HISTORY

- 1842
  - Italian scientist Carlo Matteucci realizes that electricity is associated with the heart beat
- 1876
  - French physiologist Etienne-Jules Marey analyzed the electric pattern of a frog's heart
- 1895
  - Dutch physician and physiologist Willem Einthoven was credited for the invention of EKG and later received the Nobel Prize in Physiology and Medicine in 1924
- 1906
  - Using the string electrometer EKG, Willem Einthoven diagnosed some heart problems

WHAT IS AN ECG?

- A recording of the electrical potentials generated by electrical currents from the heart into the adjacent tissues, which are detected by electrodes placed on the surface of the body.
- ECGs help in the diagnosis of cardiac and noncardiac disorders:
  - Arrhythmias and Conduction Blocks
  - Myocardial ischemia and infarction
  - Chamber hypertrophy
  - Pericarditis
  - Pericardial effusion/tamponade
  - Electrolyte disturbances
  - Drug toxicity

ACTION POTENTIAL, ELECTRICAL STIMULATION, AND CARDIAC CONTRACTION

- **Action Potential:** Depolarization and Repolarization phases of the cardiac myocyte membrane
  - It will cause electrical stimulation, which will then be the ultimate cause of cardiac contraction.
  - What is recorded in the ECG is the depolarization and repolarization sequence (it is not contraction but action potential).

The ECG in relation to the Cardiac Cycle

Big Picture First

- The ECG records **electrical activity** of the heart.
- The cardiac cycle describes **mechanical activity** (contraction and relaxation).

→ Electrical events trigger mechanical events.

- So on the timeline: **ECG change → short delay → muscle contracts or relaxes**
- Think of electricity as the **spark**, and contraction as the **engine movement**.

One Complete Cardiac Cycle – Imagine one heartbeat as a loop:
The atria fill with blood → atria contract → ventricles fill → ventricles contract → blood is ejected → ventricles relax → filling starts again.
Now we layer the ECG on top of that story.

P WAVE → ATRIAL DEPOLARIZATION → ATRIAL CONTRACTION

What the P wave is

- The P wave represents **depolarization of atrial muscle fibers**.
- Depolarization = sodium entering atrial cells → membrane potential becomes less negative → cells become electrically active.

What happens mechanically after

- A short moment after the P wave begins, the atria **contract**.
- This contraction is called **atrial systole**.
- Atrial systole pushes the last bit of blood into the ventricles — about **15–20% of ventricular filling** (called the *atrial kick*).

Simple chain
P wave
→ atrial depolarization
→ atrial contraction
→ final ventricular filling

NOTE: If P waves disappear (e.g., atrial fibrillation), ventricular filling decreases → ↓ cardiac output, especially in elderly or stiff ventricles.

PR Interval → AV Nodal Delay

- The PR interval is measured from the **start of the P wave** to the **start of the QRS complex**
- It represents:
- Time for the impulse to travel SA node → atria → AV node → His-Purkinje system
- Why the delay exists – The AV node conducts **slowly**. This delay allows:
  - ✔ Atria to finish contracting
  - ✔ Ventricles to finish filling
    → Before ventricles contract.
- Clinical meaning
  - Normal PR interval: 0.12–0.20 seconds
  - Prolonged PR = first-degree AV block.

QRS Complex → Ventricular Depolarization → Ventricular Contraction

What the QRS complex is

- Represents **rapid depolarization of both ventricles**.
- Large amplitude because ventricular muscle mass is large.
- Atrial repolarization happens here too, but it's hidden inside QRS.

What happens mechanically after

- Shortly after QRS begins:
- Ventricles start to **contract** → ventricular systole begins.
- This creates a rapid rise in ventricular pressure.

Two phases follow:

1. **Isovolumetric contraction –** Valves closed, pressure rising

2. **Ejection phase –** Semilunar valves open, blood exits heart

QRS
→ ventricular depolarization
→ ventricular contraction
→ blood ejection

NOTE: Wide QRS = abnormal ventricular conduction (bundle branch block, ventricular rhythm).

ST Segment → Ventricles Fully Depolarized → Plateau Phase

- The ST segment is the flat line after QRS.

- It represents the time when ventricular muscle cells are:

Uniformly depolarized
This corresponds to the **plateau phase (phase 2)** of ventricular action potentials.

Mechanical meaning
Ventricles are **actively contracting and ejecting blood** during ST segment.

NOTE:
- ST segment ≈ **middle of ventricular systole**
- CLINICAL NOTE: ST elevation or depression = myocardial ischemia or injury.

T Wave → Ventricular Repolarization → Ventricular Relaxation

What T wave is
- Represents **repolarization of ventricular muscle**.
- Potassium exits cells → membrane potential becomes negative again.

What happens mechanically after
- Ventricular muscle **relaxes** → ventricular diastole begins.
- Pressure in ventricles falls.
- Once ventricular pressure drops below atrial pressure:
- AV valves open → ventricular filling starts.

Simple chain
T wave
→ ventricular repolarization
→ ventricular relaxation
→ filling phase

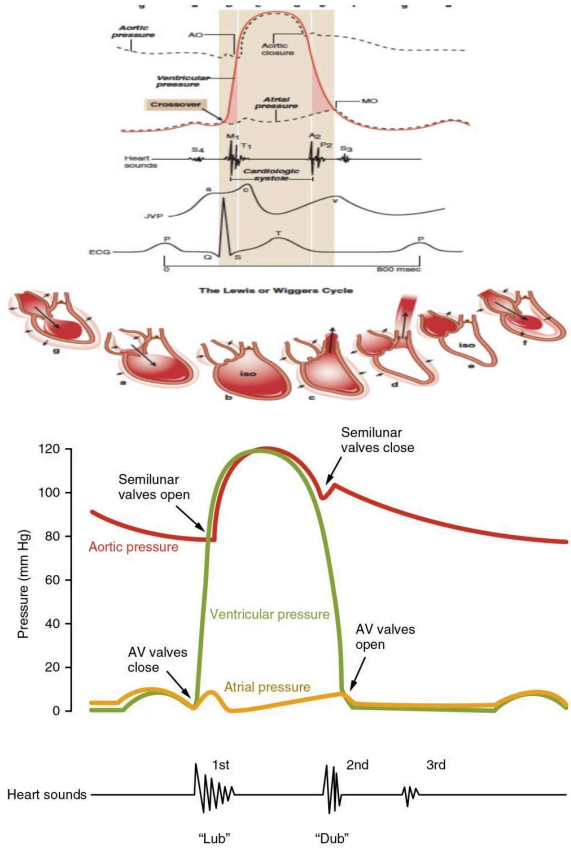
ECG Component	Electrical Event	Mechanical Event
P wave	Atrial depolarization	Atrial contraction
PR interval	AV nodal delay	Ventricles filling
QRS	Ventricular depolarization	Ventricular contraction
ST segment	Ventricles fully depolarized	Blood ejection
T wave	Ventricular repolarization	Ventricular relaxation

Key Principle (Very Testable): Electrical events always precede mechanical events. ***Never the other way around.***

If a question asks:
"What ECG wave occurs during atrial systole?"
Answer: **P wave (slightly before contraction)**

Relationship to Heart Sounds
- **S1 (lub)** occurs just after QRS
  → AV valves close at start of ventricular systole
- **S2 (dub)** occurs near end of T wave
  → Semilunar valves close at start of ventricular diastole

NOTE:
❌ Saying P wave = atrial contraction
✔ Correct: P wave = atrial depolarization
❌ Saying QRS = ventricular systole
✔ Correct: QRS = ventricular depolarization; systole follows
❌ Thinking T wave = diastole
✔ Correct: T wave = repolarization that leads into diastole



Major ECG Waveforms

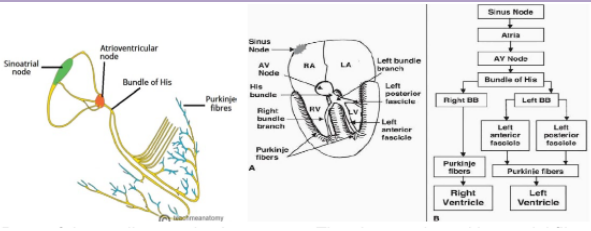
- P and QRS complex occur before major cardiac contractions
- P wave occur during atrial contraction
- QRS complex occur before ventricular contraction
- ST and T wave occur during the diastolic (filling) phases

The Cardiac Conduction System

- Generates electrical impulses to initiate rhythmical contraction of the heart muscle.
- Conducts these impulses rapidly through the heart.
- The heart as an organ is composed of cardiac myocytes (atrial and ventricular myocytes).
- There are specialized myocytes in the heart that function as the electrically conductive tissue of the heart.
- The heart beats itself independent of central nervous system from a few weeks gestation up to the entire life.
- The cause of this battery is the cardiac conduction system.

PARTS OF THE CARDIAC CONDUCTION SYSTEM

The sinus node and interatrial fibers will all converge into the AV node.



- In a normal heart, there should be no other electrical communication between the atria and the ventricles except through the AV node.

- ➔ If there are abnormal pathways communicating the atria to the ventricle, it is called a bypass tract. If it is a functional bypass tract, it will show abnormalities in the ECG.
- ➔ Any block anywhere in the conduction system will have characteristic ECG changes. Major blocks of the right and left bundle are called complete right bundle branch block (CRBBB) and complete left bundle branch block (CLBBB) respectively.
- ➔ From the AV node, it penetrates the interventricular septum as the penetrating bundle or bundle of His.
- ➔ It then divides into 2 major bundle branches, the right and the left branches, and then it will terminate just below the endocardium as the Purkinje fibers or Purkinje system.
- ➔ The left bundle branch before terminating as Purkinje fibers, it will divide into the left anterior and posterior fascicles.

SINOATRIAL NODE (SA NODE)

- The heart's natural pacemaker
- It discharges the fastest rate, at 60–100 beats per minute at rest.

AV NODE

- Receives impulse from SA Node
- Delivers impulse to the His-Purkinje System
- It discharges at 40–60 BPM if SA Node fails to deliver an impulse

BUNDLE OF HIS

- Begins conduction to the ventricles
- At AV junctional tissue
- It discharges at 40–60 beats/minute

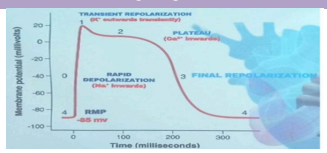
THE PURKINJE NETWORK

- Bundle Branches/Purkinje Fibers
- Moves the impulse through the ventricles for contraction
- Provides "escape rhythm"
- It discharges at 20–40 beats/minute
- In a cardiac arrest patient, for every second that the heart cannot be revived, there is successive ischemia of the cardiac conduction system. The AV nodes, bundle of His and Purkinje fibers will take over (ventricular escape rhythm), which is at a very slow rate.
  - The stroke volume is very dependent on the heart rate.
  - A heart rate that is dependent on ventricular escape rhythm at 20–40 bpm rarely produces a blood pressure that is enough to generate a perfusing blood pressure.

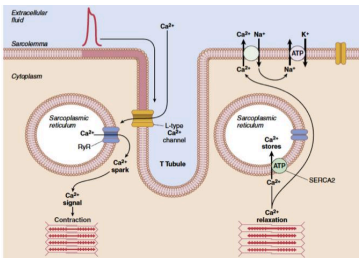
THE CARDIAC ACTION POTENTIALS

If electrodes are placed in the cardiac myocyte membranes, it will generate action potentials, especially if stimulated electrically.

