BASIC ELECTROGRAPHY

UNIT-1

Learning objective:

- Fundamental principles of electrocardiography: Cardiac electrical field Generation during activation, Cardiac wave fronts
- Cardiac electrical field Generation during ventricular recovery

Fundamental principles of electrocardiography: Cardiac electrical field Generalisation*:*

What is an ECG?

An ECG is simply a representation of the electrical activity of the heart muscle as it changes with time, usually printed on paper for easier analysis. Like other muscles, cardiac muscle contracts in response to electrical *depolarisation* of the muscle cells. It is the sum of this electrical activity, when amplified and recorded for just a few seconds that we know as an ECG.

Basic Electrophysiology of the Heart (see Figure 1)

The normal cardiac cycle begins with spontaneous depolarisation of the sinus node, an area of specialised tissue situated in the high right atrium (RA). A wave of electrical depolarisation then spreads through the RA and across the inter-atrial septum into the left atrium (LA).

The atria are separated from the ventricles by an electrically inert fibrous ring, so that in the normal heart the only route of transmission of electrical depolarisation from atria to ventricles is through the atrioventricular (AV) node. The AV node delays the electrical signal for a short time, and then the wave of depolarisation spreads down the interventricular septum (IVS), via the bundle of His and the right and left bundle branches, into the right (RV) and left (LV) ventricles. Hence with normal conduction the two ventricles contract simultaneously, which is important in maximising cardiac efficiency.

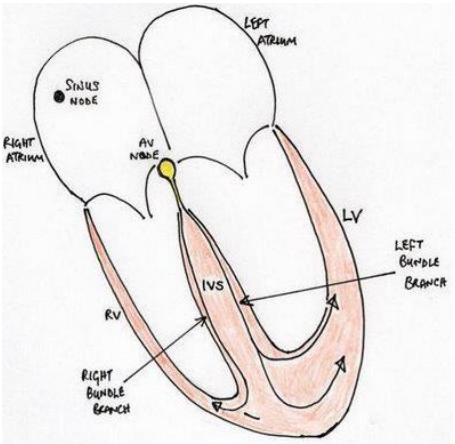


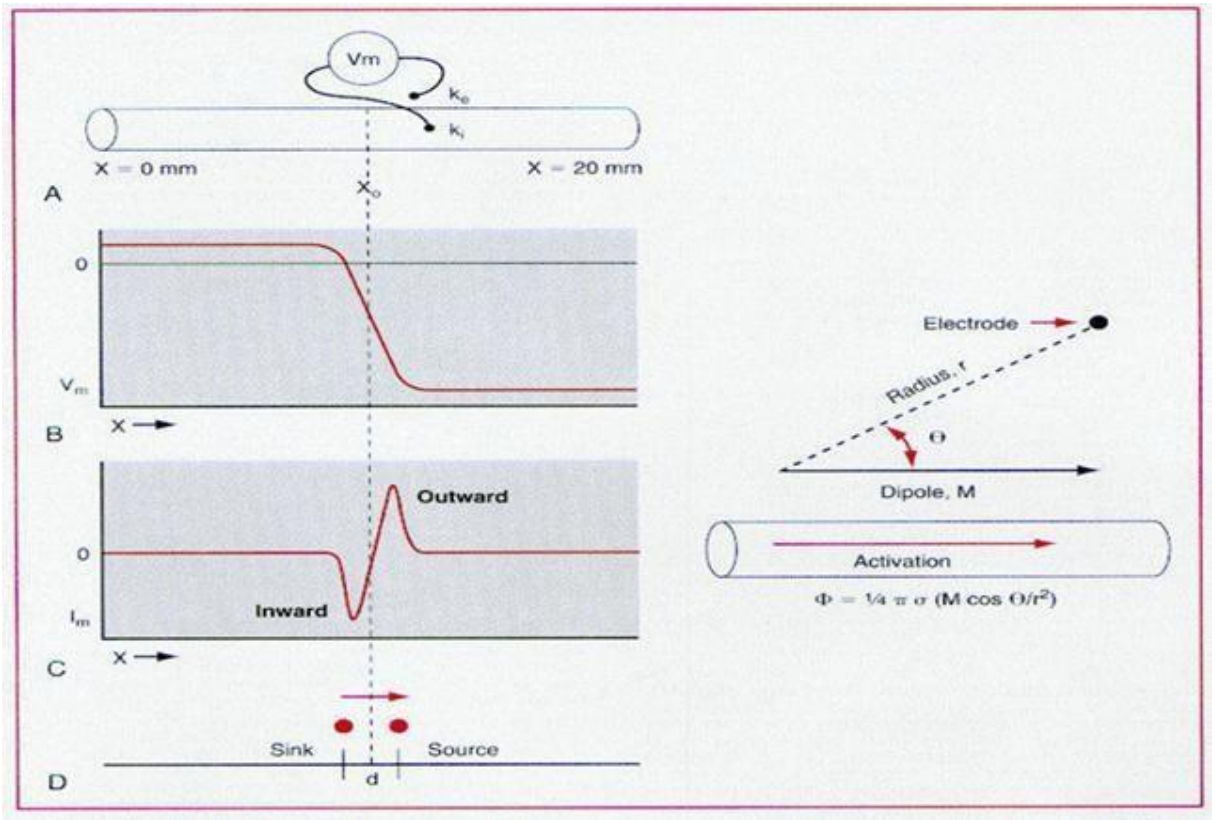
Figure 1. Basic electrophysiology of the heart

After complete depolarisation of the heart, the myocardium must then *repolarise*, before it can be ready to depolarise again for the next cardiac cycle.

Cardiac electrical field Generation during activation:

Transmembrane ionic currents are ultimately responsible for the cardiac potentials that are recorded as an ECG. Current may be analysed as though carried by positively charged or negatively charged ions.

A positive charge moving in one direction is equivalent to negative current of equal strength moving in the opposite direction. The process of generating the cardiac electric field during activation can be illustrated by considering the events in single cardiac fibre that is activated by a stimulus applied to its left most margin. Transmembrane potential (Vm) are recorded as the difference between intracellular and extracellular potentials(Ki and ke)



Left panel B plots Vm along the length of the fibre at the instant during the propagation(t_0) at which activation has reached the point designated as X_0.

As each site is activated the polarity of transmembrane potential is converted from negative to positive. Thus sites to the left of the point X_0 , which have already undergone excitation, have positive transmembrane potentials (i.e. inside of the cell is positive) whereas those to the right of X_0 (which remains in resting state) have negative transmembrane potentials. Near the site undergoing activation (site X_0) the potentials reverse the polarity over a short

distance. Left panel C displays the direction and magnitude of transmembrane currents (Im) along the fiber at the instant (t_0) at which excitation has reached X_0

Current flows is inwardly directed in fiber regions that have undergone activation and outwardly directed in neighbouring zones still at rest(that is to right of X_0).

Sites of outward current flow are current sources and those with inward current flow are current sinks, current flow is most intense in each direction near the site of activation X_0.

Because of border between inwardly and outwardly directed currents are relatively sharp, we can visualize these currents as though they were limited to the sites of maximal current flow, as depicted in panel D and separated by distance d that is usually 1.0mm or less.

As activation proceeds along the fiber, the source sink pair moves to the right at a speed of propagation for the particular type of fiber.

Two point sources of equal strength but of opposite polarity located near each other, left panel D may be represented as a current diapole(arrow in panel D). thus, activation of a fiber can be modelled as a current dipole that moves in the direction of propagation of activation. Such a dipole is fully characterized by three parameters- strength of dipole movement, location and orientation.

