Rhythm and EKG Interpretation

Presented for Nursing

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Avera eCARE Clinical Nurse Educator

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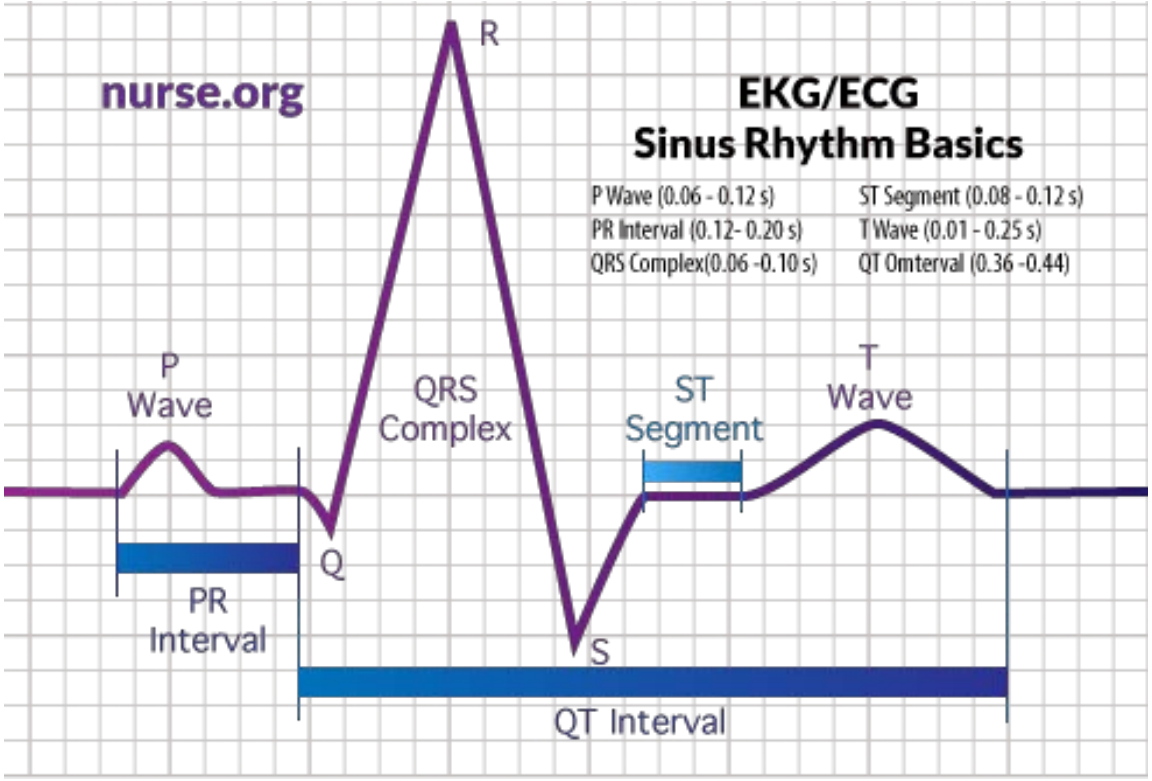
EKG Basics



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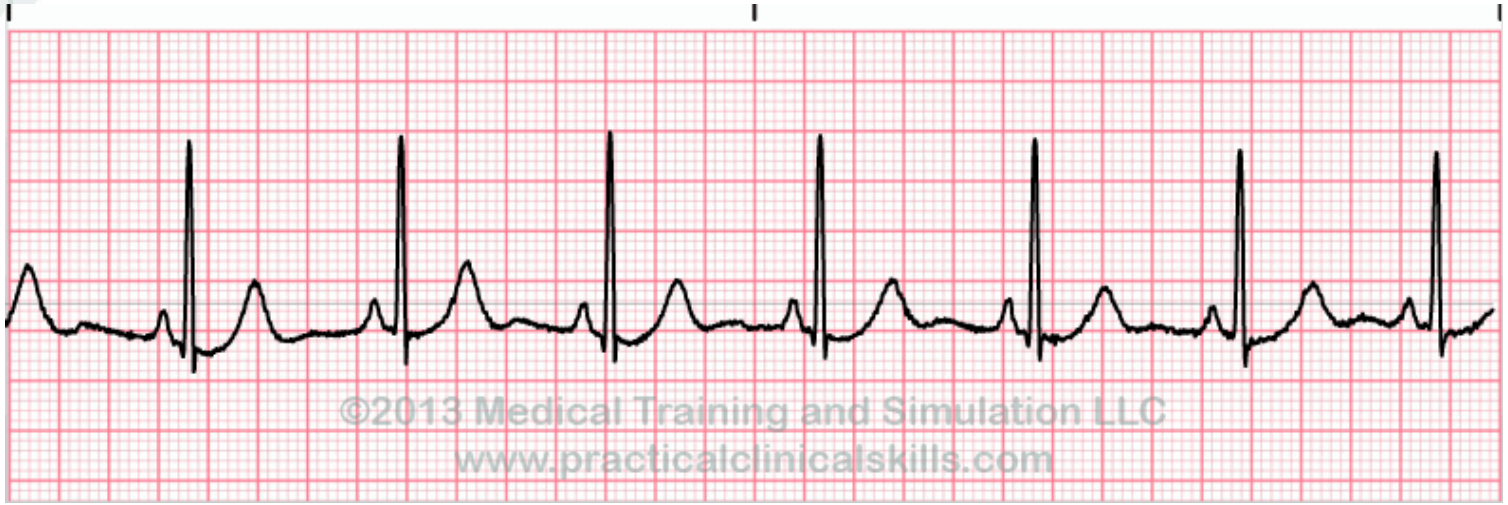
Assess Your Patient

- Are they having chest pain?
- Are they short of breath?
- How does their skin look? (Pale, normal for race, dry, diaphoretic, etc.)
- Can you palpate peripheral pulses?
  - Nurse.org

Sinus Rhythms – Normal Sinus Rhythm



60-100bpm

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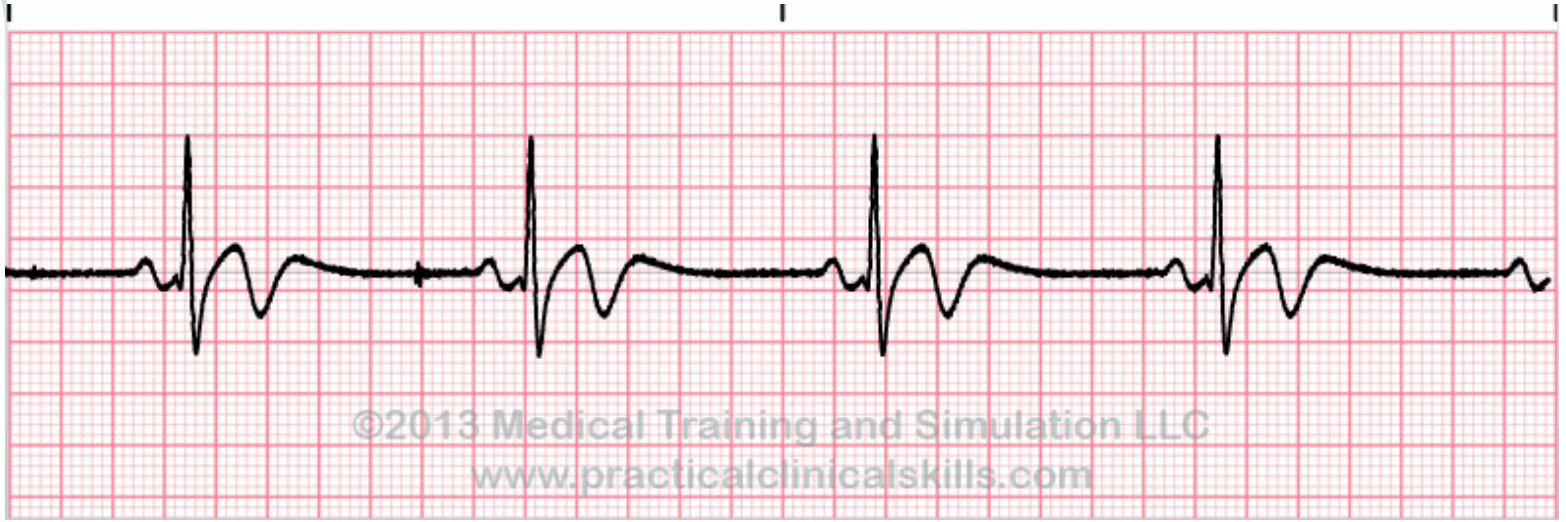
Sinus Bradycardia



<60 bpm and regular

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Sinus Bradycardia

Causes

- Normal during sleep
- Increased vagal tone (athletes)
- Vagal stimulation
- Inferior MI
- Hypothyroidism
- Hypothermia
- Anorexia nervosa
- Myocarditis
- Medications
  - Opiates, beta-blockers, etc
- Electrolyte abnormalities
  - Hyperkalemia, hypermagnesemia

Treatments

- May not require treatment if “normal” for patient
- Depends on cause
- Atropine
- Pacing

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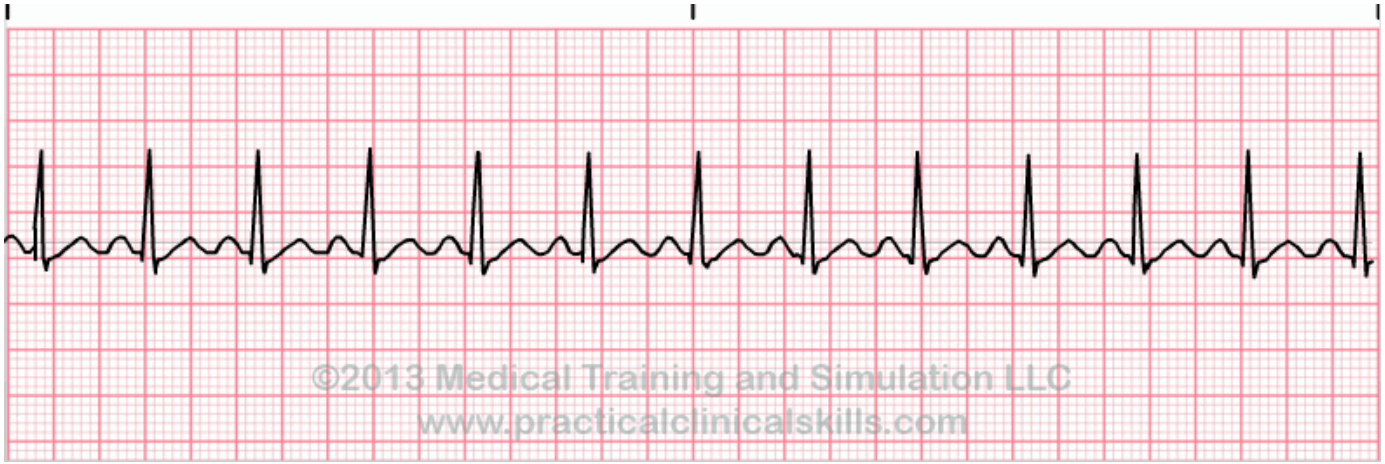
Sinus Tachycardia