The Ventricular Myocyte Action Potential



- It starts in phase 4, which is the **resting membrane potential** (**-85 mV**), which means -85 mV inside the membrane.
- In phase 0, it will shoot up and is called **rapid depolarization**.
  - ❖ It is caused by Na^+ moving inwards (the inside of the membrane from a very negative value, it will become positive, about 20 mV).
  - ❖ If it reaches that level, it will return back to negative transiently.
- In phase 1, it will return back to negative transiently and is called **transient repolarization**.
  - ❖ It is caused by K^+ going out of the cell, loosing positivity inside the
  - ❖ cell, but is only transient.
- In phase 2, the transient repolarization is offset by a **plateau** phase.
  - ❖ This is characteristic of a myocyte and is not seen anywhere else in the body (not in the skeletal, smooth muscle, or sinus node)
  - ❖ It is caused by slow Ca^{2+} movement towards the inside, that is why positivity is maintained for a few milliseconds.
  - ❖ The transport channel responsible for this is the **L-type calcium channels** which are characteristic of the ventricular myocytes.
- In phase 3 (**final repolarization**), it goes back to a more negative value at resting -85 mV
  - ❖ It is caused by K^+ going out of the cell.
- Anything that brings the potential back to the negative is usually K^+ going outwards.
- The usual ions causing membrane potentials to become more positive are Na^+ and Ca^{2+}.



Mechanism of excitation-contraction coupling and relaxation in cardiac muscle

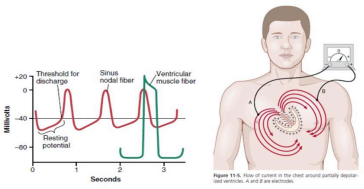
- In this depolarized membrane, this is ready to contract and then it will be propagated all throughout the myocardium before contraction would occur.

The Pacemaker Action Potential

- The **resting membrane potential** (phase 4) is much less negative at around -55 to -60 mV (-85 mV in cardiac myocytes)
  - ❖ The RMP does not rest and it slowly drifts upward (unlike in ventricular myocytes that it becomes isoelectric)
  - ❖ It is due to the **"funny" channels**. It is called as such because it is naturally leaky to Na^+ and Ca^{2+} and does not absolutely close.
    - ➢ It constantly leaks Na^+ out of the cell membrane such that constantly, the inside of the cell is gaining positivity, that is why the RMP drifts slowly towards the positive.
- When it reaches -40 mV, it is the threshold and there is nowhere to go but **rapid depolarization** (phase 0).
  - The rapid depolarization of the sinus node action potential is mainly secondary to Ca^{2+} going in because at this level, the "funny" sodium channels will close.
- There is no **transient repolarization** (phase 0) and **plateau** (phase 1) in the sinus node (unlike in cardiac myocytes).

- It goes to **final repolarization** (phase 3) right away.
  - ❖ It is due to K_+ going out of the cell (loosing positivity inside the cell)
- It then reaches baseline around -55 mV and then it slowly drifts again to -40 mV because of the "funny" sodium channels.
- It is the **pacemaker** because naturally it does not rest in the RMP but it slowly drifts upward, and then threshold, and then it fires.

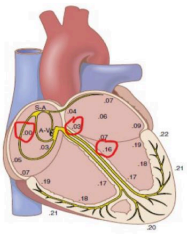
○ **Differences between Sinus-Nodal and Ventricular Muscle Action Potentials**



Flow of Electrical Currents in the Chest Around the Heart

- Current flows from negative to positive primarily in the direction from the base of the heart toward the **apex** during almost the entire cycle of depolarization.
- The waveform of depolarization starts at the base of the heart and then towards the apex.
- The average current flow occurs with negativity towards the base of the heart and with positivity toward the apex.

TRANSMISSION OF CARDIAC IMPULSE THROUGH THE HEART



The numbers indicate the time interval from the sinus node to the rest of the heart (the arrival of the electrical potential)

○ SA to AV = **0.03 second**
- Starting from 0.00. The **sinus node** is located in the junction of the SVC and the RA. Then electrical potential will travel to both **atria** and then it arrives in the **AV node** after 0.03 second.
- In normal hearts, it should be accurate to the millisecond. Any abnormality in the length of time will be visible in the ECG.

○ Within AV Node = **0.09 second**
- There is the **natural atrioventricular nodal delay**.
- When the impulse arrives, it has a 0.09 second delay which is normal because it is for proper atrioventricular synchrony. The atria are not passing chambers and they also contract. When the atria contracts, the ventricles has to relax so that there is proper flow of blood. When the ventricles contract, the atria has to relax.

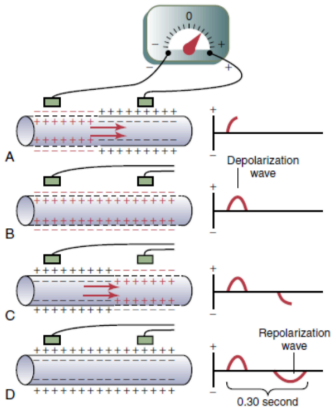
○ AV Node to Penetrating bundle (Bundle of His) = **0.04 second**
- There is an additional 0.04 seconds delay in the bundle of His

○ SA to Penetrating bundle = **0.16 second**

FUNDAMENTAL CARDIAC ELECTROPHYSIOLOGY PRINCIPLES

- **Transmembrane ionic currents** are generated by ion fluxes across the cellular membrane and between adjacent cells.
- These currents are synchronized by cardiac activation and recovery sequences to generate a **cardiac electrical field** in and around the heart that varies with time during the cardiac cycle.
- This electrical field passes through numerous other structures, including the lungs, blood, and skeletal muscle, that perturb the cardiac electrical field.
- The currents reaching the skin are then detected by electrodes placed in specific locations on the extremities and torso that are configured to produce leads, representing the difference in potentials sensed by pairs of electrodes or electrode combinations.
- The outputs of these leads are amplified, filtered, and displayed, using a variety of devices, to produce an ECG recording

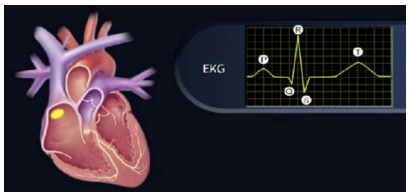
○ **Depolarization vs. Repolarization Waves**



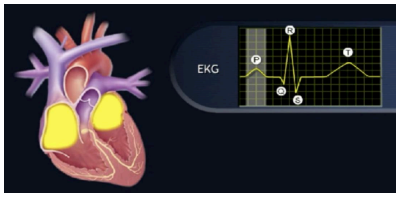
- Electrical impulse that travels toward the positive electrode produces an upright ("positive") deflection.
- Depolarization is the inside of the cell is going positive.
  - In an ECG lead, a lead has a positive input and a negative input.
  - If the depolarization wavefront is going to the positive input of that lead, it will record a positive deflection in the ECG (an upward wave).
  - The reverse is true with repolarization.

THE NORMAL ECG WAVEFORMS

Impulse Generation in the Sinus Node

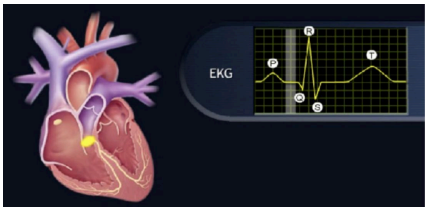


Atrial Depolarization



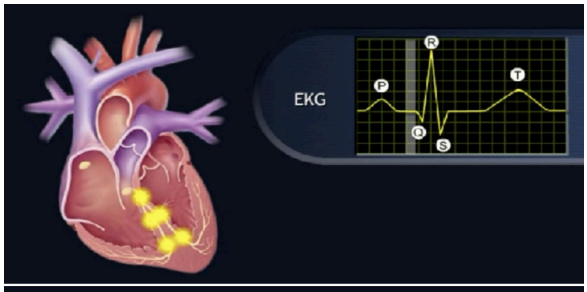
- P wave represents atrial depolarization, the left and right.
  - Mainly the left because ECG mainly records the left because everything in the right is overshadowed by the left (there is more electricity and muscles in the left)

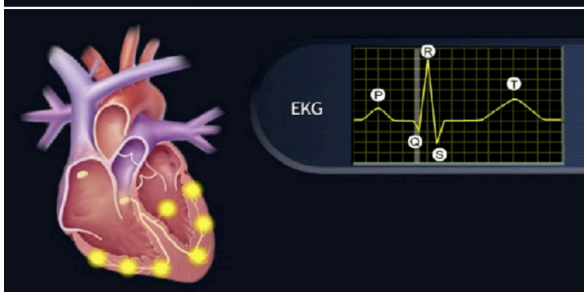
Atrioventricular (AV) Delay



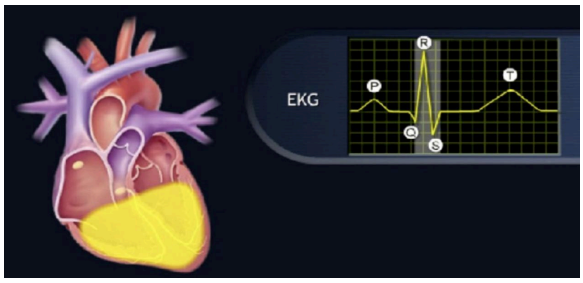
- PR interval is the interval between the P and the QRS. This is the normal AV delay.
    - The AV node is discharging at 0.30 millisecond (for example) from the normal 0.09 second (or 0.16 if including the penetrating bundle), there is more delay than usual in the AV node, the PR interval will prolong because the PR interval represents the AV node.

Conduction through the Bundle Branches and Ventricular Septum



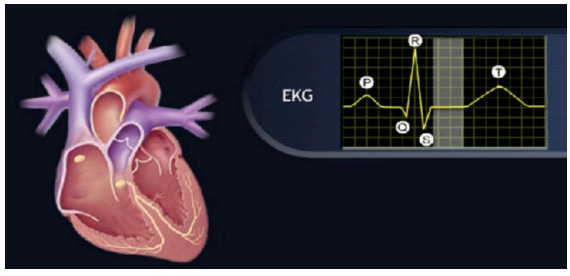


Ventricular Depolarization



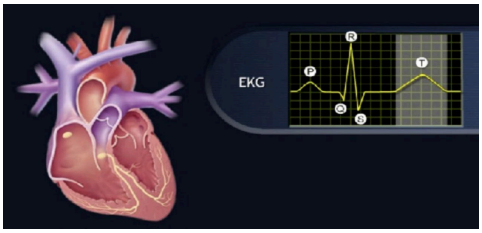
- QRS complex represents ventricular depolarization.
    - The early part of the QRS is the septal but the entire QRS is the depolarization of the ventricles.

Plateau Phase



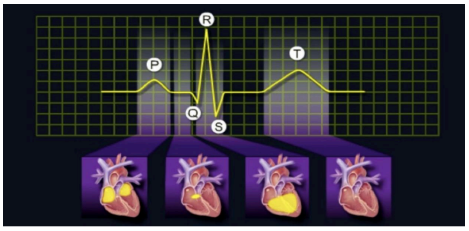
    - ST segment is the plateau (phase 2)

Final Rapid (Phase 3) Repolarization



- T wave is the rapid repolarization (phase 3)

The Major ECG Waveforms



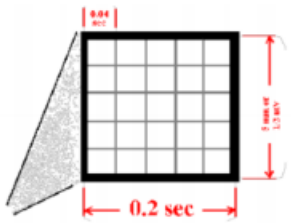
- ECG waveforms are P, QRS, and T.
- Some ECG will produce a U but is specific for a certain disorder.

