profile (nausea, diarrhea, arrhythmias, seizures, interactions with other medications), it is rarely used anymore. Patients on theophylline usually have poorly controlled asthma, and any physical activity should be closely monitored.
  - **See Table 31-1 for a list of governing bodies and their rules for pharmacologic treatment of asthma.**
- **Nonpharmacologic treatment and prevention**
  - Conditioning—may help to reduce the severity of the EIB.
  - Patients can be coached to "run through" their EIB. This means that the athlete continues to exercise while having symptoms and some patients can push through the transitory symptoms and continue to perform their sport.
  - Short burst of vigorous exercise (e.g., wind sprints) may extinguish EIB and induce short-term refractoriness. Attributed to endogenous release of prostaglandins.
  - Warming up prior to activity induces bronchodilation and refractoriness to EIB.
  - Cooling down after strenuous exercise decreases EIB symptoms postexercise.

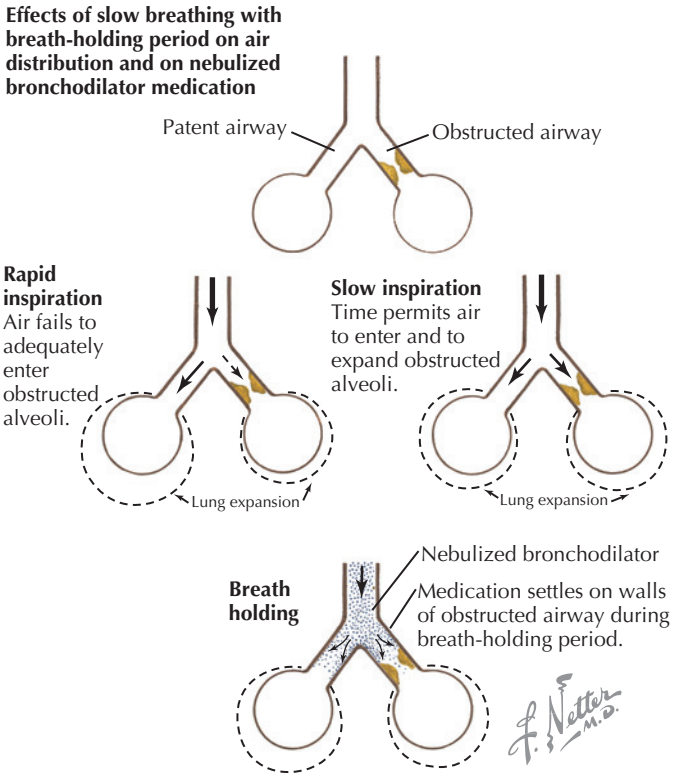

Figure 31-1 Bronchospasm.

Table 31-1 GOVERNING BODIES FOR PHARMACOLOGIC TREATMENT OF ASTHMA

Governing body	Policy on EIB/EIA	Medications
NCAA	Need a written prescription from doctor for the medication.	Albuterol is allowed in aerosolized form only.
International Olympic Committee	Need a positive exercise challenge or BPV test (Bronchial Provocation Test) Need a therapeutic use exemption (TUE) form for medications.	All beta-2 agonists and their D- and L-isomers are prohibited. Exceptions (with a valid TUE) are formoterol, albuterol/salbutamol, salmeterol, and terbutaline.

EVH, eucapnic voluntary hyperpnea.

- Avoid hyperventilation.
- Cold weather may exacerbate EIB, so dressing appropriately may help. Also, in cold weather a scarf may help retain warmer and more humid air, thus reducing EIB symptoms.
- Avoid exercising in areas that have high pollen counts or heavy pollution.
- Avoid vigorous activity when the patient has a cold, or when allergies are not well-controlled.
- Use nasal breathing (as opposed to mouth breathing) to help warm air when exercising.

CHOLINERGIC URTICARIA

Definition: Cholinergic urticaria is the name given to hives that are precipitated by an increase in core body temperature. Also referred to as generalized heat urticaria.

Epidemiology: Cholinergic urticaria is believed to account for 5% of all cases of chronic urticaria and approximately 30% of all physical urticaria. About 15% of the general population will experience at least one episode in their lifetime. Typical onset is 2nd or 3rd decade of life. Familial cases have been reported but are rare (all affected patients in these cases were males with father-to-son transmission). There may be a slight predominance in male gender.

Triggers: Include exercise, strong emotions, and bathing in hot water (essentially any trigger that causes a rise in core body temperature may provoke an onset of cholinergic urticaria). Ingestion of spicy foods or hot foods can also be a trigger. All these factors lead to increased sweat production, which may have some bearing on the pathogenesis of the condition.

Pathogenesis: Exact mechanism not known, but thought to be due to an abnormal cutaneous response to the presence of cholinergic agents, releasing mediators that cause an urticarial response. Studies have shown elevated plasma histamine in these patients.

Clinical signs and symptoms: Classic appearance is that of numerous punctate wheals (1 to 3 mm) surrounded by large flares. As the response progresses, the flares may coalesce to form large areas of erythema. The wheals typically begin on the trunk and neck and spread distally to involve face and extremities (Fig. 31-2). Patients will often have sensations that can include itching, burning, or tingling. Can progress to include systemic symptoms such as hypotension, angioedema, and bronchospasm (this is rare). Can also have signs of cholinergic stimulation such as lacrimation, salivation, and diarrhea (also rare).

Testing: The presence of classic lesions in the context of a typical inciting trigger is often enough to suggest a diagnosis, but confirmatory testing can be attempted. Provocation testing:

- Classically a methacholine injection should be positive in patients with cholinergic urticaria. This is done by an intradermal injection of methacholine in saline, and a positive test produces hives around the injection site. However, this test is only positive in about one third of patients who do have cholinergic urticaria.
- Can also conduct specific provocation testing to provoke a response. This would include having the patient mimic the activity (e.g., exercising, eating certain foods) to try and produce a reaction, including exercising in a warm room.
- Can also try and raise the core body temperature by having the patient partially submerged in a hot water bath at 40° C until the body temperature has been raised 0.7° C. The appearance of generalized urticaria confirms the diagnosis. Note: Aquagenic urticaria may be precipitated by this procedure, but those wheals should only occur in submerged portions of the skin.

Treatment: Nonpharmacologic: Identification and avoidance of known triggers is the first step in controlling cholinergic urticaria. Avoid strenuous exercise in hot weather. Avoid bathing in overly hot water. **Pharmacologic:** Antihistamines are the treatment of choice and, of those, hydroxyzine is the agent of choice. A low dose should be initiated and gradually increased until the urticaria is controlled (typical is 100 to 200 mg divided over 24 hours).

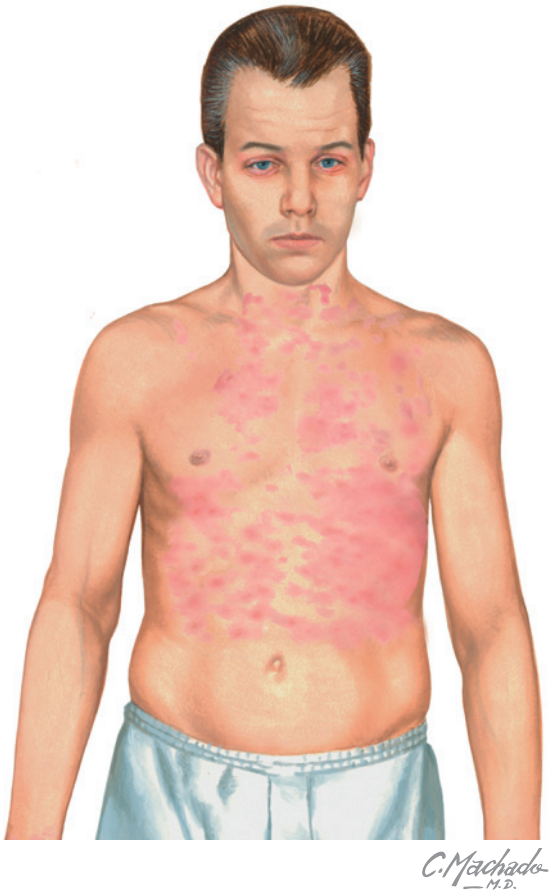

Figure 31-2 Urticaria.

EXERCISE-INDUCED ANAPHYLAXIS

General Considerations

Definition: Exercise-induced anaphylaxis is a distinct form of physical allergy in which exercise can cause a spectrum of symptoms including pruritis, urticaria, angioedema, wheezing, hypotension, syncope, and even death. In these patients, urticaria is an early sign of true anaphylaxis.

Epidemiology: Age of onset ranges from early childhood through adulthood. Seems to be more prevalent in females. About 50% of patients have a history of atopy.

Triggers: Can be triggered by any physical activity, but most commonly seen in aerobic sports and jogging. Factors that have been associated with exercise-induced anaphylaxis include menstruation, use of aspirin and nonsteroidal anti-inflammatory drugs (NSAIDs), and exposure to cold weather. Some patients have food-dependent variant where they need to ingest a certain food and then exercise to provoke an anaphylactic reaction. Common foods associated with these reactions are celery, shellfish, cheese, eggs, chicken, hazelnuts, oranges, apples, peaches, grapes, wheat, and cabbage.

Pathogenesis: Exact mechanism unknown, but mast cells are known to play a factor because of studies that demonstrate elevations of levels in serum histamine and tryptase during attacks.

Studies have also shown mast cell degeneration on skin biopsies done after attacks. May be a priming phenomenon at work where one stimuli acts as a cofactor for the reaction to occur. The food, medication, etc., may act as the primer, and exercise then triggers the event.

Clinical signs and symptoms: Within several minutes of exercising, patients experience a prodromal phase that consists of fatigue, warmth, pruritis, and erythema. These symptoms then progress to large hives that become confluent and eventually appear as angioedema. If the patient continues to exercise, the attack progresses to systemic anaphylaxis with CV (hypotension, syncope), respiratory (wheezing, stridor), and gastrointestinal (nausea, vomiting, abdominal pain and colic) symptoms. Once the attack occurs, it lasts from 30 minutes to 4 hours. A late phase has been described that can cause headache, fatigue, and warmth lasting from 24 to 72 hours.

Testing: Exercise testing is the preferred method for diagnosis. Testing needs to be done in a controlled environment, with the appropriate medical personnel, epinephrine, and resuscitative equipment present. Vital signs and spirometry should be monitored. It is recommended that an IV be in place to both draw serum markers and administer medications. False-negative challenges are common, so may need to repeat the testing on multiple occasions.

Treatment: Nonpharmacologic: The first step in effective management is to identify and avoid and specific foods, medicines, or other triggers. May need to limit the intensity and/or frequency of exercise. Avoid exercising 4 to 6 hours after eating. Avoid exercise during extremely hot, humid, or cold weather. **Pharmacologic:** Patients should always carry self-injectible epinephrine at all times. They also need to exercise/train with a partner who can inject them with the epinephrine if necessary. Antihistamine therapy has demonstrated only partial benefits in prevention of anaphylaxis.

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Neurologic Problems in the Athlete

32

David J. Petron and Jonathan C. Crist

EPILEPSY AND SEIZURE ACTIVITY IN ATHLETES

Definition

- Seizure: a transient occurrence of signs and/or symptoms resulting from abnormal excessive or synchronous neuronal activity in the brain.
- Epilepsy: an enduring predisposition to generate epileptic seizures. Prevalence is approximately 8.2 per 1000 of the general population worldwide. Seventy-five percent of epileptics experience their first seizure before the 3rd decade of life.

Classification

Seizures can be systematically classified as focal seizures and generalized seizures.

Generalized

Overview: Seizures involve both cerebral hemispheres of the brain, are of abrupt onset, and involve alteration in consciousness.

Tonic-clonic seizures (grand mal): Occurs in all ages. May have prodrome lasting hours to days. Classically starts with tonic phase (rigid extension) lasting up to 20 seconds, followed by clonic phase (synchronous muscle jerking) lasting 1 to 2 minutes. Most last less than 1 minute. Characteristics include loss of consciousness, convulsions, muscle rigidity, and urinary but not fecal incontinence. May progress to generalized status epilepticus. Postictal phase (confusion, headache, fatigue) is common. May be followed by focal weakness or paralysis, including vision and speech function, reflecting postictal depression of the epileptogenic cortical area. This phenomenon, known as Todd's paralysis, is reversible and usually resolves within 48 hours.

Absence (petit mal): Occur generally in children. No prodrome or postical phase reported. **Typical:** Onset is acute, as is recovery, with seizure activity lasting less than 10 seconds. **Atypical:** Last greater than 10 seconds, more gradual onset and recovery. Sudden loss of awareness with associated staring, rhythmic blinking, and possibly clonic jerks.

Generalized status epilepticus: Defined as continuous seizure activity lasting 30 minutes, or as two or more discrete seizures between which consciousness is not fully regained. Seizures lasting greater than 5 minutes have a high likelihood of progressing to status epilepticus and should be treated aggressively. **This is a medical emergency. Activate emergency medical services (EMS).** Ensure adequate airway, breathing, and circulation. Attempt to obtain IV access and to prevent aspiration. Benzodiazepines are the drugs of choice for initial treatment because they are fast acting and effective. Lorazepam at a dose of 0.1 mg/kg IV over 2 minutes is considered first line. Consider metabolic etiologies. Complications include dysrrhythmias, metabolic abnormalities, hyperthermia, pulmonary edema, rhabdomyolysis, and pulmonary aspiration.

Other generalized seizures: Atonic, clonic seizures; bilateral myoclonus; eyelid myoclonia; myoclonic atonic; negative myoclonus; reflex seizures; seizures of posterior neocortex; neocortical temporal lobe seizures; myoclonic seizures.

Focal

Overview: Seizures begin in one focal part of the brain and cause symptoms appropriate to the part of the cortex in which they originate. Keep in mind that partial seizures may become secondarily generalized.

BROAD TYPES

Simple focal: May include motor, sensory, autonomic, or psychic symptoms. No alteration of consciousness occurs. May be isolated or progress to complex focal or generalized seizure. Associated with Todd's paralysis (see "Tonic-clonic seizures").

Complex focal: Most common type of seizure in epileptic adults. Characterized by focal repetitive, purposeless, complex movements (e.g., chewing, gesturing, lip smacking, snapping fingers) with alteration of consciousness. Associated with auras that represent sensory and psychoillusory phenomena. Most originate in the temporal lobe. Generally last less than 90 seconds and are associated with postictal phase. Amnesia specific to the event is common.

SPECIFIC TYPES

Focal sensory seizures, focal motor seizures, gelastic seizures, hemiclonic seizures, secondarily generalized seizures, reflex seizures in focal epilepsy syndromes, and focal status epilepticus.

Posttraumatic Seizures

Description: Type of provoked seizure following traumatic brain injury (TBI). Classified as to when exactly the seizure activity occurs.

Immediate (concussive convulsions): Occurs within the first 24 hours after TBI. Controversial classification because they are not felt to represent a true seizure but rather a fairly benign phenomenon immediately following concussion. These lack the rhythmic neuronal discharges characteristic of seizures. Usually do not require anticonvulsant therapy.

Early: Occurs within 1 week of TBI. Risk factors include young age, severity of injury, alcoholism, intracerebral or subdural hematoma. Incidence is approximately 6% to 10% but higher in groups with significant head injury. Seizure activity is usually tonic-clonic within the first 24 hours progressing to more focal symptoms after this period. Computed tomography (CT) of the head indicated. Not felt to represent epilepsy but often treated with prophylactic antiepileptic medication to minimize risk of status epilepticus and risk of secondary injury. Treatment with antiepileptic medications does not affect the risk for posttraumatic epilepsy.

Late: Occur greater than 1 week after TBI with most occurring before 2 years after injury. Risk factors: early posttraumatic seizures, severity of injury, age older than 65 years, alcoholism, brain contusion, intracerebral or subdural hematoma. Overall incidence is approximately 2% but strongly correlated with severity of TBI. Seizures are also associated with underlying brain pathology. Seizures may recur in up to 70% and often require long-term anticonvulsant medication.

Precipitating Factors for Seizure

Idiopathic, new onset epilepsy, stress, sleep deprivation, fatigue, prenatal or perinatal brain injury, hyperthermia, metabolic (dehydration, hypoglycemia, hyponatremia, etc.), infectious (e.g., meningitis), trauma, drugs/alcohol (intoxication and withdrawal), febrile (usually occur in ages 6 months to 5 years), intracranial lesion (mass, hematoma, etc.).

Evaluation of the Epileptic Athlete

Prior to participation the physician should be familiar with certain aspects of seizure history including type of seizures (frequency, duration, manifestations), precipitating factors, postictal recov-

ery, any history of status epilepticus, current anticonvulsant use including side effects and medication adherence, and history of head trauma.

On-Field Treatment

- Standard guidelines for management of airway, breathing, and circulation should be followed.
- Assist the patient to the ground and clear the area of potential hazards.
  - Do not restrain the athlete.
  - Rolling the athlete to his or her side while he or she is convulsing may lead to injury. Wait until the seizure is over before attempting this.
- Do not place anything in the athlete's mouth, especially fingers. The mouth guard may be removed if this can be done safely.
- If there is any concern for status epilepticus, activate EMS for transport to a medical facility.
- If this was a first seizure or atypical for seizure history, further workup including imaging is indicated.

Epilepsy and Sports

- Points to consider:
  - Does the specific type of seizure disorder place the athlete at increased risk of injury (e.g., absence vs. simple focal)?
  - Will the athlete's condition place others at risk of injury?
  - How intent is the athlete on playing the sport?
- Sports participation is associated with factors that may alter seizure threshold.
  - Stress: Known risk factor for seizures and this should be addressed on an individual basis.
  - Hyperventilation: Resting hyperventilation may predispose to decreased seizure threshold. Exercise-induced hyperventilation, however, is a physiologic response and does not seem to have a negative effect on seizure threshold.
  - Alterations in drug metabolism: Studies have found no significant difference in metabolism in pre-exercise, exercise, and postexercise periods. However, if seizure activity increases, this should be considered.
  - Exercise-induced seizures
    - Although there are some cases of exercise lowering seizure threshold, this seems to be the exception. Overall, it appears that seizure frequency is decreased by aerobic activity. This decrease in frequency with activity is supported by EEG findings.
    - Prolonged exercise, as seen with endurance athletes, may alter physiologic parameters associated with seizure activity (hyponatremia, hypoglycemia, etc.).
    - Consider diagnosis of seizure with episodes of exercise-induced syncope.
- People with epilepsy are less active, less physically fit, less likely to participate in sports, and are at risk for social isolation.
- There is currently no evidence that contact sports are harmful to most athletes with epilepsy.
  - There is evidence to suggest that severe head injury may cause or exacerbate preexisting epilepsy, but this does not apply to repeated mild TBI.
- Aerobic exercise should be encouraged in epileptic patients.
- Contact sports should be evaluated on an individual basis based on presumed danger the athlete may present to themselves or others.

Specific Sport Guidelines

Contact sports: No restriction unless newly diagnosed or unclear course.

Water sports: Generally permitted with proper precautions (avoid open water, wear flotation device, supervised by qualified personnel). Scuba diving, competitive underwater swimming, and diving prohibited with active epilepsy though may be considered after prolonged seizure-free period (e.g., 5 years free of seizures without medication is suggested by the U.K. Sport Diving Medical Committee).

Motor sports: Discouraged with active epilepsy.

Aerobic sports: No restriction. Wear appropriate protective gear.

Sports at heights: Equestrian sports should be avoided with active epilepsy. Certain gymnastic events (e.g., high bar) should be discouraged. Skydiving, hang gliding, and free climbing should be discouraged. Pilot's license prohibited.

Shooting sports: Specific consideration for type and frequency of seizures, pattern of occurrence, and type of weapon fired.

BRACHIAL PLEXUS AND CERVICAL ROOT INJURIES ("STINGERS/BURNERS")

Overview: Transient neurologic injury accompanied by burning pain in the upper extremity believed to be the result of unilateral nerve traction or compression generally involving C5 or C6 levels or the superior trunk of the brachial plexus (Fig. 32-1). Unclear if this results from brachial plexus or cervical root injury. One of the most common injuries in sports medicine; reported by up to 65% of college football athletes in their career.

Proposed mechanisms of injury: Neck extension with lateral bending resulting in a compression injury; distraction of the shoulder from the head and neck causing nerve traction; or direct blow to the supraclavicular region at Erb's point (where the brachial plexus is most superficial), causing compressive injury. Cervical stenosis predisposes to stingers and may contribute to more complicated courses.

Presentation: Symptoms are unilateral and include transient burning pain or numbness with or without weakness from the neck/shoulder radiating into the fingers. Symptoms in lower extremity or bilateral upper extremities should prompt consideration of central cord injury. Care should be taken to distinguish this condition from transient quadriplegia, which may present with bilateral sensory changes, motor changes, or combined sensorimotor deficits. Generally not associated with neck pain or limited range of motion (ROM). Symptoms generally last seconds to minutes; if symptoms last longer or if there is

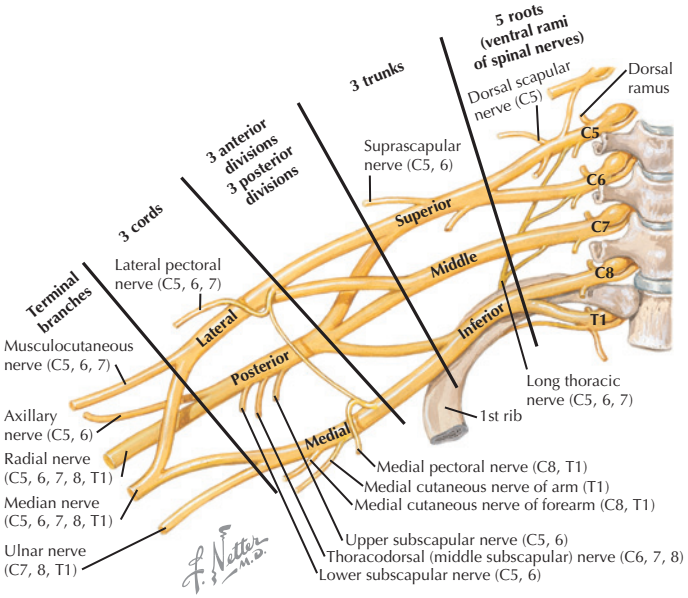


Figure 32-1 Brachial Plexus Schema.

cervical pain or limited range of motion (ROM), cervical spine magnetic resonance imaging (MRI) should be strongly considered before return to play (RTP). Up to 10% have neurologic deficit lasting from hours to weeks.

Physical exam: Athletes frequently leave the field shaking their arm and hand and may elevate the arm to decrease neural tension. Initial evaluation should focus on the cervical spine for any evidence of fracture (bony tenderness [especially midline], deformity, swelling). If initial evaluation is normal, assess ROM. Conduct a complete neurologic examination including strength, sensory, and muscle stretch reflex testing. Weakness is variable in time of onset and severity. C5 and C6 are the most common levels affected. Most common sites of weakness are deltoid, biceps, supraspinatus, and infraspinatus, resulting in weak shoulder abduction, elbow flexion, and upper arm external rotation. Percussion of Erb's point may elicit radiation of pain. A Spurling maneuver has been shown to recreate symptoms in 70% of cases; good specificity, but variable sensitivity. Contralateral exam should be normal. If bilateral symptoms present, full cervical precautions should be initiated and the athlete transported for further evaluation and imaging. Shoulder examination to rule out other injury should follow.

Diagnostics: Diagnosis based on physical exam. Radiographs usually normal. Consider MRI for prolonged symptoms or recurrent stingers. Electromyography (EMG) may be appropriate if symptoms persist beyond 3 weeks. The role of EMG is controversial with RTP decisions as the EMG findings may remain abnormal long after clinical symptoms have resolved.

Treatment: Athletes should be removed from play until completely asymptomatic. Immediate supportive treatment includes rest and use of a sling. More chronic symptoms may necessitate anti-inflammatory medications, physical therapy and, occasionally, nerve root blocks. Prevention of recurrence is central to treatment. Rehabilitation for neck and shoulder strengthening should be instituted.

Preventative equipment: To limit excessive neck extension and lateral flexion, high-riding shoulder pads and neck rolls may be considered, but efficacy is debatable. Straps connecting the helmet and shoulder pads are not recommended. Evaluate tackling technique.

Prognosis and return to play: Decision is clinical. Athletes may return to full contact when they have pain-free, full range of motion of the neck and shoulder, and normal neurologic examination, including normal symmetric strength. Longer rest periods are indicated for symptoms lasting longer than 15 minutes and for recurrent stingers in same season. Continue to evaluate for delayed weakness during the same event and for the next 2 weeks. Repetitive or prolonged episodes warrant further diagnostics. Chronic and recurrent stingers have been associated with cervical disk disease, spinal stenosis, and/or neural foraminal narrowing. Prolonged time to symptom resolution (especially greater than 3 weeks) and recurrent stingers over a short period of time have a poorer prognosis. Some advocate that if cervical foraminal stenosis is discovered, the athlete should discontinue contact sports.

CERVICAL CORD NEURAPRAXIA (TRANSIENT QUADRIPLEGIA)

Overview: Transient neurologic episode characterized by a temporary loss of motor or sensory function, or both. This is likely secondary to central cord contusion with or without associated ischemia. Most common with contact sports. Prevalence is estimated at 7 per 10,000 football participants.

Mechanism of injury: Varied mechanisms (usually hyperflexion, hyperextension, or axial loading of the neck) resulting in cervical cord contusion and decreased blood flow. Suspected to be associated with spinal stenosis and the shape of the spinal canal. Degree of stenosis may be estimated by the Torg ratio (ratio of spinal canal width to vertebral body diameter). Ratio of less than 0.80 is considered indicative of "significant cervical stenosis." This ratio has been shown to be an unreliable measure in larger athletes because of the relative vertebral body width yielding poor positive predictive value, and it has been replaced by MRI evaluation.

Symptoms: Bilateral sensory changes, motor changes, or combined sensorimotor deficits. Total body numbness may occur. Neck pain is usually absent.

Physical exam:

- **On the field**
  - Assume cervical spine injury until proven otherwise. Maintain cervical spine precautions and use cervical collar and spine board.
  - Athlete should be stabilized and transported to the nearest appropriate facility by EMS for further evaluation.
  - For football, remove the face mask, but helmet and shoulder pads should remain in place. According to the Inter-Association Task Force for Appropriate Care of the Spine-Injured Athlete, if any of the criteria below are met, the helmet and the shoulder pads should be removed maintaining cervical spine precautions:
    - If the helmet and chin strap do not hold the head securely, such that immobilization of the helmet does not also immobilize the head.
    - If the design of the helmet and chin strap is such that, even after removal of the face mask, the airway cannot be controlled or ventilation provided.
    - If after a reasonable period of time, the face mask cannot be removed to gain access to the airway.
    - If the helmet prevents immobilization for transportation in an appropriate position.
  - Standard guidelines for management of airway, breathing, and circulation should be followed.
- **Off the field**
  - Thorough cervical spine and neurologic evaluation is necessary.
  - Remember pain and tenderness over the cervical spine is not associated with transient quadriplegia and should prompt consideration of other injury such as fracture.

Diagnostics: Plain cervical spine trauma radiograph series should be obtained to rule out fracture or dislocation. Once a fracture is definitively ruled out, flexion and extension views may be obtained to evaluate for ligamentous instability. CT to rule out occult facture. MRI should be obtained to evaluate spinal stenosis, spinal cord, and associated injuries.

Treatment: Initial treatment should focus on cervical spine immobilization, medical stabilization, and transport via EMS. Steroids are frequently used in spine-injured athletes and may be started in transport if suspicion for injury exists and transportation time is prolonged. Steroids were initially used with spinal cord compression from malignancy, and their efficacy with trauma is unknown.

Prognosis and return to play: Temporary condition by definition, but extent and duration of neurologic dysfunction both factor in the overall prognosis. Average rate of recurrence for players who returned to football is 56%. Repeat episodes may be associated with cervical radiculopathy and myelopathy. Predisposition to permanent neurologic injury after episode of cervical cord neurapraxia is controversial, but case reports exist. Return-to-play decisions are made on an individual basis taking into account history of injuries, anatomical predisposition, and future risk. Discussing all possible scenarios is beyond the purpose of this text. A few examples: If the athlete is asymptomatic without documented stenosis, it is generally accepted that he/she be allowed to return to play. If athlete is asymptomatic with documented stenosis, significant controversy exists about return to play. Stenosis is more accurately defined by adequate cerebrospinal fluid around the cord as opposed to absolute canal diameter.

Opinions range from considering this a relative contraindication to an absolute contraindication. Multiple episodes without documented stenosis should prompt serious consideration of stopping contact sports.

COMPLEX REGIONAL PAIN SYNDROME (CRPS)

Overview: A regional pain syndrome of unclear etiology generally affecting the limbs. Synonyms include reflex sympathetic dystrophy (corresponds with CRPS I), causalgia (corresponds with CRPS II), posttraumatic pain syndrome, Sudeck's atrophy, and shoulder-hand syndrome. The diagnosis of CRPS is often missed. The average time between symptom onset and diagnosis in children has been reported to be 1 year.

Diagnostic criteria: Multiple diagnostic criteria published but the current criteria published by the International Association for Study of Pain (IASP) are the most widely accepted. Note that CRPS type II is associated with a definitive nerve injury whereas type I is not.

- **IASP Diagnostic Criteria for Complex Regional Pain Syndrome I** (criteria 2 through 4 must be satisfied)
  1. The presence of an initiating noxious event, or a cause of immobilization (not required for diagnosis—up to 10% of patients will not experience this).
  2. Continuing pain, allodynia, or hyperalgesia in which the pain is disproportionate to any known inciting event.
  3. Evidence at some time of edema, changes in skin blood flow, or abnormal sudomotor (sweating) activity in the region of pain.
  4. Diagnosis excluded by the existence of other conditions that would otherwise account for the degree of pain and dysfunction.
- **IASP Diagnostic Criteria for Complex Regional Pain Syndrome II** (all criteria must be satisfied)
  1. The presence of continuing pain, allodynia, or hyperalgesia *after a nerve injury*, not necessarily limited to the distribution of the injured nerve.
  2. Evidence at some time of edema, changes in skin blood flow, or abnormal sudomotor (sweating) activity in the region of pain.
  3. This diagnosis is excluded by the existence of other conditions that would otherwise account for the degree of pain and dysfunction.
- Mean age for adults is 36 to 46 years with 60% to 80% of patients being female.
- Children ages 9 to 15 years are most commonly affected, at a girl-to-boy ratio of 3:1.

Presentation: Pathophysiology is unclear. Common associated precipitating conditions include immobilization, fracture, strains/sprains, minor trauma, postsurgical, and contusion or crush injury. In some cases, no initiating event is found. A deviation from the normal course of recovery may be the first indication of CRPS. Symptoms include neurogenic pain (especially "burning pain"), allodynia (e.g., minimal stimulus such as bed sheets over affected area cause severe pain), hyperesthesia, asymmetry of color/temperature, increased sweating of the affected limb, edema, sensory deficits, stiffness, weakness, night pain, and skin mottling (especially after showers). Stressors in the lives of pediatric patients with CRPS may play a greater role than compared to their adult counterparts. Lower extremity involvement is more common in children at a ratio of 5:1 whereas upper extremity involvement is more common in adults.

Physical exam: Pain assessment is of great importance. General exam should include range of motion and a thorough neurologic examination. Specific testing for unilateral weakness, skin temperature changes, allodynia, edema and sudomotor changes should be included. The key to the exam may be in comparing the affected and unaffected limbs.

Diagnostics: The diagnosis of CRPS is clinical. No objective test is needed to make this diagnosis. Nerve conduction velocities and electromyography are advocated by some to objectively evaluate nerve function. Additional studies including radiographs, MRI, and bone scans are generally normal and should be used only to rule out other diagnoses. More chronic cases may display radiographic changes associated with atrophy, osteopenia, or osteoporosis.

Treatment: Goal should be to improve function. A multidisciplinary approach including a pain specialist is recommended. If multidisciplinary resources are not available, sports physicians may design a home program consisting of desensitization exercises (rubbing affected body part with various stimuli such as washcloth, toothbrush, shaving cream, hot and cold), stretching and strengthening exercises. Initial exercise of short duration (few seconds) and progress through tolerated discomfort for increasing periods. Serial follow-up is important, especially initially. Psychological support is recommended if pain lasts longer than 2 months. Rehabilitation is the core treatment. Additional therapeutics should be aimed at facilitating therapy. Many pharmacologic therapies have been incorporated into the treatment of CRPS, including antidepressants, antiepileptic medications, opioids, topical analgesics, NMDA antagonists, bisphosphonates, etc.; rotating schedules should be employed. Interventional pain management (e.g., epidural anesthesia, sympathetic nerve blocks, neurostimulation, etc.) is common.

Prognosis and return to play: Prognosis and return-to-play decisions are based on individual cases and vary widely; generally a more prolonged course is associated with poor prognosis. Return to play should not be the primary goal of treatment with pediatric athletes. Rather, symptom management and activities of daily living should be prioritized. Studies have reported that 50% of pediatric athletes with CRPS will return to competitive sports. Be mindful of the fact that this presentation may offer the pediatric athlete a way out of competitive sports without parental disapproval.

AXILLARY NERVE INJURY

Overview: The axillary nerve is derived from the posterior cord of the brachial plexus at the C5 or C6 level with occasional contribution from C4. It travels across the anterior inferior aspect of the subscapularis muscle before entering the quadrilateral space and dividing into two branches. The anterior branch passes around the surgical neck of the humerus; posterior branch travels adjacent to the inferior aspect of the glenoid rim before dividing into the upper lateral brachial cutaneous nerve and the nerve to the teres minor (Fig. 32-2). The quadrilateral space is bordered by the long head of the triceps medially, the humerus laterally, teres minor superiorly, teres major and latissimus dorsi muscles inferiorly, and the subscapularis anteriorly. Branches:

- Anterior branch: motor innervation to the deltoid.
- Posterior branch: motor innervation to the teres minor.
  - Superior lateral cutaneous nerve: sensory innervation over the inferior portion of the deltoid (upper, lateral arm).
- Uncommon nerve injury, representing less than 1% of all nerve injuries.

Common sports for injury: baseball, football, hiking, hockey, martial arts, rugby, volleyball, and wrestling.

Mechanism of injury: Most commonly incurred with other brachial plexus injuries.

- Traumatic: Direct (e.g., contusion to anterolateral deltoid or humeral fracture) or indirect (e.g., shoulder traction with dislocation).
- Compression: Quadrilateral space syndrome. This is a rare condition characterized by compression of the posterior hu-

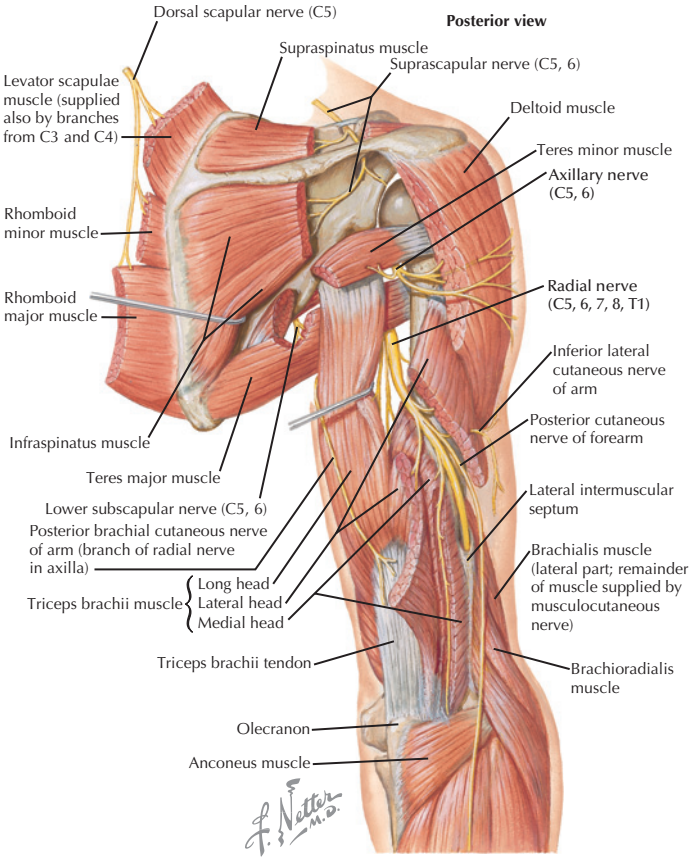


Figure 32-2 Scapular, Axillary, and Radial Nerves.

meral circumflex artery and axillary nerve as they pass though the quadrilateral space. Compression is believed to be caused by fibrotic bands within the space or by hypertrophy of the muscles that form the borders but the exact etiology is unclear.

- Iatrogenic (e.g., rotator cuff surgery).

Symptoms: Variable. The athlete may not complain of frank weakness. Possible symptoms include early fatigue or weakness especially with overhead activities or abduction. The athlete may notice numbness at the lateral upper arm. Night pain is frequently reported.

Differential diagnosis: Cervical pathology (usually C5/C6), rotator cuff injury, vascular compression, suprascapular nerve injury.

Physical exam: Visual inspection may reveal deltoid or teres minor atrophy with late presentation. Examine the neck, shoulder, and arm. Range of motion and strength in all planes should be tested. Palpate the deltoid for contraction during the initiation of abduction. Be sure to evaluate all three heads of the deltoid because nerve injury may not be uniform throughout. Weakness may occur with external rotation, abduction, and/or forward flexion. Evaluate sensation in the upper lateral arm.

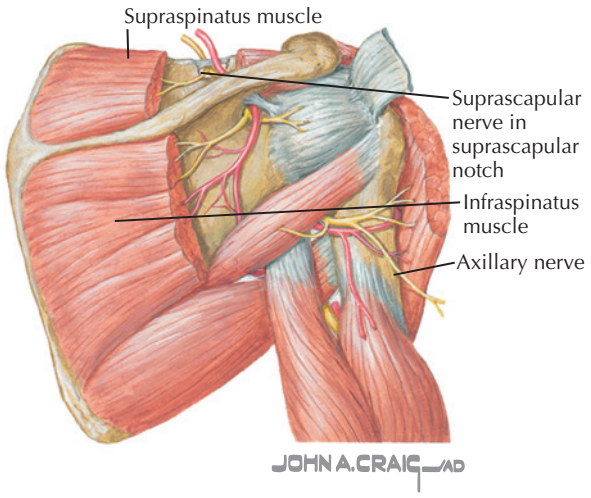
Diagnostics: Radiographs should be obtained to assess for fracture of the humerus. Cervical spine films assess bony cervical pathology. MRI may reveal indirect indicators of nerve injury (e.g., fat and water composition in muscle). Nerve conduction velocities and EMG obtained at least 3 weeks after the injury can help confirm the diagnosis. These may be repeated at 3 months if no clinical improvement.

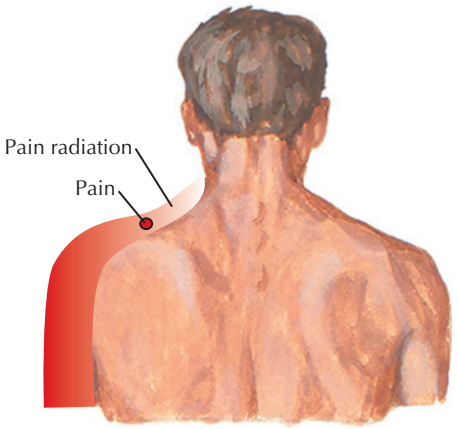
Treatment: Depends on the underlying etiology and injury to surrounding structures. Address underlying shoulder dislocation and/or fracture. Conservative treatment includes rest and physical therapy for active and passive range of motion and strengthening. Electrical stimulation of the deltoid is an optional treatment. Indications for surgical consultation include a symptomatic patient with no clinical, EMG, or nerve conduction velocity evidence of recovery by 3 to 6 months after injury; penetrating injury; or iatrogenic cause. Surgery should occur within 6 months of injury because some studies have demonstrated poorer prognosis with delayed intervention.

Prognosis and return to play: Full recovery with nonoperative treatment is expected in 85% to 100% of cases within 12 months if they are associated with dislocation or fracture. Various case series describing injuries from direct contusion have a poorer prognosis with respect to recovery of deltoid function, but a good number of these patients returned to contact sports despite deltoid paralysis. Return-to-play decisions depend on the associated injuries involved, but full shoulder range of motion and good strength are recommended.

SUPRASCAPULAR NERVE INJURY

Overview: The suprascapular nerve originates from the superior trunk of the brachial plexus (C5 and C6 with variable contribution from C4). It travels laterally across the posterior triangle of the neck, through the suprascapular notch and under the superior transverse scapular ligament before sending off branches to the supraspinatus. The nerve then travels around the spinoglenoid notch to enter the infraspinatus fossa, where it divides to supply the infraspinatus (Fig. 32-3). Innervates the supraspinatus and infraspi-





Compression of suprascapular nerve may cause lateral shoulder pain and atrophy of supraspinatus and infraspinatus muscles.

Figure 32-3 Neuropathy about Shoulder: Suprascapular Nerve.

natus with sensory branches to the acromioclavicular and glenohumeral joints. Sensory innervation of the proximal-lateral arm is reported in 15% of patients. The nerve is the most frequently injured peripheral branch of the brachial plexus in athletes.

Common sports for injury: volleyball, baseball, basketball, tennis/racquetball, football, weightlifting, hiking, wrestling, cycling, ballet dancing.

Mechanism of injury:

- Trauma: Direct (e.g., scapular or clavicular fracture, shoulder dislocations, penetrating trauma) and indirect (e.g., traction or repetitive overuse).
- Compression: Usually via surrounding ligaments or mass lesions (e.g., cyst lipoma, fibrous band). Overhead activities such as throwing lead to greater degrees of friction, traction, and compression. Also implicated are external rotation, cross-body adduction, and forward flexion. Proposed "sling effect" refers to friction of the nerve against surrounding structures of the suprascapular notch with depression and retraction, or hyperabduction, of the shoulder.
- Iatrogenic

Symptoms: Include dull pain at the posterior aspect of the shoulder exacerbated by overhead maneuvers and/or weakness of the affected shoulder, especially external rotation and abduction. Atrophy present in up to 80% of patients (see Figure 32-3). Onset is usually insidious but may follow acute event. Lesions at the spinoglenoid notch often present with asymptomatic atrophy of the infraspinatus only, whereas lesions at the suprascapular notch often have atrophy of both infraspinatus and supraspinatus.

Differential diagnosis: Cervical spine pathology, brachial plexopathy, biceps tendonitis, adhesive capsulitis, impingement syndrome, rotator cuff and intra-articular glenohumeral pathology.

Physical exam: Visual inspection may reveal atrophy of the infraspinatus or supraspinatus. Atrophy of the infraspinatus better visualized from behind and above. Supraspinatus atrophy may be difficult to visualize. Physical exam may reveal tenderness at site of injury, and cross-arm adduction may increase pain. Physical exam findings are similar to impingement syndrome. Include thorough neurologic evaluation including neck and upper extremity specifically evaluating external rotation and abduction.

Diagnostics: Often considered a diagnosis of exclusion. May try injection of a local anesthetic into the suprascapular notch for diagnostic purposes but this is nonspecific. Radiographs of the shoulder and possibly cervical spine are usually normal. An anteroposterior radiograph directed caudally at 30 degrees or a Stryker notch view may be useful to assess the shape of the suprascapular notch. MRI is considered the optimal imaging modality for evaluating suprascapular nerve palsy, because it should reveal both mass lesions and other rotator cuff or soft tissue pathology. Ganglion cysts causing suprascapular nerve compression are often associated with labral tears. Ultrasound may also be useful but is operator dependent. Nerve conduction velocities and EMG may confirm diagnosis and/or exclude other pathology but results may not support clinical findings.

Treatment: Treatment and prognosis depend on the underlying etiology. Initial treatment is conservative and includes avoidance of aggravating activities, anti-inflammatory medications, and rehabilitation for stretching and strengthening of surrounding structures. Structural lesions (e.g., labral pathology) correlating with clinical symptoms should be treated operatively. Nonoperative treatment of ganglion cysts has a high failure rate and cysts are frequently drained surgically. Occasionally, exploration and decompression of compressive lesions or bands may be required. If symptoms are not caused by known structural lesion or if atrophy continues for more than 6 months, surgical exploration should be considered. Follow-up EMG may aid in the decision to proceed with surgical options.

Prognosis and return to play: The natural history of the injury varies greatly but resolution of symptoms in the absence of a mass lesion is expected within 6 to 12 months of the diagnosis. The patient may continue his or her chosen sport at a competitive level despite muscle atrophy if appropriate strength permits safe participation. Return-to-play decisions are individual and activity specific.

LONG THORACIC NERVE INJURY

Overview: The long thoracic nerve arises from the anterior branches of C5 through C7. Twenty percent have contributions from intercostal nerves. At 22 to 24 cm in length, it descends posteriorly to C8 and T1 rami, passes beneath the clavicle and continues distally on the external surface of the serratus anterior (Fig. 32-4). This nerve innervates the serratus anterior, which protracts and rotates the scapula as well as stabilizing the scapula in abduction.

Common sports for injury: Tennis/racquetball, backpacking, archery, basketball, bodybuilding, football, golf, gymnastics, martial arts, hiking, shooting, volleyball, and wrestling.

Mechanism of injury:

- Traumatic: Direct (e.g., contusion to shoulder or lateral thorax) and indirect (e.g., stretching of the nerve can occur with the neck turned to the contralateral direction and the arm raised overhead or shoulder depression in conjunction with contralateral neck bending).
- Compression: Multiple possible areas for compression by surrounding structures, crutches, or inflamed bursa.
- Iatrogenic (e.g., postoperative).

Symptoms: Classic winging of the scapula or popping of the scapula during movement secondary to compromised glenohumeral bio-

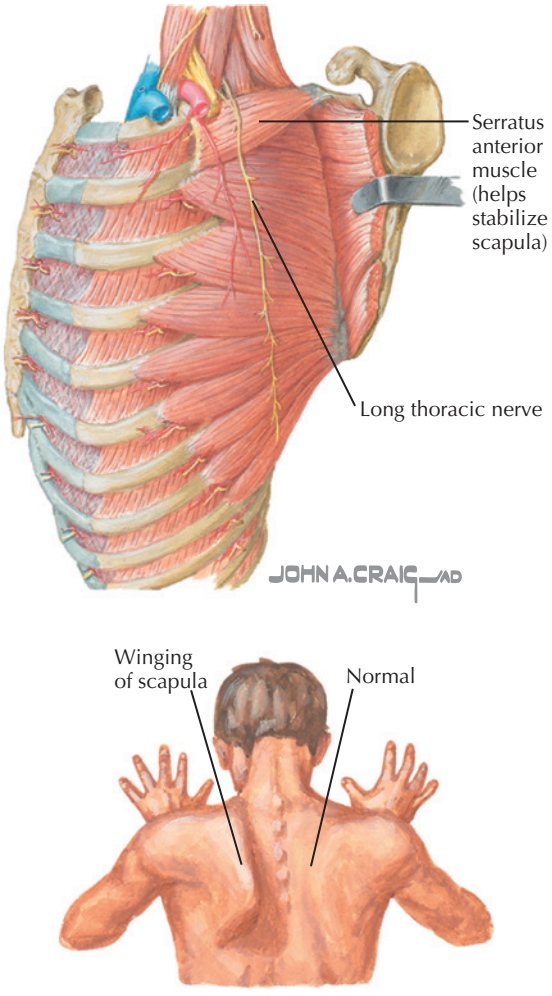


Figure 32-4 Neuropathy about Shoulder: Long Thoracic Nerve.

mechanics. Others may complain of shoulder, neck, and/or scapular area pain lasting up to a few weeks followed by insidious weakness with overhead activities or forward elevation.

Differential diagnosis: Cervical disc disease, rotator cuff pathology, brachial neuritis (Parsonage-Turner syndrome), adhesive capsulitis, glenohumeral instability or arthritis, acromioclavicular joint arthritis, thoracic outlet syndrome, spinal accessory nerve injury.

Physical exam: Visual inspection will reveal winging of the affected scapula exaggerated by forward elevation or pushing off a wall (see Fig. 32-4). Atrophy of the serratus anterior may be visible. Winging is associated with both long thoracic and spinal accessory nerve injuries. Winging associated with spinal accessory nerve injury is generally not exaggerated by forward elevation as is seen with long thoracic etiology. Physical exam reveals weakness with forward elevation often limiting movement to 110 degrees. Altered scapulohumeral rhythm may be found.

Diagnostics: Considered a clinical diagnosis. Plain radiographs generally normal. CT and MRI generally not useful unless ruling out other pathologies. Nerve conduction velocity and EMG may confirm the diagnosis and track recovery. Include the spinal accessory nerves in the EMG.

Treatment: Conservative treatment includes rest, reassurance, anti-inflammatory medications or medications for neurogenic pain, maintaining range of motion, and strengthening of surrounding structures. Bracing may be considered for improved function or pain control but has not been highly effective. Indications for surgical consultation include symptoms persisting beyond 1 year with no improvement on EMG and iatrogenic injury.

Prognosis and return to play: Isolated atraumatic long thoracic nerve palsy generally resolves in 1 to 2 years. Return-to-play decisions should be individualized and based on the athlete's strength and the demands of the sport.

SPINAL ACCESSORY NERVE INJURY

Overview: The spinal accessory nerve is a pure motor cranial nerve. It enters the foramen magnum and then exits the jugular foramen before passing through the upper third of the sternocleidomastoid. The nerve then assumes a subcutaneous course in the posterior cervical triangle to the trapezius. This nerve provides motor innervation to the trapezius and sternocleidomastoid (SCM).

Common sports for injury: Hockey, backpacking, lacrosse, wrestling, and martial arts.

Mechanism of injury:

- Traumatic: Direct (e.g., penetrating or blunt trauma to the posterior neck) or indirect (e.g., traction).
- Compression: Mass lesions or external (e.g., backpack).
- Iatrogenic: Subject to insult during lymph node dissection, carotid endartectomy, etc.

Symptoms: Pain around the shoulder, weakness, difficulty with abduction and overhead activities, and sagging of the shoulder. Radicular pain caused by traction from the drooping shoulder may also occur.

Differential diagnosis: Long thoracic nerve injury, cervical pathology.

Physical exam: Visual inspection may reveal asymmetry of the neckline, drooping of the affected shoulder, loss of normal scapulohumeral rhythm, and "lateral" scapular winging. Atrophy of the trapezius and SCM with associated spasm of the levator scapulae and rhomboids may be present. Physical exam may reveal weakness with abduction and forward elevation and inability to shrug the affected shoulder. Winging of the scapula may occur but is generally not as severe as that seen with long thoracic nerve injury. Winging associated with spinal accessory nerve injury is not exaggerated by forward elevation as is seen with long thoracic etiology. Scapular stabilization against the back by the physician may relieve symptoms.

Diagnostics: Radiographs of the cervical spine, chest, and shoulder are indicated but usually negative. MRI only if ruling out mass lesion. Nerve conduction velocities and electromyography are generally diagnostic. Repeat studies at 6-week intervals may be helpful in assessing recovery and need for surgical intervention.

Treatment: Treatment and prognosis depend on the underlying etiology. Conservative treatment consists of anti-inflammatory drugs, electrical stimulation, limitation of overhead activities, and physical therapy focusing on shoulder girdle and scapular rehabilitation. A sling may be used as needed but be wary of frozen shoulder. Braces to stabilize the scapula have not been highly effective. Indications for surgical consultation include symptoms and atrophy continuing for more than 6 months, penetrating trauma, and iatrogenic injury.

Prognosis and return to play: Injuries caused by blunt trauma typically recover in less than 1 year with conservative treatment. With injury caused by penetrating trauma or laceration, there is improved prognosis if surgical intervention is not delayed more than 6 months. Return-to-play decisions are individual and activity specific.

RADIAL NERVE INJURY

Overview: The radial nerve originates from the posterior cord of the brachial plexus (C5 to T1). It runs with the deep artery before passing into the cubital fossa and descending between the brachioradialis and brachialis. At the level of the lateral epicondyle, it branches into the superficial and deep branches (Fig. 32-5). The deep branch courses around the neck of the radius and enters the posterior compartment, terminating as the posterior interosseous, which continues under the supinator in the radial tunnel. The superficial branch passes anterior to the pronator teres, eventually entering the dorsum of the hand. Branches:

- Forearm branches: motor innervation to the triceps brachii, anaconeus, brachioradialis, extensor carpi radialis longus.
- Posterior interosseous nerve: motor innervation to the extensor carpi radialis brevis, extensor carpi ulnaris, extensor digiti minimi, extensor digitorum, supinator, extensor indicis proprius, abductor pollicis longus, and extensor pollicis longus and brevis.
- Posterior cutaneous nerve: sensory innervation to posterior arm and forearm.
- Inferior lateral cutaneous nerve: sensory innervation to lateral arm.
- Superficial branch: sensory innervation to proximal dorsal three and a half digits and dorsal hand.

Common sports for injury: Arm wrestling, baseball, football, tennis/racquetball, weightlifting, frisbee/discus, rowing, gymnastics.

Mechanism of injury:

- Traumatic: Direct (e.g., humeral shaft fracture) or indirect (e.g., muscle strain).
- Compression: Multiple possible sites of compression. Repetitive pronation/supination increases compressive forces. More proximal lesions may occur with tourniquet use, improper use of axillary crutches, and "Saturday night compression palsy."
  - Radial tunnel syndrome—compression at the elbow.
  - Wartenberg syndrome—caused by direct trauma to or compression of the superficial branch in the forearm.

Symptoms: Symptoms about the elbow may mimic lateral epicondylitis. Of patients with lateral epicondylitis, 5% to 10% have associated radial nerve entrapment.

- Radial tunnel syndrome (RTS): Deep ache distal to the lateral epicondyle that is worse at night and after throwing. Generally not associated with sensory loss or weakness. Syndrome is controversial. May occur with racquet sports because of repetitive supination/pronation.

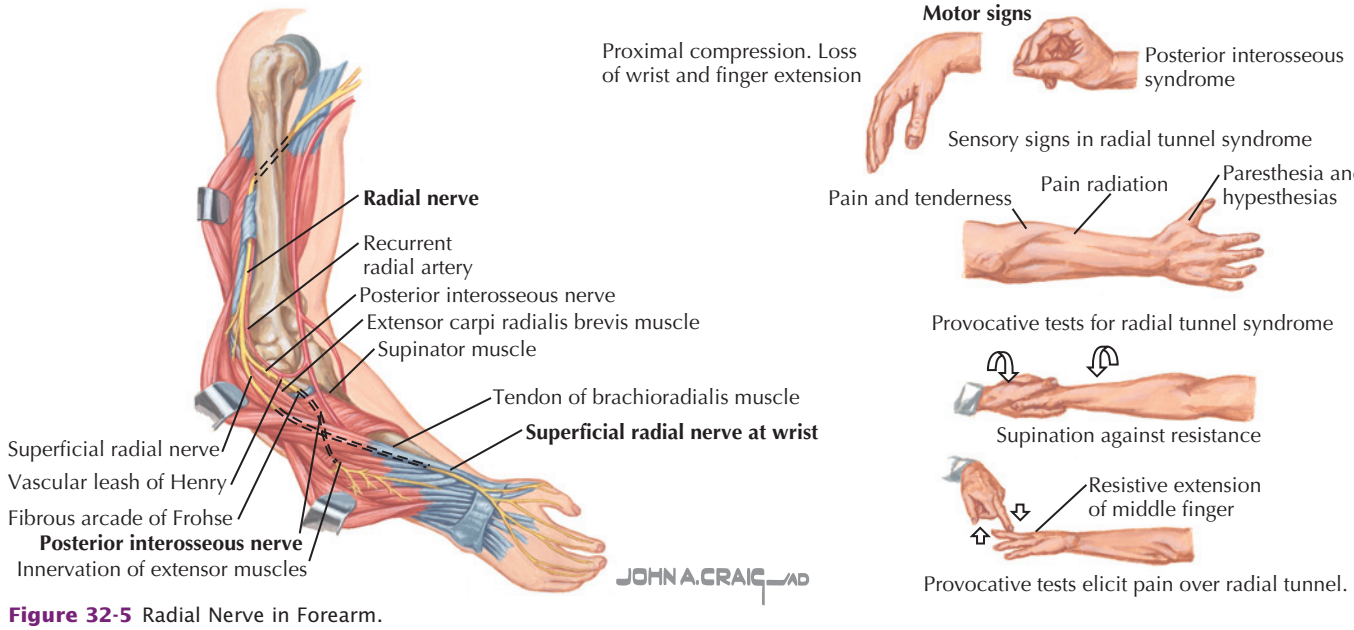


Figure 32-5 Radial Nerve in Forearm.

- The radial tunnel is the area between the lateral epicondyle and the supinator muscle, which exists as a potential space with numerous sites of compression.
- Posterior interosseous nerve compression syndrome (PIN): The same structures are compressed as with RTS but symptoms are predominantly weakness of the purely motor PIN-supplied muscles after a prodrome of lateral forearm or elbow pain. Throwers may note decreased control and velocity.
- Wartenberg syndrome: May be caused by any tight-fitting strap or band about the wrist. Symptoms include pain and decreased sensation over the dorsoradial hand, dorsal thumb, and index finger. Not associated with throwing.

Differential diagnosis: Varies depending on symptoms.

Physical exam: Full neurologic examination of the upper extremities is warranted. Specific tests (see Fig. 32-5):

- RTS: Resisted extension of the middle finger with the elbow extended, forearm pronation with the wrist flexed, elbow flexion and supination, or supination with the elbow extended all may elicit pain. Some of these tests will be positive with lateral epicondylitis. Some support the use of diagnostic blocks.
- PIN: Same as above but with weakness with extension of the wrist, thumb, and index finger. Forearm supination and grip is also weak. Symptoms may be reproduced by compression three fingerbreadths below the lateral epicondyle and can also worsen if misdiagnosed as lateral epicondylitis and counterforce brace applied over site of entrapment.
- Wartenberg syndrome: Tinel's sign should be positive. Not associated with weakness. Pseudo-Finkelstein test can be positive with forced pronation and ulnar flexion of the wrist.
- Evaluate for cervical radiculopathy and thoracic outlet syndrome.

Diagnostics: Plain radiographs are generally negative in the absence of trauma but may reveal calcifications. MRI is helpful only if a mass lesion is suspected. Nerve conduction velocities and electromyography (EMG) are usually not helpful with RTS but may be diagnostic in PIN. Negative test does not rule out entrapment. Consider testing if associated with trauma.

Treatment:

- RTS: Standard conservative treatment includes rest (may be assisted with splinting), avoidance of provocative movements, anti-inflammatory medications, physical therapy, and pain management.
- PIN: All of the above for RTS plus splinting to maintain function.
- Wartenberg syndrome: standard conservative treatment includes rest (may be assisted with splinting in supination) and anti-inflammatory medications.
- Indications for surgical consultation are lack of clinical or EMG improvement after 3 months of conservative treatment. Surgical intervention for RTS is rare.

Prognosis and return to play: Generally responds to conservative treatment. Limited case series show a good prognosis with surgical decompression.

MEDIAN NERVE INJURY

Overview: The median nerve arises from two cords of the brachial plexus, lateral (C6 and C7) and medial (C8 and T1). Occasional input from C5. It descends on the medial arm and enters the cubital fossa before passing between the heads of the pronator teres, where it gives off the anterior interosseous branch. Then it descends between the flexor digitorum superficialis (FDS) and the flexor digitorum profundus (FDP), giving off the palmer cutaneous branch before passing within the carpal tunnel to reach the hand (Fig. 32-6). Branches:

- Forearm branches: motor innervation to the pronator teres, flexor carpi radialis, and palmaris longus.
- Anterior interosseous nerve: motor innervation to the FDP (radial half), flexor pollicis longus (FPL), pronator quadratus.
- Terminal motor branches: motor innervation to the FDS, abductor pollicis brevis, opponens pollicis, flexor pollicis brevis, lateral two lumbricals.
- Palmer cutaneous nerve: sensory innervation to lateral palm.
- Digital cutaneous branches: sensory innervation to volar and distal dorsal surfaces of the radial three and a half digits.

Common sports for injury: Wrist: archery, basketball (wheelchair), bicycling, weightlifting, football, golf, and wrestling. Palmer branch: cheerleading and golf. Pronator teres: archery and baseball.

Mechanism of injury:

- Traumatic: Direct or indirect.
- Compression: Most commonly within the carpal tunnel (carpal tunnel syndrome) but occurs at multiple sites including within the pronator teres (pronator syndrome) and along the

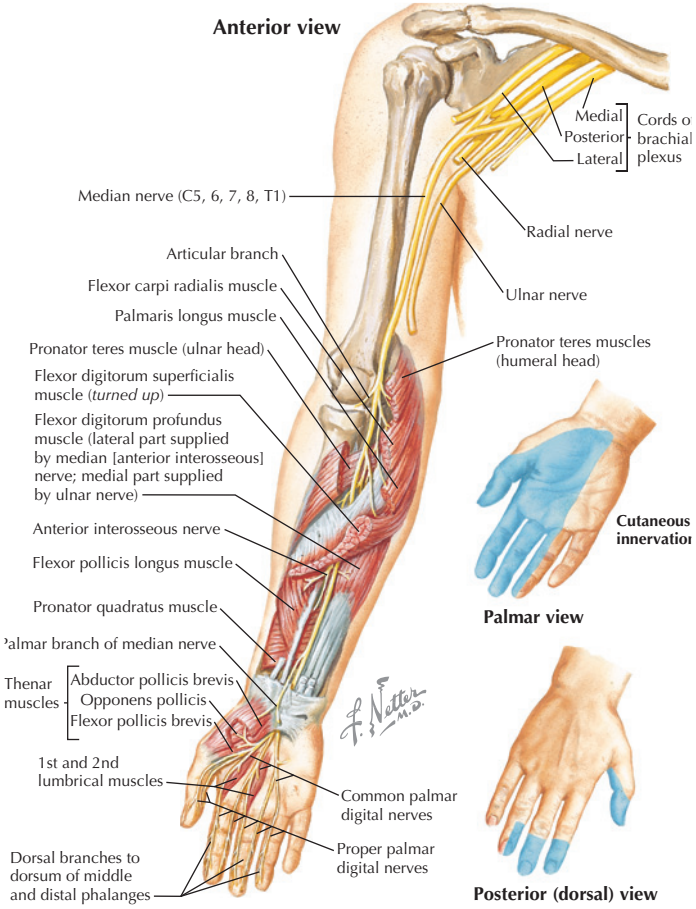


Figure 32-6 Median Nerve.

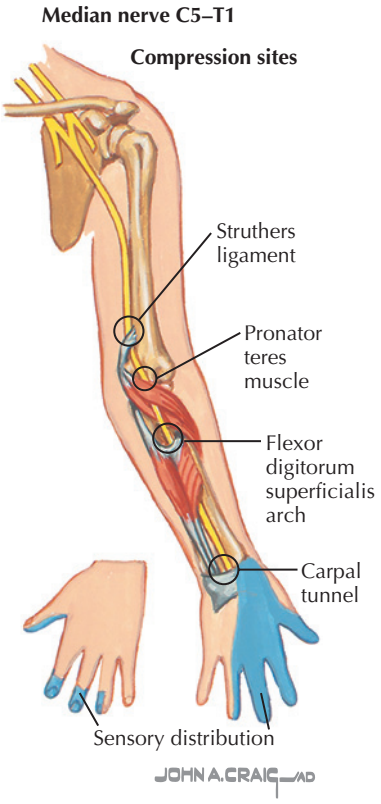


Figure 32-7 Common Sites of Upper Extremity Nerve Entrapment.

anterior interosseous nerve (anterior interosseous syndrome) (Fig. 32-7).

Symptoms:

- Carpal tunnel syndrome (CTS): Paresthesias and weakness in the radial three and a half digits of the hand. Increased symptoms with repetitive movements. Pain may radiate proximally. Nighttime symptoms are common.
- Pronator syndrome (PS): Pain in the proximal volar surface of the forearm often radiating distally that generally increases with activity. Often described as fatigue. More distal sensory symptoms may occur but are uncommon. Not typically associated with nighttime symptoms.
- Anterior interosseous syndrome (AIS): Deep constant pain in the proximal volar forearm preceding gradual weakness of the FPL and FDP. Sensory loss is not noted.

Differential diagnosis: Varies depending on symptoms.

Physical exam: Thenar muscle wasting may be present in advanced cases, usually with CTS. Thorough neurologic examination including two-point discrimination should be performed. Specific tests:

- CTS: Tinel's sign (tapping over the median nerve at the wrist, reproducing index symptoms) and Phalen's sign (wrist flexion for 60 seconds producing paresthesias in a median nerve distribution) are classic.
- PS: Pronator compression test in which compression over the pronator teres reproduces symptoms. Tenderness over the pronator is also common. Tinel's sign over the cubital fossa and reproduction of symptoms with resisted pronation in the extended elbow support the diagnosis. May cause numbness in the thenar eminence in the distribution of the palmar cutaneous branch of the median nerve (spared with CTS).
- AIS: Weakness of the FDP and FPL manifesting as inability to make an appropriate circle with the index finger and thumb.
- Evaluate for cervical radiculopathy and thoracic outlet syndrome.

Diagnostics: Plain radiographs may reveal osteophytes or bony abnormalities causing compression. MRI should be obtained if suspicion exists for compressive masses. Nerve conduction velocities and electromyography (EMG) are warranted with CTS and AIS but are unreliable with PS. New advancements using the relative sensory latency differences may allow earlier detection of CTS. Negative studies do not rule out CTS.

Treatment:

- CTS: Conservative treatment of splinting the wrist in neutral position including at night, rest, physical therapy, and anti-inflammatory medications. Corticosteroid injections into the carpal tunnel may relieve symptoms.
- PS: Rest, anti-inflammatory medications, and immobilization with elbow flexion at 90 degrees and the forearm in neutral for 3 to 6 weeks. Corticosteroid injection is debatable.
- AIS: Same as PS.
- Indications for surgical consultation include failed conservative management and progressive symptoms. Specific time periods for trial of conservative treatment are 6 months for CTS and 3 to 6 months for PS and AIS but these vary in the literature.

Prognosis and return to play:

- CTS: Prognosis is variable but generally responds well to conservative treatment within a few months with very good prognosis expected if surgical treatment required.
- PS: 50% resolution in 6 to 8 weeks with conservative treatment. Very good prognosis if surgical decompression required.

- AIS: Frequently resolves with conservative management. Good prognosis if surgical intervention required.

ULNAR NERVE INJURY

Overview: The ulnar nerve arises from the C8 and T1 roots. Contribution from C7 is not uncommon. It descends distally, just medial to axillary artery. After passing posterior to the medial epicondyle of the humerus within the cubital tunnel, it enters the anterior compartment of the forearm between the heads of the flexor carpi ulnaris (FCU). Descending between the FCU and flexor digitorum profundus (FDP), the nerve gives off the palmar cutaneous branch and then the dorsal cutaneous nerve. After passing through Guyon's canal, the nerve splits into superficial sensory and deep motor branches (Fig. 32-8). The nerve may move as much as 7 mm anterior-medially, and lengthen as much as 4.7 mm during flexion. Branches:

- Forearm branches: motor innervation to FCU and FDP (ulnar half).
- Superficial motor branch: motor innervation to the palmaris brevis.
- Deep motor branch: motor innervation to the hypothenar muscles, adductor pollicis, all interossei, medial two lumbricals, and deep head of flexor pollicis brevis.
- Dorsal and palmar sensory branch: sensory innervation to medial palm/dorsal hand and volar and distal dorsal surfaces of ulnar one and a half digits.

Common sports for injury: Elbow: baseball; tennis; bicycling; bodybuilding/weightlifting; judo, karate, and kickboxing; cross-country skiing; and wrestling. Wrist: basketball (wheelchair), bicycling, football, cross-country skiing, and snowmobiling. Flexor carpi ulnaris: weightlifting and golf. Deep motor branch: weightlifting.

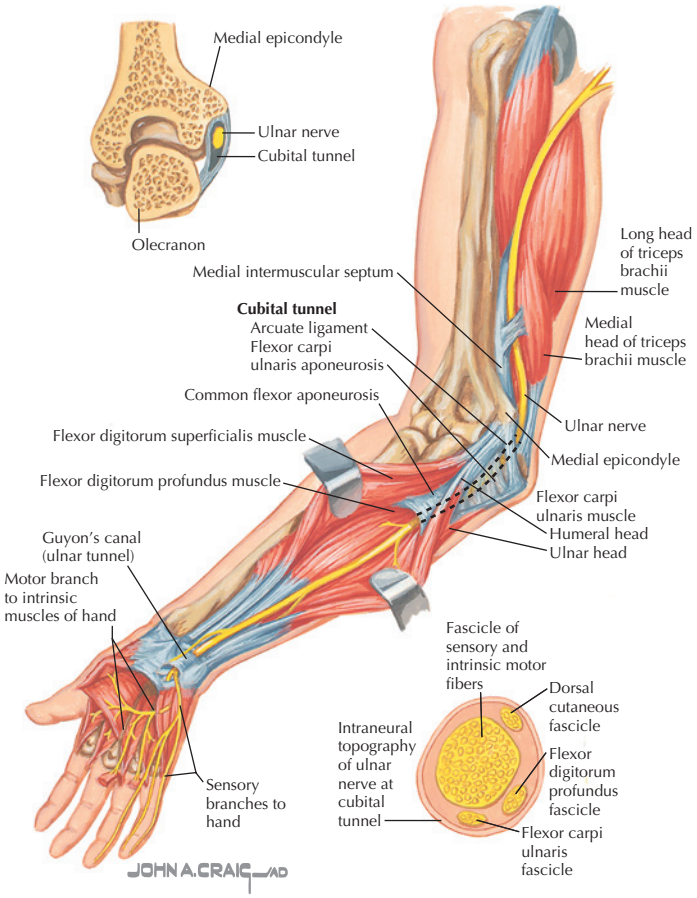


Figure 32-8 Compression of Ulnar Nerve.

Mechanism of injury:

- Trauma: Direct and indirect trauma (i.e., traction, friction).
- Compression: Common sites for compression at the elbow include the ulnar groove, below the medial epicondyle (also known as the cubital tunnel), and above the medical epicondyle (also known as the "arcade of Struthers"). Compression at the wrist usually occurs at Guyon's canal (see Fig. 32-8). May also occur with muscular hypertrophy of the FCU, ulnar artery aneurysm, lipoma, etc.

Symptoms:

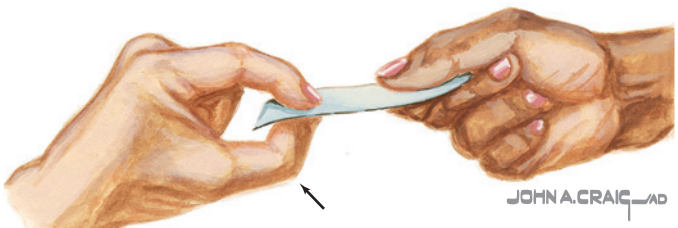
- Cubital tunnel syndrome: Common in baseball pitchers because of the large valgus stress at the elbow and repetitive flexion/extension (valgus overload syndrome). Pain at the medial joint line or paresthesias during late cocking or early acceleration may occur. Radiation to the hand is common. Snapping of the nerve may occur with flexion/extension. Symptoms may also focus more distally, mimicking ulnar tunnel syndrome. Sleeping with elbows fully flexed can increase symptoms.
- Ulnar tunnel syndrome (Guyon's canal syndrome): Also known as handlebar or cyclist's palsy because of repetitive loads on the palmar aspect of the wrist in cycling. Symptoms include paresthesias and pain in the fourth and fifth digits, decreased grip strength (40% of the grip strength is derived from ulnar nerve musculature), and pain at the volar wrist ulnar aspect. Fourth and fifth digit abduction and adduction weakness imply poorer prognosis.

Differential diagnosis: C8 and/or T1 radiculopathy, brachial plexopathy, and polyneuropathy. Other causes of wrist pain include hypothenar hammer syndrome, fracture of the hook of the hamate, ganglion cysts, ulnar carpal instability, and ulnar artery aneurysm.

Physical exam: Visual inspection for flexion contractures or valgus deformity at the elbow, muscle hypertrophy or atrophy, claw hand deformity. Palpate for a subluxing ulnar nerve as the elbow is flexed. Stress the ulnar collateral ligament (UCL) for laxity. Thorough neurologic examination including intrinsic muscles of the hand. Dorsal symptoms rule out the possibility of ulnar nerve entrapment at the wrist. Specific tests:

- Elbow flexion test: holding elbow in maximal flexion with full wrist extension for one minute to test for symptom reproduction.
- Tinel's sign: palpation of the ulnar nerve reproducing symptoms at the cubital tunnel (especially combined with elbow flexion) or Guyon's canal.
- Phalen's sign: wrist flexion eliciting paresthesias in an ulnar distribution.
- Froment's sign: weakness of adduction of the thumb (Fig. 32-9).
- Evaluate for cervical radiculopathy and thoracic outlet syndrome.

Diagnostics: Choice of imaging is based on lesion location. Plain radiographs may reveal bony changes or osteophytes about the elbow (ulnar sulcus or cubital tunnel view) or hamate fracture at



When pinching a piece of paper between thumb and index finger, the thumb IP joint will flex if the adductor pollicis muscle is weak (ulnar nerve paralysis).

Figure 32-9 Positive Froment's Sign.

the wrist (carpal tunnel view). MRI studies and angiograms may reveal occult hamate fracture and/or ulnar artery thrombosis. Nerve conduction velocities and electromyography (EMG) may help localize the injury and monitor recovery, though utility at Guyon's canal is limited because of the technical difficulty of the exam.

Treatment: Treatment and prognosis depend on the underlying etiology and injury to surrounding structures.

- Cubital tunnel syndrome: Rest, ice, anti-inflammatory medications, and physical therapy. Splinting in mild flexion (elbow pad worn in "reverse," or fiberglass volar flexion "block" splint) or an elbow pad to avoid pressure on the cubital tunnel may help.
- Ulnar tunnel syndrome: Similar to cubital tunnel syndrome treatment, plus wrist splinting in position of function with slight dorsiflexion. Corticosteroid injection into Guyon's canal may be considered but often yields only transient relief.
  - In cases of cyclist's palsy, use of padded gloves, specialized grips, altering and frequently changing hand position, and appropriate bicycle fitting may decrease symptoms.
- Indications for surgical consultation include failure to respond to conservative treatment, persistent motor weakness, intrinsic paralysis, or progression of symptoms. Surgical intervention for cubital tunnel syndrome frequently includes decompression of the nerve and/or anterior transposition for neuropathy associated with elbow deformity or subluxation of the nerve.

Prognosis and return to play: Return-to-play decisions depend on the associated injuries involved and treatment required.

TIBIAL NERVE INJURY

Overview: The tibial nerve derives from the L4 to S3 roots as part of the sciatic nerve. It branches from the sciatic nerve in the distal thigh and continues through the popliteal fossa, entering the calf between the two heads of the gastrocnemius muscle. At the medial ankle, the nerve passes into the foot through the tarsal tunnel. The tarsal tunnel is a fibro-osseous tunnel inferior and posterior to the medial malleolus, formed by the bony floor and the flexor retinaculum. Within the tunnel it splits into the medial and lateral plantar nerves (Fig. 32-10). The medial plantar nerve passes distally between the abductor hallucis and flexor digitorum brevis. The lateral plantar nerve passes between the quadratus plantae and flexor digitorum brevis. Branches:

- Direct branches: motor innervation to the semimembranosus, semitendinosus, biceps femoris (long head), plantaris, popliteus, gastrocnemius, soleus, tibialis posterior, flexor hallucis longus, and flexor digitorum longus; sensory innervation to posterolateral calf via sural branches.
- Medial plantar nerve: motor innervation to the abductor hallucis, flexor digitorum brevis, flexor hallucis brevis, and first lumbrical. Also sensory innervation to the medial sole and medial three and a half toes.
- Lateral plantar nerve: motor innervation to the quadratus plantae, flexor digiti minimi, adductor hallucis, interossei, abductor digiti minimi, and lateral three lumbricals. Also sensory innervation to the lateral sole and lateral one and a half toes.
- Medial calcaneal nerve: sensory innervation to the plantar and medial heel.

Common sports for injury: Running, hockey, hiking, skiing, ballet, and gymnastics.

Mechanism of injury:

- Traumatic: direct or indirect trauma (most commonly to the distal tibia or ankle), overuse, fractures.
- Compression: mass lesion, exostosis, accessory flexor digitorum longus, varicose veins, overpronation, rearfoot valgus deformity, muscular hypertrophy.
- Systemic disorders: rheumatoid arthritis, diabetes, etc.

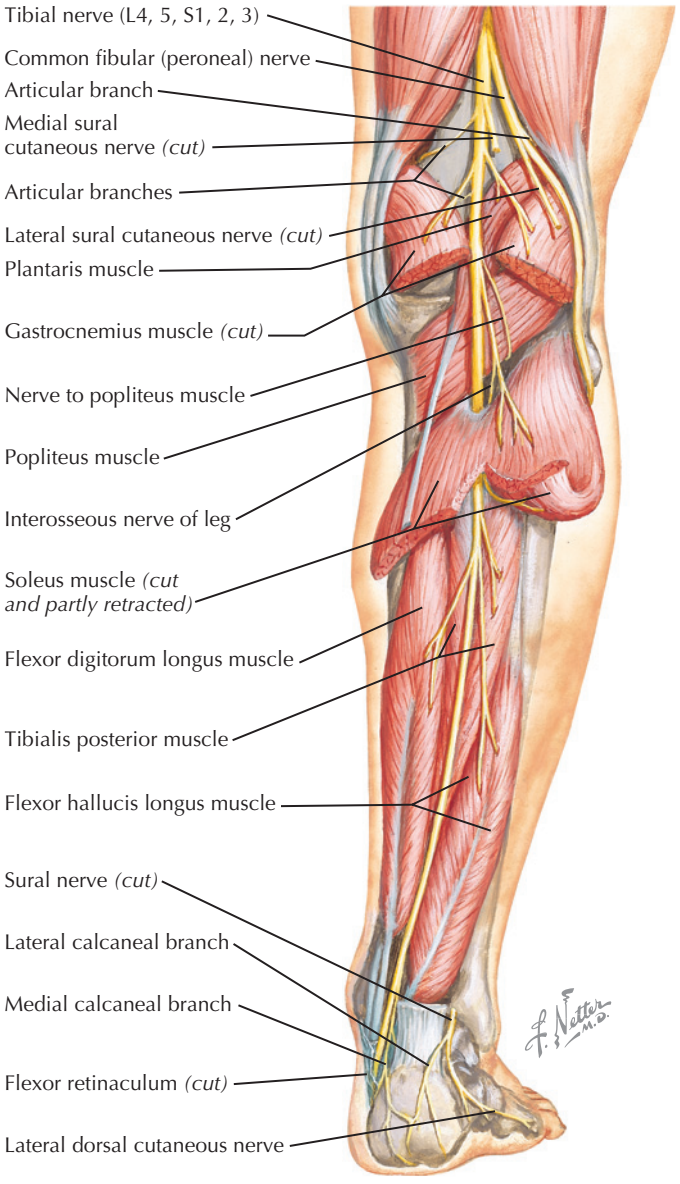


Figure 32-10 Tibial Nerve.

Symptoms: Posterior tibial nerve injury (tarsal tunnel syndrome) is the most common neuropathy involving this nerve. Compression occurs within the tarsal tunnel. Patients experience burning pain on the medial plantar foot, which is worse with prolonged standing or walking. Proximal radiation to the calf is not uncommon. Motor weakness is generally not reported, but weakness of toe flexion may occur. Occasional night pain may be reported.

Differential diagnosis: Varies depending on symptoms.

Physical exam: Visual inspection in advanced cases may reveal atrophy of intrinsic foot muscles. Physical inspection should include thorough examination of the soft tissues for evidence of mass lesion. Complete neurologic examination of bilateral lower extremities should be included. Inspect the lower back for signs of radiculopathy. Specific tests:

- Pain when the ankle is placed in extremes of dorsiflexion; Tinel's sign behind medial malleolus at the tarsal tunnel or at various entrapment sites distal to the tunnel reproducing symptoms; decreased sensation along the planter aspect of the foot; tenderness and/or mass or swelling at the tarsal tunnel; occasionally weakness with great toe plantarflexion.

- Two-point discrimination test on medial and lateral sides of the foot may localize lesions.

Diagnostics: Plain radiographs may reveal fracture or osteophytes. MRI to rule out mass lesions. Nerve conduction velocities and electromyography (EMG) may show prolonged conduction.

Treatment: Determined by the underlying cause. Conservative treatment includes rest, change in footwear or running posture, orthotic with medial support, splinting, anti-inflammatory medications, and corticosteroid injection. Indications for surgical referral include mass lesions and failed conservative treatment after 3 to 6 months.

Prognosis and return to play: Conservative treatment is successful in the majority of cases. If surgical intervention is required, approximately 80% to 90% of patients will experience improvement or resolution of symptoms. This estimate drops to 75% if the specific cause is not known.

Less common tibial nerve injuries:

- Medial plantar nerve (MPN) injury ("jogger's foot"): symptoms include burning heel pain, aching in the arch, and decreased sensation in the plantar foot behind the great toe. Compression occurs distal to the tarsal tunnel, most commonly in the abductor tunnel behind the navicular tuberosity. There may also be tenderness of the MPN at the entrance to the abductor tunnel and weakness of intrinsic foot musculature. Difficult to differentiate from plantar fasciitis.
- Lateral plantar nerve (LPN) injury: Symptoms may include decreased sensation at the lateral one third of the plantar foot. There may be weakness of the abductor digiti quinti but this is difficult to determine.

COMMON FIBULAR (PERONEAL) NERVE INJURY

Overview: The common fibular (peroneal) nerve derives from the L4 to S2 roots as part of the sciatic nerve. The fibular nerve branches from the sciatic nerve in the upper popliteal fossa before giving off a lateral sural cutaneous branch, which becomes part of the sural nerve. Traveling posterior to the fibular head, the nerve enters the peroneal (fibular) tunnel. Upon entering the tunnel the nerve splits into the superficial and deep fibular nerves (Fig. 32-11). Traveling within the lateral compartment, the superficial branch continues distally along the anterior intermuscular septum eventually piercing deep fascia at the distal third of the leg to become subcutaneous. The deep fibular nerve (DFN) travels on the interosseous membrane in the anterior compartment before crossing the distal end of the tibia and continuing under the extensor retinaculum and through the anterior tarsal tunnel (a flattened space between the inferior extensor retinaculum and the fascia overlying the talus and navicular) to enter the foot. Branches:

- Direct branches: motor innervation to biceps femoris (short head). Also sensory innervation to lateral leg.
- Lateral sural cutaneous: sensory innervation to the lateral and posterior leg.
- Deep fibular nerve: motor innervation to tibialis anterior, extensor hallucis longus and brevis, peroneus (fibularis) tertius, and extensor digitorum longus and brevis. Also sensory innervation to first web space.
- Superficial fibular nerve: motor innervation to peroneus longus and brevis and sensory innervation to dorsum of foot and toes except lateral fifth toe and first web space.

Common sports for injury: Running, bicycling, soccer, auto racing, football, ballet dancing, hockey, martial arts, surfing, horse racing, and skiing.

Mechanism of injury:

- Traumatic: Direct contusion, repetitive motion injury, or stretch injury, the latter mostly occurring where the nerve passes through the peroneus longus muscle. May occur with knee dislocations, fibular fracture, proximal tibiofibular instability, and severe ankle inversion.
- Compression: Most common at the peroneal tunnel. Also occurs with internal masses (e.g., fabella), casting, weight loss, and after prolonged bed rest or prolonged positions such as squatting, kneeling, or sitting cross-legged.
- Iatrogenic: During surgical approach (i.e., knee arthroscopy) or caused by positioning.

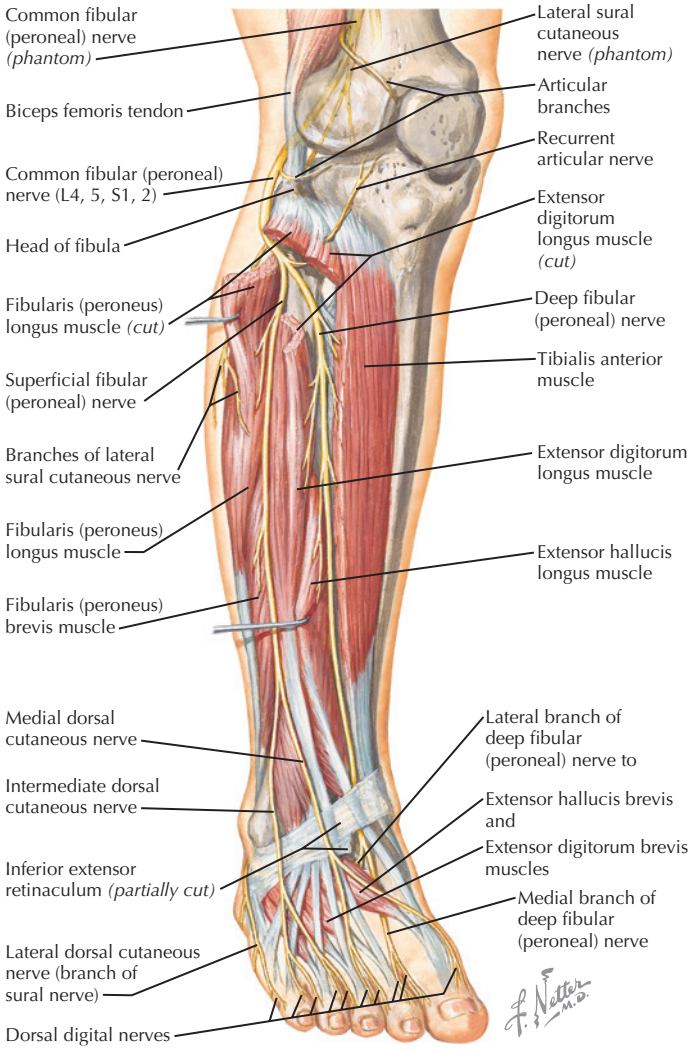


Figure 32-11 Common Fibular (Peroneal) Nerve.

Symptoms:

- Common fibular nerve (CFN) injury: Partial or complete foot drop. May be insidious or acute. This may present as tripping or falls. Lateral lower leg and dorsal foot paresthesias are common. Occurs with compression at the peroneal tunnel.
- Superficial fibular nerve (SFN) injury: Pain or paresthesia over the lateral calf, lower leg, and/or dorsum of the foot reproduced by resisted ankle dorsiflexion and eversion. Etiology usually undetermined but may be associated with fascial defect. Associated with lateral compartment syndrome.
- Deep fibular nerve (DFN) injury (anterior tarsal tunnel syndrome): Pain over the dorsomedial aspect of the foot with radiation into or numbness within the first web space. Occurs with compression of the nerve at the ankle as it passes the talonavicular joint. Associated with anterior compartment syndrome.

Differential diagnosis: Varies depending on symptoms.

Physical exam: Visual inspection in chronic cases may display atrophy in the anterior and lateral compartments of the leg. Observation of the patient ambulating may reveal steppage gate or hip hiking. Physical inspection should include thorough examination of the soft tissues about the knee for evidence of mass lesion or fascial hernia. Complete neurologic examination of bilateral lower extremities should be included. Consistent findings include difficulty heel walking, weakness with dorsiflexion, great toe extension, and weakness with foot inversion while in dorsiflexion but not plantarflexion. Forced inversion may increase pain. Sensory deficits are more common at the first web space (deep fibular nerve) than over the superficial fibular nerve and common fibular nerve distribution. Reflexes should be normal. Specific tests:

- CFN: Weakness of ankle and toe dorsiflexors and ankle eversion; hypoesthesia to touch and pain in the lower two thirds of the lateral leg and dorsum of the foot; Tinel's sign may be positive at the CFN (fibular head).
- SFN: Worsening symptoms with plantar flexion and inversion of the foot.
- DFN: Weakness of the extensor digitorum brevis muscle; sensory deficit in the first web space; ankle eversion normal; Tinel's sign may be positive at the DFN (anterior compartment, mid-distal tibia).
- Consider palpation of the muscular compartments after exercise to evaluate for compartment syndrome, especially with DFN symptoms.
- Inspect the lower back for signs of radiculopathy. Be sure to differentiate back pain secondary to abnormal gait from primary lower back pathology associated with radiculopathy.

Diagnostics: Plain radiographs to rule out fracture or compressive exostosis. MRI should rule out mass lesions. Nerve conduction velocities and electromyography are considered the gold standard with fibular nerve injuries. Compartment pressure testing may be warranted if clinically suspected.

Treatment: Conservative treatment includes avoidance of continued compression by object or position (this may include use of protective pads), rest, anti-inflammatory medications, and physical therapy. Wearing a looser shoe for DFN compression may help. Corticosteroid injections are also used. Depending on the degree of weakness, bracing with an ankle-foot orthosis may be required for foot drop and prevention of contracture. Indications for surgical referral include nerve laceration, compression by a mass lesion, and lack of clinical improvement with conservative measures. Concurrent anterior compartment syndrome with DFN lesions may require decompression.

Prognosis and return to play: Resolution should be expected within 2 to 6 months, depending on the etiology. SFN is less likely to respond to conservative treatment and usually requires surgery. If surgical intervention necessary, generally a good response is expected but this may vary with site of compression and degree of palsy.

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Headache in the Athlete

33

Kinshasa C. Morton

GENERAL PRINCIPLES

- **Headache is one of the most common disorders and symptoms reported to primary care, emergency department, and team physicians.**
- Complaint of headache accounts for 1% to 4% of primary care office and emergency department visits.
- In the general population, the prevalence of headache in a 1-year period is more than 90%, and lifetime prevalence is 93% to 99%.
- Headaches are one of the most reported pain complaints among children and adolescents.
  - In a population study of adolescents aged 11 to 21, more than 90% had experienced headaches, regardless of type, over 1 year.
  - Of children aged 4 to 17 years in the United States, 6.7% had frequent headache pain over a 12-month period.
- Various studies done with athletes have shown a headache prevalence of 35% to 50% related to participation in their sport.

Causes of Headache

- The exact cause of many headaches is the source of much debate and likely differs for each specific type of headache. Physiologic changes in the head and brain that consist of, but are not limited to, changes in neurotransmitter regulation, vascular dilation and constriction, and cranial nerve irritation causing activation of pain signals are all contributors to the development of headache. Cyclical hormonal changes and genetic predisposition are also thought to play an important role.
- Tension-type, migraine, and cluster headaches combine to comprise more than 90% of headaches experienced by the general population.
- Adults experience mostly tension-type headaches, whereas children are more likely to have migraine headaches.
  - Among children aged 3 to 18 years seen at pediatric neurologic clinics, vascular/migrainous headaches account for 52%, chronic/tension-type headaches for 21%, and unclassified headaches for 19%. Remainders are mixed tension-migraine, psychogenic, or posttraumatic.
- Of headache-caused emergency department visits:
  - Migraine and tension-type headaches account for 25% to 55% of visits.
  - Headache associated with systemic illness account for 33% to 39% of visits.
  - Headache caused by a serious neurologic condition (subarachnoid hemorrhage, intracranial mass, meningitis, hemorrhage) accounts for 1% to 16% of visits.

Classification of Headache Disorders

- **In 1988 the International Headache Society (IHS) created its first classification of headache disorders. This classification was updated and revised in 2003.**
- Headache has many causes. The IHS classification gives the physician a logical approach to create a rapid and accurate differential diagnosis.
- Headache disorders are classified as either primary, secondary, or those caused by cranial neuralgias.
  - Primary headaches are those that have no underlying cause; there are three main types: migraine headache, tension-type headache, and cluster headache.
  - Secondary headaches are those that may be attributed to some underlying pathologic condition, i.e., infectious, neoplastic, vascular, psychiatric, traumatic, drug-induced, or homeostatic changes.
  - Cranial neuralgia headaches are those that are initiated by compression, distortion, or irritation of specific cranial nerves and subsequently cause pain in the area that those nerves innervate.
- The athletic population will suffer from the same headache disorders as the general population, but may also have a predisposition to suffer from other headache disorders and subtypes because of the effects of exercise and because of participation in their specific sports.
  - Sports/exercise-related primary headaches are exercise/effort-induced migraine and primary exertional headache.
  - Sports/exercise-related secondary headaches are acute and chronic posttraumatic headache, cervicogenic headache, high-altitude headache, and diving headache.
  - Sports/exercise-related cranial neuralgia headaches are external compression headache and cold-stimulus headache.

ASSESSING HEADACHE IN THE GENERAL AND ATHLETIC POPULATION

Like all other medical evaluations, the assessment of headache begins with a thorough history and physical exam. Most patients will have a completely normal general physical and neurologic exam, so obtaining a detailed history is key. After history and physical exam have been completed, further investigation may be warranted depending on history and physical findings. This investigation may include lab work, imaging, and/or diagnostic procedures.

Headache History

- When was the first onset of headache?
- What is the frequency?
- Where is the location and is there any radiation of pain?
- What is the character of the pain? (dull, throbbing, sharp)
- What is the intensity of the pain?
- Are there any associated symptoms?
  - Nausea/vomiting
  - Photophobia/phonophobia
  - Confusion
  - Blurry vision
  - Gait disturbances
- Were there any medications taken?
- Are there any alleviating or exacerbating factors?
- Is there a prior history of headache?
- Is there a family history of headache?
- Has there been a change in the headache characteristics? (if prior history is positive)
- Is there a history of other medical issues?
- What medications is the athlete taking?

Physical Exam

- Assess for level of consciousness, orientation, alertness, mini-mental status, overall affect.
- Check vital signs to rule out hypertension.
- Evaluate head, eyes, ears, nose, and throat to assess for papill-edema. Evaluate for central nervous system abnormalities including cranial nerve testing, ptosis, pupil reactivity, head trauma.
- Conduct musculoskeletal evaluation to assess for nuchal rigidity, temporomandibular joint problems, cervical spine range of motion, areas of scalp tenderness, neck tenderness.

- Conduct neurologic examination to assess for motor, sensory, reflex response. Also include evaluation of gait.
- Check for evidence of systemic illness (assess abnormalities in the cardiovascular, respiratory, gastrointestinal [GI] systems).

Additional Investigation

Laboratory evaluation: To rule out metabolic problems. Labs that may aid in diagnosis are erythrocyte sedimentation rate, complete blood count, liver function tests, thyroid function tests, antinuclear antibodies, antiphospholipid antibodies, and drug screen. Testing should be obtained as indicated by the history and physical examination.

Imaging tests: Possibilities include cervical spine films, CT (with or without contrast), MRI, EEG, and MR angiography. Choice of tests should be individualized.

- Cervical spine x-ray (flexion/extension views) in patients with trauma/neck pain, possible fracture, instability.
- CT scan with contrast for patients with new-onset exertional headache.
  - Some do not advocate CT unless neurologic exam is abnormal.
  - May not be helpful for suspected posterior fossa lesions.
- CT better for acute bleeding, bony fracture; detects surgical lesions as well as MRI scan.
- MRI has greater tissue contrast resolution than CT.
  - Better more than 48 hours after trauma and in patients with arteriovenous malformations (AVMs) or tumor.
  - Study of choice when looking for lesions in posterior fossa.
  - Less radiation exposure compared to CT.
- According to Quality Standards Subcommittee of American Academy of Neurology, routine use of brain imaging is not warranted for adults with recurrent headaches that have been defined as migraine with no recent change in pattern, no history of seizure, and no other focal neurologic signs or symptoms.

Diagnostic procedure: Lumbar puncture (LP) to rule out subarachnoid hemorrhage from aneurysm. Detects minor leaks, which occur in 39% of patients who later have rupture. CT scans are negative in 55% of patients with minor leaks.

General Consideration

- Consider the same diagnostic evaluation in athletes as in nonathletes, except in cases of trauma with increased risk for intracranial bleed.
- Workup should be complete when diagnosis is in question. This may include, but is not limited to, evaluation for drug abuse, hypertension, vascular lesions, neoplasms, intracranial bleeding, and psychiatric issues.
- Be cognizant of "red flags" within the history or physical exam that should prompt further neurologic evaluation in the form of either imaging or diagnostic procedure (Box 33-1).

PRIMARY HEADACHE DISORDERS

See Table 33-1.

Migraine headache

- **Definition:** Migraine is a chronic, idiopathic neurologic disorder characterized by episodic headaches of high intensity that may be preceded by, accompanied with, or followed by other associated symptoms (Figs. 33-1 and 33-2).
- Associated symptoms
  - **Premonitory (prodrome) symptoms:** Occur hours up to 1 to 2 days prior to development of migraine headache. These symptoms are often thought of as a warning of an impending migraine.
    - Fatigue
    - Difficulty concentrating
    - Yawning
    - Change in appetite
    - Change in sleep pattern
    - Change in mood
  - **Aura:** A set of reversible focal neurologic symptoms that occurs at the beginning of a migraine headache or just prior to its onset. Auras typically last less than 60 minutes. Visual symptoms are most common.
    - Visual changes (scintillating scotoma, visual field deficits, blurry vision)

BOX 33-1 *Red Flags in the Evaluation of Headache*

1. Headache onset after the age of 50
2. Sudden onset of severe headache
3. Change in headache pattern
4. Headache associated with systemic illness and fever
5. Headache associated with neck stiffness
6. Headache with focal neurologic deficits
7. Presence of papilledema
8. Headache in the setting of moderate/severe trauma
9. Alteration of consciousness, amnesia
10. Early morning nausea and vomiting without headache
11. Seizure occurring after headache

Table 33-1 CHARACTERISTICS OF MIGRAINE, CLUSTER, AND TENSION-TYPE HEADACHES

	Migraine	Cluster	Tension-type
Onset	Peaks in adolescence	30s or 40s	Variable; 20s or older
Frequency	1-2 attacks/mo, often with menses	1 or more attacks/day for 6-8 weeks	Episodic: <15 days/mo Chronic: >15 days/mo
Location	Unilateral more common than bilateral; frontotemporal or orbital	100% unilateral Generally orbitotemporal	Bifrontal, bioccipital, neck
Quality	Throbbing or intense pressure	Nonthrobbing, penetrating, boring	Squeezing, pressing, aching
Duration	4-72 hr, usually 12-24 hr	30 min-2 hr, usually 45-90 min	Episodic: several hours Chronic: all day
Prodrome	Changes in mood, energy, appetite	May include brief, mild burning in eye and internal nares	None
Aura	Up to 60 min, usually 20 min; often visual	None	None
Associated symptoms	Nausea, vomiting, photophobia	Ipsilateral ptosis-miosis, conjunctival injection, tearing, stuffed and running nose	Episodic: loss of appetite, light or sound sensitivity Chronic: light or sound sensitivity, nausea
Behavior	Go to dark, quiet room	Frenetic pacing, rocking	Generally not affected, or mild decrease in function

Modified from Marks DR, Rapoport AM: Practical evaluation and diagnosis of headache. Seminars in Neurology 17(4):307-312, 1997.

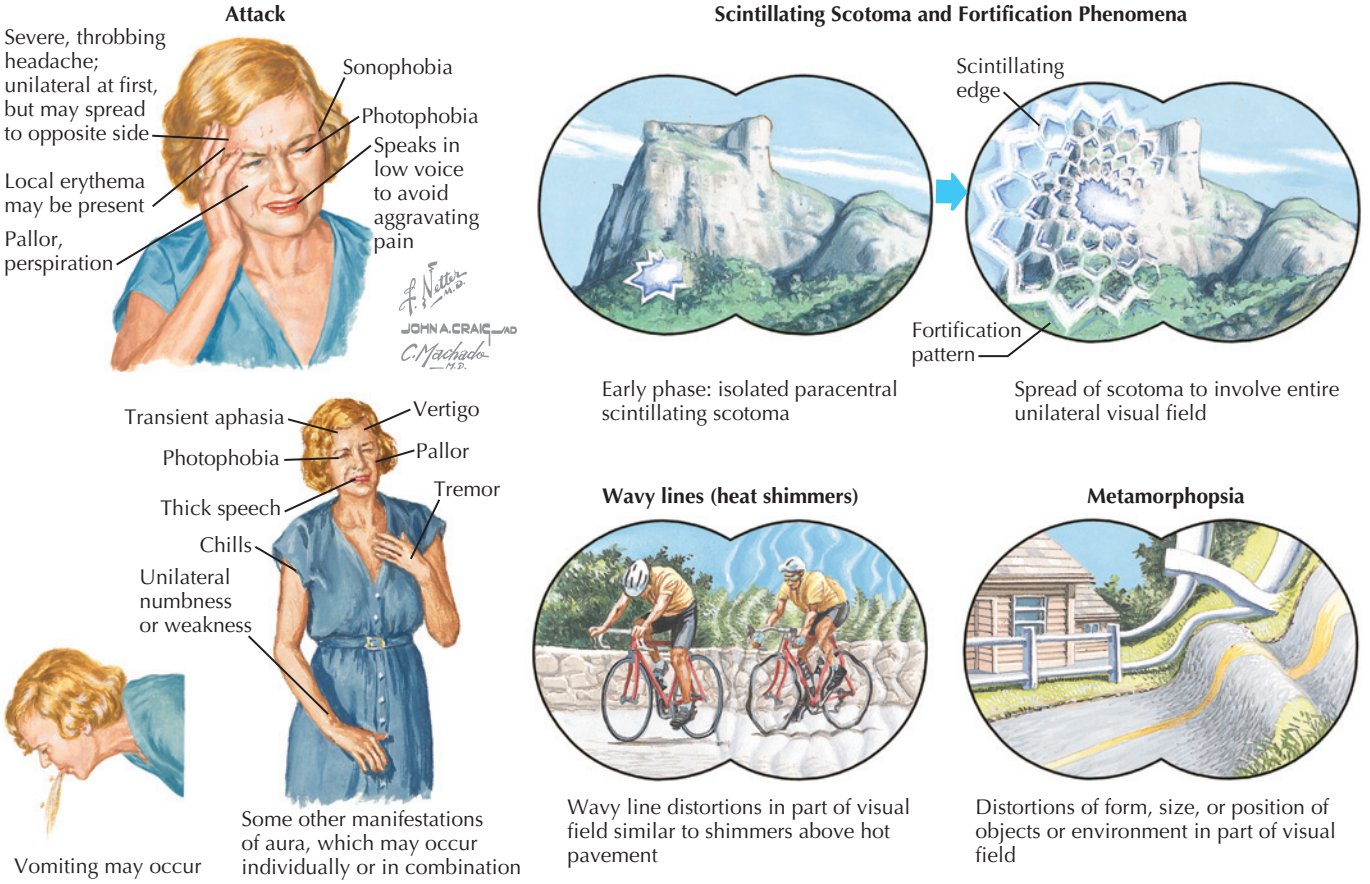


Figure 33-1 Mechanisms in Migraine.

- Sensory disturbances (pins and needles sensation, numbness)
- Speech disturbances (dysphasia)
- Motor deficits (rare)

- **Resolution (postdrome) symptoms:** These are symptoms that follow headache that may include some premonitory symptoms.
  - Exhaustion
  - Exhilaration
  - Depression
  - Nausea
  - Scalp tenderness

- Migraine headache is divided into two major subtypes by the IHS.
  - Migraine without aura
  - Migraine with aura (10% to 15% of migraines)
- Migraine headaches are typically unilateral, but can be bilateral. They also tend to have a pulsating nature, moderate-to-severe intensity, exacerbation by activity, and an association with nausea, vomiting, photophobia, and phonophobia.
- IHS diagnostic criteria for migraine are shown in Box 33-2.
- Typical age of onset is in adolescence.
- 18% of women and 6% of men report headaches that meet definition of migraine. Female-to-male ratio is 3:1.
- A survey of 791 male and female NCAA division I basketball players showed a 2.9% total prevalence rate (0.9% of males, 4.4% of females).
- 70% of females who suffer from migraines note a menstrual relationship to migraine attacks.
  - Migraine without aura: highest incidence during first 3 days of menses.
- Severe, debilitating forms of migraine include status migrainous, in which migraine lasts for more than 72 hours, and chronic migraine, in which migraines occur on 15 or more days per month.
- Common migraine triggers include aspartame, caffeine (use or withdrawal), estrogens, monosodium glutamate, nicotine, nitrates, progesterone, alcohol, cheese, chocolate, menstruation, missed meals, perfume, red grapes, sleep (too much or too little), stress, changes in environment/weather, exercise.

Treatment Options

Overview: Abortive treatment is appropriate if headaches occur once or twice per month, especially if predicted by aura. It is more effective if used as early as possible and a large single dose is more effective than multiple smaller doses.

Nonsteroidal anti-inflammatory drugs (NSAIDs): Chronic use should be avoided, especially in those with a history of peptic ulcer disease, gastritis, and renal insufficiency. Specific medication and initial dosages:

- Ibuprofen: 1200 mg PO (peak effect at 1 hour)
- Naproxen: 500 to 825 mg PO (peak effect at 2 hours)
- Indomethacin: 50 mg PO
- Ketorolac: 30 to 60 mg IM, with additional 30 mg in 8 hours if necessary

Dihydroergotamine (DHE-45): Is available in parenteral form and is typically given with an antiemetic.

- **DHE-45:** 1.0 mg IV/IM/SC, given 30 minutes after metoclopramide 5 mg or prochlorperazine 5 mg. May be repeated in 1 hour (90% effective).
- **DHE-45 (nasal):** 1.0 mg of intranasal puffs, repeating in 15 minutes.

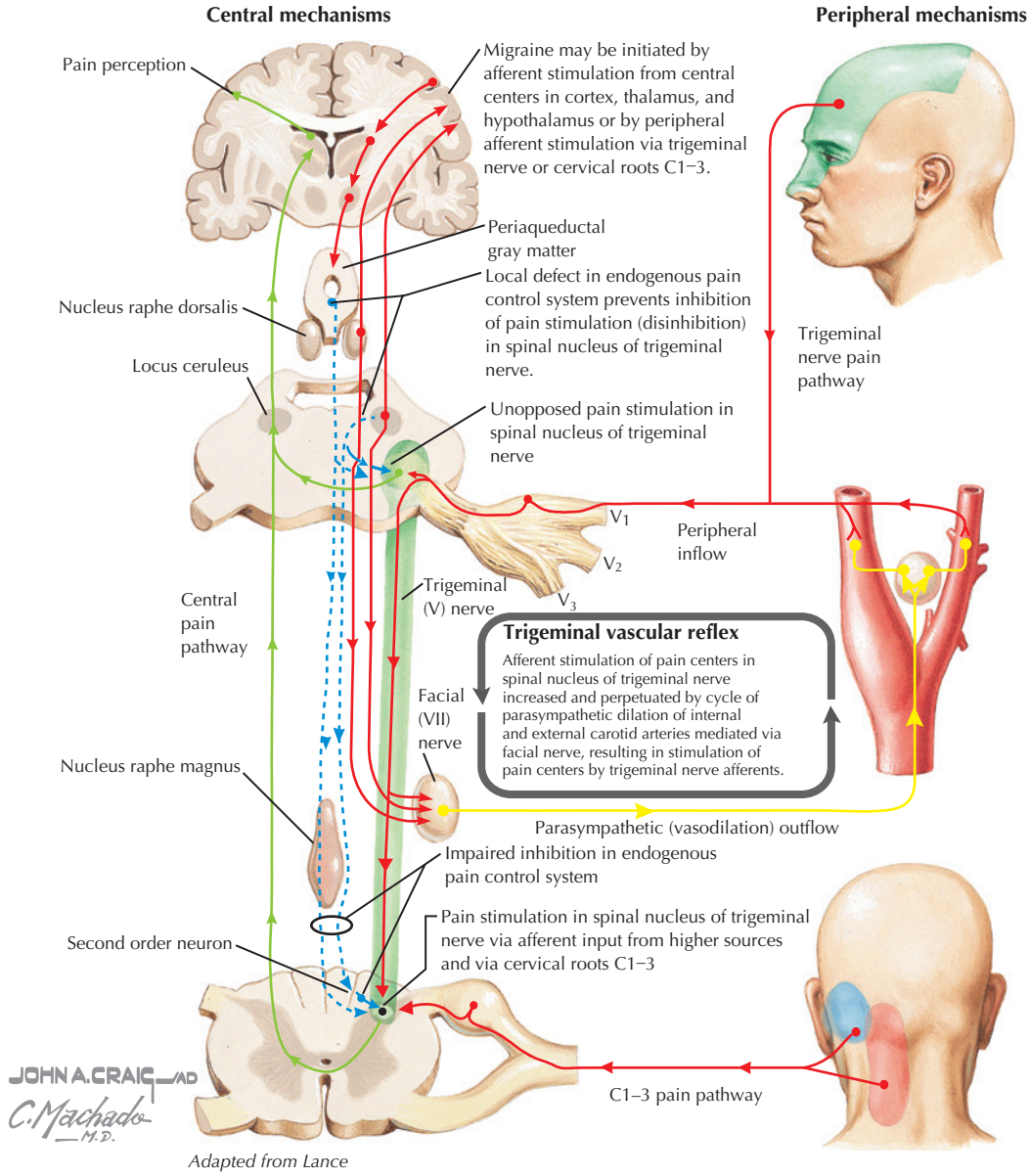


Figure 33-2 Migraine.

BOX 33-2 *IHS Diagnostic Criteria for Migraine without Aura*

A. At least five attacks fulfilling criteria B-D
B. Headaches lasting 4 to 72 hours
C. Headache has at least two of the following characteristics:
1. unilateral location
2. pulsating quality
3. moderate or severe pain intensity
4. headache aggravated by physical activity

D. During headache at least one of the following:
1. nausea and/or vomiting
2. photophobia and phonophobia

E. Cannot be attributed to another disorder

Modified from The International Classification of Headache Disorders, 2nd ed. Cephalgia 24(1):1-151, 2004.

- Favorable side effect profile when compared to ergotamine. (Minimal cardiovascular effects and nausea; no rebound headache; nausea/vomiting, GI upset, and muscle cramping have been reported.)
- DHE-45 is a vasoconstrictor and is therefore contraindicated in patients with cerebrovascular, cardiovascular, or peripheral vascular disease. It is also contraindicated in patients with severe hypertension, ischemic heart disease, renal or hepatic disease, sepsis, recent infection, or those who may be pregnant.

Triptans: This class of medication causes vasoconstriction and blocks pain pathways within the brainstem. They are selective 5-hydroxytryptamine (5-HT) receptor agonists and are used specifically to treat migraines. All medications belonging to this class have similar side effect profiles (nausea, dizziness, chest pressure, asthenia, dry mouth). They also carry the same precautions in that they should be avoided if possible in those with cardiovascular disease, peripheral vascular disease, hypertension, and hepatic dysfunction.

- **Sumatriptan (Imitrex):** 5-HT1D receptor agonist. The first triptan developed, it is available in injectable, oral, and intranasal spray forms.
  - Injectable dosage: 4 to 6 mg SQ initially; may repeat in 1 hour (maximum of two 6-mg injections in 24 hours). Recommended initial dose is 6 mg.
  - Oral dosage: 25 to 100 mg initially; may repeat dosage in 2 hours (maximum of 200 mg in 24 hours). The recommended initial dose is 50 mg because it is more efficacious than 25 mg and has fewer side effects than 100 mg without loss in efficacy.
  - Nasal spray dosage: 5 to 20 mg initially; may repeat in 2 hours (maximum of 40 mg in 24 hours). Recommended initial dose is 20 mg.
  - Tfelt-Hanson showed that 6 mg of the injectable form was more efficacious than 100 mg of the oral form and also had the fastest onset of action of all forms. The intranasal form has the same efficacy and a faster onset of action than the oral form, but limited therapeutic effects are seen for the first 30 minutes.
  - DHE versus SQ sumatriptan: DHE less effective at 2 hours (73% vs. 85%), but no difference after 2 hours. Headache recurred in 45% of sumatriptan-treated patients and 17.7% of DHE-treated patients.
- **Naratriptan (Amerge):** 5-HT1B/1D receptor agonist. Dosage: 1 to 2.5 mg PO; may repeat in 4 hours (maximum of 5 mg in 24 hours). Has lowest reoccurrence rate of headache of all the triptans.
- **Frovatriptan (Frova):** 5-HT1B/1D receptor agonist. Dosage: 2.5 mg PO; may repeat in 2 hours (maximum of 7.5 mg in 24 hours).
- **Rizatriptan (Maxalt):** 5-HT1B/HD receptor agonist. Dosage: 5 to 10 mg PO of regular or disintegrating (MLT) tablets; may repeat in 2 hours (maximum of 30 mg in 24 hours). Fastest onset of action of all triptans.
- **Eletriptan (Relpax):** 5-HT1B/1D receptor agonist. Dosage: 20 to 40 mg PO; may repeat in 2 hours (maximum of 80 mg in 24 hours).
- **Zolmitriptan (Zolmig):** 5-HT1B/HD receptor agonist. Dosage: 2.5 to 5 mg PO of regular or disintegrating (ZMT) tablets; may repeat in 2 hours (maximum of 10 mg in 24 hours).

Others:

- **Midrin** contains isometheptene (sympathomimetic amine vasoconstrictor), dichloralphenazone (sedative), and acetaminophen. Consider in patients unable to use ergots. Dosage: 2 tabs PO; may take 1 tab every 1 hour after initial dose until improvement (maximum of 5 tabs per attack).
- **Butalbital combinations** (Fiorinal, Fioricet) also may be used. Special concerns may arise about abuse potential and sedation. Dosage: 2 tabs PO; may repeat (maximum of 6 tabs in 24 hours).
- **Butorphanol intranasal (Stadol NS):** Mixed agonist-antagonist opioid analgesic. Used for severe attacks. One puff equals 5 mg morphine. Dosage: 1 spray; may repeat in 1 hour.
- **Intranasal lidocaine 4% solution** provided rapid relief (within 5 minutes) of headache, nausea, and photophobia in prospective, double-blind, placebo-controlled trial. Relapse was common.
- **Intravenous antiemetic medications** (chlorpromazine, prochlorperazine, metoclopramide) have shown some efficacy as monotherapy in the treatment of migraine. Practical use of these outside of the emergency department setting is low.

First-Line Choice in Athletes

Overview: Because of the efficacy, ease of administration, and relatively quick onset of action, athletes should use oral or injectable forms of triptans for first-line therapy of moderate migraines. Nasal DHE may be another good option secondary to ease of administration. For mild migraines, NSAIDs in any form should be tried initially.

Prophylactic treatment: Indicated in patients with more than four attacks per month or attacks lasting several days causing severe disability.

- **Beta-blockers** are agents of choice.
  - Only two are FDA approved: propranolol and timolol, which lack intrinsic sympathomimetic activity (ISA).
  - Long-acting form of propranolol increases compliance and is useful in patients with coexisting hypertension, angina pectoris, or thyrotoxicosis.
  - Contraindicated in patients with asthma, chronic obstructive pulmonary disease, congestive heart failure, atrioventricular conduction problem, or prescription for monoamine oxidase inhibitors.
  - Other beta-blockers used despite lack of FDA approval: nadolol, metoprolol, atenolol. Metoprolol is a selective beta-blocker that can be used in patients with concomitant pulmonary disease.
  - **Beta-blockers and athletes:** Negative effects on aerobic performance cause some concern for use in athletes. Also, beta-blockers are banned in certain sports and by certain athletic rules committees because they may provide an unfair advantage.
- **Calcium channel blockers:** Nimodipine, a selective cerebrovascular vasoconstrictor, and verapamil have been shown to be useful in migraine prophylaxis.
- **NSAIDs:** Various NSAIDs may be used with varying degrees of efficacy. They are more efficacious for **menstrual migraines** and should be started 2 to 3 days prior to menses (see later discussion).
- **Antidepressants (tricyclic):** Amitriptyline and clomipramine are most often used. Efficacy is attributed to antidepressant and analgesic action. They are especially effective in patients with coexisting migraine and tension-type headache. They are sedating so should be used at bedtime.
- **Antiepileptics:** Valproate, topiramate, and gabapentin are most often used. Only valproate and topiramate are FDA approved for this indication. They are category C and D drugs, respectively, so there should be concern for use in premenopausal female athletes; ensure birth control. There is no known adverse effects on exercise, although some antiepileptics are sedating.

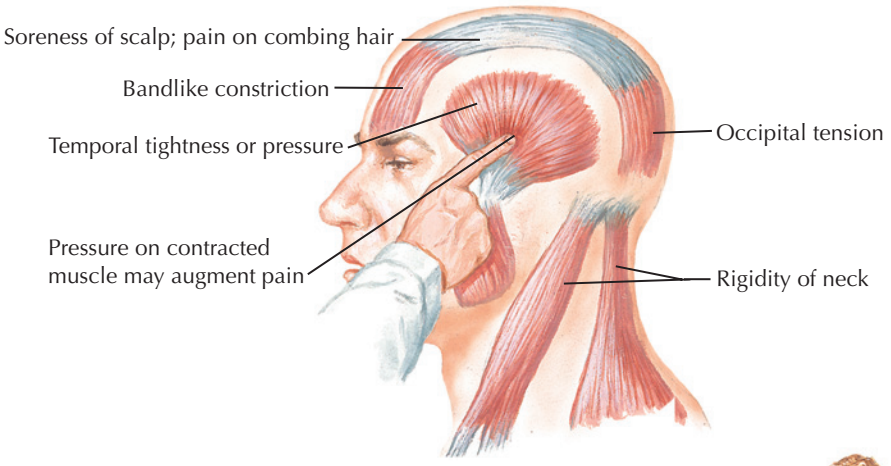
Status migraine treatment: Corticosteroids: dexamethasone, 10 mg IV followed by 4 mg IV every 6 hours. IV DHE, often given with metoclopramide, every 8 hours for 2 days.

Menstrual migraine treatment: "Mini-prophylaxis" may be achieved with the use of NSAIDs or long-acting triptans (frovatriptan and naratriptan) when started 2 to 3 days prior to menstruation and continuing until 3 days after. **Hormonal treatment** can be used to prevent the estrogen withdrawal that contributes to the development of menstrual migraine. Treatment strategies include the use of extended-cycle oral contraceptive pills, regular-cycle oral contraceptive pills with supplemental estrogen during the inactive pill week, and supplemental estrogen used alone perimenstrually.

Tension-Type Headache

- **Definition:** Tension-type headaches (TTH) are headaches of mild to moderate intensity that can be either episodic with varying frequency or chronic. Pain is usually bilateral and patients often report it being "vice-like" in nature. The most significant exam finding is pericranial tenderness to palpation (Fig. 33-3).
- IHS diagnostic criteria for TTH are listed in Table 33-2.
- TTH is the most common type of primary headache, with prevalence reaching 80% in the general population.

Intermittent, recurrent, or constant head pain, often in forehead, temples, or back of head and neck. Commonly described as "bandlike," "tightness," or "viselike"



Sleep disturbances common. Diurnal incidence: headache occurs most often between 4 and 8 AM and 4 and 8 PM

Psychogenic factors: emotional conflict and depression often seen in chronic headache

Figure 33-3 Tension-Type Headache.

Table 33-2 IHS CRITERIA FOR EPISODIC TTH

Infrequent episodic TTH	Frequent episodic TTH
A. At least 10 episodes occurring on < 12 days per year and fulfilling criteria B-D	**A.** At least 10 episodes occurring on > 1, but < 15 days of the month for 3 months and fulfilling criteria B-D
B. Headache lasts from 30 mins to 7 days	
C. Headache has at least two of the following: 1. bilateral location 2. pressing/tightening (non-pulsating) nature 3. mild or moderate intensity 4. not worsened by physical activity	
D. Headache has both of the following: 1. no nausea or vomiting 2. no more than 1 of photophobia or phonophobia	
E. Cannot be attributed to another disorder	

Modified from The International Classification of Headache Disorders, 2nd ed. Cephalgia 24(1):1-151, 2004.

- Both episodic and chronic TTH may exhibit photophobia and phonophobia, but nausea is usually isolated to chronic TTH.
- Chronic tension-type headache (CTTH): defined by IHS as headache present for more than 15 days per month for more than 3 months.
- CTTH prevalence in general population is 2% in men, 5% in women.

Treatment Options

Overview: TTHs are often responsive to nonpharmacologic therapy or over-the-counter headache preparations. Those who suffer from frequent TTH may benefit from stress reduction and relaxation techniques. For athletes, alteration of training regimens may be helpful. Counseling for patients with psychosocial issues including, but not limited to, anxiety and depression is also beneficial.

Abortive treatment:

- **NSAIDs:** Ibuprofen, naproxen, indomethacin, ketorolac, and others are used. Smaller dosages than used in migraine are typically effective for TTH.
- **Butalbital combinations** (Fiorinal, Fioricet) also may be used. Less frequent dosing interval than that used for migraine is usual. Special concerns may arise about abuse potential and sedation. Dosage: 2 tabs PO; may repeat (maximum of 6 tabs in 24 hours).
- **Tramadol (Ultram):** An opioid analog. It may have some abuse potential. Dosage: 100 mg PO four times daily (maximum of 500 mg in 24 hours).

Prophylactic treatment:

- **Tricyclic antidepressants:** Amytriptyline is most commonly used.
- **Beta-blockers:** Propanolol and timolol are used. Not a great choice for athletes because they impair aerobic exercise. They do this by reducing $\dot{V}O_2$ max and maximum heart rate and increasing fatigue. They are banned by the International Olympic Committee in diving and shooting.

General TTH treatment considerations: Watch for headache secondary to analgesic use and "rebound pain." Concerns and side effects in athletes differ from those in the general population; individualize treatment if possible.

Cluster Headaches

- **Definition:** Cluster headaches present as a series of reoccurring headaches that persist from weeks to months at a time

followed by remission periods. Pain is always unilateral and is of a severe, penetrating, stabbing nature. The location of these headaches is orbital, periorbital, or temporal (Fig. 33-4).

- Most important differentiating feature of cluster headaches is the presence of transient autonomic symptoms.
- Recur throughout the day, and wake approximately 50% of patients from sleep.
- IHS diagnostic criteria for cluster headaches are listed in Box 33-3.
- Occur more frequently in men than in women, with research showing male-to-female ratios that range from 2:1 to 6.7:1.
- Typical age of onset is between the 3rd and 5th decades.
- Alcohol, nitroglycerine, and histamine may trigger headache.
- Patients pace, rock, or bang head against wall (vs. patients with migraine, who sleep).
- Chronic cluster headache is a more severe form in which headache attacks occur for more than 1 year either without remission or remission lasting less than 1 month.

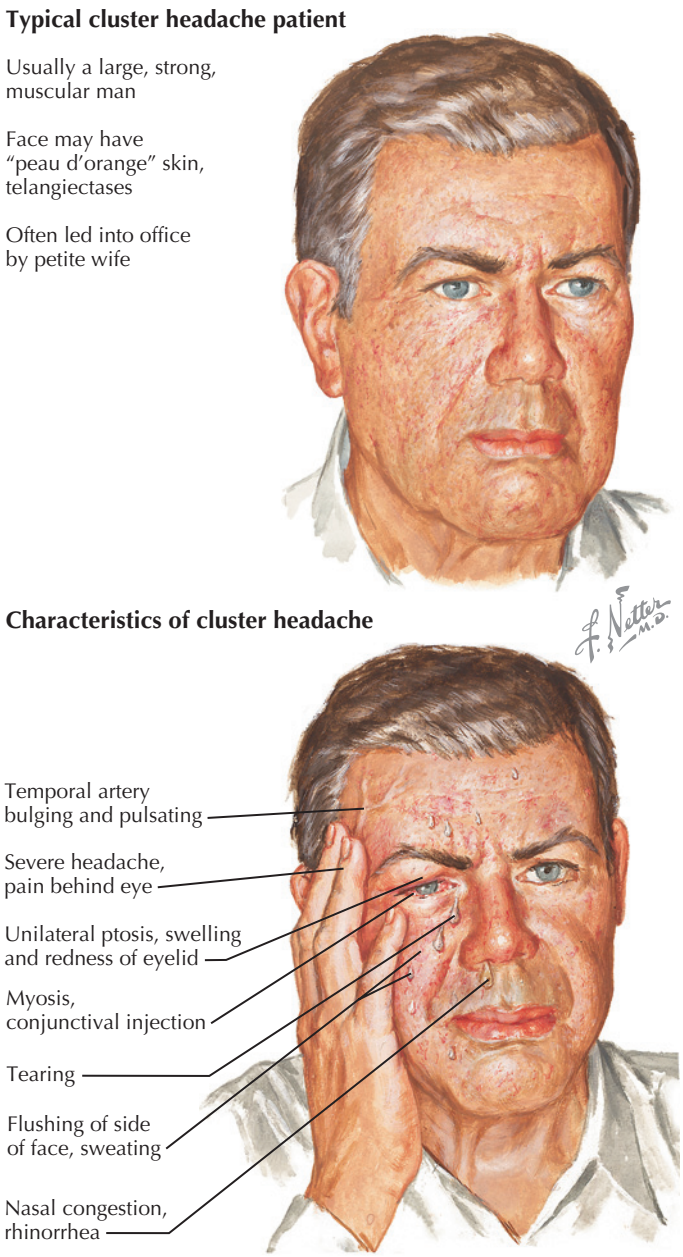


Figure 33-4 Cluster Headache.

BOX 33-3 *IHS Criteria for Cluster Headaches*

A. At least five attacks fulfilling criteria B-D
B. Severe unilateral orbital, supraorbital, and/or temporal pain lasting 15 to 180 minutes
C. Headache is accompanied by at least one of the following transient autonomic symptoms:
1. ipsilateral conjunctival injection and/or lacrimation
2. ipsilateral nasal congestion and/or rhinorrhea
3. ipsilateral eyelid edema
4. ipsilateral forehead and facial sweating
5. ipsilateral miosis and/or ptosis
6. a sense of restlessness or agitation

D. Attacks have a frequency of one every other day up to eight per day
E. Cannot be attributed to another disorder

Modified from The International Classification of Headache Disorders, 2nd ed. Cephalgia 24(1):1-151, 2004.

Treatment Options

Abortive treatment: Limited duration of headache makes most abortive treatment ineffective.

- **DHE-45** as used with migraine; DHE (0.5 to 1.0 mg) IV, IM, or intranasal.
- **Oxygen:** Inhalation with non-rebreathable mask; 8 L per minute over 10 to 15 minutes; 70% response.
- **Sumatriptan SQ** as used with migraine. Because of its rapid onset it is considered by many to be the most effective agent.
- **Intranasal lidocaine 4%:** Injected into sphenopalatine fossa, effective in some.
- Oxygen and sumatriptan are abortive treatments of choice in athletes.

Prophylactic treatment:

- **Corticosteroids:** Used as bridge between abortive and prophylactic therapy, they are effective in halting the cluster cycle. **Prednisone:** 40 to 60 mg PO daily for 5 days and tapered over 2 to 4 weeks.
- **Lithium carbonate:** One of the first drugs used for prophylactic treatment of chronic cluster headache. Dosage: 900 mg daily in divided doses. Blood levels need to be monitored and thyroid function tests followed. Concern that in athletes dehydration may cause increase in lithium levels.
- **Calcium channel blockers:** They inhibit the initial vasoconstrictive phase of cluster headaches. Verapamil is most often used and is the drug of choice for prophylaxis against cluster headaches in athletes. Verapamil 120 to 360 mg daily divided into two, three, or four doses. One-time daily dosing may be achieved with use of extended-release tabs.
- **Anticonvulsants:** Valproate and topiramate may play a role in nonpharmacologic treatment of cluster headaches.
- **Methysergide (Sansert)** is a synthetic ergot alkaloid that is structurally related to the potent hallucinogen LSD. It is a potent serotonin receptor agonist that is used for the prophylaxis of severe vascular headaches (migraine and cluster). Dosage: 2 to 8 mg PO daily, not to be used continuously for more than 6 months. May produce hallucinogenic effects. Main adverse effect is retroperitoneal fibrosis with prolonged use.

SPORTS/EXERCISE-RELATED PRIMARY HEADACHES

Athletes comprise a special subset of the headache-suffering population. This is because they may suffer from specific types of headaches that are the direct result of their participation in their sport.

Exercise/Effort-Induced Migraine

- **Definition:** Exercise/effort-induced migraines are headaches that fit the criteria for migraine headache (mainly in that they

are severe, unilateral, throbbing, and last 4 to 72 hours) and are brought on by exercise. Those who experience exercise/effort-induced migraines usually have a history of non–exercise-related migraines.
  - In one study, high-intensity bicycling for a duration of 30 seconds was shown to precipitate a typical migraine 4.5 to 5.5 hours after the cessation of the exercise in women with a previous history of migraine.
- Headache occurs after activity and may be with or without aura.
- Activities that usually trigger the migraines are aerobic, such as running and swimming.
- Dehydration, excessive heat, hypoglycemia, and hypomagnesemia are predisposing factors.
- Elevation of nitric oxide, which has been shown to increase during exercise and is responsible for causing central vasodilation and subsequent increased central nervous system sensitivity, has been proposed as a potential cause of these headaches.
- **Treatment:** Responds to the same prophylactic and abortive treatments as ordinary migraine. A slow warm-up regimen has been recommended as an adjunct in the prevention of exercise/effort-induced migraines.

Primary Exertional Headache

- **Definition:** Primary exertional headaches (PEH) are headaches that are precipitated by any form of exercise and do not meet migraine criteria. This IHS classification lumps together two headache entities that were previously well known to the sports medicine community as "benign exertional headache (BEH)" and "effort headache."
  - BEH are thought to arise from brief, strenuous physical exercise that involved increasing intrathoracic pressure and performing Valsalva maneuvers (e.g., weightlifting, sprinting, specific swimming events). These headaches are brief in duration.
  - Effort headaches are thought to arise from longer aerobic exercise activity common to a multitude of sports. These headaches typically have more migrainous features.
- This type of headaches was first described by Tinel in 1932 and termed "la cephalee a l'effort."
- PEH is the most common headache classification in athletes.
- PEH occurs in trained athletes and in those who are infrequent to exercise.
- IHS diagnostic criteria for primary exertional headache are listed in Box 33-4.
- This type of headache often occurs with exercise in hot weather or higher altitude.
- Thorough evaluation should be done prior to making the diagnosis of exertional headache because it may not always arise from primary causes.
  - Early study in 1968 followed 103 patients diagnosed with benign exertional headache; 10 eventually had underlying organic disease.
  - In another series of 221 patients with brain tumor, 60% had headache, and 22% of these had headache that worsened with cough and exertion; in 18%, pain increased with stooping or bending over.
  - There have also been case reports of spontaneous cerebrospinal fluid leaks causing headache with Valsalva being misdiagnosed as BEH.
- **Treatment:** May be prevented by limiting the amount of exercise and controlling the exercise environment. Most primary exertional headaches respond to NSAID therapy. Indomethacin can be used prophylactically, taken in 25- to 50-mg doses 1 to 2 hours prior to exercise. Also, twice daily or three times daily dosing of 50 to 150 mg of indomethacin may be used to abort theses headaches in those who have begun to have frequent episodes.

BOX 33-4 *IHS Criteria for Primary Exertional Headaches*

A. Pulsating headache fulfilling criteria B and C
B. Lasting from 5 minutes to 48 hours
C. Brought on by and occurring only during or after physical exertion
D. Cannot be attributed to another disorder

Modified from The International Classification of Headache Disorders, 2nd ed. Cephalgia 24(1):1-151, 2004.

SPORTS/EXERCISE-RELATED PRIMARY HEADACHE PRETENDERS

- There are many conditions that may present as primary exercise-related headaches. The intuitive physician will at least be cognizant of this fact and will take into account all of the differentials prior to making the final diagnosis.
- **Cardiac cephalgia:** Case reports describe these as headaches that begin with vigorous exercise and are relieved by rest. The severity of the headache may increase as the intensity and duration of the exercise increases.
  - Headache is caused by cardiac ischemia.
  - Neurologic evaluation is normal.
  - Diagnosis is made with exercise stress testing. The onset of the patient's headache may correlate with ECG changes indicative of myocardial ischemia.
  - Headache may improve with nitroglycerin treatment (vasodilator) and worsen with triptan treatment (vasoconstrictor), which is in stark contrast to primary exercise-related headaches such as exercise-induced migraine that will likely worsen with nitroglycerin and improve with triptan treatment.
  - **Treatment:** Myocardial revascularization has been shown to lead to complete resolution of these headaches.
- **Other differential diagnoses of exercise-related primary headaches**
  - Intracranial: brain tumors, subacute and chronic subdural hematomas, vascular malformations.
  - Craniospinal abnormalities: platybasia, Arnold-Chiari malformation, basilar impression.
  - Metabolic disorders: pheochromocytoma, Cushing's disease, myxedema, thyrotoxicosis, hypoglycemia of any etiology.
  - Neurologic diseases: hydrocephalus, chronic central nervous system infections, multiple sclerosis.

SPORTS/EXERCISE-RELATED SECONDARY HEADACHES

Acute and Chronic Posttraumatic Headache

- **Definition:** Posttraumatic headache (PTH) is any form of headache (migraine, TTH, cluster) that occurs after a known head trauma. They are categorized by the IHS as either acute or chronic and also as either occurring from mild or moderate/severe head trauma.
  - Acute PTH occurs within 7 days after trauma and resolves within 3 months.
  - Chronic PTH also occurs within 7 days after trauma, but persists for more than 3 months. Often occurs with others symptoms including poor concentration, irritability, depression, sleep disturbances.
  - IHS definitions of mild and moderate/severe head trauma are listed in Table 33-3.
- Headache is the most common neurologic symptom after head trauma; it persists for more than 2 months in 60% of patients.

TABLE 33-3 IHS DEFINITION OF MILD VERSUS MODERATE/SEVERE HEAD TRAUMA

Mild head trauma	Moderate/severe head trauma
All of the following: 1. either no LOC, or LOC less than 30 min 2. Glasgow Coma Scale = 13 or better 3. Symptoms or signs diagnostic of concussion	At least 1 of the following: 1. LOC > 30 min 2. Glasgow Coma Scale < 13 3. Posttraumatic amnesia for > 48 hr 4. Imaging showing traumatic brain injury or skull fracture

LOC, Loss of consciousness.
Modified from The International Classification of Headache Disorders, 2nd ed. Cephalgia 24(1):1-151, 2004.

- Location, severity, and pain characteristics vary considerably. Most studies show that PTH is *less* common when the head trauma is *more* severe.
- Tension-type headache is the most common PTH.
- Women have a higher risk for developing PTH.
- In a study of 443 high school and collegiate football players, 85% reported developing headache as the direct result of hitting.
- **Etiology** is controversial: both organic and psychogenic theories exist.
  - May be related to axonal injury or excitotoxic amino acid release, which results in neural injury.
  - May be related to altered cerebral hemodynamics and/or slowed cerebral circulation.
- **Posttraumatic migraine (footballer's migraine)** has been seen in sports such as boxing and soccer where taking blows to the head or repetitively heading the ball causes migraine.
  - Does not require significant trauma; not associated with significant amnesia.
  - After symptom-free interval of several minutes, visual, motor, sensory, or brainstem signs and symptoms begin (scintillating scotoma, flashing lights, blurred vision, or other sensory symptoms); lasts 15 to 30 minutes.
  - Then severe, throbbing headache develops, associated with nausea and vomiting.
  - Predisposing factors include history of non–sports-related migraine and family history of migraine (60% have parent with migraine, 70% have parent or sibling with migraine, 77% have parent, sibling, or grandparent with migraine).
- **Important to rule out organic basis for symptoms**
  - Do not return athlete to play if he or she remains symptomatic.
  - Immediate concerns include second-impact syndrome, vascular abnormalities, and other neurologic disorders.
  - **Intracranial bleeds that cause PTH**
    - **Epidural hematoma:** Usually caused by a tear in the middle meningeal artery secondary to temporal bone fracture; 20% to 50% have "lucid interval" following brief loss of consciousness or short period of confusion. Patients soon develop headache and focal neurologic deficits.
    - **Subdural hematoma:** Caused by tears in the subdural bridging veins. Signs and symptoms are slower than that of epidural hematomas, which may take between 24 hours and a few weeks to manifest. Headache and focal neurologic deficits are again routine features of this posttraumatic sequela.
    - **Subarachnoid hemorrhage:** Bleeding into the subarachnoid space. Patients present with "the worst headache of my life" complaint. This may quickly progress to seizures, which is a sign of meningeal irritation, and focal neurologic deficit.
- **Treatment:** Once organic cause is ruled out, treatment depends on the type of headache pattern (migraine, TTH, cluster). Acute PTH that is nonmigrainous in nature and occurs within hours of the trauma should be treated in the short term with non-NSAID analgesics. This is done to prevent worsening of an intercranial bleed by the antiplatelet effect of the NSAID, in the event an intercranial bleed has occurred.

Cervicogenic Headache

- **Definition:** A cervicogenic headache is a headache that is caused by abnormalities within the structures of the cervical spine, specifically C1 to C3. These structures include the muscles, ligaments, nerves, blood vessels, joints, and intervertebral discs.
- Pain typically starts in the neck and spreads to the oculofrontotemporal area. Pain is "dull" or "boring" in nature and fluctuates in intensity.
- Headache is constant and may last from hours to weeks.
- Often accompanied by restricted range of motion of the neck, and by ipsilateral, nonradicular, neck, shoulder, or arm pain.
- Often seen in response to moderate/severe head trauma.
- Affects women more than men with a female-to-male ratio of 4:1.
- IHS diagnostic criteria for cervicogenic headache are listed in Box 33-5.
- **Treatment:** Physical therapy has been shown to provide the most long-term relief of cervicogenic headache. This may include cervical traction, massage, and strengthening. Anesthetic blockade may provide temporary relief, but is not a long-term solution. Those who are provided relief with blockade but are not helped by physical therapy may benefit from radiofrequency neurotomy.

High-Altitude Headache

- **Definition:** Headache that occurs with ascent to altitudes above 2400 to 3000 meters and is caused by hypoxia. They are dull in nature and are of mild to moderate intensity. They typically develop within 6 to 24 hours of ascent and resolve within 8 hours after descent.
- Headache is the main symptom of acute mountain sickness (AMS).
- 20% to 50% of skiers and mountaineers experience headache at altitudes of 3000 to 5000 meters.
- **Treatment:** Main treatment is descent to lower altitude. High-altitude headaches may be prevented by allowing for

BOX 33-5 *IHS Criteria for Cervicogenic Headache*

A. Pain referred from a source in the neck and perceived in one or more regions of the head or face
B. Clinical, laboratory, or imaging evidence of a disorder within the cervical spine or soft tissues of the neck that is known to cause headache
C. Evidence that the pain caused by the neck disorder is based on at least one of the following:
  1. Demonstration of clinical signs that implicate the source of pain in the neck
  2. Abolition of the headache following diagnostic blockade of the cervical structure or its nerve supply
D. Pain resolves within 3 months after treatment of the causative disorder

Modified from The International Classification of Headache Disorders, 2nd ed. Cephalgia 24(1):1-151, 2004.

proper acclimation to one altitude before climbing to a higher altitude. Pharmacologic treatments include:

- **Pretreatment with aspirin:** Raises headache threshold; associated with less pronounced cardiorespiratory response to short-term exercise at altitude; can prevent headache.
- **Ibuprofen:** More effective than placebo in altitude-related headache. In randomized, double-blind crossover study, ibuprofen, 600 mg, was effective in treating headache, whereas sumatriptan, 100 mg, was ineffective.
- **Prophylaxis:** Acetazolamide, 250 mg PO twice daily or 500 mg PO once daily with use of extended-release tabs. Prophylaxis should start on the day before the climb.

Diving Headache

- **Definition:** Headache that is caused secondary to hypercapnia and most readily associated with deep-water diving. Carbon dioxide builds up in divers who practice "skip breathing," which is the act of intentionally holding respiration to conserve air or affect buoyancy in the water.
  - Hypercapnia causes cerebrovascular vasodilation and elevated intracranial pressure, which leads to headache.
  - Diving headache is nonspecific in character, but migrainous-type headache may occur.
  - Occurs at depths below 10 meters.
  - Worsens during resurfacing or decompression phase of the dive.
- **Treatment:** Taking deep, slow breaths may help prevent these headaches from occurring. Treatment with 100% oxygen will usually resolve these headaches within 1 hour.

SPORTS/EXERCISE-RELATED CRANIAL NEURALGIA HEADACHES

External Compression Headache

- **Definition:** Headache that results from continuous stimulation of the cutaneous nerves of the head. The continuous nerve stimulation is caused by externally applied pressure. This pressure usually results from the wearing of a tight headband, swim goggles, or diving mask.
- Headache is dull in nature and increases in intensity as duration of compression increases.
- May contribute to the development of diving headache.
- **Treatment:** Removal of the compressing device (headband, mask, swim goggles) results in complete resolution of the headache.

Cold-Stimulus Headache

- **Definition:** Headache that occurs after exposing the unprotected head to cold temperatures (e.g., diving in cold water).
- Headache is dull and diffuse in nature.
- May contribute to the development of diving headache.
- **Treatment:** Headache resolves with removal of the cold stimulus.

CLASSIC STUDY OF SPORTS-RELATED HEADACHE

- Williams and Nukada in 1994 assessed the prevalence of sports-related headaches in two groups of university students. One group consisted of 178 male and female medical students and the other group of 190 male and female physical education students. Each individual in both groups was surveyed about his or her experience with sports and exercise-related headaches.
- 35% of respondents reported having a sports/exercise-related headache in the past.
- **4 categories of sports-related headache were used**
  - Effort migraine: 9% overall
  - Trauma-triggered migraine: 6% overall
  - Effort-exertion headache: 60% overall
  - Unspecified, "other": 3% overall
- Men in the physical education group had a higher rate of sports/exercise-related headache than the men in the medical student group, which was probably related to the higher frequency of trauma-related headaches in contact sports. There is no significant difference in the rate of sports-related headache among women in two groups.

CHARACTERISTICS OF PRIMARY HEADACHE DISORDERS

See Table 33-1.

DRUG CHOICES FOR HEADACHES IN ATHLETES

See Table 33-4.

Table 33-4 DRUGS OF CHOICE FOR HEADACHES IN ATHLETES

	Tension headache	Migraine headache	Cluster headache
Abortive treatment	Acetaminophen NSAIDs	Sumatriptan Other 5-HT agonists DHE-45 (nasal) Intranasal lidocaine	Oxygen Sumatriptan DHE-45
Prophylactic treatment	NSAIDs TCAs at bedtime	Verapamil Valproate, topiramate TCAs at bedtime	Verapamil

DHE, dihydroergotamine; NSAIDs, nonsteroidal anti-inflammatory drugs; TCAs, tricyclic antidepressants.

RECOMMENDED READINGS

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11. Williams SJ, Nukada H: Sport and exercise headache: Part 1. Prevalence among university students. Br J Sports Med 28:90-95, 1994.
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Skin Problems in the Athlete

34

Scott H. Grindel

GENERAL CONSIDERATIONS

Although athletes are probably as susceptible to most dermatologic disorders as the general population, there are some disorders that are more common in athletes. Table 34-1 describes common dermatologic lesions and gives examples of each.

PROBLEMS CAUSED BY FRICTION AND PRESSURE

Blisters

Overview: Fluid-filled bullae that form at the site of friction (usually caused by a change in training pattern or ill-fitting equipment).

Presentation: Seen early in the workout season in sports using hard playing surfaces and repetitive activities (athlete may have recently changed the equipment responsible for the injury).

Diagnosis: Typical lesions, location, and history (differential diagnosis includes pemphigus and pemphigoid).

Treatment:

- May use protective socks, Vaseline, mole skin, or Compeed for treatment and prevention.
- Superglue may be painted on hot spots for protection from irritation, but be cautious because this may cause increased traction on the site.
- Drain bulla in a sterile manner only if tense to touch.
- Use an antibiotic ointment (e.g., bacitracin ointment) or hydrocolloid if the lesion is open.

Return to play: When the lesion is covered and athlete can tolerate the pain.

Calluses (Tylomata) and Corns

Overview: Calluses and corns are caused by a thickening of the outer layer of skin (hyperkeratosis) caused by repetitive friction; they can lead to blisters (Fig. 34-1). There is no central core as seen in verruca vulgaris and normal skin lines are maintained. Calluses generally form on the hands or feet (typically on ball of foot, heel, underside of big toe, or under proximal metatarsals), although they may form wherever there is pressure on the skin. Calluses are usually not painful and may be useful (a tennis player might develop calluses on the palm that protect his or her hand when handling a tennis racket). Corns have an inner core that can be soft or hard. A soft corn is found between toes (usually the fourth and fifth toes); a hard corn is often found over a bony part of a toe (usually the lateral fifth toe).

Presentation: The athlete may describe a recent change in equipment, ill-fitting equipment, or new or repetitive activity on hard playing surfaces.

Physical exam: Evaluate the athlete for an underlying anatomic defect such as pronounced metatarsal heads or boney deformities.

Table 34-1 DESCRIPTION OF TERMS IN DERMATOLOGY

Terminology	Description	Example
Macule	A discolored spot or patch on the skin, neither elevated nor depressed, of various colors, sizes, and shapes.	Vitiligo Café au lait spots Petechiae
Papule	A solid lesion elevated above the plane of the surrounding skin. Often precede vesicles and pustules. Generally considered less than 1 cm in diameter.	Measles Acne vulgaris
Ulcer	An open sore or lesion of the skin or mucous membranes where there has been destruction of the overlying epidermis and upper papillary layer of the dermis resulting in the formation of a crater.	Decubitus ulcers Venous stasis ulcers Aphthous ulcers
Nodule	A palpable solid round or ellipsoidal lesion deeper than a papule and is in the dermis, subcutaneous tissue, or epidermis. The depth rather than the diameter differentiate it from a papule.	Bouchard's and Osler's nodes Warts Squamous cell carcinoma Basal cell carcinoma
Wheal	A rounded or flat-topped pale red elevation in the skin that is evanescent, disappearing within hours, and often intensely pruritic. A result of edema in the upper layer of the dermis.	Urticaria Insect bites
Bulla	A large blister or skin vesicle filled with serum, lymph fluid, blood, or extracellular fluid. They are located within the epidermis, or the epidermal-dermal interface. Usually more than 0.5 cm in diameter.	Blisters Pemphigus
Vesicle	A small blister filled with serum, lymph fluid, blood, or extracellular fluid. They are located within the epidermis, or the epidermal-dermal interface. Usually less than 0.5 cm in diameter.	Herpes zoster Herpes simplex Variola Varicella
Pustule	A circumscribed elevation of the skin that contains a purulent exudate that may be white, yellow, or greenish yellow. May be associated with a hair follicle. Vesicles may become pustules.	Acne vulgaris Impetigo
Plaque	An elevation above the skin surface that occupies a relatively large surface area in comparison with its height above the skin. It may be formed by a confluence of papules.	Psoriasis Mycosis fungoides
Lichenification	Like a plaque, but the elevation above the skin surface is due to proliferation of the keratinocytes and stratum corneum caused by continued irritation. The skin appears thickened, and skin lines are accentuated.	Eczematous dermatitis
Scales	Because of an increased rate of proliferation of epidermal cells, the stratum corneum is not formed normally, causing the skin to peel in visible sheets or flakes.	Eczema Seborrhea Psoriasis
Crusts	Result when serum, blood, or purulent exudate dries on the skin surface. They may be thin, delicate, and friable, or thick and adherent.	Impetigo Ecthyma

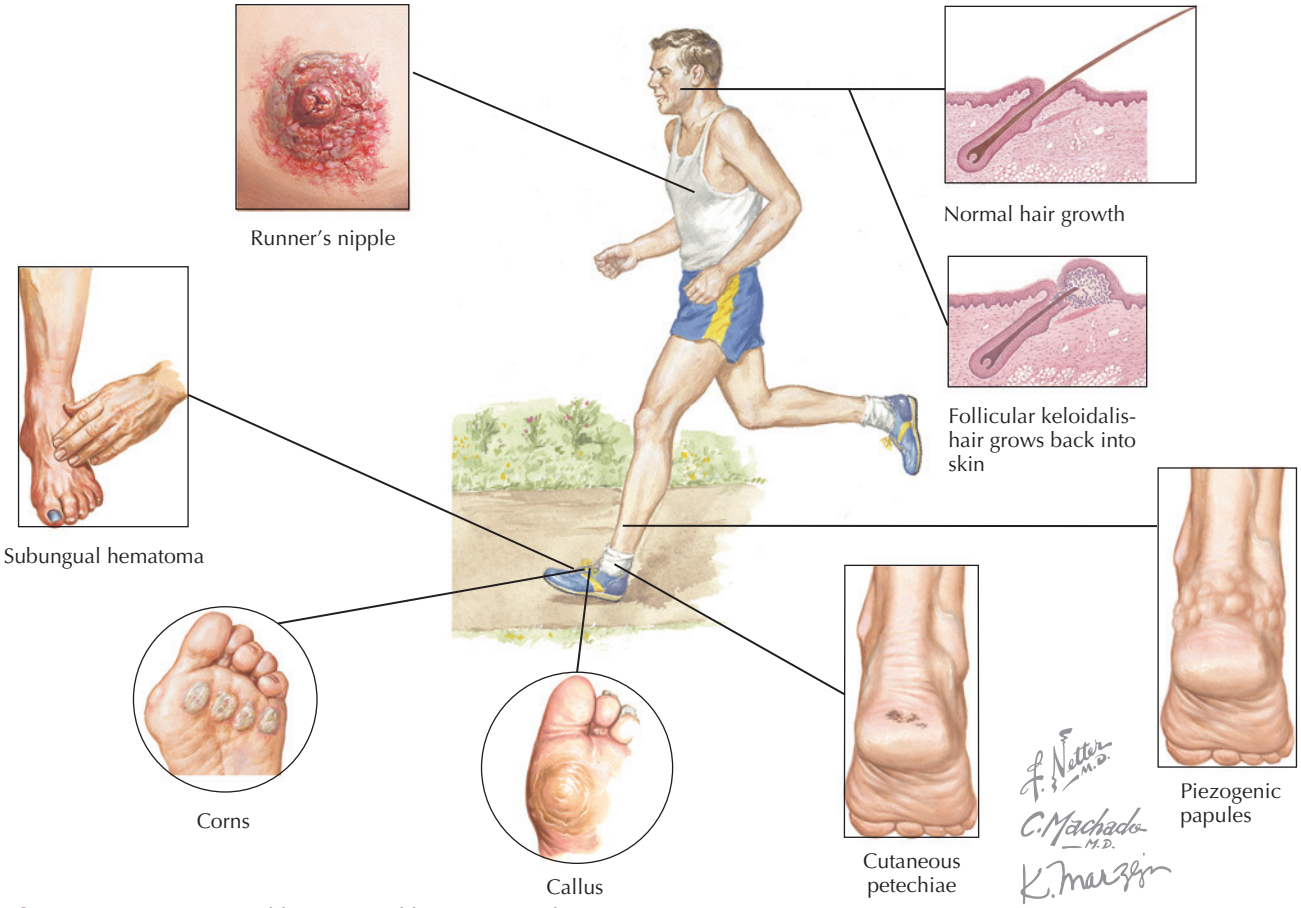


Figure 34-1 Common Problems Caused by Friction and Pressure.

Diagnosis: Diagnosis is made through typical history and characteristic location over pressure points (differential diagnosis includes plantar warts [lack "black dots" of warts], bunions, and psoriasis).
Treatment: The athlete may use a pumice stone to reduce the bulk of the callus or the athletic trainer or physician can pare it down with a scalpel. Serial trichloroacetic acid application and debridement every 2 to 3 weeks is effective and relatively painless. Use of gloves, protective socks, Vaseline, or mole skin may also protect the skin from friction. Modification of footwear to reduce or remove pressure points with metatarsal bars, cutouts, and widening of toe box (if located between toes). Foot orthotics may be used if the callus or corn is associated with mechanical defects.
Return to play: As tolerated.

Chafing

Overview: Superficial erosion of the epidermis caused by mechanical friction of opposing areas of the body (aggravated by sweat, excess muscle, or subcutaneous fat). Most commonly seen over the upper inner thighs but is also seen on the neck and in the axilla.
Presentation: More often caused by the friction of fabric against skin than skin on skin (usually seen in long-distance sports such as running and bicycling).
Physical exam: Is consistent with typical lesions located in a moist area where there is contact with opposing skin, especially between the thighs, in the axilla, and on the neck.
Diagnosis: Made by the identification of typical lesions in a characteristic location (differential diagnosis includes contact dermatitis, tinea cruris, intertrigo, and erythrasma).
Treatment: Cool compresses, exposure to air, nonirritating ointments to add lubrication (bacitracin, Vaseline, Aquaphor), low-friction fabrics with longer leggings, friction-reducing powders, weight loss.
Return to play: As tolerated.

Runner's Nipple

Overview: Also called jogger's nipple, weightlifter's nipple (see Fig. 34-1). Soreness, dryness, or bleeding of or irritation to the nipples following running or other physical exercise (superficial irritation can lead to the formation of fissures and result in bleeding). More common in men than women; more prominent in long-distance runners.
Presentation: Acute tenderness of the nipple after prolonged physical activity in a shirt with rough fabric over the nipple (caused by friction of coarse shirt fabric or the logo on a jersey against the nipple).
Physical exam: Findings include erythema, edema, scaling and/or fissuring of either or both the nipple and the areola, possibly with bleeding.
Diagnosis: Denuded erosions of the nipple and/or areola; severe cases may have bleeding.
Treatment: Wash using warm water and a mild antibacterial soap. Use a simple dressing with antibiotic cream. Men should run without shirt when possible; women should wear a sports bra. A lubricating ointment such as Vaseline can be used prophylactically on the nipples. Tape with minimal adhesive can be applied directly to the nipple and areola.
Return to play: As tolerated.

Abrasions

Overview: Also called road rash, turf burn, raspberry (term may be applied to both a fresh injury and the scar tissue left by an old one). Denuded epidermis and superficial dermis with punctuate bleeding and exudate.

Presentation: Skin and subcutaneous tissue injury caused by abrasive contact with sport surfaces such as asphalt, gravel, turf, or ice (seen in bicycling/motorcycle accidents, rollerblading, football, soccer, runners, and ice hockey).

Physical exam: Carefully examine the abrasion for foreign bodies and exposed bone.

Diagnosis: Difficult to misdiagnose with consistent history but must be watchful of underlying injuries, foreign bodies, and infection.

Treatment:

- Initial cleansing with warm water and antibacterial soap or diluted hydrogen peroxide; high-pressure irrigation helpful with dirtier wounds.
- Removal of gravel or other foreign debris may require a brush after careful anesthetic block or sedation.
- Follow tetanus protocol.
- Hydromembranes (e.g., DuoDERM), applied and left on the wound for several days, can be especially effective.
- Alternatively, an antibiotic cream or spray may be used; should be covered with a clean nonadherent dressing, changed daily.
- Be especially wary of infection; consider prophylactic antibiotics.
- For particularly large or deep wounds, consider referral to dermatology for skin grafting.
- For prevention, use additional protection in areas of potential trauma, especially elbows and knees.

Return to play: When the athlete passes functional testing and the area can be adequately covered, unless there has been a surgical repair in which case a prolonged recovery may be required.

Follicular Keloidalis (Acne Keloidalis)

Overview: Nontender, firm fibrous papules each associated with a hair shaft; may not be identified readily (see Fig. 34-1). Initially presents as an inflammatory folliculitis but may undergo a profound cystic change if untreated. Mostly seen in African-American athletes.

Presentation: Caused by friction over a recently shaved area that causes the growing hair shaft to curl back and penetrate the surrounding skin, which leads to a local foreign body reaction (seen mostly around the edges of the football helmet, rarely under shin and thigh pads in hockey players).

Diagnosis: Typical lesions in an area of friction under pads or helmet.

Treatment: Aggressive antibiotic treatment of underlying folliculitis. Serial injection of dilute triamcinolone solution directly into papules if refractory. Delay treatment until season is complete and causative gear is no longer worn. Surgical excision may be required in severe cases. Place padding over equipment to reduce friction in involved areas.

Return to play: As tolerated.

Subungual Hematoma

Overview: Also called black toe, runner's toe, tennis toe, skier's toe (see Fig. 34-1). Splinter hemorrhage of the nail bed usually involving the first or second toe. Risk factors include Morton's toe and long or deformed nails.

Presentation: Develops acutely after pressure from tight shoes or from sudden decelerations (most often seen in racket sports, football, skiers with tight boots, and distance runners, especially with downhill running).

Physical exam: Hemorrhage is easily identified and may include the entire subungual area. The hemorrhage will not usually extend beyond the border of the nail, if it does consider a biopsy to rule out melanoma. Examine the shoes for proper fit, especially proper length, and that the foot does not have too much anterior-to-posterior movement.

Diagnosis: Physical exam and supporting history (care should be taken to differentiate from subungual melanoma).

Treatment: No treatment necessary in most cases. Close trimming of toenail proximal to the distal aspect of the toe, properly fitting shoes with adequate room in the toe box, and change of running style or route usually help.

Return to play: As tolerated.

Plantar Petechiae

Overview: Also called black heel, black dot syndrome, talon noir. Intraepidermal bleeding and petechiae of the heel (occurs on the heel at the posterior edge of the foot pad) (see Fig. 34-1). Seen in volleyball, racket sports, running, lacrosse, and basketball; associated with poor fitting shoes and repetitive trauma (cutting or stops).

Presentation: Caused by sheering forces and sudden stops. May present early after acute onset or more often after it has been present for a long-standing period of time and the athlete becomes concerned.

Physical exam: Petechiae are seen in the posterior heel. With paring of the lesion, skin lines are maintained with no additional bleeding, as can be seen with melanoma.

Diagnosis: Petechiae are seen in the posterior heel with a supporting history.

Treatment: Properly fitted shoes, use of a "soft" shoe or heel pads, and thick socks.

Return to play: Athlete may return to play without restriction.

Piezogenic Papules

Overview: Protrusions of fat appear as dome-shaped papules over mediolateral surfaces of the midfoot and heel (seen mostly in serious long-distance runners) (see Fig. 34-1).

Presentation: Caused by fat herniations through defects in the dermis. Pain may be of sufficient severity to preclude continuation of activity.

Diagnosis: Condition apparent only when pressure placed on sole of foot from below (may not be tender in resting state).

Treatment: No medical or surgical interventions have been shown to be of definitive value. Usually not symptomatic during normal walking. Some sufferers benefit from special orthotics or rigid heel cups.

Prevention: Avoidance of running or prolonged standing until the pain subsides.

Return to play: As tolerated.

SUN- AND HEAT-RELATED PROBLEMS

Sunburn

Overview: The outer layer of skin is damaged from excessive exposure to the sun's ultraviolet (UV) rays (Fig. 34-2). Most sunburns cause mild pain and redness and affect only the outer layer of skin, but more significant burns can affect the deeper layers and nerves, causing blisters, bullae, and severe pain (see Fig. 34-2). Fair complexion and blue eyes are risk factors as are water sports, snow sports, and other outdoor sports. A history of sensitivity to sun exposure and severe burns increases the risk of skin cancers.

Presentation: Athletes usually present early during the warm season or after a trip to the south for early season practices. Water sports that afford little protection from the sun are at the highest risk. Ask about sun-sensitizing medications such as tetracycline, sulfa, and phenothiazines.

Treatment: Cool compresses, aloe vera lotions, topical anesthetics and/or antihistamines, antibiotic ointments if second-degree

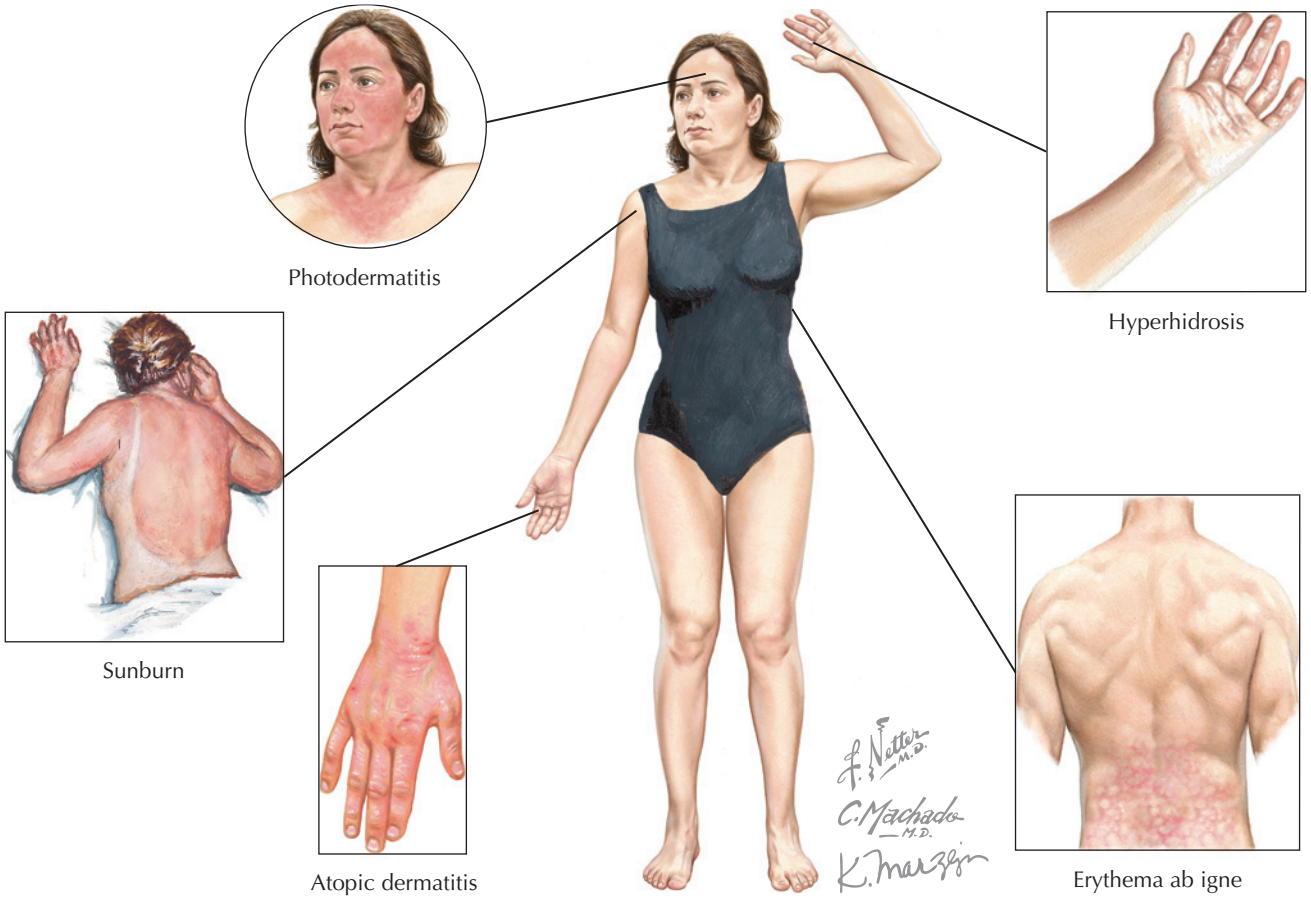


Figure 34-2 Common Sun- and Heat-Related Problems.

burns, oral fluids, maintain integrity of the overlying skin; oral and topical steroids may be required for moderate to severe burns to control inflammation and discomfort.

Prevention: Sunscreen and sunblock (sweatproof/waterproof), protective clothing and hats, avoid midday sun from 10 A.M. to 4 P.M., gradual sun exposure to develop a protective tan (tanning booths may be a more controlled environment), provide shade near workout areas.

Photodermatitis

Overview: An immune reaction directed against the skin due to the UV rays of the sun (see Fig. 34-2). It may be caused by a medication that makes the skin more sensitive, a skin product, autoimmune conditions such as systemic lupus erythematosus, or a vitamin deficiency. It ranges from nodular (sun poisoning) to solar purpura to solar urticaria (see Fig. 34-2).

Presentation: May present as hivelike lesions, nodules, purpura to generalized edema. Review history for sun-sensitizing medication or cofactors such as psoralens, coal tars, photoactive dyes (eosin, acridine orange), musk ambrette, methylcoumarin, lemon oil (may be present in fragrances), PABA (found in sunscreens), salicylanilide (found in industrial cleaners), hexachlorophene (found in some prescription antibacterial soaps), tetracyclines, NSAIDs, sulfonamides, griseofulvin, diuretics, phonothiazides, first-generation sulfonylurea agents, Benadryl, and cosmetics.

Diagnosis: Made through identification of typical lesions after sun exposure.

Treatment: Difficult to treat; avoidance of sun exposure is best or use sunblock without PABA; avoid psoralen with UVA (PUVA), antihistamines, and sun-sensitizing medications; severe cases may require IV steroids.

Return to play: Can be accomplished when stabilized, but avoidance of prolonged exposure to the sun is recommended unless an offending cofactor is identified and eliminated.

Atopic Dermatitis

Overview: Eczematous eruption that is itchy, recurrent, flexural, and symmetric; may be aggravated by heat, sweat, or exertion (see Fig. 34-2). Infants have facial and generalized or sometimes patchy body eczema. Adolescents and adults have eczema in flexural areas and on the hands. Seen in athletes exposed to dramatic temperature changes.

Presentation: It generally begins early in life, follows periods of remission and exacerbation, and usually resolves by the age of 30. There is a polygenic inheritance pattern seen. Mechanism is unknown.

Physical exam: Lesions are identified in the flexural areas of the body, and sometimes the hands, symmetrically.

Diagnosis: Made by identifying typical lesions with a supporting history.

Treatment: May be improved by sun exposure and emollients, avoidance of radical temperature changes, and topical steroids.

Return to play: As tolerated; secondary bacterial infection may preclude play.

Hyperhidrosis

Overview: Excessive perspiration; may be congenital or stress-related (see Fig. 34-2).

Presentation: May cause problems with grip, vision, and self-confidence.

Physical exam: Sweaty palms, feet, scalp, forehead, axilla, and groin may be seen but may be entirely normal.

Diagnosis: Appropriate history and physical findings.
Treatment: Aluminum chloride (Certain Dry, Drysol, etc.) applied before bed; after several weeks, may need application only once or twice a week. Iontophoresis units can be very useful.
Return to play: As tolerated.

Erythema Ab Igne

Overview: Also called erythema á calore or toasted skin syndrome (see Fig. 34-2). Reticulated erythema in acute phase directly over area of applied heat; progresses to a reticulated dark hyperpigmentation over time. Seen in athletes who regularly apply heat to certain areas of the body.
Presentation: The athlete may present acutely after reticulation fails to subside or the chronic changes may be noted at the preparticipation physical or in the training room; asymptomatic.
Diagnosis: Typical lesions are noted with a supporting history.
Treatment: Avoid application of causative heat; reassure athlete that this finding does not indicate serious disease; cessation of applied heat leads to spontaneous resolution in 3 to 6 months; present other therapy options.
Return to play: Without restriction.

COLD-RELATED INJURIES

Frostnip

Overview: See Chapter 18, Exercise in the Cold and Cold Injuries. Frostnip is damage to the superficial layers of the skin (first-degree frostbite) caused by extreme cold, at or below freezing. Damage is due to freezing of the superficial tissue and will usually result in flaking of the epidermis. Typically seen in outdoor winter sports.
Presentation: Usually present with burning pain over the affected area (Fig. 34-3).
Physical exam: May appear flushed or have rosy cheeks and ears; affects nose, cheeks, and ears most often.
Diagnosis: Typical lesions with supporting history.
Treatment: Treat symptomatically; cover all exposed skin in subfreezing conditions for prevention.
Return to play: Without restriction.

Frostbite

Overview: See Chapter 18, Exercise in the Cold and Cold Injuries. Extended exposure to subfreezing temperatures and poor blood flow can cause severe damage to skin and other tissues by freezing; typically seen in outdoor winter sports. Results in cellular disruption in the blood vessels, nerves, and soft tissue.
Presentation: Athlete may present after competition in freezing temperatures with a numb hard digit, nose, ear, or penis (see Fig. 34-3).
Physical exam: May cause first-, second-, or third-degree injuries; first- and second-degree injuries most commonly seen in athletes. Most often affects the ears and penis in athletes.
Treatment: Rapid rewarming with warm water bath; use caution in coexisting hypothermia; treat like a burn; serious or extensive injuries may require hospitalization.
Return to play: Depending on the extent of the injury—may return as soon as tolerated.

Chilblains (Pernio)

Overview: See Chapter 18, Exercise in the Cold and Cold Injuries. Caused by prolonged exposure of extremities to nonfreezing temperatures; acute and chronic forms. Results in breakdown of the dermis with resultant irritation and discomfort. Predisposes to future cold intolerance.
Treatment: Protect from further trauma and cold. May require antibiotics. Prevention is key.

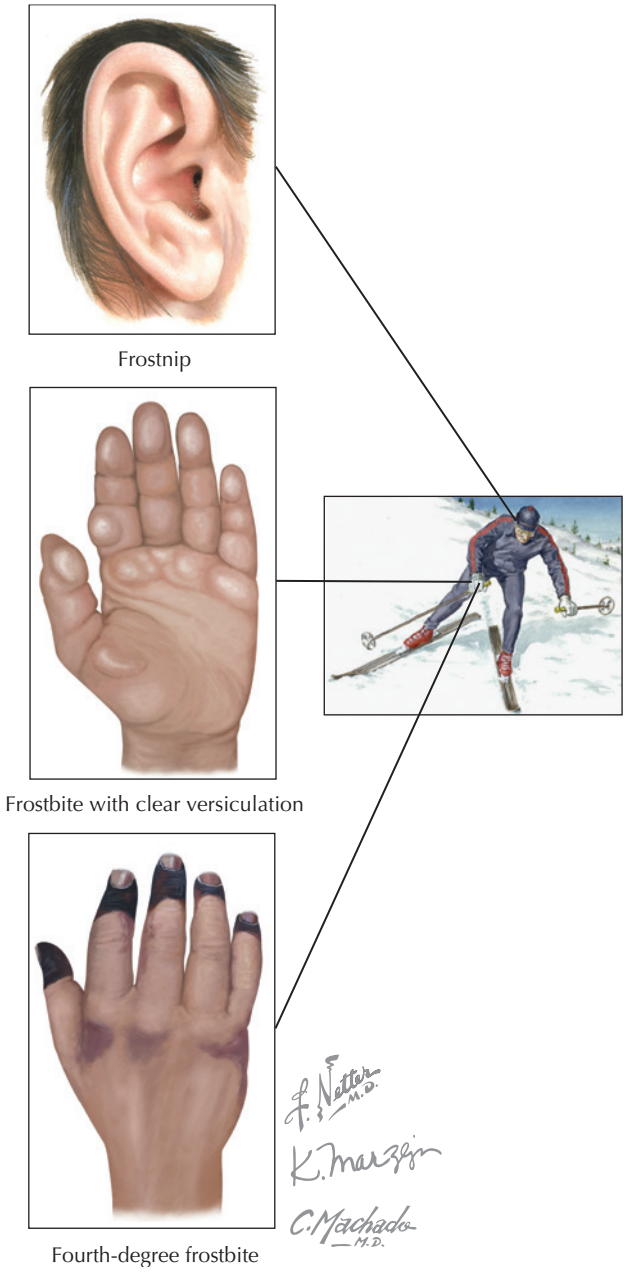


Figure 34-3 Common Cold-Related Injuries.

INFECTIONS

Fungal Infections

Tinea Pedis (Athlete's Foot)

Overview: Erythematous, often scaling eruptions on the plantar surface of the foot and between the toes. Caused by a variety of dermatophytes and yeasts (most commonly caused by *Trichophyton rubrum*, *T. mentagraphytes*, and *Epidermophyton floccusum;* sometimes *Candida*). Seen especially in those with chronically wet feet, diabetics, and immune system failure; athletes exposed to locker room and public shower floors are also at risk.
Presentation: The athlete may present after a gradual onset of pain, itch, and rash on the feet; may have tried and failed numerous over-the-counter preparations.
Physical exam: Typical lesions seen on the feet particularly between the toes; secondary bacterial infections are often present; skin is often macerated and fissures may be seen.

Diagnosis: Diagnose by typical clinical presentation, skin KOH scrapings, or fungal cultures.

Treatment:

- Over-the-counter preparations: Lotrimin Ultra (butenafine) or LamisilAT (terbinafine) are fungicidal against both dermatophytes and yeasts, but need to be used twice daily for 2 to 3 weeks, or until 7 days after rash has resolved. Yeasts do not respond to some over-the-counter preparations.
- Prescription topical preparations: Econazole (Spectazole) and oxiconazole nitrate (Oxistat) are effective against both dermatophytes and yeasts, are once daily preparations, and may deliver cure within a week.
- Difficult cases may require oral antifungal agents: terbinafine 250 mg daily for 2 weeks, or fluconazole 150 mg once per week for 2 to 4 weeks.
- Treat secondary bacterial infections with cephalexin 500 mg three times daily for 7 days.
- Keep feet clean and dry by changing damp socks often, using breathable shoes, and drying powders.
- Wear sandals or water shoes in public showers.

Return to play: As tolerated.

Tinea Cruris (Jock Itch)

Overview: A reddened scaly patch with sharp margins that begins in the moist, warm crural folds and spreads out toward the thigh in a fan-shaped pattern; may be spread to the groin by contamination from infected feet (Fig. 34-4).

Presentation: May be painful, pruritic, and weeping. Similar organisms as tinea pedis (most commonly caused by *Trichophyton rubrum*, *T. mentagraphytes*, and *Epidermophyton floccusum;* sometimes *Candida*). Seen in athletes who share undergarments, wear tight-fitting synthetic garments, or have prolonged exposure to moisture in the groin.

Physical exam: Typical lesions identified in the groin, particularly in the crural folds, that rarely involve the scrotum (see "Erythrasma").

Diagnosis: By typical clinical presentation, skin KOH scrapings, or fungal cultures.

Treatment: Similar to tinea pedis. Avoid corn starch because of its conversion to sugars that may act as a media. Use antihistamines or low-potency steroid creams to control itch. Loose-fitting, absorbent undergarments can help. Don socks before underwear. Change and shower soon after workout. Use powders to keep dry.

Return to play: As tolerated.

Tinea Corpus (Ringworm, Tinea Gladiatorum)

Overview: Fungal infection of the skin that causes a ringlike rash with small red papules, blisters, or scales (caused by the dermatophytes *Microsporum*, *Trichophyton*, and *Epidermophyton*) (see Fig. 34-4). Usually more tissue reaction at the advancing borders of infection, which accounts for the ringlike appearance, Sweating, heat, and physical exertion contribute to fungal growth so sports with close skin contact or communal mats are at risk (e.g., wrestling, martial arts, gymnastics).

Presentation: Presents with an enlarging itchy red rash, gradual in onset, and present for days to months. Usually seen on hairless portions of skin. May be spread by mats, equipment, animals, and clothing.

Physical exam: Presents as an itchy red rash with a central area of hypopigmentation and an advancing erythematous, scaling border of varying size, usually 1 to 5 cm. There may be multiple lesions on the skin in multiple stages of development.

Diagnosis: By typical clinical presentation, skin KOH scrapings, or fungal cultures.

Treatment: Similar to tinea pedis. To prevent transmission, cover with occlusive dressing for contact sports. Use proper treatment of mats and equipment with fungicidal cleaners after each practice/meet. Enforcement of skin infection rules. Prompt treatment and isolation.

Return to play: When the lesion is no longer scaling and red; as early as 3 to 5 days after treatment is initiated. May be able to cover the lesion in some cases to allow earlier return to play.

Tinea Versicolor

Overview: Common fungal infection of the skin; most commonly noted as macular hypopigmented or hyperpigmented lesions on the nape of the neck extending onto the trunk and arms (see Fig. 34-4). Pathogens include *Pityrosporum orbiculare* and *P. ovale* (both were previously called *Malassezia furfur*). More common in males.

Presentation: A painless hypopigmented or hyperpigmented rash that does not itch; may have been present for years and incidentally found on physical exam.

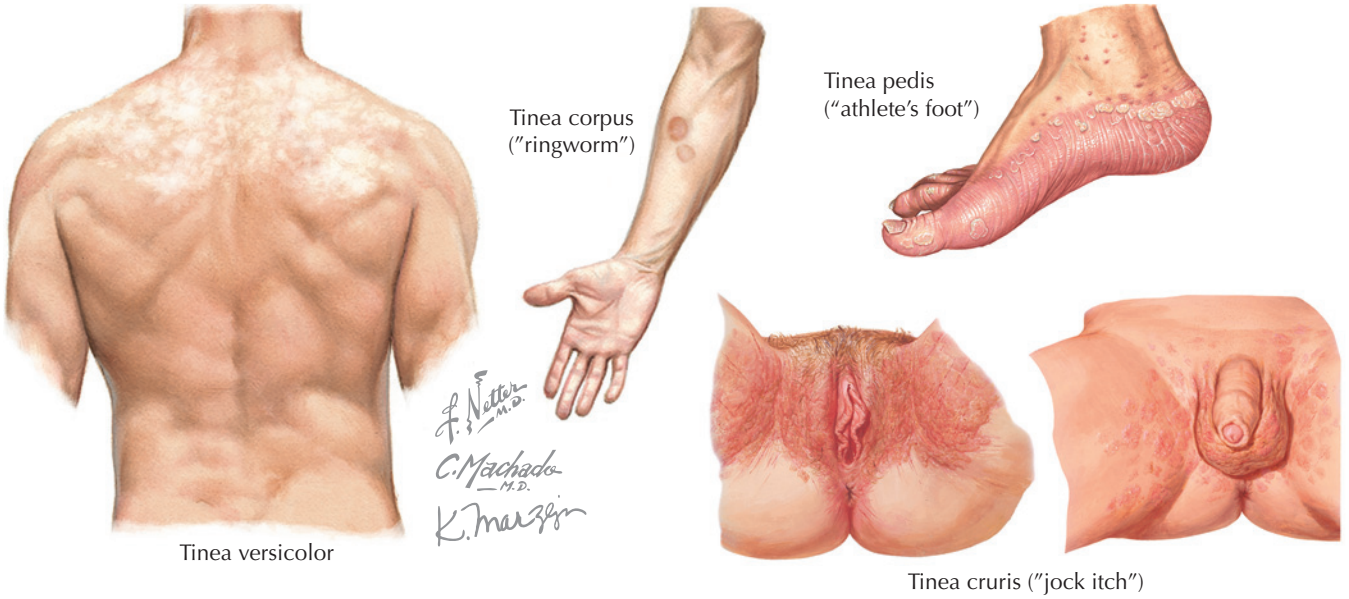


Figure 34-4 Common Fungal Infections.

Physical exam: Macular hypopigmented or hyperpigmented lesions that start on the nape of the neck and may extend onto the shoulders, trunk, and arms in a fan-shaped pattern.
Diagnosis: By typical lesions and/or pathopneumonic spaghetti-and-meatball appearance on KOH scrapings.
Treatment:
- Treatment consists of antifungal or selenium-based over-the-counter shampoo on the skin and hair daily for 2 to 4 weeks, or 2% ketoconazole cream applied daily for 2 weeks.
- Oral antifungal agents may be needed in more difficult cases: ketoconazole 200 mg daily for 7 days or itraconazole 400 mg daily for 3 to 7 days.
  - Application prior to workouts may increase skin concentrations.
- Hypopigmented regions may take 6 to 8 weeks to return to normal.
- Preventive treatment includes good hygiene with showering immediately after workouts, clean dry undershirts, and use of selenium-based or antifungal shampoo daily (may also use as a body wash).

Return to play: Immediately, without risk of infecting others.

Intertrigo

Overview: Red, macerated, half moon–shaped plaques found in the moist body folds. Caused by moist irritation allowing mixed infections of dermatophytes, bacteria, and yeast to infect the superficial layer of skin. Obesity is a risk factor.
Presentation: Athletes may present with a long-standing discoloration in their axilla, groin, or under their breasts.
Physical exam: Typical lesions are seen in the moist skin folds and may extend onto the scrotum.
Diagnosis: By typical lesions with a supporting history.
Treatment: Similar to tinea cruris. Avoid corn starch because of its conversion to sugars that act as a media. Mild cortisone or antihistamine cream may improve itch. Healthy weight and good hygiene should be maintained.
Return to play: Without restrictions.

Bacterial Infections

Acne Mechanica (Football Acne)

Overview: Typical infections consist of inflammatory papules and pustules, comedones, nodules, and/or cysts (Fig. 34-5). Located primarily on the face, back, shoulders, and chest; caused by a variety of bacteria. Worsened by sweat and occlusion from equipment that irritates the skin. Flare often seen in athletes with preexisting acne.
Presentation: Besides football players, may be seen in weightlifters from weight bench, golfers from carrying bag, hockey players from shoulder pads, and dancers from leotards; commonly seen under helmets, chin straps, shoulder pads, jock straps, etc. Causative factors include heat, occlusion, and pressure along with friction.
Diagnosis: Diagnosis is by typical lesions in moist areas under protective equipment or uniform.
Treatment:
- Area of involvement should be cleansed thoroughly immediately after workout with mildly abrasive cleanser (good hygiene).
- Avoid overdrying or scrubbing the skin.
- Protect from equipment; use **clean** absorbent underwear beneath pads.
- Use properly fitted and clean equipment.
- Apply topical astringent or 10% benzoyl peroxide.
- Apply topical 3% erythromycin plus 5% benzoyl peroxide twice daily.
- When severe may add oral antibiotics (doxycycline 100 mg twice daily or minocycline 50 mg twice daily).
- Usually greatly improves or resolves after sport season.

Furuncle

Overview: Also known as boil, furunculosis, or carbuncle. Usually begins as a *Staphylococcus* infection of the hair follicle, which then invades the surrounding tissues causing a locculated collection of pus. Multiple boils are called furunculosis and if they interconnect, a carbuncle.
Presentation: Athlete may describe a "pimple" that enlarges over days that is painful, warm, and may be draining.
Physical exam: Usually located on the upper extremity, buttocks, groin, axilla, neck, waist, or chest. Boils on the face are of particular concern because the venous access to the brain.
Diagnosis: Always consider obtaining cultures and sensitivities to guide appropriate antibiotic choice and avoid resistance. Assess for methicillin-resistant *Staphylococcus aureus* (MRSA).
Treatment: Boils will usually rupture on their own. If persistent, large, painful, or show signs of sepsis, they should be lanced under a physician's care, cultures taken, and oral antibiotics initi-

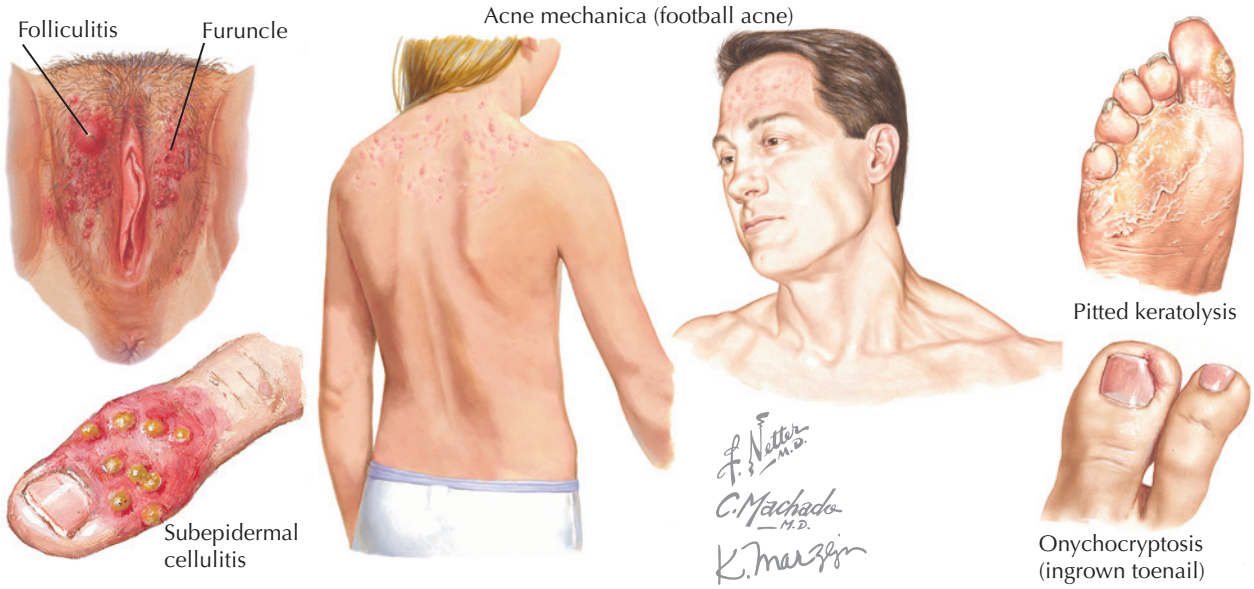


Figure 34-5 Common Bacterial Infections.

ated. Close follow-up to assess response to treatment and insure clinical cure.

Return to play: Avoid contact with other players, shared equipment, and mats until no longer infective (dry with no discharge).

Occlusive Folliculitis (Bikini Bottom)

Overview: Common infection of the hair follicles, often by *Staphylococcus aureus*, *Candida*, *Pseudomonas aeruginosa*, *demodex* and other skin infectants. May present as macules, papules, pustules, or sometimes crusted lesions at the base of the hair shaft that may produce boils or carbuncles. Tend to be worse in the summer and with spandex fiber clothing. Often seen in sports that require/promote shaving of the body (running sports, wrestling, football, and swimming).

Hot tub folliculitis: Caused by a *Pseudomonas aeruginosa* infection from exposure to infected water in hot tub or whirlpool. Generally affect the axillae, breast, and pubic area but occasionally the trunk. Usually resolves on own after 7 to 10 days.

Steroid folliculitis: Associated with use of anabolic steroids. Usually affects the trunk and occasionally the neck and face. Treatment is with cessation of steroids; treat like folliculitis.

Presentation: May be due to friction from pads, prolonged exposure to wet or sweaty bathing suit, or shaving.

Physical exam: Often seen on the back, thighs, or buttocks (inferior gluteal fold especially affected in swimmers). Area of involvement coincides with overlying equipment.

Diagnosis: Typical lesions in shaven areas with increased friction or with a history of hot tub or steroid use.

Treatment:

- Usually resolves once the irritating source is removed.
- Topical antiseptics such as benzoyl peroxide (Oxy-10).
- Use of antibacterial soaps or topical antibacterial ointment (mupirocin) or topical antibiotic solutions such as clindamycin phosphate (Cleocin T) or erythromycin (Erygel) to areas of involvement.
- Refractory cases may need oral antibiotics.
- Aggressive treatment may preclude appearance of folliculitis keloidalis.
- Remove equipment or swimwear as soon as possible after workout.
- Shower immediately after workouts.
- Keep areas clean and dry.
- Shave in the direction of hair growth.
- After shaving apply an alcohol-based aftershave.

Impetigo

Overview: Highly contagious *Streptococcus* or *Staphylococcus* infection of the skin. Characterized by small vesicles that form pustules and eventually become honey-colored weeping crusts. May cause problems with the kidney and, rarely, the heart. Close contact sports such as martial arts, wrestling, and rugby are at risk.

Presentation: Transmitted by direct contact with infected skin, towels, or equipment. Can be spread by scratching. Should be treated immediately.

Physical exam: Typically seen around the mouth and nose but can be found anywhere on the body.

Diagnosis: Appearance of typical lesions; sometimes difficult to differentiate from herpes labialis. Cultures are rarely needed unless MRSA is suspected.

Treatment: Topical antibiotics are usually sufficient. If in difficult areas to treat topically or if extensive, may treat with oral antibiotics.

Return to play: Withhold return to contact with other players, shared equipment, and mats until no longer weeping or crusted.

Cellulitis

Overview: Infection of the dermis and subcutaneous tissue usually caused by group A *Streptococcus* and *Staphylococcus aureus*. May be life threatening if it involves the face, airway, or leads to sepsis. Includes erysipelas, Ludwig's angina, and many other variants.

Presentation: Athlete usually complains of a gradual onset of pain and swelling over a circumscribed area, sometimes after a puncture, laceration, abrasion, or pustule.

Diagnosis: Made by identifying typical erythema and edema, sometimes with fever and sometimes with a history of a nidus.

Treatment: Should be treated immediately. If not severe, use of oral antibiotics is appropriate. If severe, use IV antibiotics. Warm compresses, pain medication, and if extensive, irrigation and drainage. Consider immediate blood cultures.

Return to play: When afebrile, pain-free, and as tolerated.

Onychocryptosis (Ingrown Toenail)

Overview: Infection of the subcutaneous tissue beneath the toenail; usually caused by *Streptococcus* species and *Staphylococcus aureus* (see Fig. 34-5).

Presentation: Athlete usually presents with pain and purulent discharge at the edge of the advancing nail over the course of weeks to days; often associated with improper trimming of the nails.

Diagnosis: Typical lesions are diagnostic.

Treatment: Oral antibiotics should be started immediately, warm compresses, pain medication. If extensive or very painful, toenail removal. Prevent future problems with proper trimming of nails. Shoes with adequate room in the toe box should be worn.

Return to play: May return to play as soon as they can perform functionally and tolerate the pain.

Pitted Keratolysis

Overview: Also known as stinky foot or tennis shoe foot. Characterized by many circular or longitudinal, punched-out depressions on the sole of the foot (see Fig. 34-5). Most cases are asymptomatic, but painful, plaque-like lesions may occur. *Dermatophilus congolensis* and *Micrococcus sedentarius* produce and excrete exoenzymes (keratinase) that are able to degrade keratin and produce pitting in the stratum corneum. Hyperhidrosis, moist socks, or immersion of the feet favors its development.

Presentation: Athlete may complain of foul-smelling feet and depressions on the sole of the foot present for months to years.

Diagnosis: Typical lesions, foul odor, and supporting history.

Treatment: Promoting dryness: socks should be changed frequently, feet kept clean and dry. Rapid clearing occurs with application of 20% aluminum chloride (Drysol) twice a day. Application twice a day of alcohol-based benzoyl peroxide (Panoxyl 5) may also be useful. Treatment with topical erythromycin 2% gel or 1.5% solution twice daily is also curative.

Return to play: May return without risk of infecting others.

Erythrasma

Overview: Bacterial infection *(Corynebacterium minutissimum)* may be confused with tinea cruris because of the similar, half moon–shaped plaque. Noninflammatory, it is uniformly brown and scaly, and it has no advancing border.

Presentation: Athletes may not even notice the rash and it may only be picked up on during preparticipation physical exam.

Physical exam: Uniform brown, scaly, macular rash in interdigital toe spaces, axilla, and/or groin.

Diagnosis: Typical lesions that fluoresce under Wood's light.

Treatment: Responds equally well to erythromycin orally 250 mg four times a day for 2 weeks or topical erythromycin 2% gel or 1.5% solution twice daily for 2 weeks. Topical erythromycins may be irritating when applied to the groin.

Return to play: May return to play without risk of infecting others.

Viral Infections

Verruca Vulgaris (Warts)

Overview: Caused by human papilloma viral infection of the epidermis. May occur anywhere on the body; appears as a rough hyperkeratotic area that can become quite large.

Presentation: Athletes may present with a painless lesion most often seen on the hands that has been present for weeks to months. They may have tried and failed treating the wart with cutting, scraping, or over-the-counter wart treatments.

Diagnosis: Lesion lacks normal skin lines and can be differentiated from other lesions by this fact; may have black dots in the center from the ruptured blood vessels present there.

Treatment: Usually will regress spontaneously. May treat with cryotherapy, topical abrasives, laser, or immunotherapy. The simplest method is duct tape to occlude the lesion for 1 to 2 weeks if the anatomical location is amenable to this (such as a digit). Use of Tagamet has *not* been shown to be of benefit. There is a low risk of transmission, but the lesion should be kept covered to prevent exposure to others.

Return to play: Without restriction typically.

Verruca Plantaris (Plantar Wart)

Overview: Caused by a viral infection of the plantar surface of the foot; may be painful and cause gait abnormalities.

Presentation: The athlete may present with a painful lesion on the plantar surface of the foot, usually over the heel or ball of the foot. Lesion is usually painful with weight bearing; may have been present for weeks to months.

Physical exam: Lesion lacks normal skin lines and can be differentiated from other lesions by this fact; may have black dots in the center from the ruptured blood vessels present there.

Diagnosis: Similar to warts.

Treatment: Treat early rather than late. May use a doughnut-shaped pad during the season and treat at the end of the season. Duofilm with 17% salicylic acid nightly can be used during the season. Topical over-the-counter preparations may take weeks for cure. Can be treated in the office with liquid nitrogen, cautery, or 40% salicylic acid. Topical trichloracetic acid and debridement every 2 to 3 weeks is relatively painless and effective and usually does not disrupt play.

Return to play: Return to play as tolerated, with use of sandals in public showers to help prevent transmission.

Molluscum Contagiosum (Water Warts)

Overview: This viral pathogen is much more infectious than normal warts, thus its name. Transmission is through close physical contact or autoinoculation. Appears as a flesh- or yellowish-colored papular lesion with a collapsed center. Seen especially on the hands, face, and upper body and more prevalent in contact sports such as wrestling, boxing, and rugby. Sharing razors can spread lesions.

Presentation: The athlete may present with painless lesions that have been present for weeks to months and may be spreading.

Physical exam: Umbilicated lesions, usually found about the hands, face, perinaval area, or groin.

Diagnosis: Typical appearance. Usually athlete does not give a history of contact with infected person, but often are in a sport with close physical contact.

Treatment: Usually resolve spontaneously after 6 to 9 months. Cryotherapy, topical salicylic acid, curettage, or excision by a physician may be required. It is not unusual for these lesions to spread after incomplete treatment or shaving.

Return to play: Restriction with some sports, especially wrestling, until completely resolved.

Herpes Gladiatorum (Traumatic Herpes)

Overview: Caused by herpes simplex virus (HSV) that is directly inoculated onto the skin. Appears as blisters that rupture to form a crusted surface. Can be provoked by physical stress (workouts), emotional stress, and sun exposure. Sports with close physical contact such as wrestling, boxing, and rugby are at higher risk.

Presentation: May be preceded by an itching or burning sensation. Maximally contagious for 5 days after blisters rupture; infectious until lesions are crusted over. May last for 1 to 2 weeks. Carried inactively by the majority of the population.

Physical exam: Vesicles may be noted, but often they are already ruptured with an ulcerated base sometimes with purulent discharge. Athletes may try numerous methods to cure or hide the lesions such as bleach, excoriation, and makeup.

Diagnosis: Typical lesions, but cultures may be taken. If cultures are taken, understand that this may delay return to play for days if treatment not initiated.

Treatment: Antiherpetic antiviral agents for 7 to 10 days. For those with more than six outbreaks per year consider prophylactic treatment.

Return to play: Specific return-to-play rules for some sports, but lesions must be healed and dry before other athletes are exposed. Lesions can be covered for casual contact sports, but because of the permanence of the infection, athletes in direct-contact sports with active lesions should not participate.

Herpes Labialis (Fever Blister)

Overview: Caused by herpes simplex virus (HSV) that is directly inoculated onto the skin by contact with infected host. Appears as blisters around the lips that rupture to form a crusted surface. Can be provoked by physical stress (workouts), emotional stress, and sun exposure. Sports with close physical contact sports such as wrestling, boxing, and rugby are at higher risk.

Presentation: May be preceded by an itching or burning sensation. Maximally contagious for 5 days after blisters rupture; infectious until lesions are crusted over. May last for 1 to 2 weeks. Carried inactively by the majority of the population.

Physical exam: Vesicles may be noted about the lips, but often they are already ruptured with an ulcerated base, sometimes with purulent discharge.

Diagnosis: Typical lesions, but can be confused with impetigo (cultures may be required to differentiate).

Treatment: Start antiviral antiherpetic agents as soon as the tingling and burning begin.

Return to play: There are specific return-to-play rules for some sports, but lesions must be healed and dry before other athletes are exposed.

Parasitic Infections

Scabies

Overview: Caused by the mite *Sarcoptes scabiei* var. *hominis*. African Americans rarely acquire scabies. Lesions are often vesicular or pustular and may have associated elevated "burrows." Wrestlers are at increased risk and scabies may spread through an entire wrestling team in a matter of days.

Presentation: Transmission occurs during direct skin contact with an infected person, dog, or cat. After 6 to 8 weeks becomes widespread, with intensely pruritic eruption. Patient may be comfortable during the day but itch at night (nocturnal pruritis). A mite can possibly survive for days in normal home surroundings but whether it can be acquired from bed linens or clothing is unknown.

Physical exam: Typically found in the finger webs, wrists, extensor surfaces of the elbows and knees, sides of the hands and feet, axillary areas, buttocks, waist area, and ankle area. In men, the penis and scrotum are usually involved; in women, the breast,

including the areola and nipple. Burrows may be seen on the hands, feet, and groin.

Diagnosis: Suspected when burrows are found or when a patient has typical symptoms with characteristic lesions and distribution. Definitive diagnosis is made with microscopically identified mites, eggs, egg casings, or feces (scybala).

Treatment:

- Permethrin 5% cream (Elimite cream) is the scabicide of choice; apply chin to toes, leave on 10 hours, repeat in 1 week.
  - One application is highly effective (91% resolution).
- Ivermectin given in a single oral dose (200 μg/kg) was found to be effective and safe.
- Lindane 1% cream (Kwell) (86% resolution) may be used for children of all ages and pregnant and nursing mothers.
- Team members with close contact and all members in the same household should be treated.
- Itching may continue for 1 to 2 weeks after treatment complete (because of dead mites and feces in burrows).
- Athletes should wash all clothing, towels, and bed linen (in a normal washing machine cycle) that have touched the skin.
- Emphasize that coats, furniture, rugs, floors, mats, and walls do not need to be cleaned in any special manner.

Return to play: May return to play 24 hours after treatment with permethrin or ivermectin and reinfection is not likely (linens washed and household members and teammates treated).

ALLERGIC REACTIONS AND DERMATITIS

Cold Urticaria

Overview: Papular erythema with dermal edema; papules smaller than with standard urticaria and wheal less pronounced. Cholinergic urticaria induced by factors common to athletes, such as rapid temperature changes, especially cold to hot; emotional stress; and physical exertion. Cold urticaria is nearly disabling to swimmers.

Presentation: Athlete presents with intense itching after exposure to cold air, water, or equipment.

Physical exam: Inner aspects of arms and legs and lateral flanks are common sites. Elevated wheal where cold exposure occurred.

Diagnosis: Made by observing typical lesions with a supporting history.

Treatment: Cyproheptadine (Periactin) taken regularly at bedtime seems particularly helpful. Combination therapy with h_1- and h_2-inhibiting antihistamines often works better than single-drug regimen (hydroxyzine plus doxepin). Alternate-day steroids may be required in extreme cases. Warm or cool body temperature very slowly; use biofeedback techniques for stress reduction.

Return to play: Only if able to protect the athlete, treated acceptably, or if very mild disease.

Contact Dermatitis

Overview: Varying degrees of erythema initially, with scaling and exudate later. Vesicle formation in acute cases, with strong sensitivity. People with no known allergens may experience contactants unique to athletic environment such as nickel in shoe eyelets, elastic in glove cuffs, tanning products in baseball or softball gloves, etc.

Presentation: Athletes usually present acutely after rash has been present for days with a pruritic, sometimes painful erythematous and sometimes vesicular rash.

Physical exam: Sharp margins that configure to the offending equipment is hallmark finding. Often found to be caused by shoe eyelets, metal in equipment, or latex or elastic in uniform or socks.

Diagnosis: Made by observing typical lesions with a supporting history.

Treatment:

- Corticosteroids are cornerstone of therapy.
- Gel, lotion, and spray preparations are especially effective topically.
- Use systemic "burst" of prednisone in acute cases.
- Parenteral steroids often faster-acting and eliminate problems with patient compliance.
- Antihistamines diminish reaction and relieve itching.
- Identification and elimination of allergen key to resolution.
- Barrier between skin and causative factor may include T-shirt or leggings under pads, nail polish over metal surfaces, and cordran tape for small areas.
- Special equipment such as allergy-free shoes may eliminate problem but are usually expensive.

Return to play: As tolerated.

RECOMMENDED READINGS

1. Gilbert DN, Moellering Jr RC, Eliopoulos GM, Sande MA: The Sanford Guide to Antimicrobial Therapy, 36th ed., Hyde Park, Antimicrobial Therapy, Inc., 2006.
2. Habif, TI: Clinical Dermatology: A Color Guide to Diagnosis and Therapy, 4th ed., Philadelphia, Mosby, Elsevier Science, 2003.

Connective Tissue and Rheumatologic Conditions in Sports

35

Mark E. Lavallee

CONNECTIVE TISSUE DISORDERS

Marfan Syndrome

Overview: Inheritable autosomal-dominant genetic condition affecting the processing of fibrillin. Caused by more than 400 mutations in the gene encoding fibrillin-1 (FBN-1) located on chromosome 15 at q21 loci. Incidence is 1 in 3000 to 10,000 live births; an estimated 200,000 Americans have Marfan syndrome. Fibrillin, which is the major component of microfibrils, is found in large amounts in the aortic root>aorta> lens>joints> other connective tissues.

Presentation: Athletes with Marfan syndrome may (Fig. 35-1):

- Be tall (97th percentile or above) and thin.
- Have long thin arms, legs, hands, and feet.
- Present with sudden death (aortic root dissection).
- Complain of joint laxity (including joint subluxations and dislocations).
- Have joint pain.
- Have scoliosis (60% have curvature greater than 20 degrees).
- Have visual problems.
- Have dental crowding.
- Be drawn to sports where tall, slender build is an advantage (basketball, volleyball, rowing, and track).
- Famous athletes with Marfan syndrome include Flo Hyman (U.S. 1984 Olympic Silver Medalist in Volleyball—died of aortic dissection).

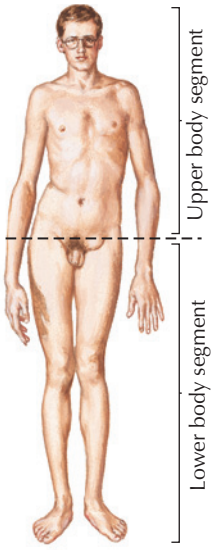
Physical exam: See Table 35-1.

Diagnostics:

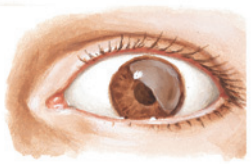
- Echocardiogram: aortic root dilation, aortic dissection, valvular issues (e.g., mitral valve prolapse [MVP]).
- Slit-lamp exam: look for lens abnormalities.
- Imaging of chest and abdomen: CT scan with IV contrast, MRI with IV contrast, ultrasound.
- Genetic testing: via blood or tissue sample for FBN-1 mutation.
- Using diagnostic criteria (see Table 35-1)
  - Index case: if no family history, then need major criteria in two organ systems and minor criteria in third organ system.
  - Relative of index case: presence of one major criterion in family history *and* one major criterion in one organ system and involvement in a second organ system.

Treatment:

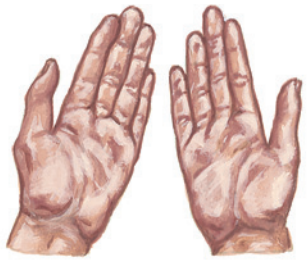
- General: Healthy lifestyle including exercise and diet to control lipids and systemic blood pressure. Medic-alert bracelet/necklace stating condition.



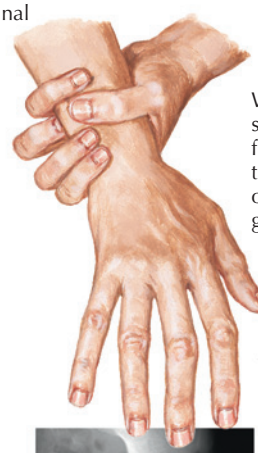
Tall, thin person with skeletal disproportion. Upper body segment (top of head to pubis) shorter than lower body segment (pubis to soles of feet). Fingertips reach almost to knees (arm span-to-height ratio greater than 1.05). Long, thin fingers (arachnodactyly). Scoliosis, chest deformity, inguinal hernia, flatfoot



Ectopia lentis (upward and temporal displacement of eye lens). Retinal detachment, myopia, and other ocular complications may occur.

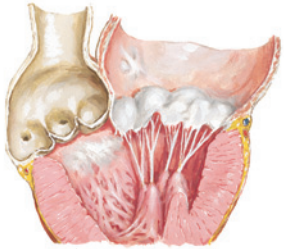


Arachnodactyly of hands

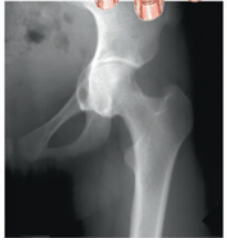


Walker-Murdoch wrist sign. Because of long fingers and thin forearm, thumb and little finger overlap when patient grasps wrist.

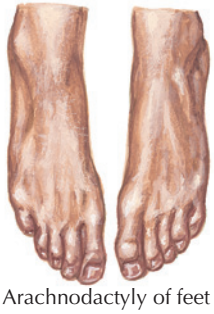
Steinberg sign. Tip of thumb protrudes when thumb folded inside fist. Thumb and index finger overlap when encircling opposite wrist.



Dilatation of aortic ring and aneurysm of ascending aorta due to cystic medial necrosis cause aortic insufficiency. Mitral valve prolapse causes regurgitation. Heart failure common



Radiograph shows acetabular protrusion (unilateral or bilateral).



Arachnodactyly of feet

Figure 35-1 Characteristics of Marfan Syndrome.

Table 35-1 PHYSICAL EXAM FOR MARFAN SYNDROME

Exam	Major criteria	Minor criteria
Skeletal *(need 2 major OR 1 major and 2 minor)*	Tall (>97th percentile) Arm span to height ratio > 1.05:1 Increased carrying angle at elbows (<170 degrees of extension) Arachnodactyly Pectus Deformity: (carinatum or excavatum requiring surgery) Thumb sign (flex thumb across where full thumbnail exposed) Walker-Murdoch wrist sign (able to overlap thumb and fifth finger around wrist) Scoliosis/kyphosis Pes planus (flat feet)	Joint laxity and hypermobility Beighton Scale (a.k.a. Carter-Wilkinson criteria) Pectus excavatum High arched palate and dental crowding Facial appearance (dolichocephaly, malar hypoplasia)
Ophthalmologic *(need 2 of minor criteria, if no major)*	Ectopic lentis or lens dislocation	Abnormally flat cornea Increased axial length of globe (via ultrasound) Hypoplastic iris or ciliary muscles Nearsightedness
Cardiovascular *(need only 1 major or 1 minor)*	Dilation of ascending aorta Ascending aortic dissection	Mitral valve prolapse (w/ or w/o regurgitation) Dilation of pulmonary artery (age < 40 and no pulmonary stenosis) Calcified mitral annulus (age < 40) Aortic insufficiency
Pulmonary *(need only 1 minor)*	None	Spontaneous pneumothorax Apical blebs on chest radiograph
Dermatologic *(need only 1 minor)*	None	Recurrent or incisional hernias Striae atrophicae (stretch marks) not associated with weight change or pregnancy
Spine/Dura *(need only 1 major)*	Lumbosacral dural ectasia as seen on CT or MRI *(enlargement of neural canal)*	None
Family/Genetic History *(need only 1 major)*	Parent, child, or sibling that meets criteria for Marfan Presence of FBN-1 mutation known to cause Marfan Presence of haplotype around FBN-1 in a family with Marfan	None

- Prevention: Annual evaluation of eyes (ectopic lentis or lens dislocation), heart (valvular issues, aortic root dilatation or dissection), imaging of chest and abdomen (identifying/following aortic dissections, aneurysms, or dilatation).
- Vascular: Monitor any changes in aorta with annual echocardiogram, avoid strenuous activities that will increase intrathoracic pressure (e.g., powerlifting). If aortic pathology worsens, consider intraluminal prosthetic device versus elective open surgical replacement of aorta. Use of a beta-blocker has been correlated with increased survival. Age in which to start beta-blocker is still debated. Valve replacement surgery is also an option, but it is not without severe risks for those with aortic valve insufficiency and ascending aorta dilation.
- Orthopedic: Mild strength training to stabilize severe joint laxity. Recommend against intense strength training involving Valsalva or increase in systolic blood pressure. Joint preservation and protection often includes avoidance of contact sports. Physical therapy (especially joint and core strengthening, proprioceptive training, modalities, strengthening).
- Psychology: Provide empathy and psychological support as per individual's and family's needs. Genetic counseling is always strongly advised, as is putting patient in contact with knowledgeable medical professionals and local/national support groups such as the National Marfan Foundation at www.marfan.org.
- Pain control: Encourage smoking cessation; control pain with oral, injectable, transdermal, and topical medicines to include acetaminophen, nonsteroidal anti-inflammatory drugs (NSAIDs), opioids, tramadol, transcutaneous electrical nerve stimulation (TENS) units, bracing, ring splints, lastly surgery.

Prognosis: In 1972, the mean age of death was 32 years (range 16-48 yrs). By 1995, the median cumulative probability of survival was 72 years.

Return to play:

- 36th Bethesda American College of Cardiology recommendations (2005):
  - May participate in low-static, low-dynamic competitive sports if they *do not* have one of the following: aortic root dilation, moderate-to-severe mitral valve regurgitation, or family history of aortic dissection or sudden death.
  - Athletes with aortic root dilation may only participate in low-intensity competitive sports.
  - Avoid sports with bodily collision.
  - Same recommendation for aortic valve regurgitation.
  - Prefer nonstrenuous, low-static, low-dynamic sports (e.g., golf, walking, billiards, riflery, bowling).
- Encourage an active lifestyle.
  - Favor noncompetitive, isokinetic exercise performed at a nonstrenuous aerobic pace.
  - Avoid activities that involve high levels of isometric workloads (e.g., Olympic weightlifting, gymnastics, etc.).
  - Mild strength training (non-breathholding) if joint laxity present. Prefer multiple repetitions at lower resistance rather than maximal repetitions at higher resistance.
  - Be careful in environments with rapid atmospheric pressure changes (scuba, flying in an unpressurized cabin) or rapid decelerations (car racing, skydiving).
  - Control lipids, blood pressure, blood sugar to decrease long-term injury to vascular endothelium.
  - Educate patient as to risks of participation.
  - Use protective eyewear whenever appropriate.
- Once orthopedic or ocular injury has healed and the injured area adequately protected, the athlete may return to sport. Support patient with reasonable adaptive measures in order to participate.

Ehlers-Danlos Syndrome (EDS)

Overview: Group of inheritable genetic conditions that affect connective tissue, especially collagen in joints, vessels, skin, and internal organs. Variable severity seen within each type and within the same family of pedigree. The defect in the genes for collagen-processing COL1A and COL3A genes is found on the long arm of chromosome 2. There are six types of Ehlers-Danlos syndrome, of which 90% seem to fall into the first three types (hypermobile, classical, vascular). Incidence is approximately 1 in 5000 live births. The term *generalized* or *benign joint hypermobility syndrome* (GJHS or BJHS) was first used by Kirk, Ansell, and Bywaters in 1967. EDS hypermobile type was described as a condition in 1946 and noted to be genetically based in 1970. The description of BJHS also uses the Beighton Scale and the same diagnostic criteria as EDS hypermobility type. Essentially, they are the same entity with only varying degrees of expressivity. A review of the literature seems to find no significant difference in the nosology between BJHS and aspects of EDS hypermobile type. BJHS seems to be a term preferred by those outside genetic circles (e.g., rheumatology, orthopedics, etc.) and EDS hypermobile type and Marfan syndrome are preferred by general medical and genetic groups.

Presentation: There are six types of EDS:

- **Hypermobile:** Most common type in North America, mild to severe joint laxity, most often involving multiple joints subluxating or dislocating, mild skin involvement, chronic joint pain with often normal imaging studies, Beighton Scale score of 5/9 or greater, 22% risk of developing aortic root dilatation.
- **Classical:** Second most common type in North America; tissue friability; wide, atrophic scars ("cigarette paper scar tissue") may resemble psoriasis; has piezogenic papules and subcutaneous spheroids; often only mild joint laxity, though dislocations are still common; 33% risk of developing aortic root dilation; early osteoarthritis especially in smaller joints.
- **Vascular:** Third most common type in North America, life-threatening (sudden death often from aortic/vascular or bowel rupture). Particular facial features: delicate nose, wide-set eyes, thin cheekbones, thin, translucent skin, very prominent veins on extremities and chest, skin friability, mild to moderate joint laxity. Very significant risk of developing aortic pathology (e.g., root dilatation, aneurysm, dissection).
- **Kyphoscoliotic:** Very rare, has risk of ocular rupture, marfanoid body habitus, diagnosis via urine test available.
- **Dermatosparaxis:** Very rare, doughy, redundant skin, autosomal recessive, diagnosis via skin biopsy.
- **Arthrochalasia:** Very rare, congenital hip dislocation, atrophic scars, joint hypermobility, diagnosis via skin biopsy.

Physical exam: See Figure 35-2.

- Beighton Scale (score > 5/9)
- Excessive subtalar motion
- Pinchable skin in a relaxed palmer aspect of hand
- Pectus deformity: excavatum more common than carinatum
- Touch tip of tongue to nose
- Skin hyperextensibile to greater than 3 cm at angle of mandible
- Widened, atrophic scars (known as "cigarette paper scar tissue") (see Fig. 35-2)
- Piezogenic papules (often found near sole of feet)
- Subcutaneous spheroids (firm, small, mobile subcutaneous)
- Joint pain (acute or chronic, episodic)
- Joint laxity or hypermobility affecting multiple joints
- Joint subluxations and dislocations
- Family history (because most cases have an autosomal-dominant transmission)
- Early osteoarthritis
- Postural orthostatic tachycardic syndrome (POTS)
- Chiari malformation
- Hernias (e.g., ventral, umbilical, inguinal)
- Mitral valve prolapse
- Dental malalignment/gingival issues
- Sudden death (due to vascular or bowel rupture)
- Positive family history of first-degree relative with EDS

Diagnostics: Echocardiogram periodically, CT scan of chest and abdomen with IV contrast or ultrasound periodically, skin punch biopsy (4 mm) put into live medium for EDS and TGF-beta (e.g., *Chlamydia* culture) (currently only for classical and vascular types), and radiographs, and other imaging of affected joints.

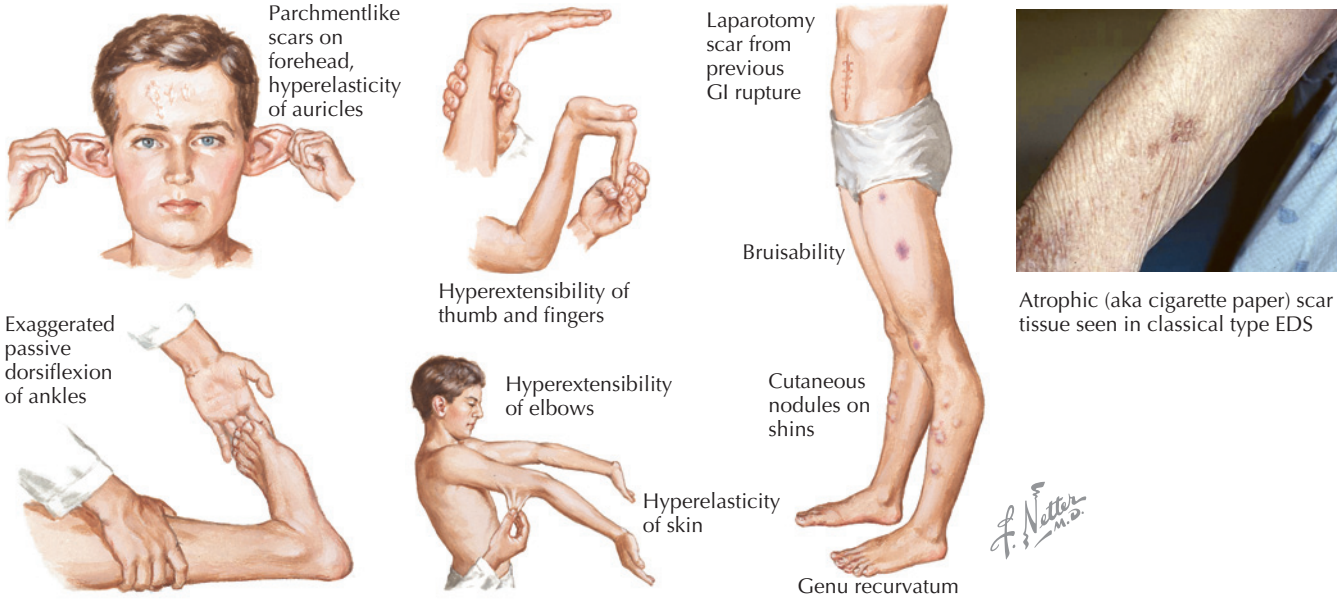


Figure 35-2 Characteristics of Ehlers-Danlos Syndrome. *(Photograph reprinted with permission from Goldman L, Ausiello D, Arend W, et al (eds): Cecil Textbook of Medicine, 23rd ed. Philadelphia: Elsevier, 2007.)*

Treatment:
- General: Healthy lifestyle that includes exercise and diet to control lipids and systemic blood pressure. Medic-alert bracelet/necklace stating condition.
- Prevention: Periodic evaluation of heart (e.g., valvular issues) and chest/abdomen (e.g., identifying/following aortic dissections, aneurysms, or dilatation).
- Vascular: Monitor any changes in aorta, avoid strenuous activities that will increase intrathoracic pressure (e.g., powerlifting). If aortic pathology worsens, consider intraluminal prosthetic device versus elective open surgical replacement of aorta. Encourage smoking cessation.
- Orthopedic: Mild strength training to stabilize severe joint laxity. Recommend against intense strength training involving Valsalva or increase in systolic blood pressure. Joint preservation and protection may often include avoidance of contact sports. Physical therapy (especially joint and core strengthening, proprioceptive training, modalities, strengthening, unweighted exercise (e.g., pool therapy, harnesses, total gym).
- Psychology: Provide empathy and psychological support as per individual's and family's needs. Genetic counseling is always strongly advised, as is putting patient in contact with knowledgeable medical professionals and local/national support groups. Have the athlete visit the Ehlers Danlos National Foundation website at www.ednf.org.
- Pain control: Encourage smoking cessation; control pain with oral, injectable, transdermal, and topical medicines to include acetaminophen, nonsteroidal anti-inflammatory drugs (NSAIDs), opioids, tramadol, TENS units, bracing, ring splints, ambulation-assist devices, and lastly surgery.

Prognosis: Percentage of patients with EDS who will develop pathology within their aortic root or aorta: 90% vascular type, 33% classical type, 22% hypermobile type.

Return to play: Once orthopedic or integument injury has healed and the injured area adequately protected, the athlete may return to sport. Support patient with reasonable adaptive measures in order to participate. According to the 36th Bethesda recommendations (2005):
- Individuals with vascular type EDS should not compete in any form of competitive athletic activity.
- Keep normal weight.
  - Underweight: not enough muscle to stabilize loose joints
  - Overweight: early osteoarthritis
- Encourage an active lifestyle.
- Control lipids, blood pressure, blood sugar to decrease long-term injury to vascular endothelium.
- Mild to moderate strength training is often helpful in joint stabilization.
- Educate patient as to the risks of participation in sport.
- Highly recommend against contact and collision sports, especially for the more severe form of EDS.
- Encourage noncontact, nontraumatic sport, especially for those with skin fragility (i.e., classical type) and severe joint laxity (i.e., hypermobility type).
- Do not recommend high-dynamic sport, which could increase intra-abdominal pressure and blood pressure, secondary to risk of aortic dilatation or colonic rupture.

Osteogenesis Imperfecta

Overview: Also known as OI and brittle bone syndrome, osteogenesis imperfecta is an inheritable genetic disorder that affects collagen (type 1) (Fig. 35-3). A classification system is commonly used to describe how severely a person is affected by OI. The genetic defect (COL1A gene) affects type 1 collagen, which is found primarily in the bone. There are two forms of OI. In the dominant (classical) form the patient has too little type 1 collagen or a poor quality of type 1 collagen. Most cases (80%) are autosomal dominant, though there are autosomal recessive types. There are eight documented types of OI (two types have autosomal recessive expressivity).

Incidence: An estimated 20,000 to 50,000 individuals in the United States have OI.

Presentation:
- Type 1: Most common and mild type.
- Bones fracture easily. Most fractures occur before puberty.
- Normal or near-normal stature.
- Loose joints and muscle weakness.
- Sclera (whites of the eyes) usually have blue, purple, or gray tint.
- Triangular face.
- Tendency toward spinal curvature.
- Bone deformity absent or minimal.
- Brittle teeth possible.
- Hearing loss possible, often beginning in early 20s or 30s.
- Collagen structure is normal, but the amount is less than normal.

Physical exam: See Figure 35-3.
- Dentinogenesis imperfecta (poorly formed teeth)
- Blue sclera
- Bone fragility
- Pathogenic and multiplicity of fractures
- Adult-onset hearing loss
- Osteoporosis
- Joint hyperextensibility
- Short stature/limb shortening or deformity in severe forms

Diagnostics: Radiographs may show bowing deformities and multiple fractures/callus formation. Bone scan may show multiple increased areas of uptake as a result of multiple areas of occult or stress fractures. Dual energy x-ray absorptiometry (DEXA) scan may show premature decrease in bone density consistent with osteopenia or osteoporosis. Of skin punch biopsy tests, both the collagen biopsy test and the DNA test are thought to detect almost 90% of all type I collagen mutations. A negative type I collagen study does not rule out OI. When a type I collagen mutation is not found, other DNA tests to check for recessive forms are available.

Treatment:
- General: Healthy lifestyle including exercise and diet.
- Prevention: Protection or avoidance of contact or collision sports is paramount. Medic-alert bracelet/necklace stating condition and severity is recommended.
- Orthopedic: Mild strength training to stabilize joint laxity. Recommend against intense strength training especially ballistic lifts (e.g., snatch, power cleans, etc.). Physical therapy may help (especially after fractures) with pain control and improvement in function after prolonged immobilization. Often because of the nature of the fracture, open-reduction internal fixation is needed (see Fig. 35-3). Unweighted exercise may be beneficial (e.g., pool therapy, harnesses, total gym). "Off-label" use of bisphosphonates has been advocated by some for between 2 and 5 years.
- Psychology: Provide empathy and psychological support as per individual's and family's needs. Genetic counseling is always strongly advised, as is putting patient in contact with knowledgeable medical professionals and local/national support groups. Have athletes visit the Osteogenesis Imperfecta Foundation website at www.oif.org.
- Pain control: Encourage smoking cessation; control pain with oral, injectable, transdermal, and topical medicines to include acetaminophen, NSAIDs, opioids, tramadol, TENS units, bracing, ring splints, ambulation-assist devices, and lastly surgery.

Prognosis: Generally, patients have a full life expectancy, but morbidity seems to be correlated directly to the number of fractures.

Return to play: Absolutely avoid contact/collision sports. Certain high-impact sports may be contraindicated depending on sever-

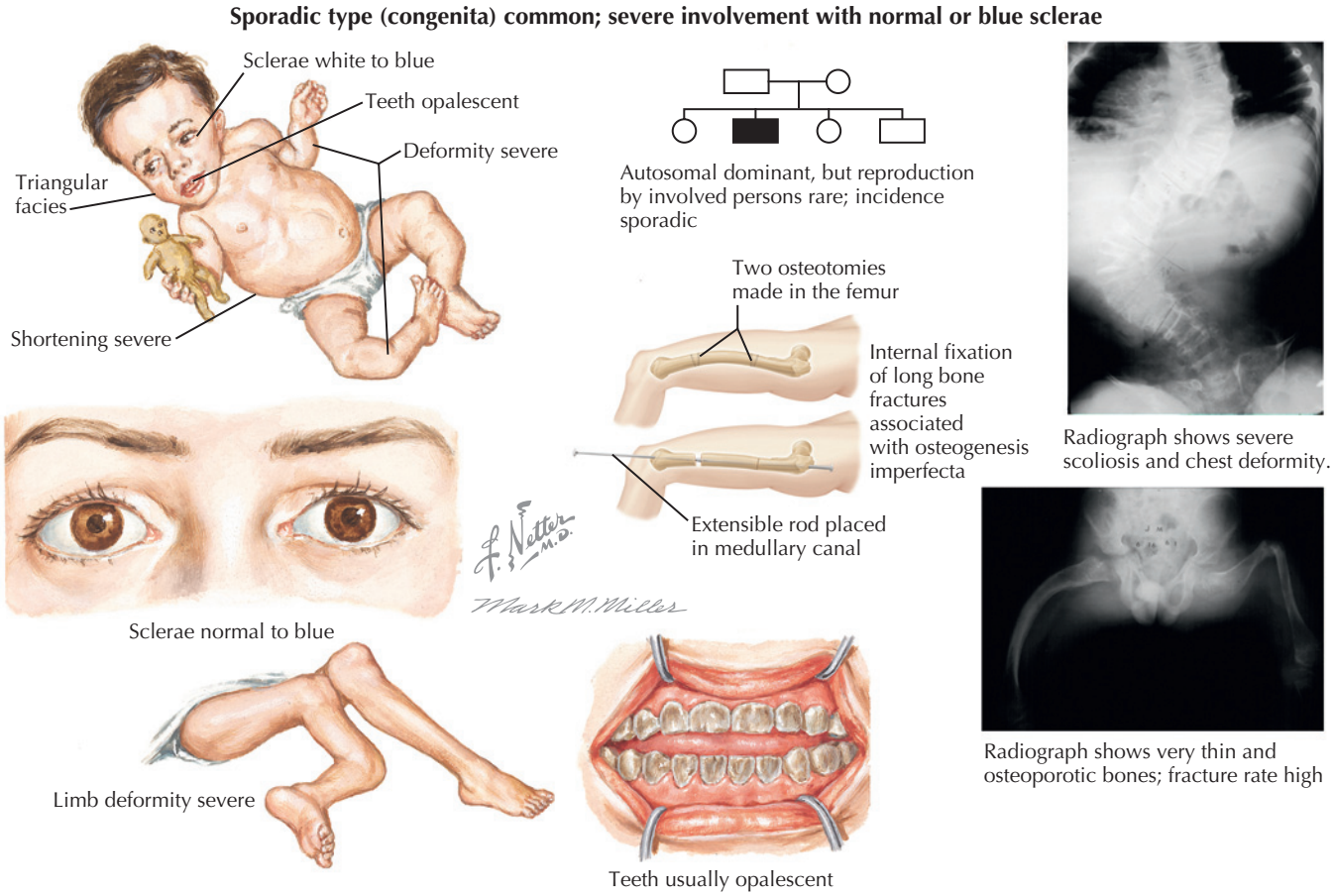


Figure 35-3 Characteristics of Osteogenesis Imperfecta.

ity of condition. Joint and bone protection is crucial both in activities of daily life and in selection of athletic and recreational activities. No cardiovascular limitation noted by the 36th Bethesda Guidelines. Allowed once fracture(s) have healed and matured both clinically and radiographically.

SERO-NEGATIVE ARTHROPATHIES

Ankylosing Spondylitis (AS)

Overview: A chronic, inflammatory rheumatic disease that causes arthritis of the spine and sacroiliac joint (Fig. 35-4). The chronic inflammation and irritation of the spinal joints (vertebrae) can eventually cause the vertebrae to fuse together, a condition known as "ankylosis."

Incidence: Approximately 350,000 Americans are estimated to have AS. About 1.8% of the U.S. population will develop AS.

Presentation:
- Back pain, acute or chronic
- Usually presents in 3rd to 4th decade
- Pain with back forward flexion
- Affects young men more than women (60% to 85% of cases are in men)
- Sacroiliitis
- History of low back pain/stiffness
- Occasional pain/inflammation in other joints (e.g., hips, knees, and ankles)
- Morning stiffness

Physical exam: See Figure 35-4.
- Pain with back flexion (lumbar/sacral area).
- Decreased range of motion in spine (cervical to sacral).
- Lateral spinal flexion exam: Patient stands erect with heels and back against the wall. The first measurement is distance from finger tips to floor. Then the patient is asked to laterally flex the spine with rotation without bending knees. Second measurement is the distance between finger tips and floor. Normal is greater than 10 cm.
- Schober test: Measure forward flexion of lumber spine. Patient is erect and a mark is placed 5 cm below the posterior superior iliac spine (PSIS), at midline at level of PSIS, and 10 cm above the PSIS over spinous process. The patient is asked to bend forward without bending knees. If the measurement between the two marks does not exceed 20 cm in length (start is 15 cm), the test is considered positive.
- Limited ability to fully expand chest with deep inhalation.
- Eye exam may show uveitis or iritis.
- Limited internal and external rotation of the hips.
- Enthesitis.
- Sacroiliac tenderness and positive FABER test.

Diagnostic:
- Blood work: HLA-B27 is positive in 90% of AS cases.
- X-ray shows squaring of vertebral bodies, "bamboo spine," fusion of vertebrae.
- Radiographic grading: modified Stokes Ankylosing Spine Score (SASS)
- MRI shows spondylitis (inflammation)/sacroiliitis.
- Indices for evaluating disease:
  - Bath Ankylosing Disease Activity Index (BASDAI)
  - Assessment in Ankylosing Spondylitis (ASAS)
  - Dougados Functional Index (DFI)
  - Health Assessment Questionnaire for AS (HAQ-S)

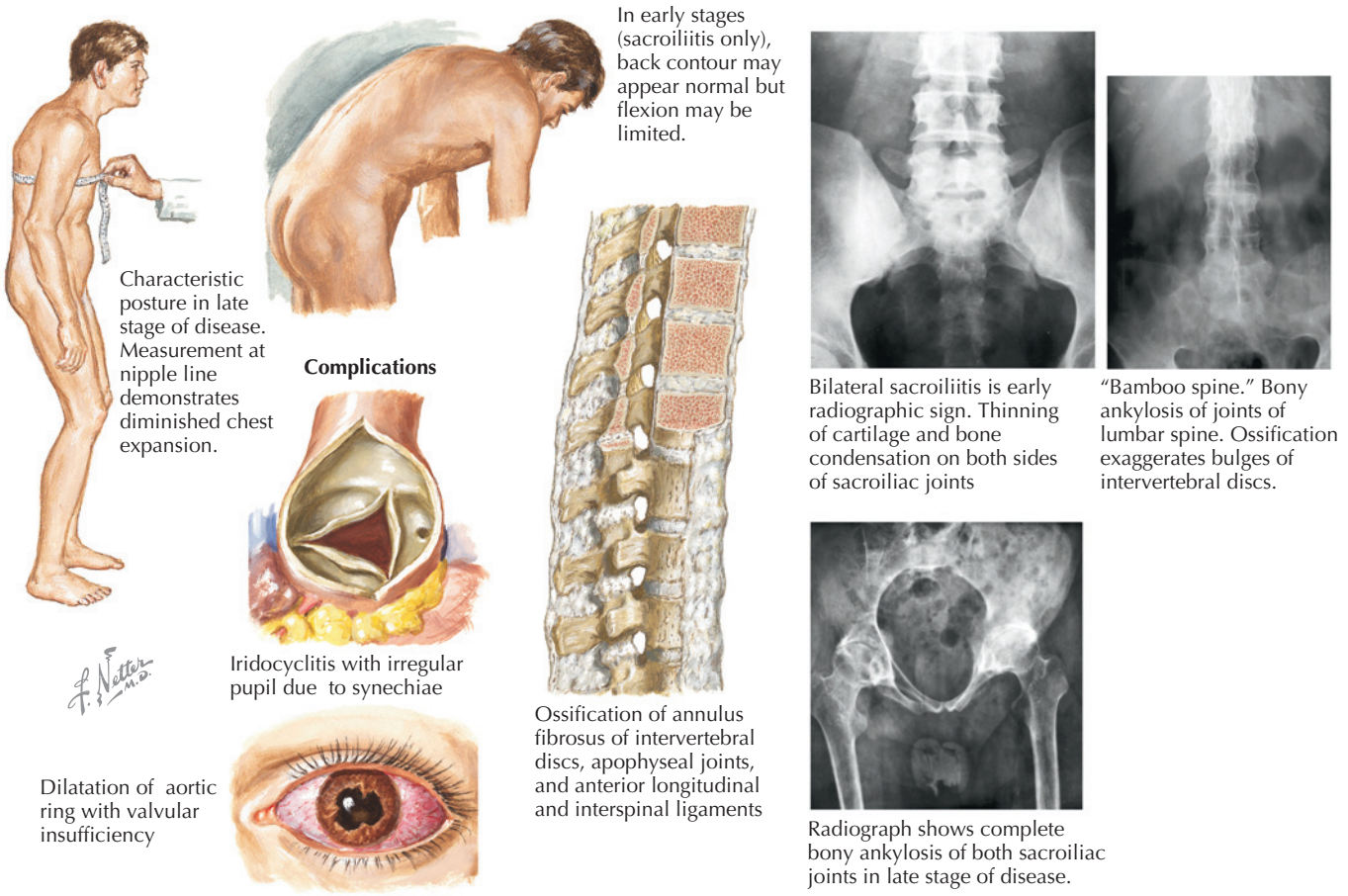


Figure 35-4 Characteristics of Ankylosing Spondylitis.

Treatment:
- Discontinue smoking because it can accelerate lung scarring and decrease pulmonary functioning.
- TNF-blockers:
  - Adalimumab 40 mg SC every other week
  - Etanercept 25 mg SC twice weekly
  - Infliximab 5 mg/kg IV every 6 weeks
- Drug-modifying antirheumatologic drug (DMARDs): methotrexate, sulfasalazine.
- Oral prednisone: for as short a time as possible, often chronic use needed.
- Injection (prednisone): epidural versus facet.
- Pain control: aspirin, acetaminophen, NSAIDs, tramadol.
- Physical therapy: Firm mattress; back extension exercises; stress core strength. Maintain spine mobility and lung capacity; improve posture.

Prognosis: Variable. Most patients with limited disease do well, although a minority of patients develop severe skeletal symptoms or life-threatening complications. Approximately 1% will "burn-out" or go into remission.

Return to play: Continuous flexibility training and core strengthening, mild to moderate exercise within limits of disease activity. Swimming is preferred because it avoids jarring impact of the spine. Patients can participate in carefully chosen aerobic sports when disease is inactive. Aerobic exercise is generally encouraged because it promotes full expansion of the breathing muscles and opens the airways.

Osteoarthritis or Degenerative Arthropathy

Also see Chapter 11, The Senior Athlete.

Overview: Most common joint disorder; about 5% of the population between ages 35 and 54 show signs of osteoarthritis in knee joint on radiograph. Directly related to severity of occurrences of trauma, joint-related surgery, age, and activity in which normal articular cartilage is damaged (Fig. 35-5). Currently, it is thought that metalloprotease enzymes (e.g., collagenase, stromelysin, gelatinase) are active in the degradation of cartilage. Other mediators that seem to play a role in osteoarthritis are cytokines, other proteases, nitric oxide, calcium crystals, sex hormones, aging, and familial genetics.

Presentation: Early morning stiffness and joint pain that generally improves with activity, asymmetrical joint involvement, often monoarticular, especially at knees (see Fig. 35-5).

Physical exam:
- Heberden's nodules: joint enlargement of the distal interphalangeal joint (DIP).
- Bouchard's nodules: tendon nodules located around the proximal interphalangeal joint (PIP) (see Fig. 35-5).
- Stiff, enlarged joint, often monoarticular or asymmetric.
- Decreased range of motion.
- Antalgic movement of that joint (seen often in weight-bearing joints).

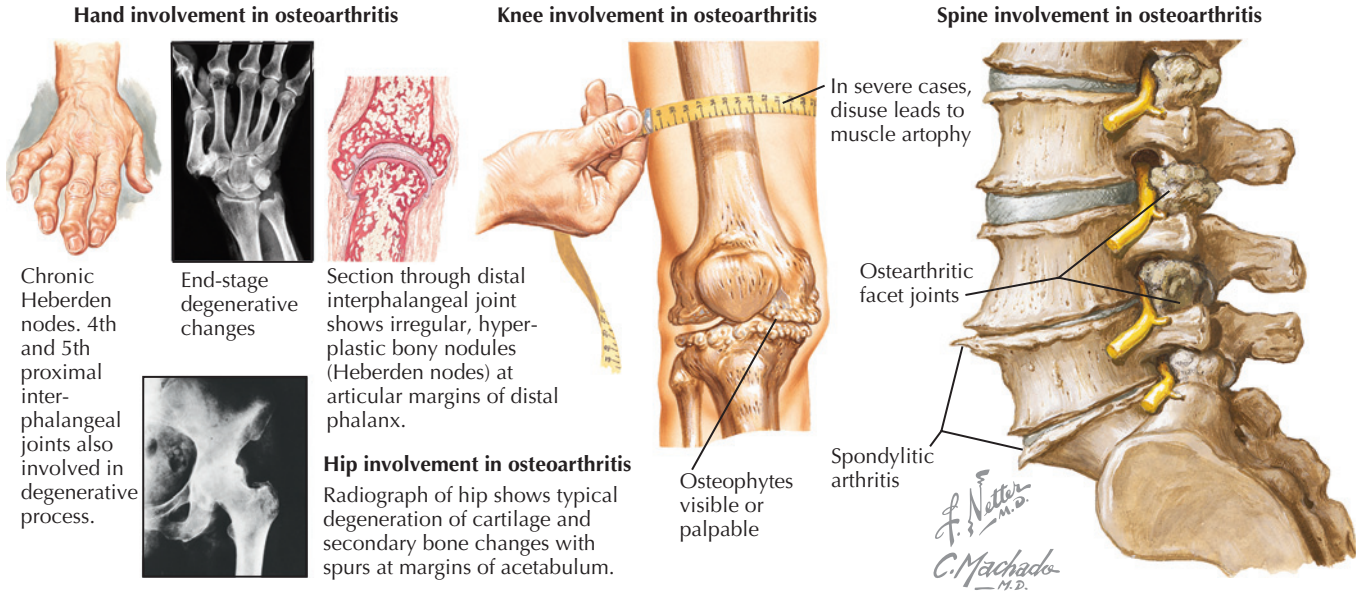


Figure 35-5 Characteristics of Osteoarthritis or Degenerative Arthropathy.

Diagnostic: History and physical exam. Radiographs (weight-bearing preferred in lower extremity). Bone scan will show moderate to severe arthritic changes in all joints of the body.

Treatment:

- Pain control (acetaminophen, tramadol, NSAIDs).
- Physical therapy should address range of motion, core strength, mobility, modalities, mild strength training to stabilize joints, home exercise program (HEP).
- Intra-articular injections: steroids and hyaluronic acid.
- Weight loss if patient is overweight.
- Flexibility training.
- Improve joint mechanics and shearing forces across joint.
- Daily stretching and exercise program.
- Joint replacement surgery, when conservative measures fail.

Prognosis: Varies among patients. It depends on severity of condition, number of joints involved, concomitant diseases, and activities (occupation/recreation).

Return to play: As tolerated by pain and mechanical defect. Long-term impact loading of affected joint(s) may advance disease at a quicker pace.

SERO-POSITIVE ARTHROPATHIES

Rheumatoid Arthritis (RA)

Overview: The most common chronic inflammatory arthritis, RA is thought to be caused by a complex interaction between T-cell and B-lymphocytes, macrophages, and fibroblast-like synoviocytes. Bimodal expressivity of this condition as juvenile onset versus adult onset.

Incidence: Approximately 1% of the population in Great Britain and U.S. is affected by rheumatoid arthritis.

Presentation: Joint pain in multiple joints (usually symmetric), non–trauma related, swollen joints, finger deformities or weakness (often episodic in nature), myositis/vasculitis, drug-induced myopathy (glucocorticoids, statins, antimalarials) (Fig. 35-6).

Physical exam:

- Metacarpophalangeal joint inflammation, especially first.
- Heberden's nodules: joint enlargement of the distal interphalangeal joint (DIP) (more common in OA).
- Bouchard's nodules: tendon nodules located around the proximal interphalangeal joint (PIP) (see Fig. 35-6).
- Tender proximal interphalangeal joints of the hand.
- Fingernail: subungual hemorrhage or infarct (see Fig. 35-6).
- Synovitis of multiple joints.
- Ulnar deviation of phalanges *(in long-term cases)*.
- Symmetrical joint pain and swelling; destruction of joint, synovium, and surrounding soft tissue.
- Swan neck deformity.
- Ulnar deviation.
- Eye involvement (see Fig. 35-6).
- Keratoconjunctivis sicca, episcleritis.
- Lung involvement: pulmonary toxicity secondary to methotrexate or gold salts
- Cardiac involvement: pericarditis, myocarditis.
- Foot deformities (see Fig. 35-6).
- Spine involvement.

Diagnostic: History and physical exam, radiographic changes (joint space narrowing or destruction). Serum laboratories: rheumatoid factor, sedimentation rate, anti-CCP ab, ANA, CBC: anemia (normocytic hypochromic). Neutropenia: consider Felty's syndrome.

Treatment:

- DMARDs
  - Methotrexate 7.5 to 25 mg IV or IM weekly
  - Hydroxychloroquine 200 mg PO twice daily with food
  - Azathioprine 1 mg/kg daily PO divided doses, once or twice a day
  - Penicillamine 250 to 500 mg two to three times a day
  - Cyclosporine 2.5 mg/kg PO divided doses, two times a day
  - Cyclophosphamide 1.5 to 3 mg/kg PO daily
  - Sulfasalazine 2 g daily PO divided doses, twice a day
  - Gold salts
- Prednisone orally burst then taper
- Monoclonal antibody drugs
  - Infliximab 5 mg/kg IV on weeks 0, 2, and 6, then every 6 weeks
  - Adalimumab 40 mg SC every other week
  - Etanercept 25 mg SC two times a week
  - Abatacept 500 mg IV every month
- Intra-articular injections: steroids and hyaluronic acid
- Interleukin-1 inhibitors (e.g., Anakinra SC daily)

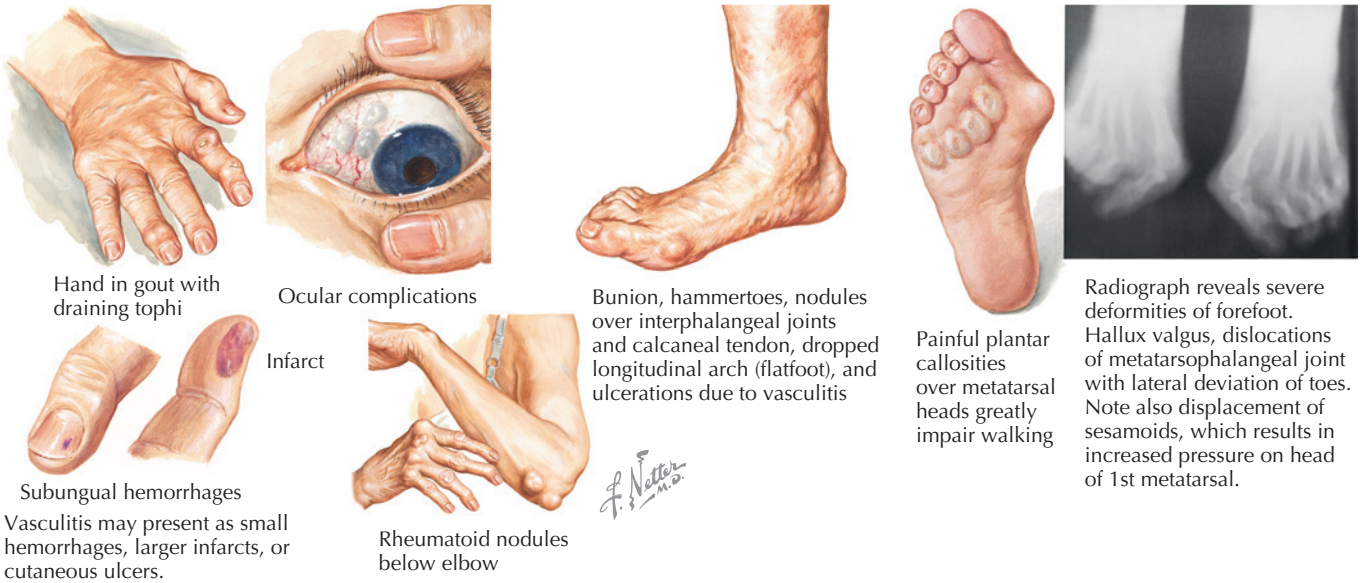


Figure 35-6 Characteristics of Rheumatoid Arthritis.

- Physical therapy
- Physical activity and healthy lifestyle
- Orthotics for foot deformities/pain
- Joint replacement surgery, when conservative measures fail

Prognosis: Depends on severity, responsiveness to medicines, juvenile versus adult onset. Many times juvenile RA may "burn-out" and become quiescent in early adulthood.

Return to play: Avoid combative/collision/contact sports, especially during the early phases of the condition as the DMARDs are starting to work.

- Disease quiescent with complete functional recovery: moderate to vigorous activity (e.g., running).
- Disease quiescent with minimal to moderate crippling: mild to moderate activity (e.g., swimming, cycling).
- Asymptomatic with effusion and synovial changes in weight-bearing joints in active therapy: mild activity with participation at own tolerance.
- Some evidence-based studies have shown that moderate athletic activity (e.g., swimming, cycling) had no negative impacts on deterioration of hand function and preserved hand function compared to nonactive controls.

Systemic Lupus Erythematosus (SLE)

Overview: Lupus is a chronic inflammatory immunologic disease that can affect multiple organ systems, including dermatologic, pulmonary, cardiac, renal, immune, musculoskeletal, and nervous systems, with the production of antinuclear antibodies (ANA).

Presentation: SLE presents more often in young women than men. The organ systems that are involved early in the disease are the same organs systems that continue to be involved later in the disease process. Most patients present with fatigue, fever, myalgias, nonspecific abdominal pain and/or weight loss, and migratory, asymmetrical arthralgias; the small joints of hands, wrists, and knees are the most frequently involved.

Physical exam: See Figure 35-7.

- Fever, weight change (loss or gain)
- Mucocutaneous: photosensitivity, malar/"butterfly" rash, serositis, discoid lesion often scar, alopecia, painless nasal/oral ulcers
- Hematologic: anemia, arthralgias
- Renal: glomerulonephritis, proteinuria more than 3.5 g in 24 hours, cellular casts, renal failure

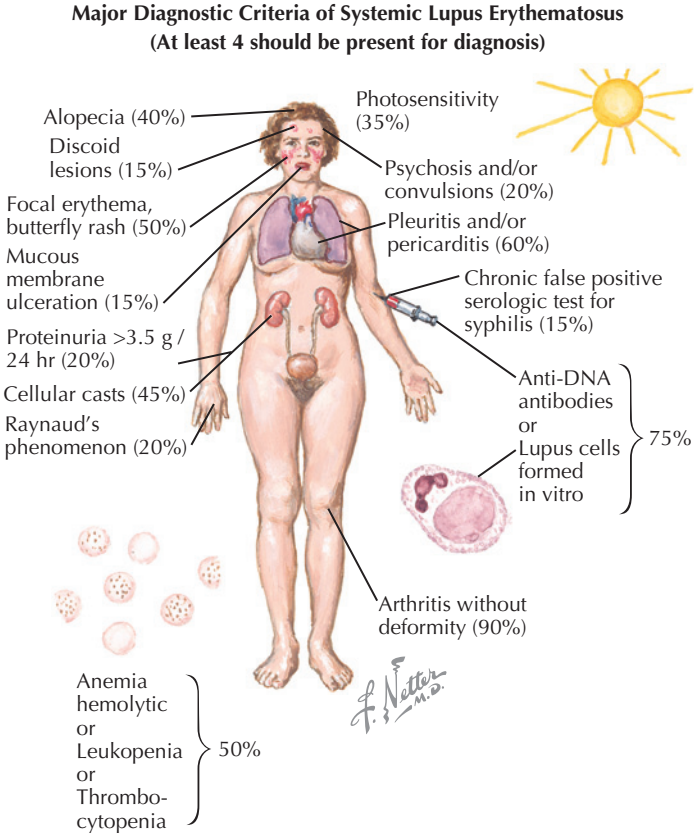


Figure 35-7 Characteristics of Systemic Lupus Erythematosus.

- Gastrointestinal/vascular: vasculitis with SLE often can lead to pancreatitis, peritonitis, and colitis; Raynaud's phenomenon
- Pulmonary: pleurisy, pleural effusions, pneumonitis, pulmonary hypertension, alveolar hemorrhage
- Cardiovascular: pericarditis, valvular insufficiency
- Neurologic/psychologic: psychosis, depression, anxiety
- Ophthalmologic: keratoconjuctivitis sicca, episcleritis or scleritis, retinal vasculitis, anterior uveitis

Diagnostic:
- Common hematologic abnormalities:
  - Leukopenia (white blood count below 4500 cells 10^3/mL in 43% to 66% of cases)
  - Mild anemia
  - Thrombocytopenia
- Elevated sedimentation rate.
- ANA greater than 1:160 *(large number of false positives)*.
- Confirmatory antibody blood tests include anti-double stranded DNA, anti-Smith, anti-SSB (La), anti-SSA (Ro); antiphospholipid antibodies may be present in those with SLE, associated with higher risk of thrombotic event.
- Libman-Sacks endocarditis (also known as verrucous endocarditis)—may cause valvular insufficiency.
- Premature degenerative joint disease may be noted on radiographs.

Treatment:
- Fever usually responds to NSAIDs, acetaminophen, or low-dose corticosteroids.
- Limit exposure to sunlight.
- Difficult to differentiate between true SLE and drug-induced SLE. Review all recent medications/supplements.
- Infections may initiate or cause a relapse of SLE.
- Hydroxychloroquine 400 mg PO once or twice a day with food.
- Other medications may include cyclophosphamide, methotrexate, azathioprine, immune globulins, mycophenolate, and prednisone.

Prognosis: Variable, can be episodic with long periods of remission to rather progressive and debilitating; occasionally leads to significant morbidity and some mortality.

Return to play: Protect from sun exposure because this can worsen or prolong dermatologic manifestations such as rash. Assess patient for signs of renal involvement by looking for proteinuria, calculating glomerular filtration rate via plasma creatinine levels, and, rarely, by doing a renal biopsy on a scheduled basis if patient suffers a flare. Discourage patient from participating during a flare that includes fever because of increased risk of endocarditis, vasculitis, and worsening synovitis. Baseline echocardiogram and subsequent follow-up echocardiograms prior to clearing for endurance or competitive sports with moderate to severe intensity to look for any valvular insufficiency. Patients with long history of SLE should be screened before starting a strenuous exercise regimen because they are at higher risk of having coronary artery disease. Stress, surgery, and pregnancy have been known to trigger relapses, so caution is warranted with these events.

OTHER RHEUMATOLOGIC CONDITIONS

Buerger's Syndrome (Thromboangiitis Obliterans)

Overview: Nontraumatic nonatherosclerotic inflammatory occlusion of small-to-medium sized arteries or vasculitis. It often presents with pain and claudication of lower extremities and is associated with heavy smokers and men younger than 50 years; also seen in users of smokeless tobacco.

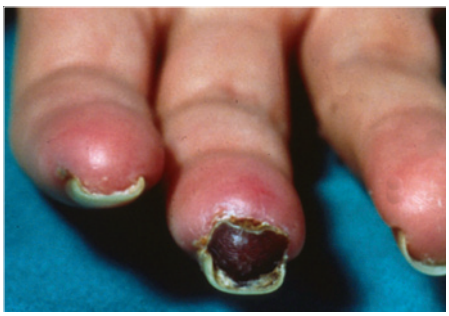
Etiology: Unknown, but possibly immunologic phenomenon with vasodysfunction and inflammatory thrombi.

Incidence: The incidence of Buerger's is approximately 12.6 per 100,000 persons in the United States. Incidence ranges from 1% to 5% of the population in western Europe to upward of 80% in Israel among Ashkenazi Jews with peripheral arterial disease.

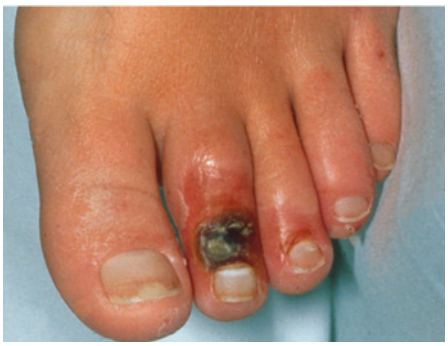
Presentation:
- Male-to-female ratio: 3:1
- Coldness, numbness, cyanosis of hands and feet
- Cold weather exacerbation
- Multiple amputations and/or ischemic ulcers
- Arch pain (most common site of foot claudication)
- Pain at rest (at advanced stages)
- Three phases
  - Acute phase: inflammatory
  - Intermediate phase: progressive organization of thrombus
  - Chronic phase: thrombus and fibrosis, no inflammation

Physical Exam: See Figure 35-8.
- Mottled skin appearance
- Ulcers or gangrene on distal extremities
- Delayed capillary refill in extremities (>2 seconds)
- Abnormal Allen test
- Tests for thoracic outlet syndrome (Wright's, Addson's, Roos) may be positive
- Decreased or absent distal pulses
- 20% to 50% will also have migratory thrombophlebitis
- To differentiate atherosclerosis from Buerger's
  - Buerger's usually affects both upper and lower extremities
  - Buerger's does not affect proximal arteries
  - Associated with tobacco use; improves when tobacco use stops
  - History of thrombophlebitis
  - Age younger than 45 years

Diagnostic:
- Serum labs (normal): ESR, CRP, ANA, complement, RF, Anticentromere Ab, Scl-70 Ab, Antiphospholipid Ab.
- Histopathologic examination of vessel identifies acute-phase lesion in a patient with an active tobacco history.



A Ischemic finger of a young male patient with Buerger's syndrome



B Ischemic toe of a 28-year-old woman with Buerger's syndrome

Figure 35-8 Characteristics of Buerger's Syndrome (Thromboangiitis Obliterans). *(Photograph reprinted with permission from Goldman L, Ausiello D, Arend W, et al: Cecil Textbook of Medicine, 23rd ed. Philadelphia: Elsevier, 2007.)*

- Arterial angiography
  - Early findings: consistent with claudication
  - Late findings: show multiple fine "cork-screw" shaped branches of distal arteries that end abruptly

Treatment:
- Eliminate contact with *all* tobacco products.
- Echocardiogram to screen for intracardiac thrombus.
- Common methods to assist patient quit smoking include hypnosis, nicotine replacement (via oral, inhaled, transdermal), bupropion (Zyban), varenicline (Chantix), etc.
- Symptoms should improve if patient is compliant (Cohn et al, 1990).
- Wear proper footwear and inspect feet daily.
- Avoid cold weather and vasoconstricting drugs.
- Cilostazol and pentoxifylline have not been shown to be effective.
- Iloprost: 6 hour IV daily decreased risk of amputation.
- Pneumatic compression stocking and sympathectomy have been used with minimal success.
- Surgery for revascularization is sometimes considered if patient is noncompliant with smoking cessation. Results are often less than optimal.
- Amputation if gangrene or osteomyelitis occurs.
- Clinical trials for phVEGF 165 (vascular endothelial growth factor) are underway and may show promise.

Prognosis: Good, if patient stops smoking.

Return to play: Once symptoms have begun to abate, then slow and progressive return to mild to moderate exercise is encouraged. Current review of the literature shows no evidence-based studies giving recommendations on either sports participation or return to play.

Polymyalgia Rheumatica (PMR)

Overview: A rheumatic condition that is associated with giant cell (temporal) arteritis (GCA), polymyalgia rheumatic (PMR) is characterized by chronic aching and morning stiffness in shoulders, hip girdles, neck, and torso in older persons.

Presentation: Prevalence is approximately 700 per 100,000 in persons older than 50 years. Of those with PMR, 50% also have GCA. In addition to the shoulder and hip stiffness and achiness, patient complains of malaise, fatigue, weight loss and anorexia, and mild fever.

Physical exam: See Figure 35-9.
- Decreased active range of motion at shoulder, neck, and hips.
- Normal muscular strength, though weakness/atrophy has been noted because of disuse.
- Often shoulder, neck, and hip musculature is not tender.
- Tenosynovitis around carpel tunnel has been noted.
- Soreness in affected joints seems to be related to a bursitis/synovitis.

Diagnostic:
- Criteria: Patient must:
  - Be older than 50 years.
  - Have bilateral, aching, morning stiffness in neck, shoulders, or hips for 30 minutes.
  - Have erythrocyte sedimentation rate (ESR) of 40 mm per hour or higher.
- *Differentiation from GCA:* PMR rarely expresses temporal tenderness, headache, jaw pain, visual loss, noncranial ischemia (e.g., arm claudication).
- Hematologic: ESR is elevated, often above 100 mm per hour but up to 20% may have an ESR as low as 40 mm per hour (Proven et al, 1999).
- Elevation of C-reactive protein (CRP).
- Immunologic: PMR and GCA share the polymorphism on HLA-DR4. A secondary hypervariable region of the HLA-DRB1 gene has been associated with PMR. IL-6 may be elevated.
- Imaging:
  - Radiographs: rarely reveal any changes of inflamed joints.
  - MRI: may show inflammation of tenosynovial sheaths or bursae.
  - Ultrasound: effusions of shoulder bursa
  - PET scan: may hold promise but more studies need to be done.

Treatment: Initial: moderate doses of corticosteroids (7.5 mg to 20 mg daily). **Chronic:** lower doses of corticosteroids are used (note: *ESR, CRP, and IL-6 levels may return to normal once corticosteroid treatment is started).* **Additional agents:** methotrexate, infliximab.

Prognosis: In most persons, PMR runs a self-limited course over months to years and corticosteroids can be discontinued. Ongoing surveillance for development of GCA symptoms should be assessed.

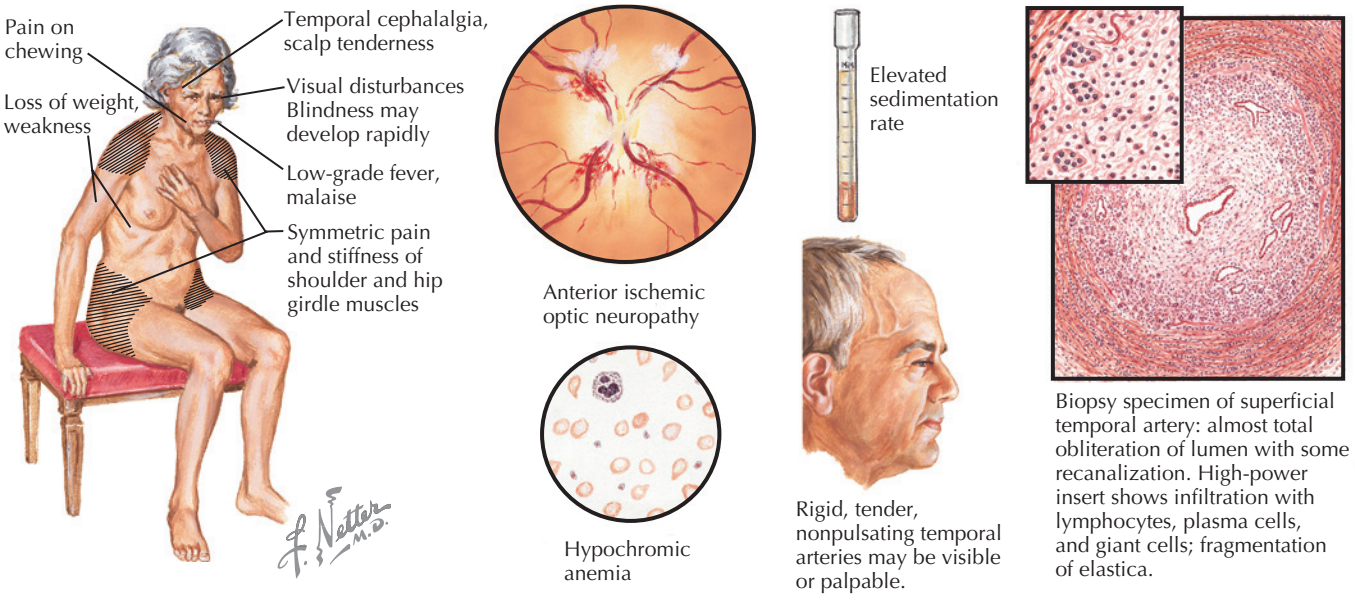


Figure 35-9 Characteristics of Polymyalgia Rheumatica and Giant Cell Arteritis.

Return to play: Once a patient has been indentified and appropriately treated, there are relatively little contraindications for participation outside of those issues associated with chronic steroid use (e.g., weight gain, glucose intolerance, osteoporosis, etc.).

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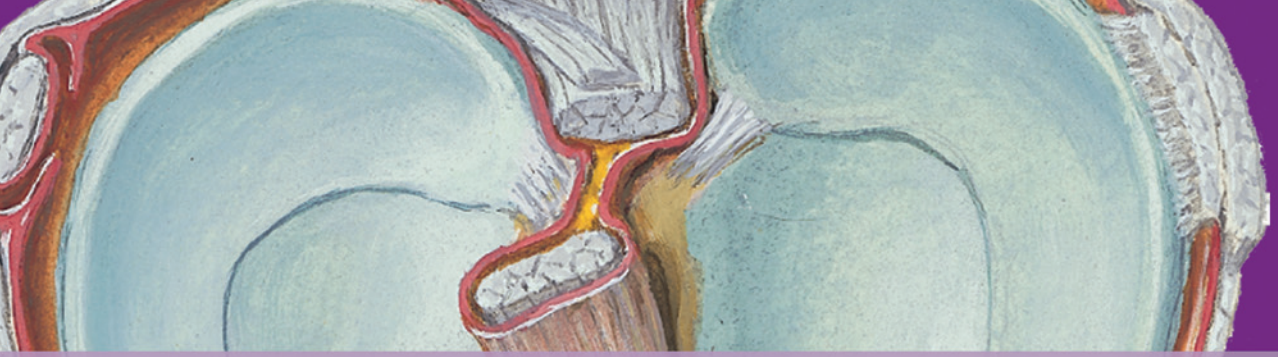

SECTION VII

Injury Prevention, Diagnosis, and Treatment

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Musculoskeletal Injuries in Sports

36

Eric C. McCarty, W. Michael Walsh, Ronnie D. Hald, Laura E. Peter, and Morris B. Mellion

GENERAL CLASSIFICATION OF MUSCULOSKELETAL SPORTS INJURIES

Musculoskeletal sports injuries can be classified as **traumatic** injuries or **overuse** injuries.

Traumatic Injuries

Description: Result from specific episode(s) of trauma, whether recent (acute) or in the more distant past (subacute or chronic).

Bone

Description: Traumatic injury to bone most commonly results in **fracture,** though rarely another process occurs, such as **subperiosteal hematoma.**

Descriptive terms:

- **Closed fracture** is a fracture that does not produce an open wound in the skin.
- **Open fracture** is when an open wound in the skin communicates with the fracture site.
- Descriptive terms for direction of fracture line:
  - Fracture at right angles to long axis of bone is called **transverse.**
  - Fracture line at other angle to long axis of bone is called **oblique.**
  - Bone twisted apart creates **spiral** configuration of fracture.
- **Comminuted fracture** is when a bone is broken into three or more pieces.
- **Avulsion fracture** is a "pull-off" fracture; a piece of bone is pulled off by ligament or tendon attachment.
- **Greenstick fracture** is an incomplete fracture in children. One side of a bone is broken, whereas the other side appears bent.
- **Torus fracture** is localized buckling in the cortex of the bone, common in children.
- **Epiphyseal fracture** is a fracture that involves the growth center at the end of a long bone in children.

Joint

Description: Traumatic injury to joint and supporting structures (capsule, ligaments) often results in an instability episode referred to as **dislocation** or **subluxation.** Rarely, some other process occurs from a direct blow, such as joint contusion or hemarthrosis.

Classification:

- **Dislocation** is complete displacement of joint surfaces so that they no longer make normal contact at all. Important to distinguish **first time** or **recurrent** dislocation.
- **Subluxation** is partial displacement of joint surfaces, usually transient in nature. Important to distinguish **first time** or **recurrent** subluxation.
- Dislocation or subluxation implies damage to ligaments or other supporting structures of joint. Important to ascertain injury to those tissues; discussed in following section.

Ligament

Description: Traumatic injury to ligament is referred to as **sprain.**

Classification:

- **First-degree sprain:** Tear of only a few ligament fibers. Mild swelling, pain, disability. No instability of joint created.
- **Second-degree sprain:** Tear of a moderate number of ligament fibers, but ligament function is still intact; however, ligaments may be somewhat stretched. Moderate amount of swelling, pain, disability. Slight to no instability of joint.
- **Third-degree sprain:** Complete rupture of ligament. Severe swelling and disability. Definite joint instability. Instability may be classified as:
  - **1**+ Joint surfaces normally stabilized by ligament(s) displaced 3 to 5 mm from their normal position.
  - **2**+ Joint surfaces separated by 6 to 10 mm.
  - **3**+ Joint surfaces separated by more than 10 mm.

Muscle-Tendon Unit

STRAIN

Description: Traumatic injury to muscle or tendon caused by **indirect force** (i.e., contraction of muscle itself) is referred to as **strain.**

Classification:

- **First-degree strain:** Tear of only a few muscle or tendon fibers. Mild swelling, pain, disability. Can also be characterized by patient's ability to produce strong, but painful, muscle contraction.
- **Second-degree strain:** Disruption of moderate number of muscle or tendon fibers, but muscle-tendon unit still intact. Moderate amount of pain, swelling, disability. Characterized by patient's weak and painful attempts at muscle contraction.
- **Third-degree strain:** Complete rupture of muscle-tendon unit. May be at origin, muscular portion, musculotendinous junction, within tendon itself, or at tendon insertion. Characterized by extremely weak attempts at muscle contraction.

DEEP MUSCLE CONTUSION

Description: Traumatic injury to muscle caused by **direct force** may produce deep muscle contusion. Typically affects quadriceps or brachialis muscles involved in contact or collision sports. May lead to **myositis ossificans** and therefore permanent loss of function.

MYOSITIS OSSIFICANS

Description: Heterotopic bone formation caused by deep muscle contusion or strain, especially after marked hematoma formation.

Common sites: Quadriceps; biceps, triceps, brachialis; hip girdle; groin; lower leg.

Risk factors: Severe contusion; continuing to play after injury; massaging injured area; early application of heat; passive, forceful stretching; overly rapid rehabilitation; premature return to sport; reinjury of same site; individual propensity to heterotopic bone formation.

Calcification: Follows injury by 3 to 6 weeks.

- May continue to develop for 6 weeks or more.
- May remodel or reabsorb over 3 to 12 months, especially if close to musculotendinous junction.

Treatment:

- Treat strain or contusion with basic athletic first aid (see Chapter 37, Comprehensive Rehabilitation of the Athlete) represented by the mnemonic **PRICES: p**rotection, **r**est, **i**ce, **c**ompression, **e**levation, **s**upport. Followed by progressive symptom-guided rehabilitation.
- **Excision rarely necessary**
  - Warranted only in cases of persistent weakness or limited range of motion.
  - Only after calcification matures (6 to 12 months).
  - High rate of recurrence if excised too early.

EXERTIONAL RHABDOMYOLYSIS

Description: Breakdown of skeletal muscle cells with leakage of cellular contents, including myoglobin, creatine kinase (CK), and aldolase through damaged sarcolemma into serum as result of prolonged, heavy, or repetitious exercise.

Presentation: Muscle pain and tenderness, muscle swelling, muscle cramps, reddish brown urine.

Laboratory tests: Urinalysis (urine dipstick positive for hemoglobin; microscopic exam reveals few to no red blood cells); elevated CK (normal = < 200 U/L; subclinical to mild rhabdomyolysis = > 600 U/L; most clinical cases = > 10,000 U/L; severe cases: reports > 200,000 U/L); and elevated serum myoglobin (normal = 5 to 70 μg/L; elevated only up to 6 hours after injury if renal function is normal).

Etiology: Intense exercise causes local tissue hypoxia, resulting in elevation of adenosine triphosphate (ATP) and consequent failure of sodium-potassium pump with potassium efflux and calcium influx; anaerobic glycolysis with lactic acid overproduction and, in severe cases, metabolic acidosis; sarcolemma permeability; and, in severe cases, cell death.

Common predisposing features: Heat, humidity, dehydration, poor physical conditioning (also may occur in well-conditioned athlete with very intense or repetitive exercise), high altitude, recent viral infection, sickle cell trait, and hereditary defects in ATP synthesis.

Clinical course:

- Most cases are **subclinical** and never diagnosed or **minimal** (visible myoglobinuria without muscle pain and spontaneous healing).
- **Mild-to-moderate cases** respond well to aggressive hydration. Without nephrotoxic cofactors, dehydration, and acidosis, damage is usually not persistent. Lasting systemic complications are rare; muscle has remarkable capacity for repair.
- **Severe cases** are common in patients with dehydration or acidosis. They are characterized by **systemic complications:**
  - **Acute tubular necrosis and renal failure** caused by combination of renal hypoperfusion, acidosis, and myoglobin sludging in renal tubules.
  - **Acute compartment syndrome** (rhabdomyolysis causes muscle swelling, which increases intramuscular pressure, causing vicious cycle of damage; in athletes with chronic compartment syndrome, increased compartment pressure may reduce tissue perfusion and cause rhabdomyolysis).
  - Hyperkalemia
  - Hypocalcemia
  - Hyperphosphatemia
  - Diffuse intravascular coagulopathy (DIC)
  - Cardiac dysrhythmia

Treatment:

- **Aggressive early hydration** to maintain renal perfusion and clear myoglobin, thereby preventing acute tubular necrosis and renal failure.
  - May require 4 to 12 L of normal saline during first 24 hours.
  - Diuretic (furosemide) may be necessary to maintain kidney function.
- **Alkalinization of urine with bicarbonate**
  - Myoglobin is less nephrotoxic and more soluble in alkaline urine.
  - Uric acid is more soluble and less likely to crystallize in alkaline urine.
  - **Caveat:** Alkalinization with bicarbonate may increase precipitation of calcium in injured muscles, causing heterotopic bone formation. Safer approach may be oral acetazolamide, 250 mg three times daily, if plasma bicarbonate level is greater than 13 to 15 mEq/L.
- Correct hyperkalemia, hyperphosphatemia, and hypocalcemia. Avoid IV calcium, except to treat tetany, because of risk of heterotopic bone formation.
- **Fasciotomy,** if necessary for compartment syndrome.
- Treat DIC if it does not resolve spontaneously.
- Dialysis if necessary.

Other Soft Tissues

Description: Traumatic injury to bursa with bursal swelling is referred to as **traumatic bursitis,** it is usually caused by bleeding into bursa. Traumatic injuries to other soft tissues include various **contusions** and **hematomas.** Lacerations may involve musculoskeletal tissues.

Shearing injuries: Avulsions, abrasions, blisters.

Overuse Injuries

Description: Account for more than 50% of injuries seen in sports medicine practices.

General overuse concepts: If viewed as function of Newton's third law of motion, athletic injury can be described as resulting from equal and opposite reactions, which in turn result in macrotrauma or microtrauma.

- Macrotrauma: Equal and opposite forces exceed strength of specific anatomic structure, and therefore the structure fails (see "Traumatic Injuries").
- Microtrauma: Microscopic subliminal injury from repeated activity. Can be cumulative over time and result in inflammation; characterized by pain and dysfunction.

- **Predisposition:** Equally important are intrinsic or extrinsic factors that predispose the athlete to overuse injury.
  - **Intrinsic:** Malalignment of limbs, muscular imbalances, other anatomical factors.
  - **Extrinsic:** Training errors, faulty technique, incorrect surfaces and equipment, poor environmental conditions.
- **Degenerative processes:** May influence traumatic injuries as well, but more commonly have effect on overuse injuries. Normal degenerative processes occur in many musculoskeletal tissues with aging. May add to likelihood of certain injuries. Common examples are rotator cuff and Achilles tendon problems.
- **General classification:** Overuse injuries are classified according to four stages, depending on pain:
  - **Stage 1:** Pain after activity only.
  - **Stage 2:** Pain during activity, does not restrict performance.
  - **Stage 3:** Pain during activity, restricts performance.
  - **Stage 4:** Chronic, unremitting pain, even at rest.

Bones

Description: Overuse injury of bone may be stress fracture or apophysitis.

Classification:

- **Stress fracture:** Most often found in lower extremity, but can also be found in spine (see Chapters 47and 53 for discussion of spondylolysis and spondylolisthesis) and upper extremity when it is subjected to weight-bearing (e.g., gymnastics, weight training).
- **Apophysitis:** In skeletally immature athletes, traction injuries can affect apophysis. Appear to result from repeated stress at tendinous insertion into bony growth center, followed by reactive bone formation. Most common apophysitis is Osgood-Schlatter disease (see Chapter 51).

Joints

Description: Overuse joint injuries almost invariably result from mechanical factors. Although they may create a condition that could be called "arthritis," it may be more valid when treating athletes to think of it as **synovitis.** Synovitis may be generalized,

with swelling, warmth, pain, and, occasionally, redness. Some synovitis is more localized (e.g., synovial plica of knee, peripatellar synovitis in extensor mechanism malalignment of knee; see Chapter 49).

Ligament

Description: There are few examples of pure overuse injuries to ligaments. Theoretically, they may occur whenever ligament is subjected to repeated stress. Examples include:

- **Medial elbow injuries:** Part of this spectrum may include overuse injury to medial collateral ligament of elbow, resulting from repetitive throwing with valgus loading.
- **Breaststroker's knee:** Probably most common example of pure ligament injury through overuse. Typically involves medial collateral ligament of knee at femoral attachment, secondary to breaststroke kick.
- **Plantar fasciitis:** Technically a ligament connecting bone to bone, the plantar fascia is commonly involved in overuse syndromes of the foot (see Chapter 54).

Description: Overuse injury of muscle-tendon unit may be myositis, tendinitis, or tenosynovitis.

Classification:

- **Myositis** overuse injuries of muscle tissue are rather nondescript. Can involve practically any muscle in the body. More distinct syndromes occur with overuse syndromes at muscular origin and attachment to bone/periosteum. Examples include lateral epicondylitis of elbow, medial epicondylitis of elbow, chronic groin strain, and shin splints.
- **Tendinitis** is inflammatory reaction within the tendon tissue itself. Closely related to concept of normal aging and degenerative changes within tendons (tendinosis), which may predispose to microtrauma. Common examples are bicipital tendinitis, rotator cuff tendinitis, and Achilles tendinitis.
- **Tenosynovitis (peritendinitis, tenovaginitis)** is inflammatory change that involves tissue surrounding the tendon itself. Classic physical finding is crepitation or "dry leather creaking" sensation over involved tendon as tendon is moved through its sheath. Common locations include extensor tendons of forearm and tibialis anterior in lower leg.

Other Soft Tissue

Description: Most common overuse musculoskeletal injury involving other soft tissue is **bursitis.** Bursae lie between tissue planes and help to reduce frictional stress between those structures. Common sites for mechanical bursitis in athletes include subacromial bursa, greater trochanteric bursa of hip, and retrocalcaneal bursa just anterior to the Achilles tendon insertion.