>100 bpm and regular

Sinus Tachycardia

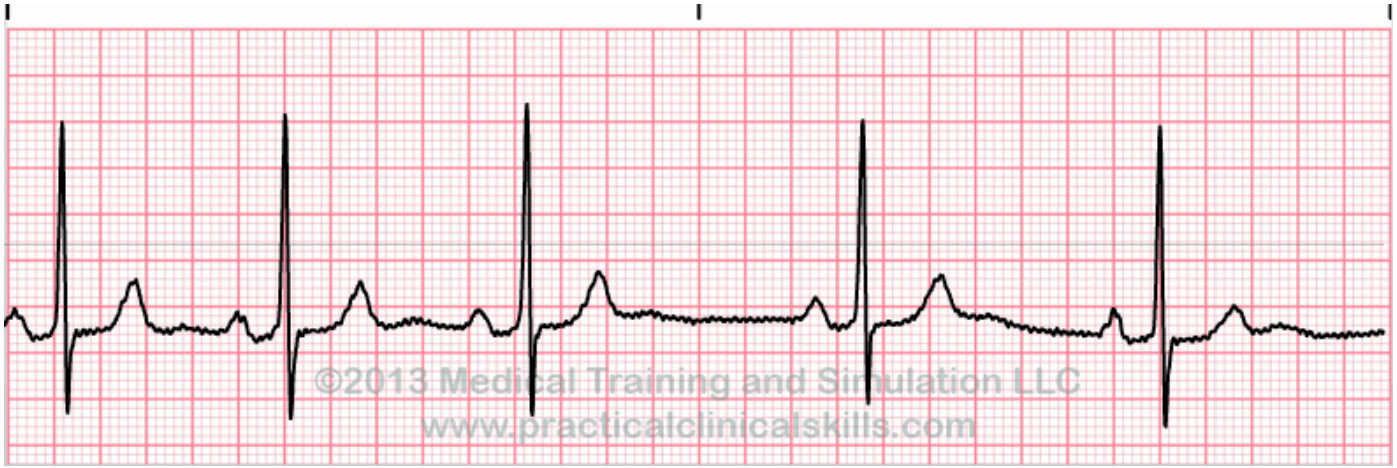
Causes

- Exercise
- Pain, Anxiety
- Hypoxia
- Sepsis
- Pulmonary Embolism (PE)
- Hyperthyroidism
- Medications
- Beta-agonists, antihistamines, etc
- Caffeine
- Marijuana

Treatments

- Medications to slow heart rate
- Depends on cause

Sinus Arrhythmia

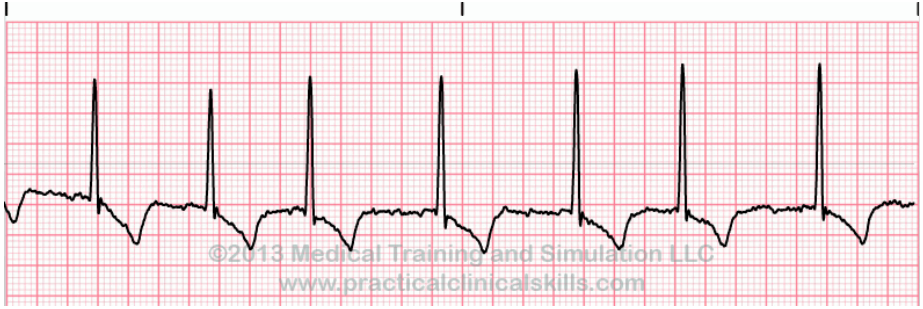


60-100 bpm and irregular

Sinus Arrhythmia

- Normal physiological phenomenon
- Commonly seen in young, healthy persons
- Incidence decreases with age
- Inspiration increases the heart rate by decreasing vagal tone
- With expiration, vagal tone is restored which decreases heart rate

Atrial Rhythms – Atrial Fibrillation



Irregular, rate may be slow, normal or fast

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Atrial Fibrillation

Potential Causes

- Hypertension
- Valvular heart disease
- Acute infections
- Electrolyte disturbances
- Hypokalemia and hypomagnesia
- Pulmonary Embolus
- Cardiomyopathies
- Ischemia heart disease

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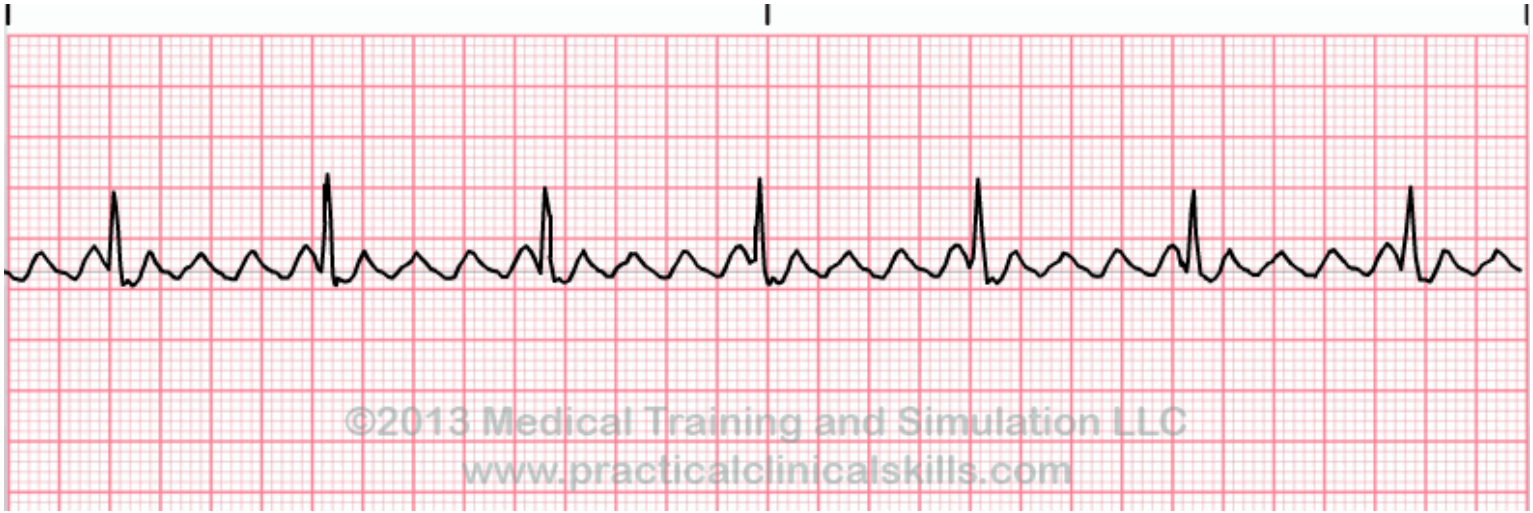
Atrial Flutter



Regular or Irregular

P Wave and PR Interval not measurable/observable

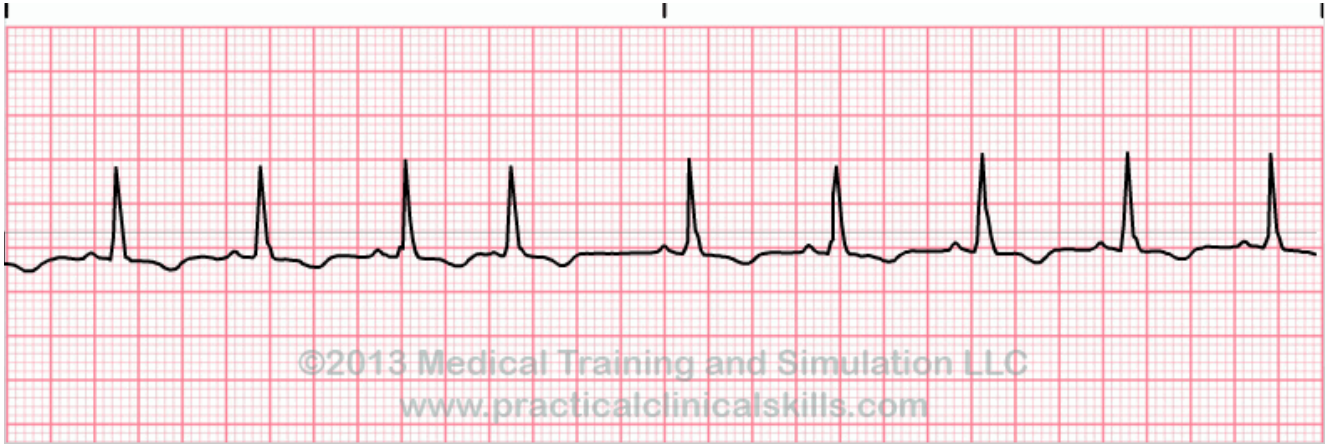
Atrial Flutter

A type of supraventricular tachycardia

Treatment:

- May try vagal maneuvers
- May try Adenosine
  - Usually will not convert with these treatments

Premature Atrial Complex (PAC)



Irregular
Premature P Wave

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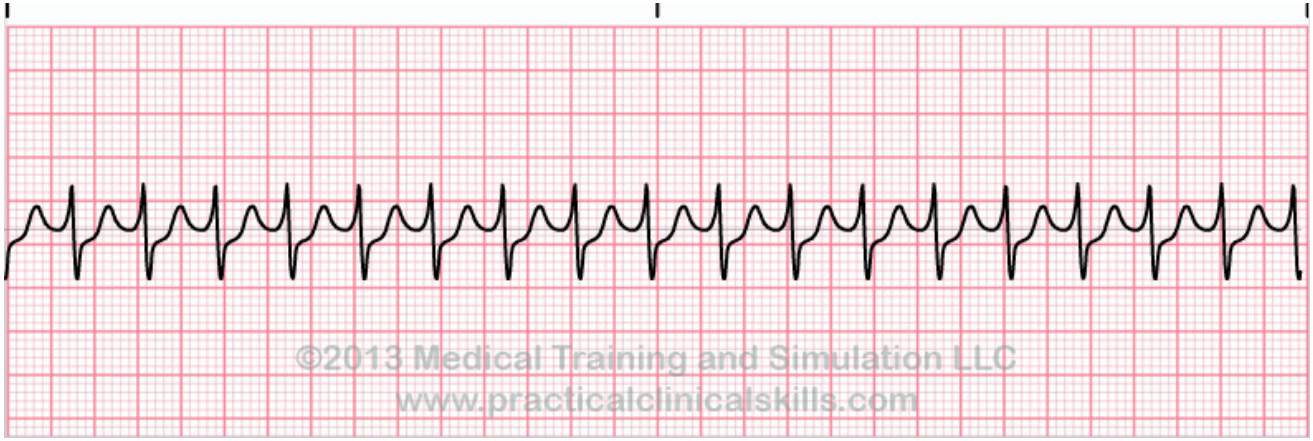
Premature Atrial Complex (PAC)

- Normal electrophysiological phenomenon not usually requiring investigation or treatment
- Frequent PACs may cause palpitations and a sense of the heart "skipping a beat"
- Potential causes:
  - Anxiety
  - Digoxin toxicity
  - Excess caffeine
  - Medications
  - Low potassium/magnesium
  - Myocardial ischemia

