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# Diagnosis of acute anterior coronary infarction by use of limb leads

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# THE DIAGNOSIS OF ACUTE ANTERIOR CORONARY INFARCTION BY USE OF LIMB LEADS

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## Introduction

Electrocardiography has given the clinicians the confidence of knowledge in diagnosis. It has made prognosis more exact. It has rationalized therapy. Although the cardiovascular examination is incomplete without the aid of an electrocardiogram, an attempt to extract more information than it is able to furnish often leads to confusion. The purpose of this paper therefore is to give to the busy practitioner a practical understanding and working knowledge of the basic mechanisms involved, both in recording and interpreting the electrocardiograms of acute anterior coronary infarction, because it is in this capacity that electrocardiography has reached its prime usefullness.[23]

In order to impart to the practitioner this knowledge of interpretation, which will serve him not only in myocardial infarctions, but also in other disorders of the heart, herein is included a summary of the physiologic basis of electrocardiography, expressing the most popular theory of polarization and depolarization as are used in modern electrocardiography. In order to limit the size of this article, however, interpretation is restricted to the use of the three standard limb leads. It is

well recognized that the chest leads are a valuable adjunct in evaluation of cardiac disorders; in fact, almost indispensable. However, the three standard leads remain as the main criteria in diagnosis of acute myocardial infarction.

## The Instrument

Since 1903, when Einthoven introduced the string galvanometer, electrocardiographs have been improved and simplified. The string galvanometer, however, is still one of the most widely used instruments. In this type of apparatus, the minute heart action electric currents cause deflections of a gold plated quartz fiber stretched in a strong magnetic field. The projected shadow of this fiber, highly magnified, moves across an illuminated field and falls on a continuously moving strip of film; thus, the string deflections are photographed continuously. Horizontal lines to measure the amplitude of excursion of the string shadow, and vertical lines to measure time intervals are photographed on the film also.

Time lines in the finished record are one millimeter apart and represent 0.04 second; every fifth (heavy) line represents 0.20 second. The horizontal lines are one millimeter apart, equivalent to 0.1 millivolt of current after proper standardization of the machine; while the space between every fifth (heavy) line is five millimeters and represents a deflection of 0.5 millivolt of current. These lines are used to determine the duration of the intervals and amplitude of the waves of an electrocardiogram respectively." (Fig. 1).

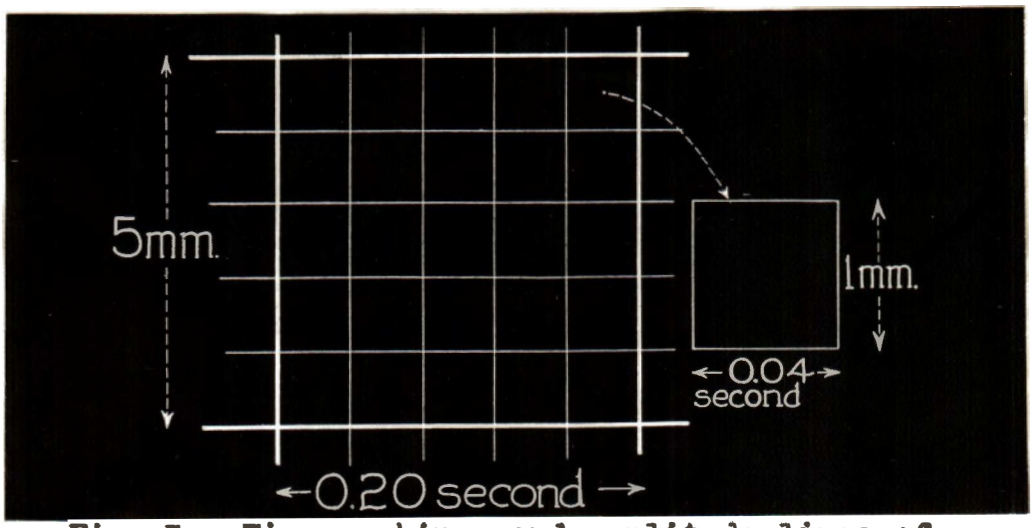


Fig. I.- Time markings and amplitude lines of the electrocardiogram.

