Fundamentals of Electrocardiography

A Quick Guide to Read, Understand and Treat.

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Acknowledgements

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Chapter 1

Electrocardiography

Electrocardiography is the graphical representation of the electrical activity in the heart during each heartbeat. This technique was first introduced and recorded in 1872 by a Scottish electrical engineer, Alexander Muirhead, in St Bartholomew's Hospital. He specialized in wire telegraphy, using this technique he attached wires to the patient's wrist and recorded the very first ECG. Following his pioneering work, a number of scientists played great contributions to developing the ECG we see today.

Figure 1.1. Alexander Muirhead's fax machine used for recording ECG.

Rune Elmqvist, a well-known engineer for his remarkable work in the field of heart implants was the first one to develop an ink-jet ECG printer, called Mingograph to record the heart's electrical activity on paper in the late 1940s.

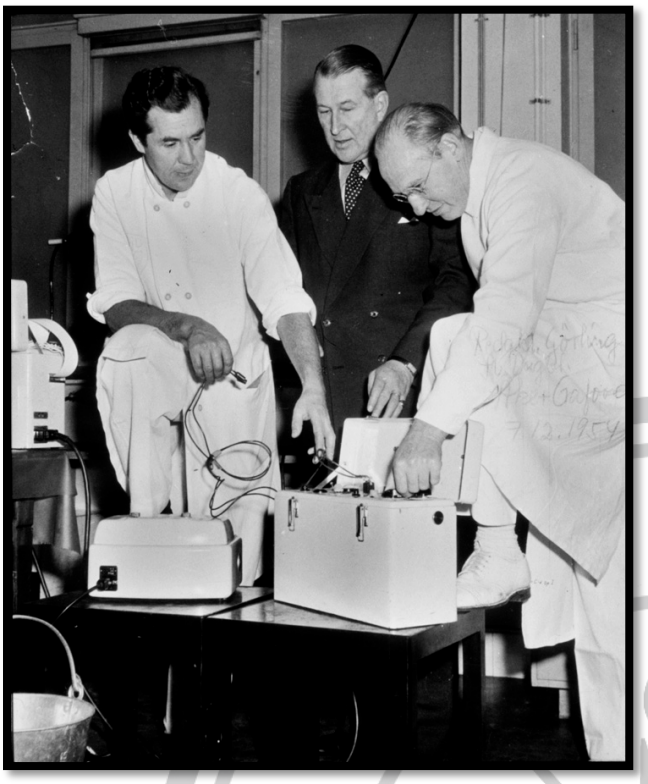

Figure 1.2. Rune Elmqvist and his colleagues with Mingograph.

The ECG we know today is a series of waves on paper generated by each cardiac cycle. The waves carry magnitude and deflections in correspondence to the base line. Each terminology needs explanation to make ECG easier to read.

Baseline

Baseline is an isoelectric line which plays key role to determine the direction and magnitude of waves. Each wave generated by the heart can have either positive, negative deflection or isoelectric representation on the ECG. By rule, if the deflection is above the base line it is termed as a positive deflection but if under then negative deflection, as shown in (Figure 1.3.).

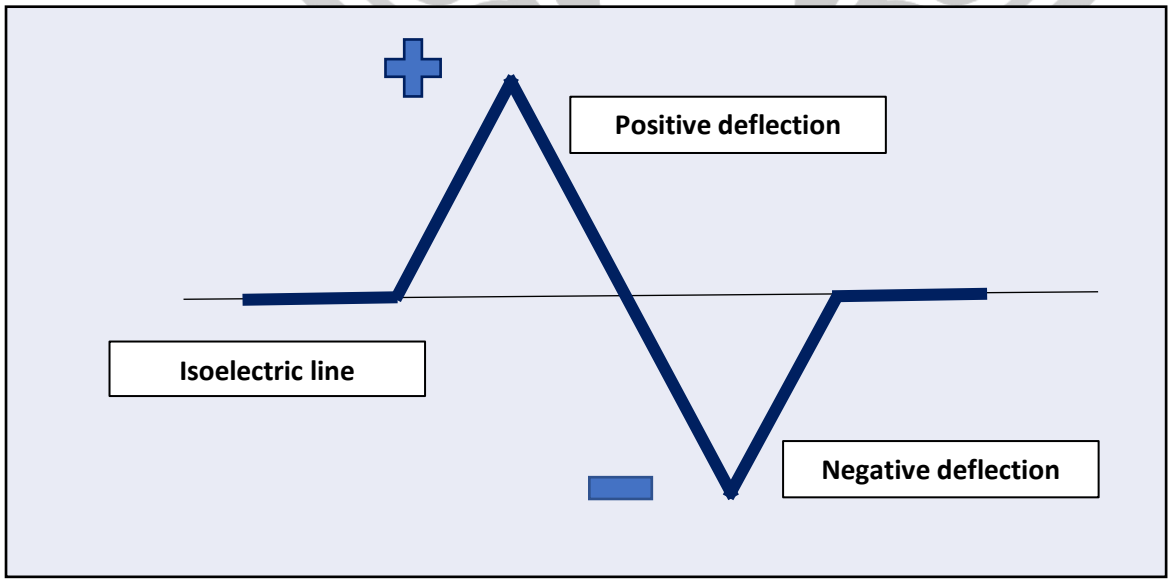

Figure 1.3. Direction of Deflections

- Positive deflection-above isoelectric line
- Negative deflection- below isoelectric line

Direction of waves

The direction of the deflection depends on the flow of current and placement of electrodes. The electrical current generated in the heart during each beat is recorded as waves on ECG. Therefore, if the current flows towards the electrode will generate a positive deflection while, away a negative deflection.

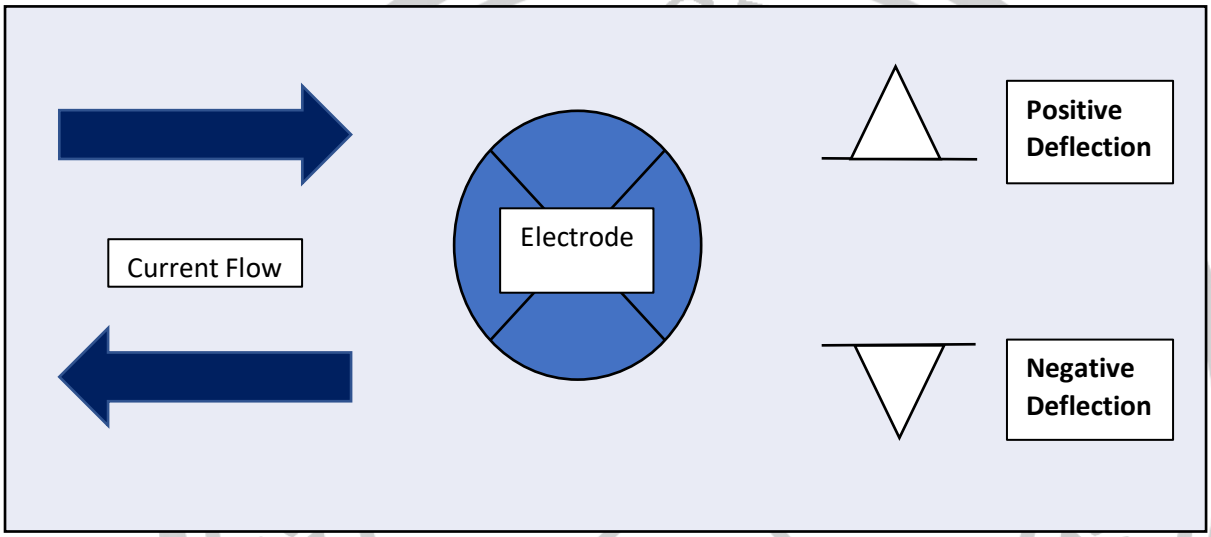

Figure 1.4. Current flow and Deflections

- Current flow towards- Positive deflection
- Current flow away- Negative deflection

Amplitude of waves

Amplitude of the wave is basically the voltage that is measured in millimeters.. It is the vertical height of positive or negative deflection from the baseline. In addition, the height depends on two factors;

1. Strength of voltage
2. Thickness of heart tissue

The strength of voltage is directly proportional to the amplitude of waves recorded on the ECG. In simple words, more the electrical force generated during each cardiac beat, higher would be the wave height and vice versa. For example, if the strength of atria to ventricular contraction is taken into consideration, ventricles contract with a much greater force in comparison to the atria. Therefore, generating a strong and taller wave in amplitude as seen in Figure 1.5.

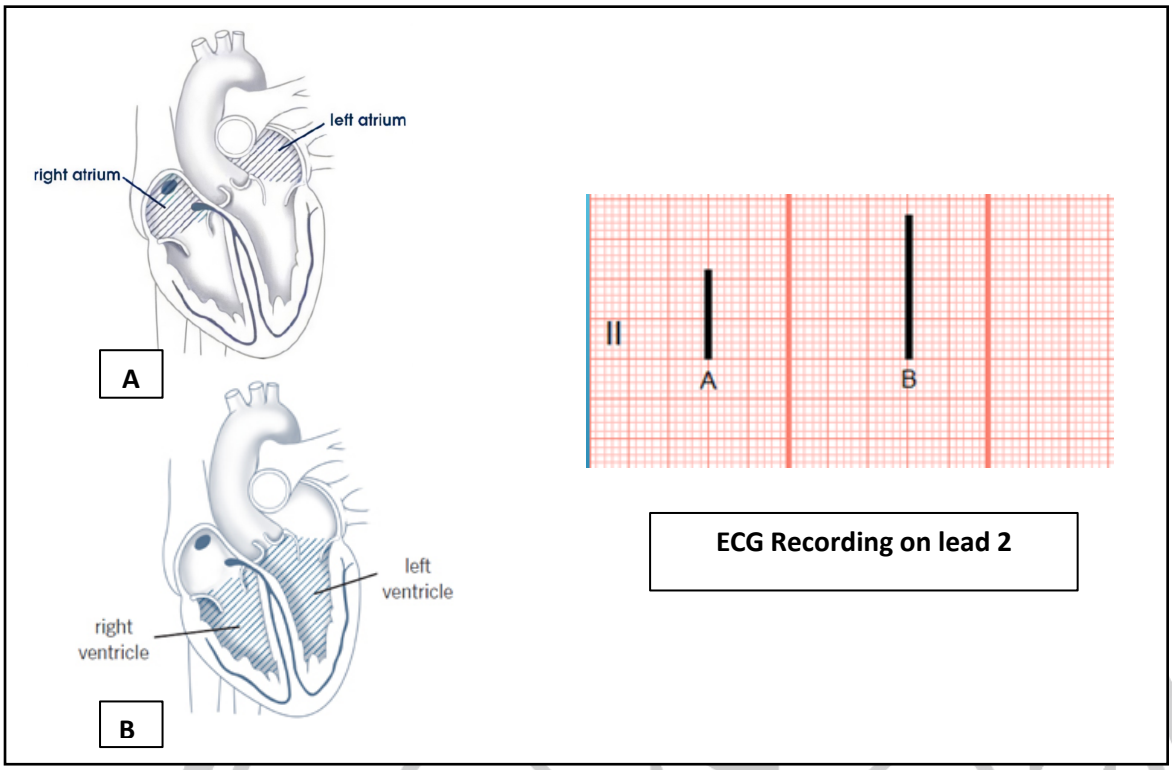

Figure 1.5. Relation of voltage strength to amplitude.

A. Atrial contraction
B. Ventricular contraction

Similarly, the thickness of heart chambers can generate a magnitude based on the strength of individual contraction. In certain conditions like hypertrophy of heart chambers like, atrial or ventricular hypertrophy characterized by thickening of the heart walls result in stronger contractions. Hypertrophic walls generate taller impulses that are seen on ECG.

In contrast, skin thickness shows an inverse relation to the amplitude and magnitude in the ECG as seen in figure 1.6.

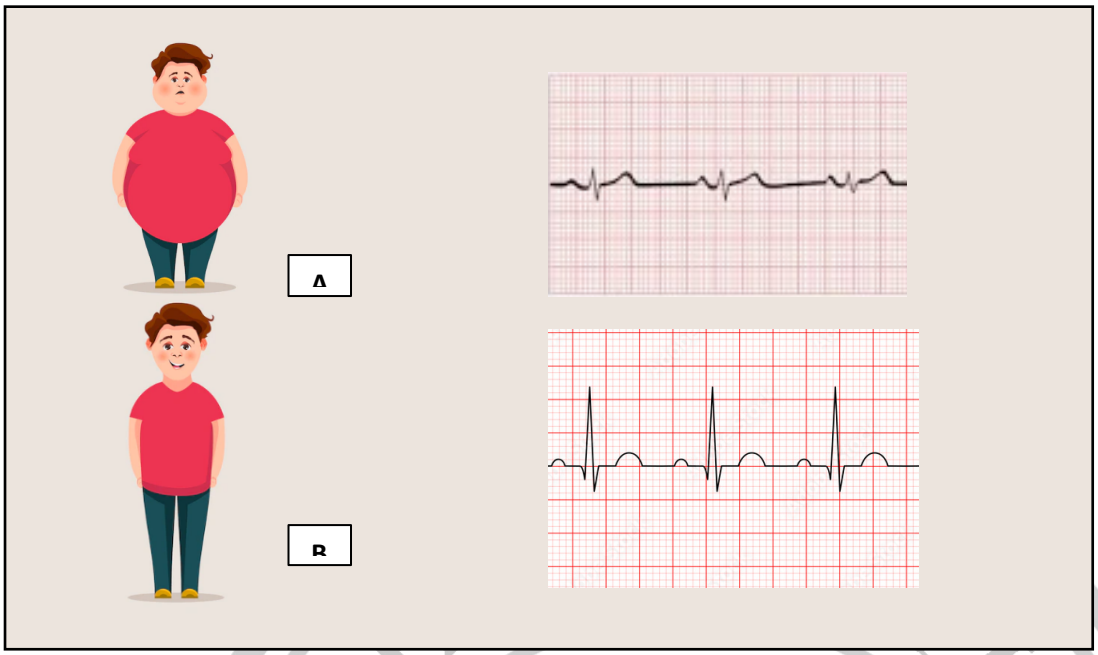

Figure. 1.6. Relation of chest wall thickness to amplitude.

A. Thick chest wall-low amplitude waves
B. Thin chest wall-high/normal amplitude waves

In simple terms with increase in the chest wall thickness, the electrical impulses recorded by the heart would be weaker due to increase in distance between placed electrode and the heart. Therefore, someone with a thicker chest wall due to being overweight or accumulation of fluid in the heart sac called pericardium (pericardial effusion or cardiac tamponade) can have low voltage waves whereas, on the other hand a thin individual due to reduced thickness can have taller or even normal voltage impulses on the ECG.

Chapter 2

Electrophysiology of Heart

The electrophysiology of heart is the understanding of the electrical current that generates through a well-defined circuit to bring a rhythmic contraction of its muscle during a single beat. The genesis of such a complex electrical system can only be understood by starting off with basic anatomy of the heart. The human heart has four chambers divided into two smaller atria's and two large ventricles separated by segments. The separating septums include,

1. Interatrial septum
2. Interventricular septum
3. Atrioventricular septum

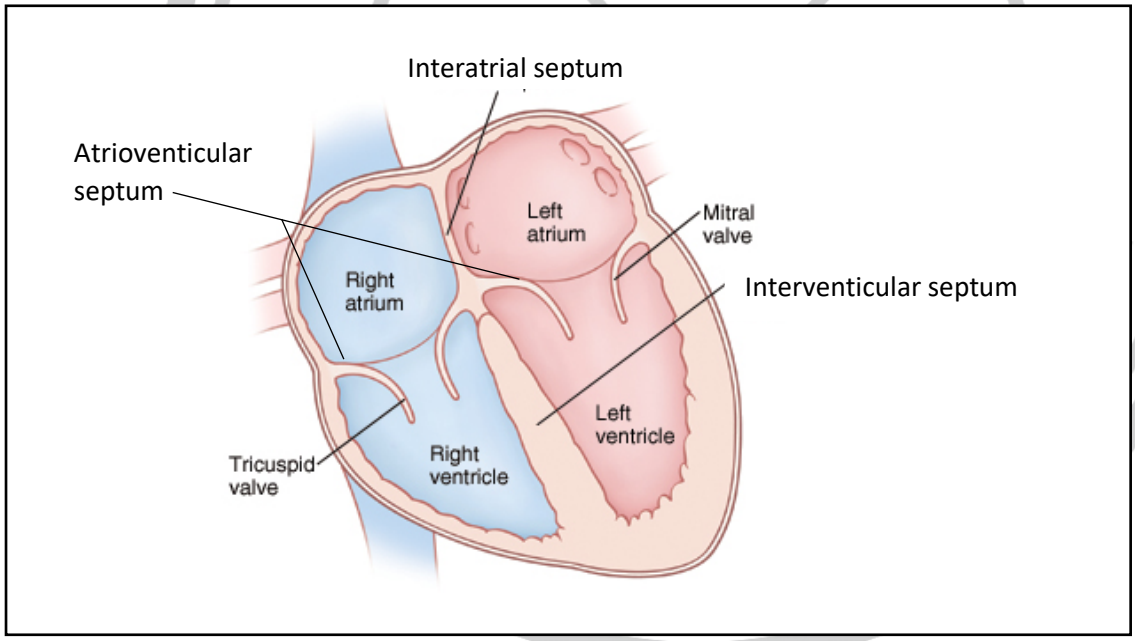

Figure 2.1. Heart chambers and septums.

As seen in figure 2.1, septum separating atria is called interatrial septum and that between ventricles is known as interventricular septum. However, the atria are having a division called atrioventricular septum that is provided with two gateways that allow blood to flow from atria to ventricle during each cardiac cycle. These two gateways are termed as tricuspid and mitral valve which play significant role to not only control flow through them but also prevent any back flow. The atria and ventricles have to contract simultaneously to generate a waveform on the ECG. To understand the mechanism of their contraction we will discuss at cellular level.

The heart comprises of three layers, namely from outer to inner most:

1. Pericardium
2. Myocardium
3. Endocardium

Pericardium is the outermost, protective layer of the heart with two sub layers, outer parietal and inner visceral pericardium that contains a fluid filled sac called pericardial sac. Myocardium, middle muscular layer that is composed of myocytes that contracts in response to the changes in ion flow during the electrical activity that leads to a synchronous contraction. Endocardium, the inner most layer forms the lining of chambers and heart valves as seen in figure 2.2.

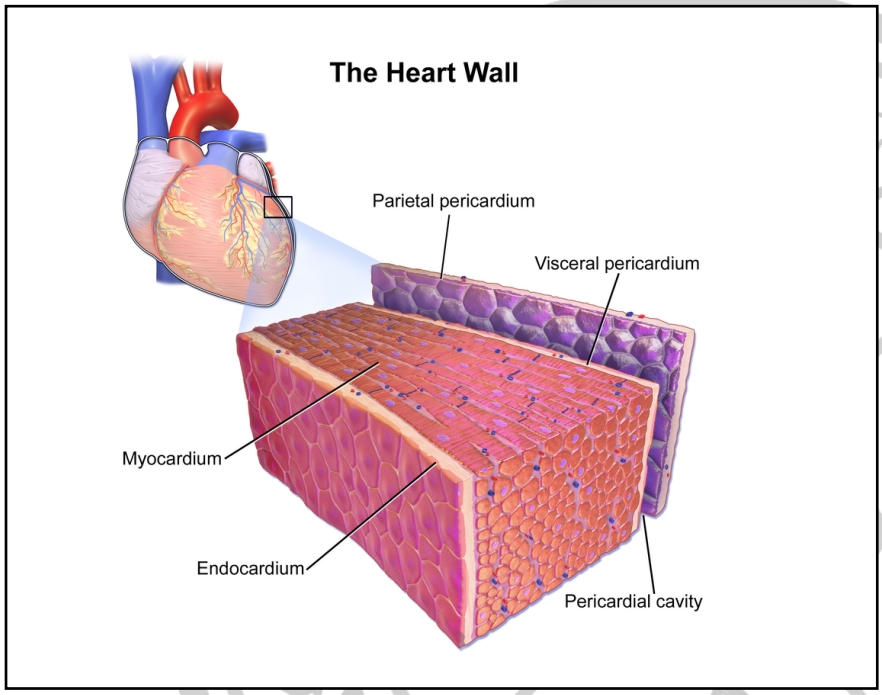

Figure 2.2. Three layers of the heart: Pericardium, Myocardium and Endocardium.

As the contractile system form the basis of a heartbeat, only myocardium will be discussed for this reason.

Myocardium:

Myocytes form the cellular component of the myocardium consisting of small contractile filaments called actin and myosin that lock and unlock with each contraction. To have a better understanding of this phenomenon, we need to understand a few terms.

- Membrane potential:

The charged potential that exists across the membrane of a cell due to ionic difference is called membrane potential. In simple terms, a cell membrane can carry a negative or a positive charge due to flow of ions across it through specialized pumps to create that gradient.

In a resting state, the outer surface of myocytes carries a negative charge with a voltage of –90mV which is almost equivalent to the equilibrium potential for potassium ions having a concentration of 4 mM outside the cell. In such a situation, the actin and myosin filaments are relaxed or unlocked.

In an active state, the calcium pump on the surface of myocytes opens allowing a flow of positively charged ions into the cell. As a result, the membrane potential increases to +20mV that causes actin as well as myosin filaments to interlock and lead to muscle fiber contraction. The process of electrical activity generated and running through the muscle layer is termed as depolarization.

After completion of contraction, the myocardium returns back to its resting potential by opening the potassium channels and closing of the calcium pumps. In other words, loss of positive charge through these pumps will slowly cause a drop in voltage back to –90mV for the myocytes to relax. Henceforth, the return of membrane potential back to its resting state is called repolarization (figure 2.3).

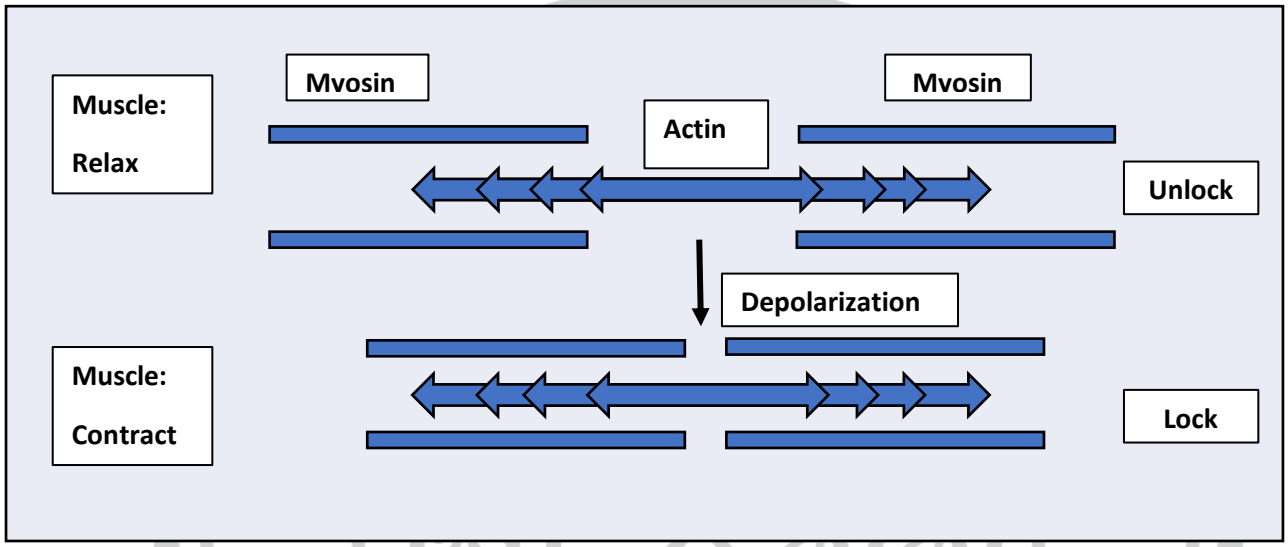

Figure 2.3. Muscle contraction with lock and unlock mechanism.

Depolarization and repolarization follow synchronous fashion in the myocardium of both the atria and ventricle. So, they can contract and relax in a rhythm.

Electrical Circuit of heart:

The heart constitutes of cells that have the capacity to generate electrical impulses through the heart to bring a sequential beat. These are called;

1. Sinoatrial node
2. Atrioventricular node

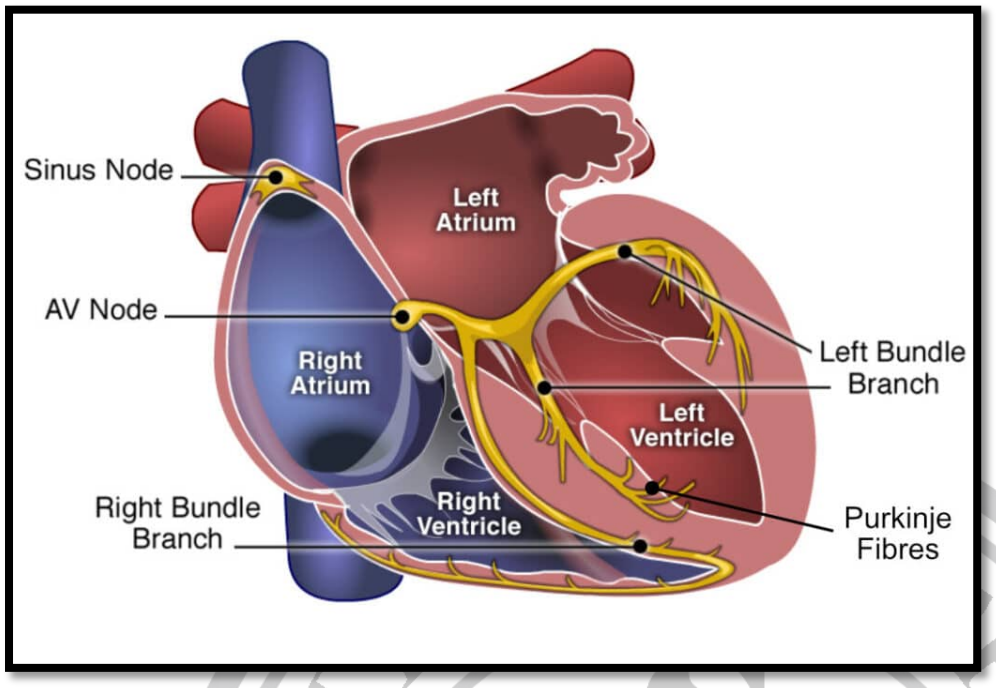

Figure 2.4 Conductive system of heart.

