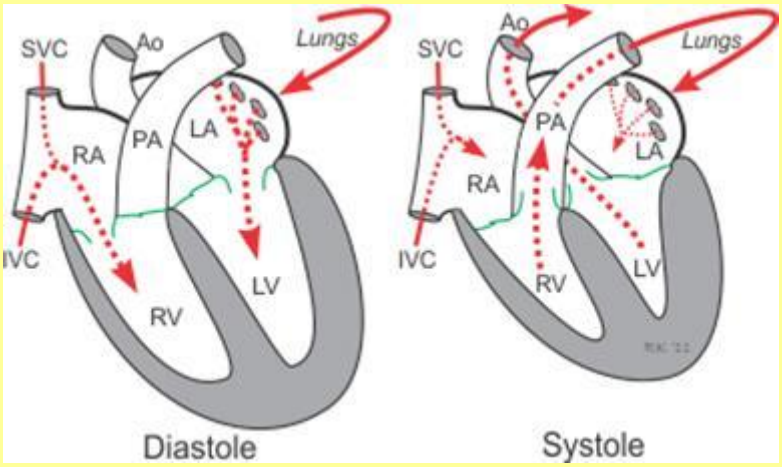
ELECTROCARDIOGRAPHY AS A METHOD OF OBSERVATION

Cardiac Cycle

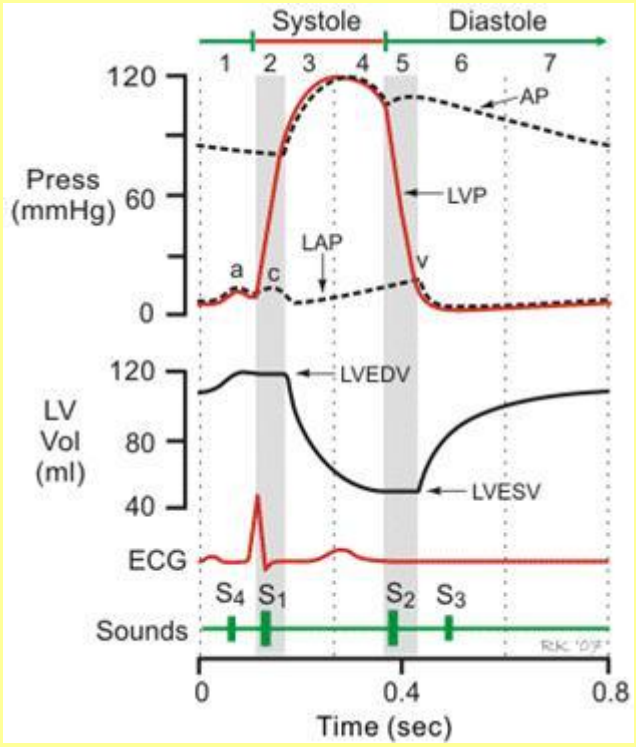


A single cycle of cardiac activity can be divided into two basic phases - ***diastole and systole***.

Diastole represents the period of time when the ventricles are relaxed (not contracting).Throughout most of this period, blood is passively flowing from the left atrium (LA) and right atrium (RA) into the left ventricle (LV) and right

ventricle (RV), respectively (see figure at right). The blood flows through atrioventricular valves (mitral and tricuspid) that separate the atria from the ventricles. The RA receives venous blood from the body through the superior vena cava (SVC) and inferior vena cava (IVC). The LA receives oxygenated blood from lungs through four pulmonary veins that enter the LA. At the end of diastole, both atria contract, which propels an additional amount of blood into the ventricles.

Systole represents the time during which the left and right ventricles contract and eject blood into the aorta and pulmonary artery, respectively. During systole, the aortic and pulmonic valves open to permit ejection into the aorta and pulmonary artery. The atrioventricular valves are closed during systole, therefore no blood is entering the ventricles; however, blood continues to enter the atria though the vena cavae and pulmonary veins.



The cardiac cycle diagram shown to the right depicts changes in aortic pressure (AP), left ventricular pressure (LVP), left atrial pressure (LAP), left ventricular volume (LV Vol), and heart sounds during a single cycle of cardiac contraction and relaxation. These changes are related in time to the electrocardiogram. Aortic pressure is measured by inserting a pressure catheter into the aorta from a peripheral artery, and the left ventricular pressure is obtained by placing a pressure catheter inside the left ventricle and measuring changes in intraventricular pressure as the heart beats. Left atrial pressure is not usually measured directly, except in investigational procedures; however, left atrial pressure can be estimated by recording the pulmonary capillary wedge pressure. Ventricular volume changes can be assessed in real time using echocardiography or radionuclide imaging, or by using a special volume conductance catheter placed within the ventricle. To analyze systole and diastole in more detail, the cardiac cycle is usually divided into

seven phases. The first phase begins with the P wave of the electrocardiogram, which represents atrial depolarization, and is the last phase of diastole. Phases 2-4 represent systole, and phases 5-7 represent early and mid-diastole. The last phase of the cardiac cycle ends with the appearance of the next P wave, which begins a new cycle. Detailed descriptions of each phase can be obtained by clicking on each of the seven phases listed below.

Phase 1 – Atrial Contraction

Phase 2 – Isovolumetric Contraction

Phase 3 – Rapid Ejection

Phase 4 – Reduced Ejection

Phase 5 – Isovolumetric Relaxation

Phase 6 – Rapid Filling

Phase 7 – Reduced Filling

The Genesis and Conduction of Cardiac Rhythm

There are four main electrophysiological properties of the Cardiac Muscle:

- Automaticity

- Conductibility

- Excitability

- Contractibility

Automaticity is the cardiac cell's ability to spontaneously generate an electrical impulse (depolarize).

Contractility is property to contract replying to irritation.

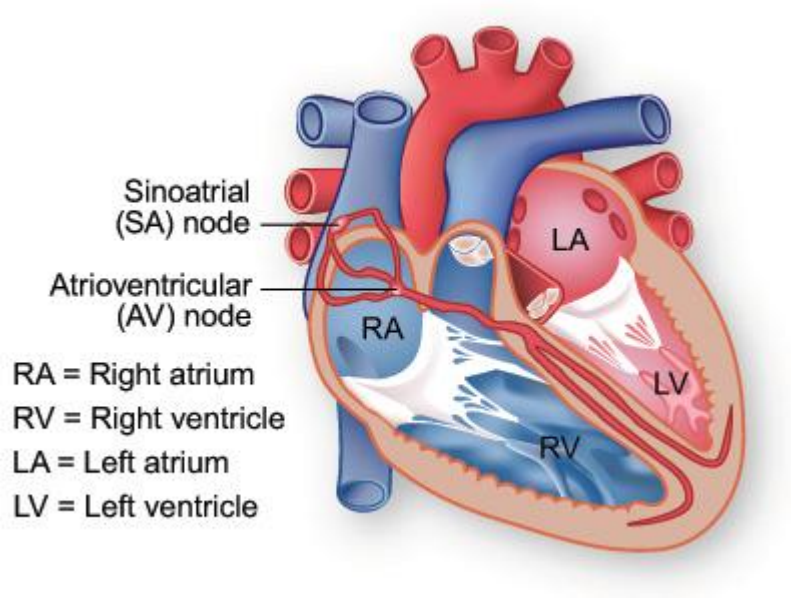
Conductibility is property to spread electrical impulses through the conduction system and contractive myocardium.

Excitability is property to reply the irritation.

Automaticity of heart

Cells that are dedicated to the purpose of generating an impulse to maintain a heart rate commensurate with the body's need are called ***pacemaker cells***. Cells normally capable of generating an impulse are the sinus node, cells of the AV node, the bundle of His and Purkinje cells. The lower the site of the latent pacemaker, the slower the intrinsic rhythm.

The sinus node is located at the intersection of the superior vena cava and the right atrium laterally.



While the sinus node cells have an intrinsic automaticity of their own, they are influenced by the sympathetic and parasympathetic nervous system. The former increases the heart rate while the latter slows the heart rate.

During sleep and in highly trained athletes, the parasympathetic nervous system tone is elevated and the heart rate is slow. During exertion and any other condition that the sympathetic nervous system tone is high, the heart rate is elevated. Cardiac cells with automaticity displaying the fastest rate take over pacemaker cells with slower rates. The sinus node has the fastest intrinsic rate, other cells capable of generating a rhythm (automaticity) are termed latent pacemakers. The can take over pacing the heart if the sinus node becomes dysfunctional (sick sinus syndrome).

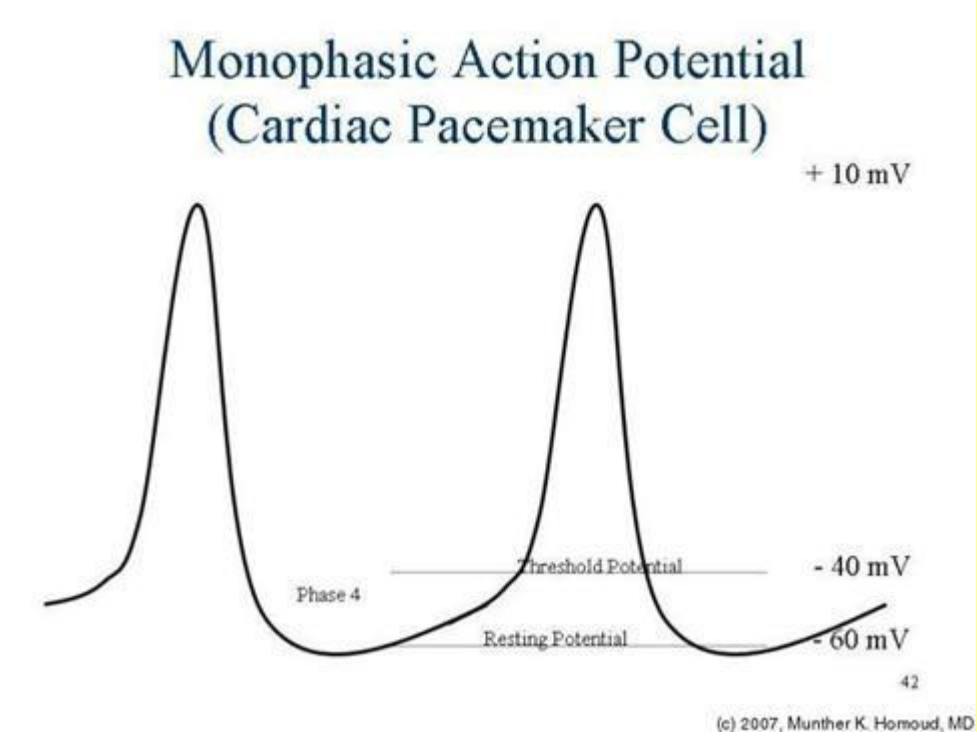
Normally, the interior of cardiac cells are more negative than the exterior due to the distribution of the main intra and extracellular electrolytes; Na+, K+, Ca++ and Cl-. The voltage differential between the interior and the exterior at rest (diastole) is called the ***Resting Potential***. In myocardial cells, the interior is maintained more negative than the exterior by the extrusion of 3 Na+ ions for every 2 K+ ions pumped in by the Na+/K+ATPase pump. The resting potential of sinus node cells is -50 to – 60 mV, for ventricular muscle cells it is -80 to – 90 mV. Cardiac cells endowed with automaticity depolarize spontaneously at a rate commensurate with the intrinsic rate of the cell. Once the membrane potential reaches a certain voltage

it fires (depolarized) spontaneously. This voltage is called the ***threshold potential***. For sinus node cells this threshold potential is approximately – 40 mV. Compared with ventricular muscle cells, cells with automaticity spend little time at the resting potential beginning to depolarize soon after reaching their resting potential. The rate of spontaneous depolarization (automaticity) is dependent upon the slope of Phase 4 of the action potential (If), the level of the membrane resting potential and the level of the threshold potential (see figure below).

Movement of electrolytes across the impermeable cell membrane is through a number of channels (Na channels (INa), K channels (IK), e.g.) that permit or prevent the movement of ions depending upon transmembrane voltage. The channel responsible for spontaneous depolarization (Phase 4) is called If . The monophasic action potential (MAP) describes 2 the changes in transmembrane potential plotted against time. The MAP of pacemaker cells such as the sinus node differs from the MAP of conduction and myocardial cells.

Whereas calcium is the main electrolyte responsible for pacemaker cell depolarization, sodium is the main electrolyte responsible for depolarization of myocardial cells and cells dedicated to conduction of impulses.

The monophasic action potential of a cardiac cell endowed with automaticity. The rate of firing is dependent upon 1) the slope of Phase 4 (the steeper the faster the rate of depolarization), 2) the level of the resting membrane potential (the less depolarized the faster for any given phase 4 slope and firing threshold level) and 3) the level of the threshold potential (the closer to the resting potential, the faster the rate of spontaneous) depolarization.



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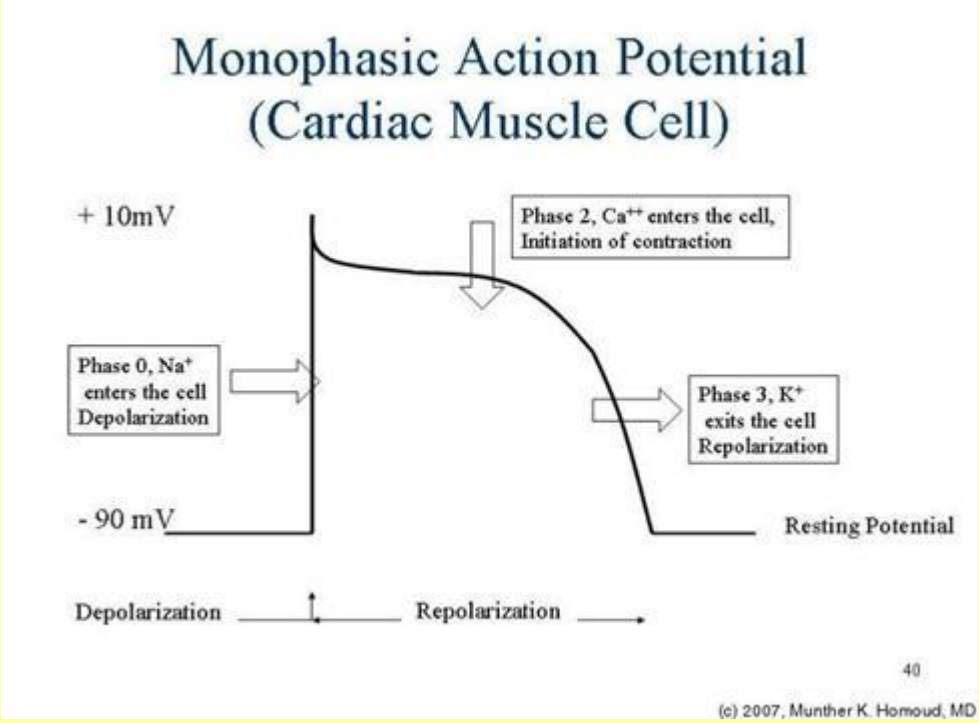


Conductive system of the heart

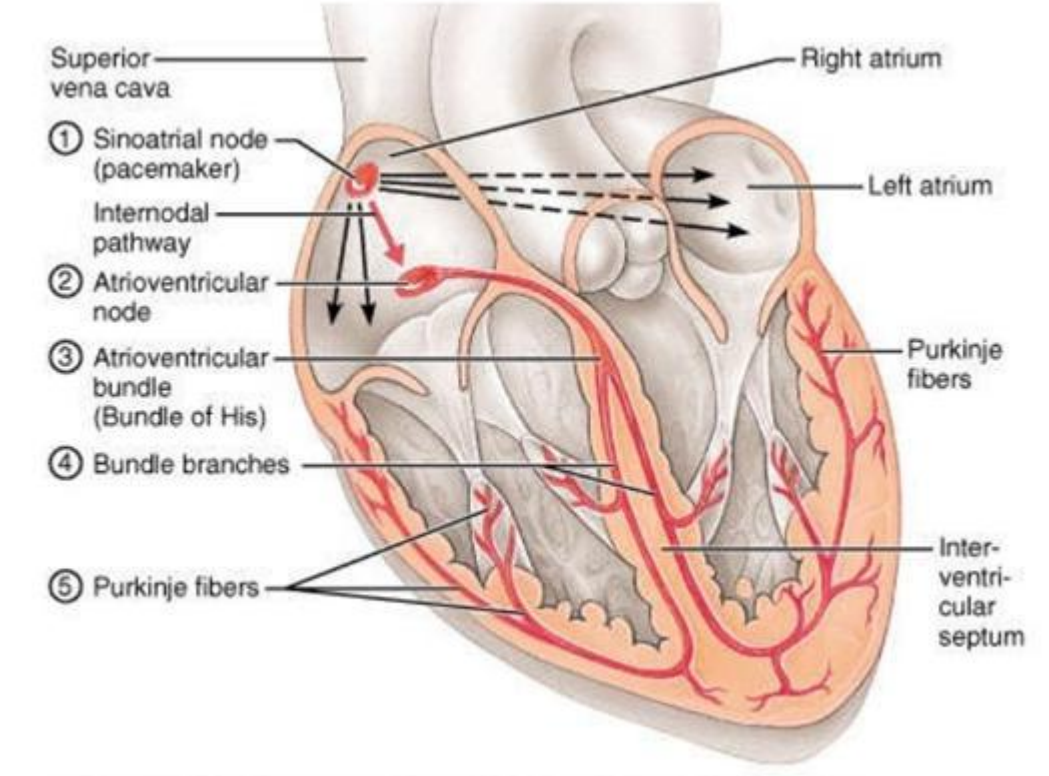
1. Sinus node "SA" node: also called sinoatrial node, located in the right atrium. It is concerned with the generation of rhythmical impulse; it is the pacemaker of the heart that initiates each heart beat. This automatic nature of the heart beat is referred to as automaticity.

