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TABLE 2.1-3. **Bradyarrhythmias and Conduction Abnormalities (continued)**

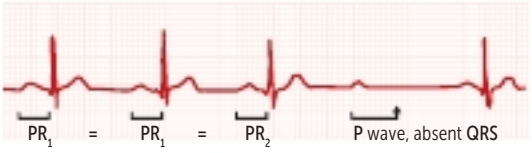
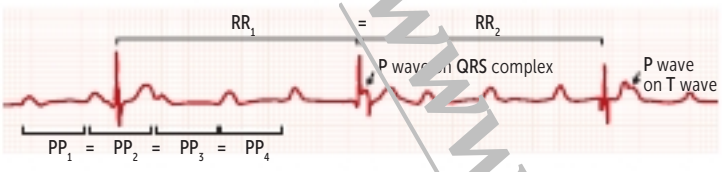
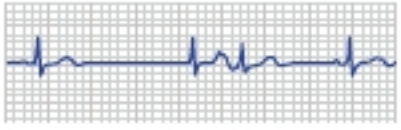
TYPE/ETIOLOGY	SIGNS/SYMPTOMS	TREATMENT
Second-degree AV block (Mobitz type II)  	**ECG findings:** Unexpected dropped beats without a change in PR interval	
Results from fibrotic disease of the conduction system or from acute, subacute, or prior MI Suggests intrinsic disease of His Purkinje system	Occasionally syncope; frequent progression to third-degree AV block	Pacemaker placement (even if asymptomatic)
Third-degree AV block (complete)  	**ECG findings:** P and QRS waves occur regularly but at different rates (different PP and RR intervals shown in the figure; ie, atrial contraction is dissociated from ventricular contraction). Note: Some P waves are not visible or are partially visible due to fusion with QRS complex	
No electrical communication between the atria and ventricles Suggests disease of His Purkinje system	Syncope, dizziness, acute heart failure, hypotension, cannon A waves	Pacemaker placement
Sick sinus syndrome/tachycardia-bradycardia syndrome 	**ECG findings:** ECG shows an SA pause (no P waves generated, suggesting no activation at the SA node), followed by a junctional escape beat (QRS with no preceding P wave), and then reappearance of P waves (resumption of SA node activity). Other supraventricular tachyarrhythmias and bradyarrhythmias may occur intermittently in sick sinus syndrome (see ECGs earlier)	
Heterogeneous disorder that leads to intermittent supraventricular tachyarrhythmias and bradyarrhythmias	Secondary to tachycardia or bradycardia; AF and thromboembolism may occur → syncope, palpitations, dyspnea, chest pain, transient ischemic attack (TIA), and/or stroke	Most common indication for pacemaker placement Anticoagulate in AF/flutter to prevent systemic emboli

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TABLE 2.1-4. Supraventricular Tachyarrhythmias

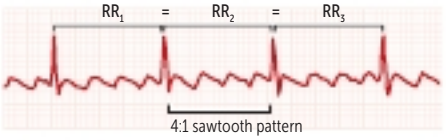
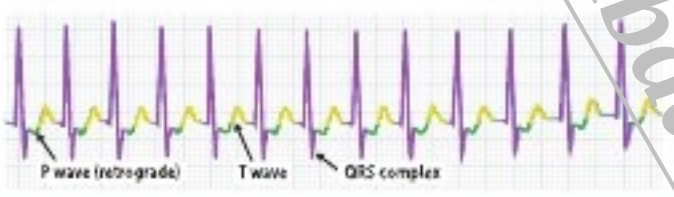
TYPE/ETIOLOGY	SIGNS/SYMPTOMS	TREATMENT
Sinus tachycardia	**ECG findings:** Sinus rhythm, ventricular rate >100 bpm	
Normal physiologic response to fear, pain, and exercise Can also be secondary to hyperthyroidism, volume contraction, infection, or pulmonary embolism (PE)	Palpitations, shortness of breath	Treat the underlying cause
Atrial flutter 	**ECG findings:** Regular rhythm; "sawtooth" appearance of P waves; atrial rate is usually 240–320 bpm, ventricular rate depends on conduction block through AV node (in example, atrial rate 300 bpm, ventricular rate 60 bpm)	
Circular movement of electrical activity around the atrium at a rate of approximately 300 times per minute. Reentrant circuit most commonly passes between inferior vena cava and tricuspid annulus (cavotricuspid isthmus). Interventions to ablate the cavotricuspid isthmus may break the reentrant circuit	Usually asymptomatic but can present with palpitations, syncope, and lightheadedness	Anticoagulation, rate control, and cardioversion guidelines as in atrial fibrillation (see earlier)
Atrioventricular nodal reentry tachycardia (AVNRT) 	**ECG findings:** HR about 150 bpm, with retrograde P waves. Note no P waves before the QRS complex	
A reentry circuit in the AV node depolarizes the atrium and ventricle nearly simultaneously	Palpitations, shortness of breath, angina, syncope, lightheadedness. AVRT and AVNRT are often indistinguishable on ECG. P waves may occur during or after QRS. These P waves may appear as a pseudo R' in V_1, or pseudo S in inferior leads (II, III, aVF), a finding that supports AVNRT over AVRT	Cardiovert if hemodynamically unstable If stable, initial trial of vagal maneuvers (eg, Valsalva, carotid sinus massage, [CSM], ice immersion), followed by adenosine if ineffective CSM contraindicated in MI/TIA/stroke in previous 3 months, carotid stenosis/atheroma, ventricular fibrillation (VF)/ventricular tachycardia (VT), or previous adverse reaction to CSM
Atrioventricular reentrant tachycardia (AVRT)	**ECG findings:** Patient's baseline ECG may show preexcitation (see WPW syndrome later). During tachycardia, ECG is similar to AVNRT noted earlier. A retrograde P wave is often seen on the ST segment or T wave.	