GENERAL TREATMENT OF MUSCULOSKELETAL INJURIES

- Basic athletic first aid: Use PRICES mnemonic (discussed earlier).
- **Nonsteroidal anti-inflammatory drugs** (NSAIDs) are commonly used in treating musculoskeletal sports injuries. Many different types and brands exist. Their use is usually based on clinician's empiric results rather than on objective scientific studies. Choice should always be tempered by known side effects (e.g., renal damage). Best thought of as adjunctive treatment to other modes.
- **Physical modalities:** Cold, heat, ultrasound, iontophoresis, and electrical muscle stimulation are commonly used.
- **Therapeutic exercises:** Most important, but the most commonly underused means of treating musculoskeletal sports injuries. Important to correct not only deficits that may result from injury, but also those that predispose to injury.
- **Injection therapy:** Most commonly injected material is corticosteroid, with or without local anesthetic. **Studies have demonstrated direct harmful effect of steroid on articular cartilage and weakening effect on tendon.** (See Chapter 56.)
  - **Never inject corticosteroid into young athletes' joints, or into major joint (e.g., knee, shoulder)** in athlete of any age, without objective evidence of degenerative change, **or into major load-bearing tendons** (e.g., patellar tendon, Achilles tendon). **To do so may hasten rupture.**
  - **Acceptable** to inject corticosteroid into muscular trigger points, bursae, and small non–weight-bearing joints (e.g., acromioclavicular joint) and large joints (e.g., knee, shoulder); however, numerous injections in a normal large joint is not recommended. Also acceptable to inject corticosteroid into **muscular** attachments to bone, such as lateral epicondyle (**total number** of injections should be limited); ligament attachments to bone where subsequent rupture of ligament would not be disastrous (e.g., plantar fascia attachment to calcaneus); **tendon sheath,** but not tendon itself (e.g., for de Quervain's disease at wrist); and already degenerated joint in older athletes.
- **Braces, supports, and other devices.** Various products have been developed to aid in treatment of athletic injuries, ranging from simple compressive sleeves for various joints to expensive custom-made braces. They are discussed in chapters on anatomic parts and individual sports.
- Calcification excision: rare.

SELECTED MUSCULOSKELETAL EVALUATION TECHNIQUES

Flexibility Testing

Description: Flexibility is limited by length of muscle across joint. Lack of flexibility in two-joint muscles (muscles that cross two joints) is often indicated as cause of musculoskeletal problems. In testing for flexibility, consider whether restriction seen is due to muscular tightness or other sources, such as lack of joint range of motion or pain.

Heel cord flexibility: Athlete sits with knee extended and is asked to actively dorsiflex ankle. Measurement is made goniometrically (Fig. 36-1). Normal value is at least 10 degrees beyond plantigrade. This may also be done with knee flexed to assess tightness within soleus (normal value is at least 20 degrees beyond plantigrade).

Hamstrings flexibility: Athlete lies supine with hip maintained at 90-degree flexion and is asked to extend knee actively without repositioning hip (see Fig. 36-1). Measurement is made goniometrically. Normal value is less than 10 degrees short of full extension.

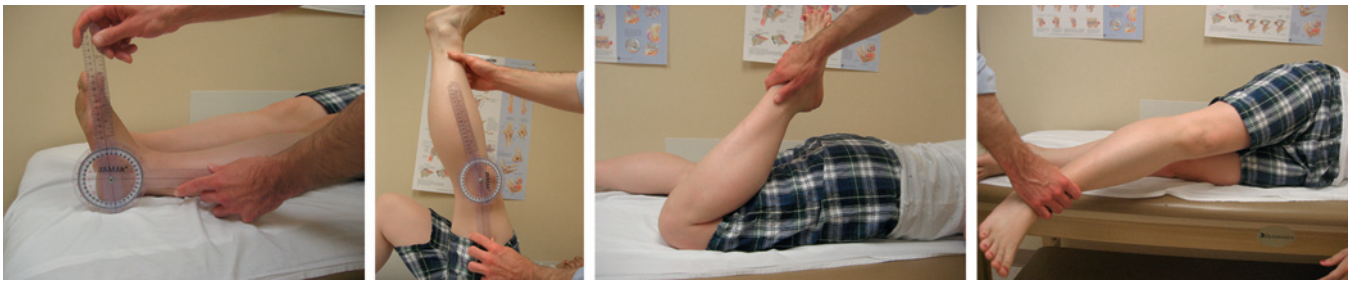
Quadriceps flexibility: Athlete lies prone and knee is flexed passively by examiner. Normal value is full knee flexion without tilting of pelvis (see Fig. 36-1).

Iliotibial band flexibility: Athlete lies on opposite side, near edge of examining table, facing away from examiner (see Fig. 36-1). Hip on side to be examined is slightly extended and passively adducted by gravity. Take care not to let iliotibial band slip anterior or posterior to greater trochanter or to allow lateral tilting of pelvis. Normal is considered to be when the knee drops level or below level of table. Also called modified Ober's test.

Strength Testing

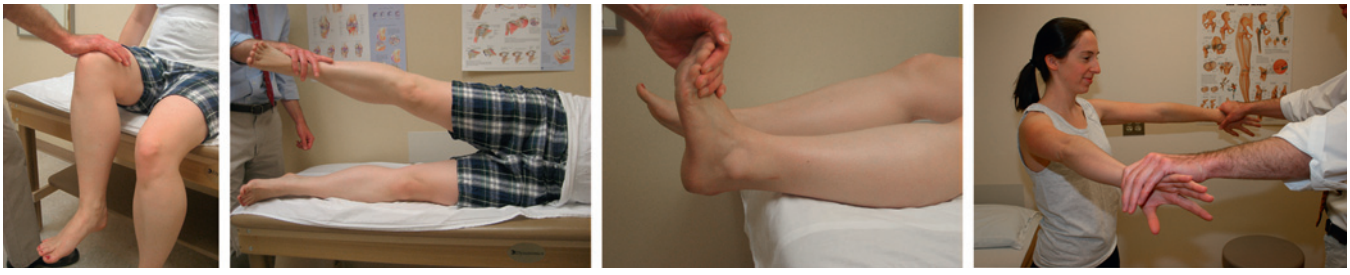
Description: Although there are many ways to assess strength, the authors prefer manual muscle "break" test technique. Athlete generates maximal contraction of muscle in shortened range, and examiner applies opposite force in attempt to move athlete from testing position. Common muscle testing rule is not to ap-

Flexibility testing



Heel cord flexibility | Hamstring flexibility | Quadriceps flexibility | Iliotibial band flexibility

Strength testing



Hip flexion strength | Hip abduction strength | Ankle dorsiflexion strength | Supraspinatus strength

Figure 36-1 Musculoskeletal Evaluation Techniques.

ply forces across adjacent joints, but athletes are generally able to support adjacent joints adequately, thus allowing examiner to apply more force to area in question. Strength is usually graded on a 0 to 5 scale (0 = zero, 1 = trace, 2 = poor, 3 = fair, 4 = good, 5 = normal). Most athletic applications are in upper range of this scale and subjective in nature. Although more objective methods are available, the manual muscle test is the most easily administered. Hip flexion, hip abduction, and supraspinatus strength tests are included, because weakness may indicate new condition or unrehabilitated condition more distal in kinetic chain. Ankle dorsiflexion strength test is included because of relationship to patellofemoral problems.

Hip flexion strength: Athlete sits at edge of table with arms crossed. Athlete flexes hip and examiner performs a manual muscle "break" test (see Fig. 36-1). If a break occurs, observe whether the weakness identified is located in the hip or in the abdominals.

Hip abduction strength: Athlete lies on opposite side, facing away from the examiner. Athlete abducts hip and examiner performs a manual muscle "break" test (see Fig. 36-1). If a break occurs, observe whether the weakness identified is located in the hip or in the abdominal obliques.

Ankle dorsiflexion strength: Athlete sits with knee extended and is asked to dorsiflex ankle. Examiner performs a manual muscle "break" test (see Fig. 36-1).

Supraspinatus strength: Athlete sits or stands with shoulders abducted to 90 degrees, horizontally adducted to 30 degrees, and in a "thumbs down" position (fully internally rotated). Examiner performs a manual muscle "break" test, taking care to eliminate substitution from the trapezius (see Fig. 36-1).

MYOFASCIAL PAIN SYNDROME

Definitions

- **Myofascial trigger point:** Intensely irritable spot in muscle and/or adjacent fascia that stimulates and sends distress signals to central nervous system.
  - Feels like indurated nodule or "ropey" taut band of muscle.
  - Occurs only in characteristic anatomic sites.
  - Each site has specific **"reference zones"** of radiating/referred pain or paresthesia. Reference zone pain is often presenting complaint.
  - **May trigger a spasm-pain-spasm cycle** (discussed in later section).
  - **Active trigger point**
    - Symptomatic reference zone pain.
    - Palpation reproduces both trigger point tenderness and reference zone pain.
  - **Latent trigger point**
    - Tender on examination.
    - No reference zone pain.
- **Myofascial pain syndrome** (myofascial syndrome): Presence of one or more active trigger points with characteristic reference zone pain.
- **Scapulocostal syndrome:** Clustering of trigger point spasms in the trapezius, levator scapula, and posterior cervical muscles.

Diagnosis

- **Knowledge of precise anatomic sites** of trigger points and reference zones. Common trigger point sites:
  - Levator scapula, splenius capitis, trapezius, sternocleidomastoid
  - Infraspinatus, supraspinatus, rhomboids
  - Quadratus lumborum, gluteus medius, tensor fascia lata
  - Biceps femoris, vastus lateralis, adductor longus
  - Gastrocnemius/soleus
- **Initiating, precipitating, and perpetuating phenomena**
  - **Physical:**
    - Trauma (major/minor, old/recent)
    - Overuse (sports/exercise, work with repetitive motion, muscle cramps)
    - Inadequate warm-up
    - Cold exercise/work environment

    - Poor posture, poor body mechanics, anatomic abnormalities, poorly designed or sized workstation (especially computer worksite)
    - Disease (rheumatoid arthritis, multiple sclerosis)
  - **Mental:** fatigue, anxiety/stress, depression
- **Palpation of trigger points:** "Rubbery" or "ropey"; indurated; tight; exquisitely tender.

Pain-Spasm-Pain Cycle

- Trigger point activation
  - First pain
  - Then local muscle activation and fatigue
  - After that, increase in pain spreads
  - Then additional trigger points recruited
  - This causes more pain, which spreads further
- "Key" or "matrix" trigger point recruits "satellite" trigger points.

Treatment

- **Stretch and spray:** Passive stretching using vapocoolant (fluoromethane) for distraction.
- **Massage:** Deep friction or pressure (acupressure); manual, elbow, dowel.
- **Trigger point injection**
  - 0.5% procaine
    - Other local anesthetics may be myotoxic.
    - Dilute 2% procaine with 3 parts of normal saline.
  - "Needling" by inserting 18-gauge needle without local anesthetic may inactivate trigger points.
  - Corticosteroids typically do not provide additional benefits because trigger points contain no inflammatory cells.
- **Therapeutic exercise** improves strength and flexibility.
- **Ice** (heat may exacerbate trigger points).
- Ultrasound.
- Muscle energy manipulation techniques.

Levator Scapula Syndrome

- Strain of levator scapula insertion with trigger point spasm of muscle body.
- Treat trigger point as discussed earlier; may also need to treat muscle insertion with corticosteroid injection, iontophoresis, or phonophoresis.

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Comprehensive Rehabilitation of the Athlete

37

Kevin E. Wilk and Charles D. Simpson II

INTRODUCTION

The overall goal of rehabilitation is to enhance recovery of injured tissues and avoid stresses that may prove deleterious to the healing process. This is accomplished by understanding normal function, pathomechanics, and the healing processes of specific tissues. Current research and scientific evidence must establish guidelines for rehabilitation. Rehabilitation specialists must integrate the medical team's diagnosis and conduct a functional examination of the musculoskeletal system.

PRINCIPLES OF REHABILITATION

Overview

- Communication with the sports medicine team along with an accurate and differential diagnosis is the beginning of a successful rehabilitation process.
- Communication between the rehabilitation specialist and physician should concern the type of injury, surgical procedure performed, method of surgical fixation, results of any diagnostic tests, integrity and quality of patient's tissue, and physician's patient-specific expectations so the proper rehabilitation program can be designed and implemented.
- Rehabilitation specialist must determine specific functional impairments and specific structures involved by thorough and systematic examination.
- Rehabilitation program must be designed based on the patient's unique response to injury and the athlete's specific functional needs.
- Program phases are designed to emphasize goals that are specific to the proper time frame of tissue healing at that particular point in rehabilitation:
  - Each phase will have specific goals that must be met, such as full range of motion (ROM), before progressing to the next phase.
  - Patients will reach milestones at different times, so criteria-based progression should be promoted over time-based progression.
  - Criteria-based progression assists with locating areas in which the patient may be improving slowly and thus may need to be more heavily emphasized.
- Establishing a differential diagnosis based on involved structures and causes contributing to the lesion is a fundamental part of designing the rehabilitation program.

Create a Healing Environment

- Clinician must promote healing while being careful not to overstress healing tissue.
- Program must be progressive and sequential with each phase building on the prior.
- Advancing a patient too quickly can result in inflammation, soreness, and potentially tissue failure, as opposed to controlled application of specific stresses, which can benefit healing tissues.

Decrease Pain and Effusion

- First goal in many rehabilitation programs is to decrease pain and effusion.
- Swelling stimulates sensory nerves and leads to an increase in the athlete's pain perception.
  - Pain and inflammation can work as muscle inhibitors, thus causing disuse atrophy the longer the effusion is present.
- Treatment options for swelling reduction include elevation, cryotherapy, high-voltage electrical stimulation, and joint compression.
- Patients with chronic joint effusion may benefit from using a knee sleeve or compression wrap to apply constant pressure while they perform everyday activities; such devices can minimize joint effusion.
- Patients with acute inflammation benefit from ice and elevation.
- Pain may play a role in muscle activity inhibition that is observed with joint effusion.
- Pain can be reduced passively through use of cryotherapy and analgesic medication.
- Commercial cold wraps immediately following surgery can be extremely beneficial.
- Passive ROM may also provide neuromodulation of pain during acute or exacerbated conditions.
- Therapeutic modalities such as ultrasound and electrical stimulation may be used to control pain via the gate control theory of pain.
- Speed of progression of rehabilitation, particularly weight-bearing and ROM, may affect pain and swelling; thus any increase in pain and effusion in the involved joint should be monitored as the patient progresses and adds additional exercises (Fig. 37-1).
- New exercises should be carefully monitored to ensure the pace of rehabilitation is appropriate and the tissue is not being overstressed; this is particularly important with articular cartilage procedures.
- Persistent pain, inflammation, and swelling may result in long-term complications involving ROM, voluntary quadriceps control, and a delaying of the rehabilitation process; thus it is imperative that these symptoms be minimized.

The Science of Rehabilitation

- When progressing a patient through rehabilitation, thought must be given to the healing tissue itself.
- Consider if the patient is ahead of schedule and has no complaints: can the patient continue at an accelerated rate without compromising the long-term health of his or her tissue?
- Another consideration: does someone returning at 4 months have better outcomes than someone returning at 6 months?
- Several characteristics must be considered when deciding the speed of rehabilitation:
  - The patient's age, genetics, nutrition, concomitant injuries, and unique healing characteristics can all affect the rehabilitation timeline.
  - Injuries to the meniscus and or collateral ligaments can slow the rehabilitation process, following anterior cruciate ligament (ACL) surgery for example.
  - Clinicians must also be aware of nonvisible concomitant injuries, such as bone bruises, that are associated with ACL injuries.
- Critical decisions have significant effects on metabolic activity of the injury site and the return to normal joint homeostasis.
- The risks and consequences of accelerated rehabilitation must be evaluated for each patient.

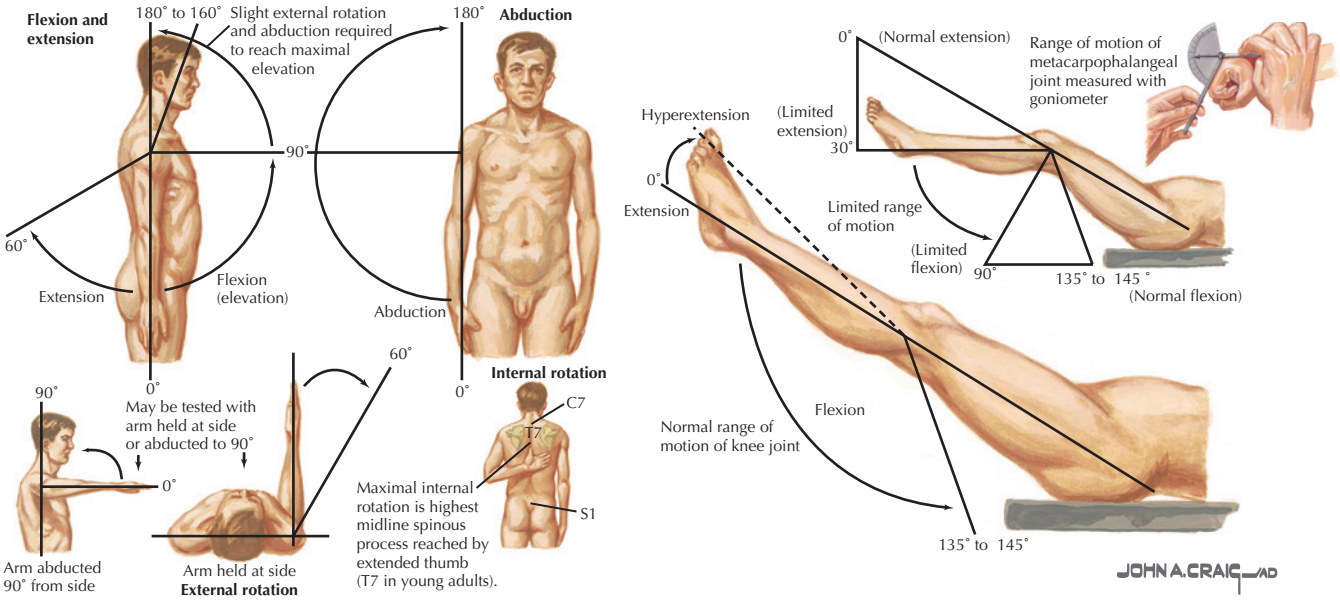

Figure 37-1 Range of Motion.

- The science of rehabilitation should be applied to all injuries and surgeries, especially rotator cuff repairs, SLAP (superior labral anterior posterior) repairs, meniscus repairs, and related procedures.

Prevent the Deleterious Effects of Immobilization

- Restriction of motion is often necessary in acute stages to promote tissue healing. ROM restriction can cause (see Fig. 37-1):
  - Quick loss of muscular girth and strength
  - Joint contracture
  - Loss of proteoglycan and weakening of articular cartilage
- "Motion is lotion for the joint."
- Deleterious effects of immobilization must be minimized and immobilization should be avoided in almost all cases.
- Current research indicates immediate controlled motion is critical to a successful outcome.
- Passive range of motion (PROM) is often performed by a skilled clinician but can also be applied in the form of continuous passive motion (CPM).
- PROM can also be applied by an isokinetic device set in passive ROM setting.
- CPM following surgery has several benefits, including avoidance of arthrofibrosis.

Retard Muscular Atrophy

- Rehabilitation must also focus on the retardation of muscular atrophy and facilitation of volitional muscle activity following injury or surgical procedure.
- Effusion can decrease voluntary control of surrounding musculature; this can affect the patient's ability to control his or limbs and ambulate with normal gait pattern.
- Exercises designed to enhance muscular volition begin with basic isometric contractions.
- Isometrics (Fig. 37-2):
  - Allow firing of the muscle fibers without joint motion.
  - Are a safe and effective method of exercise during early rehabilitation.
  - Are most often used at multiple static angles throughout the available ROM.

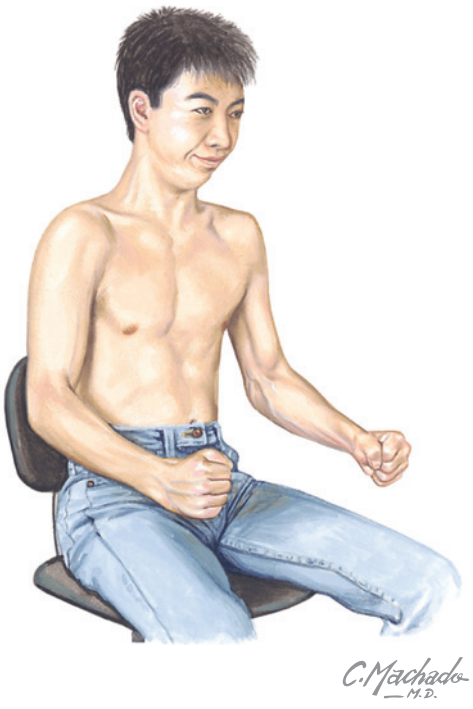

Figure 37-2 Isometric Exercise.

  - Have been shown to be one of the most efficient forms of exercise to increase muscular tension and improve strength.
- Muscle re-education with electrical muscle stimulation (EMS) may assist in restoring the patient's voluntary control of inhibited musculature.
  - EMS is often used concomitantly during isometric and isotonic exercises to increase recruitment of muscle fibers during contraction.
  - Several recent studies have found that patients that add neuromuscular electrical stimulation (NMES) to postoperative exercises have stronger quadriceps and more normal gait patterns than nonusers.

- Biofeedback can also be used to enhance voluntary control of injured musculature.
- Clinically, NMES is used following injury or surgery while the patient performs isometric and isotonic extremity exercises.
- NMES is typically used prior to biofeedback when the patient presents acutely with the inability to activate the musculature.
- Once independent muscle activation is present, NMES may still be used to recruit more motor units, thus resulting in greater strength gains.
- NMES is typically used 4 to 8 weeks post–ACL surgery or following selected shoulder surgeries.
- Biofeedback is used for patellofemoral patients when they are unable to actively recruit their vastus medialis; the biofeedback causes the patient to concentrate on neuromuscular control.

Restoration of Dynamic Stability

- Dynamic stability refers to the patient's ability to stabilize a joint during functional activities to avoid injury.
- Dynamic stability involves neuromuscular control and the efferent (motor) output to afferent (sensory) stimulation from the mechanoreceptors.
- Dynamic stability of the glenohumeral joint is primarily achieved through interaction of rotator cuff muscles as they blend into the joint capsule.
- Contraction of the rotator cuff produces tension within the joint capsule, which centers the humeral head on the glenoid.
- Muscle weakness or strength imbalances of the posterior cuff muscles may have deleterious effects on shoulder mechanics.
- Exercises to enhance dynamic stability are emphasized immediately following injury or surgery through the use of rhythmic stabilization drills.
- Alternating isometric contractions are performed to facilitate co-contractions of the anterior and posterior rotator cuff.
- Drills are progressed to include stabilization at end range of motion and with the patient's eyes closed, especially in the overhead-throwing athlete, in whom dynamic stability is compromised during the throwing motion.

Restoration of Proprioception and Neuromuscular Control

- Early proprioception and kinesthesia exercises are important for patients returning to sports because researchers have shown a decrease in these abilities following injury.
- Basic exercises designed to enhance the athlete's ability to detect the joint position and movement in space are performed to establish a baseline of motor learning for further neuromuscular control exercises that will be integrated at a later time.

Proprioceptive Training

- Proprioceptive training initially begins with basic exercises such as joint positioning and closed kinetic chain weight shifting.
- Joint repositioning drills begin with an athlete's eyes closed; the rehabilitation specialist passively moves the extremity in various planes of motion and then returns to the starting position.
  - The patient is then instructed to actively reposition the extremity to the location.
  - A therapist may increase the challenge to the patient's proprioceptive system by altering external stimulus such as vision and hearing.
- Weight shifting should be performed in the medial-lateral direction and in diagonal patterns.
- Mini-squats on a force platform to ensure equal weight distribution are beneficial (see Fig. 37-3).
- Advancement to mini-squats on an unstable surface such as a tilt board may be used as the patient progresses.

Mini-squats on a force platform that can provide objective feedback of the amount of weight distributed between lower extremities (Balance Trainer, Uni-Cam Inc., Ramsey, NJ).

Figure 37-3 Proprioceptive Training.

- Several studies have shown that the wearing of an elastic bandage may have a positive effect on proprioception and joint position sense.
- As proprioception is advanced, drills to encourage preparatory agonist-antagonist coactivation during functional activities are incorporated.

Dynamic Stabilization Drills

- Dynamic stabilization drills begin with single leg stance on flat ground and unstable surfaces, cone stepping, and lateral lunge drills.
- The patient may perform forward, backward, and lateral cone step over drills to facilitate gait training, enhance dynamic stability, and to train the hip to help control forces at the knee joint.
- Cone drills may be performed at various speeds to train the lower extremity to dynamically stabilize with different amounts of momentum.
- Lateral lunges are also performed, with the patient advancing from straight plane lateral lunges, to multiplanar/diagonal lunges, to lunges with rotation, and finally to lateral lunges onto foam (Fig. 37-4A).
- Concentration may be challenged by adding a ball toss to any of these exercises, which challenges preparatory stabilization.
- Single leg balance exercises are progressed by altering a patient's center of gravity and incorporating movement of the upper extremity and uninvolved lower extremity (Fig. 37-4B).
- Perturbation training may also be incorporated into such exercises with single or double leg balance exercises on a tilt board (Fig. 37-4C).
  - The patient performs an isometric hold of the tilt board with the knee flexed to 30 degrees while catching a light medicine ball.
  - The patient is instructed to stabilize the tilt board in reaction to the sudden outside force produced by the weighted ball.

A. Lateral lunges using a sport-cord onto an unstable surface.

B. Single leg balance on an unstable surface while incorporating alternating upper extremities movements with a weighted ball to alter the patient's center of gravity.

C. Single leg balance on a tilt-board while the patient tosses a ball against a rebound device. The rehabilitation specialist may create a perturbation by striking the board.

D. Rhythmic stabilization to promote co-contraction of the rotator cuff. The patient is asked to hold the arm steady while the therapist gives alternating forces to the extremity.

E. Upper extremity lyometrics. a. Chest pass using a trampoline and medicine ball. **b.** Single arm wall throws with the patient's arm at 90° of abduction and 90° of elbow flexion. **c.** Side to side throws using a medicine ball and trampoline.

F. Manual resistance during sidelying external resistance. The rehabilitation specialist resisted both external rotation and retraction of the scapula. Rhythmic stabilizations may also be performed at end range.

Figure 37-4 Dynamic Stabilization Drills.

- The rehabilitation specialist can also challenge the athlete by providing manual perturbations by striking the tilt board with his or her foot to create a sudden disturbance in the static support of the lower extremity; this requires the patient to stabilize the board with dynamic muscular contractions.
- Exercises such as balance beam walking, lunges onto an unstable surface, and step-up exercises while standing on an unstable surface are also used to strengthen the knee musculature while requiring the muscles located proximally and distally within the kinetic chain to stabilize and allow coordinated functional movement patterns.
- Plyometric jumping drills are performed to facilitate dynamic stabilization and neuromuscular control (Fig. 37-4D).
  - Plyometrics use the muscle's stretch-shortening properties to produce maximal contraction following a rapid eccentric loading of the muscle tissue.
  - Plyometric training is used to train the extremities to produce and dissipate forces to avoid injury.

Neuromuscular Control Drills

- The final aspect of rehabilitation regarding neuromuscular control involves enhancing muscular endurance.
  - Proprioceptive and neuromuscular control has been shown to diminish once muscular fatigue occurs.
  - Exercises such as bicycle, stair climbing, and elliptical machines are used to increase endurance.
  - High-repetition, low-weight resistance weight training can also increase muscular endurance.
- An additional strategy to protect the athlete from reinjury is to perform neuromuscular control drills at the end of treatment sessions after cardiovascular training.
  - This challenges the neuromuscular control of the knee joint after the dynamic stabilizers have been fatigued.
- Enhancement of neuromuscular control is equally important in the upper extremity.
  - Efficient dynamic stabilization and neuromuscular control of the glenohumeral joint is necessary for athletes to avoid injuries during competition.
  - Dynamic stabilization exercises for the upper extremity also begin with baseline proprioception and kinesthesia drills.
  - Rhythmic stabilization is also incorporated to facilitate co-contraction of the rotator cuff and dynamic stability of the glenohumeral joint (Fig. 37-4E).
  - Exercise involves alternating isometric contractions designed to promote co-contraction and basic reactive neuromuscular control (Fig. 37-4F).
- These dynamic stabilization techniques may be applied as the athlete progresses to provide advance challenges to the neuromuscular control system.
- As an athlete progresses through the program, it is necessary to train the upper extremity to provide adequate dynamic stabilization in response to sudden forces, particularly at end range of motion (referred to as *reactive neuromuscular control*).

Gradually Restore Muscular Strength and Endurance

- Gradually restore muscular strength after volitional muscle activity is achieved.
- Baseline levels of muscular strength are needed before the athlete can progress to the later stages of rehabilitation.
- Strengthening can be performed through a variety of different methods of isotonic exercises.
  - Weight is gradually applied and increased as the athlete progressively improves strength.
- Isotonics can be used in the form of single-joint or multijoint exercises.
- Exercises can also be performed in either an open kinetic chain (OKC) or closed kinetic chain (CKC).
  - OKC exercise is defined as a movement in which the distal extremity is not fixed, such as a leg extension.
  - CKC exercise is defined as an exercise in which the distal extremity is fixed, such as a leg press.
  - These exercises both have a place in rehabilitation, although they have different effects on both muscular activity and biomechanics of the joint, as shown by multiple electromyographic activity studies.
- Muscular endurance is an important factor in rehabilitation programs.
  - Many activities related to athletics involve repetitive and microtraumatic events.
  - Training the musculature to endure these events in necessary to prevent injuries.
  - Fatigue has been shown to result in decreased proprioception and altered biomechanics of the joints, which may result in further pathology.

Normalization of Soft Tissue Mobility and Flexibility

- Rehabilitation of soft tissue to restore tissue balance applies to both soft tissue around the joints, such as retinacular tissue surrounding the patella, and also muscular flexibility around each joint.
- Deviations in the balance of soft tissue forces will promote altered arthrokinematics and excessive forces to the joints.
- Muscular flexibility is vital to normal joint function because it allows the musculature to absorb force and align the joint in neutral position. For example:
  - Soft tissue tightness of the quadriceps muscle is common to those with patellar tendonitis and patellofemoral pain.
  - In the upper extremity, it is common to see patients with tightness of the anterior structures, which can lead to several pathologies such as impingement syndrome.
- It is critical for the clinician to identify the causes for loss of motion and treat involved structures based on assessment.

Emphasis on the Entire Kinetic Chain

- Rehabilitation must be focused on not only regaining strength and neuromuscular control of the affected joint but also on the surrounding areas. For example, neuromuscular control of the shoulder involves stability of not only the glenohumeral joint but also the scapulothoracic joint.
- Core stabilization drills are used to further enhance proximal stability with distal mobility of the extremities:
  - This is based on the kinetic chain concept, in which imbalance within any point of the kinetic chain may result in pathology throughout.
  - Movement patterns, such as throwing, require a precise interaction of the entire body kinetic chain to be performed efficiently.
  - A multiphase approach, progressing from baseline core and trunk strengthening, to intermediate core strengthening with distal mobility, to advanced stabilization in sport specific movement patterns, is used.
- Imbalances of strength, flexibility, endurance, or stability may result in fatigue, abnormal arthrokinematics, and subsequent compensation.
- It is important to not neglect the uninjured extremity:
  - Numerous studies have pointed to a crossover effect when the contralateral extremity is exercised, which may lead to improvements in proprioception and strength of the involved extremity.
  - The neuromuscular control system may have a certain amount of central mediating function receptive to bilateral training techniques.
  - When rehabilitating a patient with a joint injury, the rehabilitation specialist must consider having the patient perform either bilateral exercises or unilateral reciprocal exercises.

Gradual Return to Functional Activities

- Following successful completion of the rehabilitation program, the athlete must begin a gradual return-to-sport program.
- Interval sport programs (ISP) are designed to gradually return motion, function, and confidence to the athlete after injury or surgery by slowly progressing through graduated sport-specific activities.
  - Goal of this phase is to gradually and progressively increase functional demands on the athlete.
- Criteria before returning to sport-specific activities are:
  - Full functional ROM
  - Adequate static and dynamic stability
  - Satisfactory muscular strength and endurance
  - Satisfactory clinical examination
- Once criteria is met a gradual return-to-sport activity in a controlled manner is initiated.
- Healing constraints based on surgical technique and fixation, as well as the patient's tissue status, should be considered.
- ISP is set up to minimize chance of reinjury and emphasize precompetition warm-up and stretching.
- There should be no set time table for completing the ISP because of individual differences.
  - Variability will exist based on skill level, goals, and injury of each athlete.
- The ISP is developed based on the specific sport and stresses observed during these athletic activities.
  - For example, overhead-throwing athletes must perform an interval throwing program that includes a limited amount of throws using a flat-ground long toss.
- Other goals are to maintain a patient's muscular strength, dynamic stability, and functional motion established in the previous phase.
  - A stretching and strengthening program should be performed on an ongoing basis to maintain and improve on these goals.
- Rate of progression with functional activities is dictated by the patient's unique tolerance to the activities.
  - Exercise must be performed at a tolerable level without overstressing the healing tissue—referred to as the patient's *envelope of function.*
- The athlete's return to sport-specific drills progresses through a series of transitional drills designed to progressively challenge the neuromuscular control system. Examples include:
  - Pool running prior to flat-ground running.
  - Backward and lateral running prior to forward running.
  - Plyometrics prior to running-and-cutting drills and, finally, sport-specific drills.
- Integration of functional activities is necessary to train the injured patient to perform specific movement patterns necessary for everyday activities.
- The intention of sport-specific training is to stimulate the functional activities associated with sports while incorporating

peripheral afferent stimulation with reflexive and preprogrammed muscle control and coactivation.
  - Drills may be modified based on specific functional movement patterns that are unique to the patient's sport.
- Sport-specific training can include side-to-side shuffle, carioca, sudden starts and stops, 45-degree cutting, 90-degree cutting, and various combination movements.
- Sport-specific patterns learned throughout the rehabilitation program are integrated to provide challenges in a controlled setting.
- Drills are performed to train the neuromuscular control system to perform during competition in a reflexive pattern.

SPECIFIC REHABILITATION PROGRAMS

Anterior Cruciate Ligament (ACL)

- ACL rehabilitation programs have dramatically changed over the past 15 years.
- Currently, a scientifically based program that takes into account patient's functional demands is critical to maximize outcomes.
- Emphasis is now on immediate ROM, full passive knee extension, immediate weight bearing, and activation of the quadriceps musculature through NMES.
- The rehabilitation program begins preoperatively through education of the patient and family on both the surgical procedure and rehabilitation progression.
- Preoperative goals include reduction of pain and swelling, restoration of ROM, gait normalization, and prevention of muscular atrophy.
- Postoperative rehabilitation begins 1 to 2 days following surgery.
- Initial sessions include the initiation of ROM activities and to assure patient that weight bearing with the use of crutches is encouraged.
  - Primary goals at this point are to reduce swelling, restore full extension, and activate quads.
- Expectations are that the patient should be full weight bearing in a hinged brace without the use of crutches at 10 to 14 days after surgery.
- Full passive knee extension is attained as quickly as possible with a gradual restoration of flexion ROM and patella mobility.
- Open kinetic chain exercises with the use of NMES followed by incorporation of closed kinetic chain exercises can be introduced 2 weeks postoperatively.
- Machine weights may be introduced approximately 3 to 4 weeks postoperatively, including leg press and multi-hip (abduction, extension, and adduction).
- Closed kinetic chain exercises and neuromuscular control drills may be progressed as tolerated to include perturbation training on unstable surfaces.
- Light plyometric jumping may be initiated 8 to 10 weeks after surgery.
- Functional activities such as jogging may begin shortly after plyometrics with progression to running and jumping at 10 to 14 weeks.
- Careful attention should be made to avoid overstressing the athlete during this phase; the development of patella tendonitis is possible, especially if adequate quadriceps strength has not yet returned.
- Educating the female athlete on proper jumping and landing techniques should be included; this has been shown to reduce the incidence of future ACL injuries in the athletic population.
- Finally, the athlete can gradually return to cutting sports such as baseball, football, and tennis approximately 4 to 6 months postoperatively, but a return to jumping sports (basketball and volleyball) should be delayed until 6 to 8 months postoperatively.

Superior Labral Anterior to Posterior (SLAP) Repairs

- Pathology to superior labrum presents significant challenge to the rehabilitation specialist.
- Better understanding of the normal labral anatomy and healing constraints following a repair procedure will make a successful return to unrestricted function very likely.
- Successful return to the patient's prior level of function can be attained through communication by the surgeon with the rehabilitation team and patient; activity should be restricted until adequate healing response is obtained.
- Prior to initiating rehabilitation, a thorough subjective and clinical examination should be performed to identify the nature of the labral pathology and mechanism of injury.
  - This will aid the rehabilitation specialist postoperatively while advancing the patient through the protocol.
- There are certain restrictions to a patient's rehabilitation process that can be unique based on the method of injury. For example:
  - Patients who sustained labral injury after falling onto an outstretched hand would be encouraged to avoid closed-chain and weight-bearing activities.
  - Patients with traction injuries should avoid heavy biceps resistance activities, particularly those involving the eccentric phase of muscle contraction.
  - The overhead-throwing athlete with superior labral injuries should avoid excessive external rotation until adequate healing time of at least 8 weeks has occurred.
- Mechanism of injury is an important factor to consider as the patient progresses through the rehabilitation program.
- Rehabilitation following a SLAP type II repair presents a significant challenge but should render the patient with a good to excellent outcome.
  - This type of labral lesion is commonly seen in overhead-throwing athletes with the biceps tendon detached from its glenoid rim attachment.
- Initially the goal of rehabilitation is to control the forces placed on the healing tissue.
- Gradual passive and active-assisted range of motion activities are performed below 90 degrees of flexion for the first 4 weeks (see Fig. 37-1).
  - Passive external rotation is allowed only to approximately 15 degrees in the scapula plane.
  - The patient is instructed to sleep in an immobilizer for 4 weeks.
- ROM activities are progressed beyond 90 degrees of flexion with full flexion at 6 to 8 weeks postoperatively.
- External rotation ROM is progressed to 90 degrees of abduction at 4 weeks after surgery and motion is gradually increased until fully restored (115 to 125 degrees) by approximately 8 weeks postoperatively.
- Isometric strengthening activities are performed immediately to prevent muscle atrophy as a result of the immobilization.
- Rhythmic stabilization drills are also initiated with the patient in supine position and the shoulder in the scapula plane and neutral rotation.
- These activities promote dynamic stability and neuromuscular control of the humeral head within the glenoid while the scapula is stabilized by the table.
- Active joint repositioning exercises following passive displacement are also used to enhance proprioception.
- External rotation/internal rotation tubing exercises are initiated during week 3 to 4 with the arm at 0 degrees abduction along with lateral raises, full can (scaption), and scapula stabilization drills.

- At approximately week 6 to 7, the Thrower's Ten program is initiated; this program places emphasis on external rotator and scapula strengthening.
- No isolated biceps strengthening should be performed for the first 8 weeks to protect the healing biceps attachment into the labrum.
- Isotonic strengthening can be progressed by 1 pound per 7 to 10 days as long as there is no increase in pain or soreness.
- More aggressive strengthening such as manual resistance exercises, proprioceptive neuromuscular facilitation (PNF), and two-handed plyometrics may be added at 10 to 12 weeks following surgery.
- Functional activities such as one-handed plyometrics in the 90/90 position into a trampoline may be initiated approximately 2 to 3 weeks after initiation of two-handed plyometric drills.
- At week 12, machine weights such as lat pull downs and seated presses may be incorporated.
- The interval hitting program may also be initiated at week 12; this consists of a gradual progression beginning with hitting of a tee, progressing to soft toss, and finally taking batting practice off a live pitcher.
- At week 16, the athlete may begin the interval throwing program.
  - Athlete begins at 45 feet and progresses to 60, 90, and 120 feet.
  - Following the 150-foot phase, the athlete may begin flat-ground throwing using pitching mechanics.
  - After completion of the flat-ground throwing step is when the athlete can begin throwing fastballs off the mound at 50% of the athlete's normal velocity.
  - Velocity and number of pitches are gradually increased.
  - The throwing program takes approximately 4 to 6 months depending on timing of the upcoming season, position played, or any concomitant procedures performed during surgery that may delay onset or progression of the program.
- Return to play following a type II SLAP repair typically occurs at approximately 9 to 12 months following surgery.
- Rehabilitation should be based on specific injury (mechanism, location), surgery performed, and ultimate functional goals of the patient.
- Gradual restoration of ROM, strength, endurance, and dynamic stability is critical but it must be applied in a controlled manner to minimize stress on the healing tissue.
- Ultimate goal is to return the patient to prior level of function as safely and as quickly as possible without deleterious consequences.

Ulnar Collateral Ligament (UCL) Reconstruction

- The overhead-throwing athlete is susceptible to numerous elbow injuries because of the strong forces that act on the elbow during the throwing motion.
- The ulnar collateral ligament (UCL) is the main medial stabilizer of the elbow and is subject to high valgus stresses, particularly during the late cocking phase of the throwing motion.
- Injuries are caused by chronic stresses or repetitive microtrauma to the medial soft tissue structures that result from the athlete's attempts to stabilize the elbow joint.
- There exists a medial shear force of 300 N and a compressive force of 900 N.
  - The valgus stress that is applied to the medial elbow during the late cocking and acceleration phase of throwing is 64 Nm, which exceeds the strength of the UCL.
- Surgical reconstruction is employed to regain normal stabilizing function of the UCL, particularly the anterior bundle.
  - Autologous graft sources are typically either the palmaris longus tendon or the gracilis tendon.
  - Ulnar nerve transposition is often concomitantly performed at the time of reconstruction as well.
- Rehabilitation following UCL reconstruction is vital to fully restore normal function and return the athlete to competition as quickly and safely as possible.
  - Rehabilitation must be progressed to restore full ROM, strength, dynamic stability, and neuromuscular control while protecting the healing tissues.
- Rehabilitation generally commences prior to surgery through an educational component with the athlete and the family; this should include progression, brace use, and prognosis.
- Preoperative ROM measurements are generally obtained to gauge how much motion recovery after surgery should be expected.
- A general strength assessment is also performed to both the elbow joint and the shoulder joint musculature.
- Immediately following surgery the elbow is placed in a 90-degree splint for the first 5 days; this allows for adequate healing of the UCL graft and soft tissue sling of the transposed ulnar nerve.
- The patient performs wrist ROM and gripping exercises to improve overall circulation of the distal upper extremity.
- After approximately 5 days, the athlete is placed in a hinged elbow brace, which is adjusted to allow 30 to 100 degrees of ROM.
  - Motion is increased 10 degrees in each direction per week until full ROM is obtained at approximately 4 to 5 weeks postoperatively.
- Strengthening exercises progress from isometric contractions to isotonic exercises by week 4.
- The full Thrower's Ten program may be performed using light weights by week 6.
- Progressive resistance exercises for the elbow and shoulder dynamic stabilizers are emphasized through postoperative weeks 8 to 10.
- Wrist flexor and extensor stretches to increase flexibility are also incorporated at this time.
- Aggressive exercises involving eccentric and plyometric contractions are included beginning weeks 9 to 12.
- Two-handed plyometrics are added at week 10 and one-handed plyometrics are added approximately 2 to 3 weeks later.
- Interval throwing begins at approximately week 16.
- Return to competitive throwing usually occurs at approximately 8 to 10 months following surgery.

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Physical Modalities in Sports Medicine

38

Russell G. Steves

INTRODUCTION

Modalities are best thought of as an adjunct to the body's own recovery process. It would be a mistake to think of them as all that any patient needs to rehabilitate. However, they do have a role in therapy, especially in sports medicine, where any tool that hastens the return to play is valuable. Some may claim that certain modalities are an integral part of healing and recovery from injury, although minimal evidence exists to support many claims.

SUPERFICIAL HEAT

- Modality requires direct contact with skin.
- Greatest heating effect is achieved superficially.
- If maintained long enough, heat energy gets conducted into deeper structures.
- Increase in tissue temperature has the following beneficial effects:
  - Vasodilation
  - Increased blood flow to and from the site
  - Increased cell metabolism
  - Increased elasticity of collagen tissues
  - Decreased pain
  - Decreased muscle tone
  - Decreased muscle spasm

Devices for Heat

- Devices conduct thermal energy across the skin to provide a rise in tissue temperature.
- **Moist heat packs**
  - Canvas covers filled with silica gel
  - Heated in hot water tanks (approximately 160° F)
  - Packs placed in insulating layers of towels or cloth covers
  - Covers need to be kept clean to prevent spread of skin disorders
  - Heat dissipates quickly, usually within 15 minutes
  - Easier to get patient in comfortable position
- **Dry heating pads**
  - Plug-in electrical or microwaveable varieties
  - Do not heat tissue as rapidly nor as comfortably as moist heat packs
  - Heat does not dissipate as rapidly so can be used for longer time
  - Skin can be burned by prolonged or overly intense heat exposure
- **Whirlpools**
  - Body part immersed in tub with motor that moves water
  - Water temperature usually between 102° F and 110° F
  - Lower temperatures used when more of body is immersed
  - More of body part heated as it is surrounded by water
  - Water circulation keeps temperature next to skin constant and not dissipated into body part
  - Circulating water has massaging effect
  - Water is an excellent transmitter of bacteria into open wounds so care must be used in keeping the wound and the tank clean
  - Whirlpool can be used for debridement of superficial wounds
  - Clean whirlpools thoroughly to prevent disease transmission
- **Paraffin baths**
  - Mixture of wax and mineral oil melted to a liquid state (118° F to 126° F)
  - Apply to body part by dipping or brushing on
  - Effective with irregularly shaped body parts such as hands and feet
  - Multiple coats applied and allowed to cool
  - Cooling wax solidifies and thereby transfers heat into tissues
  - Low specific heat of wax allows for comfort at higher temperatures

Exercise for Heat

- Heat is by-product of muscular work
- The more intense the exercise, the greater and quicker the heating
- Heat is transmitted from muscle into other body tissues as well as carried away by the bloodstream
- Temperature increase occurs deeper in the tissues
- Combination of exercise and superficial heat modalities results in the greatest increase in tissue temperature

Uses of Treatment

- Injuries no longer in the acute inflammatory phase
- Chronic pain
- Injuries resulting in decreased range of motion (ROM)
- Swelling
- In preparation for therapeutic exercise
- Injuries in which the goal of treatment is to increase circulation

Contraindications

- Acute injuries (risk of increased swelling)
- Uncovered open wounds
- Tumors
- Thrombophlebitis
- Nerve sensitivities

Precautions

- Fair skin that burns easily
- Areas of decreased sensation
- Dermatologic problems and disease transmittal

CRYOTHERAPY

- Cryotherapy involves the application of cold to affect changes in the body for therapeutic benefit. Various methods are used (see "Methods of Cryotherapy").
- Each method attempts to draw heat from the body's tissues through the skin, which raises the temperature of the cold device rather than having the cold penetrate the body. This loss of thermal energy and subsequent decrease in tissue temperature results in:
  - Vasoconstriction
  - Decreased blood flow to the area
  - Decreased swelling
  - Reduction in inflammatory mediators and pain-producing substances
  - Decreased cell metabolism
  - Reduction of elasticity in collagen tissues
  - Slowed conduction of nerve impulses; analgesia
  - Decreased muscle spasm
  - Decreased force production in muscle
- Because of these responses to the cold application, cryotherapy is most successful as a treatment modality for acute injuries. The reduction in pain, swelling, and inflammatory reaction helps the patient's healing process progress more quickly, which allows a faster return to activity. Other conditions that can benefit from cryotherapy include:

  - Injuries in which pain is the predominant symptom
  - Postoperative conditions
  - Preexisting injuries (immediately following activity)
  - Problems in which pain inhibits activity or therapy
  - Situations in which anesthesia is desired
- Elevation of the injured body part to levels higher than the heart causes a decrease in vascular hydrostatic pressure. The force of gravity also increases venous and lymphatic return, further reducing the fluid collection in the injured area.
- The reduction in swelling at the time of injury decreases the pain for the patient as well as the time it will eventually take to return to full activity.
- The duration of cold application in acute injuries should not be a concern. Prolonged cold application, as occurs in the previously listed cryotherapy methods, has not been shown to result in a reflexive body warming. Longer cold application results in more beneficial effects in acute injuries.

Methods of Cryotherapy

- Ice bags
  - Crushed ice in plastic bags
  - Smaller ice cubes allow for greater surface coverage
  - Less air in ice bag allows for more ice in contact with skin
  - More conforming to body part, which also results in greater cold effects
  - Longer application means deeper cooling
  - Application usually 15 to 20 minutes
- Reusable ice packs
  - Silica gel pack kept in freezer
  - Allows for multiple uses
  - Requires insulating layer between pack and skin (wet towel) because pack can get colder than ice bags and result in skin irritation
  - Application 15 to 20 minutes
- Ice massage
  - Water frozen in paper cup and applied directly to skin
  - Peel paper from cup to expose ice
  - Effective at cooling superficial tissues
  - Movement and pressure of application has similar benefits to massage
  - Smaller area of cooling and labor intensive are drawbacks
  - Application 10 to 15 minutes
- Chemical cold packs
  - Water and ammonium nitrate separated within pack
  - Chemicals mix when barrier is broken by squeeze
  - Can be stored indefinitely and broken when needed
  - More expensive version of icing
  - Chemicals can irritate skin if bag leaks
  - Cold is not as long-lasting
- Ice immersion
  - Ice and water mixture in small container for lower legs and arms
  - Surrounds entire body part; helpful for joint injuries
  - Make immersion as cold as tolerable
  - With appendage immersion there is less core temperature reduction
- Ice water circulating units
  - Commercially available units that combine cold and compression
  - Cold therapy can be applied longer; stays cold longer
  - Use in different locations
  - Commonly used postoperatively or in chronic conditions; easier for multiple applications
  - More costly
- Cold baths
  - Larger-sized tanks for immersion of more of the body
  - Used for cooling larger, deeper body areas
  - May use whirlpool action to keep colder at skin contact areas
  - To achieve therapeutic benefit while avoiding too large a drop in core temperature, recommend bath temperatures 50° F to 60° F.
  - Used more frequently for muscle recovery than injury care
  - Application 10 to 15 minutes
  - Similar worries as warm whirlpool concerning open wounds and hygiene
- Vapocooling sprays
  - Chemical spray (ethyl chloride) applied to skin
  - Cools rapidly and evaporates quickly
  - Cools only superficially
  - Used whenever quick numbing is desired; trigger point treatment

R.I.C.E.

- Use rest, ice, compression, and elevation (RICE), especially with acute injuries.
- Compression can take the form of:
  - Tightly applied ice bag
  - Ice bag applied over top a wetted ace wrap (to enhance thermal exchange)
  - Adhesive expandable tape job
  - Commercially designed device
- Compression works by:
  - Increasing heat exchange by squeezing tissues together and closer to the surface
  - Decreasing tissue spaces where fluid can accumulate
  - Decreasing blood flow so that less warm blood gets into affected area
  - Increases contact area of applied cold to skin

WHEN TO USE HEAT VERSUS COLD

- It is not uncommon for a clinician to be confronted with the dilemma of when to use heat or when to use cold.
- Most simplistic guide to follow is, **if pain is the primary symptom, use cold; if stiffness is the chief complaint, use heat.**
- Injuries in the acute inflammatory phase need to have ice applied. As an injury gets more swollen, it becomes more painful and will also take longer to get better. Modalities that increase temperature will result in more swelling. The duration of the acute injury phase can be variable, but certainly no less than 24 hours postinjury.
- Application of ice is effective as an anesthetic. When the patient has pain at rest or has high levels of pain, use some form of cryotherapy.
- Heat can be applied safely whenever the patient is out of the acute inflammatory phase.
- The application of heat prior to exercise can often make the workout more comfortable. The tissues are more elastic and flexible. Most bodily functions work more effectively at slightly higher temperatures, especially those relating to athletic activity.
- After the completion of a workout, especially if the workout produced any pain in the injured area, ice is the proper treatment. This can reduce any inflammatory effects brought on by the activity.
- Some chronic conditions may still benefit from cryotherapy. With the presence of a long-term injury it is not always so clear which will be the most effective modality. The clinician may have to use trial and error or revert to the guidelines listed above.
- When in doubt, apply ice. There is less risk of improper application of ice compared with heat.

ULTRASOUND

- Therapeutic ultrasound has thermal and nonthermal effects.
- **Thermal effects**
  - Increased tissue temperature

  - Increased cellular activity
  - Increased blood flow
  - Increased tissue extensibility
  - Reduced muscle spasm
  - Reduced pain
- **Nonthermal effects**
  - Acoustic streaming—movement of fluids along cell membranes.
  - Cavitation—formation of gas-filled bubbles.
  - Both of these nonthermal effects are thought to facilitate tissue repair.
- Ultrasound is most effective when there is a relatively small treatment area. Target area is two to three times the size of the device's sound head, called the effective radiating area (ERA). Conditions that require treatment over a large area will result in longer treatment durations that may become too time-consuming for the clinician.
- Sound head must be kept moving (2 inches per second recommended) during the treatment. Accumulation of the thermal energy can become uncomfortable.
- Amount of ultrasound energy delivered is the dosage. This varies based on the desired treatment effect. As the dosage gets higher, the thermal effects get greater and become the limiting factor because of discomfort. The clinician must select from treatment parameters (Table 38-1).
- Therapeutic ultrasound is often thought of as a deep heating modality, but it can also be used for heating superficial structures and can have nonthermal effects.
- Indications for ultrasound:
  - Conditions that benefit from increased heating include tendinitis, myositis, arthritis, sprains, strains, etc.
  - Relief of pain and muscle spasm.
  - Acute conditions (using nonthermal settings).
- Contraindications for ultrasound: ischemic areas; deep vein thrombosis; anesthetic areas; active infections; tumors; external fixation devices; injury to eyes, heart, skull, genitals; over the trunk during pregnancy or menstruation; over stress fractures or osteoporotic areas.

Phonophoresis

- Phonophoresis involves the use of ultrasound energy to assist the diffusion of medication through the skin and into the target tissues.
- Corticosteroids are used to reduce inflammation; salicylates are used to relieve pain.
- Clear gels are better at conducting media than thick, white creams.
- Standard parameters for phonophoresis have not been established; use general ultrasound protocols.
- There is little understanding of how much medicine gets delivered or is required to have an effective outcome.
- Factors affecting how much medicine reaches tissues include:
  - Skin's water content
  - Patient's age
  - Skin composition
  - Skin thickness
  - Skin vascularity

Table 38-1 ULTRASOUND TREATMENT PARAMETERS

Frequency	1 MHz—penetrates to deeper tissues; up to 1 inch 3 MHz—affects superficial structures
Intensity	The higher the intensity, the greater the thermal effects When goal is heating, select the highest comfortable intensity
Duty cycle	Regulates the "on" time of the machine The higher the percentage (up to 100%), the greater the thermal effects
Duration	Longer durations for larger treatment areas

Ultrasound Bone-Growth Stimulators

- Ultrasound energy used to facilitate fracture healing
- Low-intensity, pulsed ultrasound used
- Devices made specifically for this purpose
- Application is 20 minutes daily
- Ultrasound energy thought to apply low-level mechanical force to reduce fracture healing time
- Limited studies demonstrating effectiveness
- Few reports of adverse effects

ELECTRICAL STIMULATION

- Some of the body's tissues with higher water content, such as nerves, muscles, and cell membranes, are directly excitable by electrical current.
- Structures such as bones, cartilage, tendons, and ligaments may be affected by the electric fields caused by the current.
- Electrical muscle stimulation (EMS) is used for:
  - Pain reduction
  - Swelling reduction
  - Muscle spasm reduction
  - Facilitating muscle contractions
  - Minimizing muscle atrophy
  - Muscle strengthening
  - Facilitating fracture healing
  - Facilitating inflammation reduction
- In **direct current (DC)** electrical stimulation, one electrode is positive and the other is negative.
- **Alternating current (AC)** electrical stimulation, the more common application, can be either *monopolar*, with electrodes of unequal size (one designated "active," the other "dispersive"), or *bipolar*, in which the electrode polarity changes many times per second and both electrodes are of equal size.
- EMS has been shown to stimulate sensory, motor, and pain nerve fibers given the appropriate stimulation parameters.

Transcutaneous Electrical Nerve Stimulation

- Transcutaneous electrical nerve stimulation (TENS) is the most common form of EMS used for pain reduction.
- **High-rate (sensory) TENS**
  - Pulse frequency: 60 to 120 Hz
  - Phase duration: short (<150 μsec)
  - Intensity: highest tolerable without eliciting muscle twitch
  - Treatment goal: pain reduction through stimulation of large diameter (A-beta) nerve fibers; quicker acting, shorter duration pain relief
- **Low-rate (motor) TENS**
  - Pulse frequency: 2 to 10 Hz
  - Phase duration: long (200 to 300 μsec)
  - Intensity: highest tolerable muscle twitch
  - Treatment goal: pain reduction through stimulation of smaller diameter (A-delta) nerve fibers; slower acting, longer duration pain relief
- **Noxious TENS**
  - Pulse frequency: high or low
  - Phase duration: very long (>300 msec)
  - Intensity: painful; highest tolerable
  - Treatment goal: pain reduction through stimulation of smallest nerve fibers (C)
- Small, portable TENS units with current generated by batteries instead of wall outlets are used to help patients get pain relief for longer periods of time.

Interferential Current

- Interferential current is another variation of EMS used for pain relief.
- Main advantage is deeper penetration.
- Uses four electrodes (two pairs of slightly different medium frequencies).
- Arrange electrode pairs diagonally so that the currents interfere.
- Resultant current is what determines treatment.
- Can be high-rate TENS or low-rate TENS.
- Configure electrodes so that effect is felt over desired area.

High-Voltage Stimulators

- High-voltage stimulators are also known as high-volt galvanic stimulators.
- Primarily used for pain modulation.
- Use monophasic current of ≥150 volts.
- Higher voltage results in deeper penetration.
- Average current is low, so safe for patients.
- Phase duration is too short to mimic uncomfortable effects of DC.
- One smaller electrode is "active"; the other, larger electrode is "dispersive".
- Electrode polarity is interchangeable.

Neuromuscular Electric Stimulation (NMES)

- Neuromuscular electric stimulation (NMES) is used for muscle activation and strengthening.
- NMES activates muscles by stimulating alpha motor neurons that often become inhibited with joint injury pain and swelling.
- Muscle activation can be thought of as lower-level strengthening.
- NMES current needs long phase duration to recruit as many motor units as possible.
- Current frequency of 35 to 50 pulses per second is needed to achieve tetany.
- The stronger the muscle contraction, the more rest incorporated into treatment cycle.
- Larger fast-twitch fibers are recruited first and more easily fatigued.
- By convention, use 5:1 rest-to-work time ratio.
- NMES for strengthening is more apt to be uncomfortable.
- Better strengthening effect if voluntary muscle contraction is superimposed over NMES stimulation.

Iontophoresis

- Iontophoresis uses electric current used to move ions across skin barrier for reduction of pain and inflammation.
- It involves a DC generator with monopolar setup: one large dispersive electrode, one small active electrode.
- Clinician selects electrode's polarity depending on polarity of medication.
- Polarity of commonly selected medications:
  - Salicylate (−)
  - Hydrocortisone (+)
  - Lidocaine (−)
  - Dexamethasone (−)
- Mixture of lidocaine-dexamethasone most commonly used.
- Lidocaine acts to reduce skin irritation at treatment site and can also reduce the amount of dexamethasone pushed through the skin.
- Now manufacturers make buffered electrodes that eliminate the need for lidocaine.
- Maximum current for each treatment should be 5 microamps; maximum dosage 80 microamps per minute.
- For each treatment, set maximum dosage first, then highest tolerable current; treatment time will result from that.
- No standard number for iontophoresis treatments has been determined.

Stimulation of Denervated Muscle

- In this treatment, denervated muscle from injury or disease is stimulated by DC.
- Current is applied directly to muscle to lessen atrophy.
- Alpha motor neurons can regenerate, as opposed to injured upper motor neurons in the spinal cord, which cannot regenerate.
- Because muscle cells are being stimulated directly and their capacitance is large, current phase duration must be very long (too long for AC).
- Stimulation is uncomfortable to patient because small diameter C nerve fibers that carry noxious pain are stimulated.
- Efficacy of DC stimulation of denervated muscle has not been proven.

Microcurrent

- Microcurrent treatments use very low current (microamps) to modulate pain, reduce swelling, and heal skin lesions.
- The current used is too low to depolarize nerve.
- Theoretically, subthreshold stimulation enhances adenosine triphosphate (ATP) production, which then affects cell function.
- Very little research exists to support claims of microcurrent's benefit.

Electrical Bone-Growth Stimulators

- Electrical bone-growth stimulators attempt to introduce electrical field to aid fracture healing.
- They use an electrical field similar to that produced by normal body activities, thus stimulating bone growth in accordance with Wolff's law.
- Devices will have external electrodes placed transcutaneously using AC or implanted electrodes using DC.
- Effectiveness of electrical bone-growth stimulators has not been shown conclusively.

Contraindications for EMS

- Heart/respiratory conditions: current through the chest and neck may disrupt normal functioning.
- Applied over carotid sinus, esophagus, larynx, pharynx, eyes, upper thorax, temporal regions: all have enhanced nerve sensitivity and may be easily intolerable.
- Pacemakers: current can interfere with device's function.
- Pregnancy and menstruation: avoid current over abdomen, pelvis, or lumbar areas.
- Tumors: current can stimulate growth.
- Active infections.
- Exposed metal implants: results in electrical shock.
- Electronic monitoring equipment: interferes with device's proper function.

DIATHERMY

- Diathermy uses high-frequency electromagnetic waves to heat tissues; also known as shortwave diathermy.
- This treatment is chosen for its ability to penetrate deeper and over a larger area than other heating modalities.

Methods of Application

- Capacitance technique
  - Body part placed between two electrodes
  - Resultant electric field cause structures in body that have a negative and positive pole (dipoles) to rotate
  - Dipole movement causes heat in the tissues

  - Muscle tissue has more dipoles and thus greater capacitance and requires more current to achieve desired effect
  - Fatty tissue and skin tissue have fewer dipoles and so have greatest heating
- Inductance technique
  - Electromagnetic field generated by passing current through coiled wire
  - Coiled wire wrapped around patient or inside a drum
  - Patient not part of electrical circuit
  - Tissues such as blood vessels and muscle, which are better conductors, see more increase in temperature
  - Adipose and skin see less increase in temperature

Indications

- Same as for any other heating modalities
- Has larger effective treatment area and longer heat retention than other deep-heating modality and ultrasound.

Contraindications

- Acute inflammatory conditions
- Metal implants or jewelry
- Cardiac pacemakers
- Over the eyes or genitalia
- Pregnancy
- Tumors
- Open wounds
- Infection
- Peripheral vascular disease
- Water collection over the skin
- Areas of sensory loss

LOW-LEVEL LASER THERAPY

- Low-level laser therapy (LLLT) makes use of light energy (laser) delivered to body's tissues for therapeutic benefit.
- Laser delivers its energy to a small target area.
- Therapeutic use of lasers is tightly controlled in the United States by the Food and Drug Administration (FDA) and approval has been granted only for:
  - Pain resulting from minor neck and shoulder problems
  - Carpal tunnel syndrome
- Medical lasers' classifications (1 to 4) are based on amount of energy produced and safety risks.
- LLLT devices are class 3b lasers
  - Medium power devices (5 mW to 500 mW)
  - Maximum power allowed: 90 mW
  - May be referred to as "cold lasers"
  - Hazard if viewed directly
  - Not normally a fire hazard
  - Cannot heat tissues above 36.5° C
- LLLT devices use different gases to produce laser energy; different gases result in different wavelengths; longer wavelengths penetrate deeper into tissues.
- LLLT effects have been shown to occur to 15 mm.
- LLLT has multiple therapeutic uses. Treatment dosage depends on the intended therapy, and is defined by the machine's output power, which is not adjustable, the duty cycle, and the treatment time.
- **Precautions with the use of LLLT:**
  - Pain may occur the day following treatment, which is thought to be an activation of the tissue-healing mechanism.
  - Avoid use within 6 months of radiation therapy.
  - Patient dizziness may occur and with continued occurrence must cease treatment.
  - Avoid treatment to trunk during pregnancy and to unfused epiphyseal plates.
  - Avoid use with small children.
- Contraindications for LLLT include use over cancerous areas, over the eyes, and in areas of bleeding.

MAGNETS

- It may be useful to understand what lies behind magnet therapy, although it is not an obvious therapeutic modality.
- Magnetic fields are commonly used for therapeutic benefit; magnetic fields used for therapy result from an AC source.
- Therapeutic magnets are low-power static magnets worn on the body. (Static magnets have a positive and negative pole.)
- Proponents state that magnets can be used to restore the body's natural magnetic field alignment, which becomes abnormal with injury or disease.
- The power of therapeutic magnets ranges from 500 to 1000 Gauss (G).
- Magnets over 1000 G are regulated by the FDA. (The power of an average refrigerator magnet is 4 G.)
- The power of magnets used for magnetic resonance imaging (MRI) is 15,000 G.
- The World Health Organization (WHO) has deemed magnets up to 20,000 G safe.
- There are two techniques used for the application of therapeutic magnets:
  - Unipolar magnet: one pole (usually negative) touches skin.
  - Bipolar magnets: both poles touch skin and are arrayed in patterns to make optimal use of polar effects.
- Little or no scientific evidence exists to demonstrate the therapeutic value of these low-power magnets. Reports of benefits are all anecdotal.

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Head Injuries

39

Margot Putukian and Christopher C. Madden

GENERAL PRINCIPLES

- Head injuries in sports are comparatively mild compared with those in high-velocity motor vehicle accidents, yet remain significant and important injuries for team physicians to evaluate and manage.
- **Concussion** is the most common head injury in sports. Information is evolving regarding pathophysiology, diagnosis, natural history, and treatment of concussion in sports.
- Consider focal, vascular, and associated injuries (e.g., cervical spine, skull fractures) when evaluating head-injured athletes.
- Cumulative injury, postconcussive syndrome, and other sequelae of head injury may contribute to significant morbidity.

EPIDEMIOLOGY

- **Many head injuries go undetected,** especially in younger age groups.
- Athletes often underreport symptoms; thus injuries often are underreported.
  - Athletes may take injuries lightly, viewing them as "part of the game."
  - Athletes may fear being taken out of game if they admit symptoms.
  - Head injuries may be evaluated on sideline but not referred for further evaluation or athlete may fail to follow up with appropriate referral.
- According to the National Head Injury Foundation, sports cause 18% of minor head injuries compared with motor vehicle accidents (46%), falls (23%), and assaults (10%).
- Head injury accounts for 4.5% of all high school sports injuries and 19% of all nonfatal injuries in football.
- Every year 300,000 head injuries occur, with an average of eight deaths in football players per year.
- The National Collegiate Athletic Association (NCAA) Injury Surveillance System (ISS) uses injury definition of time lost from participation and provides denominator of athlete exposure, allowing true incidence of injury to be determined.
  - For the period from the 1988-1989 school year through the 2002-2003 year, concussions accounted for between 5% and 18% of all reported injuries, 14% of which were associated with 10 or more days of restriction from activity. Overall incidence of concussion has increased 7% from 1988-1989 to 2002-2003; the increase is likely related to increased reporting, greater awareness of injury, and better identification by medical staff (see Table 39-1).
  - Injury rates are comparable in sports with and without head protection.
  - Concussion rates are highest in women's ice hockey, followed by football, men's ice hockey, men's and women's soccer, field hockey, and men's lacrosse. Player contact is primary mechanism of injury in all sports but field hockey (contact with stick) and baseball, softball, and women's lacrosse (contact with ball).
  - There appears to be a higher incidence of injury in women when compared to men playing similar sports (soccer, basketball, ice hockey) and also some evidence to suggest that recovery is also different in men than women. Though women may be more likely to report symptoms and/or be more truthful about their injuries, the exact reasons for these gender differences are unclear.

Table 39-1 NCAA INJURY SURVEILLANCE SYSTEM DATA FOR 1988-1989 THROUGH 2002-2003

Head protection required	% of all game injuries	No head protection	% of all game injuries
Men's ice hockey*	9.0	Women's lacrosse*	9.8
Women's ice hockey	21.6	Wrestling	4.8
Men's lacrosse*	8.6	Women's soccer	8.6
Football*	6.8	Men's soccer	5.8
Spring football* (practice)	5.6	Field hockey	9.4
Softball	6.0	Women's basketball	6.5
Baseball	3.3	Men's basketball	3.6
Concussion injuries in games per 1000 athlete exposures			
Head protection required		*No head protection*	
Football*	2.34	Wrestling	1.27
Men's ice hockey*	1.47	Men's soccer	1.08
Women's ice hockey	2.72		
Men's lacrosse*	1.08	Women's soccer	1.42
Spring football*		Women's lacrosse*	0.7
Softball	0.25	Field hockey	0.52
Baseball	0.19	Women's basketball	0.5
		Men's basketball	0.32
Concussion injuries in practices per 1000 athlete exposures			
Head protection required		*No head protection*	
Football*	0.21	Wrestling	0.14
Men's ice hockey*	0.10	Men's soccer	0.08
Women's ice hockey	0.33		
Men's lacrosse*	0.12	Women's soccer	0.12
Spring football*	0.54	Women's lacrosse*	0.15
Softball	0.07	Field hockey	0.09
Baseball	0.03	Women's basketball	0.15
		Men's basketball	0.12

*Mouth guard required.

Data from Hootman J, Agel J, Dick R: Epidemiology of collegiate injuries for 15 sports: Summary and recommendations for injury prevention initiatives. J Ath Training 42(2):311-319.

- **Look at epidemiology when considering injury prevention.** Before making rule or equipment change, consider incidence of injury and how change may affect sport.
  - Use of helmet in women's lacrosse and field hockey may decrease incidence of facial lacerations, nasal fractures, or dental injuries but may not significantly affect incidence of concussions. Increased aggressiveness with added head protection may negatively affect sport.
  - One study in ice hockey reported that 75% of 246 head injuries involve violence unrelated to on-ice activities (high sticking, deliberate pushing, fistfights).
  - Proper enforcement of existing rules (avoiding head-to-head hits) is essential.
  - Use of helmets significantly decreases risk of head injury in bicycling, baseball, skiing, snowboarding, and softball without negatively affecting sports.
  - No evidence that helmets or mouth guards prevent concussion. Helmets can decrease incidence of skull fracture because of their protective plastic shell, and mouth guards decrease incidence of tooth injury.

TYPES OF HEAD INJURY

Head injuries occur across a large spectrum. Classifications such as focal versus diffuse or structural versus nonstructural are not absolute but permit organized discussion of specific pathologies.

Diffuse Brain Injury

Diffuse Axonal Injury (DAI)

- Involves diffuse axonal disruption in white matter of brain and brainstem.
- Severity of injury determined by clinical course.
  - Mild DAI: comatose for 6 to 24 hours; mortality rate is approximately 15%.
  - Moderate DAI: comatose for more than 24 hours; often associated with basilar fracture; mortality rate is approximately 25%.
  - Severe DAI: prolonged coma, severe disability, or persistent vegetative state common if patient survives; high mortality rate; death often caused by infectious complications, associated intracranial pathology, and other complications of prolonged coma.
- Caused by **shear or tensile forces;** often results from falls or motor vehicle accidents.
- **Presentation:** All patients present in coma, may exhibit decorticate or decerebrate posturing, severe posttraumatic amnesia, and cognitive deficits after awakening (moderate DAI); severe DAI often includes hypertension and hyperpyrexia (autonomic dysfunction), increased intracranial pressure (ICP), posturing, and herniation syndromes.
- **Treatment:** Supportive during coma; medical or surgical measures as needed for increased ICP and associated injuries.

Cerebral Concussion

- First International Conference on Concussion in Sport defined concussion as a complex pathophysiologic process affecting the brain, induced by traumatic biochemical forces.
- Several common features that incorporate clinical, pathologic, and biomechanical injury constructs that may be used in defining the nature of a concussive head injury include:
  - May be caused by direct impact to head or elsewhere on body with "impulsive" force transmitted to head.
  - Results in rapid onset of short-lived impairment of neurologic function that usually resolve spontaneously.
  - May result in neuropathologic changes, but acute clinical symptoms reflect functional rather than structural disturbance.
  - Results in graded set of clinical syndromes that may or may not involve loss of consciousness. Resolution of symptoms typically follows sequential course.
  - Typically associated with grossly normal structural neuroimaging studies.
- Cerebral concussion is the **most common head injury in athletes;** isolated concussion has low mortality rate.
- Caused by **acceleration/deceleration** (tensile), **rotational** (shearing), and **impact** (compressive) forces. Coup injuries often result from direct impact, contrecoup injuries from acceleration/deceleration forces (e.g., athlete falls and strikes ground with head).
- **Presentation:** Hallmark of concussion is confusion; other signs and symptoms may occur immediately or several minutes later (Table 39-2).
- **Treatment:** Rest (physical and cognitive), protection from further injury, and serial follow-up evaluations (see detailed discussion below).

PATHOPHYSIOLOGY OF CONCUSSION

- Concussion is associated with neurochemical and metabolic changes with changes in glutamate, potassium, lactate, and glucose, as well as changes in cerebral blood flow.

Table 39-2 SIGNS AND SYMPTOMS OF CONCUSSION

Early (minutes to hours)	Late (days to weeks)
Cognitive	Persistent low-grade headache
Confusion	Lightheadedness
Vacant stare	Poor attention and concentration
Slow to answer questions or follow instructions	Memory dysfunction
Easily distracted	Anomia (cannot think of word one wants to say)
Inability to focus	Easy fatigability
Feeling "in a fog"	Irritability and frustration
Disoriented: unaware of time/date/place	Difficulty with focusing vision
Slurred or incoherent speech	Photophobia
Memory deficits	Phonophobia
Repeatedly asks same question (e.g., what happened?)	Anxiety and/or depression
Retrograde amnesia (RGA): Cannot remember events before injury	Sleep disturbance
Posttraumatic amnesia (PTA): Cannot remember events after injury	Persistent cognitive deficits
Loss of consciousness	Postconcussive syndrome
Somatic	
Gross uncoordination: cannot walk straight line	
Headache	
Dizziness, disequilibrium or vertigo	
Visual disturbances (blurry vision, photophobia)	
Phonophobia	
Fatigue	
Headache	
Nausea and/or vomiting	
Dizziness, disequilibrium or vertigo	
Visual disturbances (blurry vision, photophobia)	
Phonophobia	
Fatigue	
Seizure	
Affective	
Emotional lability: may cry for no apparent reason	
Irritability	
Nausea and/or vomiting	
Seizure	

- No current objective neuroanatomic or neurophysiologic measurements can be used practically and reliably to determine if athlete has concussion. See "Diagnostic Testing."
- After concussion brain cells may be in state of **injury-induced vulnerability;** second injury during this time of heightened vulnerability may produce irreversible damage (e.g., second-impact syndrome).
  - Injury-induced vulnerability characterized by **fuel need–fuel delivery mismatch.** Brain's need for glucose increases acutely (hyperglycolysis), and cerebral blood flow and oxidative metabolism are relatively reduced (sometimes called disruption of metabolic autoregulation).
  - Increased levels of extracellular potassium probably activate adenosine triphosphate (ATP)-dependent sodium-potassium pumps, which add to metabolic stress (e.g., need for glucose).
  - Glutamate (excitatory amino acid) increases extracellularly and may contribute to increased flux of potassium.
  - Increased intracellular calcium may be related to regional reduction of cerebral blood flow.
  - Not known whether normalization of injury-induced neurometabolic and neurochemical abnormalities correlates with resolution of concussive signs and symptoms, but timeline of changes correlates with changes in neuropsychological function.

Focal Brain Injury

Subdural Hematoma

- Low-pressure venous bleed into potential space (subdural) between arachnoid and dura mater; classified by time to clinical presentation (see Fig. 39-1).
  - **Acute:** within 24 hours; often associated with other intracranial pathology (e.g., contusion, axonal injury).
  - **Subacute:** 24 hours to 2 weeks.
  - **Chronic:** 2 weeks or more.
- Leading cause of death related to head injury; overall mortality rate of 35% to 50%; loss of consciousness implies poor prognosis.
- Elderly and alcoholics at greatest risk because of increased space between brain (atrophy) and dura.
- Caused by brain movement within skull: acceleration-deceleration, rotational, shearing injuries.
- **Presentation:** Decreased/altered level of consciousness, lucid interval followed by declining mental status, headache; patients may have pupil inequality, motor deficit (e.g., unilateral weakness or paralysis), or other findings of brain swelling.
- **Treatment:** Usually prompt surgical evacuation; patient may be observed if prognosis is poor (e.g., prolonged loss of consciousness) or if elderly and asymptomatic (see Fig. 39-1).

Epidural Hematoma

- High-pressure arterial bleed between inner table of skull and dura mater (epidural space); middle or other meningeal arteries often disrupted; 80% associated with skull fracture in temporoparietal region (fracture less common in children); occasionally caused by tear of underlying dural sinus (most common in posterior fossa) (Fig. 39-2).
- Other associated intracranial pathology common (e.g., subdural bleed, contusion).
- Mortality rate low if diagnosed acutely; comatose state associated with highest mortality rate (≈20%); mortality rate higher with associated injuries.
- Caused by direct blow to head.

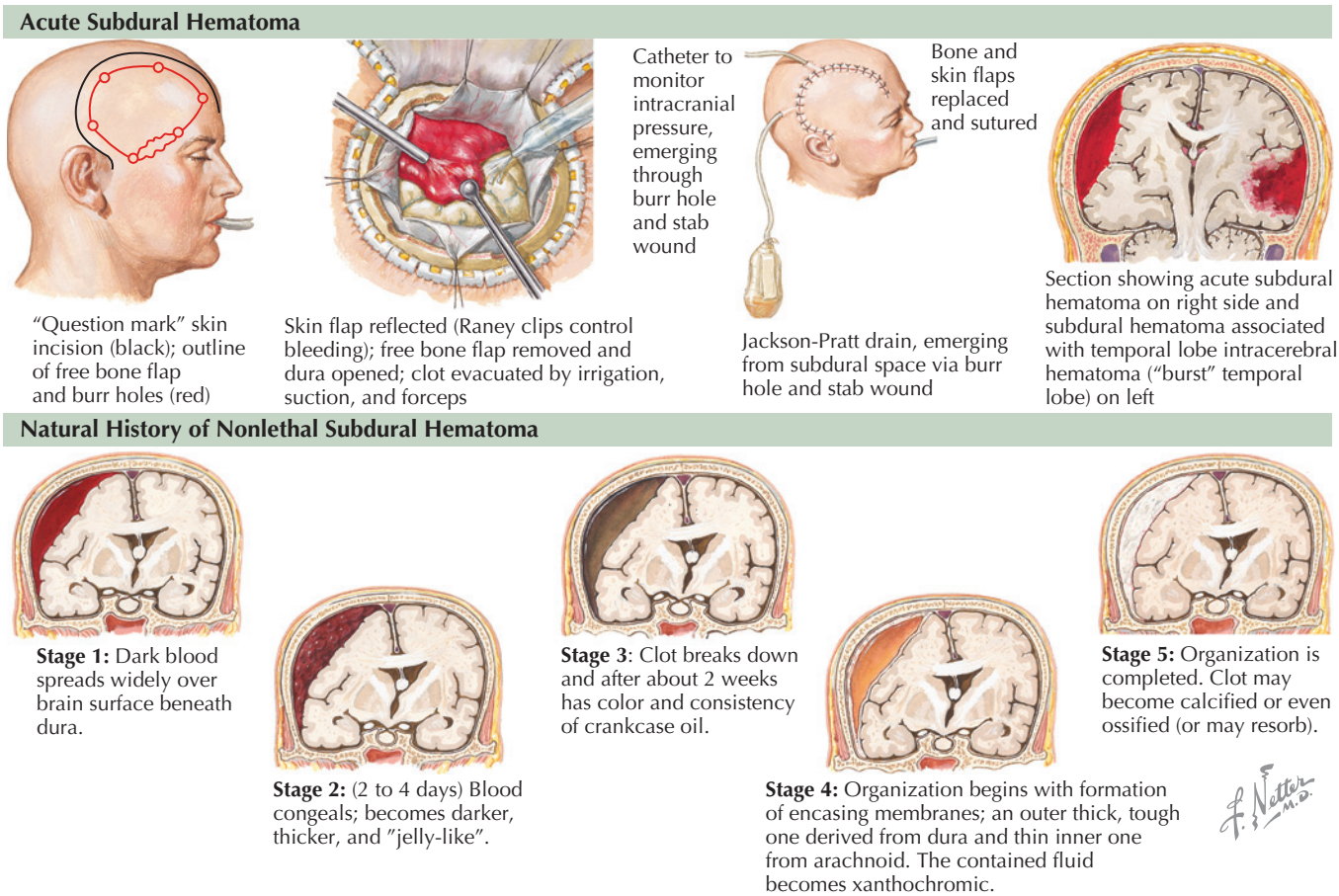


Figure 39-1 Subdural Hematoma.

- **Presentation:** Decreased level of consciousness followed by lucid interval, deteriorating mental status with eventual loss of consciousness, headache, confusion, sleepiness, nausea, vomiting. Only one third present classically with loss of consciousness followed by lucid interval and focal deficits.
- **Late signs:** Ipsilateral dilated pupil, contralateral muscle weakness, coma.
- **Lucid interval may last for several hours and lead to false reassurance and missed diagnosis.**
- **Treatment:** Craniotomy/evacuation of hematoma.

Intracerebral Hemorrhage/Hematoma

- Bleeding from small-caliber arterioles within brain parenchyma; frontal and temporal lobes affected most often; may be accompanied by **brain laceration** (Fig. 39-3).
- Mortality rate low if patient is conscious before intervention; may approach 45% in unconscious patients.
- Caused by tensile or shearing forces that stretch brain (coup or contrecoup mechanisms of injury).
- **Presentation** varies with size and location of lesions as well as associated pathology (e.g., contusion, postinjury edema); loss

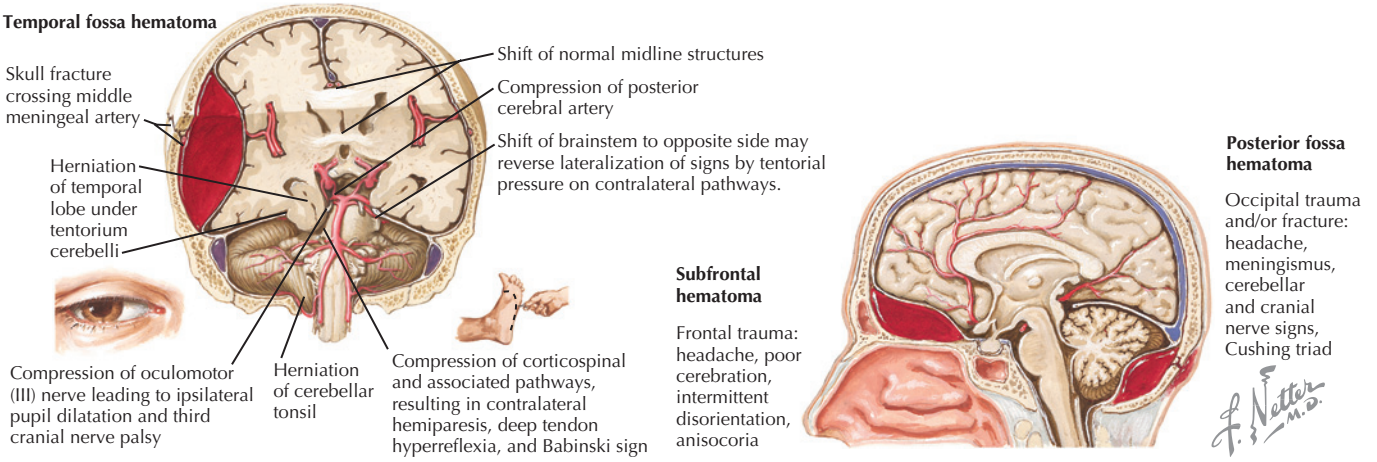


Figure 39-2 Epidural Hematoma.

Pathology	CT scan	Pupils	Eye movements	Motor and sensory deficits	Other
Caudate nucleus (blood in ventricle)		Sometimes ipsilaterally constricted	Conjugate deviation to side of lesion; slight ptosis	Contralateral hemiparesis, often transient	Headache, confusion
Putamen (small hemorrhage)		Normal	Conjugate deviation to side of lesion	Contralateral hemiparesis and hemisensor loss	Aphasia (if lesion on left side)
Putamen (large hemorrhage)		In presence of herniation, pupil dilated on side of lesion	Conjugate deviation to side of lesion	Contralateral hemiparesis and hemisensory loss	Decreased consciousness
Thalamus		Constricted, poorly reactive to light bilaterally	Both lids retracted; eyes positioned downward and medially; cannot look upward	Slight contralateral hemiparesis, but greater hemisensory loss	Aphasia (if lesion on left side)
Occipital lobar white matter		Normal	Normal	Mild, transient hemiparesis	Contralateral hemianopsia
Pons		Constricted, reactive to light	No horizontal movements; vertical movements preserved	Quadriplegia	Coma
Cerebellum		Slight constriction on side of lesion	Slight deviation to opposite side; movements toward side of lesion impaired, or sixth cranial nerve palsy	Ipsilateral limb ataxia; no hemiparesis	Gait ataxia, vomiting

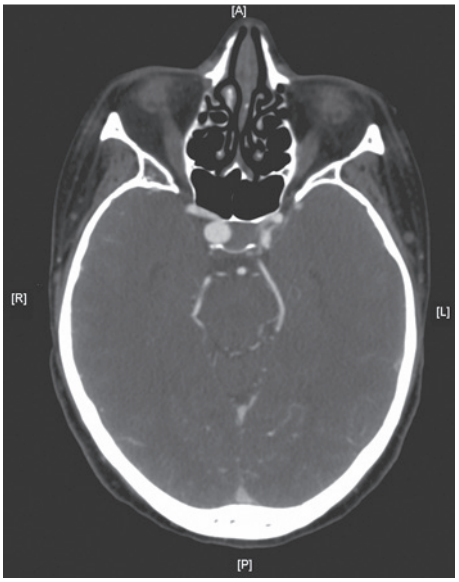
F. Netter M.D.

Figure 39-3 Intracerebral Hemorrhage: Clinical Manifestations Related to Site.

of consciousness (≈50%), headache, confusion, nausea, vomiting, focal deficits (affected areas) (see Fig. 39-3). Symptoms may develop over hours or days.
- **Treatment:** Many require emergent intervention to lower ICP and/or stop bleeding; depends on severity of clinical presentation, bleed, and associated pathology.

Subarachnoid Hemorrhage (SAH)

- Bleeding between arachnoid and pia mater (subarachnoid space) into cerebrospinal fluid (CSF); may be **traumatic** or **spontaneous.**
- Traumatic SAH results in small tears of subarachnoid vessels; spontaneous SAH (and sometimes traumatic SAH) often associated with intracranial aneurysms and arteriovenous malformations and sometimes with hypertension and arteriosclerosis (Fig. 39-4).
- May be most common abnormality after head injury (all causes); isolated traumatic SAH has low mortality rate; associated pathology (e.g., contusion, skull fracture) leads to less favorable outcome.
- Caused by tensile or shearing forces.
- **Presentation** depends on associated pathology and whether SAH is traumatic or spontaneous. Symptoms include headache ("worst ever"), photophobia, nausea, vomiting, dizziness, confusion, neck stiffness, focal deficits (affected areas). Injury often complicated by posttraumatic cerebral vasospasm 2 days to 2 weeks after acute bleed; causes neurologic deterioration and may be mistaken for second bleed.
- Must be differentiated from "bad" migraine and meningitis. Lumbar puncture done only after funduscopic exam (to rule out papilledema) and CT scan. Xanthochromia of CSF may be most specific finding with SAH.
- **Treatment** varies with individual case and pathology; may involve surgical intervention (e.g., clipping of aneurysm), medical management (e.g., calcium channel blocker for vasospasm), and conservative measures.



CT Angio source image showing an aneurysm.

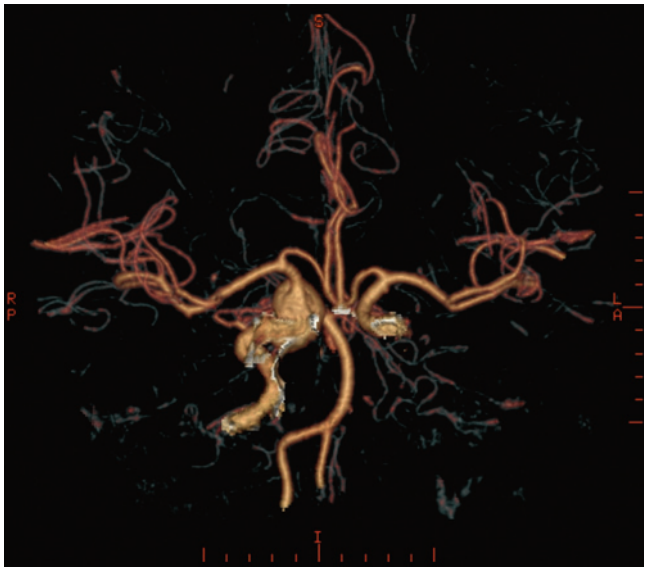

CTA 3-D reconstruction showing detailed anatomy of the aneurysm.

Figure 39-4 Subarachnoid Hemorrhage.

Other Injuries

Scalp Laceration

- Scalp has many layers and generous blood supply. Large lacerations or avulsions may be significant source of bleeding.
- All scalp lacerations should be examined for "step-off" deformities that indicate underlying depressed skull fracture.
- Hemostasis may be obtained with direct pressure (if no skull fracture is present) followed by quick, careful closure of wound, usually in single layer. Wounds should be debrided and irrigated well prior to repair.

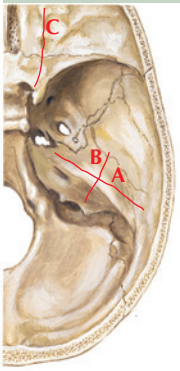
Skull Fracture

- Uncommon in athletics, but must always be considered.
- Classified as linear or depressed (open or closed).
- Caused by direct impact; usually force of impact large enough to cause underlying brain injury.
- Presentation varies with type of fracture and associated brain injury.
- **Nondepressed linear fractures** may cause only localized pain and swelling; basilar fractures (linear) often occur in petrous portion of temporal bone and may present with hemotympanum, otorrhea, or rhinorrhea (CSF leak), periorbital ecchymosis (raccoon eye), or retroauricular ecchymosis (Battle sign) (Fig. 39-5).
- **Depressed fractures** are noted by palpating "step-off" beneath skull laceration (considered open if scalp is disrupted) (see Fig. 39-5). Some are associated with loss of consciousness, nausea, vomiting, and other neurologic deficits, depending on extent of underlying brain tissue injury.
- Open skull fractures have increased risk of infection and seizures.
- CT scan with bone window is more accurate than plain skull x-rays and provides information about depressed skull fragments and associated intracranial pathology.
- Neurosurgical consultation is advised with all depressed skull fractures. Treatment is individualized and based on the specific location and pathology. Open depressed skull fractures often require prophylaxis for posttraumatic seizures and infection.

EVALUATION

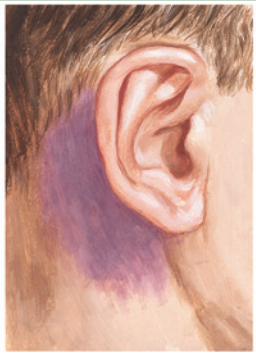
Evaluation and management of head-injured athletes must be complete, rigorous, and performed by trained personnel. Highly organized approach must be used.

Basilar Skull Fractures

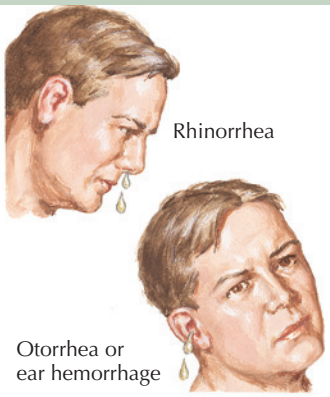


Longitudinal (A) and transverse (B) fractures of petrous pyramid of temporal bone and anterior basal skull fracture (C)

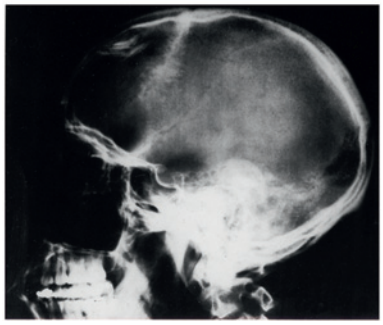
"Panda bear" or "raccoon" sign due to leakage of blood from anterior fossa into periorbital tissues. Note absence of conjunctival injection, an important differential from direct eye trauma.



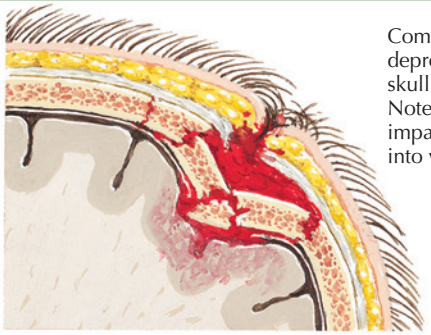
Battle's sign: postauricular hematoma



Compound Depressed Skull Fractures



Left lateral skull film showing left frontal depressed skull fracture.



Compound depressed skull fracture. Note hair impacted into wound.

Figure 39-5 Skull Fracture.

Early Evaluation of Head Injuries

- On field and sideline
- **A**irway, **b**reathing, **c**irculation, **d**isability, **e**xposure (ABCDE)
- Glasgow Coma Scale; evaluates best eye, verbal and motar response. Useful for predicting prognosis in severe head injury.
- **If athlete is conscious** and has no neck pain, and normal neurologic evaluation, remove to sideline for further evaluation and observation.
- **If athlete is unconscious** or confused or has neurologic abnormality, skull fracture, or neck pain, protect airway, immobilize cervical spine, and transfer to hospital for further evaluation (e.g., cervical spine films, other imaging).
- **Evaluation of associated injuries**
  - Unstable cervical spine injury should be assumed until proved otherwise; ask about neck pain.
  - Maintain high suspicion for vascular or focal injuries (change in level of consciousness, focal deficits, other neurologic symptoms).
  - Assess for skull fracture (see "Skull Fracture").
- **Thorough history at time of injury and focused neurologic exam** with attention to mental status and cognitive functioning.
  - Determine level of consciousness, and memory of event before (retrograde amnesia) and after (posttraumatic amnesia) injury.
  - Specifics of events (e.g., game score, special plays, teammates), events before game, previous game score. Teammates and coaching staff may be useful in validating information.
  - Neurologic assessment: early cognitive, somatic, and affective signs and symptoms (see Table 39-2).
  - Orientation: time, place, date.
  - Short-term memory, long-term memory, and concentration assessment.
    - Examples include asking the athlete about teammates, coach, specific plays, home phone numbers. What and who they ate with prior to the event and what classes they attend on specific days.
    - Confusion may be quickly assessed by using questions proposed by Maddocks and colleagues: "Where are we?, Which team are we playing?, Who is your opponent?, Which half is it?, How far into the quarter is it?, Which side scored last?, Which team did we play last week?, Did we win last week?"
    - Ask athlete to remember five items, count backward (digit spans, e.g., 1-4-2, 6-9-3-1 and so on up to six digits or more), name months in reverse, give number of dimes in a dollar, other simple tasks.
  - Assess upper and lower extremity motor and sensory function, deep tendon reflexes.
  - Assess for cranial nerve deficits.
  - Assess cerebellar function: finger to nose, heel to shin, Romberg test, tandem gait with eyes opened and closed.
  - Assess balance; may used computerized platform or clinical assessment. Modified Balance Error Scoring System (BESS) can be utilized.
- **History of prior concussions and risk factors**
  - Details of previous injuries, including alterations in level of consciousness, associated injuries, time lost from participation and date of most recent concussion.

- Pay particular attention to repeat concussions with lesser impact forces and/or increasing duration of symptoms.
- Assessment of other risk factors: intoxication, childhood and adolescence (to age 18), learning disability, use of anticoagulants, hemophilia, inadequate postinjury supervision.

- **Close observation and periodic reevaluation** are highly important. Signs and symptoms worsen with time.
- Athlete's helmet, if applicable, should be held to prevent reentry into game, and the coach should be made aware that the athlete is not available to participate.
- Athletes may need to be serially evaluated after injury and a plan should be arranged with the athlete, parent, and/or other teammates/roommates such that the athlete can be watched for signs of deterioration and transported to an appropriate facility if need be.
- If Neuropsychological testing (NPT) available, consider performing post-injury NPT at least 24 to 48 hours after injury (as long as athlete is asymptomatic).

Standardized Assessment Tools

- Developed to establish valid standardized, systematic sideline evaluation for immediate assessment of concussion in athletes.
- **Not meant to replace individual assessment** or more comprehensive formal clinical NPT; **do not diagnose concussion.**

Standardized Assessment of Concussion (SAC)

- Baseline testing recommended for comparison with postconcussion scores.
- If appropriately used in addition to individualized assessment, may provide good starting point for less experienced health professionals.
- Objectively assesses orientation, immediate memory, concentration, and delayed recall but does not assess more complex neurocognitive function.
- **Advantages:** Practical and portable with some validity takes about 5 minutes to administer; may be administered by athletic trainer; three equivalent forms minimize practice effects.
- **Disadvantages:** Potential for inappropriate use to diagnose concussion and make return-to-play decisions; sensitivity questioned by some as screening tool for concussion.

Sport Concussion Assessment Tool (SCAT and SCAT2)

- Standardized tool developed for use with both physician assessment of sports concussion and patient education; recommended for use by the Prague Second International Agreement Statement, and SCAT2 recommended by Zurich 3rd International Consensus Statement.
- Combined multiple existing assessment tools into standardized tool, including SAC.
- Similar uses, advantages, and disadvantages of SAC, but with additional memory questions, symptom diary, return-to-play guidelines, and patient education material.
- Takes longer to administer; there have been no validation studies, but SCAT2 is a better tool than SAC.
- SCAT2 similar to SCAT but adds Glascgow Coma Scale, SAC, and modified BESS. No validation studies.
- **Transfer for further evaluation** of athletes with:
  - Suspected cervical spine injury.
  - Deteriorating mental status, deteriorating or persistent neurologic deficits (e.g., focal signs) behave unusually or are very irritable, become lethargic and can't be woken up, have worsening headache, persistent nausea and vomiting.
  - Have difficulty with balance, weakness or numbness in arms or legs, and/or develop seizures.
  - High-risk condition (e.g., hemophilia, intoxication, anticoagulants).

Delayed Evaluation

- Serial sideline, postgame, training room or office.
- All head-injured athletes should be reassessed serially as needed. History is again essential. Family members often help validate history. Athletic trainer's notes and consultation may be useful.
- Same evaluation as on-field and initial sideline assessments.
- Consideration of repeat or additional neuropsychological testing.

INJURY SEVERITY AND MANAGEMENT OF CONCUSSION

- Numerous concussion grading systems and return-to-play (RTP) guidelines are available. **Many are empirical and lack scientific basis.**
- Early guidelines use loss of consciousness (LOC) as indicator of injury severity, which has subsequently been shown not to be true.
- Newer guidelines, such as those published by the National Athletic Trainers Association (NATA), the First International Conference on Concussion in Sport (Vienna Guidelines), as well as Team Physician Concussion Consensus Statement (TPCC; see Appendix A), and most recently, the Zurich 3rd International Consensus Conference Guidelines (in press), likely represent the most comprehensive.
- The Second International Conference on Concussion in Sport (Prague Group) categorized or graded concussion as **simple** or **complex** for management purposes:
  - **Simple:** Injury progressively resolves without complication over 7 to 10 days; treatment involves rest until all signs and symptoms resolve, followed by graded exertion progression.
  - **Complex:** Injury associated with persistent signs and symptoms (includes exertional), specific sequelae (e.g., concussive convulsions, prolonged LOC (>1 minute), or prolonged cognitive impairment. Includes athletes suffering multiple concussions and/or repeated concussions occurring with less impact force. Additional individualized management considerations, including consideration of NPT, replaces simpler RTP decisions made with simple concussion.
- There are significant limitations in using the system discussed because NPT is recommended for complex concussions, with baseline data important, yet it cannot be determined if the concussion is simple or complex until after symptoms have resolved. Third Zurich International Consensus Statement on Concussion in Sport abandoned the use of "simple" and "complex" concussion, and made revisions regarding RTP as discussed previously.
- According to the Team TPCC guidelines both encourage determining severity of concussion only after all symptoms resolve and cognitive and neurologic exams normalize. Emphasis is appropriately placed on individualized assessment, not on a rigid timeline, when making RTP decisions.
- Severity; based on nature, burden, and duration of symptoms.
- Zurich 3rd International Consensus Statement. Substantiate the approach to RTP made by the Vienna Guidelines, NATA, and TPCC Statements. Individualized RTP with progressive step-wise exercise program as advocated above. Zurich guidelines allow for same-day RTP and speeded progression but only in select situations and only in adult athlete.

Return-to-Play Guidelines

- Vienna and Prague Guidelines do not recommend same day RTP. NATA TPCC Guidelines allow consideration of same day RTP as long as symptoms do not persist for more than 15 to 20 minutes, no LOC occurred, and/or no significant memory dysfunction occurred. Zurich Guidelines state that

same-day RTP can be considered in the adult athlete when symptoms are limited, resolve quickly, and a comprehensive evaluation by an individual with knowledge regarding sports concussion and one that includes normal cognitive and balance testing is performed, with no additional modifiers present. All agree that **symptomatic athletes should never be allowed to return to play.**

- Areas of controversy for *same day* RTP decisions include:
  - Returning athlete with fully resolved symptoms.
  - Returning athlete with certain specific symptoms, such as LOC, PTA, or RGA.
  - Duration and severity of symptoms are primary determining factors for RTP.
- Conservative considerations with emphasis on a period of prolonged asymptomatic rest should be considered in athletes with history of repeat concussions; in **young athletes;** and in athletes with a history of prolonged signs or symptoms following concussion, current or previous injury associated with disproportionately lesser force, and other risk factors. May be able to speed progression and allow same-day RTP in "elite" athlete, when defining "elite" as those athletes with certified athletic trainers and/or team physicians on site, specialized programs for concussion including a sideline assessment, additional testing such as neuropsychological testing and balance testing, and accessibility to additional expertise. One exception to this definition of elite is the youth athlete, who should always be treated more conservatively.
- If athlete allowed to return to play on same day, perform exertional challenge: e.g., 40-yard sprint, sit-ups, push-ups, knee bends. If athlete becomes symptomatic, hold from play.
- If delayed RTP, formal NPT and balance testing should be considered before RTP if available (see later discussion). If unavailable, serial evaluations with individualized progression of activity and conservative advancement important.
- **Timing** and **speed** of progression is controversial. Consider symptom-free interval prior to initiating and advancing progression if individual risk factors exist. Most important modifying factors include age, prior history of concussion and temporal relationship, pre-existing headache or learning disorder, or other medical comorbidities. Progression should occur at slower rate for young athletes than in adult. Zurich Guidelines allow for same-day RTP and more rapid progression in selected situations, consistent with the NATA and TPCC Guidelines.
- No long-term data assess return-to-play decisions.

Additional Further Diagnostic Testing

- Computed tomography (CT) scan should be the first tool if concerned about intracranial bleed or fracture; better at detecting blood, fracture; as good as magnetic resonance imaging (MRI) at identifying surgical lesions.
- Electroencephalogram (EEG), MRI, and CT scan may be normal despite significant clinical symptoms and abnormalities in cognitive function.
- Positron emission tomography (PET), diffusion tensor imaging (DTI), and functional MRI scans may play role; correlate with pathophysiologic data. Clinical applicability not practical in most settings at this time; these are mainly research tools.
- Neuropsychological testing (see later discussion).
- Laboratory evaluation of athletes with significant head injury should be individualized. Consider complete blood count, electrolytes, serum glucose, urinalysis, coagulation studies, toxicologic and ethanol screens, and blood type and crossmatch.
- Cervical spine x-rays should be considered in all athletes with significant head injury. Skull films may help localize and determine severity of depression of underlying skull fractures.

NEUROPSYCHOLOGICAL TESTING (NPT)

- **NPT provides reliable assessment and quantification of brain function** by examining brain–behavior relationships. Neuropsychologists likely best trained to interpret NPT results.
- Tests measure broad range of cognitive function: speed of information processing, memory, attention and concentration, reaction time, scanning and visual tracking ability, and problem-solving abilities.
- Preliminary studies using paper and pencil testing used in athletics demonstrate NPT as a useful tool in assessment of concussion.
- Newer, more portable, computerized test batteries are available: Immediate Postconcussion Assessment and Cognitive testing (ImPACT), CogStateCogSport, Automated Neuropsychological Assessment Metrics (ANAM), Headminder. These computerized tests are shorter and easier to perform than standard paper and pencil tests. Controversy surrounds optimal protocol of tests and when to use.
- Several studies using computerized NPT have shown the "value added" of NPT in addition to symptoms in demonstrating cognitive deficits in athletes after concussion.
- Recent studies have questioned the sensitivity and specificity of various computerized formats.
- Remains "one piece of the puzzle" when managing concussion.
- May be useful in assessment and recovery phases of head injury. Most clinicians advocate use after acute injury and symptoms have resolved, compared with preinjury baseline assessment.
- NPT is currently used in the National Football League, the National Hockey League, and U.S. Soccer and U.S. Lacrosse, as well as at various college and high school programs in the assessment of concussion.
- NPT may detect acute and chronic head injury; it is more sensitive in assessing cognitive function than classic medical testing (neurologic exams with MRI, CT, EEG).
- NPT provides additional useful information in assessment of concussion and may supplement, but not replace, comprehensive individualized assessment. NPT is "only one tool in the toolbox."
- NPT may become more widely available for sideline use in the future, especially with development of more sophisticated programs for laptop and hand-held computers.

Take Home Messages for Concussion

- Important to individualize treatment.
- Any athlete suspected of having a concussion or other head injury should be removed from play and evaluated by a health care provider.
- No athlete should be allowed to participate with symptoms
- Any athlete with worsening symptoms, altered mental status, or other symptoms of intracranial bleeding should be transferred immediately to an emergency facility. Athletes with suspected cervical spine injury should be immobilized and transported immediately to an emergency facility.
- Severity of injury is more closely related to the burden and duration of symptoms, presence of amnesia, and prolonged confusion.
- Children and adolescents must be treated with more caution than adults. Available research demonstrates that it takes longer for the younger athlete to recover than their older counterparts.
- Once the athlete is asymptomatic, an individualized, gradual, step-wise return to play progression can be initiated.

- Treat each injury individually based on several factors including burden and duration of symptoms, age of athlete, prior history of concussions and sport, mismatch between force of impact, and subsequent injury (see Appendix C).
- **No athlete who is symptomatic (at rest or with exertion) after head injury should be allowed to return to sport until symptoms have resolved completely.**
- "If in doubt, sit them out."

HEAD INJURY COMPLICATIONS

Second-Impact Syndrome (SIS)

- Rapid brain swelling and herniation after second head injury in athlete still recovering from initial head injury (during period of injury-induced vulnerability).
- Vascular congestion, increased ICP, and brain (uncal) and brainstem herniation probably result from loss of autoregulation of cerebral vasculature.
- Second impact may be mild (e.g., blow to chest or back that "snaps" head). Athlete may initially appear dazed. Precipitous collapse, rapidly dilating pupils, coma, and respiratory failure ensue in seconds to minutes; end result is often death.

Postconcussive Syndrome

- Characterized by persistent concussive symptoms for extended period (often weeks to months).
- May be related to altered neurotransmitter function; severity and duration of symptoms may correlate with duration of posttraumatic amnesia.
- Link to depression in former professional football players with history of concussion.
- MRI or CT and, if available, NPT should be considered for athletes experiencing concussive symptoms for more than 1 to 2 weeks.
- Treatment involves multidisciplinary approach that may include psychotherapy, physical therapy, biofeedback, and medication (e.g., antidepressant).

Posttraumatic Seizure

- Three types of seizures may follow head injury: immediate, early, and late.
  - **Immediate posttraumatic seizures (concussive convulsions)** associated with no underlying structural or permanent brain injury; occur seconds after impact and involve brief tonic phase, followed by bilateral myoclonic jerking. Seizures cease spontaneously and are followed by concussive symptoms; usually do not require anticonvulsant therapy. Recurrence uncommon.
  - **Early** (<1 week) or **late** (>1 week) **posttraumatic seizures** may be partial or generalized. Many involve temporal lobe; most are associated with underlying brain pathology (e.g., contusion, hemorrhage, skull fracture) and require long-term anticonvulsants. Seizures may recur in 20% to 25% of early and in up to 70% of late cases of posttraumatic epilepsy.
- **Differential diagnosis** for posttraumatic seizures includes idiopathic generalized epilepsy (poorly controlled or new onset), focal (partial) epilepsy associated with preexisting brain lesion or seizure focus, secondary epilepsy (e.g., drug-induced), convulsive syncope, and posttraumatic seizures.
- **Risk factors** for chronic posttraumatic seizures include depressed skull fracture, dural penetration, posttraumatic amnesia that lasts more than 24 hours after injury, acute intracranial hemorrhage, and early or late posttraumatic epilepsy.

Chronic Traumatic Brain Injury (CTBI)

- CTBI (also called chronic traumatic encephalopathy and dementia pugilistica) represents chronic and cumulative neurologic dysfunction after repetitive head trauma. Observed most often in boxers. More recent reports in professional wrestlers and American football players. Area of significant ongoing research.
- Characterized by central nervous system dysfunction that may include cognitive impairment, ataxia, behavioral changes, parkinsonism, and pyramidal tract dysfunction.
- Apoprotein E ϵ4 allele has been noted to be a risk factor for CTBI in boxers.
- Varying degree and duration of neuropsychological function have been observed after head injuries (sport-related and non–sport-related trauma).
- Recurrent head injury, cognitive dysfunction, and learning disability may be risk factors for reduced cognitive performance after head injury.
- Potential link to Alzheimer's disease, depression, and suicide recently reported.

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Neck Injuries

40

R. Lance Snyder

INTRODUCTION

Cervical spine injuries are most often seen in football and hockey but have occurred in wrestling, rugby, baseball, lacrosse, and mountain biking.

Anatomy

- There are seven cervical vertebrae and eight cervical nerves.
- Spinal nerves exit above the vertebral body for which they are named; for example, the sixth cervical nerve exits at the C5-6 disc space (Fig. 40-1).
- The cervical spine is divided into upper and lower segments: The upper segment includes C1 (the atlas) and C2 (the axis).
  - The spinal cord occupies little space because the canal is funnel shaped.
  - The atlas (C1) and the occiput account for 40% of cervical flexion.
  - The axis (C2) has a fingerlike projection, the dens, about which the atlas rotates. This accounts for 60% of cervical rotation.
  - The key for C1 and C2 stability is the transverse atlantal ligament, which lies posterior to the body of C2 and connects C1 to C2. Disruption of this ligament can cause atlanto-axial instability (see Fig. 40-1).
- The lower segment of the cervical spine includes C3 through T1.
  - Bony structure is relatively constant with anterior column support provided for by the anterior longitudinal ligament, the vertebral bodies, and the discs.
  - Posterior column is supported by the posterior longitudinal ligament, facet articulation, facet capsule, intraspinous ligament, and supraspinous ligament.
  - The spinal cord occupies 75% of the space of the canal at this level.
  - Space available for the spinal cord (SAC) typically is between 14 and 23 mm. The cord is kenotic when there is less than 13 mm available. Cord compression is typically present when there is less than 10 mm available.

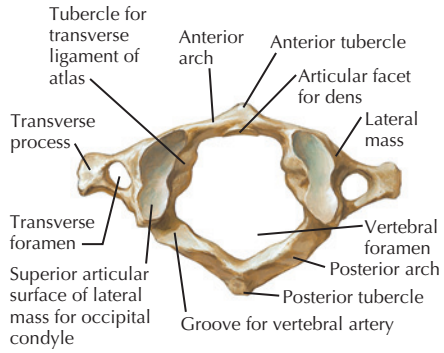
History and Physical Exam

History

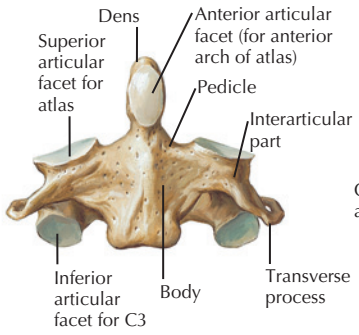
- Before moving the player, the physician must first ask if the player is hurt and if the player has full sensation and movement.
- Physician should ask about (1) direction of a player's helmet at the time of injury and (2) radiation of symptoms and their resolution.

Physical Exam

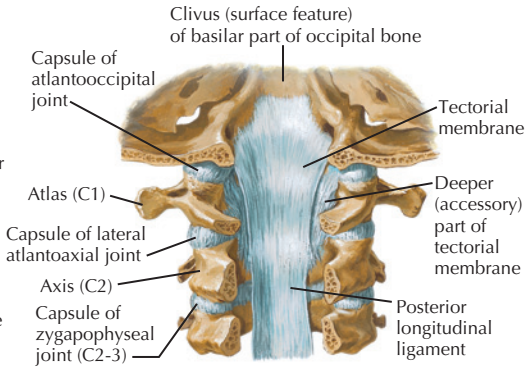
- Player must first be removed from the field in a safe and protected manner.



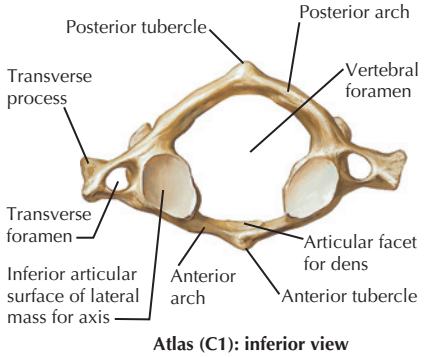
Atlas (C1): superior view



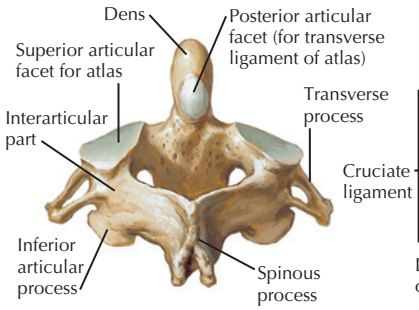
Axis (C2): anterior view



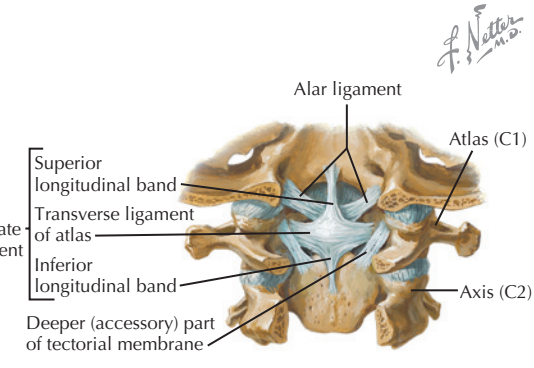
Upper part of vertebral canal with spinous processes and parts of vertebral arches removed to expose ligaments on posterior vertebral bodies: posterior view



Atlas (C1): inferior view



Axis (C2): posterosuperior view



Principal part of tectorial membrane removed to expose deeper ligament: posterior view

Figure 40-1 Anatomy of a Vertebra.

- Six persons, who have practiced the maneuver prior to injury, must logroll the athlete onto a spine board and transport the athlete safely to the sidelines.
- Helmet should not be removed but the airway must be protected and maintained.
- The patient with only pain is examined with palpation.
- A fracture of the cervical spine may present with minimal pain and the physician and medical staff should be aware of this fact.
- Athletes with suspected injuries would be immobilized.
- If only radicular symptoms are present the athlete should be examined for range of motion of the cervical spine, followed by a brief neurologic exam.
- This exam is by no means complete and a thorough neurologic exam and work-up must be carried out at a later time.
- Strength is tested by comparing both sides on a scale of 0 to 5 (with 5/5 being full strength). The deltoids are checked with corresponding C5 nerve root.
- The biceps are checked with corresponding C6 nerve root and then the triceps are checked with the corresponding C7 nerve root.
- Sensation is then checked with the thumb dorsally corresponding to C6, the long finger involving C7, and the little finger C8. Reflexes are then checked at the elbow (C6) and the triceps (C7).
- Spurling's test may then be performed by extending the player's neck and rotating the head toward the affected side.
- A positive test causes radiating pain and can be caused by soft disc herniations or osteophytes combined with a hard disc protrusion.

STINGERS OR BURNER

Description: This is a stretch injury of the nerve root.

Mechanism of injury: There is typically hyperextension of the player's neck with ipsilateral deviation of the player's head.

Presentation: There is typically a stinging or burning pain into the shoulder, arm, or hand. There may be numbness and tingling. There may also be weakness in the affected upper extremity. Symptoms typically last seconds to minutes but can last days to weeks. There is a 65% incidence in college football players with an 87% chance of recurrence.

Physical exam: Test for muscular weakness. The deltoid corresponds to C4-5, the biceps to C5-6, and the triceps to C6-7. Test for sensory loss over the biceps (C5), thumb (C6), and long finger (C7). Test for reflexes at biceps, triceps, and the brachioradialis. Check for focal tenderness in the neck. Perform a range of motion exam on the neck. Check for a positive Spurling's sign (hyperextension of the neck with rotation toward the side of injury causes pain).

Differential diagnosis: Cervical fracture, cervical herniation.

Diagnostics: Cervical x-rays in flexion and extension should be taken to rule out fractures, and instability should be taken if pain and weakness persist. The T1 vertebral body must be seen to attain an adequate exam. Magnetic resonance imaging (MRI) may be necessary as well if symptoms persist.

Treatment: The player is rested until full strength and sensation has returned. Pad modification that prevents lateral deviation of the cervical spine may be of benefit. Positional change (e.g., changing a right guard to a left guard) may be of benefit as well.

Prognosis and return to play: Athletes are allowed to return to play when they have a normal neurologic exam and full cervical range of motion.

TRANSIENT QUADRIPARESIS

Description: Pathologic insult to the spinal cord, which or may not be accompanied by a transient hypoxemia in the cord.

Mechanism of injury: With flexion the spinal cord is pinched between the superior spinolaminar line and the superior aspect of the posterior lower vertebral body.

Presentation: The player may experience a sudden period of paralysis after making a tackle or being struck. The paralysis may be in all four limbs or limited to the upper body. The weakness typically is short-lived, lasting only minutes, but can last hours to days. Sensation changes may be present as well.

Physical exam: The airway must be cleared of any obstruction if there is loss of consciousness and adequate ventilatory support must be obtained. A gross neurologic exam must be performed. The physician must check for upper motor neuron lesions as well as lower motor neuron lesions. Reflexes of the upper and lower extremity must be tested. The strength and sensation in the upper and lower extremity also must be checked.

Differential diagnosis: Cervical herniations, fracture, or fracture/dislocation.

Diagnostics: Radiographs can be used to determine the Torg ratio. This ratio compares the space from the back of the vertebral body to the spinolaminar line with the width of the vertebral body. The Torg ratio should be greater than 0.8. A number less than this indicates spinal stenosis and is sensitive for the risk of recurrence. An anterior to posterior radiograph as well as a lateral film should be obtained. Flexion or extension films should not be performed if cervical tenderness exists. A computed tomography (CT) scan can help show the bony structures. A myelogram can be added to show the space available to the spinal cord. An MRI can help demonstrate any pathologic changes to the soft tissues.

Treatment: The player should be observed and frequent neurologic exams should be performed. The diagnostic studies should be performed. There is no clear-cut answer as to whether the steroid protocol should be administered. This protocol, when started with 8 hours after neurologic damage, has been shown to improve neurologic function. The dosage of methylprednisolone is 30 mL/kg bolus over 15 minutes and then 15 mL/kg over 23 hours.

Prognosis and return to play: Return to play is highly controversial. Many doctors believe that if there is no stenosis and no evidence of structural damage then a player should be allowed to return to play. The player must have full strength and sensation before returning to play.

CERVICAL DISC HERNIATIONS

Description: Disc material protrudes through a tear in the annulus, causing root and, rarely, cord impingement (Fig. 40-2).

Mechanism of injury: Compression and rotation of the disc causes the annulus to tear and the nucleus to retropulse against the nerve root or spinal cord.

Presentation: A nerve's root exits in the neuroforamen at the level above its vertebral body; therefore, a herniation at C5-6, for example, will typically affect the sixth cervical nerve root. The athlete may experience radiculitis (radiating pain) or weakness that radiates (radiculopathy) into the extremities.

Physical exam: Spurling's sign may be present, which is lancinating pain into the upper extremity when the athlete's head is extended and rotated toward the affected side. The athlete must be examined for sensory loss in a dermatomal pattern and checked for muscular weakness correlating to a corresponding nerve root (Table 40-1).

Diagnostics: MRI is typically diagnostic and will indicate the presence of a hard or soft disc.

Treatment: Oral steroids or epidural injections are often beneficial. Anterior cervical discectomy and fusion or a foraminotomy may be needed to decompress the nerve root.

Prognosis and return to play: The athlete may be allowed to return to play when he or she has regained full function without signs of neurologic damage.

SPEAR TACKLER SPINE

Description: When an athlete experiences one or more episodes of cervical neurapraxia. Often seen in football players with a propensity to hit or tackle with the crown of the head. The Na-

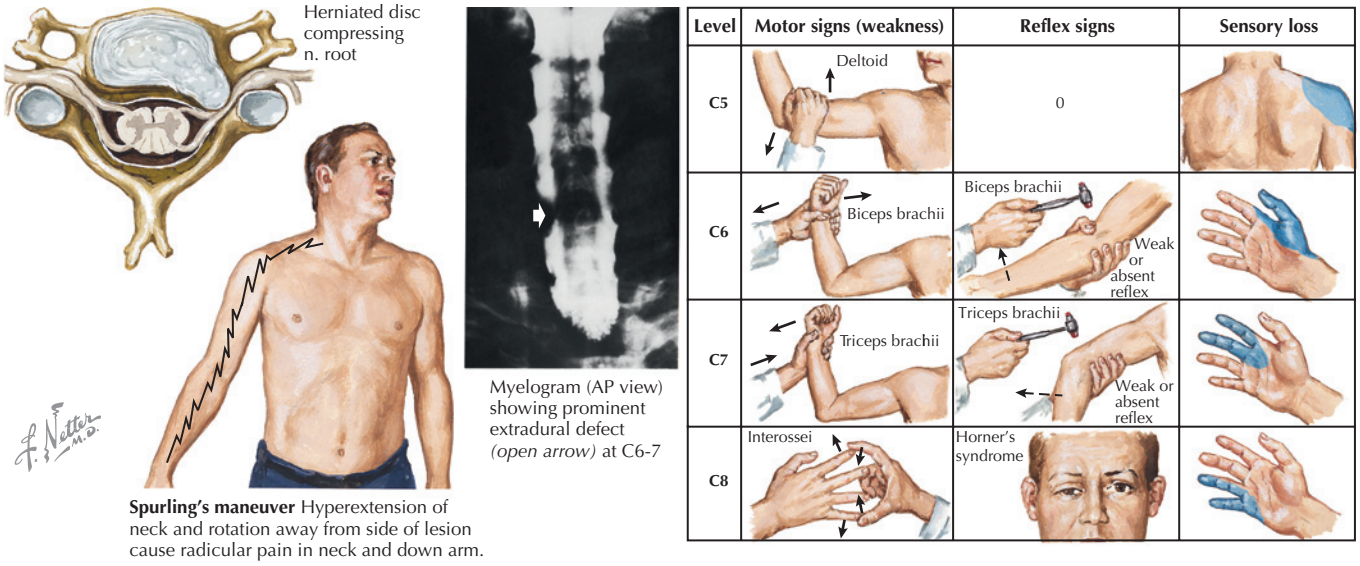


Figure 40-2 Cervical Disc Herniation: Clinical Manifestations.

Table 40-1 SENSORY LOSS TEST

Nerve root	Sensation	Motor	Reflex
C5	Outer arm	deltoid	n/a
C6	Thumb and index finger	biceps	biceps
C7	Long finger	triceps	triceps
C8	Little finger	finger extensor	n/a

tional Collegiate Athletic Association (NCAA) has banned the use of hitting with the crown of the head. After this technique was banned there was a drop in the rate of catastrophic cervical injuries.

Diagnostics: X-rays may show cervical stenosis with a positive Torg ratio (<0.8). There is usually a loss of cervical lordosis and some cervical kyphosis may be present. Torg described that the player may have degenerative radiographic abnormalities as well. Video analysis will document that the player is leading with his head and therefore spear-tackling.

Prognosis and return to play: This player should not be allowed to return to athletic activity that involves contact.

FRACTURES

C1 Fractures (Jefferson Fracture)

Description: Traumatic burst fracture of C1 (Fig. 40-3).

Mechanism of injury: Axial load.

Presentation: The athlete will likely present with neck pain. This injury is likely to cause a neurologic injury because of the wide mount of space available for the spinal cord at this area.

Physical exam: Palpate for any tenderness. Check for lack of range of motion.

Differential diagnosis: Cervical strain.

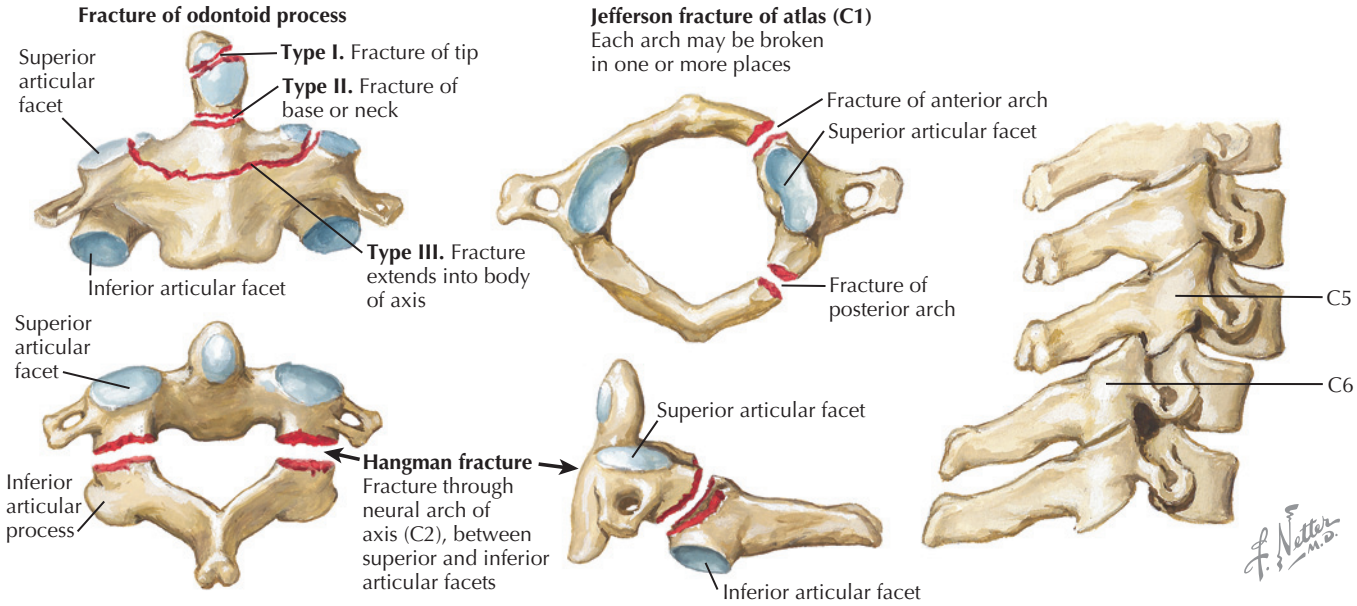
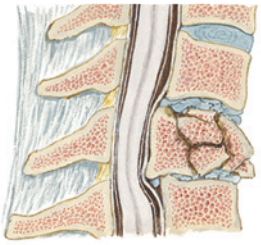


Figure 40-3 Fracture and Dislocation of Cervical Vertebrae

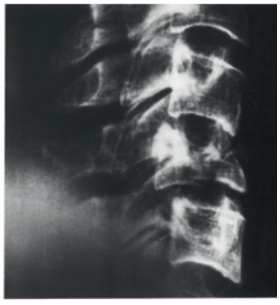
Diagnostics: Plain films and CT scan should be diagnostic.
Treatment: This is an unstable injury and referral to an orthopedic spine surgeon or neurosurgeon should be performed.
Prognosis and return to play: This player will likely not be allowed to return to play.

C2 Fractures (Hangman's Fractures)

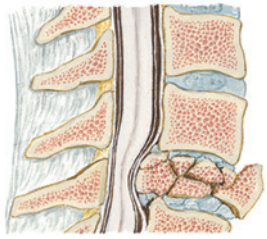
Description: Traumatic spondylolisthesis of C2.
Mechanism of injury: Axial load and extension.
Presentation: The athlete will experience pain and may experience a sense of instability.
Physical exam: Palpate the neck area. Check for range of motion. This injury does not typically result in paralysis or death because this injury actually widens the amount of area available to the spinal cord.
Differential diagnosis: Cervical strain.
Diagnostics: Lateral radiograph can be diagnostic if there is displacement in the fracture. CT scans can further define the injury.



Type III. Fracture through entire vertebral body with fragmentation of its anterior portion. Posterior cortex intact but projects into spinal canal causing damage to cord and/or nerve roots.

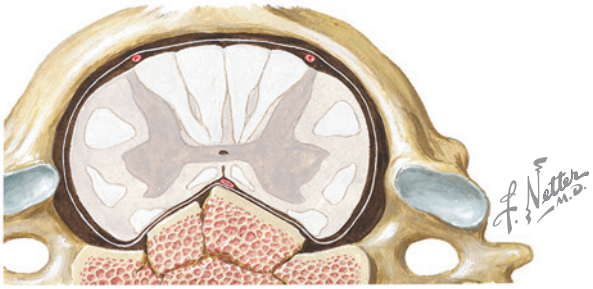


X-ray film: Type III fracture of C5



Type IV. "Burst" fracture. Entire vertebral body crushed, with intraspinal bone fragments.

X-ray film: Type IV fracture of C6



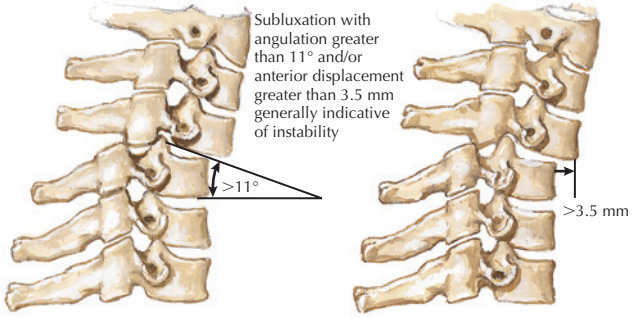
Dislocated bone fragments compressing spinal cord and anterior spinal artery. Blood supply to anterior two thirds of spinal cord is impaired.

Figure 40-4 Compression Fractures of Cervical Spine.

Treatment: The head should be immobilized until referral to a spine specialist.
Prognosis and return to play: Poor.

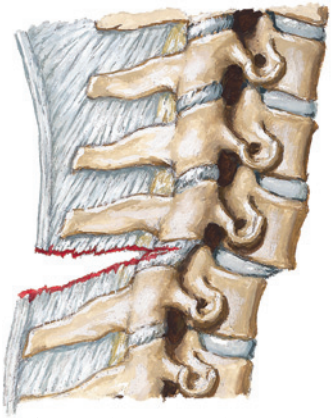
Burst Fractures

Description: This injury involves a fracture of the vertebral body in the coronal and saggital plane. There can be retropulsion of fragments, which often results in spinal cord damage (Fig. 40-4).
Mechanism of injury: When a pure axial load is applied to a straightened cervical spine, failure can result because the surrounding soft tissues cannot dissipate the force.
Presentation: The athlete commonly complains of neck pain, which may be the only symptom. There may be a root lesion or incomplete or complete paralysis.



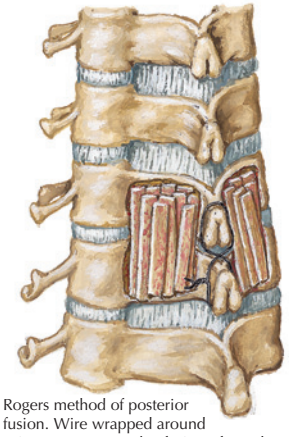
Subluxation with angulation greater than 11°

Anterior displacement greater than 3.5 mm

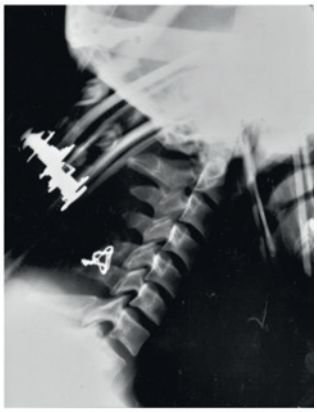


Tear of interspinal and supraspinal ligaments characteristic of anterior dislocation of spine.

Lateral radiograph shows severe kyphotic angulation in cervical dislocation.

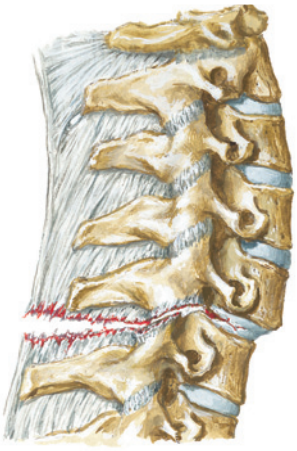


Rogers method of posterior fusion. Wire wrapped around spinous processes plus fusion of vertebrae.

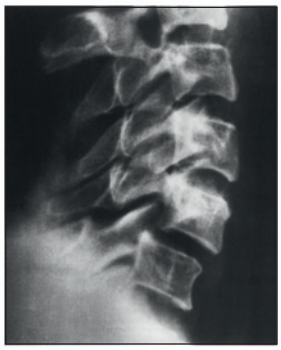


Postoperative radiograph shows corrected alignment and fixation wire in place.

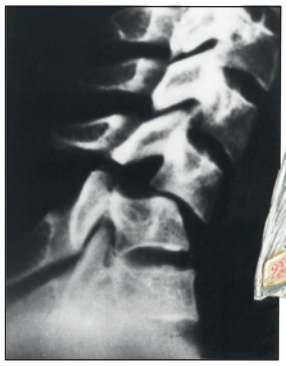
Figure 40-5 Subluxation and Ligamentous Instability of Cervical Spine.



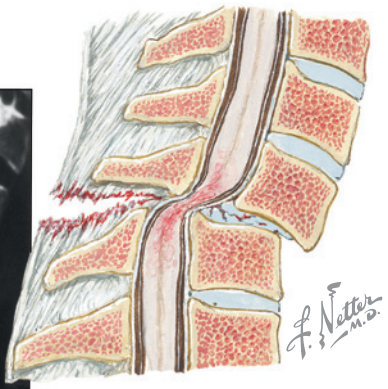
Anterior dislocation of C5 on C6 with tear of interspinous ligament, facet capsules and posterior fibers of intervertebral disc



X-ray film showing moderate (1st-degree) dislocation of C5. If there is no evidence of spinal cord injury, spontaneous healing may occur following reduction by traction and prolonged bracing.



X-ray film: 3rd-degree dislocation of C5



Severe spinal cord injury in 3rd-degree cervical dislocation. Any evidence of cord or nerve root damage is indication for prompt:
- **Reduction** by traction
- **Decompression** by disc removal
- **Fixation** by interbody fusion

Figure 40-6 Dislocations of Cervical Spine.

Physical exam: Pain in the neck region. Often there is loss of motion in the cervical spine. There may be sensory or muscular strength loss.

Differential diagnosis: Cervical strain.

Diagnostics: X-rays, CT scans, and MRI test should be performed. A flexion and extension series should not be performed when there is an obvious fracture. The physician must be aware of the seemingly mild anterior teardrop fracture, which may indicate an unstable sagittal split in the anterior and posterior column of the spine. Radiographs must include from C1 to T1 on the lateral films. An indication of fracture is loss of height (3 mm or more) of a vertebral body when compared with another vertebral body. Angulation of greater than 11 degrees between adjacent vertebral bodies is also a mark of instability. The measurement must be made between the vertebral bodies, not just between the vertebral body and the inferior vertebral body line (Fig. 40-5).

Treatment: The ABCs of trauma management must be applied. The sports medicine team members must be practiced in turning a patient into the prone position and transporting the patient onto a spine board. This often requires at least six individuals because athletes can be quite large. The helmet should not be removed on the field, but the facemask should be removed to gain access to the airway while the cervical spine is stabilized. The medical professional must be familiar with the headgear the athlete is wearing and have all the necessary tools, nonelectric and electric, to remove the facemask. After transport to the emergency department, appropriate steps can be taken for further equipment removal.

DISLOCATIONS

Description: An injury in which one or both of the facets are dislocated. An associated facet fracture may be present (Fig. 40-6).

Mechanism of injury: A flexion distraction injury.

Presentation: Pain or tenderness in the cervical spine. Pain with range of motion. Muscular strength or sensation loss. There may be associated neurologic injury, which can include a nerve root injury or a complete lesion.

Differential diagnosis: Cervical strain, herniations.

Diagnostics: Radiographs will usually show greater than 25% displacement on the posterior vertebral body line from the adjacent posterior vertebral body line, for a unilateral facet dislocation. There may be greater than 50% displacement for a bilateral facet dislocation. Greater than 3.5 mm of displacement of the posterior vertebral line of one vertebra compared to the other vertebral body denotes instability. CT scans should be performed as well, which will pick up fractures of the facets and give an overall indication of the force and treatment pattern. An MRI will help pick up the presence of myelomalacia or cord damage as well as the presence of a herniated disc. A herniated disc may be present in up to 55% of cases with bilateral facet dislocation.

Treatment: On the field, the head and neck are immobilized until the athlete is safely transported to a hospital. An orthopedic spine surgeon or a neurosurgeon should then be consulted. A reduction is typically performed with weight applied to Gardner-Wells tongs. This procedure is usually performed with the patient awake so as to be aware of any worsening neurologic injury. The herniated fragment can retropulse into the spinal canal and cause paralysis. Some physicians have made the case for prereduction MRI, to look for a herniated disc and lessen the likelihood of retropulsion.

RECOMMENDED READINGS

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Eye Injuries in Sports

41

David E. Olson, Robby S. Sikka, Thomas Pulling, and Michael Broton

INTRODUCTION

- More than 42,000 sports and recreation-related eye injuries were reported in 2000. Seventy-two percent occurred in individuals younger than 25 years; 43% in people younger than 15.
- About 1.5% of all sports injuries involve the eye or ocular adnexa; these injuries have a high morbidity rate.
- In the United States, basketball is the leading cause of sports-related eye injuries; in Europe and South America, soccer is the leading cause.
- Although eye protectors cannot eliminate the risk of injury, appropriate and well-fitted eye protection can reduce the risk of significant eye injury by as much as 90%.
- The American Association of Pediatrics and American Academy of Ophthalmology 2004 position statement on protective eyewear in young athletes categorizes sports by the risk of eye injury to the unprotected eye (Box 41-1). Though there is no ideal collecting system for data, the National Collegiate Athletic Association (NCAA) Injury Surveillance System (ISS) tracks injuries in college sports (Table 41-1).

MECHANISMS OF INJURY FOR EYE INJURIES IN SPORTS

- The severity of eye injuries can be positively correlated with the total impact force, the rate of force onset, and the kinetic energy of an impacting object.
- Ocular injuries fall into several broad categories (Table 41-2).
- Open globe injuries are full-thickness wounds to the eye wall (cornea or sclera) and result from rupture or laceration. Sports that cause ruptured globes typically have a stick or projectile that fits into the orbit. Previous surgery or eye disease increases the risk of open globe injury.
- Lacerations may be caused by objects that "slice" or penetrate the eye, which may lead to open globe injuries.
- Closed globe injuries are those that do not completely penetrate the cornea or sclera. These include lamellar lacerations, corneal abrasions, contusions, hyphema, or injury to the choroid, macula, retina, or optic nerve.

BOX 41-1 *Risk Categories for Sports*

High risk	**Moderate risk**
Small, fast projectiles	Fishing
Air rifle/BB gun	Football
Paintball	Soccer/volleyball
Hard projectiles, fingers, "sticks," close contact	**Low risk**
Baseball/softball/cricket	Bicycling
Basketball	Noncontact martial arts
Fencing	Skiing
Field hockey	**Eye safe**
Ice hockey	Gymnastics
Lacrosse, men's and women's	Track and field
Squash/racquetball	
Street hockey	
Intentional injury	
Boxing	
Full-contact martial arts	

Adapted from Vinger PF: A practical guide for sports eye protection. Phys Sports Med 28(6), 2000. Committee on Sports Medicine and Fitness: Protective eyewear for young athletes. Pediatrics 113:619-622, 2004.

Table 41-1 RELATIVE RISK OF EYE INJURIES AS REPORTED IN NCAA ISS

	Annual risk of eye injury	
	Men (%)	**Women (%)**
Wrestling	1.67	
Basketball	0.97	
Field hockey		0.88
Basketball		0.50
Softball		0.50
Soccer	0.26	0.24
Baseball	0.20	
Volleyball		0.12
Football	0.11	
Ice hockey	0.08	0.00
Lacrosse	0.06	
Gymnastics	0.00	0.00

Data from Dick R, Agel J, Marshall SW: National Collegiate Athletic Association Injury Surveillance System. J Athl Train 42(2), 2007.

Table 41-2 RELATIVE FREQUENCY OF EYE INJURIES

Most common	**Relatively infrequent**	**Eye emergencies**
Corneal abrasion	Chemical burns	Corneal laceration
Corneal foreign body	Vitreous hemorrhage	Retinal detachment
Conjunctival foreign body	Retinal hemorrhage	Lens dislocation
Subconjunctival hemorrhage	Retinal edema	Blowout fracture of the orbit
Eyelid laceration	Hyphema	Optic nerve injury
	Injury to the lacrimal system	

- Blunt injuries, typically causing contusions, globe rupture or adnexal injury, account for most sports-related eye injuries. Contusions are usually caused by blunt objects smaller than the orbit (e.g., golf ball or finger). Additionally, many objects will deform significantly on impact (e.g., soccer ball), producing a "knuckle" that will impact the eye (Fig. 41-1).
- Radiant energy injuries are less common yet, may occur in those activities taking place at a high altitude or on snow.

PRINCIPLES OF PROTECTION FROM SPORTS EYE INJURIES

- Protective devices work by deflecting the impact energy away from the eye and dissipating the energy over time and area. This is typically done with either a lens or mechanical grid (e.g., wire-framed face guard or mesh fencing helmet).
- Improper fit of protective gear can decrease the protection offered, placing the eye at an increased risk.
- Gear must be comfortable and not interfere with performance of athlete.
- Contact lenses offer no protection. Athletes that wear contact lenses should wear one of these three options:
  - Contact lenses plus the appropriate protective eyewear.
  - Polycarbonate lenses in sports frames that pass the appropriate ASTM standard.

Figure 41-1 Large Object Causing Injury to the Globe.

- Over-the-glasses protector that conforms to the appropriate ASTM standard.

CERTIFICATION AND SELECTION OF EYEWEAR

- Several organizations certify sports protective eyewear. These include the Protective Eyewear Certification Council (PECC), Canadian Standards Association (CSA), Hockey Equipment Certification Council (HECC), and National Operating Committee on Standards in Athletic Equipment (NOCSAE). Equipment approved by these organizations commonly bears their seal and should be selected when available (Table 41-3).
- ASTM International has written performance standards, based on design and strength, upon which many of these organizations base certification (see Table 41-3).
- For sports in which there are no appropriate ASTM standards or certified equipment, ANSI (American National Standards Institute) should be considered.

VISUAL RISK FACTORS

- Best corrected visual acuity less than 20/40 in either eye or spectacle correction for myopia or hyperopia greater than six diopters; disease, degeneration, or structural weakness of the eye itself; thin sclera; history of retinal degenerative disease; history of eye surgery that weakens outer wall of eye, especially cataract or refractive surgery. Athletes with such risk factors should be evaluated by an ophthalmologist before engaging in high-risk or extremely high-risk sports.
- Disability from high corrective spectacle lenses can sometimes be mitigated by contact lenses; however, contact lenses themselves can be risk factor.
- Functionally one-eyed athletes face additional risk. A person is functionally one-eyed when loss of the better eye would result in a significant change in lifestyle owing to poor vision in the remaining eye.
  - For the child, vision worse than 20/40 should be considered functionally one-eyed. The assessment of an adult is more difficult because their judgment and values determine the visual impairment they are willing to accept. Special considerations are necessary for these athletes.
  - The only sports absolutely contraindicated for the functionally one-eyed athlete are boxing and full contact martial arts, because the risks of serious injury are high and there is no known effective eye protector. Wrestling and the noncontact martial arts have a lower incidence of eye injury, but also do not have effective eye protection. They should be discouraged for the functionally one-eyed and banned for the monocular athlete.

EXAMINATION AND FUNCTIONAL TESTING AFTER INJURY

History

- Mechanism of injury is important. Historical features such as type of trauma (blunt versus penetrating), the direction of force, size of the object, and whether eye protection was worn influence the type of injury.
- Relevant signs and symptoms include decreased visual acuity, diplopia, flashers, floaters, and halos around lights.
- Historical features such as previous anisocoria, extreme myopia, past ocular surgery, or family history of Marfan syndrome are useful.

Physical Exam

- Visual acuity is the single most important physical exam feature in evaluation of the eye. Because changes in visual acuity are more important than absolute values, acuity should be routinely assessed prior to injury and the results available to the team physician. Any acute decrease in acuity necessitates immediate further evaluation and referral (Box 41-2).

Table 41-3 STANDARDS AND CERTIFYING ORGANIZATIONS FOR SELECTED SPORTS

Sport	Eye protection	Standards	Certifying organizations
Baseball	Polycarbonate or wire face guard attached to a helmet while batting. Sports goggles with polycarbonate or TriVex lenses while on the field.	ASTM F910	PECC
Basketball	Sports goggles with polycarbonate or TriVex lenses.	ASTM F803	PECC
Field hockey	Full face mask for the goalie. Sports goggles with polycarbonate lenses or wire mesh goggles while on the field.	ASTM F803	PECC
Football	Wire face mask and polycarbonate eye shield attached to the helmet.		NOCSAE
Ice hockey	Helmet with full face protection.	ASTM F1587 ASTM F513	CSA/HECC
Men's lacrosse	Helmet with full face protection.		NOCSAE
Women's lacrosse	Full face protection or sports goggles with either polycarbonate lenses or wire mesh goggles.	ASTM F803	PECC
Paintball	Full face protection.	ASTM F1776	PECC
Racket sports	Sports goggles with polycarbonate or TriVex lenses.	ASTM F803	CSA/PECC
Skiing	High impact-resistant eye protector.	ASTM F659	PECC

From the American Academy of Ophthalmology (AAO) (www.aao.org).

BOX 41-2 *Indications for Referral*

Signs and symptoms
Any loss of visual acuity
Visual field cuts
Pupil asymmetry or abnormal pupillary reaction
Perception of flashing lights
Orbit asymmetry
Hyphema
Laceration of eye or complex laceration of lids
Orbital pain with movement of the eye
Halos around lights
Abnormal EOM
Abnormal mass on inspection
Diplopia

EOM, Extraocular movements.

- Confrontational visual field testing: Examiner tests visual fields in all four quadrants of each eye using his or her own eye as a control.
- Inspection: Look for signs of external trauma, bruising, fullness, or subcutaneous emphysema. Mild external trauma can be a sign of more severe internal ocular injury. Do not manipulate or forcibly open an eye if mechanism and exam cannot rule out a ruptured globe.
- Ocular motility testing: Check for the cardinal movements of the eye. Deficiencies in upward gaze may suggest entrapment with orbital blow-out fracture, or neuro-ophthalmological pathology.
- Pupillary examination: Evaluate the pupils initially for uniform roundness and symmetry. Anisocoria may denote an injury along the pupillary pathways. Check direct and consensual responses. If a defect is found, a swinging flashlight exam may be done by quickly moving the light back and forth between the eyes.
  - An afferent pupillary defect, or injury to the retina or optic nerve, results in paradoxical dilation when the light hits the ipsilateral eye. In this case the consensual response would be intact because the efferent pathway from the contralateral eye would be maintained.
  - An efferent lesion would limit direct and consensual response in a lesion ipsilateral to the affected eye.
- Anterior chamber depth can be assessed by shining a light from the temporal side of the eye to observe the amount of medial shadow present. In addition, inspect the iris for a symmetric round opening and for the presence of hyphema.
- Anterior chamber
  - Assess for layer of blood that accumulates at the 6 o'clock position. Look at the iris for uniformity.
  - Assess depth of anterior angle by shining light from temporal to medial. Increased medial shadowing can suggest a narrow angle, though a special slit lamp exam is needed to visualize and fully assess the anterior angle.
- Funduscopic exam: Often impractical on the sideline, but can add useful information for the more complete ophthalmologic exam. This exam is very useful in evaluation of acute visual loss.
  - Red reflex: Opacification of the structures of the anterior chamber, vitreous, or retina can cause a change in this reflex.
  - Funduscopic exam: Assess optic disk margins, cup-to-disk ratio, blood vessels, and retina.
  - Retinal edema resulting from any interruption of circulatory dynamics produces loss of retinal transparency and obscuring of background choroidal circulation. This changes the intensity of red reflex and also obliterates image detail of choroid.
  - Retinal edema occurs in contusion injuries to globe and is also seen in retinal detachments from variety of mechanisms.
  - Contraindications to pupillary dilation:
    - Neurologic monitoring of pupillary response with significant head trauma
    - Suspected acute angle glaucoma or narrow anterior chambers
    - Iris-supported ocular lens implants
- Neurologic: Sensory exam features such as infraorbital hypesthesias may add to diagnosis of orbital floor fracture.
- Special tests
  - Fluorescein staining: Instill anesthetic drops and have the patient blink. The sterile individual strips can then be moistened with sterile water and touched to the inferior cul-de-sac with care taken to not brush the cornea. Use cobalt light or Wood's light to examine for defects, which fluoresce yellow-green.
  - Tonometry: Hand-held electronic tonometers are convenient with readings consistent with applanation. Instill anesthetic drops and apply tonometer per manufacturer instructions.
  - Pressure can also be estimated by palpating globe and using palpated tension in fellow eye for a rough comparison.
    - Care should be taken to avoid compression of ruptured or perforated globe by this test.
    - Intraocular pressure may be elevated with hemorrhage or swelling of orbital contents and may be decreased in certain cases of blunt injury to globe.

SPORTS INJURIES

Corneal Abrasion

Description: Results from cutting, scratching, or abrading the thin, protective surface of the anterior portion of the ocular epithelium. Disruption of the cornea near the central visual axis interferes with visual acuity. Patients often report a history of ocular trauma, or foreign body, and subsequent acute pain. Aggressive eye rubbing can also cause injury. Predisposing factors include foreign body, contact lens, and previous history of corneal abrasion.

Signs and symptoms: Red eye, pain, photophobia, conjunctival injection, tearing, decreased visual acuity if central corneal area is involved, foreign body sensation, and gritty feeling. Symptoms are worsened by blinking, rubbing, and light exposure.

Examination: After fluorescein staining, corneal abrasions and foreign bodies will appear yellow-green against the blue background illumination. Topical anesthetic may be necessary to allow examination. Additional ocular injury must be ruled out, and any foreign bodies should be removed.

Treatment:

- Healing is best facilitated by management of pain and discomfort and controlling lid movement.
- Postinjury infection is uncommon; however, topical antibiotics may be useful in cases with contamination of the eye with debris as well as in immune-compromised patients. Bacitracin, erythromycin, or gentamicin are more lubricating than drops and are considered first-line treatment.
- In contact lens wearers, an antipseudomonal antibiotic such as ciprofloxacin should be given, and contact lens use should be discontinued until the abrasion is completely healed and the antibiotic course completed.
- Patching is no longer recommended. Several randomized, controlled trials (RCTs) have failed to show an increase in healing rate or improvement in pain with patching. Patching may cause increased pain, decreased oxygen delivery, and increased moisture with increased risk of infection.
- In large abrasions associated with significant pain and photophobia, topical nonsteroidal anti-inflammatory drugs (NSAIDs)

such as Voltaren or Acular are modestly useful in reducing pain.
- Generally, avoid topical corticosteroid preparations except in complicated cases, because they can encourage fungal and viral infections.
- In corneal abrasions in contact lens wearers, patients need gram-negative antibiotic coverage. **An ophthalmologist should follow these patients within 24 hours.**

Prognosis:
- Uncomplicated corneal epithelial injuries heal completely within 24 to 72 hours without scarring. Even though frequently contaminated, they rarely become infected.
- **Long-term use of topical anesthetics for pain management interferes with corneal re-epithelialization and is absolutely contraindicated. Topical anesthetics should never be prescribed for home use.**
- **Recurrent epithelial erosion** is infrequent, though patients should be advised that this may occur.
- **Topical mydriatics have not been proven to be beneficial.** Mydriatics were previously thought to relieve ciliary muscle spasm. However, one RCT showed pain was similar in patients using eye lubricant alone or combined with topical NSAID.

Foreign Bodies on Eye and Eyelid Surfaces

Description: May result from penetrating ocular trauma, which is the most common cause of blindness in teenage and young males. Size, shape, and momentum of the object at the time of impact affect the site of ocular penetration. Deeper penetrating objects can lodge within the orbit and cause retinal injury. Superficial corneal foreign bodies are much more common than deeply embedded foreign bodies. However, corneal scarring or infection may occur.

Signs and symptoms: Same as for abrasions.

Examination:
- Tiny foreign bodies may require magnification to be properly visualized and removed.
- If a metallic foreign body has been embedded for hours to days, a rust ring may be present.
- Localization can be enhanced by fluorescein stain. Foreign bodies will appear yellow-green against the blue background illumination. Use of topical anesthetic may be necessary to facilitate examination.
- Both upper and lower conjunctival fornices should be examined carefully for presence of foreign body.
- Upper eyelid should be everted and the conjunctival surface inspected for the presence of foreign body.
- A positive Seidel test will help detect leaking aqueous or exposed vitreous. Fluorescein ophthalmic strips are wetted with normal saline. The concentrated fluorescein is dark orange, but if it becomes diluted with aqueous, then it turns bright green under blue light. The presence of an intraocular foreign body (IOFB) suggests globe penetration. The patient may be asymptomatic if the foreign body is below the epithelial or conjunctival surface. Over a few days epithelium may grow over small corneal foreign bodies, with pain reduction. If a corneal infiltrate is noted, an infectious cause should be considered. Foreign bodies can cause a small sterile inflammatory reaction around the foreign object. However, if a large infiltrate, ulceration, significant anterior chamber reaction, or significant pain is present, it should be managed as an infection.
- If there is concern for an intraocular foreign body, helical computed tomography (CT) scan with 1 mm axial and coronal cuts or biomicroscope ultrasound can be considered. Referral should be made to an ophthalmologist for a dilated exam and management.

Treatment: For corneal foreign bodies, apply short-acting topical anesthetic. Removal of the foreign body using irrigation, sterile needle, and foreign body removal instrument should be performed, however, if likelihood of penetration through more than 25% of the cornea exists, referral should be made to an ophthalmologist for surgical removal. Cotton tip applicators are not appropriate because the large surface area may cause an epithelial defect.
- Topical antibiotic ointments such as Bacitracin or Ciloxan should be prescribed to prevent infection.
- Rust rings may be removed by a well-trained physician with an Alger brush or automated burr. Rust rings should be visualized using a slit lamp.

Eyelid Lacerations

Description: May occur in association with blunt trauma as well as from sharp objects and can result from the propulsion and shattering of eye protection equipment (Fig. 41-2).

Signs and symptoms: Swelling, hemorrhage, anatomic disruption of lids. Damage may be subtle and appearance normal.

Examination: Evaluation of normal anatomic relationship of lid margins and front surface of globe as well as symmetry with uninjured side. Opening and closing functions are assessed specifically, especially to verify that lids can be spontaneously closed. Attention needs to be directed to rule out possibility of upper eyelid ptosis or lacerations in lacrimal drainage system. Globe must be thoroughly inspected for signs of damage.

Treatment: Lacerations require individual suturing of lid tissue layers and additional repairs specific to any injuries involving integrity of lacrimal drainage apparatus.

Prognosis: Because of rich vascular supply to eyelids, healing is rapid and deformities are minimal in cases of minimal tissue loss and good anatomic approximation. **Particular injuries requiring ophthalmology referral include:**
- Lacerations involving the upper or lower lid margin.
- **Lacerations suspected to involve the lacrimal sac or duct.**
- Lacerations with exposure of orbital fat.
- Horizontal lacerations with ptosis and possible disruption of the tarsal plate.
- Any laceration with avulsion of eyelid tissue.

Contusion Injuries

Blunt injuries to the globe are the most common sports injury to the eye and may be associated with ruptured globe or hyphema. They result from facial blows directly to orbital contents or from

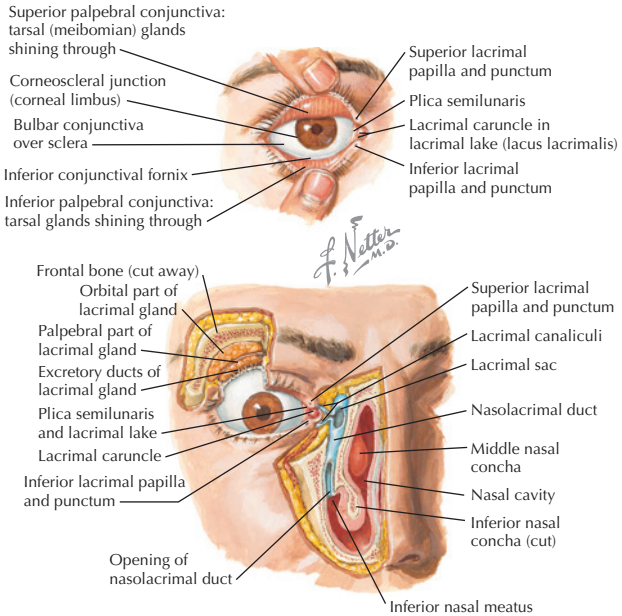

Figure 41-2 Orbit Eyelids and Lacrimal Apparatus.

sudden pressure increases transmitted to eye from surrounding orbital tissue.

Blunt Trauma to Orbit

RUPTURED GLOBE

Description: Occurs when the full thickness of the cornea or sclera is breached. The potential for serious morbidity of the eye is present with this injury.

Epidemiology: One study of open globe injuries ordered the following injuries from most common to least common: fishing, hunting/shooting recreation, baseball/softball, golf, basketball, and racket sports.

Mechanism of injury: History should elicit the mechanism of injury, change in visual acuity, pain, previous visual impairment, use of eye protection, and time of last meal. Injury often occurs by one of two methods:

- Rupture occurs when hard objects increase intraocular pressure, causing rupture at the weakest point.
- Lacerations occur when sharp (e.g., sticks) or small hard objects (e.g., shrapnel, BB's, shattered eye protection) enter the globe at high velocity.

Physical exam:

- Evaluate anterior segment of globe for signs of subconjunctival hemorrhage.
- The pupil should be round, central, and symmetric with the fellow eye.
  - Lacerations of cornea frequently incarcerate iris tissue, causing distortion and displacement of pupil.
  - Scleral lacerations also may displace location of pupil because of herniations of uveal tract through defect.
- Prolapsed uveal tissue presents as dark brown or black mass, even in those with fair complexion and blue-eyed people.
- Lacerations of globe may result in subluxation of crystalline lens.
- Intraocular bleeding is also a frequent complication, causing obscuration of ocular media and loss of red fundus reflex.
- Intraocular pressure may be decreased.

Treatment: Requires emergent ophthalmology referral:

- Manage other trauma including safe clearance of other head or neck trauma.
- Patient should be given nothing by mouth.
- Keep a **rigid** shield over eye at all times.
- No topical medicines.
- Tetanus status should be updated.
- Other considerations to discuss with ophthalmologist include narcotics, prophylactic antibiotics, imaging.
- Penetrating objects remaining in place should be secured in place without removal and covered for protection. A Styrofoam cup may be a useful adjunct for this.

Prognosis: In patients with penetrating eye trauma, predictors of excellent final visual acuity (defined as 20/60 or better) were initial visual acuity of 20/200 or better, wound location anterior to the plane of insertion of the four rectus muscles, wound length 10 mm or less, and sharp mechanism of injury. Predictors of poor outcomes were initial visual acuity of light perception or no light perception, wounds extending posterior to rectus muscle insertion plane, wound length greater than 10 mm, and blunt or missile injury. Only 50% of children with ruptured globe injuries recover good visual acuity.

Return to play: After a ruptured globe injury, return-to-play decisions should include consultation with ophthalmologist and include discussion of risks and benefits of participation as outlined above.

ORBITAL FRACTURE

Description: An orbital blowout fracture occurs when blunt trauma to the eye or orbit is transmitted to the bony walls of the orbit, causing fracture. This may result in entrapment of contents of the orbit, including the muscles of the eye, leading to restricted gaze and diplopia (Fig. 41-3).

Signs and symptoms: A high index of suspicion for this injury should be present when an object larger than the orbit of the eye strikes the eye. History should elicit the mechanism, change in visual acuity, pain, previous visual impairment, and use of eye protection. Additional symptoms suggestive of injury are diplopia, especially with upward gaze, and ipsilateral nosebleed.

Examination:

- Rule out ruptured globe.
- Findings include:
  - Enophthalmos, which may have the appearance of relative ptosis.
  - Periorbital ecchymoses/abrasions suggest significant mechanism to cause injury.
- Restricted vertical gaze occurs with orbital floor fractures and entrapment of the inferior rectus muscle, tethering the eye.
- Infraorbital hypesthesia of cranial nerve V2 can occur because of neurapraxia of V2 that runs through the orbital floor/maxillary sinus roof.
- Coronal CT with 2 mm slices enables visualization of soft tissue densities, such as prolapsed orbital fat, extraocular muscle, and hematoma.
- Plain radiographs can have a false negative rate of up to 50%.
- MRI does not visualize bone well.

Treatment: Surgical treatment is usually necessary only when there is actual interruption or herniation of orbital tissue. Diplopia may be transient after these injuries; surgical intervention should be deferred until a significant portion of contusion injury has resolved. Conversely, in cases with little contusion injury and

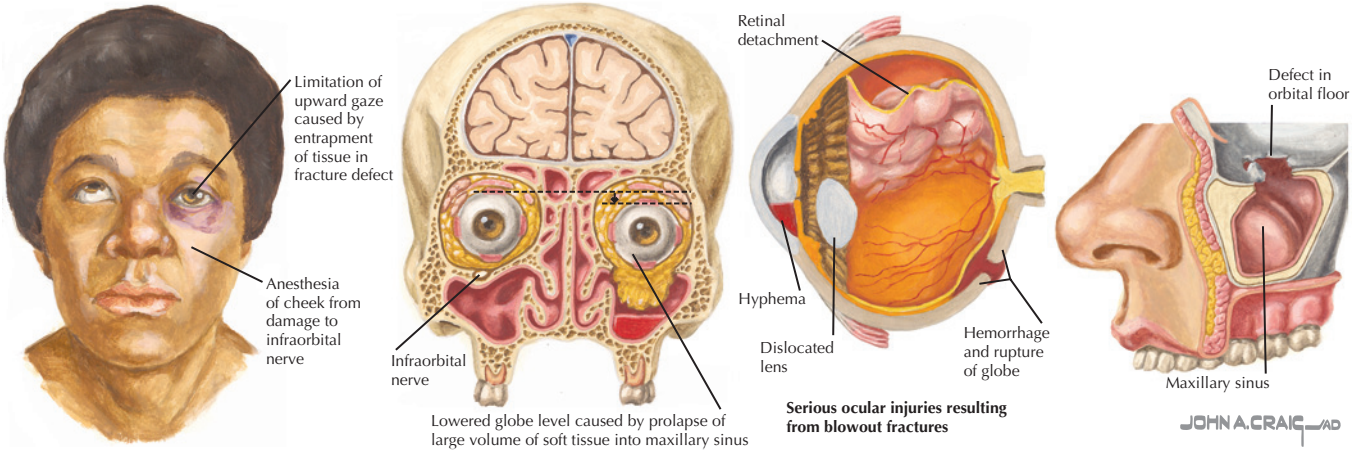

Figure 41-3 Orbital Blowout Fractures.

obvious interruption or herniation of orbital tissue, there is no need to delay definitive repair.

Prognosis: Varies depending on degree of injury. Prognosis is best when orbital tissue damage is minimal. Prolonged tissue entrapment and inflammation can result in fibrosis and contractures, which can lead to permanent functional disabilities.

HYPHEMA

Description: Refers to presence of blood in anterior chamber and can result from a tear in peripheral iris vessels. Anteroposterior compression of the globe leads to expansion of the globe with damage to the blood vessels of the iris or ciliary body. There is a high frequency of concomitant injury, including blunt or penetrating trauma. Projectiles that strike the exposed portion of the eye are a common cause of hyphema. Projectiles may include balls, rocks, toys, air gun blasts, hockey pucks, paint balls, and fists. Baseball and softball players are commonly affected. Hyphema frequently occurs in microscopic quantities and, consequently, can be easily overlooked.

Signs and symptoms: Because many hyphema are small, visual acuity may be unaffected. There may be mild injection of globe with moderate transient discomfort and photophobia. In addition to hyphema, other injuries associated with contusion injuries may also be found: papillary paralysis, pupillary contour irregularities, and tearing of uveal tract.

Examination: Slit lamp examination is necessary to identify any turbid microscopic hyphema before it has had time to settle out in chamber; however, careful penlight exam can identify hyphema in acute settings. Intraocular pressure (IOP) should be measured. Of hyphema patients, 25% have other ocular injuries, including vitreous or retinal hemorrhage or subluxated lens.

Treatment:

- Patients should be managed as if they have an open globe.
- Shielding of the eye should be done with a protective metal shield for 2 weeks.
- Physical activity should be severely restricted with bed rest for the first 4 days and light activity for the first 2 weeks. Topical cycloplegics (atropine sulfate twice daily for 2 weeks), and topical corticosteroids should be given. Salicylates and NSAIDs should be restricted. Acetaminophen can be given for pain relief. Allergan should be followed by latanoprost (Xalatan, Pharmacia) and timolol maleate (recommended for elevation of IOP).
- Patients should be examined for 3 consecutive days to monitor for rebleeding or IOP elevation. Recurrent bleeding is most frequent during the first 5 days after injury and occurs in 4% to 6% of cases. IOP may lead to glaucoma. Patients with sickle cell trait or disease are more susceptible to this complication. **Increased intraocular pressures may accompany hyphemas of any size.**
- Corneal bloodstaining is possible.
- Sickle cell status should be established in all African-American patients or Hispanic patients who have IOP greater than 21 mm Hg.
- Patients should be referred to an ophthalmologist immediately and the eye should be shielded.
- Patients should be seen again if there are no problems 2 weeks after the injury.

Prognosis: Excellent, especially if blood clears rapidly, and no recurrent bleeding, or other injuries are noted. Usual duration of an uncomplicated hyphema is 5 to 6 days.

- **Recurrent bleeding may lead to permanent loss of visual acuity.**
- Most microhyphemas can be treated on an outpatient basis, unless rebleeding or IOP is uncontrolled. If the hyphema occupies more than one third of the anterior chamber, intraocular pressure is elevated beyond 30 mm Hg, or both, hospitalization is recommended.

VITREOUS HEMORRHAGE

Description: When vitreous hemorrhage is caused by trauma, significant force is involved and additional injury to the eye is generally present. It can result from blunt trauma or shaking, or proliferative retinopathy. Blood obscures the light path through the vitreous cavity of the eye and reduces visual acuity.

Signs and symptoms: Assessing and documenting the patient's vision prior to symptoms of hemorrhage is crucial. Patients may report seeing "floaters, visual haze, smoke, shadows, or cobwebs." More severe hemorrhages may result in the sensation of dark streaks that break up into numerous, minute black spots or vision reduction to only light perception. Isolated vitreous bleeding is not associated with symptoms such as pain or discomfort.

Examination: Measure visual acuity and pupil response. On funduscopic examination, fundus detail is blurred or disappears entirely and may be seen as a "black reflex." On slit lamp examination: Fresh blood is identified readily by adjusting the slit beam to a tangential position and viewing the anterior vitreous directly behind the lens.

Treatment: Generally conservative. Severe cases may require surgical removal of blood and vitreous. Such procedures are often done at time of repair of associated ocular injuries.

Prognosis: Guarded.

RETINAL HEMORRHAGES AND DETACHMENT

Description: Can result from direct trauma to eye by transmission of force to retinal surface. This produces immediate loss of visual function within the detached segment. In the absence of treatment, the entire retina eventually becomes involved, and total retinal detachment develops. Can be caused by retinal instability seen with violent exercise performed in conditions of decreased oxygen saturation or elevated venous pressure from Valsalva maneuvers. Such findings have been documented in activities such as mountain climbing and weightlifting. **Retinal detachment** produces immediate loss of visual function within detached segment. Any form of blunt or perforating trauma can produce retinal detachment. Indirect trauma, such as severe head injury, myopia, and vitreous traction, are risk factors.

Signs and symptoms: Assessing and documenting the patient's vision prior to symptoms of hemorrhage is crucial. Multiple asymptomatic areas of retinal hemorrhage and edema are not uncommon, especially if affected areas are confined to peripheral retina. Involvement with macula results in decreased visual acuity or distortion of visual perception. Patients often report positive scotoma at edge of visual field or seeing "floaters, visual haze, smoke, shadows, or cobwebs." As detachment progresses, the patient may describe visualization of "lightning flashes" or "flying sparks." An enlarging scotoma may be seen as a waving, black curtain encroaching on central vision. Severe hemorrhages may result in the sensation of dark streaks that break up into numerous, minute black spots or vision reduction to only light perception.

Examination: Visual field and acuity measurement. Funduscopic examination reveals both flame-shaped hemorrhages and round-blot hemorrhages or may show a "black reflex." On slit lamp examination: Fresh blood is identified readily by adjusting the slit beam to a tangential position and viewing the anterior vitreous directly behind the lens. As the retina becomes more elevated, it is necessary to add additional convex or plus lenses to the funduscopic viewing port to maintain sharp focus on internal retinal surface. It is necessary to dilate the pupil widely to visualize a detachment during its early stages because detachments begin in the far periphery.

Treatment: Symptomatic. Surgical intervention is almost invariably necessary after actual separation of retina has occurred. If surgery is not indicated, then close observation for 1 to 2 weeks allows time for spontaneous clearing of some hemorrhage, but it may take several months for complete vision to return. Upright positioning

for sleep may enhance settling of the hemorrhage. Emergent consultation is required if hemorrhage resulted from trauma.

Prognosis: Varies depending on location, extent, and severity of involvement. A chance for recovery of good central vision is generally poor if retina becomes detached in the area of macula.

DISLOCATED LENS

Description: Results from tearing of lens zonules and loss of support in its normal position. May result in subluxation or movement of lens away from site of injury, causing it to decenter slightly but remain in relatively normal position. More extensive damage may displace lens entirely, causing it to fall either into anterior or posterior chamber.

Signs and symptoms: Visual acuity is affected by even the slightest shift in position of crystalline lens:

- Lens decentering causes irregular astigmatism with poor near vision secondary to loss of accommodation power and decreased distance visual acuity.
- Complete dislocation results in condition of aphakia.
- Shifts of lens position, even though slight, can also cause loss of iris stability and resulting tremulousness to slight ocular movements or vibrations (iridodonesis).
- Monocular diplopia.

Examination:

- Visual acuity may be initially reduced but can frequently be corrected by change in refraction.
- Slit lamp examination may reveal iris undulations (iridodonesis) at pupillary margin after rapid eye movements.
- Strabismus is not uncommon (secondary to amblyopia).
- Pupillary dilatation can aid in assessment of lens position and can evaluate retina.
- Enophthalmos with facial myopathic appearance may be seen in patients with Marfan syndrome.
- IOP should be assessed because lens dislocation can result in secondary glaucoma.
- If Marfan syndrome, homocystinuria, or other collagen vascular diseases are suspected cardiac workup should be performed including a cardiac echocardiogram. Check serum and urine levels of homocysteine or methionine for homocystinuria. Genetic-associated lens dislocation is usually bilateral.

Treatment: Variable and may necessitate surgical removal of lens. Immediate referral should be made to an ophthalmologist if this injury is suspected.

Prognosis: Varies with extent of injury.

CHAMBER ANGLE RECESSION

Description: Generally results from blunt trauma to globe, causing sudden increase in pressure within anterior chamber, which is transmitted to lens-iris diaphragm, propelling it backward. Dynamics of this injury are similar to that of lens dislocation; however, in angle recession, lens position is usually normal. In few cases, force of injury is sufficient to produce associated injury to trabecular meshwork, which may eventually lead to glaucoma.

Signs and symptoms: In cases in which there is development of glaucoma, the onset is almost invariably delayed from the time of the injury and progresses slowly. As with other forms of chronic glaucoma, visual loss is insidious, beginning with the peripheral areas of the visual field involved initially. Angle recession glaucoma should always be suspected in cases of unilateral chronic glaucoma.

Examination: Usual methods for evaluating chronic glaucoma including elevation of intraocular pressure and visual field examination, with special attention to classic field defects commonly seen in chronic glaucoma.

Treatment: Angle recession glaucoma is frequently not responsive to medical therapy and may require surgical treatment.

Prognosis: Varies with extent of pressure elevation, length of time it has been present, and responsiveness to treatment.

RED EYE

Description: The aim of management should be differentiation of the symptom of red eye and assessment of the underlying disease. Generally, redness of the eye can be caused by hyperemia with dilation of the conjunctival, episcleral, or scleral vessels (caused by trauma, chemical burns, immunologic reactions); inflammatory reactions from infections (bacterial, viral, fungal); or chronic reactions of the external eye from systemic causes (Box 41-3).

Signs and symptoms:

- Association with pain.
- Pain, itching, visual decrease or loss.
- Mucopurulent discharge, watering, blepharospasm (lagophthalmus), or systemic (fever, nausea) findings.
- Foreign body sensation.
- Decreased visual acuity.

Examination: Should include the anatomic location of redness (eyelids, conjunctiva, cornea, sclera and episclera, or intraocular). Measure IOP (to rule out acute closed angle glaucoma) and check visual acuity (if decreased, needs urgent referral to ophthalmologist). Measure pupil size and response to light. Fluorescein testing may be necessary.

Treatment: Depends on the cause of symptoms.

Prognosis: Depends on the cause of symptoms. Athlete should not return to play until the cause of symptoms is ascertained.

INJURY PREVENTION

Risk Factors to be Considered

- Physical development and skill level.
  - Beginners may have increased risk because of lack of necessary refinement of skill of sport.
  - Advanced players, especially in some high-risk sports, may play more aggressively and be at greater risk for eye injury.
- **Existing visual impairment** increases risk of injury.
- **Preexisting eye disease** may present increased risk factor to athletes in all risk groups.
  - Conditions that may lead to serious eye disorders or get worse after even minor trauma to the eye include retinal detachment, retinal degeneration, severe myopia, thin sclera, and prior ocular surgery.
  - Systemic eye diseases and previous serious injuries may be risk factors as well.

Types of Eye Protectors

Total head protector: Combination of helmet and face shield designed to protect eyes, teeth, jaw, and larynx and transfer forces to skull. Designed for use in high-risk sports that require total head protection: football, hockey, and lacrosse.

BOX 41-3 *Most Common Causes of Red Eye*

Conjunctivitis
Episcleritis and scleritis
Keratitis and corneal ulcer
Iritis and intraocular infections (endophthalmitis)
Glaucoma (acute and chronic)
Dry eye
Subconjunctival hematoma (hyposphagma)
Corneal and conjunctival foreign body
Corneal abrasion
Corneal flash burn
Chemical burns
Blunt or penetrating trauma to the eye
Allergic reaction
Blepharitis

Full face protector: Designed for use in conjunction with eye protectors for high-risk sports that do not require protection for brain: fencing, some positions in baseball and softball.

Helmet with separate eye protectors: For use in sports with low risk for injuries to lower face and neck: cycling, snowmobiling, skiing, automobile racing, and bobsled racing.

Helmet only: Helmets are designed to protect brain only. They afford little protection to face or eyes and are used in boxing and cycling.

Sports eye protectors: Used only to protect eyes and are recommended in all high-risk sports for which additional head and face protection is impractical: all racquet sports, baseball, soccer, basketball, and softball (see Table 41-3).

"Sports" sunglasses: Most are inadequate for both impact resistance and ultraviolet radiation blockage. Adequate eyewear of this type should:

- Contain manufacturer's statement recommending intended sport.
- Block light from sides and below.
- Protect from glare (transmit < 30% of light).
- Be lightweight, cosmetically acceptable, and aerodynamically designed to prevent drying in wind.

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Maxillofacial Injuries

42

Scott Escher, Michael Case, and Lawrence Kent

INTRODUCTION

General Considerations

Epidemiology

- 3% to 29% of facial injuries are a result of sporting activity.
- 60% to 90% of facial injuries in sports occur in males between the ages of 10 and 29.
- Approximately 75% of facial fractures occur in the zygoma, mandible, or nose.
- The most commonly injured teeth are the maxillary central incisors, followed by the lateral incisors and then the mandibular incisors.

Initiation of Care in the Head-Injured Athlete

- Airway injury or compromise
  - Follow ABCs (airway, breathing, circulation) of basic life support.
  - May need to secure airway before making any other assessment.
  - If neck is injured, use jaw thrust maneuver.
  - May be difficult to maintain airway with unstable mandibular fracture or some soft tissue injuries.
  - Can use oral airway or endotracheal tube as indicated in unconscious patient. Nasal trumpet works well in awake patient without midface fracture.
  - Cricothyrotomy may be only option in emergency.
- Cervical spine injury
  - Need to stabilize spine if there is any doubt regarding injury.
  - If athlete is unconscious or of dubious mental status, the cervical spine should be stabilized.
  - Discussed in more detail in Chapter 40, Neck Injuries.
- Concussion
  - When mental status is altered, the physician needs to be more vigilant regarding search for associated injuries. Conversely, with facial injuries, the physician needs to look for associated cervical spine injuries or concussion.
  - Discussed in more detail in Chapter 39, Head Injuries.

History

- Determination of mechanism of injury can help physician direct exam and make diagnosis.
- Query the athlete regarding what has happened in the contest to see if there is a possibility of concussion.
- Ask the athlete if there is a history of abnormality such as crooked nose, missing teeth, anisocoria (unequal-sized pupils), etc.

Physical Exam

Inspection

- Look for facial asymmetry, widening of the midface, or ocular asymmetry. Need to examine from different angles.
- Best to do this early before swelling causes asymmetry.
- Observe for malocclusion.
- Bleeding or bruising sites should alert physician to other possible injuries in area.

Palpation

- Palpate bony structures systematically.
- Can palpate maxilla and mandible bimanually with gloved finger in oral cavity.
- Conduct sensory exam for nerve injuries. Three branches of trigeminal nerve supply the face: ophthalmic innervates the forehead, maxillary innervates the cheek and midface, mandibular innervates the jaw (Fig. 42-1).

Range of Motion

- Assess mandibular motion. Pain with opening or closing can indicate fracture.
- Ocular motions important to document in facial fractures.

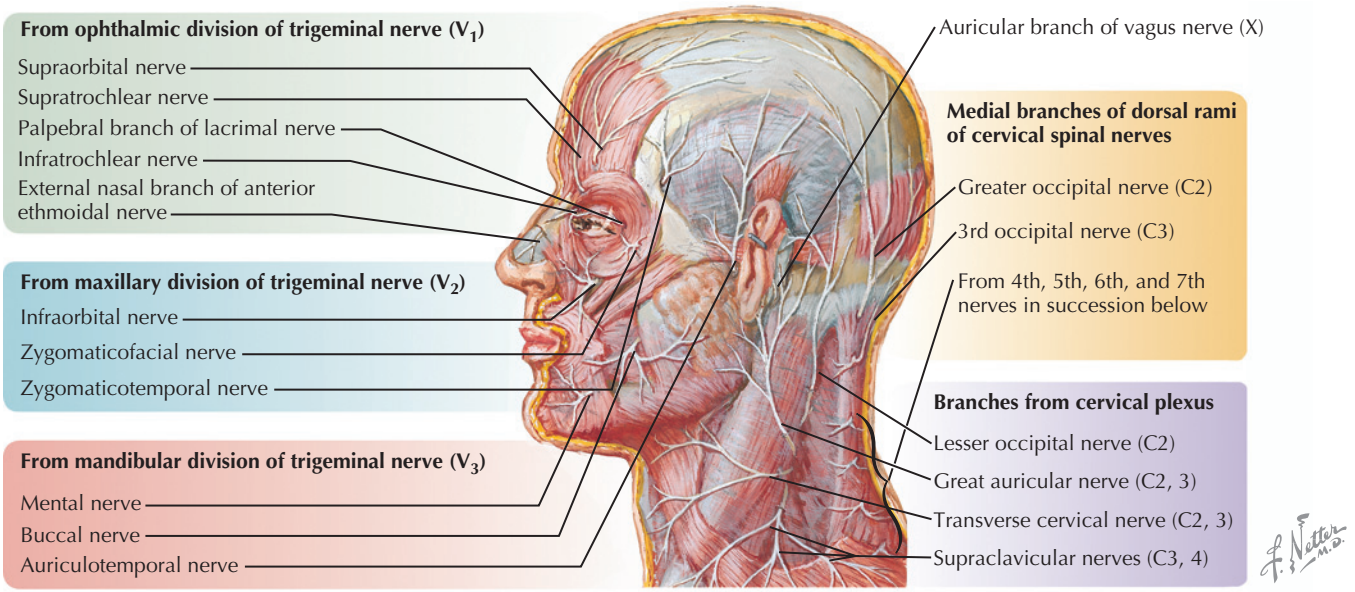


Figure 42-1 Cutaneous Nerves of Head and Neck.

Imaging Studies

Conventional Radiography

- Conventional radiography is rarely used for nasal bone evaluation because imaging adds little to clinical exam; however, if needed, most common x-rays of the nose include right and left lateral, superoinferior axial occlusal, and Waters' views.
- For a facial bone series, submentovertex, Waters', lateral obliques (right and left), Towne's, and posteroanterior views can be used.

Cross-Sectional Imaging

- Computed tomography (CT) and magnetic resonance imaging (MRI) are greatly superior to conventional radiographs in showing both normal and abnormal anatomy.
- A facial bone CT offers much better anatomic detail and is the study of choice over plain radiographs in trauma.
- CT is superior in showing bony anatomy.
- MRI is superior in soft tissue imaging. Also displays vascular anatomy without using contrast. Rarely used in trauma.

INJURIES

Soft Tissue Injuries

Lacerations

Mechanism of injury: Projectile (e.g., puck), implement (e.g., bat), or contact (e.g., chin on opponent's helmet or ice) can cause laceration.

Examination: If abundant blood supply around face makes exam difficult, apply superficial pressure over dominant blood vessel in area to slow bleeding. Assess for both sensory and motor nerve injury. Remember that underlying bone can be injured.

Treatment: Cleanse well with sterile water or irrigating solution. Anesthetize locally with lidocaine with epinephrine to help with hemostasis. Some advocate not using epinephrine on ear or nose. Can use dissolving suture to reapproximate the subcuticular tissue. May need a layered closure on deep wounds. Skin closure can be with 5-0 or 6-0 suture or tissue adhesive if skin edges closely aligned. Proper alignment of vermilion border of lip important for cosmetic purposes. If the laceration is minor and bleeding can be controlled, the athlete can return to competition until definitive care can be arranged. Need to cover with occlusive dressing prior to return to competition.

Ear Injuries

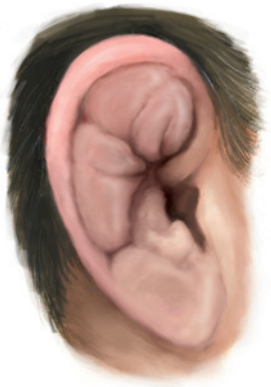
Auricular Hematoma ("Cauliflower Ear")

Mechanism of injury: Usually caused by shear forces on ear; commonly seen in wrestlers without head protection. Blood and/or serum between ear cartilage and perichondrium or between sheared layers of perichondrium can result in permanent deformity of ear.

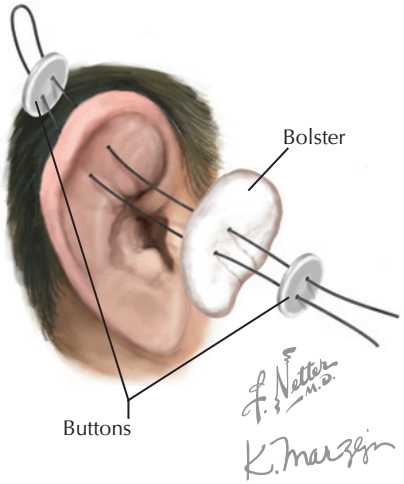
Examination: Swelling or fluctuant area in cartilaginous area of ear usually on outer or lateral side of ear (Fig. 42-2).

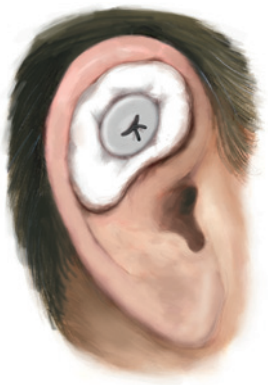
Treatment: Can usually allow continued participation in event in which injury occurs. Initially treated by aspiration of hematoma or incision and drainage after anesthetizing:

- Permanent disfigurement can occur if not treated properly.
- Field block anesthesia can be accomplished by infiltrating posterior sulcus as well as skin anterior to the helix and tragus with 1% or 2% lidocaine without epinephrine.
- Hematoma will reoccur if not bolstered. Several methods exist to prevent the hematoma from reaccumulating. To apply pressure, can use bolster in which sutures hold packing onto area with slight pressure. Bolster can be dental roll on both sides of the pinna held in place by 3-0 or 4-0 monofilament nonabsorbable suture. Some advocate a button or silicone splint on either side of the ear instead of dental roll. Need to prevent further trauma with wrestling headgear and leave bolster in place for 7 days (see Fig. 42-2).



Appearance of auricular hematoma (cauliflower ear).





Bolster properly placed.

Figure 42-2 Cauliflower Ear.

- Always use antibiotics with *Staphylococcus* coverage with a bolster in place.
- If infection occurs, bolster must be removed and area must be incised and drained. Antibiotic should be appropriate for *Staphylococcus* and *Pseudomonas*.

Prevention: Proper headgear.

Ear Laceration

- See "Lacerations."
- Sew ear in layers with cartilage, perichondrium, and finally skin. On lateral portion of ear, all three layers may need to be sutured together because they can be closely adherent.

- Attempt to minimize sutures in cartilage.
- Use undyed absorbable 6-0 suture for cartilage and perichondrium.

Otitis Externa ("Swimmer's Ear")

Mechanism of injury: Anything that injures the thin ear canal skin, especially in athletes with water in the canal, can bring about otitis externa. Usually caused by *Pseudomonas aeruginosa* or *Staphylococcus aureus.*

Examination: Signs and symptoms include pain and discomfort with motion of pinna and swollen, inflamed, and erythematous external auditory canal. Purulent discharge usually present.

Treatment: Treat with combination of antibiotic and corticosteroid drops (e.g., hydrocortisone + polymyxin + neomycin) for 5 to 7 days (ciprofloxacin with steroid if allergic to neomycin). Suction ear to remove debris. May need wick to allow antibiotics to penetrate to innermost portion of ear canal if significant swelling present; leave in place for 3 to 5 days. Consider treating significant infection with oral antibiotics; make sure it includes *Pseudomonas* coverage (e.g., levofloxacin). Athlete usually should stay out of water until asymptomatic.

Prevention: Dry ears after swimming; a hair dryer to ear can help reduce moisture. A combination of alcohol and vinegar or commercially available alcohol-based products such as Swim Ear can help lessen moisture and may reduce incidence of otitis externa.

Tympanic Membrane Perforation

Mechanism of injury: Can occur after skydiving or scuba diving with pressure changes, or after a blow to the ear by a large ball or in boxing. A fall onto the ear while water-skiing can also cause perforation.

Examination: Hearing loss, serous or bloody drainage from ear, hole visible in tympanic membrane (TM) with otoscopic exam; associated vertigo can signify ossicular disruption.

Treatment: Most (85% to 90%) will heal without treatment; if not healing within 2 to 3 weeks refer to otolaryngologist. In water sports, use earplug to keep water out of canal until perforation heals. Need to curtail sports with large changes in pressure such as platform diving until perforation heals. If this injury occurs in river or lake water, consider antibiotic eardrops. If you suspect ossicular disruption, athlete needs immediate referral to an otolaryngologist.

Dental Injuries

History:

- Is there spontaneous pain, which indicates pulp exposure?
- Are any teeth tender with chewing, which indicates injury to the periodontal ligament?
- Are any teeth sensitive to extremes of temperature, which indicates pulp exposure or inflammation?
- Is there a change in the bite (malocclusion), which suggests facial fracture or dental subluxation?

Examination: Examine teeth for fractures and laxity and soft tissue for associated lacerations and bruising. Radiographs should be done with dental fractures or luxation. Can assess for root or bony fracture, or in the case of younger athletes for permanent tooth bud displacement.

Treatment:

Primary Teeth: Main goal in treatment of primary teeth is to prevent injury to permanent teeth. Children with displaced or significantly loose primary teeth should see dentist as soon as possible. Do not replace avulsed or extruded (completely dislocated) teeth because this may injure permanent teeth. Mildly subluxed teeth, those which appear in normal position but have pain with chewing, should be treated with a soft diet; the child can see a dentist in a few days. Children with tooth fractures should be referred to a dentist:

- Crown fractures involving the pulp need prompt treatment to prevent infection.
- Children with crown fractures not involving the pulp and affecting only the enamel and dentin should also see a dentist within a few days.
- Children with root fractures should see a dentist within a few days.

Permanent Teeth: Subluxed permanent teeth that are crooked should be seen by a dentist as soon as possible. The on-site physician or athletic trainer does not need to attempt to straighten the tooth immediately. Fractures of teeth can be treated hours after the injury. Fractures involving the pulp can be painful. One can transport fractured teeth in tap water as long as the root is not involved. Avulsed teeth should be transported in Save-A-Tooth as noted later. Tetanus prophylaxis should be given if tooth is contaminated with dirt and last tetanus shot was more than 5 years previous. Avulsed or displaced teeth should be reimplanted immediately if possible. Tooth survival diminishes quickly with the amount of time out of the socket. The survival of the delicate periodontal ligament cells on the root of the tooth is necessary for tooth survival. Little chance for dental survival after 1 hour out of the socket:

- Tooth should be handled by the crown.
- Tooth should be rinsed with saline or tap water. Do not rub or attempt to sterilize root.
- The athlete can then keep the tooth in place with finger pressure or by biting on a gauze pad until he or she sees a dentist.
- Dentist should be seen as soon as possible after the tooth is replaced.
- If immediate reimplantation is not possible, the tooth should be stored in culture medium, which is commercially available as Save-A-Tooth. This will maintain the viability of the periodontal ligament cells longer, increasing the likelihood of successful reimplantation. Cold milk is a good alternative to this. Saline solution can also be used if the other two alternatives are not available. Water is not helpful in extending the viability of the periodontal ligament cells.

Prevention: Mouth guards can prevent dental injury. Basketball players are seven times more likely to have an orofacial injury when not wearing a mouth guard.

Nasal Fracture

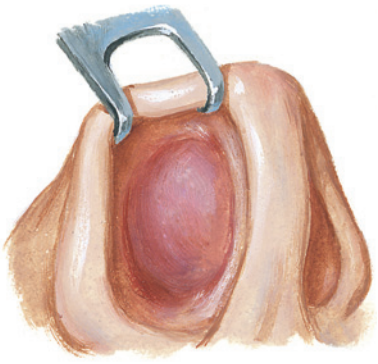
Mechanism of injury: Trauma to nose causing fracture of bone. Can appear similar to injury to bone-cartilage interface.

Examination: Signs and symptoms include epistaxis, nasal asymmetry, crepitus on palpation, swelling, and nasal airway obstruction. Assess for septal hematoma (Fig. 42-3), submucosal swelling on one or both sides of septum.

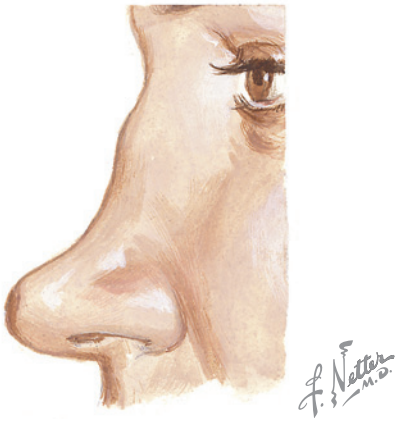
- Septal hematoma is suggestive of injury to blood supply to nasal cartilage.
- If untreated, cartilaginous deformity can result (see Fig. 42-3).

Imaging: X-rays may show fracture. Radiograph not necessary. X-ray may be normal if injury is at the bone cartilaginous interface. Some advocate prereduction photograph for medicolegal purposes.

Treatment: Control hemorrhage with compression or nasal packing; most bleeding will stop with time. Topical vasoconstriction or thrombin may be used. Need evacuation and treatment of septal hematoma within 24 to 48 hours to prevent permanent damage to underlying nasal cartilage (i.e., saddle nose deformity). Use antibiotic with *Staphylococcus* coverage if packing or if hematoma is present. Reduce displaced fractures immediately or can wait 5 to 7 days for swelling to diminish; waiting longer can make reduction more difficult. May be able to control fracture with splinting; complex fractures may need operative reduction. Restrict aerobic activity for a few days.



Examination for septal hematoma.



Cartilaginous deformity secondary to untreated septal hematoma.

Figure 42-3 Nasal Fracture.

Return to play: Facial protection can be purchased off the shelf or individually fabricated for athletes in contact or collision sports such as basketball or soccer. Athletes can return to contact/collision sports with the facemask in approximately 4 weeks. Many athletes return earlier than 4 weeks with the understanding that they may refracture and require further treatment.

Epistaxis

Mechanism of injury: Usually occur spontaneously at Kiesselbach's plexus on nasal septum in area of thin nasal mucosa overlying blood vessels. Trauma is second most common cause of epistaxis. Epistaxis can occur without nasal fracture. Posterior bleeding is less common and can be difficult to control.

Examination: If bleeding site is difficult to find, a nasal telescope can be used.

Treatment: Most nosebleeds resolve spontaneously in a few minutes; apply pressure to the upper lip over the superior labial artery. Pinch the nostrils against the nasal septum, which may constrict blood flow from the anterior ethmoidal artery and the sphenopalatine artery (Fig. 42-4). If the bleeding site is at Kiesselbach's plexus, one can insert a folded dental roll into the naris temporarily, which will apply pressure over the bleeding site and allow the athlete to finish the contest. If time allows, consider vasoconstriction of the area with topical oxymetolazone, cocaine, or epinephrine 1:100,000. An alternative is to use a combination of lidocaine, epinephrine, and tetracaine topically. Then chemically cauterize the area with a silver nitrate stick or use electrocautery. Anterior nasal bleeding that does not stop with these maneuvers can be treated with nasal packing. Posterior bleeds and those anterior bleeds not controlled with nasal packing need an urgent visit with an otolaryngologist. Petrolatum applied nightly to the nasal septum can prevent drying and cracking of the nasal mucosa, which may help prevent epistaxis.

Mandibular Fractures

Mechanism of Injury: Usually direct trauma to jaw by another athlete, fall, or projectile impact (e.g., ball, bat, puck). Athlete notes jaw feels loose or complains of crepitus or inability to occlude teeth together.

Examination: Signs and symptoms include pain with palpation, malocclusion; may have numbness with injury to the mandibular nerve, loose teeth, loose segments of jaw; laceration of oral mucosa overlying mandible may be an indicator of underlying fracture; inability to open or close jaw (Fig. 42-5).

Imaging: Panorex x-ray, CT.

Treatment: Can initially immobilize with an ace wrap until athlete can get to facility with imaging capabilities (if the athlete can breathe through the nose). Reduction and stabilization needed. Bone immobilized with maxillomandibular fixation (wiring of

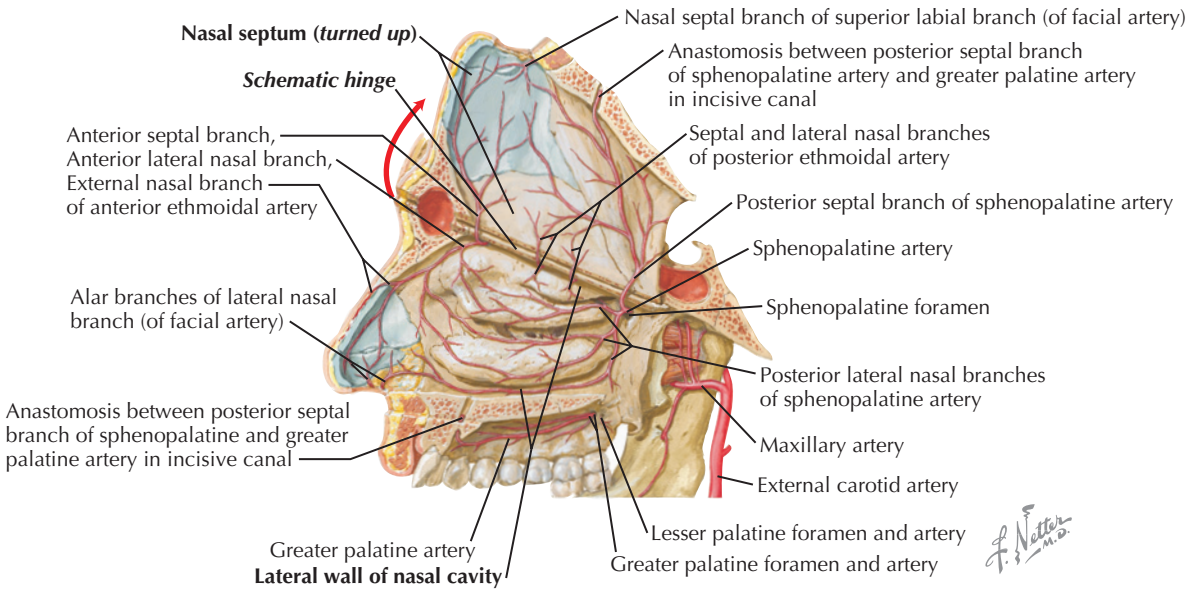


Figure 42-4 Arteries of Nasal Cavity: Nasal Septum Turned Up.

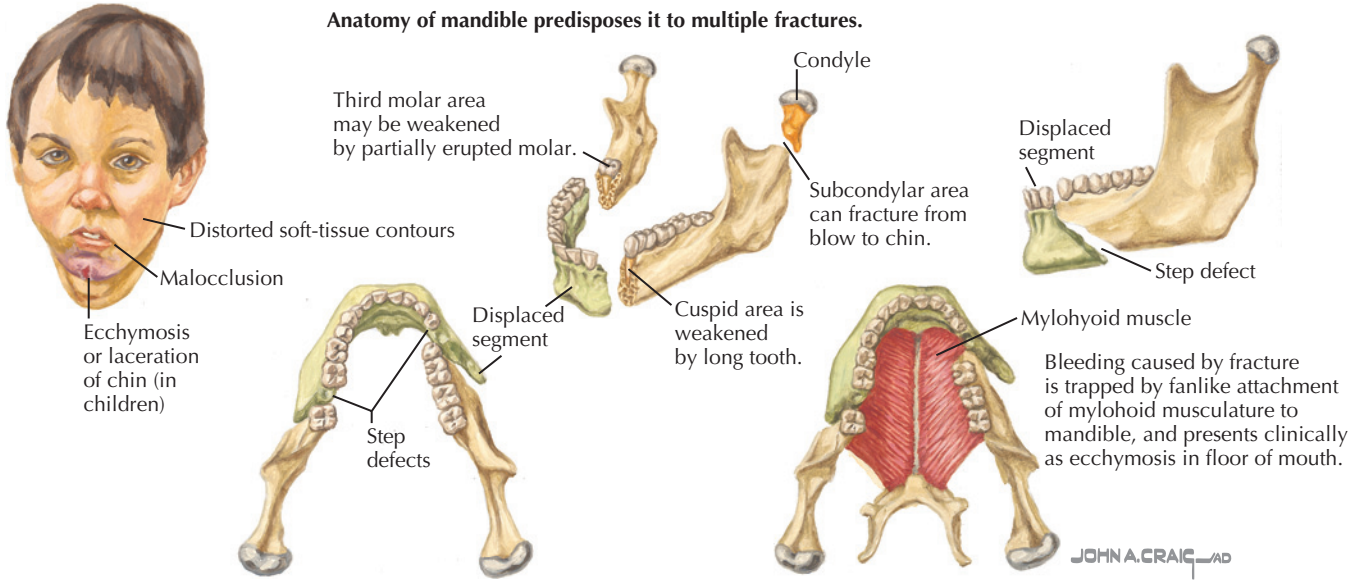


Figure 42-5 Mandibular Fractures.

teeth) or bone plating. With wiring, the jaw is immobilized for 4 to 6 weeks; with bone plating, can move jaw immediately but chewing is limited for approximately 4 weeks. With bone plating, airway issues, oral hygiene, and diet are less problematic. Infection, unfortunately, is not uncommon.

Rehabilitation: May try therapy after jaw wiring to improve jaw opening.

Return to play: Return to noncontact sports in 4 weeks; contact sports in 2 to 3 months.

Maxillary Fractures

Mechanism of injury: Usually secondary to trauma directly to midface from the usual culprits—balls, bats, pucks, sticks. Classified using Le Fort scheme (Fig. 42-6). Injuries may include combination of the classic Le Fort schema. May see associated brain, cervical, or ocular injuries (see Chapters 39, 40, and 41).

Examination: Signs and symptoms include asymmetry or altered contour of face, flattening of the midface; midface is mobile when applying a force directed anteriorly to the anterior maxillary alveolus; epistaxis; malocclusion and loose dentition; and diplopia or globe malposition. Document function of all cranial nerves. Injury to the infraorbital nerve common in midface fractures; resulting in altered sensation of cheek and upper lip.

Radiology: Waters' view, CT scan with coronal and axial imaging most used, or MRI.

Treatment: Secure airway. If athlete is able, a forward sitting position will allow blood to drain outside the body and may facilitate mouth breathing. Nasotracheal airway contraindicated without fiber-optic placement in these injuries. Most surgeons advocate open reduction with internal fixation with titanium miniplates and microplates as soon as medically prudent based on other injuries. Can treat Le Fort I fractures with intermaxillary fixation (wiring).

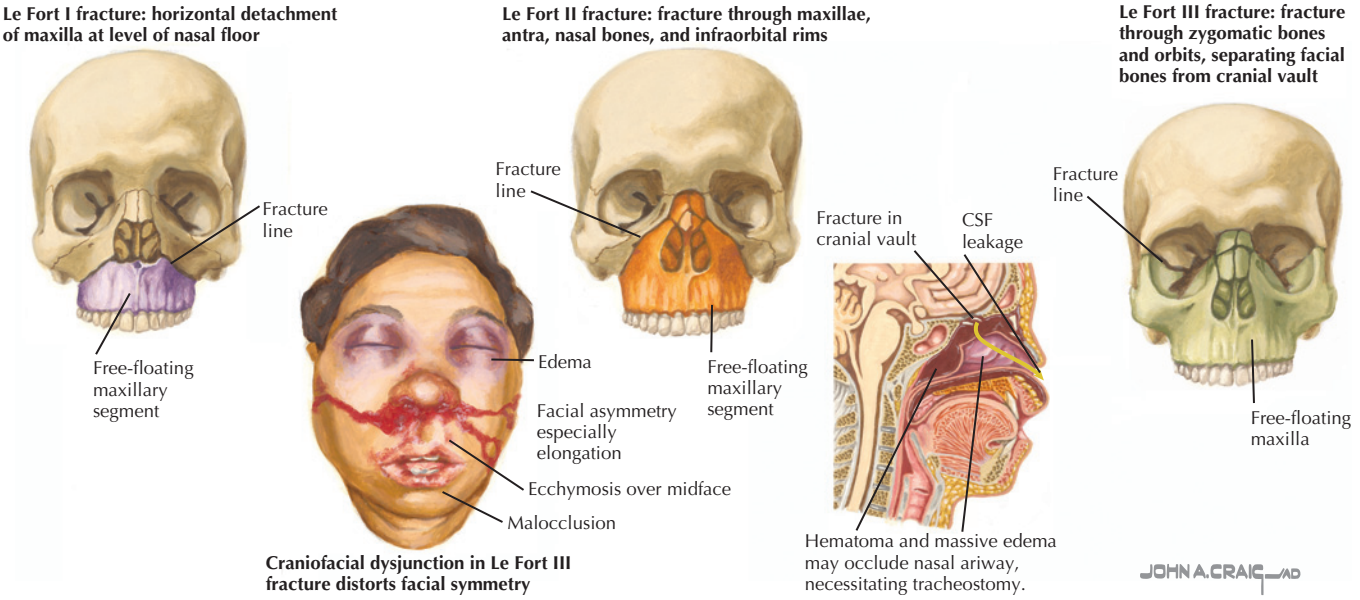


Figure 42-6 Midface Fractures: Le Fort.

Zygoma Fractures

Mechanism of injury: Usually from direct force to cheek. Often associated with orbital floor fracture. Can affect root of maxillary teeth. Affects the anterior wall of the maxillary sinus. Also known as tripod fractures, tetrapod fractures, and malar complex fractures.

Examination: Signs and symptoms include:

- Facial asymmetry
  - Flattening of malar eminence of the involved side. Masseter muscle pulls the malar eminence inferiorly. May not be apparent until swelling goes down after a few days.
  - Enophthalmos (sinking of the eyeball into the orbital cavity) if orbital floor is sufficiently damaged.
- Diplopia if inferior rectus or inferior oblique muscles are entrapped indicating orbital floor fracture.
- May see pain with mastication. If the zygomatic arch is depressed, the action of the temporalis muscle may cause pain with chewing or opening the mouth.
- Numbness of affected cheek.
- Hallmark of zygoma fracture is the triad of bloody nasal drainage, subconjunctival hemorrhage, and paresthesia of the ipsilateral infraorbital nerve (Fig. 42-7).

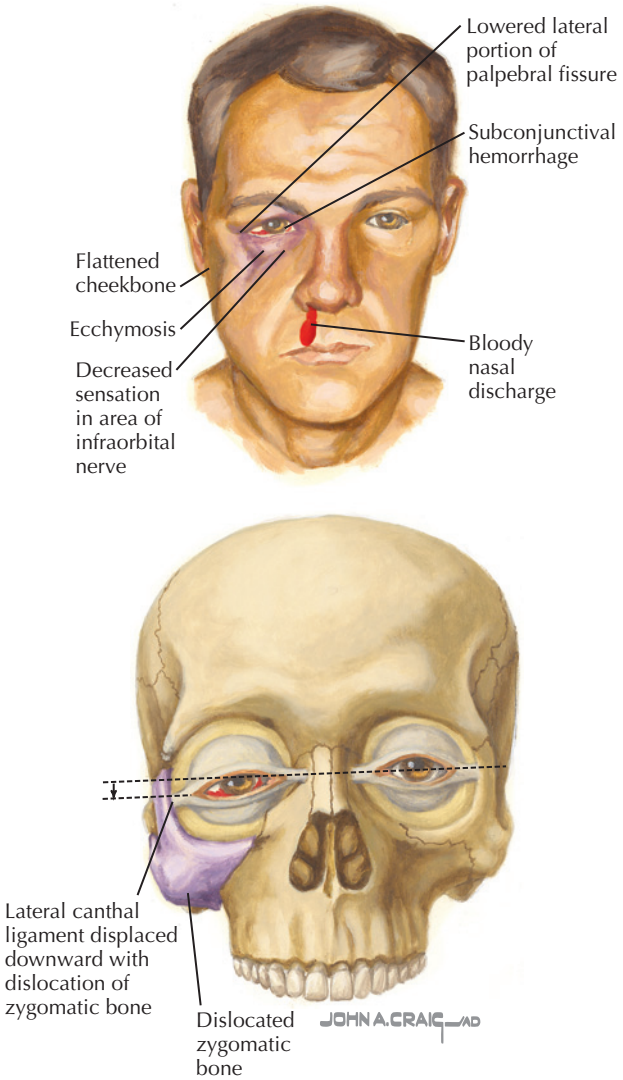


Figure 42-7 Zygomatic Fractures.

Imaging: X-rays—Caldwell, Waters', and submentovertex views will identify most fractures except orbital fractures. CT scans with axial and coronal imaging usually sufficient for surgeon to plan treatment.

Treatment: If less than 2 mm of displacement, can usually be treated nonsurgically. However, cosmetic deformity can become apparent when facial swelling diminishes. Surgical fixation after reduction is often performed 3 to 7 days after injury. Athletes with nonsurgically treated fractures should use a soft diet for 1 to 2 weeks to minimize tension on fracture from masseter muscle; those with surgically treated fractures can use soft diet for 1 week.

Return to play: Return to noncontact sports in 3 to 4 weeks and contact sports in 6 to 8 weeks.

Frontal Sinus Fracture

Mechanism of injury: Direct trauma to sinus. Uncommon fracture. May see in some sports played without helmet. More common in adults because pediatric population may not have developed sinuses. Anterior wall of sinus is strongest portion of sinus.

Examination: Signs and symptoms include frontal headache, epistaxis, forehead numbness from supraorbital nerve injury, anosmia (alteration in sense of smell) if associated fracture of anterior fossa floor, cerebrospinal fluid leakage if posterior wall of frontal sinus fractured, and depression of frontal area of skull can be seen and palpated (swelling may make this difficult).

Imaging: CT imaging of face and head delineates extent of injury.

Treatment: Referral to a maxillofacial surgeon is indicated. Nondisplaced fractures limited to the anterior wall of the frontal sinus may be treated nonsurgically. Most frontal sinus fractures need to be surgically explored. This is done to prevent complications such as deformity, damage to the frontonasal duct, and mucocele formation. In fractures of the posterior wall, one needs to be aware of the possibility of dura damage and possible route of infection to the meninges and brain.

Orbital and Ophthalmic Injuries

See Chapter 41, Eye Injuries in Sports.

RECOMMENDED READINGS

1. Andreasen JO, Andreasen FM, Andersson L: Textbook and Color Atlas of Traumatic Injuries to the Teeth, 4th ed. Blackwell.
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4. Lee KJ: Essential Otolaryngology. Stamford, Conn: Appleton & Lange, 1999.
5. McTigue KJ: Evaluation and management of dental injuries in children. Up To Date 2007.
6. Romeo SJ et al: Sideline management of facial injuries. Curr Sports Med Rep 155-161, 2007.

Shoulder Injuries

43

Kevin M. Honig and Eric C. McCarty

HISTORY

- A careful history will help establish the diagnosis and formulate a treatment plan.
- Important factors include the chief complaint, mechanism of injury, hand dominance, what sport the athlete plays, and prior treatments.
- Common complaints are "pain with overhead activities," "pain at night when I lie on that side," "a feeling of the shoulder coming out of the joint."

PHYSICAL EXAMINATION

Evaluation of shoulder pathology should include an examination of the cervical spine to rule out referred pain.

Inspection

- Important to be able to visualize the entire shoulder during exam and compare to the unaffected side.
- Assess for muscle atrophy, which can indicate neurologic dysfunction or chronic injury such as a rotator cuff tear.
- Look for scapular dyskinesia or winging.
- Note any prominence that could represent an acromioclavicular separation, clavicle fracture, sternoclavicular subluxation, etc.

Palpation

- Should include bony landmarks for tenderness or crepitus—acromioclavicular joint, clavicle, sternoclavicular joint, greater tuberosity, and coracoid.
- Extending and internally rotating the arm delivers the greater tuberosity from under the acromion.

Motion

- Important to compare active and passive range of motion to the unaffected side. Forward flexion, abduction, and external rotation are measured in degrees from neutral rotation. Internal rotation is measured in relation to the spinal level that can be reached posteriorly (Fig. 43-1).
- Does motion cause pain or produce feeling of instability?
- The normal ratio of glenohumeral to scapulothoracic motion is 2:1 (see Fig. 43-1).

Manual Muscle Testing

Supraspinatus: (Jobe test) Resistance is applied with the patient's arm abducted 90 degrees, forward flexed 30 degrees, and internally rotated (thumb pointing down) (see Fig. 43-1).

Infraspinatus/Teres Minor: Resistance to external rotation with the arm at the patient's side and elbow flexed 90 degrees preferentially tests the infraspinatus. Resistance to external rotation with the arm abducted and elbow flexed 90 degrees tests both the infraspinatus and teres minor (see Fig. 43-1).

Subscapularis: Resistance to internal rotation with the arm at the patient's side and elbow flexed 90 degrees (see Fig. 43-1).

SPECIFIC TESTS

Impingement

Positive tests produce pain at the anterior or lateral aspect of the shoulder.

Hawkins' test: The arm is passively forward flexed to 90 degrees and then forcibly internally rotated (Fig. 43-2).

Neer's sign: The patient's arm, with the forearm pronated, is passively forward flexed while the scapula is stabilized (see Fig. 43-2).

Rotator Cuff Tear

Jobe test: Isolates the **supraspinatus** (see earlier discussion).

Drop arm sign: The patient fully elevates the arm in the plane of the scapula and then tries to lower it slowly. Sudden dropping of the arm or pain while doing so suggests a rotator cuff tear.

External rotation lag sign: With the arm adducted to the side and elbow flexed 90 degrees, the arm is passively brought into maximal external rotation. Inability of the patient to actively maintain the arm in the externally rotated position indicates a **massive tear** involving the **infraspinatus.**

Lift-off test: The dorsum of the patient's hand is placed against the lumbar spine (see Fig. 43-2). The patient then lifts the hand away from the back, maintaining the elbow in the coronal plane. Inability to do so indicates a **subscapularis** tear.

Belly-press test: The patient places both hands on the abdomen, internally rotates to bring the elbows forward beyond the coronal plane, and then presses the hands into the abdomen. Inability to keep the elbow beyond the coronal plane indicates a **subscapularis** tear (see Fig. 43-2).

Biceps Tendon Pathology

Speed's test: With the forearm supinated and elbow extended, the patient forward flexes the arm against resistance. A positive test produces pain in the anterior shoulder (see Fig. 43-2).

Yergason's test: With the elbow in 90 degrees of flexion, the patient supinates the forearm against a resistive force. A positive test produces pain in the biceps region (see Fig. 43-2).

Instability

It is important to perform these tests on the contralateral extremity for comparison and assessment of the patient's normal laxity.

Anterior

Apprehension test: Best performed supine. The arm is passively abducted to 90 degrees and then progressively externally rotated while the patient's response is noted. A positive test produces a patient response of "apprehension" by reproducing the patient's symptoms of anterior instability (see Fig. 43-2).

Relocation test: A posteriorly directed force is applied to the proximal humerus while performing the **apprehension test.** The test is positive when the patient's "apprehension" is relieved and greater external rotation can be achieved (see Fig. 43-2). **Note:** If the **apprehension test** produces pain (as opposed to a feeling of instability) that is relieved by the **relocation test,** it is suggestive of **internal impingement.**

Load and shift test: Can be performed in the supine or upright position. The humeral head is loaded to center it within the glenoid. It is then translated anteriorly and posteriorly (see Fig. 43-2). Amount of translation graded as follows: grade 0—minimal, grade I—up to the rim of the glenoid, grade II—over the glenoid rim but spontaneously reduces, or grade III—over the glenoid rim and does not spontaneously reduce.

Posterior

Load and shift test: See previous discussion.

Posterior stress test: Performed supine; the arm is flexed to 90 degrees and internally rotated. A posteriorly directed force is then applied to the humerus. A positive test causes subluxation.

Jerk test: Performed upright; the arm and elbow are flexed 90 degrees. The arm is internally rotated and the humerus is loaded posteriorly. A positive test can cause posterior sublux-

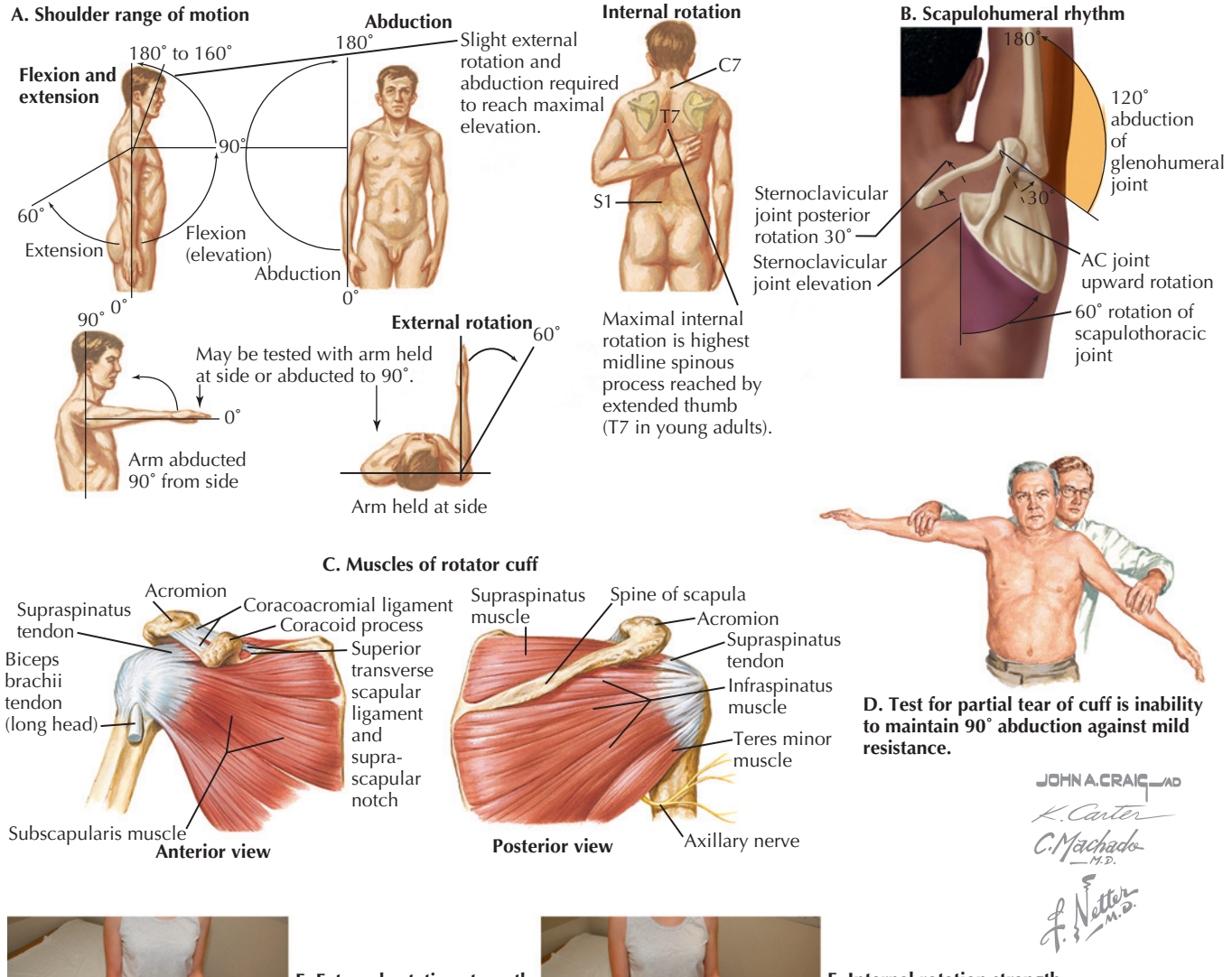


Figure 43-1 Physical Examination of Shoulder.

ation of the humeral head that is then reduced with a "jerk" when extending the arm (see Fig. 43-2).

Multidirectional

Sulcus sign: Traction is applied to the arm in an inferior direction by grasping the elbow while observing the area lateral to the acromion for a "sulcus." Presence of a sulcus indicates **inferior** laxity. Presence of a sulcus when repeating the exam maneuver in some external rotation indicates involvement of the rotator interval.

Labrum

Clunk test: With the patient supine, the arm is fully abducted and the examiner's hand is placed on the posterior aspect of the humeral head. An anterior force is applied to the humerus while the other hand rotates the humerus. Positive findings include a "clunk," pain, and grinding.

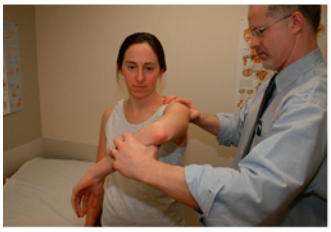
O'Brien's test (active compression test): The arm is positioned in 90 degrees of flexion and 10 to 15 degrees of adduction. A downward force is applied by the examiner as the patient resists, first with the arm internally rotated (thumb down) and then with the arm externally rotated (thumb up). A positive test causes pain felt deep within the joint, which is reduced or relieved with the arm externally rotated (see Fig. 43-2).

Biceps load test: Performed supine. The arm is abducted 120 degrees, maximally externally rotated, the forearm supinated, and the elbow flexed 90 degrees. Active elbow flexion is then performed against resistance. A positive test produces pain suggestive of a superior labrum from anterior to posterior (SLAP) tear.

Anterior slide test: The patient's hands are placed on the hips with the thumbs pointing anteriorly. An axial load is applied at the elbow toward the glenohumeral joint against patient resistance. A positive test produces pain suggestive of a SLAP tear.

Acromioclavicular Joint

Direct palpation over the acromioclavicular (AC) joint causes pain.

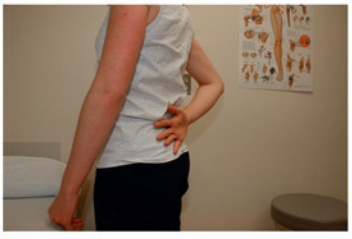


A. Hawkins' test. The arm is passively forward flexed to 90° and then forcibly internally rotated.

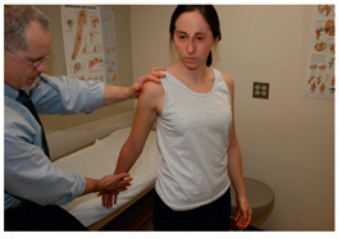
B. Neer's impingement sign. Performed by passively bringing the patient's arm into full forward flexion with the forearm pronated.

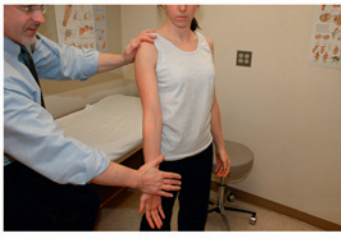
C. Lift-off test (no resistance). The dorsum of the patient's hand is placed against the lumbar spine. The patient then lifts the hand away from the back maintaining the elbow in the coronal plane.

D. Belly press. The patient places a hand on the abdomen, internally rotates to bring the elbow forward beyond the coronal plane, and then presses the hand into the abdomen. Inability to keep the elbow beyond the coronal plane or the hand on the abdomen against resistance indicates a **subscapularis** tear.

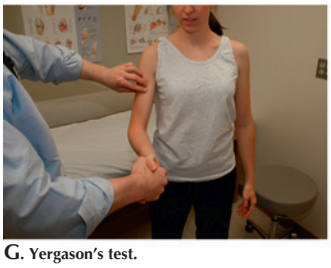


E. Positive subscap tests.

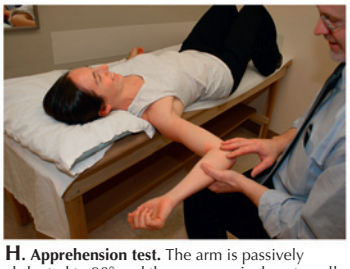




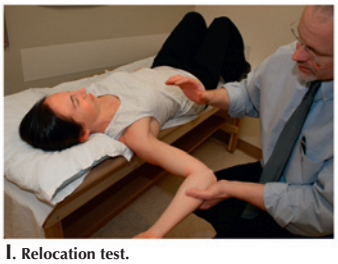
F. Speeds.

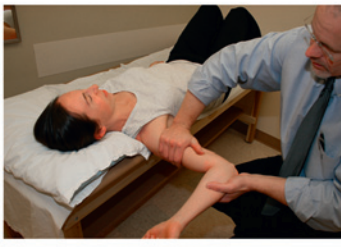


G. Yergason's test.

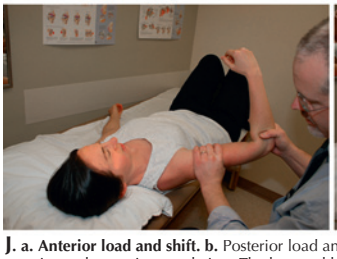


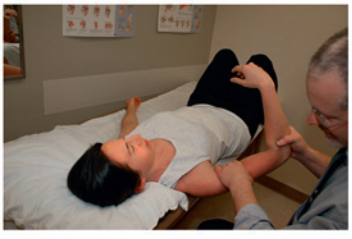
H. Apprehension test. The arm is passively abducted to 90° and then progressively externally rotated while noting the patient's response. A positive test produces a patient response of "apprehension" by reproducing their symptoms of anterior instability.



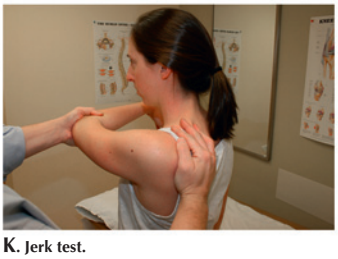


I. Relocation test.





J. a. Anterior load and shift. b. Posterior load and shift. **Load shift.** The load and shift judges anterior and posterior translation. The humeral head is loaded to center it within the glenoid. It is then translated anteriorly and posteriorly.



K. Jerk test.

L. a. Internal rotation. b. External rotation. O'Brien's test. The arm is positioned in 90° of flexion and 10–15° of adduction. A downward force is applied by the examiner as the patient resists, first with the arm internally rotated (thumb down) and then with the arm externally rotated (thumb up).

M. Cross arm adduction for AC joint. Arm is flexed 90° and then adducted across chest.

Figure 43-2 Special Tests.

Cross-arm adduction test: The arm is flexed 90 degrees and then adducted across chest. A positive test causes pain at the AC joint (see Fig. 43-2).

O'Brien's test: Can cause pain localized to the AC joint and should be distinguished from pain deep within the shoulder.

IMAGING OF THE SHOULDER

X-rays

AP: Taken in plane of thorax. Provides oblique view of the glenohumeral joint because of the anteverted position of the scapula on the posterolateral aspect of the thoracic cage.

True AP (Grashey view): Taken in plane of scapula. Provides true anteroposterior (AP) view of glenohumeral (GH) joint by angling beam approximately 45 degrees in medial-to-lateral direction or by rotating patient and placing scapula flat on x-ray cassette.
Axillary view: Important for evaluating dislocations. Useful for evaluating fractures of the coracoid and large fractures of the anterior or posterior glenoid rim. Will reveal an os acromiale.
West Point axillary lateral: Provides a tangential view to the anteriorinferior glenoid rim useful for evaluating instability cases.
Scapular Y view: Lateral x-ray taken in plane of the scapula. Helpful in evaluating relationship of humerus to glenoid fossa for dislocations. Tilting the beam 10 degrees caudal produces a supraspinatus **outlet view,** which allows assessment of acromion morphology.
Stryker notch view: Evaluates compression fractures or defects in the posterolateral aspect of the humeral head (Hill-Sachs lesion).
Serendipity view: A 40-degree cephalic tilt view for visualization of the sternoclavicular joint.
Zanca view: Provides clear view of AC joint by directing x-ray beam 10 to 15 degrees cephalad. Allows assessment of AC separations, distal clavicular osteolysis, and distal clavicle fractures.

MRI

Magnetic resonance imaging (MRI) is the gold standard for evaluating soft tissue structures and cartilage; magnetic resonance arthrogram (MRA) preferred for labral pathology.

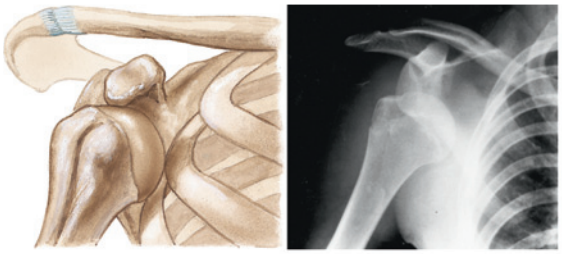
SPECIFIC SHOULDER INJURIES

Instability

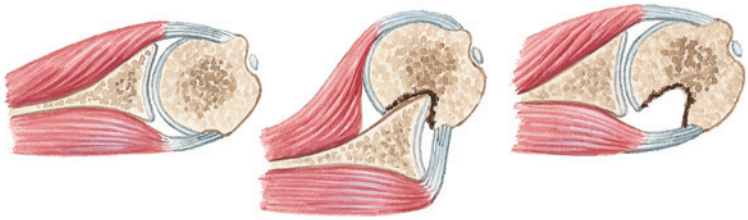
Defined as symptomatic, abnormal translation of the humeral head on the glenoid. Instability can be classified in many ways—*direction of instability* (**anterior, posterior, multidirectional**), *traumatic versus atraumatic*, and *degree of instability*.

Anterior Glenohumeral Instability

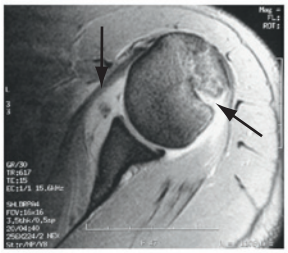
Description: The most common direction of instability.
Mechanism of injury: Direct or indirect trauma leading to tearing and attenuation of the anterior capsulolabral complex. Direct trauma involves a blow to the posterior shoulder. Indirect trauma involves injury to the arm in a position of abduction, extension, and external rotation position.
Presentation: Can present with pain, feeling of weakness, feeling of instability, or recurrent dislocations. Dislocations in younger patients are associated with *bankart lesions*, whereas patients over 40 years of age typically have associated rotator cuff tears.
Physical exam: In acute dislocations the lateral shoulder will lose its normal contour and a fullness will be present anteriorly. The arm is held in slight abduction and in external rotation. A careful neurovascular exam is important. The nerve most commonly injured is the axillary nerve (Fig. 43-3). The following exam maneuvers will be positive in patients with recurrent anterior instability: **apprehension test, relocation test, load and shift test.**
Differential diagnosis: Multidirectional instability (MDI), rotator cuff tear, SLAP lesion, proximal humerus fracture.
Diagnostics:
- **X-rays:** A standard shoulder series (AP, axillary, scapular-Y) to ensure the humeral head is reduced. Hill-Sachs lesions can also be detected (see Fig. 43-3). The **West-Point axillary view** will provide better evaluation of the anterior glenoid rim for possible *bony Bankart lesions*.
- **MRI or MRA:** Will demonstrate *Bankart lesions* and anterior capsule pathology (see Fig. 43-3).



A. Anterior shoulder dislocation.

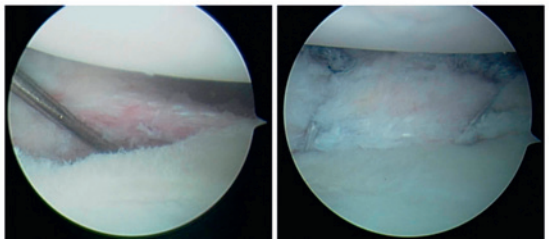


B. Anterior humeral dislocation with resulting Hill-Sachs deformity.



C. MRI Bankart and Hill-Sachs.

D. Reduction maneuvers for anterior shoulder dislocation.



E. Arthroscopic images of Bankart.

Figure 43-3 Anterior Glenohumeral Instability.

Treatment:

- Acute anterior dislocations require reduction. Several reduction methods have been described. The Stimson technique is a relatively atraumatic technique. The patient is placed prone and weights are placed on the affected wrist (see Fig. 43-3). A variation of this technique can be done on the field by placing the athlete supine and applying traction on the wrist in forward flexion and counter traction on the chest (see Fig. 43-3). Intra-articular injection of local anesthetic has been shown to be effective in aiding reduction.
- The value of traditional postreduction treatment of sling immobilization is controversial. There is no consensus in the literature on the use of a sling or the period of immobilization. Recent literature suggests that immobilization in external rotation reduces the incidence of recurrent anterior instability.
- Initial treatment is physical therapy focusing on strengthening the dynamic stabilizers of the GH joint (rotator cuff muscles, deltoid and scapula stabilizers) and maintaining glenohumeral motion.
- Recurrent instability should be treated surgically with an anterior stabilization. Current arthroscopic techniques have equivalent results to open techniques (see Fig. 43-3). Bony deficiencies of the anterior glenoid or large, engaging Hill-Sachs lesions need to be addressed for a successful outcome.

Prognosis and return to play: Traditionally, the redislocation rate is approximately 90% in patients younger than age 20 and decreases with increasing age. The decision to treat initially with surgical stabilization versus nonoperative therapy must take into account several factors—patient age, activity level, sport, etc. Return to play after nonoperative therapy requires near normal range of motion, strength, and functional ability. Return to play after surgical stabilization is typically 4 to 6 months.

Posterior Glenohumeral Instability

Description: Tear or stretching of posterior capsulolabral structures leading to dislocation or subluxation.

Mechanism of injury: Posterior subluxation or dislocation can result from a traumatic event with the arm in a position of flexion, adduction, and internal rotation causing a *reverse Bankart lesion* (Fig. 43-4). Or it can result from repetitive trauma causing a labral tear or capsular attenuation. This mechanism is commonly associated with an offensive lineman in football jamming his opponents while blocking.

Presentation: A posterior dislocation is rare compared to an anterior dislocation. However, posterior dislocations are easily missed. Patients presenting after a seizure or electrical shock should raise suspicion of a posterior dislocation. More commonly, posterior instability presents as pain or a feeling of instability posteriorly with a load to the arm in a position of forward flexion, adduction, and internal rotation.

Physical exam: In acute posterior dislocations, the arm is held in adduction and internal rotation. A prominent coracoid anteriorly and a posterior fullness are present. The following maneuvers will be positive in patients with recurrent posterior instability: **load and shift test, posterior stress test, jerk test.**

Differential diagnosis: MDI, rotator cuff tear, SLAP tear, proximal humerus fracture.

Diagnostics:

- **X-rays:** A standard shoulder series (AP, scapular-Y, and axillary view) to ensure the humeral head is reduced. Plain films will also reveal reverse Hill-Sachs lesions indicative of a posterior dislocation and allow for evaluation of bony contributions to posterior instability such as glenoid fractures, hypoplasia, or excessive retroversion (see Fig. 43-4).
- **CT:** Better for assessment of glenoid deformities contributing to instability.
- **MRI or MRA:** Will demonstrate *reverse Bankart lesions* or a redundant, attenuated posterior capsule.

Treatment:

- Acute posterior dislocations require reduction. This is often more difficult than reduction of an anterior dislocation. With the patient supine, traction is applied in line with the deformity while the humeral head is guided into the joint. External rotation is avoided to prevent proximal humerus fracture.
- For recurrent posterior instability, the majority will improve with physical therapy focusing on dynamic stabilizers of the shoulder, particularly the posterior deltoid and external rotators. Surgery is indicated for patients who fail nonoperative treatment. The surgery is directed at the offending pathology. Capsular plication for attenuated capsule and repair of reverse Bankart lesions. Bony abnormalities of the glenoid need to be identified and addressed if present.

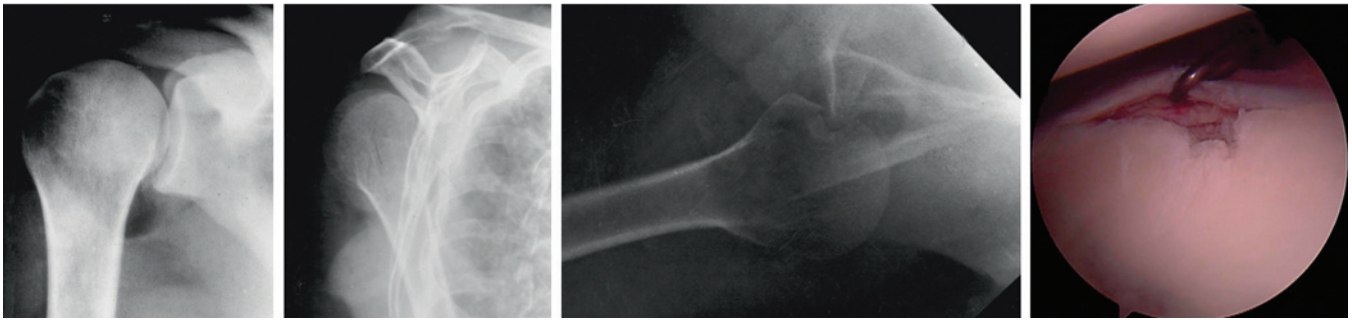
Prognosis and return to play: Criteria are similar to those for anterior instability.

Multidirectional Instability (MDI)

Description: Symptomatic instability in more than one direction—inferior plus anterior or posterior.

Mechanism of injury: Often atraumatic in the setting of generalized laxity or from repetitive microtrauma in athletes such as swimmers, volleyball players, etc. The primary pathology is an attenuated inferior capsule.

Presentation: The majority are young adults. Injury often is bilateral. Patients present with pain, instability, and sometimes transient neurologic symptoms. Symptoms when carrying heavy



A. Anteroposterior radiograph. Difficult to determine if humeral head within, anterior, or posterior to glenoid cavity.

B. Lateral radiograph (parallel to plane of body of scapula). Humeral head clearly seen to be posterior to glenoid cavity.

C. True axillary view. Also shows humeral head posterior to glenoid cavity.

D. Posterior labral tear.

Figure 43-4 Posterior Glenohumeral Instability.

objects at one's side is indicative of instability in the inferior direction.

Physical exam: Look for signs of generalized laxity, such as hyperextension of the elbows or ability to bring the thumb to the forearm. A positive **sulcus sign** indicates inferior instability. Apply the tests described earlier to evaluate for anterior and posterior instability.

Differential diagnosis: Unidirectional instability, rotator cuff tear, SLAP lesion.

Diagnostics:

- **X-rays:** Usually are normal but may reveal a Hill-Sachs or bony Bankart lesion.
- **MRA:** Can demonstrate excessive capsular volume and, similar to unidirectional instability, it will demonstrate Bankart lesions.

Treatment: The majority respond to physical therapy to strengthen the dynamic stabilizers of the shoulder. Those that fail nonoperative treatment are candidates for surgical stabilization via open capsular shifts or arthroscopic capsular plications.

Prognosis and return to play: Even in the group requiring surgical stabilization, the majority return to a competitive level. The time frame for return to play after surgery is similar to unidirectional instability.

Biceps Tendon Pathology

Tendonitis

Description: *Primary* tendonitis is an isolated inflammatory condition of the proximal biceps tendon in the intertubercular groove. More commonly, it occurs as a *secondary* process in conjunction with pathologic changes to surrounding structures in the shoulder such as rotator cuff impingement or tearing. This results in fraying or degeneration of the proximal biceps tendon.

Mechanism of injury: Overuse injury causing repetitive trauma to the tendon of the long head of the biceps.

Presentation: Pain in the anterior aspect of the shoulder that may radiate down the biceps. Usually no history of trauma.

Physical exam:

- Tenderness anteriorly over the bicipital groove.
- Speed's test is positive.
- Yergason's test is positive.
- Because of its association with impingement, Hawkins' and Neer's tests are often positive.

Differential diagnosis: Rotator cuff pathology, labral tear.

Diagnostics:

- X-rays are normal in primary bicipital tendonitis. They may show an acromial spur suggestive of impingement associated with secondary bicipital tendonitis.
- MRI will show edema in or around the tendon, or a thickened tendon.

Treatment: Begins with nonoperative therapy consisting of rest and nonsteroidal anti-inflammatory drugs (NSAIDs), followed by range of motion exercises. Corticosteroid injections have utility in the bicipital sheath (done via ultrasound guidance) or in the intra-articular space or subacromial space (especially for secondary tendonitis). Surgery is indicated for those who fail conservative therapy. Surgical options include tendon debridement, release of the synovial sheath, tenotomy, and tenodesis.

Prognosis and return to play: Once pain has resolved enough to allow near normal range of motion and strength.

Instability/Subluxation

Description: The long head of the biceps tendon subluxates out of the bicipital groove.

Mechanism of injury: Invariably associated with complete or partial tear of the subscapularis and structures of the rotator interval that comprise a pulley system.

Presentation: Similar to bicipital tendonitis. Patients may also report popping during shoulder motion.

Physical exam: Similar to bicipital tendinitis.

Differential diagnosis: Bicipital tendonitis, rotator cuff pathology.

Diagnostics: X-rays will be normal. MRI will reveal injury to the subscapularis and dislocation of the proximal biceps tendon from the bicipital groove if present.

Treatment: Conservative therapy is similar to that for bicipital tendonitis. Surgical intervention (tenotomy or tenodesis) is more appropriate as primary treatment in the young, active patient or in those who fail conservative therapy. It is important to address associated cuff pathology as well.

Prognosis and return to play: Those undergoing tenotomy can return to play when near normal strength and range of motion has returned. Tenodesis procedures will require 4 to 6 months to allow for adequate healing and rehabilitation.

Rupture

Description: Disruption of the long head of the biceps tendon.

Mechanism of injury: Most commonly caused by forceful elbow flexion against resistance.

Presentation: In acute, traumatic ruptures, patients present with pain and ecchymosis anteriorly. Ruptures in older patients are often the result of attritional degeneration. These patients are often unaware that they have sustained a rupture.

Physical exam: Acutely, patients will be tender over the anterior shoulder and upper arm. Swelling and ecchymosis will be present. The classic "Popeye" deformity will be present because the biceps will be more prominent in the middle of the arm (Fig. 43-5). Patients may have a slight decrease in elbow flexion and forearm supination strength.

Differential diagnosis: Proximal humerus fracture, bicipital tendonitis/instability/subluxation, rotator cuff tear, pectoralis tear.

Diagnostics: X-rays are unremarkable. MRI will confirm the diagnosis.

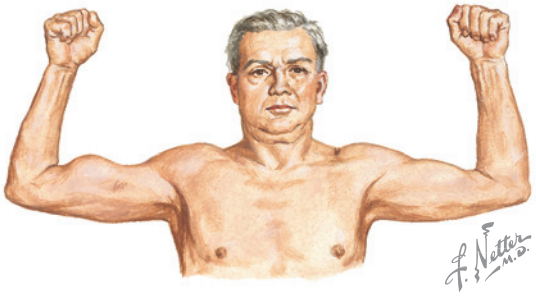
Treatment: Pain control and therapy to maintain motion in the elderly. Surgical tenodesis may be more appropriate for young, active patients or those concerned with the cosmesis of the "Popeye" deformity.

Prognosis and return to play: Same as discussed earlier for tenotomy and tenodesis.

SLAP Lesion

Description: The superior labrum anterior and posterior injury (SLAP) injury was first defined by Snyder in 1990 to describe injuries to the superior glenoid labrum.

Mechanism of injury: Many are atraumatic with an insidious onset of pain. Traumatic mechanisms include traction, compression, and direct blow injuries. Traction injuries occur in overhead-throwing athletes or in a sudden pull in an inferior or superior direction, such as catching oneself from falling. Com-



Rupture of tendon of long head of right biceps brachii muscle indicated by active flexion of elbow.

Figure 43-5 Rupture of Long Head Biceps Brachii Muscle.

pression injury occurs when falling onto an outstretched slightly abducted arm.

Classification: The original classification by Snyder described four types of lesions. Many additional types have since been added. The original four types are described below. Type II lesions are the most common clinically significant lesion (Fig. 43-6).

- **Type I:** Fraying of the superior labrum but the biceps anchor and labrum are still attached.
- **Type II:** The superior labrum and biceps anchor are detached from the superior glenoid. Further classified as anterior, posterior, or combined. Posterior and combined type II SLAPs are often associated with throwing athletes because of the *"peel-back" phenomenon* as the arm is abducted while in an externally rotated position.
- **Type III:** A bucket handle tear of the superior labrum, but the biceps anchor is still attached to the glenoid.
- **Type IV:** The bucket handle tear of the superior labrum extends into the biceps tendon.

Presentation: Complaint is frequently pain especially with overhead activities and decreased function. Patients also report mechanical symptoms of popping, clicking, or catching with motion. The symptoms are usually difficult to distinguish from those associated with impingement or rotator cuff tears.

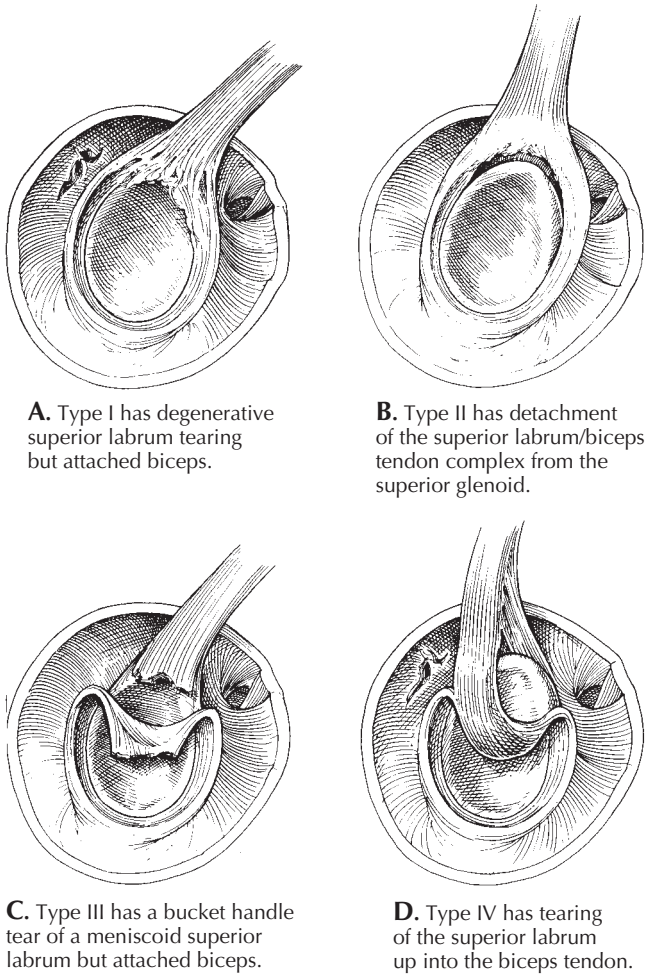
Physical exam: Many provocative exam maneuvers have been described but no one test has proven to be consistently successful in diagnosing a SLAP tear. SLAP lesions are often associated with rotator cuff tears or instability complicating the diagnosis. The following exam maneuvers are considered positive for SLAP lesions if the patient describes pain as deep within the joint: **O'Brien's test, biceps load test, anterior slide test.**

Differential diagnosis: Instability, impingement, rotator cuff tear, glenohumeral arthritis.

Diagnostics: MRI is the best imaging study to evaluate for a SLAP lesion. Great variability in the normal appearance of the superior labrum exists, making interpretation difficult, but presence of a superior paralabral cyst has a high correlation with a SLAP lesion.

Treatment: Nonoperative treatment begins with rest, NSAIDs, and physical therapy to focus on strengthening and stretching because many overhead athletes will have an associated tight posterior capsule and loss of internal rotation. Patients who fail conservative therapy are indicated for surgery. Surgical treatment is dictated by the type of SLAP lesion. In general, types I and III undergo debridement, type II's undergo repair, and type IV's undergo repair or tenodesis depending on degree of biceps tendon involvement.

Prognosis and return to play: Throwing athletes will require 6 to 7 months to fully rehabilitate; nonthrowing athletes can return to sport at 4 months.



A. Type I has degenerative superior labrum tearing but attached biceps.

B. Type II has detachment of the superior labrum/biceps tendon complex from the superior glenoid.

C. Type III has a bucket handle tear of a meniscoid superior labrum but attached biceps.

D. Type IV has tearing of the superior labrum up into the biceps tendon.

Figure 43-6 Superior Labrum Anterior and Posterior (SLAP) Injury. *(Illustration reprinted with permission from DeLee J, Drez D, Miller M: DeLee & Drez's Orthopaedic Sports Medicine: Principles and Practice, 2nd ed. Philadelphia: Saunders, Elsevier, 2002.)*

Rotator Cuff Pathology

Impingement Syndrome

Description: *Impingement syndrome* encompasses a spectrum of pathology including subacromial bursitis, rotator cuff tendinopathy, and partial thickness rotator cuff tears. Three types of impingement entities have been described: **subacromial impingement**, **internal impingement,** and **coracoid impingement.**

Mechanism of injury:

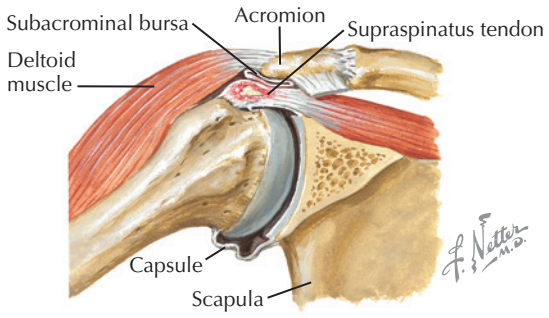
- **Subacromial impingement:** Impingement of the rotator cuff on the undersurface of the acromion and coracoclavicular ligament (Fig. 43-7). This can be the result of acromion morphology, rotator cuff muscle fatigue, degenerative tendinopathy, or AC joint spurring/hypertrophy.
- **Internal impingement:** Particularly in throwers, the articular surface of the rotator cuff comes into contact with the superior glenoid labrum. Although considered anatomically normal, overuse combined with instability or loss of internal rotation can lead to rotator cuff and/or SLAP tears.
- **Coracoid impingement:** Contact between the rotator cuff and a prominent coracoid. The prominence can be idiopathic or iatrogenic (after osteotomy).

Presentation:

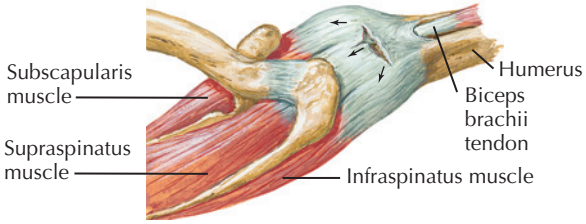
- **Subacromial impingement:** Presents with symptoms typical for rotator cuff pathology. This includes anterolateral shoulder pain that radiates to the lateral arm. Pain is exacerbated by overhead activities. Pain at night and when lying on affected shoulder is very common.
- **Internal impingement:** Posterior-superior shoulder pain or pain described as deep in the joint. Exacerbated by activities that place the arm in an abducted, externally rotated position.
- **Coracoid impingement:** Anterior shoulder pain exacerbated by activities that involve forward flexion and internal rotation.

Physical exam:

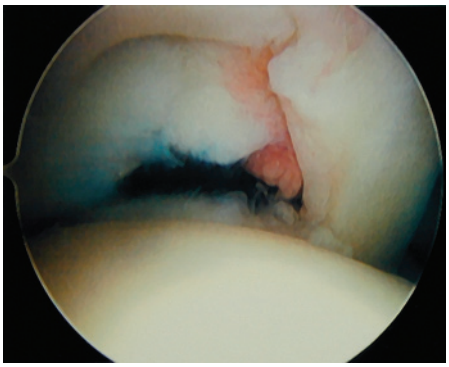
- Neer's impingement sign.
- Hawkins' test.
- Painful arc: The patient actively elevates the arm in the scapular plane and then lowers it in the same plane. Pain during range of motion between 60 and 120 degrees is positive.
- Coracoid impingement sign: The patient's arm is passively placed in a position of forward flexion, adduction, and internal rotation.



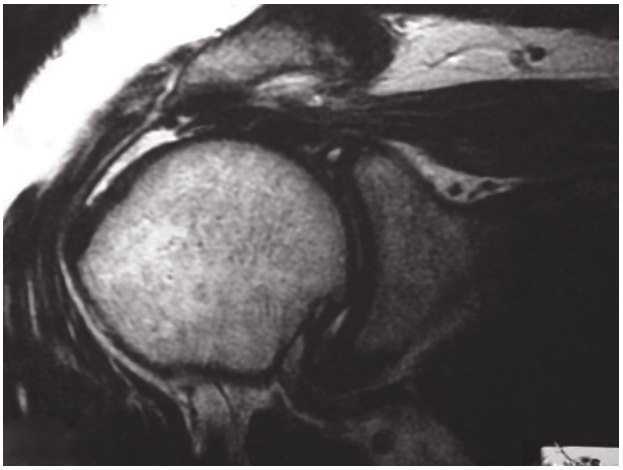
Rotator cuff disease



Acute rupture (superior view). Often associated with splitting tear parallel to tendon fibers. Further retraction results in crescentic defect as shown at right.



Arthroscopic image of small rotator cuff tear as viewed from the joint.



MRI large cuff tear.

Figure 43-7 Rotator Cuff Injury.

- Jobe relocation test: Initially described for anterior instability. Patients with *internal impingement* report posterior-superior shoulder pain with the arm abducted and externally rotated. This pain improves when applying a posteriorly directed force on the humerus, effectively centering the humeral head on the glenoid and reducing the internal impingement.

Differential diagnosis: Instability, GH arthritis, adhesive capsulitis, cervical radiculopathy, calcific tendonitis, AC joint arthritis, thoracic outlet syndrome.

Diagnostics:

- **Neer's test:** A subacromial lidocaine injection is given. Resolution of pain when performing Neer's impingement sign maneuver is a positive test.
- **Coracoid impingement test:** Lidocaine injection just lateral to the coracoid. Resolution of symptoms when performing the coracoid impingement sign maneuver indicates a positive test.
- **X-rays:**
  - AP of the shoulder to rule out GH arthritis.
  - Outlet view allows evaluation of acromion morphology (type I—flat, type II—curved, type III—hooked). Type III acromions are associated with a higher incidence of rotator cuff pathology.
  - Axillary view allows evaluation of the coracoid.
- **MRI:** Will demonstrate rotator cuff tendinopathy, partial or full-thickness rotator cuff tears, and subacromial bursitis.
- **Coracohumeral index:** The distance between the coracoid and humerus can be measured on a computed tomography (CT) scan or MRI. Normal is 8.6 mm.

Treatment:

- NSAIDs, and physical therapy to strengthen the rotator cuff and scapular stabilizers. Subacromial corticosteroid injections are used in subacromial impingement.
- The majority of patients improve with conservative therapy, but those who fail may be indicated for surgical decompression. *Subcoaracoid decompression* involves a partial resection of the coracoid. *Subacromial decompression* involves a bursectomy and acromioplasty. Surgical treatment for *internal impingement* is directed at the SLAP lesion or rotator cuff tear as necessary.

Prognosis and return to play: Athletes involved in overhead activities can return to sports once pain has resolved enough to allow for normal range of motion and near normal strength.

Rotator Cuff Tear

Description: Disruption of the tendon or tendons of the rotator cuff muscles. The supraspinatus tendon is the most commonly involved (see Fig. 43-7).

Mechanism of injury: Tears can occur acutely from direct or indirect trauma. Alternatively, chronic tears can be the result of long standing tendinopathy that eventually progresses to a tear.

Presentation: Similar to subacromial impingement, patients present with anterolateral shoulder pain exacerbated by overhead activities. Night pain and pain sleeping on the affected side are common. Weakness may be present depending on the acuity or size of the tear.

Physical exam: Inspection for atrophy of the supraspinatus or infraspinatus, which if present would indicate a more chronic condition. Evaluate active and passive external rotation, internal rotation, and forward flexion. Positive findings during the following exam maneuvers are suggestive of a rotator cuff tear:

- Lift-off test
- Belly-press test
- External rotation (ER) strength at 0 and 90
- Supraspinatus (SS) strength testing
- External rotation lag sign
- Drop-arm sign
- Impingement tests

Differential diagnosis: Impingement, AC joint arthritis, biceps pathology, GH instability, adhesive capsulitis, cervical radiculopathy.

Diagnostics:

- **X-rays:** AP to look for proximal migration of the humeral head, which would indicate a chronic tear.
- **MRI:** Very sensitive and specific for rotator cuff disease. Can distinguish partial and full thickness tears. Other notable findings that can influence treatment plan and prognosis are fatty infiltration and atrophy of the rotator cuff muscles, and degree of tear retraction (see Fig. 43-7).
- **Ultrasound:** Also very sensitive and specific for rotator cuff tears. It is cheaper than MRI but is very technician dependent.

Treatment: The decision making should be individualized and take into account the patient's age and activity level. Nonoperative treatment tends to be less successful in patients who present with a duration of symptoms greater than 1 year and significant weakness. Nonoperative treatment is similar to that for impingement. Patients who fail nonoperative treatment are indicated for surgical repair. Acute traumatic tears are best treated with prompt surgical repair.

Prognosis and return to play: Overhead athletes undergoing surgical repair may require 6 months to 1 year before being able to fully return to sports.

Acromioclavicular Joint Injuries

Sprains/Separations

Description: Involves sequential injury to the acromioclavicular (AC) and coracoclavicular (CC) ligaments. Includes involvement of the deltoid and trapezial muscle and fascia in higher degree injuries.

Classification:

- **Type I:** Sprain of the AC ligament. AC and CC ligaments are intact. X-rays are normal (Fig. 43-8).
- **Type II:** Rupture of the AC ligament. CC ligaments are intact. X-rays will show slight elevation of the clavicle. The AC joint is unstable to exam but a stress x-ray will *not* produce 100% separation.
- **Type III:** Complete Rupture of the AC and CC ligaments with 100% superior displacement of the clavicle.

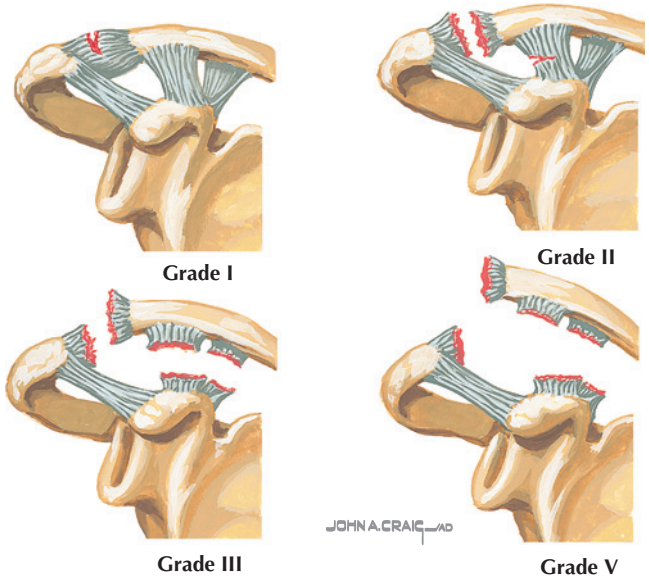


Figure 43-8 Acromioclavicular Dislocation.

- **Type IV:** Complete separation of the AC joint with the clavicle displaced posteriorly through the trapezial fascia.
- **Type V:** More severe Type III with complete rupture of the AC and CC ligaments. Also involves disruption of the trapezial and deltoid fascia off of the acromion and clavicle. The clavicle is displaced superiorly 100-300%.
- **Type VI:** A rare inferior dislocation of the clavicle into a subcoracoid (lodged behind the conjoined tendon) or subacromial position.

Mechanism of Injury: The vast majority are the result of direct trauma by a fall or blow onto the shoulder with the arm adducted (see Fig. 43-8). An indirect mechanism involves falling onto an outstretched hand, which drives the humerus proximally into the acromion.

Presentation: The patient will usually report the above mechanism and complain of pain at the anterior/superior aspect of the shoulder. Common examples include hockey players being checked into the boards, football players taking a blow to the shoulder pads, or a cyclist falling off of a bike.

Physical exam: Tenderness will be present over the AC joint. Prominence of the distal clavicle will be evident in types II, III, and V. Stability of the distal clavicle can be assessed although it may be difficult in the acute setting. The **cross-arm adduction test** will be positive. The **O'Brien test** has been shown to have high specificity for AC sprains if the pain localizes to that region.

Differential diagnosis: Clavicle fracture, distal clavicular osteolysis, shoulder contusion.

Diagnostics: In standard x-rays of the shoulder, the AC joint is overpenetrated and poorly visualized.

- **Zanca view:** See earlier discussion under "Imaging of the Shoulder."
- **Axillary view:** Allows evaluation for anterior or posterior displacement of the clavicle

Treatment:

- Types I and II are treated nonoperatively with a brief period of immobilization in a sling, ice, and analgesics followed by physical therapy. A local anesthetic injection can be used for in-game situations to allow the athlete to resume play. Chronic type II injuries that are symptomatic can be treated surgically with distal clavicle excision and anatomic reconstruction.
- Type III: Treatment is controversial, but most lean toward nonoperative treatment. If the patient has persistent pain or is unable to return to the desired level of activity, then distal clavicle excision and anatomic reconstruction can be performed.
- Types IV, V, and VI are treated surgically.

Prognosis and return to play: Those treated nonoperatively will be able to return to sports in 1 to 6 weeks depending on the severity of the injury and sport played. Those treated surgically will require 4 to 6 months before returning to sports.

Distal Clavicle Osteolysis

Description: A painful condition of the AC joint caused by lysis and resorption of bone of the distal clavicle.

Mechanism of injury: Overuse injury producing repetitive microtrauma to the distal clavicle that leads to bone resorption.

Presentation: It is most common in young males and often bilateral. Frequently associated with weightlifting. Patients present with pain over the anterior/superior aspect of the shoulder. Pain is exacerbated with more demanding activities. Weightlifters are more symptomatic with specific exercises such as bench pressing, dips, and push-ups.

Physical exam: Tenderness over the AC joint will be present. There may be a prominence of the distal clavicle and crepitus with motion. **Cross-arm adduction test** will elicit pain. Stability of the distal clavicle should be assessed because this may impact surgical planning.

Differential diagnosis: Rotator cuff disease, infection, hyperparathyroidism, clavicle fracture, AC separation.

Diagnostics:

- Zanca view reveals osteopenia and expansion of the distal clavicle. Joint space widening and cysts may be present as well.
- Bone scan is sensitive and is useful in cases in which x-ray findings are not obvious.
- A diagnostic injection of local anesthetic into the AC joint is useful in cases in which the diagnosis is unclear.

Treatment: Initial treatment should be nonoperative and consist of modification of activities and weightlifting technique, NSAIDs, and corticosteroid injections. Distal clavicle resection is indicated in patients who fail nonoperative treatment or cannot tolerate the extended course usually required for complete resolution of symptoms.

Prognosis and return to play: Those treated nonoperatively with a local injection or symptomatic treatment can return to sports as tolerated. Those treated surgically will require at least 4 to 6 weeks before returning to sports.

Sternoclavicular (SC) Joint Injuries

Sprains, Subluxations, and Dislocations

Description: Involves injury (ranging from stretching to partial tearing to complete rupture) of the ligaments about the sternoclavicular joint. It is these ligaments (intra-articular disc ligament, extra-articular costoclavicular ligament, capsular ligament, and interclavicular ligament) that provide most of the stability to this very incongruent joint. Acute sprains are classified as mild (stable joint), moderate (joint subluxation), and severe (joint dislocation). Dislocations are further classified according to the direction of dislocation: anterior (more common) or posterior (Fig. 43-9). In the skeletally immature, the injury may be through the medial clavicular physis, which is the last of the long bones in the body to close. Can also present as spontaneous, atraumatic anterior subluxation.

Mechanism of injury: These injuries are the result of direct or indirect forces.

- Indirect forces are more common and are applied to the SC joint from the anterolateral or posterolateral aspects of the shoulder. Compression and rolling forward of the shoulder causes a posterior dislocation whereas compression and rolling backward of the shoulder causes an anterior dislocation. Such mechanisms can be seen in football pile-ons.
- Direct force applied over the anteromedial aspect of the clavicle will result in a posterior dislocation.

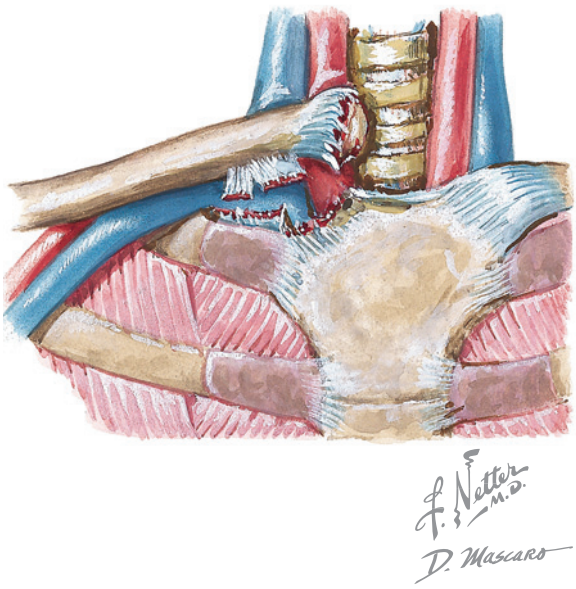


Figure 43-9 Dislocation of Acromioclavicular or Sternoclavicular Joint.

Presentation:

- Acute, traumatic dislocations or physeal fractures present with significant pain and will often be supporting the arm with the uninjured side. Pain is exacerbated by shoulder motion. Dislocations will give an appearance of shortening of the shoulder.
- The majority of spontaneous, atraumatic subluxations are not painful and occur with overhead elevation of the arm. The subluxation reduces when the arm is brought back down. These patients are usually in their teens to 20s and many have generalized ligamentous laxity.

Physical exam:

- Anterior dislocations: Tenderness and swelling exists over a prominent medial end of the clavicle.
- Posterior dislocations: The medial end of the clavicle may not be palpable because it sits posterior to the sternum. Significant swelling anteriorly over the SC joint has been known to mask a posterior dislocation. Patients may have difficulty breathing or swallowing. Venous congestion can be present in the neck or extremity. Compression on the trachea or great vessels can make these injuries a medical emergency.

Differential diagnosis: Sternum fracture, rib fracture, contusion.

Diagnostics:

- **X-rays:** Because of its location and the surrounding anatomy, the SC joint is difficult to image with plain x-ray. Several special views have been developed over time to maximize visualization.
  - Serendipity view (see earlier description under "Imaging of the Shoulder"): Anterior dislocations will appear displaced superiorly to a horizontal line off of the normal clavicle. Posterior dislocations will appear displaced inferiorly.
  - Heinig view: A lateral view centered at the manubrium and shot tangential to the SC joint and parallel to the opposite clavicle.
- **CT scan:** The best imaging modality for evaluating the SC joint.

Treatment:

- Mild sprain: Ice in the first 12 to 24 hours. Sling immobilization for comfort for the first 3 to 4 days followed by progressive range of motion and return to activities.
- Moderate sprain/subluxation: Ice in the first 12 to 24 hours. Figure-of-eight strap to hold shoulders back and reduce SC subluxation. Sling to support upper extremity.
- Dislocation:
  - Anterior: Gentle reduction followed by figure-of-eight strap for 6 weeks and a sling. Anterior dislocations are often unstable after reduction and may re-dislocate after figure-of-eight strap is discontinued. Conservative versus surgical treatment is then based on patient's symptoms and whether or not activities are limited.
  - Posterior: Closed reduction is the treatment of choice for acute injuries. It usually requires general anesthesia and results in a stable SC joint. Appropriate consultants should be available given the associated vascular injuries that have been known to occur. Again, postreduction care includes a figure-of-eight strap and a sling.
- Atraumatic spontaneous subluxation: Symptoms resolve spontaneously. Surgery is not indicated.
- Physeal fracture: Reduction and immobilization similar to dislocations; however, period of immobilization is shorter (3 to 4 weeks) because of fracture healing.

Prognosis and return to play: Patients with mild and moderate sprains can return to sports in 2 to 4 weeks once the pain has resolved and motion has returned. Those with anterior disloca-

tions should be withheld from sports for 6 to 8 weeks. Those with posterior dislocations should be withheld for a longer duration to allow for complete ligament healing, given the potential complications associated with a recurrent posterior dislocation.

Little Leaguer's Shoulder

Description: Growth plate injury (epiphysiolysis) of the proximal humerus.

Mechanism of injury: Caused by overuse and repetitive microtrauma in overhead, skeletally immature athlete. Poor throwing technique can contribute to the cause of injury.

Presentation: Diffuse shoulder pain, worse with throwing or extremes of motion. Usually gradual in onset and may present after a recent increase in throwing activity.

Physical exam: Pain with palpation about the proximal humeral physis and possibly at extremes of shoulder range of motion. Patients may present with external rotation contractures and decreased internal rotation.

Differential diagnosis: Osteochondrosis of the proximal humerus, instability, impingement.

Diagnostics: Plain x-rays of the shoulder including an AP with the arm externally rotated allows for evaluation of the physis. Usually reveals physeal widening but depending on severity may demonstrate metaphyseal fragmentation and periosteal reaction. MRI will also reveal the injury in occult cases not detected on x-ray.

Treatment: Most of these injuries are subtle with minimal displacement. Treatment for these cases begins with cessation of throwing for about 3 months. Then a progressive throwing program should be initiated. Attention should be given to the athlete's throwing mechanics. In cases of significant displacement and/or angulation, treatment is based on the age and growth remaining of the patient.

Prognosis and return to play: Adherence to the treatment protocol of rest followed by a throwing program with proper mechanics will return the vast majority to play without return of symptoms.

Glenohumeral Internal Rotation Deficit (GIRD)

Description: Loss of glenohumeral internal rotation of more than 25 degrees. Overhead athletes will often gain external rotation adaptively to increase velocity. The gain in external rotation usually leads to a loss of internal rotation. If the loss of internal rotation exceeds the gain in external rotation, it is considered pathologic.

Mechanism of injury: Posterior-inferior capsular contracture. The posterior capsular contracture alters the kinematics of the shoulder, leading to SLAP lesions (most commonly type II) or impingement.

Presentation: Patients complain of shoulder pain and decreased performance. They may report difficulty reaching across their body or up their back.

Physical exam: External and internal rotation are documented with the patient supine and the scapula stabilized. Impingement tests and O'Brien's test will be positive if impingement or a SLAP tear has developed.

Differential diagnosis: Adhesive capsulitis, physiologic loss of internal rotation (internal rotation loss is *not* greater than external rotation gain).

Diagnostics: The diagnosis is clinical. MRI will demonstrate associated SLAP lesions or cuff pathology indicative of impingement.

Treatment: Physical therapy to focus on posterior capsular stretching. Patients who do not regain internal rotation with a therapy program are candidates for arthroscopic release of the posterior-inferior capsule. Patients with recalcitrant pain despite improvement in internal rotation may have a SLAP lesion that needs to be addressed surgically.

Prognosis and return to play: Ninety percent of patients respond to a stretching program. A prophylactic stretching program will protect against GIRD and the potential development of an associated SLAP lesion.

Adhesive Capsulitis

Description: Characterized by pain and gradual loss of both active and passive motion of the glenohumeral joint caused by soft tissue contracture.

Mechanism of injury: Capsulitis can be idiopathic, which is termed "primary adhesive capsulitis." Etiology is unknown. When the condition is due to a known intrinsic or extrinsic cause, such as postsurgical or posttraumatic, it is termed "secondary adhesive capsulitis." More common in women. Diabetics are more susceptible.

Presentation: Depends on stage of the disease, but the hallmarks are pain and limited active and passive motion. Four stages have been described.

- Stage 1: Pain with active and passive range of motion. Positive rest and night pain. Symptoms present less than 3 months.
- Stage 2 ("freezing stage"): Chronic pain and progressive loss of range of motion. Positive rest and night pain. Symptoms present 3 to 9 months.
- Stage 3 ("frozen stage"): Significant shoulder stiffness. Minimal rest and night pain. Symptoms present 9 to 15 months.
- Stage 4 ("thawing stage"): Minimal pain and progressive improvement in range of motion.

Physical exam: Careful examination of passive and active range of motion of the glenohumeral joint. Make sure to stabilize scapula so as not to be fooled by scapulothoracic motion.

Differential diagnosis: Glenohumeral arthritis, posttraumatic deformity, rotator cuff disease, polymyalgia rheumatica, posterior shoulder dislocation.

Diagnostics: Adhesive capsulitis is a clinical diagnosis. X-rays are unremarkable but are useful to rule out posttraumatic deformity or glenohumeral arthritis.

Treatment:

- Options include benign neglect, physical therapy, NSAIDs, intra-articular corticosteroid injections, manipulation under anesthesia, and surgical capsular release.
- Treatment should be individualized depending on the stage of disease at presentation. NSAIDs and corticosteroid injections are beneficial for the inflammatory process associated with stages 1 and 2. Physical therapy during these stages focuses on gentle range of motion and modalities for pain and inflammation. Range of motion exercise should be more aggressive during stages 3 and 4.
- Surgery is indicated for those who fail nonoperative treatment. Many would advise against surgery during stages associated with an inflammatory phase.
- Secondary adhesive capsulitis should be treated more aggressively.

Prognosis and return to play: The majority of patients will improve with conservative therapy but it is a protracted course. Small residual deficits in motion and functional limitations may exist.

Pectoralis Major Tear

Description: Tear of the pectoralis major tendon. More commonly isolated to sternal head. Usually a distal injury occurring as a tendon avulsion or rupture at myotendinous junction. Can occur as a proximal injury to the muscle belly.

Mechanism of injury: Distal injuries are associated with strenuous activity such as football, wrestling, and weightlifting (during eccentric phase of bench press exercise). Proximal injuries to the muscle belly usually are the result of direct trauma.

Presentation: Patients may remember specific incident and report feeling a tearing sensation and possibly a "pop." Complaints of

pain around chest, axilla, and upper arm associated with weakness and painful limited motion.

Physical exam: Acutely, swelling and ecchymosis will be present over anterior chest wall and upper arm (if distal injury). Weakness will be evident with resisted adduction and internal rotation. Distal injuries will often have a palpable defect in the axilla and noticeable asymmetry compared to contralateral side in complete tears.

Differential diagnosis: Biceps tendon rupture, anterior dislocation that spontaneously reduced, rotator cuff tear, contusion.

Diagnostics: Although x-rays are usually normal, avulsion injuries and loss of the normal pectoralis major shadow may be detected. MRI is the modality of choice. It can distinguish between partial and complete tears, and acute and chronic tears.

Treatment: Nonsurgical management applies only to proximal injuries or partial tears distally. Treatment consists of rest, ice, and physical therapy to maintain shoulder motion. Resisted strengthening should be incorporated after about 6 weeks. Surgical management is indicated for complete distal tears.

Prognosis and return to play: Athletes undergoing surgical repair can expect a return to full or near-full strength.

Clavicle Fracture

Description: Accounts for about 2.5% of all fractures. Eighty percent occur in the middle third of the clavicle.

Mechanism of injury: Caused by direct or, more commonly, indirect force. Indirect force involves fall onto an outstretched arm or more commonly onto the point of the shoulder. Direct blows to the clavicle resulting in fracture have also been reported.

Presentation: The patient is usually splinting the injured extremity and can give a clear history of the injury-causing event (Fig. 43-10). A visible deformity with marked swelling and ecchymosis may be present in displaced fractures.

Physical exam:

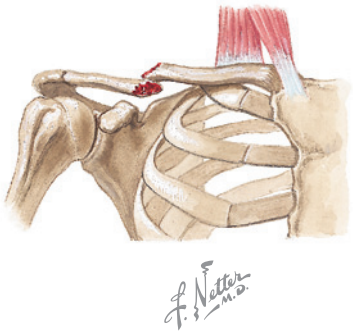
- Important to check the neurovascular status of the involved extremity.
- Evaluate the overlying skin at the fracture site to determine if it is at risk for breakdown, converting a closed fracture into an open fracture.
- Rule out any associated musculoskeletal injuries to the cervical spine or ipsilateral upper extremity, and any visceral injuries such as a pneumothorax.

