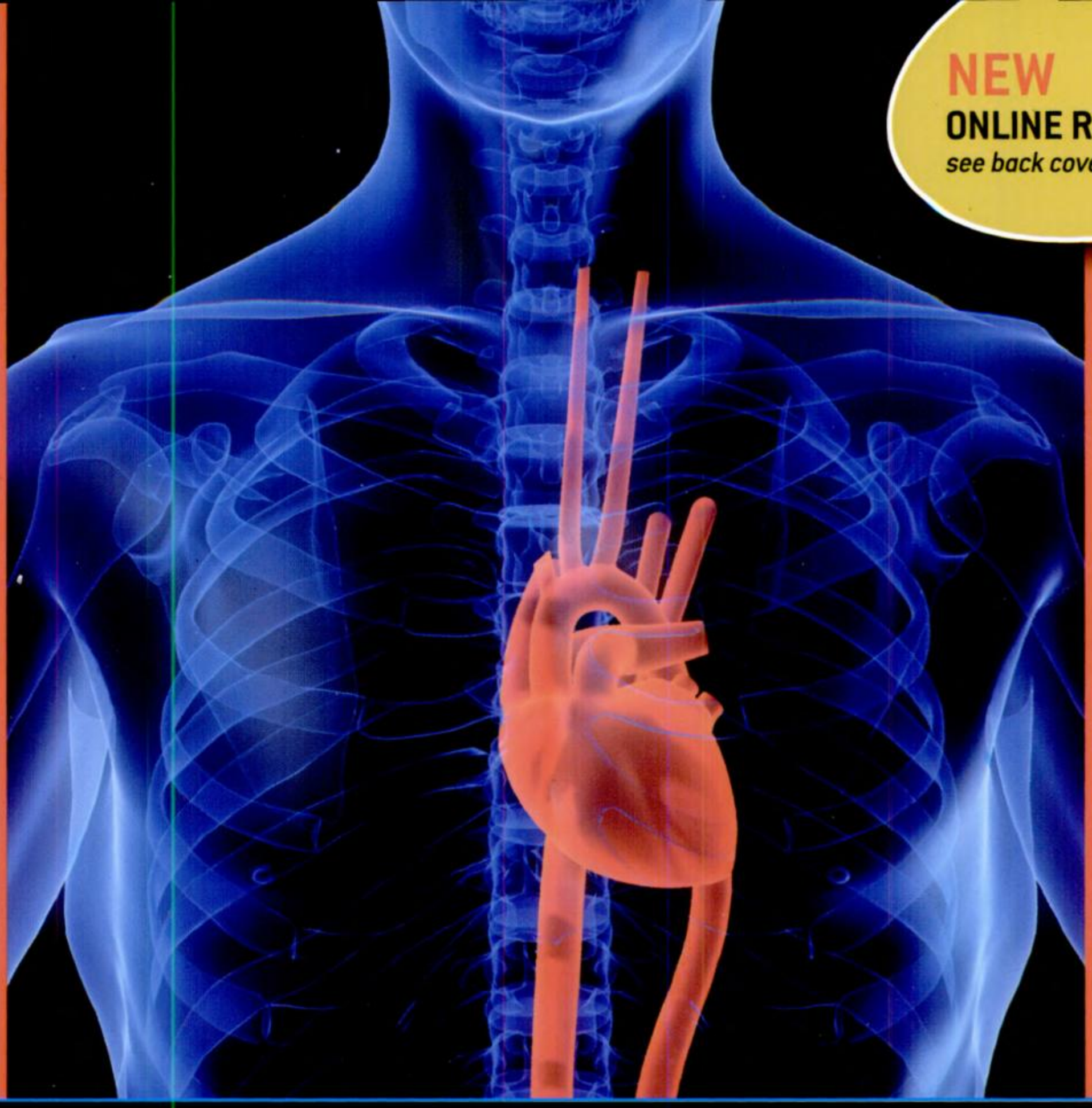


NEW
ONLINE RESOURCE!
see back cover for details

Fourth Edition

PHYSICAL EXAMINATION *of the* HEART *and* CIRCULATION

JOSEPH K. PERLOFF

People's Medical Publishing House
2 Enterprise Drive, Suite 509
Shelton, CT 06484
Tel: 203-402-0646
Fax: 203-402-0854
E-mail: info@pmph-usa.com

09 10 11 12/*PMPH*/9 8 7 6 5 4 3 2 1

ISBN 978-1-60795-023-3
Printed in China by People's Medical Publishing House
Copyeditor/typesetter: Newgen; Cover designer: Mary Mckeon

Sales and Distribution

Canada
McGraw-Hill Ryerson Education
Customer Care
300 Water St
Whitby, Ontario L1N 9B6
Canada
Tel: 1-800-565-5758
Fax: 1-800-463-5885
www.mcgrawhill.ca

Foreign Rights
John Scott & Company
International Publisher's Agency
P.O. Box 878
Kimberton, PA 19442
USA
Tel: 610-827-1640
Fax: 610-827-1671

Japan
United Publishers Services Limited
1-32-5 Higashi-Shinagawa
Shinagawa-ku, Tokyo 140-0002
Japan
Tel: 03-5479-7251
Fax: 03-5479-7307
Email: kakimoto@ups.co.jp

United Kingdom, Europe, Middle East, Africa
McGraw Hill Education
Shoppenhangers Road
Maidenhead
Berkshire, SL6 2QL
England
Tel: 44-0-1628-502500
Fax: 44-0-1628-635895
www.mcgraw-hill.co.uk

Singapore, Thailand, Philippines, Indonesia, Vietnam, Pacific Rim, Korea
McGraw-Hill Education
60 Tuas Basin Link
Singapore 638775
Tel: 65-6863-1580
Fax: 65-6862-3354
www.mcgraw-hill.com.sg

Australia, New Zealand
Elsevier Australia
Tower 1, 475 Victoria Avenue
Chatswood NSW 2067
Australia
Tel: 0-9422-8553
Fax: 0-9422-8562
www.elsevier.com.au

Brazil
Tecmedd Importadora e Distribuidora de Livros Ltda.
Avenida Maurilio Biagi 2850
City Ribeirao, Rebeirao, Preto SP
Brazil
CEP: 14021-000
Tel: 0800-992236
Fax: 16-3993-9000
Email: tecmedd@tecmedd.com.br

India, Bangladesh, Pakistan, Sri Lanka, Malaysia
CBS Publishers
4819/X1 Prahlad Street 24
Ansari Road, Darya, New Delhi-110002
India
Tel: 91-11-23266861/67
Fax: 91-11-23266818
Email:cbspubs@vsnl.com

People's Republic of China
PMPH
Bldg 3, 3rd District
Fangqunyuan, Fangzhuang
Beijing 100078
P.R. China
Tel: 8610-67653342
Fax: 8610-67691034
www.pmph.com

Notice: The authors and publisher have made every effort to ensure that the patient care recommended herein, including choice of drugs and drug dosages, is in accord with the accepted standard and practice at the time of publication. However, since research and regulation constantly change clinical standards, the reader is urged to check the product information sheet included in the package of each drug, which includes recommended doses, warnings, and contraindications. This is particularly important with new or infrequently used drugs. Any treatment regimen, particularly one involving medication, involves inherent risk that must be weighed on a case-by-case basis against the benefits anticipated. The reader is cautioned that the purpose of this book is to inform and enlighten; the information contained herein is not intended as, and should not be employed as, a substitute for individual diagnosis and treatment.

1 Introduction with a Brief Historical Survey

I hope to demonstrate to you that knowledge essential to the understanding of the patient's complaint can be made out by our unaided senses, and that artificial and mechanical aids can, in the nature of things, only be accessory aids.[1]

Sir James Mackenzie

The physical examination of the cardiovascular system includes the heart and circulation *per se*, the chest and abdomen, and the physical signs of coexisting noncardiac diseases. Examination of the heart and circulation includes (1) physical appearance (sometimes called inspection), (2) the arterial pulse, (3) the jugular venous pulse and peripheral veins, (4) the location and movements of the heart: percussion, palpation, and observation of the precordium, and (5) auscultation. Examination of the chest (thorax, lungs) and abdomen frequently reveals signs secondary to or associated with cardiac or vascular disorders. In infants, children, and young adults, diseases of the heart usually occur in patients who are otherwise normal. With advancing age, coexisting noncardiac diseases accrue and modify the cardiovascular physical signs.

Systematic assessment of each physical sign sets the stage for a synthesis of the information so derived. It is axiomatic that emphasis focuses on the relationship of the parts to the whole, a relationship that ideally results in a harmonious picture devoid of contradictions, not an assembly of loosely related observations. Maximum information should be extracted from each physical sign while relating information from one sign to that of another.

No organ system lends itself better to a close association between signs, structure, and function than the heart and circulation. Laboratory characterization of cardiovascular anatomy and physiology has validated these signs, which in turn permit remarkably precise pathophysiologic inferences. Excitement, satisfaction, and a feeling of confidence and security are experienced when accurate, practical information is assembled with the unaided senses apart from a stethoscope, a pocket flashlight, a sphygmomanometer, and an ophthalmoscope.

My purpose is to describe the physical signs and how they are best elicited. Mechanisms are dealt with chiefly when doing so serves this end. Emphasis is placed on the physical *examination* rather than on physical diagnosis.

The fabric of the cardiovascular physical examination is woven from threads of the past. Franciscus de le Boe Sylvius, at the University of Leiden, was one of the most celebrated clinical teachers of the seventeenth century and was the first champion of bedside

teaching. He would lead his students by the hand to the practice of medicine, "taking them every day to see patients in the public hospital, that they may hear the patients' symptoms and see their physical findings."[2] Two and a half centuries later, Harvey Cushing's biography of William Osler (Fig. 1–1) began with the dedication, "To medical students . . . lest it be forgotten who it was that made it possible for them to work at the bedside in the wards."[3]

Each of the five essential components of the cardiovascular physical examination is rooted in history. ***Physical appearance*** was the subject of the introductory paragraph of the chapter "Preliminary Examination of the Patient" in James Mackenzie's *Diseases of the Heart*[4] (Fig. 1–2):

> The attempt to appreciate the patient's condition should begin when first he presents himself before us. On his appearance in the consulting room, his bearing, his gait, the condition of his respiration, the color of his face, any nervous peculiarity in his manner of speech and behavior, and so forth, should be noted. If he is in bed, note the position he assumes, and any change in his color or respiration in response to such exertions as talking or turning over. By habit, one unconsciously notices these things.[4]

The ***arterial pulse*** served as the basis for an entire system of diagnostic medicine circa 2600 B.C. in China with *The Yellow Emperor's Book of Medicine.*[5] *Nei Ching Su Wen*

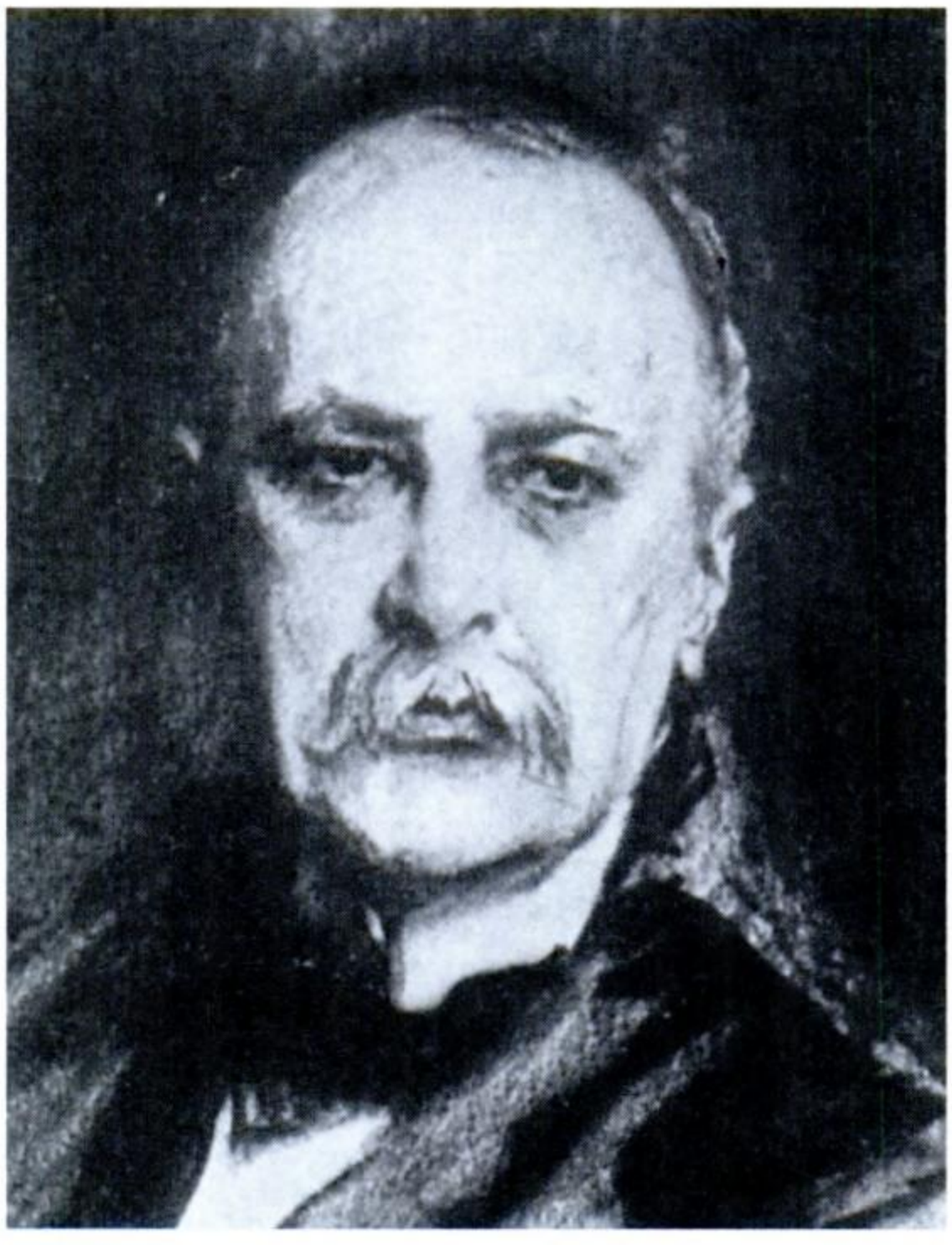


Figure 1–1 Sir William Osler (1849–1919). (Courtesy of the College of Physicians, Philadelphia.)

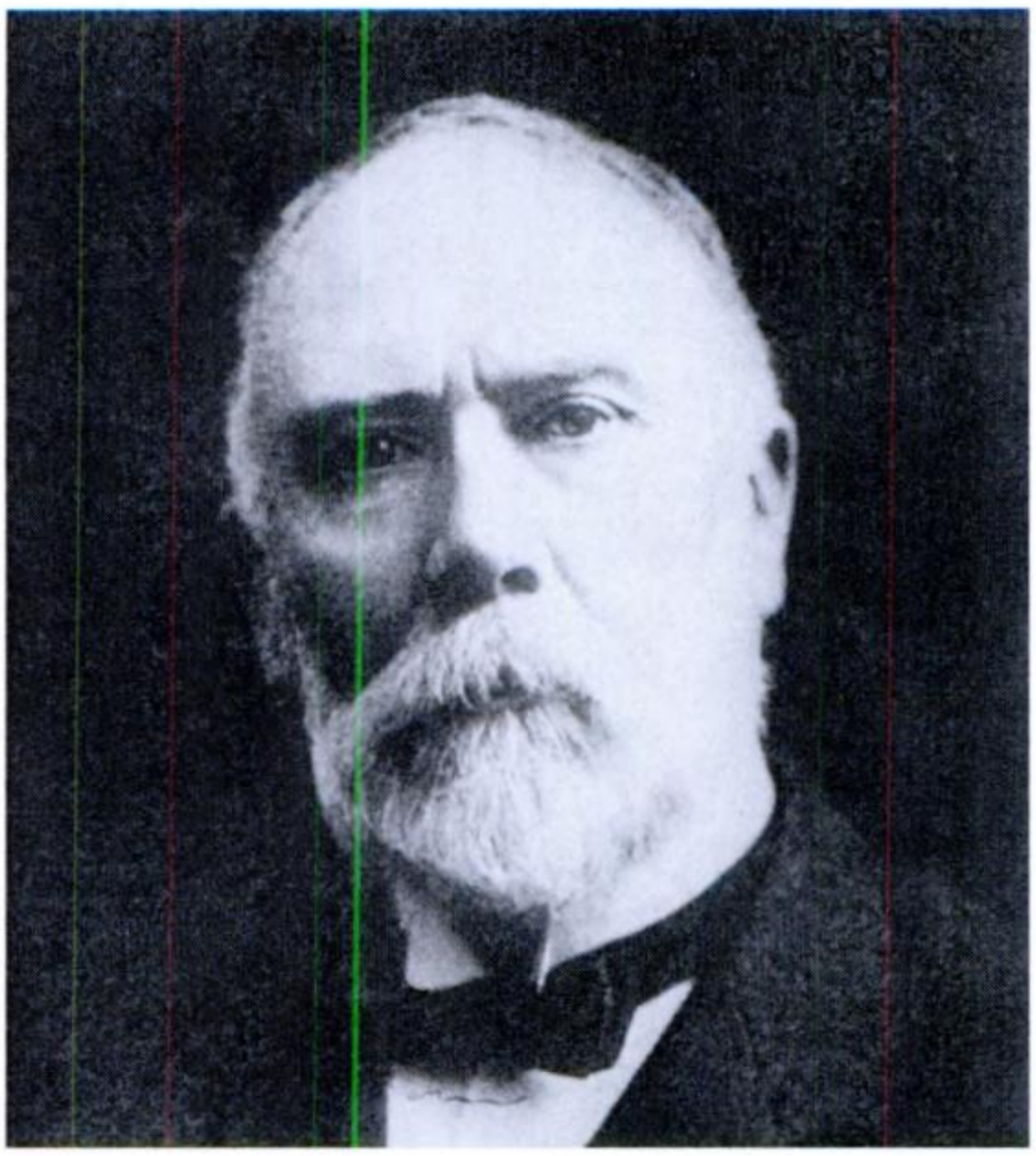


Figure 1–2 Sir James Mackenzie (1853–1925).

is believed to be the oldest medical book extant.[5] The text is in the form of a dialogue between the legendary Huang Ti (The Yellow Emperor, 2696–2598 B.C.) (Fig. 1–3) and his minister Ch'i Po. The chief means of diagnosis in *Nei Ching* is the examination of the arterial pulse. All other methods were secondary. The state of disease was judged by the volume, strength, or weakness of the pulse and by the regularity or interruption of the beats. The examination was a time-consuming affair, taking hours and involving palpation in nearly a dozen sites while the pulse rate was timed by the physician's respiratory excursions. The patient was examined in the early morning before the physician took food and before the cares of the day distracted him from full concentration. If the patient were female, the right pulses were palpated first (Fig. 1–4) and *vice versa*.

Despite the significance attached to the pulse, its origin in the heartbeat was not remotely considered. It was long in coming before Herophilus (344 B.C.) recognized that the arterial pulse and the cardiac impulse were synchronous. Herophilus used a water clock to count the pulse, analyzed rate and rhythm, was influenced by musical theories, and evolved an entire rhythmical pulse lore.[6] In 1700, Sir John Floyer commissioned an English watchmaker to construct a timepiece with a minute hand that permitted accurate determination of the rate of the pulse.[7] In *The Physician's Pulse Watch*, Floyer describes the normal and abnormal arterial pulse, its rate, rhythm, amplitude, forcefulness, and compressibility.

The first recorded account of the *cervical venous pulse* was published in 1728 when Lancisi (Fig. 1–5) described "systolic fluctuation" of the jugular vein in a patient who at necropsy had tricuspid regurgitation.[8] Rhythmic pulsations of the large veins in the neck were subsequently defined by John Hunter (Fig. 1–6) in 1794 in his *Treatise on the Blood, Inflammation,*

Figure 1–3 Huang Ti, the Yellow Emperor. (From an eighteenth-century Japanese scroll.)

and Gun-Shot Wounds.[9] "The larger veins, near to the heart, have a pulsation which arises from the contraction of the heart preventing the entrance of blood at that time."[9] Almost a century elapsed before Chauveau and Marey[10] published their classic graphic records of the *jugular* venous pulse. At the turn of the century, the jugular pulse was shown to have such striking similarity to waveforms recorded directly from the right atrium that Carl Wiggers was prompted to write, "It became increasingly more obvious to many physiologists that records of the venous pulse might be of service in the interpretation of dynamic events in the heart."[11] In 1902, Sir James Mackenzie applied this principle at the bedside in his *Study of the Pulse,*[12] which brought together 20 years of meticulous observation. Mackenzie firmly established the value of examination of the jugular venous pulse and stimulated a lively interest in its study. Paul Wood (Fig. 1–7) rekindled this interest in the 1950s.

Figure 1–4 If the patient were female, her *right* pulses were palpated first, as shown here. If the patient were male, his *left* pulses were examined first. (From Veith I [trans]. *Huang Ti Nei Ching Su Wen: The Yellow Emperor's Book of Medicine*. Berkeley: University of California Press, 1972; reprinted with permission.)

The practice of ***precordial palpation*** was recorded in the Ebers papyrus (Fig. 1–8) in a chapter entitled, "Knowledge of the Heart's Movement," which physicians can appreciate by applying their hands or fingers "upon the place of the heart."[13] Systolic movements imparted to the precordium are the most visible evidence of the action of the heart and were familiar to William Harvey (Fig. 1–9) in the early seventeenth century. "The heart is erected and rises up to a point so that at this time it strikes against the breast and is felt externally."[14] In 1857, Chauveau confirmed Harvey's contention that precordial movement observed when the heart strikes against the breast resulted from ventricular contraction.[15] Validation awaited Chauveau's animal experiments done in collaboration with Marey, in which a precordial apex beat was correlated with intracardiac pressure pulses recorded via a catheter.[15]

Applying an ear directly to the chest wall was an ancient method of auscultation. Hippocratic writings circa 400 B.C. described the "succussion splash" of hydropneumothorax.[16] William Harvey, in 1616, referred to the heartbeat as "two clacks of a water-bellows," and in *De Motu Cordis* (1628) Harvey wrote that "with each motion of the heart when there is the delivery of a quantity of blood from the veins to the arteries . . . a pulse takes place and can be heard within the chest."[14] This description leaves no doubt that Harvey was aware of sounds originating in the beating heart. Robert Hooke was not only familiar with heart sounds but also foresaw the value of auscultation, stating in his Cutlerian Lectures published in 1705: "I have been able to hear very plainly the beating of a man's heart . . . . Who knows, I say, but that it may be possible to discover the Motions of the Internal Parts of Bodies by the sound they make."[17]

Figure 1–5 Giovanni Maria Lancisi (1654–1720). (From Castiglioni A. *A History of Medicine*. New York: Alfred A. Knopf, 1947; reprinted with permission. Iconographic source—the Electa Archive.)

The modern era of cardiac auscultation began in 1816 with Rene Theophile Hyacinthe Laennec who initially practiced direct auscultation, as illustrated in an eighteenth-century painting showing him in the Necker Hospital in Paris sitting at the bedside of a patient with his ear applied to the patient's thorax (Fig. 1–10). Notwithstanding, Laennec was sensitive to the shortcomings of direct auscultation, writing, "As inconvenient for the physician as for the patient, distaste alone renders it almost impractical in the hospital; it cannot even be proposed to most women, and in most of them the volume of the breast is a physical obstacle to its use."[18] The alternative was the stethoscope (Greek *stethos* "the breast or chest" and *skopein*, "to view or examine"), the discovery of which is legendary:

> In 1816, 1 was consulted by a young woman labouring under general symptoms of diseased heart, and in whose case percussion and the application of the hand were of little avail on account on her great degree of fatness. The other method just mentioned being rendered inadmissible by age and sex of the patient, I happened to recollect a simple and well-known fact of acoustics, and fancied at the same time, that it might be turned to some use on the present occasion. The fact I allude to is the augmented impression of sound when conveyed through certain

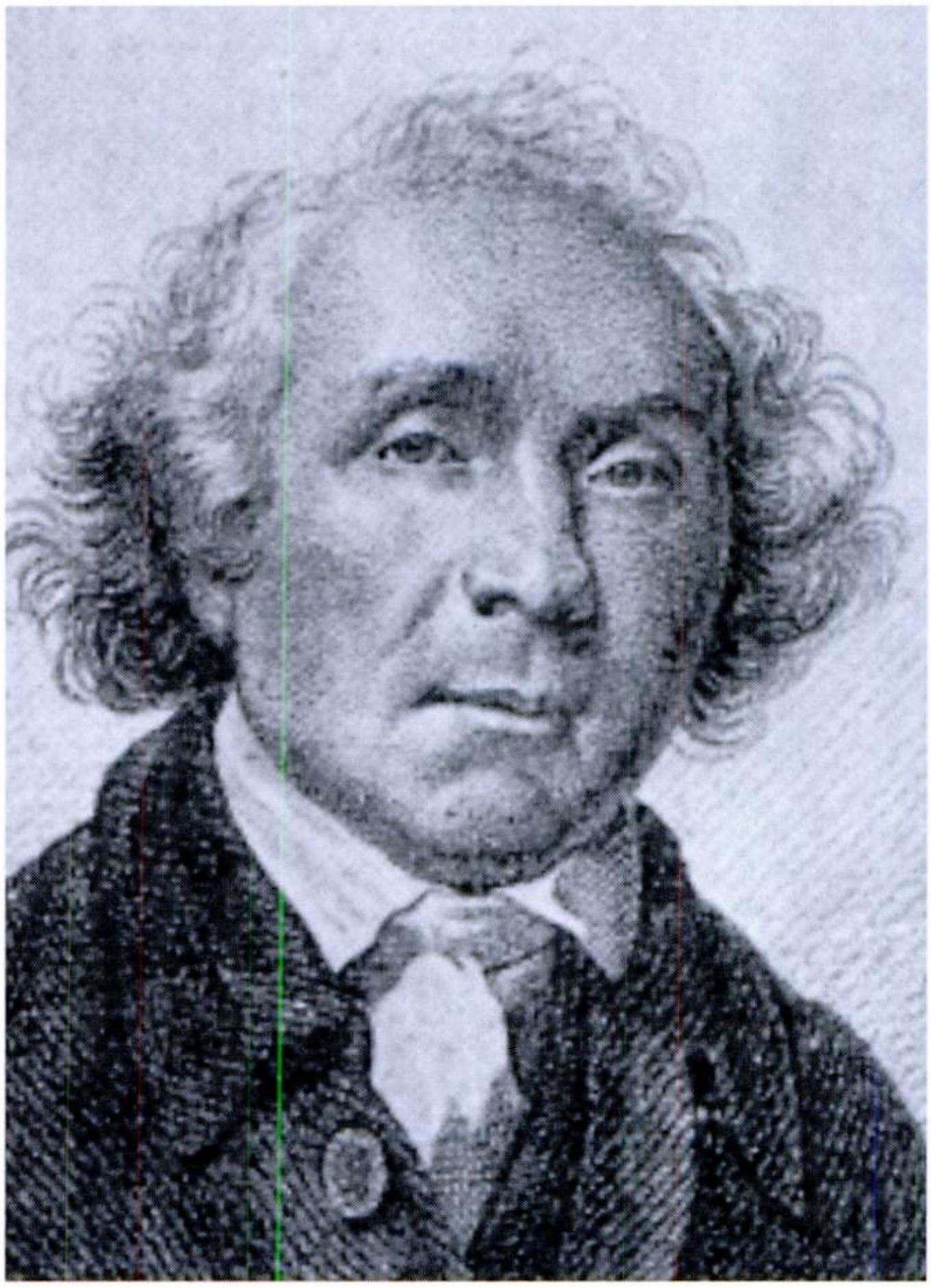


Figure 1–6 John Hunter (1728–1793), one of the greatest figures in English medical history.

solid bodies as when we hear the scratch of the pin at one end of a piece of wood on applying our ear to the other. Immediately, on this suggestion, I rolled a quire of paper into a sort of cylinder and applied one end of it to the region of the heart and the other to my ear, and was not a little surprised and pleased, to find that I could thereby perceive the action of the heart in a manner much more clear and distinct than I had ever been able to do by the immediate application of the ear. From this moment, I imagined that the circumstance might furnish means for enabling us to ascertain the character, not only of the action of the heart, but of every species of sound produced by the motion of all the thoracic viscera.[18]

The ridicule and satiric cartoons that greeted Laennec's new device were soon replaced with enthusiasm. The London *Times* in September 1824, announced, "A wonderful instrument called the stethoscope is now in complete vogue in Paris." Physicians became so attached to the "wonderful instrument" that the soon-to-come flexible binaural stethoscope struggled for recognition. As late as the turn of the century, an article

Figure 1–7 Paul Hamilton Wood, MD, OBE (1907–1962). Director, Institute of Cardiology, London. (Courtesy of Dr Arthur Selzer, San Francisco.)

in the *Lancet* hotly argued that "the double stethoscope should be altogether done away with."[19] At the same time, methods for the graphic recording of auscultatory information were moving ahead with the investigations of Otto Frank, Carl Wiggers, and Orias and Braun-Menendez.[20] The term "phonocardiogram" came into use, and Einthoven published an account of the string galvanometer for recording heart sounds.[21]

Physical signs elicited from the ***chest*** are important adjuncts to the cardiovascular examination and originated in Auenbrugger's treatise on percussion:

> I here present the Reader with a new sign which I have discovered for detecting diseases of the chest. This consists in Percussion of the human thorax, whereby, according to the character of the particular sounds thence elicited, an opinion is formed of the internal state of that cavity. In making public my discoveries respecting this matter, I have been actuated neither by an itch for writing, nor a fondness for speculation, but by the desire of submitting to my brethren the fruits of seven year's observation and reflection.[22]

Leopold Auenbrugger (Fig. 1–11), in his father's inn, witnessed the practice of percussion of wine barrels to determine the level of their fluid content. It later occurred to him that the same technique might be applied to the examination of the human thorax. In 1761, "a kindly and unassuming junior physician at the Vienna Hospital" published his "new invention—*inventum novurn*—for diagnosing thoracic disease by chest percussion."[22] Joseph

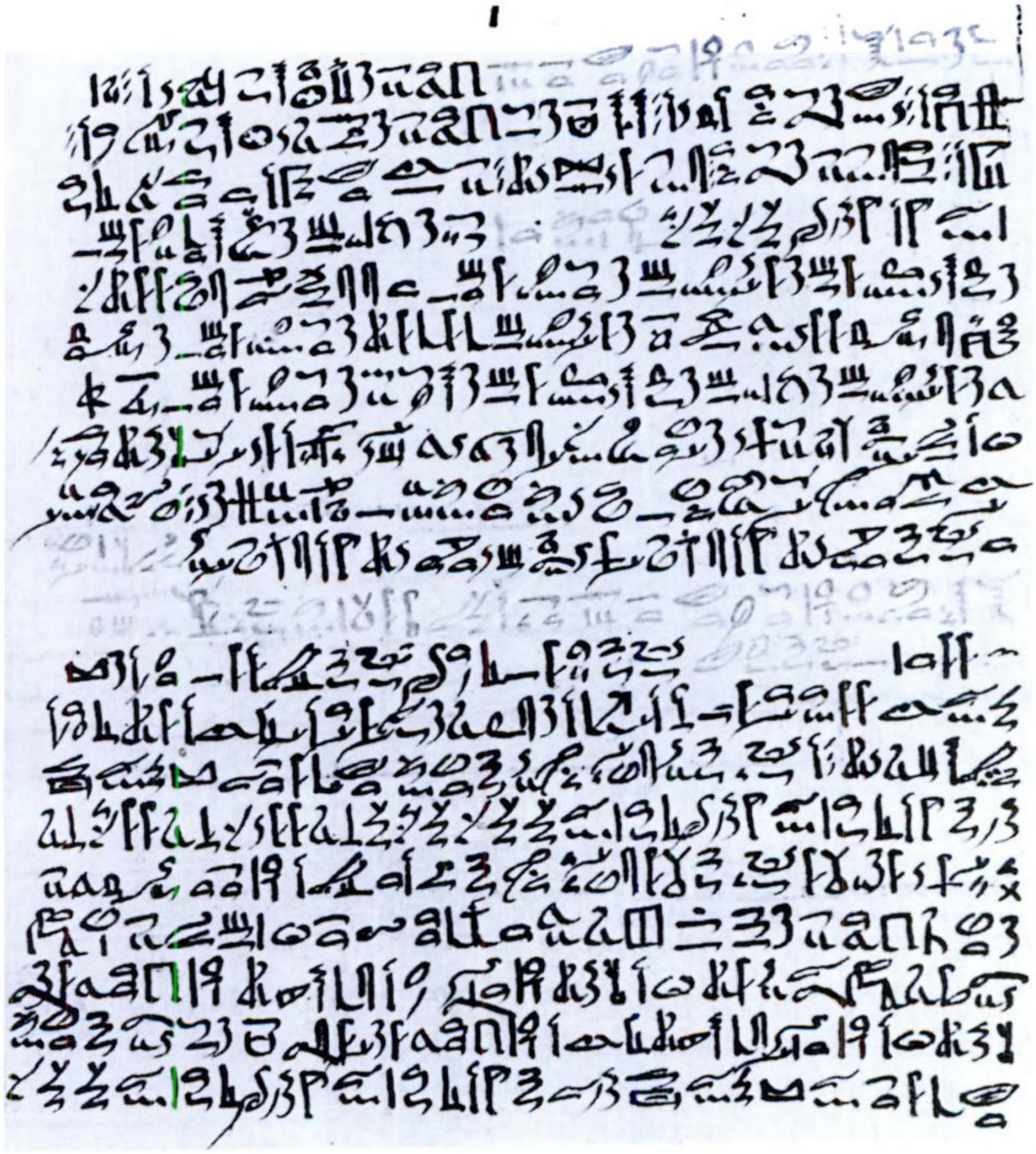


Figure 1–8 A section of the Ebers papyrus (circa 1550 B.C.) discovered by George Ebers at Thebes in 1872. (Courtesy of the UCLA Biomedical Library.)

Skoda hailed the discovery as "the beginning of modern diagnosis," but Auenbrugger's epochal work lay virtually dormant until Corvisart, the celebrated physician to Napoleon, published an elaborately annotated 480-page French edition of Auenbrugger's unpretentious 95-page book. The stage was set for Laennec's *A Treatise on the Diseases of the Chest.* In his chapter on pneumothorax, Laennec recalled the dictum of Hippocrates:

> Convinced by these symptoms of the existence of a pneumothorax combined with a pleuritic effusion, I confidently expected that the Hippocratic succussion

Figure 1–9 William Harvey (1578–1657), one of the greatest figures in the history of medicine. (Courtesy of the National Portrait Gallery, London.)

of the chest would let us hear the fluctuation of the liquid, and I was not mistaken.[18]

Richard C. Cabot's landmark book *Physical Diagnosis* (1915) contained three comprehensive chapters on diseases of the lungs and pleural cavity.[23]

Examination of the ***abdomen*** by palpation (Fig. 1–12) was practiced by the Athenian physician Jason in second century B.C.

Avicenna (980–1037), in his *Canon of Medicine*, described dropsy as "cold swellings composed of watery fluid."[24] Whether Avicenna referred to cardiac ascites is open to question, but William Withering's account of cure of the dropsy by the foxglove given by a Shropshire gypsy leaves little doubt.[25] Nevertheless, well into the twentieth century, Cabot was less than sanguine about the value of the abdominal examination:[23]

> Our methods are crude and inexact compared to those applicable to the chest. Auscultation, despite Canon's brilliant foundation studies, is of practically no use. Inspection is helpful in but few cases. Palpation, our mainstay, is often rendered almost impossible by thickness, muscular spasm, or ticklishness of the abdominal walls. Percussion is of great value in some cases, but yields no useful results in the majority.[23]

Figure 1–10 Laennec applying his ear to a patient's chest—direct auscultation. Note the wooden monaural stethoscope in Laennec's left hand (*arrow added*). (Courtesy of the National Library of Medicine, Bethesda, MD.)

Figure 1–11 Leopold Auenbrugger (1722–1809), discoverer of percussion of the thorax. (From Castiglioni A. *A History of Medicine*. New York: Alfred A. Knopf, 1947; reprinted with permission. Iconographic source—the Electa Archive.)

Figure 1–12 Stela (second-century B.C. Greek) showing palpation of the abdomen. (Courtesy of the British Museum, London.)

REFERENCES

1. Mackenzie J. *Principles of Diagnosis and Treatment in Heart Affections*. London: Oxford University Press, 1916.
2. Linfors EW, Neelson FA. The case for bedside rounds. *N Engl J Med.* 1980;303:1230.
3. Cushing H. *The Life of Sir William Osler.* Oxford: Clarendon Press, 1925.
4. Mackenzie J. *Diseases of the Heart.* London: Henry Frowde, Hodder, & Stoughton, 1908.
5. Veith IH, trans. *Huang Ti Nei Ching Su Wen: The Yellow Emperor's Book of Medicine.* Berkeley, CA: University of California Press, 1972.
6. Osler W. *The Evolution of Modern Medicine.* New Haven, CT: Yale University Press, 1921.
7. Floyer J. *The Physician's Pulse Watch.* London: Smith and Walford, 1707.
8. Lancisi JM. *Mortu Cordis et Aneurysmabitus.* Roma 1728.
9. Hunter J. *A Treatise on the Blood, Inflammation, and Gun-Shot Wounds.* London: George Nicol, Bookseller to His Majesty, Pall-Mall, 1794.

10. Chauveau JBA, Marey EJ. Appariels et experiences cardiographics. *Memoirs Acad Med.* 1863;26:268.
11. Wiggers CJ. *The Pressure Pulses in the Cardiovascular System.* London: Longmans, Green, 1928.
12. Mackenzie J. *The Study of the Pulse, Arterial, Venous, and Hepatic, and of the Movements of the Heart.* Edinburgh: Young J. Pentland, 1902.
13. Basta LL, Bettinger JJ. *The Cardiac Impulse: A New Look at an Old Art. Am Heart J.* 1979;97:96.
14. Harvey W. *An Anatomical Disquisition on the Motion of the Heart and Blood in Animals.* London, 1628 (translated from the Latin by Robert Willis, Barnes, Surrey, England, 1847). In: Willius FA, Keys TE, eds. *Classics of Cardiology*, Vol. 1. Malabar, FL: Robert E. Krieger, 1983:19.
15. Chauveau JBA, Marey EJ. Determination graphiques des rapports de la pulsation cardiaque avec les mouvements de Foreillette et du ventricle, obtenue au moyen d'un appareil enregistreur. *C R Soc Biol. (Paris)* 1861;13:3.
16. McKusick VA, Sharp WD, Warner AO. An exhibition on the history of cardiovascular sound including the evolution of the stethoscope. *Bull Hist Med.* 1957;31:463.
17. Hooke R. The posthumous works of Robert Hooke, containing his Cutlerian Lectures and other discourses read at the meeting of the Illustrious Royal Society, etc. In: McKusick VA, ed. *Cardiovascular Sound.* Baltimore: Williams & Wilkins, 1958:4.
18. Laennec RTH. *A Treatise on the Diseases of the Chest* (translated from the French by John Forbes, MD). London: T and G Underwood, Fleet Street, 1824.
19. Sayers HW. The decay of auscultation and the use of the binaural stethoscope. *Lancet* 1902;1:369.
20. Orias O, Braun-Menendez E. *The Heart Sounds in Normal and Pathological Conditions.* London: Oxford University Press, 1939.
21. Einthoven W. Die registrierung des menschlichen herztone mittels des sartengalvanometers. *Pflugers Arch Physiol.* 1907;117:461.
22. Auenbrugger L. Inventum Novum ex Percussione Thoracis Humani. Vienna, 1761 (translated by John Forbes). London: T and G Underwood, Fleet Street, 1824.
23. Cabot RC. *Physical Diagnosis*, 6th ed. New York: William Wood, 1915.
24. Gruner OC. *A Treatise on the Canon of Avicenna.* London: Luzac, 1930.
25. Withering W. *An Account of the Foxglove and Some of its Medical Uses.* Birmingham: M. Swinney, 1785.

2 Physical Appearance

The eye is the chief means whereby the understanding may most fully and abundantly appreciate the infinite works of nature.

Leonardo da Vinci

What the eye sees—the patient's general and detailed physical appearance—is the subject of this chapter. In order *to see*, however, the eye must be trained. The "innocent eye, which should see the world afresh, does not see it at all."[1]

Certain distinctive physical appearances are associated with certain specific cardiac diseases, whereas other appearances are *caused* by cardiac or vascular diseases. *General* appearance should first be focused upon, and then *detailed appearance* (Table 2–1). I have selected examples of practical interest. No attempt was made to be comprehensive.

Systematic assessment of a patient's appearance is an integral part of the physical examination of the heart and circulation. I shall deal in turn with (1) general somatic features, (2) gestures and gait, and (3) detailed appearance of face, eyes, mouth, hands, feet, skin, muscles, tendons, thorax, and abdomen.

GENERAL APPEARANCE

General appearance can distinguish at a glance acute from chronic illness, but a patient may look well despite the presence of cardiac or vascular disease. The picture of certain acute illnesses is well known. Pulmonary edema is characterized by a struggling, frightened, diaphoretic patient, dyspneic while sitting bolt upright. Or witness the patient with an acute myocardial infarction—anxious, ashen, diaphoretic, reacting to oppressive chest pain with its real and symbolic meanings. The picture of cardiogenic shock, familiar to us all, was perfectly described in Shakespeare's death of Falstaff:

> So he cried out, "God, God!" three or four times. Now I, to comfort him, bid him he should not think of God; I hoped that there is no need to trouble himself with any such thoughts yet. So he bade me lay more clothes on his feet; I put my hand into the bed and felt them and they were as cold as any stone; I then felt to his

Table 2–1 Physical Appearance

General appearance
Gestures and gait
Detailed appearance
Face
Eyes—external and internal
Mouth—external and internal
Hands and feet
Skin
Muscles and tendons
Thorax
Abdomen

knees; and they were as cold as any stone, and so upward and upward, all was as cold as any stone.[2]

The general appearance of chronic illness is exemplified by the catabolic effects of congestive heart failure—wasted pectoral, shoulder girdle, and arm muscles, edematous legs, protruding ascitic abdomen, and drawn face. The appearance of the coronary-prone patient is a stereotype—a mesomorphic, balding, hirsute, middle-aged, overweight male of good appetite whose fingers are stained with nicotine, whose ashtray is filled with the butts of cigarettes that preceded the one he is smoking, while close by is a briefcase bulging with papers—symbols of deadlines he feels compelled to meet.

The general appearance of thyrotoxicosis is typified by a lean, perspiring, tremulous individual with quick movements and bright eyes with or without exophthalmos. Hypothyroidism is the antithesis—lethargic and pallid, with slow movements and coarse features.

A patient may be distinctively tall[3] (gigantism) or distinctively short (dwarfism). Marfan syndrome is an example of a strikingly tall person with excessively long extremities and sparse subcutaneous fat. Gigantism in the neonate is represented by the infant of the diabetic mother (Fig. 2–1). The description of James W. Farquhar is apt:

> The infants are remarkable not only because, like foetal versions of Shadrach, Meshach and Abednego, they emerge at least alive from within the fiery metabolic furnace of diabetes mellitus, but because they resemble one another so closely that they might well be related. They are plump, sleek, liberally coated with vernix caseosa, full-faced and plethoric. The umbilical cord and the placenta share in the gigantism. During their first *24* or more of extrauterine hours, they lie on their backs, bloated and flushed, their legs flexed and abducted, their lightly closed hands on each side of the head, the abdomen prominent and their respiration sighing. They convey a distinct impression of having had such a surfeit of both food and fluid pressed upon them by an insistent hostess that they desire only peace so that they may recover from their excesses.[4]

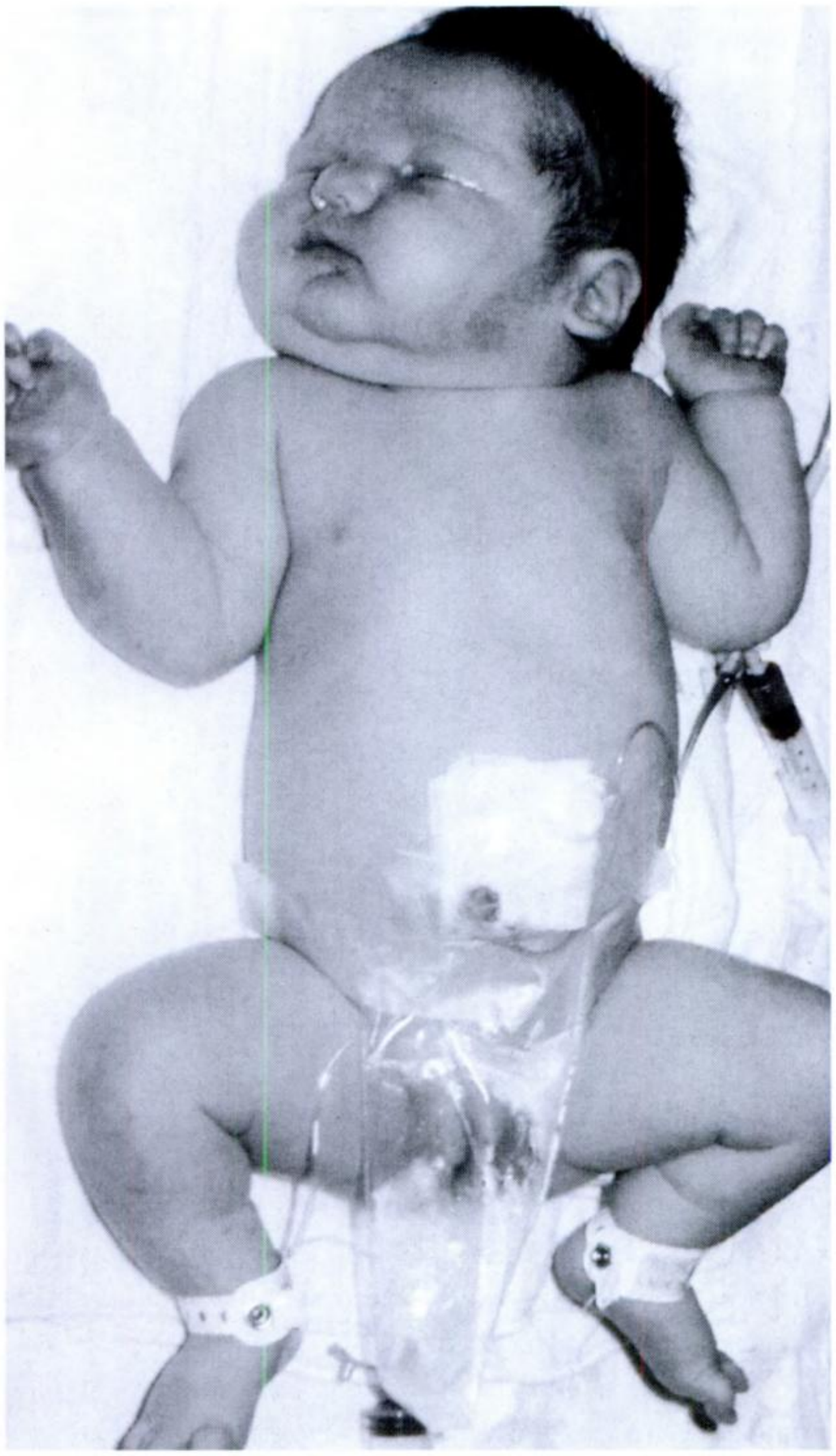
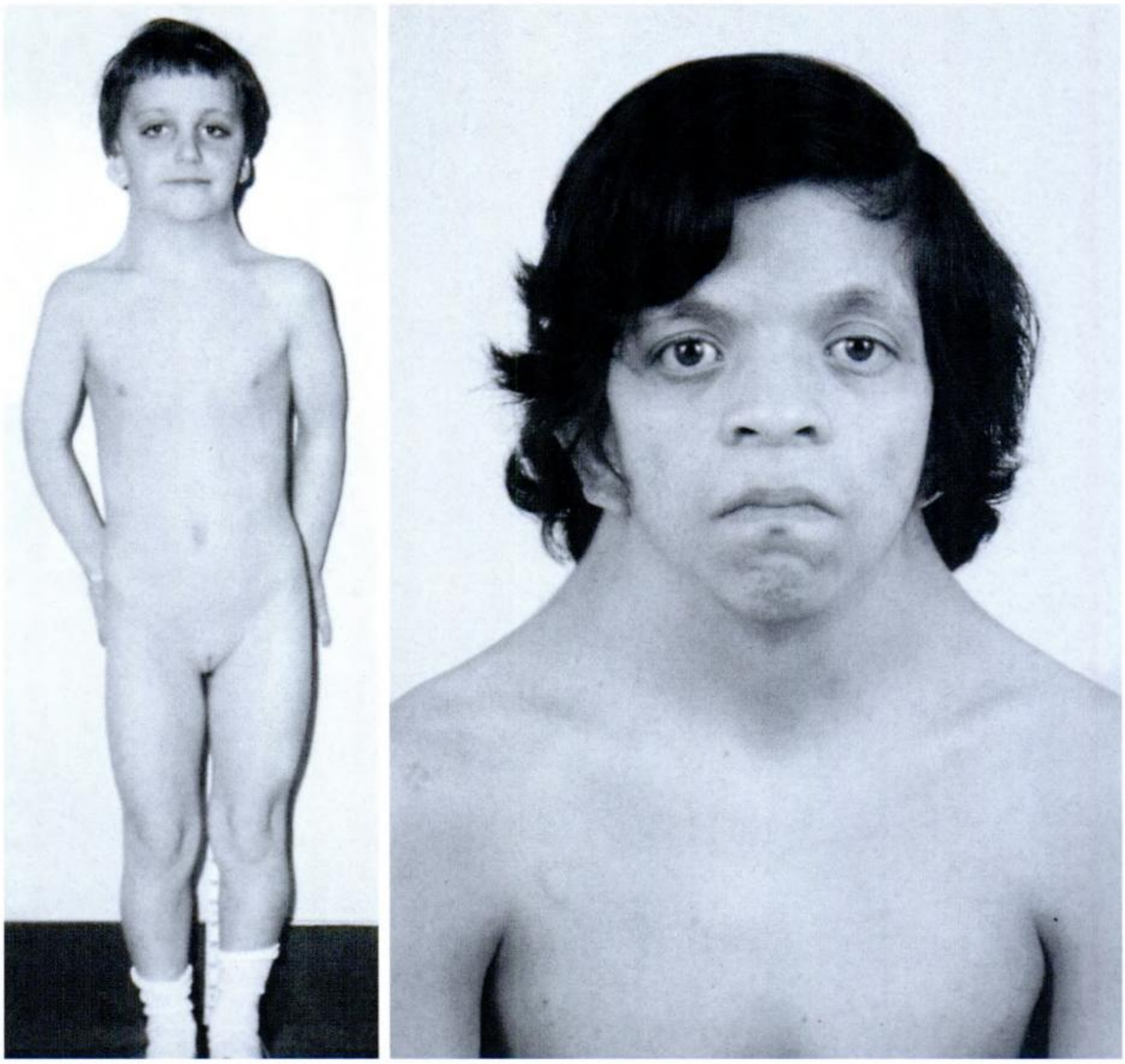


Figure 2–1 Infant of a diabetic mother showing typical neonatal gigantism—plump, full-faced, plethoric, bloated, and flushed. "They lie on their backs, bloated and flushed, their legs flexed and abducted, their lightly closed hands on each side of the head, the abdomen prominent."[4]

Short stature as represented by the 45/X0 Turner syndrome (Fig. 2–2*A*) is characterized by a phenotypic female with webbing of the neck, sexual infantilism (absent or scanty pubic and axillary hair), wide-set nipples, low hairlines usually concealed by bangs, and long hair, small chin, and wide carrying angles of the arms.[5] The "female Turner" (45/X0) predicts the presence of coarctation of the aorta, dilated aortic root, and bicuspid aortic valve. Noonan syndrome is a Turner phenotype with normal chromosomal composition (46/XX female, 46/XY male) (Fig. 2–2*B*), but dwarfism is not the rule. The cardiac disease is likely to consist of dysplastic pulmonary valve stenosis or hypertrophic obstructive cardiomyopathy.[5] The Ellis–van Creveld syndrome is characterized by dwarfism and polydactyly.[5]



A B

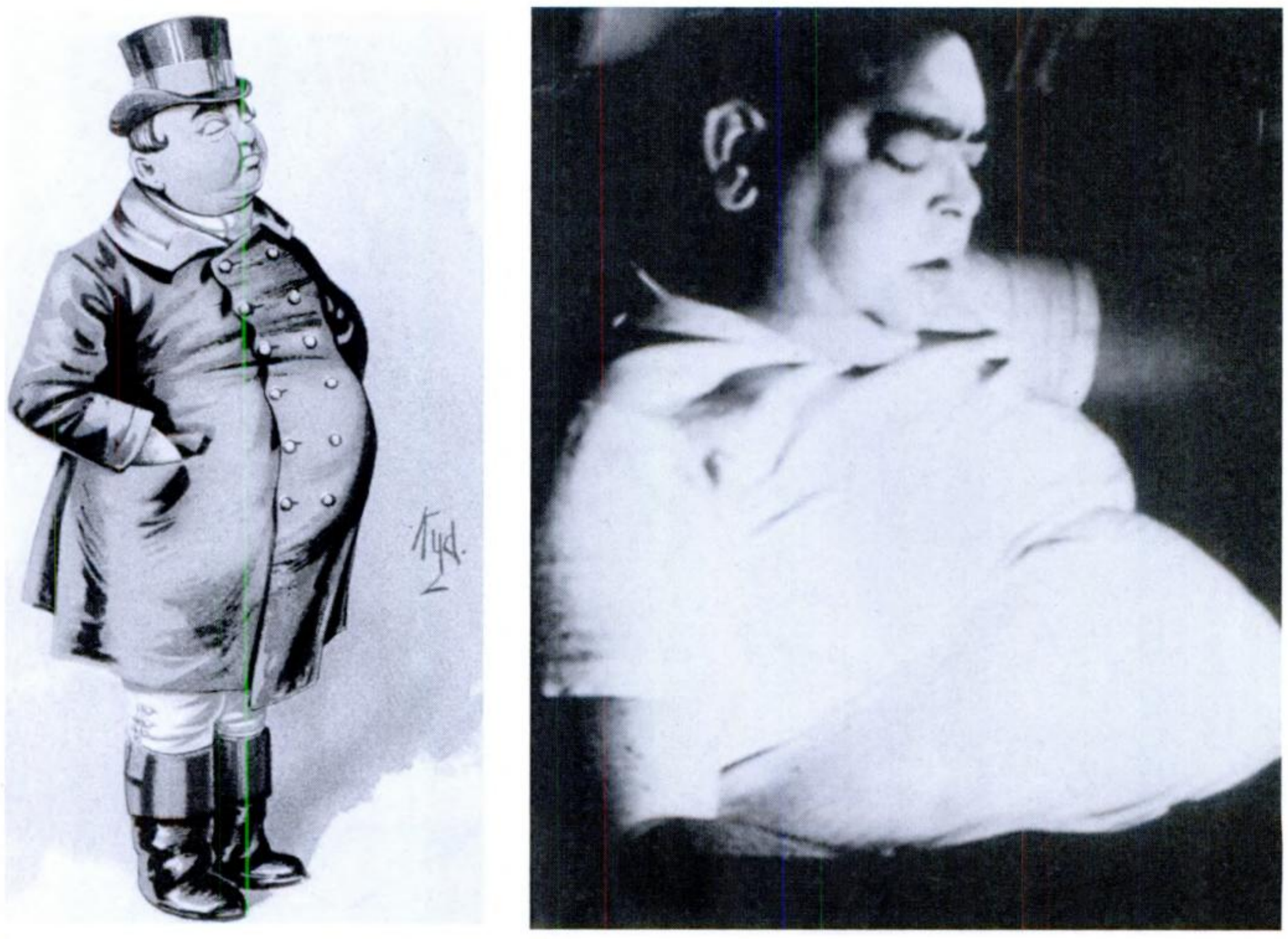
Figure 2–2 *A,* A 13-year-old girl with 45/X0 Turner syndrome and the typical phenotype of short stature, webbing of the neck, absence of pubic hair, wide-set nipples, and small chin. Bangs cover a low anterior hairline. The patient had coarctation of the aorta. *B,* An 18-year-old boy with 46/XY Noonan syndrome and typical webbing of the neck. Long hair obscures low anterior and posterior hairlines. The patient had dysplastic pulmonary valve stenosis and an atrial septal defect.

Obesity varies in appearance and should be carefully characterized. The combination of obesity and somnolence—*the pickwickian syndrome*—was so named by C. Sidney Burwell based upon the description of Charles Dickens's fat boy (Fig. 2–3*A*):

> A most violent and startling knocking was heard at the door. The object that presented itself to the eyes of the astonished clerk was a boy—a wonderfully fat boy—standing upright on the mat, with his eyes closed as if in sleep. He had

> never seen such a fat boy, and this, coupled with the utter calmness and repose of his appearance, so very different from what was reasonably to have been expected of the inflictor of such knocks, smote him with wonder. The extraordinary boy spoke not a word; but he nodded once and seemed to the clerk's imagination to snore feebly.[6]

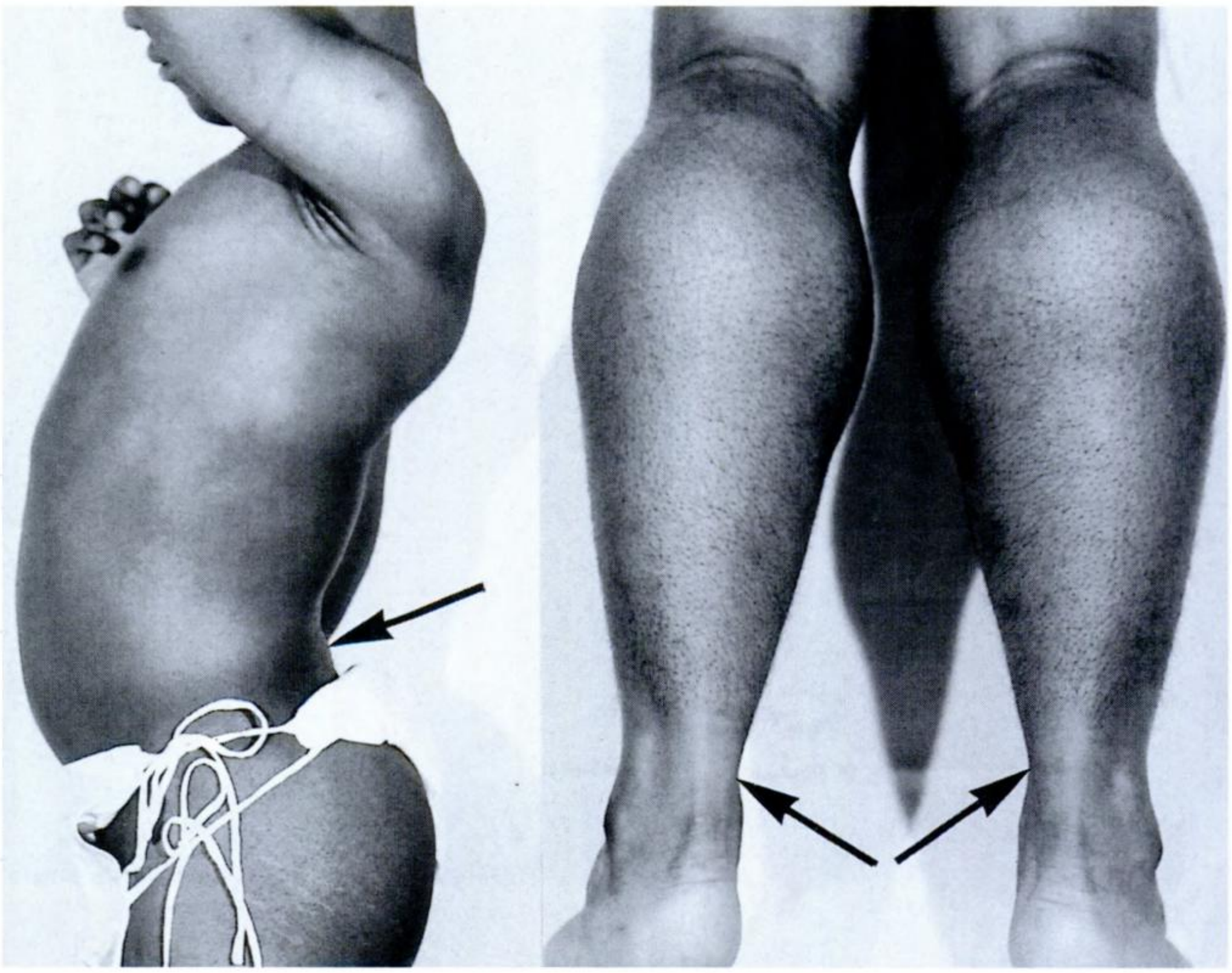
Alveolar hypoventilation that accompanies pickwickian obesity results in hypercapnea, somnolence (Fig. 2–3*B*), hyoxemia, increased pulmonary vascular resistance, and pulmonary hypertension. By contrast, the obesity of Cushing syndrome coincides with systemic hypertension. The cushingoid appearance is typified by central obesity with rounding of the face, thick fat pads in the supraclavicular fossae, and a marked increase in thoraco-abdominal paniculus adiposus. Abdominal obesity can progress to grotesque proportions while the extremities remain remarkably slender.



A B

Figure 2–3 *A,* Charles Dickens' *Fat Boy:* "He's gone to sleep again. Be good enough to pinch him, sir—in the leg, if you please; nothing else wakes him" (*Pickwick Papers,* Chapter IV). *B,* An obese 32-year-old man with the pickwickian syndrome photographed by the author as the patient fell asleep while his history was being taken.

Gestures and gait can be important features of physical appearance. A clenched fist pressed against the sternum is a time-honored gesture of the oppressive pain of myocardial infarction. The importance of gait was emphasized by James Mackenzie: "When the patient presents for examination, the physician naturally scrutinizes him and makes a mental note of his gait and how he deports himself generally."[7] However, except for neurologists, physicians seldom routinely observe their patient's walk, even though an abnormal gait may be the first evidence of a systemic neuromuscular disease with cardiac involvement. Pseudohypertrophic Duchenne muscular dystrophy, for example, results in a characteristic slow, clumsy waddling gait caricatured by exaggerated lumbar lordosis and protuberant abdomen (Fig. 2–4). When such a patient is asked to rise from a recumbent position, he is apt to use the distinctive gestures originally described by Gowers in 1888 (Fig. 2–5). The cardiac involvement in Duchenne dystrophy targets the posterobasal left ventricular wall.



A B

Figure 2–4 *A,* Young boy with classic pseudohypertrophic X-linked Duchenne muscular dystrophy and exaggerated lumbar lordosis (*arrow*). *B,* Striking calf pseudohypertrophy with shortening of the Achilles tendons (*arrows*) causing the patient to stand on his toes. Cardiac disease was characterized by regional dystrophy of the posterobasal left ventricular wall.

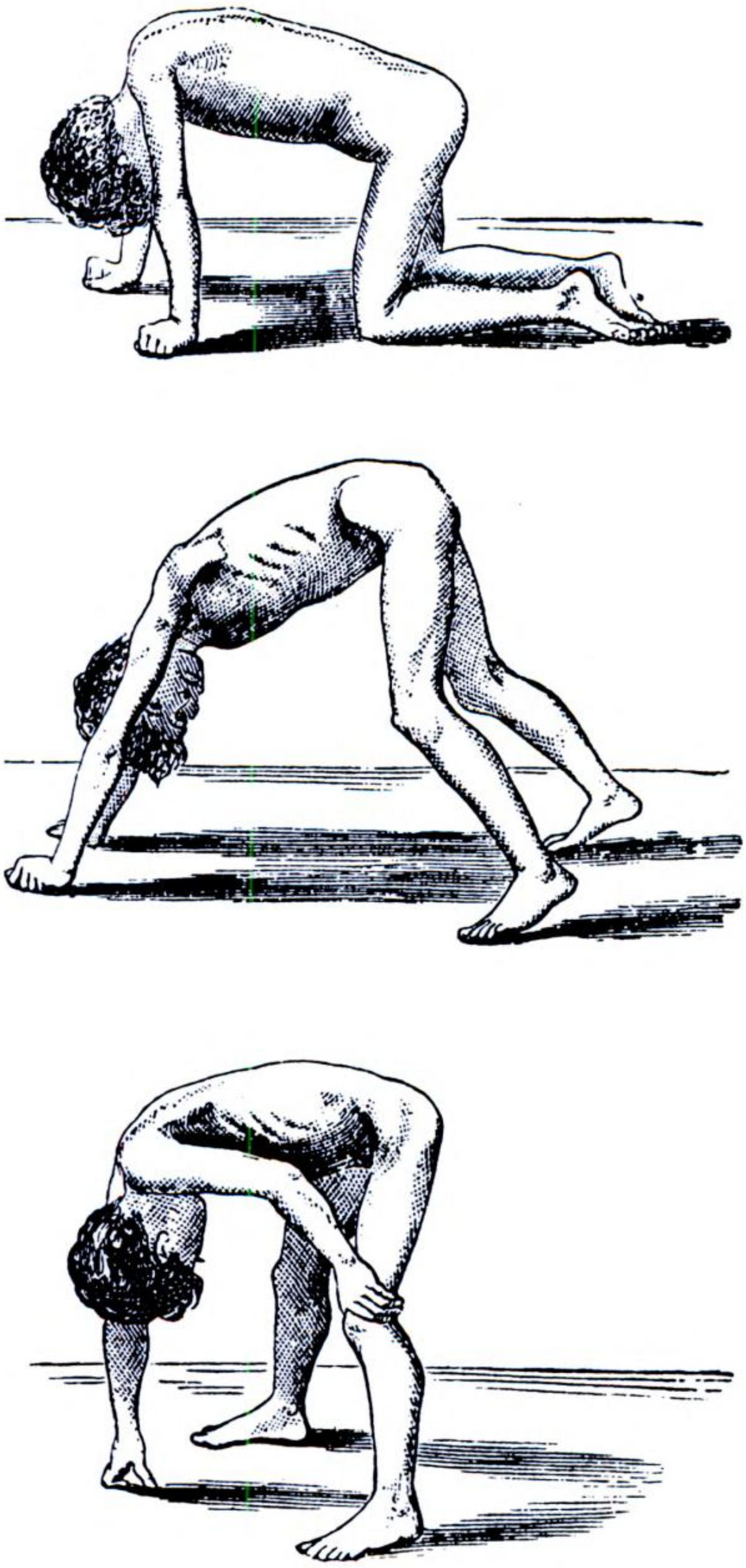


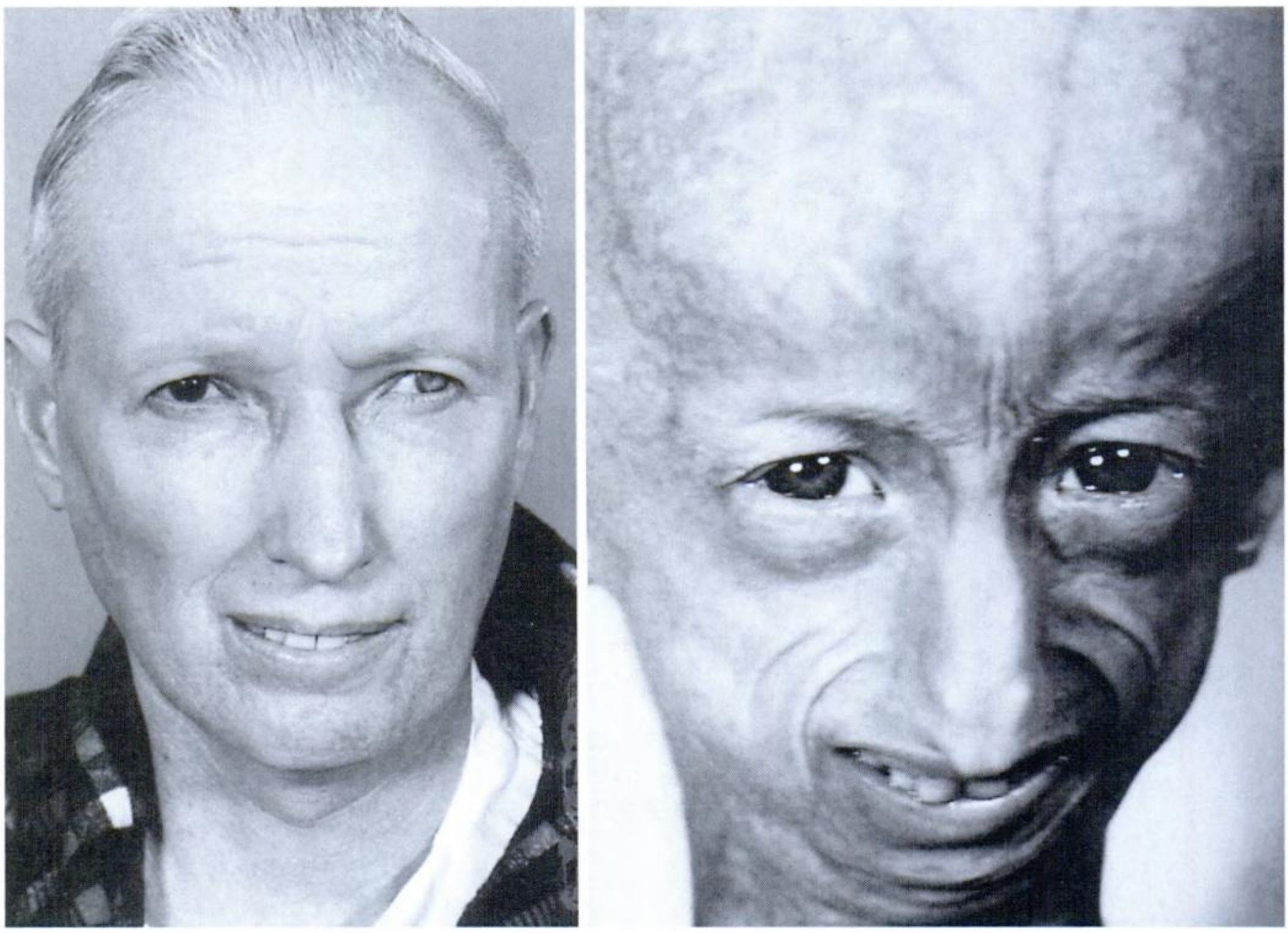
Figure 2–5 Gowers sign: "Mode of rising from the ground in pseudohypertrophic paralysis." (From Gowers WR: Diseases of the Nervous System. Philadelphia, R Blakiston, Son & Co, 1888.)

DETAILED APPEARANCE

For by his face straight shall you know his heart.[8]

Facial Expression

The face can express anxiety or calm. Anxiety can provoke a variety of cardio-circulatory disturbances in the absence of organic heart disease, or may be the result of real or imagined fear of cardiac disease. The face is often a barometer of age—chronologically appropriate or inappropriate. Premature aging takes one of its most dramatic forms in Werner syndrome[9] (Fig. 2–6*A*), a disorder characterized in part by premature graying of the hair, frontal baldness in males, cataracts, taut skin, and beaking of the nose. Coronary artery disease and systemic atherosclerosis are strikingly premature.



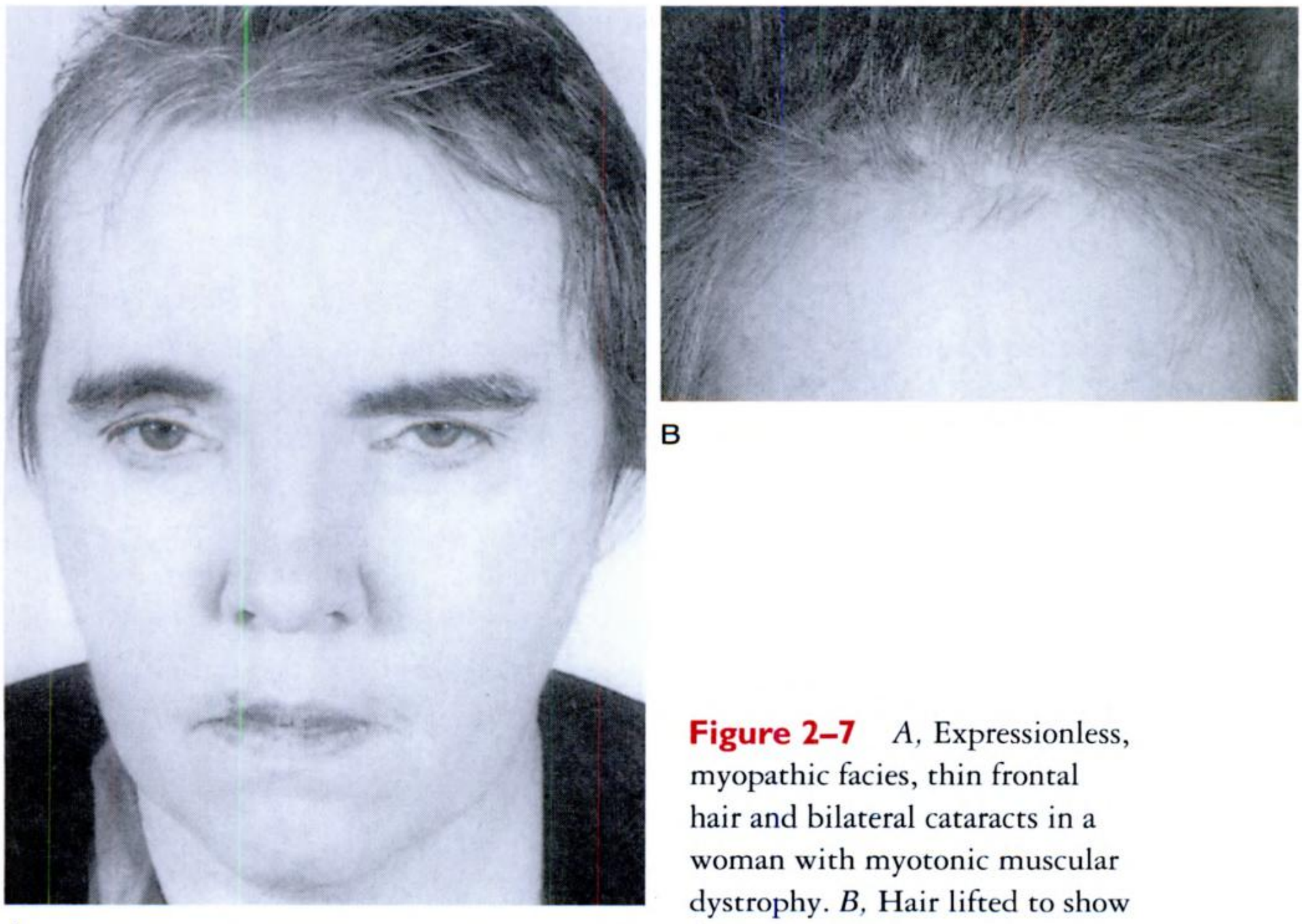
A B

Figure 2–6 *A,* A 34-year-old man with the typical premature aging of Werner syndrome characterized by graying of the hair, frontal baldness, and a cataract in the left eye. Atrophic skin is pulled tightly over the bridge of the nose. *B,* A 10-year-old boy with the Hutchinson–Gilford syndrome consisting of progeria (premature aging) with nanism (dwarfism). There is complete alopecia and tightly drawn atrophic skin. The boy died of myocardial infarction.

In children, a dramatic variation on this theme is the Hutchinson–Gilford syndrome (Fig. 2–6*B*), with alopecia, senile appearance, premature aging, childhood atherosclerosis, and death from cardiac infarction. Patients with myotonic muscular dystrophy appear prematurely old, with graying of the hair, frontal baldness or thinning even in females, cataracts, and an expressionless, myopathic facies (Fig. 2–7). Abnormalities of cardiac impulse formation and conduction are typical.

A facial "butterfly rash" with malar depigmentation are features of systemic lupus erythernatosus. In the carcinoid syndrome, a cardinal facial appearance is a sudden bright red to violaceous hue accompanied by facial and periorbital edema. During the flush, hypotension, even shock, sometimes occurs. Cardiac involvement usually takes the form of pulmonary valve stenosis and tricuspid stenosis/ regurgitation.

Facial appearance in hypothyroidism is characterized by dull, coarse features, an enlarged tongue, thickened skin, dry hair that does not maintain a curl, puffy eyelids, and sparse eyebrows, the outer thirds of which may be absent. In the Hurler's syndrome, *gargoylism* refers to grotesque facial features recognizable at a glance (Fig. 2–8*A*). The skull is malformed, the supraorbital ridges are prominent, the bridge of the nose is depressed, the lips are thick, and the teeth are peg-shaped as in a gargoyle (Fig. 2–8*B*). Hurler's syndrome is a form of mucopolysaccharidosis that involves the mitral and aortic valves together with thickening of the walls and narrowing of the lumina of the coronary arteries.[10]



A

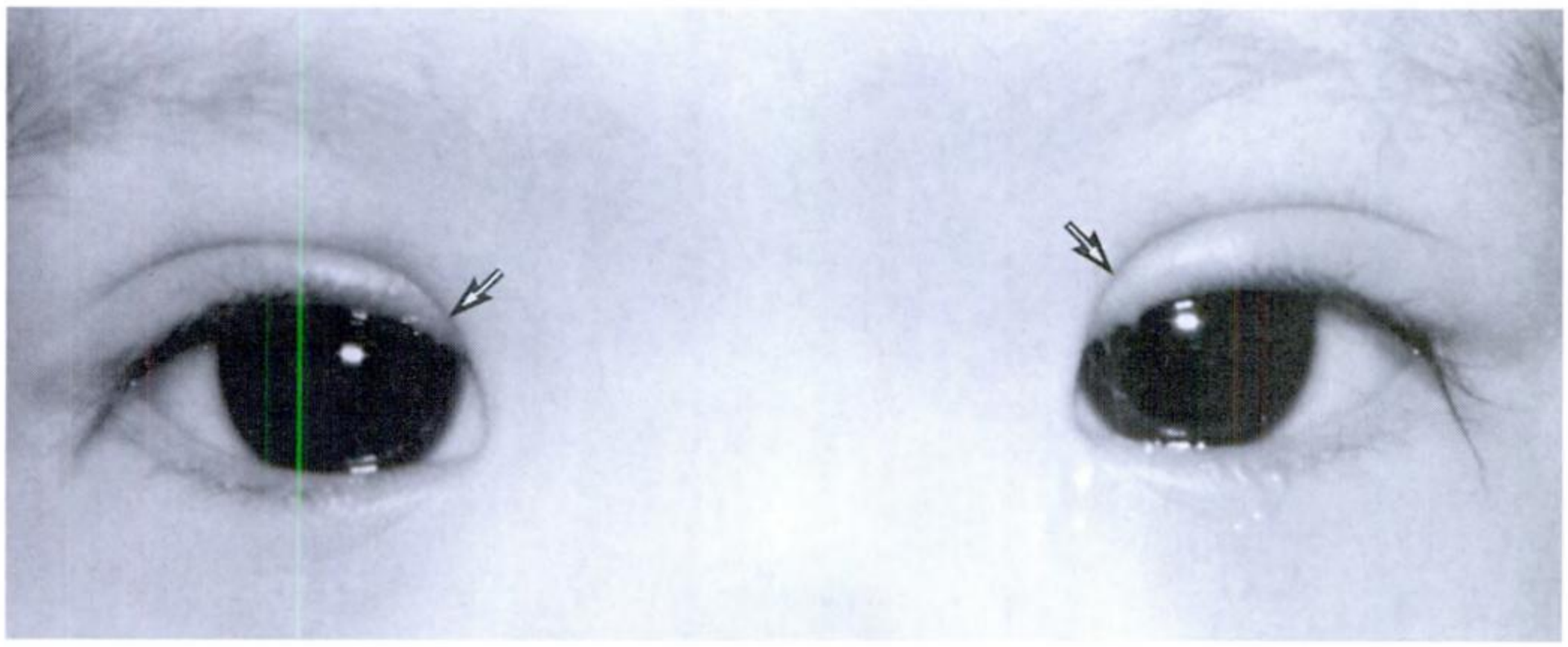
B

Figure 2–7 *A,* Expressionless, myopathic facies, thin frontal hair and bilateral cataracts in a woman with myotonic muscular dystrophy. *B,* Hair lifted to show frontal baldness.

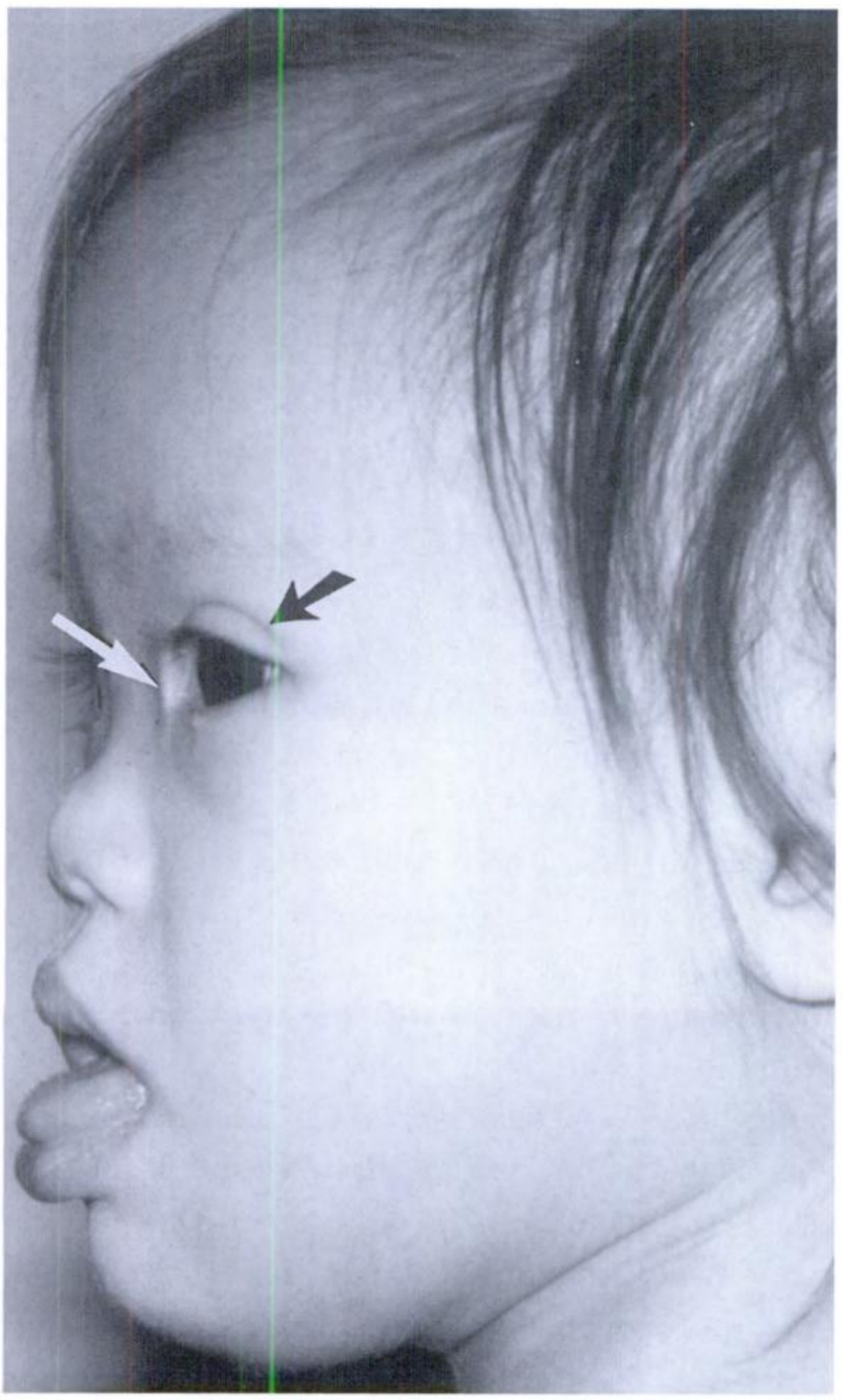
A B

Figure 2–8 *A,* Child with typical coarse physiognomy of Hurler syndrome (gargoylism). Necropsy disclosed thickened mitral leaflets and severely narrowed coronary arteries. (Courtesy of Dr Hines Zellweger, University of Iowa, Iowa City.) *B,* Gargoyle showing the typical grotesque bulging eyes, prominent supraorbital ridges, blunt upturned nose, thick lips, and peg teeth. (Courtesy of Professor Robbert Flick, Department of Fine Arts, University of Southern California, Los Angeles.)

A number of facial appearances are associated with specific or relatively specific *congenital cardiac or vascular diseases.* Figure 2–9*A* is an example of Down syndrome with typical inner epicanthic folds and depressed nasal bridge. Figure 2–9*B* is the profile of an Asian child with Down syndrome, *inner epic*anthic folds, normal horizontal Asian epicanthic fold above the *outer* canthus, flat nose with depressed nasal bridge, and a characteristically protrubant tongue. Another distinctive facial appearance typifies Williams syndrome that coexists with nonfamilial supravalvular aortic stenosis and pulmonary artery stenosis. The face is characterized by a large mouth, patulous lips, small chin, baggy cheeks, blunt upturned nose, wide-set eyes, and malformed teeth (Fig. 2–10). The fetal alcohol syndrome is a consequence of heavy maternal drinking during gestation. Offspring have short palpebral fissures, a hypoplastic upper lip with thin vermilion, diminished to absent philtrum, micrognathia, and midfacial growth deficiency[11] (Fig. 2–11). Coexisting congenital cardiac malformations usually take the form of atrial or ventricular septal defect alone or with pulmonary valve stenosis. The *cardiofacial syndrome* consists of distinctive unilateral partial lower facial weakness that appears only when the child cries.[12]



A



B

Figure 2–9 *A,* Down syndrome with typical inner epicanthic folds *(arrows)* and depressed nasal bridge. *B,* Profile of a Chinese child with Down syndrome, *inner* epicanthic fold (*white arrow)*, normal horizontal Asian epicanthic fold above the *outer* canthus (*black arrow*), *flat* nose with depressed nasal bridge, and a characteristically protuberant tongue.

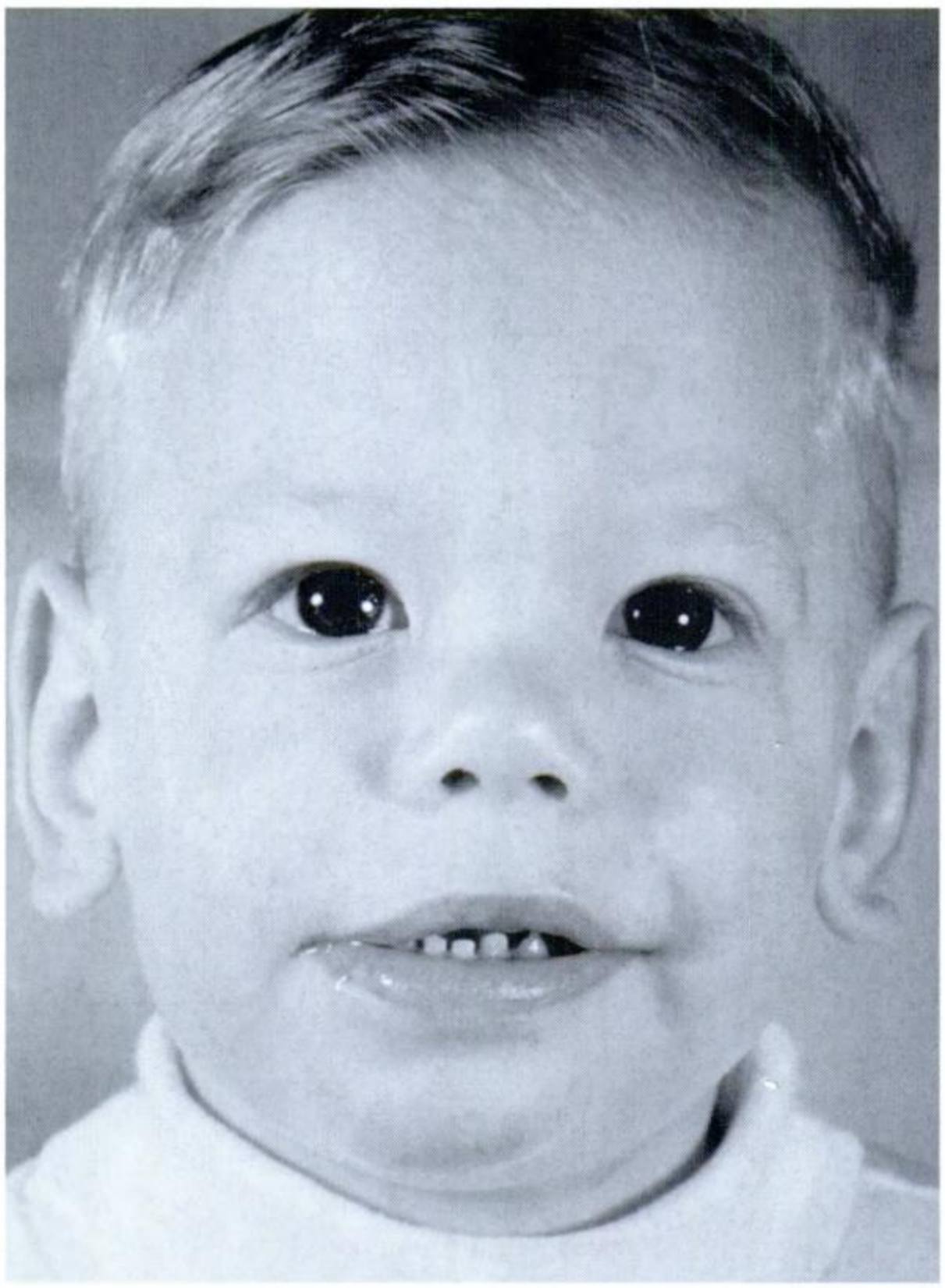


Figure 2–10 Facial appearance of Williams syndrome with the typical small chin, large mouth, patulous lips, blunt upturned nose, wide-set eyes, broad forehead, baggy cheeks, and malformed teeth. The patient was mentally retarded and had infantile hypercalcemia, bilateral stenosis of the pulmonary arteries, and supravalvular aortic stenosis.

External and Internal Appearance of the Eyes

External examination of the eyes is part of the examination of the face. Some abnormalities are recognizable at a glance (strabismus or conspicuous exophthalmos), while detection of other abnormalities requires careful inspection of lids, lacrimal glands, palpebral fissures, eyebrows, conjunctivae, sclerae, cornea, iris, and lens.

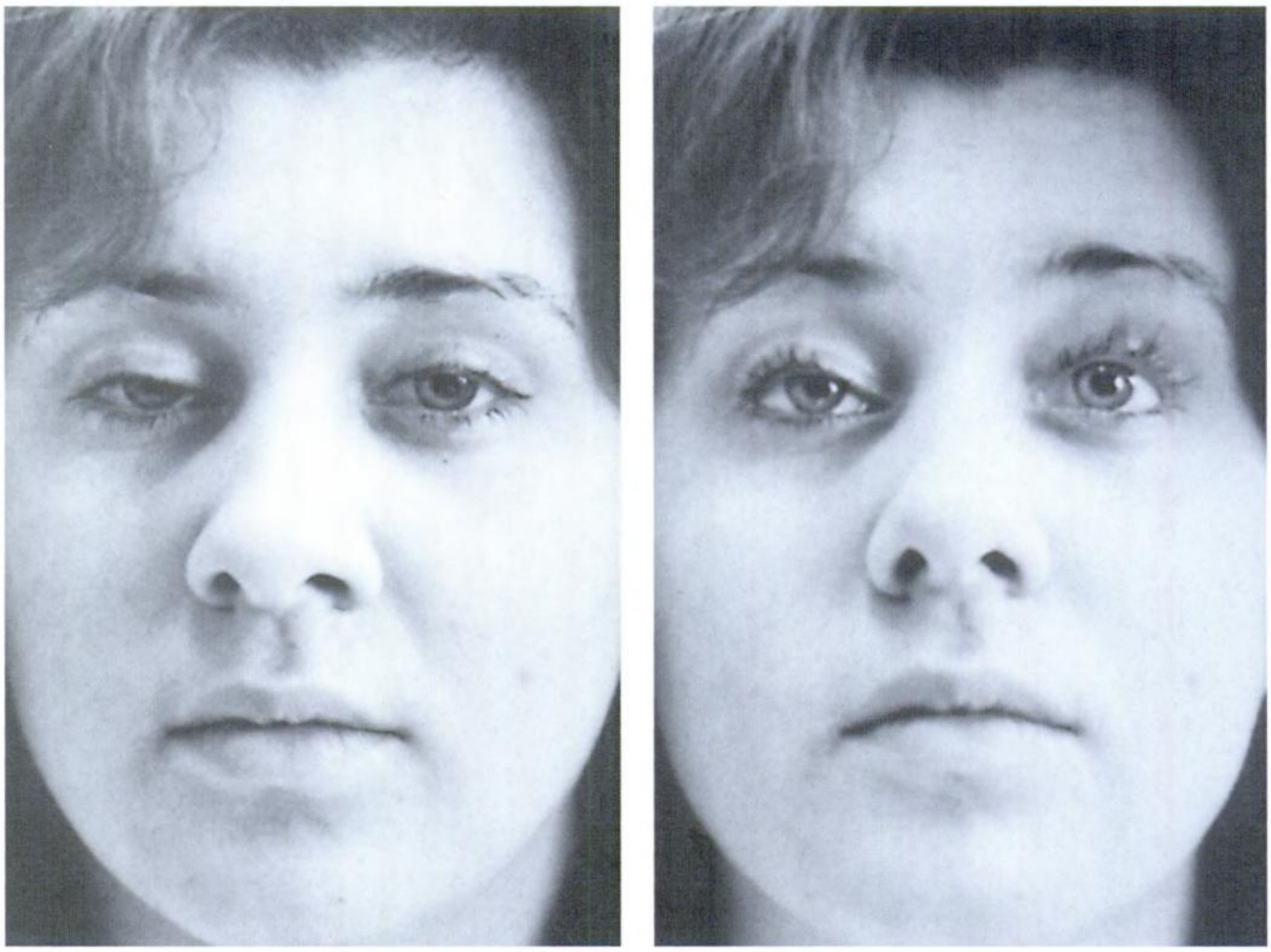
The **bulbus oculi** (ocular bulb or eyeball) is normally well contained within its bony cavity, the orbit. In hyperthyroidism, the appearance of the ocular bulb varies from symmetric or asymmetric protrusion (exophthalmos) to mild lid retraction and stare. Slight proptosis and stare in euthyroid patients are occasionally due to chronic elevation of systemic venous pressure. Proptosis in patients with severe tricuspid regurgitation, may be accompanied by systolic anterior movement of the ocular bulb that is best detected when the examiner observes the eyes with an oblique light while the patient sits and looks directly forward. Conversely, shrunken eyeballs occur when the catabolic effects of congestive heart failure cause loss of retro-orbital fat and connective tissue. Nystagmus—involuntary oscillations of the ocular bulb with lateral gaze—is a feature of Friedreich ataxia.

The **eyelids** themselves sometimes disclose telltale signs. A common example is *xanthalasma*, which are circumscribed, yellowish cholesterol-containing plaques.

Figure 2–11 Fetal alcohol syndrome in a 21-month-old girl. There is a hypoplastic upper lip, micrognathia, absent philtrum, and short palpebral fissure. The cardiac malformation was ventricular septal defect with pulmonary valve stenosis. (Courtesy of Dr David A. Ferry, Encino, CA.)

Xanthalasma reside near the inner canthus and are best seen when the eyelids are gently closed. Their presence, especially in young adults, is accompanied by hypercholesterolemia and premature atherosclerotic coronary artery disease. Lid-lag, another ocular feature of hyperthyroidism (see above), is characterized by sclera visible above the iris as movement of the upper lid lags behind the eyeball during downward gaze. Ptosis, often asymmetric, is part of the external ophthalmoplegia and pigmentary retinopathy of Kearns–Sayre syndrome (Fig. 2–12), which is associated with infranodal conduction abnormalities culminating in complete heart block.[13] In sarcoidosis, enlarged lacrimal glands conspicuously protrude the lateral margins of the upper lids (Fig. 2–13). Eversion of the lid permits direct inspection of the enlarged lacrimal glands.[14]

The **conjunctivae** are best examined with a pocket flashlight as the lower lid is retracted downward while the patient looks up. The pallor of anemia or the suffusion of cyanosis is then apparent. White-centered lesions of infective endocarditis are located on the conjunctiva of everted lower lids (Fig. 2–14*A*). Conjunctivitis with urethritis points to the diagnosis of Reiter's disease. Acute cardiac involvement takes the form of pericarditis and abnormalities in atrioventricular (AV) conduction, while chronic involvement is represented by AV block and aortic regurgitation.[15]



A B

Figure 2–12 An eighteen-year-old girl with Kearns–Sayre syndrome and typical asymmetric ptosis. She also had pigmentary retinopathy. Cardiac involvement took the form of bifascicular block that required a pacemaker.

The sclera can disclose discoloration as familiar as jaundice, or as unusual as the blue sclera of osteogenesis imperfecta.[16]

In young patients, a corneal arcus (Fig. 2–15) prompts suspicion of hypercholesterolemia and premature coronary artery disease. In Down syndrome, the iris may reveal distinctive Brushfield spots, a circle of punctate, depigmented dots circumferentially arranged at the periphery (Fig. 2–16*A*). Brushfield spots cannot be seen in patients with darkly pigmented irises. Congenital coloboma or fissures of the iris cause a change in pupillary shape from circular to oblong, creating the appearance of a cat's eye[17] (Fig. 2–17).

The size, shape, and reaction of the pupils to light and accommodation are diagnostically important. In diabetes mellitus, the pupils are sometimes unequal and nonreactive to light. The distinctive Argyll Robertson pupil, which is small, irregular, and usually constricts in response to convergence accommodation but not to light, is a feature of neurosyphilis (tabes dorsalis), often accompanied by aortic regurgitation.[18]

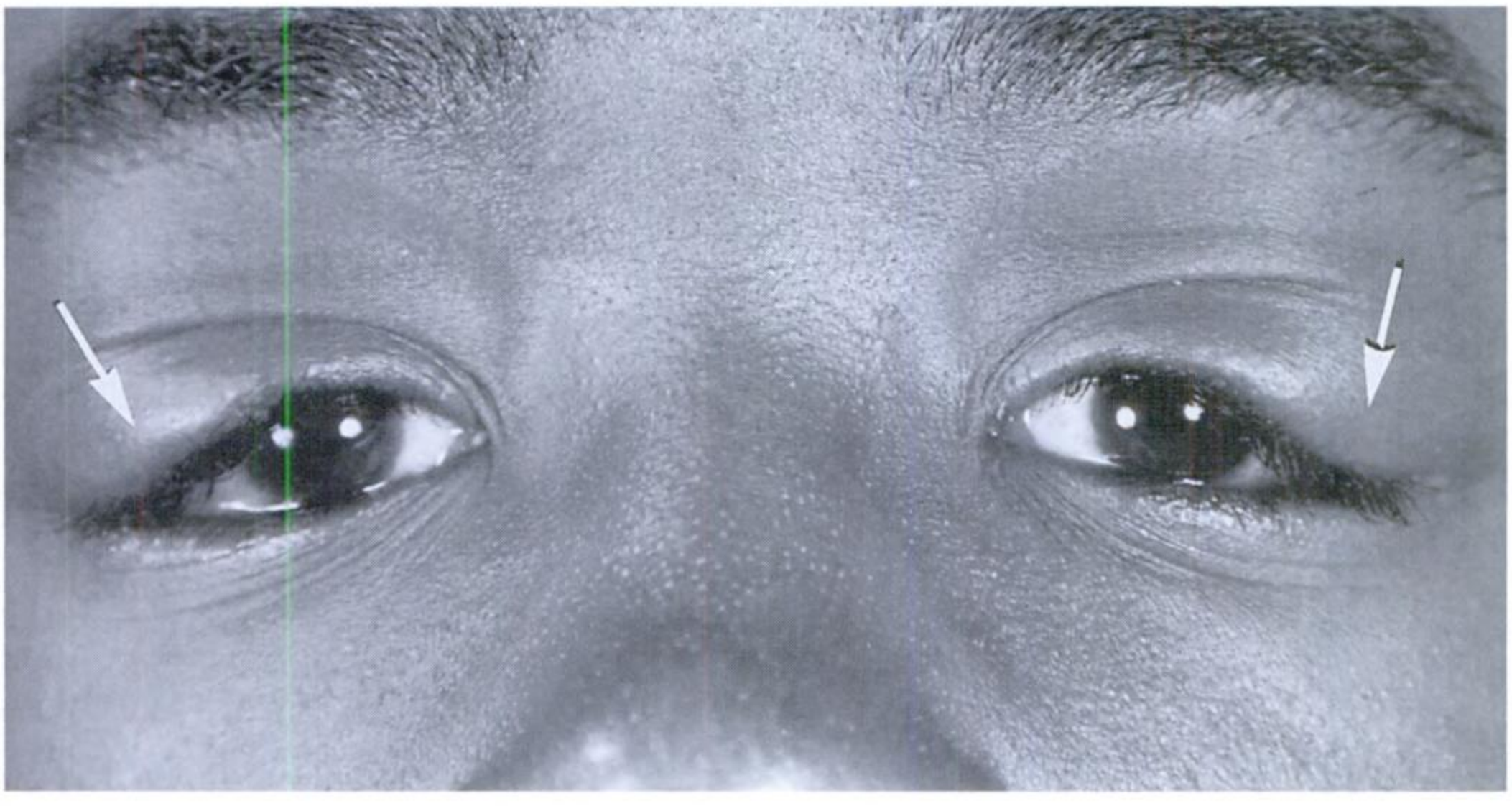
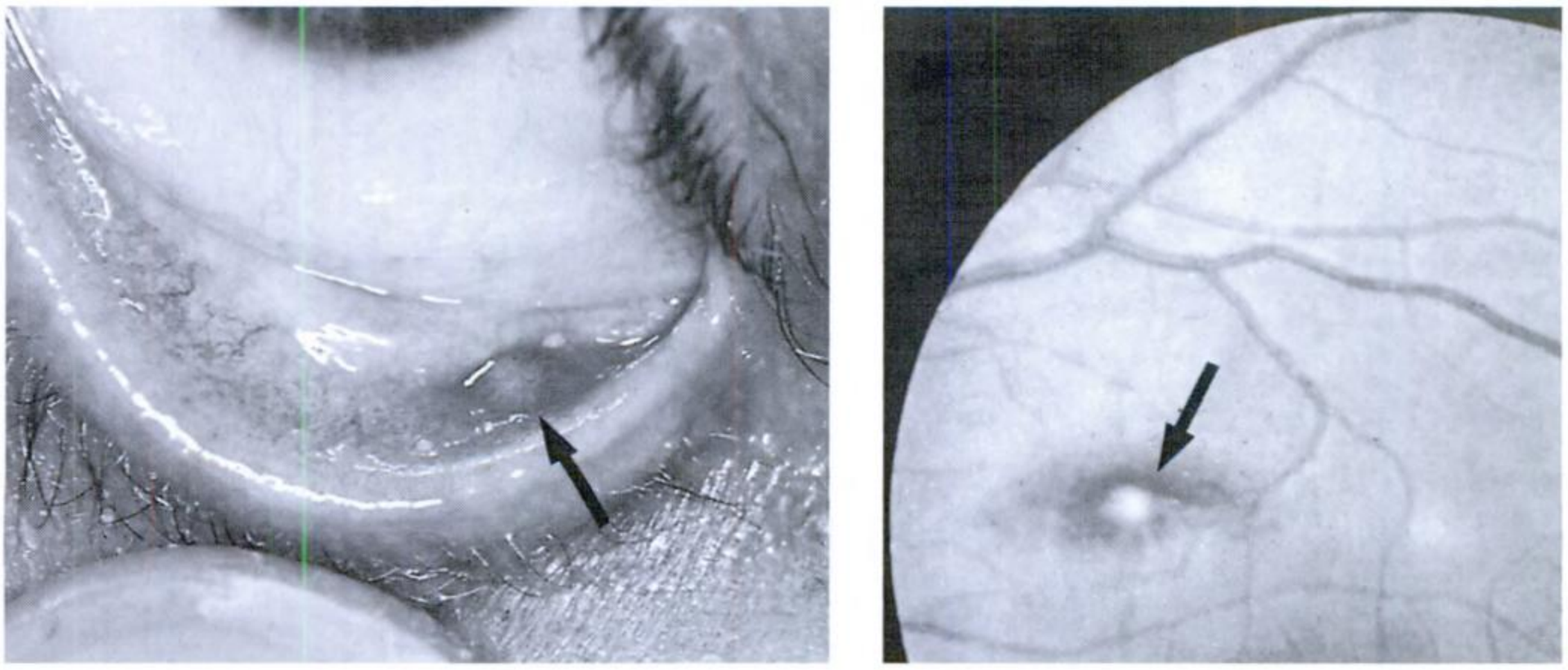


Figure 2–13 Young boy with sarcoidosis and typical enlarged lachrymal glands that caused protrusion of the lateral aspects of the upper lids (*arrows*). Pulmonary hypertension and prolonged atrioventricular conduction coexisted.



A B

Figure 2–14 *A,* Eversion of the lower lid discloses a typical white-centered conjunctival lesion (*arrow*) of infective endocarditis. *B,* Retinal Roth spot of infective endocarditis with white center surrounded by hemorrhage (*arrow*).

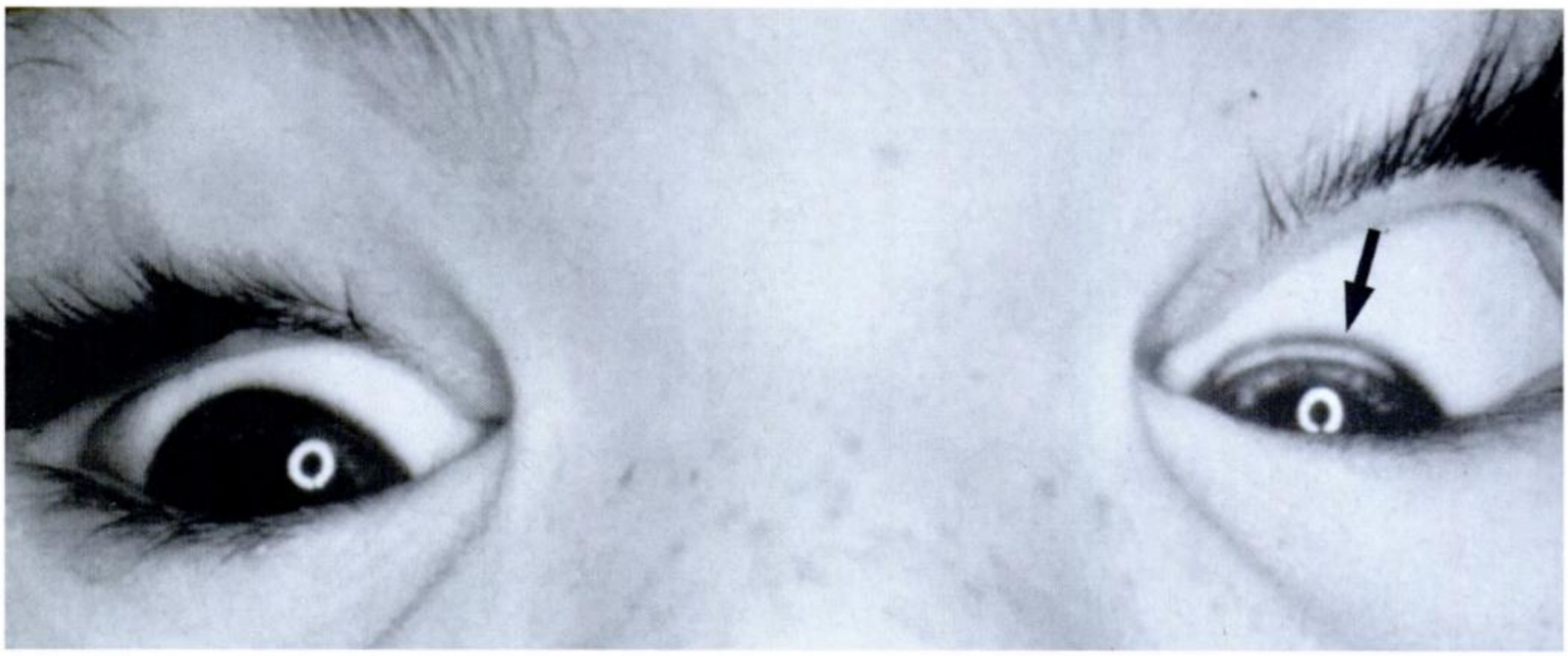
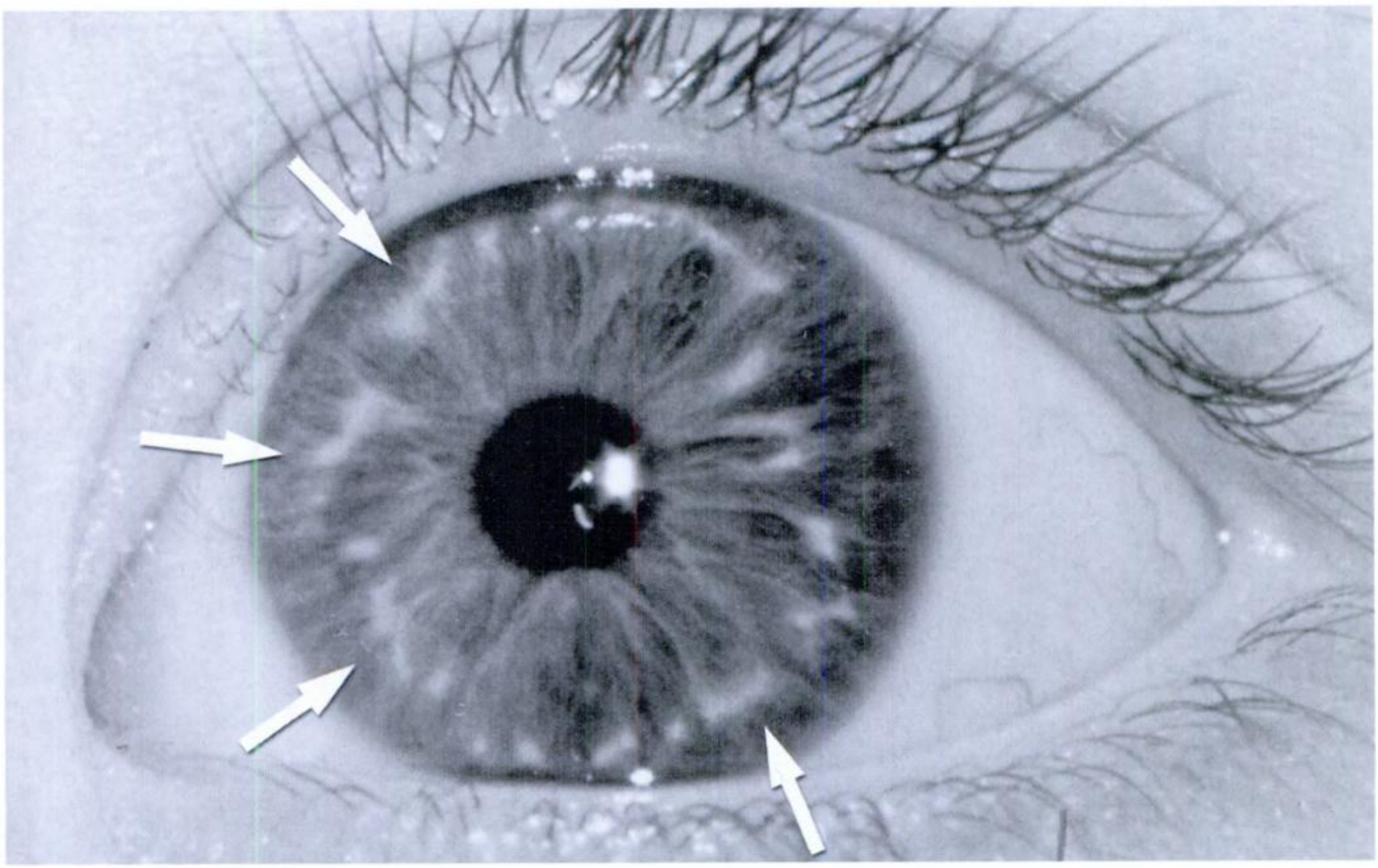


Figure 2–15 Left corneal arcus (*arrow*) in a 4-year-old girl with homozygous familial hypercholesterolemia.

A common abnormal appearance of the **lens** is the cataract. Premature cataracts occur in myotonic muscular dystrophy (Fig. 2–7), in Werner syndrome (Fig. 2–6*A*), and in offspring of mothers with first-trimester rubella.[5] Subluxation of the lens is an important feature of the Marfan syndrome[3] (Fig. 2–18) and homocystinuria. The unsupported lens imparts a distinctive shimmer to a transverse light beam when lateral motion of the eye is abruptly halted, an observation best made in a darkened room. In Marfan syndrome, lens subluxation is typically upward (Fig. 2–18), whereas in homocystinuria, downward dislocation is the rule. Homocystinuria is accompanied by systemic arterial and venous thromboses, myocardial infarction, and pulmonary embolism.

Examination of the **internal appearance** of the eye—retina, retinal arteries, retinal veins, and optic disc—requires an ophthalmoscope, the skilled use of which is a necessary part of the cardiac and vascular physical examination. The optic fundi provide visual access to small arteries, arterioles, veins, venules, and capillaries. In hypertensive retinopathy, permanent or relatively permanent changes consist of increased light reflex, copper wire appearance, arteriovenous nicking, and arteriolar tortuosity with irregular caliber. Changes that tend to disappear with control of blood pressure include hemorrhages, exudates, cotton wool patches, and papilledema. Less well-known in hypertensive patients is the distinctive appearance of retinal arteries when the hypertension is due to coarctation of the aorta.[5] The retinal arteries show "U turns" (Fig. 2–19) but do not exhibit hypertensive retinopathy.[5]

Retinal arterial emboli have characteristic appearances that may reveal their sources. Calcific emboli tend to be white, dull, and in close proximity to the disc margin. Cholesterol emboli (Hollenhorst plaques) are highly refractile.[19] Fibrin-platelet emboli are whitish plugs that are sometimes seen moving through the retinal arteries. In cyanotic congenital heart disease and at high altitude, there is an increase in retinal vascularity

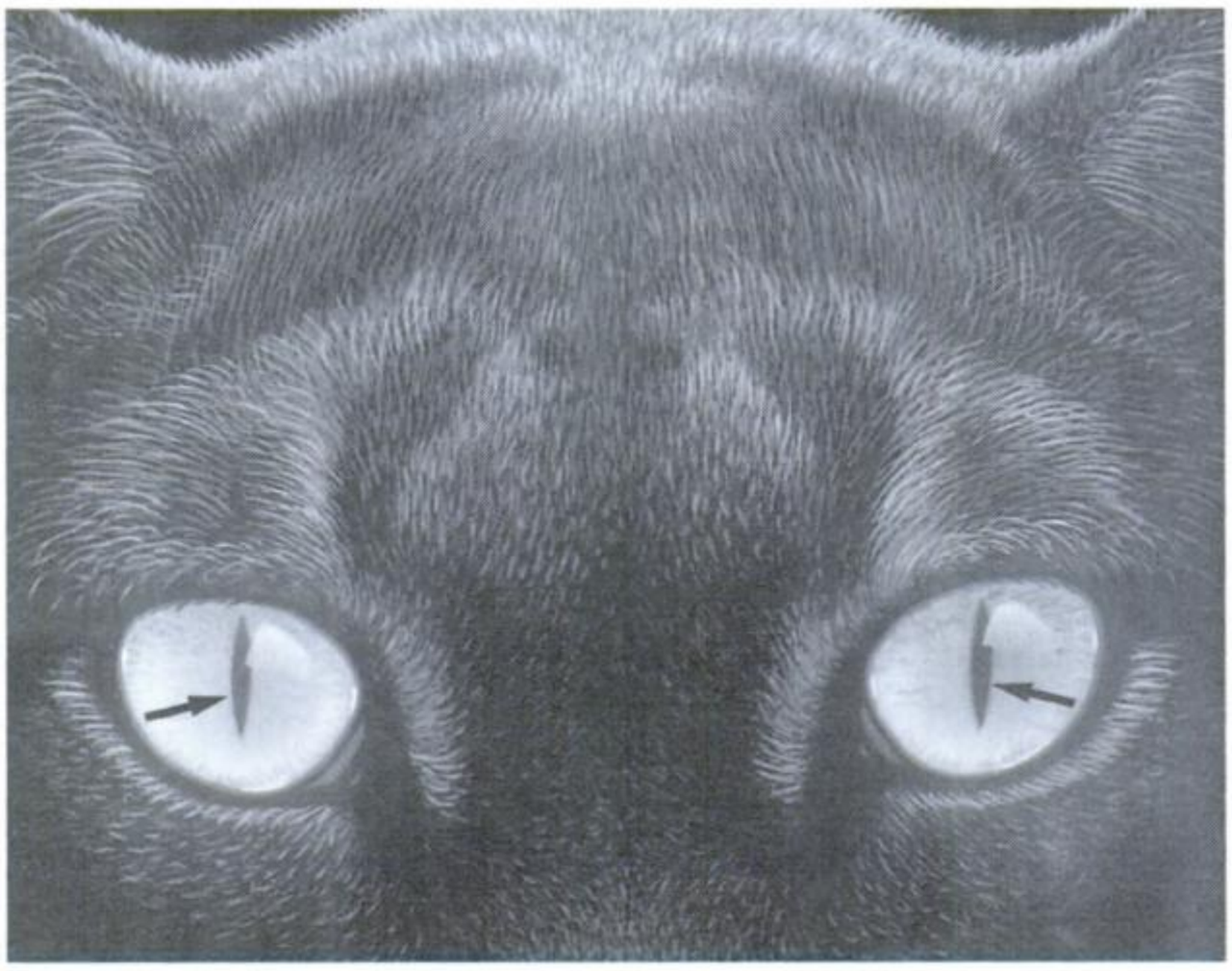


A

B

Figure 2–16 *A,* Brushfield spots consisting of depigmented foci (*arrows*) along the outer circumference of the iris together with sparse, thin eyelashes in a child with Down syndrome and an atrioventricular septal defect. *B,* Simian crease (*arrow*) traversing the palm of a 6-month-old child with Down syndrome and an atrioventricular septal defect.

