I

Diagnostic Examinations And Procedures

CARDIOVASCULAR PHYSICAL EXAMINATION

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CHAPTER 1

Editor's Note to Readers: *For an excellent and more detailed discussion of the cardiovascular physical examination, read* Physical Diagnosis Secrets, *2nd edition, by Salvatore Mangione.*

1. What is the meaning of a slow rate of rise of the carotid arterial pulse?

A carotid arterial pulse that is reduced (*parvus*) and delayed (*tardus*) suggests the presence of *aortic valvular stenosis*. Occasionally, this also may be accompanied by a palpable thrill. If ventricular function is good, a slower upstroke correlates with a higher transvalvular gradient. In left ventricular failure, however, parvus and tardus may occur even with mild aortic stenosis (AS).

2. What is the significance of a brisk carotid arterial upstroke?

It depends on whether it is associated with *normal* or *widened* pulse pressure. If associated with the *normal pulse pressure*, a brisk carotid upstroke usually indicates two conditions:

- *Simultaneous emptying of the left ventricle into a high-pressure bed (the aorta) and a lower pressure bed*: The latter can be the right ventricle (in patients with ventricular septal defect [VSD]) or the left atrium (in patients with mitral regurgitation [MR]). Both will allow a rapid left ventricular emptying, which in turn generates a brisk arterial upstroke. The pulse pressure, however, remains normal.
- *Hypertrophic cardiomyopathy (HCM)*: Despite its association with left ventricular obstruction, this disease is characterized by a brisk and bifid pulse, due to the hypertrophic ventricle and its delayed obstruction. An example of the carotid pulsation in HCM and other conditions is given in Fig. 1.1.

If associated with the *widened pulse pressure,* a brisk upstroke usually indicates aortic regurgitation (AR). In contrast to MR, VSD, or HCM, the AR pulse has rapid upstroke *and* collapse.

3. In addition to aortic regurgitation, which other processes cause rapid upstroke and widened pulse pressure?

The most common are the hyperkinetic heart syndromes (high-output states). These include anemia, fever, exercise, thyrotoxicosis, pregnancy, cirrhosis, beriberi, Paget disease, arteriovenous fistulas, patent ductus arteriosus, AR, and anxiety—all typically associated with rapid ventricular contraction and low peripheral vascular resistance.

Examples of the carotid pulse waveform and its correlation to heart sounds are provided in Fig. 1.2.

4. What is pulsus paradoxus?

Pulsus paradoxus is an exaggerated fall in systolic blood pressure during quiet inspiration. In contrast to evaluation of arterial contour and amplitude, it is best detected in a peripheral vessel, such as the radial artery. Although palpable at times, optimal detection of the pulsus paradoxus typically requires a sphygmomanometer. Pulsus paradoxus can occur in cardiac tamponade and other conditions.

5. What is pulsus alternans?

Pulsus alternans is the alternation of strong and weak arterial pulses despite *regular rate and rhythm*. First described by Ludwig Traube in 1872, pulsus alternans is often associated with alternation of strong and feeble heart sounds (auscultatory alternans). Both indicate severe left ventricular dysfunction (from ischemia, hypertension, or valvular cardiomyopathy), with worse ejection fraction and higher pulmonary capillary pressure. Hence, they are often associated with an S_3 gallop. A tracing indicating pulses alternans is given in Fig. 1.3.

6. What is the Duroziez double murmur?

The *Duroziez murmur* is a to-and-fro double murmur over a large central artery—usually the femoral but also the brachial. It is elicited by applying gradual but *firm* compression with the stethoscope's diaphragm. This produces not only a systolic murmur (which is normal) but also a *diastolic* one (which is pathologic and typical of AR). The Duroziez murmur has 58% to 100% sensitivity *and* specificity for AR.

7. What is the carotid shudder?

Carotid shudder is a palpable thrill felt at the peak of the carotid pulse in patients with AS, AR, or both. It represents the transmission of the murmur to the artery and is a relatively specific but rather an insensitive sign of aortic valvular disease.

