I. GENERAL EXAMINATION

CARDIOVASCULAR PHYSICAL EXAMINATION

Salvatore Mangione, MD

Editor’s Note to Readers: For an excellent and more detailed discussion of the cardiovascular physical examination, read Physical Diagnosis Secrets, ed 2, 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) argues for 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 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.

If associated with 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’s disease, arteriovenous fistulas, patent ductus arteriosus, aortic regurgitation, and anxiety—all typically associated with rapid ventricular contraction and low peripheral vascular resistance.

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 usually 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 S3 gallop.
6. What is Duroziez’s double murmur?  
Duroziez’s 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). Duroziez’s 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 insensitive sign of aortic valvular disease.

8. What is Corrigan’s pulse?  
Corrigan’s is one of the various names for the bounding and quickly collapsing pulse of aortic regurgitation, 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?  
By placing your bell on the neck in a quiet room and with a relaxed patient. 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?  
It’s high. In fact, bruits presenting with transient ischemic attacks (TIAs) 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 presence of a bruit significantly increases the likelihood of high-grade carotid stenosis, its absence doesn’t 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 (CVP)?  
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 central venous pressure reflects right ventricular end-diastolic pressure (in the absence of tricuspid stenosis).

12. Which veins should be evaluated for assessing venous pulse and CVP?  
Central veins, as much in direct communication with the right atrium as possible. The ideal one 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 as compared with 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 not, practically yes. Not 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 S1, because it represents atrial contraction against a closed tricuspid valve.

15. How do you estimate the CVP?
- By positioning 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 doesn’t 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.
- By identifying 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.
- By finding 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. (The standard zero for central venous pressure is instead the center of the right atrium.)
- By measuring 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 (Fig. 1-1). The extrapolated height between the sternal angle and meniscus represents the jugular venous pressure (JVP).
- By adding 5 to convert jugular venous pressure into central venous pressure. This method relies on the fact that the zero point of the entire right-sided manometer (i.e., the point where central venous pressure 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.

16. 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 central venous pressure. 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 argues in favor of an underlying cardiac etiology.

17. What is Kussmaul’s sign?
Kussmaul’s sign is the paradoxical increase in JVP that occurs during inspiration. Jugular venous pressure normally decreases during inspiration because the inspiratory fall in
intrathoracic pressure creates a “sucking effect” on venous return. Thus, Kussmaul’s 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’s are those that interfere with venous return and right ventricular filling. The original description was in a patient with constrictive pericarditis. (Kussmaul’s is 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.

18. 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/parasternal areas, both right and left. This may lead to misdiagnoses of carotid disease, patent ductus arteriosus, or AR/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 also is encountered 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.
19. **Which characteristics of the apical impulse should be analyzed?**

- **Location:** Normally 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 mitral regurgitation) tend to displace the apical impulse downward and laterally. Conversely, *pressure loads* (such as aortic stenosis 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 (nickel, quarter, or an old Eisenhower silver dollar) 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 S2 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 aortic regurgitation and ventricular septal defect) but may also be felt in normal subjects with very thin chests. Similarly, a *hypodynamic* impulse can be due to simple obesity but also to congestive cardiomyopathy. In addition to being hypodynamic, the precordial impulse of these patients is large, somewhat sustained, and displaced downward/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 S1), early systolic, and nonsustained impulse, localized over the fifth interspace midclavicular line.

20. **What is a thrill?**

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

**BIBLIOGRAPHY, SUGGESTED READINGS, AND WEBSITES**

HEART MURMURS
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Editor’s Note to Readers: For an excellent and more detailed discussion of heart murmurs, read Physical Diagnosis Secrets, ed 2, by Salvatore Mangione.

1. What are the auscultatory areas of murmurs?
Auscultation typically starts in the aortic area, continuing in clockwise fashion: first over the pulmonic, then the mitral (or apical), and finally the tricuspid areas (Fig. 2-1). Because murmurs may radiate widely, they often become audible in areas outside those historically assigned to them. Hence, “inching” the stethoscope (i.e., slowly dragging it from site to site) can be the best way to avoid missing important findings.