A



B

Figure 2–17 *A,* Painting by Lyonel Feininger, an American-born German Expressionist (photographed by the author). The distinctive oblong shape of the pupils creates the appearance of a "cat's eye" (*arrow*), suggesting that the model had congenital coloboma (fissures) of the irises. In the original painting, the cheeks had the reddish blue hue of cyanotic congenital heart disease, probably Fallot's tetralogy, a malformation that coexists with the cat's eye syndrome. *B,* Oblong pupils of a cat's eye *(arrows).*

and an increase in vascular caliber probably due to vascular endothelial growth factor elaborated in response to hypoxemia. Exceptionally, papilledema and retinal edema occur in response to low systemic arterial oxygen saturation.

Retinal Roth spots that accompany infective endocarditis have a white, cotton wool appearance (perivascular collections of lymphocytes in the nerve layer) surrounded by hemorrhage (see Fig. 2–14*B*). Bilateral atypical pigmentary retinopathy is a feature of Kearns–Sayre syndrome mentioned earlier.[13] Retinal detachment is an occasional feature of the Ehlers–Danlos syndrome, in addition to angioid streaks that radiate from the optic discs as reddish brown or gray lines that are conspicuously wider than the retinal vessels. Angioid streaks also occur in pseudoxanthoma elasticum, Paget bone disease, and sickle cell anemia.

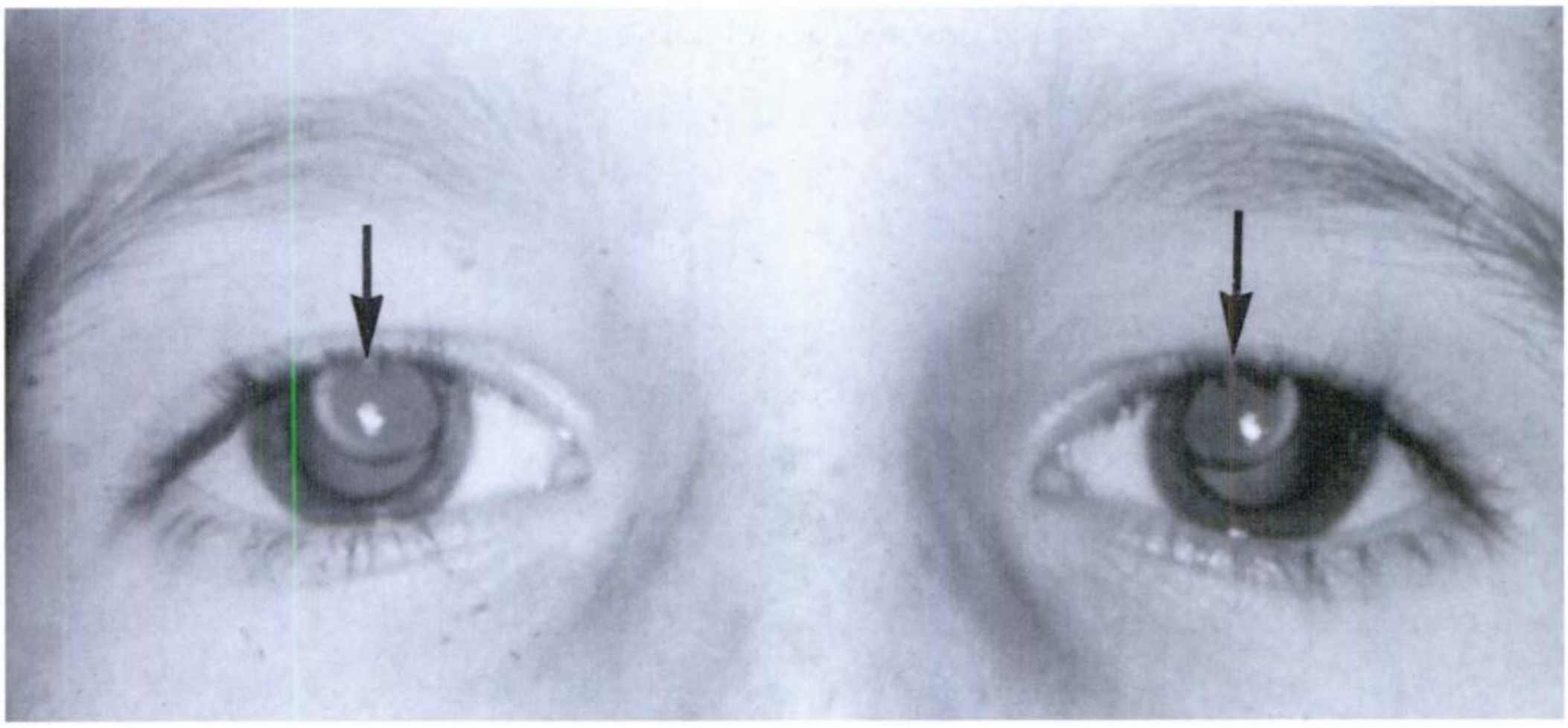


Figure 2–18 Typical *upward* dislocation of the lenses *(arrows)* in a young woman with Marfan syndrome.

External and Internal Appearance of the Mouth

Examination of the mouth includes the lips, mucous membranes, teeth, gums, tongue, and palate. The color of the lips may disclose the pallor of anemia or the discoloration of cyanosis. Thick lips are features of Hurler's syndrome (see Fig. 2–8 mentioned earlier). Attention has already been drawn to the absent philtrum of the fetal alcohol syndrome (see Fig. 2–11).

Mucous membrane lesions are best examined by everting the upper and lower lips, and by examining the mucosa of the oral cavity with a flashlight. Petechiae of infective endocarditis can be identified. In hereditary hemorrhagic telangiectasia (Rendu–Osler–Weber syndrome), clusters of tiny ruby patches appear on the lips, oral mucous membranes, palate, and tongue (Fig. 2–20), and are associated with pulmonary arteriovenous fistulae. These mucous membrane lesions were well described in an annotation from the *Lancet*:

> Every large general hospital is certain to have on its list of frequent attenders a small group of unfortunate adults who come to the casualty department complaining of recurrent bleeding from the nose, lips or mouth. The blood is seen to stem from an insignificant leak in the center of a small ruby patch, many of which are usually to be found gathered here and there on mucous membranes . Although the flow of blood is seldom vigorous, it may eventually by its persistence, draw some concern. Its arrest can be infuriatingly difficult. Each ruby patch marks the position of a tiny arteriovenous communication at the capillary level. Whatever the cause, they can be induced to bleed by the most trivial of injuries.[20]

An important aspect of the examination of the mouth is the oral hygiene of teeth and gums in patients with cardiac or vascular lesions that are substrates for

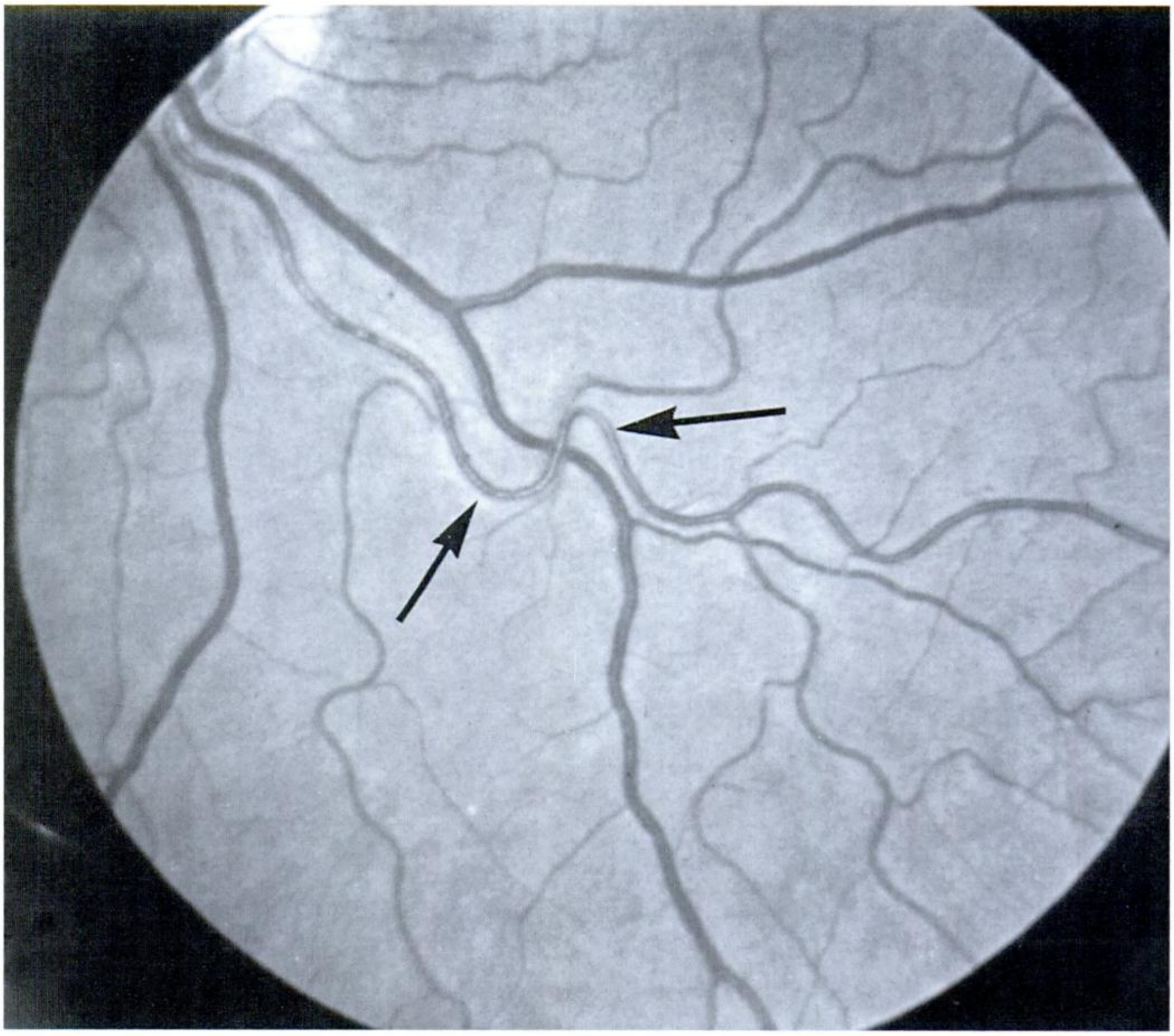


Figure 2–19 Retina of a 28-year-old woman with coarctation of the aorta. *Arrows* identify typical U-shaped turtousity of a retinal artery. Hypertensive retinopathy was conspicuously absent.

infective endocarditis. Malformations of the teeth are diagnostically important in Williams syndrome, the nonhereditary form of supravalvular aortic stenosis with pulmonary artery stenosis (see Fig. 2–10). Peg teeth are common in Hurler's syndrome (gargoylism) (see Fig. 2–8). An even more distinctive malformation of the teeth is a feature of the Ellis–van Creveld syndrome in which abnormal, prematurely erupted teeth are present at birth, in addition to gingival hypertrophy and multiple frenuls.[5] Phenytoin, which is occasionally used as an antiarrhythmic agent, may cause gingival hyperplasia.

The appearance of the **tongue** should be judged according to size, texture, position, and surface lesions. A large, protruding tongue is a feature of Down syndrome (see Fig. 2–9) and Hurler's syndrome (Fig. 2–8) mentioned above. An odd variation is

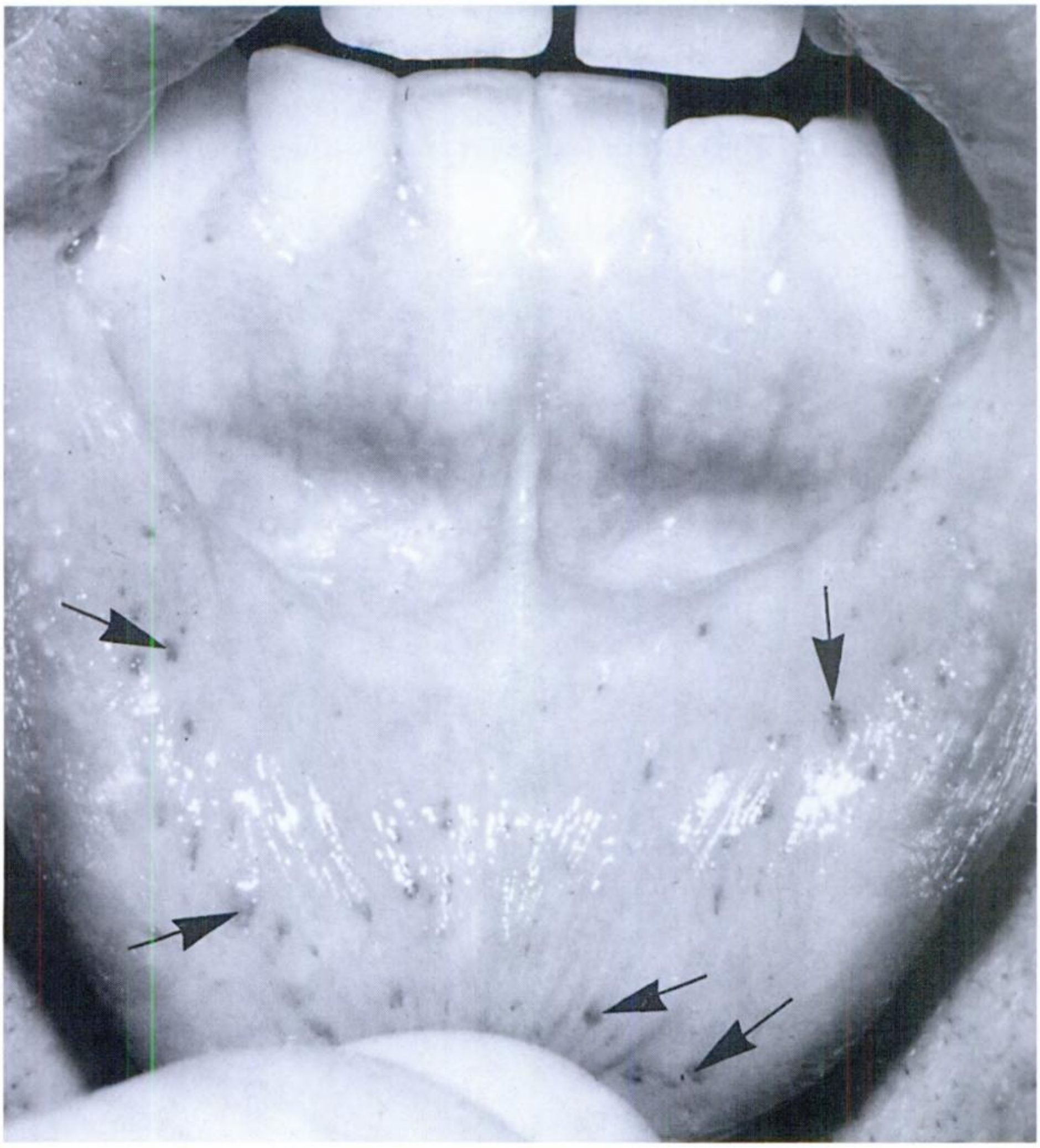


Figure 2–20 A 28-year-old man with Rendu–Osler–Weber hereditary hemorrhagic telangiectasia. Clusters of ruby patches are on his everted lower lip (*arrows*). Bilateral pulmonary arteriovenous fistulae were present.

retraction of the tongue—glossoptosis—in the Pierre Robin syndrome. The retraction may cause upper airway obstruction and pulmonary hypertension.[21] Macroglossia is an important albeit infrequent feature of primary amyloidosis which is accompanied by restrictive cardiomyopathy.

Examination of the hard palate may disclose innocuous *torus palatinus*, a bony protuberance or ridge found at the junction of the intermaxillary and transverse palatine sutures. A distinctive high-arched palate is a feature of Mafan syndrome.

The Hands and Feet

After the face, the hands are the most expressive parts of the human body. A thumb capable of apposing each finger was a major step forward in mammalian evolution. Strictly speaking, the hand is equipped with four fingers (triphalangeal digits) and one thumb (a biphalangeal digit). For the purpose of this discussion, the hands and feet will be considered together. Their appearance is assessed in terms of color and texture of the skin, structure, and the presence of specific lesions.

An abnormality of color is represented by *cyanosis*, the *blue condition* from the Greek *kyanos, blue*, and *osis, condition*. Cyanosis accompanied by clubbing (Fig. 2–21) is a feature of a right-to-left shunt in cyanotic congenital heart disease. In older adults, digital clubbing may accompany cyanotic cor pulmonale of chronic obstructive lung disease.

Digital clubbing (Fig. 2–21) differs from an increase in the convexity of otherwise normal nails.[22] Clubbing requires sufficient elevation of the *base* of the nail to eliminate the angle with the contiguous soft tissue (Fig. 2–21). Clubbing therefore consists not only of an

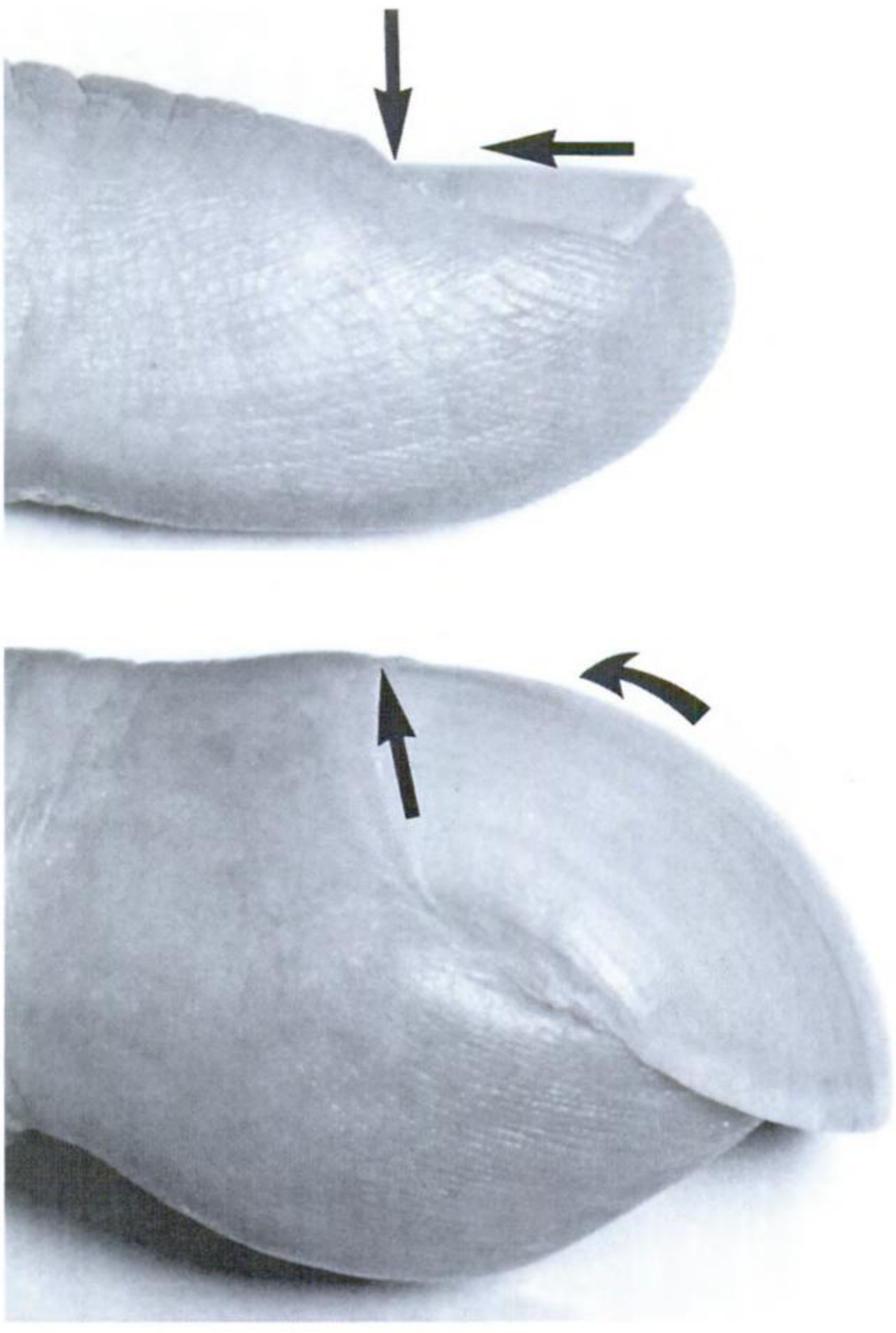


Figure 2–21 *A,* Profile of normal finger. *B,* Profile of typical clubbing of cyanotic congenital heart disease. The dorsum of the nail is strikingly convex (*curved arrow*), and the angle at its base is absent (*vertical arrow*).

exaggeration of the normal convexity of the nail, but an extension of that convexity to include the *base* of the nail. Clubbing is best detected by observing the digit in profile (Fig. 2–21). The elevated base of the nail has a spongy texture detected by applying light pressure.

Cyanosis and clubbing are diagnostically important not only because of their presence and degree, but also because of their *distribution.* When equally represented in the hands and feet, the causative right-to-left shunt is proximal to the brachiocephalic arteries. Conversely, *differential* cyanosis and clubbing refers to cyanosis and clubbing of the feet but not the hands. The diagnostic implication of differential cyanosis is a right-to-left shunt that delivers unoxygenated blood from the pulmonary trunk into the aorta *distal* to the left subclavian artery, so the toes are affected but the hands are spared (Fig. 2–22).

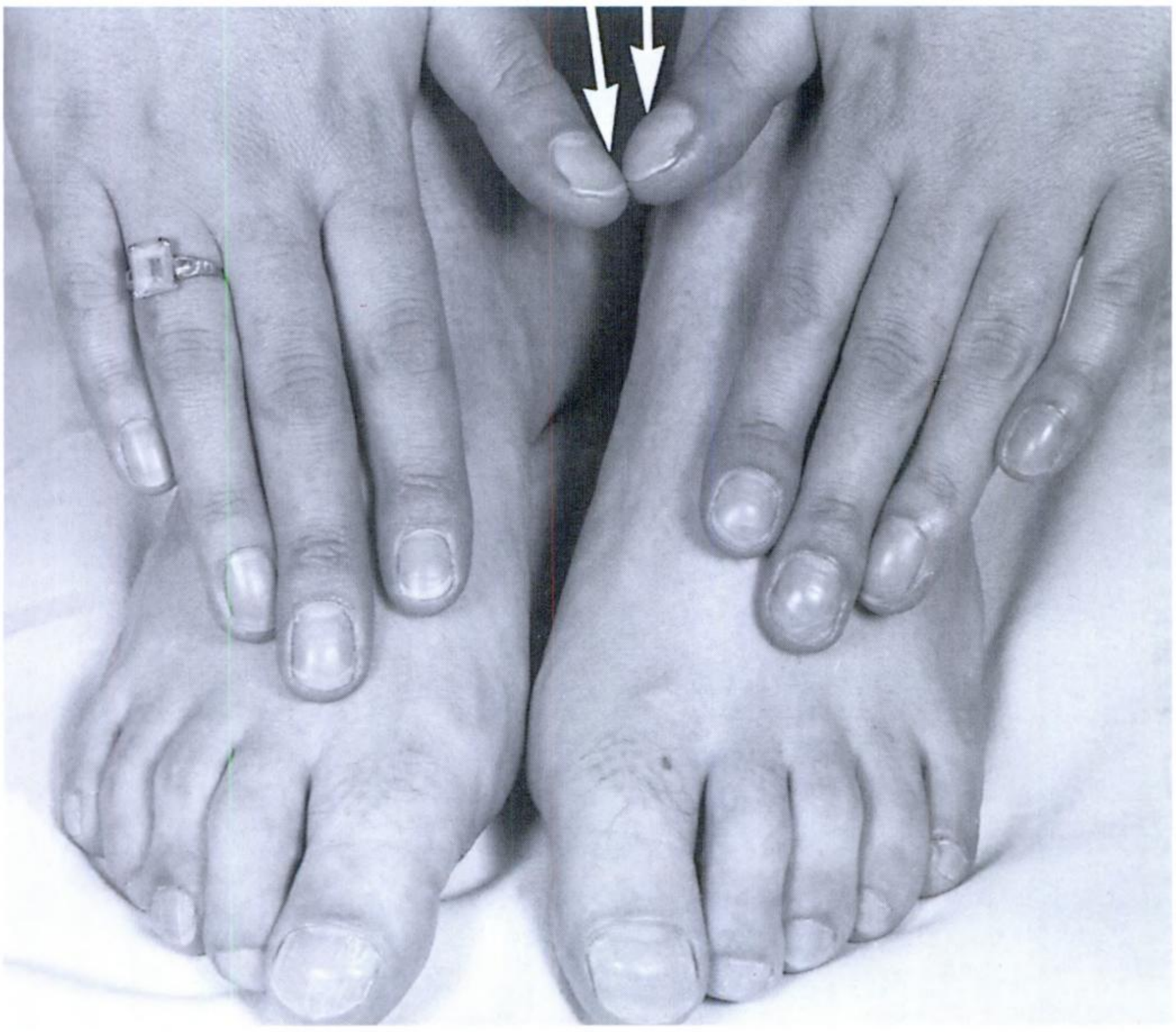


Figure 2–22 Differential cyanosis in a 28-year-old woman with patent ductus arteriosus, pulmonary vascular disease, and reversed shunt. The hands were placed on the dorsa of the feet to compare the fingers and toes. Because the right-to-left shunt was immediately distal to *the left* subciavian artery, the spared right hand is acyanotic and the fingers are not clubbed, the left hand is mildly cyanotic and clubbed (*arrows*), and the toes are cyanosed and clubbed.

Patent ductus arteriosus with suprasystemic pulmonary vascular resistance and reversed shunt is the cause.[5] Differential cyanosis and clubbing are best identified when the patient sits with knees flexed and hands placed upon the dorsa of the feet (Fig. 2–22). Differential cyanosis is highlighted by exercise or immersion of the hands and feet a warm bath. Exercise increases the right-to-left shunt, and warmth increases skin blood flow.

Normal young females sometimes have *peripheral* cyanosis, especially of the feet, because of innocuous vasoconstriction. The feet are conspicuously cooler than the hands. Diagnostic error can be prevented by warming the hands and feet, an intervention that abolishes peripheral cyanosis but exaggerates central cyanosis, as just noted.

Clubbing of the digits and hypertrophic osteoarthropathy are responses to platelet-derived growth factor and transforming growth factor β, cytokines, and mitogens derived from the cytoplasm of shunted systemic venous megakaryocytes.

In patients with the protein-losing enteropathy of chronic constrictive pericarditis or a Fontan operation, discoloration may appear at the distal border of the fingers. *Terry nails* are characterized by a distal brownish band, with the proximal 80% of nail bed white[22] (Fig. 2–23). *Lindsay nails* associated with chronic renal disease are characterized by red, pink, or brown discoloration occupying approximately half the distal border of the nail bed.[22]

In peripheral vascular disease (see Chapter 3), the *texture and color* of the atrophic skin of the feet and toes is shiny, tightly drawn, and often interrupted by ischernic ulcers (Fig. 2–24). The ischemic foot is pallid when the leg is elevated and red (rubor) when the leg is dependent.

Raynaud phenomenon, sometimes seen in primary pulmonary hypertension, is characterized by intermittent alterations in color of the fingers, less commonly of the toes.

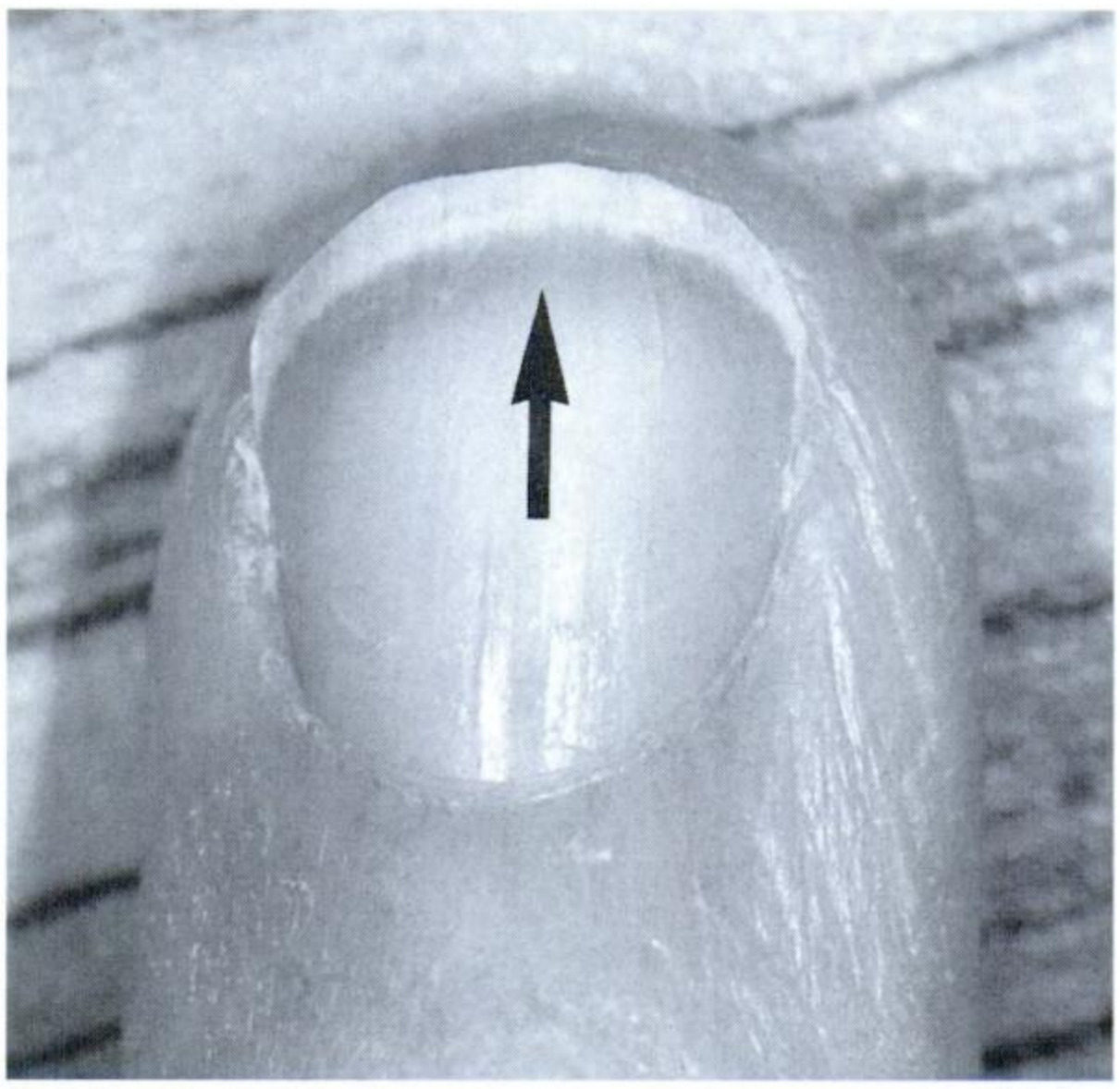


Figure 2–23 A brownish band 1- to 2-mm at the distal end of the nail bed (*arrow*). Terry nails is typically associated with *hypoalbuminemia*.

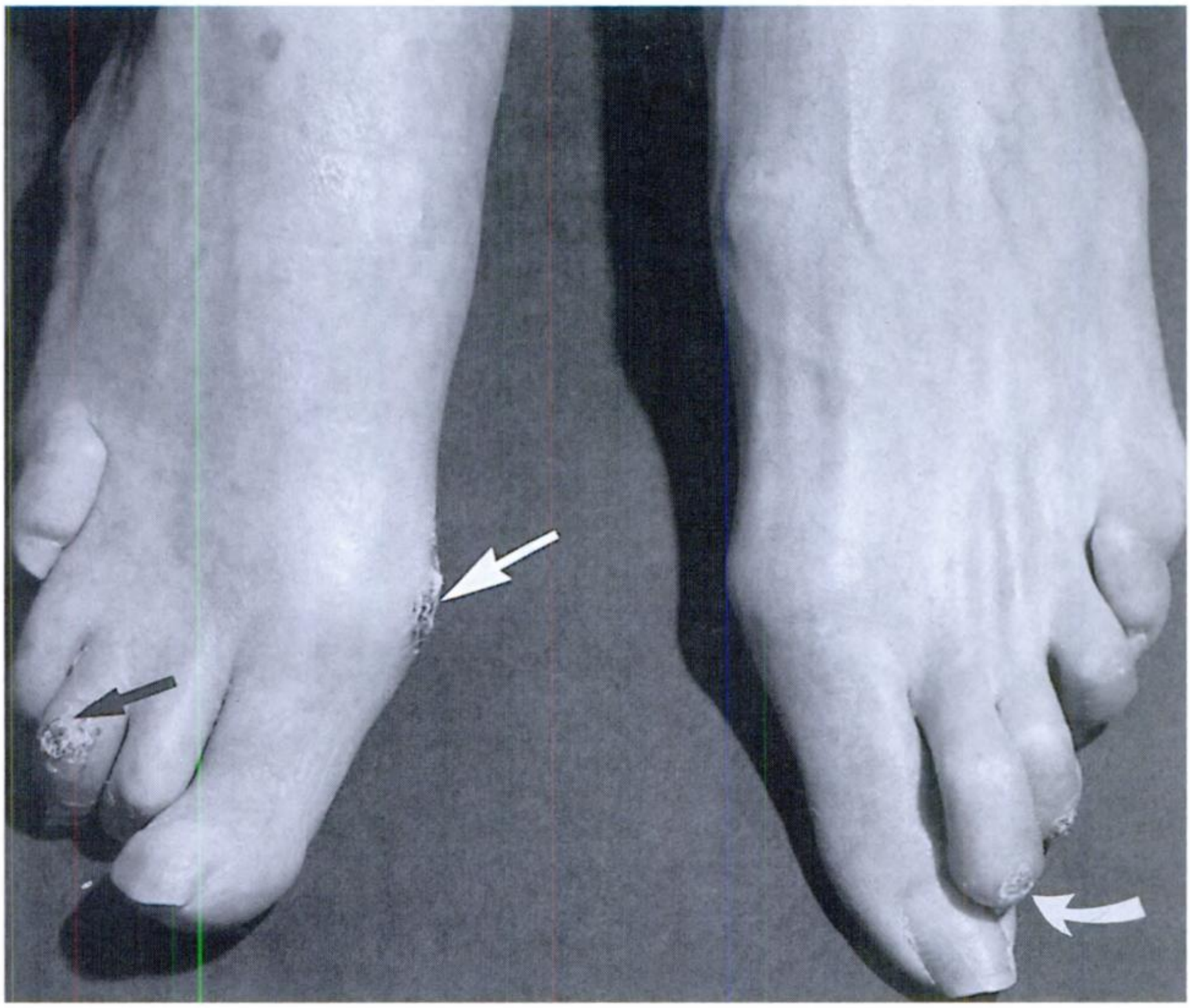


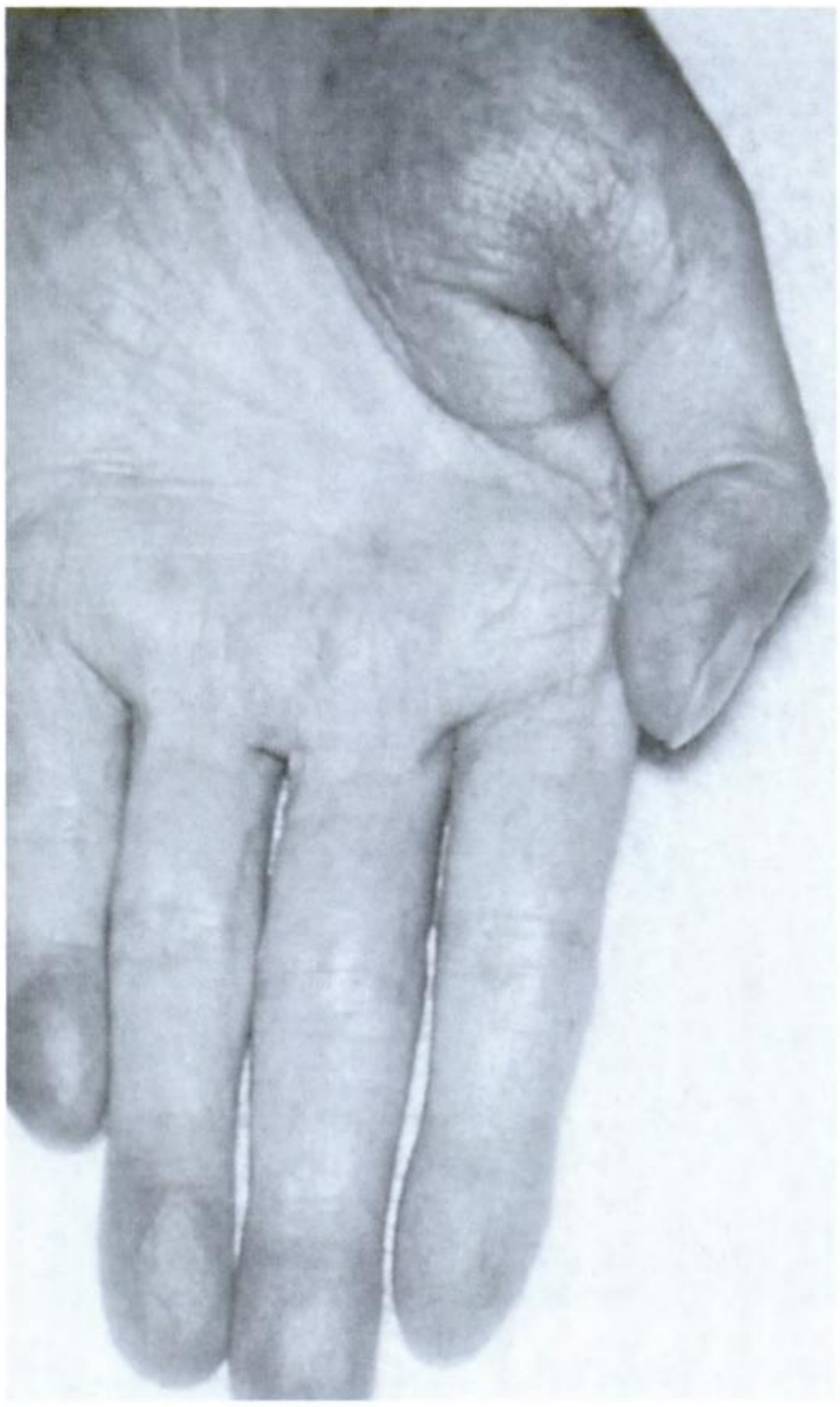
Figure 2–24 Atrophic, tightly drawn skin, ischemic ulcers (*black and white arrows*), and hypoplastic nails (*curved white arrow*) in patient with peripheral vascular disease associated with Werner syndrome and premature aging (see Fig. 2–6*A*). Note the ischernic loss of the tips of several toes.

The affected digits initially manifest intense pallor followed by cyanosis. Before restitution of normal color, the digits may develop intense rubor (reactive hyperemia).

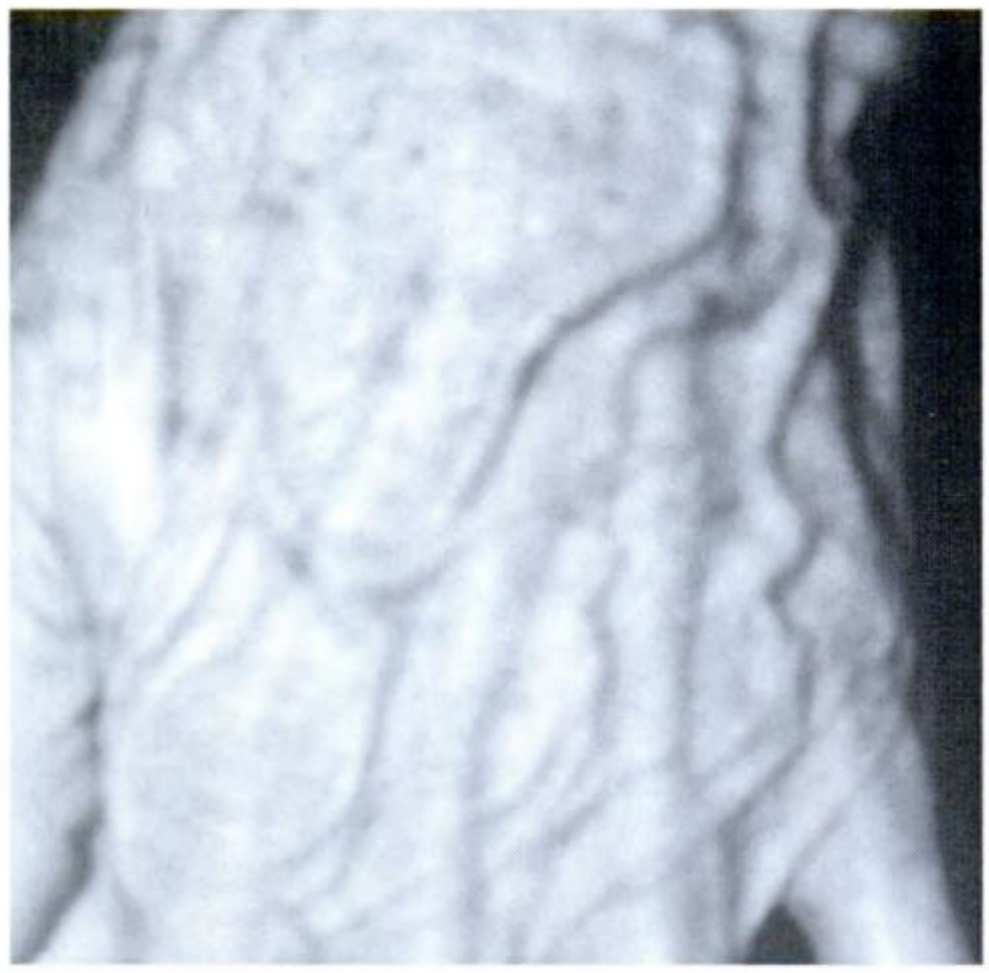
The postmyocardial infarction *shoulder–hand syndrome* is characterized by painless erythema of the palms and fingertips (Fig. 2–25*A*). The skin over the dorsa of the hands subsequently becomes swollen, tense, discolored, and wrinkled (Fig. 2–25*B*). The changes may culminate in Dupuytren contractures.[23] Pain, stiffness, and limitation of motion in the shoulder girdle, usually the left, may coexist.[23]

In progressive systemic sclerosis (scleroderma), which is accompanied by interstitial pulmonary fibrosis and pulmonary hypertension, the skin of the dorsa of the fingers is thickened with loss of the fine transverse creases (Fig. 2–26).

Abnormalities of the hands and feet may be sufficiently distinctive to establish a diagnosis and predict the coexisting cardiac disease. A prime example is Marfan syndrome, with its remarkable appearance of the hands[3] (Fig. 2–27). In osteogenesis imperfecta, another heritable disorder of connective tissue,[16] the joints of the hands are



A



B

Figure 2–25 *A,* Painless erythema of the hands and fingertips in a 56-year-old man with postmyocardial infarction shoulder–hand syndrome. *B,* Wrinkling of skin of the dorsum of the hand after a myocardial infarction.

hyperextensible but the fingers are not elongated (Fig. 2–28). Jaccoud's arthritis (sometimes mistaken for rheumatoid arthritis) is a postrheumatic fever abnormality in which the deformity can be voluntarily corrected because the cause is subluxation of the joints rather than erosion and fusion of articular surfaces[24] (Fig. 2–29).

The hands in Down syndrome are characterized by a simian (Latin *simia* ape) palmar crease, a distal triaxial radius, increased space between the fourth and fifth fingers, and a short fifth finger that is curved inward[5] (see Fig. 2–16*B*). The association of Down syndrome with atrioventricular septal defect was mentioned earlier. In Hurler's syndrome (see Fig. 2–8), inward curvature of the fourth and fifth fingers results in a claw-like appearance of the hands, which are wider than they are long.[10] Broad thumbs and toes are characteristic of the Rubinstein–Taybi syndrome[25] in which the coexisting cardiac malformation is usually patent ductus arteriosus.

Structural abnormalities of the hands and feet are sometimes represented by accessory digits or deficient digits. The extra digit is almost always an accessory finger as in Ellis–van Creveld syndrome, which is accompanied by common atrium (absence of

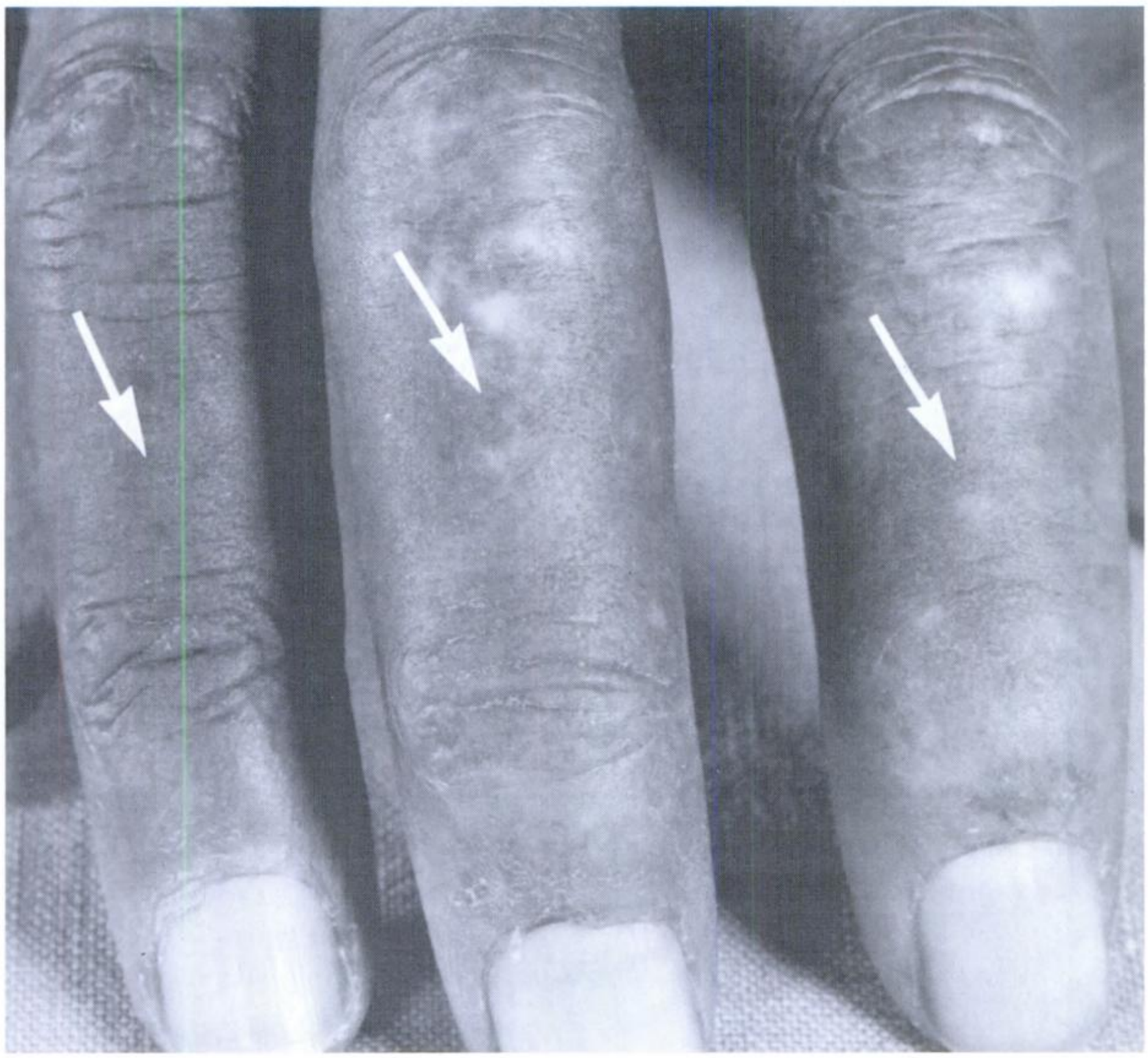
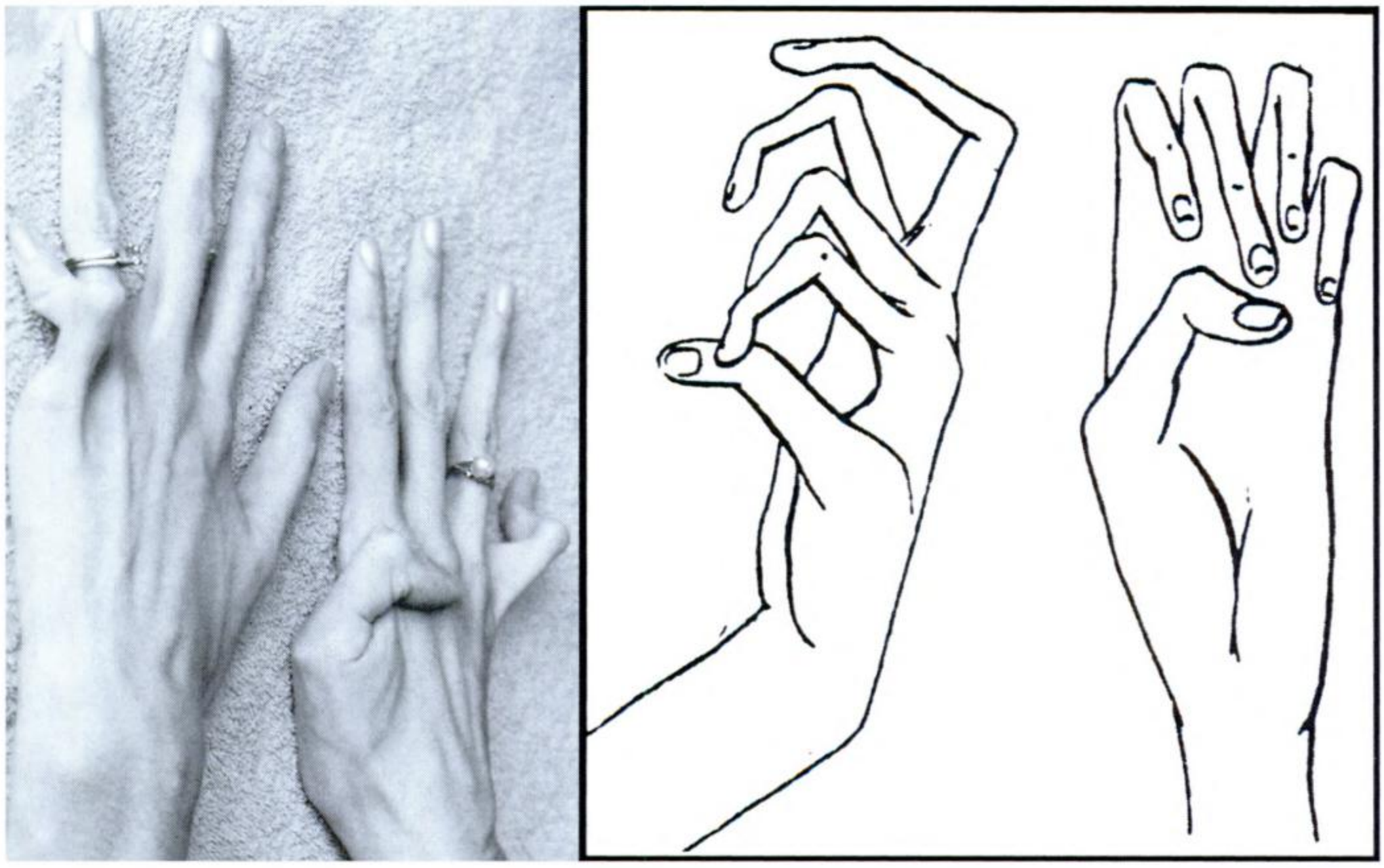


Figure 2–26 Hands of a 56-year-old woman with progressive systemic sclerosis (scleroderma). The skin of the dorsum of the fingers (*arrows*) is thickened and smooth, with loss of fine transverse creases.

the atrial septum), as mentioned earlier. Polydactyly of the hands is invariable in Ellis–van Creveld syndrome, but polydactyly of the feet occurs in only 10 percent of patients. The converse of polydactyly is a reduction in the number of digits, generally loss of the thumb, as in Holt–Oram syndrome.[26] The thumb may be present but hypoplastic with an accessory phalanx (triphalangial) that imparts a crooked appearance to the digit and makes apposition with fingertips difficult. The Holt–Oram abnormality becomes more obvious when the palm is supinated (turned up). The most common coexisting cardiac anomaly is an ostium secundum atrial septal defect.[5] Syndactyly is an innocuous variation in digital reduction, an autosomal disorder characterized by extension of interdigital webbing so that adjacent digits are fused. Syndactyly is the most common congenital anomaly of the fingers or toes and is of no special diagnostic significance.

Pes cavus with hammer toe (Fig. 2–30) is an abnormality of the foot in Friedreich ataxia[27] (see earlier). An even more distinctive and diagnostically useful structural



A B

Figure 2–27 *A,* Elongated, hyperextensible fingers of a young woman with Marfan syndrome. *B,* Illustration from Marfan's original report. (*Un cas de déformation congénitale des quatre membres plus prononcée aux extrémités caractérisée par l' allongement des os avec un certain degré d'amincissement.* Bull Mém Soc Méd Hôp Paris 13:220, 1896.)

abnormality is the "rocker-bottom" appearance of the foot caused by the protruding heel in trisomy 18, which is usually associated with patent ductus arteriosus or ventricular septal defect.[5]

Discrete lesions of the hands and feet may be subtle and unimportant, such as subungual splinter hemorrhages. Conversely, white-centered (actually yellowish gray) lesions are important physical signs of infective endocarditis (Fig. 2–31). Osler nodes are tender, pea-sized, raised areas on the pads of the fingers or toes (Fig. 2–31), on the thenar or hypothenar eminence, or on the soles of the feet. Osler nodes, while uncommon, are relatively specific for infective endocarditis. Not to be mistaken for Osler nodes are focal, discolored, tender, painful lesions on the tips of the fingers and toes caused by small emboli from prosthetic cardiac valves, left ventricular mural thrombi, or marantic endocarditis.

The Skin

Appearance of the skin is important in terms of color, texture, swelling (edema), and focal lesions. Evaluation of small focal lesions benefits from use of a magnifying glass or an ophthalmoscope as a means of magnification.

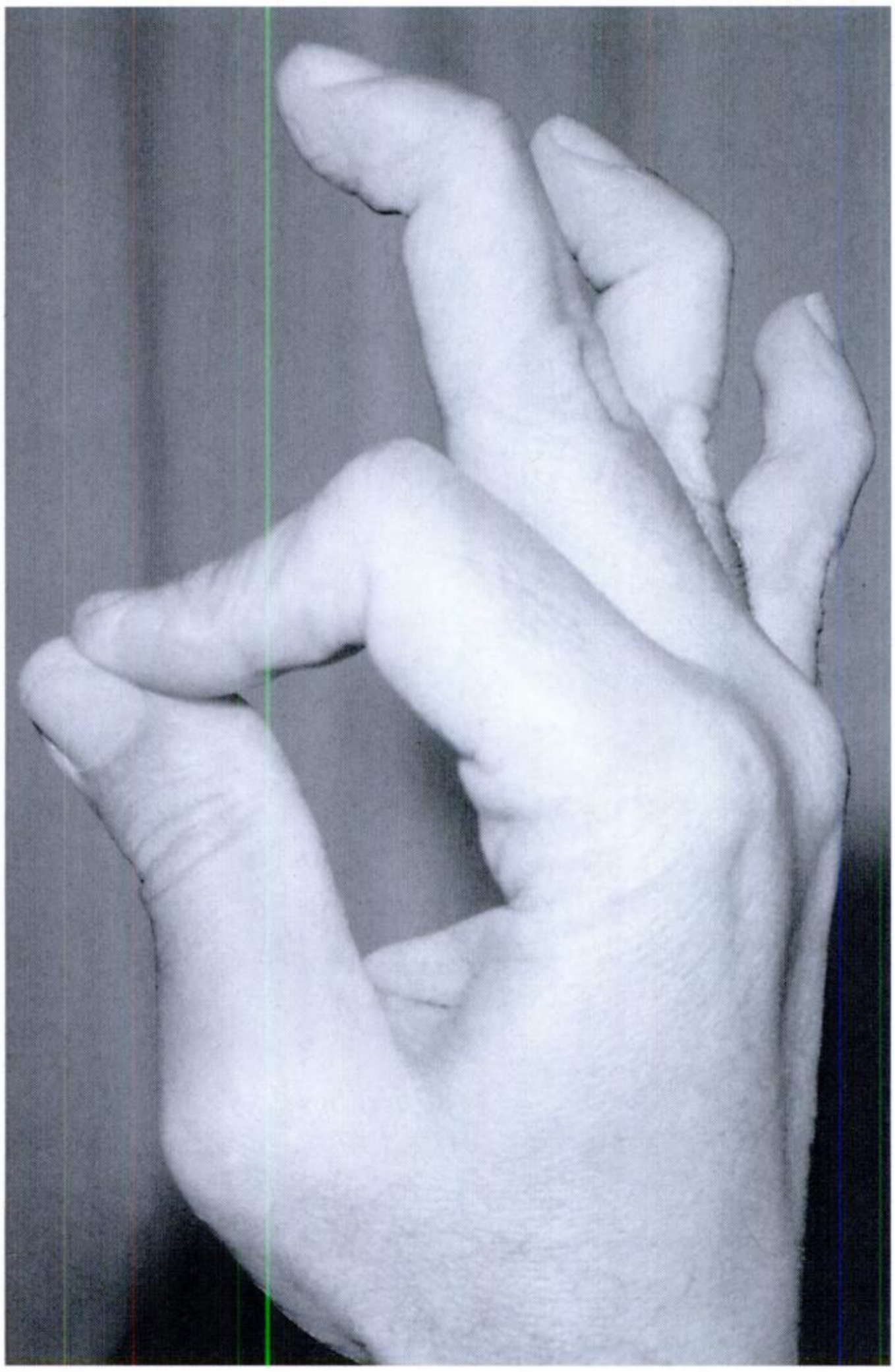
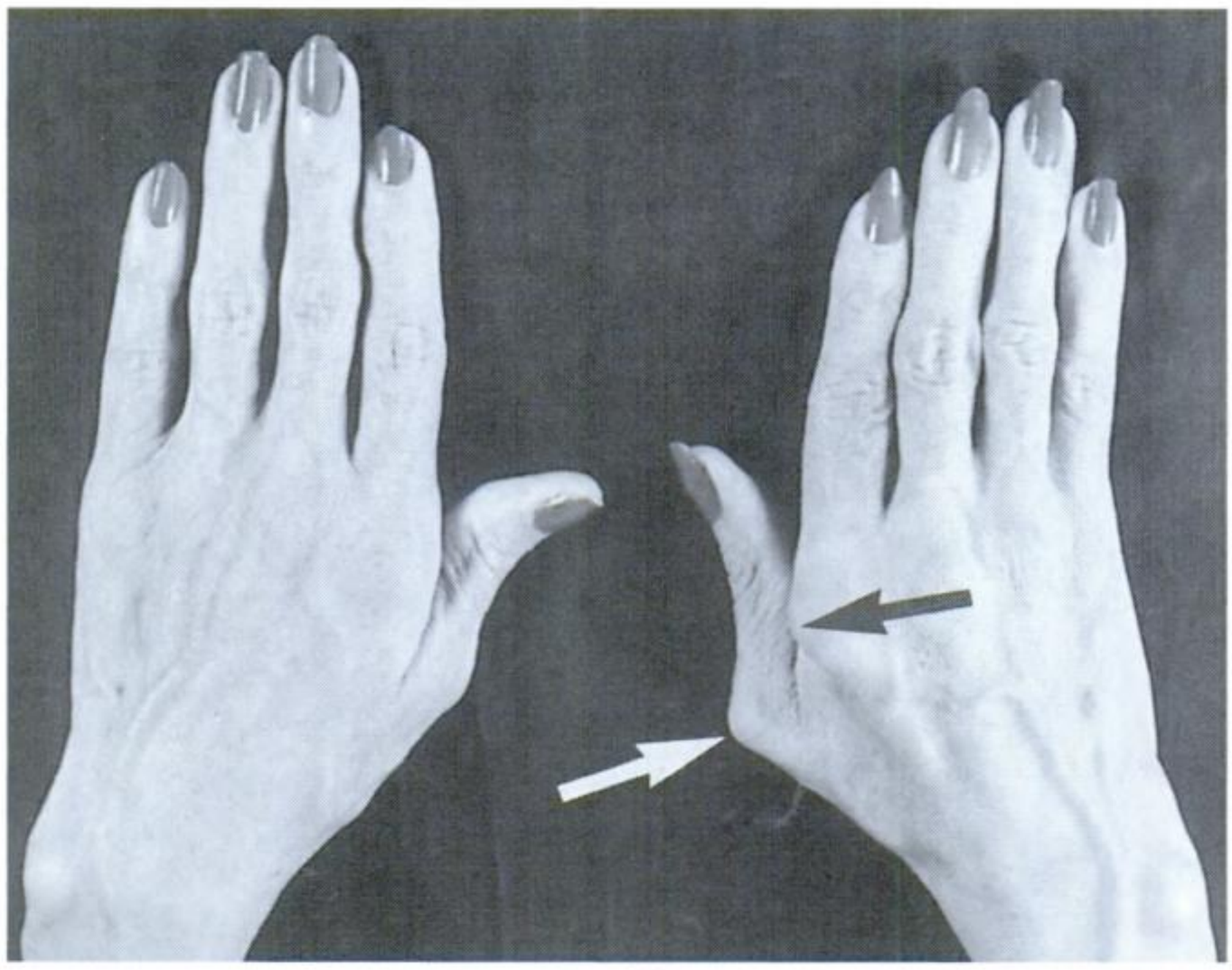
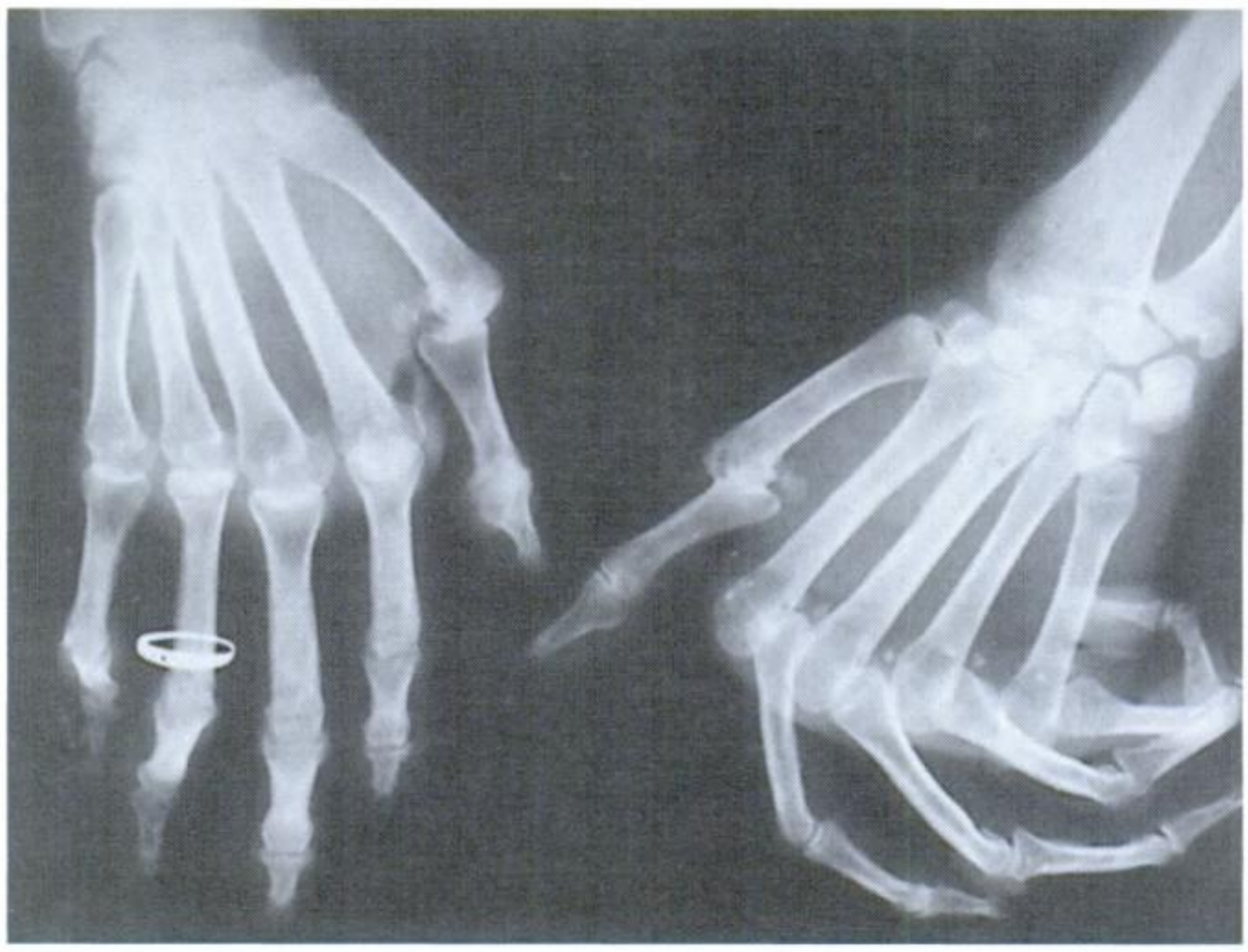


Figure 2–28 Hyperextensible but otherwise normal fingers in a young man with osteogenesis imperfecta and aortic regurgitation.

Cyanosis, apart from fingers, toes, and mucous membranes (see above), is detected in areas where the skin is thin, such as the ears, the tip of the nose, and the soles of the feet in infants. Cardiac implications of *jaundice* include chronic passive hepatic congestion, hemolysis from microangiopathic anemia of prosthetic cardiac valves, and from hemolysis following a large pulmonary infarct. Brawny induration of the legs and feet is a common consequence of the chronic peripheral edema of congestive heart failure. The bronze pigmentation of hemochromatosis is most prominent on exposed skin surfaces and is diagnostically important because of coexisting restrictive or dilated cardiomyopathy, which is potentially reversible by phlebotomy.[28] *Cafe au lait* spots (Fig. 2–32), freckles (especially axillary), and neurofibromas (skin-colored pedunculated tumors and subcutaneous nodules) are features of von Recklinghausen's disease that is accompanied by pheochromocytoma and hypertrophic cardiomyopathy. Symmetric vitiligo, especially



A



B

Figure 2–29 *A,* Hands of a patient with postrheumatic fever (Jaccoud) arthritis. The right hand shows typical flexion subluxation and u1nar deviation of the metacarpophalangeal joints (*arrows*). In the left hand, the deformity has been voluntarily corrected. *B,* Radiograph of the hands in Jaccoud arthritis. Rheumatic heart disease was manifested by mitral regurgitation.

of the distal extremities, is an occasional feature of Graves disease. In myxedema (see earlier), the texture of the skin is coarse, thick, and dry, with brittle, sparse head hair that doesn't sustain a curl. In Ehlers–Danlos syndrome (see above), the skin exhibits remarkable hyperextensibility, with a rubber-like response to stretch, and with cigarette paper scars that result from fragility and poor healing. In pseudoxanthoma elasticum (see above), the skin of the neck and axillae is reticular and telangiectatic with small tannish yellow papules that create the texture and appearance of a plucked chicken.

Subcutaneous edema, a time-honored hallmark of congestive heart failure, is typically pitting and dependent, involving the ankles and feet in the upright position

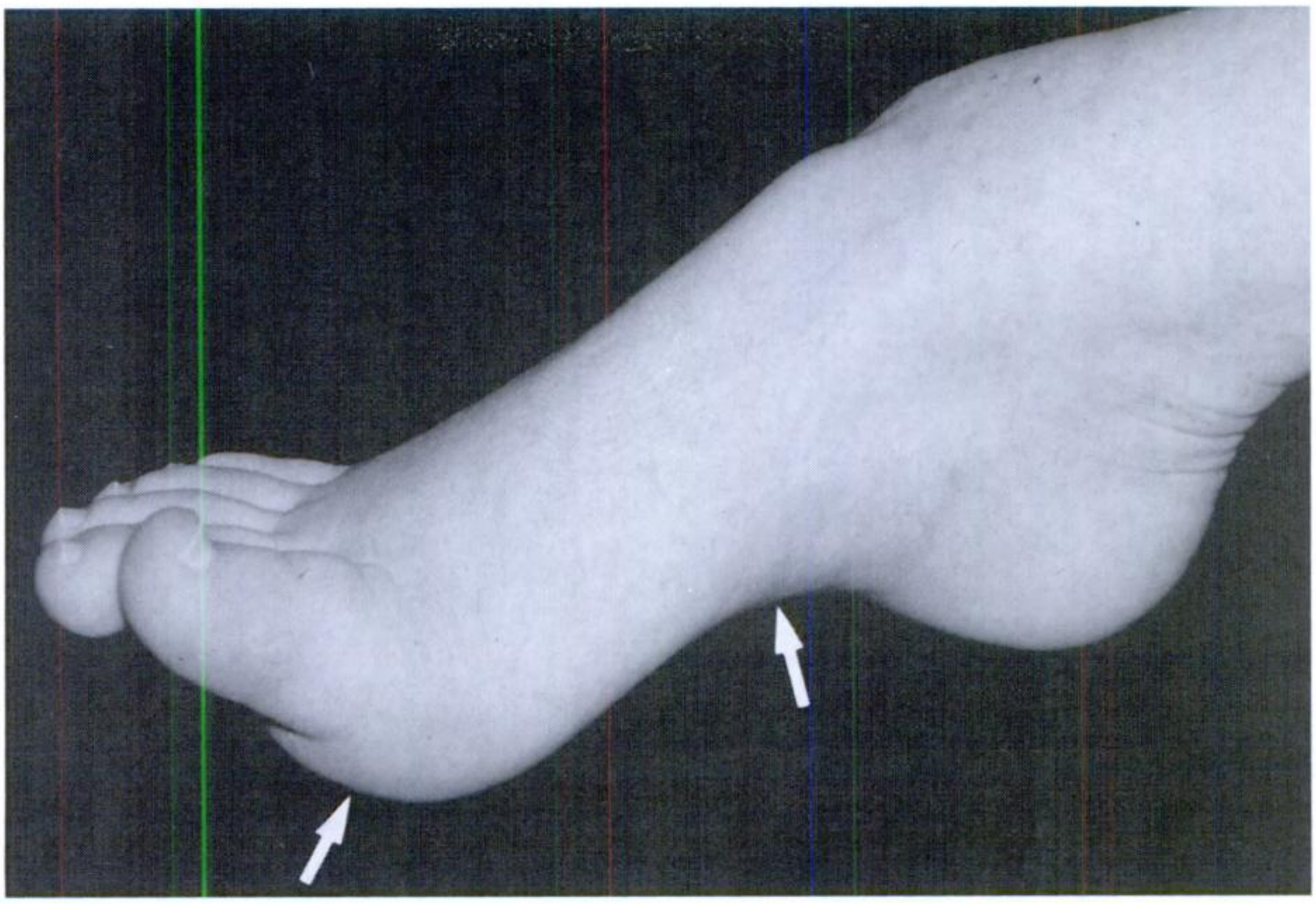


Figure 2–30 Pes cavus with hammer toe (*arrows*) in a patient with Friedreich ataxia and hypertrophic cardiomyopathy.

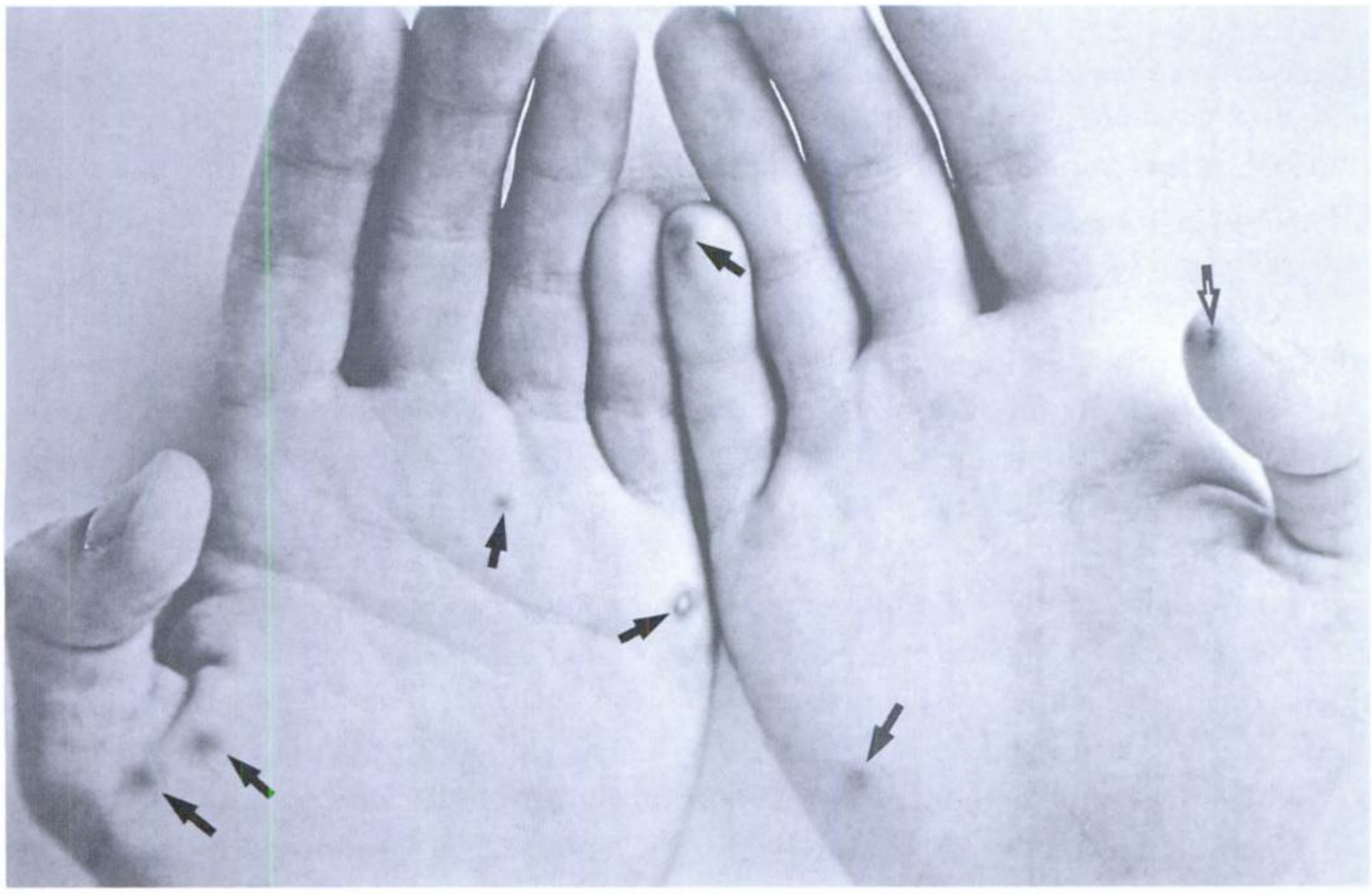


Figure 2–31 Typical white-centered (actually yellowish gray), nontender palmar lesions (*black arrows*) in infective endocarditis. The pea-sized lesion on the pad of the left thumb (*black/white arrow*) was a tender Osler node.

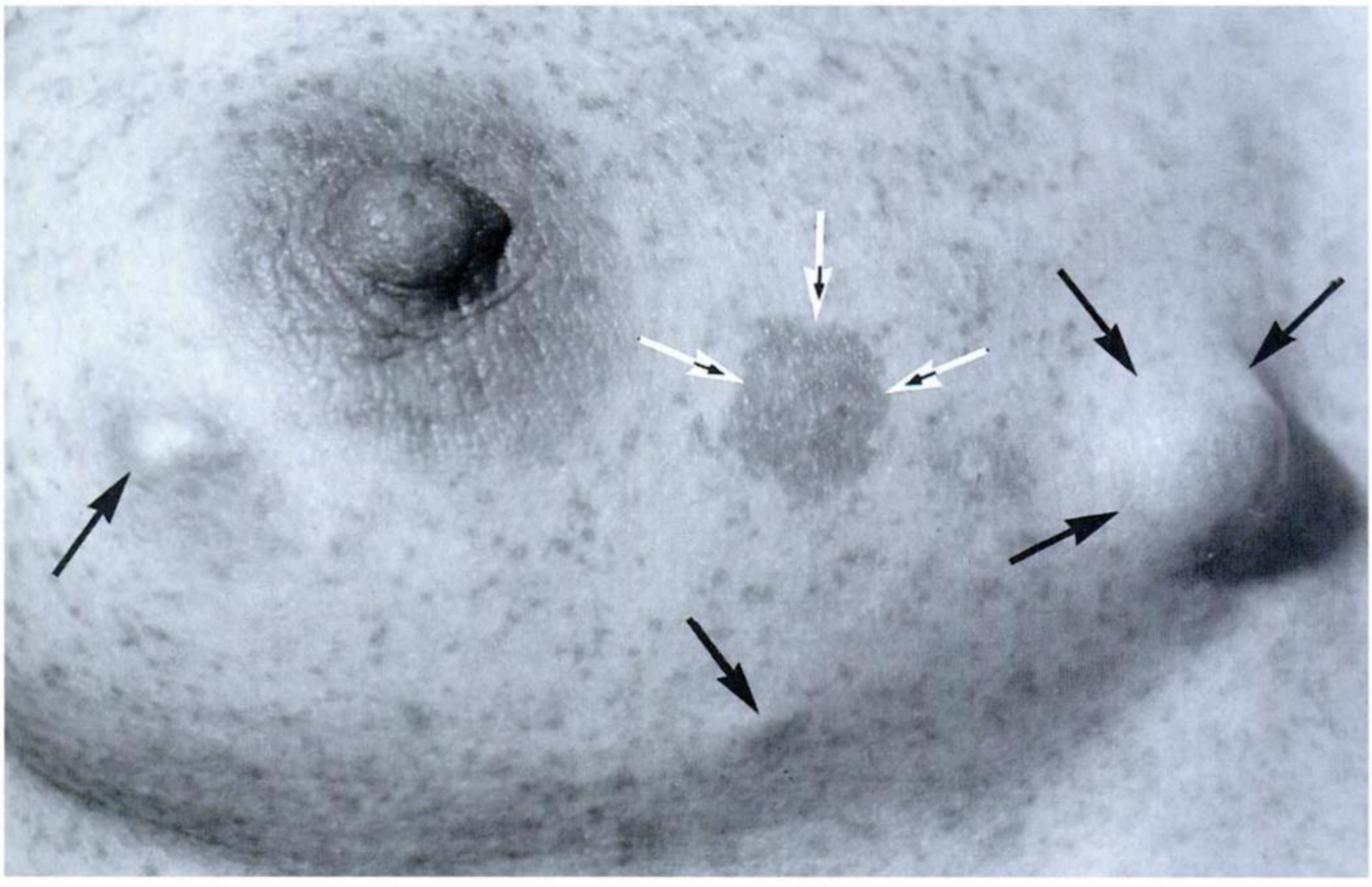


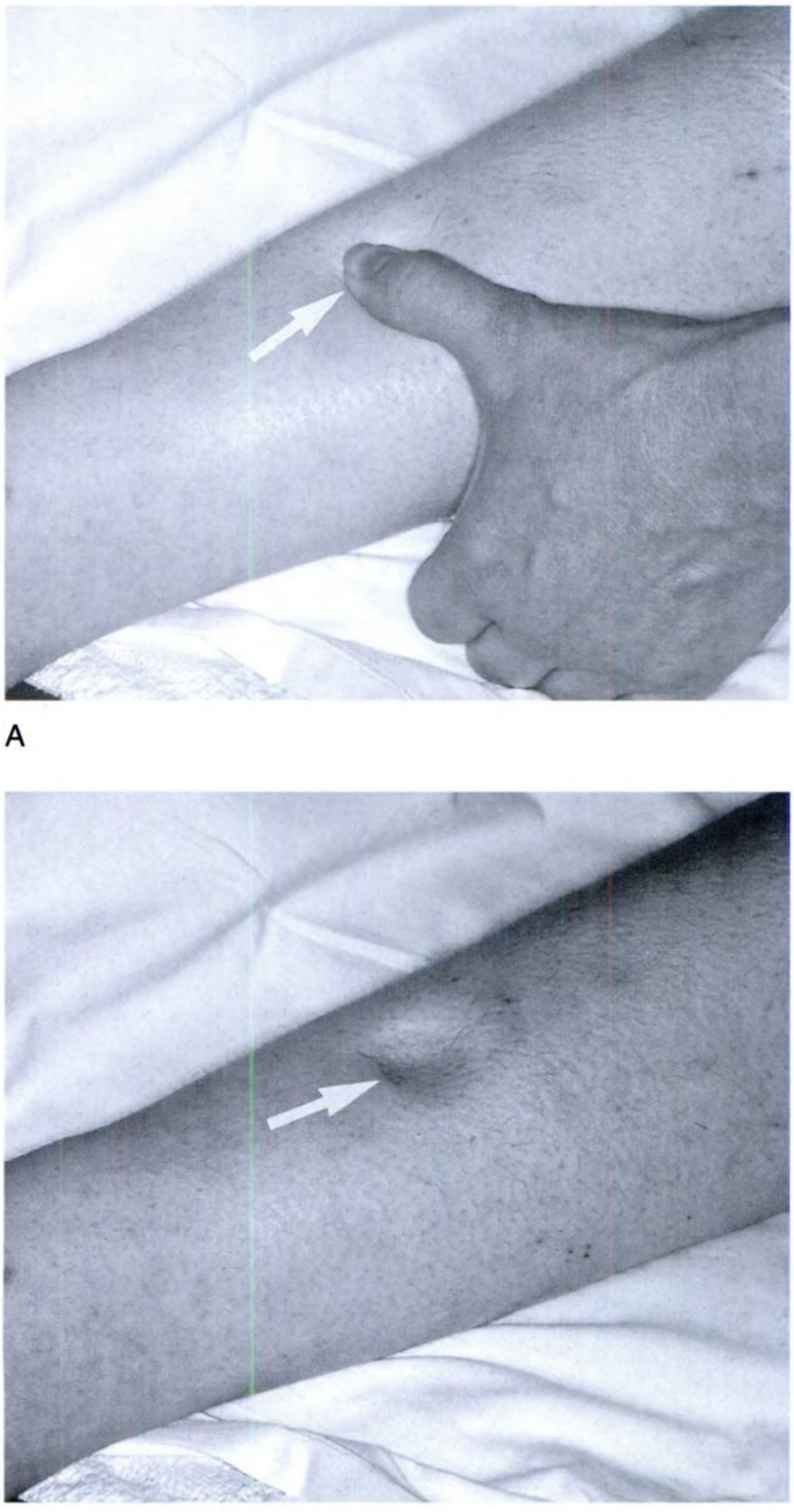
Figure 2–32 *Cafe au lait spot* (*white arrows*), freckling, and cutaneous neurofibromas (*black arrows*) in a 38-year-old woman with von Recklinghausen disease and hypertrophic cardiomyopathy.

(Fig. 2–33) and involving the sacrum in the supine position. Facial edema, especially periorbital, occurs in recumbent infants with congestive heart failure unless the baby is put into an infant seat so its trunk is elevated. Localized *nonpitting* pretibial edema is related to hyperthyroidism, rather than to congestive heart failure.

Acne, a form of localized skin lesion, most commonly found on the face, less commonly on the shoulders or back, incurs the risk of staphylococcic infective endocarditis. Detection sets the stage for prophylactic skin care. An analogous risk results from biting or picking the fingernails and the periungual skin (onychophagia) which causes paronychial infection (Fig. 2–34), staphylococcic bacteremia, and infective endocarditis. Striated xanthomas (yellowish streaks) along the palmar creases (Fig. 2–35) are caused by type III (broad beta) hyperlipoproteinemia, and are associated with coronary artery disease. Xanthomas (soft, yellowish plaques at the inner canthus of the eyelids) were mentioned earlier. Tuberous xanthomas are yellowish, coalescing nodules most commonly found on the elbows or knees. Eruptive xanthomas are crops of yellow-orange papules surrounded by erythematous halos, generally on the arms, legs, and buttocks.

In systemic amyloidosis, discrete localized cutaneous lesions may take the form of subtle, small, translucent, waxy, flat-topped nodules. An ophthalmoscope or hand lens is especially useful in identifying these small lesions, which are ideal for punch biopsy.

Axillary freckling—Crowe sign—is a useful sign of Von Recklinghausen neurofibromatosis (see earlier).[29] Large widespread freckling or lentiginosis (leopard syndrome)



A

B

Figure 2–33 *A*, Pretibal digital compression with the thumb. *B*, Pitting edema induced by digital compression.

is sometimes associated with hypertrophic cardiomyopathy, especially when the lentigines are present from the first year of life.[30]

Localized linear scars along the course of antecubital veins imply "mainline" intravenous drug abuse (Fig. 2–36) with the risk of staphylococcic bacteremia and right-sided infective endocarditis. A small localized cutaneous scar may be the only visible remnant

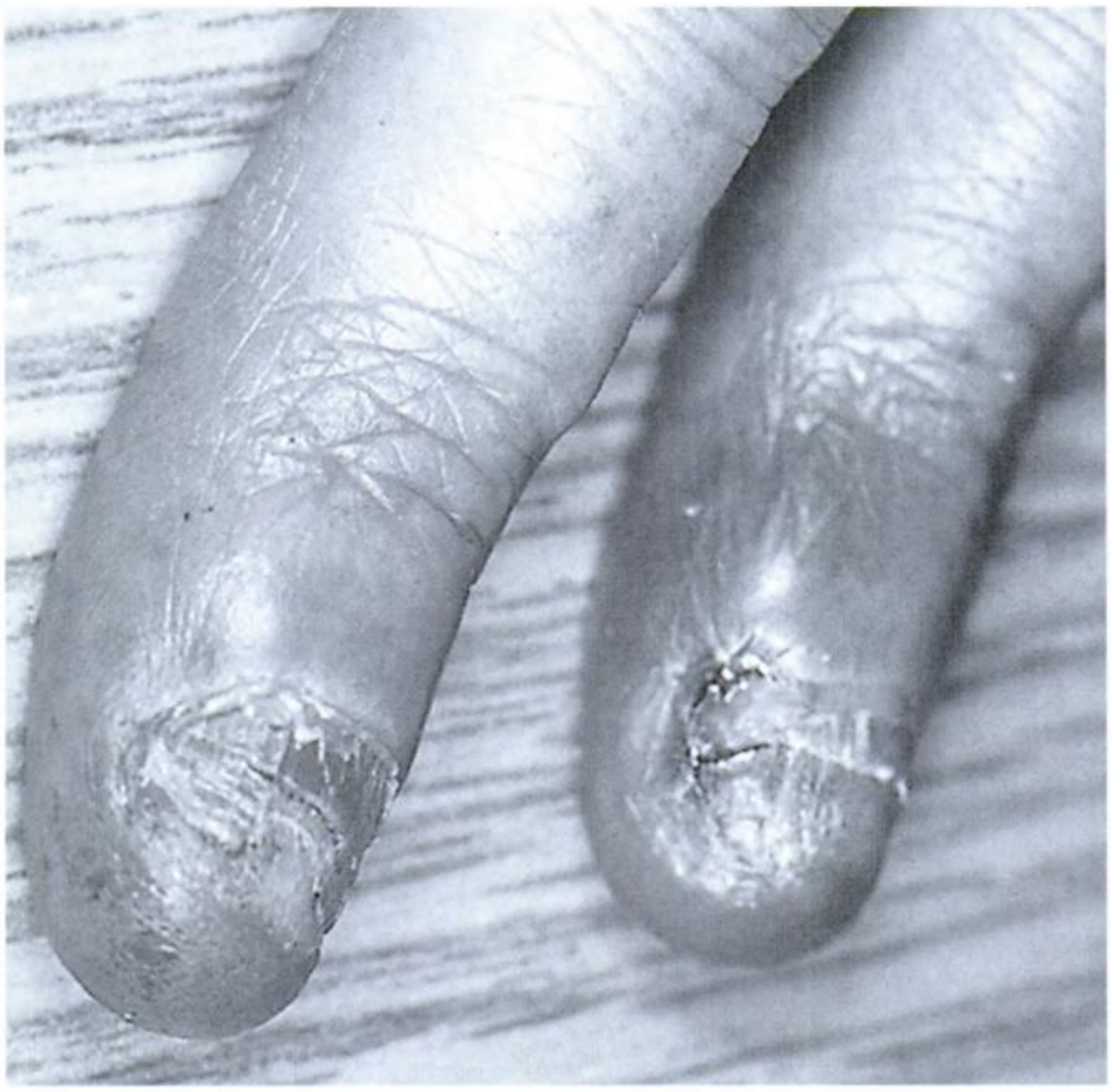


Figure 2–34 Badly bitten infected nails, in a 31-year-old woman with Fallot tetralogy and pulmonary atresia. Staphylococcic bacteremia caused aortic valve infective endocarditis.

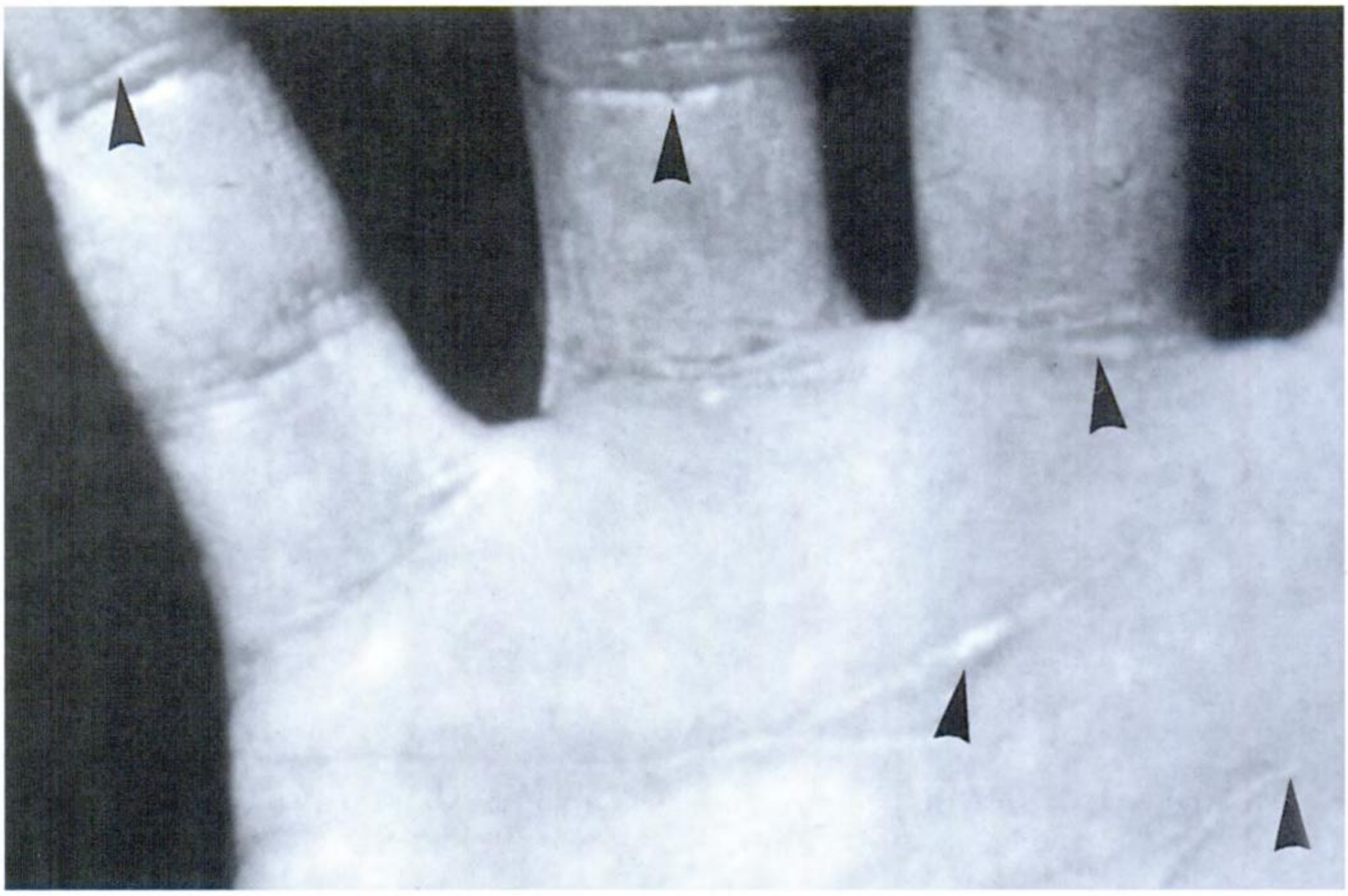


Figure 2–35 Striated xanthomas (yellowish streaks, *arrowheads*) along palmar creases in broad beta-hyperliproteinemia.

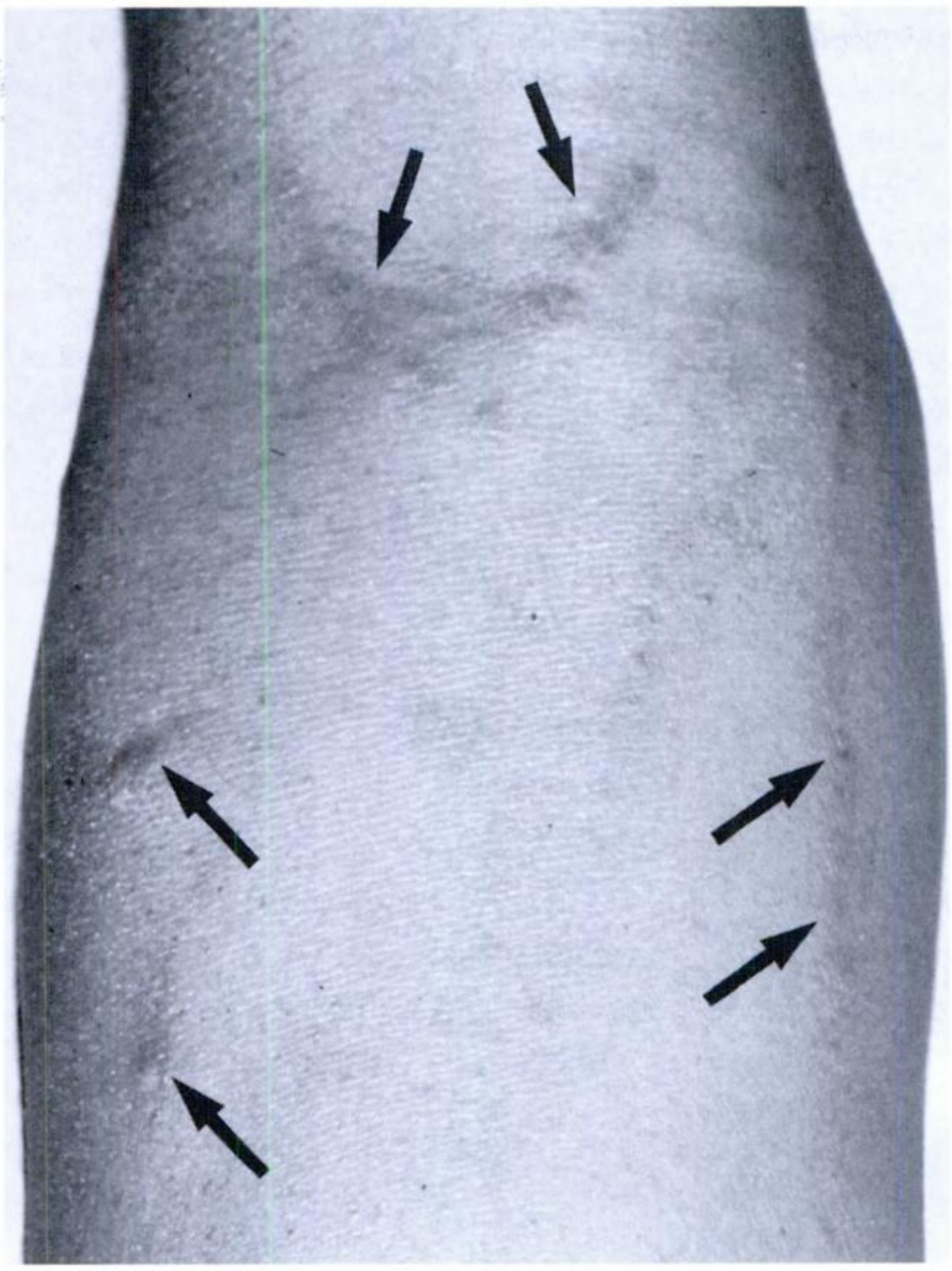


Figure 2–36 Mainline scars (*arrows*) along the course of arm veins in a 21-year-old female heroin addict with pulmonary valve infective endocarditis.

of the entrance site of the penetrating injury responsible for an acquired systemic arteriovenous fistula.

Subcutaneous nodules in acute rheumatic fever tend to reside over bony prominences of the elbows, the dorsa of the hands or feet, and the malleoli or vertebrae. These nodules vary from pinhead to 1 to 2 cm, and are unattached to the skin so that the integument moves freely over them. In tuberous sclerosis, the cutaneous lesions are yellow to orange-red nevi varying in size from a few millimeters to a centimeter, symmetrically distributed on the malar and nasolabial skin. The lesions are also called adenoma sebaceum and are sometimes accompanied by cardiac rhabdomyoma.

Muscles and Tendons

Abnormal appearance of **skeletal muscles** generally implies a decrease in mass (atrophy, dystrophy, wasting). A generalized decrease in muscle mass accompanies the catabolic effects of chronic congestive heart failure. When these catabolic effects occur in infants and young children, they are accompanied by growth retardation.

Alternatively, the decrease in muscle mass can be highly selective, reflecting specific phenotypic features of a systemic neuromuscular disease. An example of selective (regional) muscle wasting (dystrophy, atrophy) is myotonic muscular dystrophy (see Fig. 2–7) with involvement of facial, temporalis, and sternocleidomastoid muscles, distal forearm muscles, and dorsiflexors of the feet. Cardiac involvement targets specialized conduction tissues with abnormalities of impulse and conduction, as mentioned earlier.[31]

The converse of muscle wasting is represented by "pseudohypertrophy" especially but not only of the calves, in boys with classic X-linked Duchenne muscular dystrophy (see Fig. 2–4*B*). Cardiac involvement is common and highly specific[31] (see above).

As a rule, muscle weakness accompanies a *loss* of muscle mass, but occasionally flaccidity occurs *without* muscle wasting. An example is the hypotonia of infants with Down syndrome[5] (Fig. 2–37). The hypotonia decreases or disappears altogether as the child grows older.

Examination of tendons discloses abnormalities varying from subtle to obvious. The Achilles tendons should be palpated routinely in adults to detect thickening caused by tendon xanthomas, which, when large and nodular, are evident at a glance (Fig. 2–38). The diagnostic implication is coronary artery disease. Xanthomas attached to dorsal tendons of the hands may not be seen unless the fingers are gently and rhythmically flexed, imparting motion to the xanthomas that are attached to the tendons but not to the skin. Shortening of Achilles tendons is an important abnormality in boys with Duchenne muscular dystrophy (see Fig. 2–4*B*), causing them to walk on their toes, compromising an already tenuous balance.

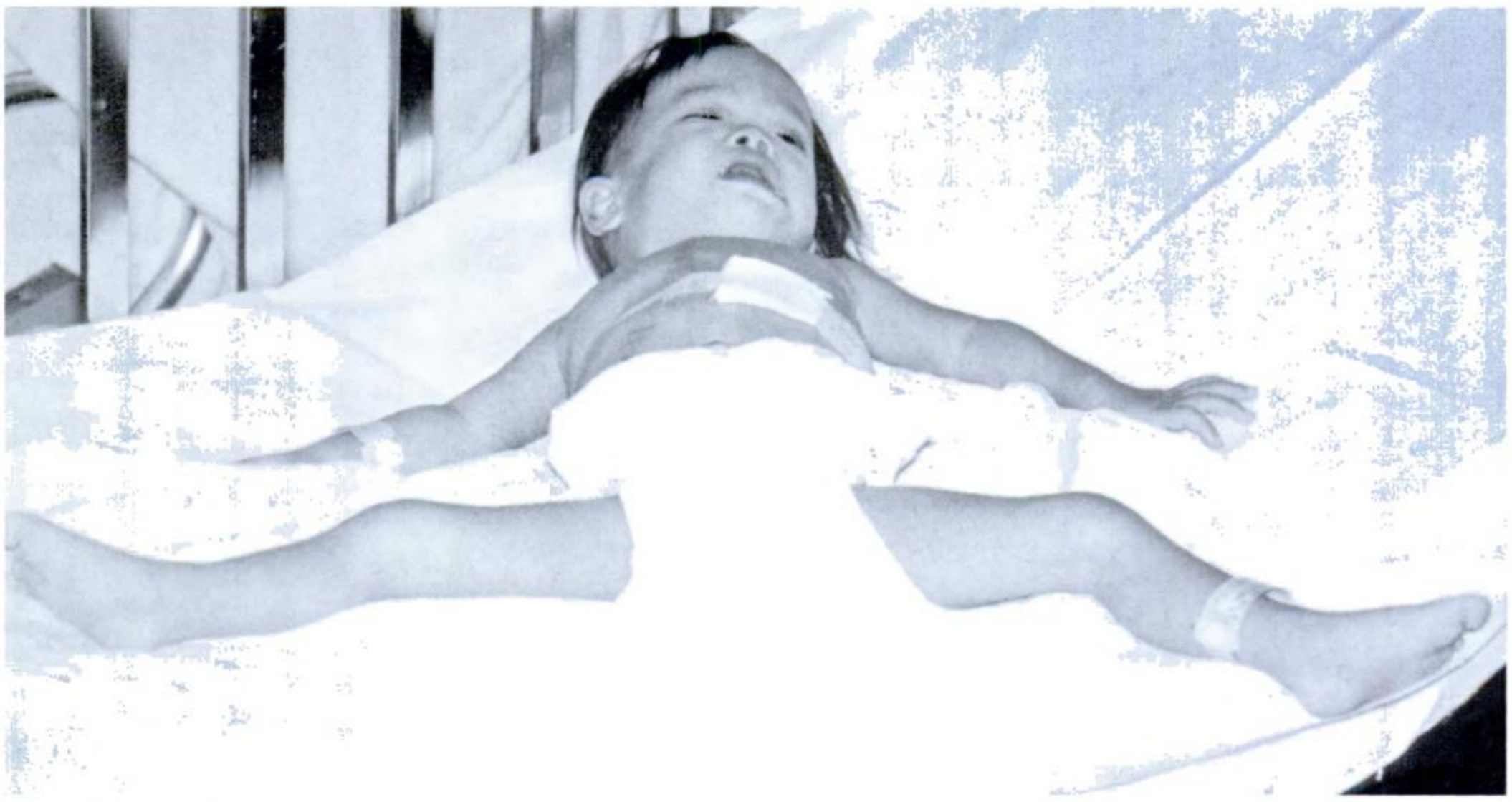


Figure 2–37 Hypotonic legs and hyperflexible hips in a child with Down syndrome. The bandage is postoperative for atrioventricular septal defect repair.

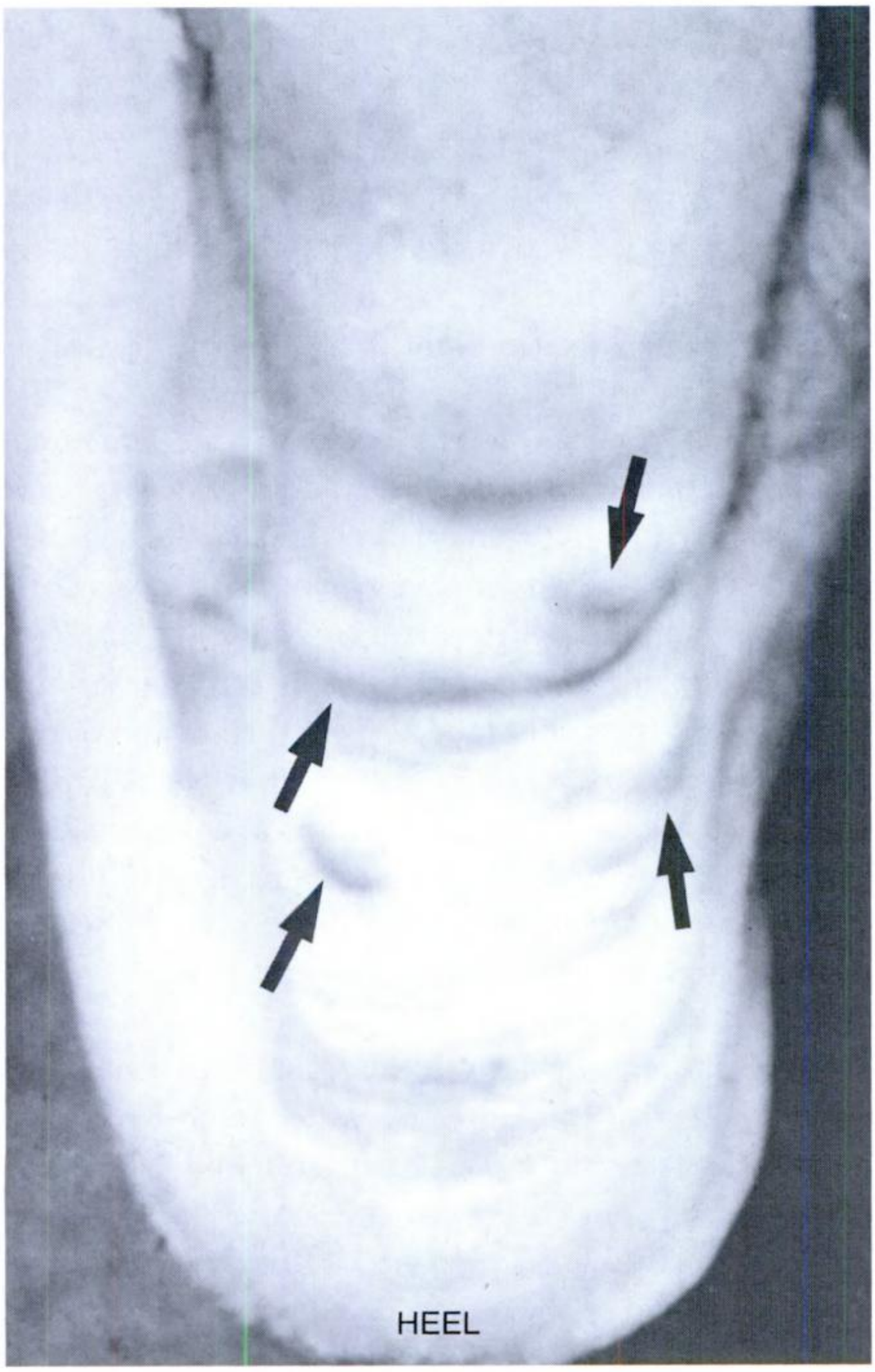


Figure 2–38 Large, nodular Achilles tendon xanthomas (*arrows*) in a young adult male with heterozygous familial hypercholesterolemia and premature coronary artery disease.

The Thorax

The diagnostic value of thoracic appearance lies in its general configuration and in regional abnormalities of bone or soft tissue. Normal and abnormal movements of the thorax during active respiration are dealt with in Chapter 7.

One of the most common clinical abnormalities of thoracic appearance results from pulmonary emphysema (chronic obstructive lung disease). Richard C. Cabot's description of large-lunged emphysema is a case in point:

> The diagnosis can be made by inspection alone. In typical cases, the anteroposterior dimension of the chest is greatly increased, the interspaces are widened, and the costal angle is blunted, while the angle of Ludwig (formed by the junction of

the manubrium with the second piece of the sternum) becomes prominent. The shoulders are high and stooping and the neck is short (see Chapter 7).[32]

Structural abnormalities of the thoracic spine and/or sternum vary from marked distortions that are obvious at a glance to subtle abnormalities that reveal themselves only to a pointed search. Kyphoscoliosis, pectus excavatum, and carinatum are well-known features of Marfan syndrome (see earlier, and see Chapter 7). An innocuous loss of normal thoracic curvature (straight back) varies from subtle to obvious, and is best identified with the patient sitting bolt upright (see Chapter 7). The accompanying decrease in anteroposterior chest dimensions results in physical signs that are sometimes mistaken for heart disease—a left parasternal systolic impulse and a pulmonary midsystolic murmur, for example. Importantly, this form of straight thoracic spine leaves the vertebrae mobile, in contrast to ankylosing spondylitis, in which the thorax is characterized by a rigid spine fused in flexion, and in which aortic regurgitation is a common feature. Pectus excavatum of moderate degree is easily overlooked in females with well-developed breasts. Marked pectus excavatum shifts the heart to the left and results in a parastemal systolic impulse, and a pulmonary midsystolic murmur, signs similar to those described above in patients with a loss of thoracic kyphosis and a decrease in anteroposterior chest dimension. Minor degrees of scoliosis are often recognized during a routine physical examination, but are readily identified in an anteroposterior chest radiograph. The scoliosis is almost always convex to the *right.* Convexity to the *left* arouses suspicion of a systemic neuromuscular disease or poliomyelitis.

Abnormalities of thoracic configuration are relatively frequent in patients with congenital heart disease, but the hypothesis that these alterations are due to an increase in right ventricular mass has not withstood scrutiny.[33] The usual variety of chest deformity in such patients is anterior asymmetry, especially a left precordial bulge. When cardiac dyspnea in infants and young children causes chronic repetitive traction on the diaphragmatic insertions of the rib cage during growth and development, nonrachitic Harrison's grooves commonly result.

Soft tissue abnormalities of the thorax are diagnostically important, such as the scar of a previous thoracotomy. A midline sternotomy scar is not likely to be overlooked, but lateral thoracotomy scars may be missed. A lateral thoracotomy scar together with *ipsilateral* absence of the brachial arterial pulse is a feature of a classic Blalock–Taussig shunt. A *left* thoracotomy scar together with absence of the *left* brachial pulse is presumptive evidence of a right aortic arch and a left Blalock–Taussig shunt.

Abnormalities of the breasts occasionally take the form of unilateral or bilateral gynecomastia in males receiving digitalis glycosides[34] (Fig. 2–39). Conversely, *hypomastia* is part of an asthenic habitus in females. In 45/ XO Turner syndrome, widely spaced nipples are components of the broad shield chest (see Fig. 2–2*A*).

Abdomen

For information on the physical appearance of the abdomen, see Chapter 8.

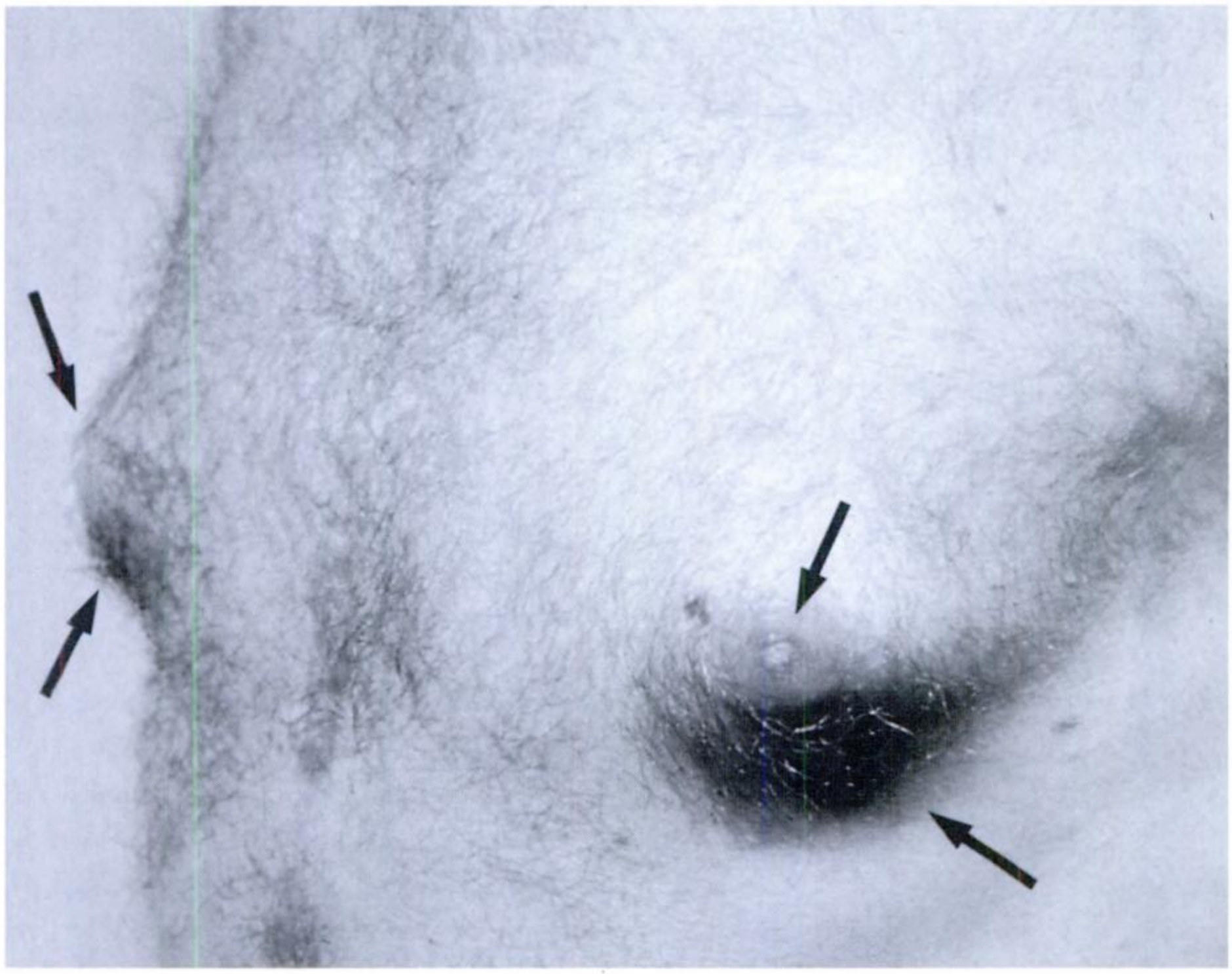


Figure 2–39 Male gynecomastia in response to digitalis glycosides in an average maintenance dose.

REFERENCES

1. Gombrich EH. *Meditations on a Hobby Horse.* London: Phaidon, 1978.
2. Shakespeare W. *King Henry V,* Act 11, Scene 111.
3. Milewicz DM. Molecular genetics of Marfan syndrome and Ehlers-Danlos, type IV. *Curr Opin Cardiol.* 1998;13:198.
4. Farquhar JW. The child of the diabetic woman. *Arch Dis Child.* 1958;34:76.
5. Perloff JK. *The Clinical Recognition of Congenital Heart Disease,* 5th ed. Philadelphia, PA: WB Saunders, 2003.
6. Burwell CS, Robin ED, Whaley RD, Bickelmann AG. Extreme obesity associated with alveolar hypoventilation—A pickwickian syndrome. *Am J Med.* 1956;21:811.
7. Mackenzie J. *Principles of Diagnosis and Treatment in Heart Affections.* London: Oxford University Press, 1916.
8. Shakespeare W. *King Richard III,* Act 111, Scene IV.
9. Perloff JK, Phelps ET. A review of Wemer's syndrome, with a report of the second autopsied case. *Ann Intern Med.* 1958;48:205.
10. Renteria VG, Ferrans VJ, Roberts WC. The heart in the Hurler syndrome: Gross, histologic and ultrastructural observations in five necropsy cases. *Am J Cardiol.* 1976;38:487.
11. Clarren SK, Smith DW. The fetal alcohol syndrome. *N EngI J Med.* 1978;298:1063.

