ECG

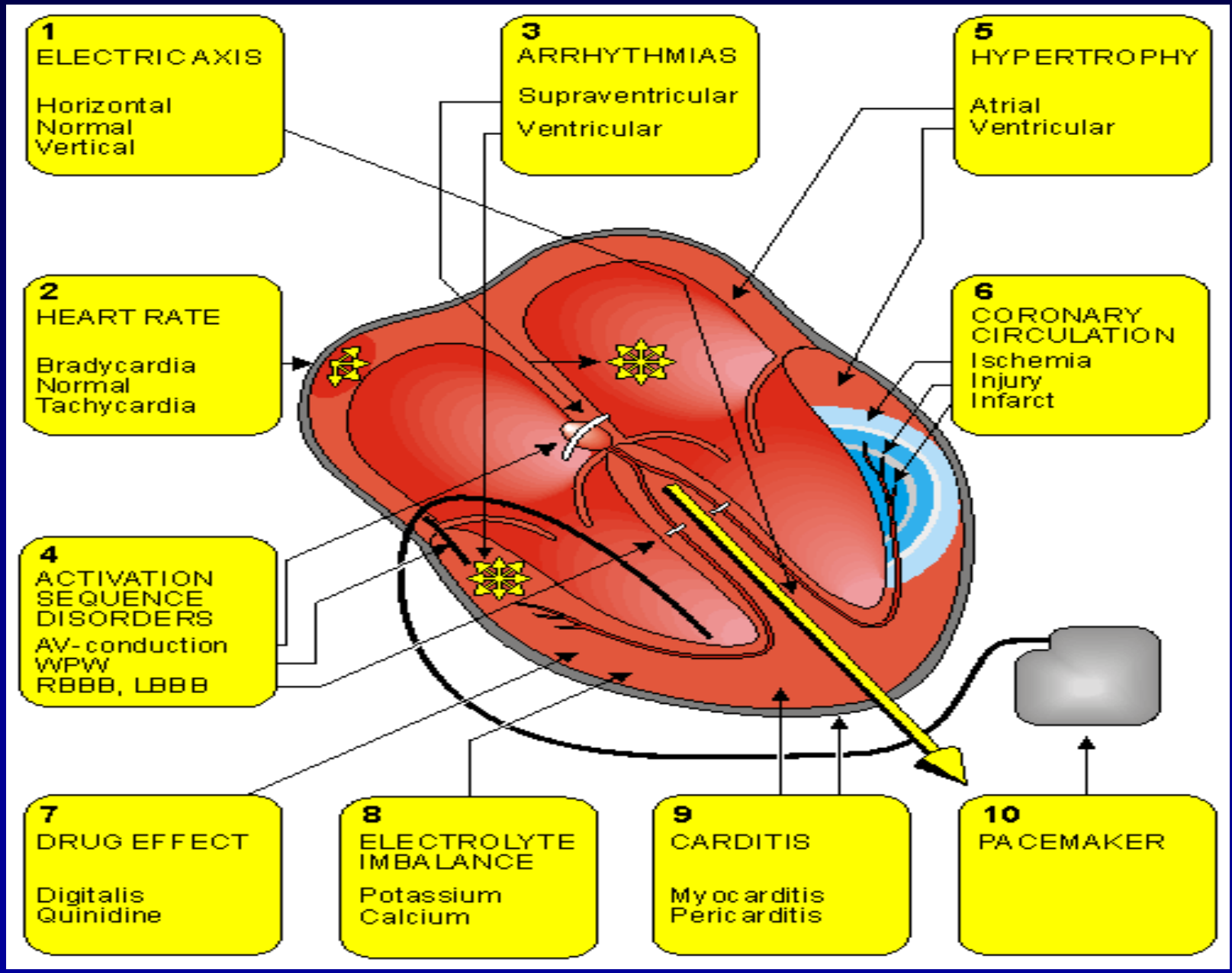
BASICS

ECG

- The ECG records the electrical signal of the heart as the muscle cells depolarize (contract) and repolarize.

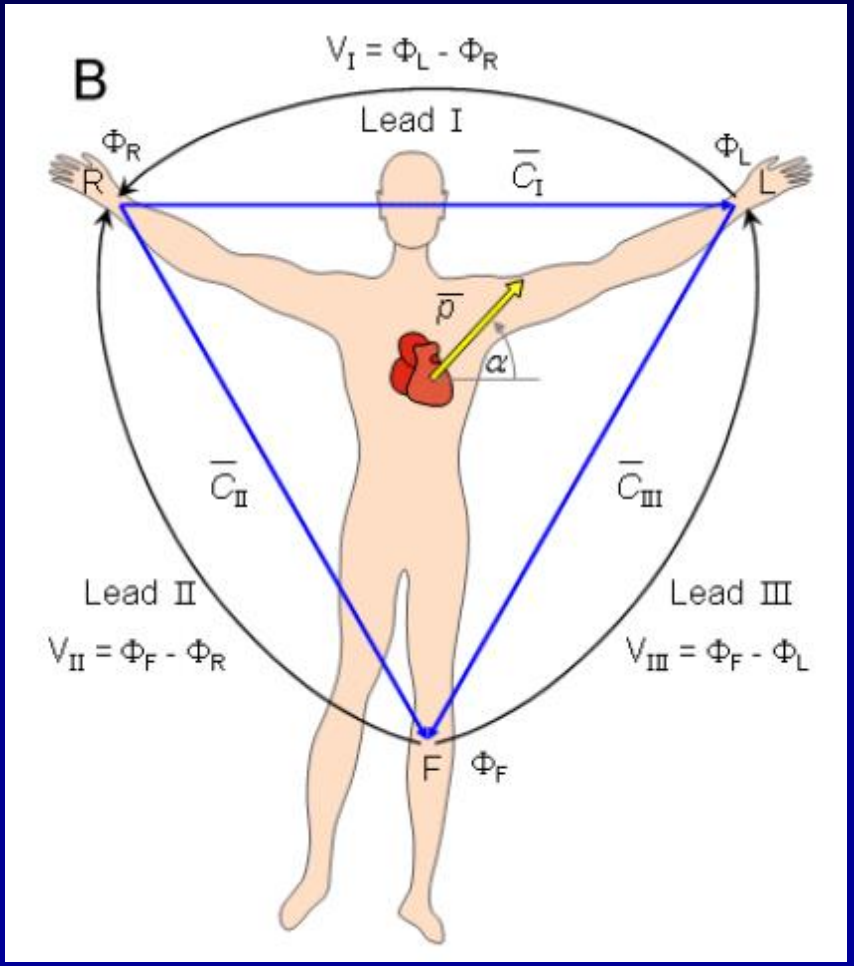
- Normally, the SA Node generates the initial electrical impulse and begins the cascade of events that results in a heart beat.

ECG Diagnosis



ECG Leads

- In 1908, Willem Einthoven developed a system capable of recording these small signals and recorded the first ECG.
- The leads were based on the Einthoven triangle associated with the limb leads.
- Leads put heart in the middle of a triangle

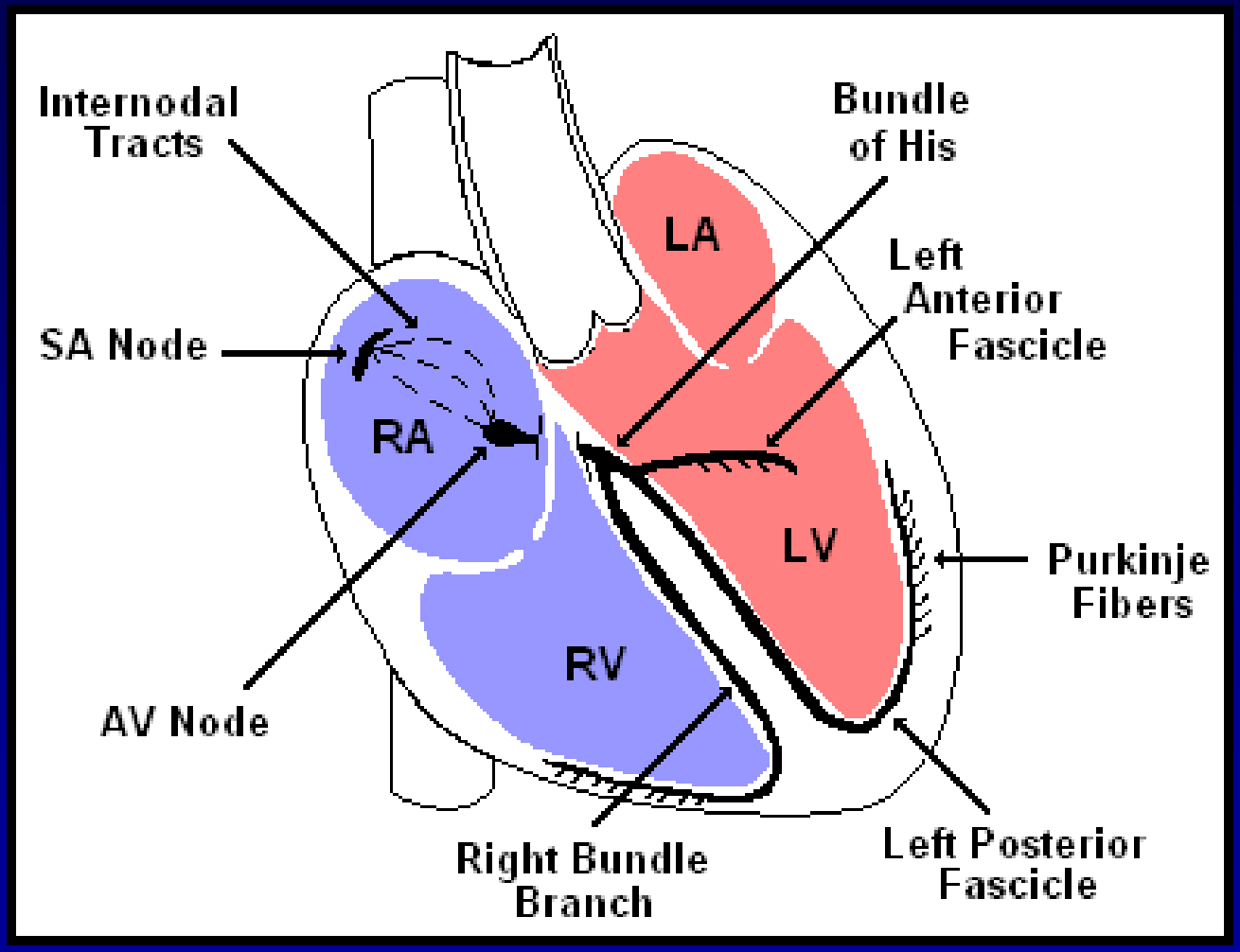


Rules of ECG

- Wave of depolarization traveling towards a positive electrode causes an upward deflection on the ECG

- Wave of depolarization traveling away from a positive electrode causes a downward deflection on the ECG

The Normal Conduction System



ORIENTATION OF THE 12 LEAD ECG

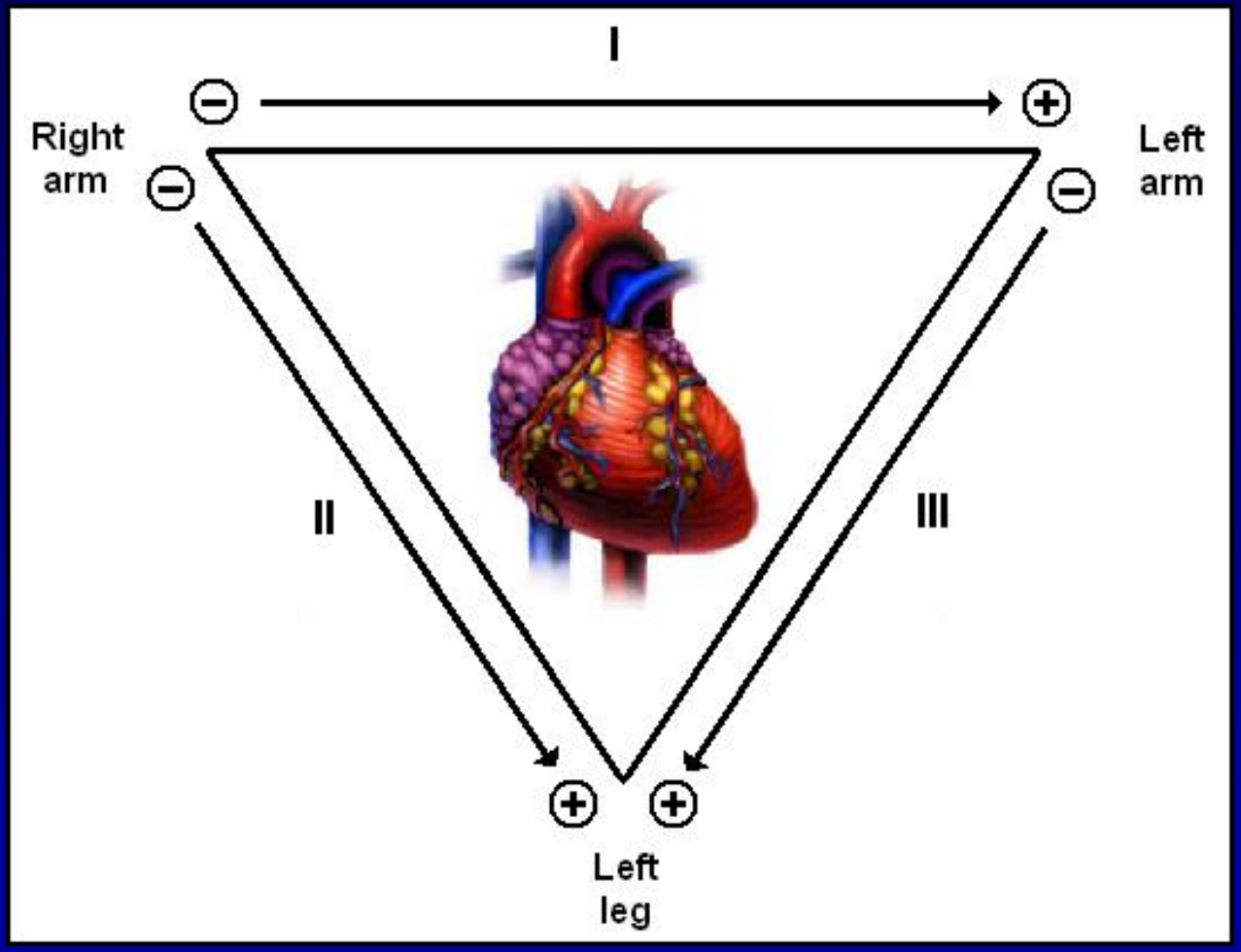
EKG Leads

The standard EKG has 12 leads:

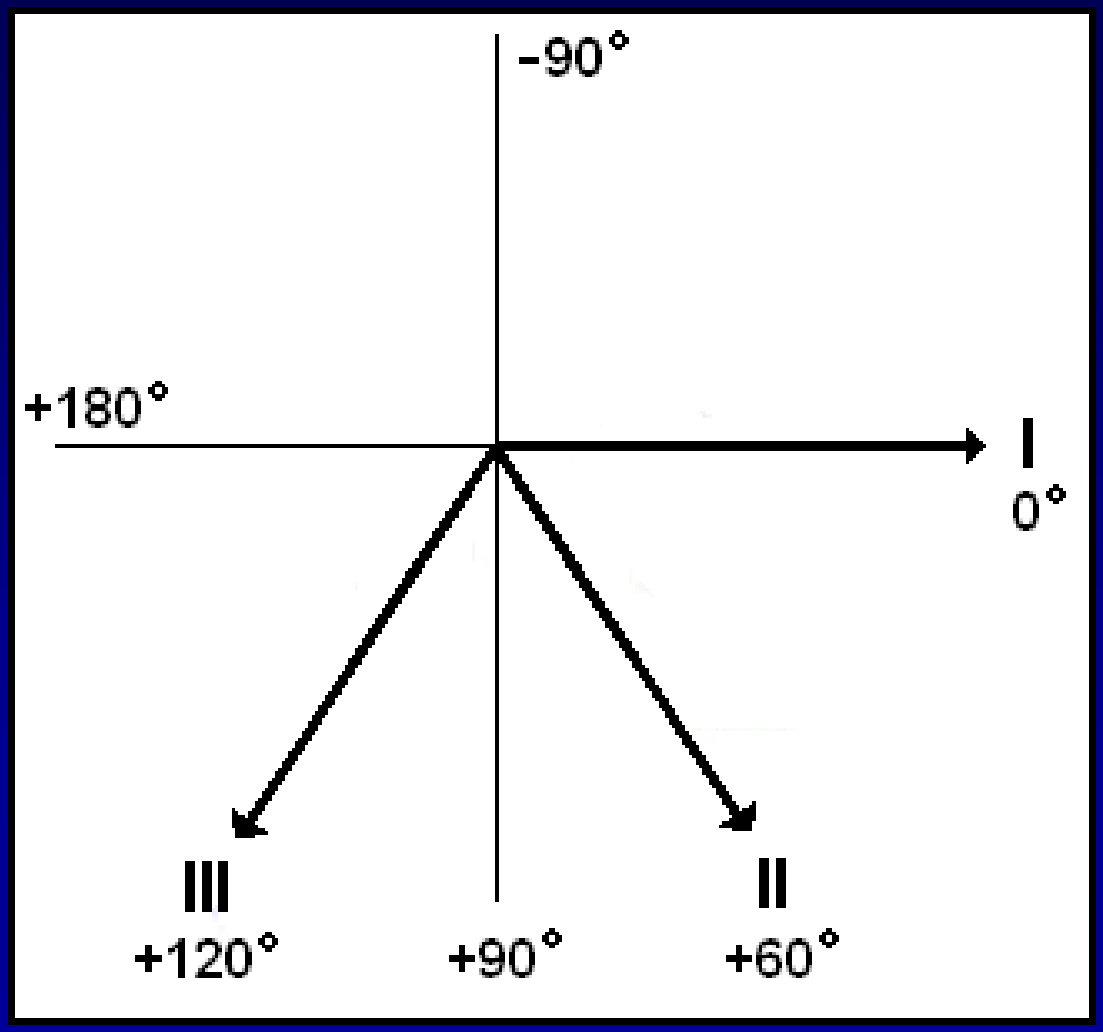
- 3 Standard Limb Leads
- 3 Augmented Limb Leads
- 6 Precordial Leads

The axis of a particular lead represents the viewpoint from which it looks at the heart.

