ACLS / BLS
Rhythm Strips

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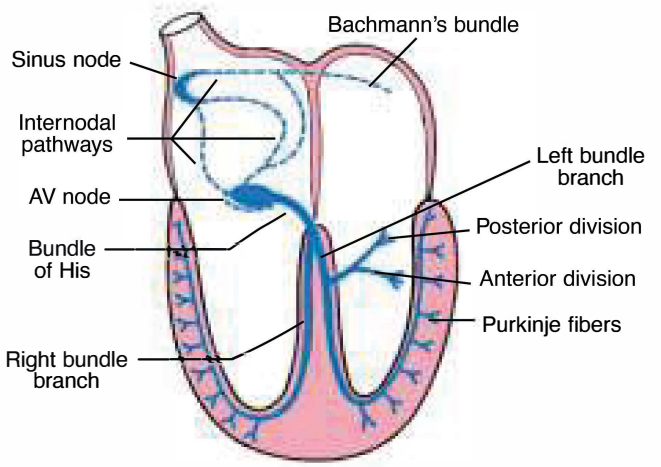
Healthcare Certifications

January 2024

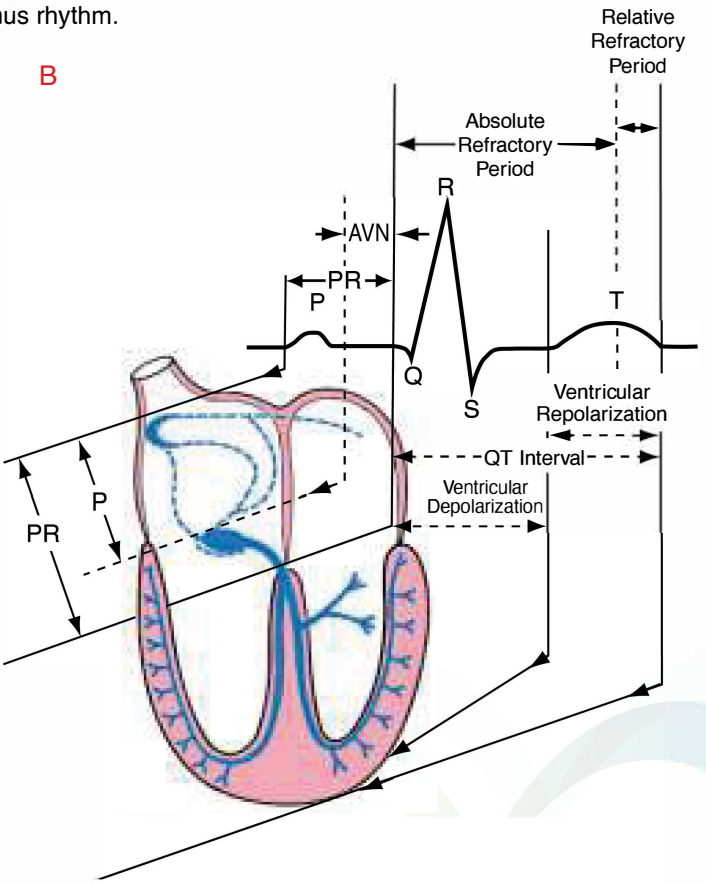
The Basics

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.

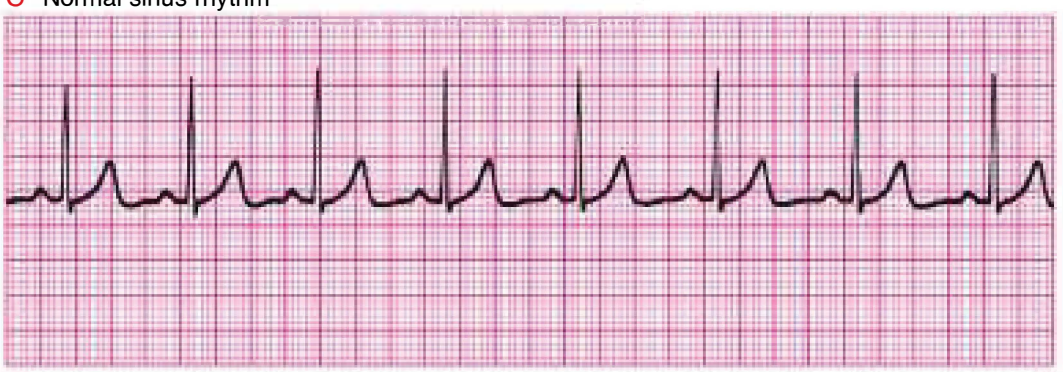
A



B

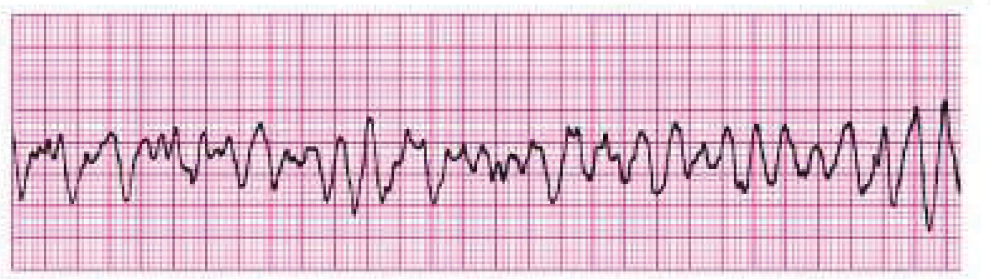


C Normal sinus rhythm

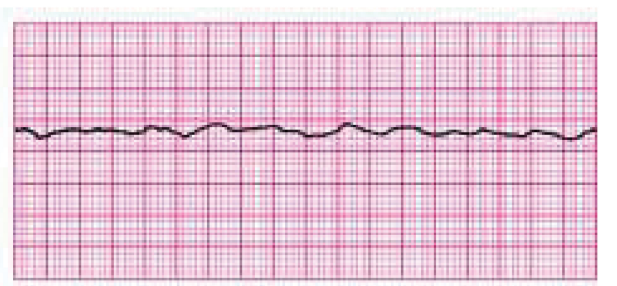


Ventricular Fibrillation/Pulseless Ventricular Tachycardia

Pathophysiology	Ventricles consist of areas of normal myocardium alternating with areas of ischemic, injured, or infarcted myocardium, leading to chaotic pattern of ventricular depolarization
Defining Criteria per ECG	**Rate/QRS complex:** unable to determine; no recognizable P, QRS, or T waves **Rhythm:** indeterminate; pattern of sharp up (peak) and down (trough) deflections **Amplitude:** measured from peak-to-trough; often used subjectively to describe VF as *fine* (peak-to-trough 2 to <5 mm), *medium-moderate* (5 to <10 mm), coarse (10 to <15 mm), *very coarse* (>15 mm)
Clinical Manifestations	Pulse disappears with onset of VF Collapse, unconsciousness Agonal breaths apnea in <5 min Onset of *reversible death*
Common Etiologies	Acute coronary syndromes leading to ischemic areas of myocardium Stable-to-unstable VT, untreated PVCs with R-on-T phenomenon Multiple drug, electrolyte, or acid-base abnormalities that prolong the relative refractory period Primary or secondary QT prolongation Electrocution, hypoxia, many others
Recommended Therapy *Comprehensive ECC algorithm, page 10; VF/pulseless VT algorithm, page 77*	Early defibrillation is essential Agents given to prolong period of reversible death (*oxygen,* CPR, intubation, *epinephrine, vasopressin*) Agents given to prevent refibrillation after a shock causes defibrillation *(lidocaine, amiodarone, procainamide, β-blockers)* Agents given to adjust metabolic milieu *(sodium bicarbonate, magnesium)*



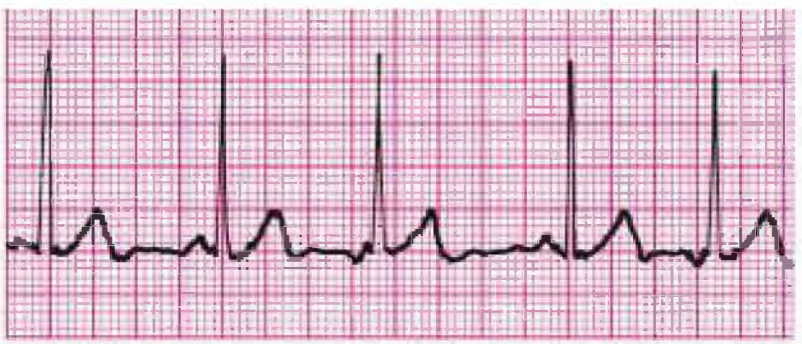
Coarse VF



Fine VF

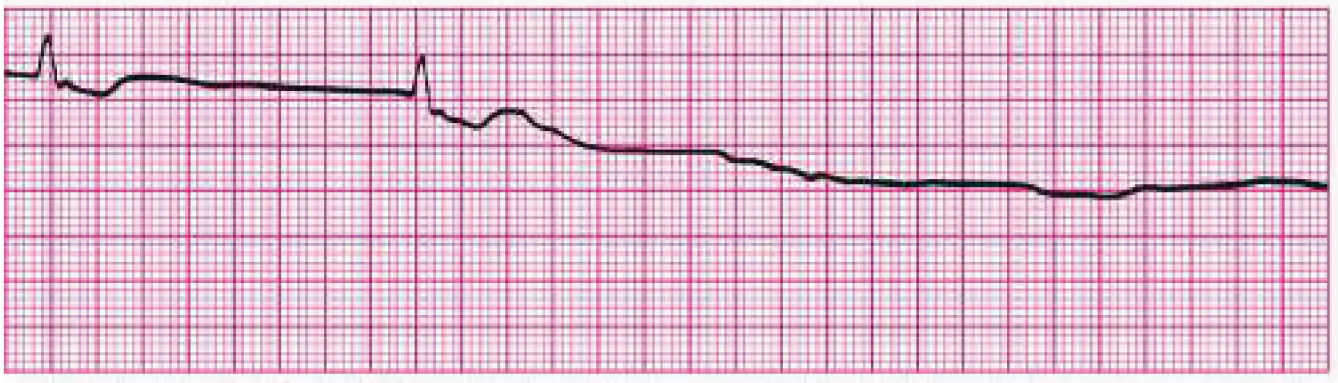
3. PEA (Pulseless Electrical Activity)

Pathophysiology	▯ 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
Defining Criteria per ECG	▯ Rhythm displays organized electrical activity (not VF/pulseless VT) ▯ Seldom as organized as normal sinus rhythm ▯ Can be narrow (QRS <0.10 mm) or wide (QRS >0.12 mm); fast (>100 beats/min) or slow (<60 beats/min) ▯ Most frequently: fast and narrow (noncardiac etiology) or slow and wide (cardiac etiology)
Clinical Manifestations	▯ Collapse; unconscious ▯ Agonal respirations or apnea ▯ No pulse detectable by arterial palpation (thus could still be as high as 50-60 mm Hg; in such cases termed *pseudo-PEA*)
Common Etiologies	*Mnemonic of 5 H's and 5 T's aids recall:* ▯ Hypovolemia ▯ Hypoxia ▯ Hydrogen ion—acidosis ▯ Hyperkalemia/Hypokalemia ▯ Hypothermia ▯ "Tablets" (drug OD, ingestions) ▯ Tamponade, cardiac ▯ Tension pneumothorax ▯ Thrombosis, coronary (ACS) ▯ Thrombosis, pulmonary (embolism)
Recommended Therapy *Comprehensive ECC Algorithm, page 10; PEA Algorithm, page 100*	▯ Per PEA algorithm ▯ Primary ABCD (basic CPR) ▯ Secondary **AB** (advanced airway and ventilation); **C** (IV, *epinephrine, atropine* if electrical activity <60 complexes per minute); **D** (identify and treat reversible causes) ▯ **Key:** identify and treat a reversible cause of the PEA