Supraventricular Tachycardia



Regular
Fast Rate (150-250 bpm

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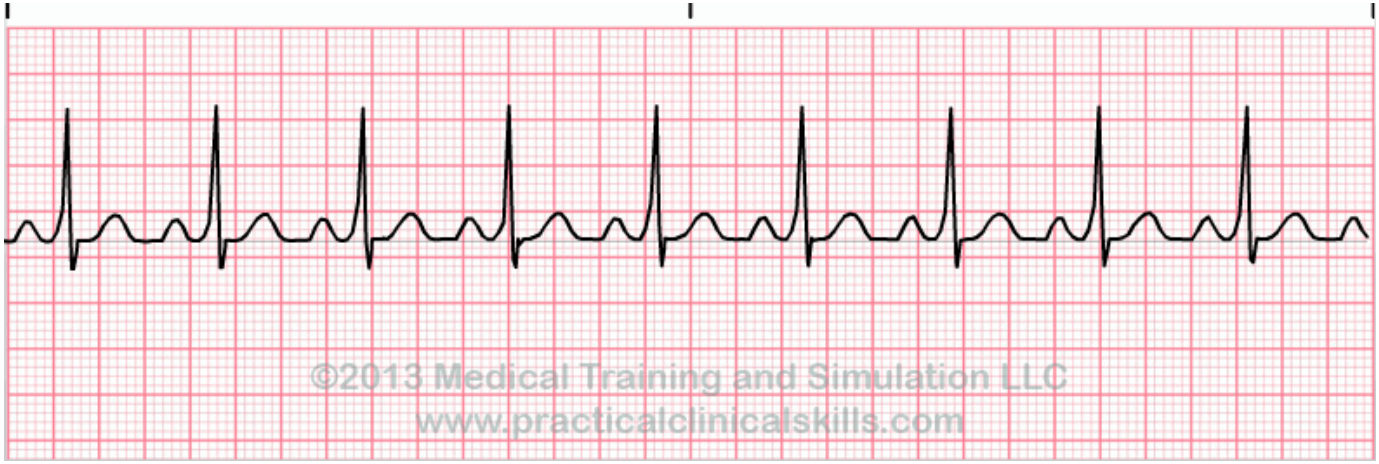
Supraventricular Tachycardia

- May respond to vagal maneuvers
- If no response, may try Adenosine
  - Large bore IV in antecubital
  - Elevate patient's arm
  - Three-way stopcock
  - Administer fast and flush quickly as Adenosine has quick half-life
  - First dose: 6mg then may try 12mg if no conversion
- Cardioversion is rarely required

Wolff-Parkinson-White Syndrome



Regular
Short PR Interval, Wide QRS

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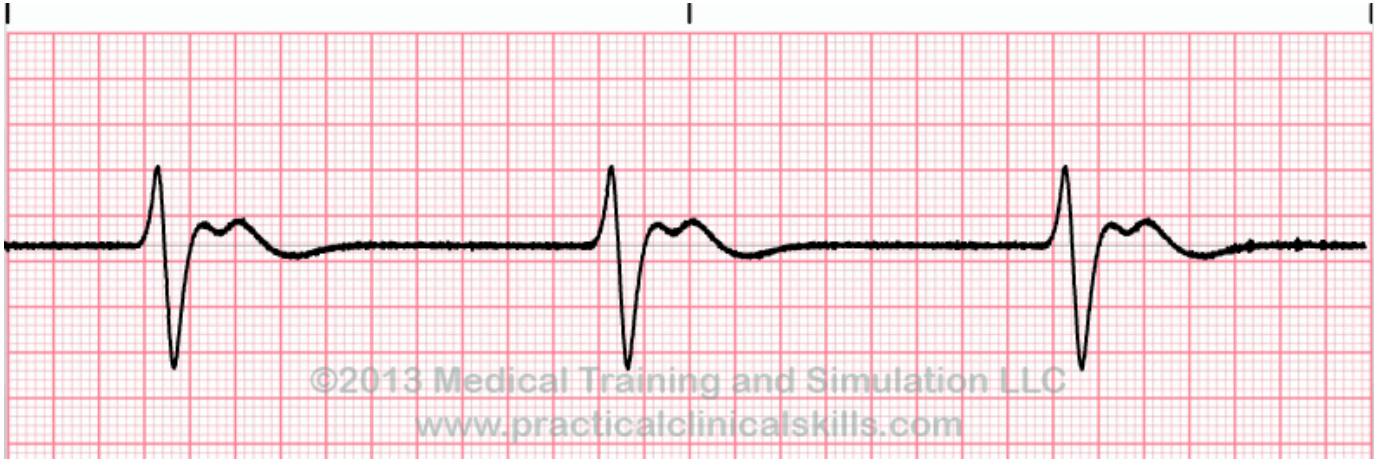
Ventricular Rhythms – Idioventricular Rhythm



Regular rhythm with slow rate (20-40 bpm)
Absent P Wave and unmeasurable PR interval

•Premature complexes frequently occur in
• rhythms, but may occur almost any time.
•PVC's occur when an early electrical impulse occurs from a location in either ventricle.

Premature Ventricular Complex (PVC)

- Normal electrophysiological phenomenon not usually requiring investigating or treatment

- Frequent PACS may cause palpitations and a sense of heart "skipping a beat"

Causes may include:

- Anxiety
- Excess Caffeine
- Low Potassium/Magnesium
- Dig Toxicity
- Beta-Antagonists
- Myocardial Ischemia

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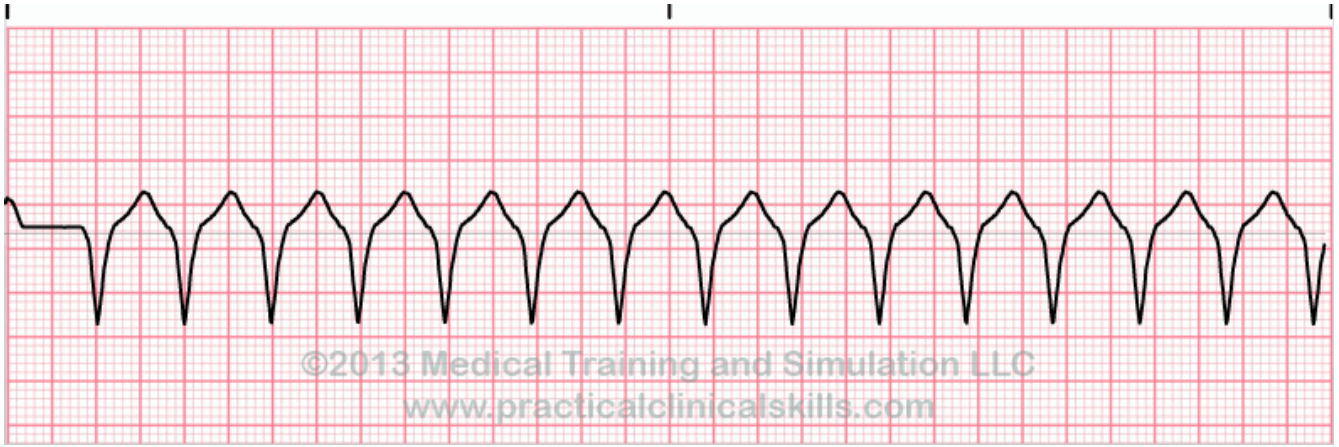
Ventricular Tachycardia



Regular rhythm and fast rate

Absent P Wave and unmeasurable PR interval

Ventricular Tachycardia

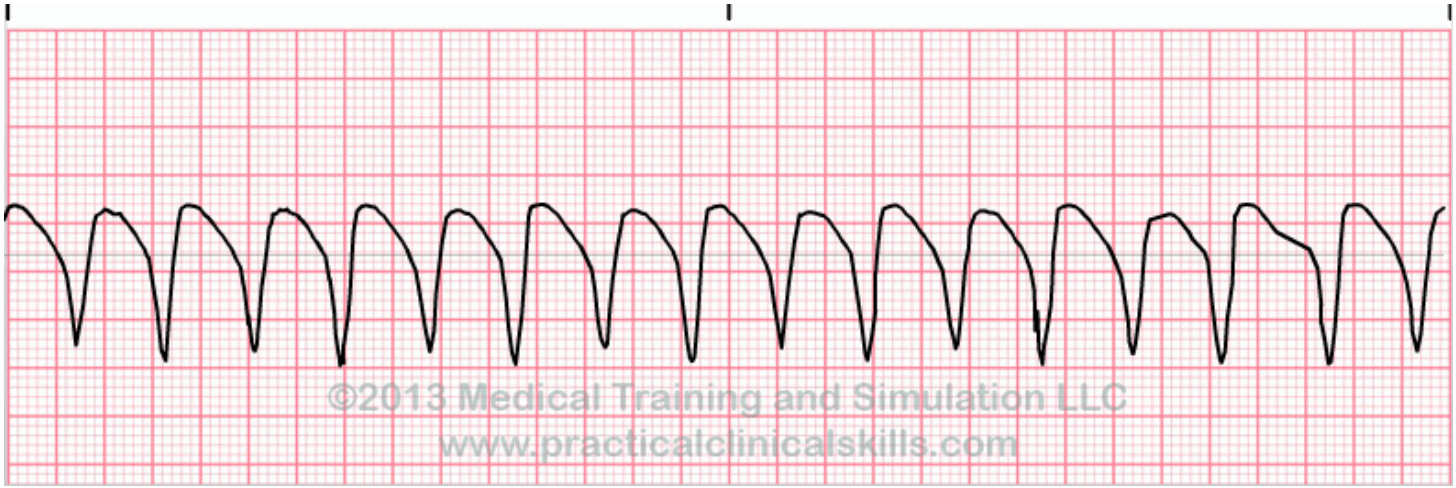
May be hemodynamically stable

Hemodynamically Unstable:

- Hypotension
- Chest pain
- Heart failure
- Decrease LOC
  - May impair cardiac output due to hypotension and acute cardiac failure through loss of atrial kick
  - Decreased cardiac output may decrease myocardial perfusion leading to vfib

Prompt recognition and initiation of treatment is required in all cases of vtach!