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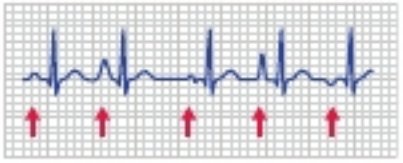
TABLE 2.1-4. **Supraventricular Tachyarrhythmias (continued)**

TYPE/ETIOLOGY	SIGNS/SYMPTOMS	TREATMENT
Multifocal atrial tachycardia 	**ECG findings:** Three or more unique P-wave morphologies are visible *(red arrows)*; rate >100 bpm	
Multiple atrial pacemakers or reentrant pathways; associated with many cardiopulmonary conditions, eg, chronic obstructive pulmonary disease (COPD), hypoxemia, CHF	May be asymptomatic. At least three different P-wave morphologies	Treatment of underlying condition is first step Consider intravenous (IV) non-dihydropyridine CCBs and β-blockers for acute management If recurrent and symptomatic, oral non-dihydropyridine CCBs and β-blockers chronically (unless contraindicated)

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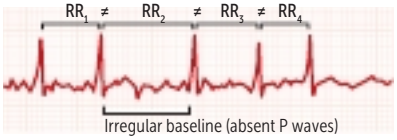


FIGURE 2.1-10. **ECG findings in atrial fibrillation.** No discernible P waves, with variable and irregular QRS response (RR interval varies irregularly). (Reproduced with permission from USMLE-Rx.com.)

History/PE

- Presentation ranges from asymptomatic to hemodynamically unstable. May have palpitations, fatigue, and dyspnea. Sometimes chest discomfort or even syncope.
- Importantly, can present with thromboembolic complications initially (eg, stroke, mesenteric).
- PE: Irregular pulse, irregular jugular venous pulsations.

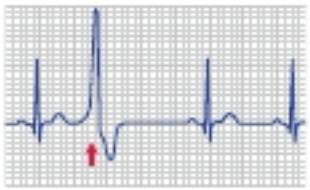
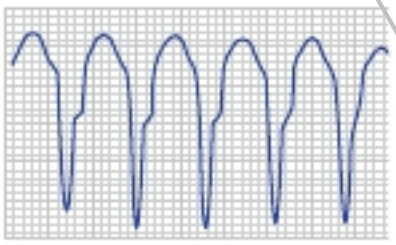
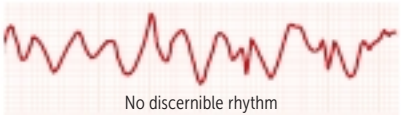
Investigations

- **12-lead ECG:** Diagnosis confirmed on ECG (Fig. 2.1-10).
- **Labs:** Electrolytes, complete blood count (CBC), and thyroid-stimulating hormone (TSH; hyperthyroidism should always be considered).
- **Transthoracic echocardiography (TTE):** To identify structural issues (eg, atrial size, valve disease).

Treatment

Patients with AF may require anticoagulation to prevent thromboembolism. Also, either a rate control or rhythm control strategy may be implemented to manage the arrythmia (see p. 32).