12. Cayler GG, Blumenfeld CM, Anderson RL. Further studies of patients with the cardiofacial syndrome. *Chest.* 1971;60:161.
13. Roberts NK, Perloff JK, Kark RAP. Cardiac conduction in the Kearns-Sayre syndrome (a neuromuscular disorder associated with progressive external ophthalmoplegia and pigmentary retinopathy). *Am J Cardiol.* 1979;44:1396.
14. Silverman KH, Hutchins GM, Bulkley BH. Cardiac sarcoid: A clinicopalhologic study of 84 unselected patients with systemic sarcoidosis. *Circulation.* 1978;58:1204.
15. Ruppert GB, Lindsay J, Barth WF. Cardiac conduction abnormalities in Reiter's syndrome. *Am J Med.* 1982;73:335.
16. Criscitiello MG, Ronan JA, Besterman EM, Schoenwetter W. Cardiovascular abnormalities in osteogenesis, imperfecta. *Circulation.* 1965;31:255.
17. Freedom RM, Gerald PS. Congenital heart disease and the "cat eye" syndrome. *Am J Dis Child.* 1973;126:16.
18. Heggtveit HA: Syphilitic aortitis. A clinicopathologic autopsy study of 100 cases, 1950 to 1960. *Circulation.* 1964;29:346.
19. Hollenhorst JZW. Significance of bright placques, in the retinal arterioles. *JAMA.* 1961;178:23.
20. Editorial. Pulmonary arteriovenous fistula in hereditary hemorrhagic telangiectasia. *Lancet.* 1960;1:158.
21. Jeresatz RM, Husyar RJ, Basu S. Pierre Robin syndrome. *Am J Dis Child.* 1969;117:710.
22. Beaven DW, Brooks SE. *A Color Atlas of the Nail in Clinical Diagnosis.* Weert, Netherlands: Wolfe Medical Publications, 1984.
23. Edeiken J. Shoulder-hand syndrome following myocardial infarction with special reference to prognosis. *Circulation.* 1957;16:14.
24. Bittl JA, Perloff JK. Chronic post-rheumatic fever arthropathy of Jaccoud. *Am Heart J.* 1983;105:515.
25. Gellis SS, Feingold M. Rubinstein-Taybi syndrome. *Am J Dis Child.* 1971;121: 327.
26. Holt M, Oram S. Familial heart disease with skeletal malformation. *Br Heart J.* 1960;22:236.
27. Child JS, Perloff JK, Bach PM, et al. Cardiac involvement in Friedreich's ataxia. *J Am Coll Cardiol.* 1986;7:1370.
28. Dabestani A, Child JS, Henze IN, et al. Primary hemochromatosis: Anatomic and physiologic characteristics of the cardiac ventricles and their responses to phlebotomy. *Am J Cardiol.* 1984;54:153.
29. Crowe FW. Axillary freckling as a diagnostic aid in neurofibromatosis. *Ann Intern Med.* 1964;61:1142.
30. Somerville J, Bonham-Carter RE. The heart in lentiginosis. *Br Heart J.* 1972;34:58.
31. Perloff JK. Neurological disorders and heart disease. In: Braunwald E, ed. *Heart Disease*, 5th ed. Philadelphia, PA: WB Saunders, 1997: p1865.
32. Cabot RC. *Physical Diagnosis.* New York: William Wood, 1915.
33. Maxwell GM. Chest deformity in children with congenital heart disease. *Am Heart J.*1957:54:368.
34. LeWinn EB. Gynecomastia during digitalis therapy. *N Engl J Med.*1953:248:316.

3 The Arterial Pulse

With careful practice, the trained finger can become a most sensitive instrument in the examination of the pulse.[1]

James Mackenzie trained his palpating finger by meticulous documentation with his polygraph (Fig. 3–1). The ancient art of feeling the pulse (Fig. 3–2) can still be applied with considerable advantage[2]:

> In examining the pulse, our object is to obtain the most complete and exact knowledge attainable and to interpret accurately the facts we observe; the method to be followed must therefore be carefully described.[3]

This chapter describes methods of examining the arterial pulse and calls attention to the clinical information so derived. *Examination* refers to palpation, observation, and auscultation of arterial pulses. Certain pulses are examined routinely; others are examined depending upon the clinical circumstances. *Palpation* in infants routinely includes the brachial and femoral arteries. In children and young adults, the carotid is palpated routinely, whereas in still older adults, routine palpation includes the carotids, brachials, radials, femorals, dorsalis pedis, posterior tibials, and abdominal aorta. Palpation of additional arterial pulses is performed chiefly but not exclusively in older adults, and includes the subclavian, popliteal, ulnar, and digital (fingertip) pulses.

Observation refers to visible arterial pulsations. Certain pulses are readily seen, such as the carotid pulsations of severe aortic regurgitation, or the serpentine brachial pulse of Monckeberg sclerosis (see Fig. 3–3). Others pulses are less obvious, less important, and are overlooked unless specifically sought such as the pulsating dorsalis pedis in an infant with a large left-to-right shunt through a patent ductus arteriosus.

Auscultation of arterial pulses is based chiefly on patient age. In children and young adults, normal supraclavicular systolic murmurs are common, and are elicited by placing the bell of the stethoscope over the subclavian arteries in the supraclavicular fossae (see Chapter 6). In older adults, auscultation of the carotids, subclavians, and femoral arteries should be routine because of the incidence of atherosclerotic obstruction. Auscultation over the spine between the scapulae is reserved for patients with coarctation of the aorta. Auscultation of the abdominal aorta and its bifurcation and over the renal arteries is selectively undertaken.

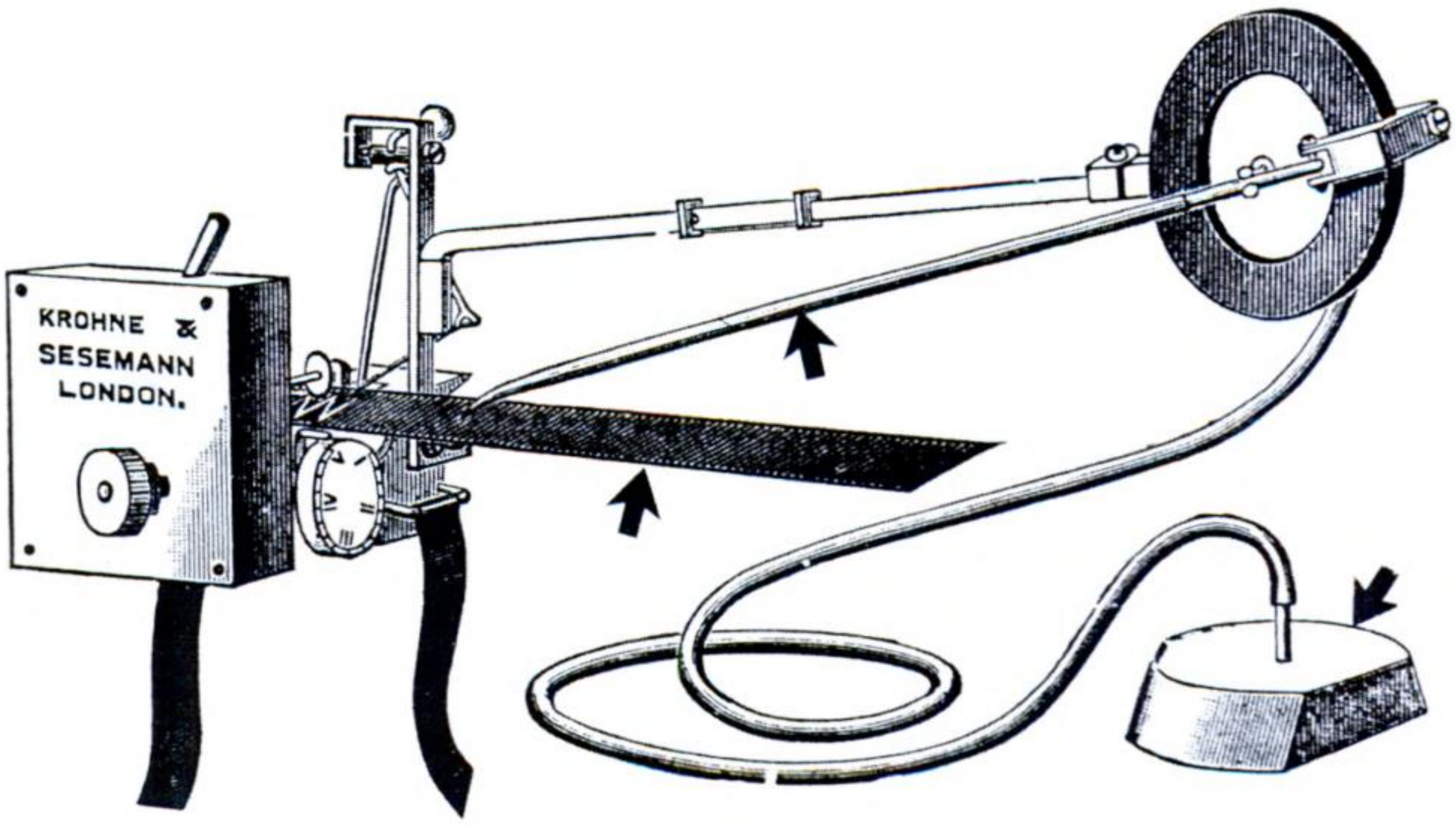


Figure 3–1 Mackenzie's polygraph designed to record a sphygmogram which is a diagrammatic representation of the variations of pressure within an artery. When the spring of the sphygmograph (*lower right arrow*) is so accurately adjusted on an artery that it does not obliterate the artery when the arterial pressure is at the lowest, and still slightly compresses the artery when the arterial pressure is at the highest, the spring will oscillate with each variation of pressure within the artery. This oscillation being communicated to the lever (*middle upper arrow*) and recorded on the tracing paper (*left lower arrow*), gives us a series of wavy lines, which represent the variations of the pressure within the artery. (Arrows my additions.) (From Mackenzie J: The Study of the Pulse Arterial, Venous and Hepatic and of the Movements of the Heart. Edinburgh, Young J. Pentland, 1902.)

Figure 3–2 Franz van Mieris, "The Doctor's Visit." (Courtesy of, Kunsthistorisches Museum, Vienna.) In female patients, decorum precluded palpation of any pulse except the radial.

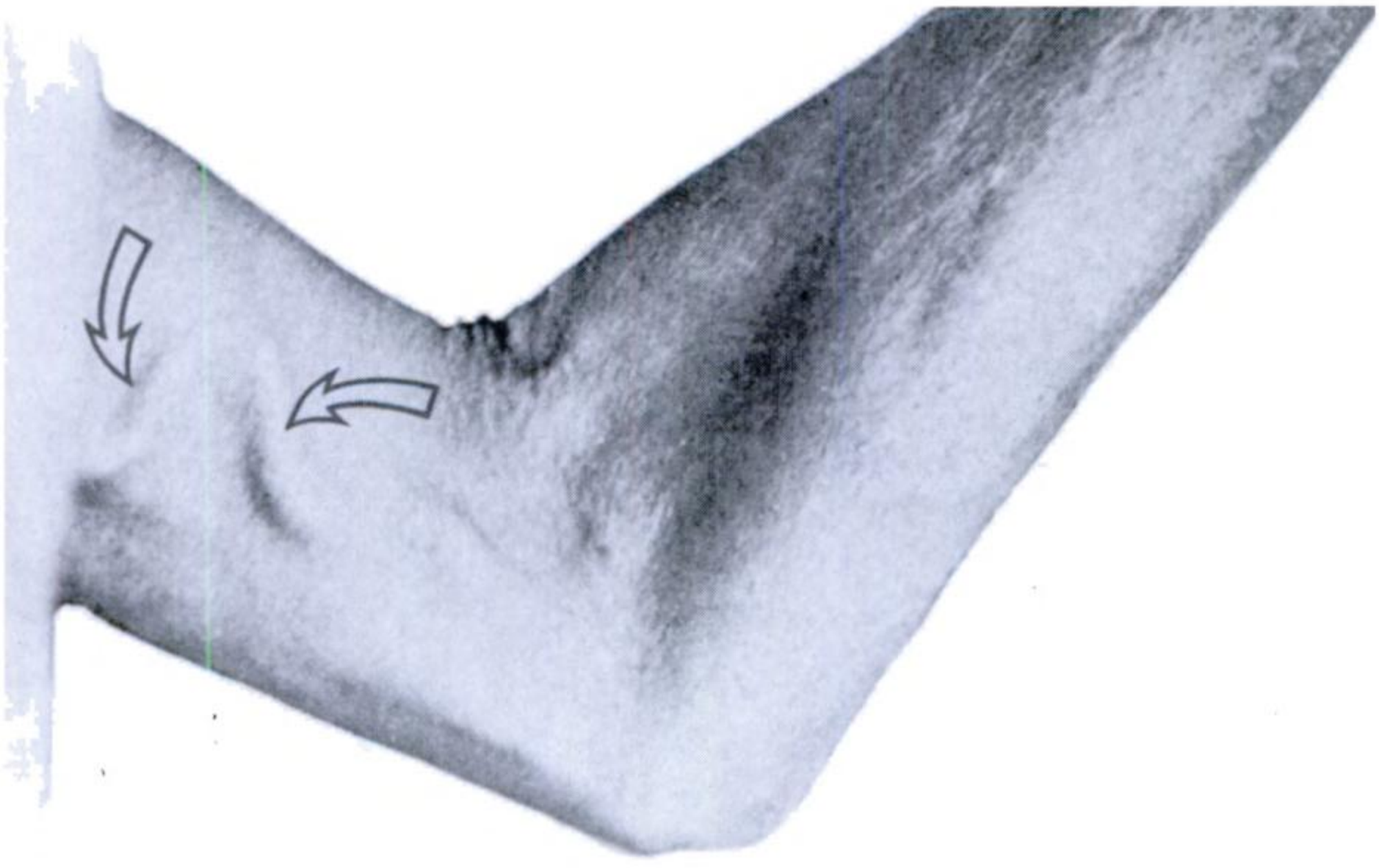


Figure 3–3 The thickened, serpentine brachial artery of Monckeberg's sclerosis visible beneath the skin of the antecubital fossa (*arrows*). (From Cabot RC: Physical Diagnosis. New York, William Wood and Co, 1915.)

INFORMATION DERIVED FROM THE ARTERIAL PULSE

> The point first to be noted is the frequency—the number of beats per minute—the regularity or irregularity of the beats as to time, and their equality or inequality in force…. We should naturally wish, in the next place, to estimate the force or strength of the pulse…. An important point to be investigated is the degree of constant pressure prevailing in the arteries…. The character of the beat is another matter for study; and brief as is the period occupied by it, each pulse wave presents a rise, duration, and fall.[3]

A SYSTEMATIC APPROACH

To avoid oversight, examination of the arterial pulse should be systematic. A consistent sequence should be employed (Table 3–1), although the sequence might differ from examiner to examiner.

Blood Pressure

Before the sphygmomanometer was introduced, Mackenzie argued that

> the trained finger is as yet the best guide we have in judging the pressure in an artery. The fingertips become so educated in the course of time, that we readily appreciate the sensation conveyed in compressing an artery.[1]

Table 3–1 Examination of the Arterial Pulse

1. Blood pressure
2. Cardiac rate and rhythm
3. Waveform
4. Differential pulsations:
   Right/left, upper/lower
   Selective diminution, absence, or augmentation
5. Arterial thrills and murmurs
6. Structural properties

Systolic arterial pressure can be estimated by the amount of brachial arterial compression required to obliterate the ipsilateral radial pulse palpated by the examiner's other hand. When relatively mild brachial arterial compression obliterates the radial arterial pulse in adults, the systolic pressure is generally less than 120 mmHg. When considerable compression is required to achieve this end, the systolic pressure generally exceeds 160 mmHg.

Indirect Measurement

Indirect measurement of blood pressure using an occluding pressure cuff and arterial auscultation was one of the earliest quantitative measurements of major importance employed at the bedside. Potain (1845–1901) introduced the sphygmomanometer.[4] The occluding cuff was developed almost immediately by Hill and Barnard in England, and in Italy by Riva-Rocci, a pupil of Potain.[4] However, for practical clinical use, a supplement to these two devices was required, namely, a reliable yet simple method for measuring blood pressure at the bedside. A pivotal figure in filling this need was the young Russian surgeon Nikolai Korotkoff, who wrote in 1905,

> Immediately below a completely compressed artery (with obliteration of the lumen) no sounds are heard. As soon as the first drops of blood escape from under the site of pressure, we hear a clapping sound very distinctly. This sound is heard when the compressed artery is released and even before the appearance of pulsation in the peripheral branches.[5]

The next step was the binaural stethoscope which facilitated detection of brachial arterial Korotkoff sounds. The modern binaural stethoscope, and a mercury aneroid, or digital cuff provide contemporary clinicians with a refined bedside method for accurate determination of systemic arterial pressure. The stethoscope should be equipped with a bell and diaphragm. The sphygmomanometer's compression cuff, the hand-operated bulb for inflation, and the adjustable valve for cuff deflation should be in perfect working order. The inflatable bladder must be completely and securely contained within the sealed inelastic cuff so that pressure is distributed evenly over the area to which the cuff is applied. Standard portable aneroid and digital manometers should be periodically recalibrated against a mercury manometer.

Taking the blood pressure is routine, but errors abound because of faulty technique. In addition to an instrument in perfect working order, correct cuff size is mandatory because an undersized cuff causes falsely high readings and an oversized cuff causes falsely low readings.[6] Cuff size refers to the width and length of the inner inflatable bladder, not to the cloth covering. Selection of appropriate cuff size is determined by the circumference of the limb to which it is applied, not the age of the patient.[6] The width of the inflatable bladder should be approximately 40 percent of the circumference of the limb, and the length of the bladder within the cuff must completely encircle the extremity without overlapping.

Brachial arterial pressure is best determined in a quiet setting with the patient comfortably supine and the arms extended and relaxed parallel to and at the approximate level of the heart. Although upper extremity blood pressure is routinely determined in the supine position, a sitting position is acceptable in a young child who may be more relaxed when sitting in the parent's lap, or for home blood pressure in adults who while sitting, should rest the cuffed arm on a table at the approximate level of the heart.

Sufficient time must elapse for the patient to recover from recent activity and to relax, especially in an outpatient setting. Blood pressure should be taken in both arms because systolic pressure tends to be somewhat higher in the right arm, as much as 15 mmHg in normal adults. It is useful to palpate the brachial artery during cuff inflation to be certain that the inflation pressure is 20 to 30 mmHg above the pressure required to obliterate the brachial pulse. The adjustable screw valve permits controlled, slow deflation of the cuff. Peak systolic pressure is identified by the appearance of Korotkoff sounds for two or more consecutive beats. Continued slow deflation determines the diastolic pressure which is the point at which Korotkoff sounds disappear rather than the point of muffling, because the disappearance point is closer to the intra-arterial diastolic pressure. Inappropriately slow cuff deflation should be avoided because the accompanying venous congestion decreases the intensity of Korotkoff sounds, so that systolic pressure is underestimated while diastolic pressure is overestimated. Once the diastolic pressure is established, the cuff is rapidly deflated. At least 1 minute should elapse before blood pressure measurements are repeated. Audibility of brachial arterial Korotkoff sounds is improved, sometimes appreciably, by having the patient open and close the fist vigorously a dozen or so times while the arm is elevated.

An "auscultatory gap" occasionally separates initial Korotkoff sounds from subsequent audibility at a lower pressure. The importance of the auscultatory gap is the risk of misjudging systolic pressure when the cuff is inflated to a level within the gap itself. This error is avoided by inflating the cuff well above the level necessary to obliterate the brachial arterial pulse (see above).

The seemingly outmoded technique of palpating the pulse for estimating arterial pressure occasionally serves a useful purpose in an acute care setting when Korotkoff sounds are indistinct or inaudible. The approximate peak systolic pressure is the level at which a brachial pulse is first palpated when the cuff is slowly deflated. The diastolic pressure is then estimated by detecting a distinctive snapping quality of the palpable pulse as the cuff is deflated further.

There is a misconception that cuff brachial arterial pressure is inherently inaccurate in patients with obese arms. Error is avoided by selecting a cuff of appropriate size (ie, width and length of the inflatable bladder as recommended above). A cuff of proper width (40 percent of the limb circumference) should completely encircle the arm and exert pressure evenly over the enclosed area.[6]

Blood pressure should be measured beginning at 3 years of age with normal blood pressure in children judged according to height and weight. Elderly patients may experience a significant postprandial fall in systolic and diastolic blood pressure. A higher cuff pressure is required to compress a rigid sclerotic artery in the elderly than to compress a normally compliant artery at any age. A sclerotic artery of reduced compliance can be detected if the ipsilateral brachial or radial pulse remains palpable when the blood pressure cuff is inflated above systolic—spurious systolic hypertension. This observation was made by William Osler in 1892 before blood pressure was routinely measured in clinical practice:[7]

> It may be difficult to estimate how much of the hardness and firmness is due to the tension of the blood within the vessel and how much to the thickening of the wall. If, for example, when the radial artery is compressed with the index finger, the artery can be felt beyond the point of compression, its walls are sclerosed.

A potentially useful variation on routine blood pressure is *proportional pulse pressure* calculated as the difference between systolic and diastolic pressures divided by the systolic pressure. A proportional pulse pressure 25 percent or less identifies left ventricular failure and cardiac indices of 2.2 L/min/M^2 or less in about 90 percent of patients.

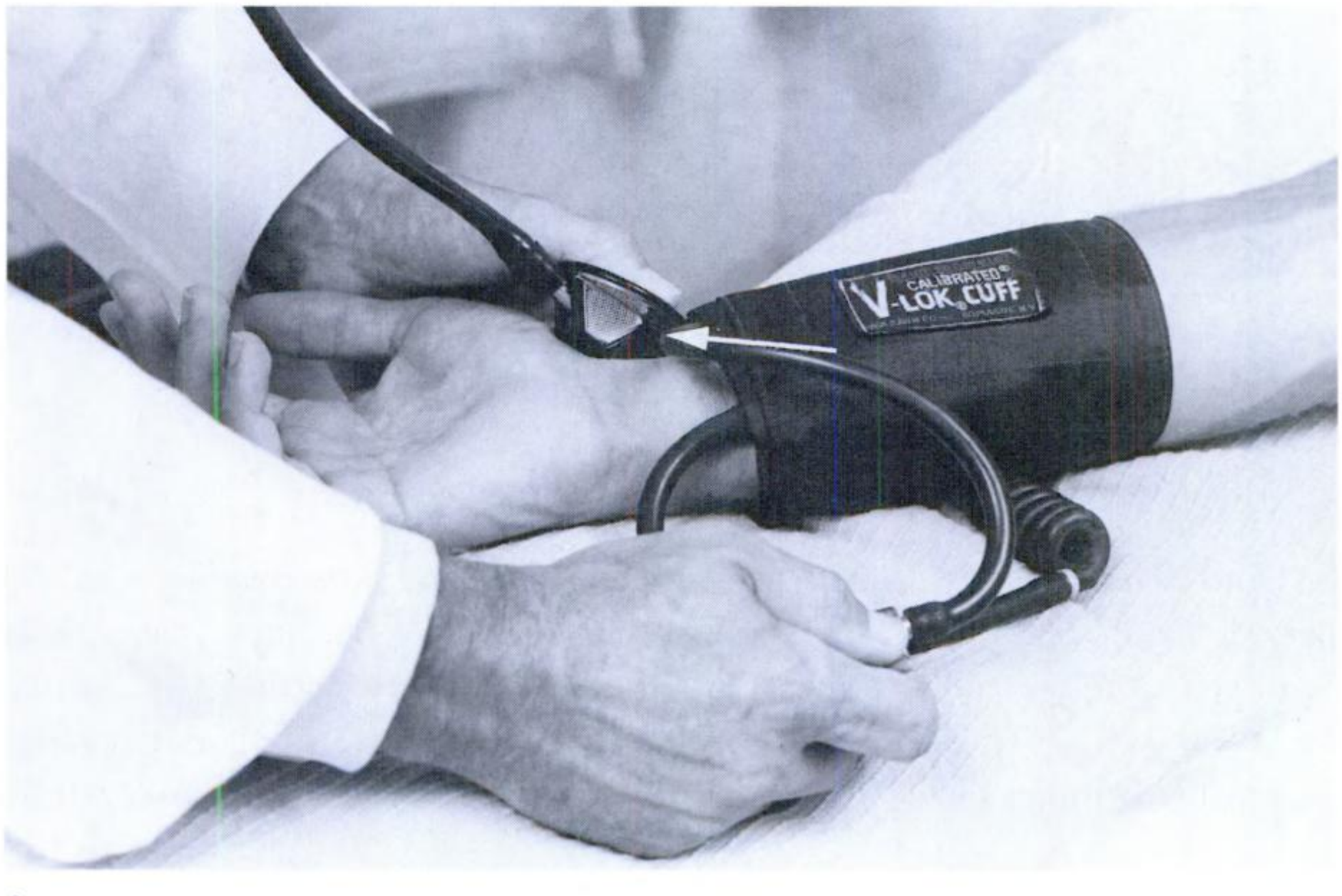
If the only available cuff does not adequately encircle the upper arm, the same cuff might be tried on the forearm while the bell of the stethoscope is applied to the radial artery for the detection of Korotkoff sounds (Fig. 3–*4A*). This is a satisfactory technique provided that the forearm is relatively uniform in circumference, but not when a conical forearm prevents an even distribution of pressure over the encircled area. Properly determined forearm blood pressure is a reasonable approximation of brachial arterial pressure.

Orthostatic Hypotension

In adults beyond middle age, orthostatic hypotension can be identified by recording brachial arterial pressure in the supine and upright positions—either with the patient standing or with legs dangling over the edge of the examining table or bed. A change in position from supine to upright is normally accompanied by no more than a 5 to 15 mmHg decline in systolic pressure, whereas diastolic pressure tends to rise. These changes are accompanied by slight reflex tachycardia. Symptomatic orthostatic hypotension is a feature of the Shy–Drager syndrome, a rare, progressive degenerative disease of the autonomic nervous system with loss of involuntary control of blood pressure and heart rate.

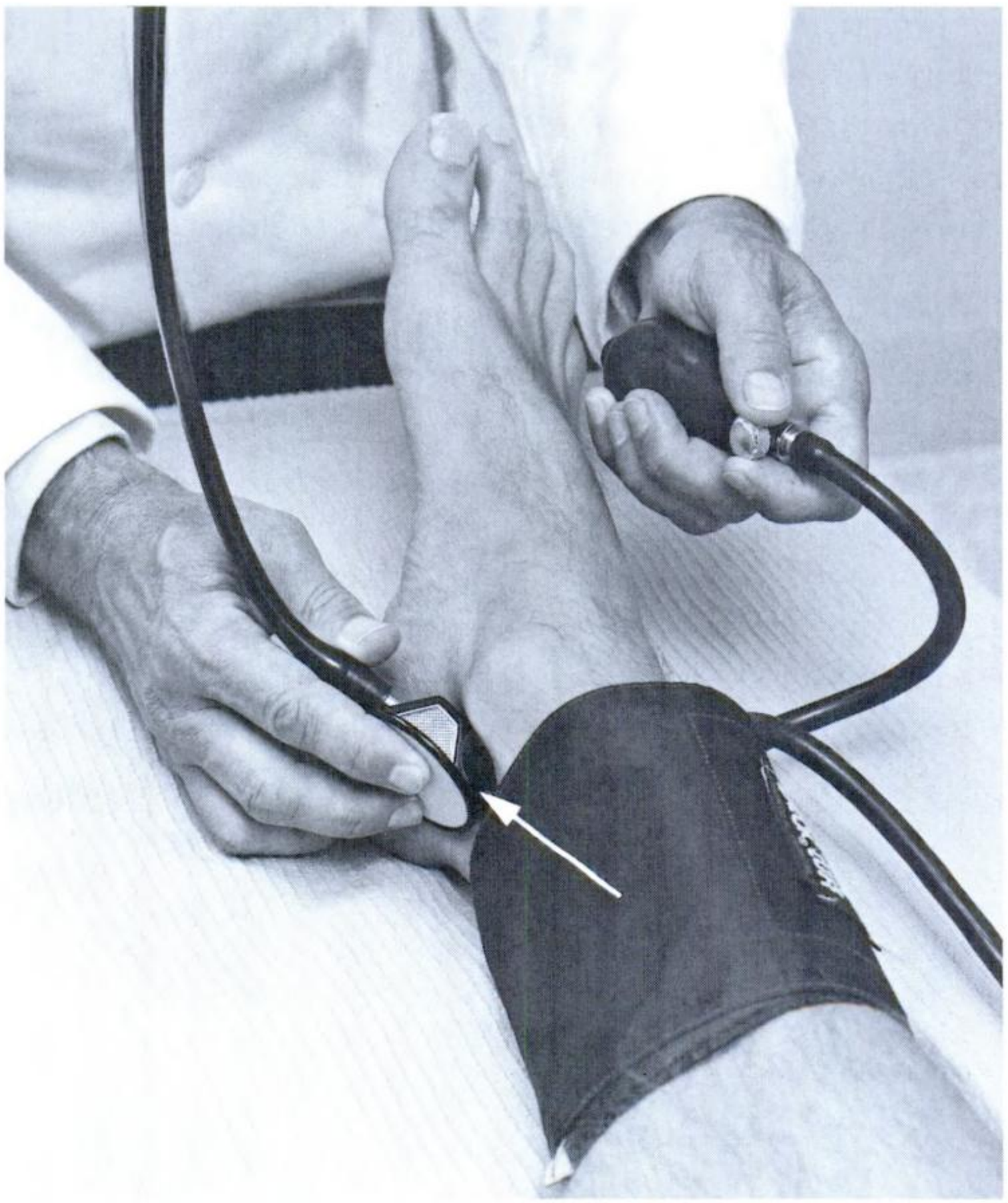
A special and important form of postural (positional) hypotension is provoked by the *left* lateral decubitus position.[8] To elicit left lateral decubitus hypotension, a cuff is placed on both arms, and blood pressure recorded in the supine position, bearing in mind that, in some normal adults, right arm systolic pressure exceeds left arm systolic pressure (see above). The patient is then asked to turn into the *right* lateral decubitus position while blood pressure is taken in the *left* arm. After a brief return to the supine position, the patient is asked to turn into the *left* lateral decubitus position while blood pressure is recorded from the *right* arm. Normally, there is no difference in blood pressure from supine to right or left lateral decubitus position. Left lateral decubitus hypotension includes a fall in both systolic *and* diastolic pressure, sometimes accompanied by dyspnea, palpitations, weakness, and light-headedness.

When an infant is quieted with a bottle or pacifier, blood pressure can be obtained by the auscultatory method employing a cuff of appropriate size. Alternatively, the "flush" method can be used. An uninflated cuff is applied to the infant's elevated forearm, while the limb distal to the cuff is gently massaged to induce blanching. The cuff is then inflated above anticipated systolic pressure while the arm remains



A

Figure 3–4 *A,* If a cuff appropriate for the upper arm is not available, a standard upper arm cuff can be applied to the forearm as shown here, provided the forearm is relatively cylindrical. The bell of the stethoscope (*arrow*) is placed over the radial artery for detection of Karotkoff sounds. *B* (*see next page*), If a cuff appropriate for the leg is not available, a standard upper arm cuff can be applied to the distal calf as shown here. Korotkoff sounds are elicited by placing the bell of the stethoscope over the dorsalis, pedis or posterior tibial artery (*arrow*), the latter generally providing a better skin seal. *Continued*



B

Figure 3–4 *Continued*

elevated and blanched. The arm is then lowered to a horizontal position and the cuff is slowly deflated. The point at which the blanched hand becomes flushed is an estimate of the *mean* arterial pressure. To perform the flush method two persons are required, and even then only a mean arterial pressure is secured. Alternatively, a simple bedside Doppler device can be used by a single observer to record systolic and diastolic pressures.

In patients with coarctation of the aorta *lower extremity* blood pressure is obtained from the popliteal artery with the patient prone. The popliteal artery is identified by palpation (see Fig. 3–5*A*). A large-size cuff is applied to the thigh,[6] and inflated to a level just above the brachial arterial systolic pressure. The cuff should be inflated slowly to avoid discomfort, if not pain. Systolic and diastolic pressures are estimated by auscultation with the stethoscope applied in the poplitieal fossa for Korotkoff sounds originating in the popliteal artery. Not uncommonly, only systolic Korotkoff sounds are clearly heard, but this is not a shortcoming in the assessment of differential blood pressure in

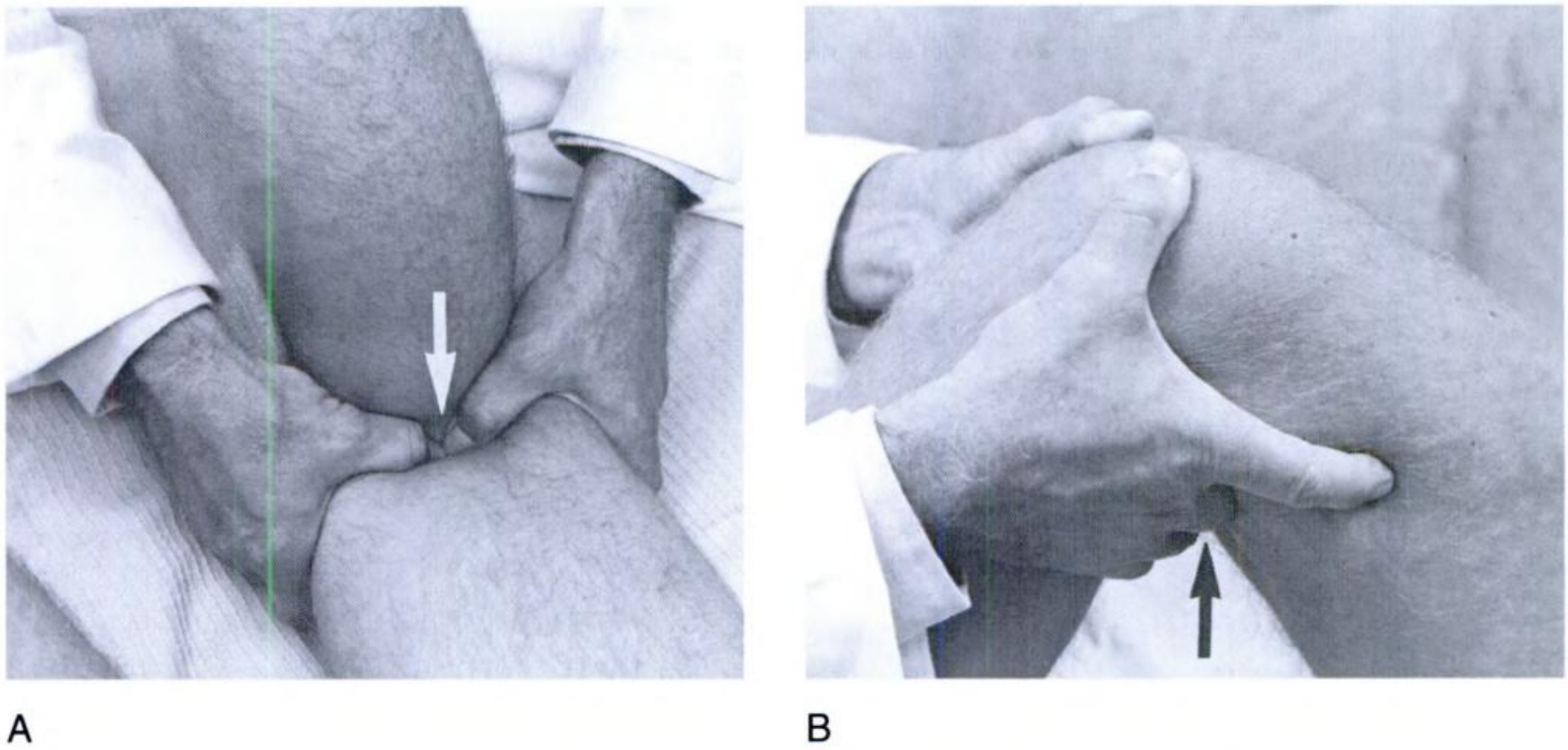


Figure 3–5 *A,* Palpation of the popliteal artery while the patient is prone with knees flexed and muscles relaxed by resting the leg against the examiner's shoulder. The midline of the popliteal fossa is firmly pressed with both thumbs (*arrow*). *B,* Alternative method of palpating the popliteal artery is more convenient but less sensitive. With the patient supine, the knee is flexed while the fingers of both hands are firmly applied to the midline of the popliteal fossa (*arrow*).

coarctation of the aorta because diagnostic differences in arm and leg pressures are based upon *systolic*, not diastolic levels. The *auscultatory popliteal* systolic pressure is normally about 10 mmHg higher than the *intra-arterial femoral* systolic pressure, but the diastolic pressures are the same.

If the above technique for determining lower extremity blood pressure proves unsatisfactory, the patient can be returned to the supine position and an arm cuff applied to the lower half of the calf (Fig. 3–4*B*). Korotkoff sounds are then sought by placing the bell of the stethoscope over the dorsalis pedis or for a better skin seal, over the posterior tibial artery after identifying these pulses by palpation. The disadvantages of this method are that it cannot be used in infants, and Korotkoff sounds are at best inconsistently heard over the dorsalis pedis or posterior tibial arteries in older patients even when those arteries are palpable. Exercising the limb sometimes increases audibility of Korotkoff sounds from these two arteries.

Cardiac Rate and Rhythm

> The point first to be noted is the frequency—the number of beats per minute—the regularity or irregularity of the beats as to time, and their equality or inequality in force. This is simple and easy.[3]

Heart rate is usually determined by palpating an arterial pulse, although cardiac auscultation is sometimes employed (see below). Accurate estimates of rate are now taken for granted, requiring nothing more than a palpable artery and a watch or clock with a

second hand. However, an appropriate time piece was long in coming as part of the clinician's bedside accoutrements:

> I caused a Pulse-Watch to be made which run 60 seconds, and I placed it in a box to be more easily carried, and by this I now feel pulses.[9]

Counting the pulse rate should encompass at least 30 seconds, but when the rhythm is irregular, a full minute provides a more accurate basis. Under certain circumstances—atrial fibrillation or marked pulsus alternans, for example—the rate of the palpable arterial pulse is slower than the ventricular rate, a pulse deficit readily identified by comparing a palpable arterial pulse rate with the heart rate as judged by simultaneous precordial auscultation. It is self evident that normal cardiac rates vary with age, from person to person, and under different circumstances in the same person. In 1890, Broadbent wrote:

> The average frequency of the pulse in the adult male is 72 beats per minute; in the female about 80; in the child it is much more frequent, and it gradually loses in frequency from infancy onwards. There are slight diurnal variations, but independently of any such influences, the pulse is more frequent in the evening than in the morning...and it would appear from various considerations that during a long night's sleep the circulation runs down in vigour as well, and not only in frequency.[3]

There are no deviations from the normal character of the pulse so easy to recognize as irregularities in rhythm.[1] Mackenzie went on to divide cases into two groups, namely, those in which the duration of systole was *regular* and those in which systole was *variable* in duration. Characterization of arrhythmias based on the arterial pulse is, with important exceptions, imprecise, but Mackenzie's grouping remains a useful point of departure. If the rhythm is irregular (variable duration of systole), it is necessary to determine whether the irregularity has a pattern. Simple atrial premature beats do not interrupt subsequent cycle lengths, provided the sinus node recovery time is normal. Conversely, ventricular premature beats are followed by *compensatory pauses*. In either case, it is apparent from the pulse that the basic rhythm is regular. A pronounced sinus arrhythmia sometimes clouds the picture and leads to the mistaken conclusion that the basic rhythm is irregular. This error can be avoided by determining the rhythm with the patient's breath held in the respiratory mid-position, a maneuver that abolishes sinus arrhythmia which is coupled to respiration.

Group beating generally means that the basic rhythm is regular but is systematically interrupted by recurrences of premature beats or cyclic absence of a beat. In 1899, four years before Einthoven's string galvanometer, Karel Frederik Wenckebach, using radial arterial pulse curves (sphygmograms) and a tuning fork for measuring time, described the periodicity that bears his name as an eponym.[10] In 1906, employing the newly developed electrocardiograph, Wenckebach demonstrated that PR intervals lengthened before the dropped beat, confirming his previous observations on second degree atrioventricular block in frogs.[10] Complete loss of regular rhythm (beat-to-beat variation in the duration of systole) is characteristic of atrial fibrillation. Even so, a *slow* ventricular

response in atrial fibrillation may be accompanied by differences in cycle lengths that are too subtle to be detected by palpation of the arterial pulse. Multiple ventricular premature beats sometimes disturb the basic rhythm to a degree that precludes confident distinction from atrial fibrillation. Examination of the jugular venous pulse serves to make this distinction (see Chapter 4).

The Valsalva maneuver, described in detail in Chapter 6, is mentioned here because the associated changes in heart rate are detectable at the bedside.[11] Phase 2, specifically is accompanied by reflex tachycardia and a small pulse, while Phase 4 (overshoot) is accompanied by reflex bradycardia. Congestive heart failure results in loss of the reflex tachycardia of Phase 2 and of the reflex bradycardia of Phase 4. Accordingly, determination of the pulse rate during Phases 2 and 4 of the Valsalva maneuver is a simple bedside method that sheds light on left ventricular function.

One of the most important aspects of examination of the arterial pulse is analysis of **waveform**. A necessary precondition is an appreciation of the waveform of the *normal* arterial pulse in all ages under varying conditions of relaxation and stress.

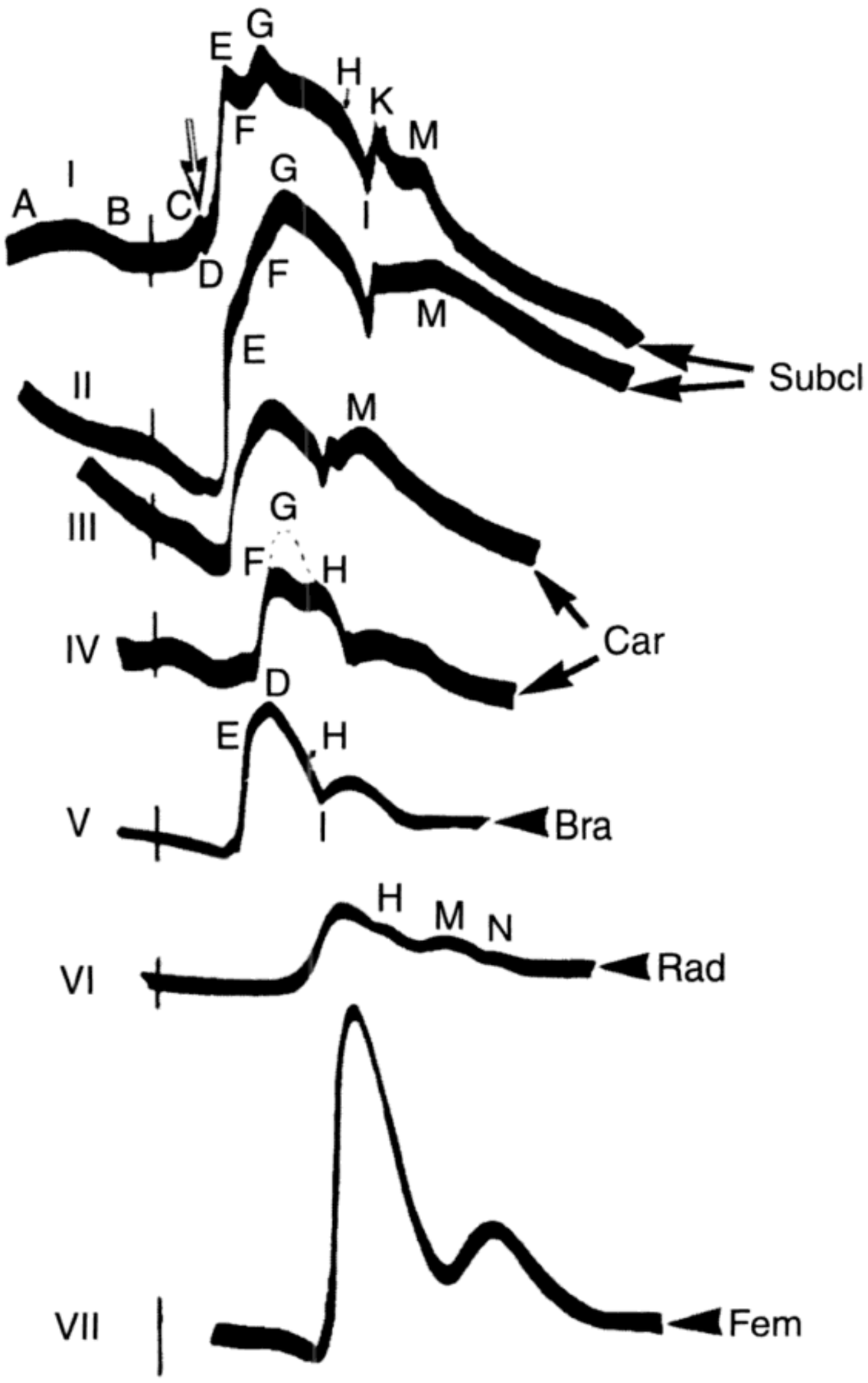


Figure 3–6 Retraced pulse curves from different arteries showing modification in contour from the central pulses (subclavian, "Subcl" and carotid, "Car") to distal pulses (brachial, radial, femoral). (Arrows and labels are my additions.) (From Wiggers CJ: The Pressure Pulses in the Cardiovascular System. London, Longmans, Green and Co, 1928, reprinted with permission.)

Assessment of waveform begins with selection of the most appropriate pulse for examination. The basis for judgment depends on the clinical objective, the structural state or physical properties of the arterial wall, and the patient's age. Because a prime objective is to secure the most accurate representation of the *central* arterial pulse, it is well to remember a point made by Carl Wiggers (Fig.3–6):

> By the time the pulse wave reaches the radial artery, friction has modified the fundamental contour and in addition has wiped out all of the smaller oscillations which exist in the central pulse.[12]

The carotid pulse (Fig. 3–7) provides a relatively close approximation of the waveform of the central aortic pulse and is readily accessible for palpation except in infants. Before palpating the carotid, however, certain variables must be taken into account, such as atherosclerotic obstruction in adults, kinking of the right carotid (see below), and the presence of a thrill or shudder. Alternatively, certain waveforms are more obvious in *peripheral pulses* such as pulsus alternans which is best detected in radial and femoral arteries, and pulsus bisferiens which is best detected in the brachial artery. However, these exceptions do not reduce the value of information derived from palpation of the carotid pulse in older children and adults.

The *technique of palpation* is all-important. It is best to begin the examination of the waveform of the arterial pulse by palpating the brachial arteries which are readily accessible from infancy through adulthood. Start with the right brachial pulse (Fig. 3–8). Mackenzie emphasized that "One's whole attention should be concentrated upon the observation." Physicians in Ancient China were enjoined to banish all other thoughts from their minds in order to concentrate on the pulse. Although these directives are excessive in contemporary context, it still holds that, without care and attention to arterial pulse waveform, errors are bound to occur.

Routine palpation of the *right brachial pulse* is best achieved with the thumb of the examiner's right hand as the patient's arm lies supinated (Fig. 3–8*A*). Alternatively, the patient's elbow can rest in the palm of the examiner's right hand, while the free left hand passively raises and lowers the patient's forearm to achieve maximum relaxation of muscles around the antecubital fossa (Fig. 3–8*B*). In infants, gentle restraint of the right hand or wrist can reduce undesirable arm movement, but forceful restraint must be avoided (see below).

The ease with which palpation of the brachial pulse is achieved is influenced by the thickness and compressibility of surrounding tissues and by the firmness of the bed upon which the artery rests. Accordingly, a brachial artery is easier to palpate when it is relatively superficial and rests on a firm support. The pulse is difficult to palpate in an obese or muscular arm. Pulsatile, tortuous, thick-walled brachial arteries in elderly adults with Monckeberg's sclerosis (Fig. 3–3) may roll away when palpated, but despite this difficulty, the thumb can almost always fix the serpentine artery against its bed.

Once the brachial artery is identified, progressive gentle pressure is exerted by the thumb until the maximum systolic impact of the pulse is elicited. The examiner should

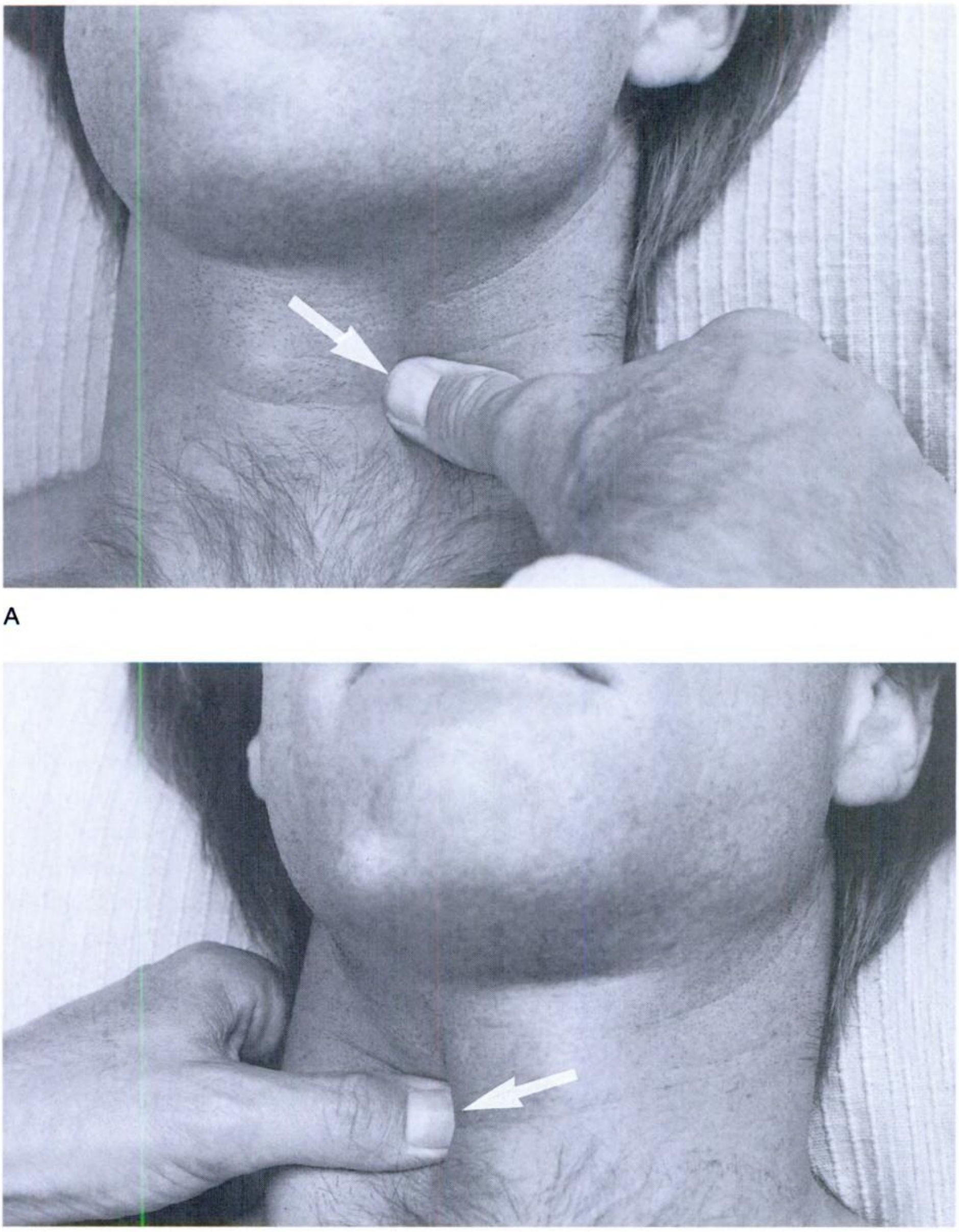
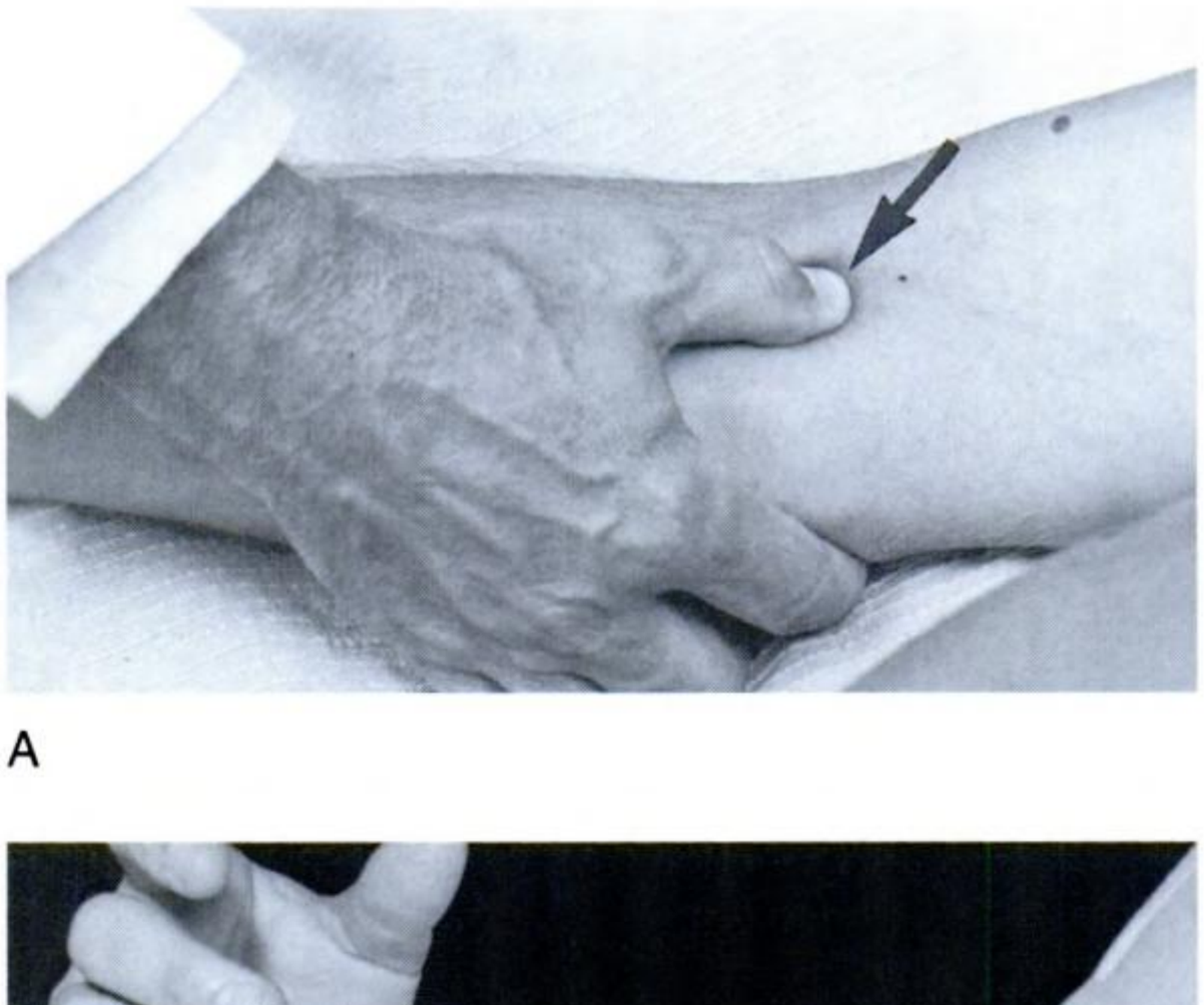


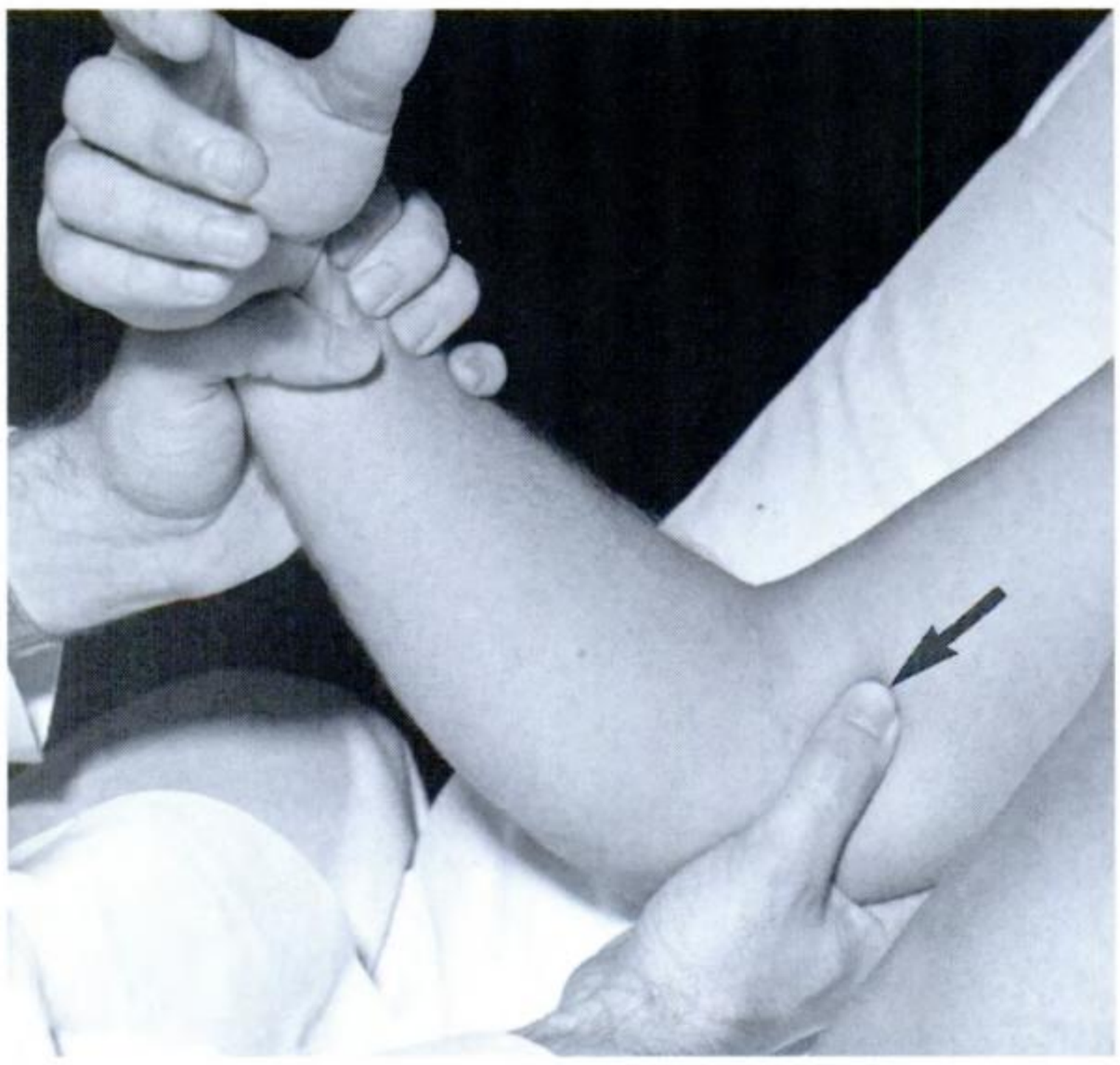
Figure 3–7 Palpation of the carotid pulse. *A,* The examiner places the right thumb (*arrow*) on the right carotid artery. *B,* The left thumb (*arrow*) is then applied to the left carotid which is used to time the contralateral jugular venous pulse.

then vary the pressure ever so slightly while forming a visual image of the components of the waveform (Fig. 3–9).

Palpation of the *carotid* pulse is best achieved with the examiner at the patient's right side. The right thumb is applied to the patient's left carotid (Fig. 3–7*A*), and the



A



B

Figure 3–8 *A*, Palpation of the right brachial pulse with the thumb (*arrow*) while the patient's arm lies at his or her side palm up. *B*, Palpation of the right brachial pulse while the patient's elbow is supported by the palm of the examiner's hand. The thumb is applied to the antecubital fossa (*arrow*) while the patient's forearm is passively raised and lowered to achieve maximum relaxation of muscles around the elbow.

left thumb is applied to the patient's right carotid (Fig. 3–7*B*). This technique permits application of the thumb without awkward bending of the wrist that decreases sensitivity in the fingertips. It is obvious that simultaneous palpation of both carotids is ill-advised, especially in elderly adults. Sequential palpation avoids risk while permitting accurate comparative assessment. The thumb should gently and slowly exert pressure on the carotid pulse to elicit the maximal systolic impact as described above. The artery should be palpated in the lower third of the neck to avoid inadvertent stimulation of the carotid sinus, especially in elderly adults.

Palpation of the *femoral arterial* pulse is best achieved with the examiner at the patient's right side. The right thumb is applied to the patient's left femoral artery, and the left thumb is applied to the right femoral artery (Fig. 3–10*A*). The examiner's right thumb is used for comparative palpation of the left brachial pulse (Fig. 3–10*B*).

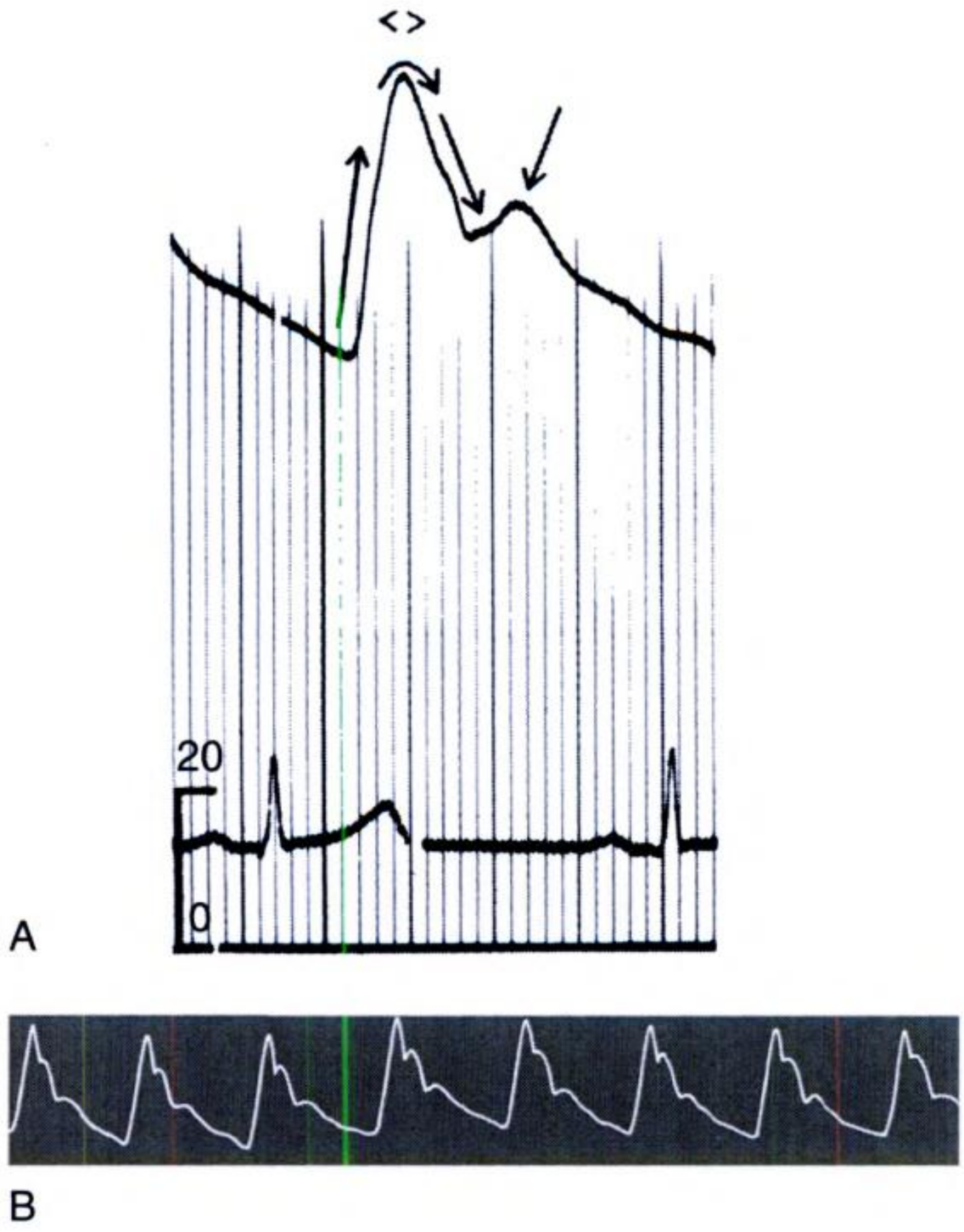


Figure 3–9 *A,* Normal intra-arterial brachial pulse showing an ascending limb, peak, descending limb, and dicrotic wave. *B,* Normal indirect radial pulse recorded by Mackenzie with his polygraph (see Fig. 3–1) applied to the wrist. (From Mackenzie J: The Study of the Pulse, Arterial, Venous, and Hepatic, and of the Movements of the Heart. Edinburgh, Young J. Pentland, 1902.)

The radial pulse was the time-honored site for palpation when females were not to be disrobed for examination (see Fig. 3–2). Mackenzie gave impetus to use of the radial pulse because it was the most convenient site to which he could fix his polygraph (see Fig. 3–1). However, the modified contour of the radial pulse[12] makes it less useful than the more central brachial and carotid pulses (see Fig. 3–6), except for detection of pulses alternans (see below). In infants, palpation of the radial pulse has inherent limitations (James Mackenzie 1902):

> The infant's radial artery is naturally very small. We are not conscious of its presence, being unable to differentiate it from the surrounding structures, and we only recognize its pulse when the finger presses the artery against the bone. This inability to feel the artery as a distinct structure is due not only to the smallness of the vessel, but to the fact that very often the padding of subcutaneous fat is relatively great in the very young.[1]

For palpation of the radial pulse, the patient's hand should be supinated. The examiner's thumb (Fig. 3–11*A*) or the tip of the index finger (Fig. 3–11*B*) is then applied. The index finger is somewhat more sensitive than the thumb, an observation used to advantage when palpating a relatively small pulse, such as the radial.

Normal Arterial Pulse

Recognition of abnormal waveforms assumes an understanding of the waveform of the normal arterial pulse (see and Fig. 3–9) which varies according to physiologic state, patient age, and distance from the aortic root (see Fig. 3–6).

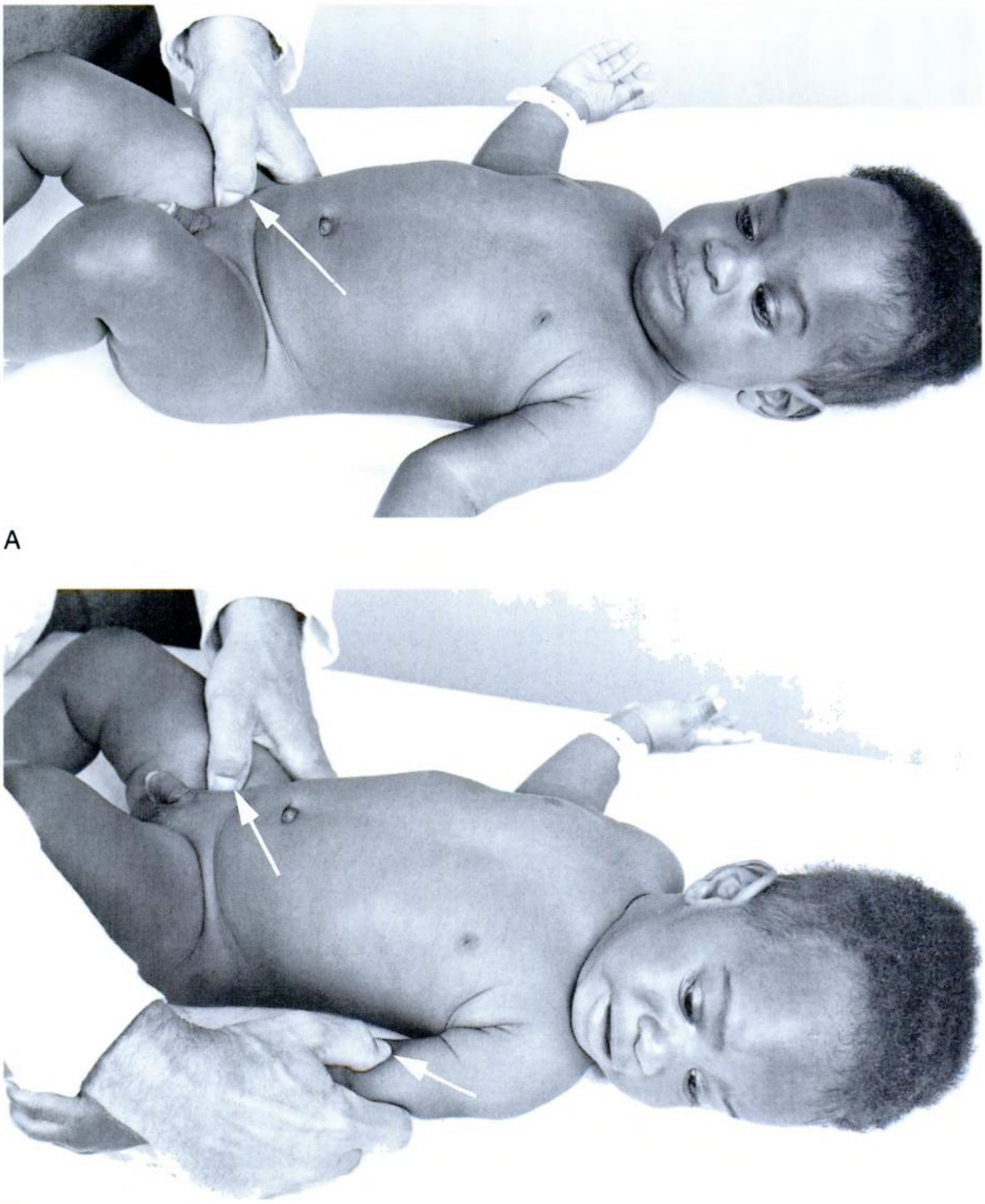
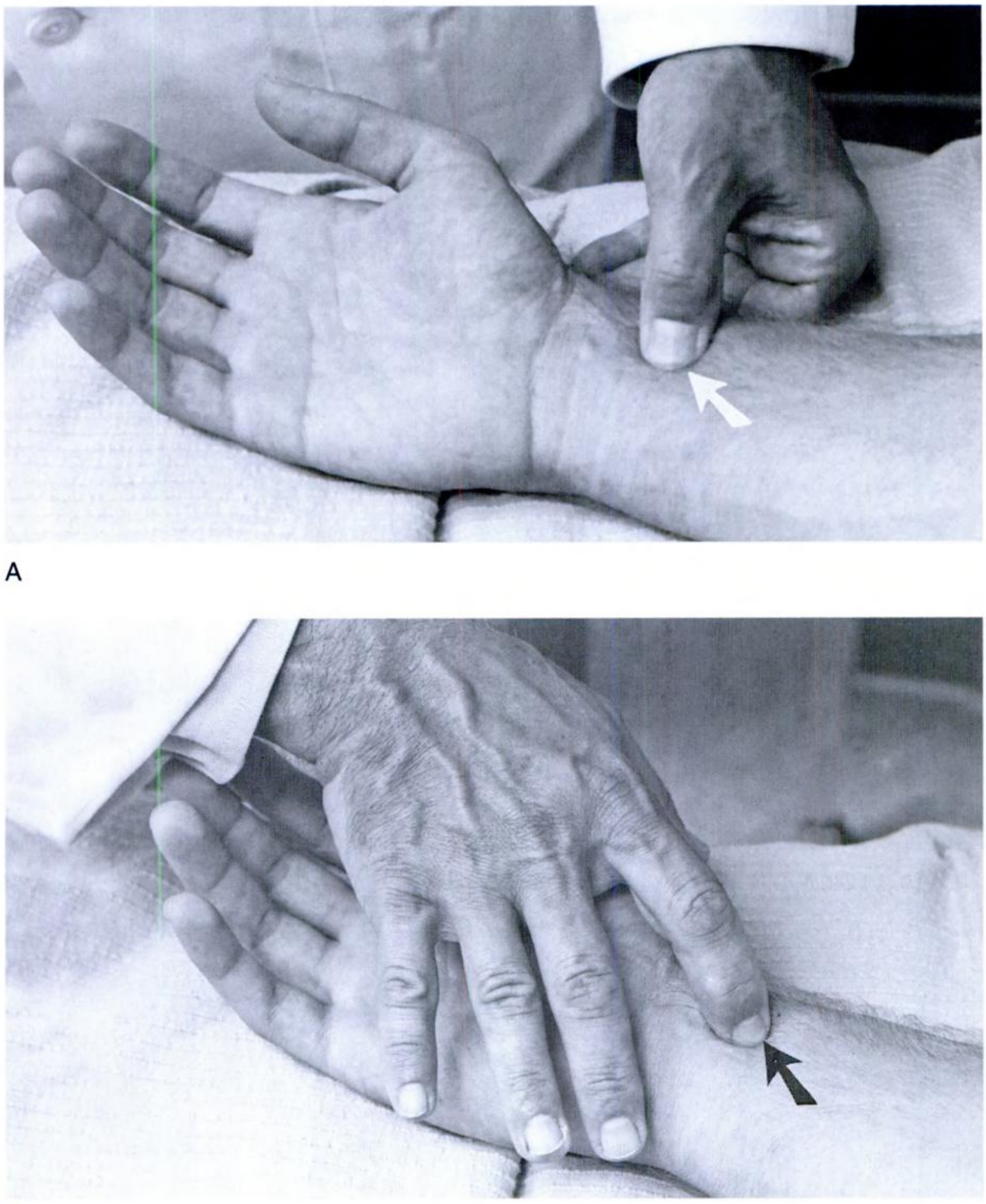


Figure 3–10 Palpation of the femoral pulse. *A,* The examiner's left thumb is applied to the patient's right femoral pulse (*arrow*), then *vice versa*. *B,* With one thumb palpating a femoral artery, the free thumb is applied to the contralateral brachial artery (*arrow*) for simultaneous comparative assessment.

Mackenzie's description of the normal arterial pulse still applies:

> There is first an abrupt rise, then a fall, followed by a continuation of the wave at about the same level. This period is usually described as being divided into two, the abrupt rise spoken of as the primary or percussion wave, and the latter portion



A

B

Figure 3–11 Palpation of the radial pulse. The thumb *A* or the index finger *B* is applied to the radial artery (*arrows*).

as the tidal or predicrotic wave. With the closure of the aortic valve, the aortic pressure falls rapidly to the bottom of the aortic notch. This fall is interrupted by a distinct rise in pressure, represented by the dicrotic wave.[1]

In characterizing the normal arterial pulse from childhood to young adulthood, attention is paid to the ascending limb, the peak, and the descending limb (see Fig. 3–9).

Table 3–2 Abnormal Waveforms

Diminished	Small, hypokinetic, with or without a delayed, sustained peak
Increased	Hyperkinetic with or without a wide pulse pressure
Double peaked	Systolic or systolic/diastolic

With advancing age, reduced arterial distensibility (see section on Blood Pressure) results in a steeper rate of rise, a higher peak, and a more rapid descent. Otherwise, the ascending limb normally rises to a single, somewhat rounded peak. The descending limb is less steep, and is interrupted by an incisura (ie, a brief downward displacement associated with closure of the aortic valve). The pulse then rises slightly (dicrotic wave), before gradually falling throughout diastole. The incisura, the dicrotic wave, and the anacrotic notch (see Fig. 3–6) are seldom palpable.

Abnormal Arterial Pulse

Abnormal waveforms of the arterial pulse are designated as diminished (small, weak, hypokinetic), increased (large, strong, hyperkinetic), and double-peaked (double systolic peak or systolic/diastolic peaks) (Table 3–2).

A **diminished arterial pulse** is sensed as a gentle impact because the rate of rise is decreased. The peak is relatively distinct but reduced, ill-defined, and sometimes delayed. A diminished, weak, hypokinetic arterial pulse occurs with depressed left ventricular function. In severe aortic stenosis, the arterial pulse is also diminished with a slow ascending

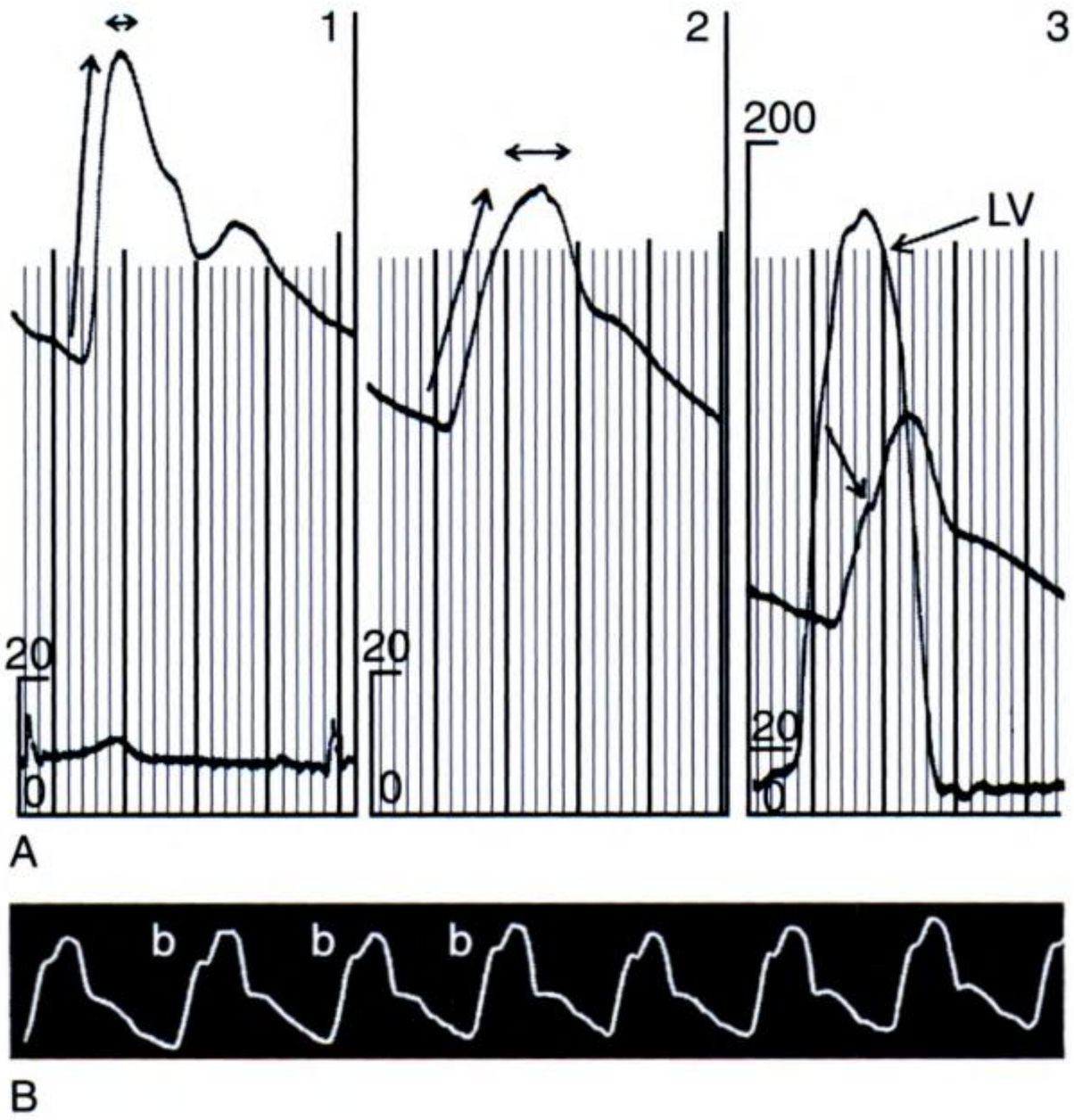
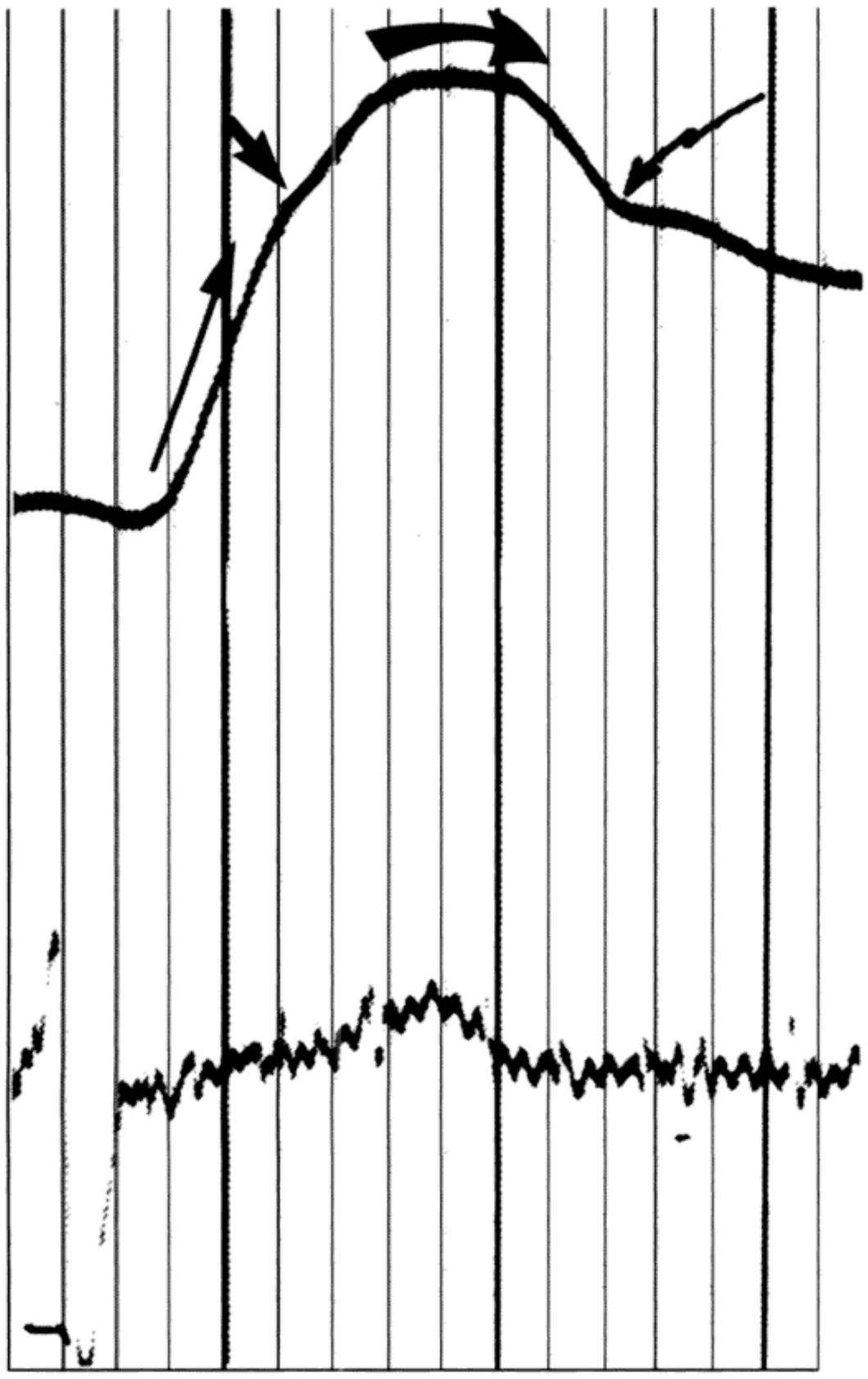
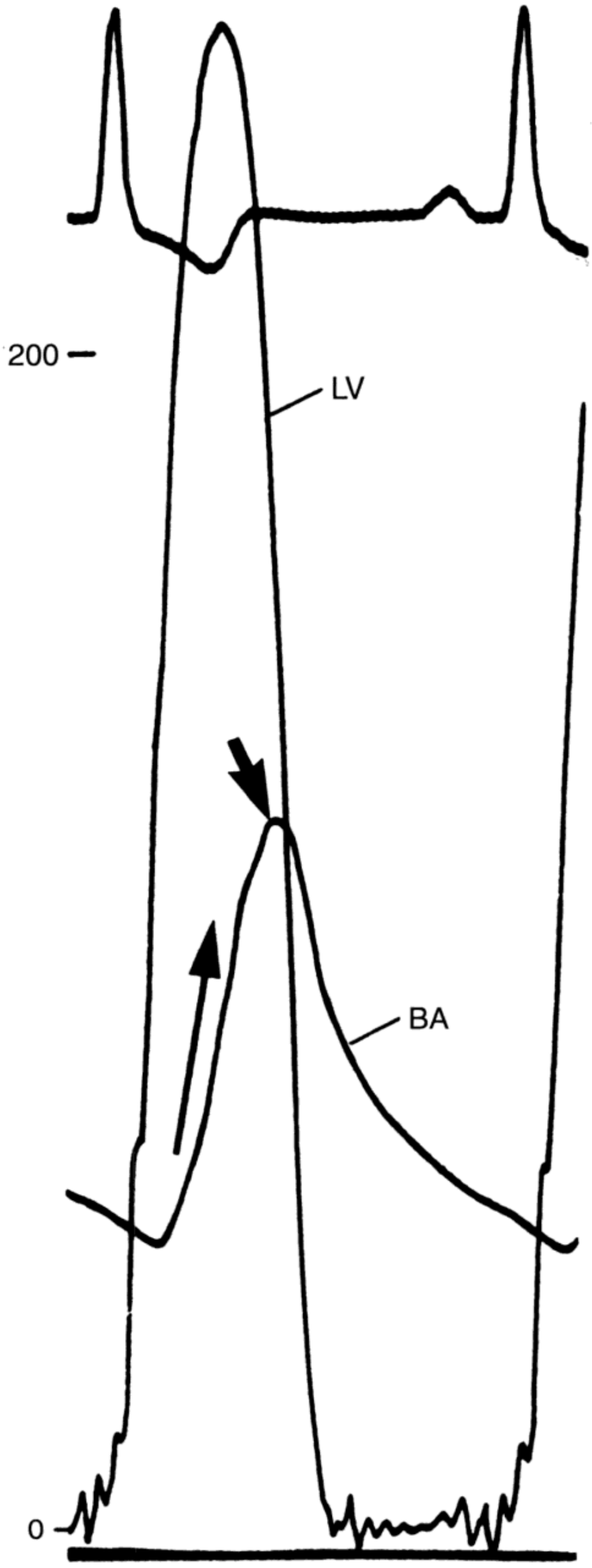


Figure 3–12 *A,* Panel 1 shows a normal brachial arterial pulse for comparison with Panel 2, the pulse in aortic valve stenosis, that shows the typical slow rate of rise and delayed, broad crest. Panel 3 shows simultaneous left ventricular (LV) and brachial arterial pulses of aortic stenosis. The lower left arrow identifies a non-palpable anacrotic notch. *B,* "Anacrotic pulse in a case of aortic stenosis." (From Mackenzie J: The Study of the Pulse, Arterial, Venous, and Hepatic, and of the Movements of the Heart. Edinburgh, Young J. Pentland, 1902.)



A



B

Figure 3–13 *A,* Indirect right brachial arterial pulse and electrocardiogam from a young male with severe bicuspid aortic stenosis. There is a characteristic slow rate of rise (*first arrow*), a non-palpable anacrotic notch (*second arrow*), a broad crest (*curved arrow*), and a non-palpable dicrotic notch (*fourth arrow*). *B,* Left ventricular (LV) and brachial arterial (BA) pulses from an elderly male with acquired calcific stenosis of a trileaflet aortic valve. The rate of rise of the brachial arterial pulse is *not* slow (*first arrow*), and the crest is *not* broad (*second arrow*).

limb (parvus) and an ill-defined late peak (tardus) (Figs. 3–12*A* and 3–13*A*). Mackenzie wrote (Fig.3–12*B*):

> When there is marked narrowing of the aortic orifice, the full effect of the ventricular systole upon the arterial column is not at once developed, as the aortic stenosis offers an increased resistance. Hence, the impact of the pulse wave on the

> finger is not sudden, but feels to push against the finger in a somewhat leisurely fashion. The tracing in such a case represents a slanting of the stroke with an interruption near the top—an anacrotic-pulse tracing.[1]

An exception is the pulse in elderly patients with calcific aortic stenosis, in whom decreased systemic arterial compliance (stiffness) accelerates the rate of rise and counters the sustained peak (Fig. 3–13*B*).

A **brisk arterial pulse** with normal pulse pressure and a single systolic crest characterizes the rapid ejection of severe mitral regurgitation with hyperkinetic left ventricular contraction (Fig. 3–14). A wide pulse pressure with double systolic crests (bisferiens) characterizes the rapid ejection of pure severe aortic regurgitation. It is open to question whether the rapid *fall* of an arterial pulse (Fig. 3–15*A*) is sensed by palpation. The distinction between an increased arterial pulse with or without wide pulse pressure cannot always be established by palpation, but an attempt can be made by determining the degree of brachial arterial compression required to obliterate a simultaneously palpated radial pulse. A brisk arterial pulse with *normal* pulse pressure requires only normal brachial compression to obliterate the radial pulse. If the pulse pressure is *increased*, it must then be determined whether the increase is due to *systolic hypertension* (older adult) or to systolic hypertension with low *diastolic* pressure in aortic regurgitation (Figs. 3–15*A* and 15*B*), or to a large left-to-right shunt through a patent ductus arteriosus (Fig. 3–16). When a wide pulse pressure is due to high systolic and low diastolic pressure of severe aortic regurgitation, the rate of rise is not only brisk, but markedly so (Figs. 3–15 and 3–16).

The *Corrigan pulse* refers to the *visible* pulsations of severe aortic regurgitation described by Dominic J. Corrigan in 1832:

> When a patient affected by the disease is stripped, the arterial trunks of the head, neck and superior extremities immediately catch the eye by their singular

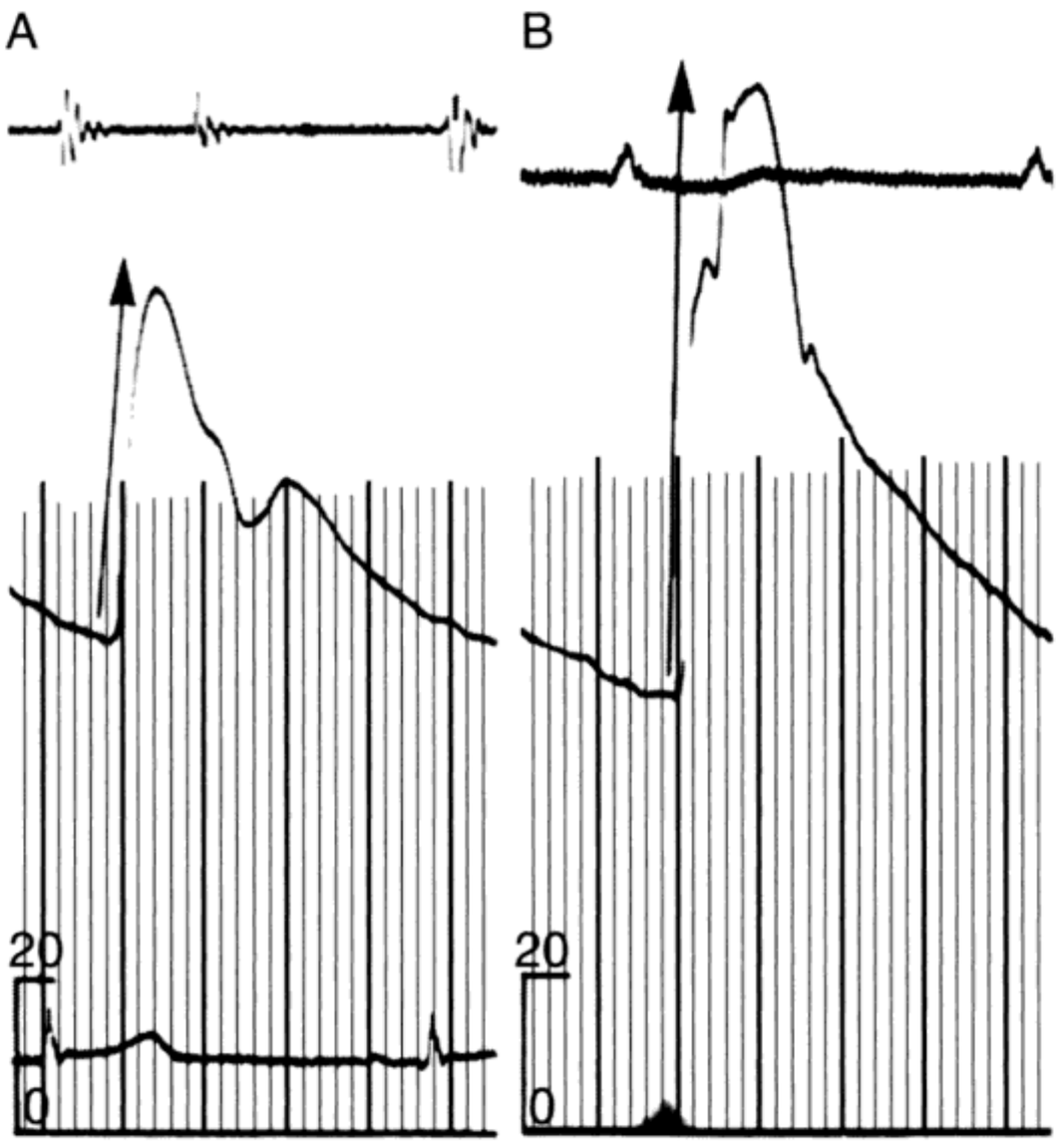


Figure 3–14 *A*, Normal brachial arterial pulse for comparison with the brachial pulse in pure severe mitral regurgitation (*B*) that exhibits a brisk rate of rise (*arrow*) with only a modest increase in pulse pressure.

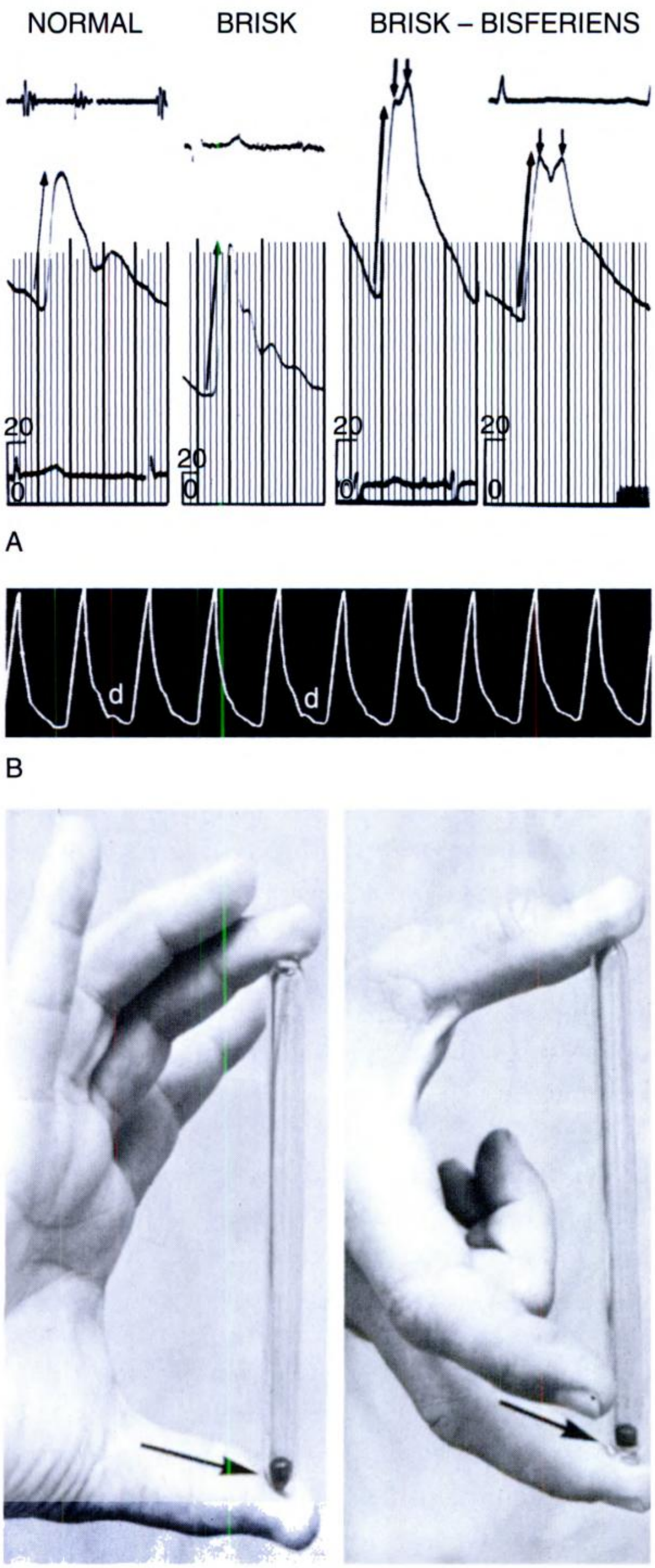


Figure 3–15 *A, First panel*—Normal brachial arterial pulse. *Second panel*—Brisk arterial pulse with wide pulse pressure but single brief crest. *Third panel*—Brisk bisferiens pulse of pure severe aortic regurgitation with a rapid rate of rise and two unequal crests *(small vertical arrows)*. *Fourth panel*—Brisk bisferiens pulse with a rapid rate of rise and two equal crests (*small vertical arrows*). *B,* "Pulse of extreme aortic regurgitation." (From Mackenzie J: The Study of the Pulse, Arterial, Venous, and Hepatic, and of the Movements of the Heart. Edinburgh, Young J. Pentland, 1902.) *C,* Toy water hammer consisting of a sealed glass tube containing mercury (*arrows*) in a vacuum. As the tube is quickly inverted back and forth, the mercury falls abruptly from one end to other, imparting a jolt or impact to the thumb or fingertip.

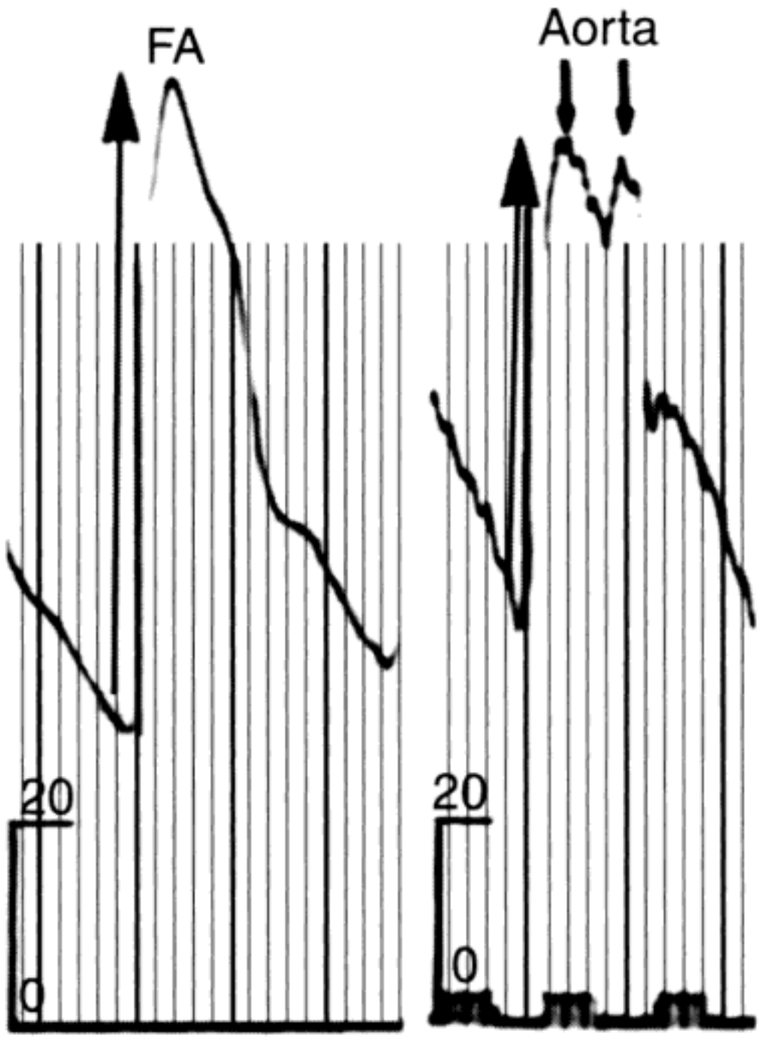


Figure 3–16 *First Panel*—The *femoral arterial* (FA) pulse in an infant with large patent ductus arteriosus. The rate of rise is brisk (*arrow*), the pulse pressure is wide, and the systolic crest is single. *Second Panel*—The *central aortic* pulse in the same patient is bisferiens *(double arrows)*.

pulsation. From its singular and striking appearance, the name of *visible pulsation* is given to this beating of the arteries. It is much more marked in the arteries of the head and neck in the erect than in the horizontal posture; and a patient suffering under the disease himself, first points it out.[13]

The wide pulse pressure of free aortic regurgitation has been compared to a *water hammer*, a term coined in 1844 by Thomas Watson, an English physician. The water hammer was a popular toy in Victorian England and consisted of a sealed glass tube containing mercury in a vacuum. Solids and liquids fall at the same rate in a vacuum, so when the glass tube is quickly inverted, the mercury column falls abruptly from one end of the tube to the other (see Fig. 3–15*C*), imparting an impact or jolt to the fingertip at the inverted end. The Victorian toy has long since vanished, but the term is still applied the arterial pulse of pure free aortic regurgitation.

Double-peaked pulses are of two types—those with two systolic peaks and those with one systolic and one diastolic peak. The term "double-peaked" is preferable, because the two crests are not necessarily identical (see Figs. 3–15*A* and 3–16). By convention, the double-peaked *systolic* pulse is designated *bisferiens*, whereas a paired systolic/diastolic pulse is designated *dicrotic* (Fig. 3–17). The two peaks of a bisferiens pulse are often palpable, but that is not the case with a dicrotic pulse. However, terms matter less than clarity. It is not beside the point that, literally translated, "dicrotic" and "bisferiens" mean the same thing. *Dicrotic* has a Greek root (*di,* "twice," and *krotos*, "beat"). *Bisferiens* is Latin for "beat twice."

The term *anacrotic* is unimportant at the bedside as a designation for systolic double peaking because the anacrotic pulse is seldom palpable even when clearly recorded (see Figs. 3–12 and 3–13*A*).

Double systolic peaks are most commonly represented by the bisferiens pulse of pure aortic regurgitation (see Fig. 3–15*A*). The two peaks can be equal, or the first or second crest can be the larger of the two (see Figs. 3–15*A* and 3–16). The two

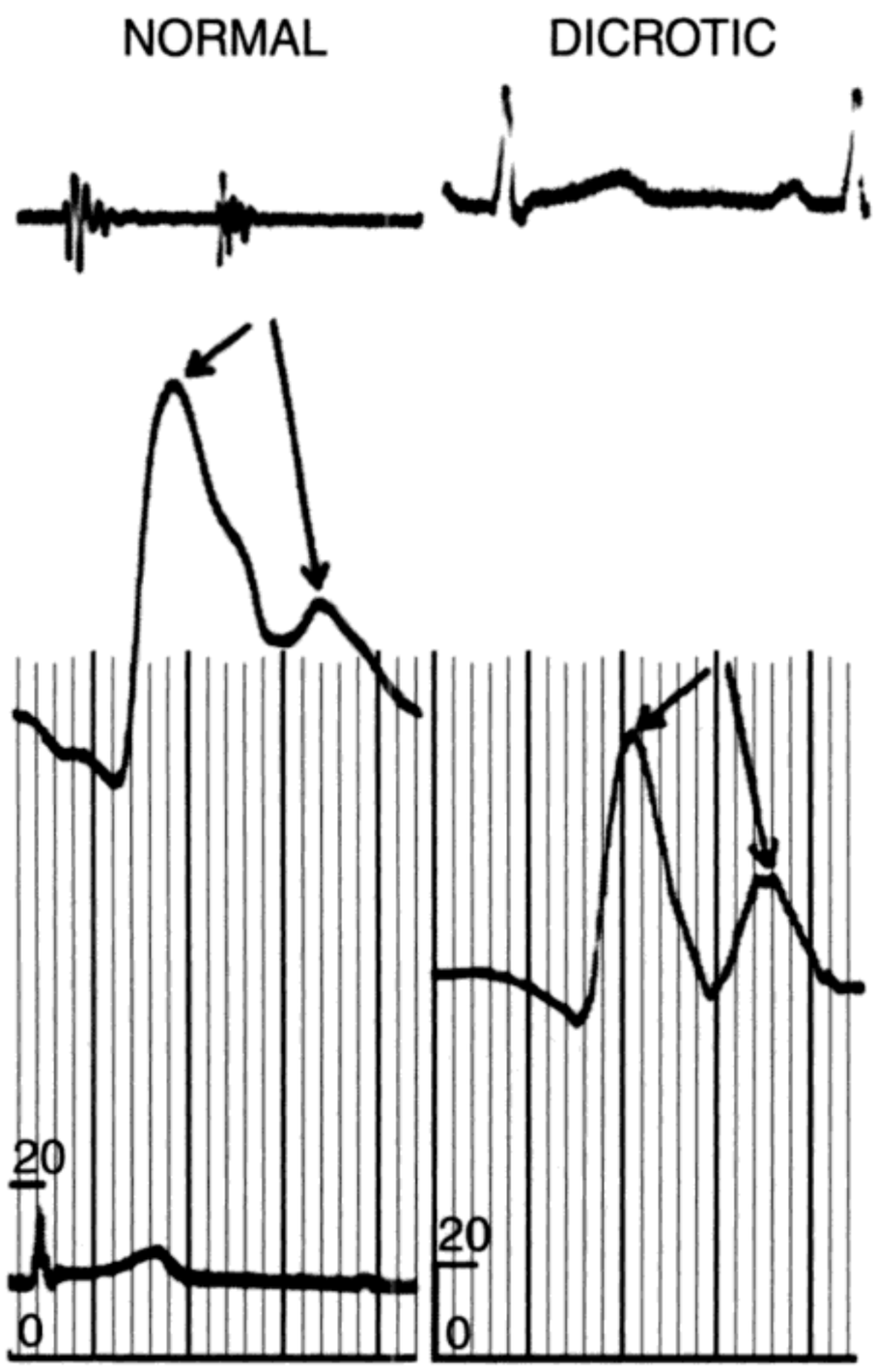


Figure 3–17 First panel shows a *normal brachial arterial pulse*. Second panel shows a *dicrotic pulse* characterized by a single systolic crest *(short arrow)* and an amplified dicrotic wave *(long arrow)*.

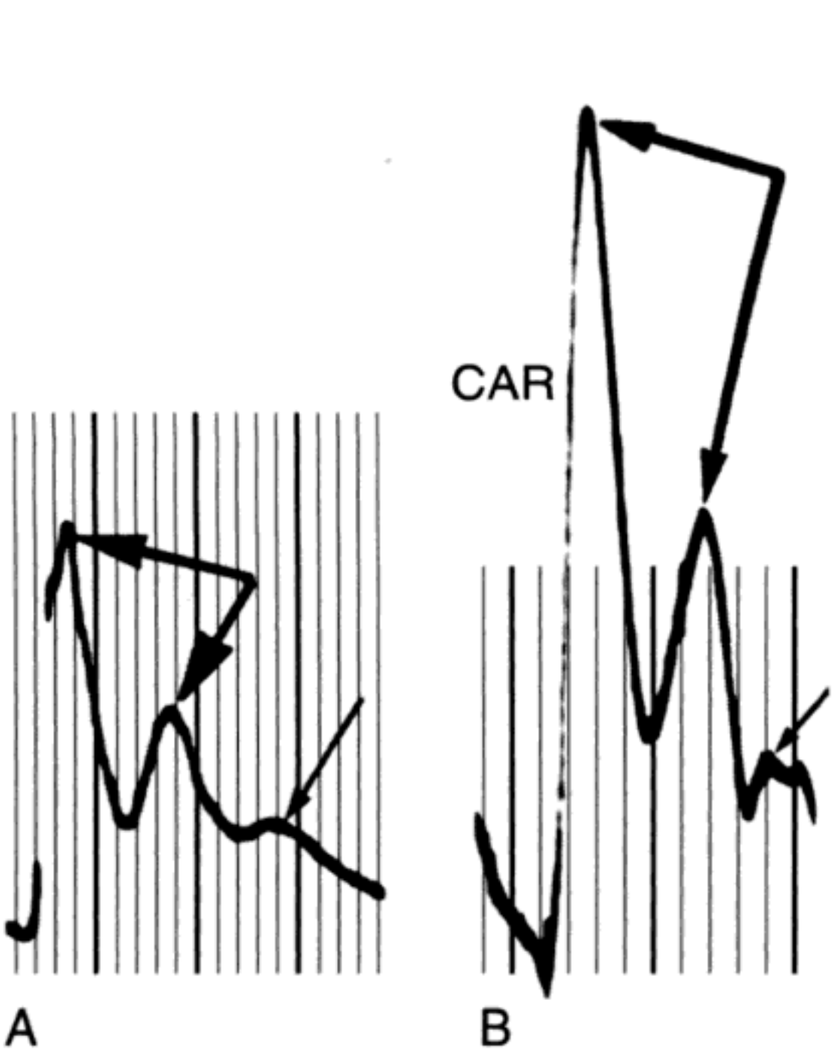


Figure 3–18 *A,* Direct brachial pulse and *B,* indirect carotid pulse (CAR) from two patients with hypertrophic obstructive cardiomyopathy. Similar waveforms show brisk rates of rise and double systolic peaks *(bold arrows)*. *Thin arrows* point to low-amplitude dicrotic waves, confirming the systolic timing of the two preceding crests.

peaks are not only palpable but are sometimes audible as double Korotkoff sounds. Double systolic peaks of the arterial pulse also occur in hypertrophic obstructive cardiomyopathy, but palpation usually detects only the brisk rise and sharp initial peak (Fig. 3–18).

Pulsus alternans, originally described by Traube in 1872,[14] refers to alternation of the *strength (force* or *impact*) of the pulse sensed by palpation (Fig. 3–19*A*) in the

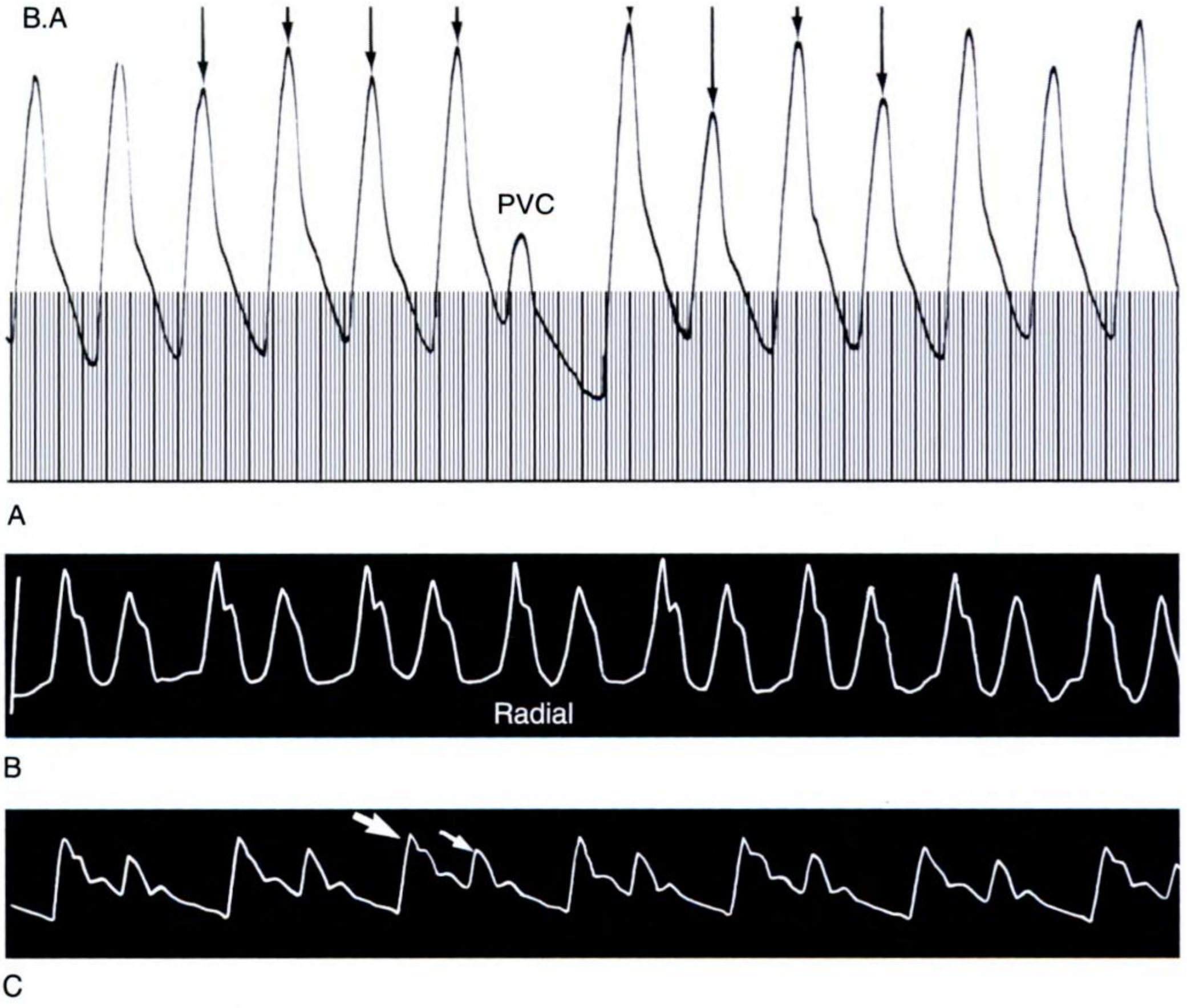


Figure 3–19 *A,* Brachial arterial (BA) pulse showing typical *pulsus alternans* (*vertical arrows*) that is exaggerated after a premature ventricular contraction (PVC). There is beat-to-beat alternation in peak systolic pressures and alternation of the rates of rise of the ascending limbs. *B,* "Radial pulse showing the rhythmical irregularity (pulsus alternans)."[1] *C,* The strong/weak beats of a bigerninal pulse recorded by Mackenzie must be distinguished from the strong/weak beats of pulsus alternans as illustrated above. (Arrows my additions) (*B* and *C* are from Mackenzie J: The Study of the Pulse, Arterial, Venous, and Hepatic, and of the Movements of the Heart. Edinburgh, Young J. Pentland, 1902.)

absence of an arrhythmia (Fig. 3–19*B*) or of a significant variation in interval between beats. Pulsus alternans must be distinguished from the alternating strong/weak beats of a bigeminal pulse in which the weaker beat is premature (Fig. 3–19*C*). Traube drew a careful distinction between pulsus bigeminus and pulsus alternans.[14]

Pulsus alternans can also be identified by beat-to-beat differences in the intensity of Korotkoff sounds (see below). Alternation of a systemic arterial pulse is a sign of depressed left ventricular systolic function. Alternate beats differ from each other in their peak systolic pressures, but what is sensed by palpation is the alternating rates of rise of the ascending limbs (Fig. 3–19*A*). Alternation is sometimes so marked that the weak

beat is not perceived—total mechanical alternans—effectively halving the cardiac rate as judged by the arterial pulse.

Alternans tends to increase as the pulse wave moves peripherally. Accordingly, the radial and femoral pulses are more revealing than the carotids and brachials. Pulsus alternans is best elicited with the thumb applied to a femoral artery or the thumb or finger applied to a radial artery. The digital compression is gradually increased until the impact of the pulse is maximal. Slow release of compression discloses a decrease in impact of the alternating beats. The pulse should be examined for alternation during quiet breathing or with the breath held comfortably in the respiratory mid-position to avoid respiratory variations in the arterial pulse (see below). When quiet breathing or breath holding is impractical because of supine dyspnea, the radial pulse should be palpated for alternation while the patient sits with legs hanging over the bedside or examining table, a position that reduces or eliminates dyspnea, decreases venous return, and tends to exaggerates alternation. Nitroglycerin has the same effect. It is important to anticipate premature ventricular contractions that transiently provoke or exaggerate pulsus alternans, as shown in Figure 3–19*A*.

Pulsus alternans can also be detected by sphygmomanometry. The cuff is inflated to above systolic pressure and then is slowly lowered. Auscultation at the brachial artery initially detects Korotkoff sounds at half the ventricular rate. When the cuff pressure is lowered further, Korotkoff sounds suddenly double.