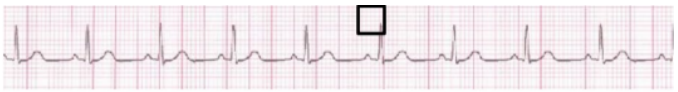
Summary:

- P wave- atrial depolarization
- PR interval- AV node
- QRS complex- ventricular depolarization
- ST segment- plateau
- T wave- repolarization

THE STANDARD ELECTROCARDIOGRAPHIC RECORDING (ECG PAPER)

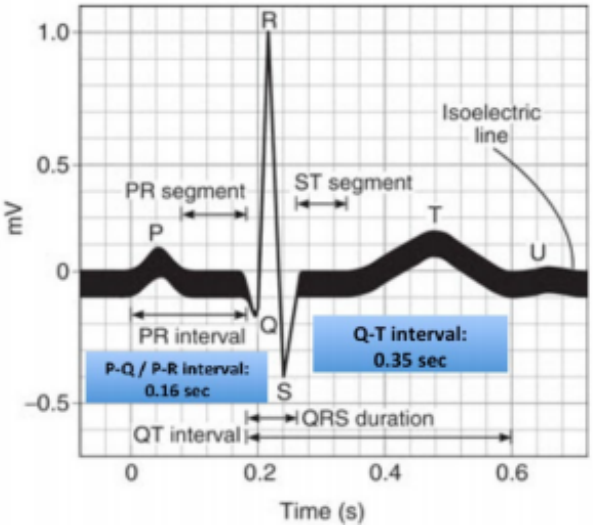
- In the ECG, there are **dark** lines and **light** lines.
- **Voltage and Time Calibration of the ECG**
    - **Horizontally** (time)
        - One small box is **0.04 second**
        - One large box is **0.20 second**
    - **Vertically** (Amplitude)
        - One small box is **0.1 mV**
        - One large box is **0.5 mV**



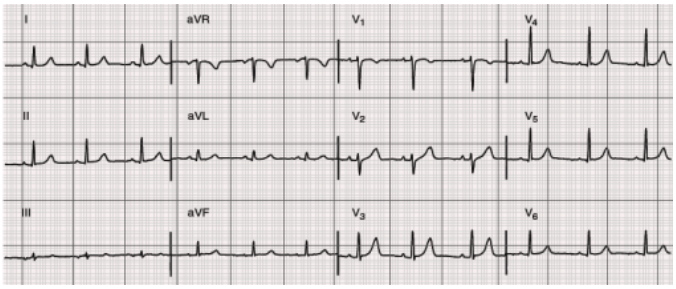


THE NORMAL ECG WAVEFORMS AND INTERVALS

- Normal P-R interval is **0.16 second**, but the range is up to 0.2 seconds, beyond 0.2 (>0.18) is already abnormal.
- Normal Q-T interval is **0.35 second** (represents the entire sequence of ventricular depolarization, plateau, and repolarization)



The Normal ECG



ECG Intervals

Intervals	Normal Durations (second, s) Average	Range	Events in the Heart during Interval
PR interval*	0.18[b]	0.12–0.20	Atrioventricular conduction
QRS duration	0.08	to 0.10	Ventricular depolarization
QT interval	0.40[c]	to 0.43	Ventricular action potential
ST interval (QT minus QRS)	0.32	...	Plateau portion of the ventricular action potential

THESTANDARD ELECTROCARDIOGRAPHIC LEADS

- Measure the difference in electrical potential between two points.
- Two Types
  - **Bipolar Leads**: Two different points in the body (positive and negative input)
    - There are 3 Bipolar Leads
  - **Unipolar Leads**: One point in the body (the positive input) and a virtual reference point with zero electrical potential located in the center of the heart.
    - The negative end is determined by the machine, called the negative (in augmented leads) or Wilson Central Terminal (in precordial leads)
    - 3 Unipolar Augmented Leads
    - 6 Precordial leads
- The Standard ECG recording is taken from 12 standard leads:
  - **3 Bipolar Limb Lead**s

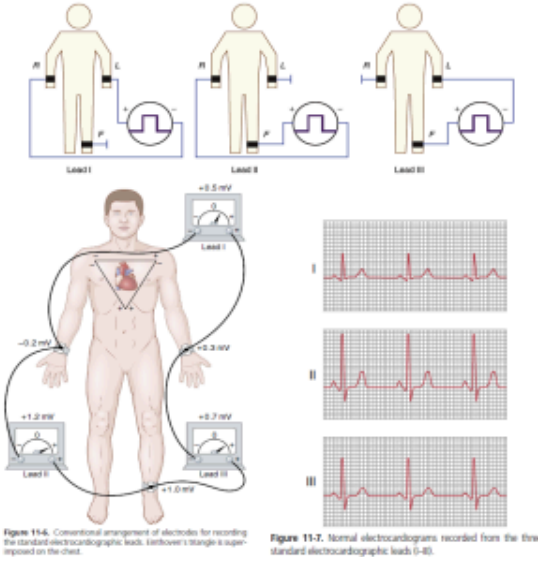


Figure 11-6. Conventional arrangement of electrodes for recording the standard electrocardiographic leads. Einthoven's triangle is superimposed on the chest.

Figure 11-7. Normal electrocardiograms recorded from the three standard electrocardiographic leads (I-III).

- Lead I- (-) right arm; (+) left arm (right to left arm)
- Lead II- (-) right arm; (+) left foot (right arm to left foot)
- Lead III- (-) left arm; (+) left foot (left arm to left foot)
- The **vector** is the direction of depolarization of each lead.
- All of the limb leads are **positive deflections** (above the baseline) because the normal depolarization vector in the normal heart goes from left to right and from up to down. The vectors in the bipolar leads run in parallel.

3 AUGMENTED UNIPOLAR LIMB LEADS:

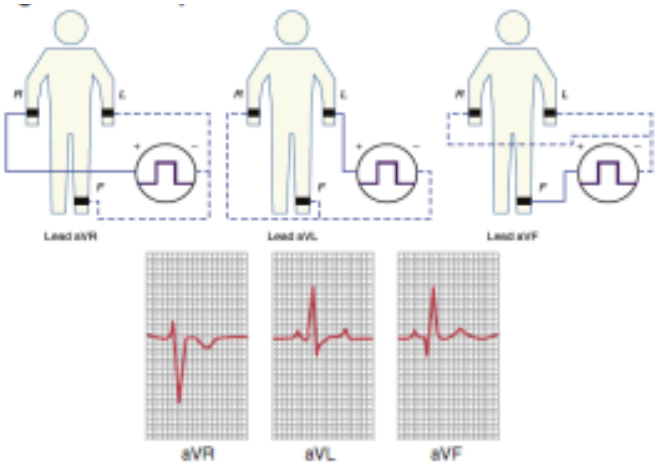
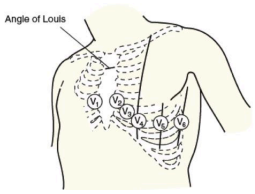


Figure 11-10. Normal electrocardiograms recorded from the three augmented unipolar limb leads.

- The negative input is determined by the machine (extremities that are not used) so only the positive inputs will be placed.
- Nothing will be placed on the right leg (only ground electrode)
  - **Lead aVR**- (+) right arm; (-) combination of the left arm and left leg
  - **Lead aVL**- (+) left arm; (-) combination of the right arm and left leg
  - **Lead aVF**- (+) left foot; (-) combination of the left arm and right arm.
- Lead aVR has the **negative deflections** because the vector in the aVR goes into the right arm and since the normal depolarization rate wavefronts in the normal heart has not wavefronts going to the right arm (exactly opposite the normal depolarization wavefronts in a normal heart)

- Unless if the heart is in the right chest (dextrocardia or wrongly placed leads), the aVR should register negative waveforms.
- 6 **Precordial (Chest)** Leads



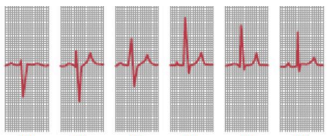
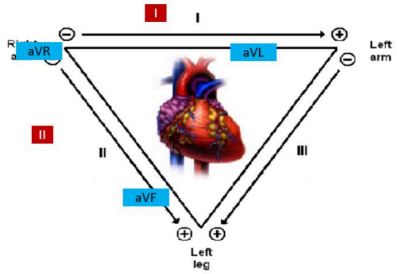
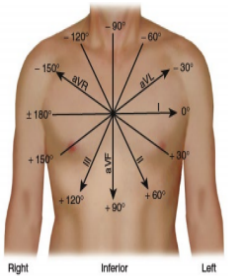


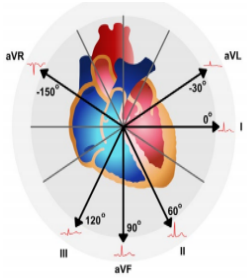
Figure 11-9. Normal electrocardiograms recorded from the six standard chest leads.

- **The Einthoven's Triangle**



  - An imaginary triangle drawn around the area of the heart with the 2 arms and the leg forming the apices of the triangles.
  - It illustrates that the 2 arms and the left leg form apices of a triangle surrounding the heart.
  - Represent the points at which the two arms connect electrically with the fluids around the heart and the lower apex is the point at which the leg connects with the fluids.
- Limb Leads Axes (The Hexaxial System)

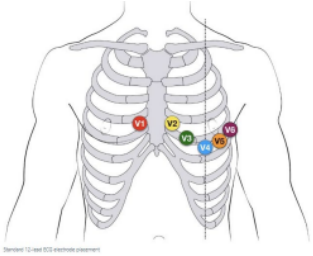


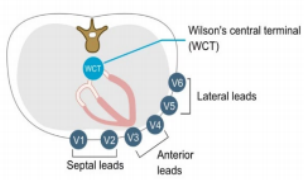


  - A pair of electrodes (positive and negative) connected to the body on opposite sides of the heart, and the direction from negative to positive is the axis or vectors.
  - Composed of the 3 bipolar leads and 3 augmented unipolar leads.

Lead I	going to 0°	**aVL**	going to -30°
Lead II	going to +60°	**aVF**	going to +90°
Lead III	going to +120°	**aVR**	going to -150°

- Precordial **(Chest)** leads

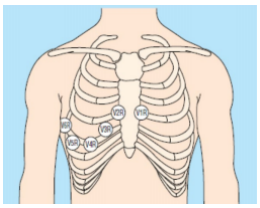


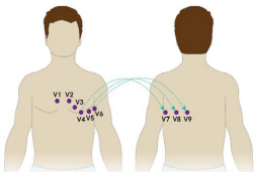


- **V1**- 4th right ICS parasternal (close to the sternum)
- **V2**- 4th left ICS parasternal (opposite to V1)o
- **V4**- 5th left ICS midclavicular line
- **V3**- between V4 and V2 (approximately)o
- **V5**- 5th left ICS anterior axillary line
- **V6**- 5th left ICS midaxillary line

V1 and V2 represents right ventricle; V3-V6 represents left ventricle

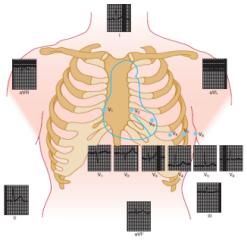
- **Right-sided and Posterior Chest Leads (Non-standard)**





  - The chest leads can be extended to the right when dealing with right ventricular infarction which is represented in the standard ECG.
  - The chest leads can also be extended to the back.

- **12-Lead ECG Recordings**



THE CONTIGUOUS LEAD GROUPINGS

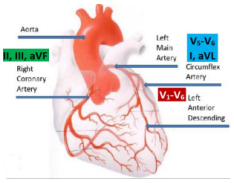
Leads are grouped together according to the general area of the heart (the Left Ventricle) that they represent.

- **Limb Leads** generally view the heart in a Supero-Inferior and MedioLateral dimension
- **Precordial Leads (Chest Leads)** generally view the heart (Left Ventricle) in the Antero-Posterior dimension

- **Arrangement of Leads on the ECG**

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 Based on Contiguous Lead Groupings**
  - **Septum**- When examining a suspected pathology involving the ventricular septum, look at the V1 and V2.
  - **Anterior Wall (Anteroseptal)**- Look at V3 and V4 (V1 to V4 in some sources)
  - **Lateral Wall**- Look at V5, V6, Lead I (going to the left arm), aVL
  - **Inferior Wall**- Look at Lead II, Lead III, aVF (leads going down)
- **The Coronary Artery Distribution based on the Contiguous Lead Groupings**



THE CARDIAC ACTION POTENTIALS

Coronary Artery Territories and ECG Leads

- Left Anterior Descending (LAD) – supplying most of the anterior surface of the heart; V1–V6
- Circumflex Artery (LCX) – supplies the lateral portion of the heart; V5–V6, Lead I, aVL
- Right Coronary Artery (RCA) – supplies the inferior portion of the heart; Lead II, Lead III, aVF

SUMMARY:

- Anterior wall → LAD → V1–V4
- Lateral wall → LCX → I, aVL, V5–V6
- Inferior wall → RCA → II, III, aVF

Example:

- A patient has myocardial infarction in the ECG involving the anterior wall. The culprit vessel is the LAD.
- Since the pathology of MI is acute thrombosis in the coronary vessel, so a thrombus in the left anterior descending is suspected.
- The patient's artery will be opened and to save time, look at the ECG, the thrombus is probably in V1–V4, then canulate the LAD first.

