ACLS Core Rhythms

Part 1—Recognition of Core ECG Arrest Rhythms

The Basics Figure 12 shows the anatomy of the cardiac conduction system and its relationship to the ECG cardiac cycle.

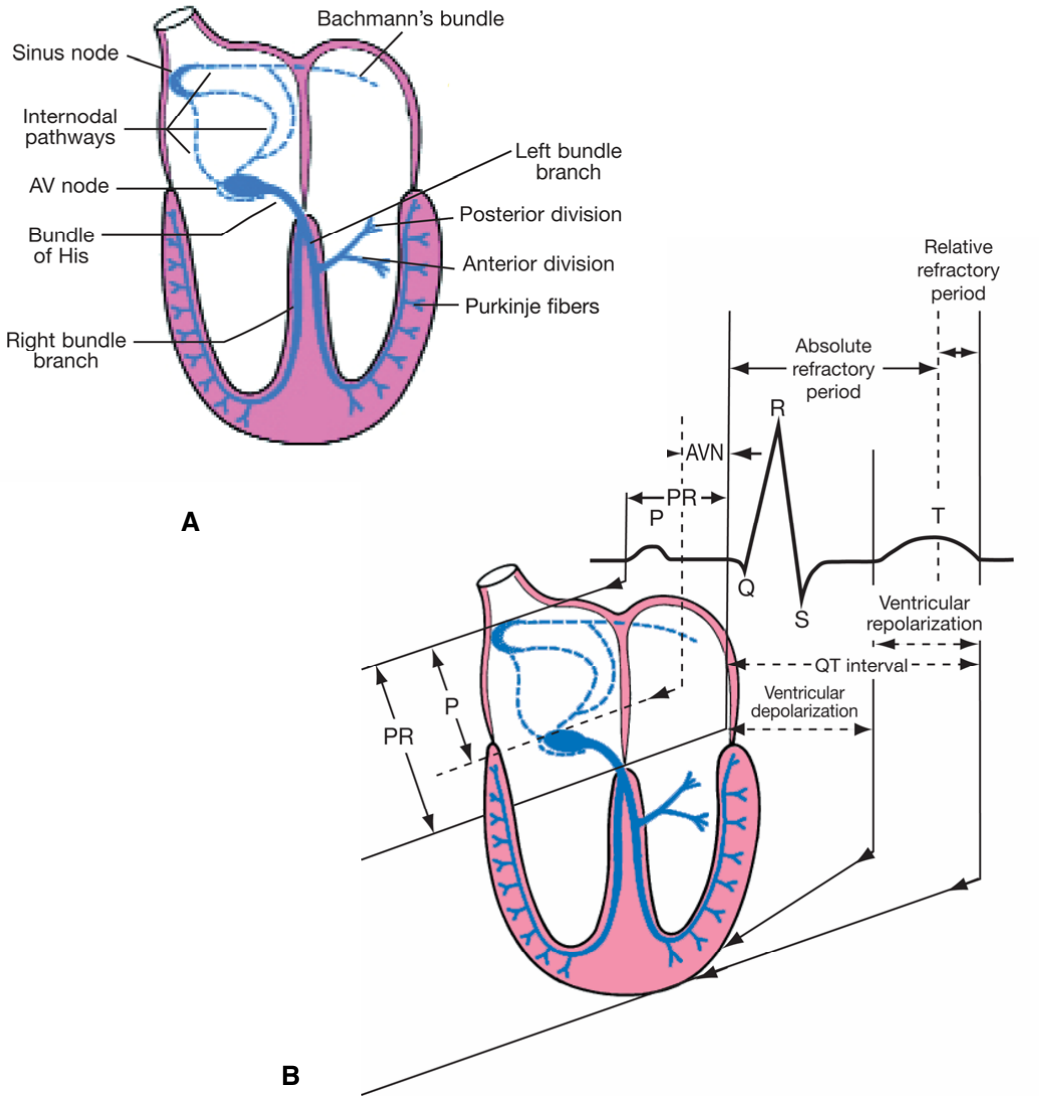


Figure 12. Anatomy of the cardiac conduction system: relationship to the ECG cardiac cycle. **A,** Heart: anatomy of conduction system. **B,** Relation of cardiac cycle to conduction system anatomy.

