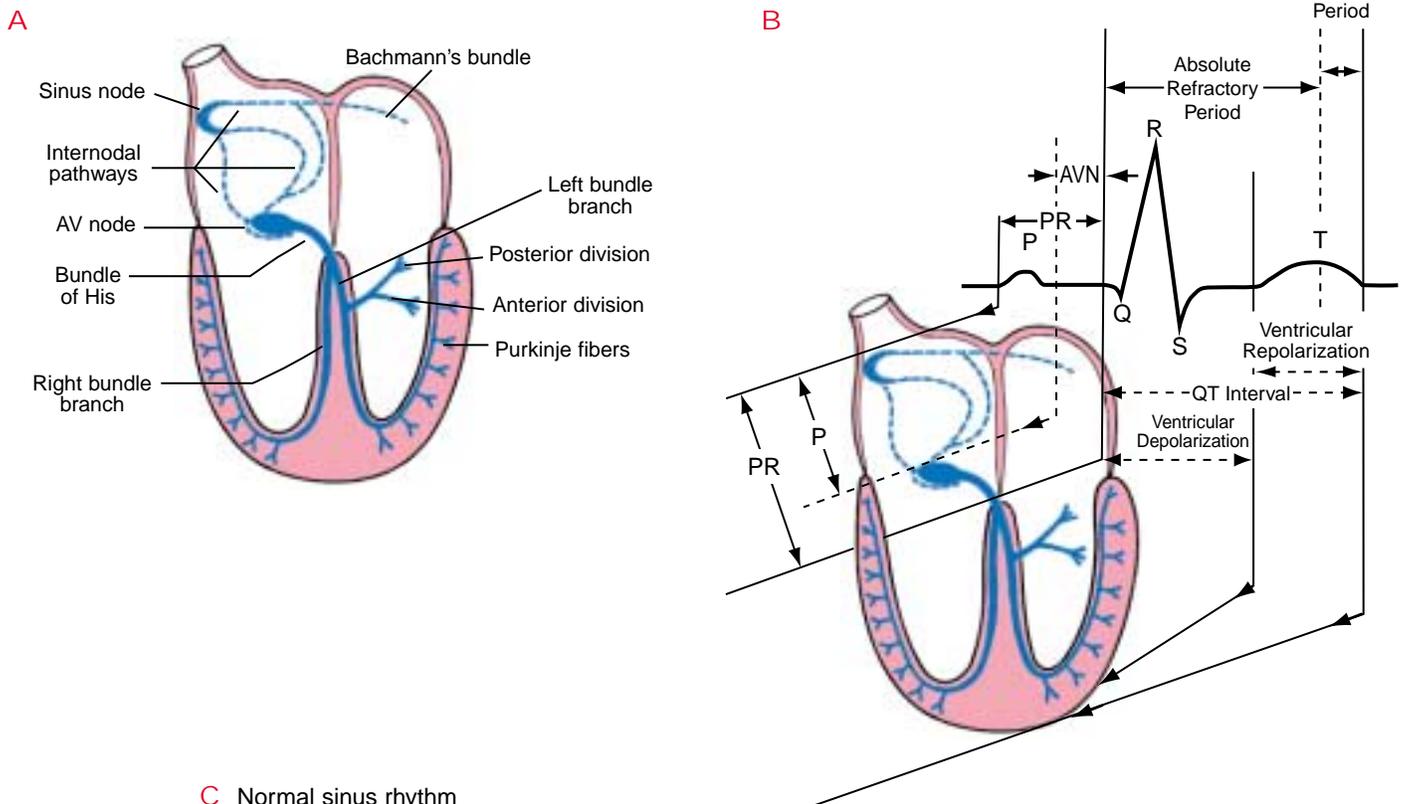


## ACLS Rhythms for the ACLS Algorithms

### The Basics

1. Anatomy of the cardiac conduction system: relationship to the ECG cardiac cycle. **A**, Heart: anatomy of conduction system. **B**, P-QRS-T complex: lines to conduction system. **C**, Normal sinus rhythm.

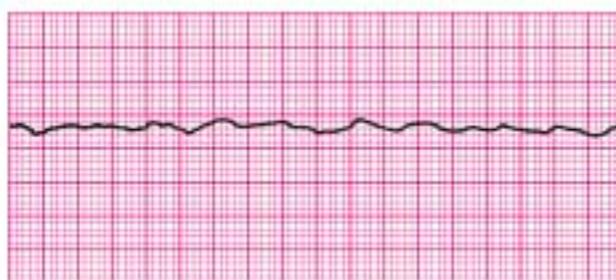


## The Cardiac Arrest Rhythms

2. Ventricular Fibrillation/Pulseless Ventricular Tachycardia	
<b>Pathophysiology</b>	<ul style="list-style-type: none"> <li>■ Ventricles consist of areas of normal myocardium alternating with areas of ischemic, injured, or infarcted myocardium, leading to chaotic pattern of ventricular depolarization</li> </ul>
<b>Defining Criteria per ECG</b>	<ul style="list-style-type: none"> <li>■ <b>Rate/QRS complex:</b> unable to determine; no recognizable P, QRS, or T waves</li> <li>■ <b>Rhythm:</b> indeterminate; pattern of sharp up (peak) and down (trough) deflections</li> <li>■ <b>Amplitude:</b> measured from peak-to-trough; often used subjectively to describe VF as <i>fine</i> (peak-to-trough 2 to &lt;5 mm), <i>medium-moderate</i> (5 to &lt;10 mm), <i>coarse</i> (10 to &lt;15 mm), <i>very coarse</i> (&gt;15 mm)</li> </ul>
<b>Clinical Manifestations</b>	<ul style="list-style-type: none"> <li>■ Pulse disappears with onset of VF</li> <li>■ Collapse, unconsciousness</li> <li>■ Agonal breaths → apnea in &lt;5 min</li> <li>■ Onset of <i>reversible death</i></li> </ul>
<b>Common Etiologies</b>	<ul style="list-style-type: none"> <li>■ Acute coronary syndromes leading to ischemic areas of myocardium</li> <li>■ Stable-to-unstable VT, untreated</li> <li>■ PVCs with R-on-T phenomenon</li> <li>■ Multiple drug, electrolyte, or acid-base abnormalities that prolong the relative refractory period</li> <li>■ Primary or secondary QT prolongation</li> <li>■ Electrocutation, hypoxia, many others</li> </ul>
<b>Recommended Therapy</b> <i>Comprehensive ECC algorithm, page 10; VF/pulseless VT algorithm, page 77</i>	<ul style="list-style-type: none"> <li>■ Early defibrillation is essential</li> <li>■ Agents given to prolong period of reversible death (<i>oxygen, CPR, intubation, epinephrine, vasopressin</i>)</li> <li>■ Agents given to prevent refrillation after a shock causes defibrillation (<i>lidocaine, amiodarone, procainamide, <math>\beta</math>-blockers</i>)</li> <li>■ Agents given to adjust metabolic milieu (<i>sodium bicarbonate, magnesium</i>)</li> </ul>



Coarse VF



Fine VF

3. PEA (Pulseless Electrical Activity)			
<b>Pathophysiology</b>	<ul style="list-style-type: none"> <li>■ Cardiac conduction impulses occur in organized pattern, but this fails to produce myocardial contraction (former “electromechanical dissociation”); or insufficient ventricular filling during diastole; or ineffective contractions</li> </ul>		
<b>Defining Criteria per ECG</b>	<ul style="list-style-type: none"> <li>■ Rhythm displays organized electrical activity (not VF/pulseless VT)</li> <li>■ Seldom as organized as normal sinus rhythm</li> <li>■ Can be narrow (QRS &lt;0.10 mm) or wide (QRS &gt;0.12 mm); fast (&gt;100 beats/min) or slow (&lt;60 beats/min)</li> <li>■ Most frequently: fast and narrow (noncardiac etiology) or slow and wide (cardiac etiology)</li> </ul>		
<b>Clinical Manifestations</b>	<ul style="list-style-type: none"> <li>■ Collapse; unconscious</li> <li>■ Agonal respirations or apnea</li> <li>■ No pulse detectable by arterial palpation (thus could still be as high as 50-60 mm Hg; in such cases termed <i>pseudo-PEA</i>)</li> </ul>		
<b>Common Etiologies</b>	<p><i>Mnemonic of 5 H's and 5 T's aids recall:</i></p> <table style="width: 100%; border: none;"> <tr> <td style="vertical-align: top;"> <ul style="list-style-type: none"> <li>■ Hypovolemia</li> <li>■ Hypoxia</li> <li>■ Hydrogen ion—acidosis</li> <li>■ Hyperkalemia/Hypokalemia</li> <li>■ Hypothermia</li> </ul> </td> <td style="vertical-align: top;"> <ul style="list-style-type: none"> <li>■ “Tablets” (drug OD, ingestions)</li> <li>■ Tamponade, cardiac</li> <li>■ Tension pneumothorax</li> <li>■ Thrombosis, coronary (ACS)</li> <li>■ Thrombosis, pulmonary (embolism)</li> </ul> </td> </tr> </table>	<ul style="list-style-type: none"> <li>■ Hypovolemia</li> <li>■ Hypoxia</li> <li>■ Hydrogen ion—acidosis</li> <li>■ Hyperkalemia/Hypokalemia</li> <li>■ Hypothermia</li> </ul>	<ul style="list-style-type: none"> <li>■ “Tablets” (drug OD, ingestions)</li> <li>■ Tamponade, cardiac</li> <li>■ Tension pneumothorax</li> <li>■ Thrombosis, coronary (ACS)</li> <li>■ Thrombosis, pulmonary (embolism)</li> </ul>
<ul style="list-style-type: none"> <li>■ Hypovolemia</li> <li>■ Hypoxia</li> <li>■ Hydrogen ion—acidosis</li> <li>■ Hyperkalemia/Hypokalemia</li> <li>■ Hypothermia</li> </ul>	<ul style="list-style-type: none"> <li>■ “Tablets” (drug OD, ingestions)</li> <li>■ Tamponade, cardiac</li> <li>■ Tension pneumothorax</li> <li>■ Thrombosis, coronary (ACS)</li> <li>■ Thrombosis, pulmonary (embolism)</li> </ul>		
<b>Recommended Therapy</b> <i>Comprehensive ECC Algorithm, page 10; PEA Algorithm, page 100</i>	<ul style="list-style-type: none"> <li>■ Per PEA algorithm</li> <li>■ Primary ABCD (basic CPR)</li> <li>■ Secondary <b>AB</b> (advanced airway and ventilation);  <ul style="list-style-type: none"> <li><b>C</b> (IV, <i>epinephrine</i>, <i>atropine</i> if electrical activity &lt;60 complexes per minute);</li> <li><b>D</b> (identify and treat reversible causes)</li> </ul> </li> <li>■ <b>Key:</b> identify and treat a reversible cause of the PEA</li> </ul>		



Any organized rhythm without detectable pulse is “PEA”