2. Internodal pathways conduct the impulse generated in SA node to the AV node.

3. The AV node (atrioventricular node), located near the right AV valve at the lower end of the interatrial septum, in the posterior septal wall of the right atrium. At which impulse from the atria is delayed before passing into the ventricles.

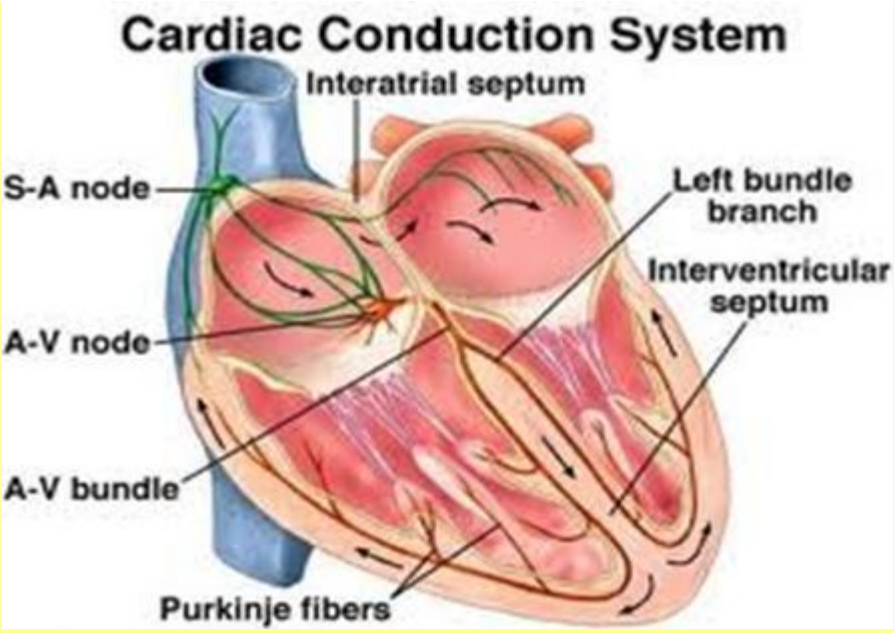
4. The AV bundle (bundle of His) conducts the impulse from the atria into ventricles.

5. The left and right bundles of Purkinje fibers, which conduct the cardiac impulse to all parts of the ventricles. The purkinje fibers distribute the electrical excitation to the myocytes of the ventricles.



Cardiac Conduction System

The impulse generated in the ***sinus node*** depolarizes the surrounding atrium and conducts to the ventricle via the ***AV node***. Conduction below the AV node and into the myocardial cells is via the specialized ***His-Purkinje system***. These cells are specialized conduction cells and are localized to the ***Right and Left Bundle Branches***. Cell-to-cell conduction across normal cells occurs across gap junctions. Within the gap junctions are different types of proteins that are responsible for cell-to-cell conduction of electrical impulses called connexins. Electrical conduction along the longitudinal axis of the cell is faster than it is perpendicular to its long axis.



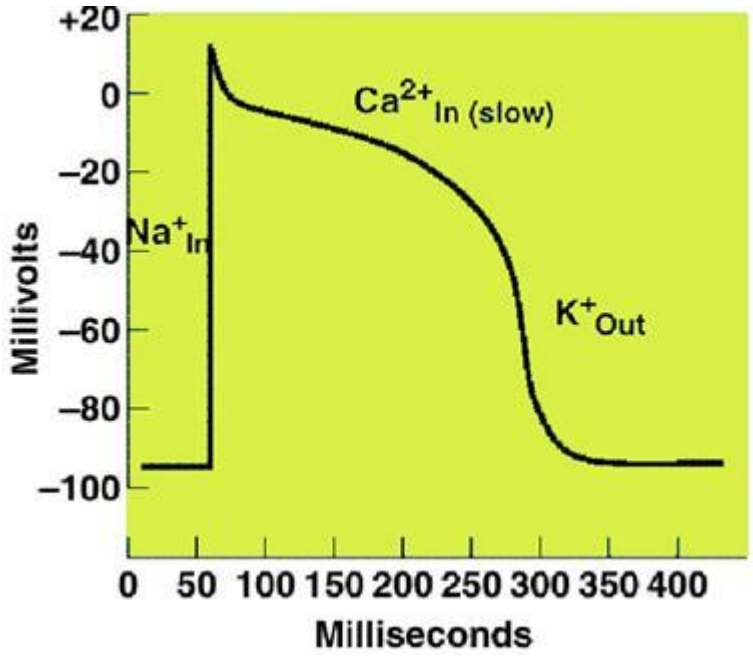
The rhythmic sequence of contractions is coordinated by the sinoatrial (SA) and atrioventricular (AV) nodes. The sinoatrial node, often known as the cardiac pacemaker, is located in the upper wall of the right atrium and is responsible for the wave of electrical stimulation that initiates atrial contraction. Once the wave reaches the AV node, situated in the lower right atrium, it is delayed there before being conducted through the bundles of His and back up the Purkinje fibers, leading to a contraction of the ventricles. The delay at the AV node allows enough time for all of the blood in the atria to fill their respective ventricles. In the event of severe pathology, the AV node can also act as a pacemaker; this is usually not the case because their rate of spontaneous firing is considerably lower than that of the pacemaker cells in the SA node and hence is overridden.

Excitability of heart

In common with other muscle tissue, the heart muscle cell membrane is an excitable membrane i.e., it is capable of transmitting an action potential. Also in common with other muscle tissue, the depolarisation is due to the opening of the fast sodium channels. The unique characteristic of cardiac muscle action potential is the plateau phase i.e. the maintenance of the potential at a positive level. The plateau is the result of slow sodium/calcium channels that remain open for severalhundred milliseconds.

Repolarisation is produced by the closing of the fast sodium and slow sodium/calcium channels and opening of the potassium channels.

The prolonged depolarisation ensures that the absolute refractory period (ARP) of the heart muscle cells is relatively longer than that of other muscle cells. This prolonged ARP results in the fact that the heart muscle cell can never be tetanised - important as for normal pumping action of the heart requires some diastole to fill with blood.



Factors influencing Excitability

Factors that increase excitability:

- Sympathetic stimulation - Lowers the resting membrane potential - becoming more p
- Mild Hyperkaleamia (increase in potassium concentration) - partial depolarisation
- Hypocalcaemia (decrease in calcium concentration) - partial depolarisation
- Adrenaline - similar to sympathetic stimulation
- Digitalis - increase atrial muscle excitability
- Mild Hypoxia - Partial depolarisation
- Ischaemia - Partial depolarisation

Factors that decrease excitability:

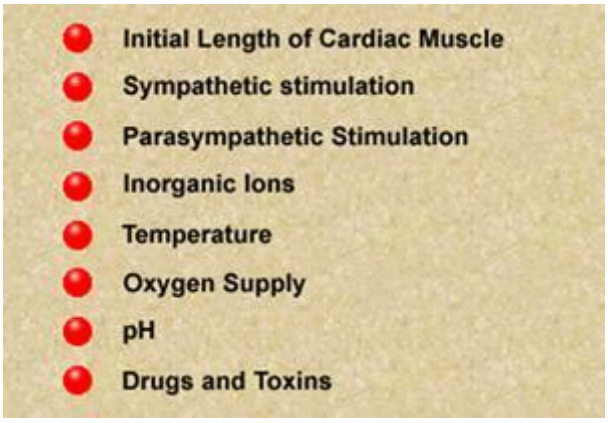
- Parasympathetic Stimulation - decreases excitability only of the atrial muscle cells
- Hyponatreamia (decrease in sodium concentration)
- Marked Hyperkaleamia - Marked depolarisation
- Hypokaleamia - Hyperpolarisation
- Hypercalceamia - decreases permeability to sodium

- Acetyl choline - same as parasympthetic stimulation
- Digitalis - decreases ventricular muscle excitability
- Marked Hypoxia - marked depolarisation

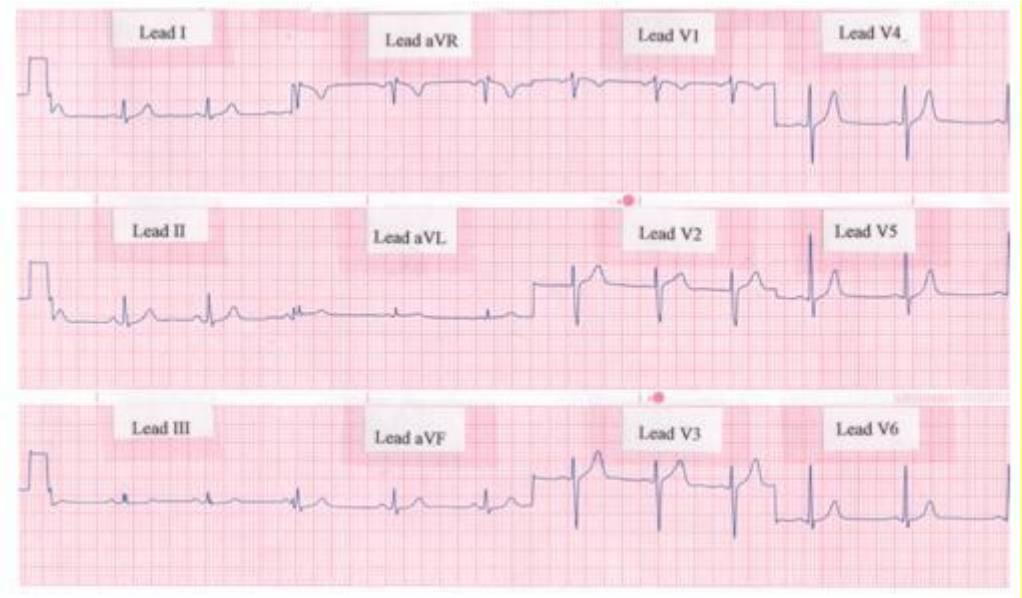
Contractility of heart

- Contraction of the heart is called **systole** whilst relaxation of the heart is called **diastole**
- As the function of the heart is that of a pump, both actions are important - a pump has to can pump it out. The filling process occurs during diastole.
- One systole and its following diastole is called **one cardiac cycle**.
- In the normal heart, beating at a rate 0f 75 beats/min, the duration of ventricular systole diastole is around 0.5 sec (total length of cycle 0.8 sec)

Factors that influence cardiac contractility include:



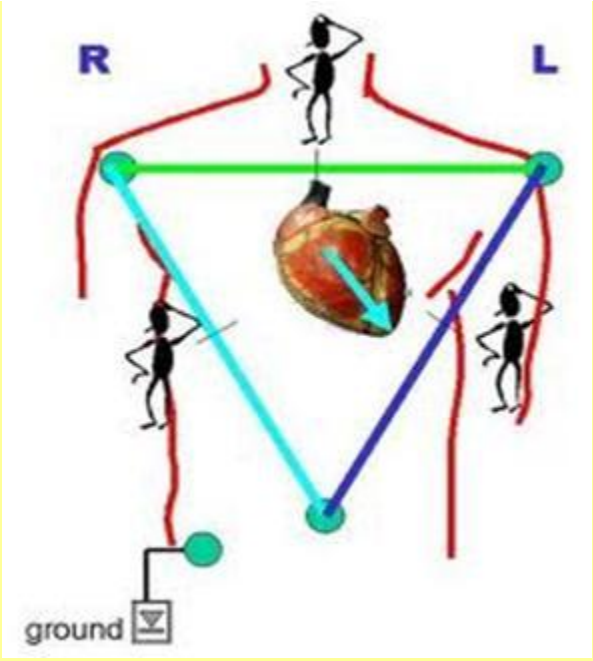
FORMATION OF NORMAL ELECTROCARDIOGRAM



An electrocardiograph is an instrument that measures and records the electrocardiogram (ECG), the electrical activity generated by the heart. Electrodes placed on various anatomical sites on the body help conduct the ECG to the electrocardiograph. The ECG alone is not sufficient to diagnose all abnormalities possible in the pacing or conduction system of the heart. The interpretation of the 12-lead ECG provides a differential diagnosis for many arrhythmias