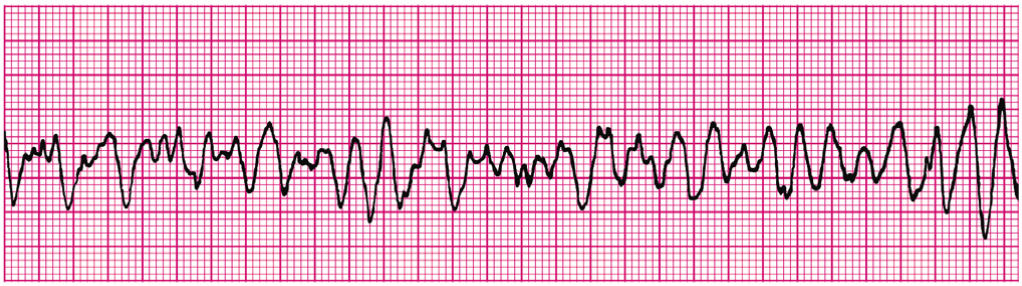
Cardiac Arrest Rhythms

The ECG rhythms for patients who are in cardiac arrest are

- Ventricular fibrillation (VF)/pulseless ventricular tachycardia (VT)
- Pulseless electrical activity (PEA)
- Asystole

These ECG rhythms are shown below:

Ventricular Fibrillation (Figure 13)	
Pathophysiology	• Ventricles consist of areas of normal myocardium alternating with areas of ischemic, injured, or infarcted myocardium, leading to a chaotic asynchronous pattern of ventricular depolarization and repolarization. Without organized ventricular depolarization the ventricles cannot contract as a unit and they produce no cardiac output. The heart "quivers" and does not pump blood.
Defining Criteria per ECG	• **Rate/QRS complex:** unable to determine; no recognizable P, QRS, or T waves. Baseline undulations occur between 150 and 500 per minute. • **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 or moderate* (5 to <10 mm), *coarse* (10 to <15 mm), or *very coarse* (>15 mm)
Clinical Manifestations	• Pulse disappears with onset of VF (the pulse may disappear before the onset of VF if a common precursor to VF, rapid VT, develops prior to the VF) • Collapse, unresponsiveness • Agonal gasps • Onset of *irreversible death*
Common Etiologies	• Acute coronary syndromes (ACS) leading to ischemic areas of myocardium • Stable to unstable VT, untreated • Premature ventricular complexes (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



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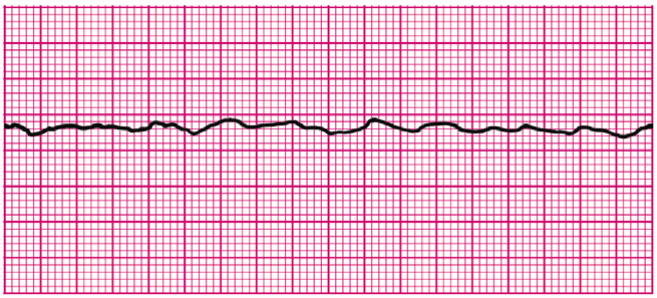

B

Figure 13. A, Coarse ventricular fibrillation. Note high-amplitude waveforms, which vary in size, shape, and rhythm, representing chaotic ventricular electrical activity. The ECG criteria for VF are as follows: (1) QRS complexes: no normal-looking QRS complexes are recognizable; a regular "negative-positive-negative pattern (Q-R-S) cannot be seen. (2) Rate: uncountable; electrical deflections are very rapid and too disorganized to count. (3) Rhythm: no regular rhythmic pattern can be discerned; the electrical waveforms vary in size and shape; the pattern is completely disorganized. **B,** Fine ventricular fibrillation. In comparison with Figure 13A, the amplitude of electrical activity is much reduced. Note the complete absence of QRS complexes. In terms of electrophysiology, prognosis, and the likely clinical response to attempted defibrillation, adrenergic agents, or antiarrhythmics, this rhythm pattern may be difficult to distinguish from that of asystole.

PEA	
Pathophysiology	• Cardiac conduction impulses occur in an organized pattern but do not produce myocardial contraction (this condition was formerly called electromechanical dissociation); or insufficient ventricular filling during diastole; or ineffective contractions
Defining Criteria per ECG	• Rhythm displays organized electrical activity (not VF/pulseless VT) • Usually not as organized as normal sinus rhythm • Can be narrow (QRS <0.10 mm) or wide (QRS >0.12 second); fast (>100 per minute) or slow (<60 per minute) • May be narrow (noncardiac etiology) or wide (often cardiac etiology) and can be slow (cardiac etiology) or fast (often

	noncardiac etiology).
Clinical Manifestations	• Collapse, unresponsive • Agonal gasps or apnea • No pulse detectable by palpation (very low systolic blood pressure could still be present in such cases termed *pseudo-PEA*)
Common Etiologies	Use the H's and T's mnemonic to recall possible causes of PEA: • Hypovolemia • Hypoxia • Hydrogen ion (acidosis) • Hypo-/hyperkalemia • Hypoglycemia • Hypothermia • Toxins ("tablets," ie, drug overdose, ingestion) • Tamponade, cardiac • Tension pneumothorax • Thrombosis, coronary (ACS) or pulmonary (embolism) • Trauma