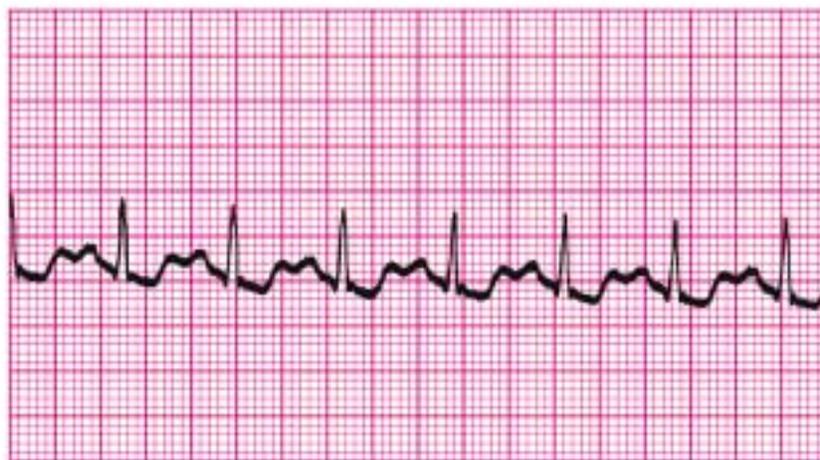
## 4. Asystole

<p><b>Defining Criteria per ECG</b></p> <p>Classically <i>asystole</i> presents as a “flat line”; any defining criteria are virtually nonexistent</p>	<ul style="list-style-type: none"> <li>■ <b>Rate:</b> no ventricular activity seen or <math>\leq 6</math>/min; so-called “P-wave asystole” occurs with only atrial impulses present to form P waves</li> <li>■ <b>Rhythm:</b> no ventricular activity seen; or <math>\leq 6</math>/min</li> <li>■ <b>PR:</b> cannot be determined; occasionally P wave seen, but by definition R wave must be absent</li> <li>■ <b>QRS complex:</b> no deflections seen that are consistent with a QRS complex</li> </ul>
<p><b>Clinical Manifestations</b></p>	<ul style="list-style-type: none"> <li>■ Early may see agonal respirations; unconscious; unresponsive</li> <li>■ No pulse; no blood pressure</li> <li>■ Cardiac arrest</li> </ul>
<p><b>Common Etiologies</b></p>	<ul style="list-style-type: none"> <li>■ End of life (death)</li> <li>■ Ischemia/hypoxia from many causes</li> <li>■ Acute respiratory failure (no oxygen; apnea; asphyxiation)</li> <li>■ Massive electrical shock: electrocution; lightning strike</li> <li>■ Postdefibrillatory shocks</li> </ul>
<p><b>Recommended Therapy</b></p> <p><i>Comprehensive ECC Algorithm, page 10; Asystole Algorithm, page 112</i></p>	<ul style="list-style-type: none"> <li>■ Always check for DNAR status</li> <li>■ Primary ABCD survey (basic CPR)</li> <li>■ Secondary ABCD survey</li> </ul>



Asystole: agonal complexes too slow to make this rhythm “PEA”

5. Sinus Tachycardia	
<b>Pathophysiology</b>	<ul style="list-style-type: none"> <li>■ None—more a physical sign than an arrhythmia or pathologic condition</li> <li>■ Normal impulse formation and conduction</li> </ul>
<b>Defining Criteria and ECG Features</b>	<ul style="list-style-type: none"> <li>■ <b>Rate:</b> &gt;100 beats/min</li> <li>■ <b>Rhythm:</b> sinus</li> <li>■ <b>PR:</b> ≤0.20 sec</li> <li>■ <b>QRS complex:</b> normal</li> </ul>
<b>Clinical Manifestations</b>	<ul style="list-style-type: none"> <li>■ None specific for the tachycardia</li> <li>■ Symptoms may be present due to the cause of the tachycardia (fever, hypovolemia, etc)</li> </ul>
<b>Common Etiologies</b>	<ul style="list-style-type: none"> <li>■ Normal exercise</li> <li>■ Fever</li> <li>■ Hypovolemia</li> <li>■ Adrenergic stimulation; anxiety</li> <li>■ Hyperthyroidism</li> </ul>
<b>Recommended Therapy</b> No specific treatment for sinus tachycardia	<ul style="list-style-type: none"> <li>■ Never treat the tachycardia per se</li> <li>■ Treat only the causes of the tachycardia</li> <li>■ Never countershock</li> </ul>



Sinus tachycardia

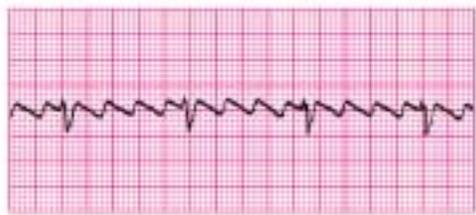
Rhythmic Algorithm No. 1: Tachycardias Overview



Tachycardia



Atrial fibrillation



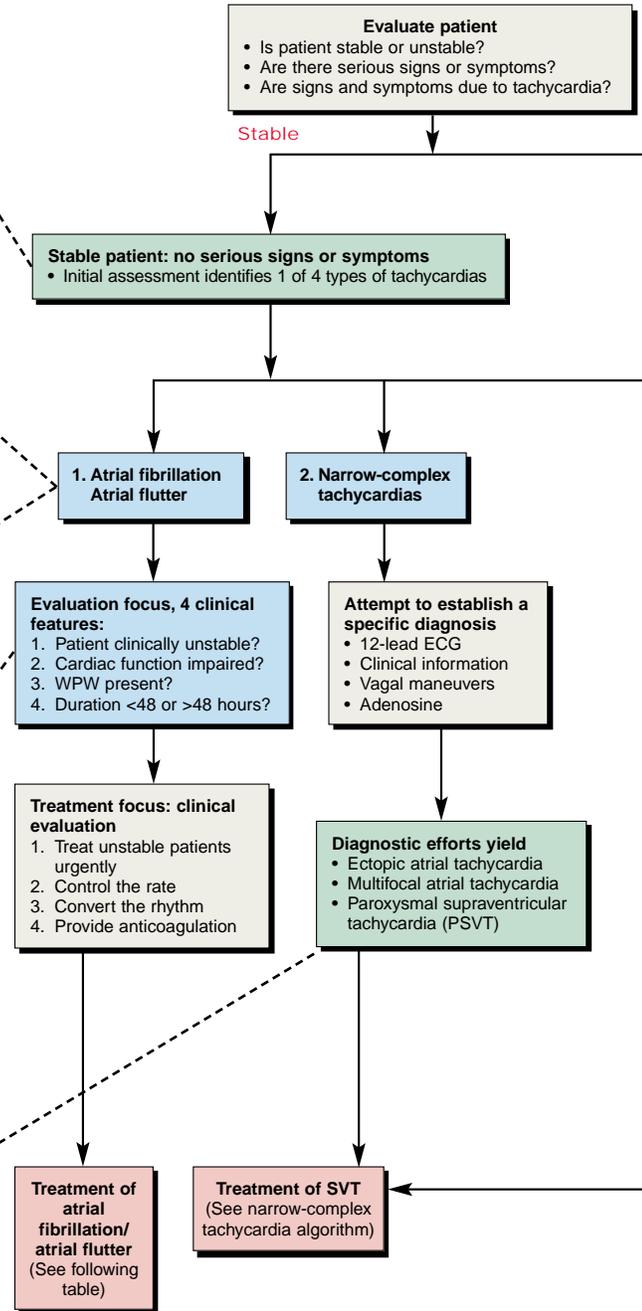
Atrial flutter

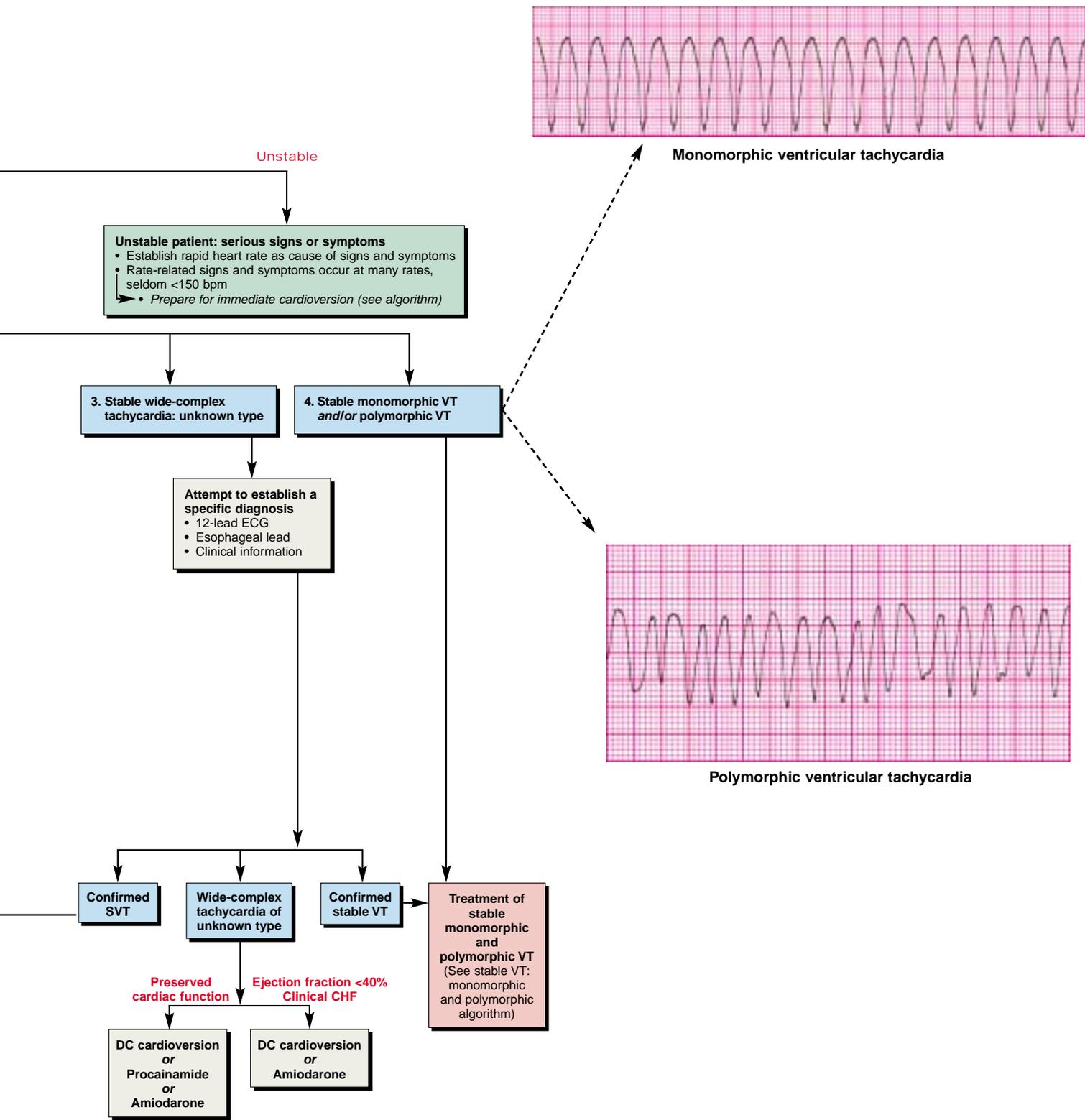


Sinus rhythm with WPW syndrome



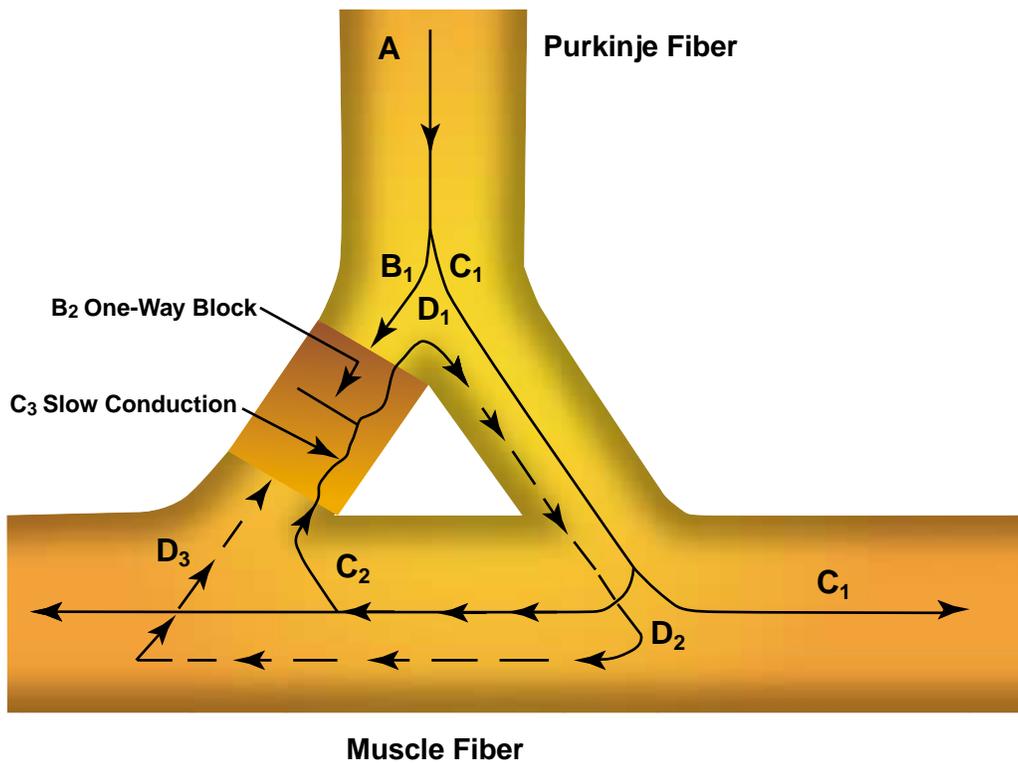
Initial sinus rhythm with paroxysmal onset of supraventricular tachycardia (PSVT)





