MEDAVIE HealthEd ÉduSanté

3-Lead Rhythm Interpretation

Primary Care Paramedicine

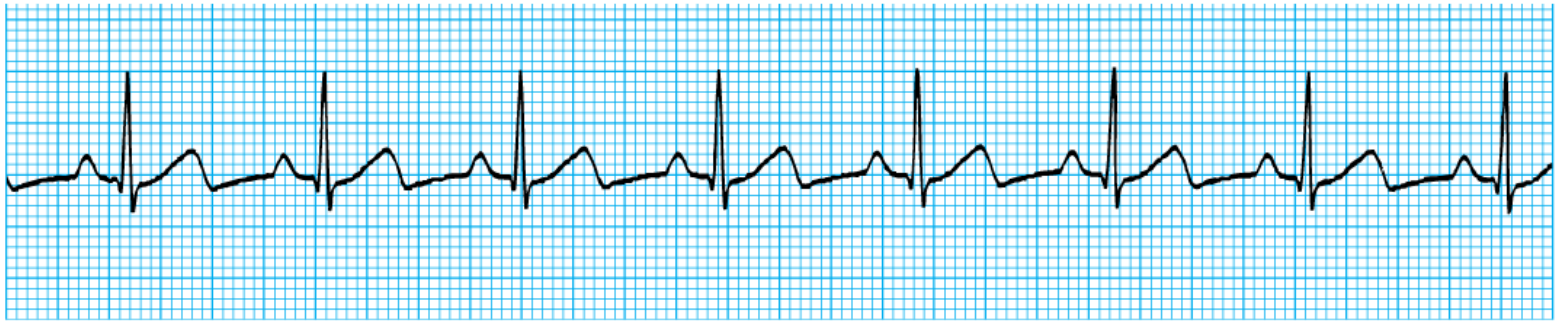
Module: 04

Section: 02b

3-Lead Rhythm Interpretation

RHYTHMS

Normal Sinus Rhythm



- Rate
  - 60–100 bpm
- Rhythm
  - Regular
- P waves
  - Normal, upright, only before each QRS complex
- PR Interval
  - 0.12–0.20 seconds
- QRS Complex
  - Normal, duration of <0.12 seconds

Dysrhythmias

- Any rhythm that is not Normal Sinus Rhythm is called a **Dysrhythmia**
- Not all dysrhythmias require treatment. Many times patients live with known dysrhythmias
- Some dysrhythmias, however can be life threatening and require immediate intervention

Causes of Dysrhythmias

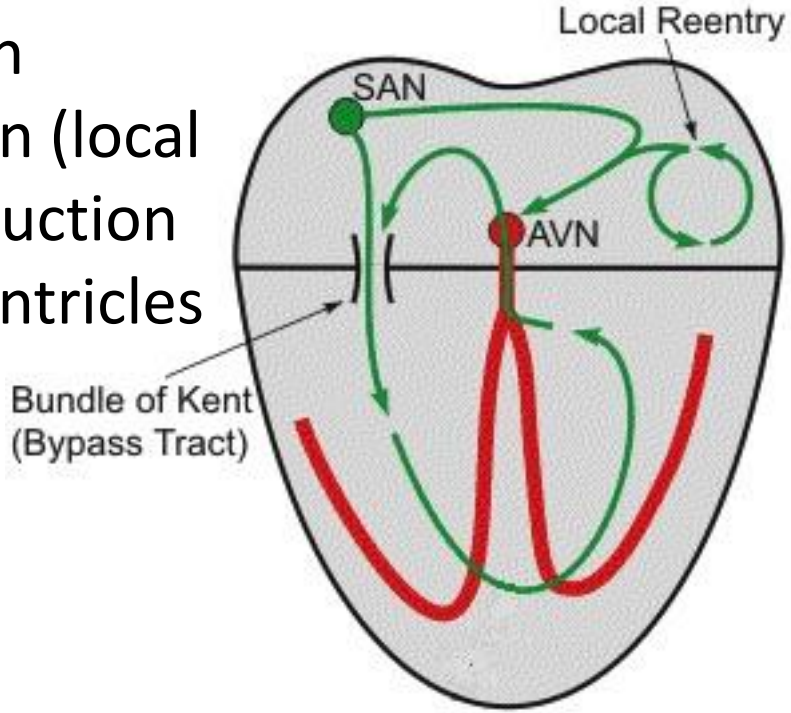
- Some common causes of dysrhythmias are:
  - Myocardial Ischemia, Necrosis, or Infarction
  - Autonomic Nervous System Imbalance
  - Distention of the Chambers of the Heart
  - Blood Gas Abnormalities
  - Electrolyte Imbalances
  - Trauma to the Myocardium
  - Drug Effects and Drug Toxicity
  - Electrocution
  - Hypothermia
  - CNS Damage
  - Idiopathic Events

Impulse Formation

- Normally, cardiomyocytes in the conduction pathway of the heart conduct the impulse for depolarization
- However at times, other cardiomyocytes outside the conduction pathway can also form an impulse
  - When this occurs, these cardiomyocytes are known as **ectopic foci**
  - These irritable cells can produce a single extra beat = **ectopic beat**

Impulse Formation

- Other than ectopic foci, another impulse abnormality that can occur is re-entry
- Re-entry
  - Disease or ischemia produces two branches of a pathway
  - Impulses re-enter conduction pathway in opposite direction (local re-entry) or via a direct conduction path from the atria to the ventricles (global re-entry)



Classification of Dysrhythmias

- When interpreting rhythms, dysrhythmias are classified based on:
  - Nature of origin
    - Changes in automaticity versus disturbances in conduction
  - Magnitude
    - Major versus minor
  - Severity
    - Life threatening versus non-life threatening
  - Site of origin

Classification by Site of Origin

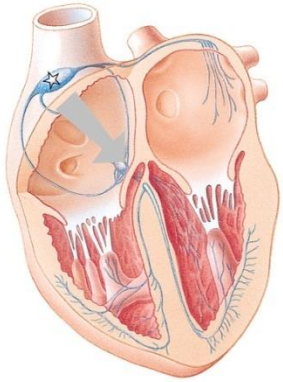
- Dysrhythmias originating in the SA Node
- Dysrhythmias originating in the atria
- Dysrhythmias originating within the AV junction
- Dysrhythmias originating in the ventricles
- Dysrhythmias resulting from disorders of conduction

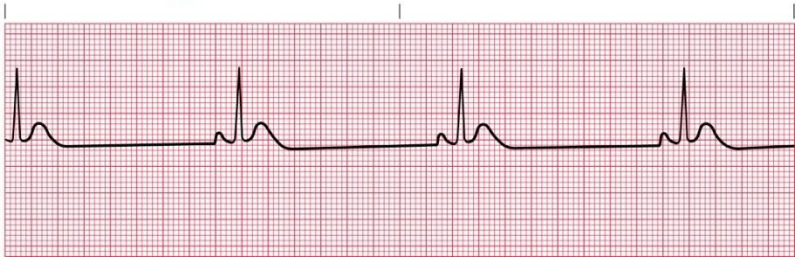
Dysrhythmias Originating in the SA Node

- Sinus Bradycardia
- Sinus Tachycardia
- Sinus Dysrhythmia
- Sinus Arrest

Sinus Bradycardia

Rules of Interpretation **Sinus Bradycardia**	
Rate	***Less than 60***
Rhythm	***Regular***
Pacemaker Site	***SA node***
P Waves	***Upright & normal***
PRI	***Normal***
QRS	***Normal***



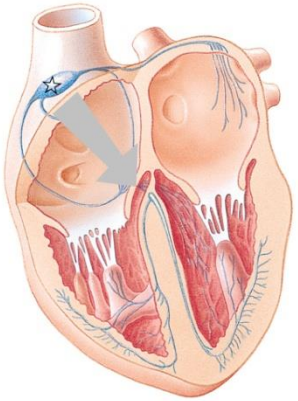


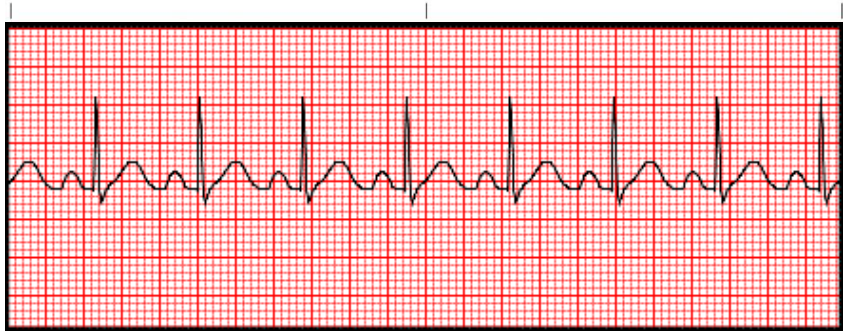
Sinus Bradycardia

- Etiology
  - Increased parasympathetic (vagal) tone, intrinsic disease of the SA node, drug effects
  - May be a normal finding in healthy, well-conditioned persons
- Clinical Significance
  - May result in decreased cardiac output, hypotension, angina, or CNS symptoms
  - In healthy, well-conditioned person, may have no significance
- Treatment
  - Generally unnecessary unless hypotension or ventricular irritability is present

Sinus Tachycardia

Rules of Interpretation **Sinus Tachycardia**	
Rate	***Greater than 100***
Rhythm	***Regular***
Pacemaker Site	***SA node***
P Waves	***Upright & normal***
PRI	***Normal***
QRS	***Normal***



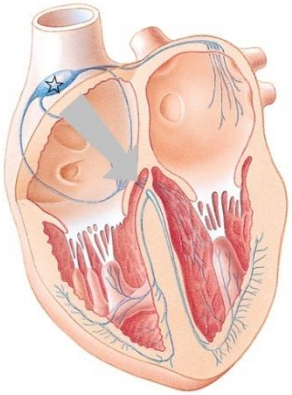


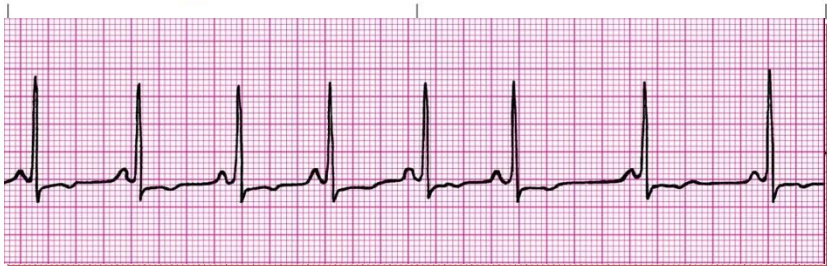
Sinus Tachycardia

- Etiology
  - Results from an increased rate of SA node discharge.
  - Potential causes include exercise, fever, anxiety, hypovolemia, anemia, pump failure, increased sympathetic tone, hypoxia, or hypothyroidism
- Clinical Significance
  - Decreased cardiac output for rates >140
  - Very rapid rates can precipitate ischemia or infarct
- Treatment
  - Treatment is directed at the underlying cause

Sinus Dysrhythmia

Rules of Interpretation **Sinus Dysrhythmia**	
Rate	***60–100***
Rhythm	***Irregular***
Pacemaker Site	***SA node***
P Waves	***Upright & normal***
PRI	***Normal***
QRS	***Normal***