Pulsus paradoxus is a term that was introduced into clinical medicine by Adolph Kussmaul (1873) to describe the marked inspiratory fall in systemic arterial blood pressure associated with constrictive pericarditis.[15] The inspiratory decline is not really paradoxical, but instead is an exaggeration of the normal inspiratory decrease in systolic pressure. What Kussmaul referred to, however, was a pulse that dropped excessively hence *paradoxically* with inspiration despite the fact that the heart rate and rhythm remained unchanged.

In a healthy adult, normal inspiration results in no more than a 3 to 4 mmHg decline in systolic blood pressure. Even a relatively deep breath seldom causes a decline that reaches 10 mmHg. Accordingly, the term "pulsus paradoxus" applies when the inspiratory decrease exceeds 10 mmHg, especially during average respiratory excursions.

Pulsus paradoxus accompanies certain cardiac diseases and certain forms of pulmonary disease. In cardiac disease, a paradoxical pulse is most often associated with pericardial tamponade, less frequently with chronic constrictive pericarditis which was the basis for Kussmaul's original description (Fig. 3–20*A*). Pulsus paradoxus is characterized by an exaggerated fall in systolic *and* diastolic pressure because of an abnormal inspiratory fall in left ventricular volume. The most common *noncardiac* cause of a paradoxical pulse is pulmonary emphysema (Fig. 3–20*B* and 20*C*). A decrease in lung compliance coupled with airway obstruction causes an excessive fall in systolic and diastolic pressures, while exhalation is accompanied by an excessive rise in systolic pressure (Fig. 3–20*B* and 20*C*).

The trunk should be raised to a level that minimizes respiratory effort. The patient is instructed to breathe as regularly and quietly as comfort permits. The examiner's thumb is applied to a brachial arterial pulse with just enough pressure to elicit the maximal systolic impact. Respiratory movements are monitored by glancing at the

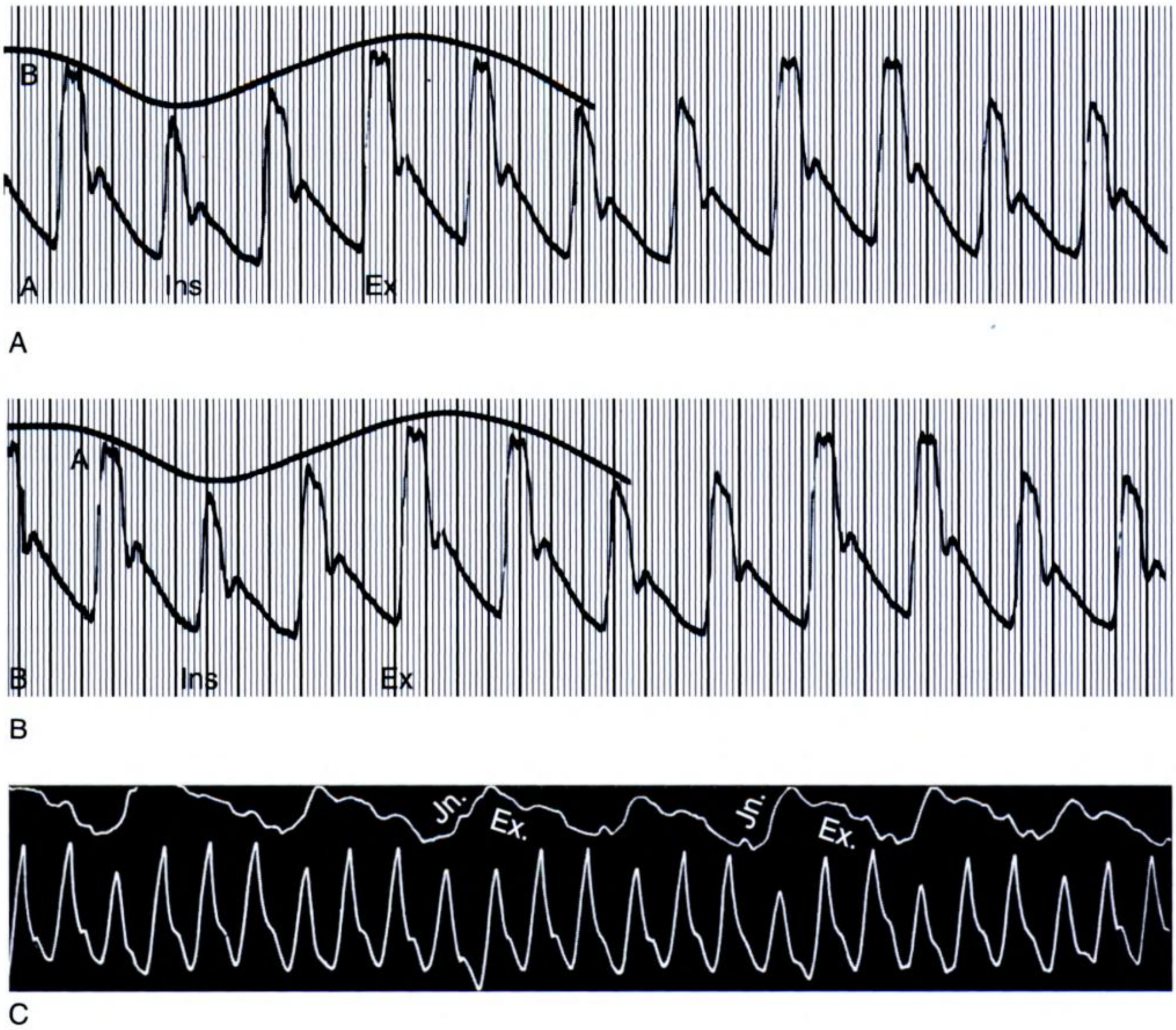


Figure 3–20 *A,* Direct brachial arterial (BA) tracing in a young adult with pulsus paradoxus caused by pericardial tamponade. There is an abnormal fall in systolic *and* diastolic pressures during inspiration (Ins). (Ex—exhalation). *B,* Direct BA pulse in an elderly adult with chronic obstructive lung disease and emphysema. During inspiration (Ins) there is an exaggerated fall in systolic and diastolic pressures, while during exhalation (Ex) there is an exaggerated rise in *systolic pressure. C,* Radial arterial pulse showing pulsus paradoxus in "...a case of capillary bronchitis and catarrhal pneumonia."[1] (From Mackenzie J: The Study of the Pulse, Arterial, Venous, and Hepatic, and of the Movements of the Heart. Edinburgh, Young J. Pentland, 1902.)

patient's chest or abdomen as brachial arterial compression is slowly decreased, seeking the point at which the pulse diminishes or even vanishes altogether during inspiration.

The paradoxical pulse is readily detected with a sphygmomanometer which is best reserved for clinical settings in which the anticipated paradox is not elicited by palpation. With the cuff inflated above systolic pressure, Korotkoff sounds are sought over the brachial artery while the cuff is deflated at a rate of approximately 2 to 3 mmHg per heartbeat. This method permits identification of peak systolic pressure during exhalation. The cuff is then deflated further in order to determine the level at which Korotkoff sounds are

audible during both exhalation *and* inhalation. Pulsus paradoxus is considered present when the peak systolic pressure levels at exhalation and inhalation exceed 16 mmHg.

Differential Pulsations and Selective Absence, Diminution, or Augmentation

Palpation can be used to grade an arterial pulse from absent to augmented and for comparison with established norms for age. Palpation also permits comparison of right vs left and upper vs lower extremity pulses (Figs. 3–10 and 3–21). A numerical system employs 0 to 4+ for grading an arterial pulse with 0 as absent, 1+ as present but diminished, 2+ as normal, 3+ as moderately increased, and 4+ as markedly increased . Information thus obtained is, in part, an extension of the preceding sections that dealt with normal and abnormal waveforms.

Palpation is most revealing when a single digit rather than several fingertips is used to grade and compare arterial pulses. Sensitivity differs from digit to digit, so application of more than one fingertip compromises the delicate control that is desirable when an artery is palpated. As a rule, the *thumb* is preferable for palpation (see earlier) because it can be moved on its metacarpophalangeal hinge without bending the wrist, thus avoiding a decrease in sensitivity of the fingertips. The thumb is also convenient for simultaneous or sequential comparison of right and left arterial pulses (Figs. 3–7 and 3–21), and for comparison of upper and lower extremity pulses (Fig. 3–10*B*).

Contralateral Pulses

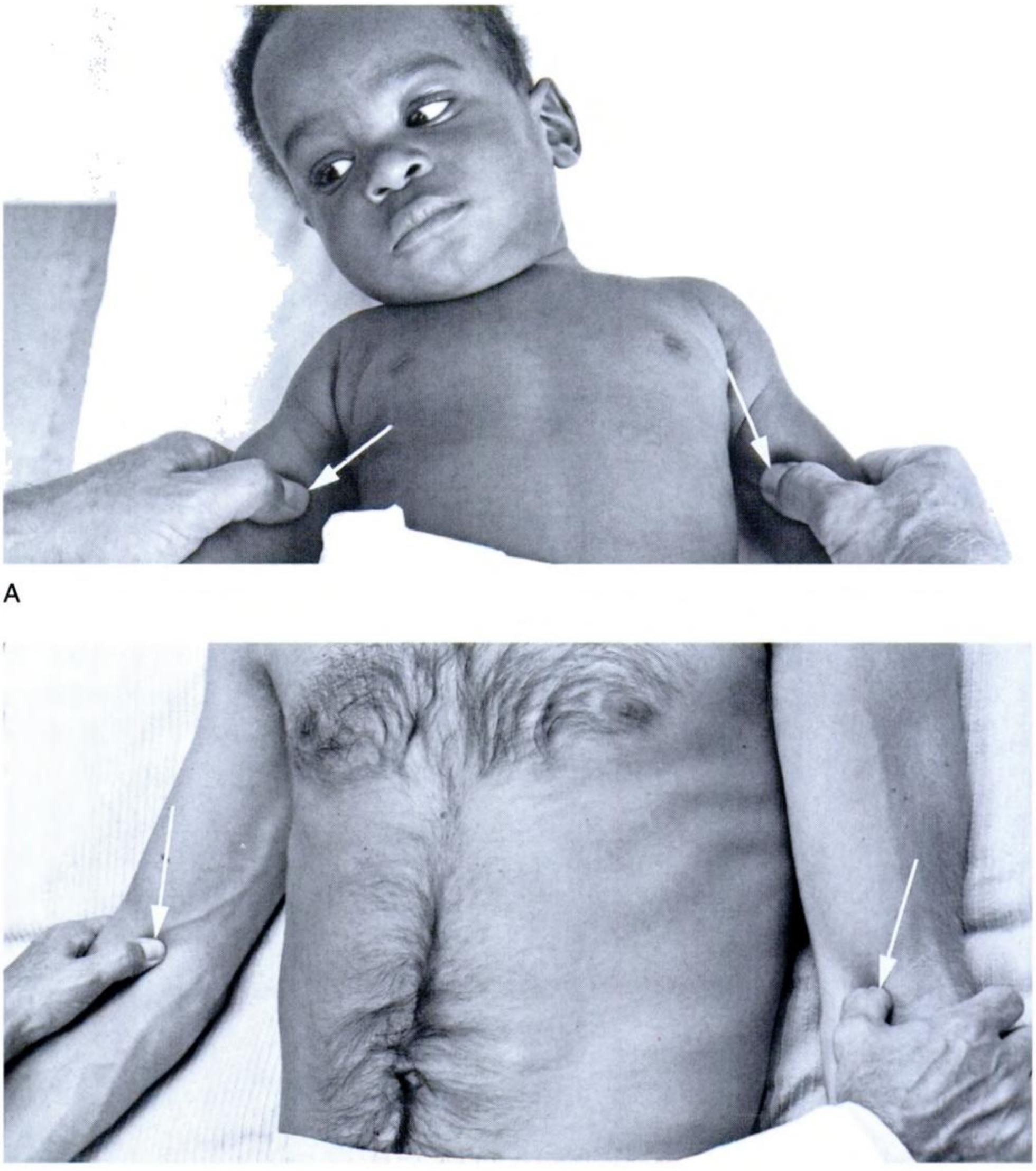
Techniques for comparing contralateral pulses are shown in Figure 3–21. *Brachial* pulses are compared by applying a thumb to the right and left antecubital fossae (Fig. 3–21*A*). In infants, the examiner must wait until the arms are voluntarily relaxed. Restraint is likely to provoke resistance, compromising rather than facilitating the examination. Brachial pulses are compared simultaneously or sequentially.

In elderly adults, the most common cause of *diminution or absence* of a brachial pulse is atherosclerotic obstruction. When coarctation of the aorta obstructs the orifice of the left subclavian artery, the *left* brachial pulse is diminished or absent in addition to diminished or absent femoral pulses, while the right brachial pulse is increased. When aortic stenosis is supravalvular, the *right* brachial pulse is greater than the left because of amplification of systolic pressure in the former.

Selective diminution of the *left* brachial pulse in adults, rarely in children, arouses suspicion of a *subclavian steal*. The suspicion is reinforced if palpation immediately above the left clavicle finds the ipsilateral subclavian pulse absent or diminished. Asymmetry of brachial pulses demands that blood pressure be determined in *both* arms irrespective of age.

The right and left *femoral arterial pulses* are compared by simultaneous application of the thumbs. Comparative palpation of the femoral pulses is obligatory in elderly patients in whom asymmetric diminution indicates atherosclerotic obstruction.

Carotid pulses should be routinely compared in adults, especially elderly adults. Prudence demands that comparison of right and left carotids be accomplished *sequentially*, not simultaneously (see Fig. 3–7), a precaution that minimizes the risk of reducing cerebral blood



A

B

Figure 3–21 Comparison of the right and left brachial arterial pulses in a child *A* and in an adult *B* by simultaneous palpation with the thumbs (*arrows*).

flow if partially obstructed carotids are compressed simultaneously. Care should be taken to avoid palpation in the vicinity of the carotid sinus, especially in the elderly (see earlier).

Selective diminution of a carotid pulse is relatively common in elderly adults, but selective augmentation is unusual at any age. An example of the latter is the kinked right carotid[16] (Fig. 3–22) which can be mistaken for a pulsating aneurysm. When the carotid artery becomes too long for the distance it normally occupies, the vessel loops upon itself, ie, *kinks* (Fig. 3–22), a change that occurs almost exclusively in the *right* carotid, generally but not always in females beyond middle age.[16] Comparative palpation

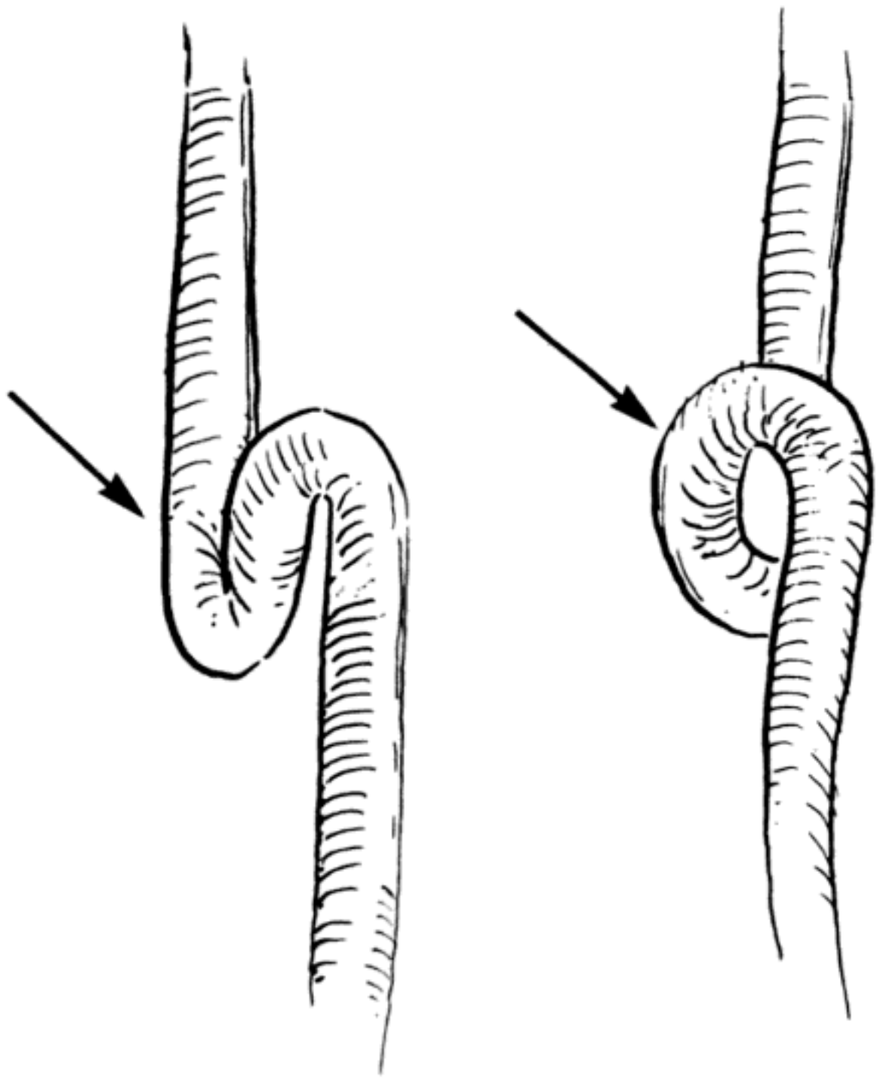


Figure 3–22 Schematic illustrations of kinked right carotid arteries.

of *radial* pulses in adults is best achieved by simultaneously applying the thumbs while the patient's hands lie comfortably at the side with the palms supinated. Diminution or selective absence of a radial pulse, particularly in elderly adults, requires assessment of radial and ulnar flow using *Allen's test*[17] (Fig. 3–23). Patency of the ulnar artery is determined by sequentially compressing the right and left radial arteries while the patient briskly opens and closes the fist a dozen or so times (Fig. 3–23*A*, *B*). Selective arterial refilling is judged by the rate of color return to the opened but *not hyperextended* palm (Fig. 3–23*C*), while pressure is maintained on the radial artery (Fig. 3–23*D*). Prompt return of normal color or transient reactive rubor of the palms and fingers indicates that the ulnar artery contributes normally to the circulation of the hand. Persistent pallor means ulnar occlusion. To assess patency of the *radials arteries*, the test is repeated while the right and left *ulnar* arteries are compressed.

The fingers as well as the palm should be observed during sequential release of radial and ulnar arteries (Figs. 3–23*C* and 3–23*D*) because delayed flushing of an individual digit sometimes indicates local arterial obstruction.

The dorsalis pedis and posterior tibial arteries are palpated routinely in adults. A fingertip is sometimes more convenient and sensitive than the thumb. This is especially so when applying the index or middle finger to the posterior tibials (Fig. 3–24). Pulsations in the dorsalis pedis and posterior tibial arteries vary from time to time in the same subject. Accordingly, diminution or absence during a single examination does not necessarily imply a persistent reduction. A tablet of sublingual nitroglycerin resolves doubt by rendering a temporarily absent dorsalis pedis or posterior tibial pulse readily palpable. The posterior tibial pulse is absent in 10 to 15 percent of normal subjects.

Upper vs Lower Extremity Pulses

Comparison of *upper* and *lower* extremity pulses (Fig. 3–10*B*) should be routine irrespective of age. Two methods are advocated. Some clinicians recommend comparison of femoral and *radial* arterial pulses, whereas others advocate comparison of femoral and *brachial* pulses. The radial and femoral pulse waves are normally sensed as synchronous, so that *any* femoral delay is considered abnormal. Positioning the patient's wrist next to the groin brings the radial and femoral arteries into proximity, a technique believed to facilitate comparison. My preference is for simultaneous palpation of *brachial* and *femoral arteries* which is accomplished by placing a thumb on each (Fig. 3–10*B*). In infants, especially newborns,

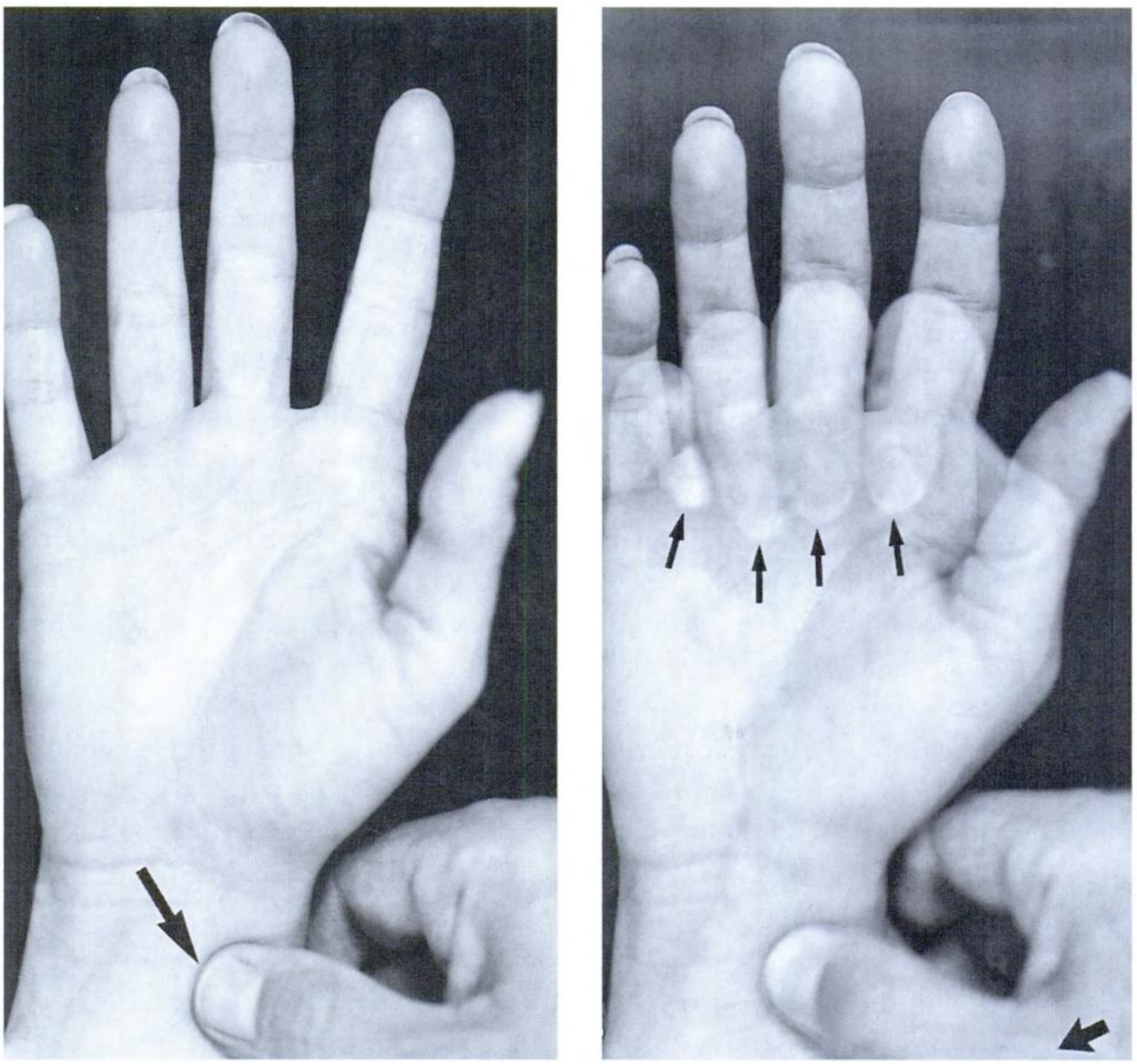
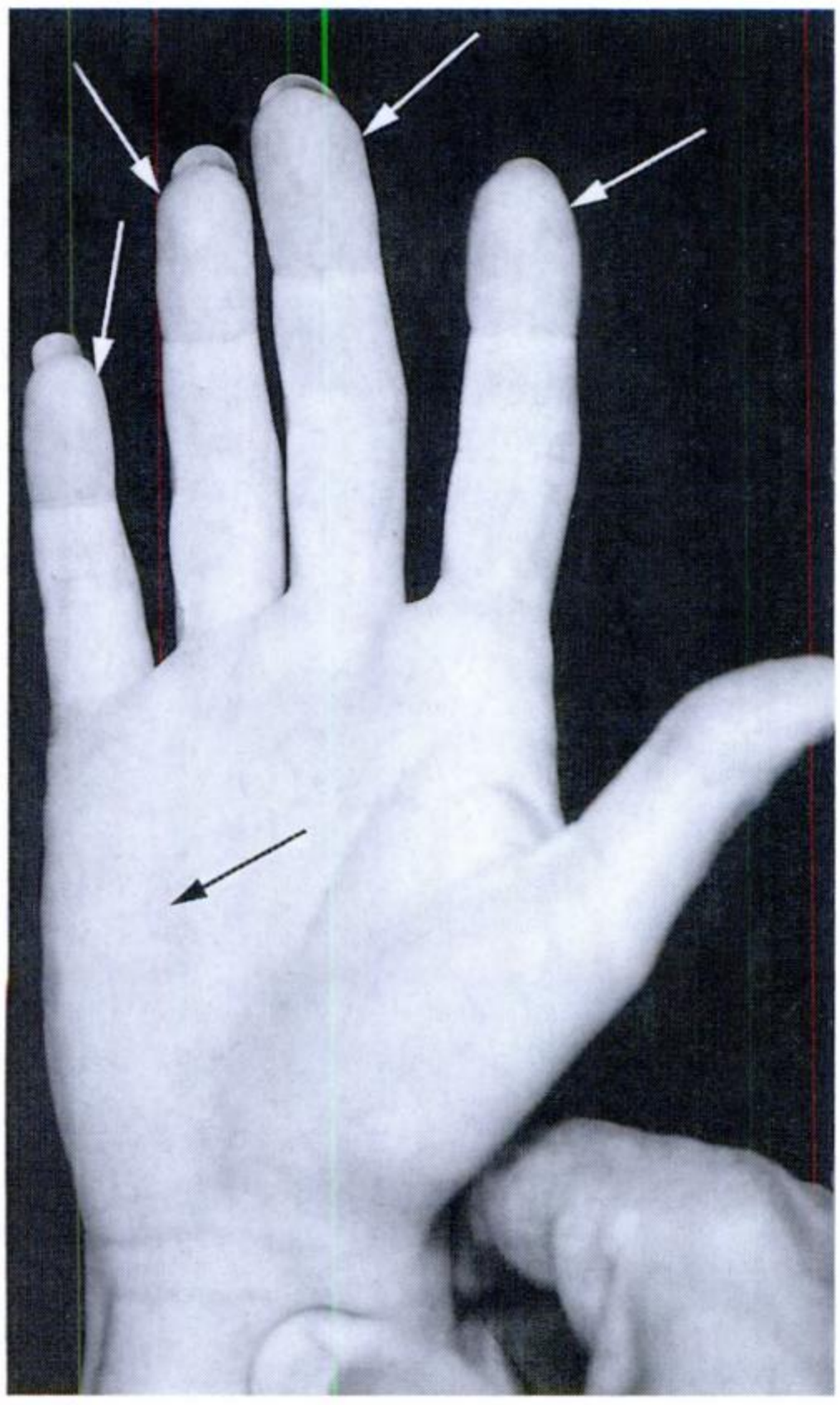


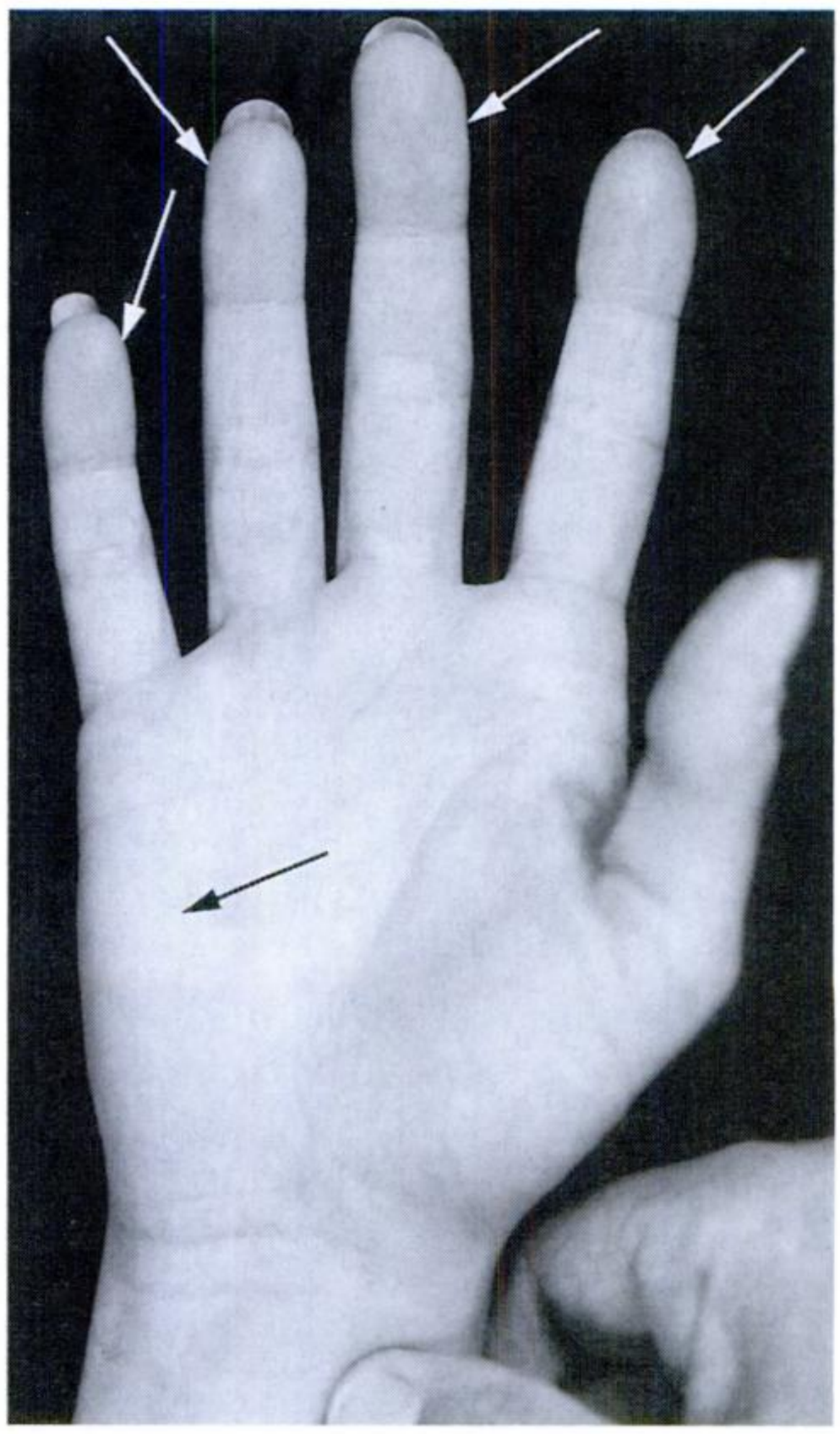
Figure 3–23 Allen's test for assessment of ulnar arterial patency. *A,* Selective occlusion of the radial pulse with the examiner's thumb (*arrow*). *B,* The patient rapidly opens and closes the fist to induce blanching as shown in *C (black palmar arrow)*. There is prompt return of color (*arrows)* to the *opened but not* hyperextended hand, while radial compression continues. *D*, indicating normal patency of the ulnar artery. *Continued*

palpation of the tiny radial pulse is impractical. When using the technique illustrated in Figure 3–10*B*, the examiner must wait for the baby to relax its legs and arms. Restraint is counterproductive and can render a normal femoral pulse impalpable. When comparing the *brachial* and femoral pulses, the trivial normal delay in perceived arrival time of the femoral pulse (on the order of 15 milliseconds in adults, less in infants and children) is a norm against which even slight deviations are readily judged. Furthermore, in all except a minority of patients with coarctation of the aorta, the femoral arterial pulses are *distinctly* reduced (Fig. 3–25) if not absent altogether, so diagnostic differences between upper and lower extremity pulses are readily apparent. In the occasional patient with mild coarctation, the femoral pulse is palpable, so that meticulous technique and timing are pivotal. In any event, what the examiner senses as *femoral delay* in coarctation is not a delay in *arrival* of the femoral arterial pulse, but instead a delay in its rate of rise (Fig. 3–25).

In adults, especially elderly adults, a relative reduction in femoral compared to brachial arterial pulsations generally means atherosclerotic iliofemoral obstruction. Asymmetry of right vs left femoral pulse is common.



C



D

Figure 3–23 *Continued*

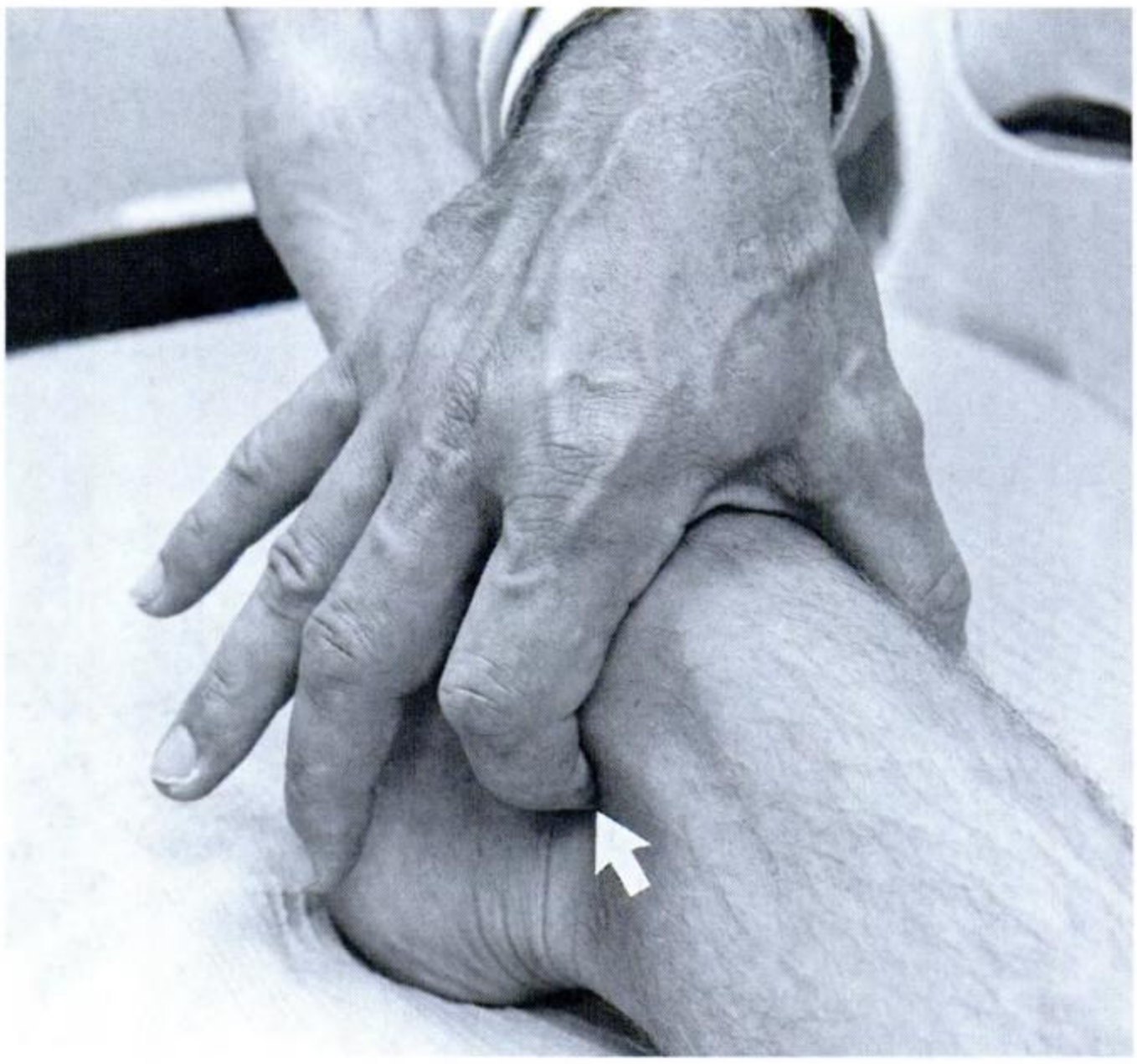


Figure 3–24 Palpation of the posterior tibial arterial pulse with the index finger (*arrow*).

Certain arterial pulses are not routinely palpated, but instead are examined only under circumstances that arouse suspicion. When lower extremity peripheral atherosclerotic arterial disease is suspected, *popliteal* pulses should be palpated in addition to femorals, dorsalis pedis, and posterior tibials. Two techniques of popliteal palpation are shown in Figure 3–5. The better technique (Fig. 3–5*A*) requires a prone position with flexed knee at less than a right angle, and muscles relaxed by resting the flexed leg against the shoulder of the examiner who sits or stands at the bedside or examining table. The midline of

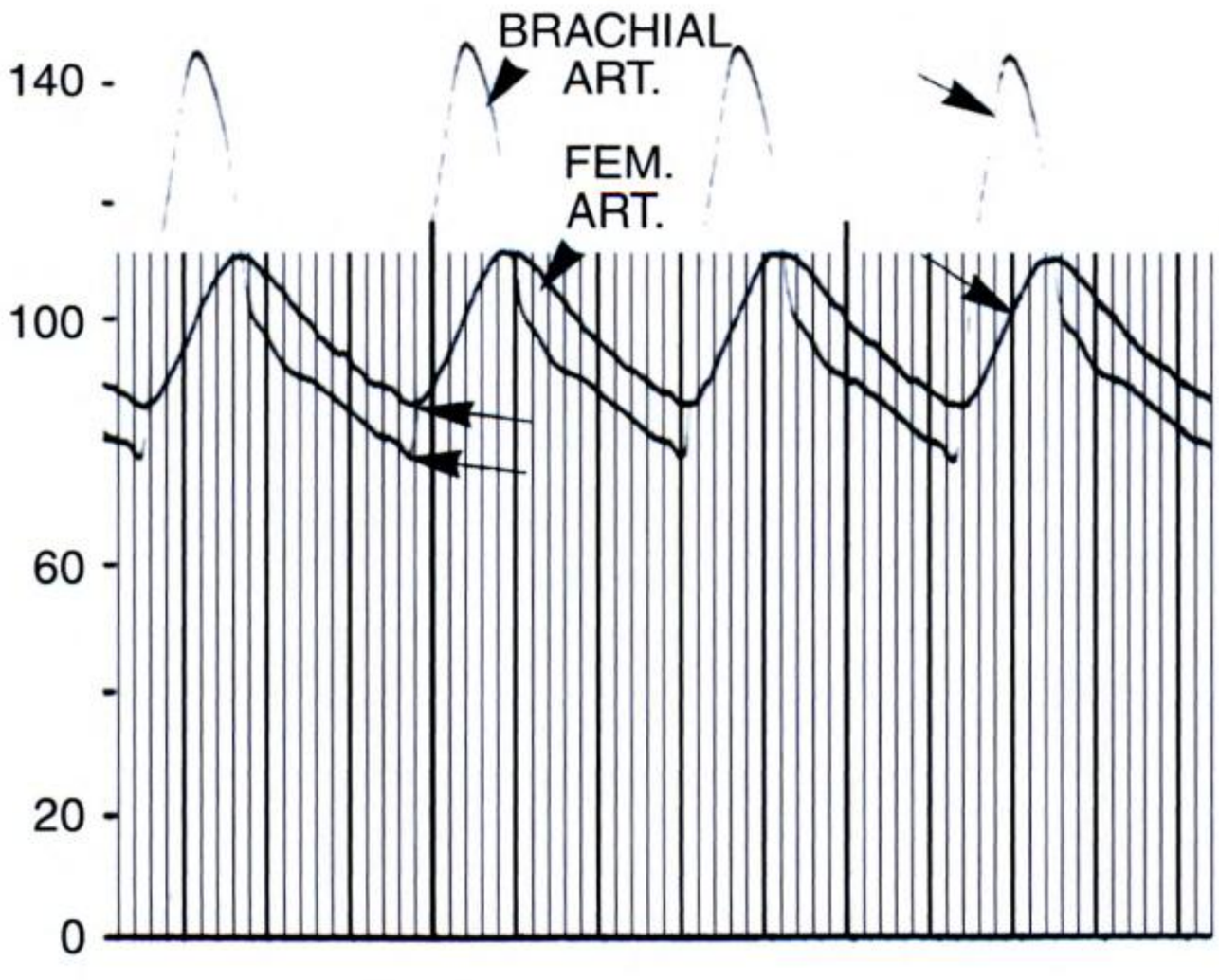


Figure 3–25 Simultaneous brachial and femoral arterial pulses in coarctation of the aorta. The *arrival times* are virtually identical (*lower left arrows*). The so-called femoral delay that is perceived by the examiner reflects dissimilarity in the rates of rise, not in the arrival times. The femoral rate of rise is slower than the brachial rate of rise (*upper right arrows*).

the popliteal fossa is firmly palpated with both thumbs (Fig. 3–5*A*). An alternative but less sensitive method of palpating the popliteal artery is with the patient supine and the knee flexed while the examiner firmly applies the fingers of both hands to the midline of the popliteal fossa as in Figure 3–5*B*.

In coarctation of the aorta, intercostal collateral arterial pulsations can sometimes be palpated in young adults. Normal intercostal arteries are not palpable, but in coarctation, the apex of the axillae provides access to dilated pulsatile intercostal collaterals.

In Chapter 2, brief comment was made on the skin of the feet in peripheral arterial occlusive disease. Diagnostic changes in color are elicited as follows. Examination begins with the patient supine. Each leg is passively elevated, a maneuver that intensifies pallor in the ischemic limb, whereas the skin color of a limb with normal arterial circulation is not affected. It is useful to elevate both legs simultaneously to induce or intensify the pallor. The patient should then change promptly from supine to sitting with both legs lowered passively over the side of the bed or examining table. The rate of color return can be delayed for as long as 60 seconds. Each foot should be compared with the other. When color returns, continued dependency results in intensification of rubor in the ischemic foot or in individual ischemic toes.

Digital (fingertip) pulsations are palpated under specific circumstances, such as severe aortic regurgitation. The patient's fingertips are gently but snugly gripped by the fingertips of the examiner (Fig. 3–26). To be certain that the fingertip pulsations are those of the patient and not the examiner, the digital pulses can be timed by simultaneously

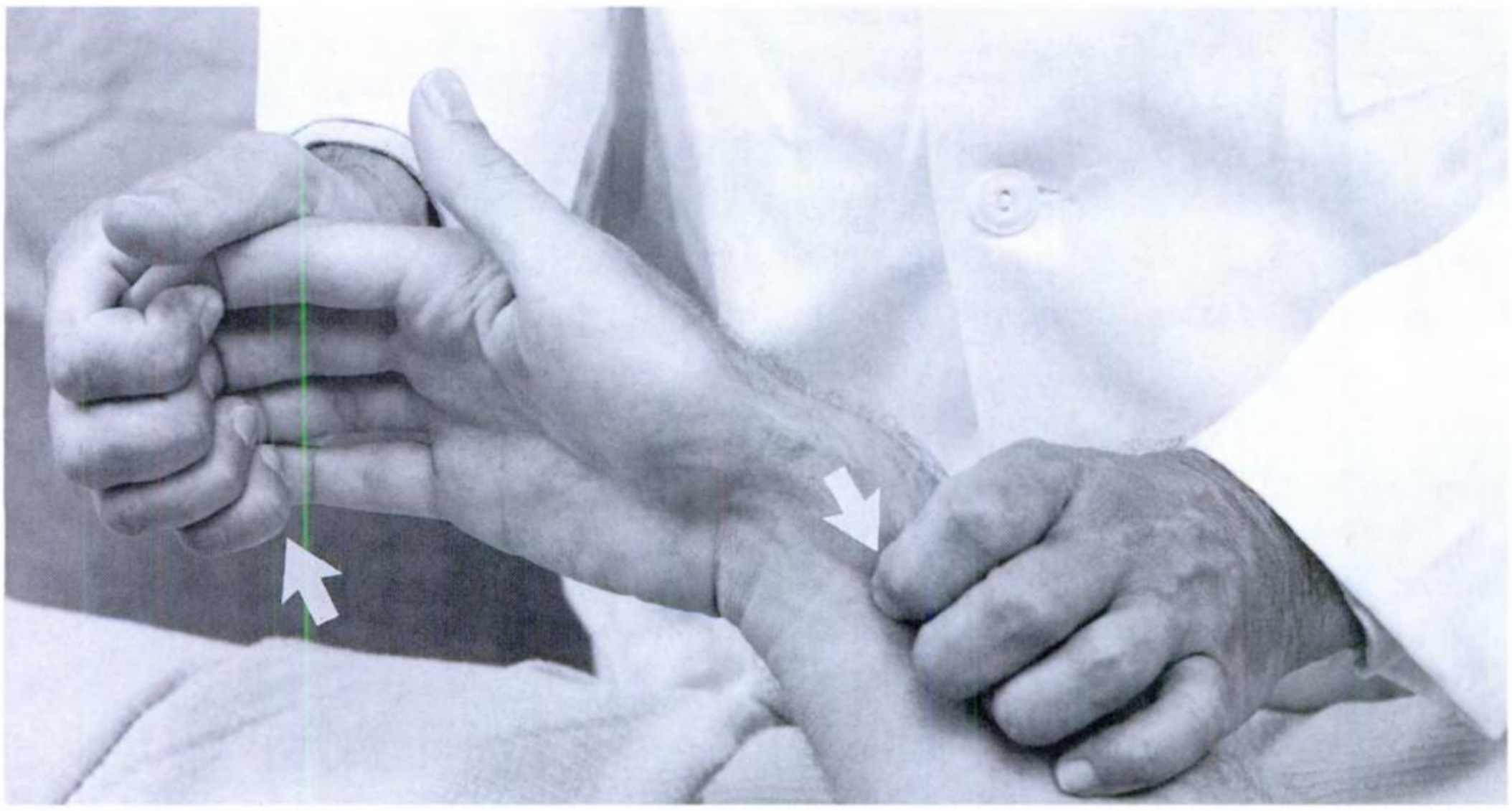


Figure 3–26 Upper left portion of the figure shows palpation of the patient's fingertips by the examiner's fingertips. Lower right portion of the figure shows simultaneous palpation of the patient's radial pulse (*arrow*) to provide assurance that the examiner is not sensing his or her own fingertip pulse.

palpating the ipsilateral radial artery with the examiner's free hand (Fig. 3–26). The visual counterpart of increased digital pulsations in severe aortic regurgitation is the Quincke pulse[18] best elicited by trans-illuminating the fingertip with a pocket flashlight or otoscope applied in a darkened room to the pad of the fingertip opposite the nail. The examiner's free hand encloses the finger to provide a relatively focused field that highlights the phasic reddening and blanching of the nailbed with each systole and diastole. Quincke wrote[18]:

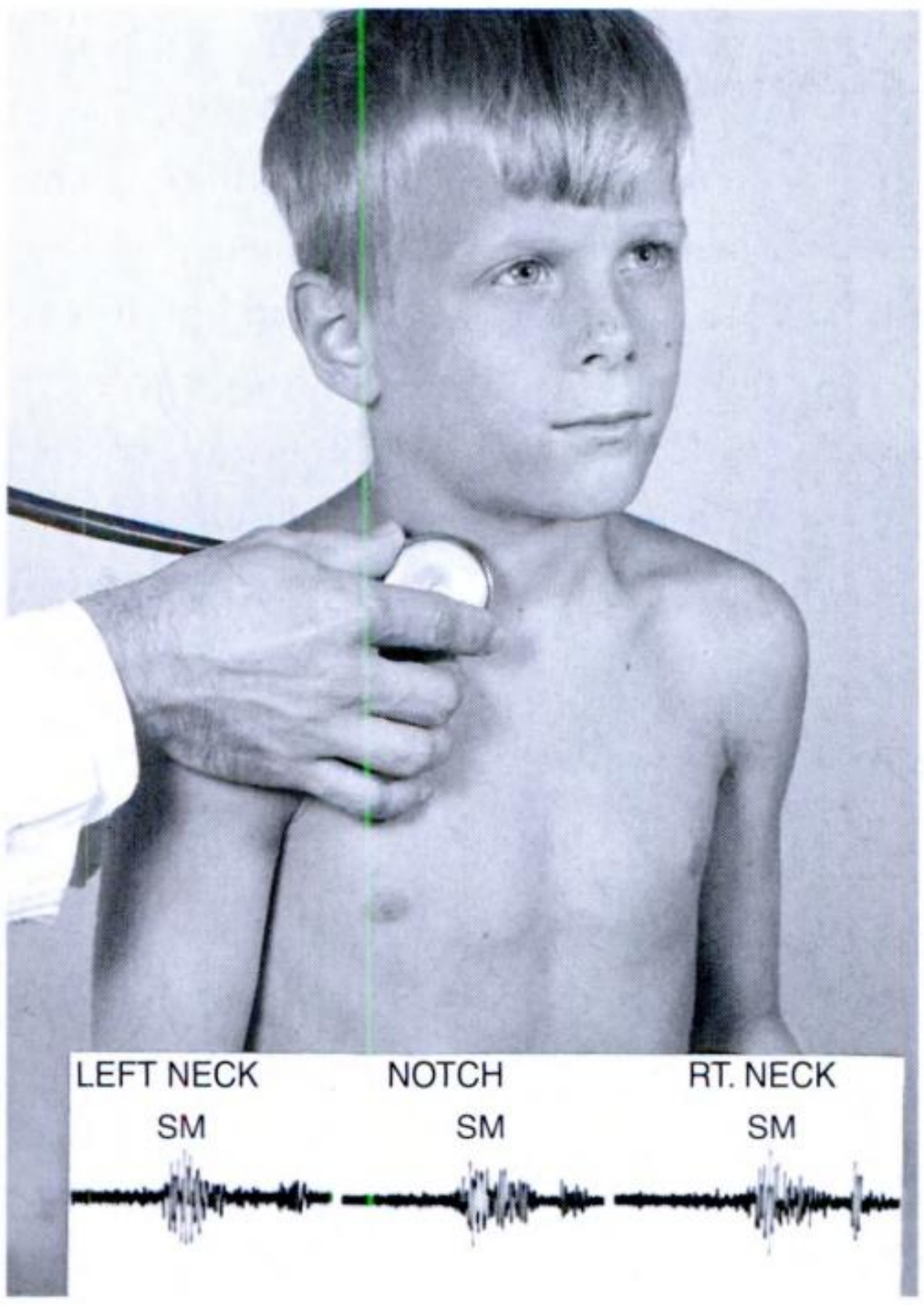
> One observes a distinct lightning-like and momentary accentuation of the reddening, so that the manner of the appearance and disappearance of the capillary pulse is objectively as characteristic a sign of aortic insufficiency as the exquisitely abrupt pulse to the palpating finger.

Thrills and murmurs should be routinely sought over certain arteries, with the sites varying according to patient age. In normal children and young adults, an innocent supraclavicular systolic murmur can be sufficiently loud to transmit below the clavicles as a thrill. Auscultation is performed while the patient sits and looks forward with shoulders relaxed and forearms and hands resting on the lap (Fig. 3–27*A*). The bell of the stethoscope is placed in the supraclavicular fossa over the subclavian artery. The shoulders are then hyperextended with the elbows brought back sharply until the shoulder girdle muscles are taut (Fig. 3–27*B*). In response to this maneuver, the innocent supraclavicular systolic murmur diminishes considerably or disappears altogether.

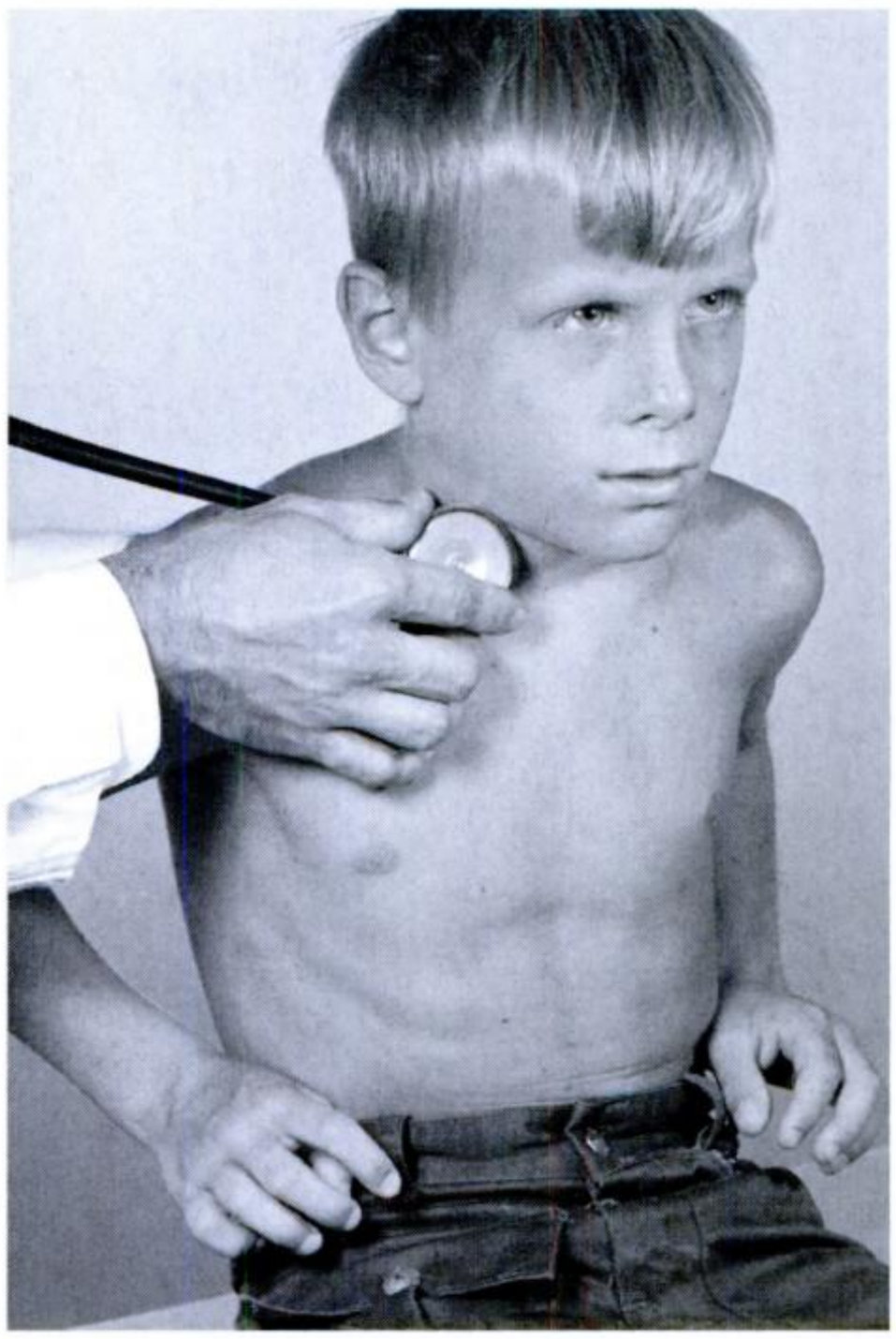
Auscultation over carotid, subclavian, and femoral arteries is obligatory in elderly adults even if palpation is unrevealing. Carotid and subclavian auscultation is undertaken while the patient is either supine or sitting, with chin pointed straight ahead. The bell of the stethoscope is applied just firmly enough to achieve a skin seal over each internal carotid artery in its lower, middle, and upper thirds. A carotid arterial murmur is not necessarily louder on the side on which the pulse is diminished pulse. In fact, the converse may be the case. The right and left *subclavian* arteries are then examined by applying the stethoscope in the supraclavicular fossae. Femoral arterial auscultation requires that the patient lie supine while the bell of the stethoscope is applied sequentially in the right and left inguinal regions. Each femoral artery is compared to the other, as the carotids and subclavians were compared. As with the carotid artery, a femoral arterial murmur is not necessarily louder on the side of a diminished pulse.

Arterial auscultation at unorthodox sites is based on patient age and clinical suspicion. When coarctation of the aorta is suspected, the diaphragm of the stethoscope should be applied to the posterior thorax over the vertebral column at the estimated level of the coarctation. In infants, this observation is best made with the patient prone and the stethoscopic diaphragm applied over the vertebral column between the scapulae. In older children and adults, auscultation for this purpose is accomplished with the patient sitting and the shoulders and thorax relaxed. The murmur overlying the coarctation can be systolic or continuous, depending on the degree of isthmic obstruction.

In elderly adults, auscultation of the *abdominal aorta* at its bifurcation is useful especially when palpation of the femoral arteries discloses iliofemoral atherosclerotic



A



B

Figure 3–27 *A,* Supraclavicular auscultation begins while the patient is sitting with shoulders relaxed and forearms resting on the lap. Inset shows the phonocardiogram of a normal supraclavicular systolic murmur in the left neck, right neck and suprasternal notch. *B,* When the patient hyperextends his shoulders by bringing the elbows well behind the back, the murmur diminishes or disappears altogether.

obstruction. In hypertensive patients, auscultation in the flanks over the right and left kidneys may identify the soft, high-frequency systolic murmur that occasionally accompanies renal artery stenosis (see Chapter 8).

Structural Properties

> We recognize the yielding nature of the arterial coats in healthy arteries. In degeneration of the coats, the arterial walls may be universally thickened or contain bead-like patches of induration as in atheroma, or the artery may have become a rigid tube, as in calcareous degeneration.[1]

Structural abnormalities of systemic arteries can be visually evident. The elongated brachial artery of Monckeberg's sclerosis can be seen beneath the skin of the antecubital fossa (see Fig. 3–3) as each cardiac cycle imparts distinctive movement to the snake-like tortuous

vessel. When the artery is "rolled" under the thumb, it imparts the distinctive feel of a thickened, firm arterial wall as Mackenzie described above. The radial and the dorsalis pedis pulses are occasionally visible as well as palpable in free aortic regurgitation or with a large left-to-right shunt through a patent ductus arteriosus. The distinctive tactile and visual properties of a kinked carotid artery (see Fig. 3–22) can be mistaken for an aneurysm.

Conversely, true aneurysms are sometimes overlooked unless meticulously sought, such as an abdominal aortic aneurysm in elderly adults, especially hypertensive (see Chapter 8). Pulsations of a normal abdominal aorta do not extend below the umbilicus, even in thin patients with relatively flaccid abdominal walls. When abdominal pulsations extend below the umbilicus, an aortic aneurysm is the presumptive reason. Systolic murmurs in the vicinity of abdominal aortic aneurysms originate from iliofemoral obstruction rather than from within the aneurysm.

Femoral arterial aneurysms are, as a rule, readily identified because of unilateral exaggeration of a femoral pulse, especially if that pulse is visible and the contralateral femoral pulse is not. Small asymptomatic popliteal aneurysms, typically unilateral, are sometimes discovered incidentally when the popliteal pulse is palpated. The pulsation of an aneurysm in the popliteal fossa is more apparent when the patient is examined prone with the knee flexed (see Fig. 3–5*A*). An obvious popliteal aneurysm can be overlooked when examination is performed with the patient supine. Diagnosis depends on assessment of the width of the pulsation when the thumbs are firmly applied to the midline of the popliteal fossa (Fig. 3–5*A*).

REFERENCES

1. Mackenzie J. *The Study of the Pulse, Arterial, Venous, and Hepatic, and of the Movements of the Heart.* Edinburgh: Young J. Pentland, 1902.
2. O'Rourke MF, Kelly R, Avolio A. *The Arterial Pulse.* Philadelphia, PA: Lea & Febiger, 1992.
3. Broadbent WH. *The Pulse.* London: Cassell, 1890.
4. Lewis WH. The evolution of clinical sphygmomanometry. *Bull N Y Acad Med.* 1941;17:871.
5. Geddes LA, Hoff HE, Badger AS. Introduction of the auscultatory method of measuring blood pressure including a translation of Korotkoff's original paper. *Cardiovasc Res Center Bull (Baylor).* 1966;5:57.
6. Manning DM, Kuchira C, Kaminski J. Miscuffing: Inappropriate blood pressure cuff application. *Circulation.* 1983;68:763.
7. Osler W. *Principles and Practice of Medicine.* New York: Appleton-Century, 1892.
8. Wood FC, Wolferth CC. Tolerance of certain cardiac patients for various recumbent positions (trepopnea). *Am J Med Sci.* 1937;193:354.
9. Floyer J. *The Physician's Pulse Watch.* London: Smith and Walford, 1707.
10. Upshaw CB, Silverman ME. The Wenckebach phenomenon; a salute and comment on the centennial of its original description. *Ann Intern Med.* 1999;130:58.
11. Nishimura RA, Tajik AJ. The Valsalva maneuver revisited. *Mayo Clin Proc.* 1986;61:211.
12. Wiggers CJ. *The Pressure Pulses in the Cardiovascular System.* London: Longmans, Green, 1928.
13. Corrigan DJ. On permanent patency of the mouth of the aorta, or inadequacy of the aortic valve. *Edinb Med Surg J.* 1832;37:225.

14. Traube L. A case of pulsus bigeminus. *Berl Klin Wochenschr.* 1872;9:185. Translated in Willius FA, Keys TE, eds. *Classics of Cardiology*. Malabar, FL: Robert E. Krieger, 1983;p 590.
15. Kussmaul A. Euber schweilige mediastinopericarditis and den paradoxen pulse. *Klin Wochenschr.* 1873;10:433.
16. Metz H, Murray-Leslie RM, Bannister RG. Kinking of the internal carotid artery in relation to cerebrovascular disease. *Lancet.* 1961;1:424.
17. Allen EV. Thromboangiitis obliterans: Methods of diagnosis of chronic occlusive arterial lesions distal to the wrist with illustrative cases. *Am J Med Sci.* 1929;178:237.
18. Quincke H. Observations on capillary and venous pulse. *Berl Klin Wochenschr.* 1868;5:357. Translated in Willius FA, Keys TE, eds. *Classics of Cardiology*. Malabar, FL: Robert E. Krieger, 1983;p 569.

4 The Jugular and Peripheral Veins

The internal and external jugular veins are sources of anatomic, hemodynamic, and electrophysiologic information "within the chambers of the heart"—the right atrium and right ventricle.[1] Veins of the extremities and thoracic inlet reflect primary, if not intrinsic, venous disease.

THE JUGULAR VEINS

In 1902, James Mackenzie established the jugular venous pulse as an essential part of the cardiovascular physical examination:

> We come now to the study of a subject which gives us far more information of what is actually going on within the chambers of the heart. In the study of the venous pulse we have often the direct means of observing the effects of the systole and diastole of the right auricle, and of the systole and diastole of the right ventricle.[1]

In the 1950s, Paul Wood rekindled interest in the jugular pulse, emphasizing that

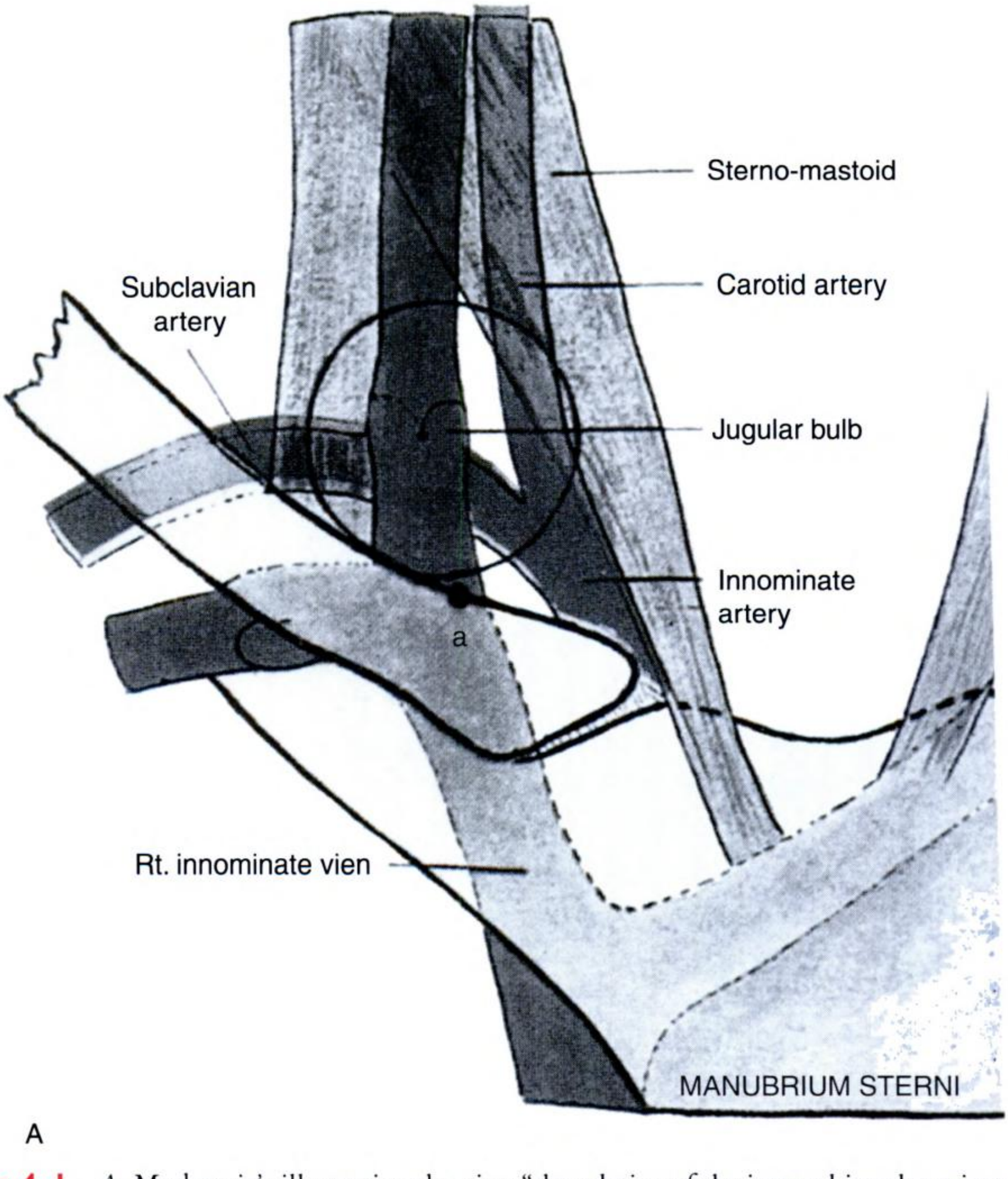
> Precise analysis of the cervical venous pulse and measurement of the height of each individual wave with reference to the sternal angle is not only possible at the bedside but highly desirable.[2]

Information derived from examination of the jugular veins includes (1) waveform and pressure, (2) anatomic/physiologic inferences, and (3) arrhythmias and conduction defects. The technique described below permits analysis of the jugular veins in most if not all patients except in infants whose short necks and lack of cooperation make examination impractical, although not impossible.

External and Internal Jugular Veins

The external jugular vein provides estimates of mean right atrial pressure, while the internal jugular reflects right atrial waveform *and* pressure. The *internal* jugular is

equipped with a bicuspid venous valve at the thoracic inlet, and lies within the carotid sheath behind the sternocleidomastoid, an important anatomic relationship emphasized by Mackenzie[1] (Fig. 4–1*A*). The jugular bulb, a slight dilatation of the internal jugular vein immediately proximal to the venous valve, lies in a hollow between the two clavicular insertions of the sternocleidomastoid muscle.



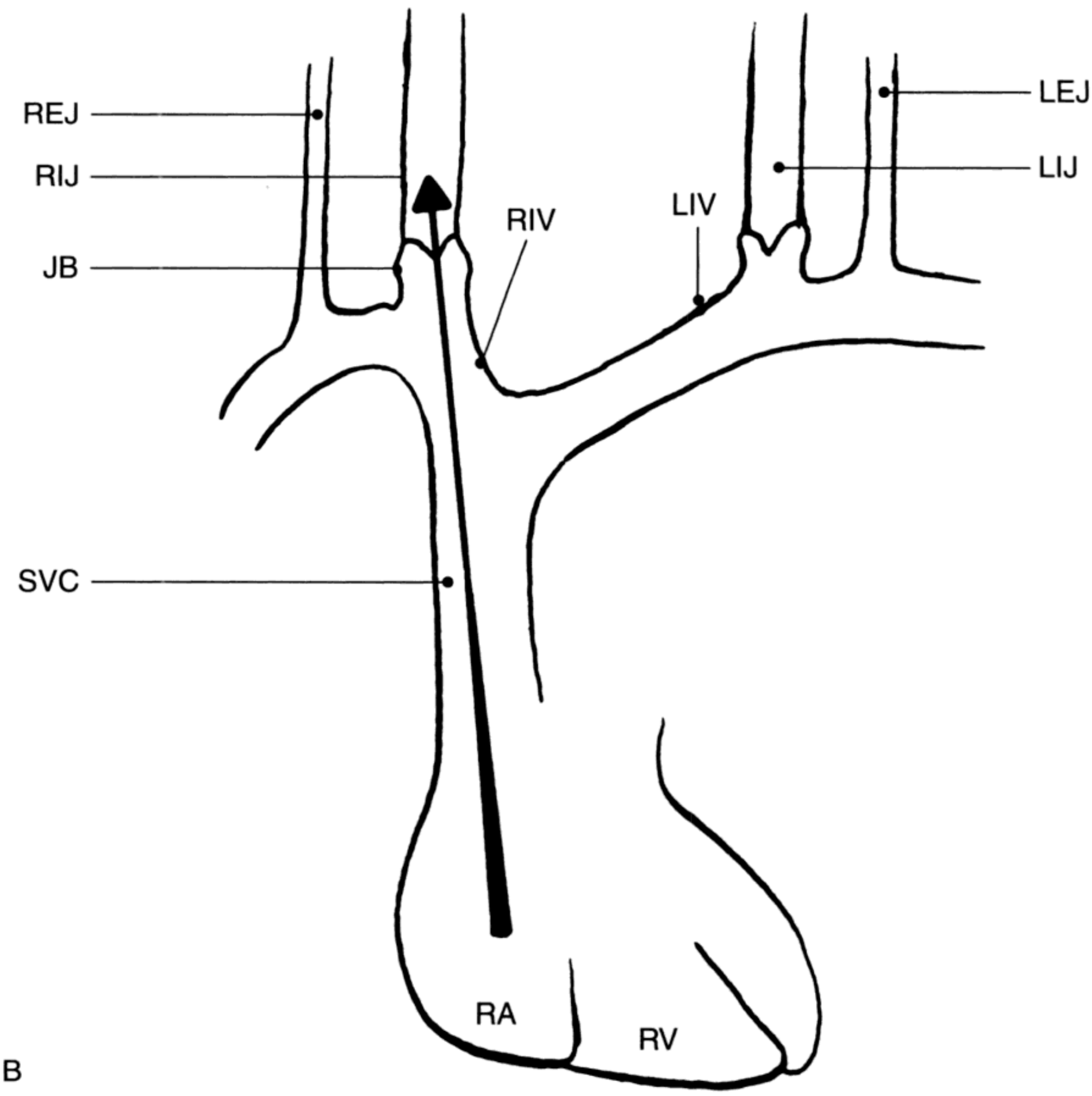
A

Figure 4–1 *A*, Mackenzie's illustration showing "the relation of the internal jugular vein to the carotid and subclavian arteries, and to the sterno-mastoid muscle." He identified the valve in the jugular bulb. (From Mackenzie J: Diseases of the Heart. London, Oxford University Press, 1908.) *B*, Sketch illustrating Mackenzie's contention that "the direct communication of the right internal jugular through the right innominate vein and superior vena cava in about a straight line renders it susceptible to the movements from the right heart." REJ, RIJ = right external jugular and right internal jugular; LEJ, LIJ = left external jugular and left internal jugular; RIV, LIV = right innominate and left innominate veins; JB = jugular bulb; SVC = superior vena cava; RA = right atrium; RV = right ventricle. *Continued*

Phasic pressure imparted to the internal jugular vein accurately reflects the right atrial waveform. Accordingly, more information is derived from examination of the internal jugular pulse than from the external jugular vein, as Mackenzie originally stated (Fig. 4–1*B*):

> The movements communicated by the heart to the blood in the veins are usually best observed in the internal jugular veins. The direct communication of the right internal jugular through the right innominate and superior cava in about a straight line renders it susceptible to the movements of the right heart.[1]

The right internal jugular is therefore more important than the left in the physical examination, because it provides a more direct and therefore a more accurate reflection of right atrial mechanical activity, and of right atrial and right ventricular pressure–volume relationships. When pulsations of the internal jugular vein are more prominent on the *left* rather than the right side of the neck, the cause is almost always a persistent left superior



B

Figure 4–1 *Continued*

vena cava. Pulsations transmitted from the right atrium into the internal jugular vein are generated when the steady flow of systemic venous return becomes phasic upon reaching the right atrial chamber. The only significant difference between the waveforms of the internal jugular vein and the right atrium is the impact of the carotid pulse—the C wave—upon the former (Fig. 4–2*A, B*) (see below).

Normal Waveform of the Internal Jugular Vein

Potain described the waveform of the internal jugular vein in 1867,[3] and Mackenzie provided the nomenclature that remains in use with few or no modifications (Fig. 4–2). Mackenzie designated the two crests as A and V, and went on to write:

> There are two rises in the auricular pressure curve—a large and a small one, with of course two falls. The first rise in pressure immediately precedes the rise in ventricular pressure. It can only be due to the systole of the auricle. Immediately after the auricle ceases to contract, there is a great fall (x) in the pressure due to the diastole of the auricle. The auriculo-ventricular valves being closed, the blood pouring into the auricles from the veins, the pressure gradually rises, producing the second small wave in the curve. This wave is terminated by the opening of the auriculo-ventricular valves at the beginning of the ventricular diastole. When the pressure becomes lower in the ventricles than in the auricles, the valve opens and the contained blood passes through, reducing the auricular pressure, and causing the second fall (y). After this, the pressure slowly rises by the accumulation of blood in both chambers, until it is suddenly increased by the next auricular systole.[1]

These remarks need little or no change. The A wave (Fig. 4–2*A*) reflects right atrial contraction and occurs just before the carotid arterial pulse and the first heart sound. The X descent (decline of the A wave) is initiated by right atrial relaxation, and is reinforced as right ventricular contraction causes descent of the floor of the right atrium. The initial portion of the descent (atrial relaxation) is sometimes designated X while the remainder of the descent during right ventricular contraction is designated X′. For practical purposes, the designation X is generally used for the entire descent, while not ignoring the fact that the descent is due sequentially to right atrial diastole followed by descent of the floor of the right atrium during ventricular systole.

The X descent, which is often the most conspicuous feature of the normal jugular venous pulse, is interrupted by the C wave (Figs. 4–2*A* and 4–2*B*), which is the impact carotid itself. Mackenzie assigned the letter C to this movement, which he correctly ascribed to the carotid pulse. A relatively small C wave is sometimes generated within the right atrium proper as right ventricular isovolumetric contraction displaces the tricuspid leaflets upward, but this small intra-atrial C wave is overshadowed in the jugular pulse by the large carotid pulsation with which it coincides.

The V wave (Fig. 4–2) was so designated by Mackenzie because its ascent begins during ventricular systole, hence V. The ascending limb reflects passive filling of the right atrium as venous return confronts a closed tricuspid valve. The Y descent represents

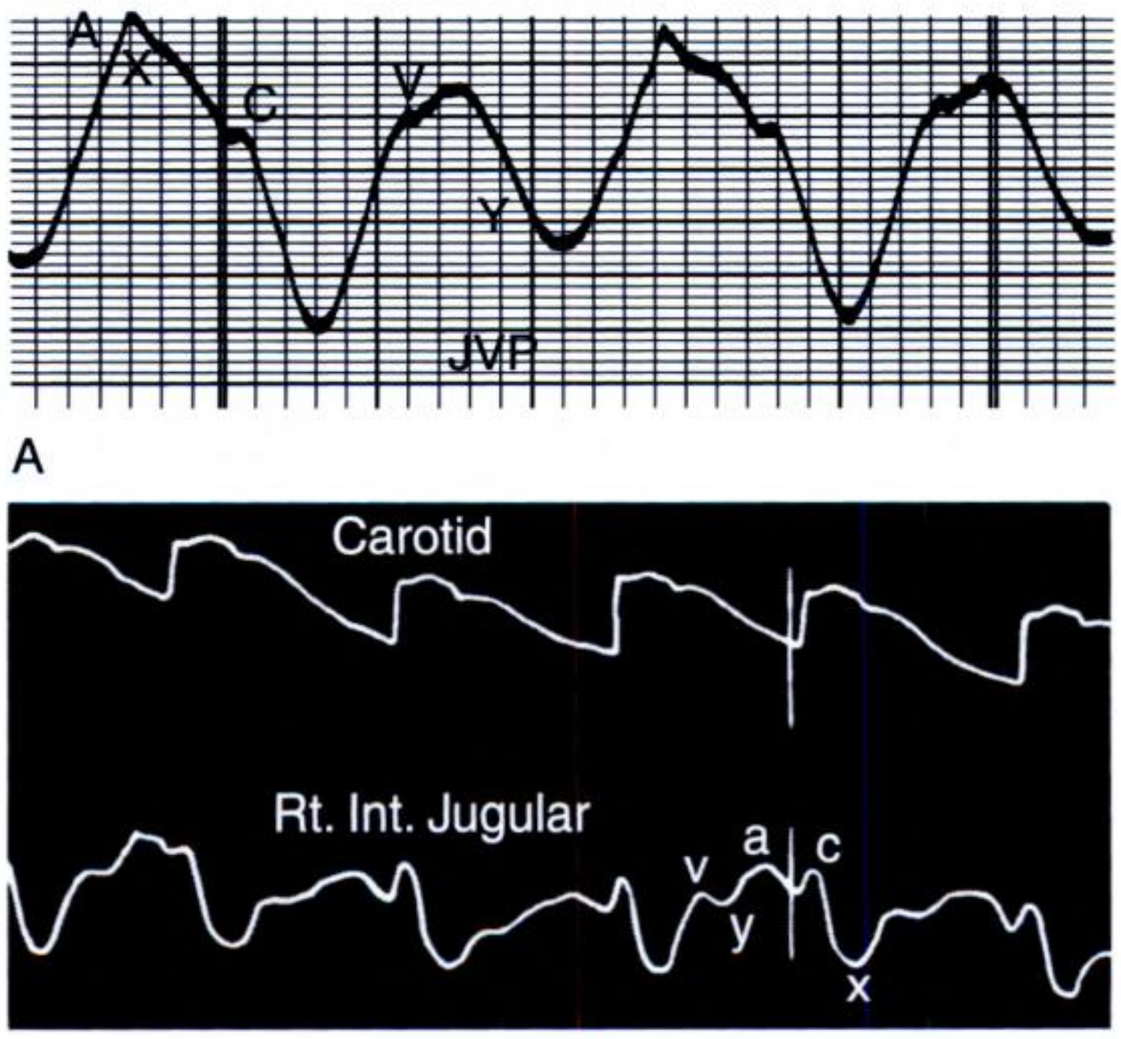


Figure 4–2 *A*, Normal jugular venous pulse (JVP) showing the A wave, the X descent, the C wave, and the V wave followed by the Y descent. *B*, James Mackenzie's "simultaneous tracings of the carotid pulse and the jugular pulse, showing the exact synchronism of the carotid wave (c) in the jugular pulse." The designations of the crests and troughs of the jugular pulse as originally used by Mackenzie are the same as those in tracing *A*. (From Mackenzie J: The Study of the Pulse, Arterial, Venous, and Hepatic, and of the Movements of the Heart. Edinburgh, Young J. Pentland, 1902.) *C*, Normal right atrial (RA) pressure pulse. The slight rise preceding the "a" wave is called the "h" *wave (arrows).* The heart rate is slow.

sudden termination of the V wave as the right ventricle relaxes, the tricuspid valve opens and the right atrial pressure rapidly falls. The Y descent is normally less conspicuous than the X descent, and the Y trough is not as deep as the X trough (Fig. 4–2).

Following the Y trough, right atrial and right ventricular pressures slowly rise in parallel (diastases), especially when diastole is prolonged by a slow heart rate. A slight rise preceding the next A wave is called the *h* wave (Fig. 4–2*C*), so designated by Hirschfelder in 1907.[4] The *h* wave is seldom, if ever, identified in the normal jugular venous pulse, even when the cardiac rate is slow.

Technique of Examination

Patient Position and Light Source

Proper examination of the jugular venous pulse demands that the patient lie supine on a bed or examining table that is readily adjustable above the horizontal (Fig. 4–3). An electrically controlled adjustment is ideal.

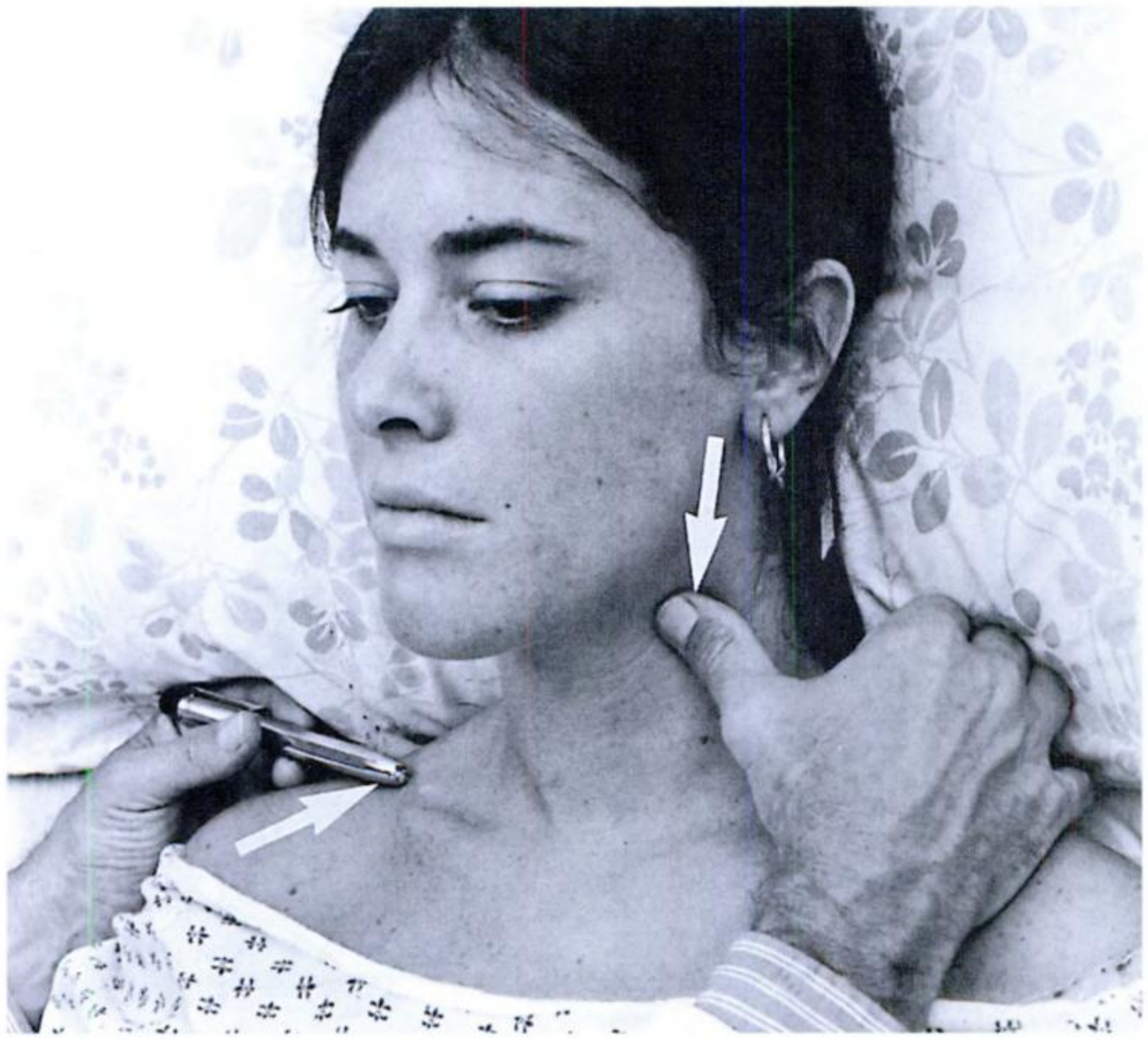
The trunk should be positioned at an angle that corresponds to the maximum visible oscillations of the right internal jugular vein. Begin with a 30 degree elevation, then lower or elevate the trunk to achieve the maximum oscillations. If the jugular venous pressure is low, the trunk is lowered until pulsations become visible. If oscillations of the internal jugular are not visible at 15 degrees above the horizontal, gentle abdominal compression with the flat of the hand increases venous return and transiently reveals the oscillations (see below under Assessment of Jugular Venous Pressure). The higher the central venous pressure, the greater the required elevation of the trunk above horizontal. If the crests of the jugular venous oscillations cannot be seen with the patient sitting bolt upright, the legs should be dangled over the side of the bed or examining table, a maneuver that generally brings into view the crests of high jugular venous pulsations. If the oscillations still cannot be seen, the jugular pulse should be examined with the patient standing.

The head should be adjusted from a neutral position slightly upward or downward and to the *right* (Fig. 4–3*A*) by gently moving the chin, but the head should *not* be tilted too far upward or turned too far to the left (Fig. 4–3*B*), because these maneuvers tense the right sternocleidomastoid (Fig. 4–1*B*) and compress or obliterate the internal jugular pulse.

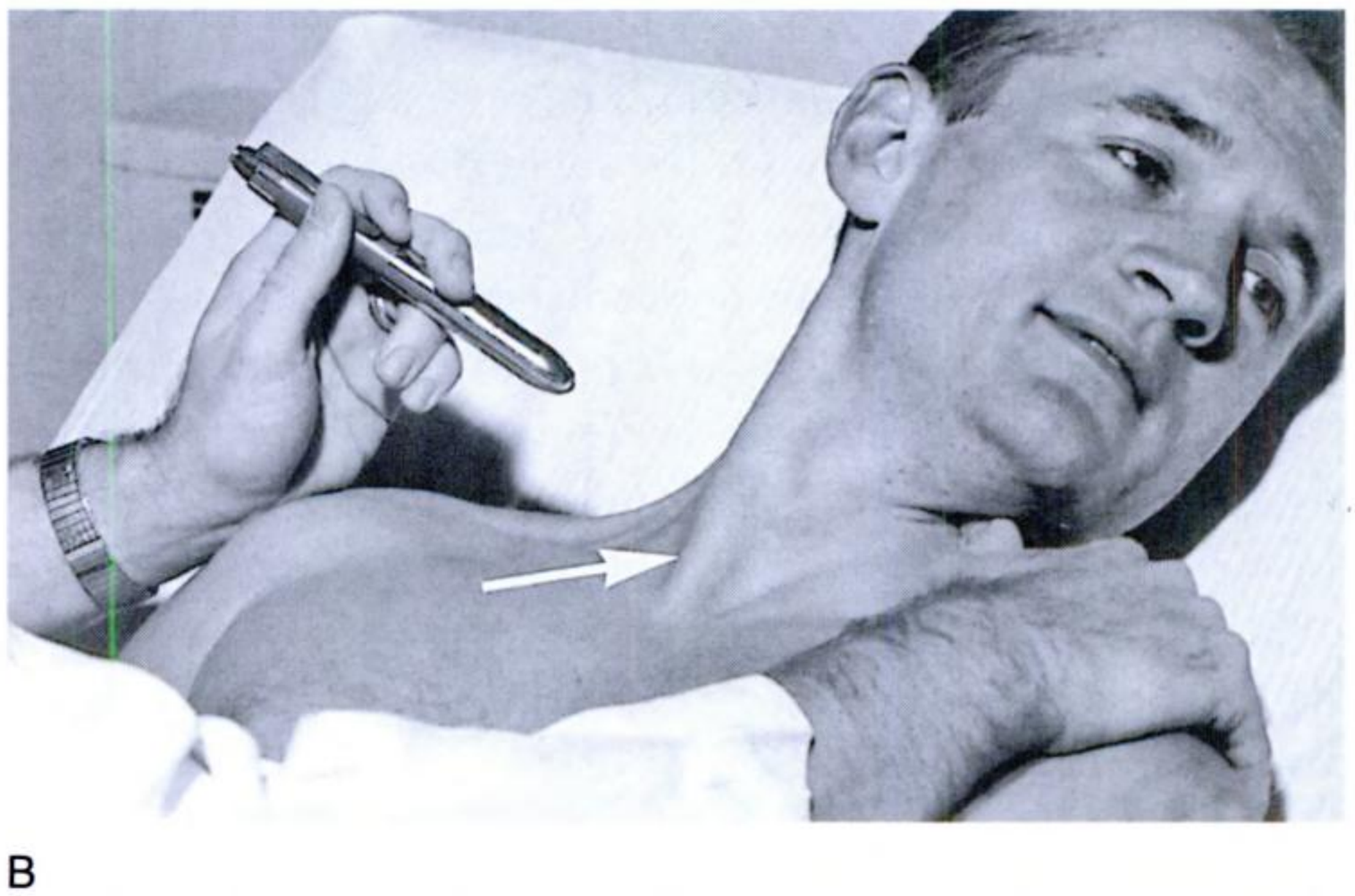
The light source such as a small pocket flashlight with a focused beam is mandatory. Illumination should be tangential, ie, across the area under scrutiny (Fig. 4–3*A*) to highlight the shadow cast by the external jugular and the fluctuations of the internal jugular vein. It is best for the examiner to use the left hand to adjust the light source and the right hand to palpate the left carotid artery for timing purposes (Fig. 4–3*A*). The right hand can also be used to apply the stethoscope to the chest when the jugular pulse is timed with the heart sounds (see below).

External Jugular Vein

The right external jugular vein may not be visible unless mechanically distended by digital compression at the root of the neck (Fig. 4–4*A*). A visible right external jugular vein

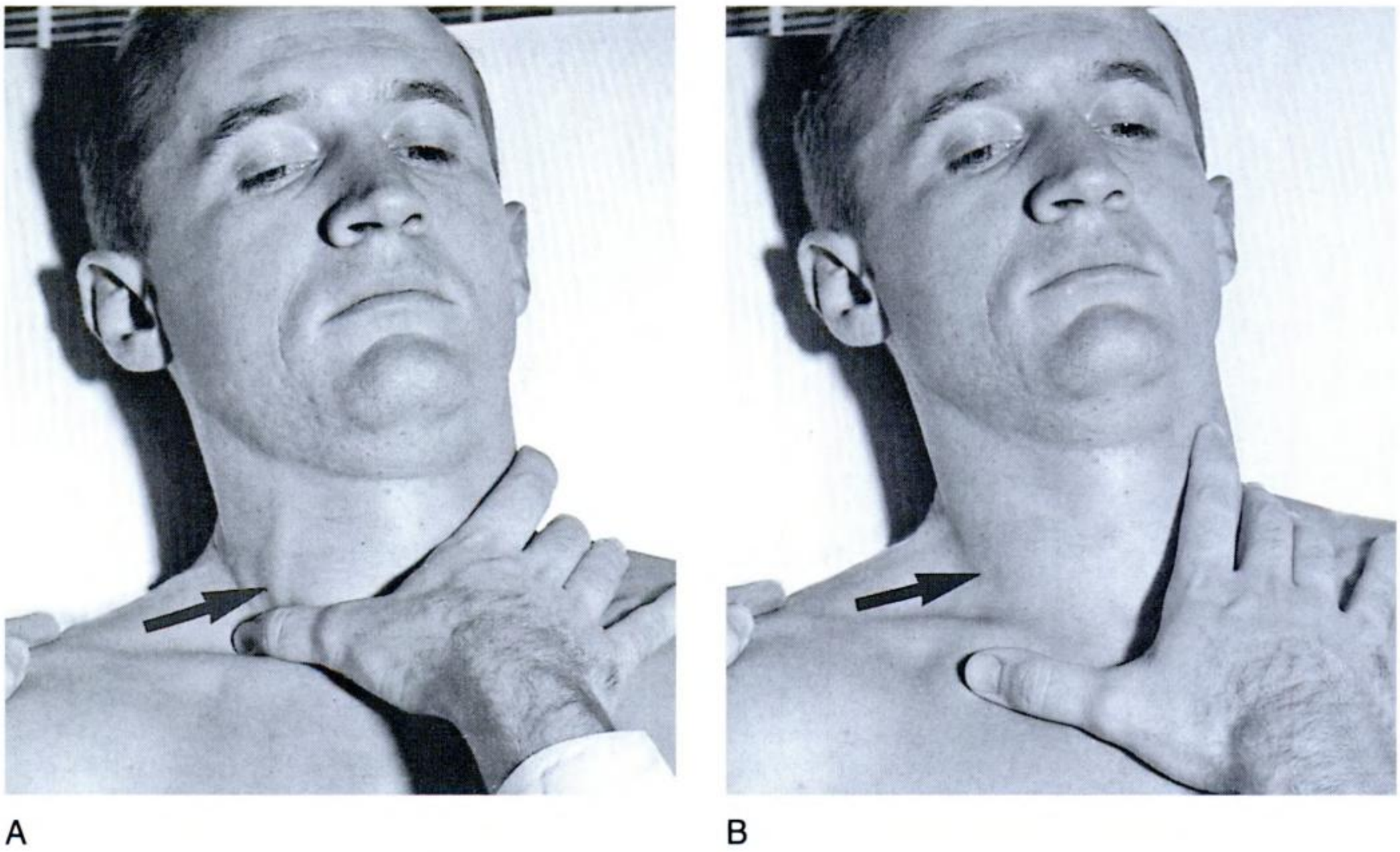


A



B

Figure 4–3 *A*, The trunk is elevated 30 degrees above the horizontal. The head is adjusted to look forward or slightly to the right to relax the ipsilateral sternocleidomastoid muscle. The examiner's right thumb palpates the left carotid for timing purposes (*upper arrow*) while a tangential light (*left horizontal arrow*) shines across the right lower neck to highlight the crests and troughs of the jugular pulse. *B*, Rotating the head to the left tenses the right sternocleidomastoid (*arrow*) and compresses or obliterates the jugular pulse. This position is to be avoided.



A B

Figure 4–4 *A*, The external jugular vein distends when compressed just above the clavicle (*arrow*). *B*, The distended vein disappears (*arrow*) when compression is released.

is a static nonpulsatile column that becomes apparent when it casts a shadow. The external jugular provides an estimate of the mean vena caval and right atrial pressure, which reflect the right ventricular end-diastolic pressure in the absence of tricuspid stenosis.

Before proceeding, there are three caveats. First, in patients with congestive heart failure, elevation of the trunk above the horizontal serves to decrease venous return and lower the central venous pressure.[5] Second, veno-constriction occasionally obliterates the *external* jugular vein despite an increase in mean right atrial pressure. Third, distention of the external jugular vein does not necessarily indicate that the mean right atrial pressure is elevated. If the external jugular is not visible because of veno-constriction, examination of the pulsatile internal jugular vein prevents error (see below). If distention of the external jugular is *not* caused by an elevated mean right atrial pressure, the following simple maneuver clarifies this point. The distended vein is first emptied by applying digital pressure to its superior aspect while blood is mechanically expressed by running a thumb or finger downward along the course of the vessel. The collapsed vein then lies between the two sites of digital compression. If release of the *inferior* site results in prompt filling from *below*, the cause is a high mean right atrial pressure. As the vessel fills from below, the column of blood occasionally stops at a distinct bulge that represents a competent internal jugular venous valve (Fig. 4–1). If the external jugular does not fill from below, the vein is once again mechanically emptied, and the *superior* site of digital compression is released. Prompt distention of the vein indicates that filling is from above, which is of no clinical significance.

Except for these qualifications, the crest of the external jugular vein is a convenient manometer for estimating mean right atrial or central venous pressure. When the trunk is elevated 30 degrees above horizontal, the crest of the normal right external jugular vein is seen just above the clavicle. Digital compression at that site impedes flow from above and elevates the column (Fig. 4–4*A*). When compression is released, the column falls out of sight (Fig. 4–4*B*) or is seen just above the clavicle. In any event, the crest of a normal external jugular vein does not exceed 3 cm above the sternal angle. When the crest of the external jugular vein cannot be seen at 30 degrees above the horizontal, the trunk should be elevated until the crest comes into sight. The height is then measured in centimeters above the sternal angle, as shown in Figure 4–8 (Assessment of Jugular Venous Pressure).

Internal Jugular Vein

In 1867, Potain recommended timing the internal jugular pulse by precardial palpation or auscultation of the heart sounds:

> I studied them then in this relationship by combining palpation or auscultation of the precordial region with inspection of the cervical pulsations.[3]

Mackenzie subsequently employed the carotid artery for timing (see Fig. 4–2*B*). The carotid pulse and the heart sounds, properly applied, achieve the same end; but the carotid is preferable because the physical examination does not have to be interrupted for auscultatory orientation, which is especially undesirable in patients with complex heart sounds and murmurs.

The right thumb palpates the left carotid pulse, while the left hand shines a tangential light across the undulations of the right internal jugular vein (see Fig. 4–3*A*). Cervical venous pulsations that are not synchronous with the carotid cannot be arterial, and therefore must be venous. The A wave and the beginning of the X descent precede the carotid (C), which is followed by continuation of the X descent to its trough (see Fig. 4–2). The V wave commences immediately after the carotid pulse and is followed by the Y descent, after which a low amplitude h wave is generated but seldom seen even if diastole is prolonged (Fig. 4–2*C*).

Timing of the jugular venous pulse with auscultation requires selection of a precordial site at which the first and second heart sounds are clearly identified. The stethoscope is positioned with the right hand while the light source is adjusted with the left hand. The first heart sound occurs immediately before the carotid pulse. The A wave and the beginning of the X descent precede the first heart sound by a space that coincides with the PR interval (see later). The remainder of the X descent continues beyond the first sound. The second heart sound immediately precedes the crest of the V wave and is followed by the Y descent.

What the Eye Perceives

The normal jugular venous pulse is a sequence of gentle, undulating crests and troughs.[6] The descents, especially the X descent, are usually more obvious than the crests (see

above). A relatively brisk X descent, for example, makes an otherwise subtle A wave more apparent. The converse is the case with the carotid pulse, which is more obvious in its ascent than in its decline. Mackenzie recognized these points:

> The sudden collapse of the tissues covering the vein is more striking than the protrusion, whereas the carotid pulse is always abrupt and sudden in its protrusion of the covering tissues, and gradual in its shrinking.[1]

Effects of Respiration

Mackenzie also observed that

> When the steady stream of venous blood approaches the heart, it is subjected to the intermittent influences of the respiratory and cardiac actions.[1]

The jugular venous pulse is usually more obvious during inspiration. A low normal jugular pulse is sometimes evident *only* during inspiration. This is so chiefly because the descents, especially the X descent, are brisker during inspiration and are therefore more apparent.

Despite the inspiratory fall in intrathoracic pressure and the accompanying increase in venous return, there is a decline in *mean* pressure in the right atrium but an augmentation of right atrial contractile force. This preserves or increases the crest of the A wave, thus accentuating the initial portion of the X descent as the right atrium relaxes more rapidly after its vigorous contraction. In addition, the inspiratory augmentation in right ventricular volume and contractile force results in a brisker descent of the floor of the right atrium, and accordingly in a brisker descent of the portion of the X descent that follows the C wave. The Y descent is increased during inspiration because tricuspid flow during the rapid filling phase is reinforced by the inspiratory fall in right ventricular diastolic pressure. Kussmaul's sign (see below) refers to an *abnormal* response to inspiration, namely, a *rise* in right atrial mean and jugular venous pressures.

Jugular Venous Pulse vs Carotid Pulse

> The pulse in the internal jugular vein is often mistaken by most experienced observers for beating of the carotids.[1]

However, the carotid pulse has a single rise and fall, whereas the jugular pulse has two crests and two troughs. The *rise* of the carotid is brisker than its descent, whereas jugular venous *descents* are brisker than their crests. Inhalation diminishes the carotid pulse, but makes the jugular pulse more evident. Abdominal compression (see below) has no effect on the carotid, but transiently improves visibility of the jugular pulse. The carotid artery is normally palpable, whereas the jugular pulse is not. Sitting improves visibility of the carotid pulse, while the jugular venous pulse drops from sight. Gentle digital compression above the clavicle obliterates the jugular venous pulse but not the carotid, as Potain recognized:

> A light pressure, suitably applied to the lower portion of the neck can impede them or suppress them entirely, while the pulsations of the carotid persist with all of their intensity.[3]

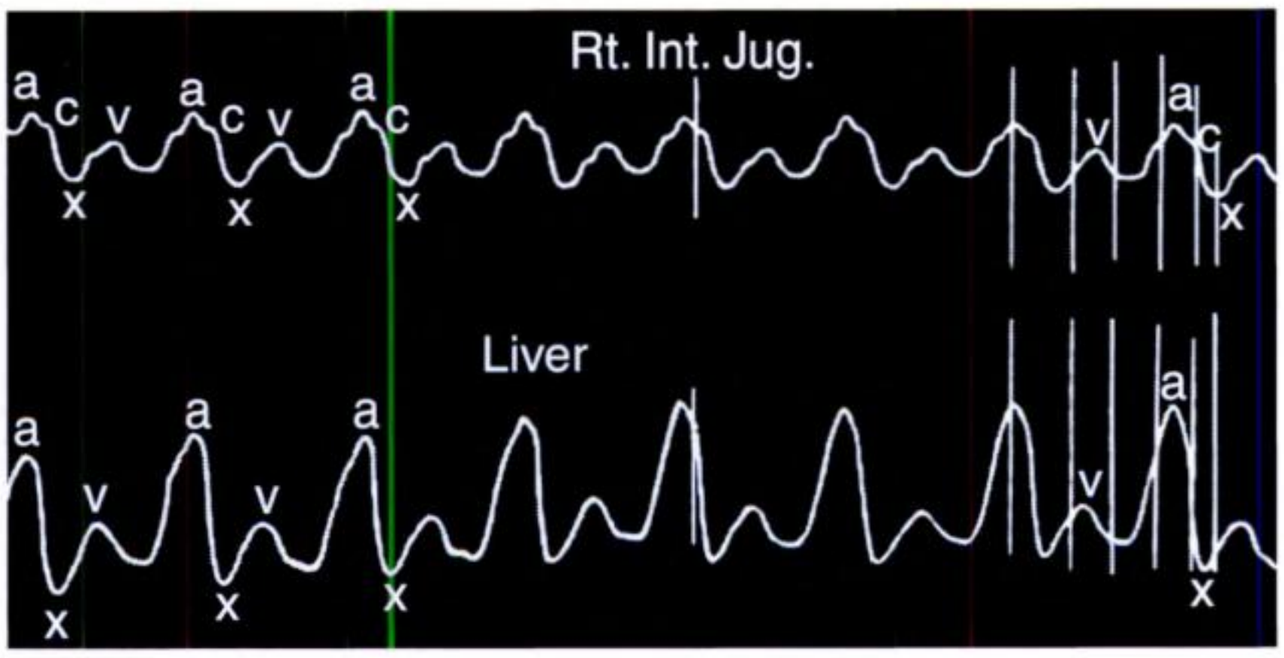


Figure 4–5 "Simultaneous tracings of the jugular and liver pulses."[1] The major waveforms are similar in both tracings except for absence of the carotid "c" wave in the hepatic pulse. (From Mackenzie J: The Study of the Pulse, Arterial, Venous, and Hepatic, and of the Movements of the Heart. Edinburgh, Young J. Pentland, 1902.)

Hepatic Pulse

The right atrial pulse is transmitted via the *superior* vena cava into the internal jugular vein (see Fig. 4–1), and the hepatic pulse (Figs. 4–5 and 4–6) is transmitted via the *inferior* vena cava into the liver.

"The relationship between the liver pulse and the jugular pulse is a very intimate one. In certain cases, waves of blood are sent back into the inferior vena cava, and these waves distend the liver, and give rise to a pulsatile swelling of the organ. When there is both a venous and a liver pulse present they are always of the same character, and one can demonstrate the changes that take place by the liver pulse as well as by the venous pulse"[1] (Figs. 4–5 and 4–6).

The hepatic pulse is detected by palpation. The palm of the right hand is placed upon the patient's right upper quadrant below the anticipated liver edge. Care should be taken to avoid bending the wrist, which decreases sensitivity in the fingertips during contact with the liver edge. A gentle liver pulse is sometimes detectable in normal thin individuals with flaccid abdominal walls. When the liver is palpable during normal respiration, the patient is instructed to hold the breath in a respiratory midposition. If the liver is not palpable, a moderately deep breath is held just long enough to permit accurate palpation of the descended liver edge.

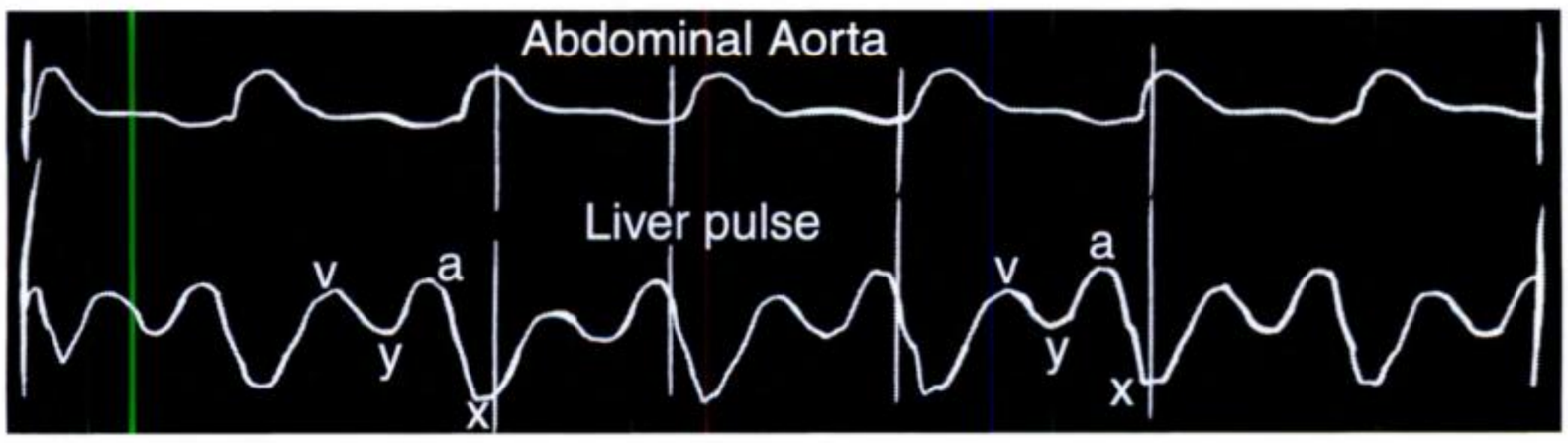


Figure 4–6 "Simultaneous tracings of the abdominal aorta and of the liver pulse."[1] The waveform of the liver pulse is not altered by the aortic pulsation. (From Mackenzie J: The Study of the Pulse, Arterial, Venous, and Hepatic, and of the Movements of the Heart. Edinburgh, Young J. Pentland, 1902.)

Assessment of Jugular Venous Pressure

Three venous beds are accessible for estimations of pressure—the external jugular vein, the internal jugular vein, and veins on the dorsa of the hand (see under The Upper Extremities). In assessing the external or internal jugular venous pressure, it is best to avoid the term "jugular venous distention" which is imprecise. The first necessity is to focus on a reference point and to adjust the patient's trunk above the horizontal (see below). A useful reference against which venous pressure can be measured is the sternal angle of Louis (Pierre Charles Alexander Louis, 1787–1872), which has the advantage of easy identification (Fig. 4–7). Recent investigations have confirmed Sir Thomas Lewis' observation regarding the accuracy and utility of the sternal angle as a reference point.[5] The simplest way of judging the height of the external jugular vein and the height of the A and V waves of the internal jugular is to measure their crests in centimeters above the sternal angle, as shown in Figure 4–8. The height of the jugular venous pressure in *centimeters of water* is not recommended for contemporary use because it relates to a time when manometric estimates were employed.

The examiner should focus first on the nonpulsatile external jugular vein (see above and Fig. 4–4) and then on the height of the A and V crests of the internal jugular (see above and Fig. 4–2). In analyzing jugular venous pressure, the trunk should be adjusted above the horizontal to an angle that coincides with the maximum excursions of the right internal jugular vein (see earlier). The patient's head is positioned to avoid compression of the right internal jugular vein by the sternocleidomastoid muscle (see Figs. 4–1*A* and 4–3). Rotating the head to the left tenses the sternocleidomastoid (Fig. 4–3*B*).

The right internal jugular vein permits assessment of the heights of the A and V crests (Figs. 4–2 and 4–3), and the nonpulsatile *external* jugular permits an approximation of mean right atrial pressure (see Fig. 4–4). With the trunk elevated 30 degrees above the horizontal, normal A and V waves undulate just above the clavicle, with the A wave slightly dominant (see Fig. 4–2). When related to the sternal angle as a reference, the crests of the normal A and V waves do not exceed 3 cm above the angle of Louis when the trunk is 30 degrees above the horizontal.

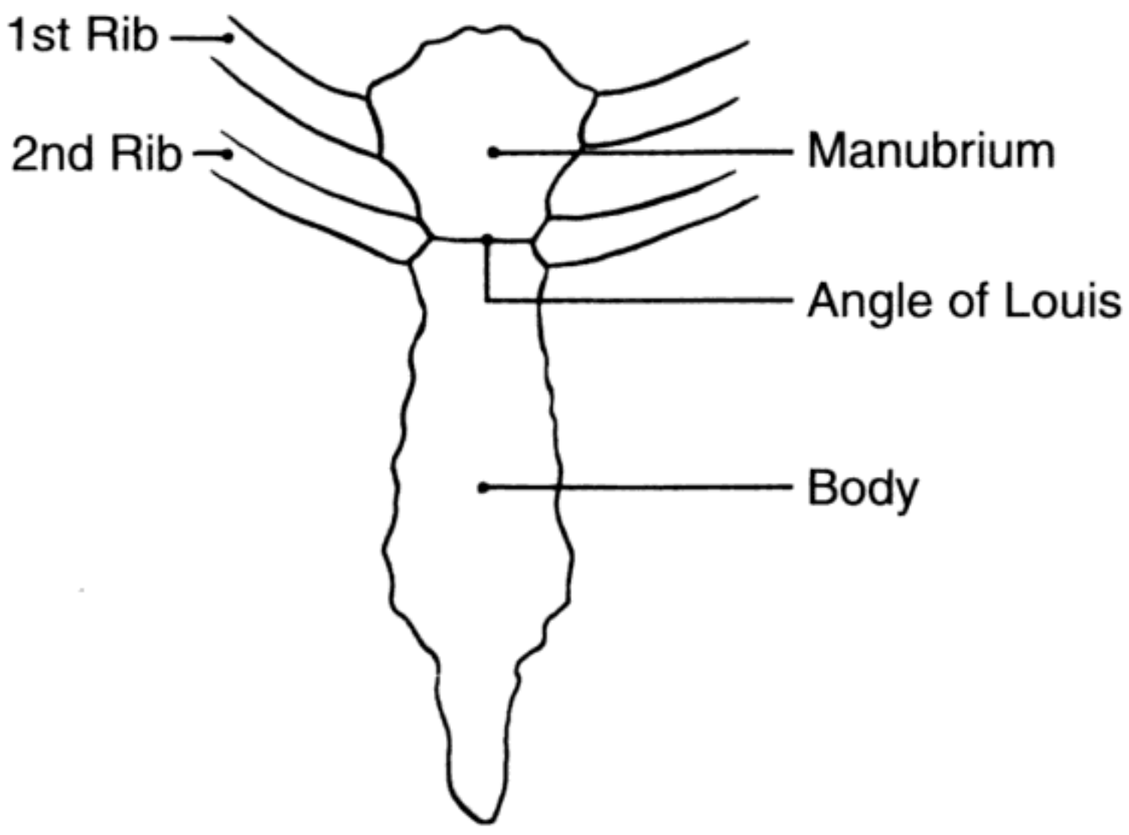


Figure 4–7 The sternal angle of Louis is a well-defined transverse ridge or junction between the manubrium above and the body of the sternum below.

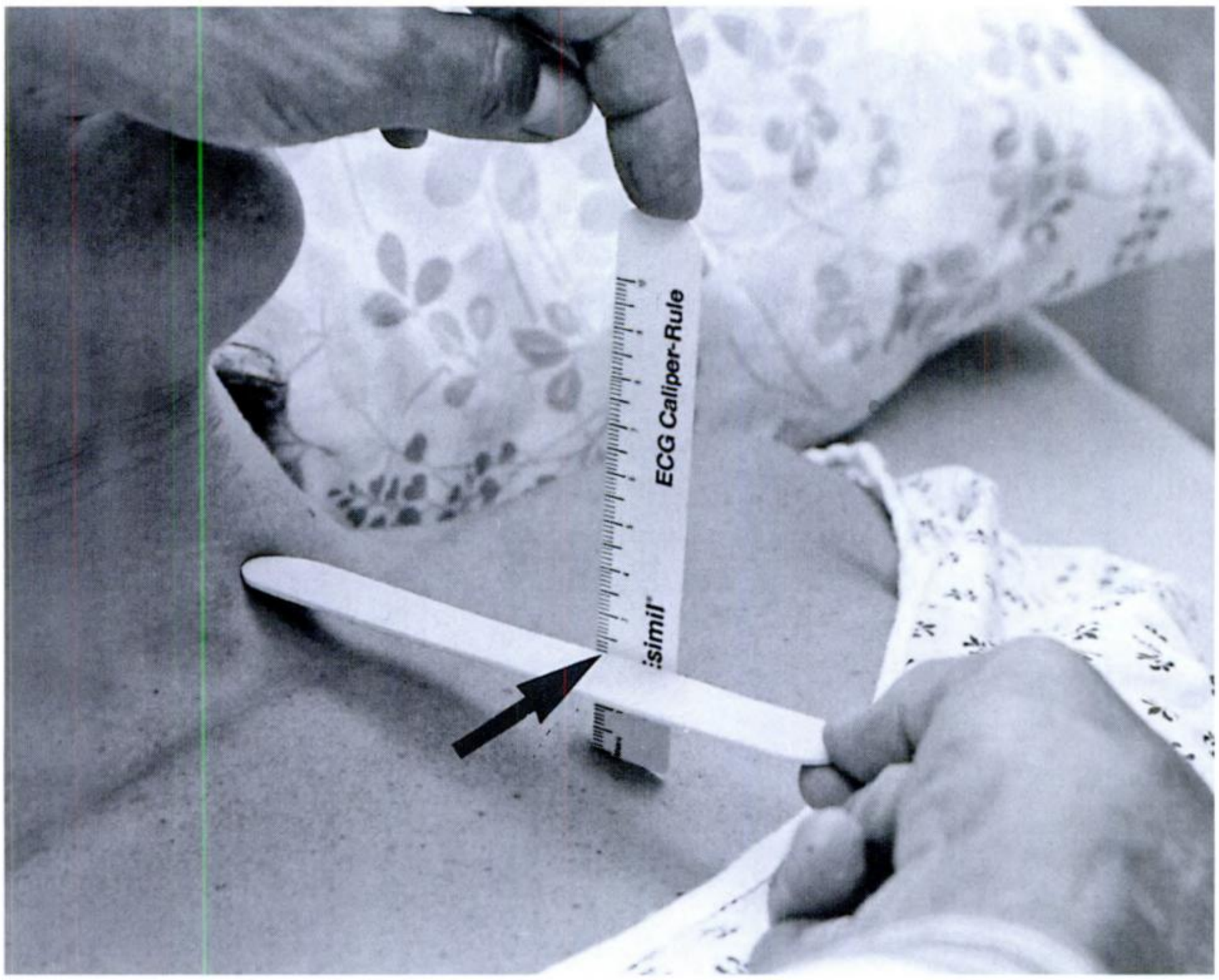


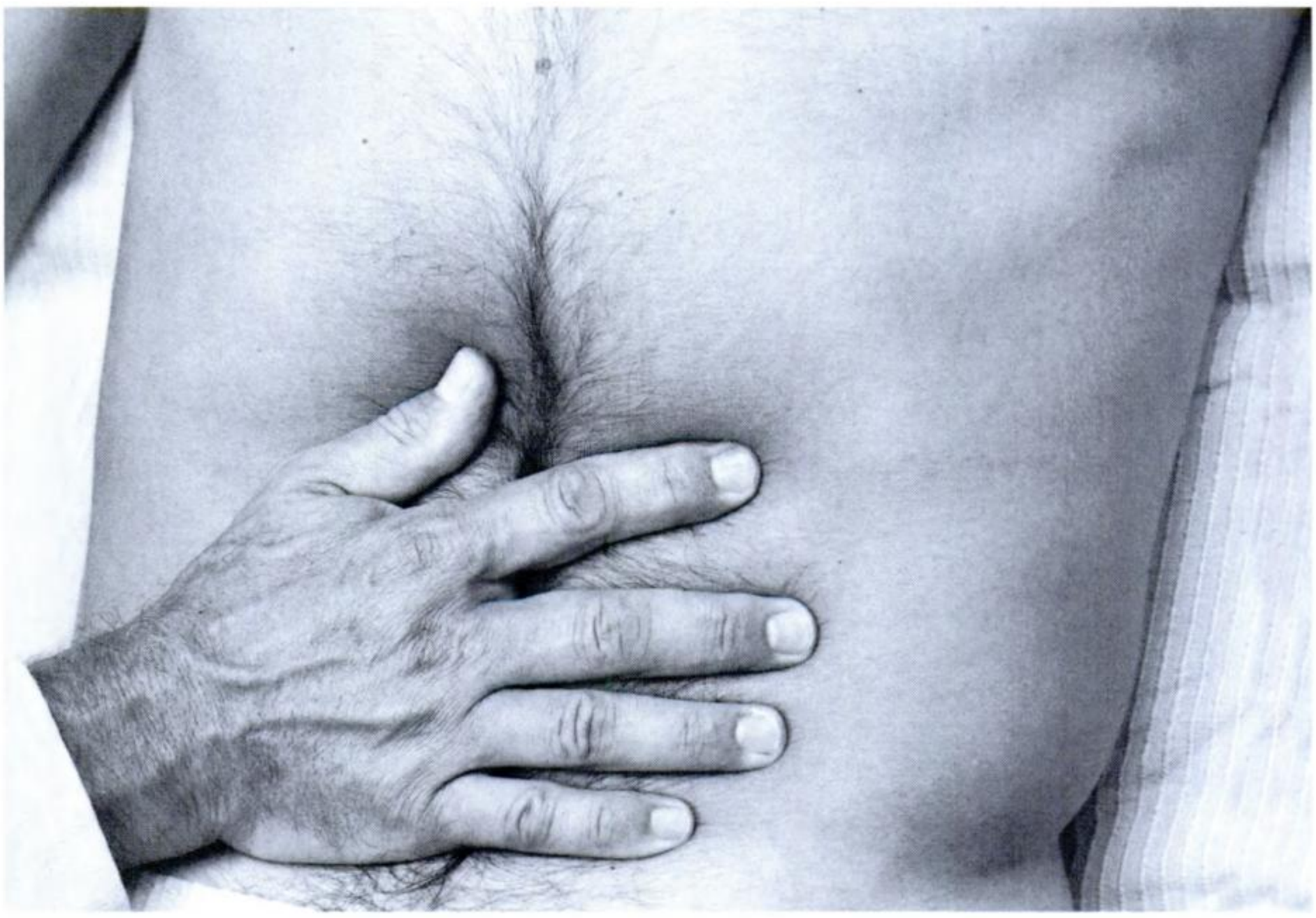
Figure 4–8 Use of a centimeter rule and a tongue depressor to measure the vertical distance (*arrow*) above the sternal angle of Louis (see Fig. 4–7).