## 6. Reentry Tachycardia Mechanism

- A** — Normal impulse comes down Purkinje fibers to join muscle fibers.
- B** — One impulse ( $B_1$ ) encounters an area of one-way (unidirectional) block ( $B_2$ ) and stops.
- C** — Meanwhile, the normally conducted impulse ( $C_1$ ) has moved down the Purkinje fiber, into the muscle fiber ( $C_2$ ); and as a retrograde impulse, moves through the area of slow conduction ( $C_3$ ).
- D** — The retrograde impulse ( $D_1$ ) now reenters the Purkinje and muscle fibers ( $D_2$ ); and keeps this reentry cycle repeating itself multiple times ( $D_3$ ).



7. Atrial Fibrillation/Atrial Flutter		
<b>Pathophysiology</b>	<ul style="list-style-type: none"> <li>■ Atrial impulses faster than SA node impulses</li> <li>■ Atrial fibrillation → impulses take multiple, chaotic, random pathways through the atria</li> <li>■ Atrial flutter → impulses take a circular course around the atria, setting up the flutter waves</li> <li>■ Mechanism of impulse formation: reentry</li> </ul>	
<b>Defining Criteria and ECG Features</b> (Distinctions here between atrial fibrillation vs atrial flutter; all other characteristics are the same) <b>Atrial Fibrillation Key:</b> A classic clinical axiom: <i>"Irregularly irregular rhythm—with variation in both interval and amplitude from R wave to R wave—is always atrial fibrillation."</i> This one is dependable. <b>Atrial Flutter Key:</b> Flutter waves seen in classic "sawtooth pattern"	<b>Atrial Fibrillation</b>	
	<b>Rate</b>	<ul style="list-style-type: none"> <li>■ Wide-ranging ventricular response to atrial rate of 300-400 beats/min</li> </ul>
	<b>Rhythm</b>	<ul style="list-style-type: none"> <li>■ Irregular (classic "irregularly irregular")</li> </ul>
	<b>P waves</b>	<ul style="list-style-type: none"> <li>■ Chaotic atrial fibrillatory waves only</li> <li>■ Creates disturbed baseline</li> </ul>
	<b>PR</b>	<ul style="list-style-type: none"> <li>■ Cannot be measured</li> </ul>
	<b>QRS</b>	<ul style="list-style-type: none"> <li>■ Remains ≤0.10-0.12 sec unless QRS complex distorted by fibrillation/flutter waves or by conduction defects through ventricles</li> </ul>
<b>Clinical Manifestations</b>	<ul style="list-style-type: none"> <li>■ Signs and symptoms are function of the rate of ventricular response to atrial fibrillatory waves; <i>"atrial fibrillation with rapid ventricular response"</i> → DOE, SOB, acute pulmonary edema</li> <li>■ Loss of <i>"atrial kick"</i> may lead to drop in cardiac output and decreased coronary perfusion</li> <li>■ Irregular rhythm often perceived as <i>"palpitations"</i></li> <li>■ Can be asymptomatic</li> </ul>	
<b>Common Etiologies</b>	<ul style="list-style-type: none"> <li>■ Acute coronary syndromes; CAD; CHF</li> <li>■ Disease at mitral or tricuspid valve</li> <li>■ Hypoxia; acute pulmonary embolism</li> <li>■ Drug-induced: <i>digoxin</i> or <i>quinidine</i> most common</li> <li>■ Hyperthyroidism</li> </ul>	

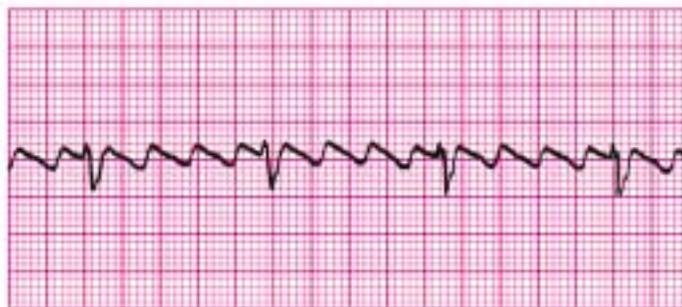
## 7. Atrial Fibrillation/Atrial Flutter (continued)

Recommended Therapy		Control Rate	
Evaluation Focus:	Treatment Focus:	Normal Heart	Impaired Heart
1. Patient clinically unstable? 2. Cardiac function impaired? 3. WPW present? 4. Duration $\leq 48$ or $>48$ hr?	1. Treat unstable patients urgently 2. Control the rate 3. Convert the rhythm 4. Provide anticoagulation	<ul style="list-style-type: none"> <li>■ Diltiazem or another calcium channel blocker <b>or</b> metoprolol or another <math>\beta</math>-blocker</li> </ul>	<ul style="list-style-type: none"> <li>■ Digoxin <b>or</b> diltiazem <b>or</b> amiodarone</li> </ul>
		<b>Convert Rhythm</b>	
		<b>Impaired Heart</b>	<b>Normal Heart</b>
		<ul style="list-style-type: none"> <li>■ If <math>\leq 48</math> hours:               <ul style="list-style-type: none"> <li>— DC cardioversion or <i>amiodarone</i> or others</li> </ul> </li> <li>■ If <math>&gt;48</math> hours:               <ul style="list-style-type: none"> <li>— Anticoagulate <math>\times 3</math> wk, <b>then</b></li> <li>— DC cardioversion, <b>then</b></li> <li>— Anticoagulate <math>\times 4</math> wk</li> </ul> </li> <li style="text-align: center;"><b>or</b></li> <li>■ IV <i>heparin</i> and TEE to rule out atrial clot, <b>then</b></li> <li>■ DC cardioversion within 24 hours, <b>then</b></li> <li>■ Anticoagulation <math>\times 4</math> more wk</li> </ul>	<ul style="list-style-type: none"> <li>■ If <math>\leq 48</math> hours:               <ul style="list-style-type: none"> <li>— DC Cardioversion <b>or</b> <i>amiodarone</i></li> </ul> </li> <li>■ If <math>&gt;48</math> hours:               <ul style="list-style-type: none"> <li>— Anticoagulate <math>\times 3</math> wk, <b>then</b></li> <li>— DC cardioversion, <b>then</b></li> <li>— Anticoagulate <math>\times 4</math> more wk</li> </ul> </li> </ul>

TEE indicates transesophageal echocardiogram.



Atrial fibrillation



Atrial flutter

8. WPW (Wolff-Parkinson-White) Syndrome	
<p><b>Pathophysiology</b></p>	<ul style="list-style-type: none"> <li>■ The prototypical <b>pre-excitation syndrome</b>: congenital malformation; strands of conducting myocardial tissue between atria and ventricles</li> <li>■ When persistent after birth strands can form an accessory pathway (eg, bundle of Kent)</li> </ul>
<p><b>Defining Criteria and ECG Features</b></p> <p><b>Key: QRS complex</b> is classically distorted by delta wave (upwards deflection of QRS is slurred)</p>	<ul style="list-style-type: none"> <li>■ <b>Rate</b>: most often 60-100 beats/min as usual rhythm is sinus</li> <li>■ <b>Rhythm</b>: normal sinus except during pre-excitation tachycardia</li> <li>■ <b>PR</b>: shorter since conduction through accessory pathway is faster than through AV node</li> <li>■ <b>P waves</b>: normal conformation</li> <li>■ <b>QRS complex</b>: classically distorted by delta wave (upwards deflection of QRS is slurred)</li> </ul>
<p><b>Clinical Manifestations</b></p>	<ul style="list-style-type: none"> <li>■ A person with WPW may never have symptoms</li> <li>■ People with WPW have same annual incidence of atrial fibrillation as age- and gender-matched population</li> <li>■ Onset of atrial fibrillation for WPW patients, however, poses risk of rapid ventricular response through the accessory pathway</li> <li>■ This rapid ventricular response can lead to all signs and symptoms of stable and unstable tachycardias</li> </ul>
<p><b>Common Etiology</b></p>	<ul style="list-style-type: none"> <li>■ The accessory pathway in WPW is a congenital malformation</li> </ul>

## 8. WPW (Wolff-Parkinson-White) Syndrome (continued)

Recommended Therapy		Wolff-Parkinson-White: Control Rate	
Evaluation Focus	Treatment Focus	Normal Heart	Impaired Heart
<ol style="list-style-type: none"> <li>1. Patient clinically unstable?</li> <li>2. Cardiac function impaired?</li> <li>3. WPW present?</li> <li>4. Duration <math>\leq 48</math> or <math>&gt;48</math> hr?</li> </ol>	<ol style="list-style-type: none"> <li>1. Treat unstable patients urgently</li> <li>2. Control the rate</li> <li>3. Convert the rhythm</li> <li>4. Provide anticoagulation</li> </ol>	<ul style="list-style-type: none"> <li>■ Cardioversion or</li> <li>■ Antiarrhythmic (IIb): <i>amiodarone or flecainide or procainamide or propafenone or sotalol</i></li> </ul>	<ul style="list-style-type: none"> <li>■ Cardioversion or</li> <li>■ <i>Amiodarone</i></li> </ul>
<b>Class III (can be harmful) in treating atrial fibrillation with WPW:</b> <ul style="list-style-type: none"> <li>■ <i>Adenosine</i></li> <li>■ <i><math>\beta</math>-Blockers</i></li> <li>■ <i>Calcium channel blockers</i></li> <li>■ <i>Digoxin</i></li> </ul>		Wolff-Parkinson-White: Convert Rhythm	
		Duration $\leq 48$ Hours	Duration $>48$ Hours
		<ul style="list-style-type: none"> <li>■ Cardioversion or</li> <li>■ Antiarrhythmic (IIb): <i>amiodarone or flecainide or procainamide or propafenone or sotalol</i></li> </ul> <b>If impaired heart:</b> cardioversion or <i>amiodarone</i>	<ul style="list-style-type: none"> <li>■ Anticoagulate <math>\times</math> 3 wk then</li> <li>■ DC cardioversion then</li> <li>■ Anticoagulate <math>\times</math> 4 wk</li> </ul>



Wolff-Parkinson-White syndrome: normal sinus rhythm with *delta wave* (arrow) notching of positive upstroke of QRS complex

