

Arrhythmias

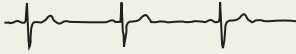




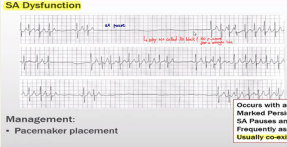
MNEMONIC

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 <i>acceptable in athletes</i>	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 lightheadedness, syncope, chest pain, or hypotension 	Sinus rhythm Ventricular rate < 60 bpm <i>narrow QRS</i> <i>Regular</i> <i>Slow HR</i> <i>Normal PR interval (3-5 small boxes)</i>	None if asymptomatic and <i>symptomatic:</i> rate > 40 bpm; atropine may be used to ↑ heart rate <i>2. B. Agonist · Dopamine (iso-proterenol)</i> 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 <i>0.2 sec = &gt; 1 Large Square</i> <i>No Dropped QRS</i>	<i>Same as Sinus Brady cardia.</i>
Second-degree AV block (Mobitz type I/ Wenckebach)	Drug effects (digoxin, β-blockers, CCBs) or ↑ vagal tone; right coronary ischemia or infarction	Usually asymptomatic 	<i>Progressive</i> 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 <i>Fixed Prolonged PR interval</i>	Pacemaker placement (even if asymptomatic)
Third-degree AV block (complete)	No electrical communication between the atria and ventricles	Syncope, dizziness, acute heart failure, hypotension, <i>cannon A waves</i> 	<i>P &gt; QRS</i> No relationship between P waves and QRS complexes <i>Complete Dissociation</i> <i>QRS &lt; Narrow -&gt; if from the Sinoatrial node of the heart</i> <i>Wide -&gt; if from the Ventricle</i>	Pacemaker placement <i>Medical Emergency</i>
Sick sinus syndrome/ <i>tachycardia-bradycardia syndrome</i>	Heterogeneous disorder that leads to intermittent supraventricular tachyarrhythmias and bradyarrhythmias <i>SNT</i> <i>in advanced Age</i>	2° to tachycardia or bradycardia; AF and thromboembolism may occur → syncope, palpitations, dyspnea, chest pain, TIA, and/or stroke	<i>SA Dysfunction</i>  Management: • Pacemaker placement <i>Tachy</i> <i>Brady</i> <i>Pause</i>	Most common <i>indication</i> for pacemaker placement Anticoagulate in atrial fibrillation/flutter to prevent systemic emboli

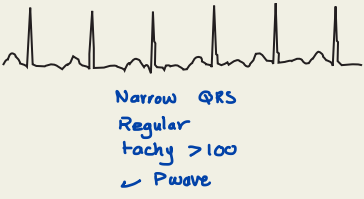
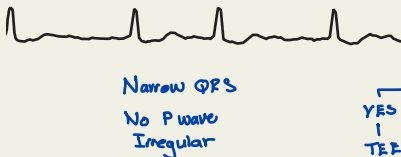
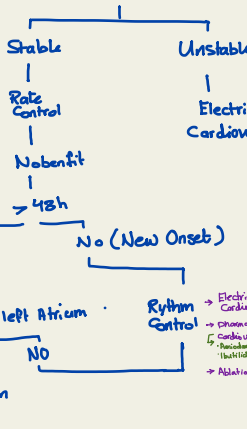
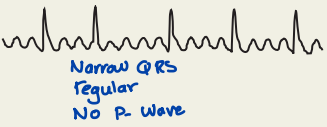
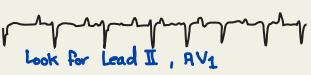
KEY FACT