A current dipole produces a characteristic potential field with positive potential projected ahead and negative potential projected behind the moving dipole. The actual potential recorded at any site within the field is directly proportional to the dipole moment, inversely proportional to the square of distance from the dipole to the recording site and directly proportional to the cosine of angle between the axis of dipole and a line drawn from the dipole to the recording site.

This example from on cardiac fiber can be generalized to the more realistic case in which multiple adjacent fibers are activated in synchrony to produce an activation front. Activation of each fiber creates a dipole oriented in the direction of activation. The net effect of all the dipoles in this wave front is a single dipole equal to the sum of the effects of all the simultaneously active component dipoles. Thus, an activation front propagating through heart can be represented by a single dipole that projects positive potentials ahead of it and negative potential behind it.

This relationship between activation, direction, orientation of the current dipole and polarity of potential is critical one in electrocardiography. It describes a fundamental relationship between the polarity of potentials sensed by an electrode and the direction of a movement of an activation front. This dipole model while useful in describing cardiac fields and understanding clinical electrocardiography, has significant theoretical limitations. These limits derive primarily from the inability of single dipole to accurately represent more than one wave front that is propagating through the heart at any instant.

Cardiac electrical field Generation during ventricular recovery:

The cardiac electrical field during recovery (phases 1 through 3 of the action potential) is generated by forces analogous to those described during activation.As a cell undergoes recovery, its intracellular potential becomes progressively more negative.

Hence, for two adjacent cells the intracellular potential of the cell whose recovery has progressed further is more negative than that of an adjacent less recovered cell. Intracellular current than flows from the less towards the more recovered cell. An equivalent dipole can then be constructed recovery. Just as for activation, its orientation however points from less to more recovered cell. Thus, the recovery dipole is oriented away from the activation front. That is in the direction opposite that of the activation dipole. The moment or strength of the recovery dipole also differs from that of the activation dipole. The strength of the activation dipole is proportional to the rate of change in transmembrane potential. Rates of change in potential during recovery phases of action potential are considerably slower than during activation. So, dipole moment at any one instant during recovery is less than that during activation.

UNIT-2

Learning objectives:

- Electrocardiographic lead systems: Standard limb leads, Precordial leads and the Wisdom central termina, Augmented limb leads
- The hexaxial reference frame and electrical axis
- Recording adult and pediatric ECGs

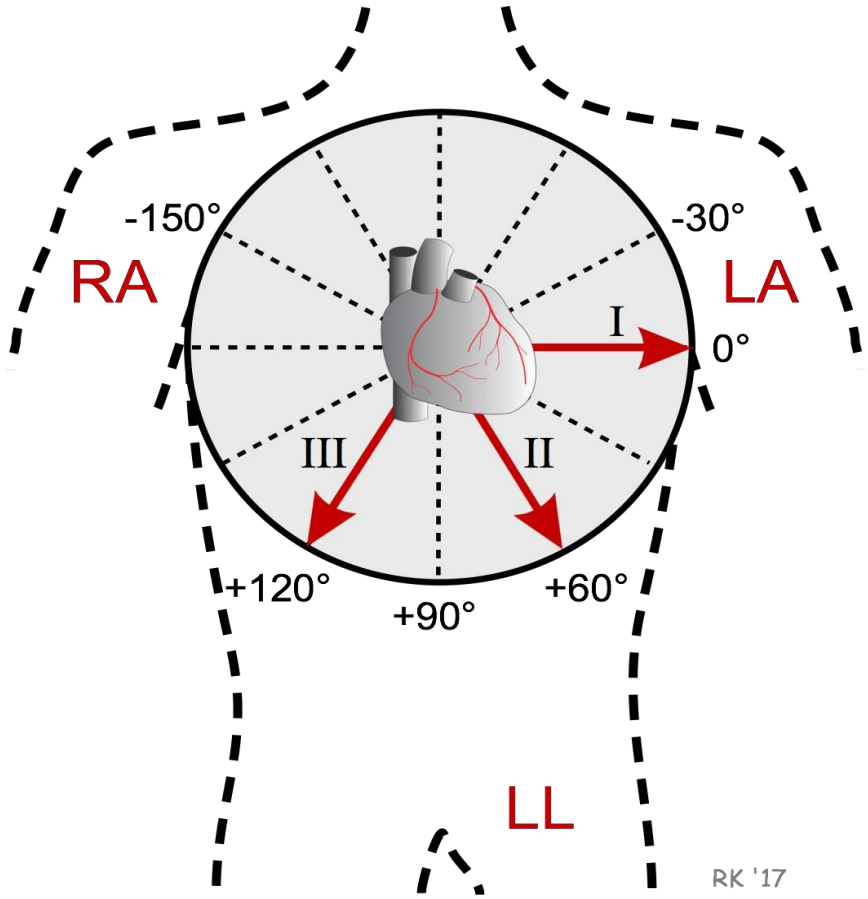
Electrocardiogram Standard Limb Leads (Bipolar):

Bipolar recordings utilize standard limb lead configurations depicted in the figure. By convention, lead I has the positive electrode on the left arm, and the negative electrode on the right arm, and therefore measures the potential difference between the two arms. In this and the other two limb leads, an electrode on the right leg serves as a reference electrode for recording purposes.

In the lead II configuration, the positive electrode is on the left leg and the negative electrode is on the right arm. Lead III has the positive electrode on the left leg and the negative electrode on the left arm. These three bipolar limbs lead roughly form an equilateral triangle (with the heart at the center) that is called Einthoven's triangle in honor of Willem Einthoven who developed the electrocardiogram in the early 1900s. Whether the limb leads are attached to the end of the limb (wrists and ankles) or at the origin of the limb (shoulder or upper thigh) makes little difference in the recording because the limb can simply be viewed as a long wire conductor originating from a point on the trunk of the body.

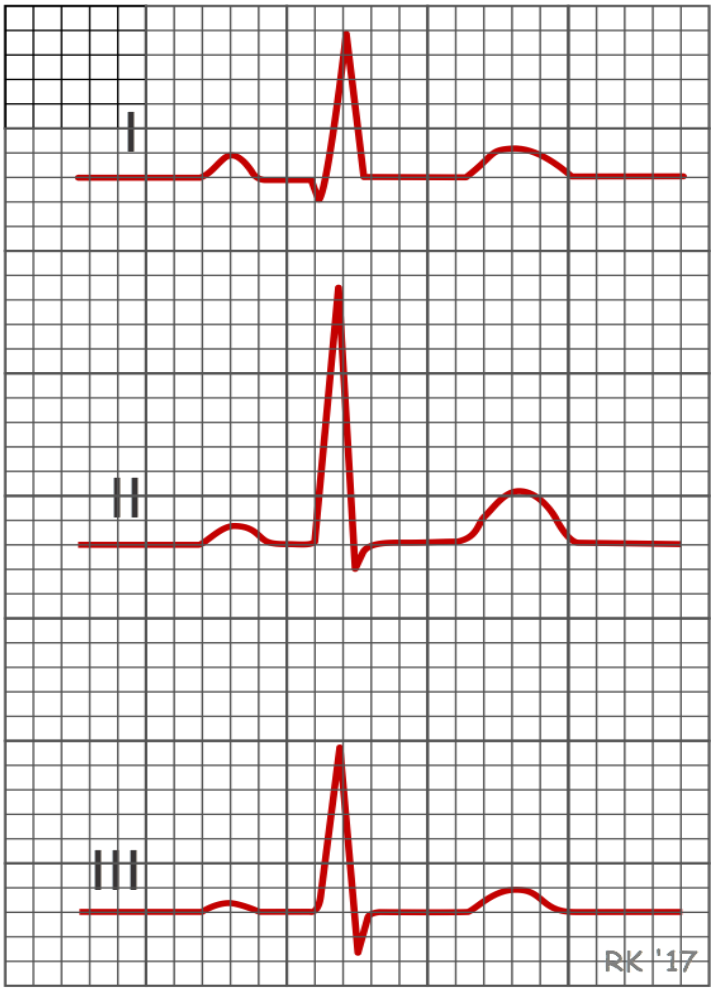
Based upon universally accepted *ECG rules*, a wave a depolarization heading toward the left arm gives a positive deflection in lead I because the positive electrode is on the left arm. Maximal positive ECG deflection occurs in lead I when a wave of depolarization travels parallel to the axis between the right and left arms. If a wave of depolarization heads away from the left arm, the deflection is negative. Also by these rules, a wave of repolarization moving away from the left arm is recorded as a positive deflection. Similar statements can be made for leads II and III in which the positive electrode is located on the left leg. For example, a wave of depolarization traveling toward the left leg produces a positive deflection in both leads II and III because the positive electrode for both leads is on the left leg. A maximal positive deflection is recorded in lead II when the depolarization

