ACLs Rhythms for the ACLS Algorithms

The Basics

## The Cardiac Arrest Rhythms

### 2. Ventricular Fibrillation/Pulseless Ventricular Tachycardia

<table>
<thead>
<tr>
<th>Pathophysiology</th>
<th>Ventricles consist of areas of normal myocardium alternating with areas of ischemic, injured, or infarcted myocardium, leading to chaotic pattern of ventricular depolarization</th>
</tr>
</thead>
</table>
| **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

![Coarse VF](image1.png)

### Fine VF

![Fine VF](image2.png)
## 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

- 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

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Any organized rhythm without detectable pulse is “PEA”
### 4. Asystole

<table>
<thead>
<tr>
<th>Defining Criteria per ECG</th>
</tr>
</thead>
<tbody>
<tr>
<td>Classically asystole presents as a “flat line”; any defining criteria are virtually nonexistent</td>
</tr>
<tr>
<td><strong>Rate</strong>: no ventricular activity seen or ≤6/min; so-called “P-wave asystole” occurs with only atrial impulses present to form P waves</td>
</tr>
<tr>
<td><strong>Rhythm</strong>: no ventricular activity seen; or ≤6/min</td>
</tr>
<tr>
<td><strong>PR</strong>: cannot be determined; occasionally P wave seen, but by definition R wave must be absent</td>
</tr>
<tr>
<td><strong>QRS complex</strong>: no deflections seen that are consistent with a QRS complex</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Clinical Manifestations</th>
</tr>
</thead>
<tbody>
<tr>
<td>Early may see agonal respirations; unconscious; unresponsive</td>
</tr>
<tr>
<td>No pulse; no blood pressure</td>
</tr>
<tr>
<td>Cardiac arrest</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Common Etiologies</th>
</tr>
</thead>
<tbody>
<tr>
<td>End of life (death)</td>
</tr>
<tr>
<td>Ischemia/hypoxia from many causes</td>
</tr>
<tr>
<td>Acute respiratory failure (no oxygen; apnea; asphyxiation)</td>
</tr>
<tr>
<td>Massive electrical shock: electrocution; lightning strike</td>
</tr>
<tr>
<td>Postdefibrillatory shocks</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Recommended Therapy</th>
</tr>
</thead>
<tbody>
<tr>
<td>Always check for DNAR status</td>
</tr>
<tr>
<td>Primary ABCD survey (basic CPR)</td>
</tr>
<tr>
<td>Secondary ABCD survey</td>
</tr>
</tbody>
</table>

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**Asystole: agonal complexes too slow to make this rhythm “PEA”**
### 5. Sinus Tachycardia

<table>
<thead>
<tr>
<th>Pathophysiology</th>
<th>None—more a physical sign than an arrhythmia or pathologic condition</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Normal impulse formation and conduction</td>
</tr>
</tbody>
</table>

| 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                                   | Never treat the tachycardia per se                                  |
|                                                       | Treat only the causes of the tachycardia                            |
|                                                       | Never countershock                                                  |

Sinus tachycardia
Rhythmic Algorithm No. 1: Tachycardias Overview

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

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

1. Atrial fibrillation
2. Atrial flutter

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

1. Atrial fibrillation
2. Atrial flutter

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)

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

1. Atrial fibrillation
2. Atrial flutter

Evaluation focus, 4 clinical features:
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Treatment of atrial fibrillation/atrial flutter
(See following table)

Treatment of SVT
(See narrow-complex tachycardia algorithm)
3. Stable wide-complex tachycardia: unknown type
   - Attempt to establish a specific diagnosis
     - 12-lead ECG
     - Esophageal lead
     - Clinical information

4. Stable monomorphic VT and/or polymorphic VT

Unstable patient: serious signs or symptoms
- Establish rapid heart rate as cause of signs and symptoms
- Rate-related signs and symptoms occur at many rates, seldom <150 bpm
  - Prepare for immediate cardioversion (see algorithm)

Confirmed SVT
Confirmed stable VT
Wide-complex tachycardia of unknown type
Ejection fraction <40%
Clinical CHF

Treatment of stable monomorphic and polymorphic VT
(See stable VT: monomorphic and polymorphic algorithm)