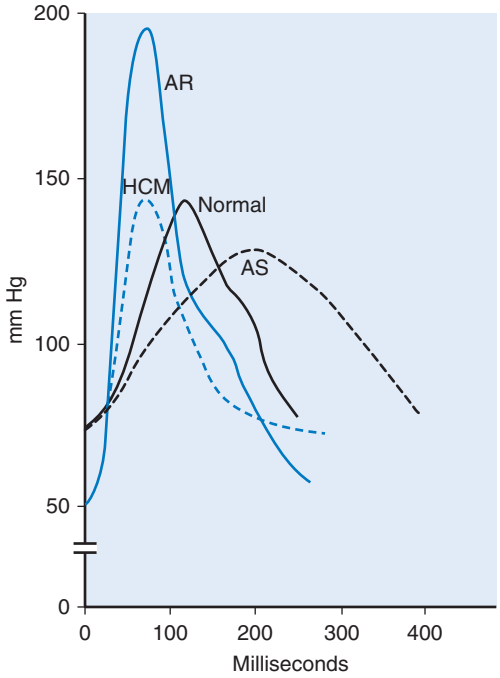

Fig. 1.1 The carotid pulsation. The waveform of the carotid pulse is characterized by the rate of rise of the carotid upstroke. In aortic regurgitation *(AR)*, the upstroke is rapid and followed by abrupt diastolic "collapse." In hypertrophic cardiomyopathy *(HCM)*, the upstroke is also rapid and the pulse has a jerky character. In aortic stenosis *(AS)*, the upstroke is slow with a plateau. (Adapted from Timmis, A. D., & Archbold, A. (2012). Cardiovascular system. In M. Glynn & S. M. Drake (Eds.), *Hutchison's clinical methods: an integrated approach to clinical practice* (23rd ed.). St. Louis, MO: Elsevier.)

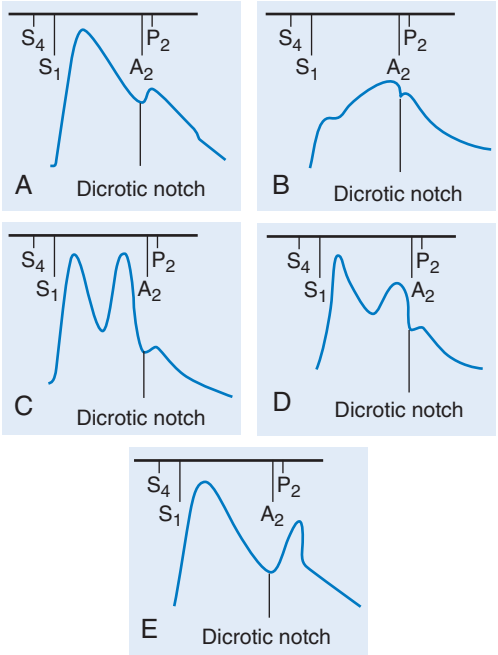

Fig. 1.2 Carotid pulse waveforms and heart sounds. **A,** Normal. **B,** Aortic stenosis: anacrotic pulse with slow upstroke and peak near S_2. **C,** Severe aortic regurgitation: bifid pulse with two systolic peaks. **D,** Hypertrophic obstructive cardiomyopathy: bifid pulse with two systolic peaks. The second peak (tidal or reflected wave) is of lower amplitude than the initial percussion wave. **E,** Bifid pulse with systolic and diastolic peaks as may occur with sepsis or intra-aortic balloon counterpulsation. A_2, Aortic component of S_2; P_2, pulmonic component of S_2. (**A–D,** From Chatterjee, K. (1991). Bedside evaluation of the heart: the physical examination. In K. Chatterjee & W. Parmley (Eds.), *Cardiology: an illustrated text/reference* (pp. 3.11–3.51). Philadelphia, PA: JB Lippincott; **E,** Braunwald, E. (2003). The clinical examination. In E. Braunwald & L. Goldman (Eds.), *Primary cardiology* (2nd ed., p. 36). Philadelphia, PA: WB Saunders.)