wave travels parallel to the axis between the right arm and left leg. Similarly, a maximal positive deflection is obtained in lead III when the depolarization wave travels parallel to the axis between the left arm and left leg.



If the three limbs of Einthoven's triangle (assumed to be equilateral) are broken apart, collapsed, and superimposed over the heart, then the positive electrode for lead I is said to be at zero degrees relative to the heart (along the horizontal axis between LL and RA (see figure). Similarly, the positive electrode for lead II (RA-LL axis) will be +60° relative to the heart, and the positive electrode for lead III (LA-LL axis) will be +120° relative to the heart as shown to the right. This new construction of the electrical axis is called the **axial reference system**. With this system, a wave of depolarization traveling at +60° produces the greatest positive deflection in lead II. A wave of depolarization oriented +90° relative to the heart produces equally positive deflections in both lead II and III. In this latter example, lead I shows no net deflection because the wave of depolarization is heading perpendicular to the 0°, or lead I, axis.

For a heart with a normal ECG and a mean electrical axis of +60°, the standard limb leads will appear as follows:



Precordial leads:

Which are the precordial leads?

The Chest Leads (or Precordial Leads)
Therefore, do not confuse these Precordial V leads with the three V limb leads (aVR, aVL, aVF). The precordial (chest leads) leads each consist of a positive electrode strategically placed on the chest of the patient.

For measuring the potentials close to the heart, Wilson introduced the *precordial leads* (chest leads) in 1944 (Wilson et al., 1944). These leads, V_1-V_6 are located over the left chest as described in Figure 3. The points V_1 and V_2 are located at the fourth intercostal space on the right and left side of the sternum; V_4 is located in the fifth intercostal space at the midclavicular line; V_3 is located between the points V_2 and V_4; V_5 is at the same horizontal level as V_4 but on the anterior axillary line; V_6 is at the same horizontal level as V_4 but at the midline. The location of the precordial leads is illustrated in Figure 3

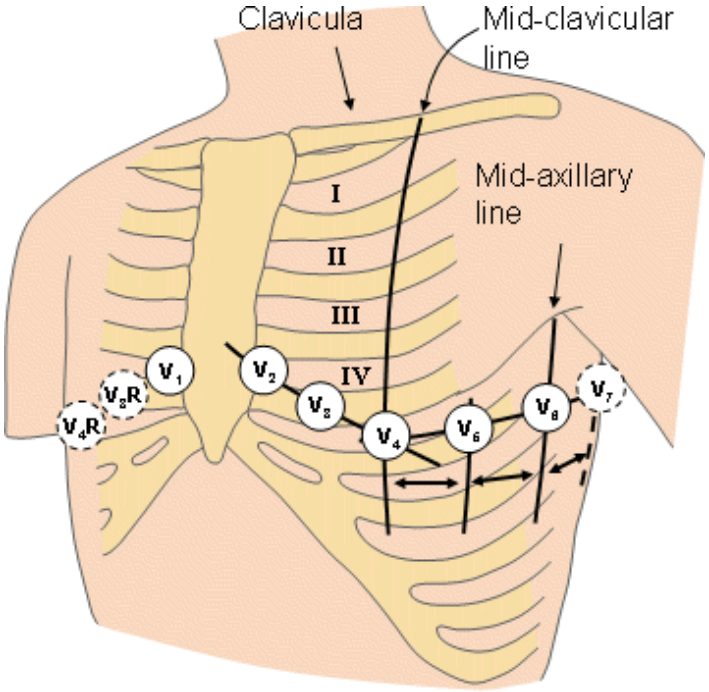


Fig: 3 Precordial Leads

WILSON CENTRAL TERMINAL:

Frank Norman Wilson (1890-1952) investigated how electrocardiographic *unipolar* potentials could be defined. Ideally, those are measured with respect to a remote reference (infinity). But how is one to achieve this in the volume conductor of the size of the human body with electrodes already placed at the extremities? In several articles on the subject, Wilson and colleagues (Wilson, Macleod, and Barker, 1931; Wilson et al., 1934) suggested the use of the *central terminal* as this reference. This was formed by connecting a 5 kΩ resistor from each terminal of the limb leads to a common point called the central terminal. Wilson suggested that unipolar potentials should be measured with respect to this terminal which approximates the potential at infinity.

Actually, the Wilson central terminal is not independent of but, rather, is the average of the limb potentials. This is easily demonstrated by noting that in an ideal voltmeter there is no lead current. Consequently, the total current into the central terminal from the limb leads must add to zero to satisfy the conservation of current (see Figure 1). Accordingly, we require that

$$I_R + I_L + I_F = \frac{\Phi_{CT} - \Phi_R}{5000} + \frac{\Phi_{CT} - \Phi_L}{5000} + \frac{\Phi_{CT} - \Phi_F}{5000}$$

from which it follows that

$$\Phi_{CT} = \frac{\Phi_R + \Phi_L + \Phi_F}{3}$$

Since the central terminal potential is the average of the extremity potentials it can be argued that it is then somewhat independent of any one in particular and therefore a satisfactory reference. In clinical practice good reproducibility of the measurement system is vital. Results appear to be quite consistent in clinical applications.

Wilson advocated 5 kΩ resistances; these are still widely used, though at present the high-input impedance of the ECG amplifiers would allow much higher resistances. A higher resistance increases the CMRR and diminishes the size of the artifact introduced by the electrode/skin resistance.

It is easy to show that in the image space the Wilson central terminal is found at the center of the Einthoven triangle, as shown in Figure below.

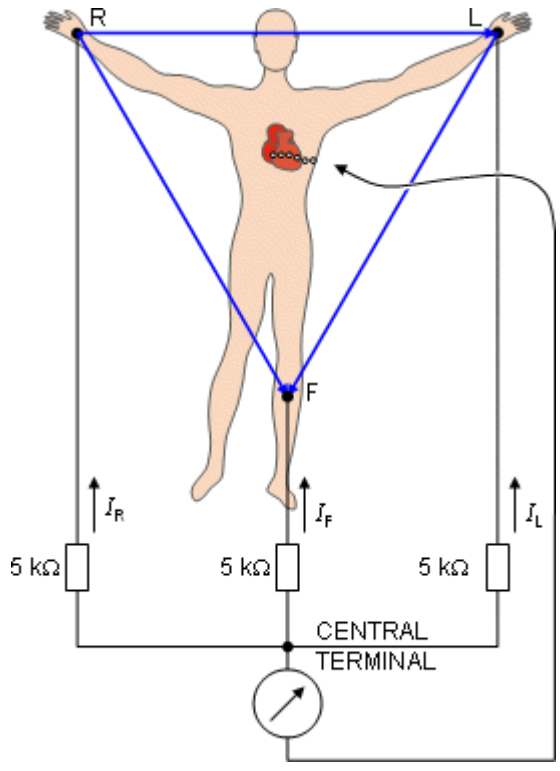


Fig. 1. The Wilson central terminal (CT) is formed by connecting a 5 kΩ resistance to each limb electrode and interconnecting the free wires; the CT is the common point. The Wilson central terminal represents the average of the limb potentials. Because no current flows through a high-impedance voltmeter, Kirchhoff's law requires that $I_R + I_L + I_F = 0$.