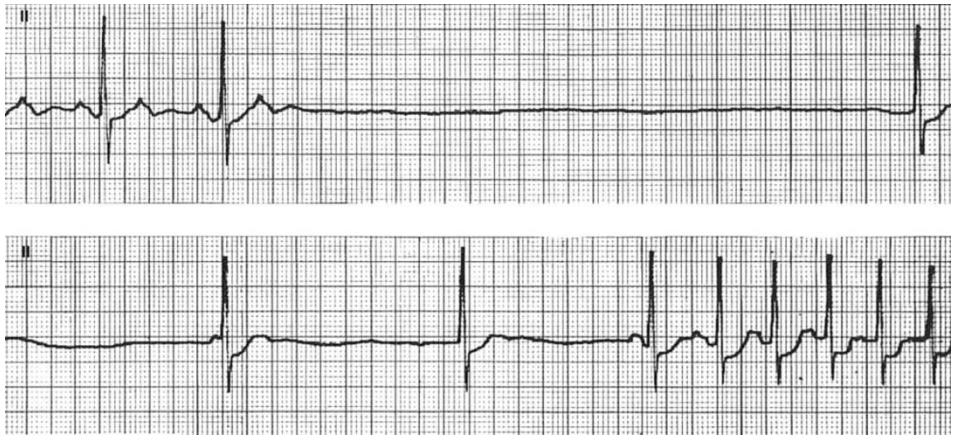


Sinus Dysrhythmia

- Etiology
  - Often a normal finding, sometimes related to the respiratory cycle
  - May be caused by enhanced vagal tone
- Clinical Significance
  - Normal variant
- Treatment
  - Typically, none required

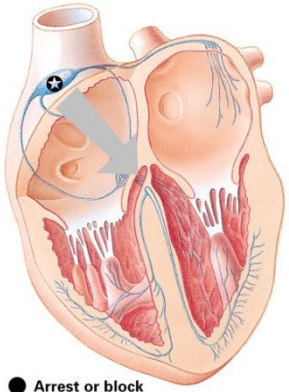
Sick Sinus Syndrome

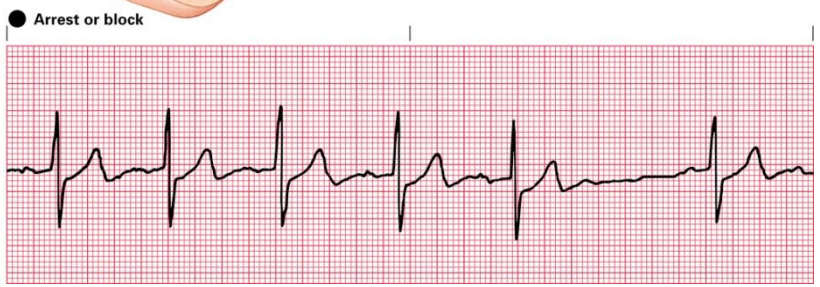
- Bradycardia-tachycardia syndrome
  - Symptomatic vs asymptomatic depends on how tachy/brady the patient becomes
  - May include:
    - Chest pain or palpitations
    - Confusion or other changes in mental status
    - Fainting or near-fainting, fatigue
    - Shortness of breath
- Treatment
  - Symptomatic
    - requires a permanent pacemaker if symptoms relate to bradycardic portion
    - Tachycardia treated with medications



Sinus Arrest

Rules of Interpretation **Sinus Arrest**	
Rate	***Normal to slow***
Rhythm	***Irregular***
Pacemaker Site	***SA node***
P Waves	***Upright & normal***
PRI	***Normal***
QRS	***Normal***





Sinus Arrest

- Etiology
  - Occurs when the sinus node fails to discharge
  - May result from ischemia of the SA node, digitalis toxicity, excessive vagal tone, or degenerative fibrotic disease
- Clinical Significance
  - Frequent or prolonged episodes may decrease cardiac output and cause syncope
  - Prolonged episodes may result in escape rhythms
- Treatment
  - None if patient is asymptomatic
  - Treat symptomatic bradycardia

Dysrhythmias Originating in the Atria

- Supraventricular Tachycardia / Paroxysmal Supraventricular Tachycardia
  - Atrial Flutter
  - Atrial Fibrillation

Supraventricular Tachycardia

- SVT is the name given to any extreme tachycardia that originates above the ventricles (specifically above the bundle of His)
- High rate of QRS complexes can make it difficult to determine whether P waves are present
- Narrow width QRS complexes confirm supraventricular origin
- Rhythm can be regular or irregular

Supraventricular Tachycardia

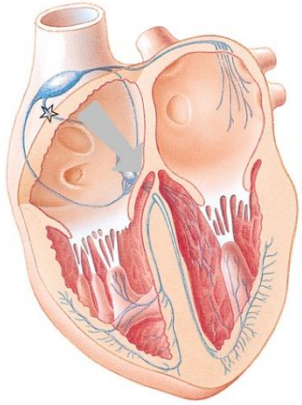
- Although SVT is a general name for a group of tachycardias, when a patient experiences SVT in an abrupt onset/offset pattern it is referred to as Paroxysmal Supraventricular Tachycardia (pSVT)
  - Typically this is the result of re-entry

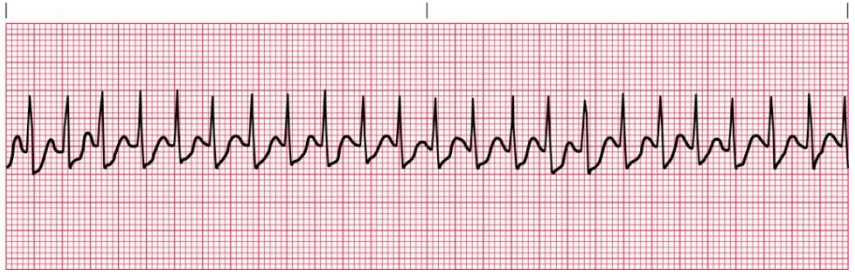
Supraventricular Tachycardia

- Rhythms that fall under the classification of SVT are:
  - Multifocal Atrial Tachycardia
  - Atrial Tachycardia
  - Atrial Flutter
  - Atrial Fibrillation

Supraventricular Tachycardia

Rules of Interpretation **Supraventricular Tachycardia**	
Rate	***Tachycardic >150bpm***
Rhythm	***Regular***
Pacemaker Site	***Varies among the SA node, atrial tissue, and AV Junction***
P Waves	***Absent***
PRI	***Unable to determine***
QRS	***Normal or narrow***





Supraventricular Tachycardia

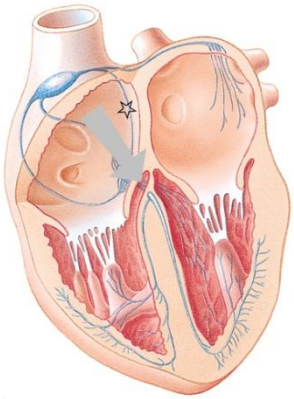
- Etiology
  - Rapid atrial depolarization overrides the SA node
  - May be precipitated by stress, overexertion, smoking, caffeine
- Clinical Significance
  - May be tolerated well by healthy patients for short periods
  - Marked reduction in cardiac output can precipitate angina, hypotension, or congestive heart failure

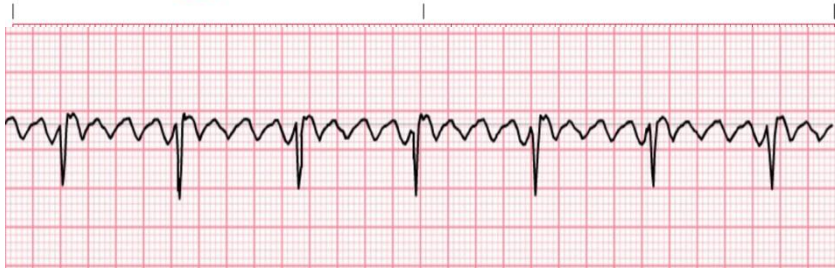
Supraventricular Tachycardia

- ACP Treatment
  - Vagal Manoeuvres
  - Pharmacological Therapy
    - Adenosine
    - Verapamil
  - Electrical Therapy
    - Consider if patient symptomatic with HR > 150.
    - Synchronized cardioversion starting at 100J

Atrial Flutter

Rules of Interpretation **Atrial Flutter**	
Rate	***Atrial rate 200-400*** ***Ventricular rate varies***
Rhythm	***Usually regular***
Pacemaker Site	***Atrial (outside SA node)***
P Waves	***Flutter waves are present***
PRI	***Usually normal when impulse is conducted***
QRS	***Usually normal***





Atrial Flutter

- Atrial Flutter produces classic “flutter wave” representing the multiple P waves between QRS complexes
  - Referred to as the “saw-tooth pattern”
- Typically atrial impulses are conducted through to the ventricles in a repeated ratio
  - Recorded as the # of P waves : # of QRS
    - Ex. 2:1, 3:1, 4:1

Atrial Flutter

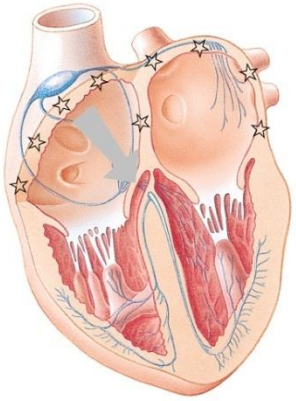
- Etiology
  - Results when the AV node cannot conduct all the impulses
    - Re-entry circuit in R atrium
  - Impulses may be conducted in fixed or variable ratios.
  - Usually associated with organic disease such as congestive heart failure (rarely seen with MI)
- Clinical Significance
  - Generally well tolerated
  - Rapid ventricular rates may compromise cardiac output and result in symptoms
  - May occur in conjunction with atrial fibrillation

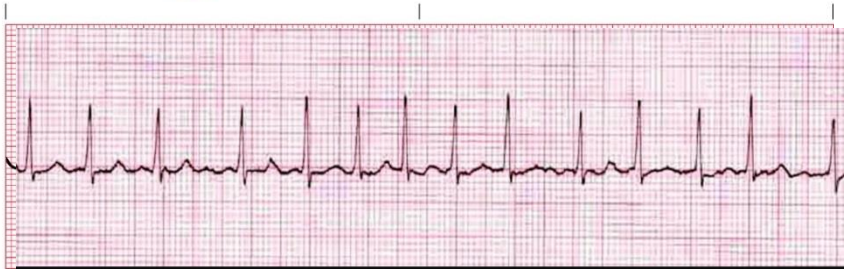
Atrial Flutter

- ACP Treatment
  - Electrical Therapy
    - Consider if ventricular rate > 150 and symptomatic
    - Synchronized cardioversion starting at 100J
  - Pharmacological Therapy
    - Sodium Channel Blockers (Procainamide)
    - Beta Blockers (Metoprolol)
    - Calcium Channel Blockers (Diltiazem or Verapamil)
    - Cardiac glycosides (Digoxin)

Atrial Fibrillation

Rules of Interpretation **Atrial Fibrillation**	
Rate	***Atrial rate 400-600 (though not discernible)*** ***Ventricular rate varies***
Rhythm	***Irregularly irregular***
Pacemaker Site	***Multiple ectopic atrial foci***
P Waves	***None discernible***
PRI	***None***
QRS	***Normal***





Atrial Fibrillation

- With AF, due to such a high atrial rate, no discernable P waves are present
- Depending on conduction of impulses through to the ventricles, ventricular rate is irregular and can vary
  - When ventricular rate is tachycardic = AF with rapid ventricular response

Atrial Fibrillation

- Etiology
  - Results from multiple ectopic foci; AV conduction is random and highly variable
  - Often associated with underlying heart disease, metabolic disturbances and toxicological emergencies
- Clinical Significance
  - Atria fail to contract effectively, reducing cardiac output
  - Well tolerated when ventricular rates are normal
  - High or low ventricular rates can result in cardiac compromise
  - Most common sustained dysrhythmia

Atrial Fibrillation

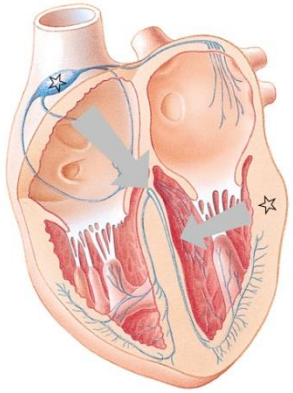
- ACP Treatment
  - Electrical Therapy
    - Consider if ventricular rate > 150 and symptomatic
    - Synchronized cardioversion starting at 100J
  - Pharmacological Therapy
    - Sodium Channel Blockers (Procainamide)
    - Beta Blockers (Metoprolol)
    - Calcium Channel Blockers (Diltiazem or Verapamil)
    - Cardiac glycosides (Digoxin)
    - Anticoagulant (Heparin or warfarin)