9. Junctional Tachycardia	
<b>Pathophysiology</b>	<ul style="list-style-type: none"> <li>■ Area of <i>automaticity</i> (automatic impulse formation) develops in the AV node (“junction”)</li> <li>■ Both retrograde and antegrade transmission occurs</li> </ul>
<b>Defining Criteria and ECG Features</b> <ul style="list-style-type: none"> <li>■ <b>Key:</b> position of the P wave; may show antegrade or retrograde propagation because origin is at the junction; may arise before, after, or with the QRS</li> </ul>	<ul style="list-style-type: none"> <li>■ <b>Rate:</b> 100-180 beats/min</li> <li>■ <b>Rhythm:</b> regular atrial and ventricular firing</li> <li>■ <b>PR:</b> often not measurable unless P wave comes before QRS; then will be short (&lt;0.12 secs)</li> <li>■ <b>P waves:</b> often obscured; may propagate antegrade or retrograde with origin at the junction; may arise before, after, or with the QRS</li> <li>■ <b>QRS complex:</b> narrow; ≤0.10 secs in absence of intraventricular conduction defect</li> </ul>
<b>Clinical Manifestations</b>	<ul style="list-style-type: none"> <li>■ Patients may have clinical signs of a reduced ejection fraction because augmented flow from atrium is lost</li> <li>■ Symptoms of unstable tachycardia may occur</li> </ul>
<b>Common Etiologies</b>	<ul style="list-style-type: none"> <li>■ Digoxin toxicity</li> <li>■ Acute sequelae of acute coronary syndromes</li> </ul>
<b>Recommended Therapy</b> If specific diagnosis unknown, attempt therapeutic/diagnostic maneuver with <ul style="list-style-type: none"> <li>■ Vagal stimulation</li> <li>■ Adenosine . . . THEN →</li> </ul>	<p><b>Preserved heart function:</b></p> <ul style="list-style-type: none"> <li>■ <i>β-Blocker</i></li> <li>■ <i>Calcium channel blocker</i></li> <li>■ <i>Amiodarone</i></li> <li>■ <b>NO DC cardioversion!</b></li> </ul> <p><b>If impaired heart function:</b></p> <ul style="list-style-type: none"> <li>■ <i>Amiodarone</i></li> <li>■ <b>NO DC cardioversion!</b></li> </ul>



Junctional tachycardia: narrow QRS complexes at 130 bpm; P waves arise with QRS

Rhythmic Algorithm No. 2: Narrow-Complex Tachycardias



Supraventricular tachycardia



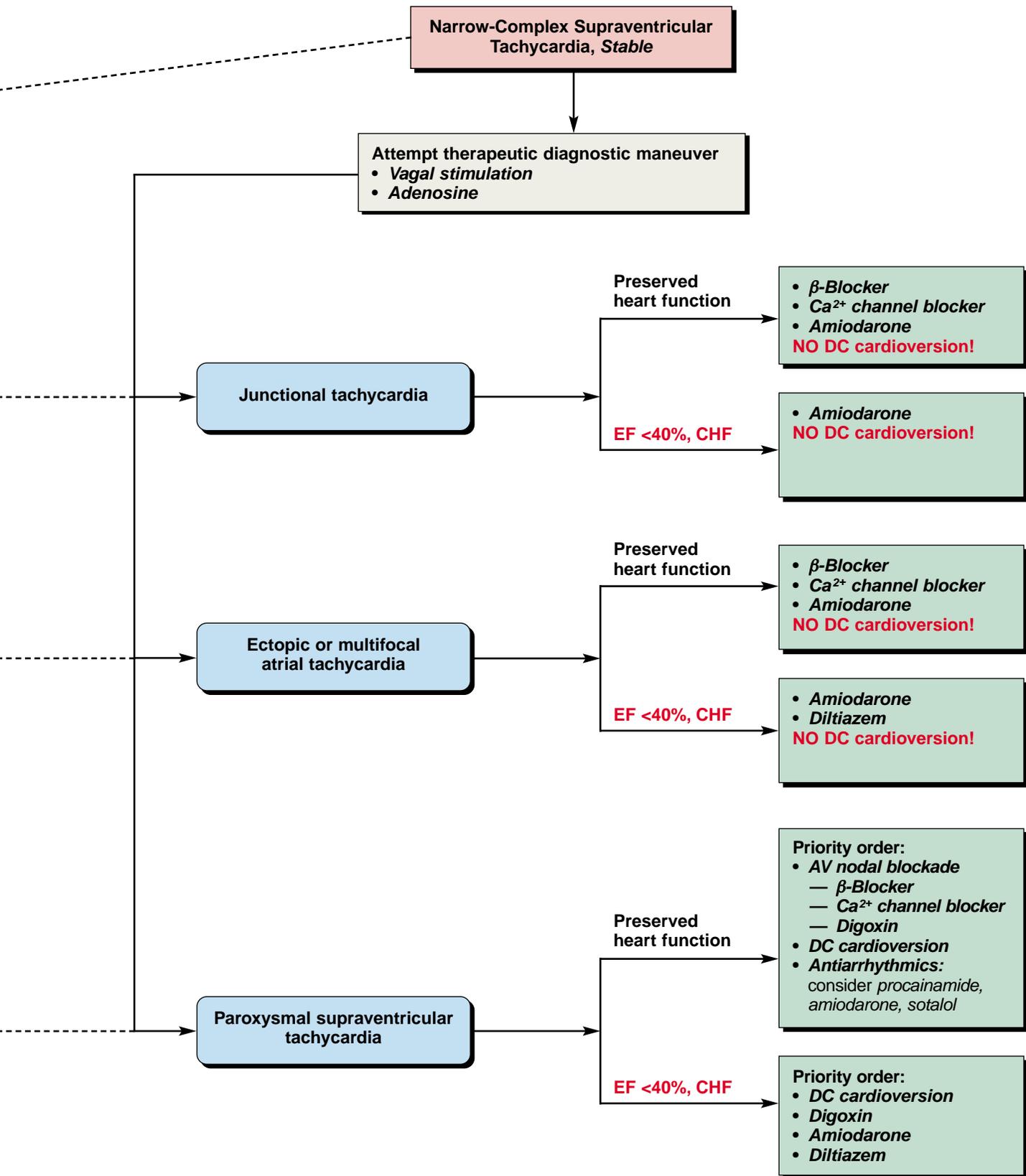
Junctional tachycardia



Multifocal atrial tachycardia



Sinus rhythm (3 complexes) with paroxysmal onset (arrow) of supraventricular tachycardia (PSVT)



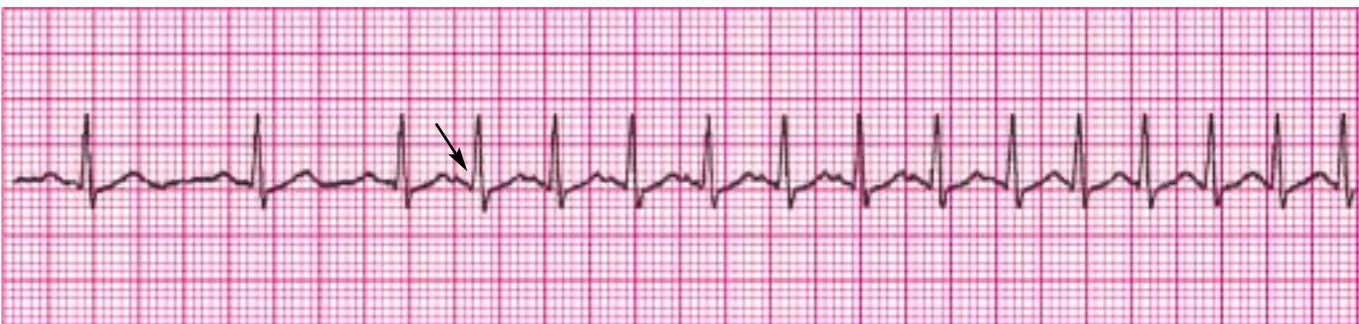
## 10. Multifocal Atrial Tachycardia

<b>Pathophysiology</b>	<ul style="list-style-type: none"> <li>■ Areas of <i>automaticity</i> (impulse formation) originate irregularly and rapidly at different points in the atria</li> </ul>
<b>Defining Criteria and ECG Features</b> If the rate is <100 beats/min, this rhythm is termed “ <i>wandering atrial pacemaker</i> ” or “ <i>multifocal atrial rhythm</i> ” <b>Key:</b> By definition must have 3 or more P waves that differ in polarity (up/down), shape, and size since the atrial impulse is generated from multiple foci.	<ul style="list-style-type: none"> <li>■ <b>Rate:</b> &gt;100 beats/min; usually &gt;130 bpm</li> <li>■ <b>Rhythm:</b> irregular atrial firing</li> <li>■ <b>PR:</b> variable</li> <li>■ <b>P waves:</b> by definition must have 3 or more P waves that differ in polarity (up/down), shape, and size since the atrial impulse is generated from multiple foci</li> <li>■ <b>QRS complex:</b> narrow; ≤0.10 sec in absence of intraventricular conduction defect</li> </ul>
<b>Clinical Manifestations</b>	<ul style="list-style-type: none"> <li>■ Patients may have no clinical signs</li> <li>■ Symptoms of unstable tachycardia may occur</li> </ul>
<b>Common Etiologies</b>	<ul style="list-style-type: none"> <li>■ Most common cause is COPD (<i>cor pulmonale</i>) where pulmonary hypertension places increased strain on the right ventricle and atrium</li> <li>■ Impaired and hypertrophied atrium gives rise to automaticity</li> <li>■ Also digoxin toxicity, rheumatic heart disease, acute coronary syndromes</li> </ul>
<b>Recommended Therapy</b> If specific diagnosis unknown, attempt therapeutic/diagnostic maneuver with <ul style="list-style-type: none"> <li>■ Vagal stimulation</li> <li>■ Adenosine . . . THEN →</li> </ul>	<b>Preserved heart function:</b> <ul style="list-style-type: none"> <li>■ <i>β-blocker</i></li> <li>■ <i>Calcium channel blocker</i></li> <li>■ <i>Amiodarone</i></li> <li>■ <b>NO DC cardioversion!</b></li> </ul> <b>If impaired heart function:</b> <ul style="list-style-type: none"> <li>■ <i>Amiodarone</i></li> <li>■ <i>Diltiazem</i></li> <li>■ <b>NO DC cardioversion!</b></li> </ul>



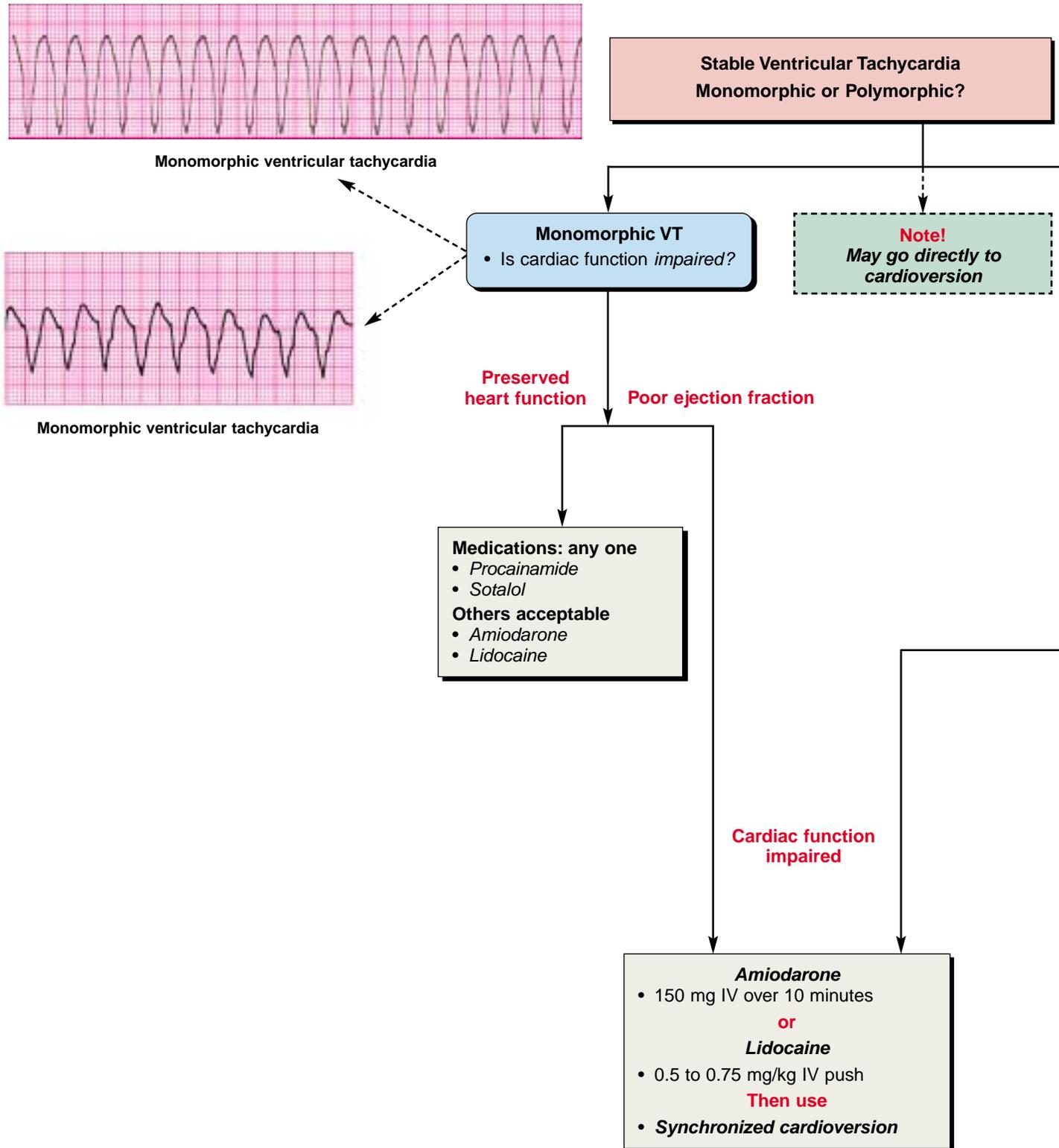
Multifocal atrial tachycardia: narrow-complex tachycardia at 140 to 160 bpm with multiple P-wave morphologies (arrows)

