MENU

Chapter 6
Ventricular Rhythms

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Ventricular Rhythms

- Objectives

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Overview

- Ventricles are the heart's least efficient pacemaker
  - Normally generate impulses at a rate of 20 to 40 beats/min
- May assume responsibility for pacing the heart if:
  - The SA node fails to discharge
  - An impulse from the SA node is generated but blocked as it exits the SA node
  - The rate of discharge of SA node is slower than that of ventricles
  - An irritable site in either ventricle produces an early beat or rapid rhythm

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Ventricular Depolarization

- Ventricular beats and rhythms may originate from any part of the ventricles
- Typically characterized by QRS complexes that are abnormally shaped and prolonged (longer than 0.12 second)

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Ventricular Repolarization

- Because ventricular depolarization is abnormal, ventricular repolarization is also abnormal
  - Results in changes in ST segments and T waves
- T waves are usually in a direction opposite that of the QRS complex

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Premature Ventricular Complexes (PVCs)

- Arise from an irritable focus within either ventricle
- A PVC:
  - Is premature, occurring earlier than the next expected sinus beat
  - QRS is typically equal to or greater than 0.12 second
    - PVC depolarizes ventricles prematurely and in an abnormal manner
  - T wave is usually in the opposite direction of the QRS complex

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PVCs – Compensatory Pause

- A full compensatory pause often follows a PVC
- To determine whether a pause following a PVC is compensatory or noncompensatory:
  - Measure the distance between 3 normal beats
  - Compare that distance between 3 beats, one of which includes the PVC

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Compensatory/Noncompensatory Pause

- The pause is noncompensatory (incomplete) if the normal beat following the PVC occurs before it was expected
  - When the distance is not the same

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Compensatory/Noncompensatory Pause

- The pause is compensatory (or complete) if the normal beat following the PVC occurs when expected
  - When the distance is the same

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PVCs – Patterns

- Pairs (couplets): two sequential PVCs
- Runs or bursts: three or more sequential PVCs are called “ventricular tachycardia” (VT)
- Bigeminal PVCs (ventricular bigeminy): every other beat is a PVC
- Trigeminal PVCs (ventricular trigeminy): every third beat is a PVC
- Quadrigeminal PVCs (ventricular quadrigeminy): every fourth beat is a PVC

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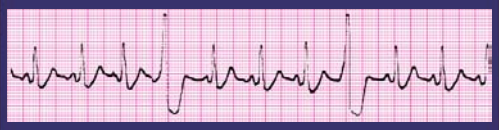
Uniform PVCs

- Premature ventricular beats that look the same in the same lead and originate from the same anatomical site (focus)



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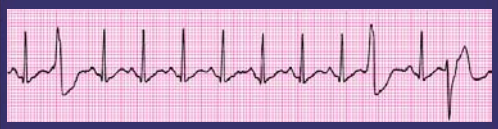
Multifocal PVCs

- PVCs that appear different from one another in the same lead
- Often (but not always) arise from different anatomical sites



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Terminology

- The terms “unifocal” and “multifocal” are sometimes used to describe PVCs that are similar or different in appearance
- Uniform PVCs are unifocal, but multiform PVCs are not necessarily multifocal

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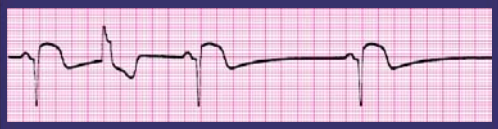
Interpolated PVCs

- Do not have a full compensatory pause
- “Squeezed” between two regular complexes without disturbing the underlying rhythm



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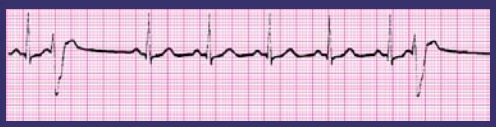
R-on-T PVCs

- Occur when the R wave of a PVC falls on the T wave of the preceding beat
- A PVC occurring during this period of the cardiac cycle may precipitate VT or VF



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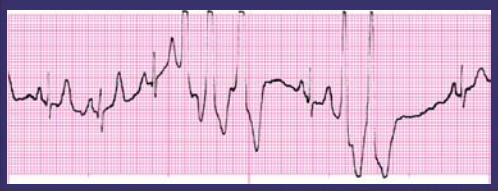
Paired PVCs