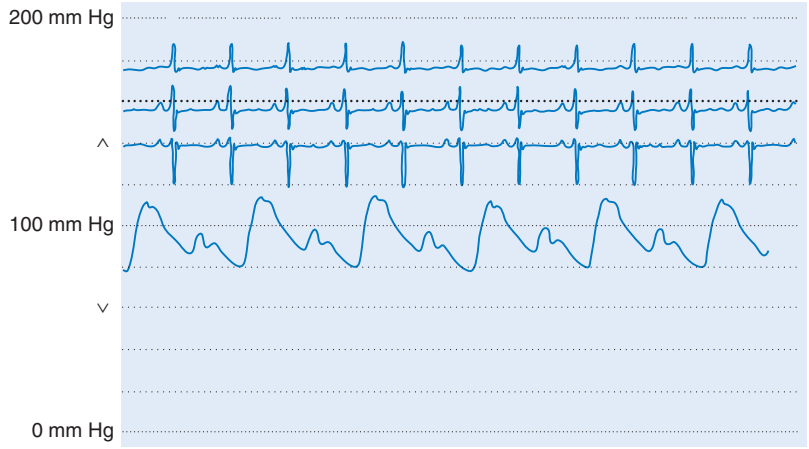

Fig. 1.3 Pulsus alternans in a patient with severe left ventricular systolic dysfunction. The systolic pressure varies from beat to beat, independent of the respiratory cycle. The rhythm is sinus throughout.

8. What is the Corrigan pulse?

The *Corrigan pulse* is one of the various names for the bounding and quickly collapsing pulse of AR, which is both visible *and* palpable. Other common terms for this condition include *water hammer*, *cannonball*, *collapsing*, or *pistol-shot pulse*. It is best felt for by elevating the patient's arm while at the same time feeling the radial artery at the *wrist*. Raising the arm higher than the heart reduces the intraradial diastolic pressure, collapses the vessel, and thus facilitates the palpability of the subsequent systolic thrust.

9. How do you auscultate for carotid bruits?

To auscultate for *carotid bruits*, place your stethescope's bell on the patient's neck in a quiet room; the patient should be completely relaxed. Auscultate from just behind the upper end of the thyroid cartilage to immediately below the angle of the jaw.

10. What is the correlation between symptomatic carotid bruit and high-grade stenosis?

This correlation is very strong. In fact, bruits presenting with transient ischemic attacks or minor strokes in the anterior circulation should be evaluated aggressively for the presence of high-grade (70%–99%) carotid stenosis, because endarterectomy markedly decreases mortality and stroke rates. Still, although the presence of a bruit significantly increases the likelihood of high-grade carotid stenosis, its absence does not exclude disease. Moreover, a bruit heard over the bifurcation may reflect a narrowed *external* carotid artery and thus occur in angiographically normal or completely occluded *internal* carotids. Hence, surgical decisions should *not* be based on physical examination alone; imaging is mandatory.

11. What is central venous pressure?

Central venous pressure (CVP) is the pressure within the right atrium–superior vena cava system (i.e., the right ventricular filling pressure). As pulmonary capillary wedge pressure reflects left ventricular end-diastolic pressure (in the absence of mitral stenosis), so does CVP reflect right ventricular end-diastolic pressure (in the absence of tricuspid stenosis).

12. Which veins should be evaluated for assessing venous pulse and central venous pressure?

Central veins, especially those in direct communication with the right atrium, should be evaluated as much as possible. The ideal vein is therefore the internal jugular. Ideally, the right internal jugular vein should be inspected, because it is in a more direct line with the right atrium and thus better suited to function as both a manometer for venous pressure and a conduit for atrial pulsations. Moreover, CVP may be spuriously higher on the left than on the right because of the left innominate vein's compression between the aortic arch and the sternum.

13. Can the external jugulars be used for evaluating central venous pressure?

Theoretically they cannot, but practically they can. Theoretical models advise against this because:

- While going through the various fascial planes of the neck, they often become compressed.
- In patients with increased sympathetic vascular tone, they may become so constricted as to be barely visible.
- They are farther from the right atrium and thus in a less straight line with it. Yet, both internal *and* external jugular veins can actually be used for estimating CVP because they yield comparable estimates.

Hence, if the only visible vein is the external jugular, do what Yogi Berra recommends you should do when coming to a fork in the road: take it.