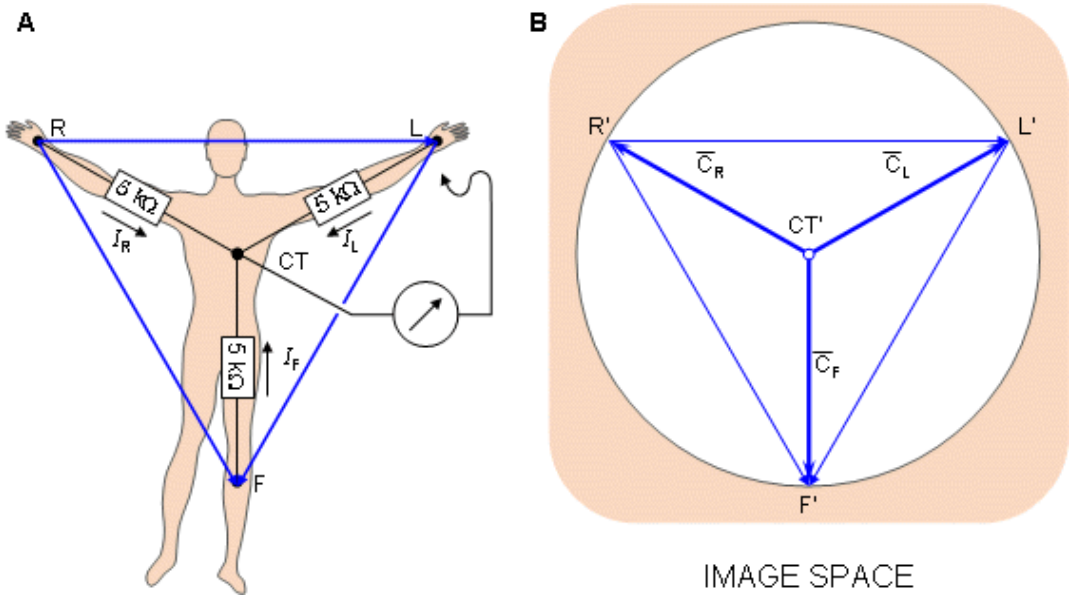
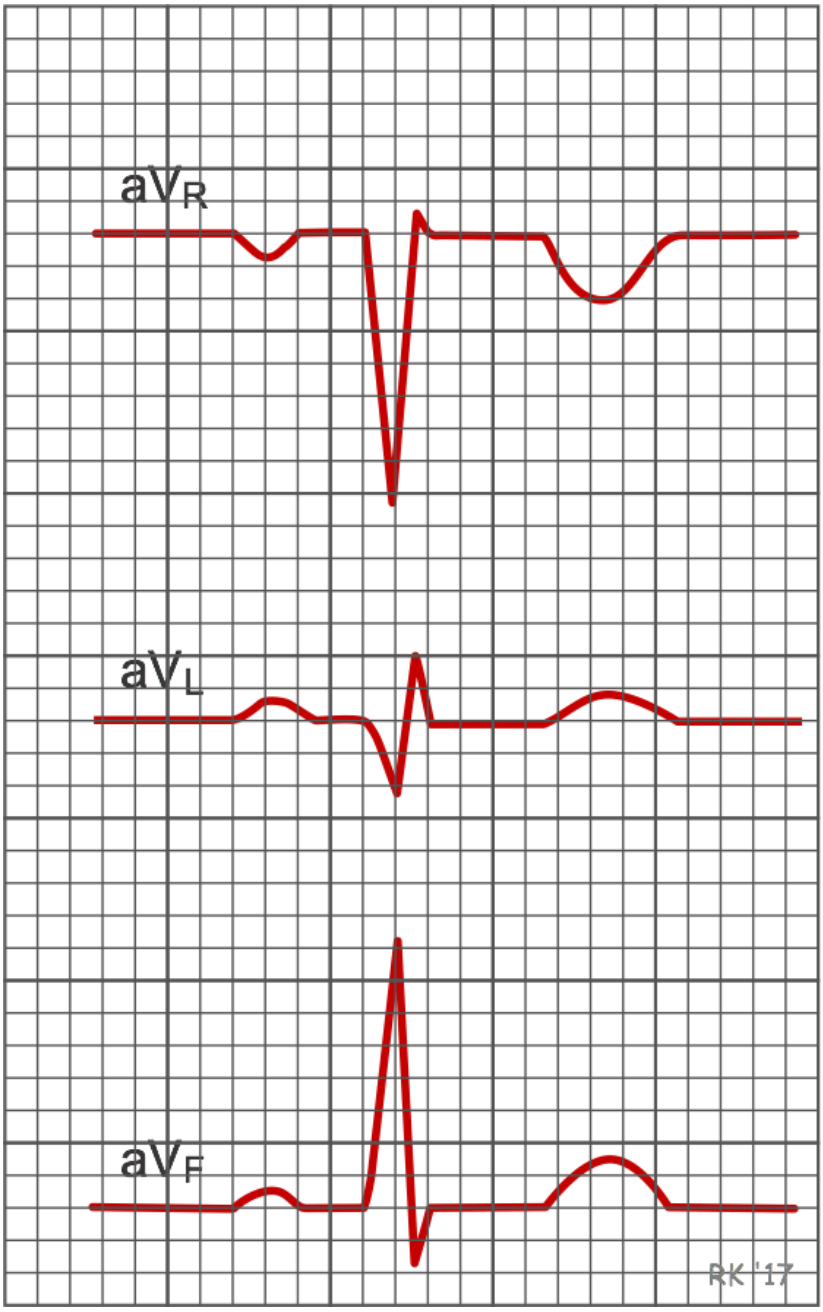


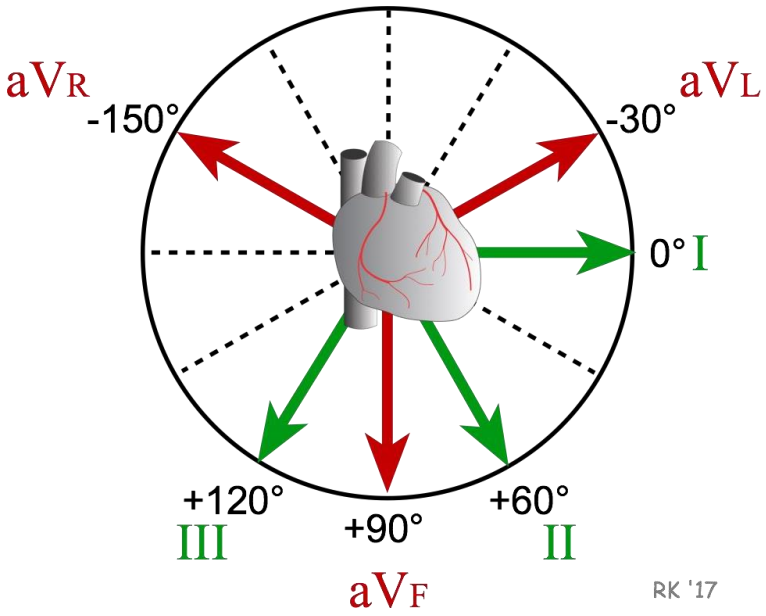
Fig. 2 (A) The circuit of the Wilson central terminal (CT).

