

KEY FACT

At usual speed (25 = mm/s), each large square = 200 msec, and each small square = 40 msec.

KEY FACT

Heart rate = 300/number of large boxes between two consecutive QRS complexes.

MNEMONIC***Axis deviation—*****RAD RALPH, the LAD from VILLA hates WOLVES****Right Axis Deviation**

Right ventricular hypertrophy

Anterolateral MI

Left Posterior Hemiblock

(also consider PE)

Left Axis Deviation

Ventricular tachycardia

Inferior myocardial infarction

Left ventricular hypertrophy

Left Anterior hemiblock

WOLVES – Wolf-Parkinson-White syndrome can cause BOTH

Electrocardiogram

Assesses the ECG for rate, rhythm, axis, intervals, ischemia, and chamber enlargement (see Figure 2.1-1).

Rate

Normal adult HR is 60–100 bpm. HR < 60 bpm is bradycardia. HR > 100 bpm is tachycardia. Common causes of sinus bradycardia are physical fitness, sick sinus syndrome, drugs, vasovagal attacks, acute MI, ↑ intracranial pressure. Common causes of sinus tachycardia are anxiety, anemia, pain, fever, sepsis, CHF, PE, hypovolemia, thyrotoxicosis, CO₂ retention, and sympathomimetics.

Rhythm

Sinus rhythm: Normal rhythm that originates from sinus node. It is characterized by a P wave (upright in II, III, and aVF; inverted in aVR) preceding every QRS complex and a QRS complex following every P wave. Sinus arrhythmia is common in young adults.

Axis

Can be determined by examining the QRS in leads I, II, and aVF (see Table 2.1-1 and Figure 2.1-2).

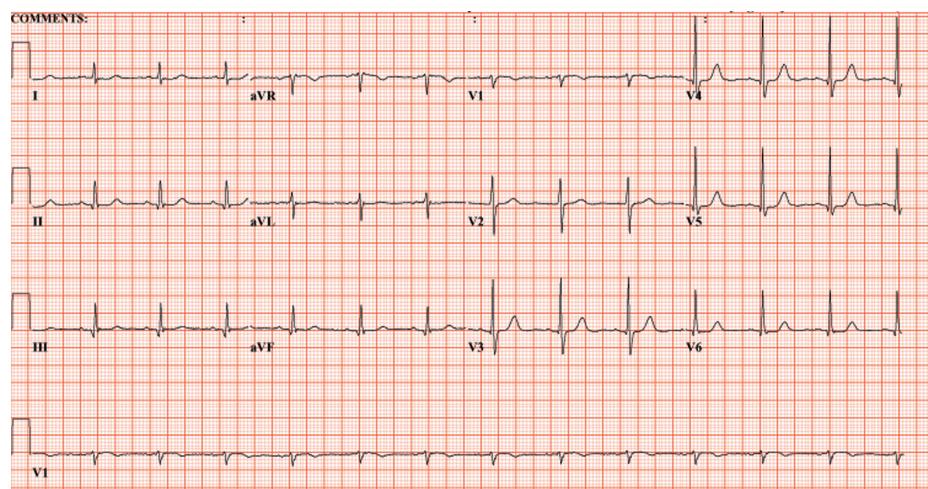


FIGURE 2.1-1. Normal electrocardiogram from a healthy subject. (Reproduced with permission from USMLE-Rx.com.)

TABLE 2.1-1. Axis Deviation by ECG Findings

	LEAD I	LEAD II	LEAD aVF	DEGREES
Normal axis	↑	↑	↑	⊖30–⊕90
Left axis deviation	↑	↓	↓	⊖30–⊖90
Right axis deviation	↓	↑	↑	⊕90–⊕180
Extreme axis	↓	↓	↓	⊖90–⊖180

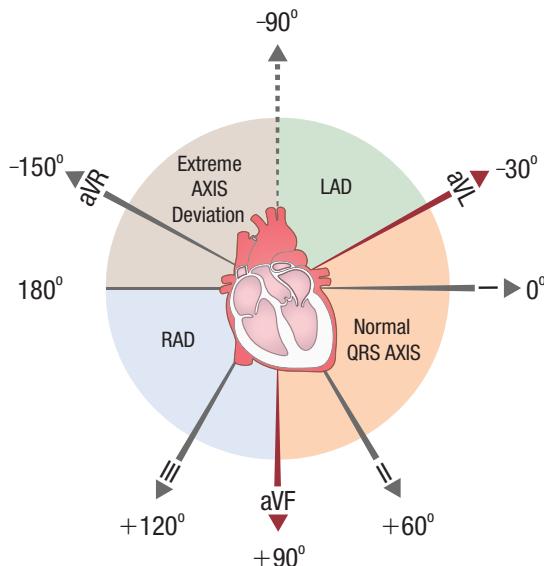


FIGURE 2.1-2. **ECG axis interpretation.** QRS axis and frontal leads. (Reproduced with permission from USMLE-Rx.com.)

Intervals

- **PR interval:** Normally 120–200 msec (3–5 small boxes).
 - Prolonged = delayed AV conduction (eg, first-degree heart block).
 - Short = fast AV conduction down accessory pathway (eg, WPW syndrome).
- **QRS interval:** Normally < 120 msec. A normal Q wave is < 40 msec wide and < 2mm deep. Ventricular conduction defects can cause a widened QRS complex (> 120 msec):
 - **Left bundle branch block (LBBB):** Deep S wave and no R wave in V₁ ("W"-shaped); wide, tall and broad, or notched ("M"-shaped) R waves in I, V₅, and V₆ (see Figure 2.1-3). A new LBBB is pathologic and may be a sign of acute MI.
 - **Right bundle branch block (RBBB):** RSR' complex ("rabbit ears;" "M"-shaped); qR or R morphology with a wide R wave in V₁; QRS pattern with a wide S wave in I, V₅, and V₆ (see Figure 2.1-4).
- **QT interval:** Normally QTc (the QT interval corrected for extremes in heart rate) is 380–440 msec (QTc = QT/√RR). Long QT syndrome (QTc > 440 msec) is an underdiagnosed congenital disorder that predisposes to ventricular tachyarrhythmias. Other common causes of prolonged QTc: acute MI, bradycardia, myocarditis, ↓ K⁺, ↓ Ca²⁺, ↓ Mg²⁺, congenital syndromes, head injury, drugs.
- **Jervell and Lange-Nielsen syndrome:** Long QT syndrome due to a defect in K⁺ channel conduction. Associated with sensorineural deafness. Treat with β-blockers and pacemaker.

Ischemia/Infarction

Acute ischemia:

- Within hours, peaked T-waves and ST-segment changes (either depression or elevation).
- Within 24 hours, T-wave inversion and ST-segment resolution.
- Within a few days, pathologic Q waves (> 40 msec or more than one-third of the QRS amplitude). Q waves usually persist, but may resolve in 10% of patients. Because of this, Q waves signify either acute or prior ischemic events.

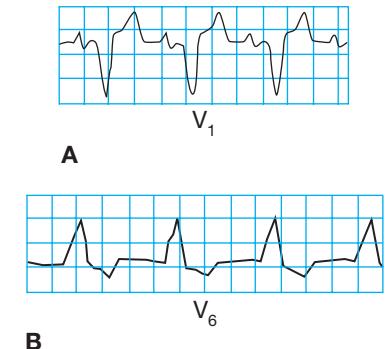


FIGURE 2.1-3. **Left bundle branch block.** Characteristic ECG findings are seen in leads V₁ (A) and V₆ (B). (Modified with permission from USMLE-Rx.com.)



MNEMONIC

Left bundle branch block—

WiLLiaM

V1 = **W** QRS pattern

V6 = **M** QRS pattern

Right bundle branch block—

MaRRoW

V1 = **M** QRS pattern

V6 = **W** QRS pattern

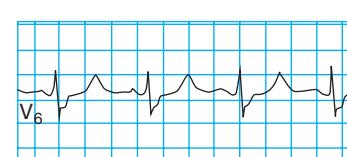
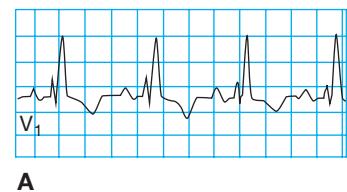


FIGURE 2.1-4. **Right bundle branch block.** Characteristic ECG findings are seen in leads V₁ (A) and V₆ (B). (Modified with permission from USMLE-Rx.com.)

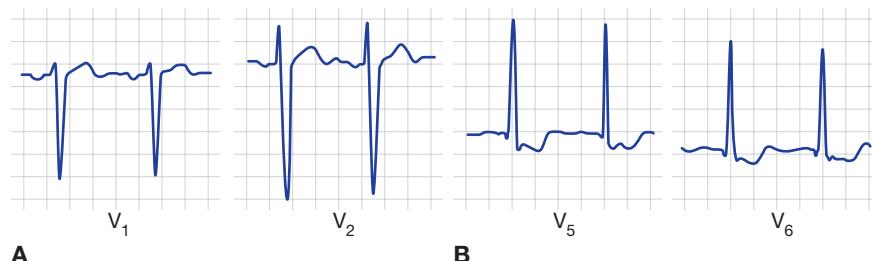


FIGURE 2.1-5. **Left ventricular hypertrophy.** Shown are leads V₁, V₂, V₅, and V₆. → S wave in V₁ + R wave in V₅ = 45 mm. Note ST changes and T-wave inversion in V₅ and V₆, suggesting strain. (Reproduced with permission from USMLE-Rx.com.)

- Non-Q-wave infarcts (also known as subendocardial infarcts) have ST and T changes without Q waves.
- In a normal ECG, R waves increase in size compared to the S wave between leads V₁ and V₅. Poor R-wave progression refers to diminished R waves in these precordial leads, and can be a sign of infarction, although it is not specific.

KEY FACT

- Pulmonale causes **P**eaked P waves.
- Mitrale causes **M**-shaped P waves.

MNEMONIC

Heart auscultation locations—

All Physicians Take Money

Aortic
Pulmonic
Tricuspid
Mitral

- Aortic
- Pulmonic
- Tricuspid
- Mitral

Chamber Enlargement

- Atrial enlargement:
 - Right atrial abnormality (**P pulmonale**): The P-wave amplitude in lead II is > 2.5 mm.
 - Left atrial abnormality (**P mitrale**): The P-wave width in lead II is > 120 msec, or terminal ⊖ deflection in V₁ is > 1 mm in amplitude and > 40 msec in duration. Notched P waves can frequently be seen in lead II.
- Left ventricular hypertrophy (LVH; see Figure 2.1-5):
 - Amplitude of S in V₁ + R in V₅ or V₆ is > 35 mm.
 - Alternative criteria: The amplitude of R in aVL + S in V₃ is > 28 mm in men or > 20 mm in women.
 - Usually associated with ST depression and T-wave changes.

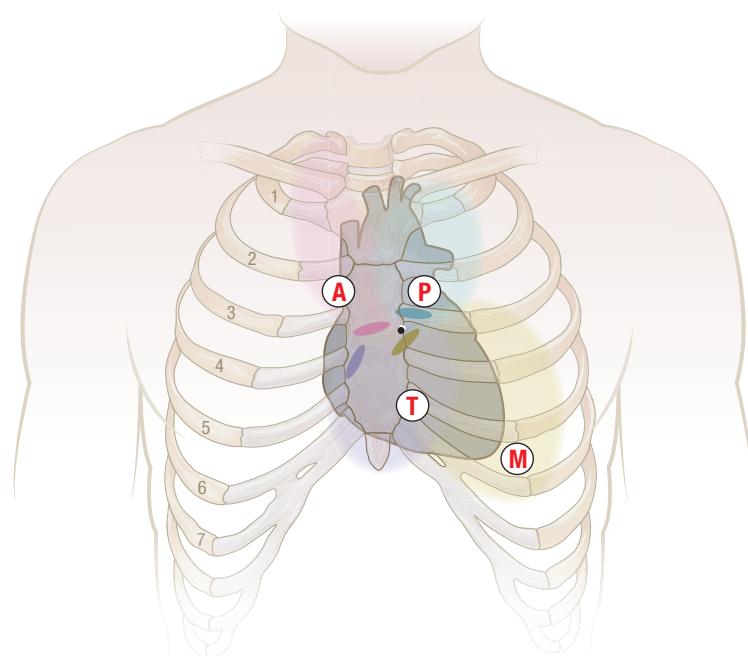


FIGURE 2.1-6. **Auscultation locations.** Auscultation sites are shown with associated valves. A, aortic valve; M, mitral valve; P, pulmonic valve; T, tricuspid valve. (Modified with permission from USMLE-Rx.com.)

- **Right ventricular hypertrophy (RVH):**
 - Right-axis deviation and an R wave in $V_1 > 7$ mm.

Cardiac Physical Exam

Key exam findings that can narrow the differential include the following:

- **Jugular venous distention** (JVD > 4 cm above the sternal angle): Most typically from volume overload, stemming from conditions such as right heart failure or pulmonary hypertension.
- **Hepatojugular reflux** (distention of neck veins upon applying pressure to the liver): Seen in same conditions as JVD.
- **Kussmaul sign** (\uparrow in jugular venous pressure [JVP] with inspiration): Often seen in constrictive pericarditis.
- **Systolic murmurs** (see Table 2.1-2 and Figures 2.1-6 and 2.1-7):
 - **Aortic stenosis:** A harsh systolic ejection murmur that radiates to the carotids.
 - **Mitral regurgitation:** A holosystolic murmur that radiates to the axilla.
 - **Mitral valve prolapse:** A midsystolic or late systolic murmur with a preceding click.
 - **Flow murmur:** Usually a soft murmur that is position-dependent (very common and does not imply cardiac disease).
- **Diastolic murmurs** (see Table 2.1-2 and Figures 2.1-6 and 2.1-7): Always abnormal.
 - **Aortic regurgitation:** An early decrescendo murmur.
 - **Mitral stenosis:** A mid to late low-pitched murmur.
- **Gallops:**
 - **S3 gallop:** A sign of fluid overload (ie, heart failure, mitral valve disease); often normal in younger patients and in high-output states (eg, pregnancy).
 - **S4 gallop:** A sign of decreased compliance (ie, hypertension, aortic stenosis, diastolic dysfunction); usually pathologic but can be normal in younger patients and in athletes.
- **Edema:**
 - **Pulmonary:** Left heart failure (fluid “backs up” into the lungs).
 - **Peripheral:** Right heart failure and biventricular failure (fluid “backs up” into the periphery), nephrotic syndrome, hepatic disease, lymphedema, hypoalbuminemia, and drugs.
- **Hands:**
 - **Finger clubbing:** Congenital cyanotic heart disease; endocarditis.
 - **Infective endocarditis:** Splinter hemorrhages; Osler nodes, Janeway lesions.