14. What is a cannon a wave?

A *cannon a wave* is the hallmark of atrioventricular dissociation (i.e., the atrium contracts against a closed tricuspid valve). It is different from the other prominent outward wave (i.e., the presystolic giant a wave) insofar as it begins just after S_1, because it represents atrial contraction against a closed tricuspid valve.

15. How do you estimate the central venous pressure?

- Position the patient so that you can get a good view of the internal jugular vein and its oscillations. Although it is wise to start at 45 degrees, it does not really matter which angle you will eventually use to raise the patient's head, as long as it can adequately reveal the vein. In the absence of a visible internal jugular, the external jugular may suffice.
- Identify the highest point of jugular pulsation that is transmitted to the skin (i.e., the meniscus). This usually occurs during exhalation and coincides with the peak of a or v waves. It serves as a bedside pulsation manometer.
- Find the sternal angle of Louis (the junction of the manubrium with the body of the sternum). This provides the standard zero for jugular venous pressure (JVP). (The standard zero for CVP is instead the center of the right atrium.)
- Measure in centimeters the vertical height from the sternal angle to the top of the jugular pulsation. To do so, place two rulers at a 90-degree angle: one horizontal (and parallel to the meniscus) and the other vertical to it and touching the sternal angle (see Fig. 1.1). The extrapolated height between the sternal angle and meniscus represents the JVP.
- Add 5 to convert JVP into CVP. This method relies on the fact that the zero point of the entire right-sided manometer (i.e., the point where CVP is, by convention, zero) is the center of the right atrium. This is vertically situated at 5 cm below the sternal angle, a relationship that is present in subjects of normal size and shape, regardless of their body position. Thus, using the sternal angle as the external reference point, the vertical distance (in centimeters) to the top of the column of blood in the jugular vein will provide the JVP. Adding 5 to the JVP will yield the CVP. Measurement of the JVP is illustrated in Fig. 1.4.

16. Can the jugular vein examination be used to diagnose cardiac pathology?

Yes. The jugular venous examination can suggest stenosed tricuspid valve, noncompliant hypertrophied right ventricle, AV dissociation (heart block, ventricular tachycardia), tricuspid regurgitation, constrictive pericarditis, and other conditions. Examples of the jugular venous examination in pathologic conditions are shown in Fig. 1.5.

17. What is the significance of leg swelling without increased central venous pressure?

It reflects either bilateral venous insufficiency or noncardiac edema (usually hepatic or renal). This is because any cardiac (or pulmonary) disease resulting in right ventricular failure would manifest itself through an increase in CVP. Leg edema *plus ascites* in the absence of increased CVP argues in favor of a hepatic or renal cause (patients with cirrhosis *do not* have high CVP). Conversely, a high CVP in patients with ascites and edema suggests the presence of an underlying cardiac etiology.

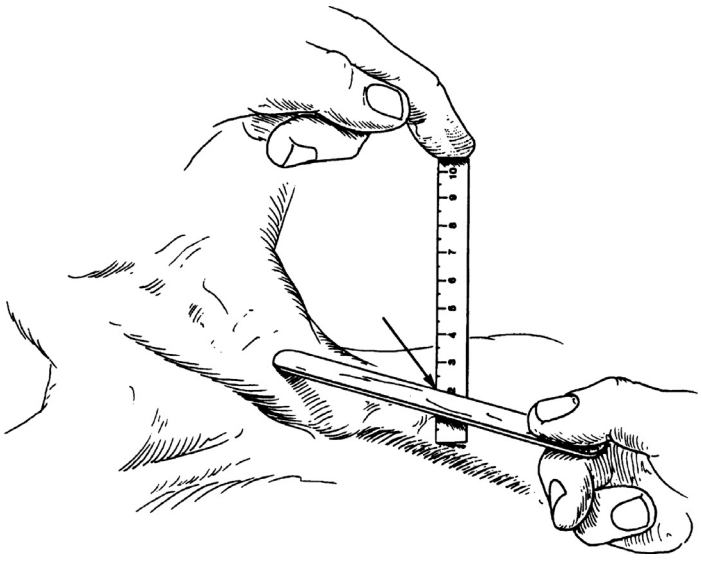

Fig. 1.4 Measurement of jugular venous pressure.