In judging the jugular venous pressure, the response to abdominal compression is useful. The term "hepatojugular reflux" was coined by Rondot in 1898,[7] although observations on the response to abdominal compression were published in England a few years earlier.[8] Right upper quadrant pressure generally provokes the most pronounced response, but neither enlargement nor compression of the liver is required to elicit the response. In the presence of congestive hepatomegaly, right upper quadrant pressure may be painful. Central abdominal pressure (Fig. 4–9*A*) avoids the tender liver. Accuracy and practicality recommend the term "abdominojugular reflux" rather than "hepatojugular reflux."[9]

The technique of applying abdominal pressure is important. The patient is instructed to relax and breathe as quietly as comfort permits. The palm of the right hand is gently applied to the center of the abdomen (periumbilical) as illustrated in Figure 4–9*A*. The examiner's hand should be comfortably warm, and the contact gentle. When the patient is breathing quietly and has become accustomed to the examiner's hand, compression is gradually increased until the desired rise in jugular venous pressure is observed while the cervical veins are scrutinized with a pocket flashlight held in the left hand. Rapid pressure provokes reflex abdominal tension. Forewarning the patient is apt to result in anxious anticipation and is best avoided. The objective

is to minimize inadvertent straining (the Valsalva maneuver) or increased respiratory excursions which interfere with, if not cancel, the desired response. Compression is maintained for 10 to 15 seconds while the external and internal jugular veins are monitored.

The normal response to augmented venous return caused by abdominal compression is a transient increase in prominence of the external jugular vein and of the crests and troughs of the internal jugular (Fig. 4–9*B*). Within a few beats, this initial increase is followed by a fall to control levels as abdominal compression continues. If the initial rise in jugular venous pressure is *not* followed by a prompt fall, but instead is maintained during the entire 10 to 15 seconds of abdominal compression (Fig. 4–9*C*), the cause is likely to be right ventricular failure. However, a sustained response to abdominal compression also occurs with constrictive pericarditis and tricuspid stenosis. In the presence of emphysema or bronchospastic pulmonary disease, abdominal compression may result in an abnormal rise in jugular venous pressure in the absence of



A

Figure 4–9 *A*, The abdominojugular reflux performed with the palm of the hand gently but firmly applied to the center of the abdomen. *B*, The normal jugular response to abdominal compression *(arrow)* is a modest transient rise in the jugular pulse followed by a prompt fall to or below baseline level. *C*, Abnormal jugular venous response to abdominal compression *(arrow)* is a prompt obvious rise that is sustained throughout the 10 to 15 seconds of compression. (Modified from Sochowski RA et al. Clinical and hemodynamic assessment of the hepatojugular reflux. Am J Cardiol 66:1002,1990. Reprinted with permission.) *Continued*

right ventricular failure, presumably because a sudden increase in intrathoracic pressure impedes venous return.

Anatomic-Hemodynamic Inferences

Observations of abnormal waveforms of the right internal jugular vein permit gratifyingly precise, anatomic, and hemodynarnic inferences. The A wave, the X descent, the V wave, the Y descent, and occasionally the h wave all come under scrutiny.

A Wave

In sinus rhythm, the A wave regularly precedes the carotid pulse. If A waves are present but do not conform to this sequential relationship to the carotid, or if A waves are absent altogether, a disturbance in rhythm or conduction is the cause (see below). Giant A waves "leap to the eye, towering above and dwarfing the other waves

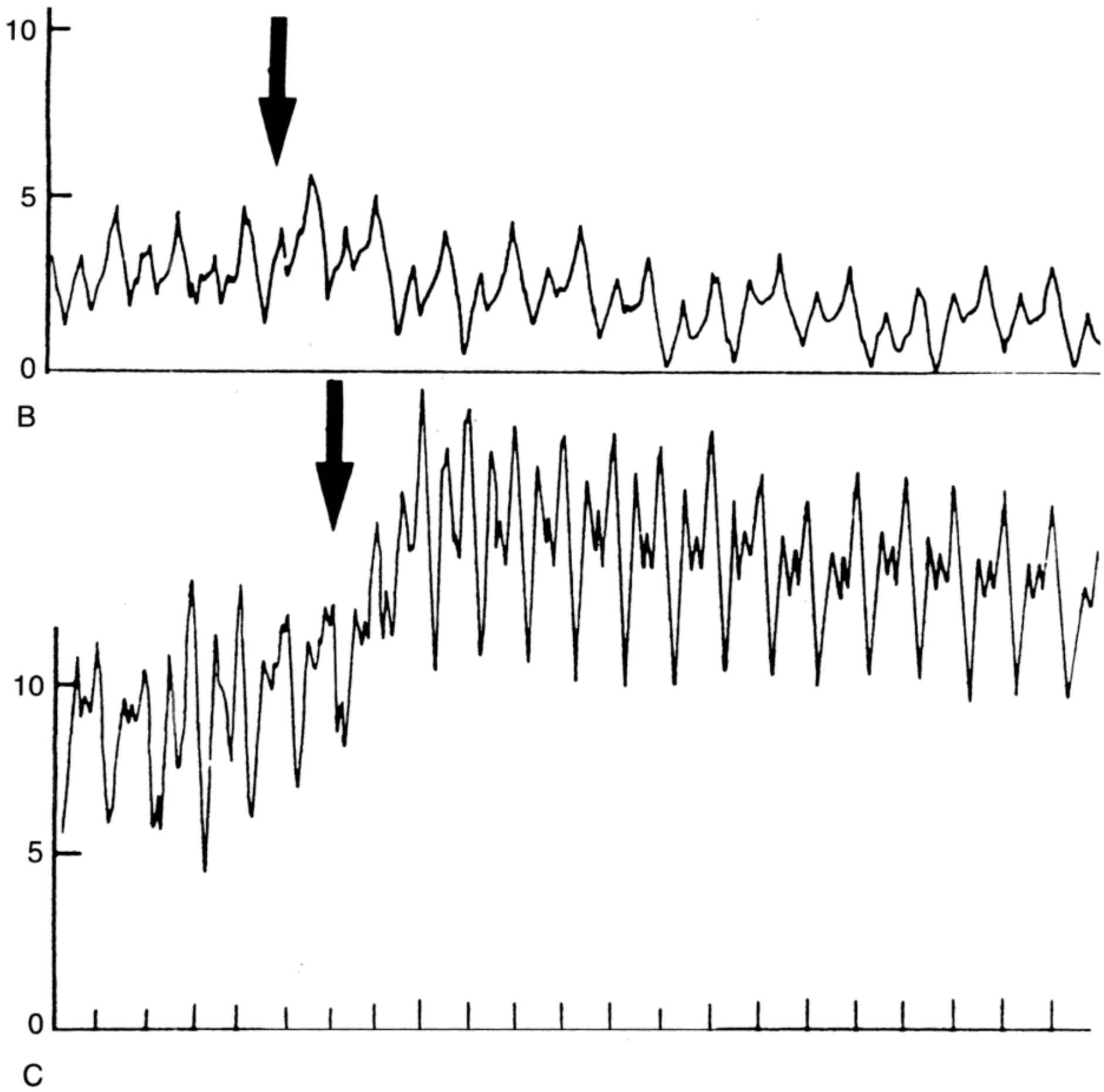


Figure 4–9 *Continued*

of the venous pulse."[2] Such a dramatic event has good reasons, as the following story illustrates:

One afternoon as I was seeing outpatients with Dr. Wood at the National Heart Hospital, he stood beside an examining table and looked intently at the neck of a young woman. Wood's comments went something like this:

> The pulsation in the neck is extraordinarily large. Even from a distance, I see amplification during inspiration, implying that the pulse is venous. Its regularity implies sinus rhythm. Assuming that this is so, the prominent pulse must be a giant A wave caused by powerful right atrial contraction against a major resistance. The resistance can only reside at the tricuspid orifice (tricuspid stenosis) or within a thick-walled hypertrophied right ventricle of severe pulmonary stenosis or pulmonary hypertension with intact ventricular septum.[2]

Wood then leaned forward, palpated the patient's left sternal edge, and remarked:

> There is no right ventricular impulse and no thrill of pulmonary stenosis. Nor can I feel a loud pulmonary component of the second heart sound. The A wave, therefore, cannot be due to either pulmonary stenosis or pulmonary hypertension, so this young woman must have tricuspid stenosis.[2]

And so she did!

Giant A waves are generated when a forceful right atrium contracts against an appreciable resistance at one of two levels—an obstructed tricuspid orifice or within the cavity of the thick-walled hypertensive right ventricle of severe pulmonary stenosis or pulmonary hypertension with intact ventricular septum. Wood referred to the giant A wave as the *venous Corrigan*,[2] indicating that he, like Dominic Corrigan, was referring to the dramatic *visual* pulsation in the neck (see Chapter 3).

Increased A waves range from mild at one end of the spectrum to giant at the other (Fig. 4–10). At the mild end, the A wave is abrupt and flicking rather than elevated. Inspiration or abdominal compression highlights this impression by transiently increasing venous return and augmenting the X descent.

X Descent

An increase in the initial portion of the X descent represents enhanced right atrial relaxation that follows augmented contraction due to the normal inspiratory increase in venous return or to the increased contractile force provoked by resistance to right atrial contraction. An increase in the briskness of the *second portion* of the X descent (descent of the floor of the right atrium) occurs in response to augmented *right ventricular* contraction. An important variation is constrictive pericarditis, in which the conspicuous X descent is due chiefly if not exclusively, to enhanced descent of the floor of the right atrium during right ventricular systole (Fig. 4–11).

In the normal heart, the X descent is brisker and deeper than the Y descent (see Fig. 4–2). Blunting of the X descent is an early sign of tricuspid regurgitation

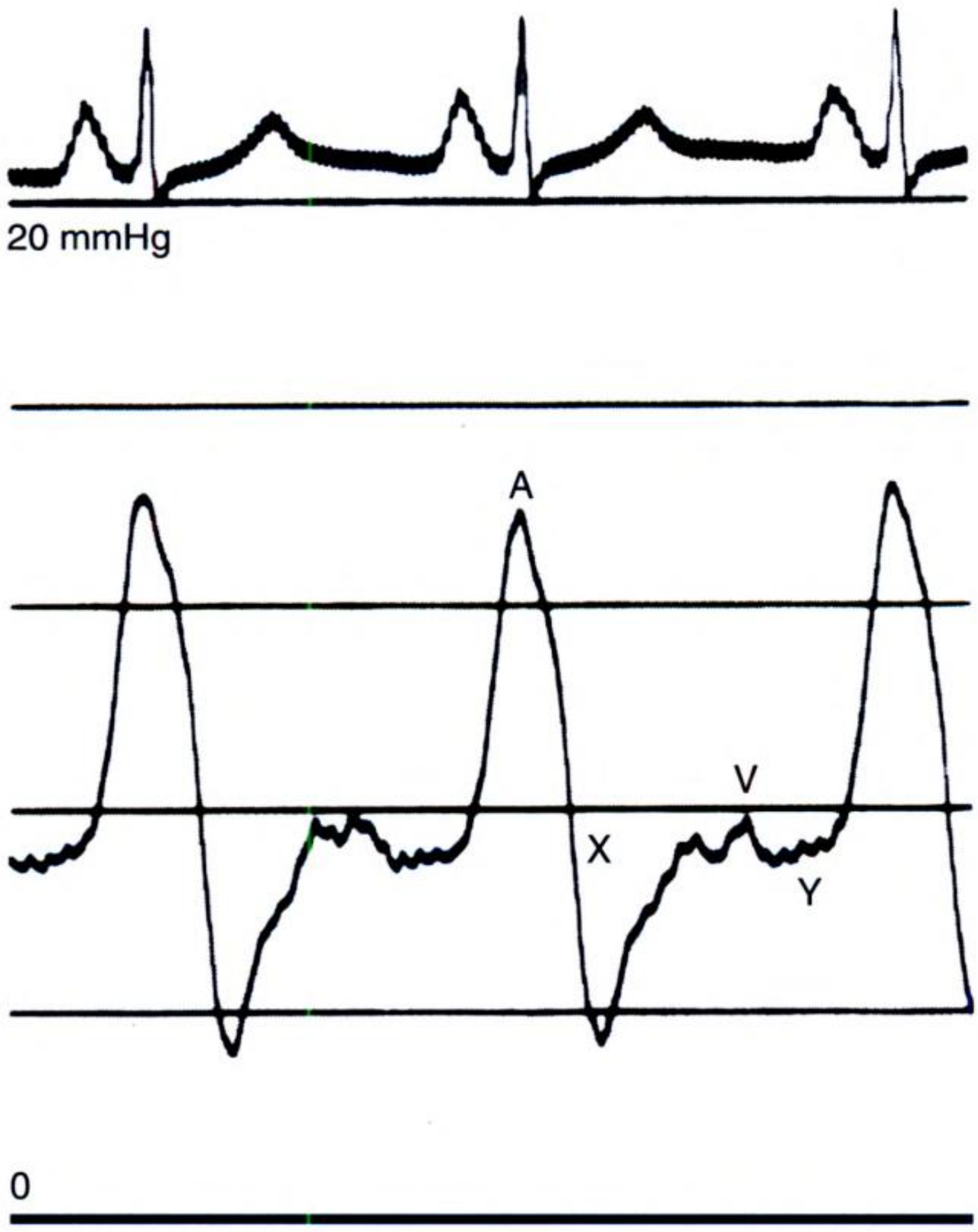


Figure 4–10 *A*, Giant A wave in an adult with severe rheumatic tricuspid stenosis. *B*, "When the auricular wave is large and stands out clear and distinct from other elements, it is evidence of a powerful right auricle." (Mackenzie J: The Study of the Pulse, Arterial, Venous, and Hepatic, and of the Movements of the Heart. Edinburgh, Young J. Pentland, 1902.)

which negates the portion of the X descent that coincides with right ventricular systole (Fig. 4–12).

V Wave and Y Descent

"One constantly finds clinical evidence of the ready occurrence of tricuspid incompetence in the study of the venous pulse."[1] In pure tricuspid regurgitation with sinus rhythm, the

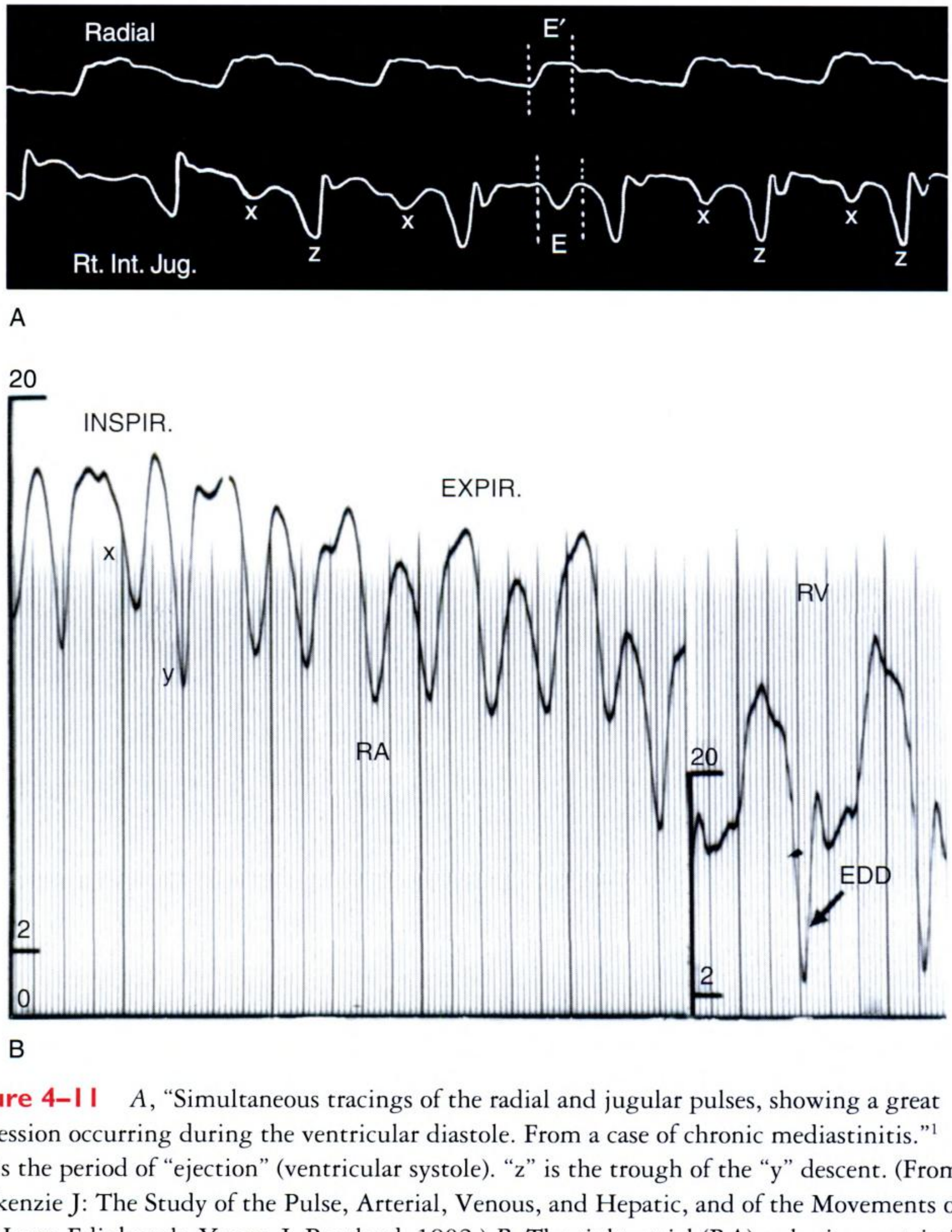
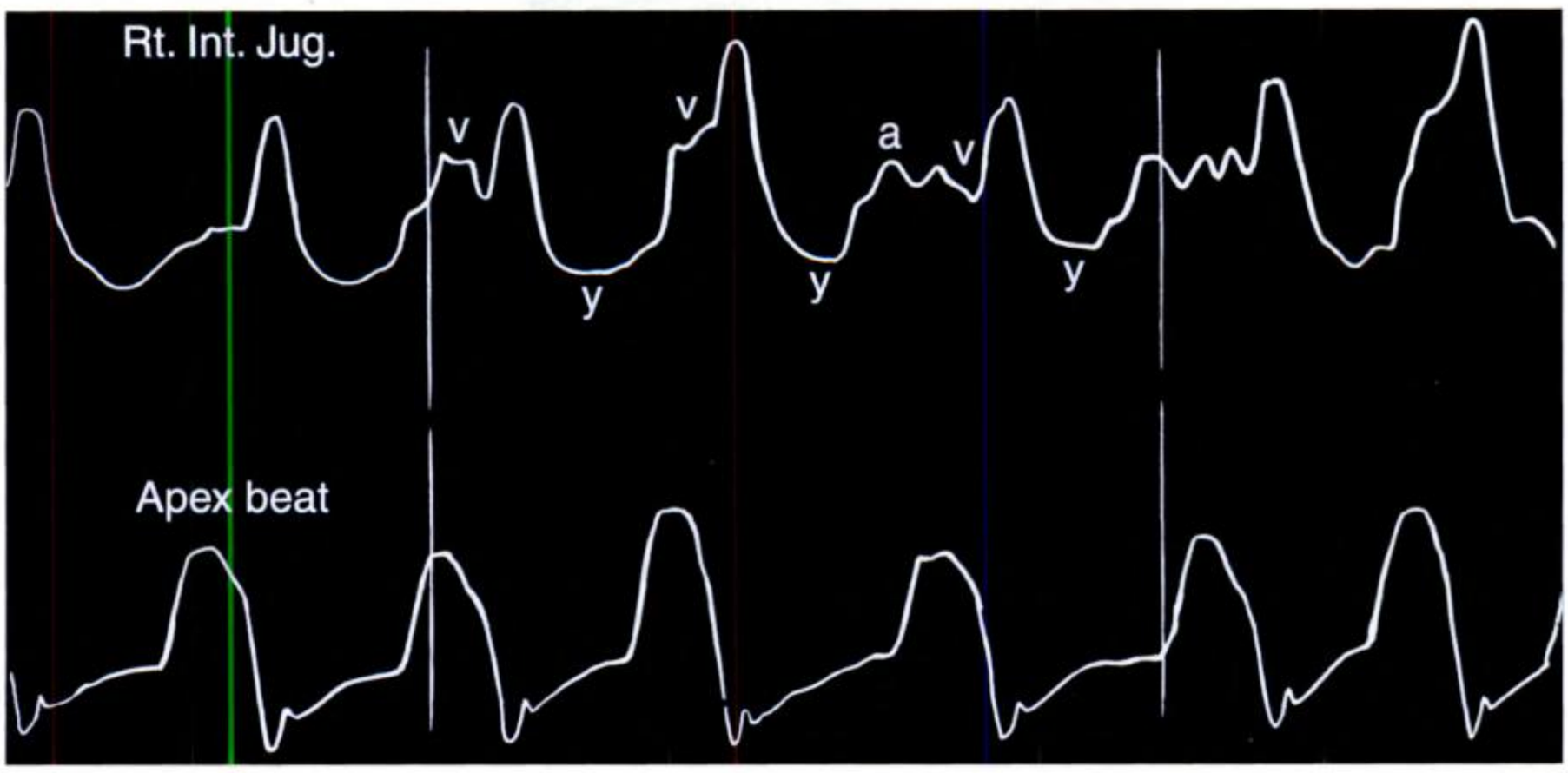
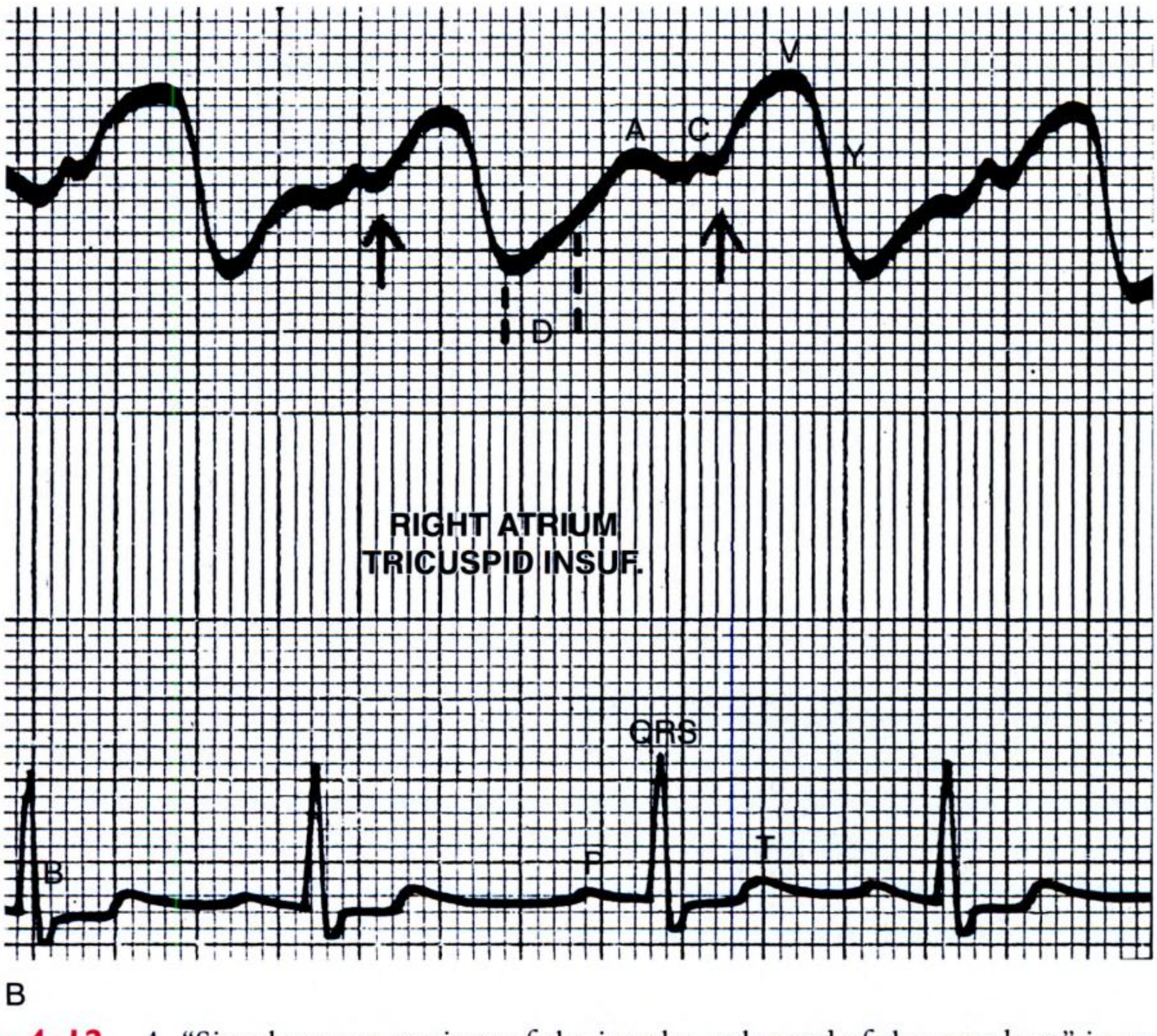


Figure 4–11 *A*, "Simultaneous tracings of the radial and jugular pulses, showing a great depression occurring during the ventricular diastole. From a case of chronic mediastinitis."[1] "E" is the period of "ejection" (ventricular systole). "z" is the trough of the "y" descent. (From Mackenzie J: The Study of the Pulse, Arterial, Venous, and Hepatic, and of the Movements of the Heart. Edinburgh, Young J. Pentland, 1902.) *B*, The right atrial (RA) pulse in constrictive pericarditis. There is a conspicuous "x" descent and an even more conspicuous "y" descent in addition to an inspiratory increase in the right atrial pressure pulse—Kussmaul's sign. In the right ventricular (RV) tracing, a steep early diastolic dip (EDD) (*arro*w) coincides with the "y" descent of the right atrial pulse.



A



B

Figure 4–12 *A*, "Simultaneous tracings of the jugular pulse and of the apex beat" in a patient "during an attack of heart failure."[1] Tricuspid regurgitation obliterated the x descent and increased the height of the v wave. (From Mackenzie J: The Study of the Pulse, Arterial, Venous, and Hepatic, and of the Movements of the Heart. Edinburgh, Young J. Pentland, 1902.) *B*, Right atrial pressure pulse in severe tricuspid regurgitation. The A/C wave is followed by obliteration of the second portion of the X descent. A tall V wave is followed by a brisk Y descent. D = diastasis.

ascent of the V wave is earlier in systole, its crest is higher, and the Y descent is brisker (Fig. 4–12). The tall V is sometimes called a "systolic venous wave."

The jugular pulse of tricuspid regurgitation in atrial fibrillation is characterized by an isolated tall, even giant V wave and a rapid Y descent (diastolic collapse) (Fig. 4–13).

> When the pulsation is due to the ventricular systole, the engorgement of the veins is usually so great, the arterial pulse so small, and the cardiac mischief so evident, that the recognition of the venous pulse is comparatively easy.[1]

In the presence of tricuspid regurgitation with a tall V wave, the valve of the right internal jugular vein (Fig. 4–1) becomes incompetent. Disproportionate systolic filling and expansion of the *right* internal jugular vein, which is in direct alignment with the right atrium (see Fig. 4–1*B*), results in a right-to-left *head bob*, best seen when the examiner observes the patient frontally.

A tall V wave with a *rapid* Y descent indicates that the tricuspid orifice is unobstructed. Conversely, a *slow* Y descent in the presence of a tall V wave is a feature of obstruction at the tricuspid orifice as in rheumatic tricuspid stenosis (Fig. 4–14). The Y descent is slow despite the tall V crest because the stenotic tricuspid orifice acts as a brake on the rate of right ventricular filling. The rate of rise of the V wave is not increased (Fig. 4–14), because the right atrium fills chiefly from the venae cavae, indicating that tricuspid regurgitation is mild or absent.

A modest increase in V wave is a subtle sign of a large atrial septal defect. The A and V waves in the jugular pulse are equal in height when a nonrestrictive atrial septal defect permits transmission of the *left* atrial waveform into the *right* atrium and internal jugular vein (Fig. 4–15).

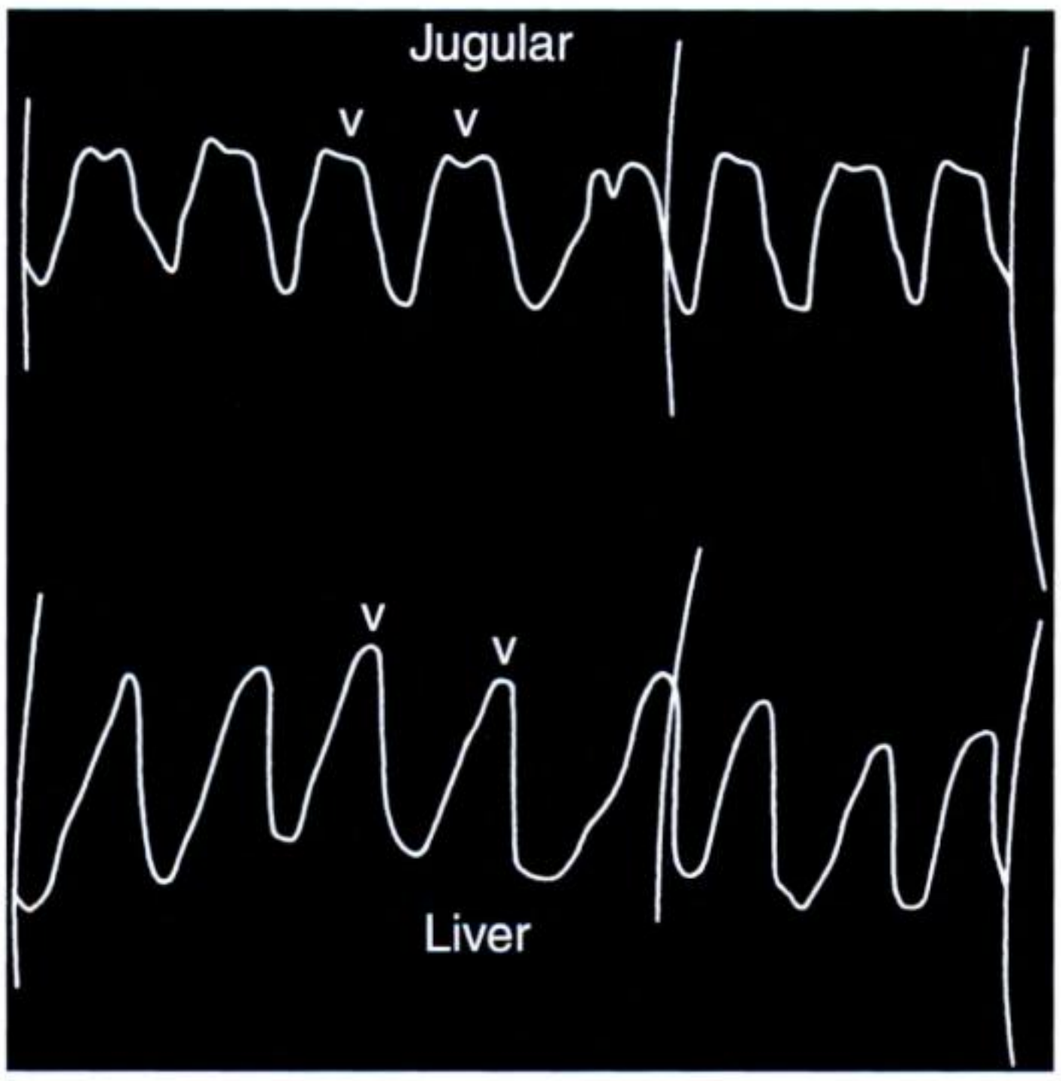


Figure 4–13 In this figure, "the venous and liver pulses of the ventricular type are given."[1] Both pulses show large v waves and brisk, if not collapsing y descents of tricuspid regurgitation. (From Mackenzie J: The Study of the Pulse, Arterial, Venous, and Hepatic, and of the Movements of the Heart. Edinburgh, Young J. Pentland, 1902.)

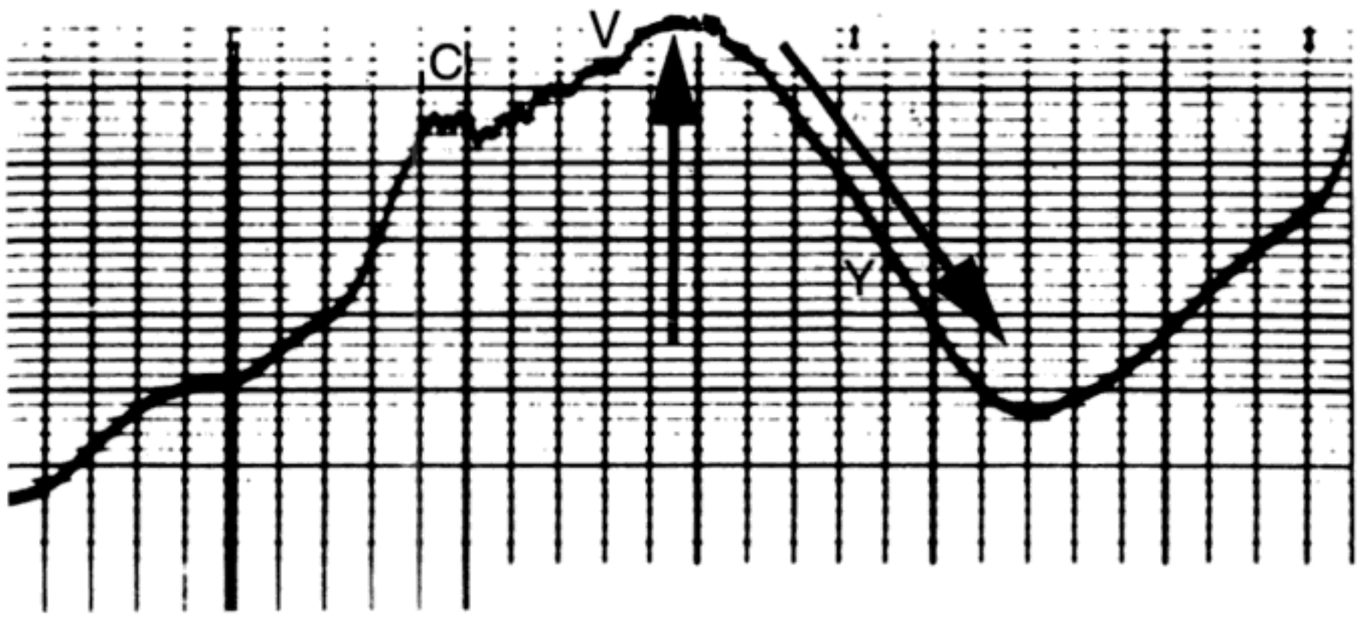


Figure 4–14 The jugular venous pulse in severe rheumatic tricuspid stenosis with atrial fibrillation. Despite a tall V wave *(vertical arrow)*, the Y descent is slow *(slanting arrow)* because obstruction at the triuspid orifice impedes the rate of flow.

Amplification of A and V Waves

The most common cause of an increase in A and V waves is right ventricular failure. Both crests are increased, but the A wave remains dominant as long as the tricuspid valve is competent. The later part of the X descent is less brisk because of diminished right ventricular systolic contribution to its descent. Rarely, right ventricular failure is associated with A and V waves that "alternate"—*right atrial pulsus alternans* (Fig. 4–16).

Tricuspid regurgitation with pulmonary hypertensive right ventricular failure is expressed in the jugular venous pulse as an increase in A and V waves with diminished X descent and a prominent if not collapsing Y trough. Pulmonary hypertension generates

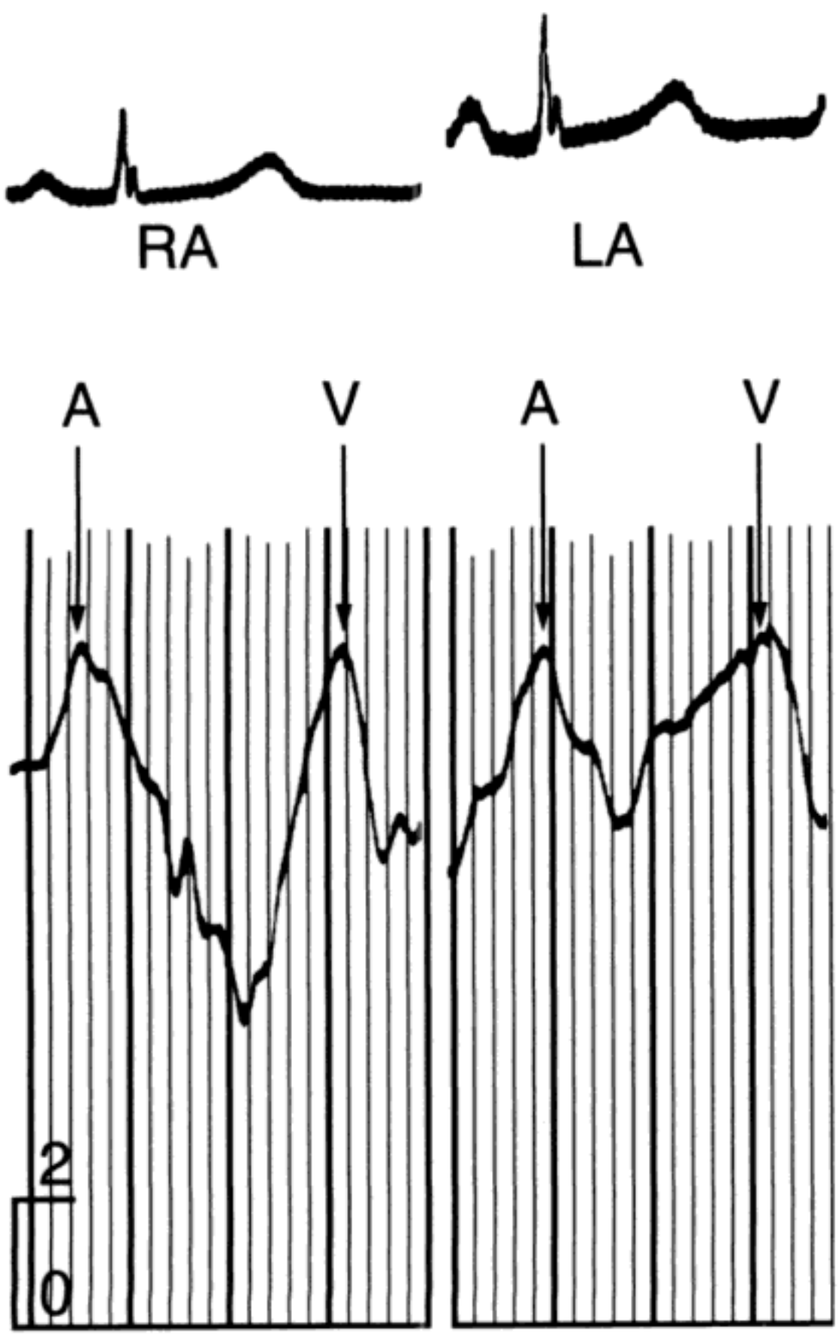


Figure 4–15 Right atrial (RA) and left atrial (LA) pressure pulses in a nonrestrictive ostium secundurn atrial septal defect. The A and V waves are equal in height and resemble a left atrial pressure pulse which is transmitted into the right atrium.

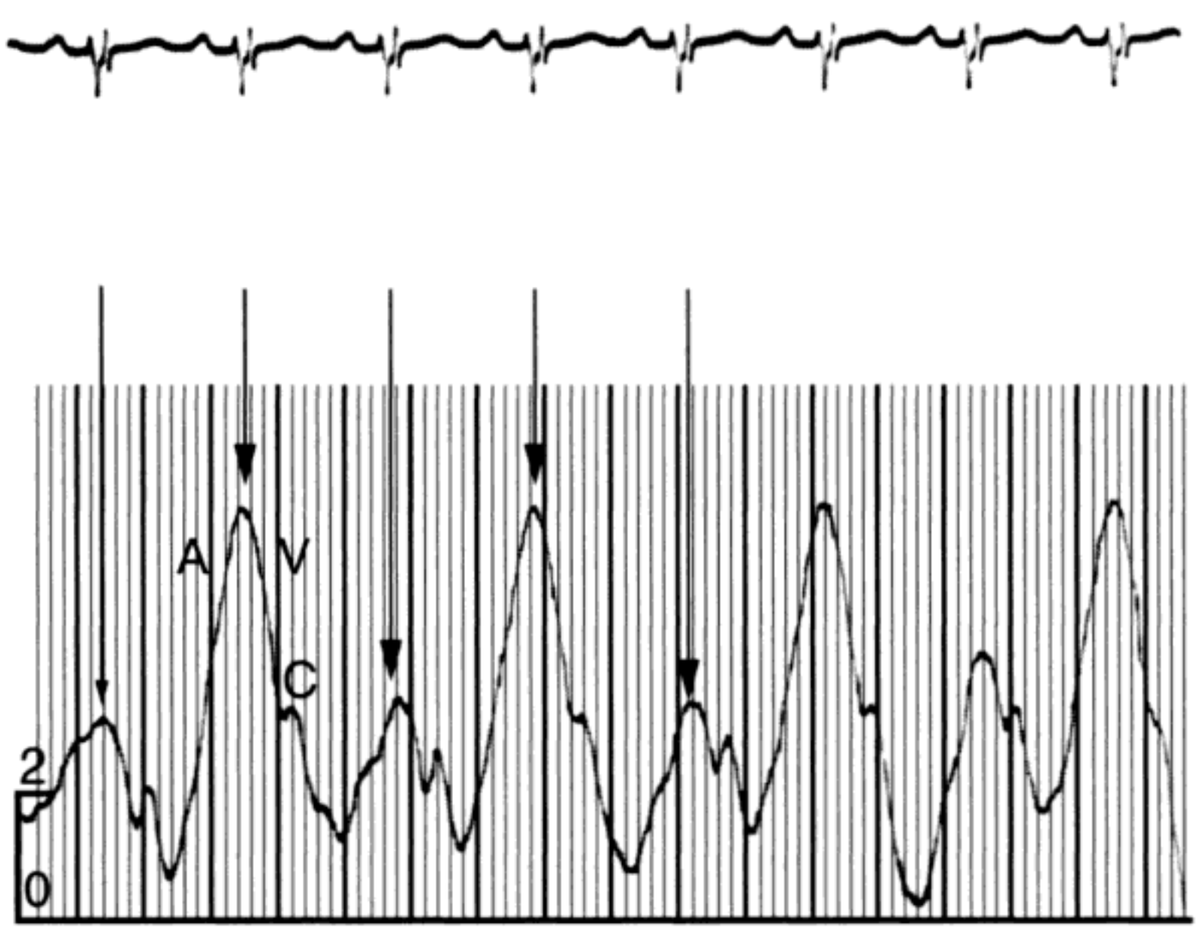


Figure 4–16 Striking right atrial alternans *(arrows)* in an infant with severe pulmonary valve stenosis and right ventricular failure. The A and V waves summate because of the rapid heart rate. C = carotid.

the large A wave, while tricuspid regurgitation blunts the X descent and increases the V crest and Y descent.

Appreciably elevated and equal A and V waves with inconspicuous pulsations but brisk X and Y descents (see Fig. 4–11) are best seen with the patient sitting bolt upright or even standing. Especially conspicuous and most consistent is the rapid Y descent to which the term "diastolic collapse" was applied by Friedreich in 1864.[10]

In chronic constrictive pericarditis, high nonpulsatile A and V crests are punctuated by rapid X and Y descents that impart a W-shaped pattern to the jugular pulse (Fig. 4–11*B*). Inspiration increases the rate of fall of both the X and Y descents, but does not significantly affect the A and V crests except as expressed in Kussmaul's sign,[11] a term still applied to an abnormal inspiratory increase in jugular venous pressure. Inspiratory augmentation in venous return results in an abnormal increase in mean right atrial pressure and in the A and V crests (see Fig. 4–11*B*) because the constricted right ventricle cannot accept the increase in volume without increasing its filling pressure.

The "*h wave*" is briefly mentioned here as a matter of completion. In 1907, Alexander G. Gibson wrote (Fig.4–2*C*):

> In the course of a study of jugular pulses in normal persons, I have noticed that in those whose pulse rate is slower than the average, a wave between the V wave and A wave is often to be seen.[12]

The *h* wave is also visible when diastasis occurs at a high right atrial pressure and a slow heart rate. This is so because flow from a high pressure right atrium into a right ventricle with impaired diastolic distensibility results in a prominent Y trough followed by a steep ascent to a plateau, the onset of which is seen as an *h* wave.

Hepatic Pulse

The hepatic pulse is cast into a secondary role because of the relative ease and clarity with which *jugular* venous pulsations can be seen. Nevertheless,

> In many cases the changes in volume of the liver offer a most constructive indication of the condition of the circulation.[1]

A liver pulse is anticipated when examination of the jugular pulse predicts transmission of an abnormal right atrial waveform into the inferior vena cava and into the liver. The fingertips perceive hepatic movements that coincide with the waveform of the jugular pulse (see Figs. 4–5 and 4–6). Hepatic pulsations are timed more easily when there is selective amplification of the presystolic or systolic component of the liver pulse. Mackenzie remarked:

> In tricuspid stenosis, when the auricle at first hypertrophies, the waves sent back by the auricle are of sufficient strength to cause a marked pulsation in the liver.[1]

The liver pulse is most dramatic in severe tricuspid regurgitation with hepatomegaly, because the right ventricle ejects across the incompetent tricuspid valve directly into the inferior vena cava and hepatic bed (see Fig. 4–13). Systolic movements of the liver impart visible motion to the right upper quadrant and the right lower thorax. Severe tricuspid regurgitation with hepatomegaly not only results in displacement of the liver edge, but also in *systolic* ex*pansion* of the liver identified by placing the palpating right hand upon the right upper quadrant while the left hand is applied directly posterior at the margin of the lower ribs.

Electrophysiologic Inferences—Arrhythmias and Conduction Defects

> I had been endeavoring to discriminate between the different forms of irregular heart action and it occurred to me to employ the jugular pulse as an aid. By this means I was able to separate the great majority of irregularities into definite groups, according to the mechanism of their production.[1]

Arrhythmias

H. J. L. Marriott's advice in diagnosing cardiac arrhythmias from the electrocardiogram, "*cherchez le P*," can be applied to the jugular venous pulse "*cherchez le A*."

Electrocardiographic analysis of complex arrhythmias requires identification of atrial and ventricular activity and their relationship to each other. The A and V waves of the jugular venous pulse provide the clinician with a bedside opportunity to do just this. Occasionally, the A wave is better seen in the jugular pulse than is the P wave in the electrocardiogram. Arrhythmias recognized in the jugular venous pulse are listed in Table 4–1.

The slow cardiac rate and regular rhythm of *sinus bradycardia* are recognized by the normal, regular sequence of A wave preceding V wave. However, 2:1 sino-atrial

Table 4–1 Arrhythmias Recognized in the Jugular Pulse

Sinus bradycardia
Sinus arrhythmia
Ectopic or premature atrial, junctional, or ventricular beats
Ectopic tachycardias or ectopic rhythms (atrial, junctional, ventricular)
Loss of coordinated atrial activity (atrial fibrillation)

exit block is indistinguishable from sinus bradycardia in the jugular pulse, as in the electrocardiogram. *Sinus arrhythmia* is recognized by rate accelerations that coincide with inspiration and are abolished when the breath is held in the respiratory midposition while the normal A-C-V sequence remains unchanged (Fig. 4–17). *Premature atrial beats* are represented by cycles that begin early but have the normal sequence of A wave, carotid pulse, and V wave. The changes in cycle length prompted by premature atrial beats are distinguished from the changes in cycle length of sinus arrhythmia because the latter are abolished during held respiration. In older subjects, premature atrial beats are sometimes accompanied by pauses because of prolonged sinus reset times. These pauses should not be mistaken for the compensatory pauses that follow premature ventricular beats. Error is avoided by observing the jugular pulse in which the atrial premature beat maintains a normal relationship between the A wave and carotid pulse.

Cannon wave refers to amplification of the A wave when right atrial contraction coincides with a closed tricuspid valve. In *ventricular* or *junctional* premature beats (Fig. 4–18), cannon waves are the rule because right atrial systole finds the tricuspid valve closed by the premature ventricular contraction. Accordingly, cannon waves distinguish ventricular premature beats followed by compensatory pauses from atrial premature beats with a prolonged sinus reset time. With junctional premature beats, cannon

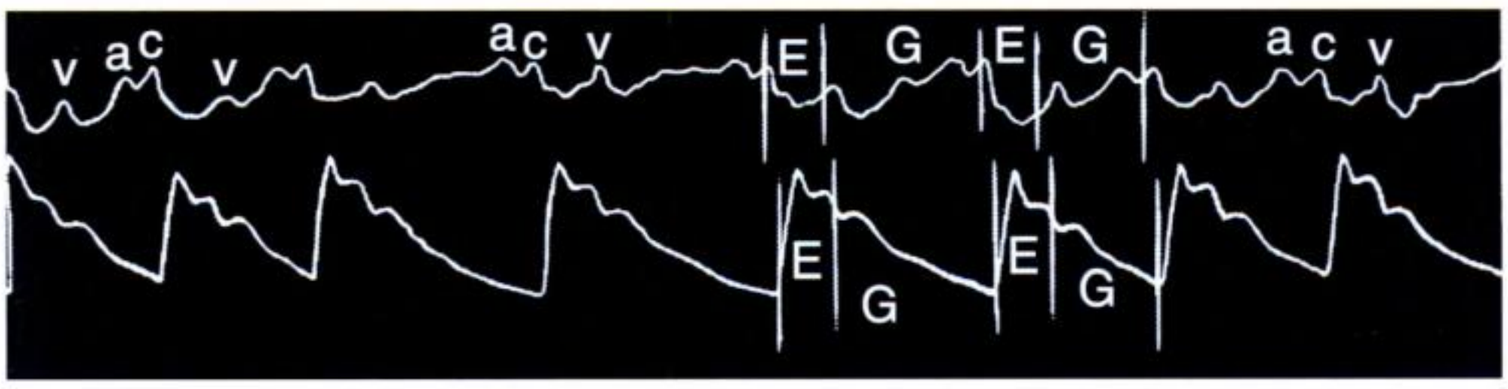


Figure 4–17 "Simultaneous tracings of the jugular and radial pulses showing agreement in rhythm of the right auricle and ventricle (waves a and v) with the radial pulse, in the youthful form of irregularity"[1] (sinus arrhythmia) "E" is the ejection period (ventricular systole). (From Mackenzie J: The Study of the Pulse, Arterial, Venous, and Hepatic, and of the Movements of the Heart. Edinburgh, Young J. Pentland, 1902.)

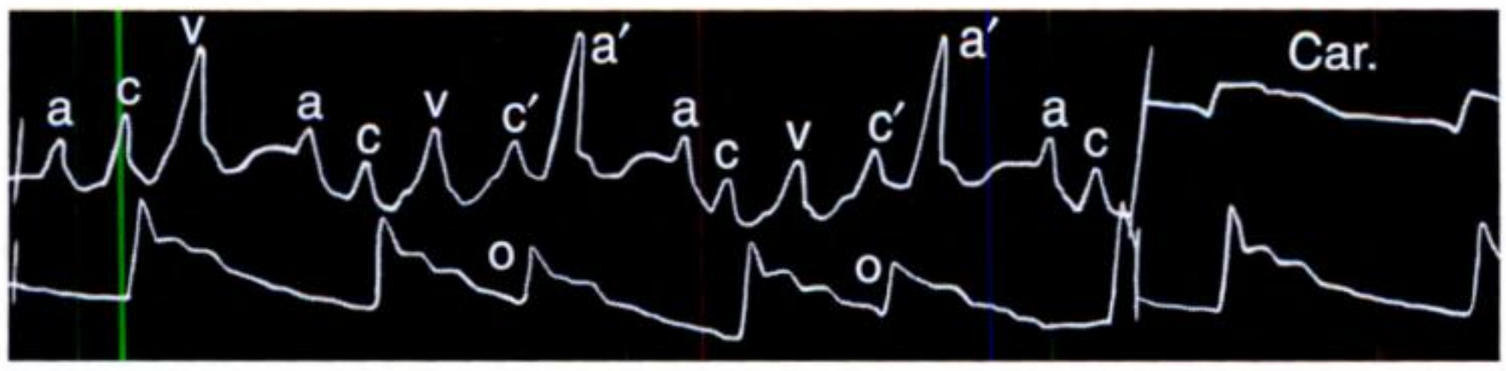


Figure 4–18 "Simultaneous tracings of the jugular and radial pulse. The auricular wave following a′ occurs earlier, as if to take on the ventricular rhythm."[1] The tracings illustrate cannon A waves (a′) that coincide with premature ventricular beats ("o") identified in the radial pulse. Car = carotid. (From Mackenzie J: The Study of the Pulse, Arterial, Venous, and Hepatic, and of the Movements of the Heart. Edinburgh, Young J. Pentland, 1902.)

waves occur whether or not the premature beats are accompanied by retrograde atrial activation (Fig. 4–19).

Recognition of *ectopic tachycardias* is, in part, an extension of the comments on individual ectopic beats. Continuous beat-to-beat cannon A waves in *junctional tachycardia* (Fig. 4–20) are examples of the sustained mechanism for generating intermittent cannon waves of individual junctional premature beats (see Fig. 4–19). Sustained junctional rhythm with rates above 100 beats per minute is called junctional tachycardia, and below 100 beats per minute is called junctional rhythm. In *atrioventricular nodal reentrant tachycardia*, a discernable A wave precedes each carotid pulse provided that the rate is not excessive. With rates above 160 beats per minute, the A and V waves

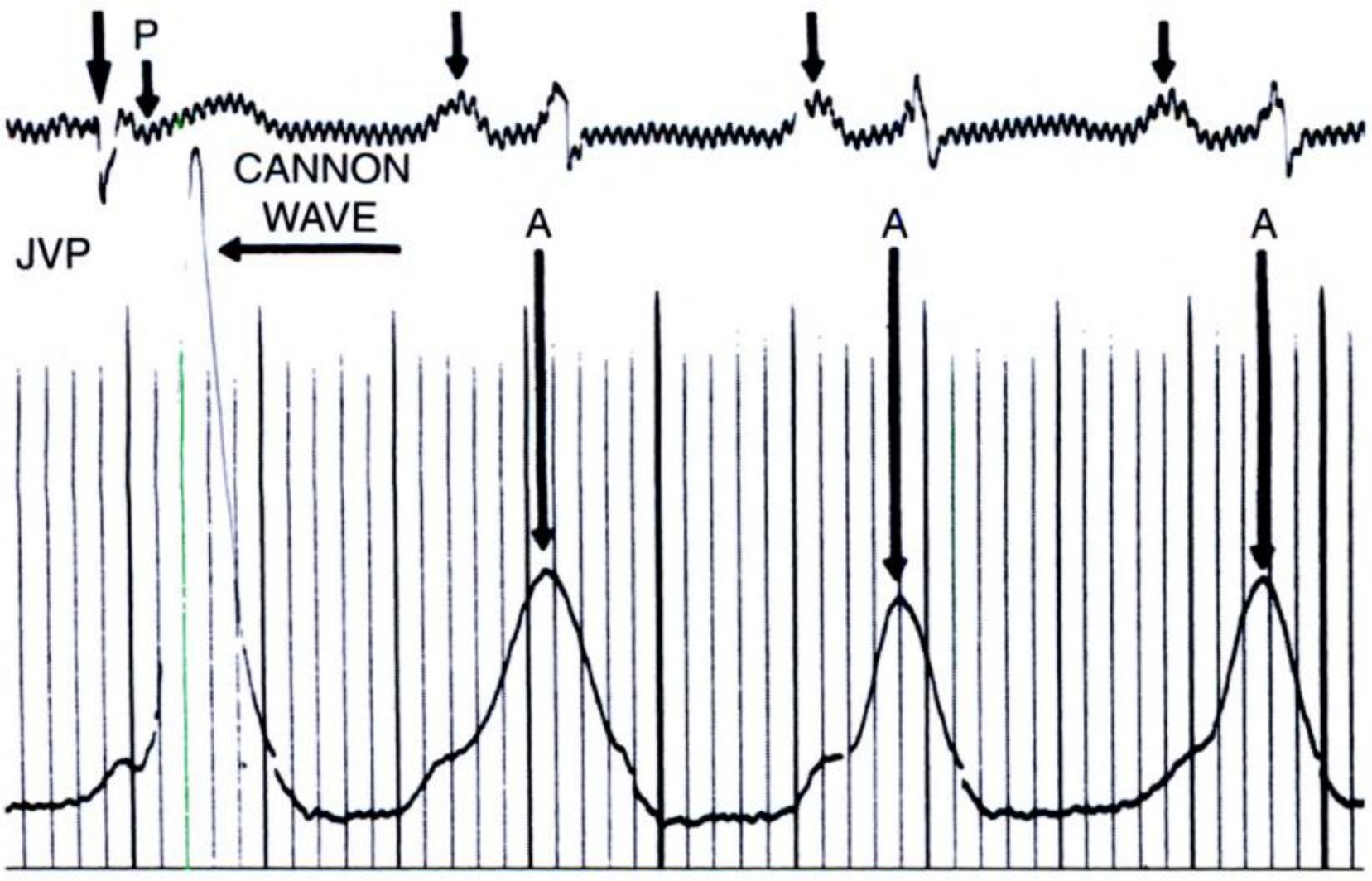


Figure 4–19 Jugular venous pulse (JVP) showing a cannon A wave (*horizontal arrow*) caused by retrograde atrial activation (inverted P wave) with a junctional premature beat (*first upper vertical arrow*).

Figure 4–20 Jugular venous pulse (JVP) with a junctional rhythm (1:1 retrograde atrial activation, inverted P waves) and regular sustained cannon waves. C = carotid.

merge into a single venous crest followed by a single descent. Mackenzie's description is relevant:

> With still greater increase of the pulse rate, the ventricular wave v becomes shortened even to such an extent that its rise is blended into that of the auricular wave, so that only one true venous pulse wave is recognized.[1] (Fig. 4–21)

These summated A and V crests resemble beat-to-beat cannon waves of junctional tachycardia (Fig. 4–20), the distinction lying chiefly in the difference in heart rates. Atrioventricular nodal reentrant tachycardia is usually associated with rates of 160 to 250 beats per minute, whereas in junctional tachycardia, the rates are slower, generally just above 100 beats per minute. However, in *atrioventricular nodal reentrant tachycardia*, when atrial and ventricular activations are simultaneous, or in *circus movement tachycardia* when ventricular activation immediately precedes atrial activation, atrial contraction is against a closed tricuspid valve, producing rhythmic cannon waves in the jugular pulse.

Ventricular tachycardia with 1:1 retrograde atrial activation results in beat-to-beat cannon waves indistinguishable from those of junctional tachycardia. More commonly, however, ventricular tachycardia produces a fundamentally different jugular venous pulse because atrial activity is *independent of* ventricular activity (atrioventricular dissociation). Ventricular tachycardia is recognized in the jugular pulse by a regular rhythm and rapid rate as judged by palpation of the carotid, accompanied by independent A waves at

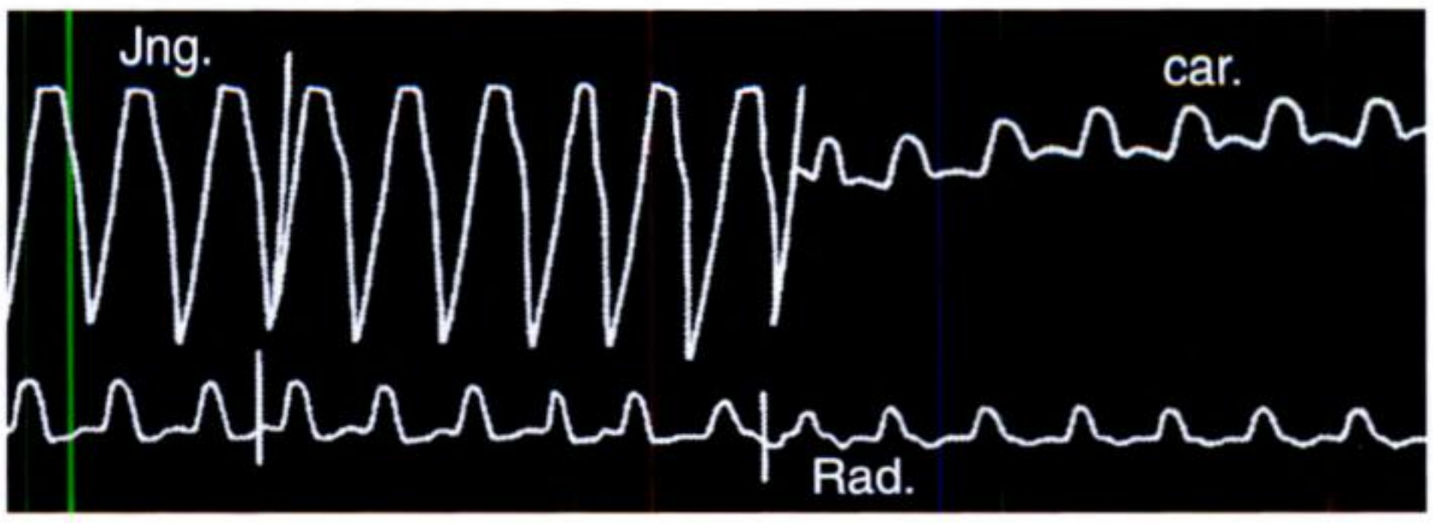


Figure 4–21 "Simultaneous tracings of the pulsation in the jugular bulb and in the radial and carotid pulses during an attack of paroxysmal tachycardia, taken 18 hours from the beginning of the attack."[1] The illustration shows summation of A and V waves into a single venous crest followed by a single descent. Jug = jugular; car = carotid; Rad = radial. (From Mackenzie J: The Study of the Pulse, Arterial, Venous, and Hepatic, and of the Movements of the Heart. Edinburgh, Young J. Pentland, 1902.)

a rate slower than the rate of the carotid pulse. Importantly, and distinctively, fortuitous atrial contraction against a closed tricuspid valve results in intermittent amplification of right atrial beats—cannon A waves. Accordingly, regular tachycardia occurring with slower independent A waves and intermittent cannon waves is characteristic of ventricular tachycardia. When P waves cannot confidently be identified in the electrocardiogram, this observation is useful in distinguishing ventricular tachycardia from supraventricular tachycardia with aberrant ventricular conduction.

Atrial flutter is represented by coordinated atrial activity at rates of about 300 beats per minute, and a regular ventricular response at 150 beats per minute (2:1 atrioventricular block). The ease with which flutter waves are seen in the jugular venous pulse (Fig. 4–22) depends on the functional condition of the right atrium prior to the onset of flutter. When *sinus rhythm* with large A waves is replaced by *atrial flutter*, the flutter waves are likely to be large as shown in Figure 4–22. Carotid sinus massage transiently slows the ventricular response, facilitating detection of flutter waves in the jugular pulse. Similarly, when atrial flutter occurs with a higher degree atrioventricular block (generally 4:1), flutter waves are easier to identify in the jugular pulse because diastole is relatively long. Flutter waves amplify in bursts when the right atrium flutters against a closed tricuspid valve (Fig. 4–22).

Atrial fibrillation is represented in the jugular pulse by absence of coordinated atrial activity. Loss of A waves in the presence of an irregular rhythm as judged by palpation of the carotid distinguishes the irregular rhythm of *atrial fibrillation* from the irregular rhythm of multiple premature atrial or ventricular beats in which A waves are present and often distinctive, as described earlier. Because absent right atrial contraction is necessarily accompanied by absence of relaxation, the initial portion of the X descent is lacking, so the X trough is relatively inconspicuous.

Conduction defects recognized in the jugular pulse are listed in Table 4–2. The *PR interval* can be estimated by the interval between the A wave and carotid pulse. The

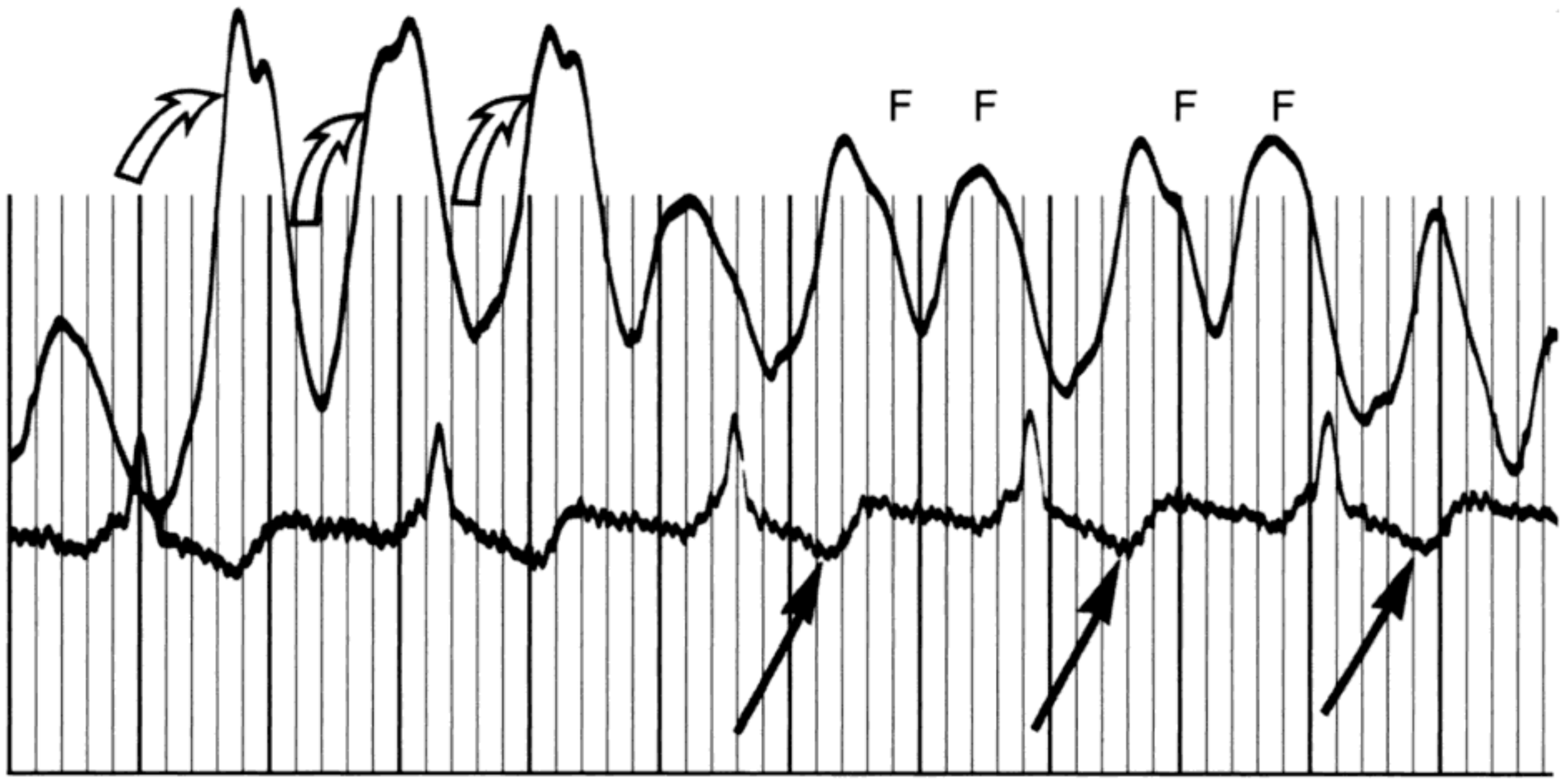


Figure 4–22 Jugular venous pulse with prominent flutter waves (F) that amplify in bursts (*upper curved arrows*) in a case of rheumatic mitral stenosis. The electrocardiogram shows flutter waves (*arrows*).

practiced eye comes to recognize normal, prolonged, or short A-C (and therefore PR) intervals. Complete *left bundle-branch block* slightly prolongs the A-C interval by delaying left ventricular activation. A corollary of A-C prolongation is *Wenckebach periodicity* that is characterized in the jugular pulse by gradual lengthening of A-C intervals, culminating in an A wave that is not followed by a carotid pulse (nonconducted beat). Mobitz I Wenckebach periods[13] differ from Mobitz II atrioventricular block that is represented in the jugular pulse by A-C intervals that do not vary but are suddenly interrupted by an isolated A wave that is not followed by a carotid pulse (a nonconducted beat). Interestingly, Wenckebach described the periodicity that bears his name in 1899, which was *prior* to the introduction of electrocardiography into clinical practice[13] (see Chapter 3). In *two-to-one atrioventricular block*, there are two A waves for every carotid pulse (Fig. 4–23). The X descent of the blocked (nonconducted) A wave is blunted because only the portion coinciding with right atrial relaxation is present. The second portion of the X descent is absent because the blocked A wave is not followed by ventricular systole and descent of the floor of the right atrium.

Table 4–2 Conduction Defects Recognized in the Jugular Pulse

PR interval prolongation
Wenckebach periods
Second degree atrioventricular block
Complete heart block

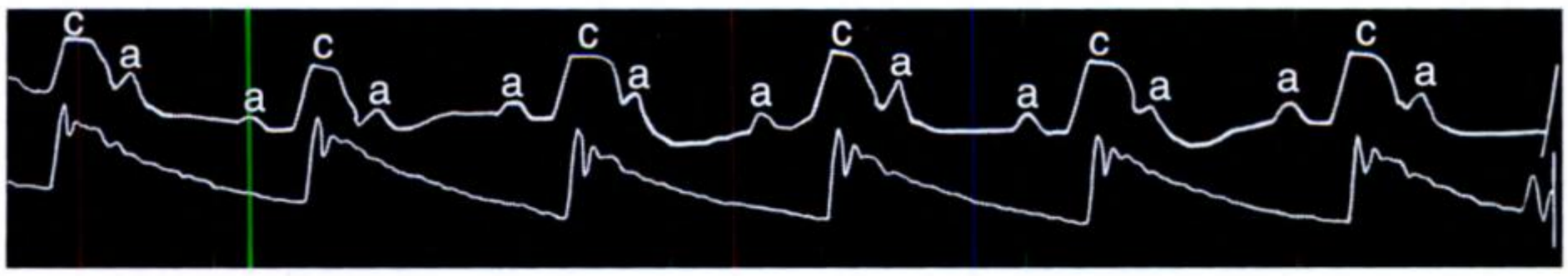


Figure 4–23 "Tracings of the pulsation in the neck due to a wave, *a*, in the jugular vein and the carotid pulse, *c*, taken at the same time as the radial. There are two auricular waves, *a*, to one carotid pulse" (2:1 atrioventricular block.) (From Mackenzie J: The Study of the Pulse, Arterial, Venous, and Hepatic, and of the Movements of the Heart. Edinburgh, Young J. Pentland, 1902.)

Complete atrioventricular block was described by William Stokes (1846) in his "Observations on Some Cases of Permanently Slow Pulse."

> A new symptom has appeared, a very remarkable pulsation in the right jugular vein. The number of the reflex pulsations is difficult to be established, but they are more than double the number of the manifest ventricular contractions. About every third pulsation is very strong and sudden and may be seen at a distance; the remaining waves are much less distinct and some very minor ones can also be perceived. The appearance of this patient's neck is very singular, and the pulsation of the veins is of a kind which we have never before witnessed.[14]

In complete heart block, coordinated atrial activity is more rapid than and is dissociated from ventricular activity that arises from a slow, regular, idioventricular focus (Fig. 4–24). The long diastoles disclose independent A waves, which are punctuated by intermittent cannon waves as right atrial contraction fortuitously coincides with right ventricular systole and a closed tricuspid valve (Fig. 4–24). Accordingly, complete heart block is diagnosed in the jugular venous pulse when intermittent cannon A waves occur with smaller independent A waves at a rate faster than the ventricular bradycardia identified by the carotid pulse (Fig. 4–24).

VEINS OF THE EXTREMITIES AND THORACIC INLET

Lower Extremities

Physical examination of lower extremity veins seeks chiefly to identify varicose veins, thrombophlebitis (superficial and deep), and signs of chronic venous insufficiency. *Superficial thrombophlebitis* is characterized by a visible and palpable thrombus in a vein just beneath the skin, hence *superficial.* Commonest sites are the saphenous veins and their tributaries. In the acute state, an indurated, palpable tender cord of varying length is accompanied by a visible red line along the course of the inflamed vessel without edema. As the acute inflammation subsides, a palpable nontender cord sometimes persists for weeks. Thromboplebitis usually destroys venous valves, rendering them incompetent when the leg is dependent.

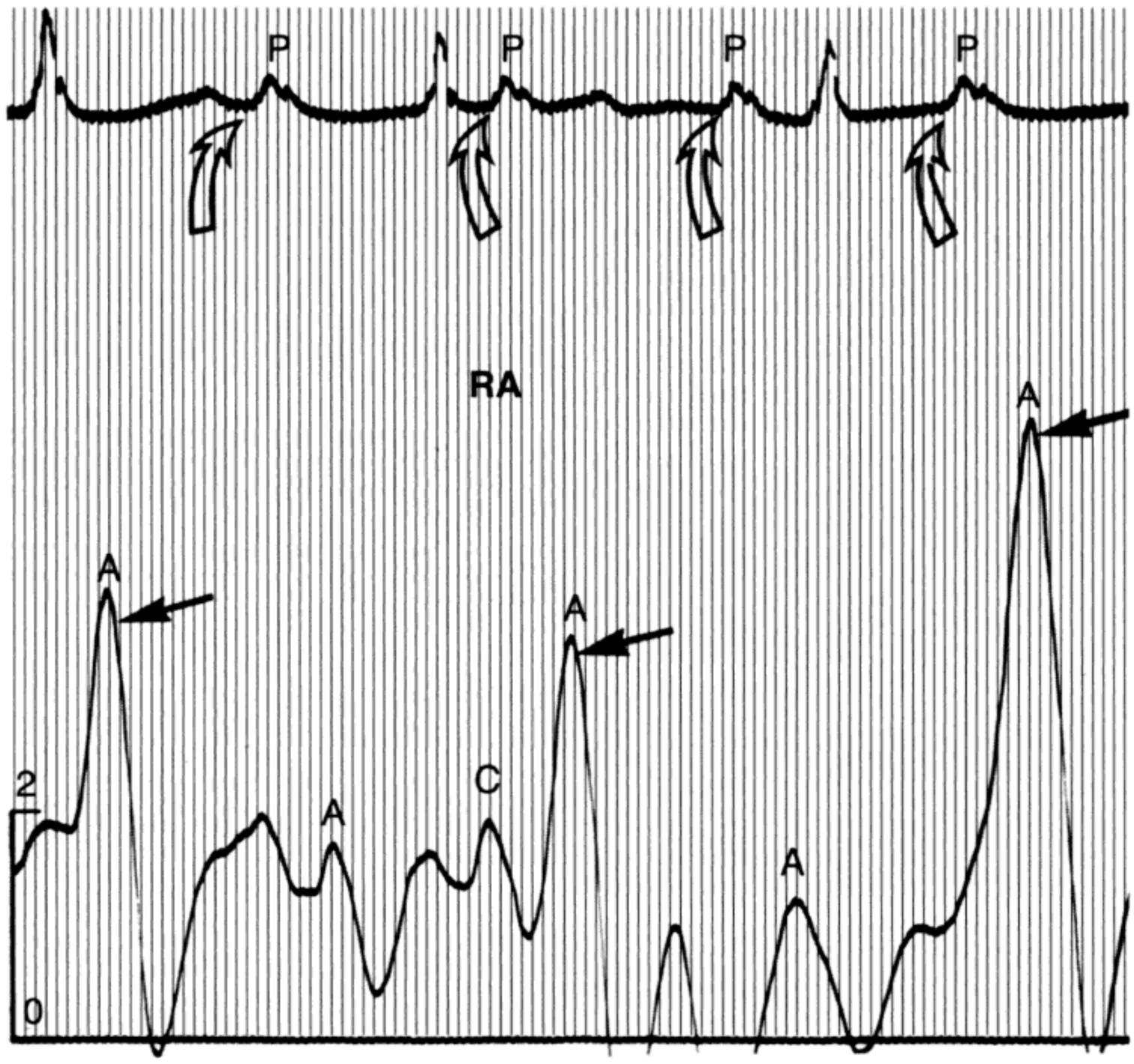


Figure 4–24 Right atrial (RA) pressure pulse in complete heart block. P waves (*curved arrows*) in the electrocardiogram are independent of the QRS. The venous waveform shows independent A waves and intermittent cannon A waves *(arrows). C* = carotid.

Deep thrombophlebitis typically involves calf veins or iliofemoral veins and may initially escape attention because physical signs and symptoms are apt to be scanty. It is therefore important to seek the signs of deep vein thrombophlebitis in clinical settings that heighten suspicion—congestive heart failure, postpartum, after major surgery, during protracted debilitating illnesses, and in elderly patients who sit for hours with legs dependent and knees flexed. Local calf tenderness is often the earliest sign. With the patient supine and the knee flexed, the relaxed calf muscles are gently compressed against the tibia (Fig. 4–25). Gentle compression with the fingertips, is preferable to squeezing the calf, a maneuver that can elicit pain even in normal subjects. *Homans' sign*—dorsiflexion of the foot—is far less useful than careful systematic compression of the calf as just described. Homans' sign is positive in only about 50 percent of patients with calf deep vein thrombophlebitis, and the sign can be elicited in the absence of venous thrombosis in patients with muscular disorders of the lower extremities. In addition to local tenderness, deep vein thrombosis is generally accompanied by swelling together with increased skin temperature and tissue turgor. Iliofemoral venous thrombosis is accompanied by tenderness, pain, edema, skin suffusion, increased skin temperature, and visible superficial venous patterns.

Varicose veins, recognized by Hippocrates 2500 years ago, are the most common clinical vascular abnormality of the lower extremities. Nevertheless, varicose veins are

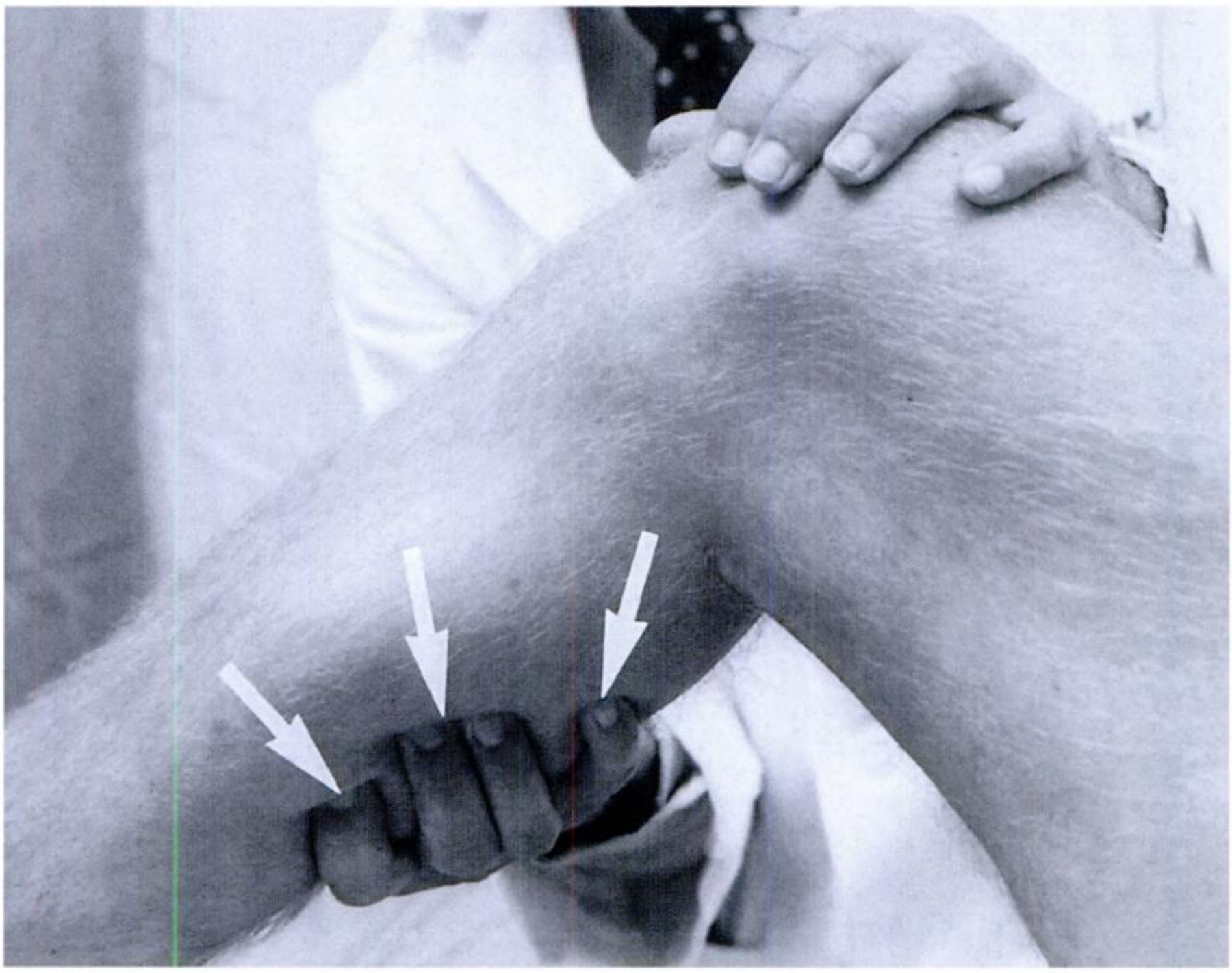


Figure 4–25 With the patient supine and knee flexed, the fingertips *(arrows)* gently compress the calf muscles against the tibia.

often overlooked during routine physical examination because the legs are not uncovered and properly examined while the patient is standing.

The word *varicose* is derived from the Latin *varicosus*, the adjectival form of *varix*, meaning "tortuous blood vessel." *Varices*, the plural of varix, is a term that remains in general use. Varicose veins, varices, or varicosities vary from cosmetically undesirable but medically unimportant clusters of small intracutaneous vessels, to the dramatic, serpentine dilatation shown in Figure 4–26. Gravity distends the vessels, so examination in the standing position is obligatory (see above).

Hieronymus Fabricus (1537–1619) described venous valves as "extremely delicate little membranes in the lumen of veins," an observation that influenced William Harvey's study of the circulation of the blood. Incompetence of venous valves—superficial (saphenous) and deep (iliofemoral)—sets the stage for chronic venous insufficiency, the signs of which are characteristic. The leg is swollen, and the skin and subcutaneous tissues of the lower third of the leg and ankle are discolored by brownish stasis pigmentation (Fig. 4–26). Cellulitis, skin erosion, and ulcerations develop, especially around the malleoli, more commonly the internal malleoli.

The high systemic venous pressure accompanying right ventricular failure aggravates distention of varicosities. Tricuspid regurgitation, especially when severe, occasionally imparts systolic pulsations to the varicose veins.

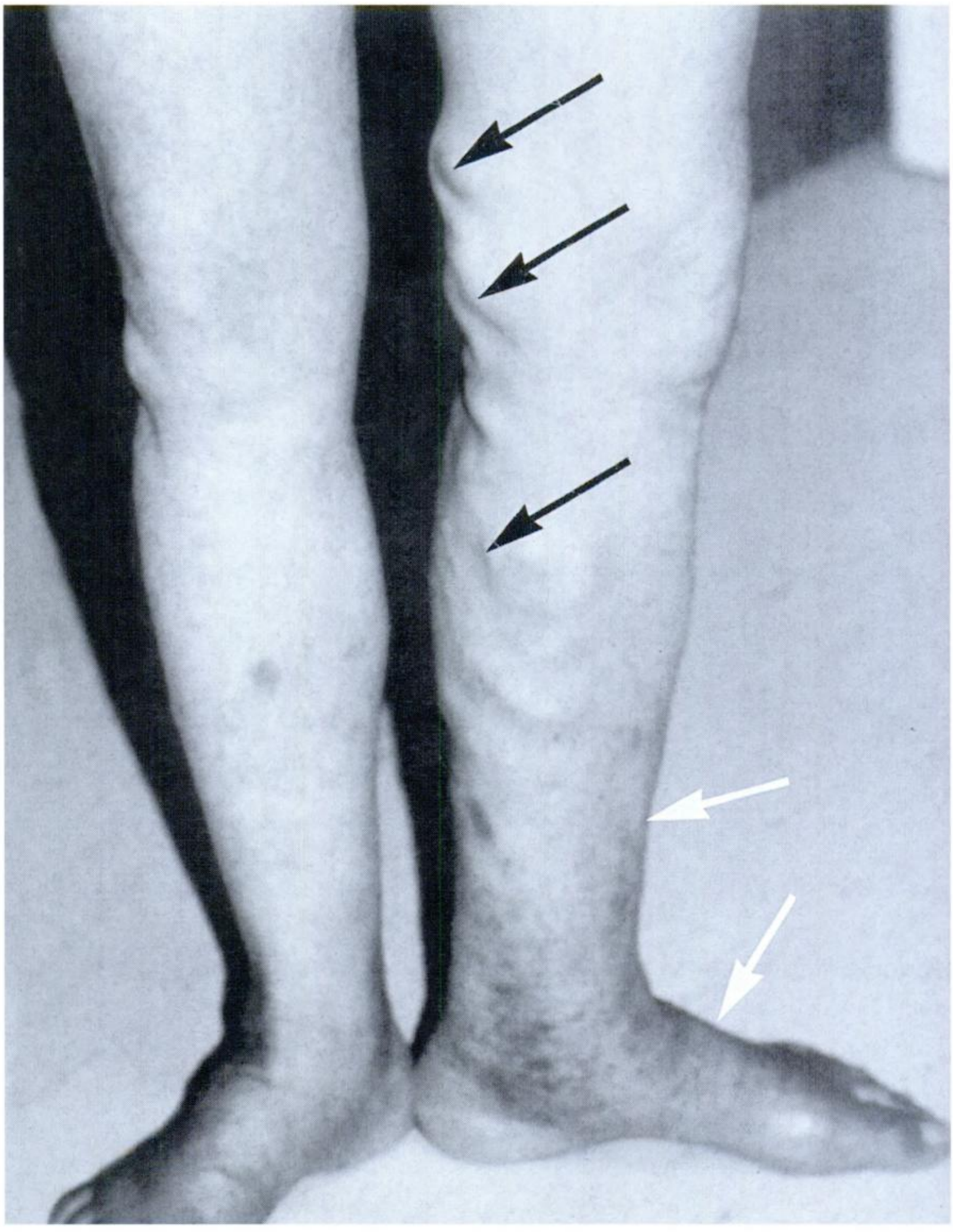


Figure 4–26 Varicose veins (*black arrows)* distend when the patient stands. In the ankle and foot, *white arrows* identify the swelling and discoloration of chronic venous stasis. (Courtesy of, Dr. Ronald W. Busuttil, Professor of Surgery, UCLA Medical Center.)

Upper Extremities

Distention of veins of the dorsa of the hands or antecubital fossae without intrinsic venous disease occurs when central venous pressure is appreciably elevated. Dorsal hand veins have been used to assess central venous pressure. With the patient's trunk above the horizontal and adjusted for comfort, the hand under scrutiny is first lowered beneath the sternal angle without flexing the elbow until its dorsal veins distend. The arm is then

gradually and passively raised while the dorsal veins are observed as the hand approaches the level of the sternal angle. Normally, these hand veins empty at that level when the patient's trunk is 30 degrees above the horizontal. Persistent distention indicates elevation of the central venous pressure, the height of which is estimated by the vertical distance above the angle of Louis at which the dorsal veins visibly collapse.

Transmission of systolic pulsations of severe tricuspid regurgitation into dorsal hand veins implies incompetence of the valve of the subclavian vein at the thoracic inlet (Fig. 4–1) in the presence of high systemic venous pressure.

Intrinsic disease of veins of the arms and hands is uncommon apart from complications of intravenous infusions and injections. The tender, indurated red cord at the site of a heparin lock is a case in point. Not so innocent are the nontender pigmented "mainline" streaks and palpable cords caused by intravenous drug abuse (see Chapter 2, Fig. 2–36). The common sites are antecubital fossae, ventral surfaces of the arms, and dorsa of the hands.

Veins of the Thoracic Inlet

Axillary and subclavian thrombophlebitis are rare but result in a relatively typical array of physical signs. Axillary vein thrombophlebitis usually occurs in young patients after trauma to or excessive exertion of the arms. The extremity becomes swollen over a period of hours, and assumes a reddish, cyanotic hue. The skin is warm, the superficial veins are distended, and there is axillary tenderness. When the subclavian vein is the site of thrombophlebitis, tenderness is elicited along its course above the clavicle.

Superior vena caval obstruction—the *superior vena cava syndrome*—can be diagnosed at the bedside.[15] The deep jugular veins are bilaterally distended but *nonpulsatile*. Sudden superior vena caval obstruction is uncommon and is characterized by dramatic distention of the neck veins together with cyanosis and pronounced edema of the face and eyes. *Chronic* obstruction is accompanied by suffusion and cyanosis of the face, neck, and arms, together with dilated, serpentine vascular collateral channels in the neck, arms and over the thorax. Edema of the eyes and face is less pronounced than with acute superior vena caval obstruction.

REFERENCES

1. Mackenzie J. *The Study of the Pulse, Arterial, Venous, and Hepatic, and of the Movements of the Heart.* Edinburgh: Young J. Pentland, 1902.
2. Wood PW. *Diseases of the Heart and Circulation.* Philadelphia, PA: JB Lippincott, 1956.
3. Potain PCE. On the movements and sounds that take place in the jugular veins. *Bull Mem Soc Med Hop(Paris).* 1867;4:3. In: Willius FA, Keys TE, eds. *Classics of Cardiology.* 1983;Vol 2: Malabar, FL: Robert E. Krieger.
4. Hirschfelder AD. Some variations in the form of the venous pulse. *Bull Johns Hopkins Hosp.* 1907;18:265.
5. McGee SR. Physical examination of venous pressure: A critical review. *Am Heart J.* 1998;136:10.

6. Wiggers CJ. *The Pressure Pulses in the Cardiovascular System.* London: Longman, Green, 1928.
7. Rondot E. Le reflux hepato-jugulaire. *Gas Hebd Sc Med Bordeaux.* 1898;19:569.
8. Pasteur W. Note on a new physical sign of tricuspid regurgitation. *Lancet.*1885;2:524.
9. Sochowski RA, Dubbin JD, Naqvi SZ. Clinical and hemodynamic assessment of the hepato-jugular reflux. *Am J Cardiol.* 1990;66:1002.
10. Friedreich N. Zur diagnose der herzbeutelverwachsungen. *Virchows Arch Pathol Anat.* 1864;29:296.
11. Kussmaul A. Uber schwielige mediastino-pericarditis and den paradoxen pulse. *Berl Klin Wochenschr.* 1873;10:433.
12. Gibson AG. The significance of a hitherto undescribed wave in the jugular pulse. *Lancet.* 1907;2:1380.
13. Wenckebach KF. Zur Analyse des unregelmassigen pulses.II Ueber den regelmassig intermitterden Puls. *Zeitschr Klin Med.* 1899;37:475.
14. Stokes W. Observations on some cases of permanently slow pulse. *Dublin Q J Med Sci.* 1846;2:73.
15. Parish JM, Marschke RF, Dines DE, Lee RE. Etiologic considerations in superior vena cava syndrome. *Mayo Clin Proc.* 1981;56:407.

5 The Movements of the Heart—Percussion, Observation, Palpation

Egyptian physicians employed the technique of palpation which was part of the physical examination in ancient Greece and in the Middle Ages (Fig. 5–1). The Ebers papyrus (1550 B.C.) contained a chapter entitled, "Beginning of the Secret of the Physician; Knowledge of the Heart's Movement and Knowledge of the Heart." William Harvey's *De Motu Cordis* (1628) dealt with the motions of the heart imparted to the chest wall:

> In the first place, then, when the chest of a living animal is laid open and the capsule that immediately surrounds the heart is slit up or removed, the organ is seen now to move, now to be at rest.[1]

Harvey made it clear that the movements of the heart could be identified on the intact chest wall:

> The heart is erected, and rises upward to a point, so that at this time it strikes against the breast and the pulse is felt externally.[1]

Laennec in 1819 not only defined the location and size of the apex beat but described movements conveyed to the chest wall by both the left and right ventricles:

> In a healthy person of moderate fullness and whose heart is well proportioned, the pulsation of this organ is only perceived in the cardiac region, that is, in the space comprised between the cartilages of the fifth and seventh ribs, and under the lower end of the sternum. The motions of the left cavities of the heart are chiefly perceptible in the former position, those of the right cavities in the latter. This is so much the case that in disease of one side of the heart only, the pulsation in these two situations gives quite different results. When the sternum is short, the pulsations extend to the epigastrium.[2]

James Mackenzie's *The Study of the Pulse (1902)* contains a chapter on "The Movements of the Heart in Health and in Disease" that represents a landmark in understanding cardiac motion imparted to the thorax:

Figure 5–1 From Augusta's fifteenth century illustrated manuscript, *Regimen Sanitatis (1482)*. The medieval physician is using palpation in the physical examination. (From Castiglioni A. A History of Medicine. New York, Alfred A. Knopf, 1947; reprinted by permission.)

> Practically all important precordial movements transmitted by a beating heart can be palpated and seen by an experienced clinician.[3]

It is exciting to witness the diagnostic conclusions that can be drawn from these observations.

Percussion has a place in the physical examination of the heart and circulation, albeit an abridged place relative to the much older art of palpation. Nevertheless, the modem art of physical diagnosis began with Auengrugger's discovery that fluid levels in wine casks (Fig. 5–2) could be determined by thumping (see Chapter 7). Referring specifically to the heart, Auenbrugger wrote

> Over the space occupied by the heart, the sound loses part of its usual clearness, and becomes dull. The whole sternum yields as distinct a sound as the sides of the chest, except in the cardiac region where it is somewhat duller.[4]

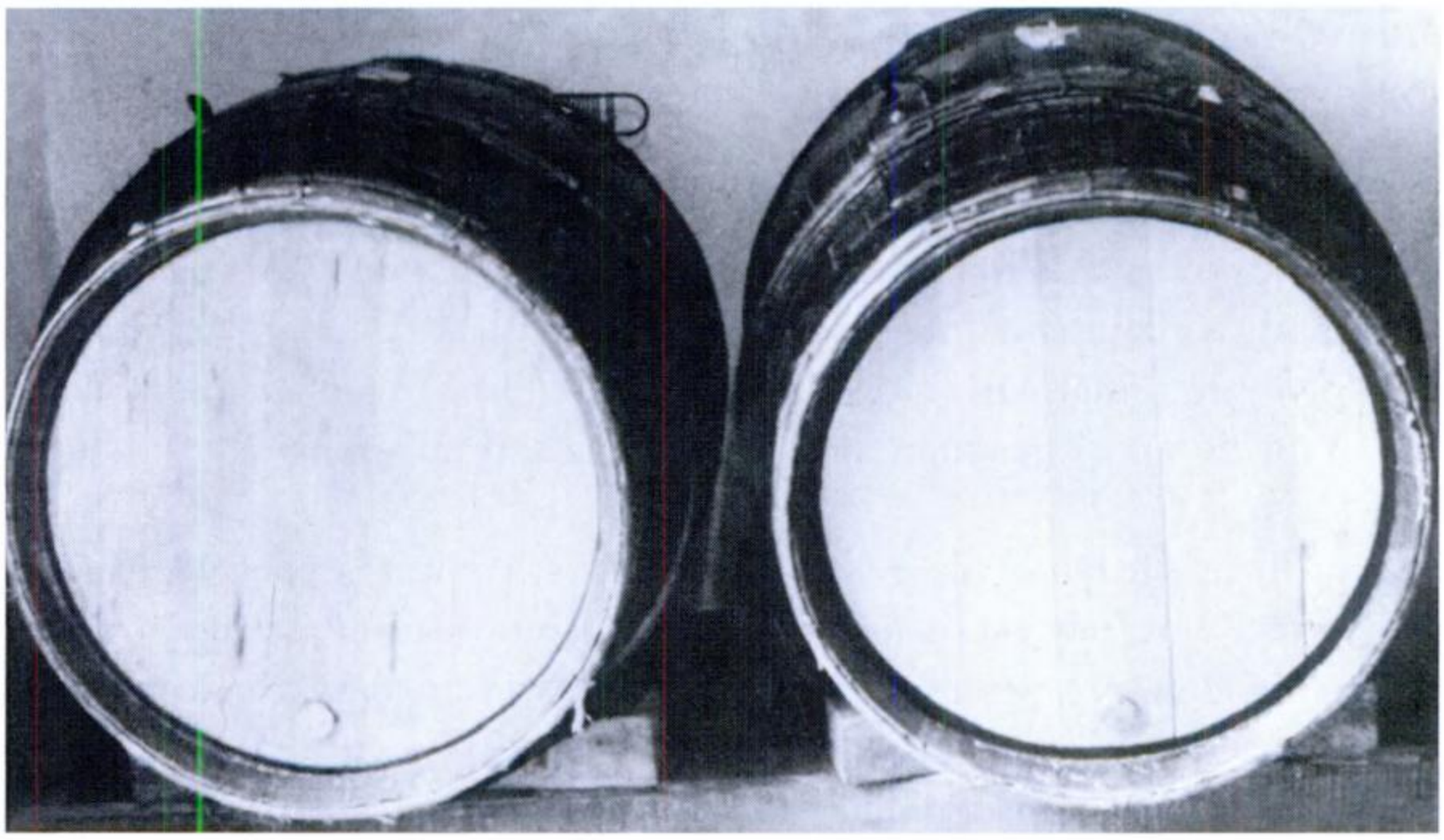


Figure 5–2 Leopold Auenbrugger witnessed his father's practice of percussing wine barrels to determine the level of their fluid contents. The observation led to "a new sign which I have discovered. This consists of the percussion of the human thorax."[4] (Burgundian wine barrels photographed by the author.)

The cardiac examination should begin with percussion, followed by observation and palpation.

PERCUSSION

The thorax ought to be struck, slowly and gently, with the points of the fingers brought close together and at the same time extended.[4]

However, percussion is best achieved using the bony intermediary of the applied finger, which serves to amplify the response. To elicit the percussion note, the entire length of the middle finger of one hand is applied firmly to the site of interest while the dorsum of that digit is struck—percussed—by the tip of the arched middle finger of the free hand using the wrist as the fulcrum (see Chapter 7, Fig. 7–7).

Information derived from percussion falls into two categories: (1) determination of visceral *situs*: heart, stomach, and liver, and (2) less importantly, approximations of the left and right cardiac borders. The location (*situs*) of cardiac and hepatic dullness and gastric tympany must first be established. *Situs solitus* with *levocardia*—the normal position—is established by percussing gastric tympany on the left and hepatic dullness on the right, while percussion to the left and right of the sternum identifies cardiac dullness on the left (*levocardia*). If tympany from the stomach is not clearly evident, the patient should inhale and swallow a few times (aerophagia), a maneuver that generally resolves the problem by making the air-filled stomach tympanitic. An infant swallows air when sucking an empty bottle.

Situs inversus with *dextrocardia* (mirror image) is the reverse of the above, with gastric tympany on the right, hepatic dullness on the left, and cardiac dullness to the right of the sternum (Fig. 5–3). Ninety-five percent of patients with mirror image *dextrocardia* have no coexisting congenital heart disease, but unless *situs inverus* with *dextrocardia* is recognized, symptoms accompanying *acquired* heart disease and accompanying *noncardiac* disease and can be misleading. In *situs inversus* with *dextrocardia*, the pain of angina pectoris is *right* precordial with radiation to the *right* arm. Acute cholecystitis presents with pain in the *left* upper quadrant, and the pain of acute appendicitis is in the *left* lower quadrant.

Except for identifying visceral *situs*, percussion competes poorly with precordial palpation, which with few exceptions, provides all information attained by percussion and much more. However, percussion is sometimes useful in approximating the location of the left cardiac border when the apex is not identified by palpation. In the presence of a large pericardial effusion, cardiac dullness is elicited lateral to the left ventricular impulse. A very large pericardial effusion causes dullness over the sternum and to the right of the sternum. A dilated *right* atrium can cause dullness to the right of the sternum, and a giant *left* atrium can form the right lower cardiac border and cause dullness to percussion.

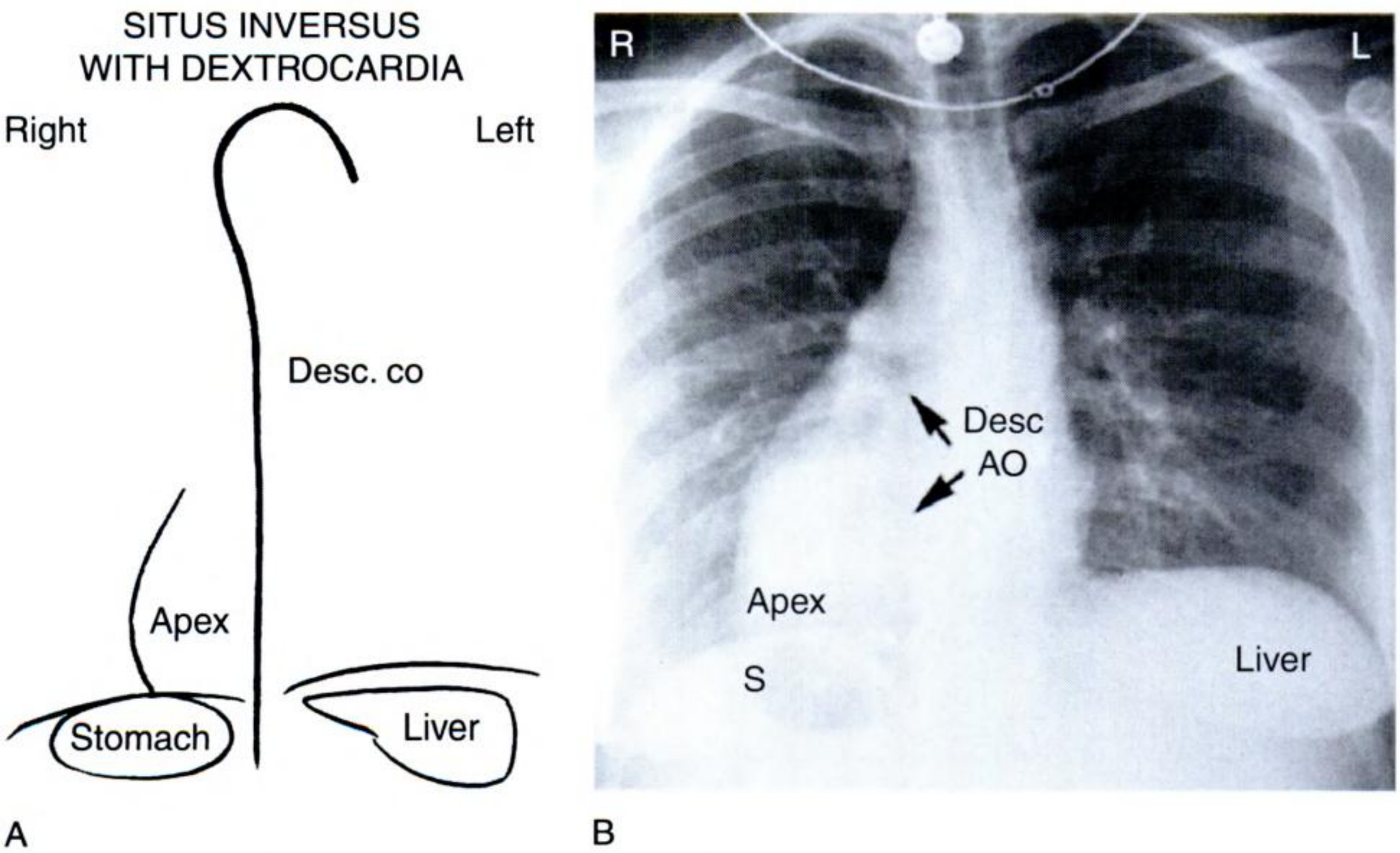


Figure 5–3 *A*, Schematic illustration and *B*, chest radiograph of *dextrocardia in situs inversus*. *Situs inversus* of the abdominal viscera is established at the bedside by percussing the stomach (S) on the right and the liver on the left. *Dextrocardia* is established by percussing cardiac dullness on the right.

OBSERVATION AND PALPATION

Topographic Anatomy of the Heart and the Designation of Precordial Sites

Diagnostic conclusions based on observation and palpation (Table 5–1) require knowledge of the topographic anatomy of the cardiac and vascular structures that impart movements or vibrations to the chest wall. These sites are best referred to by simple descriptive terms.

The chief topographic areas for routine observation and palpation in patients with *levocardia in situs solitus* (normal visceral positions) are the cardiac apex and the sternal borders (left and right; lower, middle, and upper). The examination is then refined by focused palpation of individual parasternal interspaces, the subzyphoid area, and the sternoclavicular junctions.

In the normal heart, the apex is on the left and is occupied by an anatomic *left* ventricle (Figs. 5–4 and 5–5). The anatomic *right* ventricle is anterior and inferior (Figs. 5–4 and 5–5). The inflow or sinus portion of the right ventricle underlies the fourth and fifth left intercostal spaces, the outflow portion (infundibulum) underlies the third left interspace, and the main pulmonary artery (pulmonary trunk) lies beneath the second left interspace (Fig. 5–5). The border of the right atrium is just lateral to the lower right sternal edge, and the ascending aorta is convex to the right of the sternum at the level of the second right interspace (Fig. 5–5). The left atrium is not border-forming because it is a posterior structure except for its appendage which underlies the third left interspace.

Methodology and Technique

Precordial palpation at its inception and for centuries thereafter was performed with the patient standing and facing the physician (Fig. 5–1). However, examination is currently performed with the clinician standing or sitting at the right of the bedside or examining table with the patient's he trunk as close to horizontal as comfort permits. Observation, percussion and palpation begin in the supine position, followed by a partial left lateral decubitus (Fig. 5–6). Infants are, as a rule, examined supine but can sometimes be turned into a partial left lateral decubitus. In adult females, the breasts should be retracted. The patient can assist when the examiner's hands are occupied with palpation and timing (see below).

Table 5–1 Information from Observation and Palpation

Systolic movements of the ventricles
Diastolic movements of the ventricles
Systolic movements of the great arteries
Systolic movements of the atria
Palpable heart sounds
Palpable murmurs (thrills)

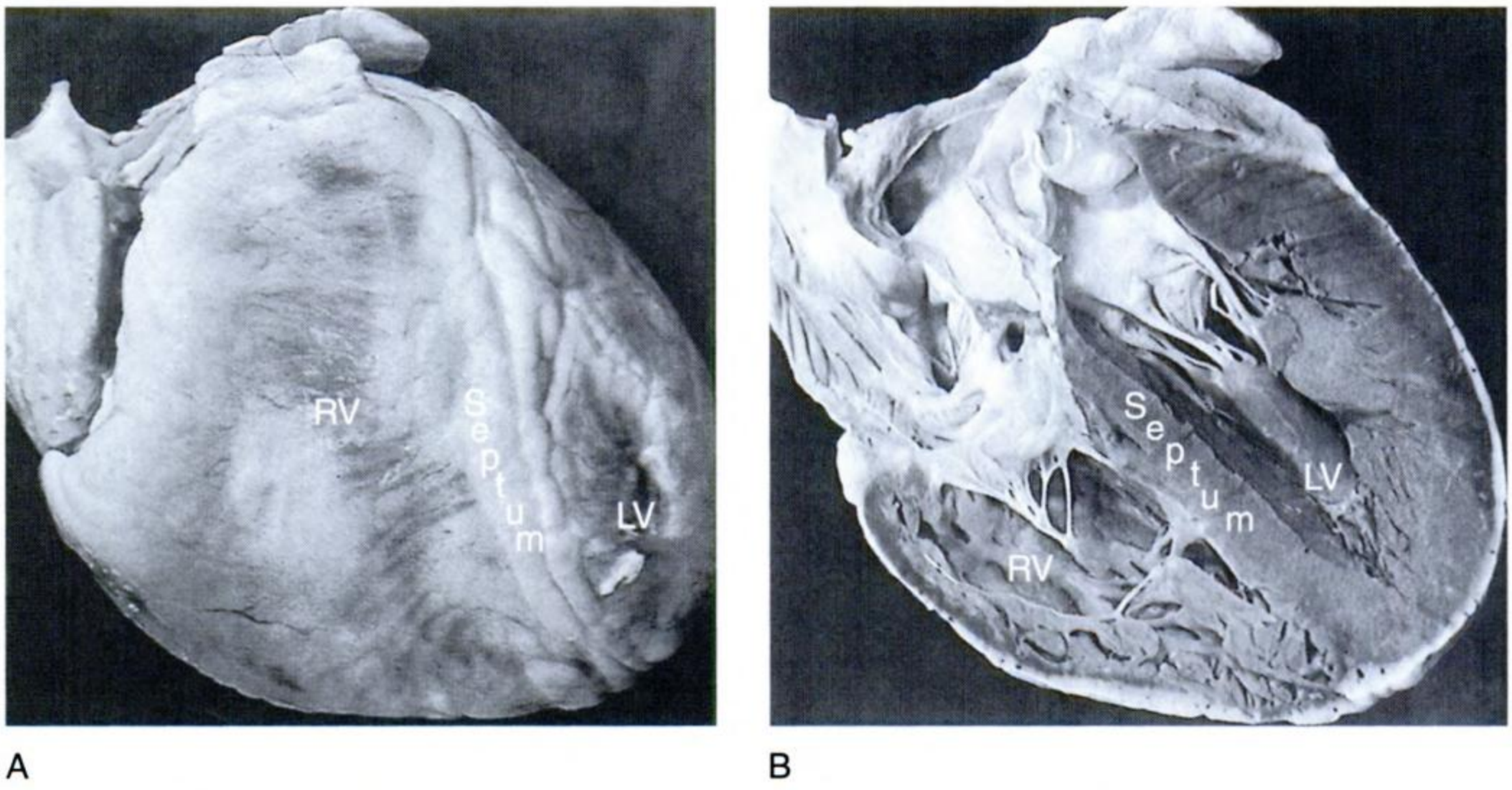


Figure 5–4 *A, In situ* position of the normal heart showing the apical left ventricle (LV) separated from the anterior right ventricle (RV) by the ventricular septum here shown as the linear interventricular sulcus. *B,* Sagittal section of the heart in the same *in situ* position showing the relative positions of the right ventricle (RV) and the left ventricle (LV) separated by the ventricular septum. (From Anderson RF, Becker AE: Cardiac Anatomy. London, Gower Medical Publishing, 1980. Reprinted with permission. Labels are my additions.)

The palmar surfaces of the hand—fingertips, distal meta-carpals, or the "heel" of the hand—and the amount of pressure applied vary according to what is anticipated from palpation and according to the precordial site under consideration. Detection of thrills, ejection sounds, and loud first and second heart sounds benefits from firm pressure of the palmar surface of the distal metacarpals. With this exception, virtually all other information from palpation is best elicited with the pads of the fingertips which detect precordial movements and precisely localize them. The "heel" of the hand applied

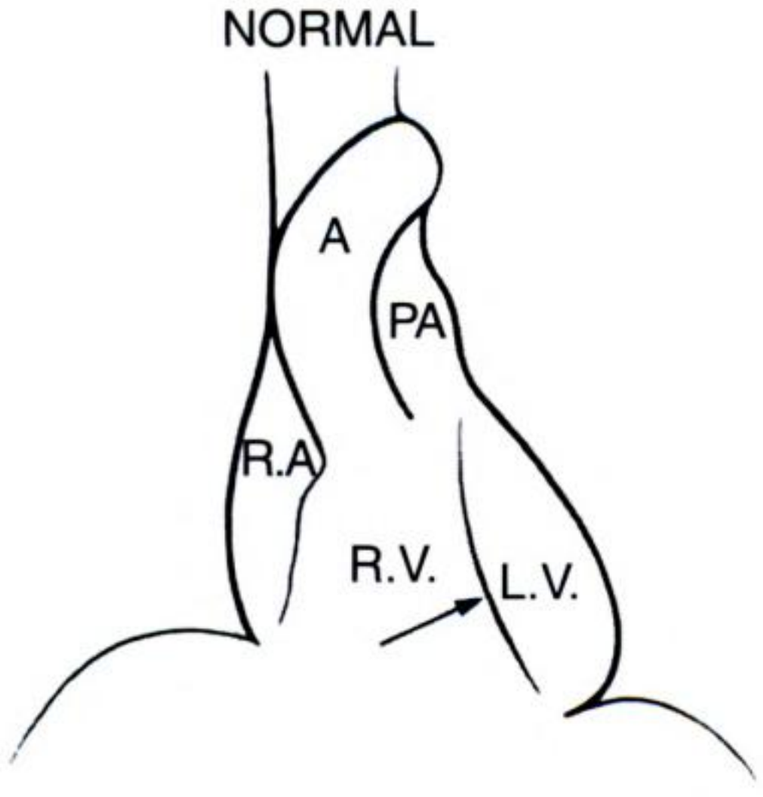


Figure 5–5 Line drawing of the topographic anatomy of the normal heart. *Arrow* points to the interventricular sulcus. The left ventricle (LV) occupies the apex. The body of the right ventricle (RV) is anterior, underlying the left lower sternal border (fourth and fifth interspaces). The infundibulum underlies the left mid-sternal edge (third interspace), and the main pulmonary artery (PA) underlies the left second interspace. The aortic root (A) is convex to the right of the sternum in the second interspace, and the right atrial border is just lateral the right lower sternal edge. Compare with Figure 5–4.