DC cardioversion or Procainamide or Amiodarone
DC cardioversion or Amiodarone

Monomorphic ventricular tachycardia
Polymorphic ventricular tachycardia
6. Reentry Tachycardia Mechanism

A — Normal impulse comes down Purkinje fibers to join muscle fibers.
B — One impulse (B₁) encounters an area of one-way (unidirectional) block (B₂) and stops.
C — Meanwhile, the normally conducted impulse (C₁) has moved down the Purkinje fiber, into the muscle fiber (C₂); and as a retrograde impulse, moves through the area of slow conduction (C₃).
D — The retrograde impulse (D₁) now reenters the Purkinje and muscle fibers (D₂); and keeps this reentry cycle repeating itself multiple times (D₃).
7. Atrial Fibrillation/Atrial Flutter

<table>
<thead>
<tr>
<th>Pathophysiology</th>
<th>Atrial Fibrillation</th>
<th>Atrial Flutter</th>
</tr>
</thead>
<tbody>
<tr>
<td>■ Atrial impulses faster than SA node impulses</td>
<td></td>
<td></td>
</tr>
<tr>
<td>■ Atrial fibrillation → impulses take multiple, chaotic, random pathways through the atria</td>
<td></td>
<td></td>
</tr>
<tr>
<td>■ Atrial flutter → impulses take a circular course around the atria, setting up the flutter waves</td>
<td></td>
<td></td>
</tr>
<tr>
<td>■ Mechanism of impulse formation: reentry</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Defining Criteria and ECG Features (Distinctions here between atrial fibrillation vs atrial flutter; all other characteristics are the same)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Atrial Fibrillation Key</strong>: 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.</td>
</tr>
<tr>
<td><strong>Atrial Flutter Key</strong>: Flutter waves seen in classic “sawtooth pattern”</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Rate</th>
<th>Atrial Fibrillation</th>
<th>Atrial Flutter</th>
</tr>
</thead>
<tbody>
<tr>
<td>■ Wide-ranging ventricular response to atrial rate of 300-400 beats/min</td>
<td></td>
<td>■ Atrial rate 220-350 beats/min</td>
</tr>
<tr>
<td>■ Ventricular response = a function of AV node block or conduction of atrial impulses</td>
<td></td>
<td>■ Ventricular response rarely &gt;150-180 beats because of AV node conduction limits</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Rhythm</th>
<th>Atrial Fibrillation</th>
<th>Atrial Flutter</th>
</tr>
</thead>
<tbody>
<tr>
<td>■ Irregular (classic “irregular irregular”)</td>
<td></td>
<td>■ Regular (unlike atrial fibrillation)</td>
</tr>
<tr>
<td>■ Ventricular rhythm often regular</td>
<td></td>
<td>■ Set ratio to atrial rhythm, eg, 2-to-1 or 3-to-1</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>P waves</th>
<th>Atrial Fibrillation</th>
<th>Atrial Flutter</th>
</tr>
</thead>
<tbody>
<tr>
<td>■ Chaotic atrial fibrillatory waves only</td>
<td></td>
<td>■ No true P waves seen</td>
</tr>
<tr>
<td>■ Creates disturbed baseline</td>
<td></td>
<td>■ Flutter waves in “sawtooth pattern” is classic</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>PR</th>
<th>Atrial Fibrillation</th>
<th>Atrial Flutter</th>
</tr>
</thead>
<tbody>
<tr>
<td>■ Cannot be measured</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>QRS</th>
<th>Atrial Fibrillation</th>
<th>Atrial Flutter</th>
</tr>
</thead>
<tbody>
<tr>
<td>■ Remains ≤0.10-0.12 sec unless QRS complex distorted by fibrillation/flutter waves or by conduction defects through ventricles</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Clinical Manifestations</th>
<th>Atrial Fibrillation</th>
<th>Atrial Flutter</th>
</tr>
</thead>
<tbody>
<tr>
<td>■ 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</td>
<td></td>
<td>■ Loss of “atrial kick” may lead to drop in cardiac output and decreased coronary perfusion</td>
</tr>
<tr>
<td>■ Irregular rhythm often perceived as “palpitations”</td>
<td></td>
<td>■ Can be asymptomatic</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Common Etiologies</th>
<th>Atrial Fibrillation</th>
<th>Atrial Flutter</th>
</tr>
</thead>
<tbody>
<tr>
<td>■ Acute coronary syndromes; CAD; CHF</td>
<td></td>
<td>■ Drug-induced: <strong>digoxin or quinidine</strong> most common</td>
</tr>
<tr>
<td>■ Disease at mitral or tricuspid valve</td>
<td></td>
<td>■ Hyperthyroidism</td>
</tr>
<tr>
<td>■ Hypoxia; acute pulmonary embolism</td>
<td></td>
<td></td>
</tr>
<tr>
<td>■ Drug-induced: <strong>digoxin or quinidine</strong> most common</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
7. Atrial Fibrillation/Atrial Flutter (continued)