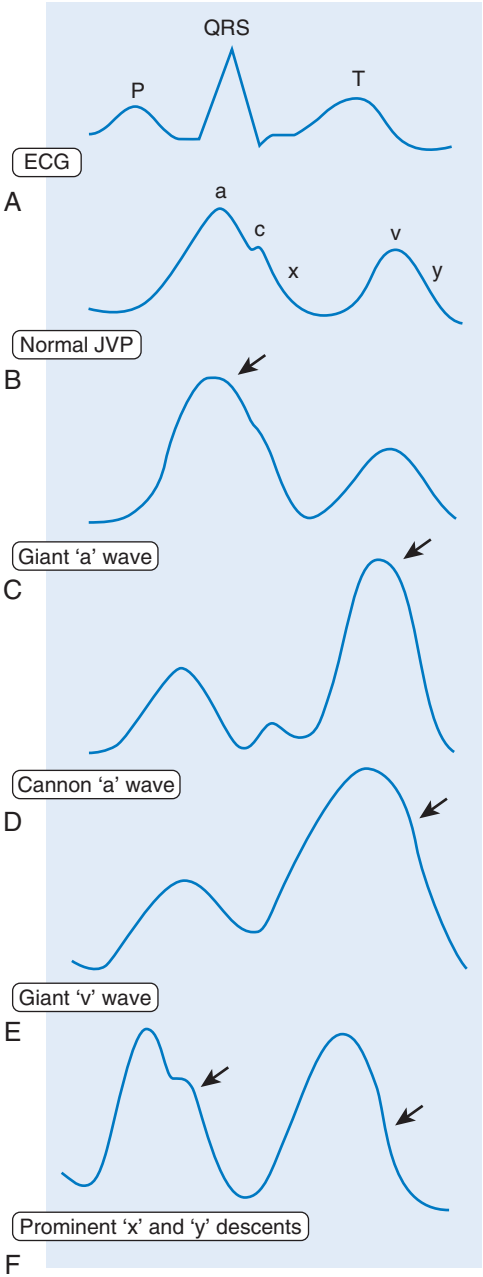

Fig. 1.5 Waveform of the jugular venous pulse. **A,** The ECG is portrayed at the top of the illustration. Note how electrical events precede mechanical events in the cardiac cycle. Thus the P wave (atrial depolarization) and the QRS complex (ventricular depolarization) precede a and v waves, respectively, of the JVP. **B,** Normal JVP. The a wave produced by atrial systole is the most prominent deflection. It is followed by the *x* descent, interrupted by the small c wave marking tricuspid valve closure. Atrial pressure then rises again (v wave) as the atrium fills passively during ventricular systole. The decline in atrial pressure as the tricuspid valve opens precedes the *y* descent. **C,** Giant a wave. Forceful atrial contraction against a stenosed tricuspid valve or a noncompliant hypertrophied right ventricle produces an unusually prominent a wave. **D,** Cannon a wave. This is caused by an atrial systole against a closed tricuspid valve. It occurs when atrial and ventricular rhythms are dissociated (complete heart block, ventricular tachycardia) and marks coincident atrial and ventricular systole. **E,** Giant v wave. This is an important sign of tricuspid regurgitation. The regurgitant jet produces pulsatile systolic waves in the JVP. **F,** Prominent *x* and *y* descents. These occur in the constructive pericarditis and give the JVP an unusually dynamic appearance. In tamponade, only *x* descent is unusually exaggerated. (From Glynn, M., & Drake, S. M. (2012). *Hutchison's clinical methods: an integrated approach to clinical practice* (23rd ed.). St. Louis, MO: Elsevier.)

18. What is the Kussmaul sign?

The *Kussmaul sign* (Fig. 1.6) is the paradoxical increase in JVP that occurs during inspiration. JVP normally *decreases* during inspiration because the inspiratory fall in intrathoracic pressure creates a "sucking effect" on venous return. Thus, the Kussmaul sign is a true physiologic paradox. This can be explained by the inability of the right side of the heart to handle an increased venous return.