VECTORIAL ANALYSIS AND DETERMINATION OF THE MEAN ELECTRICAL AXIS

THE QRS AXIS AND VECTOR

- The QRS axis represents the overall direction of the ventricles' electrical activity.
- Instantaneous mean vector is the summated vector of the generated potential.
- Abnormalities hint at:
  - Ventricular enlargement (RVH, LVH)
  - Conduction blocks (right bundle, left bundle)
  - Anatomic malposition of the heart

DIRECTION OF VECTOR

→ Denoted in terms of degrees

- Is the average direction of the QRS vector during the spread of depolarization wave through the ventricles.
- It is usually at +59° in a normal heart
- During most of the depolarization wave, the apex of the heart remains positive with respect to the base.
- The wave of depolarization is going towards the apex from base (negative to positive).

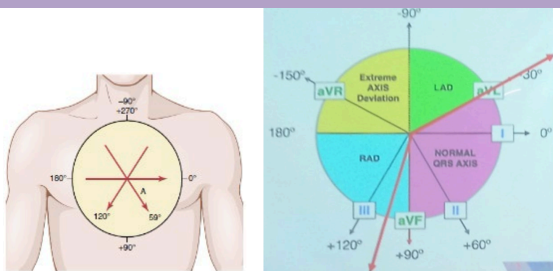
THE QRS AXIS

- Normal QRS Axis: range between -30° to +110° (mean of +59°)
- Left Axis Deviation (LAD): -30° to -90°
- Right Axis Deviation (RAD): +110° to +180°

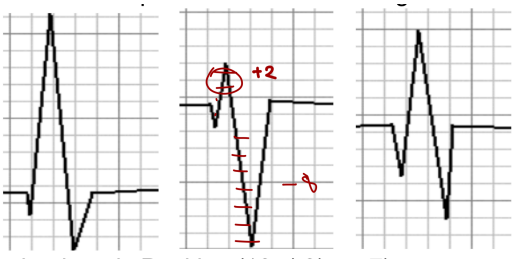
THE LIMB LEADS AND THE CARDIAC VECTOR OR AXIS

The mean QRS vector can be approximated by measuring the net differences between the positive and negative peaks of the QRS.

DETERMINING THE QRS AXIS



1. 1st: Determine the net QRS potential in 2 limb leads (Leads I and III or I and aVF)
2. 2nd: From the net QRS potential determined, you are now ready to plot

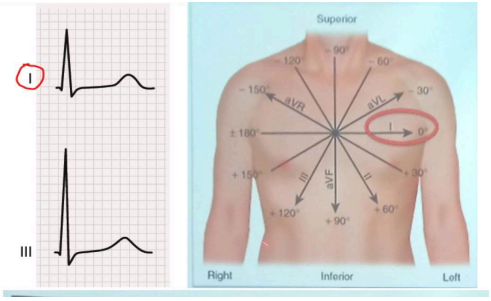


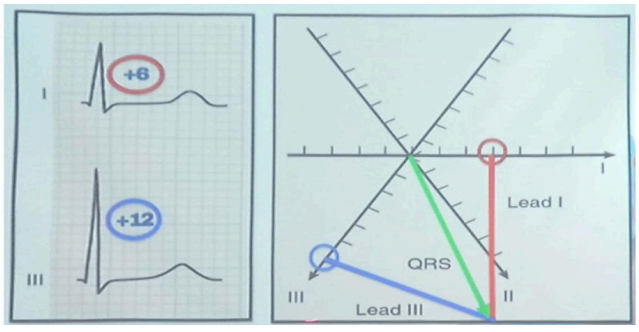
3. Determine the net QRS potential of the following QRS complexes:
   - 1st pic: Predominantly Positive (10+(-3) = +7)
   - 2nd pic: Predominantly Negative (2+(-8) = -6)
   - 3rd pic: Equiphasic (5+(-5) = 0)

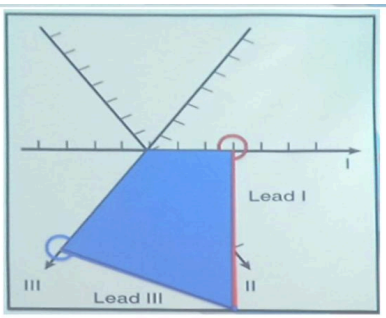
PLOTTING THE ELECTRICAL AXIS

Determine the net QRS potential of the following QRS complexes:

- The mean force during activation is represented by the area under the QRS waveforms, after plotting the vectors of 2 limb leads.
  - Look at the potential in the QRS in Leads I and III, plot and interpolate, and where they meet is the mean QRS potential.







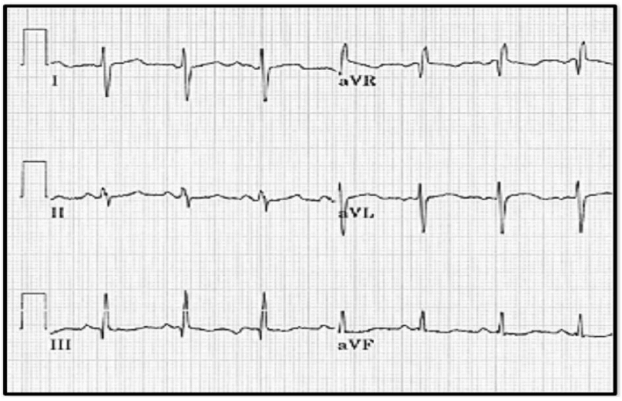
DETERMINING THE ELECTRICAL AXIS (SHORTCUT METHOD)

1. Examine the QRS complexes in leads I and aVF or III
2. Determine/Estimate the net potential of the QRS complexes (if they are predominantly positive or negative).
3. The combination should place the axis into one of the 4 quadrants below:

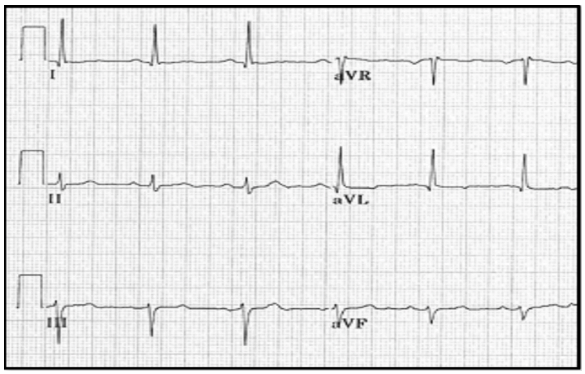
Lead I \ Lead aVF	Positive	Negative
Positive	Normal Axis	LAD
Negative	RAD	Indeterminate Axis

SHORTCUT (FOR ROUNDS PURPOSES)

- Left thumb is Lead I and right thumb is Lead III or aVF
- If positive QRS in that lead, point the thumb upward
- If negative QRS in that lead, point the thumb down
- If both thumbs are upward (both positive), it is a normal QRS axis
- If one is negative, whichever is the thumb that is remaining upwards, that is the direction of the axis.



Negative in I; Positive in aVF→ **Right Axis Deviation (RAD)**



Positive in I; Negative in aVF→ **Left Axis Deviation (LAD)**

I. PHYSIOLOGIC AXIS DEVIATIONS

- Due to changes in the position of the heart in the chest; does not indicate pathology.

A. Shift to the Left

- Occurs when the diaphragm moves upward, causing the heart to shift slightly leftward.
  - End of deep expiration: The diaphragm rises, uplifting the heart and shifting the axis to the left.
  - Supine position.
  - Obesity: Increased abdominal fat pushes the diaphragm upward.

B. Shift to the Right

- Occurs when the diaphragm moves downward, causing the heart to shift rightward.
  - End of deep inspiration: Lungs are fully expanded; the diaphragm contracts downward.
  - Standing position: Especially in patients who cannot lie flat and remain close to a 90° position.
  - Tall, lanky body habitus: The heart becomes elongated in a supero-inferior direction.

II. PATHOLOGIC AXIS DEVIATIONS

- Due to abnormal cardiac or ventricular conditions.

A. Change in the Position of the Heart

- **Dextrocardia:** The heart is located in the right side of the chest.

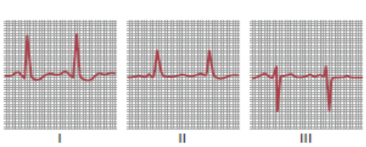
B. Ventricular Hypertrophy

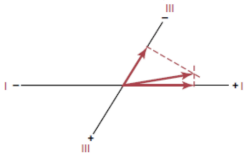
- Axis deviation occurs toward the hypertrophied ventricle because it has more myocardial mass and generates a stronger electrical potential.
  - **Left Ventricular Hypertrophy (LVH)**
  - **Right Ventricular Hypertrophy (RVH)**

C. Bundle Branch Blocks

- **Left Bundle Branch Block (LBBB)**
- **Right Bundle Branch Block (RBBB)**

III. LEFT AXIS DEVIATION RESULTING FROM LEFT VENTRICULAR HYPERTROPHY (LVH) COMMON CAUSES





Systemic hypertension

- In hypertension, there is a very elevated aortic pressure so the

ventricles will contract against higher pressure so it has to hypertrophy to overcome that pressure otherwise blood will not flow (The principle of blood flow is to create a significant pressure gradient. Without a pressure gradient, blood will not flow).
- The ventricle has to create a higher pressure to overcome the pressure in the aorta.

Aortic stenosis

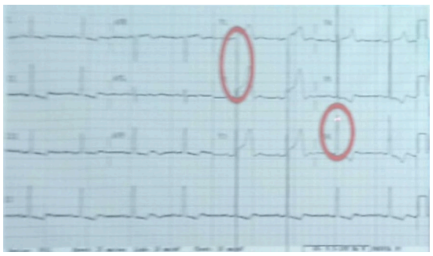
- There is obstruction in the aortic valve so there is a very small aortic opening, so the LV has to contract in a much forceful manner to overcome the obstruction

Other Causes

- **Aortic regurgitation**
- **Congenital heart diseases that causes LVH**
- **Left bundle branch blocks (LBBB)**

QRS Complexes in Left Ventricular Hypertrophy (LVH)

- Different criteria exist for the ECG diagnosis of LVH.
  - The **Sokolow-Lyon Criteria:**
    - R wave in V_5 or V_{56} + S wave in V_1 or V_2 >35mm



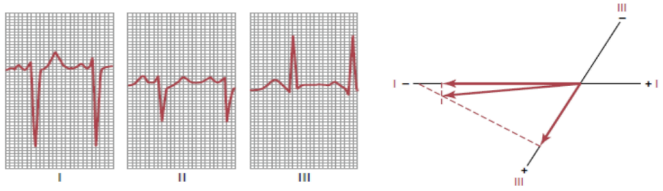
R in V_5 or V_6 and S in V_1 or V_2 when summed up is greater than 35

Common Diagnostic Criteria for Left Ventricular Hypertrophy (LVH)

MEASUREMENT	CRITERIA
Sokolow-Lyon voltages	$SV_1 + RV_5$ >3.5 mV
	RaVL >1.1 mV
Romhilt-Estes point score system*	Any limb lead R wave or S wave >2.0 mV (3 points)
	or SV_1 or SV_2 ≥3.0 mV (3 points)
	or RV_5 to RV_6 ≥3.0 mV (3 points)
	ST-T wave abnormality, no digitalis therapy (3 points)
	ST-T wave abnormality, digitalis therapy (1 point)
	Left atrial abnormality (3 points)
	Left axis deviation ≥–30 degrees (2 points)
	QRS duration ≥90 msec (1 point)
	Intrinsicoid deflection in V_5 or V_6 ≥50 msec (1 point)
Cornell voltage criteria	SV_3 + RaVL >2.8 mV (for men)
	SV_3 + RaVL >2.0 mV (for women)
Cornell regression equation	Risk of LVH = $1 / (1 + e^{-exp})$†
Cornell voltage duration measurement	QRS duration × Cornell voltage >2436 mm-sec‡
	QRS duration × sum of voltages in all leads >1742 mm-sec

LVH, Left ventricular hypertrophy; *PTF,* P terminal force; *PTFV_1,* P terminal force in lead V_1.