Sinoatrial node (SA node) is located in upper part of the right atrium, known as the natural pacemaker of heart. Electrical impulses generate at SA node cause atrial depolarization (contraction) followed by spread of current to AV node.

Atrioventricular node (AV node) is found in the inferior part of interatrial septum, atrioventricular junction that allows a short delay in transmission for the ventricles to fill with blood (repolarization) prior to contraction into the aorta and pulmonary artery.

During the AV nodal delay, the impulses make its way through a special pathway called *Bundle of His* into the ventricles. The pathways further divide into two bundle branches namely, right bundle branch and left bundle branch in accordance to the ventricle it enters. Afterwards, the bundle branches give rise to smaller terminal ramifications called Purkinje fibers that penetrates the heart tissue from endocardial to epicardial surface.

Chapter 3

Electrocardiogram and Leads

Electrocardiogram is the graphical representation of the electrical activity produced in the heart. The impulses are propagated into different directions by the activated myocardium. Electrodes are placed on several parts of the body that pick up these electrical signals and produce the ECG on paper. A pair of these electrode form a lead. Each lead constitutes of a positive and negative electrode that records electrical forces from numerous aspects of the heart.

By changing the position of these electrodes, we can alter the lead obtained as well as the angle in view. A variety of angles are recorded to lay down a detailed view of the heart. In medical practice, we use a 12-lead ECG that comprises of 6 chest leads and 6 limb leads.

1. Chest leads

The chest leads are six in number namely V1 to V6 (Figure 3.1.), often seen color coded to remember correct placement in a clinical environment. These electrodes are placed on the left side of chest with each placement indicating a single lead (Table 3a). Ideally, chest leads are placed on the left side but under special circumstances a right positioning might be required as in,

- Right ventricular infarct
- Dextrocardia (Heart on the right side)

Chest Lea d	Color Code	Location on left side	Location on left side
V1	Red	Above the fourth intercostal space, right to sternal border	Above the fourth intercostal space, left to sternal border
V2	Yellow	Above the fourth intercostal space, left to sternal border	Above the fourth intercostal space, right to sternal border
V3	Green	Midway between V2 and V4	Midway between V2 and V4
V4	Brown	Above the fifth intercostal space, midclavicular line.	Above the fifth intercostal space, midclavicular line.
V5	Black	Above the anterior axillary line at same level as V4	Above the anterior axillary line at same level as V4
V6	Purple	Above the midaxillary line at the same level as leads V5	Above the midaxillary line at the same level as leads V5

Table 3a. Chest leads V1-V6 with Location and Color Sequence.

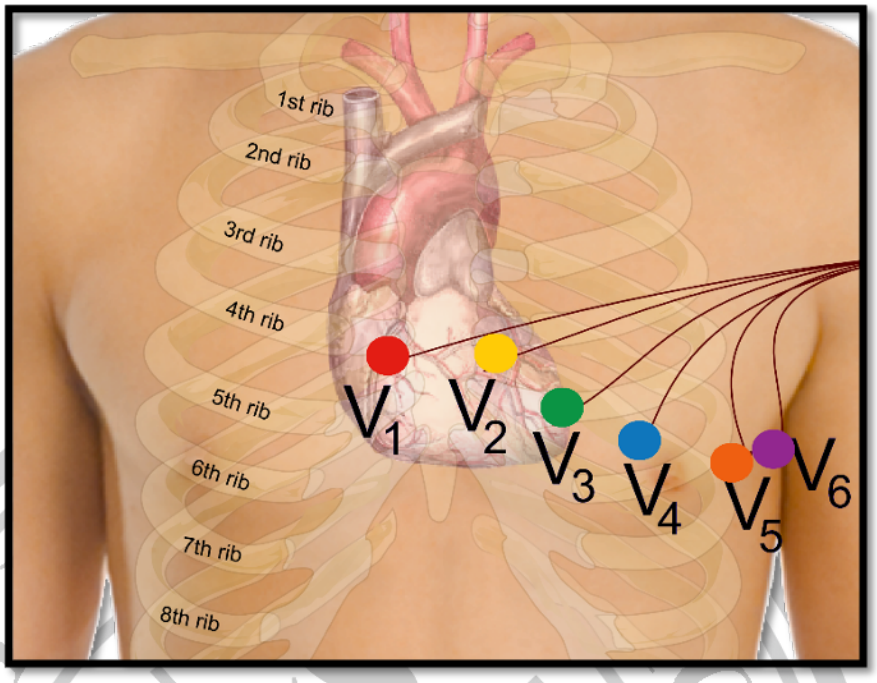

Figure 3.1. Chest leads V1-V6 with Location and Color Sequence.

2. Limb Leads

The limb leads as the name employs is applied on the limbs. They are of further two types:

- Augmented limb leads
- Standard limb leads

Augmented leads are three in number. It records the electrical forces from a single limb at a time. Moreover, they are carrying a positive charge that makes them unipolar in nature. The three leads at right arm, left arm and right leg have a positive electrode while, the center is a negative or zero potential as shown in Figure 3.2. The augmented limb leads include,

Augmented Limb leads	Location
aVR	Right Arm
aVL	Left Arm
aVF	Right Foot

In case of reversal of electrodes at arms that is accidental swapping of right with left or vice vera, changes in ECG produced are called technical dextrocardia. Variations in ECG of a technical dextrocardia to true dextrocardia can be differentiated by following:

- ✓ Mirrorlike inversion of LI
- ✓ aVR replaced with aVL
- ✓ No change in aVF.
- ✓ Lead II replaced with Lead III

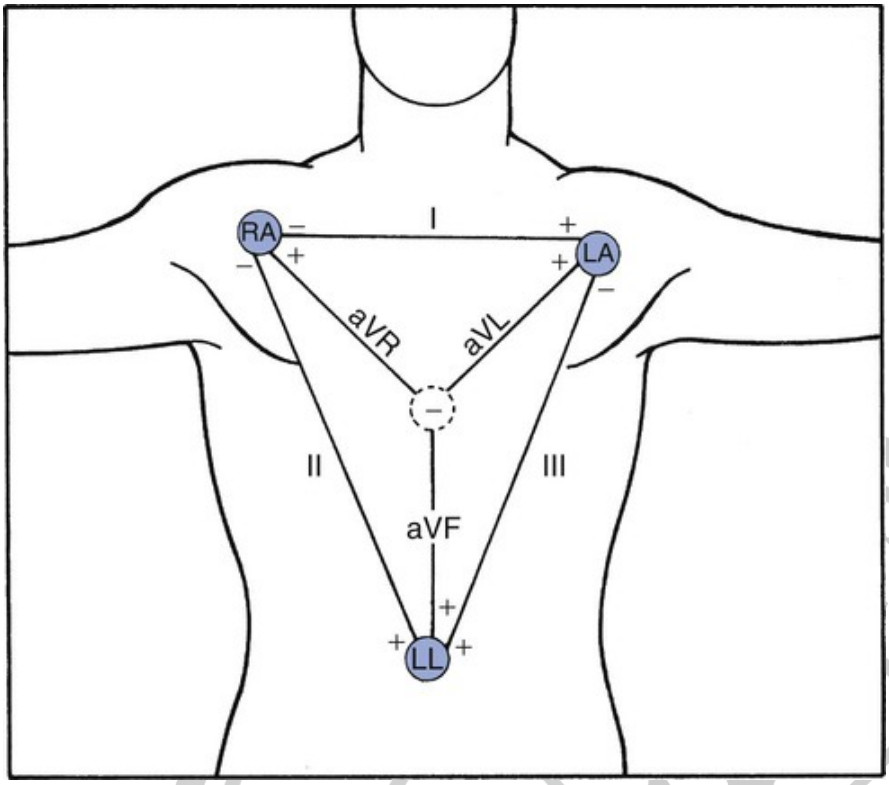

Figure 3.2. Augmented and standard limb leads

In contrast to the above leads, standard limb leads represents the electrical forces between two limbs at the same time. They are bipolar in nature, meaning that one limb carries a positive charge and other negative as seen in figure 3.2. The standard limb leads are only three in number as,

Limb Leads	Positive electrode	Negative Electrode
Lead I	Left Arm (LA)	Right Arm (RA)
Lead II	Left Leg (LL)	Right Arm (RA)
Lead III	Left Leg (LL)	Left Arm (LA)

Graphical Representation of Leads

The 12-lead ECG illustrate the heart at different angles in the form of a graph. The graph is recorded in a sequence of LI-III, aVR, aVL, aVF, V1-V6. In addition, the left ventricle is not only the clinically dominant but the strongest chamber of the heart that requires a detailed description in regards to the lead representation. Regional presentation of left ventricle on ECG can be appreciated in Table 3b.

Leads	Location on left ventricle
V1, V2	Septal
V3, V4	Anterior
V5, V6	Lateral
V1 to V4	Antero-septal
V3 to V6	Antero-lateral
LI, aVL	High lateral
LII, LIII, aVF	Inferior

Table 3b. Chest and Limb leads representation of left Ventricle

Einthoven Triangle

Keeping in mind the above information about different type of limb leads, it is understood that if one looks at the standard (Lead I-III) or augmented leads (aVR, aVL and aVF) they form an equilateral triangle called Einthoven triangle. The three standard limb leads being bipolar can bisect each other if redrawn forming a triaxial reference with each axis 60° apart with polarity and direction of three leads kept same as shown in Figure 3.3.

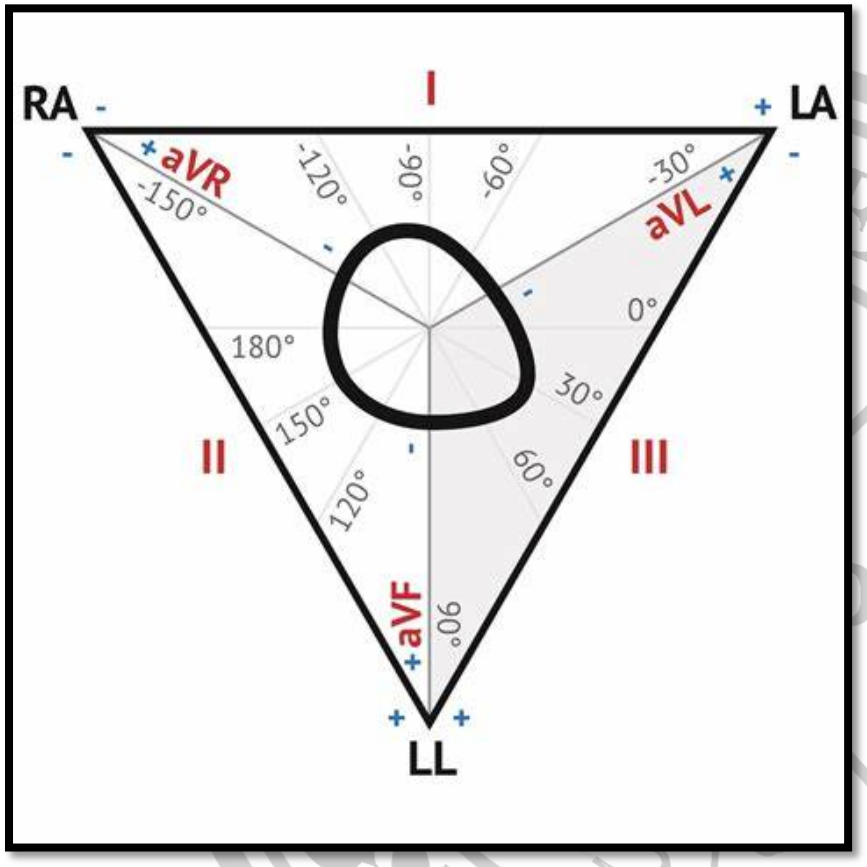

Figure 3.3. Einthoven triangle

As for the augmented leads, another triaxial reference can be drawn with each axis 60° apart and the heart being at the center of the triangle. If the two shapes are seen combined, it makes hexa-axial reference system with each axis 30° apart irrespective of lead type.

The significance of this triangle will be discussed further in chapter VI.

Chapter 4

Reading A Normal ECG

ECG is recorded on a paper by an ECG recording machine or electrocardiogram. The paper used is temperature sensitive. Therefore, the electrocardiogram has a stylus with a heated tip that runs over the paper at speed of 25mm per second ideally or 50mm per second depending on pre-determined settings. The paper roll for the ECG machine is available at a length of 20-30 meters. As the paper runs through the machine, a graph of horizontal and vertical lines is made alone the X-axis and Y axis.

The ECG strips consists of thin lines, 1mm apart from each other with a thicker line seen after every 5mm (Figure 4.1). The horizontal axis represents the time in seconds while, voltage is measured in millivolts on Y axis. During ECG tracing, the paper travels through the electrocardiogram at a speed of 25mm per second. This implies that in a duration of one second a total of 25 small squares are covered. Furthermore, a single small square has a width of 0.04 seconds which is 1/25 of the speed. Whereas, a large square having 5 small squares have a width of 0.04 x 5 or 0.2 seconds.

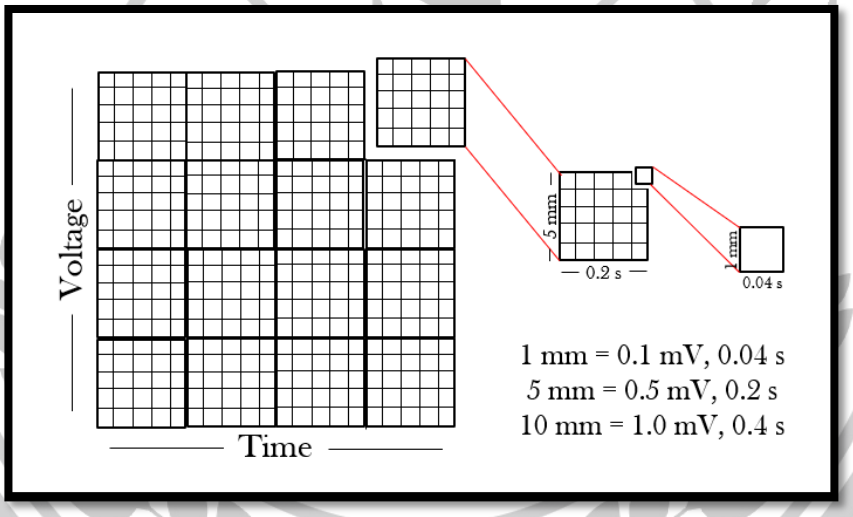

Figure 4.1 ECG paper with grids and values.

The width of an ECG interval is the length of deflection it covers in small squares on the X-axis multiplied by 0.04. So, if two small squares are equivalent to 0.04 x 2 =0.08 seconds, then 3 small squares would be 0.12 seconds and so on.

In case of vertical recordings, the electrocardiogram records these signals as 1 millivolt to 10 millimeters of deflection. Therefore, a small square represents 0.1mV and a large square 0.5mV on Y-axis. The significance of such a representation in correspondence to the base line gives us the height (positive, directly above the base line) or depth (negative, meaning under the base line) of the deflection that is equal to number of small squares on the Y-axis multiplied by 0.1mV. For example, if we have a vertical deflection of 3 small squares then multiply 3 with 0.1mV and the measure is 0.3mV. Moreover, a large square having 5 small squares have a voltage of 0.5 mV following the same rule along Y-axis. In simpler terms, the number of small squares above or under the isoelectric line can be multiplied with 0.1mV to get the voltage of the deflection as seen in figure 4.2.

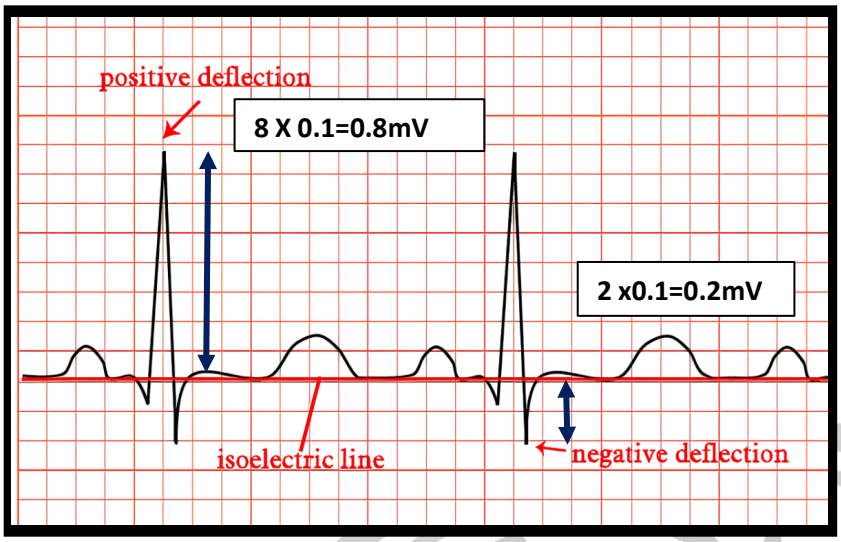

Figure 4.2. Measure of positive and deflection on ECG.

Normal ECG

An ECG consists of waves, intervals and segments. In order to read an ECG, one should be familiar with the normal values of each section.

- **ECG WAVES**

1. P-wave

P wave is a product of atrial depolarization recorded by the electrocardiogram. It is small round wave formed by the activation of both atriums. The SA node is present in the right atrium, hence the activation of right precedes the left. It is always upright in all leads with an exception to two as seen in figure 4.3. These include,

a) aVR

The direction of current away from the lead. Therefore, p wave, QRS complex and T wave are negative.

b) Lead V1

The p-wave is biphasic due to polarity difference at the two atriums. The right atrium generates a positive wave whereas, a negative deflection at left atrium is due to its activation in the reverse direction.

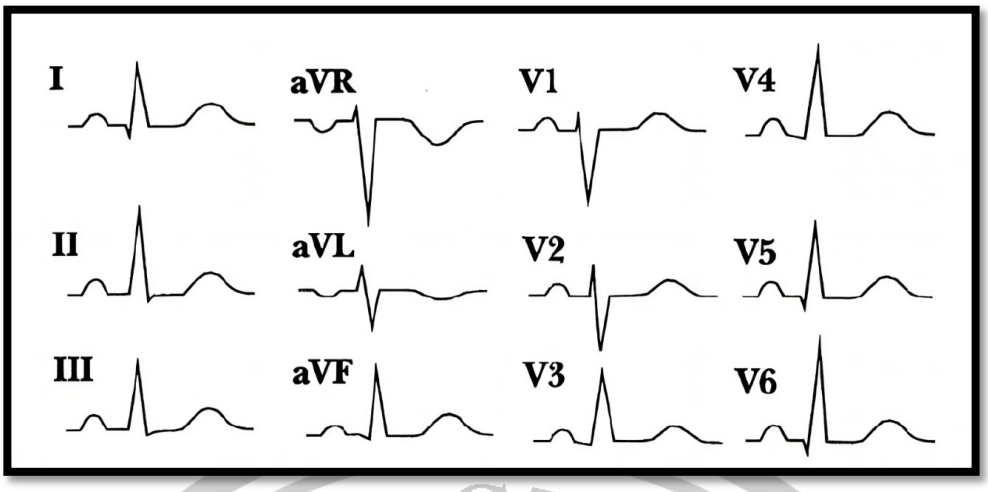

Figure 4.3 Normal P-waves in chest and limb leads.

Normal P-wave

- ◊ Single peak
- ◊ No notch or bifid
- ◊ Height < 2.5mm (0.25mV)
- ◊ Width < 2.5mm (0.10 seconds)

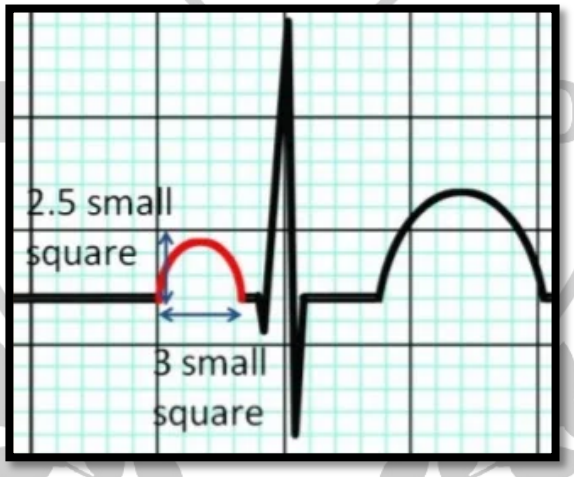

Figure 4.4 Normal P-wave and its measures.

2. QRS complex

On the ECG paper, QRS complex is the largest wave recorded. It is the representation of ventricular depolarization that is timed and synchronized as seen in figure 4.5. It is further sub divided into three waves that is

a) Q waves

Q waves are not present in all the leads but physiological Q waves can be observed in lead I, aVL, V5-V6. These waves represent interventricular septum moving in the direction away from left ventricular mass.

Criteria for physiological Q waves

- ◊ Width less than 0.04 seconds
- ◊ Height less than 25% of R wave

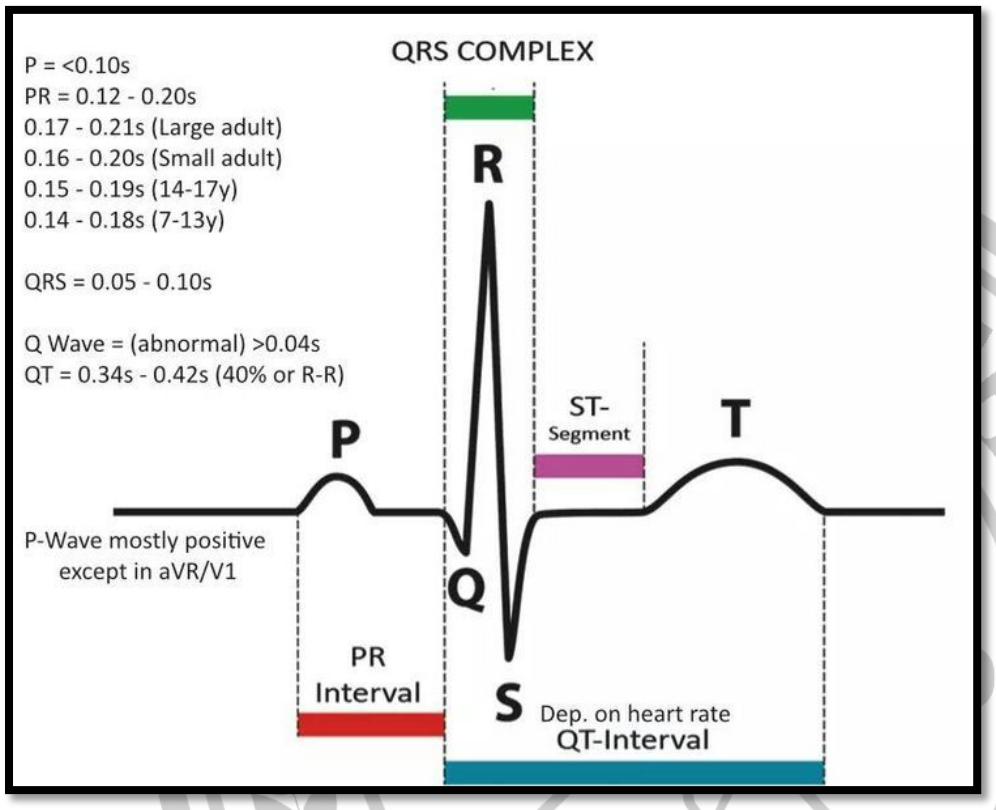

Figure 4.5. ECG with waves, intervals and segments.

The physiological Q waves appear in the leads based on orientation of left ventricle to it. If the left ventricle is in the direction of the lateral leads, then Q waves will be seen in lead I, aVR, V5-V6. However, if inferior leads then in lead II, III and aVF.

b) R wave

It is the tallest component of the QRS complex. Positive in all leads except for aVR as explained above. The height difference for R waves can be seen in limb and precordial leads due to distance from the current flow. R waves are having a voltage of at least 5mm in limb leads while ≥ to 10mm in precordial leads. Moreover, a slow rise in voltage can be appreciated as we move from V1 to V6. This phenomenon is called R wave progression. Under normal circumstances, the R wave height stays under 0.4mV or 4mm in lead V1 representing septal depolarization and below 2.5 mV (25mm) in lead V6 showing left ventricular depolarization. In V1, R wave is smaller than S wave whereas in V6, R wave is having more amplitude than s wave.

c) S wave

The QRS complex ends with S wave that is a negative deflection demonstrating ventricular depolarization of the remaining part. The S wave illustrates left ventricular depolarization in lead V1 while, in lead V6 right ventricular depolarization as s wave. Normally, the voltage of S wave remains under 0.7mV. The QRS

complex as a whole is the representation of Venticular depolarization with R and S waves in particular leads contributing to the overall amplitude of right and left ventricular depolarization. The duration corresponds to the time taken for the whole ventricle to become depolarized. Therefore, the QRS complex is a narrow wave with a sharp peak that measures less than 0.08 seconds on X-axis

3. T wave:

T wave following the QRS complex is a large rounded wave that represents ventricular repolarization. It is usually positive with exceptions to certain leads as in

- ◊ Lead aVR, always negative
- ◊ Lead V1, often inverted
- ◊ Lead V2, V3 and Lead III, occasionally negative

Amplitude like the R wave is around 5mm in Limb leads and under 10mm in precordial leads. As for atrial repolarization (Ta wave), the wave is hidden in the QRS complex due to the coincidence of timings with ventricular depolarization and low voltage. Therefore, Ta wave is not visible on a normal ECG.

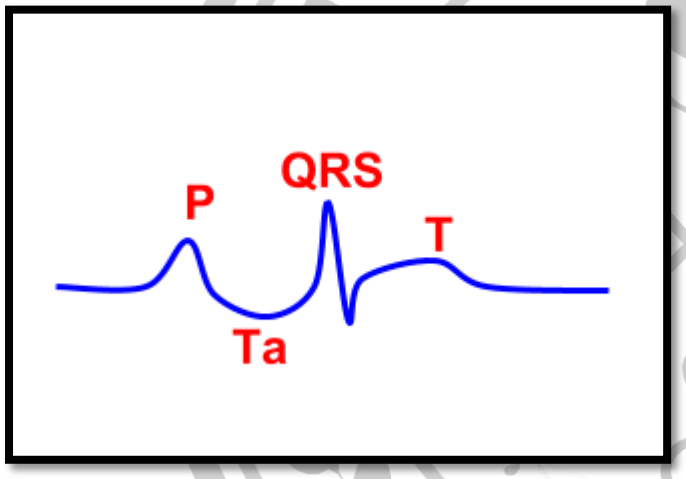

Figure 4.6. Ta wave representing atrial repolarization.