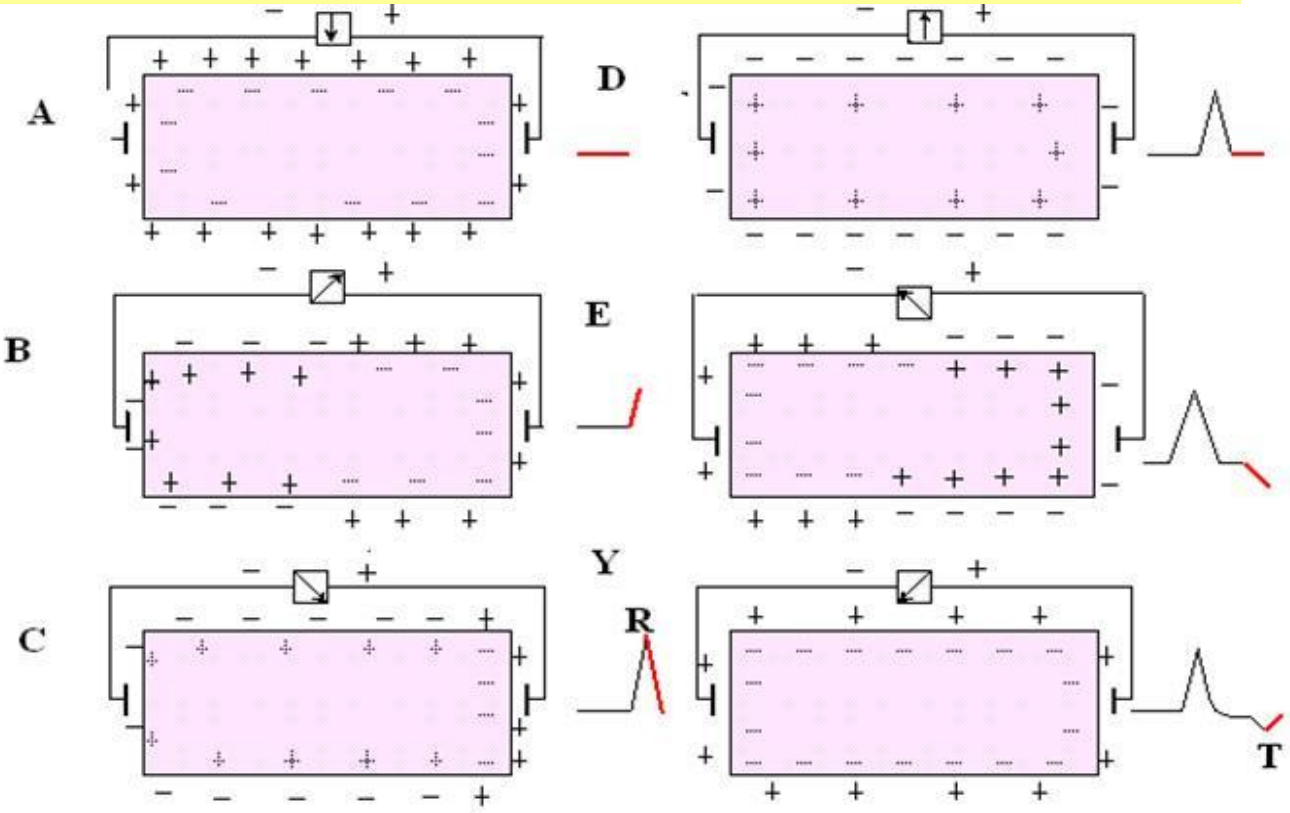
During the depolarization phase the rapid Na^+ gates open and inward diffusion of Na^+ occur. This event corresponds to formation upward part of positive wave on electro gram line. The next fast initial repolarization begins with inward Cl^- diffusion. Then electro gram returns to baseline level. When opening slow Ca^{2+} gates the potential difference temporarily isn't essential and baseline continues. During the next phase outward K+ diffusion increases and external surface of membrane becomes positive. Voltage fluctuation leads to deflection of electro gram downward further returning to baseline level. In rest period all the membrane has positive charge on external surface and baseline is recorded. Through this period ion pumps restore initial distribution of ions.

Unexcited part of cell has already positive charge. Depolarized part has already negative charge. Between positive and negative charges the electrical power is recorded. Electrical power directs towards positive voltage. When changing polarity, electrical power of entire heart has different volume and direction in every moment of cardiac cycle.



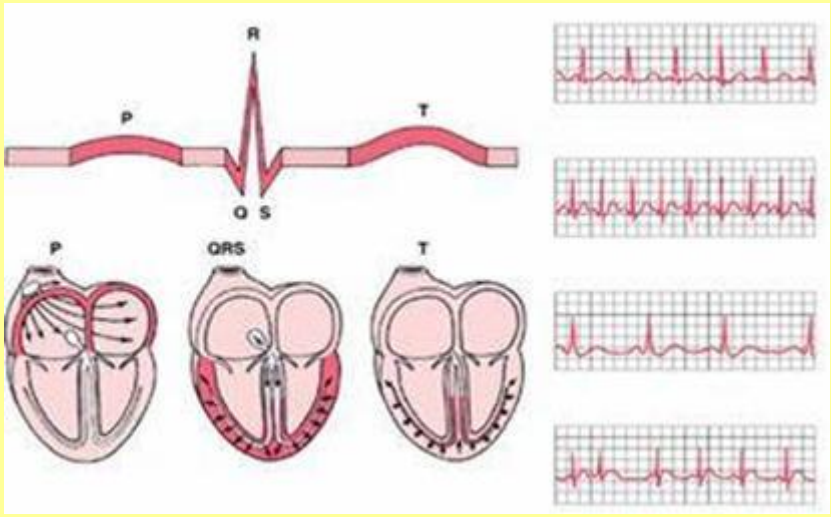
To understand formation of ECG waves in different leads it is necessary to remember some rulers of this process:

- When electrical power directs towards positive pole of lead the upward wave is recorded;
- When electrical power directs towards negative lead pole, the downward wave occurs;
- If electrical power directs perpendicular to lead axis the baseline is recorded.

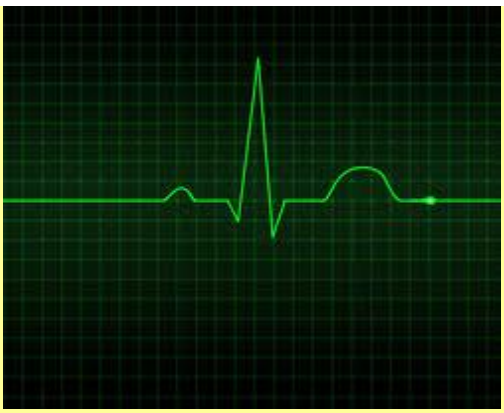


Every cardiac cycle produces ECG waves designated as P, Q, R, S and T. These waves are not action potentials. They represent potentials between rested and depolarized or depolarized and repolarized parts of whole heart. Amplitude and duration of these waves correspond to electrical power fluctuation in entire heart.

After producing impulse in SA-node depolarization begins at first in cells of right atrium and ascend part of P wave is recorded. When depolarization spreads into left atrium, the ECG line returns to baseline level. Delay of depolarization in AV-node recorded as PQ-interval in baseline. Then impulse spreads into middle part of septum and heart apex. This event recorded as descend part of Q wave. In next depolarization of right ventricle wall ECG line deflexed upward and formation of R wave begins. When impulse spreads into left ventricle wall, the ECG line returned in contrary side towards the lowest point of S wave. Depolarization of ventricles basis afterwards caused formation of S wave, which continues to baseline.



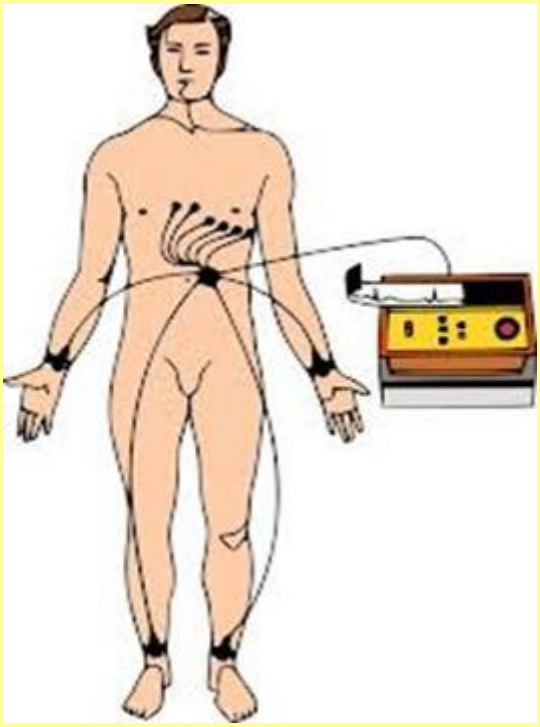
Electrocardiogram (EKG, ECG).



Electrocardiography is a transthoracic (across the thorax or chest) interpretation of the electrical activity of the heart over a period of time, as detected by electrodes attached to the surface of the skin and recorded by a device external to the body. The recording produced by thisnoninvasive procedure is termed an electrocardiogram (also ECG or EKG).

An ECG is used to measure the rate and regularity of heartbeats, as well as the size and position of the chambers, the presence of any damage to the heart, and the effects of drugs or devices used to regulate the heart, such as a pacemaker.

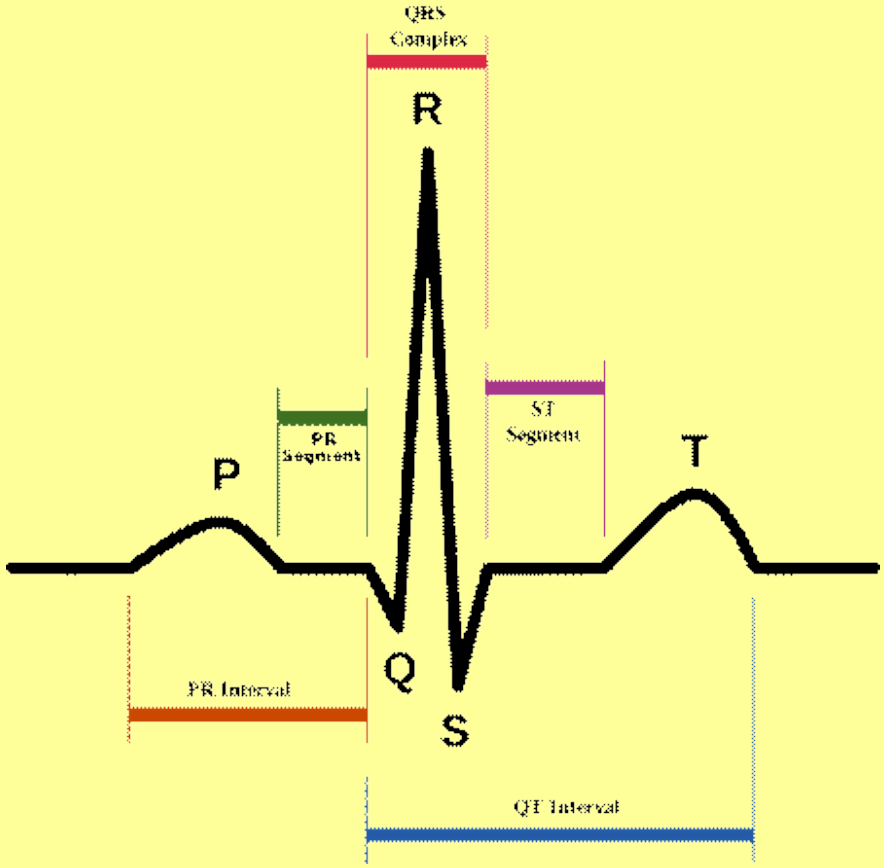
Most ECGs are performed for diagnostic or research purposes on human hearts, but may also be performed on animals, usually for diagnosis of heart abnormalities or research.



The electrical activity generated by the heart can be measured by an array of electrodes placed on the body surface.

As the heart undergoes depolarization and repolarization, the electrical currents that are generated spread not only within the heart, but also throughout the body. The electrical activity generated by the heart can be measured by an array of electrodes placed on the body surface The different waves that comprise the ECG represent the sequence of depolarization and repolarization of the atria and ventricles.

Waves and intervals



P wave

The P wave represents the wave of depolarization that spreads from the SA node throughout the atria, and is usually 0.08 to 0.1 seconds (80-100 ms) in duration.

The brief isoelectric (zero voltage) period after the P wave represents the time in which the impulse is traveling within the AV node (where the conduction velocity is greatly retarded) and the bundle of His. Atrial rate can be calculated by determining the time interval between P waves.

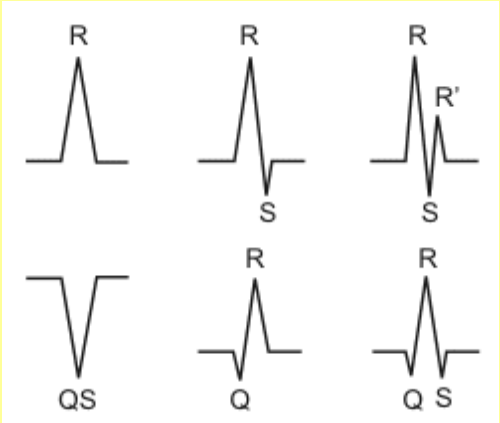
The period of time from the onset of the P wave to the beginning of the QRS complex is termed the ***P-R interval***, which normally ranges from 0.12 to 0.20 seconds in duration. This interval represents the time between the onset of atrial depolarization and the onset of ventricular depolarization. If the P-R interval is >0.2 sec, there is an AV conduction block, which is also termed a first-degree heart block if the impulse is still able to be conducted into the ventricles.

QRS complex

The QRS complex represents ventricular depolarization. Ventricular rate can be calculated by determining the time interval between QRS complexes.

The duration of the QRS complex is normally 0.06 to 0.1 seconds. This relatively short duration indicates that ventricular depolarization normally occurs very rapidly. If the QRS complex is prolonged (> 0.1 sec), conduction is impaired within the ventricles. This can occur with bundle branch blocks or whenever a ventricular foci (abnormal pacemaker site) becomes the pacemaker driving the ventricle. Such an ectopic foci nearly always results in impulses being conducted over slower pathways within the heart, thereby increasing the time for depolarization and the duration of the QRS complex.

The shape of the QRS complex in the above figure is idealized. In fact, the shape changes depending on which recording electrodes are being used. The shape will also change when there is abnormal conduction of electrical impulses within the ventricles. The figure to the right summarizes the nomenclature used to define the different components of the QRS complex.



ST segment

The isoelectric period (ST segment) following the QRS is the time at which the entire ventricle is depolarized and roughly corresponds to the plateau phase of the ventricular action potential. The ST segment is important in the

diagnosis of ventricular ischemia or hypoxia because under those conditions, the ST segment can become either depressed or elevated.

T wave

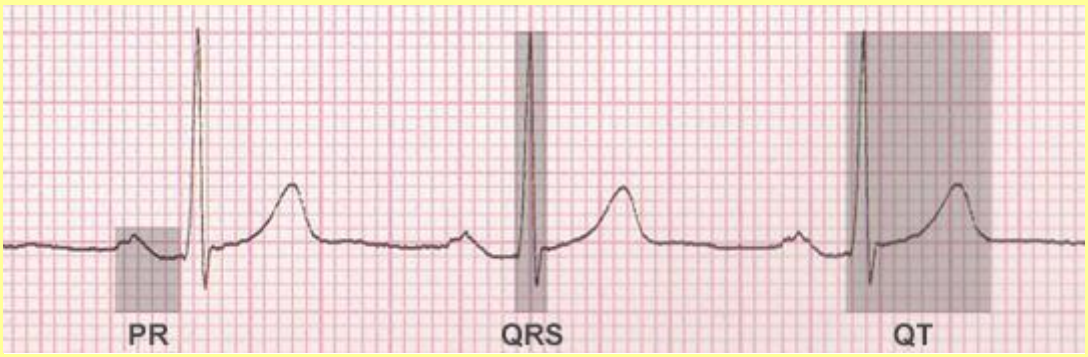
The T wave represents ventricular repolarization and is longer in duration than depolarization (i.e., conduction of the repolarization wave is slower than the wave of depolarization). Sometimes a small positive U wave may be seen following the T wave (not shown in figure at top of page). This wave represents the last remnants of ventricular repolarization. Inverted or prominent U waves indicates underlying pathology or conditions affecting repolarization.

Q-T interval

The Q-T interval represents the time for both ventricular depolarization and repolarization to occur, and therefore roughly estimates the duration of an average ventricular action potential. This interval can range from 0.2 to 0.4 seconds depending upon heart rate. At high heart rates, ventricular action potentials shorten in duration, which decreases the Q-T interval. Because prolonged Q-T intervals can be diagnostic for susceptibility to certain types of tachyarrhythmias, it is important to determine if a given Q-T interval is excessively long. In practice, the Q-T interval is expressed as a "corrected Q-T (QTc)" by taking the Q-T interval and dividing it by the square root of the R-R interval (interval between ventricular depolarizations). This allows an assessment of the Q-T interval that is independent of heart rate. Normal corrected Q-Tc intervals are less than 0.44 seconds.