<table>
<thead>
<tr>
<th>Recommended Therapy</th>
<th>Control Rate</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Evaluation Focus:</strong></td>
<td><strong>Treatment Focus:</strong></td>
</tr>
<tr>
<td>1. Patient clinically unstable?</td>
<td>1. Treat unstable patients urgently</td>
</tr>
<tr>
<td>2. Cardiac function impaired?</td>
<td>2. Control the rate</td>
</tr>
<tr>
<td>3. WPW present?</td>
<td>3. Convert the rhythm</td>
</tr>
<tr>
<td>4. Duration ≤48 or &gt;48 hr?</td>
<td>4. Provide anticoagulation</td>
</tr>
</tbody>
</table>

**Convert Rhythm**

<table>
<thead>
<tr>
<th>Impaired Heart</th>
<th>Normal Heart</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>If ≤48 hours:</strong></td>
<td><strong>If ≤48 hours:</strong></td>
</tr>
<tr>
<td>— DC cardioversion or amiodarone or others</td>
<td>— DC Cardioversion or amiodarone</td>
</tr>
<tr>
<td><strong>If &gt;48 hours:</strong></td>
<td><strong>If &gt;48 hours:</strong></td>
</tr>
<tr>
<td>— Anticoagulate × 3 wk, then</td>
<td>— Anticoagulate × 3 wk, then</td>
</tr>
<tr>
<td>— DC cardioversion, then</td>
<td>— DC cardioversion, then</td>
</tr>
<tr>
<td>— Anticoagulate × 4 wk or</td>
<td>— Anticoagulate × 4 more wk</td>
</tr>
<tr>
<td><strong>IV heparin and TEE to rule out atrial clot, then</strong></td>
<td><strong>Telediagnosis</strong></td>
</tr>
<tr>
<td><strong>DC cardioversion within 24 hours, then</strong></td>
<td><strong>Anticoagulation × 4 more wk</strong></td>
</tr>
<tr>
<td><strong>Anticoagulation × 4 more wk</strong></td>
<td></td>
</tr>
</tbody>
</table>

TEE indicates transesophageal echocardiogram.
8. WPW (Wolff-Parkinson-White) Syndrome

| Pathophysiology | The prototypical **pre-excitation syndrome**: congenital malformation; 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 | **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 |
### 8. WPW (Wolff-Parkinson-White) Syndrome (continued)

<table>
<thead>
<tr>
<th>Evaluation Focus</th>
<th>Treatment Focus</th>
<th>Normal Heart</th>
<th>Impaired Heart</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Patient clinically unstable?</td>
<td>1. Treat unstable patients urgently</td>
<td></td>
<td></td>
</tr>
<tr>
<td>2. Cardiac function impaired?</td>
<td>2. Control the rate</td>
<td></td>
<td></td>
</tr>
<tr>
<td>3. WPW present?</td>
<td>3. Convert the rhythm</td>
<td></td>
<td></td>
</tr>
<tr>
<td>4. Duration ≤48 or &gt;48 hr?</td>
<td>4. Provide anticoagulation</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Class III (can be harmful) in treating atrial fibrillation with WPW:
- Adenosine
- β-Blockers
- Calcium channel blockers
- Digoxin

**Wolff-Parkinson-White: Control Rate**

- **Normal Heart**
  - Cardioversion or
  - Antiarrhythmic (IIb): amiodarone or flecainide or procainamide or propafenone or sotalol