Differential diagnosis: AC separation, SC dislocation, scapula fracture, contusion, congenital pseudarthrosis.

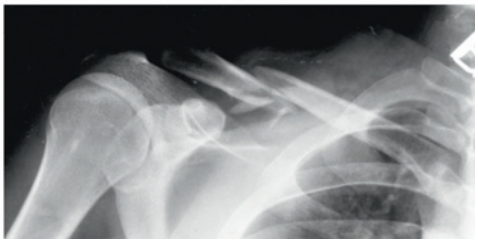
Diagnostics: Standard x-ray series of the shoulder to evaluate for associated shoulder girdle injuries (see Fig. 43-10). A standard AP x-ray alone of the clavicle will not adequately demonstrate the true displacement of the fracture. Therefore, a 45-degree cephalic tilt or axillary view with slight cephalic tilt is recommended in addition.

Treatment: Historically, clavicle fractures have been treated nonoperatively with immobilization in a sling or figure-of-eight dressing with good results in terms of union and function despite the deformity associated with healing of displaced fractures. More recently, literature suggests higher nonunion rates and lesser outcomes associated with shortening of the clavicle (>2 cm). These patients, particularly the high-level athlete, may benefit from surgical reduction and fixation. Surgical treatment is also indicated in fractures that are open, tenting the skin, or associated with a neurovascular injury.

Prognosis and return to play: Noncontact athletes can return to sports once evidence of radiographic healing is present and full, painless active range of motion with near normal strength has returned. Contact athletes should be withheld for 2 to 3 months to allow for adequate healing of the fracture.



Fracture of middle third of clavicle (most common). Medial fragment displaced upward by pull of sternocleidomastoid muscle; lateral fragment displaced downward by weight of shoulder. Fractures occur most often in children.



Anteroposterior radiograph. Fracture of middle third of clavicle.

Figure 43-10 Fracture of the Clavicle.

Proximal Humerus Fracture

Description: Commonly described by the Neer classification, which divides the proximal humerus into four parts: humeral head, greater tuberosity, lesser tuberosity, humeral shaft. A fracture fragment is considered a part if it is displaced greater than 1 cm or angulated more than 45 degrees (Fig. 43-11).

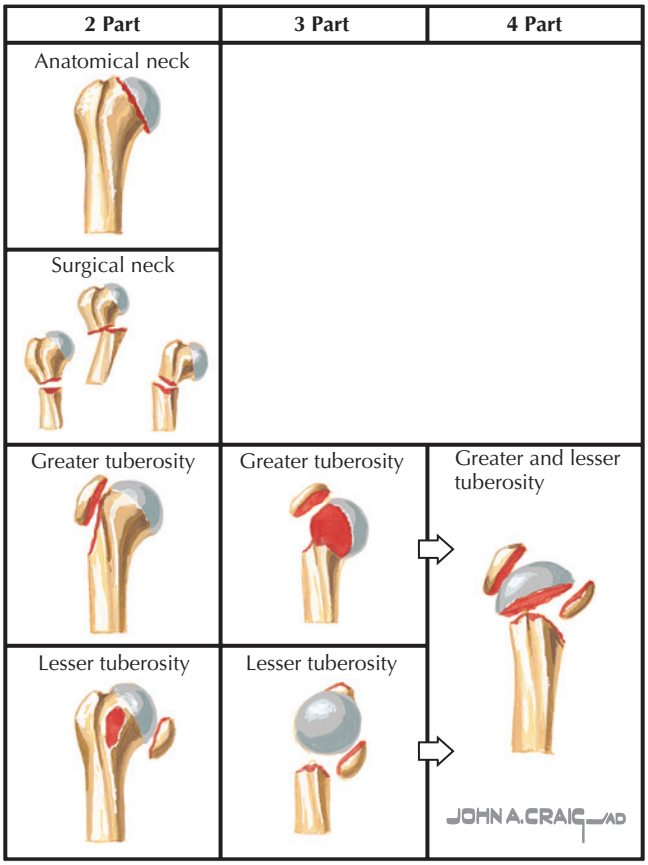


Figure 43-11 Neer Classification of Proximal Humerus Fractures.

Mechanism of injury: Sports-related fractures are usually caused by a high-energy impact or are avulsion fractures of the greater or lesser tuberosity associated with a dislocation. Fractures in the older population result from a fall onto the shoulder or outstretched arm.
Presentation: Pain, swelling, ecchymosis, and inability to move the shoulder. Patients often splint their arm against their body and support it with the uninjured arm.
Physical exam: Tenderness and swelling about the shoulder will be present. Pain and crepitus with range of motion. A good neurovascular exam is important.
Differential diagnosis: Dislocation, rotator cuff tear, contusion, clavicle fracture, scapula fracture.
Diagnostics: Plain x-rays are usually all that is required.
- Standard trauma series of the shoulder consisting of AP, axillary, and scapular-Y views. The axillary view is critical to rule out a concomitant dislocation. If the patient is not able to tolerate a traditional axillary view, a *Velpeau axillary* can be taken without moving the arm.
- CT may be helpful to better evaluate the amount of displacement of greater tuberosity fractures or evaluate articular involvement.

Treatment: Most fractures are minimally displaced and can be treated in a sling with early passive range of motion. Displaced fractures (>1 cm) require open reduction and internal fixation. Recent literature suggests that open reduction and internal fixation should be considered for greater tuberosity fractures displaced greater than 5 mm.
Prognosis and return to play: Noncontact, non-overhead athletes can return in 2 to 3 months, once adequate fracture healing has occurred. Overhead athletes will require a longer recovery time to allow for return of full range of motion and strength.

Neurologic Syndromes Affecting Shoulder

Parsonage-Turner Syndrome (aka Brachial Neuritis)

Description: Inflammation of nerves of the brachial plexus.
Mechanism of injury: Unknown etiology. Often associated with preceding upper respiratory tract infection or unusually heavy exercise.
Presentation: Initial complaint is pain in the shoulder or arm. As the pain resolves, weakness ensues.
Physical exam: Affected nerve distribution is variable but those most commonly affected are the axillary, musculocutaneous, suprascapular, and long thoracic nerves. Weakness in the corresponding muscle groups would be noted. Muscle atrophy may be apparent. Sensation is usually intact.
Differential diagnosis: Rotator cuff tear, adhesive capsulitis, stinger, suprascapular nerve entrapment, cervical spine pathology.
Diagnostics: Imaging is not generally useful. Electromyography (EMG) can be useful for the diagnosis and in localizing nerves involved.
Treatment: Pain control during the painful phase. Physical therapy to maintain range of motion and progression to strengthening exercises as weakness resolves.
Prognosis and return to play: Self-limiting condition with overall good prognosis. Time frame for complete recovery is variable; 36% recover by 1 year, 75% by 2 years, and 89% by 3 years.

Suprascapular Nerve Entrapment

Description: The suprascapular nerve provides motor function to the supraspinatus and infraspinatus muscle of the rotator cuff. Injury to the nerve can cause paralysis of these muscles.
Mechanism of injury: Can occur from a compressive lesion or from positional traction on the nerve caused by overuse (e.g., volleyball players or baseball pitchers). Injury usually occurs at one of two locations: (1) suprascapular notch or (2) spinoglenoid notch. Compression at these locations can be from a ganglion or paralabral cyst, hypertrophied/calcified transverse scapular ligament, narrow suprascapular notch, or lipoma (Fig. 43-12).
Presentation: Vague, deep pain in posterolateral shoulder with possible radiation down arm or into neck. Difficulty elevating arm past horizontal.
Physical exam: Should focus on strength testing of the rotator cuff and shoulder girdle muscles. Important to inspect for atrophy of the supraspinatus and/or infraspinatus muscles, which would be present in late stages. Distinguishing involvement of both the supraspinatus and infraspinatus versus just the infraspinatus indicates the location of injury. Injury at the suprascapular notch will involve both muscles, whereas injury at the spinoglenoid notch will be isolated to the infraspinatus.
Differential diagnosis: Cervical spine pathology, rotator cuff tear, Parsonage-Turner syndrome.
Diagnostics: Plain x-rays rarely demonstrate any positive findings. EMG can be helpful for diagnosis and to localize site of compression. MRI will demonstrate discrete compressive lesions and muscle atrophy.
Treatment: The majority of patients respond to conservative therapy consisting of activity modification, NSAIDs, analgesics, and physical therapy to strengthen the rotator cuff and scapular stabilizers. Surgical intervention is indicated in those patients who fail conservative therapy. Ganglion cysts in the spinoglenoid notch are often the result of superior labral pathology.

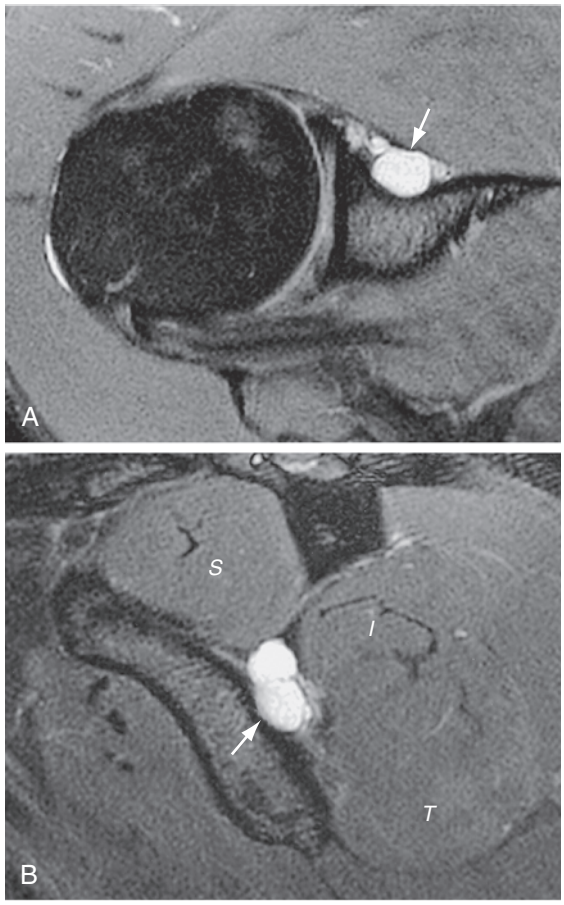


Figure 43-12 Spinoglenoid Cyst. *(Illustration reprinted with permission from DeLee J, Drez D, Miller M: DeLee & Drez's Orthopaedic Sports Medicine: Principles and Practice, 2nd ed. Philadelphia: Saunders, Elsevier, 2002.)*

Treatment to address this intra-articular pathology also successfully decompresses the cyst.

Prognosis and Return to Play: Once near normal strength and motion has returned.

Scapular Dyskinesia

Description: Abnormal motion of the scapula in relation to the thoracic cage and a visible altered position of the scapula. The dyskinesia of the scapula leads to altered kinematics of the glenohumeral and acromioclavicular joints.

Mechanism of injury: Most commonly the result of abnormal muscle activation and coordination. Contracture of shoulder muscles, ligaments, and capsular structures (e.g., GIRD) can contribute to dyskinesia). Bony abnormalities that affect shoulder girdle motion, such as malunited clavicle fractures or AC joint injuries, can also play a role.

Classification:

- **Type I:** Prominence of the inferior-medial scapular border.
- **Type II:** Prominence of the medial scapular border. Type II is a component of **SICK scapula syndrome** (**s**capular malposition, **i**nferior medial border prominence, **c**oracoid pain and malposition, and dys**k**inesia of the scapula).
- **Type III:** Prominence of the superomedial scapular border. Types I and II are associated with labral pathology whereas type III is associated with impingement and rotator cuff pathology.

Presentation: In the throwing athlete, patients often complain of pain anteriorly and decreased performance level. Onset is usually insidious.

Physical exam:

- Inspection to look for abnormal static scapular position such as winging, elevation, depression, or rotation. Also during shoulder motion to evaluate dynamic asymmetry.
- Patients with SICK syndrome will have pain anteriorly over the coracoid caused by tightness of the pectoralis minor.
- Scapular pinch test: Isometric retraction of the scapulas will elicit a burning sensation in those with scapular muscle weakness.
- Scapular assistance test: Determines if scapular dyskinesia is contributing to impingement signs. The scapula is stabilized and the inferior border is rotated with forward flexion of the extremity. If the impingement pain resolves or improves, the test is positive.
- Scapular retraction test: Scapula is stabilized in a retracted position. The test is positive if either (1) rotator cuff strength is improved or (2) pain or impingement with the Jobe relocation test is improved.

Differential diagnosis: Serratus anterior injury, long thoracic nerve injury, trapezial injury, spinal accessory nerve injury.

Diagnostics: X-rays will be normal. MRI will reveal associated pathology to the rotator cuff or labrum.

Treatment: The mainstay of treatment is physical therapy. This focuses on the periscapular muscles and their coordination in repositioning the scapula. Stretching off tight structures such as the posterior capsule and pectoralis minor is important. Therapy should also include the trunk and lower extremities because of their involvement in the kinetic chain of throwing. Surgical treatment may be necessary to address associated rotator cuff or labral pathology if indicated.

Prognosis and return to play: Once the scapula is symmetric with the contralateral side, the throwing athlete can return to play. This generally takes 3 months.

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7. Kirkley A, et al: Prospective randomized clinical trial comparing the effectiveness of immediate arthroscopic stabilization versus immobilization and rehabilitation in first traumatic anterior dislocations of the shoulder: Long-term evaluation. Arthroscopy 21(1):55-63, 2005.
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11. Tennent TD, Beach WR, Meyers JF: A review of the special tests associated with shoulder examination—Part I: The rotator cuff tests. Am J Sports Med 31(1):154-160, 2003.
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Elbow Injuries

44

John C. Carlisle, David J. Gerlach, and Rick W. Wright

GENERAL PRINCIPLES

History and Physical Exam

History: Hand dominance, location/type of pain, duration, mechanism of injury, alleviating/exacerbating factors, history of previous injuries, treatments rendered, recent changes in technique or training regimen, results of previous treatment.

Physical exam:

- **Inspection:** Compare to uninjured arm; assess atrophy/hypertrophy, edema, ecchymosis; carrying angle (cubitus valgus, cubitus varus).
- **General neurovascular examination:** Determine distal motor and sensory function; assess response to light touch; use two-point discrimination at fingertips to further define sensory deficits (normal 5 mm or less).
- **Manipulation (painful movements last)**
- **Active and passive range of motion (ROM):** Compare with uninjured elbow; feel for crepitus; average normal values: extension (0 degrees), flexion (150 degrees), pronation (75 degrees), supination (85 degrees) (Fig. 44-1).
- **Use resisted isometric movements to determine strength.**
- **Stability:** Valgus-varus stress, anterior-posterior stress, static and dynamic ulnar collateral ligament testing.

Palpation: Identify areas of tenderness as anterior, posterior, medial, or lateral. Focus on key anatomic structures.

- **Anterior elbow pain:** Biceps tendon, anterior capsule, median nerve.
- **Posterior elbow pain:** Triceps tendon, olecranon process, olecranon bursa.
- **Medial elbow pain:** Medial epicondyle, flexor-pronator mass, medial collateral ligament, ulnar nerve (Fig. 44-2).
- **Lateral elbow pain:** Lateral epicondyle, radial head, capitellum, radial nerve/posterior interosseous nerve.

Ancillary Tests

X-ray: Anteroposterior (AP) and lateral. Special views: oblique (radial head view), axial projections (olecranon fossa; gun-sight view), gravity stress view.

Arthrogram/arthrotomogram: Articular incongruity, loose bodies.

Computed tomography (CT) scan: Fracture-dislocation, exostosis.

Magnetic resonance imaging (MRI): Soft tissue mass, ligament rupture, chondral defects.

Arthroscopy (intra-articular inspection): Loose bodies, chondral lesions, synovitis.

Electromyogram–nerve conduction study: Nerve compression.

ANTERIOR ELBOW INJURIES

Distal Biceps Rupture

Description: Traumatic avulsion of distal biceps tendon from bicipital tuberosity of the proximal radius.

Mechanism of injury: Eccentric extension load applied to flexed, supinated forearm.

Presentation: Atypical injury; 97% of biceps ruptures are proximal, only 3% of biceps ruptures occur at elbow; almost all occur in males, most commonly in 5th to 6th decades of life; thought to be associated with preexisting tendon injury/degeneration or steroid use.

Physical exam: Tenderness to palpation in antecubital fossa, regional ecchymosis, palpable tendon defect in complete tears (partial tears less common), tendon retraction if lacertus fibrosis torn ("Popeye sign"); weakness with supination/elbow flexion.

Differential diagnosis: Biceps tendonitis, cubital bursitis, lateral antebrachial cutaneous nerve entrapment.

Diagnostics: Mostly a clinical diagnosis, but can obtain ultrasound or MRI to confirm.

Treatment: Acute anatomic repair considered superior to nonoperative treatment; allograft/autograft may be required in chronic cases if tendon length cannot be restored; one- and two-incision repair techniques described; historical concern for radial nerve palsy with one-incision technique and heterotopic bone formation/radioulnar synostosis with two-incision technique; radial nerve injury less common with current suture anchor and endobutton methods of fixation using one incision.

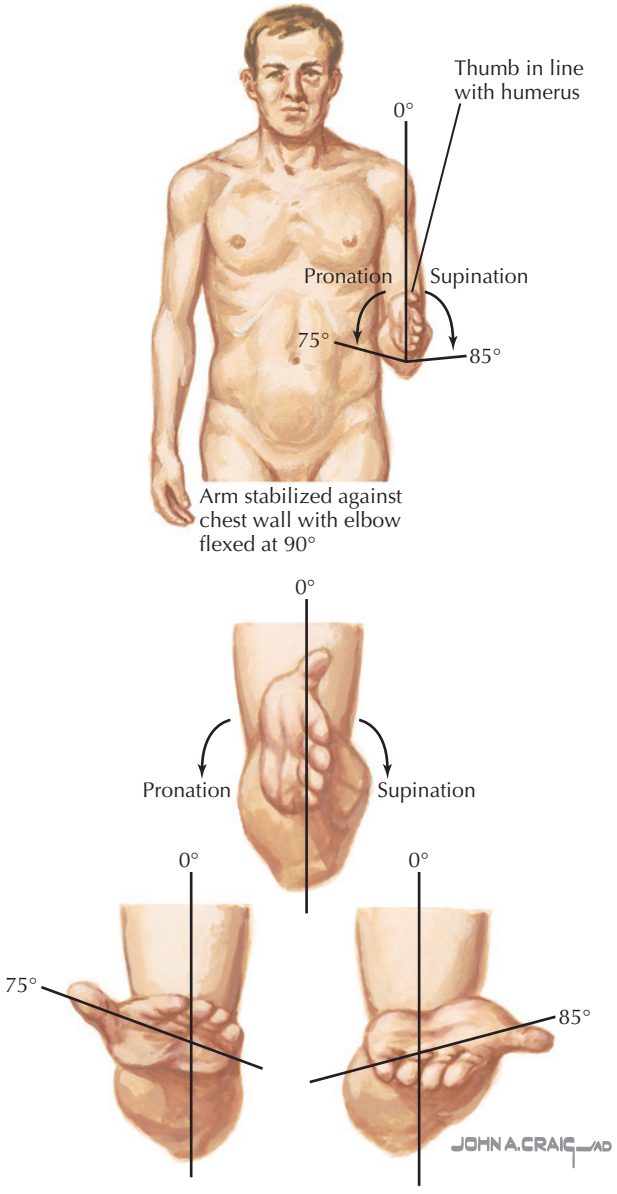


Figure 44-1 Measurement of Pronation/Supination.

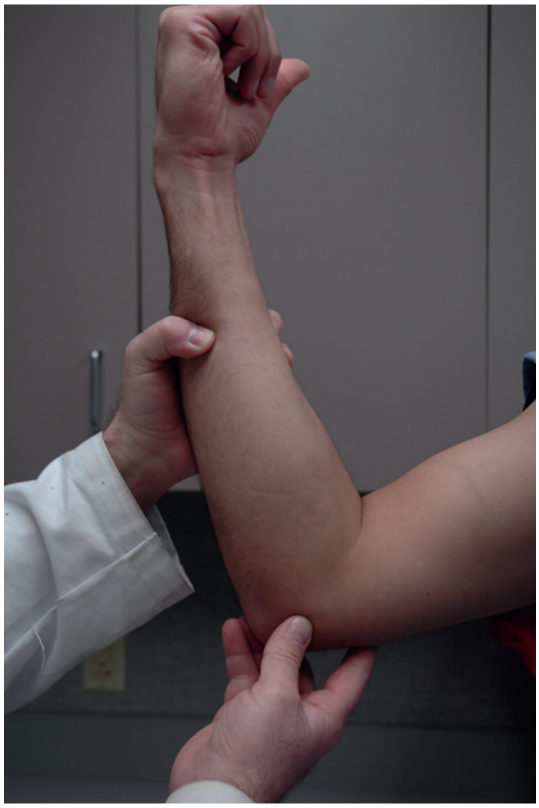


Figure 44-2 Medial Epicondyle Palpation.

Prognosis and return to play: Usually a season-ending injury; patients treated early can be expected to have full return of power and function.

Pronator Syndrome (Median Nerve Entrapment)

Description: Compression of the median nerve at the level of the elbow with resultant nerve irritation (Fig. 44-3).

Mechanism of injury: No specific mechanism of injury has been associated with this disorder; however, four possible sites of compression have been identified:

- Tendinous arch of the flexor digitorum superficialis ("sublimis bridge").
- An aberrant band of fibrous tissue that connects the sublimis bridge to the deep head of the pronator.
- Beneath the ligament of Struthers in patients with a supracondylar process.
- At the lacertus fibrosis at the level of the elbow joint.

Presentation: Patients often present with insidious onset of vague anterior elbow pain that increases with activity; associated activities include weightlifting, competitive driving, and underarm pitching.

Physical exam: Full, unrestricted range of motion; occasional hand and volar forearm paresthesias in the median nerve distribution (inconstant finding); some patients will have a positive Tinel's sign over the pronator teres; patients with a ligament of Struthers can have reproduction of symptoms with resisted flexion of the elbow at 120 to 135 degrees of flexion; a tight lacertus fibrosis can cause an indentation of the pronator muscle mass below the level of the medial epicondyle, and patients will often report pain with resisted pronation; patients with a hypertrophic pronator teres will

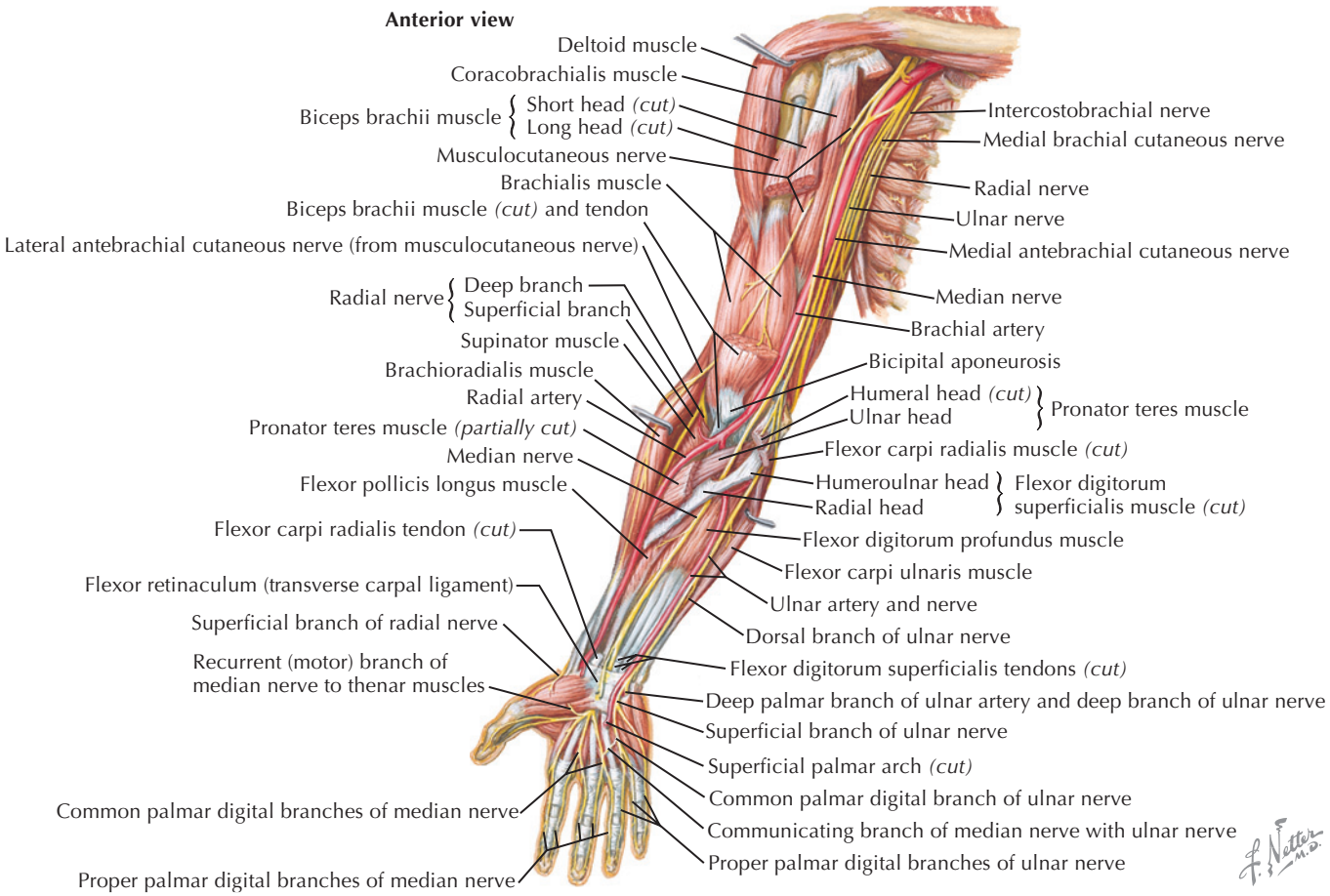


Figure 44-3 Nerves of the Upper Extremity.

often have tenderness to palpation 4 cm distal to the anterior elbow skin fold in addition to painful resisted pronation; if the nerve is compressed by the flexor digitorum superficialis (FDS) arch, patients can have pain with resisted middle finger flexion.

Differential diagnosis: Anterior elbow capsular strain.

Diagnostics: Plain radiographs are often normal, but can reveal a supracondylar process (present in 1% of people, 5 cm proximal to the medial epicondyle); electromyogram (EMG) and nerve conduction study (NCS) are an appropriate diagnostic step, but are known to be unreliable.

Treatment: Conservative treatment measures are generally not effective, but all patients should undergo a trial of rest and activity modification followed by physical therapy; sites of compression must be released operatively with an anterior approach to the elbow, targeting release of the ligament of Struthers, the lacertus fibrosis, deep head of the pronator teres, and FDS arch.

Prognosis and return to play: Allowed upon restoration of strength and range of motion (2 to 3 months).

POSTERIOR ELBOW INJURIES

Triceps Tendonitis

Description: Inflammation of the triceps tendon at its insertion on the olecranon process of the ulna.

Mechanism of injury: Overuse injury from repetitive extension/hyperextension of the elbow.

Presentation: Most commonly occurs in baseball players and weightlifters; patients report pain focal to the triceps insertion on the olecranon; usually no acute trauma identified.

Physical exam: Normal range of motion and neurovascular exam; triceps tendon is tender to palpation at, or just proximal to, its insertion site; no palpable defects; focal pain with resisted elbow extension.

Differential diagnosis: Partial tendon tear; intratendinous, subtendinous, or olecranon bursitis; olecranon stress fracture; fracture of olecranon osteophyte.

Diagnostics: Plain x-ray usually normal, but lateral view can reveal traction osteophyte or calcific deposit in terminal tendon; MRI can be useful to distinguish between inflammation and partial triceps tendon tear.

Treatment: Almost all respond to rest, ice, nonsteroidal anti-inflammatory drugs (NSAIDs), and/or rehabilitation with graduated stretching and strengthening; steroid injection can also be considered in refractory cases, though known complication is tendon rupture; patients that do not respond to a year of conservative treatment can be offered open debridement of the tendon in an attempt to stimulate healing.

Prognosis and return to play: Most athletes can continue to play with this disorder with initiation of rest and focused rehabilitation in the off-season.

Triceps Rupture/Olecranon Avulsion

Description: Traumatic avulsion of the triceps tendon from its insertion on the olecranon process of the ulna, or avulsion of the olecranon process from the ulna with triceps tendon attached.

Mechanism of injury: Most commonly occurs from fall on outstretched hand with deceleration load applied to an actively contracting triceps; also reported in weightlifters and in direct trauma.

Presentation: Rare injury; twice as common in males than females; occurs in patients of all ages, including adolescents with an incompletely fused olecranon physis; can be associated with steroid use, metabolic bone disorders, and renal osteodystrophy.

Physical exam: Tenderness to palpation along olecranon and distal triceps; regional ecchymosis and edema; palpable defect of triceps tendon or step-off at olecranon; weak elbow extension/inability to hold elbow extended against gravity; modified Thompson squeeze test (compressing the muscle bulk of the triceps) fails to cause elbow extension.

Differential diagnosis: Triceps tendonitis, olecranon bursitis, olecranon stress fracture, posterior elbow impingement.

Diagnostics: Largely a clinical diagnosis; "flake sign" (small bony avulsion fragment from olecranon process) noted in 80% of these injuries; can use MRI or ultrasound to aid in diagnosis if unclear.

Treatment: Nonoperative treatment indicated only in the elderly or in patients with partial tears; nonoperative management consists of splint immobilization with the elbow in 30 degrees of flexion for approximately 4 weeks; treatment of choice is surgical repair within 2 weeks of injury with locked Krakow stitch passed through tendon and secured to bone through drill holes in olecranon; small bony fragments can be excised; large bony fragments can be secured to bone with screw fixation/tension band construct; allograft/autograft tendon might be necessary in cases of chronic retraction of the tendon.

Prognosis and return to play: Usually a season-ending injury with at least 6 months of recovery time expected; most athletes able to return to play at similar level to preinjury state.

Olecranon Impingement Syndrome

Description: Also known as **hyperextension valgus** overload syndrome or **"boxer's elbow";** mechanical abutment of olecranon process against posterior soft tissues or the olecranon fossa that occurs with terminal extension of the elbow.

Mechanism of injury: Overuse syndrome caused by repetitive extension overloading; can occur in a stable elbow (football linemen, gymnasts, weightlifters) or can be seen in athletes with chronic attenuation of the ulnar collateral ligament (overhead throwers) causing the olecranon process to impinge against the medial wall of the olecranon fossa.

Presentation: Can produce posterior elbow pain, crepitus, and other mechanical symptoms (such as locking or catching); overhead throwers often complain of premature fatigue, loss of velocity, or loss of control.

Physical exam: Athletes may have some loss of terminal extension; posterior elbow pain with valgus stress in terminal extension; possible laxity of ulnar collateral ligament (UCL) with valgus stress; can have palpable loose bodies.

Differential diagnosis: Olecranon bursitis, olecranon stress fracture, triceps tendonitis.

Diagnostics: Plain x-rays can reveal loose bodies, hypertrophic bone formation/osteophytes in humeral fossae, calcification of the UCL, medial epicondyle avulsion fractures or can be normal; MRI can help to further assess the status of the articular cartilage and highlights areas of soft tissue edema and attenuation.

Treatment: Patients with stable elbows often respond to rest, NSAIDs, icing, and a physical therapy regimen aimed at improving flexibility and elbow strength; patients with instability can also respond to a physical therapy regimen focused on wrist flexor and extensor strengthening (especially the flexor carpi radialis and pronator teres, which are often weak in UCL-deficient elbows); athletes that fail to respond are candidates for arthroscopic debridement of the posterior fossa with or without concomitant UCL reconstruction.

Prognosis and return to play: Patients with stable elbows do well following arthroscopic debridement with full return to play expected; patients with instability do well with simple debridement in the short-term, but up to 25% of high-level throwers will need revision surgery with UCL reconstruction to fully address their symptoms long-term.

Olecranon Stress Fracture

Description: Microfracture of the proximal portion of the ulna.

Mechanism of injury: Overuse injury that results from repeated tension on the proximal ulna with throwing.

Presentation: Less common in adult throwers than adolescents and children; patients usually report gradual onset of pain in the posterior or lateral elbow that occurs during the acceleration phase of throwing.
Physical exam: Normal neurovascular exam and range of motion; focal point tenderness to palpation over olecranon without significant tenderness over triceps tendon moving proximally; no significant crepitus.
Differential diagnosis: Triceps tendonitis, olecranon bursitis, posterior impingement syndrome.
Diagnostics: Occasionally can see fracture line on plain radiographs, but often need a CT scan or MRI to confirm the diagnosis; bone scans are an alternative to other forms of advanced imaging; comparison x-rays can be useful in the skeletally immature athlete because of variability in location of the olecranon physis.
Treatment: Immediate cessation of throwing with non–weight-bearing status in the affected arm; controversy exists regarding conservative treatment versus percutaneous screw fixation of the fracture; nonoperatively treated patients must remain out of play and non–weight-bearing until point tenderness abates with gradual rehabilitation thereafter; if patients become symptomatic again with rehab, percutaneous, cannulated screw fixation of the fracture can be considered; early range of motion can be started following screw fixation; strengthening can be initiated once healing is evident on radiographs.
Prognosis and return to play: Fractures reliably heal with appropriate rest and/or screw fixation; athletes are generally able to return to play at the same level of competition after 3 to 4 months.

Olecranon Bursitis

Description: Also known as **"miner's elbow,"** or **"student's elbow";** inflammation of the bursa overlying the olecranon process of the ulna; can be acute or chronic, septic or aseptic (Fig. 44-4).
Mechanism of injury: Typically occurs because of direct (often mild) trauma to the posterior elbow; may be secondary to a single direct blow, or to repetitive trauma to the superficial tissues; septic bursitis often occurs through contamination of a skin wound or via surrounding dermatitis.
Presentation: Acute or gradual onset of swelling; acute/septic cases can be painful, whereas chronic cases are often painless; most common in football and hockey players; high association with play on artificial turf.
Physical exam: Focal posterior elbow swelling; mobile, fluctuant mass that can wax and wane in size; can have associated erythema or drainage in septic cases; surrounding forearm edema usually seen in cases of septic bursitis only; no restriction in range of motion; normal neurovascular exam.

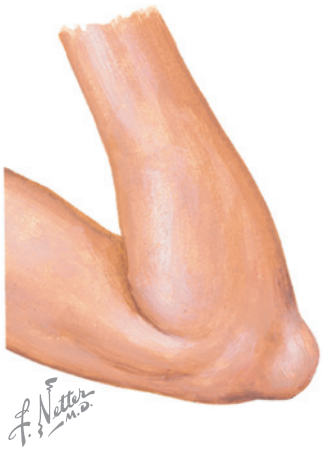


Figure 44-4 Olecranon Bursitis (Student's Elbow).

Differential diagnosis: Gouty tophus, calcium pyrophosphate deposition.
Diagnostics: X-rays occasionally will show calcification of the bursa or olecranon spur; aspiration can be performed in acute and chronic cases; fluid should be sent for cell count and differential, Gram stain/culture, and crystal analysis; aseptic fluid has a low white blood cell count with a high percentage of monocytes (>80%); gouty crystals not uncommon; *Staphylococcus aureus* most common cause of septic bursitis.
Treatment: Acute cases treated effectively with rest, short-term immobilization (3 to 5 days), compressive dressing, ice, and NSAIDs; chronic cases can be treated with aspiration and injection of corticosteroid, with a compressive dressing to be worn for 2 to 3 weeks; septic bursitis should be drained/excised with administration of intravenous antibiotics (1 to 3 weeks), followed by 2 weeks of oral antibiotics; chronic aseptic cases can also be treated with excision of the bursal sac.
Prognosis and return to play: Patients treated nonoperatively are at risk for recurrence; aseptic bursitis should not affect athlete participation in sport (however, appropriate protective padding should be used upon return to play); surgical excision typically leads to 6 weeks of absence from sport, and can be delayed until the end of an athlete's season.

MEDIAL ELBOW INJURIES

Medial Epicondylitis

Description: Also known as **"golfer's elbow";** inflammation/degenerative change of the flexor-pronator mass at its origin on the medial epicondyle.
Mechanism of injury: Stress/overuse injury of the flexor-pronator mass that occurs with repetitive wrist flexion or forearm pronation.
Presentation: Pain at the medial epicondyle; symptoms are often mild and intermittent; occurs less commonly than lateral epicondylitis; seen in pitchers, golfers, bowlers, weightlifters and football players; can be associated with ulnar neuropathy and, less commonly, triceps tendonitis and loose body formation.
Physical exam: Usually full range of motion without associated crepitus; patients have pain at the medial epicondyle and overlying the flexor-pronator mass proximally; discomfort/weakness exacerbated by resisted wrist flexion and/or pronation performed in full extension; some patients have a positive Tinel's with percussion of the flexor-pronator mass.
Differential diagnosis: Ulnar collateral ligament sprain, flexor-pronator tear, ulnar neuritis.
Diagnostics: Radiographs typically normal, though calcifications in the flexor-pronator mass occasionally present; EMG/NCS will be normal (even in cases with positive Tinel's in the region); MRI can be used to confirm the diagnosis in patients with possible conflicting sources of pain (such as throwing athletes with evidence of medial laxity on exam).
Treatment: Majority of patients will respond to conservative treatment consisting of activity modification, counterforce elbow bracing, NSAIDs, icing, and a physical therapy program aimed at flexor-pronator strengthening; corticosteroid injections can be considered in refractory cases (however, must avoid injection into or around the ulnar nerve); patients with symptoms lasting more than a year despite treatment can be offered operative debridement of the degenerative proximal portions of the pronator teres and flexor carpi radialis with gentle curettage of the medial epicondyle.
Prognosis and return to play: Athletes can generally initiate rehabilitation in the off-season, with return to play once asymptomatic (typically a 6- to 12-week process); approximately 90% have good to excellent results with surgical intervention, though only a minority of patients will fail to respond to conservative treatment.

Flexor-Pronator Strain

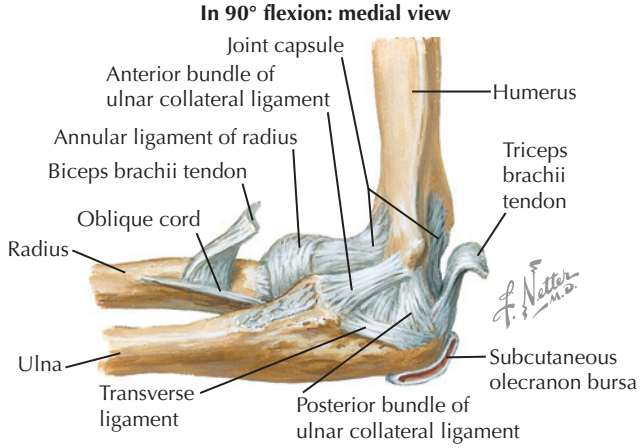
Description: Acute injury to the flexor-pronator mass immediately distal to the common tendon origin at the medial epicondyle.
Mechanism of injury: Can occur with a valgus stress to the elbow causing partial rupture/microrupture of the flexor mass; often occurs during the late cocking or acceleration phase of throwing; often seen with inadequate warm-up or in fatigued throwers.
Presentation: Similar presentation to medial epicondylitis; however, symptoms usually occur more acutely.
Physical exam: Similar to medial epicondylitis; focal tenderness at medial epicondyle or immediately distal; may have mild associated edema/ecchymosis; pain with resisted pronation or wrist flexion.
Differential diagnosis: Ulnar collateral ligament injury.
Diagnostics: Plain radiographs will be normal in the acute setting; MRI can be considered to rule out UCL injury.
Treatment: Rest, NSAIDs, followed by physical therapy (for motion and strengthening) and a gradual return to throwing.
Prognosis and return to play: Most patients will be asymptomatic after a 2- to 3-week period of rest, with gradual return to play over the following weeks; athletes should be prohibited from throwing if symptoms recur.

Ulnar Collateral Ligament Sprain

Description: Microtears or complete ruptures of the ulnar collateral ligament (Fig. 44-5).
Mechanism of injury: Repetitive valgus stress (pitching, throwing, racket sports) causes tensile loading of ulnar collateral ligament, resulting in microtears or complete rupture.
Presentation: Insidious medial elbow pain, provoked by valgus stresses and relieved by rest; throwing athletes; pain returns when throwing exceeds 75% normal velocity; complete ruptures may have single, severe episode.
Physical exam: Swelling, pain, tenderness 2 cm distal to medial epicondyle (anterior oblique ligament); pain increased by manual valgus stress (elbow 30-degree flexion) or "milking" maneuver; moving valgus stress test, Tinel's (associated ulnar neuritis) (see Fig. 44-5).
Differential diagnosis: Ulnar neuritis, medial epicondylitis, flexor-pronator muscle rupture/strain.
Diagnostics: X-rays may show avulsion fracture (acute); medial collateral ligament ossification (18%), loose bodies, marginal osteophytes (chronic); manual or gravity valgus stress views to confirm (>2 mm gapping). MRI useful for tear evaluation: partial, high-grade partial, or complete. Sensitivity increased with contrast.
Treatment: Rest, NSAIDs, physical therapy, correction of throwing mechanics; limit pitch count. Complete medial collateral ligament tear (acute or chronic) may require surgical reconstruction.
Prognosis and return to play: Variable outcome with nonoperative management; full tear is a season-ending injury; postsurgical rehabilitation lasts 12 to 18 months; 80% good to excellent results.

Medial Epicondyle Stress Lesions

Description: Also known as **"little leaguer's elbow";** medial-sided pain in the throwing elbow of adolescents.
Mechanism of injury: Repetitive tensile stress on the medial epicondyle apophysis from the flexor-pronator mass and the ulnar collateral ligament. Results from transient weakness of the apophysis in the adolescent; high valgus stresses of throwing.
Presentation: Triad of symptoms: medial elbow pain with throwing, loss of throwing speed, and diminished throwing effectiveness.
Physical exam: Point tenderness at medial epicondyle; pain with valgus stressing, but not frank instability; "milking" test may be positive.




Milking maneuver valgus stress test.

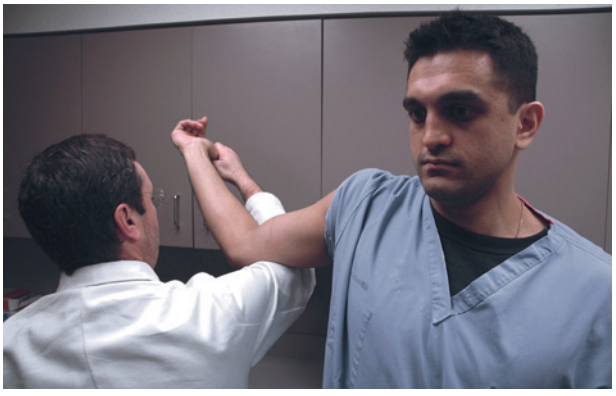

Moving valgus stress test.

Figure 44-5 Ulnar Collateral Ligament Sprain.

Differential diagnosis: Ulnar collateral ligament injury, ulnar neuropathy, medial epicondyle fracture, flexor-pronator strain.
Diagnostics: X-rays show widening of apophyseal line (versus contralateral); less commonly, fragmentation.
Treatment: Minimum of 6 weeks cessation of throwing; ice, NSAIDs, and brief immobilization; gradual return to throwing only after pain free with emphasis of proper throwing mechanics.
Prognosis and return to play: Full recovery is usually achieved with nonoperative management; recurrence possible, especially with poor mechanics; expect throwers to be out 3 months.

Ulnar Nerve Compression Syndrome

Description: Also known as **cubital tunnel syndrome;** compression of the ulnar nerve as it crosses the elbow joint.

Mechanism of injury: May be incited by trauma, cubitus valgus deformity, or subluxing ulnar nerve at the medial epicondyle. Also seen in weightlifters concentrating on triceps. However, most often, onset is insidious.

Presentation: Insidious onset of aching medial elbow/forearm pain, numbness at ring/small fingers, and grip weakness; range of motion not limited; often awake patient at night.

Physical exam: Positive Tinel's sign over cubital tunnel; positive ulnar nerve compression test; subluxation of ulnar nerve with elbow flexion; positive Froment's test; grip weakness; weak flexor digitorum profundus (FDP) to small finger.

Diagnostics: EMG/NCV**:** slowing of conduction velocity across elbow (20% to 25%). X-rays: usually normal, but may have osteophytes or cubitus valgus deformity.

Differential diagnosis: Cervical radiculopathy, thoracic outlet syndrome, ulnar nerve compression at wrist (Guyon's canal), ulnar collateral ligament injury.

Treatment: NSAIDs, modification of training, night-time splinting, elbow pads; anterior transposition if poor response to nonoperative management.

Prognosis and return to play: Dependent on severity and chronicity of neuropathy. If surgically treated, may return to full activity at 4 to 6 weeks (if transposition is subcutaneous).

LATERAL ELBOW INJURIES

Lateral Epicondylitis

Description: Also known as **"tennis elbow";** degenerative tears in extensor carpi radialis brevis (ECRB) origin with pain at lateral epicondyle (Fig. 44-6).

Mechanism of injury: Repetitive contraction of wrist extensors leads to extensor tendon degeneration.

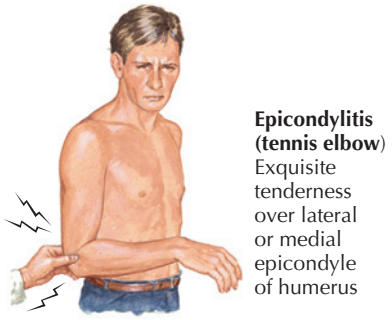
Presentation: Ten times more frequent than medial epicondylitis; increased risk with racket sports, age above 40, poor technique, dominant arm; pain with lifting objects; initially pain subsides with rest.

Physical exam: Tenderness to palpation over lateral epicondyle and ECRB; pain with resisted wrist and long finger extension; pain with resisted supination (see Fig. 44-6).

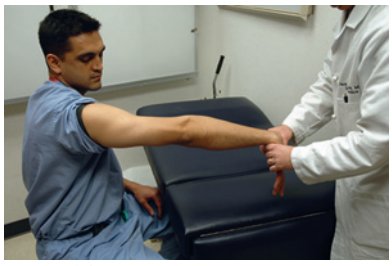
Diagnostics: Clinical diagnosis. X-rays are usually normal (22% calcific changes); MRI may show inflammation of the ECRB.

Differential diagnosis: Posterior interosseous nerve entrapment (15% comorbid), radiocapitellar arthrosis, osteochondritis dissecans, cervical radiculopathy.

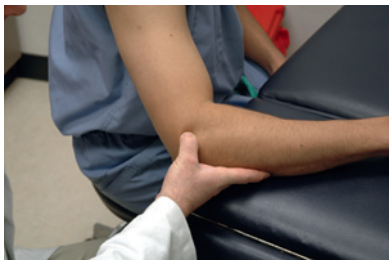
Treatment: Nonoperative management with activity modification, ice, NSAIDs, physical therapy, and counterforce bracing. Corticosteroid injection if severely painful or if symptoms persist despite therapy. Surgical treatment rare, with excision of ECRB tendon. Racket sport athletes should analyze stroke mechanics and racket grip size as part of treatment.



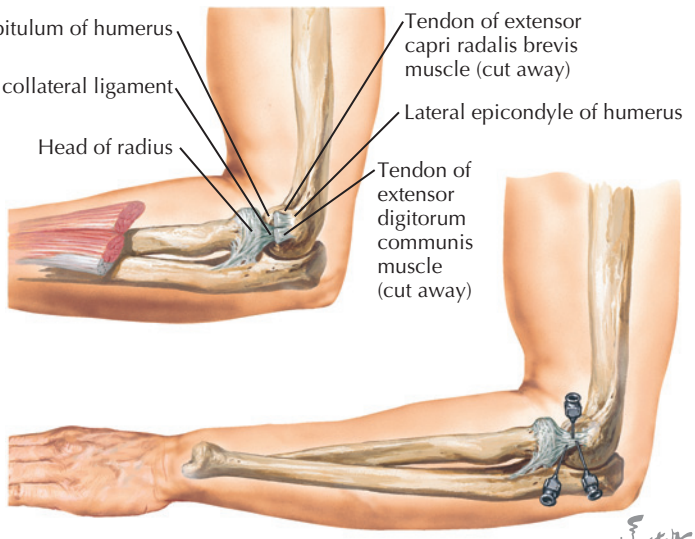
Epicondylitis (tennis elbow) Exquisite tenderness over lateral or medial epicondyle of humerus



Resisted finger and wrist extension.



Tenderness to palpation lateral epicondyle.



Technique for injection of tennis elbow

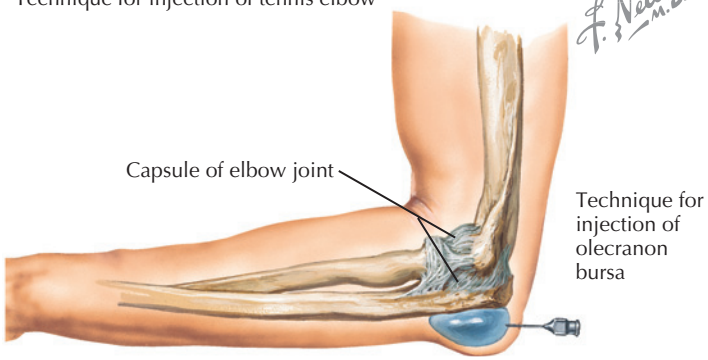


Figure 44-6 Tennis Elbow.

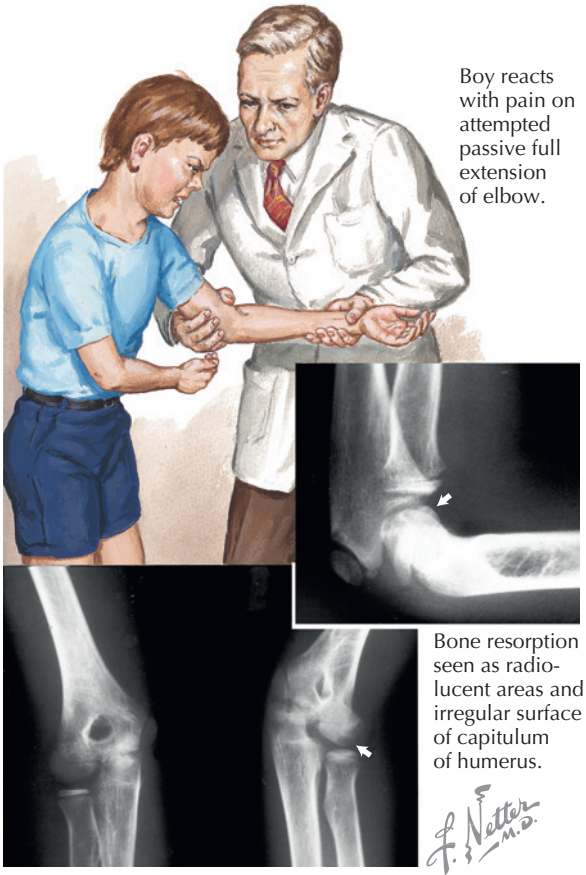
Prognosis and return to play: Nonoperative treatment is successful in 80% to 90%; 6 to 12 week course. Of surgical cases, 85% returned to full activity by 6 months.

Radiocapitellar Chondrosis

Description: Damage to the articular cartilage of the radius and capitellum secondary to compressive forced from valgus stress.
Mechanism: Valgus stress of throwing and racket sports imparts strong tensile forces to medial collateral ligament and strong compressive forces to lateral joint of elbow, leading to overload forces against the articular cartilage. May also occur following sudden, extreme compressive event or radial head fracture.
Presentation: Throwing athletes; painful swelling at lateral elbow with throwing; catching and locking.
Physical exam: Tender radiocapitellar joint, lateral swelling, crepitus and increased pain with forearm pronation-supination; decreased range of motion; assess medial collateral ligament integrity.
Diagnostics: X-ray: loss of radiocapitellar joint space; marginal osteophytes, loose bodies (late changes); MRI: loss of articular cartilage.
Differential diagnosis: Medial collateral ligament injury, proximal distal radial-ulnar joint (DRUJ) injury, radial head fracture, osteochondritis dissecans capitellum.
Treatment: Rest, NSAIDs, physical therapy, graduated activity dictated by pain; corticosteroid injection; joint debridement through lateral arthrotomy or arthroscopy with removal of marginal osteophytes and loose bodies; late stages, radial head excision versus replacement.
Prognosis and return to play: Difficult to treat once articular cartilage is damaged; gradual return to play if pain free, but recurrence of pain is common, and sign of progression.

Osteochondritis Dissecans Capitellum

Description: Focal lesion in adolescents consisting of a separation of both articular cartilage and subchondral bone (Fig. 44-7).
Mechanism of injury: Repetitive microtrauma to capitellum from high valgus stresses leads to chondral injury and subchondral fractures; fractures become necrotic and are gradually resorbed, while overlying cartilage remains viable; if cartilage remains intact, bone defect may fill in; if cartilage loses integrity, loose body may develop.
Presentation: History of activity-related lateral elbow pain in adolescent/young adult; males in throwing sports, females in gymnastics; dominant arm; history of repetitive, painful overuse; pain resolves with rest or in off-season; occasional clicking/locking.
Physical exam: Tenderness of radiocapitellar joint; swelling; lack of full extension; crepitus, clicking, popping with range of motion (suspect loose body); positive radiocapitellar compression test (pronation/supination with elbow in extension).
Diagnostics: X-rays: radiolucency and rarefaction of the capitellum, with flattening of the articular surface, possible loose bodies; MRI: early low-signal changes on T1 images; T2 images helpful for denoting intervening fluid in lesion consistent with fragment separation.
Differential diagnosis: Panner's osteochondrosis.
Management: For intact articular cartilage, period of rest with activity restriction (3 to 6 weeks), with physical therapy and gradual return to activities at 3 months, full activity by 6 months; follow with serial x-rays. If fragment is displaced, recommended treatment is excision of unstable articular fragment and drilling of capitellar defect; surgery indicated for loose bodies, continued pain despite conservative management, displacement or detachment of the cartilage or lesion; if lesions detached, debridement with drilling or microfracture of lesion.
Prognosis and return to play: Early diagnosis and treatment are essential; for early lesions, will miss remainder of season, but long-term results are consistently good and excellent; for advanced lesions, return to preinjury competition level is dubious, especially for dominant arm pitchers, catchers, and gymnasts, although long-term results remain positive, with occasional loss of extension.

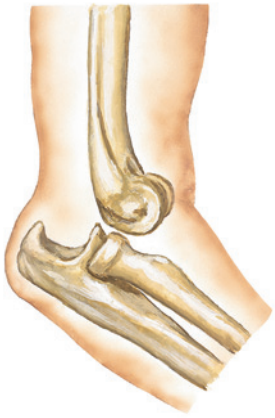


Characteristic changes in capitulum of left humerus *(arrow)* compared with normal right elbow.

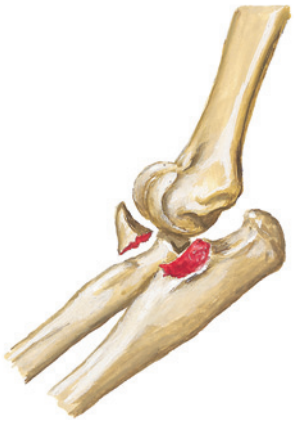
Figure 44-7 Osteochondritis Dissecans Capitellum.

Posterior Interosseous Nerve Compression Syndrome

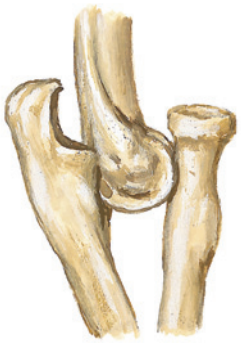
Description: Also known as **radial tunnel syndrome;** entrapment neuropathy of posterior interosseous branch of radial nerve.
Mechanism of injury: Hypoxemia of the posterior interosseous nerve, leading to paresthesia, secondary to compression under fibrous arch of supinator (arcade of Frosche) or more distally in supinator muscle.
Presentation: Arching lateral elbow pain from lateral epicondyle, radiating into dorsal forearm (arcade of Frosche); aggravated by pronation-supination activities; extensor weakness of wrist and fingers; no numbness.
Physical exam: Positive Tinel's 8 cm distal to lateral epicondyle; pain with resisted pronation/supination; pain with long finger extension; weakness of wrist extensors and extensor digitorum communis (EDC); no sensory loss.
Diagnostics: Primarily a clinical diagnosis; x-rays**:** osteophyte at radiocapitellar joint (rare); EMG/NCV: rarely confirmatory for posterior interosseous nerve compression.
Differential diagnosis: Lateral epicondylitis, C7 radiculopathy, extensor tendon rupture, distal posterior interosseus nerve syndrome.
Treatment: Rest, training schedule modification, dorsiflexion wrist splint, physical therapy for stretching and strengthening; surgical decompression in recalcitrant cases.



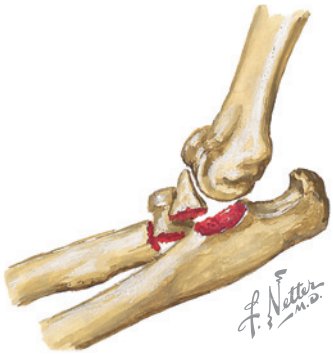
Posterior dislocation. Note prominence of olecranon posteriorly and distal humerus anteriorly.



Fracture of coronoid process of ulna with posterior dislocation of elbow. Coronoid fracture may occur occasionally without dislocation.



Divergent dislocation, anterior-posterior type (rare). Medial-lateral type may also occur (extremely rare).



Posterior dislocation with fracture of both coronoid process and radial head. Rare but serious; poor outcome even with good treatment. May require total elbow replacement.

Figure 44-8 Dislocations of the Elbow.

Prognosis and return to play: Nonoperative management usually successful (80%); full return to sports usually by 4 to 8 weeks; surgical release highly effective (90%), return 6 to 8 weeks.

DISLOCATIONS AND FRACTURE-DISLOCATIONS

Description: Fracture of the radius, ulna, or humerus, with or without dislocation or subluxation of the elbow joint (Fig. 44-8).

Mechanism: High-energy traumatic event, usually a fall onto outstretched arm.

Presentation: History of trauma; severe pain, exacerbated by subtle movement; instability.

Physical exam: Tenderness, deformity, swelling, ecchymosis; limited and severely painful motion, crepitus; possible neurologic or vascular compromise; varus/valgus instability.

Diagnostics: X-ray: fractures of radial head/neck, olecranon/coronoid, distal humerus; dislocation with or without fracture; proximal ulna fracture with associated radial head dislocation (Monteggia fracture); CT and MRI to assess bone and ligamentous damage.

Differential diagnosis: Associated ligamentous injury.

Treatment: Document neurovascular examination, if compromised, attempt reduction immediately; immobilize with splint; refer urgently to emergency department for evaluation and further treatment as indicated (closed reduction, open reduction and internal fixation, late ligamentous repair).

Prognosis and return to play: Usually a season-ending injury; extended course of treatment and rehab; simple dislocations usually have good outcomes with early range of motion; return to previous level of sports unlikely after complex fracture/dislocations.

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Hand and Wrist Injuries

45

Jeffry T. Watson, Douglas R. Weikert, and Nathan van Zeeland

GENERAL PRINCIPLES AND EVALUATION

Overview

- Fortunately, most sports-related hand and wrist injuries, when addressed in a timely manner, do not represent a significant threat to limb viability, long-term function, or eventual return to sport.
- Perhaps the greatest morbidity from these injuries results from delayed presentations or missed injuries.
- Hand function is closely linked to full mobility of the digits and a stable, mobile wrist.

Physical Examination

- Dictated by the context of the injury; no single, comprehensive evaluation applies to all maladies.
- Attention is directed toward the individual part and system (bone, joint, tendon, nerve, etc.) in question.

Observation/Inspection

- Observation is the starting point for any evaluation.
- Focal swelling, digital perfusion, digital malrotation, digital cascade, and any penetrating injuries should be noted.
- Any difference in posture of one digit relative to the others should not be dismissed or minimized, because this often signifies a displaced fracture, tendon avulsion, or joint subluxation.
- In the absence of a penetrating injury, isolated pallor of a digit usually represents spasm of the digital vessels. Although this often resolves with digital warming or reduction of associated displaced fractures or dislocations, digital viability remains in question until perfusion is actually observed.
- Even in closed fractures, the digital vessels can tear or thrombose, representing a surgical emergency.

Palpation

- Careful palpation of specific bones or ligaments in question should be emphasized.
- Focused palpation will usually localize the injured structure within an area of generalized edema demonstrating diffused swelling; for example, a swollen wrist following distal radius fracture or perilunate injury.
- Tenderness over a scapholunate ligament, even in the setting of normal radiographs, will suggest an underlying ligament tear that would do poorly if not recognized and treated.

SPECIFIC INJURIES AND CONDITIONS

Nail Bed Injury

Description: Any tear or disruption of the sterile or germinal matrix of the nail bed. May or may not be associated with an underlying distal phalanx fracture or actual disruption of the nail plate (Fig. 45-1).

Mechanism of injury: Usually caused by dorsal crush of the fingertip (such as when fingertip is crushed by another player's cleated shoe). However, may also occur with axial load to the fingertip that results in flexion fracture of the distal phalangeal shaft and tear of the overlying nail bed.

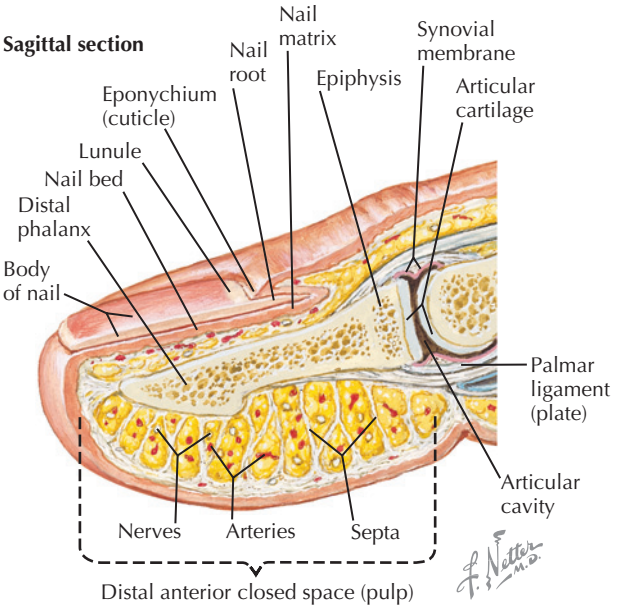
Presentation: If the nail plate is disrupted, the laceration through the matrix is usually readily visible. The tear may extend peripherally beyond the borders of the nail fold into surrounding skin. If the nail plate is intact, disruption of the matrix will always result in some degree of subungual hematoma (see Fig. 45-1).

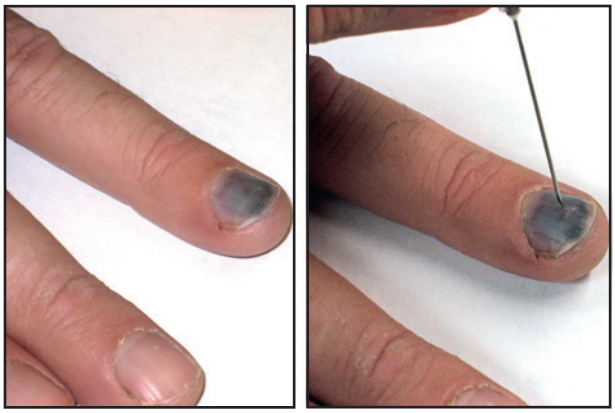
Physical exam: Angular deformity of the fingertip is suggestive of a concomitant distal phalangeal shaft fracture.

Differential diagnosis: If the base of the nail plate is flipped out dorsally over the nail fold, consider an open fracture of the proximal portion of the phalanx. In skeletally immature patients, the presence of the physis at this location may result in failure to recognize what is actually an open Salter-Harris I phalangeal fracture.

Diagnostics: Physical exam is usually sufficient. Anteroposterior (AP) and lateral radiographs reveal underlying distal phalanx fracture.

Treatment: For small subungual hematoma encompassing a portion of the nail plate, no intervention is necessary. Decompression of the hematoma through needle fenestration of the nail plate can offer pain relief. However, this may increase likelihood of wound sepsis if performed on playing field (see Fig. 45-1). For **larger hematoma** (>50% of the nail plate) with tearing beyond the nail fold borders, formal repair is required. Under a digital block anesthetic, the nail plate should be removed, the wound





Subungual hematoma | Nail plate fenestration

Figure 45-1 Nail Bed Injury.

irrigated, and the nail matrix repaired with 6-0 or 7-0 resorbable suture. The adjacent skin rip is repaired with 5-0 nylon. Underlying phalangeal tuft fracture is managed nonoperatively. Associated displaced fracture of the distal phalangeal shaft, however, requires washout and perhaps surgical stabilization.

Prognosis and return to play: If no nail bed repair is required, immediate return to play is okay. Following nail bed repair, the fingertip (including distal interphalangeal joint [DIP]) should be dressed and splinted to protect from impact. If there is an associated unstable phalangeal shaft fracture requiring pin fixation, return to competition should be delayed until pin removal. Prognosis for nail plate growth is directly related to anatomic restoration of nail bed. If there is a large span of scar in the matrix, a ridge or split in the nail plate will occur.

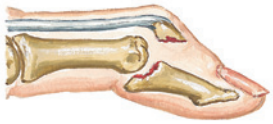
Mallet Finger

Description: Loss of terminal extensor tendon attachment to distal phalanx with resultant flexed deformity of DIP joint.

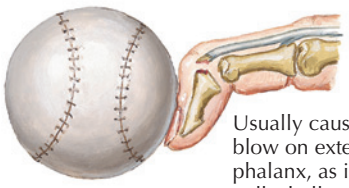
Mechanism of injury: Sudden forced flexion of the DIP joint during active extension through the terminal tendon, often as a result of a ball jamming the fingertip (Fig. 45-2).

Presentation: DIP joint is maintained in flexion with inability to actively bring joint into full extension. Varying degrees of pain, often with minimal or no pain. Swelling, ecchymosis may be noted over the dorsal aspect of the joint.

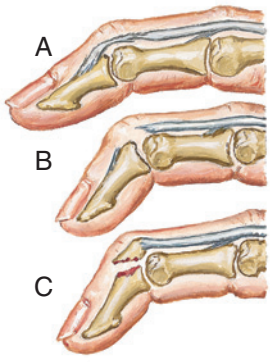
Differential diagnosis: Distal phalanx fracture or DIP dislocation.



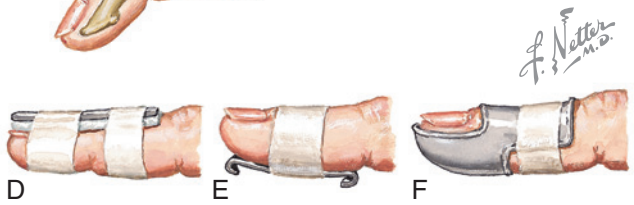
Mallet finger of bone origin. Avulsion of bone fragment and volar subluxation of distal phalanx



Usually caused by direct blow on extended distal phalanx, as in baseball, volleyball



Degrees of mallet finger injury. **A**. Extensor tendon stretched but not completely severed; mild finger drop and weak extensor ability retained. **B.** Tendon torn from its insertion. **C.** Bone fragment avulsed with tendon. In B and C there is 40° to 45° flexion deformity and loss of active extension.



Treatment for mallet finger of tendon origin. D. Padded dorsal splint. **E.** Unpadded volar splint. **F.** Stack splint. Proximal interphalangeal joint left free for active exercise.

Figure 45-2 Mallet Finger.

Diagnostics: Posterior-anterior and lateral x-ray of digit to assess if injury is limited to soft tissue (tendon only) or has associated bony avulsion (see Fig. 45-2). Lateral x-ray determines stability based on the size of the bony component and whether palmar subluxation is present.

Treatment: Acute mallet injuries with no subluxation on the lateral x-ray require *full-time* splinting of the DIP joint in extension for 6 to 8 weeks (see Fig. 45-2). Displaced bony components with joint involvement of more than 30% and/or palmar subluxation often require surgery to restore joint congruity. The DIP joint will usually require transarticular pinning in full extension with surgery.

Prognosis and return to play: Noncompliance with splint wear will negatively impact outcome and usually results in mild to moderate degrees of extension lag. The functional effect of this is variable. Most athletes treated nonoperatively will return to play within a week (while splinted, of course). With surgical treatment, return to play will depend on the athlete's ability to protect a transarticular pin. Any activity requiring grasp is likely to result in bending or breakage of the pin.

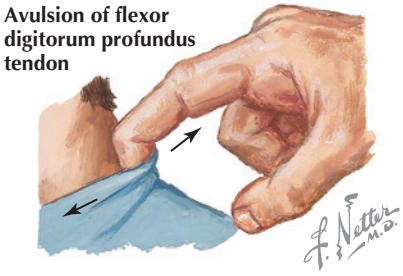
Jersey Finger

Description: Traumatic avulsion of flexor digitorum profundus (FDP) from distal phalanx (Fig. 45-3). Tendon may detach alone or avulse a palmar fragment of the distal phalanx with it. Ring finger most commonly affected.

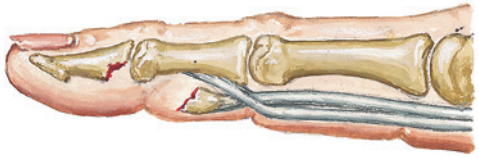
Mechanism of injury: Forced passive extension of DIP joint during active flexion of DIP joint.

Presentation: Typically seen in football and rugby players attempting to grab a jersey during a tackle. Variable degree of pain, although often the player will complain of pain proximally in the finger or palm at the level of the retracted tendon. Usual concern is inability to flex the involved DIP joint.

Physical exam: Ecchymosis may be present at DIP joint, depending on timing of presentation. The flexor tendon stump may be tender or palpable in the palm or along the digit, depending on



Caused by violent traction on flexed distal phalanx, as in catching on jersey of running football player



Flexor digitorum profundus tendon may be torn directly from distal phalanx or may avulse small or large bone fragment. Tendon usually retracts to about level of proximal interphalangeal joint, where it is stopped at its passage through flexor digitorum superficialis tendon; occasionally, it retracts into palm. Early open repair of tendon and its torn fibrous sheath indicated.

Figure 45-3 Jersey Finger.

the level of proximal retraction. Bony avulsions tend to become incarcerated along the flexor sheath (often at the A4 pulley over the middle phalanx). Loss of active DIP joint flexion is most specific finding.

Differential diagnosis: Distal phalanx fracture, DIP joint dislocation.

Diagnostics: PA and lateral x-ray of injured digit.

Treatment: Surgical reattachment of the flexor tendon within 7 to 10 days if the tendon has retracted into the palm. If tendon has retracted only to the proximal interphalangeal (PIP) level, reattachment may be successful with delay up to a few weeks. For flexor tendon avulsions with bony component, internal fixation is necessary to restore continuity of the flexor tendon.

Prognosis and return to play: Soft tissue FDP avulsions require 12 weeks of protected activity before return to full gripping and grasping activities. Bony avulsions amenable to open reduction and internal fixation require 4 to 6 weeks of protected activity. Both types of FDP avulsions also require extensive hand therapy postoperatively.

Proximal Interphalangeal (PIP) Joint Dislocation

Description: Dislocation of the proximal and middle phalanges. Usually the middle phalanx displaces dorsal to the proximal phalanx**.** However, rotatory (with the proximal phalanx condyle protruding between the lateral band and central slip), volar, and lateral dislocations, though less frequent, do occur (Fig. 45-4).

Mechanism of injury: Hyperextension of PIP joint with varying degrees of axial loading. Often occurs from ball striking finger, running into another participant, or ground striking finger.

Presentation: Usually with pain, swelling around PIP joint with or without angular deformity. Patient will be apprehensive to active or passive motion.

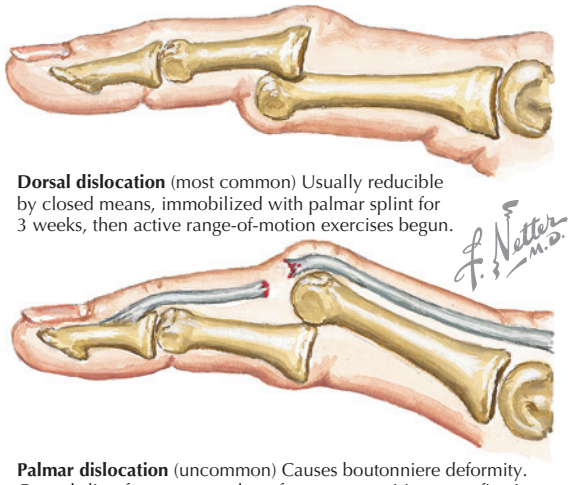
Physical exam: Pain localized to PIP joint with swelling. Occasionally will have skin laceration resulting from open injury.

Differential diagnosis: Fracture-dislocation.

Diagnostics: PA and lateral x-ray of the injured digit *must* be performed to verify congruent reduction and rule out fracture or subluxation, but can be delayed for a few days if "on the field" reduction is clinically stable. If the initial reduction attempts seem unsuccessful, films should be obtained before repeated efforts, because reduction may be prevented by fracture component or different orientation of dislocation (see Fig. 45-4).

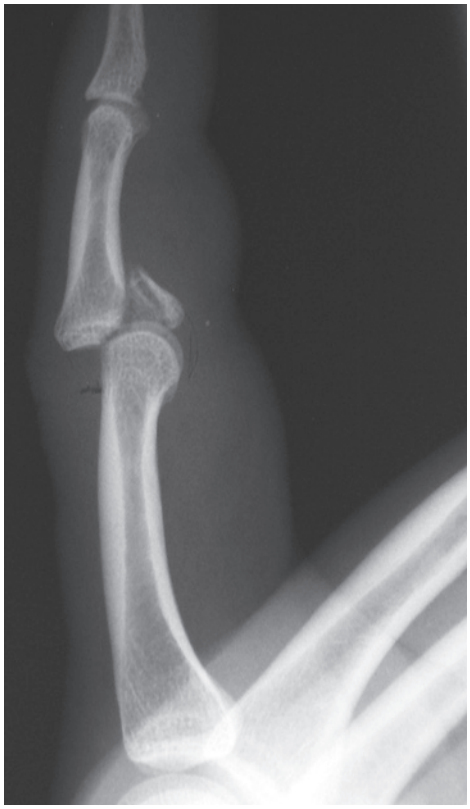
Treatment: Digital blocks may be necessary. Closed reduction employing longitudinal traction, slight extension, and dorsal pressure over middle phalanx for dorsal dislocations. Postreduction, must check range of motion (ROM) and joint stability. **X-ray or fluoroscopic confirmation of reduction is required within a few days.** Without significant periarticular fracture, instability requiring surgery is unlikely. For **rotatory dislocation,** manipulation with the metacarpophalangeal and PIP in flexed position facilitates reduction. **Volar dislocation** is reduced with slight PIP flexion and dorsal translation of the middle phalangeal base. Following reduction of volar dislocation, it is *crucial* to protect the central extensor slip insertion (which generally is always disrupted in volar dislocation) with immobilization of PIP in full extension in order to avoid inevitable progression to boutonniere deformity.

Prognosis and return to play: Return to play is usually minutes after closed reduction of dorsal dislocations. Buddy taping to adjacent digit usually facilitates this; early follow-up radiographs are mandatory. Volar dislocations need to be splinted in full extension for about 6 weeks to protect the central slip. During this time, active DIP motion is employed to promote gliding of lateral bands. Swelling and stiffness may persist for several months.



Dorsal dislocation (most common) Usually reducible by closed means, immobilized with palmar splint for 3 weeks, then active range-of-motion exercises begun.

Palmar dislocation (uncommon) Causes boutonniere deformity. Central slip of extensor tendon often torn, requiring open fixation, followed by dorsal splinting to allow passive and active exercises of distal interphalangeal joint.



Lateral radiograph of persistent PIP subluxation due to volvar fracture fragment.

Figure 45-4 Proximal Interphalangeal (PIP) Joint Dislocation.

PIP Fracture Dislocation

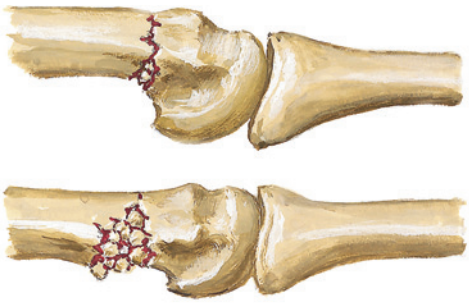
Description: Usually dorsal dislocation of middle phalanx at PIP joint with associated fracture of the palmar margin of middle phalanx base (see Fig. 45-4). Direct axial load, however, may result in comminuted pilon fracture of entire articular surface and metaphysis.

Mechanism of injury: Axial load with or without hyperextension of the digit, usually following same mechanism as PIP dislocation mentioned earlier.

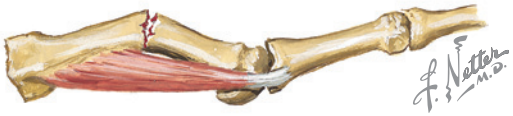
Presentation: Usually same as PIP dislocation; can be difficult to differentiate from dislocation clinically.
Physical exam: Swelling, deformity at PIP joint, point tenderness and crepitance at PIP joint.
Differential diagnosis: Volar plate injury without dislocation, PIP dislocation or shaft fracture of phalanx.
Diagnostics: PA, lateral, and oblique x-ray of injured digit. Fluoroscopy is also extremely helpful to determine stability of joint.
Treatment: Closed reduction with longitudinal traction using dorsal splint to hold PIP joint flexed may be adequate, depending on size of fractured palmar joint margin. Generally, if fracture involves less than 40% of the articular surface, this technique is useful. Joint is gradually moved into greater degrees of extension with weekly radiographic verification of maintained reduction over the ensuing 4 to 6 weeks. Fractures with persistent dorsal subluxation after closed reduction require surgical stabilization.
Prognosis and return to play: Return to sports often requires 4 to 6 weeks, with or without surgical intervention. Residual stiffness at PIP joint is common.

Metacarpal Fracture

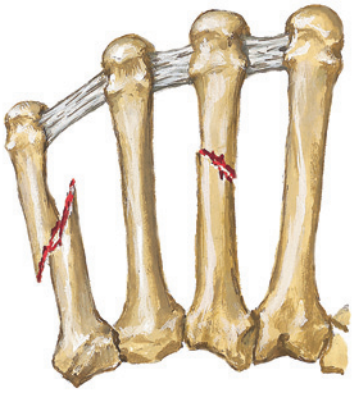
Description: Fracture of metacarpal neck or shaft.
Mechanism of injury: Direct trauma with axial load and clenched fist is common mechanism for distal or proximal metaphyseal fractures. Direct dorsal impact (such as baseball striking batter's hand or another participant stepping on hand) often results in shaft fractures (Fig. 45-5).
Presentation: Swelling of hand, with or without angular deformity of digits.
Physical exam: Point tender over metacarpal with swelling. Angular and sometimes rotational deformity (scissoring) of digit (see Fig. 45-5). Scissoring is more easily detected if patient is able to offer some degree of digital flexion.
Differential diagnosis: Contusion or metacarpophalangeal (MCP) joint dislocation.
Diagnostics: PA, lateral, and oblique x-ray of hand.
Treatment: Surgical treatment for fractures with angulation greater than 50 degrees of ring and small fingers and angulation greater than 20 degrees of index and middle fingers. Shortening of more than 5 mm and shaft fractures of the border digits (small and index) may also need open reduction and internal fixation. Significant rotational deformity/scissoring also is an indication for operative reduction and fixation. Great majority are treated nonoperatively with splint spanning wrist and hand for 3 to 4 weeks.
Prognosis and return to play: Stable fractures not requiring surgery usually require 3 to 4 weeks of splinting while swelling and soreness subsides. Transition to hand-based splint may allow skilled position players to return within 1 to 2 weeks depending on level of discomfort. Surgically treated fractures require 2 to 4 weeks before range of motion and pain allow return to sports.



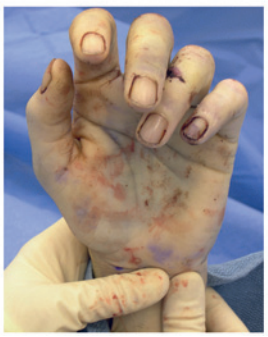
In fractures of metacarpal neck, volar cortex often comminuted, resulting in marked instability after reduction, which often necessitates pinning.



Transverse fractures of metacarpal shaft usually angulated dorsally by pull of interosseous muscles.



Oblique fractures tend to shorten and rotate metacarpal, particularly in index and little fingers because metacarpals of middle and ring fingers are stabilized by deep transverse metacarpal ligaments.

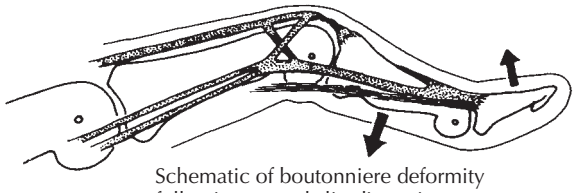


Clinical scissoring due to malrotation of 4th and 5th metacarpal fractures.

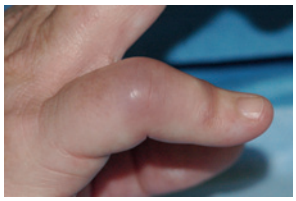
Figure 45-5 Metacarpal Fracture.

Injuries to the PIP Joint Central Extensor Slip Insertion/Boutonniere Deformity

Description: Central slip of the extensor tendon inserts on the dorsal base of the middle phalanx. Disruption of this insertion results in loss of full active PIP joint extension. Over time, adjacent lateral band tendons migrate and become fixed palmar to the axis of rotation of the PIP joint, resulting in a boutonniere deformity characterized by PIP flexion and DIP hyperextension (Fig. 45-6).
Mechanism of injury: May occur with forced passive PIP flexion against active extension through the central slip tendon, resulting in avulsion. Volar PIP dislocations often result in avulsion of the central slip insertion; dorsal PIP laceration through the central slip (hockey skate) will result in boutonniere deformity if left untreated.
Presentation: Often has a subtle presentation, and a high index of suspicion is required. Nonspecific presence of swelling is usually present about the PIP joint, and the joint may be maintained in

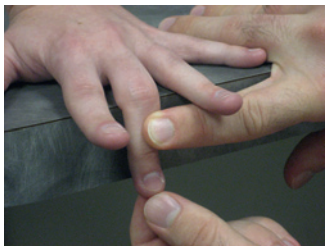


Schematic of boutonniere deformity following central slip disruption

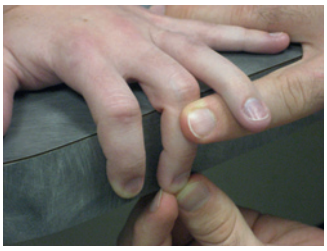


Swelling following closed central slip disruption

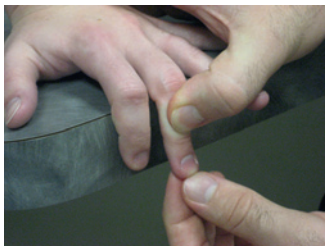
Elson test



PIP flexed over table edge, patient instructed to actively extend PIP.



While examiner resists active extension, DIP can be passively flexed without resistance.



If central slip is avulsed, the DIP cannot be passively flexed and may even extend slightly with resisted active PIP extension (positive Elson test).

Figure 45-6 Injuries to the PIP Joint Central Extensor Slip Insertion/Boutonniere Deformity. *(Illustration reprinted with permission from Green D, Hotchkiss R, Pederson W (eds): Green's Operative Hand Surgery, 5th ed. Philadelphia: Churchill Livingstone, Elsevier, 2005.)*

slight flexion (see Fig. 45-6). However, in the immediate phase, the patient may be able to maintain PIP extension through the lateral bands, which have not yet migrated palmarly.

Physical exam: Palpate for tenderness directly over the central slip insertion on the dorsal middle phalangeal base. Collateral ligament tenderness may also be present, but tenderness at the central slip insertion should raise concern. Sensitive method to assess disruption of the central slip is the Elson test. Digit is placed on a table with the PIP joint flexed over the edge. While the proximal phalanx is held firmly flat on the table by the examiner, the patient attempts active extension of the PIP joint. Any pressure felt by the examiner on the dorsum of the middle phalanx suggests some continuity of the central slip insertion. If the central slip has torn and retracted proximally, there will be loss of active PIP extension and also decreased passive DIP flexion (normally floppy and supple) during the attempt (see Fig. 45-6).

Differential diagnosis: Nonspecific swelling around PIP joint could represent anything from mild collateral ligament injuries to periarticular fractures.

Diagnostics: PA and lateral plain x-rays are needed to rule out periarticular fractures or avulsion of dorsal margin of middle phalangeal base.

Treatment: For closed injuries, seen early (within 2 to 3 weeks), the lateral bands may not have yet become fixed in a position palmar to the axis of rotation. Closed treatment with *full-time* PIP splinting in full extension and active DIP flexion/extension exercises often results in healing of the central slip to its insertion bed while preserving lateral band mobility; continue for 6 to 8 weeks. Open lacerations require primary surgical repair of the tendon followed by protection of the repair with full PIP extension splinting and active DIP motion as discussed earlier. Delayed presentations with fixed boutonniere postures are exceedingly difficult to treat. Salvage procedures in the form of terminal extensor tendon releases and even PIP fusion may be required, depending on the degree and rigidity of the contracture.

Prognosis and return to play: Prognostic factor is prompt diagnosis and initiation of closed treatment. Digit must be protected from PIP flexion for at least 6 weeks. Fixed boutonniere deformities have a poor prognosis for regaining full active motion.

Flexor Tendon Laceration

Description: Transection of flexor tendon at wrist, hand, or finger.

Mechanism of injury: Laceration to palmar aspect of wrist, hand, or digit, often from cleat, spikes, or blade of skate.

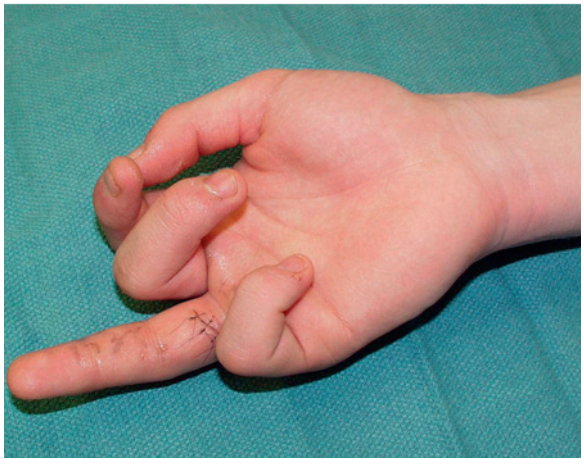
Presentation: Skin laceration with inability to flex digit(s) distally (Fig. 45-7). Resting cascade of digit in question will demonstrate less resting flexion of the DIP or PIP joint than surrounding digits.

Physical exam: Laceration with no active flexion of digit at DIP and possibly PIP joint**.** Neurovascular status may also be abnormal (digit pale, diminished capillary refill) if digital neurovascular bundles also lacerated. Two-point sensory may be altered with transaction of digital nerve.

Differential diagnosis: Open fracture or simple laceration.

Diagnostics: PA and lateral x-ray of injured area.

Treatment: Irrigation and dressing of wound with skin closure. Tendons require surgical repair within 10 days. No option for nonoperative treatment.



Typical presentation of lacerated FDS and FDP tendons in finger.

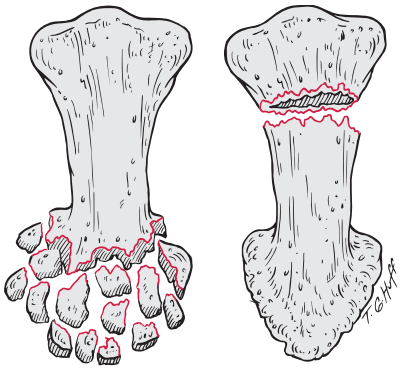
Figure 45-7 Flexor Tendon Laceration.

Prognosis and return to play: Flexor tendon rehabilitation requires 6 to 8 weeks of extensive ROM therapy and up to 12 weeks for strengthening before returning to sports that require gripping and grasping.

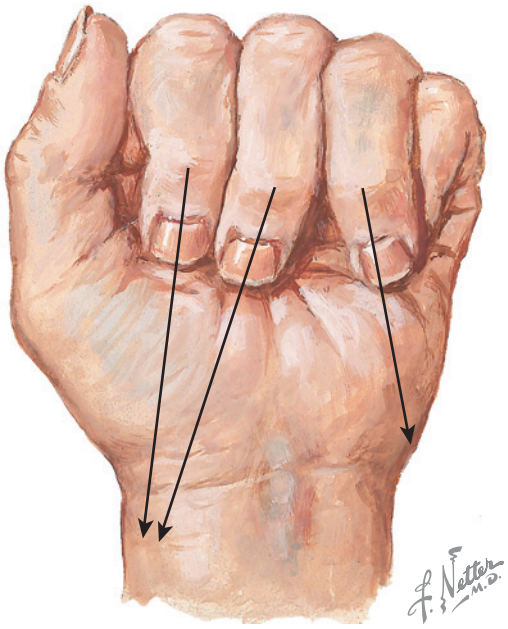
Phalangeal Fractures

Description: May occur as simple transverse patterns with minimal displacement or as more complicated, comminuted patterns with marked displacement and associated soft tissue injury. In the distal phalanx, tuft fractures occur in the very tip of the bone, usually resulting from some form of crush to the fingertip, and often have a stellate pattern; distal phalangeal shaft and base fractures may result from a crush or bending force, and can be more unstable than tuft fractures (Fig. 45-8).

Mechanism of injury: Excessive axial, torsional, or bending forces. Rate and direction of loading will determine the fracture pattern. Sudden axial load from a ball to crush from another competitor's foot can result in a variety of patterns, such as periarticular fractures or fracture-dislocations.



Comminuted Transverse



Results of healing ring finger in rotational malalignment

Figure 45-8 Phalangeal Fractures. *(Illustration reprinted with permission from Browner B, Jupiter J, Levine A, Trafton P: Skeletal Trauma: Basic Science, Management, and Reconstruction, 3rd ed. Philadelphia: Saunders, Elsevier, 2002.)*

Presentation: Nondisplaced fracture—pain, swelling, and associated apprehension to movement. Fractures from crush injuries will often have a concomitant soft tissue injury component. Tuft fractures or displaced shaft fractures usually present with a subungual hematoma. Fractures with more comminution or displacement will likely result in some angular deformity of the digit. Be leery of any open wound around a displaced phalangeal shaft fracture, because this may communicate with the fracture site.

Physical exam: Inspect soft tissues, checking for open wounds or subungual hematoma. If the base of the nail plate has flipped out of the nail fold, this frequently represents a distal phalangeal shaft or metaphysis open fracture through the nail bed. Point tenderness to palpation over the phalanx should raise suspicion. Angulation likely represents a fracture or fracture-dislocation; this is more easily detected if the patient can flex the digits (see Fig. 45-8). Open wound over the radial or ulnar neurovascular bundle mandates two-point discrimination sensory testing along that side of the fingertip. Perfusion of the digit should also be verified in settings of injuries with more energy imparted or displacement (crush injuries).

Differential diagnosis: Dislocations of the MP or interphalangeal (IP) joints may be mistaken for phalangeal fractures; clarified by plain radiographs.

Diagnostics: PA and lateral plain films of the digit should suffice for determination of fracture pattern and differentiation from joint involvement.

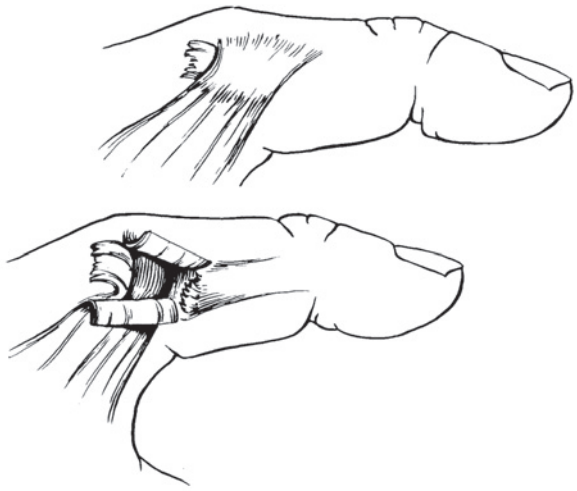
Treatment: Potentially open fracture or nonperfused digit requires emergent surgical treatment and withdrawal from competition. Otherwise, the injured digit may be bandaged in a bulky dressing together with the other digits to allow return to play. Formal radiographic and clinical evaluation of closed, perfused injuries should occur within 48 hours. **Nondisplaced, stable fractures** can usually be treated with custom splinting and gentle active assist ROM exercises over 2 to 3 weeks. **Displaced fractures** need to be reduced; digital block anesthetic is usually sufficient. Anything other than complete anatomic reduction of a transverse fracture is unstable and will require pin or plate and screw fixation.

Prognosis and return to play: Main complications usually stiffness and angular deformity. Return to play is dictated by fracture stability (or fixation rigidity), union, and participation requirements. If fixation requires pin placement across a joint, the athlete should not be allowed to participate in any sport requiring forceful grip or ball handling until the pin has been removed. Likewise, even the most stable plate and screw constructs are prone to failure under such loads, and return to activity before at least early callus formation after the third week invites fixation failure and deformity.

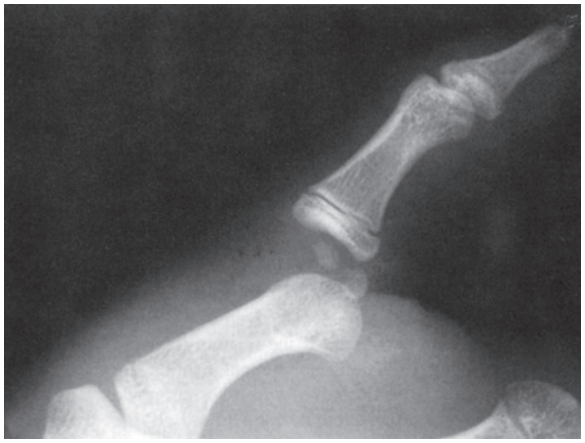
Thumb Metacarpophalangeal Joint Ligament Injuries

Description: Often referred to as "skier's" or "gamekeeper's" thumb (for ulnar collateral ligament [UCL] failure) and "reverse gamekeeper's" thumb (for radial collateral ligament [RCL] failure), these injuries can result in chronically impaired and painful grip when left untreated. UCL failure can occur anywhere along the length of the ligament; detachment usually occurs with or without avulsion fracture at the site of insertion onto the base of the proximal phalanx (Fig. 45-9). The tendon or aponeurosis of the adductor pollicis can become interposed between the torn ligament and its insertion on the phalanx, preventing healing; this is called a *Stener lesion* (see Fig. 45-9).

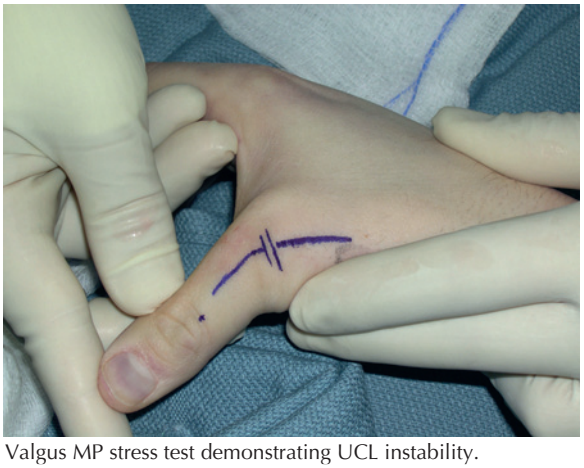
Mechanism of injury: UCL injuries (occur 10 times more frequently than RCL injuries) result from sudden valgus force to the thumb, frequently seen after a fall onto the thumb or impact from a ball. RCL injuries result from sudden varus force to the thumb, and are often underappreciated and underdiagnosed.



Schematic of stener lesion.



Radiograph showing UCL avulsion fracture.



Valgus MP stress test demonstrating UCL instability.

Figure 45-9 Thumb Metacarpophalangeal Joint Ligament Injuries. *(Illustration reprinted with permission from DeLee J, Drez D, Miller M: DeLee & Drez's Orthopaedic Sports Medicine: Principles and Practice, 2nd ed. Philadelphia: Saunders, Elsevier, 2002.)*

Presentation: Painful, impaired grip. MP joint usually diffusely swollen in the subacute phase; resting angular deformity may or may not be visible at the MP joint. In RCL injuries, as swelling subsides, metacarpal head may appear prominent because of "sagging" of the radial aspect of the joint from capsuloligamentous incompetence.

Physical exam: In acute and subacute phases, tenderness to direct palpation over the injured ligament is present. Assess UCL stability with passive valgus stress of the extended proximal phalanx while stabilizing the metacarpal with the other hand (see Fig. 45-9). Although it has been suggested that pain and deviation greater than 30 degrees is indicative of a UCL tear, this laxity should be compared with the contralateral uninjured thumb. Many patients with inherent ligamentous laxity will have that degree of mobility in that joint; repeated with the phalanx in 30 degrees of flexion to isolate the UCL and eliminate stability that may be provided from an intact volar plate. RCL is evaluated in a similar manner, except the phalanx is passively deviated in the ulnar direction while the metacarpal is stabilized.

Differential diagnosis: Ligament injuries, fractures.

Diagnostics: PA and lateral plain x-rays required to evaluate for fractures. In RCL injuries, the lateral view may reveal sagging of the proximal phalanx relative to the metacarpal. MRI may be helpful in clarifying both UCL and RCL injuries. Stener lesions have also been identified in MRI, which is an important feature in determining treatment.

Treatment: For UCL injuries, the degree of tear, avulsion fracture displacement, and presence or absence of a Stener lesion is considered. Partial tears with minimal relative laxity can usually be managed by thumb spica cast (including the IP joint) immobilization for 4 to 6 weeks, followed by a 2-week period of splinting and ROM exercises. A Stener lesion requires surgery to remove the interposed tendon and repair the ligament to its insertion bed. Controversy surrounds complete tears in which a Stener lesion is not apparent. Many advocate direct primary repair, whereas others may recommend casting. There is no consensus regarding management of acute RCL injuries. However, if sagging of the radial side of the joint is present clinically or on radiographs, operative repair of the radial collateral complex (including associated capsular hood tear) over immobilization alone is preferred.

Prognosis and return to play: If ligament complex heals, return to full activity within a couple of months. Participation in a cast may begin, however, as soon as the wound is stable. A small portion of patients may continue to have pain with forceful grip, even with a clinically stable repair requiring more prolonged splinting.

Metacarpophalangeal (MP) Dislocation

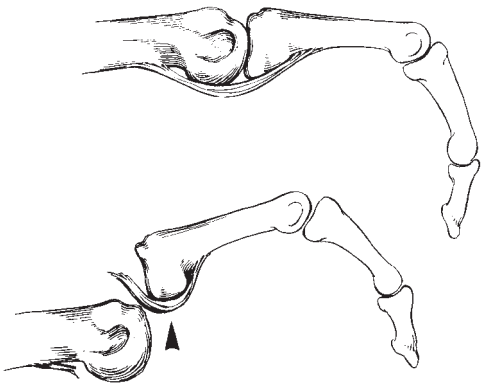
Description: Complete dislocation of proximal phalanx (usually dorsal) in relation to the corresponding metacarpal, classified as either *simple* or *complex*. Simple dislocation is one that can be manually reduced; complex dislocation requires open reduction techniques.

Mechanism of injury: Hyperextension injury to a digit at the level of the MP joint. In both simple and complex dislocations, the volar plate detaches from its metacarpal attachment and retains its attachment on the proximal phalanx. However, in a complex dislocation, the entire volar plate is interposed in the MP joint, preventing manual reduction (Fig. 45-10).

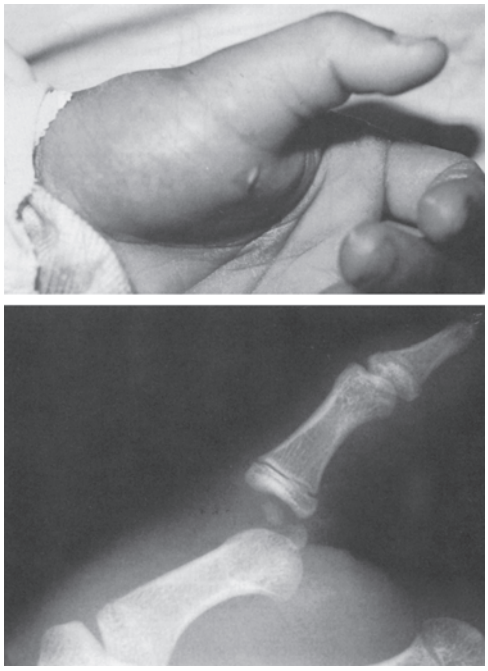
Presentation: Acute pain, swelling, and hyperextension of the MP joint; border digits are most commonly affected.

Physical exam: Differentiate between a simple and complex dislocation. Affected joint is typically hyperextended to about 90 degrees in a simple dislocation, whereas it exhibits less hyperextension in a complex dislocation. A volar "dimple" near the region of the A1 pulley is a pathognomonic finding of a complex dislocation. If a dimple is present, one must take care to evaluate the overlying skin for threatened skin or pressure necrosis. A careful neurovascular exam of the digit should be performed.

Differential diagnosis: A fracture at the level of the MP joint may occur in conjunction with a dislocation. Less likely is a "locked MP joint," characterized by sudden loss of active and passive MP extension with little or no loss of flexion.



Schematic of volar plate entrapment in irreducible dorsal dislocation.



Complex thumb MP dislocation, showing joint space widening and entrapped sesamoids within joint.

Figure 45-10 Metacarpophalangeal (MP) Dislocation.

Diagnostics: Physical examination is the mainstay in initial diagnosis. AP, lateral, and oblique radiographs are *required*, because up to 50% of such injuries have an associated fracture. Simple dislocation will demonstrate the proximal phalanx hyperextended 90 degrees; complex dislocation may demonstrate excessive joint widening or entrapped sesamoids (see Fig. 45-10). Postreduction radiographs must be obtained to assure adequate, congruent reduction and evaluate for associated fractures.

Treatment: Closed reduction is the initial treatment strategy. Technique includes wrist flexion and volar translation of the base of the proximal phalanx in relation to the metacarpal head. Excessive hyperextension and axial traction should be avoided because it may convert a simple dislocation to a complex dislocation by flipping the volar plate dorsal to the metacarpal head. The lumbrical tendon, flexor tendon, or volar plate may block reduction. For an irreducible dislocation, an open reduction must be performed, using either a dorsal or volar approach.

Prognosis and return to play: Following successful closed reduction, a dorsal blocking splint is used and early motion is encouraged. Return to play depends on the demands of the specific athlete and the ability to immobilize the digit during play. In open reduction, play may not resume until early initial wound healing is confirmed.

Carpometacarpal (CMC) Dislocation

Description: Complete dislocation of the metacarpal in relation to the adjacent carpus; metacarpals dislocate dorsal relative to carpus.

Mechanism of injury: High-energy axial load on the metacarpals. Fifth and fourth rays may dislocate in isolation because they possess a more mobile articulation with the hamate. However, the index and long fingers typically dislocate only in high-energy injuries where all four CMC joints are dislocated.

Presentation: Significant dorsal swelling, sometimes referred to as a "balloon hand." With lower-energy isolated dislocations, the dorsal swelling may be less pronounced and is typically focused over the ulnar digits.

Physical exam: Inspection for an open injury. Careful palpation of each digit is required to look for associated fractures. A neurovascular exam is imperative and there should be a high index of suspicion for an impending compartment syndrome of the hand.

Differential diagnosis: Metacarpal fractures, severe contusion, and carpal dislocations.

Diagnostics: AP, lateral, and oblique radiographs are the mainstay. Radiographic evidence of dislocation is rather obvious when all four CMC joints are involved; however, radiographic findings are more subtle when dislocations occur in isolation. A "true lateral" of the involved digit must be obtained. Lateral views of small and ring fingers require about 30 degrees of pronation at the wrist to obtain; lateral views of the index and long finger may require more supination. Distraction or traction views may be of utility.

Treatment: These injuries are inherently unstable, although isolated dislocations may be stable after a closed reduction maneuver is performed. Reduction maneuver is performed by applying axial traction in conjunction with a palmar-directed force on the dorsal base of the affected metacarpal. Most injuries require closed or open reduction combined with internal fixation (usually temporary pins).

Prognosis and return to play: Because of the unstable nature of these injuries, return to play involving gripping or hand contact is after 6 to 8 weeks to allow for sufficient healing. In an isolated CMC dislocation that is stable upon closed reduction, return to play may be earlier with protective bracing or casting.

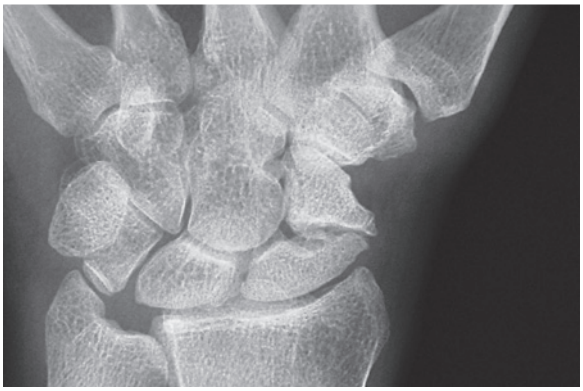
Scaphoid Fractures

Description: Common injuries in sports. A precarious retrograde vascular supply renders the scaphoid prone to slow healing and nonunion, especially in fractures of the waist or proximal pole. Left untreated, scaphoid fractures in adolescents or young adults invariably progress to symptomatic nonunion and secondary arthritic collapse (Fig. 45-11). In isolation, this fracture results in no visible deformity and often no more pain than a minor joint sprain. As such, the diagnosis is frequently missed or the player often does not complain of wrist pain until weeks or months after the precipitating injury.

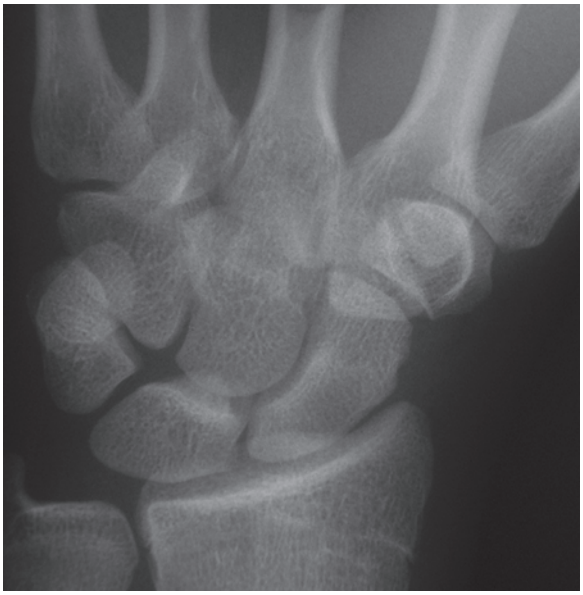
Mechanism of injury: Most commonly results from a fall on the outstretched hand with an extension load across the distal radius and carpus. The proximal pole is locked in the scaphoid fossa of the radial articular surface, resulting in failure of the waist or proximal pole of the scaphoid. A less frequent mechanism is an axial load through the carpus.

Presentation: Radial-sided wrist pain, usually following a fall or sudden axial load through the wrist. Usually no visible deformity, including lack of swelling. Patient will often still be able to move the wrist in the mid-range of motion, especially in more delayed presentations.

Physical exam: Tenderness to palpation directly over the fracture. In waist fractures, this is in the anatomic snuffbox. However, the



Radiograph of scaphoid nonunion with collapse and secondary osteophyte formation.



Normal x-ray of acute fracture.

Figure 45-11 Scaphoid Fractures.

proximal pole should be assessed by palpation at the scapholunate joint just distal to the dorsal radial tubercle. The distal scaphoid pole can be palpated at the scaphotrapezial joint, deep to the intersection of the flexor carpi radialis tendon and wrist flexion crease. Forced passive wrist extension also usually produces pain at the fracture site.

Differential diagnosis: Distal radius fractures, injuries to the scapholunate ligament, perilunate or lunate dislocations (markedly more painful with visible deformity).

Diagnostics: PA, oblique, lateral and clenched fist/ulnar deviation radiographs are essential. If plain films are negative, bone scan or MRI may demonstrate the presence of a nondisplaced fracture, although MRI may offer more specificity (see Fig. 45-11). However, simple repeat radiographs 2 to 3 weeks later following cast immobilization may reveal bone changes around a previously unrecognizable fracture. To determine displacement and potential surgical planning, CT scan offers the best bony detail.

Treatment: Displaced fractures are generally managed with arthroscopic or open reduction and internal fixation, as are fractures associated with other carpal fractures or ligament injuries. With displacement, the union rate precipitously declines with immobilization alone. Controversy surrounds management of truly nondisplaced scaphoid waist fracture, because similar union rates and functional results have been demonstrated with immediate cast immobilization compared to immediate percutaneous screw fixation. Immediate fixation may allow players to return to competition sooner while casted than without internal fixation. Proximal pole fractures, even when nondisplaced, are often managed primarily with screw fixation, owing to the poor vascularity of that portion of the bone and high nonunion rate.

Prognosis and return to play: Union rate with immobilization of nondisplaced or impacted distal pole fractures is greater than 90%. Nondisplaced waist fracture union rate with immobilization ranges from 85% to 95%. Proximal pole fracture union rates drop significantly. Return to play is dictated by fixation stability and degree of fracture union. With anatomic reduction and screw fixation, a player may be able to return to play *in a cast* within a few weeks, or once the wounds have adequately healed. Until union occurs, the wrist must be protected against forced flexion or extension.

Injuries to the Triangular Fibrocartilage Complex (TFCC)

Description: The TFCC is composed of the triangular fibrocartilage disk, dorsal radioulnar ligament (DRUL), volar radioulnar ligament (VRUL), extensor carpi ulnaris tendon subsheath, and the meniscal homologue (Fig. 45-12). It is the main structure providing stability to the distal radioulnar joint (DRUJ) throughout the full range of supination and pronation.

Classification: Traumatic (type I) and degenerative (type II) tears.

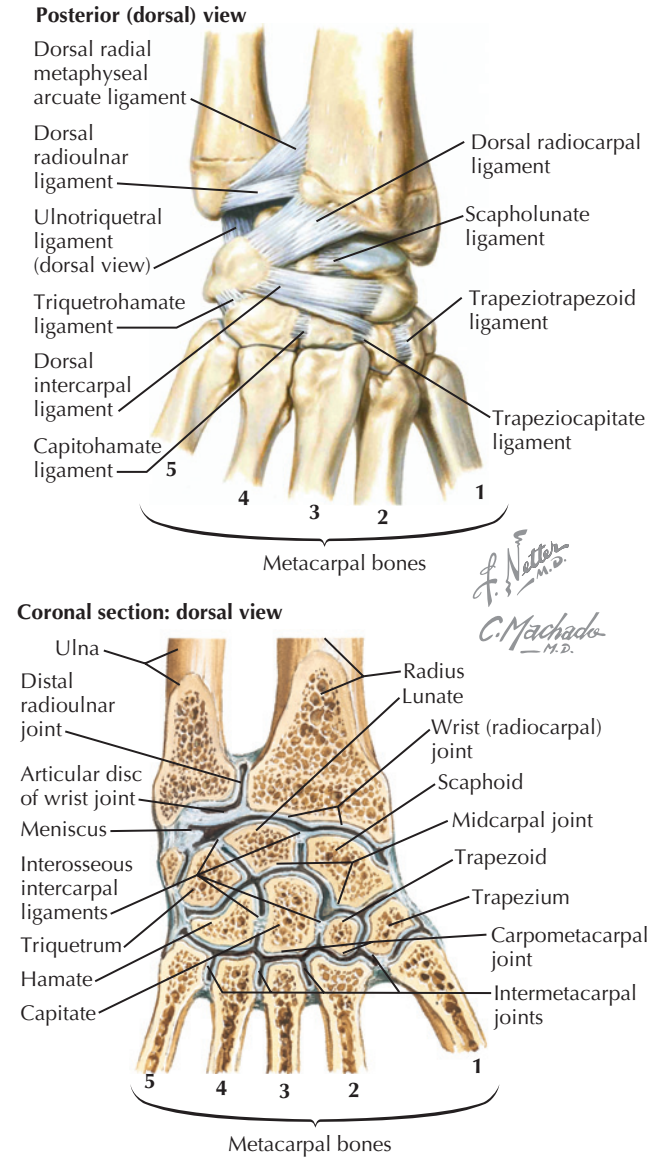
Mechanism of Injury: Most acute injuries result from a fall onto the outstretched hand. Degenerative tears of the central portion of the disk generally occur in the absence of a single inciting event.

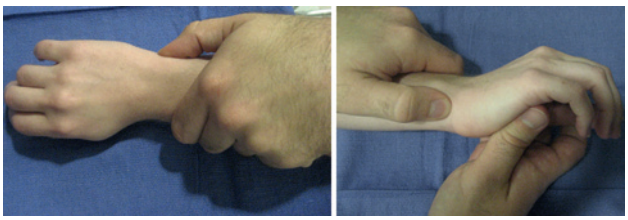
Presentation: Persistent ulnar-sided wrist pain, often with swelling, since the event. Swelling is often evident in the ulnar fovea. If there is significant disruption to the ligaments or their deep insertion at the base of the ulnar styloid, visible prominence of the ulnar head will occur with the DRUJ instability. Chronic, untreated partial traumatic tears or degenerative tears may present as recurring ulnar-sided wrist pain during activities requiring vigorous wrist motion or loading (push-ups, golf, tennis, cycling).

Physical exam: Pain or limitation with supination and pronation. Palpation tenderness is usually present over the ulnar fovea and perhaps also over the dorsal aspect of the TFCC, just distal to the ulnar head (see Fig. 45-12). DRUJ should be passively stressed by holding the distal radius in one hand and the distal ulna in the other while translating them relative to each other in neutral rotation, supination, and pronation. Pain and/or palpable "click" with the maneuver may also be noticed. Dorsal tenderness over the lunatotriquetral ligament may suggest an injury to that structure. Passive ulnar deviation of the pronated wrist that results in ulnar-sided pain is also suggested of a TFCC tear.

Differential diagnosis: Carpal fractures, lunatotriquetral ligament injuries, extensor carpi ulnaris tendon instability, and ulnocarpal synovitis without actual associated TFCC tear can also result in pain in this region.

Diagnostics: Plain PA and lateral radiographs are useful to evaluate for fractures of the distal ulna or carpus, subluxation of the DRUJ, calcification of the TFCC (suggestive of pseudogout), degenerative changes of ulnar head and ulnar corner of lunate (suggestive of ulnocarpal impaction), and ulnar variance. Positive ulnar variance occurs with the ulna longer than the radius and is more associated with ulnocarpal impaction and degenerative TFCC tears. MRI may be useful. Arthrogram may reveal tears in the complex, but is being employed less frequently. The current gold standard for TFCC evaluation is arthroscopy, which is obviously more invasive but also has the potential for





Palpation over the ulnar fovea.

Figure 45-12 Injuries to the Triangular Fibrocartilage Complex (TFCC).

offering treatment at the time of diagnosis. Isolated deep tears of the TFCC insertion may not be visible on arthroscopic evaluation and may be better seen with an MR arthrogram.

Treatment: Many TFCC tears, both traumatic and degenerative, may be minimally symptomatic and not require treatment. If the athlete is in season and does not want to be out for a possible 6 to 10 weeks following a TFCC repair, a steroid injection into the TFCC through the ulnar fovea may offer enough pain relief to allow continued participation until potential surgical intervention. In stable joint, pain may resolve with time and observation alone. Instability of the joint is best treated with surgical repair. Peripheral, traumatic tears occur in the vascularized portion of the triangular fibrocartilage and are usually reparable. Central and degenerative tears are relatively avascular (similar to the meniscus in the knee) and do not heal following repair. Degenerative tears may respond to debridement with associated shortening (either arthroscopic or ulnar shaft osteotomy) of the positive or neutral ulna.

Prognosis and return to play: Athletes may be able to return to play if a steroid injection ameliorates their symptoms and there is absence of instability. Repair of the torn TFCC will require a minimum of 4 weeks of cast immobilization followed by a period of gradual motion exercises and progressive strengthening over the next 2 to 4 weeks. Return to competition in that setting is dependent on the demands of the particular sport.

Perilunate Injuries

Description: ***(scapholunate tears, perilunate and lunate dislocations)*** injuries involving combinations of ligaments and bones surrounding the lunate.

Mechanism of injury: Wrist hyperextension and ulnar deviation during axial loading.

Physical exam: Localized wrist pain, point tenderness, limited motion, and sometimes median nerve dysfunction. With lunate dislocation, bone may be palpable at wrist flexion crease.

Differential diagnosis: Scaphoid fracture, distal radius fracture.

Diagnostics: PA, lateral, and scaphoid x-ray of wrist. With normal x-rays and clinical suspicion of injury, an MRI of wrist is indicated.

Treatment: All acute perilunate injuries require surgical repair of the ligaments and/or bony elements. Combinations of K-wires and screws are used.

Prognosis and return to play: K-wires are removed at 8 weeks and cast immobilization is required for 12 weeks. Most injuries take 4 to 5 months before range of motion and strength stabilize.

Distal Radius Fractures

Description: Some of the most common upper limb injuries seen in orthopedics. In the athlete with open physes, fractures through the growth plate are particularly common following falls onto the outstretched hand; seen commonly in snowboarders. Fracture patterns may be limited to the metaphysis or also involve the radiocarpal or ulnocarpal articulations. Associated tears of the TFCC may result in instability of the DRUJ. Degree of comminution will be determined by the quality of the bone and energy imparted to it.

Mechanism of injury: Fall onto the outstretched hand is most common mechanism.

Presentation: Nondisplaced fractures may have no visible deformity other than mild swelling with complaints of wrist pain following a fall. The more common dorsally displaced patterns may demonstrate a "dinner fork" deformity just proximal to the carpus. Patients will be apprehensive regarding any motion of the wrist. These injuries are generally more painful than scaphoid fractures, so most patients will be forced to withdraw from play.

Physical exam: Assessment of skin integrity and neurovascular status. Open fractures, seen with higher energy injuries with greater degrees of displacement at the fracture site, are fortunately uncommon in athletic competition. With greater dorsal displacement at the fracture site, the median nerve is at greater risk for traction injuries. Sensation of the radial three and a half digits should be assessed prior to any manipulation. In nondisplaced fractures, the patient will reliably be tender to palpation directly over the fracture site.

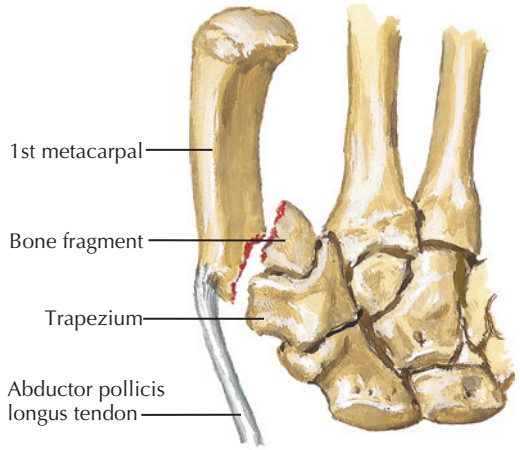
Differential diagnosis: Carpal fractures and ligament injuries should always be considered; usually differentiated by direct palpation, because patient will usually be tender over the fractured radial metaphysis as opposed to the carpus. These injuries

can occur in combination, however, and tenderness over the scaphoid as well as the radius should lead to assessment with appropriate plain films.

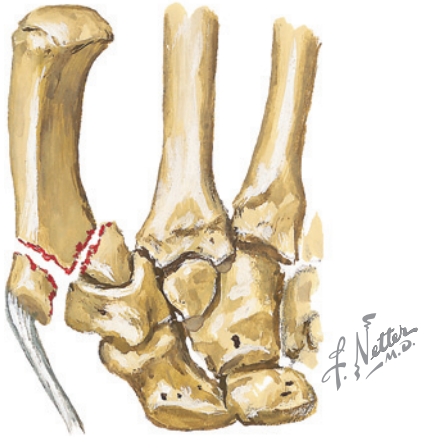
Diagnostics: PA and lateral plain films usually sufficient. CT scanning may be useful for surgical planning for fractures with intra-articular displacement or comminution.

Treatment: Wrist is splinted in neutral position. For fractures with obvious clinical displacement, there is little to be gained from immediate manipulation or attempted reduction prior to obtaining plain films or having adequate analgesia. Supportive splinting of the wrist should be applied until then. Common guidelines for accepted displacement of extra-articular fractures in skeletally mature individuals are no more than 10 degrees of dorsal tilt of the artricular surface. Most practitioners accept no more than 2 mm of gap or 1 mm step-off of the joint surface. If the reduction cannot be maintained with closed manipulation and splinting, open reduction internal fixation (ORIF) is recommended. For nondisplaced or minimally displaced fractures, ORIF may allow earlier range of motion and use of a removable splint. However, this has not been shown to result in long-term superior motion, strength, or function.

Prognosis and return to play: Participation in contact sports or load bearing of the fractured radius is generally precluded until stable union is evident, minimum of 4 to 6 weeks, depending on patient age and fracture pattern. Restoration of normal anatomy allows the best prognosis in skeletally mature individuals, whereas younger patients with open physes may still have some remodeling potential.



Type I (Bennett fracture). Intraarticular fracture with proximal and radial dislocation of 1st metacarpal. Triangular bone fragment sheared off.



Type II (Rolando fracture). Intraarticular fracture with Y-shaped configuration.

Figure 45-13 Bennett's and Rolando's (First MC Base) Fractures.

Bennett's and Rolando's (First MC Base) Fractures

Description: Intra-articular fractures of the base of the thumb metacarpal. A Bennett's fracture pattern includes a constant piece on the volar-ulnar aspect of thumb metacarpal base that is held in normal anatomic position to the adjacent trapezium by the strong *deep anterior oblique (beak) ligament*. A Rolando fracture pattern has the same volar-ulnar fragment, but also has an associated dorsal fragment, thus is often described as a T-shaped or Y-shaped thumb metacarpal base fracture (Fig. 45-13).

Mechanism of injury: Result of an axially directed force through the shaft of the thumb metacarpal.

Presentation: Acute pain and swelling localized to the base of the thumb metacarpal after a contact or impact injury. Pain is associated with any attempt to move the thumb.

Physical exam: Pain and swelling localized to the base of the thumb metacarpal.

Differential diagnosis: Thumb metacarpal shaft fracture and thumb trapeziometacarpal dislocation must also be considered.

Diagnostics: Accurate injury classification requires quality radiographs. To obtain a true lateral, the palmar surface of the thumb is placed on the flat plate and the wrist is pronated 20 degrees. The beam is then directed 15 degrees in a distal to proximal direction, centered over the thumb metacarpal base. Traction views may also be of some utility.

Treatment: Fracture patterns are unstable and surgical management is the mainstay of treatment to maintain congruity of the thumb carpometacarpal joint. Bennett fractures are often successfully managed with closed reduction and percutaneous pin fixation, whereas Rolando fractures typically require open reduction with internal fixation techniques.

Prognosis and return to play: Overall prognosis is good if articular congruity and length of metacarpal is restored. A typical course of 6 weeks of immobilization is required prior to gradual return to play with protective bracing, depending on the demands of the athlete.

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Thorax and Abdominal Injuries

46

Cindy J. Chang and Sameer Dixit

INTRODUCTION

General Overview

- Injuries to the thorax and abdomen are more often seen in sports involving sudden deceleration and impact (football, ice hockey, skiing, and snowboarding).
- Early recognition and management of these potentially life-threatening injuries is imperative. Repeated assessment and a high index of suspicion are essential for accurate evaluation. Once a severe injury has been recognized, fundamentals of emergency treatment and stabilization should be initiated until transfer can be made to a hospital.
- Torso injuries often overlap with injuries to the extremities (e.g., traction apophysitis of the iliac crest commonly presents as lower abdominal pain). Many shoulder conditions can radiate to the thorax; similarly, thoracic and abdominal conditions can radiate to the extremities, causing confusion as to the source of symptoms.

Anatomy and Physiology Issues

- Combination injuries in the upper abdomen can be divided into three regions.
  - Midline region: Left lobe of the liver, pancreas, duodenum, transverse colon, small bowel and mesentery, aorta, inferior vena cava, sternum, lower ribs, and heart.
  - Right region: Liver, right kidney, right adrenal gland, right hemidiaphragm, right lung contusion, pneumothorax or hemothorax, and rib fractures.
  - Left region: Other paired organs but the spleen instead of the liver.
- In sports, these organs can suffer damage usually resulting from compressive forces (e.g., a tackle or a bicycle handlebar) that forces a solid or viscus organ against the fixed spine.
- Deceleration forces and penetrating injuries are more uncommon in athletics, although "almost penetrating" injuries can be caused by a hockey stick or ski pole without causing a wound.
- The abdominal organs in children are more susceptible to injury from trauma because of their relative position (more anterior and lower due to the more horizontal nature of the diaphragm), the still developing abdominal musculature, and the pliable nature of the cartilaginous ribs.
- **"Getting the wind knocked out"** is a more common occurrence than significant trauma to visceral organ. An unguarded blow to the epigastric region causes temporary reflex spasm of the diaphragm. Loosening of restricting garments and flexion at knees and hips usually restores normal respiration. Danger of intra-abdominal injury exists, so careful observation and follow-up examinations are necessary.

Epidemiology, Injury Statistics, and Sports-Specific Issues

Myocardial injury: Myocardial injury may occur in up to 76% of patients sustaining blunt trauma to chest, with direct compression of heart between anterior chest wall and vertebral column.

Abdominal injury: A 1993 study looking at serious pediatric sports injuries found that abdominal injuries accounted for 7% of those hospitalized, whereas fractures were the most frequent reason for hospitalization (77%). The most frequent cause of abdominal injury in children is a bicycle accident.

Thoracic and pulmonary injuries: There is limited data defining the incidence of thoracic and pulmonary injuries in sport. Blunt chest trauma is the most common cause of both cardiac and pulmonary contusions; more than 90% of those injuries are the result of motor vehicle accidents. Pneumothoraces, with or without concomitant rib fractures, also are uncommon injuries in sports, but are increasingly reported. Spontaneous pneumothorax caused by the strenuous exertion of weightlifting has also been reported.

History and Physical Exam

History

- Accurate account of the events leading to the injury is important in establishing the diagnosis of thoracic and abdominal injuries.
- History and physical examination are sometimes unreliable, especially in children and if there is an altered level of consciousness.
- A seemingly minor trauma can be the cause of a delayed splenic rupture or other injury; careful history-taking including past injuries, surgeries, and illnesses is paramount.
- A previously undiagnosed preexisting condition such as inflammatory bowel disease or liver hemangioma can produce major clinical symptoms after minor trauma to the affected organ.
- Detailed history of the patient's pain and the use of a pain scale upon initial presentation and during serial evaluations are crucial. Examples include the PQRST principles of evaluation of pain, and a visual analog pain scale (Table 46-1).

Physical Exam

See Chapter 29 for a discussion of the cardiovascular exam; see Chapter 4 for a discussion of possible initial evaluation of a majority of injuries to the thoracic and abdominal region.

General Appearance and Vital Signs

- If thoracic and abdominal injuries are both present, the thoracic injuries are usually more symptomatic and will distract the attention from the abdominal pain, which is usually less localized and specific.
- Abdominal pain can be vague and diffuse over the entire abdomen, or localized to a quadrant. Abdominal pain is sensitive for the presence of injury, but not specific; 50% of those with

Table 46-1 PQRST EVALUATION OF PAIN

P	**Palliative/provoking**
	What causes it?
	What makes it better?
	What makes it worse?
Q	**Quality**
	How does it feel, look, or sound?
	How much of it is there?
R	**Radiation**
	Where is it?
	Does it spread?
S	**Severity**
	Does it interfere with activities?
	How does it rate on a severity of 1 to 10?
T	**Timing**
	When did it begin?
	How often does it occur?
	Is it sudden or gradual?

pain have no significant abdominal injury. Always examine the chest and spine when evaluating an abdominal complaint, and consider examining the inguinal and pelvic regions.
- Frequent monitoring of vital signs, including orthostatics, is important to gauge the cardiovascular status, and the respiratory rate, rhythm, and use of accessory respiratory muscles should be observed. If it is difficult to obtain a blood pressure by auscultation, deflate the cuff until you palpate the return of the brachial or radial pulse. The systolic pressure obtained by auscultation is approximately 10 mm Hg higher.

Inspection

- Observe the effort of breathing; listen for abnormal breathing sounds.
- Look for asymmetry, deformity, swelling, bruising, lacerations, and scars.
- Confirm that the trachea is midline and the chest has a normal anterior-posterior (AP) diameter.
- Evaluate abdominal contour and signs of increasing abdominal girth; observe for peristaltic or pulsating movements.
- Observe for splinting or guarding of the torso and upper extremities (UE), or a change in neck position.

Auscultation, Percussion, and Palpation

- Auscultate both the posterior and anterior chest, comparing each side for asymmetry. If breath sounds are decreased, the normal lung has been displaced by air (suspect pneumothorax) or fluid (hemopneumothorax or pleural effusion).
- Percuss both the posterior and anterior chest, comparing each side, and for normal diaphragmatic excursion with inspiration (symmetrical 3 to 5 cm). If hyper-resonant, suspect pneumothorax; if dull, suspect fluid. Percuss in all four quadrants; percuss the liver span (range 6 to 12 cm) and check for splenic enlargement.
- Auscultate before palpating because bowel sounds can change with manipulation. Listen for bruits over the aorta, renal, and iliac arteries. Start with gentle palpation to look for areas of tenderness, noting facial expression and presence of guarding. Follow with deep palpation to further delineate areas of pain or presence of abdominal masses. Include a complete evaluation of the genitourinary system.
- For bony and soft tissue injuries of the thorax, perform neck and shoulder range of motion (ROM) and strength tests, and palpate for crepitus, deformities, or presence of pain.

SPECIFIC INJURIES AND PROBLEMS

Chest Wall Injuries

Sternal Fracture

Description: Although fracture itself is not significant, incidence of associated intrathoracic trauma is high in acute injury.

Mechanism of injury: High-impact injuries, acute hyperflexion of cervicothoracic spine.

Presentation: Chest pain, localized tenderness over the sternum, shortness of breath.

Physical exam: Bruising, swelling, localized pain aggravated with deep inspiration, palpable or visible defect suggesting displacement.

Differential diagnosis/associated injuries: Stress fracture of sternum (develops when great stress placed on upper body during wrestling, golf), manubriosternal joint dislocation, sternoclavicular or costochondral injury, sternal contusion. Associated injuries include myocardial contusion, injury of internal mammary vessels, retrosternal and mediastinal hematoma, pulmonary laceration or contusion, rib and thoracic vertebrae fractures.

Diagnostics: Lateral chest film best to evaluate fracture (upper fragment usually displaced anteriorly over lower fragment); posterior-anterior (PA) radiograph to evaluate possible pneumothorax or widened mediastinum. Cervical, thoracic, and lumbar spine radiographs if flexion/compression mechanism. Computed tomography (CT) scan (axial cuts alone are not as sensitive; sagittal and coronal reconstruction views needed). Ultrasound (US) helps confirm radiologic diagnosis, but use remains limited in the United States. If intrathoracic trauma is suspected, electrocardiogram (ECG) and chest x-ray (CXR); consider repeat ECG in 24 hours.

Treatment: If displaced, reduction possible by lying supine and then lifting both arms above head while hyperextending thoracic spine at level just below scapular spines. **Because of high incidence of associated intrathoracic trauma, observation in hospital with cardiac monitor is advisable during reduction and for at least 24 hours afterward.** Displaced fractures may require open reduction with internal fixation, especially if there is respiratory compromise.

Prognosis and return to play: Nonunion of sternal fractures is rare, but suspect when pain persists over the sternum. If isolated fracture with no underlying thoracic injuries, progressive return to activity as limited by pain. Avoid contact sports until fracture has healed and pain resolved (range 6 to 12 weeks). If risk of reinjury is high, consider chest protector during sport.

Dislocation of the Sternoclavicular Joint (SCJ)

Description: Relatively infrequent, constituting less than 1% of somatic dislocations. Only 50% of the clavicular joint surface contacts the sternal articular surface. The posterior sternoclavicular ligament is stronger than the anterior ligament. Anterior more common than posterior disruption (ratio ranges from 9:1 to 20:1). Superior dislocation rare.

Classification:
- **Type I:** Sprain with no ligamentous damage or instability.
- **Type II:** Stretch or partial rupture of sternoclavicular and costoclavicular ligaments; joint is partially displaced.
- **Type III:** Dislocation with gross disruption of capsule and ligaments.

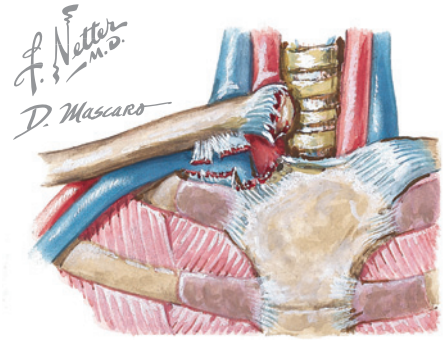
Mechanism of injury: Caused by direct or indirect trauma to shoulder girdle. In sports, injury most often seen in contact sports (martial arts, football, and rugby). For anterior dislocation, force applied at anterolateral aspect of shoulder or along abducted arm is transmitted along clavicle to SCJ, compressing and rolling the shoulder back and displacing the clavicle. For posterior dislocation, force applied to the posterolateral aspect of the shoulder when the arm is adducted and flexed is transmitted to SCJ, compressing and rolling the shoulder forward and displacing the clavicle (Fig. 46-1). Posterior dislocation can also result from direct blow on anterior aspect of medial end of clavicle.

Presentation: Severe pain, especially with any arm movement. Pain exacerbated by coughing, sneezing, or deep breathing. Because of spasm of neck muscles, head tilted toward injured side. Increased discomfort in supine position. Other symptoms may include hoarseness, dysphagia, dyspnea, numbness, and weakness or venous engorgement of the ipsilateral arm.

Physical exam: Bruising, pain, and significant swelling at joint. Noticeable prominence of medial end of clavicle in anterior dislocation. In posterior dislocation, loss of normal prominence, but often missed because of swelling. Obtain vital signs; observe ease of respiration and neurologic/vascular status of UE to rule out pressure on adjacent vital structures.

Differential diagnosis/associated injuries: SCJ sprain or subluxation; fracture of medial clavicle, fracture of medial physeal growth plate of clavicle (fusion occurs between ages 22 and 25). In patients younger than 25 years, SCJ dislocations are classified as Salter-Harris type I or II fractures. **More serious injuries are associated with post SCJ dislocations; 30% incidence of injury to vital structures traversing thoracic outlet, including major vessels of neck, brachial plexus, dome of pleurae, trachea, esophagus, and larynx. Of this group, 12.5% mortality rate.**

For posterior dislocation, force is applied to posterolateral aspect of shoulder when arm is adducted and flexed.



Posterior dislocation of sternoclavicular joint. Serious because of probable injury to trachea or vessels.

Figure 46-1 Posterior Dislocation of Sternoclavicular Joint.

Diagnostics: Obtain plain x-rays with 40-degree cephalic tilt view ("serendipity" view). Tube distance for children, 45 inches; for thicker-chested athletes, 60 inches. CXR to rule out pneumothorax. Axial CT images (3-mm cuts) have greater sensitivity and specificity and are imaging modality of choice for the SCJ; can differentiate fractures from dislocations, and allow assessment of the adjacent mediastinal structures if intravenous (IV) contrast used. Coronal plane paraxial CT reconstruction if superior component of dislocation is suspected. Occasionally arteriography/venography may be needed.

Treatment:

- **Type II injuries:** Avoid stress to joint for at least 3 to 4 weeks for adequate healing. Goal is to avoid increased symptomatic mobility at joint.
- **Type III injuries:** Immediate closed reduction for posterior dislocations with impending airway or bleeding complications. Otherwise reduce in operating suite under anesthesia with cardiothoracic surgeon present. Closed reduction method has a reported 80% success rate. Anterior dislocations can be reduced in the outpatient setting by applying gentle pressure over the displaced medial aspect of the clavicle. If closed reduction fails, open reduction can be considered if severe; however, an anteriorly displaced medial clavicle can often become relatively asymptomatic with activities of daily living. Chronic joint instability may cause pain and persistent functional limitation in active patients; this would be an indication for surgical intervention.
- **For chronic SCJ dislocation:** Evaluate for hypermobility of surrounding structures (including acromioclavicular and glenohumeral joints). Consider limited course of corticosteroid injection for symptomatic patients.

Prognosis and return to play: Despite 6 to 8 weeks of immobilization after successful reduction, healing can be inadequate and some SCJ instability may persist, with propensity for recurrent subluxation. Osteomyelitis of the clavicle can be a late complication. Operative stabilization can be difficult with unpredictable results. In the cases of recurrent stabilization failure or painful arthroses, resection of the medial clavicle is a salvage procedure.

Rib Fractures

Description: Most common serious injury of chest. Can be complete, incomplete, or stress fractures (Fig. 46-2). Often associated with other injuries, including other fractures and organ trauma. Nondisplaced fractures more common. If displaced, other injuries may include laceration of intercostal artery. Uncommon in children because thorax is more elastic and flexible.

Mechanism of injury:

- Blunt trauma: Force usually applied in anteroposterior plane; fractures located at posterior angles of fifth to ninth ribs.
- Direct force over small area of chest wall leads to fracture immediately beneath point of impact.
- Violent muscle contraction: **Floating rib** or **avulsion fractures** of attachments of external oblique muscle to lower three ribs; reported in baseball pitchers and batters. Forceful contraction, against significant resistance, of other muscles that attach to the ribs can also result in fractures.
- **Fracture of first rib:** Direct external trauma is rare cause because of protection of shoulder girdle. Other causes are indirect trauma from falling on outstretched arm, violent muscular pull (e.g., hyperabduction of arm), or repetitive stresses.
- **Stress fractures of the ribs:** Caused by excessive forceful muscular traction at attachments to ribs. Chronic opposing pulls of scalene muscles and upper digitations of serratus anterior may fracture **first rib** at its thinnest and most anatomically weak segment, where the subclavian artery crosses (subclavian sulcus), as with **weightlifting, pitching, other throwers.** Anterolateral stress fractures of **fourth ribs, fifth ribs,** and other ribs have been reported in **rowers** because of excessive action of serratus anterior muscle (see Chapter 83, Rowing).

Presentation: History of traumatic event with intense localized pain over involved rib. **If first rib injured, may complain of shoulder, scapular, or neck pain; may complain of abdominal pain if lower ribs (11th and 12th) involved.** With stress fractures, insidious onset of pain associated with specific activities, and possible radiation of pain. If fracture becomes unstable, pain is acute and knifelike. Pain aggravated by deep inspiration, coughing, or sneezing, and with twisting or side flexion (causing tension on fractured rib). May report dyspnea.

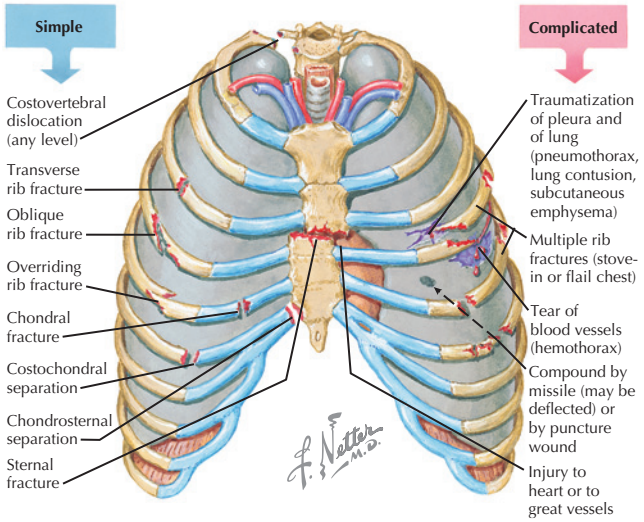


Figure 46-2 Thoracic Cage Injuries.

Physical exam: Localized tenderness, ecchymosis, and edema; crepitus over fracture site; palpable deformity of rib if fracture displaced; shallow, rapid breathing; with anteroposterior and transverse compression of rib cage, pain at site of suspected injury; subcutaneous emphysema with pleural injury.

Differential diagnosis/associated injuries:

- Severe rib contusion, costochondral separation, muscle strain (e.g., forceful contraction of thoracic muscles during tennis serve), other medical causes of chest pain including pneumothorax, pleurisy, herpes zoster.
- The more ribs fractured, the greater the incidence of intrathoracic injuries.
- **If first rib fracture displaced posteriorly, look for vascular injury** (e.g., subclavian artery, aorta).
- **Fracture of lower two ribs may damage kidneys, liver, or spleen;** splenic trauma reported in up to 20% of left lower rib fractures; liver trauma in up to 10% of right lower rib fractures.
- **Flail chest:** Fracture of at least three consecutive ribs, each in two locations, causing free-floating segment of chest wall. High chance of internal injury, especially lung and thoracic aorta. Paradoxical chest wall movement results in impaired ventilation and respiratory failure.

Diagnostics: CXR establishes diagnosis in 90% of cases if acute fracture; can also exclude complications, such as pneumothorax. Oblique views may detect anterior and lateral fractures. Bone scan if stress fracture of rib is suspected. US more sensitive than conventional radiography. CT scan most appropriate imaging modality if suspect posterior displacement of first rib. If cardiac complications possible, follow-up with ECG and echocardiogram. If upper thoracic ribs fractured, angiography may be indicated. If renal injury is suspected, IV pyelogram or other imaging modality may be performed.

Treatment: Pain relief using ice, nonsteroidal anti-inflammatory drugs (NSAIDs), analgesics. Bone stimulators have been approved by the Food and Drug Administration (FDA) for certain fractures and fracture sites, but no published data on its beneficial use for acute rib fractures or rib stress fractures. Intercostal nerve block for relief of severe pain; after aspirating, infiltrate just below the lower border of the rib, in close approximation to the intercostals vessels and nerve. Level of fracture along with two ribs above and below is infiltrated with 3 to 5 mL of lidocaine or bupivacaine. Risk of causing pneumothorax. If multiple fractures, may require open reduction/internal fixation (ORIF). Encourage deep breathing to prevent atelectasis and pneumonia; best to avoid/minimize rib belt or taping. Modification of activities until symptoms resolve, then gradual resumption of training. Changes in technique (if thought to contribute to cause of fracture). Nutritional evaluation and pertinent laboratory tests especially if suspect bone insufficiency.

Prognosis and return to play: Return to play when no pain with palpation, no use of analgesics, full ROM of thoracic cage, and ability to sprint/twist without significant discomfort. Usually minimum of 3 weeks and typically 6 to 8 weeks before return to contact sports; ability to protect fracture site should be considered. Early return inadvisable because of danger of pneumothorax. Close follow-up essential to ensure no development of delayed complications (e.g., excessive callus formation of first rib can cause thoracic outlet syndrome or Horner's syndrome).

Costochondral Sprain and Separation ("Rib-Tip" or "Slipping Rib Syndrome")

Description: Frequently occurs in contact sports such as football, ice hockey, wrestling, lacrosse, and rugby. Weakness or separation of costal cartilage as it attaches to the sternum (sternocostal ligament), or separation of anterior margin of rib from anterior end of the costal cartilage (costochondral ligament), putting pressure on intercostal nerve lying between it and rib above. More frequently involves tenth rib, followed by ninth or eighth.

Mechanism of injury: Forced compression of rib cage, twisting injury, or stretching injury to joint when arm is forcefully pulled to side. Onset sometimes insidious, occurring long after initial trauma, because loose rib can cause further stretching of supporting ligaments.

Presentation: Main symptom is upper abdominal pain or lower chest pain. History of feeling a pop; initial sharp discomfort, with severe pain lasting for several days before slowly decreasing in intensity. Pain patterns can include a dull sensation, intermittent unilateral pain in anterior ends of lower costal cartilages, or severe sharp pains during bending maneuvers with painful click as cartilage and bone override one another. Pain can radiate toward epigastric region or spine.

Physical exam: Localized swelling and tenderness at involved joint; possible deformity because of displacement of cartilage. Reproducible pain and sometimes click by hooking fingers under the costochondral junction in question and pulling rib cage anteriorly ("hooking maneuver").

Differential diagnosis/associated injuries:

- **Costochondritis or costosternal syndrome:** Both traumatic and nontraumatic; self-limiting; multiple sites of tenderness (usually second to sixth costal cartilages) but without swelling.
- **Tietze's syndrome:** Traumatic and nontraumatic; self-limiting; usually only second or third costochondral junction involved with localized swelling.

Diagnostics: CXR if chronic to rule out tumors, Paget's disease, or rheumatoid arthritis. US of costal margin during abdominal muscle contraction to demonstrate abnormal mobility.

Treatment: Ice and NSAIDs; injection of lidocaine or bupivacaine with or without corticosteroid at site of separation; rib block. Physical therapy for correction of possible posterior dysfunction at corresponding costovertebral joint. Surgical resection of affected costochondral junction or repair of the rib and cartilage for intractable pain.

Prognosis and return to play: May take 9 to 12 weeks to resolve (slow healing); subject to reinjury because it seldom completely heals.

Rupture of Pectoralis Major

Description: The pectoralis major is the most important adductor and internal rotator of shoulder and cosmetically forms anterior wall of axilla. Ruptures can be partial (grades I and II) or complete (grade III). Excessive tension on muscle causes tear of muscle belly, musculotendinous junction, or tendinous insertion on humerus lateral to bicipital groove; latter most common. Tears of proximal sternal origin rare. May be associated with anabolic steroid use because muscle hypertrophy is not accompanied by tendon adaptation. Incidence of injury has increased over past several years.

Mechanism of injury: Excessive tension on maximally, eccentrically contracted muscle while UE is externally rotated, extended, or abducted. In athletes, most often seen in weightlifters during bench press. Also reported during waterskiing, wrestling, and other sudden violent deceleration maneuvers with sudden stretching and cocontraction of muscle (blocking with outstretched arm in football, punching in boxing, attempting to grasp something to prevent fall).

Presentation: History of sudden stress or direct blow to shoulder while arm is abducted and extended; sudden onset of extreme pain on medial aspect of UE or in chest wall; tearing, snapping, or popping sensation; significant swelling and ecchymosis; painful limitation of motion; weakness of involved UE. After resolution of swelling and ecchymosis, complaints of asymmetry and persistent weakness.

Physical exam: Swelling and hemorrhage into arm and across anterior chest wall; weakness and pain during resisted internal

rotation, flexion, and adduction of arm on affected side; deformity of chest wall and palpable muscle bulge with resisted adduction; with abduction, defect in anterior axillary fold if tendon is avulsed at its insertion. Shoulder ROM limited by pain.

Differential diagnosis/associated injuries: Pectoralis muscle tendonitis/tendinosis, congenital absence of pectoralis major muscle.

Diagnostics: CXR reveals soft tissue swelling and absent pectoralis major muscle shadow. Shoulder radiographs to rule out bony avulsions/fractures. US shows uneven echogenicity and muscle thinning. Magnetic resonance imaging (MRI) accurately defines extent of injury (grade), location, amount of retraction, and helps in guiding appropriate treatment plan.

Treatment: Extent of tear may be difficult to diagnose because of the ecchymosis, swelling, and extreme pain; serial exams important. Partial tear is treated conservatively with ice, analgesia, sling for comfort, and activity restriction initially. Start with early protected ROM and gentle isometric strengthening. Regain full ROM to prevent further injuries, then start resisted strengthening exercises by 6 to 8 weeks. Activities should be resumed slowly as pain and function allow. Complete tear is surgically repaired in competitive athletes, especially those who depend on chest and shoulder strength. Without surgical repair, weakness can result, especially adduction and flexion. Repair recommended in bodybuilders for improved cosmesis. Many present with delayed diagnosis and thus adhesions, muscle retraction, and atrophy, but late repair compatible with significant strength improvement. After surgery, immobilization for 4 to 6 weeks to protect the repair. Passive pendulum exercises and passive forward flexion with arm adducted to 130 degrees can begin immediately. Between 6 to 12 weeks progress to full passive ROM and add periscapular and isometric strengthening program (avoiding shoulder adduction, internal rotation, and horizontal adduction). By 12 weeks, resistive strengthening exercises begin; by 6 months, light free weights and push-ups.

Prognosis and return to play: Surgical repair of distal pectoralis major tears results in almost full recovery of peak torque and work performed (>97%). Full recovery of those managed nonsurgically is only 56%, but normal activities of daily living are not impacted. Nonsurgical management recommended for tears at the sternoclavicular origin, although delayed repairs for persistent pain have been successful. Return to sports after surgery ranges from 8 months to a year.

Breast Injuries

Description: Contusions, hematomas, runner's/cyclist's nipple, breast pain.

Mechanism of injury and presentation: Contusions caused by direct trauma with resultant bleeding and swelling; common in softball and basketball. Nipple chafing, pain, eczema, and occasional bleeding from friction and abrasion by clothing during prolonged activity or because of evaporation of perspiration over chest (see Chapter 34, Skin Problems in the Athlete). Breast pain is often experienced during athletic activity, especially running, because of strain of Cooper's ligaments (connective tissue that holds and supports breast on chest wall). Query if pain is cyclical, and if so, how long it lasts.

Physical exam: Look for additional signs suggestive of other breast conditions, such as a mass, skin changes, or bloody nipple discharge. In runner's nipple, usually bilateral involvement with erythema, edema, oozing, crusting, and occasionally lichenification. Systematically examine the four breast quadrants in both lying and sitting positions with hands on hips and then above head. Examine axillary, supraclavicular, and infraclavicular lymph nodes.

Differential diagnosis/associated injuries: Pectoralis major muscle strain, costochondritis, rib fractures if significant trauma, fibrocystic breast disease, cyclic mastalgia, contact dermatitis, bacterial or yeast infection of the nipple, Paget's disease, breast cancer. **Fifteen percent of women with newly diagnosed breast cancer had localized breast pain as the presenting symptom.** Individuals with concomitant atopy are predisposed to develop jogger's nipples. For those with breast augmentation and blunt chest trauma, implant rupture can lead to spherical capsular contracture.

Diagnostics: In general, diagnostic studies not required if no breast masses palpated and no nipple discharge. US should be considered if the breast pain is focal and mammography should be considered in women at high risk of breast cancer.

Treatment, prognosis, and return to play:

- See Chapter 34 for information on runner's nipple.
- **For contusions:** Ice, NSAIDs, and proper support; added protective padding. Rarely, hematoma requires aspiration.
- Can lead to posttraumatic scarring and retraction or thrombophlebitis of the superficial veins (Mondor's disease). Follow closely to differentiate from breast carcinoma.
- Premenarchal athletic injuries to the breast bud (Tanner stages I and II; ages 10 to 11 years) can cause appreciable breast asymmetry (as much as one cup size or more).
- Hormonal therapy may help to diminish breast tenderness during phases of menstrual cycle.
- Nicotine may increase breast pain; it increases epinephrine levels, which stimulates cyclic AMP, a regulator of mammory tissue metabolism.
- In pregnancy, specialized breast support very important because breast can enlarge by 800 mL.

Lung Injuries

Pulmonary Contusion

Description: Blood and protein leak into alveoli and interstitial spaces, leading to atelectasis and consolidation (Fig. 46-3).

Mechanism of injury: Blunt trauma to chest.

Presentation: Chest pain, shortness of breath, cough, hemoptysis (see Fig. 46-3).

Physical exam: Hypoxemia (hallmark clinical sign), tachypnea, rales, wheezing, diminished breath sounds. Physical examination may be normal.

Differential diagnosis/associated injuries: Other causes of chest pain and hemoptysis (pulmonary embolism and pneumonia). With history of chest trauma, injury to pulmonary parenchyma most likely. Flail chest highly associated with pneumothorax or hemothorax; also pulmonary contusion and aortic rupture. **Diagnostics:** Initial CXR may not show severity of injury; nonsegmental patchy infiltrates or consolidation evident 4 to 6 hours and occasionally up to 48 hours after injury (see Fig. 46-3). CXR also underestimates degree of contusion. CT scan highly sensitive; pulmonary contusions detected twice as frequently with CT compared with CXR. However, CXR still provides clinically valuable information at minimal cost, and should be considered the primary imaging tool. Pulse oximeter or blood gas analysis helpful.

Treatment: Assisted ventilation; endotracheal intubation if necessary. Watch for onset of pneumonia and acute respiratory distress syndrome (ARDS). Severe thoracic or abdominal trauma represents major risk factor. Mechanical ventilation during first days after trauma seems to reduce risk of pneumonia, but ventilatory support for more than 5 days is associated with increased risk of pneumonia. Avoid volume overload. Use of prophylactic antibiotics and steroids remains controversial. Early research evaluating use of antioxidants, adenosine, NSAIDs, and hemoglobin-based oxygen-carrying compounds has shown promise. Extracorporeal membrane oxygenation (ECMO) for severe pulmonary contusion in respiratory failure when conventional treatments have failed. Surfactant replacement in those who are ventilated with severe hypoxemia has resulted in improved oxygenation and lung compliance.

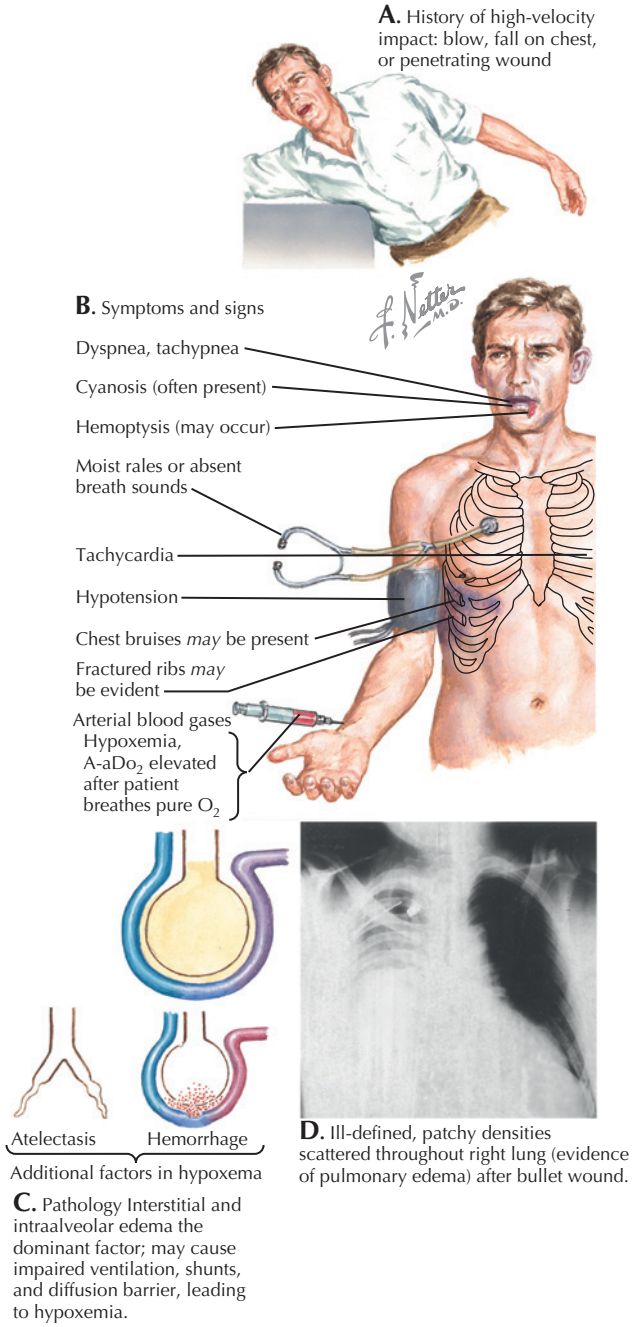


Figure 46-3 Pulmonary Contusion.

Prognosis and return to play: No sport-specific guidelines exist for return to play. With mild pulmonary contusion and no CXR findings, gradual return to progressive activity after symptoms resolve (between 2 and 10 days). Traumatic pseudocysts can develop following a contusion; follow-up imaging is essential to avoid an infection and possible surgical excision. Consider flak jacket for contact sports. Those who recover from even severe pulmonary contusions do not suffer significant late respiratory problems.

Pneumothorax

Description: Refers to air within chest cavity in pleural space (separation of the visceral and parietal pleura), which leads to collapse of lung. Classified as **spontaneous** or **traumatic;** described by approximate percentage of hemithorax occupied by free air (e.g., 10%, 50%).

Mechanism of injury: Blunt trauma most common cause, usually associated with rib fracture(s). Spontaneous pneumothorax occasionally precipitated by strenuous physical activities, especially in tall, thin, young males who smoke. Risk is increased by defects in periphery of lung; these bullae usually located in apex.

Presentation: Gradual or sudden pleuritic chest pain and dyspnea (depending on size and rate of collapse of lung); pain referred to shoulder tip.

Physical exam: Shallow, rapid respirations; cyanosis; tachycardia; hyper-resonance to percussion and decreased or absent breath sounds over affected lung; tracheal shift to contralateral side; possible subcutaneous emphysema.

Differential diagnosis/associated injuries: Traumatic rupture of left hemidiaphragm with herniated stomach bubble mistaken for loculated pneumothorax. Skin folds act as "Mach bands" and presumed to be visceral pleural lines. **Hemothorax:** Blood accumulates in pleural space as result of bleeding of intercostal or mammary blood vessels and lung parenchyma injury; massive bleeding with aortic or myocardial rupture and injuries to the hilar structures. Usually associated with pneumothorax. Symptoms include dullness to percussion, decreased breath sounds, hypotension. **Pneumomediastinum:** Excessive intra-alveolar pressures (from exacerbation of asthma, coughing, vomiting, childbirth, seizures, Valsalva maneuver) lead to rupture of perivascular alveoli; air escapes into surrounding connective tissue, with dissection into mediastinum. Symptoms include chest pain, persistent cough, sore throat, and substernal chest pain, radiating to the back, neck or shoulders. Subcutaneous emphysema is most consistent physical finding.

Diagnostics: Upright PA CXR shows absence of lung markings in periphery and increased density of collapsed part of lung. The white visceral pleural line is evident. A lateral width of 1 cm corresponds to a 10% pneumothorax. Inspiration and expiration films may make small pneumothorax more visible; a small pneumothorax on the nondependent side can be more easily detected in lateral decubitus view. Mediastinal shift seen with large pneumothorax. Thoracic US more sensitive than supine CXR and as sensitive as CT scan in detecting traumatic pneumothoraces.

Treatment: If minimal (<15% to 30%), stable, and asymptomatic, observation with serial exams and CXR. Avoid unnecessary physical exertion. If large enough to cause shortness of breath and discomfort, transport to hospital for possible insertion of chest tube. In an **open pneumothorax** with a chest wall defect, ambient air enters the injured hemithorax during inspiration, and mediastinum shifts to the uninjured side. In expiration, mediastinum swings back to the injured side and expiratory air from the normal lung enters the collapsed lung. Place foil, cloth, or other item over wound, securing it only on three sides to avoid development of tension pneumothorax.

Prognosis and return to play: No vigorous activities for 2 to 3 weeks after chest tube removed; then slow, monitored return to activity. Educate about risk of further episodes (reported in up to 50% of cases of primary spontaneous pneumothorax). No published data to suggest an increased risk for recurrence of traumatic pneumothorax. Review proper breathing techniques during sport.

Tension Pneumothorax

Description: Progressive enlargement of pneumothorax because of communication between airways or exterior and interpleural space (Fig. 46-4). **Flap valve effect** is created; with inspiration, air is drawn into pleural cavity; with expiration, air stays trapped. Positive intrapleural pressure develops, shifting the mediastinum, and further impairs ventilation of compressed noninjured lung. **Absolute medical emergency;** progressive hypoxia and hypotension lead to death.

Mechanism of injury: Similar to pneumothorax.

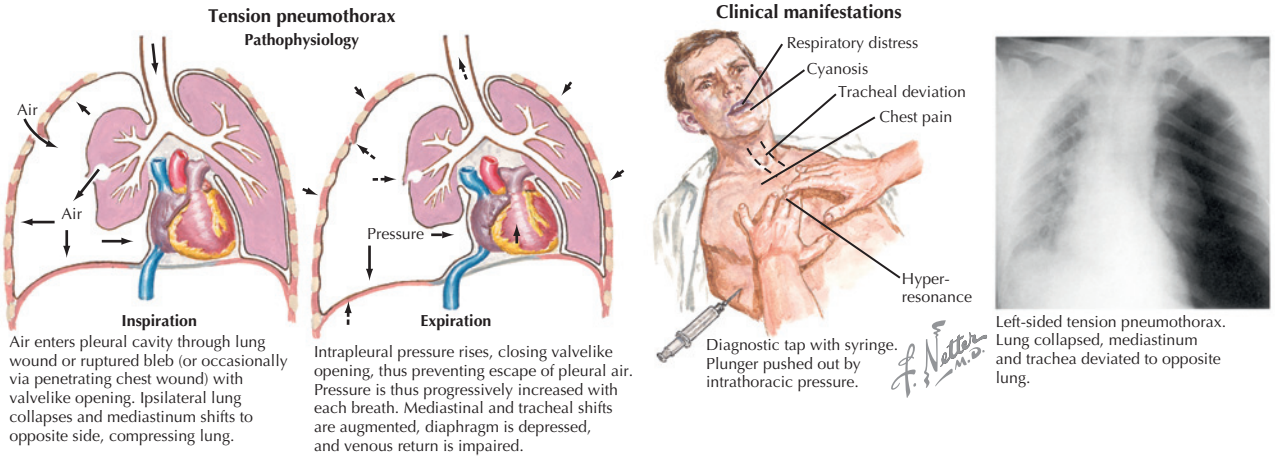


Figure 46-4 Tension Pneumothorax.

Presentation: Rapidly increasing shortness of breath, asymmetry of respiration (see Fig. 46-4).

Physical exam: Distended neck veins, cyanosis, hypotension and tachycardia, dyspnea and tachypnea, shift of the trachea away from the injured side, absent breath sounds on involved side; hyper-resonance on percussion of involved side.

Diagnostics: CXR (only if immediately available) shows distinct shift of mediastinum to contralateral side and flattening of ipsilateral hemidiaphragm.

Treatment: Usually emergently treated prior to a confirmatory chest radiograph with needle decompression; large-bore needle (14 to 16 gauge) inserted into second intercostal space in the midclavicular line on the affected side, just over superior aspect of rib to avoid intercostal vessels. Transport to hospital and chest tube placement for definitive treatment. If breathing spontaneously, hemodynamically stable, and no evidence of respiratory compromise, there may be time to obtain a portable CXR, then immediately place a chest tube once diagnosis is confirmed.

Prognosis and return to play: No specific return to play guidelines specific to tension pneumothorax. Treated as discussed previously, with gradual increase in progressive activities.

Cardiac and Great Vessel Injuries

Myocardial Contusion

Description: Most common cardiac complication of blunt chest trauma; can lead to impaired circulation, arrhythmia, or bleed into pericardium, resulting in cardiac tamponade at time of injury or as late complication several weeks after injury. Generally, myocardial contusion refers to structural damage of the heart as described in this section; commotio cordis refers to a sudden disturbance in the heart rhythm via mechanical impact in the absence of structural damage (see Chapter 29, The Athlete's Heart and Sudden Cardiac Death).

Mechanism of Injury: Blunt trauma causes "bruising" of cardiac muscle; most commonly reported from a baseball or hockey puck.

Presentation: Crushing and sudden deceleration injuries. Impact to anterior chest wall and sternum. Difficult to diagnose because signs vary with degree of myocardial damage and are typically transient. Athlete may experience minor chest pain or sudden cardiac arrest. Chest pain is nonpleuritic; relieved by oxygen but not nitroglycerin.

Physical exam: Tachycardia, arrhythmias, signs of decreased cardiac output. Cardiac exam usually normal but may show friction rub or murmur.

Differential diagnosis/associated injuries: Differential diagnosis includes acute coronary occlusion. May be obvious external chest wall injury such as sternal fractures.

Diagnostics:

- **ECG:** Findings are nonspecific, but 70% to 85% show abnormalities of ST segment and T wave changes, sinus tachycardia, and intraventricular conduction disturbances. Seen on initial evaluation and subsequent 3 days after injury; usually transient.
- **Serum creatine kinase (CK-MB) isoenzymes:** Not specific; miss 40% of contusions when used alone. Abnormal ECG and CK-MB correlate directly with complications requiring treatment. Conversely, normal ECG and CK-MB correlate with lack of complications. Right ventricle usually injured because it composes most of frontal surface of heart, yet it constitutes little of total myocardium and releases small amount of CK-MB enzyme.
- **Holter monitor:** If arrhythmias present, most are premature ventricular contractions; atrial fibrillation and other supraventricular arrhythmias also noted.
- **Radionuclide angiogram:** Depressed ejection fraction and segmental ventricular wall motion abnormalities; also nonspecific and not predictive of cardiac complications.
- **Echocardiography:** Transthoracic echocardiogram (TTE) can narrow differential diagnosis of blunt chest trauma in the setting of rising cardiac markers. If TTE limited secondary to other injuries sustained in chest trauma, use transesophageal echocardiography. Echocardiography may be more effective tool to follow suspected contusion and manage myocardial decompensation.
- **Cardiac catheterization:** Differentiate between myocardial contusion and coronary artery occlusion.
- **Serum cardiac troponin I:** Low sensitivity and predictive value in diagnosing myocardial contusion.

Treatment: For mild contusion, admit for observation and watch for dysrhythmias (more likely to occur in first 24 hours following injury). For more severe contusion associated with other injuries, may require invasive monitoring and inotropic medications.

Prognosis and return to play: Gradual return to activity when stable and showing signs of fully healing from a diagnostic and clinical standpoint. Manufacturers are developing softer baseballs to decrease incidence of morbidity and mortality. Use of chest protector, especially during batting, is advocated.

Cardiac Tamponade

Description: Accumulation of blood or edematous exudate into pericardial sac. Volume and rate of accumulation of fluid determine symptoms. Tension created within pericardial sac limits

venous inflow and diastolic filling, and cardiac output is diminished (Fig. 46-5). Shock and death can rapidly evolve without early recognition and treatment.

Mechanism of injury: Blunt trauma to chest, most commonly from high-energy collisions.

Presentation: Symptoms can be variable, but acutely will present with dyspnea, tachycardia, and tachypnea. Cold and clammy extremities caused by hypoperfusion.

Physical exam: "Beck's triad"—hypotension, jugular venous distention and distant heart sounds, pulsus paradoxus (fall in blood pressure of more than 10 mm Hg on inspiration)—tachycardia, weak pulse.

Diagnostics:

- **X-ray:** Cardiomegaly, water bottle–shaped heart, pericardial calcifications, or evidence of chest wall trauma.
- **ECG:** Shows low voltage most commonly, also sinus tachycardia, electrical alternans, PR segment depression.
- **Echocardiography:** Can visualize pericardial effusion, but cardiac tamponade is clinical diagnosis.

Treatment: IV fluids and **urgent transport to hospital;** pericardiocentesis guided by echocardiography, and emergent thoracotomy (see Fig. 46-5).

Prognosis and return to play: No evidence-based guidelines for return to play; decisions should involve cardiologist and/or cardiothoracic surgeon.

Coronary Artery Dissection and Occlusion

Description and mechanism of injury: Uncommon result of blunt trauma (e.g., high-speed vehicular collisions) resulting in rapid deceleration of body. Creates enormous shearing forces that may result in intimal dissection or disruption of coronary artery near its origin; intraluminal thrombus can form adjacent to injured arterial wall. Can be asymptomatic; may cause angina, myocardial infarction, or death.

Presentation: Dyspnea, diaphoresis, severe chest pain with or without radiation of pain, nausea.

Physical exam: Sinus tachycardia, hypotensive or hypertensive, may hear murmur particularly if any injury affects valvular function or wall motion.

Diagnostics:

- ECG: may show evidence of myocardial ischemia
- Echocardiography: to evaluate wall motion abnormality
- Coronary angiogram: to evaluate coronary vasculature

Treatment: Conservative, because lesion may heal with medical management: IV morphine, nitroglycerin; anticoagulation with heparin and acetylsalicylic acid; beta-blocker, statin, or later ACE inhibitor. Watch for postinfarction complications, including complete heart block, ventricular arrhythmias, left ventricular failure, ventricular aneurysm, and pulmonary emboli. With ongoing ischemia, coronary angiography can confirm diagnosis and assess extent of injury with possible percutaneous coronary intervention (PCI).

Prognosis and return to play: Dependent on extent of injury and treatment used; decisions should be made in conjunction with cardiologist.

Traumatic Aortic Rupture

Description: Approximately 80% to 90% fatality rate at scene of accident. Disruption extends through full thickness of aortic wall, with rapid exsanguination into mediastinum and pleural spaces. In only 20% is rupture sufficiently contained by adventitia to allow long enough survival to reach medical attention. Potentially higher risk in athletes with **Marfan syndrome** (cystic medial necrosis causes aortic dilatation); these athletes are restricted from contact and high-exertion sports.

Mechanism of injury: High-speed, deceleration-type injuries seen in motor sports, bicycling, skiing, and snowboarding. Tremendous torques that result from sudden deceleration affect junction of fixed and mobile parts of great vessels, as between aortic arch and fixed descending aorta.

Presentation: Acute onset anterior chest or interscapular pain that can migrate, dyspnea, hoarseness, neurologic deficits seen in some cases; can be difficult to detect initially.

Physical exam: Hypertension in UE with pulse pressure widening. With **ascending aortic injury,** aortic diastolic murmur radiating to back because of incompetence of aortic valve. Acute coarctation syndrome in up to 25% of **descending aortic injuries:** caused by partial obstruction of aortic lumen, UE hypertension, diminution of femoral pulses and leg blood pressure, and systolic murmur.

Diagnostics:

- **CXR:** Abnormalities present at time of admission in 75% to 90%: widening of superior mediastinum and obscuring of aortic knob shadow are most consistent findings. Others: deviation of trachea to right, depression of left main stem bronchus, left apical extrapleural density, and left pleural effusion.
- **ECG:** May show left ventricular hypertrophy or myocardial ischemia or infarction.
- **TEE:** May be both sensitive and specific enough to plan management without aortogram.
- **CT scan with contrast enhancement:** Often used in emergency department settings.

Treatment: Medical therapy to avoid sudden rupture, including antihypertensive agents and beta-blockers. Emergent operative repair results in 75% to 90% survival.

Prognosis and return to play: Mortality and morbidity rates for repair of this condition are among the highest in cardiovascular surgery; 80% to 85% of patients die before arrival to the hospi-

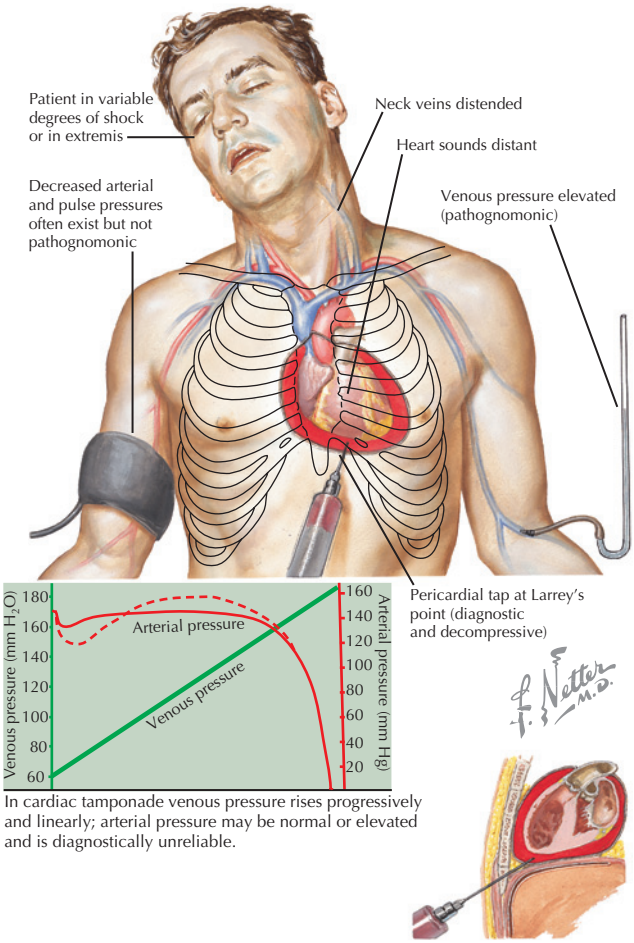


Figure 46-5 Cardiac Tamponade.

tal. Return to play decisions should be made with consultation of cardiologist and/or cardiothoracic surgeon.

Thoracic Outlet Syndrome

Description: Spectrum of signs and symptoms resulting from compression of neurovascular bundle (brachial plexus and subclavian and axillary arteries and veins) in interval between intervertebral foramina and axilla (Fig. 46-6). Clinical presentations differ according to different degrees of compression. Greater incidence in women (4:1 male-female ratio); perhaps secondary to lower position of scapula and changing shoulder posture with larger breasts.

Mechanism of injury: Related to areas of compression:

- **Supraclavicular region:** Interscalene triangle bordered by anterior and middle scalene muscles, which attach to first rib. Contributing factors include hypertrophy of scalene muscles; long transverse process of C7, cervical ribs, or other rib anomalies; fibrous bands changes in alignment and angulation of first rib, as may occur with age and postural changes.
- **Subclavicular or costoclavicular region:** Between "mobile" clavicle and "fixed" first rib. Changes in shape and mobility of clavicle, such as callus from fracture or alteration in shoulder motion, can narrow interval. Subclavius muscle lies behind clavicle just anterior to subclavian vein.
- **Infraclavicular region:** At coracoid process of scapula. Compression by pectoralis minor, which inserts at coracoid, during full abduction. Subcoracoid area has thickened costocoracoid membrane. Another contributing factor is any lesion involving the pleura, such as neoplasm.

Presentation:

- **Neural compression symptoms:** Pain from root of neck to shoulder and down arm diffusely. If lower trunk of brachial plexus compressed (most common), paresthesias involving medial aspect of elbow, forearm, and hand, especially little finger and ring finger. Weakness and occasionally atrophy of affected hand. Sometimes just vague ache and heaviness in shoulder, upper arm, and upper anterior and posterior chest,

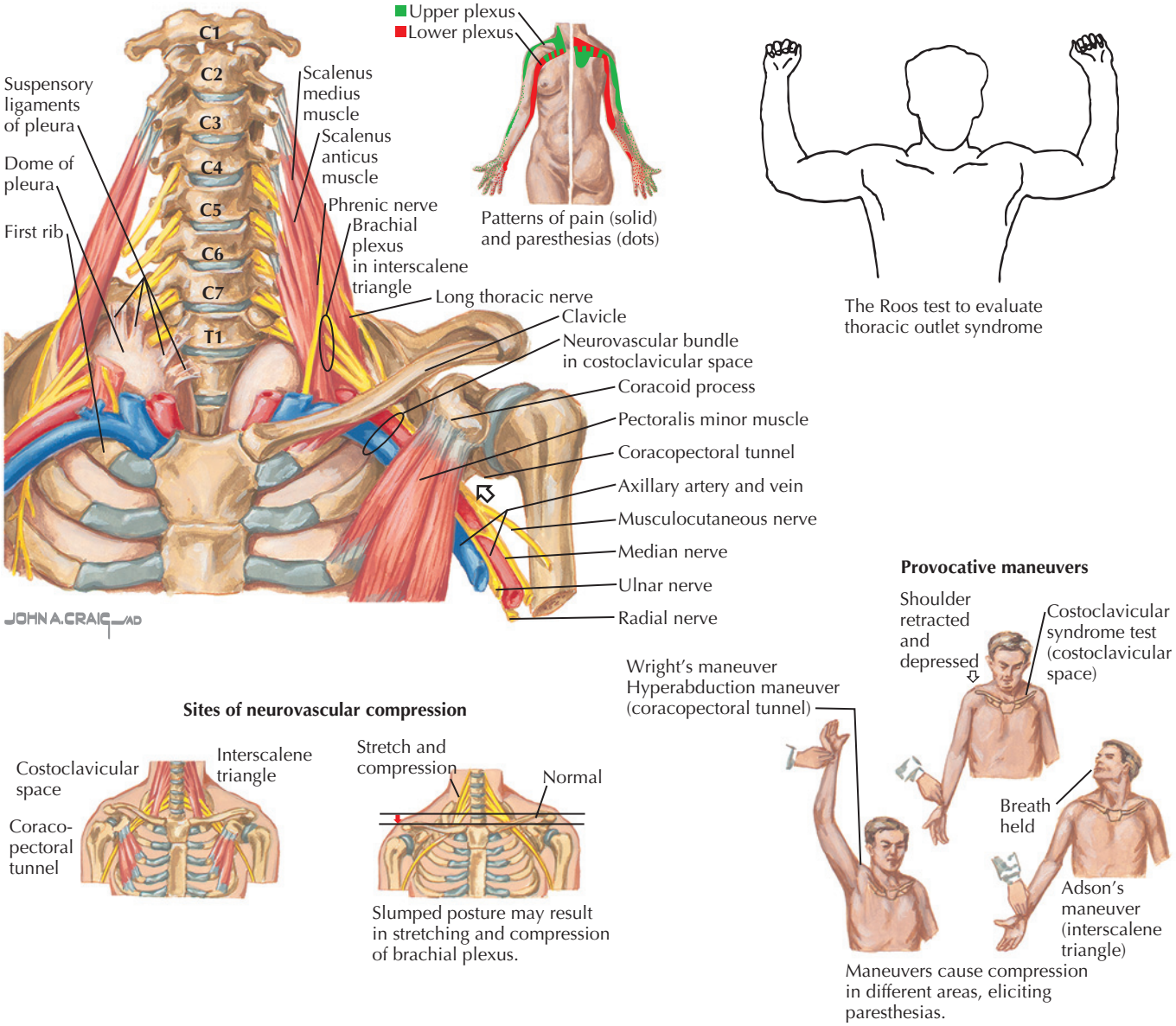


Figure 46-6 Thoracic Outlet Syndrome.

including trapezius and suboccipital region. People who sleep with arms above head may awaken with symptoms at night.
- **Arterial compression symptoms:** Hand feels cold, arm becomes numb and fatigued with rapid overhead movement.
- **Venous compression symptoms:** Swelling and discoloration of arm after exercise, prominent superficial venous pattern over ipsilateral shoulder and chest.

Physical exam: Careful exam of neck, cervical spine, shoulder, elbow, and hand; check supraclavicular fossa for masses or bruits; careful neurologic exam.
- **Adson's maneuver:** Arm abducted and externally rotated, while head extended and turned to side of lesion. Radial pulse monitored while deep breath is taken and held. Positive test equals diminution or total loss of pulse. Test is positive in up to 30% of asymptomatic population.
- **Wright's maneuver:** Similar to Adson's except arm is hyperabducted, with hand brought over head with elbow and arm in coronal plane.
- **Military brace position or costoclavicular syndrome test:** Shoulders are retracted and pulled down to narrow interval between clavicle and first rib and reproduce symptoms, including absence of radial pulse. Effective test in those with symptoms when wearing backpack.
- **Overhead exercise test (Roos test):** Arms are abducted to 90 degrees, shoulders externally rotated, and elbows flexed to 90 degrees (see Fig. 46-6). Hands opened and closed slowly for 3 minutes to reproduce symptoms of fatigue, cramping, numbness and tingling, or coolness or paleness of extremity.

Differential diagnosis/associated injuries: Shoulder instability and "dead arm syndrome"; cervical spine pathology; peripheral neuropathies; Raynaud's phenomenon (can be present with both thoracic outlet syndrome and collagen vascular diseases); complex regional pain syndrome; Pancoast's tumor or other space-occupying lesion in thoracic outlet; **myofascial syndrome** (periscapular region, base of neck, and chest wall are common areas of pain and fatigue; trigger points commonly found in supraspinous fossa near rhomboids, levator scapula, and infraspinatus can cause pain to radiate down arm).

Diagnostics: Cervical spine x-rays; myelogram or MRI if necessary; CXR to evaluate lung apex; electrodiagnostic studies (difficult to accurately determine nerve conduction velocity at thoracic outlet; perform with arm in provocative position; helpful in ruling out peripheral nerve entrapment); venography or arteriography; plethysmography; spiral CT for anatomic correlation of compressive elements.

Treatment: Conservative management results in 50% to 90% recovery. Strengthen shoulder girdle suspensory muscles (trapezius, serratus anterior, deltoid, erector spinae). Stretch scalenes, lateral neck flexors, and pectoral muscles. Other measures include weight reduction, posture training (correct "drooped" shoulder), proper brassiere support. Avoid hyperabduction of shoulder and carrying heavy packages in affected hand. Consideration of operative treatment only if diagnosis is firm and conservative treatment has failed (3 to 4 months), with symptoms of intractable pain or major neurologic or vascular complications. First rib resection is most dependable, but surgical procedure varies depending on anatomic basis for symptoms.

Prognosis and return to play: Conservative treatment generally results in recovery from injury; return to play is generally guided by the patient's clinical symptoms.

Vascular Injuries of Subclavian and Axillary Veins

- **Description: "Effort thrombosis"** (Paget-Schroetter syndrome), or primary thrombosis, describes traumatic thrombosis of the subclavian or axillary vein.
- **Mechanism of injury:** May occur after single traumatic event around shoulder or clavicle (clavicular fracture, axillary hematoma, injury to axillary or subclavian vein). **More commonly associated with repetitive overhand motions causing trauma to the vessel** (e.g., hyperabduction, external rotation). This primary thrombosis is related to inherent anatomic structure of thoracic outlet and axillary region, with compression at various points causing damage to the vein walls.
- **Presentation:** Pain and diffuse swelling in affected arm; numbness, heaviness, and easy fatigability; distention of superficial arm veins with cyanosis (bluish discoloration) of skin; onset of symptoms commonly within 24 to 72 hours of activity.
- **Physical exam:** Increase in girth of UE; may be weakness of UE, especially if venous occlusion is long-standing. Symptoms can often be reproduced with exercise test of UE or by putting arms into extreme hyperabduction.
- **Differential diagnosis/associated injuries:** Rule out secondary causes of thrombosis (sarcoidosis, infection, drug use, hypercoagulable states, metastatic tumor) and poor circulation. Arterial occlusion, including **aneurysms of subclavian or axillary arteries,** also reported in athletes. Classic symptom also early fatigue during act of throwing. Other symptoms include absent pulses, cyanosis, decreased skin temperature, and finger ischemia secondary to digital embolization. If UE thrombosis confirmed, watch for signs of pulmonary embolus (incidence approximately 12%).
- **Diagnostics:** Venogram, compression ultrasonography or duplex Doppler studies (most accurate noninvasive tests), CXR, cervical spine (AP view). Given frequent bilateral presentation, consider evaluation of both UE.
- **Treatment:** Simultaneous anticoagulation with IV or low–molecular weight heparin and subsequent warfarin therapy for 1 to 3 months. Thrombolysis with fibrinolytic agents such as streptokinase or urokinase. Thrombectomy and surgical correction of involved thoracic outlet and axillary structures if documented external compression. High incidence of late morbidity, such as swelling, pain, fatigability, and numbness, especially with conservative therapy or anticoagulation alone. Can become asymptomatic if compensatory collateral veins develop. Promising short-term results with thrombolysis alone or in combination with surgery.
- **Prognosis and return to play:** No participation in contact sports while on oral anticoagulants.

Abdominal Injuries

Rectus Sheath Hematoma

Description: Major muscle groups of abdominal wall are rectus abdominus muscles, external and internal obliques, and transverses.

Mechanism of injury: Direct blow to abdominal wall, causing hemorrhage into muscle. May damage either epigastric artery or intramuscular vessels, causing hematoma within sheath, which usually self-tamponades. With violent stretching movements, inferior epigastric artery can rupture and hemorrhage without associated indirect injury to muscle tissue.

Presentation: Sudden abdominal pain especially with trunk flexion or rotation, local tenderness, rapid swelling, greatest comfort in supported flexed position; may have nausea and vomiting. Rectus sheath hematoma can mimic an acute abdomen.

Physical exam: Abdomen may be somewhat rigid with guarding, with increased tenderness over rectus. Fixed (within the rectus sheath) palpable mass most often below umbilicus in sitting or lying position. Other signs: bluish discoloration around periumbilical region 72 hours after injury (Cullen's sign) and pain with resisted trunk or hip flexion. Hyperextension of spine causes pain in anterior abdominal wall.

Diagnostics: Laboratory studies: CBC, INR, PT, PTT. US useful diagnostic tool in hands of experienced ultrasonographer.

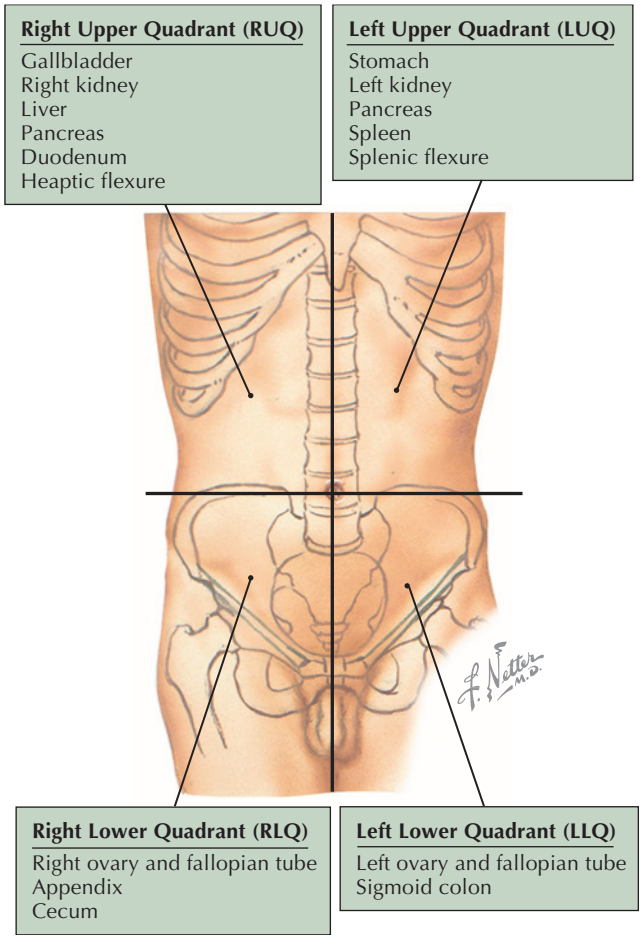
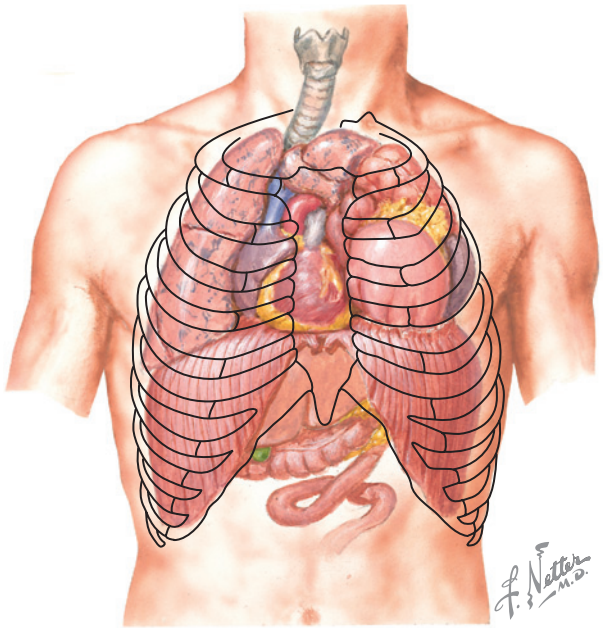


Figure 46-7 Quadrants of Abdomen.



May result from blunt impact or compression or from penetrating wound. Stomach and other abdominal viscera herniated into left thorax; left lung collapsed, right lung compressed; mediastinum shifted and trachea deviated to right.

Figure 46-8 Rupture of Diaphragm.

MRI or CT scan may be used to assist in differentiation between intra-abdominal injury and hematoma (Fig. 46-7).

Treatment: Ice, activity modification, and NSAIDs; local heat after 48 to 72 hours. Avoid activities that require rotation, stretching, or flexion of trunk or lower extremities. Rehabilitation concentrates on restoring flexibility, strength, and endurance of all abdominal muscles. If hemorrhage extensive and superficial epigastric artery is torn, operative evacuation of hematoma and ligation of artery may be required.

Rupture of Diaphragm

Description: Herniation of abdominal contents into chest (Fig. 46-8). Four times more common on left side with blunt trauma because of some protection from liver on right. Easily overlooked because of delayed onset of symptoms.

Mechanism of injury: Blunt chest or abdominal trauma.

Diagnosis: Chest pain, shortness of breath, intestinal obstruction.

Physical exam: Decreased breath sounds in affected chest, excessive percussion of tympany in chest. Bowel sounds in chest, scaphoid abdomen.

Differential diagnosis/associated injuries: Hemothorax, pneumothorax, pulmonary contusion, blunt thoracic aortic tear, elevated hemidiaphragm from other reasons. Splenic rupture in 25% of patients with blunt diaphragmatic rupture, liver lacerations in 25%, pelvic fracture in 40%, rib fracture in 52%, and thoracic aortic tears in 5%.

Diagnostics: CXR and abdominal films show dilated stomach in lower chest. Presence of nasogastric tube terminating in air space is confirmatory. Abdominal CT with reformatted images: for left-sided hernias, 78% sensitivity and 100% specificity; for right-sided hernias, 50% sensitivity and 100% specificity. MRI with clinical suspicion and indeterminate chest radiography and CT findings.

Treatment: Immediate surgical repair. Left-sided injuries explored transabdominally because of high incidence of associated intra-abdominal injuries. Right-sided injuries explored transthoracically because of location of liver. Watch for complications from pneumonia and abscess.

Prognosis and return to play: Complications include missed diaphragmatic injuries. Delayed diagnosis may result in intestinal herniation, ischemia, and necrosis.

Splenic Rupture

Description: Spleen is most commonly injured organ in sport and **most frequent cause of death related to abdominal injury in sport.** Although rib cage offers some protection, rib fractures can leave spleen more vulnerable to injury. The spleen's capacity for encapsulating bleeding delays overt signs and symptoms of rupture; may be days before clinical deterioration. The spleen can enlarge and weaken during some illnesses (infectious mononucleosis, sarcoidosis), making it more susceptible to rupture.

Mechanism of injury: Direct trauma to left lower chest from fall, sporting injury, or motor vehicle accident.

Presentation: Initial sharp pain in left upper abdomen, then continuation of dull, left-sided flank pain; abdominal distention; referred pain to either right or, more commonly, left shoulder **(Kehr's sign)** from free intraperitoneal blood irritating diaphragm. Neck pain may be referred from phrenic nerve pressure **(Seagasser's sign).**

Physical exam: Generalized abdominal tenderness; may have rebound tenderness and rigid abdomen. May have tenderness over ribs 10, 11, or 12. Tachycardia, hypotension, diaphoresis, and

rapid respirations suggest internal bleeding. Fixed dullness in left flank **(Ballance's sign).**

Differential diagnosis/associated injuries: Left-sided 11th and 12th rib fractures, abdominal contusions.

Diagnostics: Imaging studies: Flat-plate abdominal x-rays may show fading splenic outline and growing splenic shadow. CT scan with contrast (greater sensitivity and specificity and greater anatomic detail about spleen and surrounding structures than radionuclide scan) is currently diagnostic imaging standard of care; US; arteriogram; peritoneal lavage (useful but can miss subscapular tear).

Laboratory studies:

- Decreased hemoglobin and hematocrit levels.
- Markedly elevated white blood count if subcapsular hematoma has developed.
- Diagnostic peritoneal lavage is positive unless bleeding is encapsulated (10 mL of free blood, > 100,000 red blood cells/mm^3, or > 500 white blood cells/mm^3).

Treatment:

- If splenic injury suspected, immediate transport to hospital. If hypotensive, give bolus of IV fluids. Keep flat or in modified Trendelenburg position to direct blood flow to heart.
- Various grading systems, based on anatomic location of splenic injury on CT scan, help guide treatment and predict outcome. If hemodynamically stable and splenic injury is minimal or subcapsular, nonoperative management with observation in critical care setting is preferred treatment; splenic preservation is preferred over splenectomy especially in pediatric population.
- Exploratory laparoscopy/laparotomy indicated if continuing hemodynamic instability or if require more than 4 units of blood during a 48-hour period. First choice: repair of capsular lacerations (splenorrhaphy); splenectomy only for extensive injury and uncontrolled hemorrhage. After splenectomy, vaccinate for encapsulated organisms (*Streptococcus pneumoniae*, *Haemophilus influenzae*, and *Neisseria meningitides*).
- Beware of **"delayed rupture"** of spleen; occurs more than 7 days after initial negative CT scan. Need high index of suspicion and liberal utilization of other imaging techniques.

Prognosis and return to play: After 3 months in nonsurgical patients (may be longer depending on results of follow-up studies; interval studies useful in predicting return to play); after 3 to 6 months in postsplenectomy patients. Postsplenectomy patients often return prior to those treated conservatively. Postsplenectomy patients may return to full activity when surgical scars are healed and when able to tolerate activity.

Liver Laceration

Description: Relatively rare in contact sports; usually results from high-speed accidents during motor racing and skiing. Capsular hematoma most common liver injury in athletes.

Mechanism of injury: Blows to right upper quadrant (RUQ).

Diagnosis: Pain in RUQ, right shoulder, or neck pain.

Physical exam: Pain and tenderness to palpation over RUQ; tachycardia and hypotension; may be associated with right lower rib fractures.

Differential diagnosis/associated injuries: Pancreatic injury; obtain serum amylase levels.

Diagnostics: US, CT scan with contrast enhancement, arteriogram, liver enzymes (AST or ALT 130 IU/L indicates liver injury), peritoneal lavage.

Treatment: Grading systems based on anatomic location of lesions on CT scan help determine treatment and prognosis; 50% to 80% of liver injuries stop bleeding spontaneously. Rest, observation, and IV fluids for hemodynamically stable patients with no signs of peritoneal irritation and no other intra-abdominal injuries that may require surgical repair. Laparotomy may be necessary to control bleeding.

Prognosis and return to play: Length of time varies; interval radiographic studies help predict. Return to play guidelines not established; athlete should show anatomic and functional healing prior to participation.

Rupture of Stomach and Intestines

Description: Injuries are rare.

Mechanism of injury: Kicks or blows to abdomen; falls off horse or against equipment, as in gymnastics; pile-ons and spearing in football; handlebar accidents in cycling; diving decompression accidents.

Presentation: Persistent abdominal pain with signs of chemical or bacterial peritonitis, including fever, nausea, and vomiting; referred shoulder pain from irritation of diaphragm; may have blood in stool if intramucosal hemorrhage present.

Physical exam: Localized pain, guarding, rebound tenderness, absence of normal bowel sounds, rigid abdomen; clammy, sweaty skin; hypotension and tachycardia; absence of normal respiratory motion of abdomen; gross or occult blood on digital rectal exam.

Associated injuries: Intramural hematoma of duodenum may manifest as gastric outlet or high small bowel obstruction.

Diagnostics: Plain x-ray with upright and decubitus abdominal views shows free air under diaphragm or along abdominal wall; keep athlete in proper positions for 3 minutes before views are taken. Peritoneal lavage: not helpful for duodenal or large intestine injuries because of retroperitoneal position. Nasogastric tube placement to check for blood if damage to stomach suspected. Meglumine diatrizoate (Gastrografin) swallow. CT scan.

Treatment: Urgent transport to hospital if increasing pain, signs of peritonitis, or circulatory collapse develop; infuse IV fluid until transport arrives. Careful serial examinations, nasogastric tube, and IV fluids. Abdominal exploration and repair.

Prognosis and return to play: Length of time may vary. Return to play guidelines not established; decisions should be made with general surgeon.

Pancreatic Injury

Description: Injuries rare. Pancreas is relatively immobile and in the protected retroperitoneum; injuries to pancreas most often occur with direct contact. Reported injuries include lacerations and contusions to body and duct of the pancreas.

Mechanism of injury: Similar to injuries to stomach and intestine.

Presentation: Abdominal pain; can diminish within first 2 hours after injury then subsequently increase in the following 6 to 8 hours.

Physical exam: Abdominal or epigastric pain, abdominal wall ecchymosis; rebound tenderness is rare.

Diagnostics: Often, initial amylase immediately post trauma is normal. Amylase neither sensitive nor specific for pancreatic injuries, but serial monitoring of amylase found to be more specific. Lipase more sensitive. Hgb and Hct generally normal because blood loss is usually minimal.

- **Peritoneal lavage:** Not helpful because of retroperitoneal position of pancreas.
- **Contrast enhanced multislice CT:** Efficient screening modality for pancreatic trauma.
- **Focused sonography for abdominal trauma (FAST):** Often used in trauma centers as screening in blunt abdominal trauma.
- **Endoscopic retrograde cholangiopancreatography (ERCP):** Evaluates pancreatic duct; highly sensitive; risk of pancreatitis, hemorrhage, and GI tract perforations.
- **Magnetic resonance cholangiopancreatography (MRCP):** Noninvasive and accurate method of imaging pancreatic duct.

Treatment: Principally supportive, IV fluid hydration, management of metabolic complications, fasting to avoid pancreatic stimulation, parental or enteral jejunal feedings.

Prognosis and return to play: Higher morbidity and mortality when pancreatic injuries are not recognized in first 24 hours. No return to play guidelines; progressive return to play following anatomic and functional healing.

Hernias

Description: Three most common hernias in adults are indirect inguinal (50% to 70%), direct inguinal (men older than 40 years), and femoral (women). Hernias involving anterior abdominal wall include incisional, periumbilical, and linea alba defects (spigelian hernias). Potential for **incarceration** (irreducible hernia) and **strangulation** (twisting of hernia) causing bowel obstruction and toxicity.

Mechanism of injury: Repetitive heavy lifting activities cause increased intra-abdominal pressure, which can contribute to development of hernias, especially with predisposing weakness of abdominal muscle and fascia. Hernias also reported secondary to trauma, such as impact from bicycle handlebar on abdominal wall.

Presentation: Aching sensation and occasionally tender swelling in area of hernia; may be scrotal swelling with indirect hernia, as sac extends into inguinal canal.

Physical exam: Indirect and direct inguinal hernias are palpated by invaginating scrotum with finger to palpate external inguinal rings and inguinal canals. Athlete is asked to do Valsalva maneuver. Elliptic mass descending along spermatic cord and bulging against tip of finger is indirect hernia (Fig. 46-9). Globular mass close to pubis that bulges against bottom of finger is direct hernia. **Femoral hernias** occur below inguinal ligament, two fingerbreadths medial to femoral artery (see Fig. 46-9). Hernias more prominent when athlete stands or increases intra-abdominal pressure.

Diagnostics:

- **Herniography:** Intraperitoneal injection of contrast material to diagnose occult hernia sacs; 84% incidence of inguinal hernia by herniography in soccer players with groin pain; only 8% had hernias detectable by physical examination.
- Bone scan: Increased uptake in musculature in initial phase.
- MRI used to examine soft tissues in groin region.
- CT scan: Contrastographic medium combined with CT scan has been used.
- US: Helpful in hands of experienced musculoskeletal ultrasonographer.

Differential diagnosis/associated injuries:

- **Iliopectineal/iliopsoas bursitis:** Groin pain reproduced by passive hip flexion caused by inflammation of bursa between pectineus and psoas muscle. Pain can be localized over area of lesser trochanter. Position of hip flexion and external rotation is most comfortable.
- **Osteitis pubis inflammation of the pubic symphysis and surrounding muscle insertions thought to be secondary to repetitive microtrauma and/or shearing forces.**
- **Posterior inguinal wall weakness (sports hernia, groin insufficiency)** presents as gradually worsening, poorly localized groin pain that is aggravated with activity. Herniography shows bulging or areas of weakness of wall. Dynamic US can demonstrate weakening of pelvic floor musculature with Valsalva consistent with sportsman's hernia. Surgical exploration reveals tears in floor of inguinal ring (transversalis fascia). Il-

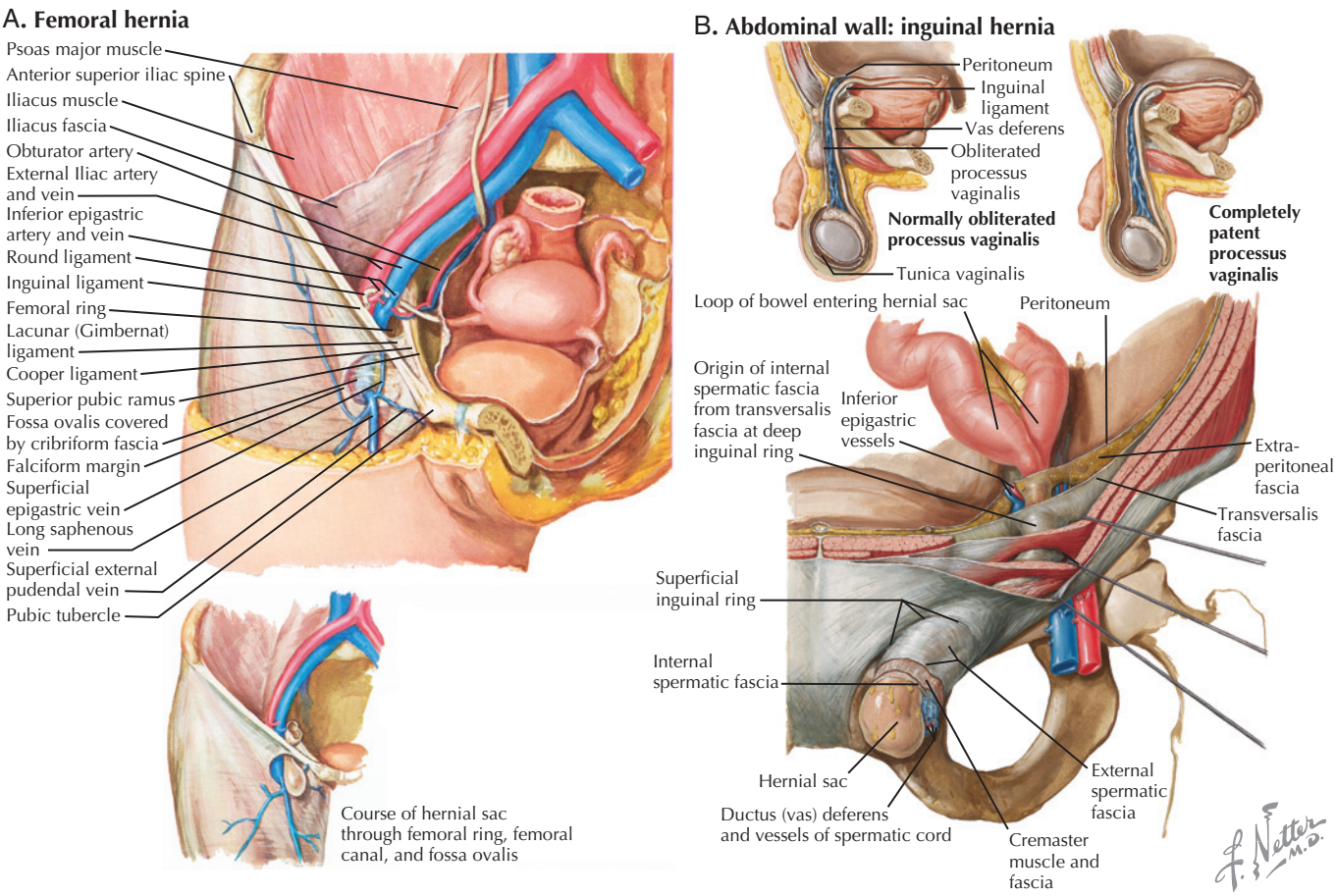


Figure 46-9 Hernias.

ioinguinal nerve is occasionally trapped in scar tissue within torn aponeurosis.
- Other: Muscular strain, hydrocele, or varicocele.

Treatment: Prompt surgical repair for large or symptomatic hernias. Surgical exploration and repair for chronic groin pain with inability to perform sport despite 6 to 12 weeks of relative rest and rehabilitation. With associated adductor injuries, tenotomy has been performed in conjunction with transversalis fascia repair to prevent recurrences. Avoid activities that stretch or pull abdominal muscles for 2 to 4 weeks after repair; then gradually resume progressive exercise and conditioning. By fourth week, begin abdominal, back, and pelvic strengthening.

Return to play: Return to noncontact sports after 6 to 8 weeks for indirect hernia repairs; contact sports, 8 to 10 weeks. More extensive repairs of direct inguinal, femoral, and anterior abdominal wall hernias require longer recovery; contact sports at 12 weeks.

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Thoracic and Lumbosacral Spine Injuries

47

Brock Schnebel

INTRODUCTION

General Principles

- With an increased number of adults and adolescents participating in fitness programs and competitive sports there has been an increase in thoracic and lumbar spinal problems.
- Most injuries are soft tissue injuries and proper training and avoidance of aggravating activities may allow participation while the pain resolves.
- Treatment of the athlete can be complicated by his or her competitiveness and the fact that the athlete will be returning to the activity that precipitated the injury.
- Primary treatment objective is the protection and preservation of the nervous system.
- Spine injuries occur with a reported incidence of 7% to 27% of injuries in sports.

Anatomy

- The spine is a mechanical structure consisting of bones, joints, ligaments, and muscles surrounding and distributing neural elements.
- The thoracic spine is more stable than the lumbar and cervical spine and is less mobile because of the thoracic cage.
- The spinal column is composed of 33 vertebrae divided into five sections: cervical (7), thoracic (12), lumbar (5), sacral (5), and coccygeal (4) (Fig. 47-1).

Osseous

Vertebral body: Large, strong, anterior weight-bearing structure (see Fig. 47-1).

Posterior vertebral arch: Semicircular-shaped structure surrounding the central canal, above and below the **vertebral foramen** through which the roots pass. Composed of **pedicles,** which project dorsally off the body, one on each side, and the **lamina,** which connects the pedicles. From the pedicles and lamina project the **transverse process.** From the lamina project the **spinous process.** These are locations for muscular attachments. Each lamina articulates with the lamina above and lamina below through **facet joints.** These **synovial-lined** facet joints are formed by a **superior articular process** from the lamina below and an **inferior articular process** from the lamina above and are surrounded by a facet capsule or ligament (see Fig. 47-1).

Pars interarticularis: Literally the "part between the joints" or the area between these facet joints. Relevant because it is the weak link anatomically and is susceptible to injury and stress fracture.

Sacrum: Coalesced lower segments of the spine that articulate with the pelvis.

Costovertebral joints: Area of articulation of the thoracic vertebrae with its rib.

Disc: Major ligament connecting each vertebral body. Composed of two types of tissue: anulus fibrosus (outer layer of fibers that functions to hold the nucleus pulposus and restrains the vertebral bodies) and nucleus pulposus (gelatinous structure located in the anulus)—both are responsible for load bearing.

Ligaments

Anterior longitudinal ligament: Strong bond of fibrous tissue that runs the entire length of the spine along the anterior vertebral bodies (see Fig. 47-1)

Posterior longitudinal ligament: Weaker than anterior; runs along the posterior surface of the vertebral bodies.

Ligamentous flavum: Very strong ligament attaching the lamina above to the lamina below.

Facet capsule: Connects each superior articulating process with its corresponding inferior articulating process.

Interspinous ligament: Connects each spinous process to the one below.

Supraspinous ligament: Runs along the dorsal surface of the tips of the spinous processes.

Costo-transverse ligaments: Thoracic area.

Muscles and Fascia

Thoracolumbar fascia: Investing tissue that separates the muscular compartments and fuses with the aponeuroses of several muscles (see Fig. 47-1).

Anterior groups of muscles: Anterior to the transverse processes, include psoas, intertransversalis, quadratus, and the levator costae.

Posterior groups of muscles: Posterior to the transverse process, include superficial muscles, erector spinae (semispinalis, longissimus, and the iliocostalis), deep muscles, mulifidus, rotares, and interspinalis.

Accessory group of muscles: Includes the abdominal muscles, the latissimus dorsi, the rhomboids, and the gluteus maximus.

Neural Elements

Neural elements of the spine include the spinal cord from occiput to about L1, the conus medullaris or lower portion of the cord thickened by the mass required to innervate the lower extremities located T11 to L1, and the cauda equina from L1 to the sacrum. The nerve roots exit at each level on both sides of the canal via the vertebral foramina (see Fig. 47-1).

Vascular Elements

Vascular elements of the spine include a complex system of intradural and extradural arteries and veins that supply the neural elements.

Biomechanics

- Spinal column has five principle functions:
  - Support of the head.
  - Support of the abdominal contents and pelvic girdle.
  - Point of attachment for thoracic cage and muscles.
  - Protection of the spinal cord and the neural elements within while allowing motion.
  - The biomechanical transfer of the weight and bending movements of the head and trunk to the pelvis.
- Understanding several basic concepts of biomechanics helps us understand how specific activities can exacerbate symptoms in certain syndromes:
  - In the spine, flexion of the lumbar spine increases the size of the intervertebral canal and the intervertebral foramina.
  - Extension decreases the size of the intervertebral canal and the intervertebral foramina.
  - Flexion increases dural sac and nerve root tension.
  - Extension decreases dural sac and nerve root tension.
  - Front flexion, axial loading, and an upright posture increase intradiscal pressure: pressure is greater in sitting, less in standing, and least in lying.
  - With flexion the anulus bulges anteriorly.
  - With extension the anulus bulges posteriorly.
  - Nuclear shift in an injured disc is poorly documented, but the disc probably shifts in the direction of the anular bulge.
  - Rotation and torsion produce anular tears and disc herniations.

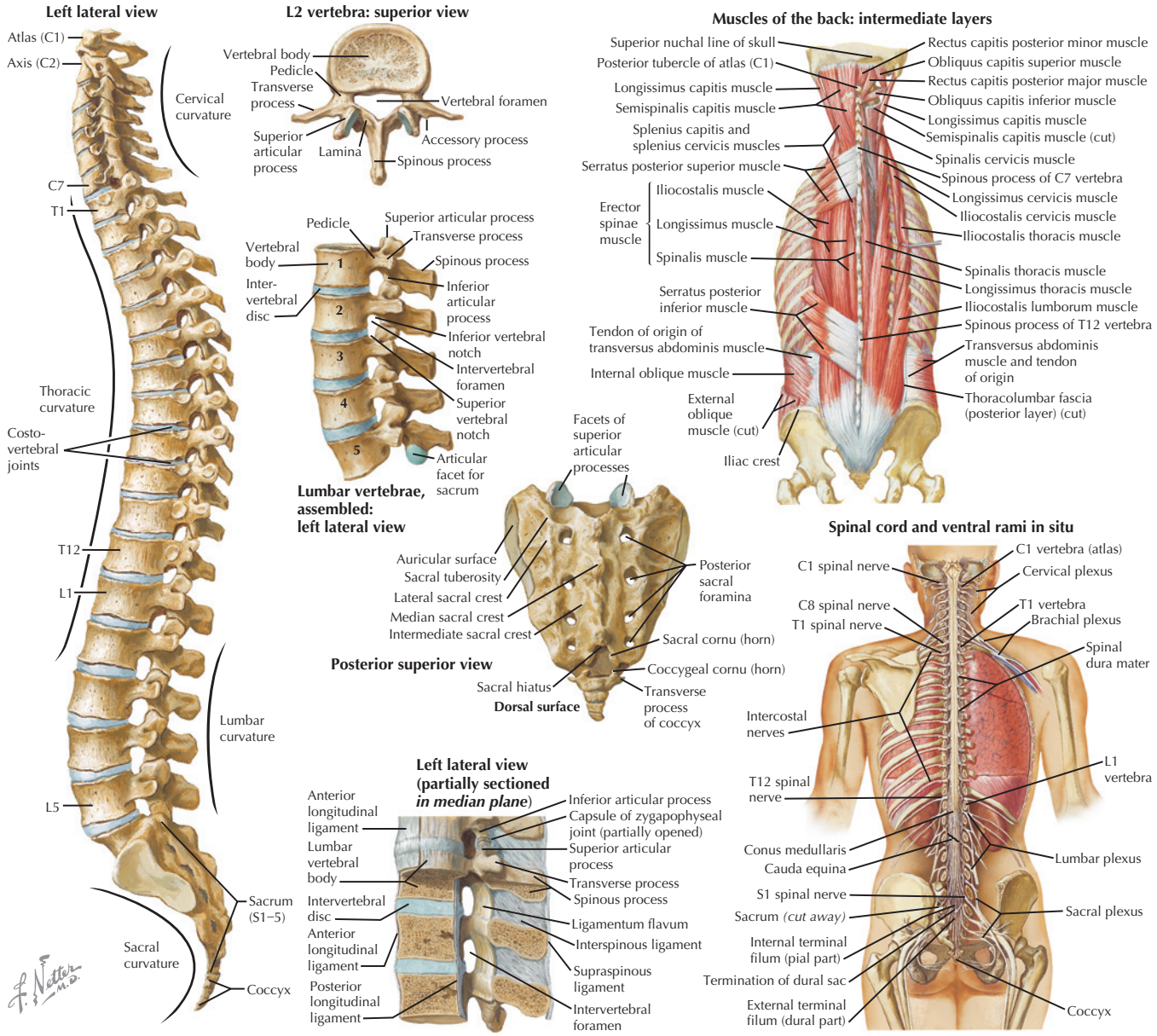


Figure 47-1 Anatomy of the Spine.

History and Physical Examination

History

Information obtained in the history and physical examination is critical. The following questions are particularly relevant.

When and how did your symptoms start? Mechanism of injury can help locate the damage. Onset can be telling because a gradual onset can be more related to a stress injury and a rapid severe onset may indicate a more acute injury.

What is the location of the pain? Neurologic involvement is likely when a patient perceives more pain in the leg than in the back. The percentage of back to leg pain is relevant. Associated neurologic symptoms such as numbness, dysesthesia, weakness, or spasticity are important to note. There are multiple nociceptive sources in the spine.

When does it hurt? Night pain may be more ominous and associated with tumors. Pain with motion and relieved by rest implies mechanical pain. Constant pain unaffected by rest may indicate an inflammatory component.

What makes it worse? Discogenic pain is usually worse with flexion or prolonged sitting and increases with a Valsalva maneuver (cough or strain with defecation). Pain from a spondylolysis is worse with hyperextension. Pain from spinal stenosis (pseudoclaudication) is worse with ambulation or prolonged standing. Sacroiliac dysfunction can be worse with hyperextension.

What makes it better? Discogenic pain can be improved with lying down with knees and hips flexed (opens foramina and unloads disc). Spinal stenosis symptoms can improve with bending forward (as in walking over a grocery cart) or with sitting.

How do you train? This may reveal a voluntary restriction that helps with a diagnosis or reveals a training error compatible with a specific syndrome.

Do you have any of these factors? Bowel, bladder, or sexual dysfunction may indicate significant neurologic involvement of the cord, conus, or cauda equina. Weight loss, anorexia, night pain, and pain at rest may suggest neoplasia. Fever may indicate infection. A visceral type of pain with referral may indicate kidney, prostate, bowel, or vascular pathology.

Physical Examination

Should address inspection, palpation, and percussion and identify:

- Exact location of tenderness, dysesthesias, or numbness
- Maneuvers that reproduce the pain
- Presence of neural tension signs
- Deficits in range of motion
- Any neurologic deficit

Inspection of posture and stance: Patients in a sitting position may tend to tripod or lean back to unload the spine. When standing, a list to one side may suggest nerve root compression. The appearance of a flat back with a vertical sacrum may be seen in advanced spondylolisthesis. Scoliosis may be detected in a standing position.

Inspection of gait: Myelopathic patients may walk with spasticity. A forward flexed posture can be seen in stenosis. Patients with discitis may have a rigid short-stride gait.

Inspection of range of motion: May be limited by painful conditions. Extension may be limited in stress fractures and facet syndromes. Discogenic pain may limit flexion.

Palpation: May detect muscle spasm, deformity with a palpable mass, scoliosis, spondylolisthesis, or a gibbus deformity. Tenderness may present at the sciatic notch.

Percussion: May elicit tenderness in trauma or infection and costovertebral angle tenderness may imply a kidney problem.

Neurologic: Most critical portion of the exam (Table 47-1 and Fig. 47-2). **Upper motor neuron findings** of spasticity, weakness, numbness, hyper-reflexia, and clonus may indicate spinal cord pathology; **lower motor neuron findings** of flaccid muscles, weakness, numbness, hyporeflexia may indicate cauda equina or root injury. Sensory levels on the trunk will help determine the level of spinal cord injury if present. A rectal and cremasteric reflex exam is recommended with any spinal cord injury.

Table 47-1 COMMON NERVE ROOT FINDING

Root	Strength	Sensation, dermatomal distribution	Reflex
L1-L2	Iliopsoas	Inguinal area and upper two-thirds of anterior thigh	
L3	Quadriceps	Oblique band of distal third of anterior thigh immediately above patella	
L4	Tibialis anterior and quadriceps	Medial side of lower leg and foot	Patellar tendon
L5	Extensor hallucis longus, and tibialis anterior	Anterior aspect of lower leg and dorsum of foot	Not reliable (posterior tibialis reflex present in only 40%-50% of population)
S1	Peroneus longus and brevis gastrocnemius	Lateral aspect of lower leg and lateral foot	Achilles tendon reflex

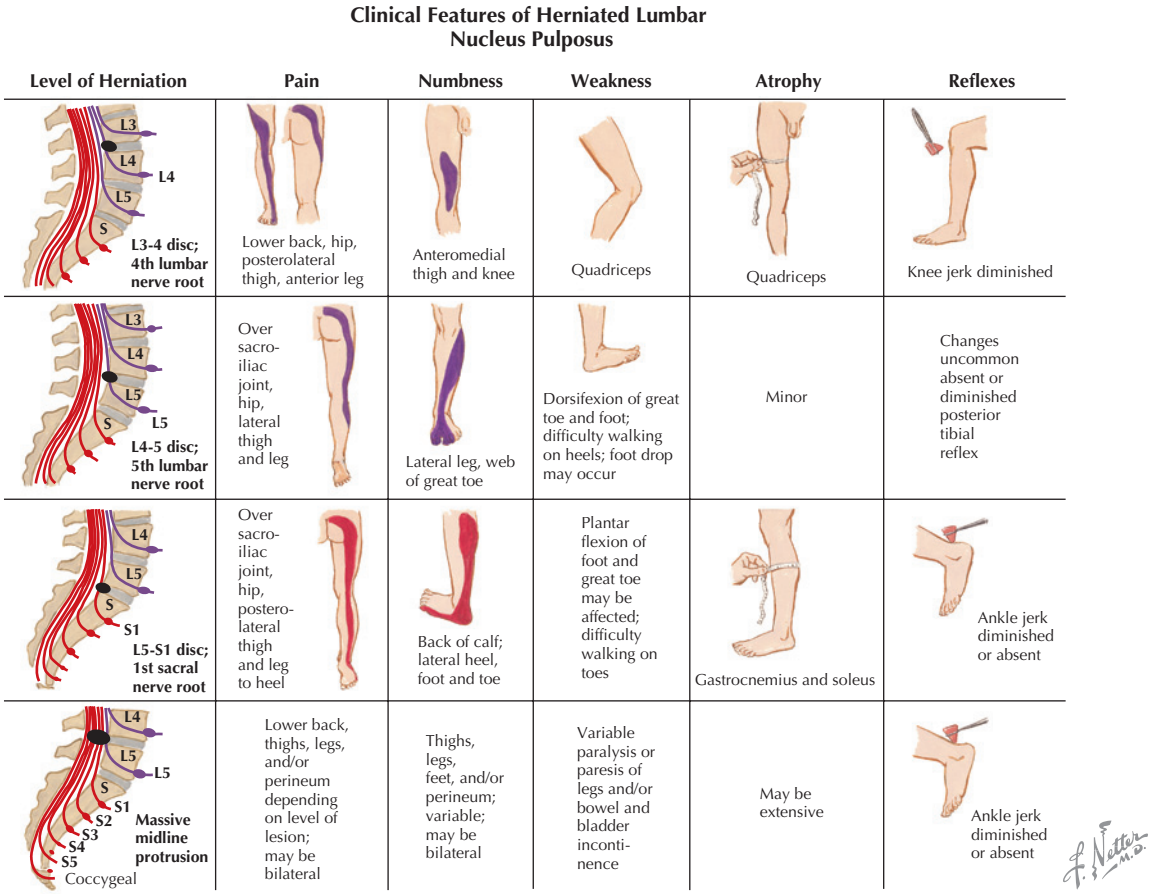


Figure 47-2 Dermatomal Distribution of Nerves.

Special Tests

Pain from Neural Source

Straight leg raise test (SLR): Tension test that indicates nerve irritation in the sciatic nerve if positive with radicular pain at less than 60 to 70 degrees of leg elevation (Fig. 47-3).

SLR with foot dorsiflexion (Lasègue's test): Tension test suggesting sciatic nerve irritation if painful.

SLR while sitting: Positive when extending the knee while patient is sitting causes radicular pain. Patient may lean back to gain relief. A positive SLR and a negative SLR while sitting can be inconsistent and suggest other etiologies.

Crossed SLR: Positive test may be pathognomonic for a herniated disc. With the patient sitting the examiner lifts the unaffected leg; in the presence of a herniated disc this may produce pain in the affected leg.

Femoral stretch test: In side-lying position with knee flexed 90 degrees, the hip is extended, stretching the femoral nerve. Pain on the anterior thigh suggests femoral nerve or upper lumbar root irritation.

Pain from Structural Source

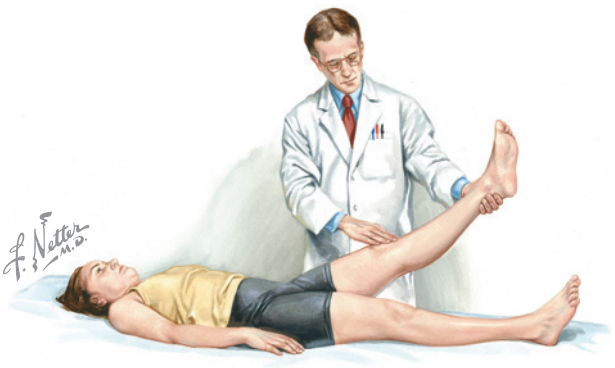
Jackson's one-legged standing hyperextension test: If painful, it suggests a stress fracture (spondylolysis), a facet syndrome, or sacroiliac dysfunction.

FABER (flexion, abduction, and external rotation of hip) or Patrick's test: Can suggest hip joint or sacroiliac dysfunction.

Supine-to-long sitting test: Examiner tests leg length at the medial malleoli with the patient first supine then sitting with both legs extended at the knees. A change in relative leg length may indicate sacroiliac dysfunction.

Radiographic and Ancillary Testing

Plain films: May be unnecessary in the acute phase in a nonathletic clinic setting but in high velocity sports and collision sports early x-ray evaluation may be needed. Plain radiographs may reveal an acute fracture or spondylolysis that may alter treatment. Plain films may also reveal spondylolisthesis, degenerative changes, signs of infection in discitis (endplate irregularities), or congenital anomalies. An anterior-posterior and lateral of the lumbar spine may be augmented by right and left obliques if a spondylolysis is suspected.



Straight leg raising test
Perform with the knee extended. Flex hip until resistance and/or pain is noted. Test places sciatic nerve, as well as hamstrings on stretch. Patients with herniated disc causing compression of sacral nerve roots often experience back pain radiating to lower extremity.

A *crossed straight leg raising test* is pain in the affected extremity when the contralateral leg is raised and is highly specific for nerve root entrapment.

Figure 47-3 Straight Leg Raise Examination Test.

Bone scan: May be used to reveal lesions that are not yet visible on plain films (e.g., stress fractures in the pars interarticularis). Because a positive bone scan indicates increased metabolic activity, this scan may reveal bone tumors or infection as well as occult fractures. It may take 5 days after the onset of symptoms for a bone scan to be positive.

Computed tomography (CT) scan: To evaluate osseous lesions and to help stage stress reactions.

Magnetic resonance imaging (MRI): To help detect discogenic lesions such as degenerative disease with desiccation, disc herniations, spinal cord lesions, fractures, stress reactions, and compressive lesions.

Myelography and CT myelography: May also be used to show compressive lesions.

Electromyography (EMG) and nerve conduction velocity (NVC): May help in localizing the location of a nerve compression lesion. It may take 3 weeks before denervation changes occur such that they are revealed on an EMG.

Laboratory studies: Can be useful in diagnosing discitis, inflammatory spondylitis, or neoplasia, but these studies are rarely needed.

SPECIAL INJURIES AND PROBLEMS

Traumatic Injuries to the Thoracic or Lumbar Spine

Description: Motor sports, high-velocity sports, and collision sports are capable of producing significant forces to the thoracic and lumbar spine. Fractures, fracture dislocations, and dislocations can occur with and without neurologic injury (Fig. 47-4). Blunt trauma may produce rib fractures and transverse process fractures. Any of the ligamentous structures may be injured, leading to very specific treatments.

Mechanism of injury: Trauma.

Presentation: Pain, limited motion, possible neurologic findings with neurologic distribution of pain.

Physical exam: Local tenderness, possible deformity, neurologic findings with lower motor neuron findings with a lumbar injury (canda equina) or upper motor neuron findings with a cord injury.

Differential diagnosis: Be aware of injuries at multiple levels.

Diagnostics: Imaging techniques, CT, MRI.

Treatment: After on-the-field management and transfer to a care facility, further treatment plans can be made depending on exam and imaging. Some traumatic injuries can be managed nonsurgically but surgical intervention may be required in others.

Prognosis and return to play: Dependent on specific injury and neurologic involvement.

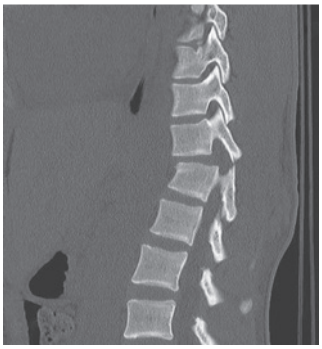
Thoracic Disc Herniation

Description: The extrusion of nucleus pulposus out of its contained position in the anulus fibrosus. Nuclear material may cause a protrusion in the anulus (contained) or escape from the anulus, termed an extrusion or free fragment.

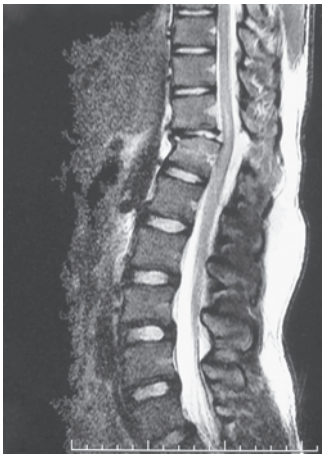
Mechanism of injury: Anular tears occur with torsional loads under pressure, through these tears the nucleus can extrude gradually or suddenly. Most herniations occur at the posterolateral portion of the anulus, where the anulus fibrosus merges with the posterior longitudinal ligament.

Presentation: Thoracic herniations may present as chest wall pain, thoracic back pain, exiting root pain along a rib, or as myelopathy if the cord is involved. Upper thoracic herniations may present as a cervical problem with pain radiating to the medial brachium. Lower thoracic herniations may present as lumbar disease.

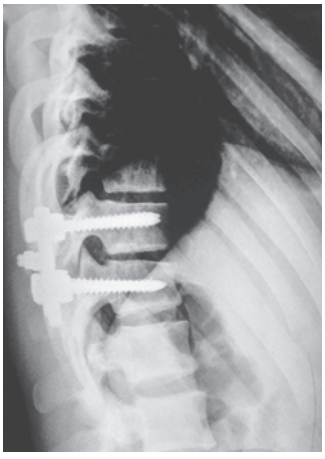
Physical exam: If the patient is myelopathic, he or she will have upper motor findings of spasticity, weakness, clonus, positive Babinski. Exiting nerve root distribution of numbness or hyperesthesia may be present.



A. CT shows facet dislocation.



B. MRI shows edema in cord, kyphotic deformity, and rupture of the ligamentum flavum.



C. Plain film shows internal fixation postoperative.

Figure 47-4 NCAA football player sustained an accidental injury in the weight room resulting in a fracture dislocation of T11 on T12, with a conus medullaris spinal cord injury.

Differential diagnosis: Cervical and lumbar herniated discs, tumors (benign, primary, metastatic, neural, bone, malignant), fractures (both stress and traumatic), spine or rib (Fig. 47-5).
Diagnostics: Diagnosis is confirmed with MRI or myelogram with CT.

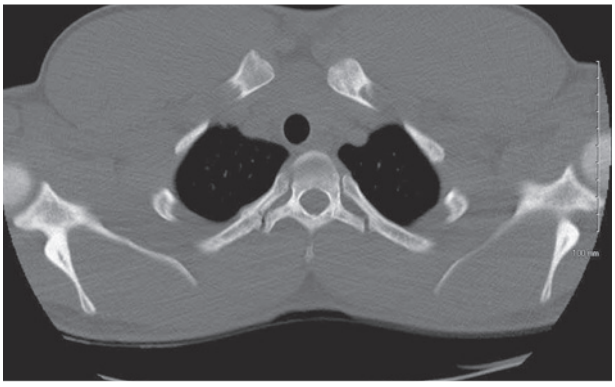


Figure 47-5 CT scan done after a bone scan of a NCAA football player shows a posterior rib fracture that enters the costo-transverse joint. This may manifest as a thoracic disc herniation.

Treatment: Nonoperative treatment is with medications, and physical therapy with a stabilization program. Surgical interventions indicated for significant progressive myelopathy and unrelenting radiculopathy.
Prognosis and return to play: Variable; return should be slow and cautious with intense rehabilitation.

Apophysitis

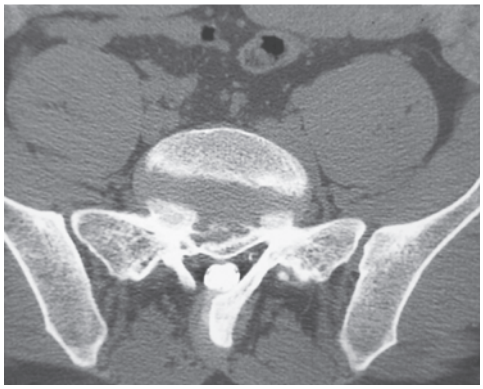
Description: Injury or inflammation of the ring apophysis of the spine.
Mechanism of injury: Caused by repetitive traction on the anterior longitudinal ligament or repetitive compressions with endplate microfractures.
Presentation: Presents as back pain that increases with activity and is relieved by rest.
Physical exam: Normal; unlike Scheuermann's disease there will not be a kyphosis.
Differential diagnosis: Stress fractures, traumatic fractures, spondyloarthropathy.
Diagnostics: Plain films will reveal irregularities of the ventral apophysis; bone scan may have increased activity.
Treatment: Rest.
Prognosis and return to sport: Excellent; return is variable depending at length of symptoms.

Slipped Apophyseal Ring

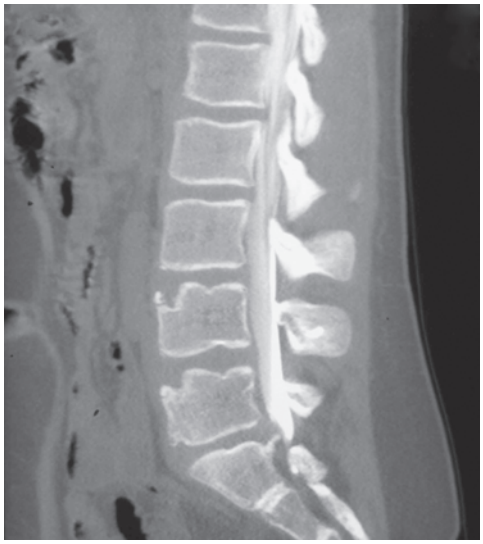
Description: Posterior fracture of the ring apophysis with protrusion of the bone rim and disc into the canal (Fig. 47-6).
Mechanism of injury: Same as a herniated disc but in skeletally immature.
Presentation: Can produce back pain only, especially if the disc herniates anteriorly or into the vertebral body producing a Schmorl's node; this is very common (see Fig. 47-6). Can present with leg pain if the protrusion enters the canal and will behave like a herniated disc.
Physical exam: May have same findings as a herniated disc: list, weakness/numbness, tension findings, reflex deficit.
Differential diagnosis: Herniated disc, stress fracture, SI joint.
Diagnostics: MRI, myelogram with CT, EMG, and NCV.
Treatment: Similar to herniated nucleus pulposus (see later section).
Prognosis: Same as herniated nucleus pulposus (see later section).

Acute Lumbar Strain

Description: Muscle or muscle tendon unit strain.
Mechanism of injury: Fatigue failure or over stretch injury to a muscle tendon unit about the lumbar spine; usually with bending, rotation, and/or improper lifting.



A. Axial CT with bone remodeling.



B. Sagittal cut showing protrusion of disc and calcified slipped apophysis at superior S1 and Schmorl's nodes at anterior L4 and L5.

Figure 47-6 Slipped apophyseal ring. CT scan of a junior high basketball player with back and leg pain shows a slipped apophyseal ring at superior aspect of S1 and anterior Schmorl's nodes at L4 and L5.

Presentation: Acute onset, back pain, no neurologic complaints.
Physical exam: Localized tenderness, with or without spasm.
Differential diagnosis: Stress reaction or fracture, SI joint pain.
Diagnostics: X-rays may not be required by history or exam but will be normal or show a straight spine related to spasm.
Treatment: Conservative care with rest, modalities, and occasional use of medications. To prevent recurrences a rehabilitation program with a spine stabilization exercises will be helpful. This is based on core strength and teaches the principle of muscle fusion; use of core muscles to brace the spine. Goal is to find the neutral position or position of least pain and use this protectively. Overall flexibility and strength is stressed through a well-defined program. Progressive aerobic training and sport-specific training is added as skills improve.
Prognosis: Excellent.

Lumbar Myonecrosis and Potential Compartment Syndrome

Description: Rare reports of compartment syndrome of the lumbar paraspinal muscles. More recently, events of lumbar myonecrosis with rhabdomyolysis have been reported; associated with sickle cell trait in athletes, and can be significant.
Mechanism of injury: Overuse, possibly associated with sickle cell trait.
Presentation: After aggressive exercise, back pain, muscular, spasm, cramping sensation, dark urine occurs if rhabdomyolysis significant. Back pain more severe and not motion related as compared to lumbar strain.
Physical exam: Posturing, spasm, neurologically intact.
Differential diagnosis: Lumbar strain, acute fracture, acute stress fracture.
Diagnostics: MRI, laboratory studies (creatine kinase), sickle cell testing.
Treatment: Rest when it occurs, observation looking for progression of rhabdomyolysis. Treatment of potential causes of rhabdomyolysis, hospitalization if needed. Long term management with appropriate guidance.
Prognosis: Excellent if detected early and protective measures taken.

Discogenic Syndromes

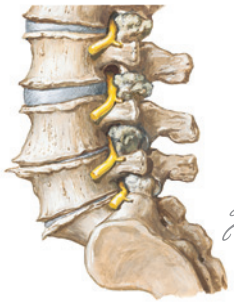
Anular Tears

Description: A tear of the anulus fibrosus. Nucleus material may or may not be extruded or displaced. As the tear occurs at the periphery, it is more likely to be painful and may be associated with referred radicular pain or a chemical neuritis.
Mechanism of injury: Rotational stress, or rotation with compression.
Presentation: Pain, local back and possible dermatomal distribution of referred pain, often with spasm and postural changes, limited motion.
Physical exam: Neurologically normal exam, but may be posturing, with spasm.
Differential diagnosis: Early herniated nucleus pulposis, stress fracture, acute lumbar stain, fracture, SI joint pain.
Diagnostics: Plain films may be normal or show degenerative changes, straight spine if spasm significant. MRI may be normal but may show an area of increased signal intensity in the anulus, especially at the outer anulus on T2 weighted images.
Treatment: Modalities, possible nonsteroidal anti-inflammatory drugs (NSAIDs) or medrol dose pack and physical therapy with a spine stabilization program.
Prognosis: Excellent, but may cause prolonged and often recurrent symptoms.

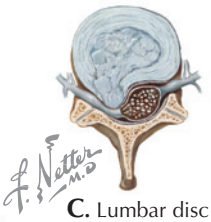
Herniated Nucleus Pulposis (HNP)

Description: The nucleus pulposis extrudes partially or completely through the anulus fibrosus, protruding into the neural elements or extruding into the canal and possibly being sequestered away from the anulus (Fig. 47-7). The herniation usually occurs to one side or the other of the posterior longitudinal ligament, which is a restraint, but may exit centrally through the posterior ligament. It can also exit far lateral to involve the root in the foramen. It is most common at the lower segments; most common in the 3rd or 4th decade.
Mechanism of injury: Repetitive flexion and rotation increases the load on the disc. Nachemson has measured the intradiscal pressures in various positions and this pressure is increased with flexion.
Presentation: May present as back pain as the anulus tears in the process of the disk extruding. As it extrudes and as the nerve becomes involved the back pain will become associated with buttock pain or extremity pain. The back pain may lessen as the radicular pain, numbness, and dysesthesias progress along a dermatomal distribution. Weakness is possible. Pain is usually worse with prolonged sitting, flexion, or Valsalva maneuvers. If severe compression occurs part or all of the cauda equina may be involved, leading to weakness, bowel, bladder, and sexual dysfunction (cauda equina syndrome).
Physical exam: Patient may have a list, may tripod, may have limited range of motion (ROM), and may have spasm. Tension

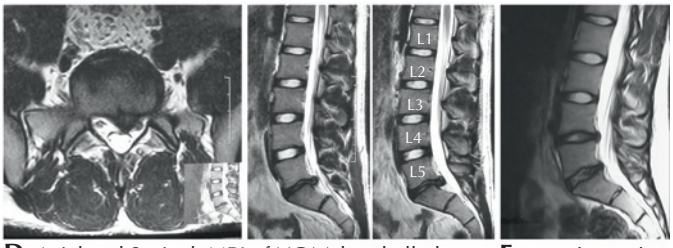
A. Radiograph of thoracic spine shows narrowing of intervertebral spaces and spur formation.



B. Degeneration of lumbar intervertebral discs and hypertrophic changes at vertebral margins with spur formation. Osteophytic encroachment on intervertebral foramina compresses spinal nerves.



C. Lumbar disc herniation. Schematic cross section showing compression of nerve root.



D. Axial and Sagittal. MRI of NCAA baseball player shows a herniated disc at L5–S1compressing the S1 nerve root.

E. MRI shows disc degeneration at two levels in a NCAA softball player.

Figure 47-7 Discogenic Syndromes.

tests are positive and a neurologic exam may reveal numbness and weakness.

Differential diagnosis: Stress fracture to lumbar spine, sacrum, or pelvis, anular tear with referred pain, plexopathy, peripheral neuropathy or nerve injury, SI joint pain, fracture (see Figs. 47-6 and 47-7).

Diagnostics: Plain films may be normal or show a narrow disc space. MRI will show the lesion (see Fig. 47-7). Myelogram and myelo-CT will also reveal the lesion but are more invasive. An MRI with gadolinium may be required to distinguish postsurgical scarring from a recurrent herniated disc. EMG and NCV studies may be helpful to rule out a peripheral neuropathy that can masquerade as a radiculopathy. This may be needed because 32% of imaging techniques may be positive in asymptomatic subjects. A positive study is not diagnostic unless it confirms the clinical picture.

Treatment: Conservative with medications, possible Medrol dose pack, followed by NSAIDs. Epidural steroid treatment may be used. Surgery may be required for intractable pain, progressive neurologic deficit, or lack of improvement after 8 to 12 weeks. A cauda equina syndrome requires urgent surgical care.

Prognosis and return to play: Many will completely recover with nonsurgical care. This followed by aggressive rehabilitation and a prolonged care program can lead to return to sport. In some studies 70% to 90% of athletes treated surgically are able to return to an elite level. Strength programs and lifting as a part of training should be adjusted appropriately.

Degenerative Disc Disease

Description: Anular tears and herniated discs are part of the spectrum of degenerative disc disease. Degeneration of the discs leads to collapse and then concomitant degeneration of the facet joints and hypertrophy of the facet capsule, which can lead to stenosis.

Mechanism of injury: Chronic degeneration of the disc.

Presentation: May present with an anular tear or herniated disc, but also as a facet syndrome with symptoms from stenosis, either foraminal or central. They may have back pain, neurologic radicular pain, referred facetal pain, or neurogenic claudication pain. Symptoms may be positional and worse with extension and improved with flexion. Some athletes may have congenital stenosis with the addition of acquired degenerative changes then become symptomatic (see Fig. 47-7).

Physical exam: May have neurologic findings, limited ROM; may have a normal exam; may be seeking postural changes for relief of symptoms.

Differential diagnosis: Stress fracture, SI joint pain, fracture, congenital stenosis.

Diagnostics: Imaging techniques, MRI, myelogram, and CT.

Treatment: Specific to symptoms; medicines and stabilization protocol for back symptoms. Facet injections for facet syndrome; epidural steroids for stenotic neurogenic symptoms. Occasionally, surgical decompression may be needed for intractable limiting pain or neurologic deficit. Surgical decompression in an athlete, however, can lead to a weakening of the lamina and predispose the athlete to pars fractures and spondylolisthesis and may limit his or her return to sport.

Spinal Stenosis

Description: Narrowing of the space available for the nerves. May be congenital with short pedicles or a narrow interpedicular distance or acquired related to trauma or degeneration. It may be central (spinal canal) or foraminal.

Mechanism of injury: Congenital, traumatic, or degenerative. May be caused by a synovial cyst or herniated disc.

Presentation: Neurologic symptoms, radicular pain, neurogenic claudication, postural radicular pain.

Physical exam: May be normal; if severe may have neurologic findings.

Differential diagnosis: Herniated disc, stress fracture, fracture, SI joint pain, peripheral neuropathy.

Diagnostics: Imaging by MRI or myelo-CT. EMGs and NCV may be useful (Fig. 47-8).

Treatment: Specific to cause. If symptoms are related to a synovial cyst, then aspiration or injection may be useful. If congenital, then surgical decompression may be required. If acquired degenerative, then conservative measures such as medication and epidural steroids may help prolong an athletic career.

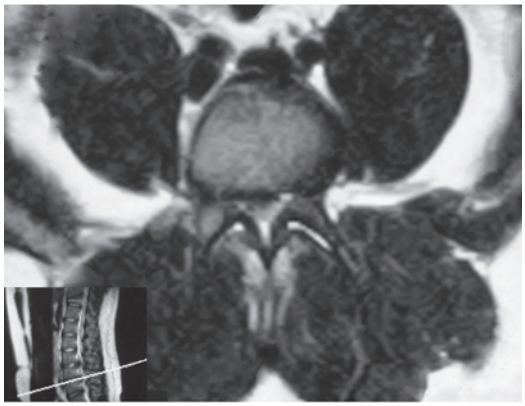
Prognosis and return to play: Variable.

Stress Reaction, Stress Fracture, Spondylolysis

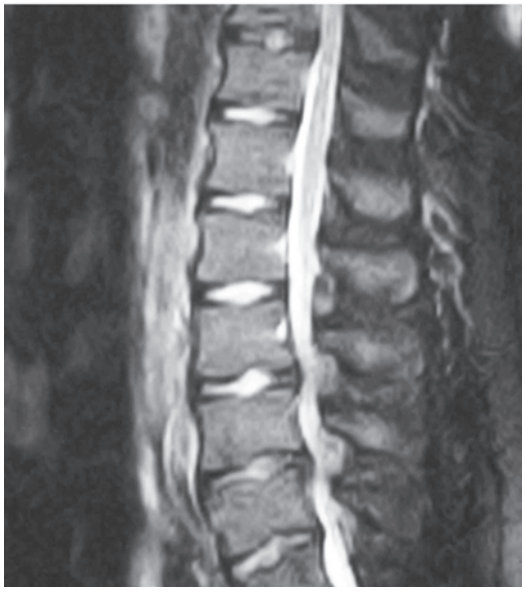
Description: A spectrum of overuse injuries to the pars interarticularis or pedicle area of the bone; very common in athletes.

Mechanism of injury: Repetitive shear forces, worse with repeated extension and hyperextension maneuvers that load this portion of the bone.

Presentation: Back pain, occasionally with some radicular pain. Worse with extension. Present as a bone stress reaction without a fracture on one or both sides, or as a nonunion of a stress fracture known as a spondylolysis (spondylo = spine, lysis = defect) on one or both sides (Fig. 47-9). Pain can be present on



A. Axial image shows congenitally short pedicles and triangular narrowed canal.



B. Sagittal image shows degeneration with acquired changes exacerbating a congenital stenosis.

Figure 47-8 Spinal stenosis. MRI of NCAA football player shows spinal stenosis.

one or both sides, worse with motion or running. Often has onset in strength and conditioning workouts.

Physical exam: Extension and one-legged extension tests are positive. Neurologic exam is negative; tension tests are negative. Pain may occur with rotation or side bending.

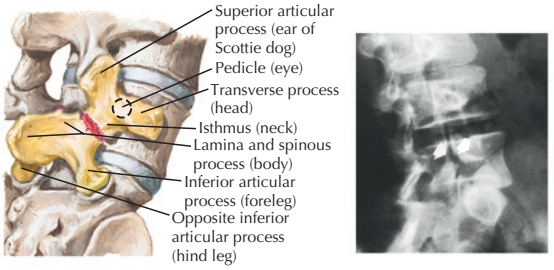
Differential diagnosis: Anular tear, HNP, SI joint pain, stress fractures; in the sacrum or pelvis, facet syndrome.

Diagnostics: Plain films include AP, lateral, and oblique views. They may be normal early in a reaction and some defects are not clearly seen. Oblique films can show a defect as "the neck of the Scotty dog" (see Fig. 47-9). Sometimes the defect can be seen on a lateral film. If plain films are negative, nuclear imaging will be helpful. A whole body conventional biplanar bone scan can visualize the pars area but also can rule out a sacral or pelvic stress fracture that mimics a lumbar injury. A bone scan combined with a single photon emission computerized tomography (SPECT) will improve sensitivity and localization of small abnormalities by screening out other tissues. A positive scan indicates an acute lesion and suggests bone healing potential.

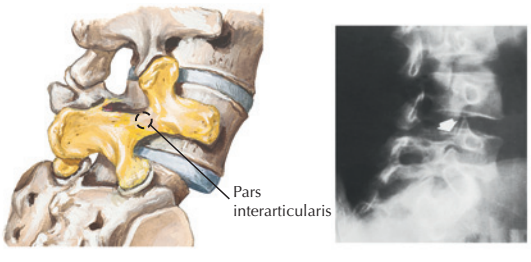
Treatment: Guided by symptoms and images; restrictions are controversial, especially bracing.

- Normal plain film, positive scan indicates an acute lesion. A positive scan indicates metabolic activity and some potential for healing. A modified Boston orthosis may be indicated. Limit activities for 4 to 8 weeks. Rehabilitation exercises to begin as symptoms abate. Strength and neutral position are stressed. On occasion with a positive scan a thin slice CT may help evaluate the lesion and help with treatment. If CT scan normal, no brace. If CT scan shows acute fracture line, brace. If CT scan shows fracture with sclerotic margins (chronic lesion), no brace required.
- Positive plain film and positive scan indicate a semiacute lesion. A zero-degree antilordotic modified Boston orthosis may be indicated. Micheli reports success with use 23 hours per day, for 12 weeks, but use is variable. Goal is to be pain free prior to starting rehabilitation exercises. The lesion may not heal with bone, so progression is based on symptoms.
- Positive plain films with a negative bone scan indicate a chronic lesion or nonunion. A negative scan suggests minimal metabolic activity and poor healing potential. Start rehabilitation when symptoms abate. Occasionally, surgery is required if pain is intractable.

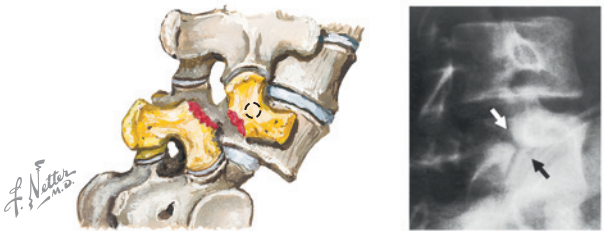
Prognosis and return to play: Excellent.



Spondylolysis without spondylolisthesis. Oblique view demonstrates formation of radiographic Scottie dog. On oblique radiograph, dog appears to be wearing a collar.



Dysplastic (congenital) spondylolisthesis. Luxation of L5 on sacrum. Dog's neck (isthmus) appears elongated. Dysplastic facets may lead to lystesis.



Isthmic type spondylolisthesis. Anterior luxation of L5 on sacrum due to fracture of isthmus. Note that gap is wider and dog appears decapitated.

Figure 47-9 Spondylolysis and Spondylolisthesis.

Spondylolisthesis

Description: One vertebral body is slipping relative to another (spondylo = spine, listhesis = slippage) (see Fig. 47-9). Usually the upper body is slipping forward on the body below, resulting in a kyphotic deformity at the slip and a reactive lordosis above.
- There are five types of spondylolisthesis:
  - Dysplastic (abnormal anatomy).
  - Isthmic (caused by a defect in the pars); this is the most common in young athletes.
  - Degenerative (caused by lax facet joints and a degenerative disc that allows the slippage)—more common with age.
  - Traumatic (high velocity injuries).
  - Pathologic (tumors or osteopenic bone).
- Isthmic (the most common type in young athletes) is most common at L5 to S1 and can be graded on a scale of I to V, based on the amount of forward displacement of the higher vertebra on the vertebra below.
  - 0% to 25% is grade I.
  - 25% to 50% is grade II.
  - 50% to 75% is grade III.
  - 75% to 100% is grade IV.
  - Complete displacement or spondylolisthesis is grade V.

Mechanism of injury: Isthmic spondylolisthesis occurs as a result of repetitive overuse at a young age, probably related to extension activities. Progression of slips is most likely to occur between ages 9 and 12 in girls and 10 and 14 in boys.

Presentation: Patients with grade I and II slips may be asymptomatic and may be participating in sport not knowing about the defect. They may have back pain and occasional radicular pain; pain is worse with extension activities. Patients with higher grade lesions may have been limited by their pain and have more difficulty with sport because of back pain, stiffness, and hamstring tightness. Localized back pain is common and radicular pain may develop as a result of nerve root compression by hypertrophic callus or the deformity or instability.

Physical exam: With high-grade slips, there will be vertical sacrum, flat buttocks, compensating tight hamstrings. There may be a palpable step-off at the deformity and a short waist. Neurologic exam is usually normal.

Differential diagnosis: Just because a patient has a spondylolisthesis that does not mean that is the origin of the pain. Other possibilities include SI joint, degeneration of other levels, and stress fracture at other levels.

Diagnostics: Standing plain radiographs usually reveal the abnormality and a lateral projection allows grading. Repeat films should be obtained yearly if the diagnosis is made before the age of 10 because progression can occur. CT and MRI are useful if other sources are suspect.

Treatment: Conservative in most cases and involves trunk stabilization exercises. Occasional limits may be required for symptomatic events. Surgical fusion may be required with high-grade slips or with slips associated with neurologic symptoms related to nerve root compression or stenosis.

Prognosis and return to play: For low-grade slips, excellent to good; but high-grade slips can be more problematic and limiting.

Deformities

Postural Roundback (Flexible)

Description: A long gentle kyphotic deformity is usually seen in older adolescents. It is flexible and can be voluntarily corrected, unlike Scheuermann's.

Mechanism of injury: Postural.

Presentation: No pain; presents as a deformity.

Physical exam: Gentle round kyphosis.

Differential diagnosis: Scheuermann's kyphosis, congenital kyphosis.

Diagnostics: Normal spine has a forward kyphosis of 25 to 45 degrees. In postural roundback the kyphosis measured on a standing lateral from the inferior margin of T12 to the inferior margin of an upper level exceeds 40 degrees. A supine lateral film with hyperextension shows correction.

Treatment: Postural.

Prognosis and return to play: Sports participation unlimited.

Scheuermann's Disease

Description: A fixed rigid thoracic kyphotic deformity with or without pain; also known as juvenile kyphosis and may present between 8 and 12 years of age (Fig. 47-10).

Mechanism of injury: It is thought to be caused by necrosis of the ring apophysis, perhaps related to repetitive trauma.

Presentation: Usually asymptomatic but some patients report pain or fatigue. Incidence in adolescent rowers and weightlifters is high. Progression later in life is unusual.

Physical exam: Rigid kyphosis accentuated with forward flexion. It tends to produce a more acute angle deformity at midthorax as compared to a postural deformity.

Differential diagnosis: Postural roundback, congenital kyphotic deformity.

Diagnostics: Radiographs will reveal the kyphosis and a lateral film must show three consecutive thoracic vertebral bodies each wedged more than 5 degrees, with endplate irregularity or with Schmorl's node.

Treatments: May include observation, exercises, bracing, or even surgery.

Prognosis and return to play: Sports participation is unlimited.

Atypical Scheuermann's Disease

Description: Also known as thoracolumbar or lumbar Scheuermann's kyphosis.

Mechanism of injury: Same as Scheuermann's kyphosis.

Presentation: This kyphosis tends to be more painful; associated with sports that have vigorous spine loads (gymnastics, especially dismounts, and weightlifting).

Physical exam: Loss of lumbar lordosis.

Differential diagnosis: Old fractures.

Diagnostics: X-ray shows loss of lordosis; will have vertebral wedging and Schmorl's nodes but may not involve consecutive vertebrae.

Treatment: Conservative with rest and therapeutic exercises. Occasional bracing may be required.

Prognosis and return to play: No limits on sports but may occasionally be limited by pain.

Scoliosis

Description: A lateral spinal curvature of more than 10 degrees. More common in girls than boys and is present to a minor degree in 2% to 3% of the population.

Mechanism of injury: Of scoliosis cases, 80% are of unknown etiology and are called idiopathic. There is a strong familial factor. Less common congenital scoliosis results from a failure of formation or segmentation. Other types may be related to neurologic problems.

Presentation: Deformity, otherwise usually asymptomatic. A careful history and exam is required to rule out other causes for the scoliosis, most of which are neurologic.

Physical examination: Inspection from behind will reveal the curve and allow to check for balance. Forward bending will allow measurement of rib elevation. Neurologic exam will be normal in idiopathic scoliosis. Skin pigmentation changes, skin defects at the spine, and neurologic defects may suggest other types of scoliosis.

Differential diagnosis: Congenital scoliosis, neuropathic scoliosis.

Diagnostics: AP and lateral view on a 14-by-36 cassette to include the entire thoracolumbar spine and to allow assessment of skeletal maturity (Risser sign).

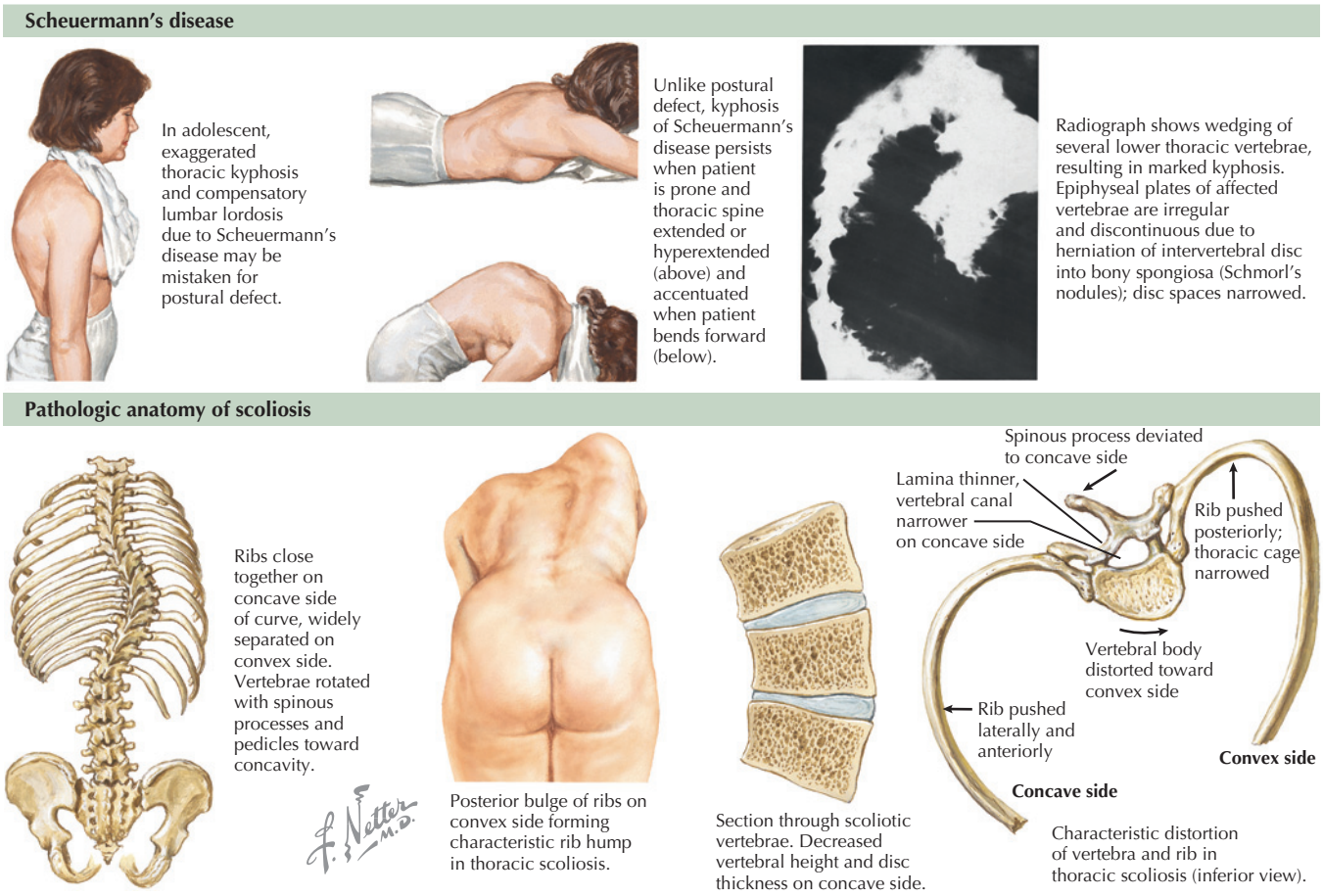


Figure 47-10 Spinal Deformities.

Treatment: Varies from observation to bracing to surgery, depending on age and type of curve. If the curvature is less than 20 degrees, then no active treatment is required but reassessment should be every 4 to 6 months until skeletally mature. If the curve is 25 degrees or more or if progressing, then bracing may be indicated. Severe curvature may require surgery.

Prognosis and return to play: Most patients are allowed to participate in sports. If surgery is required then participation in sport may be limited and decisions regarding sport should be made with the surgeon. Athletes need to understand that the fusion type of surgery required will limit flexibility and can alter performance. Fusion surgeries place additional stress on the adjacent spinal segments and can produce pathology at those levels.

Inflammatory Causes of Pain

Ankylosing Spondylitis

Description: An inflammatory spondyloarthropathy.

Mechanism of injury: Unknown cause but may be associated with the rheumatoid family of autoimmune diseases.

Presentation: Can present with spine or sacroiliac symptoms, usually at 15 to 35 years; insidious onset morning stiffness, pain, insertional tendonitis (enthesopathy); 20% have peripheral joint disease. Eyes, lungs, and heart may be involved.

Physical exam: May reveal limited chest expansion (<2 inches). Spinal flexibility may be limited as measured by Schober's test.

Differential diagnosis: Other forms of inflammatory arthropathy, degenerative disc disease, SI joint disease.

Diagnostics: HLA-B27 testing may be helpful. Radiographs of sacroiliac joints may show erosions, blurring of margins, sclerosis, and narrowing. Spine films may show straightening of the lumbar spine, squaring of vertebral bodies, and with progression of the disease syndesmophytes will bridge adjacent vertebrae, leading to the appearance of a "bamboo" spine.

Treatment: Medications helpful.

Prognosis and return to play: Allowed to participate but may be limited by pain.

Facet Syndrome

Description: Pain syndrome emanating from an irritated facet joint.

Mechanism of injury: This synovial fluid lined joint can become inflamed with injury and have associated hyaline cartilage damage. Synovitis may occur. Facet trophism (asymmetry) may lead to injury.

Presentation: Painful with extension and bending to the involved side.

Physical exam: Pain reproduced with above maneuvers. Neurologically intact.

Differential diagnosis: Stress fracture, fracture, discogenic disease, SI joint pain.

Diagnostics: Plain films may show joint changes. Bone scan may reveal increased activity in the joint and a CT scan may show arthritic changes.

Treatment: NSAIDs may be helpful. A facet injection may be diagnostic and therapeutic.

Prognosis and return to play: Participation limited by pain.

Sacroiliac Dysfunction

Description: Pain syndrome emanating from an irritated SI joint; commonly confused with low back pain. These joints are very

strong and allow limited motion while constrained with strong anterior and posterior ligaments; possess a synovial membrane.
Mechanism of injury: Injury, contracture, or inflammation of the SI joint.
Presentation: Patients may present with a traumatic history or they may be involved in jumping sports that require repetitive single-leg landing. Pain is usually over the sacroiliac joint but may also be referred into the leg, suggesting radiculopathy.
Physical exam: Local tenderness, a positive supine-to-long sitting test, a painful extension test, a painful FABER test, and pain when stresses are applied to the sacroiliac joint.
Differential diagnosis: Discogenic pain, stress fracture, facet joint pain, muscle injury.
Diagnostics: Radiographs normal unless late in course of severe spondyloarthropathy.
Treatment: Mobilization exercises, NSAIDs, modalities, and occasionally injection of the sacroiliac joint with corticosteroid.
Prognosis and return to play: Limited only by pain.

Infections

Description: Adult disc space infections, discitis, and vertebral body osteomyelitis may cause back pain, but are very rare in an athletic population.
Mechanism of injury: Infection, usually by hematogenous spread from distant focus.
Presentation: Insidious with progressively worsening back pain, may be febrile.
Physical exam: Rigid spine, pain with motion.
Differential diagnosis: Neoplasia, fracture.
Diagnostics: Laboratory studies may reveal elevated white blood cell count and sedimentation rate. Blood cultures may be positive. X-rays negative early but later may show disc space narrowing and endplate irregularities. Bone scan will be positive and an MRI will be valuable to differentiate tumor or infection.
Treatment: Specific to infection.
Prognosis and return to play: During acute phase will not be able to participate. Late participation will depend on treatment and outcome.

SUMMARY

Return-to-Play Decisions

- Each condition has its own criteria and may allow return to play at some but not all levels.
- Consultation with a spine specialist is recommended to help make the decisions and to discuss the risks and benefits of participation with the patient and family.
- The following findings may prohibit participation in contact sports:
  - Intersegmental instability on flexion-extension radiographs (e.g., instability that could potentially lead to neurologic impairment).
  - Spinal cord impingement with myelopathy.
  - Significant neurologic impairment or risk of impairment with a herniated disc or obstructing lesion.
  - Limiting pain.
  - A previous spinal fusion.
- After treatment, some athletes can be placed into the following risk categories, and allowed to return to play with the understanding that there is a level of risk involved.
  - Minimal risk: The increase in risk is small compared with play before the injury.
  - Moderate risk: There is a reasonable chance that symptoms will recur, and the patient is at some risk for permanent injury.
  - Extreme risk: The risk that symptoms will recur and cause permanent damage is high.

RECOMMENDED READINGS

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Pelvis, Hip, and Thigh Injuries

J. W. Thomas Byrd and Colin G. Looney

GENERAL PRINCIPLES

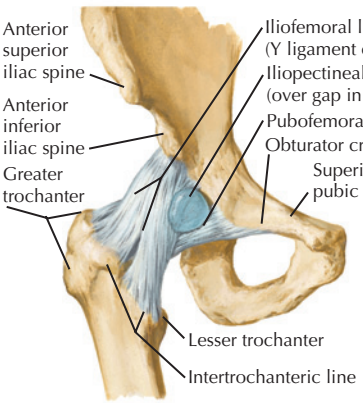
Overview

The last decade has brought a pronounced increased awareness and understanding of disorders around the hip and pelvis. More accurate diagnoses have led to more specific treatment strategies. Proper management can allow athletes to successfully recover and resume their activities.

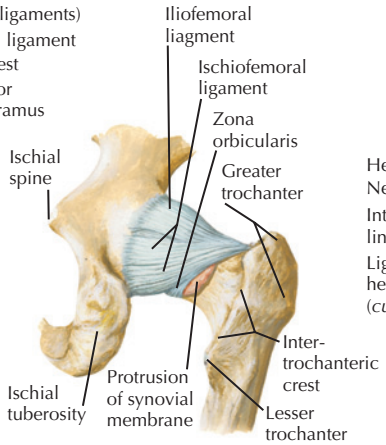
Anatomy

The constrained bony architecture of the hip provides it with greater stability, but less mobility, than the shoulder (Fig. 48-1).

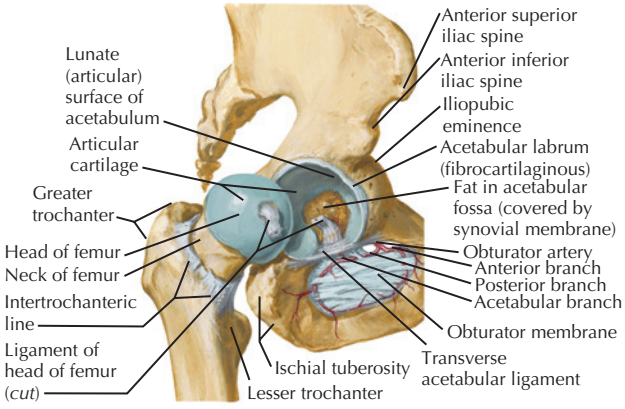
- Acetabulum
  - The articular surface of the acetabulum is encompassed by the fibrocartilaginous labrum, which is contiguous with the transverse acetabular ligament bridging the fossa inferiorly.
  - Unlike the shoulder, the labrum in the hip is of lesser importance to joint stability.
  - The abduction angle of the acetabulum relative to the horizontal plane averages 35 degrees with 20 degrees of forward flexion.
- Femoral head
  - The neck shaft angle averages 130 degrees and the femoral neck is anteverted 14 degrees relative to the bicondylar axis at the knee.



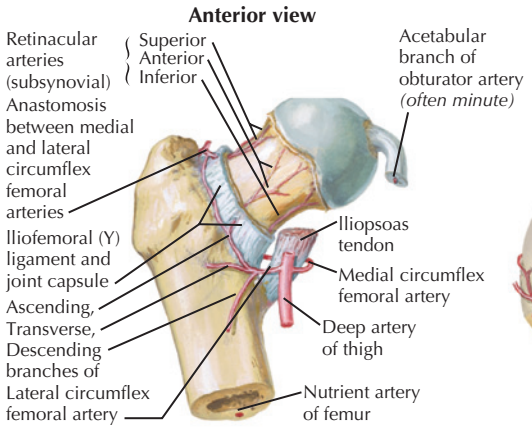
Hip joint; Anterior view. The iliofemoral ligament (ligament of Bigelow) has the shape of an inverted Y as it spirals from its attachment on the iliac portion of the superior acetabulum to its femoral attachment on the anterior neck. It is quite powerful and becomes taut in extension. The relatively weak pubofemoral ligament reinforces the inferior and anterior capsule, where it blends with the medial edge of the iliofemoral ligament.

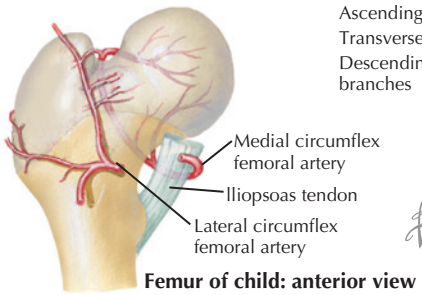


Posterior view: The ischiofemoral ligament reinforces the posterior capsule, spiraling from its attachment on the ischial portion of the posterior acetabulum to the superolateral aspect of the femoral neck.

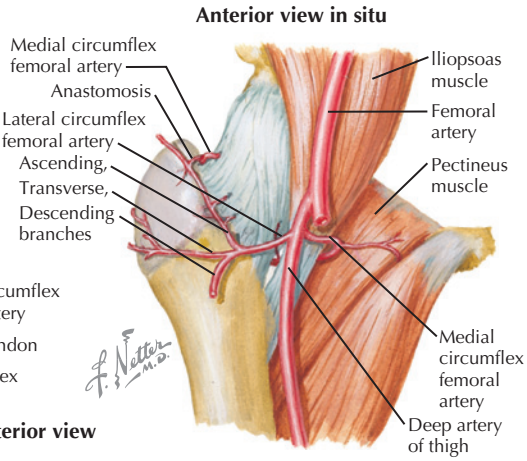


Joint opened; lateral view. Formed from portions of the ilium, ischium, and pubis, the lunate-shaped articular surface of the acetabulum surrounds the fossa containing the acetabular attachment of the ligamentum teres and fat, both encased in synovium. The labrum effectively deepens the socket and is contiguous with the transverse acetabular ligament inferiorly. The articular surface of the femoral head forms approximately two-thirds of a sphere. Medially, the ligamentum teres attaches at the fovea capitis. The diameter of the femoral neck is only 65% of the diameter of the femoral head, which allows for freer range of motion without marginal impingement.





Femur of child: anterior view



Arteries of the femoral neck. The femoral head receives arterial blood flow from an anastomosis of three sets of arteries: **(1)** The retinacular vessels, primarily from the medial circumflex femoral artery and, to a lesser extent, the lateral circumflex femoral artery; **(2)** terminal branches of the medullary artery from the shaft of the femur; **(3)** the artery of the ligamentum teres from the posterior division of the obturator artery.

Figure 48-1 Anatomy of the Pelvis, Hip, and Thigh.

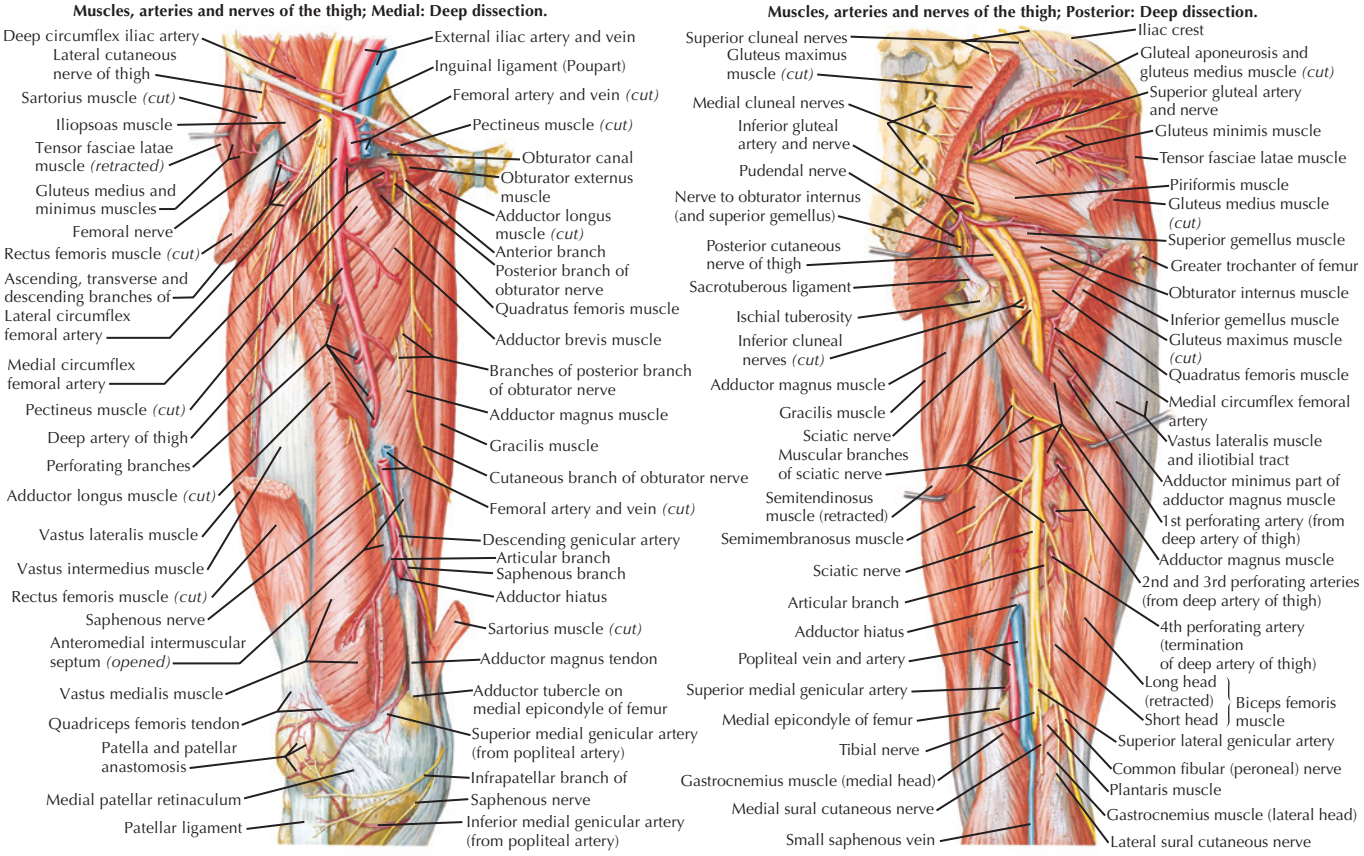


Figure 48-1, Cont'd Anatomy of the Pelvis, Hip, and Thigh.

- Capsule
  - The hip capsule is comprised of the stout iliofemoral ligament anteriorly, the ischiofemoral ligament posteriorly, and the relatively weak pubofemoral ligament inferiorly (see Fig. 48-1).
- Muscles
  - The action of individual muscles may change depending on joint position. The region is simplified by viewing the muscle groups as a superficial and a deep layer.
  - The superficial layer consists of the tensor fasciae latae, sartorius, and gluteus maximus (see Fig. 48-1).
  - The gluteus medius is transitional between the superficial and deep layers.
  - The deep layer includes posterior, lateral, anterior, and medial groups (see Fig. 48-1).
- The lower extremity receives its innervation from the lumbosacral plexus, which forms the sciatic femoral and obturator nerves as well as various smaller branches.
- The hip receives innervation from the L2 to S1 nerve roots, but principally from L3. This explains the presence of medial thigh pain often accompanying hip pathology because symptoms may be referred to the L3 dermatome.
- The lateral femoral cutaneous nerve, providing sensation to the lateral thigh, exits the pelvis under the inguinal ligament, close to the anterior superior iliac spine.

History and Physical Examination

Onset of Symptoms

- A history of significant trauma is a more favorable indicator of a potentially correctable problem.
- Insidious or gradual onset symptoms reflect underlying degenerative disease or predisposition to pathology and can reflect a less favorable long-term outcome.
- Onset from a modest acute event, such as twisting, should still lead one to suspect underlying predisposition to injury (Box 48-1).