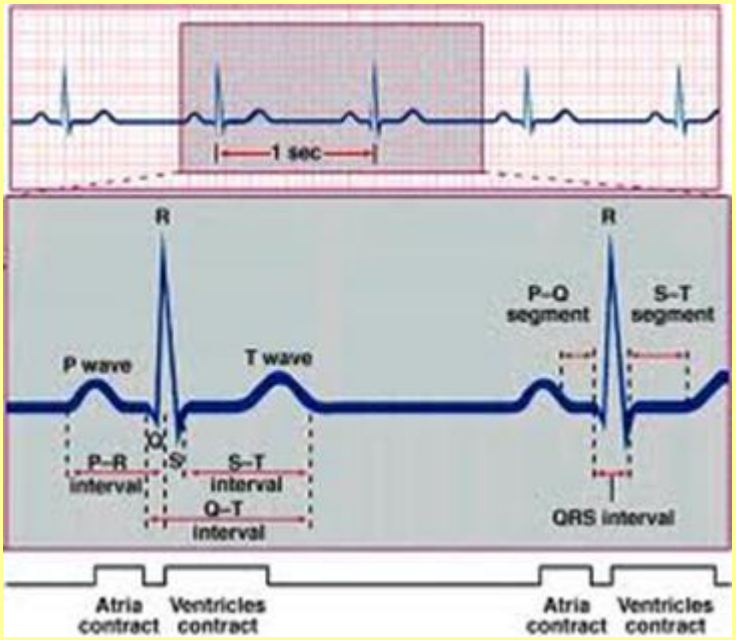
There is no distinctly visible wave representing atrial repolarization in the ECG because it occurs during ventricular depolarization. Because the wave of atrial repolarization is relatively small in amplitude (i.e., has low voltage), it is masked by the much larger ventricular-generated QRS complex.

ECG tracings recorded simultaneous from different electrodes placed on the body produce different characteristic waveforms.



PR, QT intervals, QRS complex

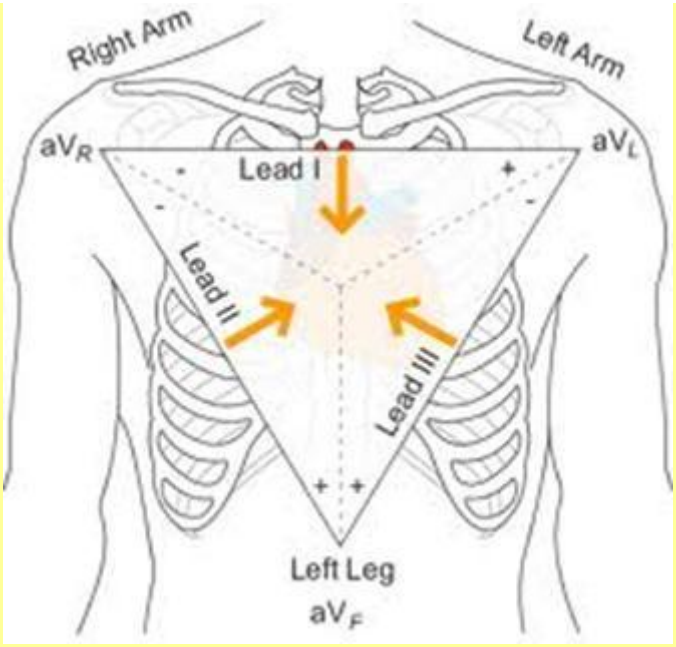
A typical ECG tracing of the cardiac cycle (heartbeat) consists of a P wave, a QRS complex, a T wave, and a U wave, which is normally visible in 50 to 75% of ECGs. The baseline voltage of the electrocardiogram is known as the isoelectric line. Typically, the isoelectric line is measured as the portion of the tracing following the T wave and preceding the next P wave.



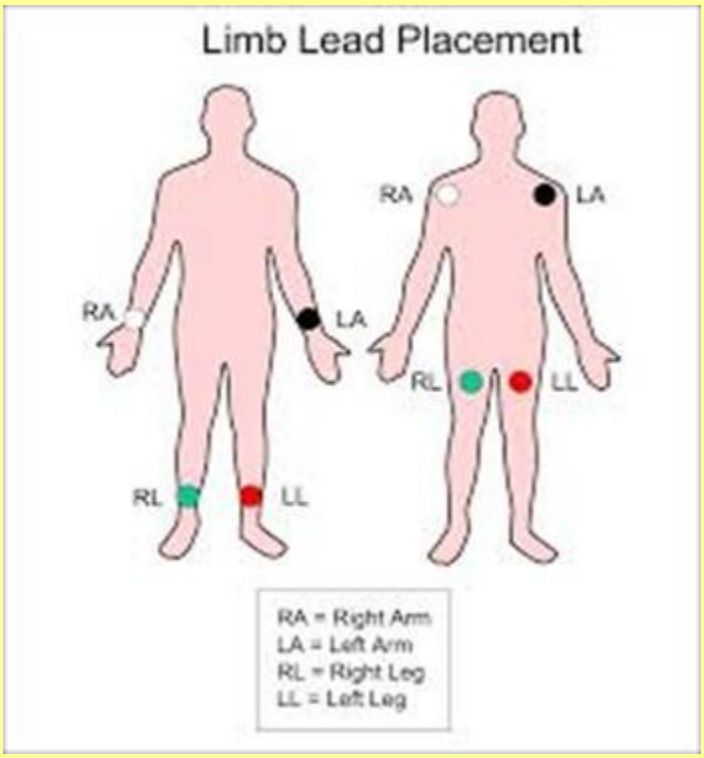
Feature	Description	Duration
RR interval	The interval between an R wave and the next R wave:Normal resting heart rate is between 60 and 100 bpm.	0.6 to 1.2s
P wave	During normal atrial depolarization, the main electrical vector is directed from the SA node towards the AV node, and spreads from the right atrium to the left atrium. This turns into the P wave on the ECG.	80ms
PR interval	The PR interval is measured from the beginning of the P wave to the beginning of the QRS complex. The PR interval reflects the time the electrical impulse takes to travel from the sinus node through the AV node and entering the ventricles. The PR interval is, therefore, a good estimate of AV node function.	120 to 200ms
PR segment	The PR segment connects the P wave and the QRS complex. The impulse vector is from the AV node to the bundle of His to the bundle branches and then to the Purkinje	50 to 120ms

	fibers. This electrical activity does not produce a contraction directly and is merely traveling down towards the ventricles, and this shows up flat on the ECG. The PR interval is more clinically relevant.	
QRS complex	The QRS complex reflects the rapid depolarization of the right and left ventricles. They have a large muscle mass compared to the atria, so the QRS complex usually has a much larger amplitude than the P-wave.	80 to 120ms
J-point	The point at which the QRS complex finishes and the ST segment begins, it is used to measure the degree of ST elevation or depression present.	N/A
ST segment	The ST segment connects the QRS complex and the T wave. The ST segment represents the period when the ventricles are depolarized. It is isoelectric.	80 to 120ms
T wave	The T wave represents the repolarization (or recovery) of the ventricles. The interval from the beginning of the QRS complex to the apex of the T wave is referred to as the absolute refractory period. The last half of the T wave is referred to as the relative refractory period (or vulnerable period).	160ms
ST interval	The ST interval is measured from the J point to the end of the T wave.	320ms
QT interval	The QT interval is measured from the beginning of the QRS complex to the end of the T wave. A prolonged QT interval is a risk factor for ventricular tachyarrhythmias and sudden death. It varies with heart rate and for clinical relevance requires a correction for this, giving the QTc.	Up to 420ms in heart rate of 60 bpm
U wave	The U wave is hypothesized to be caused by the repolarization of the interventricular septum. They normally have a low amplitude, and even more often completely absent. They always follow the T wave and also follow the same direction in amplitude. If they are too prominent, suspect hypokalemia, hypercalcemia or hyperthyroidism usually.	

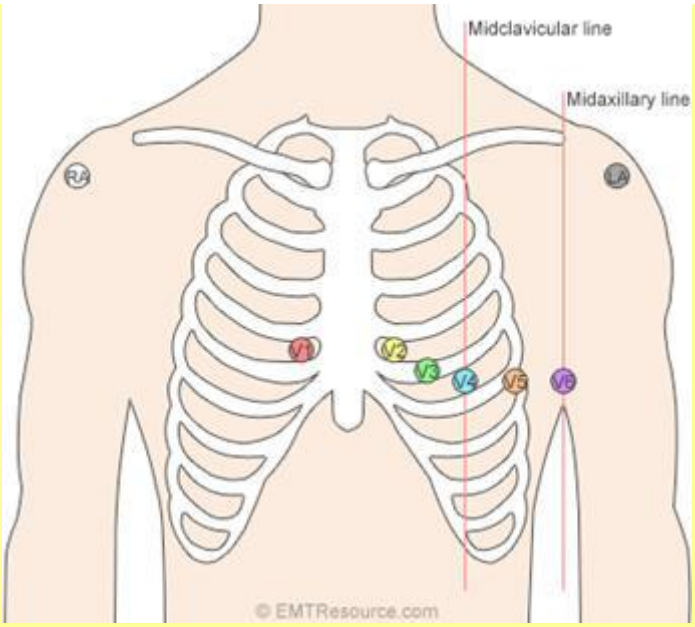
The placement of electrodes



Ten electrodes are used for a 12-lead ECG. The electrodes usually consist of a conducting gel, embedded in the middle of a self-adhesive pad onto which cables clip. Sometimes the gel also forms the adhesive. They are labeled and placed on the patient's body as follows:



The placement of the limb electrodes



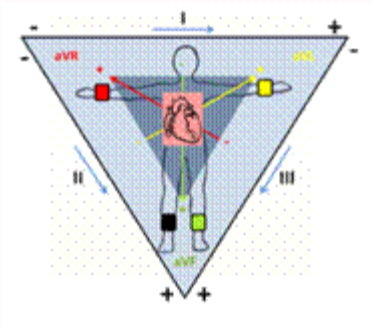
The placement of the chest electrodes

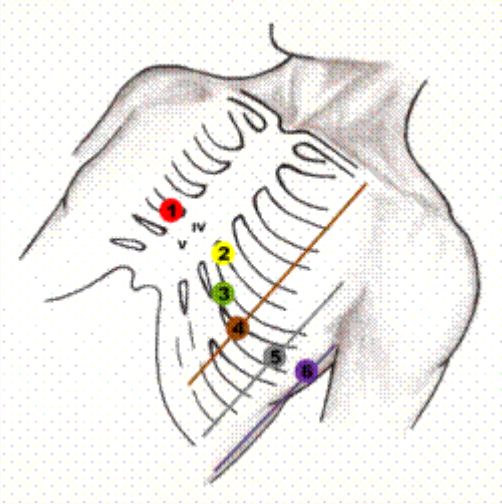
The 12-lead ECG is grouped into two electrical planes. The frontal leads (Lead I-III, aVR-F) view the heart from a vertical plane, while the transverse leads (V1-V6) view the heart from a horizontal plane.

Electrode	Placement
V1	4th Intercostal space to the right of the sternum
V2	4th Intercostal space to the left of the sternum
V3	Midway between V2 and V4
V4	5th Intercostal space at the midclavicular line
V5	Anterior axillary line at the same level as V4
V6	Midaxillary line at the same level as V4 and V5
RL	Anywhere above the ankle and below the

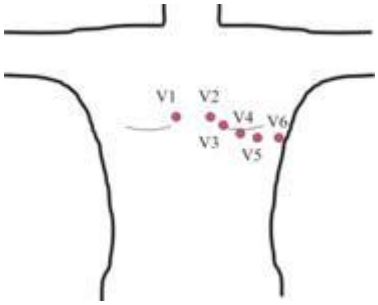
Electrode	Placement
	torso
RA	Anywhere between the shoulder and the elbow
LL	Anywhere above the ankle and below the torso
LA	Anywhere between the shoulder and the elbow

The placement of the chest electrodes

limb	**location of the lead**				
 bipolar extremity leads -Einthoven's leads-	I	+	left upper limb	-	right upper limb
	II		left lower limb		right upper limb
	III		left lower limb		left upper limb
unipolar extremity leads -Goldberg's leads-	aVR	right upper limb lead			
	aVL	left upper limb lead			
	aVF	left lower limb lead			
unipolar chest leads -Wilson's leads-	V1	fourth intercostal space, just to the right of the sternum			
	V2	fourth intercostal space, just to the left of the sternum			



V3	midway between V2 and V4, fifth intercostal space
V4	fifth intercostal space, midclavicular line on the left
V5	fifth intercostal space, anterior axillary line
V6	fifth intercostal space, midaxillary line



The description of the 12 ECG leads and the corresponding electrode positions is provided in the following:

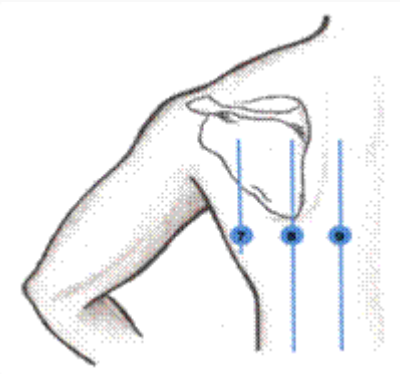
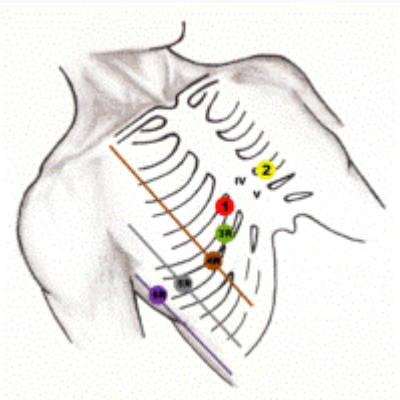
- I: is a lead obtained between a negative electrode placed on the right arm and a positive electrode placed on the left arm
- II: is a lead obtained between a negative electrode placed on the right arm and a positive electrode placed on the left foot
- III: is a lead obtained between a negative electrode placed on the left arm and a positive electrode placed on the left foot
- AVR: is a lead obtained between the average signal obtained from three negative electrodes (left arm, left leg and right foot) and the signal obtained from a positive electrode placed on the right arm
- AVL: is a lead obtained between the average signal obtained from three negative electrodes (right arm, left foot and right foot) and the signal obtained from a positive electrode placed on the left arm
- AVF: is a lead obtained between the average signal obtained from three negative electrodes (left arm, right arm and and right foot) and the signal obtained from a positive electrode placed on the left foot
- V1: is a lead obtained between the reference negative electrode and a positive electrode placed on the chest in the V1 position
- V2: is a lead obtained between the reference negative electrode and a positive electrode placed on the chest in the V2 position

- V3: is a lead obtained between the reference negative electrode and a positive electrode placed on the chest in the V3 position
- V4: is a lead obtained between the reference negative electrode and a positive electrode placed on the chest in the V4 position
- V5: is a lead obtained between the reference negative electrode and a positive electrode placed on the chest in the V5 position
- V6: is a lead obtained between the reference negative electrode and a positive electrode placed on the chest in the V6 position

Additional electrodes

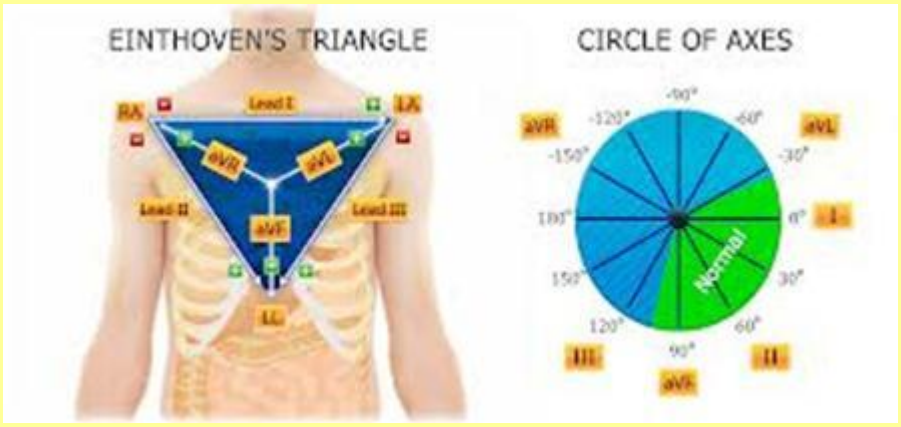
There are some leads, which are used in special situations, when conventional 12-leads ECG can not reliably show the myocardial defect.