4. U wave

It is a rounded small wave after T wave, produced by gradual late repolarization of the Purkinje fibers at the intraventricular region. The wave is appreciated best in leads V2-V4 and is smaller in size in comparison to T wave.

- **ECG Segments**

1. **P-R Segment**

The part of isoelectric line between end of P wave to start of QRS complex is called P-R segment. It is the representation of conduction delay between atrial to ventricular depolarization.

2. **S-T Segment**

The part of isoelectric line between end of S wave to start of T wave is called S-T segment. It is the representation of duration between ventricular depolarization to complete repolarization. S-T depression is

measured in reference to a point that marks the start of S-T segment called junctional Point (J point). If the depression is below 0.5mm or more in precordial leads V2-V3 than it is an indicator of clinical significance such as myocardial ischemia.

- ## ECG Intervals

1. **P-R interval**

P-R interval is recorded on the X-axis from the beginning of P wave to start of QRS complex whether it's Q or R wave. The interval measures the duration of the atrioventricular (AV) conduction time, starting from atrial depolarization to conduction delay at the AV node, followed by beginning of ventricular depolarization. P-R interval is normally between 0.12 to 0.20 seconds, variations can be seen with fluctuations in heart rate. In simple terms, a faster heart rate will have shorter interval whereas, a slower heart rate with a longer interval. Moreover, the interval tends to be shorter in younger people (upper level at 0.18 sec) as compared to elderly with a bit longer duration (upper level at 0.22 sec).

2. **Q-T Interval**

The Q-T interval is measured from the start of Q wave to the end of T-wave on x axis. This interval represents ventricular repolarization and complete ventricular systole. It ranges of 0.35 to 0.43 seconds. It is dependent on three factors including age, gender and heart rate. Therefore, the interval is known to be shorter in younger age and female gender and hence considered normal under 0.42 seconds.

A corrected QT interval can be obtained using the formula,

Q-Tc= $\frac{Q-T}{\sqrt{R}-R}$

Q-T is Q-T interval and R-R is the square-root of R-R interval. For example, if the R-R interval of heart rate is 60 (25mm x 0.04 seconds = 1 second), Q-Tc interval is 60 beats per minute.

Chapter 5

Normal Heart Rate and Rhythm

Calculation of Heart Rate

Heart rate is defined as the number of times heart beats in a minute. In other words, it the number of cardiac cycles that are completed during a whole minute. Calculating the heart rate not only has its key clinical significance but is often among the first few things that are deduced from a patient's ECG. Two methods can be opted for calculating the heart rate,

1. **Six Second Interval Method**

The ECG paper runs at a speed of 25mm/seconds which is equal to 25 small squares (single square is 1/25=0.04 seconds) or 5 large squares (single large square is 0.04 x 5=0.2 seconds) in one second. In the ECG paper, after ever 5 large squares the vertical line appears to be thicker and taller than the other lines. By adding the distance of two such lines the duration is equal to 1 second. So, if we have six such lines then it is equal to six seconds. Next step is to count the number of QRS complex in this six second strip and multiply with 10 to find the heart rate in a minute.

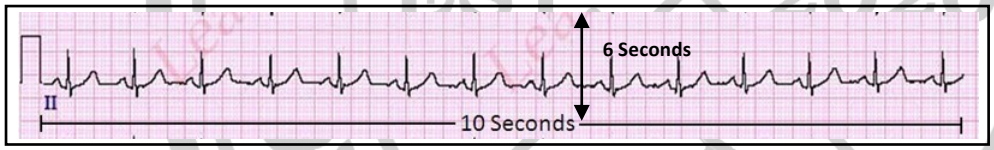

Figure 5.1. Calculation of heart rate from 6 second interval.

According to figure 5.1. there are 8 QRS complexes in 6 seconds therefore, 8 x 10= 80 beats per minute.

2. **R-R or P-P interval Method**

The ECG paper covers 25 small squares in one second at a speed of 25mm/second. In other words, 25 x 60= 1500 small squares in one minute or 60 seconds. R-R or P-P interval method is another easy way to calculate the heart rate. Moreover, the P-P interval and R-R intervals are same and can be used interchangeably.

Firstly, identify the peaks of two QRS complexes or P waves and then count the number of small squares between them. Looking at figure 5.1. the number of small squares between the first two QRS complexes is 18. Now, using the formula,

$$\text{Heart rate} = \frac{1500}{\text{Number of small squares}} = \frac{1500}{18} = 83 \text{ beats per minute}$$

But in case of large squares, replace 1500 by 300 (there are 5 large squares per second so, 5 x 60=300 large squares per minute).

Heart rate=$\frac{300}{\text{Number of Big squares}}$=$\frac{300}{3.5}$= approximately 83 beats per minute

Normal heart rate

The normal heart rate ranges from 60 to 100 beats per minute. Based on this range it can be classified into 3 categories,

1. Normal rate (60-100 beats/minute)
2. Bradycardia (< 60 beats/minute)
3. Tachycardia (>100 beats/minute)

P-P interval represents atrial rate while, R-R interval represents ventricular rate. Under normal circumstances, the two intervals are equal in measure and heart rate can be calculated by one of the above-mentioned methods. However, if the cardiac rhythm (P wave followed by QRS complex at regular intervals) is not regular then in that case, QRS complexes aren't equally spaced, therefore a mean of 5 or 10 R-R intervals are taken instead.

Heart Rhythm

A normal heart rhythm is defined as succession of all heart beats or all QRS complexes throughout the ECG. In plain words, QRS complex preceded by P wave and followed by T wave in a regular continuous pattern. The components of a heart rhythm include,

- ◊ Heart Rate
- ◊ Origin of cardiac rhythm
- ◊ Pattern of rhythm
- ◊ Atrioventricular conduction

1. Origin Of Cardiac Rhythm

The origin of electrical activity is through a pacemaker in heart. A pacemaker has the automaticity or ability to generate impulses that makes its way through the conductive system. The natural pacemaker is the SA node located in the right atrium that discharges impulses generated at a rate of 60-100 beats per minute. Therefore, SA node creates the rhythm called sinus rhythm.

In addition to SA node, there are cells in other parts of heart including atria, ventricles and atrioventricular region that can serve as pacemakers. Such cells are called ectopic or subsidiary pacemakers. However, unlike the original pacemaker they discharge impulses at a much slower rate.

- Atria or AV junction = 40-60 beats per minute
- Ventricular or Purkinje system = 20-40 beats per minute

The SA node governs the rhythm of heart by muting the ectopic pacemakers. Although, under special circumstances as insufficient SA nodal activity (sinus bradycardia or SA block) and genetic dominance of ectopic pathways, the subsidiary pacemakers can take up control over the heart rhythm. This rhythm is called escape rhythm. Therefore, when the natural conductive pathways slow down or fails, the escape

rhythm is the body's last resort to keep it alive until the original pacemaker starts working. Based on the location of alternative pacemakers, escape rhythm is classified as,

1. Junctional escape rhythm or idio-junctional rhythm.
2. Ventricular escape rhythm or idio-ventricular rhythm.

In case of genetic automaticity, a subsidiary pacemaker overrules the

SA node and acquire control over the heart rhythm, this is called idio-focal

Tachycardia. It can be further sub divided into three categories,

1) Atrial tachycardia
2) Junctional tachycardia
3) Ventricular tachycardia.

Based on site of origin, it is divided into 4 categories as shown in figure 5.2.

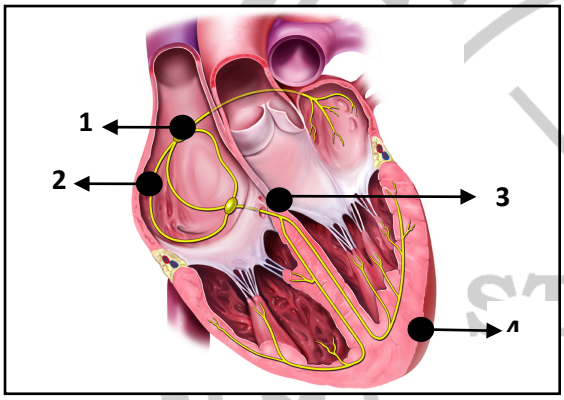

Figure 5.2. Heart showing Normal and Ectopic sites for pacemaker.

1) Sinus rhythm
2) Atrial rhythm
3) Junctional rhythm
4) Ventricular rhythm.

Let's discuss each type in detail

Sinus Rhythm

It is the normal rhythm seen on an ECG paper generated by SA nodal activity. All the waves are of normal morphology and in realtion to each other. To simplify, an upright P wave with normal PR interval followed by a narrow QRS complex in successions after one another is sinus rhythm.

Atrial Rhythm

It is a type of abnormal rhythm due to impulses produced by an ectopic pacemaker in atria other than SA node. It is identified by an inverted P wave, short PR interval but QRS complexes are normal.

Junctional Rhythm

In junctional rhythm, the atria and ventricles are activated simultaneously due to retrograde transmission of impulses through a junctional pacemaker. Therefore, P waves are inverted and can be either preceded by or merged in the QRS complex. The QRS complex like the atrial rhythm here is also normal due to intraventricular conduction.

Ventricular Rhythm

In ventricular rhythm, as the name implies the pacemaker lies at the ventricles. So, the atria are activated either by SA node producing a normal P wave or a retrograde conduction from ventricles giving inverted P waves. In such cases, the P wave is not easy to localize on ECG because is often buried in the QRS complex. The QRS complex is wide unlike the above-mentioned sub types due to slow activation of ventricular myocardium.

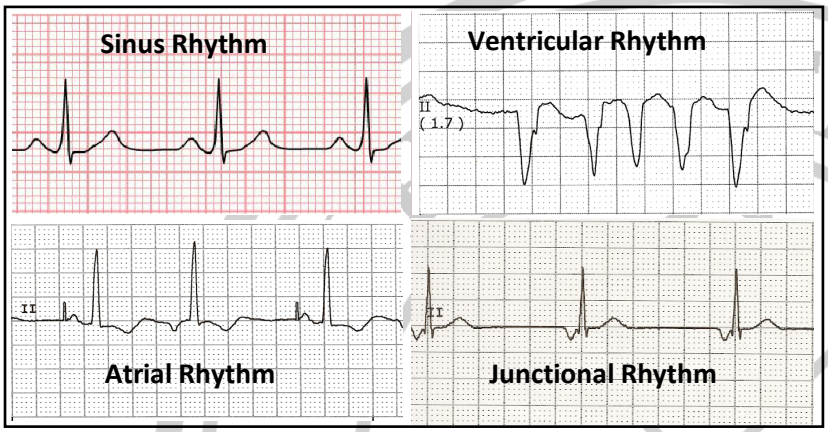

Figure 5.3. Different heart rhythms on ECG based on foci of origin

2. Pattern of Rhythm

The normal pattern of cardiac rhythm constitutes of equally spaced QRS complexes or beats. Under certain pathological events the rhythm can becomes irregular, meaning the QRS complexes are no longer equally spaced. On the basis of regularity, we can categorize the heart rhythm into three categories,

- ◊ Regular rhythm
- ◊ Regularly irregular
- ◊ Irregularly irregular

Regular rhythm includes sinus rhythm (normal, bradycardia and tachycardia) as well as fast rhythms like atrial, ventricular and junctional tachyarrhythmias.

Regularly irregular rhythm includes premature beats, regular pauses (certain heart blocks) and bigeminal rhythms or beats in pair.

Irregularly irregular rhythm constitutes of a bizarre ECG such as fibrillation. Fibrillation means functional disintegration of atrial and ventricular myocardial tissues in different stages of excitation and recovery. This leads to ineffective pumping. In rhythms like atrial fibrillation, numerous small fibrillatory waves (minimal fluctuations on ECG recorded from baseline) replaces the discrete P waves. QRS complexes appear normal with irregular R-R interval. Ventricular fibrillation has rapid onset of irregular QRS complexes of gross variation in shape, height and width. The waveforms of P wave, QRS complex and T waves are unappreciable and the isoelectric lines seems to flicker unequally.

3. Atrioventricular Conduction

The normal sequalae of cardiac activation is from SA node activating the atria to travelling down the AV node, making its way through the remainder of the conductive system to bring about ventricular activation. Atrial depolarization and ventricular depolarization are represented by P wave and QRS complex, respectively. Therefore, each P wave is followed by QRS complex and always in succession. Now, imagine a situation in which P waves and QRS complex aren't in regular sequence. As in, if the SA node led to atrial depolarization but the subsidiary controls ventricular depolarization from A-V junction. In such a scenario, P waves and QRS complex will become independent of each other and this predicament would be termed as A-V dissociation.

Examples,

1. In junctional or ventricular arrythmia, the ventricles beat at a rate faster than atria. Therefore, P waves are either dissociated or buried in the QRS complex. Also, R-R interval would be shorter than P-P interval with progressive shortening of P-R interval would also be note on ECG.
2. In complete A-V nodal block, atria beat independent of ventricles with visible segregation of P waves from QRS complex on ECG. However, P-P and R-R interval remain constant.

Rate site	**Idio-ventricular**	**Idio-junctional**	**Complete heart Block**
Atrial rate	**70-80 (normal)**	**70-80 (normal)**	**70-80 (normal)**
Ventricular rate	**70-100**	**-**	**20-40**
Junctional rate	**-**	**70-100**	**40-60**

Table 5. Arrythmias with AV dissociation.

In A-V dissociation, the SA node controls the atrial activation therefore, all produced P waves will be of normal morphology. In contrast, the shape of QRS complex depends on the subsidiary pacemaker. If the ectopic pacemaker is ventricular then QRS complexes will be wide while, in junctional the QRS complexes are normal and narrow.

Chapter 6

Heart Axis

The Electrical Axis of Heart

As the current flow through the conductive system of heart, numerous electrical signals or potentials are generated and disseminated in different directions. These are picked on by the electrodes with nearly 70-80% being annulled by equal and opposite impulses and only the net of forces is recoded on ECG. Moreover, the direction of axis is mean of all recoded vectors.

The Hexa-axial System

As previously explained in chapter 3, standard limb leads namely LI-LIII form a triangle with heart at its center, called Einthoven Triangle. The three axes are separated from each other by an angle of 60°. In comparison, the augmented limb leads also form a similar triangle with each axis 60° apart. Now, if the two triangles are placed on top of each other, it makes a hexa-axial reference system. In simple words, two tri-axial systems overlap to make a hexa-axial system with each axis 30° apart as seen in Figure 6.1.

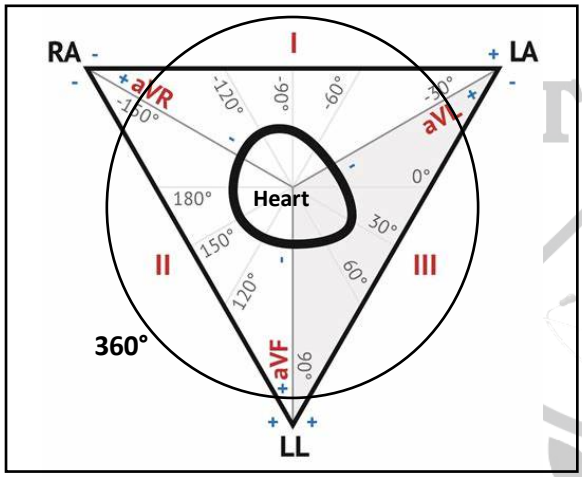

Figure 6.1. Hexa-axial reference system.

The hexa-axial system forms the basis of understanding the heart axis.

The QRS Axis

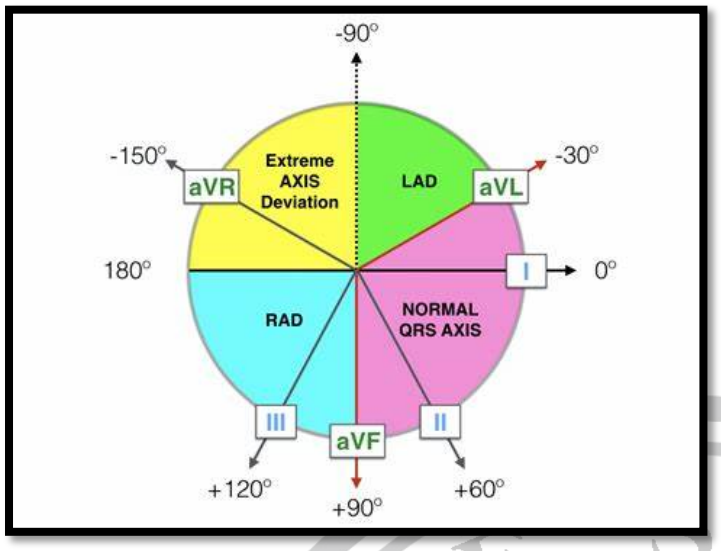

Figure 6.2. QRS axis

The QRS complex has the highest amplitude in ECG and helps in interpretation of axis using them. However, the QRS axis follow a few principals that needs to be discussed listed below,

1. The QRS axis represents the direction of electrical potentials as degrees on the hexa-axial system (Figure 6.2)
2. In any lead, the net deflection is the sum of its positive and negative vectors. For example, if positive deflection in R is +5 and negative in S is –2 then the net will be +3.
3. Based on relation of electrical impulses to any lead, it effects the recording on ECG.

◊ Parallel force, maximum deflection
◊ Oblique force, small deflection
◊ Perpendicular force, no deflection

For example, if axis is +90°, L1 will have the smallest deflection whereas, highest record in aVF. Therefore, maximum deflection in a lead determines its axis either positive or negative. So, if the major deflection in a lead is negative, means that particular lead is pointing towards the negative axis. To simplify let's consider an example,

Lead III shows a highest deflection of +7 or 7mm, so here the axis is +60°. Conversely, if it was –7 then axis will be -120°.

Determination Of Axis

Two commonly used methods to determine the QRS axis are as follows,

Method Number 1

1) Find the lead with smallest deflection.
2) Now find the lead perpendicular to first lead.
3) Observe the net deflection in the second lead
4) The axis is determined by second lead either positive or negative.

Example 1A

Smallest deflection lead is aVL

Perpendicular lead to aVL is LII

Maximum deflection in lead LII is positive, so axis here is + 60°

Example 1B

Smallest deflection lead is aVR

Perpendicular lead to aVL is LIII

Maximum deflection in lead LIII is negative, so axis here is -60°

Method Number 2

1) Find the net deflection in leads aVF and LI that are at right angle to each other.
2) Plot the net deflection of these leads between 0-10 on their respective axes.
3) Now draw a straight line from these points to where it cuts.
4) Join the circle's center to this point and then extend it to the circle's boundary.
5) The place where the line cuts the border, gives the QRS axis.

Example 2A

Deflection in limb lead LI is +5

Net deflection in aVF is 0

So, axis is 0°

Example 2B

Deflection in limb lead LI is +5

Net deflection in aVF is –5

So, axis is –45°

Method Number 3

Asses the direction of QRS deflection in leads LI and aVF to determine the axis as shown in table below.

Deflection in LI	Deflection in aVF	QRS axis
Positive	Positive	0 to +90°
Positive	Negative	0 to -90°
Negative	Positive	+90 to +180°
Negative	Negative	-90 to -180°

Table 6.1. QRS axis determined from leads LI and aVF

Abnormalities Of Axis

There are three different axes that can be picked on from reading the ECG. Each axis type holds its key clinical significance. Keeping the above QRS axis in mind, the table below list the three types of cardiac axis,

Axis Type	Degree	Causes
Normal QRS Axis	–30° to + 90°	
Right Axis Deviation	+ 90° to + 180°	1. Tall and lean adults or young children 2. Chronic lung disease 3. Ventricular ectopy 4. Hyperkalemia 5. Sodium-channel blocker toxicity 6. Pulmonary embolism 7. Congenital heart diseases (Ostium Secondum in ASD) 8. Right ventricular hypertrophy 9. Left posterior hemiblock 10. Lateral wall infarction
Left Axis Deviation	–30° to –90°	1. Obese adults 2. WPW syndrome 3. Cardiac pacing 4. Emphysema 5. Mechanical shift with expiration or raised diaphragm in conditions like pregnancy, ascites, abdominal tumor or organomegaly. 6. Congenital heart diseases (Ostium primum in ASD) 7. Left ventricular hypertrophy 8. Left anterior hemiblock 9. Inferior wall infarction
Extreme Right Axis Deviation Or Indeterminate QRS Axis NO MAN'S LAND North-west QRS Axis	–90° to –180°	1. Congenital heart disease 2. Left ventricular aneurysm 3. Misplacement or reversal of the limb leads 4. Ventricular pacing. 5. Ventricular tachycardias.

Table 6.2. Cardiac axis, its types and causes.

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Chapter 7

P waves

The P wave represents depolarization of atria. The P wave is normal and constant in morphology throughout the leads when the impulse originates from the SA node. It forms a positive deflection from the baseline and comes right before the QRS complex.

Normal P wave characteristics

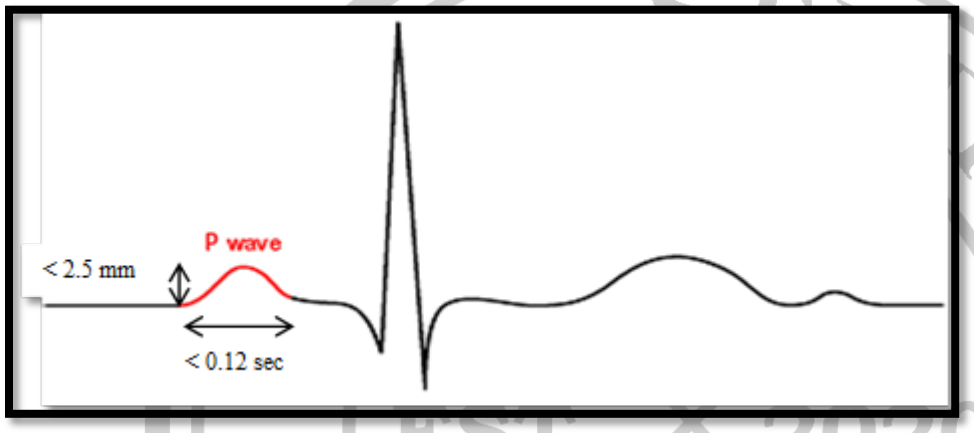


Figure 7.1. P wave with normal morphology

- Its amplitude is <2.5 mV that is less than 2.5 small squares on the vertical axis.
- Its duration is <0.12 sec that is less than 3 small squares on the horizontal axis.
- It usually has a single peak, rounded and upright without any notch.
- It has positive deflection (upright) in lead I, II, aVF, V2-V6, while negative deflection in lead aVR. However, in Lead V1 it is biphasic and variable in lead III and aVL.

Variations in P wave:

1. Tall P wave

P wave is formed as a result of both right and left atrial depolarization. Due to the presence of SA node in the right atrium, it is activated first giving rise to the first segment of P-wave. In instances where right atrium is enlarged, its deflection is superimposed on the left atrium and as a result tall P-wave is formed (Figure 7.2.)

- P-wave is labeled as tall when its height is more than 2.5 mm in lead II and aVF, while >1.5 mm in V1(biphasic lead) as shown in figure 7.3.
- It is also known as P pulmonale because pulmonary hypertension causes right atrial hypertrophy.
- Common causes of pulmonary hypertension are congenital heart disease, cor pulmonale and tricuspid stenosis.

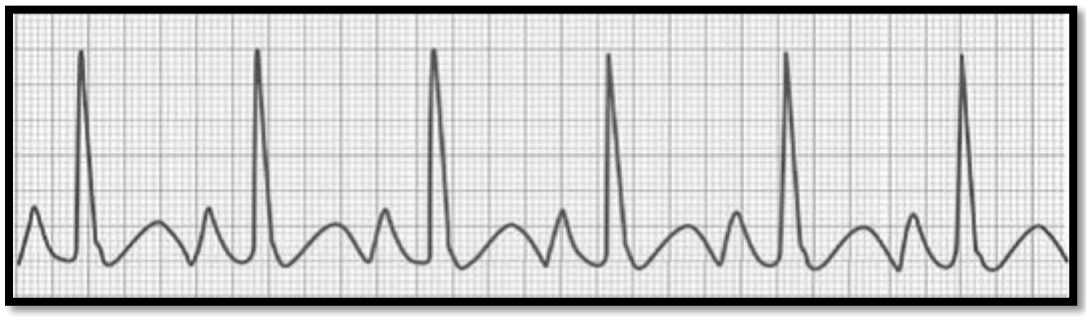


Figure 7.2. Tall peaked P waves in lead II

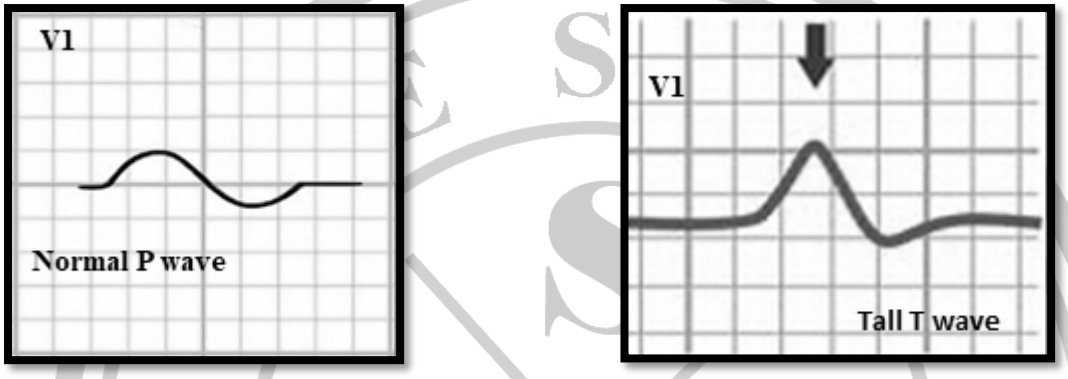


Figure 7.3. Biphasic P wave in lead V1

2. Broad P wave:

P wave is the resultant of left and right atrial activation. Initial 1/3rd of the P wave represents right atria whereas the last 1/3rd depict left atria and middle 1/3rd is the sum of the both atrium. In instances where left atrium is enlarged, its deflection is delayed compared to right atrium and as a result broad P wave is formed. This P wave is notched as well illustrating each of its left and right component.