Inspection

- Examine stance and gait.
- Observe patient's posture, standing and seated, checking for splinting or protective maneuvers used to alleviate stresses on the hip.

BOX 48-1 *Characteristic Hip Symptoms*

Symptoms worse with activities
Twisting, such as turning changing directions
Seated position may be uncomfortable, especially with hip flexion
Rising from seated position often painful (catching)
Difficulty ascending and descending stairs
Symptoms with entering/exiting an automobile
Dyspareunia
Difficulty with shoes, socks, hose, etc.

- Look for any asymmetry, gross atrophy, spinal alignment, or pelvic obliquity that may be fixed or associated with a gross leg length discrepancy.

Measurements

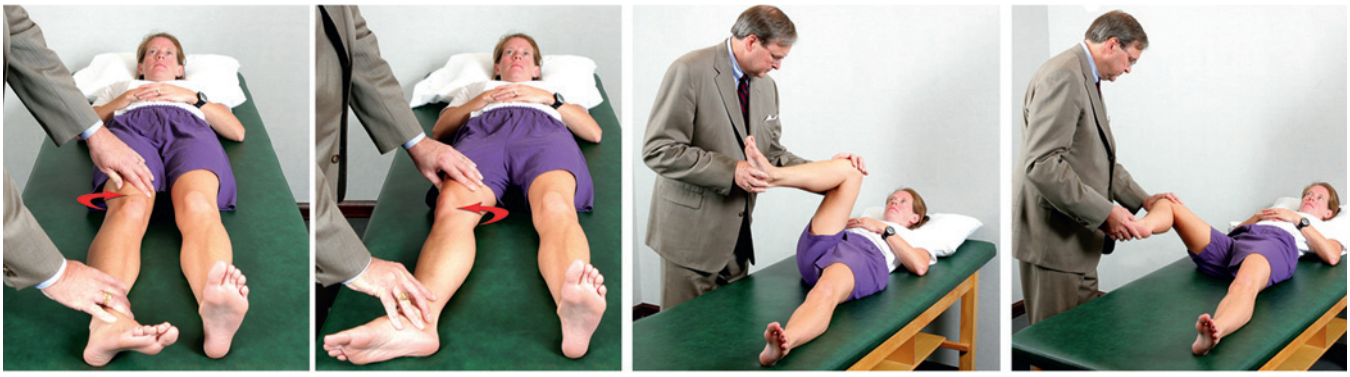
- Leg lengths are measured from the anterior superior iliac spine to the medial malleolus.
- Thigh circumference is an indicator of muscle atrophy associated with chronic conditions.
- Range of motion should be recorded in a consistent fashion, including rotational motion, flexion, extension, abduction and adduction.

Palpation

Ask the patient to point with one finger to the spot that hurts the worst. Palpation should begin away from this area to avoid exacerbating pain, which could compromise the exam. Palpation must be systematic, including the lumbar spine, sacroiliac (SI) joints, ischium, iliac crest, lateral aspect of the greater trochanter and trochanteric bursa, muscle bellies, and pubic symphysis.

Special Tests

- Log roll test is the most specific test for hip joint pathology.
- Forced flexion, adduction, and internal rotation is a more sensitive maneuver to detect hip joint irritability (Fig. 48-2); also referred to as the impingement test.
- Forced abduction with external rotation may similarly elicit hip joint symptoms (see Fig. 48-2).



A. Log roll test: Single most specific test for hip pathology. With the patient supine, gently rolling the thigh internally **(a)** and externally **(b)** moves the articular surface of the femoral head in relation to the acetabulum, but does stress any of the surrounding extraarticular structures.

B. Forced flexion, adduction, and internal rotation: Force flexion combined with internal rotation is often very uncomfortable and will usually elicit symptoms associated with even subtle degrees of hip pathology.

C. Forced abduction with external rotation: Flexion combined with abduction and external rotation similarly is often uncomfortable and may reproduce catching type sensations associated with labral or chondral lesions.

D. Patrick (or Faber) test: With the patient supine, the Patrick or Faber test is performed by crossing the ankle over the front of the contralateral knee and then forcing the knee of the involved extremity down on the table. This combination of flexion, abduction, and external rotation stresses the SI joint and when injury or inflammation is present, it markedly enhances symptoms localized to the SI area. This same maneuver can irritate the hip joint as well, but with distinctly different localization of symptoms.

E. Ober testing: a. The patient is in the lateral decubitus position with the affected side up. The knee is lowered towards the table assessing for tightness of the iliotibial band. **b.** The patient stands on the affected right leg, lifting the left leg off of the ground. With normal abductor strength, the pelvis should remain level. However, as illustrated here, with abductor weakness, the pelvis drops towards the contralateral side, reflecting a positive Trendelenburg test. *(Reprinted with permission J. W. Thomas Byrd, M.D.)*

Figure 48-2 Special Tests. *(Photographs reprinted with permission from J. W. Thomas Byrd, M.D.)*

- Patrick, or FABER (flexion, abduction, external rotation), test may provoke symptoms from either the hip or the SI joint.
- Straight leg raise test is used to assess tension signs associated with lumbar nerve root irritation. An active straight leg raise, or leg raise against resistance, may elicit hip symptoms because of the loading across the joint.
- Ober testing is used to assess tightness of the iliotibial band and the Trendelenburg test is useful for gross functional deficits of the abductor mechanism (see Fig. 48-2).

SPECIFIC INJURIES

Contusions

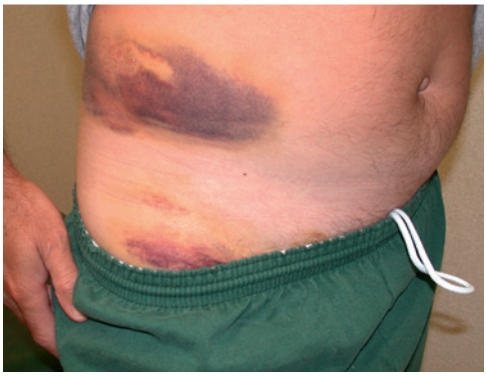
Contusions represent the most common injury of this region. The degree of injury is variable and most contusions resolve with minimal intervention. The key to treatment is proper recognition because some circumstances can have lasting consequences if neglected or mismanaged.

Iliac Crest Contusion

Description: This is referred to as a "hip pointer" in football (Fig. 48-3).
Mechanism of injury: Direct blow incurred from a fall or collision; occasionally, periostitis or exostosis may develop.
Presentation: History of trauma with acute onset of pain.
Physical exam: Pain, swelling, and ecchymoses will be evident along the iliac crest (see Fig. 48-3).
Differential diagnosis: Iliac crest fracture or abdominal wall injury.
Diagnostics: An anteroposterior (AP) radiograph is usually all that is necessary to rule out a fracture.
Treatment: Reduce swelling and pain and then gently implement range of motion followed by strengthening. Occasional judicious use of local corticosteroid injection has sometimes been advocated.
Prognosis and return to play: Generally excellent; return to play dictated by sufficient resolution of pain.

Quadriceps Contusion

Description: Trauma to the quadriceps; sometimes referred to as a "thigh bruise."
Mechanism of injury: Direct blow to the quadriceps region.
Presentation: History of trauma with acute onset of pain.
Physical exam: Pain and localized tenderness are evident. Significant swelling may occur but can be obscured by the volume of the thigh compartment. Superficial ecchymoses may be less evident or appear several days later. Stretching with passive knee flexion is painful, especially when combined with hip extension.
Differential diagnosis: Quadriceps rupture or strain.



Characteristic pattern of ecchymosis associated with iliac crest contusion.

Figure 48-3 Iliac Crest Contusion. *(Photographs reprinted with permission from J. W. Thomas Byrd, M.D.)*

Diagnostics: Although not always necessary, magnetic resonance imaging (MRI) may help to quantitate the size and severity of the contusion.
Treatment: Modalities to reduce inflammation, swelling, and pain with gentle range of motion followed by strengthening. If necessary, crutches may be used to normalize gait. Resting the extremity with the knee in flexion maintains tension on the quadriceps, which reduces pooling of blood and lessens the likelihood of contracture and scar. During the acute phase, it is important to monitor for the rare but serious complication of thigh compartment syndrome. Gentle range of motion is emphasized but aggressive passive stretching is avoided because this can potentiate the development of myositis ossificans.
Prognosis and return to play: Full recovery is generally excellent. If myositis ossificans develops, this can usually be managed conservatively. Occasionally surgical excision may be considered, but should be delayed many months until the lesion has fully matured to minimize recurrence.

Muscle Strains

Strains usually occur at the myotendinous junction. An exception to this is a previously contused muscle. With incomplete healing, the injury may occur at the site of previous muscle contusion. Most strains occur from a violent eccentric force while the muscle is attempting to contract. Muscles that cross two joints often contract eccentrically and have higher percentages of type II (fast twitch) muscle fibers and are more susceptible to this injury.

Hamstring Strain

Description: As a hip extensor and knee flexor, injury to the hamstring can occur anywhere along its course in the posterior thigh, including complete avulsion of its tendinous origin from the ischium.
Mechanism of injury: Occurs during sprinting with hip flexion and knee extension; complete avulsion of the tendinous origin usually involves a more violent force.
Presentation: Sudden severe stabbing pain; feeling a pop usually indicates a more severe injury.
Physical examination: Because of the variable location of the musculotendinous junctions, the site of involvement can be anywhere along the posterior thigh. A palpable defect indicates a more severe injury. Resisted contraction of the hamstring group will demonstrate diminished tone and pain.
Differential diagnosis: Sciatica or an injury to the popliteal region may create posterior thigh symptoms.
Diagnostics: MRI is helpful at grading the severity of the injury and may have prognostic value in estimating the length of recovery.
Treatment: Early management focuses on reducing pain and swelling with compression, ice, elevation, and rest while maintaining normal muscle length. With subsidence of acute symptoms, gentle flexibility followed by conditioning is implemented. Strengthening begins with isometric exercises and then progresses to isotonic and isokinetic methods as symptoms allow. For select cases, judicious use of corticosteroid injection into the injured area has occasionally been proposed as a method for facilitating recovery. For complete avulsions of the tendinous origin from the ischium, early surgical repair may provide more favorable outcomes. Surgical repair has resulted in significant improvement among chronic cases with residual dysfunction.
Prognosis and return to play: Recovery is favorable; return to sport is mostly dictated by the athlete's functional performance. General guidelines include full pain-free range of motion and 90% strength. In general, reinjuries tend to be more severe and recovery longer. Therefore, good judgment is necessary in determining when the athlete should return to play. Reflective of the highly variable nature of these injuries, mild strains may result in minimal lost playing time whereas more severe injuries may take months to recover.

Adductor Strain

Description: "Pulled groin muscle"; adductor injuries are especially common in ice hockey and soccer. The adductor longus is the most frequently injured (see Fig. 48-1). Adductor involvement may be a component of athletic pubalgia (see "Athletic Pubalgia").
Mechanism of injury: Forceful resisted abduction resulting in eccentric failure of the adductors is most common.
Presentation: Acute injury with pulling sensation or a pop.
Physical exam: Swelling, pain, and tenderness are present in the medial thigh. Resisted adduction is painful with diminished strength. Location of the injury is variable and can be determined by palpation. A defect may be present depending on the severity of the strain.
Differential diagnosis: Athletic pubalgia, osteitis pubis, hip flexor injury, hernia, thrombosis, fracture.
Diagnostics: Radiographs rule out a fracture or bony avulsion. MRI is useful to confirm and quantitate the injury.
Treatment: Most respond to a conservative rehabilitation program regardless of the severity of injury. Rarely is surgical repair beneficial.
Prognosis and return to play: Prognosis for a full recovery is excellent but the time frame is variable; most recover within 3 to 6 weeks.

Rectus Femoris Strain

Description: As a two-joint muscle, the rectus femoris is the most injury-prone of the quadriceps group. Acute injuries tend to occur distally in the thigh; chronic injuries occur more often near its origin at the hip. Avulsion of the anterior inferior iliac spine may occur in adolescents.
Mechanism of injury: Eccentric loading of the hip flexors and knee extensors.
Presentation: Typically precipitated by an acute event but often without a specific pop.
Physical exam: Pain is generated with either resisted hip flexion or knee extension. Careful palpation will elicit the location of the injury; a palpable defect is indicative of more severe involvement.
Differential diagnosis: Proximally, hip joint pathology or iliopsoas injury; distally, quadriceps tendon rupture.
Diagnostics: Imaging may not be necessary although MRI will aid in defining the injury.
Treatment: Most respond to a standard conservative protocol.
Prognosis: Prognosis for full recovery is generally excellent although variable depending on the severity and location of the injury. Mild strains may recover in 2 weeks whereas more severe injuries may take 2 months.

Iliopsoas Strain

Description: Not a common injury; can be a source of lingering, slowly resolving dysfunction.
Mechanism of injury: Forceful hip extension against a contracting iliopsoas muscle.
Presentation: Symptoms develop following an acute injury although they may not be incapacitating.
Physical exam: Pain occurs with resisted hip flexion and tenderness to palpation is present along the course of the iliopsoas anterior to the hip.
Differential diagnosis: Hip joint pathology, injury to the origin of the rectus femoris, adductor injury, occult hernia, athletic pubalgia, stress fracture, osteitis pubis.
Diagnostics: For recalcitrant cases, MRI may be helpful to detect inflammation and injury within the iliopsoas structure.
Treatment: Standard conservative protocol. For recalcitrant cases, judicious use of cortisone injection within the iliopsoas bursa bathing the tendon may be appropriate.
Prognosis and return to play: Prognosis for a full recovery is excellent, but can be protracted, sometimes taking 2 to 3 months for recalcitrant cases.

Bursitis

There are 13 consistent bursae and numerous other variable ones around the hip region (Fig. 48-4). Bursitis may coexist with tendinitis, tendinosis, and other friction syndromes and be difficult to differentiate.

Trochanteric Bursitis

Description: Commonly seen in association with friction of the overlying iliotibial band.
Mechanism of injury: Classically described in runners training on banked surfaces and more common in females, which is attributed to the wide pelvis and prominence of the trochanter.
Presentation: Laterally based hip pain usually develops from overuse although occasionally there may be a history of acute trauma.
Physical exam: Pain is elicited with palpation of the bursa directly over the lateral aspect of the greater trochanter. Ober testing is performed to check for associated tightness of the iliotibial band.
Differential diagnosis: Iliotibial band friction syndrome, abductor tendinopathy, stress fracture, L2 to L3 radiculopathy.
Diagnostics: Radiographs may be helpful to assess for bony abnormalities or soft tissue calcification. Further workup is not necessary except for recalcitrant cases to rule out other causes of symptoms.
Treatment: Oral anti-inflammatory medications, modification of training program, local modalities, and emphasis on stretching/flexibility of the iliotibial band. Judicious use of corticosteroid injections into the bursa can have therapeutic and diagnostic value. Surgical excision of the trochanteric bursa is rarely indicated with variable results. Careful scrutiny for other causes must be given for cases that fail conservative treatment.
Prognosis and return to play: Symptoms are rarely disabling but can be lingering. Sports participation to tolerance is acceptable, but may necessitate continued training modifications.

Iliopsoas Bursitis

Description: A cause of anterior hip pain, the iliopsoas bursa is the largest in the body and usually accompanies inflammation of the iliopsoas tendon.
Mechanism of injury: Typically occurs in conjunction with mechanical irritation of the iliopsoas tendon.

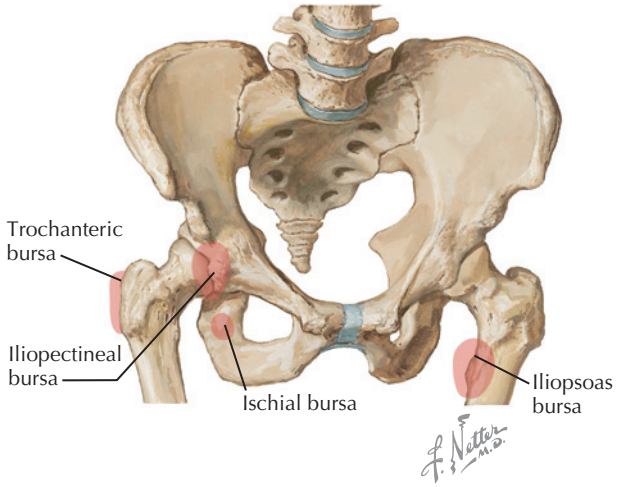


Figure 48-4 Location of Major Pelvic Bursae.

Presentation: Anterior hip and groin pain.
Physical exam: Localized tenderness to palpation is usually present. Pain with resisted hip flexion is usually present, but variable. Absence of significant discomfort with passive hip flexion with internal rotation distinguishes this from an irritable hip joint.
Differential diagnosis: Hip flexor strain, intra-articular pathology, occult hernia, athletic pubalgia.
Diagnostics: Radiographs are unrevealing, but important to assess the bony structures. MRI may be necessary to distinguish excessive fluid within the bursa from intra-articular pathology.
Treatment: A standard conservative protocol is usually effective. For recalcitrant cases, judicious use of a corticosteroid injection may have therapeutic and diagnostic value.
Prognosis and return to play: Prognosis for recovery from iliopsoas bursitis is excellent. However, in the presence of bursal swelling, other contributing factors should be considered, such as involvement of the iliopsoas tendon, that can alter the prognosis and return to play.

Ischial Bursitis

Description: Located adjacent to the origin of the hamstrings from the ischium, symptoms can be difficult to differentiate.
Mechanism of injury: Direct trauma or prolonged sitting.
Presentation: The athlete will localize symptoms to this area.
Physical exam: Tenderness to palpation directly over the ischium is present and is distinguished from hamstring involvement by absence of pain with stretching.
Differential diagnosis: Ischial stress fracture, sciatica, hamstring enthesopathy, inferior cluneal nerve entrapment.
Diagnostics: Radiographs may be helpful to rule out a bony lesion of the ischium. Further studies are rarely necessary except to rule out other causes in recalcitrant cases.
Treatment: Nonsteroidal anti-inflammatory medications, avoidance of offending activities, and other standard conservative modalities are usually effective. Judicious use of corticosteroid injections may be appropriate for recalcitrant cases.
Prognosis and return to play: Symptoms may linger, but athletic participation to tolerance is appropriate. More extensive investigation for other sources may be necessary in cases that fail to recover.

Nerve Entrapment

Any nerve arising from the lumbosacral plexus can be susceptible to entrapment around the hip (Fig. 48-5). Often no motor dysfunction is present and the areas of sensory innervation overlap, making the symptoms confusing and the diagnosis elusive. Treatment is sometimes controversial.

Lateral Femoral Cutaneous Nerve

Description: Referred to as "meralgia paresthetica," this is the most easily recognized of the nerve conditions around the hip.
Mechanism of injury: As the nerve exits underneath the anterior superior iliac spine, it is susceptible to compression from tight belts or pads, or prolonged periods of hip flexion (see Fig. 48-5).
Presentation: Athlete presents with pain or dysesthesias in the distribution of the lateral femoral cutaneous nerve along the lateral thigh.
Physical exam: Tenderness or Tinel's sign may be present as the nerve becomes superficial, distal to the anterior superior iliac spine. Dysesthesias, or diminished sensation, may be evident in the distribution of the nerve.
Differential diagnosis: Lumbar nerve root irritation.
Treatment: Elimination of the offending activity may be helpful. The value of localized injection is uncertain but may be considered for recalcitrant cases. Surgical release is rarely necessary.
Prognosis and return to play: Although painful, this usually does not prevent full activities.

Obturator Nerve

Description: Reported as a cause of medial thigh pain in athletes.
Mechanism of injury: Attributed to a fascial band compressing the nerve as it exits the obturator canal.
Presentation: Dysesthesias in the medial thigh distribution of the nerve are worsened with exercise.
Physical exam: Symptoms will be described in the distribution of the obturator nerve in absence of any other structural findings around the hip and groin. Adductor weakness may be present.
Differential diagnosis: Lumbar nerve root irritation, hip or groin pathology.
Diagnostics: Radiographs are normal. MRI may demonstrate atrophy of the adductor longus, brevis, and gracilis. Electromyogram (EMG) studies reflect chronic denervation of the adductor longus and brevis. Radionuclide scanning may demonstrate increased activity in the region of the pubic ramus at the origin of the adductors.
Treatment: Surgical release at the obturator foramen has been successfully reported for properly selected cases, but should be undertaken cautiously.

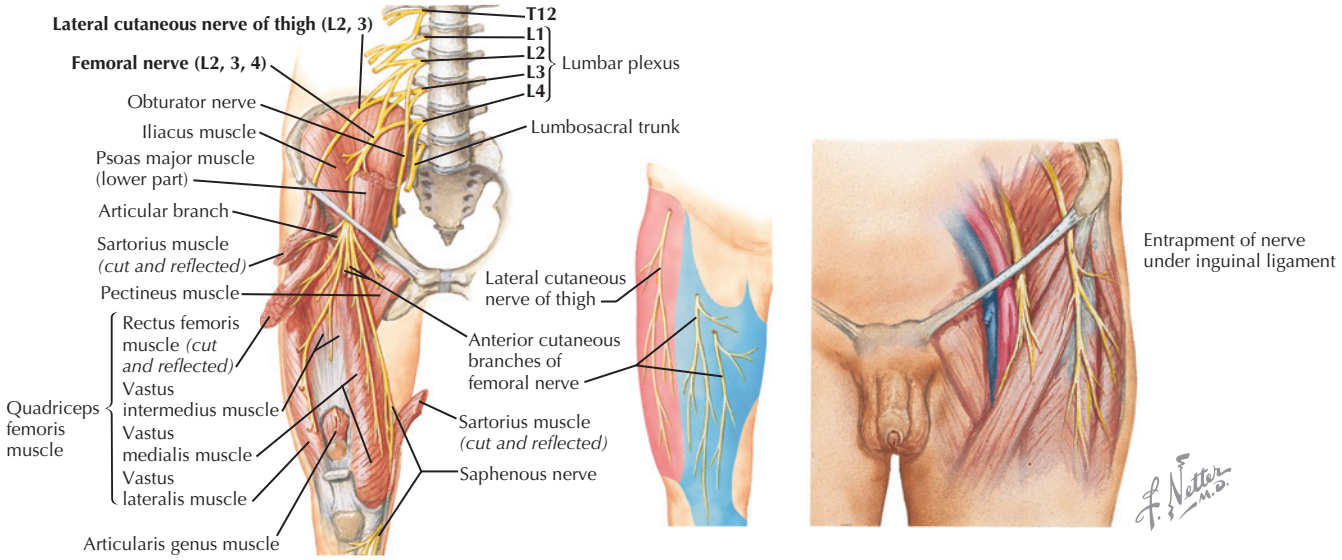


Figure 48-5 Nerve Entrapment.

Prognosis and return to play: Participation to tolerance is appropriate and successful return has been reported following surgical release.

Pudendal Nerve

Description: Common in cyclists; reported as a complication of hip arthroscopy.
Mechanism of injury: Caused by direct compression of the pudendal nerve.
Presentation: Sensory loss in the perineum and impotence in severe cases.
Physical exam: Diminished sensation is present in the distribution of the pudendal nerve.
Differential diagnosis: Lesion of the sacrum or sacral plexus.
Treatment: Avoidance of offending activity and observation.
Prognosis and return to play: Although not a problem with sports participation, the consequences of pudendal nerve neuropraxia are concerning. Most experience is that this will resolve spontaneously, usually within a few weeks, but it can take months. Permanent dysfunction, while rare, has serious personal implications.

Sciatic Nerve

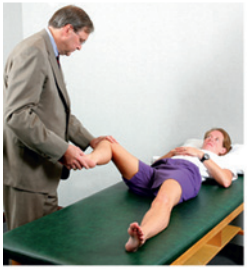
Entrapment may occur anywhere along its course, from the abdomen to the knee. Compression may occur from the piriformis muscle (discussed later) or, less commonly, attributed to the fibrous edge of the biceps femoris origin at the level of the ischial tuberosity.

Syndromes

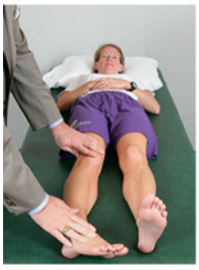
Several conditions with consistent constellations of symptoms and pathomechanics warrant distinction as syndromes.

Snapping Iliopsoas Tendon

Description: Referred to as "coxa saltans interna," this is often an incidental finding present in approximately 10% of an active population.
Mechanism of injury: The snapping occurs as the tendon flips across the anterior femoral head and capsule and the pectineal eminence; lying lateral when the hip is flexed, abducted, and externally rotated, and moving medial when the hip is extended with internal rotation.
Presentation: Following an injury or repetitive activity, an athlete may present with painful snapping, which is often audible but sometimes more of a sensation that they experience.
Physical exam: The characteristic test is bringing the hip from a flexed, abducted, externally rotated position into extension with internal rotation, eliciting the snap (Fig. 48-6). The snap consistently occurs when bringing the hip from a flexed to extended position.
Differential diagnosis: Intra-articular pathology or snapping of the iliotibial band.
Diagnostics: MRI may be helpful to assess for associated inflammation and to evaluate for intra-articular pathology. Iliopsoas bursography with fluoroscopic examination is specific but only about 80% sensitive in providing visual confirmation of the snapping phenomenon. Concomitant injection of Marcaine adds diagnostic value although it is not completely specific; and accompanying use of cortisone may have some therapeutic value. Ultrasonography has sensitivity similar to bursography with the advantage of being noninvasive, but lacks therapeutic value.
Treatment: If the snapping is not painful, assurance to the athlete that the snapping is not harmful is often all that is needed. Stretching and stabilization exercises may be beneficial. For recalcitrant cases, surgical release of the tendinous portion of the iliopsoas can be effective at eliminating the painful snapping.
Prognosis and return to play: Snapping does not contraindicate full sports participation. For surgical cases, return to sports can be anticipated at 3 to 4 months.



A. Examination maneuver for snapping of the iliopsoas tendon. a. The hip is initially placed in abduction, flexion and external rotation. **b.** The hip is then brought into extension with internal rotation, producing the snap of the ilopsoas tendon.



B. In the supine position, with the hip extended, the leg is internally rotated by the examiner, passively stretching the piriformis muscle.

C. Accentuation of symptoms with resisted abduction with the hip flexed is referred to as Pace's sign.

D. Passive piriformis stretching is a useful therapeutic maneuver but may also elicit symptoms. This includes hip flexion, adduction, and internal rotation.

Figure 48-6 Syndromes. *(Photographs reprinted with permission from J. W. Thomas Byrd, M.D.)*

Snapping Iliotibial Band

Description: Referred as to "coxa saltans externa," this may be present as an incidental finding without consequences.
Mechanism of injury: Snapping may occur from trauma, but often develops without injury. The snapping is created by the site of convergence of the tensor fascia lata and gluteus maximus flipping across the prominence of the greater trochanter.
Presentation: Sense that the hip is subluxing.
Physical exam: Snapping of the iliotibial band creates a visually evident phenomenon. With the athlete on his or her side, the snapping can sometimes be provoked as the hip is passively maneuvered between extension and flexion. The Ober test assists in checking for tightness of the iliotibial band.
Differential diagnosis: Hip instability, which is rare.

Diagnostics: Imaging studies are rarely beneficial in substantiating the diagnosis of iliotibial band, but may be useful to rule out other causes.

Treatment: It is difficult to cure the snapping, but modification of offending training activities may be beneficial in diminishing the associated symptoms. Stabilization exercises and modalities to diminish inflammation and discomfort are helpful. Judicious use of corticosteroid injections may aid in pain control as a supplement to a formal supervised rehabilitation regimen. Recalcitrant cases can often be successfully treated with surgical relaxation of the iliotibial band.

Prognosis and return to play: Snapping of the iliotibial band does not preclude full unrestricted sports participation. Return following surgery can take 2 to 4 months depending on associated conditions.

Piriformis Syndrome

Description: Uncommon condition that likely gets overdiagnosed and overlooked in equal proportions. Piriformis muscle compresses the sciatic nerve as it exits the sciatic notch.

Mechanism of injury: Often, there is a history of trauma with a direct blow to the area of the piriformis. An anomalous relationship of the piriformis muscle with the sciatic nerve may be a factor, but is often present as a normal variant.

Presentation: May be a history of trauma, and sitting is typically uncomfortable, creating both buttock pain and sciatica symptoms.

Physical exam: Provocative tests to compress the piriformis against the sciatic nerve include passive internal rotation and resisted external rotation of the extended hip; resisted abduction of the flexed hip (Pace's sign); and stretching with flexion, adduction, and internal rotation (piriformis stretch) (see Fig. 48-6). Pain on posterior palpation is present but nonspecific because of the large overlying gluteal muscle. The most characteristic examination finding is the re-creation of symptoms on palpation of the piriformis from inside the pelvis with either rectal or vaginal examination.

Differential diagnosis: Lumbar nerve root disorder, biceps syndrome, tumor, vascular anomaly.

Diagnostics: Workup should always include the lumbar spine, which is the most likely source of sciatica symptoms. MRI of the pelvis is used to rule out a soft tissue mass effect at the level of the sciatic notch. Neurodiagnostic studies are important for recalcitrant cases.

Treatment: Nonsteroidal anti-inflammatory medications and even a brief course of oral prednisone may be appropriate. Conservative measures include stretching of the piriformis muscle to reduce its compressive effect on the sciatic nerve. Injection of the piriformis muscle may have therapeutic value. Temporary alleviation of symptoms has diagnostic significance, substantiating involvement of the muscle. Precise placement of the injection is important and can be facilitated with computed tomography (CT) guidance. Surgery is rarely necessary but may be appropriate for patients that experience only temporary relief from injections.

Prognosis and return to play: Sports participation is allowed to tolerance. For rare surgical cases, alleviation of sciatica pain may be prompt but restriction of activities is still necessary for 2 to 3 months.

Athletic Pubalgia

Description: Tendinopathy characterized by breakdown of the pelvic stabilizers at a confluence of the rectus abdominis insertion, adductor origin, and pelvic floor at the pubic ramus. Common in sports such as ice hockey and soccer. Acute injury is associated with extension of the trunk and concomitant abduction of the hip. There is a significant correlation with hip disease, especially femoroacetabular impingement. It is speculated that diminished rotational motion of the hip is compensated by increased pelvic motion, placing more stress on the pelvic stabilizers and resulting in soft tissue breakdown of these structures.

Presentation: Groin pain following an acute injury or a repetitive mechanism that eccentrically loads the trunk flexors and hip adductors.

Physical exam: Tenderness is localized around the pubic ramus and there is absence of a detectable inguinal hernia. Pain is variously elicited with resisted adduction and resisted sit-ups. With acute injuries, there is associated swelling.

Differential diagnosis: Hernia, stress fracture, osteitis pubis, hip flexor strain, intra-articular pathology, nerve entrapment.

Diagnostics: Radiographs rule out bony abnormalities. MRI may show evidence of tendinopathy but is often unrevealing.

Treatment: Nonsteroidal anti-inflammatory medication and modification of offending activities is important. Therapeutic modalities may be beneficial to diminish discomfort and inflammation. Gradual restoration of flexibility and core strengthening is integral to a functional recovery. Judicious use of corticosteroid injections may be helpful for recalcitrant cases. Surgical takedown and restoration of the involved tendinous structures is advocated for those that fail conservative treatment.

Prognosis and return to play: Prognosis is highly favorable, although some may experience recalcitrant incapacitating symptoms. If the symptoms are not severe, continued sports participation may be allowed. For severe cases, recovery can take months. Following surgery, the best results have been reported in high-level athletes with lesser success among recreational athletes.

Sources of Referred Hip or Groin Pain

Upper Lumbar Disc

Nerve root irritation associated with an upper lumbar disc is less common and more easily overlooked than lower discs. Sciatica is absent as pain radiates to the anterior groin. Straight leg raising is negative, but hip extension may exacerbate nerve root irritation.

Hernias

An inguinal hernia is characterized by tenderness, fullness, or a bulge localized to the inguinal canal. The less common femoral hernia extends underneath the inguinal ligament, occupying the space medial to the femoral vein within the femoral triangle. A tender mass can be palpated. An index of suspicion is necessary to avoid overlooking this entity.

Visceral Disorders

Disease, dysfunction, and neoplasm of gastrointestinal, genitourinary, and gynecologic systems are all capable of producing hip and groin type pain. An index of suspicion must be maintained when thorough assessment does not reveal an obvious source of pathology.

Stress Fractures

Description: Occur when the forces on the bone exceed its remodeling capacity. Proximal femoral lesions usually occur in the femoral neck; pelvic-sided lesions can occur in the sacrum or the rami. Stress fractures are twice as common among females and may be part of the female athlete triad of eating disorder, osteoporosis, and amenorrhea.

Mechanism of injury: Excessive forces applied to normal bone or normal forces applied to abnormal bone (insufficiency fractures).

Presentation: Onset usually occurs in association with increased intensity or some significant alteration in the athlete's training program. Symptoms are typically worsened with weight-bearing and impact-loading activities and relieved with cessation.

Physical exam: Examination findings may be minimal in absence of impact loading; but are usually indicative of joint

irritability and can be difficult to distinguish from intra-articular pathology.

Differential diagnosis: Intra-articular pathology, muscle strain, avascular necrosis (AVN), or neoplasm.

Diagnostics: Radiographs are important but may fail to detect a lesion in almost half of cases. Radionuclide scanning is highly sensitive and relatively inexpensive. MRI has comparable sensitivity with excellent specificity and allows the advantage of assessing other soft tissue structures in the hip region. A history of multiple stress fractures may require a more extensive workup and a proper dietary and menses history should be obtained from women.

Treatment: Location of the lesion is important in determining proper treatment.

- Pelvic stress fractures are managed symptomatically with modification of activities below the threshold of pain.
- Femoral neck stress fractures require a more diligent strategy.
  - Medial-sided lesions on the compressive surface are inherently stable. These can be treated with protected weight bearing until asymptomatic, but require close monitoring for complete resolution. Surgical fixation may occasionally be indicated for protracted conditions.
  - Lateral-sided stress fractures are on the tensile surface of the neck and are "at risk" fractures for displacement. These are most properly managed with surgical fixation.

Prognosis and return to play: With proper management, most will heal completely. Complete healing takes several months, although return to sports may be sooner for stable pelvic lesions.

Osteitis Pubis

Description: Breakdown and inflammation of the symphysis pubis.

Mechanism of injury: Micromotion occurs from repetitive trauma, especially in sports that involve kicking or repetitive hip abduction/adduction activities. Occasionally observed in conjunction with breakdown of the pelvic stabilizers encountered in athletic pubalgia. Symptomatic conditions can also occur during or after pregnancy or as a sequela of infection or urologic or gynecologic pathologies.

Presentation: Vague lower abdominal, adductor, or groin symptoms that fail to localize specifically to one side.

Physical exam: Examination is characterized by point tenderness directly over the symphysis pubis.

Differential diagnosis: Inflammation or infection independent of the repetitive microtrauma of sport should be considered. Athletic pubalgia.

Diagnostics: Radiographs are important and, with chronic conditions, will usually demonstrate alterations in the symphysis pubis with variable combinations of sclerosis and lucency. However, these findings may be seen even among asymptomatic individuals. Radionuclide scanning will demonstrate increased activity in this region and MRI will show edema.

Treatment: Management is symptomatic including use of oral anti-inflammatory medication and efforts to modify the athlete's activities to diminish associated pain. Pelvic stabilization exercises can be helpful as tolerated. Corticosteroid injection is appropriate for recalcitrant cases. Various surgical procedures have been described, ranging from simple debridement to fusion, depending on the degree of instability and dysfunction. However, these are salvage procedures with limited clinical experience and reserved only for the most desperate circumstances.

Prognosis and return to play: Recovery is generally expected with conservative treatment but can take months for resolution. Sports participation is allowed as long as symptoms are tolerable. There is little evidence to support return to sports following surgical intervention because this is generally reserved for conservative failures even with inactivity.

Dislocation/Subluxation

Description: Dislocation of the hip requires substantial force. The resultant injury is incapacitating to the athlete and evident on initial assessment. Subluxation of the hip and concomitant damage may have a more subtle presentation.

Mechanism of injury: The hip is inherently stable to posterior translation yet most injuries occur in this direction because of the mechanism of injury. With the hip flexed, the leg is axially loaded, driving the femoral head posterior. Among collision sports, this commonly occurs in a pile-up of players and, in motor sports, is the classic dashboard injury. Subluxation can occur as a noncontact injury, especially with sudden deceleration as the leg is planted to stop while the hip is flexed and the knee extended. Commonly with dislocation and subluxation, there may be an associated fracture of the posterior lip of the acetabulum that does not compromise the stability of the joint.

Presentation: With a dislocation, the incapacitating nature of the injury will be evident. With subluxation or fracture-subluxation, the athlete will recount a specific injury but the amount of disability can be variable. Some athletes will be unable to bear weight whereas others may continue to participate until it becomes evident that the accompanying symptoms do not subside.

Physical exam: With a posterior dislocation, the leg will be in adduction and internal rotation and appear foreshortened. With the less common anterior dislocation, the leg will be externally rotated. Examination findings associated with a subluxation will be those characteristic of a painful hip joint. Posterior symptoms may be present because of injury to the short external rotators and posterior musculature.

Differential diagnosis: A dislocation may be difficult to distinguish from an intertrochanteric, femoral neck or complex acetabular fracture. Symptoms from subluxation will share common features with other types of intra-articular pathology or groin injuries. The presence of posterior pain may suggest an SI joint or gluteal injury.

Diagnostics: Radiographs are important to assess for concentric reduction and associated bony injury. An MRI will help to reveal the associated soft tissue injury around the joint as well as intra-articular pathology. If a bony injury is suspected, a CT scan provides better assessment of the bony architecture and fracture than that provided by MRI.

Treatment: Prompt reduction of a dislocated hip is important to lessen the likelihood of developing avascular necrosis. Whether to attempt reduction on the field without benefit of radiographs is controversial. If the clinician is experienced in the assessment and treatment of hip dislocations, then it is generally accepted that a one-time gentle attempt at reduction is appropriate. If reduction is not easily accomplished, repeated attempts are not recommended. With a documented subluxation episode, the athlete should be kept on a protected weight-bearing status until a thorough evaluation has been completed. For dislocations and subluxations, careful follow-up is important to assess for the presence of associated intra-articular pathology or the subsequent development of avascular necrosis.

Prognosis and return to play: Outcome variable, dictated by the severity of damage incurred at the moment of injury. The uncertain outcome necessitates a conservative, thoughtful approach in its management. With successful resolution of symptoms and absence of intra-articular damage or AVN, return to play can occur as early as 2 to 3 months.

Avulsion Fractures

Description: Characteristically occur in adolescent males at a period during which the growth plates remain open while muscle power is markedly increased in conjunction with the appearance of an-

drogens. Sites of involvement in order of decreasing frequency include the anterior superior iliac spine, ischium, lesser trochanter, anterior inferior iliac spine, iliac crest, and greater trochanter.

Mechanism of injury: As with strains, the injury is usually the result of a sudden ballistic maneuver with accompanying eccentric loading of the tendinous insertion site to bone. Prior to physeal closure, this is the weakest site for injury to occur.

Presentation: An acute episode in association with sprinting or other sudden acceleration activities.

Physical exam: Tenderness will be localized to the site of involvement with pain on resisted contraction of the involved muscle group.

Differential diagnosis: Muscle strain or contusion.

Diagnostics: Radiographs typically demonstrate the avulsed fragment. For subtle findings with minimal displacement, MRI can be helpful at substantiating the injury but is usually not necessary. The amount of initial displacement rarely widens over time, although follow-up radiographs may be prudent.

Treatment: Most are treated nonoperatively. Crutches may be necessary to develop a painless gait. Gentle range of motion and conditioning exercises are implemented as symptoms allow. Surgery is occasionally proposed for large fragments with significant displacement but is rarely necessary.

Prognosis and return to play: The prognosis for return to unrestricted activities is excellent. Recovery can take 6 to 10 weeks, depending on the location, severity of injury, and age of the athlete.

Apophysitis

Description: Disorder of the skeletally immature athlete; may occur anywhere within the hip girdle, but the most common site is the iliac crest.

Mechanism of injury: Overuse disorder of the skeletally immature athlete.

Presentation: Onset of symptoms may be acute or associated with a period of intense activity.

Physical exam: Pain and tenderness to palpation on the iliac crest.

Differential diagnosis: Hip pointer, contusion, or apophyseal avulsion.

Diagnostics: With chronic involvement, radiographs may demonstrate slight asymmetric physeal widening on the side of involvement.

Treatment: Symptomatic, modifying offending activities while the discomfort subsides.

Prognosis and return to play: Excellent, and return to play is allowed as symptoms tolerate but may necessitate a period of modification.

Osteonecrosis

Description: Avascular necrosis of the femoral head with diminished blood supply, resulting in osteocyte death. Because this is a disorder encountered in young adults, an index of suspicion must be maintained because it can coincidentally be present in athletes.

Mechanism of injury: Although often idiopathic, there is a causal relationship with trauma, such as dislocation or subluxation. Other systemic factors include alcohol abuse, catabolic steroids, and decompression sickness, among others.

Presentation: Symptoms usually occur insidiously without a specific precipitating event.

Physical exam: Clinical findings of hip joint irritability will be present.

Differential diagnosis: Chondral or osteochondral injury, transient regional osteoporosis and osteoarthritis.

Diagnostics: Radiographs may vary from normal to advanced collapse depending on the stage of the disease. MRI is the study of choice for diagnosis and accurate staging. Acute chondral and osteochondral injuries may demonstrate significant marrow and subchondral signal changes that can mimic AVN.

Treatment: Variable and sometimes controversial depending on the stage of the disease. Goal is to prevent progression, which may sometimes remain stable for years. Surgical options range from palliative procedures, such as core decompression, to more aggressive procedures to revascularize the bone, such as free vascularized fibular grafting. Arthroscopy has a limited role. It can help to stage the disease by assessing the integrity of the femoral articular surface and address coexistent intra-articular pathology.

Prognosis and return to play: Prognosis is guarded. Successful results in preserving the joint have been reported, but this is rarely suited for return to competitive sports.

Tumors

Description: 10% to 15% of primary musculoskeletal tumors arise within the pelvis and hip. Among older adults, metastatic disease is the most likely neoplasm.

Presentation: Neoplasms around the hip are uncommon but must be considered in cases of unexplained pain or circumstances in which the episode of trauma may seem trivial or coincidental.

Physical exam: Because of the deeply situated anatomy of this area, lesions may gain considerable size before being noticed by the patient or becoming discernable on examination.

Diagnostics, Treatment, and Prognosis: Variable based on the nature of the neoplasm.

Intra-articular Disorders

Description: Labral tears, chondral lesions, and rupture of the ligamentum teres are the three most common lesions encountered among athletes (Fig. 48-7).

Mechanism of injury: Violent blow or twisting injury (see Fig. 48-7). Training errors, pushing the joint beyond its physiologic limits, may result in breakdown in absence of major trauma. Joint morphology such as femoroacetabular impingement or dysplasia may play a significant role in many cases. Intrinsic tissue disease may also result in failure at physiologic forces.

Presentation: Onset is variable but a history of significant trauma is a more favorable indicator of a correctable problem; mechanical symptoms such as sharp, stabbing pain, catching, or locking are more favorable findings.

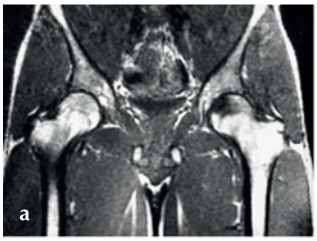
Physical exam: Groin pain or a C-sign may be present. Log roll test is the most specific test for an intra-articular disorder. Forced flexion with internal rotation and abduction with external rotation are more sensitive maneuvers to elicit symptoms of an irritable joint (see Fig. 48-2).

Differential diagnosis: Stress fracture, AVN, athletic pubalgia, snapping hip syndrome, nerve entrapment.

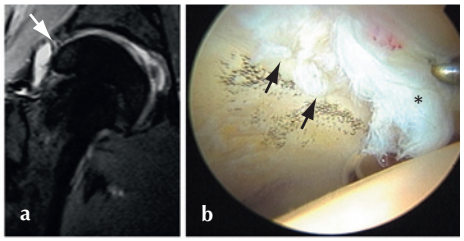
Diagnostics: Radiographs are important to assess for bony or joint changes as well as evaluate the hip morphology. High-resolution MRI is improving at detecting intra-articular pathology. Gadolinium arthrography with MRI is often more sensitive. Injection of a long-acting anesthetic along with the contrast is an important diagnostic maneuver to distinguish the amount of associated pain relief.

Treatment: A period of rest or observation may be appropriate, with close monitoring of symptoms. Persistent symptoms may warrant arthroscopic intervention. Neglect may lead to further joint damage but will be indicated by worsening pain.

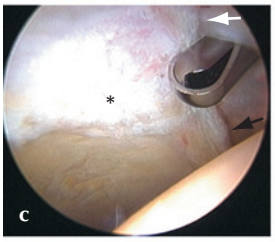
- Labral lesions can be selectively debrided (see Fig. 48-7). Indiscriminate resection is avoided because this can result in poor outcomes.
- Labral repair can be performed for appropriately selected tears (see Fig. 48-7).
- Chondral lesions are addressed with chondroplasty or microfracture for appropriate grade IV lesions (see Fig. 48-7).



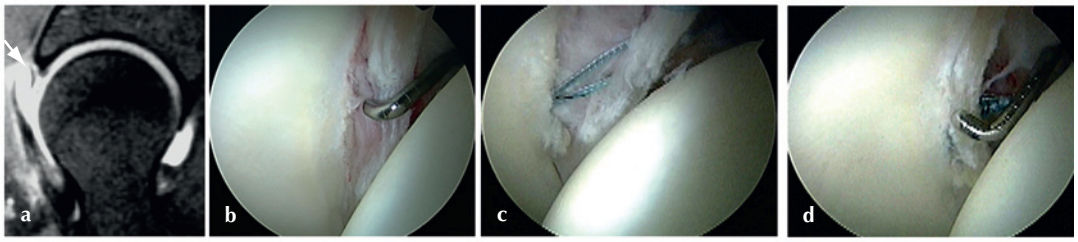
A. Fall resulting in direct blow to the greater trochanter. a. MRI of 20-year-old male collegiate basketball player with painful catching of the left hip following a fall with lateral impaction of the joint reveals extensive signal changes in the medial aspect of the femoral head characterizing the subchondral injury associated with his fall. **b.** A full-thickness chondral flap lesion (*) associated with the injury is identified. **c.** The unstable portion has been excised.



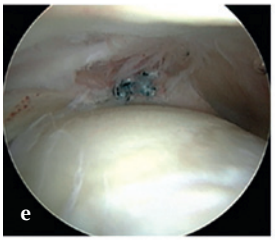
B. A 25-year-old top ranked professional tennis player sustained a twisting injury to his right hip. a. Coronal MRI demonstrates evidence of labral pathology **(arrow). b.** Arthroscopy reveals extensive tearing of the anterior labrum **(asterisk)** as well as an adjoining area of Grade III articular fragmentation **(arrows)**.



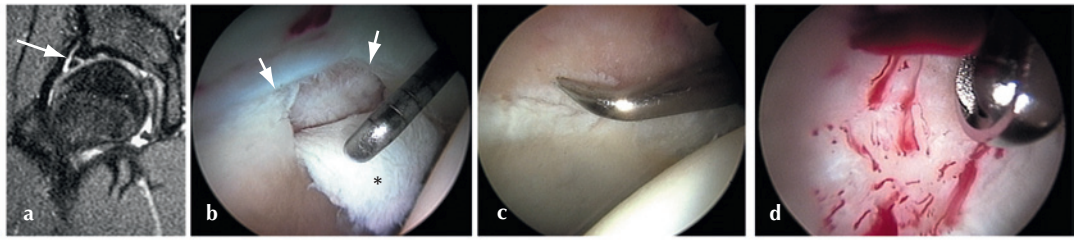
Bc. The labral tear has been resected to a stable rim **(arrow)** and chondroplasty of the Grade III articular damage **(asterisk)** is being performed.



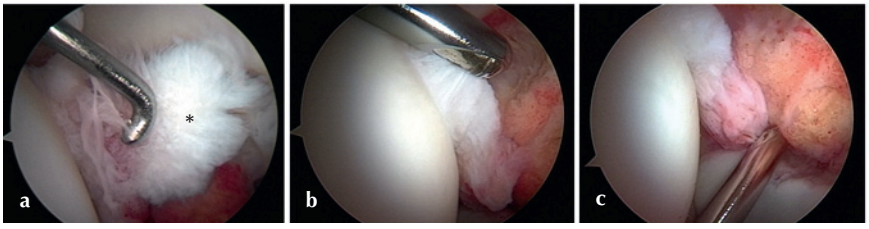
C. A 37-year-old female with recalcitrant. a. Sagittal MRA image demonstrates an anterior labral tear **(arrow)**. **b.** Arthroscopy reveals a traumatic detachment of the anterior labrum **(probe)**. **c.** An anchor has been placed with suture limbs passed in a mattress fashion through the detached labrum. **d.** The labrum has been reapproximated to the articular edge.



Ce. Viewing the peripheral aspect of the labrum demonstrates the suture on its capsular surface, avoiding contact with the articular surface of the femoral head.



D. A 23-year-old elite professional tennis player sustained an injury to his right hip. a. Coronal MRI demonstrates evidence of labral pathology **(arrow)**. **b.** Arthroscopy reveals the labral tear **(arrows)**, but also an area of adjoining Grade IV articular loss (*). **c.** Microfracture of the exposed subchondral bone is performed. **d.** Occluding the inflow of fluid confirms vascular access through the areas of perforation. The athlete was maintained on a protected weight-bearing status emphasizing range of motion for 10 weeks with return to competition at three and a half months.



E. A 16-year-old cheerleader has a 2-year history of catching and locking of the left hip following a twisting injury. a. Arthroscopic view from the anterolateral portal reveals disruption of the ligamentum teres (*). **b.** Debridement is begun with a synovial resector introduced from the anterior portal. **c.** The acetabular attachment of the ligamentum teres in the posterior aspect of the fossa is addressed from the posterolateral portal.

Figure 48-7 Intra-articular Disorders. *(Photographs reprinted with permission from J. W. Thomas Byrd, M.D.)*

- Disrupted fibers of the ligamentum teres can be selectively debrided (see Fig. 48-7).
- Careful evaluation must be performed to assess for associated etiologic factors such as impingement or dysplasia, which may need to be addressed.

Prognosis and return to play: Prognosis is variable.

- Simple isolated labral tears are uncommon but have a highly favorable outcome; return to play within 2 months.
- Labral tears usually have a variable amount of associated articular damage, which is a more limiting factor on the outcome with regard to duration and completeness of recovery.
- Microfracture may necessitate a protected weight-bearing status for 2 months before gradual resumption of functional activities.
- More complex procedures for impingement may obviate a 4- to 6-month recovery period.

Femoroacetabular Impingement

Description: Condition that leads to intra-articular pathology and subsequent early onset osteoarthritis. Cam type is caused by a nonspherical femoral head associated with subclinical slipped capital femoral epiphysis or premature physeal closure; most prevalent among young adult males. Pincer type is caused by overcoverage of the anterior acetabulum associated with numerous conditions including acetabular retroversion and protrusion; more commonly seen in mature females.

Mechanism of injury: Culmination of cyclical repetitive microtrauma during hip flexion as the joint is subjected to the abnor-

mal forces created by the altered morphology. With cam impingement, the bump on the anterolateral femoral head glides underneath the labrum, engaging the articular surface, resulting in delamination with a variable amount of associated labral pathology. With pincer impingement, the labrum becomes crushed against the neck of the femur, resulting in primary labral pathology and secondarily articular failure.

Presentation: Athlete may describe an acute injury, but close questioning will often elicit a prior history of less severe intermittent symptoms.

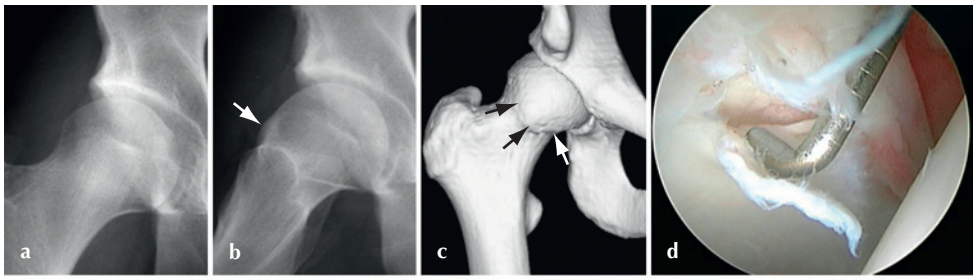
Physical exam: In addition to joint pain, diminished internal rotation is often present. Forced flexion, adduction and internal rotation is described as the impingement test.

Differential diagnosis: Impingement morphology may be a coincidental finding and other causes of hip pain must be considered.

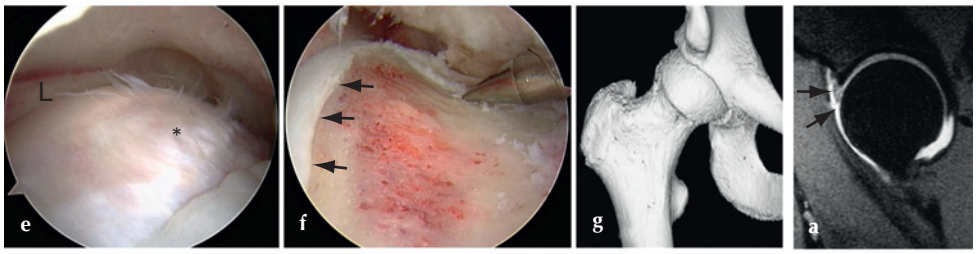
Diagnostics: Properly oriented radiographs are essential to interpreting the joint morphology. MRI and magnetic resonance arthrography (MRA) help to elicit the associated intra-articular pathology and aid in interpreting the joint morphology. CT with 3-D reconstruction is very helpful at clearly discerning the bony architecture of the joint.

Treatment: Activity modification and observation may be appropriate to monitor for resolution of the athlete's symptoms. Surgical intervention is proposed when symptoms persist in the presence of impingement and associated intra-articular pathology.

- An open technique with surgical dislocation of the hip can precisely correct the bony abnormalities with acetabular rim trimming and recontouring of the femoral head (femoroplasty) as dictated by the joint morphology. However, these extensive open procedures are not well suited for athletes returning to sports participation.
- Much of this can also be accomplished with arthroscopic methods (Fig. 48-8). Precision of the bony recontouring comes close to that which can be accomplished with the open method, although the morbidity of the arthroscopic approach is substantially less.

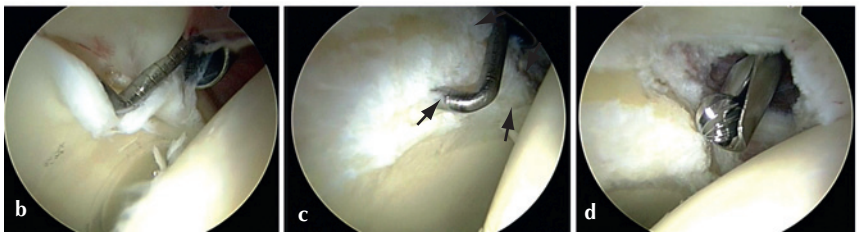


A. A 20-year-old hockey player with a four year history of right hip pain. a. AP radiograph is unremarkable. **b.** Frog lateral radiograph demonstrates a morphological variant with a bony build-up at the anterior femoral head/neck junction **(arrow)** characteristic of cam impingement. **c.** A 3D CT scan further defines the extent of the bony lesion **(arrows)**. **d.** Viewing from the anterolateral portal, the probe introduced anteriorly displaces an area of articular delamination from the anterolateral acetabulum characteristic of the peel back phenomenon created by the bony lesion shearing the articular surface during hip flexion.



Ae. Viewing from the peripheral compartment, the bony lesion is identified **(*)** immediately below the free edge of the acetabular labrum **(L)**. **f.** The lesion has been excised, recreating the normal concave relationship of the femoral head/neck junction immediately adjacent to the articular surface **(arrows)**. Posteriorly, resection is limited to the mid-portion of the lateral neck to avoid compromising blood supply to the femoral head from the lateral retinacular vessels. **g.** Postoperative 3D CT scan illustrates the extent of bony resection.

B. A 16-year-old high school football player develops acute onset of right hip pain doing squats. a. Sagittal image MR arthrogram demonstrates a macerated anterior labrum **(arrows)**.



Bb. Viewing from the anterolateral portal, a macerated tear of the anterior labrum is probed along with articular delamination at its junction with the labrum. **c.** The damaged anterior labrum has been excised, revealing an overhanging lip of impinging bone from the anterior acetabulum. **d.** Excision of the impinging portion of the acetabulum (acetabuloplasty) is performed with a burr.

Figure 48-8 Femoroacetabular Impingement. *(Photographs reprinted with permission from J. W. Thomas Byrd, M.D.)*

Prognosis and return to play: Surgical intervention has a high success rate at diminishing symptoms. However, returning to the rigors of competitive sports is more variable and is dictated by the severity of damage at the time of surgery. Whether or not these procedures alter the natural history of the disorder remains to be seen.

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Knee Injuries

W. Michael Walsh, Eric C. McCarty, and Christopher C. Madden

PHYSICAL EXAMINATION

Anatomy of the Knee

See Figure 49-1.

Observation and Measurement

Standing

Alignment of lower extremities: View patient from front, side, and back.

Angular and rotational deformities: Excessive valgus, varus, recurvatum, flexion contracture, femoral or tibial torsion.

Foot alignment and mechanics: Excessive cavus or pes planus; heels should invert and arches increase on toe rising.

Leg length inequality: Best judged by pelvic levelness on standing.

Difference in size of legs: Atrophy of one limb versus hypertrophy of opposite limb.

Popliteal masses: May be seen better in prone position.

Sitting

Patellar position: With patient's knees flexed 90 degrees, look from side to judge high or low position. Anterior patellar surface normally faces wall in front of patient sitting with legs over side of exam table. View from front to judge lateral posture. Patella should appear centered in soft tissue outline of knee.

Osgood-Schlatter's changes: Enlarged and/or tender tibial tuberosity.

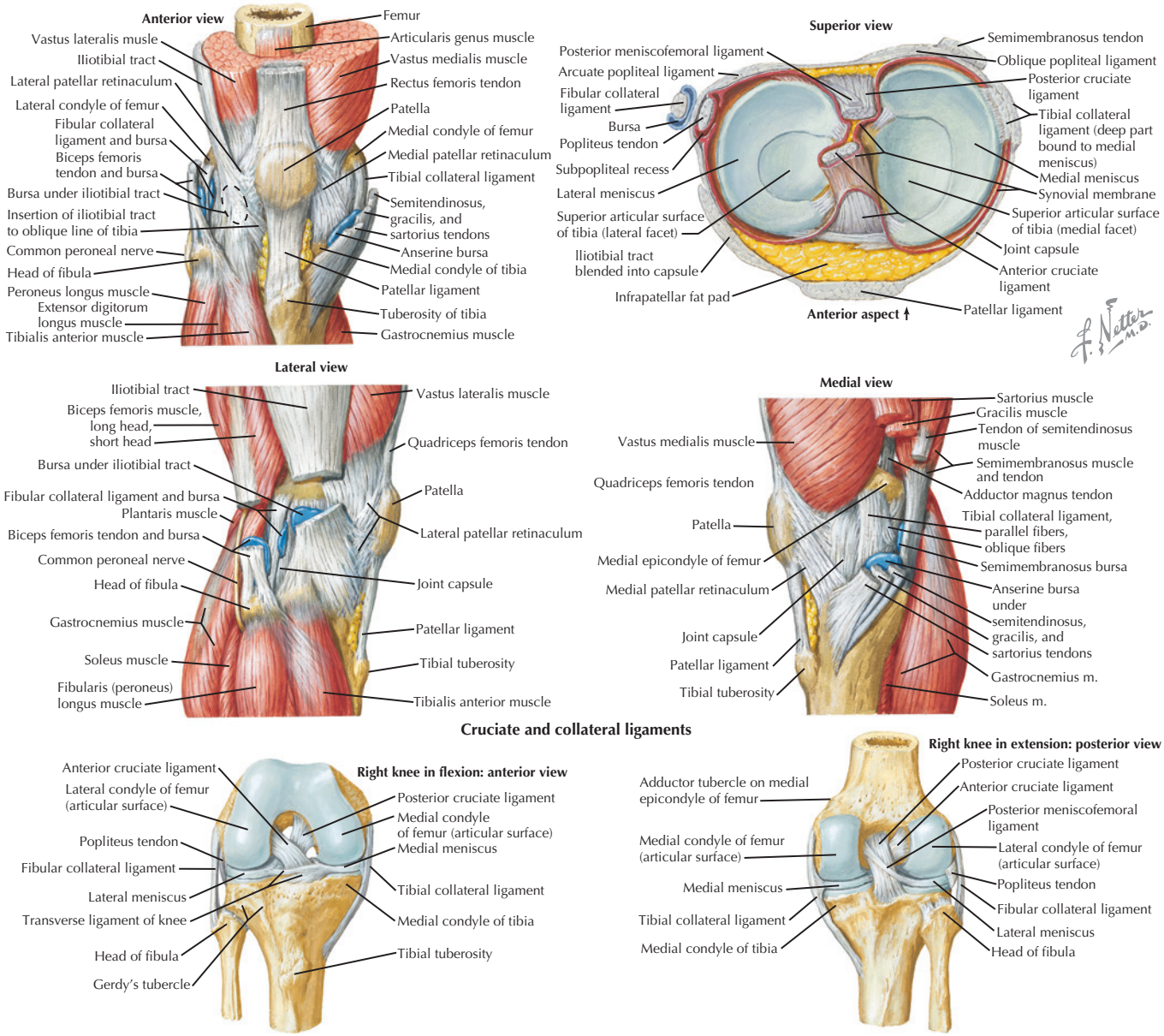


Figure 49-1 Anatomy of the Knee.

Vastus medialis obliquus/vastus lateralis (VMO/VL) relationship: With patient's knees held actively at 45 degrees of flexion (Fig. 49-2), distal one-third of vastus medialis normally should present as substantial muscle from adductor tubercle inserting into upper one-third to one-half of medial patella. Dysplastic VMO appears hollow in this normal muscular location (see Fig. 49-2). Also observe for apparent hypertrophy of VL.

Patellar tracking: Observe on active flexion and extension; watch for excessive displacement of patella.

Lying

SUPINE

Range of motion: Both active and passive; compare injured with uninjured side.

Muscle bulk: Thigh and calf; can measure circumferences.

Quadriceps (Q) angle: With quadriceps contracted, measure angle between line from anterior superior iliac spine to midpoint of patella and line from midpoint of patella to tibial tuberosity (see Fig. 49-2). Normal in males is 10 degrees or less; in females, 15 degrees or less.

Hamstring and heel cord tightness: See Chapter 36, Musculoskeletal Injuries in Sports.

PRONE

Range of motion: Lack of full knee flexion may show quadriceps tightness.

Popliteal masses: Compare contours with those of opposite knee.

Walking/Running

Mechanics of gait: Stance and swing phase from side to side is even; look for limp, other asymmetry, excessive limb rotation, limb malalignment.

Patellofemoral tracking: Observe patella closely from front view.

Palpation

Joint effusion: With patient's knee extended, milk fluid from suprapatellar pouch and palpate along medial and lateral sides of patella (see Fig. 49-2). Try to distinguish intra-articular effusion that can be moved about from extra-articular swelling that feels more like thick, soft tissues and is not movable.

Significant areas of tenderness:

- Menisci: medial and lateral joint lines.
- Ligament attachments: medial femoral epicondyle, adductor tubercle, lateral femoral epicondyle, proximal medial tibia.
- Tendons: patellar tendon, quadriceps tendon, popliteus tendon, hamstrings.
- Bursae: prepatellar, pes anserinus, tibial collateral ligament, deep infrapatellar.
- Other: patellar facets, extensor retinaculum.

Crepitation: During range of motion—from any rough joint surface (especially patellofemoral joint), fractures, or soft tissue thickness. **Patellofemoral compression—**longitudinal and/or transverse compression of patella against femur. Feel for crepitation or ask about elicited pain.

Muscle tone: Overall turgor of muscle tissue; may be decreased early after injury, even if bulk still measures normal.

Specific Tests

Perform all tests on uninjured knee first to establish "normal" for that patient (Table 49-1).

Ligaments

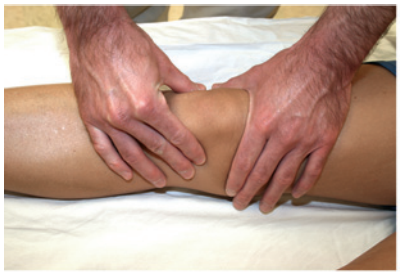
MEDIAL

Valgus stress test at 30 degrees and 0 degrees: Patient is supine and relaxed, thigh supported on table. Examiner applies valgus force at foot, while using other hand as fulcrum along lateral side of joint. Watch and feel for medial joint line opening. Perform first with knee flexed 30 degrees, then with maximum possible extension or hyperextension (Fig. 49-3).

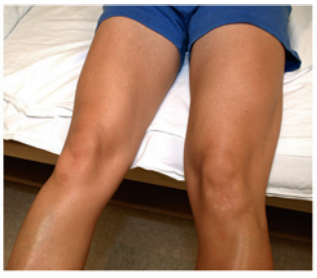
Anterior drawer test with external rotation of tibia: Patient is supine and relaxed, hip flexed to 45 degrees and knee to 90 degrees. Externally rotate foot 30 degrees, then pin foot to table with examiner's thigh. Grasp proximal tibia with both hands and pull toward examiner. Positive test is excessive anterior rotation of medial tibial condyle (see Fig. 49-3).

LATERAL

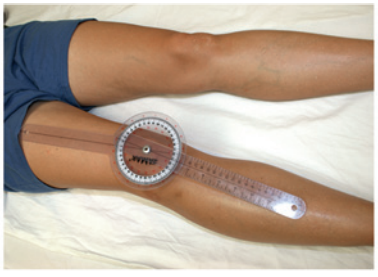
Varus stress test at 30 degrees and 0 degrees: Patient is in same position as for abduction stress test. Reverse hand position so that one hand applies varus stress, while opposite hand acts as fulcrum along medial side of joint. Watch and feel for lateral



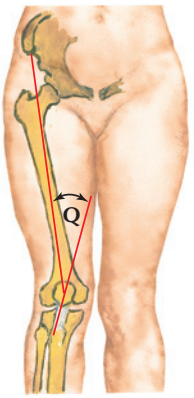
Effusion. Fluid is milked from suprapatellar pouch with one hand and with other and the fluid is palpated on the sides of the patella.



Vastus medialis obliquus (VMO). Patient with marked VMO dysplasia. This is probably the most important predisposition to all extensor mechanism syndromes.



Quadriceps (Q) angle measurement. With quadriceps contracted, proximal arm of goniometer is directed toward anterior superior spone, pivot point of goniometer is directed toward anterior superior spone, pivot point of goniometer is placed over the center of patella, and distal arm of goniometer is placed on tibial tuberosity. Normal in males, up to 10°; females, 15°.



Q angle formed by intersection of lines from anterior superior iliac spine and from tibial tuberosity through midpoint of patella. Large Q angle predisposes to patellar subluxation.

Figure 49-2 Observations and Measurement of Knee.

Table 49-1 KEY PHYSICAL EXAMINATION TESTS AND INJURED STRUCTURES

Test	Injured structure
Valgus stress test at 30 degrees and 0 degrees	30 degrees: Medial collateral ligament 0 degrees: Posteromedial corner, medial collateral ligament, posterior cruciate ligament, possibly anterior cruciate ligament
Varus stress test at 30 degrees and 0 degrees	30 degrees: Lateral collateral ligament 0 degrees: Posterolateral corner, lateral collateral ligament, posterior cruciate ligament
Lachman test	Anterior cruciate ligament
Anterior drawer test	Anterior cruciate ligament, but affected by other structures such as collaterals
Pivot shift test/jerk test	Anterior cruciate ligament
Posterior drawer test	Posterior cruciate ligament
Gravity or sag test	Posterior cruciate ligament
Posterolateral drawer test	Posterolateral corner structures
External rotation recurvatum test	Posterolateral corner structures
McMurray's test	Menisci
Apley's compression test	Menisci
Apprehension test	Medial patellofemoral ligament and retinaculum
Prone external rotation test (Dial test)	Posterolateral corner structures

joint line opening. Perform at 30 degrees of flexion and then at full possible extension or hyperextension (see Fig. 49-3).

External rotation recurvatum test: Patient is supine and relaxed. Lift entire lower extremity by first toe. Observe for excessive recurvatum and external rotation of proximal tibia (tibial tuberosity) and apparent varus deformity of knee. Indicates posterolateral corner injury.

Posterolateral drawer test: Same position as for anterior drawer test with external rotation of tibia. Examiner's hands push posteriorly on proximal tibia. Positive test is excessive posterior rotation of lateral tibial condyle (see Fig. 49-3).

Prone external rotation test (Dial test): The patient is prone with knees together and the feet are externally rotated at 30 degrees of knee flexion and then at 90 degrees. The external rotation of the foot relative to the thigh is compared with the contralateral side. Test is positive if there is more than 10 degrees of rotation of affected side compared to normal side. If asymmetry is present only at 30 degrees than isolated posterolateral corner injury is likely. If asymmetry is present at both 30 degrees and 90 degrees then a combined injury to posterior cruciate ligament (PCL) and posterolateral corner is present (see Fig. 49-3).

Reverse pivot shift test: Performed with tibia in external rotation rather than internal rotation. With knee flexed 90 degrees, lateral tibial condyle is subluxed posteriorly. With further knee extension, tibia reduces with detectable "clunk." (See later discussion of pivot shift test.)

ANTERIOR CRUCIATE LIGAMENT (ACL)

Lachman test: Patient is supine and relaxed. Examiner grasps distal femur with one hand, while other hand grasps proximal tibia. Knee flexed to approximately 15 to 20 degrees. Apply anterior force to proximal tibia. Positive test is excessive anterior translation of tibia beneath femur and lack of firm endpoint (see Fig. 49-3).

Anterior drawer test in neutral rotation: Same position as for anterior drawer with external rotation of tibia, except that foot and tibia are in neutral rotation. Anterior pull is applied to proximal tibia. Positive test is anterior translation of both tibial condyles from beneath femur (see Fig. 49-3). *Note:* This test is influenced by structures other than anterior cruciate ligament. Do not rely on this test for diagnosis of ACL tear.

Pivot shift test and jerk test: Patient is supine and relaxed. Begin with knee fully extended (pivot shift test) or flexed to 90 degrees (jerk test). Foot and tibia internally rotated. Valgus applied at knee. Knee progressively flexed (pivot shift test) or extended (jerk test). At approximately 30 degrees, watch and feel for anterior subluxation of lateral tibial condyle. Tibia suddenly reduces with further flexion (pivot shift test) or extension (jerk test) (see Fig. 49-3).

POSTERIOR CRUCIATE LIGAMENT (PCL)

Posterior drawer test: Same position as for anterior drawer test in neutral rotation. Posterior force is applied to proximal tibia. Positive test is straight posterior displacement of both tibial condyles (see Fig. 49-3). *Caution:* Make sure of neutral position as starting point. Compare position of tibia relative to femur with normal knee. It is easy to start from posteriorly displaced position and interpret reduction to neutral as positive anterior drawer sign rather than starting at neutral and interpreting as positive posterior drawer sign.

Gravity or sag test: Patient is supine and relaxed. Flex hips to 45 degrees and knees to 90 degrees with feet flat on table. With quadriceps relaxed, observe from lateral side for posterior displacement of one tibial tuberosity compared to the other. Then flex hips to 90 degrees, support both legs by ankles and feet, and observe again (see Fig. 49-3).

Valgus or varus stress test at 0 degrees: As described for abduction and adduction stress tests at 30 degrees and 0 degrees. Positive test in full extension in acute case is often due to posterior cruciate ligament rupture in addition to injury to associated collateral ligaments (see Fig. 49-3).

Menisci

Joint line tenderness: Tenderness along the medial or lateral joint lines is among the most sensitive findings for a meniscal tear (see Fig. 49-3).

McMurray's test: Patient is supine and relaxed. Have patient flex knee maximally with external tibial rotation (medial meniscus) or internal tibial rotation (lateral meniscus). While maintaining rotation, patient brings knee into full extension. Positive test is painful pop occurring over medial joint line (medial meniscus) or lateral joint line (lateral meniscus) (see Fig. 49-3).

Apley's compression test: Patient is in prone position. Knee is flexed to 90 degrees with external tibial rotation (medial meniscus) or internal tibial rotation (lateral meniscus). Apply axial compression to tibia while patient flexes and extends knee. Positive test is painful pop over medial joint line (medial meniscus) or lateral joint line (lateral meniscus) (see Fig. 49-3).

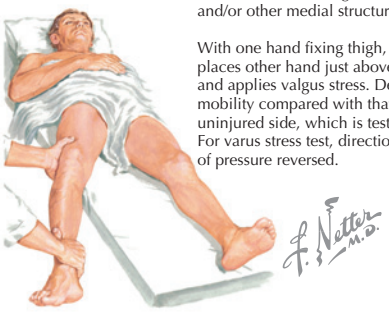
Patella

Hypermobility/apprehension test: Patient is supine and relaxed. Examiner sits on edge of table with patient's knee flexed approximately 30 to 45 degrees across examiner's thigh. With patient's quadriceps relaxed, examiner uses both thumbs to forcefully displace patella over lateral femoral condyle. Positive test is increased lateral mobility of patella compared to opposite knee or other patients. More important is discomfort or extreme apprehension that patella is going to dislocate because of lateral displacement.

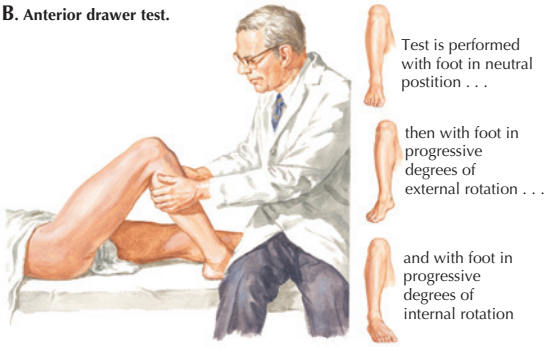
Plica tests: Patient is supine and relaxed. With tibia internally rotated, examiner passively flexes and extends knee from 30 to 100 degrees of flexion. Examining fingers placed along the medial patellofemoral joint may feel a click, possibly some tenderness, or even a pop of a pathologic plica.

A. Varus and valgus tests.
Patient supine on table, relaxed, leg over edge of table, flexed about 30° for medial ligament injury. When test is done at 0° and loose then there is typically an associated cruciale ligament injury and/or other medial structures.

With one hand fixing thigh, examiner places other hand just above ankle and applies valgus stress. Degree of mobility compared with that of uninjured side, which is tested first. For varus stress test, direction of pressure reversed.



B. Anterior drawer test.



Test is performed with foot in neutral postition . . .

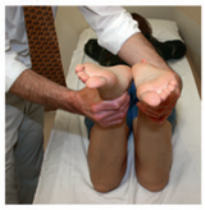
then with foot in progressive degrees of external rotation . . .

and with foot in progressive degrees of internal rotation

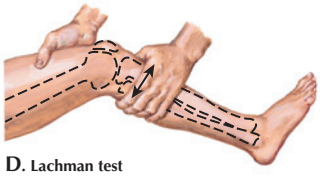
Patient supine on table, relaxed, with head on pillow; hip flexed 45°, knee flexed 90°; foot flat on table. Examiner sits partially on dorsum of patient's foot to stabilize it, places hands on each side of upper calf as shown. Anterior force applied to proximal tibia while index fingers ensure that hamstrings are relaxed. May also be done with external rotation and internal rotation of foot and tibia.

C. Prone external rotation test (dial test).

Dial test at 30°.



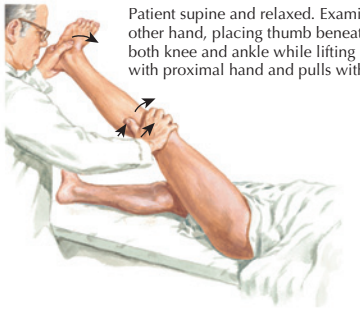
Dial test at 90°.



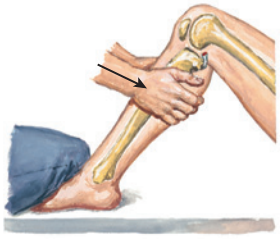
D. Lachman test
With patient's knee bent 20° to 30°, examiners hands grasp limb over distal femur and proximal tibia. Tibia alternately pulled forward and pushed backward. Movement of 5 mm or more than that in normal limb indicates rupture of anterior cruciate ligament.

E. Pivot shift test/Jerk test.

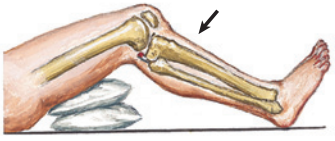
Patient supine and relaxed. Examiner lifts heel of foot to flex hip 45° keeping knee fully extended; grasps knee with other hand, placing thumb beneath head of fibula. Examiner applies strong internal rotation to tibia and fibula at both knee and ankle while lifting proximal fibula. Knee permitted to flex about 20°; examiner then pushes medially with proximal hand and pulls with distal hand to produce a valgus force at knee.



As internal rotation, valgus force, and forward displacement of lateral tibial condyle maintained, knee passively flexed. If anterior subluxation of tibia (anterolateral instability) present, sudden visible, audible, and palpable reduction occurs at about 20° to 40° flexion. Test positive if anterior cruciate ligament ruptured, especially if lateral capsular ligament also torn.

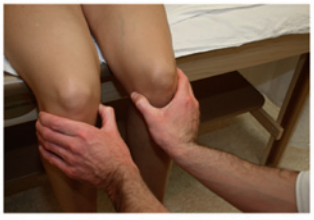


F. Posterior drawer test. Procedure same as for anterior drawer test, except that pressure on tibia is posterior instead of anterior.

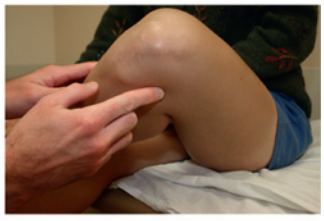


G. Sag test. Posterior cruciate ligament rupture. Left knee shows positive gravity test with sagar left tibia.

H. Joint line tenderness.



Both knees are palpated at the same time by thumbs. Tenderness of medial joint can be evaluated by comparing both sides.



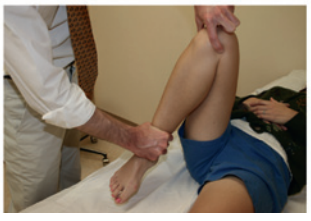
Lateral joint line is palpated with knee in figure-of-four position.

I. Apley's compression test.

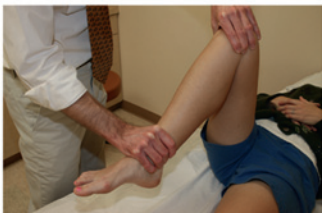
Position for lateral meniscus with pressure on sole of foot, tibia is externally rotated while knee is flexed and extended.

For medial meniscus, foot is internally rotated while knee is flexed and extended.

J. McMurray's test.



Position for lateral meniscus. With foot in internal rotation, knee is brought from full flexion to extension while fingers palpate lateral joint line.



Position for medial meniscus. With external tibial rotation, knee is brought from full extension while fingers palpate medial joint line.

Figure 49-3 Special Tests.

KNEE LIGAMENT INJURIES

Medial Ligaments

Description: Injury to medial (tibial) collateral ligament and/or medial capsular ligament (Fig. 49-4).

Mechanism of injury: Valgus force applied to knee with external tibial rotation; may be noncontact twist or a blow to lateral side of joint.

Presentation: Initial pain on medial side of knee; with complete tear, complaints of knee giving way into valgus.

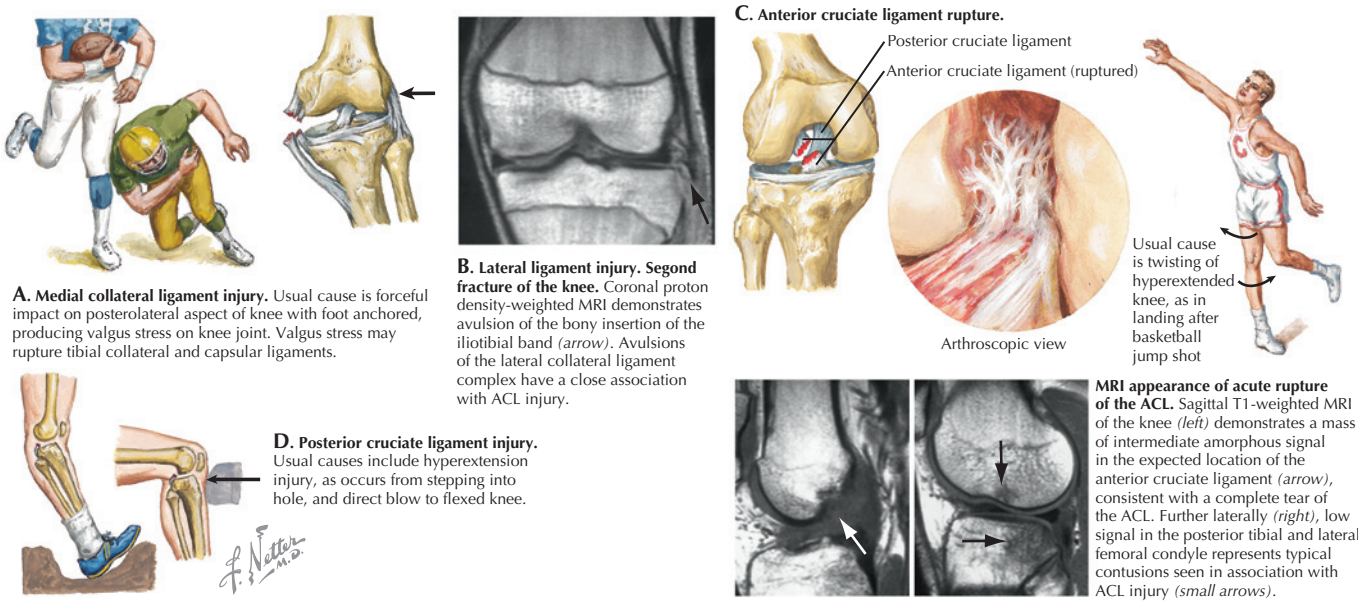


Figure 49-4 Knee Ligament Injuries. *(MRIs reprinted with permission from Adam A, Dixon A, Grainger R, Allison D: Grainger & Allison's Diagnostic Radiology, 5th ed. Philadelphia: Elsevier, 2008.)*

Examination: Positive valgus stress test at 30 degrees flexion. Compare with opposite knee. An injured medial collateral ligament (MCL) along with disrupted ACL or PCL will result in more gap occurring with a valgus stress test, particularly noticeable when knee is tested in extension. Frequently, but not always, positive anterior drawer sign results with tibia in external rotation. Medial tibial condyle rotates anteriorly.

Imaging: Abduction stress film may be used to distinguish ligament injury from epiphyseal fracture in skeletally immature athletes. Fracture opens at growth plate. Ligament tear opens at joint line. Do in 20 to 30 degrees of flexion.

Differential diagnosis: In young patients, epiphyseal fracture of distal femur or proximal tibia; patellar dislocation (may be associated with medial ligament tear); medial meniscus tear (may be associated with medial ligament tear).

Treatment:

- **Grades I and II sprains:** RICE (rest, ice, compression, elevation), crutches, rehabilitation.
- **Grade III sprain (complete ligament tear):** With other associated injuries, surgery may be considered (currently rare). If no surgery indicated, immobilization should be used for short period after acute injury. Begin rehabilitation program as soon as possible. With only mild instability, rigid immobilization may not be necessary. RICE and functional rehabilitation may be adequate treatment.

Lateral Ligaments

Description: Sprain or tear of lateral (fibular) collateral ligament and/or lateral capsular ligament; may be associated injuries to popliteus tendon, iliotibial band, popliteofemoral ligament, peroneal nerve.

Mechanism of injury: Varus or twisting injury; may be contact or noncontact. Posterolateral ligaments often injured by hyperextension mechanism, frequently with blow to anteromedial tibia.

Presentation: Pain is present over lateral ligament complex. Knee may give way on twisting, cutting, or pivoting. In chronic cases, posterolateral corner injury gives feeling of giving way into hyperextension when standing, walking, or running backward.

Examination: Compare with opposite knee. In acute case, may be increased varus stress test at 30 degrees of flexion and positive posterolateral drawer sign; chronic case shows positive reverse pivot shift test and external rotation recurvatum test. External rotation recurvatum may also be apparent on standing, giving increased varus appearance to knee.

Imaging: Lateral capsular sign shows avulsion of midportion of lateral capsular ligament with small fragment of proximal lateral tibia. Associated with high incidence of anterior cruciate tear and indicates anterolateral instability (see Fig. 49-4). Arcuate sign shows avulsion of proximal fibula with posterolateral ligament complex. Indicates posterolateral instability.

Differential diagnosis: Chronic posterolateral injury may be confused with medial compartment arthritis because of progressive varus appearance. Difficult to differentiate from posterior cruciate injury. Acute lateral ligament injury may be confused with lateral meniscus tear. Injury to middle third of lateral capsular ligament, as shown by lateral capsular sign on x-ray, usually associated with anterior cruciate ligament injury.

Treatment:

- **Grade I and II sprains:** RICE, crutches, rehabilitation.
- **Grade III sprain (complete ligament tear):** Surgical repair is usually preferable if injury involves more than just lateral (fibular) collateral ligament. Immobilization is not really useful by itself. Mild instability may be treated by RICE and functional rehabilitation.

Anterior Cruciate Ligament (ACL)

Description: Tear of part or all of two major bundles (posterolateral, anteromedial) of anterior cruciate ligament; may be associated with tears of middle one-third of lateral capsular ligament. ACL is torn from femur or tibia, or torn in its midportion; may avulse tibial spine in young patients (see Fig. 49-4).

Mechanism of injury: Hyperextension, varus/internal rotation, and extremes of valgus and external rotation are possible causes.

Presentation: Usually loud pop occurs; may be followed by autonomic symptoms of dizziness, sweating, faintness, slight nausea. Large swelling usually occurs within first 2 hours after acute injury (hemarthrosis). Conversely, most acute hemarthroses (>85%) are anterior cruciate tears. In chronic cases, complaints of giving way on twisting, pivoting, cutting.

Examination: Acute, large hemarthrosis, positive Lachman test. Chronic, positive Lachman test, positive pivot shift test or jerk test. Perhaps positive anterior drawer sign, but not reliable. Do not rely on anterior drawer sign.

Imaging: Lateral capsular sign; avulsion of tibial spine may be seen in young patients. Magnetic resonance imaging (MRI) useful in acute injury to confirm diagnosis and evaluate for injuries to other structures; reported accuracy rates as high as 95% in detecting ACL tears (see Fig. 49-4).

Differential diagnosis: Acute, differentiate from other causes of hemarthrosis (e.g., osteochondral fracture, peripheral meniscus tear, patellar dislocation); chronic, differentiate from other types of ligamentous laxity and/or meniscal tears.

Treatment:

Acute

- Various methods delineate degree of damage and associated injuries.
- Knee may be treated symptomatically followed by repeated evaluations over first 2 to 3 weeks following injury.
- Most active patients engaged in agility sports require surgical reconstruction.
- Reconstruction is now usually delayed at least 3 weeks after injury to allow decrease in swelling and increase in range of motion.
- For mild laxity with firm endpoint (partial ACL injury) and no other associated injury, may treat with PRICES, functional rehabilitation, protective bracing.
- Apparent partial injury often progresses to more obvious complete tear.

Chronic

- May attempt functional stabilization through rehabilitation, bracing, lifestyle modification; often requires surgical reconstruction.

Posterior Cruciate Ligament (PCL)

Description: Tear of part or all of two major bundles of the posterior cruciate ligament (posteromedial and anterolateral).

Mechanism of injury: Valgus/varus in full extension; in rare cases, severe twist; direct blow to anterior proximal tibia, as in fall on artificial turf or other hard playing surface.

Presentation: Usually less swelling than with anterior cruciate ligament; otherwise, in acute stage, nothing particularly distinguishing. Chronically, feeling of femur sliding anteriorly off tibia, especially when rapidly decelerating or descending slopes or stairs.

Examination: Acute, if produced by varus or valgus mechanism, may find abduction or adduction stress test positive in full extension. If produced by blow to anterior tibia, posterior drawer sign may be positive. Chronic, rely on posterior drawer sign and gravity test (see Fig. 49-4).

Imaging: Cross-table lateral view x-rays may show sag of tibia compared to opposite side; may accentuate by doing posterior drawer sign while taking cross-table lateral view. May see bony avulsion with tibial attachment of the posterior cruciate ligament. MRI shows posterior cruciate well and may help confirm diagnosis and evaluate for other injuries (see Fig. 49-4).

Differential diagnosis: Most difficult is distinguishing posterior cruciate injury from posterolateral corner injury. Posterior drawer sign and posterolateral drawer sign may appear the same. Both injuries may exist in same knee.

Treatment: Acute, most important to delineate degree of injury; may require examination under anesthesia and arthroscopy. For mild laxity (isolated PCL tear), may treat with PRICES, functional rehabilitation, protective bracing. For moderate or severe laxity, surgical repair/reconstruction is usually required. **Chronic,** may attempt functional stabilization through rehabilitation and bracing; often requires surgical reconstruction if instability is more than mild.

MENISCAL INJURIES

Medial Meniscus

Description: Disruption of medial semilunar cartilage of the knee; may be from single traumatic episode, degenerative processes, or a combination. Tears take different forms, such as radial, longitudinal, or horizontal. Most important surgical factor is whether tear is peripheral in vascular zone or more central in nonvascular zone. More common than lateral meniscus tears because medial meniscus is less mobile.

Mechanism of injury: Twisting or squatting; may be in association with ligament injuries due to any of their precipitating mechanisms.

Presentation: Usually mild swelling and joint line pain. In acute setting, important to know whether knee lacked full extension from time of injury (locked knee from displaced fragment), or knee lacked full extension next day (pseudolocking from hamstring spasm). In chronic setting, recurrent locking is typical. Otherwise, symptoms may include slipping or catching over the joint line.

Examination: Positive McMurray's and Apley's tests; results may vary considerably from one examination session to next. Joint line tenderness and mild effusion may be present. Chronically, quadriceps atrophy is common. With peripheral meniscus detachment and positive anterior drawer test, loud "clunk" may be elicited as meniscus displaces during anterior drawer testing.

Imaging: Plain films are usually normal, unless meniscal tear has been present for significant time. After that time, they may show joint line spurring and/or narrowing. MRIs have now supplanted arthrograms for diagnosis of meniscal injury. For medial meniscus, MRI has sensitivity as high as 94% (Fig. 49-5).

Differential diagnosis: Ligamentous injury (causing pain in same area), patellar problems (anteromedial joint pain that is confused with pain from medial meniscus injury), pathologic synovial plica (similar pain, swelling, catching, and popping), loose bodies may cause locking, medial compartment arthritis (medial joint pain similar to that from torn meniscus).

Treatment: Suspected meniscus tear with no ligamentous instability may be managed initially through symptomatic treatment and functional rehabilitation. If no improvement, or if time constraints do not allow initial conservative treatment, diagnostic arthroscopy is most certain way of diagnosing and treating meniscal injury. MRI may help decide whether to proceed with surgical treatment or continue with nonsurgical care. Most meniscal tears still require arthroscopic partial meniscectomy. Meniscectomy increases chance of development of future arthritic changes in knee. Vertical tears in peripheral vascular zone are now routinely treated by meniscal repair rather than removal of meniscus.

Lateral Meniscus

Description: Disruption of lateral semilunar cartilage of knee; may be from single traumatic episode, degenerative processes, or combination. With lateral meniscus injury, may also encounter injuries of congenital discoid meniscus (see Fig. 49-5). Tears take different forms, such as radial, longitudinal, or horizontal (see Fig. 49-5). Most important surgical factor is whether tear is peripheral in vascular zone, or more central in nonvascular zone. Lateral meniscus tears are less common than medial meniscus tears because lateral meniscus is more mobile (see Fig. 49-5).

Mechanism of injury: Same as for medial meniscus injury.

Presentation: Same as for medial meniscus injury, although often more pain and fewer mechanical symptoms than with medial meniscus tears. Patient may give history of cystic lesion directly over lateral joint line.

Examination: Much the same as for medial meniscus injury; may palpate localized puffiness or distinct cystic lesion over lateral joint line.

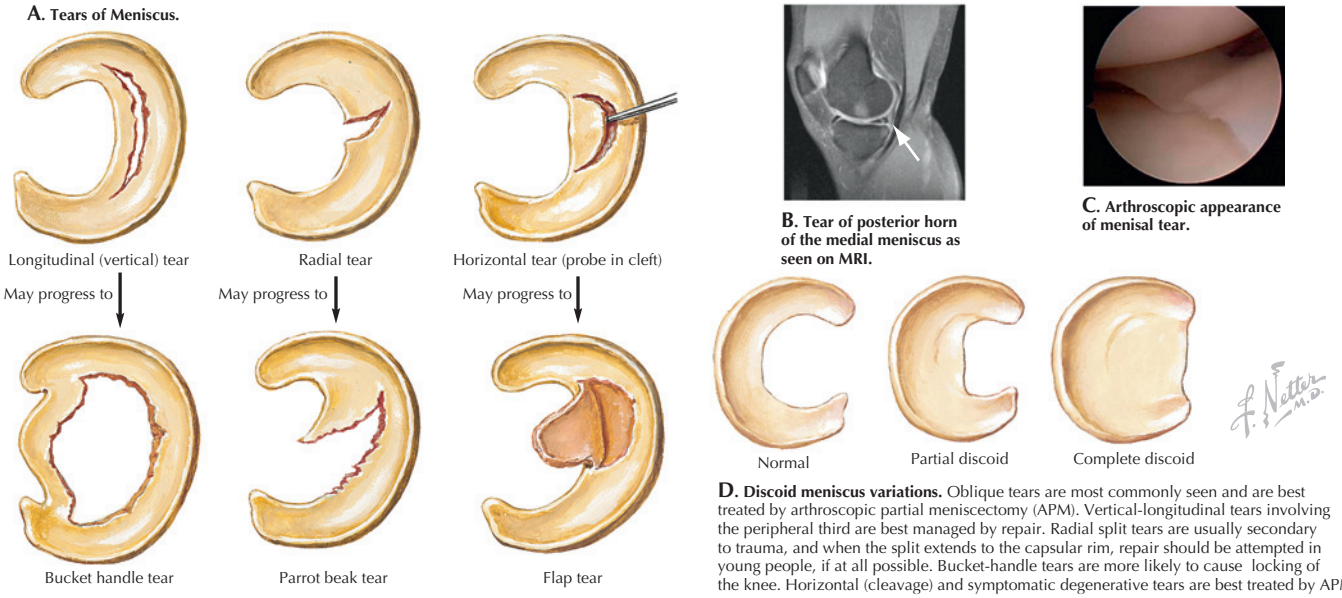


Figure 49-5 Meniscal Injuries. *(MRI and arthroscopy images reprinted with permission from Hart J, Miller M: Netter's Musculoskeletal Flash Cards. Philadelphia: Elsevier, 2007.)*

Imaging: Same findings as for medial meniscus injury. In child with congenital discoid lateral meniscus, may see widening of lateral joint space. In contrast to medial meniscus, MRI has somewhat less sensitivity (approximately 78%) in detecting lateral meniscus tears.

Differential diagnosis: Lateral ligamentous injury, loose bodies, degenerative arthritis of lateral compartment; popliteus tendinitis; iliotibial band friction syndrome.

Treatment: Same as for medial meniscus injury. Special considerations in youngsters with lateral discoid meniscus include whether to remove or repair and how much meniscus to remove.

EXTENSOR MECHANISM PROBLEMS

Instability Syndromes

Dislocation

Description: Complete, usually lateral displacement of patella from femoral trochlea that persists until reduced, usually by extending knee.

Mechanism of injury: Valgus and/or twisting with strong quadriceps contraction.

Predisposing factors: All of the stigmata indicating congenital extensor mechanism malalignment, such as vastus medialis obliqus (VMO) dysplasia, vastus lateralis (VL) hypertrophy, high and lateral patellar posture, increased Q-angle, bony deformity. Usually more easily seen in acute case on opposite uninjured side.

Presentation: May or may not be previous symptoms of instability or patellofemoral pain. Feeling of patellar dislocation when injury occurred; report of lying on ground with knee flexed; report of "something coming out" medially, which usually represents uncovered medial femoral condyle rather than patella going medially; report of "something going back into place" when knee extended. Swelling occurs within first 2 hours.

Examination: Depends on whether patella is still dislocated or has been reduced. Predisposing physical findings seen on opposite knee. If patella is still dislocated, will be located over lateral femoral condyle with prominence of uncovered medial femoral condyle (Fig. 49-6). If patella has been reduced, there may be large hemarthrosis with hypermobility and marked apprehension on hypermobility testing. May also find associated medial ligamentous instability.

Imaging: Unusual to find patella still dislocated on x-ray, because positioning on x-ray table usually reduces dislocation. Infrapatellar view may show avulsion of medial edge of patella. Large osteochondral fracture may be visible. Important to take infrapatellar view with knee flexed only 30 to 45 degrees, rather than traditional "sunrise" or "skyline" view with knee flexed beyond 90 degrees. Patella alta can be measured objectively on lateral view. Lesions of medial supporting structure often visualized on MRI.

Differential diagnosis: In acute case, differentiate from ligamentous tears; in chronic recurrent case, distinguish from meniscus disorders.

Treatment: If patella dislocated, knee extension and gentle pressure along lateral patellar edge usually reduces it easily and without anesthesia. Aspiration may be indicated for comfort or to search for fat in blood secondary to osteochondral fracture. Thoughts about rigid immobilization are changing, even with first-time dislocation, because of harmful effect of immobilization on knee joint. Immobilize first-time dislocation only as needed for symptoms, followed by extensive rehabilitation program and functional patellar bracing. Obvious disruption of VMO insertion into medial patellar edge or rupture of medial patellofemoral ligament from adductor tubercle does best with early surgical repair. Treat recurrent dislocation symptomatically with crutches, followed by functional rehabilitation and bracing. Consider surgical realignment of extensor mechanism if there is residual functional disability despite extensive conservative treatment. Surgical realignment is not always successful and should be last option.

Subluxation

Description: Transient partial displacement of patella from femoral trochlea; may occur acutely, as in patellar dislocation, or may be intermittent. There is spontaneous reduction of displacement.

Mechanism of injury: Same as for patellar dislocation; may occur with less severe force or in normal everyday activity.

Predisposing factors: Same as for patellar dislocation.

Presentation: Patient may or may not have history of complete dislocation or patellofemoral pain. Feeling of slipping when cutting, twisting, or pivoting; mild recurrent swelling.

Examination: Predisposing physical findings seen in both knees, but may be more obvious on asymptomatic side, especially if

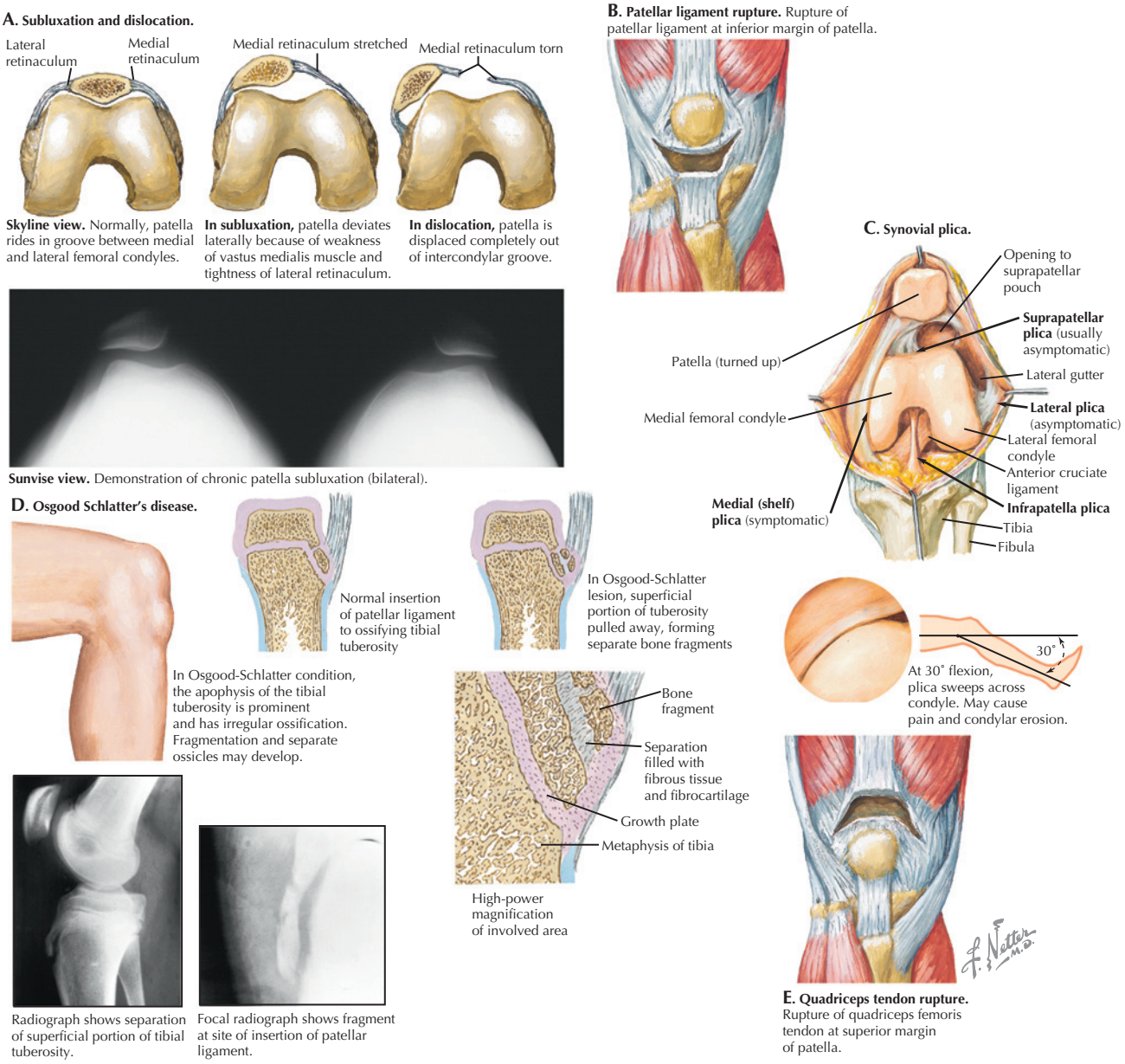


Figure 49-6 Extensor Mechanism Problems. *(Sunrise view reprinted with permission from Miller M, Cole B: Textbook of Arthroscopy. Philadelphia: Elsevier, 2004.)*

there has been an acute injury on symptomatic side. Mild effusion; positive hypermobility and apprehension test.

Imaging: Infrapatellar x-ray view must be done with proper technique and knee flexed only 30 to 45 degrees; may show lateral tilt and/or lateral subluxation or be normal in appearance. Patellofemoral indices (Merchant, Laurin, Brattstrom) show tendency to patellofemoral problems but do not give specific diagnosis (see Fig. 49-6).

Differential diagnosis: Chronic knee ligament instability, causing giving-way of knee.

Treatment: For acute subluxation, use temporary symptomatic immobilization, followed by functional rehabilitation and bracing of patella. If no acute episode, treat with functional rehabilitation, bracing, and NSAIDs. Patients disabled by subluxation may require arthroscopic lateral release or open extensor mechanism reconstruction. Surgical treatment has less than perfect results and should be considered last option (see Fig. 49-6).

Painful Syndromes

Patellofemoral Pain Syndrome

Description: Various syndromes characterized by anterior knee pain as major symptom. Imprecise term in which pain is not explained by more readily definable cause; often called "chondromalacia patella," a term that should be reserved for articular cartilage damage actually observed.

Mechanism of injury: May result from extensor mechanism malalignment, with or without an instability syndrome; may occur as overuse injury with extreme and/or repetitive loading of patellofemoral joint (e.g., knee flexion, running, jumping).

Predisposing factors: Same as for instability syndromes.

Presentation: Anterior knee pain, often worse with sitting in tight space with knee flexed and on descending stairs or slopes. Mild swelling (may be bilateral). May be snapping and popping around patella.

Examination: Findings predisposing to extensor mechanism problems in both legs; pain on patellofemoral compression test; crepitation about patella on range of motion; tenderness to palpation around patella. Mild effusion may be present. Foot malalignment or leg length inequality may aggravate symptoms.
Imaging: Same as for subluxation; may be normal.
Differential diagnosis: In preadolescents and young adolescents, consider referred pain from hip disorder (e.g., Legg-Calvé-Perthes disease, slipped capital femoral epiphysis), osteochondritis dissecans of femur or patella, bone tumor—especially in case of unilateral symptoms. In older patient, osteoarthritis or some other inflammatory joint disease.
Treatment: Functional rehabilitation program, NSAIDs, functional bracing of patella, orthotics for foot malalignment. If other treatments are unsuccessful, surgical treatment may be considered—either lateral release or extensor mechanism reconstruction. Because results are unpredictable, surgery should be considered last option. Always look for other more specific cause of anterior knee pain.

Patellar Tendinitis ("Jumper's Knee")

Description: Inflammation of patellar tendon, usually at its attachment to inferior pole of patella. Rupture of the patellar tendon may occur with or without a history of tendinopathy (see Fig. 49-6).
Mechanism of injury: Usually excessive jumping or bounding activity or other high patellofemoral stress activity; less commonly from running.
Predisposing factors: Same as for other extensor mechanism disorders; possibly ankle dorsiflexor muscle weakness, perhaps secondary to ankle injury.
Presentation: Activity, such as jumping sport, typically associated with this problem; complaint of infrapatellar pain, originally after exercise, later during exercise and while at rest. Rupture occurs with forceful knee flexion against resistance.
Examination: Tenderness at inferior pole of patella; less commonly, tenderness over body of patellar tendon. Other findings of extensor mechanism malalignment; weakness of ankle dorsiflexors; hamstring, heel cord, and/or quadriceps muscle tightness. Patellar tendon incongruity and significant knee extension weakness noted.
Imaging: X-rays occasionally show irregularity at inferior pole of patella; may show extensor mechanism malalignment, including patella alta (especially with rupture). MRI may demonstrate degenerative changes in tendon, which are often read as a partial tear of the patellar tendon by the radiologist but really are representative of changes in the tendon consistent with patella tendinosis (see Fig. 49-6).
Differential diagnosis: Usually firm diagnosis not difficult with this entity. May consider some other soft tissue lesion of patellar tendon or fat pad, such as tumor; otherwise, could be any of other causes of patellofemoral pain.
Treatment: Rehabilitative exercise program, concentrating on hamstring, heel cord, and quadriceps flexibility, as well as quadriceps strength; eccentric strengthening exercises for ankle dorsiflexors are important; anti-inflammatory medication; ultrasound, using hydrocortisone phonophoresis. Questionable benefit from infrapatellar strap. More invasive but relatively safe measures used to treat recalcitrant patella tendinosis include prolotherapy (injection at multiple points in the tendon with "sugar water") and injection of platelet-rich plasma gel. Surgical treatment should be last resort because of unpredictable results. Immediate surgical repair is indicated with rupture.

Synovial Plica

Description: Structurally, remnant of embryologic walls that divide knee into medial, lateral, and suprapatellar pouches; appears as fold of synovium attached to periphery of joint and to underside of quadriceps tendon (suprapatellar plica). May also present as free edge along medial patellofemoral joint (medial plica) or may be in both locations; rarely seen in other configurations. Edge protruding into joint may be of various sizes (see Fig. 49-6).
Mechanism of injury: Overuse with repetitive flexion and extension (e.g., running); direct blow to medial patellofemoral joint (e.g., falling on turf or dashboard injury).
Predisposing factors: Congenital presence of plica. Other extensor mechanism malalignment predispositions may increase likelihood of symptoms because of plica.
Presentation: Complaints of anterior knee pain, pain over suprapatellar or medial peripatellar regions with long periods of knee flexion (especially when accompanied by distinct snap or pop when knee is extended), painful catching episodes over medial patellofemoral joint.
Examination: Often difficult to palpate plica; best done with passive flexion and extension with tibia held internally rotated. Fingers should lie over medial patellofemoral joint. May see other extensor mechanism malalignment stigmata. Heel cord tightness and hamstring tightness aggravate significantly.
Imaging: Not helpful.
Differential diagnosis: Other painful patellofemoral conditions; possibly medial meniscus injury or loose body. Patients often dismissed as "neurotic" because of lack of findings in face of significant symptoms.
Treatment: If inflammatory process is not reversible and plica is fibrotic, persistent symptoms require arthroscopic removal of plica; promises good relief of symptoms and good future function. If inflammatory process in synovium is still reversible:
- Condition may improve with hamstring stretching, heel cord stretching, or VMO exercises (if VMO is dysplastic).
- NSAIDs, ice, activity modification.
- Simple external patellar support may help.
- Phonophoresis and/or a corticosteroid injection to plica area may also be beneficial.

Osgood-Schlatter's "Disease"

Description: Painful enlargement of tibial tuberosity at patellar tendon insertion. Rather than being a disease, condition is caused by mechanical stress and excessive tension on growing tibial tuberosity apophysis. Occurs in preadolescence and early adolescence, usually during rapid growth period (see Fig. 49-6).
Mechanism of injury: Overuse in normal childhood activities, including sports; rarely, acute onset of popping and pain over tibial tuberosity.
Predisposing factors: Patella alta, other evidence of extensor mechanism malalignment and altered extensor mechanics. Tight hamstrings, heel cords, quadriceps muscles predispose to symptoms.
Presentation: Complaints of painful enlargement of tibial tuberosity.
Examination: Enlarged, tender tibial tuberosity; stigmata of extensor mechanism malalignment, especially patella alta; tight hamstrings, heel cords, and quadriceps muscles.
Imaging: Enlarged tibial tuberosity, irregularity of tibial tuberosity, loose ossicle separated from tuberosity, patella alta shown in x-rays.
Differential diagnosis: Other forms of patellar tendinitis. In acute episode, avulsion fracture of tibial tuberosity. Tumorous processes of tibial tuberosity.
Treatment: Hamstring stretching, heel cord stretching, and quadriceps stretching exercises; VMO strengthening exercises; activity modification as necessitated by symptoms; simple modalities; local padding.

Quadriceps Tendinitis (Including VL Tendinitis and VMO Tendinitis) and Rupture

Description: Inflammation of quadriceps tendon at its insertion into superior edge of patella. May involve only VL insertion into

superolateral pole of patella or VMO insertion into superomedial pole of patella. Rupture of the quadriceps mechanism may occur with or without a history of tendonitis following forceful knee flexion against resistance (see Fig. 49-6).

Mechanism of injury: Same as for patellar tendinitis.

Predisposing factors: Extensor mechanism malalignment.

Presentation: Complaints of suprapatellar pain.

Examination: Tenderness at superior pole of patella; may be over central rectus femoris insertion, superolateral VL insertion, or superomedial VMO insertion. Other findings of extensor mechanism malalignment; hamstring, heel cord, and quadriceps muscle tightness. Quadriceps tendon defect and inability to extend knee present with rupture.

Imaging: Usually there are no findings on x-ray studies with tendonitis, but may observe patella baja with rupture.

Differential diagnosis: Suprapatellar pain from synovial plica, bone tumor of distal femur.

Treatment: Same as for patellar tendonitis; immediate surgical repair for rupture.

MISCELLANEOUS KNEE CONDITIONS

Bursitis

Description: Inflammation of any of various bursae around knee, evidenced by swelling and/or pain; typically prepatellar bursa, pes anserinus bursa, tibial collateral ligament bursa, deep infrapatellar bursa (Fig. 49-7).

Mechanism of injury: Usually overuse; may be due to direct blow with bleeding into bursa.

Predisposing factors: For pes anserinus bursitis, tight hamstrings seem to predispose.

Presentation: Complaints of swelling (if prepatellar bursa), pain in prepatellar region (for prepatellar bursitis), pain in distal patellar tendon region (for deep infrapatellar bursitis), pain in proximal medial tibia (for pes anserinus bursitis), or pain over medial joint line (for tibial collateral ligament bursitis).

Examination: For prepatellar bursa, look for localized swelling and tenderness; for others, tenderness over described areas.

Imaging: Not helpful for diagnosis.

Differential diagnosis: For deep infrapatellar bursitis, other causes of patellar tendon pain; for tibial collateral ligament bursitis, medial meniscus tear; for prepatellar bursitis, usually no differential; for pes anserinus bursitis, pain from pes anserinus tendons, tumors, other causes of proximal medial tibial pain.

Treatment:

- Acute prepatellar bursitis: ice, compression, possible aspiration, padding.
- Chronic prepatellar bursitis: NSAIDs, compression, hamstring stretching, ultrasound, possible aspiration and corticosteroid injection.
- Pes anserinus bursitis: hamstring stretching, ultrasound, NSAIDs, corticosteroid injection.
- Tibial collateral ligament bursitis: injection, both as diagnostic test and as treatment.
- Deep infrapatellar bursitis: hamstring stretching, possible injection behind patellar tendon.

Other Tendonitis

Description: Inflammation of any other tendinous structures about knee, typically semimembranosus, popliteus, or biceps femoris tendons; inflammation of gastrocnemius tendon is rare.

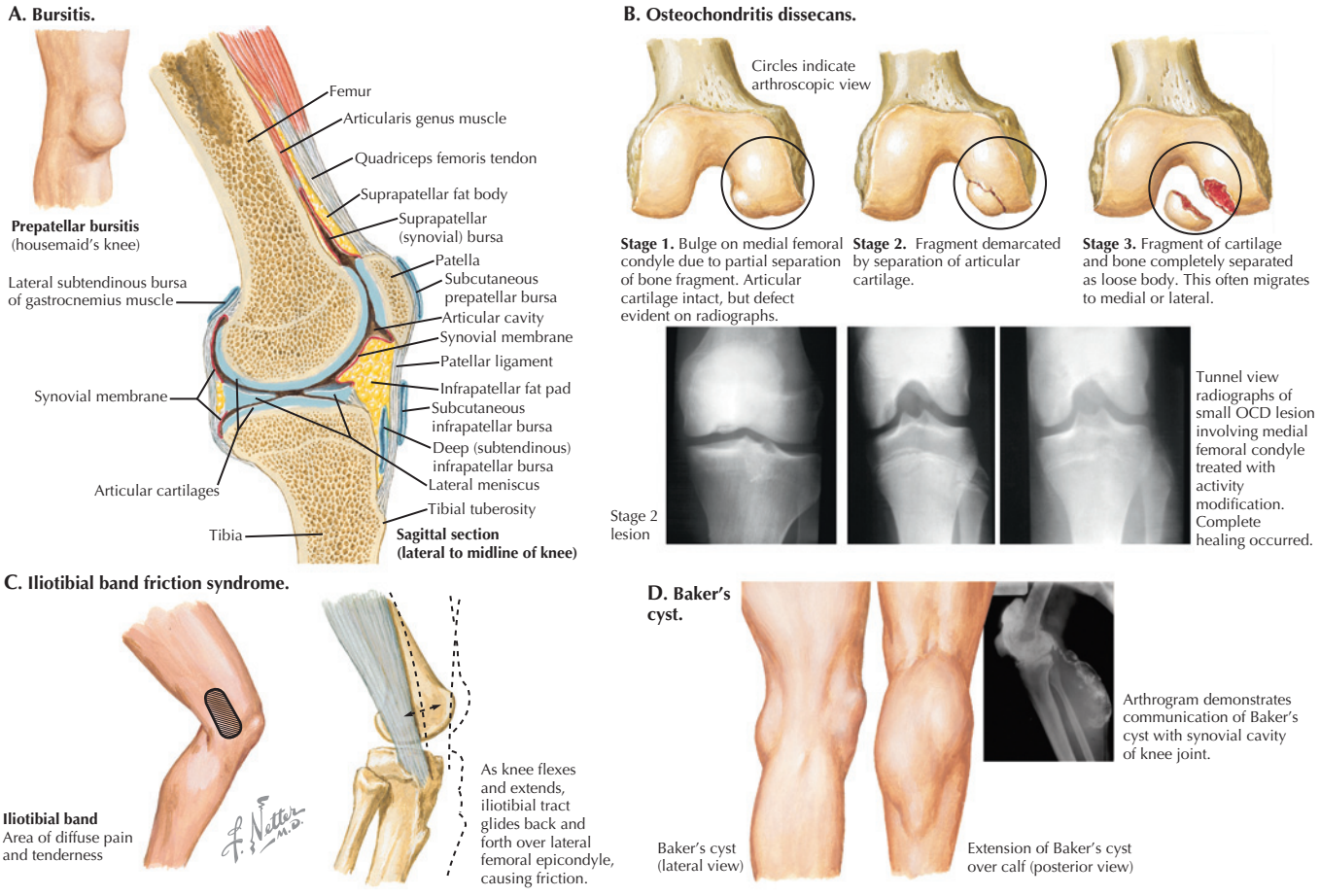


Figure 49-7 Miscellaneous Knee Conditions.

Mechanism of injury: Usually overuse; much less commonly, single episode of strain. Popliteus tendinitis is usually running injury.
Predisposing factors: For semimembranosus, pes anserinus, or biceps femoris tendinitis, hamstring tightness predisposes.
Presentation: Complaints of pain over appropriate tendon area; for popliteus tendinitis, lateral knee pain, especially while running downhill.
Examination: Tenderness over appropriate tendon; tight hamstrings. For popliteus tendinitis, painful resisted internal rotation of tibia with knee flexed. For all, initially pain on stretching tendon, later pain on active contraction of tendon.
Imaging: Usually not helpful.
Differential diagnosis: Hamstring tendinitis occasionally to be differentiated from sciatica. Semimembranosus tendinitis may be confused with medial meniscus disorders. For popliteus tendinitis, lateral meniscus injury, iliotibial band friction syndrome.
Treatment: Usual anti-inflammatory methods; hamstring stretching exercises. Patient may require corticosteroid injection. Immobilization is inappropriate except in most acute phase because of adverse effects on collagen tissue and muscle atrophy.

Neuromas

Description: Nonneoplastic enlargement of nerve, usually from direct trauma; typically involves various portions of saphenous nerve around knee.
Mechanism of injury: Direct blow, previous surgery.
Predisposing factors: Previous surgical incisions.
Presentation: Pain, particularly nervelike quality (i.e., paresthesias, burning, other alterations of sensation).
Examination: Tenderness over neuroma, positive Tinel's sign, objective changes in sensation in appropriate distribution.
Imaging: Not helpful.
Differential diagnosis: More central sources of nerve compression.
Treatment: Injection, surgical excision.

Loose Bodies ("Joint Mouse," Chondral Fracture, Osteochondral Fracture, Osteochondritis Dissecans)

Description: Cartilaginous or osteocartilaginous fragments usually free-floating within knee joint (though may be attached to synovium more or less firmly) (see Fig. 49-7).
Mechanism of injury: Dislocation of patella (see "Instability Syndromes"), other trauma to joint surface. May be result of preexisting osteochondritis dissecans; rarely due to synovial osteochondromatosis.
Predisposing factors: Predisposition to patellar dislocation, preexisting osteochondritis dissecans.
Presentation: Consistent with previous patellar instability, twisting or direct blow injury, locking episodes, subcutaneous mass that comes and goes and may be felt in various locations about knee.
Examination: Patellar findings. May feel movable mass, usually around patellofemoral joint, although it may be at anteromedial or anterolateral joint line.
Imaging: In x-rays, purely cartilaginous fragments are not visible; tunnel view may reveal small osteochondritis dessicans (OCD) lesion on lateral aspect of medical femoral condyle; very small bony loose bodies may be obscured; most loose bodies of significance containing bone are visible; source (e.g., osteochondritis dissecans) may be seen. MRI, tomogram, CT scan, or arthrogram may help in delineation (see Fig. 49-7).
Differential diagnosis: Meniscal tears as source of locking.
Treatment: Symptomatic loose bodies require surgical removal, usually arthroscopically. A few loose bodies may require replacement and internal fixation. Patellofemoral instability may require treatment. Osteochondritis dissecans may require other treatment. Large chondral or osteochondral fracture may require surgical debridement of joint surface and prolonged protected weight-bearing.

Cysts (Popliteal Cyst, Popliteal Ganglion, Baker's Cyst, Meniscus Cyst)

Description: Fluid-filled lesion about the knee arising usually as extension of synovial space, either into normal bursal structure or into soft tissue surrounding knee (see Fig. 49-7).
Mechanism of injury: Normally, no specific injury is involved.
Predisposing factors: None.
Presentation: Localized swelling in popliteal space or over meniscus.
Examination: Cystic swelling in medial popliteal space or over mid–joint line, usually lateral joint line.
Imaging: Plain films are no help; MRI is very helpful in delineating cysts (see Fig. 49-7).
Differential diagnosis: Other tumorous lesions about knee.
Treatment: Aspiration and injection with corticosteroid not very likely to give permanent cure; surgical excision is usually curative. Presence of cyst is usually secondary to another process in knee that leads to excessive synovial fluid, in turn causing cyst. Most likely meniscal tear causing popliteal cyst, or lateral meniscus tear causing lateral meniscus cyst. Underlying disorders must be treated.

Iliotibial Band Friction Syndrome

Description: Chronic inflammatory process involving soft tissues adjacent to lateral femoral epicondyle; presumably caused by chronic "friction" of iliotibial band rubbing over bony prominence of this area (see Fig. 49-7).
Mechanism of injury: Overuse; most cases caused by running.
Predisposing factors: Varus alignment of knee; running on sloped surfaces.
Presentation: Lateral knee pain on activity, occasional popping.
Examination: Tenderness over lateral femoral epicondyle, tight iliotibial band, absence of intra-articular findings.
Imaging: Not helpful.
Differential diagnosis: Other causes of lateral knee pain, especially popliteus tendinitis; lateral meniscus disorders, lateral patellofemoral joint sources such as VL tendinitis.
Treatment: Iliotibial band stretching exercises, anti-inflammatory treatment, ultrasound to lateral femoral epicondyle, corticosteroid injection. Rarely, surgery to release area of tightness.

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Ankle and Leg Injuries

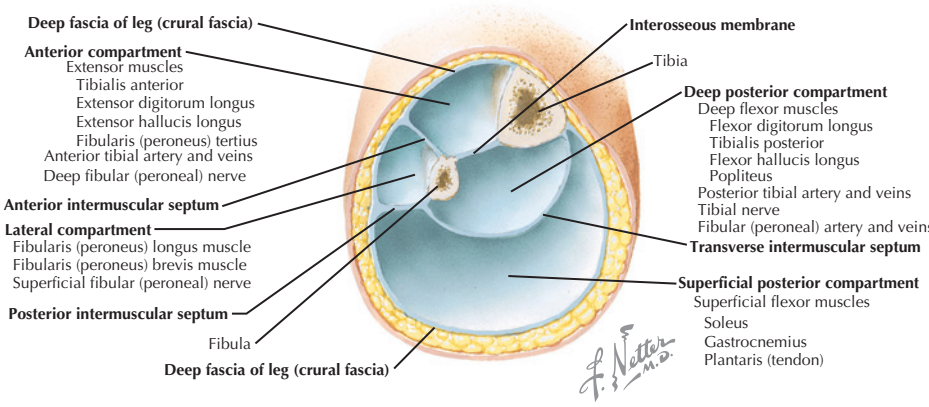
John E. Femino and Annunziato Amendola

GENERAL PRINCIPLES OF EVALUATION

- Leg and ankle are structurally and functionally united and evaluation of one must include the other (Fig. 50-1).
- Leg is composed of four fascial compartments that are in continuity with the ankle and foot via tendons, nerves, and blood vessels.
- Acute injuries to the leg that require immediate treatment include open fractures, dislocations and sprains, and acute compartment syndrome.
- Incidental finding of malignancies may first present with the evaluation of an acute or chronic injury.
- Malignant melanoma is the most common malignancy of the foot and ankle.
- Ankle sprains or inversion injuries are the most common injury of the ankle.
- Chronic pain and disability may result from lack of diagnosis of an associated injury, delayed treatment, or incomplete rehabilitation.

HISTORY

- Acute problems
  - Determine the location of pain and mechanism of injury.
  - Is the patient able to bear weight on the injured extremity, if so usually not a fracture.
  - Is there a history of prior injury, surgery, or prodromal symptoms, as with some stress fractures or overuse syndromes.
- Chronic problems
  - A detailed history of training activities should be obtained, including frequency, duration, and intensity of activity.



Muscles of Leg (Superficial Dissection): Anterior View

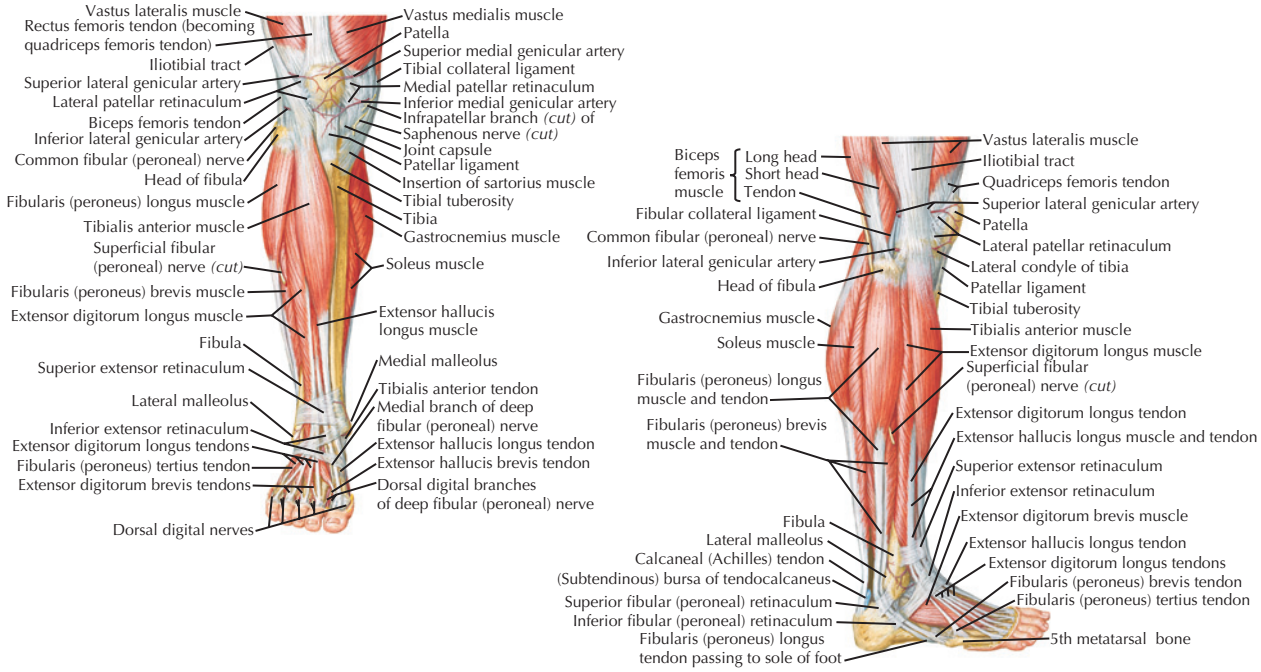


Figure 50-1 Compartments of the Leg and Ankle.

- Determine the type of shoe wear and any orthoses that have been used leading up to and just after the onset of symptoms.

PHYSICAL EXAMINATION

Inspection

- Evaluate skin for abrasions or lacerations; check for any bleeding that has fat globules that would indicate an open fracture.
- Evaluate for any deformity that may indicate a displaced fracture or dislocation.
- Evaluate for any skin color changes that may indicate autonomic instability as seen with reflex sympathetic dystrophy or cellulitis.

Palpation

- Palpation should be systematic, anatomically based, and end with the most painful area.
- Palpation of pulses may be limited because of location of pain: posterior tibial artery is palpated behind the medial malleolus, anterior tibial artery is palpated beneath the extensor hallucis longus or over the first intercuneiform joint at the dorsalis pedis, peroneal artery pulse is found anteriorly over the syndesmosis (Fig. 50-2).
- Palpation of bony prominences should include the tibial crest from the knee to the ankle (see Fig. 50-2).
- Palpation of the compartments of the leg should reveal supple compartments; hard compartments may indicate compartment syndrome. Detection of tenderness may indicate muscle or nerve injury. Palpation of the ligaments should include the lateral ligaments. Tenderness of the syndesmosis is palpated over the anterior aspect of this joint between the tibia and fibula just above the ankle joint line and extending proximally several centimeters. Palpation of the medial ankle ligaments, the superficial and deep deltoid ligaments, is somewhat difficult because of the overlying flexor retinaculum and posterior tibial tendon. The anterior band of the deltoid between the medial malleolus and navicular is superficial and can be directly palpated (see Fig. 50-2).
- Palpation of the tendons should include the Achilles, posterior tibial, flexor hallucis longus (FHL) and the peroneal; anterior tibial tendon and digital extensors can be palpated individually but are less frequently injured (see Fig. 50-2).
- Ankle joint is directly palpated medial to the anterior tibialis over the anteromedial recess and lateral to the peroneus tertius over the anterolateral recess; fullness from joint effusion can be detected in these two areas.
- Palpation of the ankle posteriorly on either side of the Achilles tendon may reveal tenderness caused by fracture or injury of the trigonal process of the talus, os trigonum, FHL tendonitis, or posterior ankle impingement.

Range of Motion

- Ankle range of motion is assessed for dorsiflexion and plantarflexion with the knee flexed and extended in the subacute setting.
- Limitation of dorsiflexion with the knee extended may indicate a contracture of the gastrocnemius portion of the Achilles tendon.
- In the acute setting, determining pain with joint motion will help the examiner to focus the exam on the supporting ligaments of the ankle and evaluation with x-rays to detect any fractures.
- Subtalar joint motion is closely linked to ankle motion but can be isolated with one hand holding the lateral side of the foot and calcaneus and inverting and everting the foot, without moving the ankle.
- Syndesmotic motion is limited but can be tested by stressing the joint (see stress tests in following section). Pain with this maneuver strongly suggests syndesmotic instability, although the degree of motion may be imperceptible.

Muscle Testing

- Muscle strength is tested by the examiner resisting all four muscle groups.
- Resisted ankle and toe dorsiflexion and plantarflexion can be done simultaneously and strength and visualization of tendon tension can be assessed.
- Weak plantarflexion and loss of resistance to passive ankle dorsiflexion are the hallmarks of an Achilles tendon rupture, but some patients may have enough strength with their toe motors to produce significant plantarflexion force.
- Resisted inversion with the foot plantarflexed and everted isolates the posterior tibialis muscle.
- Resisted eversion tests the peroneals and detection of instability is enhanced with the ankle in dorsiflexion as well; peroneal tendons can be palpated with the opposite hand from behind the ankle.

Special Tests

The following tests are used to detect ligament instability or impingement of the ankle or subtalar joints.

Anterior drawer test: Best done seated and knee flexed, to remove the gastrocnemius as a resisting force. Ankle is plantarflexed and the examiner stabilizes the tibia with one hand while the other hand is placed along the lateral side of the foot with the fingers wrapping around the posterior aspect of the heel. Ankle is then shucked back and forth gently with emphasis on detecting anterior translation (Fig. 50-3). Motion should be combined with an internal rotation of the talus because true lateral ankle instability is a rotatory phenomenon. Severity of joint instability and reproduction of pain and apprehension should be noted.

Talar tilt test: A variation of the anterior drawer test with the force being applied with the ankle in less plantarflexion and a varus force being applied to the hindfoot; some internal rotation can enhance the test, which is supposed to detect insufficiency of the calcaneal fibular ligament (CFL) (see Fig. 50-3).

External rotation stress test: Can reveal syndesmotic injury. Stabilize the tibia and externally rotate the foot and ankle, with the knee flexed to 90 degrees and the heel locked in neutral. Reproduction of symptoms would be a strong indicator of syndesmotic injury. If controlling tibial rotation is difficult, have the patient stand on the involved leg and while stabilizing with the patient's arms for balance, have the patient rotate externally on the foot. This places a more realistic force across the syndesmosis.

Impingement testing: Performed in four locations with the joint in an "open" position and placing mild digital pressure of the point of impingement; position is plantarflexion for anterior impingement and dorsiflexion for posterior position. With pressure held the ankle is then passively moved into a "closed" position, which can then entrap the redundant tissue, reproducing the soft tissue impingement and pain. For bony impingement, often the talar osteophyte can be palpated while moving the ankle in dorsiflexion or plantarflexion, with the examiner's finger over the anteromedial or lateral recess of the ankle. This same maneuver can be done for the subtalar joint with the foot in an inverted, plantarflexed position and moved into a dorsiflexed and everted position.

Compartment pressure direct measurement: Most common measuring devices are solid-state battery powered units that use a side slit catheter (see Fig. 50-3). Reliable pressure for determining when a surgical release needs to be performed is controversial. Absolute pressure above 30 mm Hg or a difference between the compartment pressure and the diastolic blood pressure that is less than 30 mm Hg are very concerning. In the setting of

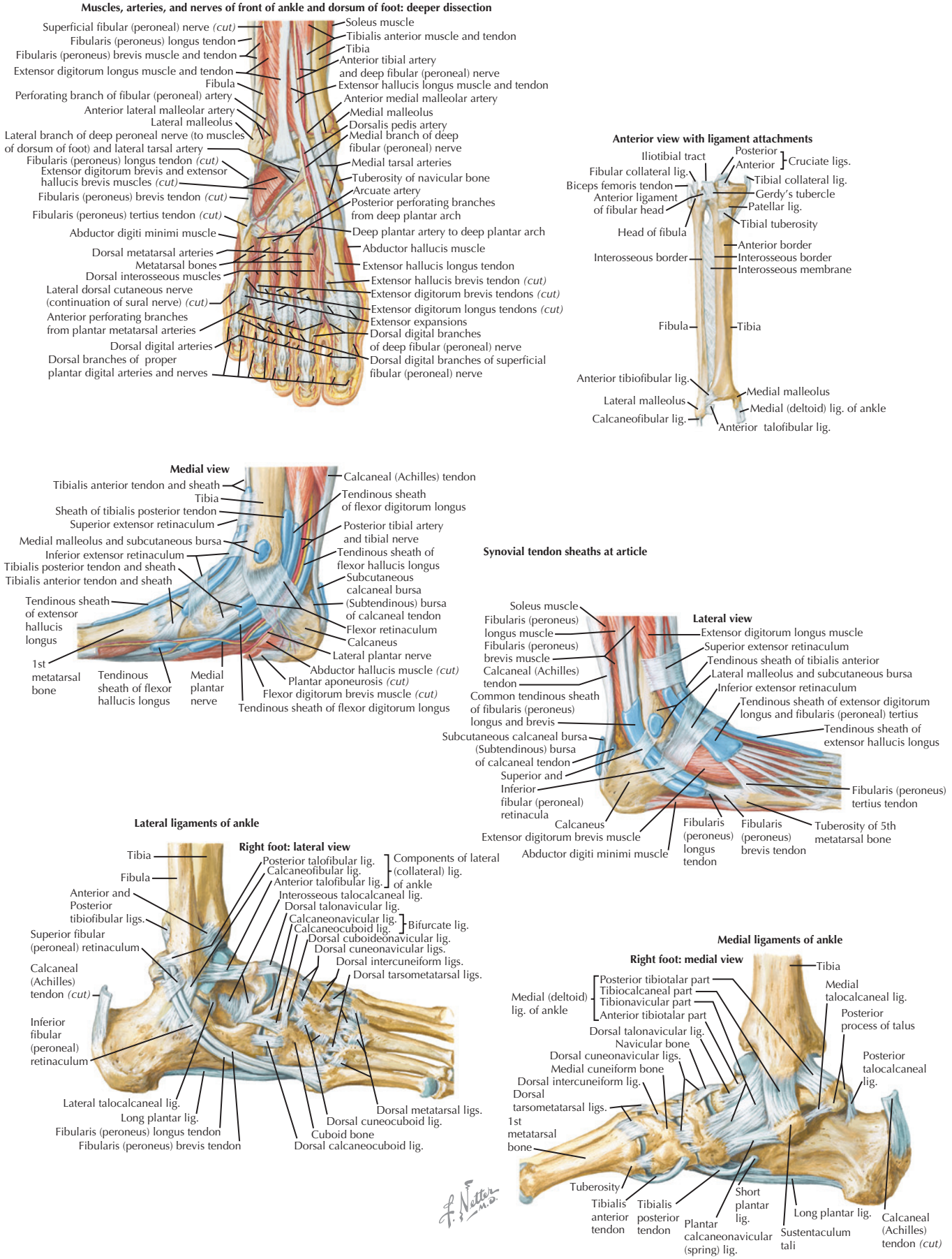


Figure 50-2 Anatomy of the Leg and Ankle.

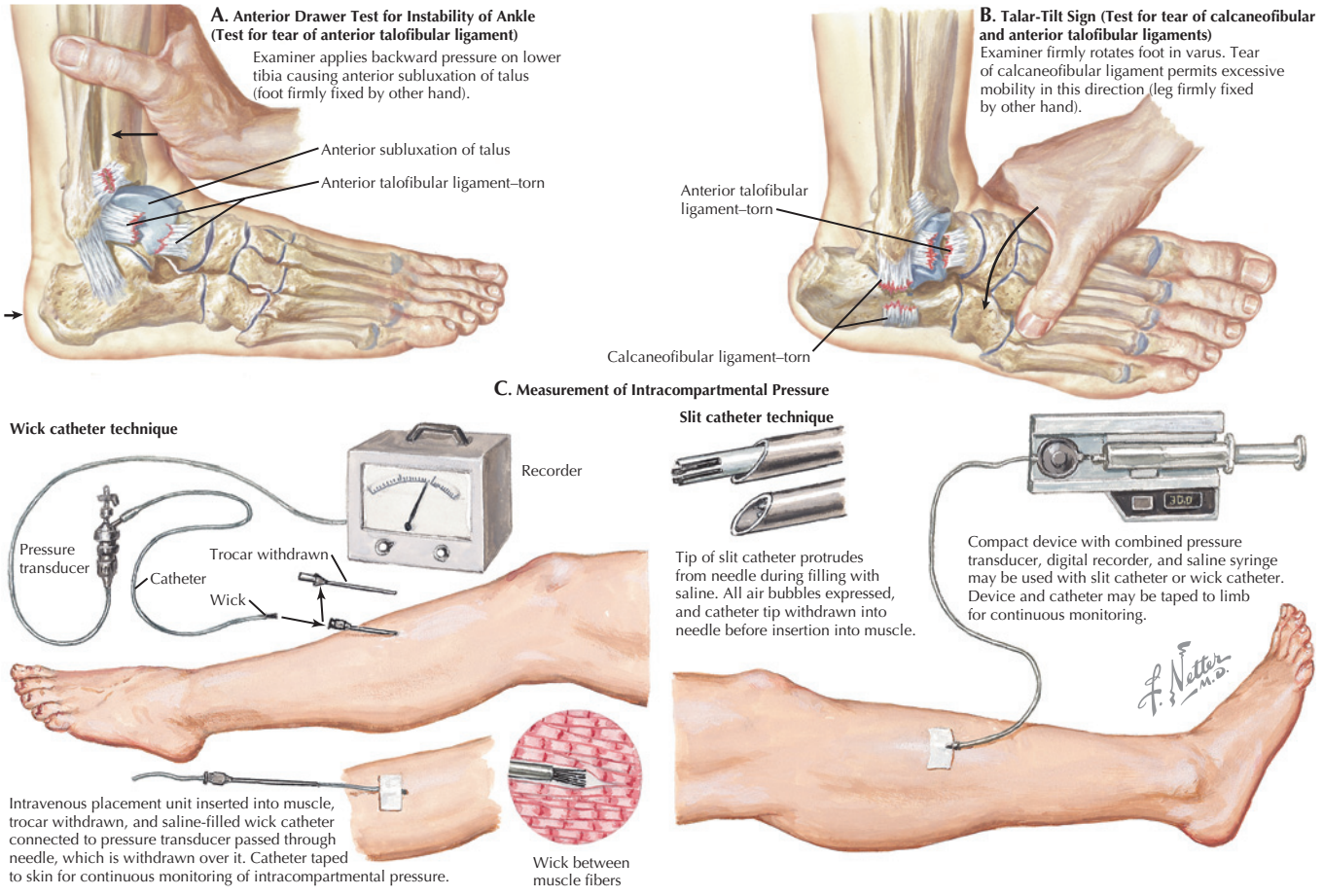


Figure 50-3 Special Tests.

an acute injury, the pressure measurement is an important part of the evaluation, but it is not the sole determinant. Pain in excess of what would be expected with the injury, palpation of a hard and tender muscle compartment, and severe pain with passive motion of the suspected muscles are strong clinical indicators of an acute compartment syndrome, and emergent surgical release may be indicated. In cases of chronic exertional compartment syndrome (ECS), the resting compartment pressure is usually around 10 to 12 mm Hg. The pressure can rise to near 80 mm Hg with activity, but it should return to the resting level within 5 minutes. If the history is consistent with exercise-induced compartment syndrome, resting pressure above 15 mm Hg, and postexercise pressure greater than 30 mm Hg that does not return to baseline values within 5 minutes, symptoms would be consistent with ECS. With these findings, fasciotomy may be indicated.

Radiological Tests

Plain x-rays: Should include injured area and likely other parts. For instance any injury to the ankle should include an anteroposterior (AP) and oblique foot view to detect peritalar injuries that may not be detected on standard AP, oblique, and lateral ankle views.

Stress x-rays: Helpful to document instability about the ankle, which may often guide treatment decisions; best compared to a normal contralateral part.

- Anterior drawer stress taken with a lateral x-ray can detect pathologic anterior translation.
- Talar tilt test on the AP x-ray view may demonstrated gross varus tilting of the talus with stress.
- External rotation stress test may show syndesmotic or associated medial ankle widening.

Nuclear medicine scans: Helpful to pinpoint bone pathology that is not seen on plain x-rays; findings, however, are not always specific enough and many times an MRI renders the bone scan unnecessary.

Computed tomography (CT) scans: Helpful in evaluating peritalar fractures; excellent at revealing details of bony anatomy and particularly the cortical bone.

Magnetic resonance imaging (MRI): Excellent visualization of soft tissue structures and bone marrow, making it very good at detecting early stress fractures.

SPECIFIC CLINICAL CONDITIONS OF THE LEG

Acute Fractures

Tibial Fractures

Description: May result in displaced and open fractures (Fig. 50-4).

Presentation: Bloody drainage with fat globules or palpable bone is an indication of an open fracture, and must be treated emergently with operative debridement and irrigation.

Treatment: Splinted initially. If severe deformity is present an urgent reduction is warranted, especially if there is any vascular compromise with a cool foot or absent pulses. Fractures with minimal or no displacement can be treated with cast immobilization; open fractures or displaced fractures treated with surgical fixation. Compartment syndrome can occur after tibial fractures and careful monitoring should be maintained, especially in the first 24 hours after injury.

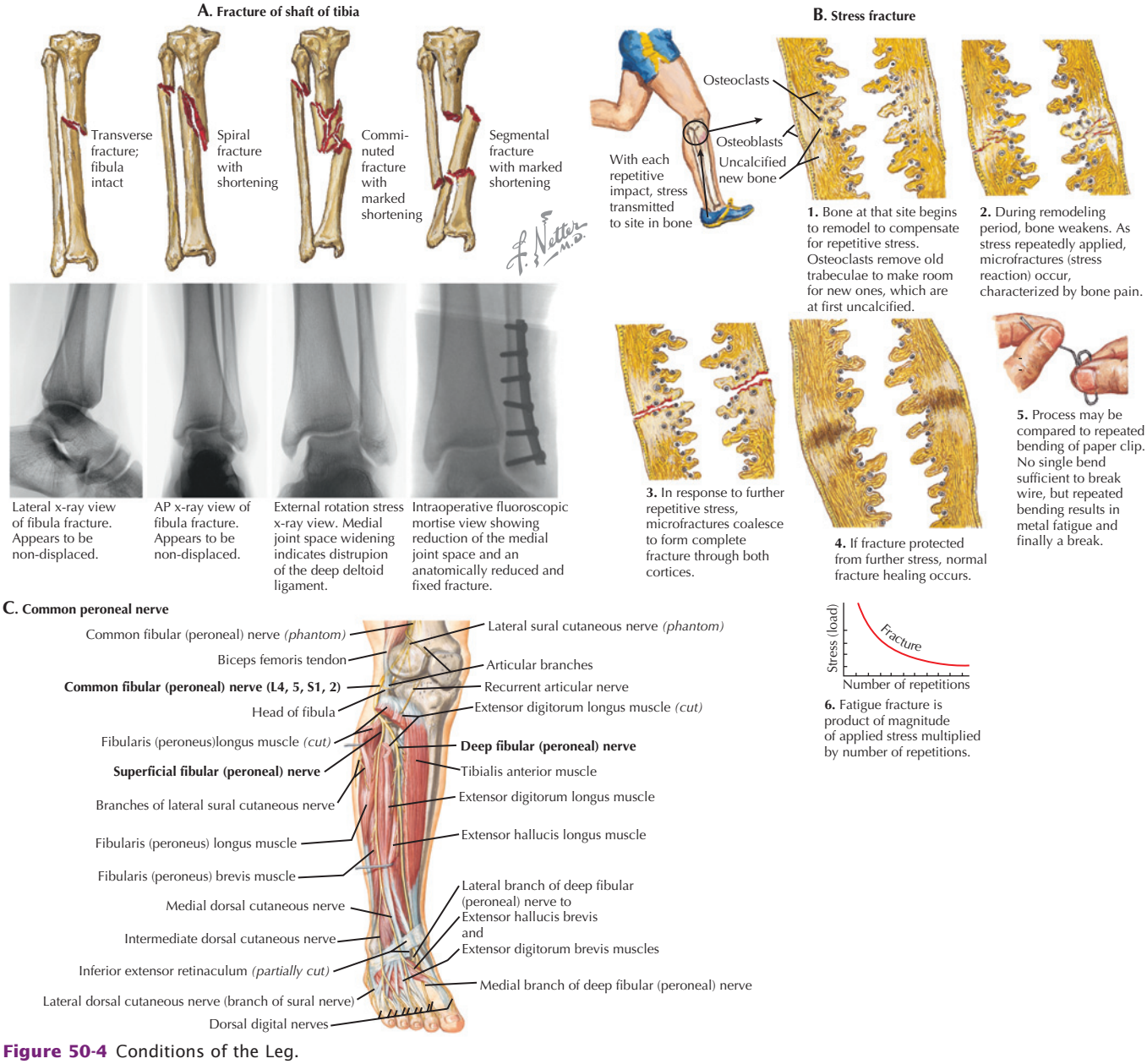


Figure 50-4 Conditions of the Leg.

Acute Fibular Shaft Fractures

Description: Associated with tibial fractures and are more likely with higher energy injuries. Fibula bears only 15% to 20% of the weight and alignment is often achieved with satisfactory alignment of the tibia. A seemingly low-energy fibular shaft fracture can be associated with significant syndesmotic injury.

Presentation: Indirect twisting mechanism of injury is likely.

Treatment: In fractures caused by a direct blow, soft tissue damage is minimal but bleeding can lead to compartment syndromes.

Syndesmotic Injuries with a High Fibula Fracture

Description: Devastating injuries and are often unstable injuries with disruption of the medial side of the ankle with a fracture of the medial malleolus or rupture of the deltoid ligament.

Examination: Displacement of the mortise may be subtle and one may see a medial malleolar fracture, a small avulsion fracture, or only subtle medial joint space or syndesmotic widening.

Imaging: In some cases, the only fracture may be the high fibula fracture, which will be missed if ankle x-rays alone are taken. This highlights the importance of performing a thorough physical examination of the entire leg when evaluating an ankle injury.

Ankle Fractures

Description: Any fracture that directly enters the ankle or is associated with a ligamentous injury that compromises the ankle mortise (see Fig. 50-4).

Classification: Based on fracture pattern or mechanism.

Treatment: Priority is to reduce any fracture where the ankle joint is subluxated or dislocated. Most fractures that require reduction are likely to be treated with operative reduction and fixation, which can reliably obtain and maintain anatomic reduction of the ankle mortise. Fractures that are minimally displaced and initially seem to have an intact mortise can be tested by stress radiographs with appropriate anesthesia to test for instability. If displacement occurs with stress then surgical reduction and fixation may be indicated. Fractures that are stable and can be

maintained anatomically reduced are well treated with cast immobilization. Transition to full weight bearing and a functional brace or removable cast can usually begin within 2 to 3 weeks, depending on the injury.

Chronic Exercise-Induced Leg Pain

Tibial Stress Fractures

Description: Can be in the proximal third, middle third, or distal third of the tibia; commonly seen in athletes who perform repetitive running and jumping such as track athletes (see Fig. 50-4).

Presentation: Pain may present acutely after a prodromal course of low-level pain.

Examination: Tenderness in the affected area and x-rays are usually diagnostic.

Imaging: When x-rays are not diagnostic, an MRI can reveal marrow edema and fracture across the bone.

Treatment: Incomplete fractures may be treated by a period of rest and immobilization for up to 8 weeks, with progressive return to training as symptoms allow. Complete fractures of the proximal and distal third of the tibia can be treated nonoperatively, but middle-third fractures are best treated by surgical stabilization with an intramedullary rod. Osteopenia is not uncommon and evaluation for bone density should be considered.

Medial Tibial Stress Syndrome

Description: Common cause of exercise-induced leg pain in athletes; seen in sports in which repetitive running and jumping are required. Considered a periostitis because of traction of the soleus and other posterior leg muscles.

Presentation: Pain along the posteromedial border of the midtibia. Pain improves with cessation of activity and rest, but does not completely resolve; as with stress fractures, focal tenderness is present.

Imaging: MRI is the best confirmatory test. The characteristic MRI finding is a linear longitudinal edema of the periosteum; this can clearly differentiate medial tibial stress syndrome from a tibial stress fracture, which has marrow edema and a transverse line of signal change.

Treatment: Initial treatment is rest and evaluation of training methods. If no response, surgery may be considered; results have been mixed. Most common surgical procedure is fasciotomy on areas adjacent to the area of pain, but this should be reserved for the most recalcitrant cases.

Chronic Exertional Compartment Syndrome (CECS)

Description: Condition in which the intracompartmental pressures lead to activity-limiting pain. Exact pathophysiology is not known, but some think in the setting of increased compartment pressure, muscle function becomes more anaerobic and results in an ischemic-like pain; has not been shown that this is due to diminished arterial blood flow.

Examination: Compartment pressure measurement before and after exercise can be used to diagnose this condition. A preexercise resting pressure higher than 15 mm Hg, a 1-minute postexercise pressure higher than 30 mm Hg, and a 5-minute postexercise pressure higher than 20 mm Hg have been suggested as diagnostic thresholds.

Treatment: Nonoperative treatment is limited to activity modification. Fasciotomy is the only treatment modality that has the potential to allow the athlete to return to play. The procedure has been reported with open, mini-open, and endoscopic techniques. The pitfalls of any technique are incomplete release and nerve damage, most commonly to the superficial peroneal nerve that exits from between the anterior and lateral compartments.

Nerve Entrapment Syndromes

COMMON AND SUPERFICIAL PERONEAL NERVES

Description: Common peroneal nerve is vulnerable at the lateral knee as it passes around the fibular neck and divides at the intramuscular septum between the anterior and lateral compartments of the leg (see Fig. 50-4). Superficial peroneal nerve is most vulnerable at the fascial exit, but it may pass through a fibrous tunnel before exiting to the subcutaneous tissues. It is within that tunnel that the nerve can become fibrotic and suffer tethering and entrapment.

Presentation: Pain that may radiate or be poorly defined, sensory changes on the dorsum of the foot, and weakness of anterior or lateral compartment muscles.

Examination: Tenderness and positive Tinel's sign is more likely to be positive immediately after running when symptoms are present. In severe cases of common peroneal nerve entrapment, transient drop foot and weakness of the anterior and lateral compartments may be presenting complaints. Electrodiagnostic testing is unlikely to be helpful with transient problems.

Treatment: Treatment with rest and modification of training with physical therapy to help with desensitization may be helpful; 4 to 8 weeks may be necessary. Surgical release of nerve compression should only be considered after complex regional pain syndrome has been ruled out, and it may be valuable to have a patient evaluated by a chronic pain specialist before proceeding with surgery.

ENTRAPMENT OF THE POSTERIOR TIBIAL NERVE

Description: Rarely occurs in the leg.

Presentation: Ankle pain and/or foot pain.

Examination: Focal tenderness, neuritic signs, and electromyography (EMG) testing.

Treatment: Nonoperative treatment is similar to other nerve entrapment problems. Avoidance of prominent orthoses in the heel and arch and treatment of pes planus with a medial forefoot posted orthosis may resolve the symptoms. Surgical release can be helpful for pain relief.

TENDON PROBLEMS AROUND THE ANKLE

Achilles Tendon Rupture

Description: Frequently seen in athletes, particularly in males in the 4th decade. Usually occurs in a zone approximately 6 cm above the insertion on the calcaneus; considered a hypovascular region of the tendon (Fig. 50-5). Degeneration of the tendon substance is presumed to be due to multiple small tears.

Mechanism: Rupture of the tendon probably requires 50% of the tendon to be degenerative.

Presentation: Patients feel as if they were struck behind the ankle and may report hearing a loud pop.

Examination: Most reliable finding is loss of resistance to passive dorsiflexion; lack of resistance and the absence of a palpable tendon can be compared to the normal side.

Imaging: MRI helpful in preoperative planning to determine the extent of tendon degeneration in some cases, but this is more applicable to the case of chronic rupture of tendinosis.

Treatment: Continues to be controversial. Satisfactory results can be obtained in some cases with nonoperative treatment. Plantarflexion casting with progressive dorsiflexion and subsequent physical therapy can provide good results without the risks of surgery. The risk of rerupture is between 7% and 10%. In general, operative treatments carry a lower risk of rerupture but the risks of infection, wound-healing problems, and sural nerve injuries are concerning. Surgical treatment ranges from open procedures with complete exposure of the tendon ends and the use of locking sutures to mini-open and percutaneous proce-

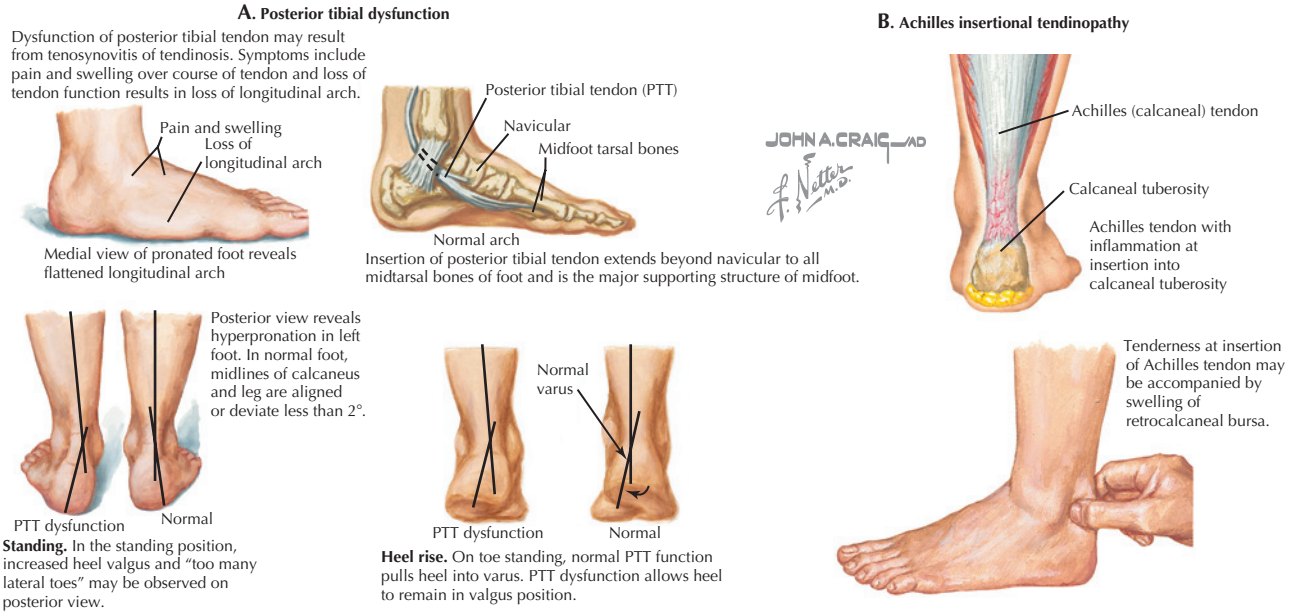


Figure 50-5 Tendon Problems around the Ankle.

dures, with no technique having clear superiority. Postoperative physical therapy and rehabilitation are critical to outcome.

Achilles Tendinopathy (Noninsertional)

Description: Peritendinitis is a thickening and inflammation of the peritendinous tissue and is associated with acute pain; tendinosis is a degenerative condition that occurs within the tendon substance with mucoid degeneration, chondroid metaplasia, and fatty degeneration of teknocytes.

Examination: History of exercise activity and warm-up activity. Physical exam findings typically show thickening of the involved area of the tendon with tenderness and varying degrees of local inflammation. Pain is enhanced with dorsiflexion and placing tension on the tendon (see Fig. 50-5). Biomechanical evaluation for a hypermobile pes planus is important because corrective orthoses and shoe wear to control motion may be of benefit.

Treatment: An improved stretching program, night splints, nonsteroidal anti-inflammatory medications, ice, rest, and even immobilization for a period of 2 to 4 weeks can be helpful. When surgery is considered to debride the involved area of painful degenerative tendon, an MRI can be essential in planning surgery because the extent of degeneration may exceed expectations.

Posterior Tibial Tendonitis and Tears

Description: Uncommon in athletes under 30 years of age. Unlike the adult acquired flat foot, many of these cases of acute tendonitis do not involve a flat foot deformity, although such injuries may indicate the earliest point in the development of this problem. Hypovascular zone of tendon, behind and below the medial malleolus, is thought to be most susceptible to injury (see Fig. 50-5).

Presentation: Focal pain with activity on the medial side of the ankle.

Examination: Palpate the course of the tendon; weakness, pain against resistance, and tenderness along the tendon are strong diagnostic findings. In a young athlete, focal tenderness at the insertion in the presence of a bony prominence may indicate a symptomatic accessory navicular bone.

Imaging: X-rays can confirm the presence, and provide information about the biomechanical alignment of the foot.

Treatment: Tenosynovial inflammation can be treated with rest; an orthosis to unload the posterior tibial tendon may be helpful. When pain is too severe, cast immobilization for a period of 2 to 4 weeks may be necessary. Steroid injections are to be avoided and can lead to rupture. A physical therapy program for posterior tibialis strengthening, and maintaining gastrocsoleus flexibility is key. An orthosis that supports the medial column of the foot is an important consideration during the subsequent several months of recovery.

Flexor Hallucis Longus (FHL) Tendonitis

Description: Reported most commonly in classical ballet dancers, but FHL tendonitis is seen in many athletes.

Examination: Pain behind the ankle can be vague and physical examination findings of focal pain over the FHL, behind the talus, can be directly palpated between the Achilles and the neurovascular bundle as approached from behind. Tenderness is increased with passive dorsiflexion of the hallux and ankle as the tendon is brought under tension. Limitation of the hallux metatarsophalangeal joint motion in this position compared to ankle plantarflexion may also be present.

Imaging: X-rays may reveal a large trigonal process of the posterior talus or an os trigonum. MRI can show edema and fluid around the tendon behind the talus but this diminishes within the fibro-osseous tunnel that the tendon passes into below this.

Treatment: Stretching, rest, night splints, and nonsteroidal anti-inflammatory drugs (NSAIDs) can lead to resolution of symptoms. Proper shoe wear is essential. A period of immobilization may be necessary to calm down any acute inflammation. Where chronic pain is present, or an unstable os trigonum is aggravating the pain, then surgical release of the tendon sheath, and tenolysis, may be necessary. Excision of an associated trigonal process or os trigonum that is also painful or irritating the tendon should be considered. Both open and posterior arthroscopic techniques have been described and the choice of procedure depends on the experience of the surgeon and the location of pathology.

Peroneal Tendon Problems

Description: Either acute or chronic dislocation and tears; occur with inversion injuries or other trauma such as calcaneal fractures.

Presentation: May include recent trauma, but this is not always the case in chronic peroneal tendon problems.

Examination: Some have suggested that the cavovarus foot posture is a risk factor for developing peroneal tendon pathology. Pain in the lateral retromalleolar region that is reproduced with palpation and resisted eversion is typical of peroneal tendon problems, which usually occur behind the lateral malleolus. Testing for transient dislocation or subluxation is essential in evaluation of the integrity of the SPR.

Imaging: MRI is the best modality for assessing pathology of the peroneal tendons behind the lateral malleolus, but the oblique course of the tendons below that make MRI much less reliable. In this region, musculoskeletal ultrasound has been shown to have particular benefit because it can be adjusted to the obliquity of the tendons; ultrasound may also be helpful in evaluating subtle instability because of the dynamic nature of the examination, which can be done with the patient performing a provocative motion for subluxation.

Treatment: If pain is not related to instability, nonoperative treatment may resolve symptoms. Orthoses with a lateral forefoot post can help offload the peroneals in a cavus foot, and rest and brief immobilization may help to initially diminish pain. Gross instability or fixed dislocation upon presentation warrant surgical consideration because there is no other treatment for this condition. Surgery for recalcitrant pain is performed to debride and repair longitudinal tears and in some cases debulk the tendons if tendinosis has led to gross thickening.

ANKLE JOINT PROBLEMS

Osteochondral Lesions of the Talus

Description: Seen in many ankle injuries, but particularly inversion injuries.

Imaging: Ankle x-rays used for diagnosis, but if a suspicion for an intra-articular problem is present, then an alternative imaging study such as a CT scan or MRI should be considered (Fig. 50-6). These studies when combined with intra-articular contrast can also indicate whether the overlying cartilage remains intact. If dye tracks beneath an osteochondral fragment, then it is considered unstable.

Treatment: Lesions that present with chronic ankle pain and are associated with necrosis and cystic changes should be treated by excision and drilling or microfracture of a healthy bleeding bone base. Newer techniques of osteochondral autograft or allograft transplant have become widely used, but may be best used for cases in which debridement and drilling have failed. Autologous cartilage transplantation has become more popular as a salvage option. If an acute osteochondral fracture fragment is large or displaced it should be fixed operatively; small displaced lesions may sometimes be treated by excision alone. Surgery may be possible by arthroscopic or open means. Generally, more anterior lesions can be treated by arthroscopic means, or by arthrotomy without osteotomy. Posterior lesions may require osteotomy of the medial malleolus for medial lesions, or by osteotomy of the Chaput tubercle of the tibia if the lesion is medial. For lesions located very posteriorly, then a prone approach by either arthrotomy or arthroscopy may be alternatives.

Chronic Lateral Ankle Instability

Description: Most commonly injured ligament is the anterior talofibular ligament (ATFL), second most is the CFL. When incomplete healing of these injured ligaments occurs, then mechanical instability of the lateral side of the ankle can result. Should be differentiated from "giving way." True mechanical instability usually occurs while walking or running and can happen when walking over uneven ground, but in severe cases even with walking on level surfaces. "Giving way" occurs first and is followed by pain, rather than the reverse.

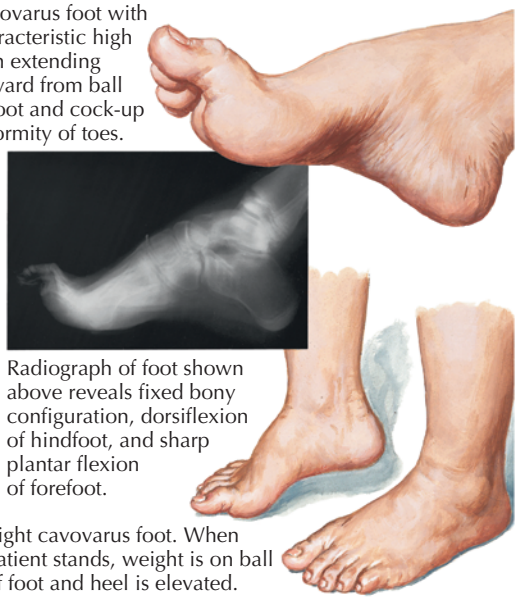
Examination: Usually a unilateral difference from the uninjured side with increased excursion and subluxation of the talus on stress examination; hyperlaxity of the ligaments can lead to a false positive test.

Treatment: Initial treatment is focused on physical therapy for overall strengthening, particularly of the peroneals, balance training, and sometimes functional bracing; physical therapy may be sufficient treatment. An orthosis with a lateral forefoot post may be necessary in conjunction with physical therapy for subtle cavus foot posture contributing to the inversion events (see Fig. 50-6). When nonoperative treatments fail, anatomic repair of the lateral ligaments is usually done.

Soft Tissue Impingement

Anteromedial and Anterolateral Impingement

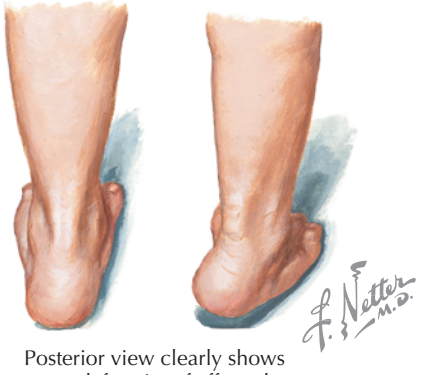
Description: Can occur after inversion injuries. Anterior capsular and synovial tissues tear; laterally the ATFL, the anterior inferior fascicle of the syndesmosis, can tear and become entrapped



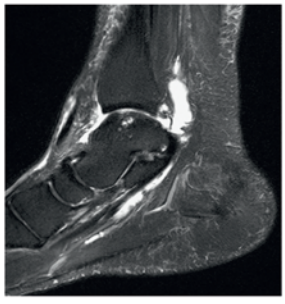
Cavovarus foot with characteristic high arch extending upward from ball of foot and cock-up deformity of toes.

Radiograph of foot shown above reveals fixed bony configuration, dorsiflexion of hindfoot, and sharp plantar flexion of forefoot.

Right cavovarus foot. When patient stands, weight is on ball of foot and heel is elevated.



Posterior view clearly shows varus deformity of affected right foot.



Sagittal T2 MRI image showing osteochondral lesion of the talar dome.

Figure 50-6 Ankle Joint Problems.

as the ankle dorsiflexes; medially the same occurs but the anterior band of the deltoid ligament is torn with the capsule and synovium to create an impingement lesion.

Examination: Physical examination can be very specific in reproducing the pain.

Imaging: MRI and arthrograms may suggest the lesion but are not clinically specific.

Treatment: Excision can be done arthroscopically and there is little indication for open debridement of soft tissue impingement alone by arthrotomy.

Posterior Ankle Soft Tissue Impingement

Description: Not common; caused by a tear of the posterior tibial-fibular ligament, which crosses behind the tibial plafond creating a labral rim.

Mechanism: Can be torn with ankle trauma and the unstable tissue can flip into the posterior ankle and become entrapped with plantarflexion.

Examination: Test for impingement.

Imaging: MRI findings on the sagittal view can suggest the presence of an impingement lesion if the labral rim is seen to be detached in this view.

Treatment: As with other impingement lesions, usually operative excision, either open or by arthroscopic means. Both supine and prone posterior ankle arthroscopy have been described.

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Cartilage Problems in Sports

Kimberly A. Turman, Jennifer A. Hart, and Mark D. Miller

GENERAL PRINCIPLES

Articular Cartilage

- Functions to decrease joint friction and distribute load across the joint; also referred to as hyaline cartilage.
- **Composition:** Water (65% to 80%), collagen (10% to 20%), proteoglycans (10% to 15%), and chondrocytes (5%). Primary collagen is type II collagen (Fig. 51-1).
- **Viability:** Chondrocytes receive oxygen and nutrients via diffusion from synovial fluid. Articular cartilage is avascular.
- **Structure:** Organized into three primary layers—superficial, middle, and deep. The tidemark separates these layers from the calcified cartilage and subchondral bone (see Fig. 51-1).

Fibrocartilage

- Functions in direct tendon and ligament insertions and constitutes the healing tissue of articular cartilage lesions.
- **Composition:** Primary collagen is type I collagen. Fibrocartilage is not as durable as hyaline articular cartilage.

Articular Cartilage Injuries

- Healing is enhanced by motion of the involved joint.
- **Deep lesions:** Cross the tidemark and penetrate the subchondral bone. Vascularity from the subchondral bone promotes fibrocartilage healing.
- **Superficial lesions:** Do not penetrate subchondral bone and therefore have no intrinsic healing potential secondary to the avascular nature of articular cartilage.

Apophysis

- Cartilaginous prominence adjacent to the physis.
- Site of tendon attachments prior to skeletal maturity.
- Secondary ossification centers later develop with eventual osseous fusion.
- **Traction apophysitis:** Repetitive microtrauma caused by the force of pull of the attached tendons results in partial avulsion and inflammation of the apophysis. Common in active children and adolescents. Excessive force may result in avulsion fracture of the apophysis.
- **Osteochondrosis:** General term for disorders affecting one or more ossification centers in children. Encompasses such conditions as traction apophysitis and avascular necrosis.

HISTORY AND PHYSICAL EXAMINATION

History

- History should focus on the nature of injury and symptoms of the involved joint.
- Acute injuries typically result in focal chondral or osteochondral injuries as opposed to the more generalized nature of degenerative lesions.

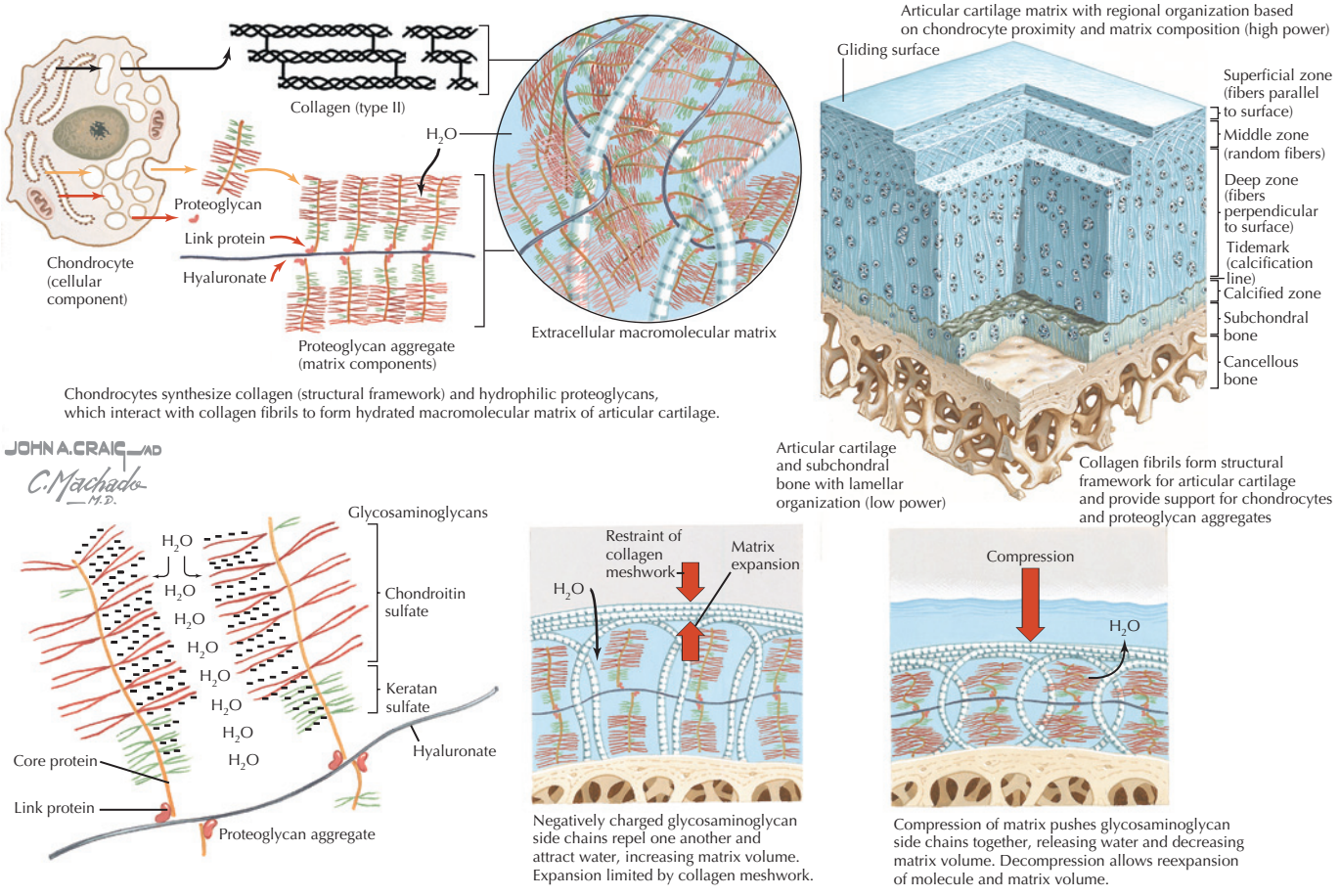


Figure 51-1 Composition and Structure of Articular Cartilage.

- These injuries may not be initially identified, however, and occasionally are only diagnosed after persistence of symptoms.
- Chronic symptoms may also be secondary to the various osteochondroses.

Physical Examination

- Few, if any, physical examination tests are specific to the evaluation of articular cartilage injury.
- A complete clinical evaluation of the involved joint should be conducted.

Imaging Studies

- Imaging studies are essential to the evaluation of cartilage injuries because the history and physical examination are often nonspecific.
- **Plain radiographs:** Useful in ruling out fractures and identifying various osteochondroses and osteochondral lesions such as osteochondritis dissecans. Also beneficial in identifying intra-articular loose bodies, assessing limb alignment, and detecting degenerative changes in the older athletic patient.
- **Computed tomography (CT):** Helpful in assessing cartilage lesions with associated osseous involvement.
- **Magnetic resonance imaging (MRI):** Gold standard for evaluation of articular cartilage. Also identifies commonly associated subchondral edema; focal chondral defects may be underestimated (Table 51-1).

Table 51-1 CARTILAGE SIGNAL INTENSITIES ON MRI

	T1-weighted images	T2-weighted images
Hyaline cartilage	Gray	Gray
Fibrocartilage	Dark	Dark

SPECIFIC INJURIES AND PROBLEMS

Hip

Focal Chondral Defect

Description: Localized, full-thickness loss of articular cartilage with exposed subchondral bone (Fig. 51-2).

Mechanism of injury: Typically a direct blow to the greater trochanter. Forces are transferred to the articular surfaces of the femoral head and acetabulum.

Presentation: History of injury with failure of full recovery, vague hip and groin pain, mechanical symptoms.

Physical examination: Nonspecific.

Differential diagnosis: Avascular necrosis, femoroacetabular impingement, hip dysplasia, degenerative arthritis, labral pathology, femoral neck stress fracture.

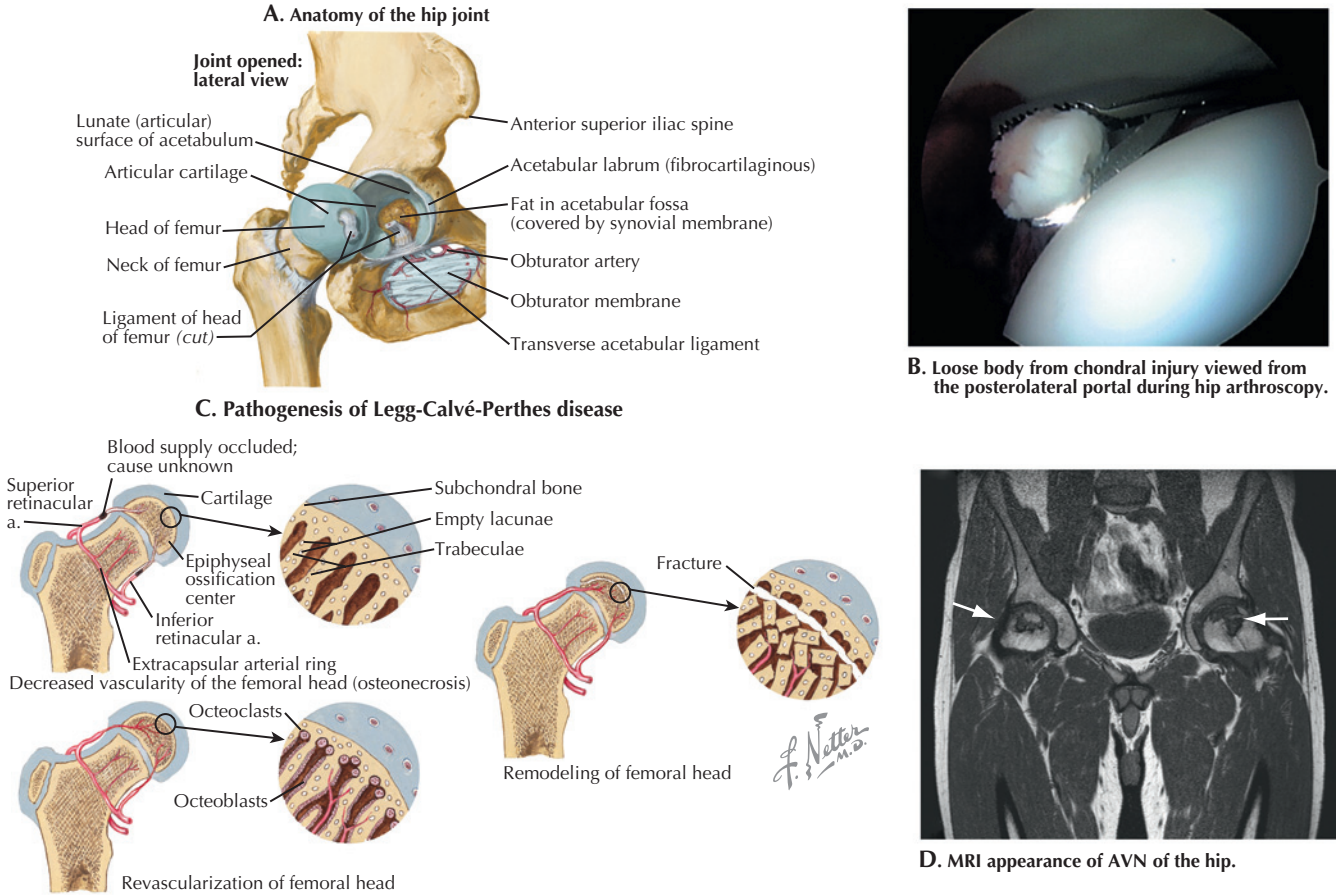


Figure 51-2 Hip Injuries and Problems. (**B,** *Reprinted with permission from Miller M, Cole B: Textbook of Arthroscopy. Philadelphia: Saunders, Elsevier, 2004.* **D,** *Reprinted with permission from Eustace S, Johnston C, O'Bryne J, O'Neill P: Sports Injuries: Examination, Imaging, and Management. Philadelphia: Churchill Livingstone, Elsevier, 2007.)*

Diagnostics: Radiographs helpful in ruling out other conditions in the differential. MRI may demonstrate localized defect or subchondral edema.
Treatment: Arthroscopic chondroplasty, drilling, or microfracture for localized lesions. Excision of unstable or loose fragments to alleviate mechanical symptoms (see Fig. 51-2).
Prognosis and return to play: Return to play when symptoms allow following debridement or excision of fragments. Chondral reparative procedures such as microfracture require partial weight bearing for 6 to 8 weeks. Early range of motion encouraged.

Femoroacetabular Impingement (FAI)

Description: Abnormal contact between the femoral head-neck junction and acetabulum. Results in injury to the articular cartilage and labrum. May be a cause of chronic hip pain in athletes. Two types defined based on the primary location of pathology:
- **Cam type:** Femoral deformity. "Pistol grip" deformity of the femoral neck with decreased head-neck offset, leading to abutment of this region with the normal acetabulum.
- **Pincer type:** Acetabular deformity. Increased acetabular retroversion leading to abutment of the normal femoral head-neck junction on the acetabular rim.

Mechanism of injury: Etiology unknown. Likely developmental component.
Presentation: Anterior hip pain with difficulty squatting, cutting, and starting/stopping.
Physical examination: Pain with flexion, internal rotation, and adduction is a positive impingement test. Range of motion often limited.
Differential diagnosis: Traumatic chondral defect or labral tear, hip dysplasia.
Diagnostics: Radiographs demonstrate the "pistol grip" deformity of the femoral neck or signs of acetabular retroversion such as a crossover or posterior wall sign. MRI may help identify chondral injury and labral tears.
Treatment: Symptomatic management often ineffective as it does not address the underlying structural pathology. Thus, cam type FAI is typically treated with arthroscopic osteoplasty and pincer type FAI with acetabular rim trimming. Chondral lesions are addressed as described previously with chondroplasty, drilling, or microfracture. Labral repair may also be required.
Prognosis and return to play: FAI may be a precursor to osteoarthritis. Arthroscopic osteoplasty, rim trimming, and labral repair require partial weight bearing for 4 weeks. When chondral lesions are addressed, 6 to 8 weeks partial weight bearing is necessary.

Apophyseal Injuries

Description: Several traction apophyses are present in the hip and pelvis of skeletally immature patients; ischial tuberosity is most commonly involved (Table 51-2).
Mechanism of injury: Traction apophysitis secondary to overuse syndromes in adolescent patients; common in running sports.
Presentation: Activity-related pain.
Physical examination: Localized tenderness, discomfort with range of motion and tension on the involved musculotendinous unit.

Table 51-2 TRACTION APOPHYSES OF THE HIP

Traction apophysis	Muscle attachments
Iliac crest	Internal and external obliques
Anterior superior iliac spine (ASIS)	Sartorius and tensor fascia lata
Anterior inferior iliac spine (AIIS)	Rectus femoris
Ischial tuberosity	Hamstrings
Greater trochanter	Abductors
Lesser trochanter	Iliopsoas

Differential diagnosis: Apophyseal avulsion fracture, muscle strain or rupture.
Diagnostics: Radiographs may show irregularity of the involved apophysis and rule out avulsion fractures.
Treatment: Activity modification, local modalities, anti-inflammatory medications.
Prognosis and return to play: Return to play as symptoms allow.

Knee

Focal Chondral Defect

Description: Localized, full-thickness loss of articular cartilage with exposed subchondral bone (Fig. 51-3). Medial femoral condyle most commonly involved, followed by lateral femoral condyle, femoral trochlea, and patellar facets. The proximal tibia articular surface is relatively protected by the overlying meniscus and thus less susceptible to injury.
Mechanism of injury: Generally traumatic injury with shear or rotational forces.
Presentation: History of injury, possible delayed effusion, pain with persistent weight bearing, mechanical symptoms.
Physical examination: Possible effusion, tenderness to palpation.
Differential diagnosis: Meniscus tear, osteochondritis dissecans, degenerative arthritis.
Diagnostics: Not visualized on radiographs. MRI best delineates, though may underestimate involvement and size of focal lesions (see Fig. 51-3).
Treatment: Nonoperative treatment (anti-inflammatory medications, steroid and hyaluronic injections, physical therapy) often of limited benefit. A spectrum of arthroscopic options exists when nonoperative measures fail.
- **Chondroplasty:** Loose chondral flaps debrided with careful preservation of surrounding normal articular cartilage. Provides temporary relief at best, particularly in the athletic population (see Fig. 51-3).
- **Microfracture:** Marrow-stimulation technique. Multiple holes created with awl or drill to penetrate subchondral bone. Bleeding into the defects promotes influx of pluripotent mesenchymal cells with resultant fibrocartilage formation.
- **Osteochondral autograft transfer (OATS):** Transfer plugs of normal osteochondral tissue from non–weight-bearing regions of the knee into the defect. Limited by donor site availability (see Fig. 51-3).
- **Fresh osteochondral allograft transfer:** Defect filled with single osteochondral plug from cadaver bone. Not limited by size of defect and donor site morbidity. Risks disease transmission.
- **Autologous chondrocyte implantation (ACI):** Two-stage procedure. First, chondrocytes are harvested and cultured. After several weeks, the cultured cells are reimplanted under a periosteal patch. The chondrocytes proliferate and produce hyaline-like cartilage. Newer techniques allow implantation on a scaffold without the need for a periosteal patch.

Prognosis and return to play: Non–weight-bearing up to 8 weeks. Early range of motion encouraged and continuous passive motion (CPM) may be beneficial. Best results with restorative procedures (OATS, ACI) that do not rely on fibrocartilage formation. Return to play typically delayed 3 to 6 months. Often longer with ACI.

Osteochondritis Dissecans (OCD)

Description: Localized separation of subchondral bone and overlying articular cartilage (see Fig. 51-3). Classic location is lateral aspect of the medial femoral condyle.
Mechanism of injury: Typically occult trauma.
Presentation: Often vague activity-related pain, possible effusion, mechanical symptoms if fragment becomes loose within the joint.
Physical examination: Effusion, tenderness to palpation.

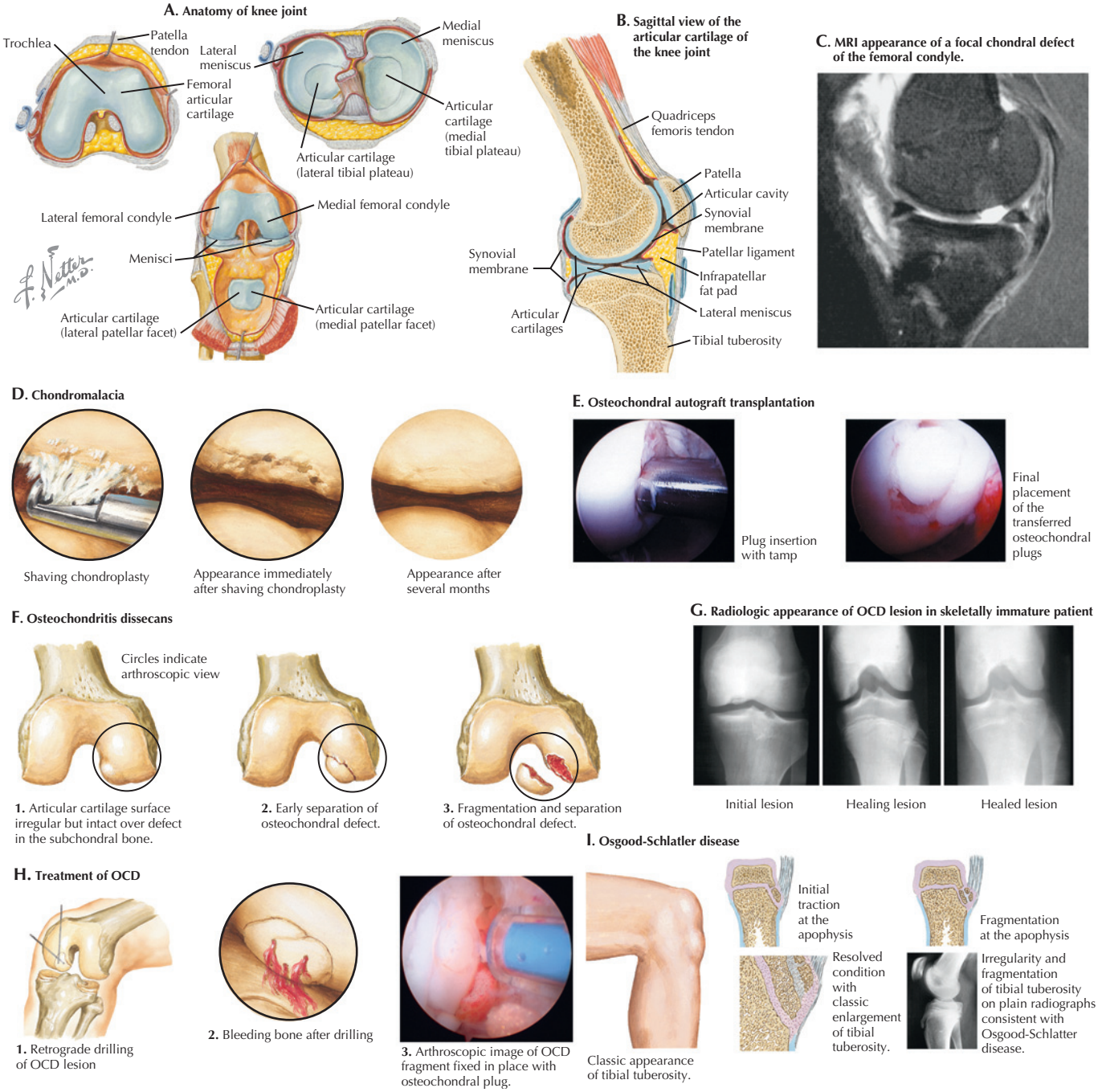


Figure 51-3 Knee Injuries and Problems. *(**C** and **E**, Reprinted with permission from Miller M, Cole B: Textbook of Arthroscopy. Philadelphia: Saunders, Elsevier, 2004.)*

Differential diagnosis: Meniscus tear, focal chondral defect, intra-articular loose body of other etiology (e.g., subsequent to patellar dislocation).

Diagnostics: Tunnel (notch) view best demonstrates OCD lesions on radiographs (see Fig. 51-3). MRI better delineates size and stability of the lesion. Synovial fluid behind the lesion indicates poor healing potential with nonoperative management.

Treatment: Adolescents with open physes have a much better prognosis and may be observed with restricted weight bearing. Surgical treatment recommended for unstable or loose fragments, failure of conservative management, and lesions in skeletally mature patients; consists of debridement, drilling, and internal fixation with addition of bone grafting in select cases (see Fig. 51-3).

Prognosis and return to play: Prognosis best in patients with open physes. Return to play when symptoms abate. With operative fixation of lesions, return to play often delayed 3 to 6 months to allow adequate healing of the lesion.

Osgood-Schlatter Disease and Sinding-Larsen-Johansson Syndrome

Description: Osteochondroses that occur at traction apophyses in the knee of skeletally immature patients. Osgood-Schlatter affects the tibial tuberosity (patellar tendon insertion) and Sinding-Larsen-Johansson the inferior pole of the patella (patellar tendon origin). Most common during periods of rapid growth (see Fig. 51-3).

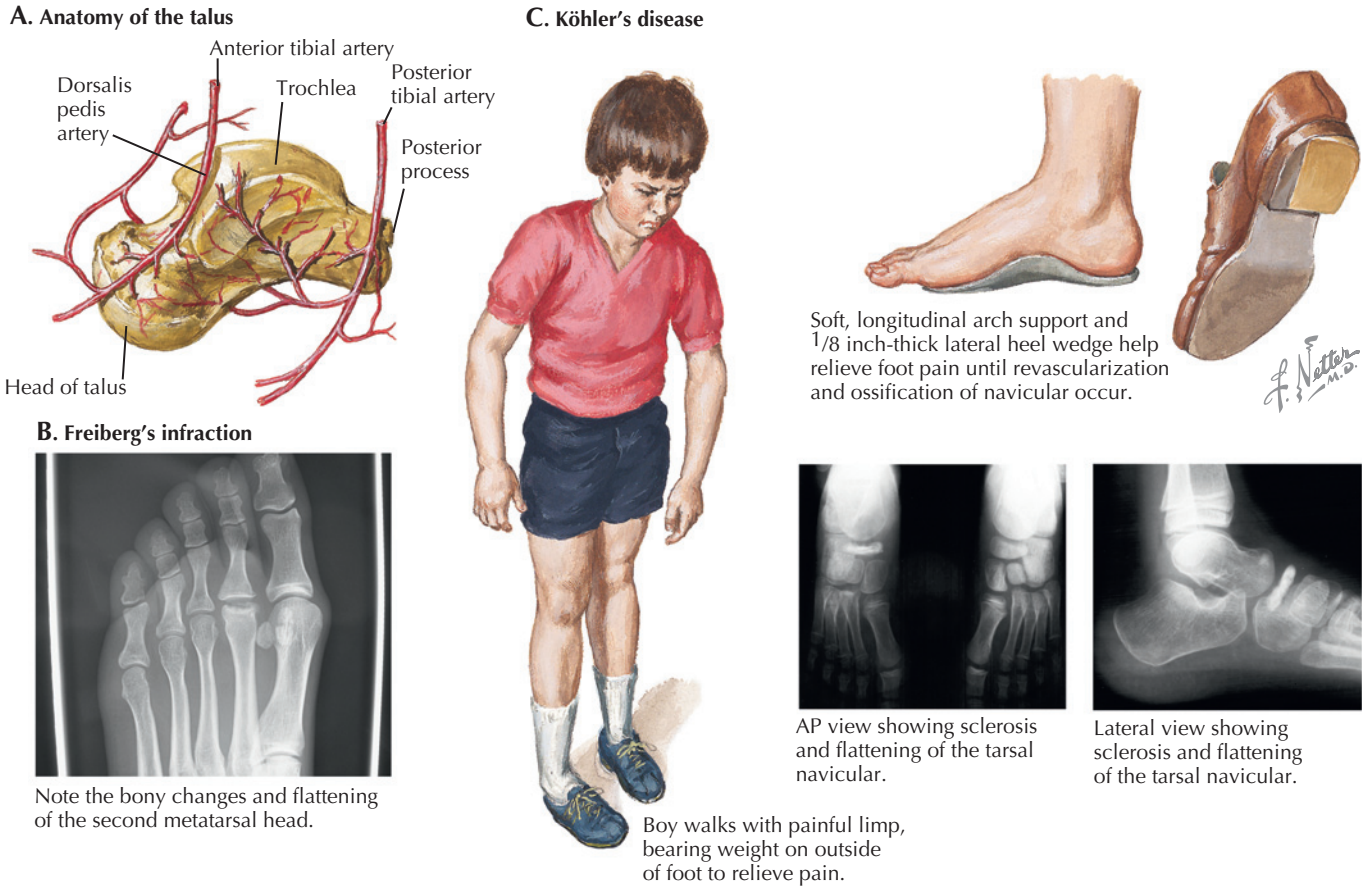


Figure 51-4 Foot and Ankle Injuries and Problems.

Mechanism of injury: Traction apophysitis secondary to mechanical stress from the extensor mechanism. Overuse injuries.
Presentation: Activity-related anterior knee pain.
Physical examination: Localized tenderness, prominent tibial tuberosity, pain with resisted knee extension.
Differential diagnosis: Patellar tendonitis, tibial tubercle physeal injury, patella sleeve fracture with acute injury.
Diagnostics: Radiographs may show fragmentation or irregularity of the tibial tuberosity or inferior patellar pole and associated soft tissue swelling (see Fig. 51-3).
Treatment: Activity modification, hamstring stretching, local modalities, bracing with compression sleeve or Cho-Pat strap, anti-inflammatory medications. Rarely, ossicle excision for recalcitrant cases.
Prognosis and return to play: Self-limited condition. Return to play as symptoms allow.

Foot and Ankle

Osteochondral Lesions of the Talus (OLT)

Description: Focal osteochondral defect involving the dome of the talus (Fig. 51-4). Anterolateral lesions are typically traumatic, shallow, and more likely to be displaced. Posteromedial lesions are usually deeper, atraumatic, and nondisplaced.
Mechanism of injury: Often occur in the setting of ankle fracture or inversion ankle sprain with recurrent lateral ankle instability.
Presentation: Persistent pain following an ankle sprain or healed fracture, possible mechanical symptoms if fragment is unstable or loose within the joint.
Physical examination: Localized ankle tenderness and swelling, may have increased talar tilt and anterior drawer consistent with associated lateral ankle instability.
Differential diagnosis: Recurrent lateral ankle instability, lateral talar process fracture.
Diagnostics: Radiographs often normal, rule out fracture. CT and MRI better to delineate size, depth, and displacement of the lesion.
Treatment: Observation with immobilization in skeletally immature patients and stable lesions in adults. Unstable, displaced, and loose fragments require surgical intervention with options similar to those for osteochondritis dissecans (OCD) lesions of the knee. Occasionally, large fragments may be reduced and fixed if recognized acutely. Other options include arthroscopic excision with curettage and drilling or microfracture to stimulate fibrocartilage formation, osteochondral autograft and allograft transfers, and autologous chondrocyte implantation.
Prognosis and return to play: Acute injuries treated conservatively and all operatively treated ankles require 6 weeks of non–weight-bearing. Return to play is often possible at 3 to 6 months.

Sever's Disease

Description: Osteochondrosis at the insertion of the Achilles tendon on the calcaneal tuberosity in skeletally immature patients. Most common during periods of rapid growth.
Mechanism of injury: Traction phenomenon on the calcaneal apophysis from the strong gastrocsoleus complex. Overuse syndrome.

Presentation: Skeletally immature patient with activity-related posterior heel pain. Bilateral in up to 50% of cases.
Physical examination: Localized tenderness of the posterior heel, pain with medial-lateral compression, tight heel cords.
Differential diagnosis: Achilles tendonitis, retrocalcaneal bursitis, calcaneal stress fracture, plantar fasciitis.
Diagnostics: Radiographs may show irregularity of the calcaneal apophysis.
Treatment: Activity modification, local modalities, heel cord stretching, anti-inflammatory medications, heel cups or lifts.
Prognosis and return to play: Self-limited condition. Return to play as symptoms allow.

Freiberg's Infraction

Description: Osteochondrosis of the second metatarsal head. Most common in adolescent and young adult females.
Mechanism of injury: May be secondary to acute trauma, repetitive microtrauma, or other conditions leading to osteonecrosis and collapse of the subchondral bone.
Presentation: Acute pain (metatarsalgia), worse with weight-bearing activity, second metatarsophalangeal (MTP) stiffness.
Physical examination: Localized tenderness of the second metatarsal head, swelling, limited MTP range of motion.
Differential diagnosis: Metatarsal fracture, stress fracture, metatarsalgia of other etiology (e.g., transfer metatarsalgia secondary to conditions of the neighboring hallux).
Diagnostics: Radiographs normal early. Later show collapse with flattening of the second metatarsal head, possible osteophytes, and articular cartilage destruction (see Fig. 51-4).
Treatment: Protective footwear, orthotics such as metatarsal pads or bars, and anti-inflammatory medications often alleviate symptoms in mild cases. Advanced cases may require immobilization in a walking cast or surgical intervention. Options include synovectomy with core decompression of the metatarsal head, cheilectomy, osteotomy, or partial resection of the metatarsal head.
Prognosis and return to play: Most patients do well with conservative management and may return to play with resolution of symptoms.

Köhler's Disease

Description: Osteonecrosis of the tarsal navicular. Usually presents in childhood.
Mechanism of injury: Idiopathic.
Presentation: Persistent midfoot pain in a young child (see Fig. 51-4). May or may not recall an injury.
Physical examination: Localized tenderness.
Differential diagnosis: Accessory navicular, navicular stress fracture.
Diagnostics: Radiographs show sclerosis and flattening of the navicular (see Fig. 51-4).
Treatment: Immobilization, activity modification.
Prognosis and return to play: Self-limited condition. Navicular reconstitutes over time. Return to activity when symptoms resolve, often 6 to 8 weeks.

Elbow

Osteochondritis Dissecans (OCD)

Description: Osteochondral fragmentation with localized separation of subchondral bone and overlying articular cartilage (Fig. 51-5). Typically involves the capitellum when present in the elbow.
Mechanism of injury: Compressive forces across the lateral elbow may damage the blood supply to the capitellum. Typically a repetitive stress injury. Common in adolescent throwing athletes and gymnasts.
Presentation: Lateral elbow pain, possible mechanical symptoms if the fragment is unstable or loose within the joint.

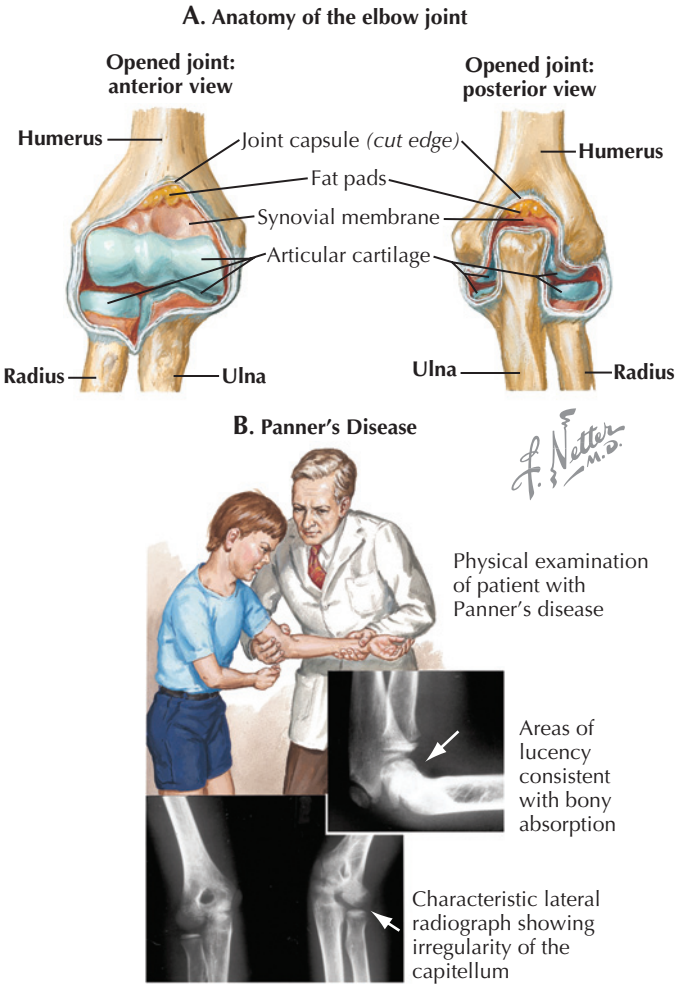
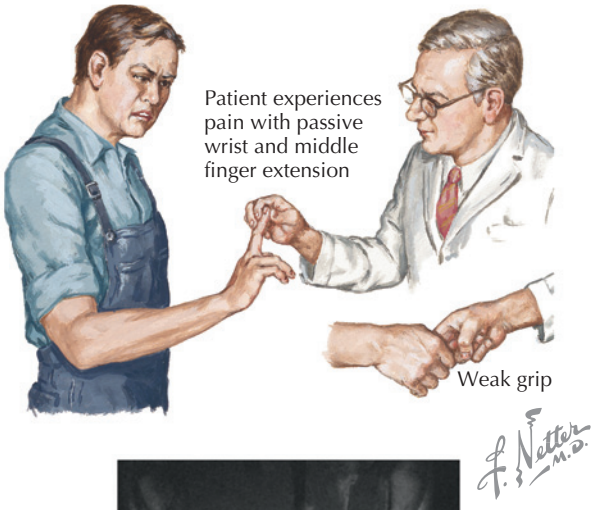


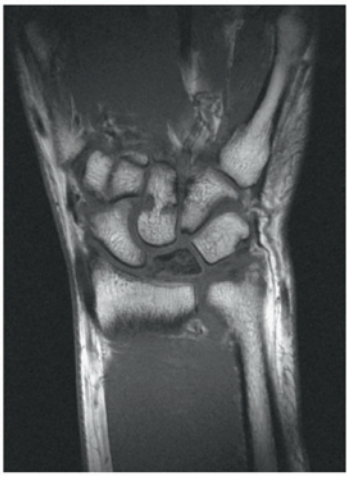
Figure 51-5 Elbow Injuries and Problems.

Physical examination: Localized tenderness, possible effusion, often lack full extension.
Differential diagnosis: Panner's disease, lateral ligament injury or tendinosis, capitellum or radial head fracture, loose body of other etiology (e.g., following elbow dislocation).
Diagnostics: Radiographs, MRI, and CT may all be useful in evaluation and classification of these lesions.
Treatment: Conservative management in adolescents with stable lesions. Large, unstable, or loose fragments typically require surgical intervention. Options include fragment removal with chondroplasty or microfracture versus fixation with drilling and possible bone grafting. Benefits of grafting and fixation over excision and chondroplasty have not been firmly established.
Prognosis and return to play: Return to play varies based on treatment regimen. Conservative management allows return to play when symptoms resolve. Operative management requires a delayed return to play, particularly if fragment reattachment is attempted. Early range of motion and strengthening are encouraged.

Panner's Disease

Description: Osteochondrosis of the capitellum in young children. An avascular segment develops and subsequently revascularizes over time.
Mechanism of injury: Likely lateral compression overuse injury.
Presentation: Activity-related lateral elbow pain. Typically younger than patients with OCD lesions.





MRI appearance of Kienböck's disease.

Figure 51-6 Kienböck's Disease.

Physical examination: Localized tenderness, may lack full extension (see Fig. 51-5).
Differential diagnosis: Capitellum OCD, stress fracture, lateral ligament injury or tendinosis.
Diagnostics: Radiographs often normal, may show irregularity of the capitellum (see Fig. 51-5).
Treatment: Activity modification, local modalities, anti-inflammatory medications.
Prognosis and return to play: Self-limited condition. Return to play when symptoms resolve.

Wrist

Kienboöck's Disease

Description: Avascular necrosis and collapse of the lunate.
Mechanism of injury: Associated with overuse and repetitive compressive loading of the wrist as well as ulnar negative wrist variance.
Presentation: Chronic wrist pain, stiffness, weak grip.
Physical examination: Tenderness and swelling localized over the lunate, pain with passive dorsiflexion of the middle digit, limited wrist dorsiflexion (Fig. 51-6).
Differential diagnosis: Tendonitis, scapholunate dissociation, occult or intraosseous ganglion cyst.
Diagnostics: Radiographs may show sclerosis and collapse of the lunate as well as ulnar negative variance. Advanced stages have associated degenerative changes. MRI may improve detection of early cases (see Fig. 51-6).
Treatment: Conservative measures as initial management. If persistent or worsening symptoms, surgical options include radial shortening versus ulnar lengthening in early stages and limited wrist fusions in advanced cases.
Prognosis and return to play: Return to play as symptoms allow.

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Acute Fractures and Dislocations in Athletes

52

Deana Mercer, Alicia Lacovara, and Robert C. Schenck, Jr.

TRANSPORTATION OF THE ATHLETE WITH FRACTURE OR DISLOCATION

- Transportation of the injured athlete is determined upon primary and secondary evaluations by the first responder, and contingent upon the extent of the injury; must always be executed so that further injury is prevented.
- Planning the mode of transport and necessary equipment can help ensure that proper technique is used (see Chapter 4, Sideline Preparedness and Emergencies on the Field).
- An athlete with a suspected spinal injury (fracture or otherwise) should not be moved by anyone other than certified medical personnel and an ambulance should be contacted immediately (see Chapter 40, Neck Injuries).
- If a serious fracture, such as tibia, femur, or pelvis fracture, is suspected, the safest and most efficient mode of transportation for a shorter distance is by stretcher that is taken onto a portable vehicle or the ambulance.
- Suspected extremity fractures must be immobilized prior to transport (Fig. 52-1).

EMERGENCY SPLINT EQUIPMENT

- Ability to take care of an acute fracture in an athlete is directly related to one's level of preparedness.
- Timely application of a splint to a fractured extremity lessens the overall surrounding soft tissue injury and allows for safe transportation of the athlete for definitive care.
- Every sports medicine physician and trainer must carry lightweight, easy-to-apply splint material in their sport medicine bag (see Chapter 4, Sideline Preparedness and Emergencies).
- **Basic mobile splint kit for medicine bag** (Table 52-1):
  - Arm sling/shoulder immobilizer (medium and large sizes)
  - Alumafoam padded splints (several lengths that can be cut to size)
  - Structural aluminum malleable (SAM) splint
  - Knee immobilizer (universal size)
  - Webril (cast padding) and ace wraps (4 and 6 inch)
  - Plaster or fiberglass splinting material for self-made splints (prepackaged splints are available)
- **Advanced facility kit** (available at all high-volume sports complexes or via emergency medical services [EMS]):
  - Extremity vacuum splints or air splints (total body kits)
  - Cervical collar
  - Spine board
  - Femoral traction splint
  - Crutches

GENERAL PRINCIPLES

General Overview

- Braces or orthoses stop or limit range of motion, facilitate movement, or guide a joint through an arc of motion.
- Splints are used to immobilize and position one or several joints (see Fig. 52-1).

Table 52-1 SPLINTS USED PER INJURY LOCATION

Clavicle and acromioclavicular (AC) joint	Arm sling
Shoulder and proximal humerus	Arm sling and 6-inch ace wrap swathe
Distal humerus and elbow	Air or vacuum splint
Forearm and wrist	Fiberglass/plaster splint, structural aluminum malleable (SAM) splint, prefabricated orthosis
Hand	Buddy taping, Alumafoam splint
Spine	Cervical collar and spine board
Hip and femur	Femoral traction splint
Knee and proximal tibia	Knee immobilizer
Tibia shaft, ankle, and foot	Air or vacuum splint

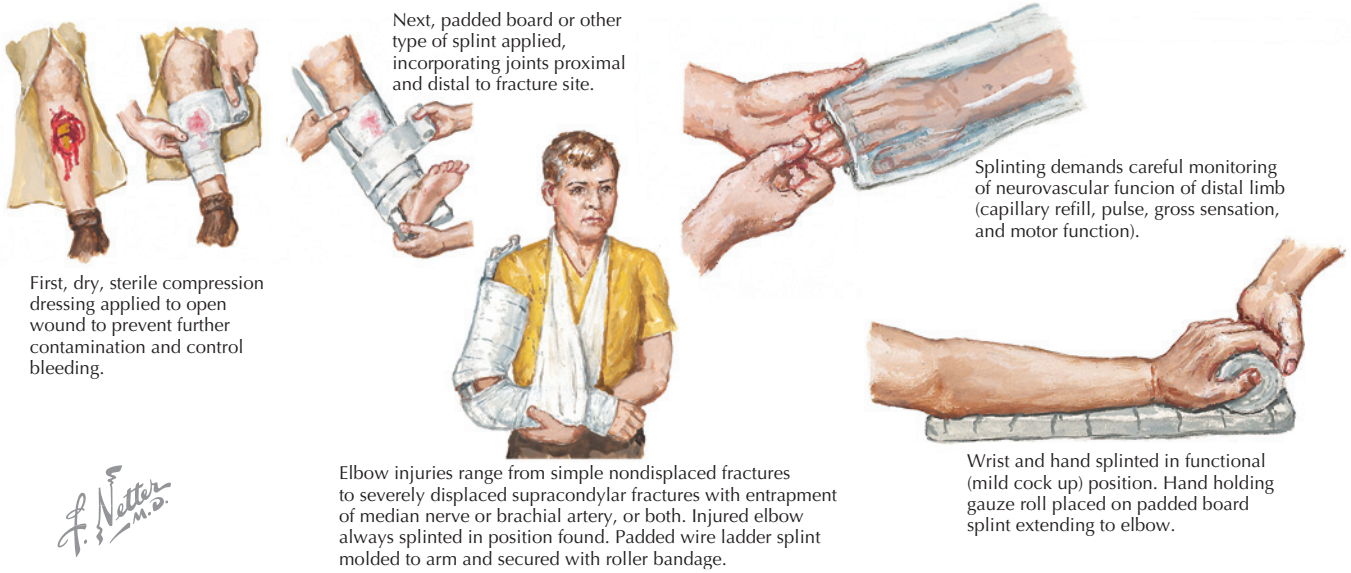

Figure 52-1 Prehospital Care of Fractures.

- Splints and braces are prescribed after a fracture to protect a partially healed fracture, or to prevent the pain that occurs with motion.
- A cast is a stress-sharing device, allowing callus formation and rapid secondary bone healing. Both joints, above and below the fracture, must be immobilized to prevent rotation and translation of the fracture fragments.

Splint Treatment

- Splints are beneficial because they provide some stabilization at the fracture site but may be removed for rehabilitation treatment.
- After a cast is first removed, splints are frequently used during activity or at night to reduce pain and discomfort.
- Joint stiffness is very common after removal of a cast, and resolves after weeks to months of rehabilitation.
- Immobilization of the joints above and below the fracture site often leads to stiffening and the need for a prolonged rehabilitation program.
- A cast brace can provide partial immobilization while allowing some range of motion and weight bearing on a limb.
- Once a fracture achieves some stability with callus formation, the cast can be replaced in the form of a hinged splint or brace allowing motion of the joints proximal and distal to the fracture, without compromising the support at the fracture site.

Initial Evaluation

- Immediately after presentation with a fracture or dislocation, neurovascular and circulatory status must be evaluated.
- Temporary immobilization measures should be taken as the patient is transported to the hospital (see Fig. 52-1).
- The mechanism of injury can provide clues as to what type of injury has occurred.
- In all upper extremity fractures, assess the following:
  - Open or closed fracture
  - Patient complaints of pain, swelling, or paresthesia
  - Finger capillary refill and sensation
  - Active and passive range of motion of wrist and/or digits
  - Radial and/or ulnar nerve function and possible compression
- In all lower extremity fractures, assess the following:
  - Open or closed fracture
  - Patient complaints of pain, swelling, or paresthesia
  - Toe capillary refill and sensation, with pink color
  - Active and passive range of motion of all metatarsophalangeal and interphalangeal joints
  - Nerve function and possible compression

UPPER EXTREMITY INJURIES

Humeral Diaphysis or Midshaft Fractures

- Radial nerve function should be assessed. If it is not intact at the time of injury, a cock-up wrist splint should be added to avoid stiffness of the wrist and fingers during recovery of radial nerve function.
- A coaptation splint is used for treatment. It is a stress-sharing device using the tissue forces and the soft tissue integrity around the fracture to stabilize the fracture (Fig. 52-2). The splint should be well padded to avoid chaffing and molded appropriately to maintain fracture reduction.
- It should be stressed to the patient that an upright body positioning will aid in fracture alignment.

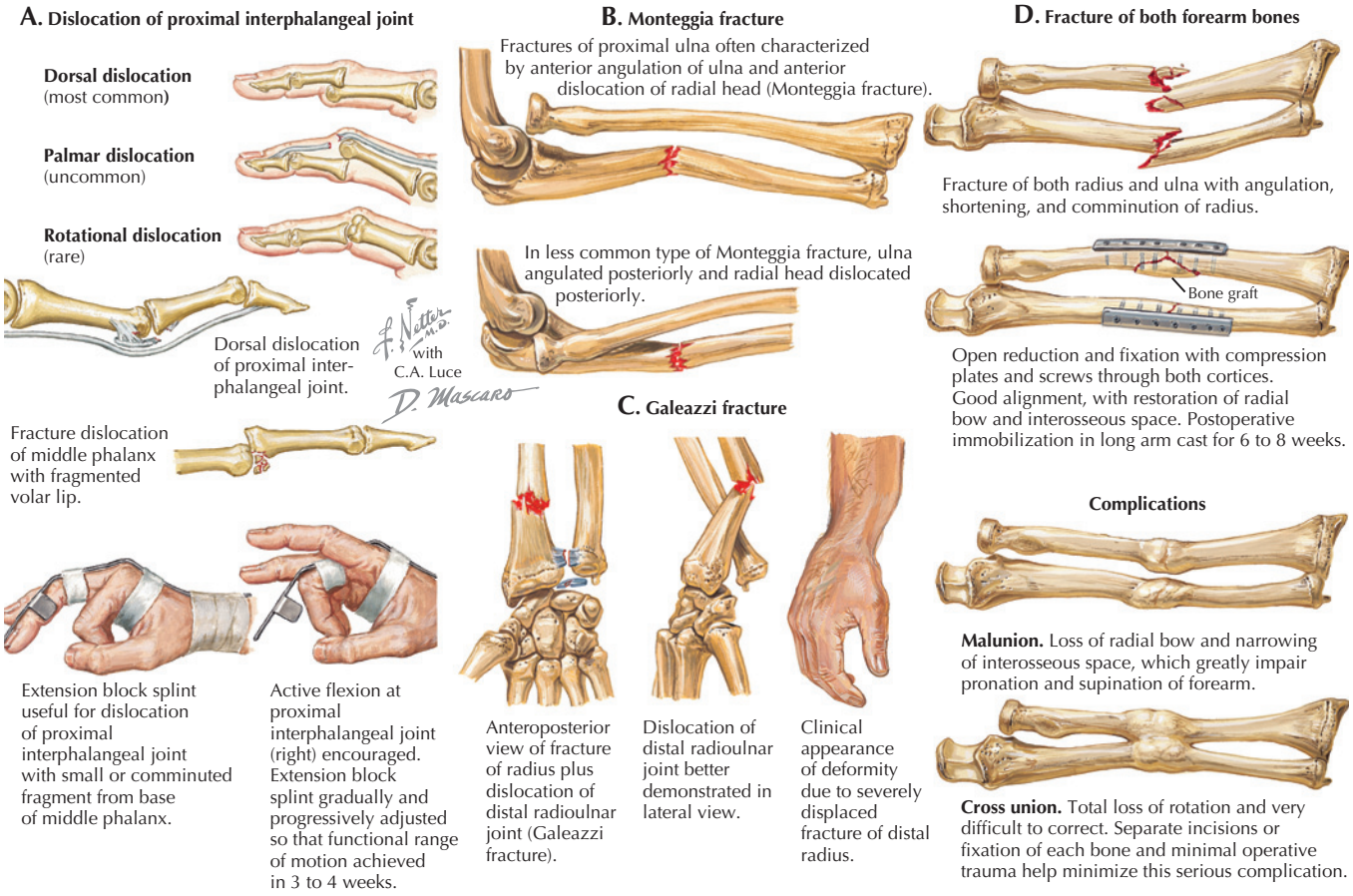

Figure 52-2 Upper Extremity Fractures and Dislocations.

Distal Humerus Fractures

- Fractures that have minimal displacement and are in near anatomical alignment can be treated with casting or splinting.
- Cast or splint treatment of these injuries should allow no motion at the elbow.
- The cast should be trimmed to the proximal palmar crease to allow full range of motion of the metacarpophalangeal joints.
- There should be no supination or pronation, and internal and external rotation movements of the shoulder should be avoided, because they can lead to fracture displacement.

Olecranon Fractures

- Minimally displaced fractures of the olecranon with less than 2 mm displacement and intact elbow extensor mechanism can be treated with cast or splint immobilization.
- The cast should be adequately padded to avoid skin breakdown at the edges of the cast or splint.
- The cast should extend to the distal palmar crease volarly and to the metacarpophalangeal joints dorsally to allow for full motion at the interphalangeal and metacarpophalangeal joints.
- Once there is evidence of healing, the elbow should be mobilized to prevent stiffness. An elbow-hinged brace can protect the fracture but maintain motion of the elbow. Often, some terminal extension is lost but functional range of motion is maintained.
- Displaced fractures of the olecranon require open reduction with internal fixation followed by early elbow range of motion to minimize the development of elbow stiffness.

Radial Head Fractures

- Minimally displaced fractures can be treated in a sling with early elbow range of motion.
- If there is significant swelling, a brace can be applied for a short period of time to allow for tissue rest, followed by initiation of early range of motion.
- For fractures requiring open reduction with fixation, rigid immobilization is needed, followed by bracing during initial mobilization.
- A hinged elbow brace is typically used to allow protection of the fixation during initiation of early range of motion.

Distal Radius Fracture

- Minimally displaced fractures can be treated with a long arm cast followed by a short arm cast (see Fig. 52-2).
- Rigid immobilization for 6 weeks followed by a removable gauntlet splint is recommended.
- Displaced fractures require reduction prior to splinting and should be immobilized in a long arm splint with the wrist in slight ulnar deviation.

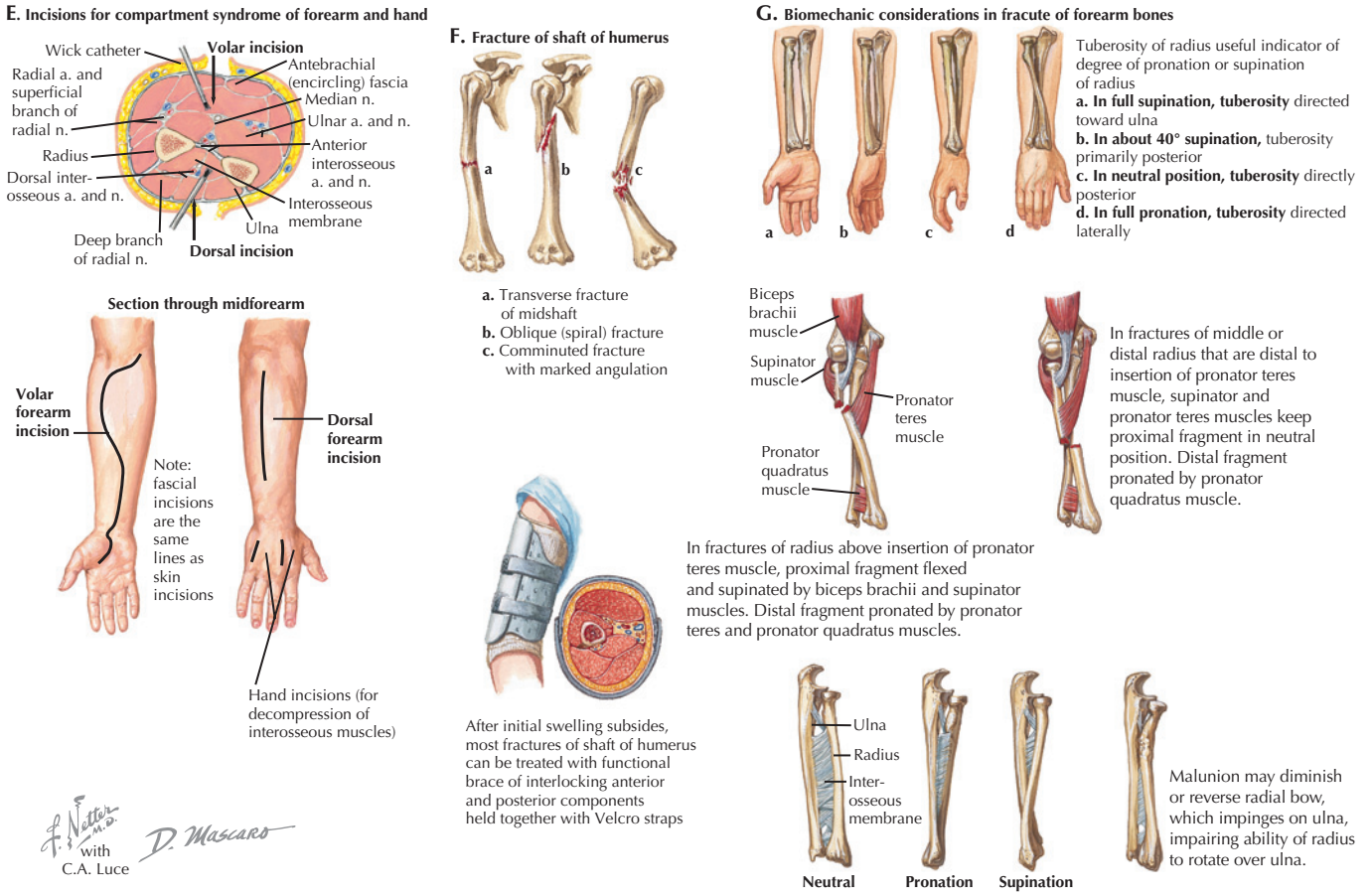

Figure 52-2, Cont'd Upper Extremity Fractures and Dislocations.

- The cast should be trimmed to the proximal palmar crease volarly and the metacarpophalangeal prominences dorsally to allow for free finger movement. The cast should be trimmed to allow the thumb full opposition with the small finger.
- Distal fractures requiring open reduction with internal fixation require long arm splint in the immediate postoperative period, followed by short arm casting or removable gauntlet splint depending on the pattern of injury (see Fig. 52-2).

Scaphoid Fractures

- For nondisplaced or minimally displaced fractures, thumb spica cast immobilization is recommended. The wrist should be in a neutral position relative to flexion, extension, and radial deviation.
- Fractures requiring open reduction should be immobilized in a thumb spica splint in the immediate postoperative period, followed by a thumb spica cast.

Metacarpal and Phalangeal Fractures

- Dislocations of the proximal interphalangeal (PIP) joint should be immobilized using static dorsal extension splinting.
- It is important to secure the proximal phalanx to the splint to ensure that the PIP joint does not extend when the patient flexes the metaphalangeal joint.
- Metacarpal neck and shaft fractures are best treated with a cast or splint.
- The splint is applied with the hand in "safe" position with the wrist in slight extension, the metacarpalphalangeal joint flexed, and the proximal and distal interphalangeal joints in extension. Displaced fractures require reduction prior to splinting.
- Phalanx fractures that are minimally displaced can be treated with "buddy taping," in which the fractured finger is taped to an adjacent finger. The adjacent finger functions as a splint but allows continued range of motion of the metacarpalphalangeal joint. Displaced fractures require reduction prior to splinting.

Clavicle Fractures

Description: Account for approximately 5% of all fractures; 85% involve the middle third of the clavicle.

Mechanism of injury: Most result from a fall onto the ipsilateral shoulder.

Initial on-field management: The fracture displacement and pain level is significant and a clinical diagnosis can be made. The athlete can be assisted off the field with the arm held at his or her side. Initial management with a sling with or without a swathe is sufficient. A 6-inch ace wrap can be used for a swathe and improves comfort by supporting the elbow and immobilizing the arm to the body.

Evaluation:

- **Inspection:** Look for obvious deformity and inspect for skin breakdown or tinting.
- **Palpation:** Check for neck, sternoclavicular joint, midclavicle, acromioclavicular joint, scapula, and proximal humerus tenderness.
- **Neurovascular exam:** Evaluate the sensory motor exam of the upper extremity including axillary nerve sensation on the lateral upper arm.

X-rays: An anteroposterior (AP) and an AP with 20 degrees of cephalic tilt are sufficient.

Treatment: Treatment for most closed clavicle fractures is nonoperative with application of a figure-of-eight collar or arm sling. Indications for operative treatment include skin compromise secondary to severe displacement, open fractures, and a floating shoulder with neurovascular compromise. Recent literature has shown improved result with open reduction and internal fixation in young active individuals with middle one-third fractures with severe shortening. Shortening and overlap greater than 2 centimeters in an active individual is a relative indication for surgical intervention. The method of fracture stabilization is generally with a contoured plate or intramedullary device.

Prognosis and return to play: Results are good in the majority with return to sporting type activity by 6 to 12 weeks.

Humeral Shaft Fractures

Overview: Uncommon, accounting for approximately 1% of fractures in trauma registries (see Fig. 52-2).

Mechanism of injury: More frequently from a fall onto the involved extremity but can occur from a direct blow.

Presentation: Painful, unstable extremity with swelling and bruising.

Physical exam: Gentle palpation of the shaft of the humerus should be performed, noting any areas of malalignment and increased pain.

Associated injuries: Associated nerve injuries are relatively common and should be documented; radial nerve injuries are most common. Most common fracture type associated with a nerve injury is a transverse mid-diaphyseal fracture. Document a motor neurologic examination of the axillary, musculocutaneous, radial, median, and ulnar nerves. Skin should also be examined to rule out an open fracture. Shoulder and elbow joint should be examined and imaged.

Diagnostics: Anteroposterior and lateral radiographs of the entire humerus should be obtained. Additionally, radiographs of the shoulder and elbow should also be taken to help evaluate for associated pathology.

Treatment: In most cases, nonoperative management is appropriate. Treat with functional bracing and by allowing active flexion and extension of the elbow during the healing process to prevent elbow stiffness; motion helps reduce the fracture and aids in the healing process. Patients who have sustained significant nerve injuries that prevent active range of motion are not good candidates for functional bracing. The humerus can heal with angulation of up to 30 degrees and shortening of 2 to 3 cm without functional problems in most patients. However, the functional needs of a high-performance athlete may be different. If functional bracing is elected to treat an athlete, great care should be taken to follow the healing process and evaluate the alignment. If alignment is not maintained in the dominant arm of a skill athlete, consideration should be given to surgical stabilization of the fracture. Surgical treatment usually involves either plating or use of an intramedullary nail (see Fig. 52-2). Shoulder pain is the most frequent complication following intramedullary nailing. It is important to warn an athlete of the risk of shoulder pain if intramedullary nailing is elected.

Prognosis and return to play: Prognosis for healing and good functional return following isolated humerus shaft fractures is good. Patients who have associated nerve injuries or shoulder dislocations have a less favorable prognosis, but still may be able to return to competition if the nerve injury resolves.

Forearm Fractures

Description: Forearm injuries represent about 5% of all fractures and include isolated ulna fractures, and fractures of both the radius and ulna. Monteggia and Galeazzi fractures involve the proximal and distal radioulnar joints, respectively (see Fig. 52-2). Monteggia fractures involve fracture of the ulna with dislocation of the radial head. Galeazzi fractures involve fracture of the radius shaft with injury to the distal radial ulnar joint.

Mechanism of injury: Isolated ulna fractures are seen in contact sports athletes resulting from a direct blow to the arm (see Fig. 52-2). Fracture of both the radius and ulna is also seen in contact athletes; fractures may be open or closed.

Initial on-field management: Injured extremity should be stabilized with a splint and the athlete taken off the field for further evaluation.

Evaluation:

- **Inspection:** Determine if the fracture is open or closed.
- **Palpation:** Assess both the elbow and wrist of the injured forearm to be sure there is no damage to either the proximal or distal radioulnar joint.
- **Neurovascular exam:** Check vascular status of the arm by palpation of the radial pulse in addition to assessment of the capillary refill at the nail bed of each digit. The radial nerve sensation is tested by lightly touching the dorsum of the first web space. The ulnar nerve sensation is tested by lightly touching the small finger. The median nerve sensation is tested by lightly touching the index finger.

X-rays: Should include two views of the forearm, three views of the elbow, and three views of the wrist.

Treatment: In skeletally mature athletes, operative treatment is recommended to restore anatomical alignment in **displaced radius and ulna shaft fractures.** The musculature of the proximal forearm makes maintenance of closed reduction difficult (see Fig. 52-2). For **undisplaced fractures of the ulna,** management of injury is symptomatic and consists of prohibition of sporting activities until clinical and radiographic evidence of fracture union exists; bracing or casting used. Serial radiographs done to ensure no further displacement and to monitor eventual healing. **Displaced fractures** may require surgical fixation. **Open fractures** require urgent treatment; the wound should be protected by a sterile dressing and the patient should be immediately taken to the operating room following all diagnostic tests.

Return to play: In general, 4 to 6 weeks.

Compartment Syndrome in Forearm

Description: An increase in pressure in a muscle compartment leading to damage of the structures within the compartment including muscles and nerves.

Mechanism of injury: Typically the result of high-energy impact (either forceful or crushing) to the forearm, and occurs more frequently in the volar compartment. In athletes with forearm injuries, a tense swelling may suggest compartment syndrome.

Evaluation:

- **Inspection:** The forearm should be evaluated for swelling and bruising.
- **Palpation:** Assess the compartment compressibility by gently pressing against the forearm compartments and comparing it to the contralateral side. Tense, noncompressible compartments or severe pain with passive motion of the fingers out of proportion to the injury should increase suspicion for compartment syndrome.
- **Neurovascular exam:** Check vascular status of the arm by palpation of the radial pulse in addition to assessment of the capillary refill at the nail bed of each digit. The radial nerve sensation is tested by lightly touching the dorsum of the first web space. The ulnar nerve sensation is tested by lightly touching the small finger. The median nerve sensation is tested by lightly touching the index finger.

X-rays: Two views of the forearm should be obtained to ensure there is no fracture.

Treatment: If the diagnosis of compartment syndrome is made, immediate fasciotomies should be performed (see Fig. 52-2).

LOWER EXTREMITY INJURIES

Tibial Plafond Fractures

- Long leg casting and closed reduction is only suitable for fractures with minimal displacement and no damage of the articular joint surface.
- Comminuted fractures can have soft tissue damage that may delay surgery, requiring temporary immobilization in a bulky cotton plaster splint prior to operative fixation. The splint allows for soft tissue rest.

Foot Fractures

- Fractures involving the forefoot, midfoot, or hindfoot can be immobilized in a short leg splint until swelling subsides.
- Those fractures that are minimally displaced can be treated with a short leg cast after swelling subsides.
- Fractures of the foot phalanges can be treated with a hard-soled shoe and "buddy taping" (Fig. 52-3).

Hip Fractures and Dislocations

Description: Intertrochanteric hip fractures and femoral neck fractures are rare in athletes; hip dislocation and femoral head fractures are more common injuries in athletes and younger individuals (see Fig. 52-3). Fracture dislocation of the hip in a young, athletic patient should be viewed as a surgical emergency. There is a correlation between the time a hip remains dislocated and the development of avascular necrosis (AVN) of the femoral head.

Presentation: Patients with posterior hip dislocations present with shortened extremity with the hip flexed, adducted, and internally rotated; those with anterior hip dislocation present with shortened extremity with the hip flexed, abducted, and externally rotated.

Physical exam: No attempt should be made at reduction of the hip prior to obtaining good quality radiographs. A neurologic examination should be documented prior to attempts at reduction.

Diagnostics: Radiographs are key and should be obtained prior to attempts at reduction, to prevent severe displacement of associated minimally displaced fractures. The patient should have AP and lateral views of the hip joint, as well as Judet views of the acetabulum. It is important to know whether there is an associated fracture, and whether that fracture is displaced. The radiographs can also provide information regarding whether there is a large fracture fragment in the joint that may prevent reduction of the dislocation.

Treatment: After obtaining radiographs and ruling out a hip fracture, closed reduction of the hip can be attempted under conscious sedation. It is important to have complete relaxation of the hip muscles prior to an attempt at reduction. If the patient has an associated femoral neck fracture, he or she should be taken to the operating room expeditiously. If there is an associated acetabulum fracture, it is reasonable to attempt a closed reduction as long as there are no large bone fragments in the joint that may block reduction. An attempt at a closed reduction should be made by an experienced physician. If that fails, the patient should be taken to the operating room for a reduction under general anesthesia. There are three primary methods for achieving closed reduction of a posterior hip dislocation.

- **Bigelow maneuver:** This is accomplished by having an assistant provide downward pressure on the anterior-superior iliac spine while the physician pulls in-line traction, flexes the hip 90 degrees, and applies internal rotation and adduction until a reduction is achieved.
- **Allis maneuver:** In this case, an assistant stabilizes the pelvis while the physician pulls in-line traction, flexes the hip to 90 degrees, and gently alternates between internal and external rotation in an attempt to reduce the hip.
- **Stimson maneuver:** In this case, the patient is placed prone with the affected extremity hanging off the end of the exam table. Both the hip and knee are flexed 90 degrees, and a downward force is applied to the calf (see Fig. 52-3).
- Anterior hip dislocations are reduced with the patient supine using traction and counter-traction until a reduction is obtained (see Fig. 52-3).

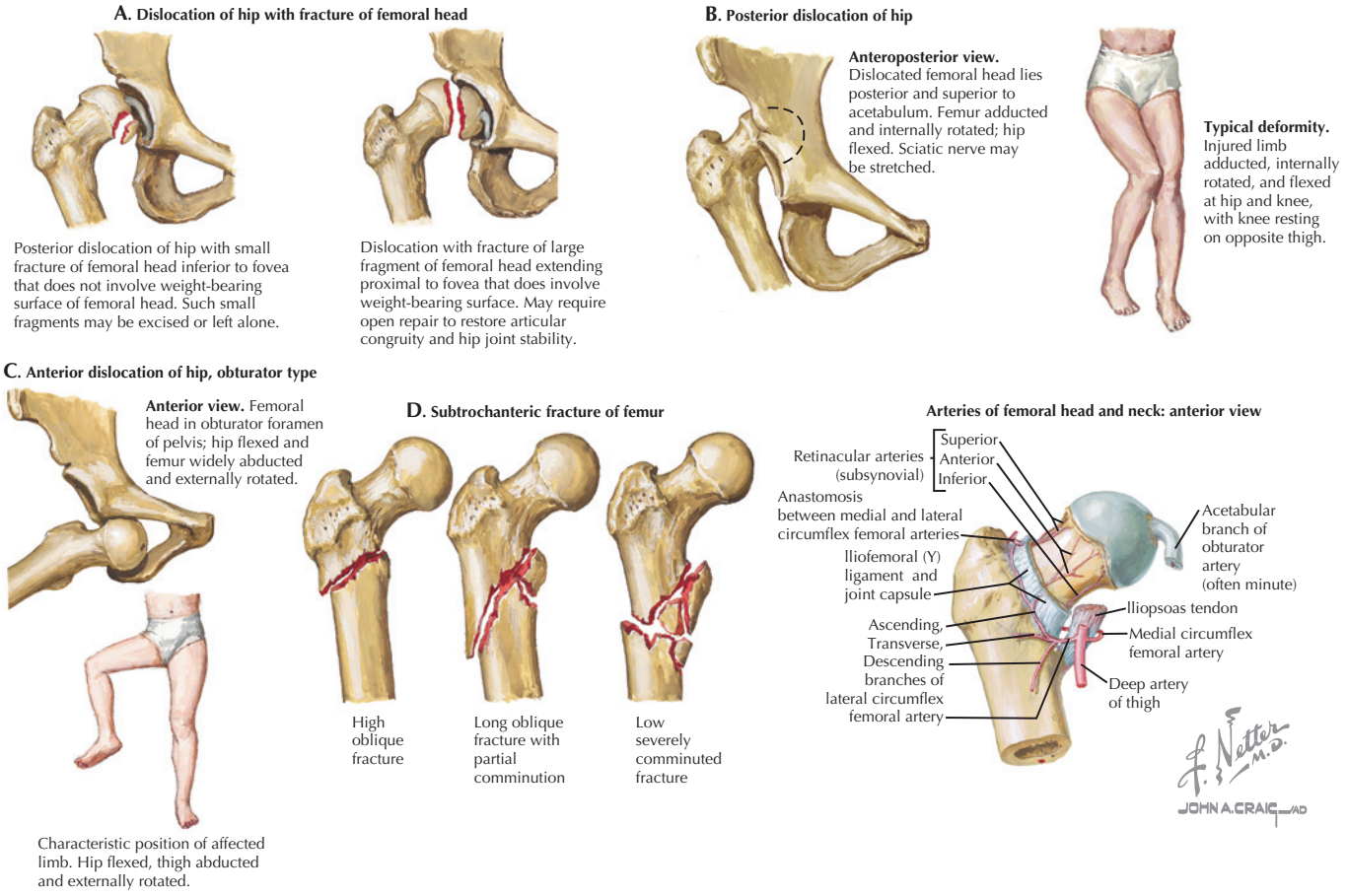

Figure 52-3 Lower Extremity Fractures and Dislocations.

Prognosis and return to play: Long-term results following hip dislocations are poor, even if reduction is prompt and appropriate. Athletes who sustain a dislocation of the hip with or without a fracture should be counseled that they may have sustained a career-ending injury.

Femur Fractures

Description: Uncommon sports injury; requires high-energy trauma.

Associated injuries: Assess athletes carefully for other injuries. Common systemic injuries include head, chest, and abdominal injuries. The most important associated skeletal injury is a fracture of the femoral neck. It is critical to diagnose these injuries and avoid any additional displacement of the neck fracture; a displaced femoral neck fracture puts the blood supply to the femoral head at risk, and may lead to avascular necrosis of the femoral head (see Fig. 52-3).

Presentation: Severe pain and inability to bear any weight on the involved leg.

Physical exam: Displaced femur fractures present with malalignment of the limb and severe pain. Document the neurologic and vascular status of the leg. Minimize movement of the injured extremity until radiographs can be obtained. Imaging should include views of the femoral neck. Evaluate for skin lacerations, which can indicate open fractures and assess for compartment syndrome. If a compartment syndrome is developing, this should raise concern for vascular injury.

Diagnostics: Plain radiographs; AP and lateral views are generally adequate to characterize the injury. If the femoral neck is not clear in hip radiographs, remove splints and an internal rotation view of the femoral neck should be obtained. If the patient has an asymmetric pulse, then emergent arteriogram or CT angiogram should be obtained.

Treatment: Initial treatment should concentrate on stabilization of the extremity (see Fig. 52-3). Gentle longitudinal traction should be applied, and then the splint should be applied. A hare traction splint can help provide pain relief. Definitive treatment of femur fractures in general requires surgery. Intramedullary nails are the most commonly used implants, whether the fracture is in the subtrochanteric, midshaft, or supracondylar regions of the femur. In some instances, fixation with a plate and screws may be indicated (see Fig. 52-3).

Prognosis and return to play: Long-term prognosis is good. Union of the fracture occurs in 96% to 98% of femur fractures, with most uniting within 3 to 4 months. Athletic activities should be deferred for 4 to 6 months. Abductor muscle weakness and dysfunction following antegrade intramedullary nailing is common. Knee pain is common following retrograde femoral nailing. Physical therapy should concentrate on the rehabilitation of the muscles around the hip, particularly the abductors.

Knee Dislocation

Description: Occurs when the tibia is no longer in contact with the femur; associated with ligament, nerve, and vascular injury (see Fig. 52-3). Seen in contact sports such as football.

Mechanism of injury: Result of a large force applied across the knee joint; knee joint is disrupted with dislocation of the tibiofemoral articulation with associated multiligament injury.

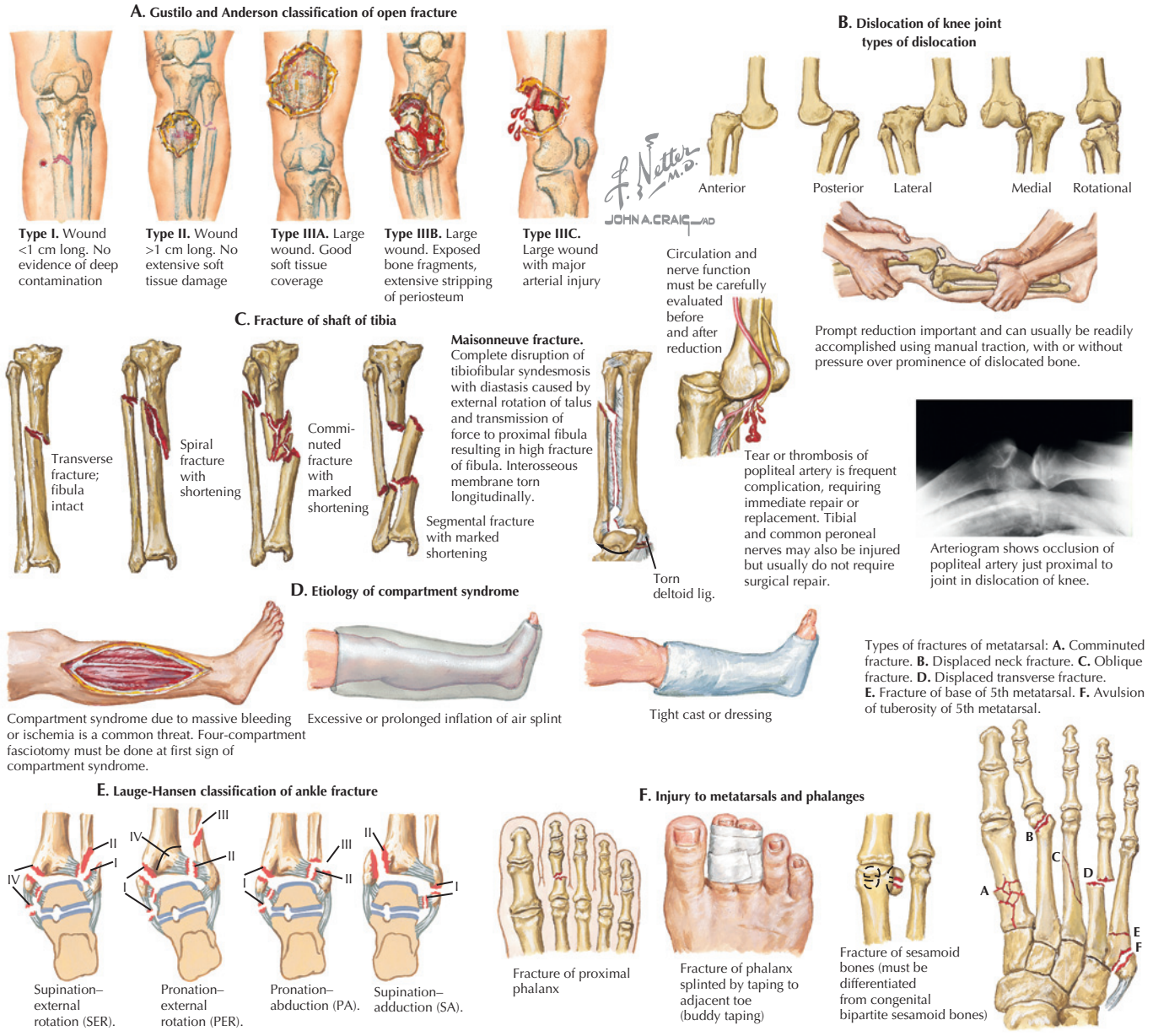

Figure 52-3, Cont'd Lower Extremity Fractures and Dislocations.

Initial on-field management and evaluation:

- Gross deformity may be apparent. There is associated swelling and guarding on physical examination.
- **Palpation:** The knee joint, leg, and thigh should be palpated to assess for possible fracture or associated soft tissue injury.
- **Neurovascular exam:** The dorsalis pedis and posterior tibial pulse should be manually palpated and compared to the contralateral side. Careful sensory and motor exam should be performed and documented.
- **Ligament exam:** Careful knee ligament examination should be performed to assess for cruciate and collateral ligament injury.

X-rays: Three views of the knee should be obtained to assess for fracture. Two views of the femur and two views of the tibia should also be obtained to assess for associated injury. MRI is recommended to confirm ligament injury and to assess the extent of the injury.

Treatment: Requires reduction of the dislocation followed by assessment of vascular status (see Fig. 52-3). Pulses of the involved extremity should be palpated. Any discrepancy in pulse should prompt immediate evaluation for vascular injury using diagnostic studies such as arteriography. Neurologic evaluation should be performed to assess tibial and peroneal nerve function. If a knee dislocation is suspected, the athlete should be immediately transferred to a medical facility for evaluation by an orthopedic and vascular surgeon. Vascular injury requires immediate intervention by the vascular surgery team. The multiple ligament injury in knee dislocations may require surgical reconstruction. Rehabilitation following a knee dislocation is intensive and prolonged (see Fig. 52-3).

Tibial Shaft Fractures

Description: Generally low- to medium-energy fractures. Despite the lower energy mechanism, fracture can be a devastating career-ending injury to an athlete. Early fracture stabilization with a well-supervised rehabilitation program is paramount to a good outcome.

Mechanism of injury: Noncontact rotational injuries occurring in sports like downhill skiing to direct blow-type injuries occurring in contact sports such as football.

Initial management: Deformity, instability, and pain confirm the diagnosis. Immediate splinting prior to transportation is advisable; air or vacuum splints facilitate splinting and transportation. In general, splint the extremity as it lies. Gentle traction while the splint is applied limits the magnitude of deformity and limits ongoing soft tissue injury.

Evaluation:

- **Inspection:** Check for angulatory and rotational malalignment, skin integrity, and for the magnitude of initial soft tissues injury.
- **Palpation:** Check for other injuries on the affected extremity.
- **Neurovascular exam:** Document the neurovascular status on the field prior to transportation and before and after splint application.
- **Stability:** While the splint is being applied the team physician can get a general sense of fracture stability or the lack thereof.

X-rays: AP and lateral x-rays of the entire tibia from knee to ankle are required. Radiographs of the joint above and below are mandatory with all long bone injuries.

Classification:

- **Tscherne classification**
  - **Grade 0:** minimal soft tissue damage
  - **Grade 1:** abrasion/contusion of skin with moderate swelling
  - **Grade 2:** deep abrasion/contusion with significant swelling and potential for compartment syndrome
  - **Grade 3:** Severe swelling and compartment syndrome
- **Gustilo classification of open fractures:** An open wound over the fracture increases the likelihood of an infection; the more severe the open wound the greater the likelihood of infection.
  - **Grade I:** open wound less than 1 cm
  - **Grade II:** larger open wounds (1 to 10 cm) with contamination
  - **Grade III:** severe soft tissue damage
    - **A:** can be closed primarily
    - **B:** severe soft tissue wound with one or all of the following: severe contamination, significant periosteal stripping, flap coverage required
    - **C:** vascular repair or reconstruction required

Treatment: Closed stable fractures can be treated with casting and functional bracing. Locked intramedullary nailing is the treatment of choice for most closed **unstable diaphyseal tibial fractures.** Open reduction with plate osteosynthesis is used in cases in which the fracture extends too close to the adjacent joint for adequate intramedullary nailing. **Open fractures** are treated with aggressive soft tissue management with debridement and irrigation and early closure. The success of treatment is related more to the adequacy of the soft tissue management than it is with the fracture fixation method. Fracture stabilization is with an intramedullary nail, plate, or an external fixator. External fixation is reserved for the **high-energy severe open grades** of tibial shaft fracture where the extent of the soft zone of injury demarcation is poorly defined on the first debridement.

Ankle Fractures

Description: Ankle injuries are among the most common injuries treated by the sports medicine physician.

Mechanism of injury: Most ankle injuries involve a planted fixed foot with a rotational force applied to the leg. The position of the foot and the magnitude and direction of the force determines the pattern of injury.

On-field management: Unstable ankle injuries and those associated with obvious deformity should be splinted on the field prior to transportation. With a stable ankle injury, the athlete can be assisted to the sidelines where further treatment can then be instigated. Initial management includes neurovascular evaluation, splint application, extremity elevation, and ice therapy.

Evaluation:

- **Inspection:** Look for deformity including foot alignment. Note areas of swelling including the syndesmotic region and foot. Document any areas of skin contusion, abrasion, or disruption.
- **Palpation:** Start by examining for associated injury to the knee or proximal fibula. Palpate the medial side, lateral side, syndesmosis, hindfoot, and midfoot. Localize all areas of direct tenderness and try to differentiate from ligamentous areas versus bone.
- **Stability:**
  - **Squeeze test:** Squeeze the midleg, compressing the tibia and fibula together, looking for syndesmotic pain.
  - **External rotation test:** In the absence of obvious deformity, a lightly applied external rotation force to the foot can help identify a fracture. Stability and the absence of pain on external rotation rules out most unstable ankle fractures.
  - **Drawer test:** Stabilize the tibia with one hand and pull the foot forward. Look for excessive anterior translation of the talus out of the mortise; compare to the uninjured ankle.
- **Neurovascular exam:** Document the presence or absence of pedal pulses, capillary refill, and color of the toes. Check all sensory dermatomes of the foot and ankle.

X-rays: Key exam findings that indicate a need for radiographs in the absence of deformity include pain at the malleoli, tenderness at the tip or posterior edge of the malleolus, severe swelling, and inability to bear weight. **Initial x-rays** should include a good quality AP, a mortise (AP with the leg 15 degrees internally rotated), and a lateral. CT scans and MRIs are sometimes required for occult fractures and to rule out osteochondral lesions of the talus.

Classification:

- The **Weber-AO system** depends on the level of the fibula fracture.
  - Type A: Fibular fracture is below the level of the plafond.
  - Type B: Fibula fracture is at the level of the plafond.
  - Type C: Fibula fracture is above the level of the plafond and is generally associated with an injury to the syndesmotic ligament.
- **Lauge-Hansen classification** is based on the position of the foot and the direction of the deforming force (see Fig. 52-3).
  - **Supination external rotation:** Foot is held supinated and an external rotation force is applied.
  - **Pronation external rotation:** Foot is held pronated while an external rotation force is applied to the tibia.
  - **Supination adduction:** Foot is held supinated and an adduction force is applied to the hindfoot and leg.
  - **Pronation abduction:** Foot is held pronated and an abduction force is applied to the hindfoot; usually combined with some external rotation of the ankle and eversion of forefoot.
- **Anatomic classification** is based on the number of malleoli fractured and the level and pattern of fracture: isolated lateral malleolus, bimalleolar, trimalleolar.
- **Syndesmotic ligament injury:** Examination findings suggestive of injury include tenderness over the anterior aspect of the syndesmosis and a positive squeeze or external rotation test. Radiographic findings include an increase in the medial

clear space (distance between the lateral wall of the medial malleolus and medial wall of the talus) greater than 4 mm and the distal tibiofibular space (distance between the posterior border of the tibia and the inner border of the fibula) greater than 6 mm. However, injury may not be apparent radiographically; thus, routine stress testing is necessary for detecting instability.

- **Maisonneuve fracture:** Fracture of the proximal fibula associated with a rotational injury to the ankle; often overlooked. Patients often do not complain of pain in the region of the proximal fibula when more painful injuries are present. More likely when deltoid is ruptured or the medial malleolus is fractured without a fracture of the lateral malleolus. Full length AP and lateral x-rays of the tibia and fibula should be obtained in these cases.

Special tests:

- **External rotation stress test:** The syndesmosis is disrupted with an external rotation force against a fixed foot in both SER (supination external rotation) stage IV and with pronation external rotation injuries. In cases in which it is not clear if the syndesmosis is completely torn, an AP x-ray can be taken with external rotation stress applied to the foot.
- **Ankle joint effusion:** A large ankle joint effusion on plain radiographs is often an indication of an occult fracture. A CT scan or MRI may be indicated in these cases.

Treatment:

- **Isolated lateral malleolus fracture:** Nonsurgical treatment of isolated distal fibular fractures without an injury to the medial side (SER II) is successful in the majority of cases; fracture can be treated with casting or an ankle orthosis with early weight bearing and early ankle range of motion.
- **Bimalleolar and trimalleolar fractures:** Displaced bimalleolar and trimalleolar fractures are best treated with open reduction and internal fixation to reestablish the integrity of the ankle mortise and joint congruity; fixation is generally with screws medially and plate and screws laterally. Fixation of the posterior malleolus is indicated with articular surface involvement greater than 25% and with persistent posterior talar subluxation.
- **Associated soft tissue ligamentous injuries**
  - **Deltoid ligament disruption:** These injuries are treated nonoperatively if the medial clear space reduces anatomically with reduction and fixation of the lateral malleolus. Persistent medial widening demands inspection of the syndesmosis. After internal fixation of the lateral malleolus, external rotation stress x-rays should be retaken to confirm stability of the syndesmosis. If syndesmotic instability is present, indicative of a SER IV injury, then this should be addressed as noted in next section.
  - **Syndesmosis:** Fixation of the syndesmosis is indicated when evidence of a diastasis is present. Fixation methods are evolving but start with anatomic restoration of both fibular length and the tibiofibular relationship at the mortise; methods include single and double screw fixation with tricortical or quadricortical fixation and newer suture repair devices with repair and reconstruction of the syndesmotic ligaments.

Compartment Syndrome of Lower Limb

Description: Surgical emergency usually associated with a fracture of the involved extremity. Acute compartment syndrome represents myoneural ischemia that is caused by elevated intramuscular pressure. Exertional compartment syndrome is a related entity that involves athletes, but it does not represent an emergency.

Presentation: Can be initiated by many conditions, including fractures, crush injuries, severe contusions, or vascular injuries (see Fig. 52-3). Reported in nearly every muscle compartment of the arms, legs, and trunk; most cases involve legs. Classic symptoms are described by the five P's: pain, pallor, pulselessness, paresthesia, and paralysis. The earliest and most reliable symptom is pain that is out of proportion with the injury; other symptoms are less reliable. Do not wait to see if the other symptoms occur prior to making the diagnosis and initiating treatment.

Physical exam: Primary diagnosis is based on physical examination, not compartment pressure measurements. Firm compartments and severe pain are adequate to make the diagnosis. Possible loss of pulses distal to compartment, pallor of the extremity, or nerve dysfunction in the form of either paresthesia or paralysis.

Diagnostics: Various commercial devices have been developed to measure compartment pressures in limbs; the pressure threshold that requires a fasciotomy is very controversial and should mainly be based on clinical examination.

Treatment: Surgical emergency, requiring surgical release of the fascia over the entire length of the involved compartment. Both skin and fascia should be left open following release. Negative pressure wound therapy to aid in the management of fasciotomy wounds can be very helpful.

Prognosis and return to play: Prognosis is highly variable and depends largely on the timing of the diagnosis. This is a limb-threatening condition with a high likelihood that the athlete will not be able to function at the same level as before injury. Best prognosis occurs in patients who have an early fasciotomy.

Open Fractures

Overview: Can be severe injuries that threaten the athlete's career, and possibly the athlete's leg. It is critical that every team physician understand some key principles regarding the treatment of open fractures. A surgeon who is experienced with severe open fractures should participate as part of the surgical team. Debridement and irrigation procedures require a great degree of judgment in order to decide what tissue to debride to minimize the risk of infection, and which tissue to retain in order to maximize long-term function. The team physician should not hesitate to engage trauma surgeons, who have a great deal of experience with severe open fractures, to assist and provide the ideal treatment.

Presentation: Significant bleeding and an obviously open wound, or it may involve virtually no bleeding and only a small wound or abrasion (see Fig. 52-3). Always assume a fracture is open until proven otherwise.

Physical exam: Remove all clothing and pads to allow a careful evaluation of the skin; inspect for lacerations and abrasions. Open fractures should be classified according to the system developed by Gustilo and Anderson (see Fig. 52-3).

Diagnostics: Orthogonal radiographs of the fracture are the primary diagnostic studies necessary. Compartment syndrome is more common with open fractures than with closed fractures; careful evaluation of the compartments is very important with pressure measurements if indicated.

Treatment: Is a surgical emergency. Initial management of the wound including a good examination with gentle cleansing of any gross contamination if possible is key. If a digital camera is available, it is extremely helpful to take a picture of the wound so once it is covered awaiting surgery, the pictures can be shared with colleagues as they arrive. Repeat exposure of the wound increases the risk of infection, and should not be done. Many open fractures that occur in athletic competition are grossly contaminated with soil and grass; it is critical to perform an aggressive and thorough irrigation and debridement in order to avoid osteomyelitis. Several new treatment options have been developed that may be useful with the most severe open fractures:

- The use of negative pressure wound therapy has recently been reported to be associated with a decreased incidence of osteomyelitis following severe open fractures.

Prognosis and return to play: Prognosis for a severe open fracture is driven by both the type of fracture and the open wound. It may be a career-ending injury, and the athlete and coach need to understand that. However, with aggressive treatment and incorporating contemporary treatment methods, it is possible to have an athlete completely recover and successfully resume his or her athletic career.

RETURN TO SPORTS AFTER EXTREMITY FRACTURE

General Principles

- Return to play has to be individualized based on the type of fracture, fixation method, healing, and type of sport activity and position played.
- Returning to a noncontact overhead sport like tennis is much different than returning to a collision sport like football.
- The individual and the team physician must weigh the benefits of an early return against the short-term and long-term risk of re-injury.
- The overriding principle is the health and well-being of the athlete.
- The certified athletic trainer and physical therapist play an important role in this decision process.
- The sport-specific evaluation on the field or court is more important than any test in the office environment. Feedback from the athletes themselves is also crucial.

Upper Extremity

In most cases, the time to return to play with an upper extremity fracture is much less than with a lower extremity fracture. Some degree of cardiovascular fitness, speed, and agility can all be maintained from the beginning. Additionally, the injury can be protected with taping, padding, splinting, and bracing. The return to sport is often some multiple of the time for fracture union (0.5 to 1.0 times the time for fracture healing). The lower extremity rule would apply to an overhead athlete.

Lower Extremity

Rehabilitation starts immediately, but because there is a period of limited weight bearing, full strength, speed, and agility return takes longer. As a general rule, time to sports return is a multiple of the time to fracture union (1.5 to 2.0 times the time for fracture healing).

Hardware

Hardware left in situ: Playing with hardware must be individualized based on the type of sport (collision, contact, noncontact), type of hardware, location of hardware, and stress concentration.

Type of hardware:

- **Plate:** Stress shielding occurs under a plate and an area of stress concentration exists at the end of the plate. Risk of fracture at the end of the plate goes up in a collision sport such as football. Protective padding to distribute loads over a broader surface area is advisable.
- **Intramedullary nail:** Minimal stress shielding occurs with a load-sharing device such as an intramedullary nail and stress concentration is better tolerated in the metaphyseal and epiphyseal regions at the ends of the nail. Minimal protection is required after fracture union.

Hardware removal: Most hardware is left in place until the athlete finishes competitive sports. Symptomatic hardware, especially intramedullary nail interlocking screws, can be removed in the off-season.

- Refracture after plate removal occurs through screw holes sites. An extended period of protection is required after removal.
- Refracture after screw removal for fifth metatarsal base fractures has been reported and screws should be left in place until the athlete has finished competitive sports.
- Intramedullary devices pose minimal risk and are left in place until competitive sports are finished.

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Stress Fractures

53

Brian Aros, Kurt Spindler, and Christopher C. Kaeding

GENERAL OVERVIEW

Definition

Stress fractures are fatigue-failure injuries of bone and are considered the **ultimate overuse injury** affecting physically active people, including military recruits, track and field athletes, and ballet dancers. With increased evidence supporting positive role of exercise for elderly people and patients with chronic disease, stress fractures may become more common and should not be overlooked in nontraditional populations with multiple risk factors.

Etiology

- Stress fractures are overuse injuries that present over a continuum of fatigue failure of bone, from microfracture to complete structural failure. During periods of vigorous exercise, excessive repetitive stress alters the balance of bone remodeling. Bone formation (osteoblastic activity) lags behind periosteal resorption (osteoclastic activity). Stress can be compressive, tensile, rotational, or axial.
- Initially, increased osteoclastic activity at site of stress leads to a more porous, weakened cortex. Above a certain threshold, additional stress cycles lead to development of microscopic cracks in porous bone, further decreasing bone strength. Continued repetitive stress leads to microfracture propagation and coalescence, resulting in stress fracture. With further stress, frank fracture and displacement can occur (Fig. 53-1).
- Moderate repetitive stress over time stimulates bone formation, is associated with adaptive increases in bone mass, and is necessary for development of strong bone.

Epidemiology

- Individual sports place stress on specific anatomic sites, which are at increased risk for stress fracture. Site specificity is determined by athletic population (Table 53-1).
- Certain sports are more commonly associated with stress fractures: **running (69%),** fitness class (8%), racket sports (5%), basketball (4%), other sports (14%).
- Female military recruits have 10 times fracture risk of males; female athletes have 3.5 times fracture risk of males.
  - Risk is activity dependent: higher risk for female runners and gymnasts.
  - Females' higher risk may be related to underlying menstrual irregularities and associated decreases in bone density (e.g., with female athlete triad) or anatomic and biomechanical factors specific to females.
- Most common bones injured, reported as percentage of all stress fractures: tibia (49.1%), tarsals (25.3%), metatarsals (8.8%), femur (7.2%), fibula (6.6%), pelvis (1.6%), sesamoids (0.9%), spine (0.6%). Bilateral injuries in 16.6% of cases.
- Age relationship: Femoral and tarsal fractures more common in older patients; tibial, fibular, and upper extremity fractures more common in younger patients.

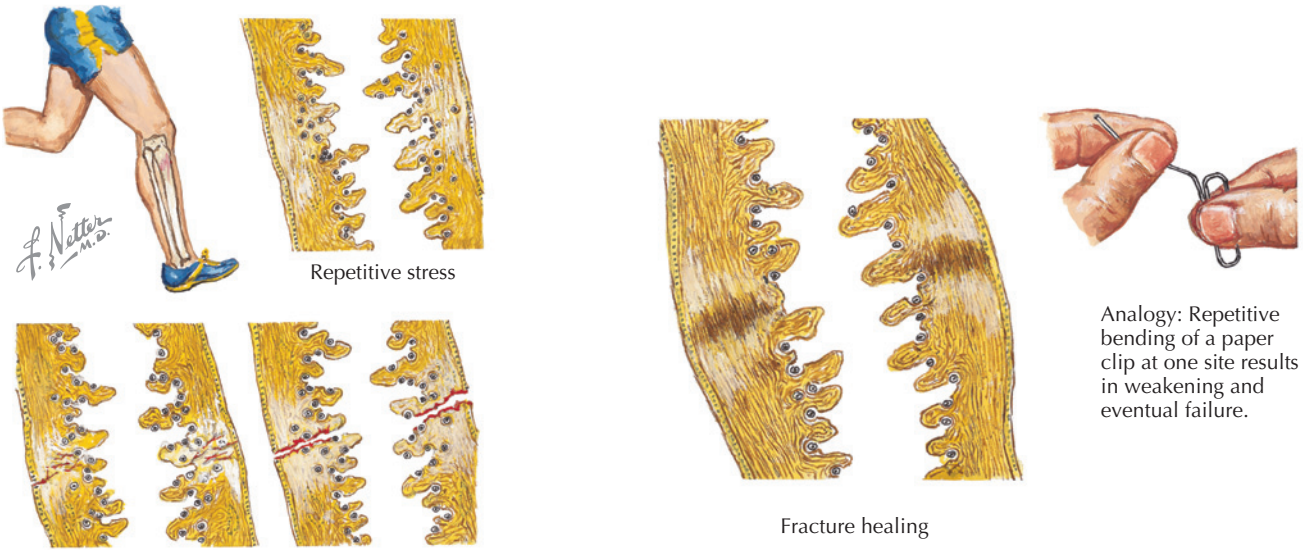


Figure 53-1 Etiology of stress fracture. Site specificity of stress fractures in track and field athletes is determined by the anatomic stress demanded by the sport.

Table 53-1 STRESS FRACTURE SITES ASSOCIATED WITH SELECTED SPORTS

Track	Football	Basketball	Gymnastics	Softball (fast-pitch)	Dance	Running
Navicular	Tibia	First rib	Pars interarticularis	Rib	Metatarsals	Tibia
Tibia	Pars	Tibia	Radius	Ulna	Fibula	Fibula
Metatarsals	5th metatarsal	5th metatarsal	Midfoot	Humerus	Pars interarticularis	Metatarsals
Fibula		Tarsal	Navicular		Tibia	

PROTECTIVE AND RISK FACTORS

See Table 53-2.

Bone Characteristics

- **Composition:** Mineral deposition around collagen matrix. Bone resists compression; collagen matrix (connective tissue) resists tension.
- **Bone remodels in response to stress** (Wolff's law).
- Allows protective increases in cortical thickness, density, and diaphyseal diameter (Fig. 53-2).
- Remodeling is function of number, frequency, and duration of loading cycles. It is influenced by strain volume, rate of application, and duration per cycle.
- Osteoclasts activated by stress. In response to stress, increased osteoclast activity over 30 to 45 days results in increased porosity of bone at stress site.
- Osteoblasts migrate into porous areas and build matrix. This process begins about 30 days after stress and culminates with new bone formation over approximately 180 days.
- If weakened (porous) bone is subject to additional stress, **osteoclastic activity can overwhelm osteoblastic activity, resulting in further weakened bone and microfractures.** Each load cycle may propagate microfractures until symptomatic stress fracture results. Increased microfractures have been demonstrated in areas of bone porosity.
- **Bone geometry** may determine risk.
  - Larger, higher density bones more resistant to fatigue fractures.
  - Long bones with increased diameter resist bending in response to load. In basic training, recruits with tibial stress fractures had significantly smaller tibial widths and cross-sectional areas than recruits without stress fracture.

Table 53-2 ETIOLOGIC FACTORS FOR STRESS FRACTURES

Intrinsic factors	Extrinsic factors
Alignment abnormalities	Overt
Femoral neck anteversion	Continued self-abuse
Pronation/supination	Improper training
Tibial torsion	Improper technique
Leg length discrepancy	Improper equipment
Muscle imbalance	Harsh environment
Muscle weakness	Covert
Flexibility	Joint instability
Genetic predisposition	Extrinsic pressure
Aging/hormonal	Biomechanical fault

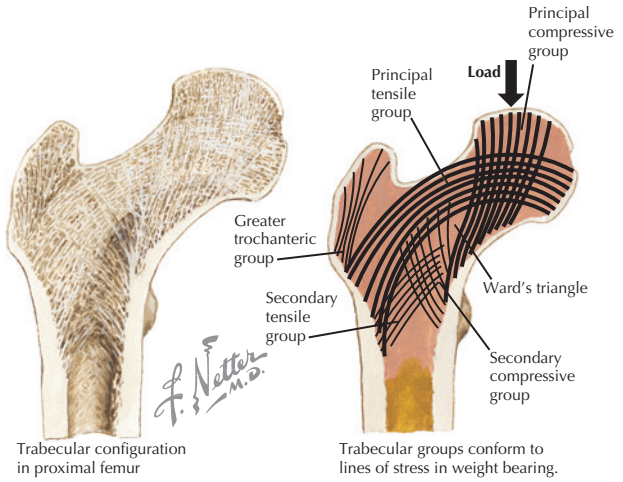


Figure 53-2 Bone Architecture in Relation to Physical Stress.

Soft Tissues (Intrinsic Factors)

- Good muscle strength is protective. Muscle contractions attenuate ground reactive forces developed at impact during running and jumping.
- Bone strain increases as muscles fatigue.
- Contraction of strong opposing muscle groups on single bone may produce tension across bone and promote stress fracture.
- No relationship between stress fracture and muscle size or flexibility.
- General fitness is protective. Military recruits with higher activity levels before enlistment had fewer stress fractures during basic training.

Endocrine

- Oligomenorrheic or amenorrheic female athletes are at risk for stress fractures secondary to decreased estrogen levels and increased osteoclastic activity.
- High-intensity training may suppress menses.
- Delayed menarche, often observed in athletes, may result in decreased bone density but probably does not increase risk for stress fractures.
- Cigarette smoking lowers estrogen in dose-dependent fashion, resulting in higher osteoclast activity.

Nutritional

- Stress fractures are associated with lower fat intake, lower caloric intake, eating disorders, and weights less than 75% of ideal body weight.
- Calcium intake correlates with bone density; low calcium intake does not correlate with stress fractures.

Training Intensity

- Training errors include rapidly escalating frequency, duration, and intensity of training; 60% of running injuries are associated with training errors.
- Stress fractures are increased in first 2 weeks after increased training intensity; with increases greater than 30% in duration or intensity over single season; and in freshman runners adjusting to collegiate training demands.
- Multiple factors compound risk.
  - Independent of risk associated with menstrual dysfunction, athletes training more than 5 hours per day have a sixfold increase in risk for stress fracture.
  - A dancer who is both amenorrheic for more than 6 months and training more than 5 hours per day is 93 times more likely to develop a stress fracture than an eumenorrheic counterpart who trains less.

Extrinsic Factors

- **Surface**
  - **Hard running surfaces** increase impact forces.
  - Unidirectional running on **cambered track surfaces** may predispose to injury.
  - Large epidemiologic studies reveal no association between stress injury and running surface.
- **Environment** (e.g., hills, uneven terrain)
- **Technical faults in training or performance—**may predispose to stress injury

DIAGNOSIS

Time to Diagnosis

It takes an average of 5 to 16 weeks to diagnose a stress fracture. Delayed diagnosis can be detrimental, especially with high-risk stress fractures.

History

- Maintain high index of suspicion when evaluating frequency, intensity, and duration of activity.
- Some affected anatomic areas are particularly challenging to diagnose.
- Recall of specific detailed historical facts may be challenging for athletes.
- Recent change in activity level over past 2 months and preseason training.
- Recent change in equipment or playing surface.
- Activities added to already demanding physical performance.
- Training errors: too rapid progression of mileage (frequency and/or duration, overall increased training volume) or intensity.
- **Insidious onset of pain,** usually initially after activity; with fracture progression pain moves earlier into activity and may eventually occur with minimal activity (e.g., walking) or even at rest.
- Vague pain in affected region, occasionally localized.
- Pain relief with rest and/or reduced activity.
- Prior stress fractures.
- **Females:** menstrual irregularities, recent weight changes, eating disorder history or behaviors, nutrition.
- **Older athletes, especially female:** osteoporosis risk factors (e.g., smoking, family history, corticosteroid use, body weight, Caucasian race, menopause, hormone replacement).

Physical Examination

- Point tenderness, edema, warmth, and palpable callus in some cases (Fig. 53-3). In areas where bone not easily palpated, such as femoral neck, gentle range of motion (ROM) may elicit pain.
- Tenderness with percussion or with sound waves (tuning fork or ultrasound).
- Specific tests:
  - **Stork test or one-legged standing hyperextension test:** Back extension with resultant hip flexion while standing on one leg increases pain in pars interarticularis stress fractures.
  - **Fulcrum test:** Performed by placing fist or arm of examiner under suspected fracture site and applying "bending" force to distal extremity while proximal extremity is kept relatively immobilized by anatomic restraints. Positive test results in pain over fracture site. Commonly used to evaluate femoral shaft stress fractures but may be used in other "long bone" areas.
  - **Hop test:** Hopping on affected leg reproduces pain at fracture site, usually with femoral shaft stress fractures.
  - **Tuning fork test:** Vibrating tuning fork over fracture site results in pain at site. High rate of false positives.

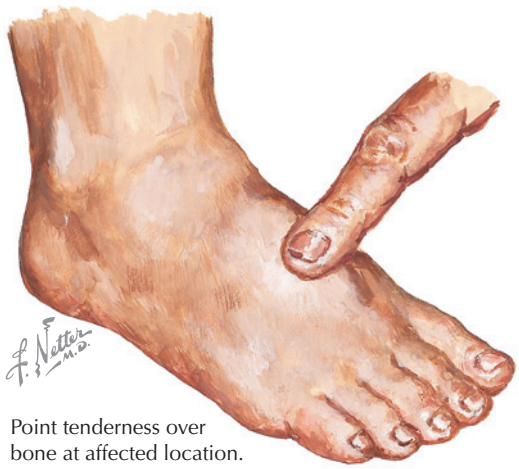


Figure 53-3 Physical Examination of Stress Fracture.

Radiologic Studies

- **Plain radiographs** usually negative early, especially first 2 to 3 weeks. Two-thirds of initial x-rays are negative, but half ultimately prove positive. X-rays are specific but not sensitive.
- **Ultrasound:** Not reliable for diagnosis; several studies in progress.
- **Bone scan:** Nearly 100% sensitive but not specific. Bone scan–negative, MRI-positive stress fractures of femoral shaft have been reported. Not helpful for guiding return to play; uptake on bone scan normalizes over 12 to 18 months, often lagging behind resolution of clinical symptoms. Especially useful in tarsal, femur, pelvis, and tibial plateau fractures. Helps differentiate stress fractures from periostitis (shin splints). Stress fractures are **usually positive in all phases of triple-phase technetium bone scan** (angiogram, blood pool, delayed). Periostitis is often negative in the angiogram and blood pool phase and positive in delayed image phase.
- **Single-photon emission computed tomography (SPECT):** More specific than planar bone scan; especially helpful in detecting stress fractures of the pars interarticularis, pelvis, and femoral neck.
- **Computed tomography (CT):** Delineates bone well, useful when diagnosis is difficult (e.g., tarsal navicular stress fractures); helpful with spinal or linear stress fractures.
- **Magnetic resonance imaging (MRI):** Expensive, but identifies soft tissue injuries. Superior sensitivity and specificity over bone scan and CT for soft tissue abnormalities and edema; may delineate early injury. Findings include periosteal or marrow edema. Musculoskeletal protocols optimize MRI use but are not available in many institutions. MRI may be highly useful with certain stress fractures (such as hip and pelvis), especially if used by musculoskeletal radiologists familiar with specific imaging protocols.