- A pair of PVCs occurring in immediate succession is called a “couplet” or “paired PVCs”
  - 3 or more PVCs occurring in immediate succession at a rate of more than 100/min are called a “salvo,” “run,” or “burst” of VT



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PVCs – ECG Characteristics

Rate	Usually within normal range, but depends on underlying rhythm
Rhythm	Essentially regular with premature beats. If the PVC is an interpolated PVC, the rhythm will be regular.
P Waves	Usually absent or, with retrograde conduction to the atria, may appear after the QRS (usually upright in the ST segment or T wave)
PR Interval	None with the PVC because the ectopic originates in the ventricles
QRS Duration	Greater than 0.12 second, wide and bizarre, T wave frequently in opposite direction of QRS

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PVCs – Causes

- PVCs can occur in healthy persons with apparently normal hearts and with no apparent cause
- Incidence of PVCs increases with age

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PVCs – Causes

- Normal variant
- Hypoxia
- Stress, anxiety
- Exercise
- Digitalis toxicity
- Acid-base imbalance
- Myocardial ischemia
- Electrolyte imbalance
  - Hypokalemia
  - Hypocalcemia
  - Hypercalcemia
  - Hypomagnesemia
- Congestive heart failure
- Increased sympathetic tone
- Acute myocardial infarction
- Stimulants
  - Alcohol
  - Caffeine
  - Tobacco
- Medications
  - Sympathomimetics
  - Cyclic antidepressants
  - Phenothiazines

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PVCs – Clinical Significance

- PVCs may or may not produce palpable pulses
- Patients may be asymptomatic or complain of:
  - Palpitations
  - "Racing heart"
  - Skipped beats
  - Chest or neck discomfort
- If the PVCs are frequent, signs of decreased cardiac output may be present

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PVCs – Intervention

- Treatment of PVCs is dependent on the:
  - Cause
  - Patient's signs and symptoms
  - Clinical situation
- Most patients experiencing PVCs do not require treatment with antidysrhythmic medications

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Ventricular Escape Beat

- A ventricular ectopic beat that occurs after a pause in which the supraventricular pacemakers failed to initiate an impulse
  - QRS measures 0.12 second or greater
  - Occur late in the cardiac cycle, appearing after the next expected sinus beat
  - A "protective" mechanism
    - Protects the heart from more extreme slowing or even asystole

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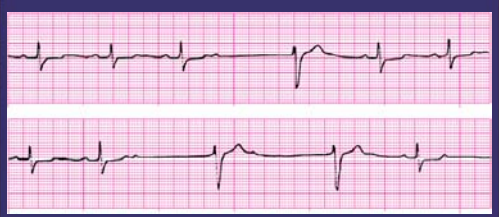
Ventricular Escape Beat



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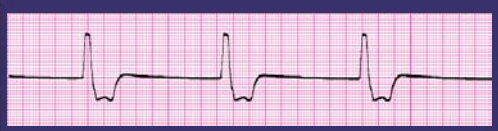
Idioventricular Rhythm (IVR)

- A ventricular escape or "idioventricular" rhythm (IVR) is three or more sequential ventricular escape beats occurring at a rate of 20 to 40 beats/min



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Idioventricular Rhythm – Causes

- May occur when:
  - The SA node and the AV junction fail to initiate an electrical impulse
  - The rate of discharge of the SA node or AV junction becomes less than the inherent ventricular rate
  - Impulses generated by a supraventricular site are blocked
- May also occur because of:
  - Myocardial infarction
  - Digitalis toxicity
  - Metabolic imbalances

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Idioventricular Rhythm – Clinical Significance

- Possible signs and symptoms due to the slow ventricular rate:
  - Severe hypotension
  - Weakness
  - Disorientation
  - Lightheadedness
  - Loss of consciousness

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Idioventricular Rhythm – Intervention

- Avoid lidocaine!
  - May abolish ventricular activity, possibly causing asystole
- If the patient is symptomatic because of the slow rate and/or loss of atrial kick:
  - Atropine may be ordered
  - Transcutaneous pacing (TCP) may be attempted

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Idioventricular Rhythm – Intervention

- Pulseless electrical activity (PEA)
  - No palpable pulse despite organized electrical activity observed on a cardiac monitor (other than VT)
- Interventions
  - CPR
  - Oxygen administration
  - Endotracheal intubation
  - IV access
  - Aggressive search for underlying cause of PEA

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Ventricular Escape Beats – ECG Characteristics