KEY FACT

Axis deviation can be a sign of ventricular enlargement.

KEY FACT

Right-sided murmurs increase with inspiration. Left-sided murmurs increase with expiration.

TABLE 2.1-2. Cardiac Murmurs

SYSTOLIC MURMURS	DIASTOLIC MURMURS
Aortic stenosis	Aortic regurgitation
Mitral regurgitation	Mitral stenosis
Mitral valve prolapse	
Tricuspid regurgitation	

Q

A college-age man passed out while playing basketball and had no prodromal symptoms or signs of seizure. His cardiac exam is unremarkable, and an ECG shows a slurred upstroke of the QRS. What are the next best steps?

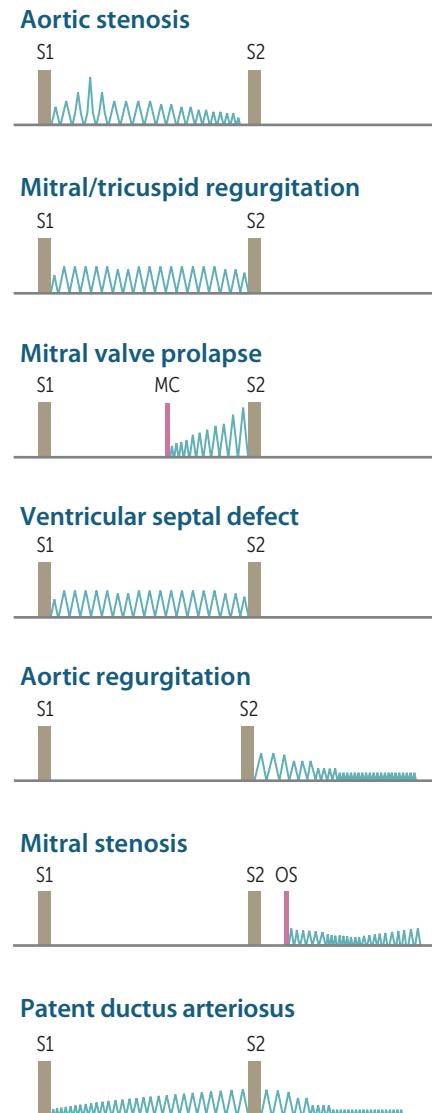


FIGURE 2.1-7. Heart murmurs. Visual representations of common heart murmurs are shown in relation to S1 and S2. MC, midsystolic click; OS, opening snap. (Reproduced with permission from USMLE-Rx.com.)

KEY FACT

An atrial myxoma is a benign tumor of the heart, commonly found within the left and right atria on the interatrial septum that can present with atrial fibrillation or mimic infective endocarditis. On auscultation, you will often hear a tumor “Plop” or see a tumor on echocardiography. Patients may develop systemic embolization from breakoff of tumor, leading to stroke. Resection of the tumor is the only treatment.

- **Peripheral pulses:**
 - **Increased:** Compensated aortic regurgitation (bounding pulses); coarctation (greater in arms than in legs); patent ductus arteriosus.
 - **Decreased:** Peripheral arterial disease; late-stage heart failure.
 - **Collapsing** (“waterhammer”): Aortic incompetence; AV malformations; patent ductus arteriosus; thyrotoxicosis, severe anemia.
 - **Pulsus paradoxus** (\downarrow systolic BP > 10 mm Hg with inspiration): Cardiac tamponade; pericardial constriction; also seen in obstructive lung diseases (eg, severe asthma), tension pneumothorax, and foreign body in airway.
 - **Pulsus alternans** (alternating weak and strong pulses): Cardiomyopathy; impaired left ventricular systolic function (LVF). Poor prognosis.
 - **Pulsus parvus et tardus** (weak and delayed pulse): Aortic stenosis.
 - **Jerky:** hypertrophic obstructive cardiomyopathy (HOCM).
 - **Pulsus bisferiens** (bifid pulse/“twice beating”): Aortic regurgitation; combined aortic stenosis and aortic regurgitation, HOCM.

This is Wolff-Parkinson-White syndrome (WPW). Advise against vigorous physical activity, use procainamide for arrhythmias, and refer for an electrophysiology study. Calcium channel blockers are contraindicated.

Arrhythmias

BRADYARRHYTHMIAS AND CONDUCTION ABNORMALITIES

Table 2.1-3 outlines the etiologies, clinical presentation, and treatment of common bradyarrhythmias and conduction abnormalities.



MNEMONIC

Management options for atrial fibrillation—

ABCD

Anticoagulate

β-blockers to control rate

Cardiovert/Calcium channel blockers

Digoxin (in refractory cases)

TABLE 2.1-3. Bradyarrhythmias and Conduction Abnormalities

Type	Etiology	Signs/Symptoms	ECG Findings	Treatment
Sinus bradycardia	Normal response to cardiovascular conditioning Can also result from sinus node dysfunction, β-blocker or CCB excess; therefore, it is important to review medications	May be asymptomatic, but may also present with light-headedness, syncope, chest pain, or hypotension	Sinus rhythm Ventricular rate < 60 bpm 	None if asymptomatic and rate > 40 bpm; atropine may be used to ↑ heart rate Pacemaker implant is the definitive treatment in severe cases
First-degree AV block	Can occur in normal individuals; associated with ↑ vagal tone, β-blocker or CCB use	Asymptomatic	PR interval > 200 msec 	None necessary
Second-degree AV block (Mobitz type I/ Wenckebach)	Drug effects (digoxin, β-blockers, CCBs) or ↑ vagal tone; right coronary ischemia or infarction	Usually asymptomatic	Progressive PR lengthening until a dropped beat occurs; the PR interval then resets 	None if asymptomatic Stop the offending drug Atropine as clinically indicated
Second-degree AV block (Mobitz type II)	Results from fibrotic disease of the conduction system or from acute, subacute, or prior MI	Occasionally syncope; frequent progression to third-degree AV block	Unexpected dropped beat(s) without a change in PR interval 	Pacemaker placement (even if asymptomatic)
Third-degree AV block (complete)	No electrical communication between the atria and ventricles	Syncope, dizziness, acute heart failure, hypotension, cannon A waves	No relationship between P waves and QRS complexes 	Pacemaker placement
Sick sinus syndrome/tachycardia-bradycardia syndrome	Heterogeneous disorder that leads to intermittent supraventricular tachyarrhythmias and bradyarrhythmias	2° to tachycardia or bradycardia; AF and thromboembolism may occur → syncope, palpitations, dyspnea, chest pain, TIA, and/or stroke		Most common indication for pacemaker placement Anticoagulate in atrial fibrillation/flutter to prevent systemic emboli

KEY FACT

Patients with persistent tachyarrhythmia (narrow- or wide-complex) causing hemodynamic instability should be managed with immediate synchronized cardioversion.

TACHYARRHYTHMIAS

Tables 2.1-4 and 2.1-5 outline the etiologies, clinical presentation, and treatment of common supraventricular and ventricular tachyarrhythmias.

TABLE 2.1-4. Supraventricular Tachyarrhythmias

Type	Etiology	Signs/Symptoms	ECG Findings	Treatment
ATRIAL				
Sinus tachycardia	Normal physiologic response to fear, pain, and exercise Can also be 2° to hyperthyroidism, volume contraction, infection, or PE	Palpitations, shortness of breath	Sinus rhythm Ventricular rate > 100 bpm	Treat the underlying cause
Atrial fibrillation (AF)	Acute AF— PIRATES: P ulmonary disease I nsomnia R heumatic heart disease A nemia/ A trial myxoma T hyrotoxicosis E thanol S eptis Chronic AF—HTN, CHF Most often caused by ectopic foci within the pulmonary veins	Often asymptomatic and incidental but can present with shortness of breath, chest pain, dizziness, fatigue, or palpitations. May present with congestive heart failure, cardiogenic shock, or devastating cerebrovascular accident Physical exam reveals an irregular pulse	No discernible P waves, with variable and irregular QRS response	For chronic AF, initial therapy: Rate control with β-blockers, CCBs, or digoxin Anticoagulate with warfarin or novel oral anticoagulant (NOAC) for patients with CHA ₂ DS ₂ -VASc score ≥ 2 For unstable AF, or new-onset AF (of < 2 days) cardiovert If > 2 days or unclear duration, must get TEE to rule out atrial clot
Atrial flutter	Circular movement of electrical activity around the atrium at a rate of approximately 300 times per minute	Usually asymptomatic but can present with palpitations, syncope, and lightheadedness	Regular rhythm; “saw-tooth” appearance of P waves can be seen The atrial rate is usually 240–320 bpm, and the ventricular rate is ~150 bpm	Anticoagulation, rate control, and cardioversion guidelines as in AF above
Multifocal atrial tachycardia	Multiple atrial pacemakers or reentrant pathways; associated with COPD, hypoxemia	May be asymptomatic. At least three different P-wave morphologies	Three or more unique P-wave morphologies; rate > 100 bpm	Treat as AF but avoid β-blockers because of chronic lung disease (if present)

(continues)

TABLE 2.1-4. Supraventricular Tachyarrhythmias (*continued*)

Type	Etiology	Signs/Symptoms	ECG Findings	Treatment
ATRIOVENTRICULAR JUNCTION				
Atrioventricular nodal reentry tachycardia (AVNRT)	A reentry circuit in the AV node depolarizes the atrium and ventricle nearly simultaneously	Palpitations, shortness of breath, angina, syncope, lightheadedness	Rate 150–250 bpm; P wave is often buried in QRS or shortly after	Cardiovert if hemodynamically unstable. Vagal maneuvers (eg, carotid massage, Valsalva, ice immersion (dive reflex). Adenosine if vagal maneuver fails
Atrioventricular reentrant tachycardia (AVRT)	An ectopic connection between the atrium and ventricle that causes a reentry circuit Seen in WPW	Palpitations, shortness of breath, angina, syncope, lightheadedness	A retrograde P wave is often seen after a normal QRS A reexcitation delta wave is characteristically seen in WPW	Except for WPW, same as that for AVNRT WPW listed separately below
Wolff-Parkinson-White (WPW) syndrome	Abnormal fast accessory conduction pathway from atria to ventricle (Bundle of Kent)	Palpitations, dyspnea, dizziness, and rarely cardiac death	Characteristic delta wave with widened QRS complex and shortened PR interval (see Figure 2.1-8)	Observation for asymptomatics Acute therapy is procainamide or amiodarone SVT gets worse after CCBs or digoxin (dangerous in WPW). Radiofrequency catheter ablation is curative
Paroxysmal atrial tachycardia	Rapid ectopic pacemaker in the atrium (not sinus node)	Palpitations, shortness of breath, angina, syncope, lightheadedness	Rate > 100 bpm; P wave with an unusual axis before each normal QRS	Adenosine can be used to unmask underlying atrial activity by slowing down the rate

Congestive Heart Failure

A clinical syndrome caused by inability of the heart to pump enough blood to maintain fluid and metabolic homeostasis. Risk factors include the following:

- Coronary heart disease.
- Hypertension.
- Cardiomyopathy.
- Valvular heart disease.
- Diabetes.
- COPD (cor pulmonale).

The American Heart Association/American College of Cardiology guidelines classify heart failure according to clinical syndromes, but alternative classification systems, including that of the New York Heart Association (NYHA), include functional severity, left-sided vs right-sided failure, and systolic vs nonsystolic failure (see Tables 2.1-6–2.1-8).

KEY FACT

Use the **CHA₂DS₂-VASc** scoring system to estimate stroke risk in atrial fibrillation, and anticoagulate with NOAC (eg, dabigatran, rivaroxaban, apixaban, and edoxaban) or warfarin (used with metal valves or mitral stenosis) for a score of 2 or more:

- **CHF** (1 point).
- **HTN** (1 point).
- **Age** ≥ 75 (2 points).
- **Diabetes** (1 point).
- **Stroke or TIA history** (2 points).
- **Vascular disease** (1 point).
- **Age** 65–74 (1 point).
- **Sex category (female)** (1 point).

TABLE 2.1-5. Ventricular Tachyarrhythmias

Type	Etiology	Signs/Symptoms	ECG Findings	Treatment
Premature ventricular contraction (PVC)	Ectopic beats arise from ventricular foci. Associated with hypoxia, fibrosis, ↓ LV function, electrolyte abnormalities, and hyperthyroidism	Usually asymptomatic but may lead to palpitations	Early, wide QRS not preceded by a P wave PVCs are usually followed by a compensatory pause	Treat the underlying cause If symptomatic, give β-blockers or, occasionally, other antiarrhythmics
Ventricular tachycardia (VT)	Can be associated with CAD, MI, and structural heart disease	Nonsustained VT (lasts < 30 seconds) is often asymptomatic; sustained VT (lasts > 30 seconds) can lead to palpitations, hypertension, angina, and syncope Can progress to VF and death	Three or more consecutive PVCs; wide QRS complexes in a regular rapid rhythm; may see AV dissociation	Cardioversion if unstable. Antiarrhythmics (eg, amiodarone, lidocaine, procainamide) if stable
Ventricular fibrillation (VF)	Associated with CAD and structural heart disease Also associated with cardiac arrest (together with asystole)	Syncope, absence of BP, no pulse	Totally erratic wide-complex tracing	Immediate electrical defibrillation and ACLS protocol
Torsades de pointes	Associated with long QT syndrome, proarrhythmic response to medications, hypokalemia, congenital deafness, and alcoholism	Can present with sudden cardiac death; typically associated with palpitations, dizziness, and syncope	Polymorphous QRS; VT with rates between 150 and 250 bpm	Give magnesium initially and cardiovert if unstable Correct hypokalemia; withdraw offending drugs

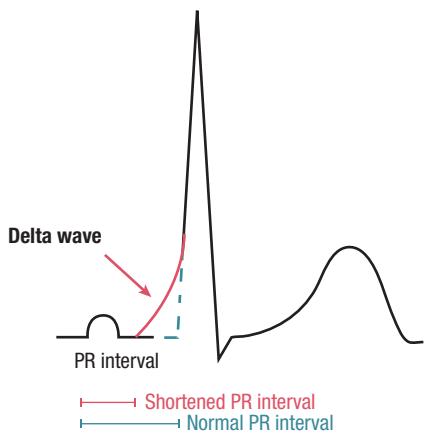


FIGURE 2.1-8. Ventricular tachyarrhythmias. Characteristic delta wave with widened QRS complex and shortened PR interval in WPW. (Reproduced with permission from USMLE-Rx.com.)