Standard Limb Leads

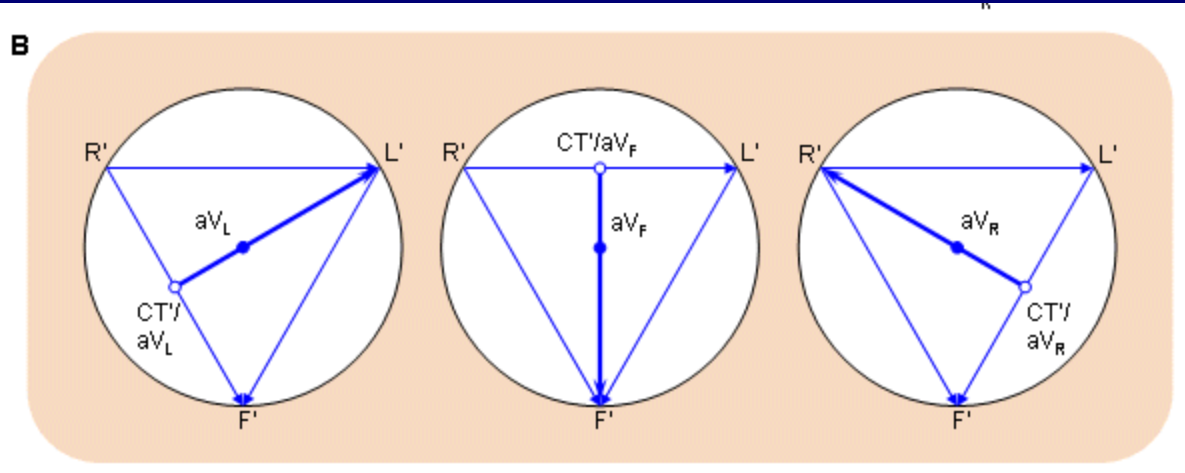


Standard Limb Leads

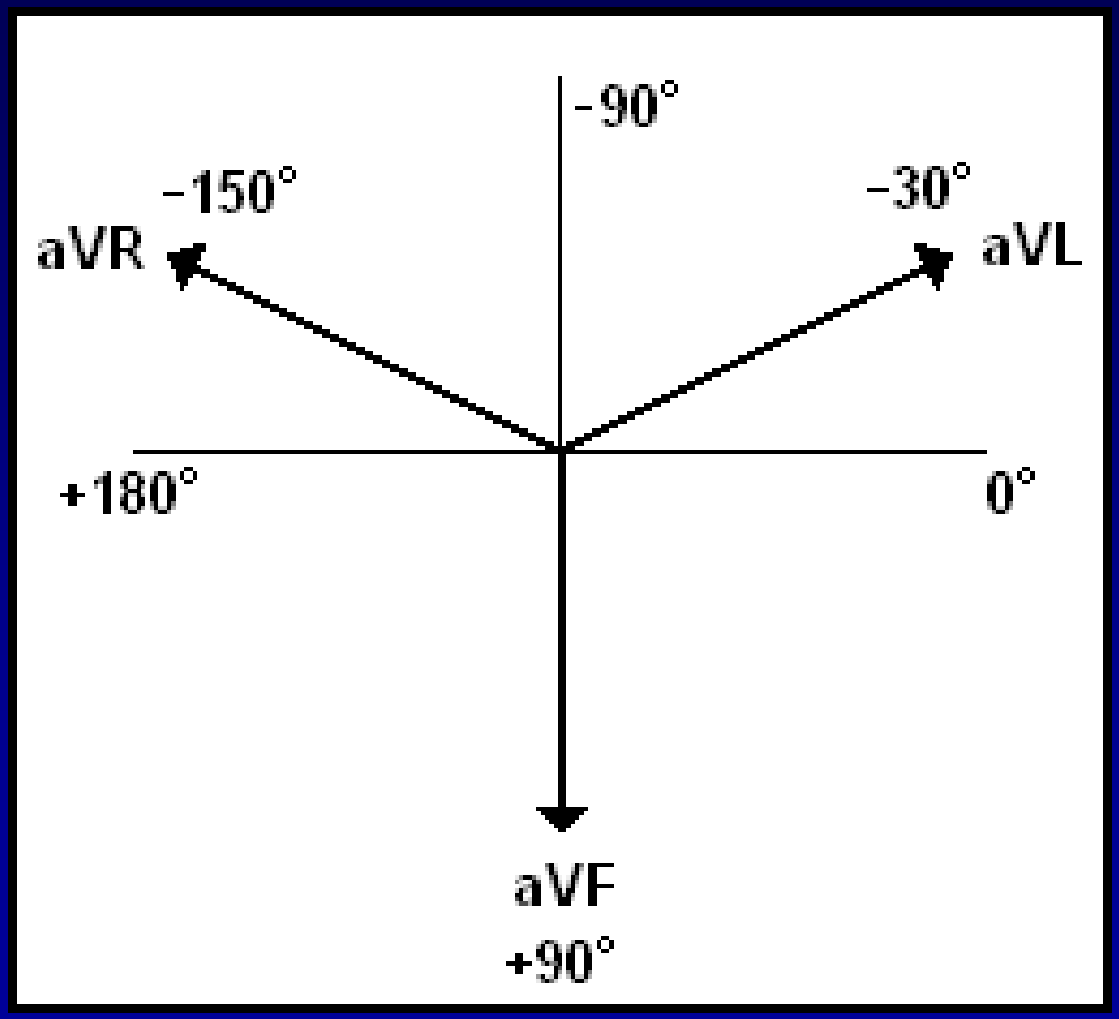


Augmented Leads

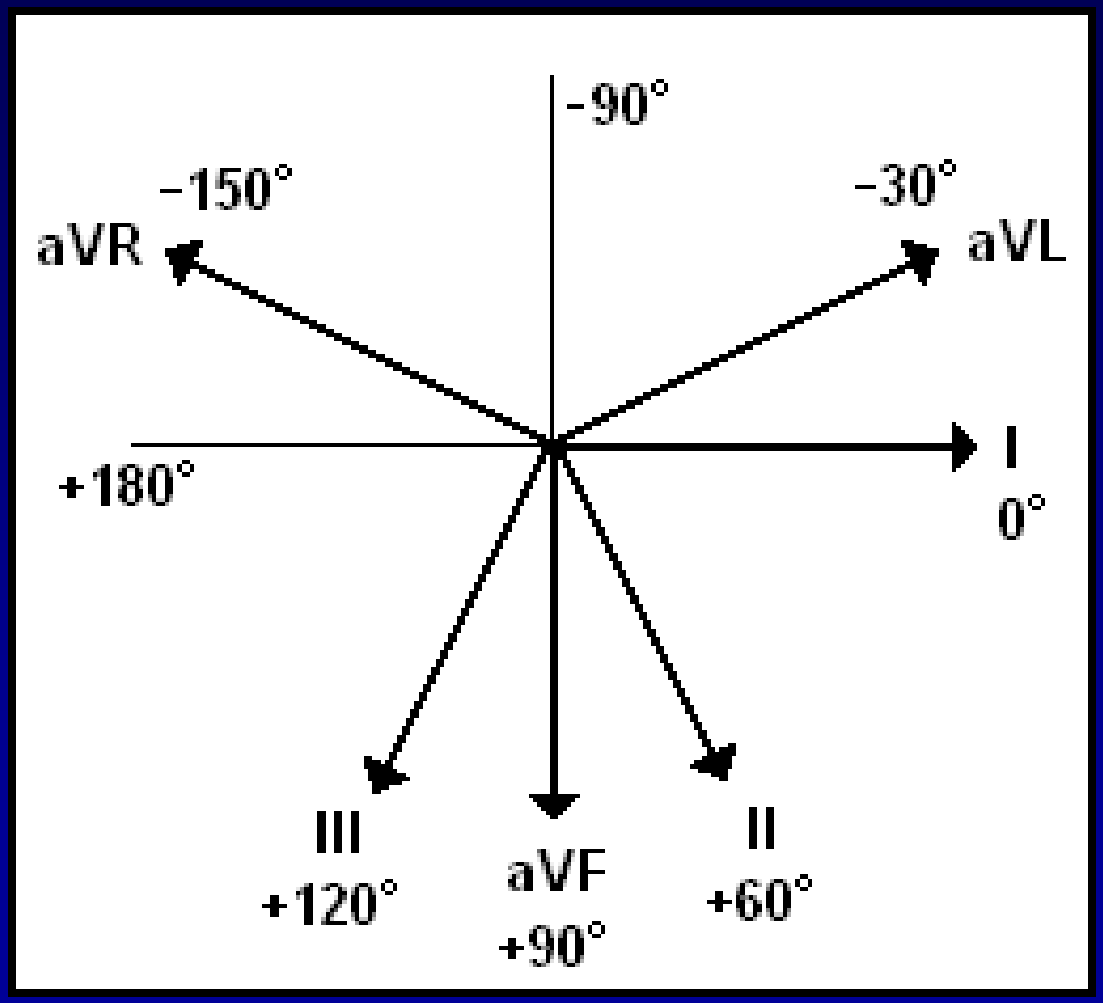
- Three additional limb leads are also used: aV_R, aV_L, and aV_F
- These are unipolar leads



Augmented Limb Leads

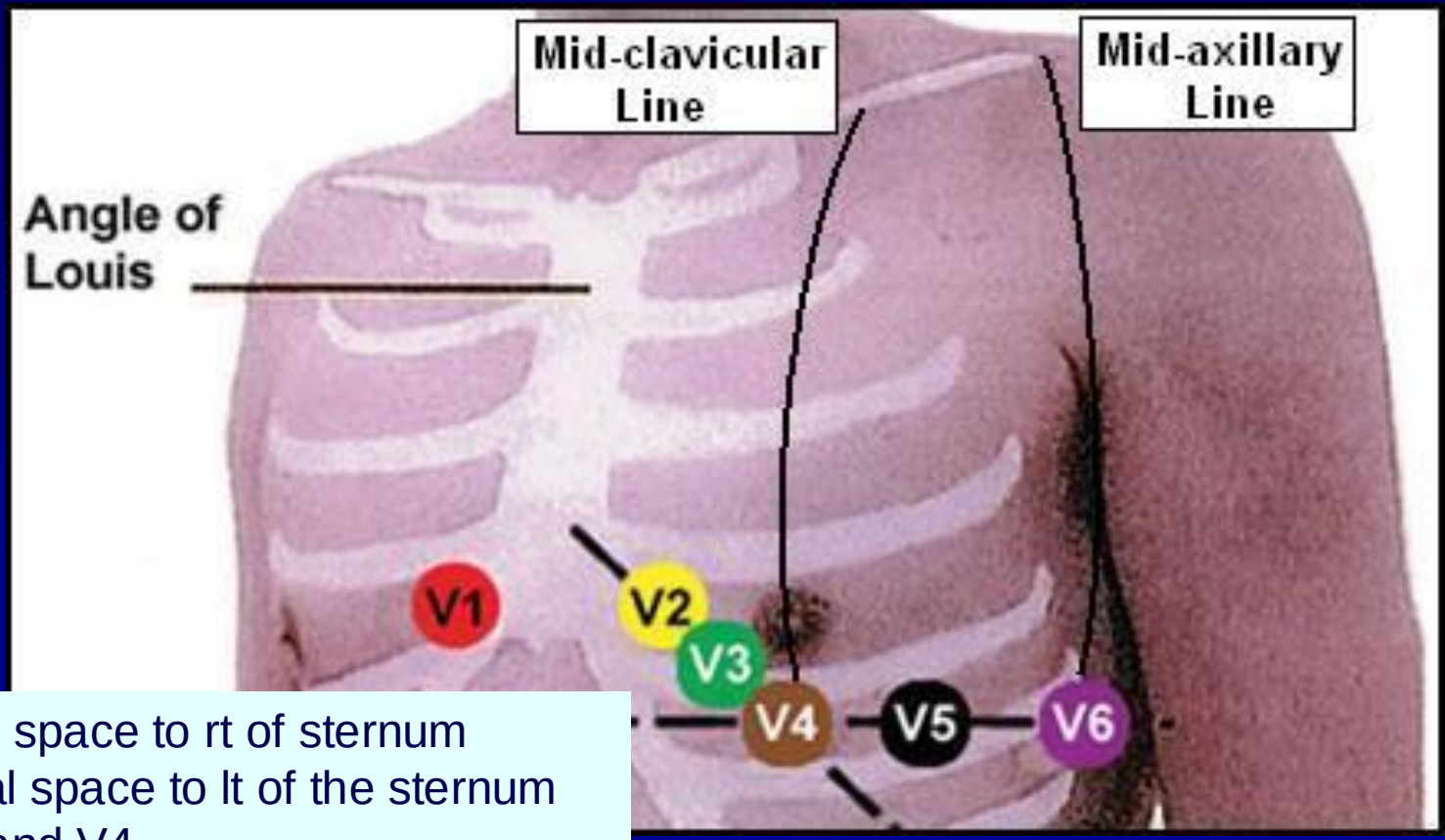


All Limb Leads



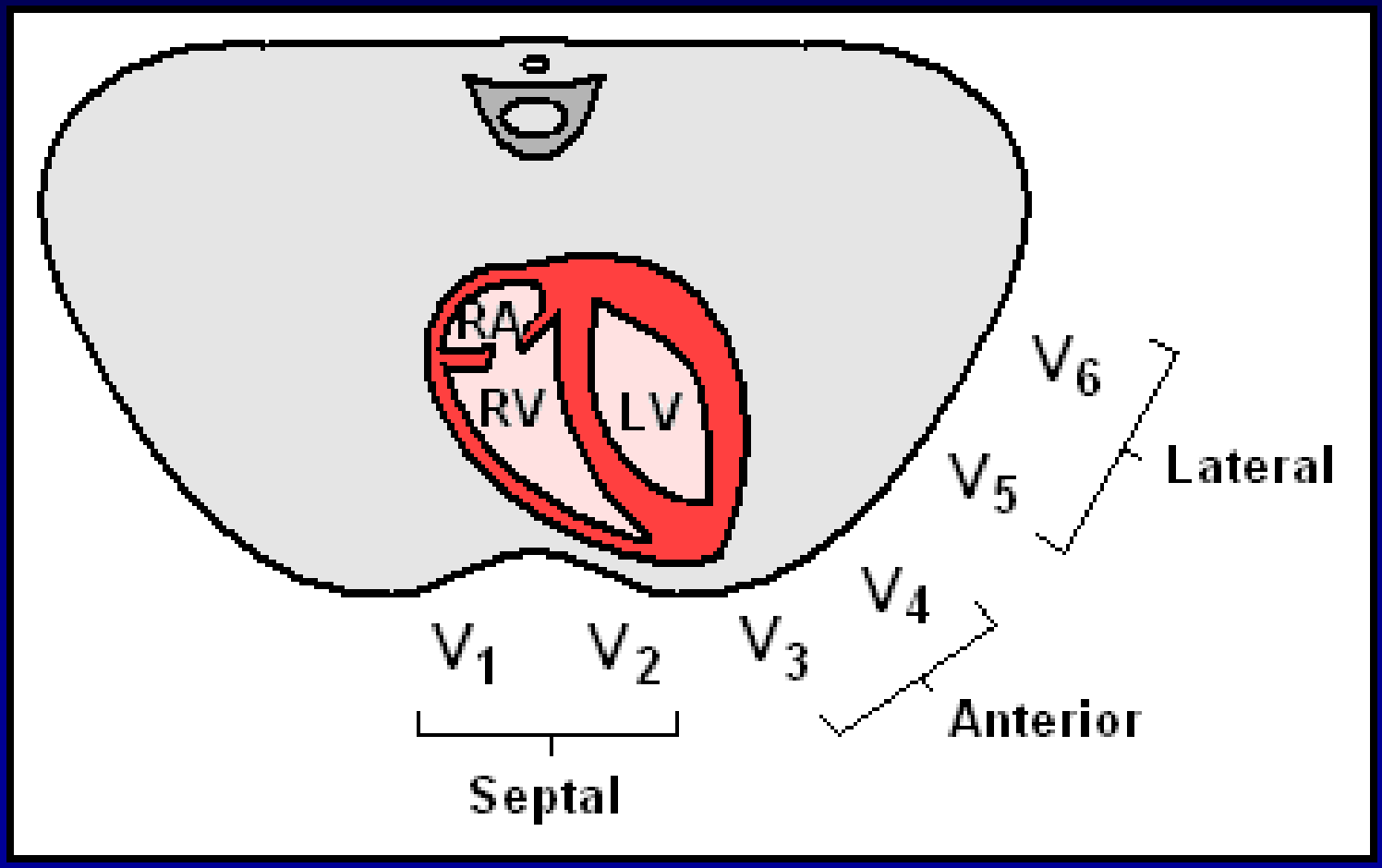
Precordial Leads

- Unipolar leads



V1 – 4 th intercostal space to rt of sternum
- V2 – 4th intercostal space to lt of the sternum
- V3 – between V2 and V4
- V4 – 5th intercostal space midclavicular line
- V5 – anterior axillary line, in line with V4
- V6 – midaxillary line, in line with V4