Asystole (Figure 14)	
Defining Criteria per ECG Classically asystole presents as a "flat line"; defining criteria are virtually nonexistent	• **Rate:** no ventricular activity seen or ≤6 complexes per minute; so-called "P-wave asystole" occurs with only atrial impulses present (P waves) • **Rhythm:** no ventricular activity seen or ≤6 complexes per minute • **PR:** cannot be determined; occasionally P wave is seen, but by definition R wave must be absent • **QRS complex:** no deflections seen that are consistent with a QRS complex
Clinical Manifestations	• May have agonal gasps (early); unresponsive • No pulse or 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 (eg, electrocution, lightning strike) • May represent "stunning" of the heart immediately after defibrillation (shock deliver that eliminates VF), prior to resumption of spontaneous rhythm

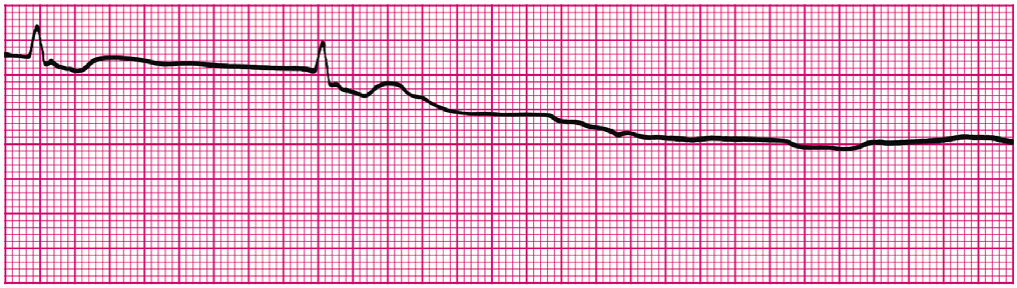

Figure 14. The "rhythm" of ventricular asystole. This patient is pulseless and unresponsive. Note the 2 QRS-like complexes at the start of this rhythm display. These complexes represent a minimum of electrical activity, probably ventricular escape beats. Does this pattern represent *pulseless electrical activity*? Note the long section in which electrical activity is completely absent. This patient is in asystole at this point.

Part 2—Recognition of Selected Nonarrest ECG Rhythms

Recognition of Supraventricular Tachyarrhythmias

Sinus Tachycardia (Figure 15)	
Pathophysiology	• None—more a physical sign than an arrhythmia or pathologic condition • Normal impulse formation and conduction
Defining Criteria and ECG Features	• **Rate:** >100 per minute • **Rhythm:** sinus • **PR:** usually <0.20 second • **P for every QRS Complex** • **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

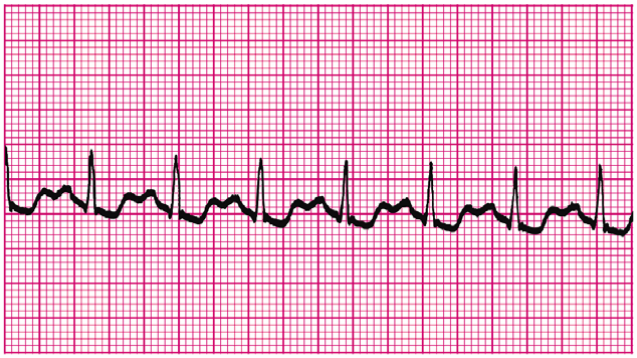

Figure 15. Sinus tachycardia.

<table>
<tr><th colspan="4">Atrial Fibrillation (Figure 16) and Atrial Flutter (Figure 17)</th></tr>
<tr><td>Pathophysiology</td><td colspan="3">• Atrial impulses faster than sinoatrial (SA node) impulses
• Atrial fibrillation: impulses take multiple, chaotic, random pathways through atria
• Atrial flutter: impulses take a circular course around atria, setting up flutter waves</td></tr>
<tr><td rowspan="5">Defining Criteria and ECG Features (Distinctions between atrial fibrillation and 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 atrial fibrillation.” This one is usually dependable. Can also be observed in multifocal atrial tachycardia (MAT).

Atrial Flutter Key: Flutter waves 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
• May be normal or slow if AV nodal conduction is abnormal (eg “sick sinus syndrome”)</td><td>• Atrial rate 220 to 350 per minute
• Ventricular response is a function of AV node block or conduction of atrial impulses
• Ventricular response rarely >150 to 180 beats because of AV nodal 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 4-to-1</td></tr>
<tr><td>P waves</td><td>• Chaotic atrial fibrillatory waves only
• Creates variable 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 to 0.12 second unless QRS complex is distorted by fibrillation or flutter waves or by conduction defects through ventricles</td></tr>
<tr><td>Clinical Manifestations</td><td colspan="3">• Signs and symptoms are a function of the rate of ventricular response to atrial fibrillation waves; “atrial fibrillation with rapid ventricular response” may be characterized by dyspnea on exertion (DOE), shortness of breath (SOB), and sometimes 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>

Common Etiologies	• Acute coronary syndromes, coronary artery disease, congestive heart failure • Disease at mitral or tricuspid valve • Hypoxia, acute pulmonary embolism • Drug-induced: digoxin or quinidine; β agonists, theophylline • Hypertension • Hyperthyroidism