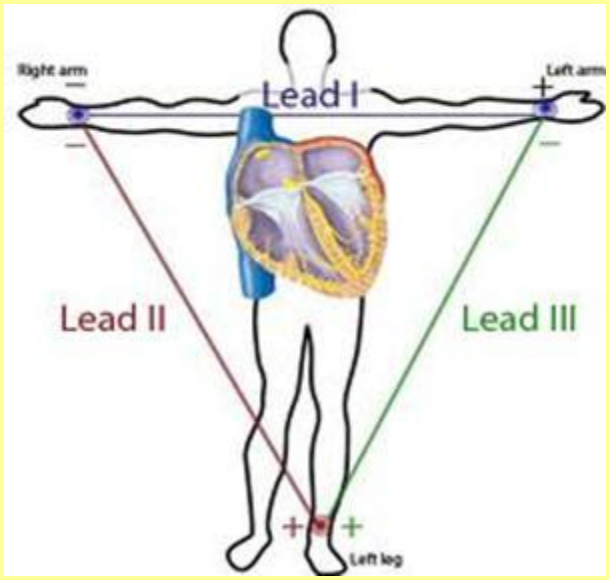
There are some examples of additional ECG leads

	lead	location of the lead	
unipolar chest leads	**V_7**	posterior axillary line, on the same level as V_6, on the left	
	V_8	scapulary line, in the same level as V_6, on the left	
	V_9	paravertebral line on the left, on the same level as V_6	
	VE	just to the left of *processus xiphoideus*	
	V3R - V6R	on the right, same location as V_3–V_6	
unipolar chest leads	**V_1' – V_6'**	about 1 intercostal space above the V_1–V_6	
	V_1'' – V_6''	about 2 intercostal space above the V_1–V_6	
esophageal leads	**E/Oe**	for example 37.5 cm (left atrium)	

Limb leads

Leads I, II and III are called ***limb leads***. The electrodes that form these signals are located on the limbs—one on each arm and one on the left leg. The limb leads form the points of what is known as Einthoven's triangle.





Einthoven's triangle

Lead I is the voltage between the (positive) left arm (LA) electrode and right arm (RA) electrode:

I lead: left arm (+) - right arm (-)

Lead II is the voltage between the (positive) left leg (LL) electrode and the right arm (RA) electrode:

II lead: left leg (+) - right arm (-)

Lead III is the voltage between the (positive) left leg (LL) electrode and the left arm (LA) electrode:

III lead: left arm (+) - left leg (-)

The two types of leads are unipolar and bipolar.

Bipolar leads have one positive and one negative pole. In a 12-lead ECG, the limb leads (I, II and III) are bipolar leads.

Unipolar leads also have two poles, as a voltage is measured; however, the negative pole is a composite pole (Wilson's central terminal, or WCT) made up of signals from lots of other electrodes. In a 12-lead ECG, all leads besides the limb leads are unipolar (aVR, aVL, aVF, V1, V2, V3, V4, V5, and V6).

Augmented limb leads

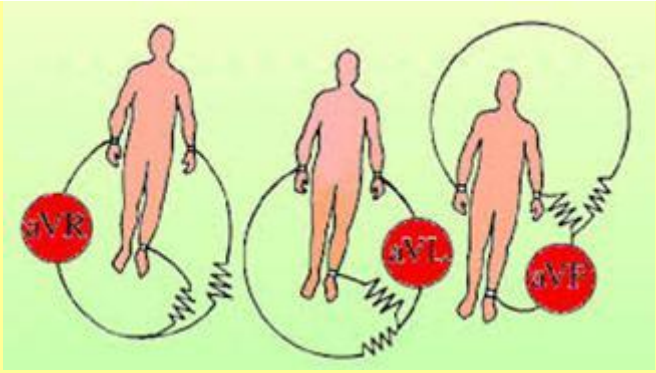
Leads aVR, aVL, and aVF are augmented limb leads (after their inventor Dr. Emanuel Goldberger known collectively as the Goldberger's leads). They are derived from the same three electrodes as leads I, II, and III. However, they view the heart from different angles (or vectors) because the negative electrode for these leads is a modification of Wilson's central terminal. This zeroes out the negative electrode and allows the positive electrode to become the "exploring electrode". This is possible because Einthoven's Law states that I + (−II) + III = 0. The equation can also be written I + III = II. It is written this way (instead of I − II + III = 0) because Einthoven reversed the polarity of lead II in Einthoven's triangle, possibly because he liked to view upright QRS complexes. Wilson's central terminal paved the way for the development of the augmented limb leads aVR, aVL, aVF and the precordial leads V1, V2, V3, V4, V5 and V6.

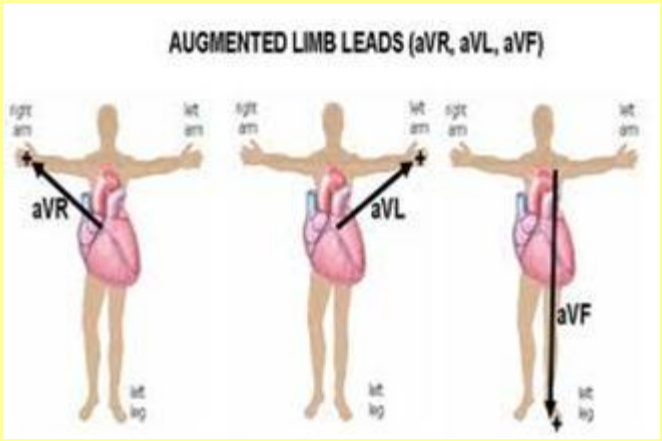
Lead augmented vector right (aVR)' has the positive electrode (white) on the right arm. The negative electrode is a combination of the left arm (black) electrode and the left leg (red) electrode, which "augments" the signal strength of the positive electrode on the right arm.

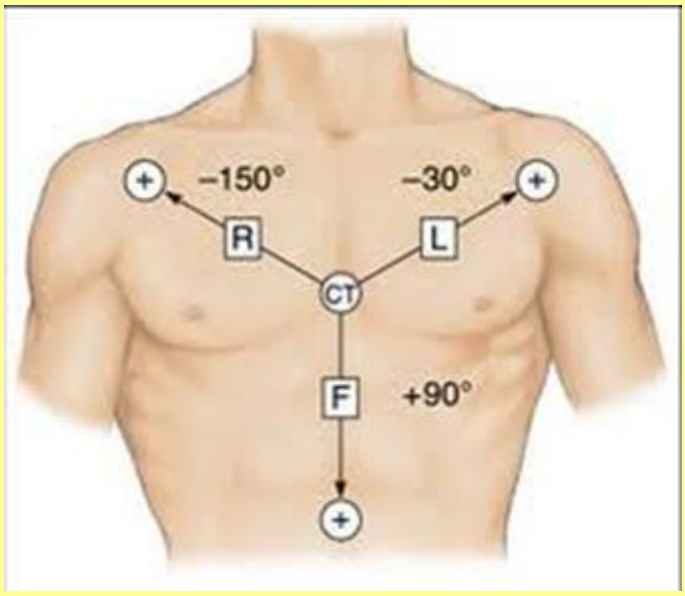
Lead augmented vector left (aVL) has the positive (black) electrode on the left arm. The negative electrode is a combination of the right arm (white) electrode and the left leg (red) electrode, which "augments" the signal strength of the positive electrode on the left arm.

Lead augmented vector foot (aVF) has the positive (red) electrode on the left leg. The negative electrode is a combination of the right arm (white) electrode and the left arm (black) electrode, which "augments" the signal of the positive electrode on the left leg.

The augmented limb leads aVR, aVL, and aVF are amplified in this way because the signal is too small to be useful when the negative electrode isWilson's central terminal. Together with leads I, II, and III, augmented limb leads aVR, aVL, and aVF form the basis of thehexaxial reference system, which is used to calculate the heart's electrical axis in the frontal plane.





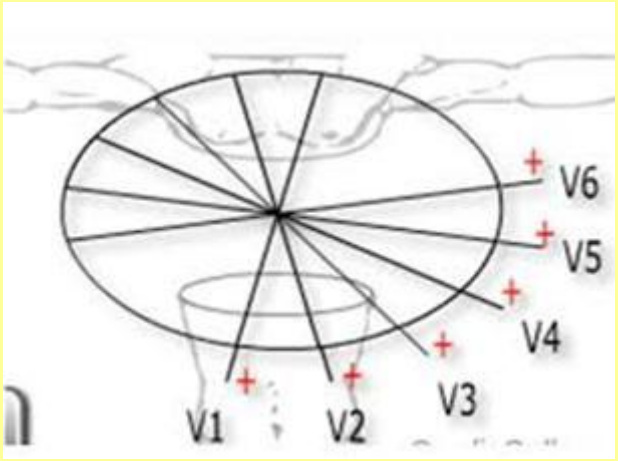


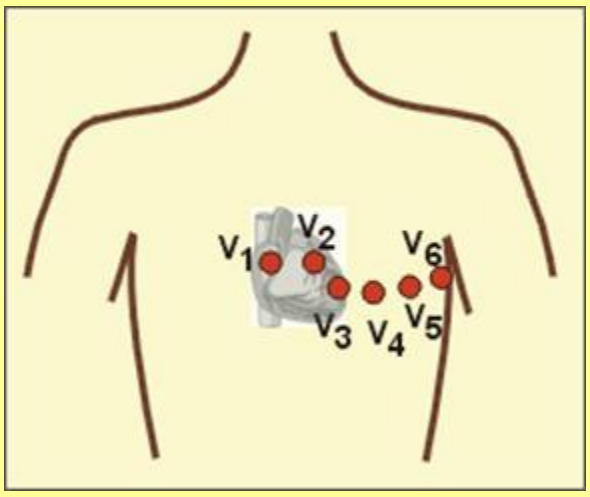
Augmented limb leads (aVR, aVL, aVF)

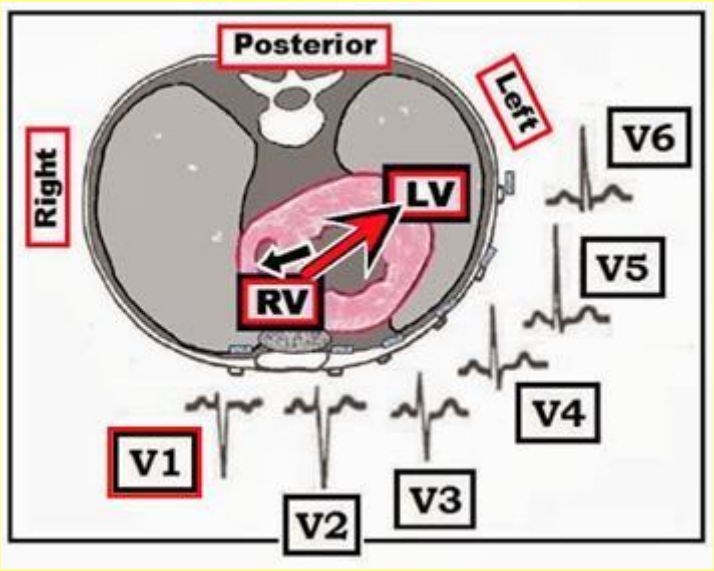
Precordial leads

The electrodes for the precordial leads (V1, V2, V3, V4, V5 and V6) are placed directly on the chest. Because of their close proximity to the heart, they do not require augmentation. Wilson's central terminal is used for the negative electrode, and these leads are considered to be unipolar (recall that Wilson's central terminal is the average of the three limb leads. This approximates common, or average, potential over the body). The precordial leads view the

heart's electrical activity in the so-called horizontal plane. The heart's electrical axis in the horizontal plane is referred to as the Z axis.



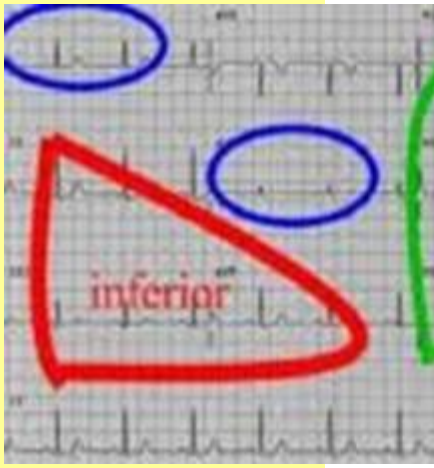




Of the 12 leads in total, each records the electrical activity of the heart from a different perspective, which also correlates to different anatomical areas

of the heart for the purpose of identifying acute coronary ischemia or injury. Two leads that look at neighbouring anatomical areas of the heart are said to be contiguous. The relevance of this is in determining whether an abnormality on the ECG is likely to represent true disease or a spurious finding.

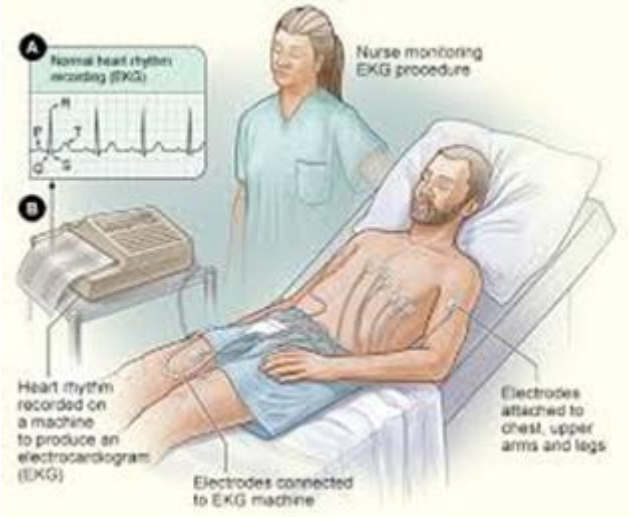
Lead	(-) Electrode	(+) Electrode	View of Heart
Lead I	RA	LA	Lateral
Lead II	RA	LL	Inferior
Lead II	LA	LL	Inferior
aVR	LA + LL	RA	None
aVL	RA + LL	LA	Lateral
aVF	RA + LA	LL	Inferior



I Lateral	aVR	V1 Septal	V4 Anterior
II Inferior	aVL Lateral	V2 Septal	V5 Lateral
III Inferior	aVF Inferior	V3 Anterior	V6 Lateral

Diagram showing the contiguous leads in the same colour

Algorithm of ECG registration

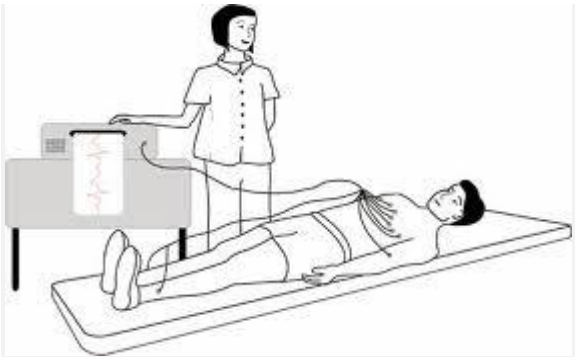


1. For this station you will need:

- Hand sanitiser
- ECG machine
- 10 sticky pads for attaching the electrodes to
- Occasionally need wipes and/or razor