2. What is the Levine system for grading the intensity of murmurs?
The intensity or loudness of a murmur is traditionally graded by the Levine system (no relation to this book’s editor) from 1/6 to 6/6. Everything else being equal, increased intensity usually reflects increased flow turbulence. Thus, a louder murmur is more likely to be pathologic and severe.
- 1/6: a murmur so soft as to be heard only intermittently and always with concentration and effort. Never immediately
- 2/6: a murmur that is soft but nonetheless audible immediately and on every beat
- 3/6: a murmur that is easily audible and relatively loud
- 4/6: a murmur that is relatively loud and associated with a palpable thrill (always pathologic)
- 5/6: a murmur loud enough that it can be heard even by placing the edge of the stethoscope’s diaphragm over the patient’s chest
- 6/6: a murmur so loud that it can be heard even when the stethoscope is not in contact with the chest, but held slightly above its surface

3. What are the causes of a systolic murmur?
■ Ejection (i.e., increased “forward” flow over the aortic or pulmonic valve): This can be:
  ○ Physiologic: normal valve, but flow high enough to cause turbulence (anemia, exercise, fever, and other hyperkinetic heart syndromes)
  ○ Pathologic: abnormal valve, with or without outflow obstruction (i.e., aortic stenosis versus aortic sclerosis)
■ Regurgitation: “backward” flow from a high- into a low-pressure bed. Although this is usually due to incompetent atrioventricular (AV) valves (mitral/tricuspid), it also can be due to ventricular septal defect.

4. What are functional murmurs?
They are benign findings caused by turbulent ejection into the great vessels. Functional murmurs have no clinical relevance, other than getting into the differential diagnosis of a systolic murmur.

5. What is the most common systolic ejection murmur of the elderly?
The murmur of aortic sclerosis. This early peaking systolic murmur is extremely age related, affecting 21% to 26% of persons older than 65 and 55% to 75% of octogenarians. (Conversely, the prevalence of aortic stenosis in these age groups is 2% and 2.6%, respectively.) The murmur
of aortic sclerosis may be due to either a degenerative change of the aortic valve or abnormalities of the aortic root. Senile degeneration of the aortic valve includes thickening, fibrosis, and occasionally calcification. This can stiffen the valve and yet not cause a transvalvular pressure gradient. In fact, commissural fusion is typically absent in aortic sclerosis. Abnormalities of the aortic root may be diffuse (such as a tortuous and dilated aorta) or localized (like a calcific spur or an atherosclerotic plaque that protrudes into the lumen, creating a turbulent bloodstream).

6. How can physical examination help differentiate functional from pathologic murmurs?
There are two golden and three silver rules:
- The first golden rule is to always judge (systolic) murmurs like people: by the company they keep. Hence, murmurs that keep bad company (like symptoms; extra sounds; thrill; and abnormal arterial or venous pulse, electrocardiogram [ECG], or chest radiograph) should be considered pathologic until proven otherwise. These murmurs should receive lots of evaluation, including technology based.
- The second golden rule is that a diminished or absent S₂ usually indicates a poorly moving and abnormal semilunar (aortic or pulmonic) valve. This is the hallmark of pathology. As a flip side, functional systolic murmurs are always accompanied by a well-preserved S₂, with normal split.

The three silver rules are:
- All holosystolic (or late systolic) murmurs are pathologic.
- All diastolic murmurs are pathologic.
- All continuous murmurs are pathologic.
Thus, functional murmurs should be systolic, short, soft (typically less than 3/6), early peaking (never passing mid-systole), predominantly circumscribed to the base, and associated with a well-preserved and normally split-second sound. They should have an otherwise normal cardiovascular examination and often disappear with sitting, standing, or straining (as, for example, following a Valsalva maneuver).

7. **How much reduction in valvular area is necessary for the aortic stenosis (AS) murmur to become audible?**
   At least 50% (the minimum for creating a pressure gradient at rest). Mild disease may produce loud murmurs, too, but usually significant hemodynamic compromise (and symptoms) does not occur until a 60% to 70% reduction in valvular area exists. This means that early to mild AS may be subtle at rest. Exercise, however, may intensify the murmur by increasing the output and gradient.

8. **What factors may suggest severe aortic stenosis?**
   - Murmur intensity and timing (the louder and later peaking the murmur, the worse the disease)
   - A single S₂
   - Delayed upstroke/reduced amplitude of the carotid pulse (pulsus parvus and tardus)

9. **What is a thrill?**
   It is a palpable vibratory sensation, often compared to the purring of a cat, and typical of murmurs caused by very high pressure gradients. These, in turn, lead to great turbulence and loudness. Hence, thrills are only present in pathologic murmurs whose intensity is greater than 4/6.