Ventricular Tachycardia: Monomorphic



Regular rhythm with fast rate

Absent P Wave and unmeasurable PR interval

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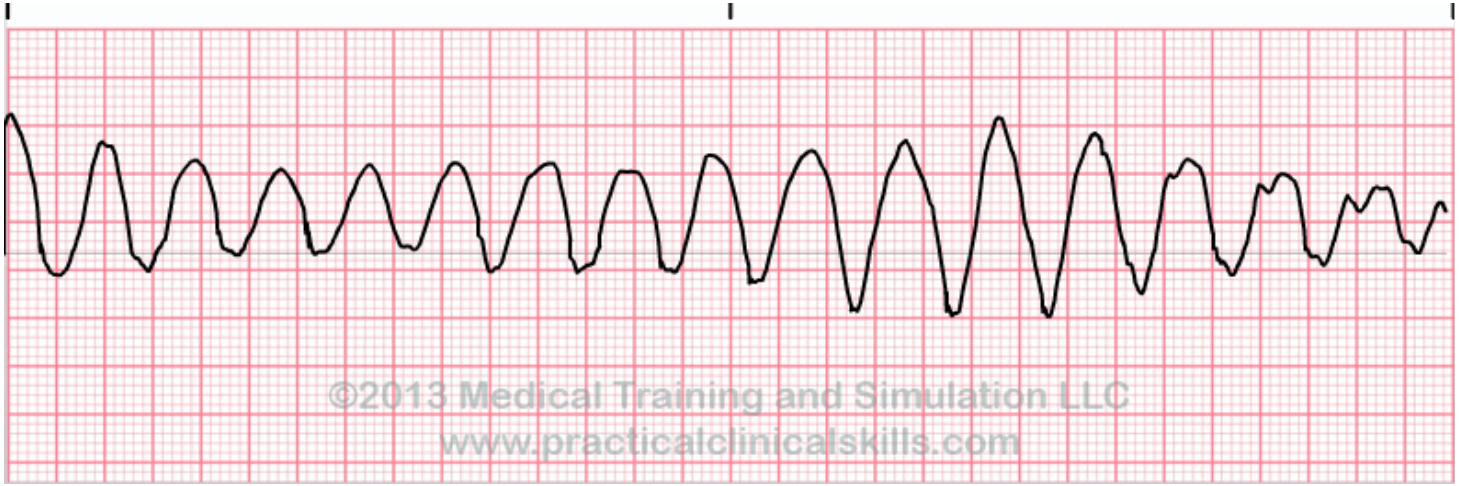
Ventricular Tachycardia: Polymorphic



Regular or irregular rhythm with fast rate
Absent P Wave and unmeasurable PR interval

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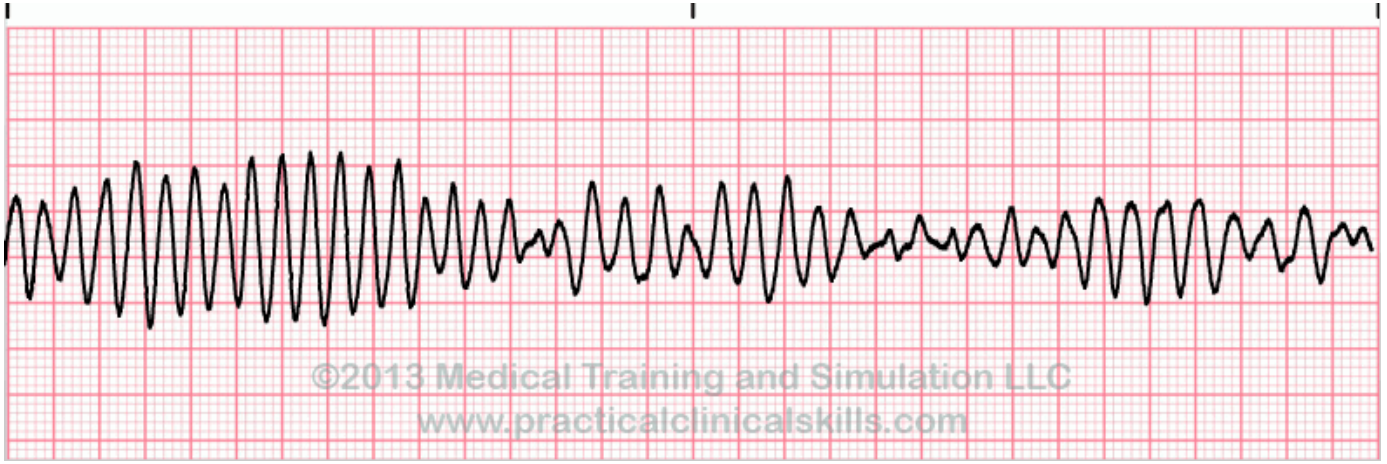
Torsade de Pointes



Regular rhythm with fast rate
Absent P Wave and unmeasurable PR interval

Torsade de Pointes

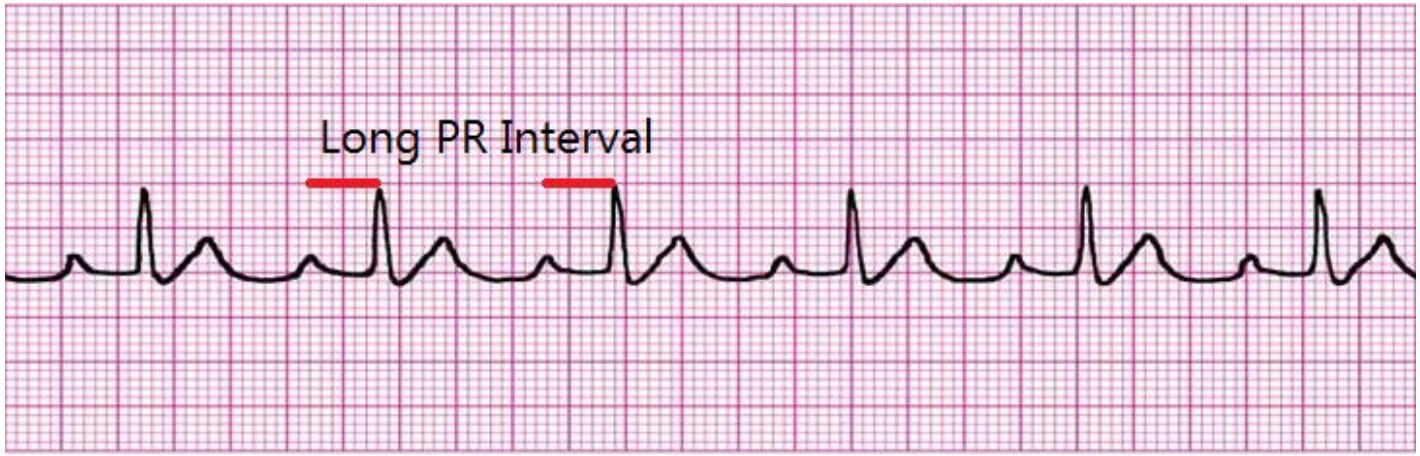
Potential Causes:

- Drugs/Poison
- Electrolyte abnormalities such as hypokalemia
- Medical conditions

- May degenerate to ventricular fibrillation

Atrioventricular Rhythms

First Degree Heart Block



Regular rhythm with prolonged PR Interval

First Degree Heart Block

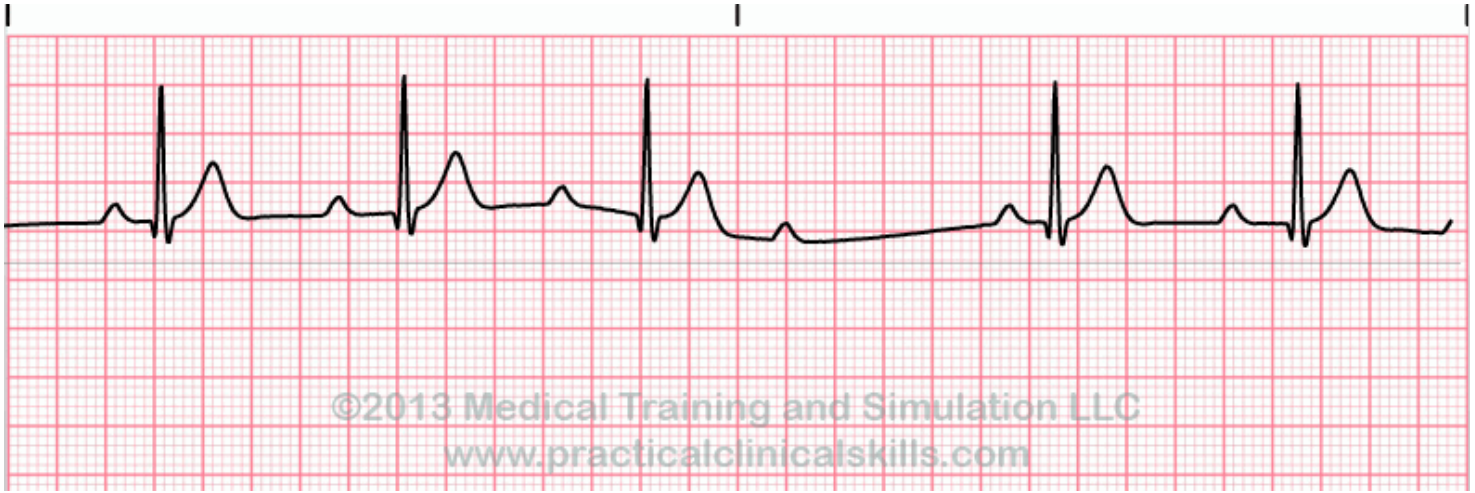
Potential Causes:

- Increased vagal tone
- Athletic training
- Inferior MI
- Mitral valve surgery
- Myocarditis (Lyme disease)
- Electrolyte disturbances (hyperkalemia)
- May be normal

Does not cause hemodynamic disturbance

- No specific treatment required

Second Degree Heart Block Type 1- Wenckebach



Irregular rhythm with progressively longer PR interval until a QRS complex is missed, then cycle repeats

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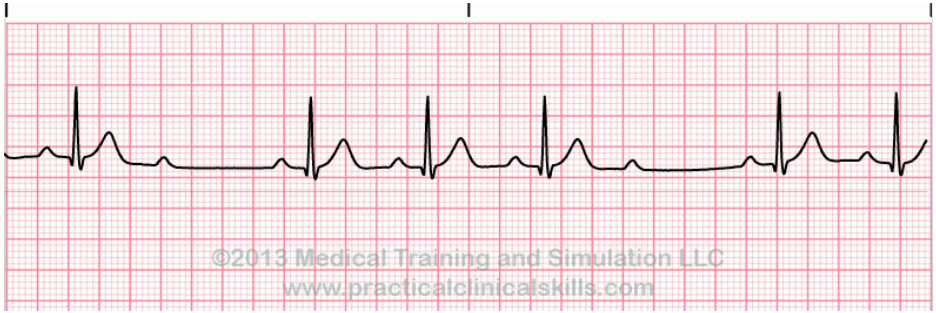
Second Degree Heart Block Type 1- Wenckebach

Potential Causes:

- Medications – beta-blockers, calcium channel blockers, digoxin, Amiodarone
- Increase vagal tone (athletes)
- Inferior MI
- Myocarditis
- Following cardiac surgery (mitral valve repair, tetralogy of Fallot repair)

- Asymptomatic patients do not require treatment
- Symptomatic patients usually respond to Atropine
- Permanent pacing is rarely required