GENERAL TREATMENT

- Because they are overuse injuries, most stress fractures respond well to relative rest and gradual reintroduction of sport. High-risk stress fractures demand rapid diagnosis and specific intervention to avoid complications.
- Stress fractures frequently heal in 6 to 8 weeks (usually low-risk), but certain fractures (mostly high-risk) heal more slowly over 3 to 4 months or longer.

Suggested Phases of Treatment

- **Phase I:** Pain control, usually 10 to 14 days.
  - Controversial to use nonsteroidal anti-inflammatory drugs (NSAIDs) at analgesic doses. Use with caution because higher (anti-inflammatory) doses may inhibit bone healing. Some preferentially use acetaminophen.
  - Physiotherapy (may include bone stimulator).
  - Flexibility and strengthening.
  - Complete or relative rest depending on fracture site and severity of symptoms.
  - Bracing may speed return to play in specific cases (e.g., long-leg pneumatic splint with anterior buttress in tibial shaft fractures).
- **Phase II:** Reintroduction of activity; may last several weeks depending on location and type of stress fracture. Initiation of phase II varies with patient symptoms and depends on size and type of stress fracture.
  - Continue phase I treatment.
  - Maintain aerobic fitness through pool running and cycling. Newer elliptical trainers may also be helpful.

- Gradually add impact activity, altering surface or equipment if possible. Run short distances on grass or soft surfaces and gradually increase running time.
- Resume sports-specific training in noncompetitive setting.
- Work out on alternating days and maximize rest between bouts of exertion.

- **Phase III:** Preparation for return to competition.
  - Increase sport-specific plyometric conditioning drills. Add running drills, cutting drills, and selected skill work.
  - Begin lower-level competitive challenges.

SPECIFIC STRESS FRACTURES

Upper Extremity

Although much less common than in the lower extremities, stress fractures of the upper extremities should be included in differential diagnosis of athletes who perform repetitive throwing, swinging, or overhead activity. Gymnasts and cheerleaders who use the upper extremities for weight bearing and twisting are at risk for stress fractures below the elbow. Young athletes and throwing athletes (e.g., goalkeepers, javelin throwers) develop stress fractures of the humerus and shoulder girdle. Swinging athletes (golf and rowing) are at risk for rib fractures.

Humerus

Description: **Proximal** and **medial epicondyle** fractures seen in younger throwing athletes and gymnasts. **Midshaft** fractures seen in adult throwing athletes and workers doing heavy lifting.
History: Pain with throwing or lifting heavy weights; may involve entire upper arm.
Examination: Normal or pain on palpation or resisted motion.
Treatment: Rest; immobilization, such as fracture brace; 6 to 8 weeks for healing; correction of biomechanical and technical faults.

Ulna

Description: Reported in baseball and softball pitchers, rodeo riders, bowlers, golfers, volleyball players.
History: Forearm pain over ulnar shaft during and after activity. Fracture location varies by activity. Fractures develop proximally in volleyball players secondary to repetitive, explosive wrist flexion. Fractures occur distally in softball players (underhand activity). Midshaft stress fractures reported in tennis. Pronation with backhand stroke at ball strike and follow-through are painful.
Examination: Pain, edema, and local heat over involved areas. Painful pronation and supination.
Radiographic studies: X-ray typically helpful, shows periosteal reaction. Bone scan or MRI used to confirm. MRI may demonstrate edema in interosseous membrane.
Treatment and return to play: Rest from activity, resume gradual activity when pain-free. Extension block splints sometimes used. Correction of biomechanical, technical faults. In all reports, return to play is in 4 to 6 weeks.

Olecranon

Description: Occurs in throwing athletes; caused by recurrent valgus extension overload. Young gymnasts may injure olecranon physis.
History: Pain with elbow extension/throwing.
Examination: Point tenderness and pain with triceps extension against resistance.
Radiographic studies: X-ray may be negative. Nonunion risk higher if x-ray shows sclerosis around area of lucency. Fracture site is under tension. X-ray demonstrates unique transverse radiolucency extending from posterior nonarticular to articular surface (Fig. 53-4).
Treatment: Short-term immobilization to reduce triceps pull. Surgery may be required for nonunion.

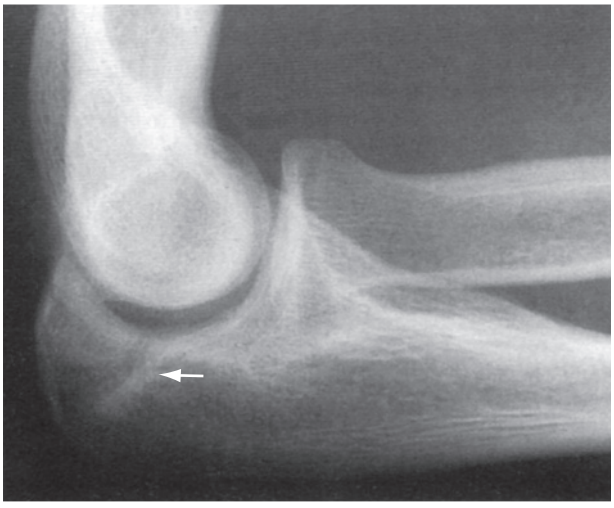


Figure 53-4 Lateral Elbow, Olecranon Stress Fracture.

Radius

Description: Uncommon. Reported in military personnel, cheerleaders, gymnasts, tennis players, volleyball players, softball players, pool players, and cyclists.
History: Pain in shaft of radius with above listed activities, especially with wrist supination.
Examination: Pain over involved area.
Radiographic studies: X-rays show periosteal thickening and occasionally bowing. Bone scan or MRI confirms diagnosis.
Treatment: Six weeks of rest. Repetitive wrist weight-bearers (gymnasts, cheerleaders) may require longer rest.

Shoulder Girdle and Trunk

Clavicle

Description: Cases reported in javelin throwers and springboard divers. Mechanism of injury hypothesized to be repetitive deltoid and pectoralis major contractions during javelin throw and repeated water entry (diving) with wrists extended and forearms pronated.
History: Insidious onset of pain over clavicle.
Examination: Pain reproduced with abduction of the shoulder above horizontal plane of shoulder.
Radiographic studies: X-ray shows periosteal reaction. Bone scan or CT used to confirm diagnosis.
Treatment: Rest from activity; return to play in 8 weeks.

Scapula

Description: Stress fracture of coracoid process reported in female trapshooter secondary to repetitive impact of rifle butt. Stress fractures at other scapular sites reported in gymnasts, joggers who carry dumbbells (superomedial scapula), and football players (associated with weightlifting; acromion).
History: Gradual onset deep aching in shoulder.
Examination: Pain with palpation of coracoid process and bicipital groove. Painful resisted adduction and forward flexion of arm.
Radiographic studies: Axillary view necessary for plain x-ray diagnosis. West Point axillary and modified oblique views helpful for acromial stress fractures. MRI or bone scan confirms diagnosis.
Treatment: Rest from trapshooting for 6 weeks. At 6 weeks, patient was pain-free with full ROM and gradually resumed shooting. Other fracture sites: return to play in 8 weeks with rest. Consider butt modification or padding on return to sport.

Ribs

Description: First rib most commonly seen in baseball pitchers and basketball players; reported in weightlifting, tennis, table tennis, rugby, judo, gymnastics, and ballet. Other rib fractures seen in softball (pitchers), golf, tennis, and rowing. Diagnosis often delayed because misdiagnosed as back strain. Typically posterolateral, although anterolateral and rib neck stress fractures have been reported in rowers. Possibly related to serratus anterior fatigue in golfers.

History: Insidious onset, but occasionally presents acutely without preceding symptoms.

Examination: Pain with arm motion over supraclavicular area (first rib) or over affected area. Pain with trunk rotation (lower ribs). Serratus anterior weakness.

Radiographic studies: X-ray often negative; usually positive if fracture is over broad, flat, thick portion of first rib. Other ribs are difficult to diagnose using x-ray. Bone scan or MRI helpful to confirm diagnosis.

Treatment: Rest, avoidance of overhead and swinging activity, maintenance of aerobic fitness, general conditioning and strengthening of serratus anterior; gradual return to play when pain-free.

Spine

Spondylolysis: Pars Interarticularis Stress Fractures

Description: Most common in athletes undergoing repetitive hyperextension of spine (gymnasts, cheerleaders, divers, weightlifters). L4 and L5 are vertebrae levels most commonly affected. Common cause of pediatric low back pain. In orthopedic referral population, 47% of pediatric patients with low back pain had spondylolysis, compared with only 5% of adults.

History: Athlete usually involved in some repetitive back **extension load** activity. Insidious onset of low back pain; patient ultimately complains of significant back spasms (often misdiagnosed as lumbar strain). Short periods of rest may temporarily relieve pain, but return to activity results in immediate exacerbation of symptoms.

Examination: May have clinical hyperlordosis; pain to palpation over affected vertebral levels; exquisite pain and muscle guarding with one-leg and two-leg standing trunk extension test (one-leg usually worse), trunk rotation and extension, hip extension test (prone), trunk extension test (prone), combined hip and trunk extension test (prone). Neurologic exam usually normal; occasionally may have associated radiculopathy.

Radiographic studies: X-rays have low sensitivity. Anteroposterior (AP), lateral and bilateral, and oblique views. If positive, classic defect of "collar" on neck (pars interarticularis) of Scotty dog is seen on oblique views. Falling out of favor because of low sensitivity and high radiation. SPECT scan has greater sensitivity and is becoming gold standard for diagnosis. Thin-cut CT scan (1.5 to 2 mm cuts) may help identify extent and age of fracture, partial versus complete, and sclerosis (Fig. 53-5). Combination of SPECT and CT findings may help determine likelihood of healing and may help design treatment protocol.

Treatment: Somewhat controversial. Initially, modify activity and avoid back hyperextension. Some consider nonrigid bracing if pain persists. At 2 to 4 weeks, begin physical therapy (trunk stabilization rehabilitation and flexibility exercises). If pain present at 4 weeks, thoracolumbosacral orthosis (TLSO) or low-profile antilordotic Boston brace (rigid) is an option to unload posterior elements and prevent hyperextension (lumbar lordosis). Treat until patient is symptom free. Healing can take 3 to 6 months and may not correlate with symptoms. Consider CT scan to assess healing. Consider spine surgical evaluation if persistent pain after rigid bracing, especially if neurologic symptoms appear or progress.

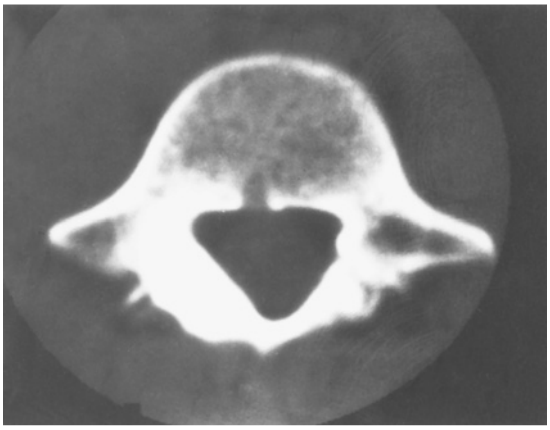


Figure 53-5 Axial CT Image, Pars Interarticularis Stress Fracture with Sclerosis.

Return to play: As early as 8 weeks if patient remains pain-free at rest, in hyperextension, and while performing aggravating activities.

Pelvis

Description: Pubic ramus usually involved. Uncommon; seen nearly exclusively in women, military recruits, long-distance runners or joggers.

History: Pain in inguinal, perineal, or adductor region that is relieved by rest. Seen in female British Army recruits forced to match official 30-inch stride length while marching. When stride length decreased to 27 inches, no further stress fractures were reported, suggesting increased stride length may promote biomechanical stress at pubic ramus.

Examination: Antalgic gait, full range of motion, pain over pubic ramus, positive "standing sign" (inability to stand unsupported on affected side).

Radiographic studies: X-rays initially negative; late in course, may show abundant callus. Bone scan or MRI needed for early diagnosis.

Treatment: Brief non–weight-bearing, then rest from running. Healing variable, typically 3 to 5 months.

Sacral Stress Fractures

Description: Fatigue fractures reported mainly in runners; high index of suspicion required. Insufficiency fractures more common in osteoporotic bone (elderly).

History: Vague, poorly localized pain in gluteal or groin area. Rapid increase in mileage or training schedule often associated.

Examination: Positive figure-4 (flexion, abduction, external rotation [FABER]) test and hopping test. Normal ROM, but deep groin pain at extremes of motion.

Radiographic studies: X-rays usually negative. Bone scan or MRI used for diagnosis.

Treatment: Cessation of running, protected weight bearing, relative rest lasting from 6 weeks to 8 months.

Femoral Stress Fractures

Femur sites most commonly involved with stress fractures, from most to least common: shaft, lesser trochanter, intertrochanteric region, neck, greater trochanter. Missed diagnosis of a femoral neck fracture can have high morbidity.

Femoral Neck

Description: Seen most frequently in runners, dancers, and military recruits. Diagnosis is typically delayed 5 to 13 weeks. Severe complications arise with progression to complete fracture and displacement. Complications may include avascular neurosis,

nonunion, varus deformity, and chronic pain. **Superior** neck fractures are under tension and are a high-risk area for progression to complete fracture. **Inferior** neck fractures are compression-sided and are often managed conservatively.

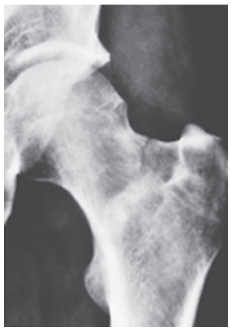
History: Earliest sign: 87% report inguinal or anterior groin pain. Aching pain precipitated by weight-bearing activity: 40% reported symptoms after long run, between 6 to 8 weeks of training. Symptoms appear with increase in training schedule. Diagnosis often delayed.

Examination: Antalgic gait (22%); pain with palpation in groin, hip, or anterior thigh (70%); pain at extremes of hip ROM (79% of 39 patients in one study). Subtle limitation of flexion and internal rotation.

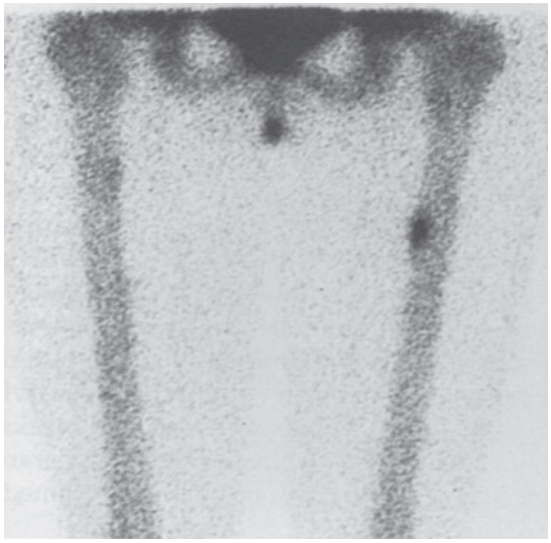
Radiographic studies: X-rays have high false-negative rate, especially early. Bone scan or SPECT scan for early diagnosis. False negatives reported up to 12 days after fracture. MRI is becoming more popular and is a sensitive study for identifying early marrow edema, which typically resolves in 8 to 12 weeks.

Treatment (Blickenstaff criteria modified by Fullerton): Complications in up to 30% of patients. Return to play may be delayed up to 2 years.

- **Compression-sided fractures** (Blickenstaff type I): inferior neck.
  - Type Ia: Callus without fracture line and positive bone scan; managed conservatively with non–weight-bearing and/or bed rest. Gradual activity progression when pain-free. Weekly radiographs until patient can walk without pain using cane.
  - Type Ib: Definite fracture line but no displacement; treat with non–weight-bearing, or surgery. Serial radiographs.
- **Tension-sided fractures** (Blickenstaff type II): superior neck (Fig. 53-6).
  - A positive bone scan without a visible fracture line on x-ray is treated with bed rest or non–weight-bearing and crutches. Serial radiographs to look for developing fracture line.
  - If fracture line present, higher risk of displacement, avascular necrosis, nonunion, and malunion. Early diagnosis and surgical management usually required. If no displacement, some advocate bed rest as first-line treatment in lieu of immediate surgery.
- **Displaced** (Blickenstaff type III): surgical fixation recommended.



AP hip, superior femoral neck (tension-type fracture).



Bone scan, increased uptake femoral shaft.

Figure 53-6 Femoral Stress Fractures.

Femoral Shaft Stress Fractures

Description: Seen mostly in runners, especially females. Most common location is at junction of proximal and middle thirds of femoral shaft.

History: Sudden increases in frequency, intensity, or duration; pain with running progresses to pain with activities of daily living.

Examination: Antalgic gait (22%); normal ROM (94%); pain with palpation in groin, hip, or anterior thigh (70%). Hopping on affected leg reproduces pain (hop test); positive in 70%. Positive fulcrum or "hanging leg" test.

Radiographic studies: X-ray negative early; callus and lucent fracture line in 2 to 6 weeks. Bone scan or SPECT scan for early diagnosis (see Fig. 53-6). MRI is much more sensitive; clearly identifies bone edema, which typically resolves in 8 to 12 weeks.

Treatment: Conservative management of shaft stress fractures is successful. First-line interventions include protected weight-bearing with crutches for 1 to 4 weeks (length of time depends on resolution of pain); activity modification; maintenance of aerobic fitness, skill, and strength. If pain-free during day-to-day activities at 14 days, begin rehabilitation with low-impact, minimal weight-bearing exercise (cycling, swimming, pool running). Time to full recovery varies, but reported as 5 to 10 weeks from diagnosis. Resumption of athletic activity may take 8 to 16 weeks.

Knee and Lower Leg Stress Fractures

Patella

Description: Rare; reported in basketball, soccer, and high-jump. May be longitudinal or transverse. Reported after anterior cruciate ligament (ACL) patellar tendon graft repair, in patients with cerebral palsy, and in young athletes.

History: Anterior knee pain, worse with jumping, may have had prior bone tendon bone graft harvest for ACL reconstruction.

Examination: Pain to palpation, pain with resisted knee extension.

Radiographic studies: X-rays may show definite fracture lines; must differentiate from bipartite patella. Bone scan. MRI clarifies diagnosis, identifies bone edema.

Treatment: Transverse fractures prone to displacement. Nondisplaced fractures are treated with long leg or cylinder cast immobilization with the knee in full extension for 4 to 6 weeks, followed by quadriceps rehabilitation. Displaced fractures are treated with open reduction and internal fixation. **Longitudinal fractures** occur in lateral patellar facet; if displaced, excise lateral fragment.

Proximal Tibia Fractures

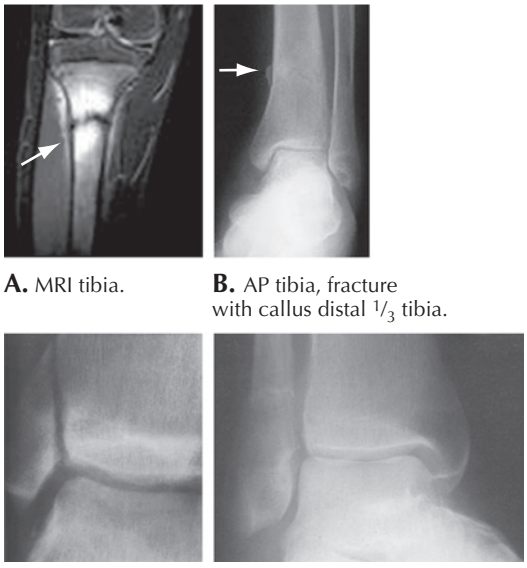
Description: Infrequent; incidence unknown.

History: Pain in anteromedial aspect of proximal tibia. Weight-bearing precipitates pain.

Examination: Localized tenderness and edema. Need to differentiate from pes anserinus tendinitis, bursitis, and saphenous nerve entrapment.

Radiographic studies: Acute stress fracture often has no radiographic findings. MRI is most sensitive study (Fig. 53-7).

Treatment: Modification of offending activity usually by decreasing frequency and duration.



A. MRI tibia. **B.** AP tibia, fracture with callus distal $^1/_3$ tibia. **C.** Medial malleolus fracture. **D.** AP ankle, distal fibula stress fracture.

Figure 53-7 Knee and Lower Leg Stress Fractures.

Tibial Shaft Stress Fractures

Description: Need to differentiate between compression stress fracture and tension stress fracture ("dreaded black line").
- **Compression stress fractures:** MRI suggests continuum between medial tibial stress syndrome and stress fracture, with varying degrees of periosteal edema. Common sites include proximal or distal third tibia, posteromedial tibia (see Fig. 53-7). Common in runners.
- **Tension stress fractures:** Located in anterior or anterolateral cortex in central third of tibia; common in ballet dancers, jumpers.

History: Initially, pain occurs after activity. Later pain occurs with activity and activities of daily living.

Examination: Localized pain, anterior or medial tibia. Edema, palpable periosteal thickening, and pain with percussion. Positive "tuning fork test" (beware false negative; useful if pretest probability is high).

Radiographic studies: X-rays may be positive if symptoms have persisted 4 to 6 weeks. Bone scan shows fusiform uptake, which differs from linear uptake of medial tibial stress syndrome. False-negative bone scans have been reported. MRI more sensitive; used for grading and prognosis for return to play. (Grade 1 and 2 less symptomatic, return to sport 4 to 6 weeks; grade 3 and 4 often need more time for healing.)
- Grade 1: mild periosteal edema (T2), normal marrow (T1, T2).
- Grade 2: moderate periosteal edema (T2), marrow edema (T2).
- Grade 3: severe periosteal edema (T2), marrow edema (T1, T2).
- Grade 4: severe periosteal edema (T2), marrow edema (T1, T2), distinct fracture line visible.

Treatment: Control pain. Stop running; use crutches if necessary.
- **Compression lesions** may take 2 to 12 weeks to heal. Return to play faster with three-panel, long-leg stirrup brace. Correct amenorrhea in females. Calcium supplementation recommended. Correct biomechanical faults (consider foot orthotics). In general, return to play guided by MRI grade.
  - Grade 1: return to play in 2 to 3 weeks, after rest.
  - Grade 2: return to play in 4 to 6 weeks.
  - Grade 3: return to play in 6 to 9 weeks (initially painful with ambulation).
  - Grade 4: return to play in 12 weeks, after casting for 6 weeks and 6 weeks of nonimpact rehabilitation (single case).
  - When pain-free: cross-training, nonimpact and reduced-impact aerobic training; progress to alternating-day graded return to running.
- **Tension lesions** achieve faster return to play with intramedullary rod. Conservative treatment may heal in 6 months but has high rate of recurrence. May progress to complete fracture if missed.

Prevention is best treatment: strength and flexibility of gastrocnemius and soleus; correction of poor running technique; correction of biomechanical issues (e.g., pronation, pes planus) at preparticipation evaluations, especially with history of stress fracture.

Medial Malleolar Stress Fractures

Description: Extend in oblique fashion from plafond. Inherently unstable and prone to nonunion. High index of suspicion key to early diagnosis.

History: Insidious onset medial ankle pain, increased with exercise and relieved by rest.

Examination: Tender over medial malleolus, ankle effusion.

Radiographic studies: X-rays usually negative early. Bone scan or MRI usually required for diagnosis.

Treatment: MRI or bone scan positive, no fracture on x-ray: stirrup immobilization for 4 to 6 weeks. X-ray or CT demonstrates fracture; consider open reduction and internal fixation (see Fig. 53-7). Nonunion requires bone graft and screw fixation.

Fibular Stress Fractures

Description: Most common in distal third just proximal to distal tibiofibular syndesmosis.

History: Limp, swelling.

Examination: Point tenderness, localized edema. Foot overpronation with hindfoot valgus common as lateral malleolus is placed at risk.

Radiographic studies: X-rays not positive for 3 to 4 weeks (see Fig. 53-7). Bone scan can assist with diagnosis.

Treatment: Conservative. For distal fractures, pneumatic ankle brace may be helpful.

Stress Fractures in Foot

Calcaneus

Description: Seen in military recruits and runners.

History: Insidious onset of heel pain with running.

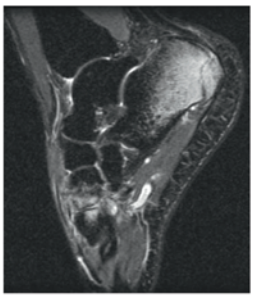
Examination: Positive heel squeeze test; may have positive hop test; edema.

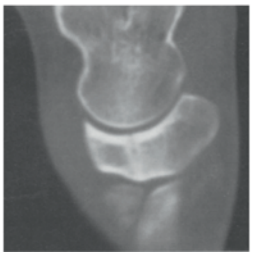
Radiographic studies: X-ray shows endosteal callus perpendicular to long axis of calcaneus. Bone scan, MRI, or CT to confirm diagnosis (Fig. 53-8).

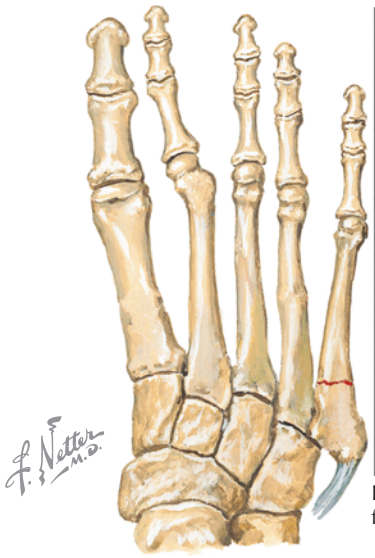
Treatment: Rapid healing with conservative treatment, including relative rest and activity modification. Brief period of non–weight-bearing if ambulation painful. Return to activity in 3 to 4 weeks.

Tarsal Navicular Stress Fractures

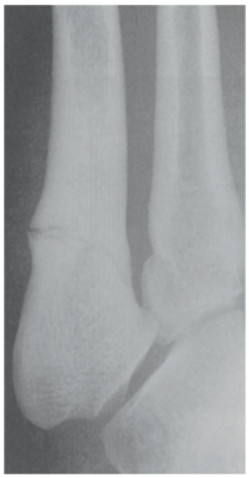
Description: Previously thought uncommon, now recognized in jumping and running athletes, soccer, basketball and track. Vague symptoms often lead to delayed diagnosis. Complications include nonunion and chronic pain. Most fractures in sagittal plane and middle third secondary to poor blood supply. Diagnosis requires high index of suspicion and early imaging (bone scan, MRI).


MRI calcaneus.


CT foot, navicular stress fracture.



Proximal diaphysis 5th metatarsal stress fracture.


Base 5th metatarsal fracture x-ray.

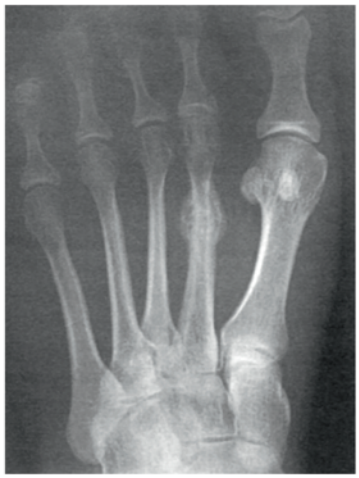

Distal $^1/_3$ 2nd metatarsal stress fracture with callus formation.

Figure 53-8 Stress Fractures in Foot.

History: Insidious onset forefoot/midfoot pain, especially with running and jumping. Pain occasionally mild and usually relieved with rest.

Examination: Pain at "N spot" (dorsal aspect of the navicular). Pain may be diffuse rather than localized.

Radiographic studies: X-rays usually negative. Bone scan or MRI can confirm diagnosis. CT or MRI helps determine fracture site and extent of healing (see Fig. 53-8).

Treatment: If no bicortical fracture, then non–weight-bearing cast immobilization for 6 to 8 weeks. If pain-free (best guide to healing), 6-week graduated program of weight-bearing activity. Consider foot orthotics on return to play. Open reduction, internal fixation (ORIF) with bone graft for displaced fractures, failure of nonoperative treatment, delayed union, and nonunion.

Metatarsal Stress Fractures

PROXIMAL DIAPHYSIS OF FIFTH METATARSAL

Description: Distal to tuberosity and prone to nonunion. Seen in basketball and, less commonly, football.

History: Insidious onset of lateral foot pain, worse during and after activity. Pain steadily worsens. Occasionally, there is acute fracture.

Examination: Point tenderness distal to tuberosity, usually in zone 3 (proximal diaphysis).

Radiographic studies: X-rays usually show sclerotic change around fracture site. Bone scans are only occasionally necessary (see Fig. 53-8).

Treatment:

- Torg classification (proximal fifth metatarsal fractures).
  - Acute: sharp fracture line with no sclerosis or cortical hypertrophy.
  - Delayed union: history of previous injury, wide fracture gap, intramedullary sclerosis.
  - Nonunion: recurrent symptoms, wide fracture gap, sclerosis.
- In high-demand and recreational athletes, intramedullary screw has been demonstrated to permit faster return to play and decrease nonunion rate. Usually weight-bearing activity in 7 to 14 days, with training progression to full unrestricted activity over 6 to 9 weeks. Small risk of fracture nonunion and fatigue fracture of screw.
- In nonathlete, short leg non–weight-bearing cast for 6 to 8 weeks may be considered.

OTHER METATARSALS

Description: First metatarsal (MT): 10% of MT stress fractures; associated with overpronation. **Second, third, and fourth MT:** 90% of MT stress fractures. Associated with running and training more than 20 miles weekly. Flatfeet (pes planus) increases impact stress to MT. Most fractures are distal in runners (see Fig. 53-8). In ballet dancers, fractures occur proximally and often involve medial second metatarsal secondary to en pointe work.

History: Localized pain and edema. Onset insidious, worse after increase in training.

Examination: Pes planus or overpronation; point tenderness and localized edema over MT.

Radiographs: Weight-bearing AP, lateral, oblique x-rays. In dancers with second MT pain, consider internal and external oblique radiographs. Usually positive; bone scan helpful if negative.

Treatment: Rest and stiff-soled shoe or low-tide cast boot. Gradual reconditioning to repetitive stress (at 2 to 3 weeks), such as pool running performed first in chest-deep water and progressing to more shallow depths as symptoms allow. Gradual progression to biking, then running. Consider foot orthotics. In distal first MT stress fractures, dorsal displacement may require casting with dorsal pressure to prevent lateral metatarsalgia. In dancers, proximal second MT stress fractures may progress to nonunion and must be aggressively managed with casting or ankle-foot orthosis for 8 weeks.

SESAMOIDS

Description: Difficult diagnosis; differentiate from sesamoiditis, bipartite and tripartite sesamoids. Thirty percent of population has bipartite sesamoids.

History: Pain over plantar aspect of first metatarsophalangeal (MTP) joint; pain with "toe-off."

Examination: Pain to palpation, pain with resisted first toe plantarflexion, pain over sesamoids with stretch into extreme of dorsiflexion.

Radiographic studies: Diagnosis of sesamoid stress fracture by x-ray is challenging, and additional imaging (e.g., bone scan, MRI) is often required to differentiate stress fracture from bipartite sesamoid. MRI helps identify marrow edema.

Treatment: Conservative, initially 6 weeks of non–weight-bearing cast to prevent dorsiflexion of first ray. Gradual return to sports over several months. Consider foot orthotics after casting. Rarely surgical excision for delayed union or chronic pain.

RECOMMENDED READINGS

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6. Fredericson M, Bergman AG, Hoffman KL, et al: Tibial stress reaction in runners. Am J Sports Med 23:472-481, 1995.
7. Kaeding CC, Yu JR, Wright RW, et al: Management and return to play of stress fractures. Clin J Sports Med 15:442-447, 2005.
8. Kahn KM, Brukner PD, Kearny C, et al: Tarsal navicular stress fracture in athletes. Sports Med 17:65-76, 1994.
9. Matheson GO, Clement DB, McKenzie DC, et al: Stress fractures in athletes: A study of 320 cases. Am J Sports Med 15:46-58, 1987.
10. Mologne TS et al: Early fixation versus casting in the treatment of acute Jones fracture. Am J Sports Med 33:970-975, 2005.
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12. Winfield AC et al: Risk factors associated with stress reactions in female marines. Mil Med 162:168-172, 1997.

Foot Problems

54

Melissa D. Koenig and Thomas O. Clanton

NORMAL ANATOMY AND PHYSICAL EXAMINATION

Observation and Measurement

Standing

Alignment of the lower extremities: View patient from front and back.

Angular and rotational deformities: Check for pelvis, patellae, and tibial tubercles, pelvic tilt and leg length discrepancy; varus, valgus, flexion, extension, and rotational abnormalities of the lower extremity.

Foot alignment and mechanics: Evaluate medial longitudinal arch to assess for pes planus, pes planovalgus, pes cavus (Fig. 54-1). Heels should invert and arch increase when rising onto toes. Evaluate forefoot for hallux valgus and pronation; crossover, cock-up, and hammering of lesser toes.

Sitting

Visible abnormalities: Note varicosities, erythema, ecchymosis, and edema.

Vascular: Palpate posterior tibial and dorsalis pedis pulses. Assess capillary refill time. Absence of hair distally may indicate peripheral vascular disease.

Skin: Note location of callus formation, scars, wounds, blisters, and ulcerations.

Range of motion: Check active and passive ankle, subtalar, transverse tarsal and first metatarsophalangeal joint motion; compare to uninjured side.

Walking

Mechanics of gait: Assess for asymmetric movement on either side of the body, degree of toeing in or out, amount of heel in-

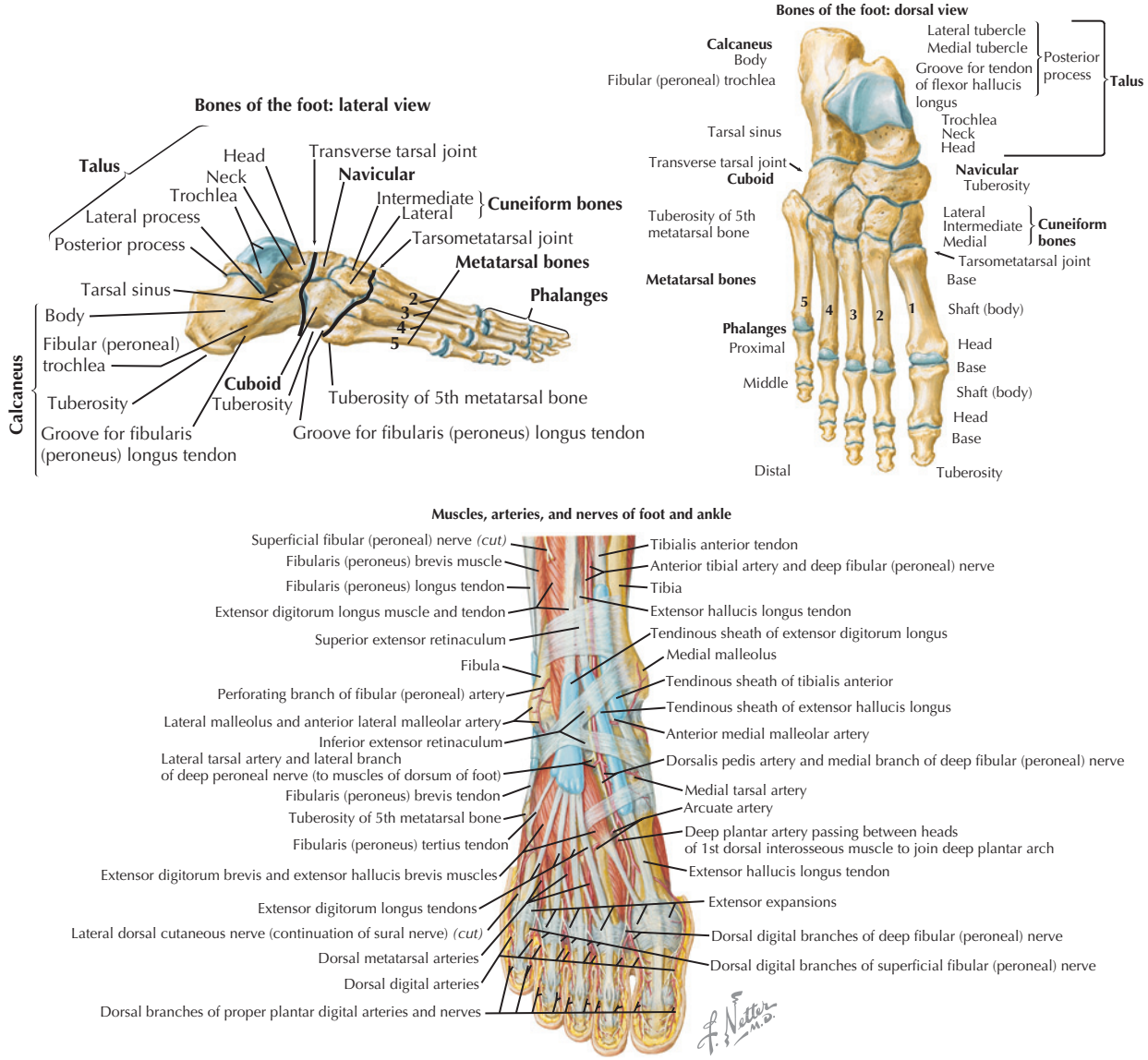


Figure 54-1 Anatomy of the Foot.

version, and supination of the foot. Observe the medial arch during stance.

Palpation

Ligament attachments: Calcaneofibular ligament, anterior talofibular ligament, superficial deltoid ligament, plantar fascia.

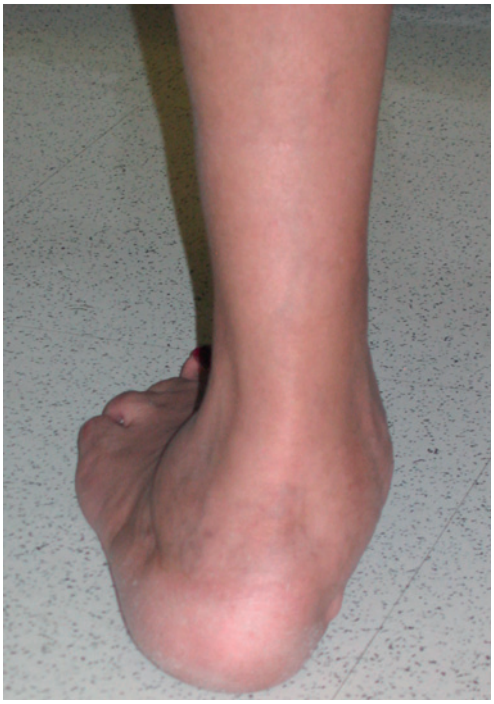
Tendons: Posterior tibialis, flexor hallucis longus, anterior tibialis, peroneal brevis and longus, extensor hallucis longus, Achilles (see Fig. 54-1).

Bursae: Calcaneal, retrocalcaneal.

Bones: Fifth metatarsal base, groove in cuboid for peroneus longus, peroneal tubercle, first metatarsocuneiform joint, navicular tubercle, head of talus, sustentaculum tali, medial malleolus (see Fig. 54-1).

Crepitation

During range of motion: From any degenerative joint surface, fractures, or soft tissue thickening.



Posterior tibial insufficiency.

Figure 54-2 Too Many Toes Test.

Specific Tests

Thompson test: Patient prone with affected leg extended. Squeeze the calf muscles to indirectly plantar flex the foot. A positive test indicated by failure of the foot to plantarflex.

Abduction stress test: Manual abduction of the forefoot while stabilizing the hindfoot. Assess for pain clinically and widening of Lisfranc joint on x-ray.

Calcaneal compression text: Squeeze the posterior heel simultaneously from medially and laterally. Pain is a positive test and suggests stress fracture.

Too many toes sign: View patient from behind and count the toes visible lateral to the heel. The finding of one or two toes visible lateral to the heel is normal; viewing three or more toes is a positive test and confirms forefoot abduction usually seen with posterior tibial tendon or spring ligament dysfunction (Fig. 54-2).

ANATOMIC VARIANTS

See Table 54-1 and Figure 54-3.

FOOT PROBLEMS

Skin and Nail Problems

See Table 54-2, Figure 54-4, and Chapter 34, Skin Problems in the Athlete.

Inflammatory Conditions

See Table 54-3.

Etiology: Stress causes macrotears (acute trauma) or microtears (chronic overuse) with resultant inflammation.

Common types: Posterior tibial, Achilles, peroneal, anterior tibial tendinitis (Fig. 54-5).

Imaging: Plain films usually not helpful; do not show tendon damage. Occasionally see associated bony changes (e.g., avulsion of base of fifth metatarsal at peroneus brevis insertion, fracture or proximal migration of os perineum, calcification at Achilles tendon insertion). Computed tomography (CT), magnetic resonance imaging (MRI), or ultrasound may help detect complete tendon tear.

Plantar Fasciitis

Etiology: Excessive tightness of gastrocsoleus complex pulling into Achilles tendon causes overload at plantar fascia origin on calcaneus during weight-bearing activities; microtears and inflammation ensue (Fig. 54-6).

Symptoms/signs: Point tenderness/pain (particularly on first step in morning), specifically along medial tubercle of calcaneus. Sometimes relieved with movement, returning after rest ("start-up pain") (see Fig. 54-6).

Imaging: Calcaneal spur at origin of flexor digitorum brevis may be seen (secondary finding and usually not a cause of pain) in radiographs.

Table 54-1 ANATOMIC VARIANTS OF THE FOOT

Variants	Description	Etiology	Functional consequence
Tarsal coalition	Bony or cartilaginous bar between bones of hindfoot and midfoot (see Fig. 54-3)	Congenital	Limits eversion/inversion Clinically may appear as rigid flat foot
Pes planus	Flat foot (see Fig. 54-3)	Structural (e.g., tarsal coalition, posterior tibial tendon insufficiency), normal variant	Abnormal stress loading (often associated with hyperpronation)
Pes cavus	High-arched foot, (see Fig. 54-3)	Structural or neurologic disorder	Less shock absorption; abnormal stress loading leads to overuse syndromes and stress overloading
Morton's toe	Elongated second metatarsal compared to first	Congenital	Abnormal stress loading from great toe to second toe; pain and callus formation; functional overuse syndromes

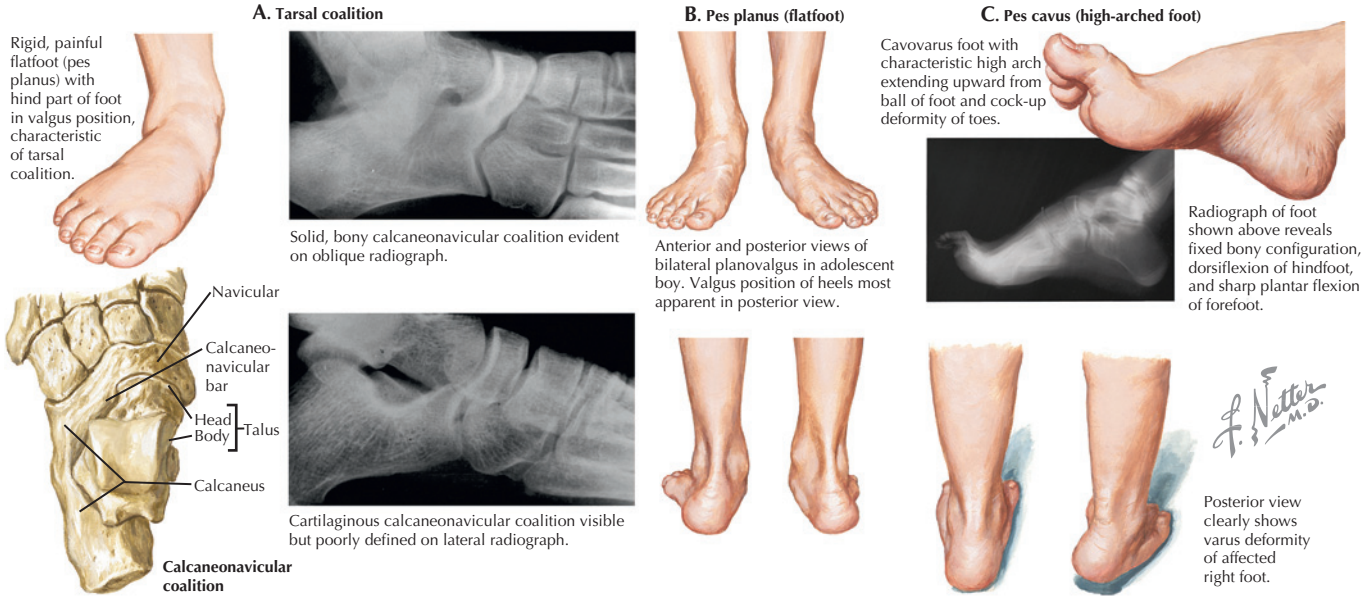


Figure 54-3 Anatomic Variants.

Table 54-2 SKIN AND NAIL PROBLEMS

Type	Cause	Signs and symptoms	Treatment
Calluses and corns	Excessive localized friction or pressure from tight shoes or structural abnormalities of feet, such as hammer toes	Pain; thickening and hardening of skin (soft corns between toes due to moisture)	Proper shoe fitting, padding (e.g., doughnut pad), pumice stone, resection of bony prominences, metatarsal pad, Budin splint
Blisters	Friction, pressure Epidermal/dermal separation by serous fluid	Painful vesicles	Unruptured: sterile dressing, pressure relief Ruptured: sterile cleansing, dressing (consider antibiotics for diabetic patient or signs of infection in any patient)
Warts	Virus (papilloma)	Pain at site; skin thickening with central core; flat or raised	Trichloracetic acid or salicylic acid, liquid nitrogen
Tinea pedis (athlete's foot)	Fungus	Dry or vesicular lesions; scaling, peeling, and cracking fissures in skin; deformed nails, hyphae and buds on KOH wet mount	Dry: miconazole, clotrimazole, terbinafine, salicylic acid Vesicular: wet dressings with Burrow's solution Erythema or other signs of infection: consider antibiotics
Paronychia	Soft tissue infection around the nail	Inflamed nail margin with or without drainage	Warm water soaks, antibiotics; partial nail resection; proper nail cutting techniques
Subungual hematoma	Trauma	Dark blood under nail; pain/pressure at site	Drainage (insert no. 18 needle or drill sterilely through nail) Note: ensure traumatic history to distinguish from subungual melanoma

Differential diagnosis: Entrapment of the first branch of the lateral plantar nerve (thought by some to be component of plantar fasciitis syndrome); plantar fascial rupture; tarsal tunnel syndrome; calcaneal stress fracture; heel pad atrophy.

Treatment: Early intervention more efficacious than late intervention.

- Achilles stretching (see Fig. 54-6).
- Nonsteroidal anti-inflammatory drugs (NSAIDs).
- Shock-absorbing heel pad and/or orthoses (see Fig. 54-6).
- Activity modification to decrease impact activities.
- Ice application/massage to area of greatest tenderness.
- Running shoes for best support of involved heel.
- Dorsiflexion night splint.
- Corticosteroid injection: usually only after more than 6 months of nonoperative treatment, risk of iatrogenic fat pad atrophy or plantar fascia rupture.
- Casting (weight-bearing) of involved extremity in refractory cases.
- Extracorporeal shock wave treatment for refractory cases.
- Surgical release (only with failure of conservative measures generally lasting more than 6 months to 1 year)—results not always successful.

Sever's Disease (Calcaneal Apophysitis)

Etiology: Unilateral or bilateral heel pain in children between 9 and 14 years of age. Contributing factors include decreased Achilles and hamstring flexibility; microtrauma from running sports, especially soccer; biomechanical abnormality contributing to poor shock absorption, such as hallux valgus, pes cavus, and pes planus; occasionally, acute trauma, such as a violent heel strike.

Symptoms/signs: Intermittent or continuous heel pain shortly after beginning new sport or season, worse during and after ac-

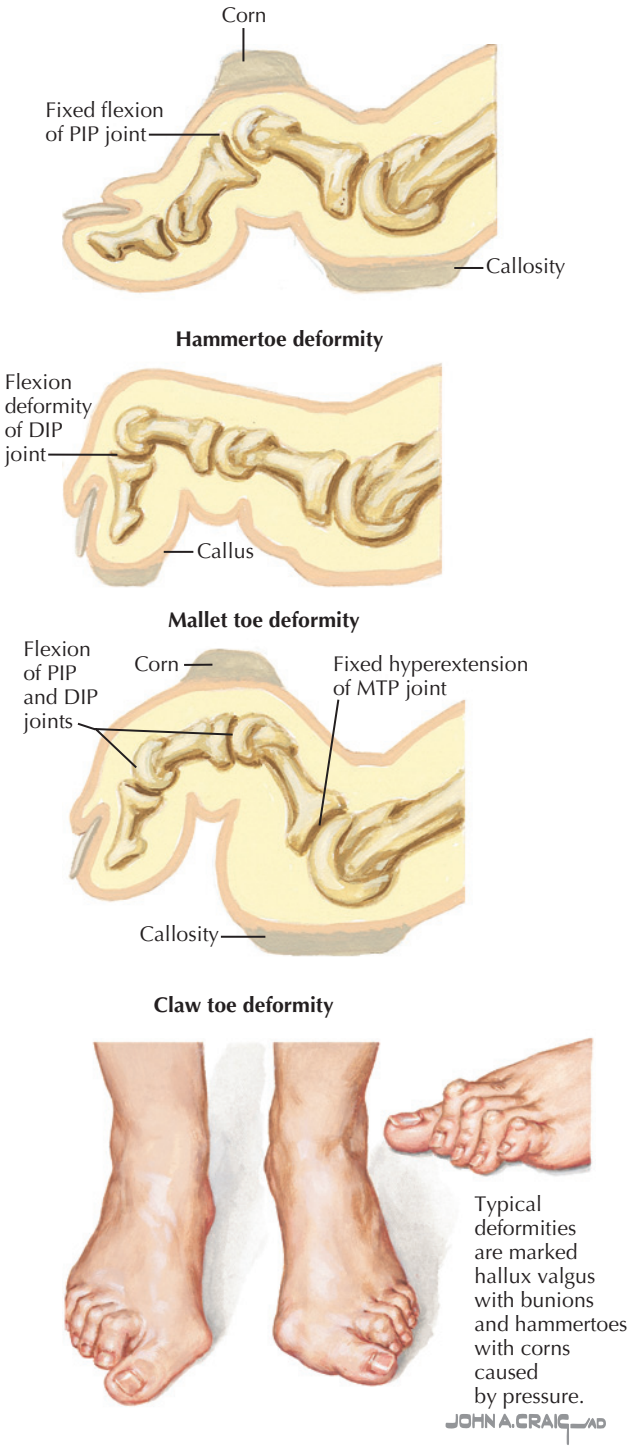


Figure 54-4 Toe deformities. See also Figures 32-1, 32-4, and 32-5.

tivity and improves with rest, tender to palpation at or just anterior to Achilles insertion. Positive "squeeze" test: compression of medial and lateral aspects of calcaneal apophysis produces pain. Positive "Sever's test": heel pain aggravated by standing on tip-toes.

Imaging: Radiographs characterized by fragmentation, sclerosis, and increased density of apophysis that mimic the appearance of osteonecrosis; changes are best seen on the lateral x-ray.

Differential diagnosis: Achilles tendinitis/strain, heel pad pain, retrocalcaneal bursitis, calcaneal stress fracture, plantar fasciitis.

Treatment: Start with rest (crutches in severe cases), activity modification, ice, heel lifts or cups, stretching and strengthening, NSAIDs. If resistant still—dorsiflexion night splint, and rarely, short leg cast for 2 weeks, orthoses.

Turf Toe

Etiology: Hyperextension of metatarsophalangeal joint (MTPJ) of great toe leads to a spectrum of injuries from plantar plate tear to dislocation (Fig. 54-7).

Mechanism of injury: Most common is axial load on a foot fixed in equinus with MTPJ in hyperextension (e.g., football linemen during push-off).

Symptoms/signs: Pain, tenderness, and swelling at great toe MTP joint; positive great toe Lachman test (see Fig. 54-7). **Assess the contralateral side.**

Imaging: Weight-bearing (WB) anteroposterior (AP), oblique, sesamoid view, comparison AP x-rays; usually normal. Look for proximal migration of sesamoids consistent with plantar plate rupture (see Fig. 54-7). Consider dorsiflexion lateral or fluoroscopy to evaluate tracking of the sesamoids. With negative x-rays but high suspicion, consider MRI.

Differential diagnosis: Phalangeal or metatarsal fracture; osteochondral injury, hallux rigidus, osteoarthritis, gout.

Treatment: Rest and protection are key: ice/NSAIDs acutely, taping to limit motion of MTPJ can assist in return to activity, walking boot, metatarsal pad to unload first metatarsal may be helpful, rigid shoe or rigid insert provides forefoot support (limits MTP hyperextension) (see Fig. 54-7). Surgery considered in unstable joint, sesamoid fracture, traumatic bunion, or chondral injury with loose body.

Hallux Valgus (Bunion)

Etiology: Occurs nine times more frequently in females than males; may be secondary to increased ligamentous laxity in females; may be component of improper shoe fit, congenital, posttraumatic, and/or inflammatory conditions.

Symptoms/signs: Usually secondary to bunion. Tenderness over medial eminence from pressure and friction against shoe. Bursa may become inflamed, thickened, and painful (Fig. 54-8). Secondary deformities (e.g., hammertoes, callosities, metatarsalgia) may be additional sources of pain and discomfort.

Imaging: Standing AP, lateral, oblique radiographs. Measure the first-to-second intermetatarsal angle by drawing a line through center of first metatarsal from head through the base. Similar line should be drawn down the second metatarsal. The point where these angles intersect proximally is the intermetatarsal angle (see Fig. 54-8). Normal is less than 9 degrees; greater than 15 degrees indicates metatarsus primus varus. Measure hallux valgus angle formed by lines that bisect the proximal phalanx and first metatarsal shaft. Normal is less than 15 degrees.

Treatment: Conservative measures to provide pain relief; properly fitting shoes with adequate width in forefoot; pads to cushion bunion; metatarsal pads and orthoses to help redistribute weight; toe spacer. Surgery is reserved for cases that fail conservative management; should be avoided in sprinters, high jumpers, pole vaulters, and ballet dancers if pain is tolerable because of possible decreased MTP range of motion after surgery. Wait until end of athlete's career if possible.

Hallux Rigidus (Painful MTPJ with Loss of Motion)

Etiology: Limitation of motion, especially dorsiflexion; mechanical block due to periarticular osteophytes.

Common causes: Posttraumatic, chronic turf toe, repetitive microtrauma (runners), primary arthrosis. Juvenile hallux rigidus (rare) is secondary to osteochondritis dissecans.

Symptoms/signs: Tender and enlarged first MTP joint; decreased first MTP dorsiflexion with pain; palpable bony ridge; pain

Table 54-3 INFLAMMATORY CONDITIONS

Type	Signs and symptoms	Treatment
Posterior tibial tendinitis	Medial arch pain, swelling, pain with resisted inversion, painful or unable to single heel rise, medial arch collapse, "too many toes sign": when viewed from behind, abducted forefoot allows more toes to be seen on affected side.	Initial: rest, ice, NSAIDs, orthoses with arch support and medial heel wedge. Rehabilitation:* plantarflexion and inversion strengthening exercises, heel cord flexibility. Surgical: treatment ranges from osteotomy with tendon transfer to arthrodesis.
Peroneal tendinitis	Lateral pain especially with active and resisted eversion; swelling; may complain of snapping sensation in cases of peroneal tendon subluxation. May be due to underlying tendon tear.	Initial: rest, ice, NSAIDs, orthoses, cast or boot. Rehabilitation:* eversion strengthening exercises. Surgery may be required for recalcitrant cases, tears, or subluxation.
Anterior tibial tendinitis	Pain in anterior medial foot, worse with active dorsiflexion; swelling and crepitation. Seen in runners, hikers, and racquet sports.	Initial: activity modification, ice, NSAIDs, walking boot. Rehabilitation:* dorsiflexion strengthening exercises.
Achilles tendinitis	Decreased gastrocnemius flexibility; pain, tenderness, swelling, and crepitation; pain with active plantarflexion. X-rays to look for Haglund's deformity or insertional calcifications.	Rule out systemic conditions such as gout or spondyloarthropathy. Initial: rest, ice, NSAIDs, heel pad. Rehabilitation:* heel cord stretching and strengthening exercises. Possible surgical debridement in chronic cases.
Retrocalcaneal bursitis	Posterior heel/ankle pain; tenderness and swelling in bursa located between the Achilles tendon and the calcaneus.	Initial: rest, ice, NSAIDs, heel cup, shoe modification. Rehabilitation:* heel cord stretching, modalities, strengthening exercises.
Sesamoiditis	Generic term. Local tenderness (tibial side most common); pain worse with weight bearing. Rule out avascular necrosis, sesamoid fracture. Bipartite sesamoid reported 10% tibial, rare fibular.	Reduce weight-bearing stress at site; orthoses with sesamoid relief or Morton's extension. Ice, NSAIDs, use of stiff-soled shoe or walking boot. If conservative treatment fails: bone scan, computed tomography, or MRI to rule out stress fracture.
Metatarsophalangeal joint synovitis	Pain at metatarsophalangeal joint (second most common), positive Lachman test (increased anterior-posterior translation), joint crepitus. Distinguish from interdigital neuroma.	Metatarsal pad, figure-of-eight taping, Budin splint, NSAIDs, steroid injection (with caution), surgery for failed nonoperative treatment.

*Rehabilitation includes maintenance of aerobic, well-leg, and upper-body fitness. Physical therapy modalities, such as phonophoresis and iontophoresis, also may be useful.
NSAIDs, nonsteroidal anti-inflammatory drugs.

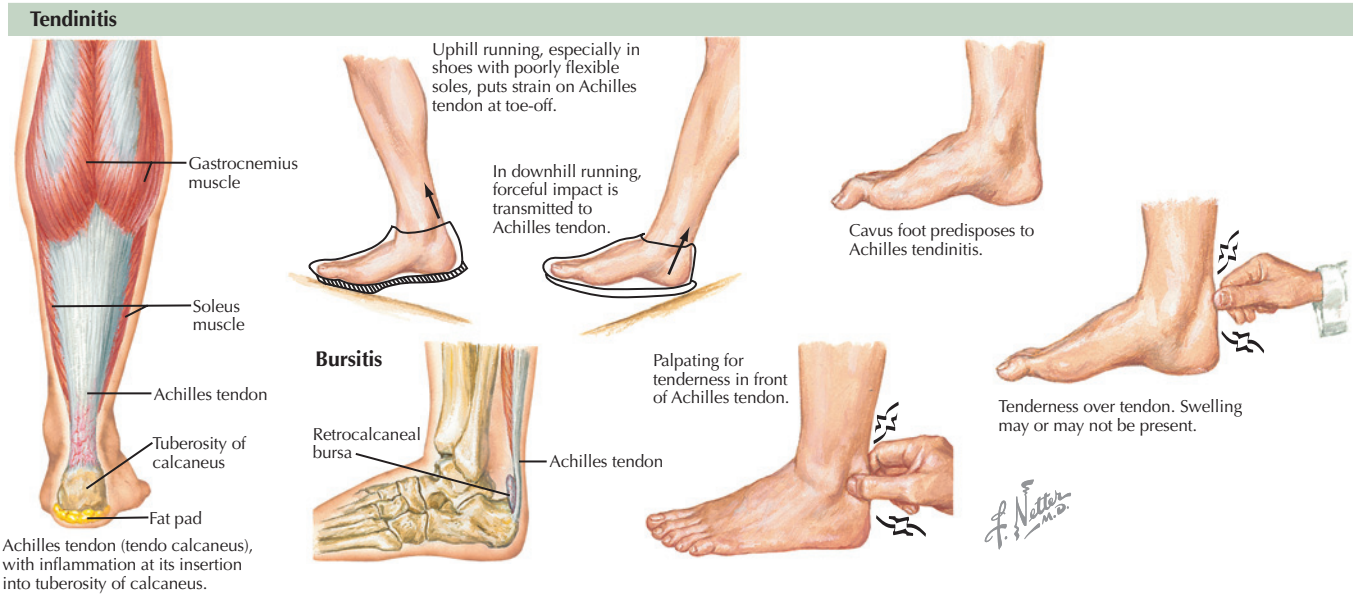


Figure 54-5 Achilles Tendinitis and Retrocalcaneal Bursitis.

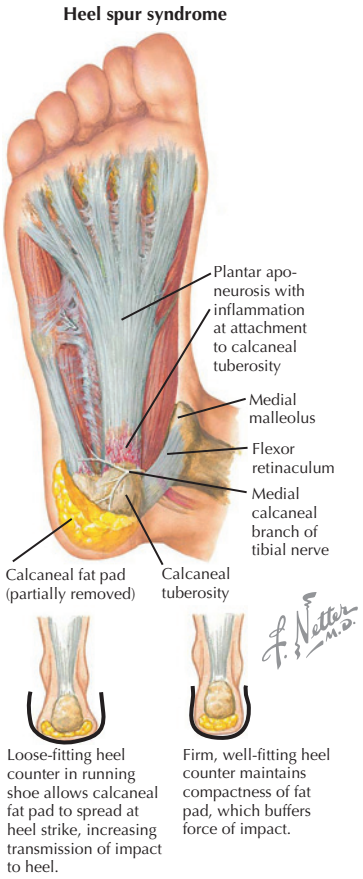


Figure 54-6 Plantar Fascia.

increased with running, incline training, and wearing shoes with elevated heel (Fig. 54-9).

Imaging: WB AP, lateral, oblique; may be normal initially; nonuniform narrowing of first MTPJ with widening and flattening of the metatarsal head, loose body, marginal osteophytes; dorsal exostosis of first metatarsal is the hallmark (see Fig. 54-9).

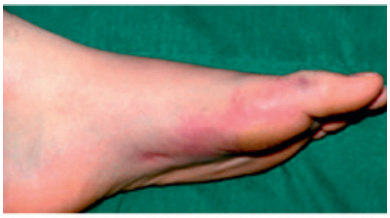
Treatment: Shoe modifications (stiff sole, rocker bottom, extra-depth toe box), metatarsal bar, NSAIDs, orthoses with Morton's extension, intra-articular steroid injection. Surgical options include cheilectomy or fusion to relieve impingement and decrease pain; dorsiflexion osteotomy of proximal phalanx (Moberg procedure) may benefit a running athlete by improving dorsiflexion movement.

Talus Fractures

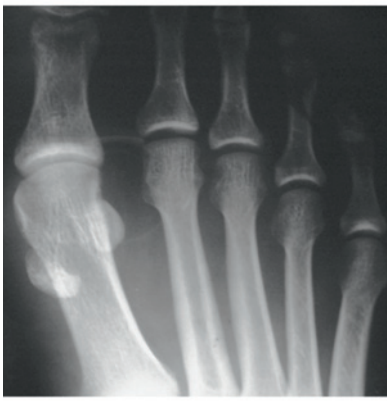
Etiology:

- **Acute trauma:** Rare in sports, usually surgical emergency; after radiographic confirmation, orthopedic referral mandatory. Talar neck fractures are associated with severe foot dislocation (Fig. 54-10). *Note:* Osteochondral fractures of the talar dome may occur with ankle sprains.
- **Stress fractures:** Also rare. Symptoms/signs include diffuse midfoot pain, anterior ankle tenderness on palpation, and painful active and passive motion of subtalar joint. *Note:* Subtalar joint allows eversion/inversion.
- **Lateral process fractures:** "Snowboarder's fracture." Frequently misdiagnosed as a lateral ankle sprain. Mechanism is acute dorsiflexion with inversion of foot and axial load.

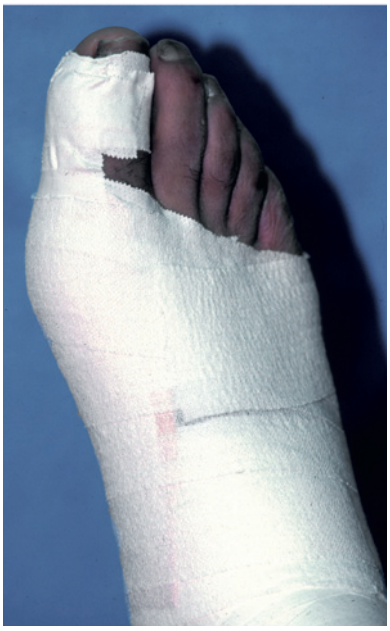
Imaging: Radiographs usually give definitive diagnosis. Subtle or stress fractures usually require bone scan or CT. Avascular necrosis of talar dome can occur after stress or traumatic fractures.



A. Turf toe clinical appearance.



B. Proximal sesamoid migration.



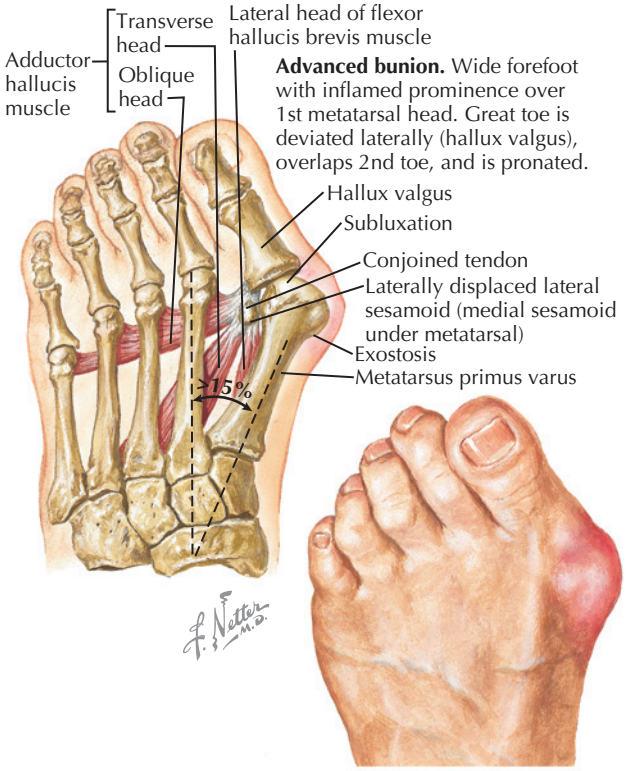
C. Taping to limit motion of metatarsophalangeal.

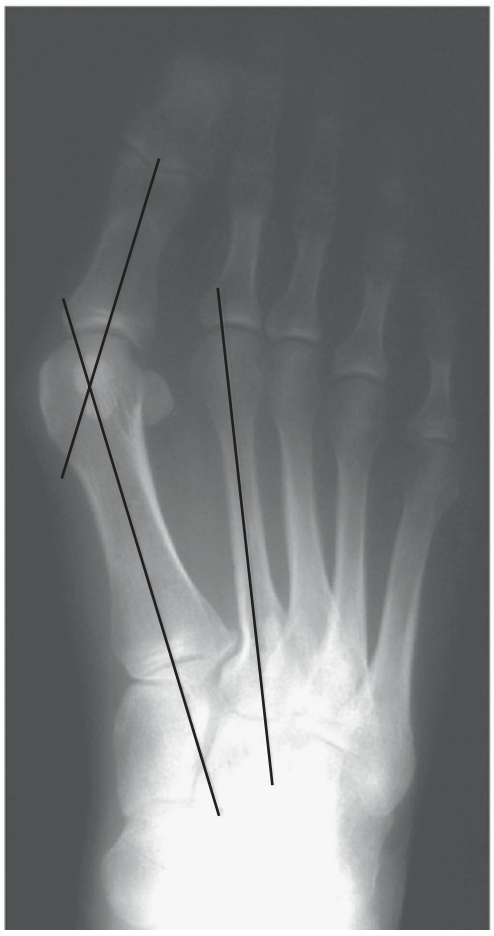
Figure 54-7 Turf Toe.

Lateral process fractures seen best on coronal CT images (see Fig. 54-10).

Treatment:

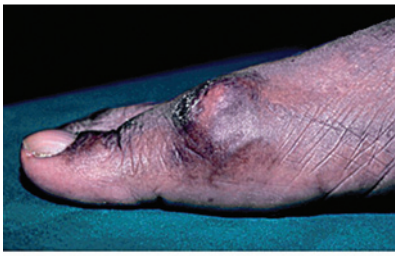
- **Stress fractures:** Modified rest/cessation of weight-bearing activity; cast or support shoe, crutches. Healing can take up to 6 months (repeat radiographs should document healing). Progressive return to weight-bearing activity with confirmation of healing. Limitation of ankle and subtalar motion on return to activities (e.g., ankle brace).
- **Lateral process fractures:** Treatment determined by fragment size, comminution and displacement. Nondisplaced—non–weight-bearing boot or cast for 6 weeks. Displaced—



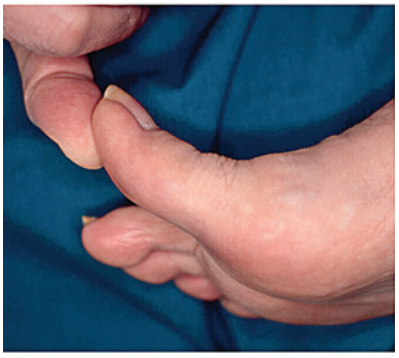


Intermetatarsal and hallux valgus angles.

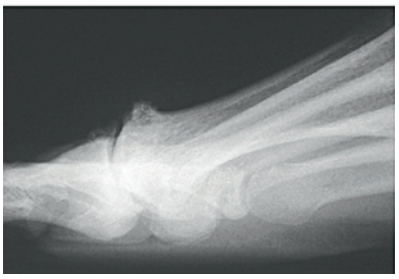
Figure 54-8 Hallux Valgus.



A. Clinical appearance.



B. Loss of dorsiflexion.



C. Dorsal osteophyte.

Figure 54-9 Hallux Rigidus.

surgical (open reduction, internal fixation [ORIF], if possible), otherwise excision of fragment(s).

Calcaneus Fractures

Etiology: Traumatic, rare; s**tress,** more common; usually related to endurance sports (Fig. 54-11).

Symptoms/signs: Localized pain in heel, accentuated by weight bearing; mild swelling; pain with medial-lateral compression of calcaneus.

Imaging: Lateral of calcaneus and heel axial x-rays: Bohler's angle may be less than 20 degrees in traumatic fractures. **Stress fracture:** May see line at posterior-superior aspect of the calcaneus perpendicular to the trabeculae (see Fig. 54-11). If negative, bone scan or CT. Assess for posterior process fracture of talus.

Treatment: Stress fractures: Modified rest with cessation of impact activities; cast or walking boot with crutches may be appropriate. Gradual return to activity when clinical symptoms abate and serial radiographic assessment documents healing. Full healing usually takes 2 to 3 months. Consider shock-absorbing orthotic device on return to activity. **Traumatic:** Closed or open treatment depending on severity of fracture.

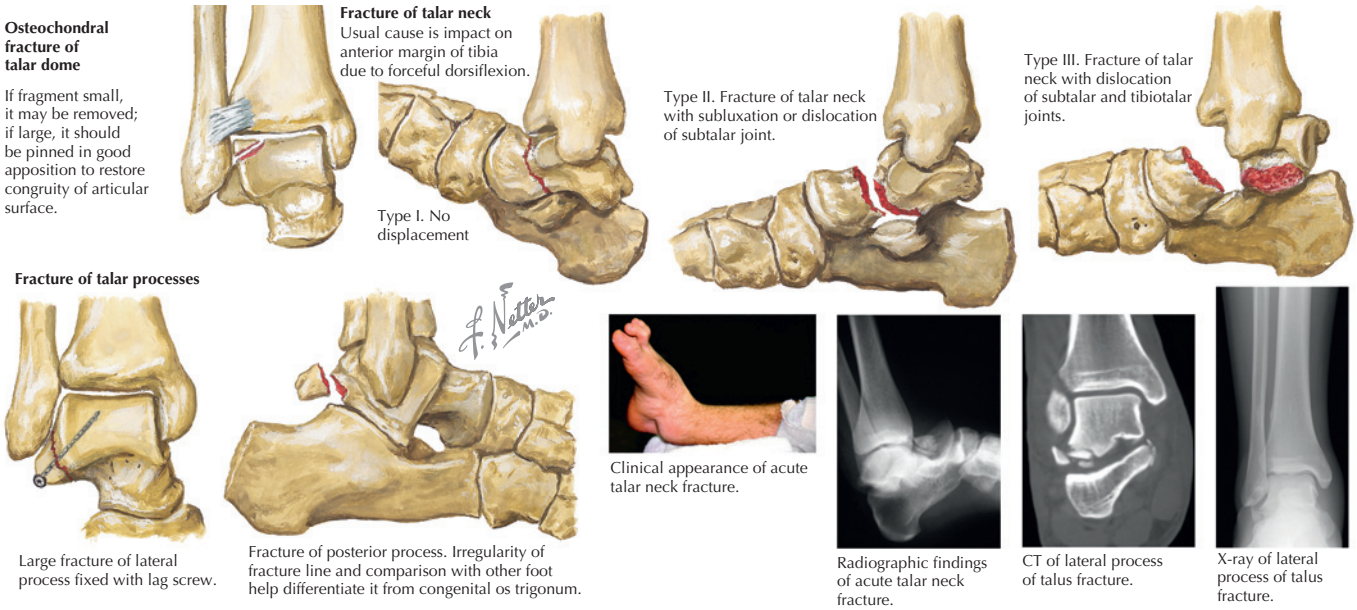


Figure 54-10 Talus Fractures.

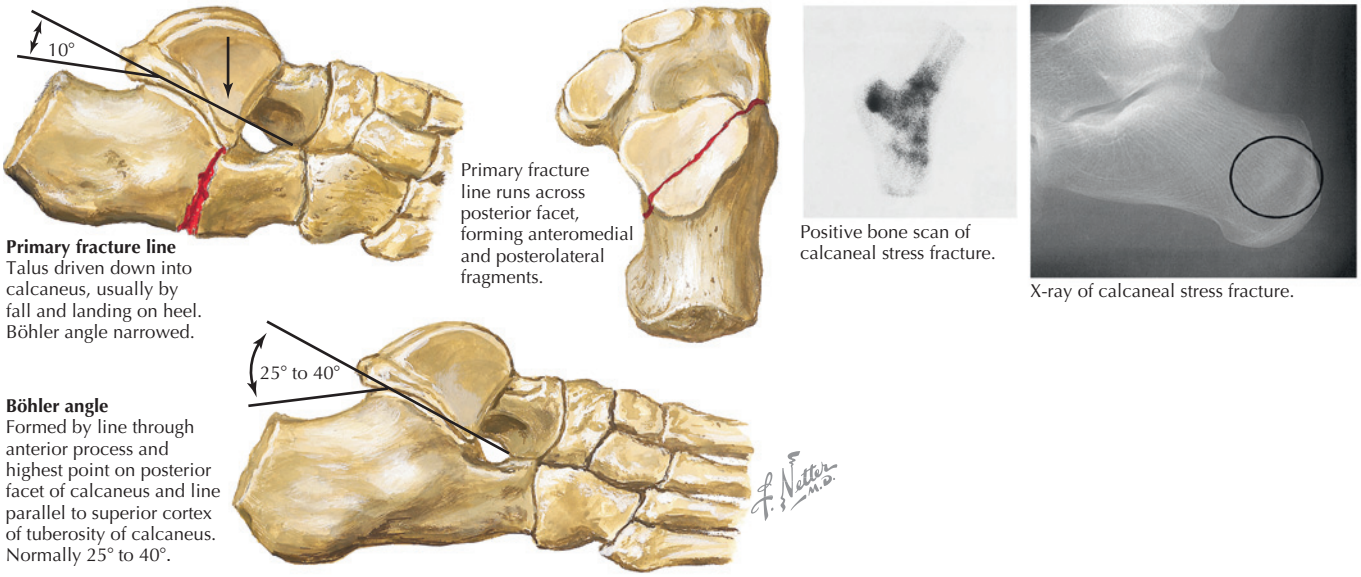


Figure 54-11 Calcaneus Fractures.

Midfoot Fractures

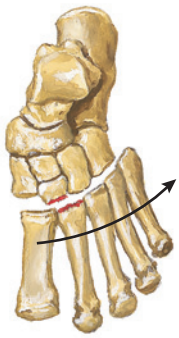
Lisfranc Injury

Etiology: Traumatic; 3 common mechanisms: twisting of forefoot (e.g., equestrian foot caught in stirrup during fall); axial load with foot in equinus (e.g., football, soccer); crush injury (Fig. 54-12). Lisfranc ligament runs from plantar-medial cuneiform to the base of the second metatarsal. No intermetatarsal ligament between the first and second metatarsals. Key is recognition of the injury.

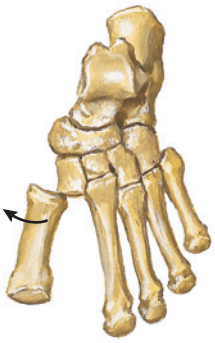
Symptoms/signs: Pain and inability to bear weight, swelling and gross deformity if severe, may have spontaneous reduction after injury.

Imaging: Weight-bearing (if possible) AP and lateral plus oblique with comparison views. Normal alignment: medial border of second metatarsal parallels the medial border of the middle cuneiform on AP view; medial border of fourth metatarsal aligns with medial border of the cuboid on oblique x-ray; first and second intermetatarsal spaces should be continuous with respective intertarsal spaces (see Fig. 54-12). Look for widening between medial and middle cuneiform and abnormal alignment at naviculocuneiform joint. CT or MRI if suspicion high and x-rays are negative, but keep in mind there are non weight-bearing studies.

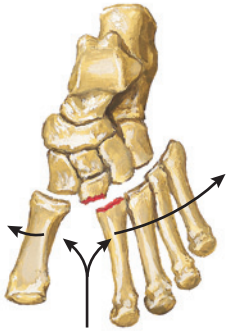
Treatment: Truly nondisplaced or sprains can be placed in non–weight-bearing cast. Displaced injuries require closed or open reduction and internal fixation using 4.0- or 4.5-mm screws.



Homolateral dislocation. All five metatarsals displaced in same direction. Fracture of base of 2nd metatarsal.

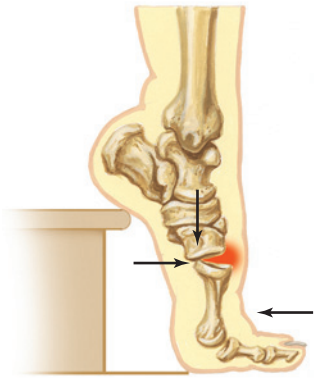


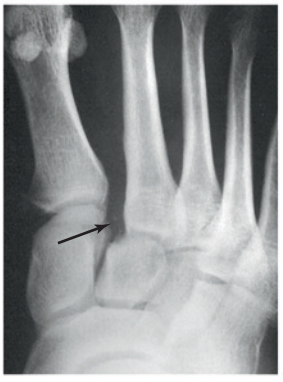
Isolated dislocation. One or two metatarsals displaced; others in normal position.



Divergent dislocation. 1st metatarsal displaced medially, others superolaterally.

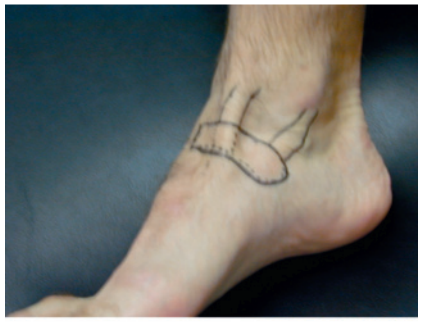
Dorsolateral dislocation often best seen in lateral view.



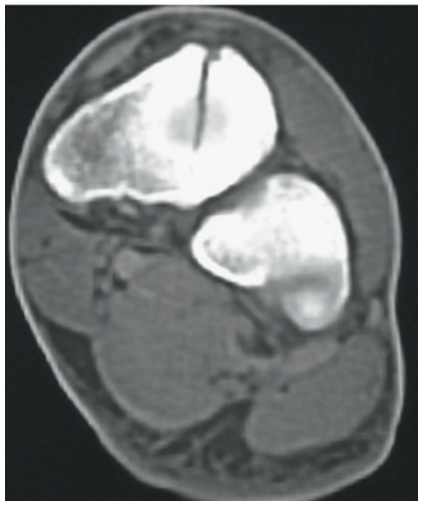


Lisfranc disruption x-ray.

Figure 54-12 Lisfranc Injury.



A. N-spot.



B. CT of fracture.

Figure 54-13 Navicular Stress Fracture.

Navicular Stress Fracture

Etiology: Common in running and jumping athletes; repetitive cyclic loading, explosive push-off. Unique vascular anatomy results in relatively avascular central one-third. Frequently missed, delay in diagnosis up to 4 to 7 months reported.

Symptoms/signs: Ill-defined midfoot to anterior ankle soreness and cramping. Insidious onset, progresses to frequent pain with activity. Pain with percussion over navicular, N-spot tenderness to palpation, symptoms exacerbated with single leg hop (Fig. 54-13).

Imaging: Foot x-rays usually negative. Bone scan highly sensitive, uptake seen in all three phases. CT: gold standard, defines location, completeness, direction and displacement (see Fig. 54-13). Fracture line usually proximal-dorsal to distal-plantar.

Treatment: Non–weight-bearing short leg cast for 6 to 8 weeks. If remains tender to palpation, replace in cast, otherwise progressive return to activity. Persistent symptoms, delayed union, or displaced fractures require surgery (internal fixation with or without bone grafting). Recovery can take up to 1 year.

Metatarsal Fractures

Etiology: Trauma: direct blow to the foot. Stress: common; seen in endurance running and jumping sports. *Note:* Presence of stress fracture in adolescent female may alert you to look for other components of female triad (anorexia, amenorrhea, and osteoporosis).

Symptoms/signs: Localized pain with weight-bearing, swelling, tenderness.

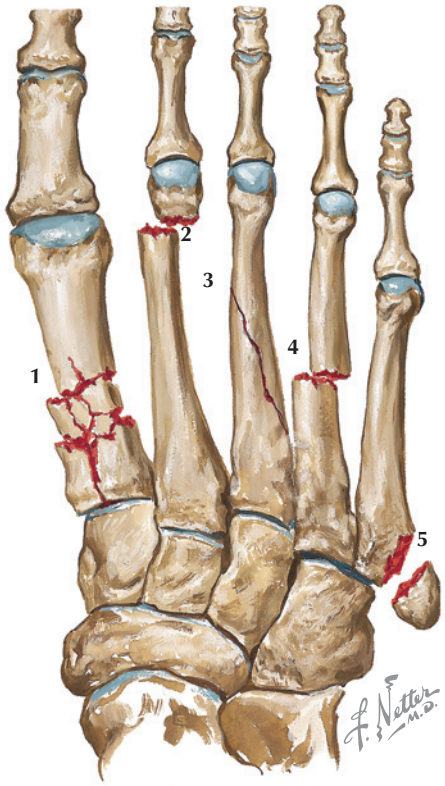
Imaging: Plain x-rays may be negative; bone scan or MRI may be necessary to detect early lesions (Fig. 54-14).

Treatment:

- **Second through fourth metatarsal stress fractures:** Nonoperative treatment (first metatarsal stress fracture rarely seen). Modified rest with cessation of weight-bearing activities, immobilization in cast or boot for 3 weeks may be used, especially for pain control). Gradual return to activity when symptoms subside. Base of second metatarsal may go on to delayed or nonunion and require bone grafting and/or ORIF.
- **Avulsion fracture of the base of the fifth metatarsal (common with ankle sprains):** Nonoperative treatment.

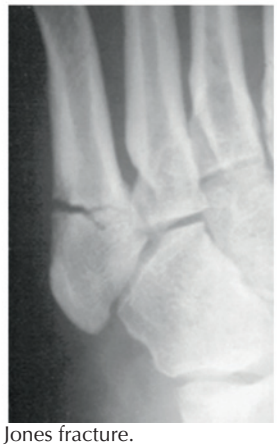
Jones Fracture

Etiology: Stress fracture of fifth metatarsal at metaphyseal-diaphyseal junction in the region of or just distal to the fourth-to-fifth metatarsal articulation (see Fig. 54-14). Frequently seen in sprinters and jumpers. Often have difficulty healing because of poor blood flow.



Fractures of the metatarsal bones.

1. Comminuted fracture
2. Fracture of neck
3. Oblique
4. Transverse
5. Avulsion of tuberosity of 5th metatarsal



Jones fracture.

Figure 54-14 Metatarsal Fractures.

Treatment: Generally cast for 4 to 6 weeks followed by 4 to 6 weeks in walking boot. Approximately 75% will heal with nonoperative treatment but 30% to 50% will refracture. Indications for surgical stabilization with intramedullary screw are controversial but considered for acute fracture in athletes, nonunion, refracture, and cavovarus foot with lateral overload. Bone grafting considered when treating delayed union or nonunion.

Phalangeal Fractures

Etiology: Trauma such as crush, direct blow, or jamming.
Symptoms/signs: Pain, deformity, swelling, ecchymosis.
Imaging: Plain films confirmatory.
Treatment: Conservative unless open fracture or intra-articular fracture of great toe with displacement.
- **Nondisplaced:** Buddy taping to adjacent toe and protection in stiff-soled shoe; symptoms dictate activity restrictions.
- **Displaced:** Reduced by manipulation; subsequent buddy-taping/splinting.
- **Complications:** Intra-articular fractures can result in joint stiffness and arthritis.

Subtalar/Pantalar Dislocations

Etiology: Severe trauma, orthopedic emergency.
Treatment: Immediate care indicated to avoid neurovascular damage.

Metatarsophalangeal/Interphalangeal Dislocations

Etiology: Trauma, relatively common.
Symptoms/signs: Pain, gross deformity of toes.
Imaging: Radiographs to rule out associated fracture.
Treatment: Reduction with steady traction; if dislocation persists, orthopedic referral to rule out tendon entrapment, which can require surgical treatment. Splint/buddy-taping after reduction. Functional toe can usually be expected.

Neurologic Injury

Tarsal Tunnel Syndrome

Description: Impingement of posterior tibial nerve beneath flexor retinaculum behind medial malleolus.
Etiology: Trauma (fractures); compression from space-occupying lesion (e.g., ganglion); systemic disorders (e.g., diabetes mellitus); biomechanical dysfunction (e.g., hindfoot valgus, tarsal coalition); idiopathic.
Symptoms/signs: Aching pain at medial foot, aggravated by prolonged weight bearing; positive Tinel's sign (tapping over tibial nerve causes radiating pain along nerve distribution); Valleix phenomenon (neural percussion causes radiation of pain proximally along course of nerve).
Diagnostics: Radiographs obtained to assess for malunion, exostosis, etc. MRI visualizes soft tissue space-occupying lesions. Electromyography (EMG) and nerve conduction studies are abnormal in 80%; however, with a supportive history, physical exam, and normal electrodiagnostic studies, tarsal tunnel syndrome is not excluded.
Differential diagnosis: Posterior tibial tendinitis, calcaneal stress fracture, gout, plantar fasciitis, herniated lumbar disc.
Treatment: Rest, NSAIDs, graduated return to activity; orthoses; local steroid injection; surgical decompression for intractable cases. Best results with surgical decompression in cases caused by space-occupying lesion.

Morton's Neuroma

Description: Impingement of interdigital nerves as they bifurcate at metatarsal heads.
Etiology: Trauma or repetitive stress leads to chronic irritation of nerves as they cross under transverse metatarsal ligament to toes. Fusiform swelling and pathologic changes occur in nerves. Histology of perineural fibrosis.
Symptoms/signs: Pain (typically burning neuralgia with radiation into involved toes); symptoms worse with activity and narrow toe-box shoes; tender to palpation between metatarsal heads; classically located between third and fourth metatarsals but can also occur between second and third. Sensation of "rock in the shoe," relieved when barefoot. Mulder's sign: squeeze the forefoot from medial to lateral while palpating the web space, positive test is a click or gritty feeling.
Differential diagnosis: Metatarsalgia, metatarsal stress fractures, MTPJ synovitis, neuropathy.

Radiographic assessment: Usually negative; evaluate for stress fracture, compression from exostosis.
Treatment: Initial: NSAIDs, metatarsal pads, wide toe-box shoes, lower heel, corticosteroid injection. **Chronic:** Surgical excision; patient left with permanent anesthesia between involved toes, but no functional deficits. Recurrence from stump neuroma may occur.

Reflex Sympathetic Dystrophy

Description: Pain syndrome accompanied by evidence of autonomic dysfunction.
Etiology: Type I: unknown etiology, insidious onset; **Type II** can occur after injury or trauma. Believed to result from dysfunction of autonomic nervous system. International Association for Study of Pain (IASP) proposed term **complex regional pain syndrome** in 1991.
Symptoms/signs: Onset is heralded by severe, diffuse, unrelenting pain with exquisite tenderness to even light touch. Decreased ROM and autonomic vasomotor signs, including warm or cool skin temperatures and decreased peripheral pulses. Skin may be moist to sweaty or dry to scaly with discoloration and swelling. Pain is usually out of proportion to original injury.
Imaging: Radiographs may reveal diffuse osteoporosis of involved part. Bone scan may show delayed pattern of diffuse increased tracer throughout foot, with juxta-articular accentuation of tracer uptake.
Differential diagnosis: Peripheral nerve injuries, polymyositis, lupus, Raynaud's disease, gout, thrombophlebitis.
Treatment: Prompt diagnosis and therapy improve chance of permanent relief. Sympathetic blockade relieves pain. Physical therapy includes vigorously active exercises, weight-bearing activities, and direct stimulation of skin. Adjuvant pharmacologic agents may include antidepressants, sedative-hypnotics, anxiolytics, anticonvulsants, narcotic analgesics, and corticosteroids. Psychological evaluation is frequently required in more difficult cases. Occasionally, chemical or surgical sympathectomy is required.

RECOMMENDED READINGS

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3. Diehl JJ, Best TM, Kaeding CC: Classification and return-to-play considerations for stress fractures. Clin Sports Med 25:17-28, 2006.
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Taping and Bracing

Thomas A. Frette and Michael E. Roberts

TEAM PHYSICIAN'S ROLE IN TAPING AND BRACING

- **Determine appropriateness** of taping/bracing.
- **Facilitate selection process.**
  - **Identify** available options.
  - **Communicate** with treatment team: certified athletic trainer, coach, athlete.
- **Evaluate effectiveness** of selected support.

SELECTION CONSIDERATIONS

Diagnosis of Injury

Location: Taping/bracing restricts undesired motion. It is contraindicated when restriction of motion may lead to decreased function or other problems.

Nature of injury: Taping/bracing provides support to acutely injured tissues and decreases effect of repetitive biomechanical forces that result in chronic injuries.

Severity: More severe injuries require more aggressive treatment.

Goals of Taping/Bracing

Prophylactic: To reduce incidence or severity of injury to uninjured normal anatomy or fully rehabilitated injuries.

Rehabilitative: To provide protection of healing injuries during rehabilitation. **Taping/bracing does not substitute for or replace need for complete rehabilitation. It is only an adjunct to rehabilitation.**

Functional: To protect against reinjury after rehabilitation or surgical reconstruction.

Resources Available

- Taping supplies are usually available. Requires application by certified athletic trainer or other skilled person; may become costly over repeated applications.
- Over-the-counter braces are instantly available, less expensive, and more time-effective; custom braces require time for custom fabrication and are more costly than their OTC counterparts. Once instructed, athlete can self-apply. Initially costly, but may be **cost-effective** over time; consult coaches and parents.
- **If taping or bracing cannot provide additional benefit, it is probably best to do without it.**

Sport and Position of Athlete

- Taping is often tailored to meet physical requirements by allowing the athlete to perform his or her skills.
- Equipment, including footwear, may need modification.
- Environment may affect choices: temperature, humidity, aquatic.
- **Use materials that do not endanger other participants.**

Athlete's Acceptance

- Taping and bracing must not be uncomfortable or decrease performance.
- Involve athlete in decision-making process. Provide choices when possible.
- Realize positive psychological effect of taping/bracing (assists athlete's confidence on returning to competition).

Research Findings

Few studies are available. Effectiveness of taping/bracing techniques used by many athletes is supported only by clinical experience or tradition. New products should be viewed with open but critical mind.

Personal Preferences

Most physicians and certified athletic trainers develop list of favorite techniques and devices, but each case should be viewed individually.

IMPLEMENTATION

Communication: Selection process, decision, and plan involves many people: physician, athlete, coach, certified athletic trainer, sports physical therapist, orthotist, parent(s) or guardian(s).

Education:

- **Taping:** Teach certified athletic trainer or coach desired technique as well as methods for prevention of skin problems (see "Skin Care").
- **Bracing:** Teach correct application and function of brace; give written directions on application and care.
- **Promote concept of "earning taping/bracing" by insisting on complete rehabilitation before resumption of sport/activity.**

Follow-up: Usually occurs at conclusion of athlete's season. Reassess injured area; encourage "prehabilitation" of deficits before next season. Re-evaluate effectiveness of method of support; other alternatives may be needed for multisport athletes. Follow-up also provides opportunity to collect data for greatly needed research.

PRINCIPLES OF TAPING

Preparation

Decide on appropriate technique. Gather needed tape supplies. Place athlete's body part in position of function and/or protection. Use appropriate table height to optimize taper's body mechanics.

Tape Selection

- Qualities of good athletic tape: good adherence to skin; adequate tensile strength to provide necessary support; allows perspiration to escape; easily unwinds and tears from roll.
- Size of body part determines appropriate width.
- Elasticity allows increased ease of application and desired movement, yet provides adequate injury protection. Many elastic tapes do not tear and require cutting with scissors; elastic tapes are also more expensive.

Skin Care

Preventive measures:

- **Shave hair:** Increases adhesion of tape, reduces irritation and build up of residue.
- **Apply taping base** (e.g., tincture of benzoin): Increases adhesion of tape, provides protective layer between tape and skin.
- **Apply tape underwrap** (e.g., thin polyester urethane foam): Decreases skin problems, increases athlete's comfort; may not be appropriate for all uses.
- **Apply lubricant to possible areas of irritation** (e.g., lace and heel areas of ankle).

Proper tape removal: Use scissors or cutters with blunt tip. Teach athlete proper removal technique. Cleanse skin to remove tape residue. Treat skin irritations and wounds promptly; these problems can prevent further taping.

Allergic reactions to tape materials: Recognize and treat problems. Consider alternative tape supplies. Investigate other forms of support and protection.

Application

- **Requires skill; proficiency results from practice.**
- **Elements of proper taping technique:**
  - Tearing tape is basic skill; tape must be torn often.
  - Every piece of tape should have distinct purpose.
  - Place anchor strips proximal and distal to injured area directly on skin.
  - Bridge across injury; duplicate anatomy needing support.
  - Weave strips to add strength, overlapping by at least one-half the width of tape.
  - Adapt two-dimensional tape to three-dimensional body part.
  - Limit pressure around body prominences, especially when vascular and neural structures are superficial.
  - Use elastic materials over muscle bellies to allow normal muscle expansion.
  - Inspect for and tape over any gaps in taping to prevent blisters and tape cuts.
- Avoid common problems that restrict circulation: applying too much tape; applying repeated circumferential strips without tearing between turns about body part; forcing tape to go in desired direction. Do not tape acute injuries; swelling causes tightness.

SELECTED TAPING PROCEDURES

Buddy Taping

Common indications: Finger sprains, minor fractures of proximal interphalangeal joint.
Materials: ¼-inch tape, felt or foam strip (optional).
Athlete position: Felt or foam strip placed between injured and adjoining finger; avoid using index or little finger as splint (Fig. 55-1).

Thumb Figure-of-Eight

Common indication: Hyperflexion injuries to metacarpophalangeal (MCP) joint of thumb.
Materials: ¼-inch tape, cloth or elastic.
Athlete position: Thumb abducted, wrist in slight extension (see Fig. 55-1).
Technique: Encircle wrist and thumb in figure-of-eight pattern; repeat as necessary.

Thumb Check-rein

Common indications: Hyperextension or hyperabduction injuries to MCP joint of thumb; can result in injuries to MCP joint of index finger.
Materials: ¼-inch and/or ½-inch tape.
Athlete position: Thumb slightly abducted (see Fig. 55-1).
Technique: Encircle proximal phalanx of thumb and proximal phalanx of index finger. Press together adhesive surfaces between thumb and index finger. Anchor by taping around checkrein.

Wrist Taping

Common indications: Wrist sprains, dorsal impingement.
Materials: 1-inch or 1½-inch tape; foam pad (for dorsal impingement).
Athlete position: Wrist slightly extended, pad on dorsum of wrist if needed to act as "block" to extension range of motion. "Fan" of tape or volar or dorsal surface to prevent flexion or extension (see Fig. 55-1).

Elbow Taping

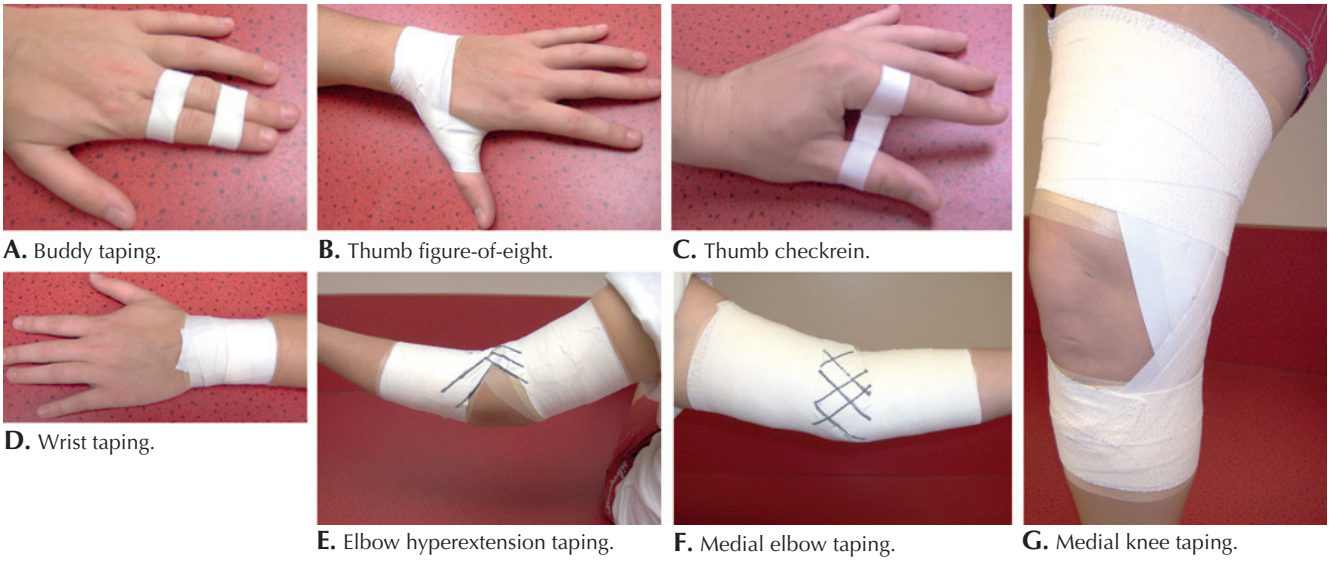
Common indications: Elbow hyperextension, varus/valgus injuries.
Materials: 1½-inch cloth tape or 2-inch elastic tape.
Athlete position: Slight flexion of elbow.
Technique for hyperextension injuries: Place anchor strips about midupper arm and midforearm (see Fig. 55-1). Criss-cross strips in "X" pattern, creating fan across anterior aspect of elbow. Repeat anchors to close, taking care not to restrict circulation.
Technique for varus/valgus injuries: modified to provide medial lateral support (see Fig. 55-1).

Shoulder Taping

- Restriction of motion required to provide adequate support decreases function.
- Time and effort are probably better spent on rehabilitation.

Hip, Hamstring, and Groin Taping

Restriction of motion required to provide adequate support decreases function. Recommend use of elastic wraps in figure-of-eight pattern about waist and upper thigh, or neoprene thigh sleeves.



A. Buddy taping. **B.** Thumb figure-of-eight. **C.** Thumb checkrein. **D.** Wrist taping. **E.** Elbow hyperextension taping. **F.** Medial elbow taping. **G.** Medial knee taping.

Figure 55-1 Taping Procedures: Various.

Anterior Cruciate Ligament Taping

Performance of custom-fitted and off-the-shelf braces far exceeds that of taping. Taping requires skilled practitioner.

Medial or Lateral Knee Taping

Common indication: Collateral ligament sprain.
Materials: 1½-inch or 2-inch cloth tape, or 3-inch elastic tape.
Athlete position: Standing with knee flexed about 15 degrees (achieve this position by placing 1-inch to 1½-inch block under athlete's heel).
Technique for medial knee injuries: Place anchor strips about midthigh and midcalf. Criss-cross strips in "X" pattern, creating fan across medial aspect of knee (see Fig. 55-1). Binding or crimping tape increases tensile strength. Repeat anchors to close, taking care not to restrict circulation. Cut area posteriorly over calf and close gap.
Technique for lateral knee injuries: Modified to provide lateral support.

Patellofemoral Taping

Common indications: Patellofemoral pain syndromes, patellar tendonitis.
Athlete position: Knee slightly flexed; may vary depending on athlete symptoms.
Technique:

- Shave hair from anterior aspect of knee; clean with Skin-Prep.
- Apply three strips of Cover-Roll stretch tape.
  - Starting laterally, place first strip over proximal half of patella, pushing knee cap distally, pulling medially and puckering the skin (Fig. 55-2).
  - Starting more laterally, place second strip over middle of patella, pulling patella medially and tilting medial edge downward, again puckering the skin (see Fig. 55-2).
  - Apply third strip over distal half of patella (see Fig. 55-2).
- Apply three strips of Sports Tape.
  - Apply first and second strips in the same manner as Cover-Roll strips (see Fig. 55-2).
  - Starting laterally, place third strip over inferior pole of patella, rotating it medially and superiorly (see Fig. 55-2).
  - Apply optional anchor strip over all three previous strips, again pulling medially (see Fig. 55-2).

Arch Figure-of-Eight

Common indications: Arch sprains, conditions resulting from excessive pronation.
Materials: 1-inch or 1½-inch tape
Athlete position: Same as for ankle taping.
Technique:

- Eliminate underwrap to allow for foot perspiration.
- Apply anchor strip loosely around metatarsal heads (Fig. 55-3); allow for expansion of foot on weight bearing.
- Apply half figure-of-eight, starting at base of great toe, angling across longitudinal arch, around heel, and returning to base of great toe (see Fig. 55-3).
- Apply other half figure-of-eight, starting at base of little toe, angling across longitudinal arch, around heel, and returning to base of little toe (see Fig. 55-3).
- Repeat previous two steps once or twice more (see Fig. 55-3).
- Apply horizontal strips, pulling medially, from heel to ball of foot (see Fig. 55-3).
- Apply "low dye" strip.
- Close by applying half anchor dorsum of foot over original anchor (see Fig. 55-3).

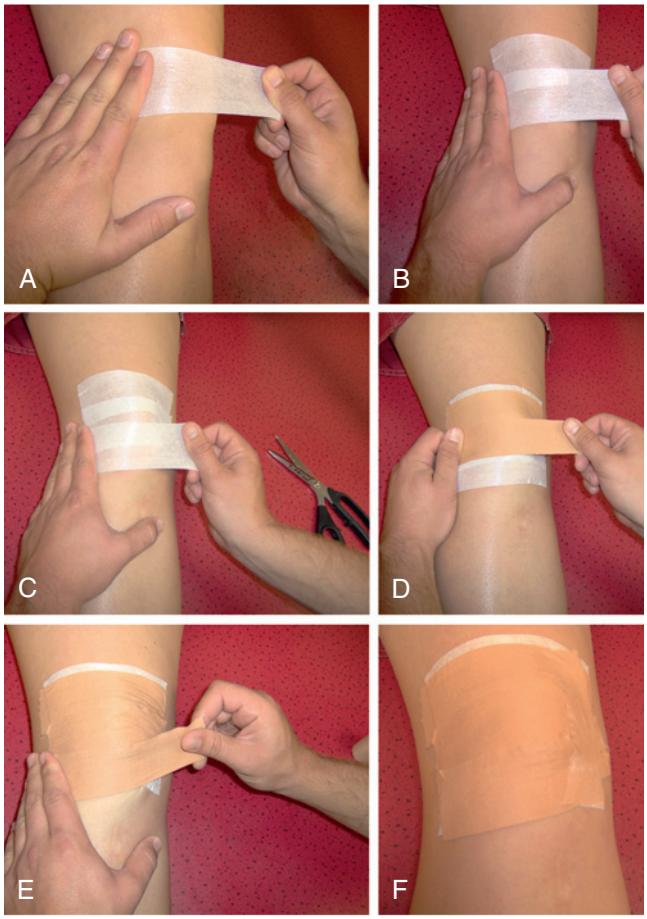


Figure 55-2 Taping Procedures: Patellofemoral Taping.

Achilles Tendon Taping

Common indications: Strain of gastrocsoleus/Achilles tendon, Achilles tendonitis.
Materials: 3-inch elastic tape, 1½-inch or 2-inch cloth tape (optional).
Athlete position: Prone with ankle plantarflexion.
Technique:

- Apply anchors around arch of foot and calf (Fig. 55-4).
- Apply support strips, repeat as needed; may use any of following alternatives:
  - Start from foot anchor, pull proximally to end at calf anchor (see Fig. 55-4).
  - Start from foot anchor, pull proximally, split tape in half to achieve better attachment to anchors (see Fig. 55-4).
- Close applying circular strips about foot and calf (see Fig. 55-4).

Turf-Toe Taping

Common indications: Sprains of first metatarsophalangeal joint and sesamoiditis.
Materials: 1-inch tape, felt/foam (optional).
Athlete position: Same as for ankle taping, positioning great toe in direction opposite of injury.
Technique:

- Apply anchor strips about midarch of foot and about proximal phalanx of great toe (Fig. 55-5).
- Apply support strips from distal anchor to proximal; dorsal strips restrict flexion and ventral strips restrict extension (see Fig. 55-5).
- Repeat anchors to close (see Fig. 55-5).

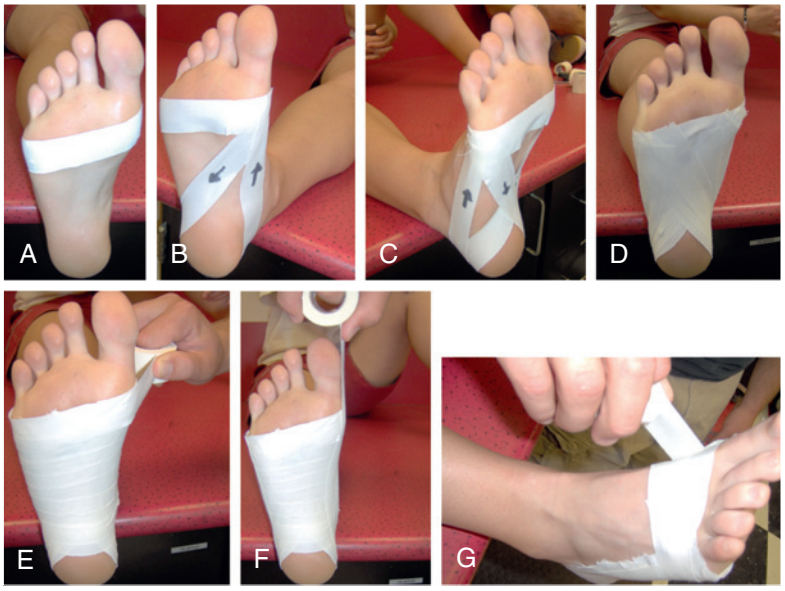


Figure 55-3 Taping Procedures: Arch Taping.

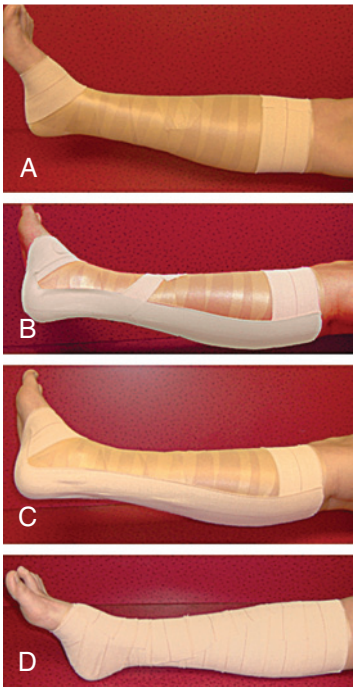


Figure 55-4 Taping Procedures: Achilles Tendon Taping.

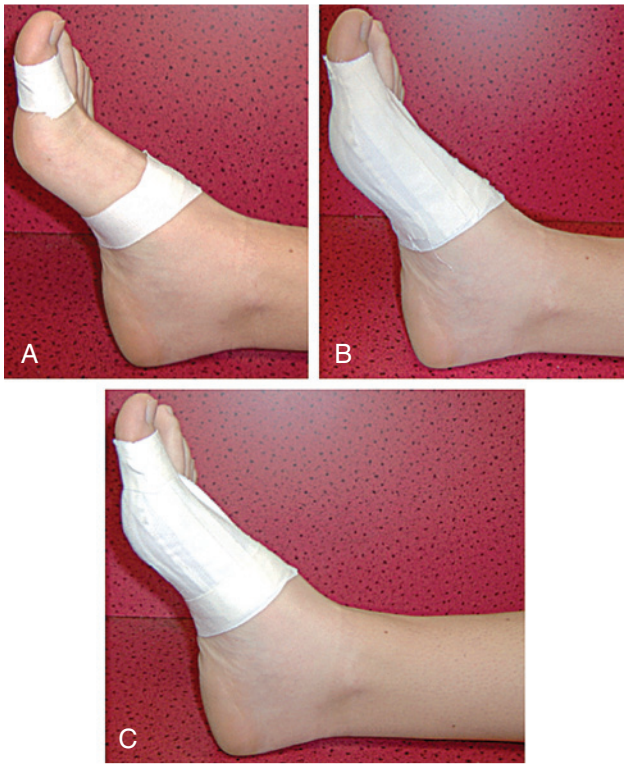


Figure 55-5 Taping Procedures: Turf-Toe Taping.

BRACING

Comparisons of Bracing to Taping

- **Advantages of bracing**
  - Does not require skilled application.
  - Sometimes more cost-effective (tape + underwrap + adherent + time = $).
  - Increased convenience.
  - Less offensive to skin.
- **Disadvantages of bracing**
  - Migration of braces during active use may lead to failure to provide support and decreased performance. Migration may be reduced by use of tape adherent or specially designed straps or undergarments.
  - Athletes commonly complain about weight of some braces; the weight is required to provide adequate protection.
  - Brace parts wear out or may break, requiring untimely replacement.
  - Athlete may be between sizes of over-the-counter braces.
  - Custom-made braces are more expensive and require time to be fabricated.
  - Brace may limit range of motion (ROM).
  - Patient compliance less certain (if tape is applied, it will be worn).

Considerations of Brace Prescription

- **Brace market full of unsubstantiated claims and disclaimers of liability.**
- **Need to be knowledgeable about and critical of new devices.**
- **Often need to evaluate a currently used or "borrowed" device.**
- **Brace-related problems** (e.g., skin irritation, altered function) **require attention.**
- **Discuss options with treatment team.**

SELECTED BRACES

Tennis Elbow "Counter-Force" Strap

Common indications: Lateral epicondylitis, medial epicondylitis (less often).
Description: Over-the-counter Velcro-and-elastic strap placed on proximal forearm, designed to reduce contractile force of wrist and finger extensors (Fig. 55-6).
Application and usage: Worn during activities (sport or nonsport) that aggravate condition.

Silicone Rubber-Moldable Plastic/Poly Blend Wrist/Hand Cast

Common indications: Hand and wrist injuries requiring immobilization to participate in contact or collision sports (e.g., moderate carpal ligament sprains, healing nondisplaced or internally fixated fractures). Physician decides if injury can be adequately supported by silicone casting.
Description: Custom-made. Scotchcast (fiberglass cast tape) currently accepted by all high school and college/university athletic associations.
Application and usage: Splint is univalved and secured by tape; bivalved cast maintains immobilization when athlete not actively participating in sport.

Lateral Prophylactic Knee Braces (Hinged)

Common indications: Decrease incidence and severity of valgus force injuries to the knee; most commonly used in football.
Description: Over-the-counter hinged single lateral upright or hinged double upright device strapped to knee.
Application and usage: Brace migration prevention: shave skin, as for taping; use tape adherent and condylar suspension. Maximal effectiveness requires team approach:

A. Examples of functional knee braces. **B.** Examples of neoprene knee sleeves. **C.** Neoprene lateral patella stabilizing brace. **D.** Patellar tendon strap.
E. Lace-up ankle support. **F.** Ankle air cast. **G.** Active ankle hinged stirrup splint. **H.** Steel shoe inserts.
I. Tuli's heel cups. **J.** Metatarsal arch pads. **K.** Tennis elbow counterforce brace. **L.** Superfeet semirigid arch support.

Figure 55-6 Braces.

- Use of audiovisual aids (e.g., videotapes, instruction, posters) supplied by manufacturers to teach players proper application and daily reminders.
- Daily check of positioning by coaches and certified athletic trainers or other skilled person.
- Weekly check on upkeep by coaches, parents, equipment managers, certified athletic trainers, and players.

Effectiveness: debatable.

Functional Knee Braces

Common indications: Mild/moderate instability, after reconstructive surgery of moderate/severe instability.

Description: Various designs using hinged double uprights with ROM stops, straps, or fitted cuff or shells; some over-the-counter braces, but most are custom-fitted via cast mold or leg tracing (see Fig. 55-6).

Application and usage:

- Migration and fit can be problems (see "Lateral Prophylactic Knee Braces").
- Exposed metal must be covered in contact-sport applications, either with sports cover or socks/pants.
- Some studies have investigated relative effectiveness of these braces; findings are mixed.

Knee Sleeves

Common indications: Conservative management of many knee-pain complaints resulting from acute or chronic inflammation, including strains, cartilage tears, and patellofemoral problems. Sleeves provide **warmth,** which expedites blood flow and increases healing agents, and **compression,** which reduces impact and gait trauma. May be used postoperatively for effusion control.

Description: Over-the-counter elastic or neoprene pull-over sleeve (see Fig. 55-6).

Application and usage:

- Easily acquired and accepted.
- Neoprene support increases sense of warmth, comfort.
- Patellofemoral problems usually require use of sleeve with patellar cut-out and buttress.
- Report of increased stability, probably due to increased proprioceptive input.
- **Similar devices available for other areas of the body—forearm, wrist, calf.**

Lateral Patella Stabilizing Brace

Common indications: Conservative or postoperative management of patellofemoral joint problems.

Description: Over-the-counter neoprene knee sleeve with patellar cut-out and a J-shaped lateral buttress strap to dynamically decrease excessive lateral patellar tracking with straps that pull medially (see Fig. 55-6).

Application and usage:

- Usually place counterbalancing strap superiorly.
- Cases of infrapatellar tendonitis may be aided by inferior placement of counterbalancing strap.
- Cases of medial patellar subluxation (rare) may be aided by using left brace on right knee and vice versa.

Patellar Tendon Straps

Common indications: Patellofemoral problems, patellar tendonitis.

Description: Most use thin circumferential strap between distal joint line and tibial tuberosity to apply counterforce to patellar tendon and reduce "play" caused by laxity/lack of muscle control (see Fig. 55-6).

Application and usage: Easily acquired and accepted; good patient compliance; fits easily under most clothes/pads.

Osteoarthritis Knee Braces

Common indications: Unicompartmental osteoarthritis.

Description: Custom-fit or off-the-shelf hinged braces that unload affected joint compartment and accommodate for varus and valgus angulations.

Application and usage: Expensive; may temporarily relieve arthritic pain and improve function in active patients.

Ankle Braces

Common indications: Healed/healing ankle sprains or fractures, tendonitis about ankle.

Description, application, and usage:

- **Slip-on elastic support** provides even compression to decrease edema; not a prophylactic support.
- **Lace-up ankle support** uses medial and lateral stays to restrict inversion/eversion; best alternative to ankle taping; may be used as prophylactic or functional support (see Fig. 55-6).
- **Stirrup splints,** based on semirigid orthosis, use Velcro straps to hold in place; designed for rehabilitative and functional uses. All are universally sized.
  - **Inflatable ankle air cast** uses adjustable air pressure linings to improve individual fit, decrease edema, and prevent excessive inversion/eversion (see Fig. 55-6).
  - **Gel cast** uses gel-filled linings to improve individual fit; can be placed in freezer to be used as one form of cryotherapy; holds in body heat for increased comfort and functional use.
  - Other devices use foam rubber linings or other materials; may be over-the-counter or custom-made.
  - **Hinged stirrup splint** has greater acceptance as prophylactic brace; provides increased plantarflexion and dorsiflexion (see Fig. 55-6).

Orthotics

Common indications: Lower extremity kinetic chain conditions resulting from excessive pronation, cavus foot, or other foot dysfunctions or mechanical faults.

Description, application, and usage:

- **Soft orthotics** provide cheaper over-the-counter solution; easy break-in, limited lifespan.
- **Hard orthotics** provide customized solution for road runners not involved in agility activities. Good control, minimal cushioning.
- **Semirigid orthotics** provide support of hard orthotics for athletes involved in agility sports (see Fig. 55-6). Good combination of support and cushioning.
- **Sorbothane viscoelastic insoles** reduce impact loading forces, provide minimal support.
- **Steel or carbon (Dynaflex) shoe inserts** provide support to metatarsal fractures, midfoot sprains, and "turf toe" and prevent hyperextension of great toe (see Fig. 55-6).
- **Heel cups** (e.g., Tuli's) decrease impact and improve shock absorption capabilities of calcaneal fat pad (see Fig. 55-6).
- **Longitudinal arch pads** or Barton's wedges provide symptomatic relief of painful foot conditions; may be held in place with tape or glue or may be self-adhesive to shoe or orthotic.
- **Metatarsal arch pads** provide symptomatic relief of painful foot conditions such as interdigital neuroma and metatarsalgia; may be held in place with tape or glue or may be self-adhesive to shoe or orthotic (see Fig. 55-6). Used to splay and elongate metatarsals and tarsals.

RECOMMENDED READINGS

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6. Deppen RJ, Landfried M: Efficacy of prophylactic knee bracing in high school football players. J Sports Phys Ther 20(5):243, 1994.
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Injections in the Athlete

56

Brett W. Gibson, Michelle Wolcott, and Armando F. Vidal

GENERAL OVERVIEW

- Knowledge of anatomy is essential to performing injections safely and effectively.
- Use of local anesthetic injections in athletes may reduce the number of games missed because of injury but carries a theoretical risk of worsening the injury.
- Corticosteroid injections are widely used in the treatment of athletic injuries because of their potent anti-inflammatory properties, but they are not without undesirable side effects.
  - Corticosteroid injections may not offer clear therapeutic advantage over anti-inflammatory medications alone.
  - Both the physician and the athlete should be aware of any restrictions regarding the use of corticosteroids in competition.
    - The World Anti-Doping Agency requires completion of a therapeutic use exemption prior to administration of corticosteroids.
    - The International Olympic Committee Medical Code requires that any team doctor wishing to administer corticosteroids to an athlete by local or intra-articular injection must give written notification to the relevant medical authority prior to the competition.
- After careful consideration, the physician and athlete must determine that the benefits outweigh the risks before proceeding with an injection.

STERILE TECHNIQUE FOR INJECTIONS

- Mark the injection site by pressing the tip of a capped needle against the skin.
- Prep the injection site with Betadine or other appropriate antiseptic solution.
- Use prepacked sterile needles and syringes.
- Use single-dose vials of injectable agent whenever possible.
- Change needles after drawing up the solution.
- Wear sterile gloves.

COMFORT MEASURES

- **Ethyl chloride** is a topical spray that rapidly cools the skin, providing topical anesthesia.
- Benefit of **subcutaneous local anesthetic** may be limited by subcutaneous injection pain.
- **EMLA cream** must be applied at least 1 hour prior to injection but has been shown to be highly effective.

ACCURACY OF INJECTION

- Knowledge of surface landmarks and tactile feedback are essential to accurate needle placement.
- Clinician experience alone does not necessarily improve the accuracy of an injection.
- Joint aspiration in the presence of an effusion may assist in intra-articular needle placement but is not a guarantee.
- Injecting 1 to 2 mL of air with the injection may help verify intra-articular injection of the knee or shoulder. The presence of an audible "squishing" with range of motion indicates a successful intra-articular injection.
- Ultrasound guidance has been shown to improve the accuracy and clinical effect of some injections in the hands of a skilled ultrasonographer.
- Ultrasound-guided injections of the plantar fascia have not shown an advantage over "palpation-guided" injections.
- Image guidance has not shown a definitive advantage over traditional techniques for most injections, although it may be useful in certain situations, such as in obese patients or in patients who have failed to respond to previous injections.

COMMON AGENTS

Local Anesthetics

- **Lidocaine (Xylocaine)** is the most widely used anesthetic. Rapid onset of action with limited duration, approximately 30 minutes. Maximum dose: 300 mg (30 mL of 1% lidocaine).
- **Bupivacaine (Marcaine)** has a slow onset of action with prolonged duration of effect, up to 8 hours. Maximum dose: 175 mg (70 mL of 0.25% bupivacaine).
- A combination of lidocaine and bupivacaine allows the rapid onset of action with a prolonged duration of effect.

Corticosteroids

- Less soluble
  - Methylprednisolone acetate (Depo-Medrol)
  - Triamcinolone acetonide (Kenalog)
  - Triamcinolone hexacetonide (Aristospan)
- More soluble
  - Betamethasone sodium phosphate (Celestone)
  - Dexamethasone sodium phosphate (Decadron)
  - Prednisolone sodium phosphate (Hydeltrasol)
- Dosage
  - Dosage requirements vary and should be individualized on the basis of the condition being treated and the response of the patient (Table 56-1).
  - In chronic cases, injections may be repeated at intervals ranging from 1 week to 5 or more weeks depending on the degree of relief obtained from the initial injection.
  - No clear safety guidelines exist regarding the frequency and maximum number of injections. Judicious use of injections is recommended.

MECHANISM OF ACTION

Local Anesthetics

- Membrane-stabilizing agents cause a reversible conduction block along nerve fibers.
- Smaller nerve fibers are more sensitive, allowing inhibition of pain signals while sparing pressure and proprioceptive fibers.
- Maximum plasma concentrations of local anesthetic are achieved within 10 to 25 minutes.
- Avoid preparations containing epinephrine for intra-articular or soft tissue injections. Epinephrine causes vasoconstriction, prolonging the anesthetic effect when used in the skin.

Table 56-1 SUGGESTED DOSES FOR CORTICOSTEROID PREPARATIONS

Corticosteroid agent	Large joint	Medium joint	Small joint	Soft tissue
Methylprednisolone acetate (Depo-Medrol)	20 to 80 mg	10 to 40 mg	4 to 10 mg	4 to 30 mg
Betamethasone sodium phosphate (Celestone)	1.0 mg	0.5 to 1.0 mg	0.25 to 0.5 mg	0.5 to 1.0 mg

Corticosteroids

- Interfere with inflammatory cell-to-cell adhesion and migration through the vascular endothelium.
- Inhibit the synthesis of cyclooxygenase-2 (COX-2) and various proinflammatory cytokines.
- Stimulate gluconeogenesis and catabolic activity in muscle, skin, lymphoid, adipose, and connective tissue.
- Solubility determines duration of action and extent of systemic effects.
  - **Insoluble:** Average duration of action is longer for less soluble agents. Display mainly local effects.
  - **Soluble:** Diffuse more readily from the injected region and may exert greater systemic effects. May be more useful in extra-articular or soft tissue injections.
- Average duration of action is 1 to 3 weeks and is longer for less soluble agents.

SIDE EFFECTS

Local Anesthetics

Anaphylaxis: Occurs from acute mast cell degranulation and is characterized by laryngeal edema, bronchospasm, and hypotension. The cause of local toxicity is allergic reaction to para-aminobenzoic acid (PABA). PABA is a metabolic product of the degradation of the ester class of local anesthetics, such as procaine (Novocain), and to a lesser degree, amide class anesthetics such as lidocaine. It is also a metabolic by-product of Methylparaben, a preservative in multidose vials of lidocaine.

Toxicity: Usually the result of inadvertent intravenous injection. Primary target organ is central nervous system resulting in altered speech, muscle twitching, and seizures. Cardiovascular toxicity (e.g., ventricular arrhythmias) may also occur. Aspirate prior to injection to avoid inadvertent intravenous injection. Compared to other local anesthetics, bupivacaine is markedly cardiotoxic.

Chondrotoxicity: Emerging evidence that both lidocaine and bupivicaine may have negative effects on articular cartilage health and chondrocyte viability.

Corticosteroids

Postinjection flare: Most common side effect. Related to steroid-crystal synovitis. Self-limited. Typically lasts less than 12 hours. Analgesic therapy and ice packs offer effective relief.

Facial flushing: Subjective sensation of warmth in the face and upper trunk.

Poor diabetic control: May increase hepatic glucose production and antagonize insulin effects. Close monitoring is suggested following injection of corticosteroid.

Subcutaneous fat atrophy and skin depigmentation: May occur after a single injection. Avoid repeated subcutaneous or superficial injections. Subcutaneous fat atrophy is especially problematic in the plantar surface of the foot. Skin depigmentation occurs more commonly in dark-skinned individuals.

Tendon rupture: Dose-dependent decrease in tenocyte proliferation and reduced collagen production by tenocytes. Care should be taken to avoid direct intratendinous injection, which can result in this complication.

Steroid-induced arthropathy and cartilage damage: Destruction of articular cartilage and a decrease in cartilage matrix production has been shown in animal studies. Although not reported in humans, a theoretical risk remains. Additionally, iatrogenic chondral trauma can result from direct contact of the needle with the cartilage surfaces. Avoid moving the needle once inserted to prevent chondral injury.

Infection: Incidence of joint sepsis varies from 1 in 3,000 to 1 in 50,000 for corticosteroid injections. Informed consent should be obtained prior to any invasive procedure. Use of good sterile technique minimizes this risk.

Anaphylaxis: May occur even after previous uneventful injections.

Hypothalamic-pituitary-adrenal axis suppression: Suppression is mild and transient. Avoid injecting multiple large joints simultaneously.

Other

Syncope: Patients who feel lightheaded or overly apprehensive should lie down. Protect the airway in the event of loss of consciousness. Reassure the patient.

CONTRAINDICATIONS

Infection: Overlying cellulitis, bacteremia, or septic arthritis.

Fracture or unstable joint: Risk of worsening injury.

Tendinopathy: Risk of tendon rupture if injected directly into tendon (e.g., Achilles tendon).

History of medication allergy or anaphylaxis following injection: Previous uneventful injection does not eliminate possibility of future reactions.

Coagulopathy or anticoagulation therapy: Risk of bleeding at the injection site.

Poorly controlled diabetes: Corticosteroids may result in temporary exacerbation of diabetes.

Prosthetic joint: Risk of infection.

UPPER EXTREMITY INJECTIONS

Shoulder

Subacromial Space

Indications: Subacromial bursitis, rotator cuff tendinitis.

Technique:

- Allow the arm to hang at the patient's side to open up the subacromial space.
- Palpate the lateral border of acromion.
- The subacromial bursa is deep to the acromion and superficial to the rotator cuff (Fig. 56-1).
- Insert the needle angled slightly superiorly at midpoint of acromion and approximately 2 to 3 cm lateral to the lateral border of the acromion
- Slowly withdraw the needle while injecting.

Considerations: Insertion of the needle too far medially may result in injection into the supraspinatus muscle belly. Up to 30% of subacromial injections may miss the subacromial space. Ultrasound guidance has been shown to improve the accuracy of subacromial injections.

Glenohumeral Joint

Indications: Labral tears, chondral defects, bone contusions, diagnostic injections.

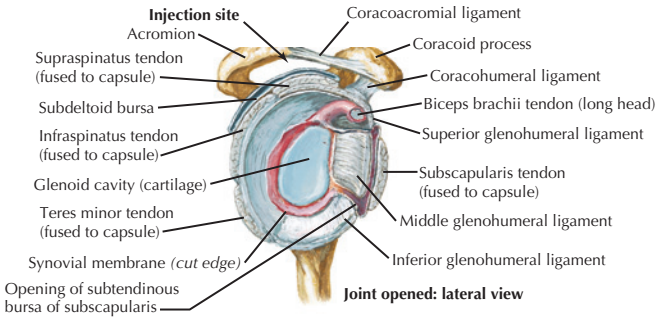
Technique (anterior):

- Approach the glenohumeral joint through the rotator interval between the superior border of the subscapularis tendon and the anterior border of the supraspinatus tendon (see Fig. 56-1).
- Insert the needle at the midpoint between the anterolateral border of the acromion and coracoid, directing the needle slightly medially. Typically a soft spot can be palpated in this region.

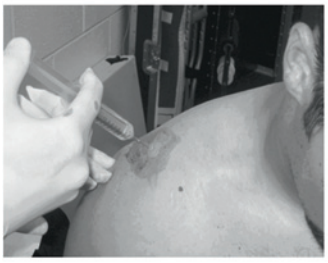
Technique (posterior):

- Palpate the posterolateral corner of acromion.
- Palpate the soft spot of the glenohumeral joint approximately 1 to 2 cm medial and 2 to 3 cm inferior to the posterolateral corner of acromion.
- Palpate the coracoid anteriorly with the opposite hand.
- Insert the needle into the soft spot, directing the needle toward the coracoid.

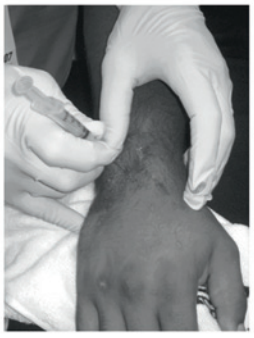
Considerations: Use caution to avoid direct injury to the articular cartilage of the humeral head and glenoid. Anterior injections may offer a higher rate of accuracy than posterior injections.



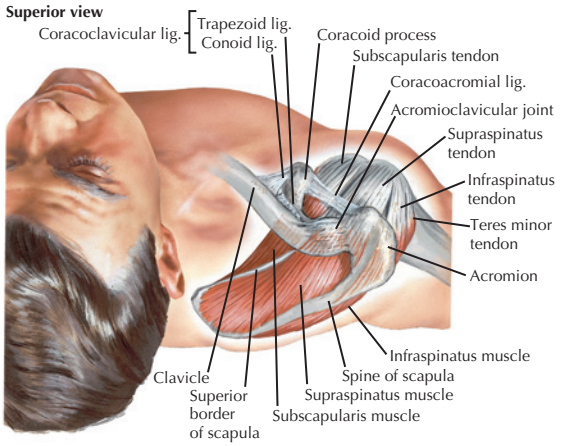
A. The subacromial bursa lies deep to the acromion and superficial to the rotator cuff. The glenohumeral joint is approached anteriorly through the interval between the supscapularis and supraspinatus tendons.



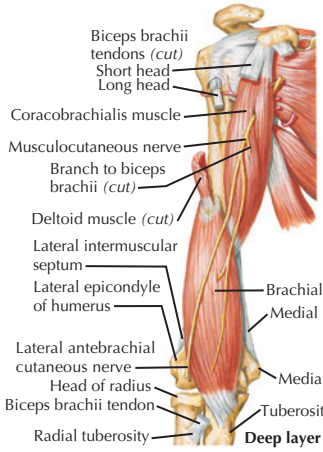
C. During an AC joint injection, the needle is directed at a 30° angle from the vertical to follow the normal anatomic orientation of the AC joint.



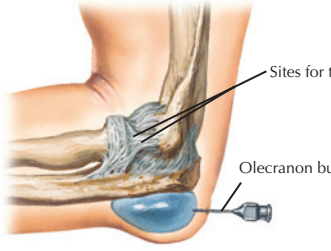
D. The needle is inserted into the soft spot distal to Lister's tubercle and angled slightly proximally to account for the normal volar angulation of the wrist joint.

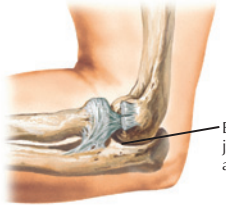


B. The acromioclavicular joint is identified by palpating medially from the anterolateral border of the acromion.

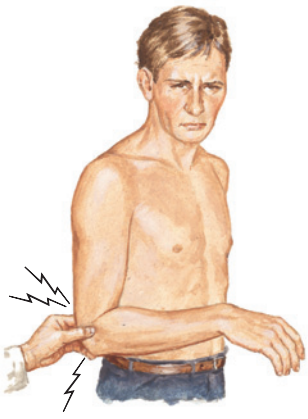


E. The long head of the biceps tendon lies within a sheath in the bicipital, or intertubercular, groove.



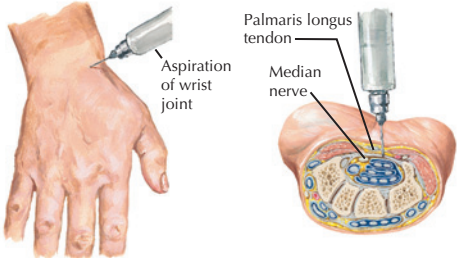


F. Approach the elbow joint through the soft spot between the olecranon, lateral epicondyle, and radial head. The area of maximum fluctuance is identifed for needle placement into the olecranon bursa.

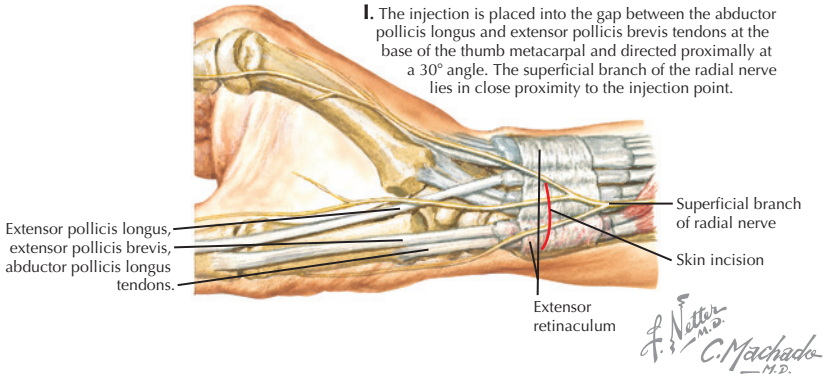


Epicondylitis (tennis elbow)
Exquisite tenderness approximately 1 cm distal to the lateral epicondyle

G. The point of maximum tenderness in lateral epicondylitis typically lies 1 cm distal to the lateral epicondyle.



H. The needle is inserted ulnar to the palmaris longus tendon to avoid direct injury to the median nerve.



I. The injection is placed into the gap between the abductor pollicis longus and extensor pollicis brevis tendons at the base of the thumb metacarpal and directed proximally at a 30° angle. The superficial branch of the radial nerve lies in close proximity to the injection point.

Figure 56-1 Upper Extremity Injections.

Acromioclavicular (AC) Joint

Indications: AC joint sprain, grades I and II AC joint separation, AC joint arthropathy, distal clavicle osteolysis.

Technique:

- Identify the lateral border of the acromion anteriorly and palpate medially to identify the superior aspect of the AC joint (see Fig. 56-1).
- The acromioclavicular articulation runs obliquely medially from superior to inferior.
- Direct the needle medially at a 30-degree angle from the vertical (i.e., parallel to the joint line) (see Fig. 56-1).

Considerations: Use a smaller total volume for the AC joint, typically 1 to 2 mL.

Long Head of the Biceps

Indications: Biceps tendinopathy.

Technique:

- The biceps tendon lies within a sheath in the bicipital groove (see Fig. 56-1).
- Palpate the point of tenderness at the anterior aspect of the shoulder.
- Insert the needle at the most superior point of tenderness, and direct the needle inferiorly, parallel with the tendon.

Considerations: Withdraw the needle slightly if resistance is encountered to prevent intratendinous injection. Ultrasound guidance may be useful to confirm needle placement in the tendon sheath.

Elbow

Elbow Joint

Indications: Chondral defects, bone contusions, posteromedial osteophytes, aspiration of effusion, synovitis.

Technique:

- Position patient in slight elbow flexion with hand resting in lap.
- Palpate the soft spot between the olecranon, lateral epicondyle, and radial head (see Fig. 56-1).
- Insert the needle into the center of the triangle formed by the above landmarks.
- Direct the needle toward the medial epicondyle.

Considerations: The joint is relatively superficial—repeated injections may result in subcutaneous fat atrophy or skin depigmentation. Avoid overpenetration of the needle to prevent direct injury to the articular cartilage of the elbow.

Olecranon Bursa

Indications: Olecranon bursitis.

Technique:

- Position elbow in slight flexion with hand resting in lap.
- Insert the needle into area of maximum fluctuance (see Fig. 56-1).

Considerations: Attempted aspiration prior to injection may be helpful in determining whether an infection is present. Avoid injection in the setting of infection.

Common Flexor and Extensor Origin

Indications: Medial epicondylitis, lateral epicondylitis.

Technique:

- Position elbow in slight flexion with hand resting in lap.
- Supinate the forearm for common flexor origin injection. Pronate the forearm for common extensor origin injection.
- Insert the needle at the point of maximum tenderness, typically 1 cm distal to epicondyle (see Fig. 56-1).

Considerations: Radial tunnel syndrome is a rare compression neuropathy that may be confused with lateral epicondylitis.

Wrist

Wrist Joint

Indications: Triangular fibrocartilage complex (TFCC) tear, scapholunate ligament sprain, bone contusion.

Technique:

- Position the wrist in slight flexion.
- Palpate Lister's tubercle on the dorsal surface of the distal radius.
- Identify the soft spot approximately 1 cm distal to Lister's tubercle.
- Direct the needle slightly proximally to account for the 11 degrees of volar angulation at the distal radius (see Fig. 56-1).

Considerations: Scaphoid fracture must be ruled out definitively prior to proceeding with wrist joint injection.

Carpal Tunnel

Indications: Carpal tunnel syndrome.

Technique:

- Position wrist in 30 degrees of dorsiflexion.
- The proximal border of the carpal tunnel lies at the distal wrist crease. The median nerve is ulnar to the flexor carpi radialis and deep to the palmaris longus (see Fig. 56-1).
- Palpate the palmaris longus tendon at the proximal wrist crease. Use the wrist midline if the palmaris longus tendon is absent.
- Direct the needle distally toward the middle/ring finger at a 30- to 45-degree angle.
- Insert the needle 1 to 2 cm until no resistance.

Considerations: Paresthesias indicate that the needle is in the median nerve—withdraw the needle and redirect more ulnarly. Direct injection into the median nerve can cause irreversible damage. Avoid injecting large volumes into the carpal tunnel because this may exacerbate compression of the median nerve.

First Dorsal Compartment

Indications: De Quervain's tenosynovitis.

Technique:

- Position the thumb in abduction and extension to facilitate identification of the radial wrist tendons.
- Palpate the abductor pollicis longus and extensor pollicis brevis tendons in the first dorsal compartment (see Fig. 56-1).
- Insert the needle in the gap between the two tendons at the base of the thumb metacarpal.
- Direct the needle at a 30-degree angle proximally toward the radial styloid.

Considerations: Redirect the needle slightly if resistance is encountered to prevent intratendinous injection. Paresthesias may indicate that the needle is in the superficial branch of the radial nerve.

Flexor Tendon Sheath

Indications: Trigger finger.

Technique:

- Position patient with forearm supinated and fingers extended.
- Palpate the nodule in the flexor tendon.
- Insert the needle at the distal palmar crease.
- Direct the needle distally at a 30-degree angle into the flexor tendon sheath.

Considerations: Withdraw needle slightly if resistance is encountered to avoid intratendinous injection. Digital nerve anesthesia occurs with injection of local anesthetic.

LOWER EXTREMITY INJECTIONS

Hip

Trochanteric Bursa

Indications: Trochanteric bursitis, bone contusion.

Technique:

- Place patient in lateral decubitus position with hip and knee flexed.
- Insert the needle perpendicular to the skin at the point of maximal tenderness over the greater trochanter.
- Inject deep to the iliotibial band into the trochanteric bursa (Fig. 56-2).

Considerations: If the needle is advanced to bone, withdraw slightly and inject. In larger patients consider using a spinal needle.

Knee

Knee Joint

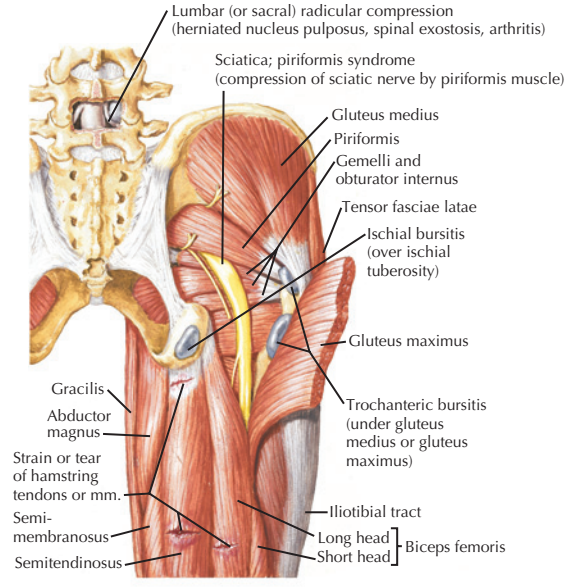
Indications: Bone contusion, meniscus tear, chondromalacia patellae, plica syndrome.

Technique (lateral suprapatellar):

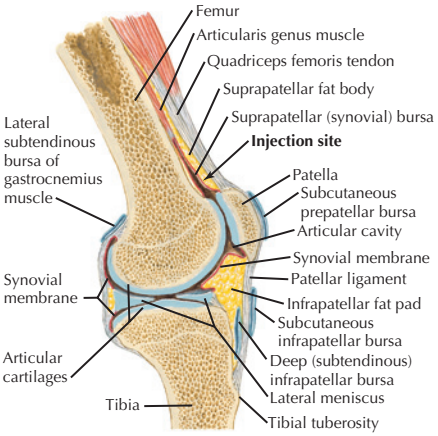
- Position patient with the knee in full extension.
- Palpate the superior border of the patella.
- Push the patella laterally to tent the capsule.
- Insert the needle inferior to the vastus lateralis tendon and parallel to the superior border of the patella (see Fig. 56-2).

Technique (anterolateral):

- Position patient with knee flexed to 90 degrees and leg hanging off the exam table to distract the joint.
- Palpate the lateral border of patellar tendon.

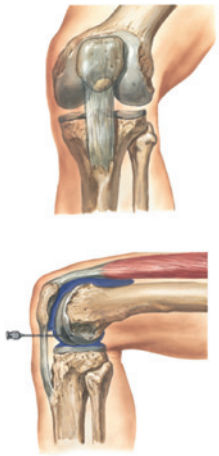


A. The trochanteric bursa lies deep to the iliotibial band over the greater trochanter. The injection is administered at the point of maximum tenderness.

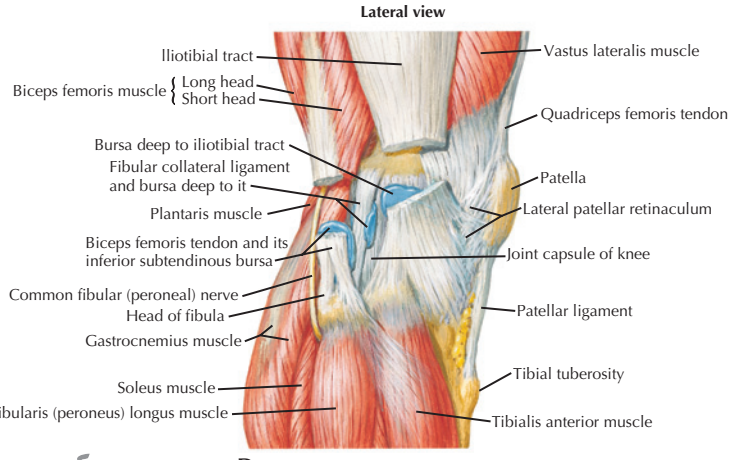


Sagittal section (lateral to midline of knee)

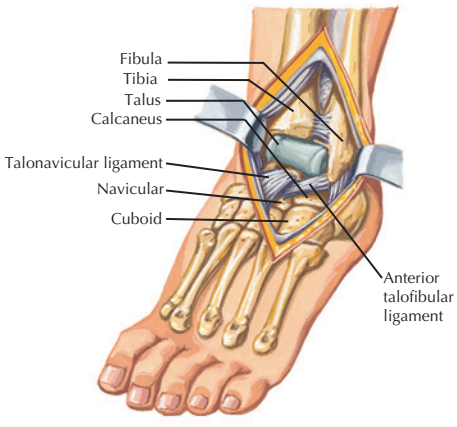
B. Knee injection using the lateral suprapatellar approach enters the joint parallel to the superior pole of the patella, just lateral to the vastus lateralis tendon. The prepatellar bursa is a subcutaneous bursa superficial to the patella.



C. Knee injection using the anterolateral approach enters the joint in the soft spot at the midpoint between the inferolateral border of the patella and the lateral joint line.



D. Knee bursa. The iliotibial bursa lies deep to the iliotibial band in the region of the lateral epicondyle.



E. The anterolateral approach to the ankle joint utilizes the soft spot in the triangular space bordered by the anterior fibula, distal tibia, and proximal talus.

Figure 56-2 Lower Extremity Injections.

- Palpate the soft spot at the midpoint between the inferolateral border of the patella and the lateral joint line (see Fig. 56-2).
- Direct the needle toward the notch approximately 0.5 to 1 cm lateral to the patellar tendon.

Considerations: The lateral suprapatellar approach may allow for more accurate intra-articular placement of the needle. With an anterolateral approach, use caution to avoid direct injury to the articular cartilage of the lateral condyle.

Prepatellar Bursa

Indications: Prepatellar bursitis.

Technique:

- Position patient with the knee extended.
- Prepatellar bursa is superficial to the patella (see Fig. 56-2).
- Insert the needle into area of maximum fluctuance.

Considerations: Attempted aspiration prior to injection may be helpful in determining whether an infection is present. Avoid injection in the setting of infection.

Pes Anserine Bursa

Indications: Pes anserine bursitis.

Technique:

- Position patient with the knee in 90 degrees of flexion.
- The pes anserine is the insertion of the sartorius, gracilis, and semitendinosus tendons. The bursa is deep to the tendons and superficial to the medial collateral ligament (see Fig. 56-2).
- Insert the needle perpendicular to the skin at point of maximum tenderness deep to the pes anserine tendons.

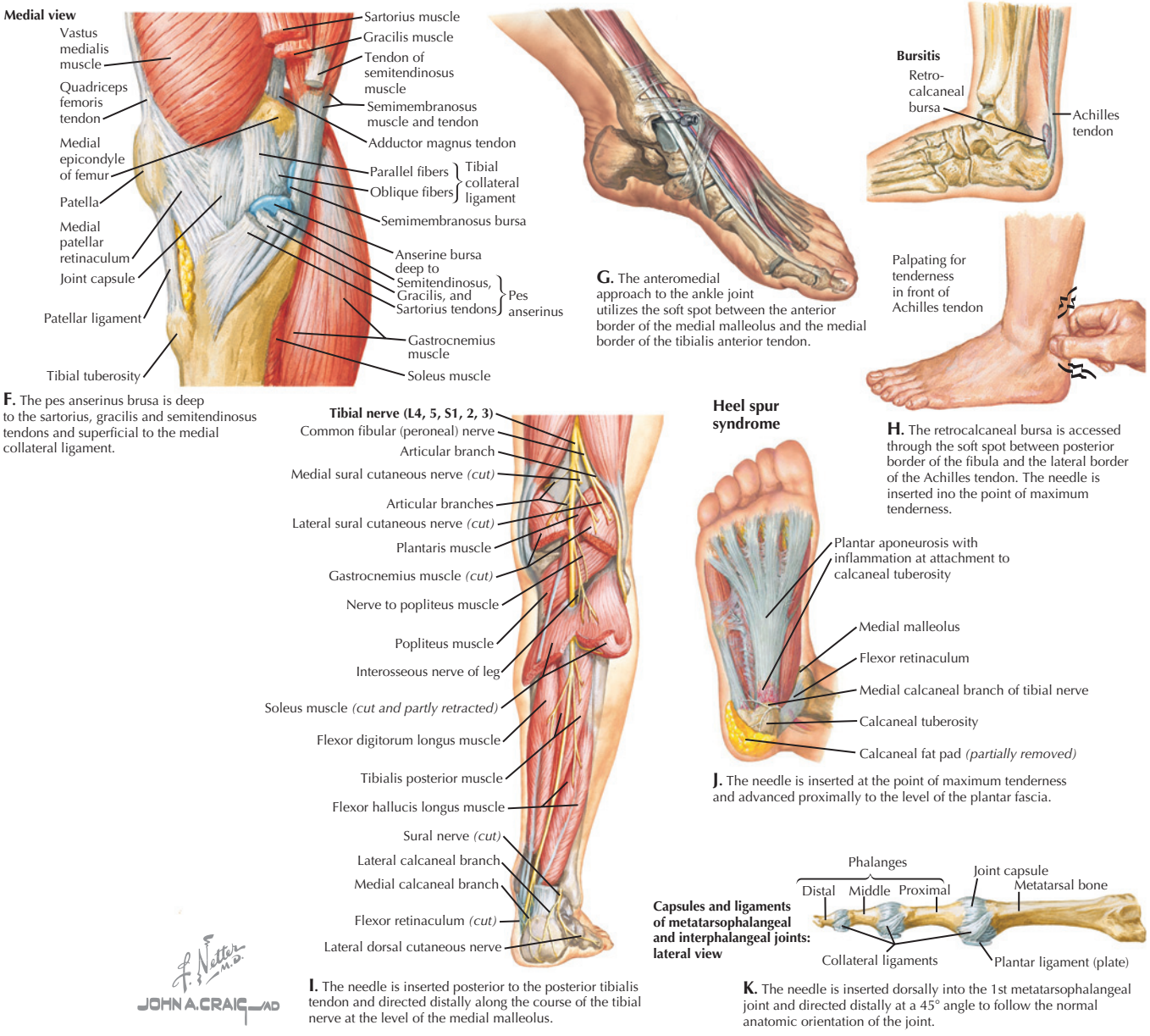
Considerations: Advance needle until no resistance is met to avoid intratendinous injection. If the needle is advanced to bone, withdraw 2 to 3 mm and inject.

Iliotibial Bursa

Indications: Iliotibial band syndrome.

Technique:

- Place patient in lateral decubitus position with hip and knee slightly flexed.



F. The pes anserinus brusa is deep to the sartorius, gracilis and semitendinosus tendons and superficial to the medial collateral ligament.

G. The anteromedial approach to the ankle joint utilizes the soft spot between the anterior border of the medial malleolus and the medial border of the tibialis anterior tendon.

H. The retrocalcaneal bursa is accessed through the soft spot between posterior border of the fibula and the lateral border of the Achilles tendon. The needle is inserted ino the point of maximum tenderness.

I. The needle is inserted posterior to the posterior tibialis tendon and directed distally along the course of the tibial nerve at the level of the medial malleolus.

J. The needle is inserted at the point of maximum tenderness and advanced proximally to the level of the plantar fascia.

K. The needle is inserted dorsally into the 1st metatarsophalangeal joint and directed distally at a 45° angle to follow the normal anatomic orientation of the joint.

Figure 56-2, Cont'd Lower Extremity Injections.

- Palpate the lateral epicondyle of the femur. The bursa lies deep to the iliotibial band in the region of the lateral epicondyle (see Fig. 56-2).
- Insert the needle perpendicular to the skin at the point of maximum tenderness and inject.

Considerations: Inflammation of the iliotibial band may occur more distally near its attachment at Gerdy's tubercle.

Ankle

Ankle Joint

Indications: Osteochondral defect, bone contusion, ankle sprain.
Technique (lateral):

- Position the ankle in slight plantar flexion.
- Identify the soft spot in the triangular space bordered by the anterior fibula, distal tibia, and proximal talus (see Fig. 56-2).
- Direct the needle slightly proximally and medially.

Technique (medial):

- Position the ankle in slight plantar flexion.
- Palpate the soft spot between the anterior border of the medial malleolus and the medial border of the tibialis anterior tendon (see Fig. 56-2).
- Direct the needle slightly proximally and laterally.

Considerations: Use caution to avoid direct injury to the articular cartilage of the tibial plafond or talar dome.

Retrocalcaneal Bursa

Indications: Retrocalcaneal bursitis.
Technique:

- Place the patient in the prone position with the foot in slight dorsiflexion.
- Palpate the soft spot between posterior border of the fibula and the lateral border of the Achilles tendon (see Fig. 56-2).
- Insert the needle at the point of maximum tenderness anterior to the Achilles tendon.

Considerations: Withdraw the needle and redirect if resistance is encountered to prevent intratendinous injection. Avoid overpenetration to prevent inadvertent injection into the medial neurovascular structures. Retrocalcaneal bursitis may be difficult to distinguish from Achilles tendinitis.

Tarsal Tunnel

Indications: Tarsal tunnel syndrome.
Technique:

- Palpate the posterior tibialis tendon.
- Insert the needle posterior to the posterior tibialis tendon at the level of the medial malleolus (see Fig. 56-2).
- Direct the needle distally at a 30-degree angle from the skin surface, parallel to the posterior tibialis tendon.

Considerations: Aspirate prior to injecting to avoid inadvertent intravascular injection. Paresthesias indicate that the needle is in the tibial nerve. Withdraw the needle slightly and redirect if resistance is encountered to prevent intratendinous injection.

Foot

Plantar Fascia

Indications: Plantar fasciitis.
Technique:

- Place the patient in the prone position with the foot in slight dorsiflexion.
- The area of inflammation typically occurs at the medial aspect of the plantar fascia near the medial process of the calcaneal tuberosity, just distal to the heel pad (see Fig. 56-2).
- Insert the needle at the point of maximum tenderness and advance proximally at a 45-degree angle to the level of the plantar fascia.

Considerations: Avoid injection into the heel pad to prevent subcutaneous fat pad atrophy. If the needle is advanced to bone, withdraw slightly and inject.

First Metatarsalphalangeal (MTP) Joint

Indications: Turf toe, chondral defects.
Technique:

- Position the great toe in slight plantar flexion. Passively flex and extend the great toe to aid in identifying the joint space.
- Insert the needle on the dorsal surface of the MTP joint (see Fig. 56-2).
- Direct the needle distally at a 45-degree angle.

Considerations: The joint is relatively superficial—repeated injections may result in subcutaneous fat atrophy or skin depigmentation. Avoid deep penetration with the needle to prevent direct injury to the articular cartilage.

RECOMMENDED READINGS

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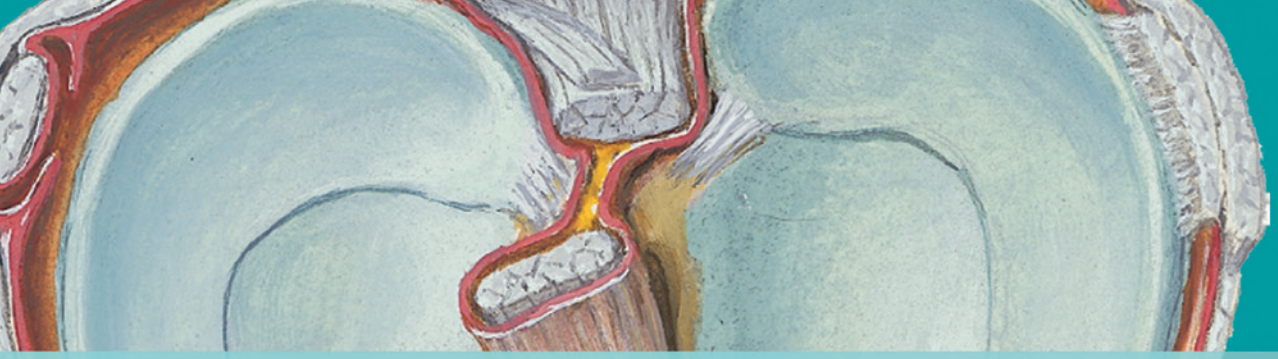

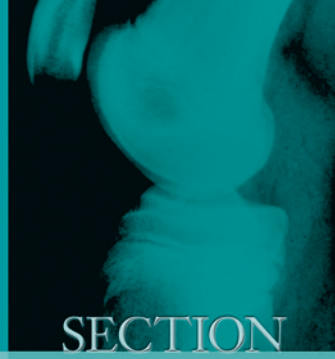

SECTION

VIII

Specific Sports

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Football

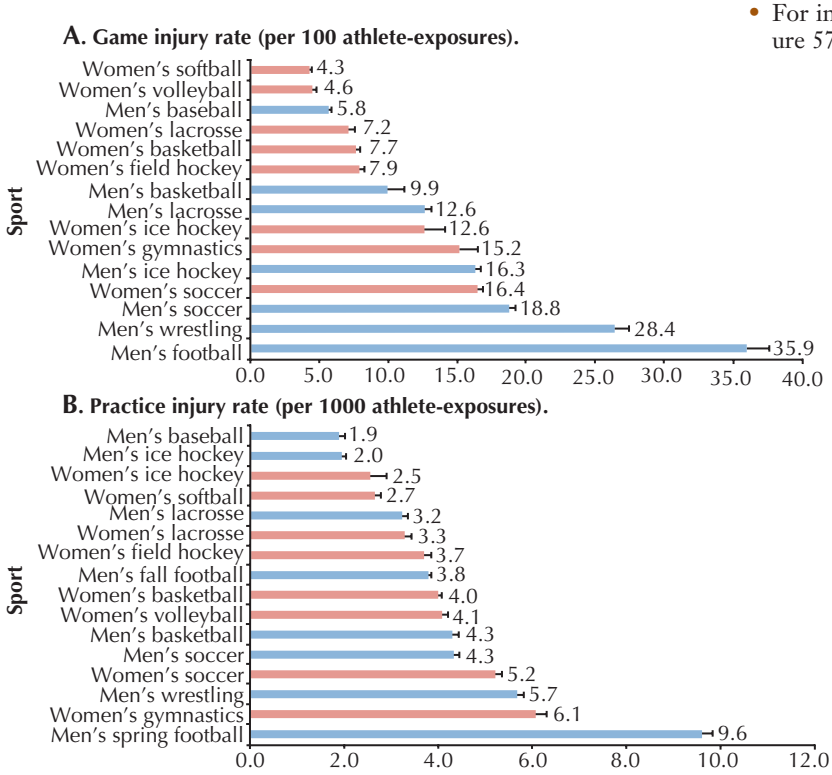
Margot Putukian, Eric C. McCarty, and Wayne Sebastianelli

OVERVIEW

- Approximately 1.5 million athletes participate in American football in the United States.
- It has been played at the college level competitively for more than 100 years. The "flying wedge" was a V-shaped formation used by Harvard in a game against Yale in 1892 and was felt to be associated with significant brutality and injuries, such that in 1894 it was banned. It was the concerns regarding the safety issues of "the flying wedge" that led to the formation of the National Collegiate Athletic Association (NCAA).
- The number of schools sponsoring varsity college football increased from 524 to 621 (18%) between the 1988-1989 and 2003-2004 school years.
- American football involves discontinuous sprint activity and requires strength; sport-specific skills vary depending on position played.
- A variety of schemes and options are used to advance the team's objective: moving the ball down the field. There are four "downs" per possession, each with the goal of moving the ball forward 10 yards, at which point four additional possessions are acquired. Teams score either by touchdown (6 points) or by kicking a field goal (3 points). Following a touchdown, the scoring team has the option of kicking for 1 point or attempting a play for 2 points. The team with the most points at the end of the game wins. Different rules regarding length of game, overtime, out of bounds, and possession are based on the level of play (high school, college, professional).
- Football is a high-velocity contact/collision sport with increased risk of injury. Physician coverage for games is optimal, and often required, depending on the level played and league and/or state regulations.
- Various medical as well as musculoskeletal issues need to be considered in treating football players at all competitive levels.

INJURY STATISTICS

- An estimated 1.2 million football-related injuries are sustained per year; risk of injury increases as teams get older and decreases with more experienced coaching staff.
- National Collegiate Athletic Association (NCAA) Injury Surveillance System injury rate was 3.7 to 4.4 per 1000 athlete exposures (AE) between 1986 and 1994.
- Injury rate for NCAA sports between 1988-1989 and 2003-2004 was highest in football compared to other sports. This is true for practices (9.6 injuries per 1000 AE) and games (35.9 injuries per 1000 AE).
- **Game injury rate is more than 9 times higher than the in-season practice injury rate, and spring football practice injury rate is 2 times higher than the fall in-season practice injury rate.**
- Between 1988-1989 and 2003-2004, the preseason injury rate was higher than both the regular season and postseason injury rate (7.05 vs. 2.02 and 7.05 vs. 1.70 per 1000 AE, respectively). Preseason is a critical time to stress proper stretching, warm-up, fluid and nutritional replacements, recovery, technique, and supervision.
- For injury rates compared with other NCAA sports, see Figure 57-1 and Table 57-1.



Overall **(A)** game and **(B)** practice injury rates for 15 sports, National Collegiate Athletic Association, 1988–1989 to 2003–2004. Although data for 15 total sports are presented, fall and spring football are reported separately for practices; because no "official games" are played during spring football, only fall football is listed for games.

Figure 57-1 Game and Practice Injury Rates in Sports. *(From Hootman JM, Dick R, Agel J: Epidemiology of collegiate injuries for 15 sports: Summary and recommendations for injury prevention initiatives. J Athletic Training 42(2):311-319, 2007.)*

Table 57-1 INJURY RATES (PER 1000 ATHLETIC EXPOSURES)

Sport	Practice	Game
Spring football	9.6	
Football	3.8	35.9
Wrestling	5.7	26.4
Women's gymnastics	6.1	15.2
Women's soccer	5.2	16.4
Men's soccer	4.3	18.8
Ice hockey	2.0	16.3
Men's basketball	4.3	9.9
Women's volleyball	4.1	7.7
Men's lacrosse	3.2	12.6
Women's basketball	4.0	7.7
Field hockey	3.7	7.9
Women's lacrosse	3.3	7.2
Women's softball	2.7	4.3
Baseball	7.9	5.8

Modified from Hootman JM, Dick R, Agel J: Epidemiology of collegiate injuries for 15 sports: Summary and recommendations for injury prevention and initiatives. J Athl Train 42(2):311-319, 2007.

- For games, knee internal derangements, ankle ligament sprains, and concussions account for the majority of injuries (17.8%, 15.6%, and 6.8%, respectively). For fall practices, knee internal derangements, ankle ligament sprains, and upper leg muscle-tendon injuries account for the majority of injuries (12%, 11.8%, and 10.7%, respectively). Heat illness accounts for 3.9% of fall season injuries. Spring practice injuries are similar to those seen in the fall in regard to the most common body parts injured. Cervical spine injuries can have catastrophic potential but fortunately have declined since rule modifications were made about tackling and blocking techniques, improved fitness, coaching, and equipment.
- Ankle sprains account for 13.6% of all injuries (0.83 injuries per 1000 AE), anterior cruciate ligament (ACL) injuries for 3% of all injuries (0.18 injuries per 1000 AE), and concussions for 6% (0.37 injuries per 1000 AE). At the NCAA level, spring football ankle sprains account for 13.9% of all injuries (1.34 injuries per 1000 AE), ACL injuries for 3.5% (0.33 injuries per 1000 AE), and concussions for 5.6% of all injuries (0.54 per 1000 AE).
- Player contact is the most common mechanism of injuries in football, and this is true for games as well as fall and spring practices.
- Player position plays a factor in likelihood of injury, with game injuries by position being most common in running backs (19.6%), followed by quarterback (17.5%), linebacker (15.5%), flanker/wide receiver (14.4%), defensive back (11.7%), defensive lineman (11.3%), and offensive lineman (9.9%).

MEDICAL PROBLEMS

Preparticipation Physical Examination

- The preparticipation physical examination is probably the most important part of initial evaluation of any athlete. Essential that evaluation is performed properly, thoroughly, and with sport-specific objectives (see Chapter 3, The Preparticipation Physical Examination).
- Three areas of importance in preparticipation physical examination: cardiac, musculoskeletal, neurologic. They are stressed not only in history, but also on physical exam.
  - Family history of sudden cardiac death, myocardial infarction before age 50, Marfan syndrome, hypertrophic cardiomyopathy.
  - History of syncope, chest pain, dizziness.
  - History of prior injury or incomplete rehabilitation.
  - History of previous head or neck injury, concussions, stingers or burners, as well as any work-up in past is important.
  - Other areas of importance: exercise-induced asthma, single organs, heat intolerance, appliances (e.g., dental plates), medications, allergies, medical conditions, sickle cell disease, immunizations.
- Historical questions to assess risk for sudden cardiac death, preexisting musculoskeletal injuries, or anatomic conditions that put athlete at risk for injury. **Thorough history is essential.**
- Physical examination
  - Emphasize cardiac, musculoskeletal, and neurologic systems.
  - Ensure adequate cervical spine protection, joint stability, absence of cardiac abnormalities, neurologic integrity.
  - Screen for inflexibility, muscle imbalances, improper rehabilitation.
- **Nutrition issues:** Need for strength gaining often puts athlete at risk for abuse of performance-enhancing drugs. Use of protein supplementation should be discussed. Athlete should understand basics about good nutritional balance and risks of excessive protein load as well as additional costs of protein supplements. Emphasis should be on healthy, natural, well-balanced diet (see Chapter 5, Sports Nutrition). The use of unsupervised nutraceuticals should be discouraged.
- **Fluids:** A study in high school football players in 50-play scrimmage simulation demonstrated no difference in anaerobic performance with 7% glucose polymer beverage containing electrolytes compared with water, but beverage had positive effect on maintaining plasma volume during recovery from anaerobic exercise.
- In 2003 as response to heat-related deaths in preseason football, the NCAA instituted rules that address heat illness by requiring 5-day acclimatization period during which practice time is limited. American College of Sports Medicine published expert roundtable recommendations, "Youth Football: Heat Stress and Injury Risk," which expounded on these risks for youth football.
- Given risk for heat-related illness with sickle cell disease, role for screening for all African- American athletes should be considered and counseling and modification of training regimens at altitude occur as needed.

Strength and Conditioning

- Important in young athlete.
- Stress maintenance of good flexibility program in conjunction with core and overall strengthening.
- Sport specificity in training; endurance work as baseline.
- Interval training and hill training for explosive speed.
- Supervision and proper technique in strengthening program, especially if free weights are used.

Infections

- General guideline is to avoid exercise during acute infection; no participation if fever is higher than 100° F. Avoid common source water outbreaks. To avoid spread of infection, athletes should not share water bottles.
- **Myocarditis** reported as cause of sudden cardiac death in athletes.
- **Skin infections** (herpes gladiatorum, ringworm) often are spread from person-to-person contact. Antiviral, antifungal, and antibacterial treatment necessary with protective covering to avoid transmission. Risk for methicillin-resistant *Staphylococcus aureus* (MRSA) of significant concern because of number of athletes participating. Important to avoid sharing towels, razors, toothbrushes, as well as ensuring proper hygiene. Rigorous cleaning of shared athletic training facilities and daily skin checks by athletic medicine staff to evaluate any suspicious skin lesions.

- **Infectious mononucleosis** is additional consideration for splenic rupture. Caused by Epstein-Barr Virus (EBV). No activity for initial 3 to 4 weeks of symptoms, then dependent on athlete's clinical condition and presence or absence of splenomegaly, which is present in majority of cases of EBV. Abdominal ultrasound useful for accurate assessment but not generally useful unless serial assessments performed. Recent position stand regarding mononucleosis in the athlete was written for the American Medical Society for Sports Medicine (AMSSM) and published in the Clinical Journal of Sports Medicine in July 2008).
- **Human immunodeficiency virus (HIV)**
  - No reason to disallow competition. Study in professional football players found 3.7 bleeding injuries per game for each team involving 3.5 players and found that 88% of bleeding injuries were abrasions, with remainder lacerations. Risk for transmission of HIV estimated to be less than 1 per 85 million game contacts.
  - Universal precautions should be used for all body fluids. More important to consider activities **off** field than on field regarding risk factors.
  - More substantial risk for **hepatitis.** Same precautions apply. Hepatitis vaccination series recommended.

HEAD INJURIES

Statistics

- Causes of minor head injury (according to National Head Injury Foundation): motor vehicle accident (46%), falls (23%), sports (18%), assaults (10%).
- Minor head injury is most common head injury, estimated to occur at a rate of 250,000 per year in contact sports. Estimated 20% of football players sustain cerebral concussions (see Chapter 39, Head Injuries).
- Team Physician Consensus Statement represents most recent consensus document to address mild traumatic head injury (concussion) in athletes.

NCAA Data on Head and Neck Injury

- Women's ice hockey had highest rate of head injuries of sports monitored, followed by football, field hockey, women's lacrosse, and men's soccer. Rate for women's ice hockey somewhat questionable given short period of time injury statistics have been tracked (Table 57-2).

Table 57-2 NCAA DATA ON CONCUSSION INJURIES: 1988-1989 THROUGH 2003-2004

	Concussion‡	
	%*	**IR†**
Sports with no head protection		
Field hockey	3.9	0.18
Women's lacrosse	6.3	0.25
Men's soccer	3.9	0.28
Women's soccer	5.3	0.41
Women's basketball	4.7	0.22
Men's basketball	3.2	0.16
Wrestling	3.3	0.25
Sports with head protection		
Ice hockey	7.9	0.41
Football	6.0	0.37
Men's lacrosse	5.6	0.26
Women's softball	4.3	0.14
Baseball	2.5	0.07

*Percentage of all reported injuries.
†Injuries per 100 athletic exposures.
‡Concussion = subset of all head injuries.
IR, injury rate.

- Injury rates in sports with no head protection, such as men's soccer, women's soccer, and field hockey, are comparable to helmeted sports of ice hockey, football, and men's lacrosse (see Table 57-2). Helmets do not prevent concussion.
- Player contact is primary injury mechanism for football.
- In football, spearing technique is associated with significant cervical injuries and head-to-head contact is associated with significant risk for head injury. Rules to prevent these mechanisms of injury exist, though enforcement is often difficult. Point of emphasis for officials in 2008 to call head-to-head contact fouls.

CERVICAL INJURIES

Neck pain and radiating arm pain, paresthesias, weakness, or loss of cervical motion are criteria for removal from the game and further work-up.

Myofascial Sprains

- Muscular or ligamentous injury to neck (most common form of neck injury).
- Presents with paravertebral spasm, decreased range of motion, usually without radicular or neurologic symptoms.
- X-rays to rule out fracture or ligamentous instability.
- Treatment includes nonsteroidal anti-inflammatory drugs (NSAIDs), rest, physical therapy.
- Return to play when full active range of motion with little or no pain, absence of neurologic findings, and negative work-up for more severe injury.

Brachial Plexus Injuries ("Burner" or "Stinger")

- Usually occur with blow to or distraction of neck and shoulder, pinching or stretching brachial plexus.
- Transient numbness or paralysis in the involved upper extremity; beware of bilateral symptoms, which may represent transient quadriparesis or spinal cord involvement.
- With first occurrence, consider cervical spine films to evaluate for congenital anomalies or spinal stenosis.
- Treatment includes NSAIDs, rest, and physical therapy.
- Return to play with full clinical strength and resolution of neurologic symptoms, especially in vertebrae C5 or C6 nerve distribution (not dependent on electromyographic findings); use protective equipment, including neck roll, which must be well fitted to tightly secured shoulder pads. Loose shoulder pads eliminate beneficial effect of neck roll. Be aware of delayed weakness. Athlete should be re-examined 24 hours later.

Fractures and Dislocations

- Less frequent after installation of rules banning "spearing" and use of top of helmet to strike another player. Injury still occurs with inadvertent hitting of crown of helmet on another player as players aggressively move to ball after initial contact is made.
- If neurologic injury present or fracture or dislocation suspected, spine must be immobilized until properly assessed, including x-rays.
- Never remove helmet or shoulder pads until the spine is properly immobilized and protected; cut or remove face guard for airway management when transporting the athlete. If removal of shoulder pads and helmet are necessary, remove both at same time to maintain neutral alignment of cervical vertebrae.
- Be aware of emergency personnel guidelines that may advocate helmet removal—protect your players! Pediatric head size is relatively larger than the shoulders—take this into consideration with young athletes.

Return-to-Play Criteria after Cervical Injury

- **Absolute contraindications** to return to collision sports
  - **Congenital:** Odontoid hypoplasia, atlanto-occipital fusion, Klippel-Feil anomaly with occipital-cervical involvement or mass fusion.
  - **Developmental**
    - Spinal stenosis with episode of cervical cord neurapraxia and ligamentous instability or cord edema by magnetic resonance imaging (MRI) or more than one recurrence.
    - "Spear tackler's spine" (stenosis with persistent straightening of cervical spine, often rigid in flexion and extension).
  - **Traumatic conditions**
    - Any C1 to C2 injury with ligamentous laxity or C1 to C2 fusion.
    - Unstable ligamentous injuries of C2 to C7.
    - Vertebral body fractures with sagittal component of displacement into the canal.
    - Healed fractures with residual instability, neurologic finding, limitation of motion.
    - Disk herniation with neurologic findings or limited motion.
    - Status postfusion of more than three levels.
  - **Relative contraindications**
    - **Developmental:** Episode of central cord neurapraxia (with resolution) and vertebral body-to-canal ratio of 0.8 or lower.
    - **Traumatic:** Full pain-free range of motion and neurologically intact with healed displaced body compression fracture or neural ring fracture; two- or three-level fusion.
  - **No contraindication**
    - **Congenital:** Klippel-Feil anomaly with stable one- to two-level fusion below C2 and full range of motion.
    - **Developmental:** Stenosis ratio of 0.8 or lower in otherwise asymptomatic patient.
    - **Traumatic:** Full, pain-free range of motion and neurologically intact with healed stable compression fracture or healed spinous process fracture; solid one-level fusion after disk excision at C3 or below.
    - Study of collegiate football players found that players with Torg ratio lower than 0.8 had three times risk of incurring stingers.

UPPER EXTREMITY

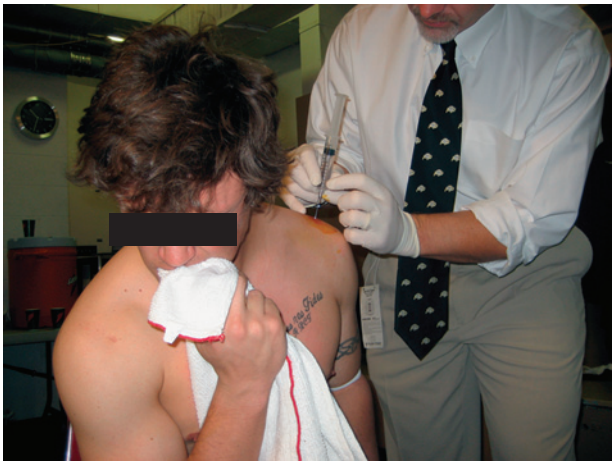
Shoulder Girdle (Clavicle Fractures)

- Mechanism or injury: direct blow (most common) or impact onto point of shoulder.
- Conservative treatment usually successful with figure-of-eight strap or sling for 6 to 8 weeks; surgery if danger of skin necrosis or soft tissue interposition. Consider surgery if overlapping fragments of 1.5 to 2 cm.
- Return to play with resumption of strength, healed fracture, and special shoulder pads for protection.

Acromioclavicular Joint

- **Osteolysis of clavicle:** Usually secondary to repetitive or traumatic cause (weightlifting vs. falling onto the point of the shoulder).
  - Attempt conservative therapy with rehabilitation, injections, acromioclavicular padding during play.
  - Distal clavicle excision if continued pain, dysfunction.
- **Incomplete acromioclavicular separations** (coracoclavicular ligaments intact, grade I and II acromioclavicular sprains) (see Chapter 43, Shoulder Injuries).
  - Acromioclavicular injuries most commonly result from fall onto point of shoulder (Fig. 57-2).
  - Diagnosed by tenderness directly over acromioclavicular joint, pain with resisted adduction of arm across the chest; x-rays with weights sometimes helpful in determining severity between type III and V.
  - Conservative treatment with RICE (rest, ice, compression, elevation), physical therapy; return to play with acromioclavicular padding after restoration of strength. Injections with a local anesthetic may be used judiciously for return to play with injury during game or to enable play in games with a sprain resolving in the semiacute phase (see Fig. 57-2).
- Complete acromioclavicular separations (grade III to VI acromioclavicular sprains)
  - Diagnosed by palpable step-off between clavicle and acromion. Can also confirm with radiographs.
  - Treatment controversial; mostly conservative unless extensive muscle stripping or penetration of the distal clavicle through trapezius is present.
  - Strength and endurance comparable in operative versus nonoperative results for grade III injuries.
  - Return to play after restoration of strength, motion, and with acromioclavicular padding.
  - Grades IV, V, and VI require surgical treatment.

Injury to acromioclavicular joint with direct mechanism of fall on to top of shoulder causing sprain of acromioclavicular ligaments.



Anesthetic injection during game for grade I acromioclavicular joint sprain with proper sterile technique.

Figure 57-2 Acromioclavicular Injury.

Glenohumeral Instability

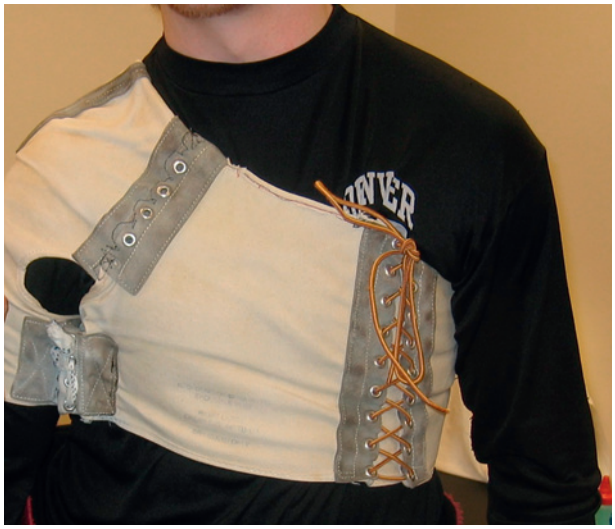
- Common football injury, typically seen as either anterior or posterior pattern. Usually traumatic in nature although multiligamentous laxity can contribute to the instability pattern.
- **Anterior** most common secondary to abduction-external rotation force; sometimes results from improper tackling technique.

- Acute injury should be treated with gentle reduction, which can be performed on field by experienced physician; immobilization following acute injury is controversial. Early rehabilitation is important and bracing can control abduction-external rotation moments (the position at risk) and allow early return to play (Fig. 57-3). Immobilization and rehabilitation, however, have not been shown to affect high redislocation rate in young people.
- X-rays including apical oblique to evaluate for reduction, Hill-Sachs, or bony Bankart lesion.
- Arthroscopy or acute stabilization in young people shown to significantly decrease the dislocation rate; decision making should be tailored to each case depending on risk factors, career goals, timing.
- Surgical stabilization of choice is surgeon dependent; trend is for arthroscopic stabilization in the football player.

- **Posterior** instability/labral tears are common in offensive lineman; however, traumatic posterior dislocation is uncommon.
  - **Diagnosis** of posterior instability/labral tear often made with history of pain in posterior shoulder when arms are extended forward and are presented with a posterior directed force, thus causing pain and/or shifting of shoulder posteriorly. Examples include an offensive lineman jamming an opponent to block or an athlete performing a bench-press lift. On examination a posterior jerk test will often reproduce the symptoms.
  - In a traumatic posterior dislocation, the exam features an anterior prominent coracoid, and the arm is adducted and internally rotated with minimal external rotation. Adequate x-rays are crucial to avoid missed diagnosis.
  - **Treatment** should consist of rehabilitation; however, no bracing can control posterior directed force on shoulder and if player remains symptomatic then surgical posterior stabilization is the treatment of choice. Typically there is a labral tear and players have been shown to have a successful return to football after surgery.

Rotator Cuff/Biceps

- Uncommon in younger population but may occur in quarterbacks; important to rule out subtle instability presenting as impingement syndrome.



Example of shoulder harness to help assist player with anterior shoulder instability in preventing abduction and external rotation during competition.

Figure 57-3 Anterior Shoulder Instability.

- Evaluate for supraspinatus weakness by testing resisted abduction with arm in 30 degrees of forward flexion from 90 degrees of straight abduction, with internal rotation; impingement test by forward flexing arm in internal rotation.
- Look for "Popeye" arm, weakness in forward flexion, and supination (Speed's test), if long head of biceps rupture suspected.
- X-rays of shoulder including anteroposterior, supraspinatus outlet view (lateral scapula with 10-degree caudal tilt) to evaluate acromial morphology (hooked vs. straight); MRI if complete tear is suspected. MRI of suspected biceps rupture helpful to rule out large intra-articular tendon fragment.
- Impingement syndrome with rotator cuff tendinopathy usually responsive to structured therapy program. Surgical repair is usually indicated if full-thickness tear in young population.
- Biceps tendon tendinitis and proximal rupture are usually responsive to rest followed by strengthening program; surgical tenodesis indicated for select high-demand positions, to include arthroscopic debridement of symptomatic intra-articular tendon fragment. Distal forearm rupture most commonly treated by surgical reattachment.

Contusions/Exostosis

- Common, especially in linebackers; occurs from direct blow to shoulder, resulting in injury similar to hip pointer. Presentation will be weakness in deltoid and rotator cuff region. Often will present with exam similar to that of someone with rotator cuff tear.
- Periosteal stripping in region of deltoid insertion may lead to "dotted veil" appearance on x-ray and eventual formation of mature exostosis.
- Initial treatment for contusions similar to treatment for quadriceps contusion—ice, rest, aspiration of hematoma if present, avoidance of heat and other modalities that may exacerbate bleeding and subsequent hematoma formation.

ELBOW AND FOREARM

Valgus Injuries (Overuse)

- Occurs occasionally in quarterbacks; diagnosed by pain and tenderness distal and posterior to medial femoral condyle with valgus stress and occasional ulnar nerve symptoms. MRI is becoming study of choice to verify diagnosis.
- Conservative treatment includes rehabilitation, protective function; may take 4 to 6 months to return to sport.
- Surgery if patient failed adequate rehabilitation and desires to continue as high-demand thrower; arthroscopy to remove loose bodies and examine degree of medial laxity followed by ulnar collateral ligament if needed.

Hyperextension/Dislocation Injuries

- Occurs with fall onto outstretched hand, often with forced extension during tackling.
- Dislocations can be reduced with gentle traction with elbow in semiflexed position (for posterior dislocations—most common).
- Both hyperextension and dislocation injuries require x-ray to rule out fracture and neurovascular assessment of hand and forearm.
- Treat both injuries with early protected range of motion; return to play with protected bracing after strength and motion restored (usually 3 to 6 weeks for dislocation, often sooner with hyperextension injury) (Fig. 57-4).

Olecranon Bursitis

- Traumatic bursitis common secondary to impact with tackling.
- Examination of skin important to rule out open communication with bursa; x-ray to evaluate for bony spurs.

Hinged elbow brace utilized to protect players elbow that has valgus laxity after elbow dislocation.

Figure 57-4 Valgus Laxity.

- Aspiration for persistent or large collection; consider sclerosing agent or excision with recurrence.
- Return to play with protective pad.

WRIST AND HAND

Wrist Sprains and Fractures

- Injuries of wrist must be evaluated carefully for location of tenderness and pain with movement; fractures and ligament injuries must be ruled out clinically and radiographically before diagnosis of wrist sprain.
- Scaphoid fractures
  - Acute nondisplaced fracture can be treated with long arm thumb spica cast for 4 to 6 weeks, followed by short arm thumb spica until healed. However, current trend is to treat these fractures intraosseous fixation in high-demand athlete who cannot afford prolonged healing period and early return can be done.
  - **Return to play** depends on position; for example, linemen can often return quickly with appropriate outer cast padding.
  - **Displaced** or symptomatic nonunion needs open fixation, often with bone grafting; return to play with protective splinting after adequate healing and strength restoration.
- Distal radius and ulna fractures
  - **Conservative versus operative treatment** depends on fracture pattern and stability.
  - **Return to play** depends on fracture stability and player position; may take 2 to 3 months before sufficient healing and strength.
- Carpal fractures other than scaphoid
  - Avulsion fractures are most common; these can often be treated with immobilization and early return to play if position allows.
  - Important to rule out carpal dislocations (e.g., lunate/perilunate) or ligamentous injuries (e.g., scapholunate) with appropriate stress x-rays; MRI if plain films are inconclusive.

Injuries to Thumb

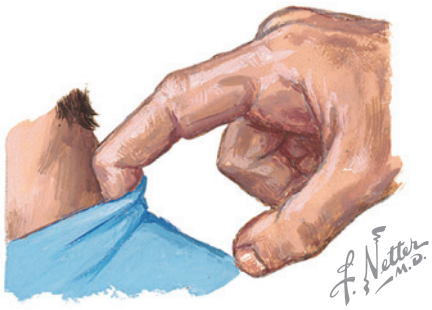
- **Collateral ligament injuries**
  - Common injuries result from adduction (radial) or abduction (ulnar ligament) forces applied to metacarpophalangeal (MCP) joint. Diagnosis by reproduction of pain with stress testing, tenderness to palpation over radial or ulnar aspect of MCP joint.
  - Radial ligament injuries and incomplete ulnar ligament injuries can be treated with protective splinting; early return to play in most positions.
  - Complete tear of ulnar collateral ligament (as confirmed by stress radiography) most commonly treated by open ligament repair because of high prevalence of Stener lesions. Could be treated with casting and early return to play after repair.
- **Fractures and dislocations of thumb**
  - Fractures and dislocations at MCP joint are also common because of precarious position of thumb.
  - MCP dislocations are most commonly dorsal; important to differentiate simple (reducible) from complex (irreducible, requiring open reduction secondary to interposition of volar plate). Can return to play early with protective splint for 4 to 6 weeks.
  - Intra-articular fractures (Bennett's) are usually unstable and require fixation. Phalanx fractures, if stable, can be treated with protective splinting and early return to play.

Hand Fractures/Dislocations Excluding Thumb

- Occur frequently from direct impact; diagnosis confirmed by x-ray.
- Fractures of fourth and fifth metacarpals can usually be manually reduced to acceptable position and treated with protective splinting. Less tolerance for displacement with second and third metacarpals; require operative fixation more frequently.
- Proximal phalanx fractures often require operative fixation to maintain length, proper rotation.
- Middle phalanx fractures more commonly amenable to closed reduction, buddy taping.
- Distal tuft fractures should be treated as soft tissue injuries; if suspected, examination of nail bed and meticulous repair if damaged or removal from fracture site if incarcerated. Time-honored hot paper-clip or electrocautery relieves painful subungual hematomas.
- Most hand fractures, if stable, can be treated with protective splinting, early return to play.
- Dislocations are most commonly dorsal and can be treated by closed reduction, continuous buddy taping for 3 to 6 weeks, early return to play
  - X-rays after game important to rule out bony injury.
  - If dislocation of proximal interphalangeal joint is volar (rare), need to treat in same manner as rupture of central slip of extensor tendon.

Tendon/Capsular Injuries

- Flexor tendon injuries usually occur with attempts to grab another player's jersey, resulting in pain and inability to flex distal interphalangeal (DIP) joint of involved finger (Fig. 57-5).
  - Important to determine level of retraction of tendon; if it retracts into palm, blood supply is significantly compromised, and repair should be performed as soon as possible (within 7 days). Less retracted tendon injuries can be repaired later (blood supply from vincula maintained, but best results with early diagnosis and repair).
  - Always obtain x-ray because bony avulsion injuries, which usually limit amount of retraction of tendon, can occur. Early repair of bony injury also yields best result.
- Extensor tendon avulsions of distal phalanx (mallet finger) usually occur by "jamming" finger, causing forced flexion of DIP joint.
  - Exam reveals inability to extend DIP joint.
  - Always obtain x-ray to determine if bony avulsion has occurred; subluxation of DIP joint (usually occurs with fragment involving more than one-third of joint surface) requires more aggressive treatment.



"Jersey Finger," example of injury to flexor tendon of hand while player trying to tackle opponent and finger getting stuck in jersey.

Figure 57-5 Jersey Finger.

- Mallet fingers should be treated with continuous extension of DIP joint with splint, free range of motion of proximal interphalangeal (PIP) joint, early return to play.
- Extensor tendon avulsions of central slip (insertion into proximal aspect of middle phalanx) also occur by "jamming" finger and are often misdiagnosed as capsular sprain.
  - With other fingers held in extension and MCP joint flexed on involved finger, test for ability to extend PIP joint. If ability absent, suspect central slip rupture.
  - X-rays important to rule out fracture.
  - Treat with PIP joint held in continuous extension for at least 6 weeks with DIP joint free. Failure to recognize and treat these injuries results in boutonnière deformity.
- Capsular sprains and lateral collateral ligament injuries can be treated by continuous buddy taping for 6 weeks and return to play.

CHEST AND ABDOMEN

Rib Fractures

- Due to direct blow to ribs with resultant pain, especially when breathing.
- X-rays important to rule out presence of pneumothorax (collapsed lung).
- Return to play with protective "flak jacket" when symptoms are tolerable.

Sternoclavicular Joint

- Dislocations or injuries caused either indirectly by blow to shoulder or directly by injury to upper chest.
- Diagnosed by pain and swelling at sternoclavicular region with exacerbation during arm range of motion.
- X-rays important to rule out posterior dislocation, which often requires surgical reduction to avoid injury to major vessels.
- Anterior dislocations most common and usually treated conservatively with return to play when strength and motion restored.

Pectoralis Major Ruptures

- May occur with forceful extension/external rotation of arm. Pain and ecchymosis most commonly along anteromedial aspect of arm (most ruptures occur near insertion onto humerus) and inability to flex pectoralis with adduction and internal rotation.
- Suspected incomplete injuries can be treated with rest and ice, with return to play upon restoration of motion and strength.
- Complete ruptures are usually treated surgically because of significant loss of strength if treated conservatively.

Rectus Abdominis Strain

- Forceful resisted contraction of abdominal musculature can lead to intramuscular strain or avulsion from iliac crest or pubis. Diagnosed by area of tenderness and pain with attempted flexion of trunk.
- Treatment is usually conservative, with ice, stretching, and taping of trunk to limit flexion and rotation on return to play.

Athlete's Groin (Sports Hernia)

- Often confused with osteitis pubis; diagnosis can be difficult.
- X-rays necessary to rule out symphysis pubis changes. Conservative management often prolonged and requires extensive physical therapy efforts for mobilization of all muscles attaching to anterior pelvic rim. Surgical treatment has mixed results, but has improved as understanding of this issue and technique has progressed.

THORACOLUMBAR SPINE

Thoracic or Low Back Strain

- Most common injury to spine in contact sports; muscle spasm or ligamentous strain usually occurs with bending or twisting during forceful activities.
- Often secondary to fatigue or inadequate conditioning of the paraspinal muscles.
- Most commonly occurs without radicular symptoms; suspect disc pathology if radiation of pain into lower extremities.
- Plain anteroposterior, lateral, and oblique x-rays helpful to rule out any osseous injury, neural arch involvement, or Schuman's kyphosis.
- Treatment consists of control of initial inflammatory phase with ice, rest; progression to back rehabilitation program when tolerated.
  - Most uncomplicated sprains respond in 7 to 10 days.
  - Flexibility and conditioning program during season and in off-season important to prevent recurrence.

Herniated Nucleus Pulposus

- Injury pattern often similar to that of strain, but with radiculopathy into one or both lower extremities, weakness, with or without diminished reflex. Straight leg raising often abnormal. Often with complaints of paresthesias and pain in leg, which are more bothersome than back complaints.
- Plain films of lumbar spine and MRI often help document injury.
- Initial treatment is same as for lumbar strain. Consider epidural steroids or disc excision if no improvement with conservative care or with progressive neurologic insult.

Spondylolysis/Spondylolisthesis

- Common in linemen secondary to repetitive loading on posterior elements in extended position.
  - Complaints similar to low back strain, but hamstring spasm and pain with resisted extension are also common. Single-legged back extension is painful.
  - Previous history of spondylolisthesis or significant back pain important to determine chronicity.
- Anteroposterior, lateral, and oblique **x-rays** necessary to evaluate for "broken neck on Scotty dog" to confirm spondylolysis and to evaluate for presence of translation of vertebral body (spondylolisthesis). Bone scan is often helpful if x-rays are equivocal or to differentiate acute neural arch injury from low back strain with previously healed spondylolysis.
- **Treatment** consists of restriction from play, abdominal and lumbar stretching, strengthening activities, and occasional use of lumbar orthosis.
  - Resolution of hamstring spasm is often reasonable gauge to begin functional progression in rehabilitation as tolerated.

- Clinical progress more reliable indicator than x-rays, which often do not demonstrate bony healing even when patient becomes symptom free.
- Known presence of spondylolysis or spondylolisthesis in asymptomatic player is not contraindication to play.
- Symptomatic, skeletally immature patient with high-grade spondylolisthesis (slippage greater than half width of vertebral body) is candidate for fusion.

Thoracolumbar Fractures

- Infrequent injuries that can occur with severe direct impact to spine or violent muscle contractions.
  - If fracture is suspected, important to maintain spine precautions before any movement of patient.
  - Neurologic assessment important in initial exam.
  - Patients generally complain of severe back pain, exacerbated with any attempted movement.
- **X-rays** of spine, including anteroposterior, lateral, and oblique views to evaluate injury.
  - MRI if any neurologic injury is present with fracture.
  - Consider CT scan or MRI of abdomen if visceral injuries (rare) are suspected.
- **Treatment** depends on fracture pattern and stability.
  - Isolated stable transverse process or spinous process fractures usually amenable to brief period of rest with return to play when symptoms tolerate (3 to 6 weeks).
  - Fracture treatment should follow accepted guidelines for particular fracture pattern (e.g., patients requiring lumbar fusion should not be allowed to return to contact sports).
  - Acute fractures with no history of prodromal symptoms can often heal if patient is adequately immobilized with lumbar orthosis and pantaloon leg extension.

HIP AND PELVIS

Soft Tissue Injuries

Hip Pointers

- Common term applied to injuries of iliac crest, including contusions from direct injury, muscle avulsions from violent contractions, and periostitis from repetitive abdominal muscular contractions or contusions.
- Skeletally immature players should be evaluated radiographically to rule out displaced apophysis from avulsion-type injuries.
- **Treatment** consists of rest, ice, stretching, and NSAIDs:
  - Most apophyseal injuries and muscle avulsions treated conservatively except for widely displaced apophyseal fractures and large muscle avulsions, which should be surgically repaired.
  - Return to play when symptoms tolerate with padding, heat, and stretching before play and ice afterward. Can consider injection with local anesthetic during game or pregame to mitigate symptoms from contusion over iliac crest.
- Trochanteric bursitis from direct blow also common injury in this region; treatment involves same protocol with judicious use of steroid injections if necessary.

Groin Pull

- Common term for injury to iliopsoas or adductor group of leg, usually caused by forced abduction, external rotation.
  - Common in older and deconditioned athletes.
  - Symptoms include pain with resisted adduction of leg or passive abduction, located along border of pubic ramus.
- X-rays of hip and pelvis to rule out avulsion fracture caused by iliopsoas (lesser trochanter) or adductor group.
- **Treatment** initially includes ice and rest, followed by stretching and strengthening when symptoms allow.
  - Best treatment is preventive by preseason strengthening program.
  - Symptoms can be persistent depending on severity; adequate strength necessary before play to prevent exacerbations.
  - Most avulsion injuries treated similarly, if not widely displaced.

Osteitis Pubis

- Common in football players, weightlifters. Exact cause unknown, but thought to be secondary to overuse of adductors and gracilis with gradual resorption of bone at muscular attachment sites (medial aspect of pubic bones).
- Symptoms include gradual onset of groin pain exacerbated by resisted adduction and tenderness along inferior and medial borders of pubic bones.
- X-rays may reveal resorption of bone at pubic symphysis with resultant widening and sclerosis of inferior pubic rami. Bone scans reveal diffuse uptake in involved areas of the pubic rami.
- Relative rest and anti-inflammatories most successful for this overuse injury, which often takes 2 to 3 months to resolve. Sometimes a corticosteroid injection maybe helpful in refractory cases.

Fractures/Dislocations

Hip Subluxation/Dislocation

- Unusual but potentially devastating injury secondary to possible osteonecrosis of femoral head.
  - Player experiences severe pain in hip with any attempted movement.
  - Posterior dislocation most common; leg appears shortened, internally rotated.
- X-rays before reduction necessary to rule out femoral neck fracture.
- Gentle reduction should be performed urgently in controlled setting after neurovascular status and x-rays are assessed.

Lesser Trochanter Avulsion/ Other Apophyseal Avulsions

- Most common under age 20 and results from severe contraction of iliopsoas muscle (or other muscular attachment).
- Symptoms include immediate severe groin pain exacerbated by attempts to flex hip joint or pain and tenderness to palpation in suspected apophyseal location with exacerbation on resisted muscular contraction.
- Treatment involves bed rest followed by gentle range of motion and partial weight-bearing when tolerated when x-rays reveal nondisplaced or minimally displaced fractures. Role of open reduction (based on degree of displacement) is controversial.
- Avulsion fracture in mature adults (age 20 or older) is often associated with neoplastic disease.

LOWER EXTREMITY

Thigh

Quadriceps Contusions/Exostosis

- Direct impact onto quadriceps muscle belly with resultant pain, decreased range of motion, and occasional palpable mass.
- Initial treatment of flexion, ice to minimize hematoma (not heat), followed by pain-free range of motion and functional rehabilitation (avoid heat, ultrasound, painful exercise). Immobilization in flexion overnight to minimize bleeding is often helpful.
- Conservative treatment for myositis ossificans unless functional impairment persists for more than 6 months.

- Return to play with extra thigh padding when full strength, function, and range of motion are achieved.

Hamstring Injuries

- Sudden pain in posterior thigh with rapid hamstring contraction; associated with fatigue, poor conditioning, inadequate stretching. Short head of biceps is most common.
- Prophylaxis with off-season conditioning, baseline hamstrings-to-quadriceps strength ratio of 0.6 or greater.
- Initial treatment of RICE, jogging, and pain-free functional rehabilitation when hamstrings 70% of baseline. If area of hamstring strain is focal then local corticosteroid injection can be considered and is supported by the literature.
- Return to play when isokinetic strength ratio approximately 0.6.

Knee and Leg

Ligament Injuries

MEDIAL COLLATERAL LIGAMENT

- Most commonly caused by direct impact onto lateral aspect of knee with applied valgus stress.
- Pain on medial aspect of knee with tenderness to palpation along course of medial collateral ligament: increased laxity to valgus stress at 30 degrees of flexion. Increased laxity when knee at full extension indicates more severe knee injury (often cruciate and/or posteromedial capsular disruption).
- Usually treated nonoperatively with protected weight-bearing, inflammation control, range of motion at 30 to 60 degrees, with full weight-bearing as tolerated. Progression to full range of motion as symptoms allow. Addition of progressive resisted exercises, and return to play with functional knee brace as injury allows.
- Complete or grade III injuries often take 4 to 6 weeks for return to full activity with protective brace.

ANTERIOR CRUCIATE LIGAMENT

- Most commonly results from noncontact twisting injury, but can result from direct blow to knee as well.
- History of hearing or feeling "pop" in knee, rapid effusion (within few hours) highly suggestive of anterior cruciate ligament disruption; exam reveals positive Lachman's sign, pivot shift.
- X-rays usually negative or may show small proximal lateral tibial plateau bony avulsion (Segond's sign).
- Treatment for cruciate-dependent patients or those who plan to continue to participate in high-risk sports is surgical in most instances; delaying surgery until knee range of motion is from full extension to greater than 110 degrees of flexion is currently believed to reduce incidence of arthrofibrosis. Concomitant injuries most commonly treated by conservative medial collateral ligament management with delayed anterior cruciate ligament reconstruction.
- Functional knee bracing on return to play remains controversial.

POSTERIOR CRUCIATE LIGAMENT

- Mechanism of injury most commonly hyperflexed knee or direct blow to anterior tibia while foot is firmly planted.
- Injury is often missed and high index of suspicion is necessary. Exam reveals increased posterior tibial translation at 90 degrees of flexion. Suspect more severe injury if posterior sag does not improve with internal rotation of the tibia.
- X-rays often negative or may reveal bony avulsion at tibial insertion.
- Treatment for isolated posterior cruciate ligament is controversial; conservative approach most common with focus on functional restoration before play. Surgical proponents recommend reconstruction because of high incidence of patellofemoral and medial arthritis with this injury, although current studies fail to show improved outcome with operative intervention.

POSTEROLATERAL INSTABILITY

- Isolated injury to this complex uncommon; usually accompanied by cruciate ligament injury. Direct blow to anteromedial tibia or severe twisting with foot planted is most common mechanism.
- Exam shows increased laxity at 30 degrees with varus stress and increased external rotation (Dial test). Effusion often absent with combined cruciate and posterolateral injuries secondary to capsular tear. Important to assess peroneal nerve function at time of injury.
- X-rays often negative but necessary to rule out avulsion fractures; MRI helps delineate structures injured. Beware of fibular avulsion of lateral hamstring.
- Treatment involves direct surgical repair/reconstruction of posterolateral structures (or biceps tenodesis if tissues severely compromised) and reconstruction of cruciate injury if present.

Meniscal Injuries

- Common injuries that can occur by contact or noncontact mechanisms; often twisting-type injury with joint line and/or posterior pain and complaints of locking, catching, or buckling. Unless tear is peripheral, effusion is usually not noticed until after first 24 hours.
- Exam often reveals joint line tenderness, effusion, pain with extremes of flexion, lack of full extension (or "locked" knee) if displaced bucket-handle is present. Nonspecific pain may be present with circumduction maneuvers.
- X-rays are negative; MRI is diagnostic test of choice when exam is equivocal.
- Arthroscopy is indicated when symptoms do not respond to rest and control of inflammation; repair instead of excision of meniscus should be attempted whenever feasible.

Patellofemoral Disorders

PATELLAR DISLOCATION

- Usually noncontact, external rotation injury to leg resulting in severe anterior knee pain with buckling or an inability to bear weight.
- Knee diffusely swollen with significant tenderness along medial retinaculum, lateral trochlea, and patellar articular surfaces; apprehension with attempts at patellar translation (usually reduces spontaneously).
- X-rays, including Merchant view, necessary to rule out patellar or trochlear ridge fracture or bone fragments in joint.
- Treatment includes RICE, with progression to range of motion and quadriceps strengthening for initial injury; return to play usually requires 3 to 6 weeks. Repeat dislocators often require surgery to reconstruct medial structures and alter mechanical alignment.

PATELLAR/QUADRICEPS TENDINITIS (JUMPER'S KNEE)

- Overuse injury common to football; secondary to frequent stops and starts and eccentric loading of extensor mechanism.
- Most common areas of tendinitis are patellar tendon at inferior pole of patella and quadriceps tendon insertion into superior pole, but can occur anywhere along extensor mechanism.
- Prevention is optimal treatment, with conditioning including eccentric strength training and adequate stretching. Symptomatic players require relative rest (exercises that do not provoke symptoms) and anti-inflammatory modalities, with progression to concentric and isometric strength training, and

finally eccentric training when asymptomatic during other therapy.

FAT PAD SYNDROME (HOFFA'S DISEASE)

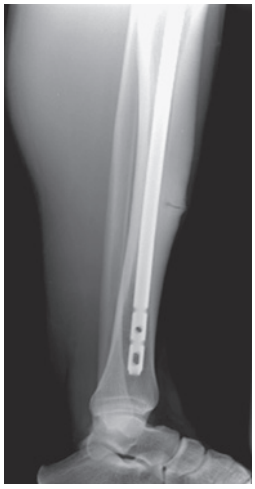
- Seen frequently secondary to contusions and resultant irritation to anterior aspect of knee.
- Tenderness to palpation usually present medial and lateral to patellar tendon and inferior patella; capsule in this region often feels "boggy."
- Treatment involves relative rest, anti-inflammatory modalities, and stretching. Sometimes corticosteroid injections can be helpful. Recalcitrant cases are uncommon, but usually respond to arthroscopic debridement.

Medial Tibial Stress Syndrome (Shin Splints)

- Characterized by diffuse pain along medial border of tibia. Common in early season with increased training, exacerbated during exercise, and may persist for hours to days.
- Area of tenderness usually along posteromedial border of tibia—less well localized than a stress fracture. Should not be exacerbated with passive ankle or foot motion.
- X-rays are usually normal but may reveal mild cortical hypertrophy. Bone scan helps distinguish from stress fracture and is negative or reveals diffuse linear uptake approximately one-third the length of tibia on posteromedial border.
- Rest is most effective treatment; anti-inflammatory modalities and stretching by themselves do not resolve condition. Gradual resumption of activities when pain free (usually 2 to 6 weeks). Orthotics may help prevent recurrence when significant pronation is present.

Tibial Stress Fractures

- Most common mechanism is rapid increase in training, especially running activities.
- Point tenderness on anteromedial aspect of tibia is highly suggestive of diagnosis.
- X-rays of stress fracture are often negative or reveal only cortical thickening; if no fracture is seen on plain films, bone scan is procedure of choice to make definitive diagnosis. MRI is often used to determine severity of stress fracture and thus predict recovery time. Beware of the "dreaded black line" (Fig. 57-6).
- Treatment involves rest followed by gradual **pain-free** progression of activities. One study suggests that use of pneumatic leg brace during rehabilitation protocol can shorten return-to-play time by several weeks. If fracture line and pain persists, then intramedullary tibial nailing is procedure of choice (Fig. 57-6).



Radiograph demonstrating intramedullary nailing for recalcitrant anterior tibial stress fracture in football player.

Figure 57-6 Recalcitrant Anterior Tibial Stress Fracture.

Ankle and Foot

Ankle Sprains

- Most common ligamentous injury. Structures injured depend on force and applied direction of injury; therefore, **history of injury important.**
  - Plantarflexion and inversion stresses injure anterior talofibular ligament (ATFL), with progressive force injuring calcaneofibular, posterotalofibular, and tibiofibular ligaments in severe injuries.
  - Inversion stress in neutral position injures calcaneofibular ligament (CFL).
  - Dorsiflexion stresses tibiofibular ligaments.
  - Eversion and external rotation injures deltoid ligament medially and tibiofibular ligament laterally, often accompanied by fibular fracture.
- **Palpation and stress tests** are used to determine ligaments injured.
  - Anterior drawer with 30 degrees of plantarflexion to assess ATFL; compare with contralateral side.
  - Inversion test: grasp heel and apply inversion stress; ability to palpate talar head indicates complete tears of both ATFL and CFL.
  - Squeeze test: squeeze fibula and tibia together near ankle joint; pain indicates probable syndesmotic/tibiofibular ligament injuries.
  - Evaluate for associated injuries, including peroneal tendon dislocation, fifth metatarsal fracture, posterior tibial tendon rupture, midfoot sprains.
- **X-rays** are necessary to rule out fracture of medial malleolus, lateral malleolus, and talar dome.
  - Stress x-rays are sometimes helpful in chronic ankle instability to evaluate ligament integrity—anterior drawer assesses ATFL, talar tilt test (inversion stress) with greater than 5 degrees of asymmetry indicates CFL instability, and loss of parallelism with Broden's view and inversion stress indicates subtalar instability. External rotation stress with mortise view to evaluate syndesmosis. Standing bilateral mortise view can identify syndesmosis laxity.
  - MRI useful to evaluate talar dome osteochondral injuries when suspected.
- **Treatment** for acute injuries includes anti-inflammatory modalities, early restoration of range of motion, and functional management dependent on severity.
  - Ankle braces to limit inversion/eversion stress used on grades II and III; cast immobilization rarely indicated in healthy athletes.
  - Proprioceptive and peroneal strengthening immediately for grade I, immediate to 1 week for grade II, when tolerated in grade III (1 week to 1 month).
  - Return to play depends on athlete's ability to demonstrate adequate performance in position-specific drills with few or no symptoms (usually less than 1 week for grade I, 1 to 2 weeks for grade II, approximately 1 month for grade III).
  - Unstable fracture patterns, syndesmotic disruption require surgical stabilization.
- **Functional instability**
  - Peroneal weakness, not ligamentous instability, responsible for approximately 50% of cases of persistent functional instability.
  - Broström lateral ligamentous repair currently most popular technique for ligamentous instability.

Midfoot Sprains

- Usually indirect injuries from forceful foot abduction when foot in fixed position or foot forced into severe plantarflexion.
- Patient demonstrates pain or tenderness in midfoot region, unable to tiptoe, may have flattening of longitudinal arch.
- Lateral and anteroposterior weight-bearing views: in normal anteroposterior view, medial border of second metatarsal base lines up with medial border of middle cuneiform; distance between second metatarsal base and lateral border of medial cuneiform should be symmetric on comparison views.
- **Treatment**
  - Conservative for normal x-rays. For severe medial sprain, use short leg non–weight-bearing cast 4 to 6 weeks followed by weight-bearing cast 2 to 3 weeks. Lateral midfoot sprains tend to resolve earlier.
  - For Lisfranc's dislocation, internal fixation to reduce second metatarsal base to medial cuneiform (weak link).
  - Return to sport with evidence of healing, such as ability to tiptoe and perform functional drills.

Fifth Metatarsal Base Fractures

- Three types
  - Tuberosity fractures from traction/avulsion-type injuries involving peroneus brevis or ligaments.
  - Metaphyseal fractures by adduction and inversion forces.
  - Jones fracture (approximately 3 cm distal to tuberosity) by repetitive or single force by sharp turning.
- Tenderness to **palpation** over fifth metatarsal base and pain with eversion forces present with all fracture types.
- **X-rays** of foot determine fracture type; bone scans help rule out Jones fracture when sclerosis, but no obvious fracture, present.
- Short leg cast or firm shoe with progression to weight bearing as tolerated for tuberosity and metaphyseal fractures. Treatment of Jones fracture controversial:
  - Conservative treatment: metatarsal support (cast, brace), weight-bearing vs. non–weight-bearing debatable.
  - In the high-demand athlete, percutaneous intramedullary compression screw improves healing time. Screw fixation is also most common treatment when significant sclerosis present or delayed union with conservative treatment.
  - Return to play with radiographic evidence of union and ability to perform functional tests.

Turf Toe

- Hyperextension or, less commonly, hyperflexion injury to first metatarsophalangeal (MTP) joint, associated with flexible footwear and more common on artificial turf. Severity of injury can range from capsular or ligamentous strain, to axial compression with resultant chondral injury, or MTP dislocation.
- Exam reveals tenderness about MTP joint, pain with passive dorsiflexion and often with decreased range of motion, and difficulty with push-off.
- **X-rays** to evaluate for bony injury to MTP joint. May see avulsions secondary to capsular injury; rarely, may have chondrolysis with repeated injuries. If plantar tenderness is present, obtain sesamoid views to rule out fractures in these structures.
- **Treatment** depends on severity of injury.
  - Capsular or ligamentous strains (characterized by localized tenderness) treated with ice, continuous play with taping to restrict motion. Also, stiff forefoot plate is helpful to limit motion of great toe.
  - Significant swelling, ecchymosis, and restricted range of motion indicate partial or complete capsular tear: treat with anti-inflammatory modalities, protective footwear, and restriction of play until clinical symptoms allow (usually 3 to 6 weeks).
  - Chondral injuries or dislocation requires treatment similar to lesser injuries, as well as limited weight-bearing and immobilization. Return to play depends on clinical symptoms (often prolonged).

PROTECTIVE EQUIPMENT

Football Helmet

Helmets must meet National Operating Committee on Standards for Athletic Equipment (NOCSAE) specifications.

Construction

- Outer shell is made of polymer plastic (polycarbonate or acryl butadiene styrene) that is both lightweight and able to withstand high impacts.
- Padding or suspension systems include pads, air and fluid-filled cells, combination of cells and pads, and suspension.
  - Air cells can be inflated or deflated to provide better fit.
  - Suspension types offer inferior protection and are being phased out.
  - Important to check for cracks in outer shell (especially at locations of face mask attachments), leaks, or other damage to padding system to ensure proper protection.
  - Helmet should have a NOCSAE label on outer shell warning of potential head or spine injury with improper helmet size.

Helmet Fitting

- Helmet should be donned by spreading ear holes apart with thumbs; helmet should turn minimally with attempts to rotate on head.
  - No space between jaw pads and face or back of head.
  - There should be one to two fingerbreadths above eyebrows; helmet should not come over eyes even with firm pressure on top of helmet.
  - Base of skull should be covered, but helmet should not impinge on cervical spine with full extension.
  - Jaw pads should provide snug fit and prevent lateral rocking.
  - Newer lightweight helmets are under development at present.
- Hair should be same length at time of fitting as it will be throughout season. Long hair should be wet when fitting to simulate sweaty conditions.
- Chin strap (four-point more secure than two-point) helps prevent forward and backward helmet motion; improper tension or strap failure can cause helmet to come off, or result in laceration to bridge of nose.
- Face masks used depend on position. Ball-carrying positions require mask that protects yet allows unobstructed view, whereas linemen need additional protection to prevent eye and facial injuries.
- Nontinted plexiglass shields are also worn for those seeking additional eye protection. Tinted plexiglass shields can be worn only by players requiring special assistance for elimination of sun because of a medical condition. They require physician's approval, and use is determined at time of play by officiating staff.
- Mouth guards (required in college and high school) can be custom-made, mouth formed by heating, or obtained in standard sizes. They help dissipate blows to chin, reducing intraoral and mandible injuries.

Shoulder Pads

- Cantilever and flat are two basic types.
  - **Flat pads** are worn by positions requiring greater glenohumeral motion.
  - **Cantilever pads** (named for bridge extending across shoulder) are worn by players in constant contact and requiring additional protection.

- Modifications depend on position and preference (e.g., linebackers and other players hit in standing position have pads that are larger anteriorly and slanted forward).

- **Shoulder pad fitting**
  - Measure from acromioclavicular joint to acromioclavicular joint, and compare with manufacturer's chart to approximate fit.
  - Neck opening should be large enough to prevent neck impingement when lifting arm overhead but not large enough to allow excessive sliding (one to two fingerbreadths between neck and inside padding).
  - Lateral aspect of pad should be just lateral to shoulder, with flaps or epaulets large enough to cover deltoid region.
  - Elastic axilla straps should be adjusted snugly but comfortably, to allow blows to be distributed across pads.
  - Anterior pads should meet but not overlap when laced up.
  - Pads should extend below nipple line anteriorly and approximately 1 inch below scapula posteriorly.
  - Important to inspect frequently for strap fraying or breakage, loose rivets, or cracks. Also check for sweat-compressed lining pads.

Other Protective Equipment

- **Standard equipment**
  - Hip pads are used to protect iliac crest and tailbone from injury.
  - Thigh pads and knee pads are inserted into pants snugly and should not slip easily.
- **Special equipment**
  - Neck rolls and collars are used frequently by players who have previously experienced "burners and stingers" or on prophylactic basis; efficacy of these devices in preventing further injury remains debatable. Neck rolls in college are often ineffective because of improper wear of shoulder pads. Shoulder pads must fit securely to chest to stabilize collar or neck roll and help decrease frequency of "burners."
  - Shoulder restraint devices restrict abduction and external rotation in chronic shoulder dislocators, but these limitations may affect player's capabilities in many positions.
  - Gloves and upper extremity pads are worn by linemen and other players to prevent finger injuries, contusions, and myositis ossificans.
  - Rib pads or vests are commonly worn by quarterbacks to prevent injuries to thorax.
  - Value of prophylactic knee brace in preventing injury remains questionable; therefore, it is not a mandatory protection device.

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Volleyball

58

Kevin Eerkes

GENERAL PRINCIPLES

Overview

- Volleyball is played by an estimated 200 million people worldwide and is growing in popularity.
- The game is played from the recreational to Olympic level.
- The focus is to hit a powerful, unreturnable ball into the opponent's court.
  - Achieved by complex setting and hitting schemes that often culminate in a "spike" or hit (a spike is a ball hit hard by a forceful overhead swing of the arm).
  - Players specialize in various aspects of the game to increase the ability to deliver an unreturnable ball (Table 58-1).
- Two most popular versions are six-player volleyball, played on a hard-surface indoor court, and two-player sand (beach) volleyball.
- Beach volleyball has been introduced most recently and has attracted players at a remarkable pace; it has boomed in popularity as a spectator sport.
- Volleyball requires explosive movements, quick direction changes, aerobic fitness, and body control.

Physical Conditioning

Off-Season Conditioning

- Endurance exercise such as running, swimming, and biking.
- Explosive exercise such as short sprints and vertical jumping.
- Resistance exercise to build gluteal, quadriceps, and calf muscles (for leaping).
- Strengthening exercise to build rotator cuff, periscapular muscles, and latissimus dorsi (for hitting ball).
- Stretching all major muscle groups after workouts, with exception of anterior shoulder ligaments, which are often already overly lax.

In-Season Conditioning

- Endurance exercise usually accomplished by frequent repetitions of volleyball drills.
- Strength training achieved by doing sports-specific tasks such as jumping, hitting/serving, and diving/digging drills.

Injury Overview

- Injuries are fairly common even though volleyball is considered a limited-contact sport.
- Certain positions are associated with specific injuries (see Table 58-1); most injuries occur in blockers and hitters.
- Most injuries are related to jumping.
  - A player landing from a jump near the net (front court) may accidentally come down on an opponent's foot that is in what's termed the "conflict zone."
  - The rules allow a player's foot to enter the opponent's side of the court as long as the foot remains in contact with the centerline that runs beneath the net.
- Higher conditioning and skill level appear to be protective; one study showed college freshmen and sophomores are injured more frequently than juniors and seniors.

Acute Injuries

- Ankle and finger sprains make up the majority of acute injuries (Fig. 58-1); strains, fractures, concussion, and facial injuries may also occur.
- Injuries occur from contact with the floor, net, other players, or the ball.
  - A hard hit ball can reach 80 miles per hour.
  - Pressure in the beach ball is only half of that in an indoor ball, so players are at less risk for some injuries.
- Acute injuries occur more in recreational players than elite.
- Most acute injuries occur near the net.

Overuse Injuries

- More common than acute traumatic injuries.
- Patellar and rotator cuff tendinitis are the most common (see Fig. 58-1).
- Lack of strength and endurance predispose the athlete to overuse injuries.
- Overuse injuries occur more often in elite players because they spend more time doing drills than recreational players.

Injury Pattern in Indoor versus Beach Volleyball

- Indoor volleyball (Fig. 58-2)
  - Most injuries occur during blocking and hitting, which frequently results in acute finger and ankle injuries.
- Beach volleyball (see Fig. 58-2)
  - Most injuries occur in field defense and in hitting.
  - More shoulder injuries than in indoor volleyball; player serves and hits more frequently in beach volleyball because there are fewer players.
  - Fewer ankle injuries than in indoor volleyball; fewer players means there is less chance of coming down on someone's foot.

Table 58-1 INDOOR VOLLEYBALL

Position	Location on court	Function	Injuries
Setter	Front court	Set ball to hitter	Wrist tendinitis Finger injuries
Hitter (Spiker)	Front court	Spike ball into opposing court	Ankle sprains Shoulder instability/impingement Spondylolysis Patellar tendinitis
Server (all players)	Back court	Serve ball	Shoulder instability/impingement
Blocker	Front court	Block or alter ball hit by opponent	Finger injuries Ankle sprains Patellar tendinitis
Passer	Back court	Receive serve Pass ball to setter May need to dive for or "dig" ball	Contusions Injuries of the upper extremities Patellofemoral syndrome Low back pain

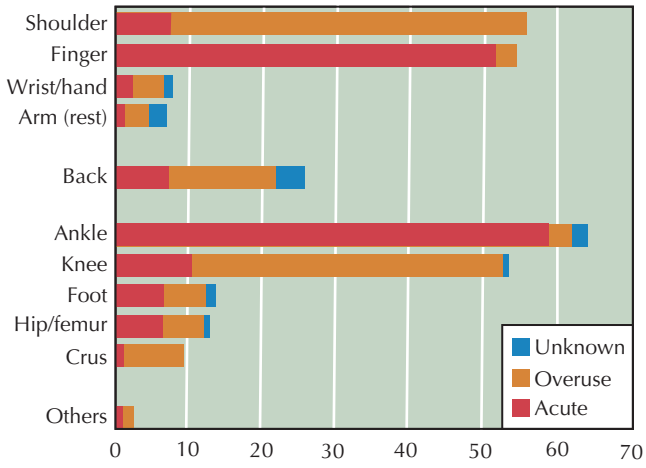


Figure 58-1 Number of Volleyball Injuries by Site and Type. *(Reprinted with permission from Aagaard H, Scavenius M, Jorgensen U: An epidemiological analysis of the injury pattern in indoor and in beach volleyball. Int J Sports Med 18:217-221, 1997.)*

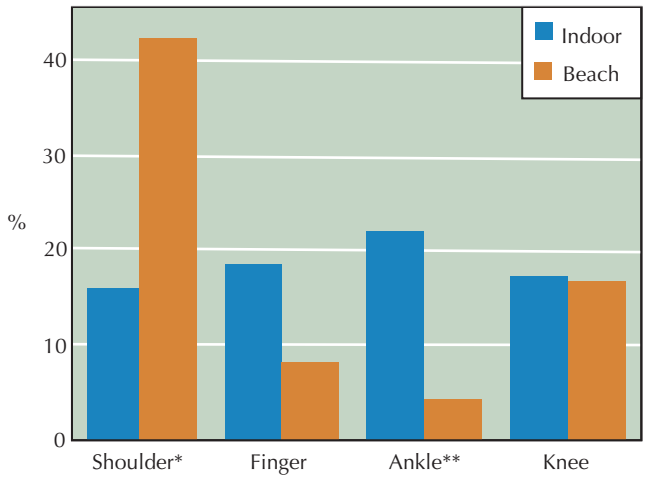


Figure 58-2 Percent of Injuries in Beach Volleyball Compared with Indoor Volleyball in the Four Main Injury Sites. *(Reprinted with permission from Aagaard H, Scavenius M, Jorgensen U: An epidemiological analysis of the injury pattern in indoor and in beach volleyball. Int J Sports Med 18:217-221, 1997.)*

- Fewer finger injuries than in indoor volleyball because (1) fewer blocks are performed in beach volleyball and (2) the ball is softer.

Prevention of Injuries

- Shock-absorbent playing surface; wood is softer than concrete or linoleum.
- Sweat on the floor should be wiped up.
- Padded apparatus (net supports and cables and officials' stand).
- Knee pads (Fig. 58-3).
- Ankle brace or taping if history of ankle sprain.
- Enforce rules; no crossing of centerline.
- Diving and digging drills to learn proper technique.
- Preseason conditioning (see "Off-Season Conditioning").

Knee pads can offer protection when digging balls.

Figure 58-3 Prevention of injuries. Knee pads can offer protection when digging balls.

Preparticipation Physical Evaluation

- Evaluate for ligamentous laxity of the shoulder
  - Could predispose to shoulder problems.
  - Athlete may benefit from strengthening program prior to and during the season.
- Marfan syndrome
  - Tall athletes with long arms tend to gravitate toward volleyball.
  - Look for stigmata of Marfan syndrome.
  - Athletes with sufficient criteria should have ophthalmology exam and echocardiogram.

INJURIES AND MEDICAL PROBLEMS

Foot and Ankle Injuries

Ankle Sprain

- Most common acute volleyball injury, accounting for up to half of all acute injuries.
- Previous sprain is an important risk factor.
- Most often occurs when a player performing a hit or block comes down on the foot of an opponent or teammate in the front court.
- Recurrence can be reduced by:
  - Not allowing return to play until the sprain has been fully rehabilitated.
  - Taping or bracing the ankle.
  - Avoiding coming down from a jump near the centerline where another player's foot may be.

Metatarsal Stress Fractures

Caused by repetitive high-impact activity, such as jumping, to which the athlete is not accustomed.

Plantar Fasciitis

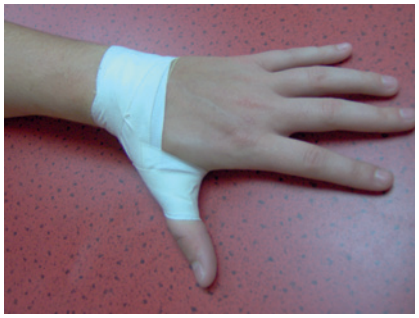
Caused by repetitive pushing off and jumping.

Wrist and Hand Injuries

Finger and Thumb Sprains, Fractures, and Dislocations

- **Mechanisms of injury:**
  - When blocking, fingers are spread apart and vulnerable to injury from the ball (Fig. 58-4).
  - If the fingers are below the ball while the player is attempting to block, they are prone to injury because of the downward trajectory of the ball onto the fingertips.
  - The fingers may also hit the net or another player during the follow-through phase of the swing.

Blockers are prone to finger injuries from the ball.



Thumb spica taping.

Figure 58-4 Finger and Thumb Sprains.

- Sprain of the collateral ligament of the thumb metacarpophalangeal joint is a common injury in volleyball.
  - Partial tears may be stabilized with thumb spica taping, allowing earlier resumption of play (see Fig. 58-4).
- Injuries to the thumb, index, and middle fingers are especially bothersome for overhead setters.
- Minor finger injuries can often be treated with short-term splinting followed by buddy taping and early return to play.
- Proper blocking and hitting techniques may decrease the frequency of finger and thumb injuries.

Wrist Fractures

Occur when the player falls on outstretched hand when losing balance coming down from a jump or tripping.

Wrist Tendonitis

Affects overhead setters, who frequently use forearm and hand muscles.

De Quervain's Tenosynovitis

- Occurs in passers because of repeated microtrauma from the ball impacting the radial aspect of the wrists.
- **Treatment** includes improving technique so the ball instead impacts more on the proximal and volar aspects of the forearms when under hand passing.

Neuropathy of the Superficial Branch of the Radial Nerve

- From repeated ball impact to the dorsoradial aspect of the distal forearms.
- Decreased sensation and paresthesias over the dorsoradial hand.
- **Treatment** includes adjustment of location of ball impact to the forearms.

Shoulder Injuries

Shoulder Instability

- Tends to occur in younger players.
- Ligamentous laxity may be congenital or acquired (from repeated hitting/serving).
- The (micro)instability of the glenohumeral joint is in an anterior direction.
- Occurs during the early acceleration phase of hitting and serving (Fig. 58-5).
- Jump serving places higher loads on the shoulder than the traditional overhead serve does.
- Rotator cuff impingement and tendinitis may occur secondarily.
- Internal impingement may also occur during anterior translation of the humeral head when the shoulder is in the cocked position.

Extreme external rotation and extension during hitting can cause shoulder problems due to anterior instability and overuse of the rotator cuff.

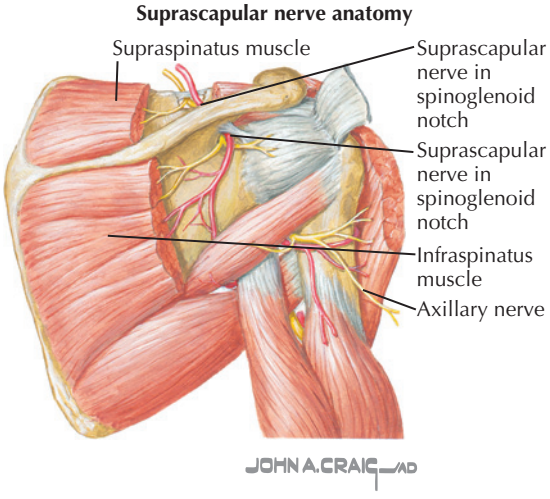


Figure 58-5 Shoulder Injuries.

Shoulder Impingement (Primary)

- Tends to occur in the older athlete.
- Often concomitant with rotator cuff muscle weakness.
- Tendinitis and bursitis tend to accompany impingement.
- Occasionally there is degeneration and tears of the rotator cuff tendons.
- Repeated hitting and serving precipitate or aggravate the condition.
- Repetitive overhead hitting strengthens the internal rotators over the external rotators, disturbing the balance of the rotator cuff muscles, which may lead to shoulder symptoms.

Suprascapular Neuropathy

- Nerve irritation in suprascapular neuropathy usually occurs at the suprascapular notch, but in volleyball players it occurs more distally at the spinoglenoid notch (see Fig. 58-5).
- Mechanism of injury is thought to be traction on the suprascapular nerve causing it to impinge at the base of the scapular spine.
  - Can occur during the follow-through of hitting and serving when the shoulder is in extreme adduction and internal rotation.
  - Traction can also occur during floating serves:
    - Arm is retracted immediately after the hitting the ball, causing the ball to "float" and be difficult to pass.
    - Movement requires forceful eccentric contraction of the infraspinatus muscle, creating traction on the nerve.
- Found in the dominant upper extremity in a fairly high percentage of volleyball players (12% to 33%, depending on the study).
- Usually asymptomatic, but occasionally may cause posterior shoulder pain. Proposed reasons for why there are usually no symptoms:
  - Nerve lesion is usually incomplete so some function of the muscle remains.
  - Teres minor muscle compensates.
  - Infraspinatus muscle is not used up to its full capacity during hitting.
- Entrapment of the nerve at the spinoglenoid notch may lead to isolated atrophy of the infraspinatus muscle and some loss of external rotation power, but again, this usually has no significant effect on the athlete's performance.
- Some athletes may have point tenderness over the spinoglenoid notch.
- Electromyography (EMG) will usually confirm the diagnosis but is not necessary in most cases.
- **Differential diagnosis** of infraspinatus atrophy:
  - Infraspinatus tendon tear
  - Brachial neuritis affecting the suprascapular nerve
- **Treatment (if symptomatic)**
  - **Nonoperative treatment**
    - Restricting overhead hitting.
    - Nonsteroidal antiinflammatory medications.
    - Physical therapy to strengthen the rotator cuff and periscapular muscles, improve posture, and stretch posterior capsule if tight.
    - Repeat EMG at 6 months, if done initially, to assess recovery.
    - If patient not improving or has significant initial disability, consider obtaining an MRI to look for possible ganglion emanating from a posterior labral tear that could be compressing the nerve.
  - **Surgical treatment**
    - Possible indications include significant pain and decreased performance despite 6 months of the above treatment, or a space-occupying lesion, such as a ganglion, compressing the nerve.
    - Is rarely needed.
    - Surgical procedure involves exploring the nerve and releasing areas of compression.
- **Prognosis/return to play:**
  - Most symptomatic cases respond to nonoperative treatment, but may take up to a year for maximal recovery. An exception is a space-occupying lesion compressing the nerve, which often will not resolve without surgery.
  - Whereas pain usually improves with operative or nonoperative treatment, muscle atrophy may not resolve. Most athletes are able to return to full athletic function despite the persistent atrophy.

Lumbosacral Spine Injuries

Mechanical Low Back Pain (Strain/Sprain)

- Passers are susceptible because of repetitive lumbar flexion. The upper extremities held in front of the body act as a long lever arm, placing additional strain on the low back (Fig. 58-6).
- Jumping positions are also susceptible because repeated landing places high forces on the spine.

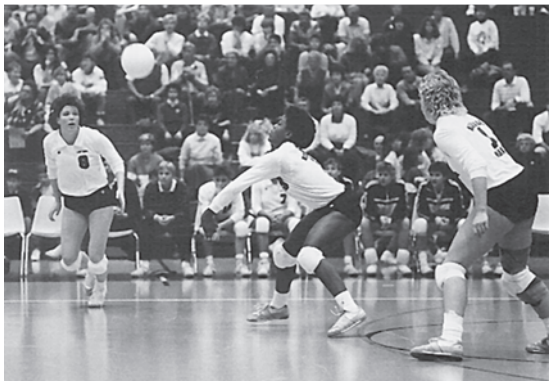
Spondylolysis

- A stress fracture at the pars interarticularis of the vertebra, most commonly the fifth lumbar vertebra.
- Occurs with repetitive hyperextension of the low back, most commonly associated with hitting but also with overhead setting and blocking.
- Suspect if persistent extension low back pain in young athletes.
- Facet joint sprain should also be considered in the differential diagnosis of extension low back pain.

Knee Injuries

Patellar Tendinitis (Jumper's Knee)

- Most common overuse injury of the knee.
- Caused by repetitive eccentric quadriceps contraction with jumping (blockers, hitters).
- **Risk factors:**
  - Higher frequency of playing (more than four times per week)
  - Higher intensity of play



Repetitive flexion of the lumbosacral spine in under hand passing can lead to low back pain.

Figure 58-6 Lumbosacral Spine Injuries.

- Playing on a hard surface rather than shock-absorbing surface
- Having higher vertical leap

- **Differential diagnosis** for anterior knee pain:
  - Patellofemoral syndrome (see following section)
  - Osgood-Schlatter disease (in early teens)

Patellofemoral Syndrome

- Common cause of knee pain in all sports.
- Especially common in volleyball positions that require jumping and squatting (hitters, blockers, passers).

Anterior Cruciate Ligament (ACL) Tear

- May be from a rotational force to the knee when coming down awkwardly.
- Occurs more frequently in female volleyball players than male.
- Reconstruction is usually required if return to a sport like volleyball is desired.

MEDIAL COLLATERAL LIGAMENT TEAR

From valgus force to the knee as a result of landing off balance.

Leg Injuries

Tibial Stress Fracture

- May be found in positions requiring frequent jumping.
- May occur along the medial or anterior edge of the tibia.

Gastrocnemius Muscle Strain

- Usually occurs at the musculotendinous junction of the medial belly.
- Associated with an eccentric load to the muscle tendon unit.

Achilles Tendon Problems

- Acute achilles rupture
  - Caused by rapid, forceful eccentric loading of the tendon when landing from a jump or sudden acceleration.
  - Most common in the 4th and 5th decades.
- Achilles tendinitis
  - Inflammation of the paratenon.
  - Caused by repetitive eccentric loads from jumping.
- Achilles tendinosis
  - From chronic overuse.
  - Usually in older players.
  - Degeneration and microtearing of the achilles tendon at its insertion to calcaneus.

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Soccer

Margot Putukian

RULES OF THE GAME

- Game lasts 90 minutes; divided into two 45-minute halves.
- Eleven players on the field (generally 10 field players and 1 goalkeeper).
- Ball cannot be played by hands (except goalkeeper), but all other surfaces of body can be used.
- Goal scored by hitting ball into opposing goal; team with most goals win.
- Rules protect against rough play (either direct or indirect fouls).
  - Direct fouls include kicking opponent, tackling from behind, using hands, pushing.
  - Indirect fouls include offside, obstruction, some unintentional fouls. Offside rule: must always have two players between offensive player and the goal. This often includes goalkeeper.
  - Many rules are enforced at discretion of referee.
- Most leagues require shin guards; no other equipment requirements.
- Ball put back in play when it goes out of touch (out of bounds).
  - Throw in: when ball goes out over sideline.
  - Goal kick: when ball goes out over endline last touched by offensive player. Kicked in from the goal box by defensive team.
  - Corner kick: when ball goes out over endline last touched by defensive player. Kicked in from corner of field by offensive team.

SPORT-SPECIFIC DEMANDS AND SKILLS

- Physical demands: discontinuous high-output activity requiring endurance base along with intermittent sprinting (aerobic and anaerobic).
- Energy expenditure requires approximately 75% $\dot{V}O_2$ max; differs with position (midfielders expend more energy than other positions). Average heart rate: 165 beats per minute. Players cover 8 to 12 km during game.
- Activity further divided into sprinting, cruising, running, walking.
- Ratio of time spent in low-intensity versus high-intensity efforts is 7:1.
- Sprints occur once every 90 seconds, high-intensity efforts (cruise and sprint) once every 30 seconds; only 2% of total distance involves time spent with ball.
- **Physiologic demands** of sport have implications for thermoregulation and nutritional needs. Energy needs increased by sport-specific skills.
- **Strength demands:** high demand on trunk and lower extremities.
- Most skills require balancing on 1 leg; emphasizes lower extremity strength, proprioception.
- Certain skills make soccer unique. Various surfaces of lower extremity are used to strike or control ball and tackle; thus various structures are at risk.
  - **Inside of foot pass/block tackle:** Foot and hip externally rotated, knee flexed, foot "locked" in dorsiflexion (Fig. 59-1). Results in significant valgus stress at knee and may be associated with traumatic injury (sprain of medial collateral ligament, fracture) or muscle strain.
  - **Outside of foot pass/shot:** Internal rotation of leg, foot in inversion, plantarflexion; may place athlete at risk for forceful inversion plantarflexion injuries, mid/forefoot injuries, ligament sprains, peroneal tendon problems (see Fig. 59-1). In young athlete, may lead to apophysitis or avulsion fracture.
  - **Instep of foot:** Extreme plantarflexion in "locked" foot, hip flexors, quadriceps, rectus, hamstrings (see Fig. 59-1). Ball velocity at release is between 17 and 28 meters per second.
  - With approach and ball strike phase of instep kick, varus torque of more than 200 Newton meters (Nm) is generated on proximal tibia; 280 Nm of extension torque is produced on proximal tibia during approach and ball strike.
  - Total of 2000 Nm generated during soccer kick. Only 15% transferred to ball; remainder absorbed by eccentric contraction of hamstrings.
  - Kicking ball on ground can injure forefoot, ankle, toes (especially great toe).
  - Can see both anterior and posterior impingement syndromes, various overuse injuries.
  - **Heading:** Purposeful, forceful striking of ball with head to control, clear, or redirect on goal. Risk for neck and head injuries because of player contact (head to head or other body part, or head to goal post or ground) that occurs while fighting for possession in air (see Fig. 59-1). No evidence that purposeful heading of the ball causes injury.
  - **Proper technique important in all skills to avoid or decrease injuries.**

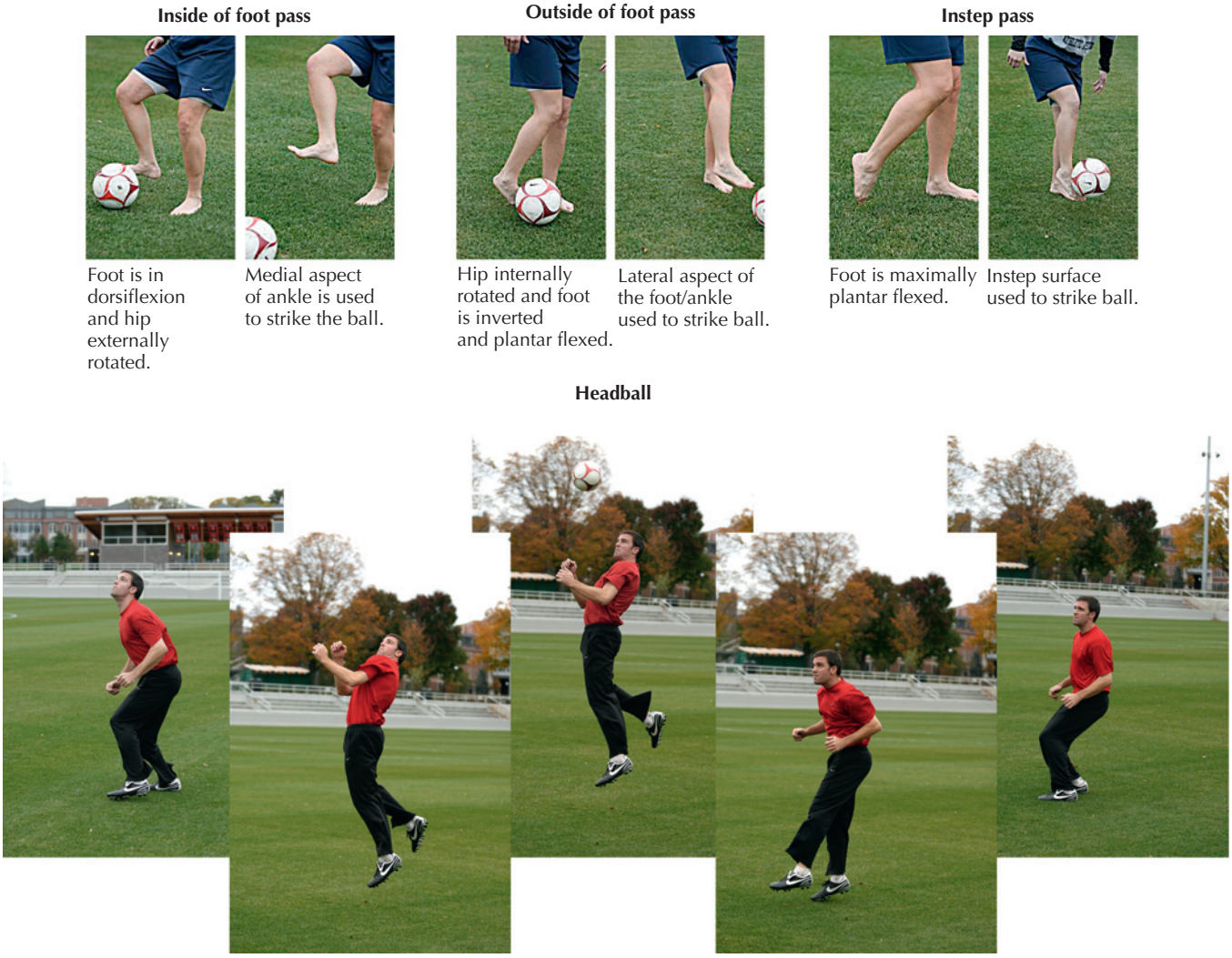
ENVIRONMENTAL AND NUTRITIONAL ISSUES

Fatigue

- Linked to many variables. Physiologic variables controllable: aerobic fitness, thermoregulation, depletion of carbohydrate reserves.
- Distance covered in the second half is 5% less than distance covered in the first half.
- Fatigue most pronounced in center backs and strikers, less so in midfielders and fullbacks, who tend to have higher $\dot{V}O_2$ max values.
- With 90 to 180 minutes of exercise at 60% to 80% $\dot{V}O_2$ max or 15 to 30 minutes of exercise at 90% to 130% $\dot{V}O_2$ max, performed in intervals of 1 to 5 minutes, muscle glycogen is depleted.
- Positive correlation between $\dot{V}O_2$ max and work rate.
- Depletion of carbohydrate stores has more effect on physiologic variables (intensity and duration of running), less effect on sport-specific skills.

Dehydration

- A concern, especially during play in warm environment.
- When athlete is 2% dehydrated, some studies demonstrate decrease in performance.
- Thirst experienced only after 3% dehydration; left on their own, athletes generally do not drink enough to replace losses.
- Soccer players over 2-week training period were allowed to drink on own for 1 week (on average, they drank 2.7 L per day), then were required to drink 4.9 L per day. **Voluntary intake resulted in deficit of 1.1 L; not enough to meet needs.**
- Once athlete is 5% dehydrated, work capacity can diminish by 30%.
- Heat illness and exhaustion are most serious side effects of dehydration; result from inability to dissipate heat.
- Acclimatization to heat can improve thermoregulation, maintain work rate.



Jumping header. These photographs show the series of technique involved in the jumping header. The ball is struck when the player is at the maximal height of the jump, with the player's eyes open. Impact with the ball is made at the forehead/hairline with the head striking forward through the ball. The arms are held up for balance and to create space for the player and thus protect him from inadvertent contact with another player.

Figure 59-1 Soccer-Specific Demands and Skills. *(Photographs © 2008 Beverly Schaefer)*

Prevention of Heat Injury

- Modifications can be made that decrease risk of heat injury; more water breaks, change game times, shorten length of play.
- Thermoregulation potentially improved with hyperhydration.
- Several factors increase risk for development of heat illness (preexisting medical disease, age, poor conditioning, inadequate acclimatization, dehydration, obesity, fatigue, prior heat injury, febrile illness, medications).
- **Level of muscle glycogen stores** before exercise important.
- Comparison of soccer players with high versus low prematch muscle glycogen content:
  - Low glycogen group covered 24% less total distance (most at high speeds).
  - Players with low pregame values had no glycogen stores at end of game compared with 10% of glycogen stores in players with high pregame values.
- Effects of high-carbohydrate (65% of calories from carbohydrate) and low-carbohydrate (35% of calories from carbohydrate) diet 48 hours before match on technical and physiologic variables were assessed in one study.
  - Technical variables included number of successful/unsuccessful "green" passes (low-risk, nonpenetrating) and "red" (high-risk, penetrating) passes, and total number of touches per possession (greater than or less than three).
  - After high-carbohydrate diet, higher pregame muscle glycogen content, 33% more high-intensity work, no difference in any technical variables.
- Prospective study examined effects of carbohydrate fluid ingestion in soccer players.
  - Muscle biopsy performed before and after game in players with glucose polymer (GP) ingested before and during soccer match.
  - Muscle glycogen content was 31% higher in players given carbohydrates.
- Effects of GP solution (750 mL Exceed) before playing match and at half-time.

  - Amount of time each player spent walking, jogging, sprinting, or cruising measured by video analysis in both halves of game.
  - Players spent same amount of time walking and jogging in first and second halves and same amount of time in cruising or sprinting in first half, but players given GP spent 40% more time sprinting and cruising in second half and covered 20% more distance overall compared with placebo controls.
- Effects of GP ingestion (300 mL, 25% glucose) between two successive 50-minute indoor matches: Players given GP ran further and faster in second game than players given placebo.
- Studies looking at effect of carbohydrate either in diet or in liquid form on skill proficiency (tackling, heading, dribbling, shooting skills) have shown no effect.
- Sport-specific and other studies make clear that **proper fluid and carbohydrate ingestion not only prevents dehydration and thermoregulatory complications but also may play role in enhancing performance by sparing muscle glycogen stores and increasing work output and time to exhaustion.** Ingesting carbohydrate as soon after exercise as possible maximizes replacement of muscle glycogen stores in preparation for next exercise.

GENERAL INJURY PATTERNS

Overall Injury Incidence

- Overall injury incidence in soccer is favorable (much lower than for American football).
- In players younger than 10 years, injury incidence is extremely low.
- Injuries predominantly minor and most often involve lower extremity (most common: muscle contusions, ankle sprains, abrasions).
- Injuries increase as skill and age increase; at high school level, injuries increase 10-fold.
- At younger age levels, girls have higher incidence of injury than boys (Table 59-1). At collegiate level, gender difference less striking (Table 59-2). Gender differences difficult to study; trends exist for certain injuries (knee, anterior cruciate ligament [ACL]).
- Injury pattern similar for indoor and outdoor soccer.
- Goalpost injuries can be fatal; should be preventable.
  - In the United States (1980-1994), of 27 injuries caused by falling goalpost, 18 were fatal (14 involved head trauma); 26 of the involved goalposts were made of galvanized pipe; 18 of the 27 were not anchored; average age of goalpost was 10 years.
  - In Denmark in 1999, there were 2 deaths and 117 injuries related to goalposts.

Injury Surveillance System

- **National Collegiate Athletic Association** (NCAA) uses Injury Surveillance System (ISS); injury is defined as time loss (Table 59-3). Recent review of all data from 1988-1989 through 2002-2003 seasons.
- Disadvantages of ISS: Injuries without time loss are missed, same injury may or may not be "time loss injury" (i.e., no time loss if in season, but time loss if off season).
- Despite these limitations, ISS data provide denominator (number of athlete exposures [AE]) and thus useful basis for comparing sports, genders, injury types.
- 1988-1989 through 2002-2003 NCAA data: Women's soccer game and practice injury rates of 16.44 and 5.23 per 1000 AE, respectively. Same time period in men's game with game and practice injury rate of 18.75 and 4.34 per 1000 AE, respectively. Both men and women have higher injury rate in preseason compared with regular season.
- Injuries that occur are generally mild; 78% of game injuries and 83% of practice injuries result in time loss of less than 10 days for women, and 81.3% of game and 85.4% of practice injuries result in time loss of less than 10 days for men.
- Knee internal derangements, ankle sprains, and concussions, in that order, account for the majority of severe injuries that occur in both men's and women's soccer.
- Injury rates during games are threefold to fourfold greater than during practices.
- Lower extremity accounts for approximately 75% of all injuries; knee, ankle, and head most common sites injured.

Table 59-1 INJURIES IN SOCCER PER 1000 PLAYER HOURS: BOYS VERSUS GIRLS

Study	Girls	Boys
Engstrom et al., 1991*	12	5
Nilsson and Roaas, 1978	32	14
Maehlum et al., 1986	17.6	8.9
Schmidt-Olsen et al., 1991	17.6	7.4
Powell 1999 (injuries per 1000 AE)	5.3	4.6

*Denotes studies using time loss from practice/play as definition of injury.

Table 59-2 NCAA DATA FOR FALL OF 1999

	Head Injury		*Concussion*	
	%	Injury rate	%	Injury rate
Women's soccer				
Practice	3.0	0.15	2.6	0.13
Game	11.7	2.13	10.8	1.95
Total	7.3	0.57	6.6	0.52
Men's soccer				
Practice	0.9	0.04	0.7	0.03
Game	9.5	1.95	6.7	1.37
Total	5.6	0.41	3.9	0.29

Adapted from Dick R: NCAA Data, 1988-1989 through 2002-2004. Indianapolis, Ind: National Collegiate Athletic Association, 2007.

Table 59-3 INJURY DATA FROM NCAA INJURY SURVEILLANCE SYSTEM 1988-1989 THROUGH 2002-2003

Sport	Practice injury rate per 1000 athletic exposures	Game injury rate per 1000 athletic exposures
Football (fall)	3.8	35.9
Wrestling	5.7	26.4
Women's gymnastics	6.12	15.2
Spring football	9.6	
Men's soccer	4.3	18.8
Women's soccer	5.2	116.4
Men's ice hockey	2.0	16.3
Women's ice hockey	2.5	12.6
Men's lacrosse	3.2	12.6
Men's basketball	4.3	9.9
Women's basketball	4.0	7.7
Field hockey	3.7	7.9
Women's lacrosse	3.3	7.2
Baseball	1.9	5.8
Women's volleyball	4.1	4.6
Women's softball	2.7	4.3

Adapted from Dick R: NCAA Data, 1988-1989 through 2002-2004. Indianapolis, Ind: National Collegiate Athletic Association, 2007.

- ACL injury rates for women and men from 1988-1989 through 2002-2003 were 0.28 and 0.28 per 1000 AE, respectively. **These data confirm increased rate of knee and, specifically, ACL injuries in women.**
- Concussion rate for women and men from 1988-1989 through 2002-2003 were 0.41 and 0.28 per 1000 AE, respectively. **These data raise concern related to the increased incidence of concussion in female players compared to their male counterparts.** The reason for this difference is unclear. Potential explanations include differences in reporting between genders, anatomical differences, hormonal differences (estrogen potentially protective), or genetic differences; more research is needed.

Indoor Soccer

- Injury data sparse; only two studies give denominator and include women.
  - In a 1994 study of indoor soccer leagues, injury incidence was 5.04 per 100 player hours for men and 5.03 for women. Injury was defined as "anything for which player requested medical attention, or play was stopped." Increased incidence of ACL injuries was found in women compared with men.
  - Lake Placid Dawn to Dark Festival in 1996 (3-day indoor tournament): injury incidence for men was 5.79 per 100 player hours; for women, 4.74. Injury was defined as time loss, with mild, moderate, and severe injuries corresponding to less than 1 week time loss, more than 1 week but less than 1 month time loss, and more than 1 month time loss. Of injuries, 65.8% were mild; 15.8%, moderate; and 18.4%, severe. No significant difference in incidence of injury between men and women; no difference in knee injuries. Severe injuries were more likely to be noncontact; 71.4% injuries were in lower extremity.
- Patterns may differ slightly, but, in general, type, location, and severity of injury appear similar in outdoor and indoor soccer.
- Natural grass compared with artificial surface: although 1996-1997 data show higher injury rate on turf versus grass, overall data show no difference.
- Recent data (2007) evaluating injury incidence, severity of injury, or mechanisms of injury for game injuries on grass versus new generation artificial turf by male and female players demonstrate no major differences for these surfaces.

Prevention of Injuries

- Early prevention program by Caraffa: Taping of injured/lax ankles, exclusion of lax knees, warm-up and flexibility program, no shooting before warm-up, shin guards, close supervision, education. Implementation resulted in 75% reduction in foul plays and injuries.
- More recent research specifically targeted toward ACL injuries in women incorporates neuromuscular (NM) training.
  - Hewett: 60- to 90-minute training sessions 3 times per week for 6 weeks in soccer, volleyball, and basketball resulted in 72% decrease in noncontact ACL injuries.
  - Mandelbaum: Prevent Injury and Enhance Performance Program emphasizing proprioception and NM training; 20 minutes of soccer-specific agility drills, plyometrics, lower extremity and trunk stretching, and strengthening exercises showed 74% reduction in ACL tears over 2 years (see "Recommended Readings").

SPECIFIC INJURY PATTERNS

Knee Injuries (see Table 59-4)

Table 59-4 KNEE STRUCTURES INJURED IN SOCCER (1989-1993)

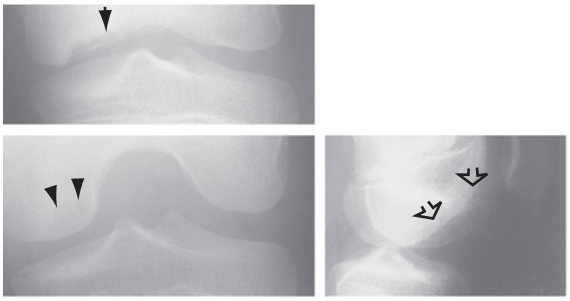
	Men's soccer		*Women's soccer*		
	No.	**Rate***	**No.**	**Rate***	**P (Variance)**
Collateral ligament	316	0.51	192	0.62	0.02
Torn meniscus	119	0.19	105	0.34	0.00
Patella/patellar tendon	130	0.21	92	0.30	0.01
Anterior cruciate ligament	81	0.13	97	0.31	0.00
Posterior cruciate ligament	22	0.04	12	0.04	
Athletic exposures	626,223		308,748		

*Per 100,000.
Data from Arendt AE: Sports Med Arthrosc Rev 5(2), 1997.

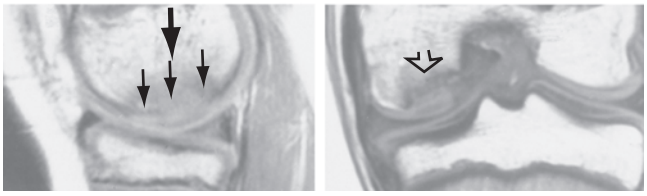
- **Overall injury patterns**
  - One needs only to watch soccer players pivot and cut back and forth, change direction while decelerating, take cross with half-volley, or cross ball from one side of field to another to understand why they may be at increased risk for knee pathology.
  - Knee injuries account for 8% to 18% of injuries in practices and games (NCAA, 1988-1989 through 2002-2003).
- **Meniscal injuries** (same sport-specific skills that put player at risk for ACL injury)
  - In setting of ACL deficiency, impact of meniscal injuries dramatic.
  - Coexistence of meniscal and ligamentous injuries to knee should not be underestimated in soccer players; isolated injury may not prohibit activity, whereas additional injuries make it difficult to return to high-level play.
- **Medial collateral ligament (MCL) injuries**
  - Inside of foot pass/redirection and block tackles put athlete at risk and also make early return difficult because of direct valgus force.
  - Risk increases with poor technique, tentative athlete, and leg extended away from body.
- **Osteochondral injuries**
  - Can occur and can be difficult to treat in soccer player.
  - Present as pain and/or swelling with activity.
  - Diagnosis made by plain radiographs, though sometimes need additional testing such as magnetic resonance imaging (MRI) (Fig. 59-2).
  - Treatment depends on clinical picture as well as diagnostic tests. If significant lesion and/or separation of fragment, arthroscopic treatment with microfracture often indicated.

Muscle Contusions

- Common in soccer; most common in pediatric age group.
- Accounted for 35.2% of injuries in one summer training camp.
- Rate of injury increases with age; boys at greater risk than girls.
- Shin guards significantly decrease risk of contusions (and fractures) to lower leg.
- Treatment: rest, ice, compression, and elevation (RICE); avoidance of aggressive passive stretching in acutely injured muscle.
- For large contusions of quadriceps, flex knee 120 degrees; use compressive wrapping in this position for 24 hours (stops bleeding, avoids more significant injury).



A. Radiographs of a 12-year-old soccer player with symptomatic medial femoral osteochondritis dissecans.



B. Magnetic resonance imaging shows loss of articular surface continuity *(small arrows)*. *Arrows* indicate the osteochondritis dissecans fragment subchondral junction.

Figure 59-2 Knee Injuries. *(Reprinted with permission DeLee J, Drez D, Miller M: DeLee and Drez's Orthopaedic Sports Medicine, 2nd ed. Philadelphia: Saunders, 2002.)*

- Reinjury is major factor in developing **myositis ossificans.**
  - Defined as new bone formation in muscle after injury; also called heterotopic (ectopic) ossification.
  - Occurs in 9% to 20% all quadriceps contusions; average time of disability is 73 days.

Muscle Strains

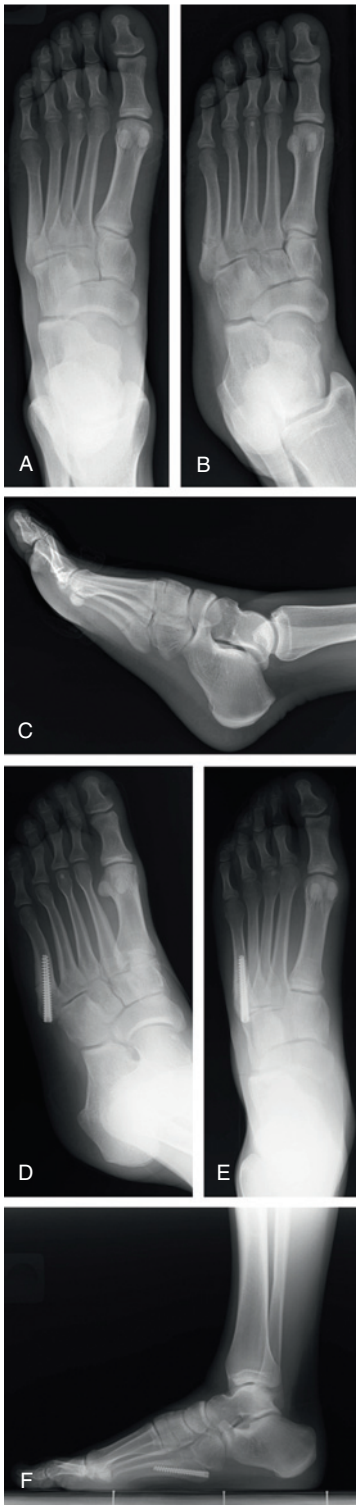
- Unusual in younger population (youth player at greater risk for apophyseal and epiphyseal injuries).
- Common, frustrating injuries in older adolescents/college athletes.
- Eccentric contraction plays key role in production of muscle strain.
- Muscles with increased content of type II fibers and muscles that cross two joints are more susceptible.
  - Rectus femoris crosses two joints, has high percentage of type II fibers, and contracts eccentrically in decelerating hip and knee.
  - Hamstring muscles contract eccentrically with running and deceleration, and have high content of type II fibers.
  - Adductor longus, gastrocnemius also commonly involved.
- Muscle tendon unit has propensity to fail under various conditions.
  - Strains result from excessive stretch or stretch while muscle is activated.
  - Muscle tends to tear near muscle-tendon junction.
- Fatigue increases susceptibility to muscle strain.
  - Amount of energy absorbed by fatigued muscle is 69.7% to 92% that in control leg.
  - Lowest energy absorption occurs in muscles that are most fatigued.
  - Fatigued muscles absorb less energy before reaching degree of stretch that causes injuries.
- Established continuum of injury during passive muscle stretch.
  - Rabbit skeletal muscle stretched at 10 cm per second to 60%, 70%, 80%, and 90% of force required to cause passive failure of muscle.
  - Continuum of diminished maximal isometric contractile force, muscle fiber disruption, edema, hemorrhage, and decreased electromyography (EMG) maximal voltage amp.
  - In previously injured muscle, failure occurs in muscle itself; hence need to protect injured muscle from further injury before healing is complete.
- **Prevention**
  - Adult players with decreased flexibility had higher incidence (not significant) of muscle strain. Dominant leg more often involved.
  - Emphasize beneficial effects of warm-up, temperature, and stretching on mechanical properties of muscle.
  - Many factors protect muscle: strength, endurance, flexibility.
  - Eccentric hamstring strengthening ("Russian Hamstring Curls") useful in preventing hamstring injuries.

Stress Fractures

- Represent overuse injury to the bone; generally caused by training regimen.
- Bone scans indicate that most overuse injuries and stress fractures occur in lower extremity: tibia (49.1%), tarsals (25.3%), femur (7.2%), metatarsals (8.8%), fibula (6.6%) pelvis (1.6%), sesamoids (0.9%), and spine (0.6%).
- Fifth metatarsal stress fractures, specifically Jones type (metaphyseal-diaphyseal junction) have unpredictable healing and thus in high-demand athlete screw placement for internal fixation should be considered (Fig. 59-3).
- Training errors accounted for 22.4% of fractures.
- Other factors: playing surface, cleat and indoor shoe requirements, changes in training regimen/intensity, and (in female athletes) hormonal and/or nutritional influences.
- Address biomechanical issues (leg length discrepancies, femoral anteversion, foot pronation, increased knee valgus) in preparticipation physical exam.
- Consider female athlete triad (disordered eating, menstrual dysfunction, and decreased bone mineral density).
  - Although more common in sports emphasizing leanness (cross-country running, swimming) and sports with subjective scoring (gymnastics, skating), triad occurs in all sports.
  - Menstrual dysfunction associated with low bone mineral density (BMD) at axial as well as appendicular sites.
  - Eating disorders also common in female athletes (same effect on BMD); several triggers may be involved.
  - Early recognition of eating disorders and menstrual dysfunction is essential.
  - Optimize prevention and early treatment through education.

Groin Injuries

- Account for 5% of all injuries in soccer; return to play difficult.
- Direct trauma can result in injury to external genitalia, muscles, vessels, or joints.
- Other injuries include osteitis pubis (symphysis pubis instability), adductor, rectus or iliopsoas muscle strain, stress and avulsion fractures, hernia, Gilmore's groin.
  - Osteitis pubis: low-grade inflammation at symphysis, positive bone scan.
  - Pain at adductor insertion (vs. adduction) for more than 2 months; pain at morning or night, with cough/sneeze, at symphysis; positive bone scan and x-ray.
    - Treated with either standard physical therapy (stretching, friction massage, modalities) or active training (strengthening, proprioceptive neuromuscular facilitation, balance).
    - 79% (vs. 14%) of those treated with active training (vs. 14% treated with standard physical therapy) returned to play.



A-C. Type IA (DeLee) fifth metatarsal fracture in collegiate soccer player. **D-F.** After fixation with variable pitch compression screw.

Figure 59-3 Stress Fractures. *(Reprinted with permission Canale T, Beaty J: Cambell's Operative Orthopaedics, 11th ed. Philadelphia: Mosby, 2007.)*

- **Sportsman's hernia:** musculotendinous disruption with torn external oblique aponeurosis, torn conjoined tendon, conjoin tendon torn from pubic tubercle, dehiscence between conjoined tendon and inguinal ligament, no hernia.
  - 90% male, 70% insidious onset, 30% report injury.
  - Affects soccer, rugby, track, hockey athletes; muscular imbalance common.
  - Strong hip flexors tilt pelvis forward, stretch, and put at risk abdominal muscles, particularly obliques.
  - Pain usually unilateral (88%); 40% in adductor region, 6% in perineum.
  - Pain worse with activity, especially sudden movement, twisting, striding, sprinting, long or dead ball kicks, sidestepping, coughing/sneezing, sit-ups.
  - Often considered "diagnosis of exclusion." X-rays, bone scans, and MRI often used to exclude other diagnoses. Incidence of coexistence of other injuries unclear. Newer use of ultrasound to diagnose sportsman's hernia.
  - Treatment: Initially conservative rehabilitation with strengthening, relative rest, NSAIDs. If no improvement and workup otherwise negative, surgical treatment indicated.
  - Surgery: In 8-year study, 97% successful; return to play within 6 weeks.
  - Laparoscopic surgery may be more beneficial than conventional surgery because of earlier return to play (median 3 weeks vs. 5 weeks, respectively).

Ankle Injuries and Impingement Syndromes

- Ankle sprains most common injury in soccer; important to detect and treat properly.
- Usual mechanism is inversion plantarflexion injury; other mechanisms in soccer: outside of foot pass/shot.
- Because soccer players rely on mobility and "touch" on ball, challenge often lies in returning athletes to 100% functional activity.
- If significant ankle laxity detected during preparticipation exam, strengthening and proprioceptive program should be initiated; consider prophylactic taping or bracing.
- Athletes with ankle sprains who experience pain and disability after injury develop "ankle impingement" syndromes; difficult problem in soccer players.
  - Consider in patients with anterolateral pain, no instability, history of prior sprains.
  - Soccer players complain of pain while trying to pivot or push off one foot or with instep shooting.
  - Cause: hypertrophic scar formation, synovium, and fibrocartilage in anterolateral tibular-talar space from anterior capsule into lateral gutter.
  - Sometimes described as "meniscoid" lesions in soccer players.

Heading and Concussions

- Heading is soccer-specific skill in which head is used to control ball, clear it from defensive area, or strike it at goal.
- In 300 games, typical European player receives 2000 blows to head.
- Regulation ball weighs 396 to 453 gm, with circumference of 68 to 71 cm, and is inflated to pressure of 1 kg/cm^2, achieving speeds of 26.82 to 53.64 meters per second during match.
- Older leather soccer balls can become waterlogged, which increases their weight by 20%; modern balls are water-resistant.
- Kicked ball achieves average speed of 114.4 km per hour from 10 meters away; average impact speed is 116 kpm (can reach 200 kpm at full force).

- Accelerometer data measuring ball speed at 15.5 meters per second (35 mph) demonstrated head acceleration forces of approximately 20 gravity, with peak forces of 1200 Nm.
- Greater than forces in boxing, in which punch generates head acceleration at 100 gravity. Forces in soccer tend to be linear, whereas they are rotational in boxing and thus associated with increased shearing forces and more damage.
- Ball in contact with head for 1/63 to 1/128 second; longer the impact, smaller the force.
- Repetitive and cumulative forces have raised concern about head and neck injury.
- Skilled players maintain neck rigidly as head impacts ball; decreases angular acceleration of head, ultimately protective.
- Applying Newton's second law (force = mass × acceleration), if neck musculature is rigid, mass of head now approximates mass of body; same force applied to greater mass yields less acceleration, thus decreasing forces acting on skull.
- May be greater risk in younger player with weak musculature, unmastered technique.

Head Injuries

- (For NCAA data, see Chapter 39, Head Injuries.) Head injuries and concussions have increased in NCAA soccer for both men and women (see Table 59-2).
- At high school level, mild traumatic brain injury occurs at rates of 1.14 and 0.92 per 100 player-seasons in girls' and boys' soccer, respectively, accounting for 4.3% and 3.9% total injuries in those sports.
- Assessment and treatment of head-injured athlete and return to play issues no different in soccer players than in other athletes (see Chapter 39).
- Neuropsychological testing techniques reliably quantify brain function by examining brain-behavior relationships (see Chapter 39).

1993 U.S. OLYMPIC SPORTS FESTIVAL STUDY

- Questionnaire study of male and female soccer players in 1993 U.S. Olympic Sports Festival determined mechanisms, frequency, sequelae of head injuries.
- Participants estimated average number of times that they headed ball during practice and game as well as frequency of heading ball collision with other players. History of concussive episodes outside soccer was noted as well as symptoms associated with heading (Colorado Guidelines grading system).
- Injury defined as event that (1) required evaluation by physician, certified athletic trainer, or dentist; (2) required removal from game/practice; (3) resulted in play stoppage; (4) resulted in sequelae; or (5) prevented participation in later games/practice.
- Weaknesses of study: self-report, retrospective, selection bias.
- What study adds to literature: highlights under-reporting of head injury and/or injuries that go undetected.
- Results of data from 100% of men and 90% of women (total of 144 athletes).
  - Men more likely than women to have sustained concussion (2.16-fold higher risk).
  - Over half of players experienced at least one headache after heading the ball, large variation in frequency and duration of headache. Most often associated with poor technique, heavy or overinflated ball, weather conditions.
  - 89% of male and 43% of female players experienced head injury during soccer (forwards reported higher rate than goalkeepers, but otherwise, no difference in history, frequency, or severity of concussion among field players).
  - 50% probability that male player would have concussion in 10 years playing versus 22% probability for female player (true rate impossible to determine given lack of denominator).
  - Grade I injuries most common.
  - If amnesia present, it lasted for median of 1.5 minutes in men, 5.75 minutes in women.
  - If confusion present, median duration for men is 5 minutes; for women, 8 minutes.
  - Sequelae reported by 5 men (6.9%), 11 women (17%). Headache most common symptom.
  - Mechanism of injury summarized in Table 59-5.

PROSPECTIVE STUDY OF TEAMS IN ACC

- Prospective study of men's and women's soccer teams in Atlantic Coast Conference (ACC) over two seasons.
- Certified athletic trainers gathered data about demographics, mechanism of injury, severity, and treatment as well as number of AE (number of players, practices, games per season).
- Concussions graded according to loss of consciousness (LOC), length of posttraumatic amnesia (PTA).
  - Grade I: PTA less than 30 minutes, no LOC
  - Grade II: LOC less than 5 minutes or PTA greater than 30 minutes
  - Grade III: LOC greater than 5 min or PTA greater than 24 hours
- Incidence of concussions: 0.96 per team per season.
- 0.6 per 1000 AE for men, 0.4 per 1000 AE for women.
- For team with 25 players and 6 AE per week, expect 1 concussion every 13.5 weeks.
- Severity of injury: 22 grade I concussions (72%), 8 grade II concussions (28%).
- Other reported injuries: orbital floor and nasal fractures, forehead hematoma, stiff neck, blurry vision, one player with multiple facial fractures.
- Mechanism of injury summarized in Table 59-6.
- Injuries caused by contact with the ball occurred when ball was kicked by opponent with full force from close range; players did not see ball coming or could not react quickly enough to get out of way.

Table 59-5 MECHANISM OF HEAD INJURY IN U.S. OLYMPIC FESTIVAL SOCCER PLAYERS

	Men	Women
Collision with another player	48	20
Collision with ground or indoor wall	10	3
Collision with goalpost	1	2
Collision with ball	15	3

Data from Barnes BC, Cooper L, Kirkendall DT, et al: Concussion history in elite male and female soccer players. Am J Sports Med 26:433-438, 1998.

Table 59-6 MECHANISM OF INJURY IN PROSPECTIVE STUDY OF ACC SOCCER TEAMS

	Absolute number	% of injuries
Collision with another player's head	8	28
Contact with soccer ball	7	24
Contact with ground	4	14
Collision with opponent's elbow	4	14
Collision with opponent's knee	1	3
Collision with opponent's foot	1	3
Collision with goalpost	1	3
Combination: opponent/ground, opponent's head/ball, opponent's head/elbow	3	10

Data from Boden BP, Kirkendall DT, Garrett WE: Concussion incidence in elite college soccer players. Am J Sports Med 26(2):238-241, 1998.

- **None of these injuries was caused by purposeful heading.**
- **Conclusions:** Concussions are more common in soccer than anticipated, and acute head injuries may have potential for long-term neuropsychologic changes.

Ongoing Debate about Heading

- Concern remains for cumulative traumatic encephalopathy or "punch drunk" syndrome described in boxing.
- **Early studies flawed** by methodology, lack of good control groups, lack of screening for acute head injuries, alcohol use, or previous motor vehicle accident. In assessing former players who played with older ball, no way of distinguishing effects of concussion and other head injuries.
- **U.S. National team players and age-matched track athletes** were assessed using MRI, surveys about symptoms of head injury, and screens for alcohol use.
  - Heading exposure index: length of season and amount of potential heading for various soccer activities used to derive participation score for each player.
  - Seven soccer players had history of previous head injury (five with LOC), all during soccer play.
  - Eight runners had previous head injury (four with LOC), five sport-related (not track), three unrelated to sports.
  - No difference in current or past alcohol use between soccer players and runners.
  - Nine soccer players with positive MRI findings (cortical atrophy, ventricular enlargement, focal atrophy, cavum septi pellucidi); similar findings in six track and field athletes.
  - Symptoms and MRI findings in soccer players did not correlate with age, number of years of play, heading exposure index, or number of headers.
  - None of MRI findings or total MRI scores correlated with number of years of participation, number of headings, number of head injuries, score of total symptoms.
  - No correlation between MRI findings and age or alcohol consumption.
  - Only significant predictor of symptoms was history of prior acute head injury ($p = 0.003$; r value = 0.63).
- **Haglund (1993) compared Swedish amateur boxers with age-matched track athletes and soccer players** (active and former first- and second-division Swedish players considered "typical headers" by managers) and track athletes. Parameters evaluated included physical findings, neurologic exam, Mini-Mental Status exam, personality trait study, CT, MRI scans, EEG, and neuropsychological tests.
  - No differences in physical, neurologic, CT, or MRI abnormalities among groups.
  - Significantly higher incidence of mild-to-moderate EEG abnormalities in boxers; no EEG changes noted in soccer players (except 1 arachnoidal cyst).
  - Boxers who had fought more than 30 matches performed poorly on both dominant and nondominant finger-tapping tests compared with the other two groups.
  - Significant negative correlation found between finger-tapping performance and length of soccer career, length of boxing career, number of boxing matches (more than 30).
  - Conclusion: No signs of chronic brain damage in soccer players known as frequent headers. Only positive finding was correlation between length of soccer career and slightly reduced finger tapping (values were still within normal limits).
- **Matser (1998) compared 53 active Dutch soccer players with 27 elite swimming and track athletes** in the Netherlands, using neuropsychological tests.
  - Classified soccer players as headers or nonheaders (goalkeeper and midfielders classified as nonheaders [rationale unclear], forwards and defenders as headers).
  - Average age: 24.4 years; no difference between percentage of athletes sustaining non–sports-related concussion; soccer players achieved higher educational level, higher number of anesthesias, lower alcohol use.
  - 54% of soccer players had one or more sport-related concussions with or without LOC, 79% sustained one or more head-to-head collisions (with or without concussion).
  - Median of 50 soccer matches per year; median amateur and professional careers of 12 and 5 years, respectively.
  - Professional soccer players performed more poorly on verbal and visual memory, planning, and visuoperceptual processing tasks than controls (even after adjusting for non–sport-related concussion, alcohol, education, number of anesthesias).
  - Increasing number of headers and sport-related concussion associated negatively with memory, visuoperceptual, and planning capacity.
  - Forwards and defenders performed significantly lower on some neuropsychological tests compared with midfielders and goalkeepers.
  - Effect of sport-related concussions and number of headers not separated; also difficult to ascertain how these groups were identified.
- **Matser's cross-sectional study (1999):** Soccer players scored significantly lower on neuropsychological tests of planning and memory than swimmers and track athletes.
  - Soccer players reported more sport-related concussions than controls.
  - Number of concussions inversely related to performance on several neuropsychological tests.
  - Study concluded that repetitive heading may explain poor cognitive function.
  - Several methodologic concerns: soccer players drank more alcohol, effect of prior concussions not separated from heading exposure, authors did not explain how they obtained some of their data about severity of concussions.
- **Other prospective studies**
  - Putukian (2000): no acute effect of heading on cognitive function as assessed by neuropsychological tests before and after practice session.
  - Putukian (2001): no effect of heading on cognitive function in men and women college soccer players as assessed by neuropsychological testing.

Prevention of Head Injury

- Helmets do not prevent concussion; no evidence to suggest helmets or headbands will play a role in decreasing concussion.
- Potential role for strengthening of neck musculature, proper technique, and enforcement of rules in preventing illegal play/contact.
- Marketing of headband products to parents and coaches with claims that use associated with decrease in concussion unfounded.
- Prospective data shows no effect of headbands on forces incurred during heading. Deformation of ball on head with current manufactured balls prevents injury.
- Unclear if headbands may change the game by making athletes more aggressive given unfounded sense of protection from headband. This would theoretically increase risk for injury.
- Mouth guards: decrease risk for dental injury. Newer materials and construction make it easier to use a mouth guard without inhibiting breathing or communicating.