10. **What is isometric hand grip, and what does it do to AS and mitral regurgitation (MR) murmurs?**
    Isometric hand grip is carried out by asking the patient to lock the cupped fingers of both hands into a grip and then trying to pull them apart. The resulting increase in peripheral vascular resistance intensifies MR (and ventricular septal defect) while softening instead AS (and aortic sclerosis). Hence, a positive hand grip argues strongly in favor of MR.

11. **What is the Gallavardin phenomenon?**
    One noticed in some patients with AS, who may exhibit a dissociation of their systolic murmur into two components:
    - A typical AS-like murmur (medium to low pitched, harsh, right parasternal, typically radiated to the neck, and caused by high-velocity jets into the ascending aorta)
    - A murmur that instead mimics MR (high pitched, musical, and best heard at the apex)
    This phenomenon reflects the different transmission of AS: its medium frequencies to the base and its higher frequencies to the apex. The latter may become so prominent as to be misinterpreted as a separate apical “cooing” of MR.

12. **Where is the murmur of hypertrophic cardiomyopathy (HCM) best heard?**
    It depends. When septal hypertrophy obstructs not only left but also right ventricular outflow, the murmur may be louder at the left lower sternal border. More commonly, however, the HCM murmur is louder at the apex. This may often cause a differential diagnosis dilemma with the murmur of MR.

13. **What are the characteristics of a ventricular septal defect (VSD) murmur?**
    VSD murmurs may be holosystolic, crescendo-decrescendo, crescendo, or decrescendo. A crescendo-decrescendo murmur usually indicates a defect in the muscular part of the septum. Ventricular contraction closes the hole toward the end of systole, thus causing the decrescendo phase of the murmur. Conversely, a defect in the membranous septum will enjoy no systolic...
reduction in flow and thus produce a murmur that remains constant and holosystolic. VSD murmurs are best heard along the left lower sternal border, often radiating left to right across the chest. VSD murmurs always start immediately after S1.

14. **What is a systolic regurgitant murmur?**
One characterized by a pressure gradient that causes a retrograde blood flow across an abnormal opening. This can be (1) a ventricular septal defect, (2) an incompetent mitral valve, (3) an incompetent tricuspid valve, or (4) fistulous communication between a high-pressure and a low-pressure vascular bed (such as a patent ductus arteriosus).

15. **What are the auscultatory characteristics of systolic regurgitant murmurs?**
They tend to start immediately after S1, often extending into S2. They also may have a musical quality, variously described as "honk" or "whoop." This is usually caused by vibrating vegetations (endocarditis) or chordae tendineae (mitral valve prolapse, dilated cardiomyopathy) and may help separate the more musical murmurs of AV valve regurgitation from the harsher sounds of semilunar stenosis. Note that in contrast to systolic ejection murmurs like AS or VSD, systolic regurgitant murmurs do not increase in intensity after a long diastole.

16. **What are the characteristics of the MR murmur?**
It is loudest at the apex, radiated to the left axilla or interscapular area, high pitched, plateau, and extending all the way into S2 (holosystolic). S2 is normal in intensity but often widely split. If the gradient is high (and the flow is low), the MR murmur is high pitched. Conversely, if the gradient is low (and the flow is high) the murmur is low pitched. In general, the louder (and longer) the MR murmur, the worse the regurgitation.

17. **What are the characteristics of the acute MR murmur?**
The acute MR murmur tends to be very short, and even absent, because the left atrium and ventricle often behave like a common chamber, with no pressure gradient between them. Hence, in contrast to that of chronic MR (which is either holosystolic or late systolic), the acute MR murmur is often early systolic (exclusively so in 40% of cases) and is associated with an S4 in 80% of the patients.

18. **What are the characteristics of the mitral valve prolapse murmur?**
It is a mitral regurgitant murmur—hence, loudest at the apex, mid to late systolic in onset (immediately following the click), and usually extending all the way into the second sound (A2). In fact, it often has a crescendo shape that peaks at S2. It is usually not too loud (never greater than 3/6), with some musical features that have been variously described as whoops or honks (as in the honking of a goose). Indeed, musical murmurs of this kind are almost always due to mitral valve prolapse (MVP).