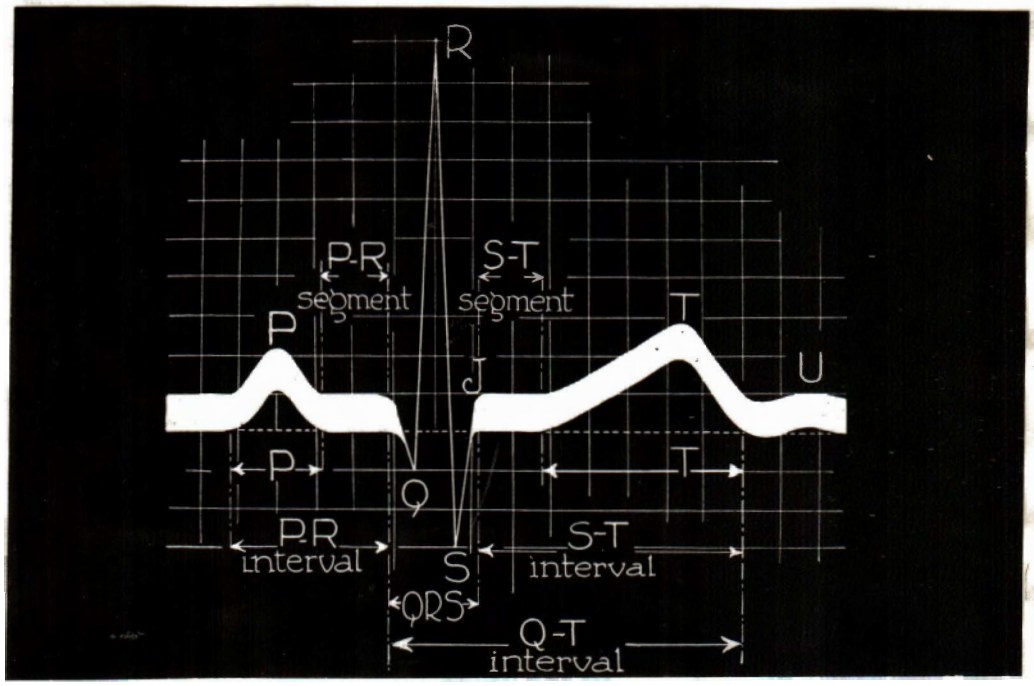


Fig.2.- Waves of the electrocardiogram.

## Physiologic Basis of Electrocardiography

The successive contractions of the auricles and ventricles are due to the passage of an excitation (depolarization) wave which precedes by a brief interval the contraction of the heart chambers so stimulated. This impulse originates in the pace-maker of the heart (sinp-auricular node), and is conducted by a peculiarly differentiated muscle tissue comprising the sino-auricular node, the atrio-ventricular node, the Bundle of His, and its right and left branches with their arborizations. From the sino-auricular node the wave spreads over the auricles in all directions, much the same as waves spread when a pebble is dropped into water. On reaching the atrio-ventricular node, there is a slight delay following which the impulse spreads swiftly to the ventricles through the atrio-ventricular node, Bundle of His, its right and left branches, and on to the Purkinje network, lining the interior surface of the ventricles. The bulk of ventricular musculature is then excited by a slower passage of the wave from the interior to the exterior surface of the ventricles.[11, 13]

## The Process of Electrocardiography

An electrocardiogram is a photograph of the oscillation of the galvanometer string. Minute changes of electric potential enter from the electrodes attached to the body surface to deflect the string. An active or injured muscle (not resting or dead) produces an electric potential. When a patien t is recumbent and in absolute repose the skeletal muscles are inactive and the main electrical potential comes from the musculature of the heart. An electrocardiograph records the site of impulse initiation, its spread and retreat, in the sense of depolarization and repolarization. An electrocardiogram is an accurate graph of the electrical activity of the heart. This electrical activity produces waves or complexes, labeled P wave, QRS complex, and T wave. The duration of a wave, complex or interval is measured from its convex curvature. The amplitude of a positive deflection is measured from the top of the base line; that of a negative deflection from the bottom of the base line. (Fig.2).