Second Degree Heart Block Type II- Mobitz



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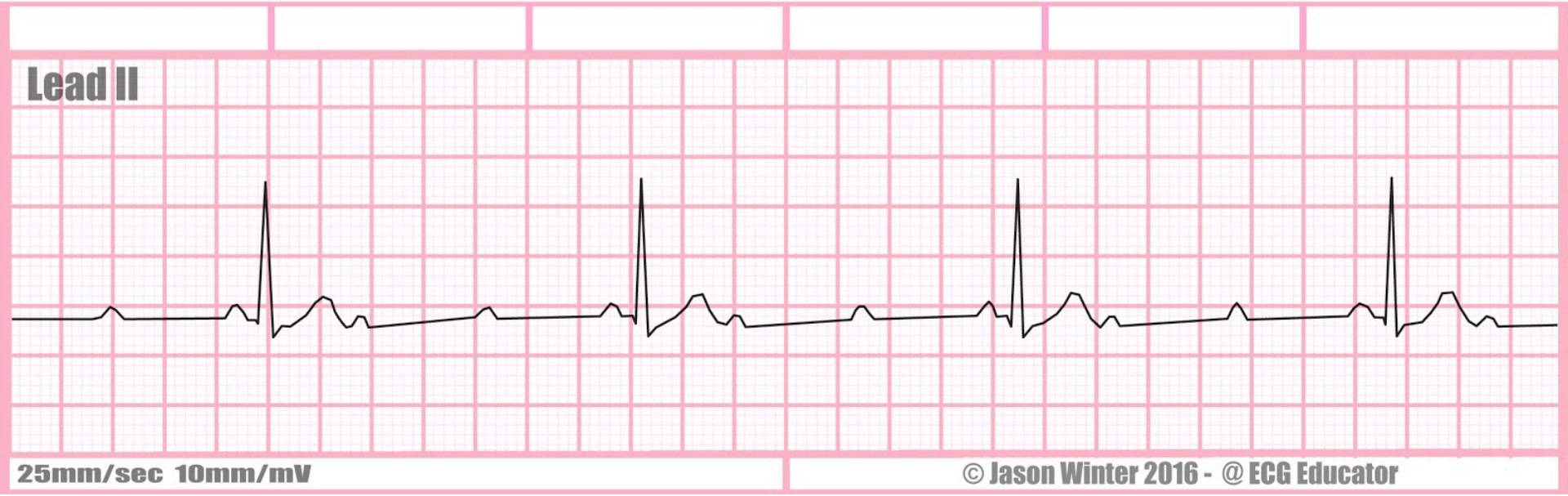
3rd Degree AV Block



Third Degree (Complete) Heart Block

Potential Causes:

- Inferior MI
- AV node blocking drugs: calcium channel blockers, beta-blockers, Digoxin
- Risk for sudden cardiac death
- May require temporary pacing and insertion of permanent pacemaker

Bundle Branch Block

- Right Bundle branch block Next EKG:
- Electrical Conduction problem
- Sometimes treatment not required.
- Possible pacemaker if symptomatic
- Left Bundle branch block
- Treat as MI until proven they have had it

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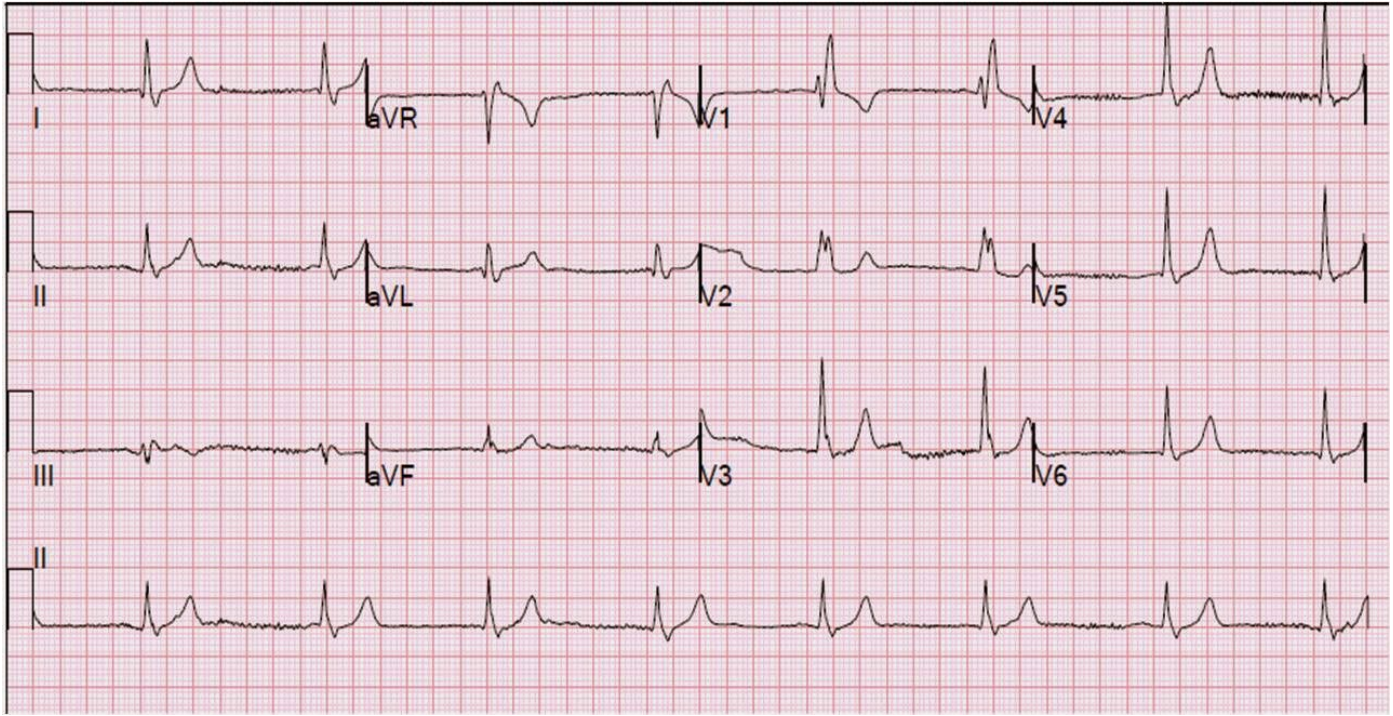
Right Bundle block Branch



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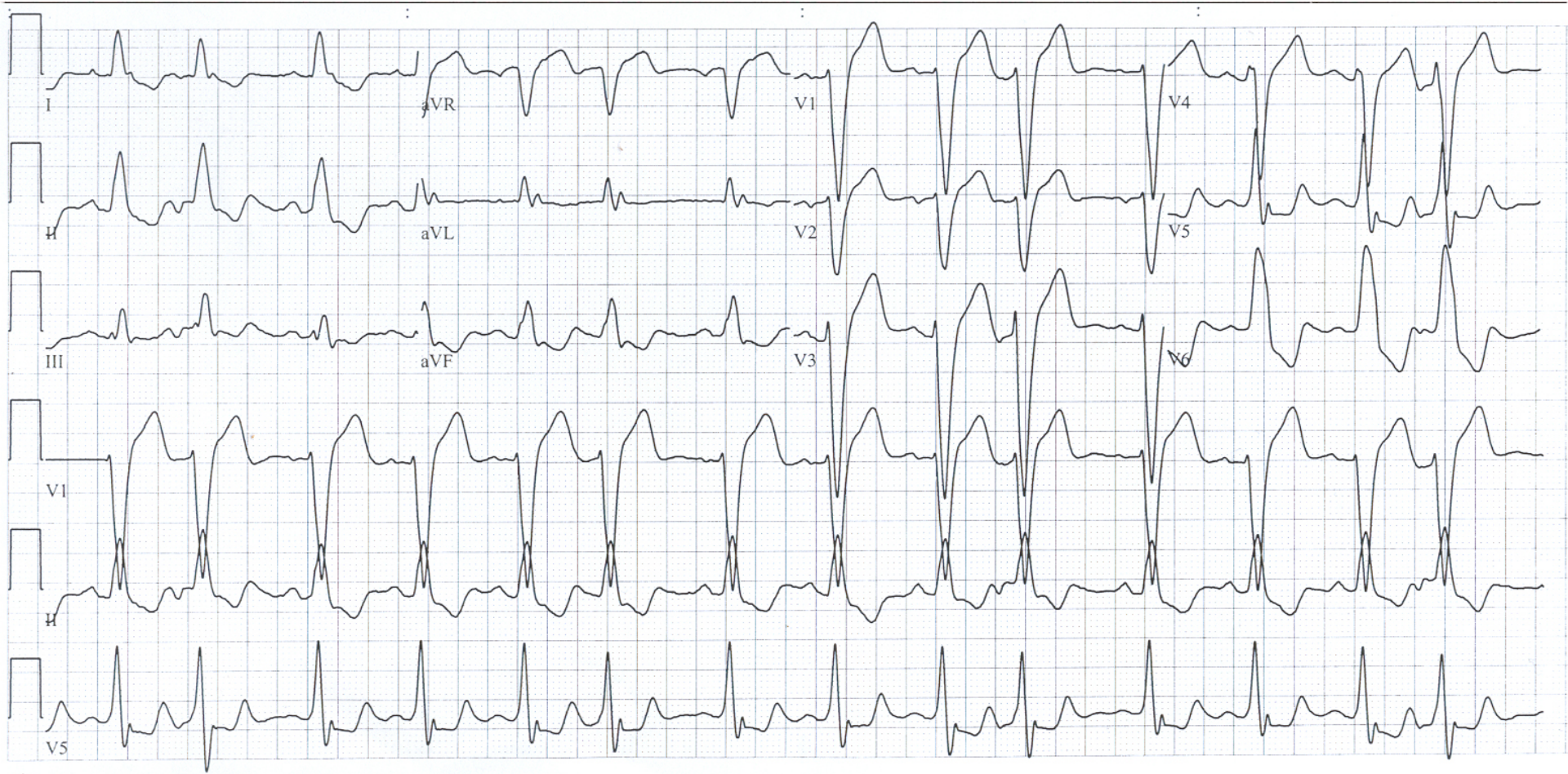
Left Bundle branch block

- See next EKG



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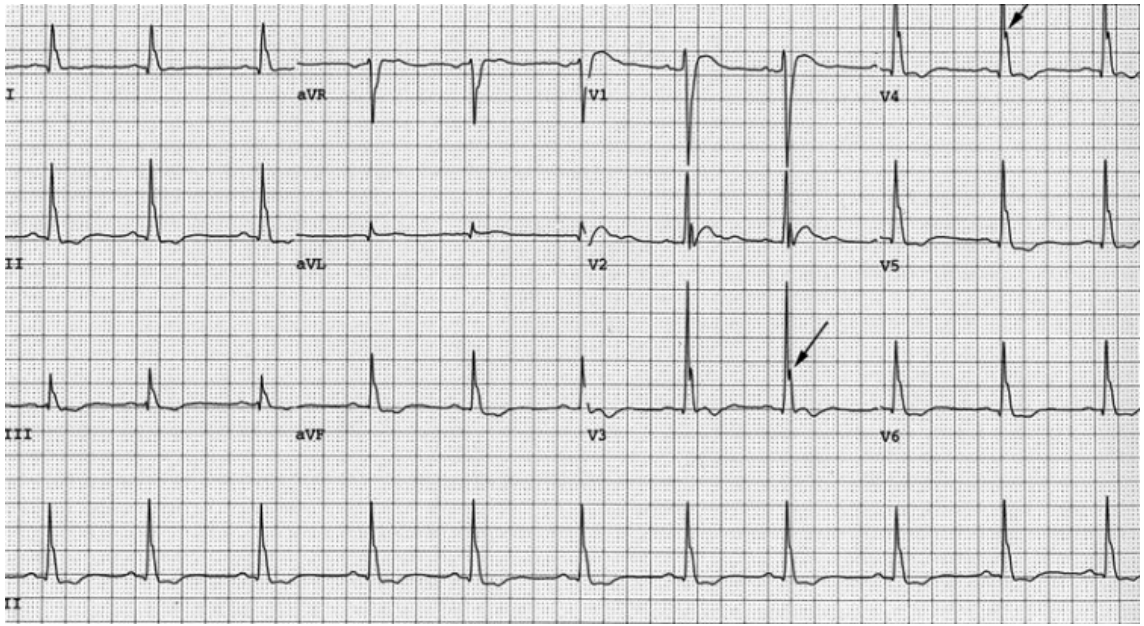
Electrolyte Imbalances