Asystole

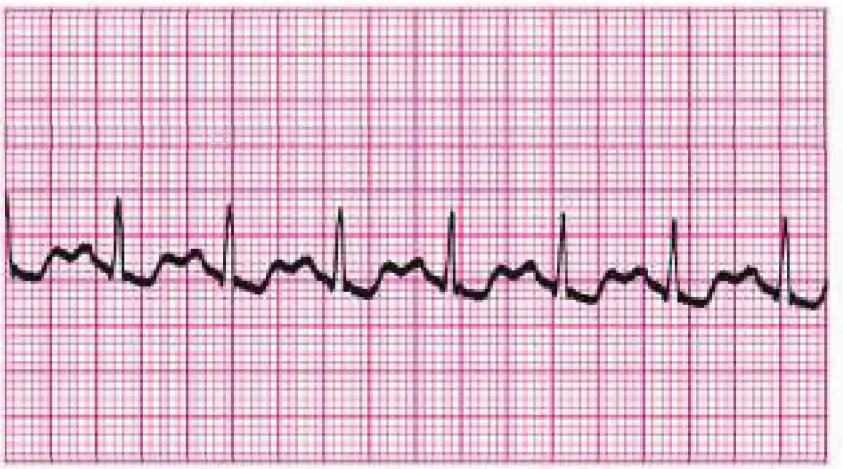
Defining Criteria per ECG Classically *asystole* presents as a "flat line"; any defining criteria are virtually nonexistent	**Rate:** no ventricular activity seen or ≤6/min; so-called "P-wave asystole" occurs with only atrial impulses present to form P waves **Rhythm:** no ventricular activity seen; or ≤6/min **PR:** cannot be determined; occasionally P wave seen, but by definition R wave must be absent **QRS complex:** no deflections seen that are consistent with a QRS complex
Clinical Manifestations	Early may see agonal respirations; unconscious; unresponsive No pulse; no blood pressure Cardiac arrest
Common Etiologies	End of life (death) Ischemia/hypoxia from many causes Acute respiratory failure (no oxygen; apnea; asphyxiation) Massive electrical shock: electrocution; lightning strike Postdefibrillatory shocks
Recommended Therapy *Comprehensive ECC Algorithm, page 10; Asystole Algorithm, page 112*	Always check for DNR status Primary ABCD survey (basic CPR) Secondary ABCD survey



Asystole: agonal complexes too slow to make this rhythm "PEA"

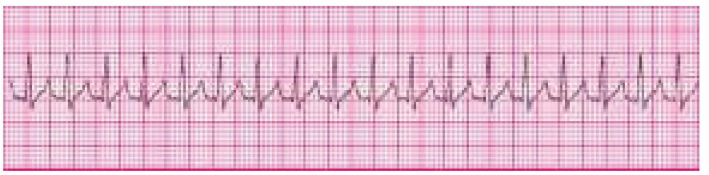
5. Sinus Tachycardia

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

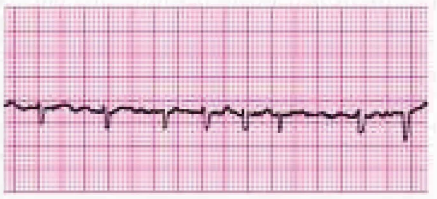


Sinus tachycardia

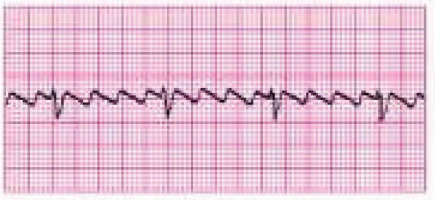
Rhythmic Algorithm No. 1: Tachycardias Overview



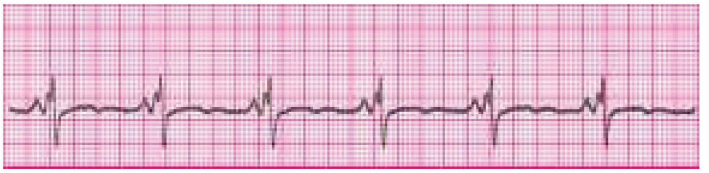
Tachycardia



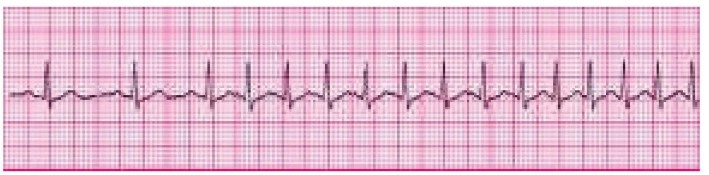
Atrial fibrillation



Atrial flutter



Sinus rhythm with WPW syndrome



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

Evaluate patient
- Is patient stable or unstable?
- Are there serious signs or symptoms?
- Are signs and symptoms due to tachycardia?

Stable

Stable patient: no serious signs or symptoms
- Initial assessment identifies 1 of 4 types of tachycardias

1. Atrial fibrillation Atrial flutter

2. Narrow-complex tachycardias

Evaluation focus, 4 clinical features:
1. Patient clinically unstable?
2. Cardiac function impaired?
3. WPW present?
4. Duration <48 or >48 hours?

Attempt to establish a specific diagnosis
- 12-lead ECG
- Clinical information
- Vagal maneuvers
- Adenosine

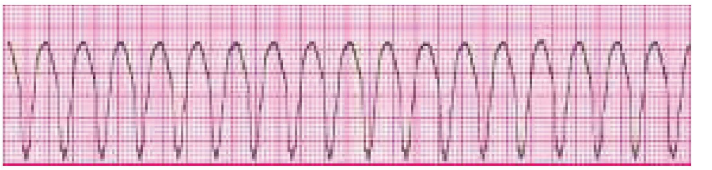
Treatment focus: clinical evaluation
1. Treat unstable patients urgently
2. Control the rate
3. Convert the rhythm
4. Provide anticoagulation

Diagnostic efforts yield
- Ectopic atrial tachycardia
- Multifocal atrial tachycardia
- Paroxysmal supraventricular tachycardia (PSVT)

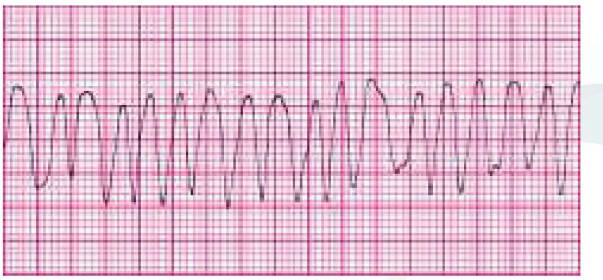
Treatment of atrial fibrillation/ atrial flutter (See following table)

Treatment of SVT (See narrow-complex tachycardia algorithm)

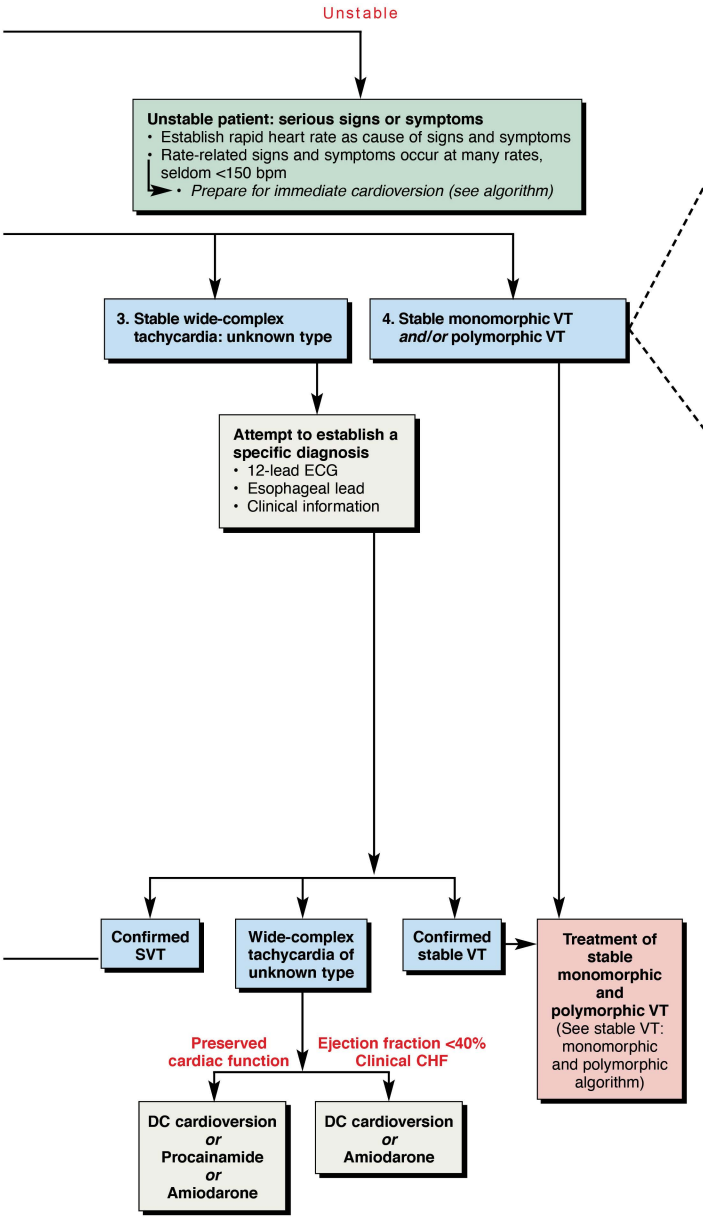
Rhythmic Algorithm No. 2: Tachycardias Overview



Monomorphic ventricular tachycardia

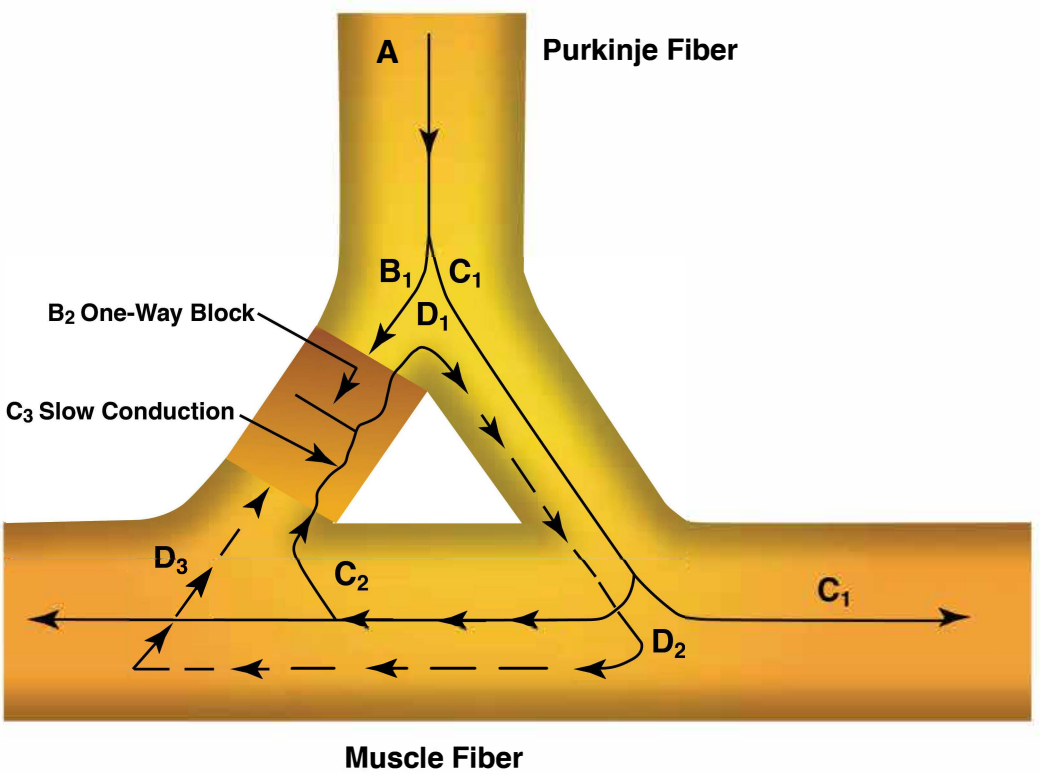


Polymorphic ventricular tachycardia



6. Re-entry 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

<table>
<tr><td>Pathophysiology</td><td colspan="3">

Atrial impulses faster than SA node impulses
Atrial fibrillation impulses take multiple, chaotic, random pathways through the atria
Atrial flutter impulses take a circular course around the atria, setting up the flutter waves
Mechanism of impulse formation: reentry