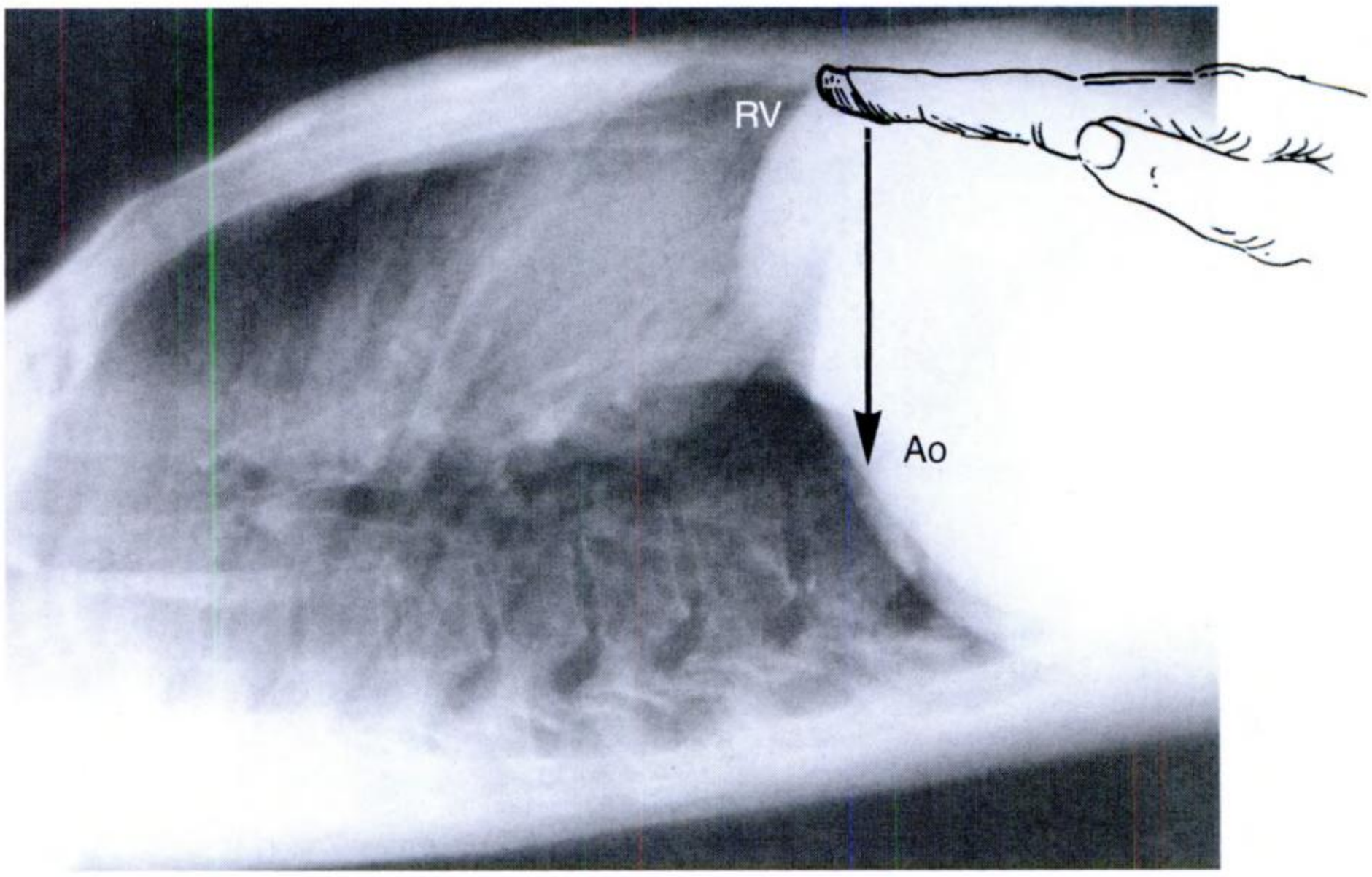


Figure 5–10 Subxiphoid palpation of the inferior wall of the right ventricle (RV) with the tip of the index finger. The relative position of the abdominal aorta (Ao) is shown by the vertical *arrow*.

ventricle at the left sternal edge. Thrills, with few exceptions, are more readily identified during full held exhalation, with the distal metacarpals firmly applied to the area of interest. More precise localization is then achieved by relatively firm application of the fingertips at specific sites.

Before beginning systematic palpation, the precordium should be *observed* for movements that are obvious at a glance. Subtle movements are enhanced by applying pen marks to the skin (see Fig. 5–6), and highlighted with the oblique illumination of a pocket flashlight. The examiner should look *across* the chest *tangentially* rather than from above. Parasternal movements are sometimes best seen when the examiner looks cephalad from the patient's feet. Precordial *retraction* is more readily *seen* than palpated, whereas outward displacement is more readily palpated than seen (Fig 5–11).

Precordial movements can be timed with the carotid pulse (see Fig. 5–12) or with the heart sounds as a reference. In older children and adults, the thumb of the left hand is applied to the carotid pulse while observing or palpating the precordium with the right hand (see Fig. 5–12). Alternatively, the stethoscope can be held in place with the left hand, freeing the right hand for palpation or observation of precordial movements. It is sometimes revealing to observe systolic movements imparted to the stethoscope while monitoring the first and second heart sounds, a point that recalls Laennec's comment on the "shock or impulse" delivered to his wooden stethoscopic cylinder as it was applied to the cardiac apex.

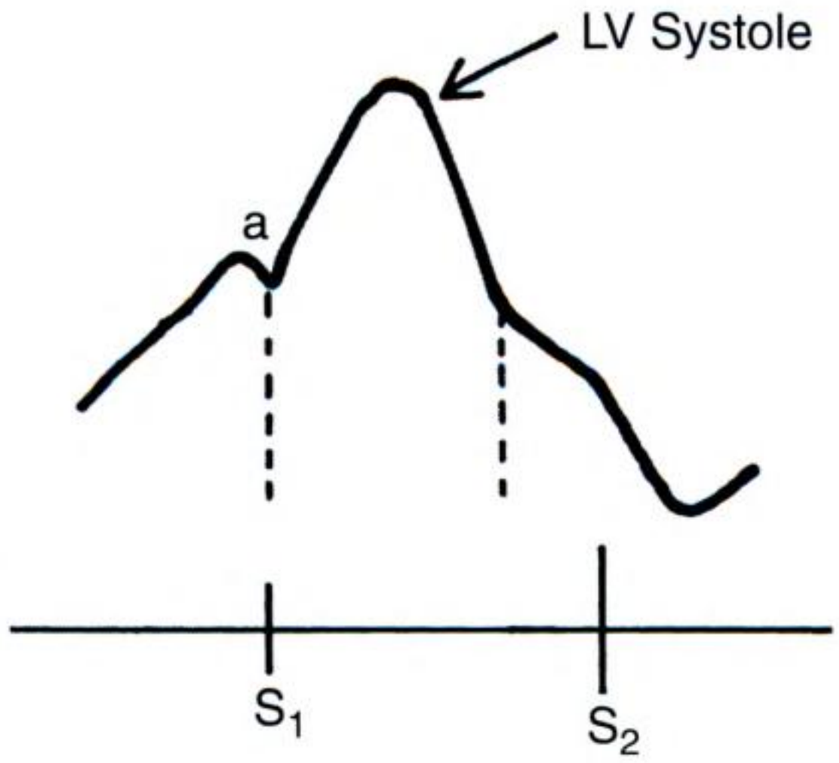
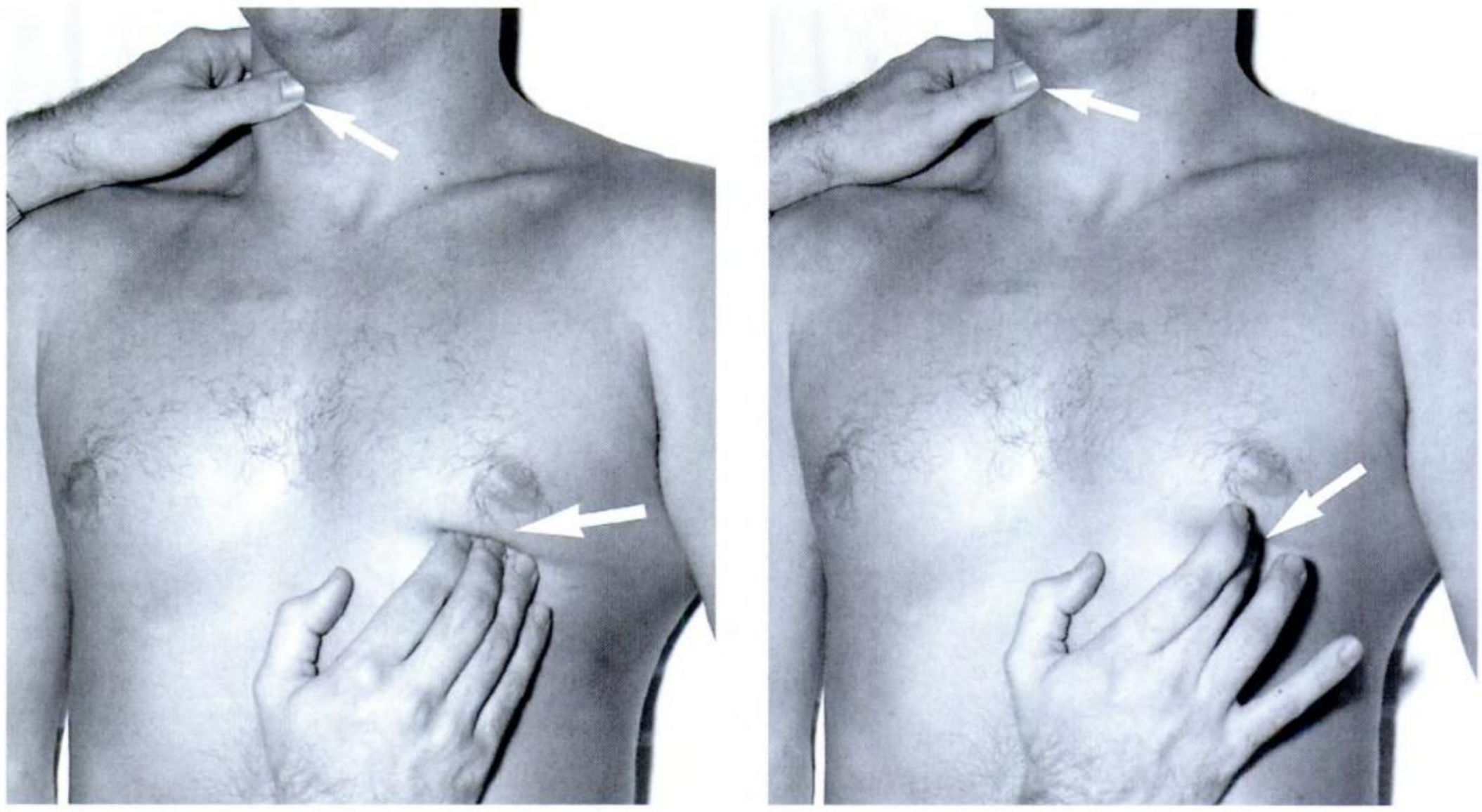


Figure 5–11 Normal apex cardiogram illustrating left ventricular (LV) presystolic filling (a) in response to atrial contraction. The LV systolic impulse returns to baseline before the last third of systole. S_1 = first heart sound, S_2 = second heart sound.

In assessing movements of the heart imparted to the chest wall, it is necessary to determine the topographic location of a given impulse as well as its timing in the cardiac cycle as early, mid , or late systole, or early, mid, or late diastole; its duration (how much of the cardiac cycle the impulse occupies); its displacement characteristics (vigor of movement, amplitude, and contour); and the maximum area occupied by the impulse, especially the left ventricular.



A B

Figure 5–12 *A,* Palpation of the left ventricular impulse by utilizing three fingertips (*right lower arrow*). The examiner's left thumb is applied to the carotid pulse for timing purposes (*upper* arrow). *B,* More refined analysis of left ventricular systolic movement is achieved by palpation with one fingertip (*lower right arrow*). The examiner's left thumb continues to palpate the carotid pulse (*upper arrow*).

Apart from and in addition to the location (*situs*) of the heart, stomach, and liver, the position of the heart and the pulsations transmitted to the chest wall are influenced by body build and thoracic configuration (see Chapter 2). Scoliosis to the right rotates the heart leftward on its long axis; pectus excavatum shifts the heart to the left; a decrease in anteroposterior chest dimensions (loss of thoracic kyphosis, saucer-shaped pectus excavatum) increases contact of the right ventricle and pulmonary trunk with the anterior chest wall. Ascites or pregnancy elevates the diaphragm and the heart, whereas a tall, thin habitus has the opposite effect. Eventration of the left hemidiaphragm (usually in infants) shifts the heart to the right, often dramatically so.

Normal Precordial Movement

Observation and palpation of normal precordial movement assumes an understanding the characteristics of left and right ventricular impulses. Systolic movements imparted by the ventricles should be assigned to the ventricle responsible for the impulse. The imprecise designation "point of maximum impulse" (PMI) should be replaced by a term that reflects the origin of the impulse—left ventricular, right ventricular—or by the topographic location of the impulse (apical, parasternal, etc.).

The normal neonatal right ventricle imparts systolic movement at the mid to lower left sternal edge and subxiphoid. The impulse is brief, gentle, early systolic, followed immediately by retraction that occupies the rest of systole. Systolic movement of the *left* ventricle is, as a rule, not identified even when the infant is turned into a left lateral decubitus position.

Elevation of the baby's trunk in an infant seat improves detection of the subxiphoid right ventricular impulse as the heart descends. In slightly built patients, and in the presence of diminished anteroposterior chest dimensions (loss of thoracic kyphosis, shallow pectus excavatum), a gentle, unsustained right ventricular impulse can be palpated at the mid to lower left sternal edge, but not subxiphoid. The left ventricle outgrows the right during normal development, so in children and adults of average body build, the only normal precordial impulse is left ventricular.

During left ventricular isometric contraction and early ejection (Fig. 5–11), the left ventricle rotates from left to right (clockwise) on its long axis, so a large portion of the chamber moves toward the chest wall. Mackenzie's comment is relevant:

> With the onset of ventricular systole a great change takes place in the position of the left ventricle. The muscle hardens and contracts upon its contents, and at the same time the heart twists round, so that the hardened apex projects forward, pressing against the chest wall.[3]

Interestingly, the physiologic mechanism responsible for the apex beat was long the subject of controversy. William Harvey correctly attributed the apex beat to ventricular systole.[1] Laennec concluded that Harvey was correct because the first heart sound was associated with ventricular systole.[2] However, Josef Skoda believed that the apex beat was a diastolic event, representing recoil after ejection.[5]

Chauveau and Marey resolved the problem by catheterizing the heart and correlating intracardiac pressure changes with the onset of the apex beat that represented contraction of heart muscle against the thorax during ventricular contraction.[6] It has now been proposed that the helical shape of the heart results in twisting that generates ejection followed by untwisting that generates suction and rapid filling in preparation for the next beat.[7]

The normal left ventricular systolic impulse is localized to one interspace, and occupies an area less than 3 cm in diameter in normal adults. The impulse consists of a localized, unsustained anterior apical systolic movement with retraction medial to the interventricular sulcus (Figs. 5–4 through 5–6). Medial retraction occurs because the anterior right ventricle is drawn away from the chest wall during systole. The zone of medial retraction is relatively localized (see Fig. 5–6), and does not extend to the left sternal edge. The horizontal supine position provides a low yield for detecting a normal left ventricular impulse. Accordingly, palpation should begin with the patient in a partial left lateral decubitus position (Fig. 5–6) that consistently permits identification of the left ventricular impulse in virtually all patients in whom that chamber occupies the apex, especially when palpation is performed during held exhalation. Obesity and emphysema are exceptions. The left ventricular impulse should initially be sought with the apposed pads of several fingers (Fig. 5–12*A*), shifting the overlying skin in search of the localized tap which is then palpated with the tips of the fingers (cupping the hand) and finally, and more precisely, with the tip of a single finger, generally the first or second (Fig. 5–12*B*). These maneuvers require just enough time and concentration to attune the examiner and heighten tactile sensitivity. Once anterior ventricular movement is palpated, medial retraction must be established. To achieve this, the palpating fingertip, without losing skin contact, can be shifted medially, a maneuver that results in loss of the impulse because medial retraction is not palpated. The fingertip can then be returned to the palpable impulse while the examiner observes medial retraction by marking the skin (Fig. 5–6). A tangential light source (described earlier) adds refinement to detection of an anterior apical impulse with medial retraction, confirming that the impulse is left ventricular.

When the left ventricular impulse is not palpable in the left lateral decubitus position despite meticulous search, percussion provides an approximate location of the left cardiac border. Palpation at that site might then detect the elusive impulse of the left ventricle.

The *location* of the left ventricular impulse is by convention, related to the midclavicular line, or to the distance from the left sternal edge, and by the interspace in which the impulse is palpable. Location should also take into account patient age and thoracic size and shape. The normal left ventricular impulse in the supine position is generally localized to the fifth intercostal space at or medial to the midclavicular line (Fig. 5–12). In infants and in adults with a short, stocky chest, the impulse is often in the fourth left interspace. In tall, thin patients in whom the heart is in a linear position, proximity of the left ventricular impulse to the left sternal edge invites the mistaken impression of a *right* ventricle impulse. Reexamination in the left lateral decubitus

position avoids this error by permitting identification of the telltale retraction medial to the interventricular sulcus. Although the left lateral decubitus position has the advantage of enhancing palpability of the left ventricle, the *location* may not accurately reflect left ventricular size. If the impulse remains palpable as the patient returns to the supine position, the location can be reassessed.

The left ventricular impulse is difficult to identify in the sitting position. In patients who must be examined while sitting (neuromuscular disease, orthopnea), the impulse can be related to landmarks described above for the supine position.

Mackenzie argued that in assessing displacement of the left ventricular impulse:

> A cardiogram (*graphic record*) is not nearly so instructive as the physical examination of the apex beat. The position, size and strength of the apex beat can be far better appreciated by the palpating hand.[3]

The *duration* of the left ventricular impulse distinguishes normal from abnormal. A single outward movement normally occurs during isometric contraction and early ejection, and returns to baseline before the last third of systole (Fig. 5–11). The three centimeter area normally occupied by the left ventricular impulse remains unchanged, even in the left lateral decubitus position.

Anxiety increases the velocity and amplitude of the left ventricular impulse, but *not* its duration or area. The relative ease with which a normal but hyperkinetic left ventricle is palpated invites the mistaken impression that duration and area are also increased.

Abnormal Systolic Movements of the Left Ventricle

An abnormal left ventricular impulse is characterized by its location (including ectopic), displacement characteristics, and contour. The impulse of an enlarged left ventricle is laterally displaced, and usually resides in the sixth rather than the fifth intercostal space. The sixth interspace at the anterior axillary line is at the same horizontal level as the fifth interspace at the midclavicular line because ribs slant upward. Dilatation of the left ventricle tends to displace the apex *inferiorly* as well as laterally (Fig. 5–13).

An *ectopic* left ventricular impulse (ventricular aneurysm, for example) occupies a site that is inappropriate for a normally positioned (*situs solitus*) heart. An ectopic impulse is most readily recognized when it is *above* and *medial* to the expected location of a normal apical left ventricular impulse. The ectopic segment is often as readily seen as palpated, and is usually caused by regional dyskinesis of ischemic heart disease. When the dyskinetic or aneurysmal segment occupies the apex, the impulse is indistinguishable from a dilated left ventricle.

Prolonged duration of a normally located left ventricular impulse is typical of pressure hypertrophy with increased wall thickness but normal internal dimensions. Laennec wrote:

> The more intense the hypertrophia, the longer the time the impulse is perceptible.[2]

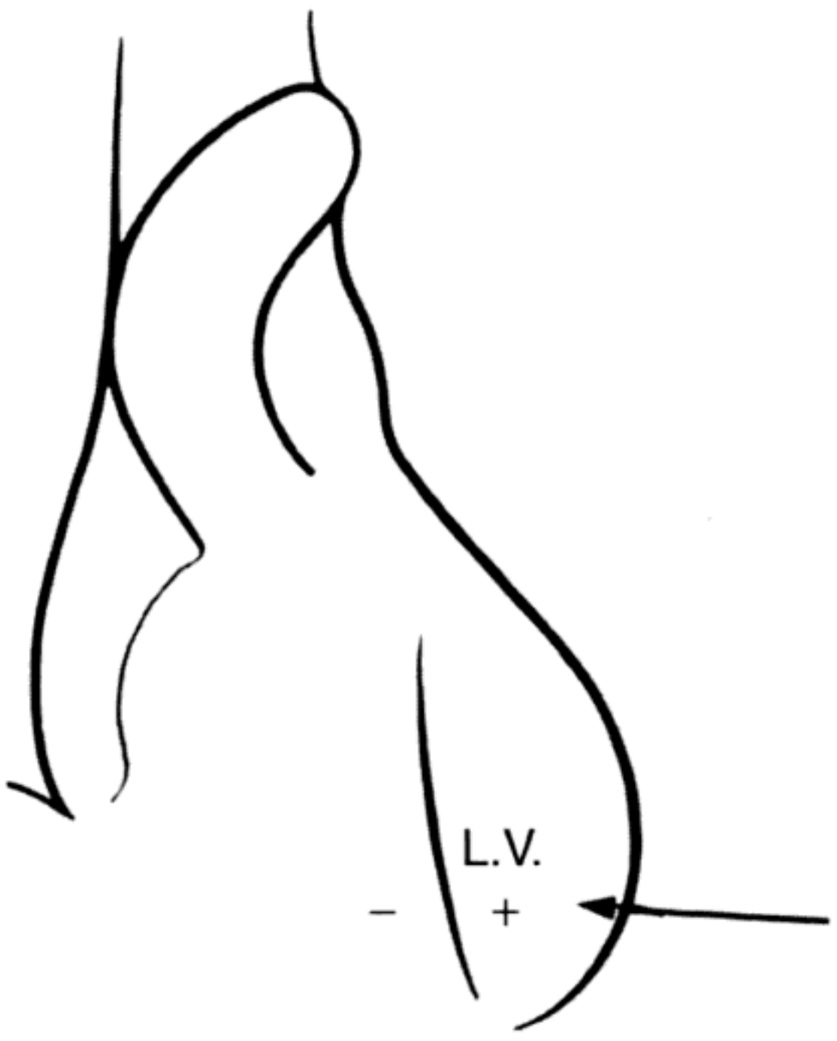


Figure 5–13 Schematic illustration of a left ventricular (LV) impulse that is displaced laterally and inferiorly (arrow). When displacement is due to LV *volume* overload, a hyperkinetic systolic impulse (+) is accompanied by exaggerated medial retraction (–).

A prolonged left ventricular impulse is maintained into the latter third of systole approaching the second heart sound, in contrast to the normal impulse illustrated in Figure 5–11.

A left ventricular impulse can be abnormal in *velocity and amplitude*, ranging from hypokinetic (decreased) to hyperkinetic (increased). A hypokinetic impulse in a patient of normal body build is a sign of decreased contractility, especially when the impulse is laterally displaced. When palpation is too medial, the impulse of a hypokinetic, laterally and inferiorly displaced left ventricle may be missed. Error is prevented by percussing in the supine position that directs attention to the true lateral cardiac border which may extend as far as the posterior axillary line.

Low velocity and low amplitude of systolic motion of the left ventricular impulse are accompanied by low velocity and amplitude of medial retraction, making detection more difficult. An ectopic left ventricular impulse of increased amplitude and duration is a feature of post-myocardial infarction dyskinesis or aneurysm (Fig. 5–14), but may occur transiently during a bout of angina pectoris. The prolonged systolic impulse of regional dyskinesis is really "paradoxic" because it bulges abnormally during mid to late systole (Fig. 5–14) at a time when normal anterior systolic movement has ceased (see Fig. 5–11).

The maximum area occupied by a normal left ventricular systolic impulse in an adult of average body build is less than 3 cm in diameter (see earlier), proportionately less in infants and children. An impulse that exceeds 3 cm in adults is evidence of concentric hypertrophy of systemic hypertension or aortic stenosis, or of the increased end-diastolic volume of a dilated left ventricle. In the absence of concentric hypertrophy, an increase in the maximum area occupied by the impulse is more reliable evidence of left ventricular enlargement than the location of the impulse relative to the midclavicular line.

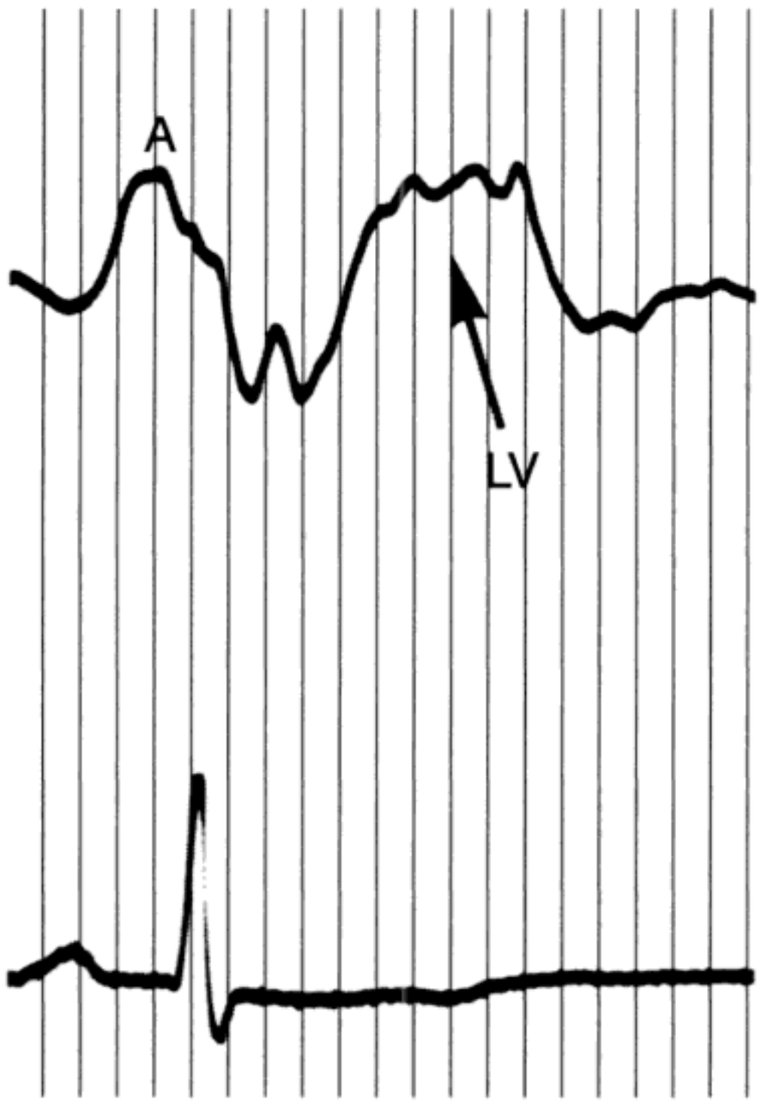


Figure 5–14 Apex cardiogram showing sustained dyskinesis and prolonged duration of the left ventricular (LV) impulse (*arrow*) in a patient with a transmural anterior myocardial infarction. The impulse was preceded by prominent presystolic distention (A). Compare with Figure 5–11.

A left ventricular impulse is, as a rule, single. A *double* systolic impulse is a feature of hypertrophic obstructive cardiomyopathy (Fig. 5–15). Identification of this contour requires precise timing with the carotid pulse (see Fig. 5–12*A*) or with the heart sounds.

When the left ventricle occupies the apex but the apex *retracts* in systole, the cause is usually chronic constrictive pericarditis.[8] Retraction includes the ribs and intercostal spaces, and is usually more pronounced at the apex but may involve most of the left anterior half of the chest.

Abnormal Systolic Movements of the Right Ventricle

Laennec remarked on an abnormal right ventricular impulse when he applied his stethoscopic cylinder to the precordium:

> The contractions of the heart, as explored by the cylinder, gave the same results nearly, whether the hypertrophia be on the right or the left side; only in the former case, the shock of the heart's action is greater at the bottom of the sternum.[2]

James Mackenzie recorded the epigastric impulse of the right ventricle, and distinguished it from the impulse of the abdominal aorta. He wrote (Fig. 5–10):

> At the postmortem examination, a needle pushed through the epigastrium at the place where the tracing was obtained, was found to have penetrated the right ventricle. The epigastric pulsation due to the abdominal aorta presents quite a different character from that due to a dilated right ventricle.[3]

For assessment of right ventricular systolic movement, the examiner stands or sits to the right of the patient who should lie supine with the trunk horizontal or elevated

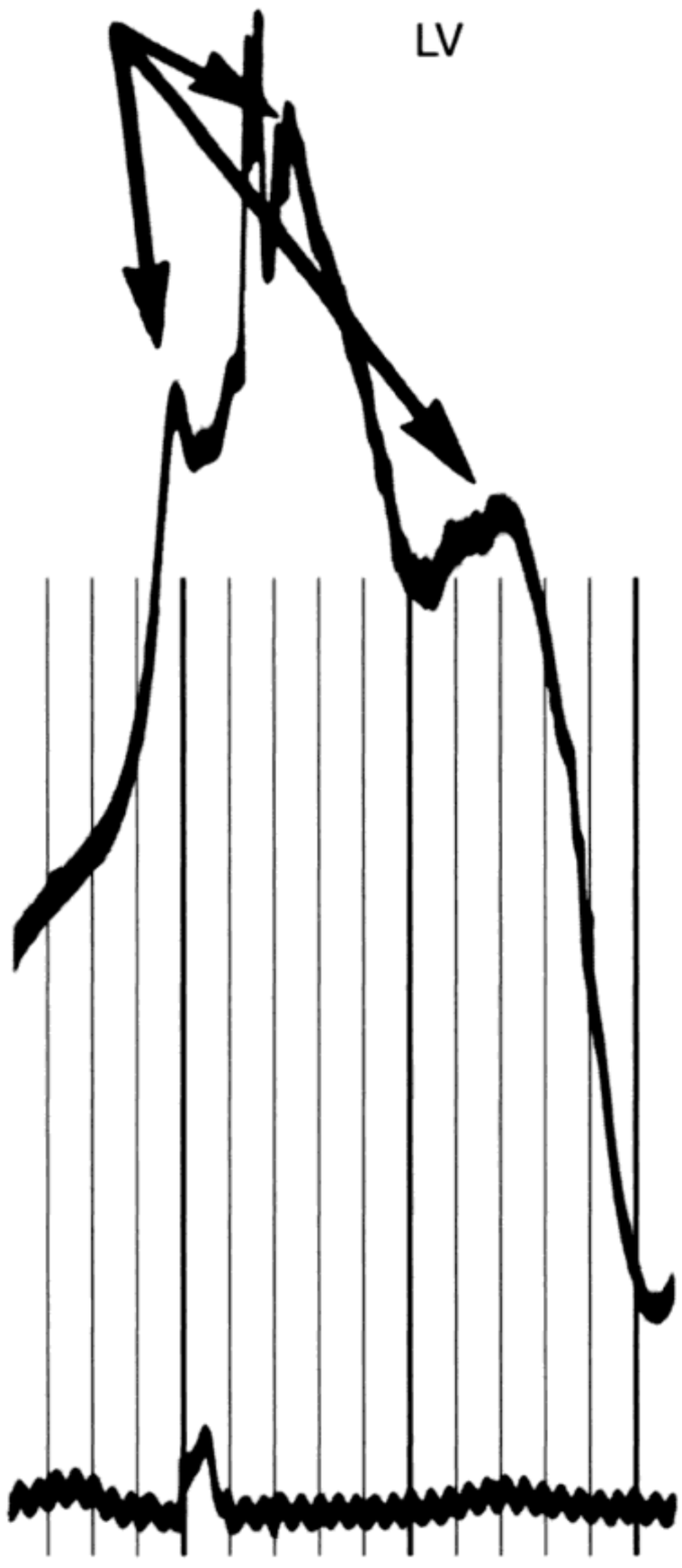


Figure 5–15 Apex cardiogram from a patient with hypertrophic obstructive cardiomyopathy. A distinctive left ventricular (LV) triple impulse is caused by presystolic and double systolic movements (*arrows*).

30 degrees. In *situs solitus*, the anatomic right ventricle is anterior and inferior,[3] and topographically behind the left sternal edge (Fig. 5–4). Its inflow portion underlies the mid to lower left sternal edge (fourth and fifth intercostal spaces), while its outflow portion (infundibulum) lies behind the third interspace. Accordingly, the area potentially in contact with the chest wall is greater than the chest wall contact of the left ventricle. The inferior portion of the right ventricle (Fig. 5–4) transmits its impulse subxiphoid, which is accessible to palpation (Figs. 5–9 and 5–10). Subxiphoid palpation should be carried out during held *inspiration*, whereas palpation of the right ventricle at the left sternal edge should be carried out during held *exhalation*.

Age and thoracic configuration significantly influence access to the right ventricle. In normal infants, right ventricular contraction generates the precordial impulse described earlier. In older children and in adults with a slight body build or decreased anteroposterior chest dimensions (loss of thoracic kyphosis, shallow pectus excavatum), contact of the right ventricle with the anterior chest wall is increased, producing a brief, gentle left parasternal impulse. Conversely, an *increase* in anteroposterior chest dimensions (barrel chest of pulmonary emphysema, see Chapter 7) reduces or abolishes contact of the right ventricle with the anterior chest wall, so its impulse may not be palpable

even when the chamber is enlarged. Subxiphoid palpation (Figs. 5–9*A* and 5–10) is an important alternative technique especially when the hyperinflation of emphysema lowers the diaphragm and with it the inferior wall of the right ventricle.

Observation plays an important but lesser role than palpation in assessing right ventricular systolic impulses, while percussion is of little or no value. Two methods of palpation are recommended, as already briefly described. The heel of the hand (distal metacarpals) can be applied to the left sternal edge during full exhalation, keeping the fingers elevated while sensing the systolic impulse and observing the motion of the hand imparted by right ventricular contraction. This technique is relatively insensitive for detecting subtle systolic movement and does not localize the impulse. A more refined method employs several fingertips applied simultaneously and in parallel to the third, fourth, and fifth intercostal spaces during held exhalation (see Fig. 5–7). The left hand is free for timing with the right carotid pulse, or for applying the stethoscope to the chest for timing with the first and second heart sounds. This method not only permits detection of a gentle right ventricular systolic impulse but localizes the impulse to the inflow portion (fourth and fifth intercostal spaces) or to the infundibulum or outflow tract (third interspace). In infants, palpation along the left sternal border is best accomplished with the tip of a single finger during active breathing (see Fig. 5–8) or during very brief arrested respiration induced by pinching the nostrils while the infant sucks a nipple or pacifier.

Abnormal anterior movement of the right ventricle during systole is accompanied by retraction in the region of the interventricular sulcus (Fig. 5–4). An *appreciably* enlarged right ventricle displaces the left ventricle from the apex, no longer makes contact with the chest wall, and imparts no precordial impulse (Fig. 5–16). Right ventricular anterior systolic movement then extends from the left sternal edge to the apex which retracts (Figs. 5–16 and 5–17). Mackenzie recalled these points in admirable detail:

> Accepting the usual clinical definition of the apex beat being the lowest and outermost part of the heart's impulse, a totally different form of beat is found when the right ventricle causes this movement. In certain cases of dilatation of the right

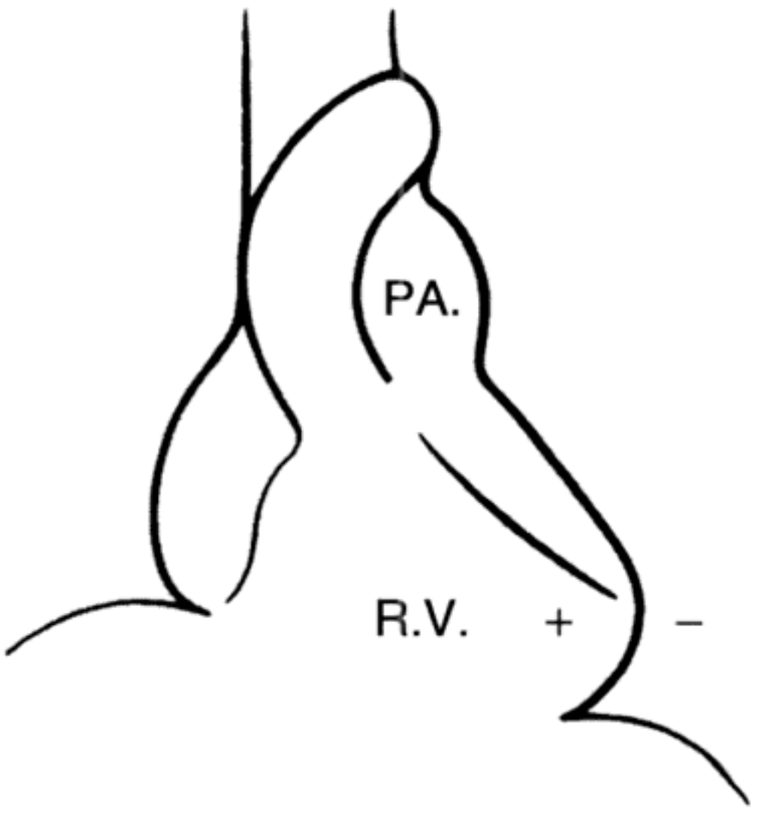


Figure 5–16 Schematic illustration of an enlarged right ventricle (RV) that displaces the left ventricle from the apex. The positive (+) right ventricular systolic impulse extends to the apex, which retracts (–). A dilated main pulmonary artery (PA) underlies the left second interspace.

heart, nearly the whole anterior aspect of the heart is composed of the right auricle and ventricle, the left ventricle forming a mere strip of the lateral border. This portion of the left ventricle is situated so far back that it is covered by the lungs and does not reach the chest wall. Therefore, the lowest and outermost part of the heart in contact with the chest wall is the right ventricle. In place of the outward thrust during systole, as in the apex beat due to the left ventricle, there is drawing in of the tissues.[3]

Systolic anterior movement of the right ventricle with lateral retraction can be highlighted by marking the chest wall, as shown in Figure 5–17. *Observation* is important in confirming that the right ventricle occupies the apex, because retraction is better seen than palpated.

Characterization of a right ventricular systolic impulse not only includes timing and location as described above, but also contour, velocity, amplitude, and duration of movement. Persistence of the brief, gentle, normal neonatal right ventricular impulse is a feature of Fallot's tetralogy in which the right ventricle continues to function as a systemic chamber as it does in the fetus and newborn. It is patient age, rather than displacement characteristics that makes the right ventricular impulse abnormal. In Fallot's tetralogy the impulse is confined to the fourth and fifth intercostal spaces, and is not present in the third interspace because infundibular pulmonary stenosis assigns the elevated right ventricular systolic pressure to the body or inflow portion of the right ventricle. A right ventricular impulse that extends to the *third* interspace means that systolic pressure is elevated in both the inflow and outflow portions, as in pulmonary valve stenosis or pulmonary hypertension. Marked to severe elevation of right ventricular systolic pressure with intact ventricular septum is accompanied by an impulse that is increased in both amplitude *and* duration, extends from the fifth to the third left intercostal space, and includes the subxiphoid area.

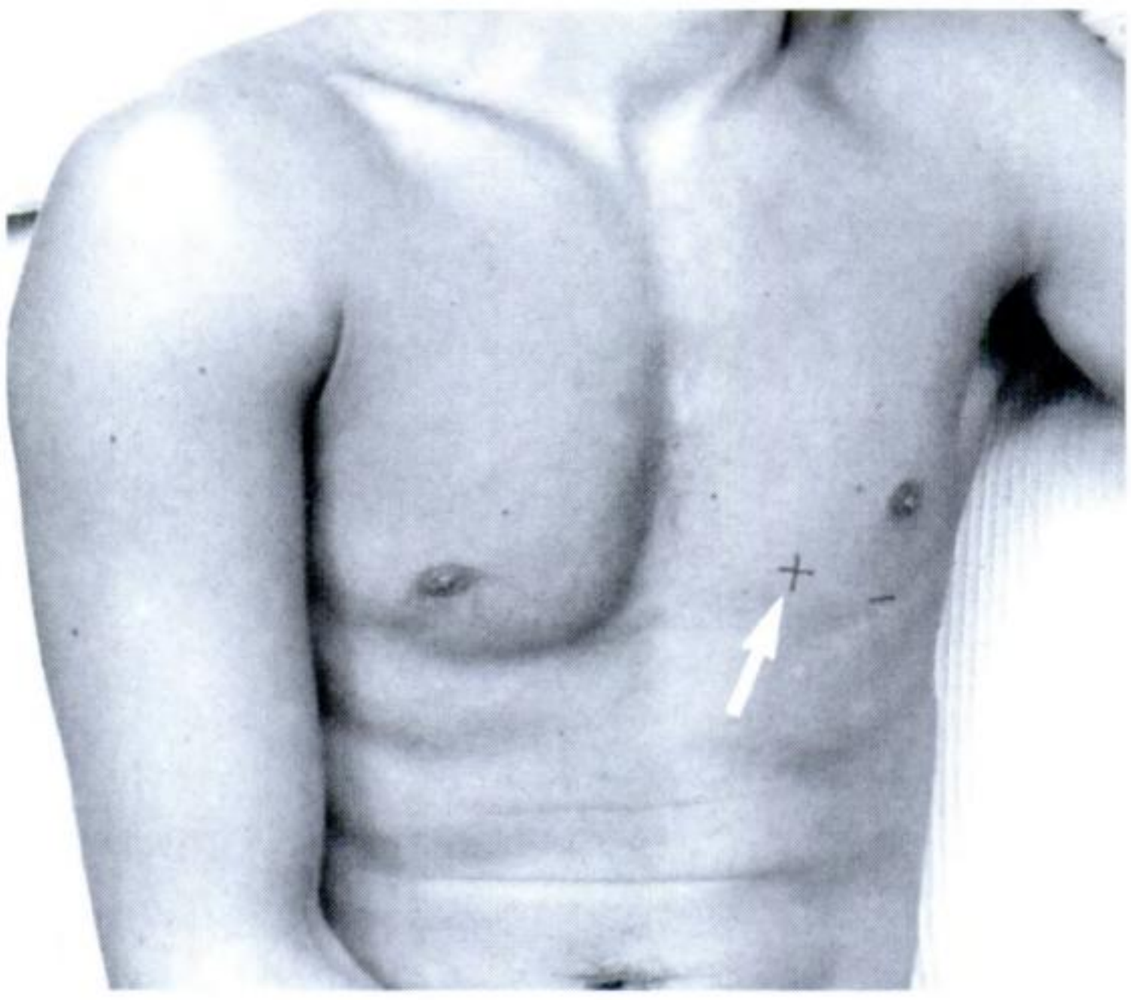


Figure 5–17 The medial mark (+ *arrow*) is over the right ventricle which moves anteriorly during systole, while the apex retracts (lateral mark –). These movements characterize a right ventricle that occupies the apex, displacing the left ventricle and interventricular sulcus.

Volume overload of mild to moderate tricuspid regurgitation generates a right ventricular systolic impulse characterized by a modest increase in velocity and amplitude, but no increase in duration. At the other end of the spectrum is the tumultuous right ventricular impulse accompanying the large left-to-right shunt of an uncomplicated ostium secundurn atrial septal defect. Although these left parasternal and subxiphoid impulses can be dramatic, and apical retraction exaggerated, the duration of the impulses is increased little if at all.

An abnormal right ventricular systolic impulse must be distinguished from movement imparted to an otherwise normal right ventricle when the chamber is displaced anteriorly during left ventricular systole. The most common cause is mitral regurgitation (Fig. 5–18). The left atrium is a posterior chamber, lying behind the heart and anterior to the rigid vertebral column. An enlarged left atrium moves the heart forward, so an otherwise normal right ventricle comes into contact with the anterior chest wall. Mitral regurgitation causes a phasic systolic increase in volume of the enlarged large left atrium, resulting in phasic systolic anterior movement (Fig. 5–18). The systolic impulse is necessarily delayed because the left ventricle must first contract in order to provide the regurgitant volume that expands the left atrium and moves the heart forward. Late systolic movement of the impulse is best timed by relating it to the first heart sound, but timing is also possible by simultaneously palpating the left ventricle while observing or palpating the parasternal systolic movement.

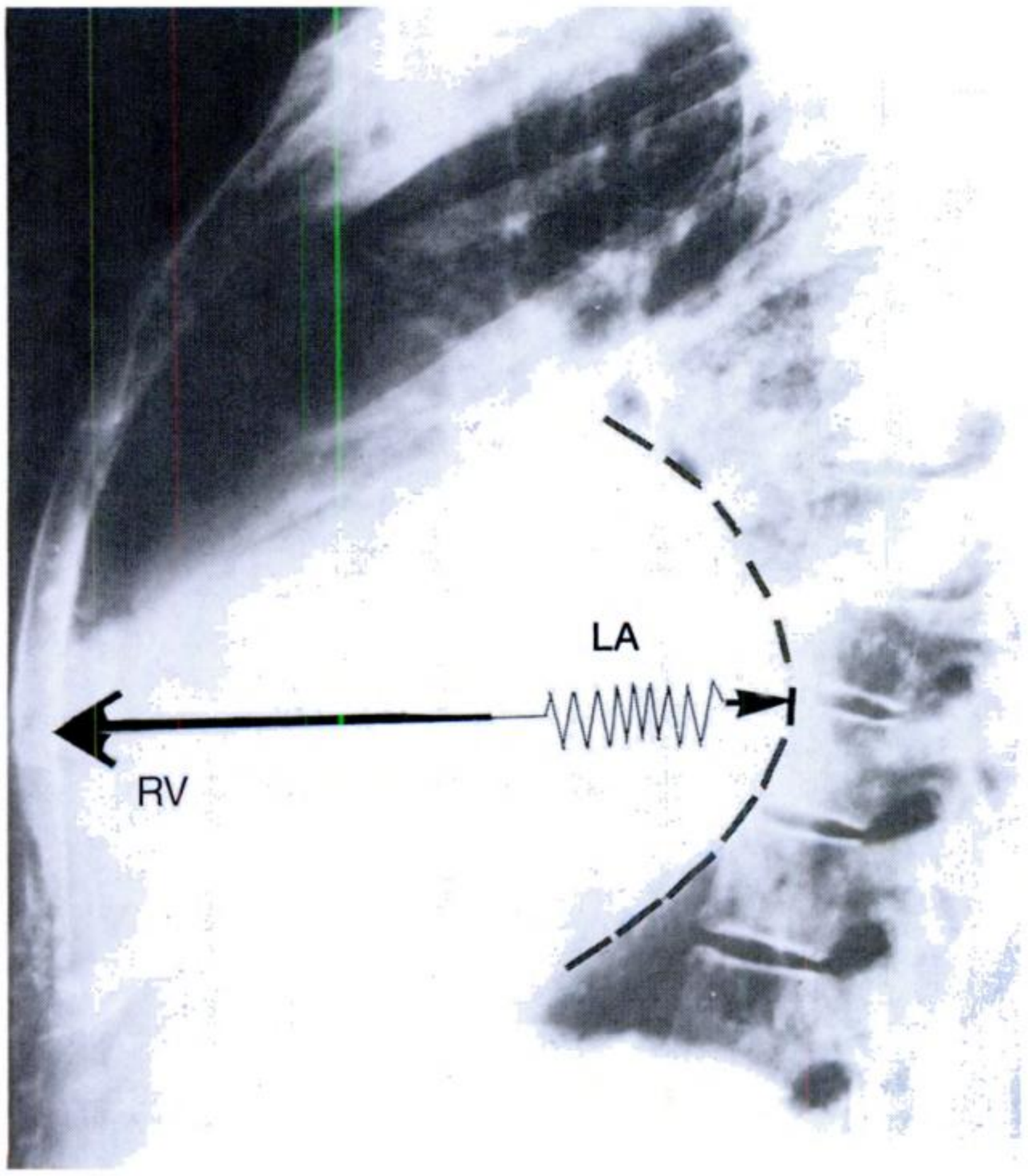


Figure 5–18 Lateral chest radiograph in a patient with severe mitral regurgitation and a large left atrium (LA) that displaces the right ventricle (RV) anteriorly, increasing its contact with the chest wall. Systolic expansion of the left atrium during regurgitant flow moves the heart forward with each left ventricular contraction (large arrow), rendering the right ventricle palpable.

Anterior systolic movement of an inherently normal right ventricle can be caused by regional wall motion abnormalities of the *left* ventricle. Dyskinetic motion of the ventricular septum during angina pectoris displaces the right ventricle and results in a transient left parasternal impulse that promptly disappears with relief of angina. Post-myocardial infarction dyskinesis of the ventricular septum is analogous, but results in persistent systolic anterior displacement of the right ventricle. In mitral stenosis, a large left atrium displaces the right ventricle anteriorly and augments the pulsations of that chamber associated with pulmonary hypertension.

Biventricular Systolic Movements

A pure *left* ventricular impulse causes apical systolic anterior movement with medial retraction (see Fig. 5–6). A pure *right* ventricular systolic impulse causes parasternal anterior movement with lateral retraction (Figs. 5–16 and 5–17). When *both* ventricles impart systolic movements to the precordium, the apical left ventricular impulse and the parasternal right ventricular impulse coexist but are separated by a zone of retraction that represents the plane of the interventricular sulcus (see Figs. 5–4 and 5–19).

Mid-Diastolic Movements of Ventricles

Passive filling of the ventricles occurs as atrial pressures rise sufficiently to open the mitral and tricuspid valves. Abnormally rapid atrioventricular flow and/or abnormal diastolic properties of the recipient ventricle impart a brief, distinct mid-diastolic movement that can be palpated and sometimes seen (Fig. 5–20). The auscultatory counterpart is an

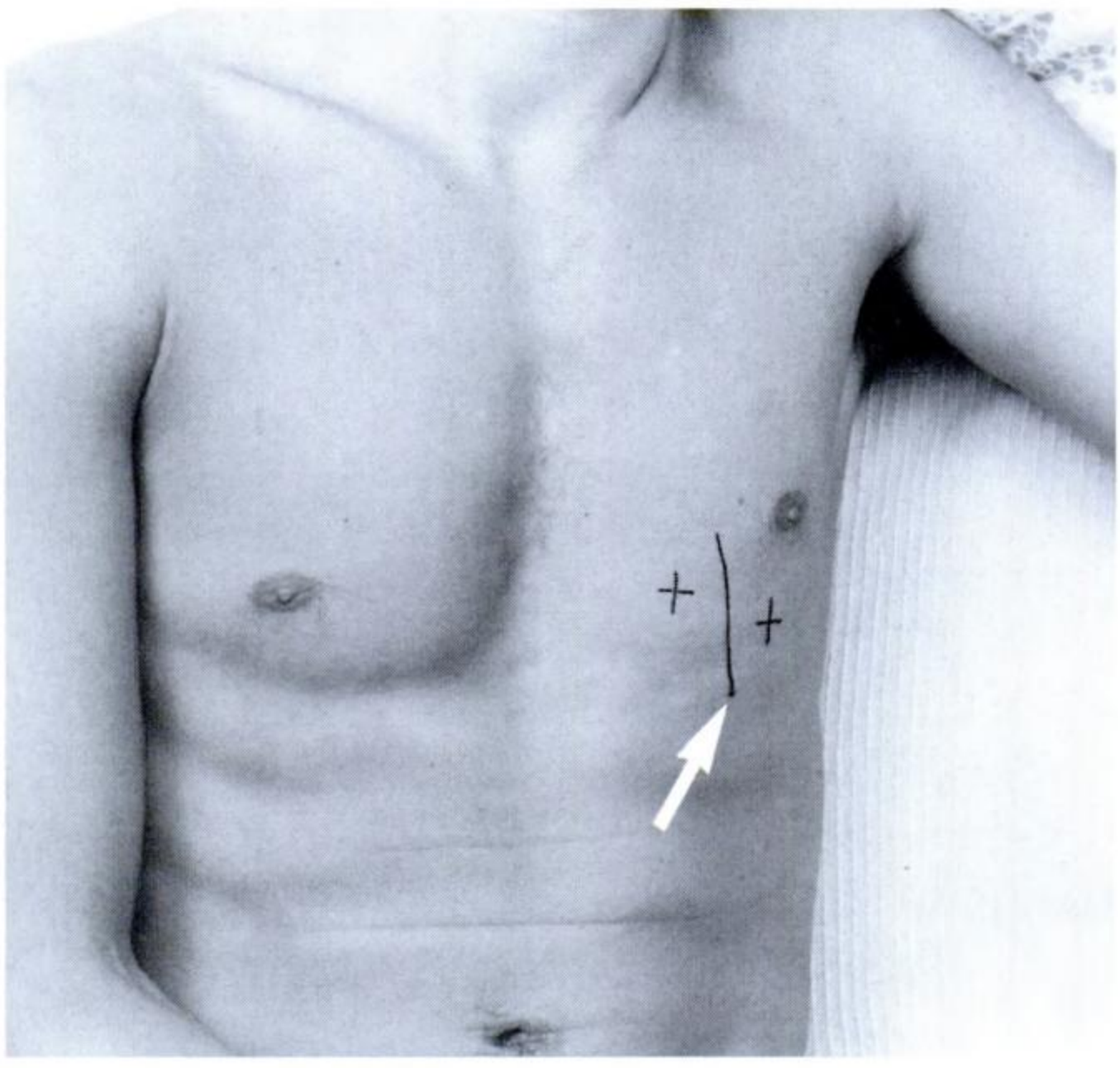


Figure 5–19 Biventricular systolic movements of the ventricles with anterior displacement (+) of both right *and* left ventricles separated by a zone of retraction along the interventricular sulcus (*arrow*).

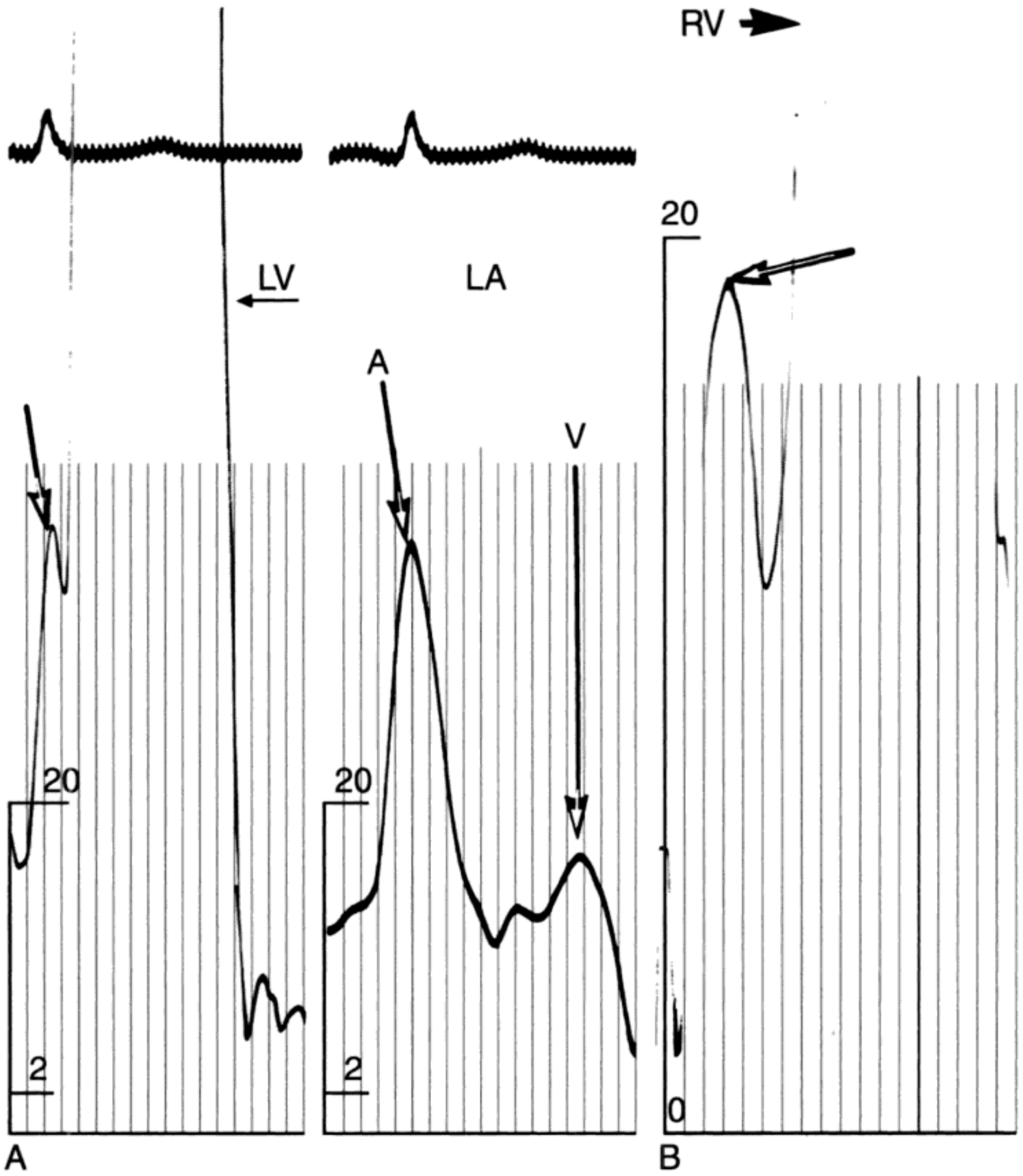


Figure 5–22 *A,* Severe aortic valve stenosis with striking presystolic distention (*arrow*) of the *left* ventricle (LV) in response to powerful left atrial (LA) contraction that generates a large A wave. The V wave is small. *B,* Primary pulmonary hypertension with striking presystolic distention (arrow) of the *right* ventricle (RV).

movement—is low frequency and is damped by firm pressure. The tactile sensation imparted to the fingertip varies from subtle, brief, and indistinct to an obvious impact immediately preceding the left ventricular systolic impulse (Fig. 5–22*A*). The auscultatory counterpart is the fourth heart sound (see Chapter 6); presystolic distention is often more readily palpated than a fourth sound is heard. In 1876, Potain called attention to this point:

> If one applies the ear to the chest, it affects the tactile sensation more perhaps than the auditory sense.[9]

A subtle presystolic impulse can be augmented when the left ventricle is transiently stressed with isometric exercise (sustained hand grip) (see Chapter 6). An X mark on the skin over the left ventricular impulse improves visualization of pre-systolic movement immediately before the major systolic impulse of the left ventricle.

Right ventricular presystolic distention is best identified in the *supine* position with the trunk horizontal or elevated 30 degrees. The fingertips are applied in the fourth and fifth left intercostal spaces during full, held exhalation (see Fig. 5–7). It is sometimes better to palpate each interspace separately, especially in infants (see Fig. 5–8). *Subxiphoid* palpation during held *inspiration* may be even more informative, because a right ventricular presystolic impulse tends to increase during inspiration.

It comes as no surprise that mid-diastolic *and* presystolic distention of the right or left ventricle may coexist (see Fig. 5–20). Identification of these dual impulses is more readily achieved when the cardiac rate is slow. A rapid rate, especially if the PR interval is long, results in summation of the two ventricular filling phases and a single, reinforced diastolic impulse.

Movements of the Atria during Ventricular Systole

Systolic expansion of a dilated left or right atrium in the presence of mitral or tricuspid regurgitation imparts distinctive movements to the chest wall. Anterior movement caused by systolic expansion of an enlarged left atrium was dealt with earlier (Fig. 5–18). A *giant* left atrium may extend into the right hemithorax. Mitral regurgitation then causes late systolic movement of the lower *right* anterior chest, occasionally as far as the right anterior axillary line. When mitral regurgitation occurs in the presence of an enlarged left atrial *appendage*, an impulse can be palpated and often seen in the third left intercostal space where the appendage is border-forming.

The right atrium normally forms the right lower cardiac border. The right lobe of the liver is just beneath the anterolateral rib cage. In the presence of severe chronic tricuspid regurgitation, the right atrium and liver enlarge appreciably, making broad contact with the anterior and lateral chest walls. Systolic expansion of the large right atrium together with systolic movement of a large right hepatic lobe cause dramatic late systolic movement of the entire right lower chest. The right ventricle, even though volume-loaded, causes relatively less impressive anterior movement, so the chest, especially when viewed from the patient's feet, exhibits a striking rocking motion with each ventricular systole. When tricuspid *stenosis* occurs with a large, powerfully contracting right atrium, a *presystolic* impulse is transmitted to the lower right anterior chest.

Movements of the Great Arteries

The ascending aorta is normally border-forming at the right thoracic inlet, but is palpable only when abnormal in size or position. An ascending aortic aneurysm in the Marfan syndrome, is an example, causing a systolic impulse in the second, or first and second right intercostal spaces near the sternum (Fig. 5–23). A saccular luetic aneurysm of the ascending aorta can present similarly or can burrow through the chest wall and present as a dramatic, visible pulsatile mass (Fig. 5–24). If an ascending aortic aneurysm is sufficiently cephalad as with a dissecting aneurysm, systolic movement can be transmitted to the *right sternoclavicular joint*. Stemoclavicular joint pulsations are seldom visible, are generally subtle, and are best sensed by relatively firm pressure with two apposed fingertips

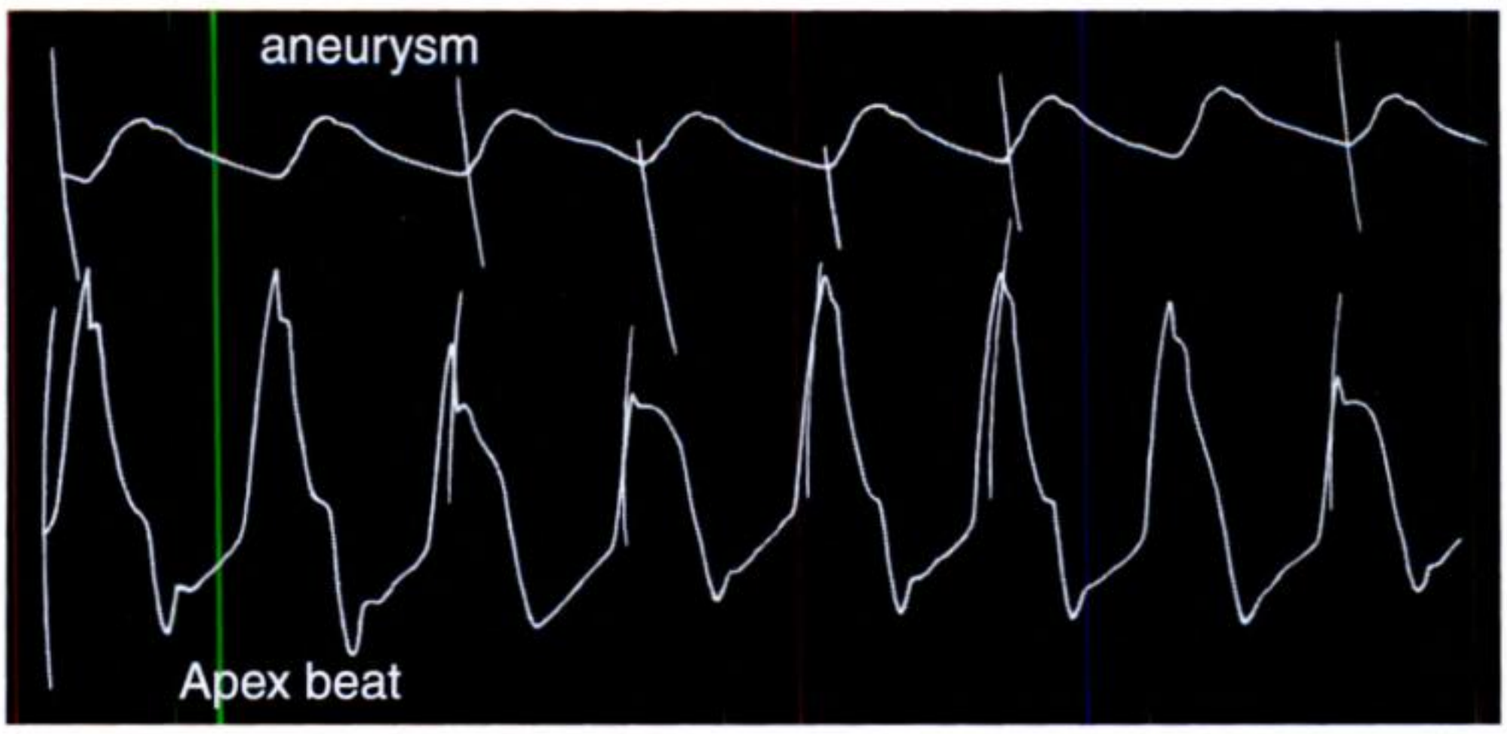


Figure 5–23 "Tracings from a large aneurysm pulsating in the second right intercostal space and from the apex beat."[3] (see Fig. 5–24). (From Mackenzie J: The Study of the Pulse, Arterial, Venous, and Hepatic, and of the Movements of the Heart. Edinburgh, Young J. Pentland, 1902.)

which fix the joint. Systolic movement of the right sternoclavicular joint is also a sign, albeit subtle, of an otherwise normal right aortic arch. When a right aortic arch is dilated as in Fallot's tetralogy with pulmonary atresia (Fig. 5–25), motion is readily imparted to the right sternoclavicular joint A systolic impulse imparted to the *left* sternoclavicular joint occasionally results from distal extension of the transverse portion of a dissecting

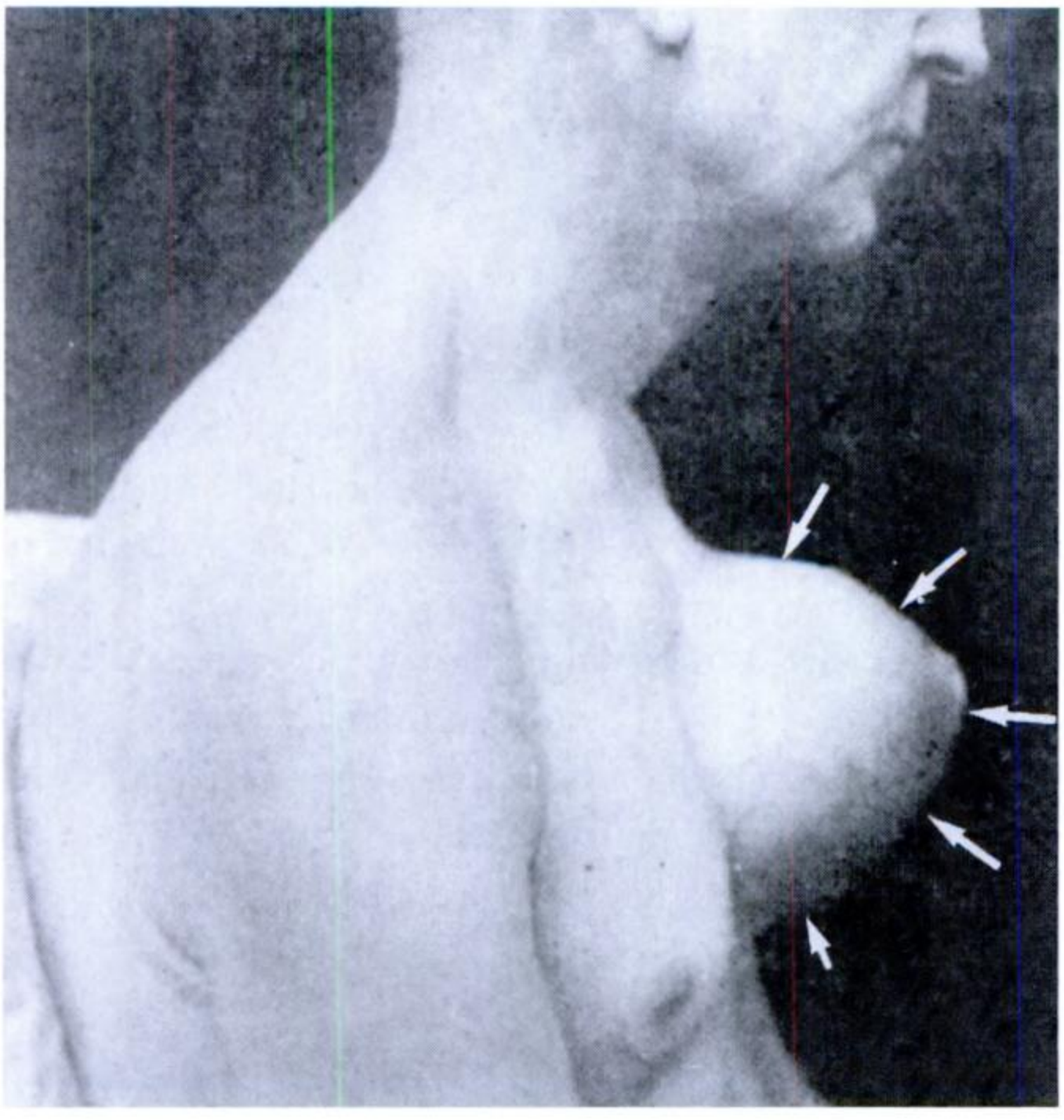


Figure 5–24 Saccular ascending luetic aortic aneurysm that burrowed through the chest wall and presented as a dramatic, visible pulsating mass *(arrows)*. (From Cabot RC: Physical Diagnosis. New York, William Wood and Company, 1915. Courtesy of Dr Sherman M. Mellinkoff, UCLA Medical Center.)

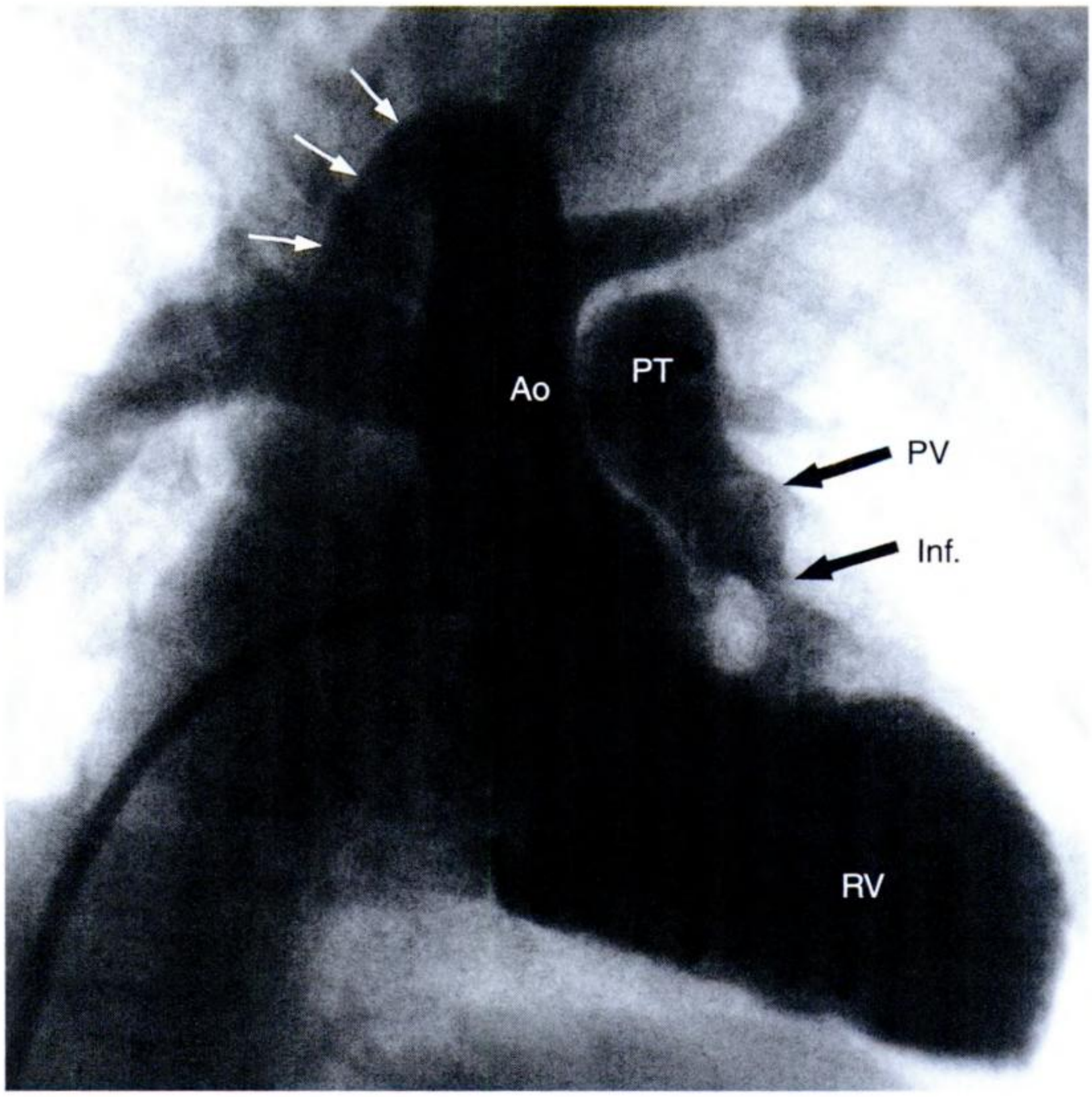


Figure 5–25 Right ventriculogram from a 15 month-old male with Fallot's tetralogy. Proximity of the right aortic arch (*three white arrows*) to the overlying right sterno-clavicular junction caused a systolic impulse. The ascending aorta (Ao) was dilated. PT = pulmonary trunk, PV = pulmonary valve, Inf = infundibulum, RV = right ventricle.

aortic aneurysm. Rarely, late systolic anterior movement of the sternum and contiguous left parasternal area are caused by a large, pulsatile *descending* thoracic aortic aneurysm as it physically moves the heart forward during ventricular systole. If this is suspected, *synchronous* late systolic movement should be sought in the *posterior* thorax to the left of the vertebral column.

The *pulmonary trunk* is normally border-forming in the second left interspace (see Fig. 5–5). To elicit a systolic impulse imparted by a dilated pulmonary trunk, the patient should be examined supine. The impulse is often better seen than palpated, especially during full held exhalation with the overlying skin marked by an X. Detection by palpation is best achieved by a gently applied fingertip. Mackenzie recorded systolic movement imparted by the pulmonary trunk (Fig. 5–26):

> Through the thin chest wall the various movements of the heart could easily be observed. In the second left interspace there was a marked pulsation. The tracings

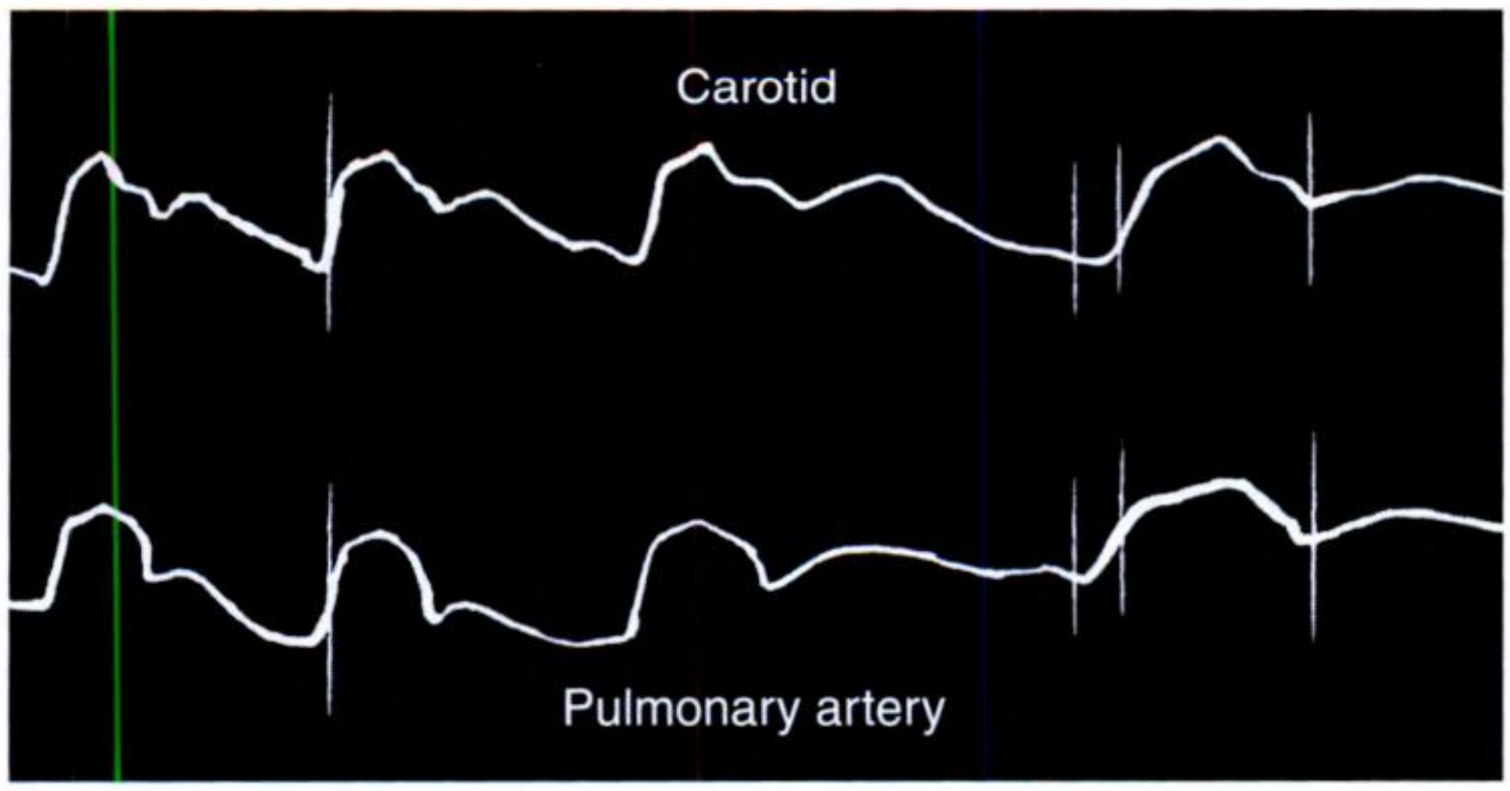


Figure 5–26 Simultaneous tracings of the pulses of the carotid and pulmonary artery. (From Mackenzie J: The Study of the Pulse, Arterial, Venous, and Hepatic, and of the Movements of the Heart. Edinburgh, Young J. Pentland, 1902.)

of the pulsation, taken at the same time as the carotid pulse, left no doubt as to its being caused by the pulmonary artery.[3]

In thin patients, especially with decreased anteroposterior chest dimensions, a normal pulmonary trunk sometimes transmits a visible and palpable impulse to the second left interspace. An *abnormal* impulse becomes evident when the trunk is dilated (pulmonary hypertensive), or when dilatation is accompanied by an increase in pulsatile excursion (ostium secundum atrial septal defect), but not when dilatation is accompanied by the proximal obstruction of pulmonary valve stenosis.

Palpable Heart Sounds

The following remarks deal with sounds that are palpable because of their intensity, not because they impart movement to the chest wall. These include loud first and second heart sounds, ejection sounds, opening snaps, and occasionally "tumor plops." Identification by palpation is analogous to the sensing of transmitted murmurs (thrills) (see below).

The mitral component of the first heart sound is sometimes palpable by relatively firm pressure of a fingertip over the left ventricular impulse in the left lateral decubitus position during held exhalation in thin, slightly built subjects, especially if the PR interval is short and the cardiac rate is rapid. In rheumatic mitral stenosis with a mobile anterior leaflet, a loud first heart sound is readily palpable and may extend to the left sternal edge and occasionally to the base. An ejection sound accompanying congenital bicuspid aortic valve stenosis (see Chapter 6) is sometimes palpable, more readily over the left ventricular impulse than in the second right interspace, and must be distinguished from a loud first heart sound. A normal PR interval and a slow heart rate assist in this distinction. Aortic ejection sounds that originate *within* a dilated aortic root in contrast to

origin in a bicuspid valve are palpated by moderately firm pressure of a fingertip applied during held exhalation at the right base over the dilated aorta, or by firmly applying the distal metacarpals. Pulmonary ejection sounds are palpable in the second *left* intercostal space and are sensed during normal exhalation because the sounds diminish or vanish altogether during inspiration (see Chapter 6). The aortic component of the second heart sound is palpable in the second right interspace in the presence of systemic hypertension and when the aortic root is dilated and the pulmonary trunk is small as in Fallot's tetralogy with pulmonary atresia, or when the aortic root is anterior to the pulmonary trunk as in complete transposition of the great arteries.

The *pulmonary component* of the second heart sound is palpated in the second left interspace in patients with pulmonary hypertension, although dilatation of the pulmonary trunk with normal pulmonary arterial pressure occasionally renders the pulmonary component palpable. In normal children and adolescents, and in thin subjects with decreased anteroposterior chest dimensions, a normal pulmonary component of the second heart sound is occasionally palpated as a gentle tap in the second left intercostal space, and is enhanced when a fingertip is applied with moderate pressure during held exhalation. A very loud pulmonary component transmits widely to the mid and lower left sternal edge, right base, and apex, especially when the right ventricle occupies the apex.

The opening snap of mitral stenosis is the most common *early diastolic sound* amenable to palpation because of intensity *per se*. The sound is palpable over the left ventricular impulse but when loud, radiates to the lower left sternal edge. A relatively rare palpable early diastolic sound is the "tumor plop" (see Chapter 6) which is generated during abrupt deceleration of a mobile, pedunculated right or left atrial myxoma as the tumor seats within the tricuspid or mitral orifice.

Palpable Murmurs—Thrills

A *thrill* by definition is a palpable murmur, so the term "palpable thrill" is redundant. A thrill should be identified as present or absent but not as palpable or impalpable.

Murmurs that reach or exceed grade IV out of VI are transmitted through the chest wall as thrills, but frequency composition also affects murmur transmission. Nevertheless, the presence of a thrill implies that a murmur is at least grade IV. The distal metacarpals are especially useful for the detection of thrills, although fingertips assist in localization. Thrills are best characterized according to location, direction of radiation, duration, and timing in the cardiac cycle ie, systolic, diastolic, or continuous: systolic thrills at the right or left base—aortic valve stenosis or pulmonary valve stenosis—are palpated with the patient supine or still better sitting and leaning forward during full held exhalation. When a thrill is prominent and widespread, application of the entire palm of the hand is helpful in sensing the direction of radiation. An aortic stenotic thrill radiates upward and to the right, and a pulmonary stenotic thrill radiates upward and to the left. If a thrill is localized, relatively firm application of a fingertip usually suffices for detection. In adults, application of the metacarpals is helpful.

It is useful to localize the maximum intensity of a left parasternal systolic thrill. Maximum intensity in the second intercostal space occurs with pulmonary *valve* stenosis, in the third intercostal space with *infundibular* pulmonary stenosis, and in the fourth or fifth intercostal space with a *restrictive ventricular septal defect*. Two or three fingertips are applied simultaneously in the second, third and fourth, or fourth, fifth, and sixth interspaces, as shown in Figure 5–7. Sequential lifting and reapplication of the fingertips refines discrimination. In infants, a single fingertip is applied in sequence to each interspace (Fig. 5–8). Most systolic thrills are better identified during exhalation, but the thrill of tricuspid regurgitation is sensed or may appear only during inspiratory amplification of the murmur (see Chapter 6).

An *early diastolic left parasternal thrill is* more likely to accompany aortic regurgitation, less likely the Graham Steell murmur of high-pressure pulmonary regurgitation which is usually grade III or less. The best technique for eliciting these left parasternal high-frequency early diastolic thrills is by applying the distal metacarpals or fingertips at the left sternal edge with the patient first supine and then sitting and leaning forward during full held exhalation. When aortic regurgitation is caused by eversion of a cusp, the pure frequency composition of the diastolic murmur sometimes results in more ready transmission than would be anticipated based upon intensity *per se*. When the diastolic thrill of aortic regurgitation is better detected at the *right* sternal edge, the cause is likely to be an ascending aortic aneurysm, as in Marfan syndrome.

Systolic and diastolic thrills over the left ventricular impulse are best assessed during held exhalation with the patient in a partial left lateral decubitus position. The thrill of mitral regurgitation commonly radiates into the axilla, sometimes to the left sternal edge, to the base, and even into the neck. Detection of the *diastolic or presystolic* thrill of mitral stenosis requires special care. With the patient in a left lateral decubitus position, the left ventricular impulse is identified because the mitral stenotic thrill is localized to that site. If the left ventricular impulse is inconspicuous, palpation of the loud first heart sound assists in identifying the apex. A fingertip is then applied with gentle to moderate pressure. Equivocal mitral stenotic thrills become evident after a brisk cough that transiently increases the heart rate and mitral flow (see Chapter 6).

When systole and diastole thrills coexist, a distinction must be made between two *separate* thrills and a single *continuous* thrill. The continuous thrill of patent ductus arteriosus is a case in point. Maximal intensity is inferior to the left clavicle. The ductus thrill begins in systole, is reinforced before and after the second heart sound, and proceeds without interruption through the timing of the second heart sound into all or part of diastole. By contrast, the systolic/diastolic thrills of aortic stenosis/regurgitation are maximal at the right base or mid-left sternal edge, and the systolic and diastolic portions are interrupted. More difficult and sometimes impossible to distinguish from a continuous thrill is the holosystolic thrill of ventricular septal defect or mitral regurgitation followed immediately by an early diastolic thrill of aortic regurgitation (see Chapter 6).

REFERENCES

1. Harvey W. An Anatomical Disquisition on the Motion of the Heart and Blood in Animals. London. 1628. In: Willius, FA, Keys TE, eds. *Classics of Cardiology.* Robert E. Krieger, 1983.
2. Laennec RTH. *Treatise on Mediate Auscultation.* 1819. (First American edition, Philadelphia, PA: James Webster, 1823.)
3. Mackenzie J. *The Study of the Pulse, Arterial, Venous, and Hepatic, and of the Movements of the Heart.* Edinburgh: Young J. Pentland, 1902.
4. Auenbrugger L. *On Percussion of the Chest.* Vienna:1761. (Translated by Forbes J. In: Diseases of the Chest. Fleet Street, London: T and G Underwood, 1824.
5. Willius FA, Dry TJ. *A History of the Heart and the Circulation.* Philadelphia, PA: WB Saunders, 1948.
6. Chauveau JBA, Marey EJ. Determination graphique des rapports de la pulsation cardiaque avec les mouvements de l'oreillette et du ventricule, obtenue au moyen d'un appareil enregistreur. *C R Soc Biol (Paris).* 1861;13:3.
7. Buckberg G. Guest Editor. Rethinking the cardiac helix. *Eur J Cardiothorac Surg.* Vol 29, Suppl.1, April 2006.
8. Boicourt OW, Nagle RE, Mounsey JPD. The clinical significance of systolic retraction of the apical impulse. *Br Heart J.* 1965:27:379.
9. Potain PC. Concerning the cardiac rhythm called gallop rhythm. *Bull. Mem. Soc. Med. Hop. Paris*, 1876. In: Major RH. *Classic Descriptions of Disease.* 3rd ed. Springfield, IL: Charles C Thomas, 1948.

6 Auscultation—The Audible Language of the Heart

With each movement of the heart, when there is the delivery of a quantity of blood from the veins to the arteries, a pulse takes place and can be heard within the chest.

William Harvey, de Motu Cordis, 1628.

The word **auscultation** is applied to the examination, made by means of the ear, of the different sounds which the circulation of the air, the reverberation of the voice, or the beatings of the heart, produce in the cavity of the chest.[1]

Auscultation may be mediate or immediate. The application of the naked ear to the different points of the chest, is called immediate auscultation. It is not only inconvenient and disagreeable in many cases both to the patient and physician; but it is besides, far from giving the satisfactory results which it would seem to promise. These numerous inconveniences prevent us from having recourse to this method as often as we would wish; but it has now been superseded by another. I mean the use of the stethoscope, an instrument as simple in its construction, as it is easy in its application, and which M. Laennec has shown to be so fruitful in results, so advantageous, I will even say indispensable, in the practice of medicine![1]

This testimony to the indispensability of mediate auscultation was written by 21-year-old William Stokes in *An Introduction to the Use of the Stethoscope* (1825).[1] But now it has been asked whether auscultation should be rehabilitated:

> Should it be reserved for the occupational therapy of a dwindling coterie of antiquarians, or should it be promoted more vigorously as a viable part of our diagnostic armamentarium?[2]
>
> The answer is ***yes***—auscultation *should* be rehabilitated, but
>
> To decipher the auscultatory language of diseases of the heart easily and accurately is an affair requiring labor and use and docility.[3]

The phonocardiogram (Fig. 6–1), which played such a seminal role in defining heart sounds and murmurs, and which served as an invaluable tool for the teaching of auscultation, has virtually disappeared. The tracings that illustrate this chapter are a rarity not to be duplicated.

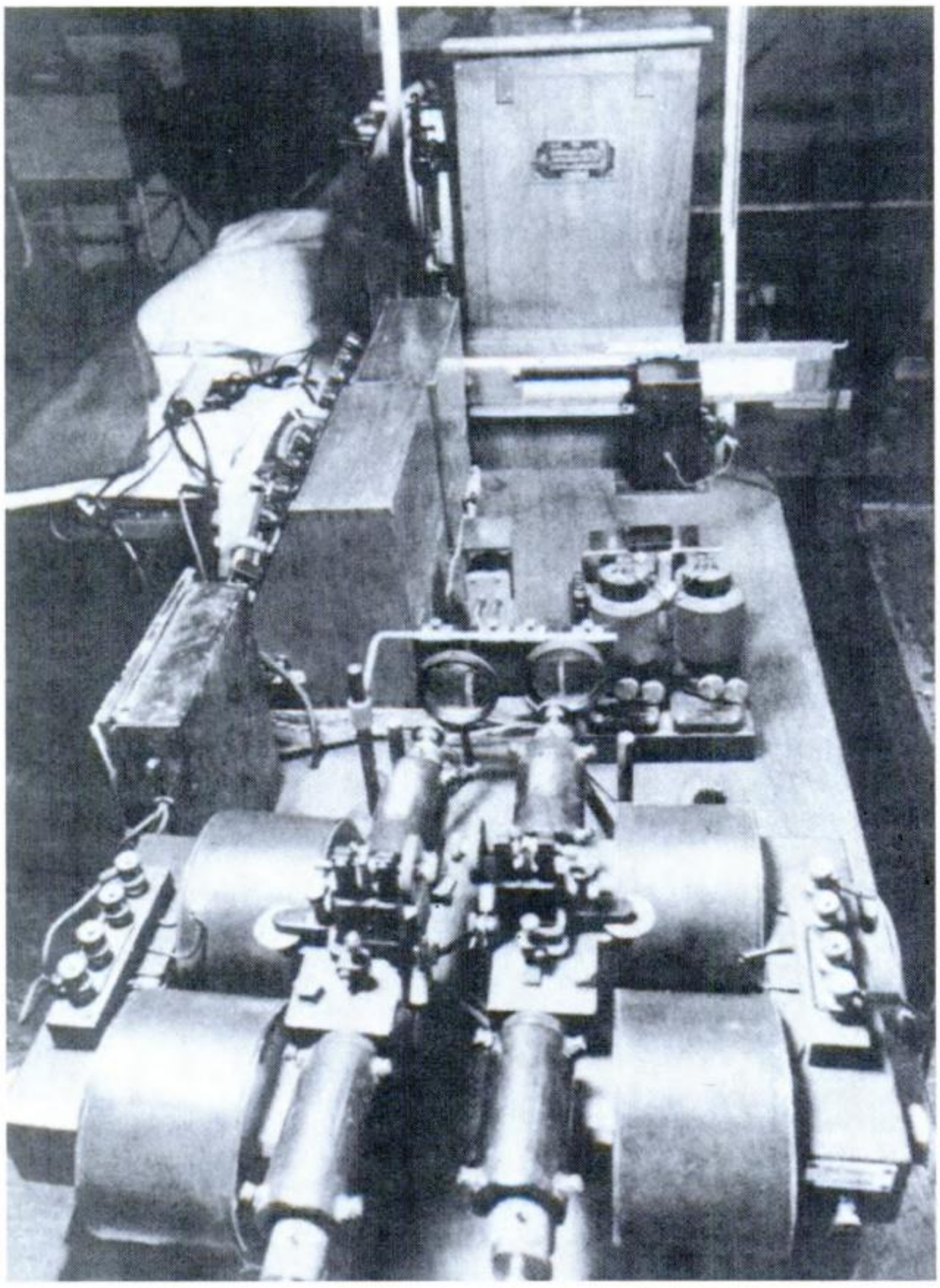


Figure 6–1 High-definition phonocardiograph consisting of two Cambridge strings and two mirror galvanometers. The recorder was constructed in the 1950s at the Institute of Cardiology, National Heart Hospital, London, and was used by Dr Aubrey Leatham. Most of the recordings in Leatham's *Auscultation of the Heart and Phonocardiography* (2nd ed. New York, Churchill Livingstone, 1975) were made with this instrument, which is now housed in the Wellcome Museum, London.

The stethoscope has superseded the head mirror as the logo of medicine, and has become more visible than ever, worn draped across the neck or over a shoulder instead of resting inconspicuously in the pocket of a laboratory coat. Regrettably, the skill with which the instrument is used tends to be inversely proportional to the prominence of its display. Is the modem stethoscope that evolved from Laennec's cylinder becoming little more than a symbolic gesture to a distinguished past? In this chapter, I hope to counter this trend.

A painting in the Necker Hospital in Paris shows Rene Theophile Hyacinthe Laennec sitting at the bedside with his ear applied to a patient's thorax (see Chapter 1, Fig. 1–10). The alternative to direct (immediate) auscultation was a quire of paper and a wooden cylinder that evolved into the binaural stethoscope. Here is Laënnec's original description:

> In 1816, I was consulted by a young woman laboring under general symptoms of diseased heart, and in whose case percussion and the application of the hand were of little avail on account of the great degree of fatness. The other method just mentioned being rendered inadmissible by age and sex of the patient, I happened to recollect a simple and well-known fact of acoustics, and fancied at the same time,

that it might be turned to some use on the present occasion. The fact I allude to is the augmented impression of sound when conveyed through certain solid bodies, as when we hear the scratch of a pin at one end of a piece of wood on applying our ear to the other. Immediately, on this suggestion, I rolled a quire of paper into a sort of cylinder and applied one end of it to the region of the heart and the other to my ear, and was not a little surprised and pleased, to find that I could thereby perceive the action of the heart in a manner much more clear and distinct than I had ever been able to do by the immediate application of the ear. From this moment, I imagined that the circumstance might furnish means for enabling us to ascertain the character, not only of the action of the heart, but of every species of sound produced by the motion of all the thoracic viscera.[4]

Nine years after Laennec's discovery of the stethoscopic cylinder, William Stokes wrote:

> We cannot in every case come to an *absolutely* certain conclusion by means of the stethoscope, still it is of infinite assistance to us, and in the hands of an experienced observer, will lead to conclusions unattainable without its aid.[1]

Stokes went on to say:

> In the examination of the heart by means of the stethoscope, we must be assured that the patient is in the calmest possible state; that he shall not have taken any exercise for some hours previously, nor indulged lately in the use of wine, or other spiritous liquors. He should be examined before his usual time of meals, for in many patients, the action of the heart is either greatly increased after a meal, or may become so irregular as to simulate disease.[1]

These instructions recall the *Yellow Emperor's Book of Medicine*[5] in which the arterial pulse was examined in the early morning before the physician took food and before the cares of the day distracted him from full concentration (see Chapter 1, Fig. 1–3).

HEART SOUNDS

Heart sounds are brief, discrete auditory vibrations of varying intensity (loudness), frequency (pitch), and quality (timbre). The first heart sound identifies the onset of ventricular systole, and the second heart sound the onset of diastole. These two auscultatory events establish a framework within which murmurs and heart sounds can be placed.

Laennec attributed the first sound to ventricular systole (*bruit ventriculaire*) and the second sound to atrial systole (*bruit auriculaire*).[4] In 1832, Joseph Rouanet published his experiments on the *valvular* origin of heart sounds,[6] conclusions in accord with current opinion that the first and second heart sounds originate in cardiac valves, and are generated by tensing of valve membranes rather than collision of cusps.

The four basic heart sounds are the first, second, third, and fourth (Figs. 6–2*A* and 6–3*A*). Each of these sounds can be normal or abnormal. Other heart sounds are usually abnormal, whereas still others are iatrogenic, ie, produced by prosthetic valves.

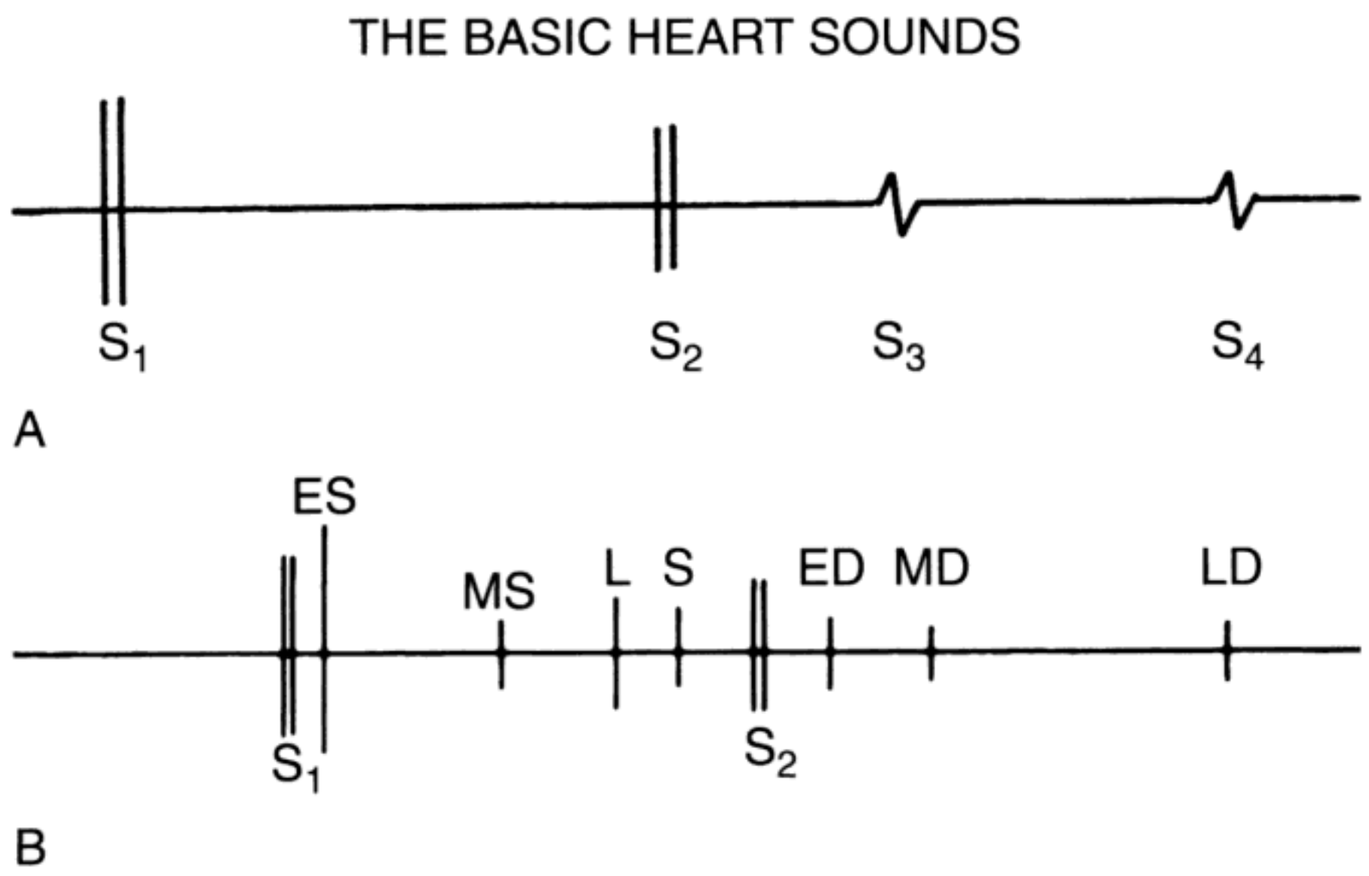


Figure 6–2 *A,* The basic heart sounds represented by the first sound (S_1), the second sound (S_2), the third sound (S_3), and the fourth sound (S_4). Each of these sounds can be normal or abnormal depending on the clinical context. *B,* Heart sounds within the auditory framework established by the first heart sound (S_1) and the second heart sound (S_2). The additional heart sounds are designated descriptively as early systolic (ES), midsystolic (MS), late systolic (LS), and early diastolic (ED), mid-diastolic (MD), and late diastolic (LD) or presystolic.

Heart sounds are assigned simple descriptive designations that identify *where* in the cardiac cycle a given sound occurs—*early systolic, midsystolic, late systolic* and *early diastolic, mid-diastolic and late diastolic (presystolic)* (Fig. 6–2*B* and Table 6–1).

The next step is to infer what a sound so characterized represents (Tables 6–2 and 6–3). An *early systolic* sound might be an ejection sound (aortic or pulmonary) or an aortic prosthetic sound. *Mid/late systolic* sounds are typified by the click(s) of mitral valve prolapse, but occasionally are "remnants" of pericardial rubs. *Early diastolic* sounds are represented by mitral opening snaps, the early third heart sound of constrictive pericarditis, the "ventricular knock" of mitral regurgitation, the opening of a rigid mitral prosthesis, or the abrupt seating of a mobile atrial myxoma (tumor plop). *Mid-diastolic* sounds are generally third heart sounds or summation sounds (synchronous third and fourth heart sounds). *Late diastolic or presystolic* sounds are almost always fourth heart sounds, rarely pacemaker sounds.

Technique and Principles

The importance of the topographic anatomy of the heart was emphasized by Henry I. Bowditch in *The Young Stethoscopist (1848)*. Bowditch provided a diagram that

> will serve to show the position of the heart, and of its valves with reference to the sternum and the ribs, during life, when fully distended. It will be well to study this diagram carefully, in order to get a definite idea of the various parts in which we should auscult in order to hear most clearly the sounds from the different valves.[7]

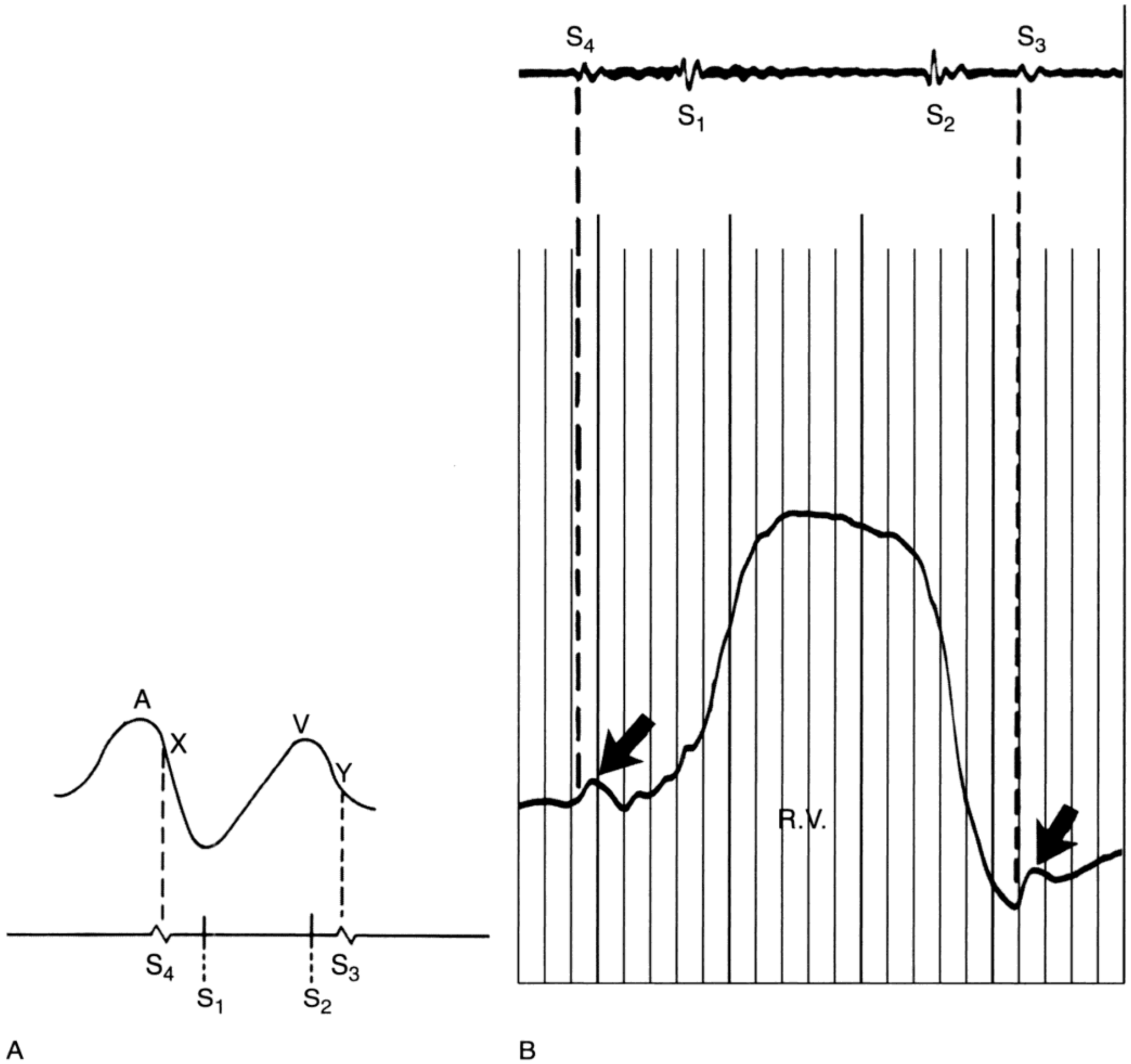


Figure 6–3 *A,* Atrial pressure pulse showing the A wave and X descent, and V wave and Y descent. The fourth heart sound (S_4) coincides with the atrial contraction phase of ventricular filling. The third heart sound (S_3) coincides with the Y descent (the phase of rapid ventricular filling). S_1 = first heart sound; S_2 = second heart sound. *B,* Intracardiac phonocardiogram recorded from within the right ventricular (RV) cavity. The fourth heart sound (S_4) coincides with presystolic filling of the right ventricle *(first arrow)* in response to right atrial contraction. The third heart sound (S_3) coincides with rapid, passive filling of the right ventricle (*second arrow*).

Table 6–1 Heart Sounds Within the Framework of the First and Second Heart Sounds: Descriptive Terminology

Systolic sounds:	Diastolic sounds:
Early systolic	Early diastolic
Mid systolic	Mid diastolic
Late systolic	Late diastolic

Table 6–2 Systolic Sounds

Early systolic	Mid/Late systolic
Ejection sounds (aortic or pulmonary)	Mitral clicks
Prosthetic aortic valve sounds	Remnants of pericardial rubs

The topographic areas for cardiac auscultation are designated descriptively as the cardiac apex, left and right sternal borders interspace by interspace, and subxiphoid. These designations are analogous to those recommended for palpation (see Chapter 5). Terms such as "mitral area, tricuspid area, pulmonary area, and aortic area" are avoided because they assume *situs solitus without ventricular inversion and normally related great arteries*. Percussion should precede auscultation in order to establish visceral and cardiac *situs*, so that the stethoscope can be applied with topographic confidence.

Auscultation should begin at the cardiac apex and contiguous lower left sternal edge (inflow), proceeding upward interspace by interspace to the left and right base (outflow). This permits the examiner to think physiologically by using a sequence that conforms to the direction of blood flow—inflow/outflow. It has been recommended that auscultation begin at the base where the first and second heart sounds are clearly identified. With few exceptions, however, timing of the first heart sound is readily established by simultaneous palpation of the carotid artery with the thumb of the free left hand (Fig. 6–4). In addition to the routine sites described above, auscultation should be conducted at certain *nonprecordial thoracic sites*, especially the anterior chest on the opposite side, the axillae, and the back.

Bowditch called attention to signs of cardiac malpositions:

> The organs of the body are exactly reversed in their position. . . . *inspection* shows the apex of the heart beating to the right of the sternum. *Auscultation and percussion* prove

Table 6–3 Diastolic Sounds

Early diastolic:
Opening snaps
Early diastolic sound of constrictive pericardidis
The "ventricular knock" of mitral regurgitation
Prosthetic mitral valve sound
Tumor plop
Mid-diastolic:
Third heart sound
Summation sound ($S_3 + S_4$)
Late diastolic (presystolic):
Fourth heart sound
Pacemaker sound

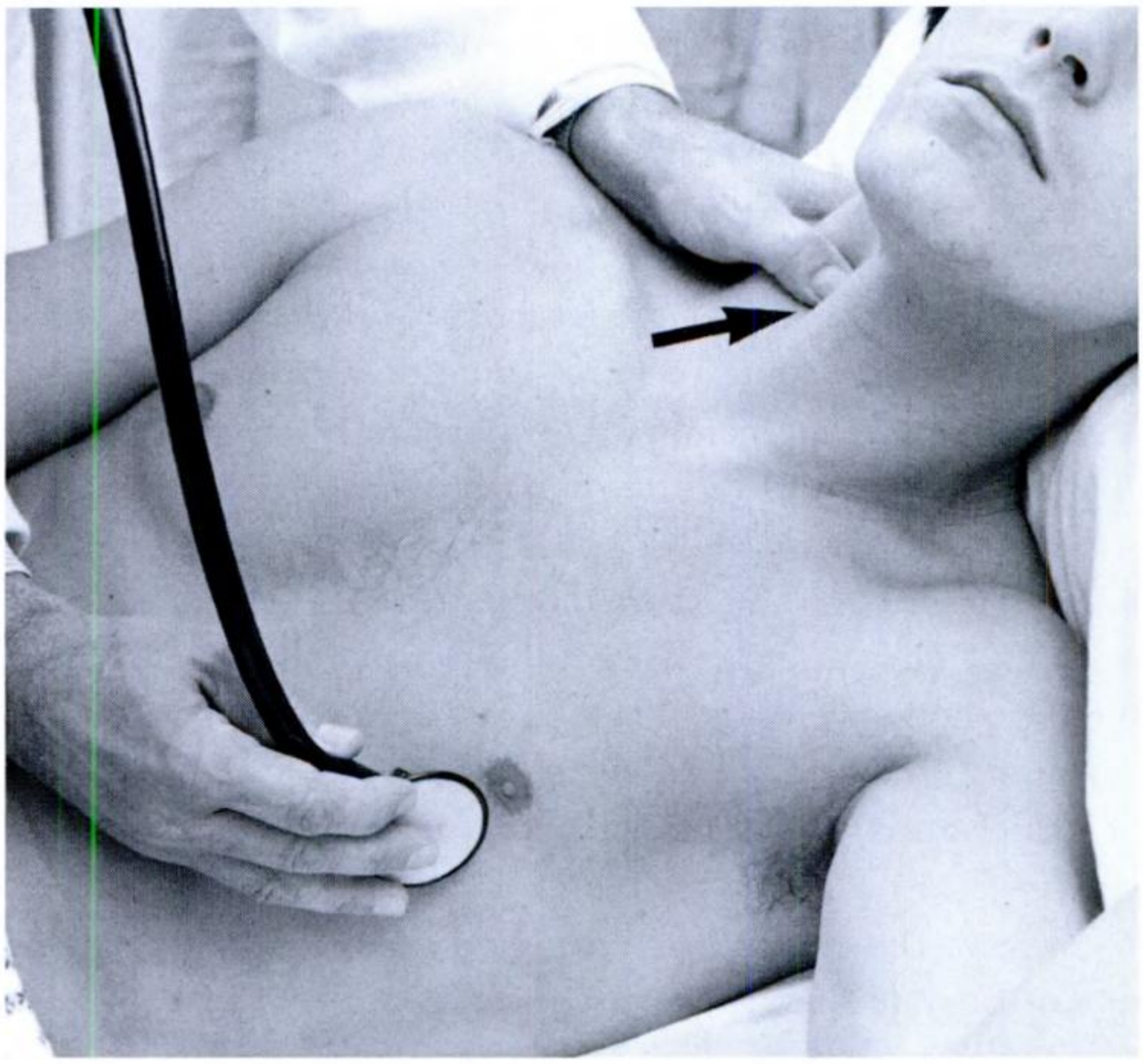


Figure 6–4 The bell of the stethoscope applied to the cardiac apex while the patient lies in a partial left lateral decubitus position. The thumb of the examiner's left hand palpates the carotid artery for timing purposes (*arrow*).

> the same; the sounds being heard at the right side of the sternum, perfectly normal, and the *percussion* gives dull sounds on the right of the breast, while the left is clear.[7]

In *situs solitus* with a *left thoracic heart*, the stethoscope is first applied to the cardiac apex with the patient in a partial left lateral decubitus position (Fig. 6–4). Once the first heart sound is identified by simultaneous carotid palpation (Fig. 6–4), auscultation proceeds by systematic attention to early, mid, and late systole, the second heart sound, then early, mid-, and late diastole (presystole), returning to the first heart sound (see Fig. 6–2). Orderly auscultation should not be distracted by dramatic signs such as a loud murmur, because less obvious signs will be overlooked.

Pitch or frequency can be assessed within a range from low to moderately high by applying variable pressure with the stethoscopic bell, reserving the diaphragm for higher frequencies. A practical sequence is to begin with the stethoscopic bell applied with varying degrees of pressure at the apex and lower left sternal edge, changing to the diaphragm when the base is reached. Low frequencies are heard when the bell applied just lightly enough to establish a skin seal. High-frequency events are elicited by firm pressure of the diaphragm.

Respiratory patterns, position, and physical interventions will be dealt with subsequently.

First Heart Sound

The initial component of the first heart sound is most prominent at the cardiac apex when the apex is occupied by the left ventricle. A second component can be heard at the lower left sternal edge, less commonly at the apex, and seldom at the base (see below). The first component coincides with closure of the mitral valve and abrupt arrest of leaflet motion when the cusps—especially the larger and more mobile anterior cusp—reach their fully closed position (maximal excursion into the left atrium). The sound is "more a matter of tensing of the valve membranes than of collision of the cusp margins," as Rouanet originally proposed.[6] Calcification renders a valve immobile and hence inaudible. The origin of the second component of the first heart sound is attributed to closure of the tricuspid valve based upon an analogous line of reasoning.

Ejection into the great arteries (ascending aorta and pulmonary trunk) is normally inaudible, although phonocardiograms may record a low-amplitude sound coinciding with maximal opening excursions of the aortic cusps.

The first heart sound is characterized according to quality and intensity in addition to the presence and degree of splitting. Because the two major components are believed to originate in the closing movements of the two atrioventricular valves, the quality/pitch is similar and is best appreciated with relatively firm pressure of the stethoscopic bell or with the diaphragm. When the first heart sound is split, its first component is normally the louder. The softer second component is confined to the apex and lower left sternal edge. Only the first component is heard at the base. The intensity of the first heart sound, particularly its first component, depends chiefly on the position of the bellies of the mitral leaflets, especially the anterior leaflet, at the onset of left ventricular systole, less on the velocity of ventricular contraction. Accordingly, the initial component of the first heart sound is loudest when the onset of left ventricular systole finds the mitral leaflets maximally recessed into the ventricular cavity as in mitral stenosis, and in the presence of a short PR interval, a rapid heart rate, and short cycle lengths in atrial fibrillation.

Conversely, the first heart sound is soft, even inaudible when the onset of left ventricular systole finds the mitral leaflets in or near their closed position, as in the presence of a long PR interval, bradycardia, or long cycle lengths in atrial fibrillation. In acute severe aortic regurgitation, the steep rise in left ventricular diastolic pressure exceeds an elevated left atrial pressure in later diastole, prematurely closing the mitral valve, sometimes before inscription of the P wave. Accordingly, the first heart sound is attenuated or absent, an auscultatory sign that stands out because the attenuation occurs despite tachycardia and a normal PR interval. In complete heart block, the random relationship between P wave and QRS results in random variability in intensity of the first heart sound—loud when the PR interval is short, and soft when the PR interval is long (see Fig. 6–35*A*).

Systolic Sounds

Ejection sounds (aortic and pulmonary) are the most common early systolic sounds (see Fig. 6–2*B*), coinciding with timing of the maximum cephalad excursion of the relevant

semilunar valve at its fully opened position, as in bicuspid aortic valve stenosis or dilated aortic root in the left heart, or pulmonary valve stenosis or dilated pulmonary trunk in the right heart. Ejection sounds are relatively high-frequency, hence the designation ejection "click." The term ejection "sound" is preferable, however, reserving the term "click" for the mid to late systolic sounds of mitral valve prolapse (see below), thus avoiding the awkward term "nonejection click" for the latter.

Ejection sounds, because of their frequency composition, should be assessed with the stethoscopic diaphragm or with firm pressure of the bell. The ejection sound of a congenital bicuspid valve is transmitted into the left ventricular cavity and is heard best over the left ventricular impulse rather than at the right base (Fig. 6–5*A*). An ejection sound implies that the aortic valve is mobile, because the sound is caused by abrupt cephalad doming (Fig. 6–5*B,C*). In congenital pulmonary valve stenosis, the ejection sound coincides with abrupt cephalad doming of the mobile pulmonary valve, and is transmitted to the overlying second left intercostal space (Fig. 6–6). A distinctive feature of the pulmonary ejection sound is its selective decrease in intensity during normal inspiration (Fig. 6–6*A*). That is so because the inspiratory increase in right atrial contractile force is transmitted into the right ventricle and onto the undersurface of the mobile stenotic valve, moving its cusps upward *before* the onset of right ventricular contraction. The result is diminished caphalad excursion of the valve during inspiration, accounting for the inspiratory decrease in intensity. The source of an ejection sound in a dilated aortic root or dilated pulmonary trunk is also probably valvular (the timing is appropriate).

An aortic ejection sound following a first heart sound at the cardiac apex (see Fig. 6–5*A*) must be distinguished from a split first heart sound. The quality of the two sounds is similar if not identical, and both are well heard with the diaphragm of the stethoscope or firm pressure of the bell. However, a split first heart sound is characterized by a louder *first* component (mitral), whereas a first heart sound followed by an aortic ejection sound is represented by a louder *second* component, as shown in Figure 6–5*A*.