- **Impaired Heart**
  - Cardioversion or
  - Amiodarone

**Wolff-Parkinson-White: Convert Rhythm**

<table>
<thead>
<tr>
<th>Duration ≤48 Hours</th>
<th>Duration &gt;48 Hours</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cardioversion or</td>
<td>Anticoagulate × 3 wk then</td>
</tr>
<tr>
<td>Antiarrhythmic (IIb): amiodarone or flecainide or procainamide or propafenone or sotalol</td>
<td>DC cardioversion then</td>
</tr>
<tr>
<td>If impaired heart: cardioversion or amiodarone</td>
<td>Anticoagulate × 4 wk</td>
</tr>
</tbody>
</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 | - **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 | **Preserved heart function:**
| If specific diagnosis unknown, attempt therapeutic/diagnostic maneuver with | - β-Blocker
|                               | - *Calcium channel blocker*
|                               | - Amiodarone
|                               | - **NO DC cardioversion!**
| If impaired heart function: | **If impaired heart function:**
|                               | - Amiodarone
|                               | - **NO DC cardioversion!**

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

Junctional tachycardia

Multifocal atrial tachycardia

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

<table>
<thead>
<tr>
<th>Pathophysiology</th>
<th>■ Areas of <em>automaticity</em> (impulse formation) originate irregularly and rapidly at different points in the atria</th>
</tr>
</thead>
</table>
| Defining Criteria and ECG Features | ■ **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 | ■ **Preserved heart function**:  
■ β-blocker  
■ Calcium channel blocker  
■ Amiodarone  
■ **NO DC cardioversion**!  
If impaired heart function:  
■ Amiodarone  
■ Diltiazem  
■ **NO DC cardioversion**! |

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**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

<table>
<thead>
<tr>
<th>Key</th>
<th>Regular, narrow-complex tachycardia without P-waves, and sudden, paroxysmal onset or cessation, or both</th>
</tr>
</thead>
<tbody>
<tr>
<td>Rate</td>
<td>exceeds upper limit of sinus tachycardia (&gt;120 beats/min); seldom &lt;150 beats/min; up to 250 beats/min</td>
</tr>
<tr>
<td>Rhythm</td>
<td>regular</td>
</tr>
<tr>
<td>P waves</td>
<td>seldom seen because rapid rate causes P wave loss in preceding T waves or because the origin is low in the atrium</td>
</tr>
<tr>
<td>QRS complex</td>
<td>normal, narrow (≤0.10 sec usually)</td>
</tr>
</tbody>
</table>

#### 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)*
Stable Ventricular Tachycardia
Monomorphic or Polymorphic?

Monomorphic VT
- Is cardiac function impaired?

Preserved heart function

Medications: any one
- Procainamide
- Sotalol
Others acceptable
- Amiodarone
- Lidocaine

Monomorphic ventricular tachycardia

Poor ejection fraction

Note!
May go directly to cardioversion

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
Polymorphic VT
- Is baseline QT interval prolonged?

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 (suggests torsades)
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

<table>
<thead>
<tr>
<th>Normal Heart</th>
<th>Impaired Heart</th>
</tr>
</thead>
<tbody>
<tr>
<td>Any one of following parenteral antiarrhythmics:</td>
<td><strong>Amiodarone</strong> or <strong>Lidocaine</strong> then <strong>DC cardioversion</strong> if persists</td>
</tr>
<tr>
<td>- Procainamide</td>
<td><strong>Amiodarone</strong></td>
</tr>
<tr>
<td>- Sotalol</td>
<td>or <strong>Lidocaine</strong></td>
</tr>
<tr>
<td>- Amiodarone</td>
<td>then <strong>DC cardioversion</strong> if persists</td>
</tr>
<tr>
<td>- Lidocaine</td>
<td></td>
</tr>
</tbody>
</table>

---

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 phenomenon 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:**

<table>
<thead>
<tr>
<th>Normal Heart</th>
<th>Impaired Heart</th>
</tr>
</thead>
<tbody>
<tr>
<td>Parenteral medications: any one</td>
<td></td>
</tr>
<tr>
<td>- β-Blockers or</td>
<td></td>
</tr>
<tr>
<td>- Lidocaine or</td>
<td></td>
</tr>
<tr>
<td>- Amiodarone or</td>
<td></td>
</tr>
<tr>
<td>- Procainamide or</td>
<td></td>
</tr>
<tr>
<td>- Sotalol</td>
<td></td>
</tr>
<tr>
<td>- <strong>Amiodarone</strong> or</td>
<td></td>
</tr>
<tr>
<td>- <strong>Lidocaine</strong> then</td>
<td></td>
</tr>
<tr>
<td>- <strong>DC cardioversion</strong> if persists</td>
<td></td>
</tr>
</tbody>
</table>
### 14. Torsades de Pointes (a Unique Subtype of Polymorphic Ventricular Tachycardia)