</td></tr>
<tr><td rowspan="5">Defining Criteria and ECG Features
(Distinctions here between atrial fibrillation vs atrial flutter; all other characteristics are the same)
Atrial Fibrillation Key: A classic clinical axiom: “Irregularly irregular rhythm—with variation in both interval and amplitude from R wave to R wave—is always atrial fibrillation.” This one is dependable.
Atrial Flutter Key: Flutter waves seen in classic “sawtooth pattern”</td><td></td><td>Atrial Fibrillation</td><td>Atrial Flutter</td></tr>
<tr><td>Rate</td><td>

Wide-ranging ventricular response to atrial rate of 300-400 beats/min

</td><td>

Atrial rate 220-350 beats/min
Ventricular response = a function of AV node block or conduction of atrial impulses
Ventricular response rarely >150-180 beats because of AV node conduction limits

</td></tr>
<tr><td>Rhythm</td><td>

Irregular (classic “irregularly irregular”)

</td><td>

Regular (unlike atrial fibrillation)
Ventricular rhythm often regular
Set ratio to atrial rhythm, eg, 2-to-1 or 3-to-1

</td></tr>
<tr><td>P waves</td><td>

Chaotic atrial fibrillatory waves only
Creates disturbed baseline

</td><td>

No true P waves seen
Flutter waves in “sawtooth pattern” is classic

</td></tr>
<tr><td>PR</td><td colspan="2">

Cannot be measured

</td></tr>
<tr><td>QRS</td><td colspan="2">

Remains ≤0.10-0.12 sec unless QRS complex distorted by fibrillation/flutter waves or by conduction defects through ventricles

</td></tr>
<tr><td>Clinical Manifestations</td><td colspan="3">

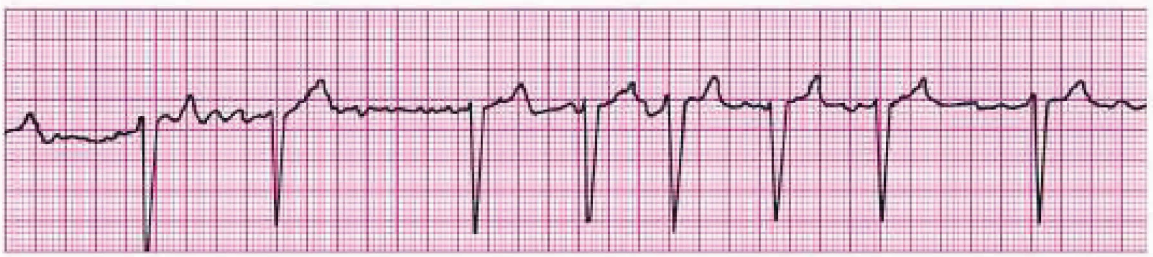
Signs and symptoms are function of the rate of ventricular response to atrial fibrillatory waves; “atrial fibrillation with rapid ventricular response” DOE, SOB, acute pulmonary edema
Loss of “atrial kick” may lead to drop in cardiac output and decreased coronary perfusion
Irregular rhythm often perceived as “palpitations”
Can be asymptomatic

</td></tr>
</table>

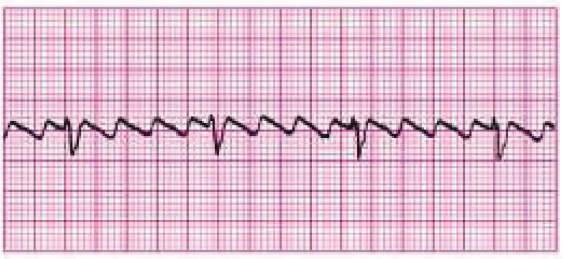
7. Atrial Fibrillation/Atrial Flutter (continued)

<table>
<tr><th colspan="2">Recommended Therapy</th><th colspan="2">Control Rate</th></tr>
<tr><th>Evaluation Focus:</th><th>Treatment Focus:</th><th>Normal Heart</th><th>Impaired Heart</th></tr>
<tr><td rowspan="4">1. Patient clinically unstable?
2. Cardiac function impaired?
3. WPW present?
4. Duration ≤48 or >48 hr?</td><td rowspan="4">1. Treat unstable patients urgently
2. Control the rate
3. Convert the rhythm
4. Provide anticoagulation</td><td>▯ Diltiazem or another calcium channel blocker or metoprolol or another β-blocker</td><td>▯ Digoxin or diltiazem or amiodarone</td></tr>
<tr><td colspan="2">Convert Rhythm</td></tr>
<tr><td>Impaired Heart</td><td>Normal Heart</td></tr>
<tr><td>▯ If ≤48 hours:
— DC cardioversion or amiodarone or others
▯ If >48 hours:
— Anticoagulate × 3 wk, then
— DC cardioversion, then
— Anticoagulate × 4 wk
or
▯ IV heparin and TEE to rule out atrial clot, then
▯ DC cardioversion within 24 hours, then
▯ Anticoagulation × 4 more wk</td><td>▯ If ≤48 hours:
— DC Cardioversion or amiodarone
▯ If >48 hours:
— Anticoagulate × 3 wk, then
— DC cardioversion, then
— Anticoagulate × 4 more wk</td></tr>
</table>

TEE indicates transesophageal echocardiogram.



Atrial fibrillation



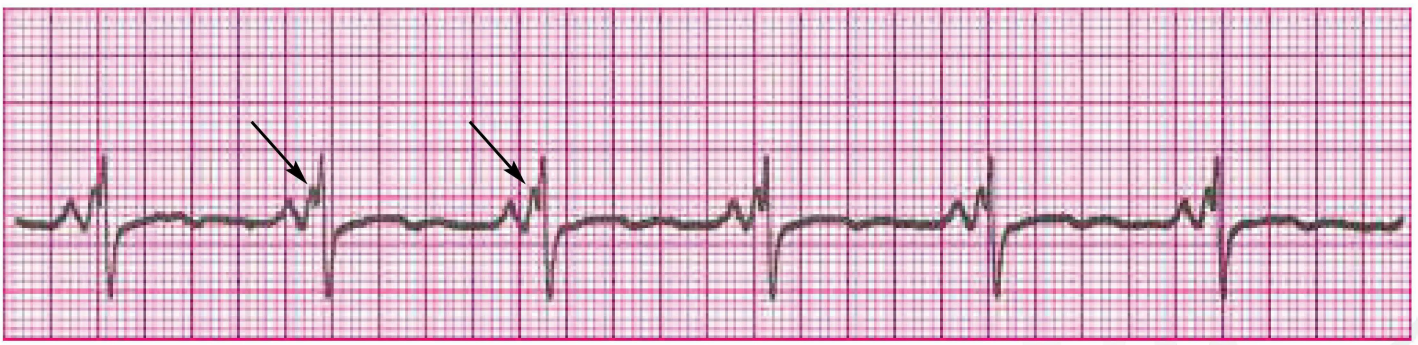
Atrial flutter

8. WPW (Wolff-Parkinson-White) Syndrome

Pathophysiology	▯ The prototypical **pre-excitation syndrome:** congenital mal-formation; strands of conducting myocardial tissue between atria and ventricles ▯ When persistent after birth strands can form an accessory pathway (eg, bundle of Kent)
Defining Criteria and ECG Features **Key: QRS complex** is classically distorted by delta wave (upwards deflection of QRS is slurred)	▯ **Rate:** most often 60-100 beats/min as usual rhythm is sinus ▯ **Rhythm:** normal sinus except during pre-excitation tachycardia ▯ **PR:** shorter since conduction through accessory pathway is faster than through AV node ▯ **P waves:** normal conformation ▯ **QRS complex:** classically distorted by delta wave (upwards deflection of QRS is slurred)
Clinical Manifestations	▯ A person with WPW may never have symptoms ▯ People with WPW have same annual incidence of atrial fibrillation as age- and gender-matched population ▯ Onset of atrial fibrillation for WPW patients, however, poses risk of rapid ventricular response through the accessory pathway ▯ This rapid ventricular response can lead to all signs and symptoms of stable and unstable tachycardias
Common Etiology	▯ The accessory pathway in WPW is a congenital malformation

WPW (Wolff-Parkinson-White) Syndrome (continued)

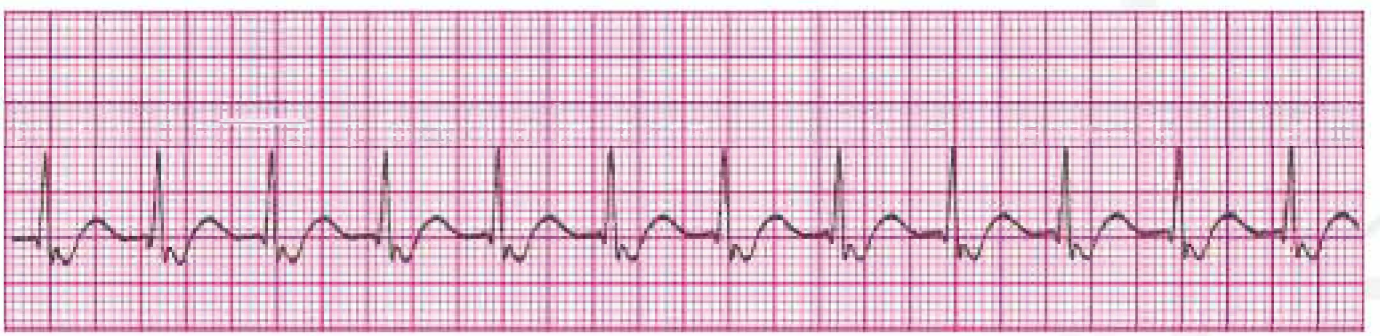
<table>
<tr><th colspan="2">Recommended Therapy</th><th colspan="2">Wolff-Parkinson-White: Control Rate</th></tr>
<tr><th>Evaluation Focus</th><th>Treatment Focus</th><th>Normal Heart</th><th>Impaired Heart</th></tr>
<tr><td>1. Patient clinically unstable?
2. Cardiac function impaired?
3. WPW present?
4. Duration ≤48 or >48 hr?</td><td>1. Treat unstable patients urgently
2. Control the rate
3. Convert the rhythm
4. Provide anticoagulation</td><td>▯ Cardioversion or
▯ Antiarrhythmic (IIb): amiodarone or flecainide or procainamide or propafenone or sotalol</td><td>▯ Cardioversion
or
▯ Amiodarone</td></tr>
<tr><td colspan="2" rowspan="2">Class III (can be harmful) in treating atrial fibrillation with WPW:
▯ Adenosine
▯ β-Blockers
▯ Calcium channel blockers
▯ Digoxin</td><td colspan="2">Wolff-Parkinson-White: Convert Rhythm</td></tr>
<tr><td>Duration ≤48 Hours</td><td>Duration >48 Hours</td></tr>
<tr><td colspan="2"></td><td>▯ Cardioversion or
▯ Antiarrhythmic (IIb): amiodarone or flecainide or procainamide or propafenone or sotalol
If impaired heart: cardioversion or amiodarone</td><td>▯ Anticoagulate × 3 wk
then
▯ DC cardioversion
then
▯ Anticoagulate × 4 wk</td></tr>
</table>