2. Wash your hands and introduce yourself to the patient and clarify their identity.

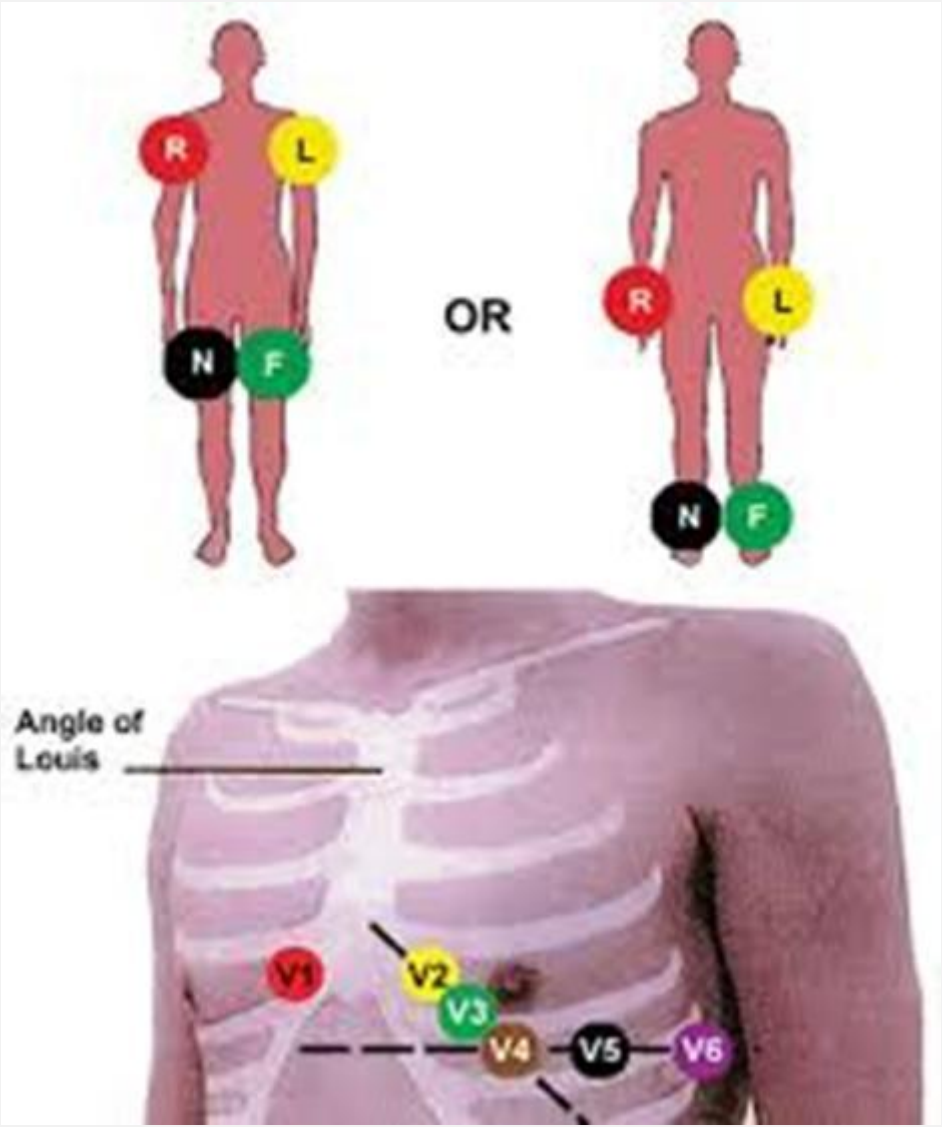
4. Explain what you are going to do and gain consent to proceed. Clarify whether the patient knows why the test is being performed e.g. chest pain.
5. Ensure the patient is comfortable.



6. For the ECG pads to stick there must be good contact with the skin, therefore ensure there is nothing which could prevent this e.g. shave hair or wipe off any cream/lotion.

7. Input the patients data into the machine correctly, thus ensuring the correct details are printed onto the ECG.

7. Although this is called a 12 lead ECG you only need 10 sticky pads, 4 for the limb leads and 6 for chest leads. This is where you place them:



- Right wrist (or shoulder)
- Left wrist (or shoulder)
- Right ankle, laterally

- Left ankle, laterally
- V1 in 4th intercostal space at right sternal edge
- V2 in 4th intercostal space at left sternal edge
- V3 between V2 and V4
- V4 in 5th intercostal space in the mid-clavicular line
- V5 horizontal to V4 in anterior axillary line
- V6 horizontal to V4 and V5 in mid-axillary line

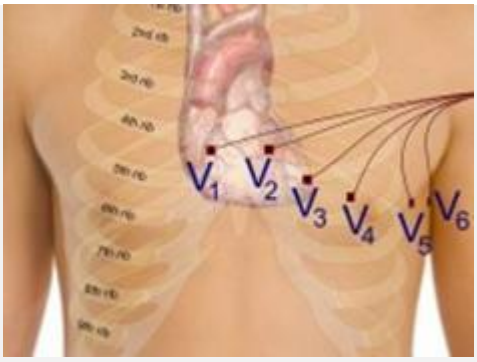
8. Attach the leads to the pads.

The limb leads are coloured: red, yellow, green and black. One way to remember is the order of traffic lights:

- red (R wrist)
- yellow (L wrist)
- green (L ankle)
- black (R ankle)

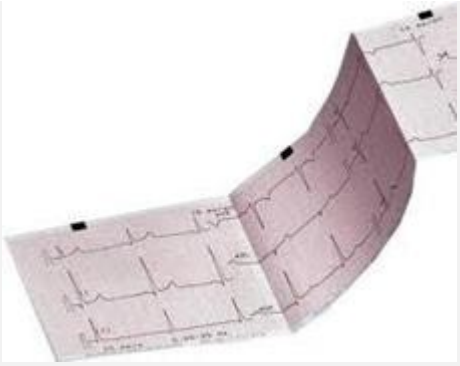
9. Note that these colours are used within Europe. America uses a different colour scheme

The chest leads are usually numbered V1-6, attach as shown:



Precordial leads in ECG

10. Once all the leads are attached ask the patient to lie as still as possible. Record the reading and print a copy. If the patient had any chest pain at the time of recording you should record this on the ECG.



11. Disconnect the leads from the pads and allow the patient to remove the pads themselves, or offer assistance if needed. Offer a tissue as the pads are sticky.

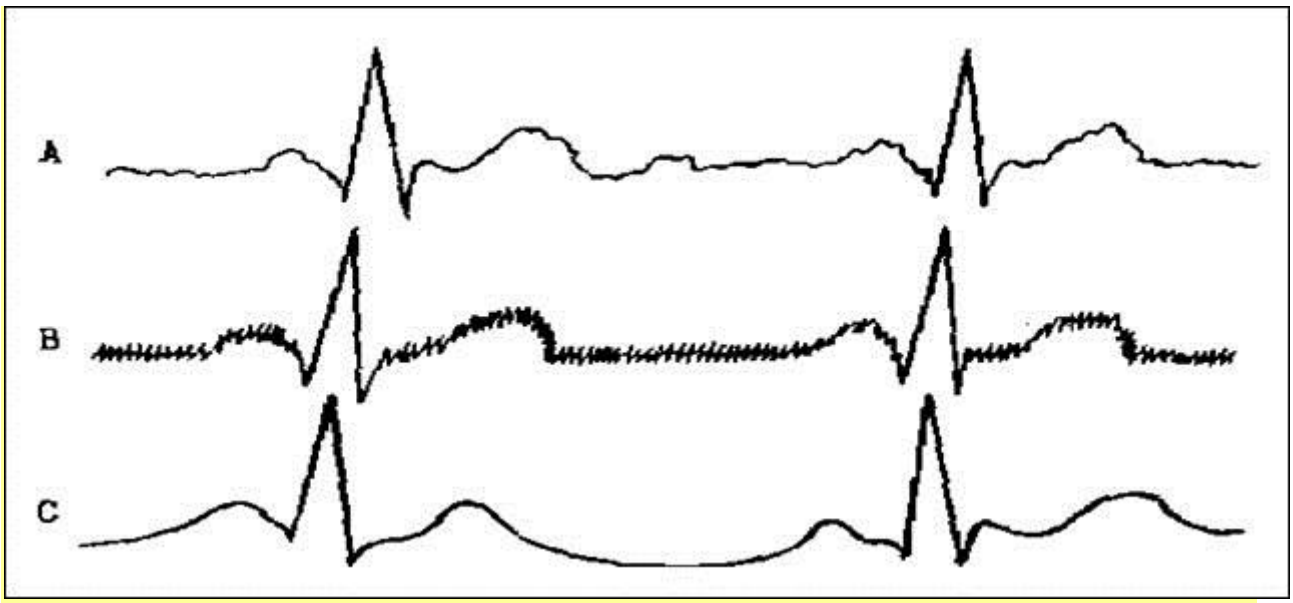
12. Allow the patient to dress and thank them.

An extension to this station could be ECG interpretation; you should therefore be familiar with the most common ECG findings.

Registration performs fare from electric motors and other electrical devices.

Tested person may have rest before registration in 10-15 minutes. This procedure needs 2-hour interval after eating or worm procedures.

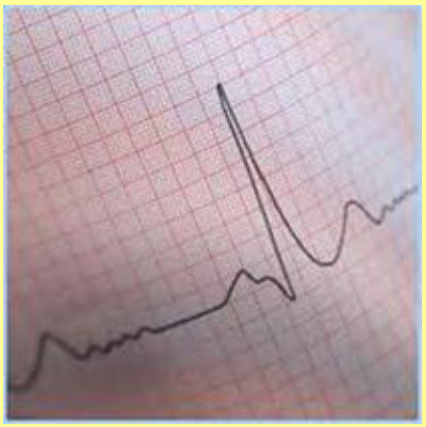
For better contact between electrodes and skin use solution NaCl 5-10 % or special electrode past or electrode gel. Otherwise hindrances in ECG curve may occur. They will stand in the way of ECG analysis:



ECG registration performs in quiet breathing in patients.

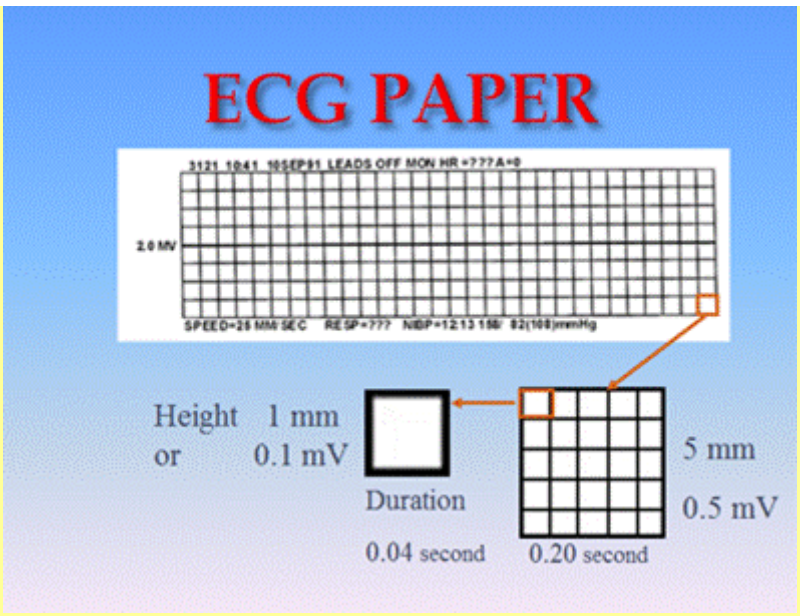
Registration begins from standard voltage 1 mV from the electrocardiograph for regulation of amplitude in ECG. Usually standard voltage amplitude is 10 mm. Then continue registration of bipolar limb leads, the next - unipolar limb leads and afterwards - unipolar chest leads.

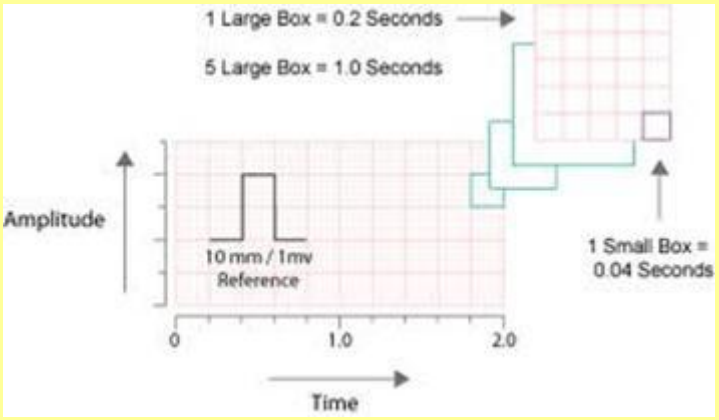
An Approach to ECG analysis



Step 1. ECG analysis begins with estimation of control voltage and paper speed.

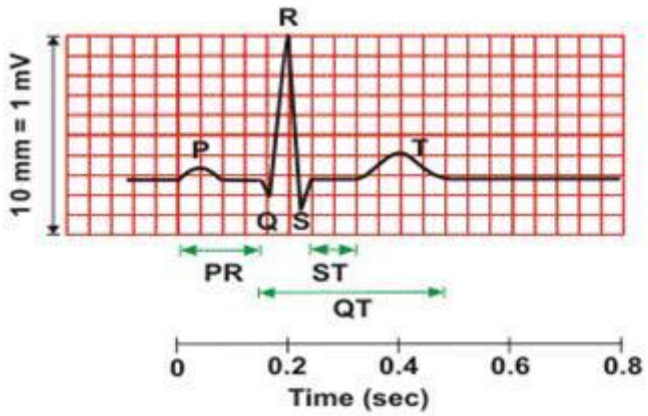
1) The ECG is recorded at a speed of 25 mm/sec, and the voltages are calibrated so that 1 mV = 10 mm in the vertical direction. Therefore, each small 1-mm square represents 0.04 sec (40 msec) in time and 0.1 mV in voltage. Since a large block is five small blocks wide and five high, each large block represents 0.20 sec (horisontal) and 5 mm (vertical).





2) The ECG is recorded at a speed of 50 mm/sec, and the voltages are calibrated so that 1 mV = 10 mm in the vertical direction. Therefore, each small 1-mm square represents 0.02 sec (20 msec) in time and 0.1 mV in voltage. Since a large block is five small blocks wide and five high, each large block represents 0.10 sec (horisontal) and 5 mm (vertical).

Because the recording speed is standardized, one can calculate the heart rate from the intervals between different waves.



