

# *Telemetry Study Packet*



# Review of Cardiac Electrophysiology And the Conduction System

## *Cardiac Electrophysiology Terminology:*

**Action potential:** the graphic representation of cellular electrical events, describes the changes in intracardiac voltage that lead to impulse formation and conduction and, ultimately, to cardiac contraction.

**Automaticity:** the ability to initiate impulses spontaneously

**Conductivity:** the ability to conduct an impulse along the cell membrane

**Contractility:** the ability to contract in response to a stimulus

**Depolarization:** A change in the resting electrical state of the cardiac cell  
The inside of the cell becomes more positive and impulse is spread through the heart

**Excitability:** the ability of cells to respond to a stimulus

### **Refractory period:**

**Absolute refractory period:** the cardiac cell is unable to accept or respond to a stimulus

**Relative refractory period:** a stronger than normal impulse may stimulate the cardiac cell to depolarize

### **Repolarization:**

Return of the cardiac cell to the resting membrane state  
The inside of the cell becomes more negative

**Resting Membrane Potential:** the electrical charge across the cardiac cell membrane between impulses, generally about  $-70$  mV

**Threshold:** the electrical level at which the cell will continue to depolarize without additional stimulus, generally about  $-60$  mV

## ***Components of conduction system:***

***Sinoatrial node (SA node):*** Located high in the right atrium, near the junction of the superior vena cava. It is the primary pacemaker of the heart with normal rate of automaticity of **60-100 beats/min.**

***Internodal atrial pathways:*** Conduct the impulse from the SA node through the right atrium musculature to the AV node. Consists of three tracts: Bachmann's tract (anterior, "fast" tract), Wenckebach's tract (middle), and Thorel's tract (posterior, "slow" tract)

***Atrioventricular node (AV node):*** Located low in the right atrium, adjacent to the inter-atrial septum. Delays impulse from atria to allow atrial contraction and synchronizes atrial contribution to ventricular pumping. The impulse is then sent to the ventricles via the bundle of His, normally the AV node is the only structure that is capable to conducting impulses to the ventricles. The AV node can serve as a backup pacemaker. Inherent rate of automaticity is **40-60 beats/min.**

***Bundle of His:*** Arises from the AV node and conducts impulse to bundle branch system.

***Bundle Branch system:*** Pathways that arise from the Bundle of His. Composed of:

**Right bundle branch (RBB):** a direct continuation of the bundle of His. Transmits impulse down right side of the interventricular septum to the myocardium of the right ventricle.

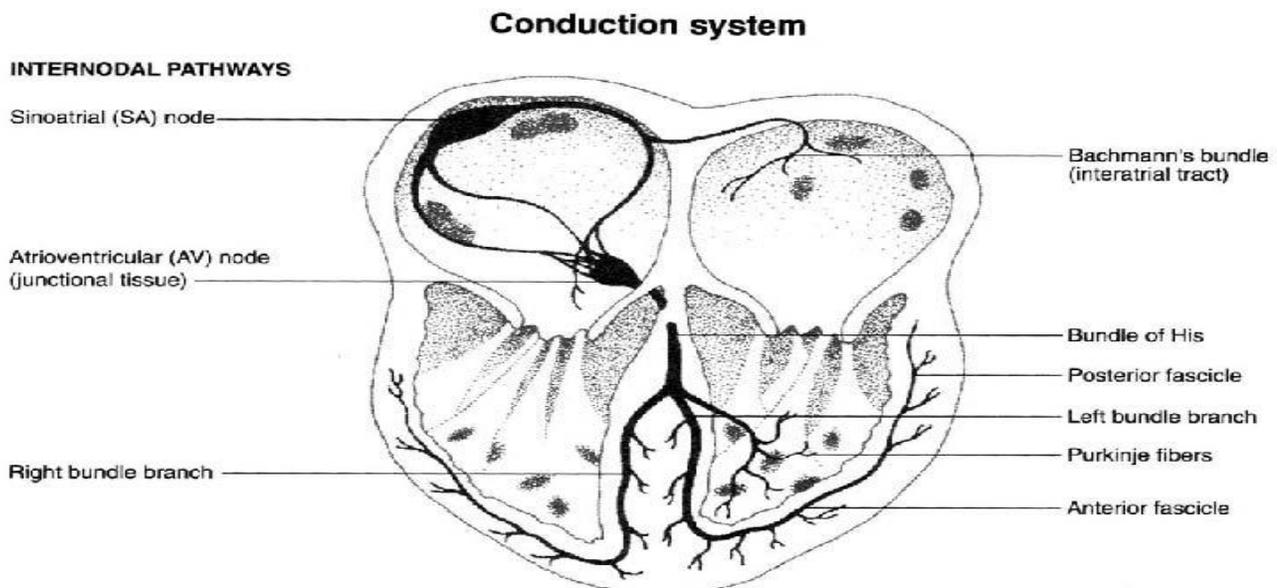
**Left bundle branch (LBB):** separates into three fascicles:

**Left posterior fascicle**—transmits impulse over posterior and inferior endocardial surface of the left ventricle

**Left anterior fascicle**—transmits impulse over anterior and superior endocardial surfaces of the left ventricle

**Septal fascicle**

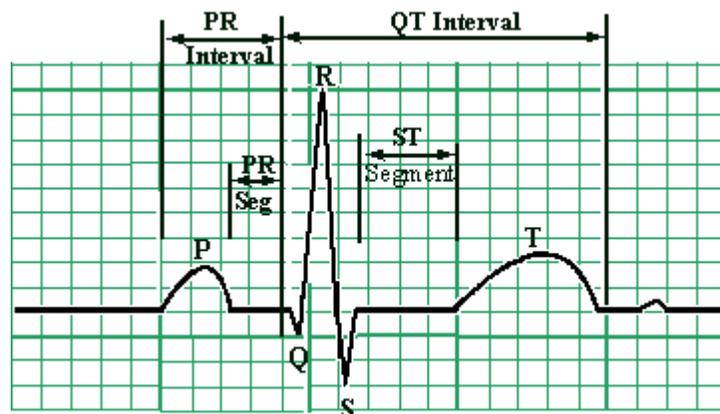
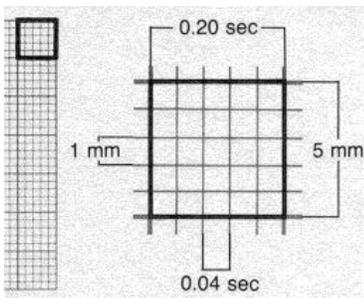
***Purkinje Fibers:*** Arises from distal portion of bundle branches, transmits impulses into subendocardial layers of both ventricles; provides for the depolarization; followed by ventricular contraction and ejection of blood out of the ventricles. Intrinsic rate of **20 to 40 beats/min.**



# RHYTHM ANALYSIS

## I. ECG Markers:

1. Isoelectric Line: flat line between complexes.
2. **P Wave:**
  - a. Indicates that the impulse originated at the SA node and the atrial are normally depolarized.
  - b. Duration: **0.10 seconds**
3. **PR Interval**
  - a. Represents the time required for the electrical impulse to travel from the SA node, through the atria, AV node, Bundle of his, bundle branches and Purkinje fibers.
  - b. Measured from the beginning of the P wave to the beginning of the QRS complex.
  - c. Duration: **.12 - .20 seconds**
4. **QRS complex**
  - a. Represents the time required for the electrical impulse to depolarize the ventricles.
  - b. Measured from the Q wave where the line leaves baseline to the S where the line returns to baseline.
  - c. Duration: **0.04 - .10 seconds**
5. **ST segment**
  - a. Represents the end of ventricle depolarization and beginning of ventricular repolarization.
  - b. Measured from the end of the QRS complex and ends with the onset of the T wave.
  - c. Duration: **.08 seconds**
6. **T wave**
  - a. Represents the latter phase of ventricular repolarization.
  - b. Measured from the beginning as the deflection slopes upward from the ST segment and ends with the line returning to baseline.
  - c. Amplitude: **<5mm**

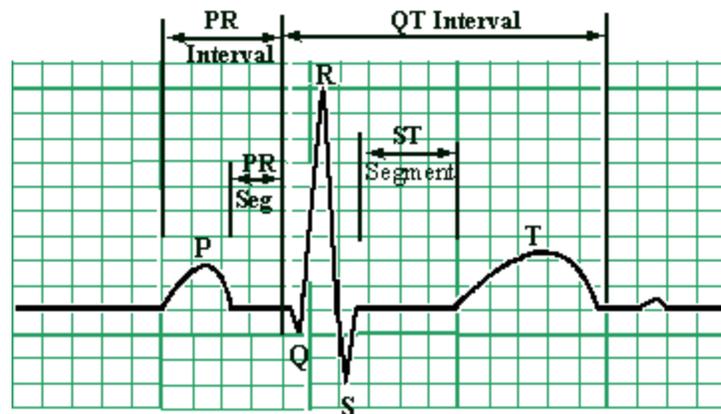
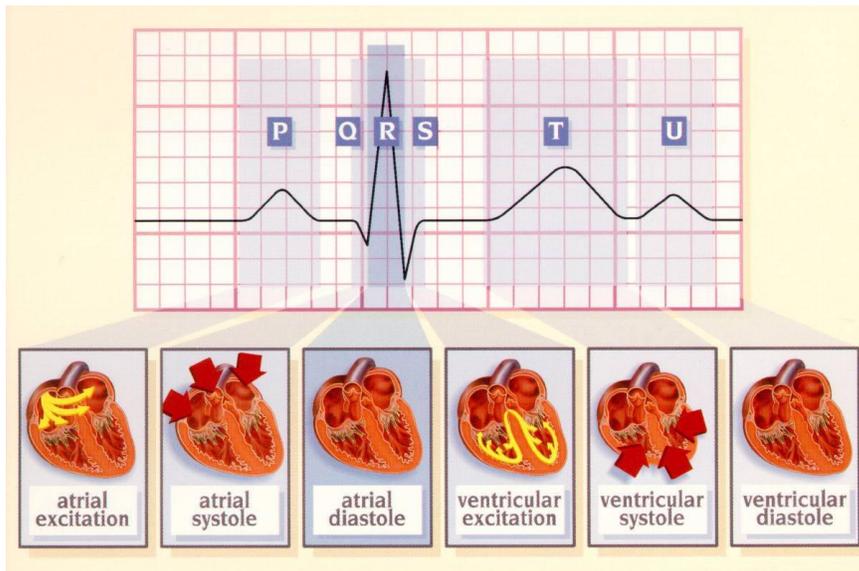


## 7. QT Interval

- Represents the time between the onset of ventricular depolarization and the end of ventricular repolarization.
- Measured from the beginning of the QRS complex to the end of the T wave where the line returns to baseline.
- Duration: **.30 - .40 (varies with the heart rate)**

## 8. U wave

- Represents the repolarization of the Purkinje fibers.
- Usually not seen on an EKG
- May be found with electrolyte disturbances