TABLE 2.1-5. **Ventricular Tachyarrhythmias**

TYPE/ETIOLOGY	SIGNS/SYMPTOMS	TREATMENT
Premature ventricular contraction (PVC) 	**ECG findings:** Early, wide QRS *(red arrow)* not preceded by a P wave; PVCs are usually followed by a compensatory pause	
Ectopic beats arise from ventricular foci. Associated with hypoxia, fibrosis, ↓ LV function, electrolyte abnormalities, and hyperthyroidism, but may be a normal finding	Usually asymptomatic, but may lead to palpitations	Treat the underlying cause. Decrease caffeine and alcohol consumption. If symptomatic, give β-blockers or, occasionally, other antiarrhythmics
Ventricular tachycardia (VT) 	**ECG findings:** Wide QRS complexes in a regular rapid rhythm; may see AV dissociation (P wave not seen in this example)	
Can be associated with coronary artery disease (CAD), MI, and structural heart disease	Three or more consecutive PVCs Nonsustained VT (lasts <30 seconds) often asymptomatic; sustained VT (lasts >30 seconds) can lead to palpitations, hypotension, angina, and syncope Can progress to VF and death	Synchronized cardioversion if hemodynamically unstable Defibrillation if pulseless VT Antiarrhythmics (eg, amiodarone, lidocaine, procainamide) if stable
Ventricular fibrillation (VF) 	**ECG findings:** Totally erratic wide-complex tracing	
Associated with CAD and structural heart disease Also associated with cardiac arrest (together with asystole)	Syncope, absence of BP, no pulse	Immediate electrical defibrillation and advanced cardiac life support (ACLS) protocol

(continues)

Q

A college-aged man passes out without any inciting factors and has no prodromal symptoms or signs of seizure. After recovery, his cardiac exam is unremarkable, and an ECG shows a slurred upstroke of the QRS. What are the next best steps?

Etiology

- HFrEF (aka systolic HF) is caused by compensatory mechanisms (sympathetic nervous system [SNS] and renin-angiotensin-aldosterone system [RAAS] activation) to inciting conditions (eg, valvular disease, HTN) that may be acutely beneficial but may become maladaptive chronically (Fig. 2.1-11).
- Chronic activation of the SNS and RAAS results in cardiac and vascular remodeling (eg, hypertrophy, fibrosis, vasoconstriction), as well as sodium and water retention. Activation of the SNS leads to increased afterload (vasoconstriction/hypertension), whereas activation of the RAAS results in increased preload (salt and water retention).

KEY FACT

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

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: Weight gain, bilateral pulmonary rales, increased JVP, positive hepatojugular reflex, peripheral edema, elevated and sustained LV impulse, and an S_3 gallop.

KEY FACT

Hyponatremia parallels the severity of HF and is an independent predictor of mortality in these patients.

Diagnosis

- HFrEF presents with the clinical syndrome of HF, with typical signs and symptoms, in addition to reduced EF(<40%).
- Studies that may support the diagnosis include the following:
  - **Best initial test:** Echocardiogram (transthoracic echocardiogram). ↓ EF helps establish HFrEF; structural abnormalities may help identify cause (eg, AF, old MI, or LVH).
  - **ECG:** May show MI, heart block, arrhythmia, or other diagnostic clues.