<table>
<thead>
<tr>
<th>Pathophysiology</th>
<th>Specific pathophysiology for classic torsades:</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>- QT interval is abnormally long (see below for etiology of QT prolongation)</td>
</tr>
<tr>
<td></td>
<td>- Leads to increase in the relative refractory period (&quot;vulnerable period&quot;) of the cardiac cycle</td>
</tr>
<tr>
<td></td>
<td>- Increases probability that an irritable focus (PVC) will occur on the T-wave (&quot;vulnerable period&quot; or &quot;R-on-T phenomenon&quot;)</td>
</tr>
<tr>
<td></td>
<td>- R-on-T phenomenon often induces VT</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Defining Criteria per ECG</th>
<th>Key: QRS complexes display &quot;spindle-node&quot; pattern ➔ VT amplitude increases then decreases in regular pattern (creates the &quot;spindle&quot;) ➔ 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 &quot;node&quot;)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td><strong>Atrial Rate</strong>: cannot determine atrial rate</td>
</tr>
<tr>
<td></td>
<td><strong>Ventricular rate</strong>: 150-250 complexes/min</td>
</tr>
<tr>
<td></td>
<td><strong>Rhythm</strong>: only irregular ventricular rhythm</td>
</tr>
<tr>
<td></td>
<td><strong>PR</strong>: nonexistent</td>
</tr>
<tr>
<td></td>
<td><strong>P waves</strong>: nonexistent</td>
</tr>
<tr>
<td></td>
<td><strong>QRS complexes</strong>: display classic &quot;spindle-node&quot; pattern (see left column: &quot;Key&quot;)</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Clinical Manifestations</th>
<th>Majority of times patients with torsades have symptoms of decreased cardiac output (orthostasis, hypotension, syncope, exercise limitations, etc)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Asymptomatic torsades, sustained torsades, or &quot;stable&quot; torsades is uncommon</td>
</tr>
<tr>
<td></td>
<td>Tends toward sudden deterioration to pulseless VT or VF</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Common Etiologies</th>
<th>Most commonly occurs with prolonged QT interval, from many causes:</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>- Drug-induced: tricyclic antidepressants, procainamide, digoxin, some long-acting antihistamines</td>
</tr>
<tr>
<td></td>
<td>- Electrolyte and metabolic alterations (hypomagnesemia is the prototype)</td>
</tr>
<tr>
<td></td>
<td>- Inherited forms of long QT syndrome</td>
</tr>
<tr>
<td></td>
<td>- Acute ischemic events (see pathophysiology)</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Recommended Therapy</th>
<th>Review most recent 12-lead ECG (baseline):</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>- Measure QT interval just before onset of the polymorphic tachycardia</td>
</tr>
<tr>
<td></td>
<td>- QT interval prolongation? (if YES see below; if NO go to the polymorphic VT algorithm)</td>
</tr>
<tr>
<td>Long baseline QT interval:</td>
<td>..................................................................................................................</td>
</tr>
<tr>
<td>- Treat ischemia</td>
<td>- Correct electrolytes if abnormal</td>
</tr>
<tr>
<td>Then therapies (any one):</td>
<td>..................................................................................................................</td>
</tr>
<tr>
<td>- Magnesium</td>
<td>- Overdrive pacing</td>
</tr>
<tr>
<td>- Isoproterenol (pharmacologic overdrive pacing)</td>
<td>- Phenytoin</td>
</tr>
<tr>
<td>- Lidocaine</td>
<td>- Torsades de pointes</td>
</tr>
</tbody>
</table>

---

**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 QTc 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 QTc 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)
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

Type II second-degree AV block or Third-degree AV block?