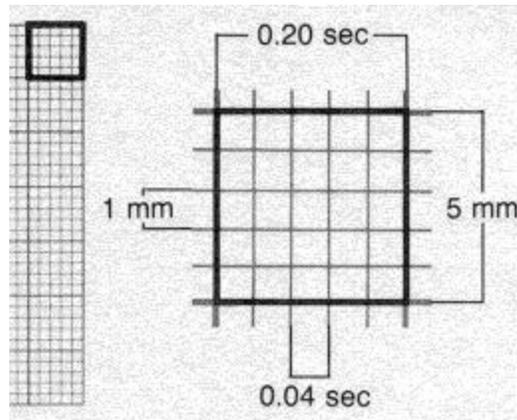


## II. Systematic ECG Interpretation:

### 1. ECG Paper

#### ◦ Horizontally (**TIME**)

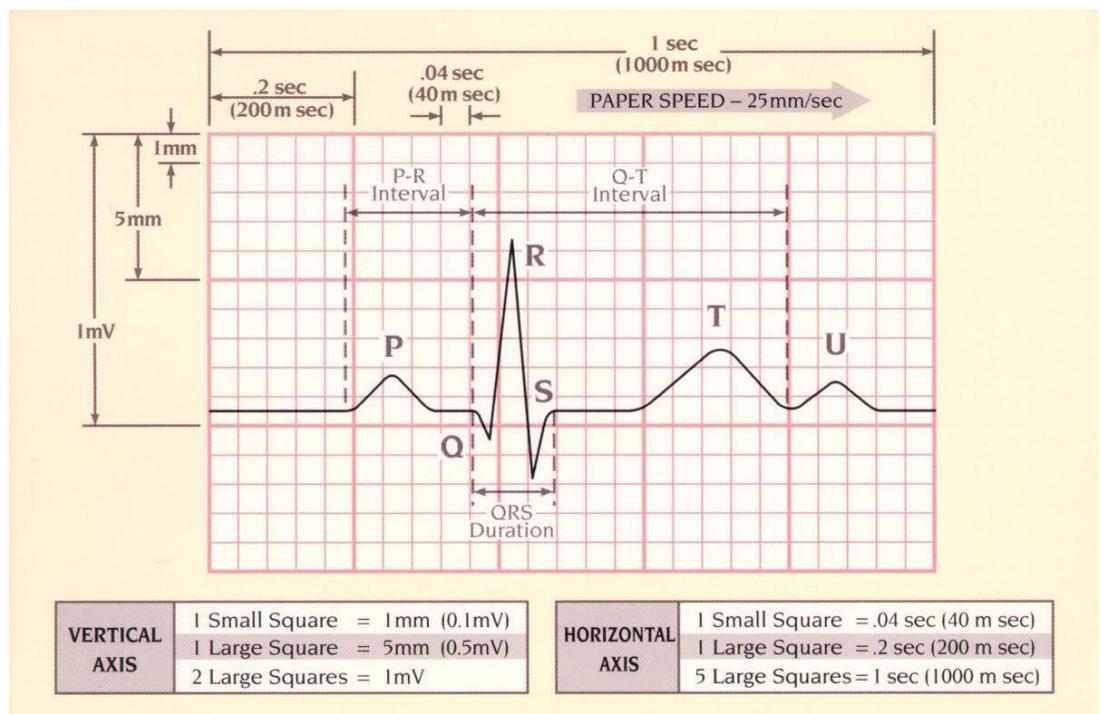
- 1 mm square (small square) represents 0.04 seconds of elapsed time.
- Each (large square) holds 5 small squares and represents 0.20 seconds.
- 1500 small squares represent one minute.
- 300 large squares represent one minute
- 15 large squares represent 3 seconds
- 30 large squares represent 6 seconds



#### ◦ Vertically (**VOLTAGE**)

- 1 mm square (small box) represents 1 mm in height and 0.1 mV of voltage.
- Each (large square) holds 5 small squares and represents 5mm in height or 0.5 mV.

With this information, you can measure the duration of any complex or interval by determining the number of small squares and multiplying by 0.4 seconds.



## 2. Analyzing an EKG Strip

### a. Calculate the *Heart Rate*

1) Two common Methods of calculating the heart rate:

- **Method #1-** (Irregular or Regular Rates) Use the 3 second or 6 second markers on the EKG paper (vertical lines in the upper margin of the EKG paper are measures of time standard to all EKG graph paper. The distance between 2 "tic" marks is 3 seconds, 3 "tic" marks equal 6 seconds)

Count the number of complexes in a 3 second strips (15 large squares) and multiply by 20

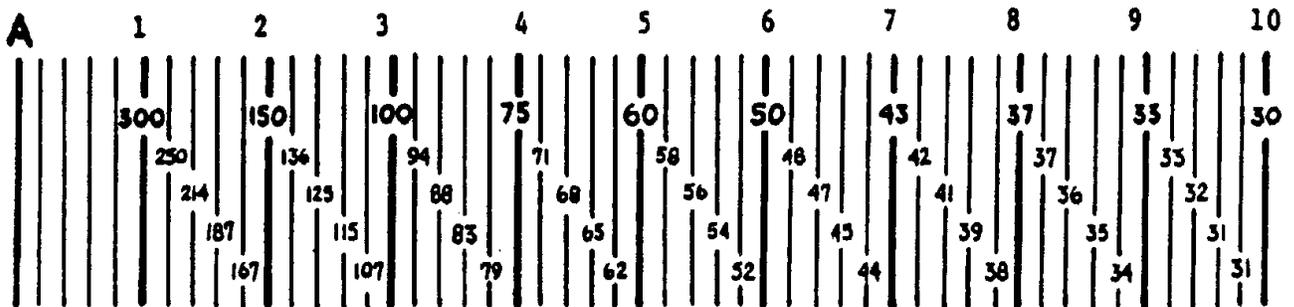
OR

Count the number of complexes in a 6 second strips (30 large squares) and multiply by 10

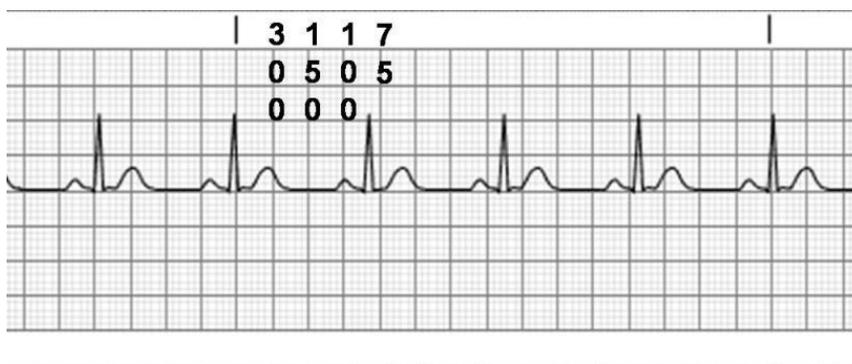
\*\*\*Using **Method #1** count the number of complexes within this 6 second strip. **HR is about 80.**\*\*\*



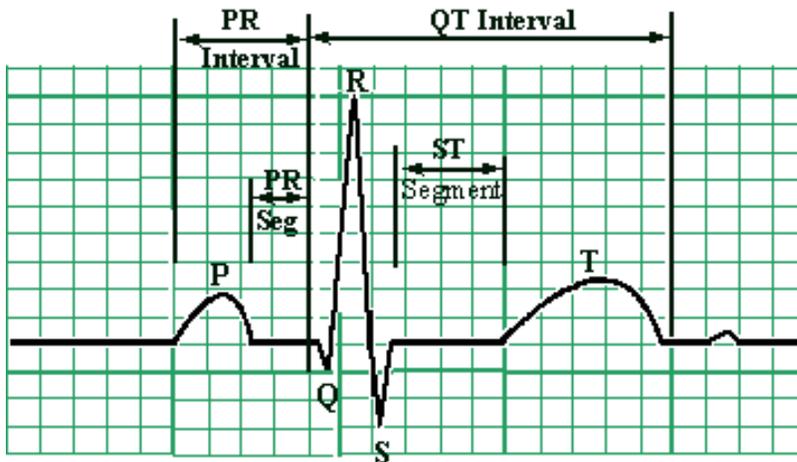
- **Method #2-** (Regular Rates) Start from a QRS complex on a darkened line. Count down using the ruler below, the number of dark lines from this complex to the next. Count is as follows:  
Start- 300-150-100-75-60-50



FOR Example:



1. **Identify and examine the Waveforms:** Evaluate the P waves: a QRS complex should follow every P wave and all P waves should be identical.
2. **Determine the regularity of the R waves.** Is it regular, patterned irregularity, or irregular? Measure the distance between 2 R waves and compare to other intervals, if the intervals are regular then the ventricular rhythm is regular.
3. **Measure the PR interval (PRI):** Normal duration 0.12-.20 seconds
4. **Measure the QRS complex:** Normal duration is  $\leq 0.12$  seconds.



## SINUS RHYTHMS

### What are “sinus” rhythms?

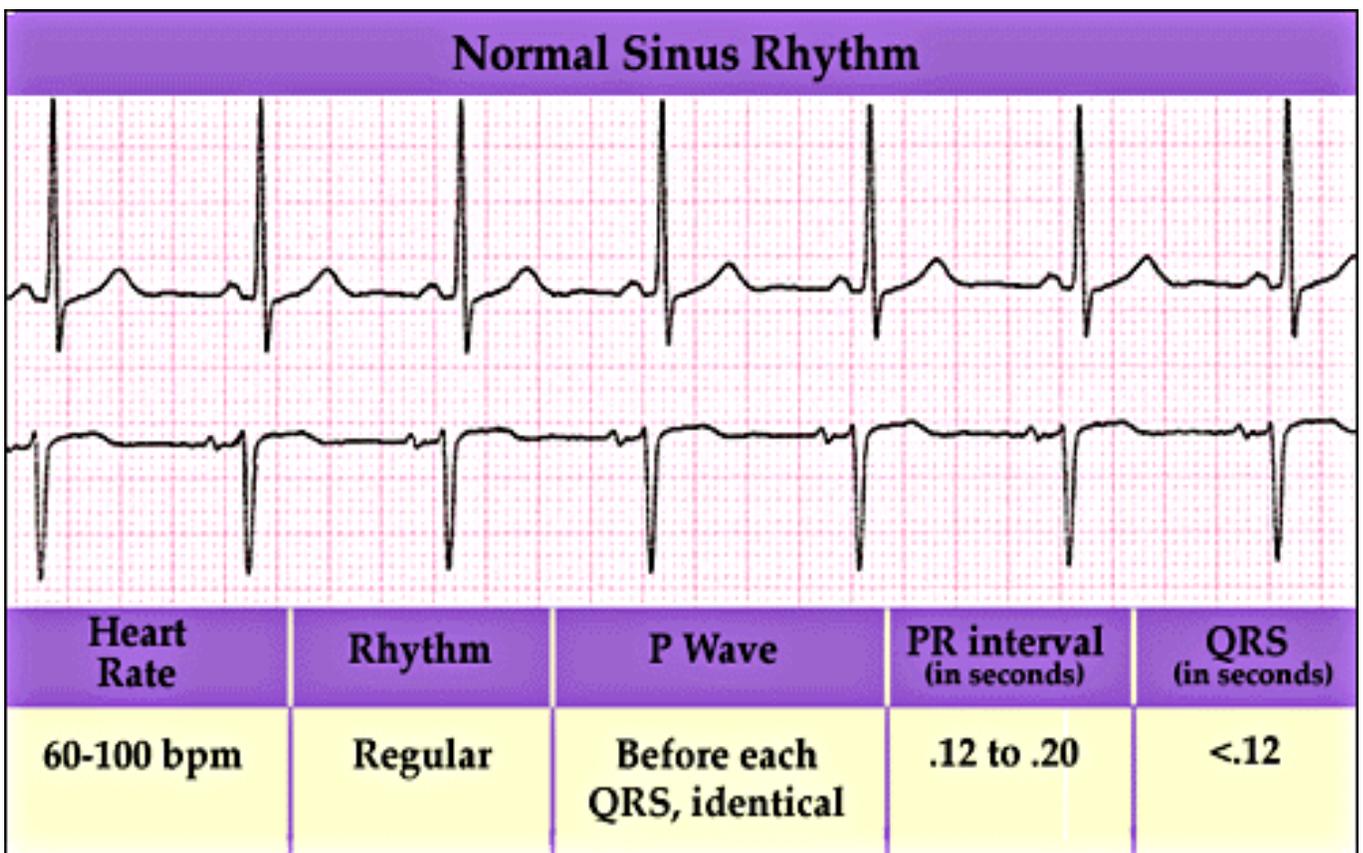
Sinus simply means that the rhythm is produced by electrical impulses formed within the SA node (also called the *sinus* node).