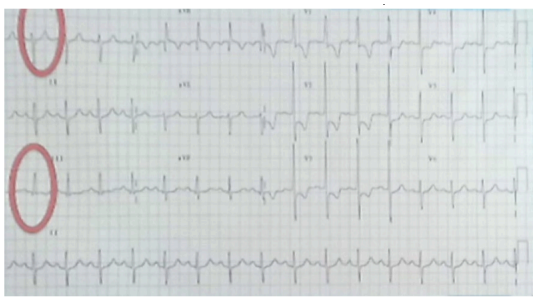
III. RIGHT AXIS DEVIATION RESULTING FROM RIGHT VENTRICULAR HYPERTROPHY (RVH) COMMON CAUSES



- **Pulmonary stenosis**
- **Conditions that result in RV volume or pressure overload:** COPD.
- **Congenital heart diseases that cause RVH:** Tetralogy of Fallot, Ventricular septal defects.
- **Right bundle branch defects (RBBB)**
  - Same principle, there is increased pressure in the right heart so that right heart should hypertrophy.

QRS Complexes in Right Ventricular Hypertrophy (RVH)

- **Criteria**: R axis deviation + R wave in V1 > 7 mm



Tall R in V_1 > 0.6 mV
Increased R/S in V_1 > 1
Deep S in V_5 > 1.0 mV
Deep S in V_6 > 0.3 mV
Tall R in aVR > 0.4 mV
Small S in V_1 < 0.2 mV
Small R in V_{5-6} < 0.3 mV
Reduced R/S ratio in V_5 < 0.75
Reduced R/S ratio in V_6 < 0.4
Reduced R/S in V_5 to R/S in V_1 < .04
$(R_I + S_{III}) - (S_I + R_{III})$ < 1.5 mV
Max R_{V1-2} + Max $S_{I, aVL} - S_{V1}$ > 0.6 mV
$RV_1 + S_{V5-6}$ > 1.05 mV
R peak V_1 > 0.035 msec
QR in V_1 present

IV. CONDITIONS THAT CAUSE ABNORMAL VOLTAGES OF THE QRS COMPLEX

- **Increased voltage:**
  - LVH
  - RVH
- **Decreased voltage:**
  - Cardiac myopathies (primary muscle disorders of the heart)
  - Abnormal conditions surrounding the heart:
    - Pericardial effusion (too much fluid in the pericardial sac)
    - Pleural effusion (too much fluid in the lungs)
    - Pulmonary emphysema (too much air in the lungs
    - What is recorded are electrical potentials from the heart projected to the skin. If there is bigger interference from the heart to the skin (too much fluid from the heart to the skin or too much air), the complexes might record very small.

IV. CONDITIONS THAT CAUSE BIZARRE QRS COMPLEX MORPHOLOGIES

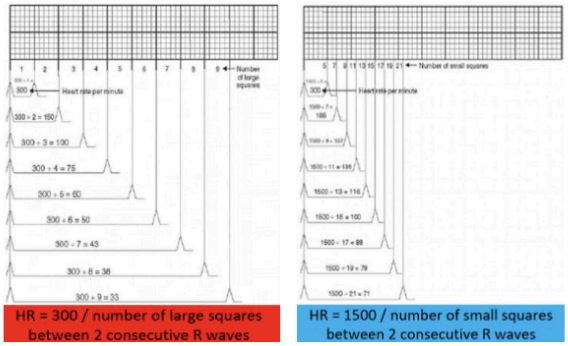
- **Cardiac hypertrophy or dilation** prolong the QRS complex.
- **Destruction of cardiac muscle** in various areas throughout the ventricular system, with replacement of this muscle by scar tissue.
- **Multiple blocks** in the Purkinje system.

ELECTROCARDIOGRAPHIC DETERMINATION OF HEART RATE

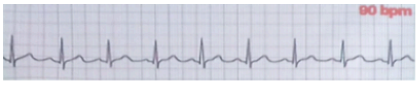
DETERMINING THE HEART RATE

- **Heart Rate** is the reciprocal of the time interval between two successive heartbeats.
  - If the interval between two beats as determined from the time calibration lines is **1 second**, the heart rate is **60 beats per minute**.
  - The normal interval between two successive QRS complexes in the adult person is about **0.83 second**.
    - HR of **60/0.83 times per minute**, or **72 beats per minute**.

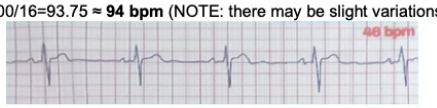
CALCULATING THE HEART RATE



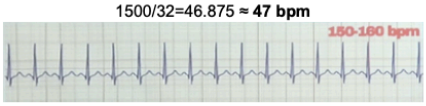
- If looking at the ECG, look at succeeding R waves. 1 R to the next R is one cardiac cycle. Count the number of squares between those 2.
- When using the big squares (dark lines), use 300 (300 divided by the number of big squares in between the R to the next R).
- When using the small squares, use 1,500 divided by the number of small squares from 1 R to the next R.



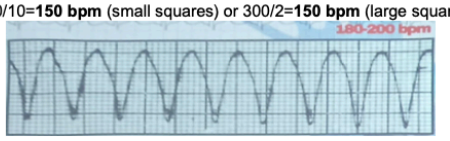
1500/16=93.75 ≈ **94 bpm** (NOTE: there may be slight variations)



1500/32=46.875 ≈ **47 bpm**



500/10=**150 bpm** (small squares) or 300/2=**150 bpm** (large squares)

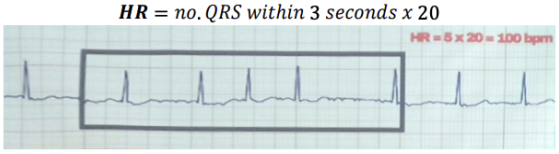


1500/8=187.5 bpm ≈ **188 bpm**

For Irregular Rhythms

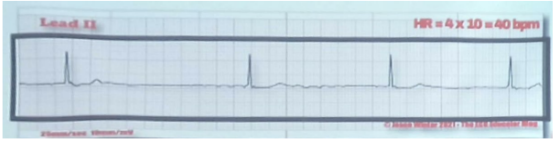
- Calculating HR using a **3-second rhythm strip:** Heart rate is calculated by counting the number of QRS complexes within the 3- second strip and multiplied by 20.
  - A 3-second rhythm strip has **15** big squares. Count the number of R included in that 3-second strip and multiply by 20.

$$HR = no.\,QRS\ within\ 3\ seconds\ x\ 20$$



- Calculating HR using a **6-second rhythm strip:**
  - A 6-second rhythm has **30** big squares. In a very slow irregular rhythm, use a longer strip.

$$HR = the\ no.\,QRS\ complexes\ x\ 10$$



- Calculating HR using a **12-second rhythm strip:**
  - A 12-second rhythm strip has **60** big squares.

$$HR = the\ no.\,QRS\ complexes\ x\ 5$$

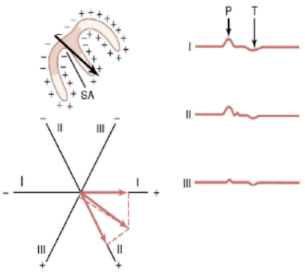
- **Shortcut Method**

No. of Big Boxes	Rate
1	300
2	150
3	100
4	75
5	60
6	50

ANALYSIS OF THE NORMAL ELECTROCARDIOGRAPHIC WAVEFORMS AND INTERVALS

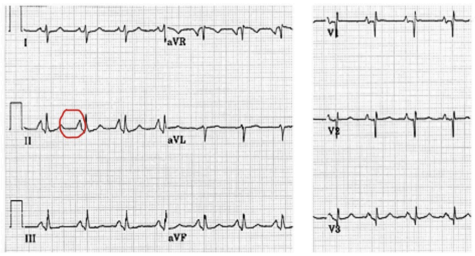
A. P WAVE (ATRIAL DEPOLARIZATION)

- The normal P wave is directed towards the left arm a bit inferiorly and is parallel with leads I, II, and III.
- Always **positive** in lead I and II
- Always **negative** in lead aVR
- **<3** small squares in duration
- **<2.5** small squares in amplitude
- Commonly biphasic in lead V_1
- Best seen in **lead II**



Right Atrial Enlargement

- Tall (**>2.5 mm**), pointed P waves: **P Pulmonale**



Left Atrial Enlargement

- Notched/bifid (M-shaped), Biphasic P waves in limb leads: **P Mitrale**

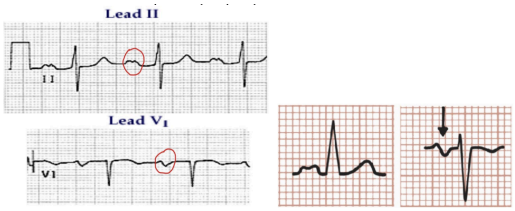


TABLE 14.3 Common Diagnostic Criteria for Left and Right Atrial Abnormalities

LEFT ATRIAL ABNORMALITY	RIGHT ATRIAL ABNORMALITY*
Prolonged P wave duration to >120 msec in lead II	Peaked P waves with amplitudes in lead II to >0.25 mV
Prominent notching of P wave, usually most obvious in lead II, with interval between notches of >40 msec	Prominent initial positivity in lead V_1 or V_2 >0.15 mV
Ratio between duration of P wave in lead II and duration of PR segment >1.6	Increased area under initial positive portion of P wave in lead V_1 to >0.06 mm-sec
Increased duration and depth of terminal-negative portion of P wave in lead V_1 (P terminal force) so that the area subtended by it is >0.04 mm-sec	Rightward shift of mean P wave axis to >+75 degrees
Leftward shift of mean P wave axis to between −30 and −45 degrees	

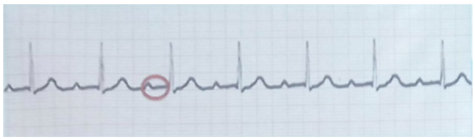
*In addition to criteria based on P wave morphologies, right atrial abnormality is suggested by QRS changes as described in the text.
Reference: Hancock EW, Deal BJ, Mirvis DM, et al. Recommendations for the standardization and interpretation of the electrocardiogram. Part V. ECG changes associated with cardiac chamber hypertrophy. *J Am Coll Cardiol*. 2009;53:992–1002.

B. PR INTERVAL (ATRIOVENTRICULAR/AV DELAY)

Prolonged PR Interval

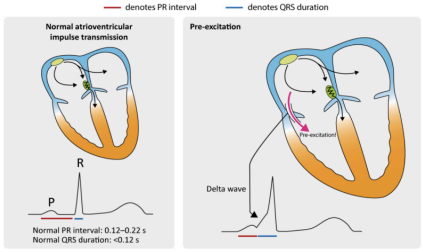
- **1st-degree AV block:** More than 200 milliseconds (>5 small squares)
  - PR interval is from the start of the P wave to the start of the

QRS.
- Common and does not have to be treated; usually secondary to beta-blocker use (down titrate the dose), usually asymptomatic.



o Shortened PR Interval

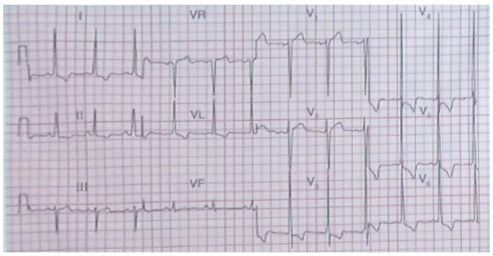
- **Preexcitation Pattern/Wolff-Parkinson-White Pattern:** Triad of Short PR-interval, presence of Delta wave, Prolonged QRS duration
  - Normally, the atria and ventricle should not have any other communication electrically except through the AV node. If there is a bypass track, it may shorten the PR interval (there is an accessory pathway).
  - A bypass track will not exhibit the normal delay exhibited by the AV node and the impulse will travel faster, making the PR shorter.
  - If the impulse will come across the impulse coming from the normal AV node, both will clash, the first part of the QRS will slur (delta wave), making the QRS be prolonged.
  - This pattern is called **WPW pattern** or **pre-excitation pattern** if you only read the ECG pattern and do not know the patient. It is called a syndrome if you know the symptoms of the patient.