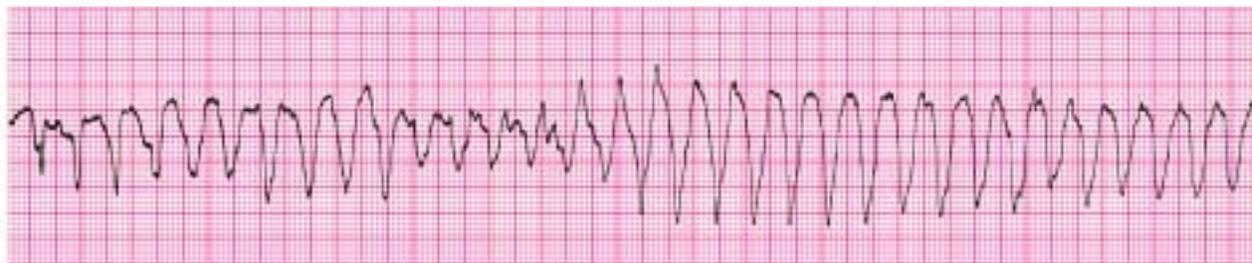
11. PSVT (Paroxysmal Supraventricular Tachycardia)	
<b>Pathophysiology</b>	<ul style="list-style-type: none"> <li>■ <b>Reentry phenomenon</b> (see page 260): impulses arise and recycle repeatedly in the AV node because of areas of unidirectional block in the Purkinje fibers</li> </ul>
<b>Defining Criteria and ECG Features</b> <b>Key:</b> Regular, narrow-complex tachycardia without P-waves, and <u>sudden</u> , <i>paroxysmal</i> onset or cessation, or both <b>Note:</b> To merit the diagnosis some experts require capture of the paroxysmal onset or cessation on a monitor strip	<ul style="list-style-type: none"> <li>■ <b>Rate:</b> exceeds upper limit of sinus tachycardia (&gt;120 beats/min); seldom &lt;150 beats/min; up to 250 beats/min</li> <li>■ <b>Rhythm:</b> regular</li> <li>■ <b>P waves:</b> seldom seen because rapid rate causes P wave loss in preceding T waves or because the origin is low in the atrium</li> <li>■ <b>QRS complex:</b> normal, narrow (<math>\leq 0.10</math> sec usually)</li> </ul>
<b>Clinical Manifestations</b>	<ul style="list-style-type: none"> <li>■ Palpitations felt by patient at the paroxysmal onset; becomes anxious, uncomfortable</li> <li>■ Exercise tolerance low with very high rates</li> <li>■ Symptoms of unstable tachycardia may occur</li> </ul>
<b>Common Etiologies</b>	<ul style="list-style-type: none"> <li>■ Accessory conduction pathway in many PSVT patients</li> <li>■ For such otherwise healthy people many factors can provoke the paroxysm, such as caffeine, hypoxia, cigarettes, stress, anxiety, sleep deprivation, numerous medications</li> <li>■ Also increased frequency of PSVT in unhealthy patients with CAD, COPD, CHF</li> </ul>
<b>Recommended Therapy</b> If specific diagnosis unknown, attempt therapeutic/diagnostic maneuver with <ul style="list-style-type: none"> <li>■ Vagal stimulation</li> <li>■ Adenosine . . . THEN →</li> </ul>	<p><b>Preserved heart function:</b></p> <ul style="list-style-type: none"> <li>■ AV nodal blockade               <ul style="list-style-type: none"> <li>— <math>\beta</math>-Blocker</li> <li>— Calcium channel blocker</li> <li>— Digoxin</li> </ul> </li> <li>■ DC cardioversion</li> <li>■ Parenteral antiarrhythmics:               <ul style="list-style-type: none"> <li>— Procainamide</li> <li>— Amiodarone</li> <li>— Sotalol (not available in the United States)</li> </ul> </li> </ul> <p><b>Impaired heart function:</b></p> <ul style="list-style-type: none"> <li>■ DC cardioversion</li> <li>■ Digoxin</li> <li>■ Amiodarone</li> <li>■ Diltiazem</li> </ul>



Sinus rhythm (3 complexes) with paroxysmal onset (arrow) of supraventricular tachycardia (PSVT)

Rhythmic Algorithm No. 3: Stable Ventricular Tachycardias





Torsades de pointes

**Polymorphic VT**  
 • Is baseline QT interval prolonged?

**Normal baseline QT interval**

**Prolonged baseline QT interval (suggests torsades)**

**Normal baseline QT interval**

- Treat ischemia
- Correct electrolytes

**Medications: any one**

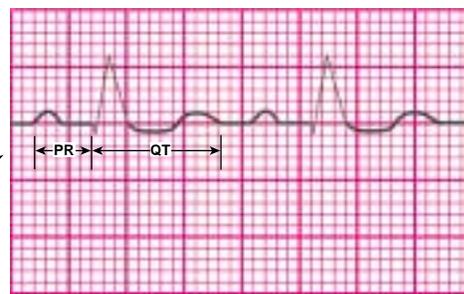
- $\beta$ -Blockers or
- Lidocaine or
- Amiodarone or
- Procainamide or
- Sotalol

**Long baseline QT interval**

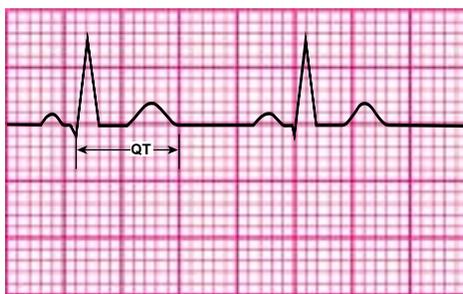
- Correct abnormal electrolytes

**Therapies: any one**

- Magnesium
- Overdrive pacing
- Isoproterenol
- Phenytoin
- Lidocaine



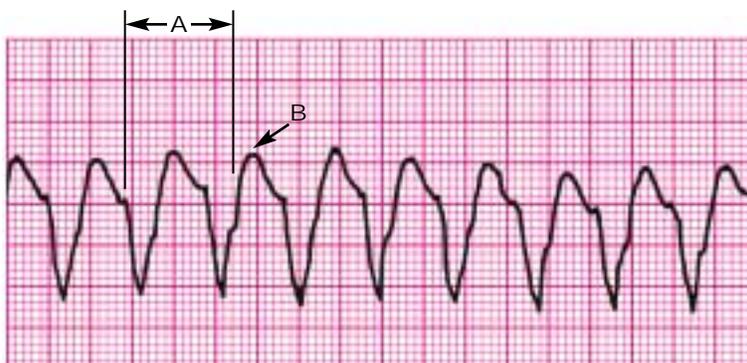
Prolonged baseline QT interval



Normal baseline QT interval

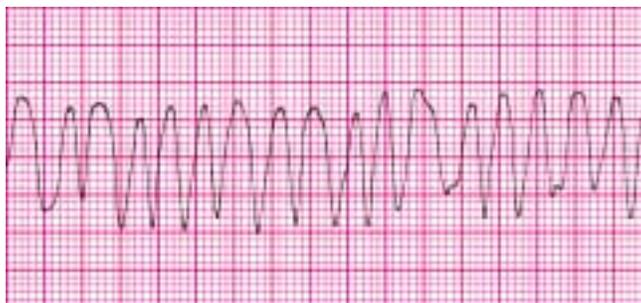
## 12. Monomorphic Ventricular Tachycardia (Stable)

<b>Pathophysiology</b>	<ul style="list-style-type: none"> <li>■ Impulse conduction is slowed around areas of ventricular injury, infarct, or ischemia</li> <li>■ These areas also serve as source of ectopic impulses (<i>irritable foci</i>)</li> <li>■ These areas of injury can cause the impulse to take a circular course, leading to the reentry phenomenon and rapid repetitive depolarizations</li> </ul>	
<b>Defining Criteria per ECG</b> <b>Key:</b> The same morphology, or shape, is seen in every QRS complex <b>Notes:</b> <ul style="list-style-type: none"> <li>■ 3 or more consecutive PVCs: <i>ventricular tachycardia</i></li> <li>■ VT &lt;30 sec duration → <i>non-sustained VT</i></li> <li>■ VT &gt;30 sec duration → <i>sustained VT</i></li> </ul>	<ul style="list-style-type: none"> <li>■ <b>Rate:</b> ventricular rate &gt;100 bpm; typically 120 to 250 bpm</li> <li>■ <b>Rhythm:</b> no atrial activity seen, only regular ventricular</li> <li>■ <b>PR:</b> nonexistent</li> <li>■ <b>P waves:</b> seldom seen but present; VT is a form of AV dissociation (which is a defining characteristic for wide-complex tachycardias of ventricular origin vs supraventricular tachycardias with aberrant conduction)</li> <li>■ <b>QRS complex:</b> wide and bizarre, "PVC-like" complexes &gt;0.12 sec, with large T wave of opposite polarity from QRS</li> </ul>	
<b>Clinical Manifestations</b>	<ul style="list-style-type: none"> <li>■ Monomorphic VT can be asymptomatic, despite the widespread erroneous belief that sustained VT always produces symptoms</li> <li>■ Majority of times, however, symptoms of decreased cardiac output (orthostasis, hypotension, syncope, exercise limitations, etc) are seen</li> <li>■ Untreated and sustained will deteriorate to unstable VT, often VF</li> </ul>	
<b>Common Etiologies</b>	<ul style="list-style-type: none"> <li>■ An acute ischemic event (see pathophysiology) with areas of "ventricular irritability" leading to PVCs</li> <li>■ PVCs that occur during the relative refractory period of the cardiac cycle ("R-on-T phenomenon")</li> <li>■ Drug-induced, prolonged QT interval (tricyclic antidepressants, procainamide, digoxin, some long-acting antihistamines)</li> </ul>	
<b>Recommended Therapy</b>	<b>Normal Heart</b>	<b>Impaired Heart</b>
	Any one of following parenteral antiarrhythmics: <ul style="list-style-type: none"> <li>■ <i>Procainamide</i></li> <li>■ <i>Sotalol</i></li> <li>■ <i>Amiodarone</i></li> <li>■ <i>Lidocaine</i></li> </ul>	<ul style="list-style-type: none"> <li>■ <i>Amiodarone</i></li> <li style="text-align: center;">or</li> <li>■ <i>Lidocaine</i></li> <li style="text-align: center;">then</li> <li>■ <i>DC cardioversion</i> if persists</li> </ul>