Yes

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

Prepare for transvenous pacer
If symptoms develop, use transcutaneous pacemaker until transvenous pacer placed

Observe

No

Yes
### 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 | ■ **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 |
| Key: Regular P waves followed by regular QRS complexes at rate <60 beats/min  
| Note: Often a physical sign rather than an abnormal rhythm |
|---|---|
| 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 |

Sinus bradycardia: rate of 45 bpm; with borderline first-degree AV block (PR ≈ 0.20 sec)
17. First-Degree Heart Block

| Pathophysiology | ■ Impulse conduction is slowed (*partial block*) at the AV node by a fixed amount  
|                 | ■ Closer to being a physical sign than an abnormal arrhythmia |
| Defining Criteria per ECG | ■ Rate: First-degree heart block can be seen with both sinus bradycardia and sinus tachycardia  
| Key: PR interval >0.20 sec | ■ 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)**

<table>
<thead>
<tr>
<th>Pathophysiology</th>
</tr>
</thead>
<tbody>
<tr>
<td>Site of pathology: AV node</td>
</tr>
<tr>
<td>AV node blood supply comes from branches of the right coronary artery</td>
</tr>
<tr>
<td>Impulse conduction is increasingly slowed at the AV node (causing increasing PR interval)</td>
</tr>
<tr>
<td>Until one sinus impulse is completely blocked and a QRS complex fails to follow</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Defining Criteria per ECG</th>
</tr>
</thead>
<tbody>
<tr>
<td>Key: There is progressive lengthening of the PR interval until one P wave is not followed by a QRS complex (the dropped beat)</td>
</tr>
<tr>
<td><strong>Rate:</strong> atrial rate just slightly faster than ventricular (because of dropped beats); usually normal range</td>
</tr>
<tr>
<td><strong>Rhythm:</strong> regular for atrial beats; irregular for ventricular (because of dropped beats); can show regular P waves marching through irregular QRS</td>
</tr>
<tr>
<td><strong>PR:</strong> 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”)</td>
</tr>
<tr>
<td><strong>P waves:</strong> size and shape remain normal; occasional P wave not followed by a QRS complex (the “dropped beat”)</td>
</tr>
<tr>
<td><strong>QRS complex:</strong> ≤0.10 sec most often, but a QRS “drops out” periodically</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Clinical Manifestations—Rate-Related</th>
</tr>
</thead>
<tbody>
<tr>
<td>Due to bradycardia:</td>
</tr>
<tr>
<td><strong>Symptoms:</strong> chest pain, shortness of breath, decreased level of consciousness</td>
</tr>
<tr>
<td><strong>Signs:</strong> hypotension, shock, pulmonary congestion, CHF, angina</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Common Etiologies</th>
</tr>
</thead>
<tbody>
<tr>
<td>AV nodal blocking agents: β-blockers, calcium channel blockers, digoxin</td>
</tr>
<tr>
<td>Conditions that stimulate the parasympathetic system</td>
</tr>
<tr>
<td>An acute coronary syndrome that involves the right coronary artery</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Recommended Therapy</th>
</tr>
</thead>
<tbody>
<tr>
<td>Key: Treat only when patient has significant signs or symptoms that are due to the bradycardia</td>
</tr>
<tr>
<td>Intervention sequence for symptomatic bradycardia:</td>
</tr>
<tr>
<td><strong>Atropine</strong> 0.5 to 1 mg IV if vagal mechanism</td>
</tr>
<tr>
<td><strong>Transcutaneous pacing</strong> if available</td>
</tr>
<tr>
<td>If signs and symptoms are severe, consider catecholamine infusions:</td>
</tr>
<tr>
<td><strong>Dopamine</strong> 5 to 20 µg/kg per min</td>
</tr>
<tr>
<td><strong>Epinephrine</strong> 2 to 10 µg/min</td>
</tr>
<tr>
<td><strong>Isoproterenol</strong> 2 to 10 µg/min</td>
</tr>
</tbody>
</table>

![Second-degree heart block type I. Note progressive lengthening of PR interval until one P wave (arrow) is not followed by a QRS.](image-url)
### 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 ($\leq 0.10$ 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                                                   | Intervention sequence for bradycardia due to type II second-degree or third-degree heart block:  
|                                                                              | - Prepare for *transvenous* pacers  
|                                                                              | - 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: | обычно 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

<table>
<thead>
<tr>
<th>Rhythm Strip</th>
<th>Comments</th>
</tr>
</thead>
</table>
| **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: prepping 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)