Hypercalcemia



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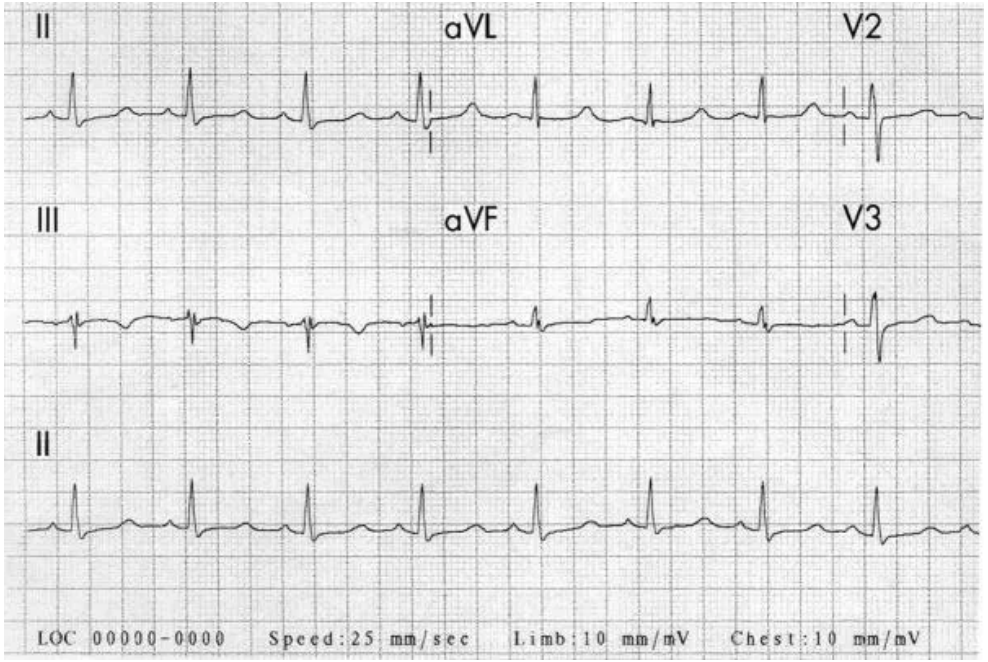
Hypocalcemia



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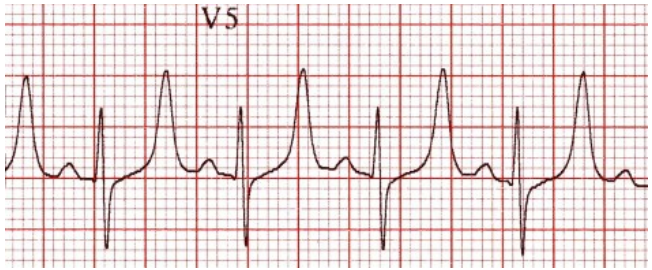
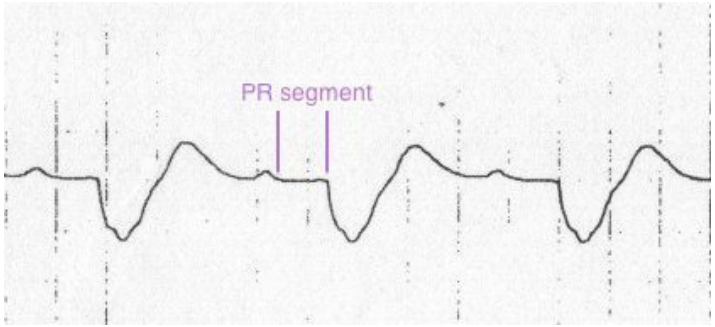
Hyperkalemia



Peaked T Waves

Loss of P Waves



Prolonged PR Interval

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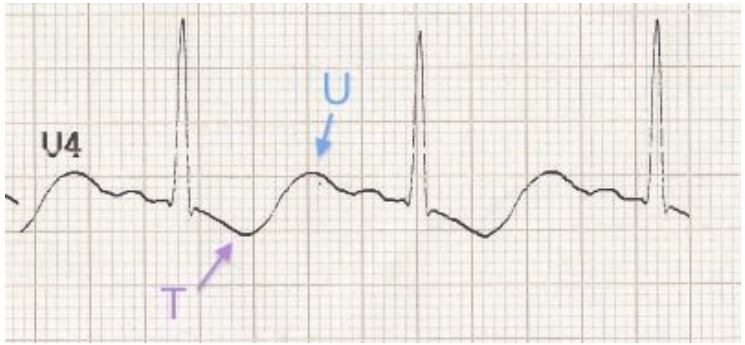
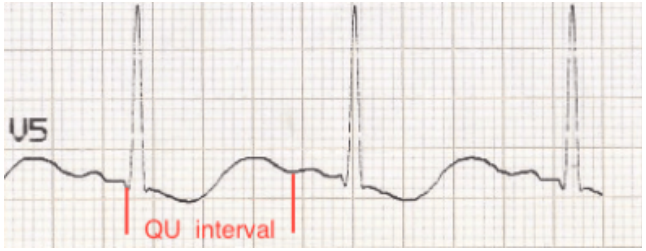
Hypokalemia



T Wave Inversion and Prominent U Waves



Long QU Interval

Pacemaker Rhythms

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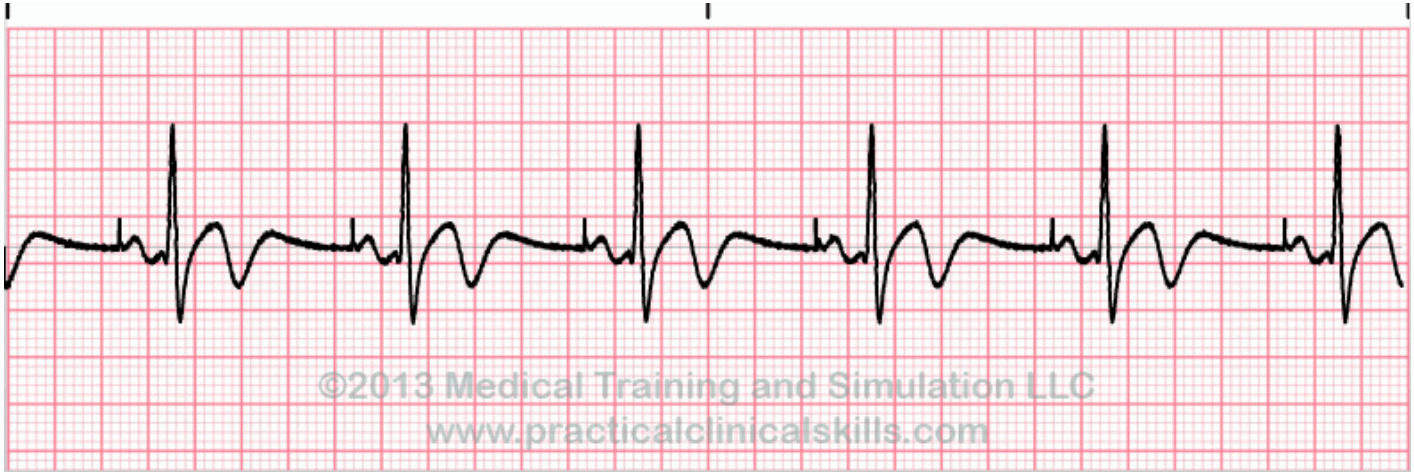
Normal Single Chamber Pacemaker



Pacemaker spike before every beat

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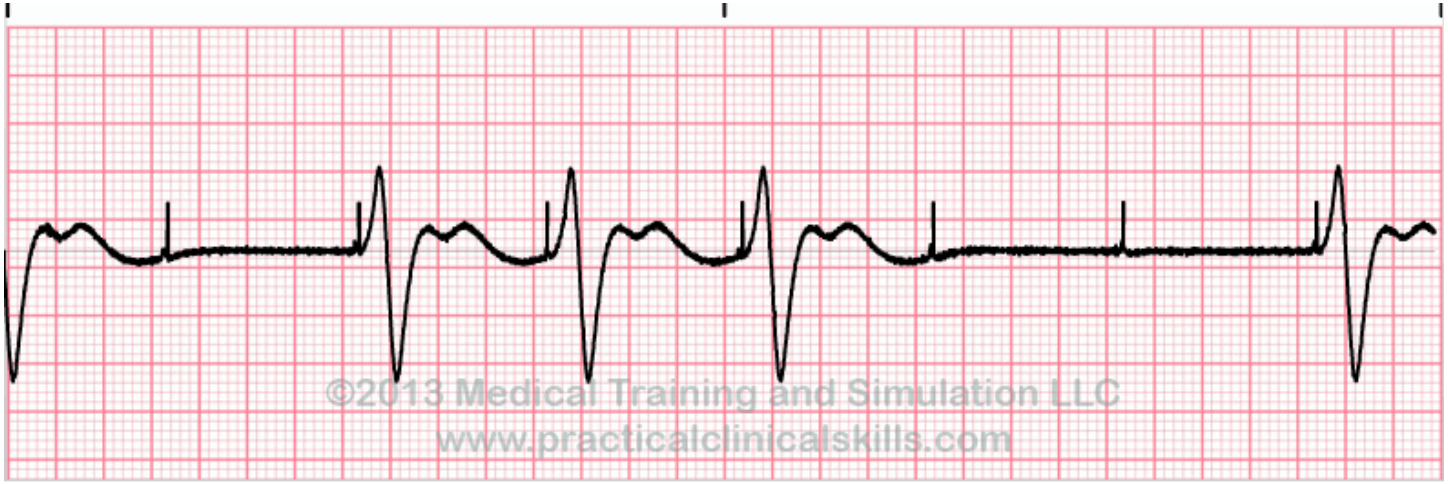
Failure to Capture