Monomorphic ventricular tachycardia at rate of 150 bpm: wide QRS complexes (arrow A) with opposite polarity T waves (arrow B)

13. Polymorphic Ventricular Tachycardia (Stable)					
<b>Pathophysiology</b>	<ul style="list-style-type: none"> <li>■ Impulse conduction is slowed around multiple areas of ventricular injury, infarct, or ischemia</li> <li>■ These areas also serve as the source of ectopic impulses (<i>irritable foci</i>); irritable foci occur in multiple areas of the ventricles, thus “<i>polymorphic</i>”</li> <li>■ These areas of injury can cause impulses to take a circular course, leading to the reentry phenomenon and rapid repetitive depolarizations</li> </ul>				
<b>Defining Criteria per ECG</b> <b>Key:</b> Marked variation and inconsistency seen in the QRS complexes	<ul style="list-style-type: none"> <li>■ <b>Rate:</b> ventricular rate &gt;100 bpm; typically 120 to 250</li> <li>■ <b>Rhythm:</b> only regular ventricular</li> <li>■ <b>PR:</b> nonexistent</li> <li>■ <b>P waves:</b> seldom seen but present; VT is a form of AV dissociation</li> <li>■ <b>QRS complexes:</b> marked variation and inconsistency seen in the QRS complexes</li> </ul>				
<b>Clinical Manifestations</b>	<ul style="list-style-type: none"> <li>■ Rare: asymptomatic polymorphic VT</li> <li>■ Majority of times: symptoms of decreased cardiac output (orthostasis, hypotension, syncope, exercise limitations, etc) are seen</li> <li>■ Seldom → <i>sustained VT</i>; seldom → “stable” VT</li> <li>■ Tends toward rapid deterioration to pulseless VT or VF</li> </ul>				
<b>Common Etiologies</b>	<ul style="list-style-type: none"> <li>■ An acute ischemic event (see pathophysiology) with areas of “ventricular irritability” leading to PVCs</li> <li>■ PVCs that occur during the relative refractory period of the cardiac cycle (“R-on-T phenomenon”)</li> <li>■ Drug-induced prolonged QT interval (tricyclic antidepressants, procainamide, digoxin, some long-acting antihistamines)</li> </ul>				
<b>Recommended Therapy</b>	<p><b>Review most recent 12-lead ECG (baseline)</b></p> <ul style="list-style-type: none"> <li>■ Measure QT interval just prior to onset of the polymorphic tachycardia</li> <li>■ QT interval prolongation? (if YES go to <i>Torsades de Pointes</i>; if NO see below)</li> </ul> <p><b>Normal baseline QT interval:</b></p> <ul style="list-style-type: none"> <li>■ Treat ischemia</li> <li>■ Correct electrolytes if abnormal</li> </ul> <p><b>Then:</b></p> <table border="1" style="width: 100%; border-collapse: collapse;"> <thead> <tr> <th style="width: 50%; text-align: center;">Normal Heart</th> <th style="width: 50%; text-align: center;">Impaired Heart</th> </tr> </thead> <tbody> <tr> <td style="vertical-align: top;"> <p><b>Parenteral medications: any one</b></p> <ul style="list-style-type: none"> <li>■ <i>β-Blockers</i> or</li> <li>■ <i>Lidocaine</i> or</li> <li>■ <i>Amiodarone</i> or</li> <li>■ <i>Procainamide</i> or</li> <li>■ <i>Sotalol</i></li> </ul> </td> <td style="vertical-align: top;"> <ul style="list-style-type: none"> <li>■ <i>Amiodarone</i></li> <li style="text-align: center;">or</li> <li>■ <i>Lidocaine</i></li> <li style="text-align: center;">then</li> <li>■ <i>DC cardioversion</i> if persists</li> </ul> </td> </tr> </tbody> </table>	Normal Heart	Impaired Heart	<p><b>Parenteral medications: any one</b></p> <ul style="list-style-type: none"> <li>■ <i>β-Blockers</i> or</li> <li>■ <i>Lidocaine</i> or</li> <li>■ <i>Amiodarone</i> or</li> <li>■ <i>Procainamide</i> or</li> <li>■ <i>Sotalol</i></li> </ul>	<ul style="list-style-type: none"> <li>■ <i>Amiodarone</i></li> <li style="text-align: center;">or</li> <li>■ <i>Lidocaine</i></li> <li style="text-align: center;">then</li> <li>■ <i>DC cardioversion</i> if persists</li> </ul>
Normal Heart	Impaired Heart				
<p><b>Parenteral medications: any one</b></p> <ul style="list-style-type: none"> <li>■ <i>β-Blockers</i> or</li> <li>■ <i>Lidocaine</i> or</li> <li>■ <i>Amiodarone</i> or</li> <li>■ <i>Procainamide</i> or</li> <li>■ <i>Sotalol</i></li> </ul>	<ul style="list-style-type: none"> <li>■ <i>Amiodarone</i></li> <li style="text-align: center;">or</li> <li>■ <i>Lidocaine</i></li> <li style="text-align: center;">then</li> <li>■ <i>DC cardioversion</i> if persists</li> </ul>				



Polymorphic ventricular tachycardia: QRS complexes display multiple morphologies (“polymorphic”)

## 14. Torsades de Pointes (a Unique Subtype of Polymorphic Ventricular Tachycardia)

<b>Pathophysiology</b>	<p>Specific pathophysiology for classic torsades:</p> <ul style="list-style-type: none"> <li>■ QT interval is abnormally long (see below for etiology of QT prolongation)</li> <li>■ Leads to increase in the relative refractory period (“vulnerable period”) of the cardiac cycle</li> <li>■ Increases probability that an irritable focus (PVC) will occur on the T-wave (“vulnerable period” or “R-on-T phenomenon”)</li> <li>■ R-on-T phenomenon often induces VT</li> </ul>
<b>Defining Criteria per ECG</b> <b>Key:</b> QRS complexes display “spindle-node” pattern → VT amplitude increases then decreases in regular pattern (creates the “spindle”) → initial deflection at start of one spindle (eg, negative) will be followed by the opposite (eg, positive) deflection at the start of the next spindle (creates the “node”)	<ul style="list-style-type: none"> <li>■ <b>Atrial Rate:</b> cannot determine atrial rate</li> <li>■ <b>Ventricular rate:</b> 150-250 complexes/min</li> <li>■ <b>Rhythm:</b> only irregular ventricular rhythm</li> <li>■ <b>PR:</b> nonexistent</li> <li>■ <b>P waves:</b> nonexistent</li> <li>■ <b>QRS complexes:</b> display classic “spindle-node” pattern (see left column: “Key”)</li> </ul>
<b>Clinical Manifestations</b>	<ul style="list-style-type: none"> <li>■ Majority of times patients with torsades have symptoms of decreased cardiac output (orthostasis, hypotension, syncope, exercise limitations, etc)</li> <li>■ Asymptomatic torsades, <i>sustained</i> torsades, or “<i>stable</i>” torsades is uncommon</li> <li>■ Tends toward sudden deterioration to pulseless VT or VF</li> </ul>
<b>Common Etiologies</b>	<p>Most commonly occurs with prolonged QT interval, from many causes:</p> <ul style="list-style-type: none"> <li>■ Drug-induced: tricyclic antidepressants, procainamide, digoxin, some long-acting antihistamines</li> <li>■ Electrolyte and metabolic alterations (hypomagnesemia is the prototype)</li> <li>■ Inherited forms of long QT syndrome</li> <li>■ Acute ischemic events (see pathophysiology)</li> </ul>
<b>Recommended Therapy</b>	<p><b>Review most recent 12-lead ECG (baseline):</b></p> <ul style="list-style-type: none"> <li>■ Measure QT interval just before onset of the polymorphic tachycardia</li> <li>■ QT interval prolongation? (if YES see below; if NO go to the polymorphic VT algorithm)</li> </ul> <p><b>Long baseline QT interval:</b></p> <ul style="list-style-type: none"> <li>■ Treat ischemia</li> <li>■ Correct electrolytes if abnormal</li> </ul> <p><b>Then therapies (any one):</b></p> <ul style="list-style-type: none"> <li>■ Magnesium</li> <li>■ Overdrive pacing</li> <li>■ Isoproterenol (pharmacologic overdrive pacing)</li> <li>■ Phenytoin</li> <li>■ Lidocaine</li> </ul>

**Torsades de pointes**

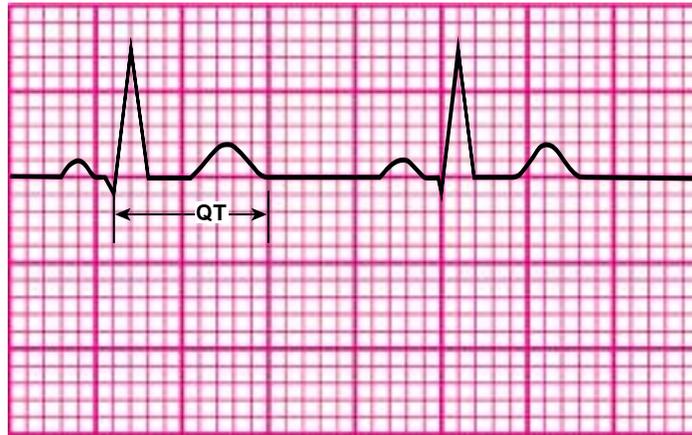
(a unique subtype of polymorphic ventricular tachycardia)

Arrows: A — Start of a “spindle”; note negative initial deflection; note increasing QRS amplitude

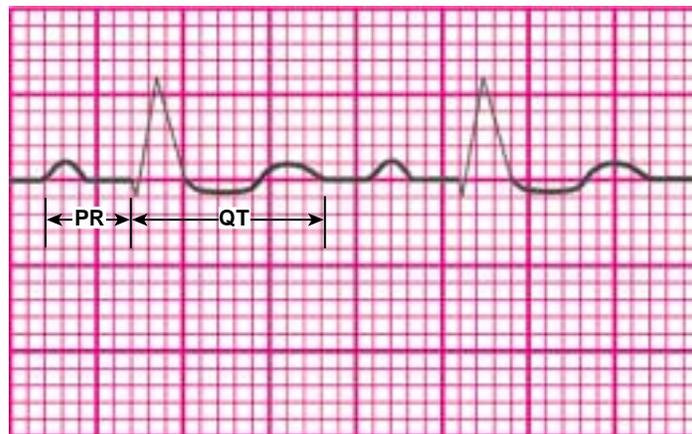
B — End of “spindle”; start of “node”

C — End of “node”; start of next “spindle”; note positive initial deflection; increase-decrease in QRS amplitude

15. Normal and Prolonged Baseline QT Interval



**Normal baseline QT interval**  
**Rate:** 80 bpm  
**QT interval:** 0.36 sec  
 (within  $QT_C$  range of 0.32 – 0.39 sec  
 for a heart rate of 80 bpm)



**Prolonged baseline QT interval**  
**Due to drug toxicity**  
**PR interval:** >0.20 sec  
**Rate:** 80 bpm  
**QT interval:** prolonged, 0.45 sec  
 (above  $QT_C$  range of 0.32 – 0.39 sec  
 for a heart rate of 80 bpm)  
**QRS complex:** widened, >0.12 sec