19. **How are diastolic murmurs classified?**
By their timing. Hence, the most important division is between murmurs that start just after S2 (i.e., early diastolic—reflecting aortic or pulmonic regurgitation) versus those that start a little later (i.e., mid to late diastolic, often with a presystolic accentuation—reflecting mitral or tricuspid valve stenosis) (Fig. 2-2).

20. **What is then the best strategy to detect the mitral stenosis (MS) murmur?**
The best strategy consists of listening over the apex, with the patient in the left lateral decubitus position, at the end of exhalation, and after a short exercise. Finally, applying the bell with very light pressure also may help. (Strong pressure will instead completely eliminate the low frequencies of MS.)
21. What are the typical auscultatory findings of aortic regurgitation (AR)?
Depending on severity, there may be up to three murmurs (one in systole and two in diastole) plus an ejection click. Of course, the typical auscultatory finding is the diastolic tapering murmur, which, together with the brisk pulse and the enlarged/displaced point of maximal impulse (PMI), constitutes the bedside diagnostic triad of AR. The diastolic tapering murmur is usually best heard over Erb’s point (third or fourth interspace, left parasternal line) but at times also over the aortic area, especially when a tortuous and dilated root pushes the ascending aorta anteriorly and to the right. The decrescendo diastolic murmur of AR is best heard by
having the patient sit up and lean forward while holding breath in exhalation. Using the diaphragm and pressing hard on the stethoscope also may help because this murmur is rich in high frequencies. Finally, increasing peripheral vascular resistances (by having the patient squat) will also intensify the murmur. A typical, characteristic early diastolic murmur argues very strongly in favor of the diagnosis of AR.

An accompanying systolic murmur may be due to concomitant AS but most commonly indicates severe regurgitation, followed by an increased systolic flow across the valve. Hence, this accompanying systolic murmur is often referred to as comitans (Latin for “companion”). It provides an important clue to the severity of regurgitation. A second diastolic murmur can be due to the rumbling diastolic murmur of Austin Flint (i.e., functional mitral stenosis). The Austin Flint murmur is a mitral stenosis–like diastolic rumble, best heard at the apex, and is due to the regurgitant aortic stream preventing full opening of the anterior mitral leaflet.

22. **What is a mammary soufflé?**

   Not a fancy French dish but a systolic-diastolic murmur heard over one or both breasts in late pregnancy and typically disappearing at end of lactation. It is caused by increased flow along the mammary arteries, which explains why its systolic component starts just a little after S1. It can be obliterated by pressing (with finger or stethoscope) over the area of maximal intensity.

**BIBLIOGRAPHY, SUGGESTED READINGS, AND WEBSITES**

1. What are the most commonly used criteria to diagnose left ventricular hypertrophy (LVH)?
   - R wave in V5-V6 + S wave in V1-V2 > 35 mm
   - R wave in lead I + S wave in lead III > 25 mm

2. What are the most commonly used criteria to diagnose right ventricular hypertrophy (RVH)?
   - R wave in V1 ≥ 7 mm
   - R/S wave ratio in V1 > 1

3. What criteria are used to diagnose left atrial enlargement (LAE)?
   - P wave total width of > 0.12 sec (3 small boxes) in the inferior leads, usually with a double-peaked P wave
   - Terminal portion of the P wave in lead V1 ≥ 0.04 sec (1 small box) wide and ≥ 1 mm (1 small box) deep

4. What electrocardiogram (ECG) finding suggests right atrial enlargement?
   - P-wave height in the inferior leads (II, III, and aVF) ≥ 2.5 to 3 mm (2.5–3 small boxes) (Fig. 3-1)

Figure 3-1. Right atrial enlargement. The tall P waves in the inferior leads (II, III, and aVF) are more than 2.5 to 3 mm high.
5. **What is the normal rate of a junctional rhythm?**
The normal rate is 40 to 60 beats/min. Rates of 61 to 99 beats/min are referred to as *accelerated junctional rhythm*, and rates of 100 beats/min or higher are referred to as *junctional tachycardia*.

6. **How can one distinguish a junctional escape rhythm from a ventricular escape rhythm in a patient with complete heart block?**
Junctional escape rhythms usually occur at a rate of 40 to 60 beats/min and will usually be narrow complex (unless the patient has a baseline bundle branch block), whereas ventricular escape rhythms will usually occur at a rate of 30 to 40 beats/min and will be wide complex.