Lead Orientation



Summary of Leads

	Limb Leads	Precordial Leads
Bipolar	I, II, III (standard limb leads)	-
Unipolar	aVR, aVL, aVF (augmented limb leads)	V_1-V_6

Arrangement of Leads on the EKG

I	aVR	V_1	V_4
II	aVL	V_2	V_5
III	aVF	V_3	V_6

Anatomic Groups
(Septum)

I Lateral	aVR None	V_1 Septal	V_4 Anterior
II Inferior	aVL Lateral	V_2 Septal	V_5 Lateral
III Inferior	aVF Inferior	V_3 Anterior	V_6 Lateral

Anatomic Groups
(Anterior Wall)

I Lateral	aVR None	V_1 Septal	V_4 Anterior
II Inferior	aVL Lateral	V_2 Septal	V_5 Lateral
III Inferior	aVF Inferior	V_3 Anterior	V_6 Lateral

Anatomic Groups
(Lateral Wall)

I Lateral	aVR None	V_1 Septal	V_4 Anterior
II Inferior	aVL Lateral	V_2 Septal	V_5 Lateral
III Inferior	aVF Inferior	V_3 Anterior	V_6 Lateral

Anatomic Groups
(Inferior Wall)

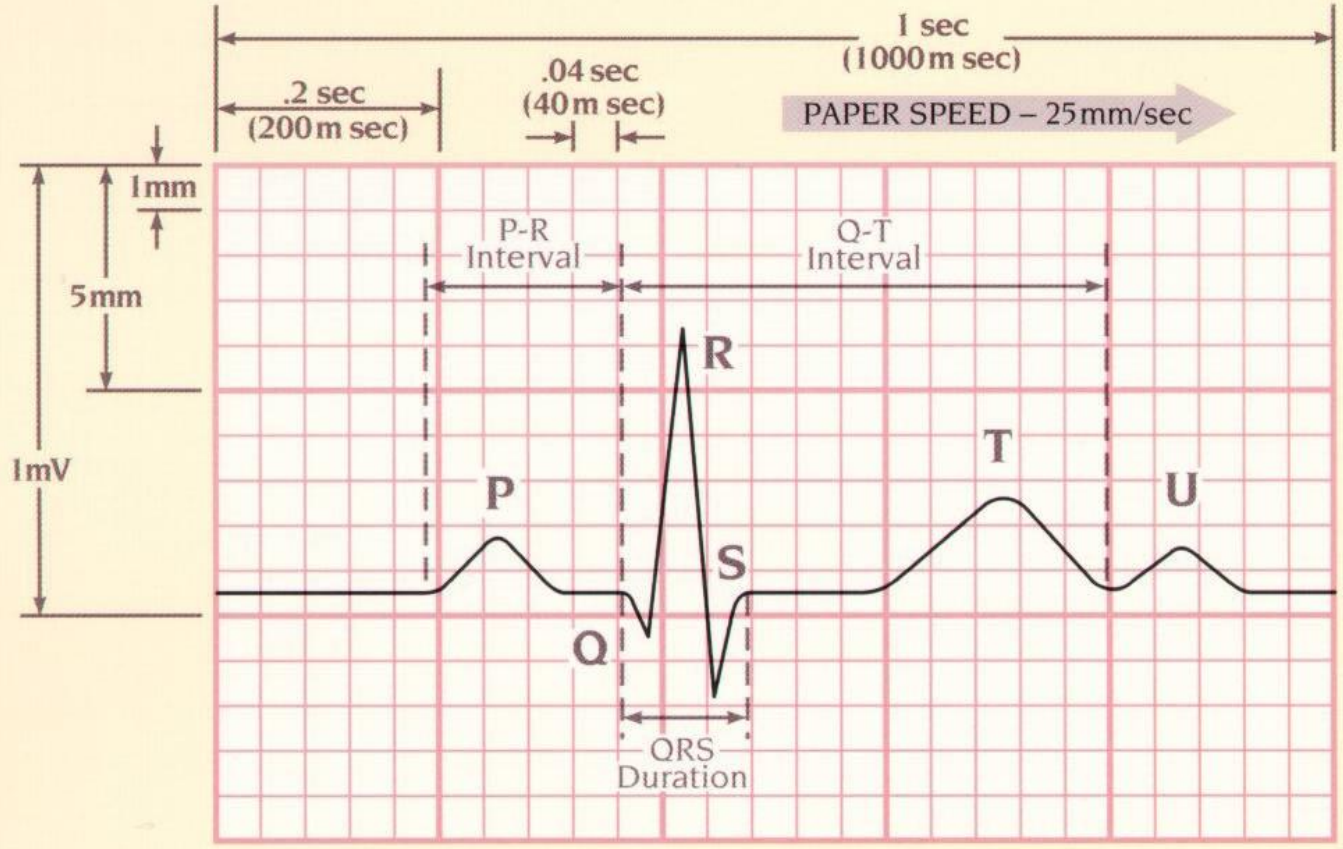
I Lateral	aVR None	V_1 Septal	V_4 Anterior
II Inferior	aVL Lateral	V_2 Septal	V_5 Lateral
III Inferior	aVF Inferior	V_3 Anterior	V_6 Lateral

Anatomic Groups
(Summary)

I Lateral	aVR None	V_1 Septal	V_4 Anterior
II Inferior	aVL Lateral	V_2 Septal	V_5 Lateral
III Inferior	aVF Inferior	V_3 Anterior	V_6 Lateral

NORMAL ECG TRACINGS

ECG



VERTICAL AXIS	
	1 Small Square = 1mm (0.1mV)
	1 Large Square = 5mm (0.5mV)
	2 Large Squares = 1mV

HORIZONTAL AXIS	
	1 Small Square = .04 sec (40 m sec)
	1 Large Square = .2 sec (200 m sec)
	5 Large Squares = 1 sec (1000 m sec)

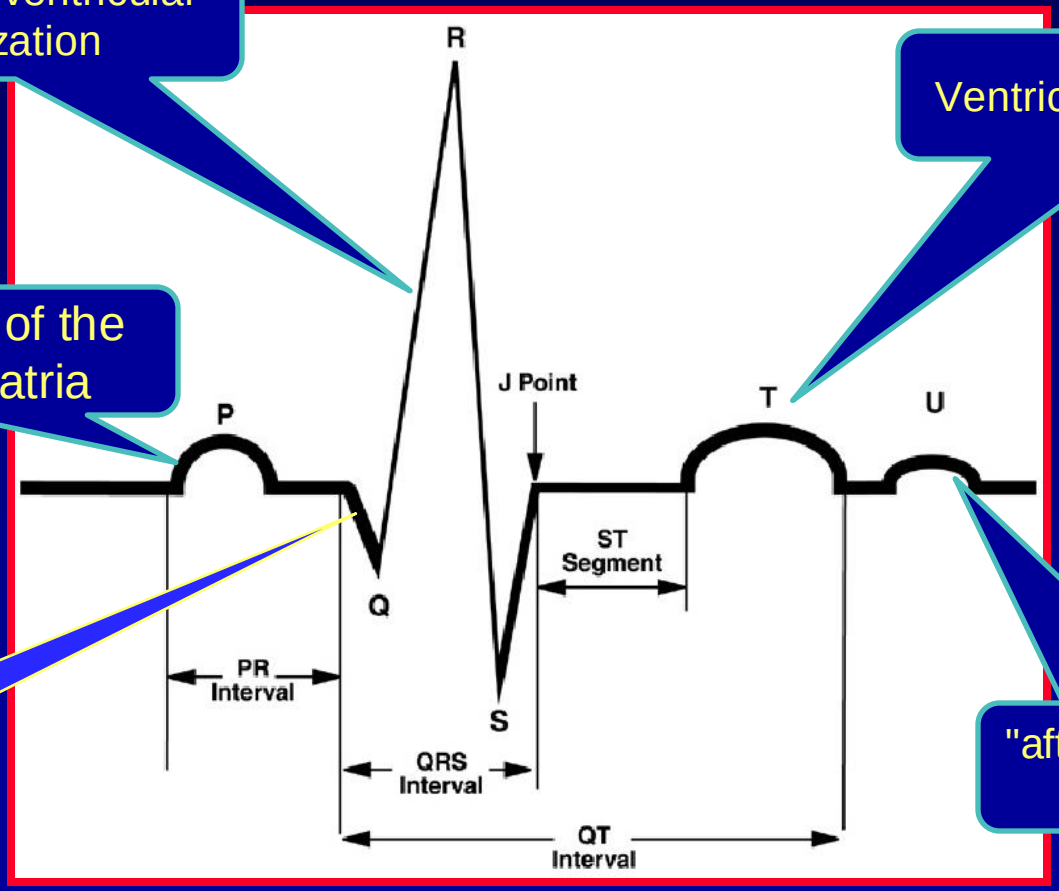
ECG

Right and left ventricular depolarization

Ventricular repolarization

Ti

Depolarization of the right and left atria



Septal depolarization

"after depolarizations" in the ventricles

Wave definition

- P wave
- Q wave – first downward deflection after P wave
- Rwave – first upward deflection after Q wave
- R` wave – any second upward deflection
- S wave – first downward deflection after the R wave

Waveform Description:

- **P Wave**

  It is important to remember that the P wave represents the *sequential* activation of the right and left atria, and it is common to see notched or biphasic P waves of right and left atrial activation.

  P duration < 0.12 sec

  P amplitude < 2.5 mm

QRS Complex

The QRS represents the *simultaneous* activation of the right and left ventricles, QRS duration $\leq$ 0.12 sec

Two determinates of QRS voltages are:

Size of the ventricular chambers (i.e., the larger the chamber, the larger the voltage)

Proximity of chest electrodes to ventricular chamber (the closer, the larger the voltage)

QRS Complex

Normal q-waves reflect normal septal activation (beginning on the LV septum); they are narrow (<0.04s duration) and small (<25% the amplitude of the R wave).

They are often seen

In leads I and aVL when the QRS axis is to the **left** of $+60^{o}$, and

in leads II, III, aVF when the QRS axis is to the **right** of $+60^{o}$.

Septal q waves should not be confused with the pathologic Q waves of myocardial infarction.

QRS Complex

Small r-waves begin in V1 or V2 and progress in size to V5. The R-V6 is usually smaller than R-V5.

In reverse, the s-waves begin in V6 or V5 and progress in size to V2. S-V1 is usually smaller than S-V2.

ST Segment and T wave

ST-T wave is a smooth, continuous waveform beginning with the J-point (end of QRS), slowly rising to the peak of the T

Normal ECG the T wave is always upright in leads I, II, V3-6, and always inverted in lead aVR.

Normal ST segment configuration is ***concave upward***

Convex or straight upward **ST segment elevation is abnormal and suggests transmural injury or infarction**

ST segment depression characterized as "upsloping", "horizontal", or "downsloping" is always an abnormal finding

METHOD OF ECG INTERPRETATION

Method of ECG interpretation

1. **Measurements**

Heart rate	(state atrial and ventricular, if different)
PR interval	(from beginning of P to beginning of QRS)
QRS duration	(width of most representative QRS)
QT interval	(from beginning of QRS to end of T)
QRS axis	

2. **Rhythm Analysis**

State basic rhythm (e.g., "normal sinus rhythm", "atrial fibrillation", etc.)
Identify additional rhythm events if present (e.g., "PVC's", "PAC's", etc)

3. **Conduction Analysis**

Normal" conduction implies normal sino-atrial (SA), atrio-ventricular (AV), and intraventricular (IV) conduction

AV block - 1st, 2nd (type I vs. type II), and 3rd degree

Method of ECG interpretation Cont.

4 .Waveform Description

P waves : Are they too wide, too tall,

QRS complexes : look for pathological Q waves, abnormal voltage.

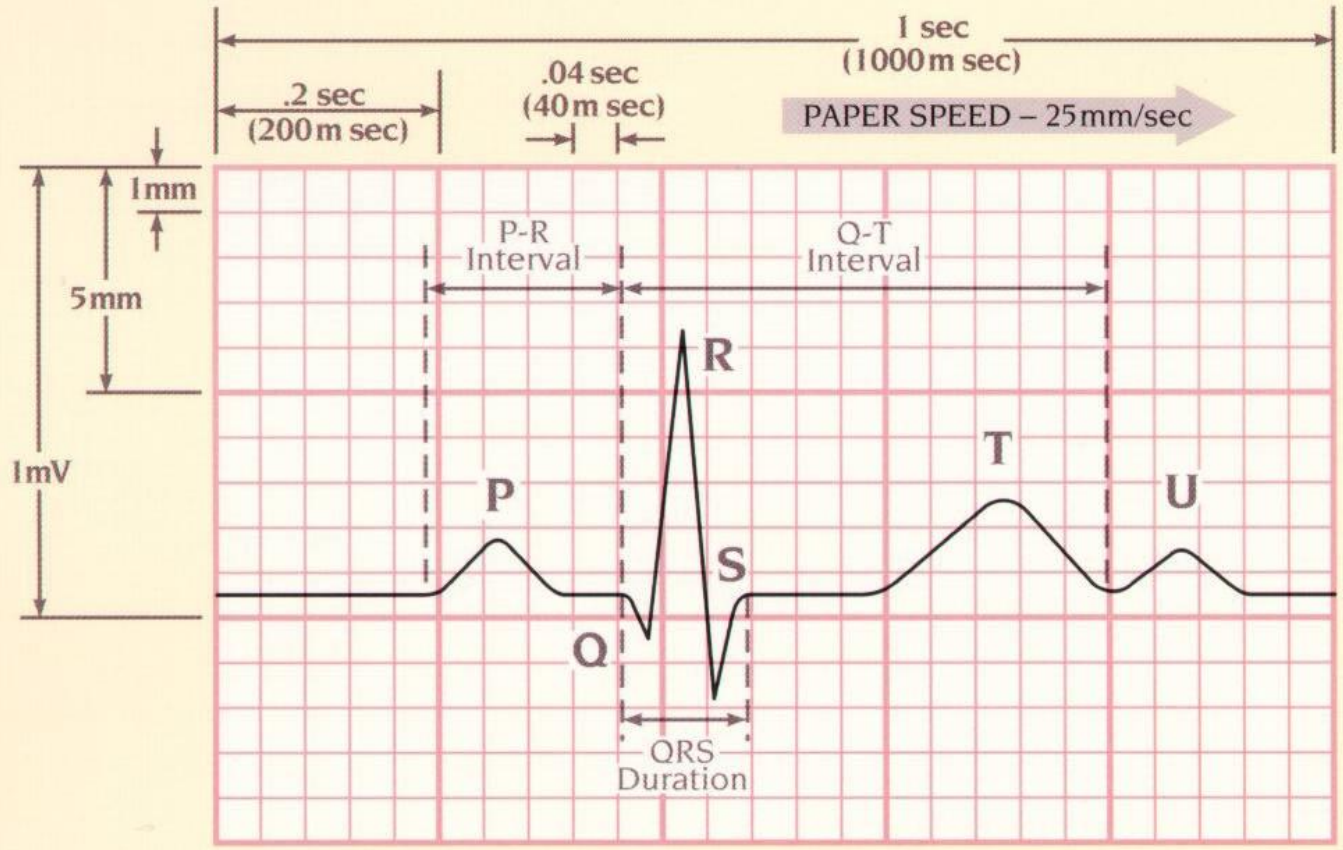
ST segments : look for abnormal ST elevation and/or depression.

T waves : look for abnormally inverted T waves.

U waves : look for prominent or inverted U waves

ECG- Heart rate

- ECG paper moves at a standardized 25mm/sec
- Each large square is 5 mm
- Each large square is 0.2 sec
- 300 large squares per minute / 1500 small squares per minute
- 300 divided by number of large squares between R-R
- 1500 divided by number of small squares between R-R



VERTICAL AXIS	1 Small Square	= 1mm (0.1mV)	
	1 Large Square	= 5mm (0.5mV)	
	2 Large Squares	= 1mV	

HORIZONTAL AXIS	1 Small Square	= .04 sec (40 m sec)
	1 Large Square	= .2 sec (200 m sec)
	5 Large Squares	= 1 sec (1000 m sec)

1. Measurements (Normal)

Heart Rate: 60 - 100 bpm

PR Interval: 0.12 - 0.20 sec

QRS Duration: 0.06 - 0.12 sec

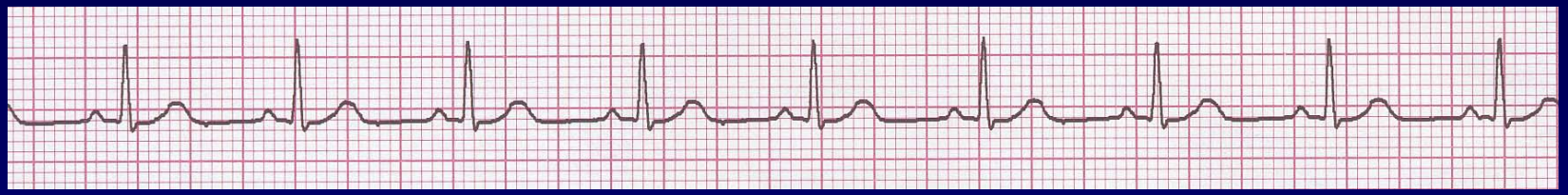
QT Interval ($QT_c \leq 0.45$ sec)

Frontal Plane QRS Axis: +90 ^{o} to -30 ^{o} (in adults)

ECG Rhythm Interpretation

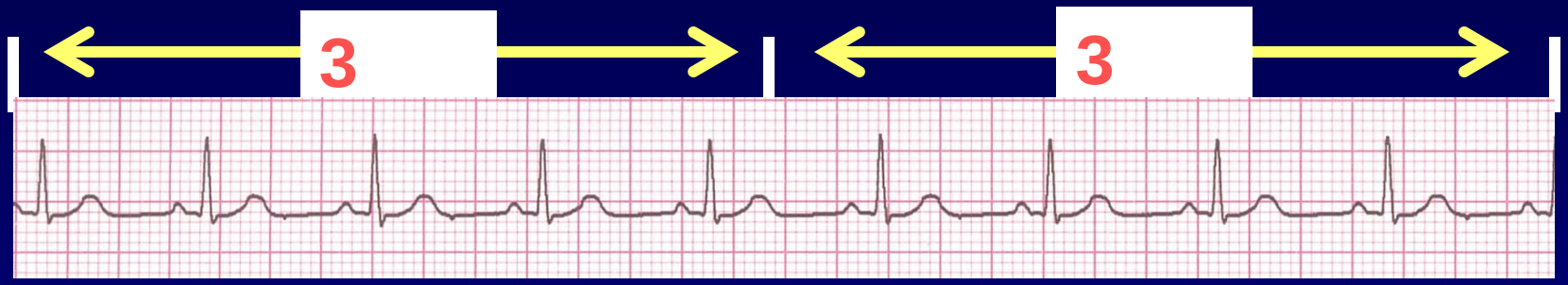
How to Analyze a Rhythm

Normal Sinus Rhythm (NSR)



- Etiology: the electrical impulse is formed in the SA node and conducted normally.
- This is the normal rhythm of the heart; other rhythms that do not conduct via the typical pathway are called arrhythmias.