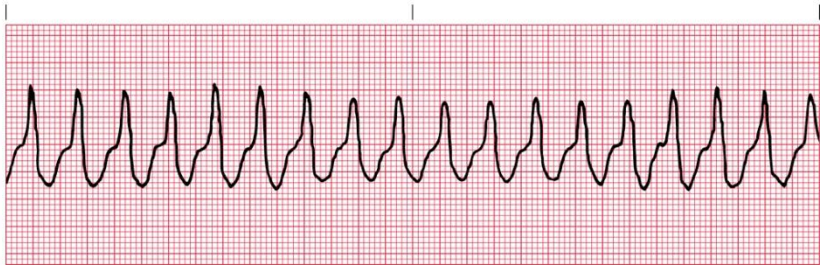
Dysrhythmias Originating in the Ventricles

- Ventricular Tachycardia
  - Torsade de Pointes
- Ventricular Fibrillation

Ventricular Tachycardia

Rules of Interpretation **Ventricular Tachycardia**	
Rate	***100–250***
Rhythm	***Usually regular***
Pacemaker Site	***Ventricle***
P Waves	***None***
PRI	***None***
QRS	***>0.12 seconds***





Ventricular Tachycardia

- Etiology
  - 3 or more ventricular escape complexes in succession at a rate of >100 bpm
  - Causes include myocardial ischemia, increased sympathetic tone, hypoxia, idiopathic causes, acid–base disturbances, or electrolyte imbalances
  - VT may appear monomorphic or polymorphic
  - If rhythm ceases before 30 seconds it is referred to as a run of Vtach
  - If rhythm persists for > 30 seconds it is referred to as sustained VTach

Ventricular Tachycardia

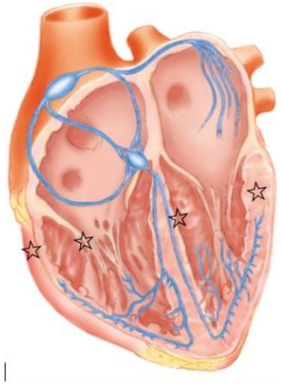
- Etiology
  - 3 or more ventricular escape complexes in succession at a rate of >100 bpm
  - Causes include myocardial ischemia, increased sympathetic tone, hypoxia, idiopathic causes, acid–base disturbances, or electrolyte imbalances
  - VT may appear monomorphic or polymorphic
- Clinical Significance
  - Decreased cardiac output, possibly to life-threatening levels
  - May deteriorate into ventricular fibrillation

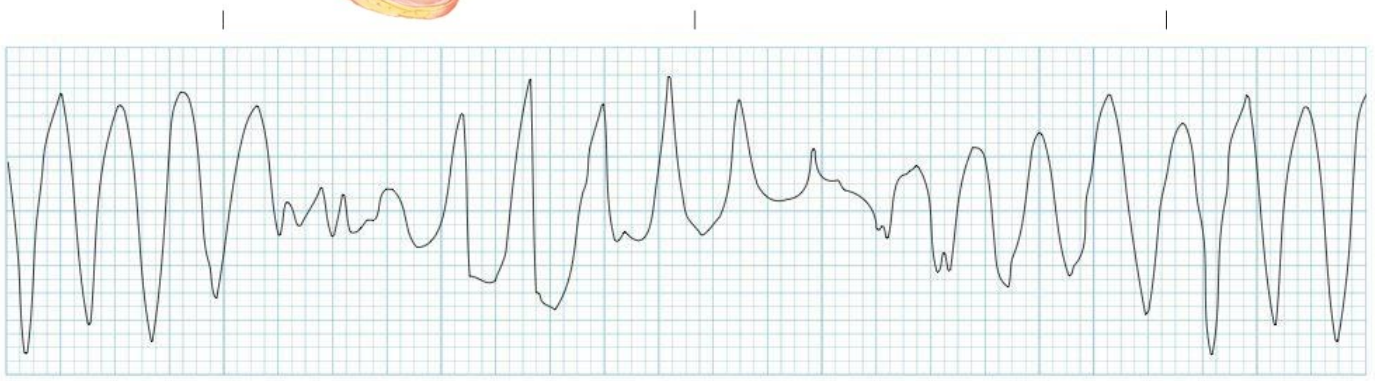
Ventricular Tachycardia

- Clinical Significance
  - Decreased cardiac output, possibly to life-threatening levels
  - May deteriorate into ventricular fibrillation
- Treatment
  - Perfusing patient
    - Administer oxygen and establish IV access
    - ACP may consider immediate synchronized cardioversion starting at 100J for hemodynamically unstable patients
    - ACP may administer Amiodarone 150–300 mg IV
  - Non-perfusing patient
    - Follow ventricular fibrillation protocol

Torsade de Pointes

- Polymorphic VT
- Caused by the use of certain antiarrhythmics



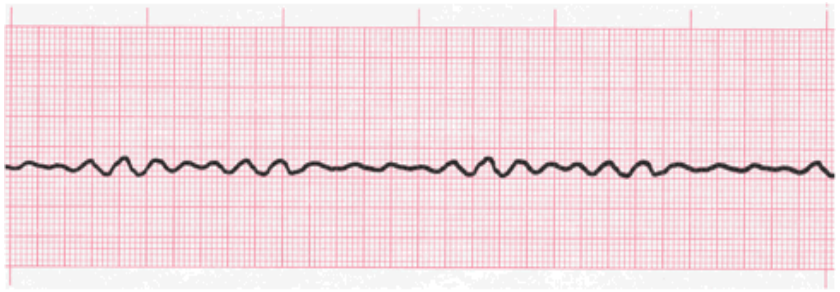


Torsade de Pointes

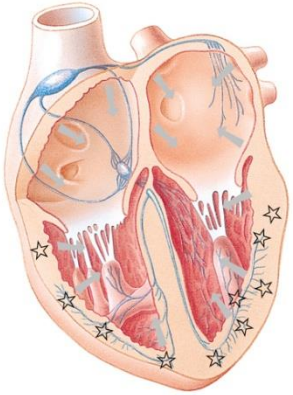
- Typically occurs in non-sustained bursts
  - Prolonged Q–T interval during breaks
  - QRS rates from 166–300
  - RR interval highly variable
- ACP Treatment
  - Administer magnesium sulfate 1–2 g diluted in 100 ml D_5W over 1–2 minutes
  - Amiodarone 150–300 mg is also effective

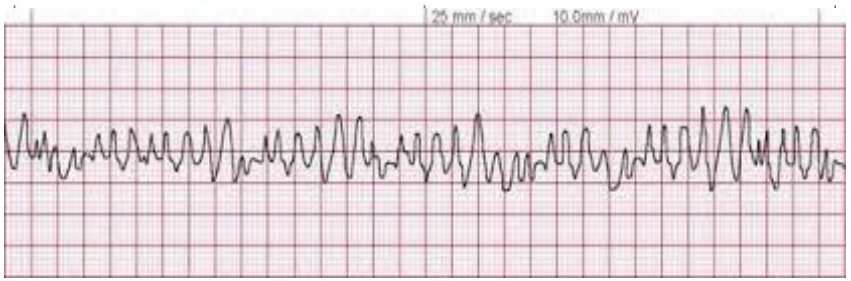
Ventricular Fibrillation

Rules of Interpretation **Ventricular Fibrillation**	
Rate	**No organized rhythm**
Rhythm	**No organized rhythm**
Pacemaker Site	**Numerous ventricular foci**
P Waves	**Usually absent**
PRI	**None**
QRS	**None**



Fine VF





Coarse VF

Ventricular Fibrillation

- Etiology
  - Wide variety of causes, often resulting from advanced coronary artery disease
- Clinical Significance
  - Lethal dysrhythmia with no organized electrical pattern, therefore no mechanical squeeze and no cardiac output and

Ventricular Fibrillation

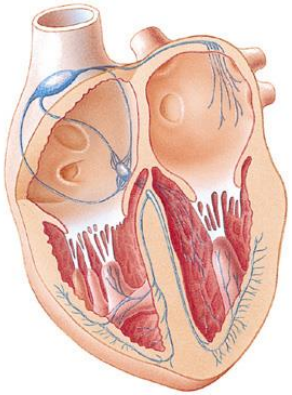
- Treatment
  - Initiate CPR
  - Defibrillate with 200J (360J if monophasic)
  - Control the airway and establish IV access
  - ACP may consider
    - Epinephrine 1:10,000 every 3–5 minutes
    - second-line drugs
      - Lidocaine (1.0 mg/kg 1st dose, 0.5 mg/kg 2nd dose q10 min to a max of 3.0 mg/kg)
      - May consider bretylium, amiodarone, procainamide, or magnesium sulfate (if torsades)

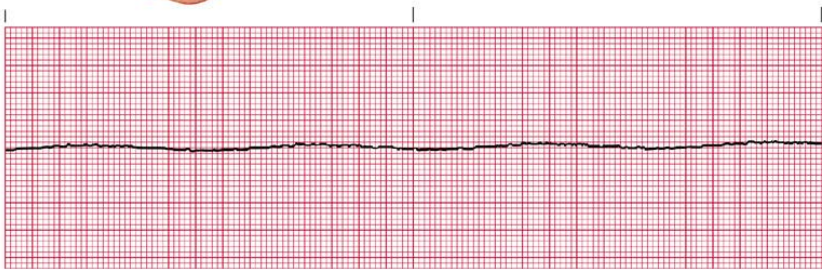
Other Dysrhythmias

- Asystole
- PEA
- Pacemaker Rhythms

Asystole

Rules of Interpretation **Asystole**	
Rate	***No Electrical Activity***
Rhythm	***No Electrical Activity***
Pacemaker Site	***No Electrical Activity***
P Waves	***Absent***
PRI	***Absent***
QRS	***Absent***





Asystole

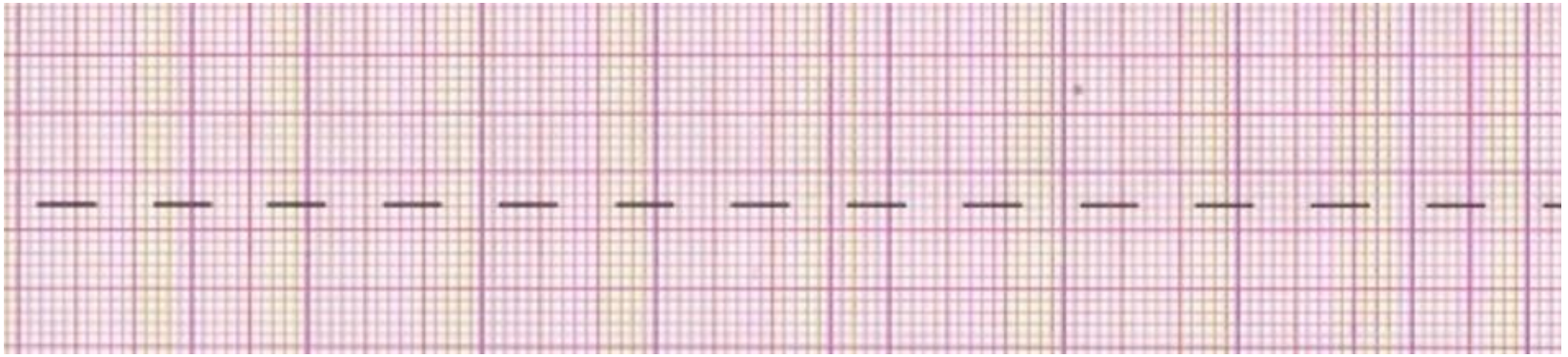
- Etiology
  - Primary event in cardiac arrest, resulting from massive myocardial infarction, ischemia, and necrosis
  - Final outcome of ventricular fibrillation
- Clinical Significance
  - Asystole = cardiac arrest
  - Poor prognosis for resuscitation

Asystole

- Treatment
  - Administer CPR and manage the airway
  - Treat for ventricular fibrillation if there is any doubt about the underlying rhythm
  - ACP may administer medications
    - Epinephrine, atropine, and possibly sodium bicarbonate

Asystole

- Don't panic when you see this:



- This represents an electrode (or electrodes) that are not connected
- Troubleshoot your setup as this is not asystole!