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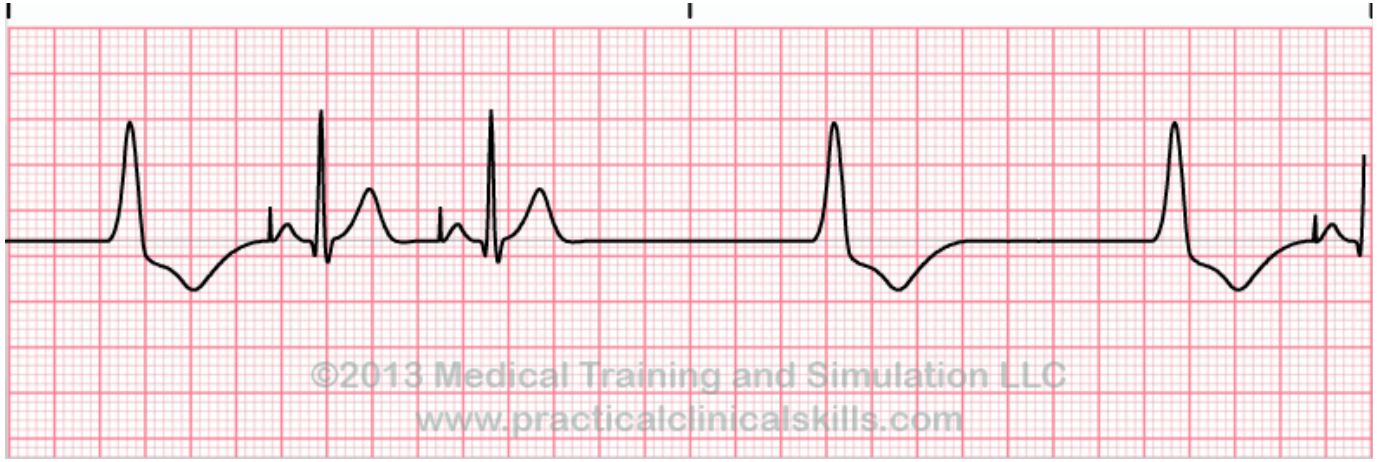
Failure to Pace



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Myocardial Infarctions

Background

- **Troponin** is *more specific to cardiac injury* and remains elevated longer than other cardiac markers
  - With contemporary Troponin assays, CKMB and Myoglobin are not useful for diagnosis of Acute Coronary Syndromes

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Troponin T

- Cardiac Troponin is the preferred marker for myocardial necrosis
  - A Troponin level should be drawn when the patient is first assessed
  - Troponin T levels *rise 4 to 6 hours after the onset of chest pain* and *peak at 48 hours*
  - Troponin T levels remain elevated and plateau for another 2-5 days, typically returning to normal (reference range) levels 7-14 days after onset of infarction

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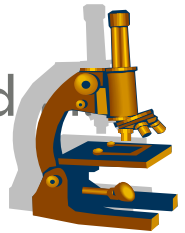
Troponin

- Troponin is primarily elevated due to myocardial injury
  - However, Troponin can be elevated
    - Unstable angina
    - Cardiac contusions/Myocardial stunning
    - Cardiac transplant
    - CABG surgery
    - HF and other conditions that may damage the myocardium



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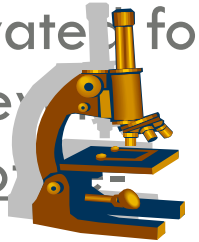
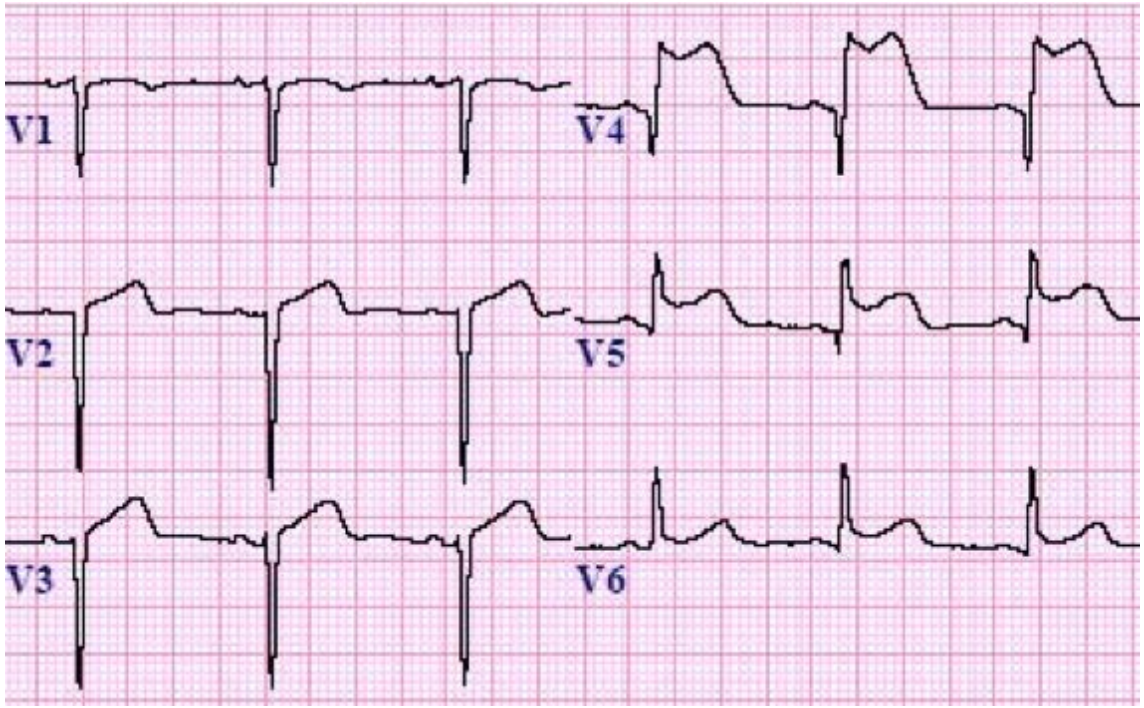
CK-MB

- CK-MB levels in blood *increase 4-6 hours* after onset of infarction and *peak at 12-24 hours*
  - CK-MB levels return to normal (reference range) 2-3 days after onset of infarction
  - Since Troponin levels remain elevated for several days after the initial MI ex… CK-MB is the preferred marker o… infarction



Anterior MI



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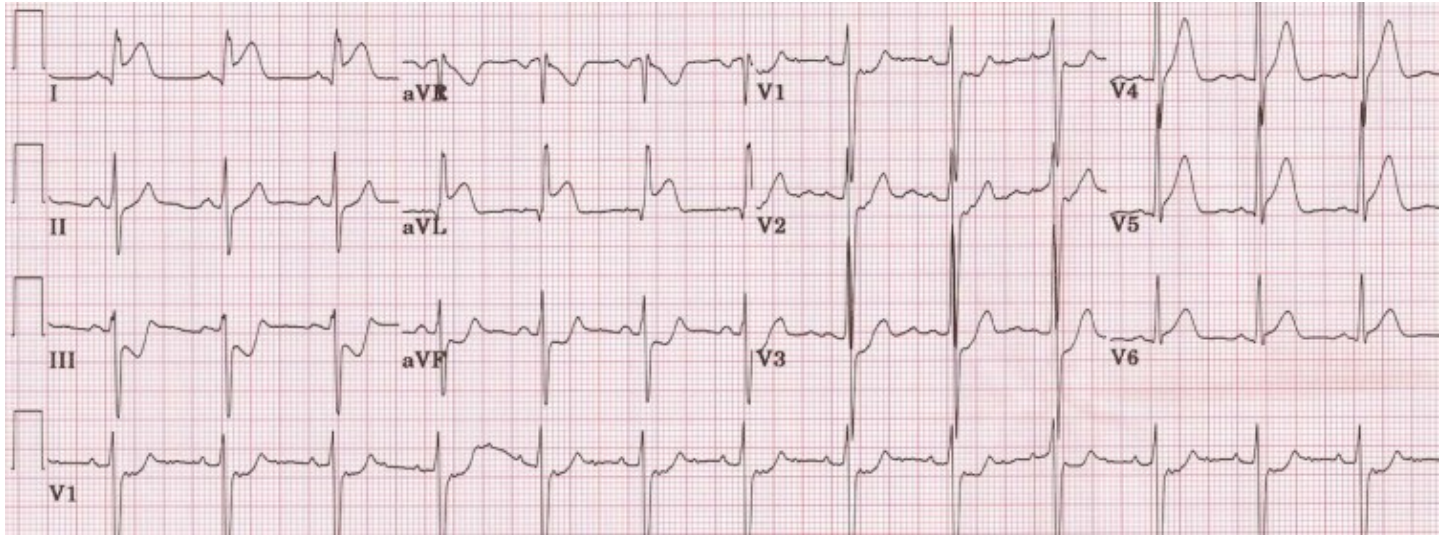
Lateral MI



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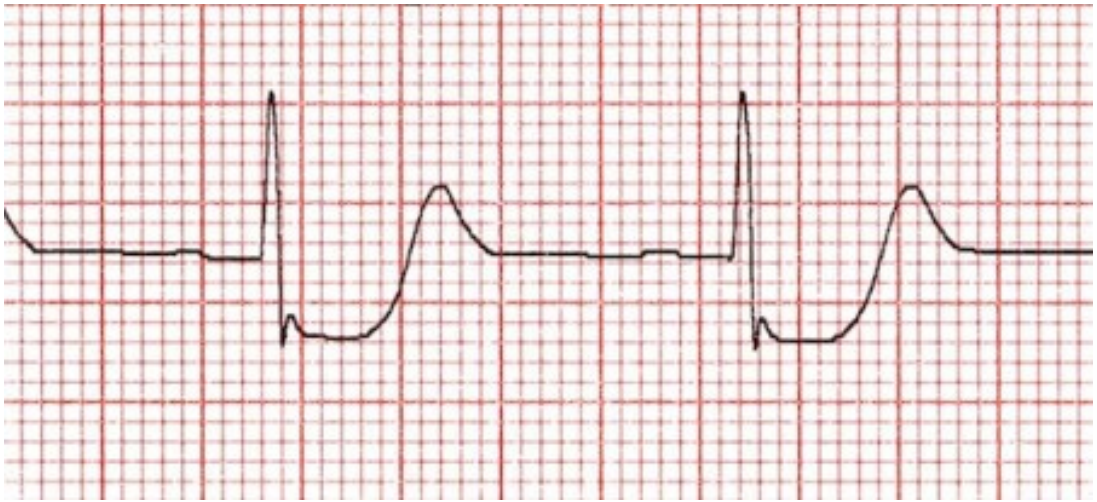
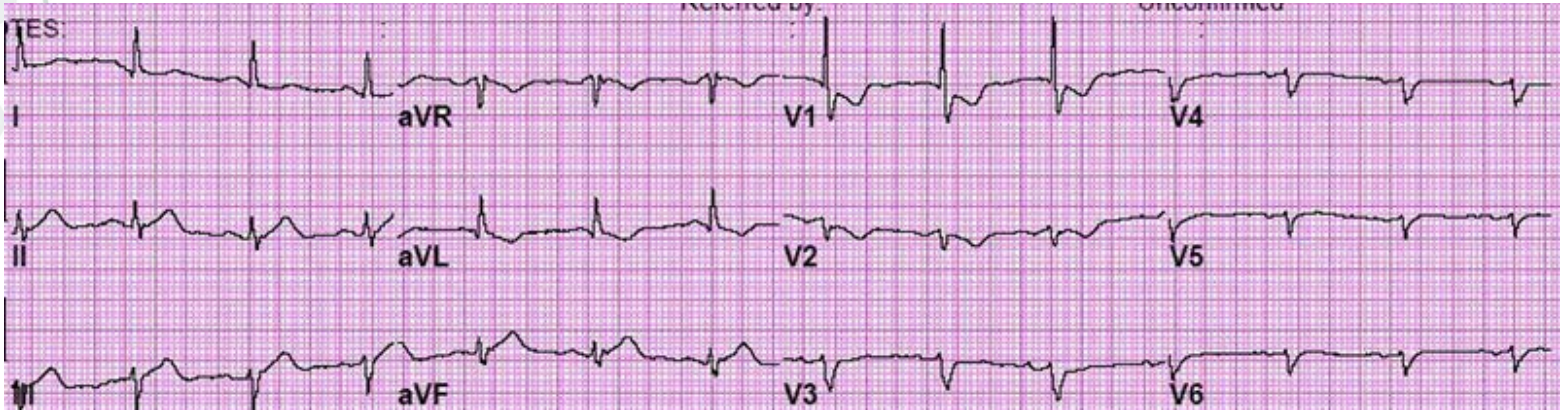
Posterior MI