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
<b>ATRIAL</b>				
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  Narrow QRS Regular tachy > 100 P wave	Sinus rhythm Ventricular rate > 100 bpm	Treat the underlying cause β Blocker Ibutilide Ablation (End Game)
Atrial fibrillation (AF)  <small>Paroxysmal &lt; 48h Persistent &gt; 7 days Long Persistent &gt; 4 year Permanent forever</small>	Acute AF— <b>PIRATES</b> : Pulmonary disease Ischemia Rheumatic heart disease Anemia/Atrial myxoma Thyrotoxicosis or Hypothyroid Ethanol Holiday Heart Syndrome Sepsis Chronic AF—HTN, CHF Most often caused by re-entry ← 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  Narrow QRS No P wave Irregular	No discernible P waves, with variable and irregular <sup>Narrow</sup> QRS response  	For chronic AF, initial therapy: Rate control with β-blockers, CCBs, or digoxin (BP not affected → Good for PT with low BP) Anticoagulate with warfarin or novel oral anticoagulant (NOAC) for patients with CHA <sub>2</sub> DS <sub>2</sub> -VASc score ≥ 2 (0 - Platelet Only, 1 - Female → 1 - For other Cause) 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 APib w/ mechanical Valve Severe mitral Stenosis LVAD [Warfarin is a must]
Atrial flutter  easy for Ablation	Circular movement of electrical activity <sup>macro circuit</sup> around the atrium at a rate of approximately 300 times per minute	Usually asymptomatic but can present with palpitations, syncope, and lightheadedness  Narrow QRS regular No P wave	Regular rhythm; "sawtooth" 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 Ablation is Favorable Here
Multifocal atrial tachycardia	Multiple atrial pacemakers or reentrant pathways; associated with COPD* hypoxemia	May be asymptomatic. At least three different P-wave morphologies  Look for Lead II, AV <sub>1</sub>	Three or more unique P-wave morphologies; rate > 100 bpm Irregular } Like AF Narrow QRS } But there is P waves	Treat as AF but avoid β-blockers because of chronic lung disease (if present) Oxygenation (Breathe ONE) → Avoid electrical Cardioversion even if Unstable

(continues)

TABLE 2.1-4. Supraventricular Tachyarrhythmias (continued)

TYPE	ETIOLOGY	SIGNS/SYMPTOMS	ECG FINDINGS	TREATMENT
<b>ATRIOVENTRICULAR JUNCTION</b>				
<p><i>Short RP interval</i></p> <p><b>Narrow QRS</b> No P wave Regular</p> <p>→ Saw-tooth Absent → SVT</p> 	<p>A reentry circuit in the AV node depolarizes the atrium and ventricle nearly simultaneously</p> <p>(AVNRT)</p>	<p>Palpitations, shortness of breath, angina, syncope, lightheadedness</p>	<p>Rate 150–250 bpm; P wave is often buried in QRS or shortly after</p> <p><i>Preventative therapy:</i></p> <ol style="list-style-type: none"> <li>1. cCB, BB</li> <li>2. Ablation (if not worked)</li> </ol>	<p>Cardiovert if hemodynamically unstable. <i>Acute:</i> <b>Vagal maneuvers</b> (eg, carotid massage, Valsalva, ice immersion (dive reflex)).</p> <p><i>I-V 6 mg:</i> <b>Adenosine</b> if vagal maneuver fails, cCB, BB</p>
<p><i>Start in accessory Pathway</i></p> <p><b>Antidromic</b> → <b>Wide QRS - regular - no P wave</b></p> 	<p>Atrioventricular reentrant tachycardia (AVRT)</p> <p>Seen in WPW</p> <p>→ <b>Orthodromic</b> → <b>Narrow Complex - Regular - no P wave</b></p>	<p>Palpitations, shortness of breath, angina, syncope, lightheadedness</p>	<p><i>antidromic:</i> - A <b>retrograde P wave</b> is often seen after a normal QRS</p>	<p>Except for WPW, <b>same as</b> that for AVNRT</p> <p>WPW listed separately below</p>
<p><i>Start in Normal Pathway</i></p> <p><b>Orthodromic</b> → <b>Narrow Complex - Regular - no P wave</b></p> 	<p>Seen in WPW</p> <p>→ <b>Orthodromic</b> → <b>Narrow Complex - Regular - no P wave</b></p>	<p>Palpitations, dyspnea, dizziness, and rarely cardiac death</p>	<p>Characteristic <b>delta wave</b> with <b>widened QRS complex</b> and <b>shortened PR interval</b> (see Figure 2.1-8)</p> <p><i>Regular Wide QRS No P wave Delta Wave</i></p>	<p>Observation for asymptomatics</p> <p><b>Acute therapy is procainamide</b> or amiodarone</p> <p><i>acid</i> <b>SVT gets worse after CCBs, BB or digoxin</b> (dangerous in WPW). Radiofrequency catheter ablation is curative → <b>Preventive</b></p> <p><i>2. Cardio-version</i></p>
<p><i>Part Start with Normal and Part Start with accessory</i></p> <p><b>Preexcitation + SVT = WPW</b></p> 	<p>Wolff-Parkinson-White (WPW) syndrome</p> <p>Abnormal fast accessory conduction pathway from atria to ventricle (<b>Bundle of Kent</b>)</p>	<p>Palpitations, shortness of breath, angina, syncope, lightheadedness</p>	<p>Rate &gt; 100 bpm; P wave with an unusual axis before each normal QRS</p>	<p>Adenosine can be used to unmask underlying atrial activity by slowing down the rate</p>