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

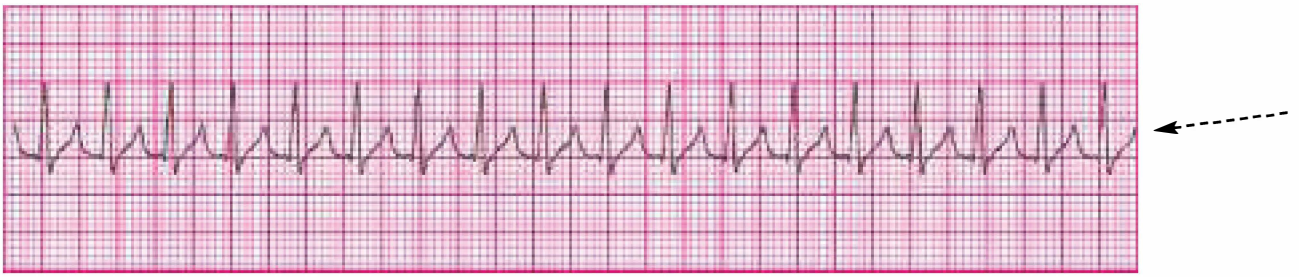
9. Junctional Tachycardia

Pathophysiology	▯ Area of *automaticity* (automatic impulse formation) develops in the AV node ("junction") ▯ Both retrograde and antegrade transmission occurs
Defining Criteria and ECG Features ▯ **Key:** 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	▯ **Rate:** 100-180 beats/min ▯ **Rhythm:** regular atrial and ventricular firing ▯ **PR:** often not measurable unless P wave comes before QRS; then will be short (<0.12 secs) ▯ **P waves:** often obscured; may propagate antegrade or retrograde with origin at the junction; may arise before, after, or with the QRS ▯ **QRS complex:** narrow; ≤0.10 secs in absence of intraventricular conduction defect
Clinical Manifestations	▯ Patients may have clinical signs of a reduced ejection fraction because augmented flow from atrium is lost ▯ Symptoms of unstable tachycardia may occur
Common Etiologies	▯ Digoxin toxicity ▯ Acute sequelae of acute coronary syndromes
Recommended Therapy If specific diagnosis unknown, attempt therapeutic/diagnostic maneuver with ▯ Vagal stimulation ▯ *Adenosine* . . . THEN →	**Preserved heart function:** ▯ *β-Blocker* ▯ *Calcium channel blocker* ▯ *Amiodarone* ▯ **NO DC cardioversion!** **If impaired heart function:** ▯ *Amiodarone* ▯ **NO DC cardioversion!**

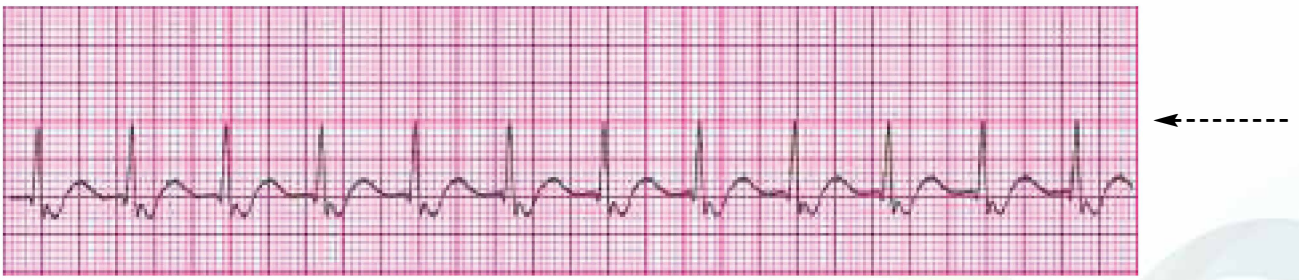


Junctional tachycardia: narrow QRS complexes at 130 bpm; P waves arise with QRS

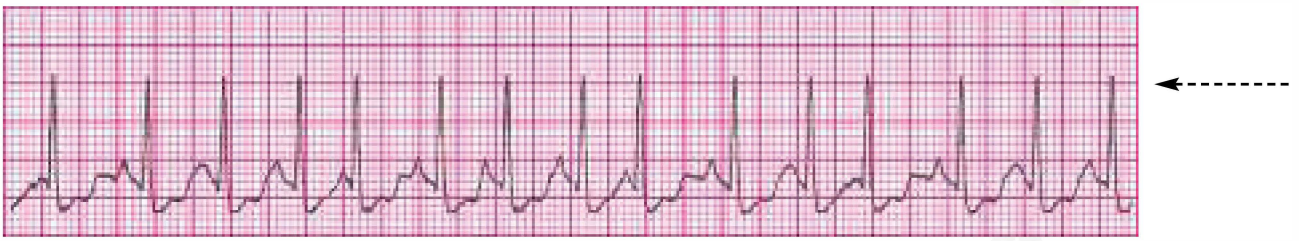
Rhythmic Algorithm No. 2: Narrow-Complex Tachycardias



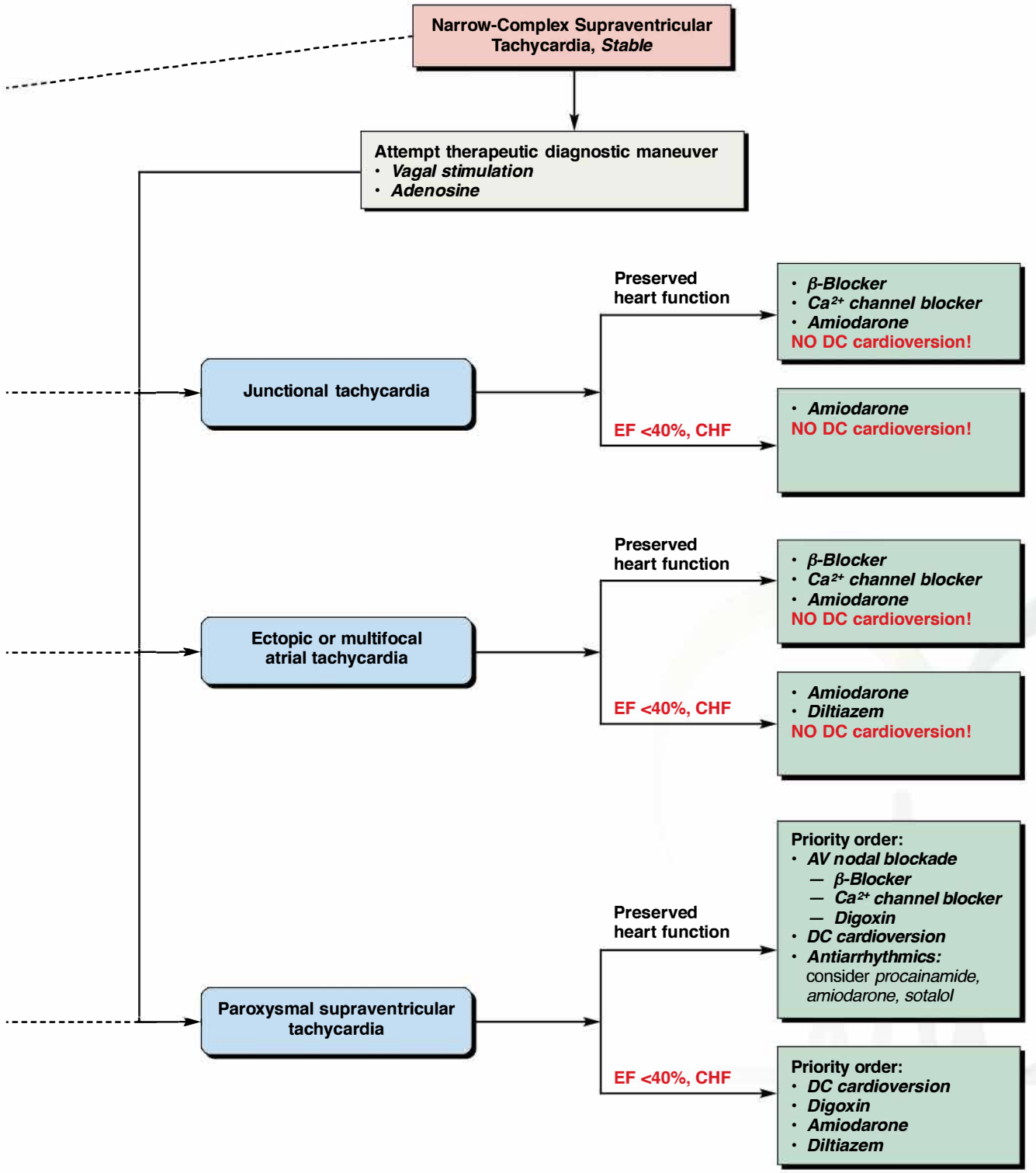
Supraventricular tachycardia



Junctional tachycardia

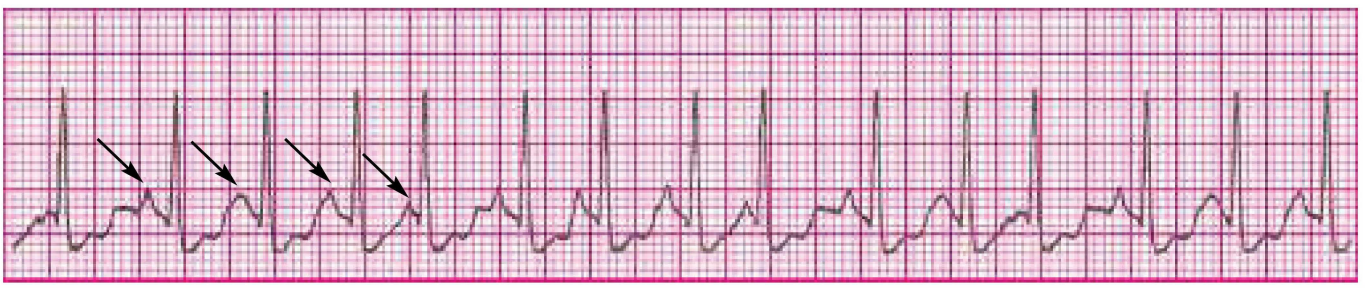


Multifocal atrial tachycardia


Narrow-Complex Supraventricular Tachycardia, *Stable*
Attempt therapeutic diagnostic maneuver
• *Vagal stimulation*
• *Adenosine*
Junctional tachycardia
Preserved heart function
• *β-Blocker*
• *Ca²⁺ channel blocker*
• *Amiodarone*
NO DC cardioversion!
EF <40%, CHF
• *Amiodarone*
NO DC cardioversion!
Ectopic or multifocal atrial tachycardia
Preserved heart function
• *β-Blocker*
• *Ca²⁺ channel blocker*
• *Amiodarone*
NO DC cardioversion!
EF <40%, CHF
• *Amiodarone*
• *Diltiazem*
NO DC cardioversion!
Paroxysmal supraventricular tachycardia
Preserved heart function
Priority order:
• *AV nodal blockade*
— *β-Blocker*
— *Ca²⁺ channel blocker*
— *Digoxin*
• *DC cardioversion*
• *Antiarrhythmics:* consider *procainamide, amiodarone, sotalol*
EF <40%, CHF
Priority order:
• *DC cardioversion*
• *Digoxin*
• *Amiodarone*
• *Diltiazem*