TABLE 2.1-6. NYHA Functional Classification of CHF

Class	Description
I	No limitation of activity; no symptoms (palpitations, dyspnea, and fatigue) with normal activity
II	Slight limitation of activity. Comfortable at rest or with mild exertion
III	Marked limitation of activity; comfortable only at rest
IV	Any physical activity brings on discomfort; symptoms (palpitations, dyspnea, and fatigue) present at rest

TABLE 2.1-7. Comparison of Systolic and Diastolic Dysfunction

VARIABLE	SYSTOLIC DYSFUNCTION/HEART FAILURE WITH REDUCED EJECTION FRACTION (HFrEF)	DIASTOLIC DYSFUNCTION/HEART FAILURE WITH PRESERVED EJECTION FRACTION (HFpEF)
Patient age	Often < 65 years of age	Often > 65 years of age
Comorbidities	Dilated cardiomyopathy, valvular heart disease, myocardial infarction	Restrictive or hypertrophic cardiomyopathy; renal disease or HTN
Physical exam	Displaced PMI, S3 gallop ("KEN"-tuc-ky)	Sustained PMI, S4 gallop ("Tenn"-es-SEE)
CXR	Pulmonary congestion, cardiomegaly	Pulmonary congestion
ECG/echocardiography	Q waves, ↓ EF (< 40%), dilation of the heart	LVH, normal/preserved EF (> 55%), abnormal LV diastolic indices

SYSTOLIC DYSFUNCTION/HEART FAILURE WITH REDUCED EJECTION FRACTION

A ↓ EF (< 40%) and ↑ left ventricular end-diastolic volumes. It is caused by inadequate left ventricular contractility or ↑ afterload. The heart compensates for ↓ EF and ↑ preload through hypertrophy and ventricular dilation (Frank-Starling law), but the compensation ultimately fails, leading to ↑ myocardial work and worsening systolic function.

HISTORY/PE

- Exertional dyspnea that progresses to orthopnea, paroxysmal nocturnal dyspnea (PND), and finally dyspnea at rest.
- Chronic cough, fatigue, and peripheral edema may be reported.
- Exam: parasternal lift, an elevated and sustained left ventricular impulse, an S3/S4 gallop, JVD, rales on lung exam, and peripheral edema.
- Look for signs to distinguish left- from right-sided failure (see Table 2.1-8).

DIAGNOSIS

- CHF is a clinical syndrome whose diagnosis is based on signs and symptoms.
- Diagnostic studies that may support diagnosis include the following:
 - **Best initial test:** Echocardiogram (transthoracic echocardiogram). ↓ EF and ventricular dilation may help pinpoint underlying cause (ie, AF, old MI, or LVH).
 - **ECG:** May show MI, heart block, arrhythmia.

KEY FACT

The most common cause of right-sided heart failure is left-sided heart failure.

KEY FACT

Hyponatremia parallels severity of heart failure and is an independent predictor of mortality in these patients.

MNEMONIC

CXR findings in CHF diagnosis—ABCDE

Alveolar edema ("Bat's wings")
Kerley B lines (interstitial edema)
Cardiomegaly
Dilated prominent upper lobe vessels
Effusion (pleural)

TABLE 2.1-8. Left-Sided vs Right-Sided Heart Failure

LEFT-SIDED CHF SYMPTOMS	RIGHT-SIDED CHF SYMPTOMS
Dyspnea predominates	Fluid retention predominates
Left-sided S3/S4 gallop	Right-sided S3/S4 gallop
Bilateral basilar rales	JVD
Pleural effusions	Hepatojugular reflux
Pulmonary edema	Peripheral edema
Orthopnea, paroxysmal nocturnal dyspnea	Hepatomegaly, ascites

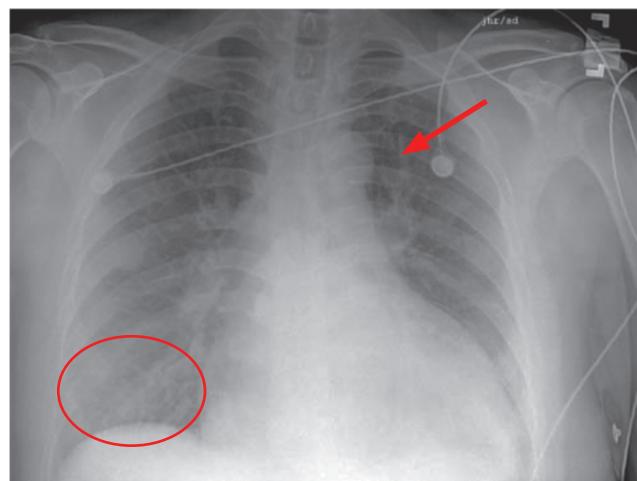


FIGURE 2.1-9. Chest x-ray (CXR) with evidence of congestive heart failure. Frontal CXR demonstrates marked cardiomegaly, cephalization of vessels (arrow), interstitial edema (circle), and left-sided pleural effusion that raise concern for CHF. (Reproduced with permission from Tintinalli JE et al. *Tintinalli's Emergency Medicine: A Comprehensive Study Guide*, 7th ed. New York, NY: McGraw-Hill; 2011.)

KEY FACT

Atrial tachycardia with AV block occurs 2° to digoxin toxicity.

MNEMONIC

Acute CHF management—

LMNOP

Lasix (furosemide)
Morphine
Nitrates
Oxygen
Position (sit upright)

- **CXR:** May show cardiomegaly, cephalization of pulmonary vessels, pleural effusions, vascular congestion, pulmonary edema, and prominent hila (see Figure 2.1-9).
- **Lab abnormalities:** Brain natriuretic peptide > 500 pg/mL, ↓ CBC (anemia), ↑ creatinine (sometimes), ↓ sodium in later stages, ↑ or ↓ TSH/T4 levels.

TREATMENT

Acute:

- **Pharmacologic therapy** (see Table 2.1-9):
 - Loop diuretics (most commonly) for aggressive diuresis.
 - ACEIs or ARB in combination with loop diuretics.
 - β -blockers should be avoided during decompensated CHF but should be restarted once patient is euvolemic.
- Correct underlying causes such as arrhythmias, myocardial ischemia, and drugs (eg, CCBs, antiarrhythmics, NSAIDs, alcohol, anemia, thyroid and valvular disease, high-output states).

TABLE 2.1-9. Types of Diuretics

CLASS	EXAMPLES	SIDE EFFECTS
Loop diuretics	Furosemide, ethacrynic acid, bumetanide, torsemide	Ototoxicity, hypokalemia, hypocalcemia, hyperuricemia, dehydration, gout
Thiazide diuretics	Hydrochlorothiazide, chlorothiazide, chlorthalidone	Hypokalemic metabolic alkalosis, hyponatremia, and hyperGLUC (hyperGlycemia, hyperLipidemia, hyperUricemia, hyperCalcemia)
K ⁺ -sparing agents	Spironolactone, eplerenone, triamterene, amiloride	Hyperkalemia, gynecomastia, sexual dysfunction. Eplerenone does not have antidiuretic effects that lead to gynecomastia
Carbonic anhydrase inhibitors	Acetazolamide	Hyperchloremic metabolic acidosis, neuropathy, NH ₃ toxicity, sulfa allergy
Osmotic agents	Mannitol	Pulmonary edema, dehydration. Contraindicated in anuria and CHF

- Treat acute pulmonary congestion with LMNOP (see Acute CHF management mnemonic).
- Acute decompensated heart failure:** Inotropic agents (eg, dobutamine) reduce left ventricular end-systolic volume for symptomatic improvement.

Chronic:

- Lifestyle:** Control comorbid conditions, and limit dietary sodium and fluid intake.
- Pharmacologic therapy:**
 - β-blockers and ACEIs/ARBs:** Help prevent remodeling of the heart and ↓ mortality for NYHA class II–IV patients. Avoid CCBs (can worsen edema).
 - Low-dose spironolactone:** Shown to ↓ mortality risk in patients with NYHA class III–IV heart failure.
 - Diuretics** (most commonly loop diuretics): Prevent volume overload.
 - Digoxin:** Symptomatic control of dyspnea and ↓ frequency of hospitalizations.
 - Daily ASA and a statin are recommended if the underlying cause is a prior MI.
- Advanced pharmacologic therapy:**
 - Sacubitril/valsartan:** angiotensin receptor-neprilysin inhibitor (ARNI) is a new drug class used in patients who continue to be dyspneic despite using the initial pharmacologic regimen. Provides mortality benefit for systolic dysfunction.
 - Ivabradine:** Reduces heart rate through SA nodal inhibition of the “funny channels.” Indicated in patients with systolic dysfunction if pulse is > 70 bpm or β-blockers are contraindicated.

Advanced treatments:

- Implantable cardiac defibrillator (ICD) in patients with an EF < 35%. Shown to ↓ mortality risk.
- Biventricular pacemaker in patient with an EF < 35%, dilated cardiomyopathy, and widened QRS complex with persistent symptoms.
- Left ventricular assist device (LVAD) or cardiac transplantation may be necessary in patients who are unresponsive to maximal medical therapy and biventricular pacemaker failure.

NONSYSTOLIC DYSFUNCTION/HEART FAILURE WITH PRESERVED EJECTION FRACTION

Defined by ↓ ventricular compliance with normal systolic function. The ventricle has either impaired active relaxation (2° to hypertension, ischemia, aging, and/or hypertrophy) or impaired passive filling (scarring from prior MI; restrictive cardiomyopathy). Left ventricular end-diastolic pressure ↑, cardiac output remains essentially normal, and EF is normal or ↑.

HISTORY/PE

Associated with stable and unstable angina, shortness of breath, dyspnea on exertion, arrhythmias, MI, heart failure, and sudden death.

TREATMENT

- Best initial treatment:** Diuretics (see Table 2.1-9).
- Maintain rate and BP control via β-blockers (first-line), ACEIs, ARBs, or CCBs.
- Digoxin and spironolactone are not beneficial in these patients.

KEY FACT

ACEIs/ARBs, ARNI, β-blockers, spironolactone or eplerenone, hydralazine/nitrates, and implantable defibrillator have mortality benefit in systolic dysfunction. Diuretics and digoxin (as well as other positive inotropic agents) are for symptomatic relief only and confer no mortality benefit. CCBs may ↑ mortality.

KEY FACT

Loop diuretics lose calcium; thiazides take it in. Both cause hypokalemia and hyperuricemia.

Q

1

A man was admitted for a CHF exacerbation with low EF. The patient is now ready for discharge, and his medications include furosemide and metoprolol. Assuming no contraindications, what medication would be appropriate to add to his treatment regimen?

Q

2

A woman with HTN and prior MI has an exam notable for a displaced PMI, an S3, a nonelevated JVP, and bibasilar rales. What is the next best step in diagnosis?

Cardiomyopathy

Myocardial disease; categorized as dilated, hypertrophic, or restrictive (see Table 2.1-10 and Figure 2.1-10).

KEY FACT

An S3 gallop signifies rapid ventricular filling in the setting of fluid overload and is associated with dilated cardiomyopathy. A S3 gallop sounds similar to the word "KEN-tuc-ky."

KEY FACT

An S4 gallop signifies a stiff, noncompliant ventricle and ↑ "atrial kick," and may be associated with hypertrophic cardiomyopathy. A S4 gallop sounds similar to the word "Tenn-es-SEE."

DILATED CARDIOMYOPATHY

The most common cardiomyopathy. Left ventricular dilation and ↓ EF must be present for diagnosis. Most cases are idiopathic, but known 2° causes include alcohol, postviral myocarditis, postpartum status, drugs (doxorubicin, AZT, cocaine), radiation, endocrinopathies (thyrotoxicosis, acromegaly, pheochromocytoma), infection (coxsackievirus, HIV, Chagas disease, parasites), genetic factors, and nutritional disorders (wet beriberi). The most common causes of 2° dilated cardiomyopathy are ischemia and long-standing hypertension.

HISTORY/PE

- Often presents with gradual development of CHF symptoms such as dyspnea on exertion, and diffuse edema of the ankles, feet, legs, and abdomen.
- Exam often reveals displacement of the left ventricular impulse, JVD, rales, an S3/S4 gallop, or mitral/tricuspid regurgitation.

DIAGNOSIS

- Echocardiography is diagnostic.
- CXR shows an enlarged, "balloon-like" heart and pulmonary congestion.

TREATMENT

- Address the underlying etiology (eg, alcohol use, endocrine disorders, infection).
- Treat CHF as noted in above section with lifestyle changes, and pharmacologic and advanced treatments.

TABLE 2.1-10. Differential Diagnosis of Cardiomyopathies

VARIABLE	TYPE		
	DILATED	HYPERTROPHIC	RESTRICTIVE
Major abnormality	Impaired contractility	Impaired relaxation	Impaired elasticity
Left ventricular cavity size (end diastole)	↑↑	↓	↓
Left ventricular cavity size (end systole)	↑↑	↓↓	↓
EF	↓↓	↑ (or normal)	Normal
Wall thickness	Usually ↓	↑↑	Usually ↑

1

A

Add an angiotensin-converting enzyme inhibitor (ACEI) to this patient's current regimen. ACEIs have been shown to have a ⊕ mortality benefit when used with β-blockers in NYHA class II–IV heart failure patients.

2

A

This patient has evidence of dilated cardiomyopathy. An echocardiogram would be the next best diagnostic step.