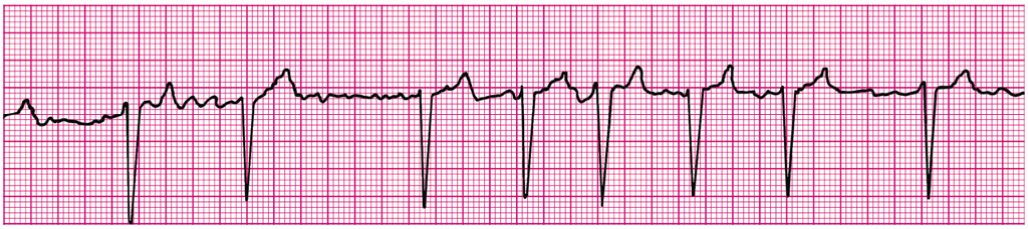

Figure 16. Atrial fibrillation.

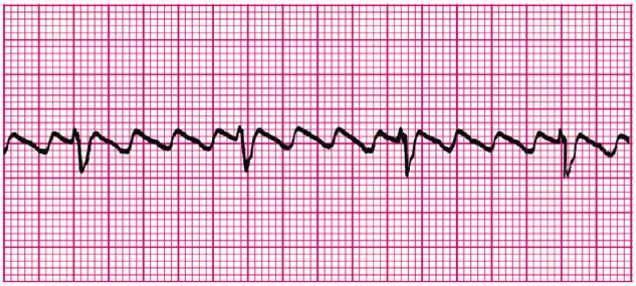

Figure 17. Atrial flutter.

Accessory-Mediated SVT (Figure 18); May include AV nodal reentrant tachycardia or AV reentry tachycardia.	
Pathophysiology	Reentry phenomenon: impulses recycle repeatedly in the AV node because an abnormal rhythm circuit allows a wave of depolarization to travel in a circle. Usually, the depolarization travels antegrade (forward) through the abnormal pathway and then circles back retrograde through the "normal" conduction tissue.
Defining Criteria and ECG Features **Key:** Regular, narrow-complex tachycardia without P waves and sudden onset or cessation **Note:** To merit the diagnosis of reentry SVT, some experts require capture of the abrupt onset or cessation on a monitor strip	**Rate:** exceeds upper limit of sinus tachycardia at rest (>120 to 130 per minute), seldom <150 per minute, often up to 250 per minute **Rhythm:** regular **P waves:** seldom seen because rapid rate causes P wave to be "hidden" in preceding T waves or to be difficult to detect because the origin is low in the atrium **QRS complex:** normal, narrow (usually ≤0.10 second)
Clinical Manifestations	• Palpitations felt by patient at onset; becomes anxious, uncomfortable • Exercise tolerance low with very high rates • Symptoms of unstable tachycardia may occur
Common Etiologies	• Accessory conduction pathway in many SVT patients • For such otherwise healthy people, many factors can provoke the reentry SVT: caffeine, hypoxia, cigarettes, stress, anxiety, sleep deprivation, numerous medications • Frequency of SVT increased in unhealthy patients with coronary artery disease, chronic obstructive pulmonary disease, and congestive heart failure

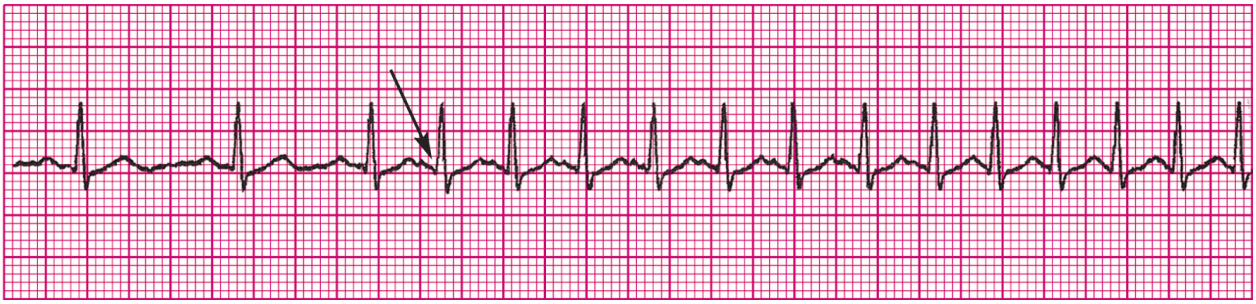

Figure 18. Sinus rhythm with a reentry supraventricular tachycardia (SVT).