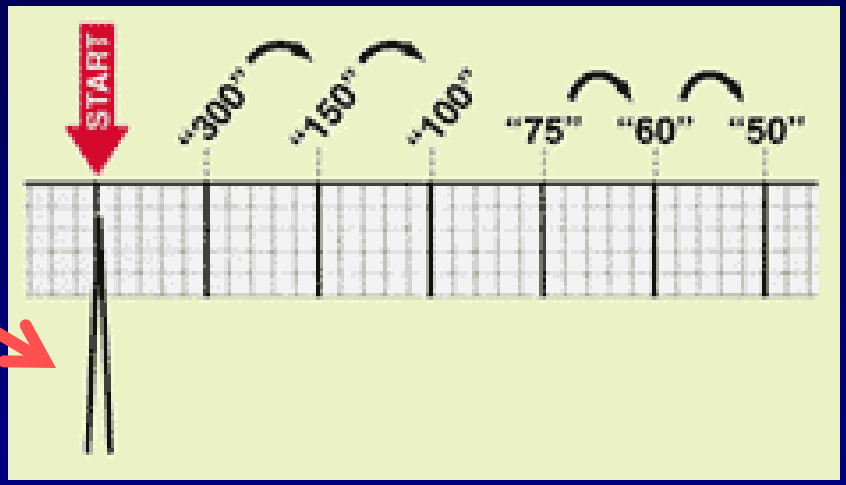
Step 1: Calculate Rate



- Option 1
  - Count the # of R waves in a 6 second rhythm strip, then multiply by 10.
  - Reminder: all rhythm strips in the Modules are 6 seconds in length.

Interpretation? *9 x 10 = 90 bpm*

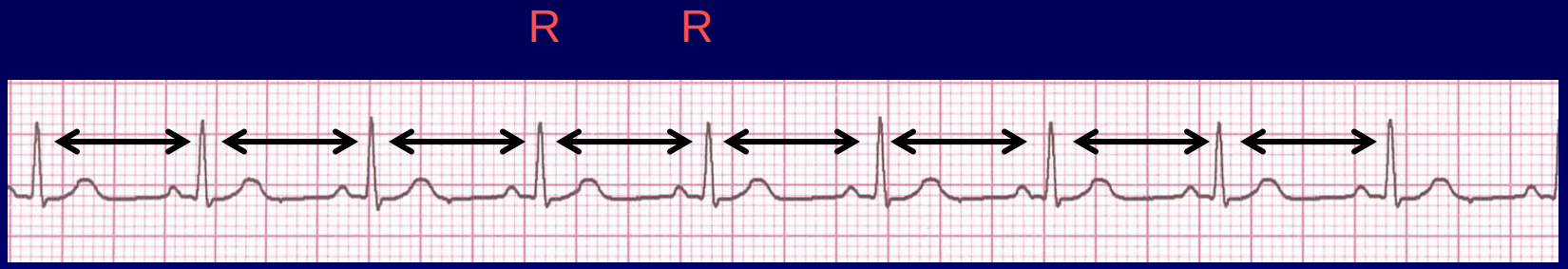
Step 1: Calculate Rate



R
wave

- Option 2
  - Find a R wave that lands on a bold line.
  - Count the # of large boxes to the next R wave. If the second R wave is 1 large box away the rate is 300, 2 boxes - 150, 3 boxes - 100, 4 boxes - 75, etc. (cont)

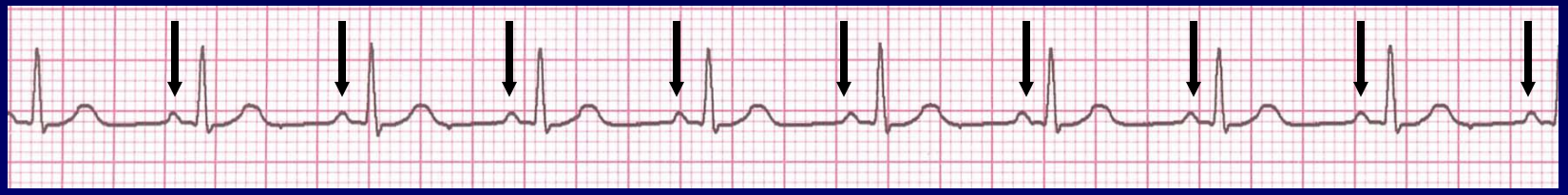
Step 2: Determine regularity



- Look at the R-R distances (using a caliper or markings on a pen or paper).
- Regular (are they equidistant apart)? Occasionally irregular? Regularly irregular? Irregularly irregular?

Interpretation? *Regular*

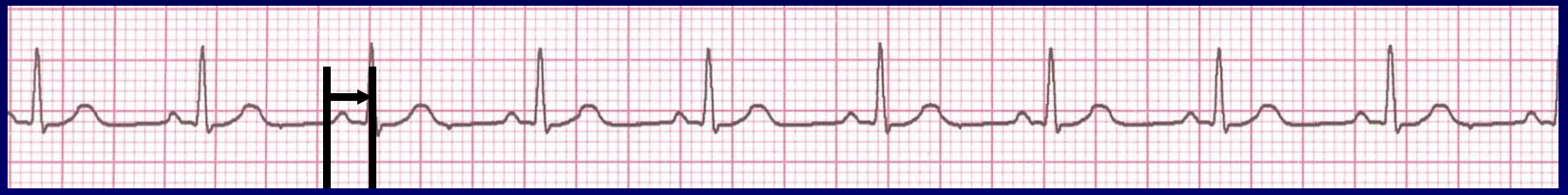
Step 3: Assess the P waves



- Are there P waves?
- Do the P waves all look alike?
- Do the P waves occur at a regular rate?
- Is there one P wave before each QRS?

Interpretation? *Normal P waves with 1 P wave for every QRS*

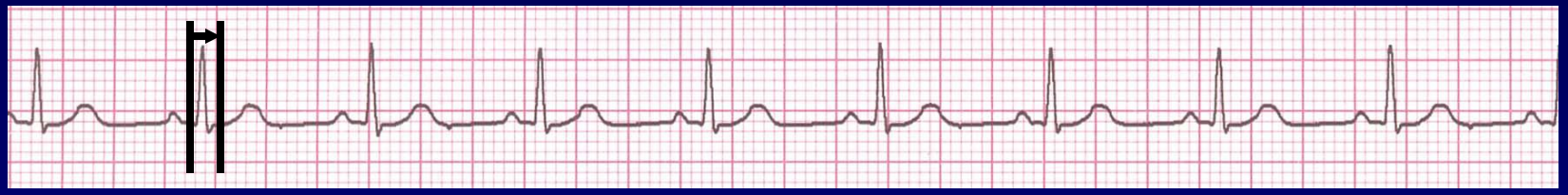
Step 4: Determine PR interval



- Normal: 0.12 - 0.20 seconds.
  (3 - 5 boxes)

Interpretation? *0.12 seconds*

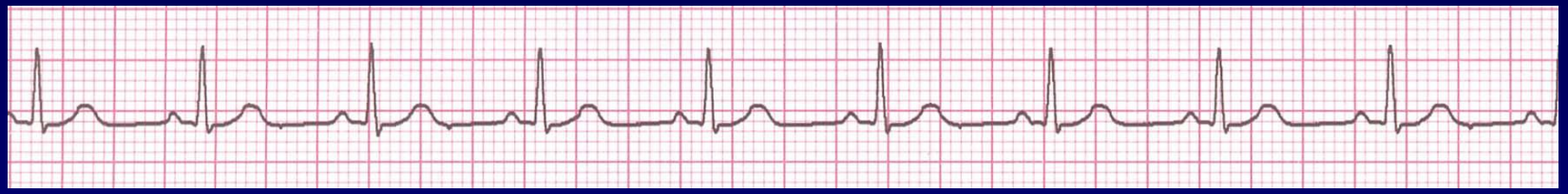
Step 5: QRS duration



- Normal: 0.04 - 0.12 seconds.
  (1 - 3 boxes)

Interpretation? *0.08 seconds*

Rhythm Summary



- Rate 90 bpm
- Regularity regular
- P waves normal
- PR interval 0.12 s
- QRS duration 0.08 s

Interpretation? *Normal Sinus Rhythm*

RHYTHM DISTURBANCES

Arrhythmia Formation

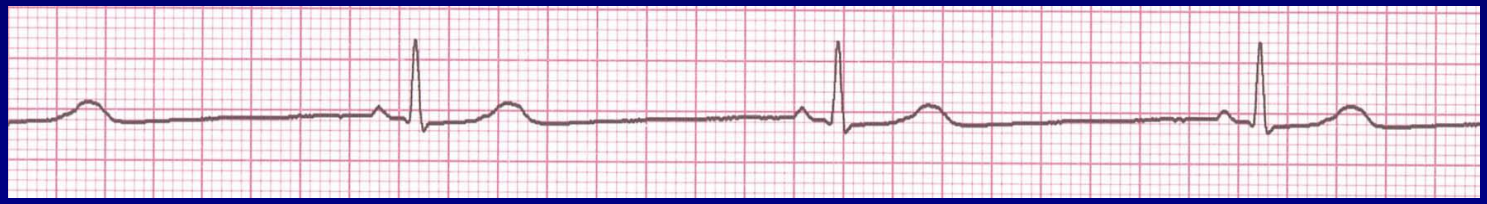
Arrhythmias can arise from problems in the:

- Sinus node
- Atrial cells
- AV junction
- Ventricular cells

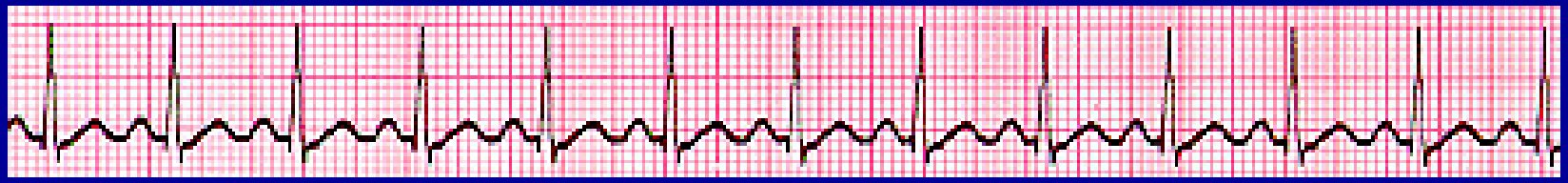
SA Node Problems

The SA Node can:

- fire too slow *Sinus Bradycardia*



- fire too fast *Sinus Tachycardia*



Atrial Cell Problems

Atrial cells can:

- fire occasionally from a focus — *Premature Atrial Contractions (PACs)*
- fire continuously due to a looping re-entrant circuit — *Atrial Flutter*

Atrial Cell Problems

Atrial cells can also:

- fire continuously from multiple foci

or

fire continuously due to multiple micro re-entrant "wavelets"

Atrial Fibrillation

AV Junctional Problems

The AV junction can:

- fire continuously due to a looping re-entrant circuit
- block impulses coming from the SA Node

Paroxysmal Supraventricular Tachycardia

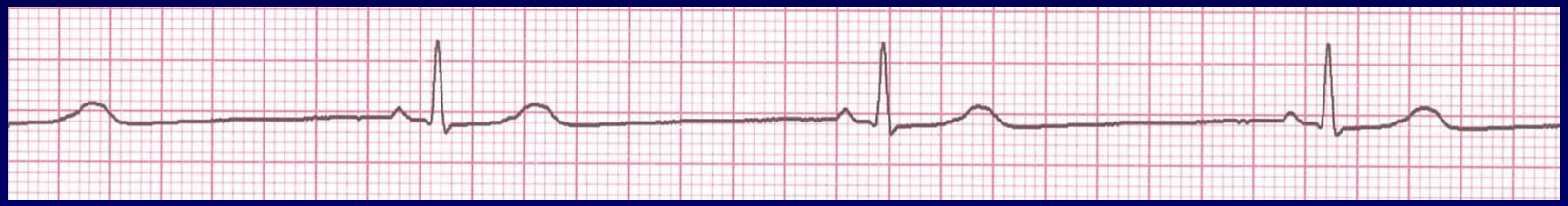
AV Junctional Blocks

Ventricular Cell Problems

Ventricular cells can:

- fire occasionally from 1 or more foci — *Premature Ventricular Contractions (PVCs)*
- fire continuously from multiple foci — *Ventricular Fibrillation*
- fire continuously due to a looping re-entrant circuit — *Ventricular Tachycardia*

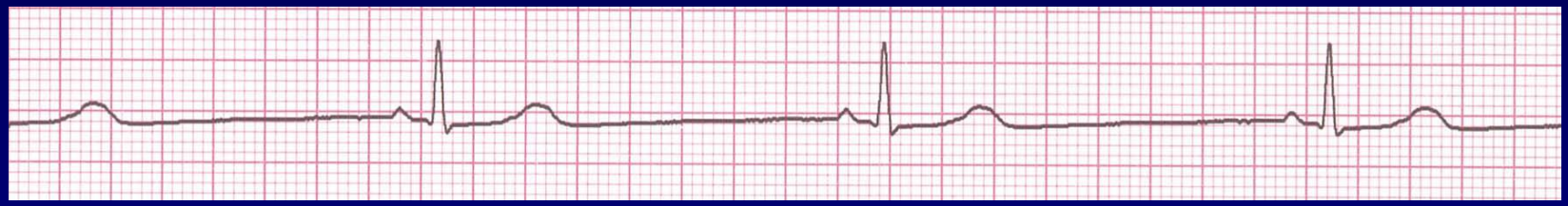
Sinus Bradycardia



- Rate? 30 bpm
- Regularity? regular
- P waves? normal
- PR interval? 0.12 s
- QRS duration? 0.10 s

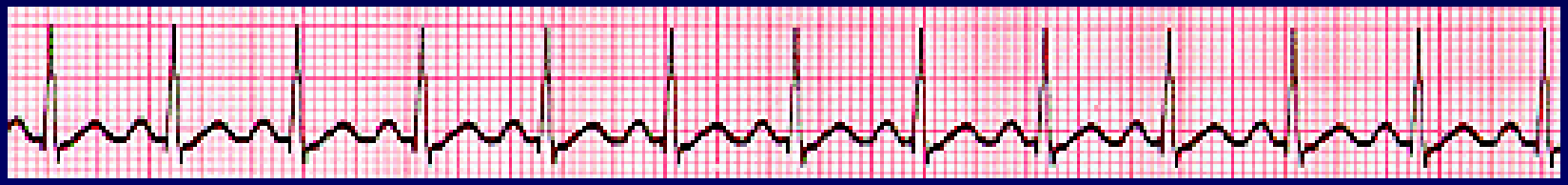
Interpretation? *Sinus Bradycardia*

Sinus Bradycardia



- Etiology: SA node is depolarizing slower than normal, impulse is conducted normally (i.e. normal PR and QRS interval).