Electrocardiogram Augmented Limb Leads (Unipolar)

In addition to the three bipolar limb leads, there are three augmented unipolar limb leads. These are termed unipolar leads because there is a single positive electrode that is referenced against a combination of the other limb electrodes. The positive electrodes for these augmented leads are located on the left arm (aV_L), the right arm (aV_R), and the left leg (aV_F). In practice, these are the same electrodes used for leads I, II and III. (The ECG recorder does the actual switching and rearranging of the electrode designations). The three augmented leads are depicted as shown to the figure using the axial reference system. The aV_L lead is at -30° relative to the lead I axis; aV_R is at -150° and aV_F is at +90°. It is very important to learn which lead is associated with each axis.

For a heart with a normal ECG and mean electrical axis of +60°, the augmented leads will appear as shown below:


aVR
aVL
aVF
RK '17



The three augmented unipolar leads, coupled with the three standard bipolar limb leads, comprise the six limb leads of the ECG as shown in the figure. These six leads record electrical activity along a single plane, termed the **frontal plane** relative to the heart. Using the axial reference system and these six leads, one can define the direction in the frontal plane of an electrical vector at any given instant in time. If a wave of depolarization is spreading from right-to-left along the 0° axis, then lead I will show the greatest positive amplitude. If the direction of the electrical vector for depolarization is directed downwards (+90°), then aV_F will show the greatest positive deflection. If a wave of depolarization is moving from left-to-right at +150°, then aV_L will show the greatest *negative* deflection according to the rules for ECG interpretation.

The hexaxial reference frame :

The hexaxial reference system is a diagram that is used to determine the heart's electrical axis in the frontal plane. Diagram showing how the polarity of the QRS complex in leads I, II, and III can be used to estimate the heart's electrical axis in the frontal plane.

The hexaxial reference system, better known as the Cabrera system, is a convention to present the extremity leads of the 12 lead electrocardiogram, that provides an illustrative logical sequence that helps interpretation of the ECG, especially to determine the heart's electrical axis in the frontal plane. The most practical way of using this is by arranging

extremity leads according to the Cabrera system, reversing polarity of lead aVR and presenting ECG complexes in the order (aVL, I, -aVR, II, aVF, III). Then determine the direction the maximal ECG vector is "pointing", i.e. in which lead there are most positive amplitude - this direction is the electrical axis - see diagram. Example: If lead I has the highest amplitude (higher than aVL or -aVR), the axis is approximately 0°. Conversely, if lead III has the most negative amplitude it means the vector is pointing away from this lead, i.e. towards -60°.

An alternative use is to locate the most isoelectric (or equiphasic) lead (I, II, III, aVR, aVL, or aVF) on a diagnostic quality ECG with proper lead placement. Then find the corresponding spoke on the hexaxial reference system. The perpendicular spoke will point to the heart's electrical axis. To determine which numerical value should be used, observe the polarity of the perpendicular lead on the ECG.

For example, if the most isoelectric (or equiphasic) lead is aVL, the perpendicular lead on the hexaxial reference system is lead II. If lead II is positively deflected on the ECG, the heart's electrical axis in the frontal plane will be approximately +60°.

- Normal axis: -30° to +90°
- Left axis deviation: -30° to -90°
- Right axis deviation: +90° to +180°
- Extreme axis deviation: -90° to -180°

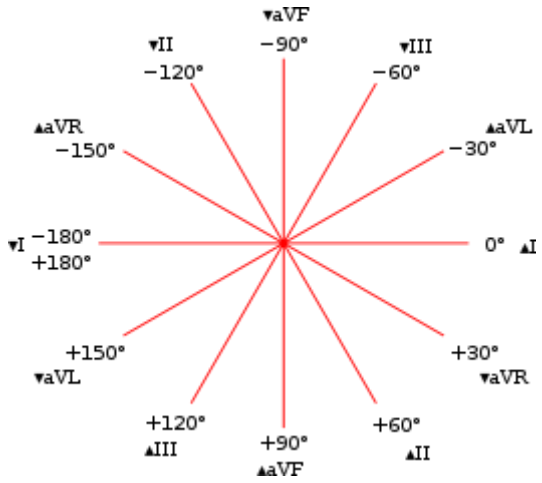


Fig:The hexaxial reference system is a diagram that is used to determine the heart's electrical axis in the frontal plane.

How to perform an Electrocardiogram to a child?

The Electrocardiogram is performed in a child in the same way as adult EKG.

10 electrodes are placed in their usual positions, and should prevent the child from moving during EKG acquisition (this is the hard part).

Limb electrodes can be placed on the torso to reduce movement artifacts.

In newborns and infants must also make V3R and V4R (right-side leads) for a better study of the right ventricle.

When done, it should be reviewed before removing the electrodes, ensuring proper calibration and the absence of artifacts or poorly recorded Leads.

Differences between the Paediatric and Adult Electrocardiogram

The paediatric electrocardiogram has different features, these differences are more pronounced in new-borns, and, as the patient grows, are varying through adolescence.

Electrocardiogram of a Newborn:

In newborns there is a predominance of the right ventricle to the left ventricle due to the fetal circulation

On the EKG trace can be observed:

- Heart rate between 90 and 160 bpm.
- Right-axis deviation (between 70º and 180º).
- Tall R waves in lead V1 and deep S waves in lead V6.
- Shorter waves (P, T) and intervals (PR, QRS).
- Positive T waves in precordial leads at birth, becoming negative in leads V1-V3 after the first week of life.
- Deep Q wave in inferior leads and V5-V6.

Electrocardiogram Changes With Age:

- **Heart axis:** The QRS axis direction is moving toward normal values (between -30º and 90º).
- **Precordial leads:** R wave in lead V1 and S wave in lead V6 are becoming smaller, while S wave in lead V1 and R wave in lead V6 increase their amplitude.
- **Heart rate:** As the child grows, heart rate decreases. In the healthy adult it is between 60 and 100 bpm.
- **Length of waves and intervals:** The length of the waves and intervals of the electrocardiogram increases with age (wider waves and longer intervals).
- **T wave:** T wave is positive in precordial leads in newborns, but after the first week of life becomes negative in leads V1-V3 and persists through adolescence and even, in young adults (juvenile T wave pattern).

Normal Values of Pediatric Electrocardiogram

- The following table shows the normal values for heart rate, heart axis, length of waves and intervals and amplitude of the R waves and S waves in leads V1 and V6 in each pediatric age range. .