C. QRS COMPLEX (VENTRICULAR DEPOLARIZATION)

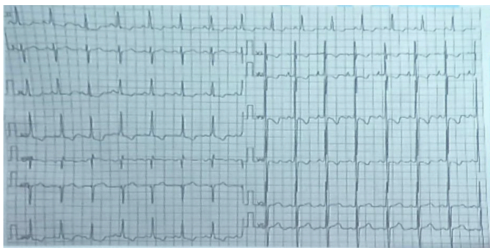
Left Ventricular Hypertrophy (LVH)

- The **Sokolow-Lyon Criteria:**
  - R wave in V_5 or V_{56} +S wave in V_1 or V_2 > 35 mm



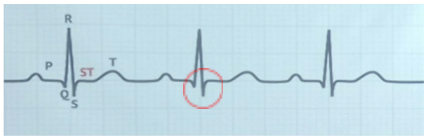
Right Ventricular Hypertrophy (RVH)

- **Criteria:** R axis deviation + R wave in V_1 > 7 mm

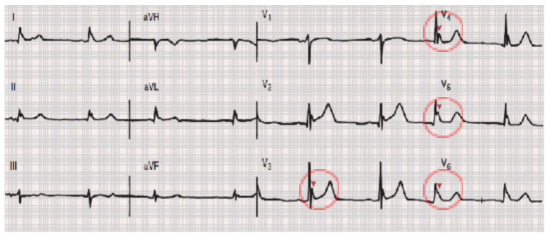


D. ST SEGMENT (PLATEAU PHASE)

- ST Segment is flat (isoelectric).
- Deviation: Elevation or depression of ST-segment by **> 1 mm**.
- **"J" (Junction) point** is the point between QRS and ST-segment.

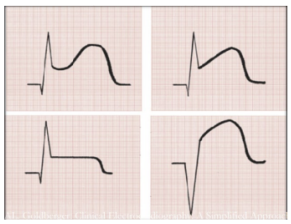


- J point elevation in systemic hypothermia: **Osborn Wave** (not MI)



Variable Shapes of ST-segment Elevations in Acute Myocardial Infarction (AMI)

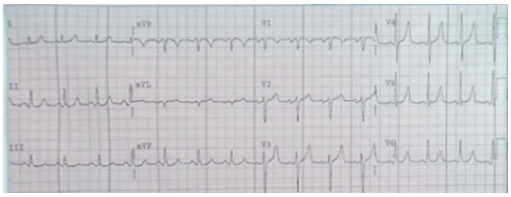
- If there is a deviation of the ST-segment, always think of AMI unless proven otherwise.
- The higher the ST segment, the bigger the myocardial involvement



E. T WAVE (VENTRICULAR REPOLARIZATION)

Abnormalities in the T Wave

- The T wave becomes abnormal when the normal sequence of repolarization does not occur.
- Several factors can change the sequence of repolarization:
  - **Slow conduction** of the depolarization wave.
  - **Shortened depolarization** in portions of the ventricular muscle.
- As a general rule, T wave amplitude corresponds with the amplitude of the preceding R wnave, though the tallest T waves are seen in leads V_3 and V_4.
  - **General Rule**: If the P wave and QRS is positive, the T wave should also be positive.
- Tall T waves may be seen in **acute myocardial ischemia** and are a feature of **hyperkalemia** (electrolyte abnormalities or dialysis patients who missed their dialysis are sensitive to potassium levels).



F. QT INTERVAL (VENTRICULAR DEPOLARIZATION & REPOLARIZATION)

- Extends from the onset of the QRS complex to the end of the T wave.
- Total duration of Depolarization and Repolarization.
- QT interval is **rate-dependent**: Decreases when heart rate increases.
- **Bazzett formula**: Corrects the measured QT interval to the effects of heart rate:

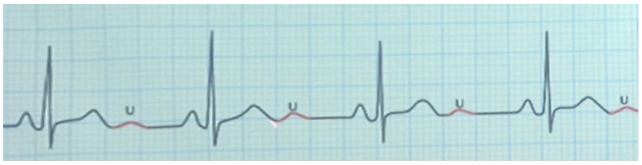
$$QTc = QT/\sqrt{RR}$$

- If the heart rate is abnormal (bradycardic or tachycardic), it is

usually affected and the Bazzett formula will correct for the variation in the heart rate.
- Only compute for the Bazzett formula if the heart rate is grossly abnormal.

G. U WAVE

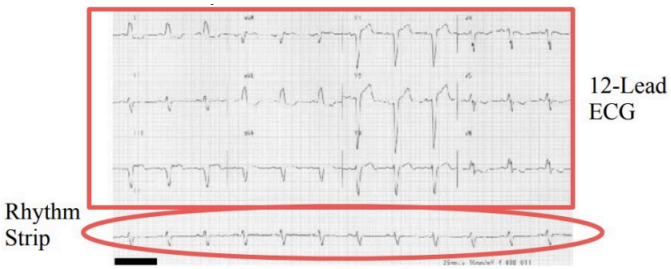
- U wave is related to afterdepolarizations which follow repolarization.
- U waves are small, round, symmetrical and positive in lead II, with amplitude < 2 mm.
- U wave direction is the same as the preceding T wave; more prominent at slow heart rates. Seen in **hypokalemia**.



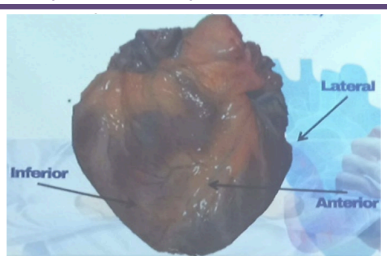
ELECTROCARDIOGRAPHIC DIAGNOSIS OF MYOCARDIAL INFARCTION (MI)

DIAGNOSING MYOCARDIAL INFECTION

- To diagnose myocardial infarction, you need to look beyond a rhythm strip and obtain a standard **12-Lead ECG**.
  - In MI, it is not enough to know there is an MI. You should also have an idea as to where the thrombus is.
  - In a normal ECG, after the 12-Lead is recorded, usually the machine will record a long strip (usually lead I or lead II, 10 second strip).
  - If the problem is more on arrhythmias or conduction abnormalities, the rhythm strip is used, but in MI, the 12-Lead is used because it will show a 3D representation of the heart.

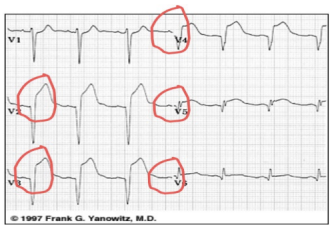


Views of the Heart (Left Ventricle)



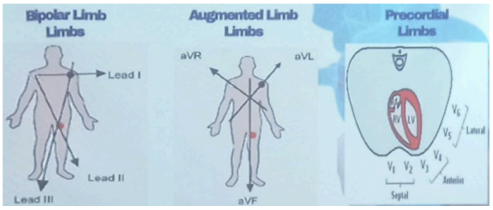
ST-Segment Elevation

- One way to diagnose an acute MI is to look for elevation of the ST segment.
- New ST elevation at the J point in 2 contiguous leads with the following cut points:
  - Any **0.1 mV deviation** in all leads (except V_2 and V_3) should be considered elevation unless proven otherwise, but it should be contiguous lead (the group of leads that would make sense).
    - V_1 and aVF does not represent the coronary distribution.
  - In leads V_2-V_3, the following cut points apply:
    - > 0.2 mV in men 40 years old and above
    - > 0.25 mV in men less than 40 years old
    - > 0.15 mV in women



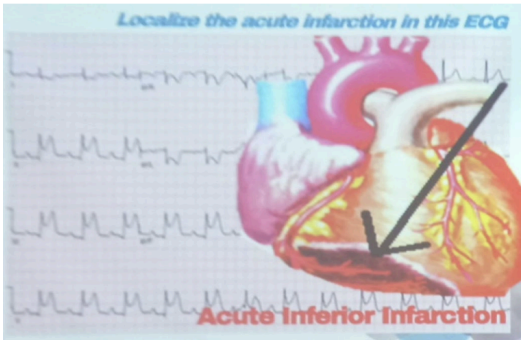
MYOCARDIAL INFARCTION LOCALIZATION

- Remember that the 12-Leads of the ECG look at different areas of the heart (LV and RV):
  - The **limb** and **augmented leads** "see" electrical activity moving to the left or laterally (I, aVL), and to the right (aVR) and inferiorly (II, III and aVF).
  - The **precordial leads** "see" electrical activity in the posterior to anterior direction.

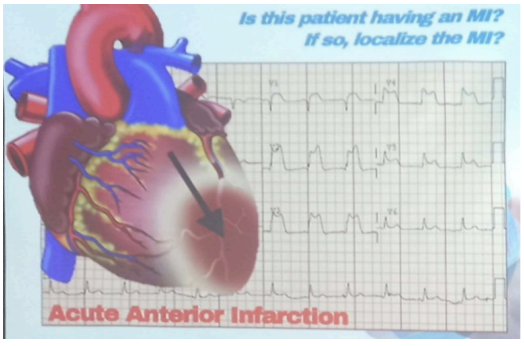


- **Anterior Myocardial Infarction**
  - The anterior portion of the heart is best viewed using leads V_1-V_4.
- **Lateral Myocardial Infarction**
  - The lateral portion of the ventricle is best viewed in Leads I, aVL, V_5-V_6.
- **Inferior Myocardial Infarction**
  - The inferior portion of the heart is best viewed in Leads II, III, aVF.

Localize the Myocardial Infarction:

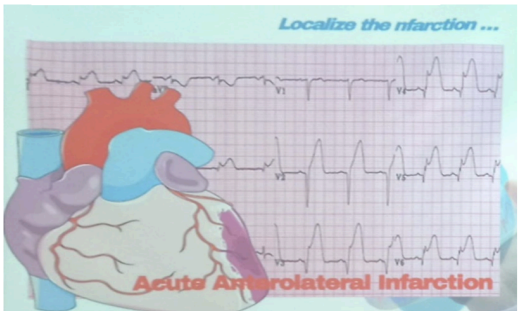


- There is an elevation in the **inferior leads** (leads II, III, and aVF) = MI is in the **inferior**. The **right coronary artery** is the culprit vessel.



- There is an elevation in the **anterior leads** (V_1, V_2, V_3, V_4, and V_5) = MI is in the **anterior**, so there is an acute anterior infarction in the left anterior descending. The more contiguous leads involved,

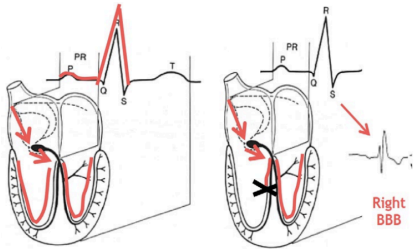
the more proximal the thrombus because the distribution is bigger.



- There is an elevation in all the **anterior** and **lateral leads** (V_1, V_2, V_3, V_4, V_5, aVF). The more **proximal** the thrombus is, the more **acute** is the treatment.