7. **Describe the three types of heart block.**
- **First-degree heart block:** The PR interval is a fixed duration of more than 0.20 seconds.
- **Second-degree heart block:** In Mobitz type I (Wenkebach), the PR interval increases until a P wave is nonconducted (Fig. 3-2). The cycle then resets and starts again. Mobitz type I second-degree heart block is sometimes due to increased vagal tone and is usually a relatively benign finding. In Mobitz type II, the PR interval is fixed and occasional P waves are nonconducted. Mobitz type II second-degree heart block usually indicates structural disease in the atroventricular (AV) node or His-Purkinje system and is an indication for pacemaker implantation.
- **Third-degree heart block:** All P waves are nonconducted, and there is either a junctional or ventricular escape rhythm. To call a rhythm third-degree or complete heart block, the atrial rate (as evidenced by the P waves) should be faster than the ventricular escape rate (the QRS complexes). Third-degree heart block is almost always an indication for a permanent pacemaker.

8. **What are the causes of ST segment elevation?**
- Acute myocardial infarction (MI) due to thrombotic occlusion of a coronary artery
- Prinzmetal’s angina (variant angina), in which there is vasospasm of a coronary artery
- Cocaine-induced MI, in which there is vasospasm of a coronary artery, with or without additional thrombotic occlusion
- Pericarditis, in which there is usually diffuse ST segment elevation
- Left ventricular aneurysm
- Left bundle branch block (LBBB)
- Left ventricular hypertrophy with repolarization abnormalities
- J point elevation, a condition classically seen in young African-American patients but that can be seen in any patient, which is felt due to “early repolarization”
- Severe hyperkalemia
9. **What are the electrocardiographic findings of hyperkalemia?**

Initially, a “peaking” of the T waves is seen (Fig. 3-3). As the hyperkalemia becomes more profound, “loss” of the P waves, QRS widening, and ST segment elevation may occur. The preterminal finding is a sinusoidal pattern on the ECG (Fig. 3-4).

*Figure 3-3. Hyperkalemia. Peaked T waves are seen in many of the precordial leads. (Adapted with permission from Levine GN, Podrid PJ: *The ECG workbook: a review and discussion of ECG findings and abnormalities*, New York, Futura Publishing Company, 1995. p. 405)*

*Figure 3-4. Severe hyperkalemia. The rhythm strip demonstrates the preterminal rhythm sinusoidal wave seen in cases of severe hyperkalemia. (Adapted with permission from Levine GN, Podrid PJ: *The ECG workbook: a review and discussion of ECG findings and abnormalities*, New York, Futura Publishing Company, 1995. p. 503)*
10. **What are the ECG findings in pericarditis?**

The first findings are believed by some to be PR segment depression caused by repolarization abnormalities of the atria. This may be fairly transient and is often not present by the time the patient is seen for evaluation. Either concurrent with PR segment depression or shortly following PR segment depression, diffuse ST segment elevation occurs (see ECG example in Chapter 53 on Pericarditis). At a later time, diffuse T-wave inversions may develop.

11. **What is electrical alternans?**

In the presence of large pericardial effusions, the heart may “swing” within the large pericardial effusion, resulting in an alteration of the amplitude of the QRS complex (Fig. 3-5).

![Figure 3-5. Electrical alternans in a patient with a large pericardial effusion. Note the alternating amplitude of the QRS complexes. (From Manning WJ: Pericardial disease. In Goldman L: *Cecil medicine*, ed 23, Philadelphia, Saunders, 2008.)](image)

12. **What is the main ECG finding in hypercalcemia and hypocalcemia?**

With hypercalcemia the QT interval shortens. With hypocalcemia, prolongation of the QT interval occurs as a result of delayed repolarization (Fig. 3-6).

![Figure 3-6. Electrocardiographic findings of hypercalcemia and hypocalcemia. With hypercalcemia, the QT interval shortens. With hypocalcemia there is prolongation of the QT interval due to delayed repolarization. (From Park MK, Guntheroth WG: *How to read pediatric ECGs*, ed 4, Philadelphia, Mosby, 2006.)](image)

13. **What ECG findings may be present in pulmonary embolus?**

- Sinus tachycardia (the most common ECG finding)
- Right atrial enlargement (P pulmonale)—tall P waves in the inferior leads
- Right axis deviation
- T wave inversions in leads V1-V2
- Incomplete right bundle branch block (IRBBB)
- S1Q3T3 pattern—an S wave in lead I, a Q wave in lead III, and an inverted T wave in lead III. Although this is only occasionally seen with pulmonary embolus, it is said to be quite suggestive that a pulmonary embolus has occurred.
14. **What is torsades de pointes?**

Torsades de pointes is a ventricular arrhythmia that occurs in the setting of QT prolongation, usually when drugs that prolong the QT interval have been administered. It may also occur in the setting of prolonged QT syndrome and other conditions. The term was reportedly coined by Dessertenne to describe the arrhythmia, in which the QRS axis appears to twist around the isoelectric line (Fig. 3-7). It is usually a hemodynamically unstable rhythm that can further degenerate and lead to hemodynamic collapse.