### Determinants of sinus rhythm:

- P wave must be singular and present before each QRS complex
- P wave must be upright and of normal morphology in leads I and II. P wave may be upright, inverted or biphasic in Lead III.
- PR interval and QRS must be constant and of normal duration

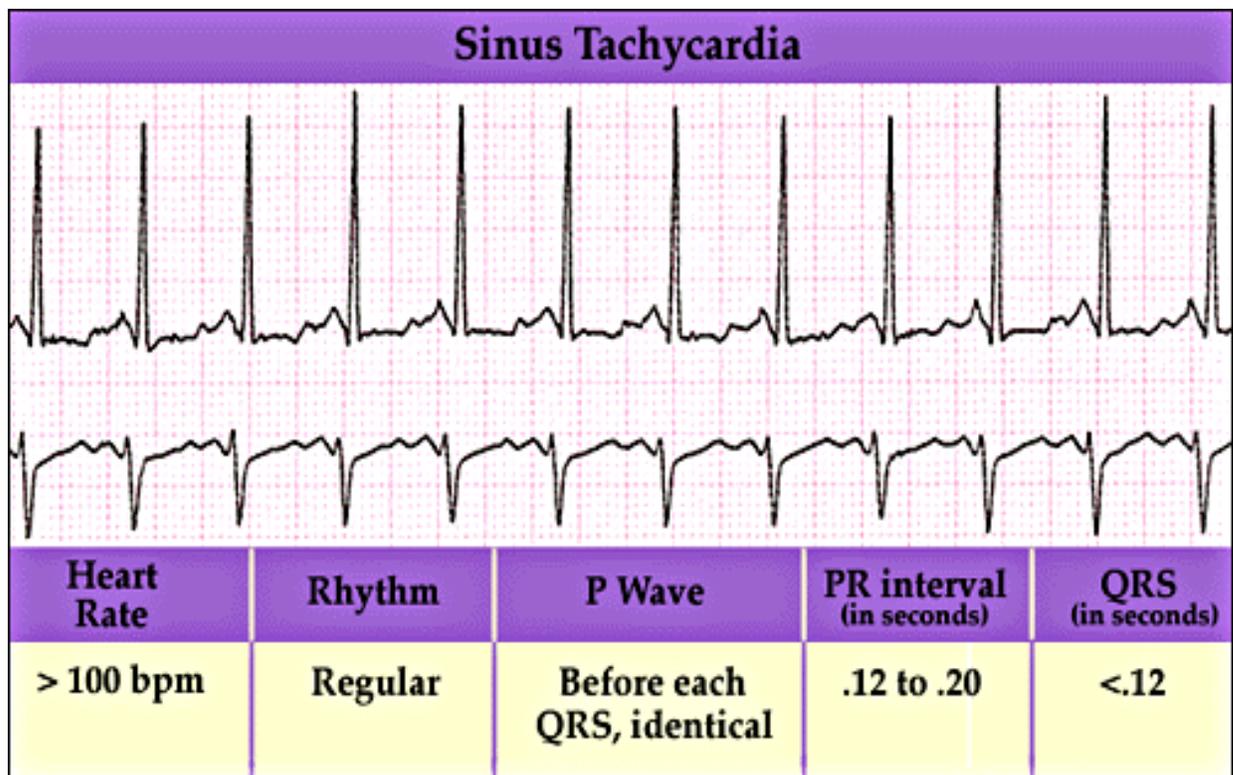
## ***Normal Sinus Rhythm:***

1. Mechanism: Normal cardiac rhythm
2. Significance: Normal cardiac rhythm
3. Clinical Features:
  - a. Rate: 60-100
  - b. Rhythm: Regular
  - c. P waves: 1 occurs before each QRS complex, normally shaped
  - d. PR Interval: 0.12-0.20 seconds
  - e. QRS Complex: 0.04-0.10 seconds
  - f. QT Interval: less than  $\frac{1}{2}$  of the R-R interval



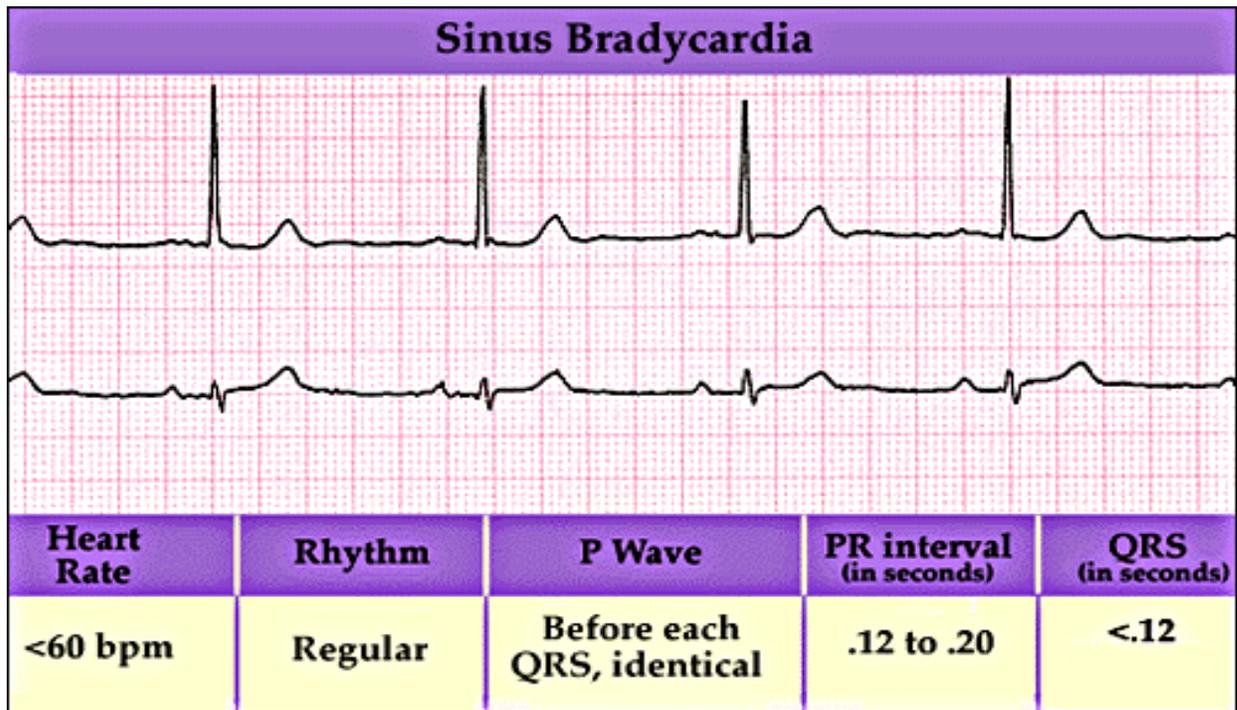
## ***Sinus Tachycardia:***

1. Mechanism:
  - a. Normal response to metabolic needs: exercise, anxiety, fear
  - b. Disease processes: MI, PE, fever, CHF; heart rate responds to compensate for reduced stroke volume, hypoxia, hypovolemia, hyperthyroidism
2. Significance:
  - a. Tachycardia increases work of the heart and oxygen consumption, which may lead to heart failure, ischemia, and increased size of infarction.
  - b. Risks associated with tachycardia depend on etiology. The risk is low if the etiology is stress, anxiety, fever, or physical activity. The risk increases if the etiology is hypoxemia, heart failure, or cardiac disease.
3. Clinical Features:
  - a. Rate: 100-150
  - b. Rhythm: regular
  - c. P waves: 1 occurs before each QRS complex, regularly shaped
  - d. PR Interval: normal
  - e. QRS complex: normal
  - f. Symptoms: May lead to angina, dyspnea.
4. Treatment:
  - a. Investigate and treat the underlying cause
  - b. Pharmacologic interventions: oxygen, pain/fever relievers, beta-blockers, calcium channel blockers



## ***Sinus Bradycardia:***

1. Mechanism:
  - a. Vagal stimulation, normal in athletes, during sleep and inactivity
  - b. Disease: early MI, inferior wall MI, increased intracranial pressure, hypoparathyroidism, hypothermia
  - c. Drugs: digitalis, propranolol, morphine
2. Clinical Significance:
  - a. Reduced cardiac output resulting in decreased coronary and cerebral blood flow leading to angina, syncope or heart failure.
  - b. Slowly discharging SA node may allow a more rapidly discharging ectopic focus to take over role of cardiac pacemaker, predisposing patient to atrial and ventricular dysrhythmias including ventricular ectopy.
3. Clinical Features:
  - a. Rate: less than 60
  - b. Rhythm: regular
  - c. P waves: 1 occurs before every QRS complex, regularly shaped
  - d. PR interval: normal
  - e. QRS complex: normal
  - f. Symptoms: fatigue, angina, dyspnea, syncope, hypotension
4. Treatment:
  - a. Only treat if symptomatic!
  - b. Atropine if symptomatic
  - c. Cardiac pacing
  - d. Avoid medications with known bradycardic effects (digitalis, morphine, beta-blockers)



# SUPRAVENTRICULAR TACHYCARDIA (SVT)

1. Mechanism:
  - a. A narrow complex tachycardia or atrial tachycardia which originates in the 'atria' and is so fast the origin cannot be determined
  - b. Disease:
  - c. Drugs:
  - d. Other
  
2. Clinical Significance:
  - a. Increased rate will cause increase in myocardial oxygen demands creating ischemia for those with heart disease
  - b. The ability to tolerate the increased rate will determine the urgency of treatment
  
3. Clinical Features:
  - a. Rate: 140-220 beats per minute
  - b. Rhythm: regular
  - c. P waves: difficult to find due to the rate
  - d. PR interval: cannot measure but if slowed the PR will vary depending on the location of the stimuli in the atria
  - e. QRS complex: normal
  - f. Symptoms: fatigue, angina, dyspnea, syncope, hypotension
  
4. Treatment:
  - a. Cardiovert if unstable!
  - b. Decrease rate with beta blocker, calcium channel blockers, or prainamide

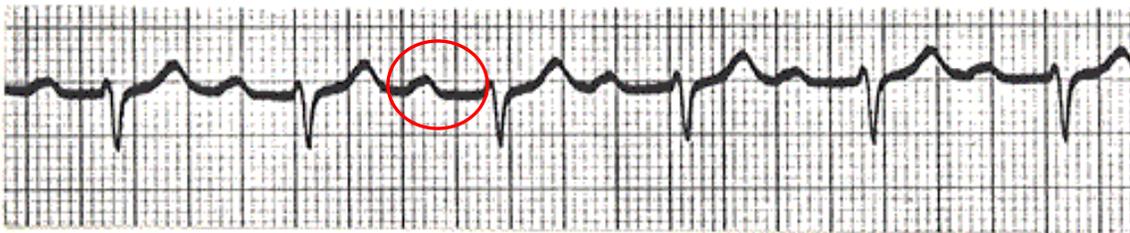


# HEART BLOCKS:

*Alteration in conduction through the AV Node. There are 3 main types-1<sup>st</sup>, 2<sup>nd</sup>, and 3<sup>rd</sup> degree. Second degree AV blocks have 2 types- Mobitz I and Mobitz II*

## *First Degree A-V Block*

1. Mechanism-
  - a. Progress of the impulse slows because of a prolonged conduction delay at the AV Node thus increasing the time for the impulse to reach the ventricles.
  - b. Diseases: Ischemia, Myocardial Infarction (acute inferior wall), infection, vagal stimulation, Hyperthyroidism, rheumatic fever, Hyperkalemia, and congenital abnormality
  - c. Medications: side effect of Digoxin, Beta and Calcium channel blockers, Quinidine and Procainamide
2. Clinical Significance- Usually clinically asymptomatic, symptoms are more likely to be present if underlying rhythm is bradycardia. Can be a warning sign of advancing heart block especially when acute myocardial infarction is present.
3. Clinical Features
  - a. Rate: Normal to fast
  - b. Rhythm: regular
  - c. P Waves Present, normal and P wave precedes every QRS
  - d. QRS: present, normal duration < .12 seconds
  - e. PR Interval ***Prolonged > 0.20 seconds and remains constant with QRS complex measuring normal***



**1st degree AV block (PR = 280 ms)**

PRI: > 0.20 seconds!!!