Pulseless Electrical Activity

- Characteristics
  - Electrical impulses are present, but with no accompanying mechanical contractions of the heart
  - Treat the patient, not the monitor
  - If pulseless initiate cardiac arrest protocols

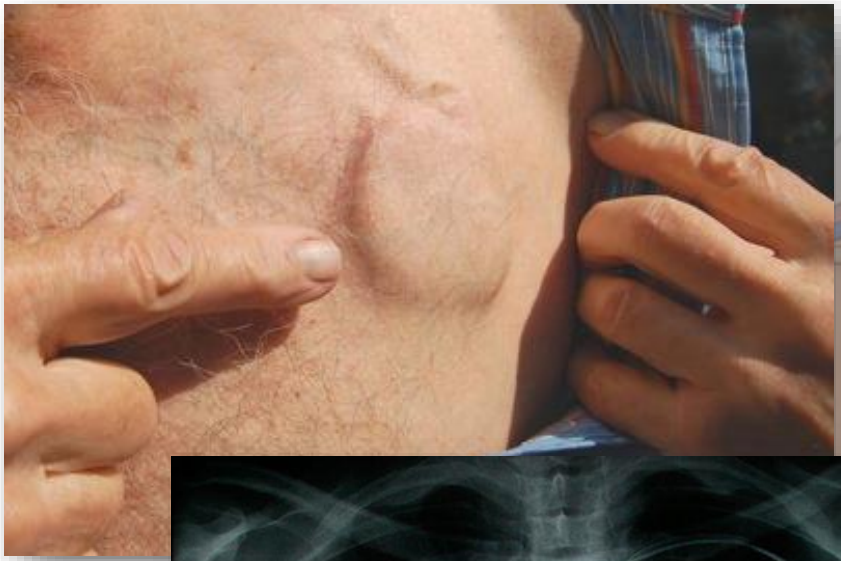
PEA/Asystole

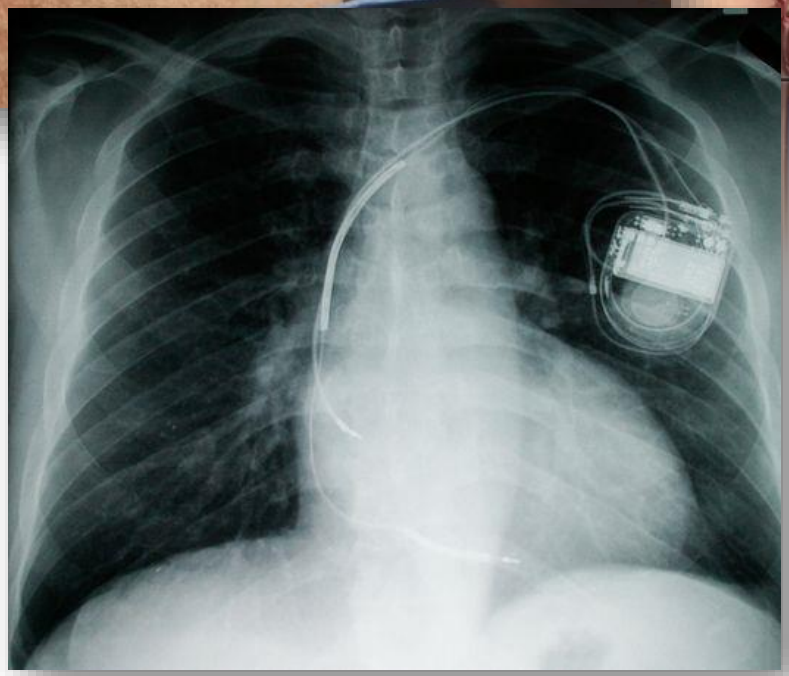
- When attempting to determine underling cause of cardiac arrest remember the H's and T's
  - Hypovolemia
  - Hypoxia
  - Hydrogen Ion (Acidosis)
  - Hypo/hyperkalemia
  - Hypoglycemia
  - Hypothermia
  - Toxins
  - Tamponade (cardiac)
  - Tension Pneumothorax
  - Thrombosis (coronary or pulmonary)
  - Trauma

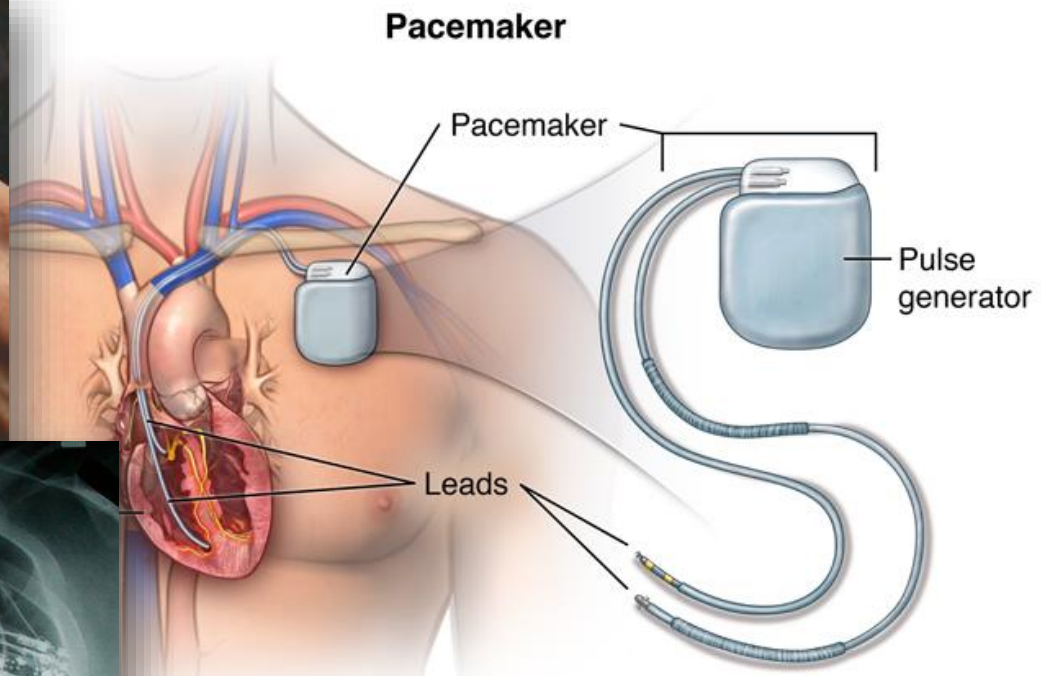
Pacemaker Rhythm

- As a result of underlying dysrhythmias, some patients have a surgically implanted cardiac pacemaker
- Two main types:
  - Single chamber: only one pacing lead is placed in the R atrium or R ventricle
  - Dual chamber: two pacing leads, one in the R atrium and R ventricle
- For both types, the device can either:
  - monitor the patient's underlying rhythm and take over pacing when needed = demand pacemaker
  - or automatically pace the patient at a set rate = fixed pacemaker

Pacemaker Rhythm

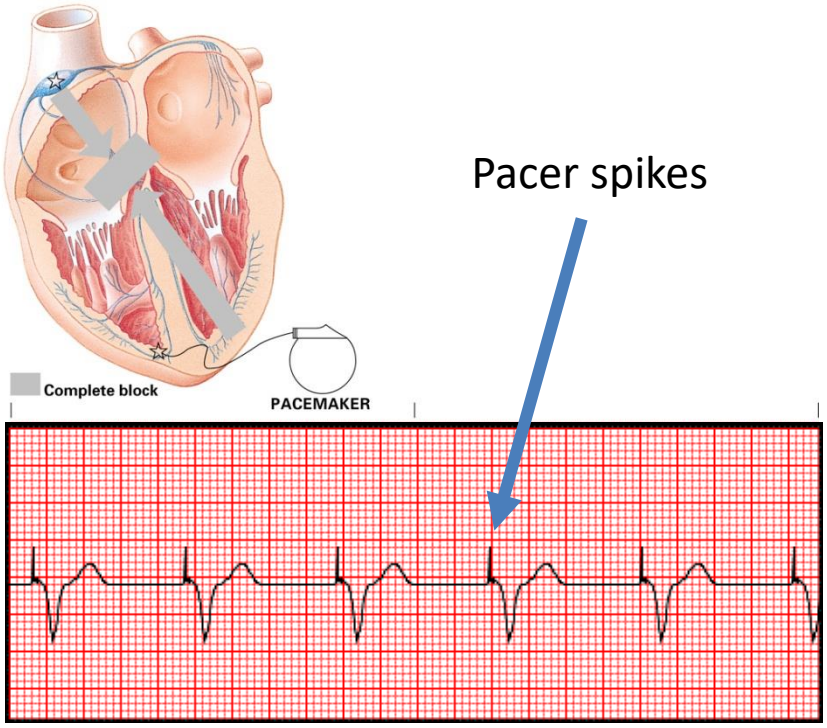






Pacemaker Rhythm

Rules of Interpretation **Artificial Pacemaker Rhythm**	
Rate	***Varies with pacemaker***
Rhythm	***May be regular or irregular***
Pacemaker Site	***Depends upon electrode placement***
P Waves	***None produced by ventricular pacemakers; pacemaker spike***
PRI	***If present, varies***
QRS	***>0.12 seconds, bizarre***



Pacemaker Rhythm

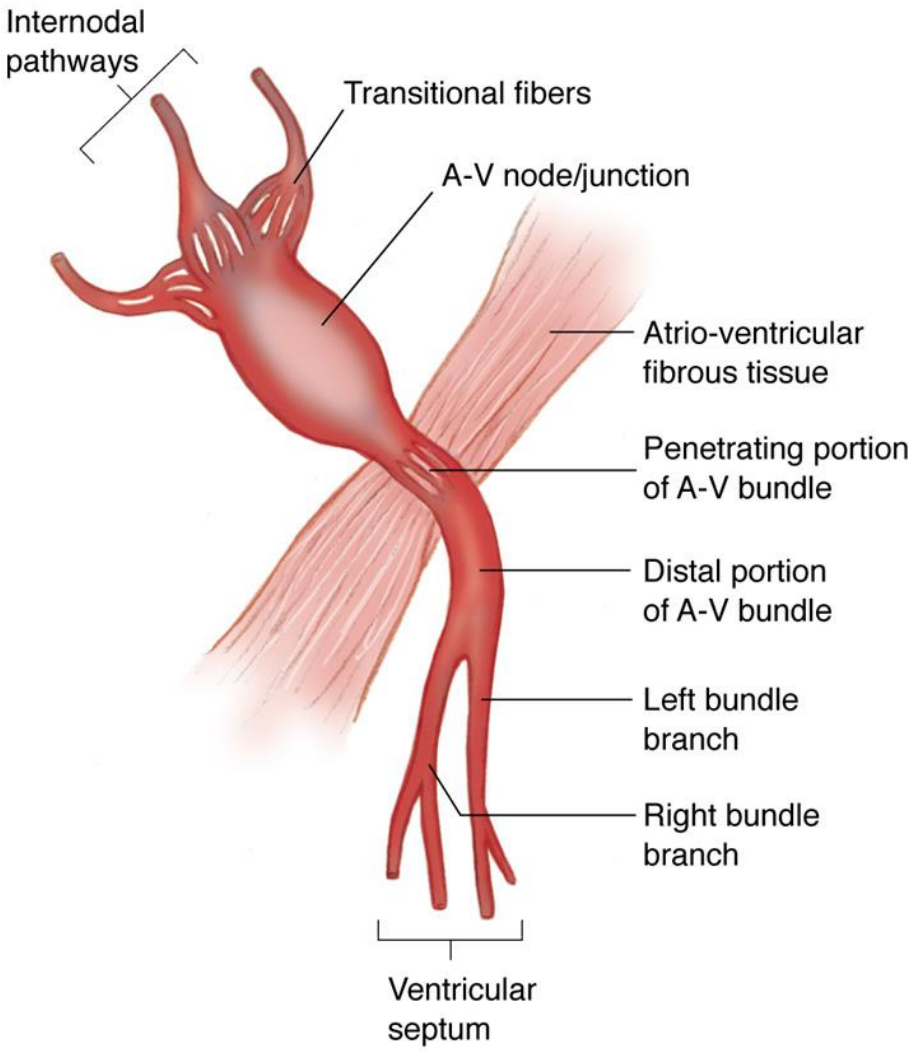
- Problems with Pacemakers
  - Battery failure
  - “Runaway” pacers
  - Displaced leads
- Management Considerations
  - Identify patients with pacemakers
  - Treat the patient
- In hospital, use of a Magnet
  - Inhibits all sensing and resets pacemaker to a predetermined rate (~70)