Recognition of Ventricular Tachyarrhythmias

Monomorphic VT (Figure 19)	
Pathophysiology	• Impulse conduction is slowed around areas of ventricular injury, infarct, or ischemia • These areas also serve as sources 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 indicate *VT* ° VT <30 seconds duration is *nonsustained VT* ° VT >30 seconds duration is *sustained VT*	• **Rate:** ventricular rate >100 per minute; typically 120 to 250 per minute • **Rhythm:** regular ventricular rhythm • **PR:** absent (rhythm is AV dissociated) • **P waves:** seldom seen but present; VT is a form of AV dissociation, a defining characteristic for wide-complex tachycardias of ventricular origin versus supraventricular tachycardias with aberrant conduction • **QRS complex:** wide and bizarre, "PVC-like" complexes >0.12 seconds, with large T wave of opposite polarity from QRS • **Fusion beats**—Occasional chance capture of a conducted P wave. Resulting QRS "hybrid" complex, part normal, part ventricular • **Nonsustained VT—** lasts <30 seconds and does not require intervention
Clinical Manifestations	• Typically symptoms of decreased cardiac output (orthostasis, hypotension, syncope, exercise limitations, etc) do develop • Monomorphic VT can be asymptomatic despite widespread belief that sustained VT always produces symptoms • Untreated and sustained VT 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 relative refractory period of cardiac cycle ("R-on-T phenomenon") • Drug-induced, prolonged QT interval (tricyclic antidepressants, procainamide, digoxin, some long-acting antihistamines)

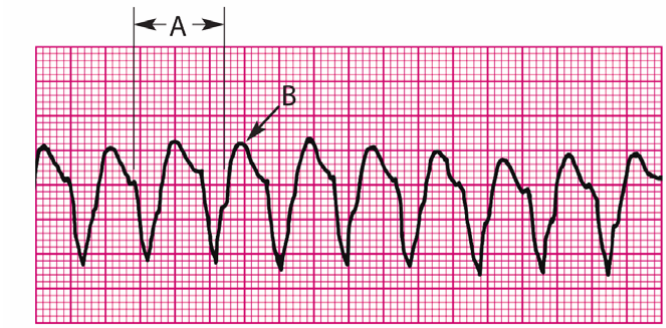

Figure 19. Monomorphic VT at a rate of 150 per minute: wide QRS complexes (arrow A) with opposite polarity T waves (arrow B).

Polymorphic VT (Figure 20)	
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 and thus are "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 QRS complexes	• **Rate:** ventricular rate >100 per minute; typically 120 to 250 per minute • **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 QRS complexes
Clinical Manifestations	• Typically will rapidly deteriorate to pulseless VT or VF • Symptoms of decreased cardiac output (orthostasis, hypotension, poor perfusion, syncope, etc) present before pulseless arrest • Seldom sustained VT
Common Etiologies	• Acute ischemic event (see Pathophysiology) with areas of "ventricular irritability" • PVCs that occur during relative refractory period of cardiac cycle ("R-on-T phenomenon") • Drug-induced prolonged QT interval (tricyclic antidepressants, procainamide, sotalol, amiodarone, ibutilide, dofetilide, some antipsychotics, digoxin, some long-acting antihistamines) • Hereditary long QT interval syndromes

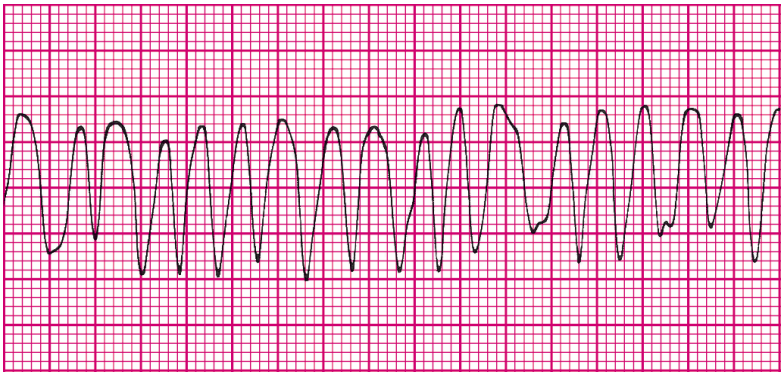

Figure 20. Polymorphic VT: QRS complexes display multiple morphologies.

Torsades de Pointes (a Unique Subtype of Polymorphic VT) (Figure 21)	
Pathophysiology	Specific pathophysiology of classic torsades: • QT interval is abnormally long (baseline ECG) (see the Maximum QT Interval table in the ECC Handbook) • Leads to increase in relative refractory period ("vulnerable period") of cardiac cycle. This increases probability that an irritable focus (PVC) will occur on T wave (vulnerable period or R-on-T phenomenon) • R-on-T phenomenon often induces VT
Defining Criteria per ECG **Key:** QRS complexes display a "spindle-node" pattern, in which VT amplitude increases and then decreases in a regular pattern (creating the "spindle"). The initial deflection at the start of one spindle (eg, negative) will be followed by complexes of opposite (eg, positive) polarity or deflection at the start of next spindle (creating the "node").	• **Atrial rate:** cannot determine • **Ventricular rate:** 150 to 250 complexes/min • **Rhythm:** only irregular ventricular rhythm • **PR:** nonexistent • **P waves:** nonexistent • **QRS complexes:** display classic spindle-node pattern (see "Key" at left)