GUIDELINES FOR SUCCESSFUL PARTICIPATION IN SOCCER

- Preparticipation physical examination: focus on previous ankle or knee injuries, careful assessment for ligamentous laxity as well as history of concussion.

- Prophylactic taping or bracing for ankle laxity.
- Further evaluation and strength testing for knee laxity, orthopedic consultation.
- Optimize sport-specific cardiovascular training; endurance and intermittent sprints.
- Proper warm-up before practice and game; no shooting on goal before warm-up.
- Well-designed 15- to 20-minute flexibility program stressing adductors, hamstrings, quadriceps, gastrocsoleus complex, iliotibial band, upper extremities, and neck.
- Flexibility program after warm-up or conclusion of practice/game.
- Ensure adequate equipment, including shoes, clothes, shin guards; consider mouth guards.
- Proper nutrition and hydration; education about performance enhancement.
- Adequate strength of major muscle groups: trunk, abdomen, lower extremities, neck.
- Proprioceptive training; may be sport-specific (ball work, juggling).
- Emphasis on jumping skill and technique (landing on flexed knee).
- Practice exercises emphasizing motor coordination, agility, and balance.
- Ensure proper technique for all skills, especially heading, blocking, and tackling.
- Use smaller ball for younger participants.
- Do not allow children to move or hang on goals; secure all goals.
- Communication with parents and coaches before and throughout season about methods to decrease injuries and treatment of injuries.
- Proper education of athletes about injury and prevention. Explain increased risk with foul play.
- Proper supervision of practice sessions by certified athletic trainer and of games by trainer and physician.

SUMMARY

- Soccer enjoys a relatively low injury profile; significant demands physiologically, both aerobically and anaerobically, for balance and proprioception; ideal sport for youth and adults.
- Soccer-specific nutrition important, may improve performance.
- Lower extremity injuries predominate; ankle most common, knee most severe.
- ACL injuries in female players remain of paramount concern.
- Head injuries of increasing concern, especially in women's game. No acute effect of heading on cognitive function, and no effect of prior concussion on cognitive function in soccer players. Long-term effects of severe or cumulative concussions less certain. Why concussions are more common in women than men is unclear. More prospective research is needed to answer these questions.

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Basketball

60

D. Thompson McGuire

INTRODUCTION

- Basketball has one of the highest overall injury rates among noncollision sports.
- It is a contact sport and is a fast and aggressive game.
- High-risk sports have been defined to involve contact, a high jump rate, and indoor participation.

EPIDEMIOLOGY

See Table 60-1.

SPECIFIC INJURIES

Head and Facial Injuries

- Basketball is the sport **with the second greatest number of facial injuries,** including eye and oral trauma, in the United States.
- **Lacerations**
  - Most frequently to head and facial area; common areas are eyebrows, lips, and chin.
  - **Mechanism of injury:** Collision with another player's bony prominences (elbows, knees, head, hand) or contact with the court.
  - **Treatment:** Follow Occupational Safety and Health Administration (OSHA) guidelines; athlete with bleeding injury must be removed from competition until bleeding controlled and blood cleaned from body and uniform. Use focal pressure, sutures, adhesive dressing (Steri-strips may be used temporarily for immediate return to play).
- **Nasal fractures**
  - Ascertain any airway obstructions (see Chapter 42, Maxillofacial Injuries).
  - Hard face masks may permit player to resume activity.
  - Order x-rays if degree of swelling, tenderness, or deformity warrant.
  - Closed reduction may be performed in a few days.
- **Concussions**
  - Close assessment of level of consciousness, memory loss (see Chapter 39, Head Injuries).

Table 60-1 EPIDEMIOLOGY OF BASKETBALL

	Men's	Women's	High school	Professional
Rate of injury	Game = 9.9 per 1000 Practice = 4.3 per 1000 A-Es **(Athlete Exposures)**	2× higher in games than in practice (7.68 vs. 3.99 per 1000 A-Es)	Injury risk 9× more likely in games than in practice	Game, WNBA = 24.9 per 1000 A-Es NBA = 19.3 per 1000 A-Es
Common injuries	60% of all injuries are to lower extremity Ankle ligament sprains are most common Knee internal derangements most common injury causing athlete to miss more than 10 days of participation	60% of all injuries are to lower extremity Most common is ankle sprains (30% of sprains were recurrent) Ankle ligament sprains (24.6% of game injuries, 23.6% practice) Knee internal derangements (15.9% game, 9.3% practice) Patellar injuries (2.4% game, 4% practice) Concussions (6.5% game, 3.7% practice)	Sprains and contusions most common injuries Ankle is most common site of injury, followed by knee	65% NBA injuries, 66% WNBA injuries to lower extremity Knee most frequently injured structure (19% of cases in NBA, 22.5% WNBA) Ankle most common site of injury in game (21% NBA, 20% WNBA) Lateral ankle sprain most common diagnosis in both leagues
Mechanism of injury	Player contact (52% game, 44% practice) Other contact (balls, standards, ground) (24%) No contact (22%)	Player contact (46% game, 31% practice) Other contact (24% game, 18% practice) No contact (29% game, 47% practice)		
Anterior cruciate ligament (ACL) injuries	Mechanism: No contact—60.3% Player contact—23.1% Other contact—15.4%	8% of all game injuries (0.66 per 1000 A-Es) 64% occurred as result of noncontact mechanism		Study of WNBA ACL injury over five seasons (1999-2003) revealed white European players had an 11× greater rate of ACL tear than black and African-American players
Other	Trend seen in increasing number of injuries to head and face			Overuse and inflammatory conditions (tendonitis, bursitis, synovitis) accounted for greatest amount of time lost from practices and games in both leagues (22% NBA, 27% WNBA)

Data from Agel J, Arendt EA, Bershadsky B: Anterior cruciate ligament injury in National Collegiate Athletic Association basketball and soccer players: A 13-year review. Am J Sports Med 33:524-530, 2005; Agel J, Olson DE, Dick R, et al: Descriptive epidemiology of collegiate women's basketball injuries: National Collegiate Athletic Association injury surveillance system, 1988-1989 through 2003-2004. J Athl Train 42:202-210, 2007; Deitch JR, Starkey C, Walters SL, Moseley JB: Injury risk in professional basketball players: A comparison of Women's National Basketball Association and National Basketball Association athletes. Am J Sports Med 34:1077-1083, 2006.

- Do not return symptomatic player to competition.
- Preparedness for severe concussions:
  - Use emergency resuscitative measures if necessary. Equipment should be readily available.
  - Transport to designated medical facility.
  - Document neurologic deficits at all times. Observe for any neurologic deterioration, including changes in personality and cognitive status.
  - Consult neurologist or neuropsychologist if necessary.
- **Eye injuries**
  - Corneal abrasion is most common (see Chapter 41, Eye Injuries in Sports).
  - Trauma from opponent's fingers or fingernails.
  - Check visual acuity and extraocular muscular function.
  - Corneal abrasion diagnosed with fluorescein strip and blue light.
  - Abrasion resolves spontaneously within 48 to 72 hours.
  - If injury not straightforward, refer to ophthalmologist for complete exam.
  - Most injuries **preventable** with protective goggles.
- **Dental injuries, mouth guards**
  - Dental injuries can be permanent and disfiguring (see Chapter 42, Maxillofacial Injuries).
  - Corrective treatment is expensive.
  - Mouth guard users have significantly lower rates of dental injuries.

Cervical Spine Injuries

- Most are cervical strains (see Chapter 40, Neck Injuries).
  - **Nerve root stretching:** Player complains of unilateral *stinging*, *burning*, or painful numbness extending from side of neck and shoulder down to fingertips.
  - **Mechanism of injury:** Diving after loose ball; blow to head with force greater than cervical musculature can withstand, stretching of brachial plexus with resulting neurapraxia.
  - **Treatment:** Rest, nonsteroidal anti-inflammatory drugs (NSAIDs), muscle relaxants.
  - If cervical spine is tender to physical exam or neck is painful with range of motion (ROM), then radiographs are necessary to rule out fracture or dislocation.
- Cervical fractures and dislocations are uncommon in basketball.

Lumbar Spine Injuries

Lumbar spine injuries associated with basketball are usually low back strains related to jumping and high-impact activities combined with lateral rotation (see Chapter 47, Thoracic and Lumbosacral Spine Injuries).

Shoulder Injuries

- Shoulder joint has the greatest ROM of all joints in the human body (see Chapter 43, Shoulder Injuries). Stability depends on static restraints of joint capsule, glenoid labrum, and glenohumeral ligaments and on the neuromuscular control of rotator cuff and dynamic positioning of scapular body and glenoid fossa.
- **Dislocations**
  - Anterior instability accounts for 98% of all shoulder instabilities.
  - **Mechanism of injury** is usually external rotation and forward elevation. May occur when diving for a loose ball, landing awkwardly onto outstretched hand, or reaching out to side to defend against another player and having arm forced backward.
  - **Diagnosis** apparent on physical examination: humeral head is palpable anteroinferiorly, defect below acromion. Player is unable to move the shoulder. Appropriate x-rays are usually necessary before reduction.
  - Requires immediate attention and reduction.
- **Rotator cuff injuries**
  - Tendinosis, tears (partial and complete).
  - **Mechanism of injury:** Single traumatic event (rare), repetitive microtrauma (more common).
  - **Differential diagnosis:** Biceps tendinitis, labral tear, biceps anchor pathology (SLAP [superior labrum from anterior to posterior] lesion).
- **Acromioclavicular (AC) joint injuries**
  - **Mechanism of injury:** Usually caused by landing on superolateral shoulder. Often occurs when a player is undercut while coming down from a jump.
  - **Examination:** Tenderness and possible deformity at AC joint.

Elbow Injuries

- Elbow and forearm injuries are frequent but rarely result in fractures or dislocations (see Chapter 44, Elbow Injuries).
- **Common injuries:** Abrasions, lacerations, olecranon bursitis.
- **Causes:** Diving after loose ball, defending against "posted-up" opponent, controlling rebound while swinging elbows.
- **Treatment:** Protective pad, aspiration/injection. Consider bursectomy for chronic cases.

Wrist Injuries

- **Common mechanism of injury:** Falling onto court with outstretched hand when undercut by another player and unable to land on feet. Other frequent cause of wrist problems is repetitive use (see Chapter 45, Hand and Wrist Injuries).
- Sprains, strains, tendinitis, triangulofibrocartilage complex (TFCC) tears.
- Distal radius fractures.
- Scaphoid fractures.
  - Deep palpation of "snuff box" elicits tenderness.
  - Radiographs of wrist, including scaphoid view. X-rays may not reveal a nondisplaced fracture until 2 weeks after the injury when resorption makes the fracture line more obvious.
  - **Treatment:** Err on side of overtreating occult injuries if snuffbox tenderness is significant. Initial use of thumb spica splint; cast after swelling reduced. Surgery for displaced fractures. Consider immediate internal fixation, even for nondisplaced fractures, because of more predictable healing in shorter time with less immobilization and quicker return to participation.
- Other serious but potentially overlooked wrist injuries: dislocation of lunate, scapholunate dissociation.
- Any persistent (more than a few days) wrist pain and swelling should be radiographed and followed closely. If x-rays are negative and symptoms persist, follow-up x-rays are obtained and, if warranted, magnetic resonance imaging (MRI) is done.

Finger Injuries

- Very common in basketball players (see Chapter 45, Hand and Wrist Injuries).
- Frequently caused by direct trauma from ball, opponent, or rim of basket.
- **Swan neck deformity:** Hyperextension of proximal interphalangeal (PIP) joint with flexion of distal interphalangeal (DIP) joint resulting from volar plate injury of PIP joint (Fig. 60-1).
- **Boutonnière deformity:** Hyperextension of DIP joint with flexion of PIP joint caused by disruption of extensor tendon insertion (central slip) into base of middle phalanx (see Fig. 60-1).
- **Mallet finger:** Flexion deformity of DIP joint. Trauma to tip of extended finger results in avulsion of insertion of extensor tendon dorsally into distal phalanx (see Fig. 60-1).

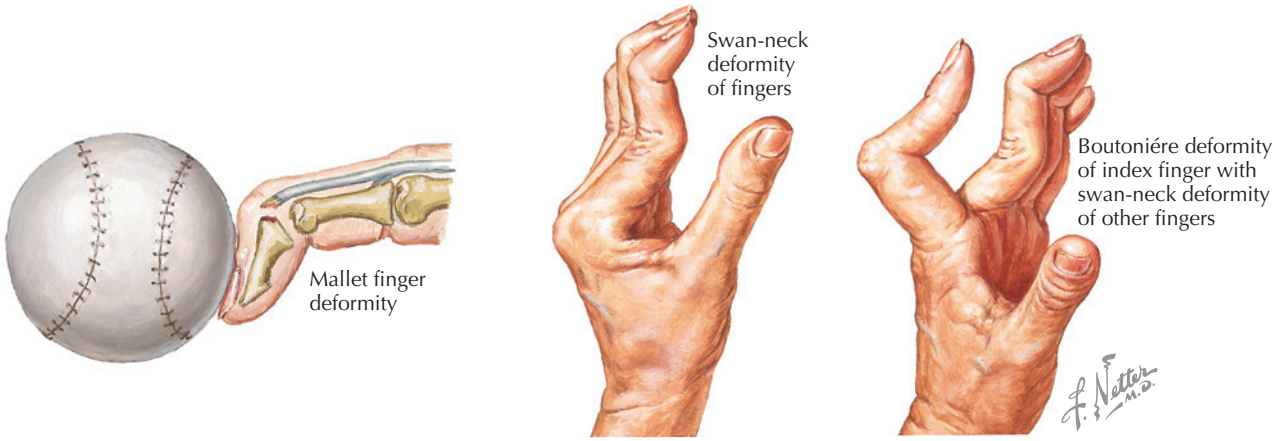

Figure 60-1 Finger Injuries.

- **Treatment:** Strict immobilization with splint for 6 weeks. Compliance is poor. Surgical repair may be required. Consider percutaneous pinning of mallet finger injuries to ensure maintenance of reduction, allow skin care, and facilitate earlier return to play.

Quadriceps and Hamstring Injuries

- **Mechanism of injury:** Commonly a result of direct blow from another player's knee. Muscle strains also common because of excessive tension generated in muscle-tendon unit during basketball participation (see Chapter 48, Pelvis, Hip, and Thigh Injuries).
- **Prevention:** Warm-up muscles adequately; stretch out muscles before competing.
- **Treatment:** Ice/heat, rest, physical therapy modalities; gradual resumption of activities as symptoms lessen.

Knee Injuries

- **Anterior cruciate ligament injuries** (see Figure 60-2 and Chapter 49, Knee Injuries)
  - ACL tears are up to 4 times more common in female than in male basketball players (see Table 60-1).
  - Factors include hormonal differences, absolute differences in graft size: weight between genders, biomechanical differences (valgus knees, wider pelvis in women), behavioral differences (e.g., landing with knees more extended and femora internally rotated).
  - Preventive training programs have been developed to attempt to address behavioral differences.
- **Jumper's knee** (see Chapter 49)
  - Overuse injury; often persistent and recurrent problem.
  - **History:** Insidious onset of symptoms, including pain and tenderness at any of three locations: insertion of quadriceps tendon onto superior patellar pole; insertion of patellar tendon onto tibial tubercle; origin of patellar tendon (infrapatellar ligament) from inferior patellar pole (most common).
  - **Assessment:** Look for possible contributing factors, including malalignment; patella alta; abnormal patellar laxity; tightness of hamstrings, quadriceps, or heel cord; muscular imbalance; weakness of ankle dorsiflexion; recent change in training regimen or shoewear.
  - **Treatment:** Activity modification, ice, NSAIDs; correction or improvement of predisposing factors listed above; taping or bracing to unload inferior patellar pole, infrapatellar straps. Occasionally, highly selective steroid injection for recalcitrant cases. Educate patient about risk for rupture. Surgical management is done in worst cases when athlete can no longer compete (drilling, multiple slits in tendon, excision of area of tendinosis).

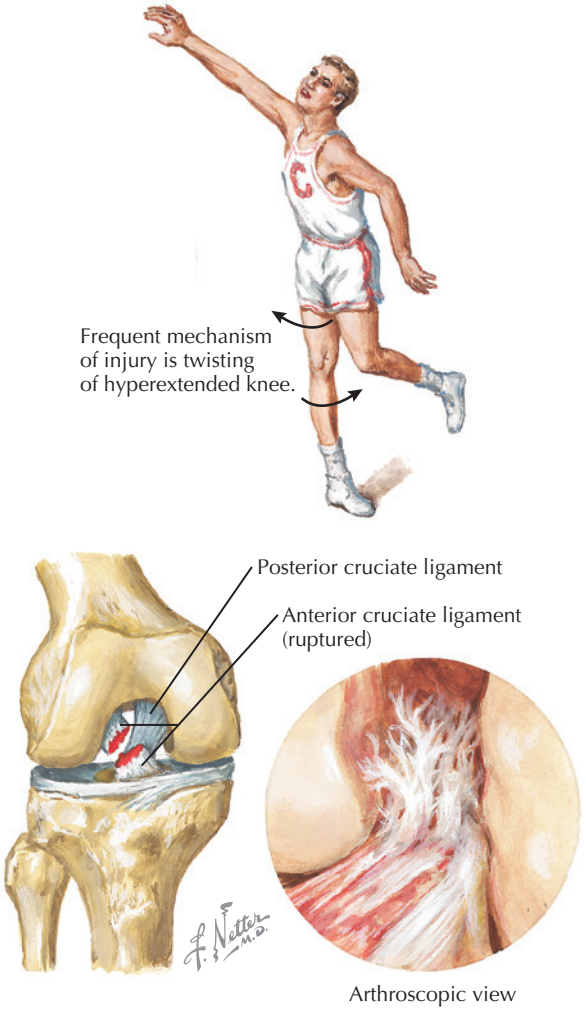

Figure 60-2 Anterior Cruciate Ligament (ACL) Injury.

Ankle and Foot Injuries

- **Ankle sprains** (see Chapter 50, Ankle and Leg Injuries)
  - Most common injury (see Table 60-1).
  - **Risk factors:**
    - History of ankle sprain may increase risk of another sprain to 5 times greater than risk for sprain in players without history of injury.
    - Players wearing shoes with air cells in the heel may be 4 times more likely to sprain their ankle.

  - Players that do not stretch before participation may be 2.5 times more likely to sprain their ankle.
  - **Mechanism of injury:** Usually inversion injury—player frequently lands on foot of another player and inverts (rolls) the ankle. Another frequent mechanism is sudden lateral movement in the opposite direction of the ankle that is injured.
- **Ruptured achilles tendon** (see Chapter 50)
  - More commonly occurs in players in their 3rd and 4th decades.
  - **Mechanism of injury:** Sudden, intense, and often unexpected eccentric or concentric contraction of ankle plantarflexors.
- **Foot ligamentous injuries** (see Chapter 54, Foot Problems)
  - Medial subtalar dislocation ("basketball foot"): dislocation of talocalcaneal and talonavicular joints; treated with immediate reduction and immobilization.
  - Subtalar sprains
  - Midtarsal (Chopart) joint: talonavicular and calcaneocuboid joint sprains, subluxations, dislocations.
  - Tarsometatarsal (Lisfranc) joint sprains, subluxations, dislocations.
- **Fifth metatarsal stress fractures**
  - Consider early operative intervention with intramedullary screw fixation.
  - If sclerosis or atrophic nonunion is present, debridement and bone grafting may be required.
  - Surgery eliminates need for prolonged immobilization and non–weight-bearing, thereby avoiding secondary problem of disuse osteopenia and allowing earlier return to basketball.

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Wrestling

John J. Wilson, Jesse J. Donnenwerth, and Gregory L. Landry

GENERAL CONSIDERATIONS

Wrestling Styles

Greco-Roman wrestling: Grew out of a form of 19th-century show-wrestling characterized by upper-body throws. The use of legs is prohibited in this style and points are often scored during frequent throws. Takedown points are awarded based on the degree of skill exhibited in throwing an opponent to the mat, with more points awarded for throws that place the other wrestler in a position exposing the shoulders to the mat. There are three 2-minute periods (one standing, and up to two ground phases), and a match is won after a fall (pin) or when a wrestler has won two of the three periods.

International freestyle wrestling: Popular throughout the world. Unlike Greco-Roman, the use of legs is allowed and is an important part of many wrestling holds and maneuvers. Trips, throws, and below-the-waist holds are permissible in freestyle wrestling. Like the Greco-Roman style, participants wrestle three 2-minute periods. Points are awarded for takedowns, reversals, exposure of opponent's back to mat (also known as the "danger position"), and forcing the opponent out-of-bounds. Wrestlers may win by fall (pin) by holding the opponent's shoulders on the mat simultaneously.

Intercollegiate freestyle wrestling (also known as folkstyle wrestling): Practiced throughout the United States at the high school and collegiate levels. Intercollegiate freestyle wrestling is most similar to international freestyle, but considerable differences exist. The match consists of three periods, and additional overtime periods as necessitated by a tie at the end of three periods. Points are awarded for takedowns, escapes, reversals, near falls, penalties, and time advantages (riding time; collegiate level only). As in other wrestling styles, a wrestler wins by achieving a fall (pinning the opponent). Table 61-1 lists various weight classes and divisions.

Other wrestling related sports: Include beach wrestling, sambo, grappling, and mixed martial arts.

Mechanisms of Injury

- **Direct wrestler contact** results in contusions and lacerations, which commonly occur during takedowns, head-to-head contact, or blows from an errant body part.
- **Friction** occurs with constant contact with the mat and/or opponent.
- **Falls** occur during takedowns, trips, or throws and can be compounded by the weight of the opponent's body landing on the bottom wrestler.
- **Twisting and leverage** mechanisms are common with various holds and maneuvers in which joints are forced past their normal range of motion.

Patterns of Injury

- The combative nature of wrestling and forceful body contact results in a relatively high incidence of injuries which is comparable to that of football, soccer, lacrosse, and ice hockey.
- Unlike other sports, there are more injuries that occur in practice compared to competition. However, when comparing injury rates (i.e., number of injuries per exposure time), injuries are more likely to occur during competition.
- Catastrophic injuries in wrestling are rare. When they do occur, serious injuries commonly involve the head, cervical spine, and lumbar spine. The majority of catastrophic injuries occur to the defensive wrestler during the process of being taken down from the neutral position.
- The neutral position and the defensive position are associated with higher injury incidence rates.
- Wrestlers often contract communicable and noncommunicable skin lesions because of frequent skin-to-skin contact, frequent abrasions, and exposure to microbes on wrestling mat surfaces.

SPECIFIC INJURIES AND CONDITIONS

Head and Face Injuries

Lacerations

Description: Occur frequently during wrestling and usually result from a direct blow from another wrestler's head or an errant elbow. Lacerations commonly occur around the orbit, scalp, zygomatic arch, lips and mouth.

Treatment: A sturdy, supportive, and protective dressing on top of the wound is often necessary to allow continued participation.

Precautions: Following competition, lacerations should be thoroughly irrigated, properly repaired, and closely monitored for signs of infection. Heavy nylon (3-0 to 5-0) sutures are commonly used to provide adequate closure while providing durability for continued participation in wrestling. Abrasions can be treated with petroleum-based ointments prior to wrestling.

Epistaxis

Description: Occurs frequently in wrestling following a direct blow or cross-face maneuver contact with the nose. The nasal septum is a highly vascular structure and epistaxis may present with mild-to-profuse bleeding.

Treatment: Mild epistaxis may resolve spontaneously with little intervention. Epistaxis should be treated with direct nasal septal pressure over affected nares while the head is tilted slightly forward with the athlete sitting or standing. Ice application to the nasal bridge may provide helpful vasoconstriction. A **cotton pledget or "nose plug"** treated with petroleum jelly or a mild vasoconstrictor such as oxymetazoline 0.05% may be inserted by gently twisting into affected nostril. The use of phenylephrine and cocaine are no longer allowed at many levels of competition and are banned by the National Collegiate Athletic Association (NCAA).

Precautions: Athletes should avoid blowing their nose after significant bouts of epistaxis to prevent rebleeding. Recalcitrant, severe bleeding may require referral to a medical facility for nasal packing or definitive treatment. **Recurrent bouts** of epistaxis may be related to allergic rhinitis or sinus infection. Rarely, they are associated with a blood clotting disorder such as von Willebrand disease. If these causes are ruled out, either nasal cauterization with silver nitrate sticks or electrocautery can be done by a trained professional.

Nasal Fractures

Description: Can occur with a forceful blow to the nose.

Treatment: When the associated bleeding subsides, the nasal septum should be examined for a hematoma. Septal hematomas require urgent incision and drainage to prevent destruction of the nasal cartilage and resulting saddle nose deformity. When the injury is 5 to 7 days old, consider referral to an otorhinolaryngologist for fracture reduction if there is septal deviation or persistent obstruction of a nares.

Return to play: Wrestlers can often return to activity by wearing a protective face guard that adequately protects the nose.

Table 61-1 U.S. AGE DIVISIONS AND WEIGHT CLASSES FOR WRESTLING, 2008

USA kids division (pounds)													
Bantam (born 2000-2001)		**Midget (born 1998-1999)**		**Novice (born 1996-1997)**		**School boy/girl (born 1994-1995)**				**FILA cadet (born 1991-1993)**			
Boys/Girls		**Boys/Girls**		**Boys/Girls**		**Boys**		**Girls**		**Men**		**Women**	
40	65	50	87	60	100	70	136	70	136	86-92.5	167.5	79.25-83.75	123.5
45	70	55	95	65	105	77	144	77	144	101.25	187.25	88	132.25
50	75	60	103	70	112	84	152	84	152	110.25	187.25-220.5	94.75	143.25
55	75+	65	112	75	120	91	160	91	160	119	275.5	101.25	154.25
60		70	120	80	130	98	175	98	175	127.75		108	172
		75	120+	85	140	105	190	105	190	138.75		114.5	185
		80		90	140+	112	210	112	210	152			220.5
						120	235	120	235				
						128		128					
USA wrestling division (pounds)													
Junior (born > 9/1/88)		**FILA Jr. World (born 1988-1990)**				**University (born 1984-1990)**				**Senior (born ≤ 1988)**			
Men only		**Men**		**Women**		**Men**		**Women**		**Men**		**Women**	
98	145	101.25-110.25	154.25	88-97	138.75	121.25	163	105.75	138.75	121.25	185	105.75	138.75
105	152	121.25	163	105.75	147.5	132.25	174	112.25	147.5	132.25	211.5	112.25	147.5
112	160	132.25	174	112.25	158.75	138.75	185	121.25	158.75	145.5	264.5	121.25	158.75
119	171	138.75	185	121.25	175.25	145.5	211.5	130	169.75	163		130	
125	189	145.5	211.5	130		154.25	264.5						
130	215		264.5										
135	285												
140													
NCAA intercollegiate freestyle (pounds)													
						125	165						
						133	174						
						141	184						
						149	197						
						157	HWT (183-285)						

FILA, Fédération Internationale des Luttes Associées; HWT, heavyweight.

Corneal Abrasions

Description: Can occur when a wrestler is poked or scratched in the eye.

Treatment: Consideration should be given to empiric topical antibiotics. More severe lacerations, especially those that do not heal overnight, should be evaluated by an eye specialist with a slit lamp.

Orbital Floor "Blowout" Fractures

Description: May result from an errant blow to the orbit. This injury may not cause much pain and is associated with double vision with upward gaze. Periorbital crepitus may occur as a result of an air leak from the maxillary sinus.

Examination: This injury requires evaluation with orbital computed tomography (CT) and ophthalmology consultation to determine if operative treatment is necessary.

Auricular Hematoma ("Cauliflower Ear")

Etiology: Blunt trauma to the auricle may shear the perichondrium away from the underlying cartilage, causing bleeding and a subsequent hematoma. Left untreated, the hematoma may precipitate new cartilage formation that results in deformity of the auricle known as **"cauliflower ear"** (Fig. 61-1).

Diagnosis: Moderately painful auricular swelling with loss of the normal convexities of the ear, occurring minutes to hours after inciting trauma.

Differential diagnosis: Cellulitis, perichondritis.

Treatment:

- **Immediate aspiration** with an 18- to 22-gauge needle. Occasionally multiple aspiration sites may be required for complete hematoma evacuation. Incision drainage is usually not necessary. Utilize aseptic technique.
- **Local anesthesia** with 1% lidocaine with or without epinephrine or regional auricular block may be employed for anesthesia but is usually not necessary.
- **Compression dressing** after aspiration prevents hematoma reaccumulation. Ear compression dressings can be created with a variety of materials. One simple technique is by applying petroleum gauze within the anterior pinna. Gauze placed behind the pinna will provide adequate posterior pressure. Fluffed gauze can then be applied over then entire ear and held in place with elastic bandage. Specialized silicone ear molds exist and are sometimes applied by specialists. Pressure dressings may be required for 3 to 5 days.
- Cotton roll suture dressings are not advised because of increased risk of further ear injury and infection.

Precautions: Monitor daily for signs of infection and hematoma reaccumulation. Disfiguring if left untreated. Wrestlers should be informed that a deformity may occur even if hematoma is treated properly.

Concussions

Description: Results from acceleration or deceleration forces and direct blows to the head.

Prevention: There is no evidence that mouth guards or protective gear prevent or minimize severity of concussions in wrestling, but their use should be encouraged to prevent dental injuries.

Return to play: Graduated return-to-play recommendations should be followed for wrestlers who have sustained a concussion. Wrestlers who are symptom free at rest may progress to aerobic exercise. When asymptomatic with exertional activities, they may progress to drilling/sparring. If asymptomatic with sparring, return to full activity is permissible. Wrestlers may benefit from baseline preseason, and postconcussive neuropsychological testing to monitor symptoms and guide return to play after a concussion.

Neck Injuries

Description: Occur frequently in wrestling. Sprains, strains, degenerative disk changes, and brachial plexus injuries are encountered in wrestling. Because of rules prohibiting slams and certain types of throws and maneuvers, catastrophic neck injuries in wrestling have declined.

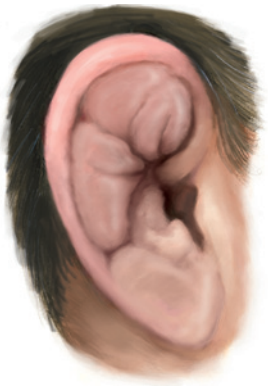
Etiology: Initiating contact with the head held in hyperextension, or **"bulling,"** is a common wrestling technique. This position carries increased risk of neck injury. **Stretch and pinch** are common during the takedown process, when head and neck are forced laterally while shoulder is depressed. This maneuver may cause a brachial plexus injury on the side that is depressed or a pinch injury to the nerve roots on the side that is compressed. **Extension and axial compression** may occur during a takedown in which the wrestlers head is the first to contact the mat.

Chest and Trunk Injuries

Common to wrestling.

Rib and Costochondral Injuries

Etiology: Forced compression injuries such as contusion or fracture are observed when one wrestler lands on another opponent's chest during a takedown maneuver. **Muscle strain** is common because of torsional wrestling maneuvers.



Presentation of auricular hematoma (cauliflower ear).

Protective headgear is worn to prevent auricular hematomas.

Figure 61-1 Auricular Hematoma ("Cauliflower Ear"). *(Photograph © 2008 Del Brown Photography, LLC)*

Diagnosis: Dyspnea, tenderness, and pain, mostly associated with inhalation, trunk motion, or chest compression; x-ray examination is helpful for identifying fractures.

Treatment: Rib injuries may be quite painful. Although further injury is unlikely, participation may be limited by pain.

- Rest, ice, heat, and anti-inflammatory medications
- Local corticosteroid injection may be beneficial in persistent localized costochondritis.
- Topical anesthetic patches (e.g., 5% lidocaine transdermal patches) may offer relief from rib pain.

Precautions: Consider radiographic studies to evaluate for rib fractures, pneumothorax, or other rare associated sequelae such as a hemothorax.

Muscle Strains

Description: May occur from a variety of mechanisms and forces sustained during wrestling maneuvers. Oblique and rectus abdominis muscles may be affected.

Back Injuries

Description: Occur frequently in wrestling. Common back injuries include sprain, strain, spondylolysis, disc injury, or sacroiliac sprain.

Etiology: Because of the repetitive concentric and eccentric forces generated from the neutral position, low back injuries are common.

- Torsional mechanisms of injury.
- **Extension against resistance** occurs during lifts and throws (Fig. 61-2).
- **Hyperflexion mechanisms** of injury occur with various rolling maneuvers.

Shoulder Injuries

Description: Occur frequently.

Types of shoulder injuries:

- Acromioclavicular joint separation may occur after landing forcibly on the lateral shoulder.
- Glenohumeral subluxations or complete dislocations of the shoulder are common in wrestling. The shoulder is commonly forced past its normal range of motion during certain maneuvers, especially those which abduct and externally rotate the upper arm.
- Glenoid labral injuries and proximal biceps tendon injuries can occur after a fall on an outstretched arm or during a strenuous lift maneuver.
- Sternoclavicular sprain can occur with a blow to the lateral shoulder or with compression of the chest.

Back extension against resistance during a lifting throw.

Figure 61-2 Back Injuries.

Elbow Injuries

Description: Common and include fractures, hyperextension, olecranon bursitis, and epicondylitis. One high-risk maneuver is when a wrestler completely extends elbow to break fall when landing on mat after takedown (Fig. 61-3), or when defending a "gut wrench" maneuver. This "posting" maneuver may lead to hyperextension or fracture-dislocation injury.

Prevention: Wrestlers should be coached to flex elbow slightly and slide their hands when defending the "referee" position (see Fig. 61-3).

Treatment: Hyperextension taping prevents full extension of elbow following a hyperextension injury.

Hand and Wrist Injuries

Description: Frequent hand and finger injuries include sprain, dislocation, subluxation, and fractures.

Treatment: Finger sprains can be buddy-taped for splinting. Finger dislocations can be reduced and properly splinted and padded to allow further competition.

Thumb Injuries

Description: Can be debilitating to a wrestler because of the need for repetitive grasping and gripping.

Treatment: Most thumb injuries involve the ulnar collateral ligament (UCL) following a forced abduction. Thumb spica taping or splints may be used. Rarely, ligament reconstructive surgery may be required when the UCL is completely ruptured and there is excessive laxity.

Elbow injuries often occur while attempting to break a fall.

The "referee" position, also known as the parterres or on-the-ground position.

Figure 61-3 Elbow Injuries.

Knee Injuries

Description: Most common wrestling injury and account for nearly 25% of all wrestling injuries. Certain maneuvers place extreme stress on the knee structures.

Etiology: Although exact pathomechanics of meniscal injuries are not always identified, Wroble and colleagues have cited the following mechanisms: **indirect forces**, **torsion**, **shearing/hyperextension**, and **insidious/overuse.**

Diagnosis: Knee injuries may present atypically in wrestlers; lateral, rather than medial, meniscal injuries occur more frequently; individuals may have minimal symptoms other than a complaint of "locking." Many wrestlers may attempt to "wrestle through" meniscal injuries.

Common knee injuries in wrestling include **meniscal tears, medial collateral and lateral collateral sprains, and prepatellar bursitis.**

- **Collateral ligament sprains** are the most common knee injuries and occur frequently during takedowns and defensive maneuvers (Fig. 61-4).
- **Meniscal injuries** occur frequently, especially during takedowns and defensive maneuvers. Wrestling is associated with a high frequency of lateral meniscus tears compared to other sports.
- Incidence of **anterior cruciate ligament** and multiligamentous injuries are low.
- **Leg wrestling** may carry higher potential for medial collateral ligament and lateral collateral ligament injuries. Some wrestlers ride their opponents, almost exclusively using their legs for control.

Prepatellar Bursitis

Description: Common problem and often affects the lead leg used during takedowns. Recurrences are frequent.

Etiology: Repetitive irritation and acute blunt trauma.

Treatment: Management of acute prepatellar bursitis is controversial. Some practitioners advise immediate aspiration to remove blood and reduce reactive inflammation. We find this is rarely necessary and initially manage every case with a compressive wrap, ice, nonsteroidal anti-inflammatory agents (NSAIDs), and knee pads for protection. Recurrent cases may require bursectomy. Corticosteroid injection results are mixed and should be reserved for difficult cases because this might increase the risk of infection.

Prevention: Protective knee pads should be worn while wrestling.

Septic Bursitis

Description: Should be considered in all wrestlers with bursitis that fails to respond with 24 to 36 hours of conservative management.

Many wrestling knee injuries occur during takedowns.

Figure 61-4 Knee Injuries.

Etiology: Usually *Staphylococcus aureus* (80%) or streptococcal species (5% to 20%).

Diagnosis: Often **asymptomatic** other than bursal swelling; may exhibit erythema, warmth, and sometimes fever. Culture, Gram stain, and sensitivity testing of bursal aspirate should always be performed.

Treatment: Empiric treatment with penicillinase-resistant penicillin while awaiting culture results is appropriate when local prevalence of community acquired methicillin-resistant *S. aureus* (MRSA) is low. In wrestlers with prior history of MRSA, or in communities with high (>10% to 15%) prevalence of MRSA, an antibiotic with activity against MRSA is suggested.

Ankle Sprains

Description: Second most common wrestling injury.

Treatment: Sprains can be adequately taped or braced for participation after adequate rehabilitation. Fractures occur infrequently.

Common Skin Conditions

See Chapter 34, Skin Problems in the Athlete.

Description: Data from NCAA Injury Surveillance System indicate that skin infections were the leading cause of practice-associated injuries (17.2%) and the second leading cause of time-loss injuries in wrestling (8.6%).

Prevention of transmission: Skin conditions must be adequately medicated and protected to prevent transmission of infection to another wrestler. Bandages must be impermeable and stand up to the rigors of wrestling. Categories of such conditions include the following:

- **Bacterial infections:** impetigo, erysipelas, folliculitis, hidradenitis suppurativa, furuncles and carbuncles; usually staphylococcal or streptococcal species.
- **Viral infections:** herpes simples ("herpes gladiatorum"), herpes zoster, molluscum contagiosum.
- **Fungal infections ("ringworm"):** tinea pedis, tinea cruris, tinea corporis.
- **Infestations:** scabies, pediculosis.
- **Noninfectious:** psoriasis, eczema.

General treatment strategies:

- **Proper hygiene:** Wrestlers should shower immediately following practices and competitions using antibacterial soaps; daily laundering of clothing and gear.
- **Mat cleansing and disinfection** should occur after each use.
- **Medications** for specific conditions as directed by a physician. Because of the high prevalence of herpes gladiatorum on many teams, an increasing number of wrestlers are taking antiviral medication for prophylaxis.
- **Isolation** of wrestler(s) with skin conditions until they are deemed to be noncontagious.
- **Proper wound care:** Abrasions and open wounds should be thoroughly cleaned and monitored daily for infection. Wounds should be adequately protected to prevent further injury and allow for healing.
- **Visual inspection** of wrestlers for contagious skin conditions prior to practices and at weigh-ins. **Disqualification** may be warranted if a skin condition is present. Participation may be permitted if solitary or limited lesions are adequately and securely covered.

TREATMENT GUIDELINES

Treatment on Mat

Time constraints: Both high school and college wrestling have a 1.5-minute injury time allotment. This time constraint challenges the physician and athletic trainer to work quickly yet thoroughly while evaluating and treating injured wrestlers during competition.

Epistaxis: Epistaxis treatment during competition does not count against the injury time allowed.

Rules regarding treatment:

- Taping is permissible during injury time-outs, but must allow normal joint motion.
- Use of braces or devices that limit normal joint range of motion prevent an opponent from applying a hold is illegal.
- Use of a medication during competition to treat a preexisting condition (e.g., albuterol for asthma) is prohibited.

Return to Wrestling

- Once the wrestler's allotted injury time has expired, the referee must decide whether the wrestler can return to competition, assuming the athlete has medical clearance to do so.
- Referee may end a match if he or she feels a wrestler faces further injury by continuing to wrestle. The referee's decision is final and may not be challenged by the coaching or medical staff.
- After evaluating an injury, a physician may allow a wrestler to continue competing. The referee has the authority to overrule the physician's decision to allow continued wrestling.
- Physician disqualification of a wrestler because of an injury may not be overruled by anyone, including the referee.

SPECIAL ASPECTS OF PREHABILITATION AND REHABILITATION

Prehabilitation:

- **Sport-specific** conditioning and technique training. Emphasis should be placed on proper techniques and maneuver mechanics.
- **Off-season and in-season** strength, endurance, and flexibility training.
- **Maturity assessment:** Youth and preadolescent wrestlers may benefit from maturation and skill matching with their opponents. This may assist in reducing injuries and increasing satisfaction with the sport.
- **Previous injury rehabilitation:** Prior injuries should be fully assessed adequately rehabilitated prior to a graduated return to full activity.

Rehabilitation: Should be implemented and guided by a certified athletic trainer, physical therapist, or physician.

WRESTLING FACILITIES AND PROTECTIVE EQUIPMENT

Facilities

Wrestling mats: Constructed from a dense foam core with a vinyl covering. Mat size, thickness, and shock-absorbing qualities are regulated by the NCAA and high school regulating bodies. Mats provide shock absorbency to help reduce injury from impact during throws and takedown maneuvers.

Wrestling room: Dimensions at least 50 square feet per wrestler to minimize chance of injury from collisions with other wrestlers. Walls and pillars in wrestling room should be padded at least 5 feet high.

Equipment

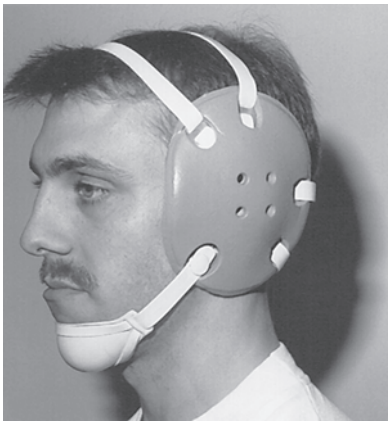
Face guard: A face guard (Fig. 61-5) may be worn to protect a wrestler with an existing facial injury or sutures.

Mouth guard: All wrestlers, especially those with orthodontic braces, should wear mouthpieces.

Knee and elbow pads: Provide protection of previously injured areas and minimize direct contact trauma. Pads should allow normal movement and be form-fitted.

Footwear: Wrestling shoes are light and heel-less and extend above the ankle. They have thin rubber soles that maximize traction with the mat. **They offer little shock absorption for running.**

A. A padded face guard may be worn to protect facial injuries.



B. Protective headgear designed to prevent injuries, including auricular hematoma.

Figure 61-5 Protective Equipment.

Headgear: Properly fitted headgear is essential for protection of ears during wrestling, and is required by most wrestling governing bodies (see Fig. 61-5).

RULES TO PROTECT WRESTLERS

Governing Bodies

- Members of NCAA Rules Committee and National Federation of State High School Associations (NFSHSA) have established individual sets of wrestling rules. Some high school rules vary by state.
- International wrestling rules and regulations are set by Fédération Internationale des Luttes Associées (FILA—International Federation of Amateur Wrestling). USA Wrestling is the governing body for amateur wrestling in the United States. USA Wrestling oversees wrestler qualification for national, international, and Olympic teams.

Specific Rules

- Subtle differences exist between high school and collegiate wrestling (Table 61-2).
- To ensure safety, referee may temporarily halt match for illegal holds, unnecessary roughness, or potentially dangerous situations.
  - **Illegal holds** are maneuvers that inflict pain and are dangerous to receiving wrestler, such as holds that cover

Table 61-2 MAJOR DIFFERENCES IN RULES FOR INTERSCHOLASTIC AND INTERCOLLEGIATE WRESTLING

Parameter	High school	College
Length of matches (minutes)		
Period 1	2	3
Period 2	2	2
Period 3	2	2
Number of matches per day	5 full matches	No limit
Weigh-ins	½ to 1 hour before meet	1 hour before meet
Weight certification	Left to discretion of individual states	Required
Pre-meet skin inspection	Highly advised	Required
Wrestling room temperature	No rule	75° F at start of practice
Minimal allowed % body fat	7%	5%

mouth, throat, and eyes. Offending wrestler is penalized with a point deduction.
- **Potentially dangerous** holds are those that force a limb to the limit of the normal range of motion, and such holds may be stopped by the referee. No penalty is assessed because potentially dangerous holds are usually unintentional. Wrestling is then resumed in the neutral position.
- **Unnecessary roughness** is deemed to be "any act that exceeds normal aggressiveness," such as forceful trip or aggressive cross-face used in a punishing fashion. A one-point penalty deduction is assessed.
- **Unsportsmanlike conduct:** First violation incurs a one-point deduction, and disqualification follows a second offense.

SPECIAL CONCERNS OF THE TEAM PHYSICIAN

Weight Loss and Weight Management

- Wrestlers of all ages commonly engage in weight loss practices in order to be certified to participate in a given weight class.
  - Fluctuations can occur frequently throughout the season.
  - Weight loss in athletes is usually the result of either hypohydration (loss of body water) or loss of body mass (adipose and lean tissue).
  - Hypohydration in excess of 3% to 5% results in decreased strength and endurance, reduced blood and plasma volume, decreased cardiac output, impaired renal perfusion, decreased glycogen stores, disturbances in thermoregulation, and electrolyte imbalances.
  - Rapid rehydration after weigh-ins is ineffective for replenishing electrolytes and replenishing glycogen stores prior to competition.
    - Effective rehydration likely occurs after 24 to 48 hours.
  - Life-threatening heat illness, rhabdomyolysis, kidney failure, and cardiac arrest may occur as a result of intentional hypohydration and rapid weight reduction practices ("cutting weight").
    - Three collegiate wrestlers died in 1997 while attempting rapid weight reduction by using fluid restriction, vapor-impermeable suits, and exercise in hot, humid environments in order to qualify for competition. Their deaths were the result of hyperthermia secondary to extreme hypohydration through intentional rapid weight loss.
- **Weight management position statements:** The practice of intentional hypohydration and rapid weight reduction ("cutting weight") as a means of weight management has been denounced by sports medicine organizations such as the American College of Sports Medicine (ACSM), the American Medical Association, and major wrestling governing organizations such as the National Collegiate Athletic Association (NCAA), the National Federation of State High School Associations (NFHS), and the National Wrestling Coaches Association (NWCA).
- **NCAA guidelines:**
  - Coaches and athletes should be educated about the adverse effects of intentional hypohydration and rapid weight loss. See www.ncaa.org/champadmin/wrestling/weight_mgmt.html and www.nwcaonline.com/sportscience.cfm.
  - Laxatives, diuretics, emetics, and self-induced vomiting should be prohibited and discouraged.
  - Exercise in vapor-impermeable suits, saunas, and steam rooms should be prohibited and discouraged.
  - A minimum acceptable weight should be determined through the use of body composition measurement during the preseason to allow for safe, gradual weight loss to the desired competition weight. The desired weight should be maintained throughout the competitive season, and compliance should be assessed using body composition and hypohydration assessments during the season.
- **American College of Sports Medicine (ACSM) guidelines** reiterate the guidelines of the NCAA and also make the following recommendations:
  - Adopt rules that schedule weigh-ins immediately prior to competition.
  - Daily weigh-ins before and after practice to monitor weight trends and hydration status. Weight loss during practice should be replenished through adequate food and fluid intake.
  - Assess body composition prior to competitive season, using valid measurement instruments.
    - Males 16 years old and younger with a body composition below 7% body fat and males older than 16 with a body composition below 5% require medical clearance prior to being allowed to compete.
    - Females require a minimal body fat of 12% to 14%.
  - Emphasize the need for daily caloric intake obtained from a balanced diet high in carbohydrates (>55% of calories), low in fat (<30% of calories), and containing adequate protein (15% to 20% of calories, 1.0 to 1.5 g per kg body weight) determined on the basis of recommended daily allowance (RDA) guidelines and physical activity levels. Minimum caloric intake levels for high school and collegiate wrestlers range from 1700 to 2500 kcal per day. Rigorous training may increase daily requirements by 1000 kcal.
  - Wrestlers should be discouraged from caloric restriction less than that of their minimal requirements.
  - Increase the number of participants per team allowed to compete by adding weight classes or by allowing for more than one representative per weight class.
  - Standardize eligibility rules for championship tournaments so that severe, rapid weight loss is discouraged (e.g., dropping an additional weight class).
  - Continue collaborative efforts between coaches, scientists, physicians, dieticians, and wrestlers to use research and education to determine the best medically sound system for selecting a weight class.
- **NCAA Wrestling Weight Certification Program:**
  - Established to determine permanent weight classes for each wrestler and eliminate incentive for unhealthy weight loss practices.

- Mandatory NCAA official weigh-in form, generated by the National Wrestling Coaches Association web site (www.NWCAonline.com) must be used for all competitions.
- Weigh-ins must occur 1-hour or less before the first match for dual, triangular, and quadrangular meets; 2 hours or less on the first day of tournament matches, and 1 hour or less on subsequent days. A 1-pound allowance is granted for the second day of tournament weigh-ins.
- NWCA optimal performance calculator (OPC) is used to establish a wrestler's lowest allowable weight (LAW) and healthy minimal wrestling weight (MWW) class.
  - The OPC serves as a clearing house for healthy MWW's and is used by most state scholastic wrestling associations, and all collegiate governing bodies (e.g., NCAA, NAIA, NJCAA).
- Assessor (physician or athletic trainer) conducts urine specific gravity testing. If specific gravity is greater than 1.020, wrestler must return no sooner than 24 hours later, in a hydrated state, for retesting. If specific gravity is less than 1.020, the wrestler's weight is recorded to the nearest pound.
- Body density (BD) is calculated with valid measurement tools (skin calipers, hydrostatic weighing, BOD POD, etc.). Equations are then used to calculate body fat percentage, fat weight, fat-free weight, and LAW (Table 61-3).
- LAW becomes wrestler's MMW. Weight class closest to MWW without going under is the wrestler's minimum weight class. No wrestler may compete below his or her MWW.
- Wrestlers wishing to compete at a weight class one higher than his or her minimum weight class may return to the original lowest certified weight class, but must not lose more than 1.5% of body weight per week while making the weight descent.

Table 61-3 SKINFOLD PREDICTION EQUATION FOR HIGH SCHOOL WRESTLERS

1. Required skinfolds:
   S1: triceps: longitudinal fold on posterior midline of upper arm midway between acromion and olecranon process.
   S2: subscapular: diagonal fold taken 1 cm below inferior angle of scapula.
   S3: abdominal: longitudinal fold taken at lateral distance of approximately 2.5 cm from umbilicus.
2. Compute body density (BD) with following formula:*
   $BD = 1.0973 - [0.000815(S1 + S2 + S3)] + [0.00000084(S1 + S2 + S3)^2]$
3. Compute percent body fat (%BF) with following equation:
   $\%BF = \{(4.57/BD) - 4.142\} \times 100$
4. Calculate minimal weight for 7%BF: 7%BF weight = 1− (%BF/100 × current weight)/0.93 = minimal weight in lbs
5. The above equations and formula should be used in conjunction with urine specificity gravity tests to ensure proper hydration in all wrestlers.

*From Lohman TG: Skinfolds and body density and their relation to body fatness: A review. Hum Biol 53:181-225, 1981, using modified constant suggested by Thorland WG, Tipton CM, Lohman TG: Midwest wrestling study: Prediction of minimal weight for high school wrestlers. Med Sci Sports Exerc 23:1102-1110, 1991.

- **NCAA-prohibited practices** include the following:
  - Use of laxatives, emetics, excessive food and fluid restriction, self-induced vomiting
  - Use of diuretics
  - Hot rooms, hot boxes, saunas, steam rooms, and vapor impermeable suits
  - Artificial means of rehydration (e.g., intravenous fluids)
- **State scholastic weight certification programs**
  - Each state determines its own weight certification process to determine how wrestlers qualify for a given weight classification for the duration of the season.
  - The majority of state scholastic wrestling associations have mandatory weight certification programs, and most use the National Wrestling Coaches Association's optimal performance calculator to determine MWW.
- Those seeking clarification of the **NCAA weight-management program optimal-performance system** may call or write to the NCAA Wrestling Committee liaison to the NWCA for the NCAA Weight-Management System: Ron Beaschler, 525 South Main St., Ada, Ohio 45810; phone (office): 419-772-2453; cell: 567-674-5133; fax: 419-772-3079; email: r-beaschler@onu.edu.

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Swimming and Diving

62

Martha I. Pyron

SWIMMING

General Overview

- One of the largest recreational and competitive sports in the United States.
- U.S. Swimming registered 280,000 athletes in 1999; 287,480 in 2006.
  - Provides liability and accident insurance for club members and coaches.
  - Requires coach education, training, and certification in first aid, CPR, and safety training.
  - Keeps records of injuries of its members.
- Swimmers span all ages from water babies to elderly.
- Competitive swimming starts around age 8 and continues through college. Masters level starts beyond the age of 25.

Events in Competitive Swimming

- Divided by sex and child's age with 2-year increments, and span from sprint, middle distance, to endurance events.
  - Freestyle: 50 m, 100 m, 200 m, 400 m, 800 m, 1500 m, 50 yd, 100 yd, 200 yd, 500 yd, 1000 yd, 1650 yd
  - Butterfly: 100 m and 200 m
  - Backstroke: 100 m and 200 m
  - Breaststroke: 100 m and 200 m
  - Individual medley: 200 m and 400 m
  - Medley relay: 200 m, 400 m
  - Freestyle relay: 200 m, 400 m, 800 m
- Swims take place in a 50-meter pool

Warm-Up, Cool-Down, and Stretching

Stretching: Swimmers have traditionally stretched each other before competition. Care should be taken to avoid overstretching, which may lead to hypermobility and loss of strength in the full range of motion.

Warm-Up: Start warm-up on deck to loosen muscle and increase heart activity; then move to water warm-up. Wear warm-up suit while waiting for event. Warm-up should be repeated before each event, especially if there is a significant time lapse between.

Rub Downs: A traditional treatment that is best performed in the hands of practicing therapists.

Swim Down: Valuable cool-down used after an event to avoid tightening of muscles. Usually occurs in adjourning pool.

Physiology of Swimming

Body Composition

- Swimming rewards extreme upper extremity strength.
- Successful swimmers are usually tall, lean, with long limbs and wide shoulders, and with a large muscle mass in their middle and upper bodies.
- Elite swimmers tend to have longer arms and larger hand surface areas.
- Freestyle and backstrokers tend to be the tallest and have greatest muscle mass.
- Breaststrokers are usually the shortest.
- Better performance is not solely because of body type, but more likely due to better application of forces in the water.

Flexibility

- Elite swimmers are more flexible around the shoulders and ankles.
- Females more flexible than males.
- Breaststrokers more flexible ankles (inversion and eversion), and greater lateral hip rotation.
- Butterfly swimmers have greater back and shoulder flexibility.

Body Fat Considerations

- Percentage of body fat of both elite male and female swimmers is lower than average.
  - 15% to 20% for males.
  - 20% to 25% for females.
  - Low percentage of body fat appears to have little effect on swimming performance.
  - Muscle strength and power play a more important role.

Strength and Power

- Ability to maintain a high percentage of peak power throughout the swim is related to swimming technique and mechanical efficiency.
- Anaerobic capacity of swimmers appears to be larger and involve smaller rates of adjustments.
- Aerobic capacity is a smaller factor in swimming performance than anaerobic capacity.

Limitations to Optimal Swimming Performance

- Energy metabolism
  - Prerace muscle glycogen depletion
  - Reduction of intramuscular adenosine triphosphate (ATP) and phosphocreatine (PCr) stores
  - Limitations in the rate of energy production and use
  - Disturbance of muscle function related to intramuscular pH
- Cardiopulmonary response
- Body fluid and thermoregulation
  - Fluid shift from gravitational effects causes a renal diuresis and hemoconcentration
  - Hypothermia is caused by cold water; hyperthermia comes from energy expenditure.
- Biomechanics, hydronamics
  - Form drag is dependent on body position; the more horizontal the less drag.
  - Wave drag is turbulence at the water surface created by the moving swimmer.
    - Waves rebound off the sides and bottom of the pool.
    - Deeper pools will have less rebound and therefore less wave drag, and provide a faster swim.
  - Frictional drag is from hair, or clothing. Less significant problem, but minimized by shaving, special body suits.

Ways to Optimize Speed

- Streamlining: mimicking a torpedo.
- Sculling: moving the hand in an oblique angle to the direction of travel, as in a propeller.
- S pattern of hand pull: pushing off still water creates more force.
- Drafting: swimming just behind the wave of the front swimmer in the next lane causes a positive draft effect.

Training

- $\dot{V}O_2$ max has been shown to reach a peak rate of development at 15.5 years.
  - Anaerobic profiles reach peak development at 16.5 years.
  - Training would seem to show benefit more greatly after these peaks are obtained.

- Overtraining
  - Low carbohydrate diets can lead to chronic muscle fatigue. Marked by decreased urinary norepinephrine and decreased plasma glutamine 2 to 4 weeks *before* fatigue develops.
- Periodization, progression, adaptation, recovery, and taper techniques are of benefit.

Facilities and Protective Equipment

- Pool configuration
  - Governing bodies specify markings, blocks, and lane lines.
  - Water temperature, acidity, and chlorine should be monitored.
  - Diving boards and water depth are regulated for divers.
  - Pool depth regulated for safety, warm-up, and competition.
- Protective equipment
  - Swimmers may use goggles to protect eyes from chlorine.
  - Rubdown oil is allowed.
  - Caps help reduce friction with the water.
  - Shaving the body and/or head also reduces friction.
  - Body suits may reduce friction as well.
- Safety issues
  - Each club, pool, or swimming organization needs to set safety standards for use of pool and for competition.
  - Designated lanes for club events and sprinters.
  - No diving unless supervised.
  - Block starts only in specified depth or deeper water.
  - No interference during competition; clear the pool before events and no jumping into the pool with a race in progress.
  - Use certified officials.
  - Lifeguard personnel should be present for competitions.

Injury Patterns

- Most are overuse injuries: develop during training and are related to changes in stroke, coaching, or improper technique.
  - Increased flexibility enables the swimmer to achieve a greater range of motion during the arm stroke but may predispose the swimmer to injury.
  - Fatigue leads to changes in stroke and improper technique.
- Acute injuries from U.S. Swimming 1999 and 2006 statistics:
  - There were 879 accidents in 1999; 898 accidents in 2006 (per approximately 280,000 swimmers).
  - Injuries spread fairly evenly over ages 8 to 18 at around 20% per each 2-year interval, with fewer injuries under age 8 and over age 18, and more injuries in the 11 to 12 age group.
  - 41% minor injury with treatment, 37% minor injury without treatment, 19% major cut or injury, 3% broken bone.
  - 38% to 45% in water, 23% to 30% on deck, 6% to 7% in locker room, 4% on blocks.
  - 49% to 52% during meet, 26% to 28% at practice, 8% to 13% during warm-ups.
  - 28% to 30% leg or foot, 14% to 19% hand/arm, 16% head or neck, 11% to 12% ENT or mouth, 5% knees, 1% to 3% internal.

Catastrophic Injuries

Neck Injury

- Avoid with low-entry angle dives and steering-up techniques in the water.
- Strive to surface in as short a distance possible by maximizing flight and having a low-angle entry dive.
- Water depth must be at least 4 feet for platform takeoff dive.
- Ten-year study in Greece showed that recreational diving accounted for 2.6% of all spinal cord injury.
- More likely in young male, during summer months.

Death in Competitive Swimming

- Drowning
  - Competitive swimmers trying to maximize breathholding technique.
    - Develop hypoxia from dropped PaO_2.
    - Slower rise in $PaCO_2$ causes a slower rise in the drive to breathe, resulting in unconsciousness and death.
  - Laryngospasm from inhaling water.
  - Diving reflex when swimming at the bottom of the pool after hyperventilation.
    - Leads to bradycardia and peripheral vasoconstriction.
    - Supraventricular arrhythmias may follow.
- Concomitant viral myocarditis predisposes to arrhythmia from hydrocution.
- Arrhythmia from cold water immersion especially in the setting of long QT interval.

Swimmer's Shoulder

- Most common orthopedic ailment for swimmers and most common reason for missed training.
  - Training 20 to 30 hours a week, may add up to more than 1 million shoulder revolutions a year.
  - Up to 30% of swimmers in season develop shoulder pain.
- Usually a combination of rotator cuff pathology and shoulder laxity.
- Includes injuries to the biceps tendon, rotator cuff, upper trapezius, rhomboids, deltoid, and serratus anterior. Subluxation, synovitis, capsulitis, and arthritis also occur. Acute ligament injuries are rare.
- Young swimmers at risk for proximal humeral epiphysiolysis similar to that seen in throwing athletes.
- More commonly seen in sprinters, and swimmers specializing in freestyle, butterfly, and/or backstroke. Usually occurs after 6 to 8 years of training (Fig. 62-1).
- Swimming aids are known to aggravate symptoms (hand paddles, drag swimsuits).
- Shoulder pain may be referred from other areas.
  - Disc dysfunction and spondylolysis at vertebrae C5, C6, C7
  - Brachial plexus or vascular impingement
  - Tumors
  - Infections of the bone or joint

Rotator Cuff

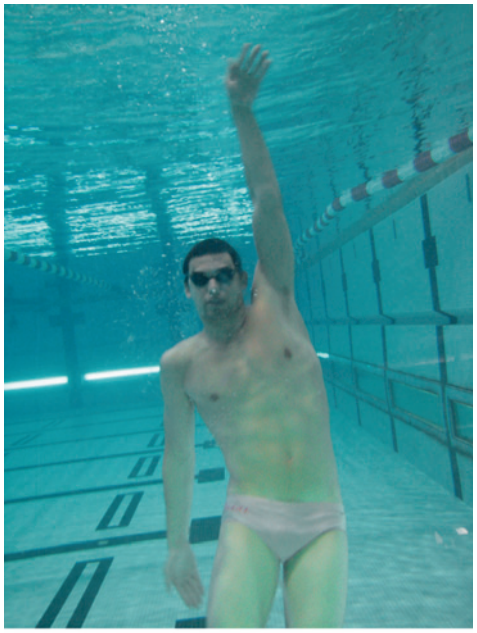
- Microtrauma or overuse syndrome leading to impingement and secondary rotator cuff tendonitis (Fig. 62-2).
- May be related to global laxity.
  - Rotator cuff centralizes the fulcrum of the joint and maintains contact of the humeral head against the glenoid articulation.
  - Overuse of the rotator cuff occurs as it tries to maintain joint alignment in the face of laxity.
- Acromioclavicular (AC) joint spurring contributes to impingement.

Multidirectional Instability

History: Pain located at the acromion or over posterior capsule; associated with overhead activities; symptoms similar to impingement syndrome; flexibility required of swimming may select those with shoulder laxity to the sport.

Muscular adaptations of swimming: Infraspinatus and teres minor (external rotators), and the abductors are relatively weak compared to the shoulder adductors and internal rotators. This may predispose the lax shoulder to tendonitis of the posterior shoulder, labral tears, subacromial impingement, or biceps tendonitis.

At-risk behavior includes: High-resistance upper extremity weight training, excessive stretching activities, including buddy stretching, and hand paddle use.



Backstroke arm motion shown underwater for clarity of picture.

Arm motion in butterfly stroke.

Figure 62-1 Swimmer's Shoulder.

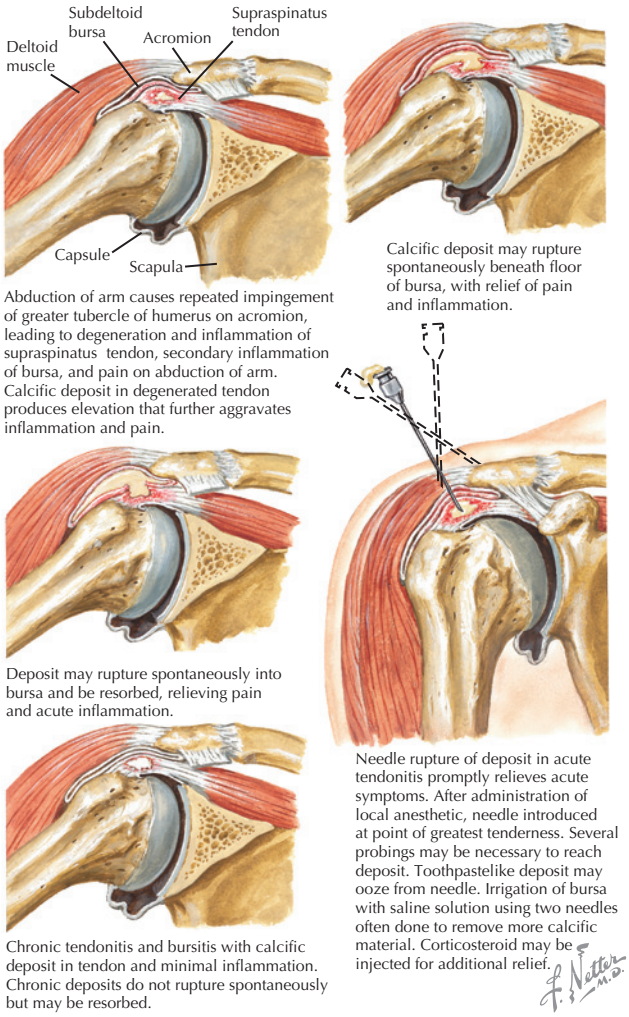


Figure 62-2 Shoulder Impingement and Tendonitis.

Physical examination: Tenderness over acromion, positive impingement tests (Hawkins, Neer), instability to anterior and posterior stresses with reproduction of symptoms.

Radiographs: Normal x-ray. Magnetic resonance imaging (MRI) or MRI arthrogram may show tear of the glenoid labrum or an abnormal rotator cuff.

Treatment:

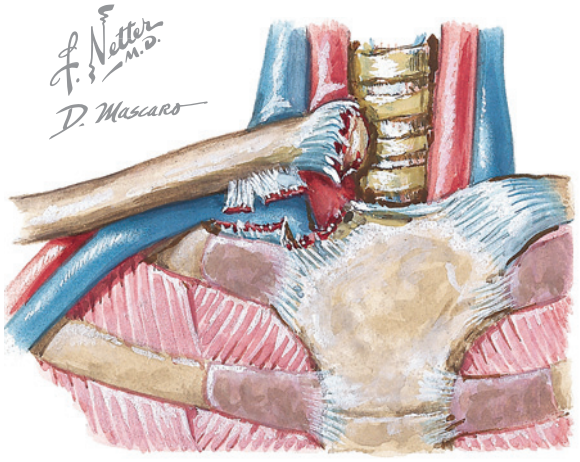
- **Nonsurgical**
  - Strengthening the rotator cuff muscles to increase the stability of glenohumeral joint.
    - Strengthening of the surrounding musculature will not prevent subluxation or dislocation.
    - Perception of stability is enhanced with rotator strengthening.
    - Remedial training of the external rotators and abductors may help prevent injury.
  - Strengthening of scapular stabilizers including the serratus anterior, trapezius, and rhomboids.
  - Decrease inflammation.
  - Adjust stroke technique.
  - Modification of activities and training volume.
- **Surgical**
  - Arthroscopic debridement of the torn or redundant glenoid labrum.
  - Acromioplasty to relieve impingement symptoms. This treatment alone will fail if laxity is the cause of the impingement.
  - There is no widely acceptable surgical solution to multidirectional laxity of the shoulder joint.
  - Capsular shrinkage procedures may help the laxity in the short term, but long-term follow-up studies are lacking. Alteration in the collagen might predispose the tissue to even further laxity in the future.

Subluxing Sternoclavicular Joints in Butterfly Stroke

See Figure 62-3.

Thoracic Outlet Syndrome (TOS)

- Entrapment of the neurovascular structures in the area of the neck (Fig. 62-4).
- May be due to a cervical rib, compression against the first rib, or from the scalenes and pectoralis minor musculature.
- Symptoms vary widely from neurogenic to vascular or both.
- Subclavian vein thrombosis associated with TOS requires an urgent need for vascular evaluation, anticoagulation, and possible surgical intervention to prevent embolism.
- Chronic or late detected thrombosis may lead to vascular scarring and permanent vascular changes, which may not respond well to surgical intervention.



Posterior dislocation of sternoclavicular joint. Serious because of probable injury to trachea or vessels. Both posterior and anterior dislocations can usually be reduced manually or with aid of towel clip. Anesthesia needed.

Figure 62-3 Sternoclavicular Dislocation.

- Various provocative tests help with the diagnosis:
  - Wright's test: Hyperabduct the arm so the arm is brought over the head with the elbow and arm in the coronal plain. Have the patient extend and rotate the neck away from the arm and take in a breath. Check for changes in the pulse during this maneuver. Decreases in pulse strength is a positive result.
  - Adson's test: Find the radial pulse. Have the patient rotate the head toward the test arm and extend the neck. Laterally rotate and extend the shoulder. Have the patient take a big breath and hold it. Disappearance of the pulse is a positive sign.
  - EAST (Roos test): Abduct arm to 90 degrees, flex elbow to 90 degrees, extend the arm so the elbows are just behind the frontal plain, and externally rotate the shoulder. Then open and close the hands slowly for 3 minutes. If the patient is unable to keep the arms in the starting position, suffers ischemic pain, heaviness or weakness of the arm, or numbness or tingling of the hand, then the test is positive.
  - Military brace maneuver: Palpate the radial pulse. Then pull the patient's shoulder down and back. Absence of pulse is a positive sign.

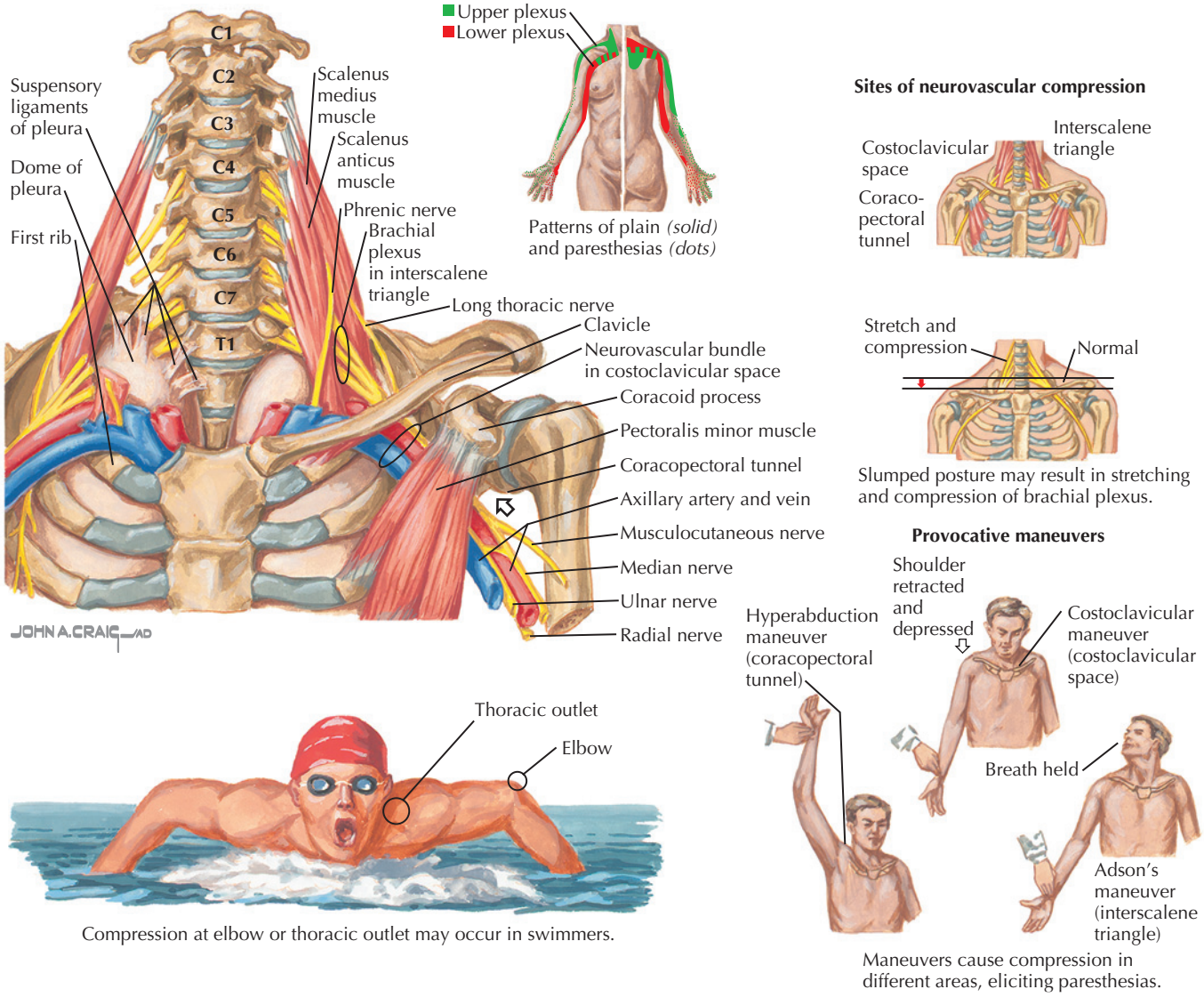


Figure 62-4 Thoracic Outlet Syndrome Anatomy.

- Labs/diagnostic tests; chest x-ray and thoracic outlet MRI to evaluate anatomical area of impingement. Urgent vascular studies such as D-dimer, magnetic resonance angiogram (MRA), or upper extremity Doppler ultrasound indicated if concern for thrombus.
- Double crush theory: Proximal nerve compression will render distal portions of the same nerve less tolerant to compression forces (e.g., carpel tunnel impingement is more likely to occur with thoracic outlet syndrome).
- **Nonsurgical treatment**
  - Anti-inflammatory medication.
  - Posture correction.
  - Muscle imbalance correction.
  - Stretching of neck musculature (scalenes, pectoralis major and minor, trapezius, levator scapulae, and sternocleidomastoid).
- **Surgical treatment**
  - Excision of the first rib.
  - Release of any soft tissue impingement.
  - Venous thrombosis treatment may include anticoagulation, thrombolytics, and angioplasty.

Hand Injuries

- From taking off the blocks or from striking the water.
- Fourth metacarpal (MC) spiral fracture in backstroker from push-off while holding onto pool edge.

Elbow Injuries

Ulnar nerve dislocation or compression from prominent medial head of triceps (Fig. 62-5).

Low Back Pain: Lumbar and Sacral

- Caused by excessive spinal load in flexion, gradual extension load, or weightlifting.
- Occurs in 16% of all swimmers; higher incidence during season.
- Can occur when diving off of blocks (Fig. 62-6).
- Especially common in butterfly swimmers (see Fig. 62-6).
- Follow up MRI studies show a higher likelihood of degenerative changes in swimmers seen after 5 years if back problems begin at a young age.
- Chronic strain of the paraspinous muscles of the lumbar spine, spondylolysis, spondylolisthesis, Scheuermann's kyphosis from turning at the end of the pool, improper technique, and diving into shallow water.
- Limitations for swimmers with injury.
  - Defective atlanto-occipital: restrict diving and water polo.
  - Disc syndrome: may need to restrict from diving or flip turns.

Head Injuries

- Lacerations and contusions from hitting the end of the pool; or collision with other swimmers can occur while drafting/turning.
- Watch for neurologic changes and signs of concussion.

Chronic Knee Pain

- Affects 25% of swimmers; mostly breast stroke and water polo athletes

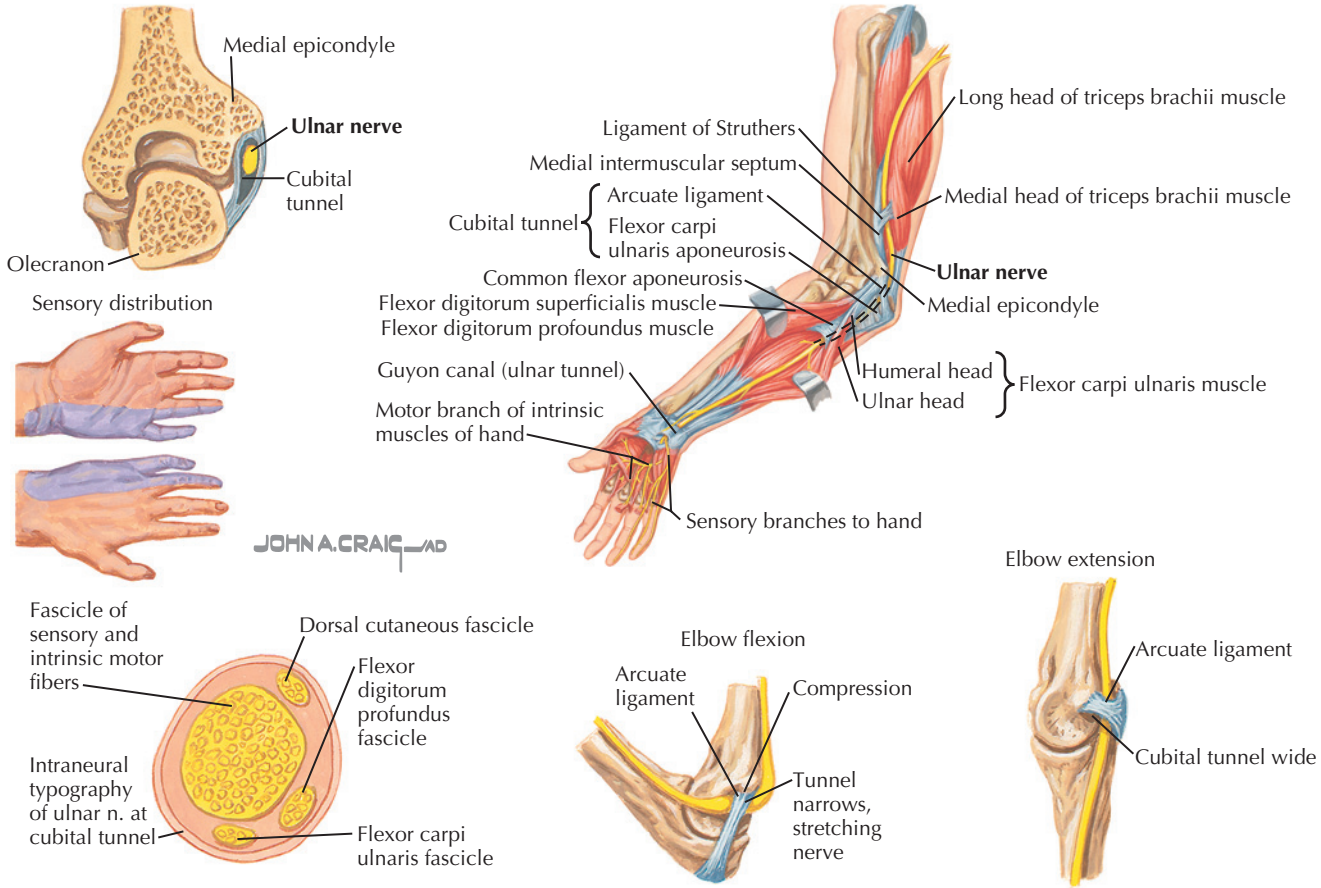


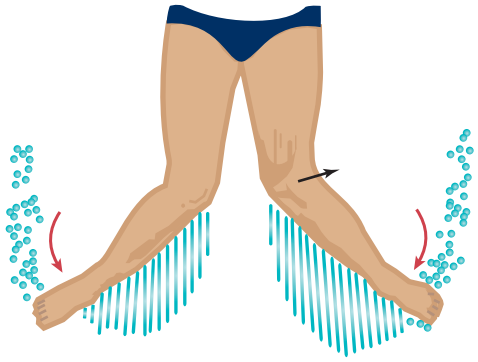
Figure 62-5 Compression of ulnar nerve. Ulnar nerve anatomy showing medial head of triceps capable of compressing.

Extreme lumbar flexion seen in this swimmer on the block predisposes the athlete to lumbar injury when takeoff forces applied.

Center swimmer seen underwater with back flexed for butterfly stroke; swimmer on the left shows back extended with head and arms out of water for this stroke. Repetitive flexion and extension predisposed the athlete to lumbar back problems.

Figure 62-6 Low Back Pain.

Correct alignment for breast stroke kick with hips held in closer adduction.



Lateral tracking of patella and/or medial knee stress with abnormal kick motion.

Figure 62-7 Knee Pain Associated with Kick.

- Chondromalacia or patellofemoral pain
  - Repetitive quadriceps contraction in the flutter kick
  - Push-off from the wall
- Medial patella facet pain or medial joint synovitis
  - Associated with abnormal whip kick.
    - Hips held in wide abduction as the hips and knees were flexed.
    - Increased hip flexion during the recovery phase.
    - Ankles are dorsiflexed and tibia externally rotated while the hips and knees are flexed.
    - Thrust phase of the kick ends with the legs in wide abduction and the knees fully extended.
    - Legs not pulled together at the termination of the kick.
    - Mechanics result in the patella being pulled laterally.
    - Normal whip kick shown in Figure 62-7.
    - Abnormal motion depicted in Figure 62-7.
  - Treatment
    - Kick modification.
    - Rest, nonsteroidal anti-inflammatory drugs (NSAIDs), ice.
    - Quadriceps strengthening, physical therapy, patellar brace.
    - Surgery: arthroscopic debridement, lateral retinacular release, thermal shrinkage or medial retinacular overlap.
- Chronic medial collateral ligament sprain
  - Medial retinaculum or gracilis tendon may also be involved.
  - Due to repetitive overuse.
  - High valgus load during the thrust of the kick.
  - Symptomatic swimmers usually have an abnormal kick.
    - Legs are abducted widely as the hips and knees flex during the recovery phase.
    - Knees reach full extension while the legs are still widely abducted.
    - Legs then pull forcefully together to complete the kick.
    - This results in higher valgus loads on the knee.
  - Treatment includes kick modification, physical therapy.
- Symptomatic medial synovial plica
- Tendonitis or bursitis of the pes anserine
- Patellar tendonitis
- Apophyseal injuries: Osgood-Schlatter, Sinding-Larson-Johannson
- Osteochondritis desiccans
- Discoid lateral meniscus

- Iliotibial band tendonitis
- Lateral patellar subluxation

Ankle Injuries

- Overuse from flutter kicks.
- Extensor retinaculum injury.
- Change stroke pattern for treatment.

Groin Injuries

- More common with the breast stroke (42.7% unable to train at some point due to groin injury).
- More likely to occur if swimmer kicks with wide abduction of the hips during the whip kick.
- Overuse or sudden stress on iliopsoas.
- Surgery may be needed.

Nonorthopedic Injuries

- **Exercise-induced asthma**
  - More common in swimming than once thought.
  - Reaction to chlorine and/or exercise.
  - Parasympathetic drive encouraged during immersion.
  - Coughing or wheezing after exercise.
  - Treat with inhaled beta$_2$-agonists prior to exercise and/or longer acting preventive medication.
  - Water sports usually less asthmagenic because of the lower pollen count over water, higher hydrostatic pressure on chest, and reduction of CO_2 from hyperventilation due to controlled breathing.
- **Otitis externa**
  - Swollen external auditory canal.
  - Treat with combination antibiotics and steroid drops.
  - Prophylaxis with products such as Swim Ear to keep the ear dry after swims.
- **Barotrauma**
  - Diving 3 feet or more below the surface can cause barotrauma.
  - Tympanic membrane can rupture with transudation of fluid into the middle ear.
  - Perilymph fistula or rupture of round or oval window.
  - Otitis media and acute mastoiditis can result.
- **Perforated ear drum**
  - Avoid diving, and swimming in pond water. Keep ear dry.
  - Earplugs and bathing caps help.
  - Ears with tubes are considered perforated.
  - Treat infection with combination antibiotic and steroid drops.
  - Follow closely. Ear should be protected from water until perforation has healed, which could take only days to 6 weeks or more. Consider surgical skin graft over perforation site if not resolving spontaneously.
- "Foot strike" anemia from striking the water.
- Skin reaction to chlorine may aggravate underlying skin conditions.
  - Atopic dermatitis
  - Ichthyosis
  - Contact dermatitis
- **Pool palms (friction dermatitis)** from repetitive contact to rough pool surface.
- **Green hair**
  - Copper-based algicides deposited in hair matrix.
  - Controlled with frequent shampooing or hydrogen peroxide bleaching.
- **Urticaria**
  - Aquagenic urticaria: results from water contact with skin sebum, which provokes release of a toxic substance that leads to mast cell degranulation.
  - Cold urticaria: cholinergic reaction.
  - Solar urticaria.
  - Exercise-induced urticaria: can be as severe as anaphylaxis.
    - Associated with pre-exercise ingestion of a specific food (e.g., shellfish, raw celery, peaches, grapes, wheat, or cabbage).
    - Exercise causes an antigen-antibody complex to induce the release of inflammatory mediators.
  - Treatment: pre-exercise antihistamines and injectable epinephrine standby.
- **Hot-tub folliculitis**
  - *Pseudomonas aeruginosa*–caused rash of mostly bathing suit covered areas with palms and soles spared.
  - Resolves spontaneously after 7 to 10 days.
  - Treat the hot tub water.
- **Athlete's foot:** Restrict barefoot from pool deck or showers to avoid exposure to others, and treat topically with antifungal medication.
- **Warts:** Hyfrecation for quick recovery or use topical treatment. Some suggest restriction from pool until healed. Restrict barefoot from pool or showers to avoid exposure to others.
- **Eye injuries**
  - Conjunctivitis: Goggles to prevent irritation, treat if infected. Ophthalmic cromolyn sodium helps with symptoms, but one may need steroid ophthalmic drops in severe cases.
  - Complications with glaucoma if high goggle negative pressure.
    - Goggle negative pressure can reach 44 mm Hg.
  - Corneal injury from anti-misting agent in goggles.
  - Eyelid masses from goggle pressure.
- **Lacerations:** repair and protect from infection
- **Hypothermia:** more common in long-distance swims
- **Sunburn, sunstroke, heat exhaustion**
- **Skin cancer:** Proper use of sunscreen helps protect the athlete.
- **Dehydration and malnutrition**
- **Gastroenteritis:** Can be caused by *Shigella*, *Vibrio*, rotavirus, *Giardia*. Have pool tested if epidemic occurs among swimmers.

DIVING

General Overview

Approximately 12,000 athletes register with U.S. Diving each year.

Events

- Springboard: 1 meter and 3 meter
- 10-meter platform
- Synchronized diving: 3 meter and 10 meter
- Variety of dives required: forward, back, reverse, inward, twisting, and handstand dive
- Optional stance: straight, pike, tuck, and free (combination of others)

Injury Pattern

- No gender differences.
- More experienced divers and older divers have higher injury rates—due to higher risk dives and overuse injury.
- Take-off phase: patellar tendonitis, quadriceps tendonitis, patellar-femoral compression syndrome, posterior tibialis and Achilles tendonitis.
- Flight or midair: shoulder instability with spin moves, long head of biceps tendon, spine overload.
- Entry is the most common time for injuries.
  - Stress fractures, sprains, strains of the wrist, and triceps tendonitis.
- Platform dives: 10-meter platform entrance velocity approaches 35 mph.

Cervical Spine Injuries

- Occur in recreation diving from lack of formal training, inadequate water depth, inadequate supervision, and alcohol ingestion.
- Competitive diving
  - In more than 100 years of competition in the United States, no fatality from cervical injury has occurred. Only two deaths worldwide.
  - One reported case of cervical instability at vertebrae C5-C6 in college diver.
    - Injury occurred upon impact with water.
  - Overuse injuries from repetitive axial loading.
    - No degenerative changes associated.
    - Sprains and strains are usually related to twisting and arching in midflight and entry.
    - Brachial plexus stretch injuries.
- **Fatal head injuries:** Only two reported and both occurred in a reverse 3½ summersault tuck dive from the 10-meter platform.
- **Carotid artery dissection** from 3-meter springboard dive, landing head first, or landing with neck twisted to side.
- **Concussion** from repetitive diving:
  - One study showed no neuropsychological changes from repetitive dives at the 1- and 3-meter height. Other heights not studied.

Lumbar Spine Injuries

- Anterior segment: vertebral body, vertebral end plate, and intervertebral disc.
  - Vulnerable to compression forces and increased load: press phase on the board, entry, or trunk flexion in flight.
  - Combination of flexion or rotation with loading most likely to end in disc herniation.
  - More commonly injured in springboard dives than in platform dives.
- Posterior segments: facet, pars interarticularis.
  - Subject to overload with rotation and maximum lumbar extension.
    - Takeoff for back or reverse dives.
    - Entry for a back rotating dive short of vertical.
    - Entry for front rotating dive beyond vertical.
  - Spondylolysis, spondylolisthesis, "kissing spines" (interspinous ligament pseudobursal formation and ossification leading to osteophytes on adjacent spinous processes), and lumbar facet arthropathy are all possible.
  - Segmental hypermobility in response to segmental hypomobility is frequently the cause of middle and low back pain in divers.

Shoulder Injuries

- Precipitated by multidirectional laxity and weakness of the scapular stabilizers.
- Repetitive microtrauma and overuse pattern is the most common.
  - Repetitive impact, swimming entries, and saving short dives.
  - Laxity progresses to instability and possibly to anterior capsule or SLAP (superior labrum from anterior to posterior) lesions, Bankart lesions, humeral avulsion of glenohumeral ligaments (HAGL), and labral splits.
  - AC arthritis, and proximal triceps tears also occur.
  - More experienced divers more likely to have instability.
- Traction tendonitis from shoulder laxity can affect the supraspinatus, infraspinatus, teres minor, and long head of the biceps.
- Subacromial bursitis.
- Supraspinatus partial tears.
- Scapular stabilizers: malposition of the scapula due to weak serratus anterior, lower trapezius, or rhomboids predisposes the athlete to anterior-inferior subluxations.
- Stress fracture of the clavicle.

Elbow Injuries

- Occur upon impact with the water when the elbow is locked in extension.
- Result in tears of the medial collateral ligament, strains or tears of the triceps, and ulnar neuritis from medial collateral ligament laxity.
- Olecranon stress fracture.

Wrist Injuries

- Occur from repetitive impact and forced dorsiflexion on flat hand technique entry (Fig. 62-8)
- Scaphoid stress fracture
- Carpel instability
  - Volar subluxation of the lunate
  - Posterior and ulnar subluxation of the scaphoid
- Dorsal impaction syndrome
- Dorsal ganglion cyst
- Flexor carpi ulnaris tendonitis
- Sprains

Hand Injuries

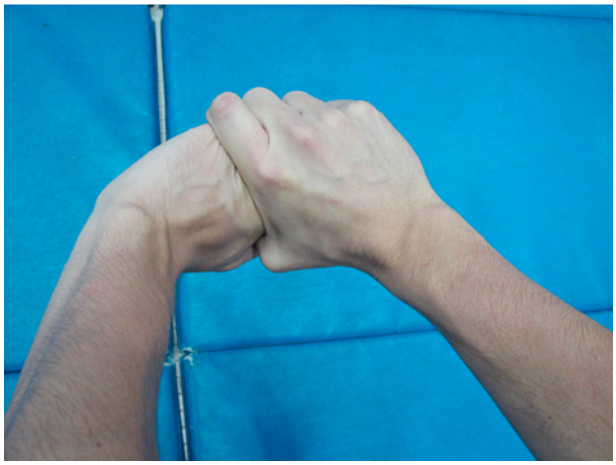
- Ulnar collateral ligament of the thumb
- Contusions and fractures of the metacarpals and phalanges
  - From striking the board
  - In younger swimmers from hands striking the head upon water entry; due to lack of muscular strength to maintain elbow extension

Lower Extremity Injuries

- Mostly associated with jumping
- Tendonitis: patellar, quadriceps, posterior tibialis, Achilles, and foot and ankle
- Maltracking patella and/or patellar compression syndrome
- Tibial periostitis and/or stress fractures of the tibia
- Fractures of the fifth metatarsal: foot lands in an awkward position during the approach or on landing from the hurdle
- Fractures of metatarsals and phalanges: striking the board during flight
- Tibial tuberosity avulsion fracture in a younger athlete
- Fracture of the subtalar joint in springboard dive

Nonorthopedic Injuries

- Perforations of the tympanic membrane, otitis externa
- Vestibular abnormalities from repetitive spins
- Retinal detachment, ocular contusion, optic nerve avulsion and corneal abrasions



Flat hand water entry postion on a dive (demonstrated on dry land).

Figure 62-8 Wrist Injuries in Diving.

- Pulmonary contusion: usually self-limiting within 48 hours
- Pneumothorax
- Scalp lacerations
- Splenic rupture with underlying splenomegaly
- Dehydration and malnutrition

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Scuba Diving

63

George A. Morris

GENERAL PRINCIPLES

Overview

- Diving is an activity done in a different environment.
- Technology allows people to enjoy the underwater experience.
- It is estimated that there are more than 1 million dives in the United States each year.
  - People dive for various reasons including the ability to experience new surroundings, meet new challenges, job requirements, military duties, and environmental awareness.
- There are few competitions for diving.
  - Breath-hold diving for records
  - Spear fishing competitions

Types of Diving

- Breath-hold diving (also known as free diving or skin diving)
  - Snorkel is the main piece of equipment used and it is used only to minimize the effort of lifting the mouth out of the water for respiration. May use fins for propulsion.
  - Often done in conjunction with spear fishing and shellfish gathering.
- Recreational scuba (self-contained underwater breathing apparatus)
  - Allows enjoyment of unique environments.
  - Done in many locations throughout the world.
- Commercial, technical, and advanced recreational diving
  - A wide range of gases used for respiration depending on the nature of the mission.
  - Specialized training for wreck, cave, and exploratory diving.
  - Different gases and mixes being tested and different decompression techniques used.

Environments for Diving

- Multiple different underwater environments, each with different risks and techniques involved for the diver.
  - Open ocean water is the most common location for recreational
  - Fresh-water lakes and rivers
  - Ice diving
  - Extreme elevations (e.g., mountain lakes or the Dead Sea)

Physiology of Diving

- Pressure at the water surface is 1 ATA (atmospheres absolute).
  - Includes the weight of all the air pressing down on a person at sea level.
  - The pressure in Denver, Colo. (elevation of 5280 feet) is 0.8 ATA, but only 6 feet under saltwater (FSW) it will be 1.2 ATA.
  - Every 33 FSW increases the pressure encountered by 1 ATA.
- Saltwater is approximately 775 times more dense than air.
  - The increased density provides buoyancy to the diver.
  - Creates a sense of weightlessness which can lead to disorientation.
- Increased viscosity of water will increase the resistance to movement.
  - Requires about 13 METS (metabolic equivalents) to swim 1.3 knots (1 knot = 101 feet per minute), approximately 1.5 mph, compared to 3.3 METS to walk 3 mph on level ground.
  - This contributes to the need for increased physical fitness in the diver.
- Increased heat capacity of water
  - Requires approximately 1000 times as much energy to heat a volume of water as it does to heat a similar volume of air.
  - Conductive heat loss in water is 25 times faster than in air, which leads to increased risk of cold illness.
- The human body's response to aquatic environment
  - Air spaces will be subjected to increased pressure.
  - Thermoregulation can be a challenge but met with proper equipment.
  - Shivering can actually hasten cooling in the water because of increased peripheral blood flow from muscle activity, with further loss of heat from the increase of warm, "core," blood now flowing through cold extremities.
  - Diving reflex leads to bradycardia, peripheral vasoconstriction, and shunting of blood to the core.

Physics of Diving

- Boyle's Law
  - As the pressure is increased on a volume of gas, the volume decreases and vice versa.
  - $V_1 \times P_1 = V_2 \times P_2$
  - As the diver descends, the air-filled flexible wall structures (e.g., lung, gastrointestinal tract) will have their volume decrease and they increase on ascent.
  - The liquid and liquid/solid organs (e.g., blood, bone, muscle, teeth, and organs) will transmit the pressures equally in all directions according to Pascal's principle, which states that pressure is distributed equally over surfaces and transmitted equally through compartments containing gas or liquids.
  - The air-filled rigid-walled structures (sinuses, middle ear, and face mask air space) will remain at surface pressure at depth due to their inability to change volume.
    - This pressure gradient is responsible for barotrauma injuries.
    - This gradient can be resolved by equalizing the spaces during depth changes.
    - The gradient change appears greatest near the surface.
- Dalton's Law
  - The total pressure exerted by a mixture of gases is equal to the sum of the pressures that would be exerted by each of the gases if it alone were present and occupied the total volume.
  - The air pressure at sea level (1 ATA) is composed of nitrogen (N_2) (0.79 ATA) and oxygen (O_2) (0.21 ATA), plus trace amounts of carbon dioxide, water vapor, and other gases
  - At greater depths, the air will be breathed in at the ambient pressure.
    - 33 FSW has a pressure of 2 ATA, which will be composed of N_2 (1.58 ATA) and O_2 (0.42 ATA).
  - This increases the partial pressures of the inspired gases and the body is sensitive to these changes.
- Oxygen
  - Our lungs and bodies can tolerate a wide range of oxygen pressure (0.158 ATA to 2 ATA) and still extract enough to meet the metabolic requirements without developing toxicity.
    - Below 0.158 ATA, the body will experience hypoxia and the diver will develop air hunger, fatigue, confusion, loss of consciousness, and death of tissues.
  - Brain cells are most sensitive, and can die if deprived of oxygen for as little as 4 minutes.

- Oxygen toxicity can occur if the partial pressure is too high for too long.
  - Effects can include nausea, disorientation, visual changes, and seizures that can be life threatening if experienced underwater
  - Enriched air mixtures have different concentrations of oxygen and different depth limits due to the increased partial pressures of oxygen.
  - The theoretical limit of 2 ATA of oxygen for 30 minutes is met at only 33 FSW when using pure oxygen.
- Henry's Law
  - The amount of gas that will dissolve in a liquid relates directly to the pressure (P) of the gas. Thus, $G_{(PD)}$ :: P where $G_{(PD)}$ is the gas physically dissolved in a liquid phase and :: stands for proportional.
  - As divers descend to greater depths and pressures, the number of molecules of the gas dissolved in their body tissues will increase.
  - Factors that can affect how fast the dissolution occurs include temperature, solubility coefficient, and metabolism of the gas.
  - Different tissues absorb and release the gas at different rates.
  - Tissue saturation eventually occurs if the diver stays at depth long enough.
  - Upon ascent, partial pressure decreases and gas will leave solution. If ascent is too rapid, bubbles may form in the local tissue or the bloodstream.
- Nitrogen
  - Usually considered an inert gas because it does not unite chemically with other substances in the body.
  - No typical effects at atmospheric pressures (0.79 ATA).
  - Nitrogen can interact with cells at increased pressures and lead to nitrogen narcosis.
  - The effects of nitrogen at depth are comparable to taking one alcoholic drink for every 50 FSW descended and at 750 FSW it has anesthesia-like effects. This effect is also called "rapture of the deep," and can cause confusion and strange behaviors.

Portions of the Dive

Surface: Swimming or wading at the surface prior to going underwater.

Descent: Involves changes in pressures of 1 ATA per 33 FSW. Need to have equilibration of pressure in air spaces.

Bottom time: Traditionally the amount of time spent at lowest depth; however, with the advent of dive computers it has evolved to the amount of time spent underwater. Activity at depth can change respiratory needs and risks of complications. Good buoyancy management can decrease the oxygen consumption and conserve the air supply.

Ascent: Risk of injuries because of changes in pressure and the release of absorbed gas back into local tissues and the bloodstream.

Surface interval: Allows the body to resume its usual physiologic status and normalize tissue gas concentration prior to the next dive. Complications or injuries encountered during the dive may not present until the diver is at the surface

Incidence of Dive-Related Injuries

- Difficult to determine because exact number of divers and dives performed each year is unknown.
- Decompression sickness (DCS).
  - Approximately 4 DCS events per 10,000 dives.
- 242 diving-related deaths reported to Divers Alert Network (DAN) in 2004.
  - 45% of the deaths occurred in divers with a BMI of 30 or greater.
  - Mean age of diving fatalities is 48 years old.
  - 45% of the deaths occur in divers with 1 year or less of diving experience and 25% in divers with more than 10 years of dive experience.
- 22 breath-hold diving deaths were reported.
- Incidence of overall drowning deaths in the United States is 1.2 per 100,000 persons. For comparison, the incidence of deaths from motor vehicle accidents in the United States is 15.4 per 100,000 persons.

Safe Dive Profiles

- Follow standard recommendations on dive tables or dive computer algorithms for duration of dives at various depths with decompression stops as needed.
  - United States Navy Air Dive tables
    - Based on rectangular or square profiles that assume the diver descends directly to the deepest depth and stays at that depth until returning to the surface
    - Designed for safety to minimize risks of DCS, oxygen toxicity, and nitrogen narcosis
- Dive computers are now very common and easy to take on a dive.
  - They allow flexibility in dive profiles by allowing the diver to be at various depths during different portions of the dive.
  - Potential for less calculation errors related to difficulty in using many of the tables.
  - May allow more dives per day.
  - Computers use different software programs to track cumulative nitrogen levels in different compartments during all portions of the dive.
  - Provides information on planning a dive profiles, safety stops, return to surface alarms, decompression times, or dive intervals.
- It is recommended that most sport scuba divers perform only no-decompression dives to minimize risks.
- No dive is 100% safe because of individual variation in conditions, health, physiology, and equipment.
  - Diving within your skill limits, equipment, and certification, makes diving safer.
  - Keep ascent rate below 30 feet per minute.
- Always dive with a buddy.

Basic Fitness for the Diver Is Important

- MET level needed for typical diving activities.
  - Average scuba and skin diving activities use 7 METS.
  - Remaining stationary against a 1-knot current can take up to 13 METS, which is similar to the level required to run between 7 and 7.5 mph.
  - Getting in and out of the water can require significant strength and coordination.
- Scuba diving is not a great way to improve fitness or lose weight; it is difficult to maintain levels of METS needed to improve fitness, and the restricted air supply limits time spent in aerobic exercise.

Guidelines for Diving

- Conditions that may disqualify a diver should generate a discussion regarding the risks versus benefits of recreational diving.
- Clearance for commercial, military, and technical divers is much more restrictive and current U.S. Occupational Safety and Health Administration (OSHA) and military guidelines should be reviewed.
- If there are any questions about medical conditions and diving, DAN is available for support, or contact a trained dive physician or appropriate specialist with knowledge of diving.

Medical Events during Diving

- Cardiac causes are implicated in about 40% of diving-related deaths.

- Important to evaluate underlying fitness for activities in a predive physical.
- A predive physical may only occur once in a person's lifetime so it is important to educate the diver about ongoing monitoring of his or her health.

SPECIFIC INJURIES

Pressure-Related Diving Problems

Barotrauma/Dysbarism

Ear squeeze: The most common pressure related injury.

Tympanic membrane (TM) rupture: Pain of the ear squeeze is relieved followed by a rush of cold water and dizziness.

Return to dive recommendations following barotrauma: Divers with middle ear symptoms may return to diving once the TM is healed or protected, hearing has improved, and the diver can equalize. Divers with perforation or round window or oval window rupture should not dive further because of the risks of permanent impairment.

Sinus squeeze: Pain and even bleeding.

Tooth squeeze: May occur if there is a small amount of air trapped under a filling or presence of a dental abscess.

Pneumothorax (PTX)

Description: Can become a tension pneumothorax (PTX) on ascent due to expansion of trapped air in pleural cavity (Fig. 63-1).

Return to dive: Consider no further diving if spontaneous. Other causes, three to six months with documented healing, normal appearance on imaging, and normal function.

Decompression Illness (DCI)

Description: A major concern of divers because it is a potentially avoidable event with possible serious consequences. DCI encompasses events that include decompression sickness (DCS) and arterial gas embolus (AGE) as well as other events that may be difficult to classify.

Etiology:

- Henry's Law is the main factor playing a role in DCI.
  - On-gassing concept
    - More molecules dissolve into tissues as a diver descends.
    - Different tissues have different rates of gas permeability
    - Fast diffusion—lung
    - Medium diffusion—blood and organs
    - Slow diffusion—joint capsules/ligaments
  - Off-gassing concept
    - The dissolved molecules need to diffuse back into the bloodstream and be transported to the lung for exchange/removal.
    - Equilibration takes different amounts of time in different tissues.
- Small venous bubbles are common during the ascent portion of a dive and they are generally filtered/exchanged without problems in the lung.
  - Problems can occur if ascent is too fast and large bubbles develop or bubbles enter the arterial side of the circulation.
  - Local effects of nitrogen bubbles in the tissue can affect skin, peripheral nerves, joints, connective tissue, and muscles.

DCI types: Can be divided into categories with some overlap possible.

- Type 1 DCS, mild: Characterized by one or more symptoms including:
  - Mild pains that resolve within minutes
  - Pruritus and skin paresthesias
  - Rash, may be violaceous, mottled, papular
  - Arthralgia and myalgias
  - Repeated DCS exposure in bone can lead to osteonecrosis
- Type 2 DCS, serious:
  - Pulmonary symptoms can include burning substernal pleuritic pain, nonproductive cough, severe respiratory distress.
  - Neurologic signs and symptoms can include focal spinal cord lesions with paresis, paresthesia, paralysis, loss of sphincter control, and truncal pain.
  - Symptoms do not follow radicular or dermatomal patterns and may change over time.
  - Cerebral type 2 DCS may present with headaches, visual changes, dizziness/vertigo, nausea, vomiting, tinnitus, hearing loss, focal neurologic deficits, and changes in mental status.
  - Hypovolemic shock, circulatory collapse, and thrombus formation can occur.

Presentation: DCS injuries have been reported up to 36 hours post dive. AGE is a catastrophic event caused by rupture of alveoli with leakage of air into the pulmonary venous system resulting in bubbles occluding the arterial circulation of the brain, heart, and other organs. The symptoms have an acute onset within 10 to 120 minutes of surfacing. Symptoms can begin with

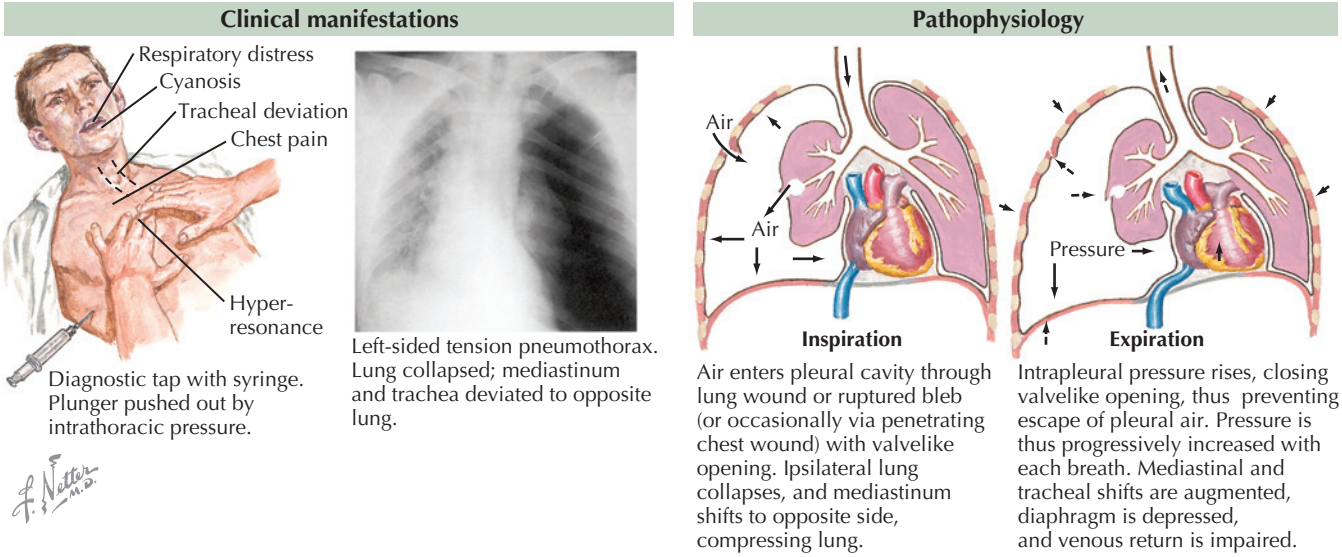

Figure 63-1 Tension Pneumothorax.

dizziness, anxiety, and headache and progress rapidly to loss of consciousness, seizures, shock, and death. Often difficult to differentiate AGE from central nervous system (CNS) type 2 DCS. Treatment is similar—recompression and oxygenation. AGE can occur with any dive. DCS injuries often involve longer, deeper dives in which tissue saturation occurs.

Prevention:
- Dive within tables/computer/conditions
- DCI has occurred in all types of dives including free-diving
- Slow ascent with stops as required

Risks of DCI :
- Dive profile that exceeds no-decompression limits for time, depth
- Rapid ascent, dehydration, exertion at depth
- Obesity, lung disease, cardiac septal defects, and patent foramen ovale
- Prior DCI

Treatment:
- Repressurization via hyperbaric oxygen treatment (HBOT).
- Dissolves gases back into the tissues and then gradually returns the patient to atmospheric pressures and air source.
- 100% O_2 during transport can displace nitrogen from the tissues and decrease symptoms and complications.
- Correct hypovolemia with intravenous fluids.
- Transportation and HBOT should begin during initial resuscitation if the diagnosis is suspected.
- Rapid resolution of symptoms with HBOT is the hallmark of DCI.

Return to dive recommendations following DCI: The diver should be evaluated for predisposing factors including equipment, dive profile, and medical causes. Medical causes should be evaluated and either corrected or decisions made regarding the advisability of further diving.
- Type 1 DCS—usually may dive in 2 days
  - Consider 2 to 4 weeks if symptoms prolonged, oxygen or recompression required
  - Joint involvement: 2 to 4 weeks
  - Paresthesias: 4 weeks
- Type 2 DCS with:
  - CNS involvement cleared with recompression: minimum of 4 weeks
  - Persistent or residual deficits: no further diving
- AGE: minimum of 3 months if all symptoms cleared and no medical causes identified

Loss of Consciousness Underwater (LOCU)

Description: Can occur for different reasons and is life threatening due to the individual's inability to protect his or her airway and return to the surface.
- Seizures
- Hypoglycemia
- Syncope
- Cardiac event, ischemia or arrhythmias
- Nitrogen narcosis
- Medication
- Alcohol or drug use
- Blackout caused by hypoxemia
  - Hyperventilation prior to breath-hold diving is common and results in a decreased level of carbon dioxide (CO_2). This leads to cerebral vasoconstriction and can reduce cerebral oxygen delivery.
  - Respiratory drive is primarily mediated by CO_2. Consequently, the diver may lose consciousness due to cerebral hypoxia before sensing a need to breathe or is too deep to get back to the surface in time
  - While ascending from a long and deep breath-hold dive, the lungs will expand and because of the partial pressure changes, O_2 may diffuse back into the lungs from the blood, causing hypoxemia with subsequent "shallow water blackout."
  - Carbon monoxide (CO) contamination can lead to hypoxemia despite adequate O_2.

Breathing/Air-Related Problems

- Air is the most common gas used and is about 21% O_2, 79% N_2.
- Oxygen toxicity can occur if pure oxygen is used or on prolonged deep dives with air.
- Nitrogen narcosis may occur on deep, prolonged dives.
- Nitrox is a gas with an increased percentage of oxygen (usually 32% or 36%) that may be used to prolong bottom time and decrease risks of nitrogen narcosis.
  - Has different risks including increased risk of oxygen toxicity and should be limited to a depth based on oxygen percentage.
  - Nitrox tanks should be specially labeled and not filled with air or vice versa.
- Heliox is a mix using helium as the inert gas, which can alleviate the risks of nitrogen narcosis.
  - Specialized support staff and equipment is needed so use of heliox is generally limited to commercial and technical divers.
  - Requires a longer decompression time.
  - Speech sounds like Donald Duck.
  - There is an increased risk of tremors, seizures, loss of consciousness, and even death if diving on heliox below 660 FSW.
- Trimix is a blend with oxygen, nitrogen, and helium, which also requires specialized equipment and training.
- Dirty air: If the compressor is set up incorrectly or malfunctioning, the air may be contaminated with carbon monoxide (CO) which binds to the hemoglobin molecule approximately 250 times stronger than oxygen.
  - Symptoms may first start during bottom time with headache, confusion, LOCU, and can lead to death.
- Bad tanks: The interior of tanks may expose the diver to contaminants via their air supply. Tanks can become rusted if the interior is exposed to moisture leading to a decreased partial pressure of oxygen inside the tank. Tanks should be inspected annually and pressure tested at least every 5 years. Tanks should be stored on their sides with about 500 PSI of gas in them to prevent exposure to moist air.

Marine Animal Exposures

- Different types of marine creatures are encountered underwater. These need to be treated with respect and caution to minimize environmental degradation and risk to the diver.
- Underwater animals may seem to be moving slowly or allow close inspection but they often can move very quickly.
- Bites from animals such as eels, rays, sharks, groupers, and barracudas can occur.
  - Risks may be increased if spear fishing or in a feeding zone.
  - Treatment should start with standard treatment for traumatic injuries. Remove the diver from the exposure while not risking your own life. Control bleeding and obtain treatment at the nearest trauma center. Clean the wound as able and use broad spectrum antibiotics that includes coverage of *Escherichia coli*, *Pseudomonas aeruginosa*, *Mycobacterium marinum*, *Staphylococcus aureus*, *Streptococcus* species, *Clostridium* species and *Vibrio* species.
- Jellyfish stings usually only cause discomfort but certain types can be fatal.
  - For most jellyfish:
    - Nematocysts on the tentacles are responsible for the envenomation.

- Local inflammatory response with pain and erythema is the main problem.
- Neurologic and respiratory depression has been seen along with nausea, vomiting, and abdominal pain from stings of certain species such as the Portuguese man-o-war.
- Initial treatment is to remove the individual from the exposure and gently remove the tentacles to prevent further nematocyst firing.
- Ice, flushing with saltwater or vinegar, meat tenderizer paste, or topical anesthetics are helpful; rinsing with alcohol may worsen the symptoms.
- Patients at risk for anaphylaxis should be monitored further.

- Box jellyfish, also called sea wasp, is another source of serious stings.
  - There are multiple species; the most venomous are found in the Indo-Pacific region and some related species are found in the Gulf of Mexico.
  - Stings from the multiple tentacles often occur in linear whiplike arrangements on exposed skin.
  - Immediate and severe pain is noted.
  - Episodes of hypotension, tachycardia, and respiratory distress may alternate with hypertension, bradycardia, and apnea.
  - Cardiogenic shock can progress quickly and death can occur within 10 minutes.
  - Treatment for these exposures needs to be started early and includes removing the tentacles and getting the diver out of the water, administering vinegar to the stings, supplying oxygen, cardiopulmonary resuscitation, and treatment for anaphylaxis.
  - Parenteral pain medications and steroids may benefit along with intubation, sedation, administration of antivenom as available, and vasopressor support in the hospital.
  - Delayed problems can include paralysis, pulmonary edema, abdominal pain, irritability, and localized skin necrosis.
- Sea bather's eruption is a reaction to thimble jellyfish larvae that can occur without the diver being aware of their presence because of their small size.
  - The larvae become trapped under the wetsuit or bathing suit and often discharge their nematocyst into the skin when they exit the water and are exposed to air or freshwater.
  - Effects can include erythema and pruritus.
  - Treatment can be started at the first sign of irritation and includes rinsing with saltwater, vinegar, 5% acetic acid solution, or using a paste with meat tenderizer.
  - Topical corticosteroids generally relieve the pruritus and erythematous rash.
- Coral abrasions, lacerations, and reactions to their chemical toxins may occur.
  - Wounds should be thoroughly cleansed to remove the foreign material.
  - Infections are common and may include *Vibrio* and *Erysipelothrix* species.
  - Dermatitis from the abrasion may persist for weeks.
- Envenomations from fish, rays, and sea urchins.
  - Stone fish, rays, and sea urchins typically reside on the ocean floor.
  - Shuffling your feet and wearing thick protective footwear can help minimize the risks.
  - Initial treatment should focus on elevation of the extremity and soaking in warm water or using hot packs because many of the toxins are heat labile.
  - Barbs and spines can be considered for removal depending on their location.
  - Antivenom for stone fish is available.

Dive Entry and Exit Injuries

- Low back pain can result from the use of heavy and awkward equipment.
- Getting in and out of the boat with equipment often requires the maximum exertion for a diver. Swells and waves may increase the risk of injury
- Shore dives have risks related to slipping on rocks or coral or being thrown into them by wave action; stepping on sea urchins, rays, or debris; wave trauma; and rip tides.

SPECIFIC CONDITIONS

Cardiovascular

Coronary artery disease (CAD): May allow diving if diver has a left ventricular ejection fraction (LVEF) greater than 50%, no abnormalities with max stress testing to at least 13 METs, no recent ischemic events, and no history of exercise-associated events. Should also wait at least 6 months following percutaneous treatment or coronary artery bypass graft (CABG) and completed cardiac rehabilitation.

Congestive heart failure (CHF): New York Heart Association (NYHA) classification II or greater should be advised against diving, or if LVEF is less than 50% and they require medications.

Hypertension: May allow diving if well controlled, no trouble with exertion, and medications are well tolerated with few side effects. There are rare cases of immersion pulmonary edema in some patients with uncontrolled hypertension, heart disease, and/or renal disease.

Murmurs, valvular abnormalities:

- Atrial septal defect (ASD) should be a contraindication to diving due to risk of right to left shunting during ascent.
- Ventricular septal defect (VSD) should be a diving contraindication if there is any evidence of right to left shunting or arterial hypoxemia. May consider diving if there is a small membranous VSD without shunting.
- Patent foramen ovale (PFO) is often undiagnosed and asymptomatic.
  - Can increase the risk of DCI because of right-to-left bubble shunting during Valsalva maneuvers or ascent.
  - If an individual experiences DCI during what is felt to be a safe dive profile and has a PFO, he or she should be disqualified from further diving.
- Aortic stenosis (AS): diving is contraindicated if severe or if the diver has ECG abnormalities, poor blood pressure response to exercise, or is symptomatic.
- Mitral stenosis (MS): diving is contraindicated if severe, symptomatic, or with an elevated pulmonary artery pressure.
- Mitral valve prolapse (MVP) and mitral regurgitation (MR) are not contraindications unless associated with arrhythmias, dilated left ventricle, pulmonary hypertension, or syncope.
- Artificial prosthetic valve patients should generally be disqualified from diving because of anticoagulation medications.
- Hypertrophic obstructive cardiomyopathy (HOCM) should be a disqualification.
- Marfan syndrome: disqualified from diving.

Arrhythmias: Long QT syndrome is a contraindication to diving. Patients with symptoms related to brady- or tachy-arrhythmias should be disqualified from diving. Sport diving may be considered on a limited and individualized basis if symptoms are controlled, exercise tolerance is adequate, and other factors are taken into consideration. Ventricular arrhythmias should disqualify a diver especially if associated with structural abnormalities or reduced cardiac function due to risk of sudden death. Patients with pacemakers may be allowed limited diving following an appropriate post-operative period, exercise testing, discussion with the cardiologist, and remaining below the depth limits to which the pacemaker has been tested. Diving

with an implantable cardio defibrillator (ICD) is not recommended.

Syncope: Contraindication to diving if triggers unknown or if triggers may be encountered during a dive or during dive-related activities.

Peripheral artery disease (PAD): Claudication can be worsened during a dive because of vasoconstriction from the dive reflex. This may not increase risks for serious complications during a dive but may increase symptoms that can be mistaken for DCI.

Pulmonary

Asthma: New research shows this is not an absolute exclusion from diving. Patients with mild, stable asthma may be allowed to dive if they have normal lung function, do not have current wheezing or symptoms, and are able to achieve appropriate levels on exercise testing. Evaluate the individual's asthmatic triggers—a cold trigger is potentially dangerous for divers. The risks associated with asthma and diving are related to air trapping with the risk of alveolar rupture and AGE.

Chronic obstructive pulmonary disease (COPD), chronic bronchitis, emphysema: The pulmonary anatomic changes and alterations in function generally make COPD patients unable to tolerate the exercise capacity needed to dive. Diving is generally contraindicated because of the decreased lung function and anatomic abnormalities that place the diver at increased risk of AGE, PTX, DCI, or drowning.

Pneumothorax: Spontaneous, has a higher incidence in tall, thin, young male smokers and up to 40% may have a recurrence. This is a contraindication to further diving. Traumatic pneumothorax should have no diving for minimum of 3 to 6 months and absolute contraindication to diving if pleurodesis is performed. If dive related, no diving for 3 to 6 months if cause can be determined and healing and normal lung function assured.

Pulmonary embolism: No diving while on anticoagulants and no diving for 6 to 12 months until normal lung function and anatomy can be determined. Concomitant hypercoagulable state may represent a contraindication

Central Nervous System (CNS)

Seizure disorder: Risk of death if a seizure occurs underwater; seizure medications may have undesired side effects and increase risk of nitrogen narcosis. May dive if stable and seizure free and off medications for 5 years. Febrile seizures as a child are not a contraindication.

Craniotomy: Is generally a contraindication to diving.

Ventricular shunts: May allow limited diving but should consider availability of local neurosurgical services in the dive area if shunt malfunction occurs.

Concussion: Avoid diving while symptomatic.

Cerebrovascular accident, transient ischemic attack (CVA/TIA): Need to have appropriate recovery and rehabilitation time. Functional limits should be evaluated in light of specific needs of diving and whether equipment can be modified. If patient is cleared to dive may need to consider having two dive buddies. Presence of carotid stenosis greater than 70% may disqualify a diver.

Motor/nerve disorder (such as amyotrophic lateral sclerosis (ALS) or multiple sclerosis (MS)): May consider allowing to dive if equipment needs can be met or modified. The patients may have increased metabolic needs. Difficulty compensating for changes associated with diving. Inability for the vascular system to adapt to the dive reflex. Pulmonary/chest/diaphragm muscle involvement may decrease respiratory capacity during the dive. Thermoregulation may be affected. Extra dive buddy is often recommended.

Gastrointestinal (GI)

Gastroesophageal reflux disease (GERD): Stomach squeeze following Nissen procedure can occur if the gas in the upper GI tract has difficulty equalizing. Reflux can increase because of frequent head down position.

Abdominal hernias: May have an increased risk of rupture or incarceration as the trapped air within the herniated segment expands upon ascent.

Bariatric surgery: An uncomplicated surgery and successful postoperative course should not preclude diving. May be advisable to wait for 1 year post procedure.

Ostomies: Not a disqualification to diving.

Genitourinary (GU)

Menses: There is no increase in shark attack risk during menstruation. No "womb squeeze" with use of tampons.

Gas-filled penile implants: Contraindicated because of risk of rupture.

Pregnancy: Diving is contraindicated during all trimesters.

Ear, Nose, Throat (ENT)

Chronic sinus disease, allergies, or eustachian tube dysfunction: Can predispose the diver to problems.

Sinus squeeze: Can cause headache, pressure, and even bleeding within the cavities.

Middle ear squeeze: May cause pain and even lead to TM perforation or round window or oval window rupture. Equalization is best done within the first few feet of a dive and intermittently at the first symptom of pressure.

TM perforation: No diving with an unprotected perforation because of the exposure of the middle ear to water and contaminants.

Motion sickness: Primarily affects people on the surface portions of their dive. Scopolamine is compatible with diving. However, many other antiemetics can increase the risk of nitrogen narcosis.

Ménière's disease: Diving is contraindicated because of risks of disorientation.

Ophthalmology

LASIK procedure: Recommended to wait at least 2 weeks with final clearance per ophthalmologist.

Retinal tear and detachment: Diving should be avoided for at least 2 months.

Glaucoma: May be disqualifying if surgically treated.

Gas-filled prostheses: Are a contraindication to diving.

Corneal implants/rings, or radial keratotomy (RK): Wait at least 3 months before returning to diving with final clearance per ophthalmologist.

Psychiatry

Anxiety: Phobias and poorly controlled anxiety may lead to panic underwater, which can place the diver and his or her buddy at risk. Medications often used to treat anxiety can affect diving performance and increase risk of nitrogen narcosis.

Depression: If controlled is not a contraindication to diving. Most antidepressants do not have significant adverse effects on diving.

Narcolepsy: Advisable to be episode free for 1 year.

Endocrinology

Diabetes mellitus (DM): Insulin use is not an absolute contraindication to diving. Restrict diving if problems with unrecognized hypoglycemia or hypoglycemia requiring treatment by others within the past 12 months. Restrict diving if there is evidence of severe secondary complications including retinopathy, nephropathy, neuropathy, or CAD. Restrict diving if patient has poorly controlled DM or poor understanding of the disease and relationship of exercise with diabetes. If allowed, the diver should monitor his or her blood glucose levels before a dive to ensure they are within an appropriate range and monitor ketones if they are elevated. The dive should be canceled if ketones are present.

- Blood glucose goal ranges in mg/dL
  - 1 hour predive: 80 to 250

  - 30 minutes predive: 120 to 250
  - immediately predive: 150 to 250
- If too low or dropping faster than 20 mg/dL per hour, diver should eat a snack, delay the dive, recheck blood glucose.
- If too high and no ketones, may treat with insulin and/or delay the dive.
- Sugar and glucagon should be available.
- Dive buddy should be aware of the disease and know emergency treatments.
- Consider a spare dive buddy.

Thyroid disease: Thermoregulation may be a problem.

Musculoskeletal

- Should not dive with recent fractures or significant injuries due to increased risk of DCI. This risk may be related to changes in blood flow around the area of the injury and endothelial permeability and susceptibility to the effects of nitrogen. The diver may return to diving when complete healing has occurred and appropriate rehabilitation has resulted in a return to near normal levels of strength and range of motion.
- Diving with an acute disc herniation or radicular symptoms is not advisable; the pain and radicular symptoms may be mistaken for DCI.

Hematologic

- Sickle cell disease is a contraindication.
- Sickle trait individuals may be allowed to dive within certain limits.
- Anemia is a relative contraindication.
- Coagulopathy and therapeutic anticoagulation is a contraindication because minor injuries and barotrauma may lead to severe bleeding.

Rheumatology

Raynaud's phenomenon: May lead to pain and inability to use the hands when diving, which places the person at increased risk of injuries or inability to perform safe diving.

Lupus: Depends on severity and extent of the disease.

Medications and Diving

- Need to consider how the pressure changes, oxygen, nitrogen, potential for bleeding, and the underlying disease may interact with the medicine.
- Medications generally considered safe for divers:
  - Antidepressants, aspirin, cholesterol-lowering agents, nonsedating antihistamines, nonsteroidal anti-inflammatory drugs (NSAIDs)
- Medications that are contraindicated in divers:
  - Anticoagulants, anxiolytics, and narcotics while diving
- Other medication considerations:
  - Decongestants
    - May lead to a reverse squeeze effect on the sinuses if the medications wear off during a dive. Should be used with caution and consider not diving if the symptoms are severe enough to warrant medications.
  - Antihypertensives: ACE inhibitors and ARBs are preferred.
  - Ultram may be low risk.
  - Antibiotics appear safe but ensure the infection is controlled.
  - PDE5 inhibitors (sildenafil, vardenafil, tadalafil) have not been associated with any adverse effects, though it may be prudent to avoid use of these medications within 2 hours of diving due to risks of hypotension.

Age-Related Concerns

- Professional association of dive instructors (PADI) recommends individuals be at least 10 years old to participate in their Junior Divers program.
  - Divers ages 10 to 11 must dive with a certified parent, guardian, or PADI professional and have limited dive depth of 40 feet.
  - Divers ages 12 to 14 must dive with a trained adult and may go to 60 feet.
  - A junior diver who has reached the age of 15 may transition to a regular certification without any additional testing.
- There is no upper age limit to diving. Base decisions on overall health and fitness.

Disabled Divers

- 15% of the U.S. population is considered handicapped or disabled.
- Handicapped Scuba Association (HSA International) is a group of divers and trainers who have developed training programs and different levels of certifications to allow challenged/disabled individuals to enjoy the opportunities to dive with safe and appropriate support.

Post Dive Care

- The diver may present with new symptoms a long distance from water due to travel.
- DCI can present up to 36 hours after exiting the water.
- Safe interval before flying
  - 12 hours is the minimum interval following a single dive.
  - 18 hours is the minimum if multiple dives have been done on the same day or multiple days of diving.
  - Above recommendations are for no-decompression dives in divers who have not experienced DCI symptoms and who will be flying in airplanes pressurized between 2,000 and 8,000 feet.
  - Longer intervals are advisable for other situations, and the longer the interval, the lower the chance of DCI.
- Skin infections
  - Cover for *Vibrio* and *Erysipelothrix* species
- Respiratory exposures
  - Increased chance of viral illnesses simply due to travel
  - Possible aspiration of water that may contain atypical organisms
- GI illness due to travel
  - Amoebic dysentery often presents with bloody diarrhea.
  - Toxins associated with eating exotic fish can cause GI symptoms and even neurologic changes and death.

SUMMARY

- Dive training agency certification cards (C-cards) are required by dive shops in order to fill tanks.
- Do not dive when ill or with recent changes in health status.
  - Acute upper respiratory infection may lead to difficulty equalizing.
  - Bronchitis or pneumonia may lead to pulmonary air trapping due to bronchial edema and mucous.
  - Acute gastroenteritis may lead to dehydration, increasing the risk of DCS or aspiration by vomiting into the regulator.
- Diving is a very dynamic activity; diving conditions in the same area can be completely different on different days.
  - Unknown risks, dangers, or emergencies can be encountered underwater.
  - Your dive buddy is depending on you and your fitness may affect your buddy's safety.

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Baseball

64

William G. Raasch

GENERAL OVERVIEW

History

- Baseball, the great American pastime, was first described by Abner Doubleday in 1839.
- The game evolved considerably in the early years, but it was with the advent of the overhand pitching motion in the 1880s, that shoulder and elbow problems become a familiar part of the game.
- Although ballplayers are subject to the usual sports related strains, sprains, bumps, and bruises, it is in understanding the common shoulder and elbow throwing injuries that the baseball team physician is defined.

Game Coverage

- A team physician is required at the professional level.
- Physician coverage is not required at the Little League, high school, or college level.
- Occasionally, a college will provide trainer coverage.

THROWING BIOMECHANICS AND ASSOCIATED PATHOLOGY

- Throwing a baseball is all about the transfer of energy from the body to the ball.
- A smooth transition will maximize ball velocity while reducing the risk of injury.
- The mechanics of throwing can be separated into five phases (Fig. 64-1).
- Because certain structures are susceptible to injury during each phase of throwing, the physician is aided in making a diagnosis by determining the phase in which the injury or pain occurs.

Windup

- Varies from pitcher to pitcher.
- Description:
  - Begins from the set position with the ball in the pitcher's glove.
  - Arms drop and body rotates 90 degrees (see Fig. 64-1).
  - Stride leg is elevated and flexed.
  - Provides rhythm and balance.
- Injury risk: No significant risk of injury.

Stride

- Description:
  - Begins when supporting leg flexes, lowering body.
  - Stride leg moves toward plate.
  - Trunk remains back.
  - Stride length averages 75% of body height.
  - On average, the stride length is offset by 0.4 cm (i.e., the lead foot lands almost directly in front of the back foot).
- Injury risk:
  - If stride is too long the athlete will be unable to rotate hips, resulting in a loss of velocity. The athlete may compensate

1. Windup

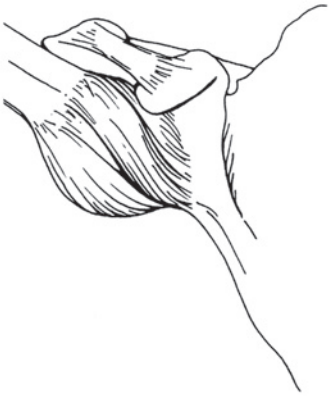
2. Cocking, late phase before maximal external rotation

3. Late acceleration, near ball release

4. Deceleration

5. Follow-through

"Winding" of anterior capsule with external rotation and horizontal abduction during cocking phase.



Valgus stress on the elbow during acceleration.

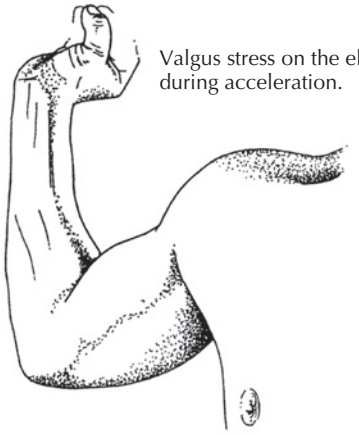


Figure 64-1 Phases of the Overhead Throw.

by overloading the shoulder, resulting in a possible rotator cuff strain.
- If stride is off line to the first base side (right-handed pitcher), the torso will be ahead of the shoulder, resulting in "opening" too soon and stressing the anterior capsule. This may result in shoulder instability.
- If stride is off line to the third base side (right-handed pitcher), the pitcher will "throw across his body" with possible shearing of the labrum.

Cocking

- Places arm in maximum external rotation.
- Description:
  - Begins when stride foot makes contact with mound.
  - Hip rotation begins with the trunk following (speed of hip rotation correlates well with ball velocity).
  - Ends when shoulder reaches maximum external rotation.
  - External rotation in a professional pitcher may approach 180 degrees.
- Injury risk:
  - When the shoulder is placed in maximum external rotation and 90 degrees of abduction, internal impingement may occur: the undersurface of the superior rotator cuff impinges against the posterior/superior labrum. This may result in undersurface tearing of the rotator cuff and fraying of the posterior/superior labrum.
  - Stretching of the anterior capsule may occur over time with resulting increased anterior/inferior laxity and possible glenohumeral instability (see Fig. 64-1).

Acceleration

- Generates ball velocity.
- Description:
  - Begins with initiation of shoulder internal rotation.
  - Ends with ball release.
- Injury risk:
  - With the rapid acceleration that occurs with ball throwing (internal rotation velocity goes from 0 to 7000 degrees per second in less than one-tenth of a second), the primary movers and stabilizers of the shoulder are stressed.
  - Rotator cuff strain—primary stabilizer.
  - Latissimus dorsi, teres major strain—internal rotation and abduction.
  - Elbow is placed under significant valgus stress—the ulnar collateral ligament (UCL) is under tension while the radiocapitellar articulation is under compression. Thus the UCL is at risk of tearing and the radiocapitellar articulation at risk for osteochondral injury.
  - Valgus extension overload—As the elbow extends while under valgus load, the posterior medial aspect of the olecranon may impinge against the olecranon fossa, resulting in posteromedial elbow pain. Over time, spurring and degenerative changes may occur at the posteromedial aspect of the ulnohumeral articulation (see Fig. 64-1).

Deceleration

- Dissipation of energy as ball is released.
- Remember, the pitcher is throwing his or her arm away from the body as well as the baseball, creating a distractive force at the shoulder equivalent to the pitcher's body weight.
- Description:
  - Begins at ball release.
  - Final elbow extension occurs.
  - Final shoulder internal rotation occurs.
  - Ends when internal rotation velocity reaches zero.
- Injury risk:
  - SLAP (superior labrum anterior to posterior) lesion—traction injury to superior labrum at the insertion of the long head of the biceps tendon.

Follow-Through

- Allows for dissipation of energy; remember, the arm as well as the ball is accelerated and should come to a gradual stop.
- Description:
  - Begins at end of shoulder internal rotation.
  - Ends when trailing leg touches the ground.
- Injury risk:
  - A proper follow-through reduces injury risk by gradual dissipation of the body's kinetic energy as it comes to a rest.

COMMON BASEBALL THROWING INJURIES

- The overhead throwing motion results in a preponderance of upper extremity injuries secondary to the significant forces generated.
- The underhanded pitching motion is not associated with significant overuse injury.

Shoulder

Rotator Cuff Injury

Description: The rotator cuff is the key to a healthy throwing shoulder, centering and controlling the humeral head as the arm is accelerated. With weakness, the cuff may impinge against the acromial arch, the capsule may stretch, or labrum tear (Fig. 64-2).

Symptoms: Pain often referred to the lateral shoulder. Posterior/superior shoulder pain with the arm in the cocking position suggests internal impingement with possible undersurface cuff tear.

Diagnosis:
- Pain and/or weakness with resisted external rotation with elbow at side.
- Positive Jobe's sign.
- Positive Hawkins' sign—subacromial impingement.
- Pain with abduction/external rotation—internal impingement.
- Magnetic resonance imaging (MRI) to define extent of injury when conservative management fails. MRI should be considered a presurgical study. History and physical exam directs initial treatment. When MRI is ordered, consider contrast to better define partial thickness undersurface tears or associated labral pathology. The ABER view (shoulder placed in abduction and external rotation) is helpful to define posterior, superior labral, and undersurface cuff tears.

Treatment:
- Relative rest—Simply refrain from any activity that triggers discomfort. This may vary from complete shut down of all

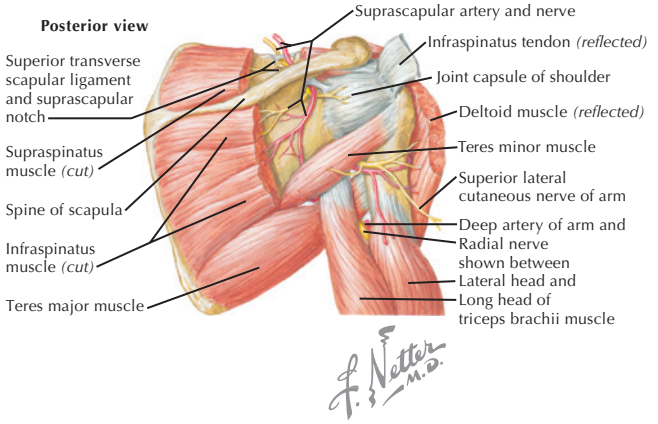


Figure 64-2 Rotator Cuff Anatomy.

throwing activity to shifting the player's position (e.g., playing first base instead of pitching).
- Physical therapy
  - Rotator cuff strengthening
    - Thera-Bands with elbow at side.
    - Advance to pulleys with arm in throwing position (shoulder abducted 90 degrees with arm internally rotating against resistance from 90 degrees of external rotation to neutral position).
    - High repetitions (30) with single set.
    - Plyometrics prior to trial of throwing.
  - Scapular stabilization—must rule out scapular dyskinesis
  - Posterior capsular stretch—must rule out tight posterior capsule
- Cortisone injection—subacromial
  - Only after failure to respond to physical therapy
  - Must shut down for 5 days after injection
- Surgery if conservative management fails
  - Arthroscopic debridement for partial thickness tears
  - Arthroscopic or open repair for partial tears greater than 50%

Labral Injury

Description: The superior/posterior labrum can be injured by impingement during the cocking phase or through distraction of the biceps anchor during deceleration (remember, the arm as well as the ball are in essence being thrown from the body and tension from the long head of the biceps can pull the labrum from its attachment).

Symptoms: Pain deep within the shoulder joint. Pain reproduced with throwing; typically not a problem with daily activities

Diagnosis: Positive provocative maneuvers (the triggered pain must be located deep within the shoulder joint) (Fig. 64-3):
- O'Brien's: Pain while resisting a downward force with the arm extended in neutral position, horizontally adducted 30 degrees, and the thumb pointing down.
- Shear: Pain with manipulation of the shoulder in 90 degrees of abduction and maximal external rotation.
- Rotation/compression: McMurray's of the shoulder—compress and rotate the shoulder in an attempt to pinch the labrum and trigger pain.

Treatment: Physical therapy:
- Correct anything that may increase the chance of pinching the labrum.
- Rotator cuff strengthening.
- Scapular stabilization.
- Posterior capsular stretch (with loss of internal rotation).

Capsular Injury

CAPSULAR LAXITY

Description: The capsule may stretch anteriorly, resulting in anterior translation of the humeral head. (Although the rotator cuff is the primary stabilizer, the capsule will provide static stability at extremes of motion—the cocking phase—and can provide proprioceptive feedback.)

Symptoms: Sensation of shoulder slipping, loss of velocity and control, tired arm (dead arm syndrome with subluxation).

Diagnosis: Must be made through history. Laxity is confirmed by physical exam: load and shift maneuver demonstrates laxity and apprehension with translation.

Treatment: Control laxity by improving rotator cuff function (remember, the rotator cuff is the dynamic stabilizer of the shoulder) with rotator cuff strengthening and proprioceptive training. Surgery with capsular placation for refractory cases.

GIRD (GLENOHUMERAL INTERNAL ROTATION DEFICIT)

Description: During the deceleration phase of throwing, the posterior capsule is tensioned. With repetitive trauma to the posterior capsule, thickening and contracture may occur. This is manifested by loss of internal rotation. With a tight posterior capsule the humeral head translates anterior and superior, resulting in cuff impingement and possible superior labral injury. Remember to check for a loss in internal rotation whenever rotator cuff impingement symptoms are present. A traction osteophyte arising from the posterior capsular attachment on the glenoid is known as a Bennett lesion.

Symptoms: Pain in the deep posterior aspect of shoulder. Anterior lateral cuff pain secondary to impingement. (With tightening of the posterior capsule, the humeral head will translate in a superior/anterior direction, increasing the risk for subacromial cuff impingement.)

Diagnosis: Exam reveals loss of internal rotation:
- Measurement is taken with arm abducted 90 degrees in the scapular plane.
- Loss of 20 degrees or more is typically symptomatic.
- Must account for the usual shift in rotation of the throwing shoulder with entire range of motion shifted in external rotation.

Treatment: Physical therapy with stretching of the posterior capsule includes adduction of the arm across the chest and internal rotation with arm abducted 90 degrees. Surgical release in refractory cases—rarely required.

Scapular Dyskinesis

Description: The scapula is the anchor for the arm during the throwing motion. Incorrect scapular position or rhythm may lead to shoulder injury (impingement of the cuff, pinching of the labrum).

Symptoms: Anterior lateral cuff impingement pain, deep anterior shoulder discomfort, pain along posterior/medial border of the scapula.

Diagnosis: Physical exam reveals the dyskinetic scapula.
- View patient from behind.
- Observe for differences in shoulder blade height and rotation.
- Observe for scapular winging when bringing the arm down from a forward elevated position.

Treatment: Physical therapy with scapular stabilization program.

Latissimus Dorsi/Teres Major Strain

Description: Often misdiagnosed injury.

Symptoms: Pain localized to the posterior inferior aspect of the shoulder. May vary from complete avulsion to minor strain.

Treatment: Conservative with physical therapy.

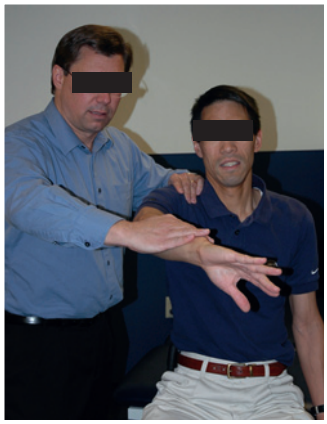
Elbow

- Valgus moment is produced with throwing—the ulnar collateral ligament is stretched, radial capitellar articulation compressed (Fig. 64-4).
- With extension of the elbow under valgus stress, the posterior medial ulnohumeral articulation compressed and has been coined the "valgus extension overload syndrome" of the throwing elbow (see Fig. 64-1).

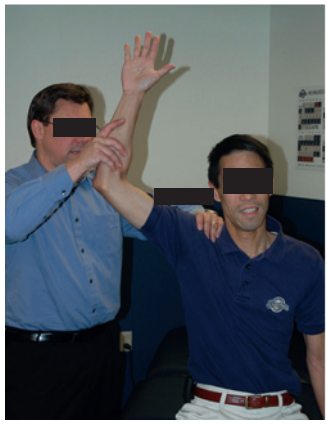
Ulnar Collateral Ligament Spain

Description: Valgus stress with throwing approaches strength of ligament. May fail with single throw, or attenuate over time. Common flexors protect ligament. (Although a diagnosis of common flexor strain may appear less severe than a ligament sprain, if the common flexor is not functioning well, the risk of UCL injury may increase.)

Diagnosis:
- Pain over UCL with palpation.
- Pain with valgus stress (elbow must be in 20 to 30 degrees of flexion to disengage the olecranon from the humeral fossa and stress the ligament).



O'Brien's Test.



Shear Test.

Figure 64-3 Elbow Exam Tests.

- Increased laxity with valgus stress.
- Soft endpoint with valgus stress.
- Pain with milking maneuver (valgus stress applied with the elbow flexed 90 degrees and the forearm in maximum pronation (Fig. 64-4).
- With increased laxity, traction to the ulnar nerve may occur, resulting in cubital tunnel syndrome—numbness in the ulnar two digits of the hand.
- MRI with contrast to confirm the injury.

Treatment:

- Complete tear in a pitcher: UCL reconstruction (the "Tommy John" procedure).
- Partial tears: a partial tear may mean many things.
- Acute partial tear more likely to respond to nonsurgical management.
  - Cessation of throwing for 6 weeks while emphasizing common flexor strengthening.
  - Plyometrics prior to throwing program.
    - Plyometrics involves bouncing a weighted ball against an angled trampoline, catching the ball with the injured arm. This provides proprioceptive training while subjecting the soft tissue to a ballistic load in preparation for throwing.
  - Progressive throwing program.

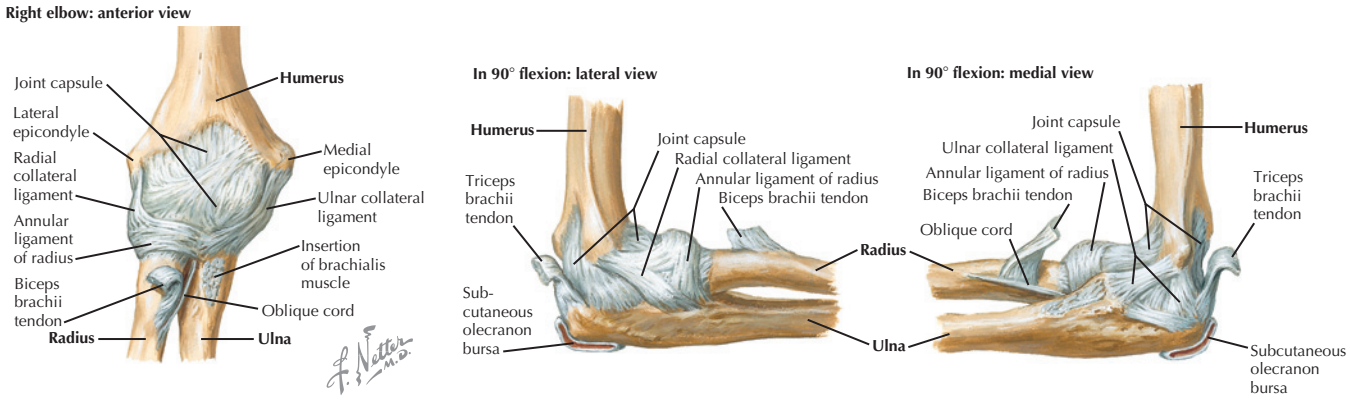
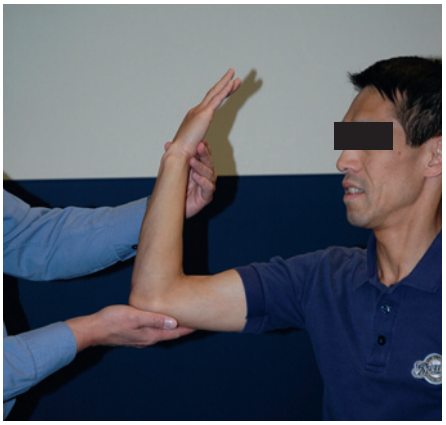


Figure 64-4 Anatomy of the Elbow.



Milking maneuver

Common Flexor Strain

Description: Dynamic medial stabilizer for the elbow. Must differentiate from a UCL sprain.

Diagnosis: Pain anterior and lateral to medial epicondyle, not over the UCL; pain with resisted wrist flexion/pronation; negative milking maneuver.

Articular Surface Injury

Description: With the elbow under a valgus stress while in extension, the posterior-medial aspect of the olecranon will grind against the medial olecranon fossa. This can lead to arthritis with spurring. The load can be increased with ulnar collateral ligament laxity. With valgus stress to the elbow the radiocapitellar joint is under compression, which may lead to early articular wear.

Ulnar Nerve Neuropathy/Instability

Description: With valgus stress at the elbow, the ulnar nerve can develop a traction neuritis at the cubital tunnel. Ulnar collateral ligament laxity may contribute to the symptoms and injury to the UCL must be ruled out with ulnar nerve symptoms. Subluxation of the ulnar nerve may occur during the pitching motion. The pitcher will experience a "zinger" down the forearm with the subluxation. The subluxed nerve can be palpated posterior to the medial epicondyle with the elbow in maximum flexion.

COMMON BASEBALL BATTING INJURIES

Contusion/fracture: A baseball weighs 5 ounces; a major league pitcher can throw the ball at up to 100 mph; when a player is hit by a pitch or fouls off a ball injury may occur. Hand fractures occur from direct impact with a thrown ball. Foot fractures occur from indirect impact from a ball fouled off the bat.

Extensor carpi ulnaris subluxation: Typically occurs when the wrist rolls over while swinging the bat (ulnar deviation with the forearm in pronation). Pain over the extensor carpi ulnaris tendon at distal ulna. Subluxation may be reproduced with resisted forearm pronation while positioned in ulnar deviation. Acute injuries may respond to cast immobilization, but more often surgical repair/reconstruction of the tendon sheath is necessary.

Hook of the hamate fracture: The hook of the hamate is fractured with swinging the bat as the hook makes contact with the nub of the bat. This may occur with a single swing or as a result of repetitive trauma resulting in a stress fracture. Pain located over the hypothenar eminence. Hand x-rays including the carpal tunnel profile view may provide diagnosis. MRI for stress fracture may be required. Excision is required with fracture.

Concussion/facial fractures: See Chapter 39, Head Injuries, and Chapter 42, Maxillofacial Injuries.

INJURIES UNIQUE TO YOUTH BASEBALL

Overview

- The skeletally immature athlete may incur injury to the upper extremity growth plates instead of the ligaments or capsule. The body will fail at the weakest site and in the Little Leaguer, the growth plate is often the weakest link.
- Whenever evaluating a growth plate, comparison views are mandatory, using the nonthrowing extremity as a control.
- In an effort to reduce the risk of throwing injuries, Little League Baseball has instituted pitching limits based on pitch count, required rest, and the age of the pitcher.

Little League Pitching Limits

- Age 17 to 18: 105 pitches/day
- Age 13 to 16: 95 pitches/day
- Age 11 to 12: 85 pitches/day
- Age 9 to 10: 75 pitches/day
- Age 7 to 8: 50 pitches/day
- Exceptions: Pitcher may continue to pitch until the current batter in which the limit is reached is put out or reaches base. A pitcher who delivers one or more pitches in a game cannot play the position of catcher for the remainder of that day.

Rest Requirements

- Age 16 and under:
  - If a player pitches 61 or more pitches in a day, 3 calendar days of rest and a game must be observed.
  - If a player pitches 41 to 60 pitches in a day, 2 calendar days of rest and a game must be observed.
  - If a player pitches 21 to 40 pitches in a day, 1 calendar day of rest must be observed.
- Age 17 to 18:
  - If a player pitches 76 or more pitches in a day, 3 calendar days of rest and a game must be observed.
  - If a player pitches 51 to 75 pitches in a day, 2 calendar days of rest and a game must be observed.
  - If a player pitches 26 to 51 pitches in a day, 1 calendar day of rest must be observed.
  - If a player pitches 1 to 25 pitches in a day, no rest is required.

Common Injuries

- Little Leaguer's elbow (medial epicondyle physical injury)
  - The ulnar collateral ligament is stronger than the epicondylar physis.
  - May be acute or chronic injury. Acute injury represents avulsion fracture of the growth plate with displacement. Chronic injury will show a widened physis on x-ray (remember the comparison view).
  - Pain over medial epicondyle with pitching and palpation.
  - Medial pain with valgus stress and milking maneuver.
  - Medial laxity with valgus stress with avulsion fracture.
  - For widened growth plate, treat with relative rest.
  - For displaced fracture, treat with anatomic repair.
- Little Leaguer's shoulder (proximal humeral physis injury)
  - Pain over the proximal arm with throwing.
  - Comparison x-rays required for diagnosis.
  - Widening of the proximal humeral growth plate on x-ray.
  - Treat with relative rest (pitching with pain may lead to fracture).
  - 4 to 8 weeks rest typically required for healing.
- Osteochondritis dissecans of the capitellum
  - Osteonecrosis of the capitellum.
  - Lateral elbow pain with throwing.
  - Locking occurs with formation of loose bodies.
  - Diagnosis with x-ray.
  - MRI may be required for staging of lesion.
  - Treat with rest with intact lesions.
  - Excision or repair required with unstable lesions.

BASEBALL THROWING PROGRAMS (REHABILITATION AND CONDITIONING)

- Following injury to the shoulder or elbow a throwing program is initiated to regain strength, form, and control.
- Prior to initiation of throwing, the following should be accomplished:
  - No pain with palpation
  - No pain with resisted firing
  - No pain with passive stretch
  - No discomfort with plyometrics (e.g., bouncing a weighted ball against an angled trampoline)
- The progressive throwing program
  - Soft toss
  - Progressive distance throwing
  - Long toss
  - Mound throwing

- Typical progressive throwing program
  - Week 1
    - Warm-up throwing for 5 minutes
    - 25 throws at 30 feet
  - Week 2
    - Warm-up throwing for 5 minutes
    - 25 throws at 45 feet
  - Week 3
    - Warm-up throwing for 5 minutes
    - 30 to 40 throws at 60 feet
  - Week 4
    - Warm-up throwing for 5 minutes
    - 40 to 50 throws at 90 feet
  - Week 5
    - Warm-up throwing for 5 minutes
    - 25 throws at 90 feet
    - Rest 5 minutes
    - 25 throws at 90 feet
  - Week 6
    - Warm-up throwing for 5 minutes
    - 25 throws at 120 feet
    - Rest 5 minutes
    - 25 throws at 120 feet

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Track and Field

Scott R. Laker, Margot Putukian, and Deborah Saint-Phard

GENERAL INFORMATION

Overview

- Track and field often attracts multisport athletes.
- The sport involves year-round competition and training.
- Differing athletic events subject athletes to differing demands. For example:
  - The shot put demands explosive power.
  - Endurance events demand high levels of aerobic conditioning and stamina.
  - Sprints (100, 200, and 400 m) demand explosive conditioning for power, flexibility, and anaerobic conditioning.
  - Middle distance races (800, 1500 m) demand a combination of anaerobic and aerobic conditioning to sustain power and stamina.
  - Distance races (3000, 5000, 10,000 m; marathon) demand stamina, high levels of aerobic conditioning, and sustained power.
  - 100 and 400 hurdles, relays (4 × 100 m, 800 m, 4 × 400 m, 4 × 800 m, 4 × 1500 m, distance medley, sprint medley) demand mental concentration, power, and stamina.
  - Long jumps, high jump, triple jump, shot put, javelin, discus, hammer, heptathlon, decathlon, pole vault, and steeplechase demand combinations of technique, stamina, power, and speed.

Statistics

- Difficult to look at injury statistics given lack of denominator (player hours or athlete exposures) as well as variability in methodology of studies.
- No National Collegiate Athletic Association (NCAA) data collected for track and field.
- Of participants in the 1985 Junior Track Olympics, 35% reported need for performance-related medical treatment.
- In general, **most injuries are overuse injuries that occur during training.**
  - Acute injuries are often muscle strains or avulsions, or are related to stress fractures; other acute injuries uncommon.
  - Majority of injuries involve lower extremities.
  - Some risk for head or neck injury during high jump and pole vault; risk of blunt trauma with javelin, hammer, discus.
  - Of particular concern are acute medical problems such as cardiovascular collapse secondary to underlying cardiac disease, dehydration, or other abnormalities related to environmental conditions. Medical concerns are primarily related to chronic training *including over-reaching and frank overtraining*.
- **Site of injury**
  - Most common injury sites are the lower leg (28%), thigh (22%), and knee (16%).
  - Most common injuries were stress reaction and hamstring muscle strains.
  - **The knee is the most commonly injured joint in runners** (48% of all injuries), followed by lower leg (20%), foot (17%), hip (6%), upper leg and thigh (4%), lower back (4%). Overuse injuries more common in the distance runners, whereas acute injuries are more typical of sprinters, hurdlers, jumpers, and multievent athletes. Recurrent injuries are very common.
- **Specific diagnoses**
  - Anterior knee pain accounts for 24% of running injuries in men and 30% of running injuries in women.
  - Medial tibial stress syndrome accounts for 7.2% of injuries in men and 11.4% in women.
  - Iliotibial band syndrome accounts for 7.2% of injuries in men and 7.9% in women.
  - Patellar tendinosis accounts for 5.1% of injuries in men and 3.1% in women.
  - Metatarsal stress syndrome accounts for 3.1% of injuries in men and 3.8% in women.
  - Achilles tendinosis accounts for 4.7% of injuries in men and 2.7% in women.

MUSCULOSKELETAL ISSUES

Basic Running Mechanics

- Important to understand normal biomechanics of running to understand abnormal biomechanics and how they can affect incidence of injury.
- **Foot strike:** at lower speeds occurs with heel, but at higher speeds occurs with forefoot.
- Ground strike occurs 800 to 2000 times per mile for average runner (5000 foot strikes per hour of running).
- **Reaction forces at foot strike are usually 1.5 to 5 times body weight.** Joint shear forces during running increase to almost 50 times that of walking. These reaction forces are augmented considerably by different surface types.
- At initial rearfoot contact, the foot is in supination. This is associated with "closed-pack," rigid position of the tarsal bones, increasing stability.
- Foot then pronates with tarsal joints assuming "open-packed" position, which is more accommodating and less rigid, allowing partial absorption of reaction forces. Internal rotation of tibia on talus.
- As runners progress to push-off, subtalar joint supinates with external rotation of tibia. Foot remains in supination during airborne phase and forward swing of leg.
- Major muscle groups all show increased electromyographic activity during running, and all lower extremity joints show increased motion during running.
- In stance phase of running, ankle generates 60% of the power generation, whereas knee and hip generate 40% and 20%, respectively.
- **Knee is principal shock absorber during running, absorbing twice as much energy as ankle and hip.**
- **Abnormal biomechanics can lead to overload of other structures.** Commonly, gluteus maximus weakness can lead to poor control of the lower limb, and reaction forces being transmitted throughout the kinetic chain. Another example, abnormal amount of rearfoot varus or pronation can abnormally load structures higher in kinetic chain, leading to increased valgus stress at knee. This is an important etiologic factor in patellofemoral dysfunction (see "Patellofemoral Dysfunction" later in the chapter).
- **Orthotics** may help prevent injury if significant biomechanical abnormalities present. Screen with gait assessment during the preparticipation exam.

Physiologic Issues

- **Demands depend on type of activity**
  - **Sprinters:** Energy requirements provided primarily by anaerobic energy pathways. Glucose major fuel source.
  - **Long distance events:** Energy requirements provided primarily by aerobic energy pathways, with fat and glucose derived from glycogen stores.
  - **Middle distance and combination events:** Combination of both aerobic and anaerobic pathways.

- Specificity of training, including periodization, to demands based on types of energy pathways used.

Strengthening and Conditioning

- **Key in track and field is sport specificity in training. This differs for each event.** Much of training continues year round. Need to emphasize variability in training and avoid overtraining.
- **Endurance and sprint athletes:** Strengthening of muscle fiber types (fast twitch vs. slow twitch) demonstrates specificity (i.e., endurance-type training leads to changes in slow-twitch fiber morphology as well as enzyme metabolism specific for endurance-type activities). Similarly, explosive strengthening programs specifically train fast-twitch muscle fibers and metabolic functions used to sustain these activities. It is more beneficial to train with sport specificity in mind when designing strength and conditioning programs.
- **Use of entire range of motion (ROM) for strengthening is important.** Strength gains seen are specific to the range and speed at which strengthening exercises are performed.
- **Field events:** Successful transfer of ground reaction force through the foot, ankle, knee, hip, trunk, shoulder, elbow, wrist, hand and finally to the implement is critical to success in throwing events. Maintain quadriceps-hamstring balance. Again, sport specificity is helpful. Shoulder scapular stabilization and rotator cuff strengthening program helpful for shot put, javelin, hammer, and discus. Reproduction of shoulder movement with manual resistance. Proprioceptive work helpful in hurdles, discus, javelin, and triple jump and long jump.
- **Develop smaller supporting muscles.** Strengthening prevents development of shin splints, plantar fasciitis, patellofemoral dysfunction, and other overuse injuries.

Flexibility

- Despite lack of reliable data about effects of increased flexibility in preventing injury, most agree that flexibility program helps avoid acute muscle strains. If muscle length at which maximal stretch felt is greater, it takes larger acute overload to "stretch" muscle past this length, leading to injury. Flexibility remains essential tool in treatment of muscle strains and joint protection.
- Flexibility program should be worked into strengthening program such that strength is improved throughout ROM without loss of motion.
- Ballistic stretching should be avoided.
- Stretching should be performed after warming up, rather than while "cold" at the start of practice.
- Stretch larger muscle groups first, followed by smaller groups.
- Hamstring flexibility important in mechanical low back injuries. If hamstring flexibility is limited, a posterior pelvic tilt is created and trunk flexion becomes limited, and stress in the low back is increased.

MEDICAL PROBLEMS

Preparticipation Physical Examination

- Stresses the importance of cardiac, musculoskeletal, and neurologic systems.
- Detailed family history emphasizing any history of heart murmur, sudden cardiac death, Marfan syndrome, hypertrophic cardiomyopathy, or premature atherosclerotic disease.
- Screen for possible anatomic abnormalities that may predispose to injury; consider orthotics and flexibility/strengthening program in the presence of muscle imbalances especially of the hip abductors and gluteal musculature assessed both statically and dynamically.
- Assess for nutritional or training errors, including any "self-restricted" food types. Inquire about lactose intolerance.
- Rule out significant medical or orthopedic problem that would preclude activity or require restrictions.
- In female athletes, pay special attention to menstrual dysfunction, hypocalcemic diet, history of disordered eating, history of stress reactions or fractures (see "Female Athlete Triad" later in the chapter).

Nutrition Issues

- Important to consider nutrition when athlete presents with symptoms of fatigue, burnout, or recurrent minor injuries (see Chapter 5, Sports Nutrition).
- Zinc, calcium, and iron are commonly deficient in athletes.
- Ideal nutritional intake: 6 to 10 g carbohydrate per kg body weight; 1.2 to 1.7 g protein per kg body weight; remainder of calories from fat. This translates into diet of 60% to 70% carbohydrate, 10% to 15% protein, and 25% to 30% fat. No performance benefit has been shown with diets consisting of 15% fat versus diets consisting of 20% to 25% fat.
- If an athlete eats an adequate caloric intake from a variety of wholesome foods, nutritional needs are often met. Proper food selection, not supplementation, is the ideal form of nutrition.
- **Iron deficiency** is common in young athletes. Iron loss can be due to hemolysis with hemoglobinuria, gastrointestinal losses, and excessive sweating. Female athletes are at increased risk of anemia because of additional loss that occurs with menses. Athletes are not immune from other medical problems; thus a complete workup of an iron-deficient athlete is important.
  - Iron deficiency or decreased iron stores can occur without anemia in as many as 9.5% to 57% of athletes, depending on study.
  - Dietary recommended intake varies based on sex and age. Females age 19 to 30 years require 18 mg of iron per day, and males age 19 to 30 years require 8 mg per day.
  - Pseudoanemia: increase in plasma volume of 6% to 25% with training results in hemoglobin and hematocrit appearing falsely low. Typically self-limited.
  - Screening hemoglobin with follow-up ferritin is reasonable for assessing iron deficiency. Ferritin is a storage form of iron, but can be falsely elevated in acute inflammation and can be decreased with high-intensity training. Iron and total iron binding capacity can differentiate pseudoanemia from true anemia.
  - Supplementation of 324 mg ferrous gluconate two or three times daily if truly iron-deficient. Increase intake of iron-rich foods and assure adequate vitamin C intake. A daily multivitamin with iron is reasonable.
- **Calories:** While obtaining an adequate total caloric intake is obviously essential, this basic ingredient of good nutrition is often neglected.
  - In an attempt to eat "healthy," many athletes restrict fat intake. This strategy results in fat-soluble vitamins (vitamins A, D, E, and K) at risk for being deficient. Diets with less than 15% fat have shown no gains in athletic performance or health.
  - Some athletes experiment by restricting calories as a means of reducing body weight, thus improving aesthetics and performance. Unfortunately, this can increase the risk for developing a frank eating disorder along with its concomitant medical problems.
  - Other deficiencies found in athletes include zinc, magnesium, folate, vitamins B6, C, and B12. Consider a nutritional consultation to formally review food intake if concerned about vitamin and/or caloric deficiencies.
- **Protein intake:** Often an issue in vegetarian athletes and those that restrict food intake.
  - Consult a nutritionist to ensure an adequate intake.
  - If vegetarian, protein complementarity (legumes and grains) can ensure adequate protein intake, but still important to

assess iron intake. Minimizing this potential deficit may minimize potential fatigue, and diet-related conditions.
  - Voluntary protein intake, along with fat and total energy intake, has been shown to be lower in athletes with menstrual irregularities than in normally menstruating athletes.
- **Calcium:** Dietary reference intakes (DRI) vary based on age. Most recent guidelines suggest 1000 mg daily for ages 9 to 18 years, and 1300 mg daily for ages 18-50 (Fig. 65-1).
  - Female athletes often consume less than the DRI for calcium.
  - Calcium and estrogen are necessary in women for normal bone deposition. If depleted, this can lead to lower bone density. Peak bone density reached in women in late teens to early 20s; thus, adequate intake in childhood and adolescence is critical.
- **Vitamin D**
  - Vitamin D is essential for bone health and incorporation of calcium. Primary sources are sunlight and vitamin-D enriched foods like milk.
  - Vitamin D levels have been increasingly associated with generalized musculoskeletal pains.
  - DRI is 25 micrograms daily and vitamin D-25-OH levels can be monitored.
  - Athletes who practice and compete in indoor sports and those living in Northern latitudes may be at risk**. Consider supplementation if levels are low.**
- **Nutritional supplements and ergogenic aids**
  - Athletes are at risk for use and abuse of supplements as well as ergogenic aids. Some of these are restricted under U.S. Olympic Committee and NCAA drug testing. Examples include ingesting excess amino acids, medium-chain fatty acids, vitamins, minerals, herb extracts, special proteins, and enzyme complexes. Often marketed to individual sports. Reports suggest up to 62% of track athletes use supplements, including multivitamins. Specific questions related to pharmacokinetics, interaction with normal foodstuffs, and prescribed medications, and side-effect profile should be asked of the pharmacist.
  - **Supplements:** If the athlete is not deficient in a vitamin, the positive effects of supplementation are unproven. In the setting of dehydration, amino acid supplementation may be detrimental if kidney function is marginal. Protein and vitamin supplementation in great excess can be dangerous. Excesses of most vitamins are eliminated from the body. Fat-soluble vitamins are stored within body; thus toxicity is possible. Most methods of supplementation are expensive.
  - **Ergogenic aids:** erythropoietin, human growth hormone, and anabolic steroids
    - Difficult to detect erythropoietin and human growth hormone with current drug-testing regimens.
    - Anabolic steroids are detectable with urine drug testing.
    - All are associated with significant side effects. Erythropoietin is associated with hyperviscosity syndrome, even death. Hyperviscosity syndrome is made worse with dehydration. Human growth hormone is associated with side effects that include acromegaly-like features. Anabolic steroid side effects are well known.
    - For up-to-date information, contact U.S. Olympic Committee drug information hotline (800-233-0393).
- **Fluid considerations**
  - During prolonged exercise, 2 to 4 pounds of body weight are lost per hour, equivalent to 1 to 2 L per hour.
  - Rate of dehydration can be estimated by changes in nude body weight; each pound weight loss equals 450 to 650 mL (16 to 24 ounces) of dehydration.

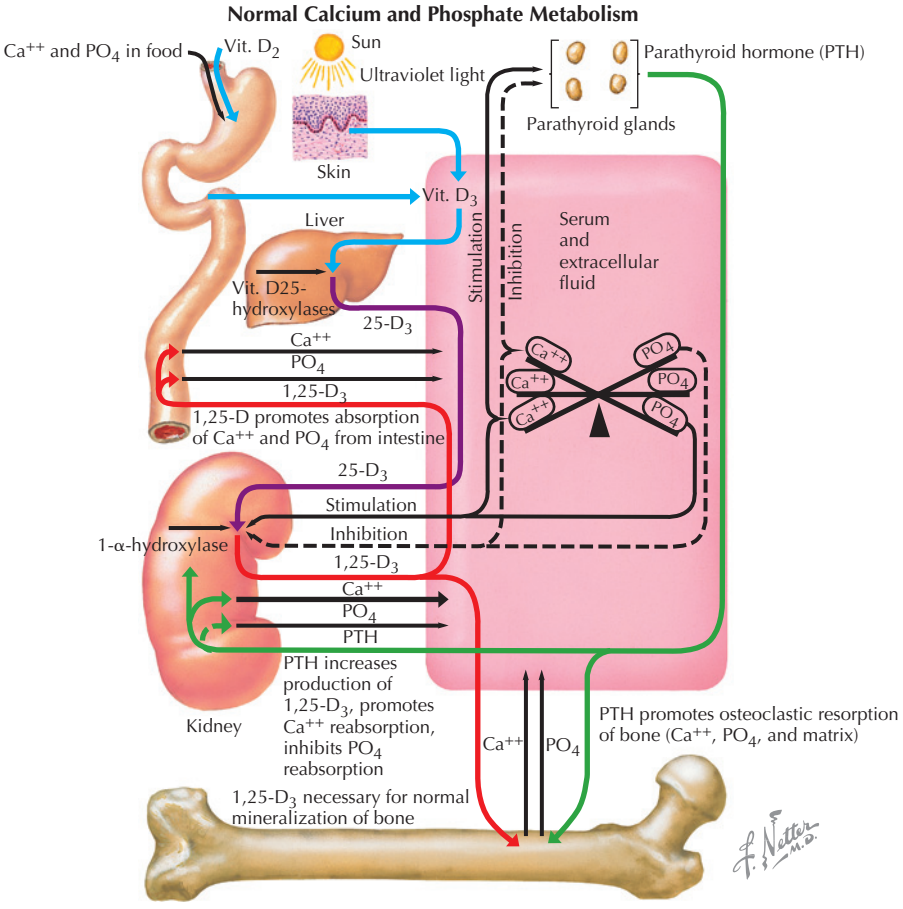


Figure 65-1 Normal Calcium and Phosphate Metabolism.

- Dehydration can incur physiologic changes; increase in core body temperature of 0.3° C, increase in heart rate of 8 beats per minute, and decrease in cardiac output of 1 L per minute, for every liter of water (2.2 pounds) lost while exercising in the heat.
- Proper rehydration essential during, after, and between events.
- Thirst is a delayed sensation and therefore is not a good indicator of hydration status.
- For athlete that weighs 68 kg (150 pounds), carbohydrate requirement is 30 to 60 g per hour. Carbohydrate and fluid needs can be met by drinking 625 to 1250 mL per hour of beverages with 4% to 8% carbohydrate.
- For a glycogen-deplete athlete, a post-race carbohydrate intake of 1.5 g per kg body weight during the first 30 minutes post-race and again every 2 hours for 4 to 6 hours will effectively replenish glycogen stores.
- Some studies relate decrease in gastric emptying once glucose concentration is above 6%. Water still excellent source for short distance events (<1 hour).
- For on-site evaluation and management of syncope or presyncope, measurement of orthostatic blood pressure and pulse is most reliable.
- Cannot assume every runner is dehydrated without proper assessment (see "Syncope" later in the chapter).

- **Nutritional recommendations are different for females and males.**
  - Societal influences have made constant dieting acceptable for girls and women, and athletes are even more likely to attempt to change body appearance if they think it will improve performance.
  - Females at increased risk for nutritional deficiency as well as eating disorders.

Female Athlete Triad

- Disordered eating, menstrual dysfunction, and low bone mass (see Chapter 10, The Female Athlete) (Fig. 65-2).
- Present in every sport but most commonly seen in sports that select for lean body weight (swimming, cross-country skiing, cross-country running) or in sports scored subjectively (gymnasts, figure skaters, divers).
- Self-worth involvement.

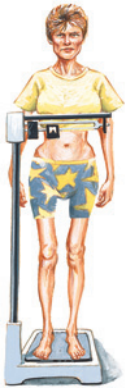
Eating Disorders

- Origins multifactorial: perfectionism, personality traits, identity, self-esteem, family dynamics, coping skills, control issues, alterations of body image, need for control. Patients often have history of sexual or physical abuse. Societal pressures increase risk.
- Women account for 90% of eating disorders.
- Increased incidence in athletes compared with general population.
- Characteristics associated with successful athletes lead to overlapping and increased risk for developing eating disorders include perfectionism, goal setting, and overachieving.
- Subtle messages by coach, parents, or teammates (often unintentional) can add to pressures that lead athlete to experiment with pathogenic weight control behaviors:
  - Misconception that lower body weight improves performance.
  - Educating coaches in how to recognize and prevent eating disorders is essential.
- Difficult to identify; team approach often necessary for proper treatment.
- Treatment team usually includes physician, psychiatrist, and nutritionist. Additional support system includes coach, sport psychologist, athletic trainer, and family. Psychological counseling is cornerstone.
- Treatment not very successful, emphasizing need for prevention through education and early identification.
- Screen for concomitant depression. Preliminary studies suggest favorable response to antidepressants such as fluoxetine.

Menstrual Dysfunction

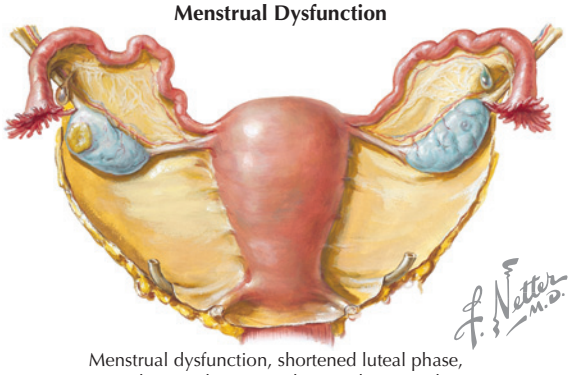
- Common in athletes, especially endurance athletes.
- Causes multifactorial. Menstrual dysfunction, shortened luteal phase, anovulation, oligomenorrhea, and amenorrhea can occur in response to chronic exercise and are associated with decreased bone mineral density (BMD) or low bone mass.
- Exercise-associated menstrual dysfunction remains a diagnosis of exclusion.
- Must rule out other conditions: pregnancy, thyroid disorders, adrenal disorders, prolactin-secreting tumors, ovarian disorders.

Eating Disorders



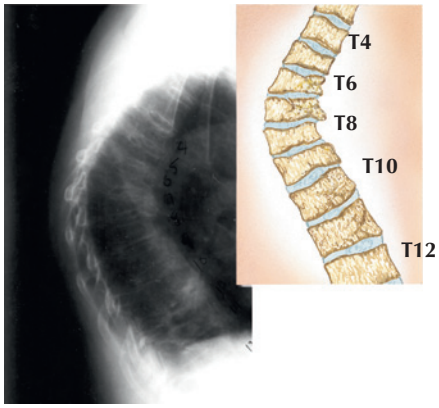
Origins multifactorial: perfectionism, personality traits, identity, self-esteem, family dynamics, coping skills, control issues, alterations of body image, need for control. Patients often have history of sexual or physical abuse. Male and female athletes should be screened for disordered eating. More common in sports in which "lean" aesthetics are favored (gymnastics, cross-county, etc.)

Menstrual Dysfunction



Menstrual dysfunction, shortened luteal phase, anovulation, oligomenorrhea, and amenorrhea can occur in response to chronic exercise and are associated with decreased bone mineral density (BMD) or low bone mass. Detailed menstrual history should be obtained.

Osteoporosis



Low BMD risk factor for early osteoporosis and stress fractures. Increased incidence of stress fractures seen in runners with amenorrhea. These athletes are at increased risk for decreased lifetime peak bone mass, which can put them at risk for postmenopausal osteoporosis.

Figure 65-2 Female Athlete Triad.

- Important to initiate workup and treatment because of long-range consequences of menstrual dysfunction. Treatment must be individualized.
- "Progesterone challenge" helpful in functionally differentiating estrogen-deficient state from estrogen- and progesterone-deficient state.
- If exercise-associated amenorrhea is diagnosed and no contraindication exists, estrogen and progesterone supplementation should be considered. Many use age 16 as younger cutoff for initiating treatment.
- Oral contraceptive pill has good side-effect profile, is well tolerated, and has convenient packaging. If positive progesterone challenge, can use monthly progesterone alone.
- Other considerations: decrease training intensity; increase body weight if underweight; assess nutritional intake; maintain high index of suspicion for eating disorders.

Osteoporosis and Stress Fractures

- Bone is constantly remodeling and is a balance between osteoblast and osteoclast activity. Stress fractures are a result of increased stress on normal bone, or normal stress on abnormal bone (Fig. 65-3).
- Amenorrheic runners have lower estrogen levels and lower BMD than runners with normal menstrual cycles.
- History of menstrual dysfunction correlated with low BMD in runners.
- Low BMD risk factor for early osteoporosis and stress fractures. Increased incidence of stress fractures seen in runners with amenorrhea. These athletes are at increased risk for decreased lifetime peak bone mass, which can put them at risk for postmenopausal osteoporosis.

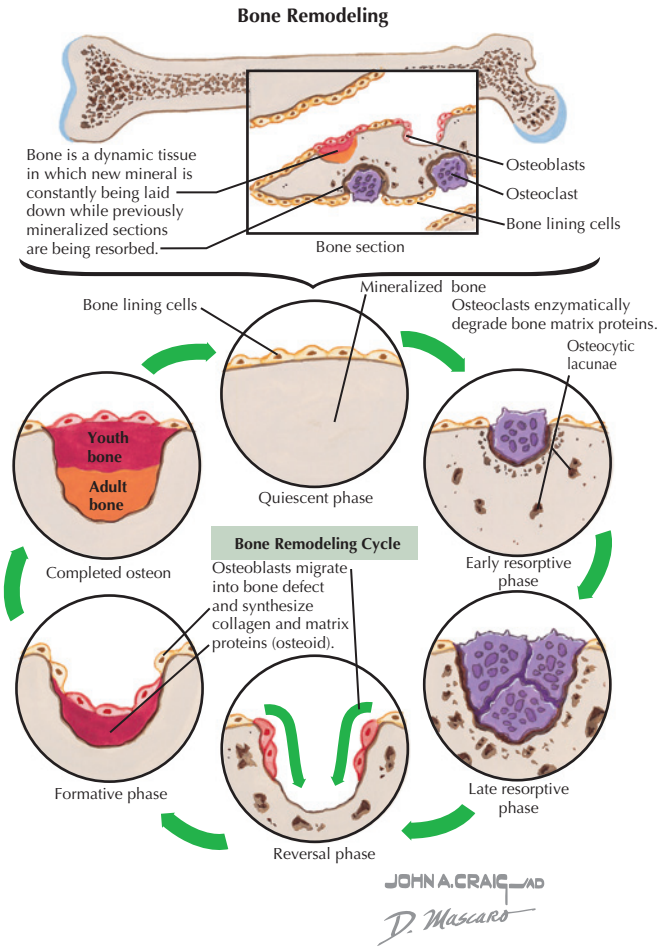


Figure 65-3 Bone Remodeling.

Recognition and Treatment

- **Detection and education critical.**
- Preparticipation physical examination offers good opportunity to address these issues with female athletes and provides opportunity to educate young athletes about importance of maintaining normal menstrual function, risks of eating disorders, and relation of both to incidence of stress fractures and early low bone mass.
- Ask athletes who present with stress fractures about current and past menstrual history.
- Supplemental history for female athletes is helpful screening tool during preparticipation physical exam.

Overtraining

Description: Often difficult to recognize and treat effectively. Common in endurance athletes with increase in training volume or intensity.

Symptoms: Nonspecific, insidious symptoms include fatigue, irritability, sleep difficulty. Often see depression, anger, and fatigue as well as decreased performance (i.e., "staleness"). Long-term underperformance shown in physiologically overtrained athletes.

Diagnostics: Generally a diagnosis of exclusion. Laboratory testing normal. Important to assess for anemia, hypothyroidism, infection, collagen vascular disease, glucose abnormalities.

Treatment: Decrease in training, increase in carbohydrate intake, gradual return to activity. Sports psychologist referral should be made.

Associated conditions: Overtraining contributes to burnout, overuse injuries, stress fractures, menstrual dysfunction, iron deficiency, and long-lasting decrements in performance.

Syncope

Description: Common medical problem with multiple causes.

Evaluation: Need to be rigorous in excluding serious medical problem, especially if syncope occurs in the midst of full exertion. Ask about history of associated chest pain, palpitation, shortness of breath, dizziness.

Differential diagnosis:

- Dehydration
- Cardiac sources: long Q-T interval, arrhythmias, hypertrophic cardiomyopathy, anomalous coronary arteries, aortic stenosis or other valvular abnormalities, neurocardiogenic syncope.
- Neurologic sources: seizures, arteriovenous malformations, aneurysm.
- Hematologic (anemia) or electrolyte abnormalities (red flag for eating disorders).

Diagnostics: History is essential because syncope during exertion is much more concerning than syncope occurring after full exertion and standing still (common in neurocardiogenic syncope). Further workup and testing as indicated by history, family history, and physical exam. Often difficult to differentiate pathologic cardiac condition from "athlete's heart." Athlete's heart is a physiologic response to training: hypertrophy, prolonged electrocardiographic intervals, functional (nonpathologic) flow heart murmurs. Echocardiography, maximal exercise testing, and tilt-table testing are often useful adjuncts to the history and physical exam.

Return to play: Guidelines are unclear, but serious, life-threatening abnormalities must be ruled out.

Gastrointestinal Problems

Alterations in gastrointestinal (GI) function: 25% to 50% of runners experience abdominal cramps, diarrhea, nausea, and abdominal pain. Blood flow to the gut typically decreases 50% during exercise.

Diarrhea: Typically self-limited and physiologic, tending not to cause dehydration or electrolyte imbalances. Antidiarrheal medications should be avoided whenever possible.

Celiac sprue (gluten sensitive enteropathy): Can lead to decreased bone mass secondary to poor absorption of calcium. Athletes will typically complain of bloating and/or diarrhea, and may have a family history. Consider this in athletes with these chronic symptoms. Also may have electrolyte abnormalities and vitamin deficiencies. Diagnosis is made with serologic testing for immunoglobulin A (IgA) and immunoglobulin G (IgG) antitissue transglutaminase. If positive, then a small intestinal biopsy is performed for definitive diagnosis.

GI bleeding: Severe GI bleeding after endurance running reported, with rare occurrences of death secondary to acute hemorrhage, though these are reported as isolated cases. Reports of bright red blood per rectum must be rigorously worked up and ulcerative colitis and Crohn's disease must be ruled out. Occult forms of GI bleeding are common. Studies have reported anywhere from 8% to 22% clinically detectable bleeding in marathoners after race. If more sensitive assays are used, increase in fecal heme concentrations is seen in 83% of marathoners. Thus, runners have increase in stool heme after race, and in approximately 20% it is detectable clinically. Bleeding self-limited and resolves in roughly 72 hours. Bleeding can be associated with excessive iron loss and resultant iron deficiency. Must differentiate from runner's pseudoanemia. Pathophysiology unclear, but must consider nonsteroidal anti-inflammatory drug (NSAID) use, bowel ischemia, traumatic shearing effect from running, and underlying GI abnormalities. Postexercise biopsy of colon to reveal congestion and vascular lesions. Evaluate with appropriate imaging. After GI pathology ruled out, treatment issues surround presence/absence of iron deficiency and consideration of antimotility agents for diarrhea. Antidiarrheals inhibit heat dissipation and should be used with caution.

Reflux/delayed gastric emptying: Common complaint in endurance athletes; may result in nausea or vomiting. Exercise increases acid secretion and in some athletes may lead to heartburn-type symptoms. Symptoms exacerbated by precompetition nervousness. Thorough workup indicated to rule out GI pathology. Treatment centers on alteration in eating patterns, trials with magnesium or aluminum hydroxide and simethicone (Maalox), or H_2-blocker trial (1 hour before events).

Exercise-Induced Asthma

Description: Common in athletes, especially when exercising in cold. **Should be discussed during pre-participation physicals, including severity and current medications.**

Symptoms: Variable, occurring with exercise: wheezing, tightness, chest pain, shortness of breath. Symptoms sometimes vague: poor exercise tolerance, cough or tightness after exercise. Symptoms aggravated by allergens, upper respiratory infections, environmental conditions (humidity, cold air).

Diagnostics: Provocative testing helps make diagnosis and assess response to bronchodilators: spirometry before exercise challenge, adequate exercise challenge (use whatever athlete describes as "typical" precipitant), postexercise spirometry. Look for decrease in forced expiratory volume in first second (FEV_1) and decrease in FEV_1 as fraction of total forced vital capacity.

Treatment: Beta-agonist, such as albuterol, often helpful as preexercise medication to prevent exercise-induced asthma. Other medications include sodium cromolyn (Intal) or nedocromil sodium (Tilade) as premedication. Inhaled corticosteroids or three to four times daily use of medications helpful for acute flare of symptoms, baseline asthma (not only exercise-induced asthma), or allergen-induced asthma. Avoid known precipitants. Proper cardiovascular warm-up can lessen symptoms and allow athlete to "run through" asthma: 15 to 20 minutes at approximately 70% $\dot{V}O_2$ max, series of 40- to 50-yard sprints. Minimize symptoms. Use scarf or other methods to warm inspired air. Breathe through nose.

Drug testing concerns: Drugs allowed change frequently. Athletes should be made aware of the acceptability of their current medications. All medication questions should be addressed to the U.S. Olympic Commission Drug Information Hotline (800-233-0393).

Renal Issues

Pseudonephritis: Proteinuria, hematuria, and cellular elements in urine after exercise. Exercise associated with decreased renal blood flow. Decreased glomerular filtration rate results, with dehydration, as exercise continues. Proteinuria, hematuria, and pyuria, as well as cellular elements seen after intense exercise, are self-limited. Workup indicated if abnormalities persist after discontinuing exercise or if other symptoms or risk factors exist.

Gross hematuria: Reported in runners. Often occurs without warning and without symptoms, as painless clots of blood or grossly bloody urine. Possibly due to traction effect on bladder. Cystoscopy can sometimes detect bleeding source, but often negative. Further workup indicated if abnormalities persist despite stopping exercise or if other symptoms or risk factors are present.

COMMON MECHANISMS OF INJURY

Precipitating Factors

- **Training errors account for approximately 80% of injuries** (Fig. 65-4). Changing to harder running surface, abrupt increase in training mileage (10%), abrupt increase in training intensity, hill running, consistent running on crowned roads, previous injury, inadequate rest/nutrition.
- **Uncorrected anatomic problems** include hip, pelvis, back, knee, foot, ankle and leg length discrepancy.

Overuse

- Most common mechanism in track and field injuries.
- Classified according to timing of pain in relation to onset of activity.

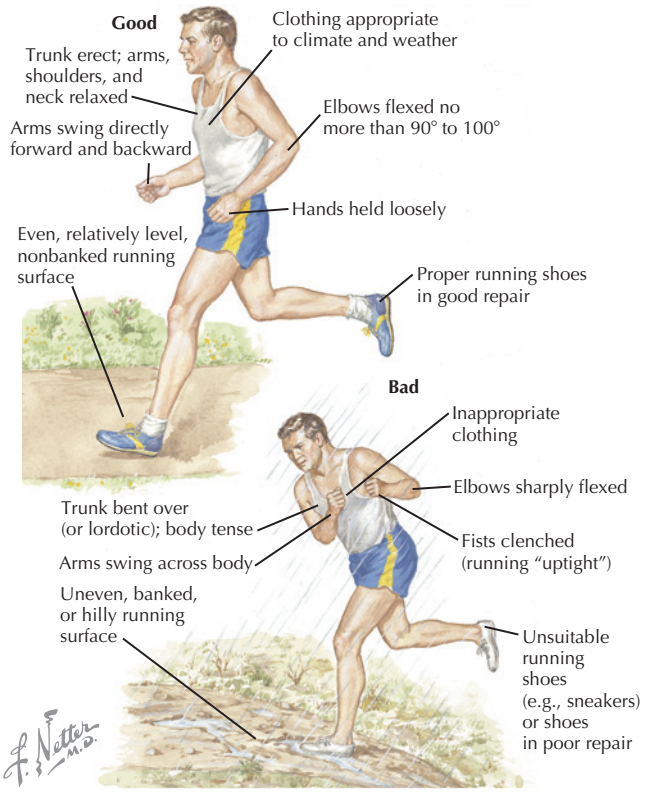


Figure 65-4 Good and Bad Running Practice.

- Type 1: pain after activity
- Type 2: pain during activity, not restricting activity
- Type 3: pain during activity, restricting activity, which restricts performance
- Type 4: chronic, unremitting pain

SPECIFIC MUSCULOSKELETAL INJURIES

Upper Extremity

Shoulder Impingement

Description: Common in field events: javelin, discus, shot put.
Causes: Rotator cuff pathology, labral tear, biceps tendinitis, multidirectional instability with muscular compensation, natural adaptations in range of motion, or overuse.
Treatment: NSAIDs, ice, decrease in training, flexibility, evaluation of biomechanics. If no response, assess need for further diagnostic studies, specialist evaluation.

Acromioclavicular Injuries

Description: Most commonly occur after fall on shoulder with arm by side.
Symptoms: Pain and swelling directly over joint, pain with adduction.
Treatment: Ice, NSAIDs, relative rest.

Fractures

Description: Usually result from direct fall.
Imaging: Required for suspected fractures.
Treatment: Depends on fracture site and type.

Medial Epicondylitis

Description: Seen in throwers.
Evaluation: Need to exclude fracture, valgus instability, and neurologic involvement.
Treatment: Biomechanical evaluation, ice, NSAIDs, physical therapy modalities, relative rest. Consider arm brace (reversal of lateral epicondylitis strap).

Lower Extremity: Knee

Knee accounts for 30% to 50% of all track-and-field injuries.

Iliotibial Band Friction Syndrome

Description: Common in runners. Iliotibial band inserts into Gerdy's tubercle along lateral tibia (Fig. 65-5). Often seen in conjunction with greater trochanteric bursitis.
Presentation: Lateral knee pain, often in midrange of knee flexion, from 20 to 70 degrees, when iliotibial band rubs across lateral femoral condyle.
Evaluation: Contributing anatomic factors (should be corrected if possible): leg length discrepancy, abnormal foot biomechanics (especially hyperpronation), tibia vara, scoliosis. Tightness of musculature and/or muscle strength imbalances often seen—important to treat with corrective rehabilitation.
Diagnostics: Ober's test is useful for identifying tight iliotibial bands. Can reproduce pain by resisting knee extension and looking for painful arc. This is sometimes difficult to differentiate from trochlear articular surface or osteochondral defect.
Treatment: Stretching, ice, oral NSAIDs, phonophoresis, iontophoresis. Avoid running on beveled surfaces. Consider corticosteroid and anesthetic agent if other measures fail.

Greater Trochanteric Bursitis

Description: Tight lateral structures (iliotibial band syndrome) often compress bursa and cause increased friction of iliotibial band over greater trochanter. Can be any combination of three bursae in the area.
Presentation: Lateral hip pain and pain with active abduction, passive adduction. No pain with passive hip rotation.
Examination: Assess biomechanics of hip, knee, and foot and ankle; leg length discrepancies; training errors, all of which can lead to increased friction at the greater trochanter.
Treatment: NSAIDs, ice massage, stretching of the iliotibial band, external hip rotators, hip flexors, use of phonophoresis, iontophoresis, corticosteroid injection (if not responsive).

Patellofemoral Dysfunction

Description: Generally felt to be caused by abnormal tracking of patella with resultant patellofemoral irritation and pain (see Fig. 65-5). New theories on patellofemoral dysfunction suggest that it results from activity that pushes the tissues of the knee beyond a "zone of homeostasis" rather than biomechanical alignment problems. Understanding static and dynamic orientation of patella is important in understanding nature of tracking dysfunction as well as in guiding rehabilitative treatment.
Contributing factors: Increased Q angle, deficient vastus medialis obliquus (VMO) or tight vastus lateralis musculature, patella alta, pronation (increases functional Q angle), genu valgum and recurvatum.
Presentation: Anterior knee pain, often made worse by climbing or usually descending stairs or prolonged sitting ("theater sign").
Treatment: Mainstay remains strengthening of VMO along with improved flexibility of lateral structures. Various patellar taping methods to allow patella to track more normally and facilitate pain-free strengthening of the VMO. Unclear how functionally effective taping is. Correction of leg length discrepancy or excessive pronation or other abnormal foot biomechanics if present. Closed-chain kinetic exercises, used in combination with physical therapy modalities, including ice, to decrease inflammation. Biofeedback is often helpful.

Leg Length Discrepancy

Description: Up to 5-mm difference in leg lengths is normal. Can contribute to iliotibial band friction syndrome, greater trochanteric bursitis, patellofemoral dysfunction, sacroiliac dysfunction, and muscular imbalances.
Examination: Measurement from anterosuperior iliac spine to the medial malleolus bilaterally to assess for "true leg length discrepancy" and umbilicus to medial malleolus bilaterally to assess for "functional leg length discrepancy." Can also bridge the pelvis to determine if the discrepancy is femoral or tibial. Pelvic obliquity, reproducibility of measurement can make this method imprecise.
Radiographic measurements: Standing postural studies; standing anteroposterior view of the pelvis to include the femoral heads and iliac crests. Then measure from bottom of film to measure discrepancy (more accurate). Does not discern exact location of discrepancy (tibia vs. femur), but this is usually of little consequence in management.
Treatment: Once discrepancy defined, **heel lift** incorporated into shoe to correct. **Usually correct for approximately 60% to 75%.** If discrepancy greater than 1.5 cm, modify shoe externally.

Patellar Tendinosis

Description: Common, especially in activities requiring jumping.
Symptoms: Pain along infrapatellar tendon, inferior pole of patella, or insertion of patellar tendon into tibial tubercle; pain during knee extension, especially terminal extension.
Treatment: Ice, stretching, NSAIDs, modalities (phonophoresis, iontophoresis), decreased activity. Can use alternate cardiovascular equipment (bicycle, aqua arc, swimming, aqua-jogging). Avoid activities that aggravate pain, such as jumping and plyometrics.

Infrapatellar Fat Pad Impingement

Presentation: Anterior knee pain, similar in presentation to patellofemoral syndrome, except typically worse with standing and rapid knee extension. Often associated with an acute hyperextension injury.

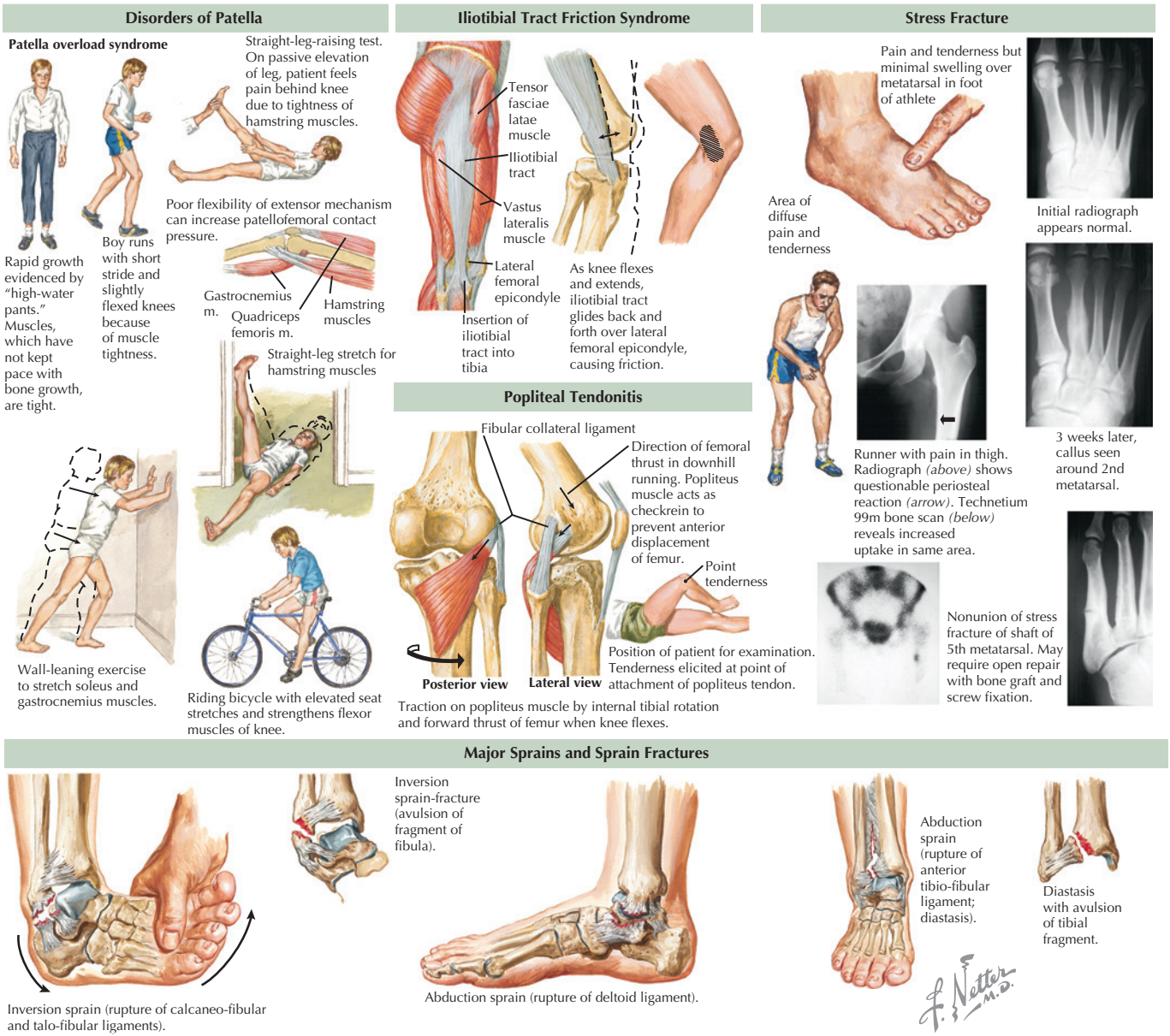


Figure 65-5 Lower Extremity Injuries.

Examination: Reveals tenderness at or below the inferior pole of the patella, often worse with passive, ballistic hyperextension.
Treatment: Patellar taping, often by compressing the fat pad even further.

Meniscal Lesions

Description: Most common acute mechanism is twisting injury in association with jumping. **Meniscal tears associated with osteoarthritis.** Chronic degenerative meniscal tears occur later in life, usually with little or no associated trauma.
Symptoms: Joint line pain, lack of full extension, joint effusion that occurs slowly (vs. immediate effusion in cruciate ligament tears), inability to go down into squat or duck walk.
Examination: Positive joint line pain, "bounce home" test, hyperflexion, circumduction/scour maneuvers (figure-of-eight movement of foot with knee in hyperflexion), McMurray's maneuver, Apley's maneuver.
Diagnostics: Magnetic resonance imaging (MRI) sometimes helpful in determining extent of tear, associated injuries, likelihood of repair versus meniscectomy.
Treatment: If not causing mechanical obstruction: non–weight-bearing (weight-bearing if pain free), decrease in activity, NSAIDs, ice, maintenance of quadriceps and hamstring strength. Gradual reintroduction of activity with pain and swelling as well as mechanical symptoms (clicking, catching, locking) as guide for further conservative as opposed to surgical management. Sport specificity and individualization necessary for proper management.

Hamstring, Adductor, Quadriceps Strains

Description: Common, especially in sprinters.
Prevention: Little published data but studies suggest preseason hamstring flexibility and strengthening programs decrease hamstring strains. Risk factors retrospectively associated with adduc-

tor strain include relative adductor weakness and decreased hip abduction range of motion.

Presentation: Sudden pain in belly of affected muscle. Athletes often describe "pulling" or "tearing" sensation. Athletes often run through initial discomfort only to experience increasing pain and disability later.

Examination: Variable amounts of swelling and ecchymosis, depending on extent of muscle damage as well as exact location of injury. The size of hematoma and cross-sectional damage can be assessed with MRI, though it does not correspond well with future risk of injury.

Most common hamstring injury: Short head of biceps femoris. Most often occurs at proximal musculotendinous junction. As the athlete's velocity increases, the hamstrings take an increasingly active role in force production, leaving them prone to injury. Injuries typically occur during the eccentric phase of high-velocity movements. Although data speculative, incidence of hamstring strains appears to increase with inadequate flexibility, quadriceps-hamstring muscle imbalances, inadequate warm-up, prior injury, poor proprioception, and fatigue.

Treatment: All of these are extremely difficult to treat. Initial management: compressive wrap, ice, stretching. **Subsequent management:** NSAIDs, possibly ultrasound if scarring occurs. **Rehabilitation** should emphasize eccentric activity, reproduction of sport-specific demands, maintenance of adequate ROM.

Prognosis and return to play: Gradually as long as athlete has full range of motion, full strength, and normal functional testing. Recurrence common and often frustrating for athlete. Progression back to full activity must be slow. Use of sport-specific progression helpful.

Lower Extremity: Lower Leg, Foot, and Ankle

Medial Tibial Stress Syndrome

Description: Pain over medial third of posteromedial tibia; association with excessive pronation.

Presentation: Area of tenderness often located in soleus muscle, which is involved in inversion of subtalar joint and plantar flexion of ankle joint.

Diagnostics: X-ray findings have poor sensitivity (10%) in the early stages of stress reactions. Findings are typically very subtle, and include thickening of medial tibial cortex common on plain radiographs; myositis and tendinosis evident on bone scan (differentiated by increased blood pool phase). Order further imaging if clinical suspicion is high. Scintigraphy helps identify stress fracture. Plain radiographs may show evidence of stress fracture after 14 days. Bone scan is very sensitive, but lacks specificity (statistics depend on site of fracture). Computed tomography (CT) scans with thin cuts over the suspected area are an option. However, false negatives are common and stress reactions are not well-visualized. Radiation exposure is involved. MRI has several advantages; no radiation exposure, multiplanar imaging, high-sensitivity, precise location of abnormalities, and ability to detect early pathology. Some reports of false-positive MRI in asymptomatic patients. Order fat-suppressed or short tau inversion recovery (STIR) images. May also have the radiologists use MRI-visible markers to localize the painful area. Cost of bone scan and MRI varies based on institution.

Associated injuries: Progression from medial tibial stress syndrome to stress fracture is major concern; treatment is aimed at preventing this.

Treatment: Ice; assessment of biomechanical abnormalities, specifically need for orthotics; eccentric strengthening of antagonistic muscle groups and flexibility exercises, relative rest with alternative methods of training useful (cycle, swim, aqua-jogging, Zuni unloader). Pain useful guide in management. Day-to-day, week-to-week adjustments in training regimen often necessary. Athletes may continue pain-free activities (swimming, cycling, hand-cycle, pool-running, ellipticals). Return to play is dictated by patient's ability to be pain-free during activities. Avoid NSAIDs (felt to inhibit osteoblast activity).

Fractures and Stress Fractures

Description: Typically associated with mileage of more than 20 miles per week, changes in training volume and intensity, or underlying bone abnormalities (see Fig. 65-5).

Treatment: Depends on site, extent, and circumstances. Discontinue the offending activity, unload the affected extremity (walker-boot, crutches, even wheelchair if bilateral involvement). Analgesia—NSAIDs have been shown to inhibit osteoblast activity in vitro in a rat model. Therefore, a non-NSAID may be a wiser choice. Consider sports psychologist and nutritionist for support. Locate and correct any biomechanical abnormalities with orthotics, shoe-type changes, assess shoe age, and consider physical therapy for stretching and strengthening. Bisphosphonates should be used with extreme caution given the negative impact on reproductive health, very little role in women of reproductive age. Navicula, tibia, and metatarsals are most common sites in track athletes. Distance runners are prone to tibial and fibular stress fractures. Studies suggest females are more susceptible than males. Likely multifactorial; female athlete triad, baseline fitness level differences, training errors body geometry differences have all been cited as possible causes.

TUBEROSITY AVULSION FRACTURES

Description: Most common fracture of proximal fifth metatarsal (Table 65-1). Often occurs where plantar aponeurosis or peroneus brevis attaches. Fracture displacement rare.

Evaluation: Look for associated lateral malleolus fracture. Differentiate from unfused tuberosity apophysis by smoothness and orientation of fracture line (fracture often perpendicular to shaft of bone). Differentiate from os peroneum by smoothness of edges and often bilaterality.

Treatment: Primarily symptomatic. Can immobilize with cast or molded hard-sole shoe for 1 to 2 weeks, followed by protected weightbearing to tolerance and close follow-up.

JONES FRACTURE

Description: Acute forefoot injury without prodrome; however, likely an acute-on-chronic phenomenon in an area of consistent microtrauma. Occurs at junction of metaphysis and diaphysis; no intermetatarsal extension.

Treatment: Varies. Conservative management with casting for 6 to 8 weeks. If not improved, surgical consultation should be ordered.

PROXIMAL FIFTH METATARSAL DIAPHYSEAL STRESS FRACTURES

Description: Fracture resulting from repetitive cyclic forces applied to foot.

Presentation: Can have prodromal symptoms or acute or chronic presentation.

Classification: Torg classification helps differentiate stress fractures by potential to heal (see Table 65-1).

- Acute, early diaphyseal stress fracture (periosteal reaction)
- Delayed union (lucent fracture line and medullary sclerosis)
- Nonunion

Treatment: Depends on patient's needs and expectations.

- Type I: Treat as acute nondisplaced Jones fracture.
- Type II: Treat operatively with bone graft or medullary screw fixation.
- Type III: Treat operatively; some prefer closed cannulated medullary screw fixation, others open methods.

Table 65-1 FOOT FRACTURES

Fracture type	Mechanism	Location	Chronicity	Prognosis
Avulsion	Hindfoot inversion	Tuberosity	Acute	Excellent
True Jones	Forefoot adduction	Metaphyseal-diaphyseal junction	Acute	Good
Diaphyseal stress	Cyclical loading	Proximal diaphyses		
Torg type I		Narrow fracture line, no medial sclerosis	Acute or chronic	Fair-good
Torg type II		Wide fracture line, some medial sclerosis	Delayed union	Variable
Torg type III		Complete medullary sclerosis	Nonunion	Variable

Adapted from Quill GE: Fractures of the proximal fifth metatarsal. Orthop Clin North Am 26:353-361, 1995.

OTHER STRESS FRACTURES

Description: Runners account for 69% of all stress fractures because of increased stresses on normal bone and normal to increased stresses on weakened bone. Occur throughout the lower extremity: tibia (34%), fibula (24%), metatarsals (18%), femur (14%), pelvis (6%).

Evaluation and treatment: Pay particular attention to the location of femoral neck fractures. Fractures occurring on the superior/tension side of the femoral neck require prompt surgical evaluation. Fractures on the inferior/compression side of the femoral neck can be managed more conservatively.

Associated injuries: Associated with gait abnormalities, leg length discrepancy, poor surface, incompletely rehabilitated prior injuries, reduced bone mineral density, training errors, and abrupt changes in volume or intensity of training.

Peroneal, Anterior Tibialis, and Posterior Tibialis Strains

Description: Common overuse injuries as well as acute overload injuries and strains. **Usually caused by training errors,** increase in training, change in shoes, poor flexibility.

Diagnostics: Can see avulsions or partial tears, especially of peroneal tendons. Peroneal tendons run in common tendon sheath, just posterior to lateral malleolus, then bifurcate and run in own tendon sheaths. MRI helps evaluate significant tendon injuries. Tendinosis rarely detected by MRI. Musculoskeletal ultrasound is increasingly used to diagnose these types of soft tissue injuries, but is not yet standard of care.

Treatment: Ice, stretching, addressing training errors or foot and ankle biomechanics.

Plantar Fasciitis

Description: Microtears caused by traction of plantar fascia and associated structures at calcaneal insertion.

Presentation: Heel or arch pain, often worst with first steps after getting out of bed.

Examination: Seen in both pes planus and pes cavus, although latter more common. Tight gastrocnemius-soleus complex and plantar flexors as well as excessive pronation increase risk of developing plantar fasciitis. Pain to palpation along plantar fascia, often at insertion into calcaneus. Pain with resisted toe flexion and passive toe extension.

Imaging: Radiographs may demonstrate heel spur, but this often presents bilaterally despite unilateral symptoms.

Treatment: NSAIDs, ice massage, arch stretches, heel cushioning, counterforce taping, phonophoresis or iontophoresis, corticosteroid injection useful if other modalities not helpful. Surgery to release the plantar fascia in severe, refractory cases. **Night splints** useful to keep plantar fascia at length by placing the foot at 90 degrees of dorsiflexion. Stretching of gastrocnemius-soleus complex and hamstrings also important as well as intrinsic strengthening of foot musculature. **Arch supports or orthotics if other rearfoot abnormalities present.**

Heel Pain: Fat Pad Contusion, Heel Spurs

Description: Common in hurdlers, jumpers, and endurance runners.

Presentation: Pain along fat pad or at insertion of plantar fascia into calcaneus. Patients often have history of increased training or increased wear of shoes without replacement.

Evaluation: Consider calcaneal stress fracture in the differential. Exaggerated heel strike in gait common.

Treatment: Ice massage; NSAIDs; shoe modification, including soft cushioning, replacement of worn shoes (recommend changing shoes every 6 months or 300 to 500 miles).

Achilles Tendinitis and Tendinosis

Description: Common in events involving jumping or landing as well as sprinting. Tendonitis represents an inflammatory process, whereas tendinosis refers to chronic intratendinous degenerative changes without acute inflammation. Pathology reveals no inflammatory cells involved.

Evaluation: Pain often approximately 2 cm proximal to insertion of achilles tendon into calcaneus or distal tendon. Palpation of nodularity may signify mucinous degeneration of tendon and/or partial tear. Can palpate crepitus along tendon sheath with passive or active motion ("squeaking tendon"). Pain along tendon, with resisted plantarflexion and passive dorsiflexion. Positive **Thompson test** signifies complete tear. Patient is placed prone and the gastrocnemius is squeezed, which creates an automatic plantarflexion of the foot. A positive test occurs if no plantarflexion occurs. Test with knee in full extension as well as 90 degrees of flexion to confirm the diagnosis.

Treatment: Ice; relative rest; NSAIDs; bilateral heel lifts to put tendon at rest; eccentric rehabilitation program that emphasizes stretching, return to functional activities, slow progression. Modify activities to pain tolerance. Assess for training errors or anatomic precipitants. Treatment of Achilles rupture should be individualized. For competitive athlete, surgical treatment may be better option for earlier functional recovery. Surgical debridement sometimes considered for recalcitrant tendinosis.

Ankle Sprains

Description: Common in cross-country runners. Studies suggest that poor proximal neuromuscular control can predispose to injuries. This can be assessed with single-leg squat balance.

Mechanism of injury: Inversion mechanism most common with involvement of anterior talofibular ligament, calcaneofibular ligament, and posterior talofibular ligament, progressively (see Fig. 65-5).

Associated injuries: Watch for associated foot or metatarsal injury. Deltoid ligament less commonly involved. Distal anterior tibiofibular ligament involvement (syndesmotic sprain) portends more extensive injury with longer recovery time.

Evaluation: Same as other sports. A "**squeeze" test** where the examiner squeezes the tibia and fibula together and pain is repro-

duced, implies a syndesmotic injury ("high ankle sprain"). Radiographs should be obtained based on the Ottawa ankle rules.

Acute treatment: Ice, elevation, compression wrap, immobilization (if severe), NSAIDs, weight-bearing as tolerated by pain as long as normal gait biomechanics maintained. X-rays, including mortise views, to assess for fracture, generally an avulsion. Consider repeat films 7 to 10 days later if initial films negative and symptoms persist or worsen to avoid missing occult fracture.

Chronic treatment: Rehabilitation important for early return of motion and strength as well as proprioception and functional activity. Bracing or taping often allows early return to sport.

Chondral and Osteochondral Lesions

Description: Occur in conjunction with ankle sprains. Ankle most stable in dorsiflexed position, at risk for injury in plantarflexion. Chondral or osteochondral injuries can occur, most commonly at talar dome, less commonly tibial plafond. Can also develop loose bodies.

Presentation: Athletes present with pain, swelling, locking with activity. Often discovered when presumed ligamentous injury fails to respond to appropriate treatment.

Imaging: Plain radiographs often negative initially. Lesions better seen on CT or MRI.

Treatment: Depends on location and size of lesion or fragments and amount of pain and disability. Arthroscopic debridement, drilling, or removal of loose bodies sometimes necessary.

Hip and Spine

Hip and spine injuries **account for close to 20% of all injuries** in track-and-field athletes.

Spondylolysis and Spondylolisthesis

Description: Spondylolysis (defect in pars interarticularis) and spondylolisthesis (movement of vertebral bodies in the sagittal plane).

Presentation: Acute-onset back pain, usually worse with activity and specifically extension; pain to palpation along spine or often paraspinally; pain with extension, aggravated by one-legged extension.

Diagnostics: Initial films may be negative. Bilateral oblique films are used to assess the integrity of the pars interarticularis. Flexion and extension views to assess for presence and severity of spondylolisthesis and stability of the spine. MRI, bone scan with single-photon emission CT or plain CT, with thin cuts through the area of interest, if suspicion is high.

Treatment: Bracing if caught before x-ray evidence present; limitation of activity; ice; neutral spine stabilization strengthening with flexion-based exercises.

Return to play: Gradual increase in sport-specific activity as tolerated with pain as guide. Follow-up bone scans not helpful because scans remain positive for several months.

Muscular Imbalance

Description: Common cause of muscular strains and overuse injuries. Unclear cause but can see in association with old injury or as response to overload, muscular weakness, leg length discrepancy, or other asymmetric anatomic abnormalities. One theory is the "lower crossed syndrome," in which tight hip flexors and erector spinae are associated with weak gluteals and abdominals. This can lead to an anterior pelvic tilt and dysfunctional lower extremity biomechanics.

Treatment: Most difficult aspect of treatment is recognition of muscular imbalances. Treatment rests on flexibility of tight structures in addition to strengthening of weak structures. Correction of anatomic abnormalities or gait abnormalities is essential.

Herniated Nucleus Pulposus

Description: Less common injury in cross-country runners, more common in sprinters.

Presentation: Central back pain, worse with flexion; pain over disk space; true nerve root irritation can present with classic radicular pain is a typical presenting symptom, with typical positive straight leg raise test and signs of neural tension

Examination: Careful neurologic exam with thorough manual muscle testing and monofilament sensory testing to exclude neurologic compromise.

Diagnostics: Plain radiographs may show degenerative changes and disc space narrowing. Flexion and extension views assess spondylolysis, spondylolisthesis, or instability. MRI helpful in assessing soft tissue and disc itself; can demonstrate central or foraminal stenosis; also helpful in ruling out tumor, multiple sclerosis, or other space-occupying lesions. Discogram or electromyelogram (EMG) may be helpful if intrinsic disc disease, or if surgery is under consideration. EMG or nerve conduction studies (NCs) are useful diagnostic tools if radiculopathy is suspected.

Treatment: Relative rest, NSAIDs, muscle relaxants, ice, physical therapy modalities. Consider epidural steroid injections, opioids or oral steroids, depending on individual situation.

Return to play: Activity may be allowed if no neurologic compromise, full strength and motion are achieved, and pain is controlled. Every situation must be individualized.

Paraspinal Muscular Strains

Description: Common, especially in hurdlers. Often secondary to acute overuse or injuries related to strengthening program.

Presentation: Paraspinal pain.

Examination: Assess for presence of other low back pathology.

Diagnostics: Additional studies indicated if no response to initial treatment or guided by history or physical examination.

Treatment: Initial treatment: ice, NSAIDs, stretching. Heat and ice for symptom reduction. Abdominal as well as neutral spine strengthening program once acute pain subsides. Flexibility program and trigger point injections may help.

ENVIRONMENTAL ISSUES

Heat Injuries

Heatstroke: Body temperature above 104° F, altered mental status, absent sweating, seizures and coma. Can be fatal, accounting for hundreds of deaths in the United States annually. Treat as medical emergency. Assess renal, neurologic, hepatic systems, and hospitalize.

Heat exhaustion: Temperature does not exceed 104° F; no mental status changes. Symptoms include profound weakness, dizziness, syncope, muscle cramps, nausea. Risks similar to heatstroke if unrecognized. Treatment often similar: assessment by physician, rest in cool environment out of the sun, oral fluids. Activity should be restricted for remainder of day, and athlete should be reevaluated on following day.

Heat cramps: Cramps often in abdomen and lower extremities; often related to fluid or electrolyte deficiency. Treat with fluid and electrolyte replacement.

Heat syncope: Decrease in vasomotor tone and venous pooling (especially immediately after stopping exercise), which leads to syncope; dehydration and increased temperature contribute.

Heat tetany: Spasm, often in wrist, seen with heat; exacerbated by hyperventilation.

Heat edema: Swelling of hands and feet in response to heat; often worst in initial phases of accommodation to new environment; self-limited; often resolves with cold compresses and elevation. Diuretics not indicated and may exacerbate dehydration.

Risk factors: Very young or very old age; preexisting dehydration; obesity; history of previous heat-related illness, heart disease, alco-

hol use, drugs (tricyclic antidepressants, amphetamines, LSD, PCP, cocaine, anticholinergics (previously discussed), antihistamines, diuretics, beta-blockers); increased humidity, temperature, no clouds, no wind; poor physical conditioning; acute febrile illness.

Treatment: Focuses on rapid cooling and timely transport to medical facility. If rectal temperature is above 104° F, cooling should occur immediately, with goal of reaching temperature of 102° F within 30 to 60 minutes. Airway protection must be ensured. Electrolyte abnormalities common.

Cold Weather Injuries

- Heat production in body maintained by:
  - **Basal heat production:** Produced by metabolic processes; ineffective in maintaining body temperature when exposure to cold environment occurs.
  - **Muscular thermoregulatory heat:** Produced by shivering. Blood flow preferentially shunted to vital organs. Can increase heat production threefold to fivefold, but requires energy; also increases cooling of distal extremities and skin.
  - **Mild-to-moderate exercise-induced heat:** Produced by low-intensity exercise; can increase heat production fivefold. Uses low-energy requirements and can thus be sustained for long period of time.
  - **High-intensity exercise-induced heat:** Can increase heat production 10-fold, but because of higher energy requirements cannot be maintained long.
- **Specific cold weather injuries include:**
  - **Frostnip:** Reversible without permanent tissue damage; blanching of skin; slow, painless crystal formation on ears, face, toes, and fingertips; common in windy conditions.
  - **Chilblains:** Repeated exposure to cold water or wet cooling; red, hot, tender swollen extremity with numbness or tingling. Irreversible damage can occur to capillaries, muscles, and nerves innervating the extremity; may progress to gangrene.
  - **Frostbite:** Freezing of soft tissue; commonly involves nose, fingertips, ears, and toes. Symptoms include local pain, numbness, redness, superficial blistering. With increased severity of exposure, deeper blistering and involvement of deep soft tissue and bone. Common in winter months and with windy or wet conditions. Frostbite may be permanent if not treated quickly.
  - **Hypothermia:** Occurs when core body temperature drops below 95° F; significant cause of mortality.
- **Treatment of cold injuries:**
  - **Best treatment is prevention.**
  - Rewarming slowly to minimize the risk of tissue damage, after warm environment secured.
  - Removal of all wet clothing, proper measurement of core body temperature using oral, rectal, or auricular thermometer.
  - Use of warm whirlpool, analgesics, skin protection.
  - Other preventive measures: layered clothing; covering for head; protection of hands and toes; avoidance of wetness; proper nutrition and hydration; adequate warm-up before activity; breathing through nose to warm inspired air; avoidance of alcohol, nicotine, and other drugs. Never train alone.

Guidelines about Environmental Issues

- **American College of Sports Medicine Guidelines remain standard.**
  - Use of wet bulb globe temperature (WBGT) ideal.
  - WBGT = (0.7 Twb) + (0.2 Tg) + (0.1 Tdb); where Twb = wet bulb thermometer temperature, Tg = black globe thermometer temperature, Tdb = dry bulb thermometer temperature.
  - Use color-coded flag system (Table 65-2).

Table 65-2 COLOR-CODED FLAG SYSTEM

Color	Risk	Comments	Restrictions
Black	Extreme risk	WBGT > 82° F	Cancel race
Red	High risk	WBGT 73-82° F	Be aware; high-risk athletes should not run
Yellow	Moderate risk	WBGT 65-73° F	Heat-sensitive athletes should slow pace
Green	Low risk	WBGT 50-65° F	No restrictions
White	Risk for hypothermia	WBGT < 50° F	Hypothermia possible, especially if wet or windy

WBGT, wet bulb globe temperature.

SAFETY AND EQUIPMENT ISSUES

- For all events, maintain proper supervision of surface and runways.
- When multiple events are going on at one time, safety issues are paramount. This is especially true for throwing events, in which risk of injury to spectators (impalement) is significant.

Track Surface

- **Equipment issues**
  - Tracks generally oval with inner length equal to ¼ mile, 440 yards, or 400 m. Knowledge of athlete training surfaces is helpful.
  - Cinder, clay, or all-weather synthetic track often used for track surface itself.
  - Proper maintenance of surface as well as adequate drainage.
- **Safety issues**
  - Amounts of shock absorption and damping characteristics depend on surface used; these factors may play role in injury.
  - Banking of track may alter biomechanics of both inside and outside leg. Banking is increased in smaller tracks. Athletes should alternate directions routinely during training.

Jumping Events

- **Equipment issues**
  - Ramps for long jump, triple jump, pole vault, and javelin often use rubberized synthetic surfaces.
  - Maintenance issues same as for track surface.
  - Take-off boards need to be replaced frequently.
  - Landing pits usually contain slightly moist sand, which needs to be turned and maintained at a minimum of 12 to 18 inches deep.
  - Pits extend from 10 feet in front of and at least 20 feet beyond take-off board.
- **Safety issues**
  - Ramps that are not wide enough can cause injury, especially inversion ankle sprains.
  - Runways must be kept clear of foreign objects, spectators, and other obstacles.

Pole Vault (Sprinter with Pole)

- **Equipment issues**
  - Foam rubber padding around planting box as well as underneath bar.
  - Planting box with sawdust or sand inside.
  - Crossbar usually fiberglass.
  - Poles usually fiberglass (lighter, more expensive) or metal, up to 18 feet long.

- **Safety issues**
  - Poles can break during take-off or from contact with vaulting box.
  - Landing mats should cover vaulting box on all sides.
  - Thickness of mat should range from 28 to 36 inches.
  - Risk of injury to cervical spine or head and risk of fracture secondary to incorrect fall mechanism. Proper cervical-spine care and backboard usage technique is a must.
  - Missing pole plant into vaulting box can lead to shoulder subluxation, dislocation, and acromioclavicular joint injuries.
  - Proper technique and supervision are important.

Hurdles (Sprint Event with Obstacles)

- **Equipment issues**
  - Hurdle designed to tip over forward.
  - Soft top hurdles minimize contact injuries with hurdle during practice.
  - Protection of ankle, heel, and knee with padding minimizes injuries in practice.
- **Safety issues**
  - Lanes clear of foreign objects, obstacles, spectators.
  - Injuries can result from direct contact with hurdles on inside of trailing leg.

Throwing Events

- Raise biggest concern about injuries to both athletes and spectators.
- Examples: shot put, discus, hammer, javelin.
- Proper technique and supervision essential.
- Proper warm-up helps prevent overuse injuries.
- Concern for spectators being struck by discus, javelin, hammer, or shot put.

RECOMMENDED READINGS

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Gymnastics

66

Amy Jo F. Overlin and Suzanne Hecht

OVERVIEW

- Types of gymnastics: acrobatic, artistic, rhythmic, tumbling, and trampoline (Tables 66-1 to 66-4).
- More than 88,000 competitive gymnasts in the United States register yearly with USA Gymnastics; there are up to 3 million recreational gymnasts in the United States.
- Injury epidemiology:
  - Rates: 3.6 injuries per gymnast per year; 2.5 to 3.3 injuries per 1000 hours of training; 2.1 to 8.5 injuries per 1000 athletic exposures.
  - Higher incidence of injuries during dismounts and floor exercise.
  - Sprains most common injury, followed by strains.
  - Ankle/foot is most commonly injured body part.
  - Competition injury incidence is two times practice incidence.
  - Higher incidence of growth plate injuries due to immature skeletal system.
  - Injury risk factors:
    - Larger size
    - Rapid growth
    - Training more than 15 to 20 hours per week
    - Life stress
  - Gymnasts are both lower extremity and upper extremity weight-bearing athletes; therefore any injury of the upper extremity will be compounded by the added forces required by gymnastics participation.

Table 66-1 ACROBATIC GYMNASTICS (MEN AND WOMEN)

Events	Levels	
Women's pairs Men's pairs Mixed pairs Women's group Men's group	**Junior Olympic** Levels 1-10 Competitive levels: 4-10 First competitive level: 4	**Elite** Junior Senior

See www.usa-gymnastics.org.

Table 66-2 ARTISTIC GYMNASTICS

Events		Levels	
Women	**Men**	**Women**	**Men**
Vault Uneven parallel bars Balance beam Floor exercise	Floor exercise Pommel horse Still rings Vault Parallel bars High bar	Junior Olympic Program Levels 1-10 Competitive levels 3-10 First competitive level: 3 Elite Program Talent Opportunity Program (TOPS) HOPES (10-12 yo pre-elite) Junior Pre-Elite Junior and Senior International	Junior Olympic Program Levels I-X Competitive levels: IV-X First competitive level: IV Elite Program Future Stars Program Junior National Team Senior Elite Team

Table 66-3 RHYTHMIC GYMNASTICS (WOMEN ONLY)

Events	Levels	
Rope Hoop Ball Clubs Ribbon	**Junior Olympic** Levels 1-8 Competitive levels: 5-8 First competitive level: 5 Individual and group competitions	**Elite** Junior Senior Individual and group competitions

Table 66-4 TUMBLING AND TRAMPOLINE (MEN AND WOMEN)

Events	Levels	
Double mini-trampoline Synchronized trampoline Trampoline Tumbling	**Junior Olympic** Levels 1-10 Competitive levels: 1-10 First competitive level: 1 (except synchronized trampoline: level 10)	**Elite** Junior Senior

SPECIFIC INJURIES

Mild Traumatic Brain Injury (MTBI)

Mechanism of injury: Hitting head on mat/floor or apparatus during fall or dismount (see Chapter 39, Head Injuries, for MTBI presentation, diagnosis, and treatment).

Return to play: When treating MTBI in a gymnast one must remember that although it is not a traditional contact sport, gymnasts make contact with the apparatus and mats frequently. In addition the aerial nature of gymnastics predisposes the athlete to catastrophic injury even when not concussed. Therefore, a more conservative approach such as one used with contact sports should be instituted.

Cervical Spine Fracture, Subluxation, and Dislocation

Mechanism of injury: Complex aerial and acrobatic nature of gymnastics places athletes at risk of catastrophic neck injuries. Cervical spine fractures, subluxations, and dislocations can occur through various mechanisms. Examples include the following: landing head first in loose foam pit, on trampoline, or on mat; failure to complete rotation on aerial or saltos; over rotating saltos; landing on upper back with neck in hyperflexed position; landing on chin or chest with neck in hyperextended position.

Specific consideration to standard evaluation and treatment: Because most gymnasts are small and an adult cervical collar may be too large, keep in mind the following when a cervical spine injury occurs in loose foam pit:

- Stabilizing neck and removing athlete with cervical spine injury from loose foam pit is difficult, because foam blocks are easily disturbed and athlete is buried in the blocks.
- Because of the difficulty of removing a gymnast with a cervical spine injury from a loose foam pit, physicians, trainers, coaches, and local paramedics should practice emergency removal as part of an emergency action plan.
- Coaches and athletes should be warned to avoid jumping into pit to help injured athlete because they may disturb foam

blocks and make injury worse and make it more difficult to removal the athlete. Gently placing a mat into the pit and then using this as a means to reach the athlete is one method to minimize disturbing the foam blocks.

Shoulder Injuries

Anterior Dislocation and Multidirectional Instability

See Chapter 43, Shoulder Injuries.

Elbow Injuries

Dislocation

- See Chapter 44, Elbow Injuries.
- Upper extremity weight-bearing activities can be introduced gradually, once gymnast has full range of motion (ROM) and strength of upper extremity and is pain-free.

Ulnar Collateral Ligament (UCL) Sprain

Mechanism of injury: Valgus stress to medial aspect of elbow causes traction injury to ulnar collateral ligament (UCL); may occur during fall from or onto apparatus with arm outstretched or during performance of skills.

History: Valgus mechanism; may be acute or chronic.

Physical examination: Findings typical of UCL injuries (see Chapter 44, Elbow Injuries). Gymnasts may have increased carrying angle and elbow hyperextension bilaterally, which may be risk factor for this type of injury.

Imaging: X-rays are important in acute injuries to rule out medial epicondyle apophyseal avulsion fracture in young athletes. For chronic cases, radiographic findings consistent with medial epicondylar apophysitis can confirm the diagnosis although the x-rays may be entirely normal. Magnetic resonance imaging (MRI) arthrogram may be needed to determine degree of ligamentous tear.

Treatment: Surgery is often indicated for complete rupture of UCL with resultant instability.

Complications: Chronic instability, ulnar neuritis.

Capitellar Osteochondritis Dissecans (OCD)

Mechanism of injury: Mechanism is similar to that proposed for capitellar osteochondritis dissecans (OCD) in throwing athletes, but in gymnasts, repetitive weight bearing causes valgus stress with medial elbow tension and lateral radiocapitellar joint compression.

History: Gradual onset, elbow pain with weight-bearing activities; pain relieved by rest. Gymnast may notice decrease in elbow extension. In more advanced cases, athlete may complain of catching and locking.

Physical examination: Tenderness to palpation over radiocapitellar joint; effusion may be present; ROM, particularly extension, may be decreased.

Imaging: X-rays may be negative or show rarefaction or radiolucency within the capitellum, irregular ossification and crater next to articular surface. MRI arthrogram helps determine integrity of articular cartilage.

Classification and treatment of OCD lesions: Type I: No displacement of lesion and no fracture of articular cartilage (Fig. 66-1).

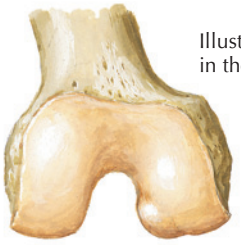
- Treatment: No weight-bearing activities or upper extremity strengthening activities until radiographs show evidence of healing and pain resolves completely. Splint may be needed for treatment of pain not relieved by discontinuing upper extremity weight-bearing activities or to guarantee compliance if there are concerns regarding compliance with treatment plan.

- Type II: Evidence of fracture of articular cartilage or partial displacement of lesion (see Fig. 66-1).
  - Treatment: Controversial; ranges from conservative to surgical intervention.
- Type III: Complete detachment of lesion with resulting loose body (see Fig. 66-1).
  - Treatment: Surgical removal of loose body with or without drilling or curettage.

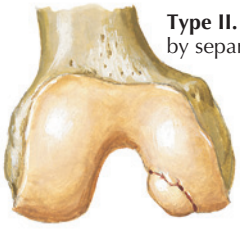
Complications: Loss of ROM, degenerative changes, chronic pain; can be career ending.

Grip Lock

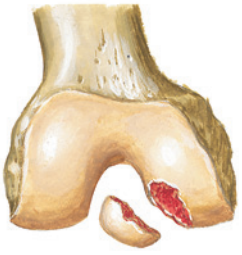
Mechanism of injury: When gymnast performs circling elements on uneven bars or horizontal bar, overlapping of leather grip against itself causes grip to lock or catch in place. Instead of allowing gymnast to circle freely around bar, hand is stuck in position as body continues to swing, causing forearm to "wrap around the bar" and fracture.



Illustrations of OCD in the distal femur.

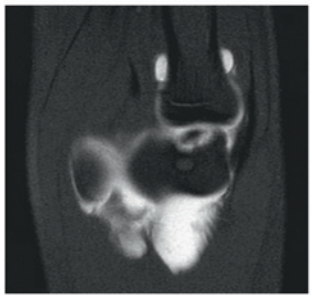


Type II. Fragment demarcated by separation of articular cartilage.

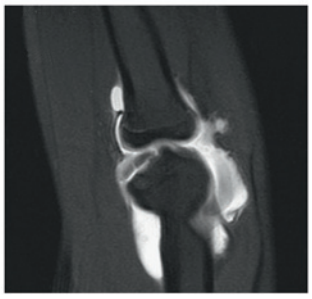


Type I. Bulge on medial femoral condyle due to partial separation of bone fragment. Articular cartilage intact, but defect evident on radiographs.

Type III. Fragment of cartilage and bone completely separated as loose body. This often migrates to medial or lateral.



OCD elbow coronal T1.



OCD elbow sagittal T1.

Figure 66-1 Capitellar Osteochondritis Dissecans (OCD).

Evaluation and treatment: Appropriate for type of fracture(s).
Prevention:
- Regular replacement of grips as the leather stretches out over time.
- Gymnasts or coaches should make routine checks to ensure that grips are not long enough to overlap around bar.
- Avoid sharing of grips between gymnasts.
- Use caution if gymnast has been training on larger-diameter bar and then switches to smaller-diameter bar.

Wrist and Hand Injuries

Distal Radial Physeal Stress Injury

Definition: Stress injury to distal radial physis.
Mechanism of injury: Thought to result from repetitive overuse of wrist as weight-bearing joint. Forces between two to six times the athlete's body weight can load the wrist joint during gymnastics maneuvers.
Prevalence: There are 2.7 distal radial physeal stress injuries per 100 gymnasts per year.
History and physical examination: Tenderness over distal radial physis, pain reproduced with wrist hyperextension and axial loading, wrist extension may be decreased.
Imaging: Radiographic and MRI findings suggest chronic stress injury to physis (Table 66-5). Consider x-rays of contralateral wrist to compare physes for subtle differences. Initial x-rays may be negative. Consider MRI for a more detailed evaluation of the physis.
Treatment:
- Decrease or eliminate upper extremity weight-bearing activities until pain resolves, which may take up to 12 weeks.
- Casting or bracing may be considered, particularly if having pain outside of gymnastics activities. A removable wrist brace worn during practice can often serve as a "reminder" for both the gymnast and the coach to avoid weight-bearing activities.
- Physical therapy to address any flexibility and strength deficits of the wrist and finger extensors and flexors. Other areas that are lacking flexibility, such as the shoulders, and might be contributing to the wrist pain can also be addressed.
- Serial examinations are helpful.
- Weight-bearing activities should be resumed gradually once pain subsides.
- Coach should review and correct poor technique on return to weight-bearing activities.
- Specialized wrist splints (e.g., Ezy Pro brace, Lion-Paws, or Teurlings wrist brace) may be recommended on return to gymnastics activities.
  - The Ezy Pro wrist brace has been shown in a cadaver study to help decrease wrist hyperextension and ulnocarpal joint forces when an axial load is applied to a pronated, extended wrist.

Complications:
- Chronic wrist pain
- Abnormal closure of physis
- Positive ulnar variance (ulna longer than radius), diagnosed by either x-rays or MRI
- Triangular fibrocartilaginous complex tears

Table 66-5 RADIOGRAPHIC FINDINGS CONSISTENT WITH DISTAL RADIAL PHYSEAL STRESS INJURY

X-ray	Magnetic resonance image
Growth plate widening	Growth plate widening
Haziness within physis	Lack of physeal homogeneity
Cystic changes on metaphysis	Physeal cartilage extension
Beaking of epiphysis	Metaphyseal "bone bruise"
	Linear striations

- Degenerative changes in triquetrum, lunate, and ulna
- Alterations in radioulnar articulation
- Extensor tendon ruptures

Scaphoid Stress Fractures

Mechanism of injury: Chronic overuse injury—repetitive upper extremity weight bearing causing dorsiflexion and abduction of the wrist, leading to increased forces across the radio-scaphoid articulation.
History and physical exam: Chronic radial-sided wrist pain, worse with upper extremity weight-bearing activities. Tenderness to palpation over the dorsum of the scaphoid or anatomical snuff box.
Imaging: X-ray may show sclerosis of the scaphoid waist or a fracture line. X-rays can be negative. MRI shows edema of the scaphoid consistent with a stress reaction with or without a definitive fracture line.
Treatment:
- Eliminate upper extremity weight bearing.
- Thumb spica cast for 8 to 12 weeks.
- After cast removal and resolution of pain, begin wrist and forearm strengthening with gradual return to upper extremity weight-bearing activities.
- Specialized wrist splints (e.g., Ezy Pro wrist brace, Lion-Paws, or Teurlings wrist brace) may be recommended on return to gymnastics activities.
  - The Ezy Pro© wrist brace has been shown in a cadaver study to help decrease wrist hyperextension and ulnocarpal joint forces when an axial load is applied to a pronated, extended wrist.
- Depending on clinical presentation and/or failure of conservative treatment an ORIF (open reduction, internal fixation) to place a screw across the scaphoid may be warranted.

Calluses, Blisters, and "Rips"

Mechanism of injury: Many events in gymnastics require hanging, swinging, and support movements on hands while gripping apparatus. Friction between hands and apparatus causes soft-tissue injuries to hands. Even with use of leather grips and chalk to decrease friction, skin develops calluses in response to repetitive forces. Calluses can tear ("rips") or blister.
Treatment:
- Prevention: Calluses should be shaved down on regular basis to decrease frequency of "rips."
- Most important principle of treatment is to avoid infection.
- Torn skin from "rip" should be trimmed away.
- Blister and "rips" should be cleaned thoroughly with soap and water to decrease risk of infection and remove excessive chalk.
- Appropriate protective padding and wrapping should be applied to prevent irritation and infection until athlete can tolerate friction in area.
- Consider using DuoDerm Extra Thin CGF Spot Dressing.
  - Apply to area of rip.
  - Athlete may attempt to swing bars with dressing in place, held on with athletic tape under grips.
  - Change dressing after each bars practice or every 7 days if bars are not being practiced.
  - Drawback: treatment can be expensive.
- Gymnasts should not share scissors to trim away torn skin, unless scissors are disinfected between uses.

Complications: Infection and other injuries related to decreased ability to grip the bar ("peeling off") secondary to pain.

Spondylolysis and Spondylolisthesis

Epidemiology: Female gymnasts have 10% to 11% incidence of pars defects. Rhythmic gymnasts may be at higher risk because of extreme lumbar hyperextension required. See Chapter 47, Thoracic and Lumbosacral Spine Injuries, for presentation, diagnosis, and treatment.

Table 66-6 PELVIC APOPHYSES

Apophysis	Attachment(s)	Age of appearance (yrs)	Age of fusion (yrs)
Anterior superior iliac spine (ASIS)	Sartorius	13-15	21-25
Anterior inferior iliac spine (AIIS)	Rectus femoris	13-15	16-18
Ischial tuberosity	Hamstring	15-17	19-25
Lesser trochanter	Iliopsoas	8-12	16-18
Greater trochanter	Gluteus medius	2-5	16-18
Iliac crest	Internal and external obliques Transverse abdominis Tensor fascia lata Gluteus medius	13-15	15-17

Pelvic Apophyseal Injuries

Definitions: Apophysis: Growth plate that adds contour and shape to bone without contributing to bone length (Table 66-6). **Apophysitis:** Irritation of apophysis. Areas include ASIS, AIIS, ischial tuberosity, lesser trochanter, greater trochanter, and the iliac crest (see Table 66-6).

Mechanism of injury: Acute: Sudden, powerful contraction of involved muscle, avulsing apophysis. **Chronic:** Repetitive tensile forces from eccentric muscle contractions, placing traction on involved apophysis.

History: Acute: Sensation of popping with resultant pain in hip; pain may be severe. **Chronic:** May be described by gymnast as "hip flexor strain"; usually painful to lift or hold legs in pike or straddle position.

Physical examination: Apophyseal tenderness at involved site; decreased ROM and strength at involved site, particularly with acute avulsions; antalgic gait common with acute avulsions.

Imaging: Acute: X-rays reveal avulsion. **Chronic:** X-rays may be normal. Comparison x-rays of contralateral apophysis may reveal differences in ossification centers. MRI may help confirm diagnosis.

Treatment: In most cases, treatment is conservative. Extensive physical therapy may be required for acute avulsions. More significant injuries may require non–weight-bearing or partial weight-bearing. Surgical treatment tends to be reserved for significant degree of fragment displacement or chronic pain and disability despite adequate conservative treatment.

Knee Injuries

Tear of Anterior Cruciate Ligament (ACL)

Mechanism of injury: Landing without fully completing twisting elements, resulting in valgus, varus, or hyperextension stress to knee. Can also result from a pure hyperextension mechanism. See Chapter 49, Knee Injuries, for presentation, diagnosis, and treatment.

Tibial Tubercle Apophysitis (Osgood-Schlatter's Disease)

History: Pain with running, jumping, tumbling, and vaulting.

Education: Make sure gymnast and parents understand that condition is limited and will resolve once apophysis fuses. Persistent pain is uncommon. Explain that tibial tubercle prominence usually remains after symptoms have resolved.

Ankle Injuries

Inversion Sprains

Mechanism of injury: Inversion or inversion plantar flexion; landing with ankle/foot rolled under from jumps, saltos, and twists; also from stepping off landing mats or landing with foot in seam of mats.

Treatment: Comprehensive physical therapy program crucial for successful and safe return to gymnastics: ROM, strength, proprioception, and evaluation and correction of entire kinetic chain. Ankle taping/bracing may be required during practice/competitions until injured ankle is completely rehabilitated. Repetitive ankle sprains require evaluation for associated injuries or true ankle instability; although many recurrent ankle sprains result from inadequate rehabilitation.

Distal Fibular Physeal Fracture

Mechanism of injury: Inversion-plantar flexion.

History: Mechanism of injury as noted above; same history as an inversion ankle sprain but may have felt or heard a pop.

Physical examination: Tenderness over distal fibular physis (located at level of tibiotalar joint line). Swelling over lateral malleolus. Decreased ankle ROM and strength.

Imaging: X-ray to classify fracture. May need x-ray of opposite ankle to compare physes for subtle changes. X-rays may be negative for Salter-Harris type I fractures; diagnosis based on physical exam.

Treatment: Relative rest, ice if not in cast, elevation. Nondisplaced, low-grade Salter-Harris fractures are treated with short leg walking cast or walking boot for 3 weeks. ROM, strengthening, and proprioception exercises are needed after immobilization and before full return to gymnastics.

Anterior Ankle Impingement Syndrome

Mechanism of injury: Abutment of dorsal portion of talus and/or navicular against anterior lip of tibia. Results from gymnasts landing "short" (i.e., athletes under-rotate backward saltos and land in either a deep squat or so low that they must place hands down in front of them to prevent falling on knees or face). Over time bone responds by forming exostoses that produce anterior ankle joint pain with dorsiflexion. Cartilage overgrowths (synovial chondromatoses), chronic synovitis, and articular damage also can occur.

History and physical examination: History of repetitive short landings with chronic anterior ankle pain, dorsiflexion may be decreased, tenderness over anterior tibiotalar joint may occur only with ankle in dorsiflexion or during landing of saltos, and pain with forced dorsiflexion of ankle by examiner.

Imaging: Lateral ankle x-rays confirm exostoses, although may be negative if the impingement consists only of soft tissue (Fig. 66-2).

Treatment: Land on soft surfaces; avoid short landings; improve ankle flexibility. Conservative treatment generally provides limited improvement. Consider steroid injection to reduce associated synovitis. Many gymnasts require surgical intervention.

Posterior Ankle Impingement

Mechanism of injury: May develops after inversion ankle sprains, and or secondary to repetitively performing skills on ¾ pointe. Posterior impingement may be caused by a combination of:

- Hypertrophy or tear of posterior inferior tibiofibular ligament or transverse tibiofibular ligament.
- Pathology of the os trigonum-talar process, subtalar joint disease, fractures.
- Flexor hallicus longus tenosynovitis.

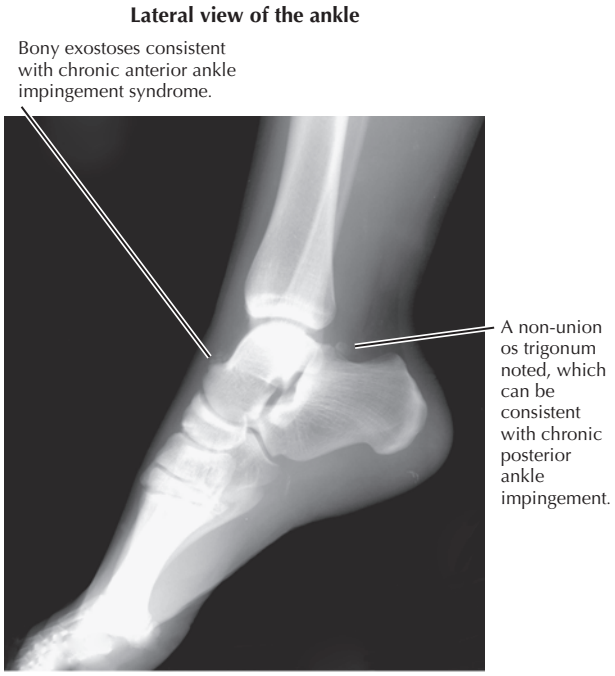


Figure 66-2 Ankle Impingement.

History and physical exam: Often fails conservative treatment; practices are very limited because of pain. Pain with plantar flexion, pain increases with skills that require gymnast to be on ¾ pointe or requires rebounding off ¾ pointe. Tenderness to palpation over posterior talus, posterior tibiofibular ligament, or os trigonum. Swelling to the same areas may also be noted. Posterior pain reproduced with forced plantar flexion by examiner.

Imaging: X-rays may show the presence of an os trigonum, or fracture of an os trigonum. It may be helpful to obtain a lateral ankle x-ray in full plantar flexion to assess for overlapping of posterior bony elements. MRI may be needed to reveal stress injury, posterior chondral injury, tenosynovitis, etc. (see Fig. 66-2).

Treatment: Physical therapy focusing on entire kinetic chain, flexibility, proprioception. Taping of ankle to minimize full plantar flexion. Corticosteroid injection around posterior talus. If a nonunion of an os trigonum is the source of pain consider surgical removal of os trigonum.

Foot Injuries

Calcaneal Apophysitis (Sever's Disease)

See Chapter 54, Foot Problems, for information on presentation and diagnosis of calcaneal apophysitis (Sever's disease).

Treatment: Relative decrease in jumping, running activities. Recommend all landings be performed on soft surfaces when at all possible. Coach should evaluate landing technique to ensure that gymnast does not land back on heels. Gastrocnemius-soleus complex stretching. Tape on heel cups for practice or use Tuli's Cheetah brace. Work-out in tennis shoes if symptoms are particularly bothersome.

Education: Make sure that gymnast and parents understand that condition is limited and will resolve once apophysis fuses.

Prognosis: Persistent pain is uncommon.

Calcaneal Contusion

Mechanism of injury: Landing over rotated dismounts, tumbling, and vaults with body weight back on heels or hitting heels on uneven, horizontal, or parallel bars during release or when swinging past low bar of uneven bars. Another mechanism involves performing a skill with poor direction and landing off the side of mat on gym floor. This trauma can cause contusion to calcaneus or heel pad. Calcaneal fracture is rare, although calcaneal stress fracture may develop from repetitive poor landings.

History and physical examination: Mechanism of injury as noted above, pain with ambulation, calcaneus tender to palpation and/or compression, possible localized heel pad swelling and ecchymosis.

Imaging: X-rays may be needed if calcaneal fracture or stress fracture is suspected. MRI may be useful to diagnose calcaneal contusion or stress fracture not seen on x-rays

Treatment:

- Ice massage.
- Padding or heel cup for comfort during weight bearing; walking boot and/or crutches (if very symptomatic).
- Heel cups can be taped on for practice, and/or knee pads (like those used in volleyball) can be worn as heel protectors on uneven, horizontal, or parallel bars; or use basket weave taping of the heel.
- Correction of poor landing technique and landing on soft surfaces are important to prevent reinjury.

FEMALE ATHLETE TRIAD

- Epidemiology: The prevalence of the female athlete triad in gymnasts is unknown. Studies have demonstrated that gymnasts, along with athletes in other aesthetic sports, are at increased risk for disordered eating and tend to have increased drive for thinness and high rates of body dissatisfaction. (See Chapter 10, The Female Athlete.)
- Gymnasts have higher bone mineral density, because of the impact loading forces of the sport, as compared to other athletes and nonathletic controls. This may offer some level of bone protection for gymnasts that suffer from one or more components of the triad.

GROWTH AND MATURATION

- Female gymnasts are short before beginning gymnastics.
- Studies suggest attenuated growth during training.
- Catch-up growth during reduced training or retirement.
- Studies unclear if "catch-up" is complete.
- Cause and effect of gymnastics training resulting in stunted growth has not been demonstrated.
- Gymnasts tend to undergo menarche later than the general female population.

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Road Biking

Marc R. Silberman

GENERAL PRINCIPLES

Races

Road Racing

Stage races: Multiday races on consecutive days with daily stage winners and overall winner based on cumulative time; mass start races where the athletes ride in a pack, or **pelenton. Grand tours:** Tour de France, Giro d'Italia, and Vuelta de España; include prologue, flat stages for "sprinters," hilly stages for "climbers," and time trials.

Road races: Mass start point to point races; start and finish in separate cities; 60 to 150 miles.

Circuit races: Multi-lap races of 60 to 150 miles, on 3- to 20-mile courses. The World Championships and Olympics are circuit races.

Criteriums: Multi-lap races of 25 to 50 miles, on 1-km to 1-mile courses, tight cornered roads through city towns. Crashes are common.

Time trials (TT): Individual races "against the clock"; riders start at 1- to 2-minute intervals on time trial bikes. Drafting prohibited. Distances 10 to 25 miles. **Triathlons** have TTs in lengths from 16 miles (sprint distance) to 112 miles (Ironman).

Team time trial (TTT): Individual teams together in a paceline.

Track Racing

Description: Held on a **velodrome** (track with banking, length 250 to 333 m); riders use **track bikes** (with no brakes and fixed gears).

Match sprint: One-on-one race of 1000 m with an explosive finishing sprint of 200 m.

Kilometer: Individual 1000 m time trial.

Pursuit: One-on-one 3000 to 4000 m time trial, where riders start on the opposite sides of the track. **Team pursuit:** Race involving teams of four riders, where cyclists draft closely, peel off the front, swing up the banking, and back down to the rear of the team.

Points race: Mass start event where points are awarded to the first five finishers of sprints.

Miss and out: Mass start event where one rider is eliminated per sprint lap, until final sprint lap between remaining riders.

Keirin: Mass start event of 2 km with six to nine sprinters competing at one time with a paced start behind a motorcycle, or **derny.** Initially slow pace, but ending at speed of 60 km per hour.

Madison: Named after Madison Square Garden. Teams of two compete, but only one rider races at any stage. The teams change riders by "tagging" their teammates, usually performing slingshot action with linked hands. The nonracing teammate then moves to the top of the track to recover.

Riders

Sprinters: High number of fast-twitch muscle fibers for explosive acceleration; reach speeds of 40 mph; produce more than 1200 watts at top end; take calculated risks in maneuvering through the pack, waiting until the last possible moment to move out of another rider's slipstream and into the wind.

Climbers: Lightweight, high power-to-weight ratio, $\dot{V}O_2$ max of 70 to 80 mL/kg/min.

Lead out riders: Break the wind for their sprinters until the very last possible moment by sustaining high speed for a kilometer; lack finishing top end speed of sprinters.

Time trialists: Ride steady state for long periods; larger cyclists who can push a big gear.

Team leaders: Riders for overall classification of stage races must be able to climb and time trial.

Domestiques: Sacrifice themselves for the sprinters and leaders by carrying water, blocking the wind, or even giving a wheel.

Organizations

USA Cycling (USAC): National governing body for bicycle racing in the United States. Covers the disciplines of road, track, mountain bike, cyclo-cross, BMX, and collegiate racing.

Union Cycliste Internationale (UCI): World governing body for cycling. Issues licenses, enforces disciplinary rules, manages the classification of races and points ranking system, oversees the World Championships.

Amaury Sport Organisation (ASO): Part of a French media group, is the organizer of the Tour de France.

International Olympic Committee (IOC): Organization that oversees the Olympics.

United States Anti-Doping Agency (USADA): A nongovernmental agency responsible for implementation of the World Anti-Doping Code in the United States. The World Anti-Doping Code, which lists drugs and methods that are prohibited in sports, was developed by the World Anti-Doping Agency (WADA).

World Anti-Doping Agency (WADA): Independent foundation created through a collective initiative led by the IOC. In November 1999, the World Anti-Doping Agency (WADA) was created to promote and coordinate the fight against doping in sports. In 2004, the **World Anti-Doping Code** was implemented by sports organizations prior to the Athens Olympics, standardizing the regulations governing anti-doping.

Court of Arbitration for Sport (CAS): International institution independent of any sports organization to facilitate the settlement of sports-related disputes through arbitration or mediation.

Epidemiology and Injury Statistics

- Survey of 81 cyclists in a well-established masters cycling club:
  - 81% with a racing license, average number of racing years 9.5, average annual mileage 6000 miles.
  - 79% seen in an emergency room, 33% admitted to hospital, 15% to intensive care unit.
  - 54% had sustained fractures: clavicle, 22; upper extremity, 20; ribs, 20; lower extremity, 11; vertebral, 11; pelvis, 6; skull, 6.
  - 45% reported a head injury; 34% reported concussion with 9% reporting more than one.
  - 75% reported breaking one or more helmets from a crash.
  - 90% reported having road rash.
  - 37% crashes involved motor vehicles, 9% road surface hazards, 12% skill errors, 10% mechanical problems.
  - Group riding injuries: 17% occurred while in a paceline, 12% during racing, more often criteriums.

Equipment and Safety Issues

- Bicycles should be inspected regularly. Tire pressure should be at proper amount, lower in wet road conditions.
- Protective gear
  - **Helmets** manufactured after 1999 must meet the Consumer Product Safety Commission (CPSC) standard by law to be sold in the United States. Twenty-one states have mandatory helmet laws. Helmets are designed for one crash

only. Write name, contact information, and medical information in helmet for emergencies.
- Other protective clothing: gloves, snug-fitting cycling wear, chamois padding in shorts, sunglasses.

Biomechanical Principles

Bicycle Anatomy

ROAD BICYCLE

- **Key frame measurements** are seat tube length, seat tube angle, and top tube length.
- **Key component measurements**
  - **Crank length:** Based on height or inseam length (Table 67-1). Too long may predispose to fatigue and knee ailment.
  - **Stem length and angle.**
  - **Handlebar width** should be close to width of shoulders.
  - **Handlebar tilt:** Bars can slip and rotate into downward tilt, which can cause excessive reach leading to hand, neck, and back symptoms.
- **Key bicycle measurements**
  - Saddle height = center of bottom bracket to height of saddle where rider sits
  - Saddle height and handlebar height difference
  - Saddle tilt
  - Saddle fore-aft
    - Plumb line from nose of saddle, measure distance behind bottom bracket
    - Distance from nose of saddle to handlebars

TRACK BICYCLE

- No brakes
- Fixed gear
- Major trauma risk with crashes

TIME TRIAL BICYCLE

- Steeper seat tube angle, of 78 to 84 degrees, aero bars, aero deep dish wheels or disc.
- Designed to go fast and straight.
- Goal is reduced frontal area, flat back, narrow arm position, and elbow flexion of 90 to 110 degrees with ear directly over elbow.
- Narrow position of arms does not restrict oxygen consumption and lung function.

Bike Fit

- Proper fit essential for comfort, safety, injury prevention, and peak performance.
- The goal: optimize power and aerobic efficiency while avoiding injury (Table 67-2).
- Static (measurements at rest) or dynamic (measurements while riding). Dynamic fit may involve video analysis with heart rate, wattage, and pedal torque readings. Wind tunnel testing: gold standard for finding the most efficient aero position.
- Changes should be made gradually.
- There are **three contact areas** a rider makes with the bicycle: shoe-cleat-pedal, pelvis-saddle, and hands-handlebar (Fig. 67-1).

SHOE-CLEAT-PEDAL INTERFACE

- First metatarsal head lies directly over the pedal axle (see Fig. 67-1).
- Leg length discrepancy: shims can be inserted under shorter leg or cleat may be moved forward (foot back). One-third to half of the difference should be corrected.
- Heel lifts and orthotics are not sufficient for cycling because the driving force is primarily through the first and second metatarsal heads. Varus forefoot wedges may be used.

PELVIS-SADDLE INTERFACE

- **Saddle height**
  - Traditional formulas designed to fit a rider for the most power at minimal aerobic cost.
  - Greg LeMond and Cyrille Guimard formula: rider's inseam length in centimeters × 0.883 = saddle height (see Fig. 67-1).
  - Knee angle method: The knee should be flexed 25 to 30 degrees from full extension, with the pedal in the 6-o'clock position (DBC = dead bottom center) (see Fig. 67-1).
- **Saddle fore-aft**
  - With the pedal positioned at 3 o'clock (KNOPS = knee over pedal spindle), a plumb line dropped from the inferior pole of the patella should hang directly over the pedal axle (see Fig. 67-1).
  - Sprinters and time-trialists may prefer a slightly more forward position so plumb line falls in front of axle.
  - Moving the saddle forward lowers the height, whereas moving it backward elevates the saddle.
  - To compete in a time trial with aerobars, a rider with one bike may move the saddle slightly forward and higher.
- **Saddle tilt**
  - Saddle tilt should be close to level.
  - About 60% of body weight centered on the narrow saddle.
  - Saddle sores (skin wounds secondary to moisture, pressure, and friction), perineal pain and numbness, or impotence may result if the saddle is not wide enough to support the ischial tuberosities or set to correct height and angle.
  - Time trialists with arms on aerobars prefer a slight downward tilt of the saddle.

HANDS-HANDLEBAR INTERFACE

- **Stem and handlebar height**
  - Stem height is a subjective measurement; important in terms of aerodynamics, power production, comfort, and injury prevention (see Fig. 67-1).
  - With the hands on the brake hoods and the arms slightly flexed, the torso should flex to about 45 degrees in relation to a nonsloping top tube.
  - When the hands are in the drops, the torso should flex to about 60 degrees.
  - The vertical distance or drop between the top of the saddle to the bars should be about 5 to 8 cm.
  - A recreational rider may prefer to sit more upright with a shorter reach and higher placed handlebars, for comfort.
  - An average-size male cyclist may decrease his frontal area by 30 degrees moving from the upright touring position to a racing position.
  - If forward-flexed excessively, maximal sustainable power may be reduced, because of diminished blood flow and/or changes in muscle lengths.

Table 67-1 CRANK ARM

Height (in.)	Crank length (mm)	Inseam (in.)	Crank length (mm)
<60	160	<29	165
60-64	165-167.5	29-32	170
65-72	170	32-34	172.5
72-74	172.5	>34	175
74-76	175		
>76	180		

Adapted from Burke ER: High-Tech Cycling, 2nd ed. Champaign, Ill: Human Kinetics, 2003.

Table 67-2 OVERUSE INJURIES: CONTRIBUTING BICYCLE POSTURE AND BICYCLE ADJUSTMENTS

Ailment	Contributing position	Bicycle adjustment
Posterior neck pain, may extend to head	Too great of a reach, handlebars too low, too stretched out	Ride more upright to shorten reach Raise stem height Shorten stem length Ride with hands on hoods or tops of bars
Scapular pain	Too great of a reach, handlebars too low, too stretched out	Ride more upright shorten reach Raise stem height Shorten stem length Ride with hands on hoods or tops of bars
Hand neuropathy (cyclist's palsy)	Too much pressure on bars, handle bars too low, saddle too far forward, excessive downward saddle tilt	Increase padding on bars and gloves Avoid prolonged pressure, change hand position often Raise stem height Move saddle back if too far forward If saddle is tilted down, position it level
Low back pain	Too stretched out	Ride more upright to shorten reach Raise stem height Shorten stem length
Tibialis anterior tendinopathy	Saddle height too high	Lower saddle height
Achilles tendinopathy	Saddle height too high (excessive stretch) Saddle height too low (with concomitant dropping of heel to generate more power)	Lower saddle height Raise saddle height
Morton's neuroma/foot pain/ numbness	Cleat position Irregular sole Shoes too tight	Usually, move cleat back, but may be forward Check sole for inner wear or cleat bolts pressing inward Wider shoes, loosen Velcro straps/shoe buckle
Perineal numbness	Saddle too high Tilt angle excessively up or down	Lower saddle height Adjust angle closer to level with the ground

From Baker A: Medical problems in road cycling. In Gregor RJ, Conconi F (eds): Road Cycling. Oxford, England: Blackwell Sciences, 2000, pp 18-45; and Silberman M, Webner D, Collina S, Shiple B: Road bicycle fit: Practical management. Clin J Sport Med 15(4):271-276, 2005.

- **Handlebar tilt** is a personal preference, but most cyclists prefer the lower curve and brake hoods to be slightly elevated.

- **Stem length or extension**
  - A rider's reach is determined by the top tube length, stem length, and stem angle or rise (see Fig. 67-1).

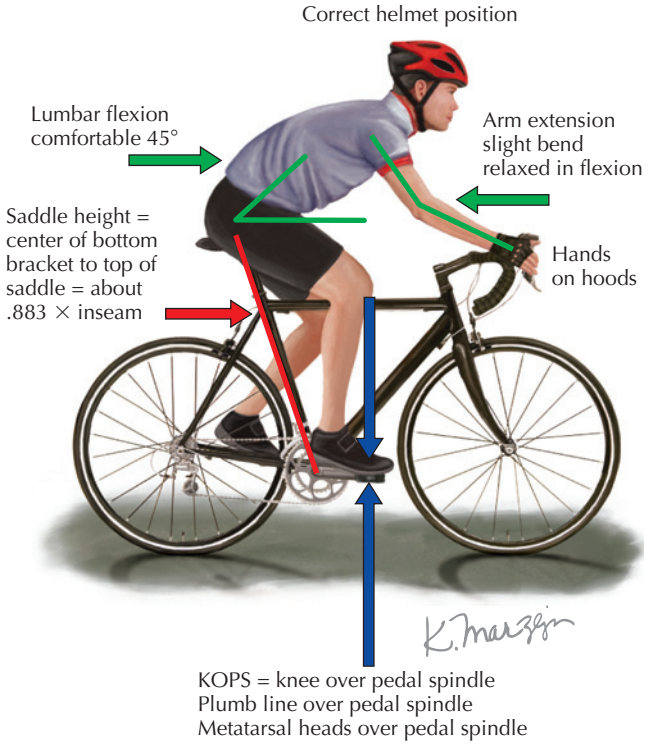

Figure 67-1 Road Bike Fit.

  - Too short a top tube or stem length and the rider will be bunched up. Too long, and the rider will be stretched out.
  - A good starting point: when the rider looks down with the arms slightly bent and the hands in the drops, the front hub should be obscured by the handlebars.
  - If the frame was properly fitted, the top tube length will allow an optimum position to be achieved with the use of a 10- to 12-cm stem.

TRAINING AND PHYSIOLOGY

Performance Testing

Conconi test: One of the first tests available to determine lactate threshold (LT) without directly measuring lactate; **pace at which linear correlation between heart rate and velocity is lost is called the deflection velocity or deflection point,** said to occur at LT; a ramp protocol of increasing watts is performed in the laboratory with HR measurement; numerous authors have found the Conconi test invalid.

Lactate threshold (LT) or anaerobic threshold (AT): No consensus definition. Original *incorrect* hypothesis: point of exertion where body goes **anaerobic or into "oxygen debt,"** rapidly produces **lactic acid** causing **fatigue and leg burn.** Onset of blood lactate accumulation **(OBLA)** originally referred to as the effort level that corresponds to the point at which lactate began to rise exponentially—a blood lactate level of 4 mmol associated with this point. Most commonly **LT** refers to effort (watts) that an athlete can maintain without a rise in lactate. The U.S. Olympic Committee Sport Science and Technology Division identifies lactate threshold (LT) as **the point at which a minimum increase of 1.0 mmol/L above baseline values is followed by another increase greater than 1.0 mmol/L.** Maximum lactate steady state **(MLSS):** effort level at which there is an equilibrium between lactate production and clearance such that prolonged exercise does not result in rising serum lactate. Critical lactate measurements are power at 2 mmol and 4 mmol. Lactate is a useable fuel; training helps the body become more

efficient at shuttling lactate for utilization to other parts of the body.

$\dot{V}O_2$ max test: Ramp protocol of increasing load of 25 watts at 1 minute increments until failure to maintain set cadence. Measures oxygen consumption and heart rate (HR) at differing workloads.

Maximum aerobic power test with lactate: Ramp protocol increasing load of 30 to 40 watts at 3- to 4-minute intervals until failure to maintain set cadence. Measure $\dot{V}O_2$, HR, watts, lactate. Higher power outputs are achieved during shorter ramp protocols of 1-minute increment of 25 watts versus 4-minute stages of 35-watt increment. $\dot{V}O_2$ max values of greater than 70 mL/kg/min are found in elite and professional cyclists. Professional cyclists appear to have a decrease in the magnitude of the **$\dot{V}O_2$ slow component** (oxygen uptake slowly rises during prolonged exercise at submaximal intensity, attributed to recruitment of type II fibers due to fatigue of type I fibers).

Cycling economy (CE): Power output generated in watts at a cost of 1 L of oxygen per minute of exercise (Coyle). For a constant load test of 20 minutes at 80% of $\dot{V}O_2$ max, the economy of world class cyclists averages 85 W/L/min.

Gross mechanical efficiency (GE): Ratio of work accomplished to energy expended = $60 \times W \div 20{,}934 \times \dot{V}O_2$ (Jeukendrup). For world class cyclists GE is 25%. Both CE and GE are positively related to percentage distribution of type 1 fibers in the knee extensors. Once a high level of fitness in obtained such as in the elite athlete, CE and GE performed at submaximal intensities of 70% to 90% of maximum heart rate are more important determinants of cycling performance than $\dot{V}O_2$ max (Lucia).

Wingate anaerobic test (WANT): Developed in Israel during the 1970s, measures **peak anaerobic power** (highest mechanical power generated during any 3- to 5-second interval of the test), **anaerobic fatigue** (the percentage decline in power compared with peak power output), and **total anaerobic capacity** (total amount of work accomplished over 30 seconds); requires athlete to pedal on a mechanically braked ergometer for 30 seconds at all out pace against a predetermined fixed resistance.

Training Periods of Professional Road Cyclists

- Rest (November to December)
  - Weekly average 200 km/week, gentle, easy
  - 90% of the time spent in Zone 1, less than 70% of maximum heart rate (HRmax)
  - 10% of the time spent in Zone 2, 70% to 90% HRmax
  - 0% of the time spent in Zone 3, greater than 90% HRmax
- Precompetition (December to Mid-February)
  - Weekly average 700 km/week of building base mileage, long steady rides, no intervals prior to 1000 miles
  - 80% of the time spent in Zone 1, less than 70% of HRmax
  - 15% of the time spent in Zone 2, 70% to 90% HRmax
  - 5% of the time spent in Zone 3, greater than 90% HRmax
- Competition (Mid-February to October)
  - Weekly average 800 km/week
  - 75% of the time spent in Zone 1, less than 70% of HRmax
  - 15% of the time spent in Zone 2, 70% to 90% HRmax
  - 10% of the time spent in Zone 3, greater than 90% HRmax
- Most riders plan for two peaks during the season.
- Training prescription traditionally not as precise as in other endurance sports; most build long steady low-intensity mileage in winter months; attend one or two training camps of 8 days with higher intensity and mileage in the late winter; "race into shape" with early season races. Traditional programs are followed to prepare riders for grand tours, with generally 30 days of racing prior to Tour de France.
- With power measurement now available on the road, more cyclists are following stricter training programs than in the past.

Training Measurements

Perceived level of exertion: Monitoring simply how one feels. Borg 10- or 20-point scale.

Time: Measure duration only, not intensity. Hours per ride for recreational rider: 1 to 1.5 hours. Amateur: 2 to 3 hours per ride, 8 to 12 hours per week. Professional: 15 to 30 hours per week.

Speed: Training based on average speed. Poor indicator of training intensity because of effects of altitude, wind, terrain, road surface, drafting. If just following average speed as indicator of intensity, will lead to overtraining.

Distance: Training based on weekly or daily mileage. Only measures duration not intensity.

Heart rate: Closely correlates with exercise intensity, power output, or rate of oxygen consumption in laboratory-type setting, not as close out on the roads. Influenced by altitude, heat, hydration, illness, sleep, overreaching, and overtraining. Heart rate responds relatively slowly to changes in exercise intensity and thus cannot be used to regulate intensity of shorter efforts **(heart rate lag).** Heart rate is not a direct determinant of performance, but is a reflection of the strain imposed on the cardiovascular system for level of exertion.

Power output: Provides a direct and immediate answer to exercise intensity. Can now be measured on the road with readily available bike-mounted power meter systems. *Power at LT or 4 mmol of lactate* is one of the most important physiological determinants of cycling performance. Functional threshold power equals average power during a 40-km (50- to 70-minute) time trial. Correlates very highly, slightly greater than, power at LT (defined as 1 mmol/L increase in blood lactate over exercise baseline). Estimate athlete's threshold power by measuring power athlete can routinely produce in training during long interval repeats of 2 × 20 minutes. Mean power for five mass start stages: 220 ± 22, range 190 W to 310 W, average HR: 142 ± 5 beats per minute (bpm). Mean power for uphill 13-km TT: 392 ± 60 W (5.5 ± 0.4 W/kg), average HR: 169 ± 3 bpm. Indirect measurement, 3-week stage races: 246 ± 44 W for high mountain stages, 234 ± 43 W for semi-mountainous stages, 192 ± 45 W for flat stages (Table 67-3).

Technique

- Pedaling
  - The driving force of forward motion is the downward push on the pedals.
  - Muscles involved in the power phase drive the crank downward in an effort to rotate the crank, whereas muscles active in the recovery phase are firing primarily to reduce resistance versus the contralateral propulsive limb.
  - Most athletes clipped into pedal systems believe they are supposed to pull up on the pedals; **pulling up on the pedals is inefficient, and may lead to overuse injury.**
  - In elite cyclists, even on the upstroke, the vector of forces is downward in the opposite direction of the pedal motion (the leg in the recovery phase is not lifted as fast as the crank is rotating). The elite cyclist exhibits reduced negative force and time during the upstroke.
  - *Maximal torque during the down stroke is what differentiates elite athletes from the recreational rider.*
- Cadence (revolutions per minute or RPM)
  - To date, no optimal cadence has been determined. Experienced cyclists will spontaneously pick the cadence that appears to work best for them. Mass start races: 90 rpm, TT: 80 to 100 rpm, mountain ascents: 70 rpm.
  - Limiting factors in determining an optimal cadence: studies failed to use well-trained cyclists on their own bikes.

Table 67-3 POWER-BASED TRAINING LEVELS

Level	Name	% of threshold power	% of threshold HR	Perceived exertion
1	Active recovery	<56	<69	<2
2	Endurance	56-75	69-83	2-3
3	Tempo	76-90	84-94	3-4
4	LT	91-105	95-105*	4-5
5	$\dot{V}O_2$ max	106-120	>106†	6-7
6	Anaerobic capacity	>120	N/A	>7
7	Neuromuscular power	N/A	N/A	Maximal

Sample workouts for the athlete who time trials at an average power of 300 W and HR 160

Level	Avg. watt	Avg. HR	Workout
1	<166	<111	Ride 1 hour
2	166-225	111-134	Ride 3 hour
3	226-270	135–152	Warm-up 30 min at L1-2, then 1.5 hour at L3, 30 min cool-down
4	271-315	153-170*	Warm-up 30 min as if for race, perform 2 × 20 minutes at level 4 with 5 minute at level 1 between efforts, warm-down
5	316-360	>171†	Warm-up as above, then 6 × 5 min at L5, with 5 min at L1 between efforts, warm-down
6	>360	N/A	Warm-up as above, then 10 × 1 min at L6 with 3 min at L1 between, warm-down
7	N/A	N/A	Warm-up thoroughly, then do 6-10 all out 10 sec sprints with complete recovery between efforts

Level	*Description of Level*
1	Easy spinning, light pedal pressure, minimal sensation of leg effort, active recovery after hard training or races
2	"All day" pace or classic long slow distance training, sensation of leg effort low but may rise when climbing, frequent days in a row possible but complete recovery may take more than 24 hours for very long rides
3	"Spirited group ride" or brisk paceline, requires concentration to maintain alone, consecutive days still possible if duration is not excessive and carbohydrate intake is adequate
4	Just below to just above TT effort, mentally very taxing, usually done in multiple repeats of 10-30 min duration, consecutive days possible if completely recovered from prior training
5	Intensity of long 3- to 8-min intervals designed to increase $\dot{V}O_2$ max, completion of 30-40 min total training difficult, consecutive days not necessary
6	Short 30 sec to 3 min intervals designed to increase anaerobic capacity, HR not useful due to non–steady state nature of effort, consecutive days not necessary
7	Very short, high-intensity efforts (jumps, standing starts, sprints) stressing musculoskeletal system versus metabolic systems, power useful as guide to compare versus prior efforts

*May not be achieved during initial phases of effort.
†May not be achieved due to slowness of heart rate response and/or ceiling imposed by maximum heart rate.
Data from Coggan AR: Training and Racing with a Power Meter. Boulder, Colo: Velopress, 2006.

- Although low cadences (50 to 60 rpm) have been found to be more economical/efficient (lower $\dot{V}O_2$), most cyclists prefer to pedal at high cadences. Improved blood flow and reduced muscular stress are possible advantages at higher cadences.
- Blood flow oxygenation to the vastus lateralis is significantly reduced during the first third of the crank cycle with a compensatory transient increase in blood supply during the relaxation phase of the upstroke.
- For a given power output, at faster cadences, force on the pedals and force of muscle contractions are reduced.
- Faster cadences may be beneficial because less intramuscular pressure causes less blood vessel constriction.

INJURIES AND MEDICAL PROBLEMS

General Overview

- Cycling is a nonimpact activity, except when crashing; overuse injuries rarely prevent athlete from continuing to ride. Unlike in running, no stress fractures occur in cycling.
- Primary muscle action is quadriceps concentric activity; acute muscle tears rarely occur.
- Overtraining more common than overuse injuries; most will not seek medical attention.

Contact Area Injuries

There are three areas at which a rider makes contact with the bicycle; each lends itself to specific injuries.

Shoe-Pedal Interface

- Burning feet, numbness, or pain is common. Riders may unclip their shoes to shake foot.
- Bike fit: adjust cleat, usually move farther back; use a shoe with wider toe box; loosen straps on cleat or pedals; inspect shoe for compression points; use a wider platform pedal; small metatarsal pads.
- **Morton's neuroma** may result from impingement of interdigital nerves, classically between third and fourth metatarsals, from tight cycling shoes, pressure on pedals, and/or toe straps.
- Treatment: Massage, nonsteroidal anti-inflammatory drugs (NSAIDs), shoe inserts, cortisone injection, or surgical excision if mass present as last resort.

Pelvis-Saddle Interface

PUDENDAL NERVE ENTRAPMENT

- The most common urogenital problem encountered; genital or perineal numbness; up to 70% of cyclists.