An early systolic sound is generated by a rigid aortic prosthesis but not by a tissue valve. A ball-in-cage valve sometimes produces a trill of early systolic sounds as the poppet moves upward during left ventricular systole.

Mid to Late Systolic Sounds

The most common mid to late systolic sound(s) are the *clicks* associated with mitral valve prolapse (Fig. 6–7*A*). The term is appropriate because these sounds are relatively high-frequency and clicking, and are best heard with the diaphragm of the stethoscope or firm pressure of the bell. The clicks coincide with maximal excursion of the prolapsed leaflet(s) into the left atrium, and are ascribed to abrupt tensing of redundant leaflet(s) and elongated chordae tendineae. Variability epitomizes systolic click(s), which from time-to-time may be present, absent, single, or multiple (Fig. 6–7*A,B*), or may be replaced by a cluster of late systolic "crackles." With physical or pharmacologic interventions that reduce left ventricular volume, such as the Valsalva maneuver, amyl nitrite (Fig. 6–7*C*), or a change

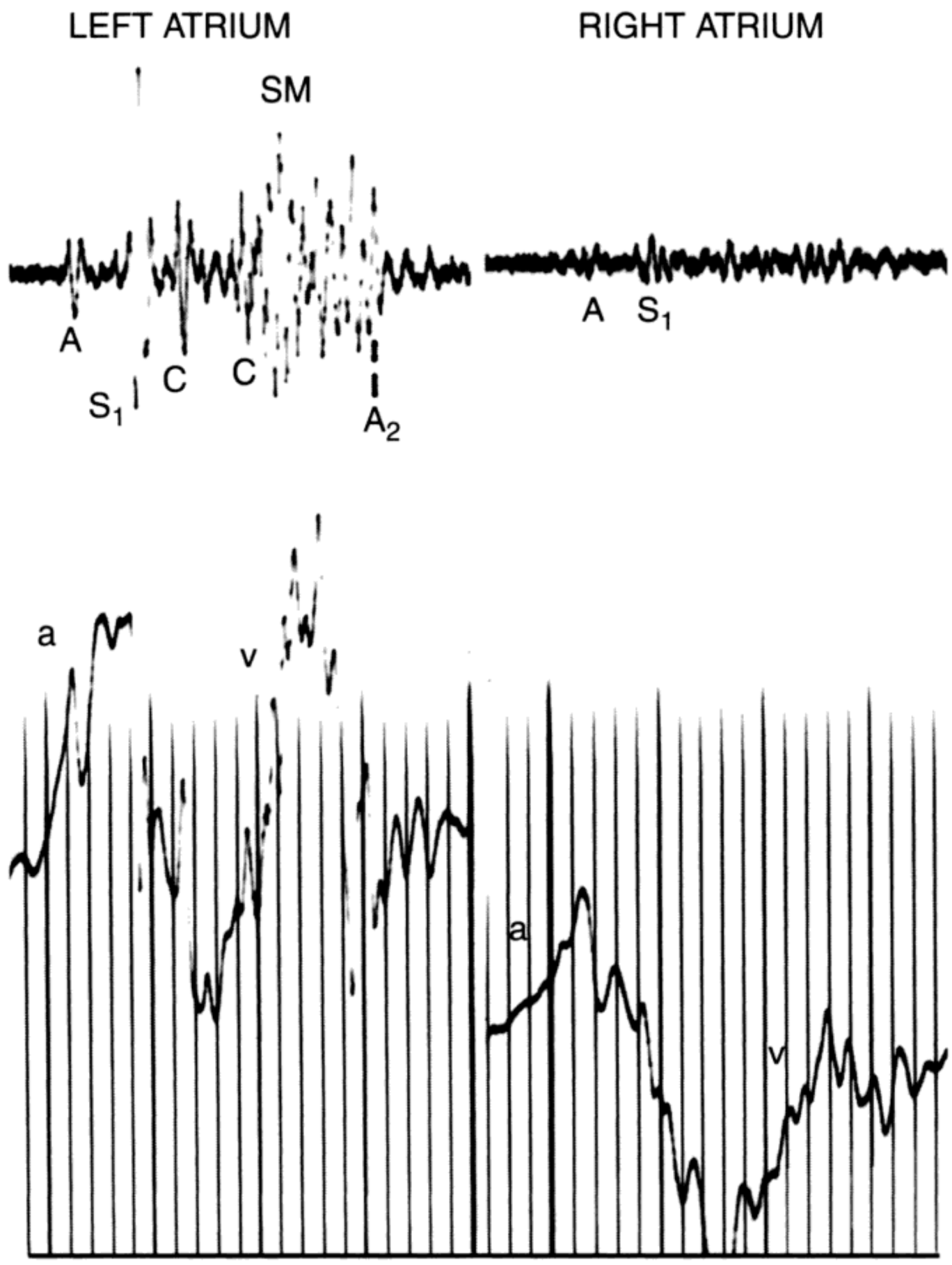


Figure 6–7 *A,* Intracardiac phonocardiogram localizing the systolic clicks (C) and late systolic murmur (SM) of mitral valve prolapse within the left atrium. The clicks (C) and late systolic murmur (SM) disappeared and the "a" and "v" waves declined as the transeptal catheter was withdrawn from left atrium into right atrium. An atrial sound (A), now termed a fourth heart sound, was more prominent within the left atrium. (From Ronan JA, Perloff JK, Harvey WP. Systolic clicks and the late systolic murmur. Intracardiac phonocardiographic evidence of their mitral valve origin. *Am Heart J* . 1965;70:319. Reprinted with permission.) *B* (*facing page*), Phonocardiograms at the cardiac apex with simultaneous brachial arterial (BA) and left atrial (LA) pressure pulses in a patient with mitral valve prolapse before (control) and after pressor amine infusion. As resistance to left ventricular discharge increased, the clicks (C) become multiple and occurred later in systole. The late systolic murmur (SM) increased in parallel with the increase in left atrial (LA) V wave. BA = brachial artery; M_1 = mitral component of the first heart sound. *C* (*see p.170*), Phonocardiograms at the apex of a patient with mitral valve prolapse before (control) and after an acute *decrease* in resistance to left ventricular discharge induced by amyl nitrite inhalation. The late systolic murmur (SM) was replaced by an early systolic decrescendo murmur that ends before the aortic component of the second heart sound (A_2). The remaining click (C) occurred earlier in systole. *Continued*

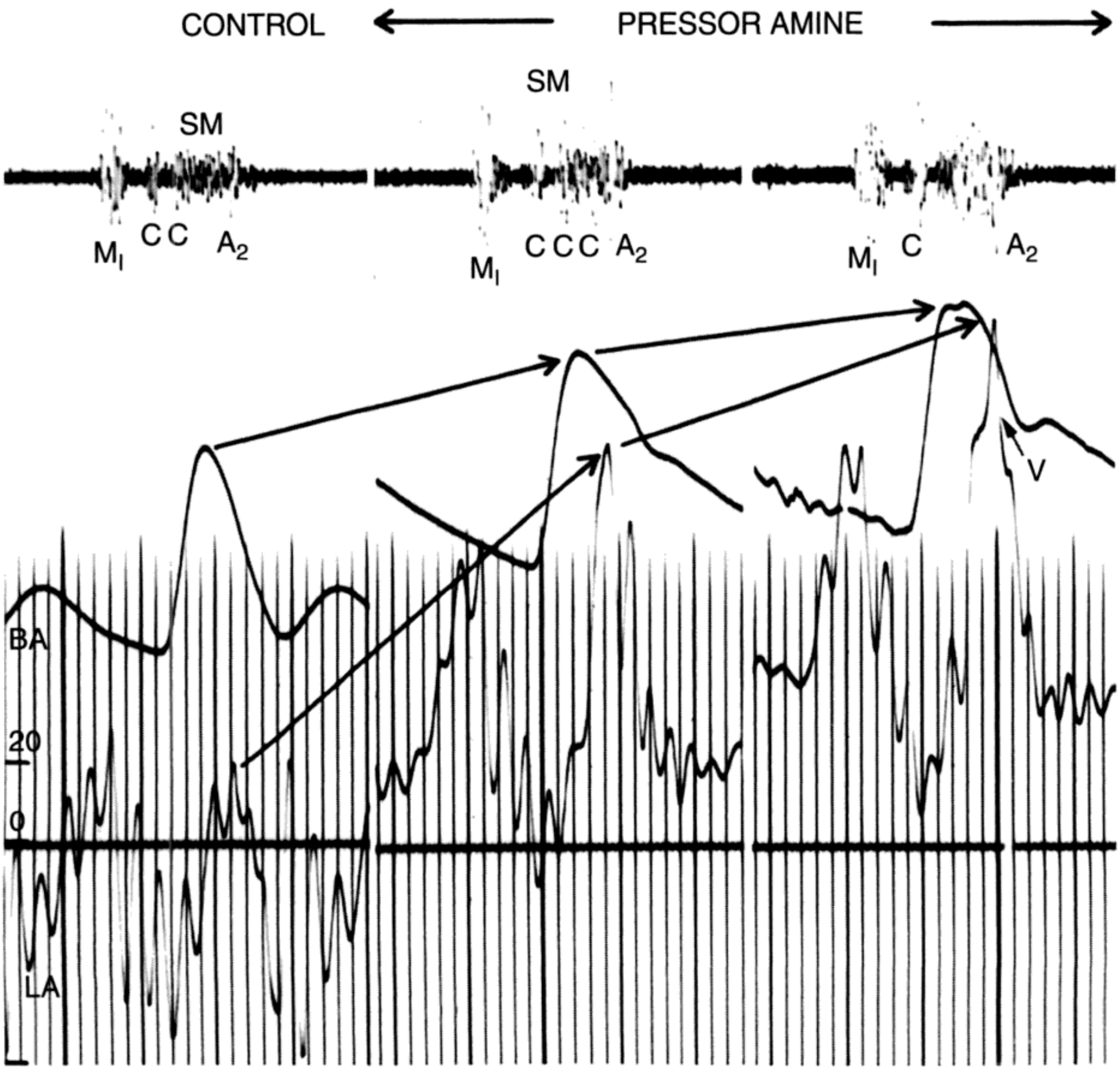


Figure 6–7 *Continued*

(capacitance) in the systemic vascular bed. Accordingly, normal respiratory variations in timing are ascribed principally to variations in impedance characteristics (capacitance) of the pulmonary vascular bed, not to an inspiratory increase in right ventricular volume as originally believed.[9] When the normal increase in capacitance of the pulmonary bed is lost because of a rise in vascular resistance, inspiratory splitting of the second sound narrows and, if present at all, reflects an increase in right ventricular ejection time and/or earlier timing of the aortic component.

Splitting of the second heart sound is best assessed with the patient supine or with a 30 degree elevation of the thorax, but reexamination in the sitting position can be useful, as commented on below. The frequency composition of the two components of the second sound calls for use of the stethoscopic diaphragm or relatively firm pressure of the bell applied in the second left intercostal space during normal respiration. It is helpful to instruct older children and adults regarding the rate and depth of respiration by asking them to "breathe in" and "breathe out." The duration and depth of breathing are established

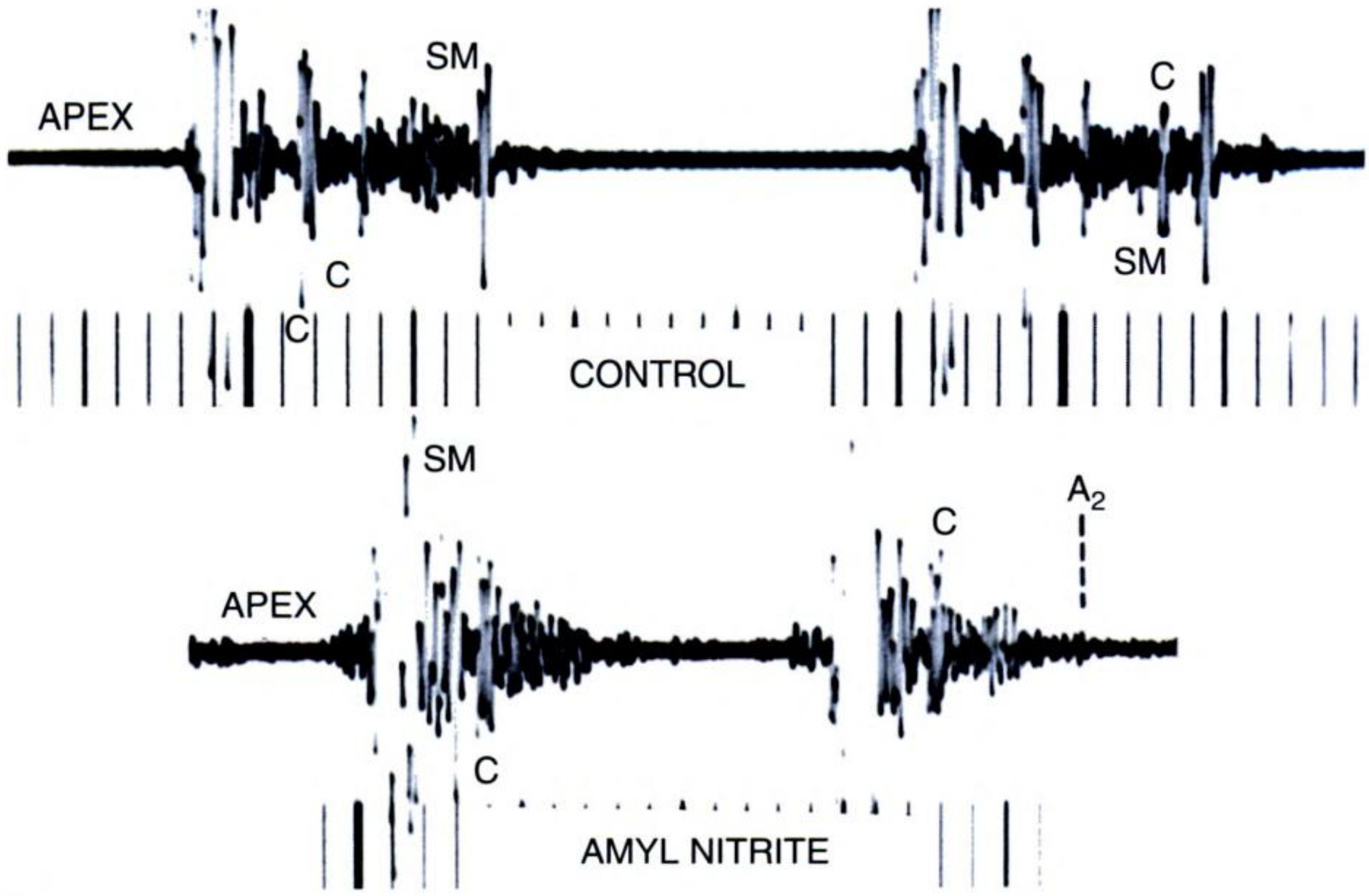


Figure 6–7 *Continued*

by the interval elapsed between the instruction to "breathe in" and the instruction to "breathe out." In infants, the technique for assessing splitting of the second heart sound differs. Rapid, nonrhythmic breathing together with a rapid heart rate conspire to prevent matching the splitting with a given phase of respiration. It is best to ignore the respiratory cycle and concentrate on the second heart sound *per se*. If it can be established with

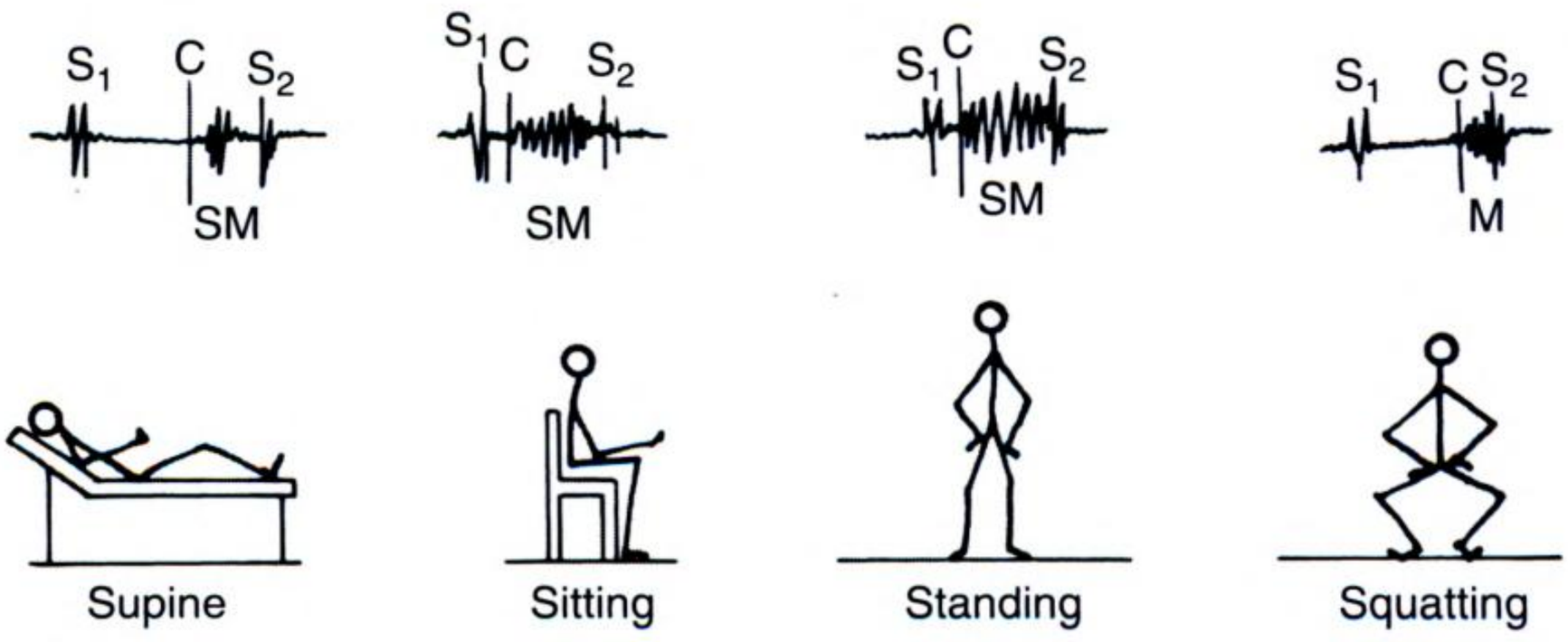


Figure 6–8 Postural maneuvers that affect the click (s) and late systolic murmur of mitral valve prolapse. A change from supine to sitting or standing causes the click(s) to become earlier and the murmur longer, although softer. Conversely, squatting delays the timing of the click, and the murmur gets shorter but louder. (From Devereux RB, Perloff JK, Reichek N, Josephson ME.Mitral valve prolapse. *Circulation*. 1976;54:3. Reprinted with permission.)

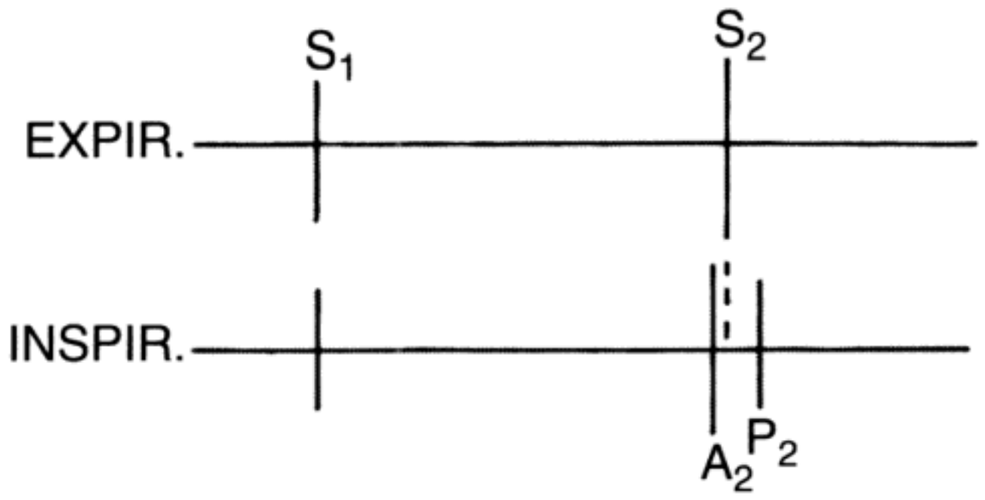


Figure 6–9 During inspiration, the second heart sound (S_2) normally splits into an aortic (A_2) and a pulmonary (P_2) component. Splitting is principally due to inspiratory delay in the pulmonary component (P_2).

confidence that the second heart sound is at times single, and at other times split, it is safe to conclude that the *sequence* of semilunar valve closure is normal (aortic followed by pulmonary), because reversed or paradoxical splitting (see below) rarely occurs in infants.

Abnormal splitting of the second heart sound falls into three categories: (1) persistently single, (2) persistently split (fixed or nonfixed), and (3) paradoxically split (reversed). When the second sound remains single throughout the respiratory cycle, one component is absent, or the two components remain synchronous. The most common cause of a single second heart sound is inaudibility of the softer pulmonary component in older adults with increased anteroposterior chest dimensions. In congenital heart disease, a single second heart sound can be due to absence of the pulmonary component (pulmonary atresia, severe pulmonary valve stenosis, a dysplastic pulmonary valve), or to the presence of transposition of the great arteries (inaudible pulmonary component because of the posterior position of the pulmonary trunk).

A single second heart sound caused by simultaneous occurrence of its two components is a feature of an Eisenmenger ventricular septal defect, in which close if not identical pulmonary and systemic vascular resistances result in aortic and pulmonary arterial dicrotic incisuras that are virtually simultaneous. A single second heart sound caused by inaudibility of the aortic component occurs when the aortic valve is atretic or when the valve is immobilized by calcific or dysplastic leaflets.

Both components of the second heart sound are sometimes absent at *all* precordial sites. This is so in older adults in whom the *aortic* component is inaudible because of calcific aortic stenosis, and the *pulmonary* component is inaudible because of a large anteroposterior chest dimension.

A single semilunar valve does not necessarily generate a single second heart sound as perceived on auscultation, and the quadricuspid valve of truncus arteriosus generates what is perceived as "splitting" but is due instead to asynchronous closure of the four unequal cusps. In systemic or pulmonary hypertension (see below), a loud single second heart sound may be sufficiently prolonged and slurred (reduplicated) to encourage a mistaken impression of splitting.

Persistent splitting of the second heart sound means that the two components remain separate and audible during both phases of respiration. Persistent splitting may be due to a delay in the pulmonary component, as in complete right bundle-branch block, or

precordial sites serves to identify the transmitted but attenuated pulmonary component and allows detection of splitting. A moderate increase in loudness of the pulmonary component of the second sound occurs with a decrease in anteroposterior chest dimension (loss of thoracic kyphosis), and with dilatation of the pulmonary trunk, as with ostium secundum atrial septal defect.

Early Diastolic Sounds

The aptly named opening snap of rheumatic mitral stenosis is the best known early diastolic sound (Fig. 6–11 and Table 6–3). An opening snap indicates mobility of the mitral valve, or at least its larger anterior leaflet, which is the source of the snap (Fig. 6–11). The opening snap of mitral stenosis is high-pitched and best detected with the stethoscopic diaphragm or firm pressure of the bell at the lower left sternal edge. The snap is less well heard at the apex, even when the left ventricle occupies the apex. A loud opening snap that radiates to the left base invites a mistaken diagnosis of wide splitting of the second heart sound. However, careful auscultation detects *two* sounds during exhalation (synchronous aortic and pulmonary components followed by the opening snap), but *three* sounds during inspiration (aortic component and pulmonary component followed by the opening snap).

The opening snap of *tricuspid* stenosis is difficult to verify by auscultation, because mitral stenosis with its snap almost invariably coexists. If the stenotic mitral valve is calcified and immobile, the audible snap becomes tricuspid by default.

The interval between the aortic component of the second heart sound and the mitral opening snap—the A_2–OS interval—can be predicted by auscultation. A short interval implies a high left atrial pressure, and significant mitral stenosis. The converse is not necessarily the case, however, because in older subjects or in the presence of systemic hypertension, tight mitral stenosis can occur with a relatively long A_2–OS interval because left ventricular systolic pressure takes longer to descend below left atrial pressure (high left ventricular systolic pressure in systemic hypertension, abnormally prolonged rate of fall of the left ventricular pressure pulse in older adults). In atrial fibrillation, the A_2–OS interval varies inversely with cycle length because the higher the left atrial pressure (short cycle length), the earlier the mitral valve opens, and *vice versa*.

Early diastolic sounds are features of constrictive pericarditis. Dominic Corrigan, in a presentation before the Pathological Society of Dublin in 1842, commented on a patient with "a very loud *bruit de frappement*."[12] *Frapper* "to knock" implies that the *bruit de frappement* was a knocking sound in Corrigan's patient who had constrictive pericarditis. The term "knock" has also been applied to an early diastolic sound in pure severe mitral regurgitation with reduced left ventricular diastolic distensibility. Corrigan's "pericardial knock" and the "ventricular knock" of mitral regurgitation are rapid filling sounds that are early and loud because a high-pressure atrium decompresses rapidly across an unobstructed atrioventricular valve into a recipient ventricle whose compliance is reduced.

An *early diastolic sound* in pure severe mitral regurgitation occasionally originates from a mobile anterior leaflet that opens rapidly because of a high left atrial V wave and

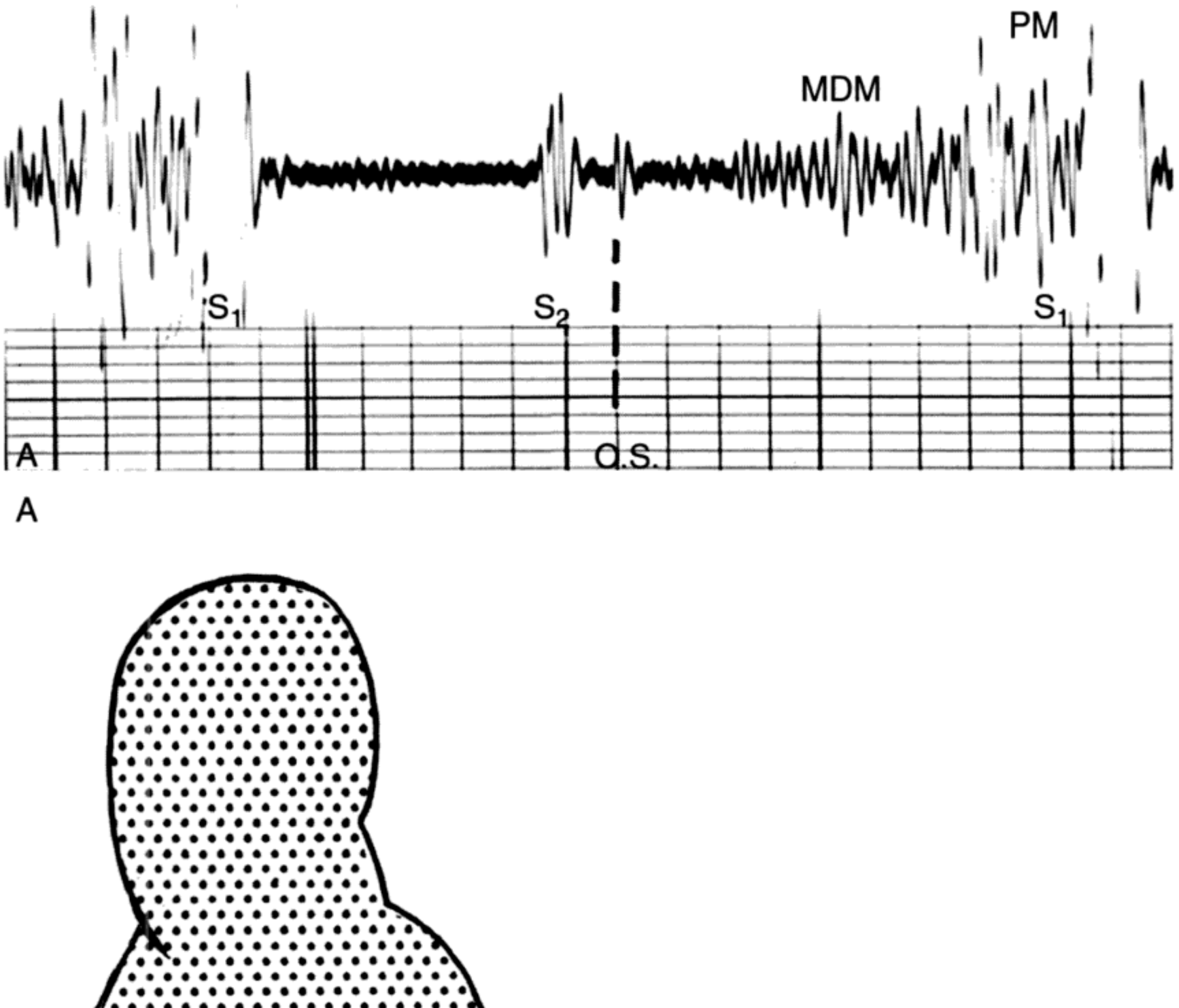


Figure 6–11 *A,* Phonocardiogram recorded over the left ventricular impulse in a patient with pure rheumatic mitral stenosis. An opening snap (OS) is followed by a mid-diastolic murmur (MDM) that merges into a presystolic murmur (PM) with a crescendo up to a loud first heart sound (S_1). S_2, second heart sound. *B,* Schematic illustration emphasizing that the loud first heart sound (S_1), opening snap (OS), mid-diastolic murmur (MDM), and presystolic murmur (PSM) of mitral stenosis are best heard over the left ventricular (LV) impulse.

a collapsing Y descent. It is difficult if not impossible to distinguish this "opening snap" from the "ventricular knock" of pure mitral regurgitation. The two sounds may coexist.

Early diastolic sounds—*tumor plops*—are sometimes caused by mobile atrial myxomas attached to the atrial septum by a long stalk. The "plop" results from abrupt

diastolic seating of the myxomatous mass within the right or left atrioventricular orifice. The opening movement of a rigid mitral prosthesis generates an early diastolic sound which is especially prominent with a ball-in-cage device (Starr–Edwards), less so with a tilting disc prosthesis and absent with tissue valves.

Mid-Diastolic/Late Diastolic (Presystolic) Sounds

In sinus rhythm, each ventricle receives blood during two diastolic filling phases (see Fig. 6–3). The first phase occurs when ventricular diastolic pressure drops sufficiently below atrial diastolic pressure to allow the atrioventricular valves to open, so blood flows from atrium into ventricle. Flow coincides with the Y descent of the atrial pressure pulse (see Fig. 6–3), ie, the rapid filling phase of ventricular diastole that accounts for about 80 percent of normal filling. The rapid filling phase is a complex, active, energy-dependent process, not a passive event in which inflow simply expands the recipient ventricle. The sound generated during the rapid filling phase is the third heart sound (see Fig. 6–3).

The second phase—*diastasis*—is variable in duration, and is usually accompanied by less than five per cent of ventricular filling.

The third phase is in response to atrial systole which accounts for about 15 percent of normal ventricular filling. The sound generated during the atrial filling phase is the fourth heart sound (see Fig. 6–3). Third and fourth heart sounds both occur *within* the recipient ventricle when that chamber receives blood. Potain attributed the third heart sound to sudden cessation of ventricular distention in early diastole, and he attributed the fourth heart sound to "the abruptness with which the dilatation of the ventricle takes place during the presystolic period which corresponds to contraction of the auricle."[13]

The presence of third and fourth heart sounds can produce a cadence called *gallop rhythm* which is a *pathologic* designation and therefore inappropriate unless the causative sound—be it a third or fourth heart sound—is *abnormal.* Potain wrote his classic description of "cardiac rhythm called gallop rhythm" in 1876.[13] The following is his account of gallop rhythm caused by a fourth heart sound:

> The formation of this rhythm of which I wish to speak, is as follows. We distinguish here three sounds, namely: the two normal sounds and an additional sound. The normal sounds show most frequently their normal characteristics, without any modification. The first especially maintains its normal relationship to the apex heart and to the arterial phase. As to the abnormal sound, it is placed immediately before it, preceding it sometimes by a very short time; always notably larger, however, than that which separates the two parts of a reduplicated sound.[13]

Gallop rhythm caused by the addition of a third or fourth heart sound is a *triple* rhythm (Fig. 6–12). The presence of both third *and* fourth heart sounds is a *quadruple* rhythm (Fig. 6–12). When diastole is short or the PR interval long, the third and fourth heart sounds merge to form a summation sound or a summation gallop rhythm (Fig. 6–12).

The presence of these sounds is more important than the cadence they create. When third or fourth heart sounds coexist with other sounds or murmurs, the gallop

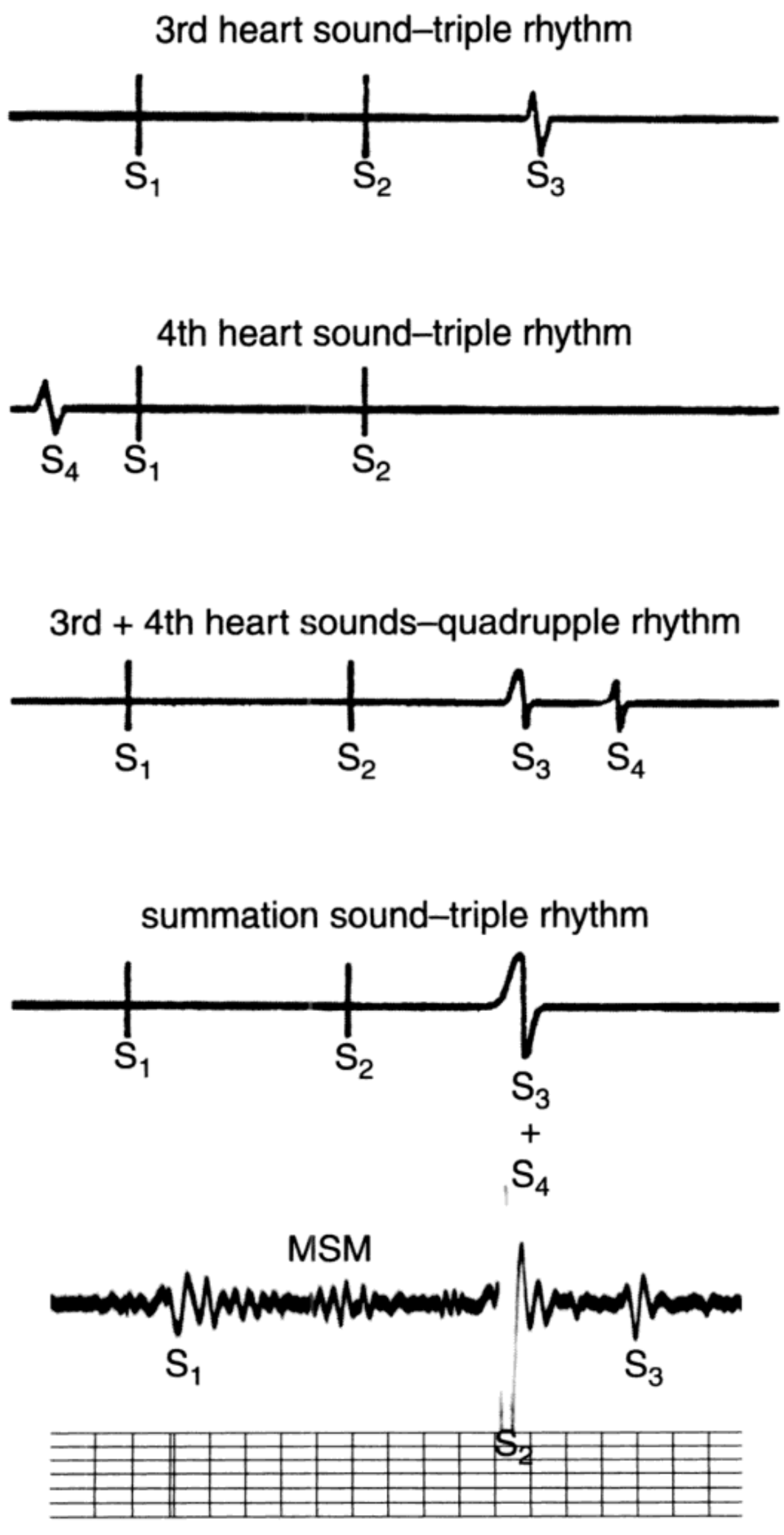


Figure 6–12 When the first and second heart sounds are accompanied by a third (S_3) and/or a fourth heart sound (S_4), the cadence of a triple or quadruple rhythm is produced. S_3 and S_4 sometimes summate (fourth tracing). The lowest tracing is a phonocardiogram recorded at the apex of a 5-year-old child with a normal third heart sound (S_3) and a trivial vibratory midsystolic murmur (MSM).

cadence is lost. It is then appropriate to use the term *abnormal third* or *abnormal fourth* heart sound rather than to say that gallop sounds exist without gallop rhythm.

How can one distinguish normal from abnormal third or fourth heart sounds? Generally by the company they keep rather than by distinctive auscultatory properties of the sounds *per se*. The abnormal filling sounds that cause gallop rhythms are not merely exaggerations of normal third and fourth heart sounds at relatively rapid heart rates, but instead result from the inappropriate presence of the sounds, whose intensities may, in fact, be soft rather than prominent. Children and young adults have normal (physiologic) *third* heart sounds (Fig. 6–12), but fourth heart sounds are abnormal. Normal third heart sounds sometimes persist beyond age 40 years, particularly in females. Thereafter, a third heart sound, especially in males, is presumptively abnormal. Fourth heart sounds are sometimes heard after exercise in healthy adults

without clinical evidence of heart disease, an observation that prompts the belief, still debated, that these fourth heart sounds are normal. In any event, there are no auscultatory differences that permit distinction between normal and abnormal third and fourth heart sounds. Further to the point, *normal* third or fourth heart sounds can create a triple rhythm cadence, but the term "gallop rhythm" is not appropriate because of its pathologic connotation. The term *normal third* or *normal fourth heart sound* should be used instead.

Third and fourth heart sounds originate in either the left or right ventricle (Fig. 6–3*B*). The fourth heart sound requires active atrial contribution to ventricular filling, so that fourth heart sounds disappear when coordinated atrial contractions cease, as in atrial fibrillation. When the atria and ventricles contract independently, as in complete heart block, fourth heart sounds or summation sounds occur randomly in diastole because the relationship between the P wave and QRS complex of the electrocardiogram is random. In light of the fact that third and fourth heart sounds are events of ventricular filling, obstruction of an atrioventricular orifice impedes ventricular inflow, and removes one of the prime preconditions for the generation of these sounds. The presence of a third or a fourth heart sound therefore implies an unobstructed atrioventricular valve on its side of origin.

Third and fourth heart sounds, whether normal or abnormal, are soft low-frequency events that require meticulous stethoscopic technique for detection. Potain called attention to this point:

> The sound is dull, much more so than the normal sound. It is a shock, a perceptible elevation, it is scarcely a sound. If one applies the ear to the chest, it affects the tactile sensation, more perhaps than the auditory sense. If one attempts to hear it with a flexible stethoscope, it lacks only a little, almost always, of disappearing completely.[13]

These soft, low-frequency sounds are best heard in a quiet room when the bell of the stethoscope is applied selectively over the left or right ventricular impulse with just enough pressure to form a skin seal. Firm pressure of the bell or use of the stethoscopic diaphragm damps low-frequency vibrations and reduces audibility of third and fourth heart sounds, or eliminates them altogether. However, these same physical properties can be used to advantage in distinguishing a split first heart sound (the two components of which are preserved or enhanced by the diaphragm or pressure of the bell) from a fourth heart sound preceding a split first heart sound (the fourth heart sound is damped by pressure while the first heart sound is not) (Fig. 6–13). This distinction was addressed by Potain, who asked

> Is the rhythm with which we are concerned nothing but a reduplication of the first heart sound? I believe this absolutely erroneous and for the following reasons. In the first place, the abnormal sound has, in no way, the timbre or usual characteristics of a valvular sound. Finally (and this is the unanswerable argument that makes unnecessary all other reasons), I have heard, in certain patients, successively and

or systemic hypertension in the left side of the heart, or the right ventricular hypertrophy of pulmonary stenosis or pulmonary hypertension in the right side of the heart. Fourth heart sounds are also common in ischemic heart disease, and are almost universal during an acute ischemic episode because the atrial "booster pump" is needed to assist a relatively stiff ischemic left ventricle maintain adequate contractile force.

A pacing catheter in the right ventricular apex may produce a presystolic pacemaker sound that is high-pitched and clicking (Fig. 6–14) and therefore different in pitch from a fourth heart sound. The consensus is that the pacemaker sound is extracardiac, resulting from contraction of chest wall muscle following spread of the electrical impulse from the pacemaker.

MURMURS—SYSTOLIC, DIASTOLIC, AND CONTINUOUS

A cardiovascular murmur is a relatively prolonged series of auditory vibrations characterized by intensity (loudness), frequency (pitch), configuration (shape), quality, duration, direction of radiation, and timing in the cardiac cycle. Once these features are established by auscultation, the stage is set for diagnostic conclusions that can be drawn from a murmur of a given description. I shall emphasize the clinical assessment, physiologic mechanisms, and interpretation of murmurs, rather than the physical principles that govern their production.

Intensity or loudness of a murmur is graded from 1 to 6 based upon the 1933 recommendations of Samuel A. Levine.[15,16] A *grade I murmur* is so faint that it is heard only after several seconds of auscultation. A *grade 2 murmur* is faint but is heard immediately. A *grade 3 murmur* is moderately loud. A *grade 4 murmur* is loud. A *grade 5 murmur* is very loud and can be heard with only the edge of the stethoscope in contact with the skin. A *grade 6 murmur* is so loud that it can be heard with the stethoscope just removed from the chest and not touching the skin.

Frequency or pitch of a murmur varies from high to low. The *configuration or shape* of a *systolic murmur* is characterized as crescendo, decrescendo, crescendo-decrescendo (diamond-shaped), plateau (even), or variable (uneven) (Fig. 6–15). *Quality* can be harsh, rough, rumbling, scratchy, buzzing, grunting, blowing, musical, whooping, squeaking, and so forth. *Duration* varies from short to long, with all gradations in between. A loud murmur radiates from its site of maximal intensity. The *direction* of radiation sometimes provides useful diagnostic information.

There are three basic categories of murmurs—*systolic*, *diastolic*, and *continuous*. A *systolic* murmur begins with or after the first heart sound and ends at or before the second heart sound on its side of origin (Fig. 6–16). A *diastolic* murmur begins with or after the second heart sound and ends before the subsequent first heart sound (Fig. 6–17). A *continuous* murmur begins in systole and continues without interruption through the timing of the second heart sound into all or part of diastole (Fig. 6–18).

The following classification of murmurs is based upon timing relative to the first and second heart sounds and is in accord with the 1967 recommendations of the Committee on Standardized Terminology of the American College of Cardiology.

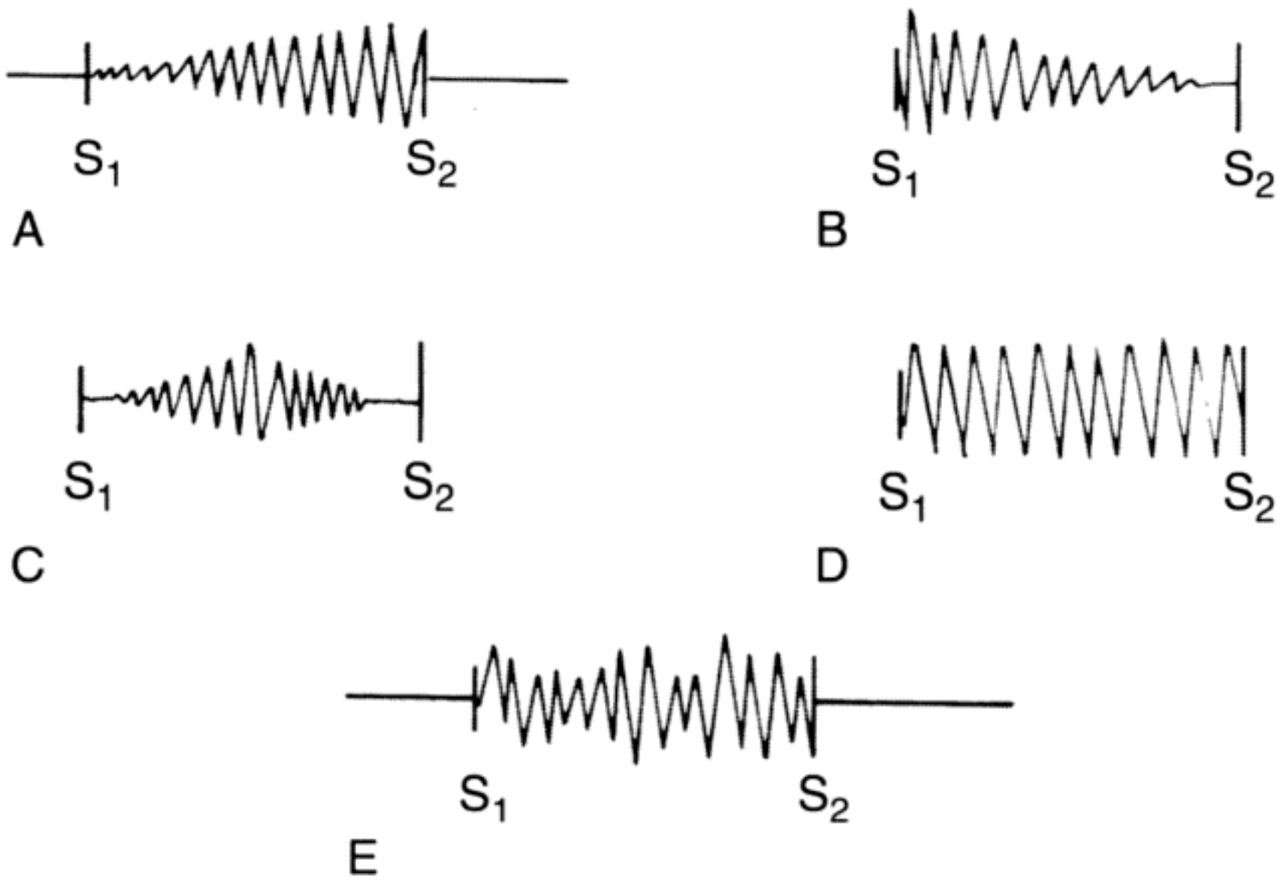


Figure 6–15 Five basic shapes or configurations of systolic murmurs: A—crescendo, B—decrescendo, C—crescendo-decrescendo, D—plateau (even-shaped), and E—variable (uneven).

Systolic Murmurs

Systolic murmurs are classified according to their time of onset and termination as mid-systolic, holosystolic, early systolic, or late systolic (Fig. 6–16). A *midsystolic murmur* begins after the first heart sound and ends before the second sound. Termination of the murmur is related to the component of the second heart sound on its side of origin (see Fig. 6–16). Midsystolic murmurs originating in the *left* side of the heart end before the

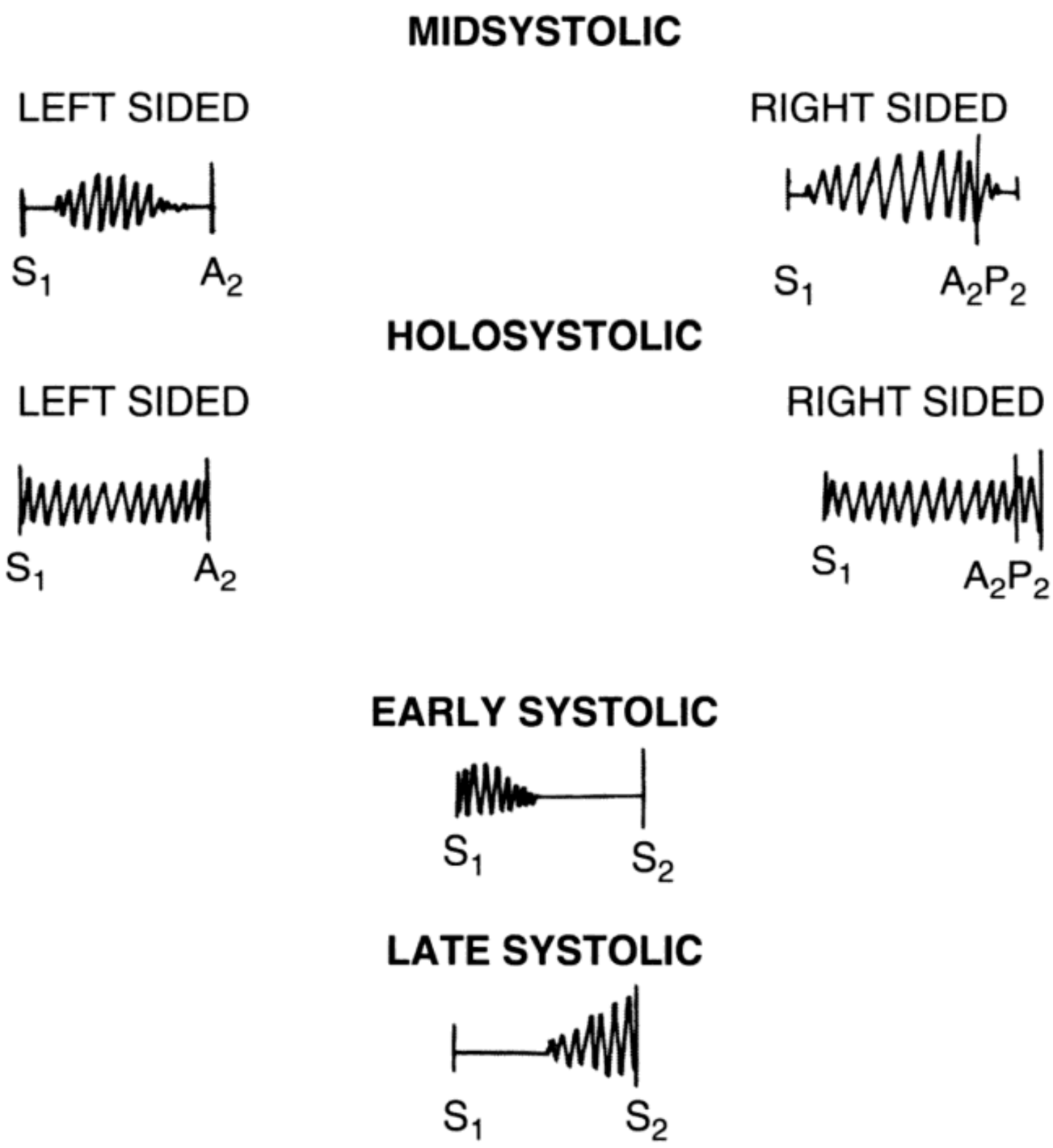


Figure 6–16 Systolic murmurs are classified according to their time of onset and termination as *midsystolic*, *holosystolic*, *early systolic*, and *late systolic*. Termination of the murmur is related to the relevant component of the second heart sound, ie, the aortic component (A_2) for systolic murmurs originating in the left side of the heart, and the pulmonary component (P_2) for systolic murmurs originating in the right side of the heart.

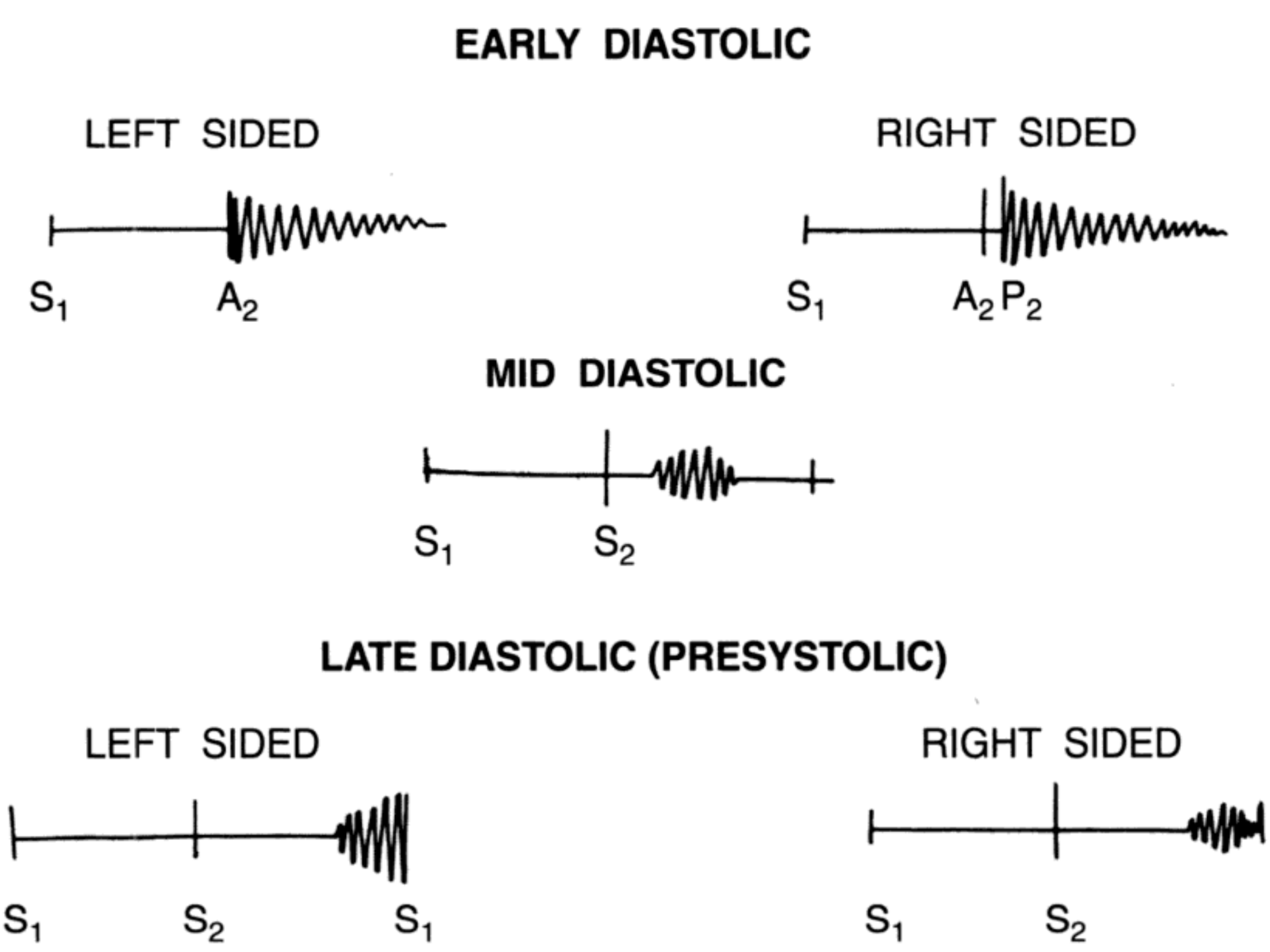


Figure 6–17 Left- or right-sided diastolic murmurs are classified according to their time of onset as *early diastolic*, *mid-diastolic*, or *late diastolic* (presystolic).

aortic component of the second sound, and midsystolic murmurs originating in the *right* side of the heart, end before the *pulmonary* component of the second sound. A *holosystolic murmur* begins with the first heart sound, occupies all of systole, and ends with the second heart sound on its side of origin (see Fig. 6–16). Holosystolic murmurs originating

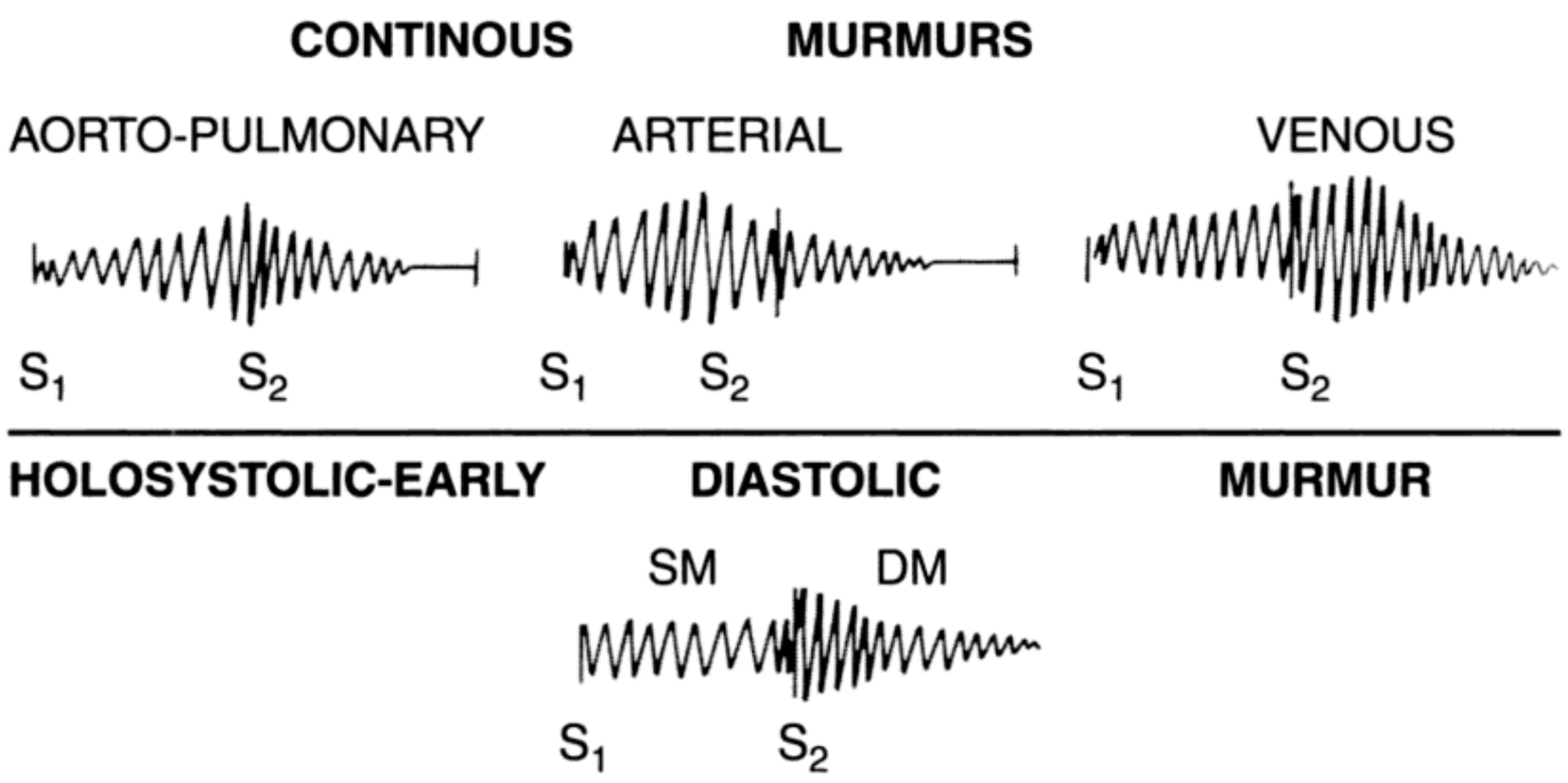


Figure 6–18 Continuous murmurs begin in systole and *continue without interruption* through the timing of the second heart sound (S_2) into *all* or *part* of diastole. Accordingly, continuous murmurs need not occupy an entire cardiac cycle. The continuous murmurs shown here are *aortopulmonary*, *arterial*, and *venous*. Note that a *holosystolic* murmur (SM) followed by a *holodiastolic* murmur (DM) is not continuous, but *two separate murmurs* that occupy all of systole and all of diastole.

in the *left* side of the heart should be related to the *aortic* component of the second sound, and holosystolic murmurs originating in the *right* side of the heart should be related to the *pulmonary* component of the second sound (see Fig. 6–16).

The term "regurgitant systolic murmur," originally applied to murmurs that occupied all of systole, has fallen into disuse because regurgitant flow can be accompanied by murmurs that are midsystolic, early systolic, or late systolic. Similarly, the term "ejection systolic murmur" originally applied to midsystolic murmurs, has fallen into disuse because midsystolic murmurs are not necessarily caused by "ejection."

Midsystolic Murmurs

Midsystolic murmurs (See Figs. 6–15 and 6–16) occur in five settings: (1) obstruction to ventricular outflow, (2) dilatation of the aortic root or pulmonary trunk, (3) accelerated systolic flow into the aorta or pulmonary trunk, (4) innocent midsystolic murmurs, including those resulting from morphologic changes in functionally normal semilunar valves (generally aortic), and (5) some forms of mitral regurgitation. The physiologic mechanism of *outflow* midsystolic murmurs reflects phasic flow across the left or right ventricular outflow tract as originally described by Leatham[17] (Fig. 6–19). Following isovolumetric contraction and the generation of the first heart sound, the rise in ventricular pressure opens the aortic and pulmonary valves. Forward flow commences, and the murmur begins. As flow proceeds, the murmur increases in crescendo; as flow declines, the murmur decreases in decrescendo, ending before ventricular pressure drops below central arterial pressure, at which time the aortic and pulmonary valves close, generating their respective components of the second heart sound (Fig. 6–19).

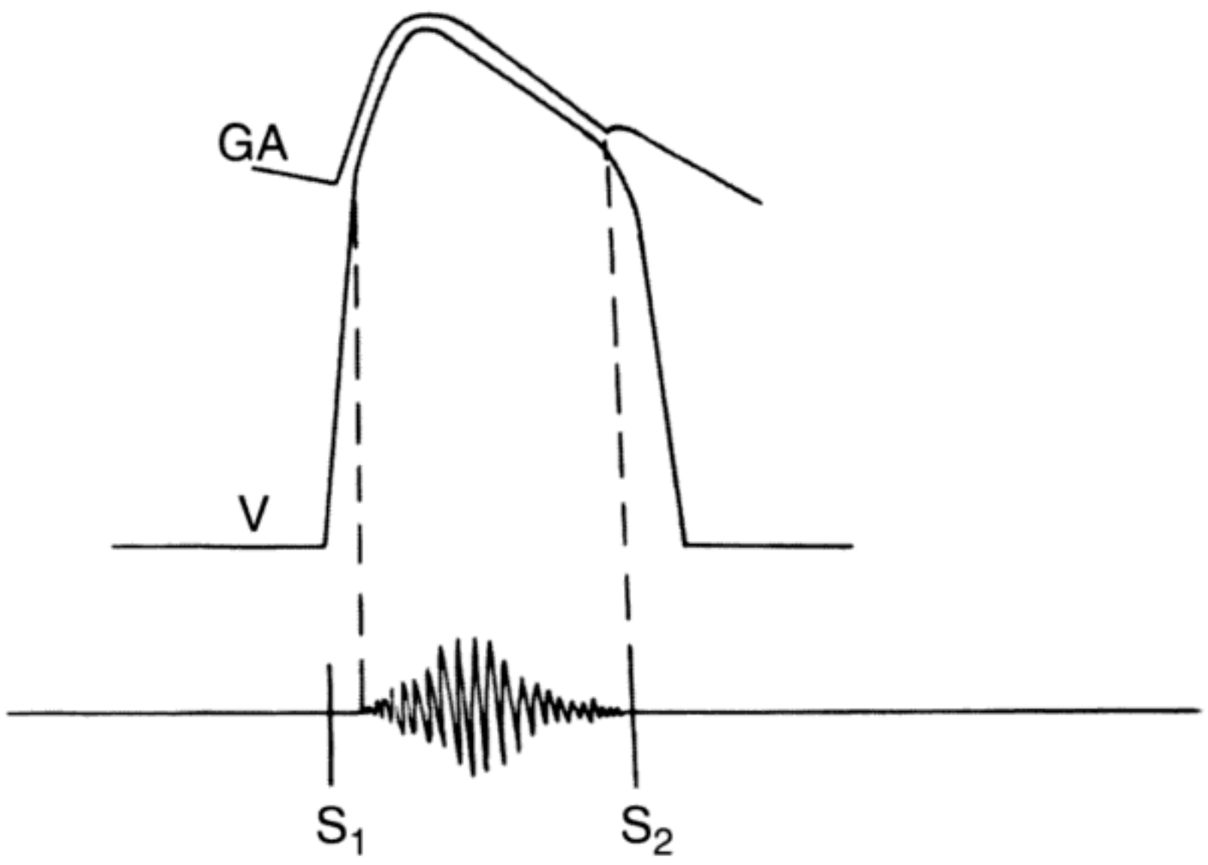


Figure 6–19 Illustration of the physiologic mechanism responsible for the configuration of an outflow midsystolic murmur into the aortic root or pulmonary trunk. Great arterial (GA) and ventricular (V) pressure pulses are shown with a representative phonocardiogram. The midsystolic murmur begins after the first heart sound (S_1) as ventricular pressure exceeds great arterial pressure. The murmur rises in crescendo to a peak as flow proceeds, then declines in decrescendo as flow declines. The murmur ends before the second heart sound (S_2) as ventricular pressure falls below the great arterial pressure.

Aortic valve stenosis is associated with the prototypical midsystolic murmur that can have an early systolic peak and a short duration, a relatively late peak and a prolonged duration, or all gradations in between. Whether long or short, however, the murmur retains its symmetric diamond shape, beginning after the first heart sound or with an ejection sound, rising to a systolic crest, and declining in decrescendo to end before the aortic component of the second hart sound (Fig. 6–16) (midsystolic, left-sided). The typical murmur of aortic valve stenosis is loudest in the second right intercostal space with radiation upward, to the right and into the neck because of the high-velocity jet within the aortic root.

An important variation is "Gallavardin dissociation" that occurs in older adults with fibrocalcific changes in previously normal trileaflet aortic valves that are either stenotic (obstruction) or sclerotic (no obstruction). The accompanying murmur in the second right interspace is harsh, noisy and impure, whereas the murmur over the left ventricular impulse is pure high-frequency and musical (Fig. 6–20*A*,*B*). The right basal murmur originates within the ascending aorta because of turbulence caused by the high-velocity jet (Fig. 6–20*A*). The musical murmur originates within the left ventricular cavity (Fig. 6–20*C*) and is ascribed to high-frequency periodic vibrations of aortic cusps that are fibrocalcific but without commissural fusion (Fig. 6–20*D*). These two distinctive midsystolic murmurs—the noisy right basal and the musical apical—were described by Louis Gallavardin in 1925,[18] and the designation "Gallavardin dissociation" is still used. The musical apical midsystolic murmur can be strikingly loud. William Stokes (1855) reported that such a murmur was heard at a distance of three feet from the chest, and that "this gentleman once observed to me that his entire body was one humming top."[19]

The pure, high-frequency apical midsystolic murmur of aortic stenosis or sclerosis must be distinguished from the high-frequency apical murmur of mitral regurgitation, a distinction that may be difficult or impossible to establish because the two murmurs may coexist. If the aortic component of the second heart sound is well heard at the base, but inaudible at the apex, the second sound may be buried in the late systolic vibrations of an apical holosystolic murmur of mitral regurgitation. However, the aortic valve may be immobile (calcified), and its closure sound soft or inaudible, so the length and configuration of the apical murmur cannot be timed with the aortic component of the second heart sound. When premature ventricular contractions are followed by pauses longer than the dominant cycle length, the apical midsystolic murmur of aortic stenosis or sclerosis increases in intensity in the long cycle length beat following the premature contraction (Fig. 6–21*A*), whereas the murmur of mitral regurgitation remains relatively unchanged in intensity (Fig. 6–21*B*). The same patterns prevail following longer cycle lengths in atrial fibrillation. The validity of these observations assumes that apical aortic and mitral murmurs do not coexist.

A midsystolic murmur originating in the right ventricular outflow tract is represented by the murmur of pulmonary valve stenosis (see Fig. 6–6*A*). The murmur begins after the first heart sound or with an ejection sound, rises in crescendo to a peak, and then declines in decrescendo to end before a delayed pulmonary component of the second heart sound (see Figs. 6–6*A* and 6–16) (midsystolic, right-sided). The murmur is maximal in the second left interspace with radiation upward and to the left, and when

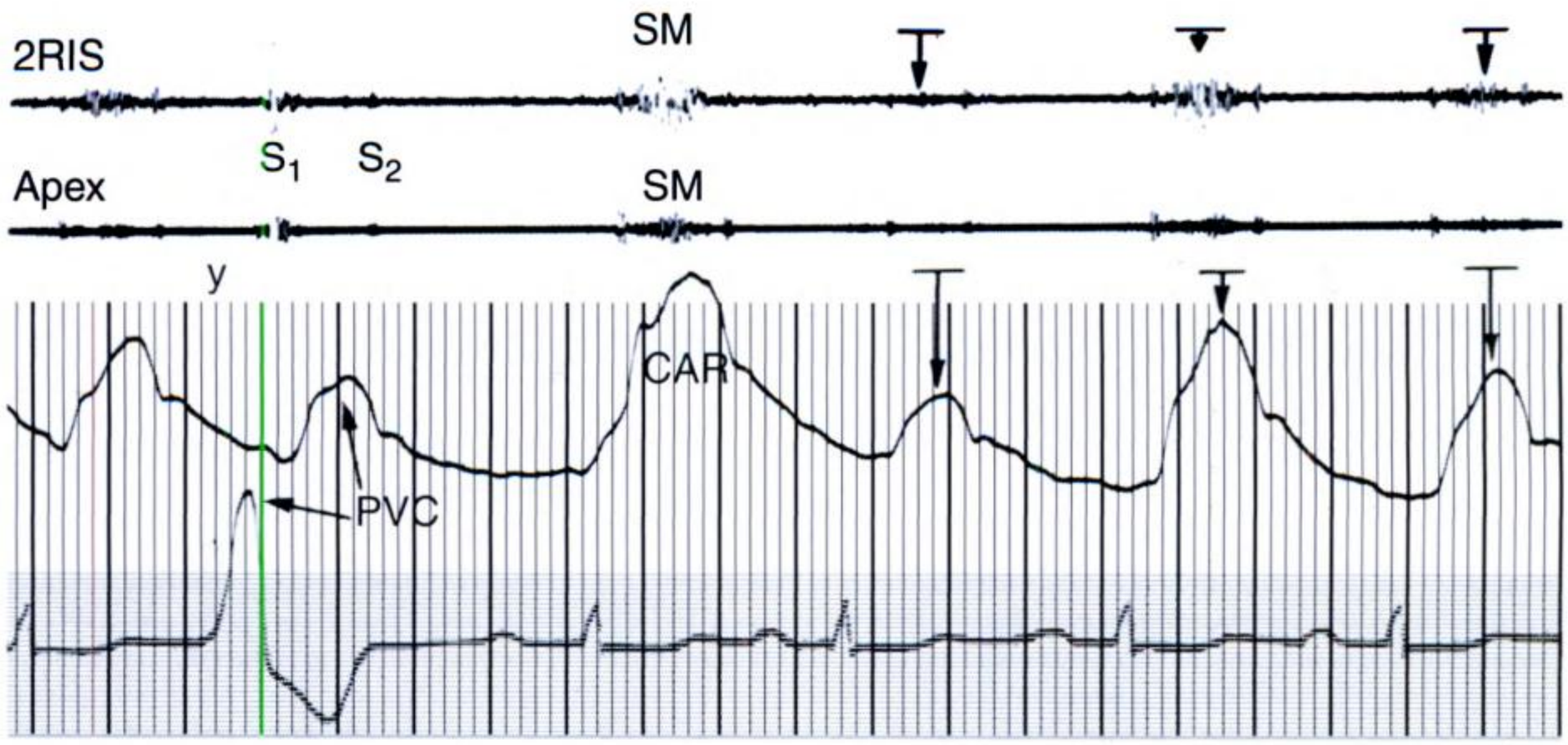


Figure 6–23 Phonocardiograms at the second right interspace (2RIS) and apex from a patient with a sclerotic aortic valve. A premature ventricular contraction (PVC) is followed by a compensatory pause and striking amplification of the midsystolic murmur (SM), after which the murmur alternates in intensity in synchrony with alternation of the carotid (CAR) pulse.

It is important to point out that some forms of *mitral regurgitation* generate midsystolic murmurs. The physiologic mechanism responsible for the midsystolic murmur of mitral regurgitation (usually associated with left ventricular wall motion abnormalities of ischemic heart disease) reflects early systolic *competence* of the valve followed by midsystolic *incompetence*, then a late systolic decrease in regurgitant flow. In any event, this is not an "ejection murmur" despite its configuration.

In elderly patients with depressed left ventricular systolic function, pulsus alternans following a premature ventricular contraction is often accompanied by alternation of the midsystolic aortic sclerotic murmur (Fig. 6–23).

An aortic stenotic murmur in patients with normal thoracic configuration is best heard at the right base (Fig. 6–24*A*). In patients with the barrel chest of pulmonary emphysema, this aortic stenotic murmur may be virtually inaudible in any thoracic location, but clearly heard over the carotids and in the suprastemal notch (Fig. 6–24*B*). Cephalad radiation is reinforced when the jet from a stenotic aortic valve is directed laterally, adheres to the aortic wall, and propagates cephalad—the Coanda effect—(Fig. 6–25), thus increasing the intensity of the murmur above the clavicles and in the suprasternal notch. Auscultation in the neck should be routine, especially but not only in elderly patients with increased anteroposterior chest dimensions (Fig. 6–24*B*).

Holosystolic Murmurs

Holosystolic (Greek *holos*, "entire") murmurs (see Figs, 6–15 and 6–16) begin with the first heart sound and occupy all of systole, going up to the second sound on their side of origin.[16] Holosystolic murmurs are generated by flow from a chamber or vascular bed whose pressure or resistance *throughout* systole is higher than the pressure or resistance in

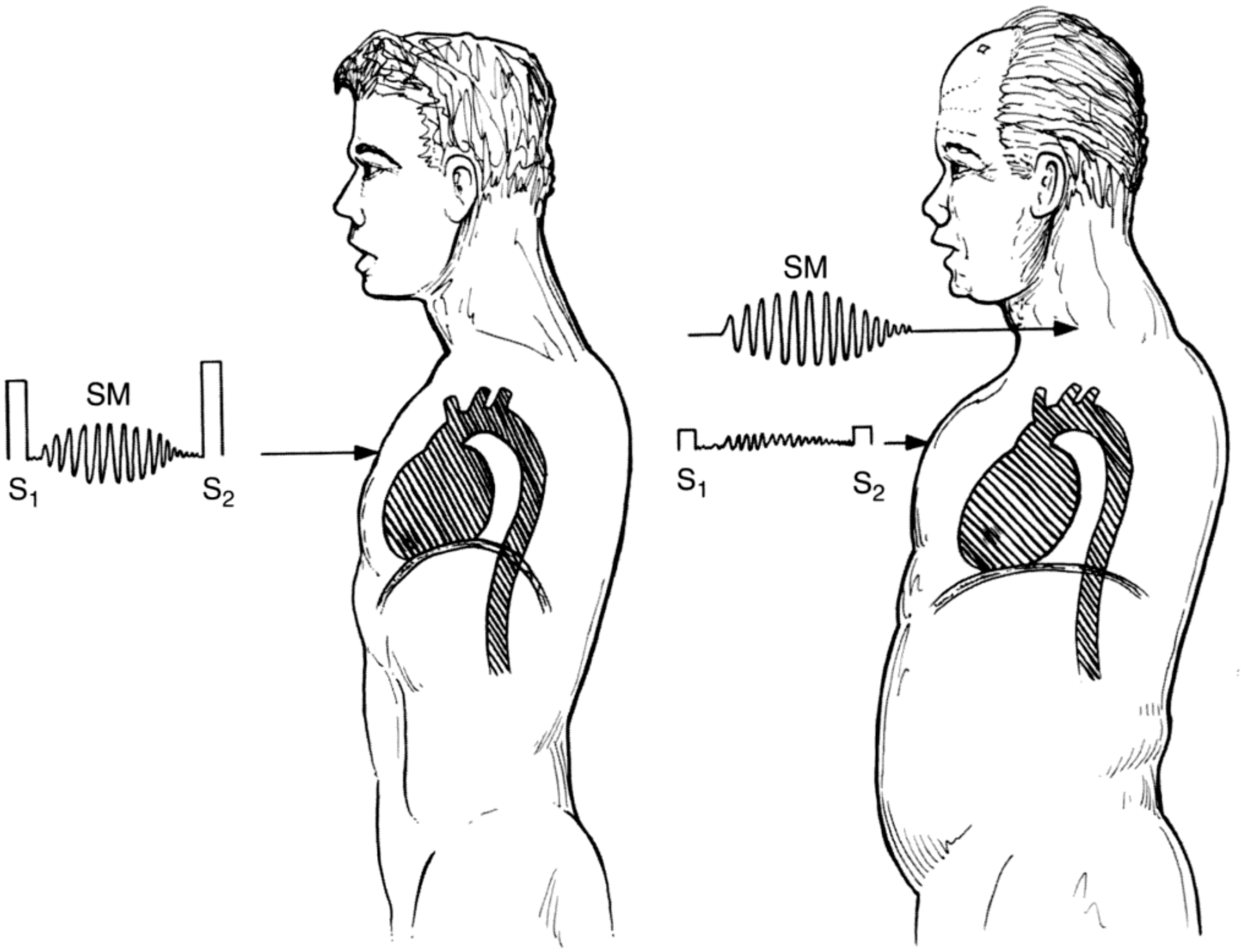


Figure 6–24 *A,* With normal body habitus, the aortic stenotic murmur (SM) is best heard at the right base. *B,* With the barrel chest of pulmonary emphysema, the aortic stenotic murmur (SM) is virtually inaudible in the thoracic locations, but clearly audible over the carotids and in the suprastemal notch.

the chamber or vascular bed receiving the flow. Holosystolic murmurs in the left side of the heart are represented by mitral regurgitation, in the right side of the heart by high-pressure tricuspid regurgitation, between the ventricles by restrictive ventricular septal defects, and between the great arteries by an aortopulmonary window or patent ductus arteriosus when elevated pulmonary vascular resistance eliminates the diastolic shunt and the diastolic portion of the continuous murmur (see below).

The timing of a holosystolic murmur within the framework of the first and second heart sounds reflects the physiologic and anatomic mechanisms responsible for its genesis. Figure 6–26 illustrates the mechanism of the holosystolic murmur of mitral regurgitation or high-pressure tricuspid regurgitation. Ventricular pressure exceeds atrial pressure from the very onset of systole, so regurgitant flow and murmur begin with the first heart sound. The murmur persists up to or slightly beyond the relevant component of the second heart sound, provided that ventricular pressure at end-systole exceeds atrial pressure and provided that the atrioventricular valve remains incompetent.

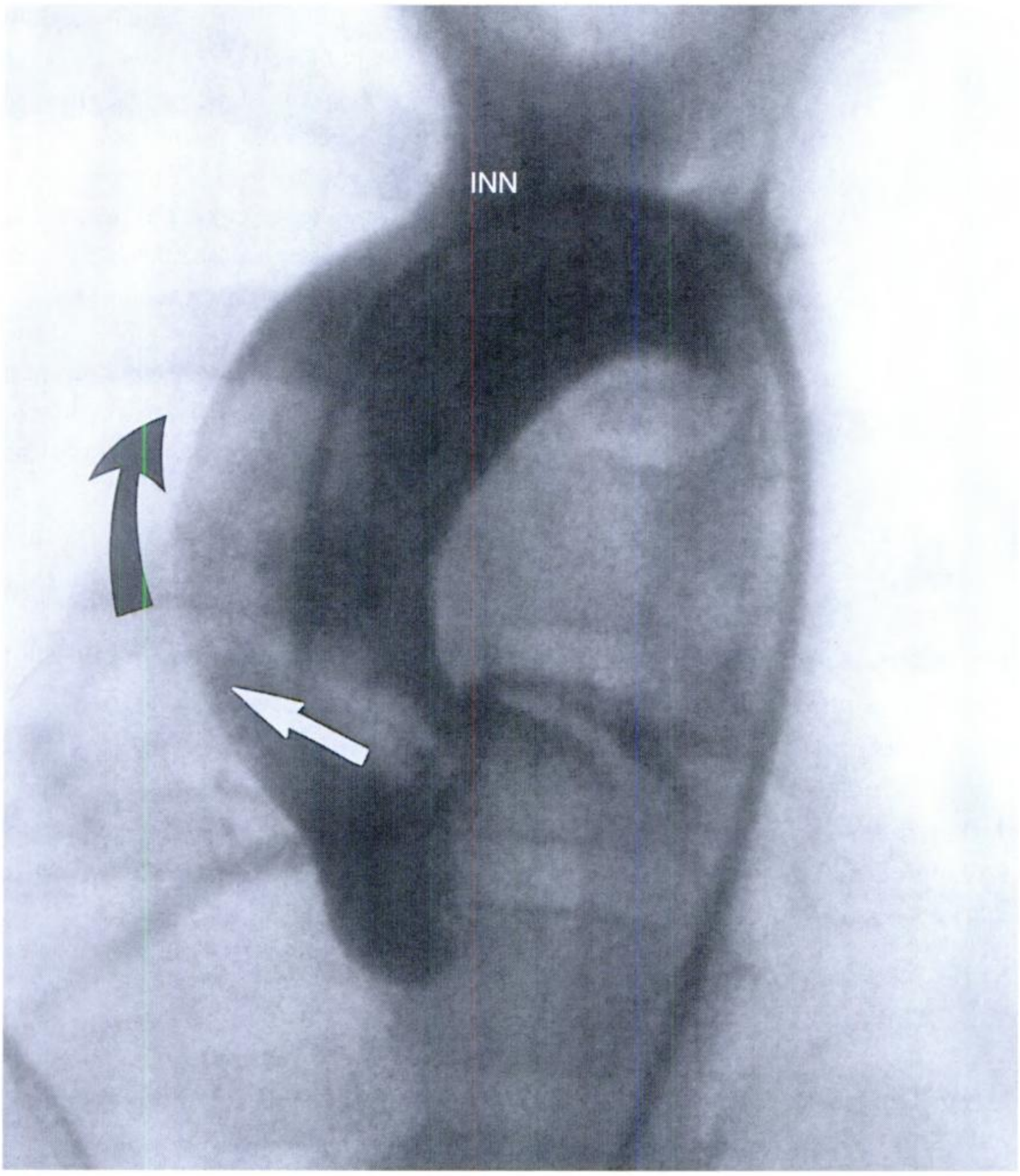


Figure 6–25 Aortogram illustrating the Coanda effect in a patient with bicuspid aortic stenosis. (Henri Marie Coandă, a Romanian inventor). The jet is directed laterally (*white arrow*), adheres to the lateral aortic wall (*curved black arrow*), and is carried cephalad toward the orifice of the innominate artery (INN). The Coanda effect is more likely to occur because of the hourglass narrowing of supravalvular aortic stenosis.

When flow generating the holosystolic murmur of mitral regurgitation is directed posterolaterally within the left atrial cavity, the murmur radiates into the axilla, toward the angle of the left scapula, and to the vertebral column, with bone conduction conveying the murmur from the cervical to the lumbar spine (Fig. 6–27*A*). When the direction of the intra-atrial jet is forward and medial toward the atrial septum near the base of the aorta, the regurgitant murmur radiates to the left sternal edge, to the base, and even into the neck (Fig. 6–27*B*).

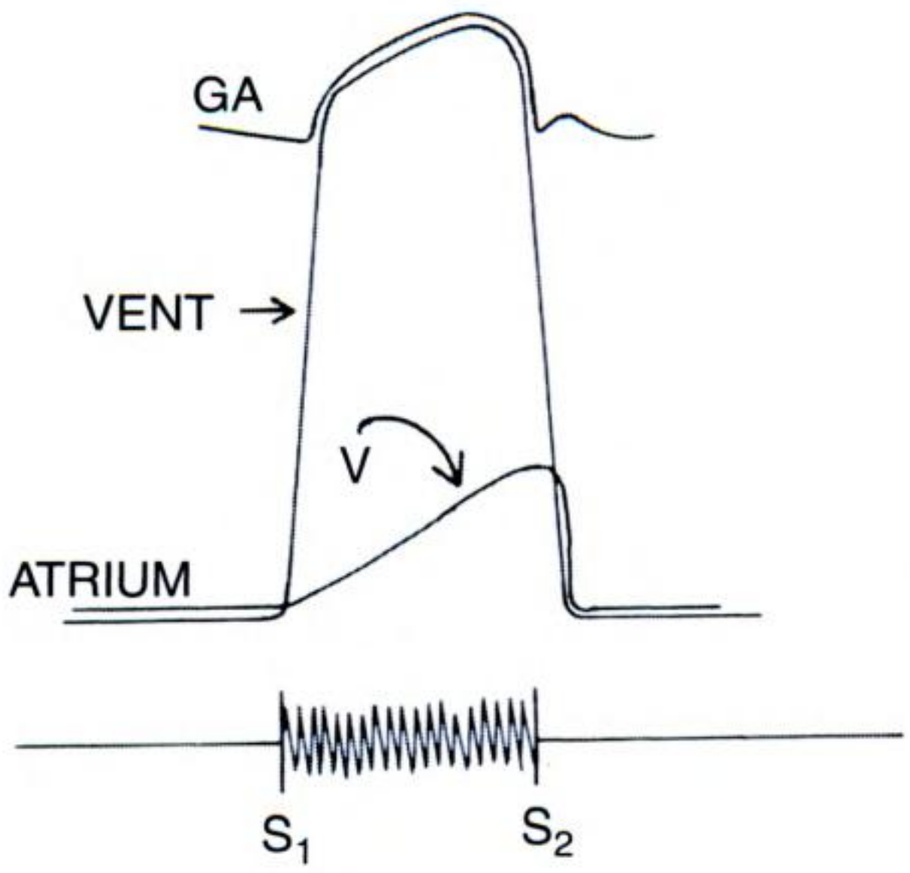


Figure 6–26 Illustration of great arterial (GA), ventricular (VENT), and atrial pressure pulses with phonocardiogram, showing the physiologic mechanism of holosystolic murmurs commonly heard in mitral and tricuspid regurgitation. Ventricular pressure exceeds atrial pressure at the onset of systole, so regurgitant flow and murmur commence with the first heart sound (S_1). The murmur persists up to or slightly beyond the second heart sound (S_2) because regurgitation persists to the end of systole at which time left ventricular pressure still exceeds left atrial pressure. V = atrial v wave.

The murmur of tricuspid regurgitation is holosystolic when right ventricular systolic pressure is substantially elevated as illustrated in Figure 6–26. A distinctive and diagnostically important feature of the murmur of tricuspid regurgitation is its increase during inspiration—Carvallo's sign[21] (José Manuel Rivero–Carvallo, National Cardiological Institute, Mexico City). The tricuspid systolic murmur may be audible *only* during inspiration. The increase occurs because the inspiratory augmentation in right

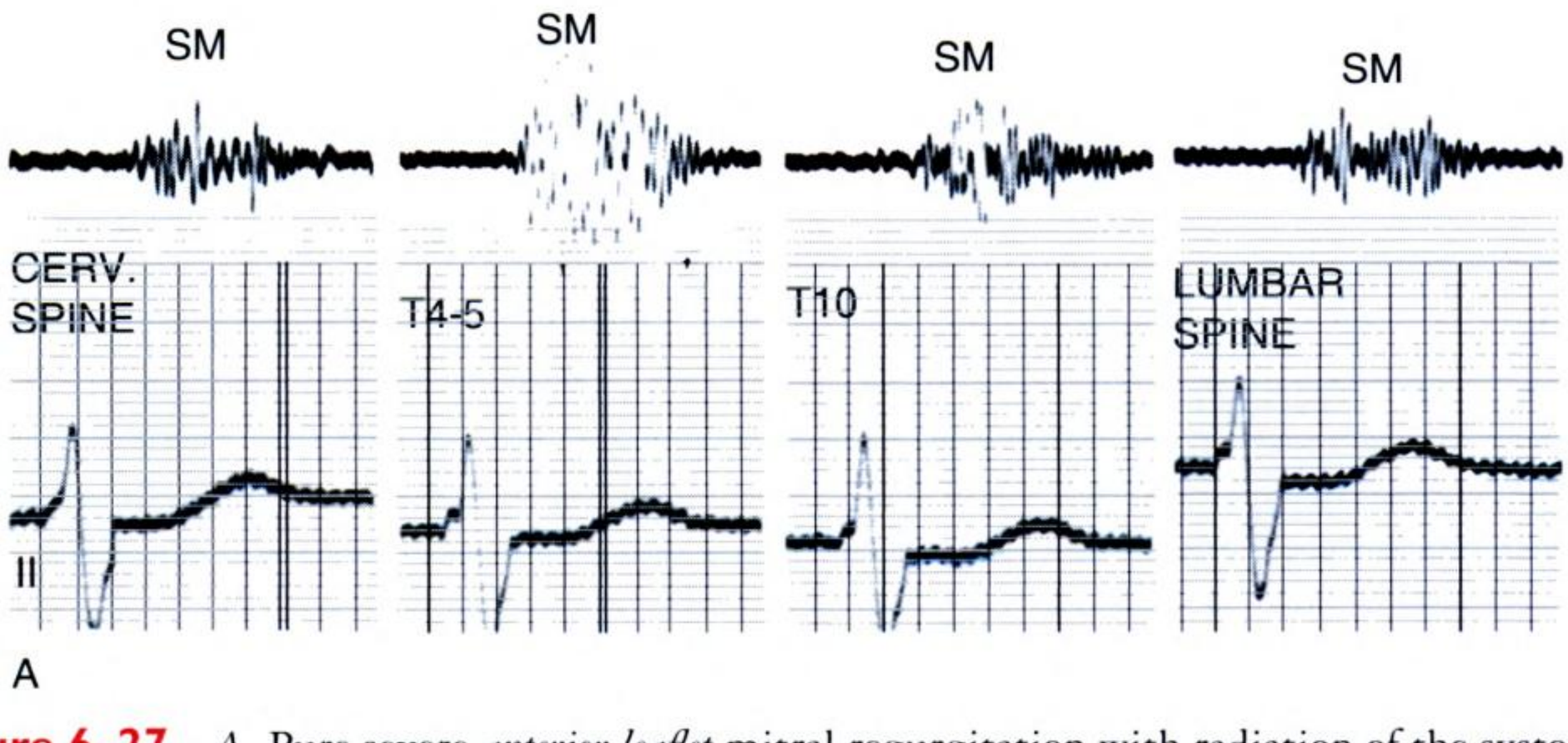
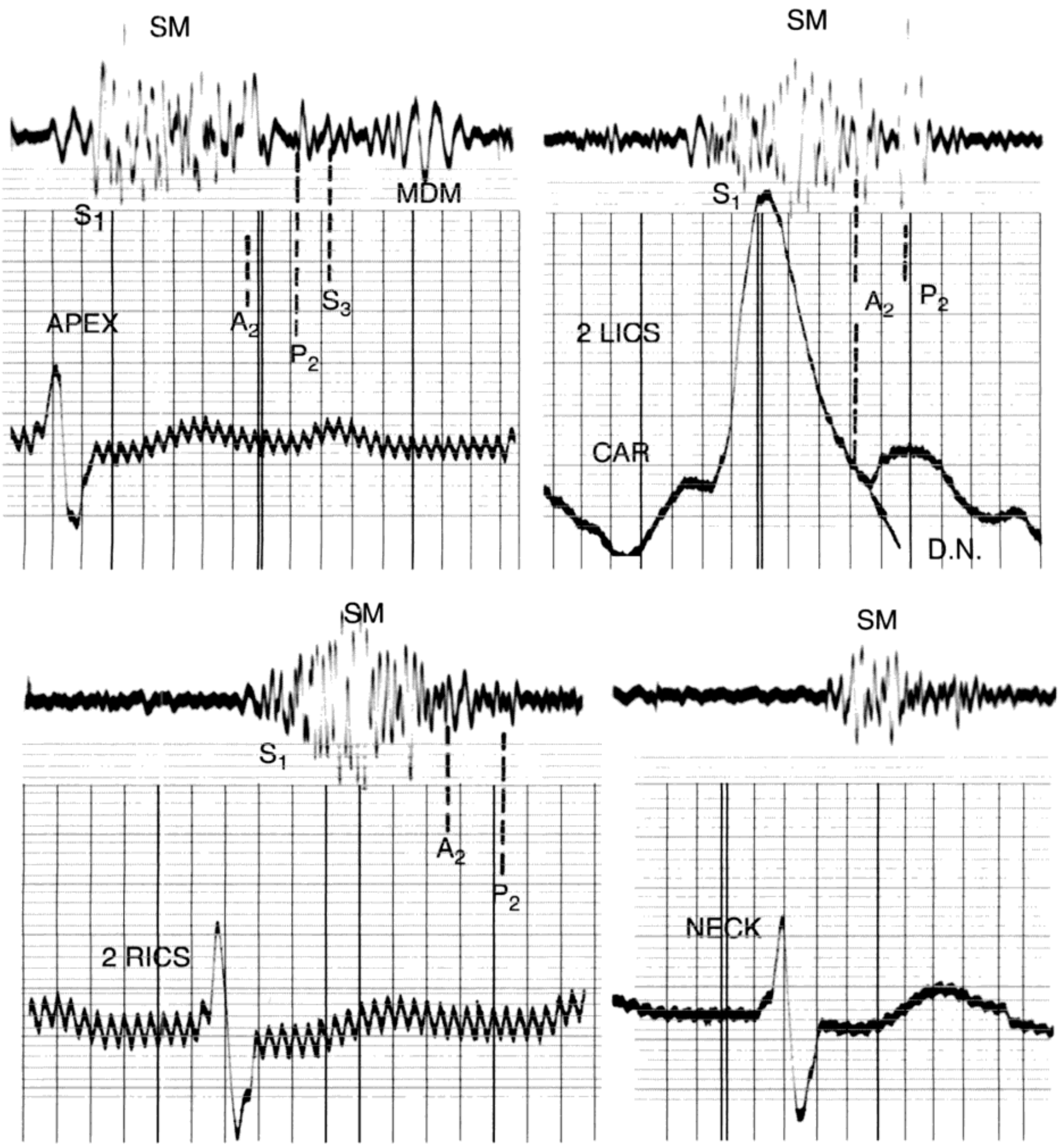


Figure 6–27 *A,* Pure severe *anterior leaflet* mitral regurgitation with radiation of the systolic murmur (SM) to the back with bone conduction from the cervical to the lumbar spine. *B,* Pure severe *posterior leaflet* mitral regurgitation with radiation of the systolic murmur (SM) to the base of the heart (2 LICS, 2 RICS, second left, and second fight intercostal spaces), and into the neck. The holosystolic murmur (SM) at the apex is followed by wide splitting of the second heart sound because of premature closure of the aortic valve associated with mitral regurgitation. The third heart sound (S_3) and the prominent but short mid-diastolic murmur (MDM) resulted from rapid diastolic flow into the left ventricle. The aortic component of the second heart sound (A_2) coincides with the dicrotic notch (DN) of the carotid pulse. *Continued*

ventricular volume is converted into an increase in the quantity and velocity of regurgitant flow. When the right ventricle fails, this capacity is lost, and so is Carvallo's sign.

The murmur of a restrictive ventricular septal defect is holosystolic because left ventricular systolic pressure and systemic vascular resistance exceed right ventricular systolic pressure and pulmonary vascular resistance from the onset to the end of systole. Holosystolic murmurs occur with large aortopulmonary connections (an aortopulmonary window or patent ductus arteriosus) when a rise in pulmonary vascular resistance abolishes the diastolic portion of the continuous murmur, leaving a murmur that is holosystolic or nearly so.



B

Figure 6–27 *Continued*

Early Systolic Murmurs

Early systolic murmurs (see Figs. 6–15 and 6–16) have their onset with the first heart sound, diminish in decrescendo, and end well before the second heart sound, generally before or at midsystole.[16] Certain types of mitral regurgitation, tricuspid regurgitation, or ventricular septal defects are examples.

Acute severe mitral regurgitation is accompanied by an early systolic murmur or a decrescendo murmur that diminishes or ends before the second heart sound (Fig. 6–28*A,B*). The physiologic mechanism responsible for a murmur of this configuration is regurgitation into a normal-sized left atrium whose distensibility is limited. A steep rise in left atrial V wave approaches left ventricular pressure at end-diastole; a late systolic decline in left ventricular pressure favors this tendency (Fig. 6–28*A,B*). The stage is set for regurgitant flow that is maximal in early systole and minimal in late systole. The systolic murmur parallels this pattern, declining or vanishing before the second heart sound (Fig. 6–28).

An early systolic murmur is also a feature tricuspid regurgitation with *normal* right ventricular systolic pressure) (Fig. 6–28*A*). The regurgitation accompanying tricuspid valve infective endocarditis in drug abusers is a case in point. The mechanisms responsible for the timing and configuration of these murmurs are analogous to the mechanisms described in the preceding paragraph and illustrated in Figure 6–28*A*. A tall right atrial V wave reaches normal right ventricular pressure in latter systole, so the regurgitation and the murmur are chiefly, if not exclusively, *early* systolic and medium or low-frequency because normal right ventricular systolic pressure generates comparatively low velocity regurgitant flow. This is in contrast to the high-frequency holosystolic murmur that accompanies tricuspid regurgitation with *elevated* right ventricular systolic pressure (see above).

Early systolic murmurs occur with ventricular septal defects under two widely divergent anatomic and physiologic circumstances. A soft, pure, high-frequency, early systolic murmur localized to the mid or lower left sternal edge is typical of a very small ventricular septal defect, because the shunt is confined to early systole (Fig. 6–29). A murmur of similar timing and configuration occurs through a nonrestrictive ventricular septal defect when elevated pulmonary vascular resistance decreases or abolishes late systolic shunting.

Late Systolic Murmurs

Late systolic applies to the murmur (Figs. 6–15 and 6–16) of mitral valve prolapse which begins in mid to late systole and proceeds up to the aortic component of the second heart sound (see Fig. 6–7*A*). One or more mid to late systolic clicks typically introduce the murmur (see Fig. 6–7). The responses of the late systolic murmur and clicks of mitral valve prolapse to postural maneuvers (see earlier discussion) are illustrated in Figure 6–8. A decrease in left ventricular volume readily achieved by prompt standing after squatting (see Fig. 6–30) and by the Valsalva maneuver, causes the late systolic murmur to lengthen and soften. An *increase* in left ventricular volume associated with squatting (Fig. 6–8) or sustained handgrip causes the murmur to shorten and become louder. Pharmacologic

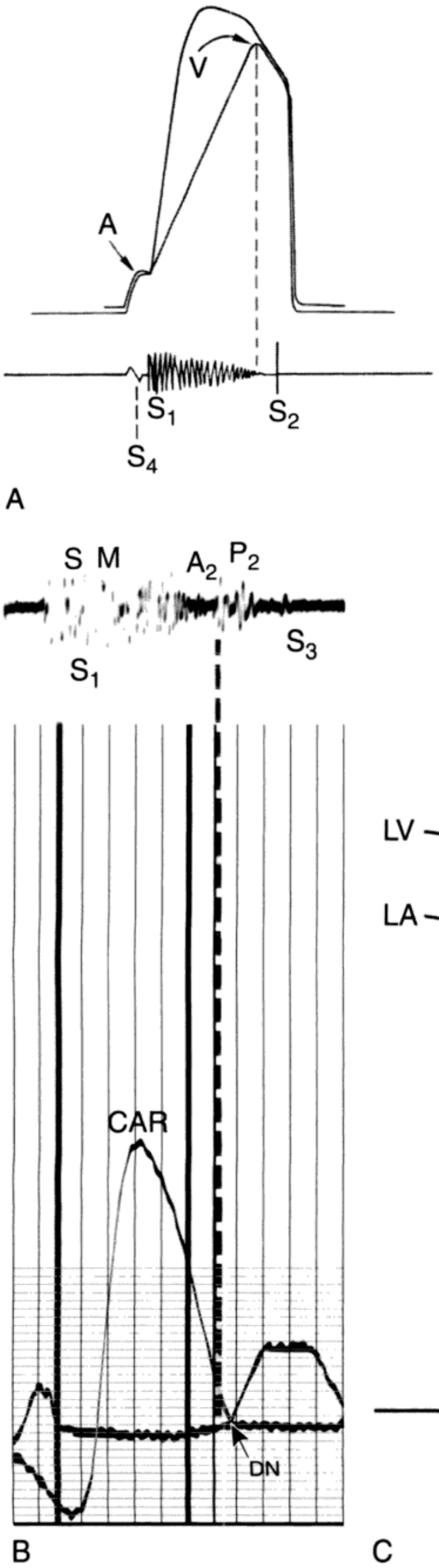


Figure 6–28 *A,* Ventricular and atrial pressure pulses with phonocardiogram schematically illustrating the mechanism of the *early* systolic murmur of acute severe mitral regurgitation or low-pressure tricuspid regurgitation. The V wave reaches ventricular pressure at end-systole (*upper curved arrow*), so regurgitant flow diminishes or ceases. Accordingly, the murmur is early systolic decrescendo, paralleling the hemodynamic pattern of regurgitant flow. The fourth heart sound (S_4) coincides with augmented atrial contraction that generates the A wave in the ventricular pressure pulse. *B,* Phonocardiogram recorded over the left ventricular impulse of a patient with acute severe mitral regurgitation resulting from ruptured chordae tendineae. An early systolic decrescendo murmur (SM) diminishes if not ends before the aortic component (A_2) of the second heart sound. P_2, the pulmonary component, is loud because of elevated pulmonary arterial pressure, and is transmitted to the apex. S_1, first heart sound; S_3, third heart sound; CAR carotid pulse. The vertical dotted line times A_2 with the dicrotic notch (DN). *C,* Schematic left ventricular (LV) and left atrial (LA) pressure pulses with phonocardiogram showing the relationship between the decrescendo configuration of the early systolic murmur and late systolic approximation of the tall left atrial V wave and the left ventricular pressure that declines in latter systole because forward flow cannot be sustained (*curved arrow*). Regurgitant flow diminishes or ceases in latter systole, so the murmur is early systolic and decrescendo, paralleling the hemodynamic pattern of regurgitant flow as illustrated.

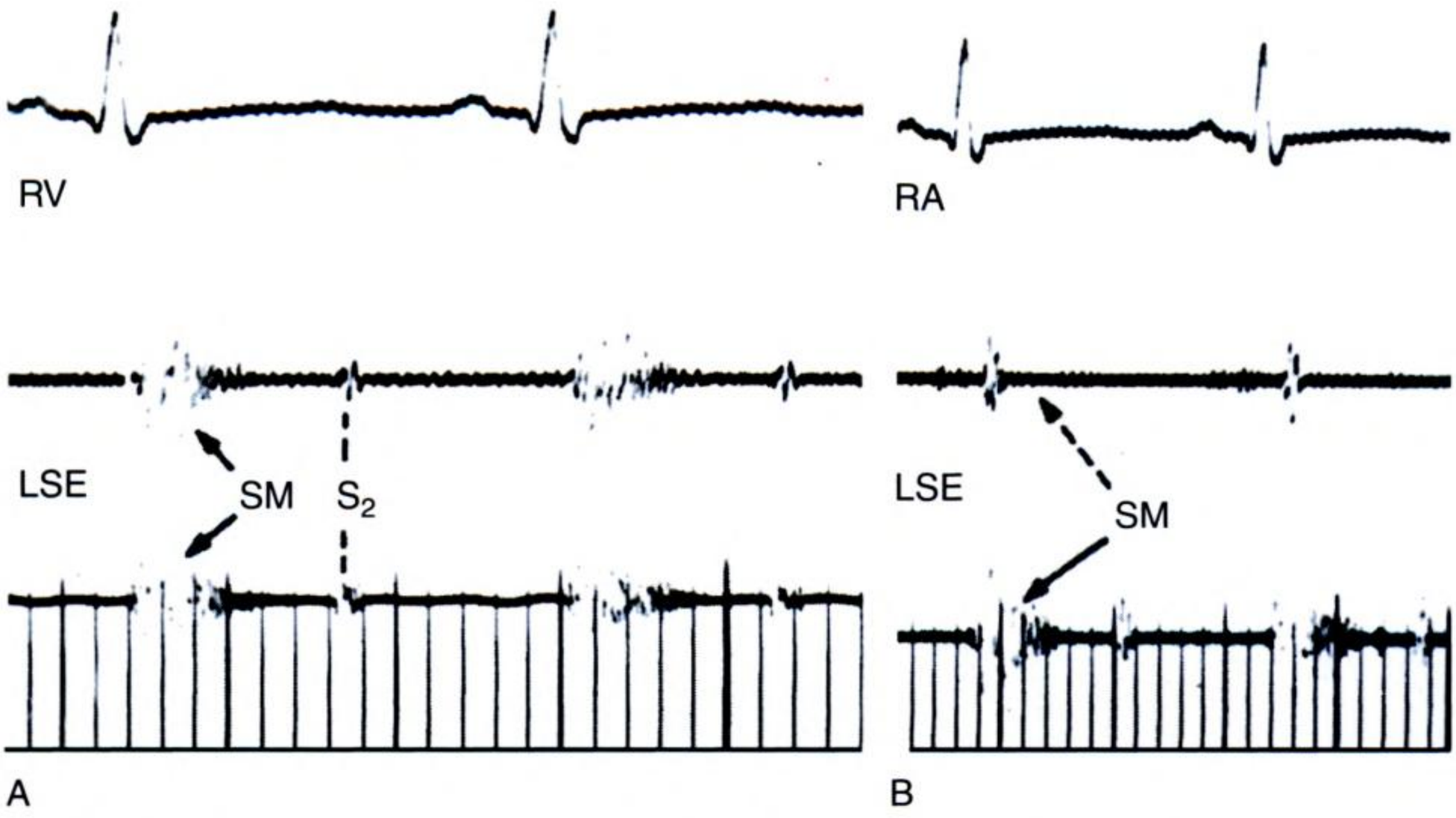


Figure 6–29 Phonocardiogram from a 4-year-old girl with a small ventricular septal defect, and a trivial early systolic shunt. *A,* A soft, pure, high-frequency early systolic decrescendo murmur (SM) is recorded *within* the right ventricle (RV) and simultaneously at the lower left sternal edge (LSE). S_2 second heart sound. *B,* When the intracardiac microphone was withdrawn into the right atrium (RA), the murmur vanished.

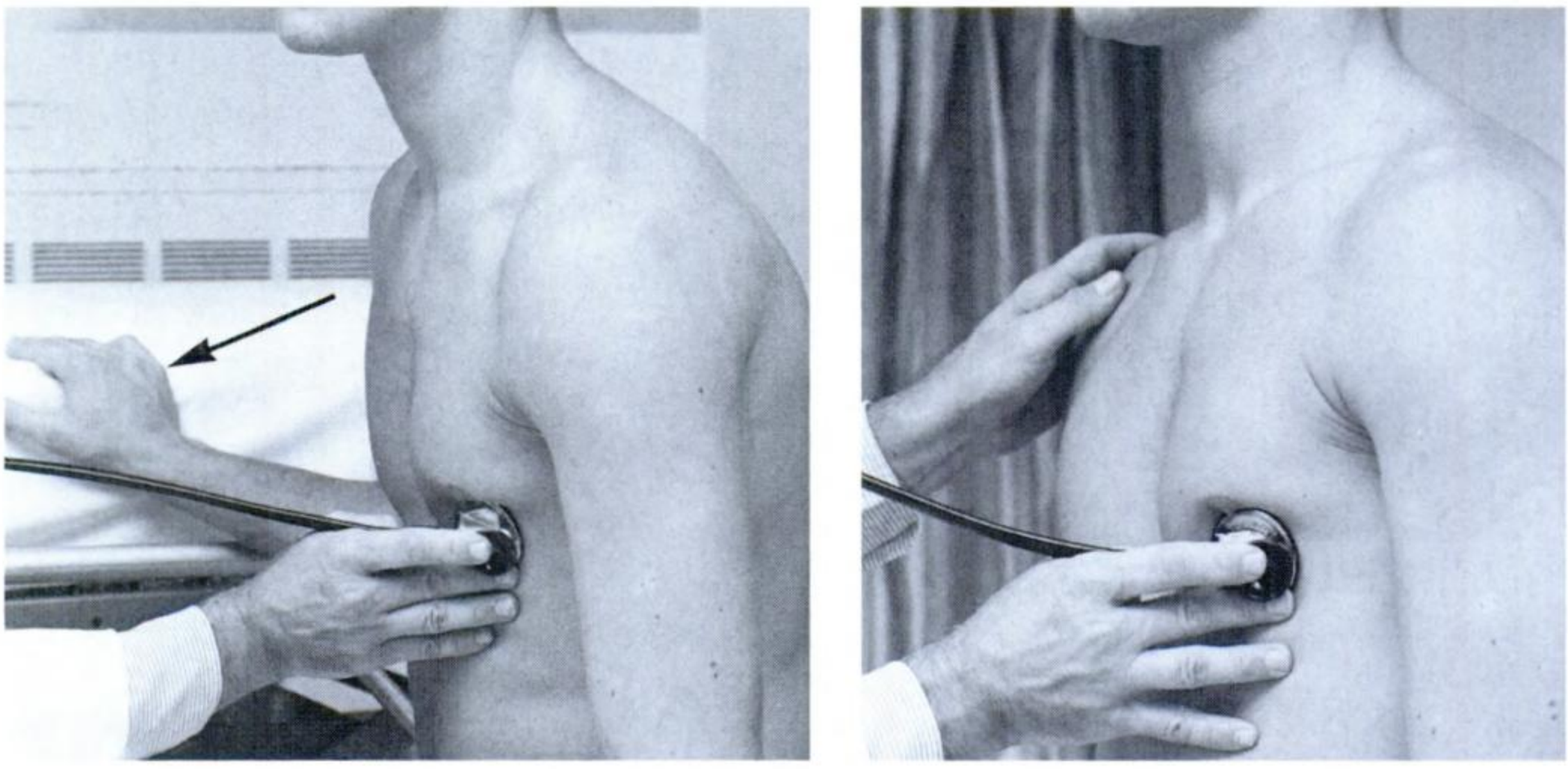


Figure 6–30 Auscultation while squatting (A), and after promptly standing (B). The right hand is used for support by holding the edge of the bed or examining table (A *arrow*).

interventions that alter left ventricular volume (Fig. 6–7*B,C*), especially amyl nitrite, produce analogous results but are less practical at the bedside.

The late systolic murmur of mitral valve prolapse is occasionally converted into an intermittent, striking late systolic *whoop* or *honk*, either spontaneously or in response to physical maneuvers. The whoop is high-frequency, musical, widely transmitted, and occasionally loud enough to be disconcertingly sensed by the patient. The musical whoop is thought to arise from mitral leaflets and chordae tendineae set into high-frequency periodic vibration. Here is William Osler's description:

> A well-nourished young girl was sent to me in May, 1888 by Dr Buller (the first Professor of Ophthalmology at McGill University), who had noticed a remarkable whistling sound, while examining her eyes. Auscultation—as she sits upright in the chair the heart sounds at the apex and base loud and clear; no murmur. When she stands, a loud systolic murmur is heard at the apex, high-pitched, somewhat musical, of maximum intensity in the fifth interspace; it varies a good deal, being loud for three or four beats, then faint for one or two succeeding ones. On removal of the ear from the chest wall, the murmur can be heard at a distance of several inches. It disappeared quite suddenly and could not be detected on most careful examination . . . . The child then suggested that she heard it most frequently when in the stooping posture; on causing her to lean forward and relax the chest, the murmur was at once heard, and with greatly increased intensity. It was distinctly audible at a distant three feet two inches by measurement, and could be heard at any point on the chest and on the top of the head.[22]

Systolic Arterial Murmurs

Auscultation at nonprecordial sites detects extracardiac systolic murmurs in systemic and pulmonary arteries. Systolic arterial murmurs originate in either anatomically normal arteries in the presence of normal or increased flow, or in tortuous or narrowed arteries. Timing with the first and second heart sounds is imprecise because extracardiac murmurs begin at variable distances from the heart. Nevertheless, the arterial murmurs dealt with here are essentially systolic and tend to have a crescendo-decrescendo configuration that coincides with the rise and fall of pulsatile flow.

A normal *supraclavicular* systolic arterial murmur is common in children and adolescents (Fig. 6–31*A*), and is believed to originate at the aortic arch origins of the brachiocephalic arteries. The configuration is crescendo-decrescendo, the onset abrupt, the duration brief, and at times the intensity is surprisingly loud with radiation below the clavicles, inviting a mistaken diagnosis of intrathoracic origin. Normal supraclavicular, systolic murmurs decrease or vanish in response to hyperextension of the shoulders that is sufficient to render the shoulder girdle muscles taut (Fig. 6–31*B*).

The most common cause of a systolic arterial murmur in older adults is peripheral vascular disease resulting from narrowing of carotid, subclavian, or iliofemoral arteries. Auscultation over these arteries should be routine in older adults.

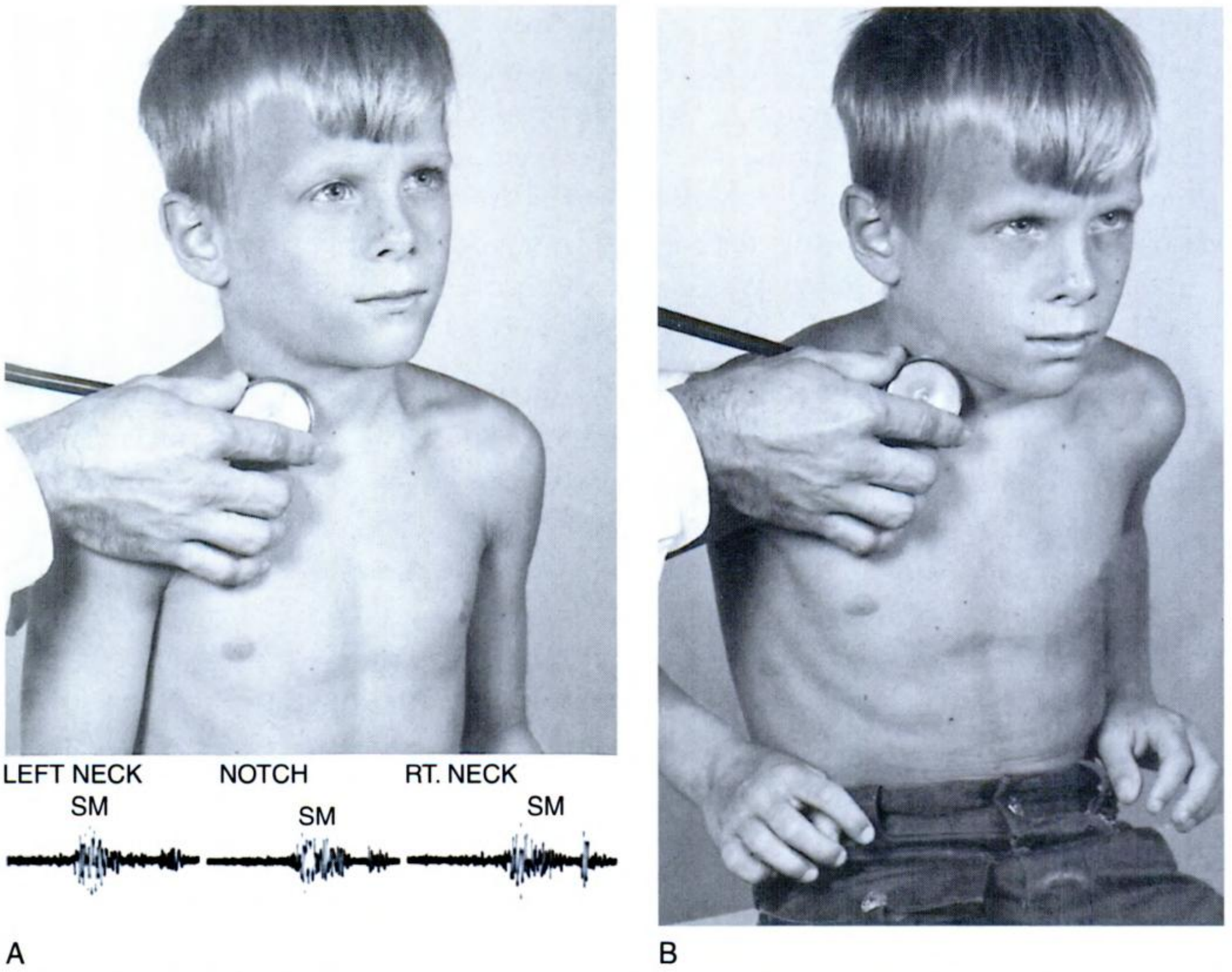


Figure 6–31 The phonocardiogram (A inset) recorded a normal supraclavicular systolic arterial murmur maximal above the clavicles in the left neck, right neck and in the suprasternal notch. Auscultation is initially carried out (*A*) while the patient sits with shoulders relaxed and arms resting in the lap. *B,* When the elbows are brought well behind the back to hyperextend the shoulders, the murmur markedly diminishes or disappears.

A variation of arterial narrowing is the "compression artifact" associated with severe aortic regurgitation. A two-phase systolic/diastolic murmur can be induced over a femoral artery in severe aortic regurgitation when the artery is compressed with the bell of the stethoscope. Moderate compression generates a systolic murmur. With further compression, the murmur becomes systolic/diastolic, a sign described in 1861 by Paul Louis Duroziez, president of the Société de Médecine in 1882. The eponym is still employed.

During late pregnancy, but especially postpartum in lactating women, a systolic "mammary souffle" is sometimes heard over the breasts because of increased flow through normal arteries. The murmur begins well after the first heart sound because of the interval between left ventricular ejection and arrival of flow at the artery of origin.