Disease processes associated with a positive Kussmaul sign are those that interfere with venous return and right ventricular filling. The original description was in a patient with constrictive pericarditis. (The Kussmaul sign is

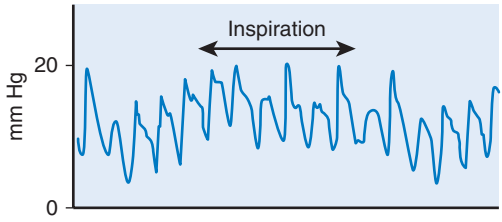

Fig. 1.6 Kussmaul sign. (From Glynn, M., & Drake, S. M. (2012). *Hutchison's clinical methods: an integrated approach to clinical practice* (23rd ed.). St. Louis, MO: Elsevier.)

still seen in one-third of patients with severe and advanced cases, in whom it is often associated with a positive abdominojugular reflux.) Nowadays, however, the most common cause is severe heart failure, independent of etiology. Other causes include cor pulmonale (acute or chronic), constrictive pericarditis, restrictive cardiomyopathy (such as sarcoidosis, hemochromatosis, and amyloidosis), tricuspid stenosis, and right ventricular infarction.

19. What is the "venous hum"?

Venous hum is a *functional* murmur produced by turbulent flow in the internal jugular vein. It is *continuous* (albeit louder in diastole) and at times strong enough to be associated with a palpable thrill. It is best heard on the right side of the neck, just above the clavicle, but sometimes it can become audible over the sternal and/or parasternal areas, both right and left. This may lead to misdiagnoses of carotid disease, patent ductus arteriosus, AR, or AS. The mechanism of the venous hum is a mild compression of the internal jugular vein by the transverse process of the atlas in subjects with strong cardiac output and increased venous flow; hence, it is common in young adults or patients with a high-output state. A venous hum can be heard in 31% to 66% of normal children and 25% of young adults. It is encountered also in 2.3% to 27% of adult outpatients. It is especially common in situations of arteriovenous fistula, being present in 56% to 88% of patients undergoing dialysis and 34% of those *between* sessions.

20. Which characteristics of the apical impulse should be analyzed?

- *Location*: Apical impulse normally occurs over the fifth left interspace midclavicular line, which usually (but not always) corresponds to the area just below the nipple. *Volume loads* to the left ventricle (such as aortic or MR) tend to displace the apical impulse downward and laterally. Conversely, *pressure loads* (such as AS or hypertension) tend to displace the impulse more upward and medially—at least initially. Still, a failing and decompensated ventricle, independent of its etiology, will typically present with a downward and lateral shift in point of maximal impulse (PMI). Although not too sensitive, this finding is very specific for cardiomegaly, low ejection fraction, and high pulmonary capillary wedge pressure. Correlation of the PMI with anatomic landmarks (such as the left anterior axillary line) can be used to better characterize the displaced impulse.
- *Size*: As measured in left lateral decubitus, the normal apical impulse is the size of a dime. Anything larger (a nickel or a quarter) should be considered pathologic. A diameter greater than 4 cm is quite specific for cardiomegaly.
- *Duration and timing*: This is probably one of the most important characteristics. A normal apical duration is brief and never passes midsystole. Thus, a *sustained impulse* (i.e., one that continues into S_2 and beyond—often referred to as a "heave") should be considered pathologic until proven otherwise and is usually indicative of pressure load, volume load, or cardiomyopathy.
- *Amplitude*: This is not the length of the impulse but its *force.* A *hyperdynamic* impulse (often referred to as a "thrust") that is forceful enough to lift the examiner's finger can be encountered in situations of volume overload and increased output (such as AR and VSD) but may also be felt in normal subjects with very thin chests. Similarly, a *hypodynamic* impulse can be due to not only simple obesity but also congestive cardiomyopathy. In addition to being hypodynamic, the precordial impulse of these patients is large, somewhat sustained, and displaced downward and/or laterally.
- *Contour*: A normal apical impulse is single. Double or triple impulses are clearly pathologic.
  Hence, a normal apical impulse consists of a single, dime-sized, brief (barely beyond S_1), early systolic, and nonsustained impulse, localized over the fifth interspace midclavicular line.

21. What is a thrill?

A *thrill* is a palpable vibration associated with an audible murmur. It automatically qualifies the murmur as being more than 4/6 in intensity and thus pathologic.

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