Electrocardiography is concerned with only two cardiac functions; Impulse production and impulse transmission. It is of service in the study of heart disease only when these two functions are disturbed. It does not record the force of the heart beat.

## Origin of Heart Currents

The electrocardiogram pictures the time relations of the invasion (depolarization) and retreat (repolarization) in the heart. The P wave represents the stage of invasion of the auricles. The QRS complex represents the stage of invasion of the ventricles. The T wave represents the retreat of activity from the ventricles. The duration of each of these waves gives the time of invasion or retreat. The amplitude and contour of the waves represent the unbalanced stresses from moment to moment during these periods. The P-R interval marks the conduction of time from the sino-auricular node to the ventricles. The S-T interval includes the end of invasion and the period of retreat of the ventricular musculature. The T wave represents ventricular repolarization, the finale of ventricular systole.

## Electrocardiographic Technique

An electrocardiographic lead is the connection of any two parts of the body by electrodes with wires to the recording instrument. The standard limb leads are called one, two and three respectively. The electrodes are connected to the extremities of the recumbent patient. Lead one consists of the right arm and left arm; lead two, the right arm and left leg; lead three, the left arm and left leg. The galvanometer is built so that an upward(positive) deflection appears on the record when the polarity of the leads are such: In lead one, when the right arm is negative and left arm positive; in lead two, when the right arm is negative and left leg positive; and in lead three, when the left arm is negative and left leg positive. These three lead points constitute the vertices of a triangle which is essentially equilateral. (Fig. 3). Electrically and geometrically, lead two is equal to the sum of leads one and three.