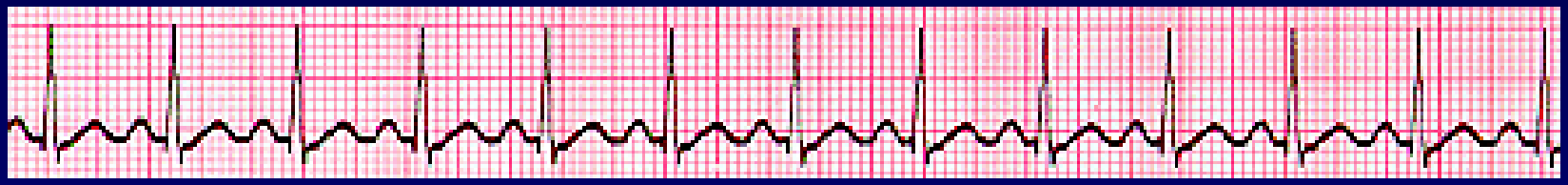
Sinus Tachycardia



- Rate? 130 bpm
- Regularity? regular
- P waves? normal
- PR interval? 0.16 s
- QRS duration? 0.08 s

Interpretation? *Sinus Tachycardia*

Sinus Tachycardia

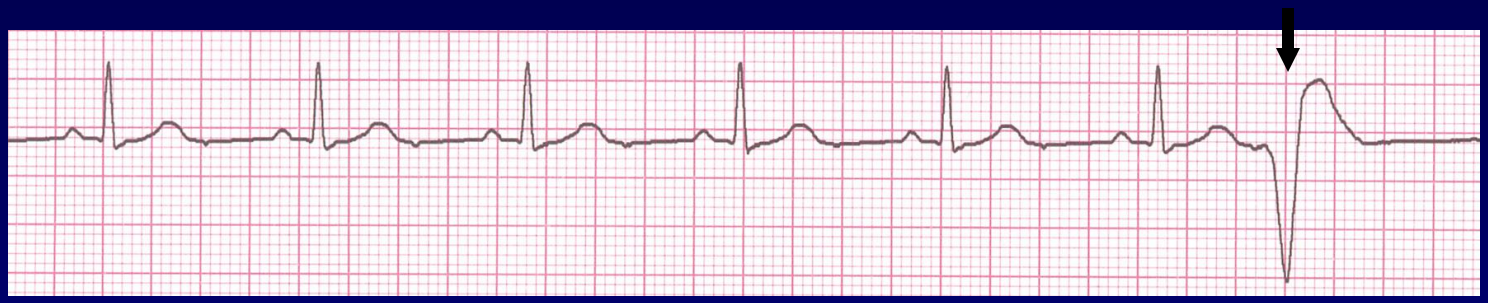


- Etiology: SA node is depolarizing faster than normal, impulse is conducted normally.
- Remember: sinus tachycardia is a response to physical or psychological stress, not a primary arrhythmia.

Premature Beats

- *Premature Ventricular Contractions (PVCs)*

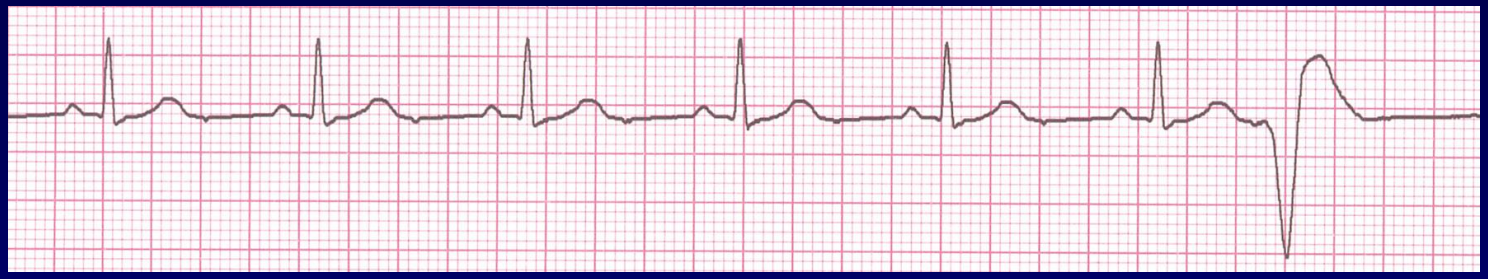
Sinus Rhythm with 1 PVC



- Rate? 60 bpm
- Regularity? occasionally irreg.
- P waves? none for 7th QRS
- PR interval? 0.14 s
- QRS duration? 0.08 s (7th wide)

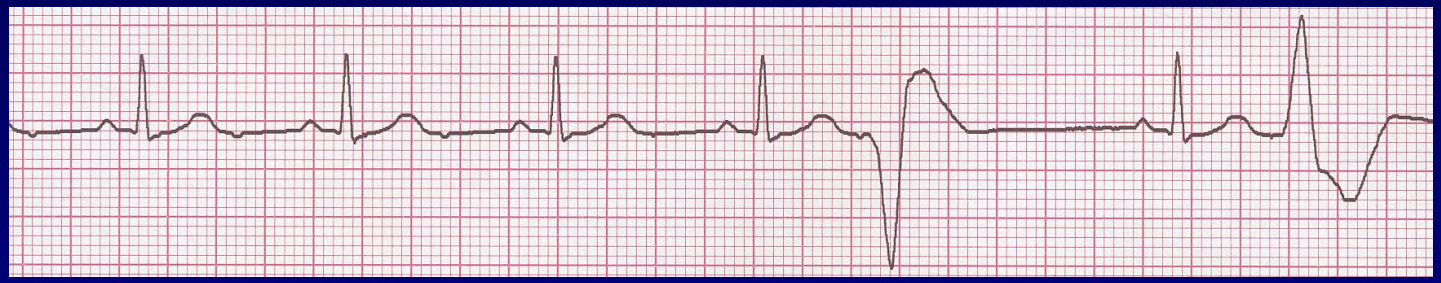
Interpretation? *Sinus Rhythm with 1 PVC*

PVCs



- Deviation from NSR
  - Ectopic beats originate in the ventricles resulting in wide and bizarre QRS complexes.
  - When there are more than 1 premature beats and look alike, they are called “uniform”. When they look different, they are called “multiform”.

PVCs



- Etiology: One or more ventricular cells are depolarizing and the impulses are abnormally conducting through the ventricles.

QRS AXIS DETERMINATION

The QRS Axis

The QRS axis represents the net overall direction of the heart's electrical activity.

Abnormalities of axis can hint at:

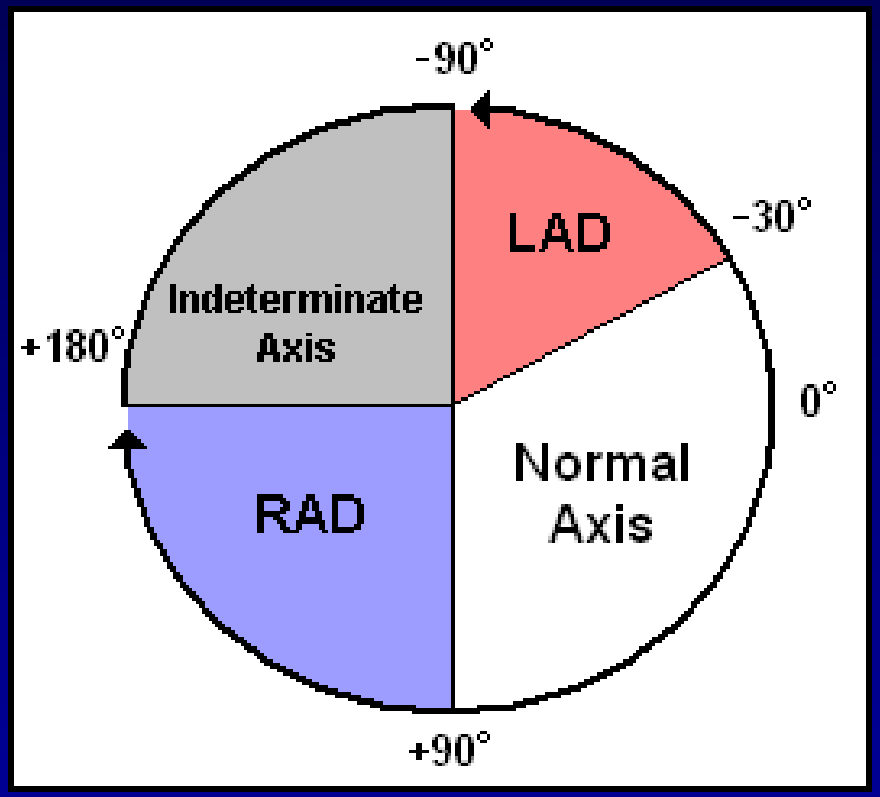
- Ventricular enlargement
- Conduction blocks (i.e. hemiblocks)

The QRS Axis

By near-consensus, the normal QRS axis is defined as ranging from -30° to +90°.

-30° to -90° is referred to as a left axis deviation (LAD)

+90° to +180° is referred to as a right axis deviation (RAD)

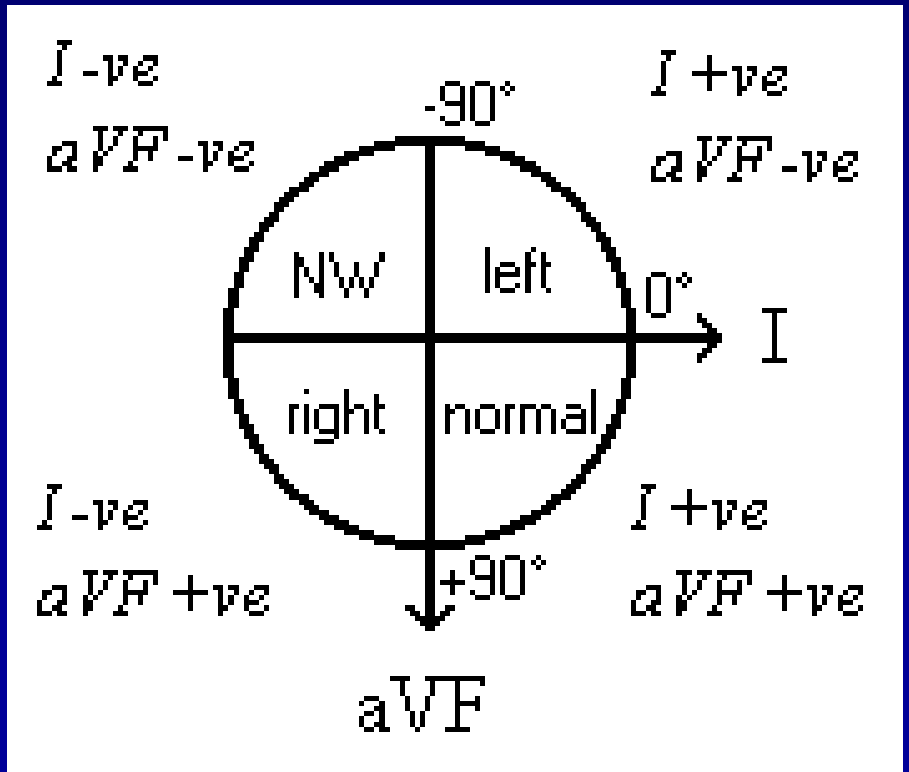


Determining the Axis

- The Quadrant Approach

The Quadrant Approach

1. Examine the QRS complex in leads I and aVF to determine if they are predominantly positive or predominantly negative. The combination should place the axis into one of the 4 quadrants below.



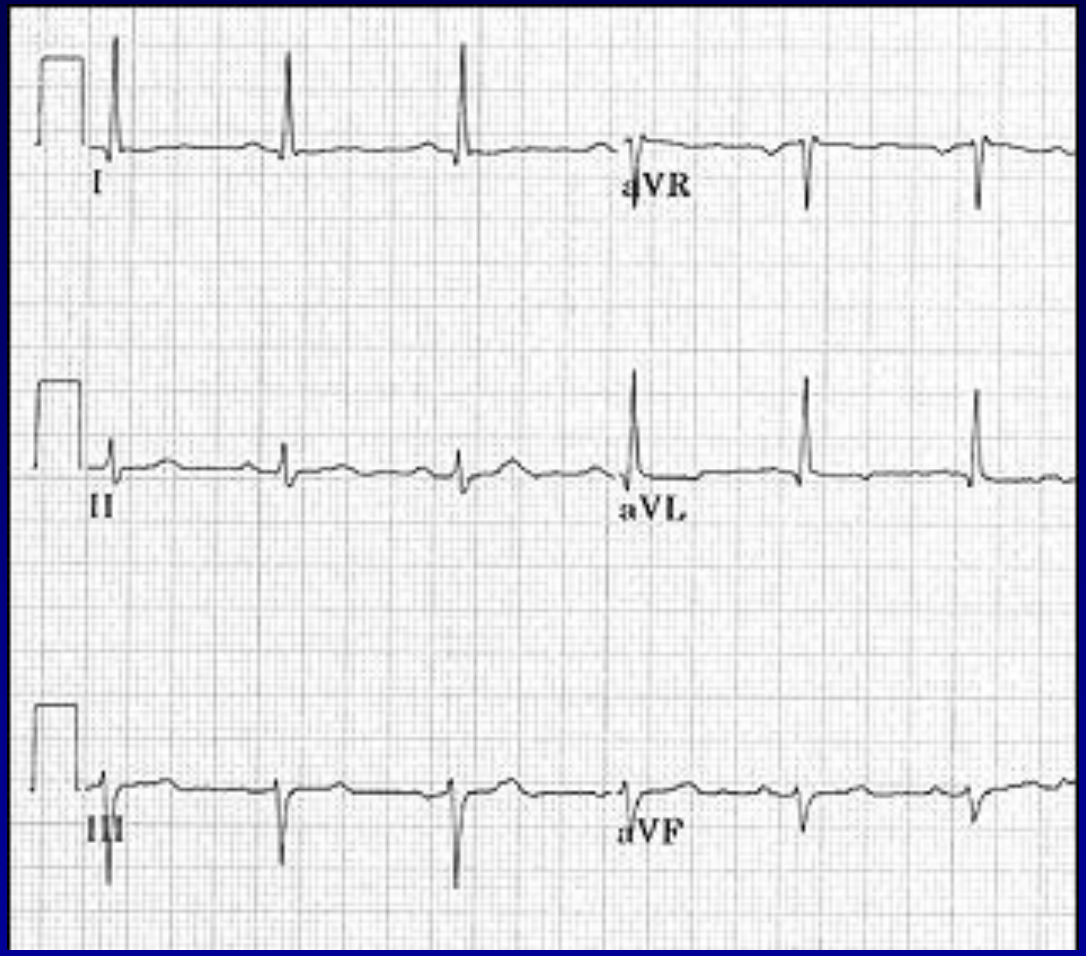
		Lead aVF	
		Positive	Negative
Lead I	Positive	Normal Axis	LAD
	Negative	RAD	Indeterminate Axis

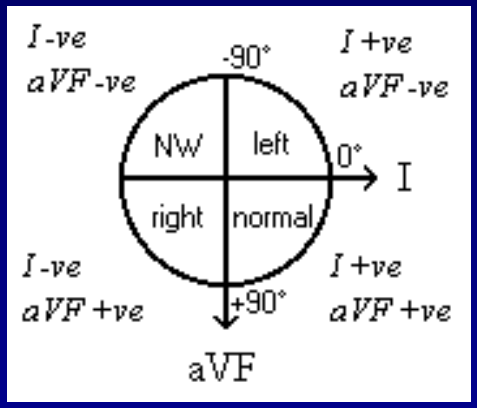
The Quadrant Approach

2. In the event that LAD is present, examine lead II to determine if this deviation is pathologic. If the QRS in II is predominantly positive, the LAD is non-pathologic (in other words, the axis is normal). If it is predominantly negative, it is pathologic.

		Lead aVF	
		Positive	Negative
Lead I	Positive	Normal Axis	LAD
	Negative	RAD	Indeterminate Axis

Quadrant Approach





The Alan E. Lindsay ECG Learning Center http://medstat.med.utah.edu/kw/ecg/

Positive in I, negative in aVF → Predominantly positive in II →

Normal Axis (non-pathologic LAD)

CONDUCTION DISTURBANCES

Measurement abnormality

PR Interval

PR Interval

Normal: 0.12 - 0.20s

Short PR: < 0.12s

Preexcitation syndromes:

WPW (Wolff-Parkinson-White) Syndrome:

An accessory pathway (called the "Kent" bundle)
connects the right atrium to the right ventricle or
the left atrium to the left ventricle, and
this permits early activation of the ventricles
(*delta* wave) and a short PR interval.

Short PR Interval

AV Junctional Rhythms

with retrograde atrial activation (inverted P waves in II, III, aVF):

Retrograde P waves may occur *before* the QRS complex (usually with a short PR interval), *in* the QRS complex (i.e., hidden from view), or *after* the QRS complex (i.e., in the ST segment).

Ectopic atrial rhythms

originating near the AV node (the PR interval is short because atrial activation originates close to the AV node; the P wave morphology is different from the sinus P)

Normal variant

Prolonged PR: >0.20s

First degree AV block

(PR interval usually constant)

Second degree AV block

(PR interval may be normal or prolonged; some P waves do not conduct)

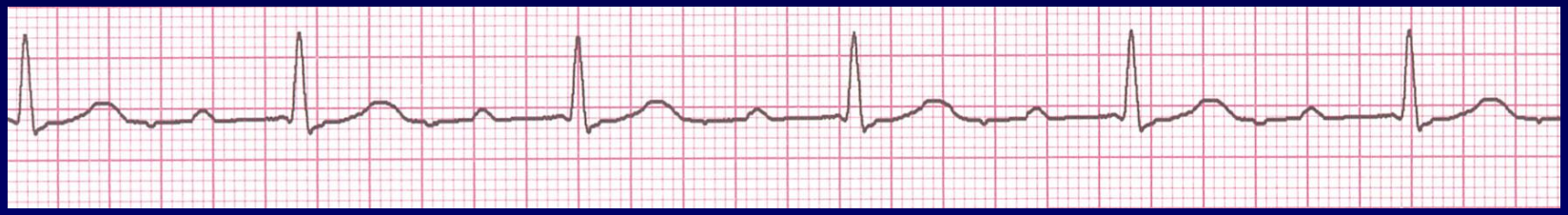
Type I (Wenckebach): Increasing PR until nonconducted P wave occurs

Type II (Mobitz): Fixed PR intervals plus nonconducted P waves

AV dissociation:

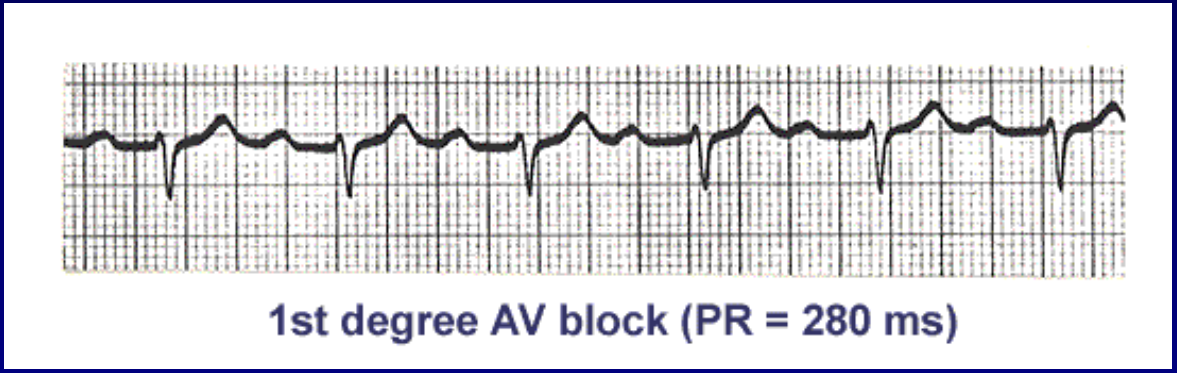
Some PR's may appear prolonged, but the P waves and QRS complexes are dissociated

1st Degree AV Block



- Etiology: Prolonged conduction delay in the AV node or Bundle of His.