- Account for 15-20% of STEMIs
- Usually occur with an inferior or lateral infarction
- Lack of obvious ST elevation (depressed, instead!)
    - This causes the diagnosis to be missed



Posterior MI



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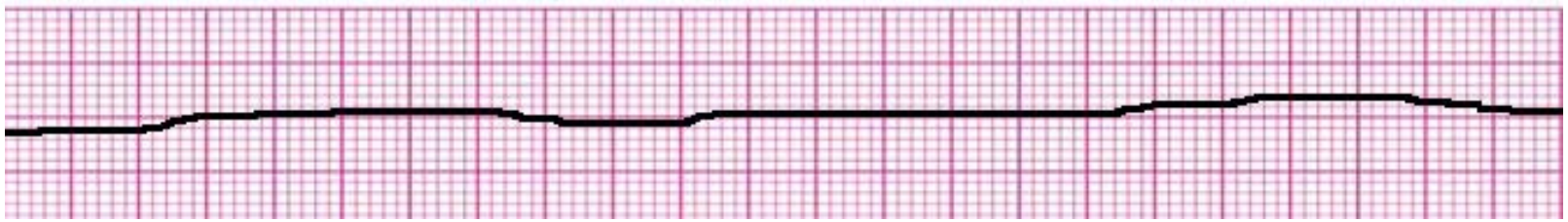
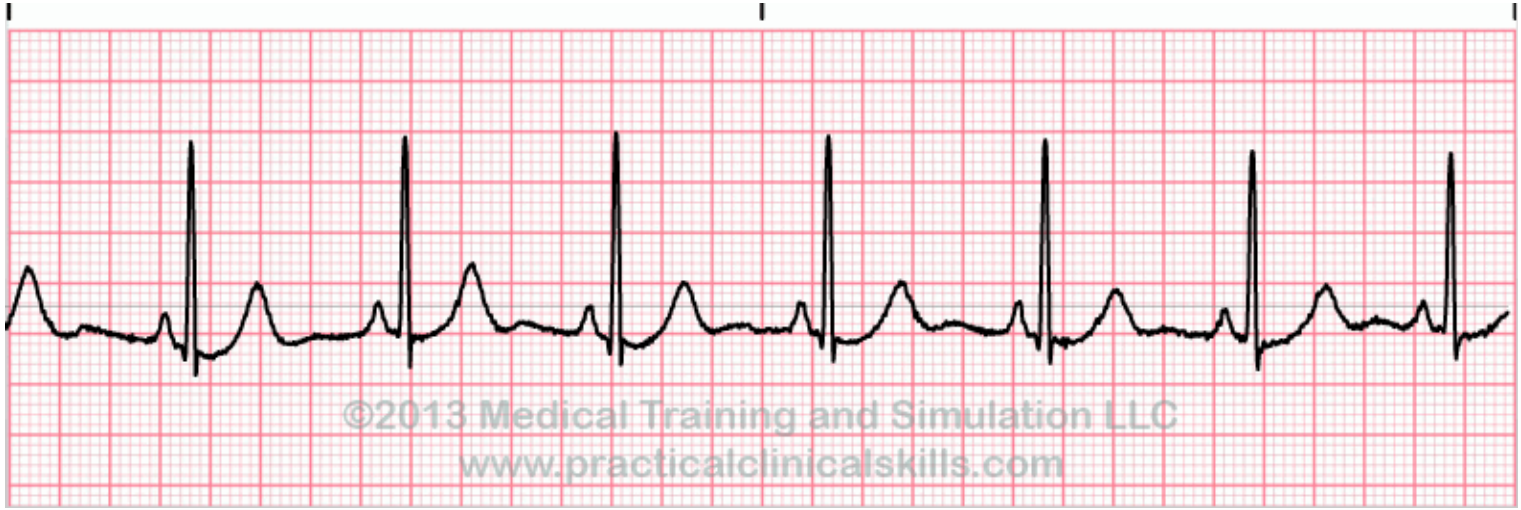
Pulseless Rhythms

Asystole

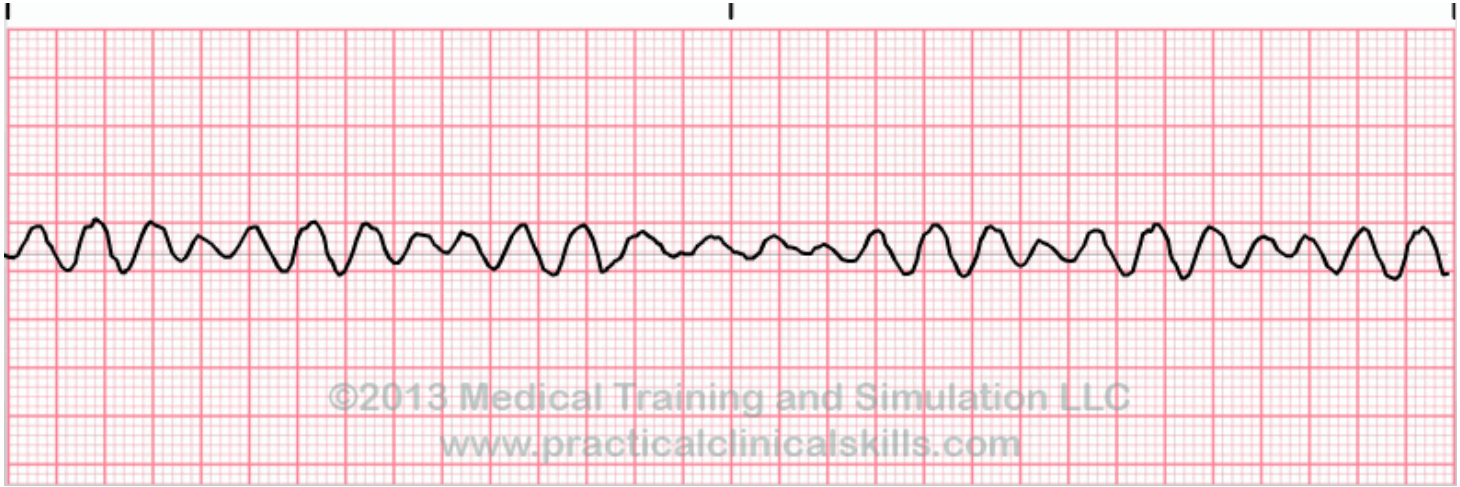


Chest compressions and epinephrine!

Pulseless Electrical Activity (PEA)



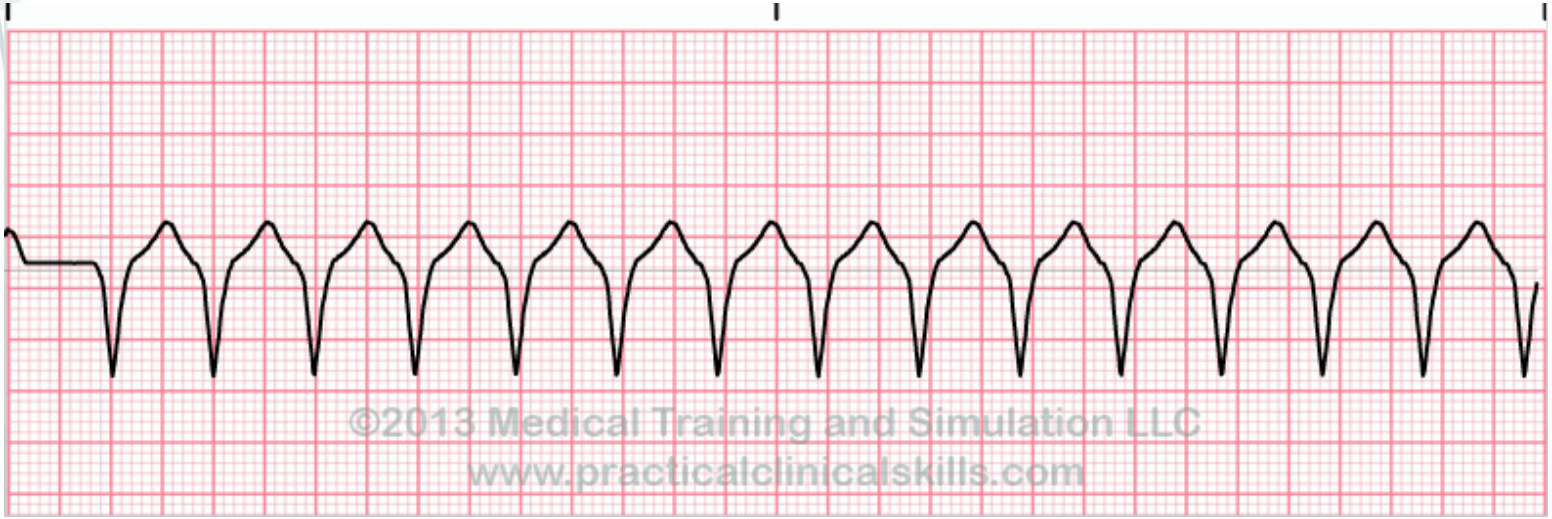
Ventricular Fibrillation



Highly Irregular

Cannot measure rate, P Wave, PR Interval and QRS

Pulseless Ventricular Tachycardia



Demonstration

Transcutaneous Pacing

Cardioversion

Defibrillation

Demonstration – Transcutaneous Pacing

- Assess your patient!
- For severe/unstable bradycardia
- Does your patient have poor perfusion?
  - If so, do not delay pacing!
- May also administer Atropine 0.5mg IV
  - May be repeated every 3 to 5 minutes
  - Maximum dose 3mg or six total doses

Demonstration – Cardioversion

- Synchronized: a LOW energy shock
- Press the “sync” button
- Look for “carrots”
- Complete a Time Out with your team
- Make sure everyone is clear
- Then press SHOCK
- Used for:
  - Unstable atrial fib, atrial flutter, atrial tachy, SVT
  - Unsynchronized cardioversion = defibrillation

Demonstration – Defibrillation

- Used for pulseless ventricular tachycardia and ventricular fibrillation
- May shock at 200J
- Complete a Time Out prior to shock
- Make sure everyone is clear

References

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