ELECTROCARDIOGRAPHIC DIAGNOSIS OF BUNDLE BRANCH BLOCKS (BBB) – NORMAL IMPULSE CONDUCTION

- **Sinoatrial (SA) node** to **Atrioventricular (AV) node** will record the P wave and PR interval.
- QRS will be the penetrating bundle to the **Purkinje system**.
- Conduction in the Bundle Branches and Purkinje fibers are seen as the QRS complex on the ECG.
  - Therefore, a conduction block of the Bundle Branches would be reflected as a change in the QRS complex.
  - If there are blocks the bundle branches in the right, it will produce **RBBB** and in the left, it will produce **LBBB**.

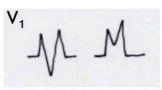


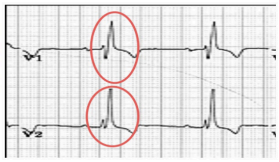
ECG CHANGES

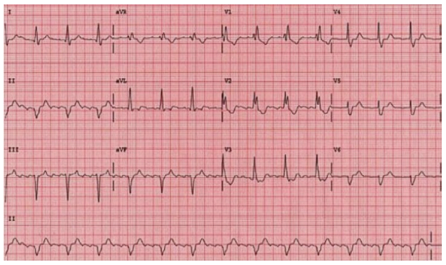
- **QRS complex widens** (> 0.12 sec if the block is complete).
- **QRS morphology changes** (varies depending on ECG lead, and if it is a right vs. left bundle branch block).

Complete Right Bundle Branch Block (CRBBB)

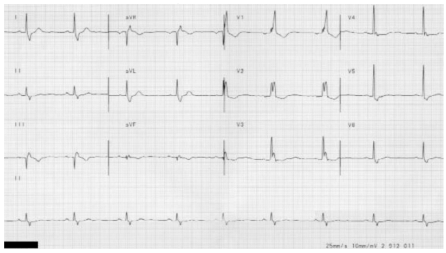
- ECG Changes: The QRS complex widens with a unique, virtually diagnostic shape in those leads overlying the RV.





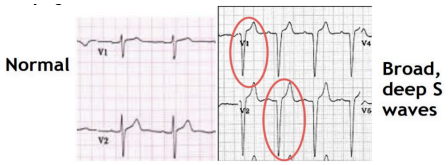


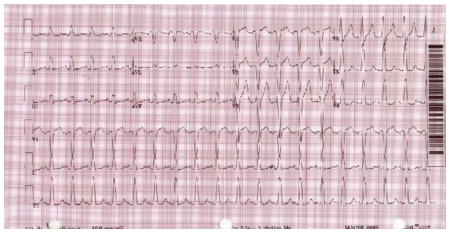
CRBBB: There is RSR in V_1 and V_2. CRBBB is common in females in their 20s. Does not cause any hemodynamic abnormalities.



Complete Left Bundle Branch Block (CLBBB)

- ECG Changes: The QRS complex widens with a **deep QS** in the leads overlying the LV.

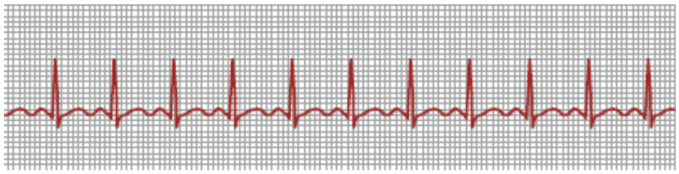




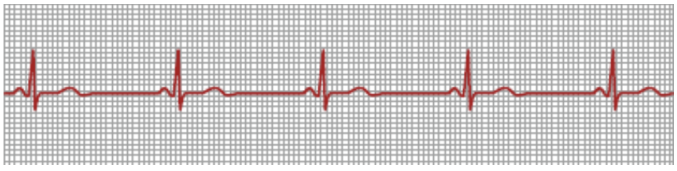
CLBBB: There is deep QS in V_1 and V_2. An acute CLBBB may be a manifestation of acute MI.

ELECTROCARDIOGRAPHIC DIAGNOSIS OF MISCELLANEOUS DISORDERS

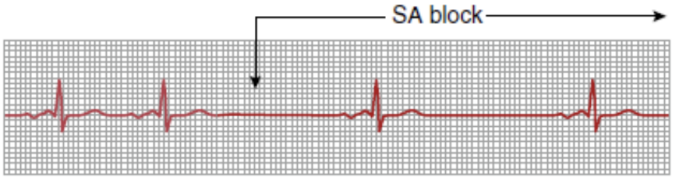
- **Sinus Tachycardia** – Everything is normal except the heart rate is more than 100 bpm.



- **Sinus Bradycardia** – Everything is normal except the heart rate is less than 60 bpm.

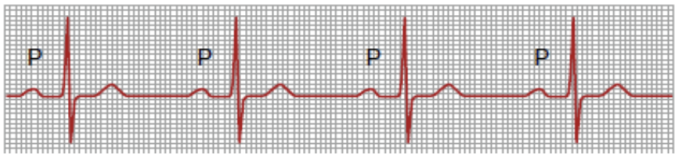


- **Sinoatrial (SA) Block** – For any reason, the sinus node stopped; significant pause will be **3 seconds**.

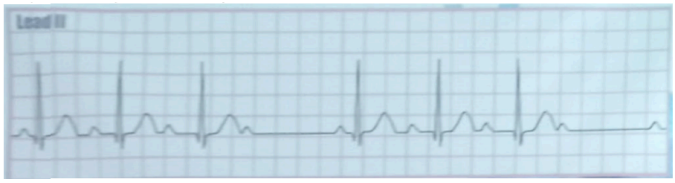


- **1st Degree AV Block** – Prolongation of the PR to more than 5

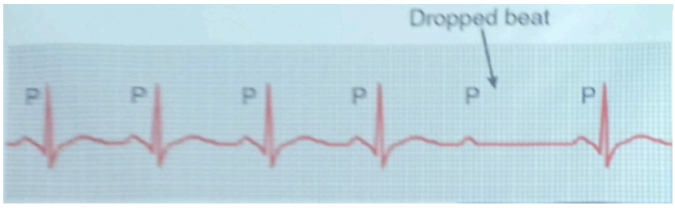
small squares.



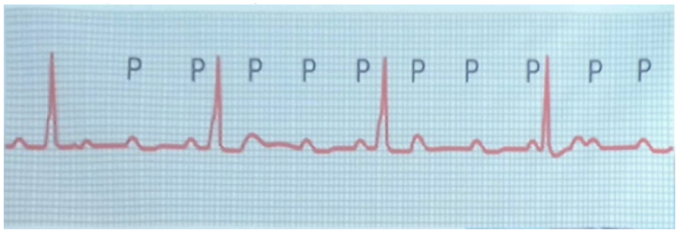
- **Mobitz Type 1 – 2nd Degree AV Block (Wenckebach)**
  - Look for P waves not followed by QRS. The AV node totally disregards the impulse from the sinus node.
  - In the long strip, a **group beating** will be seen, which is a clue for 2nd degree blocks. There are P waves not conducted, it is only up to the atria and after the AV node, it is disregarded.
  - If the PR interval is prolonging before the drop beat, it is type 1 (short, longer, longest, drop).



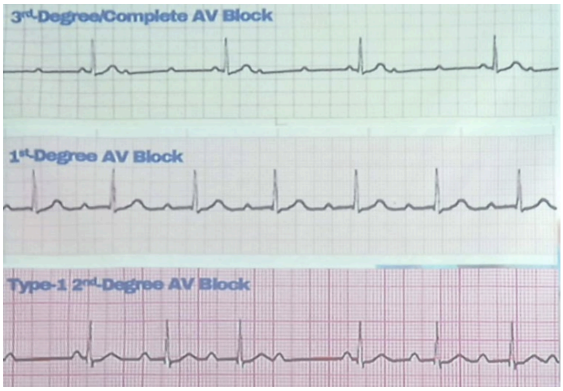
- **Mobitz Type 2 – 2nd Degree AV Block**
  - There is no prolongation of the PR, so the PR appears fixed, and the drop suddenly appears without warning.
  - **More morbid** compared to type 1.
  - A pacemaker is inserted, especially if the cause is reversible (no beta blocker therapy or electrolyte abnormality determined).



- **3rd Degree (Complete) AV Block**
  - Atria and ventricles are completely dissociated (atria and ventricles are conducting by itself and not related).
  - P to the next P and R to the next R appear regular but are not related (completely dissociated).

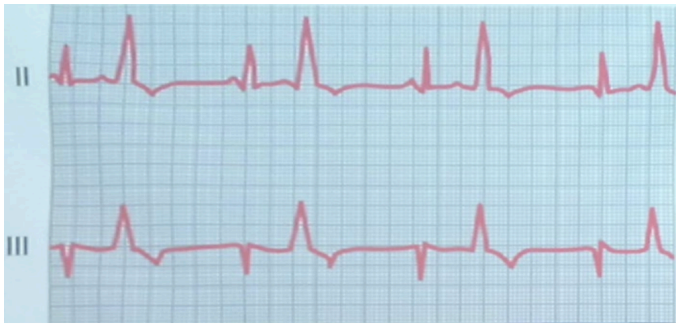


DIFFERENTIATING 1ST, 2ND, AND 3RD DEGREE BLOCKS

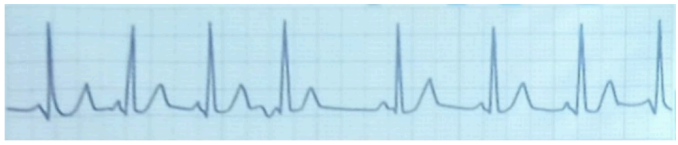


MISCELLANEOUS DISORDERS (CONTD.)

- **Premature Ventricular Contractions (PVC)** – There is an extra QRS after a normal cycle that is bizarre-looking and is not followed by a P wave.

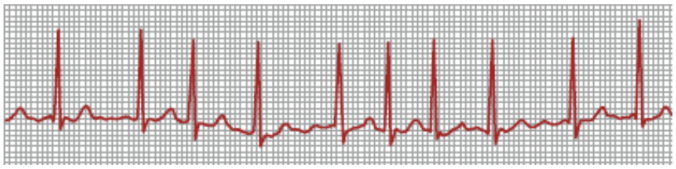


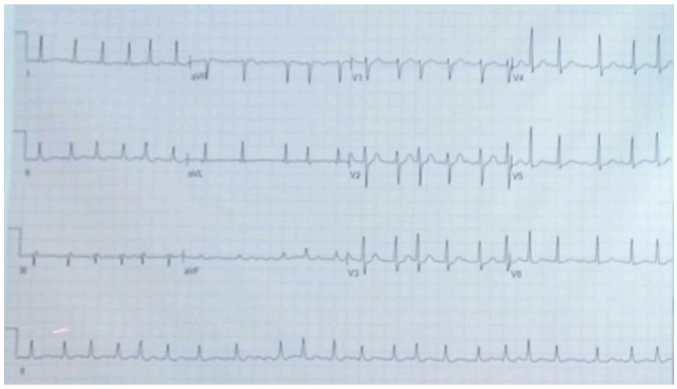
- **Premature Arterial Contractions (PAC)** – Normal looking QRS but preceded by an abnormal P wave (e.g, inverted P and early occurring QRS).



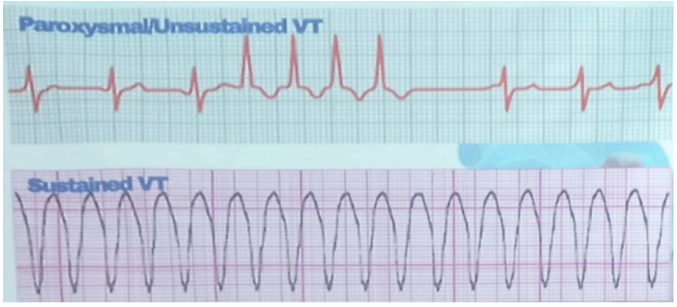
Coming from the atrium lower than the AV node, since P wave is inverted so the vector may be going up.