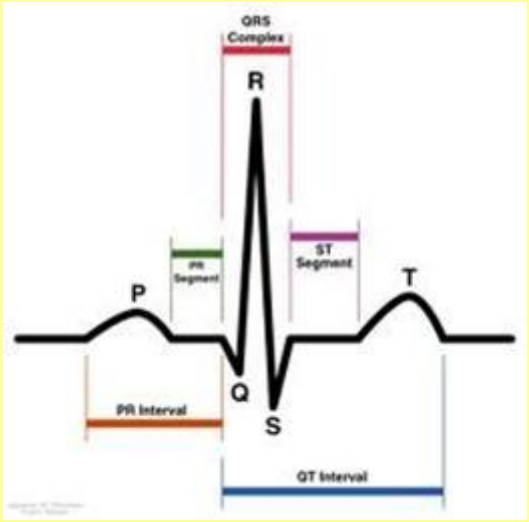
P wave (0.08 - 0.10 s) QRS (0.06 - 0.10 s)

P-R interval (0.12 - 0.20 s) Q-T_C interval (≤ 0.44 s)*

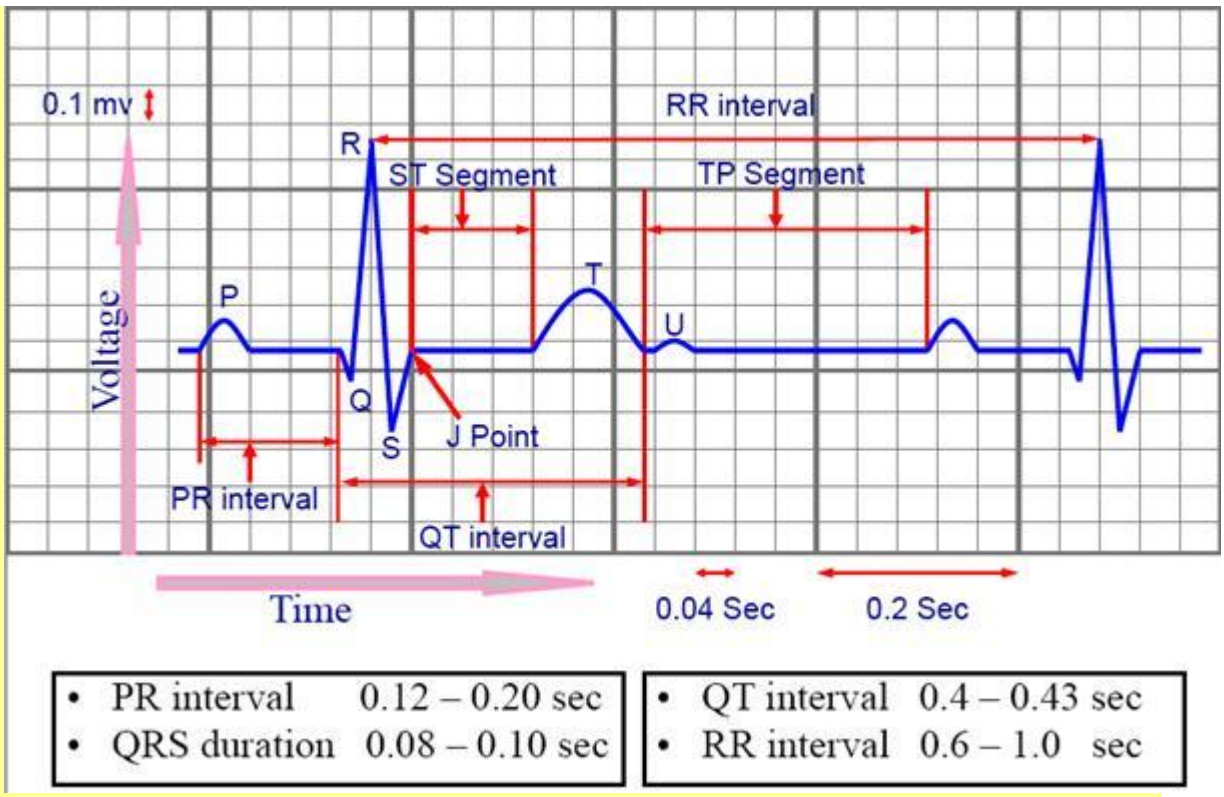
*$QT_C = QT/\sqrt{RR}$

Step 2.

Now that we know these basic measurements and are familiar with the relation on the ECG waves to the heart anatomy, let's discuss the significance of each wave and interval:



Electrocardiogram waves, intervals and segments



The main elements of ECG curve are:

- Waves P, Q, R, S and T. Sometimes U wave may occur;
- Segments - P-Q (from the end of P wave to beginning of Q wave), S-T (from the end of S wave until beginning of T wave);
- Intervals, which characterize certain time period of heart activity - P-Q (from the beginning of P wave to beginning of Q wave), Q-T (from beginning of Q wave to end of T wave);
- Complexes - atrial, which is presented by P wave, and ventricular - QRST.

P wave in healthy persons, is obligatory positive in I, II, AVF, V_2-V_6 leads. P wave may be negative in III, AVL and V1, either positive or biphasic. If it is diphasic, then the negative component comes after the positive component and is not excessively broad or deep. An absent P wave in the ECG may signify sinoatrial block, an abnormality in which the impulse from the SA node is not conducted to the AV node.

Normally in II lead its amplitude is 2,5 mm, duration – 0,1 s (not greater than 110 ms).

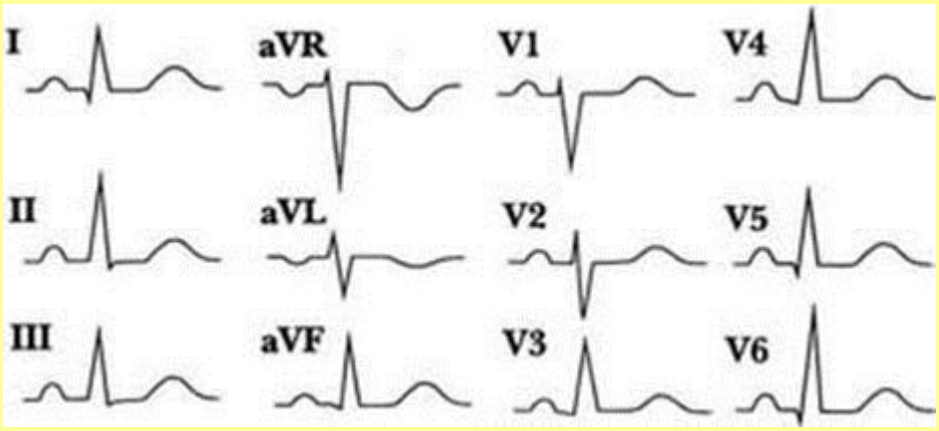
The P wave is caused by atrial depolarization. The normal shape of the P wave does not include any notches or peaks.

P-Q interval reflects duration of AV-conduction, which is spreading of potential by AV node, His bundle and its branches. This interval lasts 0.12-0.20 s and depends on heartbeat rate.

QRST complex reflects spreading of excitation by ventricles. It hole amplitude is higher 5 mm of the waves are signed by capital letters. Otherwise it used little letters. ORS duration in II lead is not more than 0.1 s.

Q wave normally in II lead is less then 1/4 of R amplitude duration is 0.03 s. Normally in AVR deep and wide Q waves may be recorded. In V_1, V_2- Q wave is particularly absent.

R-view usually is recorded in all leads; exalt AVR, which may be absent. In unipolar chest leads R amplitude gradually increases from V_1 to V_4 and some decreases in V5 and V6. So normally in unipolar chest leads both increasing R-amplitude and S-amplitude occurs. S-wave has amplitude not more than 20 mm, but it varies from lead to lead.



S-T – segment corresponds to excitation of both ventricles. Normally in bipolar and unipolar leads it lies on baseline and don't move more than 0.5 mm. In V1-V3 deviation upward to 2 mm may occur.

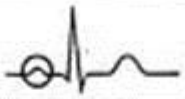
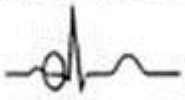
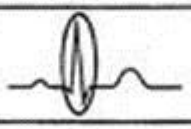
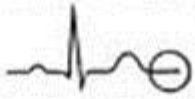
T-wave normally is positive in I, II AVF, V_2-V_6, $T_I > T_{III}$, $T_{V6} > T_{V1}$. T-wave has sloping ascend part and sleep descending part. In III, AVL, V_1 T-wave may either be positive, negative or bipolar. In II lead T-amplitude is 5-6 mm, duration – 0.16-0.24 s.

Q-T interval is electrical systole of ventricles. Its duration directly depends on heartbeat rate. Proper duration may calculated by Buzett formula:h0

Q-T=K$\sqrt{\text{R-R}}$, where

K=0.37 in male or 0,40 in female

U-wave may be recorded in unipolar chest leads, which reflects excitation fare of excitability after electrical systole of ventricles myocardium. U-wave usually is positive and small.

Electrical Activity	Graphic Depiction	Associated Pattern
Atrial Depolarization		P Wave
Delay at AV Node		PR Segment
Ventricular Depolarization		QRS Complex
Ventricular Repolarization		T Wave
No electrical activity		Isoelectric Line

How to read an ECG

ECG analysis begins with estimation of control voltage and paper speed. Another analysis at usual performs in this order.

1) Determining of impulse origin. Pay attention to proper order of waves in ECG. If P wave in II lead is positive and recorded before QRS complex is believed to determine pacemaker in SA node.

2) Heart rhythm evaluation by measuring of R-R duration. Normally adjacent R-R intervals duration may differ from each other not more 0.1 s. Usually II lead is examined.

3) Determining of heart rate. In proper rhythm 60 s is divided to R-R duration in seconds, which is calculated using paper speed.

Knowing the paper speed, it's easy to work out heart rate. It's also very convenient to have a quick way of eyeballing the rate, and one method is as follows:

1. Remember the sequence: 300, 150, 100, 75, 60, 50 (if paper speed is 25 mm/min!)
2. Identify an R wave that falls on the marker of a `big block'
3. Count the number of big blocks to the next R wave.

If the number of big blocks is 1, the rate is 300, if it's two, then the rate is 150, and so on. Rates in between these numbers are easy to interpolate.

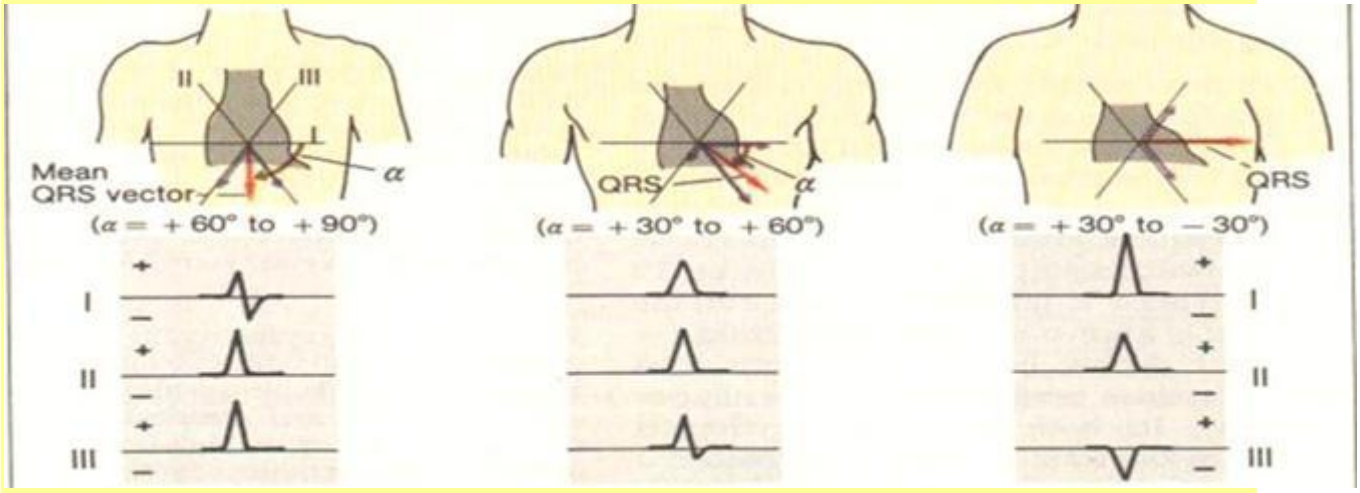
But always remember that in the heart, because we have two electrically `isolated' chambers, the atria and ventricles, that we are really looking at two rates - the atrial and ventricular rates! It just so happens that in the normal heart, the two are linked in a convenient 1:1 ratio, via normal conduction down the AV node. In disease states, this may not be the case.

Conventionally, a normal heart rate has been regarded as being between 60 and 100, but it's probably more appropriate to re-adjust these limits to 50 -- 90/min. A sinus tachycardia then becomes any heart rate over 90, and bradycardia, less than 50. Note that you have to look at the clinical context -- a rate of 85 in a highly trained athlete may represent a substantial tachycardia, especially if their resting rate is 52/minute! One should also beware of agressively trying to manage low rates in the presence of good perfusion and excellent organ function.

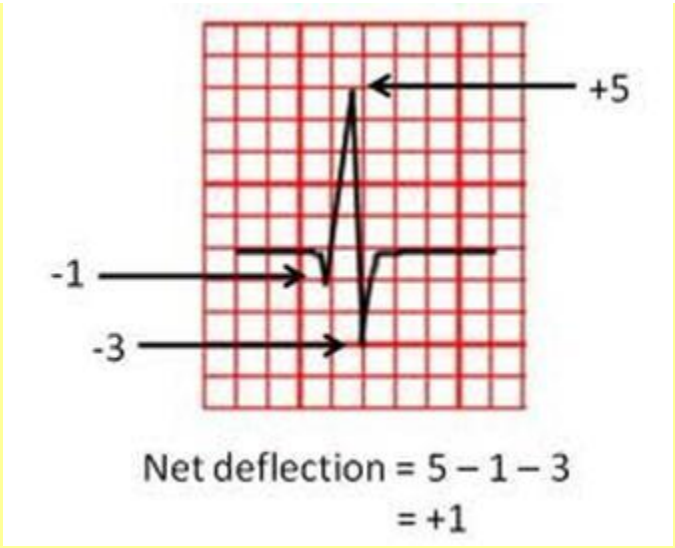
4) Evaluation of ECG voltage. If in bipolar limb leads the lowest R wave is smaller than 5 mm and RI+RII+RIII less than 15 mm, the ECG voltage is decreased. Otherwise it is normal.

5) EMP direction determining.

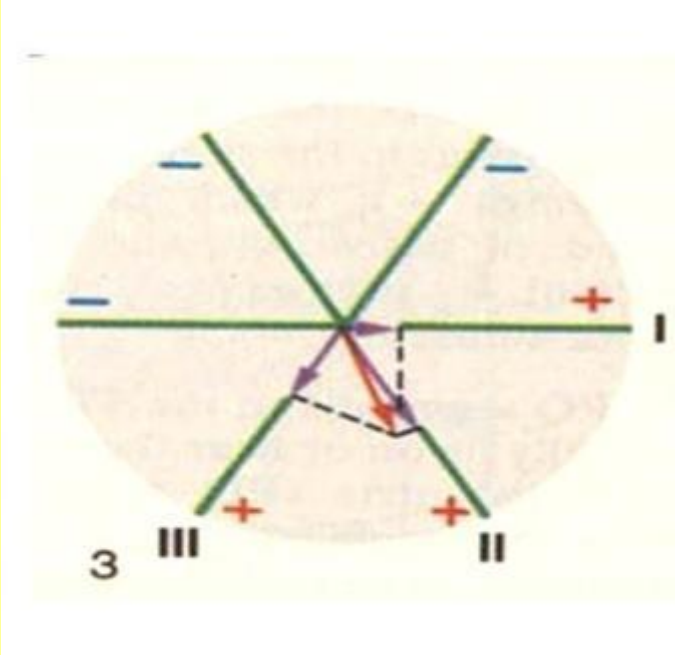
- Visual method: needs measuring R amplitude in all bipolar limb leads. If true, that RII>RI>RIII, the EMP direction is near 30º-69º, that is normal;

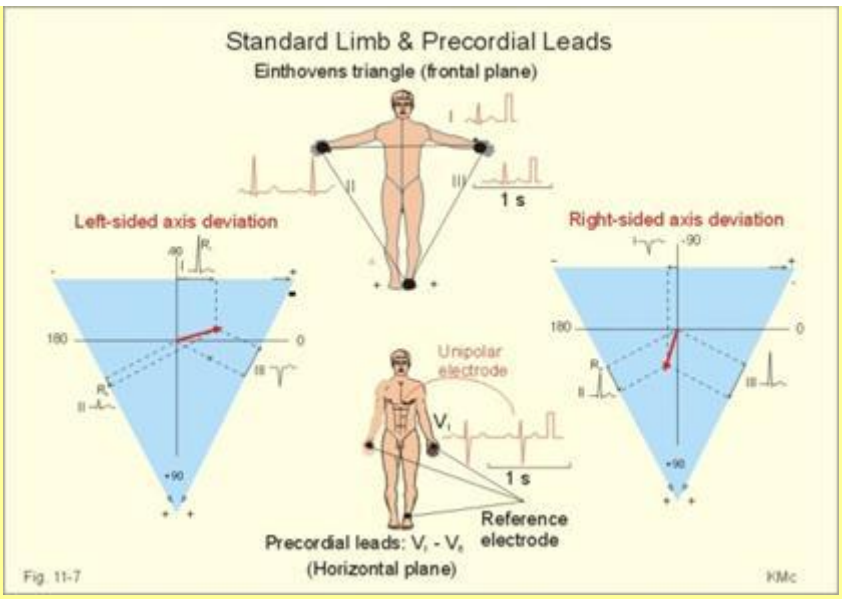


- Graphic method use Baily co-ordinate. If in Einthoven's triangle put through the center parallel to leads axes we'll get Baily's co-ordinate. Than in any two bipolar limbs leads it is necessary to determine summary amplitude of QRS waves. Upward waves have positive meaning and downward are negative.