- Bicycle factors: saddle tilt too high, saddle level too high, handlebars too low or too far forward; too much saddle padding, narrow saddle, prolonged seated riding in one position; riding on rollers worse than riding on a stationary bicycle worse than riding on the road.
- Diagnosis: Clinically, may try diagnostic lidocaine injection for confirmation, rule out urogynecological pathology.
- Treatment: Relative rest, adjust bike fit, nerve block, cortisone injection, surgical decompression for severe recalcitrant cases. Botox injection used experimentally.

ERECTILE DYSFUNCTION (ED)

- Anecdotal evidence has revealed cycling related erectile dysfunction (ED).
- Second most common urogenital bicycling disorder; reported to affect 13% to 24% of male riders.
- Vascular and neurologic mechanisms implicated.
- Compression of the perineal region during cycling may cause decreased penile perfusion.
- Prolonged hypoxemia has been associated with penile fibrosis.
- Blunt trauma to the corpora cavernosa has been considered to be a risk factor for the subsequent development of ED.
- Penile blood flow decreases significantly while cycling in a seated position. The greatest decrease is seen with narrow saddles.
- Elimination of the nose of the saddle results in a reduction in perineal pressure.
- The most important factor in safeguarding penile perfusion is a saddle wide enough to support the ischial tuberosities without compression of the perineal region—*not* saddle padding.
- Scientific studies limited; most done in laboratory settings on stationary bicycles without attention to technique and factors involved in road cycling.
- Recumbent cycling causes no compression of perineum.
- Perfusion is increased with standing; frequent position changes are beneficial.
- Time trial bicycles with aero bars cause the most compression of the perineum; may be reduced with adjustment of saddle tilt.
- Millions of cyclists are asymptomatic. Some appear to be more at risk than others. Bike fit and technique play a role in prevention.

SADDLE SORES

- Moisture, friction, and pressure lead to skin ailments in the genital region.
- **Chafing** can occur in its most mild form. Relieved with 1 to 2 days off the bike.
- **Ulceration** or more severe friction injury may require local wound care.
- **Furuncles and folliculitis** (saddle sores). Very painful, may limit riding for long periods.
- **Perineal nodular induration (third testicle)** is the most severe form. Also called ischiatic hygroma, accessory testicles, and biker's nodule.
  - Friction and pressure from the saddle induces collagen degeneration, myxoid changes, and pseudocyst formation.
  - Histologically: nodule with a dense fibrous capsule surrounding a central pseudocyst.
  - Clinically: elastic 2- to 3-cm perineal nodules fixed to the underlying soft tissue.
  - Time off from riding and cortisone injection are nonsurgical options. Surgical excision is definitive.
- Bike fit remedies: change or cut a hole in the saddle; check saddle height and tilt, raise handlebar height.
- Medical treatment: prevention, keep dry, clean chamois; emollients; avoid shaving the area; warm soaks; topical cortisone, antifungal, antibacterials; oral antibiotics; surgical incision and drainage; excision.

FEMALE ISSUES

Description: The anatomical course of the pudendal artery and nerve is the same as in males; sexual and genitourinary complaints are similar.

Saddle discomfort: Women may find the saddle uncomfortable and experience sexual and urinary dysfunction. Study on 282 female members, one third had perineal trauma, 19% associated with hematuria or dysuria and 34% with numbness. Bike fit: try women's specific frame with shorter top tube for less of a reach, women's specific saddles, shorts with padded chamois, pointing saddle down slightly.

Vulvar swelling: Mostly unilateral and caused by prolonged riding; no long-term sequelae.

Urethritis: May be caused from pressure, friction, or infection. Rule out genitourinary infection with pelvic exam for vaginitis (yeast due to moisture), and urinalysis and culture. Hematuria and dysuria may be caused by local trauma alone. Treatment: bike fit and routine management.

PRIAPISM

- Reported in the literature, a rare event from cycling; caused by unregulated cavernous arterial inflow.
- Either macrotrauma or microtrauma; presentation immediate or delayed.
- Most common etiology: vascular trauma, causing an arteriovenous fistula or shunt feeding the cavernous sinusoids directly.
- Diagnosis: ultrasound or arteriography.
- Treatment: observation, selective arterial embolization, or surgery.

PROSTATE DISORDERS

- No evidence linking cycling to prostatitis.
- The influence of bicycle riding on PSA levels remains inconclusive.
- Hormonal effects from strenuous exercise and local mechanical stress may increase PSA.
- Avoiding cycling prior to PSA sampling may be advised but is without scientific basis.

Hands-Handlebar Interface

- **"Cyclist's palsy":** Prolonged compression of ulnar nerve in Guyon's canal. Median nerve may also be compressed. Complaint of numbness, pain, and weakness; resolves with rest. Rarely is damage permanent.
- Distal motor latencies of the deep branch of the ulnar nerve to the first dorsal interosseous were significantly prolonged in a study of 28 riders in 420-mile tour.
- Study of 25 cyclists participating in a 600-km bicycle ride found that 23 of 25 cyclists experienced either motor or sensory symptoms or both, the majority ulnar neuropathy.
- A case series of three patients: severe ulnar neuropathy with an isolated lesion of the deep terminal motor branch of the ulnar nerve with the distal sensory branch intact. Because no sensory fibers are affected, the athlete is unaware of compression until severe motor lesion develops.
- Cycling factors: downhill cycling, prolonged saddle time, rough terrain, handlebars too low or forward, poor padding in gloves or bars.
- Bike fit: change hand position often, reduce training volume, change terrain, increase padding, shorten reach or raise bars, use aero bars. Gel padding may worsen problem.
- Medical treatment: rest, massage, cortisone injection, night splint, hand therapy; surgery in severe cases.

Acute Traumatic Crash Injuries

Skin Abrasion ("Road Rash")

- Most common traumatic injury.
- Treatment: Rapid cleansing with thorough scrubbing to prevent infection and staining ("tattooing"). Soap and water is

best. Peroxide can inhibit healing. Local anesthetic may be used.
- The wound should *not* be left open to the air to heal because large scabs may form.
- Traditional method: Cover wound with a nonadherent dressing with antibiotic ointment or silver sulfadiazine, pad with gauze, wrap with stretch gauze, and cover with tube stretch gauze. Daily dressing changes with cleansing of exudates, until pink healthy tissue.
- Alternatively: Semipermeable films, hydrocolloid dressings, or bioclusive bandages may be used and left in place for extended period of time until healing.

Handlebar Trauma

- More common in children with straight handlebars.
- Caused by direct blow of bars turning sideways into abdomen.
- Injuries: **splenic, liver, pancreatic, renal, bowel, and urethral.**
- Presentation often delayed.
- Most cases of organ contusion are handled with observation and serial computed tomography (CT); occasionally surgery.
- **Handlebar hernia** is a rare traumatic hernia involving disruption of the abdominal wall muscles with bowel loop herniation and potential volvulus. CT and treatment with primary repair.
- **Lacerations** result from sharp metallic end of handlebar cutting through soft rubber handle or bars with no end plug.

Concussion

- From direct blow, fall over front of handlebars; most get up and ride concussed.
- May be overlooked with associated injuries.
- If helmet has any surface scratch, look for interior damage.
- Helmet discarded after one blow.

Upper Extremity Injuries

CLAVICLE FRACTURES

- The most common cycling fracture.
- Caused by a direct blow to the shoulder.
- Of clavicle fractures, 72% to 80% occur at middle third of clavicle, 25% to 30% in distal clavicle, and about 2% at the proximal clavicle.
- May be associated with **rib and scapula fractures, and concussion.** Rarely **pulmonary contusion, pneumothorax, hemothorax, and neurovascular injury.**
- Most cyclists can ride on a trainer within 3 days to 1 week and outdoors within 2 weeks, racing in 3 to 6 weeks.
- Historically, clavicle fractures have been considered to be best treated nonoperatively with a simple sling or figure-of-eight brace.
- A prospective randomized trial found that simple sling caused less discomfort and fewer complications than figure-of-eight brace.
- Sling is more commonly used and may be worn for 1 to 2 weeks until pain free, encouraging range of motion at elbow and shoulder.
- Current management of medial clavicle fractures remains nonoperative because significant displacement is rare due to ligament stability. If displacement is present, image with CT.
- Treatment of distal clavicle fractures remains controversial because of multiple subtypes of associated ligamentous injury; most cases treated nonoperatively.
- Treatment of middle third fractures remains primarily nonoperative except when severe displacement communication or shortening is present.
  - Initial shortening at the fracture of greater than or equal to 20 mm had a significant association with nonunion and unsatisfactory result.
  - Displacement of more than one bone width on x-ray is a risk factor for nonunion.
- Late repair of painful nonunion displaced midshaft fractures has results similar to immediate fixation.
- Surgery: Intramedullary fixation, with risk of wire migration, or plate and screws with or without bone grafting, with risk of prominent painful hardware.
- Asymptomatic nonunion is not associated with any significant disability.

OTHER UPPER EXTREMITY INJURIES

Acromioclavicular separation: Direct blow, grades 1, 2, or 3. Brief use of sling for comfort. Stationary trainer workouts can begin within days. Road riding in 2 to 3 weeks. Treatment is same as for noncyclists; all but severe grade 3 treated nonoperatively.

Shoulder dislocation: From direct blow; usually involves transient dislocation and relocation with **tear of ligaments, labrum, Bankart or Hill-Sachs lesions.** Cyclists can generally return quickly; instability in everyday activity is the factor for operative repair later.

Radial head fractures (direct blow): Presents with hemarthrosis, decreased extension, and fat pad sign on x-ray without fracture (type I). Short-term sling with rapid commencement of range of motion exercises to achieve full extension. Aspiration of hemarthrosis with infiltration of anesthesia to check for mechanical block and aid in range of motion and pain relief. With nondisplaced fractures can return to riding within 1 to 2 weeks as tolerated.

Distal radius, scaphoid, or hook of hamate fracture: Fall on outstretched hand may cause one of these. In nonsurgical cases, rapid return to training within 1 to 2 weeks is possible with casting.

Lower Extremity Injuries

Hip pointer and/or greater trochanteric bursitis: Common from direct blow to lateral aspect of pelvis/femur; large **hematoma** may result; have clinical suspicion for pelvic fracture—most are stable and treated with crutches and time; CT when in doubt; most able to ride trainer before walking normal.

Knee contusion: Have clinical suspicion for patella fracture, take sunrise or merchant x-ray. **Prepatellar bursitis** common; treated with aspiration. Wrapping the patella should not be done when cycling because restriction of patella movement may lead to patella-femoral pain.

Fractures of femur and hip are less common, seen in professional cyclists and older individuals from underlying osteoporosis. More common with motor vehicle involvement.

Overuse

Knee Injuries

- Knee is most common site of overuse injury in the cyclist.
- Simple bicycle adjustments may be made based on location of knee pain (Table 67-4).
- **Anterior knee pain**
  - **Patella** tendon strain **("tendonitis"):**
    - Anterior knee pain in the tendon.
    - May become chronic or recurrent.
    - Causes: Pushing big gears, cranks too long, hills, head wind, rapidly increasing mileage or intensity; saddle too low or forward.
    - Medical treatment: Massage with assisted stretching, strapping.
    - Cycling treatment: Decrease mileage and intensity, avoid hills, spin 80 to 90 rpm. Check saddle height and fore-aft position
  - **Patella femoral pain syndrome:** Presents as pain in the patello-femoral groove or patella facets. Similar to "ten-

Table 67-4 BICYCLE ADJUSTMENT BASED ON THE LOCATION OF KNEE PAIN

Location	Causes	Bicycle adjustment
Anterior	Seat too low	Raise seat
	Seat too far forward	Move seat back
	Climbing too much	Reduce climbing
	Big gears, low rpm	Spin more
	Cranks too long	Shorten cranks
Medial	Cleats: toes point out	Modify cleat position: toe in
	Floating pedals	Consider floating pedals Limit float to 5 degrees
	Exiting clipless pedals	Lower tension
	Feet too far apart	Modify cleat position: move closer Shorten bottom bracket axle Use cranks with less offset
Lateral	Cleats: toes point in	Modify cleat: toe out Consider floating pedals
	Floating pedals	Limit float to 5 degrees
	Feet too close	Modify cleat position: apart Longer bottom bracket axle Use cranks with more offset Shim pedal on crank 2 mm
Posterior	Saddle too high	Lower saddle
	Saddle too far back	Move saddle forward
	Floating pedals	Limit float to 5 degrees

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donitis" in etiology and treatment. Limit use of cortisone injection.
  - **Prepatellar bursitis:** Swelling overlying patella. Caused by direct trauma or repetitive overuse. Treatment as above, occasional aspiration.
  - **Pes anserine bursitis:** Swelling and pain at pes anserine insertion. Cortisone and treatment as above.
- Lateral knee pain
  - **Iliotibial band (ITB) syndrome:** Lateral knee pain with or without snapping; one of the most common complaints.
  - Causes: High mileage and intensity, big gears, hills, prolonged steady state riding, windy conditions, toes pointing inward, or narrow bottom bracket.
  - Cycling treatment: Adjust cleats; check bike fit.
  - Medical treatment: Massage and assisted stretching effective first-line treatment. Foam roller. Osteopathic manipulation of sacrum and pelvis. Leg length evaluation and correction. Orthotics. Cortisone last resort.
- Medial knee pain
  - **Medial collateral ligament (MCL) bursitis:** Often overlooked; responds well to relative rest, bike fit adjustment, massage therapy, and cortisone injection.
  - **Medial meniscus tear:** Not known to be primarily caused by pedal systems; may be worsened by unclipping.
  - Asymptomatic **medial plica** may cause antero-medial knee pain and mechanical symptoms.
    - Repeated irritation from cycling may lead to fibrosis with decreased elasticity and condylar cartilage damage.
    - Diagnosis of exclusion; presence of a plica alone is not diagnostic.
    - Symptoms similar to meniscal tear.
    - MRI if suspicious.
    - If treatment with rest and/or cortisone fails, arthroscopic surgery.
- Posterior knee pain
  - Not as common, may be caused by too high or far back saddle, pedals with excessive float, improper technique of pulling up on the pedals, or by tight hamstrings.
  - Rule out lumbar radiculopathy.
  - Treatment: Proper bike fit, relative rest, and massage.

Hip Injuries

- **Flow limitations in iliac arteries**
  - Prevalence as high as 20% in top cyclists; underreported and unrecognized problem; have a high clinical suspicion.
  - Sensation of dead leg, powerlessness, pain in thigh. Disappear upon rest. Worse with increased hip flexion while riding; time trialing or climbing (steady state riding).
  - Physical exam is usually normal, may hear bruit in inguinal region at rest or post exercise.
  - Flow limitation may be caused by kinking (functional iliac artery obstruction) or endofibrosis (external iliac artery endofibrosis or EIAE).
  - Mechanical, anatomical, and hereditary factors.
    - Anatomically, the external iliac artery (EIA) may be anchored to the psoas muscle and lengthened with multiple collateral vessels.
    - Mechanically, the middle part of the EIA kinks during flexion-extension of the hip with stress lesions in the vessel wall from shearing forces created by constant cycling.
    - Hereditary and metabolic factors implicated because 75% of surgical cases presented with increases in homocystinemia and homocystinuria after load test with methionine.
  - Histopathology: Nonatheromatous vascular lesion with intimal subendothelial fibrosis leading to wall thickening and reduction of lumen caliber. May be caused by mechanical loading in susceptible individuals.
  - Functional kinking may lead to intravascular damage and poor perfusion.
  - Chevalier treated 334 lesions of endofibrosis surgically from 1991 to 2003. The first method of surgery, deemed conservative, involved endofibrosectomy with shortening of excessive length and closing with venous or arterial closing angioplasty. The second method involves saphenous bypass surgery. Mean return to competition is 3 months.
  - Schep studied 80 suspected cases (92 symptomatic legs) and demonstrated a flow limitation in 58 legs (63%). In 40 of the legs (69%), the primary cause was kinking of the EIA due to a Psoas muscle side branch or fibrous fixation of the iliac bifurcation. 23 legs (40%) underwent surgical release, with 87% able to return to high level of competition.
  - Testing
    - Provocative cycling test Ankle-Brachial Index (ABI) is the most important clinical test and has a specificity and sensitivity in detecting moderate lesions of 90% and 87%, respectively. 20 Watt per minute ramp protocol, ABI measured immediately postexercise supine with 90 degree hip and knee flexion. Positive test: ABI less than .54, ankle difference greater than 23, ankle pressure less than 107; suspicious if ABI .54 - .70.
    - Echo Doppler at rest and immediately post provocative exercise cycling test.
    - Magnetic resonance angiography with hip flexed.
    - Arteriography is the gold standard.
    - CT angiography and MR angiography do not appear as sensitive.
  - Treatment: Surgical, one or more of the following: 1) Release of artery (mobilization) for treatment of primary kinking when there is no structural narrowing or lengthening,

2) Treatment of excessive length with shortening, 3) Treatment of endofibrosis with endofibrosectomy with venous or arterial closing angioplasty, polyester patch angioplasty, or saphenous bypass surgery, 4) Inguinal ligament release, 5. NOT recommended: angioplasty, stent.
  - Medical treatment: Vitamin B1, B6, and folates when minor hyperhomocysteinemia is detected. Less hip flexion on bike. Retire (unknown natural history untreated and treated).
- **Greater trochanteric bursitis, proximal ITB tendinopathy**
  - **Proximal lateral thigh pain with or without snapping.**
  - Treatment: Relative rest, easy riding, stretching correctly, massage, osteopathic manipulation, cortisone.
- **Iliopsoas bursitis** rare when compared to running; treatment as above for greater trochanteric bursitis.

Ankle Strains

- **Including achilles, tibialis anterior, posterior tibialis.**
- Rare; usually the result of cyclists running, improper bike fit, or poor technique of pedaling (excessive pushing down heel in downstroke or pulling with foot in upstroke), leg length inequality.
- Massage and brief activity/technique modification with bike fit is curative.

Spine Injuries

- **Cervical strain with or without radiculopathy.**
  - More common in older athletes, with underlying arthritis or disc disease.
  - Worse with long rides, rough terrain, and handlebars too far forward or too low with hyperextension of the neck.
  - Cycling treatment: assume more upright cycling posture. Medical treatment: same as for noncyclists.
- **Thoracic strain** associated with **scapula dysfunction and muscle spasm at the levator scapula,** worse with aero bars.
  - Treatment: trigger point injections, manipulation, massage, bike fit adjustment, and strengthening exercises.
  - **Coronary artery disease** may present as scapula or upper back pain.
- **Lumbar spine and low back pain.**
  - May be caused by strain, spasm, or disc disease.
  - Image sooner for anyone with risk factors of other medical conditions.
  - Cycling treatment: bike fit, avoid hills and big gears, and shorten rides.
  - Medical treatment: same as for noncyclists.

Ischial Tuberosity Pain

- Discomfort in the "sits bones" where the rider makes contact with the saddle.
- Area can be tight and sore with bursitis.
- Risks: time trialing, especially early in the season and colder rides.
- Treatment: relative rest, massage, extra padding with new chamois, new saddle. Cortisone for severe cases.

Medical Conditions

Osteoporosis

- Small study found master cyclists with a long history of training exclusively in cycling had low bone mass density (BMD) compared to their age-matched peers.
- Several factors may play a role:
  - Bicycling is a nonimpact sport; when cyclists are not training they avoid weight bearing.
  - Calcium lost in sweat may play a role. Most cyclists live on a low-fat, high-carbohydrate diet with little extra calcium in the foods they eat.
  - Overtraining can lead to sex hormone imbalance, increasing bone turnover and decreasing resorption.
  - Performance enhancing substances can accelerate bone loss.
- Unlike in running, where stress fractures are common, cyclists with osteoporosis are at risk for major fractures from crashes.

Asthma

- 40% to 80% of Tour de France riders reported to "have diagnosis" or use inhalers during the race.
- Decreased forced expiratory volume in 1 second (FEV_1) greater than or equal to 10% following bronchoprovocation is the accepted standard for diagnosis of elite exercise-induced asthma.
- Licensed racers with asthma require an abbreviated therapeutic use exemption (ATUE) form from the UCI for use of permitted inhaled beta-2 agonists and glucocorticosteroids by nonsystemic routes.
- Salbutamol (albuterol) level greater than 1000 ng/mL is prohibited even with a TUE.
- For updated list of prohibited substances go to www.usantidoping.org/dro, call USADA's Drug Reference Line, 800-233-0393 or 719-785-2020, or email drugreference@usantidoping.org.

Overtraining Syndrome

- Accumulation of training and/or nontraining stressors resulting in long-term performance decrement, with or without related physiologic and psychological signs and symptoms; restoration of performance capacity may take weeks to months; exclude organic disease.
- Disparity of load and load tolerance.
- Prone to overtraining because long hours may be spent cycling without a musculoskeletal injury.
- Staleness and lack of improvement more common than frank overtraining syndrome.
- Pathophysiology: Muscle damage in fibers and mitochondria, impaired recruitment of muscle fibers, impaired hypothalamic-pituitary axis, and reduced sympathetic nervous system activity.
- Risks: High training load, nontraining stressors, sticking to a program when sick or injured, making up lost time (to illness, injury, or missed training), increase in training because of poor performance, monotonous training, and **not enough rest.**
- Early signs: Performance decrement, progressive weight loss, poor sleep, increased resting heart rate, increased fluid intake in the evening.
- Symptoms: Loss of vigor, fatigue, depression, agitation, insomnia, irritability, restlessness, loss of motivation, poor concentration, heavy legs.
- Prevention: Individualize training, build base mileage before speed work, periodization, avoid monotony, rest, carbohydrate rich diet, avoid overcompensating.
- Lab tests: **No single identifiable parameter;** exclude organic disease; complete blood count to rule out anemia, infection; complete metabolic profile to rule out renal or hepatic disease, electrolyte disturbance as clue to eating disorder; thyroid stimulating hormones, Epstein-Barr virus titer. Consider adrenal axis workup. Glutamine/glutamate decreased. Salivary immunoglobulin A decreased. May consider echocardiogram, EKG.
- Two best markers of overtraining: (1) decrease performance on standard exercise tests with HR, lactate, and $\dot{V}O_2$ max; (2) self-analysis of well-being by the athlete such as with Profile of Mood State (POMS).
- Best log book markers: Mood, muscle soreness, stress, quality of sleep, and performance decrement.

- Overtraining should be discussed not only under the clinical aspect, but more under the aspect of training content.
- Late diagnosis may result in loss of months of racing.
- Cycling treatment: Complete time off the bike weeks to months; return slowly with no hills or hard efforts for one month.
- Medical treatment: May consider antidepressant selective serotonin reuptake inhibitors (SSRI).

Gastrointestinal Distress

- Not as common as with running.
- Blood flow may be reduced to the GI system by 40% to 50% during exercise with shift toward working muscles, skin, and vital organs.
- In triathlon, majority of issues on the run are related to lower GI tract with diarrhea, and on the bike related to upper GI tract with reflux and vomiting. Swallowing water and air during swimming contributes. Increased pressure on abdomen from cycling position may contribute. Dehydration and nutrition contributes.

Nutrition

- Cyclist with average efficiency and aerodynamics requires about 21 kcal per minute to ride at 25 mph.
- Carbohydrate stores in the body (2000 to 3000 kcal) can fuel about 90 minutes of riding at 25 mph.
- During the Tour de France energy expenditure may be as high as 9000 kcal per day.
- The training diet for a cyclist:
  - 60% to 70% carbohydrate = 3.1 g per pound of body weight daily
  - 15% to 20% protein = 0.5 to 0.9 g per pound of body weight daily = 7 to 10 g/kg daily
  - 20% to 30% fat = *not* less than 20 g per day
- Pre-exercise:
  - Intake of 150 to 300 g of carbohydrate 3 hours prior to exercise will increase muscle glycogen and performance.
- During exercise:
  - 60 to 70 g carbohydrate per hour of training
  - Use of carbohydrate levels off at 1 g per minute
  - Glucose, maltodextrins, sucrose, maltose and soluble starches oxidized at high rates versus fructose and galactose (Jeukendrup and Jentjens, 2000).
  - 300 to 400 calories per hour of training
  - 400 to 600 mL of fluid per hour
  - Drinking ad libitum (according to the dictates of thirst) has been shown to have no detrimental effect on performance
  - Sodium intake of 0.15 to 0.45 g per L of fluid
- Postexercise:
  - Maximum rate of muscle glycogen resynthesis is reached at intake of 1.2 to 1.4 g per minute (75 to 90 g of carbohydrate per hour).
  - Carbohydrate intake of 1.2 g per kg body weight per hour for 4 hours post exercise, preferably within 90 minutes.
  - Carbohydrate intake of more than 1.4 g per minute provides no additional benefits in glycogen storage and can increase gastrointestinal distress.
  - Total carbohydrate intake of 8 to 10 g per kg of body weight within 24 hours.

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Mountain Biking

Christopher C. Madden and Steven J. Collina

GENERAL OVERVIEW

Definitions

- Mountain biking broadly refers to riding bikes with specific design characteristics in various off-road settings.
- Mountain bikes generally differ from road bikes in several ways: smaller frame, stronger rims, larger range of gears, wider flat or upright handlebar, stronger brakes, suspension, wider, knobby tires.
- Evolved into many riding and bike types; some overlap between bikes and riding styles.
- Four main categories include cross-country (XC), downhill (DH) and freeride (FR), all-mountain (AM), and trials and urban riding (TR) (Fig. 68-1).

Cross-Country

- Most common form; most aerobic form.
- Point-to-point or loop riding over varied terrain, including climbs and descents, but generally less aggressive and extreme than FR or DH.
- Races are mass start over a circuit course, lengths vary, but lengths up to 30 miles are common, and ultra-endurance events (many 100 miles plus) are gaining popularity.
- Lightest bikes (21 to 28 pounds); lightest parts; more aggressive frame angles; short travel suspension common (3 to 5 inches), front or both front and rear; clipless (higher level or competitive) or toe clip pedals (recreational).

Downhill and Freeride

- DH gravity time trial competition consisting of riders racing at one time, against the clock, down a trail, jeep road, fire road, or combination; FR similar to DH riding off the clock and over extreme terrain.
- DH courses and FR terrain technically challenging and feature high speeds, steeps, jumps, drop-offs (ranging from 10 to 40 feet) and other natural and man-made technical features.
- FR terrain may include rocks and cliffs, man-made structures such as interconnecting wooden bridges, drops, logs, bridges, and stunts.
- DH trials typically last 5 to 10 minutes; speeds exceed 50 mph; ultimate test of nerve and bike control.
- DH bikes shuttled to top, not ridden; FR can be ridden, sometimes pushed between destinations.
- FR also referred to as "North Shore," black diamond, and big hit riding.
- Requires significant total body, aerobic, and anaerobic strength.
- Body armor and full face helmet used.
- Heaviest bikes (32 to 50 pounds); considerable overlap between FR and DH; frames with slack angles and large tubing;

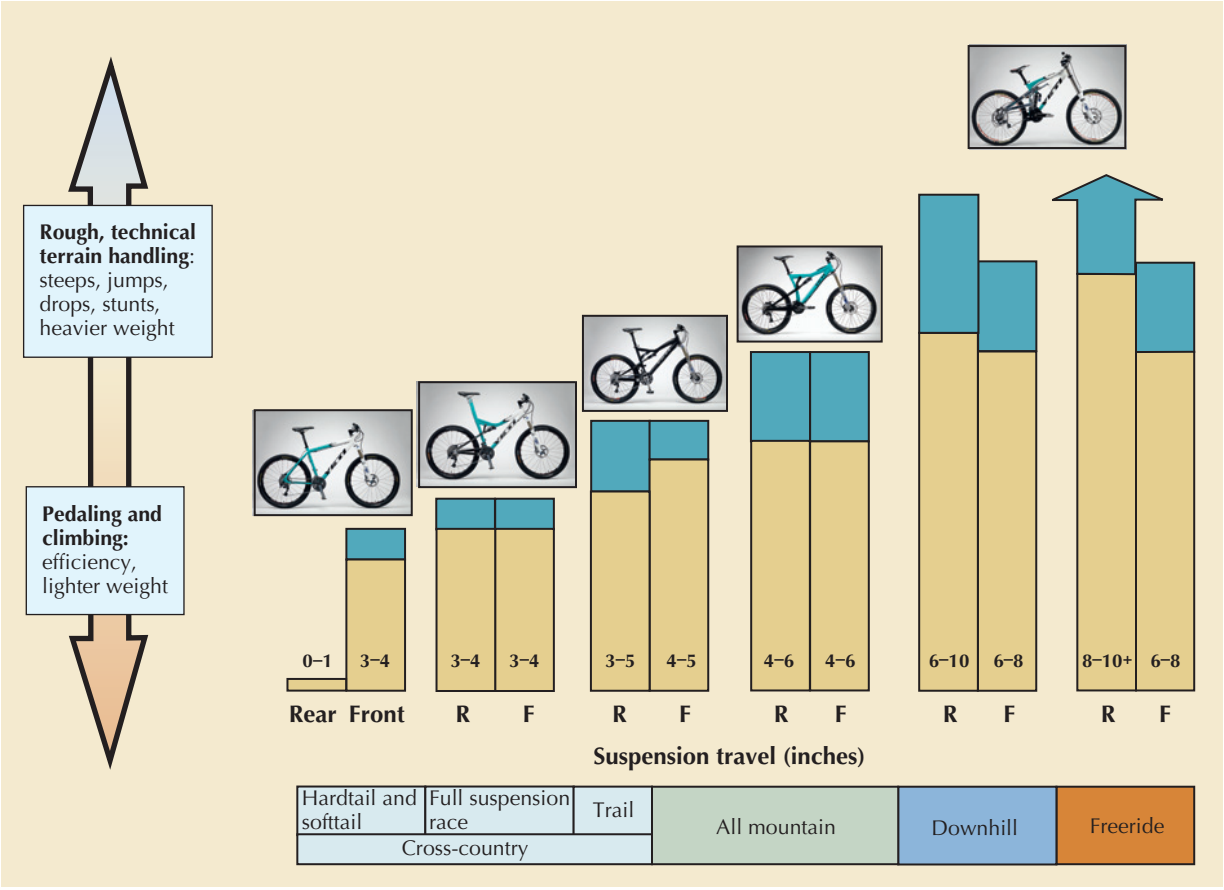

Figure 68-1 Mountain Bike Styles and Characteristics. *(Photographs from Yeti Cycles with permission. Data from Lopes B, McCormack L: Mastering Mountain Bike Skills: Techniques to Excel in all Riding Styles. Human Kinetics, 2005.)*

DH bottom bracket may be lower than FR; powerful, large disc brakes; longest travel suspension (6 to 10 inches), always front and rear; platform (FR>DH) or clipless (DH>FR) pedals (sometimes with clipless on large platform).
- "Slopestyle" riding is a variation of FR that combines big air freeride stunts with BMX style tricks; events held at mountain bike parks.
- Super D is hybrid XC and DH event; more DH than uphill sections; lack jumps and technicality of typical DH courses; tests endurance and bike-handling skills.

All-Mountain

- Increasingly popular style; broadest range of riding.
- Riding over aggressive XC (AM) to challenging natural terrain, may approach light FR at extreme end of spectrum.
- More aerobic than anaerobic.
- Bikes often referred to as "trail bikes," are heavier than XC (28 to 35 pounds, broad range depending on need); frames with slacker angles than XC and steeper than DH and FR, but heaviest of class can still climb well; bottom bracket is higher than XC and DH; longer travel suspension (AM: 5 to 6.5 inches), always front and rear; heavier and stronger parts than XC; clipless pedals.

Trials and Urban Riding

- Riding that involves hopping and jumping over obstacles.
- Can perform almost anywhere ranging from competition (observed trials) to "urban assault" (trials type riding using urban or city obstacles).
- Riding involves highly developed balance; emphasis is on style, originality, and technique.
- Broad range of trials bikes ranging from XC mountain bike conversions to smaller, specialized trials bikes; wheel size ranges from 20 to 26 inches; frames are smaller and lower; seats are low or absent; usually have front or no suspension; platform pedals.

Demographics

- All ages, mean varies depending on type of biking: 22 to 36 years average; most competitors are between 19 and 44 years; more males than females; female participation increasing; especially popular among young males.
- Mountain bikes are largest category of bikes sold in U.S. bike shops.
- Shift from XC racing to DH, FR, and other styles.
- Noncompetitive mountain bikers increasing; little known about injury epidemiology.
- Sport attracts risk-tolerant personalities.

Research

- Injuries are reported inconsistently in the literature, ranging from specific injury to general type of injury (e.g., laceration, fracture) to body area (e.g., joint, upper or lower extremity, head).
- Most data is from competitive XC with some DH and FR, some recreational, other areas minimal; AM and FR now entering picture.
- Available studies primarily descriptive and focus mainly on injuries themselves (sometimes generally defined by body or joint area); fewer details available regarding mechanisms of injury. Most injury studies with preponderance of male participants (75% to 80%). Many studies depend on victim recall.

Competition

- XC still popular, but seeing shift to DH and other events.
- Categories established based on age, gender, and rider skill.
  - Rider skill categories (applicable to 15 years old and up): Pro, Semi-Pro (men only), Expert, Sport, and Beginner.
  - Age categories: Youth (<10 years), Junior (10-18 years), Under 23 (19-22 years), Senior (23-29 years), Master (30+ years).

Bicycling Modernization and Specialization

Bike

- Modern mountain bikes have evolved into sophisticated machines, many with complex suspension systems.
- Designs are tailored to specific events, but there is considerable overlap.
- Complex front suspension and dual suspension systems:
  - Increases rider comfort, control, and performance.
  - Reduces physical trauma from excessive vibration, but makes more risky maneuvers possible.
  - May be significant differences in power output between front suspension and dual suspension during uphill climbing, but differences may not translate into increased oxygen cost or time to complete an event; newer valved or brain technology that reduces pedal "bob" probably decreases metabolic cost further.
  - Riders learn to climb using dual suspension: power preservation with spring rebound contributing to forward momentum (four-bar linkage studied, not directly applicable to other designs).
- Angles of frame tubes relative to each other vary and determine handling characteristics.
  - Common angles: head tube, seat tube.
  - Steeper angles generally create more responsiveness with turning front wheel; slacker angles create more stability in high-speed cornering, but handle more clumsily over "tight" terrain.
  - Some manufacturers offer female-specific geometry.
- Multiple shifting options: grip, trigger, integrated (same brake and shifter handle).
- Broad gearing options: three rings up front (size varies), nine gear clusters/cogs in rear (most common 11/12 to 32/34 teeth), many variations.
- Powerful brakes: Hydraulic disc, mechanical disc, and v-brakes; cantilevers rarely used; preserves forearm and hand strength and endurance; "one-finger" braking allows for better bike control.
- Multiple clipless (mini ski binding) pedal systems: vary in rotational release angle and tension, rotational and lateral float, adjustability, ease of engagement and release, and ability to shed mud.
- Toe clips mostly used by recreational cyclists; rare on aggressive or competition bikes.
- Handlebars are wider and more upright; bar ends disappearing.
- Tires available in broad range of widths (1.95 to 2.3 inches most common, larger in DH), tread patterns (semislick to knobby), and compounds (soft, sticky rubber to firm rubber, or mixed); matched to riding conditions; can be factor in crashes.
- Tubeless tires gaining popularity over tubes (tubeless specific rims and tires vs. tubeless conversions using non-tubeless rim and tire with rubber liner and sealant).

Protective Equipment

Protective torso armor and extremity padding: For FR and DH.

Helmet designs: Full face and standard; make sure Snell-, CPSC-, or ANSI-approved and tested. Recommend replacement after significant crash.

Shorts: Multiple types of synthetic moisture-wicking chamois; some have built-in hip padding.

Gloves: Varying thickness, shell protection (dorsal fingers and hand), and padding.

Eyewear: Ultraviolet protection, shatterproof, changeable light-reducing and colored lenses for varying conditions; goggles in DH and extreme cold.

Mountain Bike Fit

- Directly applicable to overuse, indirectly to acute traumatic injuries.
- Start with a professional fit (see Chapter 67, Road Biking).
- **Mountain bike fit not as straightforward as road fit;** use initial road fit to help achieve mountain bike fit window (ideal, individualized fit that may deviate slightly from virtual perfect fit).
- Static fit (using a standardized bike fit system) a good place to start to identify neutral position as starting point for individualized adjustments.
- Dynamic fit better than static, better individualization and customization, digital video sometimes used.
- Experienced physician office or high-end bike shop will offer help with fit customization.
- Riding position and repetitive forces remain relatively constant in road cycling, but mountain bikers frequently change rider position while riding over varied terrain; leads to broader, less repetitive forces.
- Fit adjustments for mountain bike follow same order as road: frame size, saddle height, saddle fore-aft (to establish neutral knee), saddle tilt, reach and stem/handlebar height. Keep in mind that changing one usually affects the others. A good fit guide is available at www.coloradocyclist.com/bikefit.
- Frame size for a mountain bike is likely smaller than that for a comparable road bike, depending on riding style.
  - Top tube should have 3 to 6 inches clearance with standover (applicable with non- or minimally-sloping tube) (shoes on).
  - Mountain bike inseam method: (____cm inseam × 0.67) − 10-12 cm = ___-___cm frame (shoes off).
  - Mountain bike frame designs broadly vary, so check specific seat tube and top tube lengths; top tube length more important than seat tube length (more adjustable) with mountain bike fit; frame angles and suspension type and design must be factored in because they will affect resultant ride.
  - Take measurements and frame angles from prior mountain bike that fit well; sometimes difficult to transfer with variations in frame and suspension designs and geometry.

Saddle Height

- Many mountain bikers lower seat at least 1 cm from road riding position to increase control, assist with steep climbing, and to allow easier dismounts; can measure crank arms and lower seat by amount equal to difference in crank arm length of mountain and road bike (e.g., 175 − 170 mm = lower mountain saddle by 5 mm).
- Quick-adjust drop seatposts available (riders often drop post before DH after long climb): lower saddle more maneuverable and controllable (DH and FR); higher achieves more aerodynamic position (XC racing).

Saddle Fore-Aft

- Plumb line or level dropped from inferior pole of patella with pedal at 3 o'clock (forward, parallel to ground) should bisect pedal axle (some drop behind crank arm—should bisect center); make sure suspension sag set appropriately.
- Long climbs: Some mountain riders prefer to set seat back from this position to increase rear wheel traction and leverage (especially if pushing large gears).
- Sprinters: Some prefer forward cleat position; neutral safest.
- Seatpost: Standard (clamp on top of post) or setback (clamp rearward from top of post) available.
- Do not use fore-aft setting to compensate for improper reach.

Reach and Stem/Handlebar Height

- Most individualized part of mountain bike fit; style of riding will affect significantly.
- Initial reach so torso is 45 degrees to ground; reach typically more upright than road; influenced by riding style, flexibility, and comfort.
- Longer reach and lower drop for competitive XC riders; shorter and higher for more upright posture with novices and certain riding styles (e.g., DH, FR, DJ).
- Reach affected by stem length and angle, flat or upright handlebars, top tube length, saddle fore-aft position, bar ends (dynamic).
- Handlebar and stem height adjustable: initial steerer tube length (determined by cutting initially long tube—do not cut too short), spacers between headset and stem, stem angle, and handlebar type (flat or upright, width, sweep).
- Handlebar width: Grips initially set at shoulder width; narrower: increases steering responsiveness; wider: increases stability and leverage.

Suspension

- Various suspension settings combined with a broad range of frame designs can make initial setup challenging; settings may include sag, preload, rebound and/or compression damping, and pedal platform adjustments.
- Sag: Amount of travel suspension compresses with static body weight over it; preload: amount of initial spring compression, which controls when it begins to move.
- Less preload = more sag; more preload = less sag.
- Sag is set by placing bike on trainer, starting with no preload or easiest valve setting and recommended air pressure, having rider gently climb on bike to weight suspension, placing zip tie around shock stanchion and measuring amount it slides (percentage of total suspension travel in mm) with rider weight; add or remove air pressure or change preload setting to achieve ideal.
- General sag recommendations: XC racers: 12% to 20%, DH: 30% to 40%, recreational: 20% (see manufacturer manual for variations).

Shoe-Cleat-Pedal Interface

- FR, TR, and DJ use platform pedals with studs or "spikes" that dig into flat shoe, can cause significant pretibial and other soft-tissue injury if slip off pedal; DH (especially competition) often uses clipless, but some use platform or hybrid platform/clipless.
- XC uses clipless (mini ski binding) with adjustable release spring; clipless mechanism and platform size varies.
- Cleat placement is adjustable fore/aft, medial/lateral, and rotational via two allen screws attaching to shoe.
- Foot moves opposite of cleat adjustment.
- Initial cleat placement so ball of foot (metatarsal heads) over pedal axle.
- Rotational "float" (amount of allowable rotation on pedal before release) varies with clipless pedal designs: Shimano and most other SPD-type (≤4 degrees) less than time ATAC (approximately 5 degrees), Crank Brothers Candy or Eggbeater (approximately 6 degrees), Speedplay Frog (20 degrees "free float").
  - Amount of ideal float varies with terrain and biomechanics of rider-bike interface, must be individualized; less float = more power, more float = increased muscle work to main-

tain foot stability on pedal, but more versatility for small adjustments over rough terrain.
- Set initial rotational cleat position (center of float; toe-in, neutral, or out) to individual foot mechanics: may estimate by observing foot rotation of rider sitting on table with legs dangling (hip and knees at 90 degrees, ankles at neutral, have rider bend forward to see if affects foot position) or may determine more exactly using rotational adjustment device (RAD).

Other

- Mountain bike shoes: less stiff than road, more traction on sole (can interfere with clipless mechanism), hybrid hiking-clipless shoes available (some ultra-endurance and epic riders use for significant time spent hiking off bike).
- Crank arm length: mountain bikers use long levers, usually 175 mm, sometimes 170 mm on smaller frames.
- Cycling orthosis and anatomical footbeds:
  - Extend through metatarsal head where force transfer occurs.
  - More rigid than running orthotics to provide better control and force transfer (with varying foot orientation on pedal).
  - Anatomical footbeds incorporate a varus wedge to accommodate canted forefoot position, longitudinal arch support to optimize force transfer, and metatarsal button to minimize nerve and vessel compression at metatarsal heads with pedaling.
- Shims, medial (varus) wedges, and spacers:
  - **Make adjustments to longest leg first.**
  - Shims: if leg length discrepancy (LLD) is more than 6 mm, use shim (especially with back and knee pain); between 3 to 6 mm may simply move cleat on long leg back and/or cleat on short leg forward 1 to 2 mm or combine shims and cleat position (especially femoral-based LLD).
  - Commercial wedges, bio-wedges, or custom wedges available (use material with minimally compressibility and high torsional rigidity).
  - Threaded spacers better than washers to achieve ideal stance and accommodate varus knee malalignment; placed between pedal and crank arm.
  - May correct femoral LLD using combination of shim on shorter leg and cleat position.
- **Rider-bike-terrain interface:** See Figure 68-2 for factors and components to consider with bike fit and terrain goals.

PHYSIOLOGY AND TRAINING

Physiology

- High intensity sport—probably higher than road stage races, especially XC.
- XC circuits average about 2 hours, performed at heart rate (HR) of approximately 90% (± 3%) of HRmax, corresponding to $\dot{V}O_2$ max of approximately 84%; more than 80% of race time spent above lactate threshold (LT).
- Intensity related to fast start, several climbs, rolling resistance, and isometric and eccentric arm and leg muscle contractions required for shock absorption, bike handling, and stability over rough terrain (increases HR response to submaximal cycling).

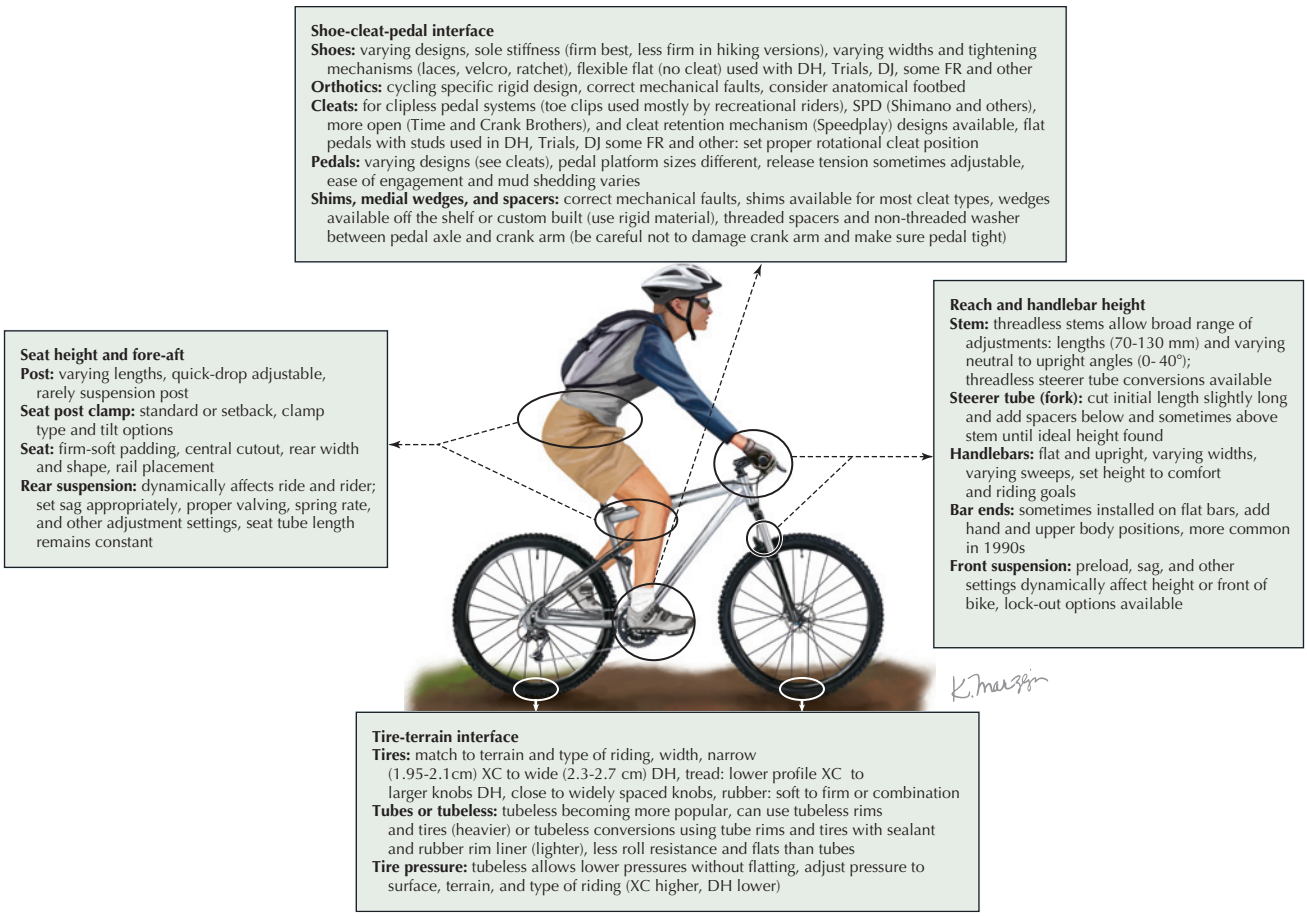

Figure 68-2 Rider-Bike-Terrain Interface.

- Start has fundamental importance to entire race; XC riders race to the narrows (where trail becomes singletrack) to achieve good position; fast starts and early steep climbs lead to high intensity and max HR early in race.
- Anaerobic energy systems taxed, especially during steep climbs (require high power output: up to 250 to 500 watts); anaerobic power and ability to sustain high work rates for prolonged periods are prerequisites for competing in high-level off-road cycling events.
- Various factors may affect off-road XC performance, especially in elite cyclists: $\dot{V}O_2$ max, peak power output (PPO), power output (PO), and $\dot{V}O_2$ at VT (ventilatory threshold) and RCT (respiratory compression threshold); studies conflicting about which most important:
  - PO and $\dot{V}O_2$ at RCT normalized to body mass are predictors of off-road performance times.
  - $\dot{V}O_2$ max, PPO, and LT normalized to body mass correlate with XC performance in some studies.
  - PO at VT may correlate with time trial performance.
- Body mass a factor: power to weight characteristics are important for success in off-road events; high power-to-weight ratio good for strong hill climbing ability; higher mass may assist with rapid descents.
- Factors other than aerobic power and capacity may affect off-road cycling performance: cycling experience and economy (specificity of principle), technical ability, and pre-, during, and post-competition nutrition.

Training

- Need to develop good aerobic endurance, anaerobic capacity, overall muscle strength, good coordination, and bike-handling skills.
- Significant upper body and core muscular strength necessary for repeated isometric and/or eccentric muscle contractions required to absorb shocks and constantly adjust to changing terrain; accomplished using weight training and riding off-road.
- Many competitive XC riders train 10 to 14 hours weekly; some XC racers train systematically similar to road racers; some train with much less structure.
- Ride at varying aerobic and anaerobic intensities (zones); helpful to use heart rate monitor.
- Mountain bikers train both on- and off-road.
  - Use different types of training similar to other endurance events (listed in order of decreasing intensity): race pace, intervals and hills, speed and tempo, endurance, strength, recovery or overdistance.
  - Training cycle (depends on peaking goals): base (4 months), intensity (4 months), peak (4 to 6 weeks), racing (8 to 12 weeks), recovery (4 to 6 weeks).
  - Off-road terrain incorporates various training types and is less flexible than road riding in controlling intensity
  - Long, off-road rides over rough, technical terrain require longer recovery compared to equidistant road rides
  - Use periodization and monitor for overtraining, especially if training at high altitude and frequently on rough terrain.

INJURY OVERVIEW

Epidemiology

- Injury types and numbers likely grossly under-reported secondary to varying, limited study design, difference in injury definitions, understudy of recreational and large, noncompetitive aggressive riders.
- Peak incidence: June to August.
- 50% to 90% riders injured in previous year (mixed competition and noncompetition data); one study shows 20% had significant injury requiring medical attention and prevented from cycling at least 1 day; competitive cyclist injured more than recreational in earlier studies, but unclear if this reflects current trend.
- Reported injury rates (multiyear data) as low as 0.45% to 0.6% per year in XC, DH, DS competitions; 0.30% recreational; some reports higher; definition of injury rate may vary (e.g., number injured cyclists per number of starts × 100; number injured cyclists per 100 hours race time).
- Injury rates are greater for DH versus XC relative to time spent on bike (0.37 injuries per 100 hours on bike for XC vs. 4.34 injuries per 100 hours on bike for DH).
- Possible association between increased hours on bike and severity of injuries, but some data points to fewer injuries in competitive cyclists who spent 1 hour per week more on bike in competition season and 3.5 hours per week more on bike in off-season compared to those who experienced major injuries.
- Cannot extrapolate injury data from road bikers because road bike crashes often specific to riding on pavement, many more collisions with motor vehicles, rare in off-road cycling (are a few case reports of serious injury resulting from mountain bike-motor vehicle collision).
- Experienced cyclists may injure bones and joints more frequently than beginners.
- Professional DH racers are more likely to get injured than amateurs.
- Young males most frequently injured population, because of popularity of sport in this group and likelihood to engage in aggressive and technically demanding riding styles.
- Injury risk in competing females may be greater than for males.
  - Loss of bike control, less upper extremity strength, fewer riding years.
- Number of injuries occurring during racing and training about equal, but traumatic more common in races, overuse in training.
- Injuries are divided into **overuse, acute traumatic,** and **environmental.**

Mechanisms of Injury and Risk Factors

- Most common reported: **excessive speed**, **unfamiliar terrain**, **loss of control** (encompasses multiple factors), **inattentiveness**, **riding beyond ability**, **riding DH**, **slippery terrain** (approximately 90% may be viewed as errors in judgment) (Table 68-1).
- **Most crashes occurred while riding DH.**
- Injuries from loss of control, loss of traction, and mechanical failure result in similar injury patterns.
- Special attention is applied to the cyclist-bike-terrain interface and how it relates to injuries.

Falls

- Forward fall over handlebar (endo)
  - **Most common direction of fall** and **mechanism of acute traumatic injury.**
  - Common causes are rapid deceleration DH (most common cause of severe injury), hitting object, improper jump landing, improper braking.
  - Reported injuries are soft tissue injuries, trauma to head, torso, shoulder, upper extremity; head, neck, face injury.
- Side falls
  - Common causes are sliding out around corners (sideslip), misjudged handling resulting in tip over, dabbing hand after losing balance in technical terrain.
  - Reported injuries are soft tissue injuries, leg injuries, especially to knee and ankle; some upper extremity (reaching out or fall on lateral shoulder).

Table 68-1 SPECIFIC REPORTED CAUSES AND RISK FACTORS

Rider related	Errors in judgment and riding technique Excessive speed Inattentiveness Riding beyond ability and loss of control Incorrect braking Improper training and overtraining Female or young male Intoxication No helmet: especially children
Bike/equipment-related	Mechanical failures (more common in DH): flat tires, brakes, chains, forks, handlebars, pedals, suspension parts, seatposts, frames Improper fit
Terrain-related	Surface: mud, gravel, loose dirt, wet Unfamiliarity Downhill Obstacles and jumps
Environment-related	Competition Heat Cold Sun Lightning Orienteering mishaps Animals and reptiles (attacks and collisions)

- Rear falls
  - Common causes are forceful wheelie, preload jump too early.
  - Reported injuries are soft tissue, upper extremity (especially hand and wrist), head, spine and torso, tailbone.

Collisions

- Collisions with other cyclists common in XC.
- Also with stationary object (trees or rocks).
- Bike parts, especially bar, stem, and pedals, frame, brakes, seat less common.
- Animals (prairie dogs and other).
- Injuries from collision and noncollision similar in severity and anatomic location.

Evaluation

History

- Review **training history** to detect common errors (e.g., volume, intensity, hill work, periodization).
- Acute traumatic or overuse?
- **Bike fit** history (professional vs. self).
- Experience, type of bike and riding, type of terrain and challenges.
- Helmet and other protective equipment use.

Exam

- Use stationary trainer in office for overuse and fit issues (dynamic evaluation); may use digital camcorder.
- Evaluate on and off the bike. Identify anatomic variations or malalignments (e.g., excessive knee valgus, LLD) and errors in bike fit; adjust rider, evaluate shoes and orthotics if used.
- Helpful tools: plumb line, long carpenter's level, goniometer, suspension pump, zip ties (for sag settings), allen wrenches and screwdrivers (for quick office adjustments).

General Treatment

- General treatment approaches for traumatic and overuse injuries in off-road cyclists are similar to approaches used with other athletes (e.g., physical therapy, cryotherapy, antiinflammatory medication).
- Specific cycling injury diagnosis and treatment is a highly individualized area; affected by multiple variables including training, experience, riding style and type, and bike fit.
- Relative rest and activity modification: temporarily decrease mileage, intensity, hill work, and spin using low-resistance and high-cadence pedaling; correct training errors.
- Consider placing rider back to neutral position, especially if bike never fit; adjust from there.
- Medication: nonsteroidal anti-inflammatory drugs (NSAIDs) for analgesia and inflammation, bacitracin ointment for soft tissue trauma.
- Ice appropriate overuse injuries after rides and intermittently throughout day; keep affected joint warm during ride (e.g., knee warmers).
- Physical therapy (see specific injuries)
  - Lower extremity (especially hamstring, iliotibial band) flexibility
  - Back and neck flexibility
  - Strengthening: lower extremity (eccentric programs for Achilles, patellar, and hamstring tendinopathy), dynamic core stabilization, upper extremity
  - Plyometrics, proprioceptive, and other cycling-specific coordination training
  - Deep tissue massage and release techniques
  - Neural stretching maneuvers
- Consider ionto- or phonophoresis in appropriate setting.
- Bracing, strapping, or taping where appropriate.
  - Knee: soft patellofemoral brace (e.g., Donjoy, Palumbo), infrapatellar strap, McConnel taping
  - Hand and wrist: off-the-shelf wrist or thumb spica split (can bend steel stay to handlebar, most cycling gloves fit over), custom-molded orthosis (e.g., Orthoplast)
- Corticosteroid injection.
  - Upper extremity: carpal tunnel syndrome, De Quervain's tenosynovitis, intersection syndrome
  - Lower extremity: pes anserine bursitis, trochanteric bursitis, adductor magnus tendonitis, Morton's neuroma

SPECIFIC INJURIES

Overuse Injuries

Epidemiology

- Retrospective, questionnaire surveys indicate 45% to 90% of mountain bikers affected.
- Body regions most commonly involved: **knee** and **low back** most common, hand, wrist, neck, and buttocks/saddle region.
- Likely grossly under-reported: poorly studied, probably common.
- Many studies evaluate injuries resulting in lost time on bike; excludes many overuse injuries.
- **Fit closely linked,** interaction between cyclist, bike, and terrain.
- Anatomic variations and fit errors (even a few mm) magnified by many hours of training, cumulative repetition; especially affects lower extremity.
- Upper extremity injuries related to weight distribution over the front of the bike (affected by bars, bar ends, grips, stem height relative to saddle), shifter type, and front suspension (travel, preload, rebound).
- Causes and risk factors: **too much training** and **improper training progression** (e.g., sudden increases in mileage or riding intensity, climbing too many hills early season, inadequate recovery), too many hours of training/racing, too many different types of riding, too much rough terrain, improper fit, anatomic variations and faults, incorrect saddle position (dynamic), insufficient stretching, incorrect gear ratio (pushing large gears too much, especially early season), not enough

warm-up, wrong shoes, not enough training (inadequate preseason conditioning), cold weather.

- **Treatment and prevention focuses on training and fit.**

Knee

May be most common joint affected by overuse; affects 30% to 40% of mountain bikers.

PATELLOFEMORAL PAIN (BIKER'S KNEE, PATELLAR TENDONITIS, QUADRICEPS TENDONITIS)

Causes: Saddle too low, aggressive sprinting, pushing big gears and aggressive climbing early season, pes planus and overpronation, excessive genu valgum, leg length discrepancy (LLD), crank arm too long.

Treatment: Pedal spacers, medial wedge, or cycling orthotic for excessive knee valgus, raise seat, move cleats rearward, move seat back, higher cadence (90 to 100 rpm), easier gears, avoid hills, shorten crank arm, correct LLD (shim), correct training errors.

PES ANSERINE BURSITIS

Causes: Saddle too high, pedals with too much float, stance too wide on pedals, LLD, overpronation, tight hamstrings, cleats inappropriately neutral or internally rotated, external tibial torsion.

Treatment: Lower saddle; correct LLD (short leg) and overpronation with orthotic, shim, wedge, or cleat position (move anterior); adjust cleats to match foot alignment (e.g., toes point out slightly).

ILIOTIBIAL BAND FRICTION SYNDROME

Causes: Stance too narrow on pedals, cleats inappropriately neutral or internally rotated, saddle too high, pedals with too little or too much float, genu varum.

Treatment: Add threaded spacer or washer between crank arm and pedal (correct varus), adjust cleats to reflect foot alignment (toe out), lower saddle, use pedals with appropriate float, cycling orthotics to control excessive lower leg rotation.

PLICA SYNDROMES (ESPECIALLY MEDIAL)

Causes: Saddle too low, genu valgum, overpronation, internal tibial torsion, excessive pedal float.

Treatment: Raise saddle, cycling orthotics, adjust cleats to reflect foot alignment (toe in), decrease pedal float.

HAMSTRING, POPLITEUS TENDONITIS, AND POSTERIOR CAPSULE STRAIN

Causes: Saddle too high and/or posterior, riding fixed gear bike (hamstrings used to decelerate), genu varum, pedals with too much float, LLD.

Treatment: Lower saddle and/or slide forward, slide forward on seat during steep climbs in granny gear (increases quad workload), pedals with minimal float, threaded spacer or washer between crank arm and pedal, correct LLD, eccentric strengthening program.

ADDUCTOR MAGNUS TENDONITIS AND ADDUCTOR TUBERCLE PAIN

Causes: Saddle too high, genu varum, pushing big gears, LLD.

Treatment: Lower saddle, spin in easy gears, high cadence, correct LLD, adjust cleats (toe in), pedal spacers, steroid injection.

Hip

TROCHANTERIC BURSITIS, ILIOPSOAS TENDONITIS, AND HIP EXTERNAL ROTATOR TENDONITIS

Causes: Weak hip external rotators, genu valgus, overpronation, pedals with too much float, LLD, saddle too high.

Treatment: Strengthen hip external rotators, decrease pedal float, cycling orthotic, medial wedge or shim, lower saddle, consider corticosteroid injection to trochanteric bursa.

Leg and Ankle

ACHILLES TENDONITIS AND RETROCALCANEAL BURSITIS

Causes: Improper pedal form (drop heel, less common toe pedaling; ankling—excessive dorsiflexion and plantar flexion during pedaling), cleat too far forward, overpronation, tight Achilles, shoe rubbing (with Haglund's deformity), saddle too high or too low.

Treatment: Correct pedal form, move cleat rearward, stretch Achilles, raise or lower saddle depending on problem.

TIBIALIS ANTERIOR TENDONITIS

Causes: Saddle too high, ankling.

Treatment: Lower saddle, anterior ankle stretching, tibialis anterior eccentric strengthening, proper pedal stroke.

POSTERIOR TIBIALIS TENDONITIS

Causes: Overpronation, ankling.

Treatment: Cycling orthotic or medial wedge, posterior tibialis eccentric strengthening, proper pedal stroke.

Foot

HOT FOOT (FOREFOOT/TOE NUMBNESS AND PAIN, METATARSALGIA, PARESTHESIAS, AND MORTON'S NEUROMA)

Causes: Improper cleat position, irregular sole (cleat bolt causing localized plantar pressure), tight or narrow shoes, small pedal platform, toe clips, improper rotational cleat adjustment.

Treatment: Move cleats back (lower saddle same amount) and adjust rotation to individual mechanics, loosen toe clips or convert to clipless pedal, install thinner insoles and/or add metatarsal or neuroma pad, larger pedaling platform, wider shoe or shoe with anatomical footbed.

PLANTAR FASCITIS

Causes: Tight plantar fascia, overpronation, excessive pedal float, saddle too low.

Treatment: Stretch plantar fascia, night splint or sock, cycling orthotic or anatomical footbed, decrease pedal float, raise saddle, neutral position or dorsiflexion night splint or sock.

Hand and Wrist

Wrist pain in 19%, hand numbness in 19%, finger tingling in 35%.

ULNAR (CYCLIST'S PALSY) AND MEDIAN NEUROPATHY (CARPAL TUNNEL SYNDROME)

Compression of nerves at Guyon's canal and carpal tunnel probably similar incidence in road and mountain cyclists; presents with motor, sensory symptoms or both; ulnar with most sensory; experienced and inexperienced cyclists equally affected.

Causes: Improper grips (size, shape, firmness), infrequent change in hand position, bumpy terrain, improper suspension settings and tire pressure relative to terrain, lack of suspension, "death grip," lack of glove padding.

Treatment: Proper grip size and comfort fit, frequent changes in hand position (bar ends can be helpful), experiment with bar angles, padded gloves, loosely grip bars, decrease suspension preload and tire pressure, consider front suspension if rigid, stretching on the bike.

DE QUERVAIN'S TENOSYNOVITIS, EXTENSOR CARPI ULNARIS TENDONITIS, AND INTERSECTION SYNDROME

Causes: Occurs especially with grip shift, sometimes with integrated shifters.

Treatment: Wrist and/or spica splint (dynamic) to minimize provocative motion, change shifters to trigger, consider local steroid injection.

Muscle Cramps

Description: Especially affects quads, hamstrings, and calves, some upper extremity.

Treatment: Hydrate with appropriate electrolyte drinks (especially when hot), decrease riding intensity slightly, soften suspension, appropriate training volume, stretching.

Low Back Pain

Description: 37% affected, one of most common overuse complaints, **may be as common as knee pain.**

Causes: Most frequently by improper fit, riding position, or inappropriate progression of training volume and intensity. Specific causes are over-reaching (stem too long or seat too posterior), too much drop, rough terrain, incorrect suspension preload and tire pressure settings relative to terrain, training errors (especially too many hills and long rides early in season).

Treatment: Proper fit, raise handlebars (especially early season), shorten stem, correct LLD, core strengthening (also addressing abdominals and iliopsoas), adjust suspension setting to terrain, lower tire pressure slightly and consider changing to wider tires, hamstring flexibility, proper riding form, avoid excessive bumpy terrain and intense hill climbs early season, change position on saddle during ride and stand intermittently, sometimes need to increase reach if pain related to crowded position (lengthen stem). Disc injuries and radiculopathy frequently worsen with hills and rough terrain.

Neck Pain

Description: Common in mountain cyclists, similar rider-bike causes and treatments to low back pain; ride with elbows appropriately flexed, change neck positions frequently, avoid tensing.

Treatment: Hydration packs worn by most mountain cyclists can augment neck pain. Make sure pack straps are adjusted appropriately. Consider decreasing weight of or eliminating pack by using water bottles and under-the-seat storage pack.

Scapulothoracic Pain

Causes: Common in mountain cyclists, similar causes to neck pain. Scheuermann's kyphosis (classic or atypical) can worsen with mountain cycling, especially over more aggressive terrain. Hydration packs especially contribute to levator scapula, trapezius, and rhomboid myofascial pain and trigger points.

Treatment: Stretching, needling or acupuncture, proper rider form, stretching on the bike (especially long rides).

Genitourinary

PUDENDAL NEUROPATHY

Description: Numbness in saddle region reported in 19%. Males affected more than females; females report sensory symptoms.

Causes: Compression of pudendal nerve (artery may be compressed too), especially after repeated long hill climbs.

Presentation: Perineal, penile shaft, and scrotal numbness, impotence/erectile dysfunction and priapism (rare).

Treatment: Proper bike fit (especially seat height and fore-aft position and tilt), make sure saddle not tilting up, lower seat, consider change to a softer with a central cutout, use quality cycling shorts with adequate padding, increase handlebar height (distributes weight more posteriorly over ischial tuberosities), stand during long climbs.

SKIN PROBLEMS RELATED TO SADDLE

Description: Chafing, callus (ischial tuberosities), maceration and ulceration, and painful nodules. Painful nodules (saddle sores) likely inflammatory (repetitive friction of hair follicle with resultant inflammation and scarring), but can be infectious; considerations: folliculitis, furuncles, carbuncles, pseudocysts.

Treatment: Aimed at prevention. Proper seat height and fore-aft, level seat position, individualize saddle fit (consider softer saddle with center cutout and/or broader rear), proper reach and bar height, proper hygiene (skin and shorts, shower immediately after ride, and wash cycling shorts between every ride), intermittent standing, avoid wearing underwear under high-quality, well-padded cycling shorts, lubricants or chamois creams frequently used (petroleum-based probably better than water-based), occasional medications (antibiotic ointment [bacitracin]) for infection, antibiotic gels (erythromycin or clindamycin) sometimes tried if recurrent folliculitis or hidradenitis suppurativa, rare corticosteroid injection of painful nodule, sometimes surgical resection.

URETHRITIS

Description: Traumatic irritation of urethra from rough terrain and/or improper rider position and fit.

Symptoms: Variable; urethral paresthesia, dysuria, hematuria, sometimes pyuria.

Treatment: Similar to pudendal neuropathy.

PROSTATE

Description: In healthy men the measurement of total prostate specific antigen (PSA), free PSA and complexed PSA is not disturbed by preceding long distance mountain biking or endurance exercise.

Treatment: Based on the present data, there is no evidence for a recommendation to limit bicycle riding or physical activity before the measurement of PSA.

Overtraining

Description: Aggressive terrain, often with repeat hill climbing, may contribute to overtraining in mountain bikers who are not careful to build in adequate recovery between rides. May especially be a factor in early season, after "melt out" in colder regions, when mountain bikers tend to get spring fever and increase training volume rapidly because of desire for trail time. Altitude training may increase need for recovery.

Treatment: See Chapter 23, Overtraining. Mountain cyclists often complain of "heavy" legs on the bike, especially during hill climbs (loss of power also noted) and with stairs.

Acute Traumatic Injuries

Epidemiology

- Most injuries are minor (e.g., skin and soft tissue wounds); second most common are orthopedic injuries such as fractures, sprains and dislocations, most commonly affecting upper extremity in XC and DH studies (Table 68-2).
- Reported more than overuse injuries in literature.
- Injury severity measures vary: level of treatment required, lost riding time; Injury Severity Score (ISS), National Athletic Injury Reporting System (NAIRS).
- General trend of frequency for body area (not specific to riding style): upper extremity (fingers and wrist most common) more common than lower extremity (knee and ankle most common), which are more common than head and trunk injuries.
- **Extremity injuries** more common than any other body area.
- General trend of injury type, in first-to-last order of greatest incidence: skin lacerations, wounds, and contusions; joint injuries (ligament sprains and dislocations); fractures; muscle injuries; brain injuries (concussions most common); dental injuries.
- General trend of FR injury type, in first-to-last order of greatest incidence: orthopedic, head, spine, chest, facial, abdominal, genitourinary.
- Ligament injury trend by location, in first-to-last order of greatest incidence: acromioclavicular (AC) joint, knee, ankle, fingers.
- Off-road cyclists sustain more fractures, dislocations, and concussions than road cyclists.

Table 68-2 BICYCLING-RELATED INJURIES

Injury	%
Orthopedic	46.5
Fractures and dislocations	68
Lower extremity	29
Upper extremity	25
Soft tissue	29
Other (including nerves and tendons)	3
Head	12.2
Fatality	Rare, most commonly due to intracranial hemorrhage
Other	
Spine	12
Chest	10.3
Facial	10.2
Abdominal	5.4
Genitourinary	2.2
Neck	1

Most riding likely FR (North Shore style riding); 1992-2002; 399 patients sustained 1092 injuries while mountain biking; 1037 total patients identified with bicycling-related injuries. 67% patients required surgery.

Data from Kim PT, Jangra D, Ritchie AH, et al: Mountain biking injuries requiring trauma center admission: A 10-year regional trauma system experience. J Trauma 60(2):312-318, 2006.

- Causes and risk factors (see "Mechanisms of Injury and Risk Factors"): Often multifactorial, some connection to fit (related to bike control), falls/unscheduled dismounts, collisions, problems releasing from pedals (inexperience and release adjustments for some pedals similar to ski bindings; avoid pulling up and off pedal), contact with rocky terrain.

Skin and Soft Tissue

- **Contusions, lacerations, and abrasions most frequently reported injuries** (up to 65% to 75% of reported injuries): contact with rocky, varied terrain, sometimes pedals and chainrings.
- Road or "rock" rash: Abrasions affecting small to large surface areas, usually lower extremities (some upper and torso), caused by contact with rocks and other rough terrain. Appropriately irrigate and cleanse, cover with hydroactive dressing (DuoDerm) and antibacterial ointment (bacitracin).
- Prepatellar bursa laceration, scalping, olecranon bursa laceration, ragged lower leg lacerations (problems releasing from clipless pedals, injury on chainrings, grease in wound, skin loss, debridement and delayed primary closure concerns).

Upper Extremity

- Upper extremities (57%) injured more frequently than lower (21%) (not FR).
- **Shoulder: most frequently injured joint in mountain biking.**
  - Dislocation and rotator cuff injuries (arm raised during forward fall, anterior or posterior depends on arm position relative to body with fall).
  - **Clavicle fracture** and acromioclavicular separation: falls on lateral shoulder common, sometimes force transmitted through arm axially.
  - **Acromioclavicular separation most common joint injury.**
  - Prevention: Learning how to fall and roll, especially over bars: snap bike under you like center snaps a football, tuck and roll (can turn endo into two-footed landing or tuck and roll); fall to side: avoid locking elbow while reaching, absorb impact with whole body.
- **Finger** dislocations and fractures, metacarpal fractures.
  - Falls and catching finger on object (e.g., sapling) while riding.
  - Buddy taping, clamshell type or custom-molded Orthoplast brace for protection (mold using handlebar).
- **Wrist injuries:** falls resulting in wrist hyperextension, bracing and collisions, momentum.
  - Sprains, including distal radioulnar joint and intercarpal ligaments.
  - Triangular fibrocartilage complex tears.
  - Extensor carpi ulnaris subluxation.
  - Fractures: radius most common, ulna, scaphoid, hook of hamate.
  - Prefabricated wrist splints and custom orthoses (Orthoplast) that limit provocative repetitive motion may allow earlier return to riding.
- **Elbow:** falls resulting in direct impact or axial load.
  - **Radial head fracture (most common elbow fracture)**
  - Subluxation/dislocation
  - Olecranon fracture
  - Medial epicondyle fracture

Lower Extremity

- Hip and pelvis: side fall with lateral impact
  - Trochanteric bursitis (traumatic)
  - Intra-articular injuries: labral tears, bone bruise, hip fractures
  - Pelvic fractures
- Knee: more frequently injured with falls in DH and FR than in XC
  - Ligament injury common (especially FR and DH), collaterals (especially medial), probably more than anterior cruciate
  - Meniscal injuries occur, other intra-articular injures less common
- Ankle and foot: forward momentum augmented by bike rider plus bike weight, combined plantar flexion and inversion on contact foot/ankle
  - **Inversion sprains most common**, syndesmosis and medial sprains sometimes occur
  - Other intra-articular injuries (e.g., talar dome lesions)
  - Can sustain midfoot sprains, but less common, probably due to relative protection from torsional rigidity of cycling shoes
  - Prevention: Stable, torsionally rigid shoes with strong heel counter, practice dismounts to enhance proprioception and reaction time, bracing, strengthening.

Fractures

- **Second most common injury behind skin and soft tissue**
- Upper extremity incidence greater than lower extremity overall.
- Women affected more than men (bone mineral density issues suggested, but not proven). Bone mineral density for male mountain cyclists higher than comparable road cyclists.
- **Clavicle most common fracture,** fingers may be second, radial head, distal radius (also common), olecranon, medial epicondyle, scaphoid, metacarpals, phalanges, and other forearm fractures.
- Other reported fractures:
  - Pelvis (few details available), proximal femur, open and closed tibia and patellar.
  - Tarsometatarsal fracture-dislocation.
  - Trunk: ribs and scapula.
  - Cervical, thoracic, and lumbar spine.
  - Likely others.

Muscle Strains

- Back, thigh, and calf common.

Spine

- Cervical and lumbar fractures reported.
- **Cervical** most common site of injury, followed by thoracic, then lumbar.
  - Most common cervical spine injury caused by fall over bars and landing on head.
  - Cord injuries reported in FR (24% total spine injuries). Some paraplegics, quadriplegics, central cord syndromes, some nerve root injuries.
  - About half of reported FR spine injuries required surgery.
    - Vertebrae C2 to C3 subluxation with cord compression (halo traction treatment, result incomplete; C3 tetraplegia).
    - Vertebrae C6 to C7 bifacetal dislocation with cord transaction and cord edema to C2 (halo traction treatment, result: C3 complete tetraplegia necessitating tracheostomy and ventilatory support).
    - Vertebra C4 burst fracture body, C3 lamina fracture, nondisplaced C1 arch fracture (C4 vertebrectomy with grafting and plating, result C4 complete motor and incomplete sensory tetraplegia).
  - All of the above listed injuries involved falls on head; mechanisms flexion-compression and axial compression; helmet showed significant damage in all three cases.
  - Assume cervical spine injury if altered mental status and damaged helmet; have low threshold to get CT and/or MRI if plain radiographs are negative and suspicion high.
  - Cervical spine least protected and most mobile region of vertebral column.
- T5 to T6 vertebral body fracture and lumbar fractures reported.

Abdomen and Chest

- Uncommon in mountain biking; blunt trauma from bike bars and bar ends.
- Chest wall injuries most common.
- Liver-subcapsular hematomas (straight bar ends).
- Pancreatic transaction.
- Small bowel evisceration.
- Renal laceration and hemorrhage.
- Hemothorax and hemopneumothorax.
- Blunt cardiac injury (uncomplicated).
- Solid organ injuries more common than hollow viscus or abdominal wall injuries.
- **Spleen most frequent solid organ injured** (49%), then liver (15%), adrenals and pancreas reported.
- Small bowel most frequent viscus, then mesentery, then colon, then omentum.
- Spleen injuries required surgery in 25%; 17% of liver injuries and most bowel injuries required surgery.

Other

- Genitourinary
  - Straddling most common mechanism of injury
  - Cavernosal artery laceration
  - Perineal and vulva laceration, contusion, hematoma
  - Scrotal and testicular injuries more common than penile shaft
  - Ureter injuries
- Brain
  - Concussion in 3% to 13%, fall over bars most common mechanism of injury
  - Intracranial hemorrhage rare, occurs with helmet use, most common cause of death; May be more common in FR than DH, followed by XC.
  - Bleeding or contusion to cerebral cortex more common than cerebellum, followed by brainstem.
  - Most mountain bikers wear helmets (80% to 90%), but less commonly used in children.
- Eye
  - Foreign objects: bugs, gravel, tree branches, sleet
  - Shatterproof lenses and goggles (DH) help prevent
- Facial and dental
  - Usually caused by fall over bars
  - Facial and dental fractures, lacerations, and abrasions
  - Most common facial fracture: maxilla (FR)
- Fatal
  - Brain injury, ruptured diaphragm, transected coronary vessel, pulmonary contusion, other nonreported

Environmental Injuries

- These injuries reported rarely or not reported in literature, but remain a concern, especially for mountain bikers who venture far into the backcountry on take epic rides.
- Pulmonary: Exercise-induced bronchospasm (especially in cold weather), dust bronchitis (mass start endurance and ultra-endurance events), high-altitude pulmonary edema (especially events at higher than 10,000 feet).
  - Pharmacologic pretreatment (salmeterol in longer events for exercise-induced bronchospasm [EIB]), wear mask or cover mouth and nose with handkerchief in dusty conditions.
- Altitude illness (acute mountain sickness most common).
  - Adequate acclimatization, pharmacologic prevention if history.
- Exposure: heat illness, cold injury, sunburn.
  - Adequate hydration pack and planning, light-colored and breathable jerseys (heat), adequate helmet ventilation, sunscreen, proper layering and coverage (gloves, head cover, shoe covers, windproof jacket and tights) for cold.
- Dehydration: "Roadies" traditionally use water bottles held in cages attached to frame, but mountain bikers typically use hydration packs that carry much larger volumes of replacement fluids and other necessities.
  - Match fluid reservoir size to needs of event, timed drinking (using slight weight loss and urine color to monitor needs in long events), carry water purification tablets or small filter in backcountry.
- Weather: sunburn, lightning, sudden rain or snow, changing temperatures.
- Wildlife: attacks rare (cougars, rattle snakes), collisions (prairie dogs and other small animals, cause crash).
- Collisions: trees, rocks most common; cliffs and drops less common; motor vehicles less common than in road cycling.
- Orienteering mishaps and getting lost.
  - Carry detailed topographic maps, compass, GPS.
  - Use buddy system, carry personal identifier, notify others of planned starts and stops.
  - "Adventure pack" with appropriate tools, extra calories, head lamp, fire-starting material, extra clothing layers, appropriate first aid, cell phone if access.

INJURY PREVENTION

- Appropriate bike fit (helps prevent overuse injury more than acute traumatic injury).
- Mountain biking skills development (acute traumatic > overuse): workshops or clinics, skills manuals, home skills drills, and riding time and practice can assist in developing mountain biking–specific skills that will improve riding and reduce injuries (Box 68-1).
- **Riders, sponsoring organizations, manufacturers:** improvements in rider training, race course design, and safety equipment.
- Rider-related: technical skill and neuromuscular coordination drills; strength and endurance training, especially upper body

and core; awareness of limitations (riding near "the edge"), especially when riding with more experienced cyclists or on unfamiliar terrain; avoid intoxicants.
- Bike/equipment-related: padded and rigid protection (e.g., bulky chest and shoulder protectors, shin guards), full face helmets for DH and FR, appropriate shoes (clipless, stiff sole, flat bottom in DH and FR), padded shorts, padded gloves, regular bike inspection and maintenance, appropriate tire setup (tubeless conversion, appropriated width, tread, and compound for setting), wheel build strength matches riding type (rim strength, number and size of spokes, type of hubs), grips (cover ends, no-slip lock, curved bar ends [if used]).
- Terrain-related: responsible race organizers; avoid exciting "spectator spots," adjust course and number of laps to skill and competition level; note risky surfaces (gravel, mud, wet).

BOX 68-1 *Basic Mountain Biking Skills*

- Mounting and dismounting in various situations
- Braking
- Cornering
- Wheelies and hops
- Drops (slow and fast)
- Jumping
- Line selection and flow
- Pedaling efficiency
- Climbing
- Switchbacks

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Tennis

69

Robert E. Mayle, Jr., Todd Ellenbecker, and Marc Safran

GENERAL OVERVIEW

Description

- More than 20 million people in the United States participate in tennis at least once a year.
- Five million people play tennis at least twice a month.
- More than 500,000 adolescents participate in tennis.
- Roughly 650,000 people in the United States play at the competitive level.

Epidemiology of Tennis Injuries

- Injuries to the lower extremity and spine account for 50% to 75% of all tennis injuries.
- Elite players tend to have more injuries to the lower extremities and spine, whereas recreational players incur more injuries to the lower extremities.
- The majority of injuries are sprains or repetitive trauma overload injuries.

Physiology

- Intermittent high-intensity exercise that requires aerobic and anaerobic fitness.
- Tennis is a noncyclical anaerobic sport (10% to 30%) with an aerobic recovery phase (70% to 90%).
- Single rallies may last only 3 to 8 seconds, but complete matches may last 3 hours.
- Over the course of a match, 300 to 500 bursts of effort may be expended.
- Tennis requires elements of quickness, endurance, strength, flexibility, reaction time/speed, agility, and coordination.
- Movements include sprinting, twisting, side-to-side running, sliding, jumping, lunging, and quick stops.
- Heart rate for singles tennis can average more than 160 beats per minute and average more than 80% of the player's maximal heart rate.
- Depending on conditioning, age, gender, intensity of play, hydration status, and environment, a player may lose 0.5 to 2.5 liters of water per hour of play.
- Conditioning includes both aerobic fitness and anaerobic fitness. Progressive resistance strengthening of key muscle groups is important, including strengthening of the core and scapular stabilizers.

Equipment/Facilities

Racket composition: Change in manufacturing materials has resulted in rackets that are larger, lighter, stiffer and more powerful than rackets of the past, though the effect of these variables in injury prevention and production is unclear.

Court surfaces: Play a role in types of injuries seen. Include clay, hard, grass, and indoor.

Clay surface: Loose surface causes the ball to lose speed rapidly and bounce higher. Allows increased time for opponent to reach/return the ball. Considered a "slow" surface. This allows for longer rallies and matches, resulting in overuse injuries. The forgiving surface is more gentle on lower extremity joints. However, due to loss of speed of the ball, power to hit the ball hard must come from the kinetic chain, resulting in upper body and back overuse injuries as well.

Hard surface: Concrete, coated asphalt, Rebound Ace. Balls bounce low, giving hard-hitting players an advantage. Considered a "fast" surface. Harder impact to the lower extremities, with stress fractures of the lower extremities and other injuries such as patellofemoral pain more prevalent. Sudden stops and starts make tennis toe and ankle sprains more common on this surface. Because powerful serve more important on this surface, injuries related to serving are more common.

Grass surface: Grass grown on hard, packed soil. Balls tend to slide and bounce low, making returns difficult. Favors the serve-and-volley player. Considered the "fastest" surface. However, uneven-ness of the surface may result in slipping and sprains.

Indoor courts: Allow for year-round play. Usually hard court type surface.

MECHANICS OF TENNIS

Kinetic Chain

- The kinetic chain is the transference of force efficiently from the ground to the racket through the coordinated sequencing of the legs, hips, trunk, and upper extremity.
- Each segment transfers more energy than the previous one, resulting in maximal racket acceleration.
- Fluid motion through the kinetic chain is essential to generate a powerful swing and minimize the risk of injury.

Strokes

- 75% of play involves the forehand and serve.

The Serve

- Considered the most important stroke of the game, and also the most commonly associated with injury.
- The service motion puts significant stress on the lower extremities, spine, abdomen, and shoulder, though certain phases of the serve are more apt to cause injury than others.
- Four phases: wind-up, cocking, acceleration, and follow-through (Fig. 69-1).

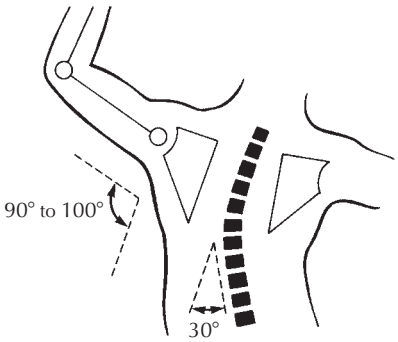
PHASES OF SERVE

Wind-up: From the standstill (ready) position, this phase is the initiation of the serving motion; it ends with the toss of the ball by the contralateral extremity (when the ball leaves the hand). The lower extremities prepare for the buildup of power that occurs in the cocking phase, as the knees and hips bend. The hips and back rotate toward the dominant extremity (see Fig. 69-1).

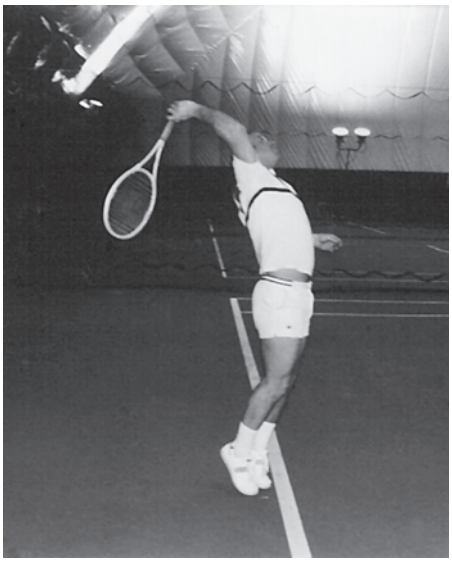
Cocking: From the release of the ball for the toss, through the point where body motion stops moving backward, toward the dominant extremity side. The arm holding the racket appears to be in maximal external rotation (see Fig. 69-1). This phase is characterized by the building up of power. Energy is stored by prestretching of the muscles. Subject to injury:

- Anterior shoulder capsule is tensioned to its physiologic limit with the shoulder in maximum external rotation.
- Glenoid labrum serves to help prevent shoulder subluxation during external rotation and translation.
- With abduction and external rotation of the humerus, internal impingement may occur.
- Muscles of the lower extremity during extension.
- Intervertebral disc, pars interarticularis with hyperextension/rotation of the spine.
- Wrist extensors.

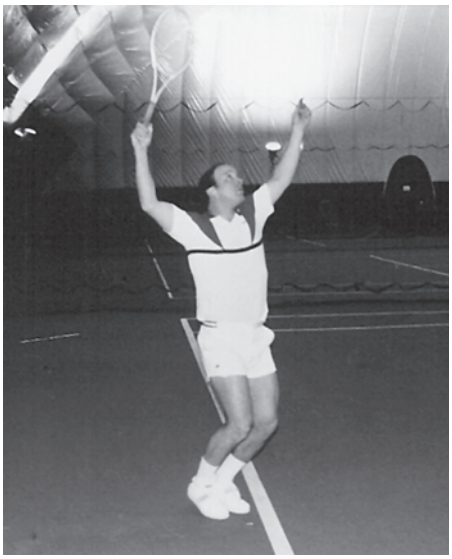
Acceleration: This phase is characterized by the body moving forward and up, as the dominant shoulder appears to be in even greater (maximal) external rotation initially and ends at ball



This view of service wind-up illustrates the addition of 30° lateral thoracic tilt to the 90° to 100° of scapulohumeral abduction.



Back and neck hyperextension should be avoided during the service motion.



Service ball toss in front of service line ensures hyperextension is avoided.

Figure 69-1 Phases of Serve.

impact (see Fig. 69-1). High muscular activity is noted with peak activity prior to ball impact as energy is transferred along the kinetic chain. Internal rotation of the humerus is responsible for 40% of the racket speed at impact. Fluid motion through the kinetic chain is crucial. Injury to one segment will lead to a loss of power and place another segment at risk for injury. Subject to injury:

- Muscular overload of the rectus abdominus, obliques, hip adductors and rotators, rotator cuff, glenohumeral internal rotators and adductors, elbow extensors, and wrist flexors may occur.
- Superior labrum with extreme abduction and internal rotation of the humerus.
- Ulnar lateral collateral ligament and the flexor-pronator muscles of the elbow secondary to valgus stress.
- Extensor carpi ulnaris tendon at the wrist with hypersupinated, ulnarly deviated wrist (for topspin/slice).

Follow-through: Ball impact through completion of the stroke. Activation of shoulder musculature is required to decelerate the humerus to maintain glenohumeral stability. Long axis rotation through the arm, via shoulder internal rotation and forearm pronation, help in the dissipation of forces. Subject to injury:

- The posterior shoulder muscles contract eccentrically to slow the internal rotation of the shoulder.
- The rotator cuff acts to maintain the humeral head within the glenoid.
- The biceps function to slow forearm pronation, elbow extension, and assist in stabilizing the glenohumeral joint.
- The posterior capsule is placed under tension as it counters distraction forces.
- Scapular motion combined with contraction of the infraspinatus muscle places the suprascapular nerve, located in the spinoglenoid notch, at risk for injury.

KINEMATICS

- Racket speeds reach a peak velocity of 62 to 83 mph.
- Ball velocities reach 83 to 153 mph.
- Above speeds are achieved in 0.2 to 0.3 seconds from the end of the cocking phase until ball contact.
- Shoulder internally rotates at 1100 to 1700 degrees per second.
- Elbow flexes to 120 degrees during late cocking and extends to 15 to 20 degrees of flexion at ball impact, resulting in an extension velocity of 900 to 1000 degrees per second.
- Forearm pronation has been recorded at 350 to 900 degrees per second prior to ball impact and has been documented to increase to 1300 degrees per second, 0.1 seconds after impact.
- Wrist speeds approach 1000 degrees per second, 0.1 seconds prior to ball impact; range of motion (ROM) of wrist during a serve is 90 to 100 degrees.

Ground Strokes

- Forehand and backhand.
- Each stroke has three phases: preparation, acceleration, and follow-through.
- Each stroke may be hit open stance (lead foot more parallel to the net) or closed stance (lead foot toward the net).

Backhand

- The backhand swing may be performed one-handed or two-handed. The one-handed backhand stroke allows the player to have a better reach and the ability to slice the ball. The two-handed backhand stroke requires less arm strength, but requires more trunk rotation and may result in greater power.
- There is an increased incidence of lateral elbow pain in novice players in the backhand stroke, particularly the one-handed

backhand. This is attributed to not using the trunk and shoulder musculature properly, placing more stress across the elbow joint, and from hitting the ball with the wrist in flexion (versus extension). Players should strive to hit the ball in front of their body to reduce injury to the elbow.

- Using a two-handed backhand reduces the risk of elbow injury, because of the greater need for trunk rotation to hit the backhand while maintaining proper elbow and wrist position.
- Wrist pain is also common with the two-handed backhand stroke, as players "flick" the racket at ball contact. Proper technique, taping of the wrists, and strengthening exercises will reduce injury to the wrist during this stroke. The nondominant wrist is more often injured in those using a two-handed backhand, during the preparation phase when this wrist is in an extended and supinated position.

Forehand

- Players may hit the forehand shot with a Western grip or an Eastern grip.
  - The Western grip allows for the production of topspin, and is more suitable for play on clay or slow hard courts. This style, however, places more of a valgus stress on the elbow.
  - The Eastern grip allows for the player to slice the ball and is used on grass or carpet surfaces by serve-and-volley type players.
- The forehand shot has three phases: preparation, acceleration, and follow-through.

SPECIFIC INJURIES

Upper Extremity

Shoulder

Overview: 35% of junior tennis players complained of shoulder pain at some point. More than 50% of older players and elite athletes note shoulder pain at some point in their career. A majority of their shoulder pain is caused by impingement and instability.

King Kong arm: Drooping and hypertrophy of the musculature of the shoulder girdle of the dominant upper extremity from repetitive use. This is attributed to eccentric stretching of the posterior shoulder and scapular stabilizers.

Rotator cuff inflammation: One of the more common causes of shoulder pain, attributed to the chronic, repetitive swinging of the racket. Rotator cuff inflammation in the young player is more often due to instability, where posterior capsular tightness, impingement, and instability are factors in the older player. Decreased strength of the scapular stabilizers and external rotators and decreased flexibility with internal rotation are associated with shoulder instability. Impingement of the rotator cuff between the acromion and humeral head is more likely to occur with serves, overhead shots, and high volleys as opposed to ground strokes (Fig. 69-2). Players will present with subacromial pain and referred pain to the lateral arm. They may claim that their arm "feels dead" during play. Persistent inflammation may lead to tears of the rotator cuff (see Fig. 69-2). Avoid activities that may further aggravate shoulder symptoms/pathology.

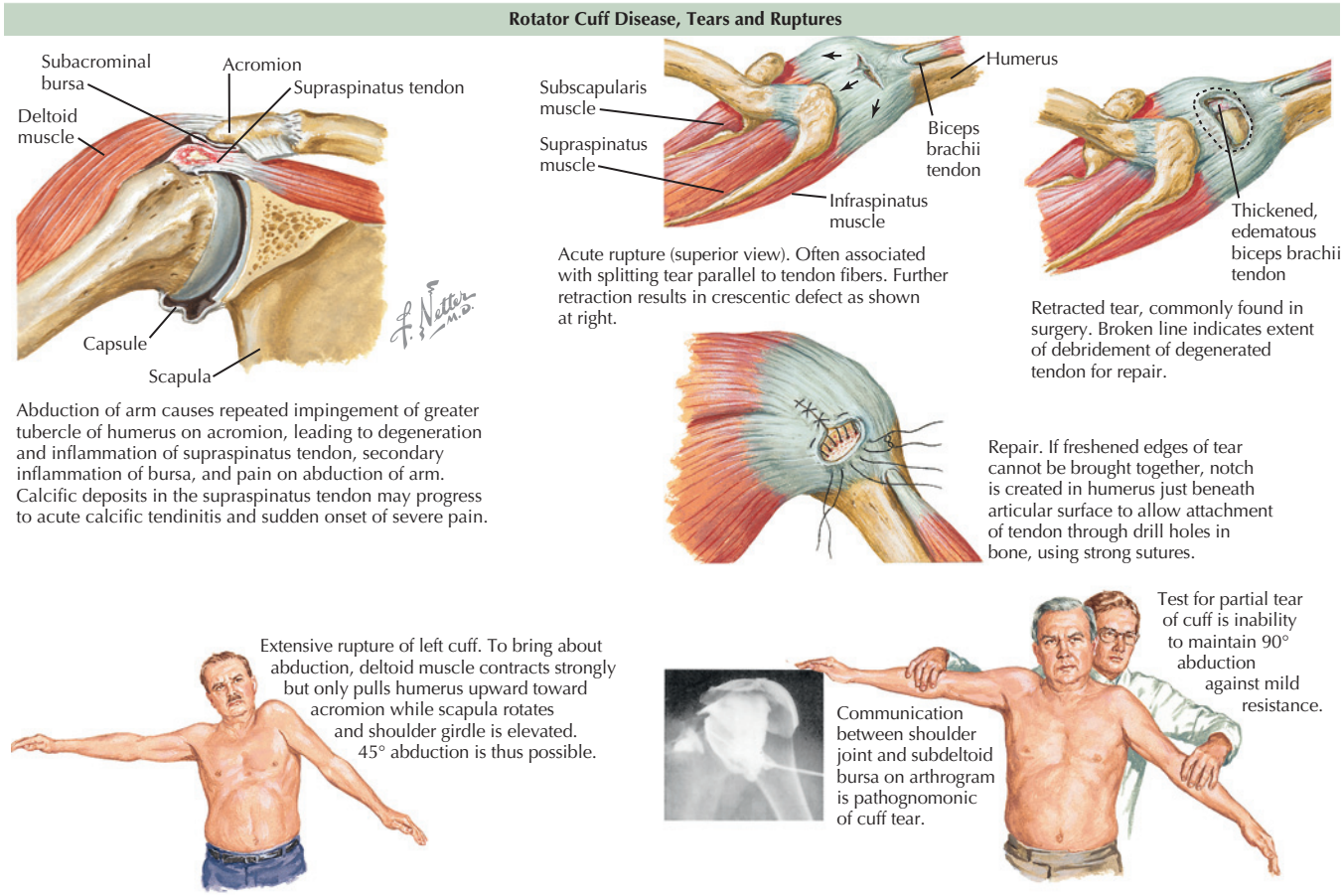


Figure 69-2 Rotator Cuff Injuries. Having the racket hand high above shoulder level, such as to serve or hit an overhead, puts the subacromial space and rotator cuff at risk for impingement.

Strengthen weak muscles; external rotators tend to be weaker than internal rotators. Increased rotator cuff strength is associated with increased velocity of serve. Additionally, strengthen latissimus dorsi, serratus anterior for all strokes, deltoid for overhead and backhand strokes, and rhomboids to counter drooping of the shoulder girdle. Maintaining flexibility of the posterior shoulder is important in prevention of injury.

Biceps tendinitis: Overuse injury, leading to pain in the front of the shoulder with activities that flex the arm at the elbow, rotate the forearm, and accelerate/decelerate the arm, such as hitting a high topspin forehand. Activity modifications, anti-inflammatories with eventual strengthening of the muscles of the shoulder, elbow, and scapula are recommended to prevent persistent tendonitis or tendon rupture. Need to rule out SLAP lesions.

SLAP (superior labrum, anterior to posterior) lesions: Two theories on etiology: First, tightness of the posterior inferior capsule leads to peeling off of the posterior superior labrum when the arm is abducted 90 degrees and externally rotated. Second, tension placed on the biceps tendon with repeated internal and external rotation of the shoulder leads to avulsion of the labrum from the superior glenoid.

Osteoarthritis (OA): In a recent study of senior elite tennis players, there was a statistically significant increase in mild osteoarthritis of the glenohumeral joint in the player's dominant shoulder. Increased instability as the result of high demands placed on the shoulder during tennis play may be the underlying cause for the development of OA.

Elbow

Overview: Biceps/triceps strengthening with improve control over the elbow, helping to reduce injury risk. Full pronation of the arm after impact will reduce excessive load and stress on the elbow.

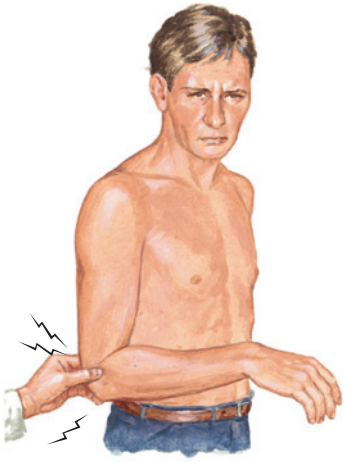
Lateral tennis elbow: 50% of recreational players sustain injury to the origin of the extensor carpi radialis brevis (ECRB) tendon (and sometimes the extensor digitorum communis [EDC]) at the lateral epicondyle (Fig. 69-3). Microtears in the tendon lead to the formation of granulation tissue and adhesions, which cause pain. Risk factors include age above 30, improper grip size, tight strings, use of a metal racket, incorrect technique, inadequate conditioning, practice more than 2 hours daily. Improper technique: wrist-flexed backhand, premature trunk rotation, leading with the elbow during the backhand. Correction of technique in addition to rest, rehabilitation, bracing, anti-inflammatories is recommended.

Medial tennis elbow: Less common than lateral tennis elbow in the general tennis playing population, but more common than lateral in professional tennis players (see Fig. 69-3). Usually caused by strain at the origin of the common flexor tendon at the medial epicondyle. Competitive players are prone to this injury from repeated wrist flexion from overhead serves or from pronation stress associated with placing topspin on the ball. Degenerative changes can be seen in FCR, PL, FDS, and FCU.

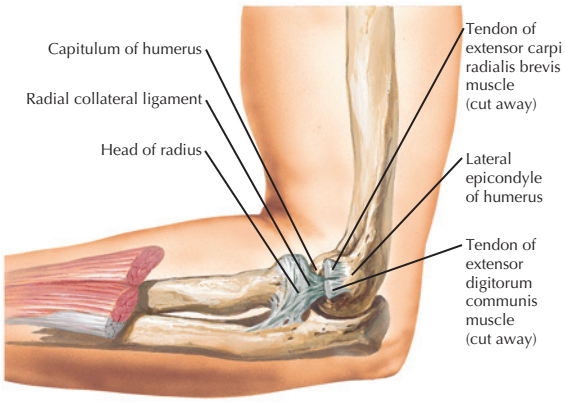
Medial and lateral elbow tendinosis: Respond more to physical rehabilitation though injections may help by reducing the pain while rehabilitating. Suspect radial nerve entrapment in a player with refractory tennis elbow.

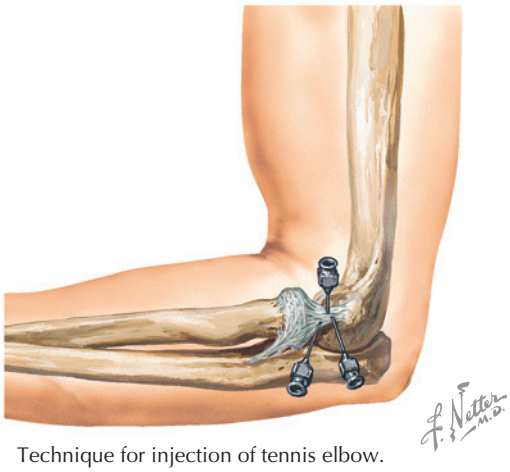
Posterior impingement syndrome of the elbow: Impaction of olecranon into the olecranon fossa of the humerus with repetitive, forceful extension of the elbow. Pain and tenderness occur about the elbow with repeated boney/soft tissue impingement. Activity modification to avoid leaning on the elbow and hitting the ball with the arm in full extension (e.g., Western forehand with fully extended arm) is recommended. Players should strengthen the biceps, triceps, and wrist flexors/extensors.

Ulnar collateral ligament (UCL) sprain: Over time, repetitive valgus stress to the elbow may stretch the UCL. This may lead to increased contact and cartilage wear on the lateral side of the elbow (between the radial head and the capitellum) and to a stretch injury of the ulnar nerve. A ruptured UCL is rare and would require surgery. Players may avoid this injury by using proper technique. Avoid hitting the Western forehand with the arm fully extended. Exercises to strengthen the biceps, triceps, wrist extensors/flexors are recommended.



Epicondylitis (tennis elbow). Exquisite tenderness over lateral or medial epicondyle of humerus.





Technique for injection of tennis elbow.

Figure 69-3 Tennis Elbow. A degenerative process of the elbow. Lateral epicondylitis is seen in recreational tennis players who are aged 30 to 50, play three to five times a week, and more often have a one-handed backhand with poor mechanics. Racket factors have also been implicated. Medial epicondylitis is seen more commonly than tennis elbow in the high-level tennis player. Factors associated with this overuse injury include hitting a lot of topspin on the forehand and the wrist snap in serving.

Wrist/Hand

De Quervain's stenosing tenosynovitis: Irritation to the abductor pollicis brevis and extensor pollicis brevis rubbing over the radial styloid with excessive ulnar deviation of the hand during grasping and swing of the racket (Fig. 69-4).

Triangular fibrocartilage complex (TFCC) tears: Painful or painless clicking noted on the ulnar side of the wrist due to tear in the TFCC. Increased association with ulnar positive variance (increased length of the ulna relative to the radius). Prevent by decreasing twisting motion at the wrist, functional bracing, and wrist strengthening exercises.

Hamate fracture: Rare injury caused by the impaction of the butt end of the racket handle into the hook of the hamate (Fig. 69-5). May require excision of the fracture fragment.

Wrist ganglion cysts: May cause pain predominately in the dorsum of the wrist. Treated with drainage, compression, anti-inflammatories, splinting, and surgically. These may recur.

Extensor carpi radialis brevis/longus, flexor carpi ulnaris tendonitis: Result from poor form, overstretching of the muscles during serve, and eccentric muscle contraction to stabilize wrist during off-center shots.

Wrist sprains: Acute overstretching or tearing of one or more ligaments of the wrist as the result of an unexpected twist, bend, or impact to the wrist. Taping, bracing, splinting of the wrist should be done until the player is completely pain free.

Recurrent dislocating extensor carpi ulnaris: Tears or stretching of the extensor retinaculum allow for subluxation of the tendon in and out of its normal groove. This commonly occurs when players hit a forehand shot with the wrist in ulnar deviation and supination imparting slice to the ball. Symptoms include painful snapping sensation over the dorsum of the wrist with rotation of the forearm and wrist. Treatment includes immobilization of the wrist for 6 weeks in supination. If nonoperative treatment fails, surgery may be necessary to repair the retinaculum. Injury prevention techniques include strengthening exercises of the wrist extensors and application of support braces, tape, or elastic bandages.

Neurovascular injury: May include ulnar nerve (cubital tunnel syndrome or entrapment at Guyon's canal), median nerve (pronator teres syndrome, carpal tunnel syndrome), radial nerve entrapment (radial tunnel syndrome), suprascapular nerve injury, or injury to palmar arteries.

Lower Extremities

Muscle strains: Partial muscle tears or pulls affecting the quadriceps, hamstrings, adductors, gastrocnemius, and soleus are common. Injury most commonly occurs at the muscle-tendon (myotendinous) junction. Muscles that span two joints are more susceptible to injury (hamstrings, gastrocnemius, and quadriceps).

Labral injuries: External rotation and hyperextension of the hip has been suggested to cause tearing of the anterior acetabular labrum (Fig. 69-6). Increased prevalence of these injuries occur in players that hit with an open stance stroke. Additionally, labral tears occur more commonly in players with hip dysplasia and femoroacetabular impingement. To confirm a diagnosis, magnetic resonance imaging (MRI) arthrogram (with Marcaine injection) is the diagnostic tool of choice. Surgical treatment via hip arthroscopy is advocated for treatment of labral injuries (see Fig. 69-6).

Knee injuries: Common in tennis due to the side-to-side, pivoting, twisting, jumping, bending, rapid acceleration and deceleration nature of the game. Most common knee injuries include patellofemoral syndrome, patellar tendonitis, meniscal injuries, and bursitis. In a survey of the U.S. Tennis Association (USTA) national team, 19% of all injuries were knee related. Of these, 70% were traumatic and 30% were from overuse. An uncommon injury that is seen more commonly in tennis is popliteus tendonitis.

Tennis leg: Incomplete/complete rupture of the medial head of the gastrocnemius. Injury is often incurred with a forceful contraction of the gastrocnemius when the knee is extended and the foot is dorsiflexed. Players aged 35 to 50 are at increased risk for this injury.

Medial tibial stress syndrome (shin splints): Periostitis along the posterior medial border of the distal one-third of the tibia from repetitive shock to the lower extremity. Muscles that normally absorb the shock in the leg fatigue, transferring the energy to the adjacent periosteum/bone. Rest, arch supports, taping, and anti-inflammatories may alleviate symptoms.

Achilles tendon rupture: More commonly seen in the player over age 40 and is associated with movements that require a quick burst of speed. Incidence is roughly 5.5%.

Ankle sprains: The most common acute injury in tennis and account for 20% to 25% of all injuries. Inversion injuries predominate. Proper footwear, taping, bracing may serve to prevent injury.

Plantar fascitis: An overuse injury that occurs due to repetitive forefoot push-offs during volleys. Treatment includes activity modification, nonsteroidal anti-inflammatories (NSAIDs), and evaluation of heel and medial arch support (Fig. 69-7). Change in footwear or use of an insert may be required.

Posterior tibial tendinitis: Pain is noted over the medial aspect of the ankle. Aggravated by jumping and quick starts. Prevention with use of a medial heel wedge, arch supports, and stretching.

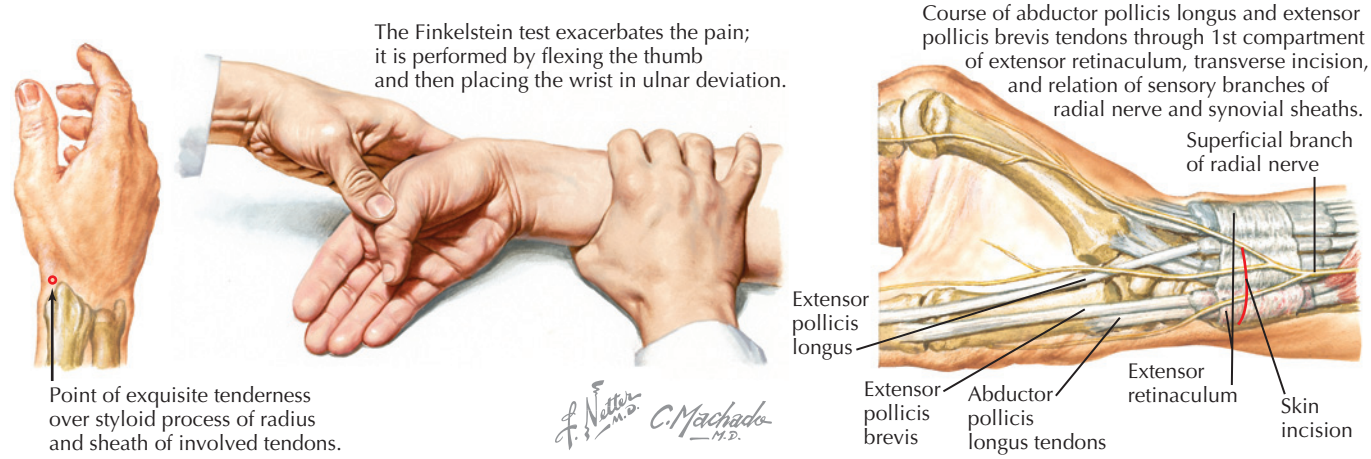


Figure 69-4 De Quervain's Tenosynovitis. Common in tennis because of the motions required to hit a ball with some spin as well as the grasping necessary to hold the racket.

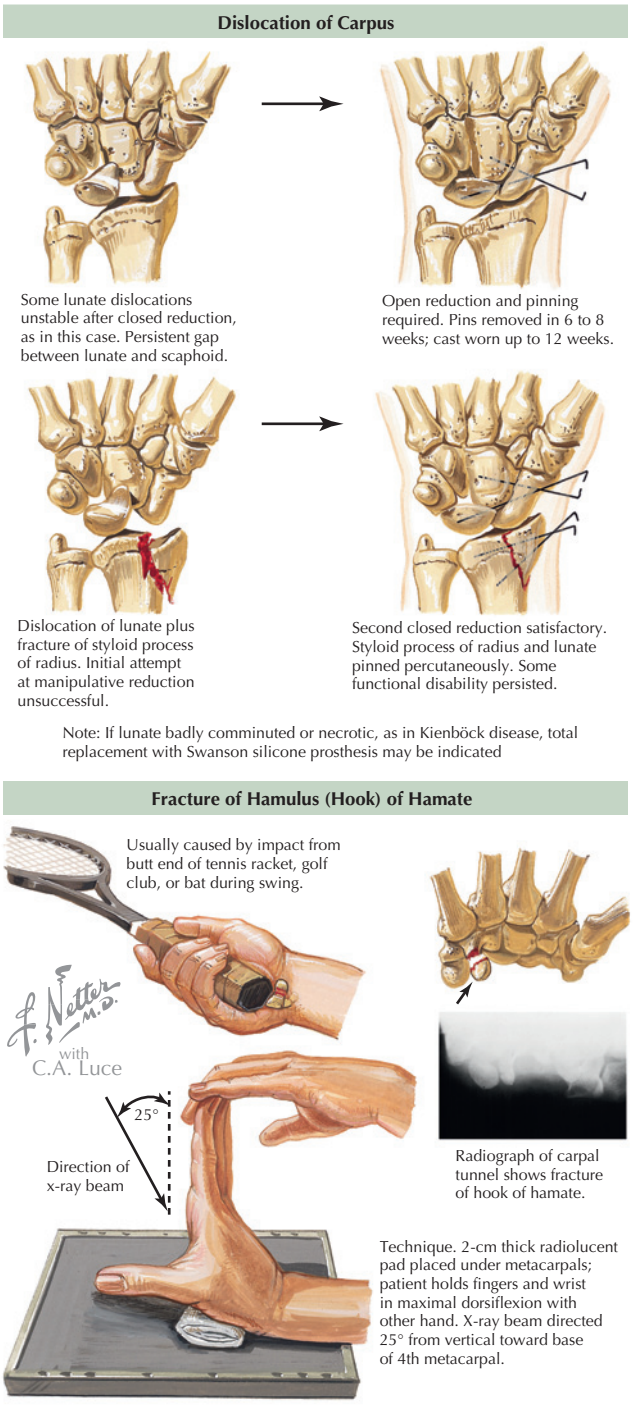
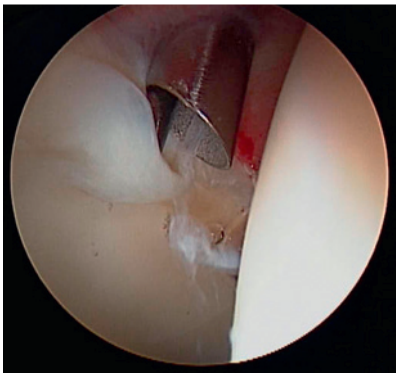


Figure 69-5 Wrist Injuries.



Hip lateral tears are becoming more common due to the open stance used in hitting the ball. The external rotation and extension of the hip result in increased stresses to the labrum.

Figure 69-6 Arthroscopic Picture of Labral Tear.

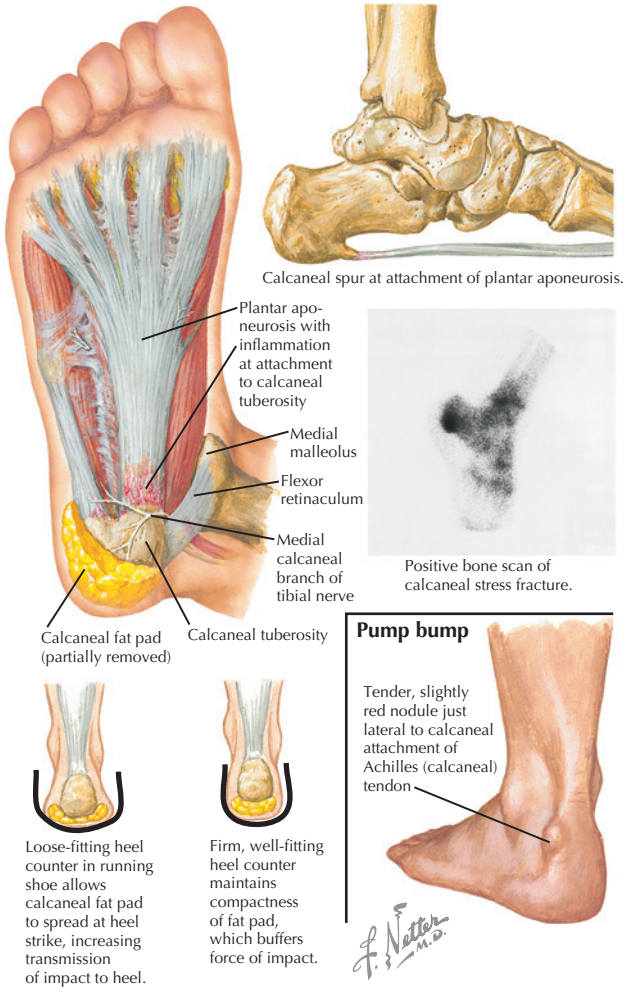


Figure 69-7 Plantar Fasciitis Associated with tightness of the calf muscles, and being on the toes for much of play; the plantar fascia may become inflamed, degenerative, and even tear.

Peroneal tendinitis: Frequently encountered in tennis players due to pivoting and rapid change in direction. Players should use tennis shoes appropriate for the surface of the court. Stabilize the ankle with braces, elastic bandaging, tape, or high-top athletic shoes. Strengthen peroneal muscles by eversion against resistance. Perform proprioceptive training.

Tennis toe: Repetitive abutment of the toes against the shoe may lead to a subungal hematoma. Players should wear appropriately sized shoes and keep their toenails cut short.

The Trunk

Low back pain (LBP): Common in tennis players of all ages: 38% of professional, 47% of elite junior female, and 31% of elite junior male tennis players have missed a tournament secondary to LBP.

Lumbar strain: Onset of symptoms often correlated to a change in intensity/duration of play. Repetitive trunk rotation and hyperextension place the erector spinae and multifidius muscles at risk for injury. When the player serves the ball, tossing the ball slightly ahead of the service line and use of the lower extremities to launch into the service will decrease the amount of hyperextension of the back and neck.

Herniated disc: Repetitive hyperextension and rotational forces applied to the lumbar spine, especially during the serve, subject the annulus to microtrauma that could lead to a tear. Correction of form and biomechanics is important to prevent a recurrence in injury.

Facet syndrome: Repetitive hyperextension, rotation of the trunk, or a combination of both may lead to cartilage wear of the facet joints. Additional load may be placed on the facet when there is loss of disc height, as seen with degeneration of vertebral discs. Players should avoid hyperextension of the lumbar spine to prevent acute facet impingement and chronic facet arthropathy.

Rib stress fracture: Repetitive contraction of muscles that have origins or insertions onto the ribs place excessive stress on the ribs and may lead to a stress fracture. Ribs susceptible to stress fracture in the tennis player include the first and fourth through ninth ribs, with the fourth and sixth most commonly affected. This is due to the action of the serratus anterior and external obliques. Players will note pain with deep inspiration, coughing, and overhead activities. Rib fractures are commonly missed on plain x-ray. Bone scans or MRI will make the diagnosis. Rib fractures heal with time, 4 to 6 weeks. Activity modification during this time is recommended.

Spondylolysis: A result of repetitive hyperextension of the lumbar spine. Prevention is similar to that for lumbar strains, as discussed earlier.

Abdominal muscle strains: Partial muscle tear or pull of the abdominal musculature (Fig. 69-8). Nondominant rectus abdominis is predominately affected. The internal and external oblique muscles are also prone to strains (see Fig. 69-8). During the cocking phase of the serve, the player is hyperextending the back, placing the abdominal musculature on greatest stretch. In the acceleration phase that ensues, the abdominal musculature is forcefully contracted—placing the muscles at risk for injury. Treatment is ice and rest. Avoid massage over the affected area to minimize risk of the development of myositis ossificans.

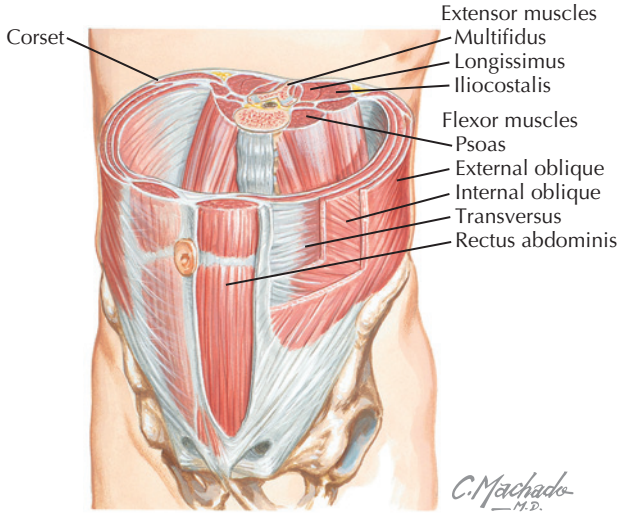


Figure 69-8 Muscles of Anterior Abdominal Wall. Abdominal muscle injuries are common in tennis as a result of the stretching and then contracting of the muscles to hit the serve and the increased use of the trunk muscles to generate force with the open stance ground strokes common in today's style of play.

Injuries Common in the Adolescent Population

- Muscle strains from overuse predominate the injuries in the young player, though the physis is the weakest link and uniquely susceptible in the skeletally immature player.
- Injuries to the lower extremity are twice as common as injuries to the spine and upper extremity.
- Injuries to the foot, leg, and wrist prevail in the female adolescent player, whereas injuries to the ankle, groin, hand, abdomen, and back prevail in the male adolescent player.
- Overall predominance of injury pattern, in order of greatest to least: strains, inflammation, sprain.

Physeal Injuries

Wrist epiphysitis: Repeated hyperextension and rotation of the wrist, causing inflammation of the distal radius epiphysis. This is commonly seen in adolescent players who attempt to put topspin on the ball. Premature closure of the growth plate is a potential complication of this process. Treatment strategies range from activity modification, wrist immobilization, to surgery for treatment of an associated fracture or for premature physeal closure. Players with wrist epiphysitis should avoid push-ups and flatten strokes, avoiding topspin.

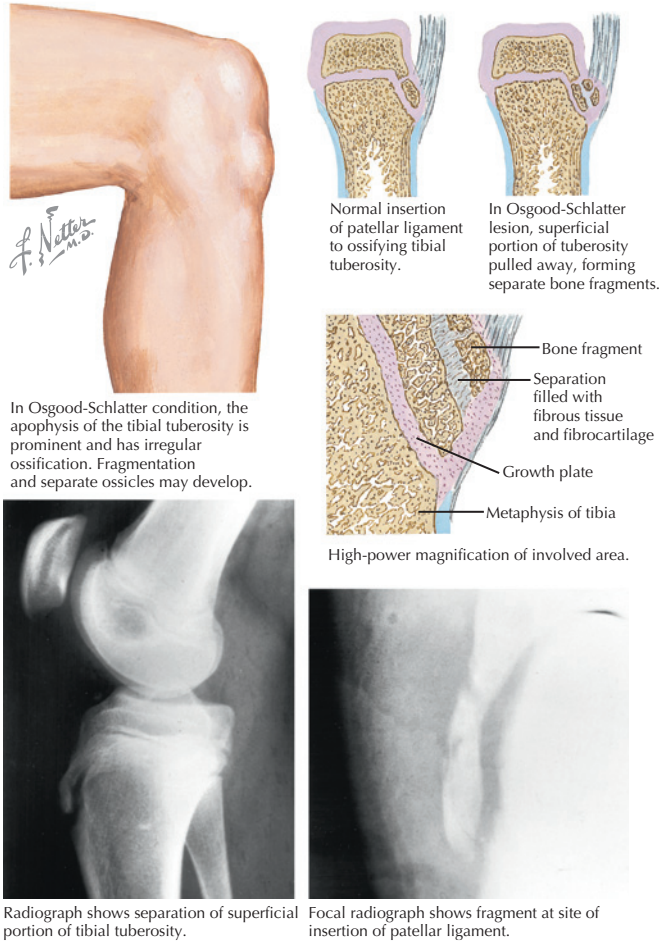


Figure 69-9 Osgood-Schlatter Lesion. Patellofemoral problems are common in tennis as a result of the repeated bending of the knees (ready position, serving, hitting low balls), lunging for the ball, and sudden stops and starts. In the young player, Osgood-Schlatter disease, inflammation of the growth plate where the patellar tendon inserts, is common, whereas in adults, patellar tendonitis or patellofemoral overload may occur.

Traction to the apophysis at the greater/lesser tuberosity of the humerus: Akin to Little Leaguer's shoulder. Rest and activity modification are the mainstays of treatment. Upon return to play, the player should start with ground strokes only. High volleys and serves should be gradually incorporated.

Humeral medial epicondyle apophysitis (adolescent medial tennis elbow): Overuse injury resulting from the repetitive muscular contractions of the forearm and wrist flexors during forehands and serves. Players may sense a decrease in their ability to serve at full speed and to fully straighten the elbow. Use of a racket with vibration-dampening characteristics, an oversized/light/stiff head, flexible shaft, a large cushioned grip (that is comfortable to the player), and low-tensioned strings of gut or high-quality synthetic strings are recommended. Activity modification with limitations on the intensity of conditioning/play and amount of serving/overhead play/throwing/heavy lifting is encouraged.

Osgood-Schlatter disease: Footwear modification for increased shock absorption and stability, stretching of the quadriceps and hamstring musculature to decrease the tension of the muscles pulling on the patellar tendon, training on soft surfaces (clay or sandy surfaces), and use of a patellar tendon strap (Fig. 69-9).

Sever's disease: Most common cause of heel pain in the adolescent player. Prevention and treatment entail proper stretching, use of a heel support that provides cushioning, shock absorption, and decreased tension on the Achilles tendon.

SUMMARY

- Tennis is a popular sport—worldwide, both genders, all ages.
- Tennis requires both aerobic and anaerobic fitness.
- Tennis may result in a variety of unique injuries.
- Lower extremity and back injuries predominate, though shoulder and elbow injuries do occur commonly.

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Alpine Skiing

70

Matthew R. Gammons

GENERAL PRINCIPALS

Background

- Alpine skiing is an immensely popular sport worldwide, with upward of 200 million participants per year.
- Alpine skiing involves high speeds, variable terrain, and weather conditions that combined with equipment can create significant opportunity for injury.
- Equipment changes have changed the nature of injuries but overall rates have not significantly decreased in the last 10 to 15 years.
- Lower extremity injuries are the most commonly reported but upper extremity injuries are frequent as well.
- Head injuries and chest wall/abdominal trauma are also of great concern because these injuries can be life threatening.
- Medical issues include cold exposure, sun exposure, altitude issues, and general travel-related problems.

Levels of Competition

- Alpine ski racing at its highest level is governed by Fédération Internationale de Ski (FIS).
- The U.S. Ski Association is the governing body in the United States.
- The U.S. national team is divided into four groups, A to D. Elite skiers are in the A team, with athletes ranked according to skill into the other divisions, down to the development team, or D team.
- Junior levels are divided by age groups (Table 70-1).
- There is often overlap between collegiate levels, high school levels, and junior race clubs.
- The highest level athletes generally compete internationally.
- Levels J3 to J1 may compete locally, regionally, and nationally. J4 and younger usually compete locally and occasionally regionally.
- There are a variety of master's race associations and recreational skiers can compete in NASTAR (National Standard Race). NASTAR racing is a program in which recreational skiers of all ages and abilities can test their skills on courses set up at resorts across the country. Times and scores are compared under a universal handicapping system similar to that used in golf. Amateur skiers can test their skills at more than 100 ski resorts in 26 states and Canada.

Events

- Events are divided into speed and technical categories.
- Speed events include the downhill and the super giant slalom (Super G).
- Technical events include the giant slalom and slalom.

Table 70-1 RACING AGE GROUPS

Level	Age Group (years)
J6	7 and below
J5	8-10
J4	11-12
J3	13-14
J2	15-16
J1	17-19

Speed Events

- Downhill is the fastest of the events, with speeds reaching 90 mph.
- Courses are hard and fast and skiers generally try to remain in the tuck position as much as possible.
- Skiers are allowed practice runs and course inspections.
- Super G combines downhill with giant slalom.
- Gates in Super G races are farther apart than the giant slalom and speeds are slightly less than downhill.
- Full speed practice runs are not allowed and only a small amount of inspection time is permitted; this requires the skiers to constantly adjust to the terrain.

Technical Events

- Giant slalom involves a course between Super G and slalom.
- The race requires more technical turns than Super G.
- Racers spend little time in the tuck position.
- Slalom is the most technical of the events; involves short arc turns and contact with the poles marking the course.
- Skiers are never in a tuck position and are constantly shifting from edge to edge.

Combined Events

- Combined races usually involve one downhill run and two slalom runs; occasionally it may combine a single slalom with a downhill or Super G race.

Event Coverage

- As with all sporting event coverage, planning and communication are the keys.
- Physician must be proficient in alpine skiing because courses are often steep and icy.
- Physicians should communicate with ski patrol prior to the event or training to learn established protocol and establish a chain of command in the event of an injury.
- Many ski areas are a significant distance from medical or trauma facilities; this combined with winter weather often necessitates that stabilizing care, pain medication, compression for bleeding, etc., be provided while transport is arranged.
- Physicians should generally station themselves high up on the course so they have access to the entire run.
- Radios allow for communication between the medical team and spotters on the course; these should be tested ahead of the training or race because most two-way radios are site-to-site and may have trouble transmitting in some areas.
- Cell phone service is also inconsistent in many mountainous areas and should be investigated prior to competition if the medical team is planning on using this mode of communication.
- Most injuries are best taken care of off the mountain; so if possible the majority of the care should be delayed until the athlete can be transported to lodge facilities.

Epidemiology

- Although rates of skiing injuries have dropped in the last 3 decades most of this drop occurred in the late 1970s and early 1980s.
- Rates have been generally stable over the past 20 years.
- Reported injury rates vary from 2 to 5 per 1000 skier days.
- Ratio of lower extremity to upper extremity injury is about 2:1.

- Highest prevalence of lower extremity injuries has changed from ankle/tibia to knee; this is likely due to equipment changes including stiffer boot materials and the advent of release bindings.
- Rates of injury and severe injury are similar with snowboarding, although snowboarders have a higher rate of upper extremity injuries.
- Catastrophic injuries are rare: 0.01 per 1000 skier days.

Equipment

- Major equipment changes occurred in the late 1970s and early 1980s; this included a move from leather to plastic boots and releasable bindings.
- A common misconception is that modern equipment protects the knees when it is in fact designed to prevent ankle and tibia fractures. Although this appears to have been effective in reducing the number of injuries, it has caused a dramatic increase in knee injuries.
- Standard binding release is based on the skier characteristics such as skill level, boot size, height, and weight; most commonly adjusted based on the Deutches Institu für Normung (DIN) standard.
- Binding mechanism may not release at slow fall speeds because torque requirement is not met.
- Expert skiers and racers will often ski at DIN settings higher than recommended to prevent prerelease.
- Lower extremity injuries, excluding knee sprains, are often associated with improper equipment adjustment.
- Parabolic or shaped skis have become the mainstay in all disciplines except downhill. These skies are shorter than the tradition ski and have increased side-cut. It is unclear what if any effect these skies have had on injuries.

Training and Physiology

- As with most sports, skiing requires a good base of lower extremity and core strengthening and aerobic fitness to tolerate training loads.
- Large eccentric and isometric loads are placed on the lower extremity so strength training should focus on these areas.
- The majority of a ski race event takes place at the anaerobic threshold so cardiovascular training regimens should focus on increasing this tolerance.
- The competitive season of elite skiers involves altitude and significant travel; because of this training should be adjusted frequently to maximize performance while minimizing the risk of overtraining the athlete.

SPECIFIC INJURIES

Lower Extremity

Knee Ligament Injuries (See also Chapter 49, Knee Injuries)

- Not unique to skiing but the mechanism of injury differs because of the ski/binding/boot interface (Fig. 70-1).
- Injuries to the medial collateral ligament (MCL) are the most commonly reported but anterior cruciate ligament (ACL) injuries have risen dramatically and now account for almost 20% of all skiing injuries.
- Injuries among elite skiers are reported at 8.5 per 100 skier seasons.
- Combination injuries (ACL/MCL) are relatively common as well.
- There is an increased risk of ACL injury in female skiers compared to male although the effect is not as dramatic as is seen in field sports.
- It is important to note that there appears to be no relationship between knee sprains and binding dysfunction.
- There are three commonly described mechanisms for knee ligament injuries in skiing (see Fig. 70-1).
  - Valgus external rotation mechanism of injury occurs when ski edge catches, forcing the leg into abduction and external rotation. This is the most common mechanism for medial collateral ligament injuries.
  - The boot-induced anterior drawer mechanism occurs when the rear of the ski contacts the snow first after jump. The ski then levers the boot forward, creating the anterior drawer movement.
  - The most common mechanism for injury of the ACL appears to be the phantom foot. This occurs when the skier's weight is posterior, and the skier's hips drop below the level of the flexed knees. Weight is on the downhill ski and, as the skier attempts to recover, increased force to the downhill ski results in greater edge pressure. This causes an abrupt internal rotation force on the downhill knee.
- Prevention is focused on education, avoidance of high-risk positions such as the described phantom foot, and equipment adjustment. It is unclear what effect these interventions may have on elite skiers.

Fractures

- Although fractures of the lower extremity have decreased, there are still two fractures commonly seen in skiers: spiral and tibial plateau.
- Spiral fractures of the tibia are common in children but are also seen in adults. These are caused by external rotation forces on the ski and have been associated with binding dysfunction (see Fig. 70-1).
- Incidence of tibial plateau fractures, most commonly of the lateral plateau, has risen along with knee ligament injuries.
- The increase of these fractures along with knee injuries likely represents a similar mechanism of injury—valgus/external rotation—as for knee ligament injuries.

Upper Extremity

- The shoulder, hand, and wrist make up the majority of upper extremity injuries.
- The shoulder accounts for the largest overall number of upper extremity injuries in skiing. The most common shoulder injuries are listed in Table 70-2.
- Skier's thumb, also known as gamekeeper's thumb, is the most common single injury of the upper extremity, accounting for 8% of all injuries.
- Sprains of the ulnar collateral ligament (UCL) occur with a fall onto an outstretched hand with a pole in the palm (see Fig. 70-1).
  - This causes a radial stress on the ligament.
  - Treatment involves protection and occasionally surgical fixation.
  - The risk of UCL injuries may be reduced by having poles with breakaway straps or using a pole without straps.
  - A modified thumb spica splint molded to a ski pole can allow continued skiing participation (see Fig. 70-1).

Trauma

Head Injuries (See also Chapter 39, Head Injuries)

- Account for a small portion of total ski injuries but account for the majority of severe injuries.
- Concussions are relatively common, particularly in the adolescent age group.
- Management of head injuries does not differ from other sports.
- Helmets are mandatory in most competitions but it is unclear what role helmets play in the prevention of these injuries.
- Several studies have shown a decrease in the risk of head injuries in helmet wearers.

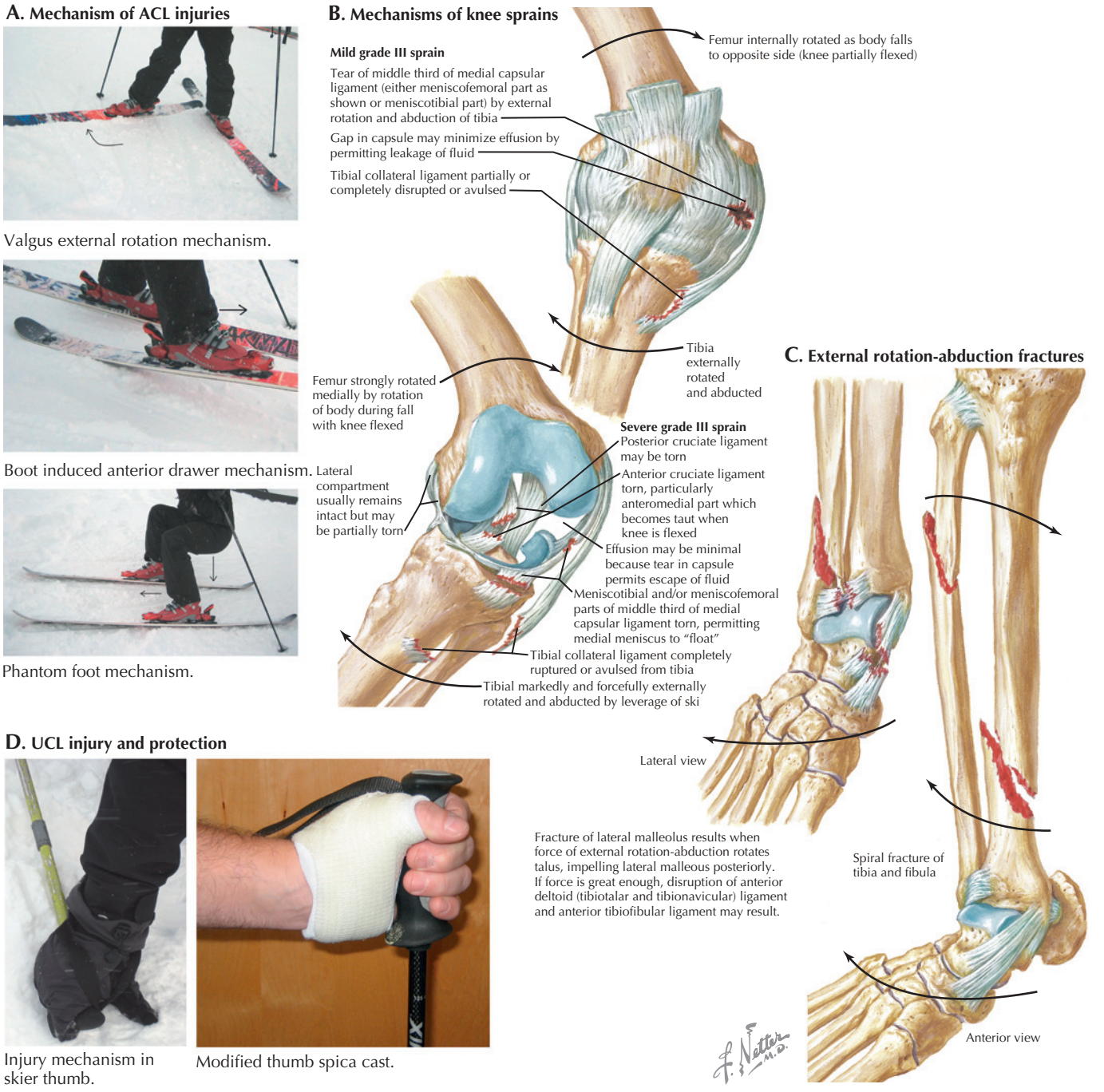

Figure 70-1 Skiing Injuries.

Table 70-2 COMMON SHOULDER INJURIES IN SKIING

Rotator cuff injuries	24%
Anterior dislocation/subluxation	22%
Acromioclavicular separations	20%
Clavicle fractures	11%

Data from Koehle MS, Lloyd-Smith R, Taunton JE: Alpine skiing injuries and their prevention. Sports Med 32:785-793, 2002.

- The topic of efficacy of helmets in preventing head injury is difficult to study due to many confounding variables, including rates of helmet use, terrain, weather, conditions, skill level, etc.
- These studies therefore have methodological flaws making it difficult to assess what overall protective effect helmets may have in skiing.

Spinal Injuries

- Severe spinal injuries are rare in alpine skiing: 0.001 to 0.004 per 1000 skier days.
- Rates in snowboarding are higher than skiing and the most common spinal injury in both is a wedge compression fracture.
- Treatment of suspected spinal injuries can be difficult slopeside.
- Ski patrol members are usually the best personnel to accomplish boarding and spine stabilization on the mountain.

Thoracoabdominal Trauma

- Minor chest wall, back, and abdominal trauma is relatively common in skiing and is generally not life threatening.
- Because of the high speeds involved, however, physicians should always be aware of the possibility of severe trauma, particularly in cases of collisions.
- Severe injuries include aortic rupture and intra-abdominal organ trauma.

Medical Problems

- Medical problems inherent to skiing include cold-related injuries and altitude illness (see Chapter 18, Exercise in the Cold and Cold Injuries).
- Because most athletes and coaches have experience with these issues they are often not a significant problem.
- Elite athletes will often log many thousands of miles during the year for both competitions and training camps.
- International travel is common and general precautions should be taken to prevent travel-related illness such as traveler's diarrhea.
- Jet lag and fatigue can also be significant problems and are best managed on a case-by-case basis.
- Because of the cold weather climates, skiers often overlook the potential for sun-related skin problems.
- A combination of altitude and reflection off the snow can increase ultraviolet (UV) exposure to extreme levels.
- Education of both coaches and athletes on sun protection is important to help prevent acute sun exposure injuries and the potential for skin cancer.

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Cross-Country Skiing

David D. Cosca and William G. Callahan

GENERAL PRINCIPLES

- Cross-country, or nordic, skiing is a multifaceted sport that can be pursued either as a simple recreational outdoor activity or as a vigorous competitive endurance sport.
- Cross-country skiing serves as an excellent means to develop and maintain cardiovascular fitness; most large muscle groups of the upper and lower body are used, in a smooth, rhythmic, low-impact manner.
- Injury rates are typically lower than those seen in alpine skiing and running.
- Very popular in Scandinavian countries, and is moderately popular in the northeastern and western mountain regions of the United States.

CROSS-COUNTRY SKIING VARIATIONS

Trail/Track Skiing

- Skiers use machine-groomed trails.
- Trails are compacted and rolled with heavy sleds pulled by Sno-Cats or snowmobiles and imprinted with a set of ski tracks and an 8-by-10 foot wide lane for ski skating.
- Groomed trails are standard at cross-country ski resorts, but many communities also set trails in the winter on snow-covered golf courses or bike trails for local citizen use, and some are lighted for night skiing.
- Trail skiing is suitable for a wide range of individuals, young and old, regardless of prior experience. Novices simply "ski-walk," employing a shuffling-like technique on the snow. More experienced skiers use the tracks with a diagonal stride (classic) technique, or the groomed lanes with a ski-skate (freestyle) technique.
- Competitive ski racing and training requires machine-groomed trails.

Backcountry Skiing/Ski Touring

- No groomed trail is required.
- Participants choose their own route (i.e., bushwhacking). Some enthusiasts pursue a midday trek in the woods and a picnic lunch, while others plan a multi-day snow camping tour, and still others seek out backcountry slopes to climb and then descend utilizing a telemark turn technique.
- A telemark turn is one in which the skier thrusts the outside (or downhill) ski forward, assuming a lunge position with the front knee at near 90 degrees of flexion and the back knee in a kneeling position. The turn is carved while holding this position.
- Backcountry skiers typically choose skis with metal edges and relatively heavy, supportive boots.

Chair Lift–Facilitated Telemark Skiing

- Could be considered a variant of alpine skiing. Participants opt for a chair lift at a ski area to repeatedly transport them to the top of a slope, then descend while making telemark turns (in contrast to the parallel turns of alpine skiers).
- Skiers use stiff plastic boots, wide metal-edged skis with significant side cut, and strong three-pin or riveted toe cable bindings that still allow the heel to lift freely off of the ski. Bindings are not designed to release during a fall.
- Skilled telemark skiers often reach speeds similar to alpine skiers, and are subject to similar injuries.

COMPETITIVE CROSS-COUNTRY SKIING

Race Events

- There has been a recent trend toward using audience-friendly formats such as mass sprint, relay, and pursuit (a race that involves switching skis and styles midway through the race).
- Venues for major ski races (e.g., the 2002 Winter Olympic venue at Soldier Hollow, Utah) have been designed to allow for better spectator viewing, with large sections of the course visible from the stands.
- World Cup and Olympic race events include (distance format for women/men respectively): 1 km sprint, 2 × 1 km team sprint, 10 km/15 km individual start, 15 km/30 km pursuit, 30 km/50 km mass start, and 4 × 5 km/4 × 10 km relay.
- Citizen race distances vary from 5 km to 55 km. Some races are specifically designated as classical technique only, whereas others use a freestyle format.
- The largest event in the United States is the American Birkebeiner in northern Wisconsin (52 km, with more than 7000 participants).

Equipment

Diagonal Stride (Classic)

Skis: Double camber, with central area for kick wax (waxless skis have a "fish scale" pattern imprinted on the ski base). When the skier's weight is evenly distributed between skis, the central kick zone should not contact the snow, allowing for maximal glide. When the ski is aggressively weighted, the kick zone is engaged in the snow, allowing for a push-off, or "kick." Skis are typically 20 to 25 cm greater than the skier's height, but the skier's weight should also be considered when choosing ski length and ski flex.

Boots: Lightweight, relatively low cut, flexible sole.

Bindings: Currently, there are two predominant systems, NNN (New Nordic Norm) and SNS (Salomon Nordic System). Both are lightweight and engage the ski boot at the toe only, allowing the heel to lift freely off of the ski while striding. The two systems differ primarily by the ridges on the binding plate that fit into corresponding slots on the boot's sole.

Poles: Carbon fiber (preferred by elite skiers) or aluminum, with variable grip and strap systems. Pole length extends to a height between the skier's armpit and top of shoulder.

Ski Skating (Freestyle)

Skis: Typically 10 to 15 cm shorter than classic skis, with more torsional rigidity to accommodate the forces generated during skate push-off. Ski design continues to evolve, particularly in relation to side cut and ski tip shape. Racing ski bases have specified "grinds" to enhance glide wax absorption and maximize glide when matched to specific snow conditions.

Boots: Skate boots are cut higher, with a stiffer upper boot that is hinged at the ankle, and provide more lateral support than classic boots. Skate boots also have a more rigid sole.

Bindings: Similar to classic bindings, but with a more rigid toe plate. The popular SNS Pilot system has a second attachment point to the boot under the toe that operates as a spring-loaded hinged plate and provides slightly more control over the ski.

Poles: Carbon fiber (best) or aluminum, with variable grip and strap systems, slightly longer than classic poles. Typical height of a skate pole is skier's midchin.

Ski Base Preparation

Ski glide: Considerable effort is directed at maximizing ski glide during cross-country ski competitions. Most elite skiers carry several different pairs of skis with variable base compositions and stone ground patterns, or "grinds," each best suited to a particular snow condition. A "rill" pattern may also be pressed onto the ski base to facilitate channeling of melting snow and reduce friction on particularly warm and humid days.

Glide wax: Typically applied to the entire length of the base of skate skis, and to the tips and tails of classic skis. Layers of wax are melted in with an iron, scraped nearly clean between layers, and then the final layer is brushed and polished after it is ironed and scraped. Modern, high-performance ski waxes contain a variable percentage of fluorocarbon, generally using a higher percentage for warmer, higher humidity conditions. 100% fluorocarbon wax costs up to $120 retail for a 30-gram container (enough for 10 wax preparations), yet this wax is often used as the final layer on a fully prepped race ski, even at the citizen race level.

Kick wax: Applied to the center section of classic skis to provide grip for forward propulsion. Waxes are specifically formulated for different temperature ranges—harder for colder snow, softer for warmer snow—and can be rubbed on like a crayon, then melted with an iron. Some conditions (icy, or warm and wet) call for Klister wax—a sticky, gluelike paste squeezed from a tube.

Clothing

Fabric and fit: Cross-country skiing is a highly aerobic sport. Race participants typically wear light, form-fitting, synthetic clothing, although newer merino wool and silk alternatives are available.

Layers: Layered clothing is a key strategy to avoid either overheating or excessive cooling that might otherwise be caused by variations in exertional effort or variations in weather, particularly during training or casual skiing.

CROSS-COUNTRY SKI TECHNIQUES

- Ski racers use both classic and skating techniques in competitions (Fig. 71-1).
- Both require a dynamic body position, with forward lean and flexion at the waist.
- Both techniques rely on aggressive poling to generate forward propulsion, and therefore both require good upper body and core strength.
- The technique of ski skating has evolved during the past 2 decades, with the result that skate skiing technique can be up to 25% faster than classical technique under some conditions.

Classic Technique

Diagonal stride (classic): The skier keeps both skis in the groomed track, pointing straight ahead, and obtains thrust by weighting a ski, engaging the kick wax (or fish scales) for grip, and pushing off from the kick zone onto the other ski as it glides forward (see Fig. 71-1). The pole is planted on the opposite side of the kick ski, similar to the arm swing during running, and the body assumes a "diagonal" position relative to the snow.

Double pole: Both poles are planted simultaneously, while the skier vigorously flexes at the waist, leaving elbows bent, and thrusts downward and backward onto the poles as the skis glide forward in the track (see Fig. 71-1).

Double pole with kick: Both poles are planted simultaneously as described above, but a single leg kick is made as the poles swing forward (see Fig. 71-1).

Ski Skating Technique

Description: Skate skiing requires the skier to push one ski outward with the ski slightly angled, applying force to the inside edge much like an ice skater. Complete weight transfer onto the gliding ski is essential for fast and efficient skiing. Ski skating has different techniques for different terrain, much like changing gears on a bicycle.

V1 skate: The technique is asymmetrical, with poling accompanying skating on one side but not the other. Double pole with arms in slightly offset position, planted simultaneously with every other step onto the glide ski, pole thrust timed with skating push off (see Fig. 71-1). Typically used for hill climbing, where poling contributes more than half of propulsive force. Requires good timing, cadence, and body position.

V2 skate: A symmetrical technique with a double pole on every skating push-off, timed just prior to the step onto the gliding ski. Used on flats or gentle uphill (see Fig. 71-1). Requires complete weight transfer, good balance, upper body strength, and aerobic fitness.

V2 alternate skate: An asymmetrical technique with a double pole on every other skating push-off. Timing similar to V2 skate, as opposed to V1 skate. Used on flats or downhill at relatively high speed.

Open field skate: Skate without poles. Used on downhill at higher speeds.

TRAINING FOR CROSS-COUNTRY SKI RACING

Aerobic Capacity of Cross-Country Ski Racers

- Elite level cross-country ski racers are among the most aerobically fit athletes in the world.

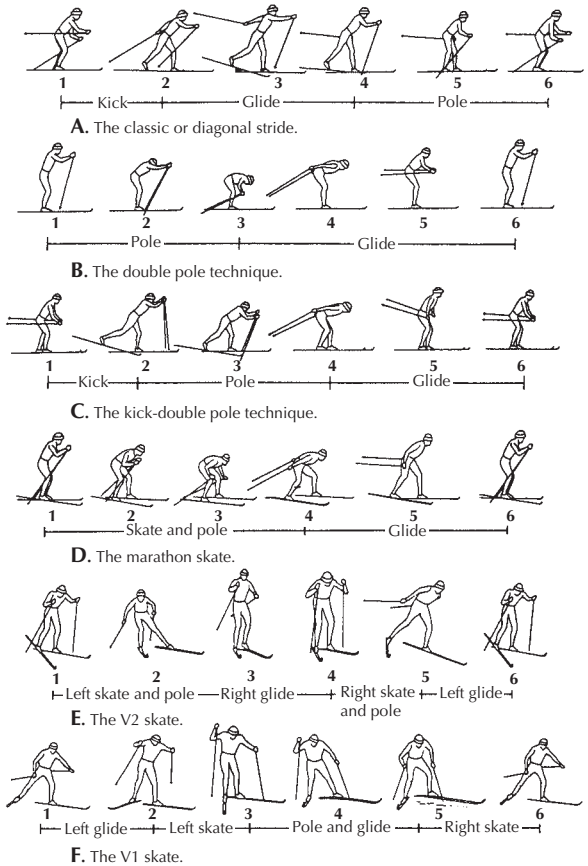


Figure 71-1 Cross-Country Skiing Techniques. *(Reprinted with permission from Elmquist LG, Johnson R, Kaplan MJ, Renstrom PA: Alpine skiing. In Fu FH, Stone DA (eds): Sports Injuries. Baltimore: Lippincott, Williams, & Wilkins, 1994, pp 481-500.)*

- Mean $\dot{V}O_2$ max levels of male Finnish and Swedish national skiers during the 1990s was 85 to 90 mL/kg per minute, with the gold medalists in the low 90 mL/kg per minute range.
- Elite Swedish women had mean $\dot{V}O_2$ max levels in the mid 70 mL/kg per minute range.
- Elite-level masters citizen ski racers in the United States in the 50- to 60-year-old age group have been shown to have $\dot{V}O_2$ max levels in the low to mid 60 mL/kg per minute range for men, and low to mid 50 mL/kg per minute range for women (unpublished data from the University of California–Davis Sports Performance Lab).

Endurance Training

- Exercise training, in season and out, must be specifically targeted to maximize both aerobic fitness and skiing efficiency (technique) in order to improve race performance. Roller skiing is the most sport-specific off-season training activity, although cycling, ski bounding, and uphill ski walking are also good cross-training modalities.
- Most elite coaches recommend that 70% to 85% of the total training volume be dedicated to a mix of long distance, low-intensity training and medium-intensity training at levels below the respiratory compensation threshold (variably referred to as "maximal lactate steady state" or "anaerobic threshold"). The remaining training volume should consist of a progressive incorporation of fast distance training at intensities near race pace (slightly above threshold, and with close attention to good technique), and short bouts of high-intensity training to improve $\dot{V}O_2$ max.
- Cardiopulmonary exercise testing on a treadmill using expiratory gas measurements can be used to measure aerobic fitness ($\dot{V}O_2$ max) and ventilatory thresholds, and provide specific heart rate training zones. Lactate threshold testing can be performed either on roller skis or on the snow, and can also provide accurate training heart rate zones.
- Ski racing does require anaerobic efforts during sustained hill climbs and end-of-race sprints, and therefore skiers should include training to improve this capability.
- Finally, it is widely recognized that both upper body strength and core strength are key contributors to ski race performance, and sufficient effort should be directed at improving these parameters in the off season. Core strengthening may also reduce the incidence of back pain, which is a relatively common complaint among ski racers.

Blood Doping

- Three medal winners in the 2002 Winter Olympic cross-country skiing events tested positive for darbepoetin.
- Skiing is an endurance sport, much like cycling, where the benefits of an elevated erythrocyte mass brought on by use of EPO or autologous blood transfusion can result in a significant competitive advantage.
- There is enormous pressure at the elite level to perform well.
- Recent evidence suggests that blood doping is both prevalent and effective in cross-country ski racing, and that current testing protocols may be ineffective.

ENVIRONMENTAL CONCERNS

- Terrain for cross-country skiing ranges from well-groomed flatland skiing in open fields to backcountry alpine wilderness, where hills, obstacles, and uneven snow surfaces factor into play.
- Participants are subject to the effects of altitude, humidity, sun exposure, temperature, and wind chill, each of which can vary during any particular outing.
- In some backcountry areas, avalanche is a concern.
- Skier awareness to snow conditions, weather, and potential hazards are foremost in the avoidance of environment-related health hazards.

Sun Injury

Description: Up to 85% of ultraviolet (UV) waves reflect off the snow surface, which magnifies the effect of sun on exposed areas of skin and unprotected eyes. Perspiration may decrease the efficacy of sunblock lotions, though "sweat-proof" preparations are available and highly recommended.

Cold Injury

Frostnip: Freezing of superficial layers of skin causing burning pain and erythema that is completely reversed with rewarming.

Frostbite: Ranges from first-degree injury to epidermis only and no resultant tissue loss, to deep penetrating fourth-degree wounds involving subcutaneous tissues, muscle, and tendon.

Corneal freezing: Affected by wind chill, ground speed, temperature, and humidity. Use of goggles or sunglasses is recommended, though evidence for primary prevention is lacking.

Hypothermia: Cooling of core temperature with resultant physiologic changes ranging from shivering, tachypnea and poor judgment to stupor, bradycardia, and death.

Risk factors: Cold injury risks inherent to skiing include exposed or inadequately protected skin, wet or improper fitting clothing, altitude, humidity, and wind chill. Injury of this nature is more common in the recreational or inexperienced skier. Prevention strategies include wearing appropriately sized clothing in removable layers; using a base layer garment designed to "wick" moisture away from skin is recommended. Synthetic fabrics such as polypropylene or naturals such as wool or silk are suitable options for next-to-skin wear. Proper nutrition and avoidance of alcohol and dehydration are highly recommended. (See Chapter 18, Exercise in the Cold and Cold Injuries.)

Altitude

Acute Mountain Illness (AMS)

- Estimated to occur in approximately 20% to 25 % of individuals with recent ascent above 2500 meters (8000 feet), but is relatively infrequent in nordic skiing, given that most ski areas are at lower elevations.
- Acute mountain illness (AMS) may develop in skiers at the lower elevations encountered at ski areas or race events, particularly if the skiers live at low elevation and travel quickly to higher elevations.
- Symptoms in these individuals are usually mild and self-limited. Altitude illness is less common in elite athletes due to experience, acclimatization, and training.
- Backcountry skiers, particularly those who enjoy mountaineering, are at higher risk of more severe AMS.
- Only a fraction of participants (0.01% to 0.05%) develop high-altitude cerebral edema (HACE) or experience high-altitude pulmonary edema (HAPE). Severe symptoms are best treated with rapid descent. See Chapter 19, High-Altitude Training and Competition, for details on diagnosis, treatment, and prevention of altitude illness.

EPIDEMIOLOGY OF INJURY AND ILLNESS

- Frequency difficult to measure. Skiers may not report minor injuries or illness, and some cross-country skiers seek out remote locations that may lead to a delay or even failure to present for medical care.
- One prospective case study reported an injury rate of 0.72 per 1000 skier days in cross-country skiers compared to an injury rate of 3.4 to 7.4 per 1000 skier days in alpine counterparts.
- Other studies report an incidence of nordic ski injury ranging from 0.49 to 5.63 per 1000 skier days.
- Most injuries occur during descents.
- Injuries and illness occurring in competitive skiers are common, yet often minor in nature.
- Complaints addressed during or after competition include muscle fatigue and soreness, tendonitis, and minor ligament injuries.

- Medical complaints such as dehydration, cold injury, gastrointestinal problems, and bronchospasm are also common.
- Exercise-induced bronchospasm is estimated to occur in more than 30% of participants.
- Cross-country skiing is a winter sport that occurs when the risk of viral upper respiratory illness is at its highest. The intense nature of endurance training may also lead to a diminished immune response.
- A common cold may strike just prior to a highly anticipated race event, negating months of training, and result in a disappointing race result.
- During race season, skiers should use all commonsense strategies to minimize risk of cold and flu: good hand washing, avoidance of close contact with infectious individuals, a flu shot, and optimization of immune function with adequate sleep and good nutrition.
- If ill, skiers should reduce their training intensity appropriately.

MUSCULOSKELETAL INJURY

- Lower extremity injuries are consistently reported as more common than upper extremity injuries (approximately 55% vs. 35%).
- Injury type and relative frequency as described in a published meta-analysis are sprains (40%), fractures (27%), contusions (16%), lacerations (9%), dislocations (6%), other (1%).
- Injury frequency and type are significantly influenced by skier experience and terrain conditions.
- Competitive athletes tend toward overuse injuries whereas less experienced skiers are more subject to traumatic injuries.
- Impact injuries from falls are most common and are predisposed by icy surfaces and obstacles buried in snow.

OVERUSE INJURIES

Upper extremity: Common injuries to all skiing styles include de Quervain's tendonitis; wrist extensor tenosynovitis; rotator cuff, bicipital, and triceps tendonitis; and medial and lateral epicondylitis. The longer poles used in **skating** predispose the skier to **triceps tendonitis.**

Lower extremity: Strains to the hip adductors, internal rotators, and flexors are common, with **adductor strain** being particularly predisposed by **skating** technique. Injuries common to running sports are similarly found in nordic athletes and include patellofemoral pain, patellar tendonitis, medial tibial stress syndrome, stress fracture, Achilles tendonitis, plantar fasciitis and rupture, and sesamoiditis. **Skier's toe,** or hallux rigidus, causes by repeated dorsiflexion of the first metatarsophalangeal (MTP) joint is common enough to have earned its own moniker, and appears to be more common with skate skiing (Fig. 71-2). Physical findings include tenderness over the dorsal joint surface, limited dorsiflexion, pain on passive plantar flexion, and possible degenerative changes and dorsal spurs on radiographs.

Trunk and spine: Low back pain is a common complaint in competitive skiers, likely related to the aggressive forward flexed skiing posture. Studies report variable frequencies between classical and skating techniques, with strongly conflicting data. Improper technique, inexperience, and training errors are thought to be contributing factors regardless of skiing style.

TRAUMATIC INJURIES

Upper extremity: Mechanism for distal upper extremity injuries is most often a fall onto outstretched hand (FOOSH) or direct trauma and can result in ulnar collateral ligament sprain of the metacarpophalangeal (MCP) joint of the thumb **(skier's thumb),** or distal radius, carpal metacarpal, and phalanx fractures (Fig. 71-3). Proximal upper extremity injuries, often caused by direct shoulder trauma, include fracture of the clavicle, shoulder subluxation and dislocation, and AC joint sprain or separation.

Lower extremity: Lower extremity traumatic injuries mimic those found in most high-velocity sports and risk is confounded by bulky equipment that may get caught in snow or obstacles thereby acting as a lever and magnifying forces. Examples include femur fractures, knee sprains (medial collateral ligament

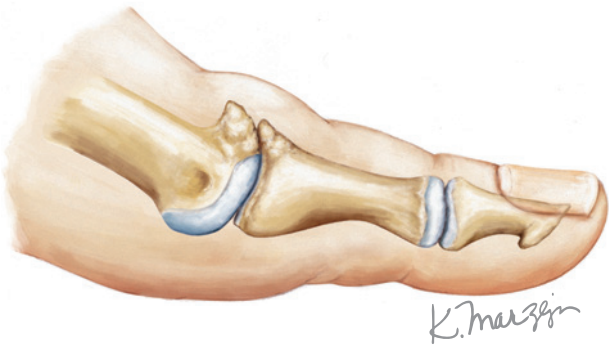
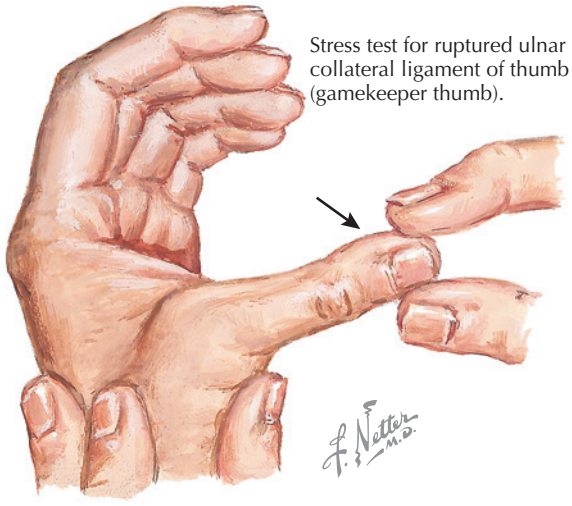
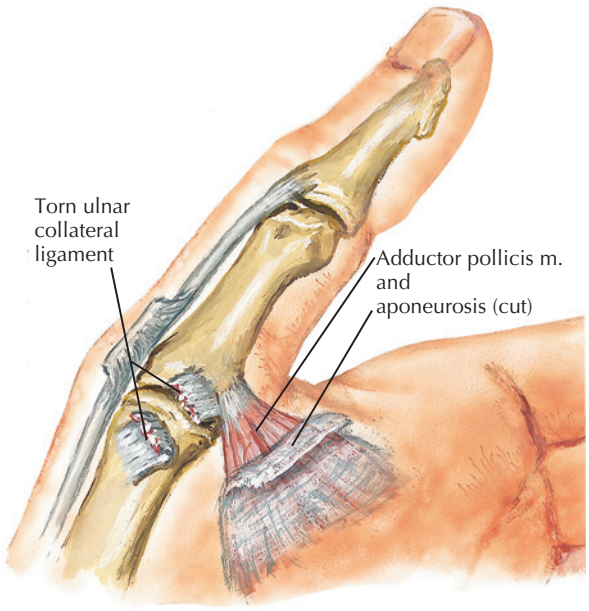


Figure 71-2 Skier's Toe.





Ruptured ulnar collateral ligament of metacarpophalangeal joint of thumb.

Figure 71-3 Skier's Thumb.

[MCL] and anterior cruciate ligament [ACL]), meniscal tear, patellar dislocation, tibial and fibular fractures, ankle sprains, ankle fractures, calf strain, and plantar fascia rupture.

Trunk and head: Soft tissue trauma may cause contusions and lacerations. Direct blows to the head can result in mild traumatic brain injury (concussion). Data that support helmet use in snow sports is growing, but current studies focus on alpine skiing and snowboarding. One report suggests a reduction in head injuries in helmeted alpine skiers when traveling at lower velocities, and might thereby infer protection for nordic skiers, particularly backcountry skiers and telemark skiers accessing ski lifts.

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Snowboarding

72

Thomas R. Sachtleben

GENERAL PRINCIPLES

Overview

- Snowboarding was developed in the 1970s and popularized in the 1980s.
- Snowboarding is one of the fastest growing sports worldwide, and there are more than 6 million riders in the United States.
- Snowboarders range in age from 4 to seniors.
- Typical riders are males in their 20s.
- The majority of snowboarders ride on slopes shared with skiers at winter resorts, but terrain parks, half-pipes, and backcountry use have seen immense popularity.
- Snowboarding became an Olympic sport in 1998.

Equipment

- Basic equipment consists of a snowboard, bindings, and snowboard boots (Fig. 72-1).
- Snowboard bindings and boots are used to fix the feet to the board and to transfer energy forces to the board. Bindings usually have straps that hold the boot on the board, or are the "step-in" variety similar to ski bindings.
- Snowboards are made of fiberglass with a wood or foam core.
- Boots can be "soft," "hard," or a "hybrid" variety. Most riders wear soft boots, which are more comfortable and allow for increased movement. The inner bladder is surrounded by a firm upper which allows flexibility for ankle movement. Hybrid boots have hard soles and are used with step-in bindings. These provide a balance between support and flexibility. Hard boots have a hard plastic shell similar to ski boots and are designed for increased control and for precision of movements.
- Additional safety equipment includes helmets, wrist guards, and goggles, as well as hip and knee pads.

Figure 72-1 Snowboarding Equipment.

Events

- Alpine-style races (parallel slalom, giant slalom).
- Half-pipe/superpipe.
- Snowboarder-cross—multiple riders race simultaneously through a course of ramps and jumps.
- Big-air events—riders jump for maximum height with aerial maneuvers.
- Slopestyle—snowboarders race through an obstacle course full of rails and tables.

Biomechanical Principles

- Feet are planted nearly perpendicular to the long axis of the board and direction of movement. This prevents the board from acting independently as a lever and applying torque on the knee, as occurs in skiing. Because the feet remain fixed to the board during falls, the upper extremities usually absorb maximal forces from impact with the snow surface.
- Snowboards are also generally shorter than skis, and fewer torsional injuries are seen due to lower forces generated by a shorter lever arm.
- Snowboarders do not have poles to help break their falls. Backward falls resulting in occipital and sacral trauma are common because of the "opposite-edge phenomenon." When the opposite edge of the board on which the snowboarder is riding comes into contact with the snow, it can cause powerful angular acceleration of the board and thus causes a forceful fall backward.
- Riders move with one shoulder and leg leading the way down the slope. This creates a partial blind side, increasing risk of collision. Approximately 75% of lower extremity injuries involve the lead foot.

Injury Patterns

- Overall injury rate is approximately 4 per 1000 snowboarder days.
- The overall rate of injury in snowboarding has diminished in recent years, partly because of advancements in boot and binding systems. Head and spinal injuries have increased, however, and several injury patterns have emerged with increased popularity of the sport.
- Falls are the most common mechanism of injury among snowboarders, followed by jumps/landings and collisions. Collisions by snowboarders with stationary objects or other skiers/snowboarders account for only about 10% of injuries; 4% to 8% of snowboarding injuries involve chairlifts.
- Most snowboarders wear flexible boots, which provide less support than ski boots. This makes snowboarders more susceptible to ankle injuries.
- Compared with skiing, there is a higher proportion of upper extremity injuries, especially in children. Approximately two-thirds of all snowboarding injuries involve the upper extremity.
- Wrist injuries are most common, because of frequent falls backward onto an outstretched arm and hyperextended wrist.
- Head injury rates are three times higher in snowboarders than in skiers. Head and spinal injuries are common and are related to the popularity of aerial acrobatics and jumping.

- Snowboarders are two and a half times more likely to sustain a fracture than skiers.

Risk Factors

- Snowboarding has a fast learning curve, which predisposes beginners, especially first-day riders, to injury. It is estimated that approximately half of all injuries occur in beginner snowboarders, and that more than 50% of injured beginners have never had formal instruction.
- Intermediate and advanced riders are often injured when riding in terrain parks or the half-pipe. Fast skill advancement and progression to aerials and jumping put many riders at risk for serious injury.
- Children are at high risk of injury, and physeal injuries are common.
- Snowboarding often appeals to those seeking risky behavior. Recreational drug use, alcohol, and sleep deprivation, as well as a rider's perception of these risk factors, can contribute to injury.
- Few snowboarders use wrist guards or other protective devices, although helmet use is gaining popularity.
- Additional risk factors include riding on hard, icy, or slushy terrain as well as riding in the half-pipe.

INJURIES AND MEDICAL PROBLEMS

Upper Extremity Injuries

- Snowboarders reach out with their arms to aid in balance and to brace falls, thus making upper extremity injuries extremely common.
- The shoulder is vulnerable, especially in advanced riders (acromioclavicular joint separations, shoulder subluxations/dislocations, and clavicle fractures).
- Elbow fractures and dislocations occur frequently, particularly in children.
- Early detection of forearm intraosseous membrane injuries is essential due to poor outcomes with delayed diagnosis. Magnetic resonance imaging (MRI) and sonography are useful in proper assessment of this injury.

Wrist Injuries

- Wrist injuries occur frequently, especially in children and beginners; 20% of all snowboarding injuries involve the wrist, and about two-thirds of these are fractures. Wrist injuries are 10 times more common in snowboarders than in skiers.
- Distal radius fractures are particularly common. Approximately two-thirds of distal radius fractures seen in snowboarders are intra-articular or comminuted fractures requiring surgical intervention. Novice riders are more likely to sustain an extra-articular fracture, whereas more experienced riders often have intra-articular fractures.
- Carpal fractures, particularly those involving the scaphoid and lunate, should be suspected in patients with point tenderness over these bones.
- Wrists are often used for speed control as well as pivoting and trick maneuvers. The majority of wrist fractures sustained by novice riders are a result of falls, whereas most wrist fractures seen in advanced riders are caused by jumping.

Head Injuries

- Head injuries encountered in snowboarding include concussions, cerebral contusions, diffuse axonal injury, subdural (most common), epidural, and intracerebral hematomas.
- Traumatic brain injury is the leading cause of severe injury and death among snowboarders.
- Collisions with stationary objects (trees, lift poles, etc.) account for approximately half of head injuries.
- Snowboarders with head injuries should be appropriately evaluated for maxillofacial injuries.

Spinal Injuries

- Spinal injuries have increased in frequency, partly as a result of young, aggressive riders participating in half-pipe and "big-air" events.
- Most spinal injuries are seen in advanced riders, and are usually caused by falls or improper landings. Falls from jumps at high speeds translate tremendous forces on the axial skeleton.
- Backward falls often involve shearing forces from multiple vectors as well as axial loading.
- Spinal injuries constitute 2% to 4% of all snowboarding injuries, and are a major cause of permanent disability
- Most of the spinal injuries involve fractures of the transverse process of the vertebra. Compression and burst fractures are also common types of spinal fractures seen in snowboarders. Axial loads are transmitted to the spine due to landing aerials and jumps in a flexed position. Spinal cord injuries usually occur at the thoracolumbar junction, and involve an anterior fracture/dislocation.
- MRI helps identify occult fractures and to assess injuries affecting spinal cord and nerve roots.

Chest/Abdominal Injuries

- Snowboarders usually sustain injuries to the chest, including rib fractures and pneumothorax, as a result of falls while jumping.
- Blunt abdominal trauma, especially splenic injuries, are quite common in snowboarders.
- Splenic lacerations, hematomas, and ruptures are observed in riders, usually after falling from great heights while jumping. The typical mechanism of injury is the snowboarder's own flexed and adducted left elbow being thrust into his or her abdomen upon hitting the ground with an outstretched hand. Another defense mechanism seen in falling snowboarders is the "fetal tuck" posture (folded arms placed on the chest at time of impact), which has also been associated with splenic rupture from the left elbow.
- Other injuries seen include liver lacerations and renal contusions.
- Contrast-enhanced computed tomography (CT) is best for rapid and accurate diagnosis of intra-abdominal injuries, and a follow-up CT is important when clinical exams suggest possible delayed splenic rupture. These injuries can be life threatening, and plain films detect an ominous sign such as pneumopertioneum in only 30% of patients with visceral rupture. Ultrasound can be used when CT is not available or in patients who are hemodynamically unstable.

Lower Extremity Injuries

- Lower extremity injuries are becoming less common as boot/binding technology evolves.
- The incidence of hip dislocations in snowboarders is five times higher than in skiers.
- There are fewer knee injuries than in alpine skiing because of fewer torsional forces. The vast majority of binding systems are nonreleasable, thus preventing most rotatory injuries to the knee. Anterior cruciate ligament (ACL) injuries are seen infrequently compared with skiing, but when they do occur, they are primarily seen in advanced snowboarders riding in the terrain park or jumping. Novice riders are at risk for ACL injury while getting on and off lifts, since this action requires one leg free for self-propulsion.
- Elite snowboarders have increased rates of knee injuries because of the higher impact and torsional forces that are observed at high levels of competition.

- Ankle injuries comprise approximately 15% to 20% of all snowboarding injuries. Approximately half of all ankle injuries are fractures. A common fracture type involves the lateral process of the talus (15% of all ankle injuries sustained while snowboarding). This fracture is frequently seen in riders wearing soft boots that allow increased ankle flexibility.

Fracture of Lateral Process of Talus (LPT)

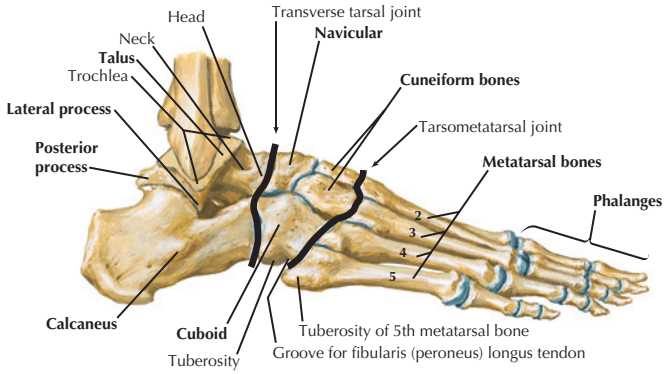
Description: This particular injury is often referred to as "snowboarder's ankle" or "snowboarder's fracture," and is relatively unique to snowboarding. This type of fracture occurs 15 times more frequently in snowboarders than in the general population. The lateral process of the talus is a large, wedge-shaped prominence that articulates with both the distal fibula and the posterior calcaneal facet (Fig. 72-2A). It is important for hinge and rotatory movements.

Mechanism of injury: Lateral process of talus (LPT) fractures usually occur as a result of sudden dorsiflexion and hindfoot inversion with axial loading, although external rotation is thought to be a key component. Axial loading in this position, along with shearing forces transferred from the calcaneus, often occurs with landing after jumps.

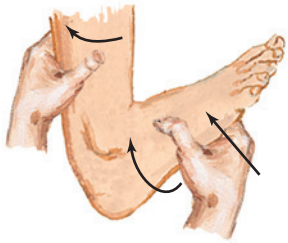
Presentation: LPT fractures present similarly to lateral ankle sprains, and thus are often missed on initial presentation. Persistent pain with weight bearing, nonresolving pain, and severely limited range of motion should invoke a high index of suspicion for talar injury.

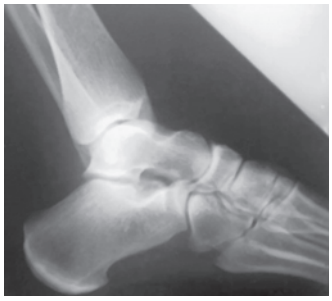
Examination: Swelling is seen anterolaterally, with maximal tenderness inferior to the tip of the lateral malleolus. Patients usually have significant pain with attempts at weight bearing. The LPT stress test has been described, which is performed by applying a dorsiflexed and everted stress to the foot while com-

A. Anatomical relationships of lateral process of talus.

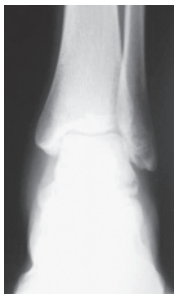


B. LPT stress test.

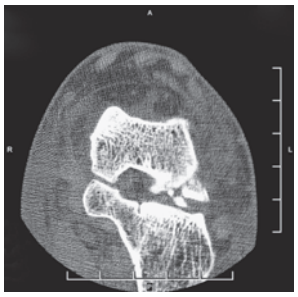




C. Radiograph of lateral process of talus fracture.

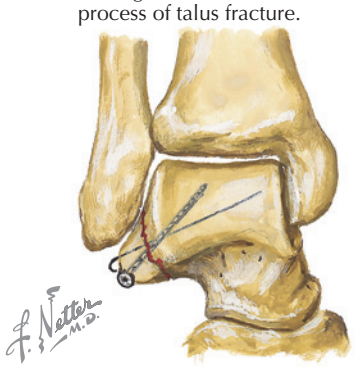


D. Radiograph of lateral process of talus fracture.



E. Computed tomography (CT) of lateral process of talus fracture.

F. Surgical treatment for lateral process of talus fracture.



Large fracture of lateral process fixed with Kirschner wire and lag screw.

G. LPT fracture treatment algorithm.

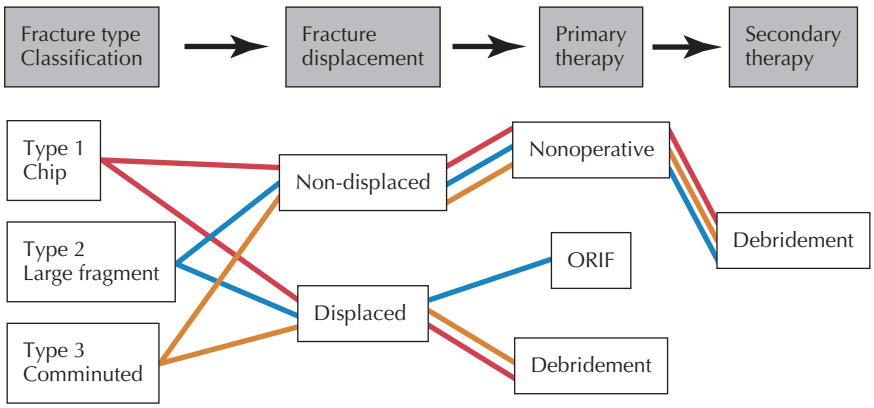


Figure 72-2 Fracture of Lateral Process of Talus. (***G,*** *Adapted from Valderrabano V, Perren T, Ryf C, et al: Snowboarder's talus fracture: Treatment outcome of 20 cases after 3.5 years. Am J Sports Med 33:871-880, 2005.)*

pressing the ankle in a cephalad direction and holding the lower leg in external rotation (see Fig. 72-2B). The LPT is compressed against the posterior calcaneal facet with this maneuver.

Differential diagnosis: Similar clinical presentations are seen with a lateral ligamentous sprain, subluxed peroneal tendon, lateral compartment syndrome, subtalar joint sprain, and Salter-Harris type 1 fracture of the lateral malleolus. Snowboarders are also at risk for malleolar bursitis and pseudotumor of the ankle, which can present with lateral ankle pain. Malleolar bursitis is a result of repetitive friction from stiff snowboard boots. Pseudotumors occur with compression of soft tissues between the lateral malleolus and snowboard boot.

Diagnostics: The best x-ray views are the mortise view with 10 to 20 degrees of inversion or the Broden view; however, this fracture is not seen well on standard radiographs (see Fig. 72-2C, D). CT is often necessary to delineate fracture patterns (see Fig. 72-2E). LPT fractures are classified into three widely recognized patterns: Type 1 fractures involve a small avulsion without extension to the talofibular articulation. Type 2 fractures involve a singe large fragment (extending from the talofibular joint to the subtalar joint), and type 3 fractures are comminuted.

Treatment: Maintaining proper anatomical alignment is paramount in managing LPT fractures. Type 1 fractures (and nondisplaced type 2 fractures, <2 mm) are generally treated nonoperatively with 4 to 6 weeks of non–weight-bearing cast immobilization if they are nondisplaced or extra-articular. After another 2 weeks of a weight-bearing cast boot, patients should begin ankle exercises to prevent stiff subtalar and tibiotalar joints. Type 2 fractures, especially if greater than 2 mm or displaced, are best treated with open reduction and internal fixation, because this approach has been shown to reduce long-term morbidities (see Fig. 72-2F). Excision of small, displaced fragments is usually necessary with type 3 fractures. Many authors recommend attempted closed reduction by an orthopedist of displaced type 2 and all type 3 fractures, with subsequent cast immobilization. If proper reduction is unsuccessful, secondary therapy with open reduction and internal fixation is generally required, followed by 4 to 6 weeks of non–weight-bearing (see Fig. 72-2G). LPT fractures have a high morbidity rate because of frequent delays in diagnosis. Unrecognized fractures can lead to nonunion, avascular necrosis, and early degeneration of the subtalar joint. Fortunately, early diagnosis and aggressive management typically results in good outcomes.

Other Risk and Prevention Measures

Environmental Risks

- Snowboarders are at risk for injury related to environmental factors because of physical exertion and prolonged exposure to winter elements.
- Appropriate prevention and protection is necessary to prevent dehydration, hypothermia, and frostbite.
- Protection with sunscreen is particularly important because of high-altitude ultraviolet light exposure.
- Altitude illness prevention and prompt recognition of symptoms is essential (see Chapter 18, Exercise in the Cold and Cold Injuries).

Backcountry

- Backcountry use by snowboarders has grown exponentially, and will continue to grow as the sport continues to gain popularity. Riders are increasingly seeking varied terrain and better snow conditions in unpatrolled areas.
- Avalanches claim the lives of many snowboarders each year, usually from suffocation and/or blunt trauma.
- Deep snow immersion can also cause asphyxiation. This occurs as a result of a fall into a deep snowbank or tree well. Tree wells are deep depressions of loose snow that form around the base of a tree with low branches. Snowboarders who fall headfirst into a tree well or deep snowbank can be easily trapped and suffocate in as little as 15 minutes. These tree-well deaths, also known as non–avalanche-related snow-immersion deaths (NARSIDS), have become an increasingly recognized fatality pattern in snowboarders. Many backcountry riders use strapless, releasable ("click-in") bindings, or hand-accessible release cords to both bindings in an effort to get off their board quickly in emergent situations.
- Skill, knowledge, good judgment, and appropriate safety gear are crucial for safe travel in the backcountry. Essential gear includes an avalanche transceiver, shovel, and probe, as well as proper avalanche training. Backcountry riders should never ride alone.

Injury Prevention

- Helmets should be strongly encouraged for all snowboarders, and they should fit properly. Children should not wear helmets that they will "grow in to" as they mature. The risk of head injury and injury severity is significantly reduced by helmet wear. There is not an increased risk of cervical spine injury in riders who wear helmets.
- Wrist guards are beneficial in preventing serious wrist injuries, especially in children and beginner riders. Unfortunately, fewer than 10% of riders wear them. There is nearly a three times higher risk of injury in those who do not wear wrist protection devices. Wrist guards that are made of high-density plastic are recommended, and some companies make gloves with built-in wrist protectors. They must be flexible enough to absorb as much energy as possible, because braces that are too rigid can generate forces at the proximal and distal ends of the brace, thus increasing fracture risk. Although there has been concern that the use of wrist protectors may transfer injury to other forearm locations, studies have not shown this to be true.
- Other types of protective equipment used by snowboarders include boots with stiff inner liners, elbow pads, knee pads, and pants with extra padding sewn in. Snowboarding equipment, including binding adjustments, should be properly maintained.

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Ice Hockey

Robert Johnson

HOCKEY ORGANIZATION AND PARTICIPATION

- **USA Hockey,** located in Colorado Springs, Colorado, is national governing body for ice hockey in the United States and official representative of U.S. Olympic Committee and International Ice Hockey Federation. It works with National Hockey League (NHL) and National Collegiate Athletic Association (NCAA).
- In 1968-1969, 3800 teams were registered with USA Hockey. In 1993-1994, 21,150 teams with approximately 340,000 players were registered. In 2005-2006, more than 442,000 players and more than 58,000 coaches were registered.
- Women's hockey is rapidly growing. Of 21,150 teams registered by USA Hockey, 352 represent about 6000 girls and women. In 1995-1996, 38 women's teams competed in interscholastic competition in Minnesota; 130 teams are expected to compete in 2007-2008. First state high school tournament for girls was held in Minnesota in 1995. In many states, girls are still competing on boys' youth hockey teams.
- The NCAA has 34 women's teams competing in Division I, 2 in Division II, and 45 in Division III. For men, the NCAA reports 59 teams competing in Division I, 7 in Division II, and 69 in Division III.
- **Age range of organized competition: 5 to over 50.**
- **Age group divisions** are determined by birth date as of August 31 of each year (Table 73-1).
- More than 545,000 are registered participants in Canada.
- **Regarded by most as fastest competitive team sport.**

GAME OF ICE HOCKEY

Structure

- **Professional, college, adult:** three 20-minute periods
- **High school:** three 17-minute periods
- **Youth:** three 12- to 15-minute periods

Team Composition

- Eighteen players and two goalkeepers (usual position distribution). Six players compete at one time: three forwards, two defensemen, one goalie.
- **Goalkeeper (goalie):** Player who tends goal to catch or deflect puck and prevent opponent from scoring.
- **Forwards (left wing, center, right wing):** Offensive-minded players who attack opponent with intent to score goal; also assist defensemen in protecting their goal.
- **Defensemen (left and right):** Primary responsibility is to protect their goal and goalie to prevent opponent from advancing to net to score.

Table 73-1 AGE GROUP DIVISIONS DETERMINED BY USA HOCKEY

Level	Boys (yr)	Girls (yr)
Mites	≤8	
Squirts	≤10	6-12
Pee Wees	≤12	13-15
Bantams	≤14	
Junior Midgets	≤19	16-19
Junior	≤19	
Senior	>19	>19

- **Substitution** may occur during play ("on the fly") or during time stoppages for violations, goals, or penalties.

Rink

- Should be 200 feet by 100 feet. Smallest recommended dimensions are 185 feet by 85 feet.
- Should be surrounded by wooden or fiberglass boards 40 to 48 inches high with yellow or light-colored kickplate at bottom. It is recommended that safety glass or other protective screen encircle rink.
- Goal should have dimensions 4 feet high by 6 feet wide with metal goalposts and crossbar and net surrounding metal framework.

Special Equipment

- **Puck** is vulcanized rubber 1 inch thick and 3 inches in diameter, weighing 5.5 to 6 ounces.
- **Hockey stick**
  - Forwards and defensemen: usually made of wood (shaft may be made of other materials) with shaft less than 60 inches, blade less than 12.5 inches long by 3 inches wide, and curve not to exceed 0.75 inch.
  - Goalie: wood shaft less than 60 inches with blade less than 15.5 inches long by 3.5 inches wide, and curve not to exceed 0.75 inch.

Skills

- **Skating** skill involves three factors: angle of propulsion (angle formed by skate blade in direction of skate), angle of forward inclination (body lean), length of stride.
- **Shooting**
  - **Types of shots: standing wrist shot** (sweeping action with stick terminating in wrist snap and follow-through); **skating wrist shot** (similar to standing wrist shot except player has forward momentum while skating; most accurate); **standing slap shot** (stick and blade are brought back variable distance, followed by vigorous forward motion, "slapping" at puck much like golf swing; least accurate); **skating slap shot** (greatest velocity).
  - **Maximal velocity** is result of strength of arm and shoulder muscles and full trunk rotation.
- **Passing**
- **Stick handling:** Ability to advance puck while maneuvering on ice.
- **Checking:** Intentional contact with opponent who is in possession of puck, using hip or shoulder. Player may check from side, diagonally or frontally, approaching with no more than two skating strides. American Academy of Pediatrics recommends checking at Bantam rather than PeeWee age group.
- **Goal tending:** Goalkeeper (goalie) tends goal, protected by special equipment and pads to catch or deflect pucks from goal.

Safety and Protection

Protective Equipment

- Goalie: helmet, mask, throat protector, chest protector, cup, thick padded shin guards, blocker (worn on one hand), trapper (device to catch puck worn on opposite hand), skates that are unique to protect goal and goalie.
- Forward and defense: helmet, shoulder pads, elbow pads, padded gloves, cup, breezers (padded hockey pants to protect sacrum, coccyx, and pelvis), shin guards, skates.

- **Face masks**
  - Full face masks required at youth and high school levels in 1975; Eastern Collegiate Athletic Conference mandated use in 1977; NCAA required use in 1980. Helmets required in NHL but face masks remain optional. This level of play accounts for most serious eye injuries.
  - **Effects of full and half face shields** (college level, Canada): full shield
    - Full shield: 61.6% had at least 1 injury.
    - Half shield: 63.2% had at least 1 injury.
    - Risk of facial, dental injury: 2.3 times greater with half shield.
    - Risk of concussion: Concussion rates are higher in those wearing the half shield compared to those wearing the full face mask.

Rules to Protect Players

- Penalties: 2-minute (minor), 5-minute (major), 10-minute (major), or combination.
  - Offending player must sit in designated penalty box and his or her team must play with one less player on ice ("shorthanded"). If two penalties are assessed against team, it must play two players short. Team never has to play more than two players short.
  - For 10-minute penalties, offending team does not have to play shorthanded. They lose services of that player for that time interval.
  - Single or multiple game disqualifications may be assessed, depending on severity of infraction.
- Goaltender protection: no unnecessary body contact with goalie; the "crease" is the goalie-protected area in front of goal where opposing players cannot enter without puck.
- Common penalties enforced for protection of players:
  - **Cross-checking:** using shaft of stick with both hands to check opponent.
  - **Hooking:** using blade of stick on opponent's body to block or impede opponent's progress.
  - **Slashing:** striking or attempting to strike opponent by swinging stick.
  - **Spearing:** poking or attempting to poke opponent with blade of stick.
  - **Interference:** impeding progress of opponent not in control of puck.
  - **Charging:** using more than two skating strides to check opponent.
  - **Checking from behind**
- Officiating: Two or three officials enforce rules, assess penalties, award goals.

PHYSIOLOGY OF ICE HOCKEY

Skating Stride

- **Three phases:** Glide during single support, propulsion during single support, propulsion during double support.
- **Propulsion:** When extending knee joint in skating thrust, quadriceps develop largest contractile force. Hamstrings and gastrocnemius stabilize knee during weight shift and push-off.
- **Stride rate is related to skating velocity.** Stride length is unrelated except in young hockey players.
- Faster skaters show better timing in push-off mechanics with resultant push-off in direction perpendicular to skating direction. Elite skaters sustain gliding phase longer.
- In players aged 8 to 15, increases in velocity are accompanied by increases in stride length and no significant change in stride rate.
- To accelerate quickly, players should attempt full extension of hip, knee, and ankle.
- With fatigue, decrease in skating velocity is caused by decreased stride rate (slower leg extension and longer glide phase) and excessive forward lean.
- Typical game skating behavior is complex activity involving repeated accelerations, decelerations, turning, and stopping. Complicating skating behavior are upper body activities of stick handling, shooting, passing, and checking.

Physical Characteristics of Hockey Players

- **Professional players** are taller and heavier on average than college and junior players.
- **Defensemen** are taller and heavier than forwards.
- **Body composition (% fat)**
  - Junior: 8.6% to 13.6%
  - College: 8.6% to 10.7%
  - Professional: 9.7% to 14.2%
  - Forwards and defensemen have equal body composition.
  - Goalies, on average, have higher body composition than forwards and defensemen.

Energy Expenditure

Physiology has been studied mainly in adult, elite hockey players, which underscores uncertainty of applying this science to youth hockey.

Game

- Shifts
  - One shift averages 45 to 90 seconds with average of two to three play stoppages per shift, lasting average of 27 seconds.
  - Average playing time per shift is 40 seconds with recovery of 225 seconds between shifts.
  - One shift plus recovery averages work capacity of 32 mL/kg per minute (66% of $\dot{V}O_2$ max).
  - Average player plays 14 to 21 shifts each game with average playing time of 21 to 28 minutes per game (based on usual practice of alternating three "lines").
- Energy requirement estimated at two-thirds anaerobic metabolism and one-third aerobic metabolism. On-ice energy requirements of college players estimated at 70% to 80% $\dot{V}O_2$ max and youth hockey players estimated in excess of 80% $\dot{V}O_2$ max.
- On-ice heart rate averages 152 beats per minute.

Time-Motion Analysis

- Adult elite players average 6400 to 7200 meters per game (3.9 to 4.4 miles/game).
- Forwards demonstrate more anaerobic activity than other positions. Aerobic system used primarily for recovery.
- Defensemen have longer playing time (+33%), more shifts (+17%), and longer playing time per shift (+21%) but less recovery time between shifts (−35%). Defensemen average about 62% of skating velocity of forwards.
- Goalie's quick, explosive movements of short duration with rest periods of submaximal activity use primarily adenosine triphosphate-phosphocreatine (ATP-PC) energy system.
- Although few, physiologic studies of youth hockey (older age groups) had similar findings.
- Adult recreational hockey players tend to stay on ice much longer per shift.
- Time-motion analyses are based on use of alternating three lines. In adult recreational leagues, only two lines may be used. At collegiate and professional levels, four lines may be used.
- Heart rate telemetry estimates on-ice intensity averaging 70% to 80% $\dot{V}O_2$ max during 60-minute stop-time game. For 30 minutes of each game, players' $\dot{V}O_2$ max exceeds 90%. Adult recreational players average heart rate intensity in excess of 70%.

Muscle Glycogen Stores (Energy Source)

- Glycogen stores decline by average of 60% for forwards and defensemen after one game.
- All muscle fibers (types I, IIa, and IIb) contribute glycogen; type I depletes (contributes) most.
- Twofold increase in plasma free fatty acids suggests small glycogen-sparing effect in muscle.
- Consecutive-day games usually do not allow complete repletion of glycogen stores (based on diet as desired).

Lactate Accumulation

- Because anaerobic glycolysis is major energy contributor, lactate accumulates over course of game (8- to10-fold increase). Because about 10 minutes are required to remove lactate from exercising muscle, there is inadequate time between shifts for full recovery. Result is mild metabolic acidosis.
- Lactate values usually higher in first and second periods. Forwards and defensemen have similar levels.
- Levels actually lower than predicted because each shift is interrupted by average of two to three play stoppages, averaging 27 seconds. This usually allows about 60% to 65% of phosphocreatine to be resynthesized before next shift.

Muscle Fiber Type

- Wide range of fiber composition.
- No difference from general population; no position-to-position variation.

Anaerobic Power and Endurance

- Forwards, defensemen, and goalies have similar results in peak power and endurance.
- Similar results occurred when younger, less experienced players were tested.

Aerobic Endurance

- Although hockey is largely anaerobic, improving aerobic capacity reduces fatigue and may enhance performance. Involvement of anaerobic system may depend on efficiency of the aerobic system.
- $\dot{V}O_2$ max ranges from 52 to 62 mL/kg per minute. Maximum aerobic capacities of youth hockey players are similar to those of adult players when adjusted for size and weight.
- NHL players have shown consistent increase in aerobic capacities over past 15 years presumably because of more effective off-season and in-season conditioning strategies.

Muscle Strength and Endurance

- Professional players were stronger than amateurs on each of six tests used for comparison.
- Comparing defensemen and forwards at similar levels, data relative to body weight showed them to be equal.
- In comparison to other sports, hockey players obtained high levels for total and relative leg force. Only elite canoeists and athletes from power events scored higher (Finnish study).

Flexibility

- Forwards and defensemen have similar flexibility.
- Goalies have significantly better flexibility (key element for that position).
- Generally, flexibility of hockey players exceeds that of other elite athletes in wrist, hip, knee, and ankle.
- Other elite athletes exceed hockey players in flexibility on neck rotation, all shoulder and elbow actions, trunk extension-flexion, and lateral flexion.

Fatigue

- Hockey players at risk for fatigue. Activities of ice skating require use of all major muscle groups. Hockey has heavy metabolic demands for energy *and* removal of waste products of energy metabolism.
- Studies of fatigue in hockey show failure of return of maximal muscle contractions to pre-exercise levels at 24 hours. Loss of ability to generate maximal force affects athlete's ability to perform peak-force activities to accelerate, stop, and turn.

Detraining

- On-ice practice and game play may not provide sufficient stimulus to maintain or improve fitness among hockey players.
- Studies suggest additional aerobic activities may be necessary during competitive season.

Practical Application of Training Studies

- Programs that have **failed** to improve skating speed: leg squats using weights; pushing partner as technique of resistance skating; speed skating with instruction; skating with ankle weights.
- Six-week preseason training program consisting of continuous running, stair running, flexibility, and strength training resulted in 11% increases in $\dot{V}O_2$ max. During subsequent season, gains in oxygen consumption were lost in absence of any specific in-season aerobic training program.
- Hockey training stimulates cardiovascular conditioning improvement similar to that of continuous training programs in untrained players. In fit, elite players, there were no improvements in cardiovascular fitness over course of season.
- Hockey practice observations show 20 minutes of actual skating during 60-minute practice. Heart rate monitoring, however, provided sufficient stimulus for aerobic training effects.
- Anaerobic endurance improved over course of season by about 16%, but not associated with increases in glycolytic enzymes.
- Muscular fatigue over 6-day routine of practices and games showed decrements in maximal voluntary muscle contractions, implying fatigue. Levels decreased through first 3 days, then reached plateau at level lower than baseline. After hockey practice, muscle output remains diminished over practice-game cycle.

Nutrition of Hockey Players

- Dietary composition: protein, 14% to 20.5%; carbohydrate, 38% to 44%; fat, 34% to 43%.
- Average daily intake is 2800 to 4900 calories.

Environmental Factors

- Ice arenas usually have lower ambient temperatures than other athletic settings, which minimizes risk of heat-related injury.
- Hockey protective equipment reduces ability to dissipate heat.
- Despite hydration between periods and shifts, hockey players lose 2 to 3 kg body weight through sweat each game.

Physiologic Studies and Their Implications for Shift Length

- Shorter shifts result in higher contribution of phosphocreatine and oxidative phosphorylation to adenosine triphosphate (ATP) (energy source) turnover, reducing contribution of anaerobic glycolysis, which reduces consumption of muscle glycogen.
- Shorter shifts result in less lactate accumulation in exercising muscles. Lactate accumulation causes muscles to be inefficient and fatigue more readily. If lactate levels are lower, lactate clears more quickly, and muscles recover more quickly.

INJURIES IN ICE HOCKEY

Epidemiology

- **Incidence and rate:** In NHL, 800 injuries per 1000 league games. Injuries caused NHL players to miss 11% of all games. One injury every 7 hours of play for elite hockey players.

- Collegiate injuries (Table 73-2)
- Age group differences
  - Age 11 to 14: 1 injury/100 hours playing time.
  - Age 15 to 18: 1 injury/16 hours playing time.
  - Age 19 to 21: 1 injury/11 hours playing time.
  - Professional: 1 injury/7 hours playing time.
  - In Finland, youth hockey injuries occurred with incidence similar to youth soccer and alpine skiing.
  - Under age 12, injury was infrequent. Beyond age 12, injuries increased evenly over older age groups.
- Average injury risk of *all* sports is 1.37%. Hockey has an average incidence of 2.71% compared with average risk of 3.95% in soccer.
- One study at elite level showed 5% of all injuries were related to fighting.
- **Catastrophic injury rate:** 2.55 per 100,000 compared with football rate of 0.68 per 100,000. Rules infractions related to 17% of all catastrophic injuries.
- **Injuries per player per year:** youth hockey, 0.02; professional, 3.0.
- **Descriptive injury data**
  - 24% to 45% of all injuries occur during practice (Table 73-3).
  - **55% to 76% of all injuries occur during games:** first period, 20.5% to 31%; second period, 30% to 38%; third period, 28% to 46.2%.
  - In youth hockey, the rate of injury in games is four to six times the incidence in practices. The higher game rates occur in older, more competitive levels. Injury rates increased at ages when checking is introduced. House leagues had lower injury rates than the equivalent travel (more competitive) leagues.
  - **Injury incidence—time in period:** 0 to 7 minutes, 12.5%; 7 to 15 minutes, 40.6%; 15 to 20 minutes, 46.9%.
  - **Acute versus overuse:** acute, traumatic, 80%; overuse, 13.5% to 20%.
  - **Location on ice:** 40% in defensive zone, 35% in offensive zone, 25% in neutral zone.
  - **Injury by position:**
    - Defensemen (107.8 per 1000 game hours): 55% minor, 30% moderate, 15% severe.
    - Forwards (71.8 per 1000 game hours): 73% minor, 21% moderate, 6% severe.
    - Goalies (39.2 per 1000 game hours): 83% minor, 17% moderate, 0% severe.
  - Male and female injury rates are identical, in spite of "no-check" rules in women's hockey.
- **Injury potential**
  - **Collisions** with players, boards, goalposts.
  - **Skating velocity** (examples): senior amateur players, 30 mph (48 km/hour); PeeWee (ages 12 to 13), 20 mph (32 km/hour).
  - **Sliding velocity** (after a fall): 15 mph (24 km/hour).
  - **Hockey puck** (6 ounces [170 gm] hard rubber) shooting velocity: professional, 120 mph (192 km/hour), senior recreational, 90 mph (144 km/hour); PeeWee (ages 12 to 13), 50 mph (80 km/hour). Maximal impact force of puck at its terminal velocity is 1250 pounds (567.5 kg). Hockey masks deform at puck speeds of 50 mph (80 km/hour).
  - **Hockey stick** velocity is measured at 100 to 200 km/hour during shooting.
  - **Hockey skates** often cause lacerations from sharp, steel blades.
  - **Nonimpact forces:** vertical reaction force during skating stride is 1.5 to 2.5 times body weight compared with 3 to 4 times body weight in runners; posterior push force during skating measured at 150 pounds.
- **Mechanism of injury** (Tables 73-4 to 73-6)
- **Anatomic sites** (Table 73-7)
- **Type** (Table 73-8)

Table 73-2 COLLEGIATE INJURIES

	Men (injury rate reported per 1000 athlete exposures)	Women (injury rate reported per 1000 athlete exposures)*
Type	Game: 13.8 Practice: 2.2	Game 12.8 Practice 2.3
Position	Goalie 2.7 Defense 5.0 Forward 5.1	Goalie 14.0% Defense 41.2% Forward 44.7%
Period	First 15.1 Second 15.1 Third 11.2	N/A
Location	Home 11.9 Away 15.6	N/A
Site		**Game/practice****
	Knee/leg 22%	Head/neck 25.4/16.2
	Head 19%	Upper extremity 30.3/22.2
	Shoulder 15%	Trunk/back 11.4/26.4
	Foot/ankle 12%	Lower extremity 31.8/31.1
	Hip/groin 9%	Other 1.1/4.2
	Back/spine 9%	
	Wrist/hand 7%	
	Other 7%	

*Checking is not permitted in women's hockey. Injuries are the result of "incidental" contact (collisions with other players, the boards, the ice, and the goal), the stick, and the puck. Fifty percent of injuries occur as a result of collisions.

**Concussion is the most common injury reported in both practices and games.

N/A, statistics not available.

Table 73-3 PRACTIVE VERSUS GAME (INJURIES/1000 PLAYERS): COLLEGE INJURY COMPARISON

Sport	Practice	Game	Total
Men's basketball	5.1	9.5	6.0
Men's gymnastics	4.7	14.8	8.9
Wrestling	6.8	28.1	8.9
Hockey	2.1	15.1	5.0

Table 73-4 MECHANISM OF INJURY IN ELITE ATHLETES

Mechanism	Beiner et al. 1973 (%)	Lorentzon 1988 (%)
Stick	25	11.8
Puck	17	14.5
Collision*	17	57.9
Skate	6	2.6
Other	36	13.2

*33% injuries in adult hockey with 14% of collisions unintentional.

Table 73-5 MECHANISM OF INJURY IN COLLEGE ATHLETES

Mechanism	%
Legal check	44.6
Accidental collision	28.6
Illegal stick check	12.2
Fighting	6.5
Illegal check	5.8
Noncontact	2.2

Table 73-6 MECHANISM OF INJURY IN YOUTH (SMALL STUDIES)

Mechanism	%
Collision*	50-86
Puck	14.3
Overuse	14.3
Stick	7.0
Skate	7.0

*10% unintentional collision. In one study, illegal checks and violations caused 66% of injuries, but penalties were assessed only 14% of the time.

Table 73-7 ANATOMIC SITE OF INJURIES

Site	%
Professional*	
Head, scalp, face	28.1-52.9
Eye	2.6
Shoulder	5.6-21.9
Hand	2.1-10.5
Thigh (groin)	15.3-35.7
Knee	11.6-17.0
Miscellaneous (back, foot/ankle, ribs)	3.6-23.3
College	
Knee	18.6
Face, eye, mouth, teeth	17.6
Shoulder, clavicle	14.9
Head, neck	10.6
Thigh, hamstring	9.0
Forearm, wrist, hand	6.9
Hip, groin, abdomen	6.4
Chest, back	4.8
Arm, elbow	3.7
Ankle	3.2
Youth†‡	
Head and neck	10-23
Upper body	23
Shoulder/arm	19-55
Trunk	13-17
Leg	17-19

*Range, four studies.
†Range, two studies.
‡At Bantam level (ages 13-14), weight differences of 53 kg and height differences of 55 cm have been reported. Smaller players are more likely to be injured.

Table 73-8 TYPES OF INJURY

Type	%
Adult (elite)*	
Contusion	25-47
Laceration	28-50
Fracture†	4-15
Dislocation	1-8
Muscle, ligament	3-12
Other	3-5
College	
Sprains, dislocations	22
Contusions	20
Lacerations	13
Strains	11
Fractures	10
Concussions	8
General trauma	6

*Range of three studies.
†Fractures are 12 times more common in leagues with checking.

- **Severity**
  - **Minor (<7 days' absence):** 61% to 73% (46% of all minor injuries caused by body checks).
  - **Moderate (8 to 30 days' absence):** 19% to 22%.
  - **Severe (>30 days' absence):** 8% (75% of all severe injuries caused by body checks).
- **Ice surface size and injury (Junior A)**
  - Injury rates: inversely proportional to ice surface size.
  - Neurotrauma: no relationship to ice surface size.
  - Aggressive penalties: no relationship to ice surface size.
- **Incidence and severity of injuries is increasing.** Possible explanations: increased participation, increase in speed of game and size of players, longer seasons and more out-of-season participation at all levels, lack of proper training, inconsistency in rule enforcement.

Acute, Traumatic Injuries

Acute, traumatic injuries account for eighty percent of all hockey injuries.

Head

- Full spectrum of injury due to closed head trauma, including death.
- **Concussions account for 8% to 14% of all hockey injuries.** Collisions with other players account for 45% of concussions; collision with boards, 34%, and being hit by a stick, 22%.
- Age-group concussion incidence (meta-analysis): 5 to 14 years, 0.0 to 0.8 per 100 player hours; high school, 0.0 to 2.7 per 1000 player hours; college, 0.2 to 4.2 per 1000 player hours; elite adult, 0.0 to 6.6 per 1000 player hours.
- Frequency of closed head trauma has decreased because of mandatory use of helmets.
- Increased angular velocity of head and neck with helmet and face mask does not appear to increase head or neck injury risk.
- In an evaluation of sport-related head injuries reported from emergency departments from 1993 to 1999 for the sports of ice hockey, soccer, and football, the overall head injury rate for ice hockey was 8.1 to 13.7 per 10,000 participants, and 6.3 to 9.7 per 10,000 and 9.4 to 13.5 per 10,000 participants for soccer and football, respectively. The concussion rate was 2.0 to 3.5 per 10,000 for ice hockey, and 1.4 to 3.1 per 10,000 and 3.1 to 5.2 per 10,000 for soccer and football.
- Under-reporting of concussions in youth hockey; study results of same populations differ depending on the sources reporting.
  - Official injury reports—0.25 to 0.61 concussions per 1000 player game hours.
  - Volunteer observer reports—4.44 to 7.94 concussions per 1000 player game hours.
  - Player surveys (elite level)—6.65 to 8.32 concussions per 1000 player game hours.
  - Player surveys (nonelite level)—9.72 to 24.30 concussions per player game hours.
- See Chapter 39, Head Injuries, for information about diagnosis and treatment.

Neck

- **0.4% to 9.2% of all hockey injuries.**
- If player has loss of consciousness, assume cervical spine injury.
- See Table 73-9 for emergency department data regarding neck injuries.
- **Since hockey helmets have been widely used, incidence of cervical spine trauma has increased. Attributed to more aggressive play associated with better and more complete protective gear.**
- **Increased incidence of severe cervical spine injury since early 1980s.** Before 1973, no spinal cord injuries caused by

Table 73-9 EMERGENCY DEPARTMENTS 1993-1999

Type of injury	Ice hockey	Soccer	Football
Sport-related neck injuries			
Total neck injuries	5038	19,341	114,706
Fracture/dislocation	105	214	1,588
Contusions/sprains/strains	4964	17,927	104,483
Lacerations	199	0	621
Rates of Injury			
Neck injuries	1.68-4.26	1.34-2.60	4.56-7.18
Fractures/dislocation	0.08-0.30	0.01-0.06	0.06-0.09
Strains/sprains/contusions	1.68-3.87	1.14-2.31	4.25-6.38

Data from Delaney JS, Al-Kashmiri A: Neck injuries presenting to emergency departments in the United States from 1990 to 1999 for ice hockey, soccer, and American football. Br J Sports Med 39:21-25, 2005.

hockey were reported. First published report in Canadian literature occurred in **1984. Between 1981 and 1985, 15 major cervical spine injuries reported each year attributable to hockey.** Currently, an average of 17 severe spinal injuries are reported each year.

- **Mechanism of serious injury:** axial load of cervical spine with head in neutral alignment. In most situations, player is pushed or checked from behind and slides headfirst into boards.
- **Cervical spine injury data**
  - 96% men, 4% women
  - Of 117 cases, 5 died. 48% had injuries to vertebrae C4-C5, C5, C5-C6; with spinal cord affected in slightly over half of cases. 29 of 117 are permanently quadriplegic.
  - Causes: impact with boards, 65.0%; impact with player, 10.3%; impact with ice, 7.6%; impact with goalpost, 0.9%.
  - 50% of spinal cord injuries occur in the 16 to 20 year age group.
- **Factors affecting high incidence of spinal cord injuries:** player taller and heavier, skating faster; increased aggressive behavior at all levels (imitating style of professionals); rules not enforced consistently; insufficient emphasis on conditioning; equipment problems (lack of shock absorption boards). See Chapter 40, Neck Injuries, for information about diagnosis and treatment.
- Cervical spine injury characteristics (1980–1996, Finland and Sweden): 16 cases with permanent disability; in 50% of cases, mechanism was checking from behind; 69% of vertebral injuries involved fractures and luxations between C5 and C7.

Eye and Face

- Significant injury reduction since mandatory face mask rule:
  - Unilateral injuries reduced from 478 to 42 in one season; blindness reduced from 37 to 12 in one season.
  - Hockey still accounts for 37% of eye injuries and 56% of blindness in sports.
  - **Face masks have been estimated to save in excess of $10 million per year in injury costs.**
- Injury types (before face mask rule): periorbital soft tissue trauma, 43%; hyphema, 19%; iris damage, 13%. Stick responsible for more eye injuries than puck.

Throat

- Goalies particularly at risk for throat injuries (blunt trauma to larynx) from high-speed pucks.
- Padded collars and other deflectors worn by both skaters and goalies to reduce injury risk.

Shoulder

- **Acromioclavicular injuries**
  - Mechanism of injury: direct blows to shoulder and falls on outstretched hand.
  - Acromioclavicular separation common.
  - In Norfray's 1977 study of 77 hockey players, 45% had asymptomatic x-ray abnormalities, including osteolysis of acromioclavicular joint and callus from united and nonunited distal clavicle fractures.
  - In a study by Lorentzon and colleagues, only one of four players with acromioclavicular separations missed more than 1 week of practice or games.
- **Glenohumeral dislocation:** 8% incidence; greater morbidity than acromioclavicular separations; high rate of recurrence.

Elbow, Wrist, and Hand

- 20% of moderate-to-severe injuries are a result of wrist and hand problems.
- **"Skier's" or "goalkeeper's" thumb:**
  - Associated with fall on outstretched hand while hockey stick is still in possession.
  - Treatment same as that outlined for other sports.
  - It is possible to fashion splint of thermoplastic, plaster, or fiberglass to fit in hockey glove; may be prohibited by rules or game officials.
  - Nonsurgical as opposed to surgical therapy recommendations are changing for early versus late repair of grade III ulnar collateral ligament sprain. Check with hand specialist.
- Scaphoid fractures: uncommon.
- **Metacarpal fracture:** usually related to stick trauma ("slashing").
- **Lacerations** of forearm, wrist, and hand may occur after fall if another player skates over fallen athlete. Skate blades can cut through thinner leather over palm of gloved hand. Wear and tear of leather of glove's palm make it paper-thin—affords little protection.
- During fights and melees, gloves are often dropped, allowing other traumatic hand injuries plus risk of human bites. Usual human bite precautions are necessary.

Back

- Infrequent site of injury, 4.3% to 7.0%.
- Spondylolysis can occur.
- Most back pain is probably of muscular origin and may be treated conservatively.
- Severe spinal injuries: T1 to T11, 9.2%; T11 to T12/L1-2, 6.0%; L2 to S5, 5.1%.

Abdomen or Groin

- Common site of lower extremity injury because of forceful hip adductor contraction during skating stride; 10% of injuries in some studies.
- Inguinal hernias, osteitis pubis, and pelvic or hip stress fractures have been reported in hockey players.
- Rectus abdominis muscle injuries can also be debilitating and chronic in hockey players. When conservative treatment fails,

surgical reinforcement has been necessary to permit athletes to return to skating.
- NHL experience (1991-1992 to 1996-1997)
  - Injuries per 1000 player hours: 1991-1992, 12.99; 1996-1997, 19.87.
  - Preseason injury rate 5 times greater than regular season, 20 times greater than postseason play.
  - 23.5% are recurrent injuries; more than 90% are noncontact injuries.
  - Mean time loss: abdominal injury, 10.6 sessions; groin injury, 6.6 sessions.
  - Groin injury accounts for 77% of injuries (68% were adductor strains).
  - Abdominal injury: 23%.
  - Position: forwards, 60.6%; defensemen, 28.9%; goalies, 5.5% (4.9% not recorded).

Thigh Contusions

- Hockey players are at high risk because of high incidence of collisions with players, goals, and boards.
- Generally players are protected, but protective padding may slide to one side.
- Treatment of quadriceps contusions is no different from other collision sports.

Knee

- **Most common lower extremity injury.**
- **Medial collateral ligament (MCL) sprain:** most common serious knee injury in most series; usually results from varus or valgus and rotational stresses.
- Anterior cruciate ligament sprains: less frequent than MCL sprains.
- Meniscal injuries usually occur in combination with ligamentous injuries.
- Hockey poses less injury risk to knee than soccer.

Ankle

- Sprains are less common than in other sports because of protection offered by skate boot.
- Lacerations just above boot often involve tendons.
- Ensure that players tuck tongue of skate boot under shin guards to minimize exposure of anterior ankle to laceration.

Foot

- Fractures of bones in foot result from direct blow, usually from puck.
- Other foot injuries are uncommon.

Overuse Injuries

- Few data, other than incidence, about types of overuse injuries.
- **Adductor tendinopathy and patellar tendinopathy are most common overuse injuries, specifically related to skating stride.**

Special Medical Situations

- **Commotio cordis:** Manifestation of concussive injury to heart resulting in ventricular dysrhythmia and cardiac asystole.
  - Despite protective equipment, rare case reports highlight slight risk of such cardiac injury in hockey. Epidemiologic studies suggest pediatric and adolescent age groups may be predisposed.
  - 70 deaths (34 organized sports, 36 recreational activity) have been reported; 40 attributed to baseball, 7 to ice hockey. Survival rate: 10%.
- **Indoor air quality problems ("ice-hockey lung")**
  - Propane- or gasoline-propelled ice-resurfacing machines (known as Zambonis) have been implicated in causing illness when engines malfunction or ventilation within arena is inadequate.
  - Players are at increased risk to minimal exposures compared with spectators.
    - Nitrogen dioxide gas is heavier than air, found at higher concentrations at ice surface.
    - Thermic inversion occurs because of ice temperature.
    - Plexiglass shields along boards alter air circulation at playing level.
    - During games and practice, players have minute ventilation up to 30 times higher than at rest.
- **Nitrogen dioxide-induced respiratory illness**
  - Nitrogen dioxide is byproduct of combustion.
  - Recommended limit: less than 0.5 ppm (parts per million).
  - Common symptoms (acute onset after unknown indoor exposure after hockey practice or game): cough, hemoptysis, dyspnea, chest pain, headache, weakness; pulmonary edema (rare).
  - Treatment: withdrawal from toxic environment, bronchodilators, corticosteroids. Untreated, most symptoms resolve within 2 weeks.
  - Late complications of bronchiolitis obliterans may develop 2 to 6 weeks after initial symptoms.
- **Carbon monoxide poisoning**
  - Source: improper combustion of fuel of Zamboni; inadequate ventilation system for arena.
  - Recommended limit: less than 30 ppm (<25 ppm in Canada; some researchers suggest 20 ppm).
  - Symptoms: acute respiratory (hemoptysis, dyspnea, chest pain, coughing spells) and central nervous system (headache, dizziness, sleepiness, nausea and vomiting).
  - Treatment: withdrawal from source of toxin; rarely requires emergency treatment.
- **Safeguards**
  - Thirteen states have tested indoor air quality of ice arenas.
  - Only three states have mandatory air quality testing in ice arenas.
  - Adequate ventilation of indoor arenas should be ensured.
  - Regular inspection and maintenance of ice-resurfacing machines should be implemented, and indoor air quality should be monitored regularly.

Injury Prevention

- Continue to update protective equipment.
- Ensure use of mouth guards.
- Fabric designs with high coefficient of friction are under evaluation to reduce sliding speeds of fallen players.
- Enforce rules. One study suggests that 39% of all injuries were attributed to foul play.
- Effective training and conditioning may minimize injury risk. Adequate nutrition, training, and hydration may reduce third-period fatigue and reduce fatigue in situations when several games are played on consecutive days as in tournaments.

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Ice Skating

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Carole S. Vetter, Emily B. Porter, and Sarah Newman

FIGURE SKATING

History

- Figure skating is a sport that focuses on a unique combination of athleticism, strength, endurance, gracefulness, and artistry on ice.
- It is an evolving sport that began in the early 1800s, when the sport consisted of complicated figures traced on the ice.
- Jumps and spins were introduced in the 1860s, and figure skaters have been developing more complex moves ever since.
- Men now routinely perform at least one quadruple jump (four revolutions in the air) in their long programs and several women have performed triple axels (three and one half revolutions in the air) in competition.
- Today there are 196,000 members and 645 clubs registered nationwide with the United States Figure Skating Association (USFSA).

Disciplines

- There are four major disciplines: singles skating, pairs skating, ice dance, and synchronized skating.
- Singles skating: Jumps, spins and connecting steps consisting of footwork and efficient use of blade edges performed by a single skater.
- Pairs skating: Jumps and spins performed separately and in tandem by a male and female skater, along with overhead lifts and throw jumps.
- Ice dance: Similar to pairs skating, but with more focus on intricate footwork and performance on deep edges, with specific rules about lifts and the amount of time the skaters can skate separately.
- Synchronized skating: Eight to 24 skaters perform on the ice together, moving simultaneously as a group.

The Athletes

- The majority of figure skaters are female, began skating when they were 5 to 8 years old, and reach the peak of their competitive careers by their teens or early 20s.
- Competitive figure skaters can spend 2 to 4 hours per day in on-ice training plus 1 to 3 hours per day in off-ice training for up to 11 months of the year. Off-ice training includes strength and flexibility training, dance, aerobic and anaerobic conditioning, and choreography.
- Training can often become all-encompassing to elite skaters and their families. It is not uncommon for young skaters to move to new cities or opt for home-schooling as they make training their first priority.
- The intensity and stress of the vigorous training can take a toll on figure skaters and lead to emotional and psychological problems such as burnout and eating disorders.

Equipment

- Figure skates are composed of a leather boot and a steel blade.
  - The boots have become increasingly stiff over the past 20 years to accommodate the stress placed on them by skaters performing triple and quadruple jumps. The increased stiffness of the boot may contribute to weaker ankles in competitive skaters and an increased number of skating injuries.
  - Boots are often custom-fit to the skater's feet; however, boots still tend to fit poorly. The shape of the boot makes placing traditional orthotics into the boot difficult and it may be necessary for special orthotics to be molded into the boot when needed.
  - Boots can cost $200 to $700 per pair depending on the level of skater and are usually replaced every 6 to 12 months, depending on how fast the skater breaks them down.
- Figure skating blades differ from hockey or speed skating blades in that they contain a large front toe pick, which is used in jump landings and some jump take-offs.

Injuries

- Common problems include acute injuries, chronic overuse injuries, and medical problems.
- Factors contributing to injuries are the boot, training regimen, environment factors, and conventions of the sport that reward high-risk moves and slender body type.
- About half of injuries are traumatic and half are related to overuse.
- Singles skaters tend to suffer more overuse injuries, whereas acute injuries are more common in pairs skaters and ice dancers.
- The lifts and throws that pairs skaters and ice dancers perform predispose them to more acute injuries, because of increased speed, momentum, and force in falls and severity of injury.
- Overuse syndromes in singles skaters increase as athletes continue to increase the intensity of their training.

Foot and Ankle Injuries

- "Lace bite" or tendonitis of the tibialis anterior and toe extensors can occur as a result of improper placement of the tongue of the boot (Fig. 74-1).
- Malleolar bursitis can occur secondary to friction from the boot at the medial or lateral malleolus.
- "Pump bumps," or Haglund's deformity, and Achilles tendonitis can occur with a skate boots that fit improperly posteriorly (see Fig. 74-1).
- Inflammation of an accessory tarsal navicular and the prominence at the base of the fifth metatarsal are also common as are corns and hammertoes (see Fig. 74-1). Most of these injuries are treated with modification of the skate boot by extra padding or friction-reducing moleskin, locally "punching out" the leather by stretching, or pressure-relieving padding such as foam or felt donuts.
- Ankle sprains also occur in ice skaters though most commonly during off-ice training sessions. This is likely due to weaker peroneal muscles from wearing stiff skating boots.

Stress Fractures

- Lower extremity stress fractures were the most frequent overuse injury recorded in a 2003 study of elite junior women skaters.
- Common in the first and second metatarsals, and occur more frequently in the skater's take-off leg for toe-pick jumps (see Fig. 74-1).
- Stress fractures are also commonly seen in the tibia, fibula, and navicular bones.

Knee Injuries

- Patellofemoral syndrome is common in figure skating as in most jumping sports. Skaters, like other athletes, are at increased risk when their vastus lateralis is relatively overdeveloped compared to the vastus medialis and the quadriceps and hamstring muscle groups have poor flexibility.

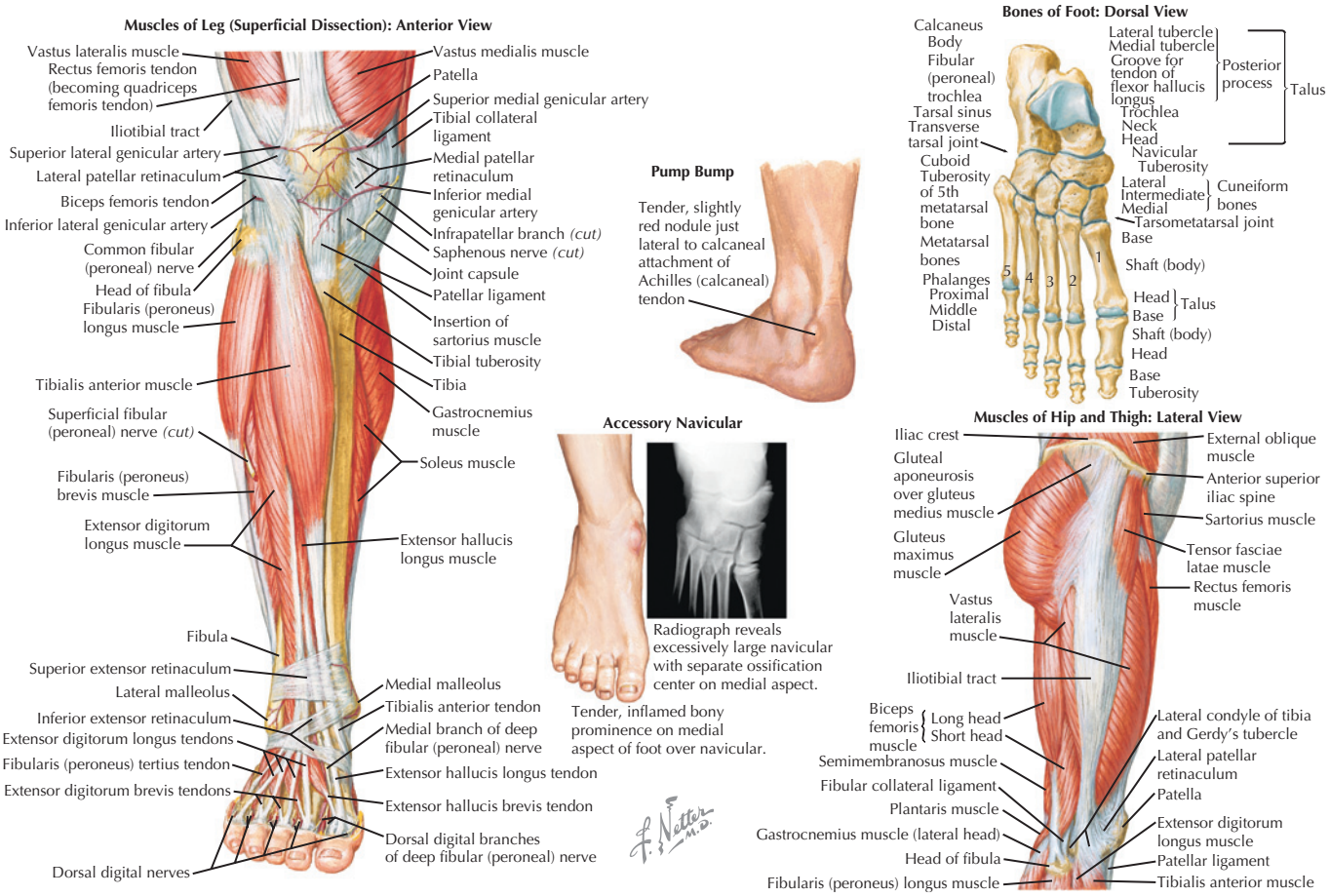


Figure 74-1 Figure Skating Injuries.

- Patellar compression injuries arise from repetitive falling, although actual patellar fractures are rare. These injuries are seen with increased frequency when performing more difficult jumps and with increased jump frequency.
- Patellar tendonitis is less common in figure skating than in other jumping sports, although it is seen more commonly in elite rather than amateur skaters.
- Ligamentous and meniscal injuries are uncommon in skaters. This is likely because skaters land gliding backward, requiring cocontraction of the quads and hamstrings, which protects the ligaments and meniscus. Also, there is a relative lack of fixation of the blade on the ice, which decreases torque at the knee joint.

Hip Injuries

- Muscle strains of the hip flexor, adductor complex, and oblique abdominal muscles are seen commonly in skaters performing triple and quadruple jumps.
- Iliac crest apophysitis is relatively common in skeletally immature skaters performing triple or quadruple jumps with edge take-offs because of the repetitive stress from jump take-offs or missed landings (see Fig. 74-1).

Back Injuries

- Low back pain is common in ice skaters as the result of acute trauma or overuse injury from repetitive falls or improper equipment.
- These injuries may also be related to the stiffness of the skater's boot. The increased rigidity limits ankle and knee motion, causing the skater to increase flexion at the hip and extend the back to maintain balance.
- These injuries may include lumbar strains, facet pain, posterior iliac crest injuries, spondylolysis, or spondylolisthesis.
- Sacroiliac dysfunction is also frequently a cause of low back pain, a result of uneven forces from repetitive falls on one hip.

Upper Extremity Injuries

- Upper extremity injuries are less common in skaters, especially in singles skaters.
- Wrist sprains and fractures may occur as a result of falling.
- Rotator cuff injuries can occur in male pair skaters because of repetitive lifting.

Medical Issues

- Exercise-induced bronchospasm (EIB) is common in these athletes because of their exposure to the cold, dry air found in ice rinks, as well as the chemicals used to maintain the ice surface or Zamboni fumes, which can contain air pollutants such as carbon monoxide and nitrogen dioxide (see Chapter 30, The Hypertensive Athlete).
- Disordered eating is a frequent problem in figure skating, likely related to the major role that a skater's appearance plays in the judges' performance evaluation. Studies examining figure skaters' dietary intake have widely varied results, suggesting that some skaters have disordered eating patterns but they are not pervasive through the sport. Restrictive eating patterns may contribute to delayed menarche in skaters. This is often perceived as an advantage by many skaters and

coaches, because the onset of puberty is often accompanied by the accumulation of additional body fat and mass that must be lifted into the air with each jump, as well as wider hips that contribute to decreased rotational speed.
- Osteoporosis has not frequently been found in women skaters; however, physicians caring for competitive figure skaters must be suspicious for the female athlete triad.

SPEED SKATING

History

- Speed skating began 3000 years ago, in the early 13th century, as a mode of transportation across frozen lakes and rivers in Scandinavia. At that time the skates were made of bone or wood.
- As the popularity of skating grew through the Netherlands and Europe so did the development of the skates and by 1400 metal skates appeared. The all-iron skate was developed in Scotland in 1572.
- The first known skating competition was held in the Netherlands in 1676 with the first recorded competition taking place in Oslo, Norway, in 1863.
- Speed skating has been an Olympic event for men since the first winter Olympics in 1924 and for women since 1960.
- Today, speed skating consists of two disciplines: long track speed skating and short track speed skating. Short track speed skating became an Olympic event in 1992 after being a demonstration sport at the 1988 Games.

Disciplines

Long Track Speed Skating

- A race against the clock.
- Skaters race in pairs on a 400-meter track. Skaters compete in separate lanes and all events are run in a counterclockwise direction.
- In order for the racers to skate the same distance, they must change lanes during each lap and begin from a staggered start. The cross-over occurs on the back stretch of the oval with the racer in the outer lane having the right of way.
- Contact between skaters is prohibited and each racer has only one opportunity to produce a winning time.
- A new team long track event, the Team Pursuit, was added to official competition during World Cups, World Single Championships, and the Olympic Games in 2003.
- A team consists of three skaters and two different teams skate against each other at the same time, starting on opposite sides of the track.
- The teams race without changing lanes. The last skater over the finish line gives the team their official finish time.

Short Track Speed Skating

- A race against other competitors and is an elimination event.
- Four to six racers compete per heat with the first and second place finishers advancing to the next heat.
- Skaters try to outskate and outwit the competition.
- Athletes skate on a 111-meter track within an international sized 30-by-60-meter ice hockey rink.
- The lead skater has the right of way and it is up to the passing skater to pass cleanly without body contact.
- Skaters often skim their fingers along the surface of the ice inside the track but must skate around the outside of the blocks.
- In addition to the individual distances, short track also has a relay event. Four skaters skate in a relay team competing against three other teams at the same time. Skaters on the same team exchange by the lead skater being pushed forward by the exiting skater. Only one skater on each team can use the racing track, the others have to rest in the middle (inside of the blocks).

The Athlete

- The average age of most competitive long track speed skaters is 20 to 24 years and the average age of most short track speed skaters is 19 to 22 years.
- These athletes average 8 hours of training a day, which includes on-ice and dry land training.
- The competition season runs from September to April and during this time the athletes travel an average of 2 weeks out of every month.
- Most of these athletes continue dry land training from May to July and take August off. Dry land training consists of technique training, aerobic conditioning, balance, agility, and weight training.
- Those athletes in high school during training are often homeschooled and those athletes in college most often take night classes or classes offered online.

Equipment

Long Track Speed Skating

- Skaters wear skin-tight racing suits (skin suits) with hoods to decrease air resistance (Fig. 74-2). The racing suit must conform to the natural contour of the racer's body and cannot be otherwise altered. There are certain restrictions for the material used.
- Racers often wear glasses to protect their eyes from wind and ice chips and to reduce glare and improve visibility.
- The early 1990s brought a change in the skates for long track racing from a "fixed blade" skate to a "klap blade."
  - Unlike fixed blade skates, the heel of the klap blade skate is not attached to the boot and the toe of the blade is affixed to the boot with a hinge (see Fig. 74-2).
  - At the end of each stride, as the skater picks up the skate, the blade briefly disconnects from the heel of the boot.
  - When the ankle has fully extended and the heel is already off the ice, the full length of the blade is still in contact with the ice pushing.
  - At the moment the blade leaves the ice, a spring mechanism mounted on the front of the boot snaps the blade back up to the boot, resulting in the clapping sound that gives the skate its name.
  - This mechanism keeps the blade on the ice longer, increasing the skater's pushing power and therefore speed.
- Pads to protect the athletes in case of falls surround the outer diameter of the track.

Short Track Speed Skating

- Skaters wear similar skin suits as long track skaters but without a hood.
- The short track skin suits are required to have kneepads, shin guards, and a neck guard to protect the skaters from the blades of other skaters. Furthermore, at international competitions, the skaters have to wear cut-resistant underwear.
- Racers wear a hard plastic helmet to prevent potential head injuries from collisions with competitors, the ice, or side walls (see Fig. 74-2).
- Short track racers also wear ceramic fingertip gloves to protect their hands from other skater's blades and the ice as they are going around a curve and skim their inside hand on the ice to help maintain balance.
- Short track skaters wear a fixed blade skate; however, the boots on short track skates lace up higher on the ankle than traditional skates.
  - These skates are constructed from customized foot molds to help stabilize the foot and the ankle so the skaters can more tightly round corners.

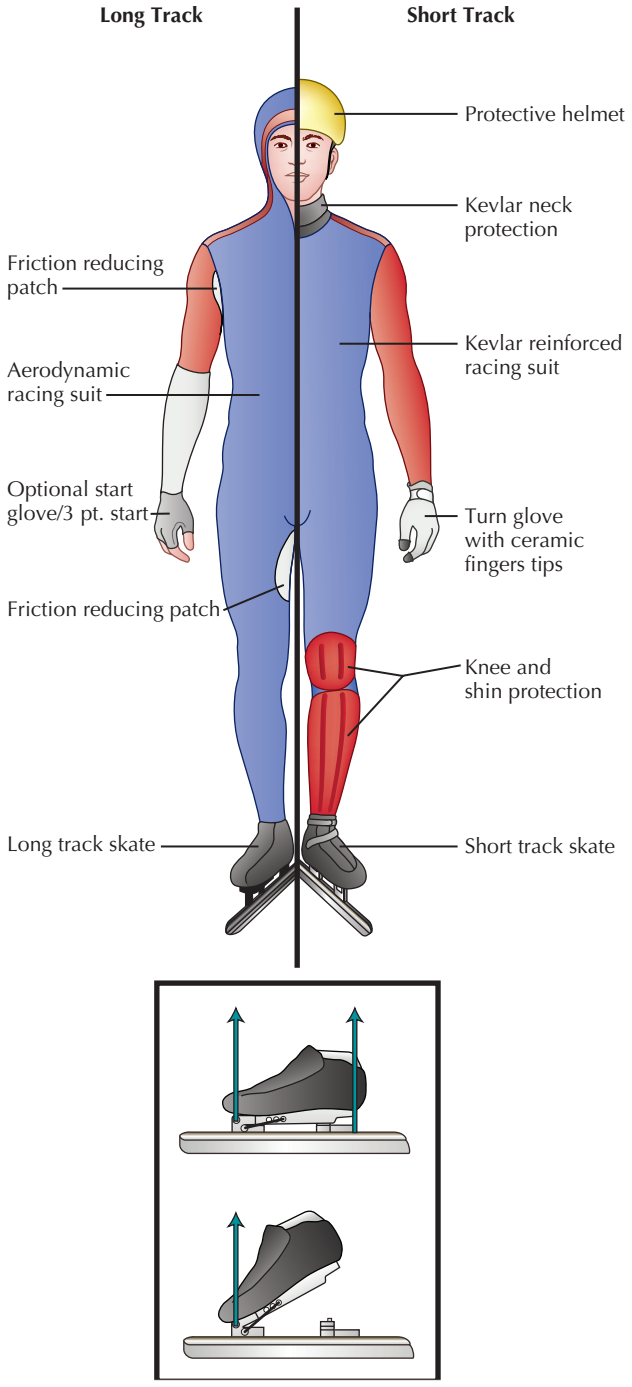


Figure 74-2 Speed Skating Equipment.

- The blades are extremely sharp and are bent in at an arc that mirrors the direction of the turn to better grip the ice.
- The blades are also placed off-center to the left to keep the boot from rubbing the ice when leaning into the left turn at high speed.

- The walls of the indoor rink enclosing the short speed track are padded for the protection of the athletes.

Injuries

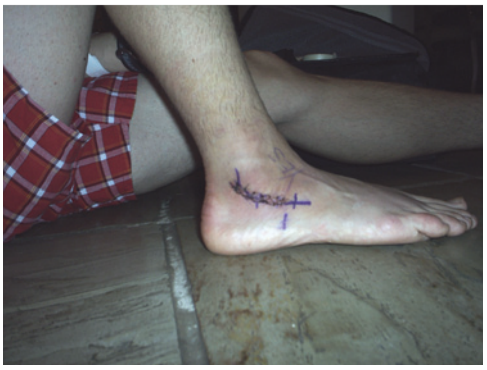
- There is very little published data regarding the incidence and patterns of injury for long and short track speed skating. Previous authors have reported personal experience and some unpublished statistics kept by the U.S. Olympic Committee. There is one retrospective study on a single season of short track speed skating.
- In general, participants in long and short track speed skating are at risk of injury during training and competition, with the short track skater at higher risk during competition because of the collision nature of the sport.
- Speed skaters travel internationally for their entire skating season and therefore have medical conditions similar to other elite athletes, including colds, upper respiratory infections, and viral syndromes.
- Reactive airway conditions are common because these athletes are constantly exposed to cold and dry air in the ice rinks where they train and compete.

Long Track Speed Skating

- Many of the overuse/chronic injuries seen in long track speed skaters are secondary to the position their bodies are in while skating and the constant repetitive motion of skating the same direction around the track counterclockwise.
- Mechanical low back pain and lumbar disc problems are common. Also, chronic hip flexor and iliotibial (IT) band tightness as well as iliopsoas tightness cause painful hip symptoms. IT band tightness and chronic knee flexion leads to the common problem of patellofemoral pain syndromes, patellar tendonitis, and IT band syndrome.
- The skates these athletes wear also lead to some common problems. The skates are worn tightly and often without socks. This can lead to callosities, corns, bunions, and hammertoes and recurrent tinea pedis. The skates are often laced tight enough to cause numbness in the foot, including the great toe and dorsal foot.
- These tightly laced skates can also cause irritation to subcutaneous tendons, resulting in Achilles tendonitis/tenosynovitis and "lace bite," also known as tibialis anterior, extensor hallucis longus, and extensor digitorum longus tendonitis.
- Somewhat similar to sprinters in track, the start of a speed skating race involves an explosive sprint from a stationary position. However, instead of quadriceps and hamstring strains, speed skaters often sustain hip adductor and hip flexor strains.
- Although falls and collisions are less common than in short track speed skating, trauma-related injuries can occur. The most common injuries due to falls are lacerations from skate blades, contusions, fractures, and joint sprains, especially of the upper extremity, from the skaters bracing for the fall (Fig. 74-3).

Short Track Speed Skating

- Short track skaters often have several of the same overuse/chronic injuries as seen in the long track athletes, including mechanical low back pain and lumbar disc problems, hip pain syndromes, and patellofemoral pain.
- More unique to the short track skaters is peroneal tendonitis due to the repetitive forceful dorsiflexion and eversion needed for the skate cross-over on the short tight curves.
- In a retrospective review of a single season for short track speed skating the most common in-competition injuries were contusions, fractures, and lacerations. The most common on-ice training injuries were strains, contusions, and sprains.
- As with any collision sport, all types of trauma can occur (see Fig. 74-3). Those most concerning for short track speed skaters are shoulder dislocations, ankle and lower extremity fractures, concussions, cervical-spine injuries, thoracic and rib contusions, and lacerations (most commonly to the lower extremity).



Lateral malleolus laceration from skate blade.

Short track skater collision.

Skaters bracing for fall.

Skaters bracing for fall.

Figure 74-3 Speed Skating Injuries.

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Sailing

Joanne B. Allen

GENERAL OVERVIEW

History of Sailing

- Sailing has been a vital mode of transportation and trade since the dawn of history when boats were built by the Phoenicians, Egyptians, Greeks, and Romans.
- The earliest representation of a ship under sail appears on an Egyptian vase from about 3500 BC.
- From the Polynesian outrigger proa, and the Chinese lugsail or "junk rig," to the Arabic triangular sail dhows, different types of sailboats were developed through the centuries.
- The oldest indication of sailing for pleasure rather than transportation, trade, or warfare came in the 16th century as the Netherlands became a maritime power.
- The first Dutch "yacht" arrived in England in 1660 as a gift to King Charles I.
- The small boat design stimulated a "sporting" new approach to yachting in Britain.
- In 1661, two more yachts were built: *Catherine*, a second yacht to King Charles, and *Anne* for the King's brother. Competition between the King and his brother ensued and the vessels raced along the Thames, in the first pleasure sailing race in history.

Competitive Sailing

- First yacht club was founded in Ireland around 1720. Named the Water Club of Cork, it was later refounded as the Cork Yacht Club in 1828.
- The first club in England was founded in 1820, named the Royal Yacht Club. The first yacht club in the United States was founded in 1844, the New York Yacht Club.
- The Golden Age of yacht clubs and sailboat racing regattas was launched with the Royal Yacht Squadron's Cowes Week regatta, held annually since 1826, in Cowes, England.
- Competitive sailboat racing continued to become popularized with the America's Cup Race, which started in 1851 off the Isle of Wight, when the yacht "America" was victorious over the British competitors and claimed the "Hundred Guinea Cup," which is now the oldest continuously contested trophy in the world and now known as the "America's Cup" (Fig. 75-1).
- As the sport of sailing grew, many other regattas and competitive events have developed to include offshore ocean racing, global solo-circumnavigation, and in-shore course racing.
- Well-known events such as the Fastnet Race, the Volvo Ocean Race, and the Around Alone Race are aided by modern day satellite navigation and other technologic advances.
- Sailing regattas have also been a part of the Olympic Games since 1900 and the Paralympic Games since 1992.
- Although professional sailors now dominate the sport in America's Cup, Grand Prix, and Volvo Ocean Race events, a vast number of amateur sailors continue to participate and enjoy the sport through international and national class championships, college and high school events, regional race weeks, and local club regattas.

The Team Physician and the Sport of Sailing

- Understanding the sport of sailing and the wide variations in the sailboat classes, events, equipment, crew positions, and physical demands is essential to caring for a sailing team.
- The specific needs of a team competing in the Volvo Ocean Race, which races offshore for weeks at a time but is able to communicate to a "home-base" hospital via satellite and requires an on-board medic to be a member of the crew, will be different than the needs of an Olympic sailing team racing on small dinghies and keelboats in day races.
- All require high-performance considerations and preparations for emergency management, but the individual event and crew demands will differ significantly.

Figure 75-1 America's Cup Yacht.

The Players

- The "players" in the sport of sailing are the sailors racing their boats in events governed by the international rules of the sport.
- From junior sailors to masters and grand masters, sailing is a sport played by all ages.
- Crews are often coed and open, but there are sometimes men's and women's divisions depending on the event and the boat class.
- Weight may be a limiting factor but there are no "weight classes"; it is used as a combined crew weight limitation that is suggested to equalize the boat's performance.

The Playing Field

- The sailboat racing "playing field" is a body of water—whether a river, a lake, or the open ocean.
- It is a constantly changing environment on which to hold a distance race or place a "course."
- Primary factors affecting the playing field include wind, waves, current, temperature, and all types of weather systems.
- The "race course" itself varies depending on the type of event. It may be around a fixed buoy course or on a course of longer distances, perhaps from one seaport to another or even from one part of the globe to another.

Rules

- Rules are essentially the same in sailboat racing regardless of where an event is being staged in the world though specific "National Prescriptions" may apply on a local basis.
- The Racing Rules of Sailing (RRS) are governed by the International Sailing Federation (ISAF), and there are slight variations to the basic RRS depending on the type of race or the event. For example, match racing rules are slightly different than fleet racing rules because of the difference in format.

- Though a jury is often present to interpret the rules in case of protests, sailing is considered a Corinthian sport in which the participants are expected to abide by the rules and take their penalties when appropriate.
- Many sailboat races are similar to race car driving; the boat that completes the "course" most quickly and crosses the finish line first, following the rules without infringement, wins.
- Some big boat races have a "handicapping" system requiring a rating such as a Performance Handicap Racing Fleet (PHRF) rating, where finish times are calculated based on the rating and distance of the race.
- There are also a few basic rules of the "road" or "waterway" that apply to sailing vessels whether competing or not.
  - Right-of-way rules apply in situations when boats meet, such as starboard/port and windward/leeward convergences.
  - Knowledge of basic sailing techniques, including the points of sail, steering the boat through a tack or a jibe, and maneuvering the boat safely, is essential for anyone who is learning to race sailboats.

Sport Classes

Types of Races

- Most common types are fleet races, match races, team races, and distance races.
  - Fleet races involve multiple boats starting together and racing around a course (Fig. 75-2).
  - Match races are done in "flights," pitting two boats against each other in a round robin format.
  - Team races involve teams of boats working together around the course to win a regatta.
  - Distance races are often from point to point and may even involve global circumnavigation.
- The events vary in duration; some are single-day events, others weekend regattas, race weeks, or even part of a longer series that occurs over a few months.
  - The America's Cup races are match races that involve series of races and an elimination ladder.
  - The Volvo Ocean race is an around the world race with many individual legs and cumulative times.
  - The Olympic and Paralympic races are a series of races with cumulative point totals.
- Races may involve many different types and sizes of boats competing against one another, each which has a "rating" or handicap assigned to it to even out the competition. Other races are "one-design" in which all the boats are exactly the same.

Figure 75-2 Rounding the Buoy.

Boat Classes and Crew

- There are many different types of boat classes—from the America's Cup class, which requires 16 crew members to operate, to the Volvo 70s, which require 13 crewmembers, to the Optimist dinghy class that junior sailors learn to race singlehanded.
- Number of crew or team members varies according to the boat type and the specific requirements of each individual class.
- The Laser is a single-person Olympic class boat that is sailed very competitively all over the world by all age groups—from juniors to great-grandmasters.
- Current Olympic sailing for 2012 will have 10 different disciplines, in which the boat class (equipment) may change from one "Games" quadrennial to the next.
- The Paralympics has three boat disciplines—single-person, two-person, and three-person boats.
- For the 2008 Olympic Games the boat classes for the 11 events selected were the Star, the Yngling, the 470 (men's and women's), the Tornado, the 49er, the Finn, the Laser, the Laser Radial, and the Windsurfers (men's and women's). In the Olympic Games 2012, the Yngling will be replaced by the Elliot 6M and will be a women's match race event, and the Tornado/multihull event has been dropped. For the Paralympic Games, the three classes in 2008 and 2012 are the Sonar, the SKUD 18, and the 2.4mR.
- Examples of other boat classes that are very well known and participated in widely include J-24s, Etchells, Lightnings, Snipes, Melges 24's, Farr 40's, and TP-52's.

Equipment and Skills

- Basic equipment includes a boat with a mast, sails, a keel or centerboard, a method to trim the sails to the wind, and a steering mechanism or helm.
- Though some boats may be very complicated with extremely advanced technology, satellite navigational systems, and carbon fiber rigging, most classes of sailboats control and limit the allowed equipment in some fashion.
- Many major one-design regattas require sail measurement and boat weigh-in, and some also require crew weigh-in on a daily basis.
- Variations in sailing equipment exist depending on the type of boat sailed—whether it is a two-person dinghy that requires hiking straps, a multihull with trapeze capabilities, or a 10-person keelboat with large "coffee grinder" winch handles for trimming sails—knowledge of the specific class is essential for proper care of the sailor.
- Skill sets required to competitively race sailboats at an advanced level are also variable, but include helming (whether on a tiller or a wheel), trimming the sails (either on block systems or large winches), hoisting and dousing different types of sails, spinnaker pole/system management, rigging the boat and making fine-tune adjustments, hiking or trapeze work, maneuvering the boat with speed and agility, and navigation of the vessel.

Protective Gear

- Sailor-athletes often requires protective equipment based on the environmental conditions in which they are sailing; for example, cold wet weather requires proper foul weather gear, and offshore distance racing requires safety harnesses, lifejackets, etc.
- Extreme sailing through the intimidating Southern Ocean around Cape Horn, for example in the Volvo Ocean Race, may require dry suits and head protection in addition to harnesses and other safety equipment due to high winds, volatile weather systems, and floating icebergs. On the other hand, a youth sailor racing a 420 dinghy in a summer regatta may only

need a bathing suit, hiking shorts, sailing gloves, boat shoes, a hat, some sunscreen, and a lifejacket.
- Constant debate occurs over the "requirement" of wearing lifejackets/personal flotation devices (PFDs) in regattas.
  - For the most part, the decision to wear PFDs is primarily the responsibility of the sailor, though the race committee or the regatta organizer may make it a mandatory requirement based on factors such as temperature, weather, and sea state conditions.
  - Because there are many documented instances of capsizes, even in favorable conditions, in which a lifejacket saved a life, use of PFDs is encouraged (see Fig. 75-3).

Safety

- Safety is a big issue because the marine environment can be a hazardous playing ground.
- From dense fog to crashing waves, an understanding of the body of water/geographic area on which a competition occurs is essential.
- Along with emergency management plan complete with contact information and appropriate seamanship skills, proper prevention of injury and illness are also required elements of safety in this sport.
- Understanding of right-of-way rules and avoiding collisions, knowledge of navigation signals, proper training, practice of man overboard rescue drills, and overall seamanship are all important for safe participation in sailing.

SAILING SPECIFIC MEDICINE

Epidemiology

- Efforts to advance evidence-based knowledge of sailing and sports medicine have been demonstrated over the last 15 years with an increase in the number of studies of various aspects of sailing.
- Interest in research by groups such as Olympic and America's Cup teams has produced many beneficial changes.
- With hundreds of boat classes, each having specific crew positions and demands, there are distinct injury profiles, optimal training regimens, and individualized physiologic stresses and demands that have been described.

Physical Demands

- Vary with boat class and crew position; injuries may differ on the basis of the specific job stressors.
- Some injuries appear to be more related to overuse dynamics, yet others may be more acute due to accidents.
- Many actions in sailing are sudden and sporadic, which may place the sailor at high risk while performing explosive, powerful moves, particularly during a tacking or jibing maneuver.
- Constant isometric contractions required during hiking on a long upwind leg add significant strength and endurance components to the sport.
- Whether the technique of straight leg hiking or droop-seat hiking, sailors may be susceptible to back and knee problems (Fig. 75-4).
- Repetitive grinding on winches while trimming can result in shoulder and arm injuries.
- Inherent postures in many crew positions also play a role in musculoskeletal issues, because some actions on a sailboat require twisting and hyperextension of joints and in particular may predispose a sailor to back injuries.
- Lifting poses a particular risk, with difficulty maintaining proper form on a moving vessel or using proper technique even on the docks.

Injury Studies

See Table 75-1 and Figure 75-5.

Environment and Illnesses

- Illnesses have been prevalent amongst sailing teams working closely on a day-to-day basis, and proper hygiene always needs to be considered—including not sharing water bottles.
- Because many elite sailors often travel internationally to events, consideration of water and food contamination is also essential.
- Environmental issues such as hypothermia, heat illness, dehydration, seasickness, poor nutrition, and sun-related problems are also prevalent within the sport of sailing.
- Water immersion, near-drowning, contact with aquatic marine life such as jellyfish, and other water-based emergencies are also obvious risks at sea.
- Special consideration for the amount of time on the water should be taken in regard to this unique field of play environment, and necessitate adequate thermoregulation with cooling/heating units, hydration kits on and off the water, and sun protection (Fig. 75-6).
- Because most events require sailors to be on the water for extended periods of time without access to land-based facilities, consideration of gastrointestinal issues, bladder problems in some of the disabled sailors, diabetic/blood sugar management, adequate nutrition, and the ability to readily manage minor emergencies on the water is necessary.
- Illness and environmental influences may impact health care within the sport of sailing; thus appropriate prevention in-

Figure 75-3 Laser Sailor in Personal Flotation Device (PDF) and Foul Weather Gear.

Figure 75-4 World Champion Yngling Team Hiking Out.

Table 75-1 COMMON AREAS OF INJURY/PAIN FOR SAILORS

Smaller boats Olympic classes	Lower back (52.9%) Other back areas (41.2%) Knees (25%-32%) Right thigh (26.5%) Neck (23.5%) Right shoulder (23.5%) Forearm or elbow (20.6%)
Larger boats America's Cup class	Lumbar spine (16%) Shoulder (16%) Knee (10%) Cervical spine (8%) Hand (7%)
Offshore endurance races (Volvo Ocean boats)	Low back Shoulder Neck Skin lesions
Disabled sailors (Paralympic classes) Boardsailing (Windsurfers)	Upper extremity (60%) Spine (20%) Lower extremities (44.6%) Upper extremity (18.5%) Head and neck (17.8%) Trunk (16.0%)

Data from Moraes J, Nery C, Fontel E, et al: Multidisciplinary assessment of the Brazilian Olympic sailing team. In Legg SJ (ed): Human Performance in Sailing Conference Proceedings: Incorporating the 4th European Conference on Sailing Sports Science and Sports Medicine and the 3rd Australian Sailing Science Conference. Palmerston North, New Zealand: Massey University, 2003, pp 92-95. Allen JB: Sports medicine injuries in the America's Cup 2000. In Legg SJ (ed): Human Performance in Sailing Conference Proceedings: Incorporating the 4th European Conference on Sailing Sports Science and Sports Medicine and the 3rd Australian Sailing Science Conference. Palmerston North, New Zealand: Massey University, 2003, pp 45-46. Rosenbaum DA, Dietz TE: Windsurfing injuries: Added awareness for diagnosis, treatment, and prevention. Physician and Sportsmedicine, 2002. Legg SJ, Smith P, Slyfield D, et al: Knowledge and reported use of sport science by elite New Zealand Olympic class sailors. J Sports Med Physical Fitness 213-217, 1997. Spalding T, Malinen T, Allen JB, et al: Analysis of medical problems during the 2001-2002 Volvo Ocean Race. In Legg SJ (ed): Human Performance in Sailing Conference Proceedings: Incorporating the 4th European Conference on Sailing Sports Science and Sports Medicine and the 3rd Australian Sailing Science Conference. Palmerston North, New Zealand: Massey University, 2003, pp 47-50. Rodriguez R: Medical dispatches from the Whitbread Sailboat Race. Physician and Sportsmed. Online submission May 1998.

cluding proper clothing, adequate nutrition, and safety measures are recommended.

Physiology of Sailing

- Research addressing the biomechanics and physiology in the sport of sailing has been recently increasing as more scientists are analyzing the performance of the sailboat racer.
- In general, the studies suggest that the dinghy classes, which require more intense dynamic hiking maneuvers, and the big boat crew positions, that require the most agility or repetitive motion, are the most physiologically demanding of the sport.
- One study of the 2002 Danish Olympic team showed Laser sailors have shown the highest $\dot{V}O_2$ max, at 58.3±4.2 mL/kg per minute whereas the Finn and Star sailors were lowest with an average value of 47.6±3.5 mL/kg per minute. Helmsmen and crew on trapeze boats have had results of 55.3±4.0 and 57.3±3.7 mL/kg per minute, respectively.

Biomechanics of Hiking

- The physical stress of hiking, using body weight to counterbalance the tilting forces of the boat, has been documented and is believed to play an important factor in performance.
- Other recent studies have recognized hiking as a dynamic activity, measuring physiologic responses and force demands within an active sailing environment, whether on a simulator or on the water.

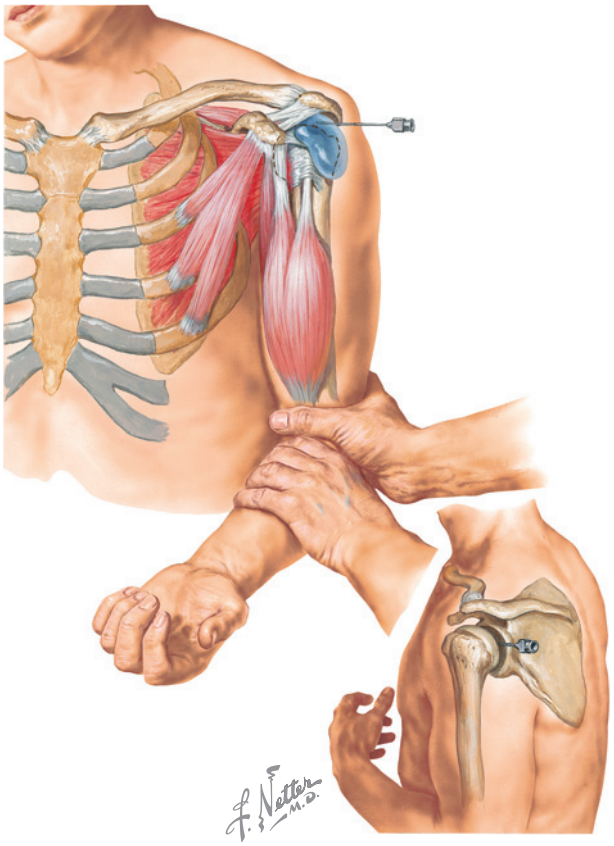


Figure 75-5 Injuries: Grinder's Shoulder May Require Injections.

Figure 75-6 Cooling Vest.

- One study found elite status and national rankings of Laser sailors were strongly correlated to quadriceps maximal voluntary contraction (MVC), isometric endurance, and tolerance of muscular fatigue (Fig. 75-7).
- Improper technique is commonly a factor in hiking-induced knee pain, resulting in unbalanced muscular forces around the knee joint.
- With fatigue, most sailors tend to isolate the vastus lateralis, leading to patellofemoral pain; turning out both feet with the legs extended increases the workload of the vastus medialis.
- Tight toe straps and plantar flexing the foot may help straighten the knees, centralize the force of gravity, and reduce the effort required by the quadriceps.

Physical demands of hiking on a Laser.

Paralympic amputee hiking on a Sonar in Sydney Games.

Figure 75-7 Biomechanics of Hiking.

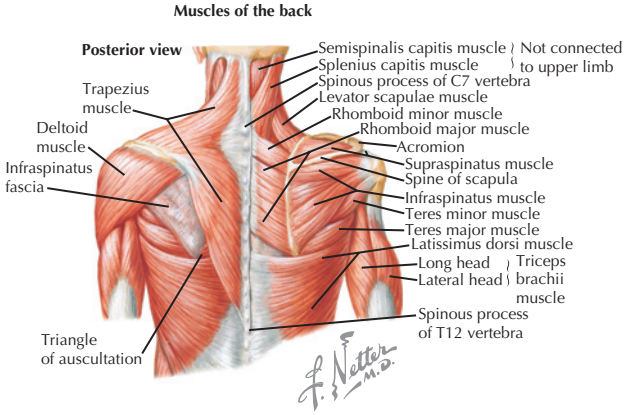
- The biomechanics of hiking in Paralympic sailing may change the dynamics, due to different force couples when using prosthetic legs for example, and should be considered when working with athletes with disabilities (see Fig. 75-7).

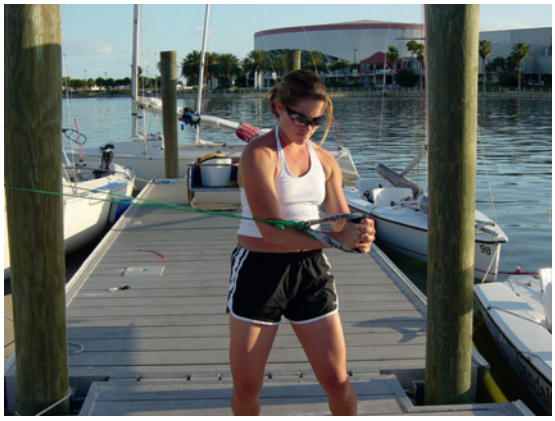
Training for the Sport

- With physical fitness joining boat speed and tactical intelligence as the main determinants of sailing performance, the issue of fitness training for sailors is accompanied by many opinion articles, and very little scientific research.
- Various boat classes, crew positions, racing conditions, and baseline fitness levels make it unfeasible to provide a uniform recommendation for fitness requirements.
- Strength, power, muscle endurance, cardiovascular fitness, weight management, flexibility, and agility all play varying roles in sailors' training regimens.
- Agility exercises may improve hand-eye coordination and the efficiency of movement about a sailboat.
- Weightlifting routines should involve commonly used muscle groups plus their antagonists to maintain proper balance of strength; this should include a core workout.
- Aerobic training and fitness has been directly related to sailors' reaction speed to wind shifts, as well as enhanced endurance, decision making, and concentration, particularly in the later stages of races.
- Physical and mental recovery between races and regattas also may improve with cardiovascular fitness.
- Position specific fitness training:
  - Hiking should include muscular strength and endurance of the core and the lower extremities, maintaining balanced force and flexibility about each joint.
  - Sailors who hike rely on muscle groups in the thighs, abdominals, hips, and arms, whereas sailors on a trapeze may focus more on upper body strength and endurance, aerobic endurance, and agility.
  - Grinders and many other big boat sailors should address aerobic endurance as well as muscular strength, power, and endurance, particularly in the upper body.
  - Board sailors require sustained isometric action of the pectoralis major, deltoid, and scapular stabilizers; therefore training should involve the shoulder girdle (Fig. 75-8).
  - Sail trimmers (any boat size) should focus on training of the arms, shoulders, and upper back (see Fig. 75-8).
- Timing of fitness training is important, whether that entails maintaining fitness in the off-season or reducing heavy training loads prior to regattas.

Nutrition and Weight

- Weight management issues for sailors commonly involve reaching a perceived ideal mass for racing a small boat or meeting weight limits of a one-design class.
- Weight loss should be gradual, focusing on reduced caloric intake while increasing aerobic exercise.
- Awareness of problems associated with the female athlete triad in women sailors is also critical, because they are often required to "make weight," which, as in many sports, can lead to eating disorders (Fig. 75-9).
- Whether sailing in a 1-day regatta or a long-distance offshore race, adequate nutrition to sustain proper blood glucose levels throughout the event is important for maintaining concentration and coordination.





Trimmer demonstrates thera-band shoulder exercises on the docks.

Figure 75-8 Training for the Sport.

Figure 75-9 Olympic Women's Keelboat Yngling Class Has Weight Limits.

- America's Cup sailors have been found to average 56 kcal per kg of body weight in daily energy expenditure.
- Dehydration can hamper performance, including increasing cognitive impairment and increasing risk of injuries.
- General guidelines for hydration in sports apply to sailors.

Sports Psychology

- In recent years, there has been a growth in understanding the importance of mental skills training, especially in high-level competition.
- Relaxation techniques, prerace routines, and mental rehearsal may be beneficial to sailors at any level.
- Sports psychology can facilitate teamwork, focus, and organization.
- Using sports psychology professionals as part of a performance enhancement team is essential to the health and wellness of the athlete and crew.

Injury Prevention

- With all of the different skill sets in mind, attention to proper mechanics, technique, posture, and positioning will assist the clinician in injury prevention.
- Injury prevention is best addressed through appropriate fitness training and properly caring for prior injuries.
- There is limited research to support injury prevention programs in sailing; however, it may be effective to include flexibility, hip flexor mobility, and core stability programs to reduce injury risk.
- Acute injuries are often related to accidents involving the boat equipment and are difficult to prevent (e.g., lacerations from hitting sharp metal edges on the rig, abrasions from mishandling sheets, finger fractures from problems with winch overrides, contusions from free flying winch handles, concussions from contact with an accidentally jibing boom, or slips and falls on a wet foredeck).
- Addressing ergonomic developments, particularly in big boat design, also holds potential for injury prevention.
- Certainly avoidance of accidents is preferable, but overuse injuries may be more easily addressed with certain considerations.
- Returning to play is dependant on the nature of injury and the physical demands for which the athlete is returning to sail.

Crew Positions and Specific Injury Examples

- **Helm:** Carpal tunnel syndrome (CTS) can be seen in both big boat sailing and dinghy sailing. Symptoms of CTS may be preventable by adjusting the grip width of the wheel at the helm of a larger yacht; avoiding a prolonged tight sustained grip position on the tiller may also decrease stress at the wrist and forearm in a dinghy (Fig. 75-10). Platforms on distance racing for the helmsman have recently been used on the large yacht in the Volvo Ocean Race to properly position the sailor's posture on long tacks, because standing on a heeled boat in


Helm on a Maxi-Yacht.


Grinding on pedestal winches aboard Stars and Stripes.


The Bowman lives dangerously up the rig.


Trapeze sailors using a harness.


Hiking straps and foot position may impact knee injuries.

Figure 75-10 Crew Positions and Specific Injury Examples.

one place for a prolonged time period can lead to overuse problems for these drivers.

- **Grinders:** Attention to the grip size and angle of rotation on winch handles, and to the height and angle of pedestal winches, may also help prevent other overuse injuries such as lateral epicondylitis, often referred to as "grinder's elbow" (see Fig. 75-10). Lumbar spine injuries also frequently affect the grinder position, and the biomechanical forces required for this skill set should be considered. These athletes must be assessed on an individual bases, because the height of the sailor, for instance, may directly impact both potential injury prevention and performance of peak power output on a pedestal winch if the height of the pedestal is not adjusted appropriately.
- **Mast:** The mast man on a larger yacht is also susceptible to injuries. Repetitive halyard hoisting at the mast with proper hand-over-hand technique using the legs and trunk for strength and stability may reduce overhead injuries to the shoulder.
- **Trimmer:** "Trimmer's neck" has been described as neck pain related to the angle of cervical rotation and extension required to visualize the sail while constantly monitoring the tale-tells. Spinnaker trimmers may be especially susceptible to such problems because of the constant attention aloft that is required to fly this specialized sail.
- **Bowman:** One of the most frequently injured crew members on a sailboat is the bowman, primarily because of the multiple physical tasks required by this position (see Fig. 75-10). Safety is essential for the bowman, who may be changing head sails in heavy weather and sea conditions or jibing the spinnaker pole when running downwind. Concussions have been reported in this position from contact with the spinnaker pole, and many bowmen must be clipped on with a safety harness in order to avoid falling overboard in heavy sea conditions.
- **Hiking crew:** Hiking is often required in heavy air conditions no matter what size boat is being sailed, but there is a difference in hiking out while sitting on a rail with a lifeline on a J-24, and hiking on a Laser. Dinghy sailing often requires a dynamic effort in heavy air conditions and will vary with the sailor and boat class. The hiking strap placement and foot position on the straps have been shown to be factors in forces generated at the knee and should be observed in any sailor with knee pain (see Fig. 75-10). The different hiking styles and the amount of torque placed on the knee when tacking the boat from a hiked out position may impact development of meniscal injuries or patellofemoral injuries in these athletes. Hiking crew members are also susceptible to lumbar spine disc problems and core stability is essential to preventing injury. In some boats that use a trapeze, the harness fit may play a role in preventing low back pain, suggesting that custom-fit harnesses should be considered (see Fig. 75-10). Finally, the hiking crew may also be susceptible to "rail rider's rump," or cutaneous rash, which can be worsened with unclean wetsuits or hiking shorts.
- **Shore crew:** Unlike other sports, most sailors also have shore-based responsibility for the boats on which they race, and care when lifting their boats onto trailers or moving large heavy sails and equipment on and off the larger vessels is essential to prevent lumbar strains and other injuries. However, as in other sports, some injuries occur during fitness training that can also impact performance on the water and should be monitored adequately.

Boardsailing

- Because boardsailing is slightly different in its rules and techniques, the injuries may differ from other classes in the sport.
- The constant pumping action of the sail by the board sailor often causes increase in forearm compartment pressures, sometimes relieved with daily massage and flushing.
- In addition the newer segment of kite-boarding will have its own unique injuries, with problems including lofting and wind-sheer dropping.

SUMMARY

- With all of this knowledge about the sport of sailing in mind, a team doctor's handbag for sailing will otherwise not differ from many other sports.
- Injuries and illnesses will occur and they will vary with the type of boat, the event, and the crew position.
- Finally, as with most teams, caring for the athlete on and off the field of play is essential to a healthy team.
- Prevention, whether through proper nutrition, adequate hydration, strength and endurance training, cardiovascular fitness, injury assessment, mental preparedness, environmentally appropriate clothing, or safety awareness, is the cornerstone to a healthy sailing team and enhancement of the team's performance.

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Rock Climbing

76

Charles S. Peterson

GENERAL PRINCIPLES

Climbing Overview

- Climbing requires endurance, strength, and agility.
  - Athletes must be in good cardiovascular shape to climb.
  - Climbing-specific training reduces injury and enhances ability.
- Safety and equipment advances have made climbing much safer.
  - Climbers must maintain and properly use equipment to optimize safety.
  - Environmental hazards can greatly increase danger.
- Rapid growth in popularity and access in recent decades.
  - According to the Outdoor Recreation Resources Review Commission in 2007, the number of recreational climbers increased from 7.3 to 9.2 million over the last decade.
  - Development of local climbing facilities and clubs continues.
    - Climbers starting at younger ages
    - Year-round climbing
  - Awareness in public eye, development as a competitive sport, events such as X-Games.

Types of Climbing

Free Climbing (Traditional Rock Climbing)

- Natural rock feature climbing (Fig. 76-1)
- "Pro" (protection) used to affix rope to the rock at intervals
  - Passive: nut, hex (see Fig. 76-2, Passive protection)
  - Active: cam (see Fig. 76-2, Active protection)
  - Aid climbing: climbing on placed or fixed pieces
- Climbers ascend in "pitches"
  - Typically 20 to 50 meters per pitch
  - Gear cleared and ropes carried with climbers for placement on ascent of next pitch
  - Belaying (see "Terminology" section) of climbers from one pitch to the next
  - Climbers rappel (see "Terminology" section) down from the top, controlling their own descent on rope
- Minimal impact on the environment

Sport Climbing

- Outdoor (see Fig. 76-1)
  - Established routes, ratings, and maps
  - Preexisting, fixed safety features
    - Bolts: anchored into rock at intervals of 2 to 4 meters, used to provide protection on the way up to the top of the climb, carabiners clip onto bolts
    - Chains: chains or welded loops anchored into rock at the top, rope runs through
    - Essential to assess for stability and safety; may be old or unstable
- Indoor
  - "Rock gyms"
  - Excellent technology and safety
  - Exercise and sport
  - Ability level pushed in safe environment
  - Risk for overuse injury
- Dynamic, or "dyno"
  - Sport competition
  - Rapid, lunging, and leaping ascents

Mountaineering/Alpinism

- Historical mountain climbing, or ascent of peaks
  - Evolution as a sport in 18th and 19th centuries
  - Major world peaks climbed by the 1960s
- Difficult access, often requires travel, may entail multiple day adventure, variable difficulty levels and techniques from hiking to rock and ice climbing
- Variety of injury risks
  - Serious injury or death from falls
  - Acute and chronic musculoskeletal injuries, most commonly to hands and extremities

Free climbing, crack climber.

Outdoor sport climb, lead climber.

Bouldering.

Canyoneering.

Ice climber.

Figure 76-1 Types of Climbing. *(Figures courtesy of Matt Turley.)*

- Illness or injury related to environment, remoteness
- Minor injury can lead to death in remote locale
- Environment-related
  - Hypothermia
  - Altitude sickness
  - Falling rocks

Bouldering

- Indoor and outdoor (see Fig. 76-1)
- Unroped climbs that typically have a maximum climb height of up to 3 to 4 meters
- Traverses and overhangs create "problems," or short climbs repeatedly attempted until successful
- Spotting and mats provide adequate protection
- High demand on upper extremities; risk of overuse, especially to fingers
- Risk for short falls on mats in climbing shoes; potential for ankle, foot, and knee injuries
- Indoor and outdoor climbing with similar risks

Rappelling

- Descent from cliff using figure-eight or other device and static or dynamic rope (see Fig. 76-3)
- Rappeller controls rate of descent with friction, holding slack rope behind back
- Use of gloves helps prevent friction burns
- Downhill partner holds end of rope to pull and increase friction to stop rapeller in danger of excessively rapid descent
- Second safety rope can be attached with belayer at top or bottom of cliff

Canyoneering

- Southwest United States
- Canyons: narrow riverbeds or channels often with steep cliffs on either side
- Slots: tight trenches cut deep into sandstone by running water over centuries, limited access, limited escape routes
- Combination of hiking, rappelling, climbing, swimming, camping, exploring (see Fig. 76-1)
- Remote locale and flash floods can present unique dangers

Spelunking

- Exploration of caves.
- More experienced explorers often refer to themselves as "cavers" and their less experienced counterparts as "spelunkers."
- Requires horizontal and vertical movement through caves, extensive use of ropes, pitches, crawling, squeezes through narrow openings.
- Guides and knowledge of terrain essential.
- Exploring can require digging or diving.
- Light source, specialized equipment, and redundant safety measures required.

Solo Climbing

- No ropes used
- High level of difficulty
- Extreme danger and risk of death, elite level climbers

Ice Climbing

- Unstable surface
- Injury risk from cold environment and falls
- Specialized equipment needs (see Fig. 76-1)

Buildering

- Climbing of man-made structures
- High technical skill required
- High risk of falls, typically fatal, unprotected usually
- Generally illegal

Terminology

Belayer: A climber at the bottom or top of a climb who controls the rope with a belay device, providing protection if the climber falls.

Top-rope: The rope is placed through chains or metal loops attached to the top of a climb; higher level of safety, alleviating the need to place protection.

Lead climber: Climber elevates attached rope while climbing, periodically "clipping-in" to protection; higher risk and difficulty. Subsequent climbers then climb with top rope.

Communication

- Climbers use standard phrases to initiate climb.
  - Climber asks, "On belay?" Belayer responds, "Belay on."
  - Climber states, "Climbing." Belayer responds, "Climb on."
- Good communication enhances safety.

Difficulty Rating Systems

- Systems vary by country
- U.S. climbing system ranges from 1 to 6:
  1. Walking
  2. Hiking
  3. Scrambling, occasional use of hands for balance
  4. Climbing with arms required, many climbers use protection with a rope
  5. Technical rock climbing
     - Rope and equipment required.
     - Developed in the 1930s by the Sierra Club, initially rated 5.0 to 5.9.
     - As climbing techniques and equipment improved, numbering system adapted.
     - Climbs typically rated 5.5 to 5.14; a, b, c, d designations given from 5.10 to 5.14.
     - Numbering system may vary by region or even area.
  6. Not climbable with hands and feet alone; aid required
- Moderate climbs rank 5.6 to 5.9, advanced to expert from 5.10 to 5.14
- International rating systems vary by country
  - German: 5 to 11, ±
  - British: seriousness rating "vs" to E9 followed by difficulty rating 4 to 7, a to c modifier (e.g., E7 6c)
  - Australian: 16 to 33
  - French: 5 to 9, a to c

Equipment and Safety

- Rope
  - Kernmantle, or jacketed-core nylon, woven sheath over braided core
  - Dynamic, stretches to lessen the impact of falls
  - Dry ropes are specially treated to repel moisture, because water makes ropes weaker
  - Nondry ropes are less expensive and suited for dry environments only
  - Static rope with little stretch better for rappelling, stronger and less supple
  - Impeccable care required with use: inspection for damage, protection from dirt or stepping upon, and dating
    - Retire in 4 to 5 years, even with light use
    - Retire sooner with damage or heavy use or long fall
  - Length choices include 50 m, 55 m, 60 m, up to 70 m
  - Diameter ranging from 7.5 mm to 11 mm
  - Typical length of 60m, diameter 10 to 11 mm
  - Ropes rated by diameter, number of falls
- Harness (Fig. 76-2)
  - Proper fit and sizing, manufactured harness
  - Inspect for damage
  - Inspect each other to ensure proper fit and use


Harness.


Climbing shoes.


Climbing gear.


Active protection.


Passive protection.


Quick draw.


Carabiner and ATC.

Figure 76-2 Climbing Equipment.