## ***Second Degree A-V Block Mobitz Type I (Wenckebach)***

1. Mechanism-
  - a. Conduction Impulse is conducted normally to the AV Node but each successive impulse has more and more difficulty passing through the AV Node, until finally an impulse does not pass through.
  - b. Diseases: Usually a transient phenomenon often caused by increased vagal tone, acute infections (rheumatic fever and myocarditis), MI (acute inferior wall),
  - c. Medications: side effect of Digoxin, Beta and Calcium channel blockers, Quinidine and Procainamide
2. Clinical Significance- Self limiting, transient and reversible and seldom serious but can advance to a higher degree of heart block. Patients are usually asymptomatic unless underlying rhythm is bradycardia.
3. Clinical Features
  - a. Rate: Atrial rate usually regular Ventricular rate irregular secondary to progressive lengthening of PR
  - b. Rhythm: irregular
  - c. P Waves Present, more P waves than QRS P waves proceed each QRS, except for blocked beat
  - d. QRS: present, normal duration < .12 seconds
  - e. PR Interval progressive prolongation of PR Interval until QRS is dropped (dropped beat)
4. Treatment Monitor patients' hemodynamic status. Pacemaker on stand-by for rapid use (1<sup>st</sup> choice) or Atropine as second choice bradycardia

**\*\*\*Hallmark\*\*\***

**PR Interval Progressive lengthening in PR interval until a QRS is dropped.**



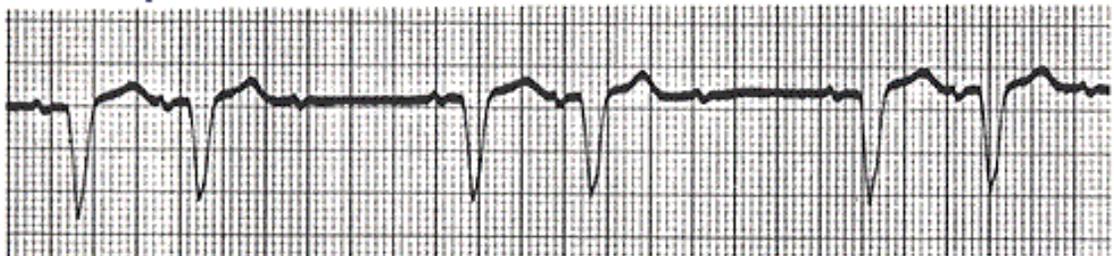
## ***Second Degree A-V Block Type II (Mobitz II)***

1. Mechanism-
  - a. Conduction: Blocking of sinus impulses is now lower at the Bundle of His or Bundle Branches, these conductive pathway becomes selective which impulses it allows through to the ventricles
  - b. Diseases: Acute anterior or anteroseptal MI, cardiomyopathy, rheumatic heart disease, severe coronary disease, age related degeneration of the hearts conduction system, Hyperkalemia, and Myocardial Ischemia
  - c. Medications: side effect of Digoxin, Beta and Calcium channel blockers, Quinidine and Procainamide
2. Clinical Significance- Can progress suddenly to Third Degree Heart Block or Asystole
3. Clinical Features
  - a. Rate: Atrial rate usually regular Ventricular rate irregular secondary to blocked beats
  - b. Rhythm: irregular a nonconducted QRS may occur in a cyclic pattern, 2:1, 3:1, 4:1
  - c. P Waves Present, more P waves than QRS P waves proceed each QRS, except for blocked beat
  - d. QRS: present, normal duration < .12 seconds Can be wide depending on the level of the block
  - e. PR Interval ***remains constant with QRS complex measuring normal***
4. Treatment Monitor patients' hemodynamic status and watch out for 3<sup>rd</sup> degree AV Block. Pacemaker, Atropine or if that is not effective Dopamine drip, support blood pressure with fluid bolus
  - \*\* Note that Atropine increases firing from SA node and the speed of conduction through the AV node, however it has little or no effect on blocks below the AV Node

***\*\*Hallmark\*\****

***PR Interval will remain constant for conducted beats but you have dropped beats***

**Lead V<sub>1</sub>**



**2nd degree AV block (type II) with LBBB**

## Complete or Third Degree AV Block

### LETHAL DYSRHYTHMIA ALERT!!!

1. Mechanism-
  - a. Conduction Impulse- the atrial impulse is blocked completely in the AV Node so the ventricle is pacing independently of the atria
  - b. Diseases: Ischemia, Acute Anterior and Inferior MI, disease of the interventricular conduction system, increased Vagal tone, rheumatic heart disease, myocarditis, endocarditis, following cardiac surgery, Arteriosclerotic heart disease, organic heart disease, chronic degeneration of the hearts conduction system in the elderly
  - c. Medications: side effect/toxicity of Digoxin, Beta and Calcium channel blockers, Quinidine and Procainamide
2. Clinical Significance- markedly affects cardiac output and results in poor central perfusion causing syncope, dyspnea, heart failure, hypotension, chest pain and decreased organ perfusion. Has potential to progress to Asystole. In the presence of MI it will require temporary pacing until healing is resolved and need for permanent pacing can be assessed.
3. Clinical Features
  - a. Rate: Atrial rate according to underlying rhythm/rate (60-100). Ventricular rate is < atrial rate, (40-60 BPM if escape is at AV Node) (20-40 BPM if escape is below the Ventricle)
  - b. Rhythm: P-P regular and R-R regular but they are not associated
  - c. P Waves Present, normal
  - d. QRS: present, width depends on escape focus, narrow if from Junctional tissue or Bundle of His and wide if from Purkinje network normal duration < .12 seconds
  - e. PR Interval varies without consistent pattern because the atria and ventricles are separated electrically
4. Treatment Assess patients' hemodynamic status. Transthoracic Pacemaker on stand-by for rapid use (1<sup>st</sup> choice) or Atropine\*\* as second choice while preparing for Transvenous Pacemaker Continuously monitor the patient and their hemodynamic response.

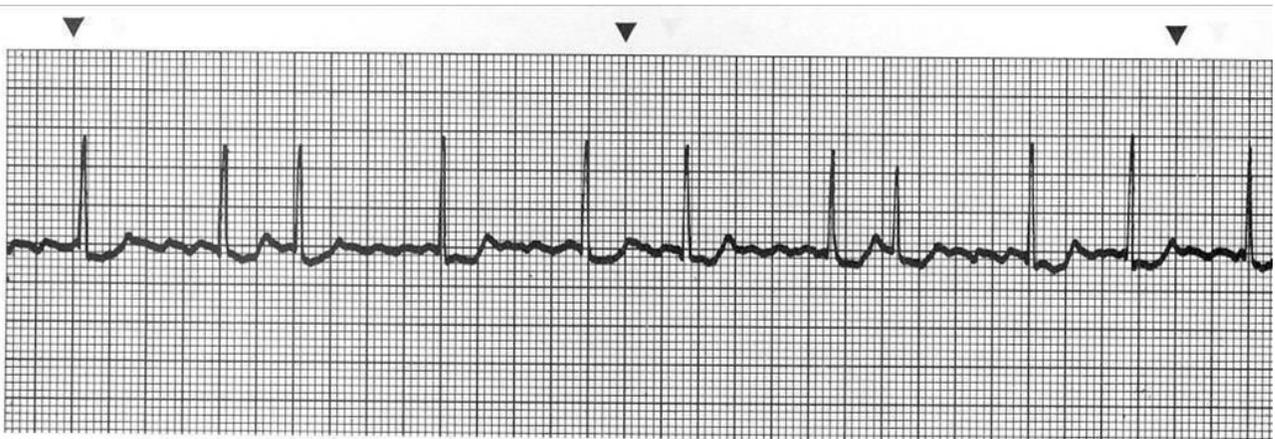
\*\* Atropine can be effective if the block is in the nodal area but is relatively ineffective in lower level blocks in this case use Dopamin or Epinephrine DRIP



*Yellow Arrows are Ventricular (QRS) and Atria (P waves) are indicated by red arrows. Note both are regular but no pattern or consistency in PR Interval. They are not associated.*

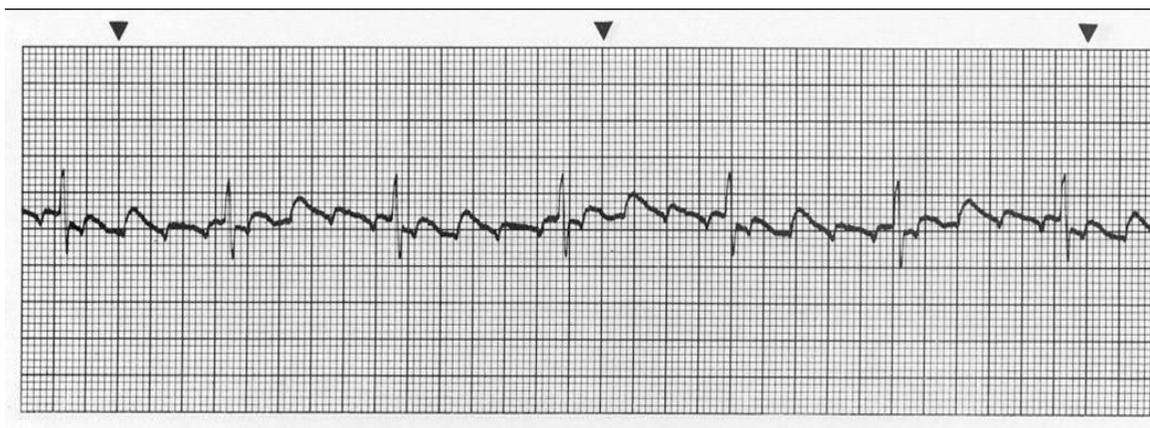
# Atrial Fibrillation

1. Mechanism-
  - a. Conduction Impulse- The atria have no organized electrical conduction just fibrillation however the conduction through the ventricles remains intact.
  - b. Diseases: **CHF**, Corpulmonale, pulmonary disease, acute MI, hypoxia, cardiomyopathy, pericarditis, thyroid toxicosis (hyperthyroidism), **rheumatic heart disease**, valvular heart disease, emotional distress, post cardiac surgery, and fibrotic changes associated with aging process.
  - c. Medications: alcohol, drugs (digitalis toxicity, quinidine, epinephrine)
  - d. May occur acutely in normal hearts or as a chronic arrhythmia with individuals who have chronic heart disease
2. Clinical Significance- : Ineffective atrial contraction with loss of “Atrial Kick” (extra atrial squeeze that provides 25-30% of total cardiac output), that has the potential of causing hemodynamic compromise and possible CHF. Lack of full emptying of the atria allows blood to pool in the chamber predisposing a person to micro emboli formation (thromboembolism).
3. Clinical Features
  - a. Rate: Atrial rate 350->400 bpm unable to discern strong waveforms. Ventricular rate is variable, Rapid Ventricular Response (RVR) is rate > 100 bpm
  - b. Rhythm: irregular
  - c. P Waves No true P waves, rather fibrillatory waves (F Waves) that may be either coarse or fine in varying shapes, amplitude and deflections **There is no discernible waveform with a totally chaotic baseline.**
  - d. QRS: Normal to narrow duration
  - e. PR Interval: none no real P waves
4. Treatment: control the ventricular response rate (80-100 bpm) and possibly convert the rhythm. Provide Oxygen and treat the underlying cause. Mode of therapy will depend on the hemodynamic stability of the patient, always monitor the patients clinical condition. Options include: Valsalva techniques, medications (cardizem, verapamil, digoxin, quinidine, and beta-blockers), synchronized electrical cardioversion, ablation, prophylactic systemic anticoagulation therapy to prevent microemboli formation. Chronic atrial fibrillation = coumadin therapy.



## *Atrial Flutter*

1. Mechanism- Single irritable focus takes over as pacemaker in atrium, producing a circadian depolarization.
  - a. Conduction Impulse- Atria have altered conduction pattern as impulse does not initiate in the SA Node but conduction through the ventricle is normal
  - b. Diseases: **CHF**, Corpulmonale, pulmonary disease, acute MI, hypoxia, cardiomyopathy, pericarditis, thyroid toxicosis (hyperthyroidism), **rheumatic heart disease**, valvular heart disease, emotional distress, post cardiac surgery, SA node disease, pulmonary embolism and fibrotic changes associated with aging process.
  - c. Medications: alcohol, drugs (digitalis toxicity, quinidine, epinephrine)
  - d. May occur acutely in normal hearts or as a chronic arrhythmia with individuals who have chronic heart disease
  
2. Clinical Significance- : Ineffective atrial contraction with loss of “Atrial Kick” (extra atrial squeeze that provides 25-30% of total cardiac output), that has the potential of causing hemodynamic compromise and possible CHF. Lack of full emptying of the atria allows blood to pool in the chamber predisposing a person to micro emboli formation (thromboembolism).
  