Clinical Manifestations	• Tends toward sudden deterioration to pulseless VT or VF • Symptoms of decreased cardiac output are typical (orthostasis, hypotension, syncope, signs of poor perfusion, etc) • "Stable" torsades, sustained torsades is uncommon • Treated with unsynchronized high-energy (defibrillation) shocks
Common Etiologies	Most commonly occurs in patients with prolonged QT interval, due to many causes: • Drug-induced: tricyclic antidepressants, procainamide, sotalol, amiodarone, ibutilide, dofetilide, some antipsychotics, digoxin, some long-acting antihistamines • Electrolyte and metabolic alterations (hypomagnesemia is the prototype) • Inherited forms of long QT syndrome • Acute ischemic events (see Pathophysiology)

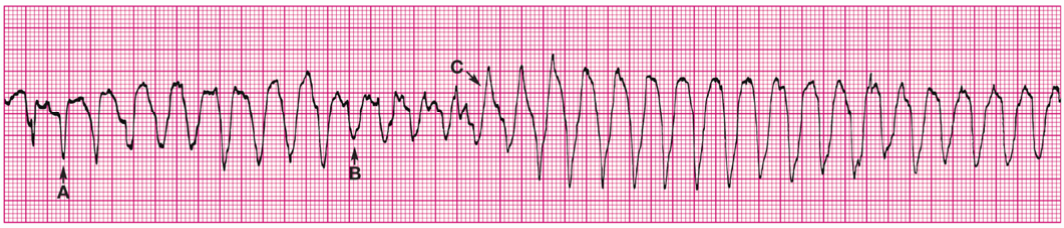

Figure 21. Torsades de pointes: a unique type of polymorphic VT. **A,** Start of a "spindle." Note negative initial deflection and increasing QRS amplitude. **B,** End of a spindle and start of a "node." **C,** End of a node and start of the next spindle. Note the positive initial deflection and "spindling" in QRS amplitude.

Recognition of Sinus Bradycardia

Sinus Bradycardia (Figure 22)	
Pathophysiology	• Impulses originate at SA node at a slow rate • May be physiologic • Can be a physical sign, as in sinus tachycardia
Defining Criteria per ECG **Key:** Regular P waves followed by regular QRS complexes at rate <60 per minute **Note:** Often a physical sign rather than an abnormal rhythm	• **Rate:** <60 per minute • **Rhythm:** regular sinus • **PR:** regular, <0.20 second • **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 second in absence of intraventricular conduction defect
Clinical Manifestations	• Usually asymptomatic at rest • With increased activity and sinus node dysfunction, a persistent slow rate can lead to symptoms of easy fatigue, shortness of breath, dizziness or lightheadedness, syncope, hypotension
Common Etiologies	• Can be normal for well-conditioned people • Vasovagal event, such as vomiting, Valsalva maneuver, rectal stimuli, inadvertent pressure on carotid sinus (“shaver’s syncope”) • Acute coronary syndromes that affect circulation to SA node (right coronary artery); most often inferior AMIs • Adverse drug effects, eg, β-blockers or calcium channel blockers, digoxin, quinidine

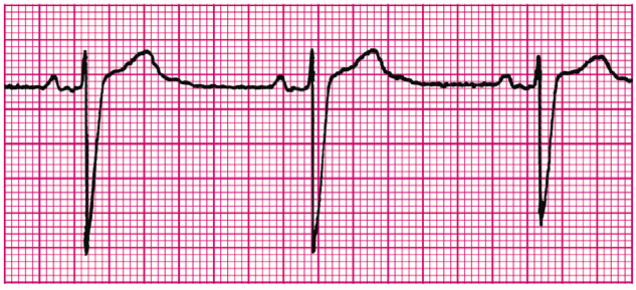

Figure 22. Sinus bradycardia.

Recognition of Atrioventricular (AV) Block

First-Degree AV Block (Figure 23)	
Pathophysiology	• Impulse conduction is slowed *(partial block)* at AV node for a fixed interval • May be a sign of another problem or a primary conduction abnormality
Defining Criteria per ECG **Key:** PR interval greater than 0.20 second	• **Rate:** first-degree heart block can be seen with rhythms with both sinus bradycardia and sinus tachycardia as well as a normal sinus mechanism • **Rhythm:** sinus, regular, both atria and ventricles • **PR:** prolonged, >0.20 second 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 P wave • **QRS complex:** narrow, ≤0.10 second in absence of intraventricular conduction defect
Clinical Manifestations	• Usually asymptomatic
Common Etiologies	• Many first-degree AV blocks are due to drugs, usually the AV nodal blockers: β-blockers, non-dihydropyridine calcium channel blockers, and digoxin • Any condition that stimulates the parasympathetic nervous system (eg, vasovagal reflex) • AMIs that affect circulation to the AV node (right coronary artery); most often inferior AMIs