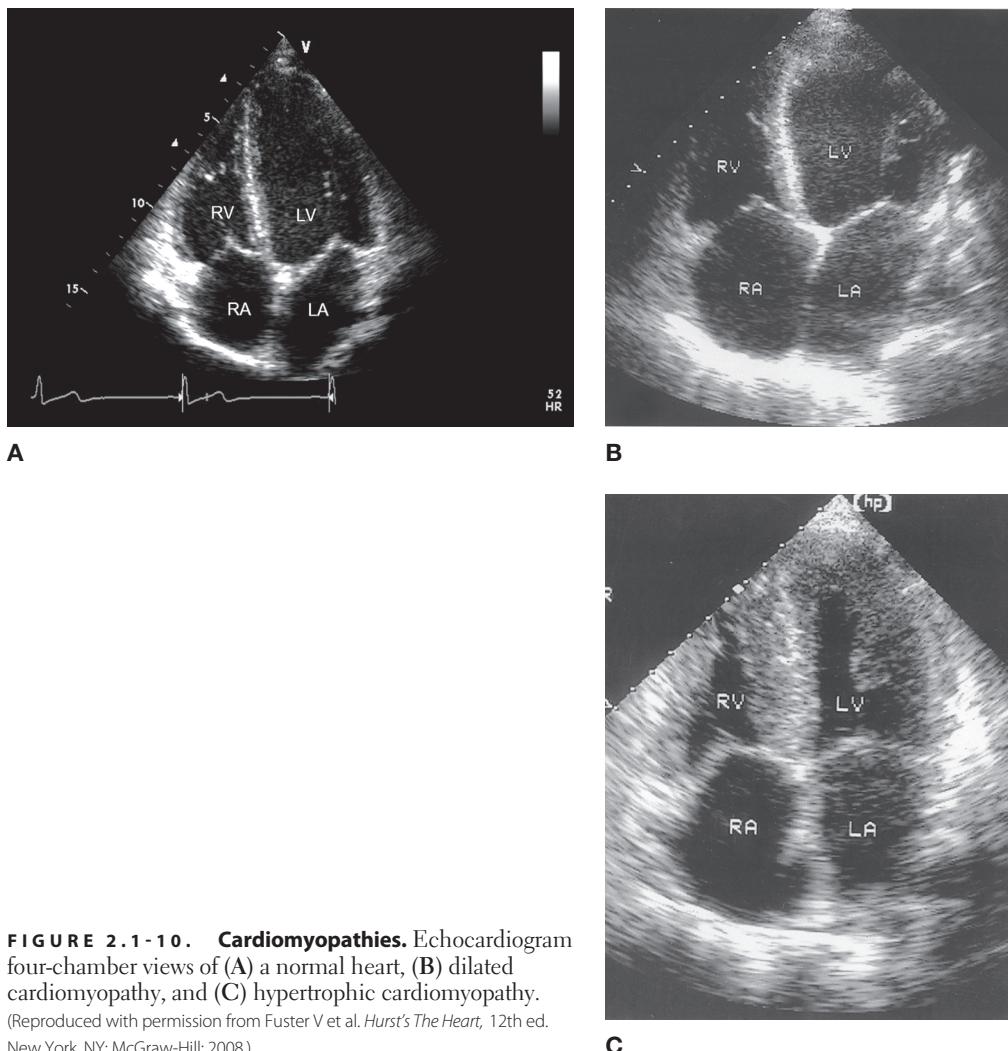


FIGURE 2.1-10. Cardiomyopathies. Echocardiogram four-chamber views of (A) a normal heart, (B) dilated cardiomyopathy, and (C) hypertrophic cardiomyopathy. (Reproduced with permission from Fuster V et al. *Hurst's The Heart*, 12th ed. New York, NY: McGraw-Hill; 2008.)

HYPERTROPHIC CARDIOMYOPATHY

Impaired left ventricular relaxation and filling (diastolic dysfunction) due to thickened ventricular walls secondary to stressors on the myocardium, such as HTN (most common cause) and aortic stenosis. Hypertrophy may also involve the interventricular septum, leading to left ventricular outflow tract obstruction and impaired ejection of blood due to asymmetric septal hypertrophy. The congenital form, hypertrophic obstructive cardiomyopathy (HOCM), is inherited as an autosomal dominant trait in 50% of HOCM patients and is the most common cause of sudden death in young, healthy athletes in the United States.

HISTORY/PE

- Patients are often asymptomatic but may also present with syncope, lightheadedness, dyspnea, palpitations, angina, or sudden cardiac death.
- Key finding is a harsh systolic ejection crescendo-decrescendo murmur in the lower left sternal edge that ↑ with ↓ preload (eg, Valsalva maneuver, standing) and ↓ with ↑ preload (eg, passive leg raise).
- Symptoms worsen with exercise, diuretics, dehydration, ACEIs/ARBs, digoxin, and hydralazine.

KEY FACT

HOCM is the most common cause of sudden death in young, healthy athletes in the United States.

- Exam also often reveals a sustained apical impulse, an S4 gallop, paradoxical S₂, and an abnormal bifid or bisferiens pulse (sudden quick rise followed by a slower longer rise due to LV outflow tract obstruction).

DIAGNOSIS

- **Best initial test:** Echocardiography is diagnostic and shows an asymmetrically hypertrophied interventricular septum and dynamic obstruction of blood flow (due to systolic anterior motion of the mitral valve against hypertrophied septum).
- ECG may be normal or show signs of LVH and nonspecific ST- and T-wave changes. Septal Q waves are common in HOCM (inferior and lateral leads).
- CXR may reveal left atrial enlargement (LAE) 2° to mitral regurgitation.

TREATMENT

- **Best initial treatment:** β -blockers are the best initial therapy for symptomatic relief in both HCM and HOCM; non-dihydropyridine CCBs (negative inotropic effect) and ventricular pacemakers are second-line agents.
- Digoxin and spironolactone are contraindicated. Diuretics may help in HCM but are contraindicated in HOCM.
- Implantable defibrillators should be used in symptomatic HOCM patients.
- Patients should avoid intense athletic competition and training.
- Surgical options for HOCM with persistent symptoms include partial excision or alcohol ablation of the myocardial septum.
- Surgical septal myomectomy is reserved for patients when medical and catheter procedures fail.

RESTRICTIVE CARDIOMYOPATHY

Decreased elasticity of myocardium leading to impaired diastolic filling without significant systolic dysfunction (a normal or near-normal EF). It is caused by infiltrative disease (eg, amyloidosis, sarcoidosis, hemochromatosis), scleroderma, Loeffler eosinophilic endocarditis, endomyocardial fibrosis, or by scarring and fibrosis (2° to radiation).

HISTORY/PE

Signs and symptoms of right-sided heart failure (JVD, peripheral edema, ascites, hepatomegaly) often predominate over left-sided failure, but dyspnea is the most common complaint.

DIAGNOSIS

- Echocardiography is key for diagnosis, with rapid early filling and a near-normal or elevated EF. CXR, MRI, and cardiac catheterization are helpful for characterization (eg, sarcoid, amyloidosis).
- Cardiac biopsy may reveal fibrosis or evidence of infiltration.
- ECG frequently shows LBBB; low voltages are seen in amyloidosis.

TREATMENT

Treat the underlying cause. Therapeutic options are limited and are generally palliative only. Medical treatment includes cautious use of diuretics for fluid overload and vasodilators to ↓ filling pressure.

Coronary Artery Disease

Also known as ischemic heart disease (IHD) or atherosclerotic heart disease. Clinical manifestations include stable and unstable angina, shortness of breath, dyspnea on exertion, arrhythmias, MI, heart failure, and sudden death.

Risk factors include the following:

- Diabetes mellitus (DM).
- Family history of premature CAD (men age < 55 years, women age < 65 years).
- Smoking.
- Hyperlipidemia.
- Abdominal obesity.
- HTN.
- Age (men age > 45 years, women age > 55 years).
- Male gender.
- CAD risk equivalents include DM, symptomatic carotid artery disease, peripheral arterial disease, and abdominal aortic aneurysm (AAA).

KEY FACT

Major risk factors for CAD include advanced age, male gender, ↑ LDL, ↓ HDL, HTN, a family history, and smoking. MI in menstruating women is rare.

ANGINA PECTORIS

Substernal chest pain 2° to myocardial ischemia (O_2 supply-and-demand mismatch). Mostly caused by atheroma. Less frequently caused by anemia, aortic stenosis, tachyarrhythmias, hypertrophic cardiomyopathy, and small vessel disease.

HISTORY/PE

- The classic triad consists of substernal chest pain that is usually precipitated by stress or exertion and is relieved by rest or nitrates (stable angina).
- The duration of stable angina is usually from 2–10 minutes (acute coronary syndrome is normally 10–30 minutes in duration).
- Pain can radiate to the neck or arm and may be associated with shortness of breath, nausea/vomiting, diaphoresis, dizziness, or lightheadedness.
- Pain is usually described as dull, squeezing, tightness, or pressure-like.
- Ischemic pain is not tender, positional, or pleuritic.

KEY FACT

Pain that is sharp or stabbing, or that changes with position, breathing, or touch, is less likely to be ischemic.

DIAGNOSIS

- **Best initial test:** ECG for any type of chest pain. Usually normal in angina pectoris, but may show ST-depression, flat or inverted T waves, or signs of past MI.
- **Cardiac enzymes (CK-MB/troponin):** Normal. May be required to be done in emergency setting after an ECG.
- **Stress testing:** ST-segment or wall-motion changes (using echo) with exercise or pharmacologic stress (dobutamine echo or dipyridamole thallium) are diagnostic of CAD. ECG stress test is not helpful without imaging for patients with abnormal baseline ECGs. (Note: Do not perform stress tests on asymptomatic patients with low pretest probability of disease.) Hold β -blockers, CCBs, and nitrates for 48 hours prior to stress test. Pharmacologic stress testing works due to coronary steal (diseased vessels are already maximally dilated while nondiseased vessels dilate in response to drugs, such as dipyridamole, leading to detectable ischemia).
- **Coronary angiography or CT coronary angiogram:** Coronary angiography, or a less invasive diagnostic test, CT coronary angiogram (availability

**KEY FACT**

Common causes of chest pain include GERD, angina, esophageal pain, musculoskeletal disorders (costochondritis, trauma), and pneumonia.

**KEY FACT**

Only ASA and β -blockers have been shown to have a mortality benefit in the treatment of angina.

varies among centers), may be used as a last resort if ECG or stress testing is equivocal.

- Rule out pulmonary, GI, or other cardiac causes of chest pain.

NONCARDIAC DIFFERENTIAL DIAGNOSIS

- **GERD:** History described as hoarseness, bad taste, and cough; relief of symptoms with proton pump inhibitors confirms diagnosis.
- **Musculoskeletal/costochondritis:** Pain is described as tender to palpation and movement.
- **Pneumonia/pleuritis:** Pain is described as worsening with breathing (pleuritic) and is often accompanied by fever and productive cough.
- **Anxiety:** Patients may have history of panic disorder or anxiety attacks.

TREATMENT

- **Chronic stable angina:** ASA, β -blockers, and nitroglycerin.
 - Nitroglycerin relieves pain due to \downarrow in left ventricular end-diastolic pressure and wall stress.
- Initiate risk-factor reduction (eg, smoking, cholesterol, HTN) through the initiation of ACEIs/ARBs, lipid-lowering therapies (ie, statins), and smoking cessation. Hormone replacement therapy is not protective in postmenopausal women.

PRINZMETAL (VARIANT) ANGINA

- Mimics angina pectoris but is caused by vasospasm of coronary vessels. It classically affects young women at rest (rather than during activity) in the early morning and is associated with ST-segment elevation. It is also associated with illicit drug use, especially cocaine.
- Patients usually do not have the standard risk factors for atherosclerosis.
- Tx: CCBs with or without long-acting nitrates. Aspirin is avoided as it can aggravate the ischemic attacks. β -blockers are contraindicated as they can \uparrow vasospasm.

CAROTID ARTERY STENOSIS

Atherosclerotic lesion of either (or both) carotid arteries. Accounts for 20% of transient ischemic attacks and embolic strokes.

HISTORY/PE

- Often asymptomatic.
- Symptomatic disease is characterized by sudden-onset focal neurologic defect in the past six months (ie, TIA or stroke).
- PE may reveal carotid artery bruit.
- **Risk factors:** Advanced age, smoking, HTN, hyperlipidemia, diabetes, obesity, and family history of CAD and/or carotid artery disease.

DIAGNOSIS

Duplex ultrasonography can determine percent occlusion.

TREATMENT

- **Definitive treatment:** Carotid endarterectomy (CEA).
 - Men with $\geq 60\%$ ($\geq 50\%$ if symptomatic) stenosis or women with $\geq 70\%$ stenosis benefit from CEA.
- Smaller lesions are monitored with serial duplex ultrasonography.

Acute Coronary Syndromes

A spectrum of clinical syndromes caused by plaque disruption or vasospasm that leads to acute myocardial ischemia.

UNSTABLE ANGINA/NON-ST-SEGMENT ELEVATION MYOCARDIAL INFARCTION

Chest pain that is (1) new onset, (2) accelerating (ie, occurs with less exertion, lasts longer, or is less responsive to medications), or (3) occurs at rest. Patient history distinguishes unstable angina from stable angina pectoris. Both stable and unstable angina have no elevated cardiac biomarkers. It signals the presence of possible impending infarction based on plaque instability. In contrast, NSTEMI indicates myocardial necrosis marked by elevations in troponin I and creatine kinase-MB isoenzyme (CK-MB) without ST-segment elevations seen on ECG.

DIAGNOSIS

- Patients should be risk stratified according to the Thrombolysis in Myocardial Infarction (TIMI) study criteria (see Table 2.1-11).
- **ECG:** Unstable angina and NSTEMI are not associated with ST elevation, but ST changes (eg, ST depression, T-wave inversion, nonspecific changes) may be seen on ECG.
- **Cardiac markers (CK-MB /troponin):** Unstable angina is not associated with elevated cardiac markers. NSTEMI is associated with elevations in cardiac markers.
- NSTEMI is diagnosed by serial cardiac enzymes and ECG.

TREATMENT

Best initial treatment:

- Admit to CCU, and monitor closely.
- If $\text{SaO}_2 < 90\%$ or breathless, administer O_2 .

TABLE 2.1-11. TIMI Risk Score for Unstable Angina/NSTEMI

CHARACTERISTICS	POINT
History	
Age ≥ 65 years	1
Three or more CAD risk factors (premature family history, DM, smoking, HTN, ↑ cholesterol, PAD, abdominal aortic aneurysm)	1
Known CAD (stenosis $> 50\%$)	1
ASA use in past 7 days	1
Presentation	
Severe angina (two or more episodes within 24 hours)	1
ST deviation ≥ 0.5 mm	1
+ cardiac marker	1
Risk score—total points^a	(0–7)

^aPatients at higher risk (risk score ≥ 3) benefit more from enoxaparin (vs unfractionated heparin), glycoprotein IIb/IIIa inhibitors, and early angiography.

KEY FACT

Acute coronary syndrome:

- Unstable angina: ECG—no ST elevation; cardiac biomarkers \ominus .
- NSTEMI: ECG—no ST elevation; cardiac biomarkers \oplus .
- STEMI: ECG—ST elevation; cardiac biomarkers \oplus .

- **Analgesia:** IV morphine with IV metoclopramide.
- **Nitrates:** IV, GTN, or sublingual.
- **Antiplatelet therapy:** ASA (\downarrow mortality in ACS) in combination with a second agent (ie, clopidogrel, prasugrel, or ticagrelor), unless contraindicated.
- Consider β -blockers as hemodynamics allow (if hypertensive/tachycardic/LV function $< 40\%$).
- Low-molecular-weight heparin (eg, enoxaparin) to prevent clot formation in the coronary arteries.