3. Clinical Features
  - a. Rate: Atrial rate 250->300 bpm unable to discern strong waveforms. Ventricular rate depends on the AV node and is variable but less than atrial, Rapid Ventricular Response (RVR) is rate > 100 bpm
  - b. Rhythm: regular if flutter wave to QRS ratio is regular, can be irregular
  - c. P Waves Normal P waves are absent and replaced by Flutter (F) Waves a saw tooth or V shaped waveform. T waves obscured by P waves
  - d. QRS: Normal
  - e. PR Interval: cannot measure as there are no real P waves
  
4. Treatment: control the ventricular response rate (80-100 bpm) and possibly convert the rhythm. Provide Oxygen and treat the underlying cause. Mode of therapy will depend on the hemodynamic stability of the patient, always monitor the patients clinical condition. Options include: Valsalva techniques, medications (cardizem, verapamil, digoxin, quinidine, and beta-blockers), synchronized electrical cardioversion, ablation, prophylactic systemic anticoagulation therapy to prevent microemboli formation. Chronic atrial fibrillation = Coumadin Therapy



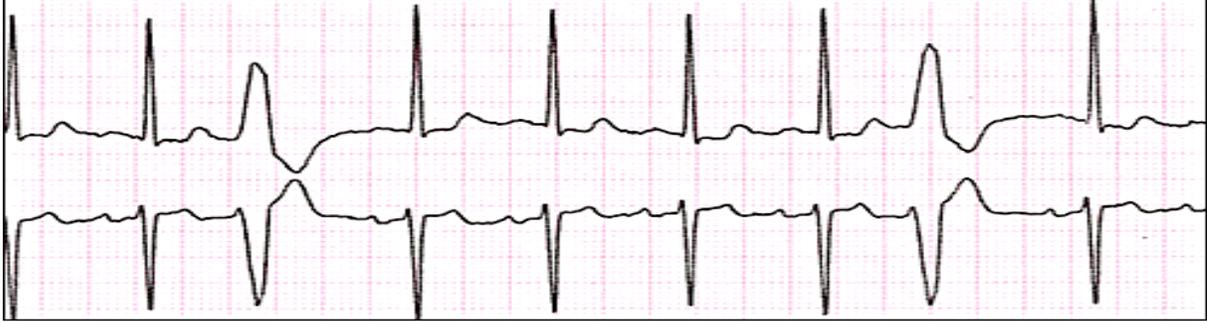
# VENTRICULAR DYSRHYTHMIAS

## *Premature Ventricular Contraction:*

The most common ventricular dysrhythmia is the premature ventricular contraction (PVC). Premature ventricular beats result from the discharge of an ectopic focus within the ventricular walls before the expected arrival of the next impulse from the SA node. These ectopic beats usually represent myocardial irritability.

1. Mechanism:
  - a. Conduction: The discharge from the ectopic focus results in a wide, distorted QRS complex. The SA node is not affected by the PVC and continues to discharge independently and on schedule.
  - b. Diseases: CAD, ischemia, MI, reperfusion, cardiomyopathy, CHF, hypoxia, acidosis, contact of the endocardium with catheters (pacing leads, PA catheter, etc) Electrolyte imbalance (hypokalemia, low magnesium)
  - c. Drugs- digoxin, epinephrine, Isuprel, aminophylline
  - d. Anxiety, stress, exercise, excessive caffeine or alcohol intake
2. Significance:
  - a. Maybe a normal occurrence
  - b. A possible precursor to a lethal arrhythmia especially if it is close to our own the T wave
  - c. A possible indicator of ischemia or electrolyte imbalance
3. Clinical Features:
  - a. Rate is that of the underlying rhythm
  - b. Rhythm: Irregular related to the early beat (PVC), the underlying rhythm may be regular
  - c. P wave seen with underlying rhythm not usually seen with PVC
  - d. QRS Complex of the PVC is WIDE AND BIZARRE
    - 1) QRS is 0.12 seconds or larger
    - 2) Generally has a compensatory pause noted on an ECG following a PVC, precedes the next normal complex
  - e. Types of PVCs
    - 1) Unifocal- one focal point (all look the same)
    - 2) Multifocal or Multifocal- more than one focal point sample (different morphologies)
    - 3) Bigeminal- every other beat is premature
    - 4) Trigeminal- every 3rd beat is premature
    - 5) Couplet- 2 PVC in a successive row
  - f. Symptoms: palpitations, if PVCs are frequent enough, cerebral perfusion may be affected.
4. Treatment:
  - a. Oxygen!!!
  - b. Assess the patient- Are the PVC's perfusing, what are the vitals and level of consciousness
  - c. Drugs only if symptomatic- amiodarone or lidocaine if rhythm is fast, atropine if underlying rate is too slow
  - d. Monitor closely for threatening or lethal arrhythmias

**Unifocal PVC's: identical shapes**  
**Note: A single PVC is labeled isolated**



**Coupled PVC's: occur in pairs**



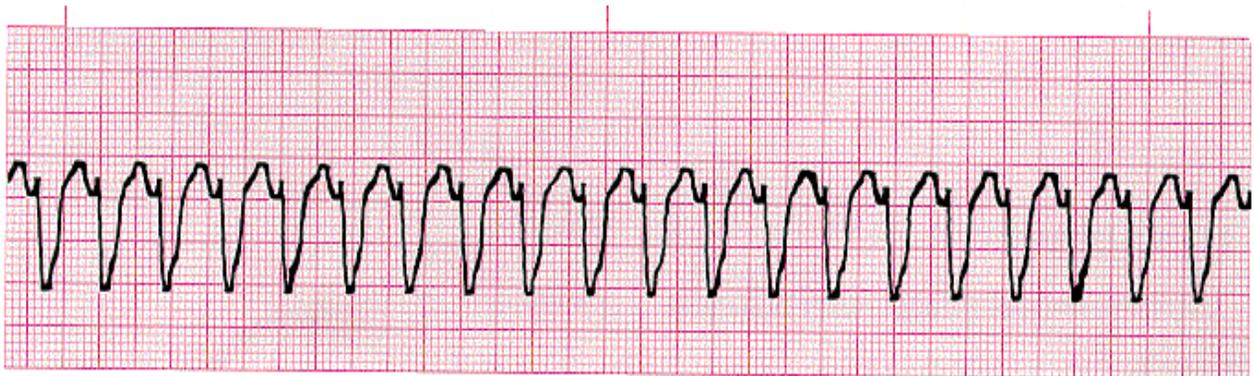
**Bigeminal PVC's: every other beat is a PVC.**



## ***Ventricular Tachycardia:***

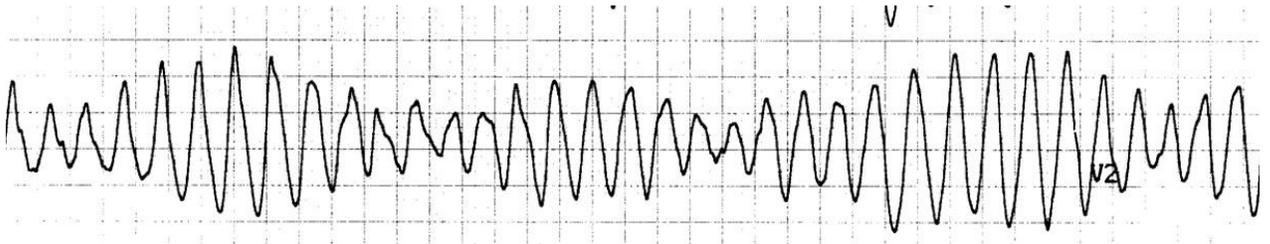
Ventricular tachycardia (VT) can be considered as a series of three or more consecutive PVCs occurring at a rapid rate (usually 140-250 per minute).

1. Mechanism:
  - a. Conduction: The electrophysiologic mechanism of their occurrence is unclear, but evidence exists for both enhanced automaticity and reentry. The occurrence of VT usually reflects marked myocardial irritability. Ventricular Tachycardia may produce a pulse for a short term but if untreated will degenerate to no pulse and increasingly lethal dysrhythmias.
  - b. Disease: CAD, MI, Ventricular aneurysm, hypoxemia and electrolyte imbalance
  - c. Drugs: those that lengthen the QT interval, Digitalis, Quinidine
  - d. Mechanical stimulation, such as pacer wire or balloon angioplasty
2. Significance:
  - a. Life threatening arrhythmia
  - b. Severely diminished cardiac output
  - c. Coronary perfusion is compromised
  - d. Usually a precursor to ventricular fibrillation
3. Clinical Features:
  - a. Rate of 100-250
  - b. Rhythm: Regular
  - c. P Wave: usually cannot see any P wave
  - d. QRS: Wide and Bizarre > 0.12 seconds
  - e. T wave is of opposite polarity of the QRS complex
  - f. Symptoms: dyspnea, palpitations, lightheadedness, angina, hypotension
4. Treatment:
  - a. Check on the patient immediately!!! If no pulse start CPR, call code and get cart
  - b. If pulse Call physician immediately!!!
  - c. If no pulse- defibrillate as soon as the cart/AED is available If pulse prepare for Cardioversion
  - d. Drugs: amiodarone, lidocaine, if no pulse- epinephrine
  - e. Cardioversion
5. Examples:



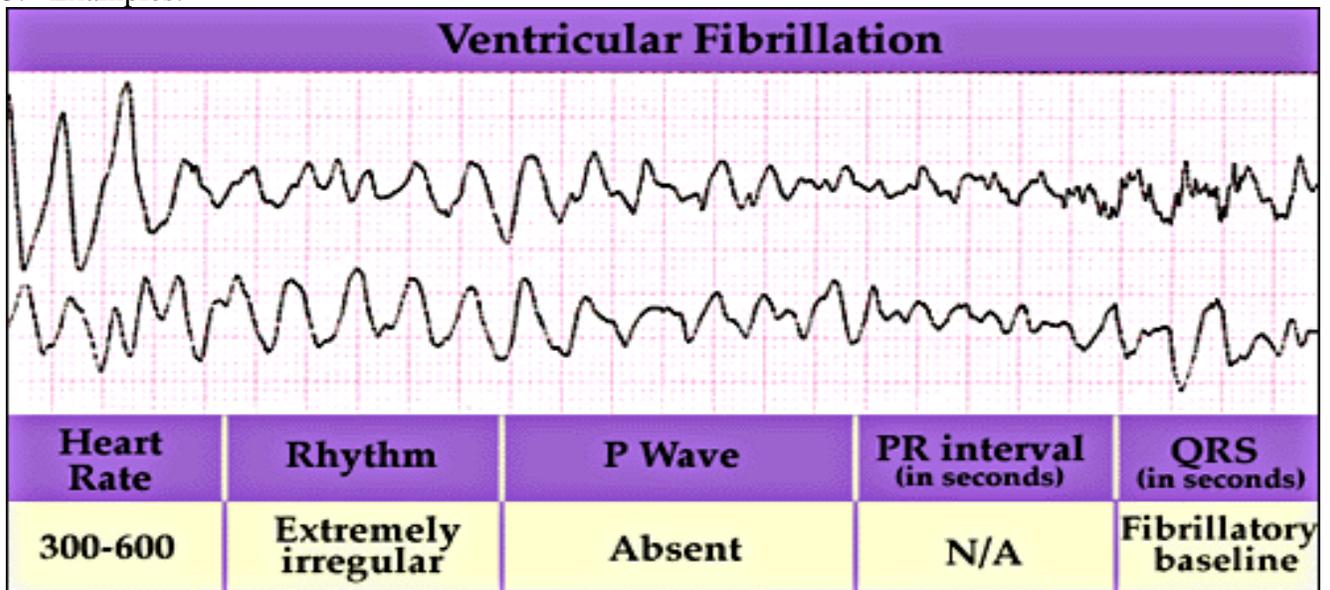
# *Torsade de Pointes (“Twisting of the Points”)*

1. Mechanism:
  - a. Conduction: initiated by a PVC striking near the apex of a delayed T wave (pts often have long QT intervals) Ventricular response is irregular with rates of 200-250/min with marked variability in amplitude and direction of a QRS wave that seems to twist around an isoelectric baseline
  - b. Diseases: myocarditis, ischemic heart disease, variant pathways
  - c. Drugs: those that prolong the QT interval, such as procainamide, tricyclic antidepressants, and quinidine, Hypokalemia, hypomagnesemia, hypocalcemia,
2. Significance:
  - a. May develop into Ventricular Standstill (asystole)
  - b. May develop into Ventricular Fibrillation
3. Clinical Features
  - a. Peaks of ventricular complexes alternate polarity, which gives a twisting appearance
  - b. Rate of 200-400 beats/minute
  - c. Symptoms: dizziness, syncope, sudden death
4. Treatment:
  - a. Discontinue offending drug (drug that is suspected that caused rhythm)
  - b. IV Magnesium (1-2 grams over 5 minutes, continue with a IV infusion)
  - c. Defibrillation if underlying rhythm is normal or Overdrive pacing if underlying rhythm is bradycardic