Age	0-7 days	8-30 days	1-6 months	6-12 months	1-5 years	5-10 years	10-15 years	adult
HR (bpm)	90 - 160	100 - 175	110 - 180	70 - 160	65 - 140		60 - 130	60 - 100
PR (ms)	80 - 150			50 - 150	80 - 150		90 - 180	100 - 200
Eje (°)	70 - 180	45 - 160	10 - 120	10 - 110	5 - 110			
QRS (ms)	40 - 70				45 - 80		50 - 90	60 - 90
QRS V1 (mV)								
Q	No Q wave							
R	0,5 - 2,5	0,3 - 2,0		0,2 - 2,0	0,2 - 1,8	0,1 - 1,5	0,1 - 1,2	0,1 - 0,6
S	0 - 2,2	0 - 1,6	0 - 1,5	0,1 - 2,0		0,3 - 2,1	0,3 - 2,2	0,3 - 1,3
T	-0,3 - 0,3	-0,6 to -0,1				-0,6 - 2	-0,4 - 0,3	-0,2 - 0,2
QRS V6 (mV)								
Q	0 - 0,2			0 - 0,3	0 - 0,4		0 - 0,3	0 - 0,2
R	0,1 - 1,2	0,1 - 1,7	0,3 - 2,0	0,5 - 2,2	0,6 - 2,2	0,8 - 2,5	0,8 - 2,4	0,5 - 1,8
S	0 - 0,9			0 - 0,7	0 - 0,6	0 - 0,4		0 - 0,2

Non-pathological Changes of Pediatric Electrocardiogram:

In children, is common to find changes in the electrocardiogram which are considered non-pathological disorder.

Sinus arrhythmia: Changes in heart rate (PP intervals) with breathing. Sinus arrhythmia occurs often in children, adolescents and young adults. Is considered a normal sinus rhythm variation.

Wandering atrial pacemaker: Sinus P waves alternating with ectopic P waves. It is observed as P waves with different morphologies in the same lead. The PR interval may also be variable. Wandering pacemaker is usually caused by increased vagal tone, rarely causes symptoms or requires treatment.

Supraventricular extrasystoles: Presence of a narrow premature QRS. It may be preceded by ectopic P wave (atrial origin) or not (node origin). No pathological significance, but may cause symptoms.

RSR' pattern in V1: The incomplete left bundle branch block is also often found in childhood and youth, in patients without heart disease. Although if it is accompanied by heart murmur should be ruled atrial septal defect.

First degree AV block and second degree AV block, type I (Wenckebach): Can be seen in children with increased vagal tone, no pathological significance (see AV blocks).

Early repolarization: Concave ST segment elevation with terminal QRS slurring or notching (J wave). It is an EKG pattern most commonly seen in adolescents and young athletes. No pathological significance, although, it has been found to be associated with a modest increased risk of ventricular arrhythmias.

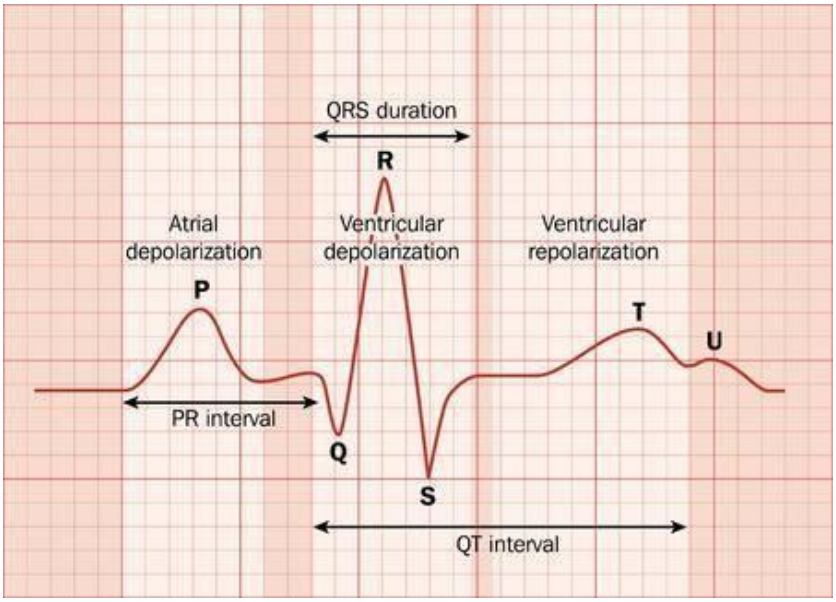
UNIT-3

Learning Objectives:

- The normal electrocardiogram, Atrial activation
- The normal P wave Atrial repolarization
- Atrioventricular node conduction and the PR segment Ventricular activation the QRS complex
- Ventricular recovery and ST-T wave, U wave Normal variants
- Rate and rhythm

The normal electrocardiogram. Atrial activation:

The normal electrocardiogram begins with a P wave, reflecting depolarization of the atria, generally from right to left, and inferiorly. Thus, the atrial activation is represented on the surface electrocardiogram by a P wave that is upright in leads I, II, and III.



Rhythm:

Normal sinus rhythm

The P waves in leads I and II must be upright (positive) if the rhythm is coming from the sinus node.

Conduction:

Normal Sino-atrial (SA), Atrio-ventricular (AV), and Intraventricular (IV) conduction

Both the PR interval and QRS duration should be within the limits specified above.

Waveform Description:

P Wave:

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

- P duration < 0.12 sec
- P amplitude < 2.5 mm
- Frontal plane P wave axis: 0° to +75°
- May see notched P waves in frontal plane

QRS Complex:

The QRS represents the simultaneous activation of the right and left ventricles, although most of the QRS waveform is derived from the larger left ventricular musculature.

- QRS duration ≤ 0.10 sec
- QRS amplitude is quite variable from lead to lead and from person to person. Two determinates of QRS voltages are:
    - Size of the ventricular chambers (i.e., the larger the chamber, the larger the voltage)
    - Proximity of chest electrodes to ventricular chamber (the closer, the larger the voltage)
- Frontal plane leads:
    - The normal QRS axis range (+90° to -30°); this implies that the QRS be mostly positive (upright) in leads II and I.
    - Normal q-waves reflect normal septal activation (beginning on the LV septum); they are narrow (<0.04s duration) and small (<25% the amplitude of the R wave). They are often seen in leads I and aVL when the QRS axis is to the left of +60°, and in leads II, III, aVF when the QRS axis is to the right of +60°. Septal q waves should not be confused with the pathologic Q waves of myocardial infarction.
- Precordial leads:
    - Small r-waves begin in V1 or V2 and progress in size to V5. The R-V6 is usually smaller than R-V5.
    - In reverse, the s-waves begin in V6 or V5 and progress in size to V2. S-V1 is usually smaller than S-V2.
    - The usual transition from S>R in the right precordial leads to R>S in the left precordial leads is V3 or V4.
    - Small "septal" q-waves may be seen in leads V5 and V6.

ST Segment and T wave:

In a sense, the term "ST segment" is a misnomer, because a discrete ST segment distinct from the T wave is usually absent. More often the ST-T wave is a smooth, continuous waveform beginning with the J-point (end of QRS), slowly rising to the peak of the T and followed by a rapid descent to the isoelectric baseline or the onset of the U wave. This gives rise to an asymmetrical T wave. In some normal individuals, particularly women, the T wave is symmetrical and a distinct, horizontal ST segment is present.

The normal T wave is usually in the same direction as the QRS except in the right precordial leads. In the normal ECG the T wave is always upright in leads I, II, V3-6, and always inverted in lead aVR.

Normal ST segment elevation: this occurs in leads with large S waves (e.g., V1-3), and the normal configuration is concave upward. ST segment elevation with concave upward appearance may also be seen in other leads; this is often called early repolarization, although it's a term with little physiologic meaning.

Convex or straight upward ST segment elevation (e.g., leads II, III, aVF) is abnormal and suggests transmural injury or infarction:

ST segment depression is always an abnormal finding, although often nonspecific (see ECG below)

ST segment depression is often characterized as "upsloping", "horizontal", or "downsloping".

The normal U Wave: (the most neglected of the ECG waveforms)

- U wave amplitude is usually < 1/3 T wave amplitude in same lead.
- U wave direction is the same as T wave direction in that lead.
- U waves are more prominent at slow heart rates and usually best seen in the right precordial leads.
- Origin of the U wave is thought to be related to after depolarizations which interrupt or follow repolarization.