Interventions:

- Assess mortality risk (eg, TIMI score, GRACE score).
- Heparin is recommended for non-ST elevation MI. Thrombolytics are only recommended in STEMI if percutaneous coronary intervention (PCI) is not available within 2 hours.
- Patients with chest pain refractory to medical therapy, a TIMI score of ≥ 3 , a troponin elevation, or ST changes > 1 mm should be given GPIIb/IIIa inhibitors (abciximab, tirofiban, eptifibatide) and scheduled for angiography and possible revascularization within 72 hours (percutaneous coronary intervention [PCI] or coronary artery bypass graft [CABG]).
- Dual antiplatelet therapy with aspirin and prasugrel or ticagrelor (also P2Y₁₂ inhibitors but superior to clopidogrel) should be considered for up to 12 months after angioplasty and stenting to prevent restenosis of stenting.
- Ensure patient is on long-term β -blockers (if depressed LV function), ACEIs/ARBs, and statin.
- Address modifiable risk factors (ie, smoking, hypertension, hyperlipidemia, diabetes).



MNEMONIC

Treatment for MI—

Patient is MOANing Big from MI

Morphine

Oxygen (to maintain saturations)

ASA + Additional second antiplatelet agent (NSTEMI)

Nitrates

β -blockers



KEY FACT

It is important to check for aortic dissection clinically prior to administering anticoagulants or thrombolytics.

ST-SEGMENT ELEVATION MYOCARDIAL INFARCTION

ST-segment elevations and cardiac enzyme release 2° to prolonged cardiac ischemia and necrosis. STEMI is a common medical emergency, and prompt treatment is absolutely necessary.

HISTORY/PE

- **Presentation:** Acute-onset substernal chest pain ($> 10\text{--}30$ min), commonly described as a pressure, tightness, or heaviness that can radiate to the left arm, shoulders, neck, or jaw. May present without chest pain ("silent" infarct).
- **Associated symptoms:** Diaphoresis, shortness of breath, lightheadedness, anxiety, nausea/vomiting, epigastric pain (more common in women), and syncope.
- **PE:** May reveal arrhythmias, hypotension (cardiogenic shock), new S4, pansystolic murmur, and evidence of new CHF. Clear lung fields are seen in right ventricular MI (inferior MI). In a young, otherwise healthy person, consider cocaine use as the etiology.
- The best predictor of survival is left ventricular EF.
- **Differential diagnosis:** Angina, myocarditis, pericarditis, aortic dissection, pulmonary embolism, esophageal reflux/spasm.

DIAGNOSIS

- **ECG:** ST-segment elevations, hyperacute (tall) T waves, or new LBBB within hours. ST-segment depressions and dominant R waves in leads V₁–V₂ can also be reciprocal change indicating posterior wall infarct. T-wave inversion and pathologic Q waves develop within hours to days.
- **Sequence of ECG changes:** Peaked T waves \rightarrow ST-segment elevation \rightarrow Q waves \rightarrow T-wave inversion \rightarrow ST-segment normalization \rightarrow T-wave normalization over several hours to days.

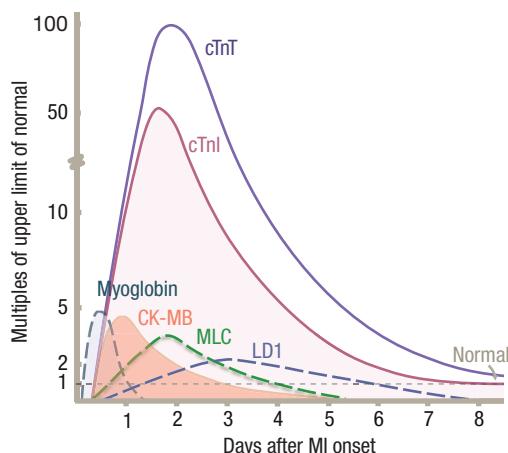


FIGURE 2.1-11. Typical pattern of serum marker elevation after an acute myocardial infarction. CK-MB, creatine kinase MB isoenzyme; cTnI, cardiac troponin I; cTnT, cardiac troponin T; LD1, lactate dehydrogenase isoenzyme 1; MLC, myosin light chain. (Reproduced with permission from USMLE-Rx.com.)

■ Cardiac enzymes:

- Troponin (T and I) is the most sensitive and specific cardiac marker.
- CK-MB and the CK-MB/total CK ratio (CK index) are also regularly checked.
- Both troponin and CK-MB can take up to 3–12 hours to rise following the onset of chest pain. Troponin peaks at 24–48 hours, and CK-MB peaks within 24 hours (see Figure 2.1-11).

■ ST-segment abnormalities:

- Inferior MI (involving the RCA/PDA):** ST-segment elevation in leads II, III, and aVF (see Figure 2.1-12). Obtain a right-sided ECG to look for ST elevations in the right ventricle.
- Anterior MI (involving LAD and diagonal branches):** ST-segment elevation in leads V₁–V₄ (see Figure 2.1-13).

KEY FACT

Women, diabetics, the elderly, and post-heart transplant patients may have atypical or clinically silent MIs.



FIGURE 2.1-12. Inferior wall myocardial infarction. In this patient with acute chest pain, the ECG demonstrated acute ST-segment elevation in leads II, III, and aVF with reciprocal ST-segment depression and T-wave flattening in leads I, aVL, and V₄–V₆. (Reproduced with permission from USMLE-Rx.com.)



FIGURE 2.1-13. Anterior wall myocardial infarction. This patient presented with acute chest pain. The ECG showed acute ST-segment elevation in leads V₁–V₆ and hyperacute T waves. (Reproduced with permission from USMLE-Rx.com.)

- **Lateral MI (involving LCA):** ST-segment elevation in leads I, aVL, and V₅–V₆.
- **Posterior MI:** ST-segment depression in leads V₁–V₂ (anterior leads) can be indicative. Obtain posterior ECG leads V₇–V₉ (15-lead) to assess for ST-segment elevations.

TREATMENT

Best initial treatment:

- **First line:** Antiplatelet therapy; ASA (\downarrow mortality in ACS). Add prasugrel or ticagrelor (both superior to clopidogrel), or clopidogrel as second antiplatelet agent with aspirin only for patients undergoing angioplasty or stenting.
- **Analgesia:** IV morphine with IV metoclopramide.
- **Nitrates:** IV, GTN, or sublingual.
- If $\text{SaO}_2 < 95\%$, breathless, or in acute LVF, administer O_2 .
- Consider β -blockers as hemodynamics allow (if hypertensive/tachycardic/LV function $< 40\%$).
- If the patient is in heart failure or in cardiogenic shock, do not give β -blockers; instead, give ACEIs provided that the patient is not hypotensive.
- In inferior wall MI (ie, right ventricular infarction), avoid nitrates and diuretics due to risk for severe hypotension (preload dependent).

KEY FACT

Contraindications to thrombolysis:

- Previous intracranial hemorrhage or major GI bleed.
- Recent major trauma/surgery/head injury.
- Ischemic stroke within the last 6 months.
- Severe hypertension ($> 180/110 \text{ mmHg}$).
- Known bleeding disorder.

Interventions:

- Emergent angiography and PCI should be performed if possible (superior to thrombolysis).
- If PCI cannot be performed < 120 minutes (door-to-balloon time should ideally be < 90 minutes), and there are no contraindications to thrombolysis, and the patient presents within 3 hours of chest pain onset, thrombolysis with tPA, reteplase, or streptokinase should be performed instead of PCI.
- Thrombolysis target time (door-to-needle time) is < 30 minutes and is contraindicated if > 24 hours. Thrombolytics can be used up to 12 hours from the onset of symptoms (mortality benefit extends to 12 hours).
- Long-term management (for all patients) includes ASA, ACEIs, β -blockers, nitrates, and high-dose statins.

- If PCI was performed, add clopidogrel, prasugrel, or ticagrelor (dual anti-platelet therapy).
- Address modifiable risk factors (ie, smoking, hypertension, hyperlipidemia, diabetes).

COMPLICATIONS

- **Arrhythmia:** VF and VT are the most common complications and the most common causes of sudden death following acute MI. Sinus bradycardia and third-degree (complete) heart block are also very common.
- Less common complications include reinfarction, left ventricular wall rupture, VSD, pericarditis, papillary muscle rupture (with mitral regurgitation), left ventricular aneurysm or pseudoaneurysm, and mural thrombi (with subsequent acute limb ischemia, TIA, or stroke).
- **A timeline of common post-MI complications:**
 - **First day:** Heart failure.
 - **2–4 days:** Arrhythmia, pericarditis.
 - **5–10 days:** Left ventricular wall rupture (acute pericardial tamponade causing electrical alternans, pulseless electrical activity, and JVD), papillary muscle rupture (severe mitral regurgitation, pulmonary edema), septal rupture (lower left sternal border murmur, increase in O₂ saturation in the right ventricle).
 - **Weeks to months:** Ventricular aneurysm (CHF, arrhythmia, persistent ST-segment elevation, mitral regurgitation, thrombus formation).
- Dressler syndrome, an autoimmune process occurring 2–10 weeks post-MI, presents with fever, pericarditis, pleural effusion, leukocytosis, and ↑ ESR.
- **Right ventricular infarction:** Caused by the occlusion of the RCA. Presents with hypotension, JVD, and clear lungs. Treat with high-volume fluid replacement (preload dependent), and avoid nitrates and diuretics.

Dyslipidemia

Total cholesterol level > 200 mg/dL, LDL > 130 mg/dL, triglycerides > 150 mg/dL, and HDL < 40 mg/dL, all of which are risk factors for CAD. Etiologies include obesity, DM, alcoholism, hypothyroidism, nephrotic syndrome, hepatic disease, Cushing syndrome, OCP use, high-dose diuretic use, and familial hypercholesterolemia.

HISTORY/PE

- Most patients have no specific signs or symptoms.
- Patients with extremely high triglyceride or LDL levels may have xanthomata (eruptive itchy nodules, orange streaks in palmar creases, or tuberous plaques on the elbows and knees); xanthelasma (yellow fatty deposits in the skin around the lids just below the eyes); lipemia retinalis (creamy appearance of retinal vessels); corneal arcus (deposition of lipid in the corneal stroma).
- Patients may have a history of familial primary hyperlipidemias.

DIAGNOSIS

- **Fasting lipid profile:** Total cholesterol, LDL, HDL, and triglycerides.
- Conduct a fasting lipid profile for patients > 35 years of age or in those ≥ 20 years of age with CAD risk factors, and repeat every 5 years or sooner if lipid levels are elevated.
- Total serum cholesterol > 200 mg/dL on two different occasions is diagnostic of hypercholesterolemia.
- LDL > 130 mg/dL or HDL < 40 mg/dL is diagnostic of dyslipidemia (not optimal levels), even if total serum cholesterol is < 200 mg/dL.
- Individuals with LDL > 190 mg/dL or triglycerides ≥ 500 mg/dL should be evaluated for secondary causes of hyperlipidemia.

KEY FACT

Indications for CABG:

- Left main coronary artery disease.
- Triple-vessel disease with ≥ 70% in each vessel.
- Two-vessel disease in diabetic patient.
- Symptomatic patient despite maximal medical therapy.

MNEMONIC

Complication of MI—

DARTH VADER

Death

Arrhythmia

Rupture (ventricular wall, septum, or papillary muscle)

Tamponade

Heart failure

Valvular disease

Aneurysm of ventricle

Dressler's syndrome

Emboli (mural thrombosis)

Recurrence/Reinfarction/Regurgitation (mitral)

KEY FACT

Right ventricular MI is caused by the occlusion of the RCA. Nitrates and diuretics must be avoided, and the condition is treated with IV fluids.

KEY FACT

Secondary hyperlipidemia causes include Cushing syndrome, hypothyroidism, nephrotic syndrome, or cholestasis.

KEY FACT

As you cannot calculate the patient's ASCVD risk on the USMLE, focus on obvious signs of ↑ risk (smoking, diabetes) or ↓ risk (young, healthy) when deciding if statin therapy is appropriate.

TABLE 2.1-12. American College of Cardiology/American Heart Association Treatment Guidelines

PATIENT AGE	CRITERIA	TREATMENT
≥ 21 years	Atherosclerotic cardiovascular disease (ASCVD) (eg, CAD, CVA, or PAD)	High-intensity statin
≥ 21 years	LDL ≥ 190 mg/dL	High-intensity statin
40–75 years	LDL 70–189 mg/dL without ASCVD or diabetes	≥ 7.5% 10-year risk → high-intensity statin 5–7.5% 10-year risk → moderate-intensity statin ≤ 5% 10-year risk → no statin
40–75 years	LDL 70–189 mg/dL with diabetes	≥ 7.5% 10-year risk → high-intensity statin ≤ 7.5% 10-year risk → moderate-intensity statin
≤ 75 years	ASCVD	High-intensity statin

TREATMENT

- Based on risk stratification using one of many cardiovascular risk calculators.
- The American College of Cardiology/American Heart Association recommendations are listed in Table 2.1-12.
- **High-intensity therapy:** Goal reduction in LDL of > 50%, moderate-intensity as lowering by 30–50% or by specific medication and dosing guidelines (ie, atorvastatin 80 mg).
- **Smokers of all ages:** Screen for dyslipidemias due to their ↑ risk.
- **First intervention:** 12-week trial of diet and exercise in a patient with no known ASCVD. Commonly used lipid-lowering agents are listed in Table 2.1-13.
- **Secondary hyperlipidemia:** Treat underlying cause.

KEY FACT

Dyslipidemia:

- LDL > 130 mg/dL or
- HDL < 40 mg/dL.