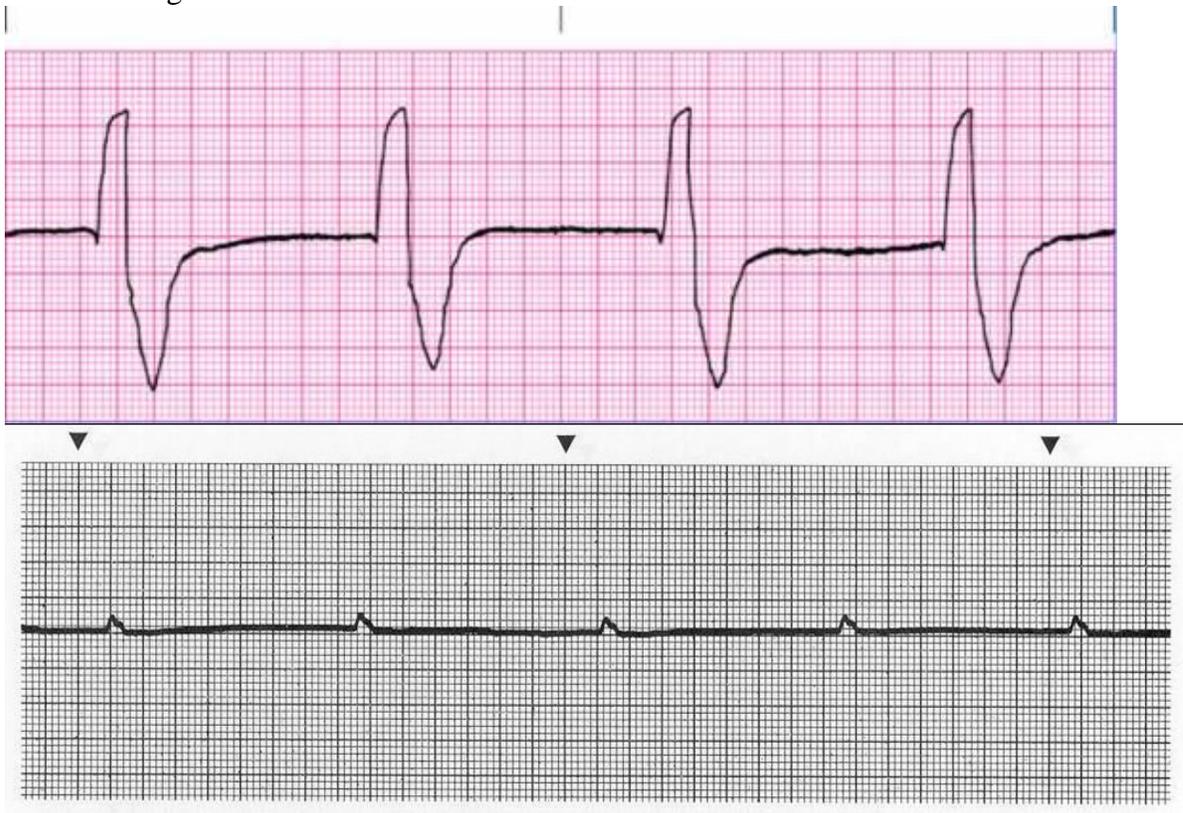
# Ventricular Fibrillation

1. Mechanism:
  - a. Conduction: Ventricular fibrillation is erratic, chaotic ventricular electrical activity that results from multiple foci stimulating the ventricles leading to a lack of coordinated ventricular activity. Muscle fibers twitch rather than contract effectively to pump blood.
  - b. Diseases: CAD, ischemia, MI, cardiomyopathy hypokalemia, hypomagnesemia
  - c. Drugs, such as catecholamine excess
  - d. Mechanical irritation, such as pacer wire and Swan-Ganz catheter
  - e. Cardioversion, and many other causes
2. Significance
  - a. Most common cause of “Sudden Cardiac Death”
  - b. No cardiac output
  - c. Requires immediate intervention, life threatening
3. Clinical Features:
  - a. Rate: a rapid fibrillating (quivering) of the ventricles
  - b. Rhythm is an erratic pattern
  - c. P wave: none
  - d. QRS Complex none
  - e. Circulatory collapse
4. Treatment
  - a. Rapid Defibrillation
  - b. CPR
  - c. Drugs: vasopressin, epinephrine, lidocaine, amiodarone
5. Examples:



## ***Idioventricular Rhythm-- Ventricular Escape Rhythm:***

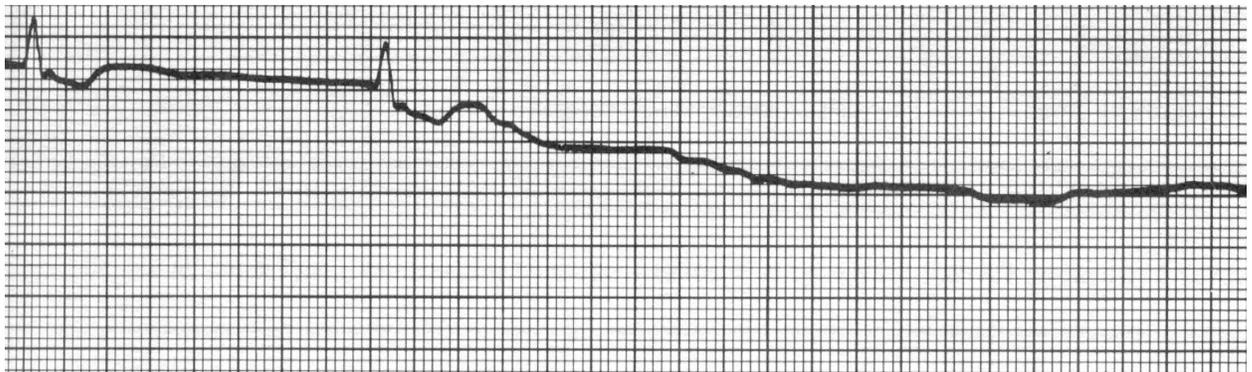
1. Mechanism:
  - a. Conduction: the ventricles initiate impulses on their own, without stimulation from a higher center and is characterized by a series of consecutive ventricular beats
  - b. Disease: sinus arrest, Heart block, MI, and Electrolyte imbalance
  - c. Drugs: digitalis, quinidine, inderal
2. Significance:
  - a. Represents failure of the Sinus Node and AV Junction;
  - b. Rate slows down to less than the inherent rate of the ventricles (20-40)
  - c. Cardiac output is diminished due to the decreased rate
  - d. May develop into asystole
3. Clinical Features:
  - a. Rate: 20-40 Occasionally this rate may increase to 50-100 beats per minute representing an accelerated idioventricular rhythm.
  - b. Rhythm: Regular
  - c. P waves: absent
  - d. QRS Complex is wide, greater than 0.10 seconds
  - e. Symptoms: hypotension, decreased cerebral perfusion
4. Treatment:
  - b. Atropine, Dopamine, or Epinephrine
  - c. Pacing



## ***Ventricular Standstill (Asystole):***

Ventricular contraction depends on an effective electrical stimulus. If for some reason In ventricular asystole,. When ventricular stimulation ceases, unconsciousness develops immediately and death occurs with a very brief period unless CPR can restore effective ventricular action.

1. Mechanism:
  - a. Conduction: electrical stimuli to the ventricles are of inadequate intensity, or if they cease entirely, the ventricles stop contracting. Sinus impulses may discharge normally and produce P waves. However, all of these impulses are blocked and none reach the ventricles. Because an inherent pacemaker does not come to the rescue, ventricular activation stops and all QRS complexes disappear on the ECG
  - b. Disease: CAD, cardiomyopathy, conduction disorders (usually involving two or three of the fascicles of the bundle branches)
  - c. 6 Hs: hypoxia, hypovolemia, hypothermia, hyper/hypokalemia, hydrogen ion (acidosis), hypoglycemia
  - d. 5 Ts: tamponade, tension pneumothorax, thrombosis (coronary), thrombosis (pulmonary), “tablets” (drug overdose)
  - e. Often preceded by VT or VF
2. Significance:
  - a. No Cardiac Output
  - b. Respirations cease shortly after onset
3. Clinical Features:
  - a. No Rate
  - b. May be an occasional P wave, but has no effect
  - c. No QRS Complex
  - d. Flat or Wavy Line
4. Treatment:
  - a. CPR
  - b. Drugs: Epinephrine, Atropine
  - c. Pacing
  - d. Treat H’s and T’s above



## *Pulseless Electrical Activity*

**\*\*Hallmark\*\***

This is a condition not necessarily a rhythm. It can be any rhythm that should produce a pulse for instance Bradycardia, Normal Sinus Rhythm, or Sinus Tachycardia. The hallmark of this condition is the **unexpected** finding of **NO PULSE**.

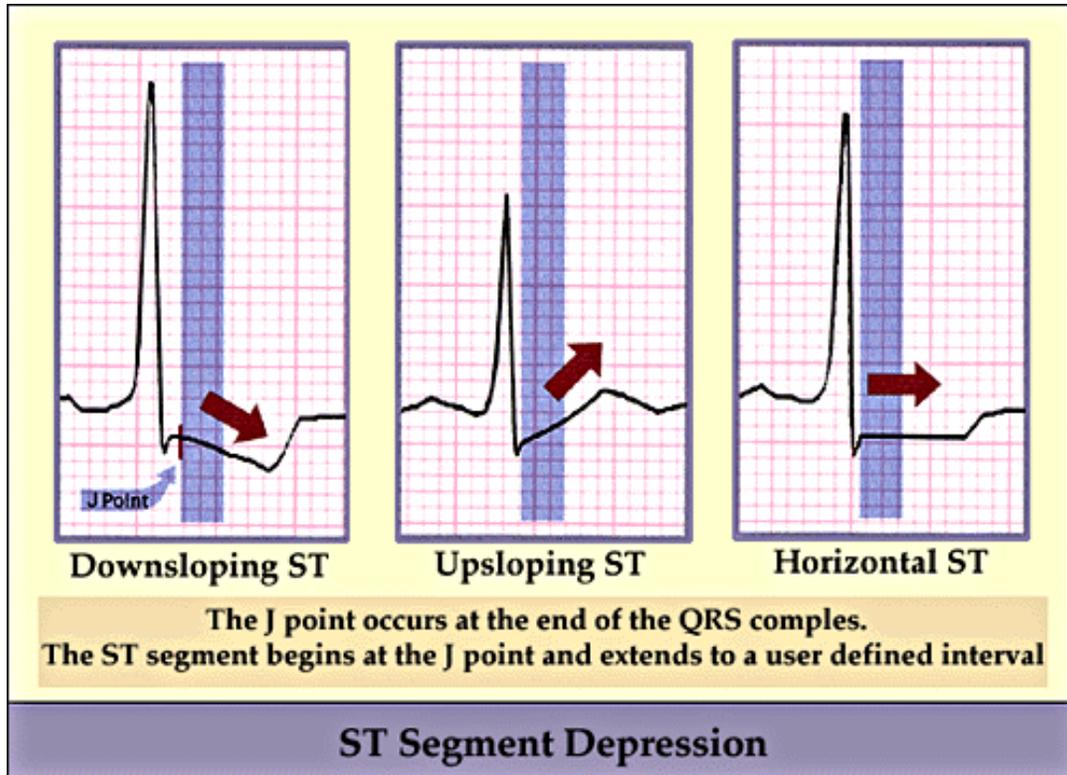


Normal Sinus Rhythm: Assess patient to see if there is a pulse: No pulse activate code Blue!!