Other Conduction Disorders

- Atrioventricular Blocks
- Premature Beats
  - Premature Atrial Contractions
  - Premature Junctional Contractions
  - Premature Ventricular Contractions
- Pre-excitation Syndromes

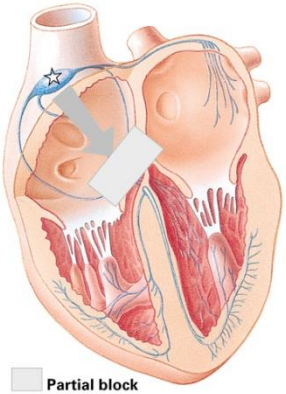
Atrioventricular Blocks

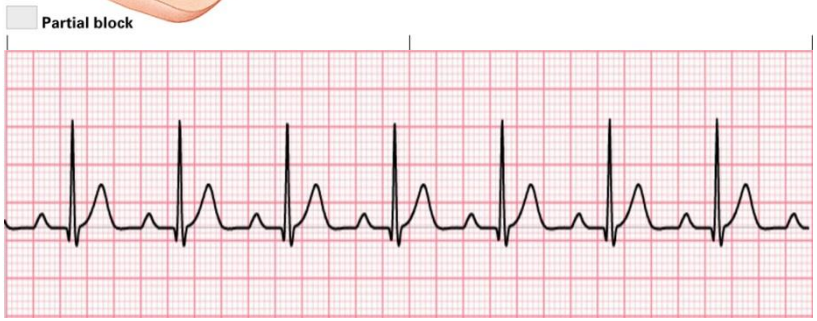
- First-Degree AV Block
- Second-Degree AV Block Type I
- Second-Degree Type II AV Block
- Third-Degree AV Block



First-Degree AV Block

Rules of Interpretation **First-Degree AV Block**	
Rate	***Depends on underlying rhythm***
Rhythm	***Usually regular***
Pacemaker Site	***SA node or atrial***
P Waves	***Normal***
PRI	***> 0.20 Seconds***
QRS	***Usually < 0.12 seconds***



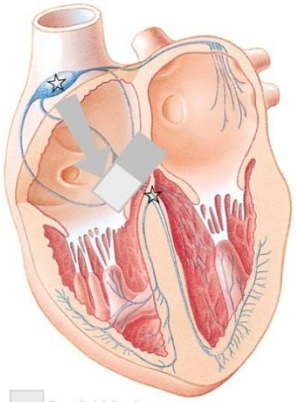


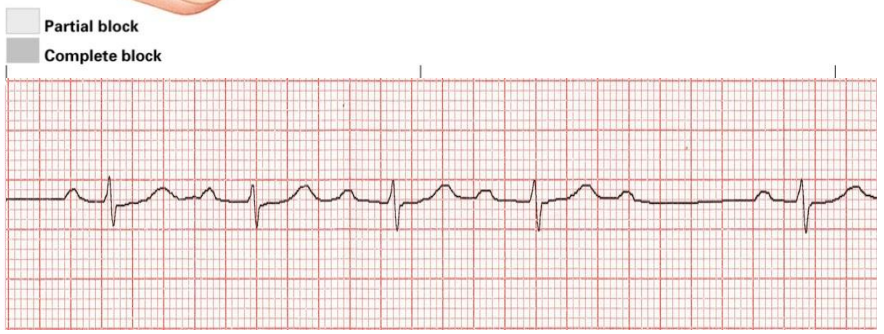
First-Degree AV Block

- Not an underlying rhythm
- Etiology
  - Delay in the conjunction of an impulse through the AV node
  - May occur in healthy hearts, but often indicative of ischemia at the AV junction
- Clinical Significance
  - Usually not significant, but new onset may precede a more advanced block
- Treatment
  - Generally, none required other than observation
  - Avoid drugs that may further slow AV conduction

Second-Degree Type I AV Block

Rules of Interpretation **Second-Degree Type I AV Block**	
Rate	***Depends on underlying rhythm***
Rhythm	***Regularly irregular***
Pacemaker Site	***SA node or atrial***
P Waves	***Normal, some P waves not followed by QRS***
PRI	***Increases until QRS is dropped, then repeats***
QRS	***Usually < 0.12 seconds***



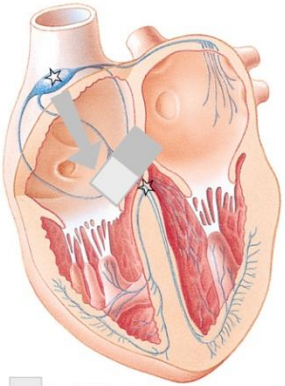


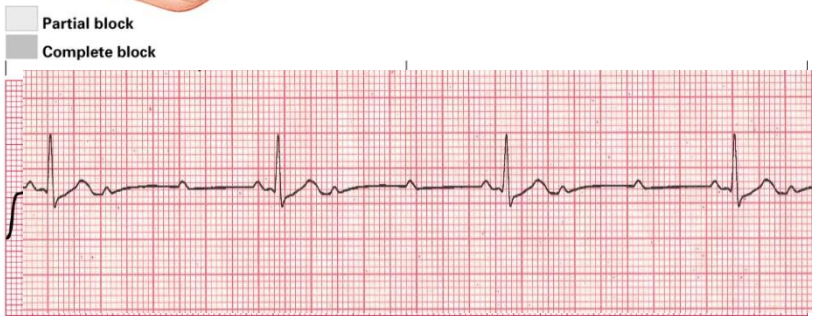
Second-Degree Type I AV Block

- Etiology
  - Also called Mobitz I, or Wenckebach
  - Delay increases until an impulse is blocked
  - Indicative of ischemia at the AV junction
- Clinical Significance
  - Frequently dropped beats can result in cardiac compromise
- Treatment
  - Generally, none required other than observation
  - Avoid drugs that may further slow AV conduction
  - Treat symptomatic bradycardia

Second-Degree Type II AV Block

Rules of Interpretation **Second-Degree Type II AV Block**	
Rate	***Depends on underlying rhythm***
Rhythm	***Regularly irregular***
Pacemaker Site	***SA node or atrial***
P Waves	***Normal, some P waves not followed by QRS***
PRI	***Constant for conducted beats, may be > 0.21 seconds***
QRS	***Normal or > 0.12 seconds***





Second-Degree Type II AV Block

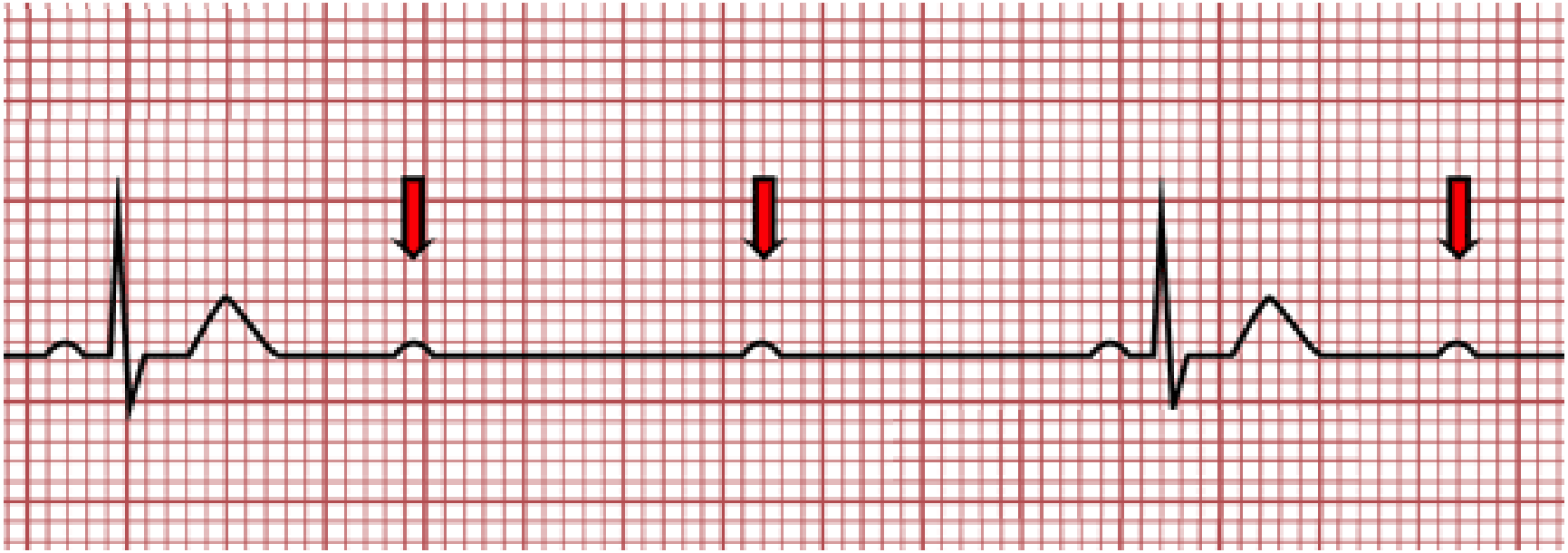
- Etiology
  - Also called Mobitz II or infranodal
  - Intermittent block of impulses
  - Usually associated with MI or septal necrosis
- Clinical Significance
  - May compromise cardiac output and is indicative of MI
  - Often develops into 3rd degree AV block
- Treatment
  - Avoid drugs that may further slow AV conduction
  - Treat symptomatic bradycardia
  - ACP may consider transcutaneous pacing

Second-Degree AV Blocks

- Both Type I and Type II typically occur in fixed ratio
  - Reported as ratio of # of P waves: # of QRS complexes
    - 4:3 Second-degree represents a dropped QRS complex every fourth atrial beat
  - It not possible to determine if a 2:1 second-degree is Type I or Type II since you cannot determine if PRI is lengthening