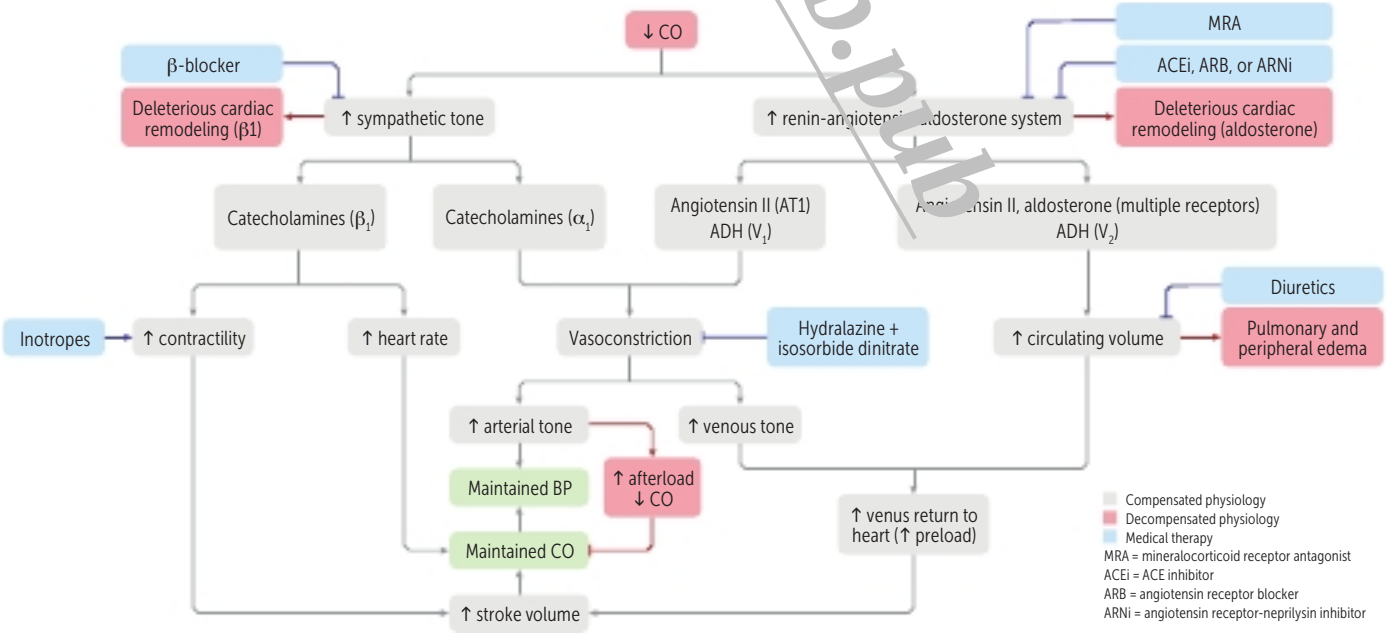


FIGURE 2.1-11. **Pathophysiology of heart failure.** Activation of the RAAS and SNS may initially help the failing heart adapt by increasing contractility, heart rate, and circulating volume. However, deleterious cardiac remodeling may lead to worsening HF and pulmonary edema over time *(red boxes)*. Drugs that target the various maladaptive processes are shown *(blue boxes)*. (Reproduced with permission from USMLE-Rx.com.)

- **CXR**: May show cardiomegaly, cephalization of pulmonary vessels, pleural effusions, vascular congestion, pulmonary edema, and prominent hila (Fig. 2.1-12).
- **Lab abnormalities**: Brain natriuretic peptide >500 pg/mL, ↓ CBC (anemia), ↑ creatinine (sometimes), ↓ sodium in later stages, ↑ or ↓ TSH/T_4 levels.

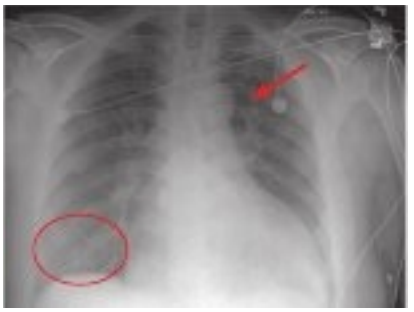


FIGURE 2.1-12. X-ray of the chest (CXR) with evidence of congestive heart failure. Frontal CXR demonstrates marked cardiomegaly, cephalization of vessels *(arrow)*, interstitial edema *(circle)*, and small left-sided pleural effusion, which raise concern for CHF. (Reproduced with permission from Tintinalli JE et al. *Tintinalli's Emergency Medicine: A Comprehensive Study Guide.* 7th ed. New York, NY: McGraw-Hill; 2011.)

Treatment

Acute congestive heart failure:

- The first step in management is clinical identification of the hemodynamic profile. Specifically, the level of congestion ("wet" vs "dry") and perfusion ("warm" vs "cold") must be evaluated. Treatment is determined based on this evaluation, as illustrated in Table 2.1-11.

TABLE 2.1-11. Hemodynamic Profiles in Heart Failure

	WET (CONGESTED)	DRY (NOT CONGESTED)
WARM (Adequate Perfusion)	**Wet and Warm** Congested, adequate perfusion ■ **Rx:** ■ Initial diuretics and vasodilators ■ Ultrafiltration if refractory	**Dry and Warm** Not congested, adequate perfusion ■ **Rx:** ■ Optimize oral therapy
COLD (Hypoperfusion)	**Wet and Cold** Congested, hypoperfusion ■ **Rx if hypotensive (systolic blood pressure [SBP] <90 mm Hg):** ■ Ionotropic agent initially; vasopressor if refractory ■ Diuretic *after* perfusion is corrected ■ Circulatory support/renal replacement therapy (RRT) if unresponsive to medication ■ **Rx if NOT hypotensive (SBP >90 mm Hg):** ■ Initial diuretics and vasodilators ■ Inotropic agents if refractory	**Dry and Cold** Not congested (hypovolemic), hypoperfusion ■ **Rx:** ■ Consider initial fluid challenge ■ Inotropic agents if still hypoperfused

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)

KEY FACT

Acute CHF management

- Upright positioning
- Vasodilators
- Diuretics
- Inotropes
- Oxygen if hypoxic
- Noninvasive positive-pressure ventilation
- Mechanical support