- P wave is labelled as broad when its duration is more than 0.12 sec (3 small squares) in lead II and aVF. (Figure-3). Meanwhile in lead V1, the negative component will be deep (>1 mm) as seen in figure 4.
- It is also known as P mitrale because of its association with pathologies of mitral valve.
- Common causes of left atrial hypertrophy are mitral stenosis, mitral regurgitation, systemic hypertension, aortic stenosis and hypertrophic cardiomyopathy.

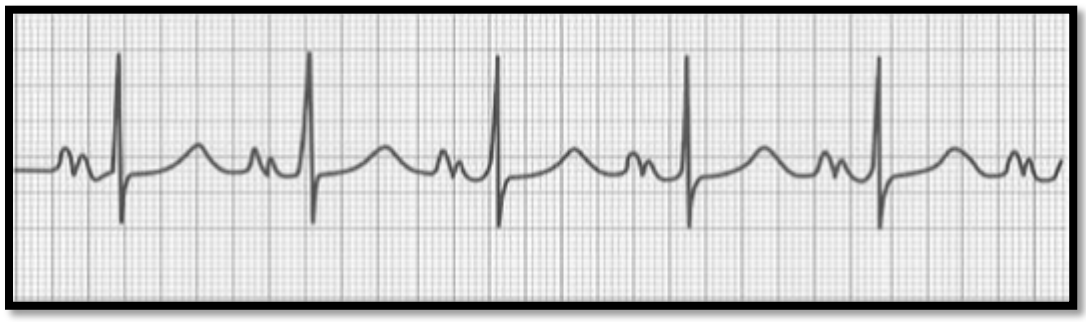


Figure 7.4. Broad, notched P wave in lead II

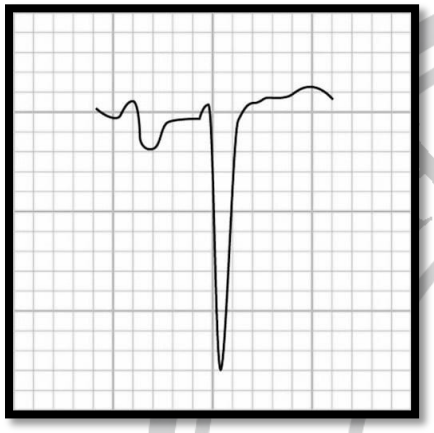


Figure 7.5. Broad P wave in V1

3. **Absent P wave:**

In few conditions, P waves are not visible.

Atrial fibrillation:

P waves are absent in the atrial fibrillation while sometimes small fibrillatory waves are noticed.

Atrial flutter:

Atrial flutter is famously known for its saw tooth impression where flutter waves are present instead of P waves.

Junctional rhythm:

Usually, impulse is generated from SA node, however in a junctional rhythm, it is originated from atrioventricular node or bundle of His. That is why, location of P wave is not normal and it can be completely absent, buried in the QRS complex or present before or after it.

Ventricular tachycardia:

In ventricular tachycardia, P wave is not appreciated as it hides under the QRS complex.

Hyperkalemia:

In addition to tall peaked T waves in hyperkalemia, P wave can be absent or low in amplitude.

4. **Inverted P wave**

As the depolarization from atria goes downward towards the inferior leads, the P wave is positive in lead II, III and aVF. Similarly, if the direction of atrial activation starts below and move upwards, P will be inverted in the same leads. P waves are inverted in the following conditions:

1. Junctional rhythm
2. By-pass tract

5. **Varying P wave:**

In a normal heart, atria follow a constant pattern of depolarization that leads to identical P waves throughout the electrocardiogram. However, this pattern changes when the impulse is not produced by the pacemaker of heart. In such cases, there is beat to beat variation in P wave. The below mentioned rhythms lead to variable P waves.

Wandering pacemaker rhythm:

As the name suggests, the pacemaker wanders from one spot to another and gives rise to changing P waves.

Multifocal atrial tachycardia:

Morphology of P wave varies with each beat because multiple foci in atria are sending impulses.

3 types of P waves can be appreciated in the above two rhythms.

- Ectopic P' waves
- Retrograde P' waves
- Fusion P'waves

Chapter 8

QRS Complex

QRS complex

The QRS complex represents depolarization of ventricles. It is produced as a result of synchronized right and left ventricle depolarization. In the absence of P wave before the QRS complex, impulse is said to be generated from the ventricles. QRS complex consist of Q wave, R wave and S wave.

Q wave: First negative deflection

R wave: Positive deflection

S wave: Negative deflection following R wave

Normal QRS complex characteristics:

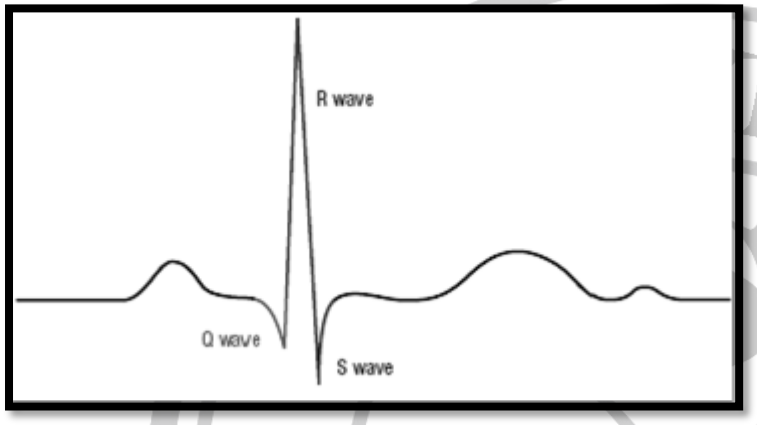


Figure 8.1. Normal QRS morphology.

- Its duration is 0.06 to 0.10 seconds roughly less than 3 small squares on horizontal axis.
- Its axis is between -30 to +90 degrees.
- It follows the P wave.
- It has positive deflection (upright) in lead I, II,III,aVL, aVF, V4-V6, while negative deflection in lead aVR, V1, V2 and biphasic in V3.
- Magnitude of R wave progressively increases from V1 to V6 that represents transition from right to left ventricle. The transition point lies normally at V3/V4.
- Its amplitude varies among all the leads. However, the voltage does not change in consecutive beats in an individual lead.
- Amplitude of R wave in V1 is equal to or less than 4mm and does not exceed 25mm in V5/V6.
- In V1, S wave is greater than R wave compared to V6 where its smaller than R wave.

Width of QRS complex:

Duration of normal QRS is between 70-100 ms. Its width gives clues to the QRS complex origin. It can be divided into narrow and broad complex.

Narrow complexes:

QRS is less than 100ms. Origin is supraventricular and arises from following area.

- Sino atrial node: Normal P wave

- Atria: abnormal P wave
- AV node: either no P wave or an abnormal P wave with a PR interval of less than 120 milliseconds.

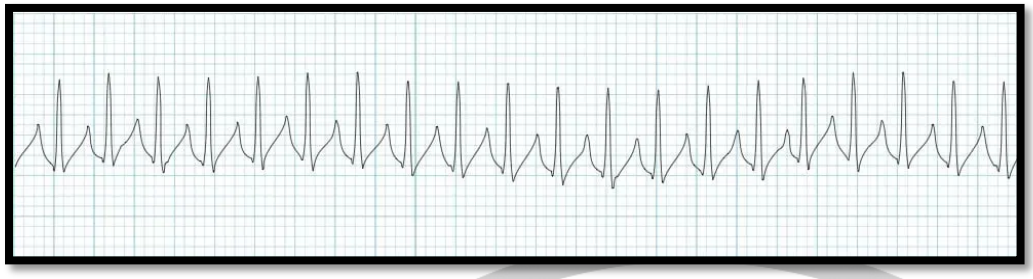


Figure 8.2. Junctional tachycardia ECG.

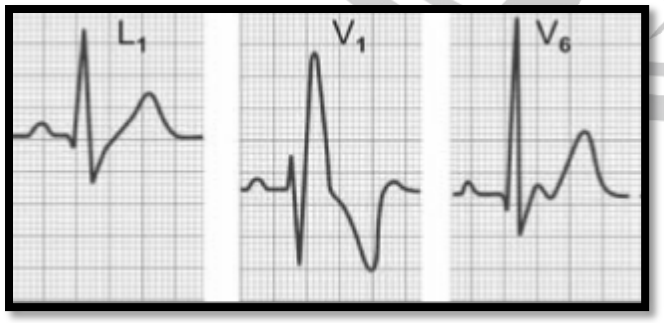
Broad complexes:

QRS is more than 100ms. Origin is ventricular or as a result of abnormal conduction of supraventricular complex due to

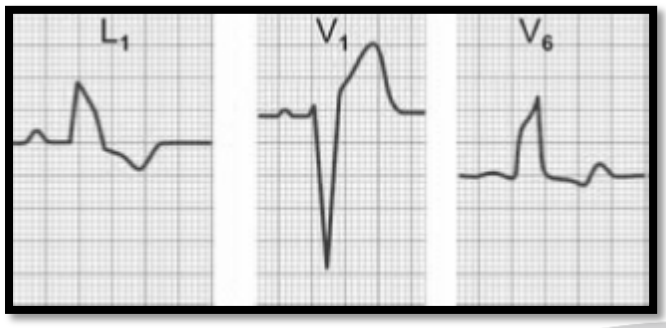
1. **Bundle branch block**

   There are 2 branches of bundle of His; right and left. If conduction through any of the branch is blocked, depolarization is not followed normally and leads to widening of QRS complex. There can be 2 types of such block, RBBB (right bundle branch block) or LBBB (left bundle branch block) according to the branch involved. In RBBB, right ventricle is activated via the myocardium instead of the fast conduction system that results in slow depolarization. On the other hand, left ventricle is depolarized slowly in LBBB.

RBBB: V1 shows RSR pattern and lateral leads show deep S waves.



LBBB: V1 shows dominant S wave with broad, notched R waves in the lateral leads.



2. **Poisoning with sodium-channel blocking agents (e.g., tricyclic antidepressants)**

   It leads to sinus tachycardia with tall R wave in aVR.

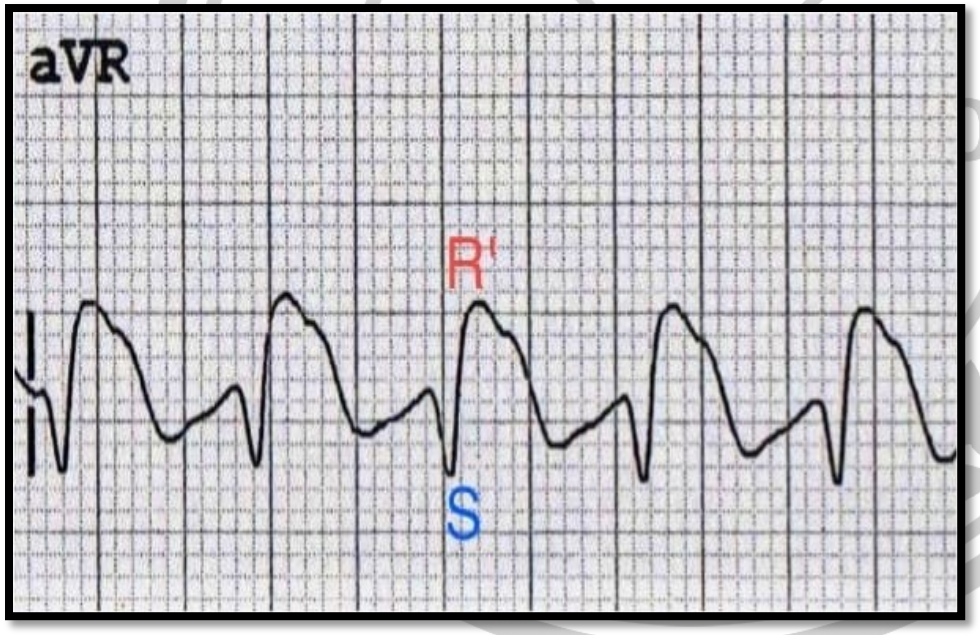


Figure 8.3. Sinus tachycardia with tall T waves.

3. **Wolff-Parkinson-White syndrome:**

   In WPW syndrome, conduction of impulse passes through an accessory pathway instead of AV node which results in premature depolarization of ventricles. It can put the patient at risk of arrhythmia.

It produces short PR interval and delta waves.

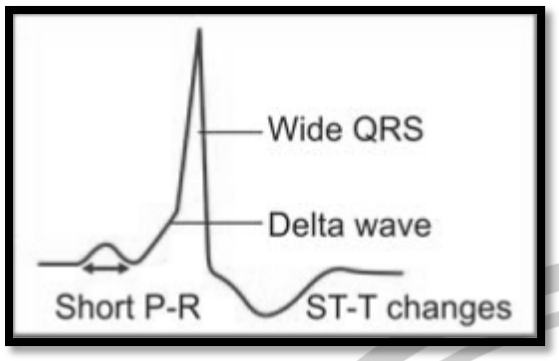


Figure 8.4. Wolf Parkinson White Syndrome ECG.

4. **Ventricular pacing:**

   A patient with pacemaker will have pacing spikes on ECG. These spikes vary according to the pacing mode, placement of lead, device threshold and presence of natural electrical activity.

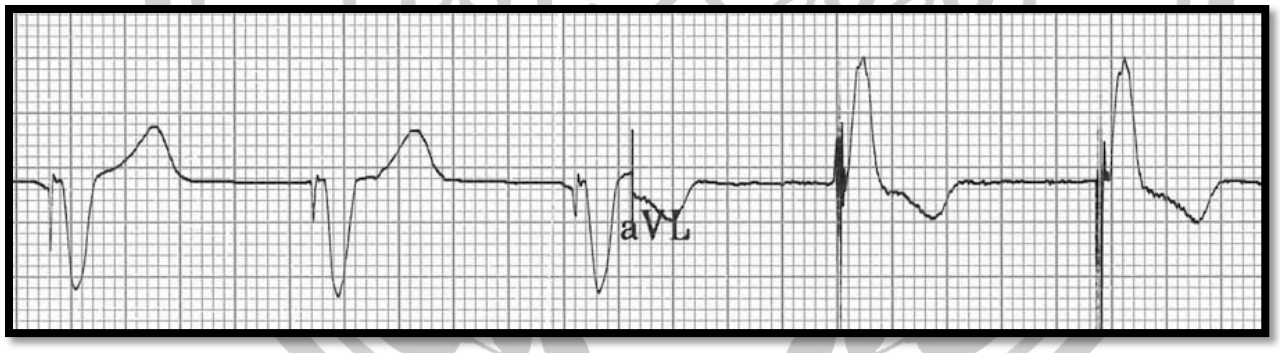


Figure 8.5. Ventricular pacing: Before QRS complex.

5. **Hypothermia:**

   Core body temperature of less than 35 °C is called hypothermia.

   ECG in such state shows bradycardia, Osborn waves, long QT and might have shivering artefact.

6. **Hyperkalemia:**

Serum K+ level of more than 5.2 mmol is called hyperkalemia. However, ECG changes mostly appear at ≥ 6.0 mmol. Tall T waves are usually the first sign on ECG. It can cause wide QRS as well.

High voltage QRS:

The most common cause of high voltage QRS complex is left ventricular hypertrophy (LVH). It can be normal in thin, athletic and <40 years of age individuals. To confirm LVH on ECG, Sokolov-Lyon criteria is used. If height of S wave in V1+ height of R wave in V5/V6 is more than 35mm it is LVH.

Low voltage QRS:

Amplitude of QRS less than 5mm in limb leads and less than 10mm in precordial leads is considered to be of low voltage. The causes of low voltage are given below

- Pleural effusion (most important)
- Obesity
- Emphysema/ pneumothorax
- Infiltrative /Connective tissue diseases like amyloidosis, haemochromatosis sarcoidosis, Constrictive pericarditis, Scleroderma
- Loss of viable myocardium as in MI, severe dilated cardiomyopathy

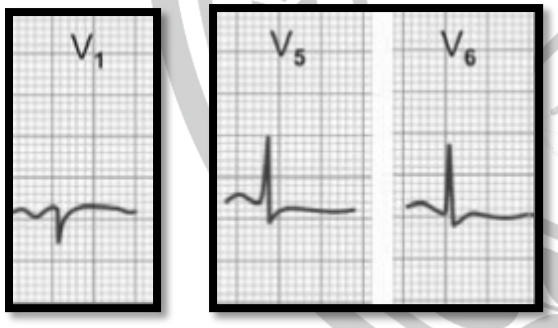


Figure 8.5. Low voltage QRS complexes.

Brugada syndrome:

It is a congenital condition due to the mutation in the gene that codes for sodium ion channels in the heart. It is notorious for causing sudden cardiac deaths. Definitive treatment is ICD. Brugada sign on ECG is considered diagnostic. It is the ST segment elevation of more than 2mm in >1 lead of V1-V3 with T wave inversion.

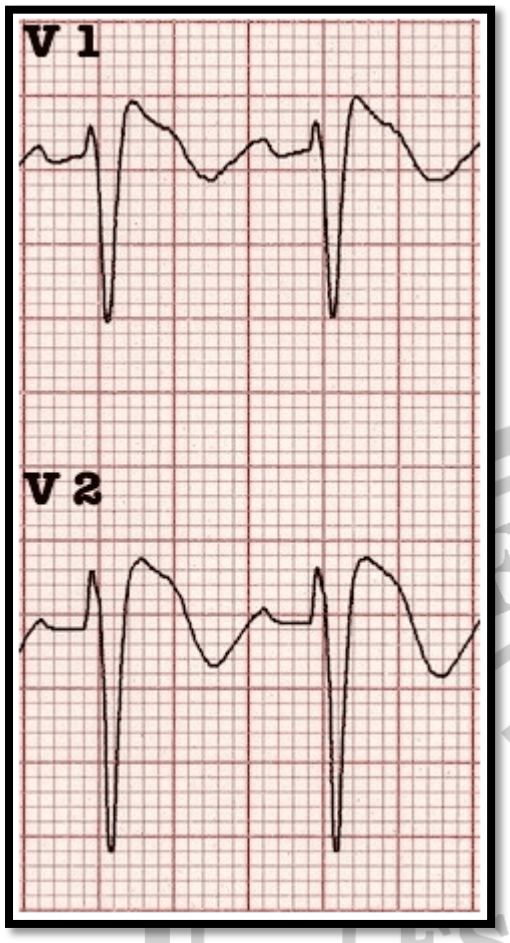


Figure 8.6. Brugada syndrome ECG findings in V1-V2.

Chapter 9

T waves

T wave represents ventricular repolarization. It is produced during the rapid phase of repolarization and is formed just after the QRS complex.

Normal T wave characteristics

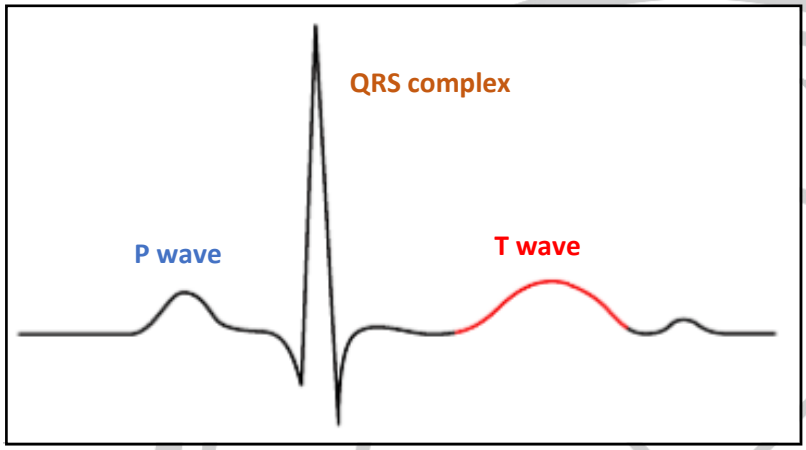


Figure 9.1. T wave with normal morphology.

- It is usually round and smooth.
- It has positive deflection (upright) in lead I, II, V3-V6, while negative deflection in lead aVR. However, it is variable in lead, aVF, aVL, III and V1-V2
- Its amplitude in limb leads is less than 5mm and less than 10mm in precordial leads.

Variation in T wave:

1. **Inverted T wave**

On the ECG graph, T wave is highly unstable constituent. Inversions of T wave are fairly common abnormality observed in practice. These T wave inversions together with ST segment depression are known as ST changes. Apart from T wave inversion, decrease in the amplitude of T wave or flattening has its significance.

Inverted T wave is considered normal in lead III. However, a new T wave inversion that is not present in previous ECGs is almost always abnormal.

T wave can be inverted due to a number of reasons as mentioned the in table (Table 9.1.). Hence why it has low specificity when making diagnosis.

Non- Specific Causes	Specific Causes
A. Physiological	A. Primary Cause
Heavy meal	Drug related: quinidine, digitalis
Anxiety	Metabolic: hypothermia, hypokalemia
Smoking	Myocardial: cardiomyopathy, myocarditis
Tachycardia	Pericardial: pericardial effusion, pericarditis
Hyperventilation	Ischemic: coronary insufficiency, infarction
B. Extra-cardiac disorders	B. Secondary abnormality
Systemic: hemorrhage, shock	Ventricular hypertrophy
Cranial: cerebrovascular accident	Bundle branch block
Abdominal: pancreatitis, cholecystitis	WPW syndrome
Respiratory: pulmonary embolism	
Endocrine: hypothyroidism	

Table 9.1. Causes of for inverted T waves.

Myocardial ischemia:

Clinically, T wave inversion is most importantly related to myocardial ischemia. These inversions occur in adjacent leads and distributed according to anatomical location of the infarction.

- Inferior leads: II, III, aVF
- Lateral leads: I, aVL, V5-6
- Anterior leads: V2-6

Inverted T waves of ischemic conditions can be differentiated on the basis of its features. In coronary insufficiency the T wave has following characteristics

- It is symmetrical
- It is peaked
- Both halves of T wave are mirror images of one another
- Apex lies midway
- Amplitude of T wave in V6 is less than V1 and greater in lead I than III

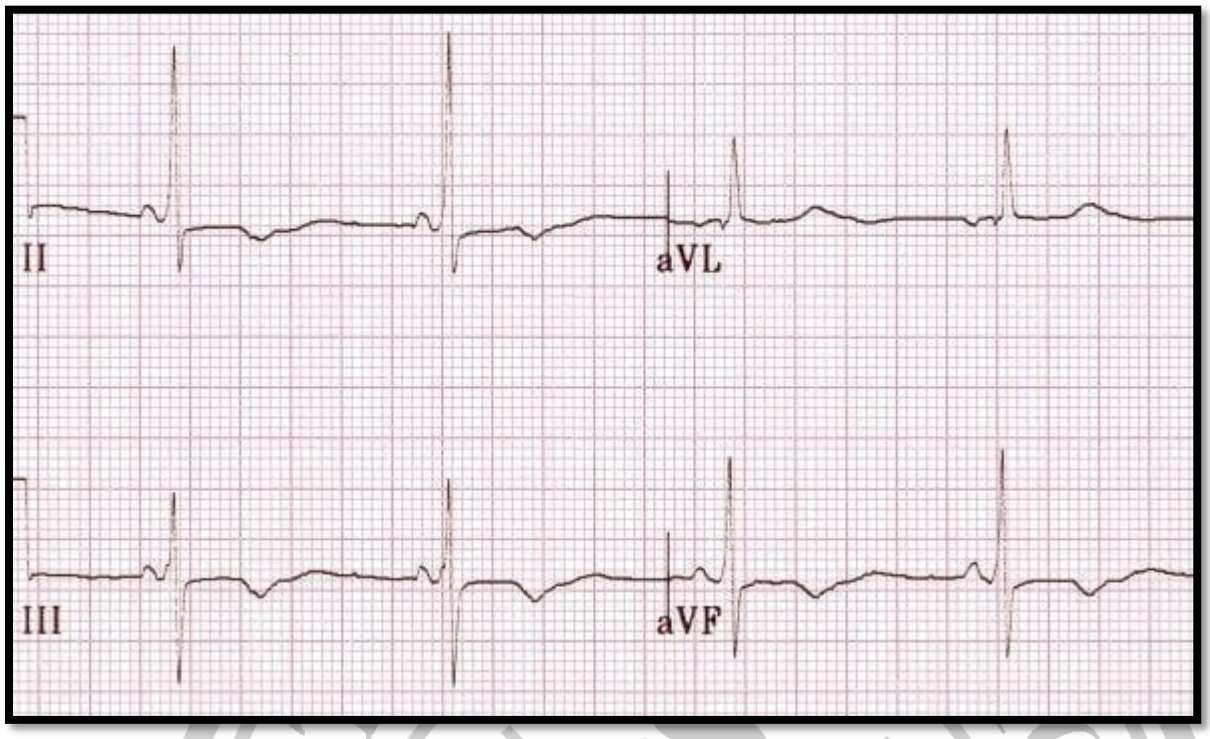


Figure 9.2. ECG with inverted T waves.

Hypokalemia:

Hypokalemia causes significant changes in the T wave. Low potassium in the blood can lead to either flattened, reduced in amplitude or inverted T waves. It is also related to the U wave prominence. U wave is a small deflection following the T wave as shown in the figure (Figure 9.4.). Prominent U wave following small T wave gives rise to 'camel-hump' effect on the ECG.

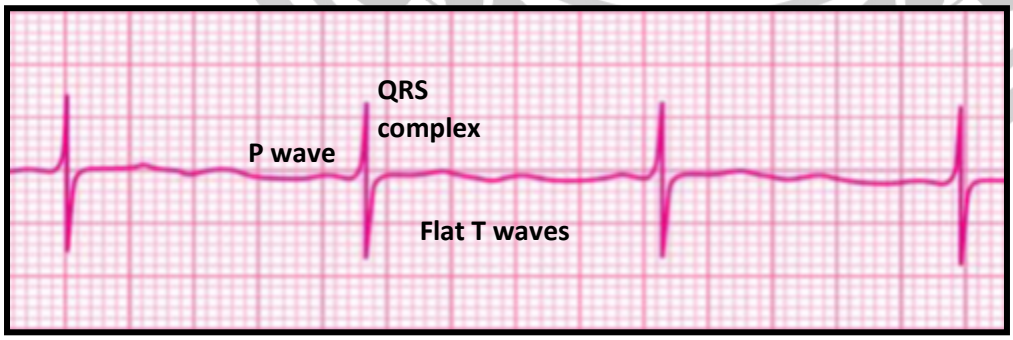


Figure 9.3. ECG with Flat T waves.