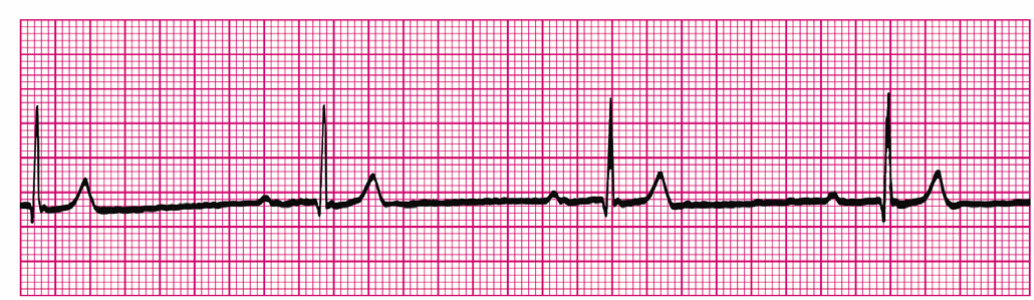

Figure 23. First-degree AV block.

Second-Degree Block Type I (Mobitz I–Wenckebach) (Figure 24)	
Pathophysiology	• Site of pathology: AV node • AV node blood supply comes from branches of right coronary artery (right dominant circulation) • Impulse conduction is progressively slowed at AV node (causing increasing PR interval) until one sinus impulse is completely blocked and QRS complex fails to follow
Defining Criteria per ECG **Key:** There is progressive lengthening of PR interval until one P wave is not followed by QRS complex (dropped beat).	• **Rate:** atrial rate just slightly faster than ventricular (because of dropped conduction); usually within normal range • **Rhythm:** atrial complexes are regular and ventricular complexes are irregular in timing (because of dropped beats); can see regular P waves marching through irregular QRS • **PR:** progressive lengthening of PR interval occurs from cycle to cycle; then one P wave is not followed by QRS complex ("dropped beat") • **P waves:** size and shape remain normal; occasional P wave not followed by QRS complex ("dropped beat") • **QRS complex:** ≤0.10 second most often, but a QRS "drops out" periodically
Clinical Manifestations—Rate-Related	**Due to bradycardia:** • Most often asymptomatic • **Symptoms:** chest pain, shortness of breath, decreased level of consciousness • **Signs:** hypotension, shock, pulmonary congestion, congestive heart failure (CHF), angina
Common Etiologies	• AV nodal blocking agents: β-blockers, non-dihydropyridine calcium channel blockers, digoxin • Conditions that stimulate the parasympathetic nervous system • Acute coronary syndrome that involves right coronary artery

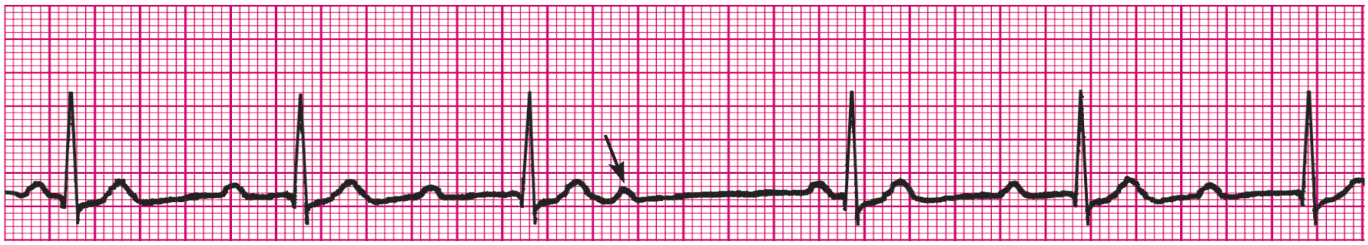

Figure 24. Second-degree AV block Type I. Note the progressive lengthening of the PR interval until one P wave (arrow) is not followed by a QRS.

Second-Degree AV Block Type II (Infranodal) (Mobitz II) (Figures 25)	
Pathophysiology	• The site of the block is most often below the AV node (infranodal) at the bundle of His (infrequent) or at bundle branches • Impulse conduction is normal through node, thus no first-degree block and no prior PR prolongation
Defining Criteria per ECG	• **Atrial rate:** usually 60 to 100 per minute • **Ventricular rate:** by definition (because of blocked impulses) slower than atrial rate • **Rhythm:** atrial = regular, ventricular = irregular (because of blocked impulses) • **PR:** constant and set; no progressive prolongation as with Mobitz Type I second-degree block—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 second) implies high block relative to AV node; wide (>0.12 second) implies low block relative to AV node
Clinical Manifestations—Rate-Related	**Due to bradycardia:** • **Symptoms:** chest pain, shortness of breath, decreased level of consciousness • **Signs:** hypotension, shock, pulmonary congestion, CHF, AMI
Common Etiologies	• Acute coronary syndrome that involves branches of *left* coronary artery