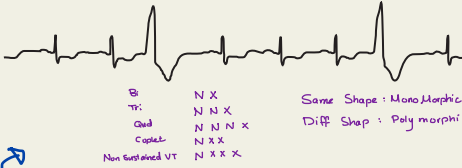



**KEY FACT**

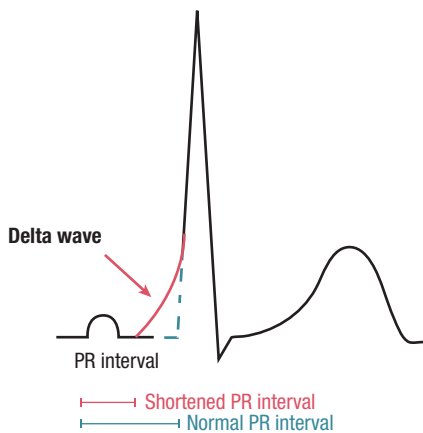
Use the **CHA<sub>2</sub>DS<sub>2</sub>-VASc** scoring system to estimate **stroke risk** in **atrial fibrillation**, and anticoagulate with NOAC (eg, dabigatran, rivaroxaban, apixaban, and edoxaban) or warfarin (used with metal valves or mitral stenosis) for a score of 2 or more:

- 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).

# CARDIOVASCULAR

**TABLE 2.1-5. Ventricular Tachyarrhythmias**

TYPE	ETIOLOGY	SIGNS/SYMPTOMS	ECG FINDINGS	TREATMENT
Premature ventricular contraction (PVC) <i>↳ normal rate</i>	Ectopic beats arise from ventricular foci. Associated with hypoxia, fibrosis, ↓ LV function, electrolyte abnormalities, and hyperthyroidism	Usually asymptomatic but may lead to palpitations 	Early, <b>wide QRS</b> not preceded by a P wave PVCs are usually followed by a compensatory pause	Treat the underlying cause If <b>symptomatic</b> , give β-blockers or, occasionally, other antiarrhythmics
Ventricular tachycardia (VT)	Can be associated with CAD, MI, and structural heart disease <i>Long QT syndrome</i> <i>Drug toxicity (macrolid, fluoroquinolones)</i>	<b>Nonsustained VT (lasts &lt; 30 seconds)</b> is often asymptomatic; <b>sustained VT (lasts &gt; 30 seconds)</b> can lead to palpitations, hypotension, 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	<b>Cardioversion</b> if <b>Acute</b> : <b>unstable</b> . Antiarrhythmics (eg, <b>IV amiodarone</b> , lidocaine, procainamide) if stable <i>Remove + treat the cause</i> <b>Preventive: ICD, EPS</b>
Ventricular fibrillation (VF)	Associated with CAD and structural heart disease Also associated with <b>cardiac arrest</b> (together with <b>asystole</b> )	Syncope, absence of BP, no pulse 	Totally erratic wide-complex tracing <i>Wide QRS</i> <i>Irregular</i> <i>No P wave</i>	Immediate electrical defibrillation and ACLS protocol <i>not awake</i> <i>awake → make sure not artifact</i>
Torsades de pointes	Associated with <b>long QT syndrome</b> , proarrhythmic response to <b>medications</b> , <b>hypokalemia</b> , congenital deafness, and <b>alcoholism</b>	Can present with sudden cardiac death; typically associated with palpitations, dizziness, and syncope 	<b>Polymorphous</b> QRS; VT with rates between 150 and 250 bpm <i>Wide QRS</i> <i>Regular</i> <i>No P wave</i> <i>Polymorphic</i>	<b>Give magnesium</b> initially and <b>car-diovert</b> if unstable <b>Correct hypokalemia</b> ; 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.)