10. Multifocal Atrial Tachycardia

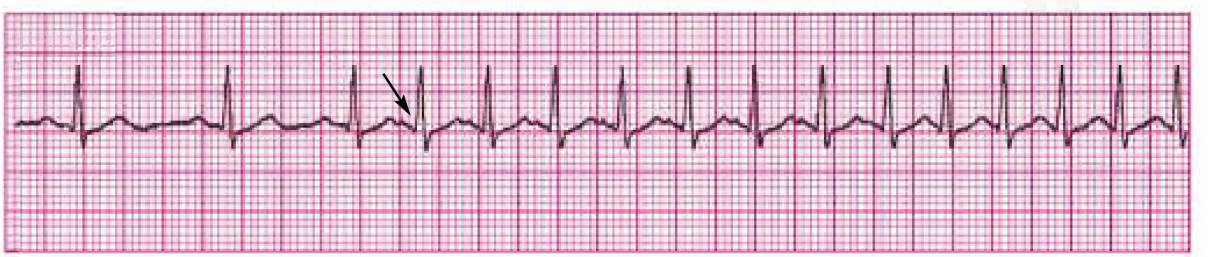
Pathophysiology	▯ Areas of *automaticity* (impulse formation) originate irregularly and rapidly at different points in the atria
Defining Criteria and ECG Features If the rate is <100 beats/min, this rhythm is termed *"wandering atrial pacemaker"* or *"multifocal atrial rhythm"* **Key:** By definition 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.	▯ **Rate:** >100 beats/min; usually >130 bpm ▯ **Rhythm:** irregular atrial firing ▯ **PR:** variable ▯ **P waves:** 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 ▯ **QRS complex:** narrow; ≤0.10 sec in absence of intraventricular conduction defect
Clinical Manifestations	▯ Patients may have no clinical signs ▯ Symptoms of unstable tachycardia may occur
Common Etiologies	▯ Most common cause is COPD *(cor pulmonale)* where pulmonary hypertension places increased strain on the right ventricle and atrium ▯ Impaired and hypertrophied atrium gives rise to automaticity ▯ Also digoxin toxicity, rheumatic heart disease, acute coronary syndromes
Recommended Therapy If specific diagnosis unknown, attempt therapeutic/diagnostic maneuver with ▯ Vagal stimulation ▯ *Adenosine* . . . THEN ➡	**Preserved heart function:** ▯ *β-blocker* ▯ *Calcium channel blocker* ▯ *Amiodarone* ▯ **NO DC cardioversion!** **If impaired heart function:** ▯ *Amiodarone* ▯ *Diltiazem* ▯ **NO DC cardioversion!**



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

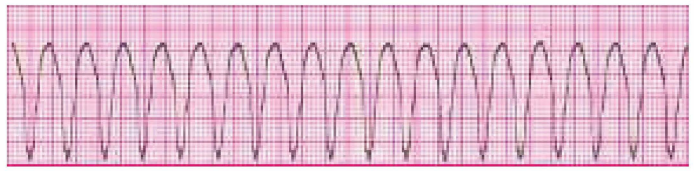
11. PSVT (Paroxysmal Supraventricular Tachycardia)

Pathophysiology	▯ **Reentry phenomenon** (see page 260)**:** impulses arise and recycle repeatedly in the AV node because of areas of unidirectional block in the Purkinje fibers
Defining Criteria and ECG Features **Key:** Regular, narrow-complex tachycardia without P-waves, and sudden, *paroxysmal* onset or cessation, or both **Note:** To merit the diagnosis some experts require capture of the paroxysmal onset or cessation on a monitor strip	▯ **Rate:** exceeds upper limit of sinus tachycardia (>120 beats/min); seldom <150 beats/min; up to 250 beats/min ▯ **Rhythm:** regular ▯ **P waves:** seldom seen because rapid rate causes P wave loss in preceding T waves or because the origin is low in the atrium ▯ **QRS complex:** normal, narrow (≤0.10 sec usually)
Clinical Manifestations	▯ Palpitations felt by patient at the paroxysmal onset; becomes anxious, uncomfortable ▯ Exercise tolerance low with very high rates ▯ Symptoms of unstable tachycardia may occur
Common Etiologies	▯ Accessory conduction pathway in many PSVT patients ▯ For such otherwise healthy people many factors can provoke the paroxysm, such as caffeine, hypoxia, cigarettes, stress, anxiety, sleep deprivation, numerous medications ▯ Also increased frequency of PSVT in unhealthy patients with CAD, COPD, CHF
Recommended Therapy If specific diagnosis unknown, attempt therapeutic/diagnostic maneuver with ▯ Vagal stimulation ▯ *Adenosine* . . . THEN ➡	**Preserved heart function:** ▯ AV nodal blockade — *β-Blocker* — *Calcium channel blocker* — *Digoxin* ▯ DC cardioversion ▯ Parenteral antiarrhythmics: — *Procainamide* — *Amiodarone* — *Sotalol* (not available in the United States) **Impaired heart function:** ▯ *DC cardioversion* ▯ *Digoxin* ▯ *Amiodarone* ▯ *Diltiazem*



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

Rhythmic Algorithm No. 3: Stable Ventricular Tachycardias

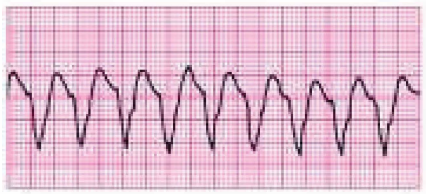


Monomorphic ventricular tachycardia

Stable Ventricular Tachycardia
Monomorphic or Polymorphic?

Monomorphic VT
- Is cardiac function *impaired?*

Note!
May go directly to cardioversion



Monomorphic ventricular tachycardia

Preserved heart function

Poor ejection fraction

Medications: any one
- *Procainamide*
- *Sotalol*

Others acceptable
- *Amiodarone*
- *Lidocaine*

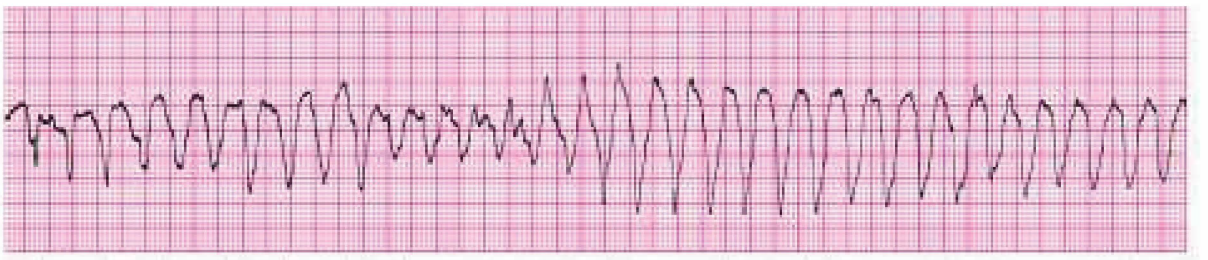
Cardiac function impaired

Amiodarone
- 150 mg IV over 10 minutes

or

Lidocaine
- 0.5 to 0.75 mg/kg IV push

Then use
- ***Synchronized cardioversion***



Torsades de pointes

Polymorphic VT
- Is baseline QT interval prolonged?

Normal baseline QT interval

Prolonged baseline QT interval (suggests torsades)

Normal baseline QT interval

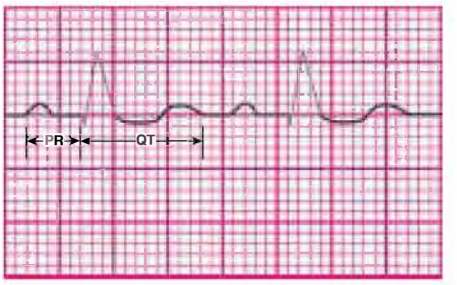
- Treat ischemia
- Correct electrolytes

Medications: any one
- *β-Blockers* **or**
- *Lidocaine* **or**
- *Amiodarone* **or**
- *Procainamide* **or**
- *Sotalol*

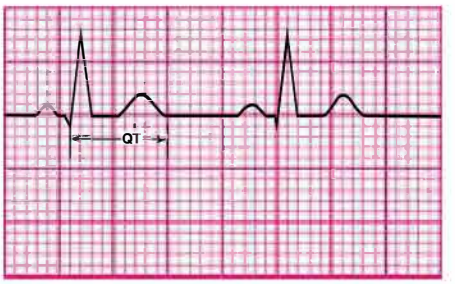
Long baseline QT interval

- Correct abnormal electrolytes

Therapies: any one
- *Magnesium*
- *Overdrive pacing*
- *Isoproterenol*
- *Phenytoin*
- *Lidocaine*



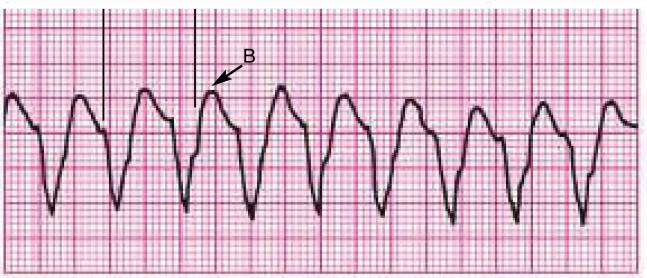
Prolonged baseline QT interval



Normal baseline QT interval

12. Monomorphic Ventricular Tachycardia (Stable)

Pathophysiology	• Impulse conduction is slowed around areas of ventricular injury, infarct, or ischemia • These areas also serve as source of ectopic impulses *(irritable foci)* • These areas of injury can cause the impulse to take a circular course, leading to the reentry phenomenon and rapid repetitive depolarizations	
Defining Criteria per ECG **Key:** The same morphology, or shape, is seen in every QRS complex **Notes:** • 3 or more consecutive PVCs: *ventricular tachycardia* • VT <30 sec duration → *non-sustained VT* • VT >30 sec duration → *sustained VT*	• **Rate:** ventricular rate >100 bpm; typically 120 to 250 bpm • **Rhythm:** no atrial activity seen, only regular ventricular • **PR:** nonexistent • **P waves:** 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) • **QRS complex:** wide and bizarre, "PVC-like" complexes >0.12 sec, with large T wave of opposite polarity from QRS	
Clinical Manifestations	• Monomorphic VT can be asymptomatic, despite the widespread erroneous belief that sustained VT always produces symptoms • Majority of times, however, symptoms of decreased cardiac output (orthostasis, hypotension, syncope, exercise limitations, etc) are seen • Untreated and sustained will deteriorate to unstable VT, often VF	
Common Etiologies	• An acute ischemic event (see pathophysiology) with areas of "ventricular irritability" leading to PVCs • PVCs that occur during the relative refractory period of the cardiac cycle ("R-on-T phenomenon") • Drug-induced, prolonged QT interval (tricyclic antidepressants, procainamide, digoxin, some long-acting antihistamines)	
Recommended Therapy	**Normal Heart**	**Impaired Heart**
	Any one of following parenteral antiarrhythmics: • *Procainamide* • *Sotalol* • *Amiodarone* • *Lidocaine*	• *Amiodarone* **or** • *Lidocaine* **then** • *DC cardioversion* if persists

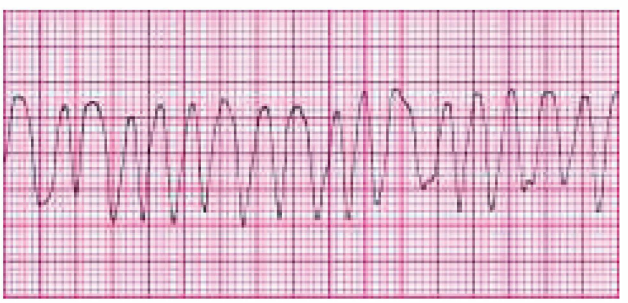


Monomorphic ventricular tachycardia at rate of 150 bpm: wide QRS complexes (arrow A) with opposite polarity T waves (arrow B)