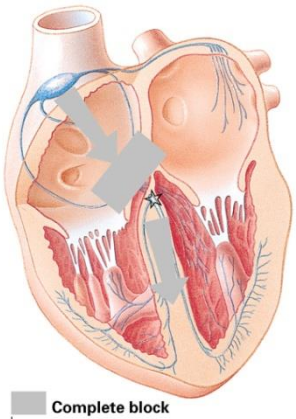
Second-Degree AV Blocks

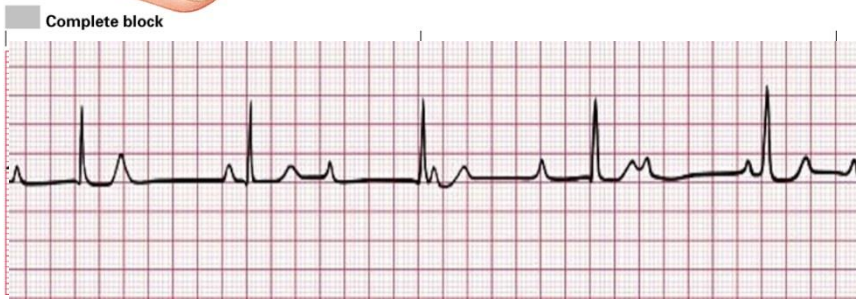
- For advanced (“high-grade”)second-degree AV blocks, there may be multiple dropped QRS complexes in a row



Third-Degree AV Block

Rules of Interpretation **Third-Degree AV Block**	
Rate	***Atrial rate is normal; ventricular, 40–60***
Rhythm	***Both atrial and ventricular are regular***
Pacemaker Site	***SA node and AV junction or ventricle***
P Waves	***Normal in appearance with no correlation to QRS***
PRI	***No relationship to QRS***
QRS	***0.12 seconds or greater***





Third-Degree AV Block

- Etiology
  - Absence of conduction between the atria and the ventricles
    - Also known as complete AV dissociation
  - Results from AMI, digitalis toxicity, or degeneration of the conductive system
- Clinical Significance
  - Severely compromised cardiac output
- Treatment
  - Transcutaneous pacing for acutely symptomatic patients
  - Treat symptomatic bradycardia
  - Avoid drugs that may further slow AV conduction

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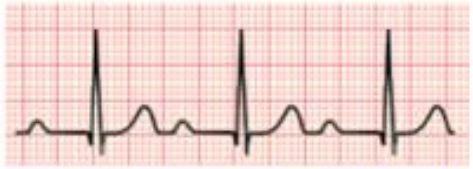
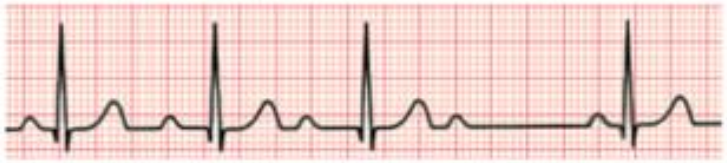
Dysrhythmias Originating Within the AV Junction

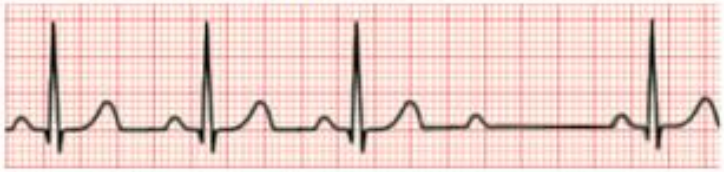
First degree AV block



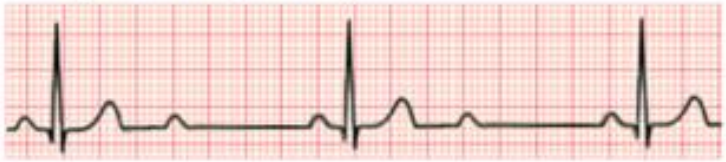
Second degree AV block (Mobitz I or Wenckebach)



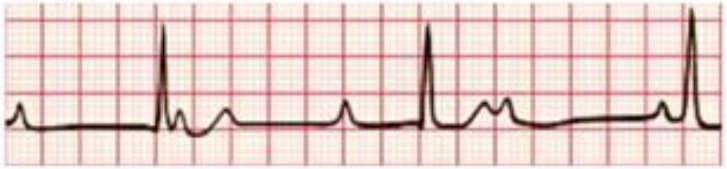
Second degree AV block (Mobitz II)



Second degree AV block (2:1 block)



Third degree AV block with junctional escape

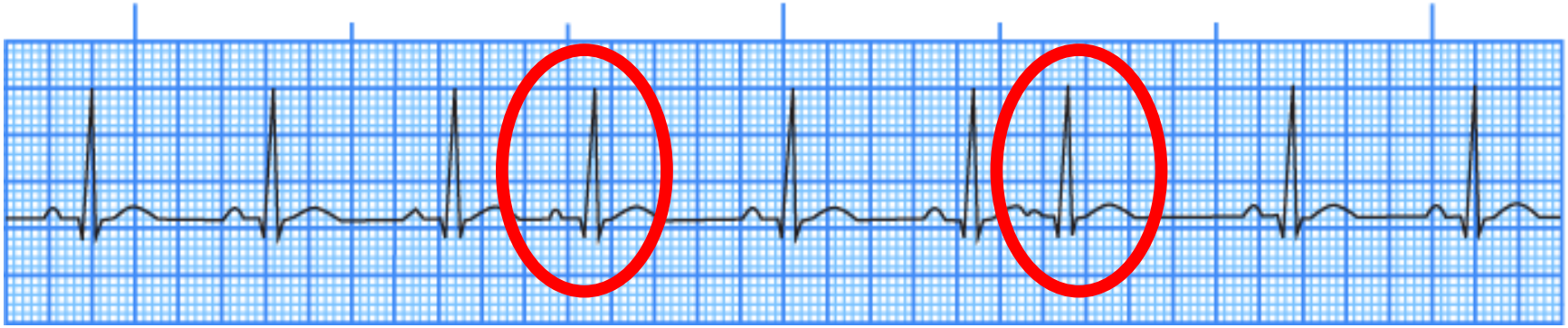


Premature Beats

- At times, non-pacemaking regions of the heart can fire, causing depolarization of the heart before the next regular beat was due
- These are referred to as premature contractions and are named based on their origin
  - Premature Atrial Contractions
  - Premature Junctional Contractions
  - Premature Ventricular Contractions
- Term is a misnomer as not all premature depolarizations produce a mechanical contraction
  - Acceptable, and more accurate to use “Complex” in place of “Contraction”

Premature Atrial Complex

- Also known as PACs, these early depolarizations originate in the atria but outside of the SA node
  - Since origin is in the atria:
    - A P wave is present
    - QRS is normal in width
  - Since impulse originates outside the SA node:
    - The P wave morphology is different than the others

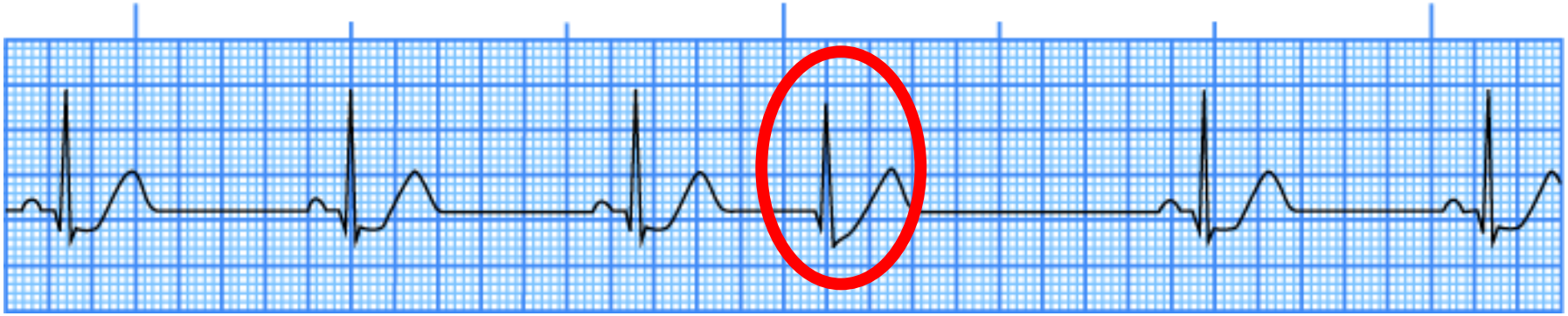


Premature Atrial Complex

- Etiology
  - Single electrical impulse originating outside the SA node
  - May result from use of caffeine, tobacco, or alcohol, sympathomimetic drugs, ischemic heart disease, hypoxia, or digitalis toxicity, or may be idiopathic
- Clinical Significance
  - Presence of PACs may be a precursor to other atrial dysrhythmias
- Treatment
  - None if asymptomatic
  - Treat symptomatic patients by administering high-flow oxygen and establishing IV access

Premature Junctional Complex

- Also known as PJCs, these early depolarizations originate in the Junction but outside of the AV node/Bundle of His
  - Since origin is in the Junction:
    - A P wave may or may not be present
    - If a P wave is present it will be inverted (due to retrograde transmission)
    - QRS is normal in width but different in appearance

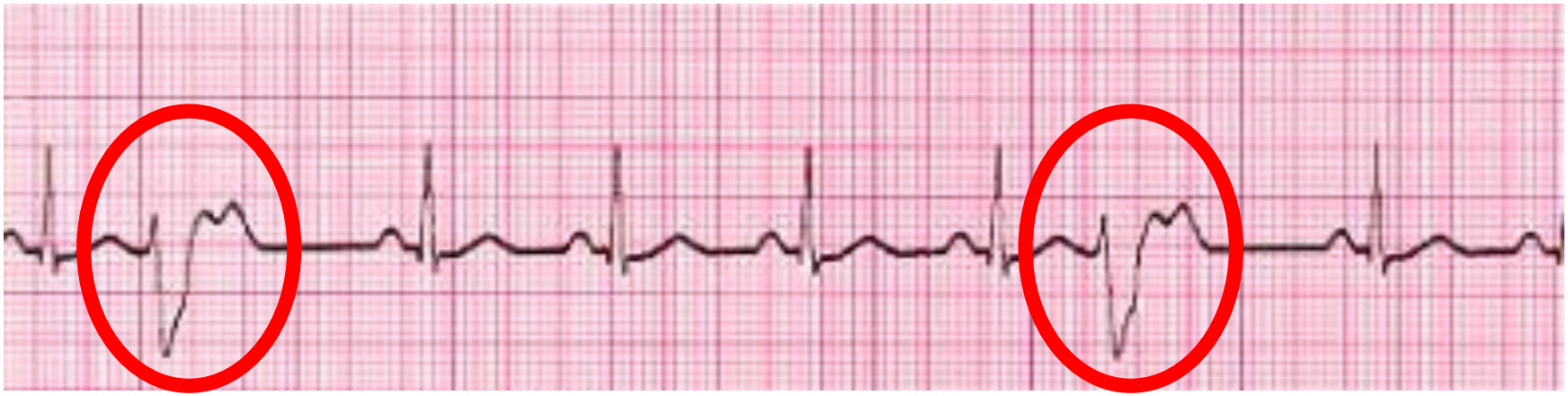


Premature Junctional Complex

- Etiology
  - Single electrical impulse originating in the AV Junction
  - May occur with use of caffeine, tobacco, alcohol, sympathomimetic drugs, ischemic heart disease, hypoxia, or digitalis toxicity, or may be idiopathic
- Clinical Significance
  - Limited, frequent PJCs may precursor other Junctional dysrhythmias
- Treatment
  - None usually required

Premature Ventricular Complex

- Also known as PVCs, these early depolarizations originate in the Ventricles but outside of the Bundle branches/Purkinje fibers
  - Since origin is in the Ventricles:
    - A P wave will not be present
    - QRS width is > 0.12s
      - At times T wave is merged with QRS due to QRS width