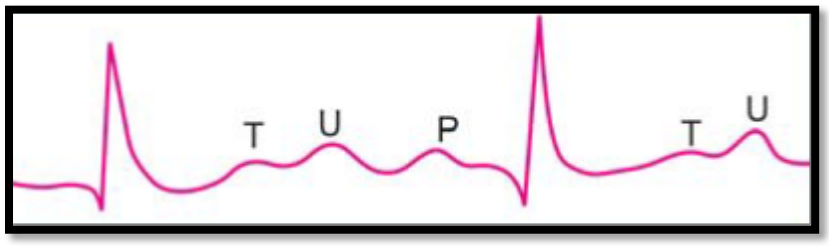


Figure 9.4. ECG with U waves.

Tall T wave:

T wave is said to be tall when its height is more than 5mm in limb leads while greater than 10mm in precordial leads.
T wave is tall in following conditions:

1. Hyperkalemia
2. Prinzmetal angina
3. Hyperacute infarction

The most common cause of tall T wave is hyperkalemia. It is usually called ‘tented T wave’ due to its shape as it is narrow at the base and peaked.

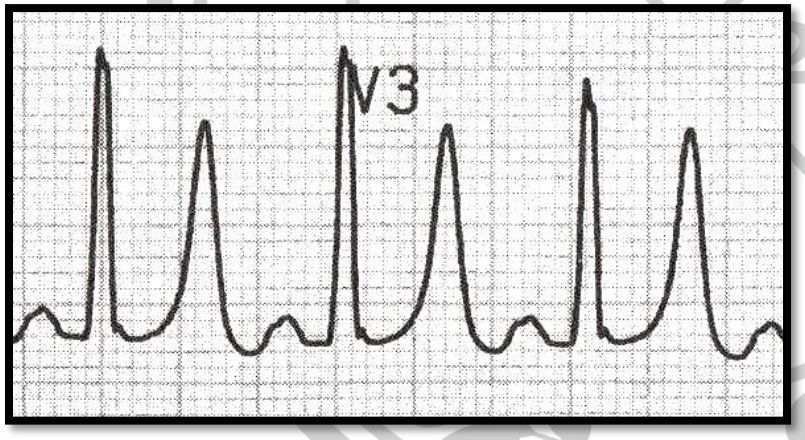


Figure 9.5. ECG with tall T waves.

Chapter 10

Pathologies of ECG segments

The two ECG segments that are P-R and S-T segments have been explained in detail in the previous chapters. This chapter will focus on pathologies of the segments while, keeping the basic knowledge in mind. As we already know both the segments under normal circumstances is at the same level as the baseline. Therefore, any deviation above or below the main segment is considered abnormal.

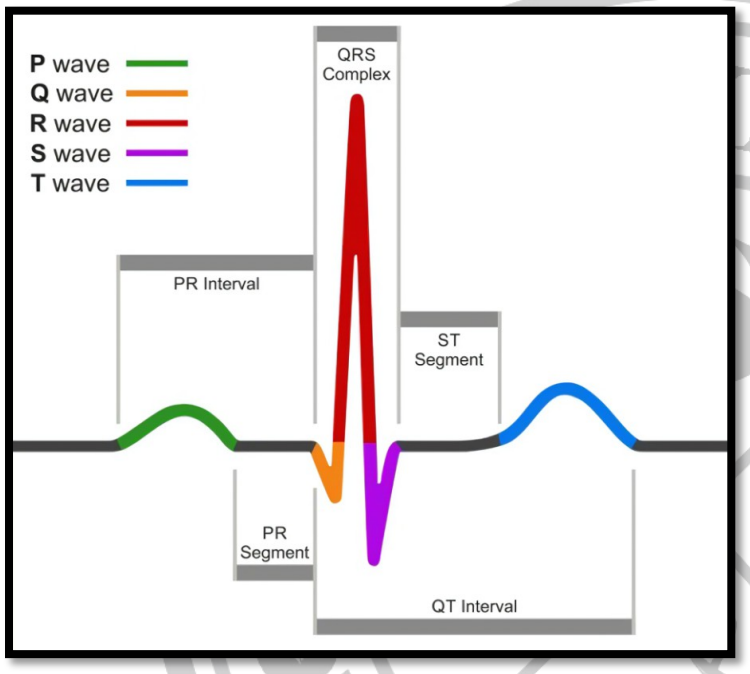


Figure 10.1 ECG with different Segments and Intervals.

P-R segment

P-R segment is the representation of AV nodal delay. P-R segment is normally along the isoelectric segment. Therefore, any deflection above or below can be clinically alarming.

- **P-R Segment Depression**

The P-R segment is the graphical illustration of atrial depolarization (P wave) to atrial repolarization (Ta wave). Ta wave is not visible on a normal ECG due a large QRS complex and coinciding timings with ventricular depolarization. However, if the Ta wave becomes evident, it will produce P-R depression.

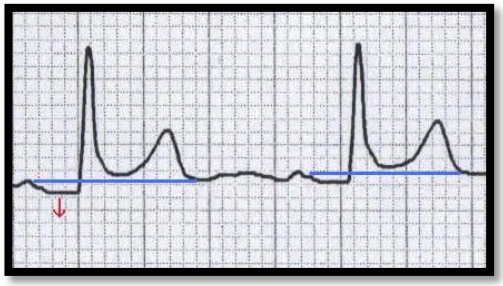


Figure 10.2 P-R depression due to prominent Ta wave.

Examples,

Primary causes

1. Pericarditis

An inflammatory condition of the outermost layer of heart called pericardium. The ECG is characterized by

- ◊ P-R segment depression
- ◊ Saddle shaped ST elevation
- ◊ PR elevation in aVR and V1 (Figure 10.3.)

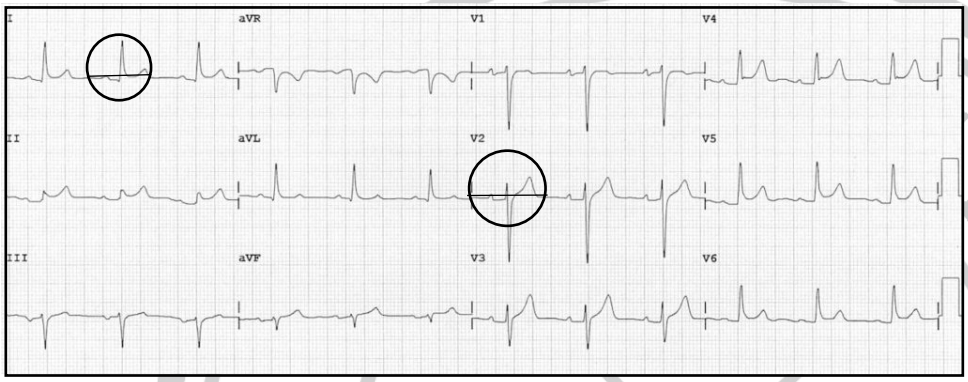


Figure 10.3. Pericarditis with P-R depression in Limb leads LI, aVL and chest leads V1-V6.

2. Atrial Ischemia

Changes in P-R segment following any acute coronary syndromes is an indicator of atrial ischemia. Such a finding is related myocardial infarction is related to poor prognosis and increase chances of heart blocks, arrythmias and worst-case scenario heart wall rupture.

Diagnosis of atrial ischemia, Liu's criteria include

- ◊ P-R segment elevation >0.5 mm in leads L I and V5-V6.
- ◊ P-R segment depression >0.5 mm in leads L II, III and V1-V2.
- ◊ P-R segment depression >1.5 mm in precordial leads whereas, >1.2 mm in standard limb leads. (Figure 10.4.)

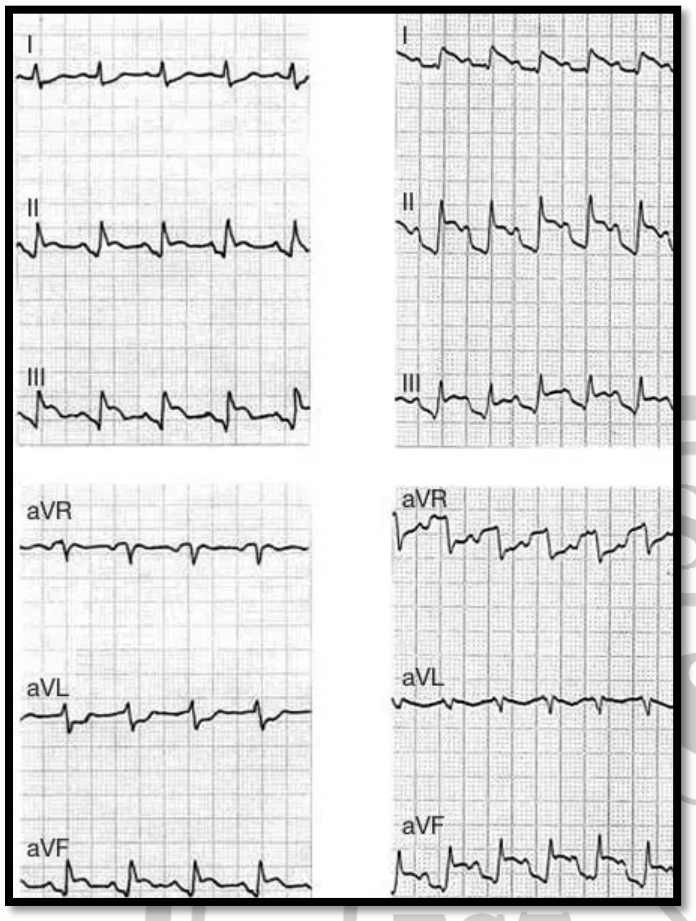


Figure 10.4. ECG for Atrial ischemia

3. Chest wall trauma

Trauma as a result of penetrating injury as in stab wound or cardiothoracic surgery that can precipitate to atrial injury or pericarditis.

Secondary causes

1. Atrial enlargement
2. Sinus Tachycardia

S-T segment

S-T segment is the demonstration of slow phase of ventricular repolarization starting from J point to the end of T wave. It is also in line with the isoelectric part as the P-R segment and any deflection above or below is considered pathological and requires urgent attention.

- **S-T segment depression**

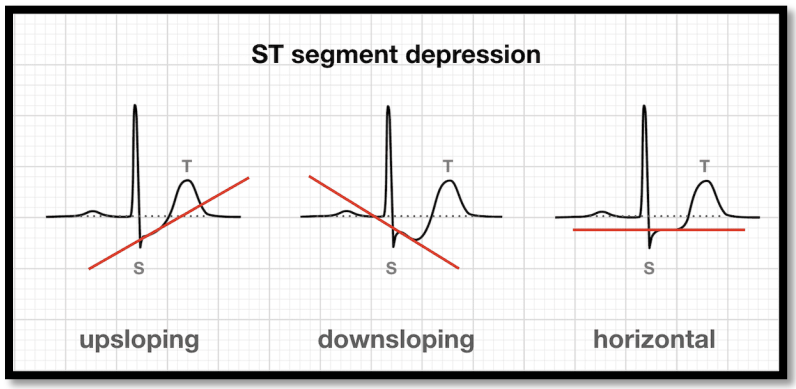


Figure 10.5. Types of S-T segment depression on ECG.

S-T segment depression of 0.5mm from the isoelectric main segment is important. This depression could be due to many reasons but always keep myocardial ischemia as the top differential due to its immediate medical intervention. S-T segment depression can vary in shape with down sloping and horizontal being the most common.

There are many causes for S-T segment depression including physiological and pathological with a few listed in table 10.1.

Specific		**Examples**
Primary	Medications	Digitalis and quinidine
	Cardiac	Myocarditis and cardiomyopathies
	Metabolic	Hypokalemia and hypothermia
	Ischemic	Vascular Insufficiency and infarction
Secondary		Ventricular hypotrophy with strain
		Bundle block
		Wolf Parkinson White syndrome
Nonspecific causes		
Physiological		Anxiety, tachycardia, hyperventilation
Systemic		Blood loss and shock
Abdominal		Pancreatitis and cholecystitis
Central nervous system (CNS)		Stroke
Respiratory		Pulmonary embolism

Table 10.1. Causes of S-T segment depression.

Let's discuss a few important examples,

- ➢ Digitalis toxicity

ECG findings include

- ◊ Short Q-T interval
- ◊ Downsloping S-T depression or Salvador Dali's moustache (figure 10.6.)
- ◊ Depression at J point
- ◊ Flattened or inverted or Biphasic T waves

Figure 10.6. Digitalis toxicity with characteristic Salvador Dali's moustache sign.

- Hypokalemia

ECG findings

- ◊ ST depression
- ◊ Prominent U wave
- ◊ Flat T wave
- ◊ Prolong P-R and Q-T/U interval

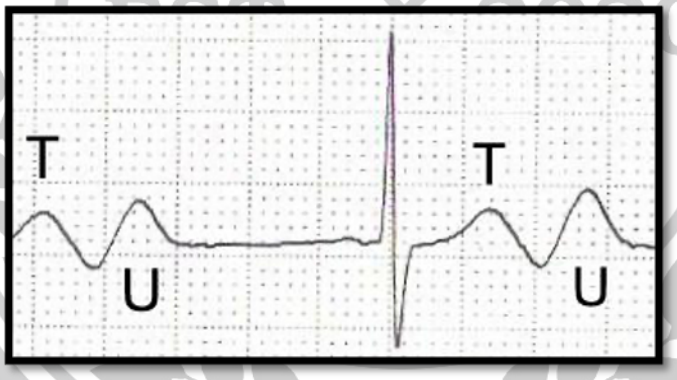


Figure 10.7. Hypokalemia-ECG Showing U waves and prolong Q-U interval

Myocardial ischemia and Infarction

Myocardial ischemia is known to be the most significant cause of ST depression where degree of depression greater than 1mm indicates severe vascular compromise. Moreover, S-T segment depression lacks diagnostic specificity due a range of cardiac and non- cardiac causes. Therefore, clinical symptoms and cardiac enzymes (raised cardiac markers e.g., creatinine phosphokinase) should be considered when establishing a diagnosis for Myocardial ischemia.

According to Task Force criteria in 2007, S-T depression is significant for myocardial ischemia if

1. Horizontal or downsloping changes in S-T segment is ≥ 0.5 mm at J point or ≥ 2 mm in contagious leads, indicative of ischemia
2. Upsloping changes in S-T segment are not specific for cardiac ischemia
3. ≥ 1 mm indicate worse prognosis

4. ≥ 2 mm in ≥ 3 leads indicate NSTEMI and mortality of 35% in 30 days.

Myocardial infarction based on Q waves can be,

1. Acute non-Q wave infarct
2. Acute Q wave infarct

Both types can produce an identical waveform on ECG but in case of non-Q waves there is prolong chest pain, raised CPK and ST-T changes in all serial ECGs. On the contrary, reciprocal depression is seen in the uninjured surface leads and S-T elevation in the affected ones for acute Q-wave myocardial infarction. For example, inferior wall MI shows S-T elevation in LII-III and aVF, while S-T depression in LI and aVL.

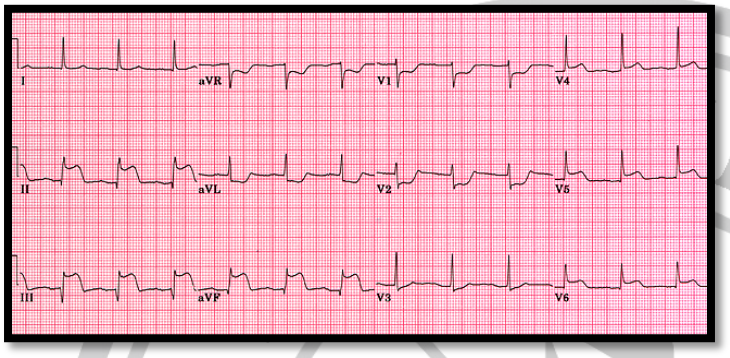


Figure 10.8. Inferior wall MI, S-T elevation in LII-III and aVF with reciprocal depression in LI and aVL.

Degree of S-T Depression

With larger amplitude of S-T depression, positivity on stress test (exercise test to assess cardiac functioning) would be greater. A depression of above 2 mm represents severe vascular insufficiency.

Shape of S-T Depression

S-T depression types from most significant to lowest include

◊ Rapid upstroke
◊ Slow upstroke
◊ Horizontal depression
◊ Downslope depression.

Timing of S-T Depression

Presence of S-T depression in the earlier part of stress indicate higher grade of positivity (vascular compromise) and vice versa.

Duration of S-T Depression

Longer the duration of S-T depression (exercise and rest period), more is the positivity on stress test. For instance, if depression continues for 8mins or longer is a marker of severe coronary artery disease.

Primary causes of S-T depression can be set apart from secondary by the shape of T waves. In primary, T waves are peaked and symmetrical whereas, in secondary they are blunt and asymmetrical.

- **S-T segment elevation**

S-T segment elevation of more than 1mm from the isoelectric line is clinically significant.

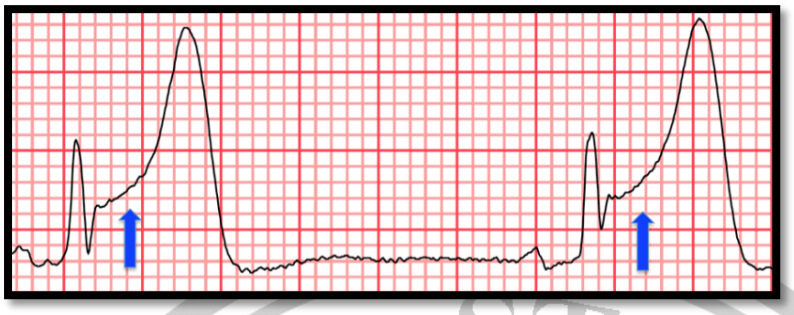


Figure 10.9. S-T segment elevation

Causes of S-T segment elevation

ECG finding of S-T segment elevation is more important than S-T segment depression as it is more specific. There are numerous cardiac causes for the elevation of S-T segment as listed in table 10.2. We will highlight a few in the upcoming text.

Causes	
Coronary vessel disease	Myocardial infarction (ACS)
	Prinzmetal angina (coronary vasospasm)
	Dressler's syndrome
Acute Pericarditis	
Ventricular aneurysmal rupture	
Left bundle branch block	
Ventricular paced rhythm	
Left ventricular hypertrophy	
Takotsubo Cardiomyopathy	
Brugada syndrome	
Raised intracranial pressure	
Bening early repolarization	

Table 10.2. Causes for S-T segment elevation.

1. Myocardial infarction

One of the most common causes of S-T segment elevation is myocardial infarction. Variations in ECG include,

- ◊ elevation of ST segment by more than 1mm
- ◊ T wave inversion
- ◊ Appearance of Q waves
- ◊ Loss of amplitude of R wave
- ◊ Arrythmias (abnormal heart rhythm)
- ◊ Conduction defects (Heart blocks)

◊ Reciprocal S-T depression in unaffected region leads
◊ Evolution of ECG with time.

Depending on the vessel and site of heart involved, leads will show S-T elevation in the affected area. For instance, S-T segment elevation will be seen in,

◊ Septal myocardial infarction → leads V1-V2
◊ Anterior wall myocardial infarction → leads V3-V4
◊ Lateral wall myocardial infarction → leads I, aVL, V5-6
◊ Inferior wall myocardial infarction → leads II, III, aVF
◊ Right ventricular myocardial infarction → leads V1, V4 (Right sided)
◊ Posterior myocardial infarction → leads V7-9

Based on duration following the myocardial infarction, ECG changes include,

◊ 0-6 hours → Acute MI
◊ 7 hours to 7 days → Recent MI
◊ 8 to 28 days → Evolved MI
◊ >29 days → healed MI

In acute phase, S-T segment slopes upwards with an upright T wave and no prominent Q wave. A week later in evolved phase, ECG shows S-T segment with upward convexity, inverted T waves, visible Q waves, and R wave of reduced amplitude.

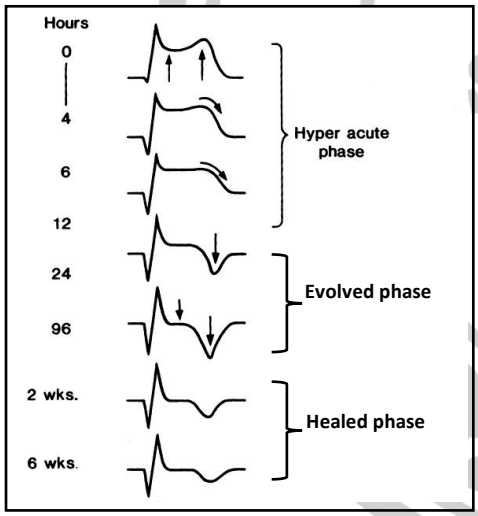


Figure 10.10. Evolution of ECG in Myocardial Infarction.

Furthermore, ECG findings for myocardial infarction is determined by factors like

a. Age of infarct; acute, evolved or healed
b. Type of infarct; thickness of heart tissue involved (subendocardial or transmural)
c. Heart wall involved; anterior or inferior wall
d. Underlying cause; myopathy, conduction defect

2. Prinzmetal's angina

ECG changes are identical to hyper acute phase of myocardial infarction (figure 10.8.) with slight variations including,

a. ECG changes don't evolve
b. Serum cardiac markers are not elevated

The principle behind it is coronary vasospasm and not thrombosis like myocardial infarction.

Triggers include intracoronary ergonovine injection.

3. Pericarditis

Pericarditis is a serious condition characterized by fever and chest pain. The pain is similar to myocardial infarction but sharp and aggravated with breathing and bending forward. The ECG features of pericarditis differentiating it from myocardial infarction are,

a. S-T segment elevation with upward concavity (saddle shaped) in all leads.
b. Upright T waves
c. P-R segment depression
d. Q waves absent
e. No reciprocal S-T depression
f. Sinus tachycardia
g. Amplitude of R- wave unchanged
h. Arrythmias and conduction disorders are rare.

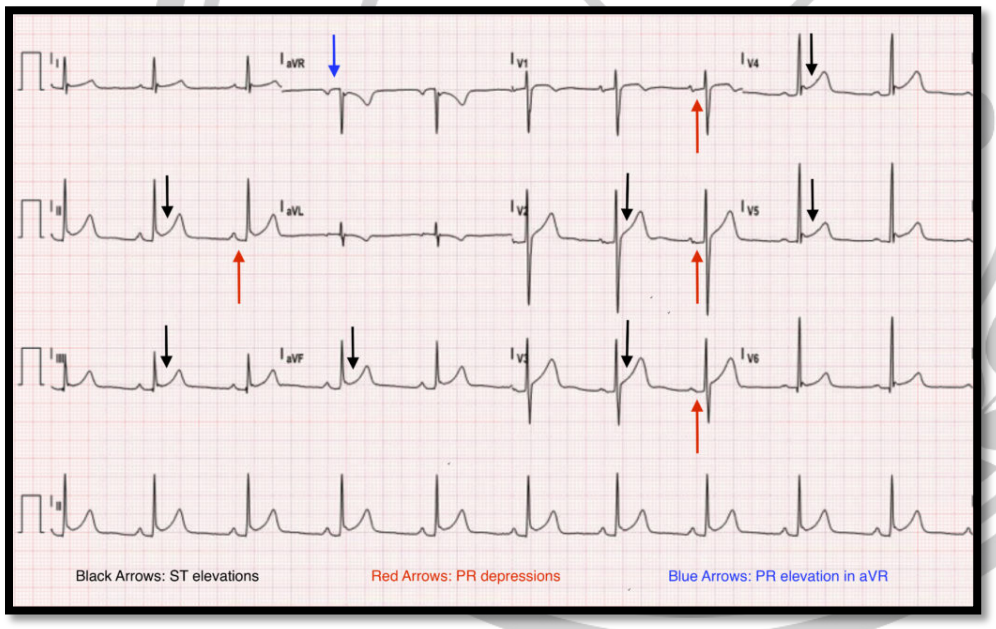


Figure 10.11. Pericarditis-ECG showing ST elevation and PR depression

4. Dressler's syndrome

Dressler syndrome is similar to pericarditis but it can appear within 2 weeks to a month following myocardial infarction, cardiac surgery, heart implant placement and chest trauma. However, unlike pericarditis it is an autoimmune phenomenon. Dressler syndrome has following features,

a. Fever and chest pain
b. Elevation of S-T segment
c. Raised serum ESR
d. Pericardial friction rub

e. Increase in pain during inspiration
f. Cardiac enzymes are normal
g. Good response to steroid therapy

5. Benign early repolarization (BER)

BER is a benign condition in which ECG shows S-T segment elevation in a healthy asymptomatic individual. It is more common in young African males with athletic build. Therefore, also called the athletic heart. These changes often return to normal after exercise. ECG findings of early repolarization syndrome are

a. Tall R waves in precordial leads V4-V6
b. Narrow and deep Q waves
c. Concave S-T segment elevation
d. Prominent J wave
e. Tall, upright and symmetrical T waves in all leads
f. Prominent U waves in mid-precordial (V2-5)

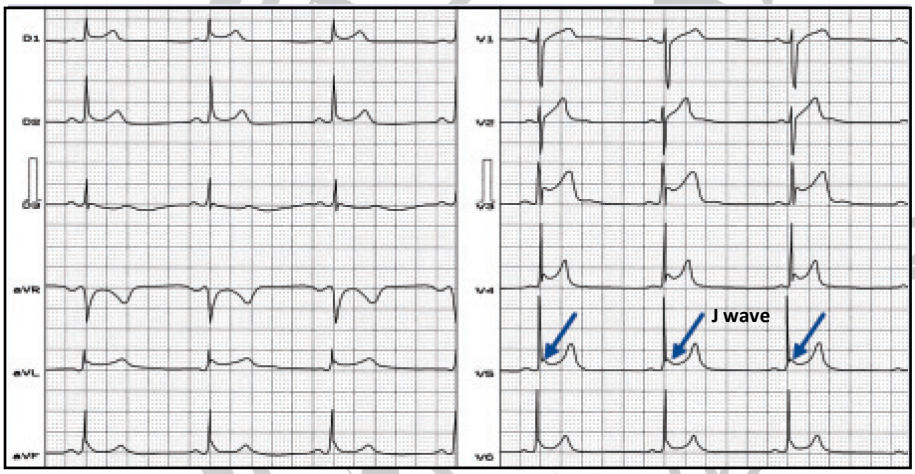


Figure 10.12. Early repolarization syndrome-ECG showing, ST elevation, J waves, tall T and R waves, and narrow Q waves.