TABLE 2.1-13. Lipid-Lowering Agents

CLASS	EXAMPLES	EFFECT ON LIPID PROFILE	SIDE EFFECTS
HMG-CoA reductase inhibitors (statins)	Atorvastatin, simvastatin, lovastatin, pravastatin, rosuvastatin	↓ LDL, ↓ triglycerides	↑ LFTs, myositis, warfarin potentiation
Lipoprotein lipase stimulators (fibrates)	Gemfibrozil	↓ Triglycerides, ↑ HDL	GI upset, cholelithiasis, myositis (especially in combination with statins), ↑ LFTs, pancreatitis
Cholesterol absorption inhibitors	Ezetimibe	↓ LDL	Diarrhea, abdominal pain. Can cause angioedema
Niacin	Niaspan	↑ HDL, ↓ LDL	Skin flushing (can be prevented with ASA, due to ↑ prostaglandins), paresthesias, pruritus, GI upset, ↑ LFTs
Bile acid resins	Cholestyramine, colestipol, colesevelam	↓ LDL	Constipation, GI upset, LFT abnormalities, myalgias. Can ↓ absorption of other drugs from the small intestine
Proprotein convertase subtilisin/kexin type 9 (PCSK9) inhibitors	Evolocumab, alirocumab (injectable medications taken every 2–4 weeks)	↓ LDL	Injection-site swelling, rash, muscle/limb pain, backache

TABLE 2.1-14. American College of Cardiology/American Heart Association BP Guidelines^a

BP CATEGORY	BP	TREAT OR FOLLOW-UP
Normal	SBP < 120 mm Hg and DBP < 80 mm Hg	Yearly evaluation Lifestyle modifications to maintain normal BP
Elevated	SBP 120–129 mm Hg and DBP < 80 mm Hg	Recommend healthy lifestyle changes Reassess in 3–6 months
Hypertension: stage 1	SBP \geq 130–139 mm Hg or DBP \geq 80–89 mm Hg	Assess 10-year risk for heart disease and stroke (ASCVD) <ul style="list-style-type: none"> ■ < 10% 10-year risk: <ul style="list-style-type: none"> ■ Lifestyle recommendations ■ Reassess in 3–6 months ■ > 10% 10-year risk or the patient has known CVD, diabetes mellitus, or CKD, lifestyle changes, and BP-lowering medication: <ul style="list-style-type: none"> ■ Reassess in 1 month for effectiveness of medication therapy ■ If goal is met after 1 month, reassess in 3–6 months ■ If goal is not met after 1 month, consider different medication or titration; continue monthly follow-up until control is achieved
Hypertension: stage 2	SBP \geq 140 mm Hg or DBP \geq 90 mm Hg	Recommend healthy lifestyle changes and BP-lowering medication (two medications of different classes) <ul style="list-style-type: none"> ■ Reassess in 1 month for effectiveness: <ul style="list-style-type: none"> ■ If goal is met after 1 month, reassess in 3–6 months ■ If goal is not met after 1 month, consider different medications or titration ■ Continue monthly follow-up until control is achieved

^a Based on 2017 report of the American College of Cardiology/American Heart Association Task Force on Clinical Practice Guidelines.

Hypertension

A major risk factor for stroke and MI. Defined as systolic BP \geq 140 mm Hg and/or diastolic BP \geq 90 mm Hg based on three measurements separated in time in adults (see Table 2.1-14). Classified as 1° or 2°.

1° (ESSENTIAL) HYPERTENSION

Hypertension with no identifiable cause. Represents 95% of cases of HTN. Risk factors include a family history of HTN or heart disease, a high-sodium diet, smoking, obesity, ethnicity (African-Americans $>$ whites), and advanced age.

HISTORY/PE

- HTN is usually asymptomatic until complications develop.
- Patients should be evaluated for end-organ damage to the brain (stroke, dementia), eye (cotton-wool exudates, hemorrhage, retinopathy), heart (LVH), and kidney (proteinuria, CKD). Renal bruits may signify renal artery stenosis as the cause of HTN.

KEY FACT

PCSK9 inhibitors are a new class of LDL-lowering drugs. They significantly increase hepatic clearance of LDL. Indicated in familial hypercholesterolemia and statin-resistant or -intolerant patients with severe hyperlipidemia.

Q

A woman is found with pulseless electrical activity on hospital day 7 after suffering a lateral wall STEMI. The ACLS protocol is initiated. What is the next best step?

DIAGNOSIS

- **Quantify overall risk:** Fasting glucose, HbA1c, lipid profile.
- **Assess end-organ damage:** ECG to check for LVH or past MI, urinalysis for proteinuria or hematuria; BUN/creatinine.
- **Exclude secondary causes (as required):** U&Es, Ca²⁺, renal ultrasound, 24-hour urine metanephrine, renin, aldosterone, urinary cortisol.
- 24-hour ambulatory BP monitoring (ABPM) may be useful sometimes (ie, “white coat” or borderline hypertension).

**MNEMONIC**

Treatment of HTN—

ABCD

ACEIs/ARBs
β-blockers
CCBs
Diuretics (typically thiazide diuretics)

**MNEMONIC**

Causes of 2° hypertension—

CHAPS

Cushing syndrome
Hyperaldosteronism (Conn syndrome)
Aortic coarctation
Pheochromocytoma
Stenosis of renal arteries

RECENT

Renal causes (renal artery stenosis, PKD)
Endocrine (Conn syndrome, Cushing syndrome, hyperparathyroidism, pheochromocytoma)
Coarctation of the aorta
Estrogen (OCP)
Neurologic (ICP)
Thyroid disorder

TREATMENT

- Begin with lifestyle modifications (weight loss, exercise, smoking cessation, diet improvement, limit alcohol and salt intake).
- BP goals vary by category and ASCVD (see Table 2.1-14).
- Diuretics, CCBs, ACEIs, and β-blockers have been shown to ↓ mortality in uncomplicated HTN. They are first-line agents unless a comorbid condition requires another medication (see Table 2.1-15).
- For patients who are not African-American, including those with diabetes, → ACEI/ARB (nephroprotective), thiazide, CCB.
- For African American patients, including those with diabetes, → thiazide, CCB.
- For patients ≥ 18 years of age with CKD → ACEI/ARB (nephroprotective).
- If BP goal is not reached within 1 month of commencing treatment, ↑ dose of initial drug or add a second drug, and if goal BP cannot be reached with two drugs, add and titrate a third drug. Poorer outcomes are seen if ACEIs and ARBs are used together.
- Periodically test for end-organ complications, including renal complications (BUN, creatinine, urine protein-to-creatinine ratio), hypertensive retinopathy (eye exam), and cardiac complications (ECG evidence of LVH).

2° HYPERTENSION

Hypertension 2° to an identifiable organic cause (~5% of cases). See Table 2.1-16 for the diagnosis and treatment of common causes.

TABLE 2.1-15. Treatment of 1° Hypertension in Specific Populations

POPULATION	AGENTS
Uncomplicated	Diuretics, CCBs, ACEIs
CHF	Diuretics, β-blockers, ACEIs, ARBs, aldosterone antagonists
Diabetes	Diuretics, ACEIs, ARBs, CCBs
Post-MI	β-blockers, ACEIs, ARBs, aldosterone antagonists
Chronic kidney disease	ACEIs, ARBs
BPH	Diuretics, α ₁ -adrenergic blockers
Isolated systolic HTN	Diuretics, ACEIs, CCBs (dihydropyridines)
Pregnancy	Methyldopa, β-blockers (typically labetalol), hydralazine

A

This patient has probably suffered a left ventricular free-wall rupture with acute cardiac tamponade. Emergent pericardiocentesis is the next best therapeutic and diagnostic step.

TABLE 2.1-16. Common Causes of 2° Hypertension

Etiology	Description	Management
1° Renal disease	Often unilateral renal parenchymal disease	Treat with ACEIs, which slow the progression of renal disease
Renal artery stenosis	Especially common in patients < 25 and > 50 years of age with recent-onset HTN Etiologies include fibromuscular dysplasia (younger patients) and atherosclerosis (older patients) Often present with headaches and bruits in abdomen/neck	Diagnose with MRA, CT angiography, or renal artery Doppler ultrasound May be treated with angioplasty or stenting. Consider ACEIs in unilateral disease. (In bilateral disease, ACEIs can accelerate kidney failure by preferential vasodilation of the efferent arteriole.) Open surgery is a second option if angioplasty is not effective or feasible
OCP use	Common in women > 35 years of age, obese women, and those with long-standing use	Discontinue OCPs (effect may be delayed)
Pheochromocytoma	An adrenal gland tumor that secretes epinephrine and nor-epinephrine, leading to episodic headache, sweating, and tachycardia	Diagnose with urinary metanephrenes and catecholamine levels or plasma metanephrenine Surgical removal of tumor after treatment with α -blockers followed by β -blockers
Conn syndrome (hyperaldosteronism)	Most often 2° to an aldosterone-producing adrenal adenoma Causes the triad of HTN, unexplained hypokalemia, and metabolic alkalosis	Metabolic workup with plasma aldosterone and renin level; \uparrow aldosterone and \downarrow renin levels suggest 1° hyperaldosteronism. Surgical removal of tumor
Cushing syndrome	Due to an ACTH-producing pituitary tumor, an ectopic ACTH-secreting tumor, or cortisol secretion by an adrenal adenoma or carcinoma. Also due to exogenous steroid exposure. (See the Endocrinology chapter for more details)	Surgical removal of tumor; removal of exogenous steroids
Coarctation of the aorta	See the Pediatrics chapter	Surgical correction
Hyperparathyroidism	Either alone or as part of MEN type 2 (with pheochromocytoma) Look for \uparrow calcium and vascular/valvular calcification	Treat underlying hyperparathyroidism

HYPERTENSIVE CRISES

A spectrum of clinical presentations in which there is a severe increase in BP (usually $> 180/120$ mm Hg) that can lead to end-organ damage.

HISTORY/PE

Present with end-organ damage revealed by acute kidney injury, severe chest pain (ischemia, MI), back pain (aortic dissection), stroke, severe headache with changes in mental status (hypertensive encephalopathy), and blurred vision. Other symptoms include nausea and vomiting, seizures, shortness of breath, and severe anxiety.

Q

A 40-year-old man presents for a routine exam. His exam is significant for a BP of 145/75 mm Hg but is otherwise unremarkable, as are his lab results. What is the next best step?

TABLE 2.1-17. Major Classes of Antihypertensive Agents

CLASS	AGENTS	MECHANISM OF ACTION	SIDE EFFECTS
Diuretics	Thiazide, loop, K ⁺ sparing Ethacrynic acid is the only nonsulfonamide loop diuretic that can be used in severe sulfa allergy patients	↓ Extracellular fluid volume and thereby ↓ vascular resistance	Hypokalemia (not with K ⁺ sparing), hyperglycemia, hyperlipidemia, hyperuricemia, azotemia
β-blockers	Propranolol, metoprolol, nadolol, atenolol, timolol, carvedilol, labetalol	↓ Cardiac contractility and renin release	Bronchospasm (in severe active asthma), bradycardia, CHF exacerbation, impotence, fatigue, depression
ACEIs	Captopril, enalapril, fosinopril, benazepril, lisinopril	Blocks the conversion of angiotensin I to angiotensin II, reducing peripheral resistance and salt/water retention. Bradykinin ↑ due to the ↓ activation of ACE	Cough and angioedema (due to ↑ bradykinin build-up), rashes, leukopenia, hyperkalemia
ARBs	Losartan, valsartan, irbesartan	Blocks the activation of angiotensin II receptor, reducing peripheral resistance and salt/water retention	Rashes, leukopenia, and hyperkalemia but no cough
CCBs	Dihydro pyridines (nifedipine, felodipine, amlodipine), nondihydropyridines (diltiazem, verapamil)	↓ Smooth muscle tone and cause vasodilation; may also ↓ cardiac output	Dihydropyridines: headache, flushing, peripheral edema Nondihydropyridines: ↓ Contractility
Vasodilators	Hydralazine, minoxidil	↓ Peripheral resistance by dilating arteries/arterioles	Hydralazine: headache, lupus-like syndrome Minoxidil: orthostasis, hirsutism
α ₁ -adrenergic blockers	Prazosin, terazosin, phenoxybenzamine	Cause vasodilation by blocking actions of norepinephrine on vascular smooth muscle	Orthostatic hypotension
Centrally acting adrenergic agonists	Methyldopa, clonidine	Inhibit the sympathetic nervous system via central α ₂ -adrenergic receptors	Somnolence, orthostatic hypotension, impotence, rebound HTN

KEY FACT

Hypertensive emergencies are diagnosed on the basis of the extent of end-organ damage, not BP measurement.

DIAGNOSIS

- **Hypertensive urgency:** ↑ BP with mild to moderate symptoms (headache, chest pain, nausea and vomiting) without end-organ damage.
- **Hypertensive emergency:** ↑ BP with signs or symptoms of impending end-organ damage such as acute kidney injury, intracranial or retinal hemorrhage, papilledema, stroke, or ECG changes suggestive of ischemia, MI, or pulmonary edema.

A

With a single BP recording and no evidence of end-organ damage, the next best step should consist of a repeat BP measurement at the end of the exam with a return visit if BP is still high.

TREATMENT

- **Hypertensive urgency:** BP can be reduced gradually over 24–48 hours with oral antihypertensives (eg, β-blockers, clonidine, ACEIs) (see Table 2.1-17).
- **Hypertensive emergency:** BP must be reduced immediately to prevent imminent organ damage. Treat with IV medications (labetalol, nitroprusside, nicardipine) with the goal of lowering mean arterial pressure by no more than 25% over the first 2 hours to prevent cerebral hypoperfusion or coronary insufficiency.

Pericardial Disease

Consists of pericarditis, constrictive pericarditis, and pericardial tamponade. Results from acute or chronic pericardial insults; may lead to pericardial effusion.

PERICARDITIS

Inflammation of the pericardial sac. It can compromise cardiac output via tamponade (extravasation of large amounts of fluid secondary to pericarditis) or constrictive pericarditis (chronic pericarditis). Most commonly idiopathic, although known etiologies include viral infection (most common infection, likely etiology Coxsackie B virus), *Staphylococcus*, *Streptococcus*, tuberculosis (TB), systemic lupus erythematosus (SLE), uremia, drugs, radiation, connective tissue disorder (ie, rheumatoid arthritis, Goodpasture syndrome), and neoplasms. May also occur after MI (either within days after MI or as a delayed phenomenon; ie, Dressler syndrome) or open-heart surgery (see Figure 2.1-14).

HISTORY/PE

- **Presentation:** Sharp pleuritic chest pain, dyspnea, cough, and fever.
- **Key feature:** Chest pain tends to worsen in the supine position and with inspiration. Classically, patient is seen sitting up (pain improves in prone position) and bending forward.
- **Exam:** May reveal a pericardial friction rub. Elevated JVP, tachycardia, muffled S_1 and S_2 , and pulsus paradoxus (a ↓ in systolic BP >10 mm Hg on inspiration) can be present with pericardial tamponade. Kussmaul sign can be present with constrictive pericarditis.

DIAGNOSIS

- **ECG:** Include diffuse ST-segment elevation and PR-segment depressions followed by T-wave inversions (see Figure 2.1-15). Classically shows concave (saddle-shaped) ST segment elevation.