First degree AV block

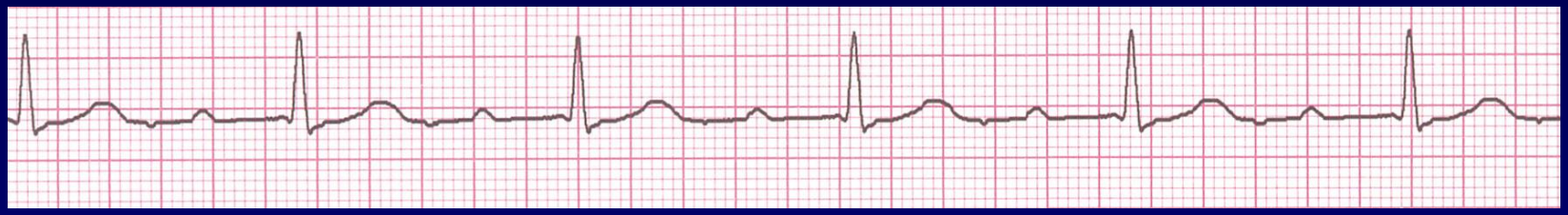


1st degree AV block (PR = 280 ms)

1st degree AV block is defined by PR intervals greater than 200 ms caused by

drugs, such as BB, CCB;
excessive vagal tone;
ischemia; or
intrinsic disease in the AV junction or bundle branch system

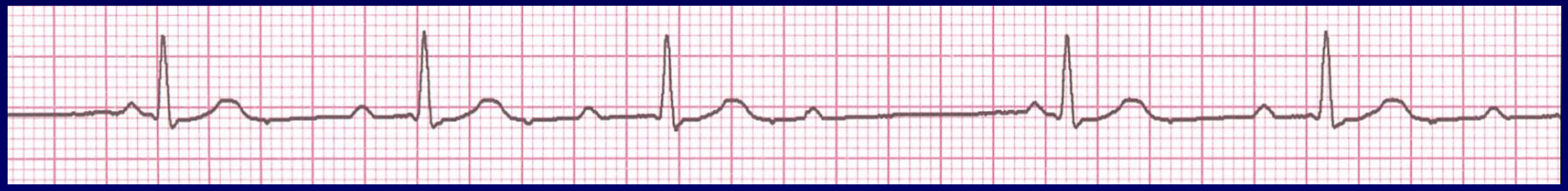
First degree AV block



- Rate? 60 bpm
- Regularity? regular
- P waves? normal
- PR interval? 0.36 s
- QRS duration? 0.08 s

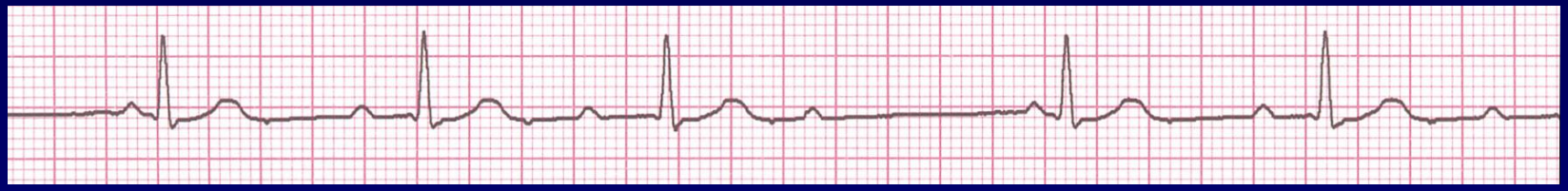
Interpretation? *1st Degree AV Block*

2nd Degree AV Block, Type I



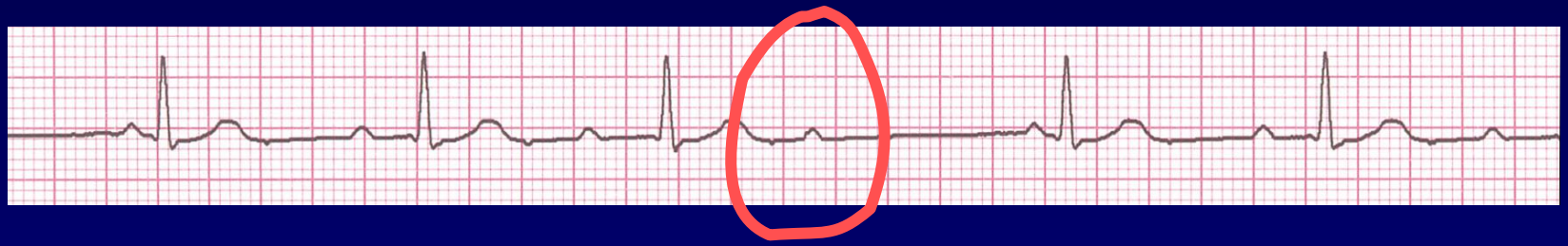
- Deviation from NSR
  - PR interval progressively lengthens, then the impulse is completely blocked (P wave not followed by QRS).

2nd Degree AV Block, Type I



- Etiology: Each successive atrial impulse encounters a longer and longer delay in the AV node until one impulse (usually the 3rd or 4th) fails to make it through the AV node.

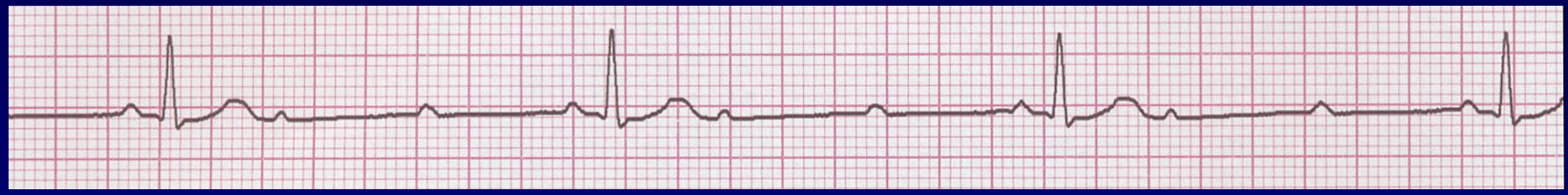
2nd Degree AV Block, Type I



- Rate? 50 bpm
- Regularity? regularly irregular
- P waves? nl, but 4th no QRS
- PR interval? lengthens
- QRS duration? 0.08 s

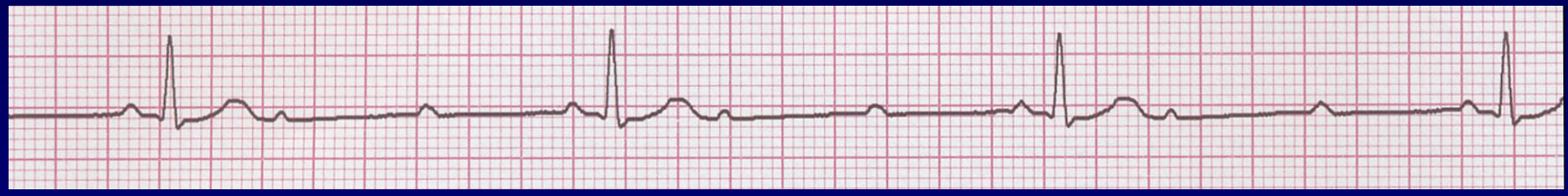
Interpretation? *2nd Degree AV Block, Type I*

2nd Degree AV Block, Type II (Mobitz)



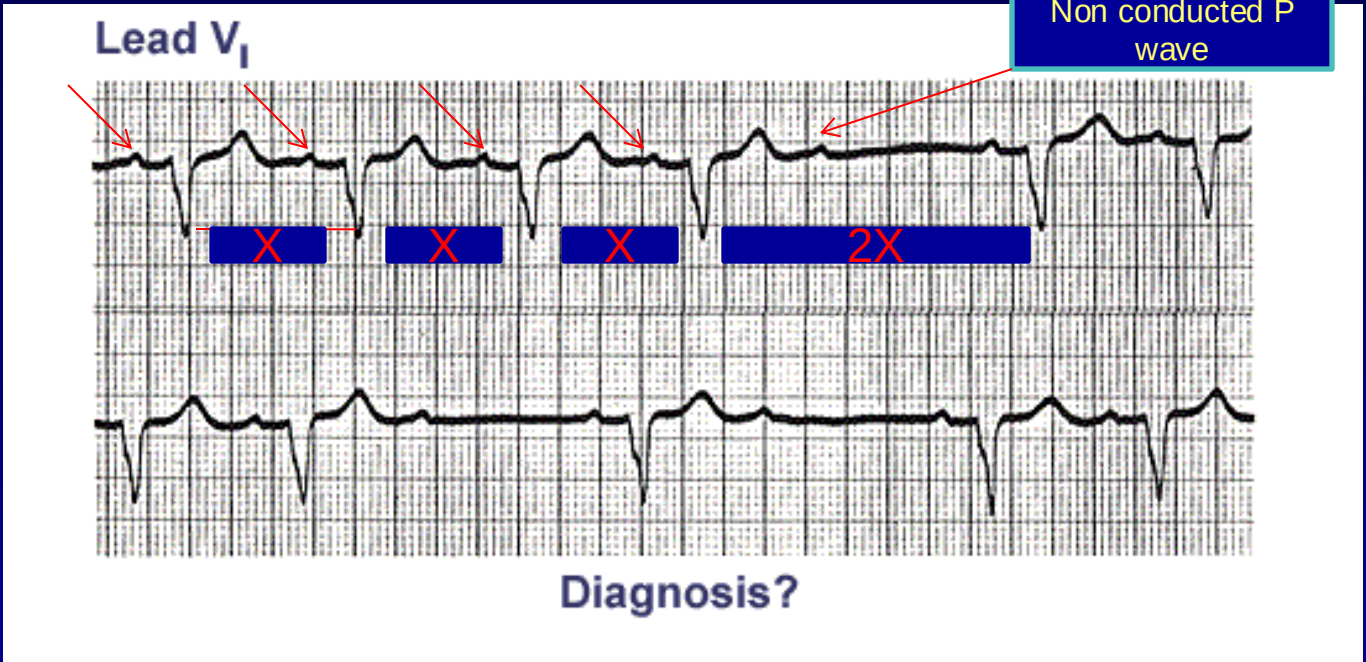
- Deviation from NSR
  - Occasional P waves are completely blocked (P wave not followed by QRS).

2nd Degree AV Block, Type II (Mobitz)



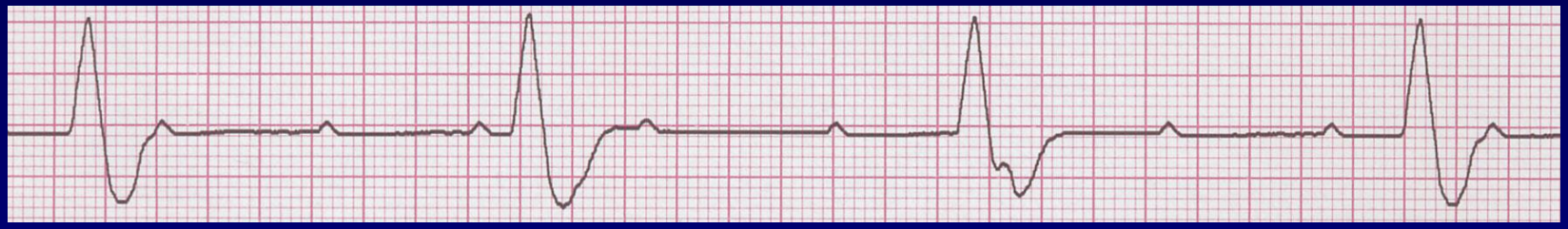
- Etiology: Conduction is all or nothing (no prolongation of PR interval); typically block occurs in the Bundle of His.

Type II - AV block (Mobitz)



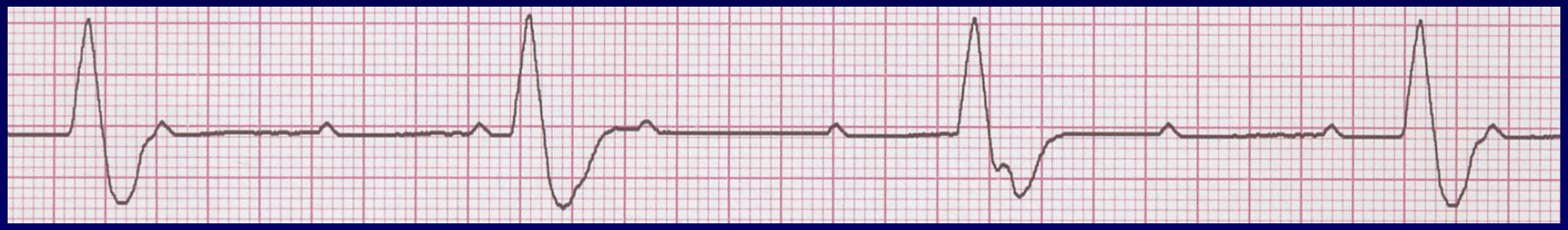
In **type II AV block**, the PR is constant until the nonconducted P wave occurs. The RR interval of the pause is usually 2x the basic RR interval.
Block may be 2:1 or 3:1

3rd Degree AV Block



- Deviation from NSR
  - The P waves are completely blocked in the AV junction; QRS complexes originate independently from below the junction.

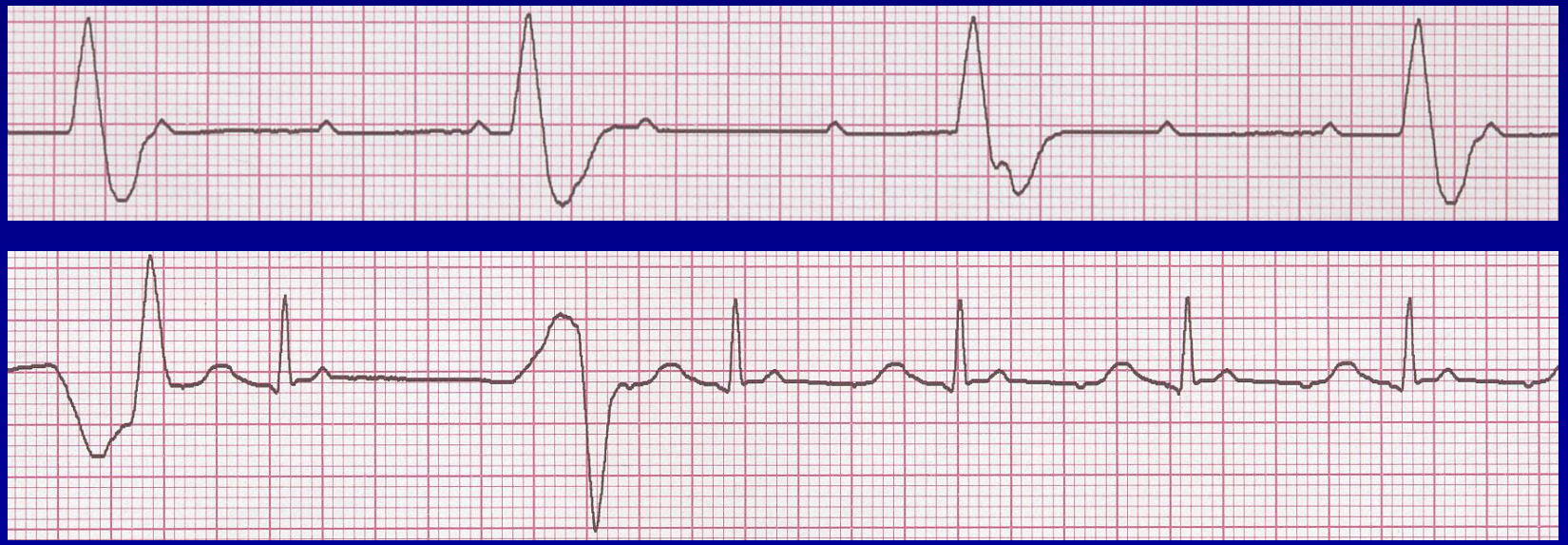
3rd Degree AV Block



- Etiology: There is complete block of conduction in the AV junction, so the atria and ventricles form impulses independently of each other. Without impulses from the atria, the ventricles own intrinsic pacemaker kicks in at around 30 - 45 beats/minute.