It is the representation of portion of myocardium that undergoes early repolarization before depolarization can take place. J or Osborne waves are also seen in hypothermia and brugada syndrome.

Additional findings on ECG are:

a. Sinus bradycardia
b. Left ventricular hypertrophy
c. T wave inversion V1 to V3

Chapter 11

Pathologies of ECG intervals

P-R INTERVAL

P-R interval is measure of duration from start of P wave (atrial depolarization) to start of Q or R wave (ventricular depolarization). The length of this interval is the estimation of atrioventricular (AV) conduction time. AV conduction time is the time taken for impulses to bring atrial depolarization, delay at AV node and to transverse through the interventricular septum prior to ventricular activation.

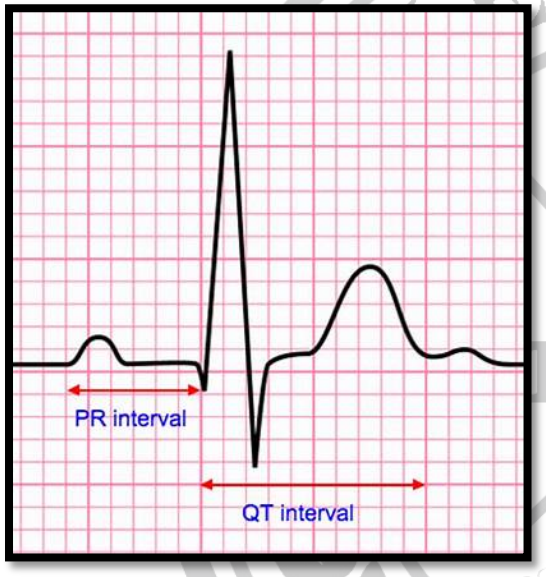


Figure 11.1. P-R and Q-T intervals.

Normally, P-R interval ranges from 0.12 to 0.20 seconds with AV nodal delay forming the major portion of this time. Abnormalities in this interval can divided into three categories,

1. Prolong P-R interval

If the P-R interval goes beyond 0.20 seconds in adults and 0.18 seconds in children, it is said to be long. Since, AV conduction time makes the most part of P-R interval therefore, an increase in AV nodal delay or first-degree heart block could be the possible factors. Causes of prolong P-R interval are,

- ◊ Physiological; Vagal stimulation in athletes, Valsalva maneuver and carotid sinus massage
- ◊ Infective; diphtheria and rheumatic heart disease
- ◊ Coronary artery disease with conduction blocks (fascicular block)
- ◊ Drugs; betablockers, calcium channel blockers and digoxin.

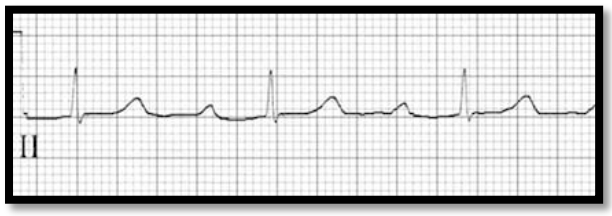


Figure 11.2. First degree heart block-prolong P-R interval

First degree heart block is characterized by delay in AV nodal conduction time by more than 0.2 seconds due to a blocked bundle branch from the above-mentioned causes. Therefore, a prolong P-R interval with normal a normal ECG will be seen (figure 11.2.). Such patients are at a risk of complete heart block and may be considered for prophylactic pacing.

2. Short P-R Interval

A P-R interval of less than 0.12 seconds reflects a decrease in AV nodal delay and increase in conduction process. In other words, a smaller AV nodal delay will present as a short P-R interval on ECG. Causative factors include,

- ◊ AV or junctional rhythm
- ◊ Pre-excitatory pathways
- ◊ Vago-lytic and anti-cholinergic drugs (atropine)

As previously explained in chapter 5, when the ectopic pacemaker in AV node or junction gains control over the rhythm, the ventricles are depolarized in a normal fashion but atria are stimulated from impulses, that are from below. In such a case, P waves are either preceded, followed or buried in the QRS complex. As a result, P-R interval becomes short.

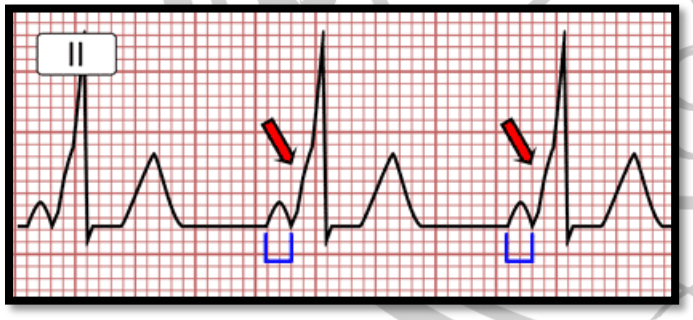


Figure 11.3. Junctional Rhythm-Short P-R interval:

Pre-excitatory pathways are accessory pathways that can directly transmit impulses from atrium to ventricles without passing the AV node. An example of such a pathway is bundle of Kent. Two types of impulses make its way from atrium to ventricle, namely slow and fast impulses. Under normal circumstances, both pass through the bundle of His. However, in the presence of bundle of Kent, the fast impulses travel through it and cause premature ventricular excitation. This condition is call Wolff Parkinson white syndrome. ECG findings include

- ◊ Short P-R interval
- ◊ Delta waves (slur on ascending limb of R wave)
- ◊ Wide QRS complex

- ◊ T wave inversion
- ◊ S-T segment depression

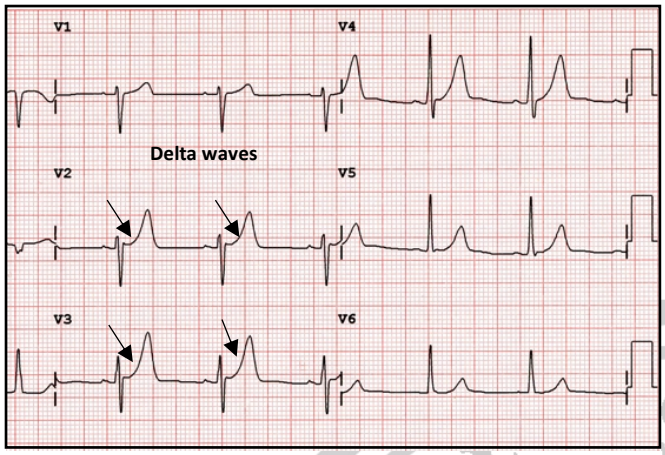


Figure 11.4. Wolff Parkinson White syndrome-delta waves.

Other accessory pathways include Lown-Ganong-Levine (LGL) syndrome, an atriofascicular tract (James's bypass) directly transmits impulses from atria to AV nodal pathway. This leads to a short P-R interval with no AV nodal delay but the QRS complexes are narrow due to normal activation of ventricles by bundle of His.

3. Variable P-R Interval

Changes in P-R interval with each beat is termed as variable P-R interval. Usually, atrial depolarization (P wave) is followed by ventricular depolarization (QRS complex) in a sequential manner so the P-R interval remains constant throughout. Although, in some pathological conditions the atria and ventricles can become independent of each other or if the AV conduction time varies with each beat, the P-R interval will not stay consistent. Causes of variable P-R interval are,

- ◊ Heart blocks (1st ,2nd and 3rd degree AV block)
- ◊ Junctional rhythm
- ◊ Wandering pacemaker (rhythm controlled by pacemaker other than SA node in atria)
- ◊ Multifocal atrial tachycardia

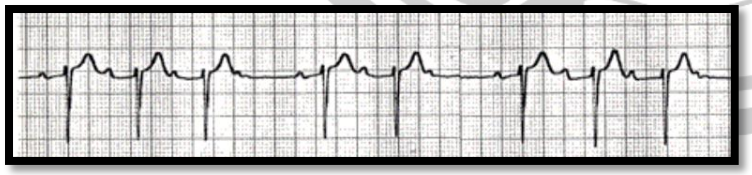


Figure 11.5. Variable P-R Interval-Second degree heart block.

Q-T INTERVAL

Q-T interval is measure of duration from start of depolarization (Q wave) to end of repolarization (T wave) of both ventricles. In short, it is the total duration of electrical systole in ventricles. The interval ranges from

0.35 to 0.43 seconds with slight a variation depending on age, gender, vagal tone, heart rate and medications.

The Q-Tc interval can be calculated using Bazett's formula:

$$\text{Q-Tc} = \frac{\text{Q}-\text{T}}{\sqrt{\text{R}-\text{R}}}$$

- Q-T is the duration of Q-T interval
- R-R is square-root of R-R interval

As previously explained in chapter 4. For example, if R-R interval is 1 second or 25mm then the value for $\sqrt{\text{R}} - \text{R}$ is 1 and Q-Tc will be equal to Q-T interval as seen below,

$$\text{Q-Tc} = \frac{\text{Q}-\text{T}}{\sqrt{\text{R}}-\text{R}} = \frac{\text{Q}-\text{T}}{1} = \text{Q} - \text{T}$$

This holds true if the heart is beating at a rate of sixty in one minute. Other formulas to calculate corrected Q-T interval includes,

◊ Fridericia formula,

$$\text{Q-Tc} = \frac{\text{Q}-\text{T}}{\sqrt[3]{\text{R}}-\text{R}}$$

◊ Framingham formula,

$$\text{Q-Tc} = \text{Q-T} + 0.154\ (1 - \text{RR})$$

Ideally, Q-T Interval remains constant in all leads in a healthy individual, but two exceptions can be seen in which it is either too long or too short.

1. Short Q-T interval

If the duration of Q-T interval is less than 0.35 seconds, it is considered to be short.

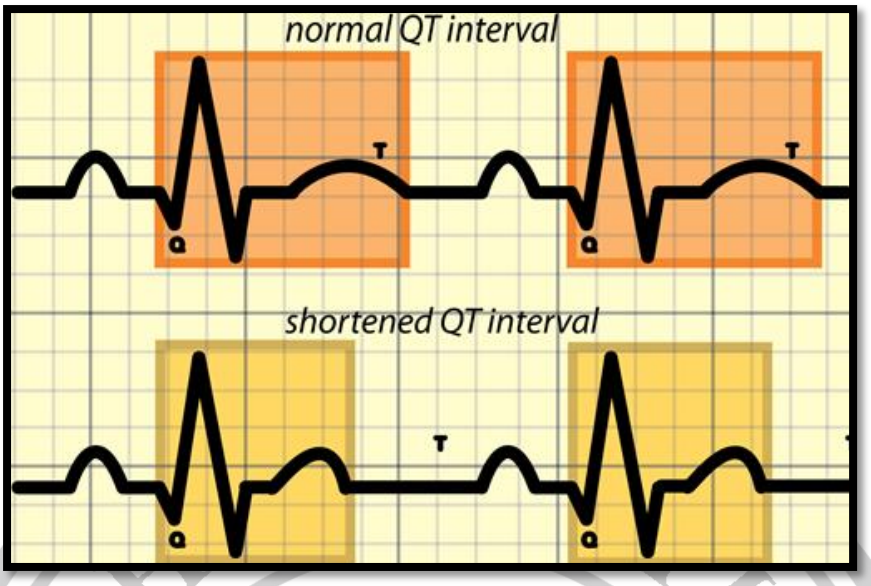


Figure 11.6. Short Q-T interval.

Causes for short Q-T interval includes,

- ◊ Metabolic → Acidosis
- ◊ Medication → Digoxin (chapter 10)
- ◊ Electrolyte imbalances → Hyperkalemia and hypercalcemia
- ◊ Temperature extremes → Hyperthermia
- ◊ Genetic → Congenital short Q-T syndrome

In conditions like hyperkalemia, an early repolarization leads to following changes on ECG

1. Short Q-T interval
2. Wide QRS complex
3. Tall T waves
4. Diminished P waves

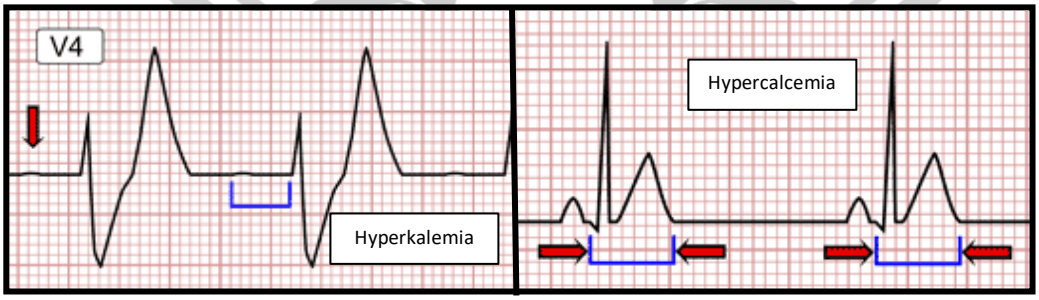


Figure 11.7. Hypercalcemia and Hyperkalemia-ECG changes

On the contrary, Hypercalcemia also has a short Q-T interval but there are no changes in the shape of P waves, QRS complex and T waves.

2. Prolong Q-T interval

If the corrected Q-T interval is more than 0.43 seconds than the interval is labelled prolonged.

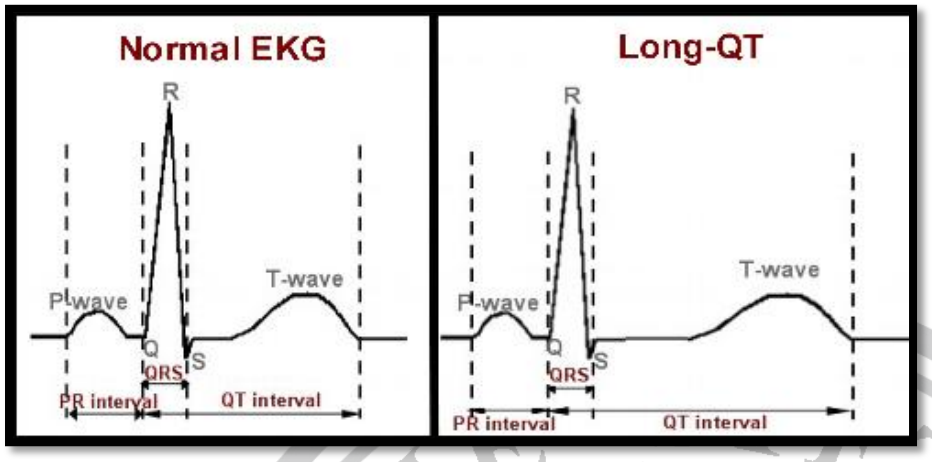


Figure 11.8. Prolonged Q-T interval.

Causes of a prolong Q-Tc interval are of two types;

◊ Congenital
→ Romano-Ward syndrome (An autosomal dominant condition without deafness)
→ Jerwell-Lange-Nielson syndrome (autosomal recessive with deafness)

◊ Acquired
→ Electrolyte imbalances like Hypokalemia and Hypocalcemia
→ Medications like quinidine and amiodarone
→ Ischemic heart diseases like myocardial infarction
→ Acute inflammatory conditions like viral myocarditis and rheumatic fever
→ CNS causes like head injury and intracranial hemorrhage
→ Arrythmias like A-V block and sinus bradycardia.
→ Anti-Psychotic drugs like tricyclic antidepressants (TCAs)
→ Miscellaneous drugs like terfenadine and cisapride.

In hypokalemia, due to low serum potassium levels the repolarization phenomenon is delayed leading to formation of long Q-T intervals. In addition, T waves are flattened and prominent U wave can be appreciated on ECG (Figure 11.9.). Sometime these U waves are mistaken for Q wave hence, Q-U interval is measured that will lead to pseudo-prolongation of Q-T interval (Figure 11.10.). Moreover, in hypocalcemia there is true prolongation of Q-T interval with no other changes in ECG.

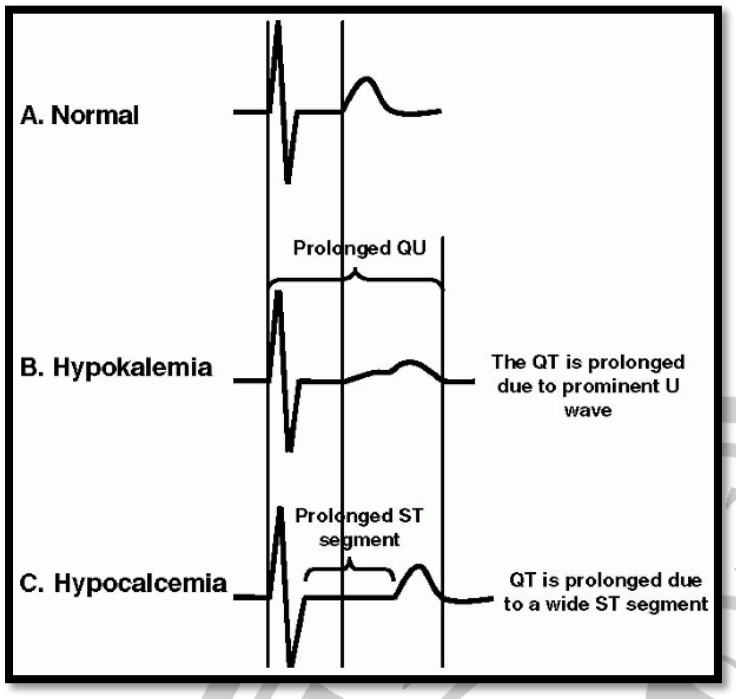


Figure 11.9. Prolonged QT interval in electrolyte deficiency.

Antiarrhythmic drugs like quinidine, procainamide and amiodarone can cause prolong QT interval and wide QRS complex (≥25% in width from baseline) by block the potassium rectifier channels, delaying repolarization. Other medications including antihistamines, terbinafine and prokinetic cisapride.

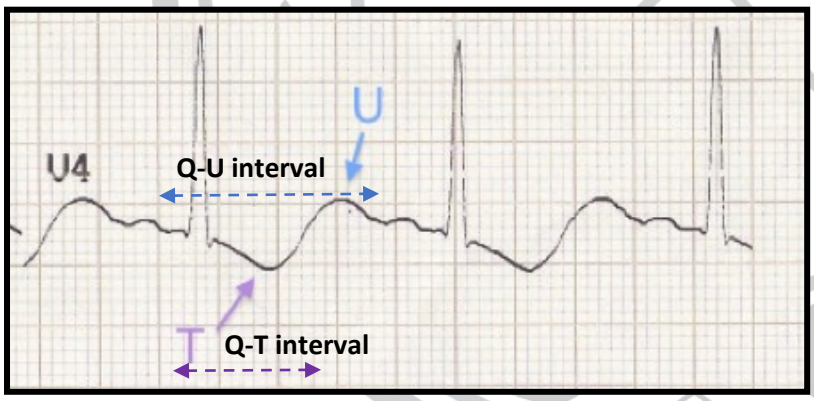


Figure 11.10. Pseudo-prolong Q-T interval in Hypokalemia

Clinical significance of prolong Q-T interval lies in risk of development of polymorphic ventricular tachycardia (Torsade de pointes or torsion around a point). Such an arrythmia needs active management before it turns into ventricular fibrillation leading to cardia arrest. Therefore, it is important to identify and stop such manifestations in ECG before they can become life threatening.

Chapter 12

Abnormal Heart Rate and Rhythm

Premature beats

A beat produced by early firing of an irritable autonomous foci other than natural pacemaker (SA- node) is called premature contractions or complexes. Based on foci of origin, it's of three types,

◊ Atrial premature beats

Premature atrial contractions (PACs) are characterized by the following ECG findings,

a. Upright P wave before normal sinus wave
b. Abnormal shape of P wave
c. Normal QRS complex
d. Pause after PACs due to SA nodal suppression.

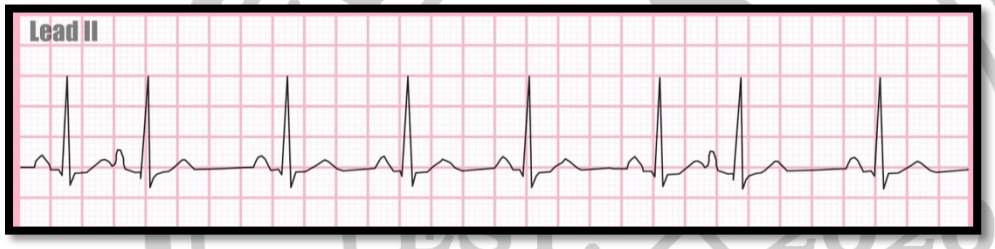


Figure 12.1. Premature Atrial contraction

Two PACs variants are,

a. Blocked PACs

PACs occurring prior to A-V nodal repolarization. The non- sinus wave will appear after QRS complex and is termed non-conducted.

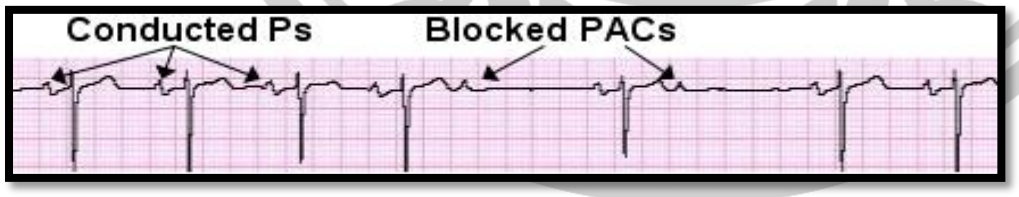


Figure 12.2 Blocked PACs.

b. PAC with aberrant ventricular conduction

PAC with bundle branch block pattern. In simple terms, origin of non-sinus P wave during refectory period of bundle branches. The resultant QRS complex will be wide.

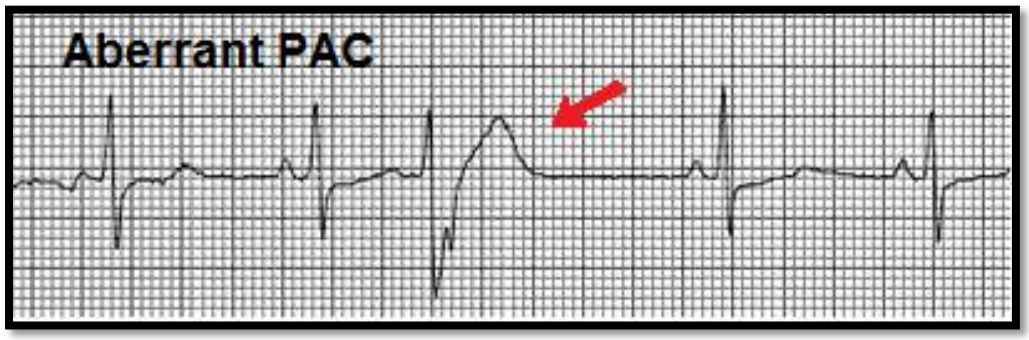


Figure 12.3. PAC with aberrant changes in QRS complex.

◊ Junctional premature beats

Junctional premature complex (JPC) is similar to PACs with a few changes at

a. Inverted P waves due to retrograde atrial conduction
b. Simultaneous atrial and ventricular contraction, P wave either merged or precedes or follows the QRS complex
c. Due to the common findings between JPC and PACs they are often termed as supraventricular premature complexes.

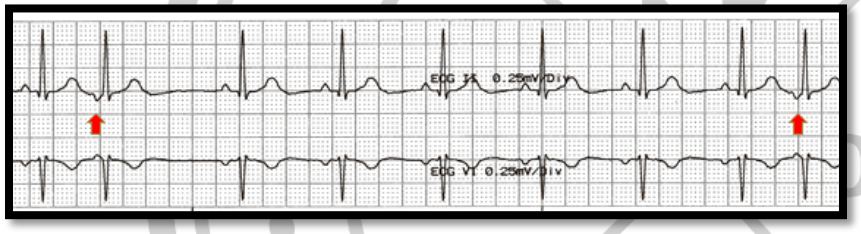


Figure 12.4. JPC with inverted P waves.

◊ Ventricular premature beats

Premature ventricular contraction (PVCs) features include,

a. Appearance of PVC prior to normal QRS beat
b. Bizarre and wide QRS complex
c. P wave buried in QRS complex
d. Compensatory Pause after PVC

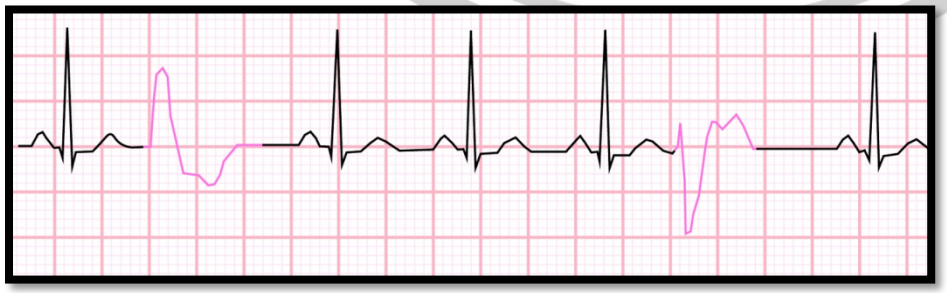


Figure 12.5. PVC with bizarre wide QRS complex.

Based on occurrence it is further subdivided into,

a. Multifocal PVCs, difference in morphology and coupling interval

b. Unifocal PVCs, similar in morphology and coupling interval

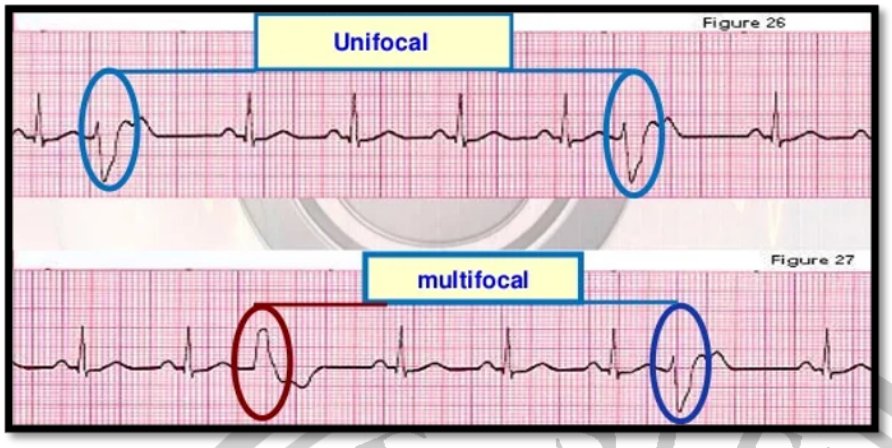


Figure 12.6. Multifocal and Unifocal PVCs.