![Figure 3-7. Torsades de pointes, in which the QRS axis seems to rotate about the isoelectric point. (From Olgin JE, Zipes DP: Specific arrhythmias: diagnosis and treatment. In Libby P, Bonow R, Mann D, et al: *Braunwald’s heart disease: a textbook of cardiovascular medicine*, ed 8, Philadelphia, Saunders, 2008.)](image)

15. **What are cerebral T waves?**

Cerebral T waves are strikingly deep and inverted T waves, most prominently seen in the precordial leads, that occur with central nervous system diseases, most notably subarachnoid and intracerebral hemorrhages. They are believed to be due to prolonged and abnormal repolarization of the left ventricle, presumably as a result of autonomic imbalance. They should not be mistaken for evidence of active cardiac ischemia (Fig. 3-8).

![Figure 3-8. Cerebral T waves. The markedly deep and inverted T waves are seen with central nervous system disease, particularly subarachnoid and intracerebral hemorrhages. (Reproduced with permission from Levine GN, Podrid PJ: *The ECG workbook: a review and discussion of ECG findings and abnormalities*, New York, Futura Publishing Company, 1995. p. 437)](image)
1. ECG Library: http://www.ecglibrary.com/ecghome.html
2. ECG Tutorial: http://www.uptodate.com
1. Describe a systematic approach to interpreting a chest radiograph (chest x-ray [CXR]) (Fig. 4-1).

Common recommendations are to:

1. Begin with general characteristics such as the age, gender, size, and position of the patient.
2. Next examine the periphery of the film, including the bones, soft tissue, and pleura. Look for rib fractures, rib notching, bony metastases, shoulder dislocation, soft tissue masses, and pleural thickening.
3. Then evaluate the lung, looking for infiltrates, pulmonary nodules, and pleural effusions.
4. Finally, concentrate on the heart size and contour, mediastinal structures, hilum, and great vessels. Also note the presence of pacemakers and sternal wires.

Figure 4-1. Diagrammatic representations of the anatomy of the chest radiograph. Aor, Aorta; IVC, inferior vena cava; LAA, left atrial appendage; LPA, left pulmonary artery; LV, left ventricle; PT, pulmonary trunk; RA, right atrium; RPA, right pulmonary artery; RV, right ventricle; SVC, superior vena cava; Tr, trachea; IVC, inferior vena cava; LPA, left pulmonary artery; LV, left ventricle; RPA, right pulmonary artery; RV, right ventricle; Tr, trachea. (From Inaba AS: Cardiac disorders. In Marx J, Hockberger R, Walls R: Rosen’s emergency medicine: concepts and clinical practice, ed 6, Philadelphia, 2006, Mosby.)
2. **Identify the major cardiovascular structures that form the silhouette of the mediastinum (Fig. 4-2).**
   - **Right side:** Ascending aorta, right pulmonary artery, right atrium, right ventricle
   - **Left side:** Aortic knob, left pulmonary artery, left atrial appendage, left ventricle

3. **How is heart size measured on a chest radiograph?**
   Identification of cardiomegaly on a CXR is subjective, but if the heart size is equal to or greater than twice the size of the hemithorax, then it is enlarged. Remember that a film taken during expiration, in a supine position, or by a portable AP technique will make the heart appear larger.

4. **What factors can affect heart size on the chest radiograph?**
   - **Size of the patient:** Obesity decreases lung volumes and enlarges the appearance of the heart.
   - **Degree of inspiration:** Poor inspiration can make the heart appear larger.
   - **Emphysema:** Hyperinflation changes the configuration of the heart, making it appear smaller.
   - **Contractility:** Systole or diastole can make up to a 1.5-cm difference in heart size. In addition, low heart rate and increased cardiac output lead to increased ventricular filling.
   - **Chest configuration:** Pectus excavatum can compress the heart and make it appear larger.
   - **Patient positioning:** The heart appears larger if the film is taken in a supine position.
   - **Type of examination:** On an anteroposterior (AP) projection, the heart is farther away from the film and closer to the camera. This creates greater beam divergence and the appearance of an increased heart size.
5. What additional items should be reviewed when examining a chest radiograph from the intensive care unit (ICU)?