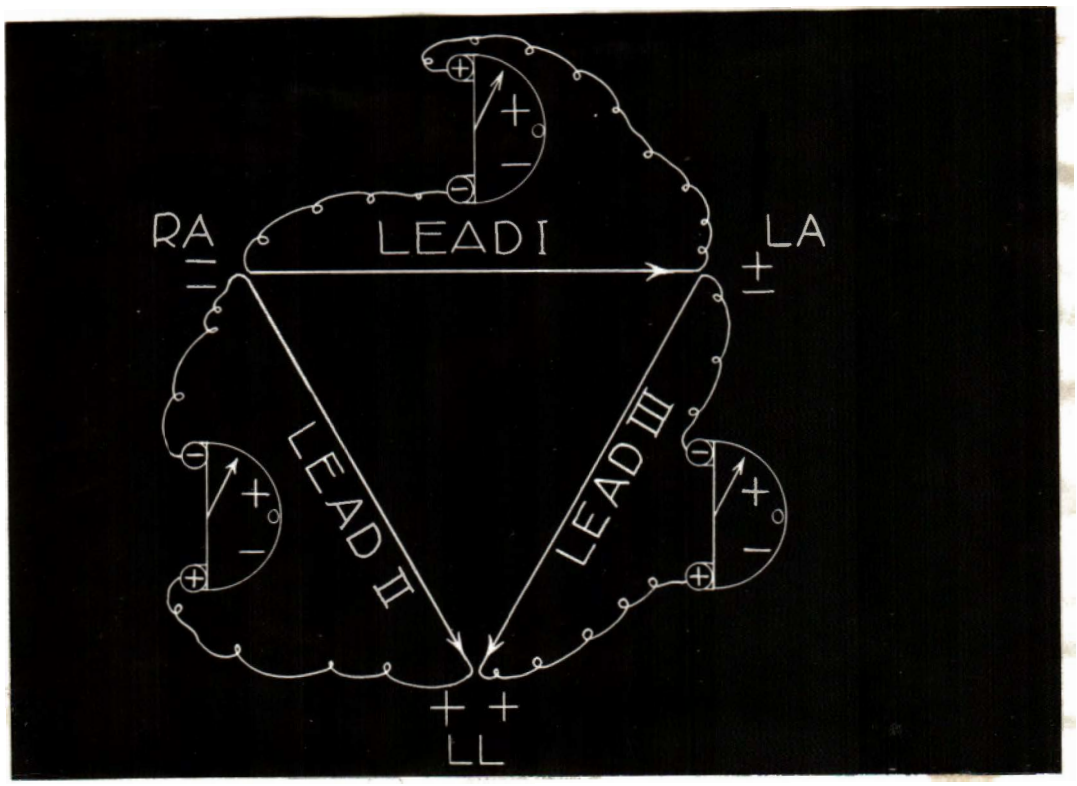


Fig. 3.- Leads I, II, and III. Current flowing in directions indicated by the arrows produces upright deflections for each of these three leads.

## Artefacts

Great care is necessary to avoid artefacts in in records. The most common cause is sixty cycle interference or muscle tremor. Artefacts may be due to unclean or improperly applied electrodes, extraneous currents, inaccurate standardization, poor illumination, faulty action of time marker or camera and defective development of records. Much care must be used to avoid interpreting artefacts as indicating myocardial disease.

## Method for Reading Electrocardiograms

Strict adherence to some routine for interpreting electrogardiograms prevents clinicians from overlooking significant changes in the record being examined. The following routine is suggested:[11, 13]

1. Determine the auricular and ventricular rates of the heart.
2. Determine the axis deviation present.
3. Detect and study irregularities of rhythm.
4. Look for abnormalities of the P wave.
5. Determine length of P-R interval.
6. Examine for abnormalities of QRS complex.
7. Determine length of QRS interval.
8. Detect and study changes in S-T interval.
9. Observe all abnormalities of T wave.
10. Summarize abnormal graphic observations.
11. Discuss interpretation of abnormalities.
12. Draw conservative clinical conclusions.

## Axis Deviation

The projection on the plane of the chest of the resultant of the distributed electrical potential developed by the heart at any instant is designated as the electrical axis of the electrocardiogram at that instant. The resultant and its projection are vector quantities; i. e., they have magnitude and direction. The mean electric axis of the QRS complex is the electrical axis at the peak of the R wave and is the only one of interest for ordinary clinical use. It may be determined from Bayley's Triaxial Reference System. This mean electrical axis has the direction of the resultant of all potentials developed by the component muscle fibers of the heart at the instant of the R spike. During the cardiac potential cycle the electrical axis corresponds to the direction at a given instant of time. The direction of the resultant electrical axis of the heart varies within wider limits than does the anatomic axis.

The position of the heart within the chest influences the graphic record. The normal electric axis lies between zero and plus ninety degrees of Einthovens triangle. If the angle is negative, there is left axis deviation; if beyond ninety degrees, there is right axis deviation.

## Left Axis Deviation

Displacements of the heart upward and leftward to a transverse position by a high diaphragm may cause left axis deviation of a normal heart. This often occurs in a short fat person with a broad chest or big abdomen, during pregnancy, with ascites, or on full expiration. Inversion of the QRS complex (deep S or Q wave) in lead three indicates left axis deviation. Left axis deviation may be caused by hypertrophy and/or dilation of the left ventricle, aortic regurgitation or stenosis, coarctation of the aorta or any other condition which places an additional load on the left ventricle. (Fig. 4).