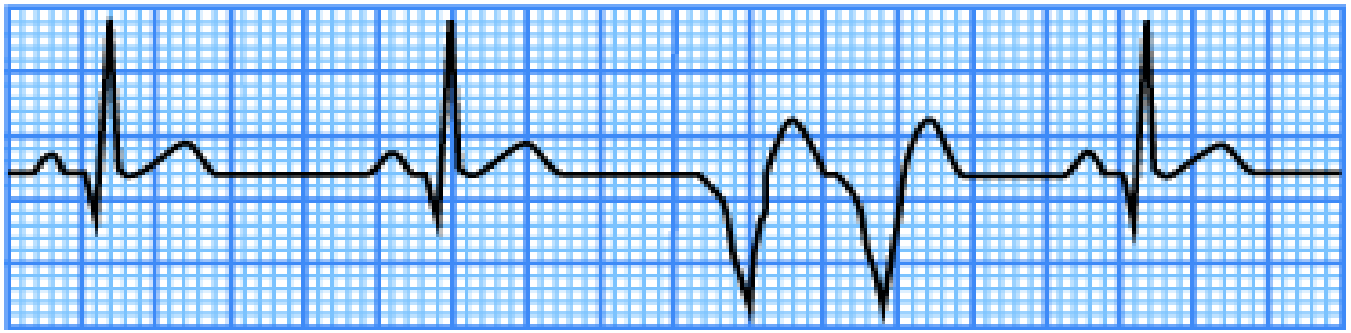


Premature Ventricular Complex

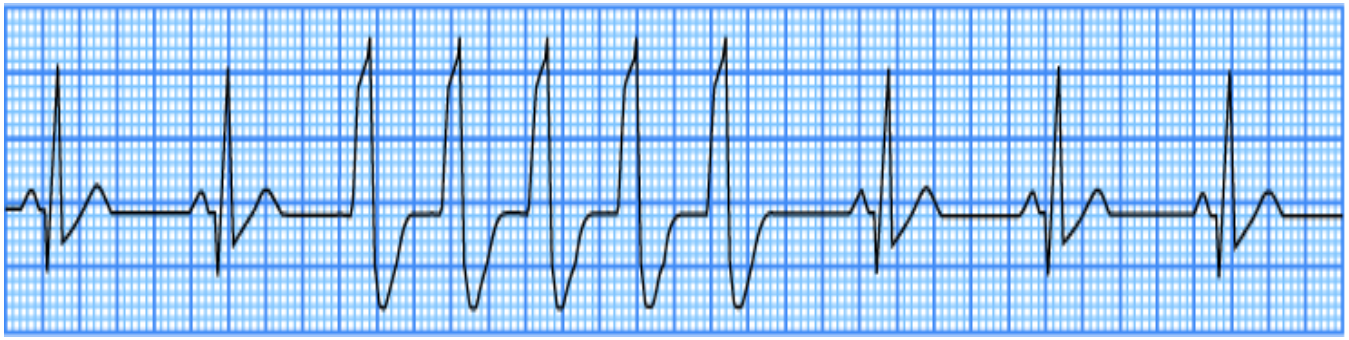
- Etiology
  - Single ectopic impulse resulting from an irritable focus in either ventricle
  - Causes may include myocardial ischemia, increased sympathetic tone, hypoxia, idiopathic causes, acid–base disturbances, electrolyte imbalances, or as a normal variation of the ECG
  - May occur in patterns
    - Bigeminy, trigeminy, or quadrigeminy
    - Couplets and triplets
    - Uni vs multifocal

Premature Ventricular Complex

- When 2 PVCs occur back to back = **couplet**

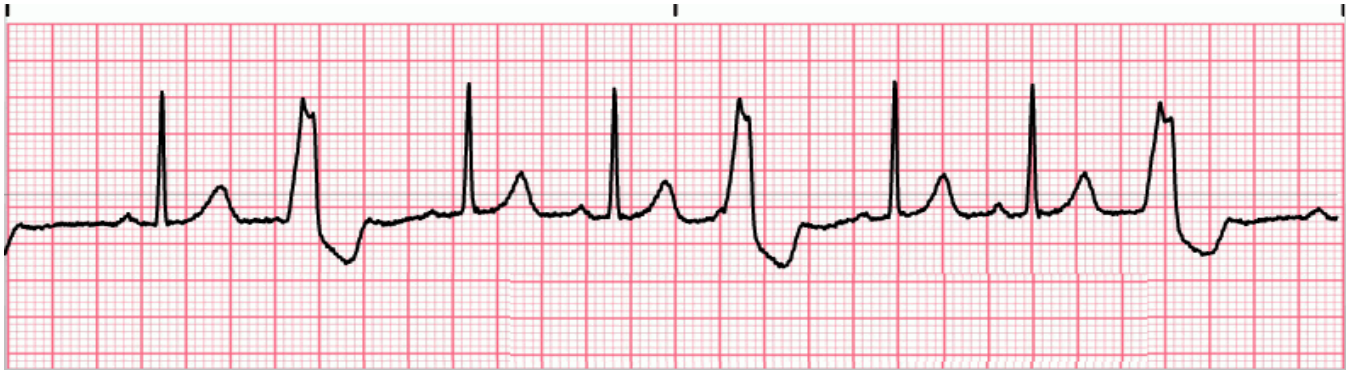


- When 3 or move PVCs occur back to back to back = **run of VTach**

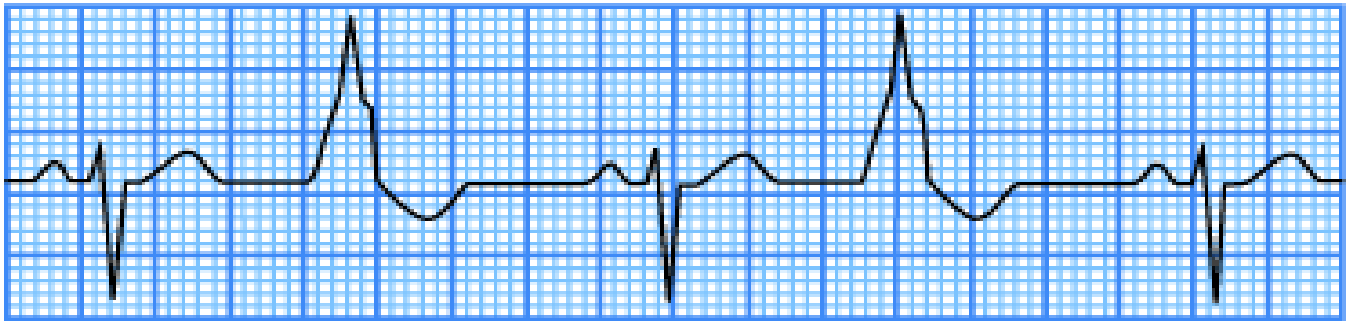


Premature Ventricular Complex

- When a PVC occurs every third complex = **trigeminy**

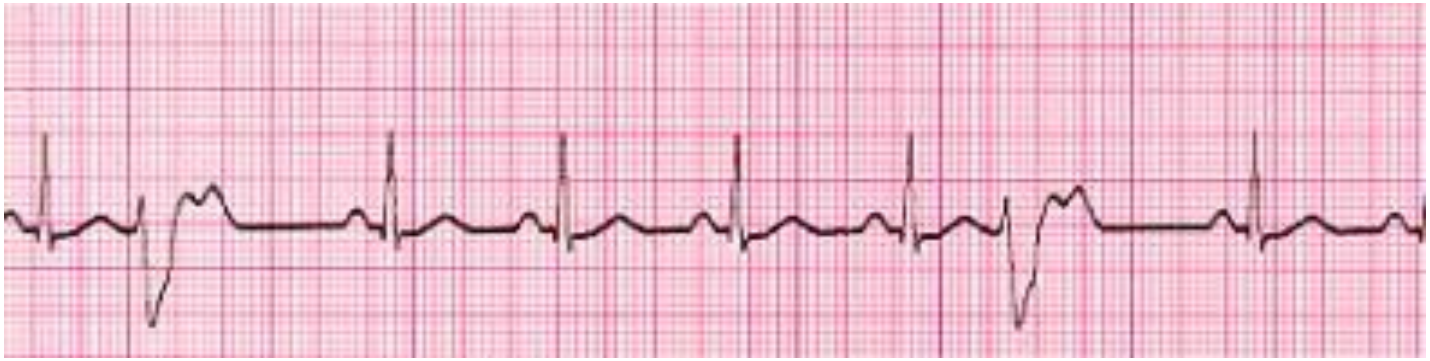


- When a PVC occurs every second complex = **bigeminy**

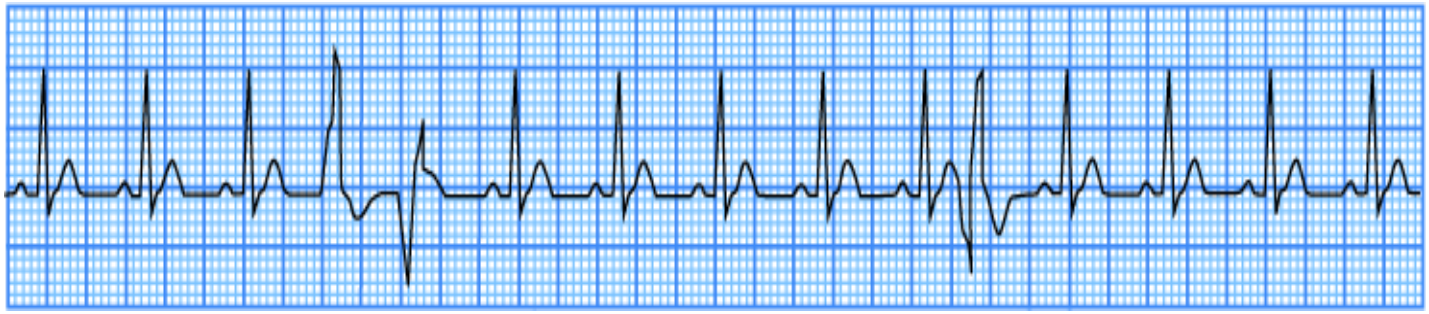


Premature Ventricular Complex

- When PVCs originate from the same irritable focus, they have the same in morphology = **unifocal PVCs**



- When PVCs originate from different irritable foci, they have the different morphologies = **multifocal PVCs**



Premature Ventricular Contractions

- Clinical Significance
  - PVCs can be **perfusing** (cause a mechanical contraction) or **non-perfusing** (electrical activity only)
  - Although PVCs can be benign, increased clinical significance with:
    - More than 6/minute
    - Couplets or runs of ventricular tachycardia
    - Bigeminy
    - Multifocal PVCs
    - PVCs associated with chest pain