Rate	Usually within normal range, but depends on underlying rhythm
Rhythm	Essentially regular with late beats. The ventricular escape beat occurs after the next expected sinus beat.
P Waves	Usually absent or, with retrograde conduction to the atria, may appear after the QRS (usually upright in the ST segment or T wave)
PR Interval	None with the escape beat because the ectopic originates in the ventricles
QRS Duration	Greater than 0.12 second, wide and bizarre, T wave frequently in opposite direction of QRS

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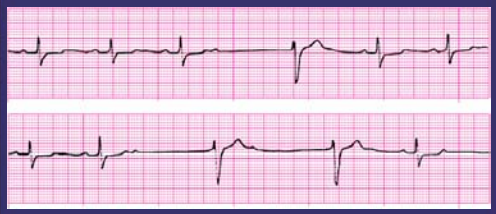
Ventricular Escape Beats



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IVR – ECG Characteristics

Rate	20 to 40 beats/min
Rhythm	Essentially regular
P Waves	Usually absent or, with retrograde conduction to atria, may appear after QRS (usually upright in ST segment or T wave)
PR Interval	None
QRS Duration	Greater than 0.12 second, T wave frequently in opposite direction of QRS

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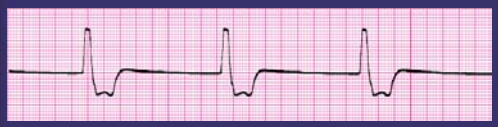
Idioventricular Rhythm



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Accelerated Idioventricular Rhythm (AIVR)

- AIVR exists when three or more sequential ventricular escape beats occur at a rate of 41 to 100 beats/min
  - Some cardiologists consider the ventricular rate range of AIVR to be 41 to 120 beats/min
- Also called “nonparoxysmal VT” or “idioventricular tachycardia”

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AIVR – ECG Characteristics

Rate	41 to 100 beats/min
Rhythm	Essentially regular
P Waves	Usually absent or, with retrograde conduction to atria, may appear after QRS (usually upright in ST segment or T wave)
PR Interval	None
QRS Duration	Greater than 0.12 second, T wave frequently in opposite direction of QRS

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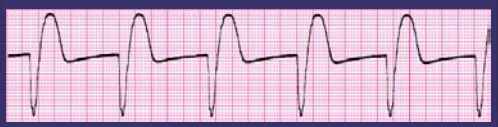
Accelerated Idioventricular Rhythm



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AIVR – Causes & Clinical Significance

- Usually considered a benign escape rhythm
  - Appears when the sinus rate slows and disappears when the sinus rate speeds up
- Often seen during first 12 hours of MI
- Common after successful reperfusion therapy

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AIVR – Intervention

- Treatment unnecessary if patient is asymptomatic
- If the patient is symptomatic because of the loss of atrial kick:
  - Atropine may be ordered
  - Transcutaneous pacing may be attempted
- Avoid lidocaine
  - AIVR is protective and often transient, spontaneously resolving on its own

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Ventricular Tachycardia (VT)

- VT exists when three or more PVCs occur in immediate succession at a rate higher than 100 beats/min
  - Non-sustained VT
    - A short run lasting less than 30 seconds
  - Sustained VT
    - Persists for more than 30 seconds
- VT may occur with or without pulses
- Patient may be stable or unstable

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Ventricular Tachycardia (VT)

- VT may originate from an ectopic focus in either ventricle
  - The QRS complex is wide and bizarre
  - P waves, if visible, bear no relationship to QRS complex
  - The ventricular rhythm is usually regular but may be slightly irregular

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Ventricular Tachycardia (VT)

- Monomorphic VT
  - QRS complexes are of the same shape and amplitude
- Polymorphic VT
  - QRS complexes vary in shape and amplitude

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Ventricular Tachycardia – Causes

- Sustained monomorphic VT is often associated with underlying heart disease, particularly myocardial ischemia
  - Rarely occurs in patients without underlying structural heart disease

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Ventricular Tachycardia – Other Causes

- Cardiomyopathy
- Cyclic antidepressant overdose
- Digitalis toxicity
- Valvular heart disease
- Mitral valve prolapse
- Trauma
  - Myocardial contusion
  - Invasive cardiac procedures
- Acid-base imbalance
- Electrolyte imbalance
  - Hypokalemia
  - Hyperkalemia
  - Hypomagnesemia
- Increased production of catecholamines
  - Fright
  - Cocaine abuse