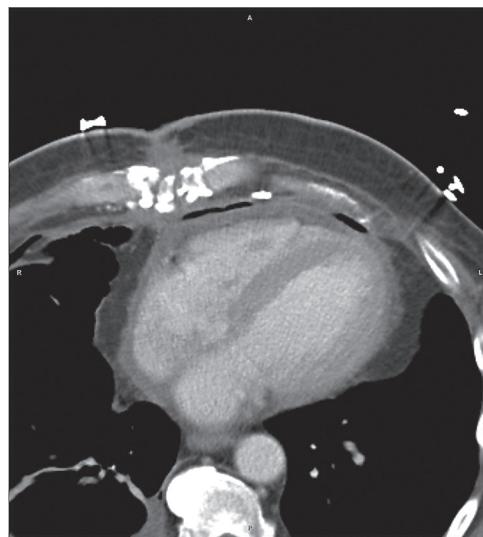


FIGURE 2.1-14. Radiographic findings in pericarditis. Contrast-enhanced CT at the level of the interventricular septum demonstrates a small pericardial effusion, with thickening and increased enhancement of the pericardium consistent with infection in this postsurgical patient. The air outlining the pericardium anteriorly is the result of dehiscence of the median sternotomy. (Reproduced with permission from USMLE-Rx.com.)

KEY FACT

Pericardial calcification seen on chest x-ray (CXR) strongly suggest constrictive pericarditis due to chronic fibrosis and calcification of the pericardium.

MNEMONIC

Causes of pericarditis—

CARDIAC RINDS

Collagen vascular disease
Aortic dissection
Radiation
Drugs
Infections
Acute renal failure
Cardiac (MI) and Connective tissue disease
Rheumatic fever
Injury
Neoplasms
Dressler syndrome
Surgery (postpericardiectomy syndrome)

KEY FACT

ST-segment elevations in pericarditis are differentiated from MI in that they are not localized to one region of the heart; widespread ST-segment elevations are seen.

- **CXR:** Cardiomegaly may indicate a pericardial effusion.
- **Blood tests:** FBC, ESR, U&Es, cardiac enzymes (troponin may be raised), viral serology, and if indicated, autoantibodies, fungal precipitins, and TFTs.
- **Echo:** Pericardial thickening or effusion may be evident.

TREATMENT

- Treat the underlying cause (eg, corticosteroids/immunosuppressants for SLE, dialysis for uremia) or symptoms (eg, ASA for post-MI pericarditis, ASA/NSAIDs for viral pericarditis or Dressler syndrome). Avoid corticosteroids within a few days after MI, as they can predispose to ventricular wall rupture.
- Treat idiopathic cases with NSAIDs such as ibuprofen, naproxen, or indomethacin. Consider colchicine for relapse or persistent symptoms.
- Pericardial effusions without symptoms can be monitored, but evidence of tamponade requires pericardiocentesis with continuous drainage as needed.

CARDIAC TAMPONADE

Excess fluid in the pericardial sac ↑ the intrapericardial pressure, leading to compromised ventricular filling and ↓ cardiac output. The rate of fluid formation is more important than the size of the effusion. Risk factors include pericarditis, malignancy, SLE, TB, and trauma (commonly stab wounds medial to the left nipple).

HISTORY/PE

- Presents with fatigue, dyspnea, anxiety, tachycardia, and tachypnea that can rapidly progress to shock and death.
- Exam of a patient with acute tamponade may reveal Beck triad (hypotension, distant or muffled S_1 and S_2 heart sounds, and JVD), a narrow pulse pressure, and pulsus paradoxus.
- Lung fields are clear on exam.

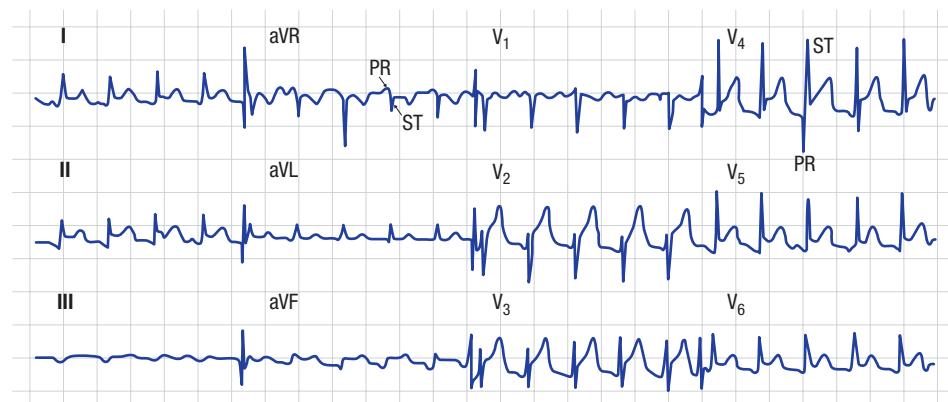


FIGURE 2.1-15. Acute pericarditis. Diffuse ST-segment elevations in multiple leads not consistent with any discrete coronary vascular territory and PR-segment depressions. (Reproduced with permission from USMLE-Rx.com.)

DIAGNOSIS

- Echo is diagnostic and shows right atrial and right ventricular diastolic collapse and echo-free zone around the heart.
- CXR may show an enlarged, globular, water-bottle-shaped heart with a large effusion (see Figure 2.1-16).
- If present on ECG, electrical alternans is diagnostic of a large pericardial effusion.

TREATMENT

- Aggressive volume expansion with IV fluids.
- Urgent pericardiocentesis (aspirate will be non-clotting blood). Send fluid to lab analysis to determine etiology.
- Decompensation or recurrent cases may warrant pericardial window.

Valvular Heart Disease

Until recently, rheumatic fever (which affects the mitral valve more often than the aortic valve) was the most common cause of valvular heart disease in US adults; the leading cause is now mechanical degeneration. Subtypes are listed in Table 2.1-18 along with their etiologies, presentation, diagnosis, and treatment.

Vascular Diseases**AORTIC ANEURYSM**

Greater than 50% dilation of all three layers of the aortic wall. Aortic aneurysms are most commonly associated with atherosclerosis. Most are abdominal, and > 90% originate below the renal arteries.

- Ascending aortic aneurysm—think cystic medial necrosis or connective tissue disease.
- Descending aortic aneurysm—think atherosclerosis.
- Complications:** Rupture, thrombosis, embolism, fistulae, pressure on surrounding structures.

HISTORY/PE

- Usually asymptomatic and discovered incidentally on exam or radiologic study. It may cause mild abdominal or back pain.
- Exam demonstrates a pulsatile abdominal mass or abdominal bruits.
- Risk factors include HTN, high cholesterol, other vascular disease, a \oplus family history, smoking (strongest predictor of rupture), gender (males > females), and age.
- Ruptured aneurysm leads to hypotension and severe, tearing abdominal pain that radiates to the back, iliac fossae, or groin, and syncope.

DIAGNOSIS

- Screening:** All men 65–75 years of age with a history of smoking are recommended for a one-time screening by ultrasound for AAA (see Figure 2.1-17).
- Abdominal ultrasound is used for diagnosis or to follow the course of an aneurysm over time.
- CT with contrast or MRA may be useful to determine the precise anatomy.

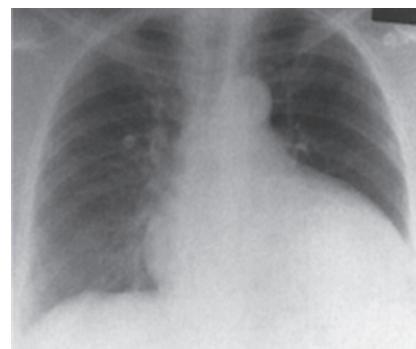


FIGURE 2.1-16. Pericardial effusion. Water-bottle-shaped heart seen on CXR with pericardial effusion. (Reproduced with permission from Chen MY et al. *Basic Radiology*, 2nd ed. New York, NY: McGraw-Hill; 2011.)

KEY FACT

Beck triad can diagnose acute cardiac tamponade:

- JVD.
- Hypotension.
- Distant S_1 and S_2 heart sounds.

KEY FACT

Size of AAA determines treatment:

- < 5 cm \rightarrow monitoring.
- > 5 cm \rightarrow surgical correction.

Q

A 20-year-old man presents with an initial BP of 150/85 mm Hg, and repeat measurement yields 147/85 mm Hg. The patient's potassium level is 3.2 mg/dL. What is the next appropriate diagnostic step?

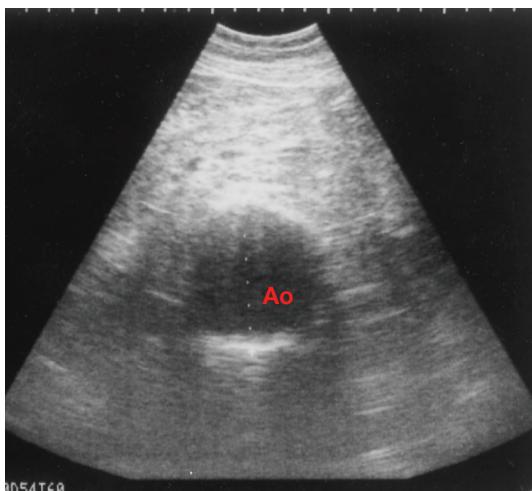
TABLE 2.1-18. Types of Valvular Heart Disease

Type	Etiology	History	Exam/Diagnosis	Treatment
Aortic stenosis	Most often seen in the elderly (senile calcific aortic stenosis) Unicuspid in childhood and adolescence. Rheumatic heart disease can predispose to AS	May be asymptomatic for years despite significant stenosis Once symptomatic, usually progresses from angina to syncope to CHF to death within 2 years Sx (also indications for valve replacements): ACS—Angina, CHF, Syncope	PE: Pulsus parvus et tardus (weak, delayed carotid upstroke) and a single or paradoxically split S2 sound; systolic crescendo-decrescendo murmur at the right second intercostal space radiating to the carotids Severe AS characterized by soft and single S2 Dx: Echocardiography	Aortic valve replacement (surgical or transcatheter methods)
Aortic regurgitation	Acute: Infective endocarditis, aortic dissection, chest trauma, MI Chronic: Valve malformations, rheumatic fever, connective tissue disorders (ie, Marfan syndrome), syphilis, inflammatory disorders	Acute: Rapid onset of pulmonary congestion, cardiogenic shock, and severe dyspnea Chronic: Slowly progressive onset of dyspnea on exertion, orthopnea, and PND. Uncomfortable heart pounding when lying on left side	PE: Early blowing diastolic murmur at the left sternal border, mid-diastolic rumble (Austin Flint murmur), and midsystolic apical murmur Widened pulse pressure causes de Musset sign (head bob with heartbeat), Corrigan sign (water-hammer pulse; wide and bounding), and Duroziez sign (femoral bruit) Dx: Echocardiography	Vasodilator therapy (dihydropyridines or ACEIs) for isolated aortic regurgitation until symptoms become severe enough to warrant valve replacement. Digoxin and diuretics have little benefit Monitor LV function and size
Mitral valve stenosis	The most common etiology continues to be rheumatic fever Uncommon in the US	Sx: Include dyspnea, orthopnea, PND, and hemoptysis. Unique features secondary to LAE include AF, dysphagia, and hoarseness	PE: Opening snap and mid-diastolic murmur at the apex; pulmonary edema Dx: Echocardiography	Antiarrhythmics (β -blockers, digoxin, or CCBs) and warfarin for AF. Mitral balloon valvotomy and valve replacement are effective for severe cases
Mitral valve regurgitation	Primarily 2° to rheumatic fever or chordae tendineae rupture after MI Myxomatous degeneration due to mitral valve prolapse Infective endocarditis	Patients present with dyspnea, orthopnea, PND, and fatigue	PE: Holosystolic/pansystolic murmur radiating to the axilla Dx: Echocardiography will demonstrate regurgitant flow; angiography can assess the severity of disease	ACEIs or ARBs to vasodilate and \downarrow rate of progression. Antiarrhythmics if necessary (AF is common with LAE). Digoxin and diuretics may be needed in CHF Valve repair or replacement for severe cases

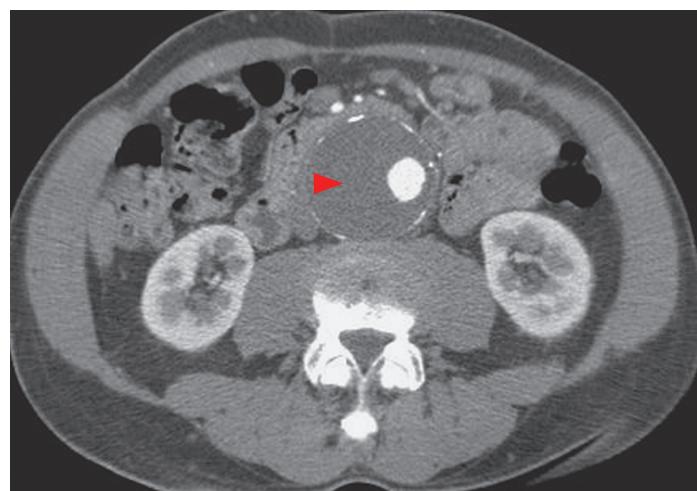
TREATMENT

- A**
- In asymptomatic patients, monitoring is appropriate for lesions < 5 cm.
 - Surgical correction is indicated if the lesion is ≥ 5.5 cm (abdominal), > 6 cm (thoracic), or smaller but rapidly enlarging (watch for bowel ischemia and infarction).
 - Emergent surgery for symptomatic or ruptured aneurysms.

A hyperaldosteronism workup with serum aldosterone and renin levels is an appropriate next diagnostic step.



A



B

FIGURE 2.1-17. Abdominal aortic aneurysm. (A) Ultrasound image of an AAA (Ao, aorta). (B) Transaxial image from a contrast-enhanced CT showing an aneurysm with extensive mural thrombus (arrowhead). (Image A reproduced with permission from Tintinalli JE et al. *Tintinalli's Emergency Medicine: A Comprehensive Study Guide*, 6th ed. New York, NY: McGraw-Hill; 2004. Image B reproduced with permission from Doherty GM. *Current Diagnosis & Treatment: Surgery*, 13th ed. New York, NY: McGraw-Hill; 2010.)

AORTIC DISSECTION

A transverse tear in the intima of a vessel that results in blood entering the media, creating a false lumen and leading to a hematoma that propagates longitudinally. Most commonly 2° to HTN, but also due to blunt chest trauma. The most common sites of origin are above the aortic valve and distal to the left subclavian artery. Most often occurs at 40–60 years of age, with a greater frequency in males than in females.