## Right Axis Deviation

The low position of the diaphragm in a tall thin person, especially on full inspiration, leads to a vertical heart and may cause right axis deviation. An inverted QRS complex in lead one indicates right axis deviation. It often accompanies mitral stenosis, pulmonary embolism, septal defects, patent ductus, emphysema, pulmonary arteriosclerosis; or any other condition causing relatively increased load on the right ventricle. Axis deviation often indicates no more than a change in QRS direction due to an altered position of the heart, but it may be associated with heart disease. (Fig. 5).

## Waves of the Normal Electrocardiogram

The electrocardiogram of a cardiac cycle consists of a P, QRS and T waves. The excitation wave responsible for P and QRS precedes auricular and ventricular contraction; the T wave represents the finale of ventricular systole. Measurements of deflections should be made on the lead in which they are the greatest. This is often lead two (Fig. 2).

The Normal P Wave. The P wave is the auricular deflection. It represents the spread of the excitation wave through the auricular musculature parallel with its endocardial and epicardial surface. This wave of depolarization precedes by a brief interval the wave of contraction in the auricles. It originates from the normal pacemaker, at the head of the sino-auricular node, and spreads throughout the auricles and finally to the atrio-ventricular node. The wave of depolarization gives rise to differences in electric potential, and these cause deflections recorded as the P wave.

Normally the P wave is a blunt, rounded, sometimes slightly notched upright deflection, less than 0.10 second in duration and less than two millimeters tall. The P wave may be upright, isoelectric, diphasic or inverted in lead three of a normal electrocardiogram.

The Normal P-R segment and Interval. The P-R segment represents auricular depolarization plus the delay in spread of the impulse through the atrio-ventricular node. The P-R interval represents the atrio-ventricular conduction time; i.e., the time required for the excitation wave to travel from the sino-auricular node via the auricular musculature and atrio-ventricular node to the upper reaches of the Bundle of His. It represents the time required for depolarization of the auricular musculature plus the delay in transmission of the wave through the junctional tissue. It is measured from the beginning of the upstroke of the P wave to the beginning of the RS complex, and normally is not more than 0.20 or less than 0.10 second in duration.

The Normal QRS Complex. The QRS Complex is the wave of ventricular systole. It represents depolarization of the ventricles. It is due to an impulse which has traversed the atrio-ventricular bundle, its main divisions, the right and left branches and their arborizations, and the ventricular muscle from the endocardial to epicardial surface. It represents the spread of a supraventricular impulse through the normal conduction system and musculature of the ventricles. This excitation wave spreads through the conduction system ten times as rapidly as through the

heart muscle.

The normal QRS wave is a sharp, spikelike, monophasic, diphasic or triphasic complex. It consists of a small (or no) initial downward deflection (Qwave), or an initial tall upward deflection (R wave), the downstroke of which, should it go below the isoelectric level, becomes the S wave of the Complex. Normally, the complex is predominately upward. If the major QRS deflection is above the isoelectric level, it is termed an upright QRS complex; if below, it is inverted; if as much above as below, it is diphasic. Slight slurring may occur normally and with axis deviation, or rotation of the heart in any direction. The QRS complex may be notched in lead three of a normal electrocardiogram. The Q and S waves in leads one and three are used in the determination of axis deviation. The normal QRS Complex varies from five to twenty millimeters in height, and does not measure over 0.10 second in duration. It varies inversely with the heart rate and age of subject.

The Junction "J". J indicates the junctional point between the QRS Complex and the S-T interval. Normal deviation is not more than one millimeter in either direction.

The Normal S-T Segment and Interval. The S-T segment represents the short period of electrical balance between cardiac apex and base. It represents final depolarization and initial repolarization of the ventricular musculature. The segment extends from the end of the QRS Complex to the beginning of the T wave. Like J it does not normally vary over one millimeter above or below the base line in limb leads. Its length is difficult to determine because it often merges with the T wave.

The S-T interval is composed of the S-T segment and the T wave. Normally the S-T interval (like J and S-T segment) does not vary more than one millimeter from the base line of the record. Its normal duration is 0.24 to 0.28 second.