Remember

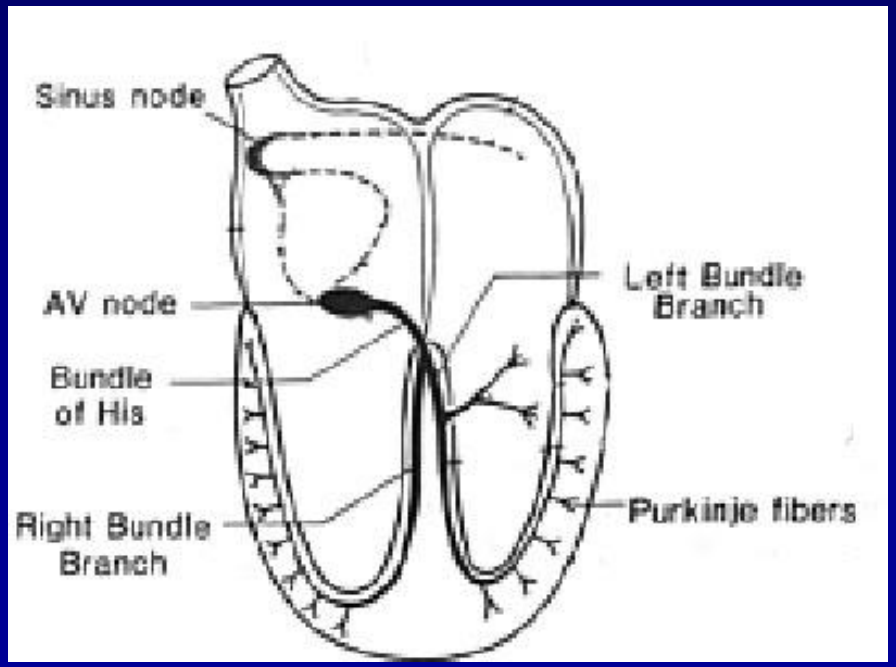
- When an impulse originates in a ventricle, conduction through the ventricles will be inefficient and the QRS will be wide and bizarre.



Bundle Branch Block

Bundle Branch Blocks

Turning our attention to bundle branch blocks…



Remember normal impulse conduction is

SA node →

AV node →

Bundle of His →

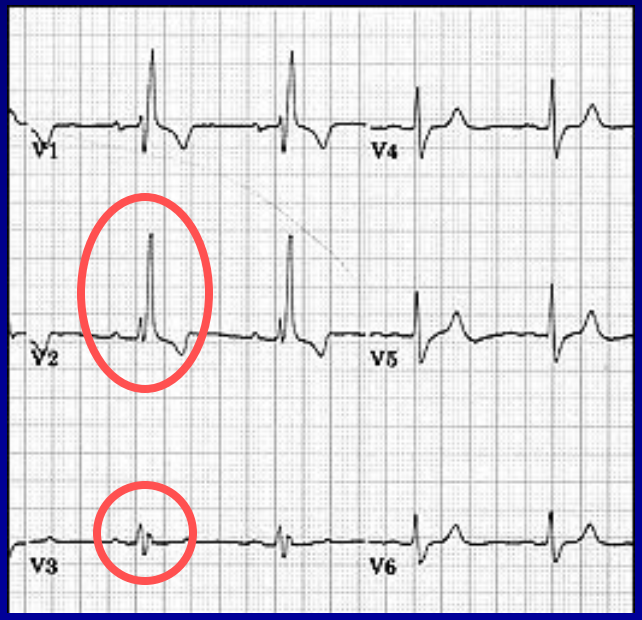
Bundle Branches →

Purkinje fibers

Bundle Branch Blocks

With Bundle Branch Blocks you will see two changes on the ECG.

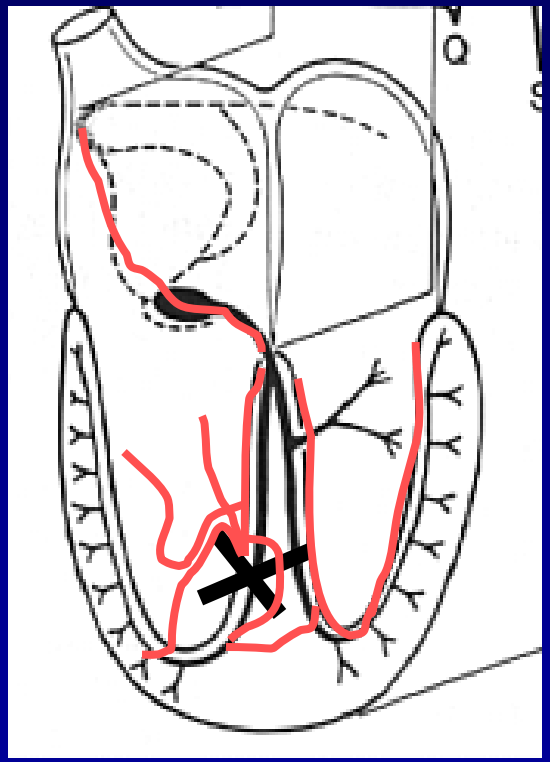
1. **QRS complex widens (> 0.12 sec).**
2. **QRS morphology changes (varies depending on ECG lead, and if it is a right vs. left bundle branch block).**



Bundle Branch Blocks

Why does the QRS complex widen?

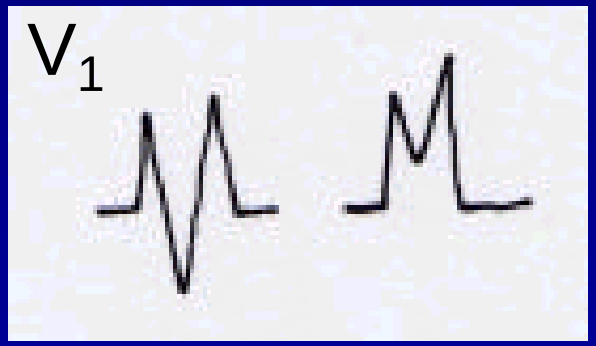
When the conduction pathway is blocked it will take longer for the electrical signal to pass throughout the ventricles.



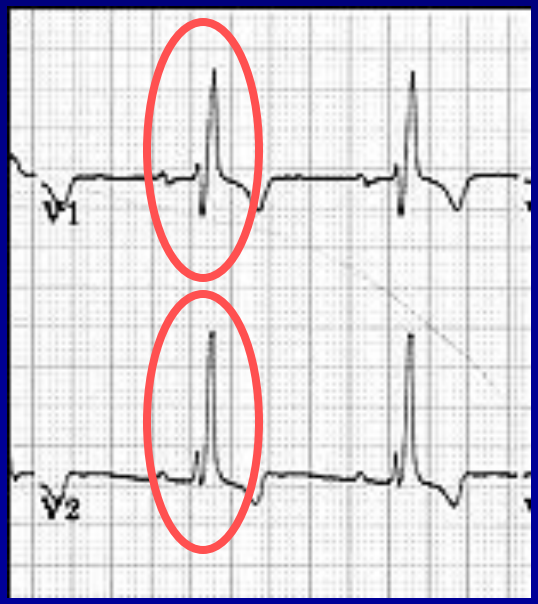
Right Bundle Branch Blocks

What QRS morphology is characteristic?

For RBBB the wide QRS complex assumes a unique, virtually diagnostic shape in those leads overlying the right ventricle (V_1 and V_2).



"Rabbit Ears"

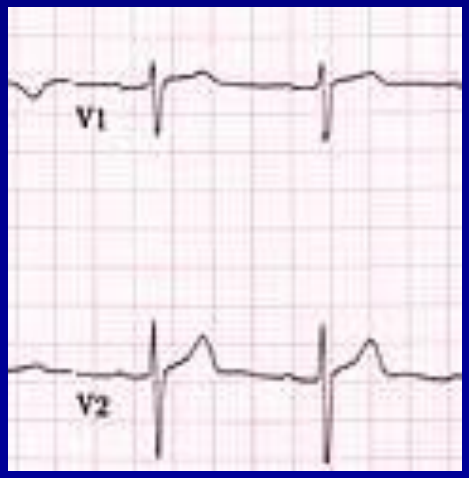


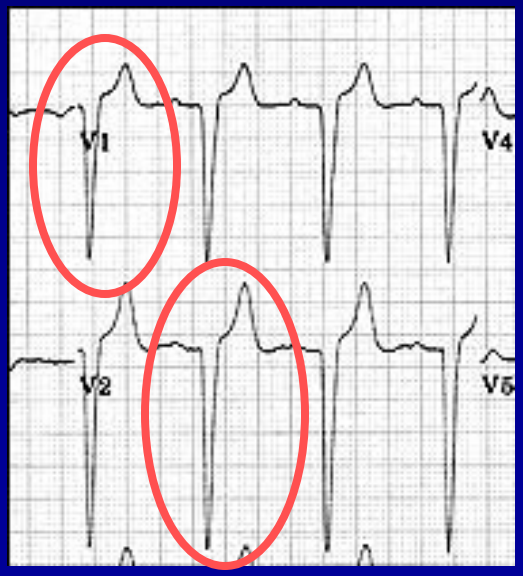
Left Bundle Branch Blocks

What QRS morphology is characteristic?

For LBBB the wide QRS complex assumes a characteristic change in shape in those leads opposite the left ventricle (right ventricular leads - V_1 and V_2).

Normal





Broad, deep S waves

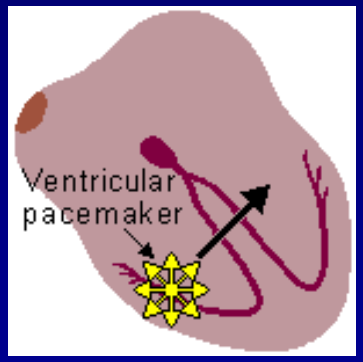
Supraventricular and Ventricular Arrhythmias

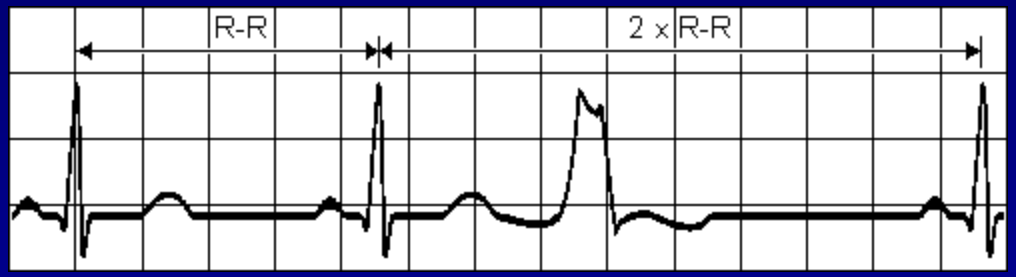
Arrhythmias

- Sinus Rhythms
- Premature Beats
- Supraventricular Arrhythmias
- Ventricular Arrhythmias
- AV Junctional Blocks

PREMATURE VENTRICULAR CONTRACTION (PVC)

- A single impulse originates at right ventricle



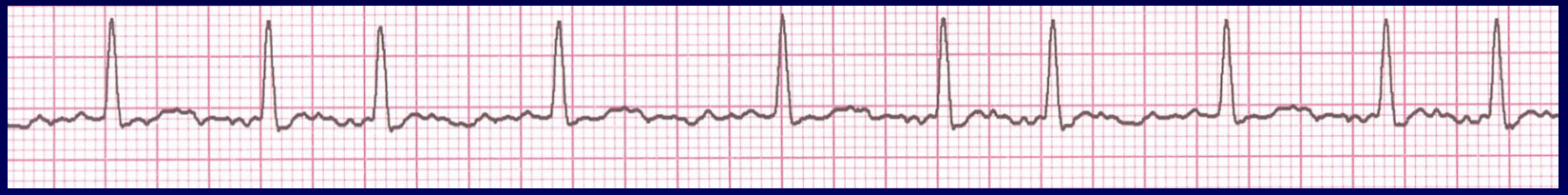


- Time interval between normal R peaks is a multiple of R-R intervals

Supraventricular Arrhythmias

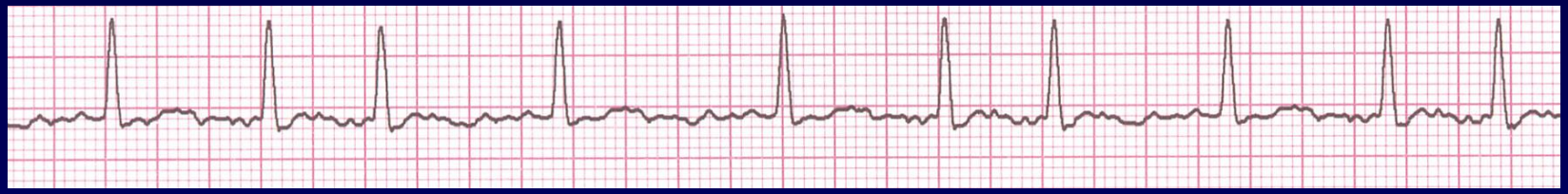
- *Atrial Fibrillation*
- *Atrial Flutter*
- *Paroxysmal Supraventricular Tachycardia*

Atrial Fibrillation



- Deviation from NSR
  - No organized atrial depolarization, so no normal P waves (impulses are not originating from the sinus node).
  - Atrial activity is chaotic (resulting in an irregularly irregular rate).
  - Common, affects 2-4%, up to 5-10% if > 80 years old

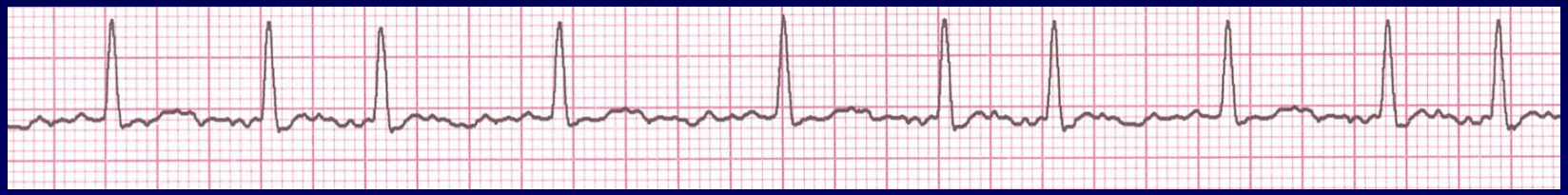
Atrial Fibrillation



- Etiology: Recent theories suggest that it is due to multiple re-entrant wavelets conducted between the R & L atria. Either way, impulses are formed in a totally unpredictable fashion. The AV node allows some of the impulses to pass through at variable intervals (so rhythm is irregularly irregular).

ATRIAL FIBRILLATION
Impuses have chaotic, random pathways in atria

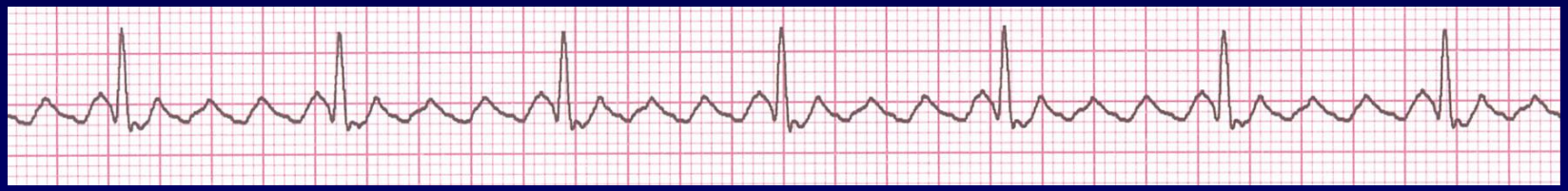
Atrial Fibrillation



- Rate? 100 bpm
- Regularity? irregularly irregular
- P waves? none
- PR interval? none
- QRS duration? 0.06 s

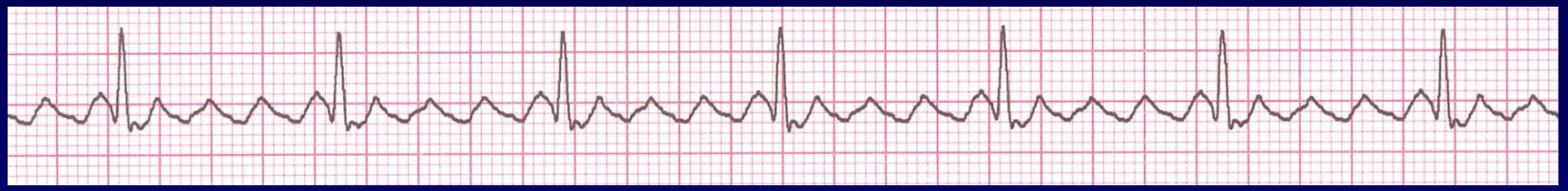
Interpretation? *Atrial Fibrillation*

Atrial Flutter



- Deviation from NSR
  - No P waves. Instead flutter waves (note “sawtooth” pattern) are formed at a rate of 250 - 350 bpm.
  - Only some impulses conduct through the AV node (usually every other impulse).