Still another type of systolic arterial murmur is heard in the back, and originates over the site of coarctation of the aortic isthmus. The murmur is elicited by applying

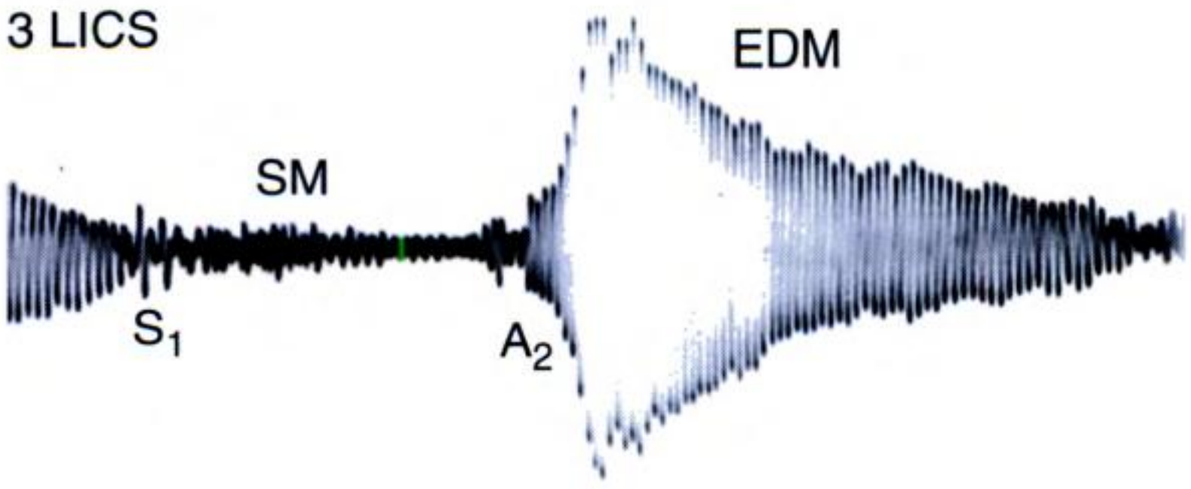


Figure 6–33 Phonocardiogram in the third left intercostal space (3 LICS) records the high-frequency, musical, decrescendo early diastolic murmur (EDM) caused by eversion of an aortic cusp. S_1 = first heart sound; SM = midsystolic murmur; A_2 = aortic component of the second heart sound.

inclined to believe that a murmur of similar mechanism occurs on the right side of the heart, when there is much obstruction to the pulmonary circulation, with a dilated pulmonary artery." My subsequent experience has only served to confirm the opinion thus cautiously expressed more than 7 years ago though my faith has from time-to-time been shaken by a case presenting a murmur which I had at first imagined to be an example, but which, on further investigation, proved to be of aortic origin.

A Graham Steell murmur begins with the loud *pulmonary* component of the second heart sound—"When the second sound is reduplicated, the murmur proceeds from its latter part"—because the elevated pressure exerted on the incompetent pulmonary valve begins at the moment that the right ventricular pressure drops below the pulmonary arterial incisura. The high diastolic pressure generates high-velocity regurgitant flow and a high-frequency murmur that may last throughout diastole. The configuration of the murmur is decrescendo, although occasionally the amplitude is relatively uniform throughout most if not all of diastole, and at times the murmur begins with a short crescendo. The auscultatory distinction between the early diastolic murmur of pulmonary hypertensive pulmonary regurgitation and aortic regurgitation was underscored by Graham Steell (see above). Even today the distinction is difficult or impossible when the pulmonary murmur is soft and the systemic arterial pulse is normal. The distinction depends on the clinical setting rather than on auscultation. Squatting and sustained handgrip augment the murmur of aortic regurgitation (see below), but leave the Graham Steell murmur unchanged.

Mid-Diastolic Murmurs

Mid-diastolic murmurs (Fig. 6–17) begin at a clear interval after the second heart sound.[16] The majority of mid-diastolic murmurs originate across mitral or tricuspid valves during

the rapid filling phase because of atrioventricular valve obstruction or abnormal patterns of atrioventricular flow, or across an incompetent pulmonary valve provided that the pulmonary arterial pressure is normal or nearly so. Rarely, a mid-diastolic murmur is due to flow through an atherosclerotic extramural coronary artery.

The mid-diastolic murmur of mitral stenosis is a useful point of departure. The murmur is present with sinus rhythm (Fig. 6–11) or atrial fibrillation (Fig. 6–34), and is characteristically introduced by an opening snap (Fig. 6–11). With the patient in the left lateral decubitus position (see Fig. 6–4), the bell of the stethoscope is placed lightly against the skin precisely over the left ventricular impulse. The murmur is maximal at that site because it originates within the left ventricular cavity. In atrial fibrillation, the duration of the mid-diastolic murmur is a useful sign of the degree of mitral stenosis, because a murmur that lasts up to the first heart sound even after long cycle lengths implies a persistent trans-mitral gradient at the end of diastole (Fig. 6–34). Soft mid-diastolic murmurs are augmented when the heart rate and mitral valve flow are transiently increased by brisk voluntary coughs.

The mid-diastolic murmur of *tricuspid stenosis* with atrial fibrillation differs from the *mitral* mid-diastolic murmur in two important respects: (1) the tricuspid murmur

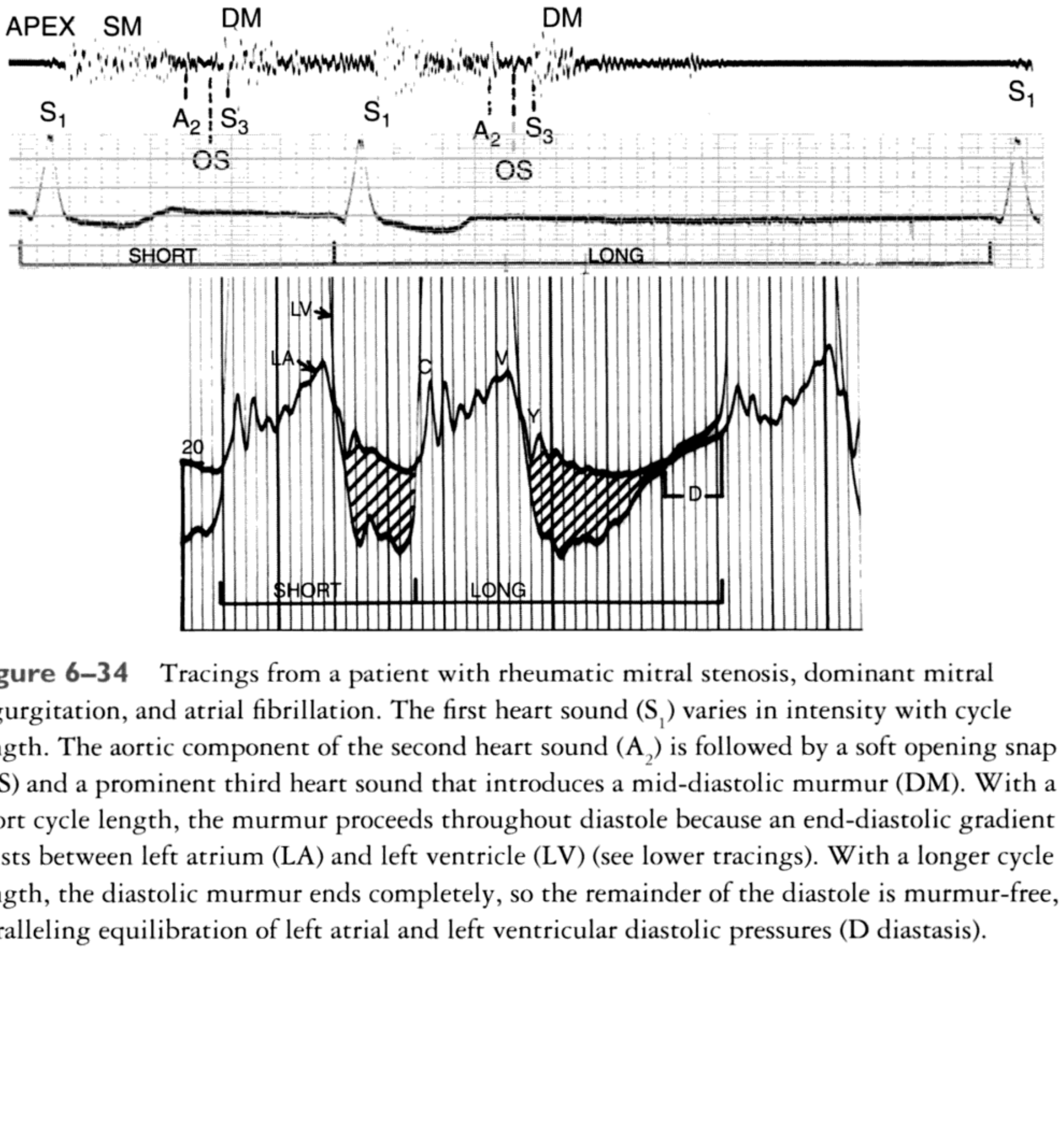


Figure 6–34 Tracings from a patient with rheumatic mitral stenosis, dominant mitral regurgitation, and atrial fibrillation. The first heart sound (S_1) varies in intensity with cycle length. The aortic component of the second heart sound (A_2) is followed by a soft opening snap (OS) and a prominent third heart sound that introduces a mid-diastolic murmur (DM). With a short cycle length, the murmur proceeds throughout diastole because an end-diastolic gradient exists between left atrium (LA) and left ventricle (LV) (see lower tracings). With a longer cycle length, the diastolic murmur ends completely, so the remainder of the diastole is murmur-free, paralleling equilibration of left atrial and left ventricular diastolic pressures (D diastasis).

selectively increases during inspiration, and (2) the tricuspid murmur is confined to a relatively localized area along the lower left sternal edge. The inspiratory increase occurs because inspiration is accompanied by an augmentation in right ventricular volume and a fall in right ventricular diastolic pressure, a combination that increases the diastolic gradient and flow rate across the tricuspid valve. The murmur is localized to the lower left sternal edge because it originates within the inflow portion of the right ventricle and is transmitted to the overlying chest wall.

Mid-diastolic murmurs occur across *nonobstructed* atrioventricular valves because of an augmented volume and velocity of flow. Examples in the left side of the heart include the mid-diastolic flow murmur accompanying pure mitral regurgitation, and the mid-diastolic flow murmur accompanying a ventricular septal defect with a large left-to-right shunt. Tricuspid mid-diastolic murmurs result from augmented flow across nonobstructed tricuspid valves in severe tricuspid regurgitation or with a nonrestrictive atrial septal defect and a large left-to-right shunt. These mitral or tricuspid mid-diastolic flow murmurs are typically short and medium pitched, occur with appreciable atrioventricular valve incompetence or large shunts, and are often preceded by third heart sounds, especially in the presence of mitral or tricuspid regurgitation.

In *complete heart block*, short, mid-diastolic atrioventricular flow murmurs occur when atrial contraction coincides with and therefore reinforces the phase of rapid diastolic filling (Fig. 6–35*B*). These murmurs result from antegrade flow across atrioventricular valves that are closing as the recipient ventricle is filling. A similar mechanism has been assigned to the Austin Flint murmur (Fig. 6–36), as Flint originally proposed[23] (see below). In acute severe aortic regurgitation, the Austin Flint murmur is necessarily confined to mid-diastole because premature mitral valve closure precludes presystolic flow.

A mid-diastolic murmur is a feature of pulmonary valve regurgitation provided that the pulmonary arterial pressure is normal or low (Fig. 6–37*A*). The cause of the pulmonary regurgitation can be acquired (pulmonary valve infective endocarditis) or congenital. The most common cause of low-pressure pulmonary valve regurgitation, however, is surgical repair of right ventricular outflow obstruction. The mid-diastolic murmur begins at a perceptible interval after the pulmonary component of the second heart sound and is crescendo-decrescendo, ending well before the subsequent second heart sound.

The physiologic mechanism responsible for the timing of the mid-diastolic murmur of low-pressure pulmonary regurgitation is illustrated in Figure 6–37*B*. Diastolic pressure exerted on the incompetent pulmonary valve at the time of the pulmonary component of the second heart sound is negligible, so regurgitant flow is negligible. As right ventricular pressure drops below the diastolic pressure in the pulmonary trunk, regurgitation accelerates, and the murmur reaches its maximum intensity (Fig. 6–37*B*). Equilibration of pulmonary arterial and right ventricular pressures in late diastolic eliminates regurgitant flow and abolishes the murmur prior to the next first heart sound. When the pulmonary component of the second sound is late, soft, or absent, the gap between the aortic component and the onset of the pulmonary diastolic murmur is even more conspicuous.

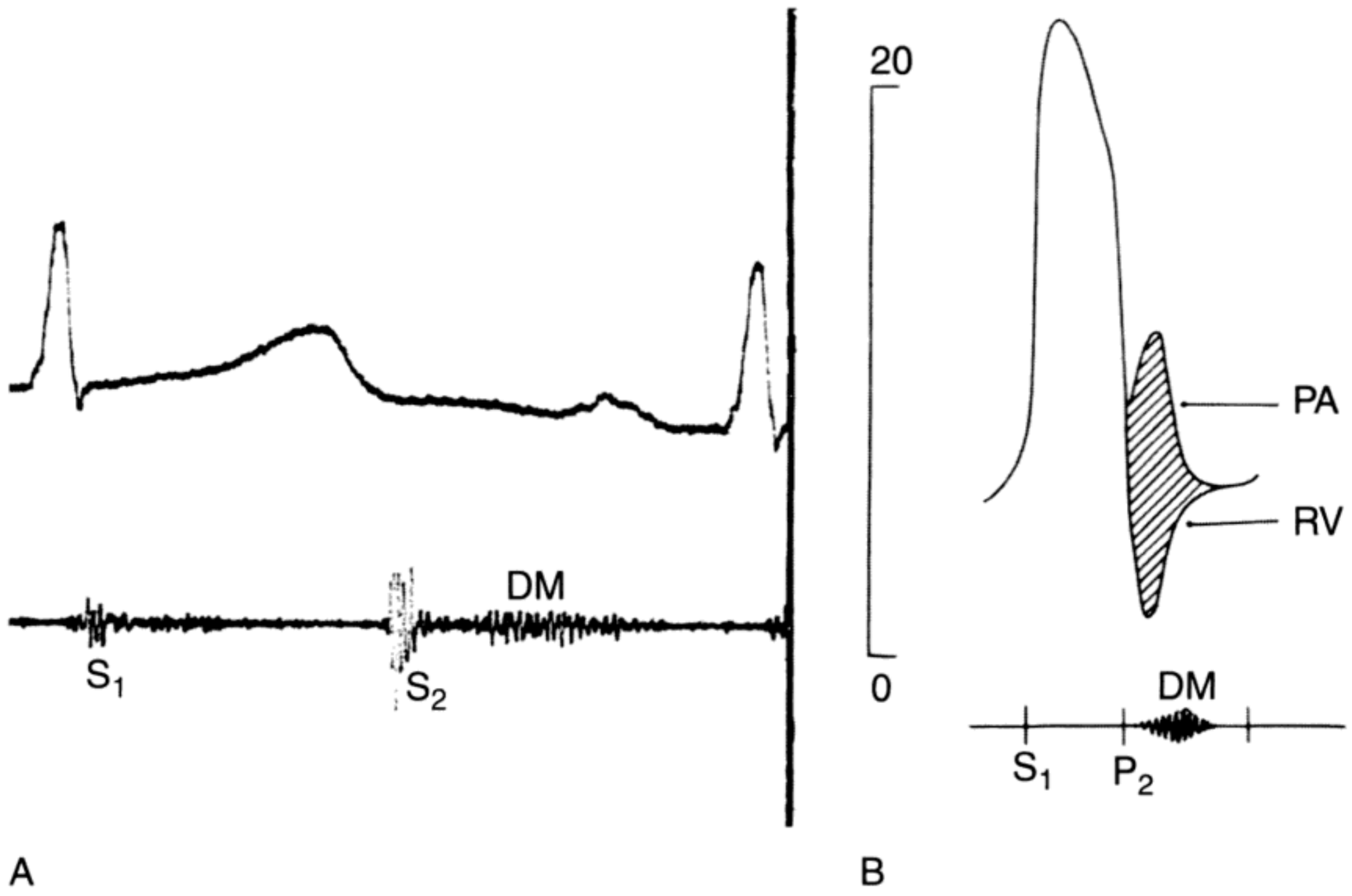


Figure 6–37 *A,* Phonocardiogram of the mid-diastolic murmur (DM) of low-pressure pulmonary valve regurgitation in a heroin addict with pulmonary valve infective endocarditis. The murmur begins well after the second sound (S_2), is medium frequency and mid-diastolic, and ends well before the subsequent first heart sound. *B,* Pressure pulses and phonocardiogram illustrating the physiologic mechanism of the mid-diastolic murmur (DM) of low-pressure pulmonary regurgitation. The pressure exerted against the incompetent pulmonary valve is low, so the murmur does not begin until well after the right ventricular (RV) and pulmonary arterial (PA) pressure pulses diverge. The murmur is maximal when the diastolic gradient is greatest (*cross-hatched area*). There is an early diastolic dip in the RV pulse. Equilibration of the pulmonary arterial and right ventricular pressures in later diastole abolishes the regurgitant gradient so the murmur disappears.

presystolic murmur of mitral stenosis, which tends to rise in crescendo to the first heart sound (Fig. 6–11). However, the most valuable auscultatory sign of tricuspid stenosis in sinus rhythm is the response to respiration. Inspiration increases right atrial volume, provoking an increase in right atrial contractile force in the face of a fall in right ventricular end-diastolic pressure. The result is a larger gradient, an increased velocity of tricuspid flow, and an increase in intensity of the presystolic tricuspid stenotic murmur, as shown in Figure 6–38.

Presystolic and mid-diastolic murmurs occasionally occur in patients with a myxoma of the left or right atrium. These murmurs can resemble the murmurs of mitral or tricuspid stenosis. The accompanying "tumor plop" can be mistaken for an opening snap (see above).

In complete heart block, short, crescendo-decrescendo presystolic murmurs are occasionally heard when atrial contraction fortuitously falls in late diastole (Fig. 6–35). However, in complete heart block, the diastolic murmur is usually mid-diastolic (see above) when atrial contraction coincides with and reinforces the rapid filling phase.

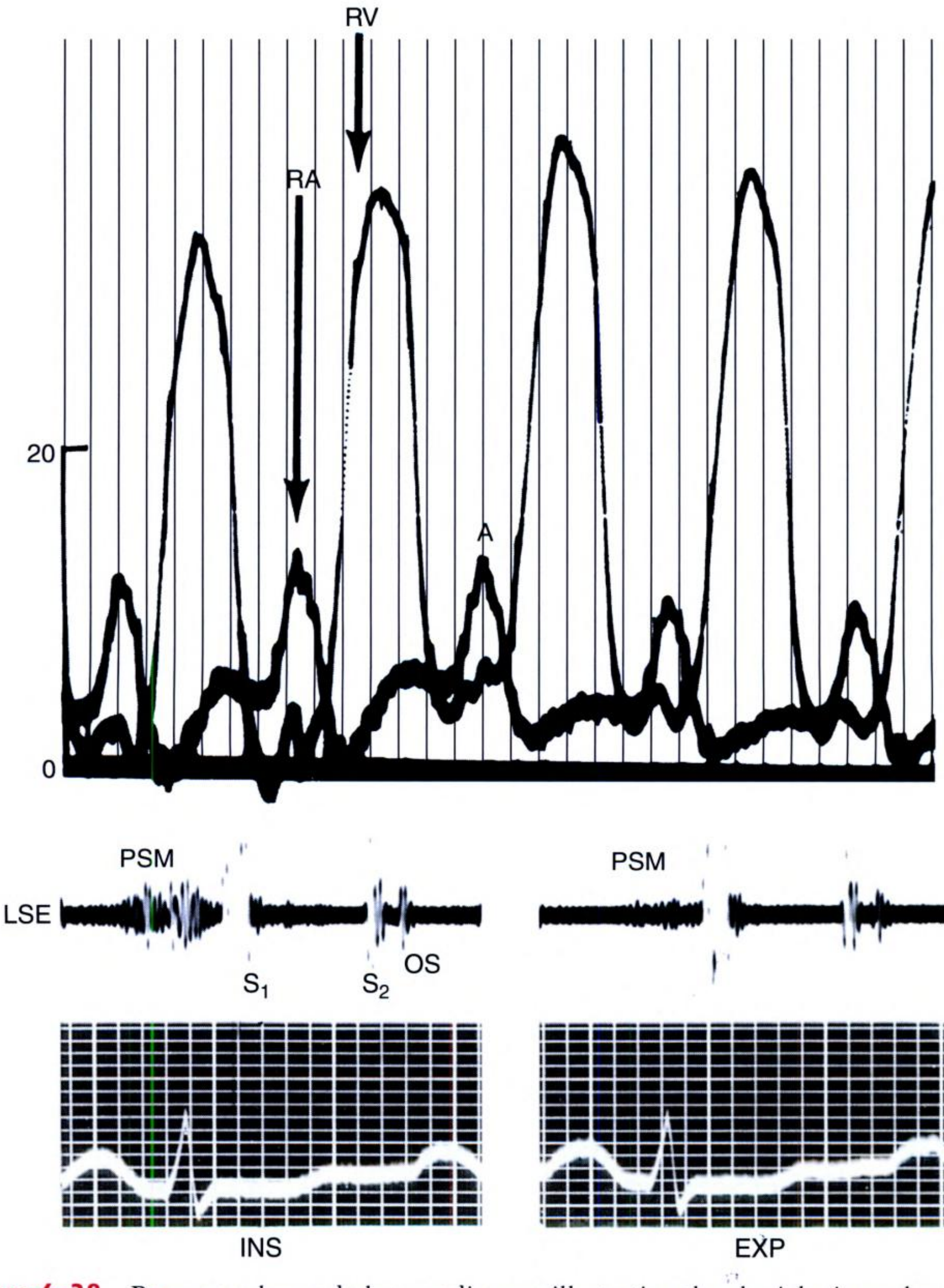


Figure 6–38 Pressure pulses and phonocardiogram illustrating the physiologic mechanism responsible for the respiratory variation in the presystolic murmur of tricuspid stenosis. During inhalation, a fall in intrathoracic pressure and an increase in systemic venous return result in an increase in right atrial (RA) A wave and a decrease in right ventricular (RV) end-diastolic pressure, so the presystolic murmur (PSM) increases. During exhalation, the right atrial A wave declines, the right ventricular diastolic pressure increases, the tricuspid gradient is minimal, and the presystolic murmur all but vanishes.

The Austin Flint presystolic murmur was described in 1862.[23] Flint was one of America's most distinguished nineteenth century physicians, and has been called America's Laennec.[24]

The mechanism proposed by Flint was perceptive:

> Is this murmur ever produced without any mitral lesions? One would *a priori* suppose the answer of this question to be in the negative. Clinical observation, however, shows that the question is to be answered in the affirmative. In May, 1860, I examined a patient, age 56. At the apex was a presystolic blubbering murmur.[23]

At necropsy days later:

> The aorta was atheromatous and dilated so as to render the valvular segments evidently insufficient. The mitral valve presented nothing abnormal.[23]

Flint examined a second case the following year and concluded:

> In both cases the mitral direct murmur was loud and had that character of sound which I supposed to be due to vibration of the mitral curtains. In both cases it will be observed, an aortic regurgitant murmur existed, and aortic insufficiency was found to exist at postmortem. A mitral direct murmur, then, may exist without mitral contraction and without any mitral lesions, provided there be aortic lesions involving considerable aortic regurgitation.[23]

Flint proposed the following mechanism:

> Now in cases of considerable aortic insufficiency, the left ventricle is rapidly filled with blood flowing back from the aorta as well as from the auricle, before the auricular contraction takes place. The distention of the ventricle is such that the mitral curtains are brought into coaptation, and when the auricular contraction takes place the mitral direct current passing between the curtains throws them into vibration and gives rise to the characteristic blubbering murmur.[23]

Current consensus regarding the mechanism of the Austin Flint murmur is in accord with Flint's original proposal. The jet accompanying chronic severe aortic regurgitation impacts and impinges on the anterior mitral leaflet, limiting its opening excursion and encroaching on the mitral orifice, a combination that results in an increase in mitral inflow velocity, an increase in vibrations from the anterior mitral leaflet, and an increase in turbulence from the combined effects of aortic regurgitant flow and mitral inflow.

Presystolic murmurs tend to be relatively low to medium frequency, so auscultation should be undertaken with just enough pressure of the bell of the stethoscope to achieve a skin seal. Presystolic murmurs originating across the mitral valve are best elicited over the left ventricular impulse with the patient in a partial left lateral decubitus position (Fig. 6–4). Presystolic murmurs across the tricuspid valve are best elicited with the patient supine while the examiner applies the stethoscope to the lower left sternal edge, moving the bell in small increments in search of the localized murmur. The presystolic murmur of tricuspid stenosis may be heard *only* during inspiration (see

Fig. 6–38), so the patient should be instructed to breathe rhythmically with a moderate increase in depth.

Continuous Murmurs

The term *continuous* applies to murmurs that begin in systole and *continue* without interruption through the timing of the second heart sound into all or part of diastole (Fig. 6–18).[16] A holosystolic murmur followed by a holodiastolic murmur occupy both phases of the cardiac cycle, but represent two murmurs, not a continuous murmur (Fig. 6–18). Conversely, a murmur that ends before the subsequent first heart sound is continuous provided the murmur begins in systole and proceeds without interruption through the timing of the second heart sound (see Fig. 6–18).

Continuous murmurs are generated by flow from a vascular bed of higher resistance into a vascular bed of lower resistance, without phasic interruption between systole and diastole. Such murmurs are associated with (1) aortopulmonary connections, (2) arteriovenous connections, (3) disturbances of flow patterns in arteries, and (4) disturbances of flow patterns in veins (see Fig. 6–18).

Best known and most celebrated is the continuous murmur of patent ductus arteriosus (Figs. 6–18 and 6–39). The murmur characteristically peaks before and after the second heart sound, and is often soft or absent in late diastole.

In 1847, the *London Medical Gazette* published the following description:

> a murmur accompanying the first heart sound, prolonged into the second so that there is no cessation of the murmur before the second sound has already commenced.[25]

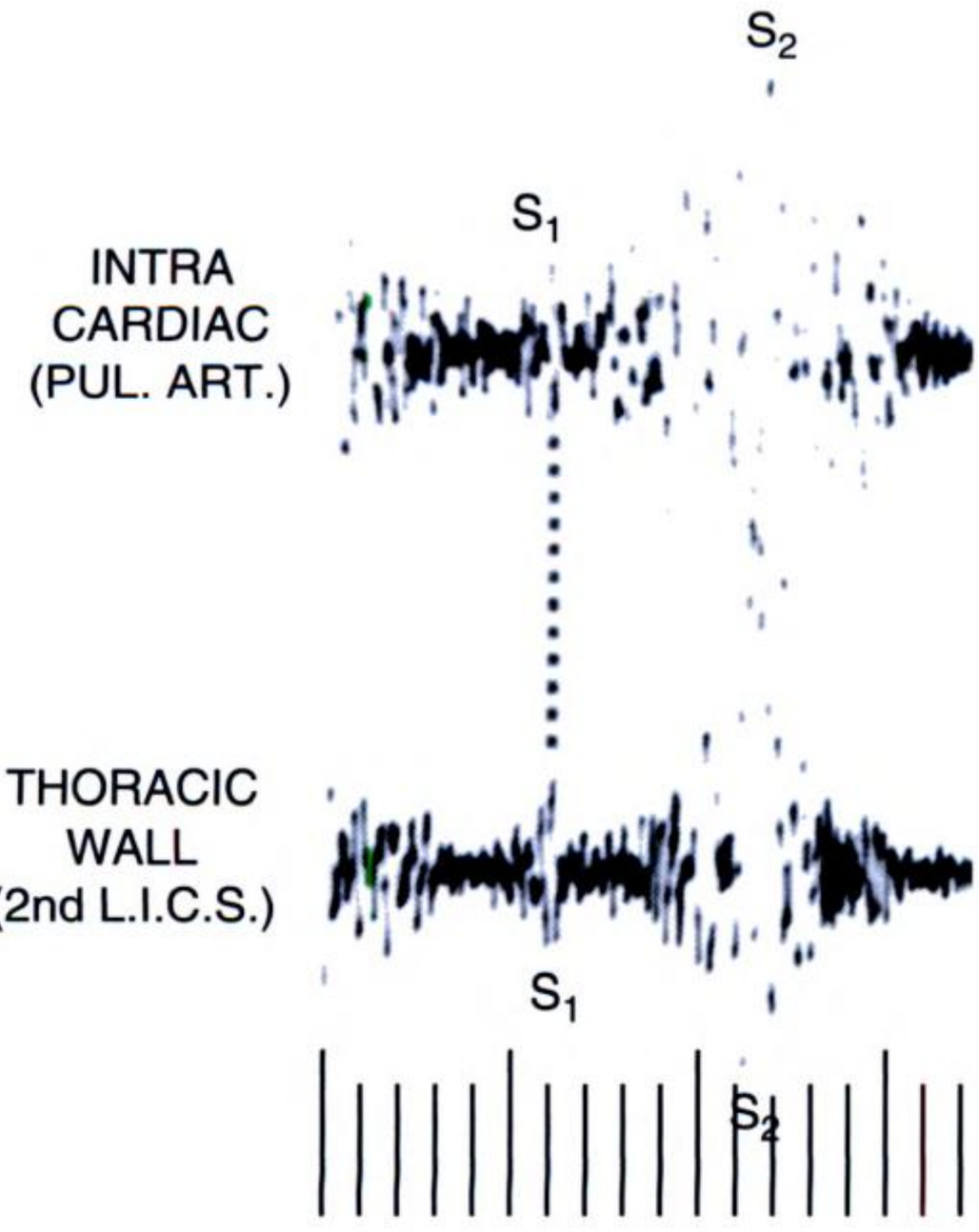


Figure 6–39 The classic continuous murmur of patent ductus arteriosus simultaneously recorded from within the main pulmonary artery (PUL ART) and at the second left intercostal space on the thoracic wall (2 LICS). The murmur "begins softly and increases in intensity so as to reach its acme just about, or immediately after the incidence of the second sound, and from that point gradually wanes until its termination," as originally described by Gibson in 1900.[26]

The cause of the murmur was correctly attributed to patent ductus arteriosus, and the term *continuous* was correctly defined as "no cessation of the murmur before the second sound has already commenced."[25]

In 1900, George A. Gibson provided a meticulous description of the murmur (Fig. 6–39) of patent ductus arteriosus that is still called the *Gibson murmur*. He wrote:

> It persists through the second sound and dies away gradually during the long pause. The murmur is rough and thrilling. It begins softly and increases in intensity so as to reach its acme just about, or immediately after, the incidence of the second sound, and from that point gradually wanes until its termination.[26]

Arteriovenous continuous murmurs can be congenital or acquired, and are associated with arteriovenous fistulae, coronary arterial fistulae, anomalous origin of the left coronary artery from the pulmonary trunk, and communications between a sinus of Valsalva and the right heart. The configuration, location, and intensity of arteriovenous continuous murmurs vary considerably. The continuous murmur resulting from a systemic arteriovenous fistula was described by the Czech–Austrian pathologist Josef Skoda. The English translation reads:

> When a moderately sized artery communicates with a vein, a very loud continuous murmur usually develops at the point of communication, the strength of which increases on each pulsation of the artery and is audible over a greater or lesser part of the surrounding area.[27]

Acquired systemic arteriovenous fistulae are represented by the surgically created forearm connections used for renal dialysis. The continuous murmur of a *congenital* coronary arterial fistula that enters the right ventricle can be either softer or louder in systole depending on the degree of compression exerted on the fistulous coronary artery by right ventricular contraction. The continuous murmur of a sinus of Valsalva aneurysm that ruptures into the right heart does not peak around the second heart sound, but tends to be louder in either systole or diastole, sometimes creating a to-and-fro impression.

Continuous murmurs can originate in *constricted* or *nonconstricted* arteries. In *constricted systemic or pulmonary arteries*, continuous flow disturbances occur when a significant persistent pressure difference exists between the two sides of the narrowed arterial segment. Arterial continuous murmurs arising in constricted arteries are characteristically louder in systole (see Fig. 6–18). Atherosclerotic obstruction of carotid or femoral arteries are examples.

Disturbances of flow patterns in *normal, nonconstricted ar*teries sometimes produce continuous murmurs. The *mammary souffle* (see above), an innocent murmur heard maximally over lactating breasts during late pregnancy and the puerperium, is typically continuous, but usually louder in systole. The souffle tends to be more prominent in the second or third right or left intercostal space. A distinct gap separates the first heart sound from the onset of the souffle because of the interval that elapses before blood ejected from the left ventricle arrives at the artery of origin. The mammary souffle is best heard with the patient supine, and may vanish altogether in the upright position. Light

pressure with the stethoscope augments the murmur and brings out its continuous features, whereas firm pressure or digital compression peripheral to the site of auscultation abolishes the murmur.

Continuous murmurs in nonconstricted arteries originate in large systemic-to-pulmonary arterial collaterals in certain types of cyanotic congenital heart disease, especially Fallot's tetralogy with pulmonary atresia. These murmurs are randomly located throughout the thorax, and must be diligently sought.

Continuous venous murmurs are represented by the normal venous hum (Fig. 6–40) which is almost universal in healthy children and is frequently present in healthy young adults, especially during pregnancy. Potain described the venous hum in 1867:

> The thrill, which is felt by placing the finger lightly above the clavicle over the course of the vessel of the neck, is sometimes continuous and frequently intermittent. It is this last case which interests us above all here. A light pressure exerted above the point of exploration can make it appear or reinforce it, while a stronger pressure extinguishes it completely, proofs positive that we are concerned with a venous phenomenon and that this thrill does not arise at all from an artery.[28]

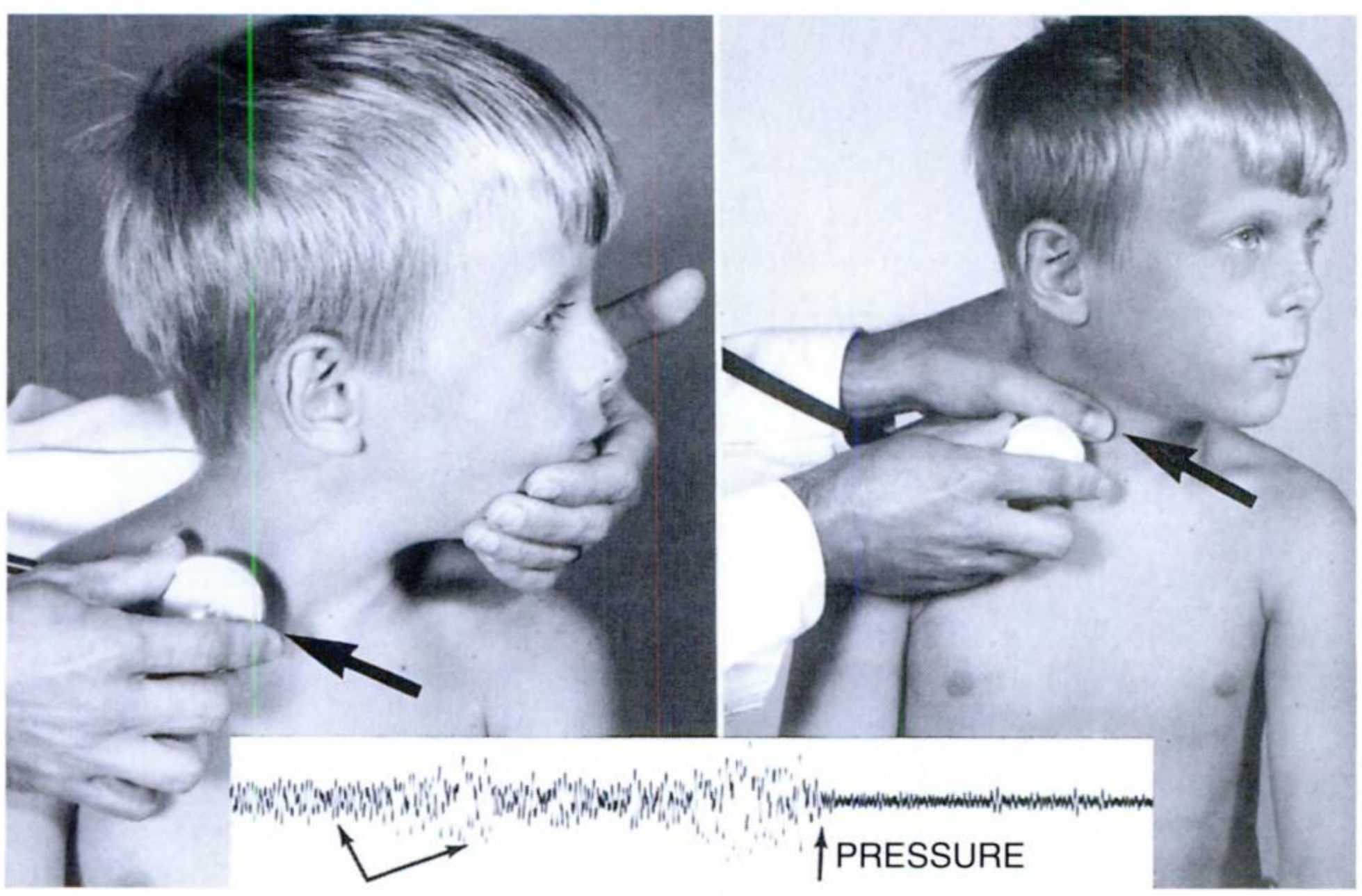


Figure 6–40 Photograph on the left shows the bell of the stethoscope applied to the medial aspect of the right supraclavicular fossa (*arrow*). The examiner's left hand grasps the chin from behind and pulls it tautly to the left and upward, stretching the neck. The photograph on the right shows digital compression of the right internal jugular vein (*arrow*) that obliterates the venous hum (*vertical arrow insert*). The patient's head has returned toward a neutral position.

The venous hum is elicited with the patient sitting (Fig. 6–40). The stethoscopic bell is applied to the medial aspect of the right supraclavicular fossa, while the examiner's left hand grasps the patient's chin from behind and pulls it tautly to the left and upward (Fig. 6–40*A*). The hum disappears when the stretch is removed by returning the head to a neutral position (Fig. 6–40*B*). Occasionally the hum appears or increases when the chin is simply tilted upward, and at other times it is prominent without neck maneuvers and irrespective of position. Venous hums in young children can appear when a child turns its head to the left or looks upward. The hum is abolished by applying pressure to the ipsilateral deep jugular vein with the thumb of the examiner's free hand, as Potain described and as shown in Figure 6–40. Compression causes instantaneous disappearance of the hum (see phonocardiographic insert, Fig. 6–40), which suddenly but transiently intensifies as pressure is released. The term *hum* does not necessarily describe the quality of these cervical venous murmurs, which can be rough and noisy, or accompanied by a high-pitched whine. The venous hum is truly continuous but is characteristically louder in diastole (Fig. 6–40), as is the case with venous continuous murmurs in general (see Fig. 6–18). A loud venous hum that radiates below the clavicles can be mistaken for an intrathoracic continuous murmur. Obliteration by digital pressure in the neck abolishes the transmitted hum and avoids error.

PERICARDIAL RUBS

The pericardial rub—aptly called a *friction* rub—resembles the sound produced by rubbing or scratching sandpaper. Less commonly, indeed rarely, the rub is musical, resembling the rubbing of a wet finger on a glass surface. Pericardial rubs have three components—one during ventricular systole and two during ventricular diastole—the latter coinciding with the rapid filling phase and the presystolic filling phase. A two-phase rub is usually systolic and presystolic. In atrial fibrillation, the presystolic component necessarily disappears. A single-phase rub, which is either systolic or presystolic, cannot be identified as such unless a two- or three-phase rub had previously been heard.

Pericardial friction rubs originate from the anterior surface of the right ventricle, and are therefore louder in response to maneuvers that increase right atrial and right ventricular filling, or that bring the right ventricle closer to the chest wall. With the patient supine, firm pressure is applied with the stethoscopic diaphragm during full held exhalation or, more awkwardly, with the patient prone but with the chest lifted (Fig. 6–41). The rub is typically louder during inhalation because of the inspiratory increase in venous return to the right atrium and right ventricle. Passive lifting of the legs with the patient supine transiently increases the rub by the same mechanism. The most common clinical setting in which pericardial rubs are heard is immediately after open heart surgery.

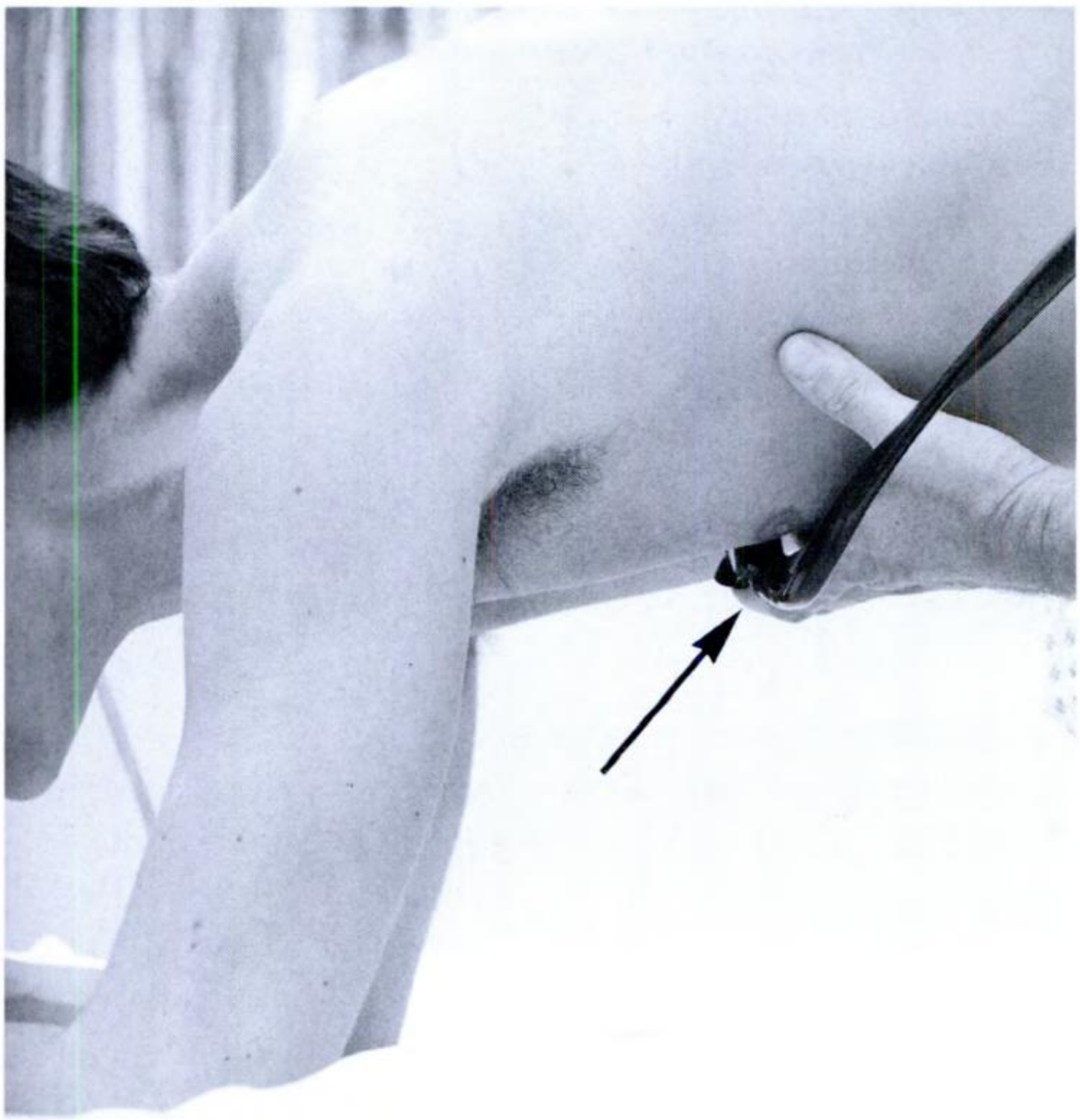


Figure 6–41 A technique for eliciting a pericardial friction rub. The diaphragm of the stethoscope is firmly applied to the precordium (*arrow*) while the patient is supported on elbows and knees.

PHYSICAL MANEUVERS AND PHARMACOLOGIC INTERVENTIONS

A number of physical maneuvers employed during auscultation have been discussed. The following remarks reemphasize the importance of these maneuvers and bring them briefly into focus (Tables 6–5 and 6–6). Pharmacologic interventions are mentioned, although these are less practical and therefore less important at the bedside.

Changes in position that are useful during auscultation are listed in Table 6–5. A partial left lateral decubitus position (see Fig. 6–4) assists in identifying the left ventricular impulse, which is an important auscultatory landmark. During the act of turning, the heart rate transiently increases, improving audibility of mid-diastolic and presystolic murmurs of mitral stenosis. The left lateral decubitus position occasionally induces premature ventricular beats that distinguish an aortic midsystolic murmur at the apex from the apical murmur of mitral regurgitation (see earlier).

Table 6–5 Positions During Auscultation

Left lateral decubitus
Sitting leaning forward
Sitting with legs dangling
Standing/squatting and *vice versa*
Hyperextension of the shoulders
"Stretching" of the neck
Passive elevation of the legs
Support of precordium on elbows and knees

Sitting and leaning forward in full held exhalation (Fig. 6–32) improves audibility of the soft, high-frequency early diastolic murmur of aortic regurgitation, or a soft Graham Steell murmur. Sitting with legs dangling over the side of the bed or examining table helps identify normal splitting of the second heart sound when the split fails to fuse during exhalation in the supine position.

Standing and squatting followed by prompt standing (Figs. 6–8 and 6–30) is achieved with the examining gown securely fastened so the garment doesn't slip off and compromise the examination. During the squatting/standing maneuver, balance is important, especially in older adults, who should squat within arm's reach of support from the bed or examining table for support (Fig. 6–30). The sequence of standing then squatting followed by prompt standing is useful in assessing the murmurs of aortic and mitral regurgitation, mitral valve prolapse, and hypertrophic obstructive cardiomyopathy.

Hyperextension of the shoulders is important in assessing supraclavicular systolic murmurs (Fig. 6–31). A venous hum is initiated or reinforced by stretching the neck upward and to the left as the stethoscopic bell is applied to the medial aspect of the right supraclavicular fossa (see Fig. 6–40).

Third heart sounds are augmented by the transient increase in venous return in response to passive elevation of the legs with the patient supine. The patient must not to assist in leg raising in order to avoid involuntary straining and a partial Valsalva maneuver.

Table 6–6 Other Physical Maneuvers During Auscultation

Respiration
Inhalation, exhalation
Full, held exhalation
Valsalva and Mueller maneuvers

The precordium can be elevated above the bed or examining table by having the patient rise on elbows and knees (Fig. 6–41), a maneuver, albeit awkward, that assists in detecting pericardial friction rubs (see above).

Physical maneuvers other than positional

Normal rhythmic inhalation and exhalation are employed routinely (see Table 6–6). Exaggerated respiratory excursions are useful in analyzing splitting of the second heart sound, right-sided third and fourth heart sounds, tricuspid systolic and diastolic murmurs, and pulmonary ejection sounds. The desired depth and rate of breathing should be demonstrated. Full held exhalation while the patient sits and leans forward (Fig. 6–32) is used to elicit the soft, early diastolic murmur of aortic regurgitation or pulmonary hypertensive pulmonary regurgitation. The innocent pulmonary midsystolic murmur associated with decreased anteroposterior chest dimensions (loss of thoracic kyphosis) amplifies when the diaphragm of the stethoscope is pressed firmly in the second left intercostal space during full held exhalation.

The Valsalva maneuver (Antonio Maria **Valsalva**: Italian anatomist, born 1666) is readily performed at the bedside, and consists of a deep inspiratory effort followed by forced exhalation against a closed glottis for approximately 10 seconds.[29] The maneuver was described in 1704 as a method for expelling pus from the middle ear by straining while the mouth and nose were closed. Simulation by the examiner is a simple means of instructing the patient on how to perform the maneuver. The flat of the hand is placed on the abdomen to provide a force against which the patient can strain, and to provide an assessment of the degree and duration of the effort.

The normal Valsalva response consists of four phases. *Phase I is* associated with a transient rise in systemic blood pressure as straining commences. This phase cannot be identified at the bedside. *Phase 2* is accompanied by a decrease in blood pressure and pulse pressure, reflex sinus tachycardia, and an increase in the intensity of the murmurs of hypertrophic obstructive cardiomyopathy and mitral valve prolapse.[29] *Phase 3* begins with cessation of straining, is associated with an abrupt, transient decrease in blood pressure not perceived at the bedside, and is followed promptly by *Phase 4* which is characterized by an overshoot of systemic arterial pressure and reflex bradycardia.

The increase in venous return and in systemic blood pressure during Phase 4 intensify outflow and regurgitant murmurs except the murmur of hypertrophic obstructive cardiomyopathy and the regurgitant murmur of mitral valve prolapse, both of which decrease as the result of larger left ventricular volume.[29] Phase 4 also identifies murmurs that originate in the right side of the heart because right-sided murmurs promptly return to baseline intensity after release of the Valsalva maneuver in response to an immediate increase in venous return. Conversely, murmurs originating in the left side of the heart take a longer time to return to baseline after release of the maneuver because of the time required for systemic venous return to

traverse the pulmonary circulation. Elderly patients tend to have impaired reflex mechanisms, so the Valsalva maneuver may cause syncope and should be performed cautiously if at all.[29]

The Muller maneuver is the converse of the Valsalva, and is less frequently employed because it is less useful at the bedside. The Muller maneuver is performed for about 10 seconds as the patient forcibly inhales against a firmly closed nose and mouth. By exaggerating the inspiratory effort, the Miller maneuver augments the murmur of tricuspid regurgitation or stenosis, but usefulness is limited.

Isometric exercise (sustained handgrip) is a simple, safe bedside maneuver. Sustained handgrip imposes a pressure load on the left ventricle by increasing systemic systolic blood pressure which is accompanied by an increase in heart rate and cardiac output, all of which promptly revert to control levels with cessation of the maneuver. To achieve the desired effect of isometric exercise, the patient simultaneously squeezes both fists. But if the fingernails are long, a firm rubber ball or an analogous object can be compressed. Care should be taken to avoid tensing proximal muscle groups, especially the shoulder girdle. Sustained handgrip is maintained until the patient is told to desist. The duration depends on whether and when the desired auscultatory response is elicited. Auscultation should therefore precede, accompany, and follow the maneuver. Twenty seconds of isometric exercise usually suffice. The response to sustained handgrip reinforces left ventricular fourth heart sounds and augments the murmurs of mitral and aortic regurgitation. The click(s) of mitral valve prolapse occur later in systole, while the late systolic murmur shortens but increases in intensity. The systolic murmur of hypertrophic obstructive cardiomyopathy softens because the increase in afterload increases left ventricular volume.

Amyl nitrite inhalation occasionally has a place during auscultation. The drug prompts a fall in systemic vascular resistance and blood pressure and an increase in heart rate, cardiac output, and ejection velocity. Inhalation is from a broken vial held close to the nose, preferably under a small cloth or handkerchief. The patient is instructed to breathe naturally while the arterial pulse is monitored for the first evidence of reflex tachycardia, at the onset of which inhalation is discontinued. The pulse rate is simpler and easier to monitor than the cuff blood pressure, and the response to nitrite is easier to monitor during continuous natural breathing than in response to several deep breaths.

The auscultatory effects of amyl nitrite inhalation are in accord with the hemodynamic effects just described. An increase in cardiac output and ejection velocity is accompanied by an increase in intensity of the systolic murmur of aortic stenosis or of isolated pulmonary stenosis (Fig. 6–42). A decrease in systemic vascular resistance is accompanied by a decrease in the systolic murmur of mitral regurgitation (Fig. 6–43*A*) and in the diastolic murmur of aortic regurgitation (Fig. 6–36). The mid to late systolic clicks and late systolic murmur of mitral valve prolapse occur earlier because of a reduction in left ventricular volume, but the murmur becomes softer because of decreased resistance to left ventricular discharge)

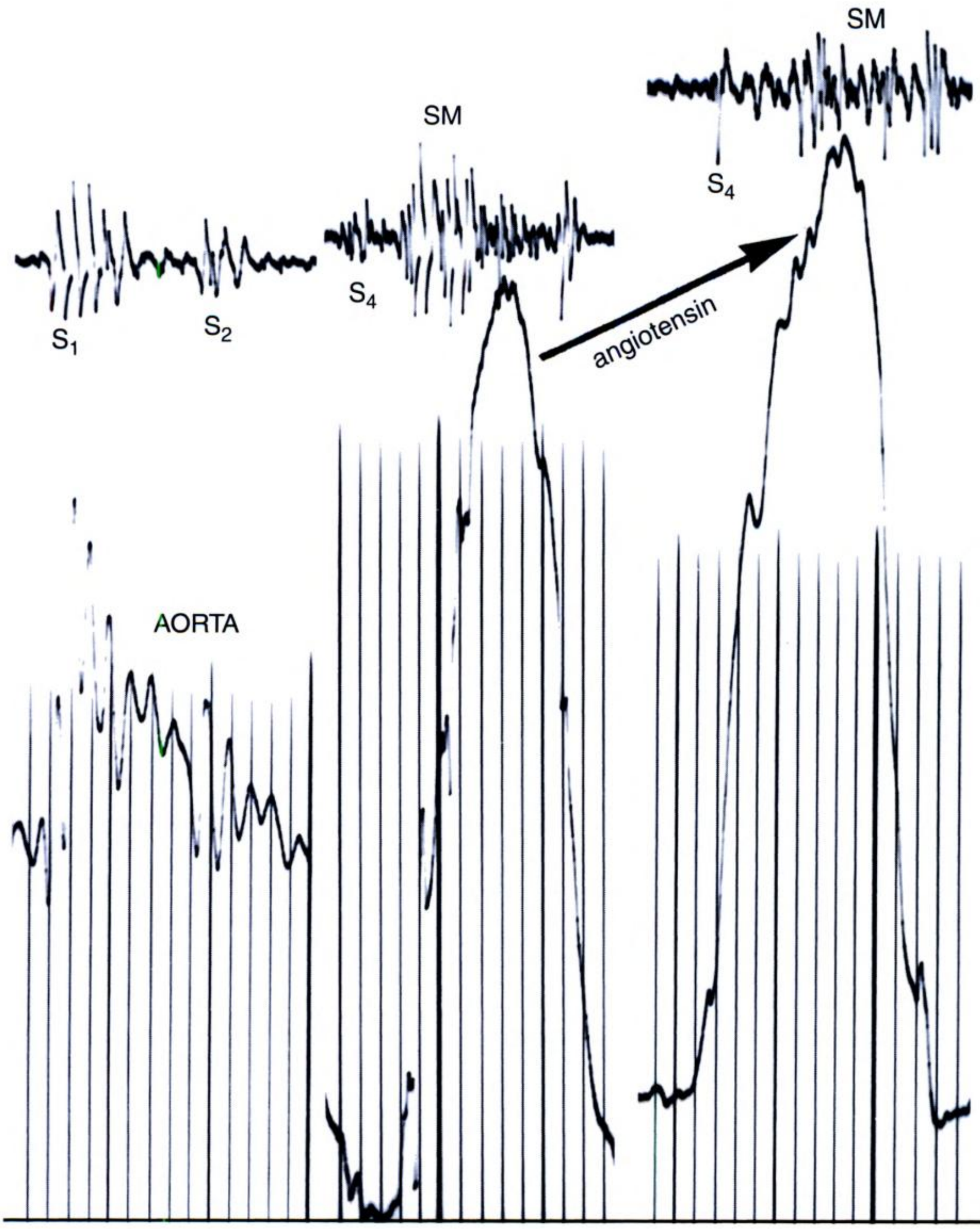


Figure 6–44 The effect of an increase in resistance to left ventricular discharge in a patient with hypertrophic obstructive cardiomyopathy. The first panel is a phonocardiogram and pressure pulse recorded within the ascending aorta just above the aortic valve. There is no systolic murmur. (S_1 S_2, first and second heart sounds.) In the middle panel, recordings are from within the left ventricular cavity (LV). There is a prominent fourth heart sound (S_4) and a prominent systolic murmur (SM). The third panel illustrates a decrease in intensity of the intracavity left ventricular systolic murmur (SM, *oblique arrow*) as resistance to left ventricular discharge increased in response to an angiotensin infusion The fourth heart sound (S_4) recorded from within the left ventricular cavity increased considerably.

REFERENCES

1. Stokes W. *An Introduction to the Use of the Stethoscope.* Edinburgh: Maclachlan and Stewart, 1825.
2. Craige E. Should auscultation be rehabilitated? *N Engl J Med.* 1988;318:1611.
3. Latham PM. *Lectures on Subjects Connected with Clinical Medicine Comprising Diseases of the Heart.* Philadelphia, PA: Barrington and Hoswell, 1847.
4. Laennec RTH. *A Treatise on the Diseases of the Chest.* Translated by John Forbes. Philadelphia, PA: James Webster, 1823.
5. Veith I (trans). *Huang Ti Nei Ching Su Wen: The Yellow Emperor's Book of Medicine.* Berkeley, CA: University of California Press, 1972.
6. McKusick VA. Rouanet of Paris and New Orleans. *Bull Hist Med.*1958:32:137.
7. Bowditch HI. *The Young Skethoscopist.* New York: Samuel S. and William Wood, 1848.
8. Potain PC. Clinique medicale de la Charite. Paris, Masson, 1894. In: McKusick VA. *Cardiovascular Sound in Health and Disease.* Baltimore, MD: Williams & Wilkins. 1958.
9. Leatham A. The second heart sound: Key to auscultation of the heart. *Acta Cardiol.* 1964;19:395.
10. Potain PC. Note sur les dedoublements normaux des bruits du coeur. *Bull Mem Soc Med Hop Paris.* 1866;3:138.
11. Steell G. The murmur of high-pressure in the pulmonary artery. *Med Chron Manchester.* 1888;9:182.
12. Connolly DC, Mann RJ: Dominic J. Corrigan (1802–1880) and his description of the pericardial knock. *Mayo Clin Proc.* 1980;55:771.
13. Potain PC. Concerning the cardiac rhythm called gallop rhythm. *Bull Mem Soc Med Hop Paris* 1876;12:137. In: Major RH. *Classic Descriptions of Disease.* 3rd ed. Springfield, IL, Charles C Thomas, 1948.
14. Ozawa MD, Smith D, Craige E. Origin of the third heart sound. *Circulation.* 1983;678:399.
15. Freeman AR, Levine SA. The clinical significance of the systolic murmur: A study of 1000 consecutive "non-cardiac" cases. *Ann Intern Med.* 1933;6:1371.
16. Silverman ME, Wooley CF, Samuel A. Levine and the history of grading systolic murmurs. *Am J Cardiol.* 2008;102:1107.
17. Leatham A. Auscultation of the heart. *Lancet.* 1958;2:703.
18. Gallavardin L, Ravault P. Le souffle de retrecissement aortique peut changer de timbre et devenir musical dans sa propagation apexienne. *J Med Lyon.* 1925;135:523.
19. Stokes W. *Diseases of the Heart and Aorta.* Philadelphia, PA: Lindsay and Blakiston, 1855.
20. Still GF. *Common Disorders and Diseases of Childhood.* London: Henry Frowde, 1909.
21. Rivero-Carvallo JM. Signo para el diagnostico de las insuficiencias tricuspideas. *Arch Inst Cardiol Mex.* 1946;16:531.
22. Osler W. On a remarkable heart murmur, heard at a distance from the chest wall. *Med Times Gaz Lond.* 1880;2:432.
23. Flint A. On cardiac murmurs. *Am J Med Sci.* 1862;44:23.
24. Berman P. Austin Flint—America's Laennec revisited. *Arch Intern Med.* 1988;148:2053.
25. Williams X. Comment in discussion of case of patent ductus arteriosus with aortic valve disease, coarctation of aorta and infective endocarditis, reported by Babington. *Lond Med Gaz.* 1847;4:822.
26. Gibson GA. Persistence of the arterial duct and its diagnosis. *Edinb Med J.* 1900;8:1.

27. Skoda J. *Abhandlung uber Perkussion and Auskultation.* Vienna: L. W. Seidl und John. Sechste Auflage, 1864.
28. Potain PC. Des mouvements et de bruits qui se passent dans, les veines jugulaires. *Bull Mem Soc Med Hop.*Paris: 1867;4:3.
29. Nishimura RA, Tajik AJ. The Valsalva maneuver revisited. *Mayo Clin Proc.* 1986;61:211.

7 The Chest

The pulmonary circulation was discovered by Michael Servetus (Miguel Servet), who was born in Villanueva de Sigena in northeast Spain. The date of his birth is disputed (1507 or 1511), but the date of death is beyond doubt. The original draft of Servetus' seminal manuscript appeared before 1546, and was published as his *Christianismi Restitutio* in 1553.[1] Because his books had theological connotations, Servetus was accused by the Calvinists of heresy. He was arrested in Geneva, and in 1553, was burned at the stake together with copies of his *Restitutio.* Servitus was burned slowly to increase his agony.

Michael Servetus wrote:

> The heart is the first organ that lives. The vital spirit is engendered by the mingling of inspired air with the more subtle portion of the blood which the right ventricle of the heart communicates to the left. This communication, however, does not take place through the septum, partition or midwall of the heart, as commonly believed, but by another admirable contrivance, the blood being transmitted from the pulmonary artery to the pulmonary vein, by a lengthened passage through the lungs, in the course of which it is elaborated and becomes a crimson colour. Mingled with the inspired air in this passage, and freed from its fuliginous vapors by the act of expiration, the mixture being now complete in every respect, the blood becomes a fit dwelling-place of the vital spirit.[1]

Servetus emphasized:

> It is in the lungs, consequently, that the mixture of the inspired air with the blood takes place, and it is in the lungs also, not in the heart, that crimson colour of the blood is acquired. The vital spirit is at length transfused from the left ventricle of the heart to the arteries of the body at large.[1]

With intuitive genius, Servetus proposed:

> Within the lungs we find a new kind of vessels proceeding from the arteries to the veins. The blood is distributed to those extremely minute vessels or capillary arteries.[1]

Servetus' *Restitutio* appeared three quarters of a century before William Harvey's *De Motu Cordis et Sanguinis* (1628), and a century before Marcello Malpighi published his discovery of the capillary circulation (*De Pulmonibus Observationes Anatomicae,* 1661).[2] It has been persuasively argued that the story of the circulation was completed by Malpighi, one of Italy's most gifted scientists. Malpighi also established the function of the uterine placenta, stating in his *De Pulmonibus*, that "the lungs of fetus were at rest," and that "a certain mass called the uterine placenta" acted vicariously for the lungs.

The only skeletal muscles upon which life depends are the respiratory muscles, especially the diaphragm. Gian Alfonso Borelli (1608–1679), a teacher of Malpighi, applied the laws of mechanics, and understood the importance of the diaphragm and intercostal muscles in respiration and ventilation.

The respiratory system comprises two essential components: (1) the lungs for gas exchange and (2) a pump that ventilates the lungs.[3] The pump consists of rib cage muscles, accessory muscles, abdominal muscles, and the diaphragm.[3] The external and internal intercostal, parasternal, interchondral, scalene, and sternocleidomastoid muscles act directly on the rib cage. The diaphragm has three functionally and anatomically distinct segments: (1) muscle fibers that originate from the costal margin and sternum and insert into the central tendon; (2) crural muscle fibers that originate from the vertebral column and insert into the central tendon; and (3) the central tendon itself.[3] The costal margin of the diaphragm displaces the rib cage and abdomen while inflating the lungs. Contraction of the crural component has no effect on the rib cage because crural fibers are not so attached. Instead, contraction of the crural portions of the diaphragm functions primarily to lower the central tendon and displace the abdominal wall, simultaneously inflating the lungs. If inspiration is performed entirely with rib cage and accessory muscles while the diaphragm remains relaxed, abdominal pressure falls and the abdominal wall moves inward rather than outward.

Breathing with intercostal or accessory muscles alone is inefficient, and does not normally occur. During quiet breathing, the diaphragm is the major component of the respiratory mechanism. In the supine position, normal respiratory movements are largely abdominal (diaphragmatic). In the upright position during normal quiet breathing, the diaphragm still predominates, but with a larger contribution from the muscles of the rib cage. The relative contributions of rib cage vs diaphragmatic/abdominal muscles is demonstrated by comparing thoracic and abdominal breathing movements when the flat of one hand is placed on the abdomen and the flat of the other hand is placed on the chest. This is a useful means of persuading patients that augmentation of rib cage (chest) muscles results in the inefficient, deep, sighing inspiratory efforts of anxiety-related hyperventilation.

It axiomatic that the physical examination of the heart and circulation includes the *chest*—its appearance, movements, palpation, percussion, and auscultation. Appearance (see Chapter 2) includes the *posterior* thorax–scoliosis, kyphosis, kyphoscoliosis, and loss of thoracic curvature (Fig. 7–1*A*)—and the *anterior* thorax (pectus carinatum, Fig. 7–1*B*), pectus excavatum, and the "barrel chest" (Fig. 7–2).

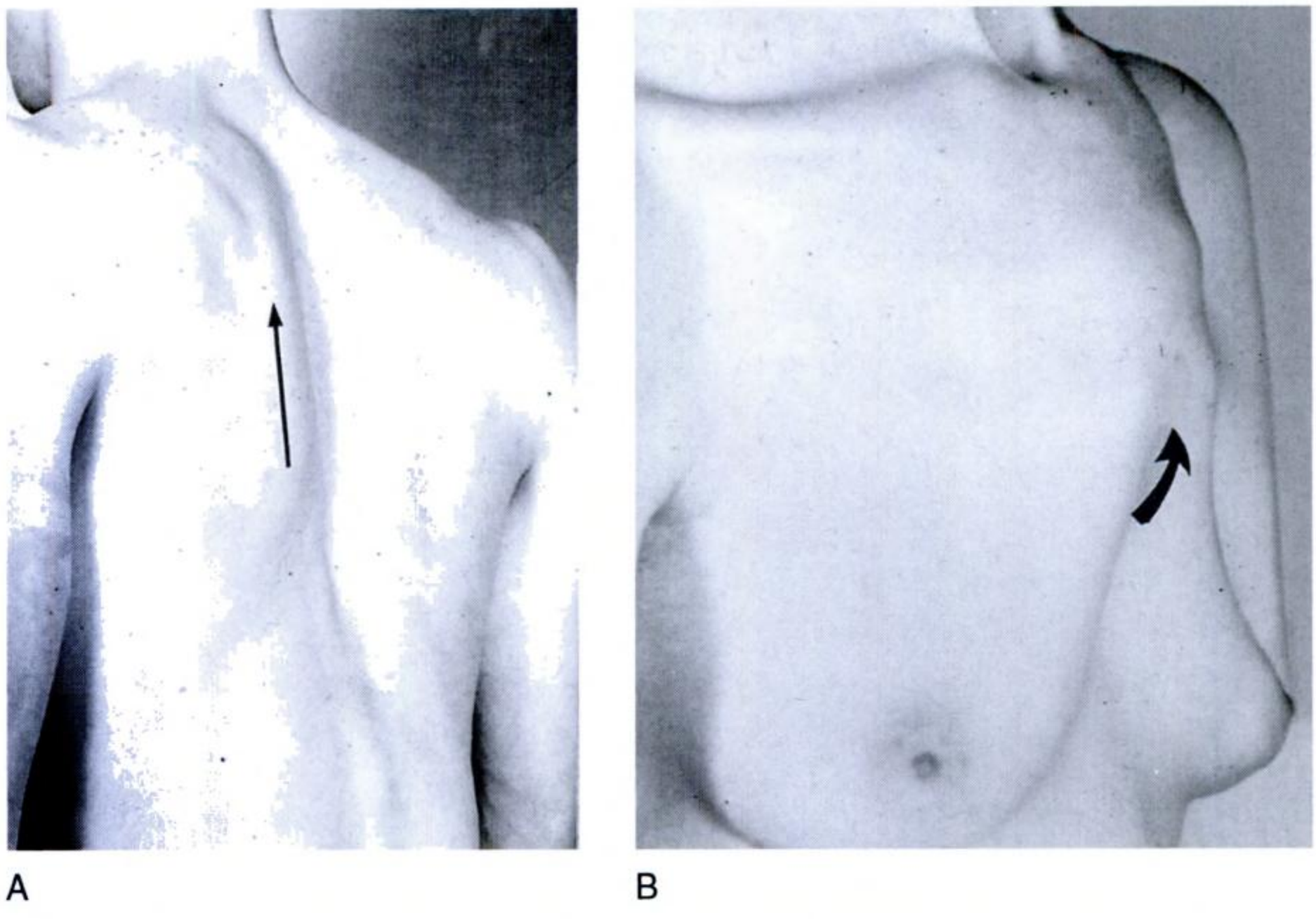
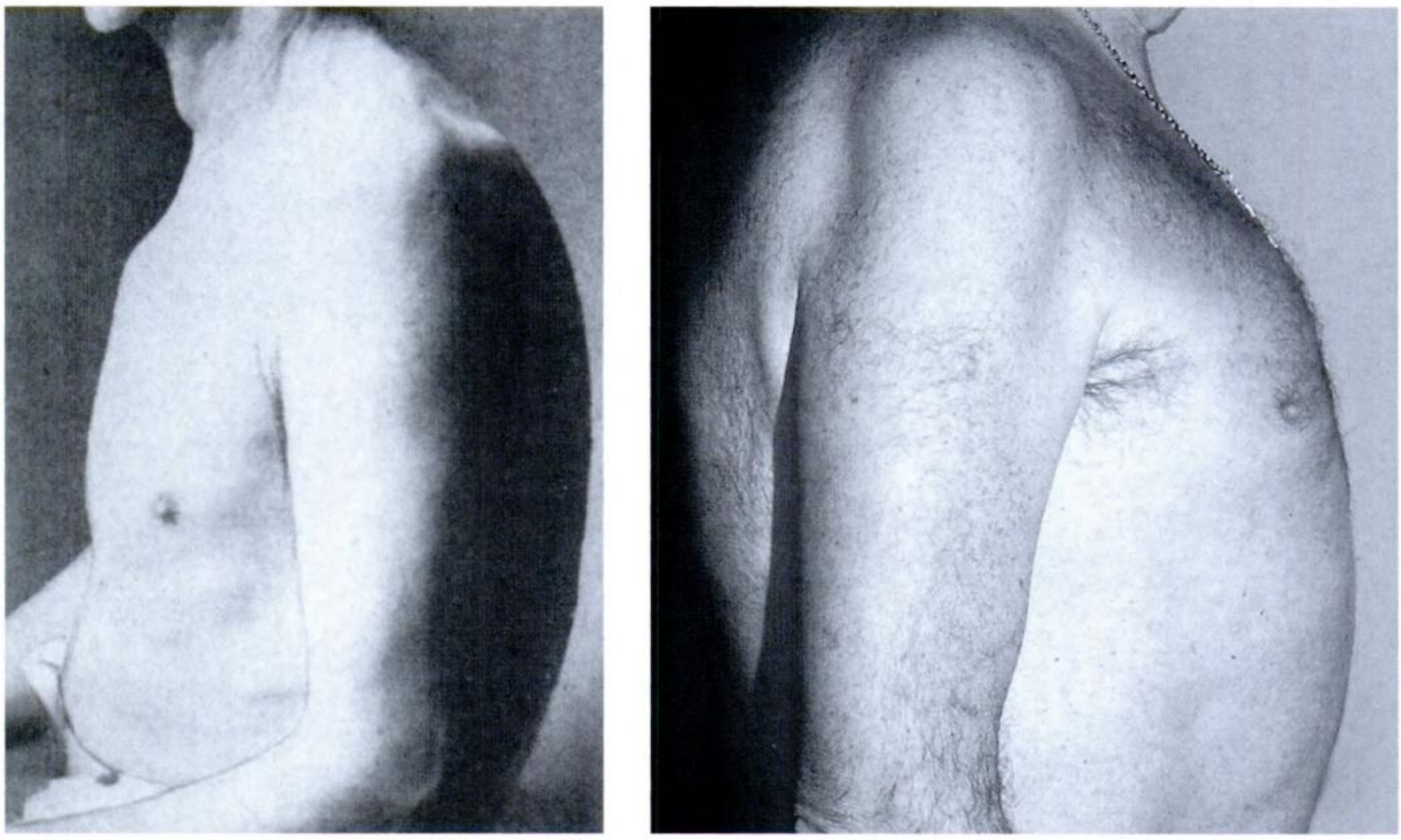


Figure 7–1 *A,* Absence of thoracic kyphosis (straight back, *vertical arrow*) in an otherwise normal young male. *B,* Marked pectus carinaturn (*curved arrow*) in a young female with Marfan syndrome.

RESPIRATORY MOVEMENTS should be assessed when the patient is unaware of the examiner's intent. The patient should *not* be told to "breathe normally," an instruction that is likely to result in a self-conscious change in breathing pattern. Respiratory movements depend in part on the position assumed to achieve the greatest comfort while breathing. Assessment should include rib muscles, accessory muscles, and the diaphragm. In an infant with pulmonary edema, an increase in the work of breathing is typically manifested by retraction of the supraclavicular fossae and intercostal muscles, and by inspiratory nasal flaring. *Orthopnea* (*orthos* = straight, *pnoia* = breath) implies that sitting upright or semi-upright is the most comfortable position. *Trepopnea* (*trepein* = to turn, plus *pnoia* = breath) refers to a condition in which breathing is most comfortable when the patient turns into a right or left recumbent position.

Once the patient is comfortable, the frequency and depth of respiration are observed. Respiratory frequency is in part age-related. The relatively rapid and somewhat irregular breathing of a normal newborn contrasts with the slower rhythmic breathing of the normal adult. Respiratory excursions can be abnormally slow (bradypnea) or abnormally rapid (tachypnea), or may transiently cease altogether (apnea). The depth of a respiratory excursion can be excessive (hyperpnea) or shallow. Hyperpnea can be persistent (the Kussmaul breathing of metabolic acidosis) or episodic (the intermittent, deep sighing



A B

Figure 7–2 *A,* "Barrel chest due to chronic bronchitis and emphysema."[15] *(*From Cabot RC: Physical Diagnosis. New York, William Wood and Co., 1915.) *B,* Barrel chest with striking increase in anteroposterior chest dimensions, anterior bowing of the sternum, and an increase in thoracic kyphosis.

excursions of hyperventilation). Inhalation and exhalation may be effortless, or labored and prolonged. A few examples suffice.

Tachypnea typically announces heart failure in infants, and is accompanied by labored breathing manifested chiefly by inspiratory efforts that call into play accessory muscles that produce subcostal and suprasternal retraction (see above). In adults, the tachypnea of acute pulmonary edema is accompanied by inspiratory and expiratory gurgling, cough, pink frothy sputum, flaring nostrils, and labored use of accessory respiratory muscles (see above). Tachypnea is a common and sometimes insidious sign of pulmonary embolism.

Periodic breathing takes different forms, among the commonest is the deep, sighing inspiratory effort of psychophysiologic hyperventilatory dyspnea in otherwise normal but anxious patients. The deep inspiratory sighs principally utilize rib cage muscles, and are associated with the sensation of an unfulfilled, unsatisfying breath (see above). A celebrated type of periodic breathing was described by William Stokes, and subsequently came to be known as Cheyne-Stokes respiration:

> The decline in the length and force of the respirations is as regular and remarkable as their progressive increase. The inspirations become each one less deep than the preceding, until they are all but imperceptible, and then the state of apparent

(consolidation) that increases transmission. Conversely, tactile fremitus is reduced by obesity (Fig. 7–4), pleural effusion, pneumothorax, or the hyperinflated lungs of emphysema (Fig. 7–2). Tactile fremitus, is evaluated to the right and left of the midline in the posterior chest, and is enhanced by a loud deep voice.

Localized pressure elicits noncardiac musculoskeletal *chest wall* pain. Pressure is selectively applied to each chondrosternal junction while asking the patient to compare the left to the right. Musculoskeletal pain thus elicited is typically to the *left* of the sternum, but may be submammary. The site of tenderness often coincides with the clinical complaint of localized, sharp chest pain.

The third role of thoracic palpation is to determine the relative degrees of *inspiratory chest excursions.* The examiner places the flats of both hands against the patient's back with thumbs parallel to the midline at approximately the level of the tenth rib. The skin beneath the thumbs is moved toward the midline. The patient is then asked to inhale deeply while movements of the thumbs away from the midline are observed. Normal movement is symmetric. Asymmetric movement of the thumbs away from the midline signifies that the chest on the side of lesser movement does not expand normally during inhalation.

PERCUSSION AND AUSCULTATION

Thoracic percussion originated with Leopold Auenbrugger (Fig. 7–5), who observed his father tapping wine barrels (Fig. 7–6) to determine their fluid level. It occurred to Auenbrugger that the same technique could be applied to the human thorax:

> The method of *percussion* is founded on the property possessed by the human thorax, in common with most hollow bodies, of giving out certain sounds when struck in a particular way.[8]

In 1761, Auenbrugger, an unassuming junior physician at the Vienna Hospital, published his *Inventum Novum Ex Percussione Thoracis Humani.*[8] Josef Skoda heralded the discovery of percussion as the beginning of modern diagnosis, but the epochal work lay dormant until Corvisart, physician to Napoleon, published an elaborate 480 page French edition of Auenbrugger's unpretentious 95 page book. Now let Auenbrugger speak for himself:

> I here present the reader with a new sign which I have discovered for detecting diseases of the chest. This consists in percussion of the human thorax, whereby, according to the character of the particular sounds thence elicited, an opinion is formed of the internal state of that cavity.
>
> The sound thus elicited from the healthy chest, resembles the sound of a drum covered with a thick woolen cloth or other envelop. To be able justly to appreciate the value of the various sounds elicited from the chest in cases of disease, it is necessary to have learned by experience on many subjects, the modification of sound produced by the habit of the body inasmuch as these various circumstances modify the sound very considerably. A clear and equal sound solicited from both sides of

Figure 7–5 Leopold Auenbrugger (1722–1809). (Courtesy of Special Collections Division, Biomedical Library, UCLA Medical Center.)

Figure 7–6 Burgundian wine casks. Auenbrugger witnessed his father's practice of percussing wine barrels to determine the level of their fluid content (see Chapter 5). (Photographed by the author.)

Four types of breath sounds are normally heard during quiet breathing: (1) vesicular, (2) bronchial, (3) bronchovesicular, and (4) tracheal. Soft low frequency *vesicular breath sounds* that resemble a gentle rustling or sigh are audible throughout most of the chest. The inspiratory phase of vesicular breath sounds is longer and more clearly audible than the expiratory phase that follows without interruption. Vesicular breath sounds are reduced or absent in the obese (Fig. 7–4) and in the hyperinflated lungs of pulmonary emphysema (Fig. 7–2). *Bronchial breath sounds*, which are normally confined to the manubrium, are loud and high-pitched with a hollow quality, and are usually interrupted by a pause between the inspiratory and expiratory phases. In normal neonates, breath sounds are bronchial, and are louder over the periphery of the lungs. *Bronchovesicular* breath sounds are intermediate between vesicular and bronchial, as the term implies, are normally confined to the vicinity of the first and second intercostal spaces anteriorly or between the scapulae posteriorly, and are usually more obvious on the right. The inspiratory and expiratory phases of bronchovesicular breath sounds are either continuous or separated only briefly, with each phase about the same duration and quality. *Tracheal* breath sounds are relatively unimportant, and are not routinely sought.

The time-honored term "rale" has been replaced in pulmonary terminology by the term "crackle." *Rale* and *crackle* are therefore synonymous, with cardiologists preferring *rale*, and pulmonologists *crackle*. Rales or crackles are classified as coarse or fine. The mechanism responsible for their genesis is believed to be the process of bubbling as gas flows through secretions, with coarse rales or crackles originating in larger airway secretions, and fine rales or crackles originating in smaller airway secretions (pulmonary edema).

Wheezes, also referred to as *rhonchi*, are relatively pure, high-frequency musical events generated by airflow within narrowed bronchi, and are augmented by exhalation, especially rapid, forced exhalation. Wheezes and rhonchi are the result of the bronchospasm or bronchial swelling of asthma or emphysema. Secretions characterize pulmonary edema.

The *pleural rub* was described by Hippocrates as "a sound like that made by leather, a grating sound produced on surfaces, the movements of which are impeded by frictional resistance."[11] Pleural rubs are best heard at the end of inhalation and at the onset of exhalation. The search must be meticulous. Firm pressure of the stethoscopic diaphragm is applied with the patient in a comfortable sitting position with the elbows away from the chest wall.

A pleural rub in a cardiac patient is usually the result of a pulmonary embolus with infarction. Special attention should focus on the lower lobes—axillae and back—which are the usual sites of embolic infarcts.

Tubular breathing is an abnormal breath sound transmitted via patent airways from a region of parenchymal consolidation. *Voice-generated* signs include *egophony, pectoriloquy, and bronchophony* (increased intensity and a nasal quality of the spoken voice heard through consolidated lungs, "e-e" is heard as "a-a"), *bronchophony* refers to the audible voice, and *pectoriloquy* to the whispered voice. The patient repeats in a quiet voice "one-two" or "ninety-nine" or "e-e" while the examiner listens with the diaphragm of the

stethoscope. The patient then repeats in a whispered voice. Voice-generated sounds are attenuated by transmission through normal air-filled lungs, and have a muffled quality with indistinct words. *Egophony refers to an increased intensity and nasal quality of the spoken voice heard through consolidated lung. "e-e" is heard as "a-a." Bronchophony* and *pectoriloquy* indicate that the spoken voice is transmitted with increased intensity and pitch so that the syllables are distinct and easily recognized. Egophony, bronchophony, and whispered pectoriloquy are auscultatory manifestations of the same acoustic properties of consolidated lungs, and therefore have the same diagnostic significance. A common site of consolidation in cardiac patients is the compression atelectasis above a pleural effusion.

The *mediastinal crunch* (Hamman sign) is a crunching, crackling, or popping sound generated by breathing movements and cardiac contraction in the presence of mediastinal emphysema. A common setting in which a crunch is detected is immediately after operation via a midline sternotomy that introduces air into the mediastinum. Air is also introduced into the mediastinum by chest trauma or cardiopulmonary resuscitation.

Thoracic auscultation occasionally detects a *cardiorespiratory murmur* which was known to Laennec, but characterized by Richard Cabot:

> Cardiorespiratory murmurs may be produced without any adhesion of the lung to the pericardium under conditions not currently understood. Such murmurs may be heard under the left clavicle or below the angle of the left scapula, as well as near the apex of the heart—less often in other parts of the chest. Cardiorespiratory murmurs may be either systolic or diastolic, but the vast majority of cases are systolic. The area over which they are audible is usually a very limited one. They are greatly affected by position and by respiration, and are heard most distinctly if not exclusively during inspiration, especially at the end of that act.[15]

James Hope's experience with two university students is picturesque:

> Both wore very tight waistcoats, preventing the expansion of the lower ribs. During this state of breathing, a bellows murmur existed in both. In both, the murmur ceased entirely when, unbuttoning their waistcoats and waistbands of their trousers, they breathed with the lungs naturally inflated. By alternating the circumstances, the murmur could be created or removed at pleasure. I presume therefore that it proceeded from a cause exterior to the heart.[16]

The mechanism responsible for the cardiorespiratory murmur remains unclear, but the location, timing, and relation to respiration are as Cabot described.[15]

REFERENCES

1. Servetus M. *Christianismi Restitutio.* Vienna: Balthasar Arnollet, 1553.
2. Malpighi M. De Pulmonibus observations anatomicae. Bononiae, 1661. (English translation by Young J. *Proc Roy Soc Med* 23:1, 1929–1930. see previous edition and style accordingly
3. Roussos C, Macklem PT. The respiratory muscles. *N Engl J Med.* 1982;307:786.
4. Stokes W. *Diseases of the Heart and Aorta.* Dublin, Ireland: Hodges and Smith, 1854.

5. Mackenzie J. *Diseases of the Heart*. London: Henry Frowde, 1908.
6. Burwell CS, Robin ED, Whaley RD, Bicklemann AG. Extreme obesity associated with alveolar hypoventilation—A pickwickian syndrome. *Am J Med*. 1956;21:811.
7. Macklem PT, Macklem DM, DeTroyer A. A model of respiratory muscle mechanics. *J Appl Physiol*. 1983;55:547.
8. Auenbrugger L. *Inventurn Novum Ex Percussione Thoracis Humani*. Vienna: Trattner, 1761.
9. Auenbrugger L. *On Percussion of the Chest*. Vienna: 1761. Translated by John Forbes. London: T and G Underwood, 1824.
10. Ewart W. Practical aids in the diagnosis of pericardial effusion in connection with the question as to surgical treatment. *Br Med J*. 1896;1:717–721.
11. The Genuine Works of Hippocrates. Translated from the Greek by Francis Adams. In: Major RH. *Classic Descriptions of Disease*. Springfield, IL: Charles C Thomas, 1948.
12. Bowditch HI. *The Young Stethoscopist*. New York: Samuel S. and William Wood, 1848.
13. Osler W. *Sir Thomas Browne. An Alabama Student and Other Biographical Essays*. London: Oxford University Press, 1908.
14. Laennec RTH. *A Treatise on the Diseases of the Chest*. London: T. and G. Underwood, 1821.
15. Cabot RC. *Physical Diagnosis*. New York: William Wood, 1915.
16. Hope J. *A Treatise on the Diseases of the Heart and Great Vessels*. London: John Churchill, 1839.

8 The Abdomen

Abdominal examination in a cardiac patient includes appearance (see Chapter 2), movements (see Chapter 7), percussion, palpation, and auscultation. The abdomen should be exposed entirely with the patient supine and as close to horizontal as comfort permits. Examination commences from the right side of the bed or examining table. Areas examined include the upper right and upper left quadrants, the lower right and lower left quadrants, three central abdominal areas—gastric, umbilical, suprapubic—and six lateral areas—right and left hypochrondrium, right and left lumbar, and right and left inguinal.

APPEARANCE AND MOVEMENTS

Physical appearance of the abdomen includes contour, striae, and incisional scars. Violaceous striae in the lower abdominal wall of a hypertensive patient suggest Cushing syndrome as the cause of hypertension. A right upper quadrant scar in a cyanotic patient suggests cholecystectomy because of calcium bilirubinate gallstones. The fluid of ascites distends the flanks when the patient is supine, and distends the lower abdomen when the patient stands. An age-related increase in abdominal girth afflicts both males and females.

Movements of the abdomen reflect the relative contributions of diaphragm vs rib cage muscles during the process of breathing (see Chapter 7).

PERCUSSION

Percussion establishes *situs* of the abdominal viscera by identifying the gastric tympany of Traube's space on the left and hepatic dullness on the right in *situs solitus*, and *vice versa* in *situs inversus* (see Fig. 5–3). *Liver size* is estimated by percussing the upper and lower hepatic borders which are no more than 10 cm apart in the average normal adult. Percussion extended laterally from the zone of gastric tympany can detect the dullness imparted by an enlarged spleen (see below).

Percussion for detection of ascites requires special technique. In the flat supine position, fluid gravitates to the flanks which are dull to percussion, while the tympanitic center of the abdomen is occupied by air-filled intestine. A line of demarcation separates flank dullness from the tympanitic center. Once the line of demarcation has been established in the supine position, *shifting dullness* is elicited as the patient turns into a partial lateral decubitus position, while the examiner again determines the dull-to-tympanitic line of demarcation. Ascites causes the demarcation line to *shift medially* as the patient turns laterally. A transmitted fluid wave is sought in the flat supine position. The patient assists by compressing the center of the abdomen with the ulnar surface of either hand. The examiner taps one flank and palpates the other flank in anticipation of a fluid wave.

PALPATION

Abdominal palpation in a cardiac patient, is concerned chiefly with the liver, spleen, abdominal aorta, and kidneys. Before attempting to palpate a liver edge, its upper and lower margins should be estimated by percussion as just described. The palm of right hand of the examiner is placed below the expected inferior hepatic border lateral to the rectus muscle, while the left hand is placed posteriorly diametrically opposite (Fig. 8–1*A*). Flexion of the knees or placement of a pillow under the knees relaxes the abdominal wall and improves the sensitivity of palpation. The patient inhales slowly but deeply as the fingertips of the right hand are pressed upward and inward, anticipating descent of the liver. Care should be taken to palpate gently, because the liver is tender in congestive hepatomegaly. The liver edge is assessed for consistency, transverse extent and level below the right costal margin expressed in fingerbreadths or in centimeters. The right lobe of the liver is more readily palpable than the left.

Horizontal extension of the inferior margin of the liver from the right upper quadrant across the epigastrium represents a *transverse liver* rather than hepatomegaly,[1] and is an important physical sign of the visceral heterotaxy of left or right isomerism (Fig. 8–1*B*).

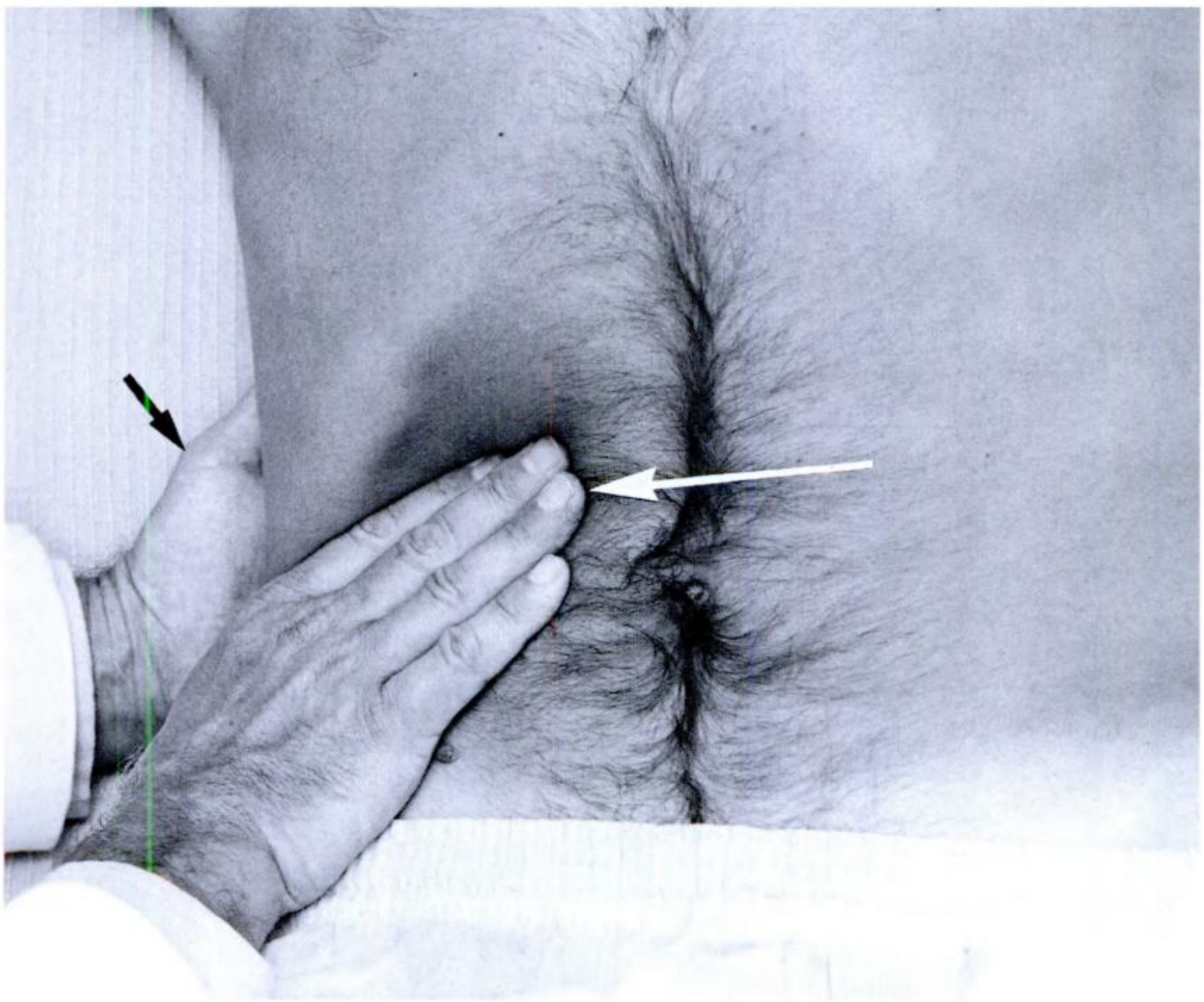
Careful palpation of the inferior margin of the liver during held inspiration permits detection of transmitted pulsations from the right atrium via the inferior cava (Fig. 8–2). Mackenzie accurately recorded a liver pulse and stated:

> When there has been enlargement of the liver, in all my cases of valvular disease, I could demonstrate that the liver pulsated. In cases with easily demonstrated liver enlargement and lax abdominal walls, this pulsation may easily be detected. The hand laid over the enlarged liver is gently heaved up and down.[2]

The eratic breathing pattern in infants does not lend itself to palpation during inspiration, and the congested neonatal liver is rounded and less distinct than in older children and adults. Palpation commences once the infant is quiet (Fig. 8–3). The hand of the examiner is placed gently on the abdomen, allowing the infant time to adjust to the contact. The right thumb is applied to the right upper quadrant for the liver while the index finger palpates the left upper quadrant for the spleen.

Palpation of the *spleen* in adults requires examination in the supine and right lateral decubitus positions. It is useful to begin by percussion lateral to Traube's space of gastric tympany. (Named after Ludwig Traube, although it was first described by a pupil in 1868.) Dullness over Traube's space heightens suspicion of splenomegaly, although obliteration can be caused by a full stomach, a left pleural effusion, and enlargement of the left lobe of the liver.

The examining table or bed should be horizontal and the patient supine with the physician on the right at a level that permits the right hand to be applied to the abdomen without flexing the wrist, thus avoiding reduction in sensitivity of the fingertips. These adjustments are best achieved when the examining table is adjustable or when the examiner sits or stands at the bedside. The palm of the right hand is placed below the left costal margin and pressed inward and upward toward the left shoulder, while the left hand is applied to the left posterior thorax to displace the spleen forward. The patient inhales deeply and slowly while the fingertips of the right hand anticipate descent of the spleen. If

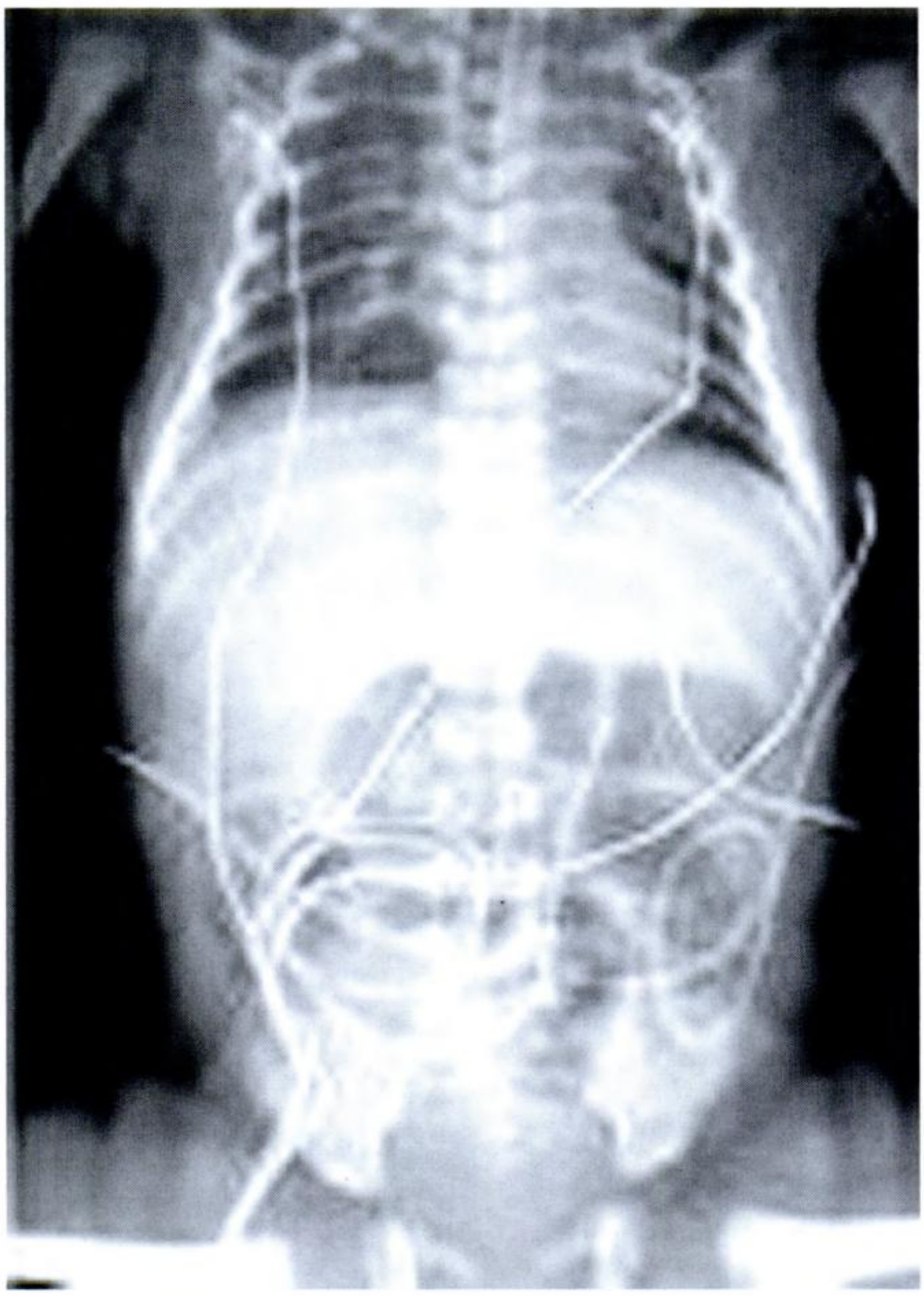


A

Figure 8–1 *A,* Palpation of the liver. The patient lies supine with knees flexed to relax the abdomen. The flat of the examiner's right hand (*white arrow*) *is* placed on the right upper quadrant just below the anticipated inferior margin of the liver, while the left hand is placed diametrically opposite (*black arrow*). *B* (*see next page*), X-ray of chest and abdomen showing the transverse liver of visceral isomerism. *Continued*

the spleen cannot be detected in the supine position, palpation should be repeated in the right lateral decubitus (Fig. 8–4), a position that moves the spleen downward and anterior. The right hand is placed beneath the left costal margin and the left hand behind the posterior thorax, as shown in Figure 8–4. The examiner's right wrist is not flexed, thus improving sensitivity of the fingertips. These maneuvers are repeated while readjusting the tilt of the patient and the position of the palpating right hand to achieve optimal tactile sensitivity.

A palpable spleen tip is a subtle physical sign that requires diligence and practice to elicit. Palpation should proceed toward the midline, because the spleen enlarges diagonally from the left hypochondrium toward the umbilicus. The principal settings in which splenomegaly is anticipated in cardiac patients are congestive heart failure and infective endocarditis. In the later case, the spleen is likely to be tender, a feature that calls for a gentle touch. Palpation of the spleen in infants is best achieved with the baby lying flat as the examiner applies the index finger of the right hand to the left upper



B

Figure 8–1 *Continued*

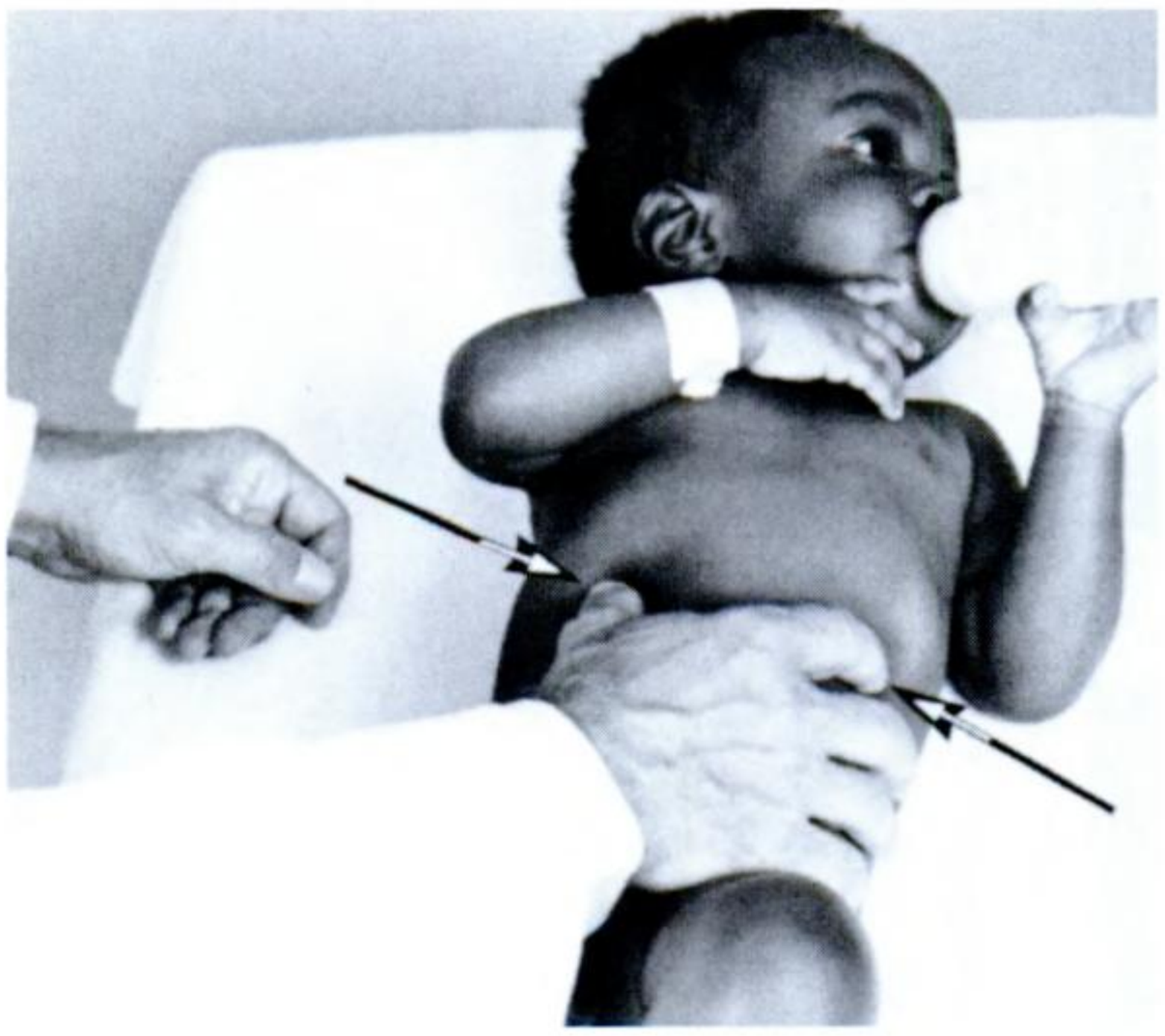


Figure 8–3 Palpation of liver and spleen in an infant. The examiner's right thumb is applied to the right upper quadrant (*left arrow*) while the index finger palpates the left upper quadrant (*right arrow*). Attention should be paid to each site, gently altering pressure first with the thumb, then with the finger as the infant breathes naturally and the liver and spleen descend during inspiration.

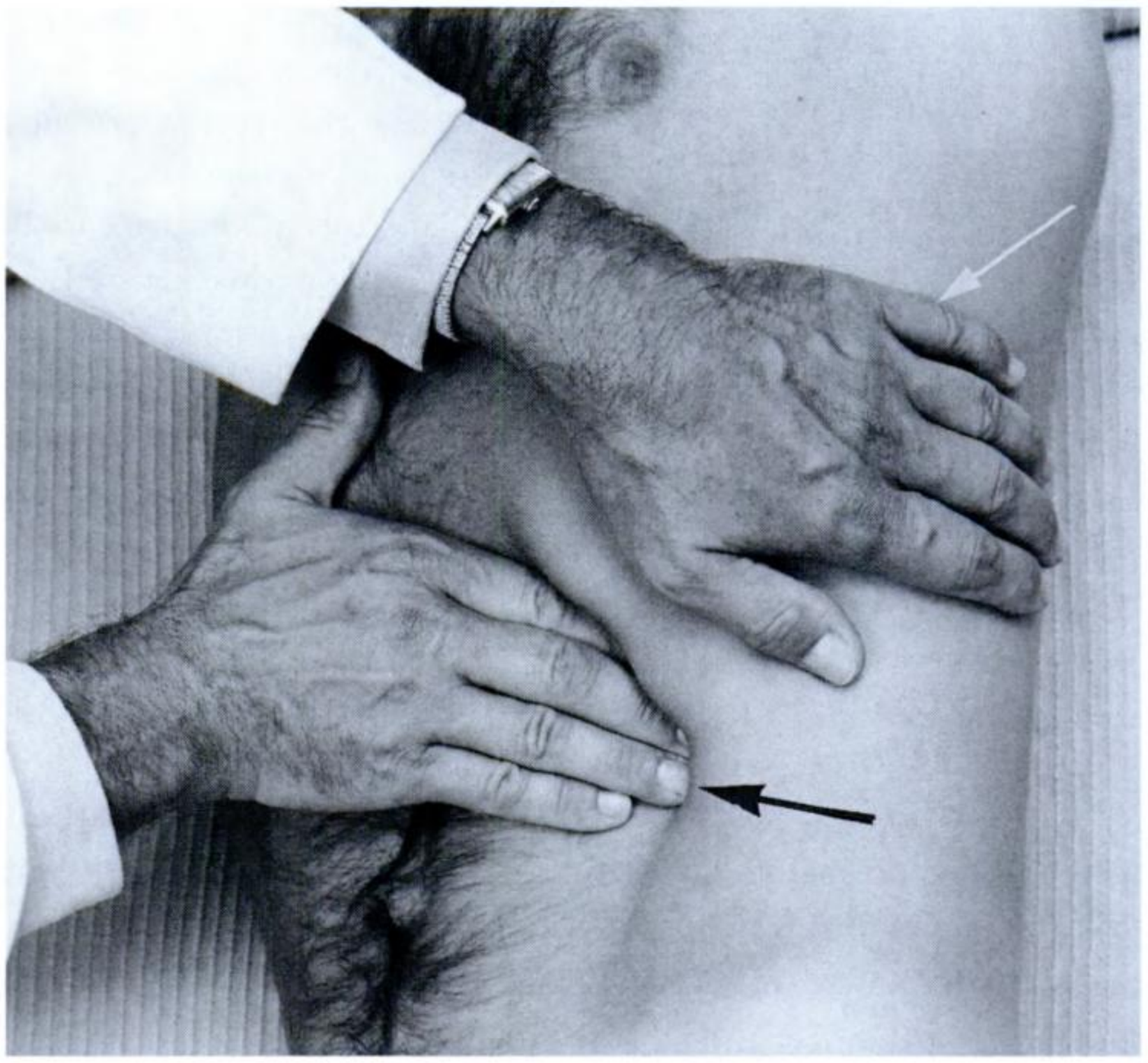


Figure 8–4 For palpation of the spleen, the patient turns toward the examiner so that the left flank is raised. The palm of the right hand (*black arrow*) is applied to the left upper quadrant without bending the wrist, while the left hand (*white arrow*) provides support diametrically opposite.

umbilicus, recalling that a normal abdominal aorta can be tender. More important, however, is palpation of the abdominal aorta *below* the umbilicus and to the left of midline. The patient should breathe quietly, utilizing thoracic respiratory muscles in order to minimize an inspiratory increase in abdominal girth. The fingertips of the examiner's right hand are applied to the left of center just below the umbilicus. Pressure is gently and gradually increased in search of an aortic pulsation. When seeking to identify the lateral margin of an abdominal aortic aneurysm, the fingertips of the right hand should attempt to "roll" the margin of the aorta. Once the margin is identified, bimanual palpation permits an experienced observer to estimate the size of the aneurysm that can be assessed by observing systolic separation of fingers placed on either side of the pulsating aorta. Width should not exceed 2.5 cm, allowing for skin thickness. Remember that an enlarged or an enlarging abdominal aortic aneurysm is tender if not painful, and when leaking, causes retroperitoneal pain in the low back or in the posterior aspect of the left flank.

Palpation of the kidneys should be routine in adults, and is obligatory in hypertensive patients. Bimanual palpation is performed in a supine position. For palpation of the right kidney, the palm of the right hand is applied to the right upper quadrant with the fingertips just below the costal margin. The left hand is placed behind the flank diametrically opposite. The patient is instructed to take a gradual deep breath while the fingertips anticipate the descent of the rounded edge of the lower pole of the kidney. Because the right kidney is lower than the left, its lower pole is occasionally palpable in normal persons. When palpating the *left* kidney, care should be taken to distinguish its lower pole from a palpable spleen. The technique for palpating the left kidney is *similar* to that just described for the spleen. The right hand is applied to the left upper quadrant with fingertips below the left costal margin. The left hand is applied to the left flank diametrically opposite. When palpating the right or left kidney, the hands of the examiner should be approximated *during* deep inhalation; if the hands are brought together *before* inspiration, the descent of the kidney is blocked, and may therefore be missed. These techniques sometimes permit palpation of the kidney by either the posterior or anterior hand of the examiner. Polycystic kidneys are readily palpable, and must be approached gently because of their delicate, cystic surfaces. Huge polycystic kidneys are large enough to be visible through the abdominal wall.

AUSCULTATION

Auscultation is the next step in the abdominal examination. Josef Skoda (1853) recognized the importance of auscultation of the abdomen, to which he devoted a section of his book, *A Treatise on Auscultation and Percussion*.[3]

With the patient supine, the diaphragm of the stethoscope is gently but firmly applied over the abdominal aorta beginning in the epigastrium, proceeding inferiorly below the umbilicus to the right and left of midline, and then toward the left and right ileofemoral areas along the courses of the iliac arteries. The degree of stethoscopic

pressure is adjusted to achieve maximal access to the abdominal aortic wall and its bifurcation. In the inguinal regions, the bell of the stethoscope is more practical than the diaphragm.

Renal arterial murmurs are uncommon, but should be sought routinely in hypertensive adults. The examination is best conducted in a quiet room, because renal arterial murmurs tend to be soft and high frequency. Renal arterial auscultation is undertaken with the patient sitting with trunk relaxed to minimize interference from tensed muscles. The diaphragm of the stethoscope is applied firmly against the skin of the posterior flank, first over one kidney and then over the other. The patient is then reexamined in the *supine* position with the diaphragm of the stethoscope applied *anteriorly* to the right and left of the abdominal aorta, at levels appropriate for each renal artery. The murmur of renal artery stenosis is high-frequency systolic. Rarely, the continuous murmur of a renal arteriovenous fistula is detected (Fig. 8–5).

Another role of auscultation of the abdomen applies to the gravid uterus. In 1853, Josef Skoda observed that

> The sounds of the fetal heart can be heard over the uterus at the sixth month of pregnancy and become more distinct as the pregnancy advances. Their presence is a sure sign of the fetus being alive. It is possible, at times, to discover the presence of twins by the fetal pulse, in those cases where the two hearts do not pulsate with equal rapidity.[3]

Skoda also called attention to the placental bruit, but stated that it was not nearly so valuable a sign in the diagnosis of pregnancy as the sounds of the fetal heart.[3]

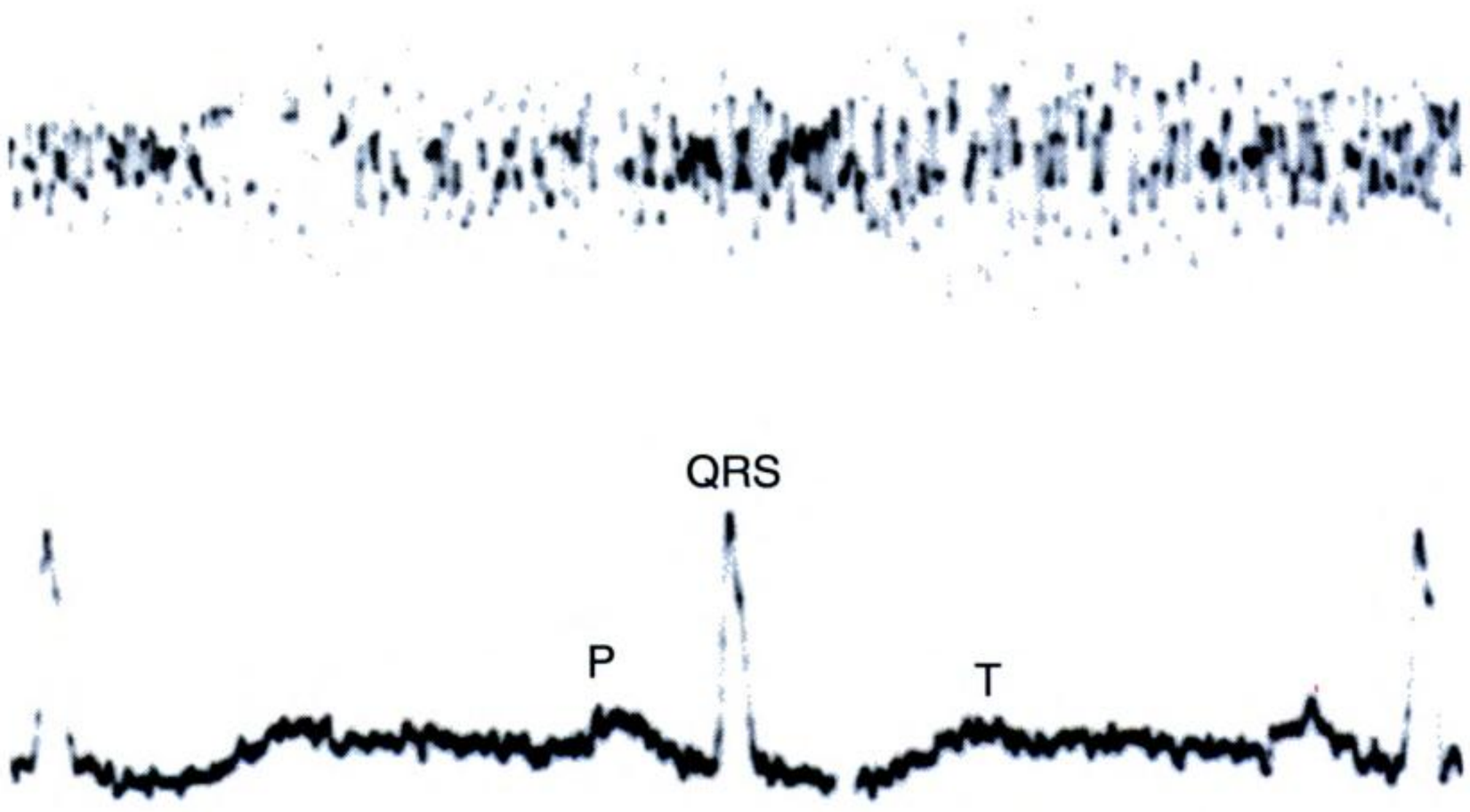


Figure 8–5 The continuous murmur of a congenital renal arteriovenous fistula in a 50-year-old woman. The murmur was heard in the flank.

REFERENCES

1. Perloff JK. *The Clinical Recognition of Congenital Heart Disease,* 5th ed. Philadelphia, PA: WB Saunders, 2003.
2. Mackenzie J. *The Study of the Pulse, Arterial, Venous and Hepatic, and of the Movements of the Heart.* Edinburgh: Young J. Pentland, 1902.
3. Skoda J. *A Treatise on Auscultation and Percussion.* London: Highley and Son, 1853.

Index

Note: Page numbers in *italics* indicate illustrations; page numbers followed by (t) refer to tables.

Fourth Edition

PHYSICAL EXAMINATION *of the* HEART *and* CIRCULATION

JOSEPH K. PERLOFF, MD

This outstanding resource emphasizes physical *examination*, rather than physical *diagnosis,* of the heart and circulation.

This book thoroughly explores the diagnostic value of cardiovascular physical signs. The cardiovascular physical examination is dealt with from birth to senescence and includes physical appearance, the arterial pulse, the jugular venous pulse, the peripheral veins, percussion, palpation and observation of the precordium, cardiac auscultation, the thorax, lungs and abdomen.

- All chapters are substantially revised
- New web site serves as a valuable audiovisual supplement to the chapters on physical appearance, the arterial pulse, the movements of the heart (percussion, palpation, and observation), and auscultation.
- Supplemental web site material on the jugular venous pulse includes Paul Wood's original audiovisual tape, narrated by Wood himself.

Visit: **www.pmph-usa.com/perloff** to gain access to video and sounds clips

PMPH-USA

People's Medical Publishing House
2 Enterprise Drive, Suite 509
Shelton, CT 06484
Telephone: 203-402-0646
Fax: 203-402-0854

ISBN 978-1-60795-023-3