The Normal T wave. The T wave represents the finale of ventricular systole and terminates when all parts of the ventricles become quiescent. It represents the process of repolarization of the heart, and subsidence of the state of electrical excitation in the ventricular musculature.

Normally the T wave is a blunt or rounded upright deflection. It varies from one to five millimeters in height and has a maximum duration of 0.25 second.

The T wave in lead three is often inverted in the normal electrocardiogram. The T wave in lead one is especially valuable for study since it shows not only many of the same changes as the T waves in lead two, but often more decided and characteristic abnormalities suggestive of certain cardiac lesions. A flat T wave in lead one often indicates myocardial damage. A negative T wave in lead one is definite evidence of heart disease.

The Normal Q-T Interval. The Q-T interval extends from the onset of the QRS complex to the end of the T wave. It represents the duration of depolarization and repolarization of the ventricles and is the best measure of the duration of electrical systole. It varies primarily with the heart rate.

## Presentation of Two Normal Electrocardiograms

At this point is presented records taken from two young healthy medical students here at the University of Nebraska. They have had no history of or any demonstrable evidence of cardiac disease. Respective ages are twenty-six and twenty-nine years. The two are presented to illustrate the normal variations of pattern in the electrocardiogram (Fig. 6).

Subject A: Interpretation. Normal rhythm is present. The P waves and P-R intervals are normal. The QRS complexes are rather low in voltage, their duration is 0.06 second, which is normal and there is normal axis deviation. The S-T segments are at the isopotential level, and the T waves are upright though appearing in lead two somewhat high and sharply peaked which is within normal limits. (Fig. 6A).

Subject B: Interpretation. Normal rhythm is present. The P waves and P-R intervals are normal. The QRS complexes are of normal voltage, their duration is 0.06 second, which is normal, and there is slight right axis deviation with a little slurring of the complex in lead three. The S-T segments are displaced very slightly upwards in leads two and three. The T waves which follow are all upright and normal. (Fig. 6B).

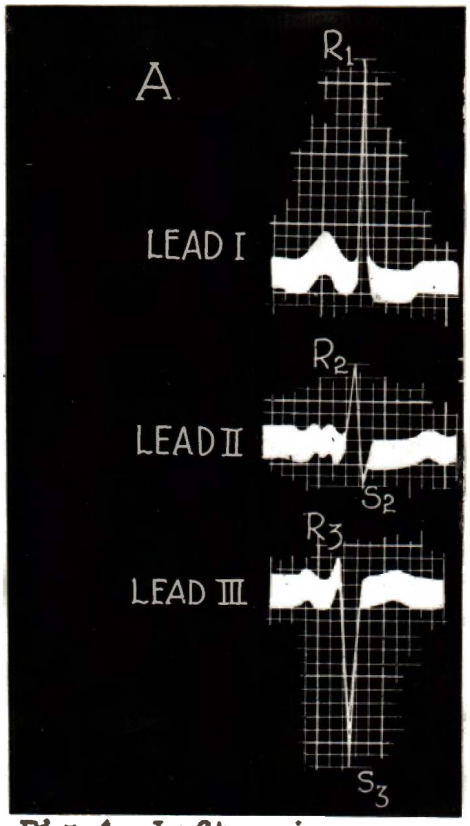


Fig.4.-Left axis deviation.