On portable cardiac care unit (CCU) and ICU radiographs, particular attention should be paid to:

- Placement of the endotracheal tube
- Central lines
- Pulmonary arterial catheter
- Pacing wires
- Defibrillator pads
- Intraaortic balloon pump
- Feeding tubes
- Chest tubes

A careful inspection should be made for pneumothorax (Fig. 4-3), subcutaneous emphysema, and other factors that may be related to instrumentation and mechanical ventilation.

6. How can one determine which cardiac chambers are enlarged?

- **Ventricular enlargement**: usually displaces the lower heart border to the left and posteriorly. Distinguishing right ventricular (RV) from left ventricular (LV) enlargement requires evaluation of the outflow tracts. In RV enlargement the pulmonary arteries are often prominent and the aorta is diminutive. In LV enlargement the aorta is prominent and the pulmonary arteries are normal.

- **Left atrial (LA) enlargement**: creates a convexity between the left pulmonary artery and the left ventricle on the frontal view. Also, a **double density** may be seen inferior to the carina. On the lateral view, LA enlargement displaces the descending left lower lobe bronchus posteriorly.

- **Right atrial enlargement**: causes the lower right heart border to bulge outward to the right.

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**Figure 4-3.** Tension pneumothorax. On a posteroanterior chest radiograph (A) the left hemithorax is very dark or lucent because the left lung has collapsed completely (white arrows). The tension pneumothorax can be identified because the mediastinal contents, including the heart, are shifted toward the right and the left hemidiaphragm is flattened and depressed. B, A computed tomography scan done on a different patient with a tension pneumothorax shows a completely collapsed right lung (arrows) and shift of the mediastinal contents to the left. (From Mettler: Essentials of radiology, ed 2, Philadelphia, 2005, Saunders.)
7. What are some of the common causes of chest pain that can be identified on a chest radiograph?

- Aortic dissection
- Pneumonia
- Pneumothorax
- Pulmonary embolism
- Subcutaneous emphysema
- Pericarditis (if a large pericardial effusion is suggested by the radiograph)
- Esophageal rupture
- Hiatal hernia

All patients with chest pain should undergo a CXR even if the cause of the chest pain is suspected myocardial ischemia.

8. What are the causes of a widened mediastinum?

There are multiple potential causes of a widened mediastinum (Fig. 4-4). Some of the most concerning causes of mediastinal widening include aortic dissection/rupture and mediastinal bleeding from chest trauma or misplaced central venous catheters. One of the most common causes of mediastinal widening is thoracic lipomatosis in an obese patient. Tumors should also be considered as a cause of a widened mediastinum—especially germ cell tumors, lymphoma, and thymomas. The mediastinum may also appear wider on a portable AP film compared with a standard posteroanterior/lateral chest radiograph.

![Figure 4-4. Widened mediastinum (arrows). (From Marx J, Hockberger R, Walls R: Rosen's emergency medicine: concepts and clinical practice, ed 6, Philadelphia, 2006, Mosby.)](image)

9. What are the common radiographic signs of congestive heart failure?

- Enlarged cardiac silhouette
- Left atrial enlargement
- Hilar fullness
- Vascular redistribution
- Linear interstitial opacities (Kerley’s lines)
- Bilateral alveolar infiltrates
- Pleural effusions (right greater than left)
10. What is vascular redistribution? When does it occur in congestive heart failure?
Vascular redistribution occurs when the upper-lobe pulmonary arteries and veins become larger than the vessels in the lower lobes. The sign is most accurate if the upper lobe vessels are increased in diameter greater than 3 mm in the first intercostal interspace. It usually occurs at a pulmonary capillary occlusion pressure of 12–19 mm Hg. As the pulmonary capillary occlusion pressure rises above 19 mm Hg, interstitial edema develops with bronchial cuffing, Kerley’s B lines, and thickening of the lung fissures. Vascular redistribution to the upper lobes is probably most consistently seen in patients with chronic pulmonary venous hypertension (mitral valve disease, left ventricular dysfunction) because of the body’s attempt to maintain more normal blood flow and oxygenation in this area. Some authors believe that vascular redistribution is a cardinal feature of congestive heart failure, but it may be a particularly unhelpful sign in the ICU patient with acute congestive failure. In these patients, all the pulmonary arteries look enlarged, making it difficult to assess upper and lower vessel size. In addition, the film is often taken supine, which can enlarge the upper lobe pulmonary vessels because of stasis of blood flow and not true redistribution.