Rhythmic Algorithm No. 4: Bradycardias



Sinus bradycardia with borderline first-degree AV block



Second-degree AV block type I



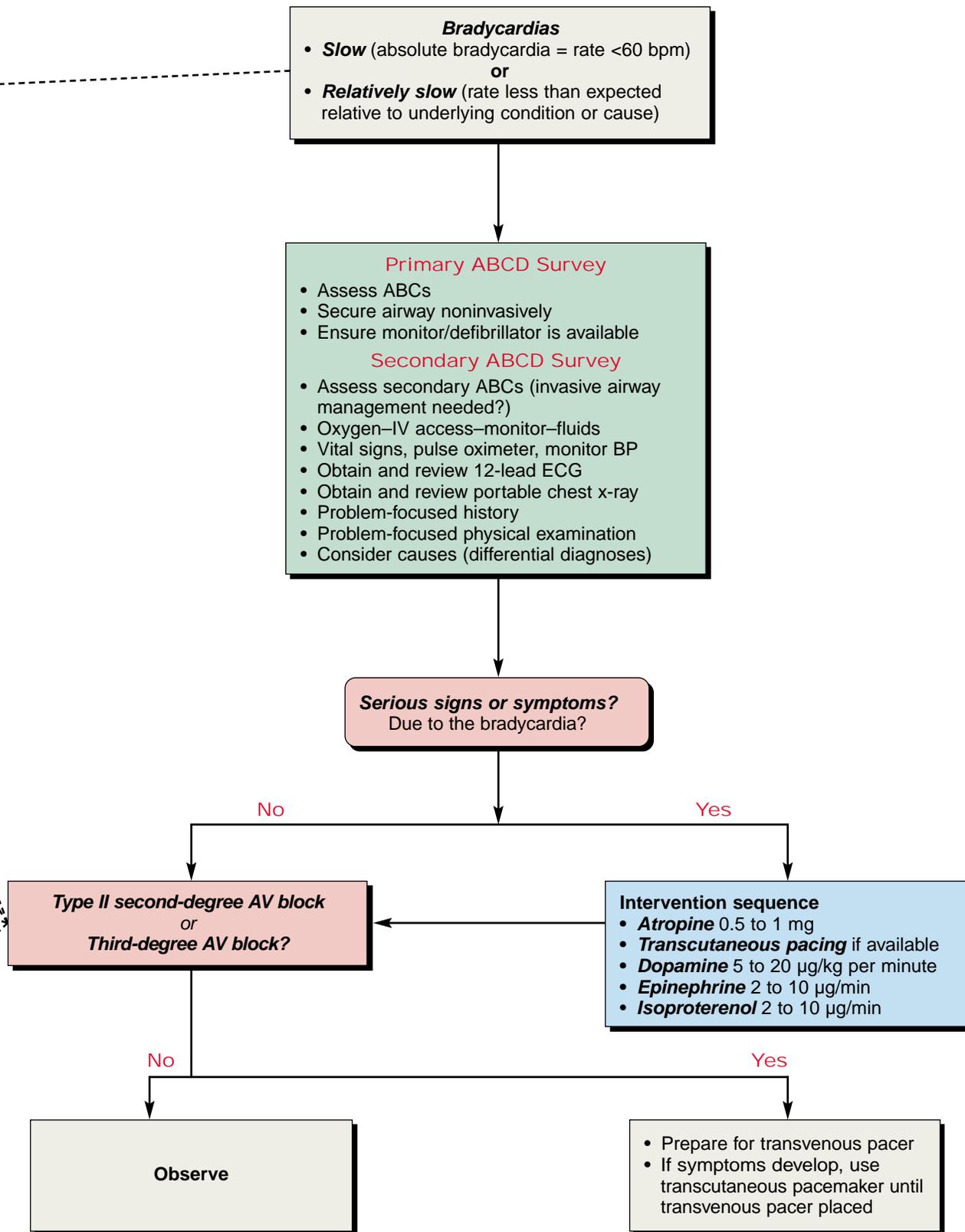
Second-degree AV block type II



Complete AV block with a ventricular escape pacemaker (wide QRS: 0.12 to 0.14 sec)



Third-degree AV block with a junctional escape pacemaker (narrow QRS: <0.12)



## 16. Sinus Bradycardia

<b>Pathophysiology</b>	<ul style="list-style-type: none"> <li>■ Impulses originate at SA node at a slow rate</li> <li>■ Not pathological; not an abnormal arrhythmia</li> <li>■ More a physical sign</li> </ul>
<b>Defining Criteria per ECG</b> <b>Key:</b> Regular P waves followed by regular QRS complexes at rate <60 beats/min <b>Note:</b> Often a physical sign rather than an abnormal rhythm	<ul style="list-style-type: none"> <li>■ <b>Rate:</b> &lt;60 beats/min</li> <li>■ <b>Rhythm:</b> regular sinus</li> <li>■ <b>PR:</b> regular; &lt;0.20 sec</li> <li>■ <b>P waves:</b> size and shape normal; every P wave is followed by a QRS complex; every QRS complex is preceded by a P wave</li> <li>■ <b>QRS complex:</b> narrow; <math>\leq 0.10</math> sec in absence of intraventricular conduction defect</li> </ul>
<b>Clinical Manifestations</b>	<ul style="list-style-type: none"> <li>■ At rest, usually asymptomatic</li> <li>■ With increased activity, persistent slow rate will lead to symptoms of easy fatigue, SOB, dizziness or lightheadedness, syncope, hypotension</li> </ul>
<b>Common Etiologies</b>	<ul style="list-style-type: none"> <li>■ Normal for well-conditioned people</li> <li>■ A vasovagal event such as vomiting, valsalva, rectal stimuli, inadvertent pressure on carotid sinus ("shaver's syncope")</li> <li>■ Acute MIs that affect circulation to SA node (right coronary artery); most often inferior AMIs</li> <li>■ Adverse drug effects, eg, blocking agents (<math>\beta</math> or calcium channel), digoxin, quinidine</li> </ul>
<b>Recommended Therapy</b>	<ul style="list-style-type: none"> <li>■ Treatment rarely indicated</li> <li>■ Treat only if patient has significant signs or symptoms due to the bradycardia</li> <li>■ Oxygen is always appropriate</li> </ul> <p><b>Intervention sequence for bradycardia</b></p> <ul style="list-style-type: none"> <li>■ <i>Atropine</i> 0.5 to 1 mg IV if vagal mechanism</li> <li>■ <i>Transcutaneous pacing</i> if available</li> </ul> <p><b>If signs and symptoms are severe, consider catecholamine infusions:</b></p> <ul style="list-style-type: none"> <li>■ <i>Dopamine</i> 5 to 20 <math>\mu\text{g}/\text{kg}</math> per min</li> <li>■ <i>Epinephrine</i> 2 to 10 <math>\mu\text{g}/\text{min}</math></li> <li>■ <i>Isoproterenol</i> 2 to 10 <math>\mu\text{g}/\text{min}</math></li> </ul>



Sinus bradycardia: rate of 45 bpm; with borderline first-degree AV block (PR  $\approx$  0.20 sec)

17. First-Degree Heart Block

<p><b>Pathophysiology</b></p>	<ul style="list-style-type: none"> <li>■ Impulse conduction is slowed (<i>partial block</i>) at the AV node by a fixed amount</li> <li>■ Closer to being a physical sign than an abnormal arrhythmia</li> </ul>
<p><b>Defining Criteria per ECG</b> Key: PR interval &gt;0.20 sec</p>	<ul style="list-style-type: none"> <li>■ <b>Rate:</b> First-degree heart block can be seen with both sinus bradycardia and sinus tachycardia</li> <li>■ <b>Rhythm:</b> sinus, regular, both atria and ventricles</li> <li>■ <b>PR:</b> prolonged, &gt;0.20 sec, but does not vary (<i>fixed</i>)</li> <li>■ <b>P waves:</b> size and shape normal; every P wave is followed by a QRS complex; every QRS complex is preceded by a P wave</li> <li>■ <b>QRS complex:</b> narrow; ≤0.10 sec in absence of intraventricular conduction defect</li> </ul>
<p><b>Clinical Manifestations</b></p>	<ul style="list-style-type: none"> <li>■ Usually asymptomatic at rest</li> <li>■ Rarely, if bradycardia worsens, person may become symptomatic from the slow rate</li> </ul>
<p><b>Common Etiologies</b></p>	<ul style="list-style-type: none"> <li>■ Large majority of first-degree heart blocks are due to drugs, usually the AV nodal blockers: β-blockers, calcium channel blockers, and digoxin</li> <li>■ Any condition that stimulates the parasympathetic nervous system (eg, vasovagal reflex)</li> <li>■ Acute MIs that affect circulation to AV node (right coronary artery); most often inferior AMIs</li> </ul>
<p><b>Recommended Therapy</b></p>	<ul style="list-style-type: none"> <li>■ Treat only when patient has significant signs or symptoms that are due to the bradycardia</li> <li>■ Be alert to block deteriorating to second-degree, type I or type II block</li> <li>■ Oxygen is always appropriate</li> </ul> <p><b>Intervention sequence for symptomatic bradycardia</b></p> <ul style="list-style-type: none"> <li>■ <i>Atropine</i> 0.5 to 1 mg IV if vagal mechanism</li> <li>■ <i>Transcutaneous pacing</i> if available</li> </ul> <p><b>If signs and symptoms are severe, consider catecholamine infusions:</b></p> <ul style="list-style-type: none"> <li>■ <i>Dopamine</i> 5 to 20 µg/kg per min</li> <li>■ <i>Epinephrine</i> 2 to 10 µg/min</li> <li>■ <i>Isoproterenol</i> 2 to 10 µg/min</li> </ul>



First-degree AV block at rate of 37 bpm; PR interval 0.28 sec

## 18. Second-Degree Heart Block Type I (Mobitz I–Wenkebach)

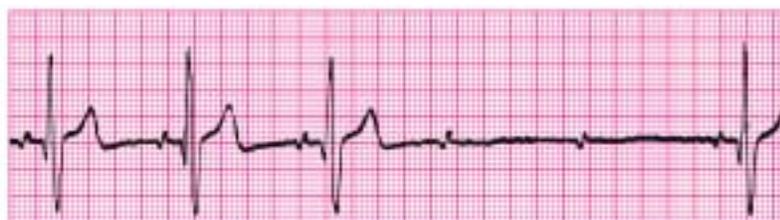
<b>Pathophysiology</b>	<ul style="list-style-type: none"> <li>■ Site of pathology: AV node</li> <li>■ AV node blood supply comes from branches of the right coronary artery</li> <li>■ Impulse conduction is increasingly slowed at the AV node (causing increasing PR interval)</li> <li>■ Until one sinus impulse is completely blocked and a QRS complex fails to follow</li> </ul>
<b>Defining Criteria per ECG</b> <b>Key:</b> There is progressive lengthening of the PR interval until one P wave is not followed by a QRS complex (the dropped beat)	<ul style="list-style-type: none"> <li>■ <b>Rate:</b> atrial rate just slightly faster than ventricular (because of dropped beats); usually normal range</li> <li>■ <b>Rhythm:</b> regular for atrial beats; irregular for ventricular (because of dropped beats); can show regular P waves marching through irregular QRS</li> <li>■ <b>PR:</b> progressive lengthening of the PR interval occurs from cycle to cycle; then one P wave is not followed by a QRS complex (the “dropped beat”)</li> <li>■ <b>P waves:</b> size and shape remain normal; occasional P wave not followed by a QRS complex (the “dropped beat”)</li> <li>■ <b>QRS complex:</b> <math>\leq 0.10</math> sec most often, but a QRS “drops out” periodically</li> </ul>
<b>Clinical Manifestations—Rate-Related</b>	<b>Due to bradycardia:</b> <ul style="list-style-type: none"> <li>■ <b>Symptoms:</b> chest pain, shortness of breath, decreased level of consciousness</li> <li>■ <b>Signs:</b> hypotension, shock, pulmonary congestion, CHF, angina</li> </ul>
<b>Common Etiologies</b>	<ul style="list-style-type: none"> <li>■ AV nodal blocking agents: <math>\beta</math>-blockers, calcium channel blockers, digoxin</li> <li>■ Conditions that stimulate the parasympathetic system</li> <li>■ An acute coronary syndrome that involves the <i>right</i> coronary artery</li> </ul>
<b>Recommended Therapy</b> <b>Key:</b> Treat only when patient has significant signs or symptoms that are due to the bradycardia	<b>Intervention sequence for symptomatic bradycardia:</b> <ul style="list-style-type: none"> <li>■ <i>Atropine</i> 0.5 to 1 mg IV if vagal mechanism</li> <li>■ <i>Transcutaneous pacing</i> if available</li> </ul> <b>If signs and symptoms are severe, consider catecholamine infusions:</b> <ul style="list-style-type: none"> <li>■ <i>Dopamine</i> 5 to 20 <math>\mu\text{g}/\text{kg}</math> per min</li> <li>■ <i>Epinephrine</i> 2 to 10 <math>\mu\text{g}/\text{min}</math></li> <li>■ <i>Isoproterenol</i> 2 to 10 <math>\mu\text{g}/\text{min}</math></li> </ul>



Second-degree heart block type I. Note progressive lengthening of PR interval until one P wave (arrow) is not followed by a QRS.