13. Polymorphic Ventricular Tachycardia (Stable)

Pathophysiology	• Impulse conduction is slowed around multiple areas of ventricular injury, infarct, or ischemia • These areas also serve as the source of ectopic impulses *(irritable foci)*; irritable foci occur in multiple areas of the ventricles, thus *"polymorphic"* • These areas of injury can cause impulses to take a circular course, leading to the reentry phenomenom and rapid repetitive depolarizations
Defining Criteria per ECG **Key:** Marked variation and inconsistency seen in the QRS complexes	• **Rate:** ventricular rate >100 bpm; typically 120 to 250 • **Rhythm:** only regular ventricular • **PR:** nonexistent • **P waves:** seldom seen but present; VT is a form of AV dissociation • **QRS complexes:** marked variation and inconsistency seen in the QRS complexes
Clinical Manifestations	• Rare: asymptomatic polymorphic VT • Majority of times: symptoms of decreased cardiac output (orthostasis, hypotension, syncope, exercise limitations, etc) are seen • Seldom → *sustained VT; seldom* → "stable" VT • Tends toward rapid deterioration to pulseless VT or VF
Common Etiologies	• An acute ischemic event (see pathophysiology) with areas of "ventricular irritability" leading to PVCs • PVCs that occur during the relative refractory period of the cardiac cycle ("R-on-T phenomenon") • Drug-induced prolonged QT interval (tricyclic antidepressants, procainamide, digoxin, some long-acting antihistamines)
Recommended Therapy	**Review most recent 12-lead ECG** (baseline) • Measure QT interval just prior to onset of the polymorphic tachycardia • QT interval prolongation? (if YES go to *Torsades de Pointes;* if NO see below) **Normal baseline QT interval:** • Treat ischemia • Correct electrolytes if abnormal **Then:**

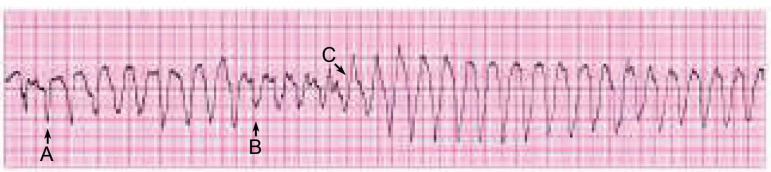
Normal Heart	Impaired Heart
Parenteral medications: any one • *β-Blockers* **or** • *Lidocaine* **or** • *Amiodarone* **or** • *Procainamide* **or** • *Sotalol*	• *Amiodarone* **or** • *Lidocaine* **then** • *DC cardioversion* if persists



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

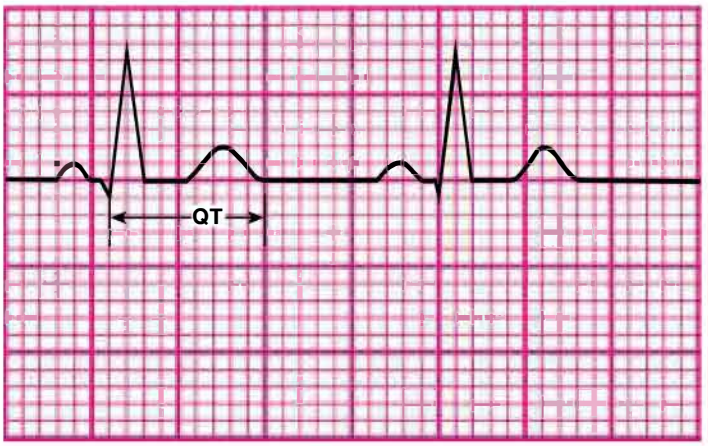
14. Torsades de Pointes (a Unique Subtype of Polymorphic Ventricular Tachycardia)

Pathophysiology	Specific pathophysiology for classic torsades: • QT interval is abnormally long (see below for etiology of QT prolongation) • Leads to increase in the relative refractory period ("vulnerable period") of the cardiac cycle • Increases probability that an irritable focus (PVC) will occur on the T-wave ("vulnerable period" or "R-on-T phenomenon") • R-on-T phenomenon often induces VT
Defining Criteria per ECG **Key:** 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")	• **Atrial Rate:** cannot determine atrial rate • **Ventricular rate:** 150-250 complexes/min • **Rhythm:** only irregular ventricular rhythm • **PR:** nonexistent • **P waves:** nonexistent • **QRS complexes:** display classic "spindle-node" pattern (see left column: "Key")
Clinical Manifestations	• Majority of times patients with torsades have symptoms of decreased cardiac output (orthostasis, hypotension, syncope, exercise limitations, etc) • Asymptomatic torsades, *sustained* torsades, or *"stable"* torsades is uncommon • Tends toward sudden deterioration to pulseless VT or VF
Common Etiologies	Most commonly occurs with prolonged QT interval, from many causes: • Drug-induced: tricyclic antidepressants, procainamide, digoxin, some long-acting antihistamines • Electrolyte and metabolic alterations (hypomagnesemia is the prototype) • Inherited forms of long QT syndrome • Acute ischemic events (see pathophysiology)
Recommended Therapy	**Review most recent 12-lead ECG** (baseline): • Measure QT interval just before onset of the polymorphic tachycardia • QT interval prolongation? (if YES see below; if NO go to the polymorphic VT algorithm) **Long baseline QT interval:** • Treat ischemia • Correct electrolytes if abnormal **Then therapies (any one):** • Magnesium • Overdrive pacing • Isoproterenol (pharmacologic overdrive pacing) • Phenytoin • Lidocaine

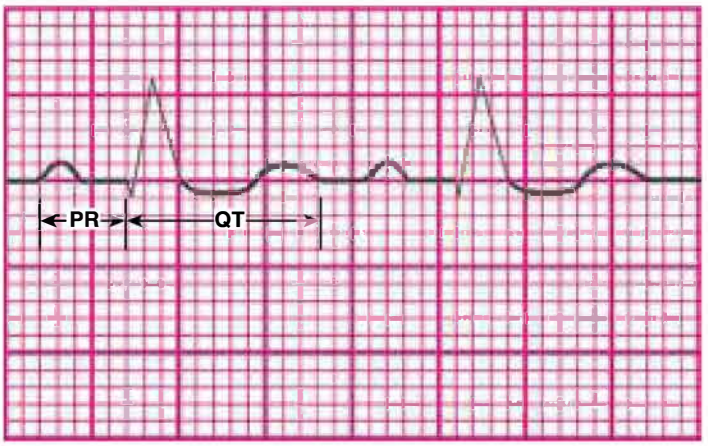


Torsades de pointes
(a unique subtype of polymorphic ventricular tachycardia)
Arrows: A — Start of a "spindle"; note negative initial deflection; note increasing QRS amplitude
B — End of "spindle"; start of "node"
C — End of "node"; start of next "spindle"; note positive initial deflection; increase-decrease in QRS amplitude

15. Normal and Prolonged Baseline QT Interval

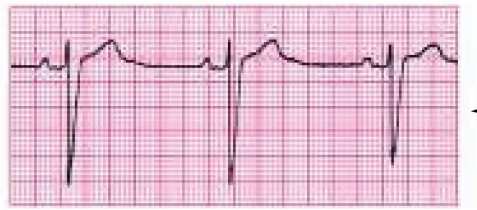


Normal baseline QT interval
Rate: 80 bpm
QT interval: 0.36 sec
(within QT_C range of 0.32 – 0.39 sec
for a heart rate of 80 bpm)

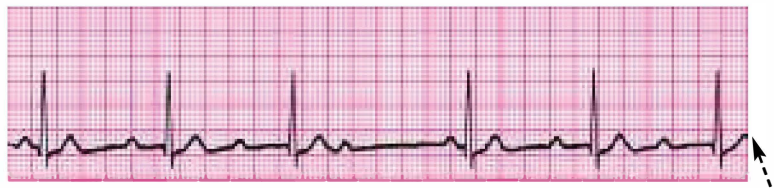


Prolonged baseline QT interval
Due to drug toxicity
PR interval: >0.20 sec
Rate: 80 bpm
QT interval: prolonged, 0.45 sec
(above QT_C range of 0.32 – 0.39 sec
for a heart rate of 80 bpm)
QRS complex: widened, >0.12 sec

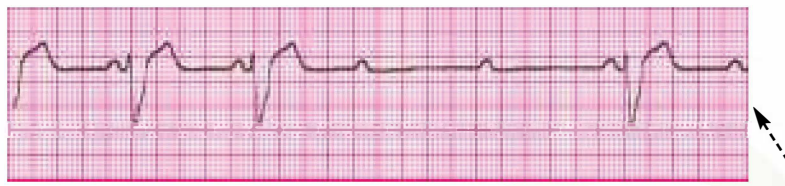
Rhythmic Algorithm No. 4: Bradycardias



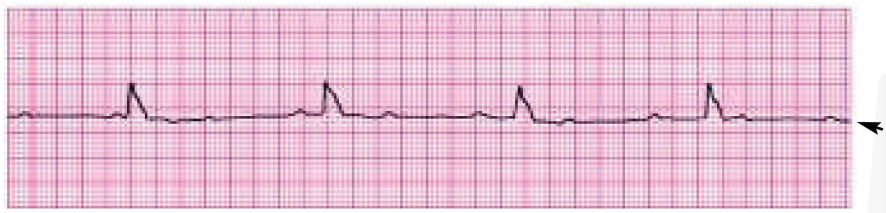
Sinus bradycardia with borderline first-degree AV block