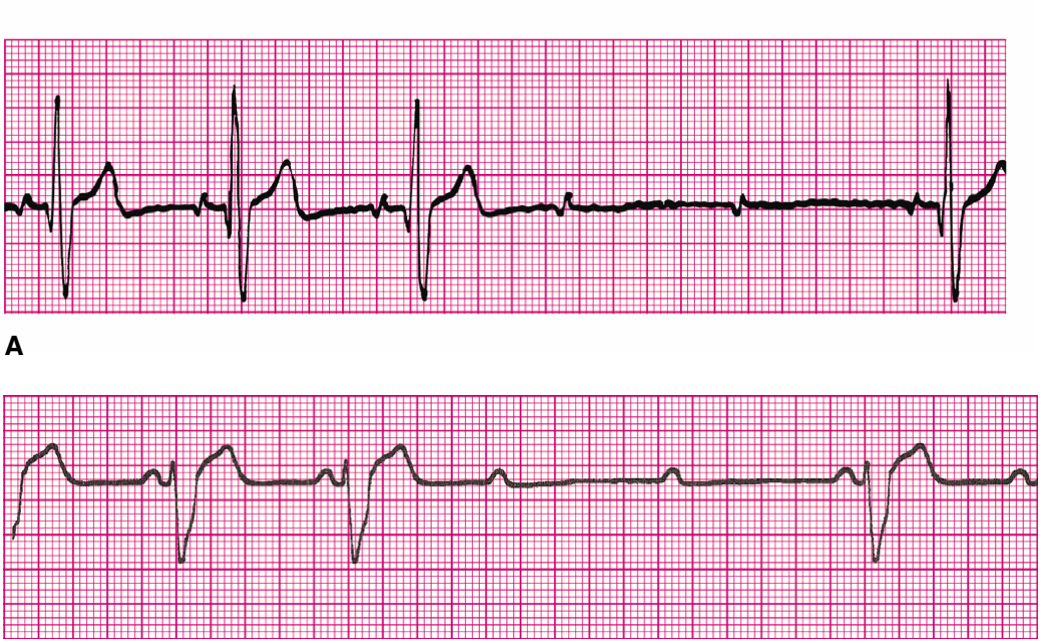

Figure 25. A, Type II (high block): regular PR-QRS intervals until 2 dropped beats occur; borderline normal QRS complexes indicate high nodal or nodal block. **B,** Type II (low block): regular PR-QRS intervals until dropped beats; wide QRS complexes indicate infranodal block.

Third-Degree AV Block and AV Dissociation (Figure 26)	
Pathophysiology **Pearl:** *AV dissociation* is the defining class; *third-degree* or *complete AV block* is one type of AV dissociation. By convention (outdated), if ventricular escape depolarization is faster than atrial rate, *AV dissociation* is present; if ventricular rate is slower than atrial rate, *third-degree AV block* is present.	• Injury or damage to 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," "supra-," or "junctional" nodal block) — Bundle of His — Bundle branches ("low-nodal" or "infranodal" block)
Defining Criteria per ECG **Key:** Third-degree	• **Atrial rate:** usually 60 to 100 per minute; impulses completely independent ("dissociated") from the slower ventricular rate • **Ventricular rate:** depends on rate of ventricular escape beats that arise:

block (see Pathophysiology) causes atria and ventricles to depolarize independently, with no relationship between the two (AV dissociation).	— Ventricular escape rate slower than atrial rate = third-degree AV block (rate = 20 to 40 per minute) — Ventricular escape rate faster than atrial rate = AV dissociation (rate = 40 to 55 per minute) • **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 second) implies high block relative to AV node; wide (>0.12 second) implies low block relative to AV node
Clinical Manifestations—Rate-Related	**Due to bradycardia:** • **Symptoms:** chest pain, shortness of breath, decreased level of consciousness • **Signs:** hypotension, shock, pulmonary congestion, CHF, AMI
Common Etiologies	• Acute coronary syndrome that involves branches of *left* coronary artery In particular, involves left anterior descending (LAD) artery and branches to interventricular septum (supply bundle branches)

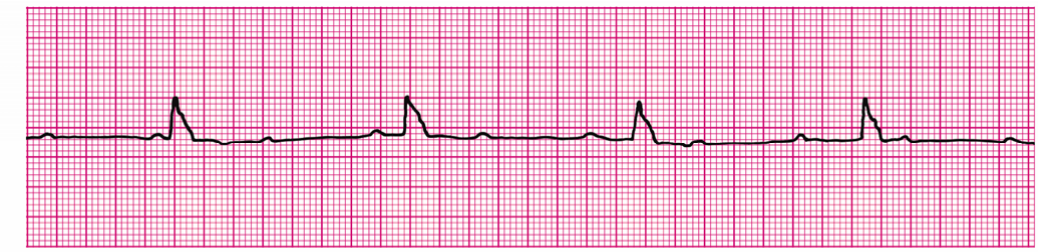

Figure 26. Third-degree AV block: regular P waves at 50 to 55 per minute; regular ventricular "escape beats" at 35 to 40 per minute; no relationship between P waves and escape beats.