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Ventricular Tachycardia – Clinical Significance

- Signs and symptoms vary
- Syncope may occur because of an abrupt onset of VT
  - The patient's only warning symptom may be a brief period of lightheadedness

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Ventricular Tachycardia – Clinical Significance

- Signs and symptoms of hemodynamic compromise related to the tachycardia may include:
  - Shock
  - Chest pain
  - Hypotension
  - Shortness of breath
  - Pulmonary congestion
  - Congestive heart failure
  - Acute myocardial infarction
  - Decreased level of consciousness

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Ventricular Tachycardia – Intervention

- Treatment is based on the patient's presentation
- Stable but symptomatic patients are treated with:
  - Oxygen therapy
  - IV access
  - Administration of ventricular antidysrhythmics to suppress the rhythm

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Ventricular Tachycardia – Intervention

- Unstable patient with a pulse
  - Usually a sustained heart rate of 150 beats/min or more
- If signs and symptoms are a result of rapid rate:
  - Administer oxygen
  - IV access
  - Sedate (if awake and time permits)
  - Electrical therapy
- If the patient is pulseless:
  - Begin CPR until a defibrillator is available

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Ventricular Tachycardia – Intervention

- When unclear whether a regular, wide-QRS tachycardia is VT or SVT with an intraventricular conduction delay, treat the rhythm as VT until proven otherwise.

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Monomorphic VT – ECG Characteristics

Rate	101 to 250 beats/min
Rhythm	Essentially regular
P Waves	May be present or absent. If present, they have no set relationship to QRS complexes, appearing between QRSs at a rate different from that of the VT
PR Interval	None
QRS Duration	Greater than 0.12 second; often difficult to differentiate between QRS and T wave

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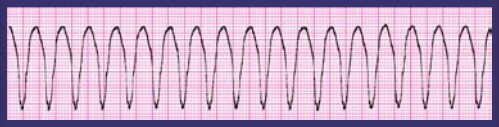
Monomorphic VT



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Polymorphic Ventricular Tachycardia

- "Polymorphic VT" refers to a rapid ventricular dysrhythmia with beat-to-beat changes in the shape and amplitude of the QRS complexes
- Two classifications of polymorphic VT
  - Long QT syndrome (LQTS)
    - Acquired (iatrogenic)
    - Congenital (idiopathic)
  - Normal QT

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Polymorphic Ventricular Tachycardia

- Polymorphic VT that occurs in the presence of a normal QT interval is simply referred to as "polymorphic VT"
- Polymorphic VT that occurs in the presence of a long QT interval is called "torsade de pointes"

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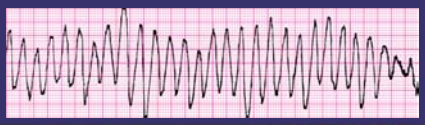
Torsade de Pointes (TdP)

- A dysrhythmia intermediary between VT and VF
- A type of polymorphic VT associated with a prolonged QT interval
- French for "twisting of the points"
  - QRS changes shape, amplitude, and width
  - Appears to "twist" around the isoelectric line, resembling a spindle



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Torsade de Pointes (TdP)

- Ventricular rate typically between 200 and 250 beats/min
  - May range from 150 to 300 beats/min
- Characterized by two or more cycles of QRS complexes with alternating polarity
  - QRS complexes twist from upright to negative or negative to upright and back

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Torsade de Pointes – ECG Characteristics

Rate	150 to 300 beats/min, typically 200 to 250 beats/min
Rhythm	May be regular or irregular
P Waves	None
PR Interval	None
QRS Duration	Greater than 0.12 second; gradual alteration in amplitude and direction of QRS

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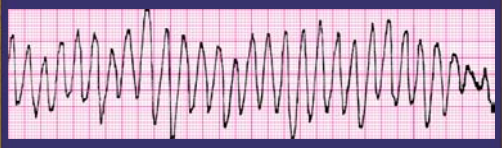
Torsade de Pointes



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Torsade de Pointes – Causes

- May be precipitated by slow heart rates
- Associated with medications or electrolyte disturbances that prolong the QT interval
- A prolonged QT interval may be congenital or acquired
- Lengthening of the QT interval may be the only warning sign suggesting impending TdP

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Torsade de Pointes – Clinical Significance

- Symptoms are usually related to the decreased cardiac output that occurs because of the fast ventricular rate
- Patients may complain of palpitations and lightheadedness or may experience a syncopal episode or seizures