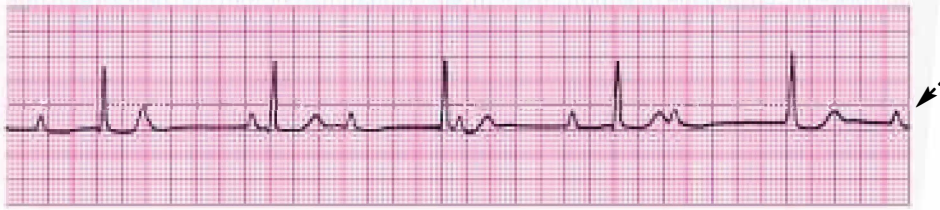
Second-degree AV block type I

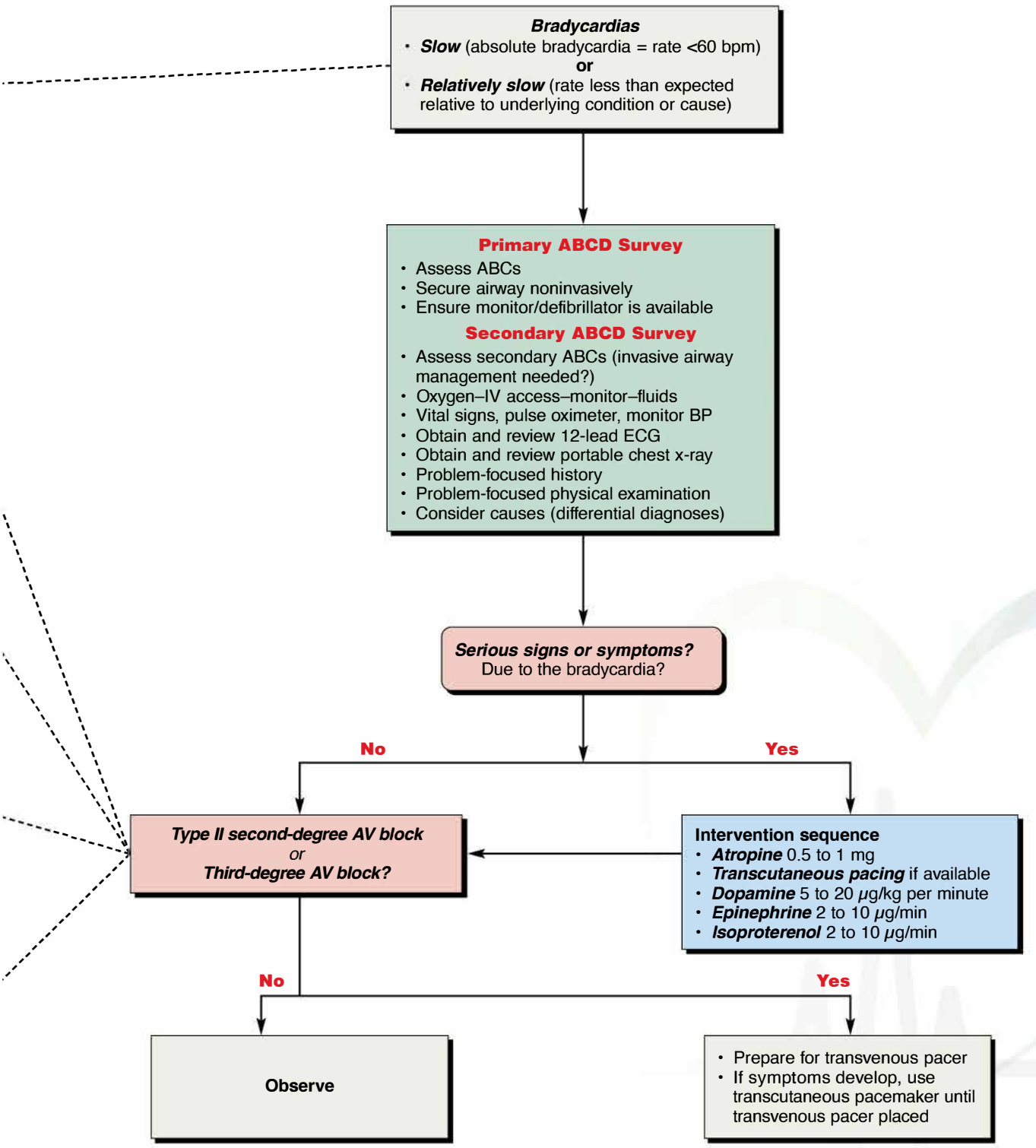


Second-degree AV block type II



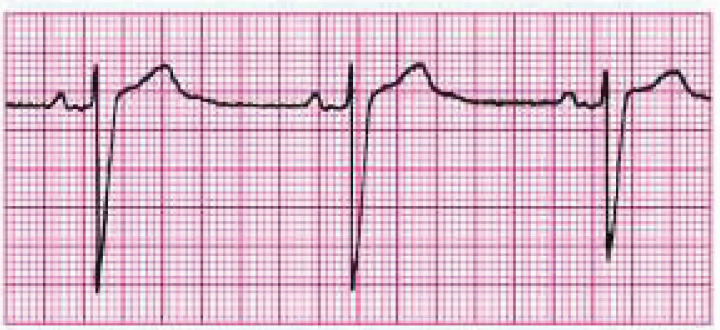
Complete AV block with a ventricular escape pacemaker (wide QRS: 0.12 to 0.14 sec)




Bradycardias
• Slow (absolute bradycardia = rate <60 bpm)
or
• Relatively slow (rate less than expected relative to underlying condition or cause)
Primary ABCD Survey
• Assess ABCs
• Secure airway noninvasively
• Ensure monitor/defibrillator is available
Secondary ABCD Survey
• Assess secondary ABCs (invasive airway management needed?)
• Oxygen–IV access–monitor–fluids
• Vital signs, pulse oximeter, monitor BP
• Obtain and review 12-lead ECG
• Obtain and review portable chest x-ray
• Problem-focused history
• Problem-focused physical examination
• Consider causes (differential diagnoses)
Serious signs or symptoms?
Due to the bradycardia?
No
Yes
Type II second-degree AV block
or
Third-degree AV block?
Intervention sequence
• Atropine 0.5 to 1 mg
• Transcutaneous pacing if available
• Dopamine 5 to 20 µg/kg per minute
• Epinephrine 2 to 10 µg/min
• Isoproterenol 2 to 10 µg/min
No
Yes
Observe
• Prepare for transvenous pacer
• If symptoms develop, use transcutaneous pacemaker until transvenous pacer placed

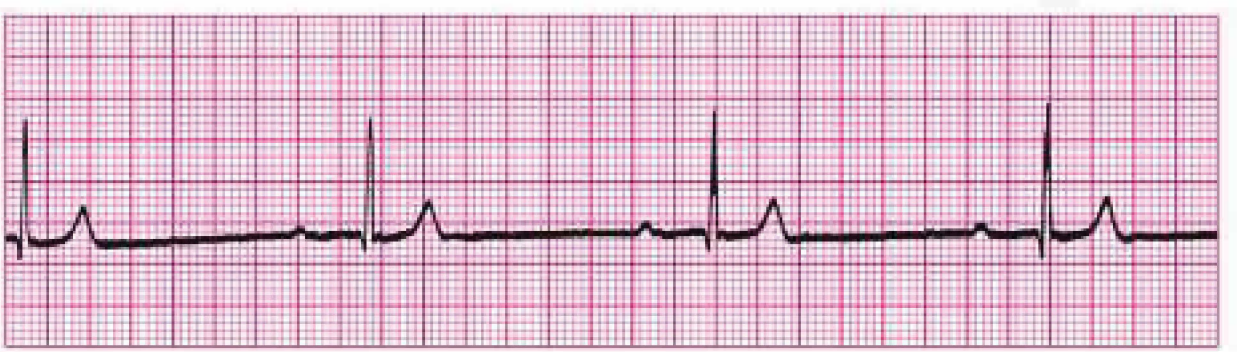
16. Sinus Bradycardia

Pathophysiology	• Impulses originate at SA node at a slow rate • Not pathological; not an abnormal arrhythmia • More a physical sign
Defining Criteria per ECG **Key:** Regular P waves followed by regular QRS complexes at rate <60 beats/min **Note:** Often a physical sign rather than an abnormal rhythm	• **Rate:** <60 beats/min • **Rhythm:** regular sinus • **PR:** regular; <0.20 sec • **P waves:** size and shape normal; every P wave is followed by a QRS complex; every QRS complex is preceded by a P wave • **QRS complex:** narrow; ≤0.10 sec in absence of intraventricular conduction defect
Clinical Manifestations	• At rest, usually asymptomatic • With increased activity, persistent slow rate will lead to symptoms of easy fatigue, SOB, dizziness or lightheadedness, syncope, hypotension
Common Etiologies	• Normal for well-conditioned people • A vasovagal event such as vomiting, valsalva, rectal stimuli, inadvertent pressure on carotid sinus ("shaver's syncope") • Acute MIs that affect circulation to SA node (right coronary artery); most often inferior AMIs • Adverse drug effects, eg, blocking agents (β or calcium channel), digoxin, quinidine
Recommended Therapy	• Treatment rarely indicated • Treat only if patient has significant signs or symptoms due to the bradycardia • Oxygen is always appropriate **Intervention sequence for bradycardia** • *Atropine* 0.5 to 1 mg IV if vagal mechanism • *Transcutaneous pacing* if available **If signs and symptoms are severe, consider catecholamine infusions:** • *Dopamine* 5 to 20 μg/kg per min • *Epinephrine* 2 to 10 μg/min • *Isoproterenol* 2 to 10 μg/min



17. First-Degree Heart Block

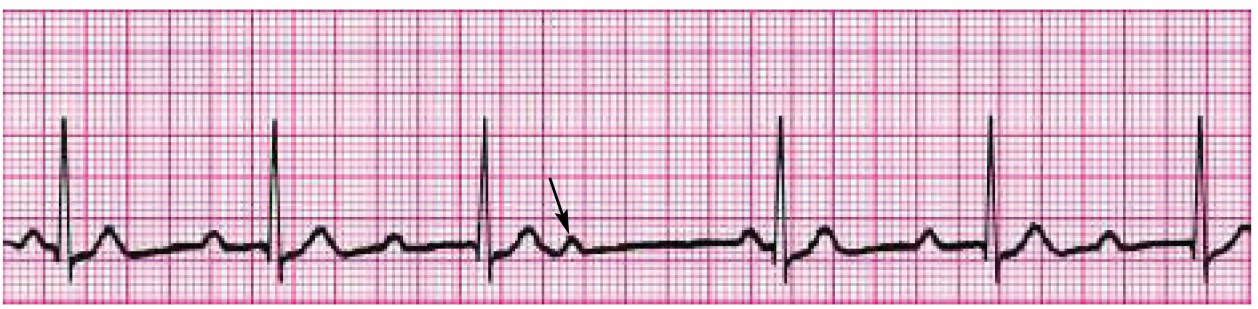
	Closer to being a physical sign than an abnormal arrhythmia
Defining Criteria per ECG **Key:** PR interval >0.20 sec	**Rate:** First-degree heart block can be seen with both sinus bradycardia and sinus tachycardia **Rhythm:** sinus, regular, both atria and ventricles **PR:** prolonged, >0.20 sec, but does not vary *(fixed)* **P waves:** size and shape normal; every P wave is followed by a QRS complex; every QRS complex is preceded by a P wave **QRS complex:** narrow; ≤0.10 sec in absence of intraventricular conduction defect
Clinical Manifestations	Usually asymptomatic at rest Rarely, if bradycardia worsens, person may become symptomatic from the slow rate
Common Etiologies	Large majority of first-degree heart blocks are due to drugs, usually the AV nodal blockers: β-blockers, calcium channel blockers, and digoxin Any condition that stimulates the parasympathetic nervous system (eg, vasovagal reflex) Acute MIs that affect circulation to AV node (right coronary artery); most often inferior AMIs
Recommended Therapy	Treat only when patient has significant signs or symptoms that are due to the bradycardia Be alert to block deteriorating to second-degree, type I or type II block Oxygen is always appropriate **Intervention sequence for symptomatic bradycardia** *Atropine* 0.5 to 1 mg IV if vagal mechanism *Transcutaneous pacing* if available **If signs and symptoms are severe, consider catecholamine infusions:** *Dopamine* 5 to 20 μg/kg per min *Epinephrine* 2 to 10 μg/min *Isoproterenol* 2 to 10 μg/min



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

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

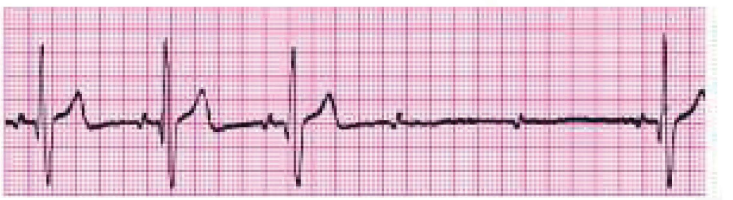
Pathophysiology	▯ Site of pathology: AV node ▯ AV node blood supply comes from branches of the right coronary artery ▯ Impulse conduction is increasingly slowed at the AV node (causing increasing PR interval) ▯ Until one sinus impulse is completely blocked and a QRS complex fails to follow
Defining Criteria per ECG **Key:** There is progressive lengthening of the PR interval until one P wave is not followed by a QRS complex (the dropped beat)	▯ **Rate:** atrial rate just slightly faster than ventricular (because of dropped beats); usually normal range ▯ **Rhythm:** regular for atrial beats; irregular for ventricular (because of dropped beats); can show regular P waves marching through irregular QRS ▯ **PR:** 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") ▯ **P waves:** size and shape remain normal; occasional P wave not followed by a QRS complex (the "dropped beat") ▯ **QRS complex:** ≤0.10 sec most often, but a QRS "drops out" periodically
Clinical Manifestations—Rate-Related	**Due to bradycardia:** ▯ **Symptoms:** chest pain, shortness of breath, decreased level of consciousness ▯ **Signs:** hypotension, shock, pulmonary congestion, CHF, angina
Common Etiologies	▯ AV nodal blocking agents: β-blockers, calcium channel blockers, digoxin ▯ Conditions that stimulate the parasympathetic system ▯ An acute coronary syndrome that involves the *right* coronary artery
Recommended Therapy **Key:** Treat only when patient has significant signs or symptoms that are due to the bradycardia	**Intervention sequence for symptomatic bradycardia:** ▯ *Atropine* 0.5 to 1 mg IV if vagal mechanism ▯ *Transcutaneous pacing* if available **If signs and symptoms are severe, consider catecholamine infusions:** ▯ *Dopamine* 5 to 20 μg/kg per min ▯ *Epinephrine* 2 to 10 μg/min ▯ *Isoproterenol* 2 to 10 μg/min



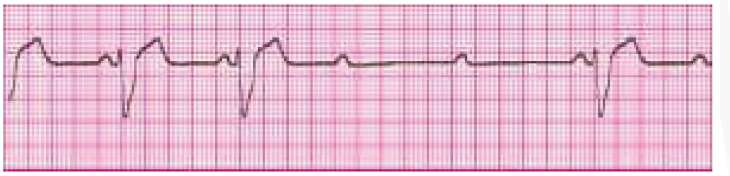
Second-degree heart block type I. Note progressive lengthening of PR interval until one P wave (arrow) is not followed by a QRS.