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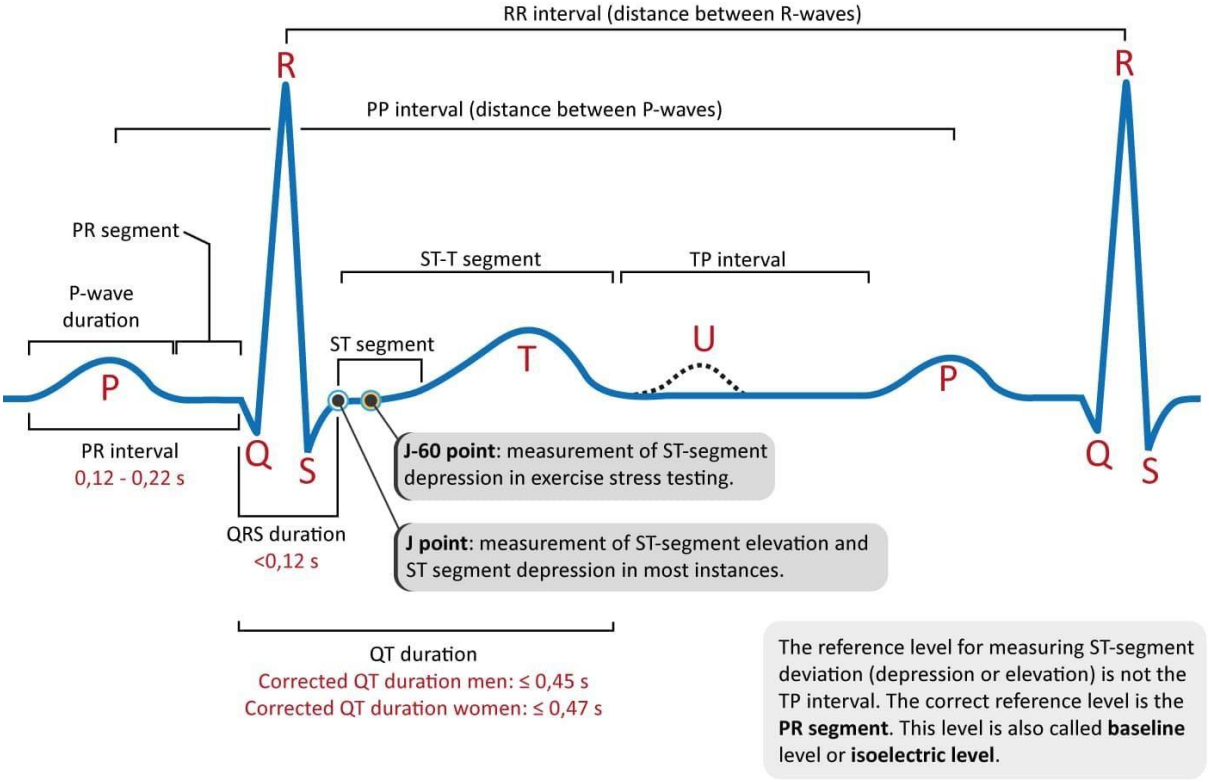
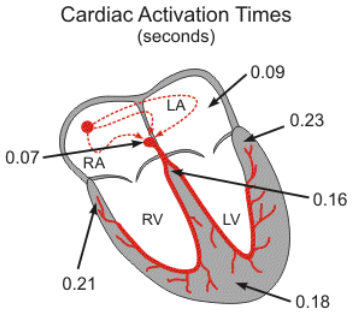


Atrial activation:

is triggered in response to the depolarization of the sinus node and spreads radially and rapidly through the adjacent right **atrial** myocardium.

<u>Atrioventricular node conduction:</u>

The AV node is a highly specialized conducting tissue (cardiac, not neural in origin) that slows the impulse conduction considerably (to about 0.05 m/sec) thereby allowing sufficient time for complete atrial depolarization and contraction (systole) prior to ventricular depolarization and contraction.



The impulses then enter the base of the ventricle at the **Bundle of His** and then follow the **left and right bundle branches** along the interventricular septum. These specialized fibers conduct the impulses at a very rapid velocity (about 2 m/sec). The bundle branches then divide into an extensive system of **Purkinje fibers** that conduct the impulses at high velocity (about 4 m/sec) throughout the ventricles. This results in rapid depolarization of ventricular myocytes throughout both ventricles.

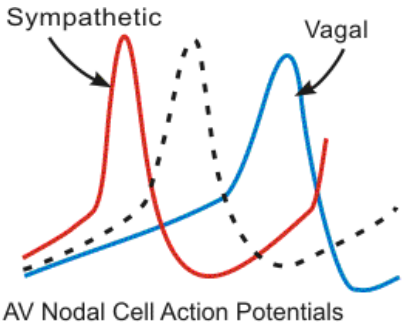
The conduction system within the heart is very important because it permits a rapid and organized depolarization of ventricular myocytes that is necessary for the efficient generation of pressure during systole. The time (in seconds) to activate the different regions of the heart are shown in the figure to the right. Atrial activation is complete within about 0.09 sec (90 msec) following SA nodal firing. After a delay at the AV node, the septum becomes activated (0.16 sec). All the ventricular mass is activated by about 0.23 sec.

Regulation of Conduction:

Conduction velocity is altered by:

- Sympathetic stimulation (increases)
- Vagal stimulation (decreases)
- Ischemia/hyoxia (decreases)
- Drugs (adrenergic & cholinergic) (increase or decrease)

The conduction of electrical impulses throughout the heart, and particularly in the specialized conduction system, is influenced by autonomic nerve activity. This autonomic control is most apparent at the AV node. Sympathetic activation increases conduction velocity in the AV node by increasing the rate of depolarization (increasing slope of phase 0) of the action potentials. This leads to more rapid depolarization of adjacent cells, which leads to a more rapid conduction of action potentials (positive dromotropy). Sympathetic activation of the AV node reduces the normal delay of conduction through the AV node, thereby reducing the time between atrial and ventricular contraction. The increase in AV nodal conduction velocity can be seen as a decrease in the P-R interval of the electrocardiogram.



Effects of Parasympathetic (Vagal) and Sympathetic Nerve Activation on AV Nodal Action Potentials

Sympathetic nerves exert their actions on the AV node by releasing the neurotransmitter norepinephrine that binds to beta-adrenoceptors, leading to an increase in intracellular cAMP. Therefore, drugs that block beta-adrenoceptors (beta-blockers) decrease conduction velocity and can produce AV block.

Parasympathetic (vagal) activation decreases conduction velocity (negative dromotropy) at the AV node by decreasing the slope of phase 0 of the nodal action potentials. This leads to slower depolarization of adjacent cells, and reduced velocity of conduction. Acetylcholine, released by the vagus nerve, binds to cardiac muscarinic receptors, which decreases intracellular cAMP. Excessive vagal activation can produce AV block. Drugs such as digitalis, which increase vagal activity to the heart, are sometimes used to reduce AV nodal conduction in patients that have atrial flutter or fibrillation. These atrial arrhythmias lead to excessive ventricular rate (tachycardia) that can be suppressed by partially blocking impulses being conducted through the AV node.

Phase 0 of action potentials at the AV node is not dependent on fast sodium channels as in non-nodal tissue, but instead is generated by the entry of calcium into the cell through slow-inward, L-type calcium channels. Blocking these channels with a calcium-channel blocker such as verapamil or diltiazem reduces the conduction velocity of impulses through the AV node and can produce AV block.