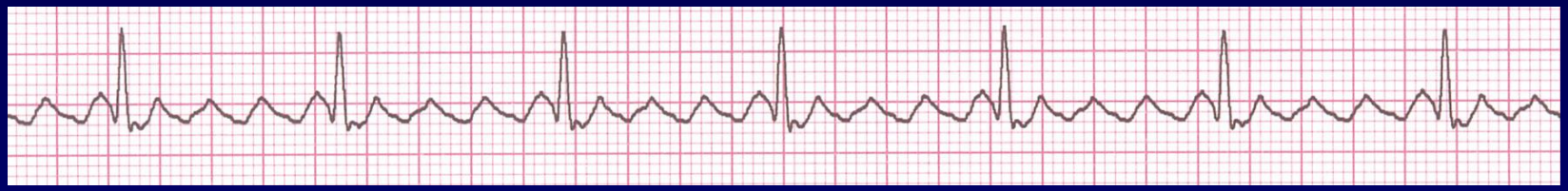
Atrial Flutter



- Etiology: Reentrant pathway in the right atrium with every 2nd, 3rd or 4th impulse generating a QRS (others are blocked in the AV node as the node repolarizes).

ATRIAL FLUTTER
Impulses travel in circular course in atria –

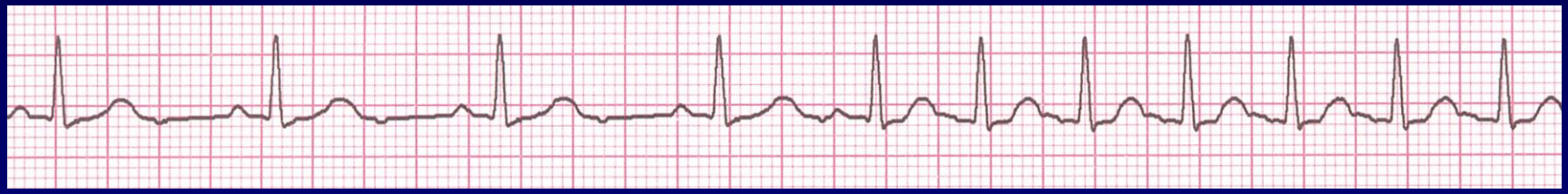
Atrial Flutter



- Rate? 70 bpm
- Regularity? regular
- P waves? flutter waves
- PR interval? none
- QRS duration? 0.06 s

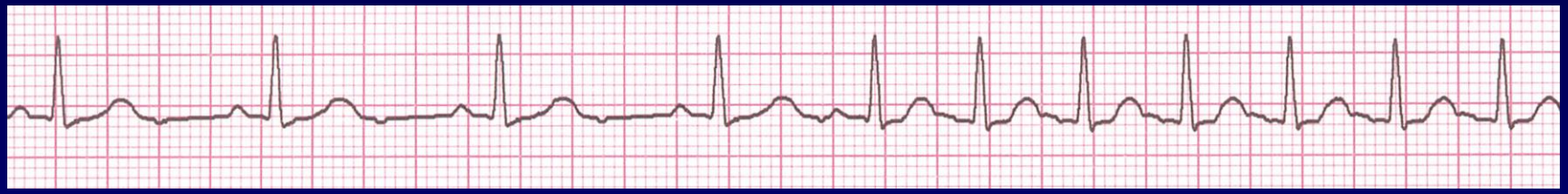
Interpretation? *Atrial Flutter*

PSVT - *Paroxysmal Supraventricular Tachycardia*



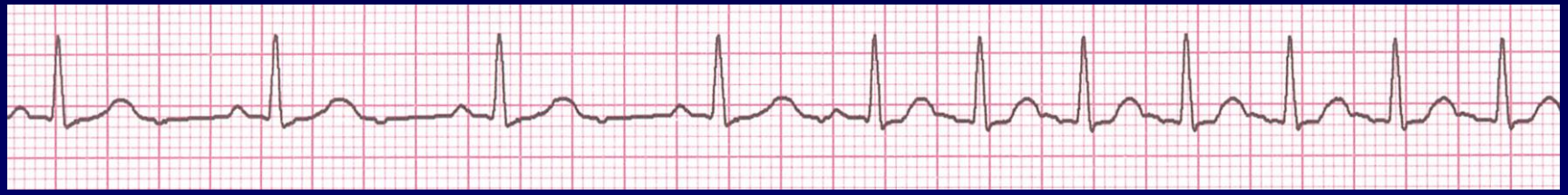
- Deviation from NSR
  - The heart rate suddenly speeds up, often triggered by a PAC (not seen here) and the P waves are lost.

PSVT



- Etiology: There are several types of PSVT but all originate above the ventricles (therefore the QRS is narrow).
- Most common: abnormal conduction in the AV node (reentrant circuit looping in the AV node).

Paroxysmal Supraventricular Tachycardia (PSVT)



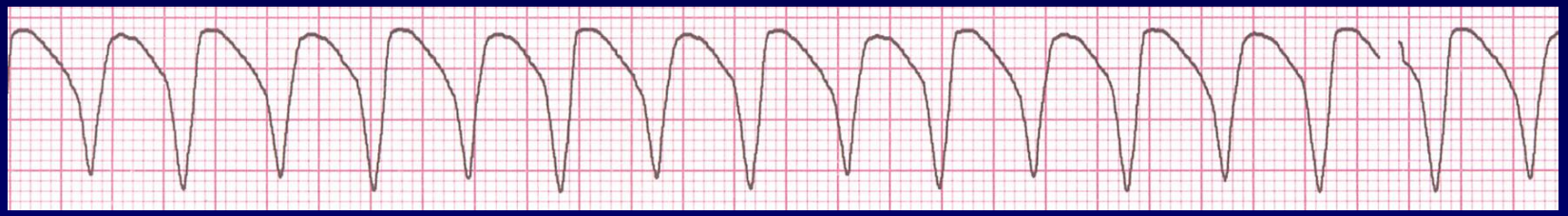
- Rate? 74 → 148 bpm
- Regularity? Regular → regular
- P waves? Normal → none
- PR interval? 0.16 s → none
- QRS duration? 0.08 s

Interpretation? *Paroxysmal Supraventricular Tachycardia (PSVT)*

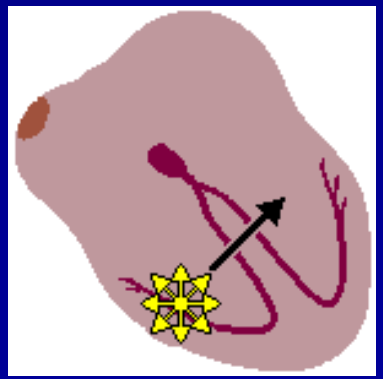
Ventricular Arrhythmias

- *Ventricular Tachycardia*
- *Ventricular Fibrillation*

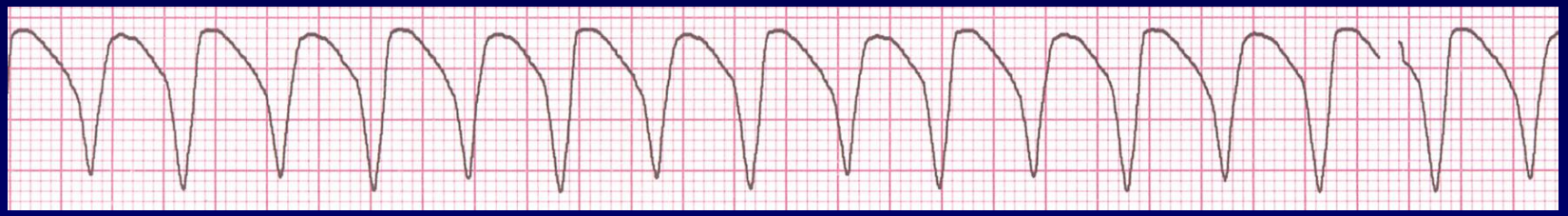
Ventricular Tachycardia



- Deviation from NSR
  - Impulse is originating in the ventricles (no P waves, wide QRS).



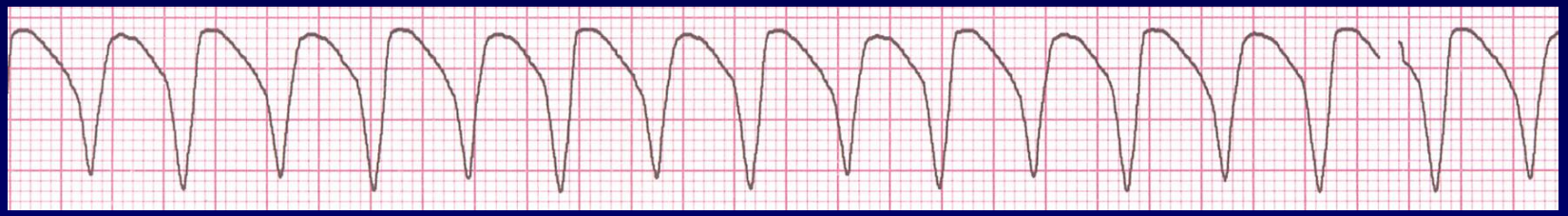
Ventricular Tachycardia



- Etiology: There is a re-entrant pathway looping in a ventricle (most common cause).

- Ventricular tachycardia can sometimes generate enough cardiac output to produce a pulse; at other times no pulse can be felt.

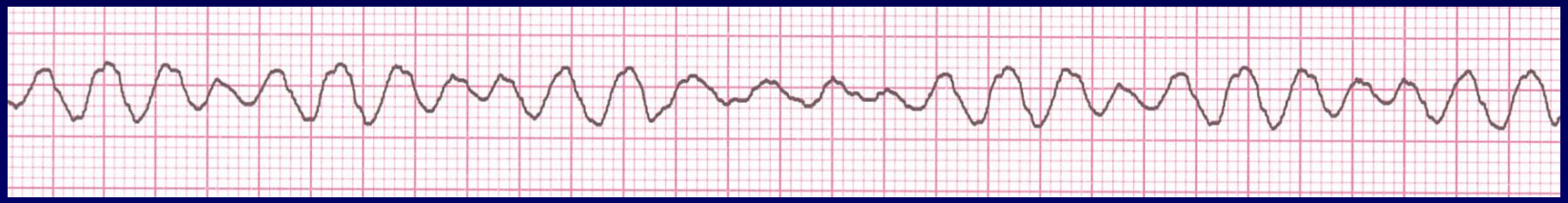
Ventricular Tachycardia



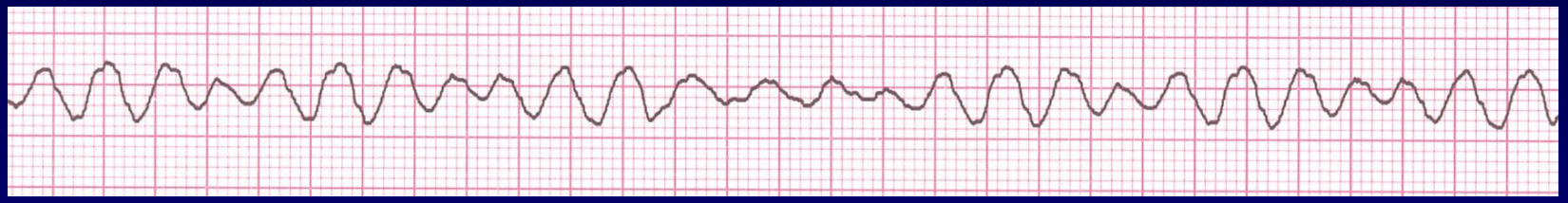
- Rate? 160 bpm
- Regularity? regular
- P waves? none
- PR interval? none
- QRS duration? wide (> 0.12 sec)

Interpretation? *Ventricular Tachycardia*

Ventricular Fibrillation



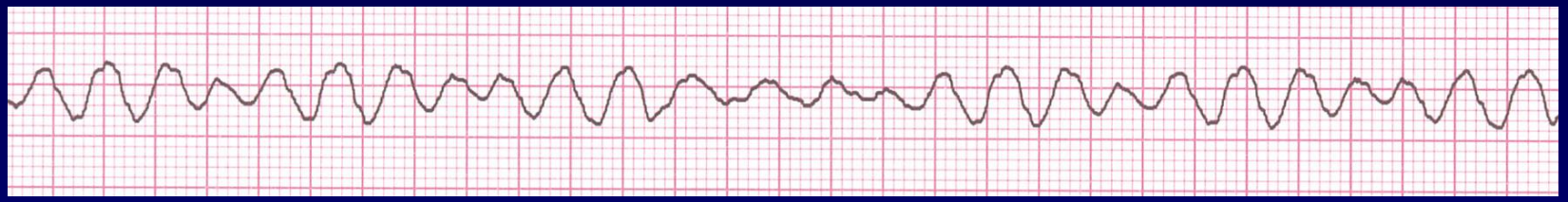
Ventricular Fibrillation



- Etiology: The ventricular cells are excitable and depolarizing randomly.

- Rapid drop in cardiac output and death occurs if not quickly reversed

Ventricular Fibrillation



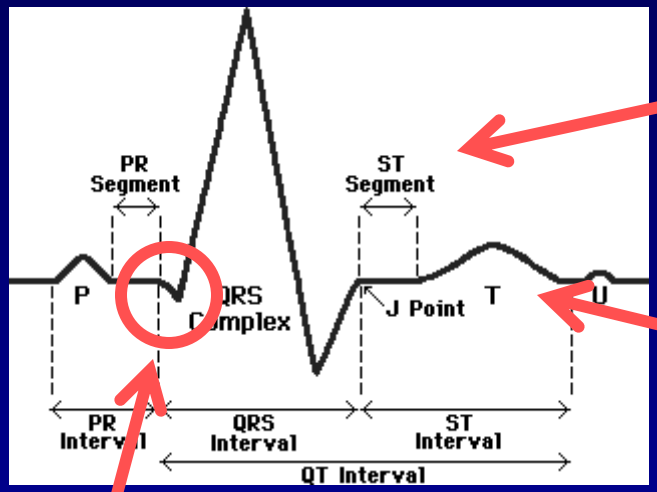
- Rate? none
- Regularity? irregularly irreg.
- P waves? none
- PR interval? none
- QRS duration? wide, if recognizable

Interpretation? *Ventricular Fibrillation*

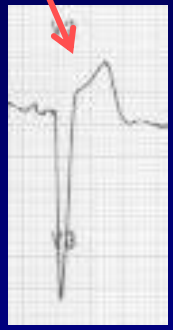
ST Elevation
and
non-ST Elevation MIs

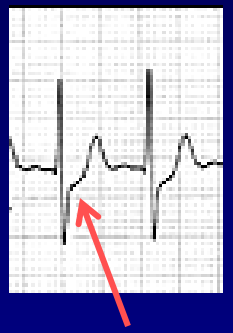
ECG Changes

Ways the ECG can change include:



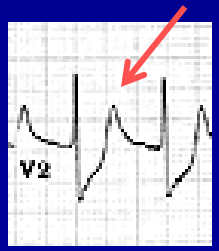
ST elevation & depression



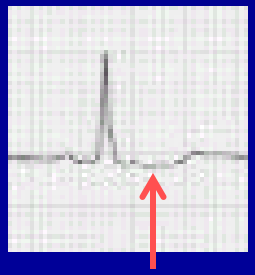


T-waves

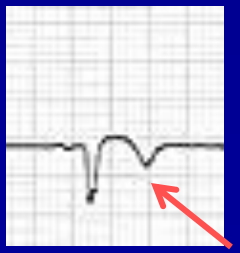
peaked



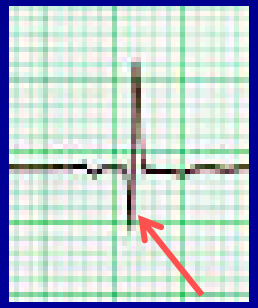
flattened



inverted

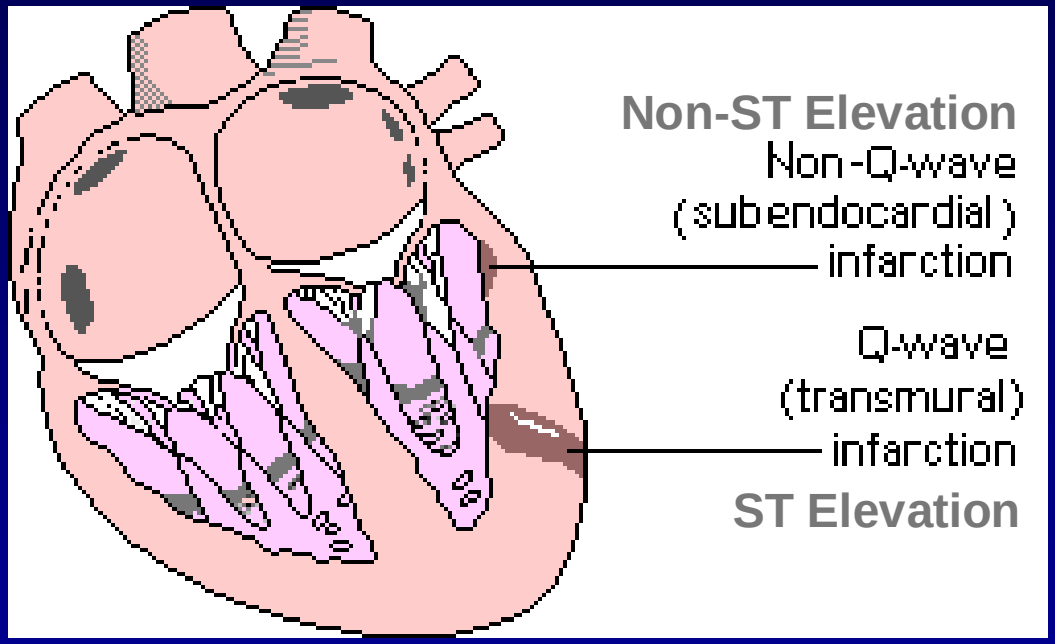


Appearance of pathologic Q-waves



ECG Changes & the Evolving MI

There are two distinct patterns of ECG change depending if the infarction is:



- ST Elevation (Transmural or Q-wave), or
- Non-ST Elevation (Subendocardial or non-Q-wave)

ST Elevation Infarction