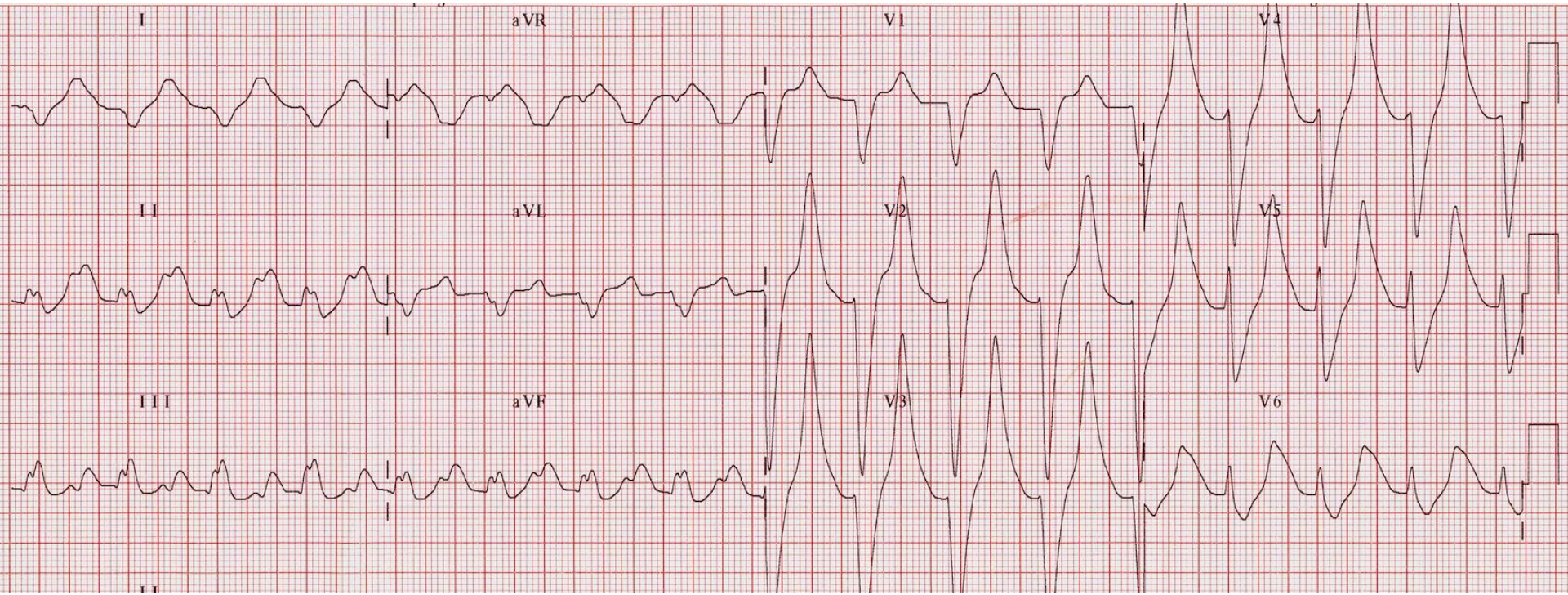
Premature Ventricular Contractions

- Treatment
  - Non-malignant PVCs do not usually require treatment in patients without a cardiac history
    - Administer oxygen and establish IV access
  - ACP may treat malignant PVCs:
    - Lidocaine 1.0 –1.5 mg/kg IV bolus.
    - If PVCs are not suppressed, repeat doses of 0.5-0.75 mg/kg to max dose of 3.0 mg/kg.
    - If PVCs are suppressed, lidocaine drip 2–4 mg/min.
    - Reduce the dose in patients with decreased output or decreased hepatic function and patients > 70 years old.

Other ECG Findings

- Hyperkalemia
  - Can produce tall ("peaked") T waves
    - Suspect in patients with a history of renal failure
  - Differentiate from Hyperacute T waves
    - Hyperacute should only be in leads affected by hypoxia whereas Hyperkalemic, peaked T waves would be global
  - Best seen in precordial leads
  - Typically not peaked until serum $K^+ > 6 - 6.5mEq/L$

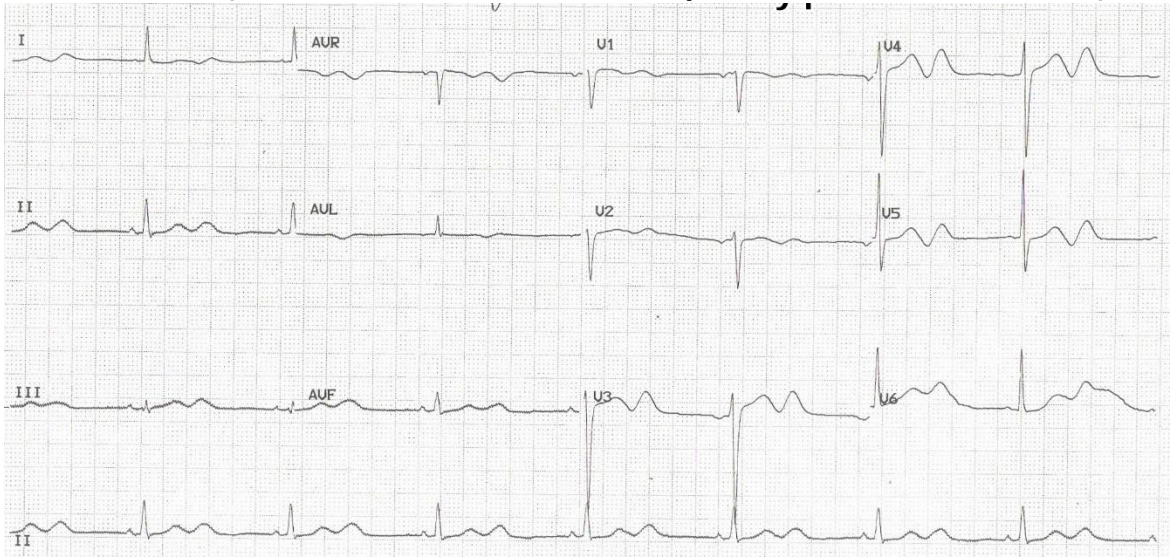
Other ECG Findings



Serum K^+ = 9.2 mEq/L

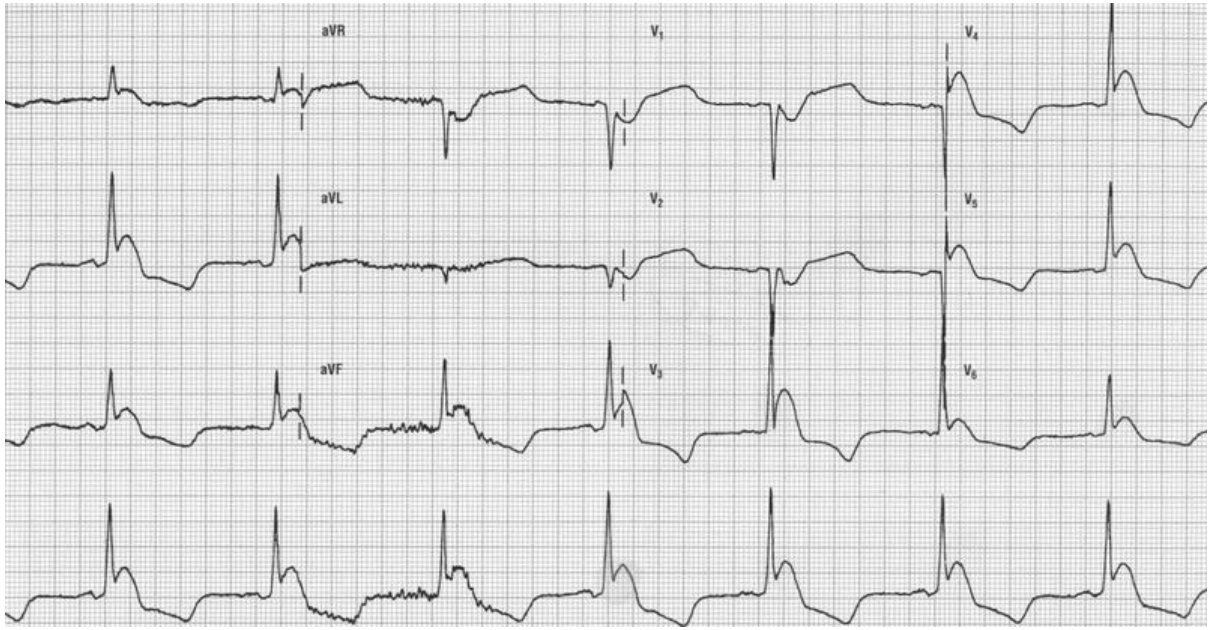
Other ECG Findings

- Hypokalemia
  - Can produce ECG changes that include:
    - P waves with decreased amplitude
    - Increase PRI
    - Flattened or inverted T waves
    - U waves (best observed in precordial leads)



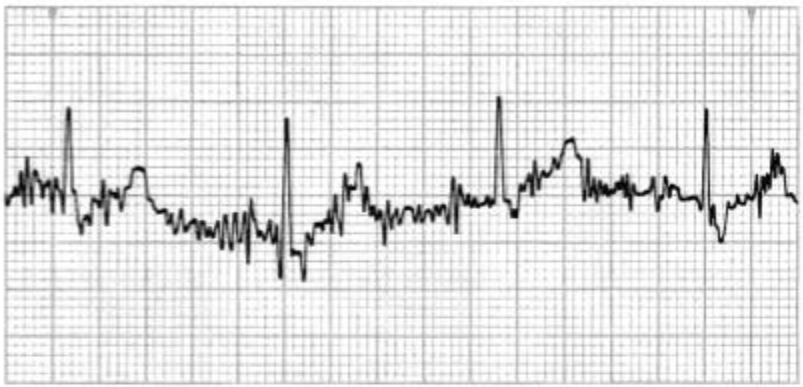
Other ECG Findings

- Hypothermia
  - Can produce ECG changes that include:
    - Osborn wave (“J” wave)
    - T wave inversion
    - Prolonged PRI and QRS

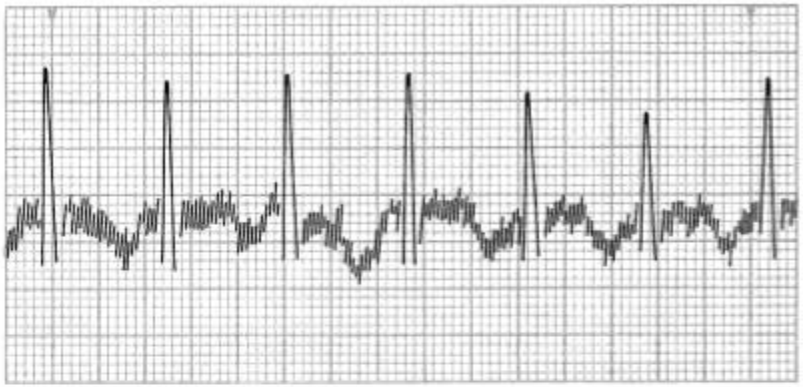


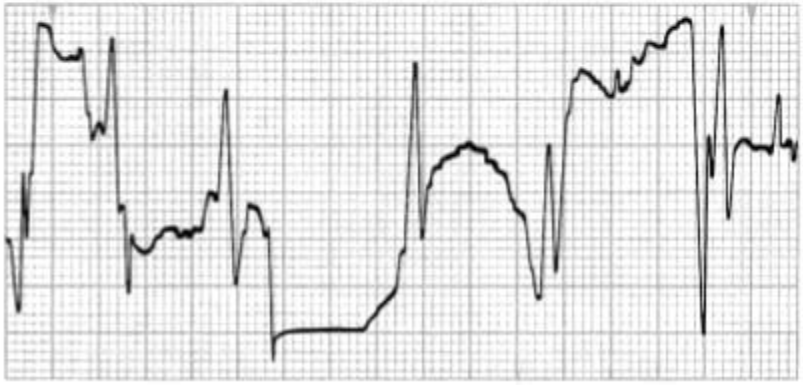
Artifact

Muscle Tremors

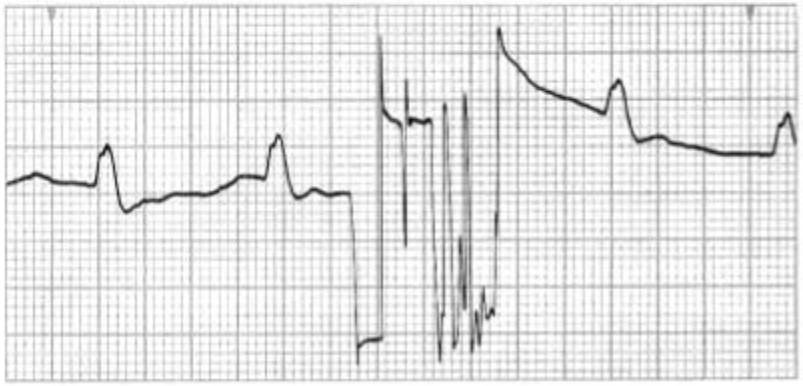


AC (60-cycle) interference





Lose electrodes



Biotelemetry

(Poor reception of signal)

Artifact

- Minimize possible artifact by:
  - Stop patient movement
  - Have patient stop talking
  - Stop ambulance is necessary to capture accurate tracing
  - Support limbs
  - Cover with blankets to reduce shivering
  - Move electrode to another location on limb to avoid interference by muscle tissue
  - Replace electrodes if not adhering to patient
  - Shave patient's hair if not allowing skin contact
  - Troubleshoot worn out/damage cables