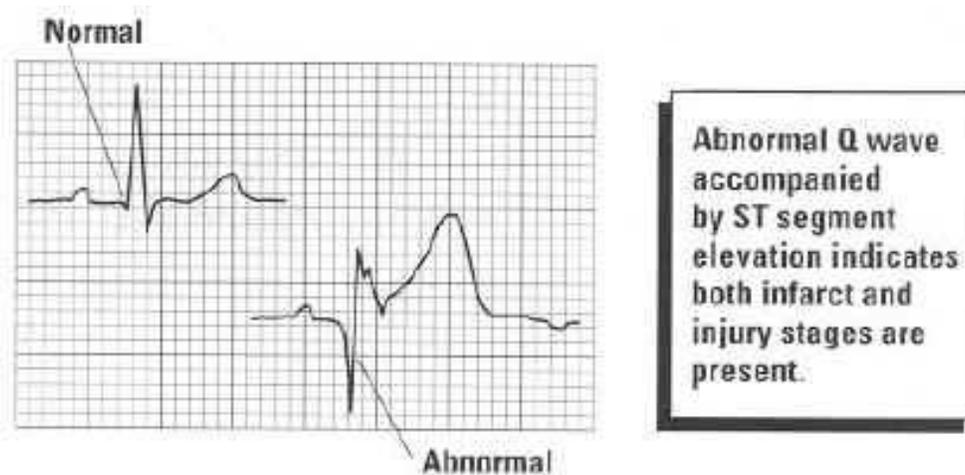
1. Mechanism:
  - a. Conduction: electrical rhythm without mechanical activity. It is the absence of a detectable pulse and blood pressure in the presence of electrical activity.
  - b. 6 Hs: hypoxia, hypovolemia, hypothermia, hyper/hypokalemia, hydrogen ion (acidosis), hypoglycemia
  - c. 5 Ts: tamponade, tension pneumothorax, thrombosis (coronary), thrombosis (pulmonary), “tablets” (drug overdose)
2. Clinical Significance: No Cardiac Output Patient is dead without cardiac output despite what is seen on the monitor The effect is comparable to asystole.
3. Clinical Features:
  - a. Rate: varies with rhythm but pulse is always absent
  - b. Waveforms will be normal for rhythm on the monitor Rhythm
  - c. UNEXPECTED finding of no pulse
4. Treatment:
  - a. CPR
  - b. Treat H’s and T’s above
  - c. Drugs: Epinephrine, Atropine
  - d. Pacing

# Identification of a Myocardial Ischemia/Infarction

- I. **Myocardial Ischemia**- Lack of oxygen to the heart tissue. Evidenced by **ST segment depression**.

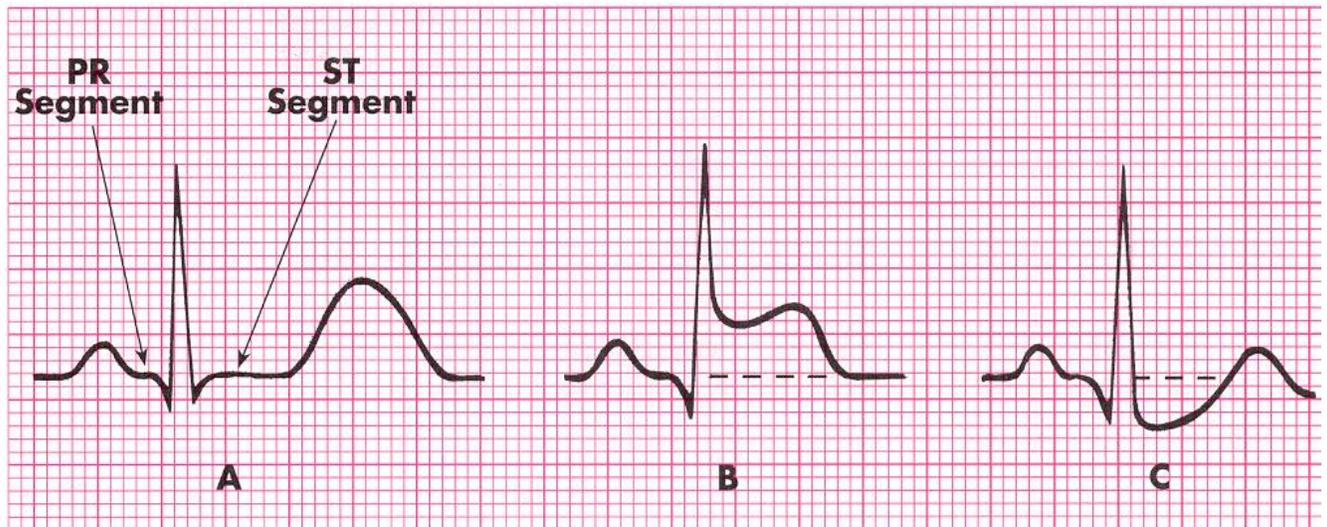


- II. **Myocardial Injury or Infarct**: Lack of oxygen to the heart, untreated can result in injury or death of the myocardial tissue (infarct). Evidenced on the EKG by **ST segment elevation**.



### III. Location of Ischemia, Injury or Infarct in Relationship to the EKG.

- A. Septal Wall ischemia, injury or infarct- changes will be noted in V1 & V2
- B. Anterior Wall ischemia, injury or infarct- changes in V3 & V4
- C. Lateral Wall ischemia, injury or infarct- changes in Lead I, aVL, V5, & V6
- D. Inferior Wall ischemia, injury or infarct- changes Leads II, III, & aVF



- A. Shows Normal Baseline ST Segment
- B. Shows ST Segment Elevation (Infarct or Injury)
- C. Shows ST Segment Depression (Ischemia)

Respond to ST Segment Changes Immediately!!!

- ◆ Assess patient
- ◆ Apply O<sub>2</sub> (Nasal Canual or Non-Rebreather Mask)
- ◆ Notify doctor
- ◆ 12 lead ECG
- ◆ All these steps occur simultaneously (use team approach)

ST Segment Alarm System

- ◆ Alarm default +/-2mm from baseline
- ◆ Alarm triggers if ST value exceeds limit longer than 1 min

## Pace Makers and Identifying them on a ECG

### Pacing Methods:

- Transcutaneous (Transthoracic)- pace through the skin and musculature-i.e. Pacemaker found on the defibrillator
- Transvenous- pacer wires are placed through the central line to stimulate the heart directly on the muscle
- Permanent- control device and wires are implanted into the chest

### Pacer Modes:

- **Asynchronous pacing (Fixed)**- pacer releases a stimulus at a fixed rate. Does not alter based on patient intrinsic beats. Rarely used unless there is no intrinsic rhythm
- **Demand Pacing**- pacer is able to recognize (sense) the hearts natural rhythm & inhibit its output when the heart beats faster than the basic rate of the pacer

### Pacer Terminology:

- **Pacer Spike**- small vertical line, “blip”, whenever a pacing stimulus enters the heart
- **Capture**- ability of pacing stimulus to result in depolarization of the chamber being paced
- **Sensing**- ability of the pacer to recognize spontaneous cardiac depolarization
- **Pacemaker’s Refractory Period**- time following paced/sensed event when the pacer “closes its eyes” & can’t see spontaneous cardiac activity

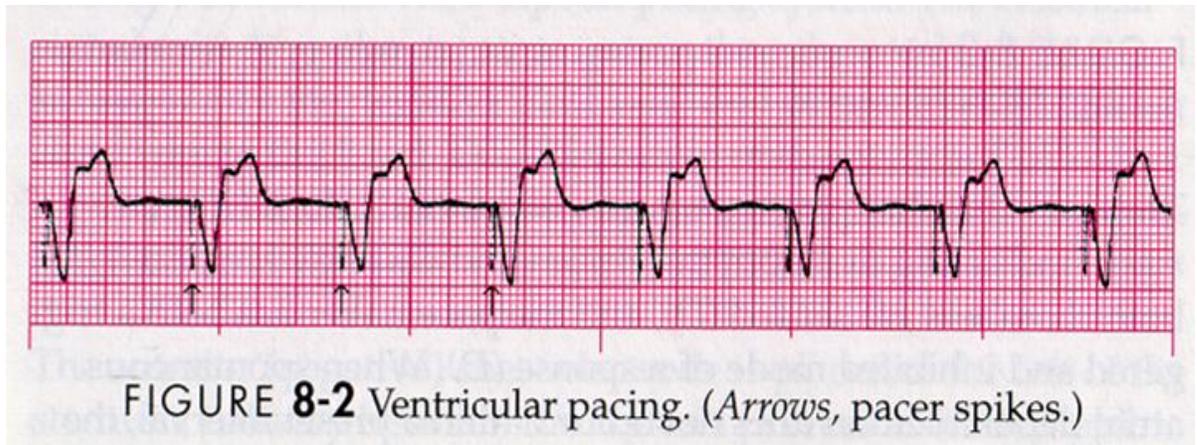
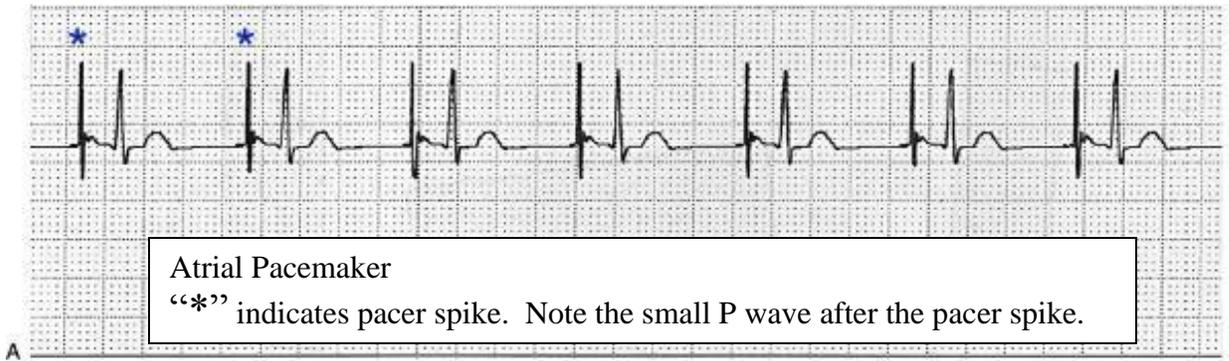


Pacer Spikes

“Capture” Notice pacer spike with a widened QRS Complex. This is a demand pacemaker, meaning it only fires when the Hearts Intrinsic Factor from the SA to AV Node connects and exceeds the set pacer rate.

### Types of permanent/transvenous pacers (see below for examples)

- Atrial- pacer wires are stimulating the atria- See a pacer spike then a P wave
- Ventricular- pacer wires are stimulating the ventricles- See a pacer spike then a wide QRS
- A-V sequential (Dual Chamber)- combination Atrial and ventricle. The pacer stimulates the atria then ventricle. Most resembles the normal electrical system.

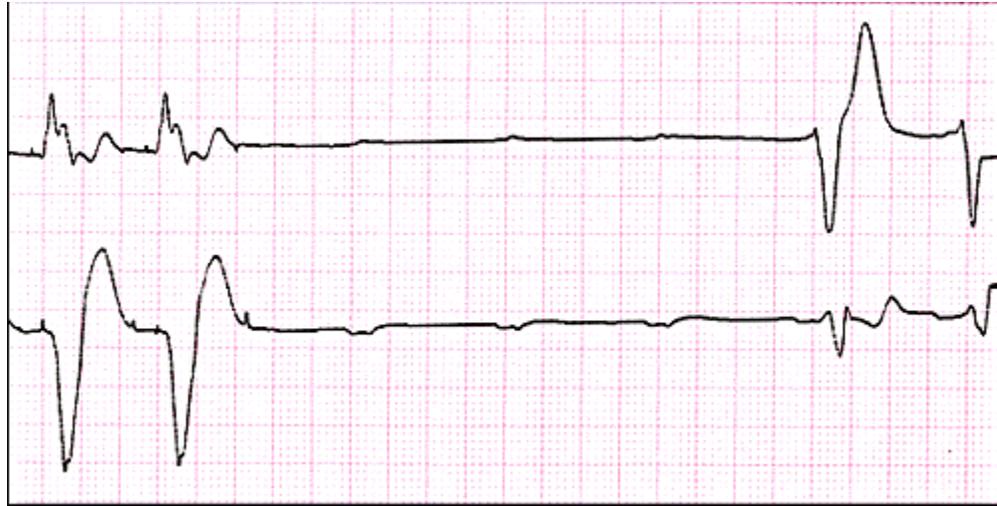


Example of dual chamber pacemaker: Notice pacer spike before each P and QRS complex



## Pacemaker Malfunctions

- **Failure to Pace (Fire):** The pacemaker is not firing when the rate decreases to below the set parameter. If the patient has no intrinsic rhythm they will experience hemodynamic compromise and possible collapse.