Because conduction velocity depends on the rate of tissue depolarization, which is related to the slope of phase 0 of the action potential, conditions (or drugs) that alter phase 0 will affect conduction velocity. For example, conduction can be altered by changes in membrane potential, which can occur during myocardial ischemia and hypoxia. In non-nodal cardiac tissue, cellular hypoxia leads to membrane depolarization, inhibition of fast Na^+ channels, a decrease in the slope of phase 0, and a decrease in action potential amplitude. These membrane changes result in a decrease in speed by which action potentials are conducted within the heart. This can have a number of consequences. First, activation of the heart will be delayed, and in some cases, the sequence of activation will be altered. This can seriously impair ventricular pressure development. Second, damage to the conducting system can precipitate tachyarrhythmias by reentry mechanisms. Click here to learn more about altered impulse conduction.

Antiarrhythmic drugs such as quinidine (a Class IA antiarrhythmic) that block fast sodium channels cause a decrease in conduction velocity in non-nodal tissue.

PR-segment:

The PR segment is the flat line between the end of the P-wave and the start of the QRS complex. The PR segment reflects the time delay between atrial and ventricular activation. The PR segment also serves as the baseline (reference line or isoelectric line) of the ECG curve.

PR segment abnormalities:

These occur in two main conditions:

- Pericarditis

- Atrial ischaemia

Pericarditis:

The characteristic changes of acute pericarditis are:
- PR segment depression.
- Widespread concave (‘saddle-shaped’) ST elevation.
- Reciprocal ST depression and PR elevation in aVR and V1
- Absence of reciprocal ST depression elsewhere.

QRS complex:

The QRS complex is the combination of three of the graphical deflections seen on a typical electrocardiogram (EKG or ECG). It is usually the central and most visually obvious part of the tracing; in other words, it's the main spike seen on an ECG line. It corresponds to the depolarization of the right and left ventricles of the human heart and contraction of the large ventricular muscles.

In adults, the QRS complex normally lasts 0.06–0.10 s; in children and during physical activity, it may be shorter. The Q, R, and S waves occur in rapid succession, do not all appear in all leads, and reflect a single event and thus are usually considered together. A Q wave is any downward deflection immediately following the P wave. An R wave follows as an upward deflection, and the S wave is any downward deflection after the R wave. The T wave follows the S wave, and in some cases, an additional U wave follows the T wave.

Formation:

Depolarization of the heart ventricles occurs almost simultaneously, via the bundle of His and Purkinje fibers. If they are working efficiently, the QRS complex is 80 to 120 ms in duration. This is represented by three small squares or less at the standard paper speed of 25 mm/s.

Clinical significance:

Any abnormality of conduction takes longer and causes "widened" QRS complexes. In bundle branch block, there can be an abnormal second upward deflection within the QRS complex. In this case, such a second upward deflection is referred to as R' (pronounced "R prime"). This would be described as an RSR' pattern.

Ventricles contain more muscle mass than the atria. Therefore, the QRS complex is considerably larger than the P wave. The QRS complex is often used to determine the axis of the electrocardiogram, although it is also possible to determine a separate P wave axis.

The duration, amplitude, and morphology of the QRS complex are useful in diagnosing cardiac arrhythmias, conduction abnormalities, ventricular hypertrophy, myocardial infarction, electrolyte derangements, and other disease states.

High frequency analysis of the QRS complex may be useful for detection of coronary artery disease during an exercise stress test.

ST, T, and U wave abnormalities:

Basic Concept: the specificity of ST-T and U wave abnormalities is provided more by the clinical circumstances in which the ECG changes are found than by the particular changes themselves. Thus the term, nonspecific ST-T wave abnormalities, is frequently used when the clinical data are not available to correlate with the ECG findings. This does not mean that the ECG changes are unimportant.

Factors affecting the ST-T and U wave configuration include:

- Intrinsic myocardial disease (e.g., myocarditis, ischemia, infarction, infiltrative or myopathic processes)
- Drugs (e.g., digoxin, quinidine, tricyclics, and many others)
- Electrolyte abnormalities of potassium, magnesium, calcium
- Neurogenic factors (e.g., stroke, hemorrhage, trauma, tumor, etc.)
- Metabolic factors (e.g., hypoglycemia, hyperventilation)
- Atrial repolarization (e.g., at fast heart rates the atrial T wave may pull down the beginning of the ST segment)
- Ventricular conduction abnormalities and rhythms originating in the ventricles

"Secondary" ST-T Wave changes (these are normal ST-T wave changes solely due to alterations in the sequence of ventricular activation):

- ST-T changes seen in bundle branch blocks (generally the ST-T polarity is opposite to the major or terminal deflection of the QRS)
- ST-T changes seen in fascicular block
- ST-T changes seen in nonspecific IVCD
- ST-T changes seen in WPW preexcitation
- ST-T changes in PVCs, ventricular arrhythmias, and ventricular paced beats

"Primary" ST-T Wave Abnormalities (ST-T wave changes that are independent of changes in ventricular activation and that may be the result of global or segmental pathologic processes that affect ventricular repolarization):

- Drug effects (e.g., digoxin, quinidine, etc)
- Electrolyte abnormalities (e.g., hypokalemia)
- Ischemia, infarction, inflammation, etc
- Neurogenic effects (e.g., subarrachnoid hemorrhage causing long QT)

Rate and rhythm:

Normal heart rhythms: The heart's normal rhythm is called sinus rhythm. Its rate is between 60 and 100 beats per minute (bpm) while you are resting. If the sinus rhythm is slower than 60 bpm, it is called sinus bradycardia. If the sinus rhythm is faster than 100 bpm, it is called sinus tachycardia. ('Brady' means slow and 'tachy' means fast.) The normal heart rate varies from minute to minute, depending on the demands on the heart. Sinus arrhythmia is a normal variation of sinus rhythm, where the heart rate increases very slightly as you take a breath in. Sinus rhythm, sinus bradycardia, sinus tachycardia and sinus arrhythmia are all normal heart rhythms where the electrical impulses travel in a normal way through the heart

What are the symptoms of abnormal heart rhythms?

If you have an abnormal heart rhythm, you may experience some or all of these symptoms:

- feeling faint, dizzy, or light-headed
- shortness of breath
- irregular pulse or heart palpitations
- chest pain
- pale skin
- sweating

What causes abnormal heart rhythms?

A number of things may cause an abnormal heartbeat, including high blood pressure. Other common causes are:

Coronary heart disease:

This serious heart problem occurs when cholesterol and other deposits block the coronary arteries.

What are the risk factors for abnormal heart rhythms?

The risks for arrhythmia include:

- smoking
- previous heart conditions, or a family history of heart conditions
- diabetes
- stress

- being overweight
- living a sedentary lifestyle
- a diet high in fats, cholesterol, and other unhealthy foods
- high blood pressure or other health problems
- excessive use of alcohol (more than two drinks per day)
- drug misuse
- sleep apnea

Diagnosing abnormal heart rhythms: doctor will perform a physical examination, which will include using a stethoscope to listen to your heart. They may also use an electrocardiogram (EKG or ECG) machine to examine the electrical impulses of heart. This will help them determine whether heart rhythm is abnormal and identify the cause.

Other tools that can be used to diagnose an arrhythmia include:

- Echocardiogram. Also known as a cardiac echo, this test uses sound waves to take pictures of your heart.
- Holter monitor. You wear this monitor for at least 24 hours while you go about your normal activities. It allows your doctor to track changes in your heart's rhythm throughout the day.
- Stress test. For this test, your doctor will make you walk or jog on a treadmill to see how exercise affects your heart.