11. How does LV dysfunction and RV dysfunction lead to pleural effusions?
- LV dysfunction causes increased hydrostatic pressures, which lead to interstitial edema and pleural effusions. Right pleural effusions are more common than left pleural effusions, but the majority are bilateral.
- RV dysfunction leads to system venous hypertension, which inhibits normal reabsorption of pleural fluid into the parietal pleural lymphatics.

12. How helpful is the chest radiograph at identifying and characterizing a pericardial effusion?
The CXR is not sensitive for the detection of a pericardial effusion, and it may not be helpful in determining the extent of an effusion. Smaller pericardial effusions are difficult to detect on a CXR but can still cause tamponade physiology if fluid accumulation is rapid. A large hourglass cardiac silhouette (Fig. 4-5), however, may suggest a large pericardial effusion. Distinguishing pericardial fluid from chamber enlargement is often difficult.

Figure 4-5. The water bottle configuration that can be seen with a large pericardial effusion. (From Kliegman RM, Behrman RE, Jenson HB, et al: Nelson textbook of pediatrics, ed 18, Philadelphia, 2007, Saunders.)
13. **What are the characteristic radiographic findings of significant pulmonary hypertension?**

Enlargement of the central pulmonary arteries with rapid tapering of the vessels is a characteristic finding in patients with pulmonary hypertension (Fig. 4-6). If the right descending pulmonary artery is greater than 17 mm in transverse diameter, it is considered enlarged. Other findings of pulmonary hypertension include cardiac enlargement (particularly the right ventricle) and calcification of the pulmonary arteries. Pulmonary arterial calcification follows atheroma formation in the artery and represents a rare but specific radiographic finding of severe pulmonary hypertension.

![Figure 4-6. Pulmonary arterial hypertension. Marked dilation of the main pulmonary artery (MPA) and right pulmonary artery (RPA) is noted. Rapid tapering of the arteries as they proceed peripherally is suggestive of pulmonary hypertension and is sometimes referred to as pruning. (From Mettler FA: Essentials of radiology, ed 2, Philadelphia, 2005, Saunders.)](image)

14. **What is Westermark’s sign?**

Westermark’s sign is seen in patients with pulmonary embolism and represents an area of oligemia beyond the occluded pulmonary vessel. If pulmonary infarction results, a wedge-shaped infiltrate may be visible (Fig. 4-7).

15. **What is rib notching?**

Rib notching is erosion of the inferior aspects of the ribs (Fig. 4-8). It can be seen in some patients with coarctation of the aorta and results from a compensatory enlargement of the intercostal arteries as a means of increasing distal circulation. It is most commonly seen between the fourth and eighth ribs. It is important to recognize this life-saving finding because aortic coarctation is treatable with percutaneous or open surgical intervention.
Figure 4-7. A peripheral wedge-shaped infiltrate (white dashed lines) seen after a pulmonary embolism has lead to infarction. (From Mettler FA: Essentials of radiology, ed 2, Philadelphia, Saunders, 2005.)

Figure 4-8. Rib notching in a patient with coarctation of the aorta. (From Park MK: Pediatric cardiology for practitioners, ed 5, Philadelphia, 2008, Mosby.)
16. **What does the finding in Figure 4-9 suggest?**

The important finding in this figure is pericardial calcification. This can occur in diseases that affect the pericardium, such as tuberculosis. In a patient with signs and symptoms of heart failure, this finding would be highly suggestive of the diagnosis of constrictive pericarditis.

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**Figure 4-9.** Pericardial calcification (*arrows*). In a patient with signs and symptoms of heart failure, this finding would strongly suggest the diagnosis of constrictive cardiomyopathy. (From Libby P, Bonow RO, Mann DL, et al: *Braunwald's heart disease*, ed 8, Philadelphia, 2008, Saunders.)

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**BIBLIOGRAPHY, SUGGESTED READINGS, AND WEBSITES**