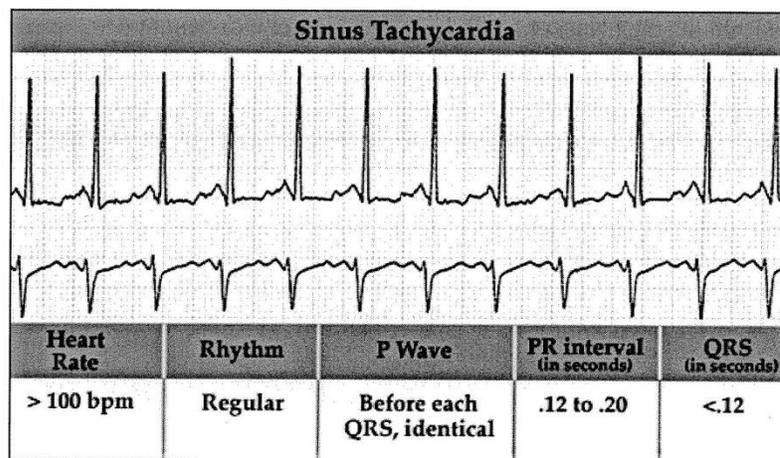
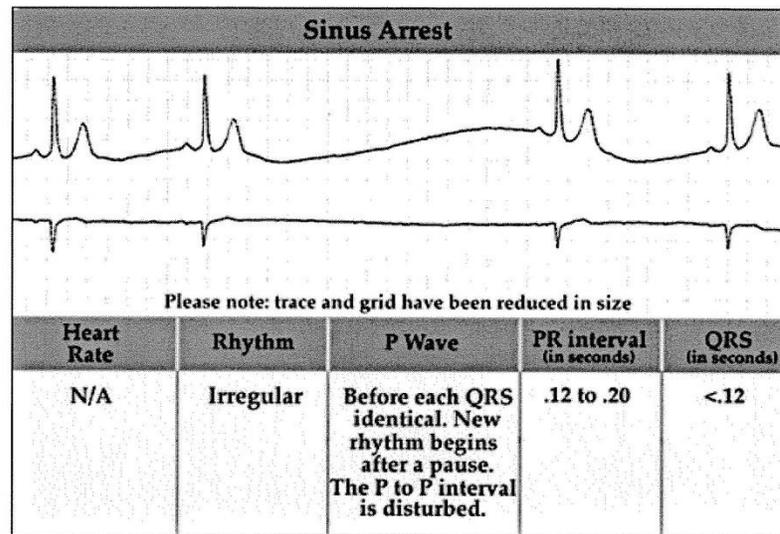
- **Failure to Capture** A pacer spike not followed by a QRS Complex. The patient is not responding to the stimulation



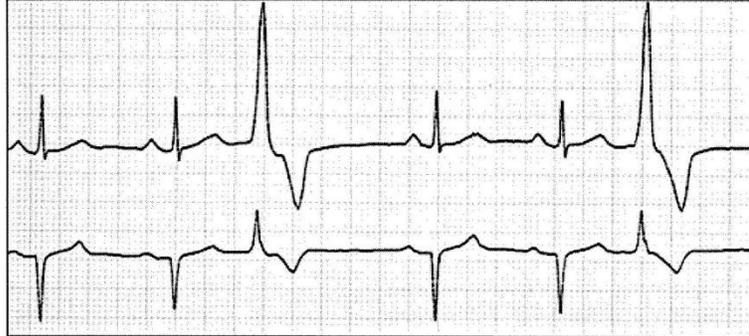
- **Failure to Sense** The pacemaker is not sensing the patient's intrinsic rhythm as competing for electrical control of the heart. If the pacer spikes hits at the vulnerable last half of the T wave (R on T phenomenon) the patient can develop Ventricular Tachycardia.



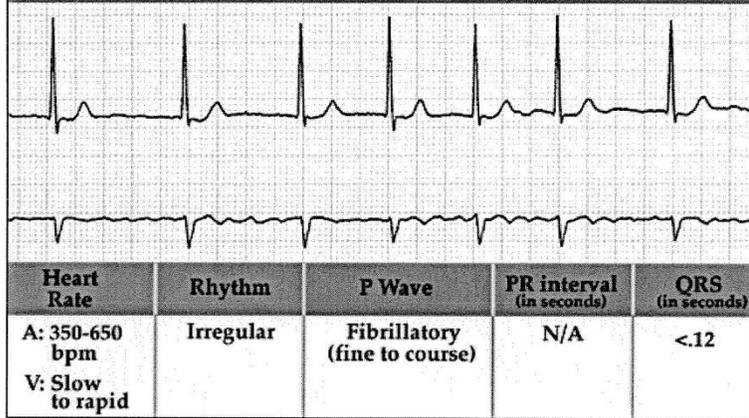
Other Examples:

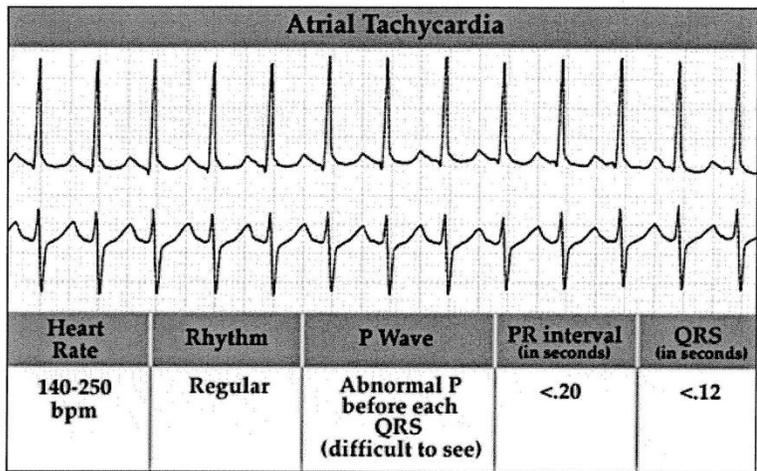
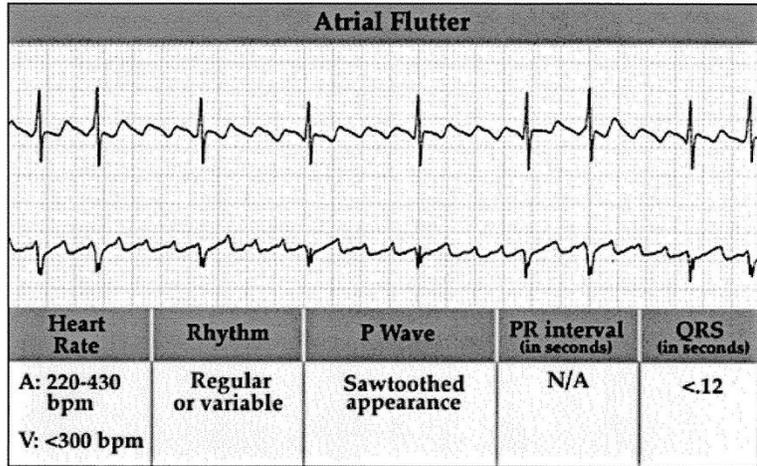


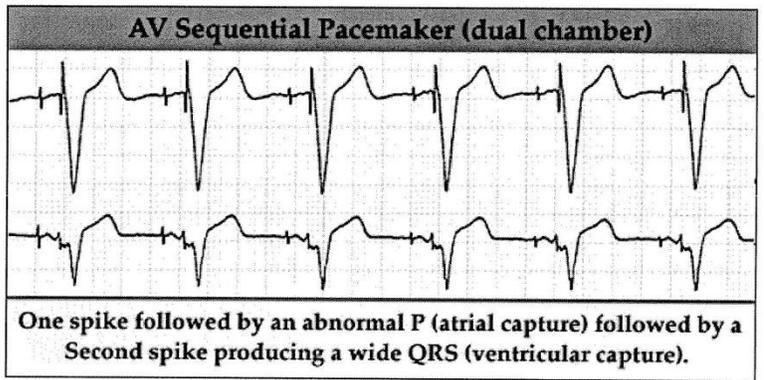
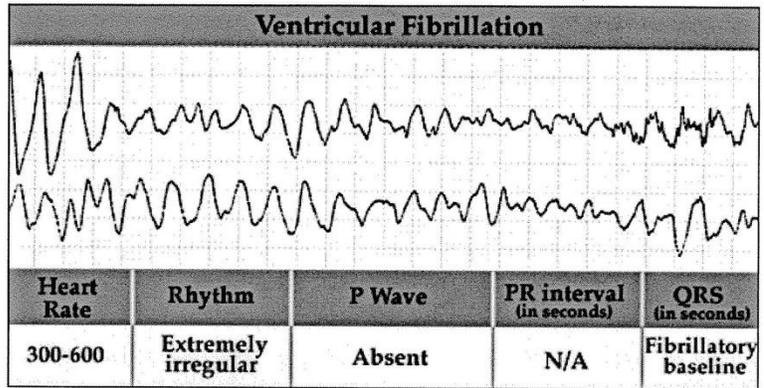
**Trigeminal PVC's: every third beat is a PVC**

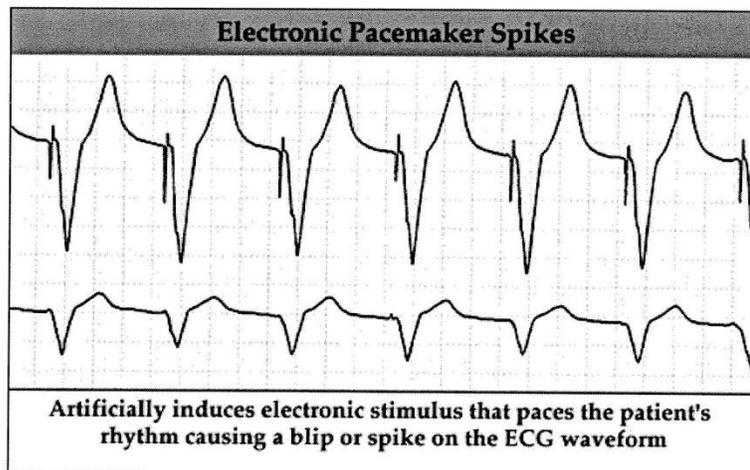
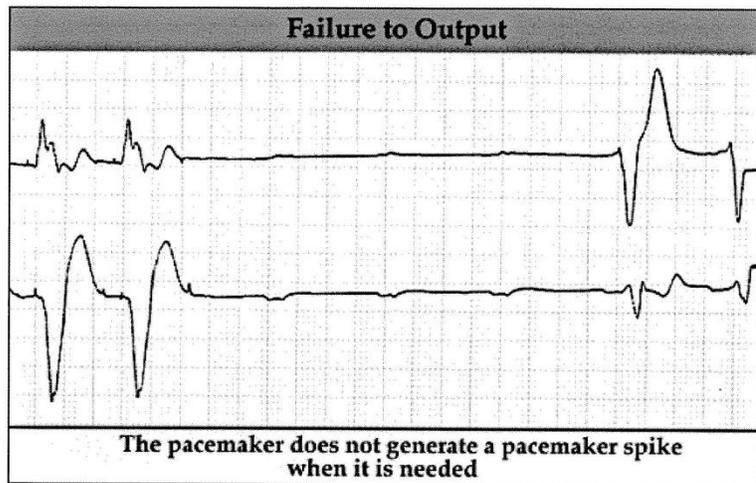


**Atrial Fibrillation**

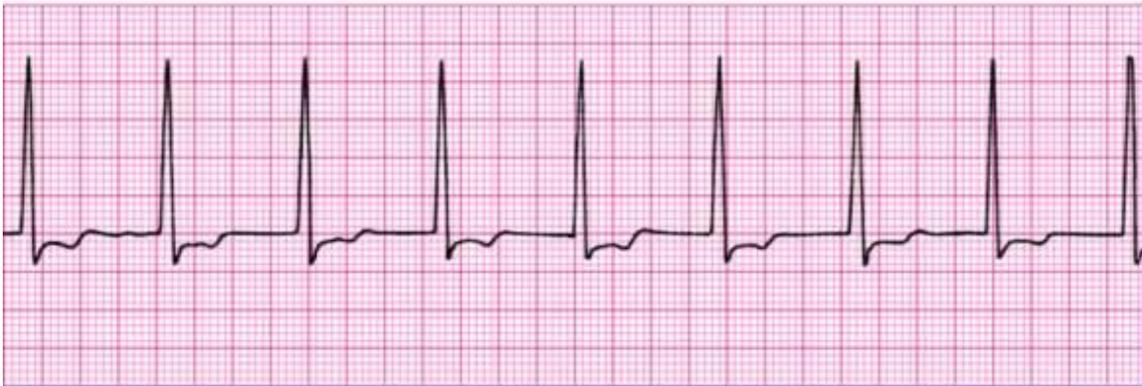








Accelerated Junctional Rhythm: Notice lack of P waves but normal QRS with increased rate



Junctional Escape Rhythm: Lack of P wave at a normal rate



Normal Sinus Rhythm with 1 Premature Junctional Contraction (PJC)