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Torsade de Pointes – Clinical Significance

- May be initiated by a PVC
- May occasionally terminate spontaneously and recur after several seconds or minutes
- May deteriorate to VF

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Torsade de Pointes – Intervention

- Obtain a 12-lead ECG
  - Rhythm may appear to be monomorphic VT in one lead but present the pattern typical of TdP in another
- Treatment includes:
  - Discontinuation of type IA antidysrhythmics (if drug-induced)
  - Overdrive pacing or administration of one of the following:
    - Magnesium, isoproterenol, lidocaine, phenytoin
- Defibrillation may be necessary for termination of sustained episodes of TdP

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Ventricular Fibrillation (VF)

- VF is a chaotic rhythm that originates in the ventricles
- No organized depolarization of the ventricles
  - Ventricular myocardium quivers
  - No effective myocardial contraction and no pulse
  - Resulting rhythm is irregularly irregular with chaotic deflections that vary in shape and amplitude
  - No normal-looking waveforms are visible

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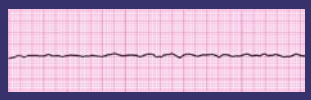
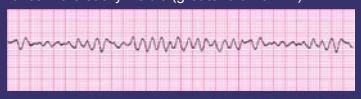
Ventricular Fibrillation

- Fine VF
  - Low amplitude waves (less than 3 mm)



- Coarse VF
  - Waves more easily visible (greater than 3 mm)



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Ventricular Fibrillation

- Because artifact can mimic VF, always check the patient's pulse before beginning treatment for VF
- The patient in VF is unresponsive, apneic, and pulseless

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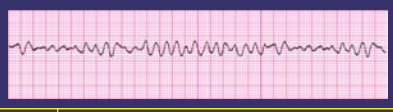
VF – ECG Characteristics



Rate	Cannot be determined because there are no discernible waves or complexes to measure
Rhythm	Rapid and chaotic with no pattern or regularity
P Waves	Not discernible
PR Interval	Not discernible
QRS Duration	Not discernible

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VF – Causes

- Extrinsic factors
  - Increased sympathetic nervous system activity
  - Vagal stimulation
  - Metabolic abnormalities
    - Hypokalemia
    - Hypomagnesemia
  - Antidysrhythmics and other medications
    - Psychotropics
    - Digitalis
    - Sympathomimetics
  - Environmental factors
    - Electrocution
- Intrinsic factors
  - Hypertrophy
  - Ischemia
  - Myocardial failure
  - Enhanced AV conduction
    - Bypass tracts
    - "Fast" AV node
  - Abnormal repolarization
  - Bradycardia

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VF – Intervention

- Begin CPR until a defibrillator is available
- On arrival of the defibrillator, deliver unsynchronized shocks
- Perform endotracheal intubation, establish IV access
- Administer medications per current resuscitation guidelines

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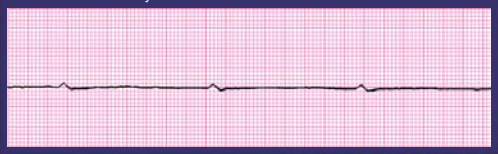
Asystole (Cardiac Standstill)

- Asystole is a total absence of ventricular electrical activity
  - There is no ventricular rate or rhythm, no pulse, and no cardiac output
- Some atrial electrical activity may be evident
  - "P-wave" asystole



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Asystole – ECG Characteristics

Rate	Ventricular usually not discernible but atrial activity may be observed ("P-wave" asystole)
Rhythm	Ventricular not discernible but atrial may be discernible
P Waves	Usually not discernible
PR Interval	Not measurable
QRS Duration	Absent

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Asystole – Causes

PATCH-4-MD

- Pulmonary embolism
- Acidosis
- Tension pneumothorax
- Cardiac tamponade
- Hypovolemia
- Hypoxia
- Heat/cold (hypothermia/hyperthermia)
- Hypokalemia/hyperkalemia (and other electrolytes)
- Myocardial infarction
- Drug overdose/accidents (cyclic antidepressants, calcium channel blockers, beta-blockers, digoxin)

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Asystole – Intervention

- Confirm the absence of a pulse
- Perform immediate CPR
- Confirm the rhythm in two leads
- Perform endotracheal intubation, IV access
- Consider possible causes of the rhythm
- Consider early transcutaneous pacing
- Medication therapy per current resuscitation guidelines