- PVCs that appear in the late diastolic phase, often precedes the sinus beat and produce a long coupling interval. These are call end diastolic PVCs.

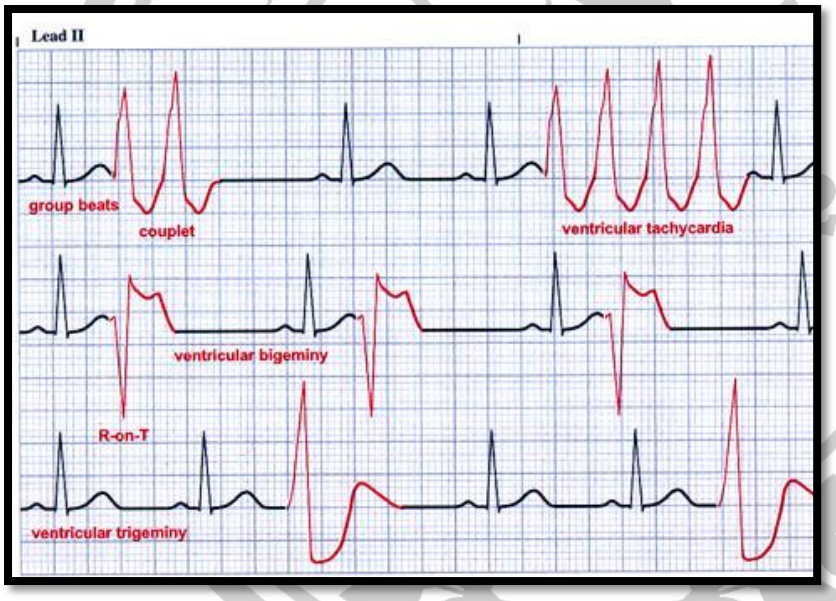


Figure 12.7. Different types of PVCs seen on ECG.

- PVCs with a very small coupling interval, precedes p wave and imposed on T wave is termed as R on T phenomenon.
- PVC that appears between two sinus beats during a slow rhythm with no compensatory pause is called interpolated PVC.
- PVC after every sinus beat constitute of a bigeminal rhythm whereas, after every two sinus beats is trigeminy and after every three would be quadrigeminy.
- Two consecutive PVCs make a couplet and three make a triplet.

PVCs are very different from PACs on the basis that the former have a wider QRS complex while, the latter have narrow QRS complex. Moreover, features unique to PACs include,

a. P wave precedes
b. Triphasic contour of QRS complex

c. Compensatory pauses are incomplete

Clinical causes of premature beats

1. Premature atrial complexes (PACs)

→ Physical factors (emotional stress and exercise)
→ Dietary (excessive intake of caffeine)
→ Smoking
→ Medication (salbutamol and theophylline)
→ Metabolic disorders (hypoxia and acidosis)

2. Junctional premature complexes (JPCs)

→ Cardiac disorders (rhematic heart disease, pericarditis and myocardial infarction)
→ Medication poisoning (digitalis toxicity)
→ Hormonal imbalances (thyrotoxicosis)
→ Cardiac surgery

Treatment for supraventricular premature complexes

◊ Avoidance of triggers (stress, excessive exercise, caffeine, adrenergic drugs and smoking)
◊ Treat underlying medical condition (cardiac disease and hormonal imbalances)
◊ Diltiazem and propranolol (first line)
◊ Amiodarone

3. Premature ventricular complexes (PVCs)

→ Physical factors (emotional stress and exercise)
→ Dietary (excessive intake of caffeine)
→ Smoking
→ Medication (sympathomimetics and theophylline)
→ Hormonal imbalances (thyrotoxicosis)
→ Coronary artery disease (ischemia, infarction and reperfusion)
→ Heart failure (cardiomyopathy, myocarditis, ventricular aneurysm, hypertension)
→ Medication poisoning (digitalis toxicity and treatment)
→ Valvular disorders, Mitral valve prolapse (MVP)
→ Cardiac surgery

Features of dangerous or malignant PVCs include

– ≥6 beats per minute
– Associated ventricular tachycardia
– Couplet or bigeminy rhythm
– R on T phenomenon
– ≥0.14 seconds of wide, bizarre or multifocal
– Underlying cardiac disease with ventricular dysfunction.

Management of PVCs

- ◊ Avoidance of triggers (stress, excessive exercise, caffeine, adrenergic drugs and smoking)
- ◊ Treat underlying medical condition (congestive heart failure or myocardial infarction)
- ◊ Anti-arrhythmic drugs → symptomatic + ventricular ectopy + failure of above two methods + organic heart disease (left ventricular hypertrophy, arrhythmogenic right ventricular dysplasia and cardiomyopathies).
- ◊ Beta blockers (Propranolol and metoprolol) → ideal choice for PVCs secondary to anxiety, vigorous exercise, MVP and thyrotoxicosis.
- ◊ Lidocaine and amiodarone → after cardiac surgery or myocardial infarction.
- ◊ Digoxin toxicity → withdraw digoxin + give phenytoin sodium.

Pauses In Rhythm

A silent gap in two successive sinus beats representing a period of electrical inactivity on the electrocardiogram is termed as rhythm pause. Causes include,

1. Sinoatrial nodal block
2. Atrioventricular nodal block
3. Premature beats

Sinoatrial nodal block

Delay in the propagation of signals from SA node to the surrounding atrial myocardium can lead to delay in atrial activation called SA nodal block. It is of three grades namely,

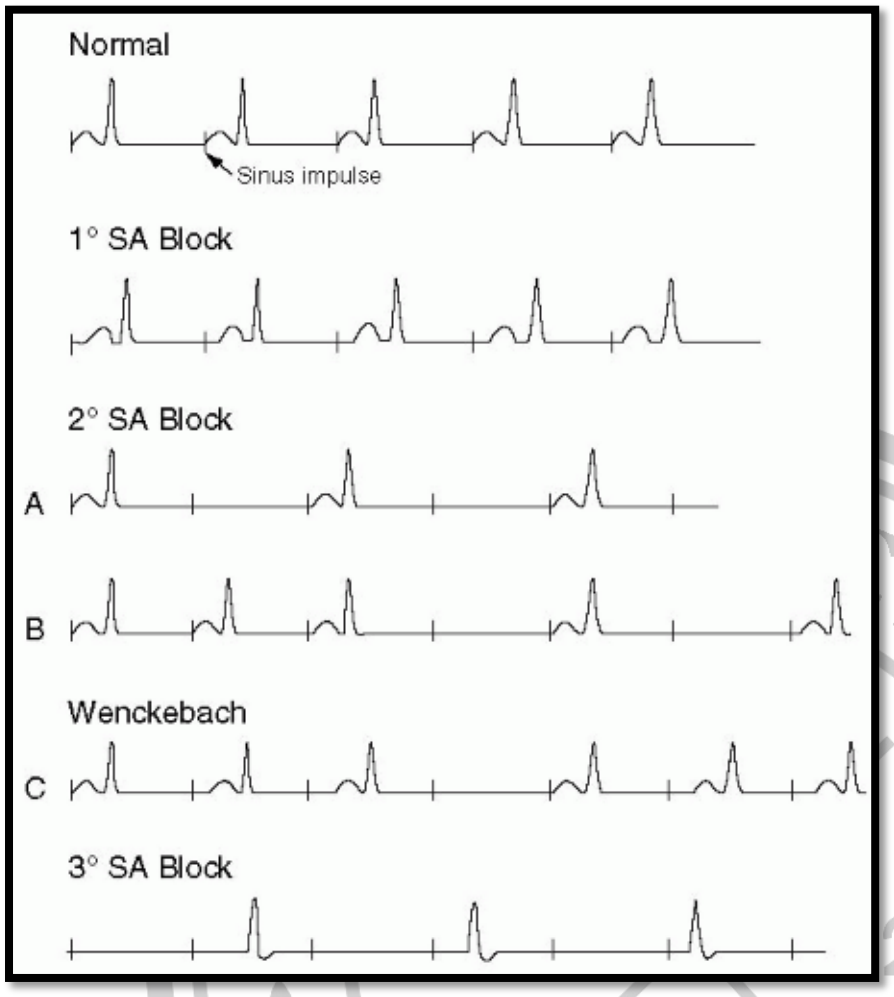


Figure 12.8. Different Degrees of Sinoatrial Block.

a. 1st degree SA block

No visible beat drop but delay in sinoatrial conduction can be seen.

b. 2nd degree SA block

Visible drop of one or more complete beats (P wave, QRS complex and T wave). For instance, if every 2nd beat is dropped in a way that one beat appears instead of two in that time frame, it is called 2:1 SA block. Similarly, if every 3rd beat dropped such that two beats appear in place of normal three beats time, it is called 3:2 SA block.

c. 3rd degree SA block

Complete blockade of transmission of impulses from SA node causes atrial standstill or sinus arrest. ECG is categorized by pause in rhythm with takeover of rhythm by ectopic pacemaker.

Atrioventricular block

Interruptions in signal transmission from atria to ventricle through AV node is called atrioventricular (AV) block.

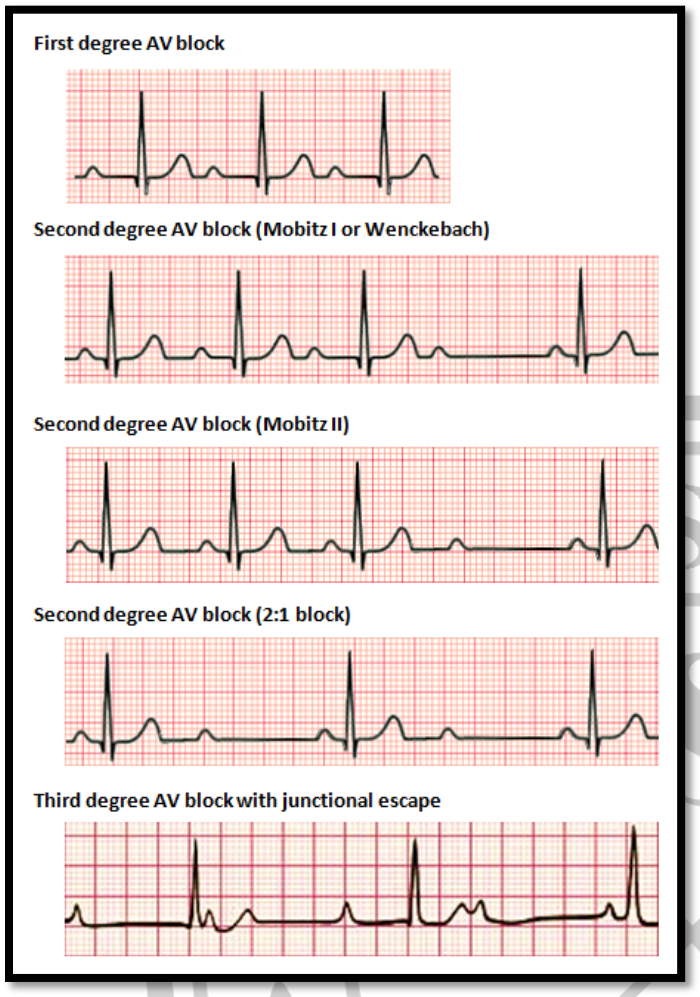


Figure 12.9. Different Degrees of Atrioventricular Block.

Types are,

a. 1st degree AV block

- Prolong P-R interval
- No beat drops

b. 2nd degree AV block

Further subdivided into,

- Mobitz type 1or Wenckebach phenomenon →successive P-R interval prolongation with beat drop.
- Mobitz type 2 → prolong P-R interval with beat drop.

c. 3rd degree AV block

Complete heart block or 3rd degree block is categorized by no transmission of signals from AV node. Therefore, there is complete AV dissociation. In simple terms, atria and ventricles are beating independent

of each other by natural and ectopic pacemakers respectively. The ECG shows no relation of P wave to QRS complexes.

Premature beat

Pause after a premature either supraventricular or ventricular is called a compensatory block. In supraventricular premature contractions, there is an incomplete compensatory pause. So, the delay in interval is less than twice of two successive sinus R-R intervals.

4. On the contrary, ventricular premature contractions have a complete compensatory pause with delay being twice more of two successive sinus R-R intervals.
5. Clinical causes of rhythm pauses:

1. Pause after premature beat → sick sinus syndrome (prolong SA nodal recovery time)

2. Sinoatrial block
→ Sinus node dysfunction
→ Peri myocarditis
→ Cardiac ischemia and infarction
→ Physiological in athletes
→ Medications like digitalis, diltiazem, beta-blockers, procainamide.

3. Atrioventricular block
→ Infections (diphtheria)
→ Rheumatic heart disease
→ Medications (Digitalis, Diltiazem, Betablockers)
→ Cardiac ischemia and infarction
→ Right coronary artery spasm.

Abnormal Rhythm

Normal rhythm in ECG is every P wave followed by QRS complex at regular intervals. Based on speed, regularity and QRS complex morphology we can divide abnormal rhythm into 3 main categories as follows,

1. Normal Rhythm
2. Fast abnormal Rhythm
3. Slow abnormal Rhythm

<u>Normal rhythm</u>

P waves and QRS complexes are in succession with a normally morphology. Since the rhythm is normal the pacemaker lies at SA node, therefore heart beats at 60-100 beats per minute. The two possible abnormalities that can arise with a normal rhythm is classified into two based on QRS complex. That is,

- Normal rhythm with narrow QRS complex

→ Atrial tachycardia

Atria beats at a much faster rate of 150-200 beats per minute whereas, ventricles due to physiological AV block at a rate of 75-100 beats minute. As a result, the ECG shows a normal rhythm, narrow QRS complexes and decrease in P-P interval.

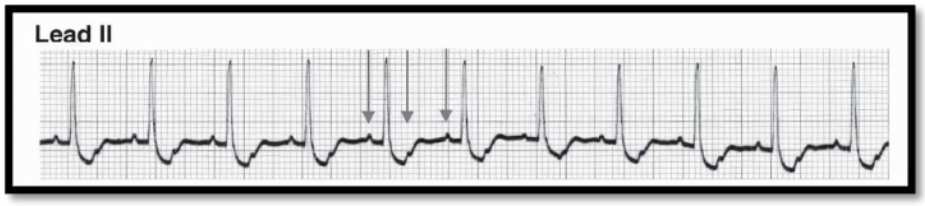


Figure 12.10. Atrial tachycardia with 2:1 AV block.

→ Atrial flutter

In atrial flutter, atria beat very fast at a rate of 220-350 beats per minute. All impulses from atria cannot pass into ventricle due to the physiological AV block. Suppose if every fourth wave is accompanied by QRS complex than it would be 4:1 AV block with ventricular rate at 60-80 beats per minute. Atrial flutter produces saw tooth waves called F waves, the F-F interval indicates atrial rate at 220-35 beats per minute while, in atrial tachycardia P-P interval has a rate of 150-200 per minute.

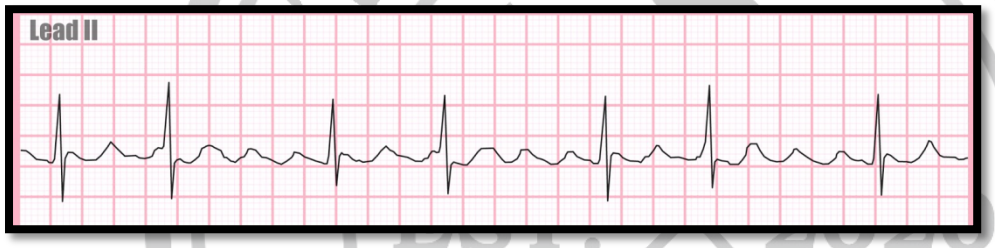


Figure 12.11. Atrial flutter with 4:1 AV block.

→ Junctional tachycardia

In Junctional tachycardia, the rhythm is controlled by an ectopic pacemaker located at the A-V nodal junction. It has a normal rate, rhythm but narrow QRS complexes. Differences from sinus tachycardia includes, inverted P waves that may precede, follow or even buried in QRS complex due to nearly similar activation timings of atria and ventricles.

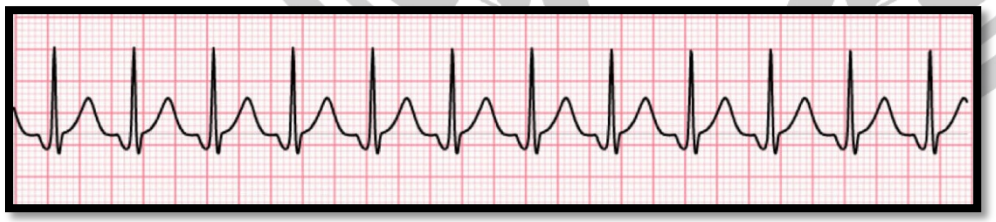


Figure 12.12. Junctional tachycardia with inverted P waves.

Causes are,

◊ Hormonal imbalances (thyrotoxicosis)
◊ Coronary artery disease (inferior wall infarction)
◊ Rhematic carditis
◊ Medication poisoning (digitalis toxicity)
◊ Cardiac surgery

- Normal rhythm with wide QRS complex

→ Idioventricular rhythm

Rhythm originating from ectopic pacemaker at ventricular myocardium is called accelerated idioventricular rhythm (AIVR). ECG findings are,

a. Normal Rythm
b. Heart rate of 60-100 beats per minute
c. Wide and bizarre QRS complex
d. AV dissociation

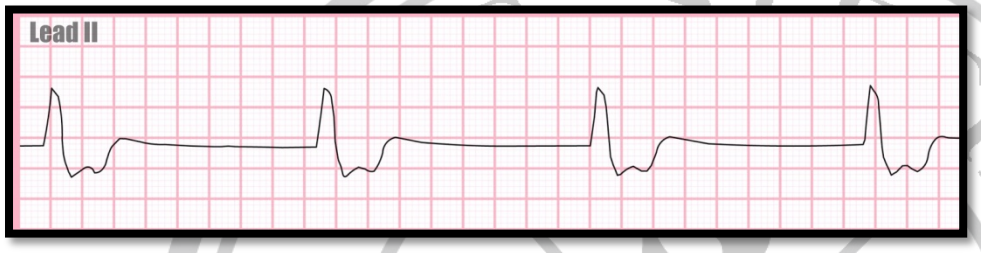


Figure 12.13. Idioventricular rhythm with wide QRS complexes and AV dissociation.

AIVR can be differentiated from ventricular tachycardia by ventricular rate. The rate in AIVR is normal that is 60-100 beats/minute while, 150-200 beats/minute in ventricular tachycardia. AIVR is a harmless type of arrythmia that is benign and transient but should be differentiated from bundle blocks. Treatment is often not required unless hemodynamic instability is present. Medical options include, atropine to speed up SA rate, overcome ventricular rhythm and terminate the AV dissociation.

Causes of clinical significance are;

1. Sinus rhythm with broad QRS complex is seen in;
   - Conduction disorders such as complete bundle branch block, Intraventricular conduction defect and ventricular pre-excitation syndrome
2. AIVR
   - Most common in acute myocardial infarction
   - Often following thrombolytic or coronary reperfusion therapy
   - Infrequent Caseses include, medication toxicity (Digitalis toxicity), Rheumatic carditis and Cardiac surgery.

- <u>Slow Regular rhythm with normal QRS complex:</u>

A rhythm is considered slow if the rate is under 60 beats per minute which can be due to,

1. Slow discharge of impulses
   - SA node
   - Junctional block

2. Conduction blocks
   - SA nodal block
   - AV nodal block
   - Block of ectopic beats

Clinically significant examples of slow rhythm are as follows,

a. Sinus bradycardia

Heart beat that is under 60 beats per minute with each P wave followed by QRS complex at regular intervals is called sinus bradycardia.

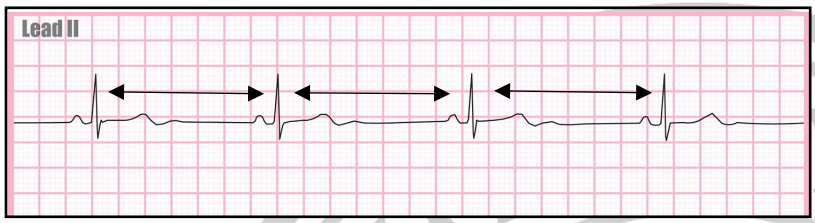


Figure 12.14. Sinus bradycardia.

For instance, figure 12.14 shows number of big squares between R-R interval to be 7, so

$$\text{Heart rate} = \frac{300}{\text{No. of big squares between R-R interval}} = \frac{300}{7} = 42 \text{ beats/minute}$$

Causes of sinus bradycardia:

- Athletes
- Increasing age
- Deep sleep
- Low temperatures such as hypothermia
- Raised intracranial pressure and Intraocular pressure (glaucoma)
- Hormonal deficiency like hypopituitarism and hypothyroidism
- Obstructive jaundice
- Medications like beta blockers and amiodarone
- Conduction disorders such as sick sinus syndrome
- Vasovagal shock

b. Junctional escape rhythm

Under certain circumstances, ectopic pacemaker at the AV junction takes control over the rhythm when the SA node is unable to generate normal impulses. ECG findings are,

- ◊ Heart rate of 40-60 beats per minute
- ◊ Normal rhythm
- ◊ Inverted P waves
- ◊ P wave precedes, follow or buried in the QRS complex
- ◊ Narrow QRS complexes

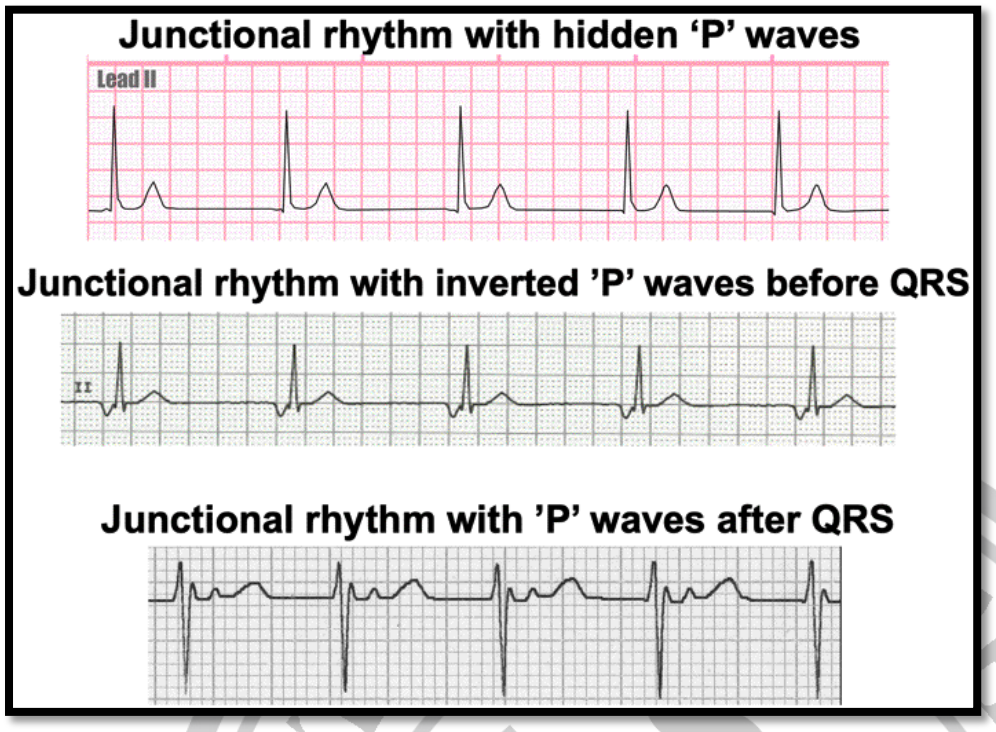


Figure 12.15. Junctional Rhythm with difference in P wave position.

Causes of junctional rhythm:

- Athletes
- Dysfunctional SA node (sinus arrest or sinus pause)
- Medications like amiodarone, betablockers, digoxin and diltiazem.

c. Sinus rhythm with SA nodal block:

In 2nd degree SA nodal block (2:1), after two successive beats there is a single drop of an entire beat (P-QRS-T wave) as neither atrial or ventricular conduction takes place (Figure 12.8.). Interestingly, in SA nodal block atropine causes an immediate doubling of heart rate while, in sinus bradycardia it leads to a slow rise in rate.

d. Sinus rhythm with AV nodal block:

In 2nd degree AV nodal block (2:1), intermittent dropping of QRS complexes cause a pause in rhythm. Such an alternative pattern will present with sinus bradycardia and normal P waves. The difference between SA nodal to AV nodal block lies in the presence of P waves, that is present in AV nodal but missing as an entire beat in SA nodal beat drop (Figure 12.9.).

Management of such 2:1 symptomatic block includes momentarily increasing the ventricular rate by injectable adrenaline or atropine. Temporary pacing is preferred in situations like myocardial infarction, carditis and drug poisoning. Lastly, permanent pacing is for long standing conditions such as sick sinus syndrome.

e. Blocked ectopic beats

PACs have a premature P wave accompanied by a normal QRS followed by a compensatory pause until next beat. If the PACs alternate with normal then it can create a slow regular rhythm of 2:1 SA block or sinus

bradycardia. The difference between such a waveform to sinus bradycardia would be bizarre p waves altering the morphology of T waves. Example of such beats are seen in older age group and digitalis toxicity.

- Slow Regular rhythm with wide QRS complex:

There are two possibilities to having a slow rhythm (heart rate under 60 beats per minute) with a broad QRS complex,

1. The rhythm is controlled by the foci in ventricles and not through the natural conduction phenomena.
2. The rhythm is governed by the foci being supraventricular but in the presence of a co-existing abnormality forming wide QRS complexes.

Examples

- Complete AV block with an idioventricular rhythm.
- Ventricular rhythm from an external pacemaker.
- Complete SA block with escape rhythm at ventricles.