- Shoes (see Fig. 76-2)
  - Glovelike and tight-fitting, but should be bearable; smaller than normal shoe
  - Creates a "hoof effect" of the foot to provide strength and grip, protection from the rock
  - Shoe types range from general use to slipper-like supple shoes for indoor and bouldering to stiffer boots for more vertical, outdoor climbing
  - Experts often have a "quiver" of shoes
- Belay device
  - Controls rope, protection from falls by feeding rope in and out
    - Belayer controls for climber
    - Rappeller controls for self
  - Technique essential for proper use
  - Multiple brands and styles available
    - ATC: "air traffic control"
    - Grigri: autolocking belay device
    - Figure-eight
- Surface
  - Indoor, outdoor
  - Type of rock, condition
    - Loose rock
    - Plants, insects introduce hazard
- Protection (see Fig. 76-2)
  - Active: cams with spring loading
  - Passive: chocks, nuts, hexes, tapers
  - Removal tool
- Carabiner (see Fig. 76-2)
  - C-shaped with hinged closure of ring
  - Provides attachment to harness, bolt, rope
  - Locking provides more safety
- Quick draw (see Fig. 76-2)
  - Two carabiners attached by webbing, used to attach rope to bolt while climbing
- Chalk
  - Held in pouch behind back to use for hand friction, moisture reduction
- Webbing, cord, slings
  - Used to carry gear, attach pro, or create a top rope with a carabiner by tying onto rock, tree, or other solid structure
- Helmet
  - Often overlooked, essential equipment
  - Should also be worn by belayer to protect from falling rocks

Biomechanics, Training, and Physiology

- Training variables more important than anthropometric determinants
- Lean, strong, low body fat, flexibility create more successful climber
- Girls make more natural climbers, using legs and hips more than arms (Fig. 76-3)
- Energy expenditure
  - Outdoor greater than indoor
  - Metabolic Equivalent of Task (METs) less than running at same heart rate
  - $\dot{V}O_2$ max plateaus with climbing
  - Handgrip fatigue lasts 20 minutes

Figure 76-3 Rappelling from Multiple Pitch Climb.

Epidemiology and Injury

- Type of climb
  - Indoor safer, more controlled
  - Proper use of pro, condition of bolts and chains
  - Equipment condition, inspection
  - Risk for death and serious injury greater with mountaineering (Table 76-1)
  - 3.1 injuries per 1000 climber hours with sport competition (Table 76-2)
- Skill level
  - Higher risk in elite climbers and higher difficulty climbs
    - More overuse injuries to upper extremities
    - Risk of death higher in more exposed environments
  - Most injuries, however, occur at or below climber's usual level
  - Inexperienced climbers experience lower extremity abrasions and lacerations
  - Greater risk in male climbers who climb harder routes, have been climbing more than 10 years, and lead climb more than top rope
- Environmental
  - Falling rocks
  - Isolation
  - Weather, sun, cold
  - Altitude
  - Lack of preparation
  - Plant and animal/insects (e.g., bees)

CLIMBING INJURIES BY ANATOMICAL REGION

Upper Extremity

- More commonly overuse injuries
- Shoulder
  - Rotator cuff tendonitis, tear, impingement
  - Rupture long head biceps
  - SLAP (superior labrum, anterior to posterior) lesion
  - Acromioclavicular (AC) sprain
  - Dislocation
  - Muscular strain: rhomboid, latissimus dorsi, lower trapezius

Table 76-1 ACCIDENTS AND INJURIES IN NORTH AMERICAN MOUNTAINEERING AND ROCK CLIMBING

Year	Accidents reported	Injured	Fatalities
2005	111 USA 19 Canada	85 USA 14 Canada	34 USA 7 Canada
1951-2005	6111 USA 958 Canada	5158 USA 715 Canada	1373 USA 292 Canada

Adapted from Williamson JE: Accidents in North American Mountaineering. Golden, CO: American Alpine Club, Inc., 2006, pp 97-98.

Table 76-2 2005 WORLD CHAMPIONSHIPS IN ROCK CLIMBING INJURY RISK

Year	2005
Climbers	443
Countries	55
Climbing days	520
Acute medical issues	18
Serious medical issues	4 (zero deaths)
Injury rate per 1000 climbing hours	3.1

Adapted from Schoffl VR, Kuepper T: Injuries at the 2005 World Championships in Rock Climbing. Wilderness Environ Med 1(3):187-190, 2006, used with permission.

- Elbow
  - Anterior
    - Climber's elbow: brachialis strain (see "Climbing-Specific Injuries")
    - Distal biceps strain or tear
    - Posterior interosseous nerve compression
  - Medial
    - Forearm flexor strain, overuse: medial epicondylitis
    - Pronator teres strain or tear
    - Ulnar nerve compression
  - Lateral
    - Extensor strain: lateral epicondylitis
    - Radiocapitellar compression
  - Posterior: triceps tendonitis
- Wrist
  - Tendonitis/tenosynovitis
    - Flexor carpi ulnaris most common
  - Sprains
  - Carpal tunnel syndrome, tendon hypertrophy, edema; 10% to 25% of elite climbers
  - Triangular fibrocartilage complex (TFCC) injury
  - Retinacular injury
- Hand
  - Climber's finger: flexor pulley injury (see "Climbing-Specific Injuries")
  - "Sausage fingers"
    - Joint effusion
    - Synovial irritation, cartilage damage
  - Flexor digitorum superficialis (FDS) and flexor digitorum profundus (FDP) tendonitis, strain, tear, laceration (Fig. 76-4)
    - Tenoperiostitis, tears at insertion
    - Loads on FDS with FDP at mechanical disadvantage with certain grips
  - Collateral ligament injury
  - Flexion contracture
  - Stress fracture
  - Epiphyseal fracture
  - Nodule/trigger finger (see Fig. 76-4)
  - Dupuytren's
  - Ganglion (see Fig. 76-4)
  - Mallet finger
  - Lumbrical tear/lumbrical shift (see Fig. 76-4)
  - Amputation
- Hand grips affect injury patterns (Fig. 76-5)
  - Open grip: strain to FDP more than FDS, easier on joints, ligaments, and tendons
  - Crimp (with closed ring): strain on FDS, distal interphalangeal (DIP) joint and volar plate
  - Pocket grip: strain to FDP, collateral ligaments
  - Vertical grip: strain to FDS
  - Pinch grip
  - Finger locks
  - Hand stacks
  - Hand and fist jams

Lower Extremity

- Hip
  - Adductor strain: wide bridging between foot holds
  - Strain to quadriceps or hamstring
- Knee
  - Meniscus tear: frog position with knee flexion and external rotation, pushing upward
  - Anterior cruciate ligament (ACL) tear: related to fall from bouldering height
- Ankle
  - Sprains more common than fractures

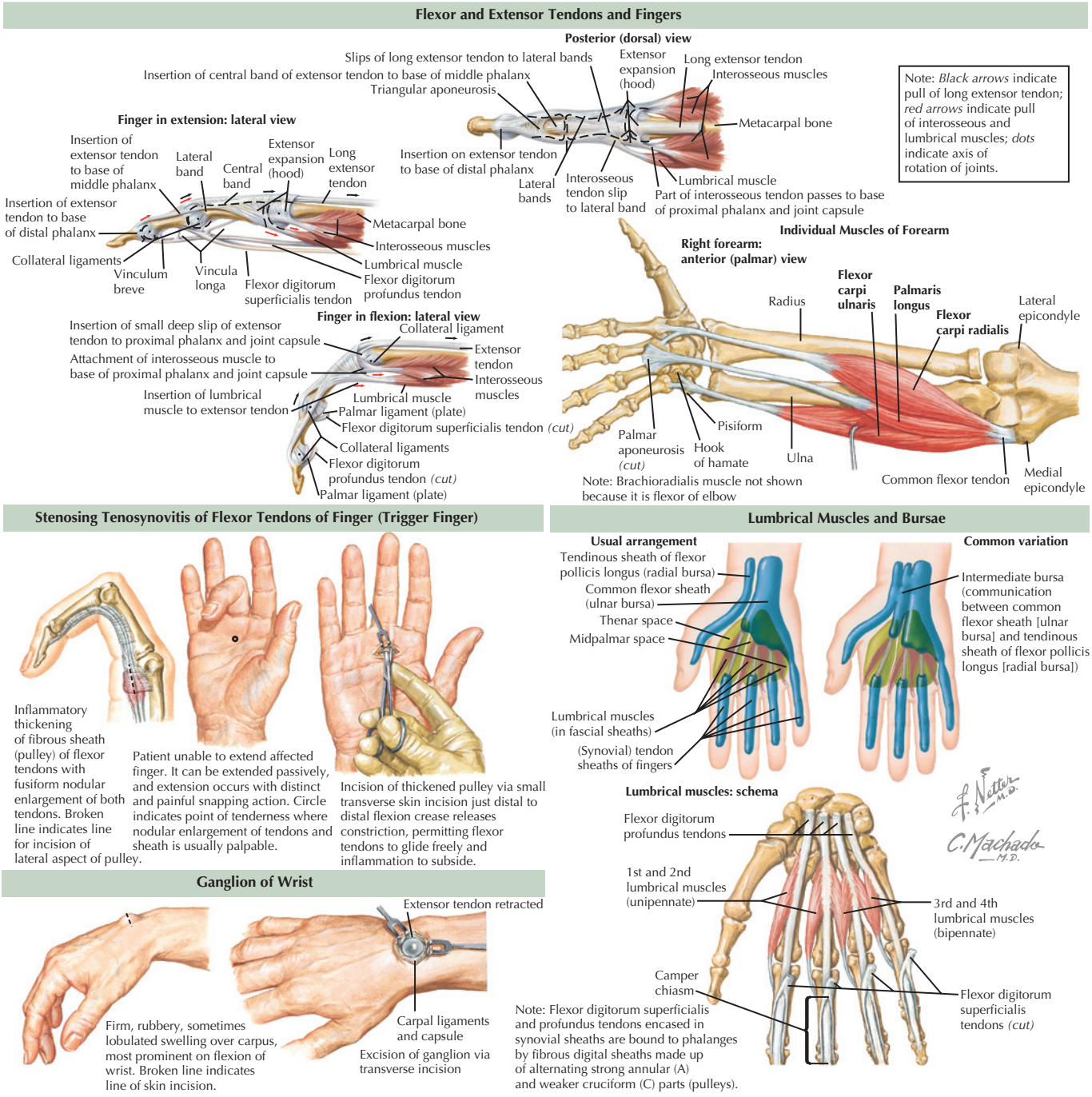


Figure 76-4 Upper Extremity Climbing Injuries.

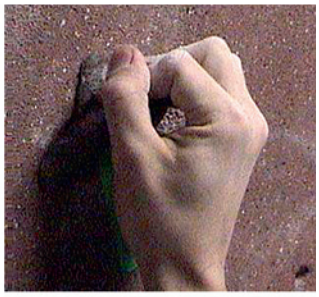
- Foot supinated in small shoes (dorsiflexion, plantarflexion-inversion)
- Bouldering falls from up to 3 meters, reduced risk with adequate mats
- Falling into climbing surface or tangling into ropes on overhang

- Foot
  - Neurologic symptoms of numbness, tingling from tight shoes
  - Hallux valgus common in climbers with 5 or more years' experience
  - Blisters, toenail contusions/loss

Spine/Abdomen

- Cervical: strains and degenerative changes
  - Falls, overhang extension
  - Belay position, looking upward in extension
- Lumbar: strains and disc injury, degenerative changes
  - Falls, impact
  - Repeated stress from falls in harness, hyperextension
  - Pectoralis imbalance, causing thoracic kyphosis and lumbar lordosis
- Abdominal: strains, contusion
  - Sustained in traverses, reaching


Open.


Crimp (closed ring).


Pocket.


Vertical.


Finger jam.

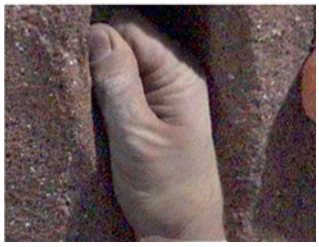

Fist jam.

Figure 76-5 Common Hand Grips.

- Falls and harness can contribute to strains, contusion, but less likely when harness properly fitting and applied

Head

- Impact
  - Falls, concussion
  - Rocks from above
  - Helmet for climber and belayer
- Eye
  - Hyphema from falling rocks or rope whip
  - Eye protection for belayer essential

Skin

- Abrasion: "climber's rash" to lower extremities of less experienced climbers
- Laceration
- Sunburn

CLIMBING-SPECIFIC INJURIES

Climber's Finger

Description: Uniquely climber's injury to the flexor pulley system. Injuries to A2, A3, or A4 flexor pulleys; multiple pulley injuries more serious (Fig. 76-6). Injury of A2 flexor pulley most common. Acute or chronic overuse. Hand grips and technique contribute to development of pulley injuries and other finger injuries. Accounts for 30% of finger injuries; half of elite climbers have some degree of pulley injury. At 90 degrees, pulley sustains more tension than the tendon. Grading of the injury very important (Table 76-3).

Presentation: Climber often presents with history of "pop," swelling, pain. May present with long-standing injury, chronic pain.

Physical examination: Bowstringing of the affected digit with acute pain and swelling. Most commonly affects the fourth digit of nondominant hand (see Fig. 76-6).

Diagnostics: Magnetic resonance imaging (MRI), ultrasound. Dynamic ultrasound 98% sensitive, 100% specific; user dependent (see Fig. 76-6).

Treatment:

- **Nonsurgical treatment** (Table 76-4)
  - Grade 1: pulley strain
    - No immobilization
    - Physical therapy 2 to 4 weeks
    - Return to light climbing 4 weeks
    - Full climbing 6 weeks
    - Taping for 3 months
  - Grade 2: complete rupture of A4 pulley or A2 or A3 partial rupture
    - 10 days immobilization
    - Physical therapy 2 to 4 weeks
    - Return to light climbing 4 weeks
    - Full climbing 6 to 8 weeks
    - Taping for 3 months
  - Grade 3: complete A2 or A3 rupture
    - 10 to 14 days immobilization
    - Physical therapy 4 weeks
    - Thermoplast or soft-cast ring
    - Return to light climbing 6 to 8 weeks
    - Full climbing 3 months
    - Taping for 6 months
- **Surgical treatment** (see Table 76-4, Fig. 76-6)
  - Grade 4: multiple pulley ruptures or A2 or A3 pulley rupture with lumbrical or ligament injury
  - Surgical repair:
    - Widstrom technique: loop and a half
    - Weilby repair as alternative
  - 14 days immobilization
  - Physical therapy for 4 weeks
  - Thermoplast or soft-cast ring
  - Return to light climbing at 4 months
  - Full climbing 6 months
  - Taping with climbing for at least a year

Prognosis: Most climbers do well with conservative care. Ten percent have persistent pain. Consider surgical treatment in climbers with residual pain or inability to return to prior climbing level.

Climber's Elbow

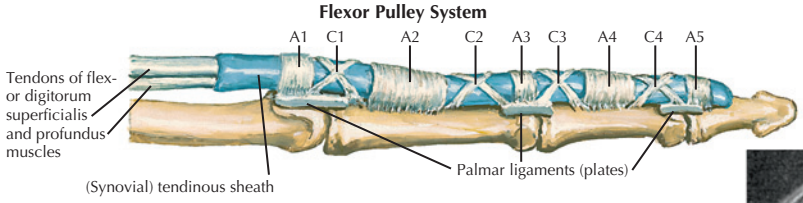
Description: Brachialis tendonitis or tear at muscle-tendon junction. Caused by flexion and pronation of the elbow typical of climbing with traverses. Insufficient firing of biceps brachii in the flexed and pronated position contributes to overuse of the brachialis (Fig. 76-7).

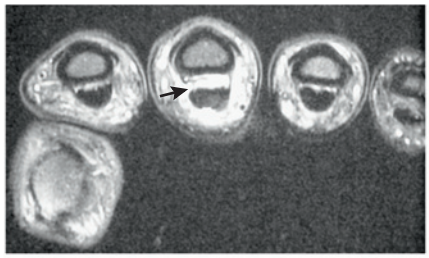
Presentation: Pain with climbing, typically presents after prolonged, hard climb or period of intense climbing over time with insufficient rest between climbs.

Physical examination: Painful to the anterior elbow, worsened with elbow flexion and pronation. Partial tear or rupture will typically have swelling, ecchymosis.

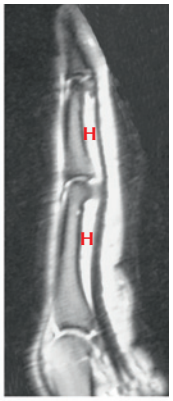
Diagnosis: Physical examination and history often sufficient to reveal climber's elbow. MRI or dynamic ultrasound can aid in identifying tendonitis or tendon tear (see Fig. 76-7).

Treatment: Most athletes with climber's elbow treated conservatively. Rest is the mainstay of treatment, typically 2 to 4 weeks of rest sufficient for strain. Modified climbing may be attempted

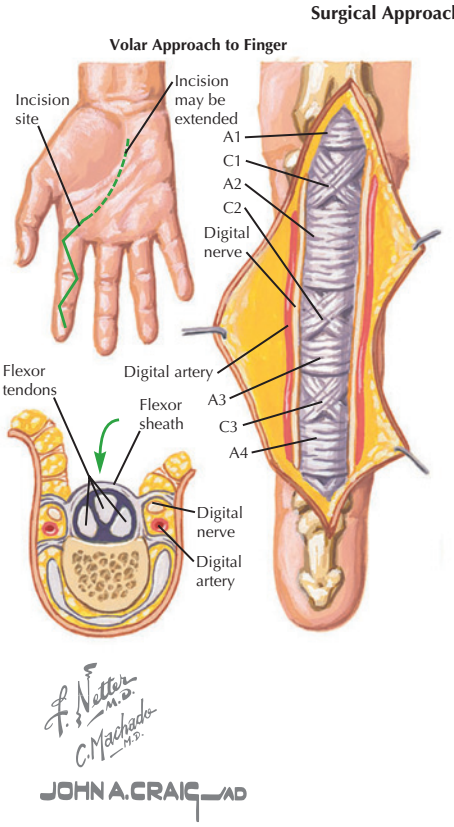


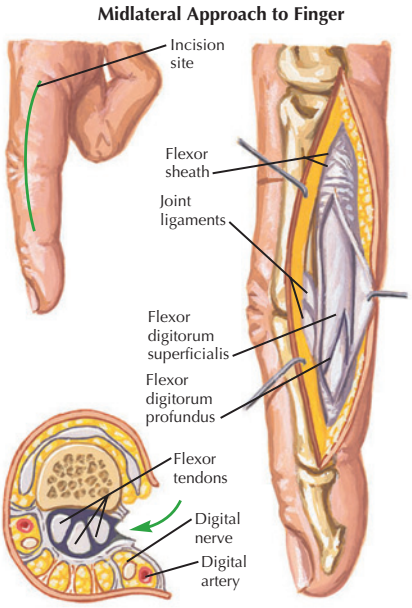


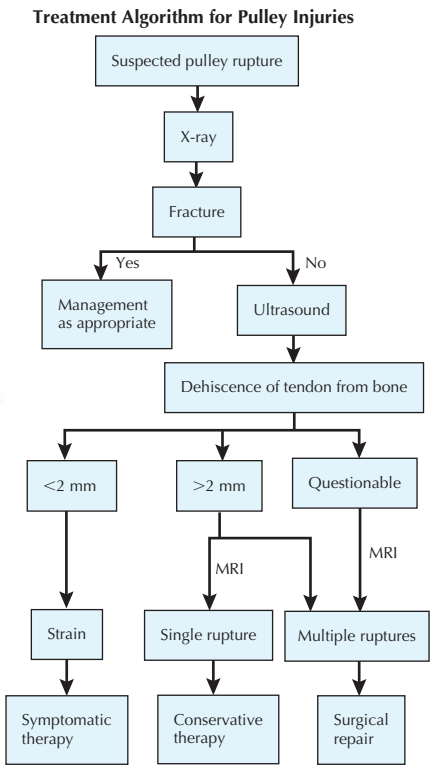
A2 pulley rupture.



Rupture of A2 and A4 pulleys.







(From Schoffl V, Hochholzer T, Winkelmann HP, et al: Pulley injuries in rock climbers. Wilderness Environ Med 14:94-100, 2003.)

Figure 76-6 Climber's Finger.

if milder injury but must be pain-free and climbing 2 to 3 levels below normal, with diminished frequency and intensity. Physical therapy highly beneficial, but requires experienced physiotherapist. Modality care, oral anti-inflammatories for inflammation and pain. Strengthening of opposing muscle groups. Complete ruptures should be treated with surgical repair without delay, within a week or two of injury for best results. Patients who fail conservative treatment should also have surgical consultation. Postoperative care includes:

- Postoperative splint at 90 degrees for 1 week, then hinged brace to 45 degrees, advancing to full range of motion by 8 weeks.
- Strengthening may begin at week 1 with isometric, low-intensity exercises to triceps and shoulder.
- Week 2 may strengthen biceps/brachialis with isometric, low-intensity flexion in forearm-neutral position.
- Active range of motion in single plane may begin at week 3 to 4 for flexion, pronation, supination.
- Initiate progressive resistance training at 8 weeks.
- May initiate light training at 3 months. Will take months to regain full activity level.

Prognosis: With adequate rest period, physical therapy, and strengthening of opposing muscle groups, many climbers return to full activity pain-free, whether conservative or surgical treat-

Table 76-3 GRADING OF FLEXOR PULLEY INJURIES

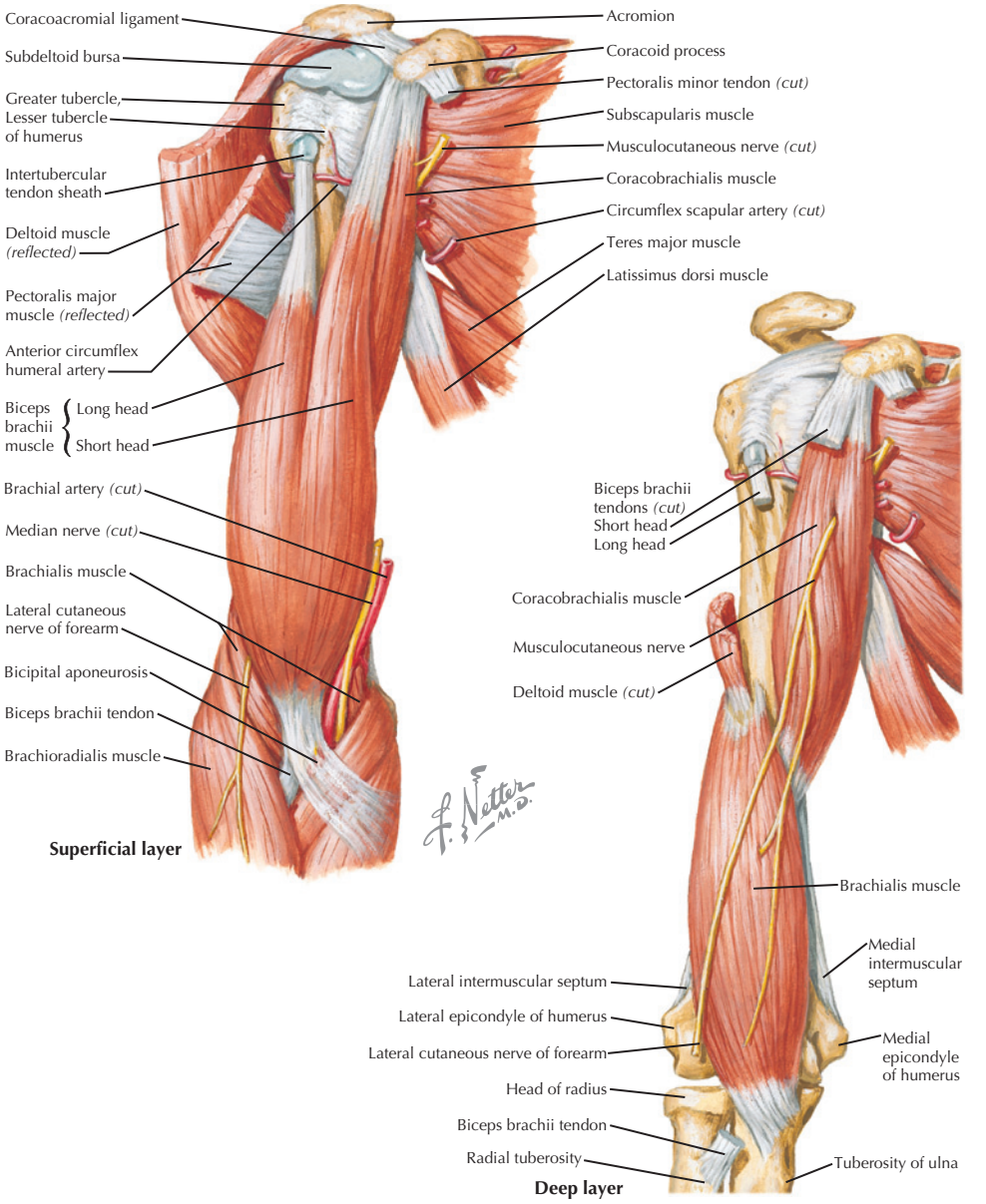
Grade	Flexor pulley injury pattern
Grade 1	Pulley strain
Grade 2	Complete rupture of A4 pulley or partial rupture of A2 or A3
Grade 3	Complete rupture of A2 or A3 pulley
Grade 4	Multiple pulley ruptures or single pulley rupture with lumbrical or collateral ligament injury

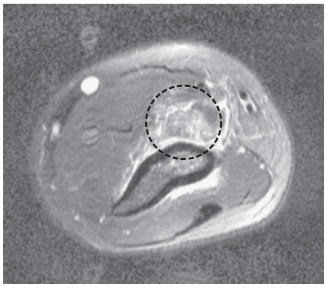
From Schoffl V, Hochholzer T, Winkelmann HP, et al: Pulley injuries in rock climbers. Wilderness Environ Med 14:94-100, 2003.

Table 76-4 TREATMENT OF PULLEY INJURIES

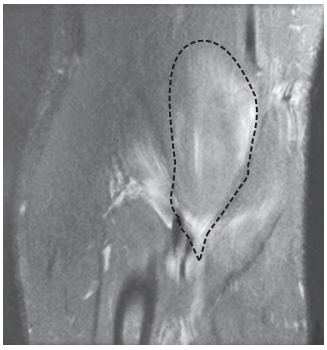
	Grade 1	Grade 2	Grade 3	Grade 4
Injury	Pulley strain	Complete rupture of A4 or partial rupture of A2 or A3	Complete rupture of or A3	Multiple ruptures, such as A2/A3, A2/A3/A4, or single rupture (A2 or A3) combined with lumbricalis muscle or ligament damage
Therapy	Conservative	Conservative	Conservative	Surgical repair
Immobilization	None	10 days	10-14 days	Postoperative 14 days
Functional therapy	2-4 wk	2-4 wk	4 wk	4 wk
Pulley protection	Tape	Tape	Thermoplastic or soft-cast ring	Thermoplastic or soft-cast ring
Easy sport-specific activities	After 4 wk	After 4 wk	After 6-8 wk	4 mo
Full sport-specific activities	6 wk	6-8 wk	3 mo	6 mo
Taping through climbing	3 mo	3 mo	6 mo	>12 mo

From Schoffl V, Hochholzer T, Winkelmann HP, et al: Pulley injuries in rock climbers. Wilderness Environ Med 14:94-100, 2003.

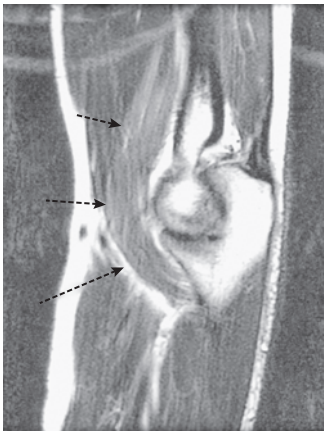




Axial T2 FSE images with fat saturation circle outline intramuscular edema in the brachialis.



Coronal proton density fat saturated FSE image circle outlines edema in the dunal brachialis.



T2 FSE sagittal image thru elbow *arrows* demonstrate increased signal intensity in the distal brachialis.

Figure 76-7 Climber's Elbow.

ment. Length of restricted activity depends on the severity of injury and resolution of symptoms.

INJURY PREVENTION, DIAGNOSIS, AND TREATMENT

Medical Care of Climbers

- Physician inexperience or misconception
  - "What do you expect when you climb?"
  - Diagnosis and treatment delayed
  - Many elite climbers find health professionals do not understand climbing stresses and are not helpful in diagnosis or treatment
- Compliance is a significant issue with climbing overuse injuries

Prevention

- Tendon strengthening, stretching, including opposing muscle groups
- Low-weight, high-repetition endurance training
- Heat prior, ice following
- Taping: pulley protection, buddy-taping when necessary
  - New method of "H-tape" produces added protection
  - Division of tape into two strips with a bridge in the middle, the bridge placed at the volar proximal interphalangeal (PIP) joint, then the strips placed circumferentially on either side of the dorsal PIP
  - Adequate rest period between climbs

Rest

- 24 hours recovery after training
- 48 hours recovery after climbing or hard lift
- May take months for significant injuries
- Splinting, ring, taping

Rehabilitation

- Can be 2 to 3 months
  - Stretches
  - Range-of-motion exercises
  - Low weights
  - Increase repetitions
- Strengthen antagonists
- Change climbing technique (e.g., rely more on open than cling grip), slowly return, start at lower level
- Formal physical therapy with experienced therapist familiar with climbing injuries
- Rock climbing can aid therapy of other injuries
  - Improves functional ankle instability with near-static movements, but care must be taken to not reinjure.

Repair

- Indication based on injury grade and injury type
- Failure of conservative therapy
- Reconstruction of fibro-osseous flexor sheath of fingers or repair of brachialis tear can fail:
  - Generally compliance issue
  - Insufficient rehabilitation

Counsel for Climbers

- Adequate instruction
- Climb with trusted companions
- Climb to your ability, know limitations
- Impeccable with equipment use and safety
  - Protect equipment from moisture, sun, heat, dirt, water, oil, abrasion
  - Appropriate protection, knots, equipment
  - Date ropes and harnesses, check damage after each use
- Plan and leave itinerary
- Cell phone, map, GPS
- First aid kit
- Food, water, sunscreen, eye protection, helmet

RECOMMENDED READINGS

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Martial Arts

77

Steven Erickson and Bryant Walrod

GENERAL OVERVIEW

Definition

- Martial arts, also known as fighting systems, are bodies of codified practices or traditions of training for unarmed and armed combat, usually without the use of guns and other modern weapons.
- People study martial arts for various reasons including improving fitness, self-realization (meditation), mental/character development, and self-defense.

Epidemiology

- Participants
  - Approximately 2 to 8 million participants in the United States, with millions more internationally
  - Male-to-female ratio is 5:1
- Injuries
  - One study in 2003 shows 70,000 injuries related to marital arts treated in hospital emergency departments and doctor's offices.
  - The incidence of martial arts injuries is actually low when compared to other sports.
  - In many styles of martial arts, much of the instruction and training is noncompetitive and noncontact, which leads to a very low incidence of injury.
  - Incidence of martial arts injuries is unknown because of a lack of reporting injuries and lack of studies regarding the sport.
- Fatalities
  - There has been one death recorded from mixed martial arts (MMA).
  - Reviewed registries of the U.S. Product Safety Commission from 1977 to 1995 found 25 cases of death caused by blunt force trauma (commotio cordis). One of the deaths was a result of an injury sustained during karate, whereas 16 of the deaths occurred while playing baseball.
  - One study shows six deaths during an 18-year surveillance of multiple disciplines of martial arts injuries: four occurred after trauma to the head (1 strike, 1 kick, 2 falls), one after trauma to the neck, and one after trauma to the chest.

FACTORS AFFECTING INCIDENCE AND PREVALENCE OF INJURY

Form of Participation in the Martial Arts

- There is no "off season" in martial arts; many participants train several times per week, year round, without a natural break in training for rehabilitation/recovery.
- Much of the emphasis of training is on personal development, both physical and mental, not on competition.
- Muscle mass and strength are typically not as important as speed, strategy, technique, mental discipline, and flexibility.
- Individuals of different levels of training may "spar" (to engage in practice competition with another individual to simulate the actual competition or bout) in practice, exploiting the mismatch in skill levels between participants.
- Tae kwon do had three times the injury rate and a fourfold risk of multiple injuries when compared to Shotokan karate.
- More major injuries and a greater incidence of multiple injuries in tae kwon do when compared to other disciplines.
- The incidence of injury is directly related to the amount of time spent in full contact, limited contact sparring, or competition.
- Setting of the competition may also influence injury rate and severity.
- Types of martial arts training include:
  - Kata—detailed choreographed patterns of movements/techniques used by practitioners to practice on their own
  - Basic hand strikes
  - Basic kicking
  - Strength training
  - Conditioning
  - Stretching
  - Flexibility
  - Breaking—using striking techniques to break boards or bricks
  - One-step sparring—noncontact sparring activity during which students practice techniques to be used during free sparring or competition
  - Grappling
  - Ground fighting
  - Arm bars—a submission move in which an opponent hyperextends an already extended arm with a fulcrum-like maneuver, forcing the person to either submit ("tap out") or risk rupture of the anterior capsule of the elbow
  - Chokes—can be employed via a variety of mechanisms using the upper or lower extremities to temporarily disrupt the vascular supply to the brain or compress the trachea to prevent breathing
  - Free sparring—to engage in practice competition with another individual to simulate the actual competition or bout
  - Point scoring competition
  - Full contact competition

Protective Equipment

Mouth guard: Increased risk of oral facial injury when mouth guards are not worn during competition.

Headgear: May reduce peak acceleration forces to the head (Fig. 77-1).

Hand and foot protection: Hand protection (as used in tae kwon do and mixed martial arts) and foot padding gear have not been shown to reduce peak acceleration to the head; however, one study shows that 10-ounce boxing gloves may reduce peak ac-

Head gear.


16 oz boxing gloves.


MMA style gloves.


Padded flooring.

Figure 77-1 Protective Equipment.

celeration forces (see Fig. 77-1). Padding may lead to decreased inhibition and poorer control of striking, which may lead to a greater number of blows with a larger amount of force. Hand and foot padding are thought to decrease the amount of superficial injuries, such as lacerations and abrasions, to both the attacker and the defender. It is important for the attending physician to observe all equipment in and around the ring for potential hazards (e.g., tape on gloves and ring stools).

Padded flooring: Padded flooring may reduce the intensity of a blow by absorbing some of the impact of falls and throws (see Fig. 77-1). The surface needs to be closely monitored for fall risks such as moisture (water, blood, perspiration) or gaps between the padding. Mat pads are common reservoirs for fungi and bacteria and therefore they need to be cared for in a similar manner to wrestling mats in order to prevent the spread of infections (e.g., community-acquired methicillin-resistant *Staphylococcus aureus*).

Age

Age is not a reliable predictor of the likelihood of injury.

Experience

- Experience and number of hours of participation appear to be associated with higher rates and severity of injury.
- One study noted a greater percentage of head injuries and fractures in professional Muay Thai kickboxers when comparing amateurs to beginners.
- Individuals with at least 3 years of experience were at twice the risk of injury than less experienced individuals.
- The number of tournaments and the months of practice were directly and significantly associated with the likelihood of eventual injury.

Setting

- Tournament and competitive situations are associated with a lower absolute number of injuries but higher rate of severity when compared with noncompetitive situations, possibly secondary to increased aggression in the tournament setting.
- Informal training sessions are associated with a higher risk and severity of injuries when compared with more formal supervised instruction.

Sex

- Males had higher rates and severity of injuries when compared with females, probably related to increased aggressiveness.
- Except in karate, where females have a higher injury rate than males.

Weight Class

- Increased risk of injury for Muay Thai kickboxing participants who compete at a heavier weight.
- Higher injury percentage in heavier weight classes in MMA when compared with lighter weight classes.

Prevention

- The American Academy of Orthopedic Surgeons offers these tips for martial arts participants to train and compete safely:
  - Consult with a physician before beginning your conditioning to establish your readiness.
  - Train under the direction of a martial arts instructor who focuses on form and technique, rather than competitive strategy.
  - Wear the appropriate protective gear for your type of activity; for example, tae kwon do, as a full contact sport, requires a head guard, a body protector, forearm and shin guards, and a groin guard.
  - Exercise to strengthen the stabilizer muscles: rotator cuff muscles and hip adductors (inner thigh muscles or groins) and abductors (muscles on the outermost part of the hip), which help to support the back and hips, are critical to this activity.
  - Maintain proper breathing techniques when practicing martial arts to avoid injury—breathing out during the contraction portion of any stretching movement, and breathing in during the extension portion of any stretching movement.

Coverage of Martial Arts Events

- General
  - Attending physician must be present when the rules of the competition are reviewed.
  - Important to identify yourself and briefly review certain medical considerations.
- Prefight examinations
  - Should be performed in a quiet, well-lit environment.
  - Be sure to inquire about previous and/or recent concussions and "knockouts."
- Match stoppage
  - Typically done by the referee; however, the referee may look to the physician for medical guidance as to whether or not the match should continue.
  - Typically intervention will be with respect to lacerations and bleeding.
- Cervical spine injuries
  - Often, the teammates or coaches will want to attend to their fighter and they may inappropriately move a fighter with an unstable cervical neck injury.
  - Review with the entire group of participants and their team the rationale and need for appropriate cervical spine immobilization.
- Postfight examinations
  - After a loss or a knockout, the participant may be confused, belligerent, or emotional.
  - It is important to maintain control of the situation to complete an appropriate examination.

Martial Arts–Specific Injury Considerations

- It is critically important to understand the unique aspects of each martial art discipline in order to best predict potential injuries.
- Certain martial arts disciplines focus more on contact and sparring whereas others focus more on technique.
- When covering a martial arts competition, or evaluating a martial artist, familiarize yourself with the techniques, emphasis, equipment, scoring, and target areas of each discipline, to anticipate various possible injuries.

COMMON TECHNIQUES IN MARTIAL ARTS

Hand Strikes

Punching: Striking an opponent with a closed fist (Fig. 77-2). Proper punching technique places the wrist in slight volar flexion, with the second and third metacarpals aligned with the long bones of the forearm. Contact is made with the second and third metacarpal heads. A "boxer's fracture" of the fifth metacarpal typically results from poor punching technique, in which an individual makes contact with the fifth metacarpal head instead of the second and third metacarpal heads. Common injuries from poor punching technique include phalanx fractures, fourth or fifth metacarpal fractures, wrist sprains, extensor tendon injuries, and first metacarpal phalangeal ulnar collateral sprains.

Knife hand chop ("karate chop"): A strike with an open hand during which contact is made with the ulnar aspect of the fifth metacarpal head.

Ridge hand (reverse knife hand chop): A strike during which the thumb is tucked into the palm and contact is made with the radial aspect of the second metacarpal head.


Proper punching technique.


Front kick.


Side kick.


Roundhouse kick.


Rear naked choke.


Triangle choke.


Guillotine choke.


Arm bar.


Leg lock.

Figure 77-2 Common Techniques in Martial Arts.

Spear thrust: Open hand technique during which contact is made with the fingertips of the second, third, and fourth fingers, most often targeting the eyes and throat.

Hammer fist: Closed hand strike with the ulnar aspect of the fist.

Spinning back fist: The attacker swivels 360 degrees and strikes opponent with the dorsum of hand and second and third metacarpophalangeal (MCP) joints, employing great power and momentum

Foot Strikes

Front snap kick: From a standing position, the hip is flexed up to bring the femur parallel to the floor (see Fig. 77-2). The leg is then extended, resulting in the ball of the foot making contact with the defender's abdomen. Note: If the target is the groin, then the foot is plantar flexed and the point of contact is the dorsal aspect of the proximal first metatarsal. If the target is the face, then the ankle is dorsiflexed and contact is made with the plantar aspect of the heel.

Side kick: Delivered sideways relative to the position of the person executing the kick. Contact is made with the heel targeting the abdomen or face (see Fig. 77-2).

Back kick ("Donkey Kick," "Mule Kick," or "Spinning Back Kick"): Kick is delivered backward, keeping the kicking leg close to the standing leg and striking with the heel. Most often it is delivered with a spinning motion generating great power.

Roundhouse kick: From the position of the hip flexed up to 90 degrees, the attacker swings their lower leg up in a circular motion, striking with the dorsal aspect of the proximal first metatarsal (see Fig. 77-2).

Miscellaneous: Some disciplines of martial arts encourage kicking with the shin opposed to the ball of the foot or instep to reduce the likelihood of injury.

Chokeholds

Rear naked choke: Attacker approaches opponent from behind, wrapping their arm around the opponent's neck and then grasping the biceps of their other arm. Then using their free hand, the attacker forces the head of the opponent into flexion, resulting in compression of the carotid arteries, temporarily rendering the opponent unconscious (see Fig. 77-2).

Triangle choke: Attacker is on their back (guard position) and wraps one leg around the neck and shoulder of the opponent with their knee next to the opponent's neck, the other leg crosses the ankle of the first leg, using the foot of the first leg to lock the second leg into position at the knee (see Fig. 77-2).

Guillotine choke: Can be applied from a standing position or the guard; the attacker faces the opponent and wraps an arm around the opponent's neck so that their humerus is on the dorsal aspect of the neck and their forearm wraps around anteriorly to apply pressure to disrupt the vascular supply to the brain, temporarily rendering the opponent unconscious or to compress the trachea in order to restrict breathing (see Fig. 77-2).

Joint Locks/Manipulation

Arm bar: A joint lock that hyperextends the elbow joint by placing the opponent's extended arm over a fulcrum such as an arm, leg, or hip (see Fig. 77-2). The opponent is controlled in this position and if they do not tap out then continued force will result in dislocation of the elbow.

Leg lock: A joint lock in which the attacker forcefully rotates a lower extremity, causing the opponent to submit (see Fig. 77-2).

Ankle lock: The ankle lock is a submission applied to the opponent or attacker in which the attacker uses their arm to secure the opponent's ankle in their armpit. Once the ankle is secured, pressure is applied by the attacker arching their back and tightening the arm on the ankle.

Miscellaneous: Small joint manipulation is typically prohibited.

TYPES OF INJURIES

Contusions: Most common overall injury. A contusion can become worrisome if, for example, an orbital contusion swells to the point of obstructing vision. Cold steel or ice can be employed between rounds to reduce edema. Higher rate of bruising in tae kwon do than in Shotokan karate.

Sprains/strains: Second most common overall injury. Most commonly sprained joints include ankles and knees. Hamstring and groin strains common from improper stretching. Aikido demonstrated a higher incidence of sprains and strains than Shotokan karate.

Abrasions/lacerations: Laceration management: direct pressure, petroleum jelly, clotting agent like silver nitrate or a vasoconstrictive agent. Physician may only examine a laceration during a fight to determine whether or not the participant may continue. The physician may not tend to or treat the laceration until after the round. Do not suture the laceration until after the competition. A competition should be stopped if the laceration bleeds to the extent that it will obstruct the vision of either competitor.

Epistaxis: Exceedingly rare to stop a competition for epistaxis. Direct pressure and petroleum jelly work well. Monitor for bleeding into either competitor's eyes such that it may obstruct vision. Careful examination to exclude septal hematoma or nasal fracture.

Fractures: More common in experienced martial artists. Finger and toe fractures common from improper kicking and striking techniques. Rib fractures common from being kicked.

Dislocations: Many digit dislocations can be reduced immediately with rapid return to competition. Postreduction radiography is recommended.

Mild traumatic brain injury (MTBI): Of severe injuries, concussion was the most common. Increased rate of concussion in younger age participants and those who demonstrate poor blocking skills. Increased rate of concussion associated with receiving a kick to the head. Decreased opportunities for head trauma in martial arts that emphasize grappling and joint locks over striking. Individuals that regularly participate in full contact sparring should have baseline neurocognitive testing for appropriate management of mild traumatic brain injury.

LOCATION OF INJURIES

Lower extremity: Instep of the foot most common lower extremity location of injury in tae kwon do. Common foot and ankle injuries are bruising, lacerations, ankle sprains, and digital fractures. Receiving and delivering a kick were the most common mechanisms associated with a lower extremity injury in tae kwon do. A roundhouse kick is most common type of kick to produce a lower extremity injury. Hamstring and adductor strains can occur secondary to overstretching or improper stretching.

Upper extremity: A unique injury with submission techniques is an arm bar, in which the anterior capsule of the elbow is stretched and may result in a complete rupture of the capsule and ligaments if continued force is applied and the participant does not tap out. An arm bar hyperextends an already completely extended elbow, stretching the surrounding capsule. If the elbow is rapidly extended, the capsule may tear and the elbow may dislocate. An arm bar also may result in a fracture of the humerus potentially causing damage to the radial nerve. Treatment of a simple arm bar with stretching of the capsule and anterior elbow pain consists of ice, nonsteroidal anti-inflammatory drugs (NSAIDs), and a sling for comfort (but be sure not to immobilize the joint to prevent stiffness). Complex arm bar injuries consisting of fractures and dislocations almost always require orthopedic evaluation for cartilaginous, ligamentous, or nerve injury. MMA size gloves (4 to 6 ounces) are typically worn to prevent hand injuries to the individual wearing the gloves. Injuries that occur from striking or kicking rather than from being struck are typically from poor technique.

Head/face/neck: These injuries are the most severe. There was an increased frequency of head injuries in martial arts disciplines that emphasize striking and head contact. Clear similarities in the force, kinematics, and biomechanics required to produce cervical neck injuries in motor vehicle accidents and common MMA maneuvers.

MARTIAL ARTS IN THE OLYMPIC GAMES

Judo ("Gentle Way")

- Founder: Jigoro Kano
- Country of origin: Japan
- Emphasis
  - Flexible or efficient use of balance, leverage, and movement in the performance of throws or techniques.
  - Skill, timing, and techniques are emphasized over brute strength.
  - Soft method in which there is an indirect application of force applied to defeat an opponent.
- Techniques
  - Throwing
    - Most throws are from a standing position.
    - Forceful throws are employed in an attempt to render an opponent unconscious.
    - Increased points are given depending on the severity of the throw.
  - Strikes (kicks and punches) are practiced but they are not permitted in competition.
  - Pinning
    - Attempt to hold an opponent to the mat.
    - Pins of 25 seconds will win the match.
    - Pins of 10 to 25 seconds will score points.
    - Significant pressure is applied to the opponent with pain-generating techniques.
    - Certain body parts, especially the ribs, are vulnerable for injury during these techniques.
  - Joint manipulation
    - Only elbow joint locks are permitted.
    - Leg locks, wrist locks, and spinal locks are not permitted.
  - Chokes (permitted depending on the age of the participants)
- Equipment
  - Judogi, "gi," meeting certain specifications and appropriately worn.
  - A gi is a Japanese name for a traditional uniform used in judo for practice and competition.
    - Consists of two parts, a heavy fabric uwagi (jacket) and a lighter fabric zubon (pants).
    - Garments are typically loose fitting and long sleeved.

Tae Kwon Do ("The Way of the Feet and Fist")

- Founder: unknown
- Country of origin: Korea
- Emphasis
  - Kicking techniques are emphasized because the leg is the longest and strongest limb in the body and thus has the potential to deliver the most forceful blow, rendering one's opponent unable to retaliate.
  - Union of physical and mental strength in such techniques as breaking of boards.
- Techniques
  - Hand techniques
    - Legal target areas: Front of the torso. This is restricted to the front of the body, starting at the hipline and going up to the base of the throat from one side seam of the uniform to the other side seam.
    - Legal hand techniques include typical punches and strikes.
    - Spinning back fists and finger strikes and other "blind techniques" are considered illegal.
  - Feet techniques
    - Legal target areas, front of torso as discussed above. The sides and back of the neck are legal as are all areas of the head including the face, mask, the sides and the top.
    - One may only strike from below the ankle, no shin or knee striking.
    - Hand and standing foot techniques to the legal torso area will score one point.
    - Standing kicks to the head, face, and neck will score one point, whereas a jumping kick to the body will also score two points.
    - A jumping kick to the head will score three points.
  - Breaking
    - Typically use hand or foot, but may also use fingertip, toe, head, elbow, knuckle, or knee.
    - It is postulated that the skeletal system, after stress is put to it, will be stronger after it heals. Thus many martial artists who practice breaking will continually stress their skeletal system, allow it to heal, and subsequently become stronger.
    - Many train to strengthen their fists by practicing bare knuckle push-ups, initially on a soft surface such as a carpet and then progressing to a harder surface like concrete.
- Equipment
  - White American Tae Kown Do (ATA) uniform
  - Mouthpiece
  - Protective cup
  - Headgear
  - Hand pads that cover the entire length of the fingers
  - Foot pads that cover the entire length of the toes
  - Chest protector

MARTIAL ART DISCIPLINES THAT FOCUS ON TECHNIQUE AND FORM

Aikido ("The Way of Harmonious Spirit")

- Founder: Morihei Eushiba
- Country of origin: Japan
- Emphasis
  - Joining with an attacker and redirecting the attacker's energy
  - Moving together rather than clashing
- Techniques
  - Body throws
  - Joint locks
  - Strong strikes or immobilizing grabs are employed but they are not emphasized

Hapkido ("The Way of Coordinating Spirit")

- Founder: Choi Youg Sul
- Country of origin: Korea
- Emphasis
  - Circular motions
  - Nonresisting motions
  - Control of opponents
- Techniques
  - Grappling
  - Throws
  - Joint manipulation—large and small
  - Pressure points
  - Punches and kicks are used, but they are not a primary emphasis

Jujitsu ("Art of Gentleness")

- Founder: Unknown
- Country of origin: Japan
- Emphasis
  - The art of breaking balance
  - Yield force provided in an opponent's attack in order to apply a counter-technique
- Techniques
  - Strikes and kicks
  - Throws—perfect throws score more points than a throw that is strong but not perfect as deemed by the judges
  - Chokes
  - Pins
  - Joint manipulation

Tai Chi Chuan ("Supreme Ultimate Boxing")

- Founder: disputed—however, origins can be traced to the Chen and Yang families
- Country of origin: China
- Emphasis:
  - Receptive "ying"—slow, repetitive, meditative, slow impact
  - Active "yang"—active, fast, high impact
  - Health—relieve the physical effects of stress on the body and mind
  - Meditation—cultivate focus and calmness to maintain homeostasis and relieve stress
  - Martial art—change in response to outside forces
  - Do not resist a violent force; instead, meet a force and redirect it
- Techniques
  - Solo form: A slow sequence of movements that emphasize a straight spine, abdominal breathing, and a natural range of motion.
  - Pushing hands for training in a practical form.
  - Pushes and open hand strikes are more common than punches.
  - Kicks are usually to the leg and torso, never higher than the hip.
  - Fingers, fists, palms, forearms, elbows, knees and feet are used to strike the eyes, throat, heart, and groin.
  - Joint locks are also used.

MARTIAL ARTS DISCIPLINES IN ADDITION TO JUDO AND TAE KWON DO THAT FOCUS ON SPARRING AND CONTACT

Karate ("Empty Hand")

- Founder: Tode Sakukawa or Gichin Funakoshi
- County of origin: possibly Ryukyu Kingdom of Japan

- Emphasis
  - Striking art.
  - Forms or techniques that demonstrate combat principles.
  - Techniques must be performed with excellent control and good form.
  - Injuring an opponent may result in a point deduction.
  - Light contact is permitted and emphasized, not full contact.
- Techniques
  - Kicks: Kicks to the head and neck score more points than kicks to the body.
  - Punches: Punches to the back and back of the head score more points than punches to the head and trunk.

Kickboxing

- Founder: Unknown, term was created by Japanese boxing promoter Osamu Noguchi as a variant of Muay Thai and karate.
- Emphasis: Strikes with punches and kicks while standing up.
- Ground techniques and grappling are *not* permitted.
- Techniques
  - Strikes above the hip with fists and feet.
  - Using elbows and knees is forbidden, and use of shins is seldom permitted.
  - Spinning back-of-the-fist punches are permitted as well as traditional punches.

Mixed Martial Arts (MMA)/Pankration/ Freestyle

- Combat sport in which participants use a multidisciplinary approach to fighting that includes jujitsu, judo, boxing, wrestling, karate, and kickboxing and other fighting disciplines in a supervised bout between two individuals.
- MMA has surpassed boxing as the most popular spectator combat sport in the United States.
- Founder: Derives from multiple sports.
- Country of origin: Major organizations exist in the United States and Japan.
- Argued to be safer than boxing because bouts may be ended by causing an opponent to submit by using arm bars and chokes rather than rendering an opponent unconscious with a strike to the head.
- Participants are required to have a preparticipation physical examination including HIV screening for most bouts.
- Techniques
  - Striking with punches and kicks to the head, face, body, and extremities.
  - Elbow and knee strikes are permitted.
  - Grappling and wrestling techniques are permitted.
  - Chokes are permitted.
  - Joint manipulation/submission/arm bars are used.
  - Points are scored for overall aggressiveness, ring control, and effective striking.
- Illegal actions (Table 77-1)
  - Head butting
  - Eye gouging
  - "Fish hooking"—inserting fingers into an opponent's mouth, and pulling intensely on the lips and perioral skin
  - Hair pulling
  - Biting
  - Small joint manipulation
  - Spiking an opponent to the canvas on the head or neck
  - Kicking a downed opponent
  - Knees to the head of a downed opponent
  - Striking with the point of the elbows
  - Strikes to the spine or back of the head
  - Groin strikes or throat strikes

Table 77-1 MIXED MARTIAL ARTS STOPPAGE OF MATCHES

Reason for stoppage of MMA matches	▪ Head impact: 28.3% ▪ Expiration of time: 27.0% ▪ Musculoskeletal stress: 16.5% ▪ Neck choke: 14.1% ▪ Miscellaneous trauma: 12.9% ▪ Disqualification: 1%
Mechanism of match stoppage	▪ Punch: 16.8% ▪ Elbow lock: 9.3% ▪ Rear choke: 6.5% Other strike to head: 5.9% Front choke: 4.2% Leg choke: 2.3% Knee strike: 2.2% Ankle lock: 2.0%

Data from Buse GJ: No holds barred sport fighting: A 10 year review of mixed martial arts competition. Br J Sports Med 40:169-172, 2005.

Tang So Do ("Way of the Chinese Fist")

- Founder: Hwang Kee
- Country of origin: Korea
- Emphasis: Empty hand and foot fighting forms with an emphasis on the development of discipline, self-confidence and self-defense.
- Techniques
  - Emphasis is placed on kicking techniques, especially turning and spinning kicks to the torso and head of the opponent.
  - Katas are used as a method of training for practitioners away from the dojo.
  - Techniques drawn from ancient Chinese and Japanese martial art traditions.

Thai Boxing (Muay Thai) ("The Art of Eight Limbs")

- Founder: Unknown—traced to Buddhist Monks
- Country: Thailand
- Emphasis: A distinguished art of fighting of attrition that employs strikes with fists, elbows, shins, and knees.
- Techniques
  - Strikes: Fists, elbows, shins, feet and knees are all permitted to strike an opponent.
  - Punching techniques score less highly than other striking techniques.
  - Emphasize entire body movements and hip rotation with strikes to increase strength.
  - Core body strength is essential.
  - Kicking techniques are encouraged to connect with the shin.
  - Grappling or clinching (Thai clinch): A clinch is not broken up as in traditional boxing because this can be an advantageous position from which to employ knee strikes.

SUMMARY

- The incidence of martial arts injuries varies with the quality of instruction and the quantity and type of combative training practiced by the participant.
- Injury rates vary greatly, depending on style of martial arts practiced, age, sex, and experience level of the participant.
- Injuries are very uncommon during practice sessions that primarily involve teaching techniques and/or practicing techniques that do not involve full-speed contact.
- There are millions of martial artists practicing in the United States who do not compete or spar and therefore have a very low incidence of injury.

- Mixed martial arts (MMA) has grown to be the most popular combative spectator sport in the United States and is arguably safer than boxing because match stoppage is not solely from knockout.
- Participation in martial arts training continues to grow in part because of the emphasis on personal growth and improved self-confidence as well as physical development and self-defense.
- Martial artists should be encouraged to use protective gear whenever possible because the use of protective gear decreases the incidence of lacerations, contusions, and abrasions and may decrease the peak acceleration forces to the head during kicks and strikes.
- Sports medicine providers should familiarize themselves with the rules and practices of the different styles of the martial arts as well as the terminology used to describe the types of training and the various kicks, strikes, and attacks used by martial artists.

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Boxing

78

Brian A. Jacobs and Jessica Ellis

HISTORY

- Origins
  - Boxing is one of the most ancient of sports.
  - Drawings of matches are preserved on the walls of Beni Hasan in Egypt dating back between 1500 BC and 2000 BC.
  - Boxing became an Olympic sport for the first time in 688 BC, with Onomastos of Smyrna garnering the first championship.
- Modern era
  - Modern boxing was born under the tutelage of James Figg and Jack Broughton.
  - Figg established a school for fighters on Tottenham Court Road in London around 1720.
  - His protégé, Broughton, called by some "the father of modern boxing," developed rules for fighting after one of his opponents died in the ring.
  - His rules revolutionized and civilized, to some degree, a sport that had been marred by bloody and violent ends.
  - Boxing rules were further modified by the Marquess of Queensbury in the latter half of the 19th century.
  - The "Queensbury Rules" allowed for a 24-foot ring, the use of padded gloves, a limited number of 3-minute rounds, a 1-minute rest period between rounds, elimination of wrestling and "clutching," delineation of certain fouls, the elimination of spiked shoes (allowed previously), and the institution of a 10-second down count.
- Olympic boxing
  - Men's boxing became a recognized competition of the 1904 Olympic Games in Saint Louis, Missouri.
  - Women's boxing matches were held as exhibition fights in that same year.
- Women in boxing
  - The history of women in boxing parallels that of men.
  - Fights are reported from the 1720s during which the female fighters were allowed to punch, kick, knee, maul, and throw their opponents.
  - As with men's fighting, the rules for women's boxing evolved.

AMATEUR BOXING

- Age categories
  - Open to males 8 to 34 years of age and to females 16 to 34 years of age.
  - A Masters category for fighters over 34 also exists, but these fighters do not progress through tournament stages.
    - Beyond the mention of the category, this chapter will be limited to the care of the non-Masters boxers.
- Weight classes
  - Assigned based on a calculation using fat free weight (FFW), following the calculation FFW/(1-[minimum body fat percentage × 0.1]), and assigning the athlete to the nearest weight class above that number (Table 78-1).

Table 78-1 AMATEUR BOXING WEIGHT CLASSES

Category	Weight class	10-oz glove	12-oz glove
Junior olympic	*−154	X	
	165-201+		X
Open boxers	101-152	X	
	165-201+		X
Open <19 (USA Boxing)	106-152	X	
	165-201+		X
Open <19 (AIBA Boxing)	106-152	X	
	165-201+		X
International (AIBA)	106-152	X	
	165-201+		X
Masters (35+)	106-152	X	
	165-201+		X

*−154, weight up to 154

INJURY PREVENTION

Equipment

Headgear: Amateur boxing requires the use of headgear, 10- to 12-ounce gloves and a fitted mouth guard. In some cases a chest protector is allowed. Well-fitted headgear is to be fitted by coaches educated to do so. A headgear appropriately fitted reduces the number of eye injuries and facial fractures, lacerations, the force of blows to the head, "cauliflower ear," and perforation of the tympanic membranes. Headgear is used when sparring and is required during amateur competition.

Gloves: Gloves with attached thumbs have decreased both eye and thumb injuries, and heavier gloves have decreased hand injuries and impact forces. Two-toned gloves are easier to see; this allows for easier visualization of both legal and illegal blows. Gloves reduce the force of impact to the hands and "thumbless" gloves and those with attached thumbs prevent injuries to the thumb. Thus, gloves with attached thumbs or no thumbs are authorized for use, and others are banned. Gloves must remain clean and in as close to "like new" condition as possible. If gloves are found to be defective, they are to be replaced. In all U.S. Championships, the sponsor must supply new gloves with the USA Boxing label or stamp affixed.

Hand wrap: Hands must be wrapped because wrapping prevents injury to the hands and further reduces the force of blows delivered. The process of wrapping of hands is to be supervised by the appropriate official.

Mouth guard: Custom-fitted mouth guards prevent dental injuries and reduce temporomandibular joint injuries. Mouthpieces are also employed to allow the athlete to "set" his or her jaw, reducing the likelihood of knockdowns by reducing the intensity of the blow. It is suspected that they may reduce forces in head trauma and possibly reduce concussion as well.

Breast protectors: Breast protectors when used must be well-fitted and not interfere with the boxer's ability to fight. It should fit well and not extend beyond the clavicles or the xyphoid.

Referee

- Referee must inspect the mouthpiece of each fighter at the prebout check.
- Referee should also inspect the fit of the headgear and give a quick head-to-toe survey of the boxer in general.
- As the chief safety official, the referee should "act minutes early rather than seconds late" when determining when to stop a bout.
- Acting with the athlete's best interests at heart may further reduce injury.

Prefight Physical Examination

- An annual physical examination is required for each athlete, with that exam to be performed by a qualified MD or DO.
- The physical is intended to reduce risk of injury by identifying potential complicating medical problems prior to competi-

tion, and through monitoring the recovery from previous injuries.

Physician Approval, Qualification, and Disqualification

- Athletes are required to have approval from their physician prior to competing.
- The physical exam must certify that the athlete is free from any injury, disability, or infection that could impair or threaten that athlete's ability to box.
- Athletes should have no history of diabetes, high blood pressure, chest pain, chronic headaches and have normal vision within the parameters elaborated upon later in the chapter.
- Disqualification of an athlete will occur if the athlete is found to have HIV/AIDS, active hepatitis B, or active herpes simplex.
- Immunization against hepatitis B virus (HBV) is recommended as is yearly testing for HIV for amateur boxers.
- Widespread screening and mandatory testing for other blood-borne pathogens is not recommended.
- Hepatitis C virus (HCV), although frequently tested for, is not currently a disqualifying condition.
- Nevada State Boxing Commission requires HIV, HBV-surface antigen (SAg), and HCV antibody testing prior to clearance for professional fighters.
- At this time, tests for blood-borne diseases are not required of professional boxers in Colorado, Hawaii, Iowa, Maine, North Dakota, Oklahoma, Vermont, Virginia, West Virginia, or Wisconsin.
- It is best to contact the individual state laws regarding testing for blood-borne diseases.

Special Considerations

Deaf and mute boxers: Eligible to participate provided they pass all other aspects of their prefight physical exams.

Vision impairment: Candidates with uncorrected vision worse than 20/400, or corrected vision of 20/60 or worse in either eye, will be disqualified. If an athlete's vision is better than 20/400 and is correctable to 20/20, that athlete may compete but glasses are not allowed during competition. Contact lenses may be worn but loss of a contact lens during a match may result in a "Referee Stop Contest" (RSC—discussed later) decision against that athlete.

Preexisting injury or illness: No boxer will be allowed to participate in a match if the boxer has a dressing on a cut, wound, abrasion, laceration, or a hematoma of the scalp, face, or ears. The fighter will be evaluated by the referee prior to the fight for conditions that may disqualify competition.

Banned substances: In amateur and Olympic competition, banned substances such as ergogenic aids and steroids are disqualifying if used. Refer to the appropriate authority for comprehensive lists of banned substances and medications.

RINGSIDE PERSONNEL AND EQUIPMENT

Coaches

- One coach and one assistant are allowed for each fighter, and they must remain seated during each round.
- Must not interact with fans or ringside officials.
- The coaches should have first-aid supplies, two clean white towels, sterile gauze pads, sterile cotton, cotton swabs, and ice bags with ice.
- First aid equipment does not include ammonia, ammonia inhalants or smelling salts, which are banned.

Cutman

- Cutmen are indispensable in the lives of boxers.
- They attend to bleeding injuries that could otherwise disqualify a boxer during a bout.
- The cutmen are responsible for stopping the bleeding.
- Amateur boxing prohibits the use of any medication to treat bleeding during a bout, but professional boxers are allowed any medication or topical treatment that the cutmen or trainers may have at their disposal.

Controlling Injury and Bleeding

- The cutman or trainer is allowed 1 minute to get the bleeding under control.
- Swelling of the eye or face is managed by pressure applied with an Enswell, an iced metal spatula applied to the hematoma that milks the blood to the surrounding tissues.
- Prevention of bleeding injuries can be done with use of petroleum jelly applied to the face.
- Medications—when allowed—to control bleeding include Qr, Avitene, Surgicel, Gelfoam, adrenaline chloride, and thrombin; these are frequently mixed with petroleum jelly and applied with cotton swabs.

PHYSICIAN'S ROLE

Advocating for the Boxers

- The physician is the ringside advocate for the fighters and is responsible for the care and safety of the fighters before, during, and after the match.
- One or more physicians (MD/DO) who are capable of initiating life-saving procedures and medications should be seated at ringside at all times during competition.
- Two physicians are preferable because this arrangement allows for one to attend to an injured boxer after completion of that boxer's match, while the other physician attends to the current bout.

Prefight

- All contestants should be thoroughly examined immediately before and after each bout.
- Female participants should furnish a signed, explicit disclaimer stating that the boxer has read Rule 101.9 (Box 78-1).
- The waiver states that to the best of the fighter's knowledge she is not pregnant.
- At the time of her prefight physical, the physician will take an appropriate history regarding menstruation, pregnancy, and breast and gynecologic disease and surgical history and note the findings within recommended sports guidelines.

Tournaments

- Tournaments present special concerns.
- Fighters should be examined before each bout because injuries may have occurred in a previous bout that could impair

BOX 78-1 *Female Boxing Rule 101.9*

Female boxers are limited to participation with additional medical restrictions. If any of the following conditions exist, the athlete is not allowed to participate:

- Confirmed pregnancy; suspect pregnancy must be confirmed or denied before starting or continuing boxing
- Painful pelvic disease states such as symptomatic endometriosis
- Abnormal vaginal bleeding of undetermined etiology
- Recent secondary amenorrhea of undetermined cause
- Recent breast bleeding
- Recently discovered breast masses
- Recent breast dysfunctions previously not present

From McCrory P: Cavum septum pellucidi—a reason to ban boxers? Br J Sports Med 36:157-161, 2002.

the safety and ability of the boxer, precluding further participation.

Disqualifying During Competition

- The following injuries disqualify initial or further competition:
  - Excessive swelling of the face or eyes that impairs vision
  - Active herpetic lesions of the face
  - Suspected or proven fractures of the nose, face, or metacarpals
  - Presence or history of a retinal detachment
  - Lacerations or wounds requiring dressings for control of bleeding, including such injuries to the face, scalp, and ears
- Refractive surgery is a relative contraindication.

Surveying the Venue

- It is imperative that the ringside physician be familiar with the venue, including the location of the training room and the location of the emergency medical system (EMS) providers and ambulances.
- The physician should also be familiar with the layout of the facility, the ring, the condition of the padding at the turnbuckles, and the condition of the ropes and mat.
- Further, the physician should get to know EMS and first-aid personnel and be aware of the location of EMS and first-aid equipment, including spine boards, oxygen, cervical collars, advanced cardiac life support (ACLS) equipment (defibrillators if available), and assure adequate phone proximity or cellular connections.
- It is mandatory that the physicians be seated at ringside with an unobstructed view of the ring and of the matches.

Gamebag/Medical Supplies

- At ringside in amateur competition, physicians are allowed to have the following available for use:
  - Sponges
  - Flashlight
  - Oral airway
  - Gloves
  - Vaseline
  - Adrenaline chloride 1/1000 to control nosebleeds and cuts
  - Thrombin for dry cuts
  - Avitene (microfiber collagen hemostat) for active bleeding
  - Ice and ice bags
  - Clean sponges
  - Gauze pads
  - Enswell pressure plates for control of hematomata
  - Two clean white towels
  - Scissors
  - Water in a clear plastic container
  - Two buckets, one containing ice and the other empty
  - Adhesive athletic tape
- Physicians involved in professional matches may have supplies they deem as necessary.

Evaluation during the Bout

- When called into the ring, the physician should always assume a cervical-spine injury has occurred and stabilize the athlete as necessary.
- As always, the physician is advised to abide the "Airway-Breathing-Circulation" principles of basic life support.
- Likewise, the physician and all ring personnel should adhere to appropriate precautions when near blood or body fluids.
- The physician should notify the athlete of his or her presence and title; also, the physician should be controlled and efficient in delivering care.
- For those athletes not requiring EMS transfer, have the athletes return for reevaluation.

THE BOUT

Rounds and Standing Eight Counts

- Amateur bouts are characterized by rounds of 2 to 3 minutes in length separated by a 1-minute rest interval.
- The total number of rounds is no greater than four.
- The referee can institute a standing eight-count whenever it is believed that a fighter has been stunned.
- Should a fighter incur three standing eight-counts in a given period or four in a given match, the fight is discontinued as RSC (H) (referee stop contest–head injury).
- The fighter is referred to physicians for immediate evaluation after the bout.

Unconscious Athletes

- In the event an athlete is unconscious, the referee signals the physician and only then the physician enters the ring.
- Only the referee and the physician attending at ringside are allowed to enter the ring unless the physician requires assistance.

Scoring

- In amateur boxing, points are awarded for blows to the upper body and head regardless of force applied and regardless whether the struck contestant was knocked down or knocked out.
- As mentioned earlier, scores in professional boxing are also tabulated as points scored but greater weight is given to blows of greater force, knockdowns, and knockouts.
- Scores in professional bouts can thus have subjective bias.

THE REFEREE, THE PHYSICIAN, AND STOPPING FIGHTS

Referee

- The referee serves as a safety official and operates to act in the best interests of the fighters in contest.
- It is the duty of the referee to assure that the rules of fair play are strictly observed.
- Also, the referee is responsible for maintaining control of the athletes at all times, and for preventing unnecessary punishment of a weaker opponent by a stronger one.
- The referee must assure that the gloves, mouthpiece, and dress adhere to the appropriate rules.

Ringside Physician

- The physician should at all times pay close attention to the match because doing so will allow for evaluation of injury mechanism, fatigue, and other factors that contribute to injury.
- If it is necessary for the physician to stop the match, the physician should be firm in doing so.
- Reasons for stopping a match include but are not limited to:
  - Cuts with arterial bleeding that cannot be controlled; bleeding of the mouth, ear, or of the eyelid that obstructs vision; cuts extending with further blows.
  - Any trauma that renders the athlete unable to defend him or herself.

Stopping the Bout

- The ringside physician may at any time suspend a bout by mounting the ring apron, but enters the ring only at the behest of the referee.
- If the physician believes that a boxer is no longer capable of fighting or that the fighter is in danger of further injury, the physician can signal the referee to terminate the bout.
- At that time, the referee can call Referee Stop Contest (RSC).

- If the fight is stopped because of concern of concussion or other head injury, the designation is RSC (H), indicating that the fight has been stopped due to head injury.
- In this instance, the fighter will then be evaluated immediately after the fight has been declared RSC (H).

POSTBOUT EXAMINATION

- Restrictions affidavit: If a bout is ended by an RSC (H), the injured boxer receives a restrictions affidavit requiring signatures of the athlete, the physician at ringside, the referee, and the coach.
- The boxer's coach is responsible for assuring the boxer gets home or to the appropriate accommodations safely after the fight.
- After any bout, it is imperative that the boxer's physician be contacted immediately of any of the following symptoms present:
  - Headache or dizziness lasting more than 2 hours
  - Increasing drowsiness or decreasing level or no consciousness following a bout (i.e., the athlete should be roused every 2 hours to assess)
  - Uncontrolled or persistent vomiting
  - Blurring of vision
  - Irrational behavior or persistent confusion
  - Any seizure
  - Inability to move a limb
  - Agitation or excessive restlessness
  - Oozing of bloody/clear fluid from the nose or ears
  - Incontinence of bowel or bladder
- Medical survey after the bout
  - All fighters should be assessed upon completion of a contest with special attention toward neurologic and orthopedic status.
  - All injuries suffered in a bout should be recorded appropriately.
  - Any medical suspension that follows from a bout should be noted on the athlete's fight card.

RESTRICTION FROM BOXING

If a decision was rendered as RSC (H), the following parameters and restrictions apply:

RSC: No restriction applies. Examples of this include matches in which fighters are clearly mismatched in skill, for instance with a fighter who is outclassed and taking an excessive number of body blows, or a fighter receiving an injury (e.g., a dislocated shoulder) that does not require RSC (H).

RSC (H) (30): A 30-day restriction from sparring and competitive boxing follows after three standing-eight counts in one round or four in one match, or when a boxer suffers a stunning head blow and does not respond normally even if the boxer remains standing. This rule also applies to a boxer knocked down by a head blow who immediately assumes an upright "ready" posture if the referee stops the contest.

RSC (H) (90): A 90-day restriction from sparring and competition applies to boxers who were rendered unconscious for a period of less than 2 minutes as determined by the ringside physician conferring with the official timekeeper. This applies whether the boxer was rendered unconscious by legal or illegal blows.

RSC (H) (180): A 180-day restriction from sparring and competition applies to a boxer rendered unconscious for at least two minutes. This applies whether the boxer was rendered unconscious by legal or illegal blows.

Second RSC (H): Should a second RSC (H) occur after the initial 30-day restriction, an RSC (H) 90 applies. Should a second RSC (H) occur after the initial 90-day restriction, an RSC (H) 180 applies. Should a second RSC (H) occur after the initial 180-day restriction, an RSC (H) 365—a 1-year restriction from sparring and competition—applies.

INJURIES COMMON TO BOXING

Comparing Injury Rates to Other Sports

Boxers suffer injuries at rates comparable to, and in many cases lower than, the rates found in other contact and noncontact sports (Table 78-2).

Head and Brain Injury

Acute Traumatic Brain Injury

- Acute brain injuries include concussions, intracerebral hematoma, epidural hematoma, subdural hematoma, and second impact syndrome.
- Of these, concussions are relatively common and tend to resolve without sequelae.
- The latter four injuries, although rare, can have devastating consequences and require a high index of suspicion and astute, efficient, and judicious evaluation and management.

CONCUSSION

- Relatively common and the sequelae are generally short-lived.
- In one study of 175 amateur college-aged fighters there were no findings of acute cognitive impairment.
  - The athletes demonstrated improvements in performance of a learning speed task, suggesting practice effects caused by repeated test administration over a relatively short time—in spite of participation in the boxing tournament.

INTRACEREBRAL HEMATOMA

- May present without loss of consciousness or focal neurologic deficit, but may present with persistent headache and periods of confusion.
- If mental status does not clear after injury, computed tomography (CT) may demonstrate the presence of an intracerebral bleed.
- These athletes require close monitoring and repeated scans because hydrocephalus or expansion of the intracerebral bleed may follow, necessitating surgical evacuation (Fig. 78-1).

EPIDURAL HEMATOMA

- Classically, when a patient sustains an injury that causes an epidural bleed, the patient may experience a brief alteration or loss of consciousness followed by arousal and even a normal level of consciousness (see Fig. 78-1).
- This may be followed rapidly by deteriorating mental status and a unilaterally dilated pupil—typically ipsilateral to the clot—and contralateral weakness and progressive decline.
- Approximately one third of patients will manifest this classic clinical triad.

SUBDURAL HEMATOMA

- Arise as a result of tears in the bridging vessels between the brain and the dura mater (see Fig. 78-1).
- Presenting signs can be subtle or can manifest as hemiparesis, seizures, photophobia, nausea and vomiting, papillary changes, elevated blood pressure, slowed heart rate, with the injury leading to uncal herniation and death.

SECOND IMPACT SYNDROME

- Is believed to occur when an athlete, who suffered an initial head injury, sustains a second head injury before the first injury has resolved.
- The second injury may be relatively minor, and even occur in whiplash fashion.
- Initially, the injured boxer may appear stunned.
- He or she may remain conscious and may remain standing for up to 1 minute but then suddenly collapse, semicomatose on

Table 78-2 INJURY STUDIES IN BOXING

Amateur boxing	In a study of 1094 amateur fighters at the 1981 and 1982 USA/ABF championships: ▪ 85 injuries of varying degrees of severity, 52 considered notable. ▪ 48 matches were discontinued because of head blows, which occurred at a rate of 4.38%. ▪ All other injuries occurred at a rate of 4.75%. ▪ Soft tissue injuries of the hands accounted for the next largest category of injury in this study, comprising sprains of the first MCP ligaments, subungual hematomas, contusions and sprains of the carpus. Fractures of the hands and wrist accounted for three injuries; likewise, nasal fractures accounted for three.
Olympic boxers	A study conducted at the United States Olympic Training Center over a 15-year period from 1977-1992 showed the following injury trends: ▪ Of 1776 reported injuries, only 6.1% met criteria for "serious injury," defined as a condition "which could not be remediated through the typical services of an athletic trainer at the USOTC" (concussions would fall within this definition). ▪ Of all injuries, contusions, muscle strains, joint sprains, and tendonitis occurred with the greatest frequency, accounting for 71.9% of all injuries in this study. ▪ Fractures—categorized as a group rather than by body region—accounted for 4.9% of all injuries, lacerations for 4.1%, "neural disorders" including concussion for 1.4%, and all others for the remaining 17.7%. ▪ The study also listed injuries by body region; upper extremity injuries accounted for 24.8% of the total, head and facial injuries for 19.4%, lower extremity injuries for 15.0%, and spinal column injuries for 9.4%. ▪ Multiple examiners with many specialties and many different levels of training were involved in data collection.
Professional boxers	In a study of professional boxers conducted over a 16-year period in Victoria, Australia: ▪ Face and head trauma comprised 89.8% of all injuries reported, with the eye, eyelid, and eyebrow injured most frequently. ▪ Concussions account for 15.9% of injuries, with all other injuries accounting for only 10.7% of the total. ▪ It is notable that in the Victoria study, most concussions were not thought to be severe, and most head injuries were lacerations, abrasions, and hematomas. ▪ Next to injuries of the head and face, injuries to the hands and fingers accounted for 2.7% of all injuries, the next largest number of injuries. ▪ Other sites of injury mentioned include sprains of the ankle, muscular strains of the low back, perforations of the tympanic membrane, dental fractures, fractures of the ribs, and contusions to the lungs.

Data based on studies from Estwanik JJ, Boitano M, Ari N: Amateur boxing injuries at the 1981 and 1982 USA/ABF national championships. Phys Sportsmed 12(10):123-128, 1984; Zazryn TR, Finch CF, McCrory P: A 16 year study of injuries to professional kickboxers in the state of Victoria, Australia. Br J Sports Med 37:448-451, 2003; Timm KE, Wallach JM, Stone JA, Ryan EJ: Fifteen years of amateur boxing injuries/illnesses at the United States Olympic Training Center. J Athl Train 28(4):330-334, 1993.

the ground, rapidly dilating pupils, a fixed gaze, and imminent respiratory failure.
- This injury is usually catastrophic, and death follows, the result of rapidly developing brain edema with herniation of the brainstem.
- The pathophysiology is suspected to be a loss of autoregulation of the brain's blood supply, leading to vascular engorgement within the cranium.
- This increases intracerebral pressure as the brain swells, and leads to uncal herniation of the temporal lobes and brainstem through the foramen magnum.
- The usual time for brainstem failure appears to be between 2 and 5 minutes.

Chronic Traumatic Brain Injury

BACKGROUND

- Literature reports of head injuries suffered in boxers range from 27% to 94% of all injuries.
- Most of these head injuries are minor, with contusions and lacerations being the most common.
- Anecdotal reports are legion that boxing contributes significantly to late and debilitating chronic brain injury; manifestations of injury from dementia resembling Alzheimer's disease and movement disorders similar to Parkinson's disease are among the most notable.
- This perspective is reinforced by the outcomes of studies conducted largely on retired boxers that were conducted retrospectively.
- However, conclusive evidence in the scientific literature about the risk factors associated with the development of injuries in either professional or amateur boxers is sparse.

EFFECT OF REPETITIVE TRAUMA

- Prolonged exposure to repeated concussive and subconcussive head impact is the major risk factor cited for chronic traumatic brain injury (CTBI).
- Fighters at risk appear to be those who have suffered a larger number of knockouts, those possessing inferior fighting and defensive skills, those who cumulatively suffered more powerful blows, and those suffering repeated concussions.
- There are compelling anecdotal data regarding risks and development of CTBI in older boxers; many boxers in the earlier 1900s had long careers spanning many decades and through greater than 1000 bouts.
- Further, many of that era had sparred and competed with fighters not matched by size nor skill, and most fought with 6-ounce gloves, which meant a greater force of impact per blow delivered.
- When sparring, there was no strict enforcement of the wearing of headgear.
- Many fighters, after their professional careers ended, became sparring partners who fought as many as 30 to 40 fights daily, adding more to the total injury burden suffered in their lifetimes.

BIOLOGIC MARKERS

- There are biologic markers that may indicate a person to be at risk for CTBI, namely the ApoE-epsilon-4 genotype.
- It appears that this genotype coupled with a greater number of heavy blows to the head may be multiplicative rather than additive in the genesis of CTBI.

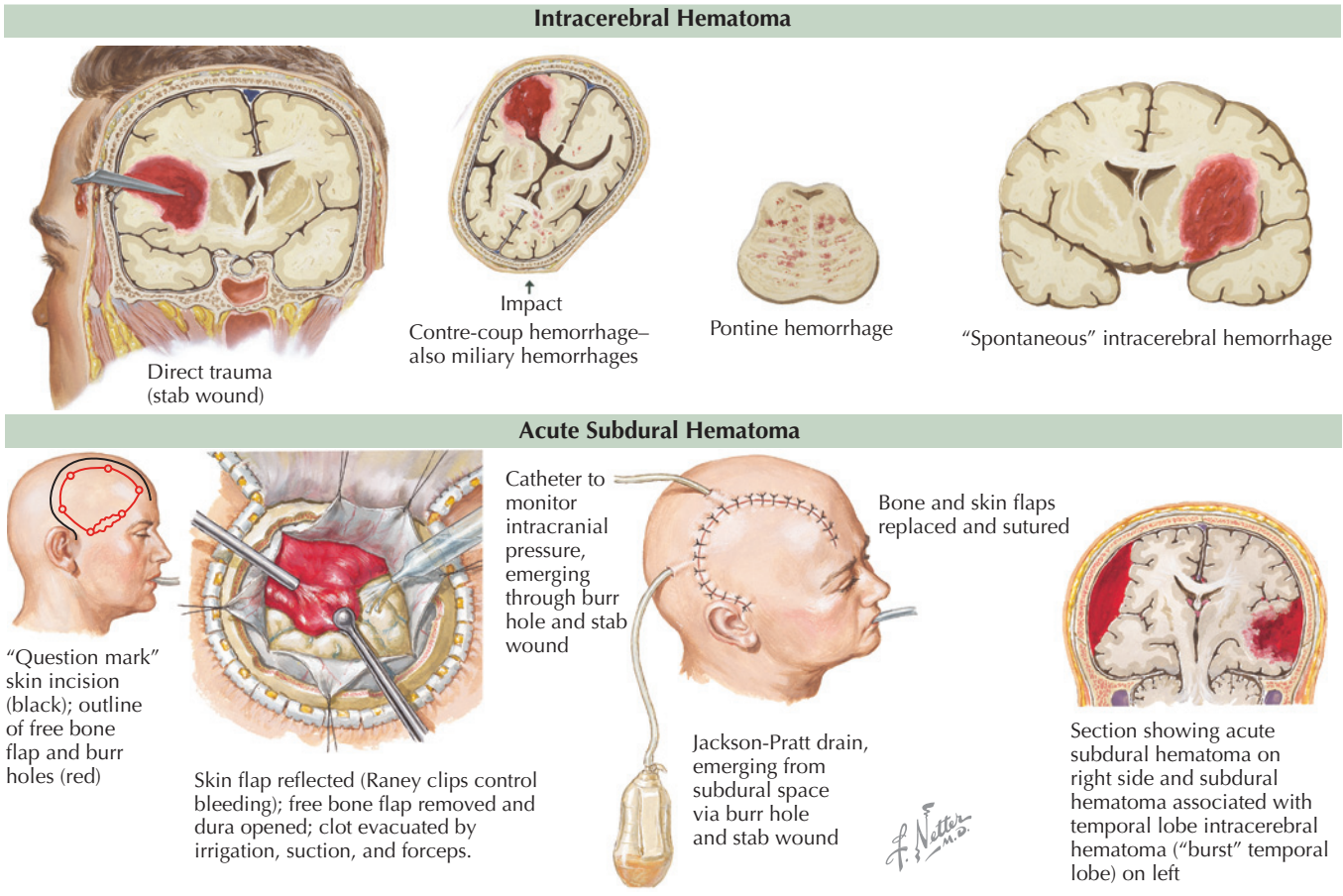

Figure 78-1 Acute Traumatic Brain Injury.

MONITORING BOXERS FOR CHRONIC BRAIN INJURY

- When considering chronic brain injury, it is imperative that the evaluating physician be attuned to subtle signs such as loss of skill or alterations of behavior.
- Maintain a high index of suspicion for this injury. An experienced and skilled fighter anecdotally should be able to avoid 90% of punches.
- As skill deteriorates, this percentage will obviously drop.

CAVUM SEPTUM PELLUCIDUM

- A cavum septum pellucidum (CSP) is an alteration to a membrane between the lateral ventricles, the presence of which has been indicated to be a marker of significant atrophy.
- It is claimed to imply in boxers a large number of knockouts and brain injury.
- John D. Spillane, MD, FRCP, was the first to note the presence of a cavum septum pellucidum in 1962.
  - He described the clinical and pneumoencephalographic findings in five retired professional boxers who had chronic neurologic problems in later life.
  - None of these fighters had fewer than 200 bouts.
  - In an autopsy study of 15 boxers who fought between 1900 and 1940 with the number of bouts per fighter ranging from 400 to 700, several findings have been put forth as markers for chronic traumatic brain injury, including abnormalities of the septum pellucidum, scarring of the cer-

ebellum and other areas of the brain, degeneration of the substantia nigra, and regional appearance of neurofibrillary tangles.

- However, according to other studies, there is no clear information regarding the persistence of the CSP in the general population at any time after the neonatal period.
- The presence of this lesion has been noted with an incidence between 0.7% and 37% at imaging studies.

NEUROIMAGING

Positron emission tomography (PET) scans: In fighters suffering chronic traumatic brain injury, hypometabolism is seen in the frontal and parietal lobes, similar to the patterns seen in Alzheimer's disease. This suggests similar pathology; indeed, it appears the outward signs and clinical progression are similar as well.

Magnetic resonance imaging (MRI) versus CT: MRI appears to be superior because it has superior ability to present detail of soft tissue. This expectation is true; MRI is superior in delineating contusions, subdural and white matter lesions, arachnoid cysts, and brainstem and cerebellar anatomy with greater detail and limited artifact.

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Dance

Craig C. Young, Selina Shah, and Laura M. Gottschlich

GENERAL OVERVIEW

Description

- Dance is an activity that is found in most cultures and dates back to ancient times.
- Although many sports medicine physicians think of dance medicine as ballet medicine, there are many other forms of dance, including ballroom, folk, jazz, and modern.
- Each of these forms has unique features and injuries.

Glossary of Common Terms

Arabesque: A pose in which the dancer stands on one leg and raises the other straight behind (at various angles); usually one arm is stretched out in front (Fig. 79-1).

Demi-pointe: Foot maximally plantar flexed with toes maximally extended—weight on metatarsal heads.

Foot positions: (see Fig. 79-1).

- First position—feet turned out with heels touching
- Second position—feet turned out with heels apart
- Third position—feet turned out, overlapping with right heel in hollow of left foot
- Fourth position—feet turned out, apart but with overlapping heels
- Fifth position—feet turned out, touching with right heel in front of left toe

Jeté: A jump in which the legs are in a split position in the air.

Plié: Bending of the knees and ankles with the legs turned out.

- Grand plié—large or deep plié in which the knees are maximally flexed (see Fig. 79-1)
- Demi-plié—"small" plié in which the knees are only partially flexed (see Fig. 79-1)

Pointe: Dancing while supporting the body on the tips of the toes (see Fig. 79-1).

Relevé: To rise up to the tiptoes or full pointe.

Turnout: Stance in which legs are rotated outward. Turnout is the sum of the external rotations of the hip, knee, tibia, ankle, and foot.

Class: Lesson.

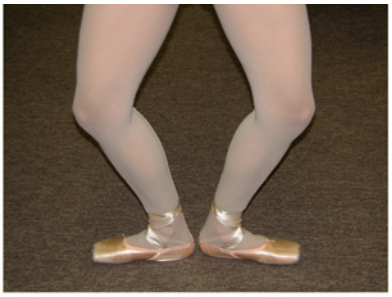
- Barre—first part of ballet class done using the railing for balance
- Center—the portion of class in which dancers perform dance movements in the "center of the room" without using the barre for assistance

TYPES OF DANCE

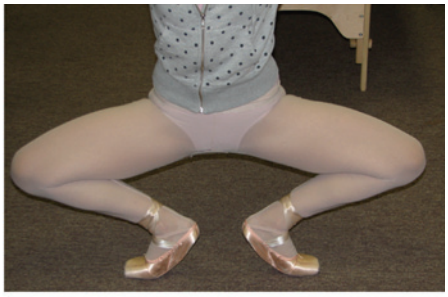
Ballet

- Classical ballet originated in the 1400s in Italy and blossomed in the 1600s in France.
  - Traditional ballet is a choreographed series of specific motions.
  - Many positions require extreme external rotation of the hips.
- Demographics
  - The average age for professional ballet dancers is 26 to 27.
  - Male body mass index (BMI): 21 to 22.
  - Female BMI: 18 to 19.
  - Females start taking dance class between ages 4 and 9.
  - Males start taking dance class between ages 12 and 16.
- Dancing on pointe consists of dancing while supporting the body on the tips of the toes.
- In traditional ballet the women wear pointe shoes that are handmade from lacquered satin and cardboard with stiff leather sole, which results in a hard but pliable shank and a rigid, unpadded toe box (Fig. 79-2).
- Traditional practice states that a girl needs to have a skeletally matured foot (usually 11 or 12 years old).
- In South America girls dance on the toes from age 4 or 5 with no apparent long-term sequelae.
- General rule of thumb—a young woman is ready to start pointe when she can do relevé passe and balance without needing to hold onto the barre for support. This movement can begin in fifth position; the working foot is drawn to the standing knee while the standing leg rises to balance on the metatarsal heads (see Fig. 79-2).

Arabesque.



Demiphlié.



Grandplié.

Basic foot positions—first to fifth from left to right.

On pointe (during relevé).

Figure 79-1 Common Terms in Dance. *(Photographs courtesy of Craig C. Young, MD, and Selina Shah, MD.)*

Ballet shoes: Pointe shoe cutaway-note lack of padding when compared to running shoe.

Ballet: Relevé passe.

Modern dancers.

Irish dance hard shoes.

Typical Irish dance position (arms tightly adducted to side and elbows locked in extension. Neutral posture is held from the waist up during all dancing and hands are closed into fists).

Figure 79-2 Types of Dance and Equipment. *(Ballet photographs courtesy of Craig C. Young, MD; Modern dance photograph courtesy of Blue 13 Dance Company, Ryuichi Oshimoto; Irish dance photographs courtesy of Laura Gottschlich, DO.)*

Jazz

- Jazz dance originated from the African-American dances of late 1800s and has beget tap dancing and some forms of ballroom (e.g., swing and Lindy).
- Traditional jazz shoes—soft leather with small heel.
- Jazz sneakers—soft leather/canvas with padded sole to increase shock absorption from jump landings.
- Positions usually require less external hip rotation than ballet.
- Jazz dancing is very free-form and includes kicks, falls, jumps, and slides and consists of quicker, sharper movements then ballet.

Modern

- Modern dance, also known as contemporary dance, originated at the turn of the 20th century in defiance of the rigidity of classical ballet.
  - Unlike ballet, modern dance uses gravity and emphasizes freedom of movement, sometimes taken to bizarre extremes.
  - Most professionals dance barefoot although a few wear some type of jazz shoe, ballet shoe, foot thong, socks, or simply use duct tape (see Fig. 79-2).
- Demographics
  - Average age for female professional modern dancers is about 30 and for males 31.
  - Male BMI: 23.6.
  - Female BMI: 20.6.
  - Most professional female modern dancers began their dance careers by studying ballet whereas the men began by studying modern.
  - Female modern dancers start taking dance class at 6.5 years of age.
  - Male modern dancers start taking dance class at about 15.6 years of age.
  - Professional modern dancers study various forms of dance outside of the time they spend in rehearsal for their companies, including ballet, pointe, jazz, tap, hip hop, African, and ballroom.
  - They spend an average of 8 hours taking various types of dance classes and about 17 hours in rehearsals for their companies.
  - Most also spend about 2 to 3 hours per week doing some form of exercise outside of dance such as yoga, pilates, gyrotonics, weightlifting, running, biking, and walking.

Ballroom

- Ballroom dancing is a set of partner dancer dances that were initially developed as social dances.
- Traditionally the purpose was to show off the woman, who does all the fancy moves while the male acts as support as well as the leader.
- Women wear high heels.
- Types of ballroom:
  - Smooth (waltz, foxtrot, tango)
    - These dances, especially waltz, involve lots of extension and flexion from the knees to create the rise and fall of the dancers.
  - Latin (cha-cha, rumba, samba, jive, paso doble)
    - Typically these dances emphasize hip motions.
  - Rhythm (East Coast swing, mambo)
  - Dance sport—competitive athletic-style dancing based on all of above styles that typically involves more lifts, throws, and slides than traditional ballroom dances.
  - Other ballroom dances include polka, two-step, Lindy, and West Coast swing.

Folk

A highly variable type of dance—depends on individual style.

Irish

- Earliest recording in literature of Irish dancing was in 1300.
- It migrated to England and then to Australia, Canada, and the United States.
- Schools are now established in 10 mainland European countries, South Africa, Israel, Taiwan, and the United Arab Emirates.
- Combines artistic elements of ballet with the rhythm of jazz and tap.
- Has had an important influence on American dancing—including square dancing, clogging, jazz, and tap.
- Has a competitive and a performance element.

- Shoes consist of a soft leather, lace-up slipper (soft, or reel, shoes) and a leather shoe with fiberglass tips and heels (hard, or jig, shoes) (see Fig. 79-2).
- Shoes are purchased to be extremely snug to give a tight toe appearance in plantar flexion.
- Competition
  - Compete at a local (Feis), regional (Oireachtas), National, and World (Rince Na Cruinne) Level
  - Must qualify by placing highly at local and regional competitions to attend National and World competitions.
  - Children divided by:
    - Age as of January 1
    - Sex
    - Level—Beginner, Advance Beginner, Novice, Prize Winner or Open Solo, Preliminary Championships, and Open Championships. Dancers progress from level to level by winning awards at a local Feis.
  - Competition may be solo or in groups up to 16 dancers.
  - Dancers compete in multiple dances in both hard (Treble Jig, Hornpipe, Set Dances) and soft shoes (Reel, Slip Jig, Jig, Figures).
  - Dancers are judged on timing to music, posture, position of feet and legs during execution of the dance, and style.
  - In order to compete, a dancer must be enrolled in a school with a TCRG (e.g., rinci gaelaca do teagasc) licensed teacher.
- Costumes
  - Female dancers must wear traditional dresses in competition.
  - Boys must wear pants or a kilt with a dress shirt and tie.
- Biomechanical principles
  - Some similarities between ballet and tap dancing.
  - Most elements of dancing are performed in the sagittal plane.
  - Hamstring flexibility must be enough to afford approximately 0 to 135 degrees hip flexion.
  - Dancing is performed in external rotation from the hip to approximately 70 degrees.
  - Front foot is routinely crossed in front of back foot, covering back toe during most dance moves and in tight plantar flexion.
  - Front knee is routinely covering back knee during most dance moves.
  - Most dancing is performed with arms tightly adducted to side and elbows locked in extension. Neutral posture is held from the waist up during all dancing and hands are closed into fists (see Fig. 79-2).
  - Most dance moves are performed with rigid landing in point/demi-point positions and the knees tightly extended.
  - Repetitive pounding of the balls of the foot occurs for minutes at a time during rhythm moves with the hard shoes and landing from jumps.
  - Turning of head for spotting for jumps and spins is discouraged.
  - In the late 1980s, dancers began to dance on pointe in their hard shoes, often jumping, spinning, and running on the fiberglass tips.
  - In 2002, the governing body of Irish dancing, An Coimisiun, banned toe or "block" movements for all children under the age of 12, citing concern over stress on immature bone with pointe movements.

Aerobic

- Differs from other form of dance because its purpose is primarily exercise.
- Emphasis is on quick motions to raise heart rate and tone muscles rather than artistic form and performance.
- Exercisers wear shock-absorbing athletic shoes.

INJURY EPIDEMIOLOGY

Modern Dance

- Up to 77.5% of modern dancers suffer at least one injury per year.
- There are 0.59 reported injuries per thousand hours of dancing.
- Annual incidence of injuries per male is 1.24 and per female is 1.65.
- Although most dancers have at least one injury per year, most do not miss any performances.
- They admit to returning to dance class as soon as possible even though they are in pain.
- Dancers may take 2 to 3 weeks off before returning to limited dancing and usually dance pain free by about 1.5 to 2 months later.
- In general, male dancers miss fewer classes and rehearsals than female dancers.
- Most common injury locations
  - Foot and ankle
  - Low back
  - Knee
- Injury types
  - Most injuries result from overuse or gradual onset rather than a result of an acute or traumatic event.
  - Most dancers suffer muscle strains, followed by ligament sprains, and then chronic inflammatory processes.
- Causes of injuries
  - Self-pressure, the demands of the role, demands from the choreographer, and ignoring pain and fatigue are the most common perceived causes of injury.
  - The characteristics of the floor (e.g., surface, resilience) are also felt to contribute to injury.
  - Dancers state that performing on raked stages (floor is angled down toward the audience for better viewing) leads to more injuries.
- Injury consultation
  - Most dancers will consult with someone within 1 week of injury.
  - Fewer than half of the dancers will consult with a physician regarding the injury because they do not think that physicians are helpful, are understanding of them as dancers; or the dancers do not have health insurance, or are worried that they will be told to stop dancing for too long.
  - Many dancers consult other health care providers such as the company physical therapists, chiropractors, massage therapists, and acupuncturists.
  - Many will also discuss their injuries with a member of the company such as the choreographer, company director, or instructor.
  - Most dancers actually adhere to the advice given to them.
  - Reasons for not adhering to the advice given to them include the lengthy amount of time recommended to refrain from dance, fear of being held out of class or rehearsal if the staff knew about the injury, not agreeing with the advice, and fear of losing their role in the performance to an understudy or rival.

Ballet

- 75% to 95% of ballet dancers suffer at least one injury per year, with an average of 2.97 to 3.2 injuries per dancer per year.
- Most common injury locations
  - Foot and ankle
  - Low back
  - Knee
- Injury types
  - Most injuries are muscle strains, followed by ligament sprains and chronic inflammatory processes.

- Intrinsic risk factors for injury: anatomic structure, inadequate strength and flexibility, improper technique, nutrition, previous injury, fatigue.
- Extrinsic risk factors for injury: choreography, cold environment.
- Students in summer intensive programs at high risk for injury.

Irish Dancing

- Lack of research in this area of dance.
- Most common areas of injury: ankle, foot, hip, knee.
- History is taken in normal fashion, but keep in mind the following issues specific to this type of dance:
  - "Studio"—50% of dance schools are not in traditional studios, but often school gyms and cafeterias
  - What the dance surface is made of
  - What type of shoe is worn for practice
  - How many hours are spent in solo class, team class, and private lessons; the dancer's strength

MEDICAL PROBLEMS

- Eating and nutritional disorders
  - Very common especially in adolescent dancers.
  - Professional female dancers believe that they weigh more than they should, even though audience members believe they appear to be of appropriate weight for dancers.
  - Problems may start in preadolescent dancers.
  - Increase risk of stress fractures.
- Menstrual disorders
  - Primary amenorrhea and secondary amenorrhea are common in dancers.
  - Increase risk of stress fractures.
- Smoking
  - Higher prevalence in dancers compared to other athletic populations.
  - Especially at risk—female dancers who are having difficulty maintaining their weight.
- Burnout
  - Beware especially in adolescent folk dancers who present with recurrent atypical injuries.
- Osteopenia
  - Amenorrheic female dancers at risk.
  - Eumenorrheic female dancers and male dancers tend to have higher bone densities than nonathletes.

MUSCULOSKELETAL INJURIES

- Core stability tests (30+ seconds)
  - Pike position—push up with forearms supporting (elbows bent 90 degrees) and on toes with ankles in neutral
  - Side lift—hold trunk linear facing side support on forearms
- Spine
  - Neck and back strains (especially trapezius, rhomboid, and latissimus).
  - "Incorrect" lifting technique—remember that choreographers and artistic directors often value appearance over optimal lifting technique.
  - Other back problems—spondylolysis, spondylolisthesis, scoliosis, and facet syndrome.
- Shoulder—rotator cuff problems, particularly in male dancers.
- Hip

Piriformis syndrome
- Pain deep in gluteal or groin region.
- Pain worsens with stair climbing, standing up, or prolonged sitting.
- Pain with extreme passive hip internal rotation (flexed approximately 90 degrees) or resisted hip external rotation.

Snapping hip syndrome
- In most nondancers, snapping hip syndrome is of the external type, which is caused by the iliotibial band (ITB) snapping over the greater trochanter.
  - Dancers with external snapping hip syndrome complain of snapping when landing jumps.
- Internal snapping hip syndrome is common in dancers and is caused by iliopsoas.
  - Usually in non–weight-bearing gesture leg (e.g., when bringing the hip from an externally rotated, abducted, flexed position to neutral extended position).
  - Reproduced by bringing hip from flexed, externally rotated position to extension with internal rotation.
  - Sometimes reproduced with resisted straight knee sit-up.
  - Both can be clinically diagnosed and, if needed, confirmed with ultrasound.

Hip labral tears
- Sharp, painful catch in groin.
- Confirm with magnetic resonance imaging (MRI) arthrogram.

Sartorius tendonitis
- Etiology: overuse as an external rotator of the hip.
- Pain with external rotation, full flexion, abduction of hip.

Rectus femoris tendinitis
- Use in forward extension of leg (e.g., battement or développé devant).
- Groin pain, especially with knee extended.
- Knee
  - Patellofemoral pain syndrome
  - Patellar tendonitis
- Leg
  - Tibial stress fracture
    - Avoid rodding—this may cause loss of motion at knee; drilling out fracture a better option.
  - Medial tibial stress syndrome
- Ankle
  - Achilles tendonitis
    - Ribbon friction—beware of where ribbons from slippers cross the achilles tendon (Fig. 79-3).
    - Character shoes—often do not have achilles tendon notch and may not have optimal sizing because the wardrobe department may have limited costumes (see Fig. 79-3).
  - Ankle sprains
  - Ankle impingement
    - Anterior: Especially aggravated by pliés; often associated with osteophyte at margin of anterior tibia and dorsum of talus.
    - Posterior: Especially aggravated by pointe and demi-pointe; often associated with os trigonum or prominent posterior process of talus.
    - Evaluation: Lateral foot x-ray in maximal plantarflexion (for anterior impingement) or maximal dorsiflexion (for posterior impingement) (see Fig. 79-3).
    - Treatment: Although surgery in dancers is often career-ending, surgery for ankle impingement may be career-saving.
  - Peroneal tendonitis, subluxation, dislocation, and dysfunction
  - Tarsal tunnel syndrome
- Foot
  - Cuboid subluxation
    - Plantar subluxation of cuboid.
    - Risk factors include tendency to be in valgus position of forefoot at metatarsal joints.
    - Can occur secondary to lateral ankle sprain.
    - Symptoms: Lateral foot pain, weakness with push-off, inability to jump because of sharp pain.
    - Diagnosis: Pressing dorsally on cuboid from plantar surface reproduces pain and possible visible depression. Radiographic studies usually unsuccessful

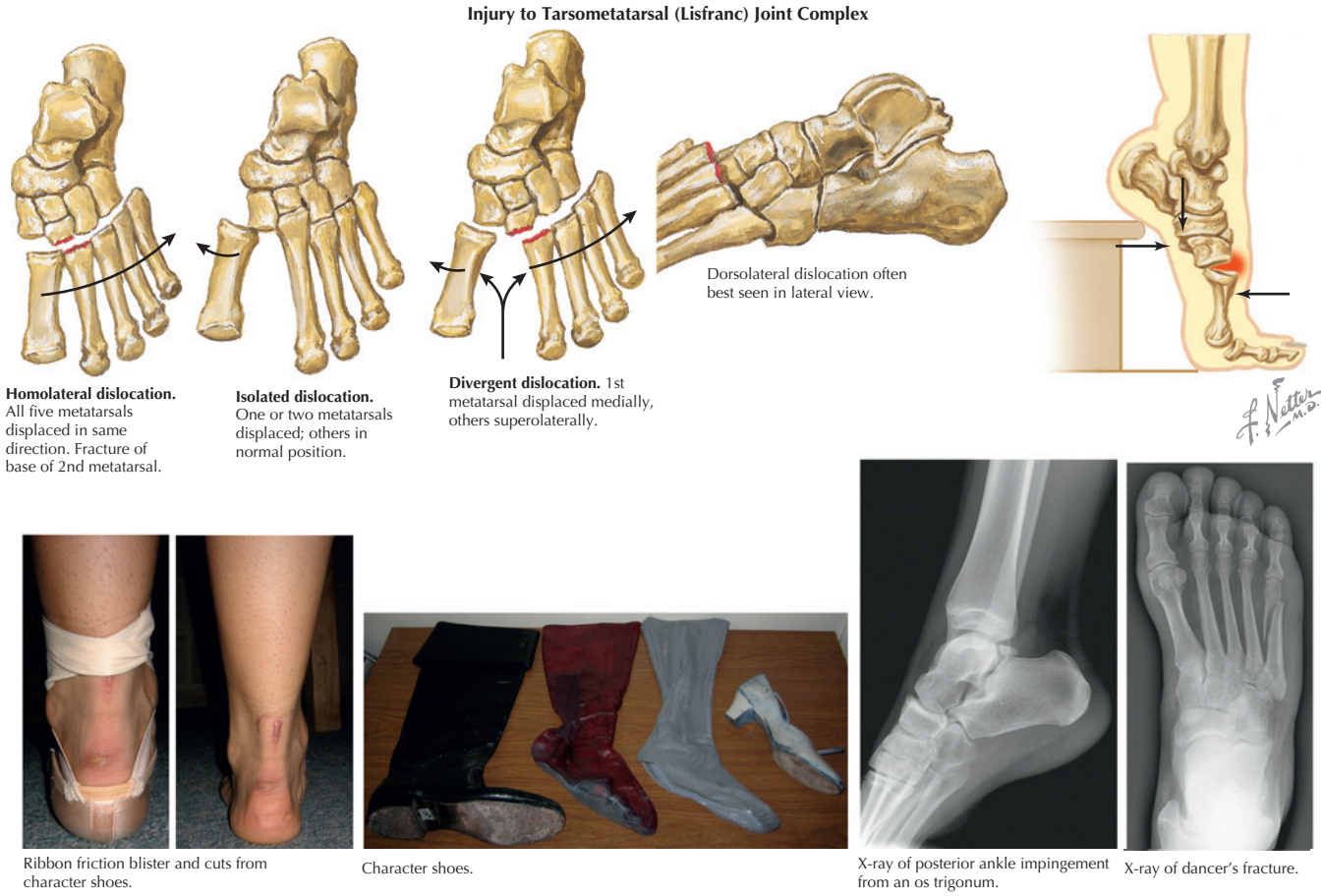


Figure 79-3 Musculoskeletal Injuries in Dance. *(Photographs © Craig C. Young, MD.)*

- Subtalar coalition
  - Bony connection that is between the calcaneus and either the navicular or the talus.
  - Limits foot motion.
  - May cause mechanical pain.
  - Usually affects young dancers.
- Navicular stress fractures
- Lisfranc joint injuries
  - Lisfranc joint: Articulation of second metatarsal with first and second cuneiforms (see Fig. 79-3).
  - Lisfranc joint has limited mobility.
  - Pointe dancers at highest risk.
  - Lack of dorsal support and immobility of second metatarsal causes dorsal displacement of second metatarsal with axial load in plantar flexion.
  - Sprain: Disruption at Lisfranc joint.
  - Fracture/dislocation: Diastasis between first and second metatarsal seen in standing anterior-posterior radiograph of foot. Line from medial edge of fourth metatarsal to cuboid is disrupted as seen on standing medial oblique view of foot.
- Spiral fracture of shaft of fifth metatarsal (dancer's fracture) (see Fig. 79-3).
  - Etiology: Inversion injury while on pointe.
  - Treatment: Cast shoe.
- Metatarsal shaft stress fractures
  - Usually in second to fourth metatarsals.
  - Especially in the longest toe.
- Flexor hallucis longus tendonitis (dancer's tendonitis)
  - Usually occurs in pointe dancers.
- Morton's neuroma and corns
  - Silicone spacers; check shoe size, especially pointe shoes; remember that each pair is handmade, thus variable in size.
- Bunion (hallux valgus), hallux rigidus, and hammertoes
  - Bunions are particularly a problem if the great toe is longer than second toe.
  - Avoid bunion surgery—minimal loss of motion may result in end of dance career.
  - Hammertoe usually occurs in second toe, particularly if it is the longest toe.
  - Shoe padding to help spread the load on the toes.
  - Silicone pad (great toe) or sleeve (toes).
  - Orthotic with rigid extension in non-dance shoes (rarely fits into dance footwear except some character shoes).
- "Stone bruise"—metatarsal head contusion
- Paronychia and subungual hematoma
  - Range of appropriate nail length small; too short paronychia/ingrown nails; too long subungual hematoma/nail trauma.
  - File nails daily to keep at optimal length.
- Sesamoiditis and sesamoid fracture
  - Especially common in folk dancers.
- Plantar fasciitis and turf toe
  - Especially common in Irish dancers.
- Calluses and corns

GENERAL TIPS FOR TREATING DANCERS

- Evaluate the dance floor for overuse injuries.
  - Sprung floors have the best impact absorption.
    - This is particularly important because dance shoes have little or no cushioning.

  - Dancers may use different floors in the class area, as opposed to performance areas.
  - Dancers in smaller programs and folk dancers are much less likely to have the luxury of dancing on sprung floors.
- Evaluate footwear worn when dancer is not dancing.
  - Alteration of this footwear may allow the dancer to continue dance activities by allowing for more effective relative rest when not dancing.
- If you don't understand the names of the motions, have the dancers describe or demonstrate them.
  - Knowledge of aggravating motions is critical in designing rehabilitation and return-to-activity plans.
- In general, avoid surgery in dancers because even minimal loss of range of motion may result in the end of a dance career.
  - Exceptions are "excisional operations" (e.g., excision of os trigonum, osteophyte shaving for ankle impingement syndrome).
  - Dancers also recover well from meniscus and hip labral surgery.
- Respect dancers as artists, but also encourage them to think of themselves as athletes who need proper conditioning, nutrition, and rest.

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Mass Participation Endurance Events

80

William O. Roberts

GENERAL CONSIDERATIONS

Overview

- This chapter develops an algorithm for management of mass participation endurance events.
- Medical director is safety and health advocate for athletes who participate in race.
- Safety of athletes is primary purpose of race medical operation.

Events

- Road running
- Cycling
- Cross-country skiing
- Triathlon
- Wheelchair
- Swimming

Approach as a "Planned Disaster"

- Mass participation events should be approached as a "planned disaster" (potential mass casualty incident).
- Mass gatherings always have potential for medical illness or injury.
- Potential casualties occur in two groups of people: participants (literature review allows estimate of injury type and incidence; individual race experience allows more accurate estimate) and spectators.
- Endurance events share common injury and illness risks that must be addressed for medical management, but each event will have a unique injury and illness profile.
- Participant safety is primary goal of race committee.
- A comprehensive medical plan will decrease the community burden and reduce the potential for emergency room overload.

Incidence and Risk

- Estimating medical encounters
  - Anticipated starters multiplied by encounter incidence.
  - Project needs: staff, supplies, equipment.
- Risk ranges
  - Running (42 km): 0.5% to 20% risk of injury.
    - Twin Cities Marathon (Minnesota): 0.5% to 3% (average 1.89% for entrants in 1983-1994).
    - Boston Marathon: 5% to 12% (recent races 1.6% to 6%).
  - Running (<21 km): 1% to 5% for Falmouth Road Race (Massachusetts); less than 1% but severe casualties.
  - Triathlon (225 km): 15% to 35%.
  - Cross-county skiing (55 km): 5%.
  - Triathlon (51 km): 2% to 5%.
  - Cycling (variable): 5%.
- Variables and unknowns: weather, event distance, event type, condition and acclimatization of participants.
  - Influence of increasing heat and humidity on marathons:
    - Medical encounters increase and race times slow.
    - Race dropouts increase.
  - Heat limits
    - Cancel at 82° F wet bulb globe temperature (WBGT) is recommended by the American College of Sports Medicine (ACSM) guideline.
    - Unpublished data implies cancelling at WBGT near 70° F may be better for nonelite runner safety and community emergency response load, especially for unacclimatized participants.
    - The cancellation level may be event specific, but the number of medical encounters and nonfinishers seems to accelerate with WBGTs in the mid to high 60s° F. Elite runners seem to tolerate hotter conditions and races may elect to run the elite racers while cancelling the nonelite race.
    - Calculate WBGT using formula (available at www.zunis.org) based on ambient temp, relative humidity, time of day, and sky cover.

Anticipating Casualty Types

- Exercise-associated collapse (EAC) is most common: hyperthermic, normothermic, and hypothermic.
- Low-frequency but potentially fatal medical emergencies can occur, including cardiac arrest, exertional heat stroke, exercise-associated hyponatremia, asthma, insulin shock, anaphylaxis, high-velocity, or impact trauma.
- Macrotrauma: musculoskeletal (fracture, dislocation, sprains and strains, contusions), vascular (closed, open), head and neck (concussion, intracerebral bleed, fracture-dislocation), visceral organs (contusions, laceration, rupture).
- Microtrauma: tendinitis, stress fracture, fasciitis.
- Dermatologic trauma: blisters, abrasions, lacerations.
- Drowning, near drowning in water-based events.

Race Medical Operations Purpose

- Prerace
  - Develop strategies to improve competitor safety and reduce race-related injury and illness.
- Race day
  - **Primary:** stop progression of injury or illness; evaluate casualties (triage, treatment, transfer); reduce community medical burden.
  - **Secondary:** prevent emergency department overload.

Role in Race Operations

- Event and runner safety
- Medical decisions
- Medical spokesperson
- Executive committee administrative functions

PREVENTION STRATEGIES

- **Primary**
  - Definition: prevent or reduced occurrence of casualties, reduce severity of casualties.
  - Passive: cooperation of participants not required. Examples: start times, course modifications, traffic control.
  - Active: cooperation or self-initiated behavior change required. Examples: education, safety advisories.
  - Enforced active: helmets, wetsuits (required).
- **Secondary**
  - Definition: early detection of injury or illness; intervention protocols to stop progression.
  - Examples: impaired runner policy: advanced cardiac life support (ACLS), advanced trauma life support (ATLS), or EAC protocol; on-course ambulance; finish line triage.
- **Tertiary**
  - Definition: treatment and rehabilitation of illness or injury.

- Examples: emergency department transfer, hospital admission, rehabilitation center.

PREPARATION

Race Scheduling

- Location (latitude and longitude)
- Season of year
- Safest start and finish times (if average high temperature is > 60° F, schedule start at sunrise)
- Time limits for competitors to be on course

Competitor Safety

- Consider the athletes' safety first in all race-related decisions.
- Use the safest start and finish times for both elite and nonelite competitors.
- Determine hazardous conditions and develop a written race administration plan to simplify decisions on race day.
  - Ensure volunteer and competitor safety.
  - Define heat, cold, traction, wind, wind chill, lightning, and torrential rain race limits.
  - Alternatives: alter, postpone, cancel.
  - Publish protocol in advance.
  - Announce risks at start.
- Impaired competitor policy
  - Define an approach to athlete who appears ill or injured during competition, especially with regard to fluid balance abnormalities and heat or cold stress.
  - No disqualification for medical evaluation. Most event rules allow medical assessment of athletes who appear ill without automatic disqualification and allow athletes deemed fit to continue participation. This is especially important for citizen-class (nonelite) runners.
  - Criteria to continue event participation: oriented to person, place, and time; straight line progress toward finish; good competitive posture; clinically fit appearance.
  - Publish policy in advance.
- Emergency department (ED) notification: Notify local EDs of date, time, and duration of event; also estimate of numbers and types of possible race casualties.
- Preparticipation screening
  - Decide whether event should require pre-event medical screening: Will it improve safety of participants? Will it be cost-effective? Will it protect event and volunteer staff from liability?
  - Generally not recommended beyond usual health screening and interventions by participant's personal physician based on risk factors and symptoms.
- Competitor education: safety measures, risks of participation, fitness level recommended for participation, hydration and overhydration (ingestion of adequate fluid to nearly replace sweat losses without excessive intake, knowledge of sweat rate, drink to thirst), volunteer identification (standard colors, visibility), nutrition.
  - Medical information on back of race bib should include training weight, prerace weight, allergies, medications, chronic medical problems, and emergency contact phone number.
  - Medical alert tags should be worn during race.
- Child and adolescent participation in endurance events: There is no data to support banning under 18 participation for medical reasons and children as young as age 7 have completed marathons without reported adverse effects. A motivated child (not parent), who is growing physically, physiologically, psychologically, and socially during training, should be allowed to participate if the race or event does not ban participation for administrative reasons.

Course

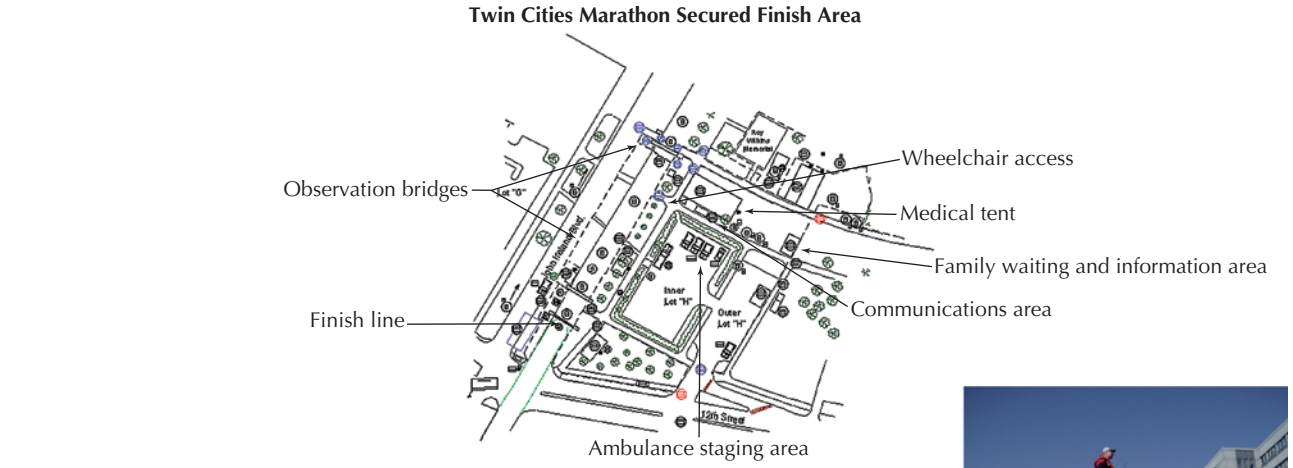
- Course survey: hills, turns, immovable objects; traffic control; altitude changes; open water (participant safety and environment influences).
- Start: downhill increases risk for wheelchair competitors; wave starts to relieve congestion and associated risks of falling.
- Aid stations
  - Major: full medical care; equipped and staffed for most anticipated problems.
  - Minor: comfort care, fluids, first aid, shelter.
  - Location: start; every 15 to 20 minutes along course; finish line.
  - Rolling aid: vehicle (bus or van) equipped and staffed to deliver medical care for expected injuries along course; requires an open lane on the course for the vehicle(s).
  - First-response teams: motorcycles or bikes; automatic defibrillator and first aid.
- Finish area (Fig. 80-1)
  - Triage: chute, postchute, and area triage (sweep teams).
  - Field hospital: major aid station (see Fig. 80-1). Subdivisions may include triage, intensive medical, intensive trauma, minor medical, minor trauma, skin, medical records.
  - Ambulance support for ED transfer (see Fig. 80-1).
  - Shelter for well finishers.
  - Dry clothes shuttle; consider clothes dryer for wet or cold conditions.
  - Fan out finish line area to spread out participants in hot races.
  - Elevated spotters to identify downed participants in crowded areas (see Fig. 80-1).

Transportation

- Well dropout competitors
  - Prevent new or increased previous injury by transporting to shelter or reducing repetitive stress: hypothermia, hyponatremia, stress fracture, strain.
  - Examples: vans, buses, golf carts, gators, snowmobiles, snowcats, public transportation, sled, toboggan, boat.
- Ill or injured competitors
  - Prevent progression of illness or injury—overuse and acute injury or illness that precludes completion of event without increasing individual morbidity or mortality.
  - Access care for more severe acute illness or injury. Minor injury can use well dropout transportation. Casualties requiring medical care need transport by ambulance to nearest ED or event medical station.
  - Examples of medically equipped vehicles for ill dropout competitors: advanced (ALS) or basic (BLS) life support ambulance, life flight helicopter.
- Finish area
  - Access medical care in finish area.
  - Examples: wheelchair, litter, stretcher, manned carriers (Fig. 80-2).
  - Access tertiary care by ambulance: ALS, BLS.

Communications

- Type: phone (cellular/digital, hard wire), ham radio systems, 2-way radio systems. Three-tiered system provides overlap and backup if a single system fails.
- Location: start; course (aid stations, pickup vans, course spotters, ambulance, race volunteers with cell phones form "line of site" spotter network to blanket course); finish area (field hospital [central dispatch for course], triage teams).
- 911 access: any volunteer to summon an ambulance, but must know exact location.




Interior of medical tent.


Ambulance support.


Elevated spotter to identify downed participants.

Figure 80-1 Finish Area of Mass Participation Events.


Finish area should be equiped with wheelchairs, litters, stretchers, and manned carriers.

Figure 80-2 Transportation at Finish Area.

- Give runner number to ensure that dispatched ambulance responds to the correct person. Notify central dispatch of pickup and disposition.

Fluids and Fuel

- Type: water, carbohydrate-electrolyte solutions, high-carbohydrate foods.
- Location: start, aid stations, finish area (postchute area, medical area).
- Amount: 6 to 12 ounces available for each competitor every 15 to 20 minutes. Double for start, finish, and transition areas. Total volume and course distribution can be estimated from similar events or past race needs. Competitors should know their individual needs based on sweat rate.
- Publish in advance: fluid and food types, locations; risks and symptoms of under- and over-hydration; calculation of personal sweat rate (measure nude weight; run at race pace for an hour in expected race conditions; strip down, towel off, and reweigh nude; difference in weight is the maximum fluid volume to be replaced every hour (sweat rate per hour).

Equipment

- Shelter (tents, vehicles like school busses, buildings)
- Security fencing
- Cots, chairs, tables
- Heating and cooling equipment
- Generator
- Defibrillator
- Back boards
- Lights
- Portable sink

- Toilet
- Point of care lab measuring devices for serum sodium, BUN, potassium, hematocrit, glucose, oxygen saturation (if available)

Supplies

- Medical
- Trauma
- Intravenous fluids (normal saline [NS] or 5% dextrose in NS)
- Medications (albuterol, epinephrine 1:1000, dextrose 50% in water, oxygen, diazepam or midazolam, cardiac arrest drug kit, 3% NaCl solution, others)

Staffing

- Personnel located at start, over course, at finish
  - Physicians
  - Acute care nurses:
    - Intensive care unit
    - Coronary care unit
    - ED
  - Paramedics
  - Emergency medical technicians
  - Athletic trainers
  - First-aid personnel
  - Nonmedical assistants
  - Physical therapists
  - Sources for volunteers: hospitals, clinics, American Red Cross, National Ski Patrol, National Mountain Bike Patrol, National Guard, Armed Forces Reserves, medical personnel training centers

Medical and Race Records

- Document care
- Calculate incidence of casualties
- Project future needs
- Research injury/illness patterns
- Design system to easily document care for common problems (Fig. 80-3)

2007 MTCM MEDICAL RECORD – CONFIDENTIAL Marathon / 10 Mile/5K **Location:** Medical tent / Aid station mile_____

Arrival time_____

Race #_____ **Name** _____ **Age**_____ **Gender** M / F **Finish time:**_____/ **DNF**

Pre-Event injury / illness: Y / N Describe _____ # Previous marathons _____ Best time _____

Medical History:

Symptoms: Exhaustion Fatigue Lightheaded Hot Cold **Mobility:** Independent With assistance Wheelchair

Mental Status: Alert Confused Unresponsive **Orientation:** Person Place Time **Neuro symptoms:** Headache Syncope Weak

Cardiac symptoms: Chest pain Tachycardia Palpitations **Resp symptoms:** SOB Wheeze Cough **Skin:** Hot Cold Sweaty Dry

GI status: Nausea Vomiting Diarrhea Stomach cramps **Muscle cramps:** **Y / N** Location: Calf Thigh Abdomen Back

Other:_____

Skin, Bones, & Joints

Complaint: Pain Blister Abrasion Bleeding Swelling Other_____**Tissue:** Skin Muscle Tendon Ligament Bone

Location: Toe R / L Foot R / L Ankle R / L Calf R / L Knee R / L Thigh R / L Hip R / L Back R / L (upper/lower)

Other:

Time	Temp (rectal)	BP	Pulse (r/ir)	Glucose check	Meds/Rx/Additional labs

Lab: **O_2 Sat** (ra) _____% **Na^+** _____ **K^+**_____ **Hct**_____ **BUN**_____

Treatment:

Leg elevation

PO Fluids:_____ **IV Fluids:** **IV #1** 1L D_5NS_____ time **IV #2** 1L D_5NS or NS_____ time **IV #3** 1L D_5NS or NS_____ time

$D_{50}W$: #1_____ time #2_____ time

Musculoskeletal: Ice pack / Compression / Stretching / Massage / Phys Rx / Other

Skin: Prep / Lance / Bacitracin / Dressing

Treatment Refused

Discharge status: Home / ER transfer (ER Follow-up: Admit/Home)

D/C instruction sheet: Blister / Fluids

Diagnosis: Blister Abrasion Sprain Tendinitis Strain Fracture (suspected)
EAC: Hyperthermic: Normothermic: Hypothermic- mild / mod / severe
Exercise Associated Muscle Cramps
Other _____

Notes: (Continue on opposite side)

Discharge time _____

Signature:_____ **MD / DO / DPM / RN / EMT / ATC** ©Wm Roberts MD

Figure 80-3 Medical Record. *(Reprinted with permission from William O. Roberts, MD, MS, FACSM.)*

MEDICAL PROTOCOLS

- **First aid:** do no harm; stay within training level.
- **Basic problems:** exercise-associated collapse, low-frequency medical emergencies, trauma, repetitive use, and skin injury.
- **Initial assessment of collapsed athlete (ABCDE): a**irway (cervical spine control), **b**reathing, **c**irculation (hemorrhage control), **d**isability (neurological status), **e**xposure and exam.
- **Initial disposition:** race medical facility, transport to emergency facility. Decide if on-course problems are triaged in race medical facility or moved directly to nearest ED. This will depend on both the medical capabilities and the accessibility of the race facility.
- **Treatment and transfer protocols**
  - Determine in advance automatic transfers (cardiac arrest, respiratory arrest, shock, symptomatic hyponatremia, severe trauma) and delayed transfers.
  - Keep protocols simple.
  - Integrate into emergency medical services (EMS) protocol.
  - Decide on level of care (first aid vs. medical treatment) in the finish area and course aid stations
- **Medical precautions**
  - Exposure to body fluids (blood, stool, emesis; *not* sweat) can transmit disease.
  - Modified universal precautions are most frequently used: handwashing, gloves, no food or eating in treatment areas.
  - Risks: hepatitis B, C, D, others; AIDS/HIV.
  - Disposal of contaminated waste: Red Bag, sharps containers.
- **Adverse event protocol**
  - If medical event has adverse outcome resulting in death or catastrophic injury, a predetermined protocol should be in place to communicate with family, public, and press.
    - Event is reported to medical director and head of event administration.
    - Event should not be discussed with or by volunteers outside immediate need for medical care.
    - Medical director or designated alternate should present incident to press.
  - Event administration and medical director should keep detailed records.

EXERCISE-ASSOCIATED COLLAPSE (EAC) CLASSIFICATION AND TREATMENT SYSTEM

- **Based on symptoms and signs of collapsed finishers**
  - Simple treatment protocols should be in place.
  - Weather influences injury patterns and warmer weather increases medical encounters and participant drop out.
  - Clinical classification system: varied presentation within each temperature class; symptoms do not reflect body temperature; similar treatment for all classes; rapid recovery for most victims.
- **Definition of EAC:** requiring assistance during or after endurance activity; not orthopedic or dermatologic.
- **Etiology:** undetermined; hypotheses include sudden loss of muscle pump blood flow from legs (secondary heart function of legs during exercise), vasovagal response, depletion of energy store, dehydration, central nervous system failure, internal fluid shifts.
- **Diagnosis:** presence of signs or symptoms. Major criteria: body temperature, mental status, ambulation status.
- **Clinical picture** (derived from clinical presentations of Twin Cities Marathon casualties)
  - Symptoms: exhaustion, fatigue, hot, cold, nausea, stomach cramps, lightheadedness, headache, leg cramps, palpitations.
  - Signs: abnormal body temperature, unconsciousness, altered mental status, central nervous system changes, inability to walk unassisted, leg muscle spasms, tachycardia, vomiting, diarrhea.
- **Classification scheme**
  - Types: **hyperthermic** (body temperature ≥ 103° F [39.5° C]), **normothermic** (temperature between (97° F [36° C] and 103° F); **hypothermic** (temperature ≤ 97° C).
  - Severity rating: **mild** (any symptom or sign, walk with or without assistance, alert, systolic blood pressure greater than 100 mm Hg, heart rate less than 100 beats per minute, weight loss < 5%), **moderate** (no oral intake, extra fluid loss, unable to walk, severe muscle spasm, weight loss 5% to 10%, temperature ≥ 105° F [40.5° C] or ≤ 95° F [36° C]), **severe** (central nervous system changes, no oral intake, extra fluid loss, unable to walk, severe muscle spasm, weight loss 5% to 10%, temperature ≥ 106° F [41° C] or ≤ 90° F [32° C]).

Management Protocol

Diagnosis and Documentation

- Initiate medical record; record presenting symptoms and medical history.
- Record vital signs.
  - Temperature: rectal measurement required for accurate core temperature estimate; tympanic membrane, temporal artery, oral, and axillary measurements (shell temperatures) are not accurate for core in athletes and not recommended.
  - Blood pressure (BP), pulse, respirations.
  - Orthostatic changes.
- Record mental status and orientation, walking status, other physical exam findings.
- Record treatment and log times.

Fluid Replacement and Redistribution

- Supine position (nonambulatory): elevate legs and buttocks; restore pooled blood to circulation; if ambulatory, assisted walking (Fig. 80-4).
- Oral fluids (preferred method): all mild cases; all moderate cases, if tolerated.
- Intravenous (IV) fluids: all severe cases; moderate cases if no response to oral fluids or unable to tolerate oral fluids.
- **Recommended fluids**
  - Oral: start with hypertonic fluid like bouillion broth and then move to simple glucose-electrolyte drinks, fruit juices, water.
  - IV: 5% dextrose in NS for first liter if Na is normal or high or if clinically dehydrated, then NS unless blood glucose is low (remember that lactated Ringer's solution contains potassium; avoid until potassium status known; do not use in

Supine position (non-ambulatory). Elevate legs and buttocks; restore pooled blood to circulation.

Figure 80-4 Fluid Replacement and Redistribution after Exercise-Associated Collapse.

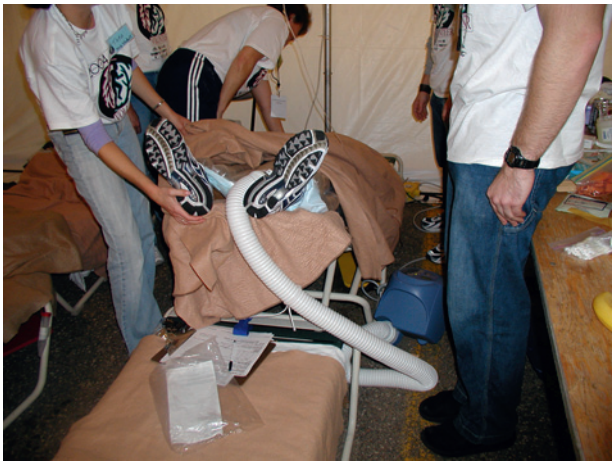
hypothermic patient because cold liver does not metabolize lactate).

- **IV access uses** (invasive procedure that should be used for set criteria)
  - Medication access.
  - Measure serum electrolytes, BUN, glucose, and hematocrit.
  - Fluid replacement in participants with normal or high Na (most finishers are mildly to moderately dehydrated and improve rapidly with IV fluids when by mouth fluids fail).
  - Hypertonic (3%) NaCl solution for symptomatic hyponatremia (in marathons usually due to inappropriate ADH levels in face of excess fluid resulting in fluid overload; in Ironman triathlons may be related to sweat Na loss and dehydration; initial treatment for symptomatic cases is the same).
    - Do not give IV fluids to athlete who appears overhydrated or has increasing confusion, severe progressive headache, or vomiting without checking Na level.
- **Selection criteria for rapid IV fluid replacement** (Twin Cities Marathon)
  - After leg elevation for at least 10 minutes with no improvement (may take 20 to 30 minutes to resolve)
  - Systolic BP less than 100 mm Hg (orthostastic BP drop after leg elevation)
  - Pulse greater than 100 beats per minute
  - Temperature above 104° F or below 95° F.
  - Severe spasms
  - Anorexia, nausea, diarrhea
  - Hypoglycemia (<60)
  - High sodium and/or hematocrit and BUN
  - Confusion with normal or high sodium
  - Not doing well and hyponatremia ruled out
- Other options: no IV starts for fluid replacement and send all who do not recover with simple first aid to ED (may overwhelm EMS transport and ED centers).

Temperature Correction

- **Hyperthermic EAC**
  - Move to cool or shaded area, remove excess clothing.
  - Active cooling (rectal temperature > 105° F): ice water tub immersion for fastest cooling rates; ice packs in neck, axilla, and groin combined with rapidly rotating ice water soaked towels to the arms, legs, trunk and head will give adequate cooling rate (Fig. 80-5). Fans to speed evaporation and increase convective losses may help, but are minimally effective alone in humid conditions.
  - Control continued muscle contractions: shivering, muscle cramping, seizure (considerations include cardiac arrest, heat stroke, and hyponatremia). Following medications may be considered: diazepam: 1 to 5 mg or midazolam 1 to 2 mg slow IV push, repeat as needed (must be prepared to intubate and no driving for 24 hours). Dantrolene should be considered in casualties who are resistant to cooling (ED administration); not generally recommended for field use.
  - Monitor temperature every 10 minutes to assess efficacy of treatment and to monitor for rebound temperature rise or overcooling. End active cooling at 99° to 102° F. (Overcooling to 97° F probably not harmful.)
  - Precool IV fluids.
- **Hypothermic EAC**
  - Move to warm area, remove wet clothing (clothes dryer in cold conditions).
  - Dry skin and insulate with prewarmed blankets (clothes dryer or microwave).
  - Use BairHugger or warm heater air to blow into blankets (see Fig. 80-5).
  - Have patient breathe warmed, humidified air (Bennett or Bird respirator).
  - Place warm packs (hot water bottles, warmed IV bags) in neck, axilla, and groin.

Hyperthermic EAC. Rapidly rotate ice water soaked towels to the arms, legs, trunk and head to give adequate cooling rate.



Hypothermic EAC. Use BairHugger™ or warm heater air to blow into blankets.

Figure 80-5 Temperature Correction.

  - Prewarm IV fluids, and consider IV dextrose 50% in water.
  - Monitor temperature at regular intervals.
  - Walk to generate intrinsic heat (if body temperature > 95° F).
- **Normothermic EAC:** maintain temperature; monitor temperature if not improving (delayed hyperthermia, postrace hypothermia).
- **Leg cramps:** salty fluid and glucose replacement, neuromuscular inhibition techniques, assisted walking, dextrose 50% in water. Avoid massage until patient is well hydrated. Consider diazepam 1 to 5 mg or midazolam 1 to 2 mg IV push (be prepared to intubate and no driving for 24 hours).

Fuel Supply

- Oral glucose solutions.
- IV glucose solutions: dextrose 5% in stock IV solutions (no dextrose 5% in water).
- Indications for dextrose 50%: low blood glucose (measure with home glucose meter at toe, ear lobe), slow response to IV hydration, slow response to temperature correction, muscle cramping, severe EAC, cardiac arrest.

Transfer or Discharge

- Transfer to emergency facility if patient does not respond to usual treatment or if severe cases do not respond rapidly. Remember automatic transfer rules.
- Discharge from race medical facility clinically stable and normothermic patients with good cognitive function.

- Instruct in fluid and food replacement.
- Reevaluate if change in status.
- Recommend follow-up exam for severe cases.

LOW-FREQUENCY MEDICAL EMERGENCIES

Cardiac Arrest

- Equipment and supplies: automatic or manual defibrillators, intubation equipment and induction medications, ACLS drug kits (epinephrine, atropine, amiodarone, lidocaine, verapamil, sodium bicarbonate, morphine, magnesium sulfate, dextrose 50% in water [check for updated ACLS recommendations]), oxygen and delivery system, IV kits.
- Assume that seizure-like activity is due to cardiac arrest and apply automated external defibrillator (AED); if pulse and breathing present, check temperature; if normal, assume hyponatremia and check serum sodium.
- ACLS standard protocol: ABCs (airway, breathing, circulation), defibrillation and cardiopulmonary resuscitation, cardiac monitor, IV access, intubation, medications.
  - Twin Cities Marathon modifications
    - Substrate replacement: Glucose has been depleted by physical activity and should be replaced for heart to respond to treatment (not a proven strategy). Use dextrose 50% in water IV push.
    - High-dose epinephrine seems reasonable alternative to low-dose regimen in face of arrest after activity that depletes catecholamines (not a proven strategy). Use 5 to 10 mg IV push.
    - Acidosis: Metabolic acidosis of activity must be reversed. Use sodium bicarbonate.
  - Amiodarone stabilized cardiac rhythm in recent Twin Cities Marathon arrest.
  - In prolonged resuscitation of marathon or longer runners, consider placement of pressure transducers in lower leg compartments to detect elevated compartment pressures.
- Sudden cardiac arrest (SCA) and sudden cardiac death (SCD) in marathon road racing:
  - Twin Cities Marathon and Marine Corps Marathon combined database: 2 SCA per 100,000 finishers and 1.1 SCD per 100,000 finishers.
  - London Marathon: 1.7 SCA per 100,000 finishers and 0.8 SCD per 100,000 finishers.
  - USA marathons 2006: 1.6 SCD per 100,000 finishers.
- Success of resuscitation for race-related SCA: Twin Cities Marathon, yes for 2 of 4; London Marathon, 6 of 11; New York Marathon, 1 of 4; Marine Corps Marathon, 3 of 8. (In major metro areas of the United States in nonrunners with out-of-hospital SCA: success with 1 or 2 of each 20.)
- Road racing by trained individuals is safer than sedentary lifestyle.
- Preventable
  - Can cardiac screening reduce deaths? Cost is problem.
  - Close attention to symptoms and risk factors with appropriate screening.

Exercise-Associated Hyponatremia

- Etiology: Water excess and dilution due to overhydration combined with inhibition of renal water clearance (inappropriate ADH) is most commonly cause; in very long events like the Ironman distance triathlon, athletes with high sweat sodium concentrations may develop sodium depletion hyponatremia with or without dehydration.
- Incidence: Low in events lasting longer than 4 hours; more common in ultra-marathon distances and long-distance triathlons; five deaths in marathons since 1998 and reports are increasing in slower participants (3 per 150,000 symptomatic finishers at Twin Cities Marathon, but 13% of Boston Marathon cohort [asymptomatic]).
- Significance: Potential for fatal outcome characterized by rapid deterioration, progressing to seizure, respiratory distress, and coma because of worsening pulmonary and cerebral edema; reason for transfer to ED. May present at home, in hotel, or in transit (subway, train, airplane, automobile) several hours postrace.
- History: High fluid (sports drink or water) intake of 1 to 2 glasses at every aid station or lack of weight loss during event in dilutional hyponatremia; severe pounding headache that progressively worsens over time; feeling of impending problem or feeling scared; puffiness in extremities; muscle cramping; sleepiness; nausea and vomiting; confusion; slow times; small body habitus; inconsistent reports of nonsteroidal anti-inflammatory drug (NSAID) use.
- Physical findings: Ashen skin (including lips), tight rings, watches, or shoes; vomiting; normal pulse, BP, and respiration (early); intact mental status (early); confusion, check for muscle spasms, tetany, clonus, seizure.
- Lab tests (if available): Sodium (<135 mmol/L); low hematocrit and BUN from fluid overload. Average sodium in fatal cases is 121 to 122 mmol/L. It is desirable to have point-of-care sodium, BUN, and hematocrit measurement available in the medical area.
- Treatment on site: If minimal symptoms may treat on site with hypertonic fluid (4 bouillion cubes dissolved in 4 ounces water) and observe until urinating freely. If symptomatic and deteriorating mental or respiratory status, start high flow oxygen supplementation and give 100 mL 3% NaCl over 10 minutes (can repeat 2 additional boluses) and continue hypertonic saline at 50 to 70 mL per hour during transfer to ED. The initial treatment for either water dilution or sodium depletion hyponatremia is hypertonic saline. Patients with sodium level of less than 135, clinical signs of dehydration, and normal to high BUN/hematocrit may be treated on site with IV NS. Have diazepam and/or magnesium sulfate ready if clonus or seizure develops.
- Disposition
  - Transfer runner with this clinical constellation to emergency facility if no point-of-care lab tests available. Start IV line for medication access but do not assume dehydration and do not administer hypotonic fluids. Alert ED of transfer and expected treatment and complications.
  - For athletes with improving clinical status and lab values who begin to urinate freely, onsite observation with discharge in the company of responsible family or friends is permissible.

Other

Anaphylaxis: types (atopic, exercise-induced), treatment (epinephrine, antihistamine).

Asthma: inhalers (albuterol metered-dose inhaler with extender), oxygen high flow, nebulizer (albuterol), subcutaneous terbutaline or epinephrine.

Insulin shock: dextrose 50% in water, glucagon.

TRAUMA

- **High-velocity activity collisions and falls:** biking, skiing, wheelchair racing.
- **Vehicle on course (collision)**
- **ATLS protocol**
  - Equipment: back boards, neck collars (semirigid), splints, cricothyroidotomy kit, oxygen, IV fluids.
  - Primary survey: airway and cervical spine control (leave helmet on if airway okay; take helmet off if unable to establish airway and breathing); breathing and ventilation; circulation and bleeding control; disability or neurologic status; exposure and examination.
  - Initial resuscitation: high-flow oxygen, shock management (fluid therapy, position, shock trousers), cardiac monitor
  - Secondary survey: look for other problems.

- Definitive field care: temperature maintenance, pain control, splint, other.
- Transport to emergency medical facility or race medical facility, if equipped and staffed to care for trauma.

POSTRACE REVIEW

- **What went right** (and can it be improved)?
- **What went wrong** (and why)?
- **Proposed changes** to improve safety and care of athletes.

BUDGET FOR MEDICAL OPERATION COSTS

- Race or event should develop budget that includes medical equipment and supplies.
- Equipment and supplies can be purchased, donated, rented, or borrowed; cost of high-tech equipment makes it difficult to purchase or borrow.
- Volunteer time is usually donated, but race should provide T-shirt, poster, pin, or similar reward for personnel.
- Ambulance service should be enlisted to help with transport of athletes off course and from finish area to local hospitals.

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Field Hockey

Cherie B. Miner

RULES OF THE GAME

- Two halves, 35 minutes each in collegiate and international play, 30 minutes in high school play.
- Eleven players per side, including goalkeeper.
- Pitch may be grass or artificial turf and is 100 yards long and 60 yards wide, divided by centerline and 25-yard line on each side of the field. Striking circle is marked 16 yards from each goalpost. All international matches are played on watered-down artificial turf.
- Goal cages are 7 feet high, 12 feet wide and 4 feet deep. Boards on back and side of cages are 18 inches high.
- Two umpires on field officiate a match.
- Ball must be passed or dribbled down field with flat side of stick.
- Goal is scored when attacker strikes ball into goal from within striking circle.
- Players may not shield ball with body or stick. All players must have equal chance to play ball.
- A 1998 rule changed placement of corner shots to the sideline. No offside rule.
- Ball does not have to be stopped on a corner shot.

EQUIPMENT

- Stick has curved head, is rounded on one side and flat on left-hand side. Ball can be touched only with flat side of stick. Stick is made of composite materials. Stick length is 36 to 38 inches.
- Ball is solid plastic, slightly larger than a baseball, and weighs between 5½ and 5¾ ounces with circumference of $8^{13}/_{16}$ to 9¼ inches.
- Goalie: helmet, mouth guard, chest protector, throat protector, leg pads, kickers, and footwear.
- Field players: footwear, shin guards now made of hard plastic over molded foam, mouth guards.
- Goggles are not mandated for high school players, but are mandated for college players. Goggles need a soft covered frame and plastic lenses.
- Soft helmets may be worn and the issue has been raised of mandating helmets for field players. Face masks may be worn but need to be smooth and fit flush to the face.

FOULS

- All players must have equal chance to gain control of ball as it is dribbled or passed down field.
- Player may not:
  - Shield or obstruct ball from opponent with body or stick.
  - Play ball with rounded side of stick.
  - Interfere in game without stick.
  - Charge, bit, shove, or trip opponent.
  - Play ball in potentially dangerous way.
  - Use foot or leg to support stick to resist opponent.
  - Rise stick in dangerous manner while approaching, attempting to play or stop ball.
  - Advance ball by any means other than with stick.
  - Stop or deflect ball in air or on ground with any part of body.
  - Hit, hook, hold, or interfere with opponent's stick.

INJURY RATES

See Tables 81-1 and 81-2.

Table 81-1 MOST COMMON WOMEN'S FIELD HOCKEY GAME INJURIES BEFORE (1988-1989 THROUGH 1995-1996) AND AFTER (1996-1997 THROUGH 2002-2003) SELECT RULE CHANGES*

Body part	Injury type	Number	Percentage of all injuries	Rate per 1000 athlete exposures	95% Confidence interval
Game injuries, 1988-1989 through 1995-1996					
Ankle	Ligament sprain	78	10.54	0.91	0.71, 1.11
Knee	Internal derangement	52	7.03	0.61	0.44, 0.77
Finger	Fracture	38	5.14	0.44	0.30, 0.59
Head	Concussion	38	5.14	0.44	0.30, 0.59
Upper leg	Muscle-tendon strain	32	4.32	0.37	0.24, 0.50
Nose	Fracture	23	3.11	0.27	0.16, 0.38
Face	Laceration	23	3.11	0.27	0.16, 0.38
Upper leg	Contusion	19	2.57	0.22	0.12, 0.32
Patella	Patella or patella tendon injury	19	2.57	0.22	0.12, 0.32
Lower leg	Contusion	17	2.30	0.20	0.10, 0.29
Total injuries		740	100.00	8.65	8.03, 9.28
Total exposures		85,508			
Game injuries, 1996-1997 through 2002-2003					
Head	Concussion	43	8.96	0.62	0.43, 0.80
Ankle	Ligament sprain	40	8.33	0.58	0.40, 0.75
Knee	Internal derangement	36	7.50	0.52	0.35, 0.69
Upper leg	Muscle-tendon strain	28	5.83	0.40	0.25, 0.55
Nose	Fracture	22	4.58	0.32	0.18, 0.45
Finger	Fracture	18	3.75	0.26	0.14, 0.38
Head	Laceration	15	3.13	0.22	0.11, 0.33
Face	Laceration	13	2.71	0.19	0.09, 0.29
Patella	Patella or patella tendon injury	12	2.50	0.17	0.07, 0.27
Pelvis/hip	Muscle-tendon strain	11	2.29	0.16	0.06, 0.25
Total injuries		480	100.00	6.91	6.29, 7.53
Total exposures		69,478			

*In 1996, provisional new rules (placement of corner shots and no offsides) were implemented in collegiate women's field hockey. The rules became permanent in 1998.

Table 81-2 1988-2003 NCAA INJURY SURVEILLANCE SYSTEM*

	Practice injuries	Game injuries
Injuries	Lower extremity 60% ▪ Upper leg strains 26.9% ▪ Ankle sprains 15% ▪ Pelvis/hip muscle strains 9.9% ▪ Knee internal derangement 7.8% Trunk/back injuries 16.2% Head/neck 8.4% with concussions at 3.4% Upper extremity at 8.1% and finger fractures 1.9%	Lower extremity 40% ▪ Ankle sprains 13.7% ▪ Knee internal derangement 10.2% ▪ Upper leg muscle strains 7% Head/neck 25.3% with concussions 9.4% Upper extremity 20.7% with finger fractures 6.5%
Injuries resulting in 10 plus days of sport time lost	Knee internal derangement 15.3% Lower leg stress fractures 9.7% Ankle ligament sprains 8.2%. Upper leg muscle tendon strains 5.2% Other injuries 61.6%	Knee internal derangement 23.1% Finger fractures 9.1% Ankle ligament sprains 9.1% Thumb fractures 5.4% Head concussion 5.4% Other injuries 47.8%
Mechanism of injury	Player vs. player contact 5% Player vs. stick, ball, or ground contact 26% Noncontact injuries 64%	Player vs. player contact 13% Player vs. stick, ball, or ground contact 60% Noncontact injuries 26% Most injuries occurred inside the 25 yard line (41.%) or around the goal (26%)
Injuries by player positions	Forwards 22.4%, mid-fielders 27.6%, back-fielders 23.6%, goalies 19.5%, and unknown 6.9%. Above the neck injuries are common in all positions except goalies. ▪ 69.3% occurred within the 25 yard line. ▪ 77.4% were from contact with stick or ball. ▪ Back-fielders had the most hand/finger/thumb injuries at 39%, with 94.9% from contact with stick or ball. ▪ Hand fractures are the most common. ▪ Has been a decline in hand injuries with the rule change not requiring the ball be stopped on a corner hit.	

*In this 15-year study, 1220 injuries reported from 10,358 games and 2066 injuries from 26,740 practices.
Practice (3.70) vs. games (7.87) per 1000 athlete exposures.
Per season practice had almost three times the injury rate as regular season practice.
Overall 2.5% decrease in game injuries over 15 years.

COMMON INJURIES

Ligament sprains: Mostly ankle and knee sprains (Fig. 81-1). Treat with rest, ice, compression, elevation, (RICE); rehabilitation with range of motion, strengthening, proprioception exercises. Return to play with bracing or taping.

Fractures: Most commonly occur in fingers and nose. Players are at increased risk with corner hits and play in the circle. Routine diagnosis and treatment. Consider padded gloves for prevention.

Stress fractures: Occur from overtraining. Consider nutritional status, amenorrhea, and female athlete triad (see Chapter 10, The Female Athlete). Evaluate biomechanics, foot/ankle alignment, and footwear. Players may benefit from orthotics. Stress fractures are diagnosed with x-ray or bone scan (see Fig. 81-1). Treat with immobilization and rest.

Contusions and muscle strains: Occur from stick or ball contact and player collisions. Muscle strains may be from overuse or lack of conditioning. Routine care is ice, maintenance of range of motion, observation of myositis ossificans.

Abrasions/lacerations: Occur frequently on turf and secondary to stick or ball trauma. Routine care is suture; blood and abrasion should be covered. Blood must be removed from field.

Low back pain: Body positioning during field hockey play puts player at risk. Training regimen should include low back strengthening and trunk extension stretching. Diagnosis and routine care for muscle strains, disc herniation, spondylolisthesis, osteoporosis, osteopenia (as cause of vertebral fractures).

Concussions: Routine diagnosis and care.

Dental injuries: Should be reduced by mouth protectors. In international male and female players incidence of dental/facial trauma is high.

- 54% sustain dental injury requiring medical care; 20% had serious dental damage (16% in women and 22% in men).
- 20% of international players reported to wear mouth protection consistently (women twice as much as men).
- In a recent National Collegiate Athletic Association study over 15 years there were seven teeth injuries.

Eye injuries: Are infrequent but severe when they occur.

Catastrophic female injuries: In field hockey from 1982 to 2006 at the high school and college levels, there were three serious nonfatal injuries:

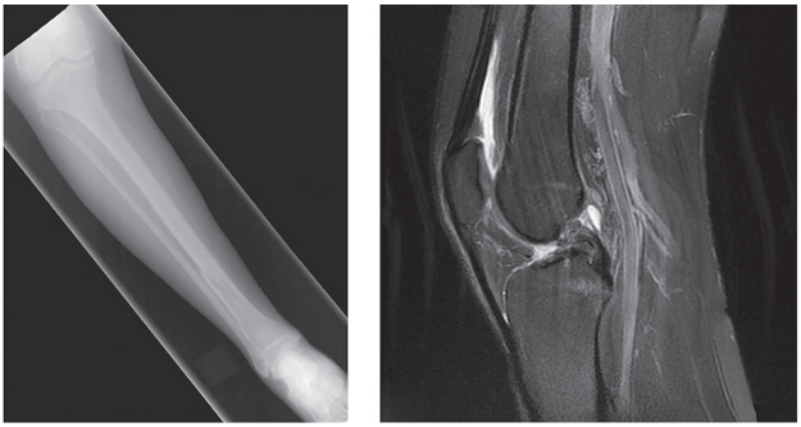
- Skull fracture from being hit with ball.
- Eye injury with loss of an eye after being hit with a stick.
- Avulsion of the distal half of the fifth phalanx after getting finger caught between ball and stick.
- No fatal injuries.

General Injury Prevention

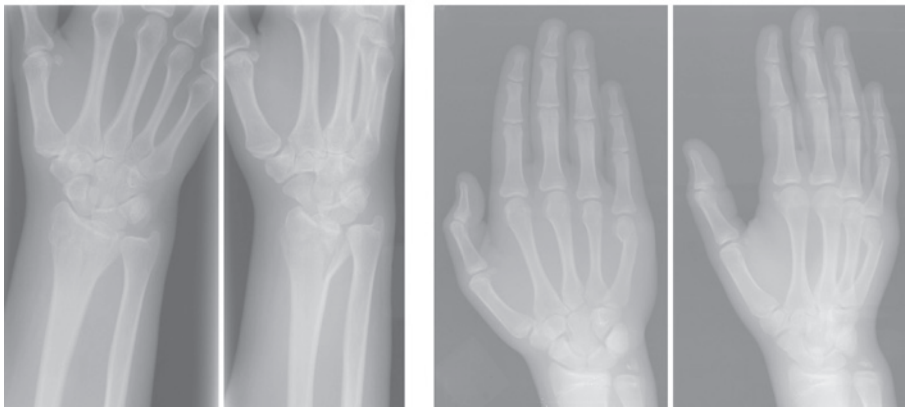
- Prophylactic bracing of and/or taping of ankles.
- Balance training exercise programs to work on proprioception. The ability to have a sense of joint position and the body's ability to react to where the joint is in space will prevent injury.
- Neuromuscular conditioning for both knee and ankle to prevent knee injuries (anterior cruciate ligament [ACL] tears) and ankle sprains.
- Data driven rule changes (to decrease congestion near goal) and equipment changes (helmets vs. goggles and padded gloves).

ENVIRONMENTAL ISSUES

Heat-related issues: Heat stroke, heat exhaustion, heat cramps, heat syncope, heat edema. Maintain adequate fluid intake and acclimatization to extreme heat. Athletes are at higher risk during preseason and tournament play. Younger athletes are at higher risk than adults because of reduced sweating capacity and greater body surface area to weight ratio.



Tibia fracture in distal third of lower leg. Knee with ACL tear. PCL is intact.



Radial fracture in proximal wrist. Metacarpal fracture of 5th metacarpal.

Figure 81-1 Common Injuries in Field Hockey.

Cold weather issues: Frost nip, chill blains, frostbite, hypothermia. Wear appropriate clothing, gloves for prevention.

Fluid considerations: Prevent and manage dehydration. Frequent water breaks during play. Postworkout body weight should equal preworkout body weight. Athletes need to rehydrate prior to continued play.

Grass versus synthetic playing surfaces: Affect lower extremity injury rates:

- An Australian study in 1989 found that the injury rates were higher on AstroTurf and most commonly involved lower extremities. Soft tissue injury appeared to be greater on AstroTurf, but joint injuries to the lower extremity were more prevalent on grass.
- Multivariant risk analysis of playing in the National Football League showed higher rates of ACL injuries on turf. Limited studies in field hockey.
- Coaches and players need to be aware of effects of different playing surfaces: wet and dry grass versus wet and dry turf. Type of shoe and friction between shoe and playing surface also have an effect.

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Lacrosse

82

Paul S. Sherbondy

GENERAL PRINCIPLES

History

- The sport of lacrosse derives its name from the netted stick, or crosse, which resembles a crosier, a staff with a hook-shaped curve at one end, used by religious figures.
- Lacrosse is a true American sport that has its roots in Native American Indian culture.

Basics of the Game

- Lacrosse is played on a rectangular field with nets at opposing ends.
- The game begins with a face-off at midfield.
- Once possession of the ball is gained, the ball is passed, caught, and carried in the netted stick.
- The object is to throw the ball into the opposing team's goal, scoring one point.
- Defensive players may attempt to intercept passes and dislodge the ball from the stick.

PARTICIPATION

- U.S. Lacrosse is the governing body of American lacrosse.
- Lacrosse is played at youth, high school, college, post-college club, professional, and international levels.
- The traditional hotbed of lacrosse is the Northeast, but participation is exploding across the United States.
  - Lacrosse is one of the top three fastest growing high school and National Collegiate Athletic Association sports.
  - There were 425,000 estimated participants in 2006, an increase of 68% since 2001.
  - Forty-seven thousand fans attended the 2006 National Collegiate Athletic Association (NCAA) Division I Finals.

MEN'S FIELD LACROSSE

- There are 10 players per side: three defensemen, three midfielders, three attackmen, and one goalie.
  - Defensemen use longer sticks for enhanced blocking and checking (Fig. 82-1).
  - Midfielders transition play from offense to defense and cover the entire field. Midfielders may use either short or long sticks.
  - Attackmen are the primarily goal scorers. They use shorter sticks for better ball control (see Fig. 82-1).
  - The goalie uses a special stick with a larger netted end and functions to save shots on goal (see Fig. 82-1).
- Four players must remain in the defensive zone and three in the offensive zone at all times.
- Possession changes if the ball is run or thrown out of bounds. The team whose player is closest to a ball shot on goal when it goes over the boundary line is awarded possession.
- Men's lacrosse is a contact sport with hitting allowed by the body and stick (checking).

Defenseman or long-stick midfielder, men's lacrosse.

Attackman, men's lacrosse.

Goalie, men's lacrosse.

Figure 82-1 Men's Field Lacrosse. *(Photographs printed with permission of Dr. Paul S. Sherbondy.)*

WOMEN'S FIELD LACROSSE

- Women's lacrosse is a noncontact sport, although controlled checking with the stick is allowed.
- There are 12 players per side: one goalie, four attackers, four defenders, and three midfielders with similar functions as in the men's game.
- Traditionally there were no fixed field boundaries in women's lacrosse but hard boundaries of variable size were instituted in 2006.
- The restraining line rule permits only seven offensive and eight defensive players within 30 yards of the goal at any time.
- Sticks are of uniform size except for the goalie.

PROTECTIVE EQUIPMENT

- Men's lacrosse equipment is designed to protect against incidental stick ball and body contact.
  - Field position players are required to wear a mouth guard, National Operating Committee on Standards for Athletic Equipment (NOCSAE)–approved helmet with full face mask and four-point chin strap, gloves, and arm, elbow and shoulder pads.
  - The goalie is also required to wear throat and chest protection and an athletic cup.
- Women's lacrosse goalies are required to wear head, throat, and chest protection (Fig. 82-2).
  - Field position players must wear a mouth guard and eye protection (see Fig. 82-2).
  - Eye protection was a controversial topic and was not mandated until 2005.

FOULS

- Men's lacrosse has both technical and personal fouls, which result in suspension from play for 30 seconds to 2 minutes and/or loss of possession.
- Women's lacrosse has minor and major fouls, which result in free position and/or loss of possession.

INJURIES

Injury Epidemiology

- The NCAA Injury Surveillance System collects data on collegiate lacrosse injuries.
- Data from the 2004 report shows that men's lacrosse has practice and game injury rates of 3.2 and 12.6, respectively, per 1000 AE (injuries per 1000 athletic exposures).
  - Twenty-one percent of both game and practice injuries resulted in time-loss from participation for greater than 10 days.
  - The top four injury types for games and practice are ligament sprains of the ankle, internal derangement of the knee, concussion, and upper leg muscle-tendon strain.
- Women's lacrosse has injury rates of 3.3 for practice and 7.2 for games.
  - Time-loss greater than 10 days occurred in 22% of game and 24% of practice injuries.
  - The top four practice and game injury types are ankle ligament sprain, internal derangement of the knee, muscle-tendon strain of the upper leg, and concussion.

Specific Injuries

Head

- Cerebral concussions are fairly common injuries.
  - Concussion injury rates are 0.25 and 0.26 per 1000 AE for women's and men's lacrosse, respectively.
  - This places the women at eighth and the men at tenth out of 16 NCAA sports for concussion frequency.
- Facial lacerations from stick contact or helmet impingement also occur.
- Eye injuries are of concern in women's lacrosse but the recently instituted mandatory eye protection should decrease eye injury rates.
- Above-the-neck injuries account for one-fifth of all game related injuries in women's lacrosse.
- Most of the injuries are concussions, contusions, and lacerations and are caused predominantly by stick or ball contact.

Shoulder

- Shoulder separations and traumatic glenohumeral instability are encountered mainly in men's lacrosse (Fig. 82-3).
- Shoulder overuse injuries are not frequent because of the mechanical advantage afforded to the thrower by the lacrosse stick.

Women's lacrosse goalie.

Women's lacrosse field position player.

Figure 82-2 Protective Equipment. *(Photographs printed with permission of Dr. Paul S. Sherbondy.)*

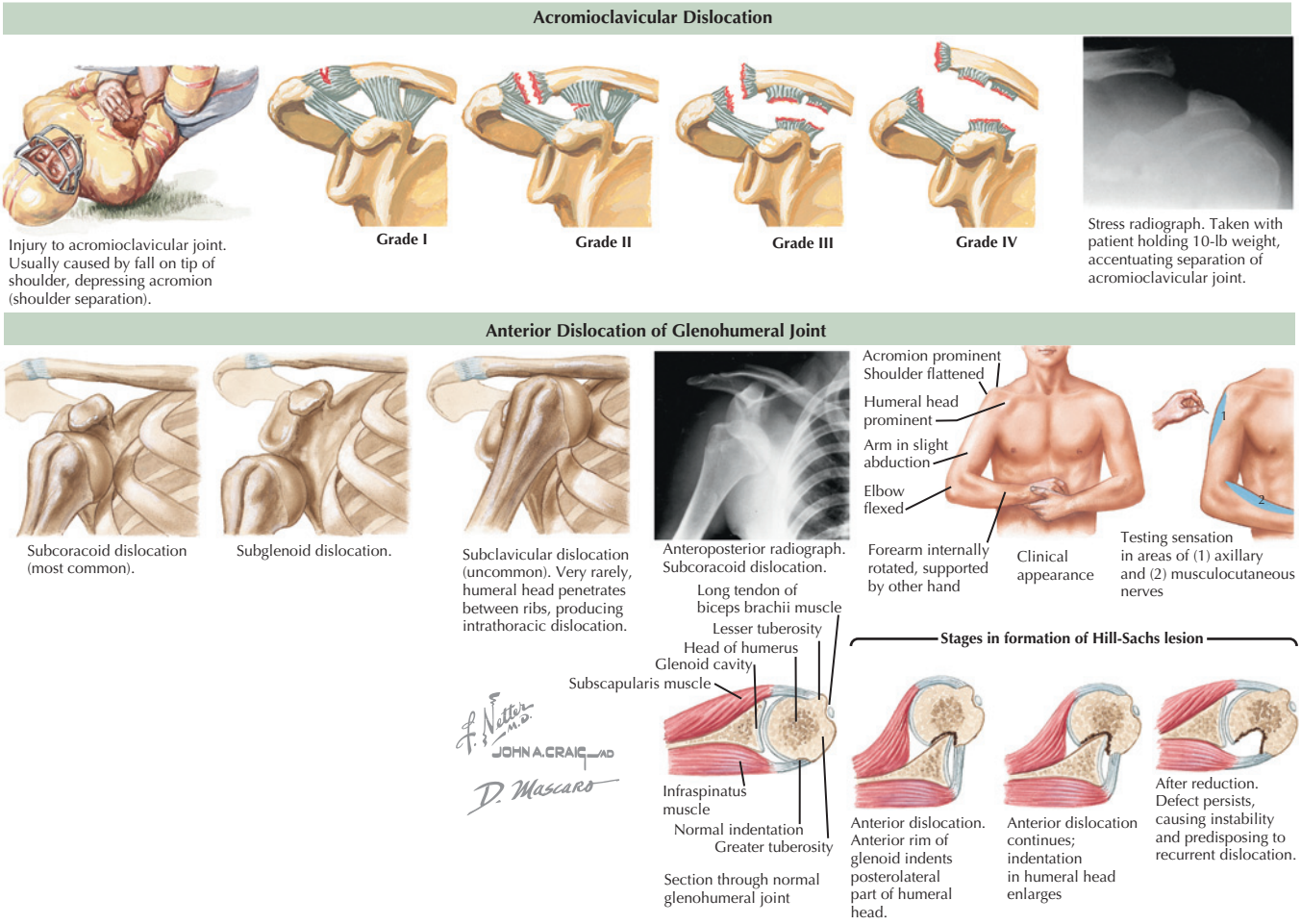

Figure 82-3 Shoulder Injuries.

Chest

- Commotio cordis is a rare cause of sudden cardiac death.
- It is seen in sports where a projectile is used and is most common in baseball.
- Commotio cordis occurs when the ball impacts over the cardiac silhouette in a specific phase of the cardiac cycle.
- This mechanical impaction causes an electrical disruption of the heart and results in an abnormal rhythm, most commonly ventricular fibrillation.
- Affected individuals collapse immediately or within a few seconds of impact.
- Commotio cordis has a high mortality rate even with CPR and defibrillation.
- It is inconclusive if chest protectors offer any protection and are not universally mandated for at-risk sports.
- It is believed that the most effective treatment is urgent defibrillation with an automated external defibrillator (AED) within 3 to 5 minutes of onset.
- It is prudent to have an emergency action plan in place for such situations and to have immediate access to an AED for at-risk sports.

Hand

- Despite protective gloves, hand and wrist fractures are seen, mainly from being slashed by the stick (Fig. 82-4).
- Lacrosse goalkeeper's thumb is a fracture of the distal or proximal phalanx of the thumb.
  - The typical mechanism is impact of the ball on the tip of the thumb as the goalie attempts to save a shot on goal.
  - This injury is treated according to standard fracture methods (see Fig. 82-4).
  - This injury can be prevented or protected by a custom-molded plastic shell within or outside the goalie's gloves (see Fig. 82-4).

Lower Extremity

- As with most explosive running and cutting type sports, strained hamstrings, noncontact anterior cruciate ligament (ACL) tears, and ankle sprains are the predominant lower limb injuries (Fig. 82-5).
- ACL injury rates for both men's and women's lacrosse place them in the middle third of the 16 surveyed NCAA sports.
  - Men's lacrosse has an ACL injury rate of 0.12 (rank 10th) and the women's game 0.17 per 1000 AE (rank 11th).
- For ankle sprains, similar injury rates are noted.
  - Women's lacrosse ranks 9th and men's 8th, with injury rates of 0.70 and 0.66 per 1000 AE, respectively.

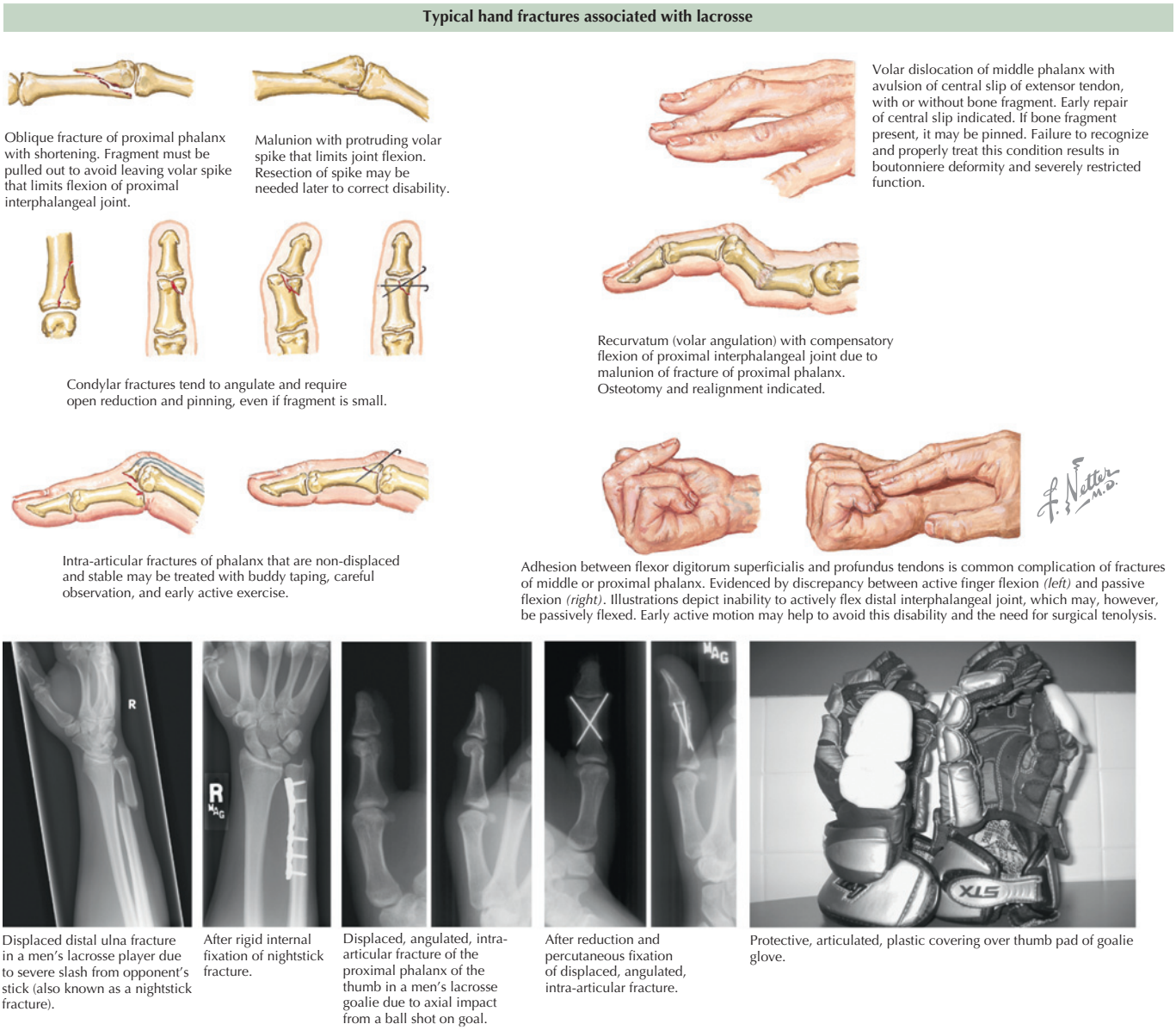

Figure 82-4 Hand Injuries.

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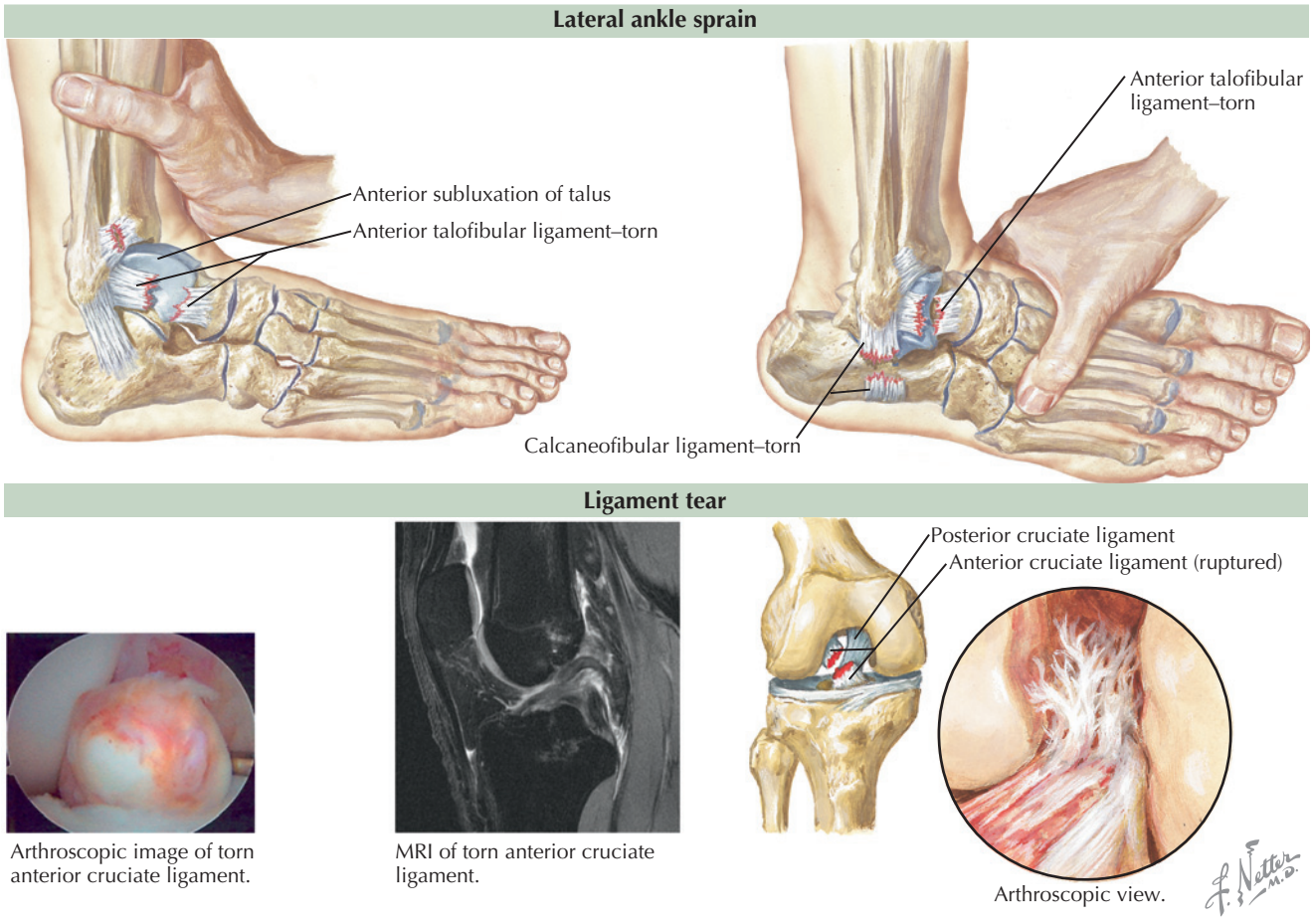

Figure 82-5 Lower Extremity Injuries.

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Rowing

Jane S. Rumball and Constance M. Lebrun

GENERAL OVERVIEW

- As a competitive sport, rowing dates back several hundred years, and was one of the original sports in the modern Olympic Games.
- Rowing was the first intercollegiate sport in the United States, the first U.S. race being held in 1852 between Harvard and Yale.
- Since Title IX regulations that require equal proportions of male and female athletes in collegiate sports, there has been a vast increase in the number of women's collegiate rowing programs; total numbers of female collegiate rowers have increased from roughly 1000 in 1981-1982 to approximately 7000 today.

PRINCIPLES OF TRAINING AND RACING

Physiology

Training

On-water: Usually high volume (1 to 3 times daily, 1 to 2 hours in length), with higher intensity pieces and interval work during racing season.

Indoor: On rowing ergometer. Simulates water training and is good monitor of fitness levels.

Cross-training: Weights, running, cycling, cross-country skiing. Used to supplement water training or during winter season. Many injuries occur as a result of improper transition to cross-training from on-water practices (and vice versa).

Racing

- Anaerobic contribution 10% to 30%; aerobic system supplies remainder.
- Ranks among most strenuous of sports because of high amounts of lactic acid incurred and cardiovascular strain. $\dot{V}O_2$ max values can reach 70 mL/kg per minute for elite rowers.

Race Distances

2000 meters: Standard distance of spring and summer racing (collegiate, club racing, and international). Boats line up side by side at starting gates in up to six lanes. Races typically last 5½ to 8 minutes or longer, depending on event class, wind conditions, and rowing ability. For example, the World Best Time in the World Championship men's eight event (raced in fast tailwind conditions) is now under 5 minutes 20 seconds.

Head racing: Predominant type of racing in fall season. Distance is usually 3-plus miles against the clock from a running start, and involves more steering on rivers that bend and turn.

Athlete Classification

- Rowers are classified by sex, age, and weight.
- Age categories are Junior (18 and under), Senior B or U23 (under 23), Senior (open), and Masters (27 and older).
- Weight categories are lightweight and heavyweight/open.
  - Both types of athletes have similar build, although lightweight rowers typically have slightly lower body fat percentages.
  - Being tall and lean allows for maximum stroke length while minimizing drag on the boat.
- Weight restrictions: Lightweight rowers must weigh in 2 hours prior to racing at or below maximal weight for national and international competition. Weight restrictions are as follows:
  - **Men:** 70-kg boat average, 72.5-kg individual maximum for national and international competition.
  - **Women:** 57-kg boat average, 59-kg individual maximum for international competition, 130 pounds with no boat average for national competition.
- Coxswains: Role is to steer the boat using rope attached to a rudder, make technical and motivational calls, and make decisions about strategy. Minimum weight of 120 pounds for men and 100 pounds for women. If underweight, must carry weight for racing, shown to the referee at the starting gate.

EQUIPMENT AND SAFETY ISSUES

Boat Types

Sweep: Rower uses one oar, which is placed either on starboard or port (left and right, respectively, from the rower's perspective). Available boats are the pair, four, and eight. Sweep boats are the only boats with the option to have a coxswain, and the eight is the only boat to always use a coxswain (Fig. 83-1).

Sculling: Rower uses two oars. Available boats are the single, double, and quadruple sculls (see Fig. 83-1).

Rowing shell: Classically made of wood, now constructed of synthetic material such as carbon fiber. Seats are attached to wheels that roll in fixed tracks or slides. Athlete faces backward and pulls away from fixed shoes. Oar is held in an oarlock attached to a rigger that extends out from the shell. Equipment is modifiable. Individual adjustments, known as rigging, are relatively easy to do. Shoes can be moved in boat either forward or backward, up or down, or with toes pointing either toward each other or away. Height of oarlock off water and relative load can be modified, and riggers can be moved forward and backward on the boat itself. Pitch refers to the angle of the pin holding the oarlock, which affects the path of the blade through the water (see Fig. 83-1).

Oars: Classically made of wood but now carbon fiber. Consists of a handle with varying grip sizes and material, a collar and sleeve that fits into the oarlock, and the blade that enters the water. Blade shape has changed over the years and is generally moving toward being able to generate more force per stroke. This may account for the rise in injury because greater load is placed on the rower during each stroke.

Indoor rowing ergometer (colloquially "erg"): Used for fitness testing, training, and racing during indoor season. Most common versions made by Concept 2 (Morrisville, Vt.); consists of a sliding seat on a single rail, fixed footplate, and handle attached to chain. Chain spins a flywheel that creates resistance, and small computer monitors power output, split times, and rate.

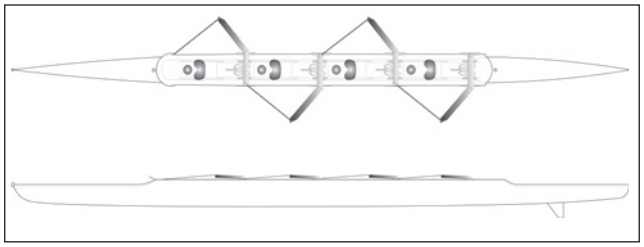
PHASES OF ROWING STROKE

Catch: Legs and back fully flexed and arms fully extended. Rower's seat is at the front of the slides and oar enters the water in the squared position (blade perpendicular to the water) (Fig. 83-2).

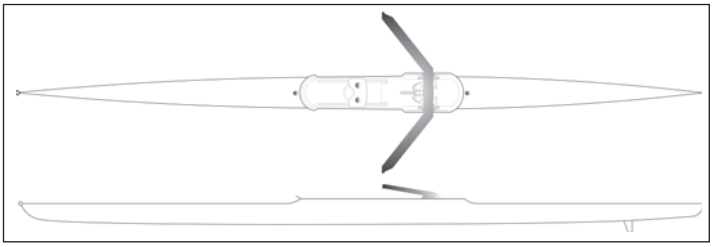
Drive: Legs extend and back begins to extend slightly, while arms and shoulders remain relatively fixed. Once back has extended to neutral position, arms begin to flex and continue acceleration of blade through the water (see Fig. 83-2).

Finish or release: Legs flat and fully extended, shoulders behind hips but back still slightly flexed, arms flexed. Blade is taken out of the water by simultaneously putting weight on the handles (tapping down) and feathering (turning wrist so blade is parallel to water). Feathering allows the blade to be carried higher off the water to pass over waves and is also more aerodynamic (see Fig. 83-2).

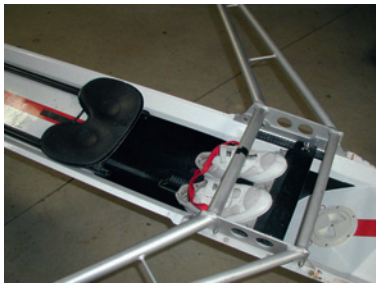
Recovery: Reverse of drive sequence; arms extend to move oar handle forward, back becomes more flexed, and knees are flexed to bring rower into position for the next catch.



Example of sweep boat: the straight or coxless four.



Example of sculling boat: the single.



Inside of rowing shell.

Figure 83-1 Types of Rowing Boats.

The catch.

The drive.

The release.

Figure 83-2 Phases of Rowing Stroke. *(Photographs © Dr. Volker Nolte.)*

APPROACH TO INJURY EVALUATION

- Vast majority for competitive rowers of all ages and abilities are chronic overuse injuries, due to repetitive nature of the sport.
- Improper stroke mechanics and asymmetries can predispose a rower to injury.
- Other factors include poor weather conditions affecting stroke mechanics, fit of equipment and rigging, and improper transition from indoor to on-water training.
- If possible, observe rowing technique on water or on the ergometer. Watch for compensatory behavior related to poor flexibility, muscle or strength deficiencies, abnormal asymmetrical movements, or undue force placed on the injury site in question.
- Most common injury sites for male rowers: lumbar spine, forearm/wrist, and knee.
- Most common injury sites for female rowers: chest, lumbar spine, and forearm/wrist.
- Rib stress fractures are also typical and unique to the rowing population.

INJURIES

Low Back

Description: Most frequently injured region, accounts for up to 25% of all reported rowing injuries.

Mechanism of injury: Great loads are placed on the lower back during the rowing stroke. Lower back muscles are relatively relaxed as the rower approaches the catch position. At the catch, the spinal extensors are quickly loaded with the resulting compressive forces at the spine, building and reaching peak compressive forces at approximately mid-drive (estimated to be more than four times a rower's body mass). Sweep rowing introduces increased rotation through the spine because the rower reaches at the catch to maximize stroke length, contributing to an increased risk of injury to the spine. Fatigue of the spinal extensor muscles has been proposed to result in decreased ability of the lumbar spine to resist further flexion forces acting on the spine during the drive and possibly increasing the load on passive spinal structures (e.g., ligaments and discs).

Types of injury:

- **Muscle strain:** Most common injury. Pain in low back, involving erector spinae muscles and/or sacroiliac joint region.
- **Sacroiliac joint dysfunction:** May result in pain over buttock, lateral thigh, anterior pelvis, and groin. Contributing factors may include leg length discrepancies, underlying hypermobility, or a constant sudden balance problem. Greatest demands on sacroiliac joint occur as forces generated by the legs are transferred to the trunk during early to mid-drive.
- **Lumbar disc herniation:** Also very common. Compressive loads applied to the lumbar spine in flexion may contribute to disc bulge or herniation. This may be associated with or progress to spinal nerve impingement, causing radicular symptoms (Fig. 83-3). However, it is important to remember that initial symptoms may be limited to centralized back pain.
- **Spondylolysis:** Observed in young rowers, likely caused by repetitive axial loading of pars interarticularis. Weight training rather than rowing induced all cases of spondylolysis seen in one group of rowers. If spondylolysis exists bilaterally at one level of the spine, then spondylolisthesis (forward slippage of one vertebra on the one below) can result (see Fig. 83-3).

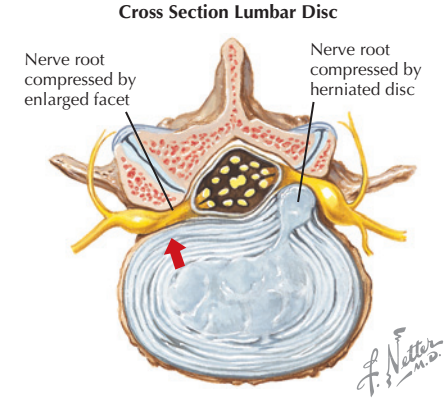
History: Usually insidious onset during periods of high-volume or high-intensity training. Pain may be localized to one region and radicular symptoms (pain or numbness radiating into legs) may be present. If pain worse with flexion and extending from flexed position, suspect muscle origin and disc pathology. Pain worse with extension may indicate spondylolysis and spondylolisthesis.

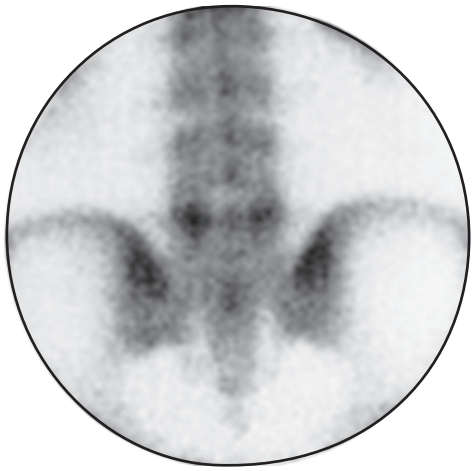
Physical exam: Standard; observe range of motion, reflexes, distal motor and sensory nerve function, and perform straight leg raise. Special addition: standing single-leg back extension for spondylolysis. (Worse back pain on the side of the standing leg.) Straight leg raise, sitting root, and neural tension tests for discogenic and herniation problems.

Diagnostics: If indicated, begin with plain x-rays with oblique views to search for pars interarticularis defect, or proceed to bone scan or computed tomography (CT) including single photon emission (SPECT) (see Fig. 83-3). If suspected disc herniation, proceed to magnetic resonance imaging (MRI).

Treatment: Include therapeutic modalities such as ultrasound and interferential current as indicated and/or mobilization/manipulation. Incorporate core stabilization exercises, relative rest, and cross-training into training regimen, with gradual return to sport. Disc herniation infrequently requires surgical intervention, although outcomes are similar. Spondylolysis usually requires extended periods of time off from training (for example, a full season).

Technical changes: Maintaining a more neutral spine during stroke may be helpful. Although changes can be made in equipment to lessen load per stroke, this may be unrealistic during the racing season if rower is in a crew boat. Rigging changes also have an effect on other crew members and run of the boat, and speed may be sacrificed. However, lowering the load setting on the rowing ergometer during the indoor season is recommended and simple to do. Address possible predisposing factors: low hamstring-to-quadriceps strength ratio, strength asymmetries between right and left erector spinae muscles, particularly during extension, and hip muscle imbalances in females. If feasible, practice may be moved until later in the day, as lumbar discs may be more vulnerable during the early morning hours because of overnight absorption of fluid from surrounding tissues. Emphasize appropriate breathing patterns: Expiring through the drive phase may offset high levels of shear force and compression. Inspiratory muscle training stabilizes the thorax and may improve rowing performance.





Bone scan of the lumbosacral spine showing bilateral spondylolysis at L4–5.

SPECT imaging of the same patient showing bilateral spondylolysis at L4–5.

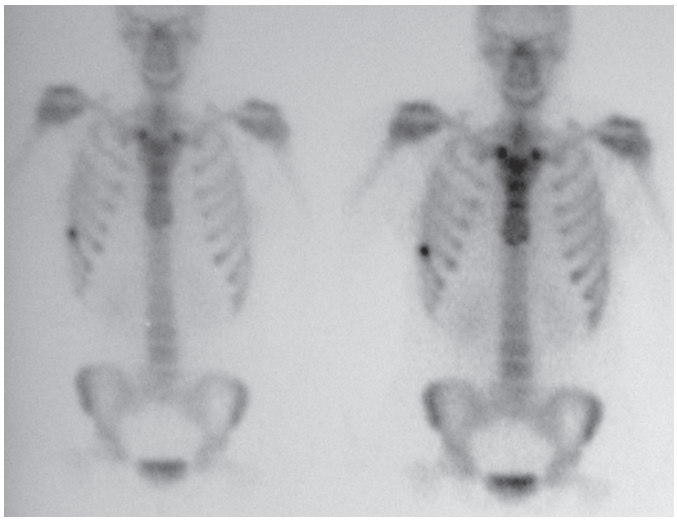
Figure 83-3 Low Back Injuries.

Thorax

Mechanism of injury: Remains controversial. Factors include weakness of surrounding musculature, repetitive strain, and muscle imbalances between serratus anterior and external obliques. May result from pull of muscular attachments, cumulative compressive forces, or specific rib architecture (angulation, diameter, etc.) (Fig. 83-4).

Types of injury:

- **Rib stress fracture:** Increased incidence after more efficient blade design in 1992; rates of occurrence range from 6% to



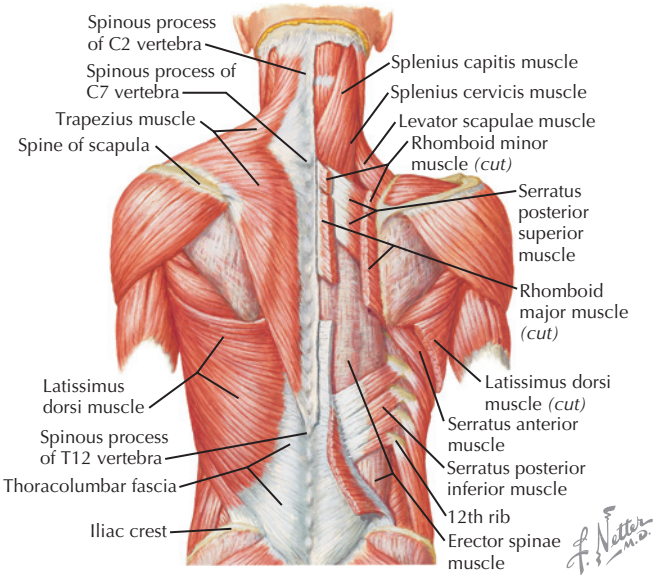


Figure 83-4 Thorax Injuries.

23%; higher incidence in female rowers. Most common in anterolateral and posterolateral aspects of fifth through ninth ribs.

- **Costochondritis and costovertebral joint subluxation:** Poorly understood condition, most likely to occur during sweep rowing because of excessive rotation.
- **Intercostal, rhomboid, and serratus anterior muscle strain:** Often confused with rib stress fracture at initial presentation. Case study of complete avulsion injury of serratus anterior.

History: Usually insidious onset with generalized chest wall pain; worse with rowing, reaching, rolling over in bed. Over time, pain associated with stress fracture becomes more localized to specific rib. Costochondritis and subluxation will exhibit pain and tenderness on costochondral or costosternal joints without swelling. Muscle strain exhibits nonspecific tenderness on palpation.

Physical exam: Stress fracture: pain localized over rib; most commonly anterolateral fifth through ninth ribs but can be posterior; worse with rib compression. Athlete may have palpable rib callus. Costochondritis or subluxation: adduction of arm on affected side and rotation of head toward the affected side will reproduce pain. Examine thoracic rib cage to ensure proper segmental rotation.

Diagnostics: Important to exclude stress fracture. Plain x-rays may detect callus formation, but bone scan remains gold standard for timely diagnosis. Single photon emission computerized tomography (SPECT) will increase sensitivity of bone scan (see Fig. 83-4).

Treatment: For stress fracture: similar to other stress fractures; relative rest (4 to 6 weeks), with gradual return to sport. Serratus anterior strengthening (scapular protraction) may be useful long term. Muscle strain: often rower is able to return to sport earlier, even with lingering pain (not indicated for stress fractures).

Technical changes: During early return-to-sport phase, rowing with less forward reach from shoulders and rotation at catch will lessen scapular protraction and may be protective. Address any muscular imbalances or hypermobility in specific regions. Female rowers should ensure they are not amenorrheic or calcium deficient, because these states will lead to decreased bone density and a higher rate of fracture.

Shoulder

Mechanism of injury: At catch phase of stroke, scapula is retracted and humerus is elevated to transfer forces from oar handle to legs and back. Most common pathology is combination of anteriorly placed glenohumeral head, tight posterior capsule, tight latissimus dorsi, and weak rotator cuff muscles. Common technical error is to activate upper fibers of trapezius at catch instead of engaging the mid-to-lower trapezius and latissimus dorsi, particularly in scullers. Usually exaggerated in outside (furthest from blade) shoulder in sweep rower.

Types of injury:

- **Rotator cuff tendinosis:** Overuse injury related to technical error and/or muscle strength imbalance.
- **Capsular instability:** May be exaggerated by reaching too far forward at the catch or not extending fully at the finish through the upper back.
- **Clavicular stress fracture:** Case report of lightweight rower with sudden resumption of training; uncommon but highlights need to consider surrounding structures of shoulder girdle when considering differential diagnosis.

Physical exam and treatment: On physical examination there will be localized tenderness over the affected structure or muscle insertion (most commonly the supraspinatus and/or biceps, although the infraspinatus and subscapularis may also be affected). Physical therapy should focus on stretching of the tight posterior capsule, correction of posture and muscle imbalance, with special attention to scapular stability. Surgical correction may be indicated in severe cases of capsular instability.

Technical changes: Limit over-reaching at catch, and ensure linear motion of shoulder girdle through entire drive.

Knee and Hip

Mechanism of injury: Improper foot and knee alignment during each phase of the stroke cycle can lead to overuse injury and pain in the knee and hip region. Vastus medialis muscle usually underdeveloped in rowers and may lead to patellar tracking problems.

Types of injury:

- **Patellofemoral pain:** Two main factors to consider for patellofemoral pain: overcompression at the catch straining surrounding structures of flexed knee, and tendency for knees to

buckle or pop up slightly at finish. If athlete cannot fully extend at the finish, vastus medialis muscle will be prevented from normal function; the three remaining muscles of quadriceps are strengthened during stroke and the imbalance will lead to lateral tracking of patella. Some female rowers may be predisposed to patellofemoral pain because of wider pelvis (Fig. 83-5).

- **Iliotibial band (ITB) friction syndrome:** Compression at the catch combined with varus knee alignment may cause friction and ensuing pain as the iliotibial tract slides over lateral condylar prominence of knee. Rowers who incorporate running into their training may encounter similar problems.

History: Patellofemoral: dull generalized pain in retropatellar area, worse going up and down stairs, or prolonged bent knee position (positive "theater sign"). ITB: pain over lateral aspect of knee, rest of knee exam typically normal.

Physical exam: Usual knee and ITB exam. Look for lateral tracking with bent knee, malalignment such as genu valgum or genu recurvatum, excessive pronation or internal tibial torsion. ITB symptoms should prompt examination of possible leg length discrepancy or pelvic malalignment.

Diagnostics: None indicated, but large knee effusion or significant locking or catching may suggest meniscal or other knee pathologies.

Treatment: Taping the patella to prevent tracking useful in the short term. Strengthening of vastus medialis and gluteus medius muscles. Bracing not advised because of range of motion limitation.

Technical changes: Modify shoe position to alleviate symptoms (angle, distance between toes and heels, height). Wide foot placement may compound the problem. Modify position of tracks to ensure full knee extension at finish of stroke.

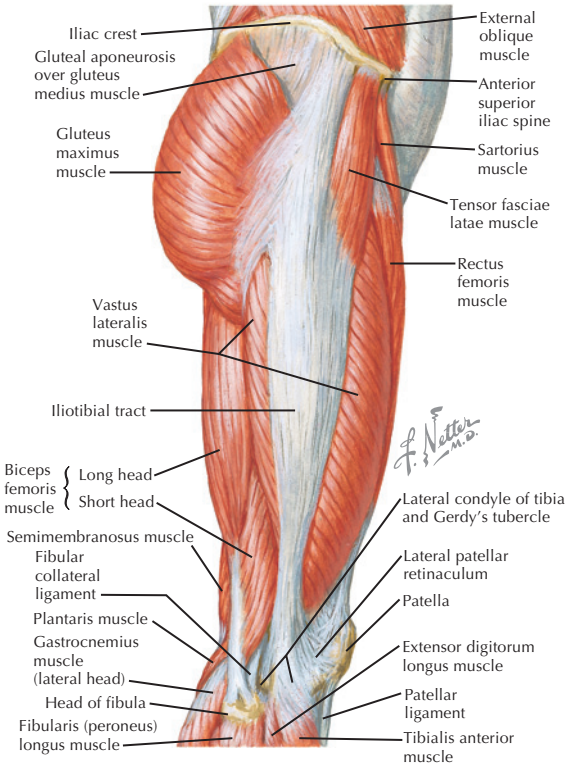


Figure 83-5 Knee and Hip Injuries.

Wrist and Forearm

Mechanism of injury: Relatively common, and most often resulting from excessive wrist motion during feathering and squaring actions, as well as excessively tight grip of the handle in rough water conditions or with inexperienced rowers. Other factors: wrongly sized or slippery grips, poor rigging, and fatigue.

Types of injury:

- **Exertional compartment syndrome:** Often volar compartment, overuse injury that may occur because of repetitive and improper initiation of pull-through with the elbow instead of the shoulder girdle at the catch, or inability to relax forearm grip at release.
- **Lateral epicondylitis:** May also occur because of factors involved with exertional compartment syndrome; characterized by pain over lateral aspect of elbow and with resisted wrist extension.
- **De Quervain's and intersection syndrome:** Tenosynovitis of first and second dorsal compartments respectively. Intersection syndrome, often misdiagnosed as de Quervain's, is also referred to as "oarsmen's wrist" (Fig. 83-6).

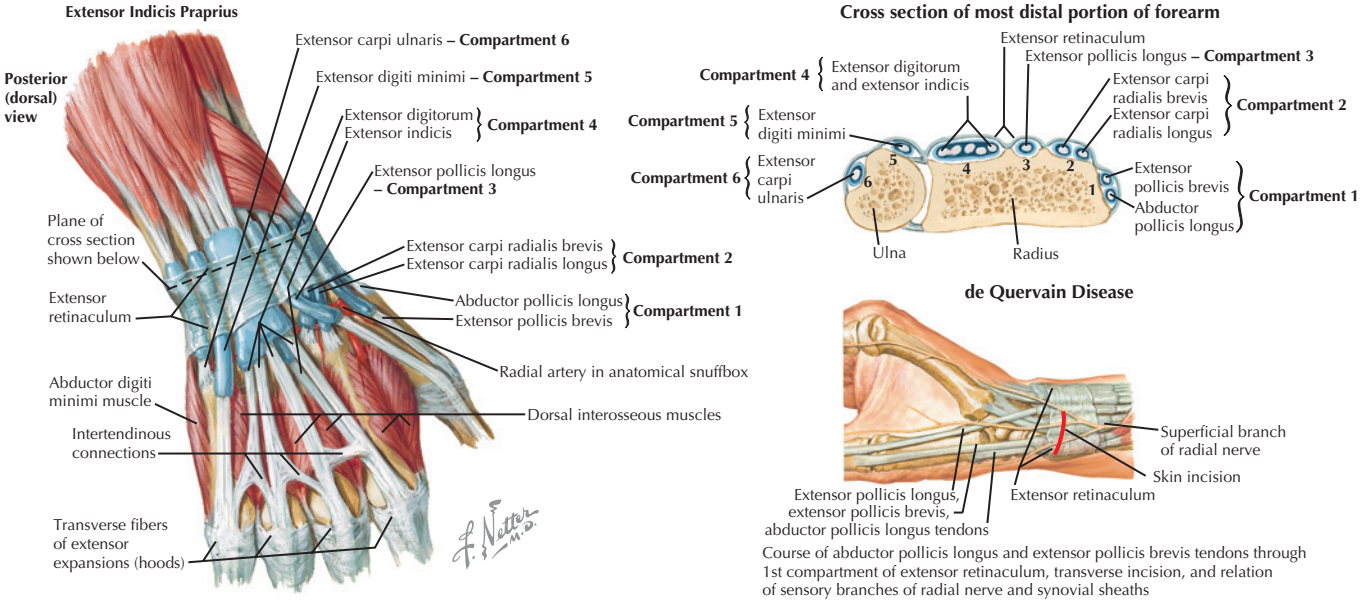


Figure 83-6 Wrist and Forearm Injuries.

- **Tenosynovitis of wrist extensors or "sculler's thumb":** Swelling over dorsal aspect of forearm, cause by hypertrophy of abductor pollicis longus and extensor pollicis brevis muscle bellies. May be due to improper use of thumb to feather the oar at the finish or by allowing palm to slide down the handle while keeping the thumb rigid against the end of the handle.
- **Carpal tunnel syndrome:** Pain over wrist and distal median nerve parethesias. Positive Tinel's and Phalen's sign and evidence of wasting of the thenar eminence in severe cases.

History: Pain in wrist or forearm region, particularly when rowing at high stroke rates, or in cold or bad weather conditions. Often pain will lead to inability to feather or square properly, burning or swelling feeling.

Physical exam and treatment: Physical examination will reveal pain and tenderness over the affected muscles, with additional discomfort on resisted muscle testing. Diagnosis of exertional compartment syndrome may require examination for pain and compartment tightness immediately after exercise, because there may be few physical findings at rest. If feathering action causes pain, advise athlete to row "on the square" or on the ergometer. Cortisone injection only when conservative treatment fails. Steroid injection (0.5 to 1 mL) into tendon sheath (for tenosynovitis) or into insertion of wrist extensors onto lateral epicondyle (for lateral epicondylitis), with return to rowing in 1 to 3 days. Surgical intervention only required in more severe cases.

Technical changes: Encourage relaxed grip without excessive wrist motion. Ensure proper grip size and material. Advise use of pogies (fleece or similar material coverings for the hands and oar handle) in cold weather.

Sciatic Nerve Compression

Mechanism of injury: Rowers' seats are generally made of wood or carbon fiber, and are molded specifically to accommodate ischial tuberosities. However, mold is usually generic and may not fit a particular rower's pelvic width, leading to constant compression of the sciatic nerve.

History: Numbness radiating down legs, only when rowing, without back pain or leg weakness.

Physical exam: Standard, rule out radiculopathy, disc disease, and piriformis syndrome (compression of the sciatic nerve by a tight piriformis muscle) (Fig. 83-7).

Technical change: Change seats or use a seat pad. Elevate or lower shoes in boat.

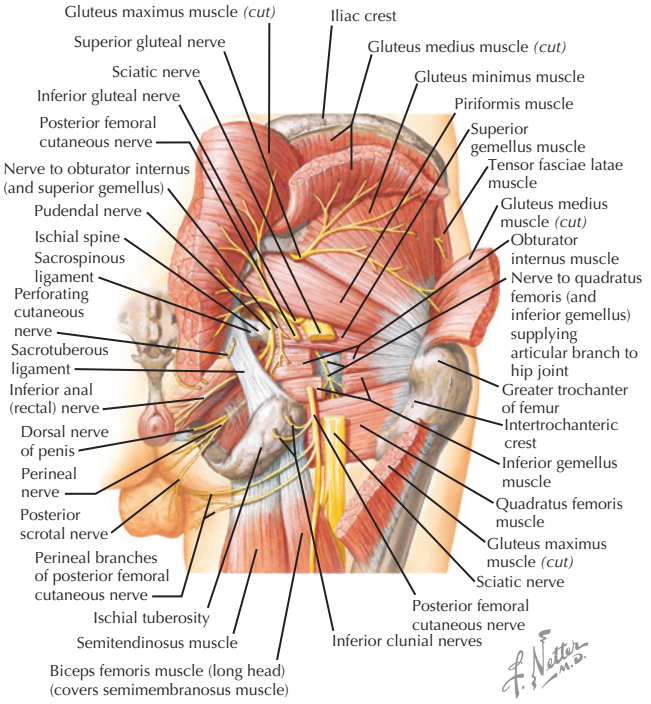


Figure 83-7 Sciatic Nerve Compression.

Hand Blisters and Skin Abrasions

Mechanism of injury: Most severe upon resumption of on-water training. Blisters and abrasions can occur at any of the three points of contact: hands on the oar, buttocks on seat, feet in shoes. Imperative to prevent secondary infection or scarring.

Types of injury:

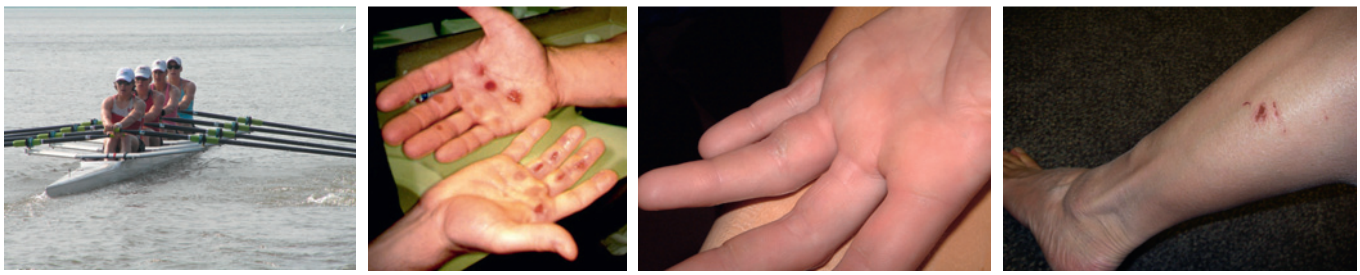
- **Hand blisters:** Found on anterior aspect of fingers and palm. Caused by repetitive friction between skin and oar handle. Usually only painful when rower resumes on-water training, but can be affected by heat, humidity, and change in grip size or material. May lead to infection ("sausage finger") if not cared for in acute stages (Fig. 83-8).
- **Sculler's knuckles:** Superficial abrasions on dorsal aspect of right hand, caused by crossover of port and starboard handles in the middle of the drive or recovery. Can be a source of significant bleeding and pain while rowing (see Fig. 83-8).
- **Track bites:** Superficial abrasions on posterior aspect of lower leg, caused by tracks for sliding seat digging into calf muscles. Can lead to scarring (see Fig. 83-8).
- **Rower's rump:** Term coined for rowing-associated lichen simplex chronicus case. Uncommon, but many rowers have superficial abrasions on the buttocks, ranging from slight indentations to skin ulcerations, secondary to repetitive chafing caused by improperly fitted seat.

History: Recent resumption of on-water training, increase of training volume, or sudden shift in weather or equipment (sweep to sculling, or grip change).

Physical exam and treatment: Standard. Monitor closely for signs of secondary infection (erythema, fever, red streaking along lymphatic drainage route).

Technical changes:

- **Hand blisters:** Change grip material and/or size of handles. Many manufacturers offer a variety of grip material (wood,



Crossover in sculling. Blisters. Sausage fingers. Slide bites.

Figure 83-8 Hand Blisters and Skin Abrasions. *(Crossover in sculling photograph © Jackie Skender.)*

hard plastic, foam, etc.), although some rowers opt to use tennis racket grip, which they tape on themselves. However, in most cases some blistering will be unavoidable. Ensure scrubbing of oar handle after each use if shared among crew. Case study of spread of hand warts caused by oar sharing among crew.

- **Track bites:** Use circumferential tape for track bites, consider moving tracks wider or toward bow of boat if possible.
- **Sculler's knuckles:** Height can be modified on oarlock to ensure larger gap between port and starboard oar handles. Circumferential tape may help in the short term. Gloves not advised because further blistering may occur and proprioceptive feedback of oar placement in water is compromised.
- **Rower's rump:** Regular application of corticosteroids in severe cases. Most cases change seat or use seat pad.

Body Composition Issues and Disordered Eating

- Rowers, particularly those who need to meet a weight requirement, often restrict calorie intake to the point of disordered eating practice.
- This can result in lowered bone density and other musculoskeletal, cardiovascular, and electrolyte abnormalities.
- In females, resultant inadequate energy intake for energy needs can result in menstrual dysfunction and altered bone mineral density (female athlete triad).
- Prevent through education, awareness, nutritional counseling, and appropriate selection of athlete's naturally maximal weight.
- Need for health care professionals to be aware of disordered eating behaviors, menstrual dysfunction, and potential for altered bone mineral density (see Chapters 10 and 22).

ENVIRONMENTAL EXPOSURE AND SAFETY CONSIDERATIONS

Personnel: Coaches should always accompany or be in sight of rowing shells, with appropriate numbers of personal flotation devices (PFDs), and communication devices to call for outside help if required. Collisions may occur as a result of rowers facing backward and not checking course or steering often enough. Rowers should be advised to stay with overturned shell and keep oars with boat as they can assist with flotation.

Storms and wind conditions: High winds and choppy water can lead to swamping of boats, because rowing shells sit very low in water. No boating of any kind should be attempted during a thunderstorm.

Sun exposure: Athletes and coaches should wear waterproof and sweatproof sunscreen, as well as sunglasses and hats during prolonged sun exposure.

Cold exposure: Early and late season rowing places the rower at risk for excessive heat loss. Rowers should dress in layers, avoid splashing with oars, and wear fleece pogies and hats during colder temperature rowing.

Water conditions: Whitecaps signify high winds and should be avoided. For rowers who train on rivers, currents and eddies may be dangerous, particularly during flood conditions.

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In-line Skating, Skateboarding, and Bicycle Motocross

84

Erica L. Kroncke and Craig C. Young

IN-LINE SKATING

History

- The existence of in-line skates dates to 1849 when Louis Legrange crafted a pair with wooden wheels to simulate ice skates for a scene in Giacomo Meyerbeer's opera *La Prophète*.
- In the 1970s in-line skating was principally limited to hockey players looking for a way to practice during the summers.
- In the 1990s the popularity of in-line skating exploded; it became the fastest growing recreational activity of the decade.
- Now more than 10 million people own in-line skates, and many additional individuals rent them to roll down the nation's parkways and pathways.

Equipment

Boots: Higher and firmer boots provide more ankle support, better control, and are used by beginners and trick skaters. Lower boots allow more ankle flexion and are used by speed skaters.

Frame: Shorter frames are more maneuverable; longer frames are faster and more stable. Most recreational skaters use four-wheeled frames. Speed skaters use five-wheeled frames. Artistic skaters use two- or three-wheeled frames.

Wheels: Wheel sizes vary depending on skating style: average recreational skate has wheels that are 72 to 90 mm in diameter, smaller wheels (48 to 72 mm) are used for tricks and dancing, larger wheels (80 to 100 mm) are used for speed skating. Harder wheels are faster and more durable, but soft wheels may have better grip. Elliptic profiles minimize friction and increase speed, whereas more rounded profiles have better grip and are more stable.

Bearings: Higher rated Annular Bearing Engineering Committee (ABEC scale) bearings are better in overall quality and theoretically translate into faster and smoother rides with less friction; however, this has never been proven to affect performance.

Brakes: Most in-line skates have a hard rubber brake built into the heel of one of the skates that creates friction against the pavement. Standard rear brake pressure does not allow for quick or efficient stopping. Older styles require the skater to lift the toe, which causes loss of contact between the front wheels and the ground and reduces stability. Expert skaters, trick skaters, figure skaters, and speed skaters often use skates that do not have brakes. Braking technique used by skaters for most of their stops: standard rear skate brake pressure (52.3%), spin stops (13.3%), "T-skate drag stops" in which a skate is dragged behind the body with the wheels perpendicular to the skate axis (11.1%), skating off into the grass (14.6%).

Competitions and Skating Styles

- Competitive in-line skating events such as the following have grown with the popularity of the sport:
  - Speed skating in sprint and long distance events.
  - Team sports such as roller hockey and roller derby.
  - Group skating as a social activity; large groups of skaters meet to skate city streets in the United States and Europe; one of the largest is the Pari Roller in France, involving up to 35,000 skaters in one night.
  - Figure skating and dancing on in-line skates.
  - Half-pipe or "vert" competitions in which the tricks are performed at or above the rim of the pipe.
  - Slalom racing thorough cones.
  - Recent growth has been in increasingly aggressive skating events such as "big air competitions" that rate competitors on height of jump, execution of tricks, and artistic performance.
    - Street competition—tricks, grinds, and jumps on railings, curbs, stairs, and ramps and other obstacles found in typical urban settings.
    - Free skating (also known as urban skating or free "riking")—the objective is to get from one point to another by the fastest possible route, by skating quickly through city streets and negotiating all obstacles with jumps and slides as needed.
    - Off-road skating on dirt trails while on skates using special "all-terrain" wheels.
    - These more extreme forms of in-line skating increase potential for injuries, and especially for the more severe injuries including head injuries.
- With increased participation came an increase in injuries.
  - The Centers for Disease Control and Prevention (CDC) estimates more than 100,000 in-line skating injuries per year are serious enough to require emergency room evaluation. This places in-line skating in the top 10 in terms of frequency of injury from a recreational activity, along with sports such as basketball, bicycling, football, baseball, skiing, and soccer.
  - In-line skate injuries tend to be more severe than other sports, with 15% to 30% or more of in-line skating injuries evaluated in emergency rooms requiring hospital admission.

Injury Patterns

Risk Factors

- Male: ratio of male-to-female injured skaters is 1.5:1 to 2:1.
- Age: 10 to 14 year olds account for the majority of injuries.
- Inexperienced skaters are at an increased risk for injury; 25% of skaters in a small study were injured their first time out
- Experienced skaters are also at increased for injury risk related to performing skating tricks and dose-response skating (i.e., the more hours per week spent skating, the higher the number of injuries, specifically more than 10 hours per week).
- Skitching is a dangerous practice of skaters hanging on to the back of a moving vehicle; it can lead to speeds of more than 70 mph and risk for significant injury.
- Deaths from in-line skating usually involve collisions with motor vehicles.

Falling Patterns

- The most common reason for involuntary falls was loss of balance.
- Other common reasons were hitting rocks or other small objects, uneven pavement, failure to stop, and collisions.
- The most frequent site of initial impact after an unintentional fall was on the hands and wrists (44.6%).

Frequency

- 10% to 25% of skaters have reported suffering at least one in-line skating–related injury.

Location

- Approximately two-thirds of the injuries occur in the upper extremity, with the wrist being the most common locus of injury.
- Wrist and forearm are the most frequent sites of injury, especially of the more severe injuries (e.g., fractures and dislocations).
- The distal radius is the location of about 50% of all in-line skating fractures. Other common fracture sites include the scaphoid bone, the radial head, and the ulna.
- The knee is the most common location for injury in lower extremity.
- Head injuries account for up to 5% of serious injuries.

Protective Equipment

- Significantly reduces the risk of injury.
- Schieber's study suggested that failure to wear wrist guards may increase the relative risk of a wrist injury by 10.4; nonuse of elbow pads may increase the relative risk of elbow injury by 9.5.
- Skaters who are not using protective equipment when injured were more likely to have a severe injury.
- Cadaveric studies that used high-speed impact loading suggest that wrist guards may reduce injuries.
- Approximately half of the in-line fatalities in the CDC database are from head injuries. The CDC estimates that in-line skating causes more than 1200 head injuries per year.
- Helmets have been shown to substantially reduce the risk of injury in bicyclists; this may be even more important to skaters who can reach speeds in excess of 40 mph on downhill stretches and have much less efficient brakes
- Fatally injured skaters universally seem to be non–helmet wearers.
- Helmets
  - For recreational skaters and speed skaters should conform to American Society for Testing and Materials (ASTM) standard F-1751. These are designed to absorb only a single hard impact and should be professionally inspected or replaced after any hard helmet impact.
  - Freestyle skaters should conform to ASTM standard F-1492 or to Snell N-94 Multi-Purpose standard (existing skateboarding helmet standard), which are designed for multiple impacts of somewhat lower energy.

Use of Protective Equipment

- Wrist guards are the most commonly worn protective equipment, worn by approximately one-half to two-thirds of skaters.
- Helmets are rarely worn (2.6%).
- A peer effect seemed to increase the use of protective equipment in observational studies. Groups of two or three skaters had a significantly increased likelihood of all members either wearing equipment or not wearing equipment.
- Adolescent skaters were much less likely to use protective equipment.
- Factors that most influenced use of protective equipment in adolescents were parents (40%), requirement of skating location (33%), friends (15%), and coaches (14%).
- Common reasons for not wearing protective equipment included lack of perceived need (47.3%) and discomfort (37.5%).
- Adolescent skaters cited discomfort (56%), lack of perceived need (47%), and appearance (25%).

Risk of Protective Equipment

- The equipment has the potential to transmit forces to areas away from the impact site, placing other areas of the body at risk for injury.
- Thus, the wrist guard may place the in-line skater at risk for "splint-top" fractures.

Other Risk Factors

- Skating location factors: pavement condition, visibility, volume of pedestrian, bicycle and motor vehicle traffic, hills.
- Experience: beginners should consider lessons, formal instruction in school-based program has been shown to have a small but significant positive effect in attitude toward and use of protective equipment.

SKATEBOARDING

History

- The modern day skateboard is thought to be an adaptation of the scooterboard (a wooden crate connected to a board and attached to rollerskate or other wheels) or soapbox. No definitive inventor is known.
- Skateboarding has experienced intermittent periods of popularity since the 1960s, most recently in the last decade.
- Currently the number of skateboarders is estimated at 9.7 million (National Sporting Goods Association).

Equipment

- Three parts make up a skateboard
  - Deck: 7 to 9 inches wide; 28 to 33 inches in length; made of aluminum, bamboo, carbon fiber, fiberglass, Kevlar, plastic, or wood. Longer, inflexible decks are safer for beginners; shorter, more flexible decks lead to increased maneuverability. Wider decks for vert (short for vertical) skating. The longboard ("sidewalk surfer") is a variant of the skateboard with a longer deck, larger wheels, and a more stable truck. Boards in the past were often made in the shape of a surfboard without concavity.
  - Modern skateboards usually have nose and tail kicks (upward curves) with a concavity between them, allowing cupping of the foot for more control.
  - Wheels: Usually polyurethane or plastic. Wide wheels ("stokers") are 6 to 10 cm and provide more stability; narrower wheels ("slicks") are 3 to 4 cm and increase maneuverability.
  - Truck: Made up of axles, frames, and hardware, the trucks connect the wheels to the deck.
- Stance
  - Regular foot (left foot forward) is most common.
  - Goofy foot (right foot forward).
- Improved technology (polyurethane wheels) has led to increased speed and maneuverability, as well as increasing numbers of skating tricks.

Competitions and Skating Styles

- Vert uses 8- to 10-foot high ramps (usually half-pipes); involves "big air" flare.
- In slalom skating (weaving between cones), the key is a technique called pumping that allows skaters to accelerate with every turn.
- Downhill usually involves coasting or racing downhill on a longboard.
- Cruising also involves longboards, but denotes riding from one place to another without focusing on tricks or speeding down hills.
- Freestyle emphasizes technical flat-ground skating; essentially "dancing" with a skateboard.
- Street skating is boarding on sidewalks, streets, parking lots, etc; it may include tricks or skating on handrails, stairs, and lower ramps.
- Pool (skating in a drained out swimming pool) or park (as in skate park) skating is named based on location.
- Off road or dirt-boarding uses a large board and wheels with the skater's feet strapped in like in snowboarding.
- New types of skateboarding continue to be developed.
- X Games have popularized competitive skateboarding.

Injury Patterns

Risk Factors

- Male: Consumer Products Safety Commission (CPSC) reviewed 11 years of injury data and found approximately 87% of injured skaters were male.
- Age: 10- to 14-year-olds are the group most often injured. Among CPSC age groups, 5- to 14-year-olds have the most injuries.
- Experience: One third of injuries occur in the first week; inexperienced and experienced skaters at increased risk as discussed in "In-line Skating" section.

Injuries

- Skateboard injuries are increasing.
- Upper extremity injuries are most common and occur as a result of falling on outstretched hands.
- Approximately half of the injuries presenting to emergency departments (ED) were fractures; forearm, wrist, and ankle most common.
- Ankle sprains also a common injury.
- Head injuries account for almost 7% of injuries.
- 3:1 ratio of left to right side injuries due to regular stance.
- Similar to in-line skating, the majority of deaths in skateboarding involve motor vehicle collisions.
- Rate of hospital ED–treated injury for skateboarding is approximately four times that of in-line skating, comparable to snowboarding, and roughly a quarter the rate for football.
- Compared with other types of skating, skateboarding injuries were two to eight times more likely to be severe or critical.
- Skateboard elbow: olecranon process fracture caused by falling directly on one or both elbows.

Skate Parks

- Skaters are at less risk to be injured from motor vehicles.
- However, studies show increased injuries following the opening of skate parks, likely because of lack of supervision and personal protective equipment.

Prevention

- Use of personal protective equipment (PPE) decreases the risk of injury.
  - Injury less likely to require hospital admission if wearing PPE.
  - Helmet use would have prevented all the head injury admissions over a 30-month period.
- Recommended PPE in skateboarding
  - American Academy of Pediatrics (AAP), American Academy of Orthopedic Surgeons (AAOS), and CPSC recommend helmets, wrist guards, elbow pads, and knee pads for skateboarding.
  - PPE made for in-line or roller skating, biking, or motorcycling can be used for skateboarding.
  - Helmets: Should have American National Standards Institute (ANSI) or ASTM stickers of approval; 18% of hospitalizations from skateboarding are due to head injuries. Nearly 50% of head injuries in youth and adolescents during sports and recreational activities occur while skateboarding, skating, and bicycle riding.
- Actual use of PPE
  - 13% to 33% of skateboarders of all ages wear PPE.
  - Requirement of skating location, parents, and peers are the most common reasons adolescent skateboarders wear PPE; discomfort, lack of perceived need, and appearance were most commonly cited by adolescents for nonuse of PPE.
  - Adolescent skateboarders report a high (defined as sometimes, usually, or always) rate of PPE use only 9% to 35% of time.
- Minimum age: AAP advises children younger than 5 years should not ride skateboards.
- Formal instruction has been shown to have a small but significant positive effect in attitude toward and use of protective equipment.

BICYCLE MOTOCROSS (BMX)

History

- Started in California in the 1960s, around the time that motocross (motorcycles) became popular.
  - For riders who wanted to participate in motocross but did not have the means, bicycle motocross (BMX) was an option.
  - Riders dressed in full motocross gear and raced on tracks they made themselves.
- Scot Breithaupt, considered the founder of BMX, organized the first race in 1971; manufacturers began making bikes with 20-inch wheels specifically for this growing sport.
- Union Cycliste Internationale (UCI, or International Cycling Association) is the governing body for cycling. It also manages the classification for cycling disciplines, which include:
  - BMX
  - Road racing
  - Track cycling
  - Mountain biking
  - Cyclo-cross
  - Indoor
  - Paracycling
- Two sanctioning bodies of BMX racing in United States:
  - NBL (National Bicycle League), which is certified under the UCI
  - ABA (American Bicycle Association)
- International BMX Federation was founded in 1981.
- In 2003, the International Olympic Committee (IOC) decided to introduce BMX in the 2008 Olympic Games in Beijing, China.
- Approximately 70%, or 4 million, of the BMX bikers in the United States are between the ages of 17 and 43 years old.
  - BMX is among the top 10 most popular extreme sports (with in-line skating and skateboarding being No. 1 and No. 2) according to the Sporting Goods Manufacturers Association.

Competitions and Styles

BMX Racing

- Track
  - Closed loop of earthen material 300 to 400 meters in length and 5 to 10 meters wide.
  - Jumps, banked corners, and other obstacles are included.
  - Electronic-controlled starting gate.
- Head-to-head competition of up to eight riders per heat (qualifying rounds, quarter finals, semifinals, finals); the top four qualify for the next round.
- Competition classes are based on age, gender, bike style, and level.
- Junior and elite levels.
- Racers may reach speeds upward of 30 mph.
- Majority of racers are male, although the number of females in the sport has increased.

Freestyle BMX

- Evolved from BMX racing when riders focused on aerial maneuvers and began to merge skateboard park riding with stunts and tricks.
- Events are timed and judged based on difficulty and originality.
- Riders usually participate in more than one discipline.

- The disciplines or styles may overlap to some extent, and as new styles of riding are developed the current may change as with skateboarding.
- Disciplines
  - Street: Riding on streets or public property; allows for creativity, as almost anything can be used as an obstacle.
  - Park (or skate park) riding can differ based on whether the skate park is predominantly wood or concrete, because concrete lends itself to a faster, smoother style.
  - Vert: Analogous to vert in skateboarding, this discipline involves riders performing tricks on a vert ramp (half-pipe). The largest vert ramp to date is the X Games big air ramp, 27 feet tall.
  - Trails: Trail riding (also known as dirt jumping) involves lines of jumps built from compact dirt. Airborne riders often perform tricks prior to landing.
  - Flatland riding terrain is smooth and flat as the name suggests. The majority of tricks involve spinning as well as balancing the rider and the bike in variety of positions.
- Competitions
  - May or may not be timed, depends on the discipline.
  - Judged (based on discipline) on combinations of difficulty, originality, creativity, style, flow, numbers of maneuvers, and height.

Equipment

- BMX bikes usually have 20-inch wheels.
- Some bikes are free-wheeling (i.e., wheels operate independent of pedal motion).
- Racing bikes
  - Standard: 20- to 22½-inch wheels.
  - Cruiser: 22½- to 26-inch wheels.
  - Typically with single brake in rear.
- Freestyle bikes
  - Street bikes are often the strongest and heaviest.
  - Dirt jumping bikes are heavier than racing bikes but lighter than other freestyle bikes; may only have a rear brake.
  - Flatland bikes have a shorter wheelbase with frames that are more reinforced.
  - Most freestyle bikes have front and rear brakes; the handlebar is designed to completely spin around without twisting the front brake cable.
  - Has axle pegs for tricks.
- Pedals
  - Platform (flat) pedals without a cage are most often used in BMX; these pedals offer grip when using short metal studs and do less damage if the rider is in an accident.
  - Toe clips generally are not used.

Injuries

- Risk factors
  - Stunts/tricks
  - Poor technique or mishandling of the bicycle (e.g., foot came off pedal)
  - Inexperience
  - Male: more than 93% of injuries treated in emergency departments are male
- Types
  - Most injuries are acute, although overuse injuries are described.
  - In studies reporting BMX injuries seen at emergency departments, mild injuries seem to be most common (abrasions, contusions, minor soft tissue injuries), although 7% of injuries in one study were head injuries.
  - Abdominal, scrotal/genitourinary, perineal, and spinal injuries are reported.
  - Extremity injuries are most common, but facial injuries are also common (21% to 22%).
  - Comparison of BMX with conventional bicycle injuries shows conflicting results.
  - Racing injuries at the 1989 European BMX Championships:
  - 61 injuries among 976 participants.
    - Injury breakdown: 42% abrasions, 29% contusions, 8% sprains, 4% fractures (majority upper extremity), 2% concussion, 2% other.
    - Females injured more often than males.
    - Injury rate: 1190 injuries per 1000 competition hours.

Personal Protective Equipment

- Helmets
  - Required for all types of riding.
  - Full face or open face (latter must be used with mouth guards).
  - CPSC set standards for bicycle helmets after 1999.
- In addition, long-sleeved shirts and pants with gloves and elbow pads required at UCI-sanctioned races
- Knee pads
- Closed-toe shoes
- Chest protectors
- Shin guards

RECOMMENDED READINGS

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Rugby

Lucien Parrillo and William W. Dexter

GENERAL INFORMATION

- Rugby is an internationally played sport, second only to soccer in popularity.
- It originally derived from soccer in 1823 when Englishman William Webb Ellis, "in a fine disregard for the rules," picked up the ball and ran with it.
- Today, rugby is one of the most popular club sports and fastest growing women's sport in the United States.
- Further, it is one of the only contact/collision sports that is played by women, under the same rules that apply to men.
- Although there are several distinct styles of rugby ("rugby league," "seven-a-side rugby," etc.), rugby union is by far the most popular and widely played.

BASICS OF PLAY

Pitch (Field)

- The field measures (not more than) 100 meters long by (not more than) 70 meters wide.
- The in-goal area extends not more than 20 meters and not less than 10 meters beyond each goal line.
- The goal posts are 5.6 meters apart, with a crossbar 3 meters from the ground and must extend a minimum of 3.4 meters high and must be padded.

Match

- There are two halves lasting 40 minutes each, with no more than a 10-minute halftime and stoppage time allotted at the end of each half.
- Play is continual; however, a player may leave the field for up to 10 minutes for injury care. During this time, a substitution may be made for that player.
- A player may return to the field later in the match after being substituted with another player.
- A front row player shall only be replaced in the front row by another front row player.
- Substitutions can be made only at stoppages in play, must occur on the nominated side of the pitch, be under the direction of the touch judge, and must be carried out quickly and efficiently.

Scoring

- A **try** occurs when a team touches the ball down over the opposing team's in-goal line; worth 5 points.
- After a try is scored, a **conversion** attempt is made by kicking the ball through the goal posts any distance back from where the try was scored; worth 2 points.
- A **goal** is most commonly made by place kick when a penalty is called, or it may also be made during open play by drop-kick; worth 3 points.

Game Play

- Rugby is a unique game whose physiologic demands are unlike any other sport played. It requires significant stamina and fitness, as well as power and physical strength.
- The ball is advanced down the field by kicking or running with it, but passed between teammates only by way of backward or lateral tosses.
- No blocking is permitted for the runner with the ball.
- Once a tackle is made, or forward progress is stopped, a **ruck** or **maul** is formed. These occur when two or more defenders challenge for the ball when it is on the ground (ruck) or while it is still being carried (maul).
- An infringement of the rules requires restart through a free kick or penalty kick or through the use of a scrummage or **scrum.**
  - The scrum is exclusive to the sport of rugby, and although it is one of the more dangerous facets of the game, it is a key method of securing ball possession.
  - The scrum consists of the eight forward players usually arranged in a "3-2-3" formation with the players "bound" together by shoulders, hands, and arms. These eight players will engage the heads and shoulders of the opposition's front forwards. The forwards then try to "hook" the ball with their feet and propel it backward through their legs to their teammates.
  - During the scrum, there is an elevated risk of cervical spine injury secondary to the great amount of forward, vertical, and shear forces generated, nearly 8000 newtons, which may greatly exceed the force the cervical spine can withstand.
- The **tackle** in rugby accounts for nearly 50% of all injuries. It is technically different from the tackle in gridiron football and quite dissimilar to the tackle in soccer.
- The **line-out** restarts the game after the ball goes out-of-bounds. Two parallel lines of players are perpendicular to the sideline and players jump (are boosted by a teammate) to contest the ball in midair. This element of the game can also represent a source for significant injury, especially to those players boosted into the air.

Equipment

- The rugby uniform consists of a jersey, shorts, socks, and cleats. The cleats may not have toe studs, and studs cannot exceed 18 mm.
- Players may wear mouth guards, mitts or gloves, shoulder pads, headgear, and chest pads sanctioned by the International Rugby Board (IRB).

POSITIONS

Players' positions are represented by the number they wear on their jersey, and this will correlate with their role on the pitch (unless they are substituted for another player).

Forwards

- These players are responsible for gaining and maintaining possession of the ball.
- They are the integral component players of the scrum and line-out.
- Although traditionally featured for their strength, forwards are now becoming increasingly athletic.
- Subdivisions of this position include:
  - **Loose head prop and tight head prop:** These two front row positions support the hooker between them and attempt to push forward during the scrum.
  - **Hooker:** This is the middle player of the front row whose job it is to hook the ball backward with his or her feet during the scrum.
  - **Lock:** Locks are second row forwards who bind into the scrum with their heads interlocked and wedged between the thighs of the props and hooker.

- **Flanker:** Also known as "wing-forward" or "loose-forward," these players on the outside of the third row attempt to push forward during scrums as well as break away from the pack if they are needed to make defensive tackles.
- **Eight man:** The middle player of the third row whose job it is to push during the scrum as well as be quick enough to make defensive stops if necessary.

Backs

- These quick and agile players are responsible for advancing the ball offensively in order to score points.
- Backs are also required to ruck, maul, and tackle as necessary.
- Subdivisions of this position include:
  - **Scrumhalf:** This player is the link between the forwards and the backs. Their duties are numerous, including putting the ball into the scrum, redistributing the ball from scrums, rucks, mauls, or line-outs and, most important, directing the offensive attack.
  - **Flyhalf:** This player is extremely adept at ball-handling and kicking, and is often required to make tactical decisions.
  - **Wing:** These players are usually extremely quick and are responsible for scoring tries, chasing down kicks, and defending.
  - **Inside center and outside center:** Along with wings and flyhalfs, these integral players are key members of the offense. They are also important for making tackles on defense.
  - **Fullback:** Thought of as "the sweeper of the rugby squad." They need to have talented offensive attacking skills as well as excellent defensive prowess.

INJURIES IN RUGBY

Overview

- Because of the unique demands of the game, injuries in rugby can occur to virtually every area of the body.
- Paradoxically, however, rugby injuries occur with an incidence closer to soccer, rather than typical collision sports like gridiron football.
- Studies that have defined injuries in terms of missed match play have reported an injury incidence of 26.8 to 60.3 per 1000 playing hours.
- Typically, injury rates for junior league and/or youth league rugby occur with less frequency than adult clubs or professional league.
- Further, women's injury rates occur at a frequency that is approximated to be about 40% of the men's injury rates.
- However, it should be noted that injuries to the anterior cruciate ligaments of the knee occur with higher incidence in women than in men.
- Injury rates are similar between backs and forwards.
- However, location and etiology of these injuries do vary according to position.
- Because of the formation of the scrum, forwards incur more head and neck injuries compared with their teammates who are backs.
- Interestingly, the rugby tackle accounts for more than half of all serious injuries.

Head and Neck Injuries

- Concussions occur at an incidence of 38 per 1000 game hours at the semiprofessional level, and account for approximately 25% of pediatric head and neck injuries.
- Cervical spine injuries, including facet fractures, disc injuries, and nerve root compressions, occur at a rate of approximately 6.5 per 100,000 participants per year.
- Traditionally, these injuries were suffered during the scrum, with the hooker being at highest risk. Probably because of recent rule changes made intending to "de-power" the scrum by controlling the engagement, the highest threat for cervical spine injuries now occurs during tackling.
- Front row forwards are at higher risk for cervical spine injury as a result of axial loading, particularly if the neck is in a flexed position during the set scrum.
- Because there are no set stoppages during the course of game play (other than halftime), it is difficult to adequately assess from the sideline a player who suffers a head injury.
- The International Rugby Board deems that players who have been concussed must "stand down" from games and training for a minimum period of 3 weeks. This may cause players to under-report head injuries.

Fractures and Dislocations

- Fractures occur in the upper extremity with greater incidence than in the lower extremity. The most common fractures occurring in the wrist, hand, and fingers.
- Other fracture sites include: radius, ulna, metacarpal ("boxer's fracture"), phalangeal, clavicle, nasal, and facial bones.
- Phalangeal and metacarpal fractures occur most commonly during tackling.
- Sideline management of fractures usually requires stabilization until definitive care can be obtained.
- Reduction of dislocations is an essential skill for the sideline physician in rugby.
- Dislocations usually occur in the glenohumeral joint or proximal interphalangeal (PIP) joint of the hand, but have been reported in the hip, elbow, ankle, and jaw.

Soft Tissue Injuries

- These account for more than 90% of all acute injuries in rugby.
- Strains and sprains represent approximately 32% of all injuries in rugby, with the preponderance of injuries occurring in the lower limb (thighs, knees, and ankles).
- Lateral ankle ligament and medial collateral ligament of the knee are the most common ligamentous injuries.
- Although medial collateral ligament (MCL) injuries outnumber anterior cruciate ligament (ACL) injuries in incidence, duration of time missed from sport is much higher after an ACL tear compared with any other knee ligament.
- Upper extremity sprains include acromioclavicular (AC) joint (most common), sternoclavicular joint, flexor/extensor tendons of the finger, and the ulnar collateral ligament of the thumb, elbow hyperextension, and wrist sprains (fall on an outstretched hand [FOOSH] injuries).
- Injuries to the foot include hematomas, turf toe, and Lisfranc sprains.

Other Injuries

- Lacerations occur commonly to the head and face, with an incidence of 0.83 per 1000 player hours.
- Because of rugby's constraints regarding substitutions during game play, a sideline physician must be keenly adept at approximating, closing, and protecting lacerations. It is not uncommon for players who suffer lacerations during a game to be efficiently treated and returned to play during the same match.
- Dental and eye injuries do occur and the sideline physician must be prepared with the necessary tools for flushing out and protecting eyes or transporting teeth.
- Chest/abdominal trauma may also occur, which requires awareness of the presenting signs/symptoms of such conditions as pneumothorax, pneumomediastinum, splenic and liver laceration, cardiac contusion, and genitourinary trauma.

INJURY PREVENTION

- It is a fallacy that rugby is equivalent to American gridiron football "without the pads."

- There have been several organizational rule changes that have helped decrease the incidence of cervical spine injury.
- There is unequivocal evidence to show that mouth guards reduce the incidence of dental injury; however, only approximately 60% of professional and 80% of youths wear them.
- Other "protective" equipment often worn includes soft helmets called "scrumcaps" and soft shoulder pads known as "shock tops." However, there is no convincing evidence that either of these prevent significant injury.
- Studies have shown that padded headgear tended to prevent damage to the scalp and ears, but do not lower the risk of concussion.
- One study has shown that support sleeves may reduce risk of sprains and strains, but other protective garments (shin guards, taping) showed no evidence of protective effect.

SUMMARY

- Rugby union is a high-speed, physically demanding contact/collision sport played throughout the world by all ages and abilities and both genders.
- Its unique features, like the set-scrum, are predictors for the types of injuries that are encountered by the sideline physician.
- Its rules regarding substitution for injured players require not only sharp diagnostic skills, but also efficacious treatment, so players may return to the game in progress.
- Much research is still being conducted regarding injury prevention, and hopefully these measures may be implemented with great success in the future.

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Cheerleading

Amy Elizabeth Valasek and Teri Metcalf McCambridge

GENERAL INFORMATION

- History
  - Cheerleading originated at a Minnesota University football game on November 2, 1898.
  - Over the next century cheerleaders functioned primarily in a supportive role at athletic competitions.
  - Since the 1980s, cheerleading has evolved into an activity demanding high levels of dance, tumbling, athleticism, and acrobatics and has become a varsity sport at some National Collegiate Athletic Association (NCAA) Division 1 schools.
- Modern forms of cheerleading include:
  - Sideline spirit raising
  - Half-time entertainment
  - Competition (All-Star Cheerleading):
    - Involves a 2-day competition.
    - The first day counts for one-third of the overall score and the second day for two-thirds.
    - The cheerleading routine is 2 minutes and 30 seconds in duration.
    - Team size varies from 5 to 35-plus males/females.
    - Teams are grouped by age, size, and ability level.
- Participation:
  - In 2002, there were an estimated 3.5 million cheerleading participants 6 years of age or older.
  - Also in 2002, there were an estimated 450,000 high school and collegiate cheerleaders.
- The American Association of Cheerleading Coaches and Administrators categorizes cheerleading as an "athletic activity." However, some high schools and Division I colleges recently made cheerleading a varsity sport.

SAFETY RULES

- The American Association of Cheerleading Coaches and Administrators (AACCA) established rules for high school, college, and all-star cheerleading teams (see www.aacca.org).
- The National Federal of State High School Associations (NFHS) publishes rules and regulations for high school cheerleading annually (see www.nfhs.org).

GLOSSARY OF TERMS

Base: A person who is in direct contact with the performing surface and supports another person's weight.

Top (flyer): A person who is supported by another individual above the performing surface or who has been tossed into the air by another person.

Middle: A person who is being supported by a base while also supporting a top person.

Spotter: A person who is responsible for assisting or catching the top person in a partner stunt or pyramid. This person cannot be in a position of providing primary support for a top person but must be in a position to protect the top person coming off of a stunt or pyramid.

Toss: A movement by one or a group of participants that propels a person into the air (i.e., free of contact with the performing surface).

Pyramid: A skill in which a top person is being supported by a middle and base layer person.

Flip: When a person is airborne while the feet pass over the head.

Dive Roll: A forward roll wherein the performer is airborne prior to the beginning of a forward roll.

Cradle: Dismount from a stunt/pyramid/toss in which the top person lands in a face-up, semipiked position.

Catching types: Bear Hug: one spotter is in front and slightly to the side. Cradle: three spotters (two on the side and one behind).

Helicopter toss: A stunt in which the top person is tossed into the air (with the body parallel to the ground) and completes a 360-degree horizontal rotation (like the blades of a helicopter).

Elevator toss: A stunt in which the top person loads in to an elevator-loading position and is then tossed into the air.

Rewind: Skill in which the top person starts with *both feet* on the ground, is tossed into the air, and performs a backward or side rotation into a stunt or loading position. Flips are limited to one rotation and twists are not permitted.

INJURY DATA

- The Consumer Product and Safety Commission in the Twenty-Third Annual Safety Report estimated 27,732 emergency room visits in 2004, ranking cheerleading as a sporting activity with the 11th highest overall injury rate (Box 86-1).
  - High school and college cheerleading combined were second overall, falling only behind football in the number of fatal, catastrophic, and serious injuries.
  - Catastrophic injuries have increased since cheerleading has incorporated gymnastic-like stunts (Fig. 86-1). In 1982-1983 there was one reported female catastrophic injury, whereas, an average of 6.1 catastrophic injuries have been reported yearly since 1993.
  - The National Center for Catastrophic Sports Injuries Research reported that cheerleading accounted for more than 50% of catastrophic injuries.

BOX 86-1 *Common Risk Factors Leading to Injury*

- Lack of conditioning
- Lack of strength training
- Lack of experience
- Insufficient supervision
- Difficult stunts
- Inappropriate surfaces
- Inappropriate shoes
- Poor nutrition

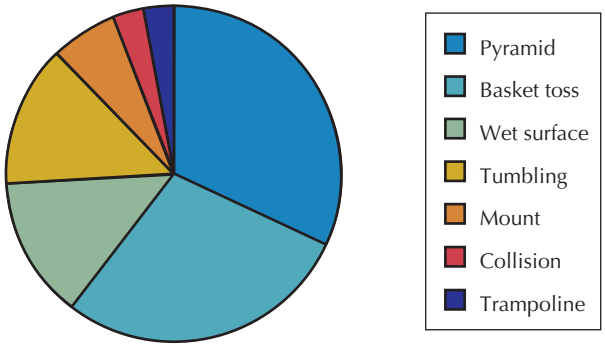

Figure 86-1 Catastrophic Cheerleading Injuries Reported 1982 to 2002 in High School and College. *(Reprinted from Boden BP, Tacchetti R, et al: Catastrophic cheerleading injuries. Am J Sport Med 31:881-888, 2003.)*

- Retrospective analysis of cheerleading-related injuries in children noted a greater number of injuries per 1000 participants sustained in the 12- to 17-year-old group compared with 6- to 11-year-old group, reflecting the greater level of performance difficulty.
  - Studies have demonstrated that the 12- to 18-year-olds are more likely to injure the lower extremity than the 6- to 11-year-olds.
  - Studies have shown more days were lost per injury in cheerleading when compared with other sports. Cheerleaders average 28.8 days lost in recovery from injury.
- The NCAA Injury Surveillance System tracks 16 intercollegiate sports, but does not include cheerleading in its survey.
  - There is currently no central tracking system for cheerleading-related injuries.
  - Cheerleading injuries occur throughout the year, with two peaks between September and November and then again between December to February.
  - These injury peaks occur during the football and basketball seasons, respectfully.

CHEERLEADING EVENTS

- Low-risk events
  - Chants and spirit yelling
  - Dance routines
  - Jumps and leaps
- Moderate-risk events
  - Tumbling
- High-risk events
  - Mounts and dismounts
    - Rules to improve safety: Spotters must be present for each person extended above shoulder level. Cradle dismounts require three bases.
  - Basket tosses
    - Rules to improve safety: Limitation to four throwers from the ground level. One thrower remains behind the flyer during the toss. Flyer should have specific training to maintain vertical alignment and head control in horizontal alignment with the body. Flips are prohibited.
  - Pyramids
    - Rules to improve safety: Height restrictions are limited to two levels in high school and 2½ body lengths in college. Pyramid height is measured by body lengths as follows: chairs, thigh stands, and shoulder straddles are 1½ body lengths; shoulder stands are 2 body lengths; extended stunts (e.g., extension, liberty, etc.) are 2½ body lengths. The top cheerleaders are required to be supported by one or more people (base) who are in direct weight-bearing contact with the performance surface. In high school, spotters are required for each person extended above shoulder level. In college, there must be at least two spotters designated for each person who is above "two persons high." One of the spotters must be in the back and the other must be at the side or in front of the pyramid.
  - Mini-trampoline or springboard
    - Use has been prohibited since the late 1980s.

INJURY TYPES

- Catastrophic injuries (see Fig. 86-1, Box 86-1)
  - Most commonly occur during a pyramid or basket toss
  - Closed head injury: subdural versus epidural versus intraparenchymal hemorrhage
  - Skull fracture
  - Vertebral fracture or subluxation
  - Spinal cord injury: complete or incomplete transaction
  - Spinal cord contusion
- Acute injuries
  - Most frequent site: ligamentous ankle injury
  - Concussion
  - Neck strain/sprain
  - Discogenic disease
  - Upper extremity injuries
    - Shoulder dislocations
    - Shoulder separations
    - Wrist and forearm fractures
  - Finger fractures
  - Lower extremity injuries
    - Ankle sprains, fractures
    - Knee injuries: ligament injury: anterior cruciate ligament (ACL), medial collateral ligament (MCL), meniscal injury, patellar dislocation
  - Contusions
  - Lacerations
- Chronic/overuse injuries
  - Most common: rotator cuff tendonitis and shoulder instability
  - Upper extremity
    - Rotator cuff and biceps tendonitis.
    - Triangular fibrocartilage complex tears
    - Distal radial physeal injury
    - Ulnar impaction syndrome
    - Dorsal wrist ganglion
    - Dorsal wrist capsulitis
  - Low back
    - Spondylosis and spondylolisthesis
  - Knee
    - Patellofemoral pain syndrome
    - Patellar and quadriceps tendonitis
    - Iliotibial band syndrome
  - Stress fractures
    - Recent study by Loud and colleagues in *Pediatrics* demonstrated cheerleading and gymnastics as independent risk factors for the development of stress fractures.
    - Atypical locations of stress fractures have been reported in gymnasts in the medial clavicle and forearm and may be present in cheerleaders training at a high level.
  - Foot
    - Plantar fasciitis

MEDICAL CONCERNS

- Dietary restriction/body image
  - Screen for the female athlete triad (see Chapter 10, The Female Athlete).
  - Recent survey results demonstrated body dissatisfaction rate of 73% in Caucasian girls and 50% in African-American girls among high school cheerleaders.
  - Discuss with coaching staff the importance of avoidance of weigh-ins or criticisms about weight or body shape.
- Fitness status of collegiate cheerleaders
  - Mean body fat level was 15.5% for females and 16.4% for males.
  - $\dot{V}O_2$ max was 48.8 ± 6.6 mL/kg per minute for men and 40.7 ± 5.8 mL/kg per minute for women.
  - Other variables tested included strength, flexibility, and aerobic fitness. The test results placed the cheerleaders well above the average fitness level for their age-matched controls and equivalent to other well-trained college-level athletes.
- Solid organ injury
  - Case report of a splenic rupture in a flyer in cheerleading after being caught.

INJURY PREVENTION

- Location of practice (Box 86-2)
  - Separate areas for stunts performed at games.
  - Sufficient space for number of individuals and stunt difficulty.

BOX 86-2 *Injury Prevention*

- Adequate and comprehensive conditioning program.
- Use and train spotters for difficult stunts.
- Restrict stunts in wet conditions.
- Implement floor mats for complex stunts such as pyramid, basket tosses, and tumbling.
- Do not perform difficult stunts with significant time constraints (e.g., during time-outs of a sporting event).

  - Proper ceiling height.
  - Appropriate floors, mats, lighting, and padding of objects.
  - Current surveys have demonstrated up to 10% of cheerleading practices occur in the cafeteria or hallway at the school.
- Proper stunt progression
  - See AACCA manual for acceptable progressions.
- Proper coaching
  - CPR/safety training.
  - Development and rehearsal of an emergency procedure and plan.
  - Completion of a coaching certification through the AACCA with recertification required every 4 years.
  - Coach with certification must be present at every practice or competition.
  - The AACCA began cheerleading safety certification in 1987.
  - Coaches should be trained in gymnastics and partner stunting and have an understanding of proper spotting techniques, conditioning techniques, and stunt progressions.

PERSONAL SAFETY

- **Proper attire** includes snug clothing, proper footwear, no hard hairpieces, no dangling jewelry or necklaces, and no body piercing on the face, mouth, or lips.
- **Game day guidelines:** No stunting while ball is in play; ensure an appropriately sized safety zone and surfaces free of water.
- **Athlete preparedness:** Proper conditioning, athletes matched by size and skills, and formal education on landing techniques (including practicing falling).
- **Preparticipation physical examination prior to onset of participation.**
- Skill restrictions based on age or ability level. See www.aacca.org to download a copy of the most recent guidelines.

CONDITIONING PROGRAMS

- Ankle
  - Strengthening (dorsiflexors and peroneals)
  - Gastrocnemius and soleus stretching
  - Proprioception training (single leg stance with eyes closed and ball toss while standing on one foot).
- Knee
  - Quadriceps strengthening (leg extension, leg press, side steps on block, wall squats, and split-leg pike jumps)
  - Calf strengthening (heel raises)
  - Hamstring stretching
- Wrist
  - Forearm strengthening (wrist curls and extensions)
  - Wrist stretching
- Upper extremity
  - Rotator cuff strengthening
  - Wall handstand push-ups or incline push-ups
  - Pull-ups and triceps dips
- Trunk
  - Side planks
  - V-ups
  - Diagonal trunk curls

GOVERNING BODIES

- American Association of Cheerleading Coaches and Administrators (AACCA)
- National Cheerleading Association (NCA)
- Universal Cheerleading Association
- U.S. Cheerleader Association
- International Cheerleading Foundation
- United Spirit Association

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Appendices

APPENDIX A: TEAM PHYSICIAN CONSENSUS STATEMENT

Team Physician Definition

The team physician must have an unrestricted medical license and be an M.D. or D.O. who is responsible for treating and coordinating the medical care of athletic team members. The principal responsibility of the team physician is to provide for the well-being of individual athletes—enabling each to realize his/her full potential. The team physician should possess special proficiency in the care of musculoskeletal injuries and medical conditions encountered in sports. The team physician also must actively integrate medical expertise with other healthcare providers, including medical specialists, athletic trainers, and allied health professionals. The team physician must ultimately assume responsibility within the team structure for making medical decisions that affect the athlete's safe participation.

Qualifications of a Team Physician

The primary concern of the team physician is to provide the best medical care for athletes at all levels of participation. To this end, the following qualifications are necessary for all team physicians:

- Have an M.D. or D.O. in good standing, with an unrestricted license to practice medicine
- Possess a fundamental knowledge of emergency care regarding sporting events
- Be trained in CPR
- Have a working knowledge of trauma, musculoskeletal injuries, and medical conditions affecting the athlete

In addition, it is desirable for team physicians to have clinical training/experience and administrative skills in some or all of the following:

- Specialty Board certification
- Continuing medical education in sports medicine
- Formal training in sports medicine (fellowship training, board recognized subspecialty in sports medicine [formerly known as a certificate of added qualification in sports medicine])
- Additional training in sports medicine
- Fifty percent or more of practice involving sports medicine
- Membership and participation in a sports medicine society
- Involvement in teaching, research, and publications relating to sports medicine
- Training in advanced cardiac life support
- Knowledge of medical/legal, disability, and workers' compensation issues
- Media skills training

Duties of a Team Physician

The team physician must be willing to commit the necessary time and effort to provide care to the athlete and team. In addition, the team physician must develop and maintain a current, appropriate knowledge base of the sport(s) for which he/she is accepting responsibility.

The duties for which the team physician has ultimate responsibility include the following:

Medical management of the athlete

- ☐ Coordinate pre-participation screening, examination, and evaluation
- ☐ Manage injuries on the field
- ☐ Provide for medical management of injury and illness
- ☐ Coordinate rehabilitation and return to participation
- ☐ Provide for proper preparations for safe return to participation after an illness or injury
- ☐ Integrate medical expertise with other health care providers, including medical specialists, athletic trainers and allied health professionals
- ☐ Provide for appropriate education and counseling regarding nutrition, strength and conditioning, ergogenic aids, substance abuse, and other medical problems that could affect the athlete
- ☐ Provide for proper documentation and medical record keeping

Administrative and logistical duties

- ☐ Establish and define the relationships of all involved parties
- ☐ Educate athletes, parents, administrators, coaches, and other necessary parties of concerns regarding the athletes
- ☐ Develop a chain of command
- ☐ Plan and train for emergencies during competition and practice
- ☐ Address equipment and supply issues
- ☐ Provide for proper event coverage
- ☐ Assess environmental concerns and playing conditions

Education of a Team Physician

Ongoing education pertinent to the team physician is essential. Currently, there are several state, regional, and national stand-alone courses for team physician education. There are also many other resources available. Information regarding team physician specific educational opportunities can be obtained from the organizations listed below:

American Academy of Family Physicians (AAFP)
11400 Tomahawk Creek Pkwy.
Leawood, KS 66211-2672
1-800-274-2237

American Academy of Orthopaedic Surgeons (AAOS)
6300 N. River Rd.
Rosemont, IL 60018
1-800-346-AAOS

American College of Sports Medicine (ACSM) (AOASM)
401 W. Michigan St.
Indianapolis, IN 46202-3233
(317) 637-9200

American Medical Society for Sports Medicine (AMSSM)
11639 Earnshaw
Overland Park, KS 66210
(943) 327-1415

American Orthopaedic Society for Sports Medicine (AOSSM)
6300 N. River Rd. Suite 200
Rosemont, IL 60018
(847) 292-4900

American Osteopathic Academy of Sports Medicine
7611 Elmwood Ave., Suite 201
Middleton, WI 53562
(608) 831-4400

Team physician education is also available from other sources such as: sport-specific (e.g., National Football League Team Physician's Society) or level-specific (e.g., United States Olympic Committee) meetings; National Governing Bodies' (NGB) meetings; state and/or county medical societies meetings; professional journals; and other relevant electronic media (Web sites, CD-ROMs).

Conclusion

This Consensus Statement establishes a definition of the team physician, and outlines a team physician's qualifications, duties, and responsibilities. It also contains strategies for the continuing education of team physicians. Ultimately, this statement provides guidelines that best serve the health care needs of athletes and teams.

APPENDIX B: SIDELINE PREPAREDNESS FOR THE TEAM PHYSICIAN: A CONSENSUS STATEMENT

Sideline Preparedness Statement Definition

Sideline preparedness is the identification of and planning for medical services to promote the safety of the athlete, to limit injury, and to provide medical care at the site of practice or competition.

Goal

The safety and on-site medical care of the athlete is the goal of sideline preparedness. To accomplish this goal, the team physician should be actively involved in developing an integrated medical system that includes:

- Pre-season planning
- Game-day planning
- Post-season evaluation

Pre-Season Planning

Pre-season planning promotes safety and minimizes problems associated with athletic participation at the site of practice or competition. The team physician should coordinate:

- Development of policy to address pre-season planning and pre-participation evaluation of athletes
- Participation of the administration and other key personnel in medical issues
- Implementation strategies

Medical Protocol Development

It is essential that:

- Prospective athletes complete a pre-participation evaluation
- In addition, it is desirable that:
- The pre-participation evaluation be performed by an M.D. or D.O. in good standing with an unrestricted license to practice medicine
- A comprehensive pre-participation evaluation form be used (e.g., the form found in the current edition of Pre-participation Physical Evaluation)
- The team physician has access to all pre-participation evaluation forms
- The team physician review all pre-participation evaluation forms and determine eligibility of the athlete to participate
- Timely pre-participation evaluations be performed to permit the identification and treatment of injuries and medical conditions

Administrative Protocol Development

It is essential for the team physician to coordinate:

- Development of a chain of command that establishes and defines the responsibilities of all parties involved
- Establishment of an emergency response plan for practice and competition
- Compliance with Occupational Safety and Health Administration (OSHA) standards relevant to the medical care of the athlete
- Establishment of a policy to assess environmental concerns and playing conditions for modification or suspension of practice or competition
- Compliance with all local, state and Federal regulations regarding storing and dispensing pharmaceuticals
- Establishment of a plan to provide for proper documentation and medical record keeping

In addition, it is desirable for the team physician to coordinate:

- Regular rehearsal of the emergency response plan
- Establishment of a network with other health care providers, including medical specialists, athletic trainers and allied health professionals
- Establishment of a policy that includes the team physician in the dissemination of any information regarding the athlete's health
- Preparation of a letter of understanding between the team physician and the administration that defines the obligations and responsibilities of the team physician

Game-day Planning

Game-day planning optimizes medical care for injured or ill athletes. The team physician should coordinate:

- Game-day medical operations
- Game-day administrative medical policies
- Preparation of the sideline "medical bag" and sideline medical supplies

Medical Protocol

It is essential for the team physician to coordinate:

- Determination of final clearance status of injured or ill athletes on game-day prior to competition
- Assessment and management of game-day injuries and medical problems
- Determination of athletes' same-game return to participation after injury or illness
- Follow-up care and instructions for athletes who require treatment during or after competition
- Notifying the appropriate parties about an athlete's injury or illness
- Close observation of the game by the medical team from an appropriate location
- Provision for proper documentation and medical record keeping

In addition, it is desirable for the team physician to coordinate:

- Monitoring of equipment safety and fit
- Monitoring of post-game referral care of injured or ill athletes

Administrative Protocol

It is essential for the team physician to coordinate:

- Assessment of environmental concerns and playing conditions
- Presence of medical personnel at the competition site with sufficient time for all pre-game preparations
- Plan with the medical staff of the opposing team for medical care of the athletes
- Introductions of the medical team to game officials
- Review of the emergency medical response plan
- Checking and confirmation of communication equipment
- Identification of examination and treatment sites

In addition, it is desirable for the team physician to coordinate:

- Arrangements for the medical staff to have convenient access to the competition site
- A post-game review and make necessary modifications of medical and administrative protocols

On-Site Medical Supplies

The team physician should have a game-day sideline "medical bag" and sideline medical supplies. The following is a list of "medical bag" items and medical supplies for contact/collision and high-risk sports:

It is highly desirable for the "medical bag" to include:

GENERAL

- Alcohol swabs and povidone iodine swabs
- Bandage scissors
- Bandages, sterile/non-sterile, band-aids
- D-50%-W
- Disinfectant
- Gloves, sterile/non-sterile
- Large bore angiocath for tension pneumothorax (14–16 gauge)
- Local anesthetic/syringes/needles
- Paper
- Pen
- Sharps box and red bag
- Suture set/steri-strips
- Wound irrigation materials (e.g,. sterile normal saline, 10–50 cc syringe)

CARDIOPULMONARY

- Airway
- Blood pressure cuff
- Cricothyrotomy kit
- Epinephrine 1:1000 in a prepacked unit
- Mouth-to-mouth mask
- Short-acting beta agonist inhaler
- Stethoscope

HEAD AND NECK/NEUROLOGIC

- Dental kit (e.g., cyanoacrylate, Hank's solution)
- Eye kit (e.g., blue light, fluorescein stain strips, eye patch pads, cotton tip applicators, ocular anesthetic and antibiotics, contact remover, mirror)
- Flashlight
- Pin or other sharp object for sensory testing
- Reflex hammer

It is highly desirable for sideline medical supplies to include:

GENERAL

- Access to a telephone
- Extremity splints
- Ice
- Oral fluid replacement
- Plastic bag
- Sling

HEAD AND NECK/NEUROLOGIC

- Face mask removal tool (for sports with helmets)
- Semi-rigid cervical collar
- Spine board and attachments

In addition, it is desirable for the "medical bag" to include:

GENERAL

- Benzoin
- Blister care materials
- Contact lens case and solution
- 30% ferric subsulfate solution (e.g., Monsel's for cauterizing abrasions and cuts)
- Injury and illness care instruction sheets for the patient
- List of emergency phone numbers
- Nail clippers
- Nasal packing material
- Oto-ophthalmoscope
- Paper bags for treatment of hyperventilation
- Prescription pad
- Razor and shaving cream
- Rectal thermometer
- Scalpel
- Skin lubricant
- Skin staple applicator
- Small mirror
- Supplemental oral and parenteral
- Tongue depressors
- Topical antibiotics

CARDIOPULMONARY

- Advanced Cardiac Life Support (ACLS) drugs and equipment
- IV fluids and administration set
- Tourniquet

In addition, it is desirable for sideline medical supplies to include:

GENERAL

- Blanket
- Crutches
- Mouth guards
- Sling psychrometer and temperature /humidity activity risk chart
- Tape cutter

CARDIOPULMONARY

- Automated external defibrillator

HEAD AND NECK/NEUROLOGIC

- A sideline concussion assessment protocol

There are many different sports, levels of competition, and available medical resources that must all be considered when determining the on-site medical bag and sideline medical supplies.

Post-season Evaluation

Post-season evaluation of sideline coverage optimizes the medical care of injured or ill athletes and promotes continued improvement of medical services for future seasons. The team physician should coordinate:

- Summarization of injuries and illnesses that occurred during the season
- The improvement of the medical and administrative protocols
- Implementation of strategies to improve sideline preparedness

Medical Protocol

It is essential for the team physician to coordinate:

- A post-season meeting with appropriate team personnel and administration to review the previous season
- Identification of athletes who require post-season care of injury or illness and encourage follow-up

In addition, it is desirable for the team physician to coordinate:

- Monitoring of the health status of the injured or ill athlete
- Post-season physicals
- An off-season conditioning program

Administrative Protocol

It is essential for the team physician to coordinate:

- Review and modification of current medical and administrative protocols

In addition, it is desirable for the team physician to coordinate:

- Compilation of injury and illness data

Conclusion

This Consensus Statement outlines the essential and desirable components of sideline preparedness for the team physician to promote the safety of the athlete, to limit injury, and to provide medical care at the site of practice or competition. This statement was developed by the collaboration of six major professional associations concerned about clinical sports medicine issues: American Academy of Family Physicians, American Academy of Orthopaedic Surgeons, American College of Sports Medicine, American Medial Society for Sports Medicine, American Orthopaedic Society for Sports Medicine, and the American Osteopathic Academy of Sports Medicine.

APPENDIX C: CONCUSSION (MILD TRAUMATIC BRAIN INJURY) AND THE TEAM PHYSICIAN: A CONSENSUS STATEMENT

Definition

Concussion or mild traumatic brain injury (MTBI) is a pathophysiological process affecting the brain induced by direct or indirect biomechanical forces.

Common features include:

- Rapid onset of usually short-lived neurological impairment which typically resolves spontaneously.
- Acute clinical symptoms that usually reflect a functional disturbance rather than structural injury.
- A range of clinical symptoms that may or may not involve loss of consciousness (LOC).
- Neuroimaging studies that are typically normal.

Goal

The goal is to assist the team physician in providing optimal medical care for the athlete with concussion. To accomplish this goal, the team physician should have knowledge of and be involved with:

- Epidemiology
- Pathophysiology
- Game-day evaluation and treatment
- Post game-day evaluation and treatment
- Diagnostic imaging
- Management principles
- Return-to-play
- Complications of concussion
- Prevention

Introduction

It is *essential* for the team physician to understand:

- The recognition and evaluation of the athlete with concussion.
- Management and treatment of the athlete with concussion be individualized.
- The factors involved in making return-to-play (RTP) decisions after injury should be based on clinical judgment.
- A game-day medical plan specific to concussion injuries be developed.
- The need for documentation.
- There is a paucity of well-designed studies of concussion and its natural history.

In addition, it is *desirable* for the team physician to:

- Coordinate a systematic approach for the treatment of the athlete with concussion.
- Identify risk factors and implement appropriate treatment.
- Understand the potential sequelae of concussive injuries.
- Understand prevention strategies.

Epidemiology

- Concussions occur commonly in helmeted and non-helmeted sports, and account for a significant number of time loss injuries.
- Published reports indicate concussion injuries occur at a rate of:
  - 0.14 - 3.66 injuries per 100 player seasons at the high school level, accounting for three to five percent of injuries in all sports
  - 0.5 - 3.0 injuries per 1,000 athlete exposures at the collegiate level.
- Self-report data suggests significantly higher incidence of concussion.
- Because of under recognition and/or under reporting, the incidence of concussion and its sequelae is unknown.

Pathophysiology

- Metabolic changes that occur in the animal model, and thought to occur in humans include:
  - Alterations in intracellular/extracellular glutamate, potassium and calcium
  - A relative decrease in cerebral blood flow in the setting of an increased requirement for glucose (i.e. increased glycolysis)
- This mismatch in the supply and demand of metabolism may potentially result in cell dysfunction and increase the vulnerability of the cell to a second insult.

Game Day Evaluation and Treatment

It is *essential* for the team physician to:

- Implement the game-day medical plan specific to concussion.
- Understand the indications for cervical spine immobilization and emergency transport.

On Field

- Evaluate the injured athlete on-the-field in a systematic fashion:
  - Assess for adequate airway, breathing, and circulation (ABC's)
  - Followed by focused neurological assessment emphasizing mental status, neurological deficit, and cervical spine status
  - Determine initial disposition (emergency transport vs. sideline evaluation)

Sideline

- Obtain a more detailed history and perform a more detailed physical examination.
  - Assess for cognitive, somatic, and affective signs and symptoms of acute concussion (see Table 1), with particular attention to retrograde amnesia (RGA), post-traumatic amnesia (PTA), and more than brief LOC (minutes, not seconds), because of their prognostic significance.
- Not leave the player unsupervised.
- Perform serial neurological assessments
- Determine disposition for symptomatic and non-symptomatic players, including post-injury follow-up (options include

return-to-play, home with observation, or transport to hospital).
- Provide post-event instructions to the athlete and others (e.g., regarding alcohol, medications, physical exertion and medical follow-up).

In addition, it is *desirable* for the team physician to:

On Field

- Have a plan to protect access to the injured player
- Have emergency medical personnel on-site
- Have medical supplies on-site for rescue, immobilization and transportation (see Sideline Preparedness for the Team Physician: A Consensus Statement, 2000).

Sideline

- Delineate the mechanism of injury.
- Perform a more detailed assessment of cognitive function (e.g., memory, calculations, attention span, concentration, speed of information processing).
- Coordinate the care and follow-up of the athlete with concussion.
- Discuss status of athlete with parents, caregivers, certified athletic trainers and coaching staff within disclosure regulations.

Selected Acute Signs and Symptoms of Concussion

COGNITIVE	SOMATIC	AFFECTIVE
Confusion	Headache	Emotional
Post-traumatic amnesia (PTA)	Fatigue	Liability
Retrograde amnesia (RGA)	Disequilibrium, dizziness	Irritability
Loss of consciousness (LOC)	Nausea/vomiting	
Disorientation	Visual disturbances (photophobia, blurry/double vision)	
Felling "in a fog," "zoned out"	Phonophobia	
Vacant stare		
Inability to focus		
Delayed verbal and motor responses		
Slurred/incoherent speech		
Excessive drowsiness		

Post Game-Day Evaluation and Treatment

It is *essential* for the team physician to:
- Obtain a comprehensive history of the current concussion, and any previous concussion.
- Perform a physical examination, including a detailed neurological/cognitive evaluation.
- Determine the need for further evaluation and consultation.
- Determine return-to-play status.

In addition, it is *desirable* for the team physician to:
- Coordinate the care and follow-up of the athlete.
- Understand the indications and limitations of neuropsychological testing.
- Post-injury neuropsychological test data are more useful if compared to the athlete's pre-injury baseline.
- It is unclear what type and content of test data are most valuable.
- It is only one component of the evaluation process.
- Educate the athlete and others about concussion.
- Discuss status of athlete with parents, caregivers, certified athletic trainers and coaching staff within disclosure regulations.

Diagnostic Imaging

It is *essential* for the team physician to understand:
- The limited value of plain skull radiographs.
- Indications of advanced imaging, such as CT or MRI, to assess associated injuries including intracranial bleed, cerebral edema, diffuse axonal injury, and/or skull fracture.
- Indications for the use of cervical imaging when cervical spine injury is suspected.

In addition, it is *desirable* for the team physician to:
- Review the results of the imaging studies and/or ancillary tests such as facial bone radiographs.

Management Principles

It is *essential* for the team physician to understand:
- Brief LOC (seconds, not minutes) is associated with specific early deficits, but does not predict the severity of injury; therefore classification systems or RTP guidelines based solely on brief LOC are not accurate.
- RGA, PTA, as well as the number and duration of additional signs and symptoms, are more accurate in predicting severity and outcome. RTP guidelines which address these issues are more useful.
- Duration of symptoms is a major factor in determining severity, therefore severity of injury should not be determined until all signs and symptoms have cleared.
- The treatment of and the RTP decision for the athlete with concussion must be individualized.

In addition, it is *desirable* for the team physician to:
- Coordinate a team for concussion management (e.g., physicians, certified athletic trainers, neuropsychologists, emergency response personnel).
- Discuss status of athlete with parents, caregivers, certified athletic trainers and coaching staff within disclosure regulations.

Return to Play (RTP) Decision

The RTP decision should be individualized, and not based on a rigid timeline. The team physician is ultimately responsible for the RTP decision. [See The Team Physician and Return-To-Play Issues Consensus Statement, 2002.]

It is *essential* for the team physician to understand:

Same Day RTP

- There is agreement that athletes with significant, persistent or worsening signs and symptoms (e.g., abnormal neurological examination, ongoing RGA or PTA, prolonged LOC) should not RTP.
- For other athletes with concussion, significant controversy exists for a same-day RTP decision and no conclusive evidence-based data are available. Areas of controversy include:
  - Returning an athlete with any symptoms to play.
  - Returning an athlete with fully resolved symptoms to play.
  - Certain symptoms, even if resolved, are contraindications to same-day RTP (e.g., any LOC, PTA, and RGA).
  - The duration and severity of symptoms are the determining factors of RTP.
- It is the safest course of action to hold an athlete out.

Post Game-Day RTP

- Determine the athlete is asymptomatic at-rest before resuming any exertional activity.
  - Amnesia may be permanent.
- Utilize progressive aerobic and resistance exercise challenge tests prior to full RTP.
- Consider factors which may affect RTP, including:
  - Severity of the current injury

- Previous concussions (number, severity, proximity)
- Significant injury in response to a minor blow
- Age (developing brain may react differently to trauma than mature brain)
- Sport
- Learning disabilities

- Understand contraindications for return to sport (e.g., abnormal neurological examination, signs or symptoms with exertion, significant abnormalities on cognitive testing or imaging studies).
- Controversy exists for post-game RTP decisions.

In addition, it is *desirable* for the team physician to:

Post Game-Day RTP

- Coordinate a team to implement progressive aerobic and resistance exercise challenge tests prior to full RTP.
- Recognize challenging cognitive effort may exacerbate symptoms of concussion and retard recovery.
- Discuss status of athlete with parents, caregivers, teachers, certified athletic trainers and coaching staff within disclosure regulations.
- Consider neuropsychological testing.

Complications of Concussion

It is *essential* for the team physician to:

- Understand cumulative concussions may increase risk for subsequent concussions.
- Determine when the athlete may RTP.

It is also *essential* for the team physician to understand other complications may occur, including:

- Convulsive motor phenomena
  - Tonic posturing or convulsive movements within seconds of the concussion
  - Dramatic, but usually benign
  - Require no management beyond on-field ABC's
  - No anticonvulant therapy required
- Post-traumatic seizures
  - Seizure occurs days to months after concussion
  - Does require seizure management and precautions
  - Usually requires anticonvulant therapy
- Post-concussion syndrome
  - Persistent post-concussion symptoms lasting months
  - Indicator of concussion severity
  - Precludes RTP while present
- Second impact syndrome
  - Occurs within minutes of concussion in athlete still symptomatic from prior brain injury
  - Prior brain injury can be earlier in same event
  - Vascular engorgement leads to massive increase in intracranial pressure and brain herniation
  - Usually with severe brain damage or death
  - May occur with associated small subdural hemotoma
  - Except for boxing, all cases in literature in adolescents (< 20 years old)

In addition, it is *desirable* for the team physician to:

- Coordinate assessment and treatment of complications
- Discuss status of athlete with parents, caregivers, certified athletic trainers and coaching staff within disclosure regulations.

Prevention

Concussions cannot be completely prevented.

It is *essential* for the team physician to understand:

- Helmet use decreases the incidence of skull fracture and major head trauma, but does not prevent, and may actually increase, the incidence of concussion.
- Improper use of the head and improper fit of helmet or protective equipment may increase the risk of concussion.
- There are rules to limit concussion (e.g., spearing, head-to-head contact, leading with the head).

In addition, it is *desirable* for the team physician to:

- During the pre-participation evaluation, obtain a concussion history.
- Discuss the enforcement of rules to limit concussion with coaching staff and officials prior to practice and competition.
- Discuss with players and coaches techniques which may increase the risk of concussion.
- Support the use of mouth guards to decrease the risk of dental and facial injury, although the protection they provide to concussion risk is unclear.
- Educate athletes, parents, and coaches on the importance of reporting symptoms of concussion to limit complications.
- Educate athletes, parents, and coaches regarding the escalation of violence in sports.

Expert Panel

Stanley A. Herring, M.D., Chair; Seattle, Washington; John A. Bergfeld, M.D.; Cleveland, Ohio; Arthur Boland, M.D.; Winchester, Massachusetts; Lori A. Boyajian-O'Neill, D.O.; Kansas City, Missouri; Robert C. Cantu, M.D.; Concord, Massachusetts; Elliott Hershman, M.D.; New York, New York; Peter Indelicato, M.D.; Gainesville, Florida; Rebecca Jaffe, M.D.; Wilmington, Delaware; W. Ben Kibler, M.D.; Lexington, Kentucky; Douglas B. McKeag, M.D.; Indianapolis, Indiana; Robert Pallay, M.D.; Hillsborough, New Jersey; Margot Putukian, M.D.; Princeton, New Jersey

This guideline for identification and planning for medical services should not be construed as including all proper methods of care and planning or excluding other acceptable methods of care and planning reasonably directed to obtain the same result. The ultimate judgment regarding specific measures to be taken by a physician or the sidelines of an athletic event must be made in light of all circumstances which might occur and the needs and resources particular to the locality or institution.

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