19. Second-Degree Heart Block Type II (Infranodal) (Mobitz II–Non-Wenkebach)

<p><b>Pathophysiology</b></p>	<ul style="list-style-type: none"> <li>■ The pathology, ie, the site of the block, is most often <i>below</i> the AV node (infranodal); at the bundle of His (infrequent) or at the bundle branches</li> <li>■ Impulse conduction is normal through the node, thus no first-degree block and no prior PR prolongation</li> </ul>
<p><b>Defining Criteria per ECG</b></p>	<ul style="list-style-type: none"> <li>■ <b>Atrial Rate:</b> usually 60-100 beats/min</li> <li>■ <b>Ventricular rate:</b> by definition (due to the blocked impulses) slower than atrial rate</li> <li>■ <b>Rhythm:</b> atrial = regular; ventricular = irregular (because of blocked impulses)</li> <li>■ <b>PR:</b> constant and set; no progressive prolongation as with type I—a distinguishing characteristic.</li> <li>■ <b>P waves:</b> typical in size and shape; by definition some P waves will not be followed by a QRS complex</li> <li>■ <b>QRS complex:</b> narrow (<math>\leq 0.10</math> sec) implies high block relative to the AV node; wide (<math>&gt; 0.12</math> sec) implies low block relative to the AV node</li> </ul>
<p><b>Clinical Manifestations—Rate-Related</b></p>	<p><b>Due to bradycardia:</b></p> <ul style="list-style-type: none"> <li>■ <b>Symptoms:</b> chest pain, shortness of breath, decreased level of consciousness</li> <li>■ <b>Signs:</b> hypotension, shock, pulmonary congestions, CHF, acute MI</li> </ul>
<p><b>Common Etiologies</b></p>	<ul style="list-style-type: none"> <li>■ An acute coronary syndrome that involves branches of the <i>left</i> coronary artery</li> </ul>
<p><b>Recommended Therapy</b>  <b>Pearl:</b> New onset type II second-degree heart block in clinical context of acute coronary syndrome is indication for transvenous pacemaker insertion</p>	<p><b>Intervention sequence for bradycardia due to type II second-degree or third-degree heart block:</b></p> <ul style="list-style-type: none"> <li>■ Prepare for <i>transvenous</i> pacer</li> <li>■ Atropine is seldom effective for infranodal block</li> <li>■ Use <i>transcutaneous pacing</i> if available as a bridge to transvenous pacing (verify patient tolerance and mechanical capture. Use sedation and analgesia as needed.)</li> </ul> <p><b>If signs/symptoms are severe and unresponsive to TCP, and transvenous pacing is delayed, consider catecholamine infusions:</b></p> <ul style="list-style-type: none"> <li>■ <i>Dopamine</i> 5 to 20 <math>\mu\text{g}/\text{kg}</math> per min</li> <li>■ <i>Epinephrine</i> 2 to 10 <math>\mu\text{g}/\text{min}</math></li> <li>■ <i>Isoproterenol</i> 2 to 10 <math>\mu\text{g}/\text{min}</math></li> </ul>



Type II (high block): regular PR-QRS intervals until 2 dropped beats occur; borderline normal QRS complexes indicate high nodal or nodal block



Type II (low block): regular PR-QRS intervals until dropped beats; wide QRS complexes indicate infranodal block

## 20. Third-Degree Heart Block and AV Dissociation

<p><b>Pathophysiology</b>  <b>Pearl:</b> <i>AV dissociation</i> is the defining class; <i>third-degree</i> or <i>complete heart block</i> is one type of AV dissociation. By convention (outdated): if ventricular escape depolarization is faster than atrial rate = “<i>AV dissociation</i>”; if slower = “<i>third-degree heart block</i>”</p>	<p>Injury or damage to the cardiac conduction system so that no impulses (<i>complete block</i>) pass between atria and ventricles (neither antegrade nor retrograde)</p> <p>This complete block can occur at several different anatomic areas:</p> <ul style="list-style-type: none"> <li>■ AV node (“high” or “supra” or “junctional” <i>nodal block</i>)</li> <li>■ Bundle of His</li> <li>■ Bundle branches (“low-nodal” or “infranodal” block)</li> </ul>
<p><b>Defining Criteria per ECG</b>  <b>Key:</b> The third-degree block (see pathophysiology) causes the atria and ventricles to depolarize independently, with no relationship between the two (AV dissociation)</p>	<ul style="list-style-type: none"> <li>■ <b>Atrial rate:</b> usually 60-100 beats/min; impulses completely independent (“dissociated”) from ventricular rate</li> <li>■ <b>Ventricular rate:</b> depends on rate of the ventricular escape beats that arise: <ul style="list-style-type: none"> <li>— Ventricular escape beat rate slower than atrial rate = third-degree heart block (20-40 beats/min)</li> <li>— Ventricular escape beat rate faster than atrial rate = AV dissociation (40-55 beats/min)</li> </ul> </li> <li>■ <b>Rhythm:</b> both atrial rhythm and ventricular rhythm are regular but independent (“dissociated”)</li> <li>■ <b>PR:</b> by definition there is no relationship between P wave and R wave</li> <li>■ <b>P waves:</b> typical in size and shape</li> <li>■ <b>QRS complex:</b> narrow (<math>\leq 0.10</math> sec) implies high block relative to the AV node; wide (<math>&gt; 0.12</math> sec) implies low block relative to the AV node</li> </ul>
<p><b>Clinical Manifestations—Rate-Related</b></p>	<p><b>Due to bradycardia:</b></p> <ul style="list-style-type: none"> <li>■ <b>Symptoms:</b> chest pain, shortness of breath, decreased level of consciousness</li> <li>■ <b>Signs:</b> hypotension, shock, pulmonary congestions, CHF, acute MI</li> </ul>
<p><b>Common Etiologies</b></p>	<ul style="list-style-type: none"> <li>■ An acute coronary syndrome that involves branches of the <i>left</i> coronary artery</li> <li>■ In particular, the LAD (left anterior descending) and branches to the interventricular septum (supply bundle branches)</li> </ul>
<p><b>Recommended Therapy</b>  <b>Pearl:</b> New onset third-degree heart block in clinical context of acute coronary syndrome is indication for transvenous pacemaker insertion  <b>Pearl:</b> <i>Never treat third-degree heart block plus ventricular escape beats with lidocaine</i></p>	<p><b>Intervention sequence for bradycardia due to type II second-degree or third-degree heart block:</b></p> <ul style="list-style-type: none"> <li>■ Prepare for <i>transvenous</i> pacer</li> <li>■ Use <i>transcutaneous pacing</i> if available as a bridge to transvenous pacing (verify patient tolerance and mechanical capture; use sedation and analgesia as needed)</li> </ul> <p><b>If signs/symptoms are severe and unresponsive to TCP, and transvenous pacing is delayed, consider catecholamine infusions:</b></p> <ul style="list-style-type: none"> <li>■ <i>Dopamine</i> 5 to 20 <math>\mu\text{g}/\text{kg}</math> per min</li> <li>■ <i>Epinephrine</i> 2 to 10 <math>\mu\text{g}/\text{min}</math></li> <li>■ <i>Isoproterenol</i> 2 to 10 <math>\mu\text{g}/\text{min}</math></li> </ul>



Third-degree heart block: regular P waves at 50 to 55 bpm; regular ventricular “escape beats” at 35 to 40 bpm; no relationship between P waves and escape beats

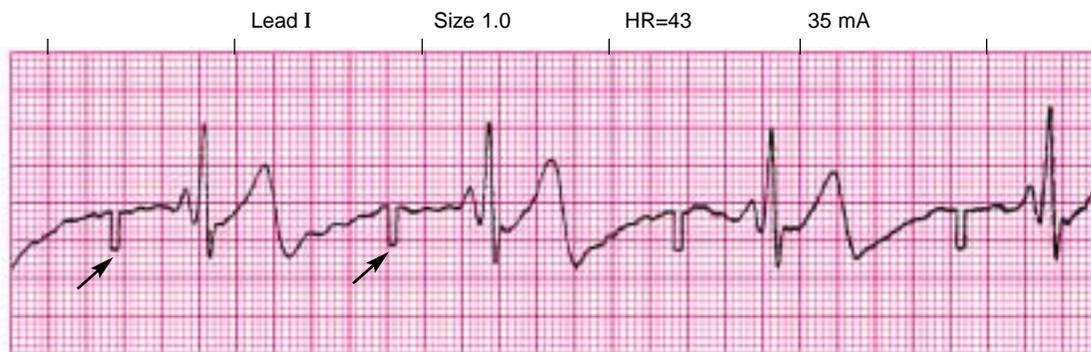
21. Transcutaneous Pacing

- A. Bradycardia: no pacing
- B. Pacing stimulus below threshold: no capture
- C. Pacing stimulus above threshold: capture occurs

Rhythm Strip	Comments
<p><b>A. Bradycardia (third-degree heart block): no pacing</b>                      (Note: Rates and intervals slightly altered due to monitor compensation for pacing stimulus)</p>	<ul style="list-style-type: none"> <li>■ QRS rate = 41 beats/min</li> <li>■ P waves seen = 125 beats/min</li> <li>■ QRS = very wide, 0.24 sec; ventricular escape beats</li> <li>■ QRS and T wave polarity = both positive</li> <li>■ Patient: SOB at rest; severe SOB with walking; near syncope</li> </ul>
<p><b>B. Transcutaneous pacing initiated at low current (35 mA) and slow rate (50 beats/min).</b>                      Below the threshold current needed to stimulate the myocardium</p>	<ul style="list-style-type: none"> <li>■ With TCP, monitor electrodes are attached in modified lead II position</li> <li>■ As current (in milliamperes) is gradually increased, the monitor leads detect the pacing stimuli as a squared off, negative marker</li> <li>■ TC pacemakers incorporate standard ECG monitoring circuitry but incorporate filters to dampen the pacing stimuli</li> <li>■ A monitor without these filters records “border-to-border” tracings (off the edge of the screen or paper at the top and bottom borders) that cannot be interpreted</li> </ul>
<p><b>C. Pacing current turned up above threshold (60 mA at 71 beats/min) and “captures” the myocardium</b></p>	<ul style="list-style-type: none"> <li>■ TCP stimulus does not work through the normal cardiac conduction system but by a direct electrical stimulus of the myocardium</li> <li>■ Therefore, a “capture,” where TCP stimulus results in a myocardial contraction, will resemble a PVC</li> <li>■ Electrical capture is characterized by a wide QRS complex, with the initial deflection and the terminal deflection <i>a/ways</i> in opposite directions</li> <li>■ A “mechanically captured beat” will produce effective myocardial contraction with production of some blood flow (usually assessed by a palpable carotid pulse)</li> </ul>



**Bradycardia: prepacing attempt**



**Pacing attempted: note pacing stimulus indicator (arrow) which is below threshold; no capture**



**Pacing above threshold (60 mA): with capture (QRS complex broad and ventricular; T wave opposite QRS)**