Now, let's discuss each example in detail as follows

a. Complete AV block

Complete block of signal transmission from atrium to ventricles cause a complete or 3rd degree AV block that constitute of AV dissociation (figure 12.9). In simple terms, the rhythm of atria is governed by SA node (beats at 60-80 beats per minute) while, ventricles beat independently by an ectopic focus at a rate of 20-40 beats per minute forming wide QRS complexes (idioventricular rhythm). Important conditions that can lead to complete AV block are,

- Congenital such as septal defects
- Coronary artery disease as anteroseptal myocardial infarction
- Valvular disease as aortic valve calcinosis or stenosis
- Fibrocalcerous degeneration as in Lev's disease
- Atrial septal defect repair surgery

Furthermore, clinically significance of AV block depends on factors such as,

- ◊ Reversibility
- ◊ Site of subsidiary pacemaker either bundle of His or ventricle
- ◊ Presence of symptoms

Among the many symptoms of complete AV block, dizziness or feeling faint is the most common. It is due to momentarily ventricular asystole that leads to a sudden drop in cardiac output. Such symptoms or syncopal episodes are termed as Strokes Adams attacks. Other possible contributors to such attacks include, carotid sinus hypersensitivity, complete SA block and subclavian steal syndrome. Although it is a transient interruption in signal transmission but can prove to be even life threatening. Therefore, indefinite cardiac pacing is required in,

- ✓ Heart rate under 40 beats per minute with broad QRS complexes
- ✓ Recurrent episodes of Strokes Adam attacks
- ✓ Acute myocardial infarction

b. External ventricular pacemaker rhythm

In case of complete heart block, external pacing is done at the right ventricle with an impulse of 60 beats per minute. These impulses can be continuous or intermittent depending upon the mode of pacing required and the intrinsic signals available. ECG findings of a paced rhythm include, asynchronous broad QRS complexes with a spike-like deflection before each beat that is termed as the pacemaker artefact.

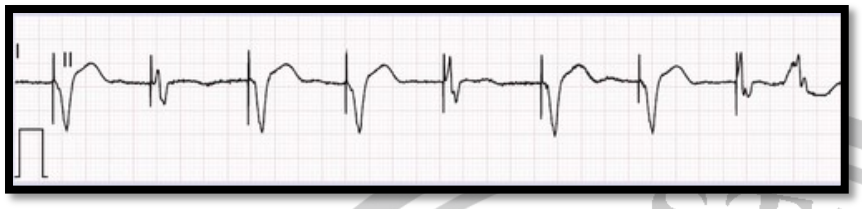


Figure 12.16. External pacemaker rhythm with broad QRS and pacemaker artefacts.

c. Complete SA block

Complete SA block also known as sinus arrest or sinus standstill, there is no ventricular depolarization from the natural pacemaker as a result junctional pacemaker governs the ventricular rhythm at a rate of 20-40 beats per minute by a phenomenon called junctional escape (Figure 12.8.). Complete SA nodal block can lead to serious conditions like Strokes Adam attack as mentioned earlier. Treatment options for symptomatic individuals involve administering medications to enhance the sympathetic activity such as increasing heart rate. For instance,

Give 0.6mg of Intravenous Atropine in stat dose and repeat every 3-5 mins to attain response. A total dose of 0.03-0.04 mg/kg body weight can be given or injection epinephrine 1mg (1:10,000 strength).

Causes of complete SA block are,

- Medications like, digitalis, diltiazem and propranolol
- Vagal stimulation by carotid sinus massage
- Sinus node dysfunction.

- Slow Irregular rhythm with Narrow QRS complex:

Heart rate under 60 beats per minute with an irregular rhythm can present with narrow QRS complexes due to following reasons,

1. Wandering pacemaker
2. Beat to beat variation
3. Conductional blocks

Sinus arrythmia is characterized by variation in heart rate to either too fast or too slow due to problem with the SA node. It is further divided into two types-based relation to the respiratory cycle namely, respiratory and non-respiratory sinus arrythmia. Moreover, P-P or R-R interval doesn't remain constant, reflecting the variations in the conductive pathways in regards to heart rate.

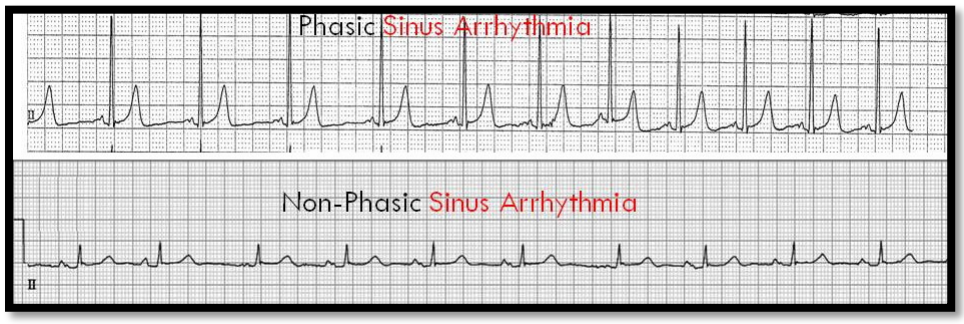


Figure 12.17. Sinus arrythmia-Phasic and non-phasic.

In respiratory sinus arrythmia, the initial 4 beats occur at a constant rate while, the next 4 if occurring in inspiration will be slower or in case of expiration, faster. It is termed as phasic sinus arrythmia as seen in figure 12.17. Causes include normal phenomena in children and young adults that require no treatment.

On the other hand, non-respiratory sinus arrythmia, there is no relation of heart rate to the respiratory cycle. Hence, termed non-phasic sinus arrythmia. It can be seen in elderly as a case of SA nodal dysfunction and will require management with pacing.

Let's discuss the origin of such waves in details.

a. Wandering pacemaker rhythm

When the impulses originate from various points surrounding the SA node; such a resultant rhythm is called a wandering pacemaker. In simple terms, the foci for impulses shift from one to another causing beat to beat variation and alteration in P wave morphology. Possible foci for origin are SA node (upright P waves), atrium (lower atrium, inverted P waves) and AV junction (inverted P waves). A variation in P-R interval can also be appreciated due to alteration in AV conduction time.

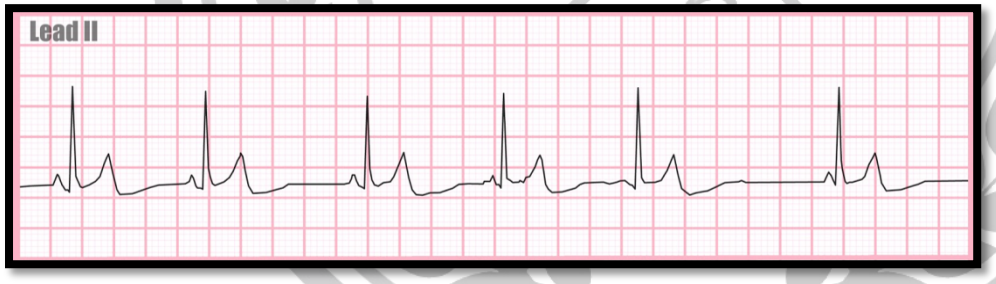


Figure 12.18. Wandering atrial pacemaker rhythm.

It can be observed in young asymptomatic adults to occasionally with digitalis toxicity and rheumatic fever. Management in asymptomatic cases is avoided while, sinus bradycardia can be treated with atropine or epinephrine alongside underlying cause in symptomatic individuals.

b. Sinus rhythm with conduction blocks

Sinus rhythm with dropped beats due to SA or AV nodal blocks can cause either missing p wave or an entire P-QRS-T complex, leading to a compensatory pause. The dropped pattern can be 2:1 or if alternative 3:2 can produce a slow irregular rhythm (Figure 12.8-12.9.). Amongst the common causes, sick sinus syndrome and acute inferior wall MI due to SA and AV block can present with slow irregular rhythm, respectively.

c. Slow Atrial fibrillation

Atrial fibrillation is a type of irregularly irregular rhythm that is known for generating fast impulses recorded on ECG as fibrillatory waves instead of P waves and ventricles beating at a rate of 100-150 beats per minute. Since the atria beat at a rate of 350 beats per minute, only a few impulses can make its way through the AV node leading to some degree of AV block. After a while, AV block becomes greater and the ventricles slow down producing a slow atrial fibrillation.

- Fast regular rhythm with Narrow QRS complex:

A regular rhythm with a heart rate above 100 beats per minute is called fast rhythm. Moreover, rapid discharge of conduction signals through myocardium results in formation of narrow QRS complexes. Examples of some important fast sinus arrythmias are as follows,

a. Sinus Tachycardia:

Heart rate that exceeds 100 beats per minute with each P wave and QRS complex following one other in succession is called sinus tachycardia. The R-R interval due to fast rate is reduced to 10mm in a rate of 150 beats per minute while, 15mm in 100 beats per minute.

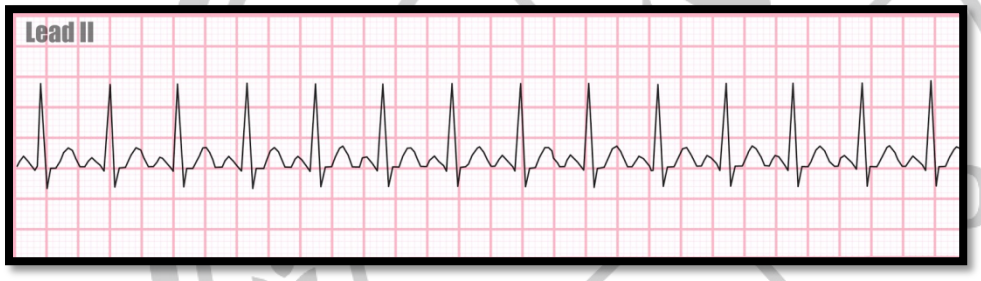


Figure 12.19. Sinus tachycardia

Causes of sinus tachycardia include:

- Physiological → Exercise and anxiety
- Hyperthermia
- Fluid loss
- Low oxygen saturation → Hypoxemia and anemia
- Hypotension and heart failure
- Hypermetabolic states → Thyrotoxicosis and pregnancy
- Sympathomimetics → Caffeine, nicotine and alcohol
- Medications → Atropine and beta-agonists
- Hemorrhage and hypoglycemia
- Infections → Pericarditis and myocarditis
- Massive pulmonary embolism

Treatment involves providing patient with symptomatic relief and addressing the underlying factors or diseases.

b. Atrial Tachycardia

A type of regular fast rhythm produced by either

- Ectopic foci in atrium (10% cases)

- Closed re-entrant circuit (90% cases

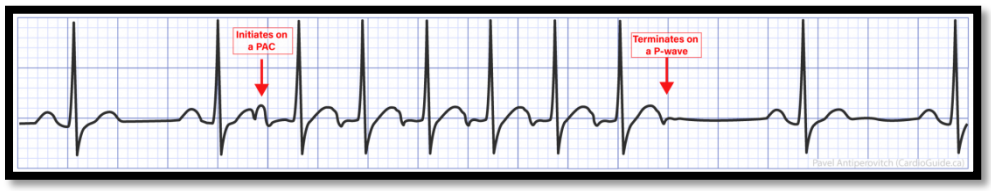


Figure 12.20. Atrial tachycardia

Features suggestive of atrial tachycardia include,

- Regular clock rhythm
- P-QRS complex normal
- QRS with width under 0.14 seconds
- Hemodynamic stability
- Termination with vagal maneuvers

ECG findings	Atrial tachycardia	Sinus tachycardia	Ectopic tachycardia	Re-entrant tachycardia
1. Heart rate	150-220 per minutes	100-150 per minutes	120-150 per minutes	≥150 per minutes
2. Onset	Clock like	Variation with breathing	Gradual	Sudden
3. P wave	Inverted or ectopic	Normal	Ectopic	Inverted
4. AV block	-	-	Possible	Never
5. Vagal maneuvers	Termination	Slowing	Slowing	Termination
6. Past history	-	-	No	Yes
7. Organic heart	-	-	Maybe	Absent

Table. 12.1. ECG findings of different types of Tachycardias.

Causes of Atrial tachycardia are,

- Infective conditions as acute myocarditis and rheumatic fever.
- Medications like adrenergic drugs and digitalis toxicity
- Hormonal conditions such as thyrotoxicosis
- Myocardial infarction
- Cardiac surgery.

Treatment options for hemodynamically stable individual include,

- Vagal maneuvers, carotid sinus massage, face in ice-cold water ice immersion and Valsalva.
- If vagal maneuvers fail then administer

Intravenous 6 mg adenosine over 3 seconds, repeat with 12 mg intravenously over 2 minutes and then again by 12 mg for another 2 minutes until the sinus rhythm is restored.

OR

Intravenous diltiazem 15-20 mg over 2 minutes, repeat with 20-25 mg intravenously over 2 minutes and then after 15mins if needed.

OR

Intravenous amiodarone 150mg over 10 minutes, repeat with 150mg intravenously over 10 minutes and again if needed.

Oral medications like diltiazem, metoprolol and amiodarone are preferred only after restoration of rhythm.

In hemodynamically unstable, cardioversion with 80-100 Joules is first line.

c. Atrial Flutter

A type of fast regular rhythm with narrow QRS due to rapid discharge of ectopic foci or re-entrant circuit at atrium. The atria beat at a very fast pace of 220-350 beats per minute such that the P wave gets replaced by saw-tooth waves called flutter (F) waves.

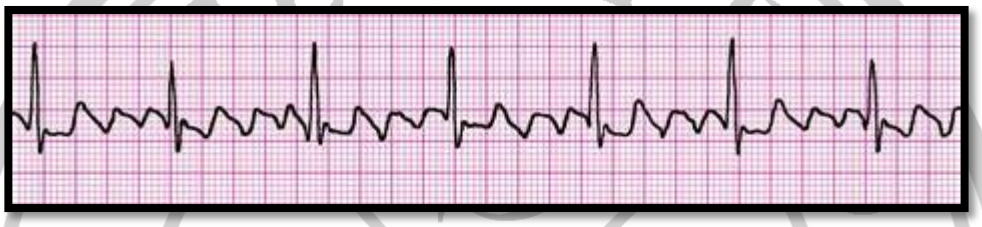


Figure 12.21. Atrial flutter with characteristic saw-tooth waves

Moreover, the ventricles are unable to catch up to atrial rate due to physiological AV block. Atrial flutter resembles that of atrial tachycardia but have few significant features that can set the two apart as seen in table 12.2.

ECG findings	Atrial flutter	Atrial tachycardia
Atrial rate	220-350 beats per minute	150-220 beats per minute
Ventricular rate	2:1 or 4:1 AV block	1:1 AV block
P waves	Saw tooth waves	Ectopic or inverted
Vagal maneuvers	Increase	Decrease or terminate

Table. 12.2. ECG differences in Atrial flutter to Atrial tachycardia.

Causes of Atrial flutter are,

- Infective conditions as pericarditis, myocarditis and rheumatic fever.
- Medications like adrenergic drugs and digitalis toxicity
- Acute respiratory failure and pulmonary hypertension
- Hormonal conditions such as thyrotoxicosis
- Myocardial infarction and ischemic heart disease
- Cardiac surgery.

Management constitutes of controlling heart rate with diltiazem or metoprolol similar to atrial tachycardia in hemodynamically stable individuals. If associated angina, hypotension or cardiac insufficiency (ejection fraction less than 45%), cardioversion with 10-50 joules is highly effective. However, in hemodynamically unstable cases, electrical cardioversion is preferred.

d. Ventricular tachycardia

Ventricular tachycardia (VT) is a type of fast regular rhythm that is due to a latent ventricular pacemaker or a closed re-entrant circuit in the ventricular tissue discharging impulses at a high rate. It can further be divided into,

- Sustained VT (lasting for more than 30 minutes)

Potential causative factors are as follows,

- ✓ Ischemic causes like myocardial infarction, aneurysm
- ✓ Cardiac diseases like cardiomyopathies, myocarditis and RV dysplasia.
- ✓ Heart failure, ischemic and hypertensive
- ✓ Valvular pathology includes rheumatic heart disease and mitral valve prolapse.

- Non-sustained VT (lasting for less than 30 minutes)

Causes,

- ✓ Pharmacological (theophylline, sympathomimetics, digitalis toxicity and quininde over dose)
- ✓ Acute myocardial ischemia and reperfusion
- ✓ Electrolyte and metabolic imbalances (hypoxia, hypokalemia and metabolic acidosis)
- ✓ Cardiac surgery

Features suggestive if VT are,

- ◊ Mild irregularity in rhythm
- ◊ Hemodynamic compromise
- ◊ Underlying cardiac disease
- ◊ Wide bizarre QRS complex (width ≥0.15 seconds)
- ◊ Lack of P-QRS association or relation
- ◊ Non-responsive to vagal maneuvers as carotid sinus massage.

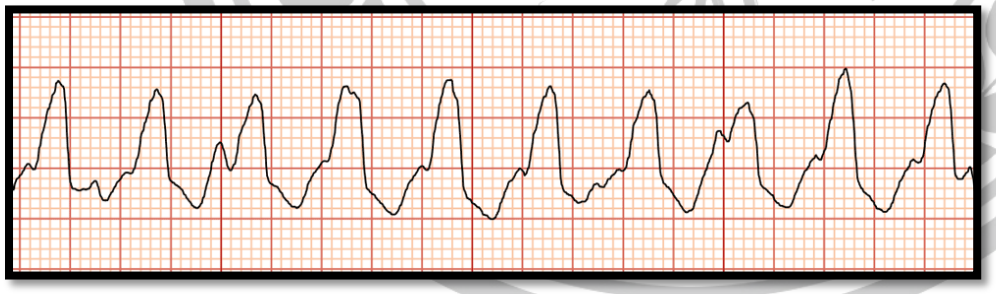


Figure 12.22. Ventricular tachycardia with wide bizarre QRS complexes.

It is important to differentiate VT from supraventricular tachycardia as their treatment slightly varies (Table.12.3.).

Findings	Ventricular tachycardia	Supraventricular tachycardia
Rhythm Regularity	Mildly irregular	Regularity (clock-like)
P-QRS complex	AV dissociation	-
Underlying heart disease	Present in majority	Absent

Response to vagal maneuvers	No response	Slowing or termination
Precordial leads	rS V1-V6	rS to Rs
QRS-morphology	Bizarre	Triphasic
QRS-width	>0.14 seconds	0.12-0.14 seconds
Capture/fusion beats	Maybe	Never
Cannon waves	Maybe	Never
QRS axis	North-west axis	Normal axis
Hemodynamic status	Unstable	Stable
Variations in Heart sounds	Variable S1	Constant S1

Table. 12.2. Differences in Ventricular tachycardia from supraventricular tachycardia based on ECG, clinical tests and examination.

Treatment options for VT depend on the type, hemodynamic status and underlying causing factor.

- Hemodynamically stable

In case of non-sustained VT, treatment of underlying cause is ideal. However, in sustained VT betablockers like metoprolol is highly effective to control the abnormal rate as for an abnormal rhythm guideline for treatment are as follows.

Injection amiodarone 150 mg given intravenously over 10 minutes, repat with another 150 mg every 10 minutes as per required

OR

Injection lidocaine 0.5-0.75 mg/kg body weight given intravenously, repeat with same dose every 5-10 minutes to a maximum of 3mg.kg body weight to revert back to sinus rhythm.

After achieving normal rhythm, maintenance dose is started as,

Injection amiodarone 360 mg given intravenously over 6 hours, maintained at 540 mg over next 18 hours.

OR

Injection Lidocaine 2-4 mg per minute given intravenously at 30-60 µg/kg/minute

- Hemodynamically unstable

A non-synchronous shock of 50-100 joules needs to be administered but if peripheral pulses are absent then 200-360 joules is preferred.

- Fast irregular rhythm:

Heart rate that exceeds 100 beats per minute with rapid asynchronous discharges from natural or ectopic pacemaker produce a rapid irregular rhythm. Clinically significant types are as follows,

a. Atrial tachycardia with AV nodal block
b. Multifocal Atrial Tachycardia
c. Atrial Flutter with Varying AV Block
d. Atrial Fibrillation

Atrial fibrillation (Afib) is a type of fast arrythmia having an irregularly irregular rhythm. Around 400-500 fibrillatory (f) waves are produced from multiple foci in the atria, out of which only 100-160 make its way

through AV node resulting in AV dissociation and a slower ventricular rate. The diagnostic finding of Afib is these f waves that replace the normal P waves. Difference between Afib to atrial flutter can be seen in table 12.3.

ECG findings	Atrial fibrillation	Atrial flutter
Atrial rate	≥350 beats per minute	220-350 beats per minute
Ventricular rate	Variable	Regular
Atrial activity (P waves)	fibrillatory waves	Flutter waves
Ventricular activity (R-R Interval)	Variable	constant
Base line		

Table. 12.4. ECG differences in Atrial fibrillation to Atrial flutter.

◊ Types and causes of Atrial Fibrillation

1. Persistent Atrial Fibrillation (> 7 days)

✓ Atrial septal defect
✓ Rheumatic heart disease
✓ Cardiomyopathies
✓ Constrictive pericarditis
✓ Hypertension related heart diseases
✓ Coronary artery disease
✓ Cardiac surgery

2. Paroxysmal atrial fibrillation (< 7 days)

✓ Lone atrial fibrillation
✓ Pulmonary embolism
✓ Conduction disorders (WPW syndrome and sick sinus syndrome)
✓ Acute alcohol intoxication
✓ Thyrotoxicosis

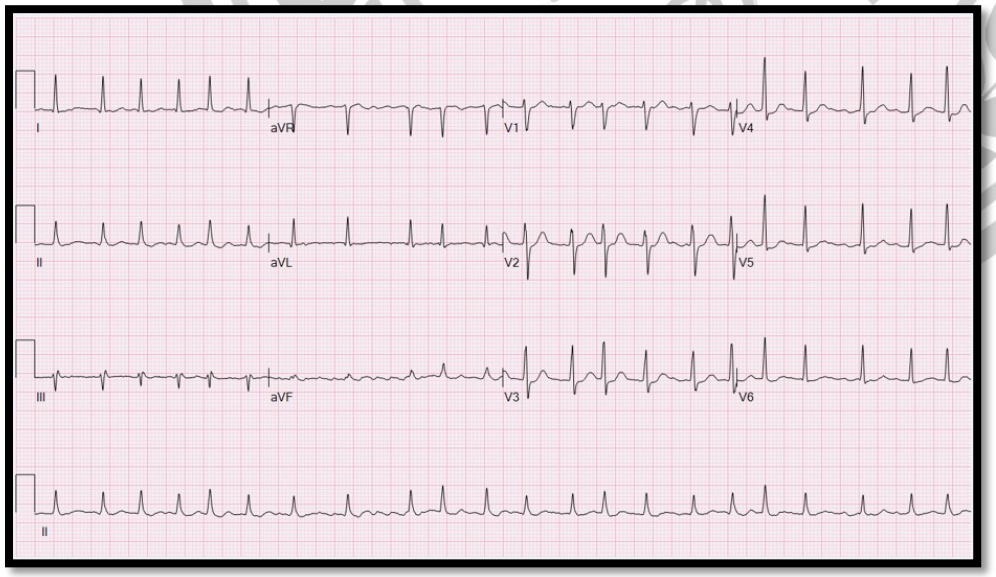


Figure 12.23. Atrial fibrillation with characteristic fibrillatory waves

◊ Clinical signs

✓ Fast irregular pulse

- ✓ Low blood pressure
- ✓ Raised JVP
- ✓ Variable intensity of 1st heart sound.

◊ Management

If patient is hemodynamically stable, the aim is to control rate and rhythm of the arrythmia. To control rate Betablocker (esmolol), calcium channel blockers (diltiazem) or digoxin is considered while, for rhythm anti-arrhythmic drugs(amiodarone) are preferred.

- ➢ Injection diltiazem 15-20 mg over 2 minutes, then repeat after 15 minutes.
- ➢ Injection esmolol 500 mcg/kg over 1-2 minutes, then repeat after 10-15 minutes.
- ➢ Injection amiodarone 150 mg over 10 minutes then repeat after 10 minutes.
- ➢ Long standing anticoagulants to prevent embolic events.

If patient is not hemodynamically stable, electrical cardioversion with 100-200 Joules energy is the treatment to revert to normal sinus rhythm. Radiofrequency ablation is considered the last resort after failure of above measures provided that,

- ➢ Age is below 70 years
- ➢ Left atrium is smaller than 5cm
- ➢ No heart failure
- ➢ No obesity.

e. Ventricular fibrillation

Ventricular fibrillation (Vfib) is among the most serious types of arrythmia that requires urgent attention and management. It is characterized by irregularly irregular rhythm with chaotic ventricular depolarization at a rate higher than 350 beats per minute.

Classical features of Vfib are,

- ➢ P-QRS-T complex is unidentifiable
- ➢ Waveforms are wide, erratic and bizarre
- ➢ Hemodynamic instability
- ➢ Pulselessness

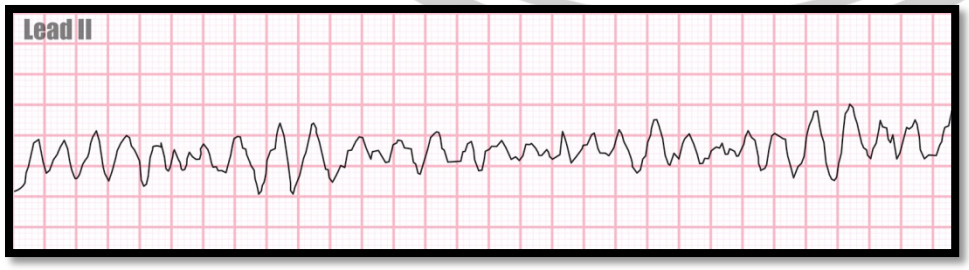


Figure 12.24. Ventricular fibrillation with bizarre waveforms.

Causes include,

- Myocardial infarction
- Cardiomyopathy
- Medication overdose (quinidine and digoxin)
- Metabolic (acidosis and hypoxia)
- Accidental electrical shocks or hypothermic conditions

This type of arrythmia has not only poor prognosis but highest mortality therefore, urgent cardioversion is required. Electrical defibrillation of 200-360 Joules of shock is ideal for Vfib. Any delay in shock treatment can increase the amount of shock voltage required to revert to normal rhythm. In case of failure after one attempt, repeat defibrillation with intravenous bicarbonate administration of 1mEq/kg body weight to correct underlying metabolic acidosis. If second attempt fails, give intravenous 300mg amiodarone or 1-1.5mg/kg body weight of lidocaine.

To avoid recurrence of Vfib, pharmacological options include phenytoin, sotalol, flecainide and propafenone. Nowadays, AICD (Automatic Implantable Cardioverter Defibrillator) devices are available to detect and revert Vfib automatically in ambulatory patients.

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