HISTORY/PE

- **Hx:** HTN, Marfan syndrome, mitral valve prolapse, trauma.
- **Presentation:** Sudden tearing/ripping pain in the anterior chest (ascending) with or without radiation to the back (descending), typically between the scapulae.
- **Exam:**
 - Patients are typically hypertensive. If hypotensive, consider pericardial tamponade, hypovolemia from blood loss, or other cardiopulmonary etiologies.
 - Asymmetric pulses and BP measurements or acute limb ischemia.
 - A murmur of aortic regurgitation may be heard if the aortic valve is involved with a proximal dissection.
 - Neurologic deficits, such as paraplegia, may be seen if the aortic arch or spinal arteries are involved.
 - Anuria may be seen if renal arteries are involved.
 - Signs of pericarditis or pericardial tamponade may be seen.

DIAGNOSIS

- **Best initial test for hemodynamically stable patients:** CT angiography. MRA can be used if contrast CT is contraindicated.
- **Best initial test for hemodynamically unstable patients:** TEE. It may also be used to visualize details of the proximal aorta and coronary vessels and can also evaluate for pericardial effusion.

KEY FACT

Aortic aneurysm is most often associated with atherosclerosis, whereas aortic dissection is commonly linked to HTN.

Q

A 70-year-old man with HTN presents for a routine appointment. He quit smoking 20 years ago but has a 20-pack-year history. What screening, if any, is indicated?

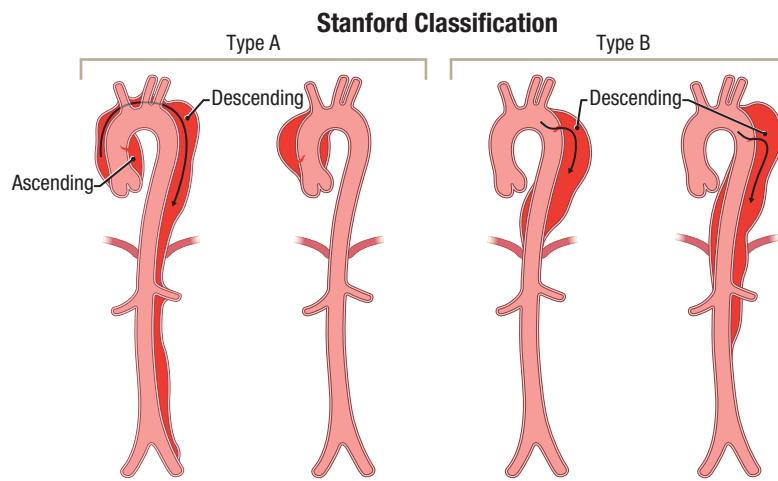


FIGURE 2.1-18. Stanford classification of aortic dissection. Type A involves the ascending aorta and may progress to involve the arch and thoracoabdominal aorta. Type B involves the descending thoracic or thoracoabdominal aorta distal to the left subclavian artery without involvement of the ascending aorta. (Reproduced with permission from USMLE-Rx.com.)

KEY FACT

Ascending aortic dissections are surgical emergencies; descending dissections are still emergencies but can often be treated medically.

- The Stanford system classifies any dissection proximal to the left subclavian artery as type A and all others as type B (see Figure 2.1-18).
- Type A (~70%) is the most common and involves the ascending aorta, irrespective of the site of the tear. Type B does not involve the ascending aorta.

TREATMENT

- **BP control:** Important to monitor and medically manage BP and heart rate as necessary. Avoid thrombolytics. Begin intravenous β -blockers (eg, IV labetalol) before starting vasodilators (nitroprusside) to prevent reflex tachycardia.
- All patients with type A thoracic dissection (ascending dissections) should have surgery.
- Patients with type B thoracic dissection (descending dissections) may be managed medically with BP and heart rate control; surgery is reserved if there is a leakage, rupture, or compromised organs.

KEY FACT

Virchow triad: (1) venous stasis, (2) trauma (endothelial damage), (3) hypercoagulability

DEEP VENOUS THROMBOSIS

Clot formation in the large veins of the extremities or pelvis. The classic Virchow triad of risk factors includes venous stasis (eg, from long-haul flights, prolonged bed rest, obesity, immobility, or incompetent venous valves in the lower extremities), endothelial trauma (eg, surgery, injury to the lower extremities, trauma), and hypercoagulable states (eg, thrombophilia, malignancy, pregnancy, OCP use).

HISTORY/PE

- Presents with unilateral lower extremity pain and swelling. Calf warmth, tenderness, and erythema may be present.
- Homans sign is calf tenderness with passive foot dorsiflexion (poor sensitivity and specificity for DVT).
- Use pretest clinical probability scoring for DVT, the Wells score.

A

The United States Preventive Services Task Force (USPSTF) guidelines recommend one-time screening for AAA by ultrasound in men 65–75 years of age who have ever smoked.

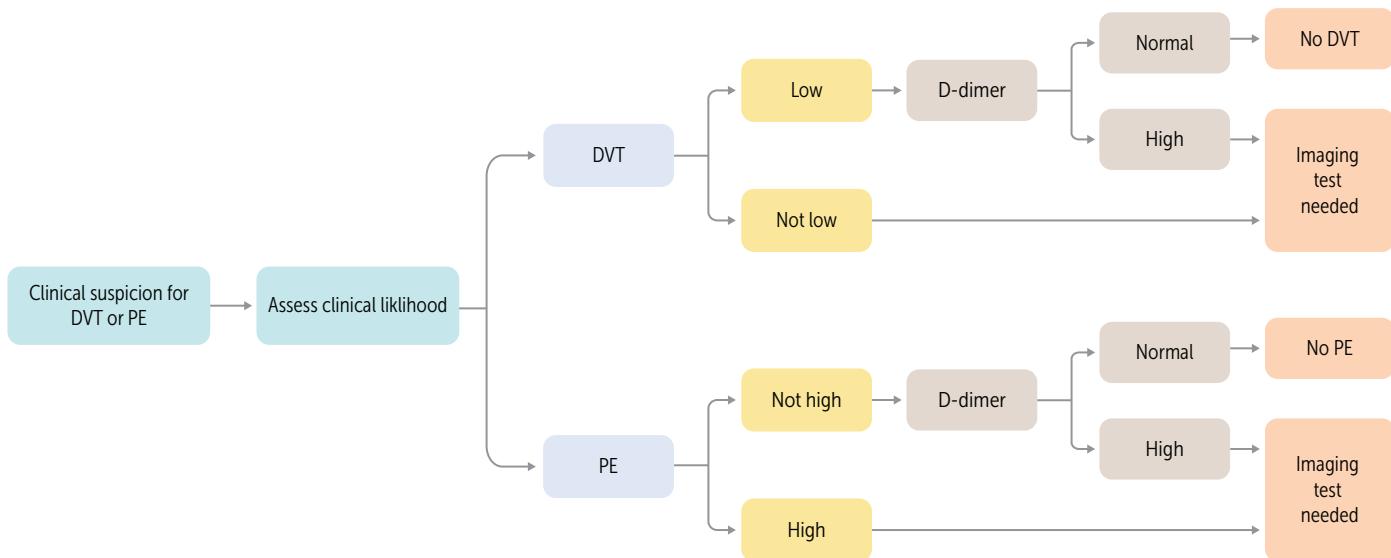


FIGURE 2.1-19. Algorithm for diagnostic imaging of deep vein thrombosis and pulmonary embolism. (Reproduced with permission from USMLE-Rx.com)

DIAGNOSIS

- D-dimer test should be ordered (sensitive but not specific) and is elevated in DVT. Also elevated in infections, malignancy, pregnancy, and postoperatively. A negative result, combined with a low clinical probability is sufficient to exclude a DVT.
- If D-dimer is elevated and the patient has a high-to-intermediate clinical probability, Doppler ultrasound is done.
- A spiral CT or V/Q scan may be used to evaluate for PE (see Figure 2.1-19).

KEY FACT

A ⊖ D-dimer test can be used to rule out the possibility of PE in low-risk patients.

TREATMENT

- Anticoagulate with subcutaneous low-molecular-weight heparin (LMWH) or IV unfractionated heparin followed by PO warfarin or NOACs for a total of 3–6 months.
- In patients with contraindications for anticoagulation, inferior vena cava filters should be placed.
- Hospitalized patients should receive DVT prophylaxis consisting of exercise as tolerated, antithromboembolic stockings, and subcutaneous LMWH or unfractionated heparin.

PERIPHERAL ARTERIAL DISEASE

Defined as a restriction of the blood supply to the extremities by atherosclerotic plaque. The lower extremities are most commonly affected. Clinical manifestations depend on the vessels involved, the extent and rate of obstruction, and the presence of collateral blood flow.

HISTORY/PE

- Presents with intermittent claudication; reproducible cramping pain in the calf, thigh, or buttock after walking for a certain distance (claudication distance) and is relieved with rest.
- As the disease progresses, it causes critical limb ischemia. Pain occurs at rest and affects the distal extremities. Dorsal foot ulcerations may develop 2° to poor perfusion. A painful, cold, numb foot is characteristic of critical limb ischemia.

**MNEMONIC****The 6 P's of acute ischemia—**

Pain
Pallor
Paralysis
Pulse deficit
Paresthesias
Poikilothermia

KEY FACT

$$\text{ABI} = \frac{P_{\text{leg}}}{P_{\text{arm}}}$$

Rest pain seen with an ABI < 0.4.

(normal ABI: 1.0–1.2)

KEY FACT

Calf claudication = femoral disease

Buttock claudication = iliac disease

Buttock claudication + impotence =

Leriche syndrome (aortoiliac occlusive disease)

KEY FACT

The major cause of mortality in patients with PAD is cardiovascular disease (MI, stroke); there is a 20–30% risk for these complications. There is only a 1–2% risk for developing limb ischemia.

- For more proximal lesions, there will be claudication and weak pulses below the area of occlusion (ie, aortoiliac disease; [Leriche syndrome] is characterized by the triad of hip, thigh, and buttock claudication, impotence, and symmetric atrophy of bilateral lower extremities).
- A Buerger angle of < 20 degrees and capillary filling of > 15 seconds are seen in severe ischemia.
- **Acute ischemia:**
 - May be due to thrombosis in situ (~40), emboli usually of cardiac origin (~38%), graft/angioplasty occlusion (~15%), or trauma. Acute occlusions commonly occur at bifurcations distal to the last palpable pulse (see mnemonic for signs and symptoms).
 - May also be 2° to cholesterol atheroembolism (“blue toe syndrome”), which is characterized by blue toes, livedo reticularis, renal failure (often 2° to catheterization).
- **Chronic ischemia:** Lack of blood perfusion leads to muscle atrophy, pallor, loss of sweat and sebaceous glands, cyanosis, hair loss, and gangrene/necrosis.

DIAGNOSIS

- Identify cardiovascular risk factors, especially smoking, diabetes, hypertension, and hyperlipidemia.
- **Best initial test:** Ankle-brachial index test; can provide objective evidence of atherosclerosis (rest pain usually occurs with an ABI < 0.4). A very high ABI can indicate calcification of the arteries.
- **Doppler ultrasound:** Identifies stenosis and occlusion. Normal ankle Doppler readings are > 90% of brachial readings.
- **Most accurate test:** Angiography; often not necessary unless revascularization is indicated.

TREATMENT

- Treat acute symptomatic ischemia with heparin and prompt revascularization.
- **Address modifiable risk factors:** Smoking (vital), hypertension, hyperlipidemia, and diabetes.
- Educate regarding careful hygiene and foot care. Exercise helps develop collateral circulation.
- Antiplatelet agents (ASA or vorapaxar) do not consistently reduce symptoms, but ↓ the risk for associated cardiovascular mortality.
- Cilostazol is effective medication in intermittent claudication.
- Surgery (arterial bypass), percutaneous transluminal angioplasty, and stenting, or amputation can be employed when conservative treatment fails or in acute limb ischemia.

LYMPHEDEMA

A disruption of the lymphatic circulation that results in peripheral edema and chronic infection of the extremities. Primary (or congenital) lymphedema is rare. Most often caused secondarily by surgeries involving lymph node dissection or, in developing countries, parasitic infections.

HISTORY/PE

History will differ by cause. Examples include the following:

- Postmastectomy patients present with unexplained swelling of the upper extremity (secondary to surgery).

- Patients originating from developing countries present with progressive swelling of the lower extremities bilaterally with no cardiac abnormalities (ie, filariasis infection).
- Children present with progressive, bilateral swelling of the extremities (1°).
- Patients with Turner syndrome will have lymphatic edema.

DIAGNOSIS

Diagnosis is clinical. Rule out other causes of edema, such as cardiac and metabolic disorders.

TREATMENT

- Directed at symptom management, including exercise, massage therapy, and pressure garments to mobilize and limit fluid accumulation.
- Diuretics are ineffective and relatively contraindicated.
- Maintain vigilance for cellulitis with prompt gram- \oplus antibiotic coverage for infection.

Syncope

A sudden, temporary loss of consciousness and postural tone 2° to cerebral hypoperfusion. Etiologies can be cardiac, neurologic, or other.

- **Cardiac:** Valvular lesions (aortic stenosis), arrhythmias, PE, cardiac tamponade, aortic dissection.
- **Neurologic:** Subarachnoid hemorrhage.
- **Other:** Orthostatic/hypovolemic hypotension, metabolic abnormalities, neurocardiogenic syndromes (eg, vasovagal/micturition syncope), psychiatric, medications.

HISTORY/PE

- Age, triggers, prodromal symptoms, and associated symptoms should be investigated.
- Syncope can be confused with seizures. Unlike syncope, seizures may be characterized by a preceding aura, tonic-clonic activity, tongue-biting, bladder and bowel incontinence, and a postictal phase.
- Cardiac causes of syncope are typically associated with very brief or absent prodromal symptoms, a history of exertion, lack of association with changes in position, and/or a history of cardiac disease.
- Neurocardiogenic syndrome is common in younger patients and older patients with difficulty voiding.

DIAGNOSIS

Depending on the suspected etiology:

- **Cardiac:** ECG, Holter monitors or 2-week event recorders (arrhythmias), echocardiograms (structural abnormalities), stress tests (ischemia).
- **Neurologic:** CT of head (ischemia or hemorrhage) and EEG (seizure).
- **Other:** Orthostatic BP readings (hypovolemia, autonomic dysfunction), glucose (hypoglycemia), and tilt-table testing (neurally mediated syncope).

TREATMENT

Tailored to the etiology.

KEY FACT

Cardiac syncope is associated with 1-year sudden cardiac death rates of up to 40%.