Summary amplitude put on corresponding axis with (+) or (-) sign. In this point lined perpendicular to lead axis. Next time determined cross point of two drown perpendiculars. When join this point to Baily's co-ordinate center we'll obtain the EMP direction outward the center.





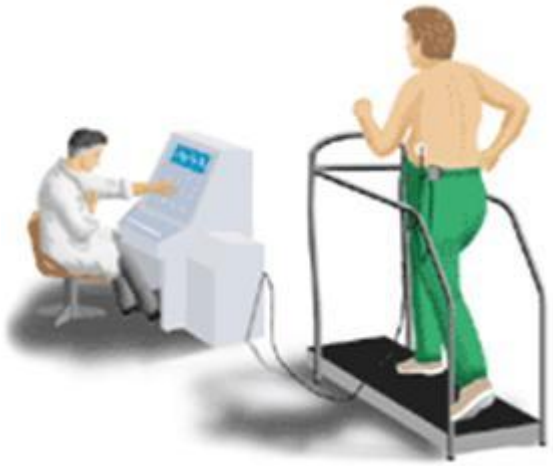
6) ECG elements analysis. Pay attention to form, amplitude and duration of waves and intervals. Measure deviation from baseline if it occurs. Compare the results with normal rate.

There are ***additional ECG procedures*** which are more involved than the basic ECG.

These procedures include the following:

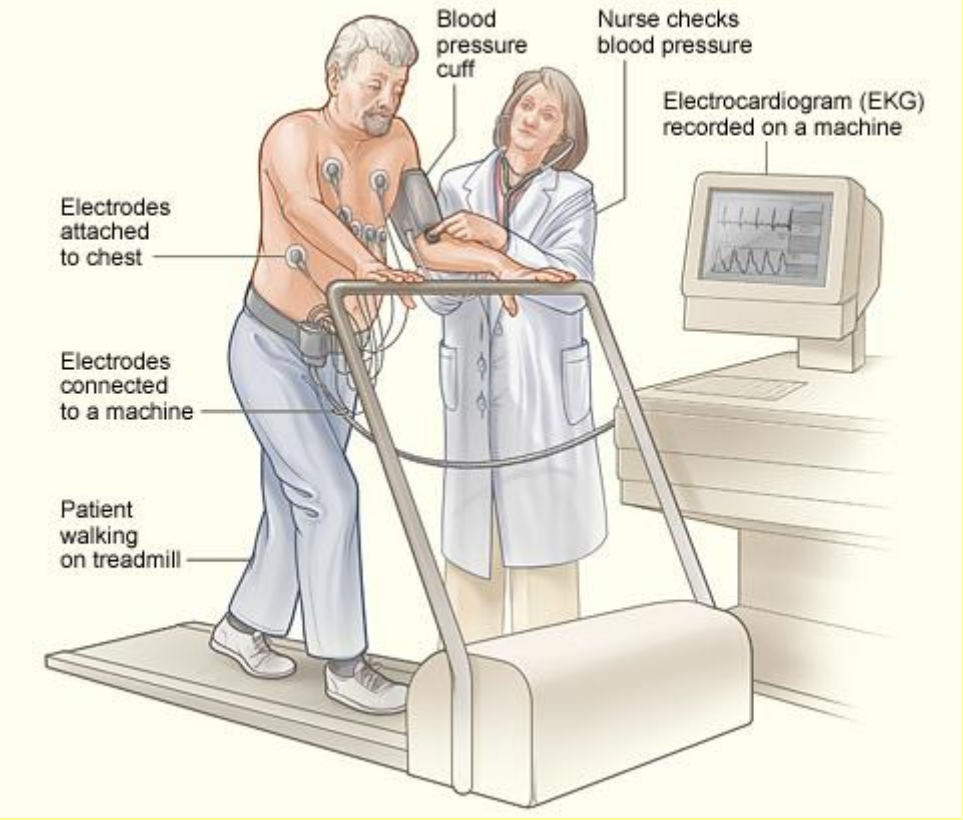
Exercise ECG, Or Stress Test

The patient is attached to the ECG machine. The patient exercises by walking on a treadmill



or pedaling a stationary bicycle while the ECG is recorded. This test is done to assess changes in the ECG during stress such as exercise.

The level of mechanical stress is progressively increased by adjusting the difficulty (steepness of the slope) and speed. The test administrator or attending physician examines the symptoms and blood pressure response. With use of ECG, the test is most commonly called a cardiac stress test, but is known by other names, such as exercise testing, stress testing treadmills, exercise tolerance test, stress test or stress test ECG.



The illustration shows a patient having a stress test. Electrodes are attached to the patient's chest and connected to an EKG (electrocardiogram) machine. The EKG records the heart's electrical activity. A blood pressure cuff is used to record the patient's blood pressure while he walks on a treadmill.

An electrocardiogram (ECG) stress test monitors a person's heartbeat at rest and during exercise, most commonly while a person walks on a treadmill. A physician observes the person, monitors the exercise level, and makes recordings until the person's heart nears a maximum predicted heart rate. The heart also is monitored during the period of cool-down or recovery that immediately follows exercise. The recordings made before, during, and immediately after an ECG stress test can show subtle changes in heart electrical activity that can help a physician:

-Determine physical fitness;

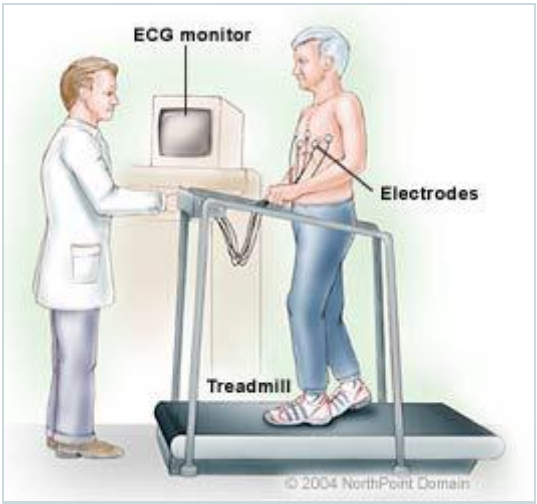
-Locate areas of the heart that receive an insufficient blood and oxygen supply;

-Reveal heart rhythm abnormalities;

-Evaluate a person's prognosis after a heart attack;

-Verify the effectiveness of medical and surgical therapies; and

-Determine an appropriate exercise program for people with known heart disease.



A typical ECG stress test setup.

PRE-TEST GUIDELINES

The physician will provide written instructions before the test, which the patient should follow closely. Typically it is recommended that people wear clothing that is comfortable to exercise in and avoid strenuous exercise and eating or drinking, and using tobacco products for approximately two hours before the test. The testing center may also request that the person refrain from using tobacco, certain prescription and nonprescription drugs, and alcohol or caffeine for 24 hours before the test.

RISK FACTORS

ECG stress testing is painless, has little risk, and does not use radiation.

WHAT TO EXPECT

In the testing room, approximately 10 electrodes are attached to the person's chest and back. The electrodes record the heart's electrical impulses and transmit them to an electrocardiograph, a machine that turns the impulses into waves on graph paper or on a monitor screen. Time is recorded horizontally, and the duration and intensity of the heart's electrical impulses is recorded vertically. People will also wear a blood pressure monitoring cuff or monitoring device during the test.

The electrodes may be placed on top of a light layer of gel that conducts electricity and helps ensure clear readings. Men should expect to have some chest hair shaved so that the electrodes will press directly against the skin.

Recordings of the heart's activity are made before exercise begins, first while the person is lying down and then again when the person stands up. These

readings can be used to make sure the person is ready for the test and to compare to the stress test with the resting heartbeat.

As exercise begins, the patient walks slowly on the treadmill. The speed and incline of the treadmill increase slowly, typically about every 3 minutes. The test ends once the person reaches the target heart rate (usually 220 beats per minute minus the person's age), or if he or she cannot continue because of fatigue, shortness of breath, or chest pain. The physician evaluates the test results and discusses them with the patient after the test.

Medication-induced stress ECG is available for people who cannot exercise. Medications that stress the heart temporarily for testing include:

- Dipyridamole (unless the patient has asthma);
- Adenosine; and
- Dobutamine.

Dipyridamole and adenosine may cause brief side effects, including:

- Headache;
- Dizziness;
- Nausea; and
- Flushing.

One disadvantage to ECG stress testing is that recordings can appear normal despite the presence of some heart dysfunction, which is called a false-negative result, or show an abnormality when there isn't one, called a false-positive result. Stress ECGs produce a high rate of false-positive results in women. In some cases, additional testing may be required.

POST-TEST GUIDELINES

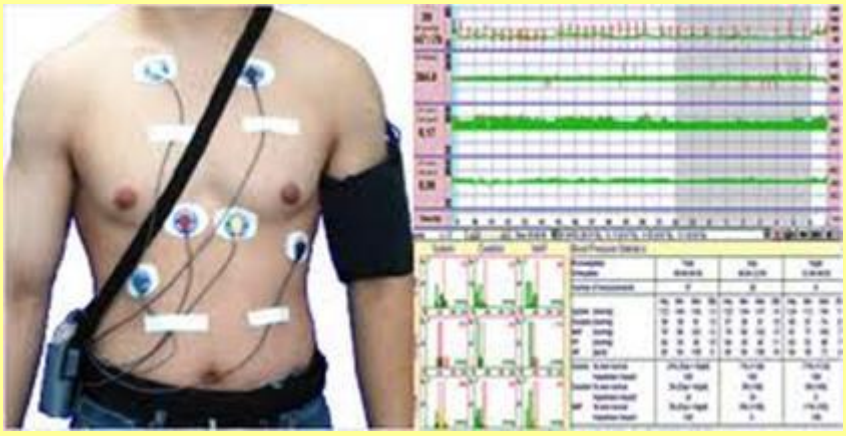
Most patients resume usual activities immediately following ECG stress testing.

Holter monitor

Holter monitoring is a form of long-term ECG recording. It is a diagnostic procedure that provides a continuous record of electrical activity of a patient's heart while the individual is engaged in ordinary activities, including sleep. Holter monitoring is used to detect abnormalities related to rhythm, rate, conduction and ischemia, which are not observed using a standard ECG.

Basic components of Holter monitoring systems are a sensing element, an appropriate recording of ECG information or significant variations in rate or arrhythmia, and a component for graphically recording ECG data or for visual or computer assisted analyses of recorded taped information.

Ambulatory electrocardiography (AECG) is used to detect, characterise and document cardiac arrhythmias in clinical practice. As some arrhythmias are infrequent or may occur only during certain activities (eg, sleep or exercise), it is usual to record the electrical activity of the heart over a period of time, usually 24 or 48 hours.



Intermittent recorders may also be used to provide brief records of recordings from a longer period of time. These recorders may have a memory loop to allow documentation of sudden change in rate or rhythm of the heart. Most modern pacemakers and implantable defibrillators can also be used to gather information about arrhythmias for retrieval.

Ambulatory ECG monitoring is suitable for patients with symptoms which may be caused by arrhythmia (eg, palpitations, light-headedness or syncope):

-Patients should be able to record symptoms in a diary.

-Patients with symptoms occurring daily or almost daily, or those who have syncope without warning, should be evaluated with a 24-hour Holter monitor.

-Patients with symptoms occurring less frequently may be better evaluated using a patient-activated event recorder.

Ambulatory electrocardiography equipment

The most commonly used method of extended ECG recording is a Holter monitor which uses a conventional tape recorder or solid-state storage system for acquiring ECG information that can then be reviewed. There are two commonly used types of AECG recorders:

- Continuous recorders:

These recorders are typically used for 24 or 48 hours to record events which might reasonably be expected to occur within that timeframe, ie frequent, or at least once a day symptoms. The patient keeps a diary of symptoms and records the time on the Holter clock when the symptoms occur, for later correlation with ECG abnormalities. The ECG recording is in digital format which allows for accurate and speedy interpretation of the recording, some recorders even providing for 'online' analysis as required. Their use is limited by cost, and reliance on computer software to analyse the results accurately (former limited storage capacity of digital data is rapidly being overcome).

- Intermittent recorders:

These are generally for recording infrequent symptoms, and are one of two types:

- Event recorders, which store only a brief recording of ECG activity when activated by the patient in response to symptoms.
- Loop recorders, which record the ECG in a continuous fashion, but store only a brief record when activated by the patient.

Both types of intermittent recorder may be worn by patients for periods of many weeks in order to capture infrequently occurring events. Newer loop recorders continuously record and erase so that data gathered from 1 to 4 minutes before and then 30 to 60 seconds after the device was activated can be retained. Recordings may often be transmitted via telephone/3G mobile/internet to a central point of analysis.

Indications for ambulatory electrocardiography

AECG may be used to assess patients in whom an arrhythmia is suspected, including:

- *Patients with syncope, near syncope or dizziness.
- *Patients with palpitations.
- *Patients who have had a cerebrovascular accident in whom paroxysmal atrial fibrillation (AF) or atrial flutter is suspected.
- *Continuous ECG monitoring of AF is useful to detect silent paroxysmal AF in patients without previously documented arrhythmic episodes, such as those with cryptogenic stroke.

- *Early diagnosis enables earlier treatment for primary or secondary stroke prevention.
- *Patients with episodic chest pain, shortness of breath or fatigue with no other obvious cause.
- AECG may be used to assess the potential risk of developing an arrhythmia - for example:
- *Patients prior to discharge from hospital after a myocardial infarction.
- *Patients with congestive heart failure.
- *Patients with hypertrophic cardiomyopathy.
- AECG may be used to assess a patient's response to anti-arrhythmic treatment - eg, the rate of AF, or pro-arrhythmic responses to drugs.
- AECG may be used to assess the function of a pacemaker device or implantable cardioversion device.
- The newer AECG monitors (incorporating multichannels, flash cards, etc) may also be used as a tool for the detection of myocardial ischaemia, by measurement of S-T segment shifts - for example:
- *Patients with suspected variant angina.
- *In the evaluation of patients with chest pain, who are unable to exercise.
- *In preoperative assessment for vascular surgery in patients who are unable to exercise.

Limitations of Holter recordings

- The sampling period is usually too short to allow capture of an infrequent arrhythmia.
- Holter monitors detect arrhythmias, which are responsible for symptoms, only 10% of the time.
- Although the period of observation could be extended, serial Holter monitor recordings are impractical and expensive.
- Observation of patients on a telemetry unit in the hospital also has severe limitations, especially poor patient acceptance.

Mobile cardiac outpatient telemetry

- This allows several days of ECG monitoring via a cellular-based transmission system.

- Mobile cardiac outpatient telemetry (MCOT) provided a significantly higher yield than standard cardiac loop recorders in patients with symptoms suggestive of a significant cardiac arrhythmia.
- MCOT can detect asymptomatic clinically significant arrhythmias, and is particularly useful to identify the cause of presyncope or syncope, even in patients with previously negative investigations.