- **Atrial Fibrillation**
  - Irregularly irregular QRS and a P wave cannot be determined.
  - **T waves** are seen (no P waves) and have no pattern.
  - When the patient's pulses are palpated, they are usually irregular. Very common in the elderly and is a very significant cause of stroke.
  - These patients are maintained on **anticoagulant therapy** because a clot may form in the LA of a fibrillating atria and if it is thrown out to the carotids, it may produce a stroke.

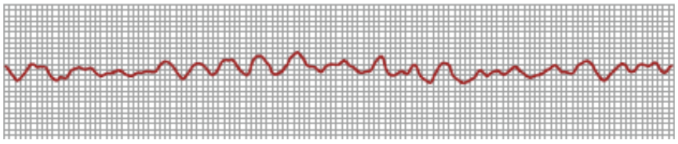


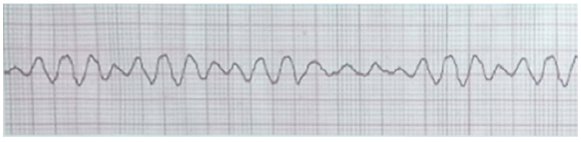


- **Ventricular Tachycardia (V Tach)**
  - Very wide QRS; no one can survive.
  - If PVC occurs at least 3 times, it is already VT; if it will still convert, it is a **non-sustained VT**.

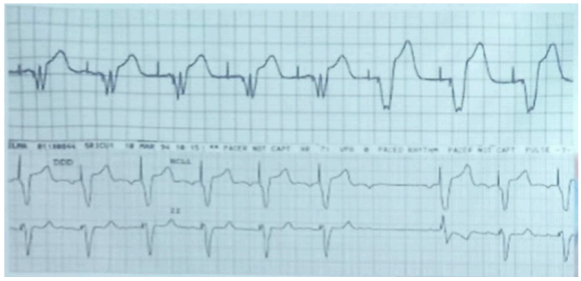


- **Ventricular Fibrillation**
  - No patterns, only wavy lines.
  - If seen and there is a defibrillator at hand, electroshock right away (or CPR). Only sustained ventricular tachycardia and ventricular fibrillation require electroshock. Do not give electroshock to other rhythms otherwise, you are promoting more death.

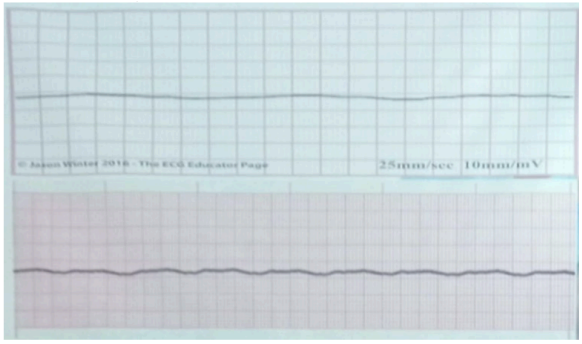




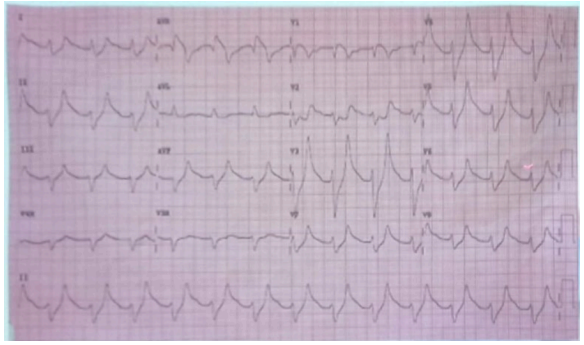
- **Pacemaker Rhythm**
  - Seen in patients with pacers inserted.
  - The blips are not artifacts but are the firing of the pacer.
  - Some pacers may have 2 leads (inserted into the RA and RV).



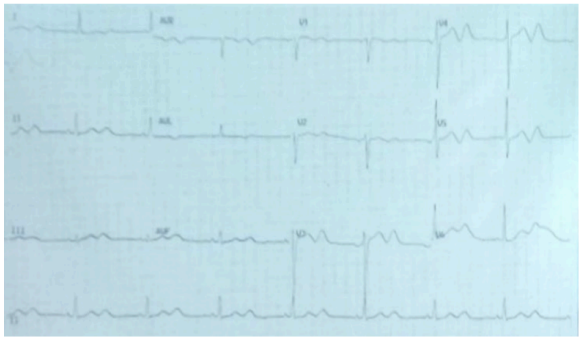
- **Asystole** – “Please see that your leads are attached”.



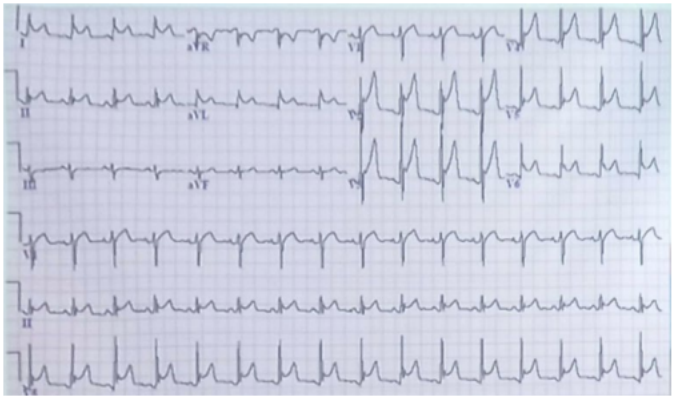
- **Hyperkalemia** – Tall or Peaked T waves; seen in lethal injection (**Potassium chloride**).



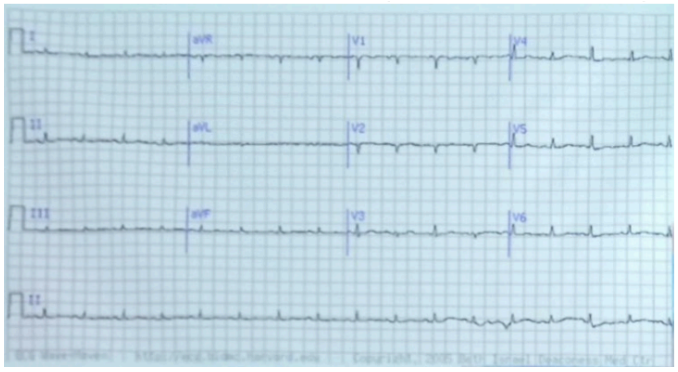
- **Hypokalemia** – Depressed or flat ST-T waves, U waves seen.

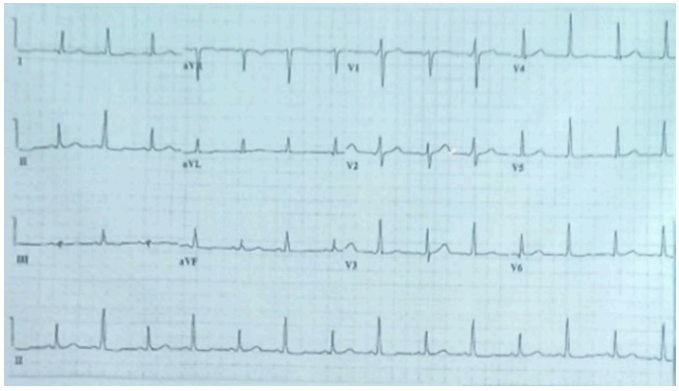


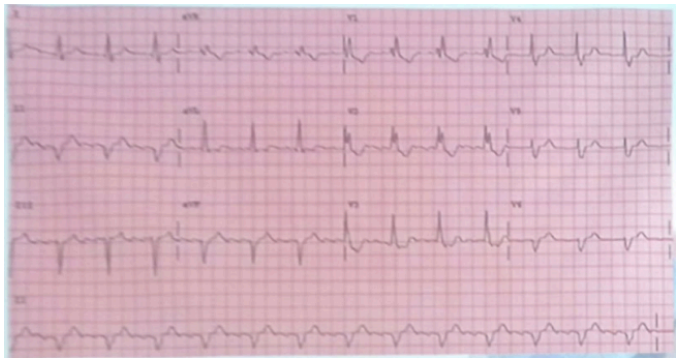
- **Acute Pericarditis**
  - Diffuse S-T elevation (ST elevation on all the leads), P-R depression.
  - Ask the patient for the characteristics of the pain.
    - Pain in MI (angina pectoris) is usually more on numbness and heaviness in the chest, often radiating to the arm or jaw.
    - **Pericardial pain** is usually pinprick and the patient can localize it and find temporary relief by leaning forward or lying on the side.
    - Also, review the patient's profile.



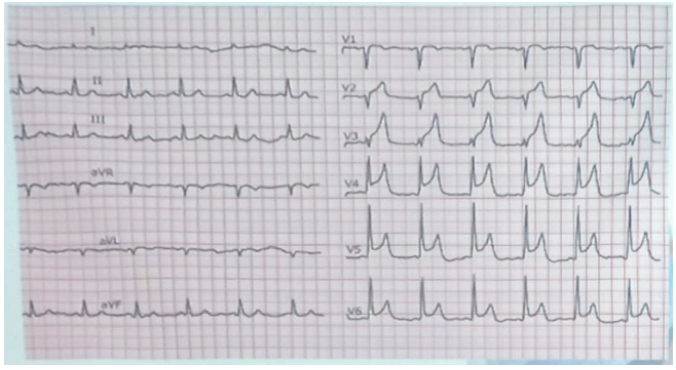
- **Cardiac Tamponade**
  - Low-amplitude complexes, presence of electrical alternans.
    - Because there is excessive interference between the skin and the heart, the QRS complexes will appear very small.
    - Electrical alternans is alternating positive and negative.
  - Too much fluid surrounds the heart in the pericardial sac such that the RA inflow of blood is impeded (low cardiac output).



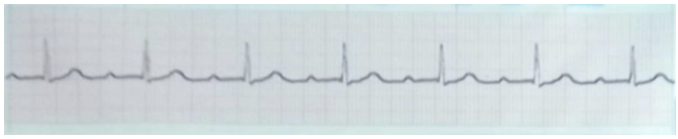




SINUS RHYTHYM WITH CRBBB

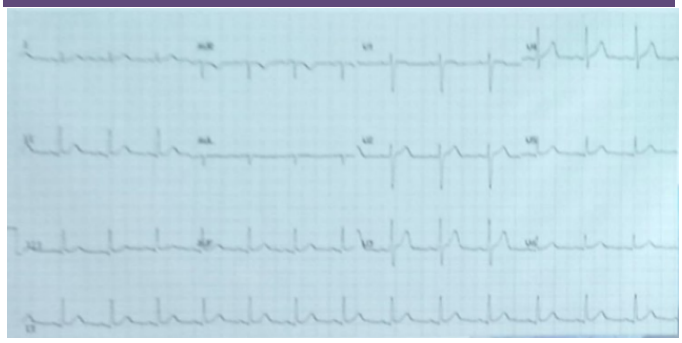


SINUS RHYTHM WITH ACUTE ANTEROLATERAL MI

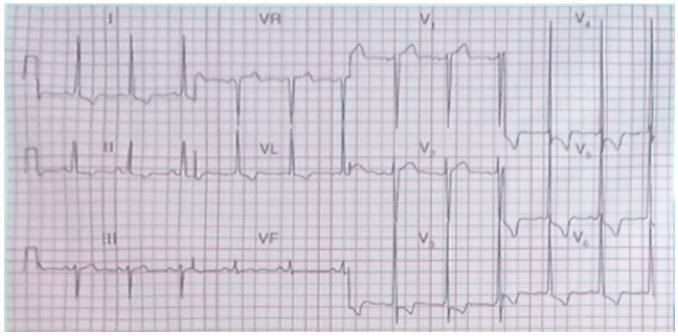


SINUS RHYTHM WITH 1ST DEGREE AV BLOCK

ECG REVIEW



NORMAL SINUS RHYTHM



SINUS RHYTHM WITH LVH BY VOLTAGE CRITERIA