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

Pathophysiology	▯ The pathology, ie, the site of the block, is most often *below* the AV node (infranodal); at the bundle of His (infrequent) or at the bundle branches ▯ Impulse conduction is normal through the node, thus no first-degree block and no prior PR prolongation
Defining Criteria per ECG	▯ **Atrial Rate:** usually 60-100 beats/min ▯ **Ventricular rate:** by definition (due to the blocked impulses) slower than atrial rate ▯ **Rhythm:** atrial = regular; ventricular = irregular (because of blocked impulses) ▯ **PR:** constant and set; no progressive prolongation as with type I—a distinguishing characteristic. ▯ **P waves:** typical in size and shape; by definition some P waves will not be followed by a QRS complex ▯ **QRS complex:** narrow (≤0.10 sec) implies high block relative to the AV node; wide (>0.12 sec) implies low block relative to the AV node
Clinical Manifestations—Rate-Related	**Due to bradycardia:** ▯ **Symptoms:** chest pain, shortness of breath, decreased level of consciousness ▯ **Signs:** hypotension, shock, pulmonary congestions, CHF, acute MI
Common Etiologies	▯ An acute coronary syndrome that involves branches of the *left* coronary artery
Recommended Therapy **Pearl:** New onset type II second-degree heart block in clinical context of acute coronary syndrome is indication for transvenous pacemaker insertion	**Intervention sequence for bradycardia due to type II second-degree *or* third-degree heart block:** ▯ Prepare for *transvenous* pacer ▯ Atropine is seldom effective for infranodal block ▯ Use *transcutaneous pacing* if available as a bridge to transvenous pacing (verify patient tolerance and mechanical capture. Use sedation and analgesia as needed.) **If signs/symptoms are severe and unresponsive to TCP, and transvenous pacing is delayed, consider catecholamine infusions:** ▯ *Dopamine* 5 to 20 μg/kg per min ▯ *Epinephrine* 2 to 10 μg/min ▯ *Isoproterenol* 2 to 10 μg/min



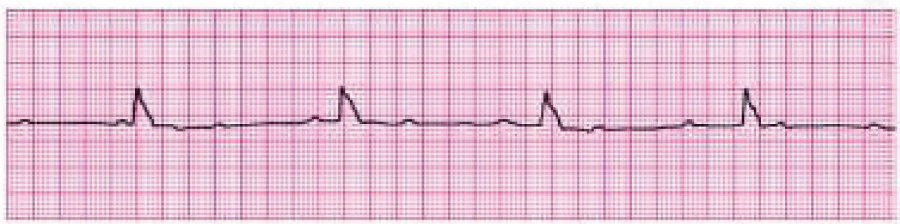
Type II (high block): regular PR-QRS intervals until 2 dropped beats occur; borderline normal QRS complexes indicate high nodal or nodal block



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

Pathophysiology **Pearl:** *AV dissociation* is the defining class; *third-degree* or *complete heart block* is one type of AV dissociation. By convention (outdated): if ventricular escape depolarization is faster than atrial rate = *"AV dissociation"*; if slower = "third-degree heart block"	Injury or damage to the cardiac conduction system so that no impulses *(complete block)* pass between atria and ventricles (neither antegrade nor retrograde) This complete block can occur at several different anatomic areas: ▯ AV node ("high" or "supra" or "junctional" *nodal block*) ▯ Bundle of His ▯ Bundle branches ("low-nodal" or "infranodal" block)
Defining Criteria per ECG **Key:** The third-degree block (see pathophysiology) causes the atria and ventricles to depolarize independently, with no relationship between the two (AV dissociation)	▯ **Atrial rate:** usually 60-100 beats/min; impulses completely independent ("dissociated") from ventricular rate ▯ **Ventricular rate:** depends on rate of the ventricular escape beats that arise: — Ventricular escape beat rate slower than atrial rate = third-degree heart block (20-40 beats/min) — Ventricular escape beat rate faster than atrial rate = AV dissociation (40-55 beats/min) ▯ **Rhythm:** both atrial rhythm and ventricular rhythm are regular but independent ("dissociated") ▯ **PR:** by definition there is no relationship between P wave and R wave ▯ **P waves:** typical in size and shape ▯ **QRS complex:** narrow (≤0.10 sec) implies high block relative to the AV node; wide (>0.12 sec) implies low block relative to the AV node
Clinical Manifestations—Rate-Related	**Due to bradycardia:** ▯ **Symptoms:** chest pain, shortness of breath, decreased level of consciousness ▯ **Signs:** hypotension, shock, pulmonary congestions, CHF, acute MI
Common Etiologies	▯ An acute coronary syndrome that involves branches of the *left* coronary artery ▯ In particular, the LAD (left anterior descending) and branches to the interventricular septum (supply bundle branches)
Recommended Therapy **Pearl:** New onset third-degree heart block in clinical context of acute coronary syndrome is indication for transvenous pacemaker insertion **Pearl:** *Never treat third-degree heart block plus ventricular escape beats with lidocaine*	**Intervention sequence for bradycardia due to type II second-degree *or* third-degree heart block:** ▯ Prepare for *transvenous* pacer ▯ Use *transcutaneous pacing* if available as a bridge to transvenous pacing (verify patient tolerance and mechanical capture; use sedation and analgesia as needed) **If signs/symptoms are severe and unresponsive to TCP, and transvenous pacing is delayed, consider catecholamine infusions:** ▯ *Dopamine* 5 to 20 μg/kg per min ▯ *Epinephrine* 2 to 10 μg/min ▯ *Isoproterenol* 2 to 10 μg/min

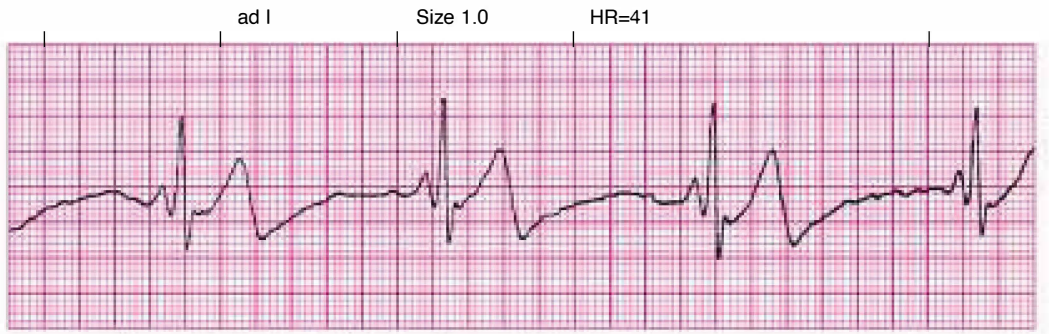


Third-degree heart block: regular P waves at 50 to 55 bpm; regular ventricular "escape beats" at 35 to 40 bpm; no relationship between P waves and escape beats

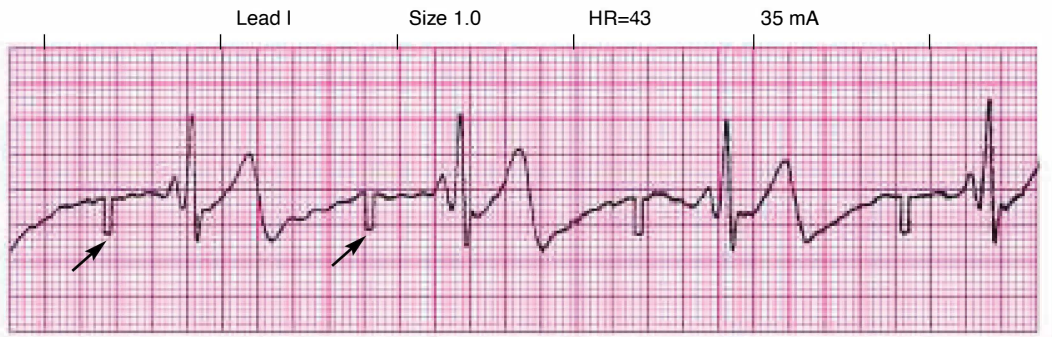
21. Transcutaneous Pacing

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

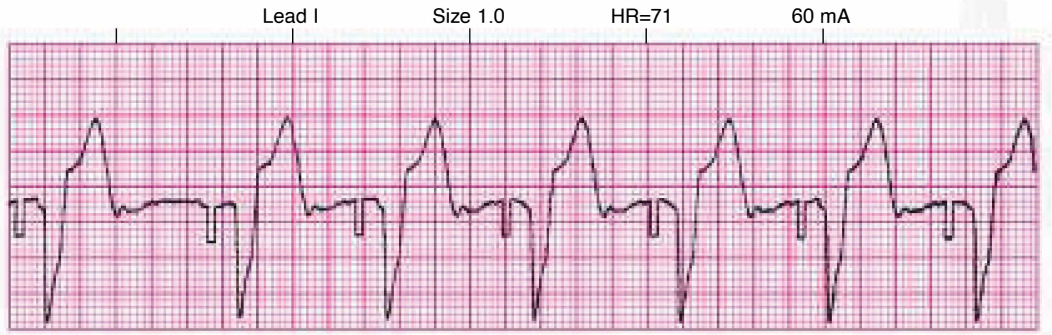
Rhythm Strip	Comments
A. Bradycardia (third-degree heart block): no pacing (**Note:** Rates and intervals slightly altered due to monitor compensation for pacing stimulus)	▯ QRS rate = 41 beats/min ▯ P waves seen = 125 beats/min ▯ QRS = very wide, 0.24 sec; ventricular escape beats ▯ QRS and T wave polarity = both positive ▯ Patient: SOB at rest; severe SOB with walking; near syncope
B. Transcutaneous pacing initiated at low current (35 mA) and slow rate (50 beats/min). Below the threshold current needed to stimulate the myocardium	▯ With TCP, monitor electrodes are attached in modified lead II position ▯ As current (in milliamperes) is gradually increased, the monitor leads detect the pacing stimuli as a squared off, negative marker ▯ TC pacemakers incorporate standard ECG monitoring circuitry but incorporate filters to dampen the pacing stimuli ▯ 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
C. Pacing current turned up above threshold (60 mA at 71 beats/min) and "captures" the myocardium	▯ TCP stimulus does not work through the normal cardiac conduction system but by a direct electrical stimulus of the myocardium ▯ Therefore, a "capture," where TCP stimulus results in a myocardial contraction, will resemble a PVC ▯ Electrical capture is characterized by a wide QRS complex, with the initial deflection and the terminal deflection *always* in opposite directions ▯ A "mechanically captured beat" will produce effective myocardial contraction with production of some blood flow (usually assessed by a palpable carotid pulse)



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)