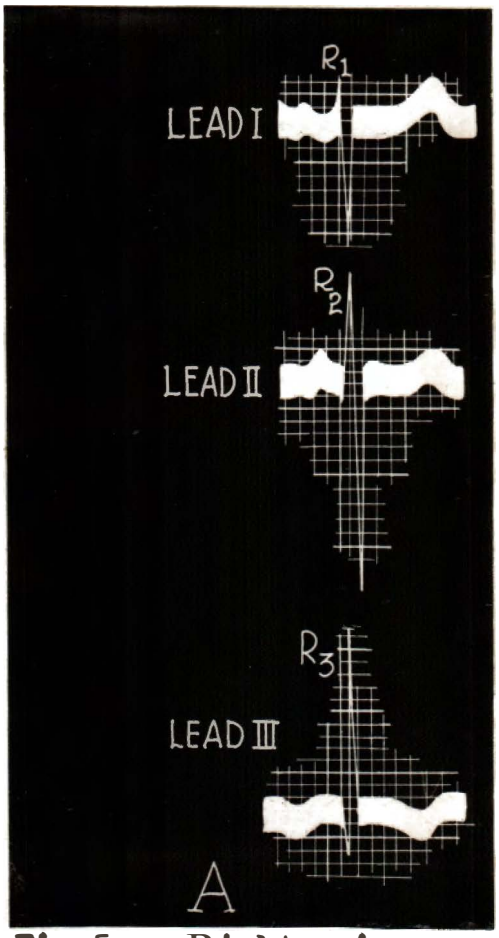


Fig.5.- Right axis deviation.

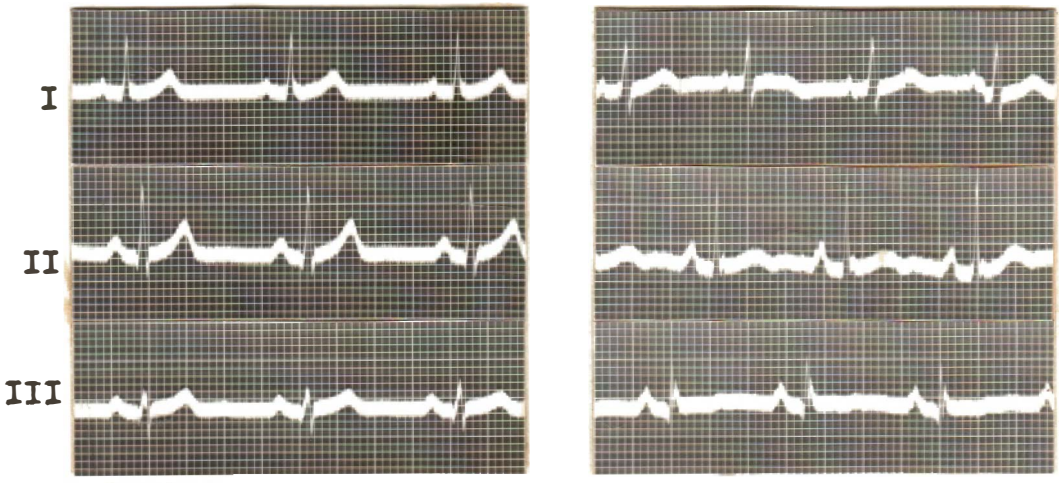


Fig.6a.

Fig.6b.

Two normal electrocardiograms.

## Mechanism of Production of Normal Electrocardiographic Pattern

That which is to follow is a brief resume' of a popular theory advocated by Burch and Winsor.[11] The ideas expressed, however, are for the most part in accord with theory proposed by Bayley.

In order to appreciate the electrocardiographic changes produced by infarction, and to interpret tracings intelligently it is necessary to understand the mechanisms involoved. The discussion to follow concerns an anterolateral infarct and the Q, T, pattern. The mechanism for lead one will be used here for convenience.

The normal resting heart is fully polarized as shown in Figure 7, and no difference in potential exists in lead one. Before an impulse enters the Purkinje system from the AV node and Bundle of His it is first delivered to the subendocardial layer of muscle on the left side of the septum, and a wave of depolarization begins. This wave migrates toward the RA (right arm) electrode and away from the LA (left arm) electrode, rendering the former positive and the latter negative causing the galvanometer to swing negative and inscribing the down stroke of the septal Q wave (Figure 8A).

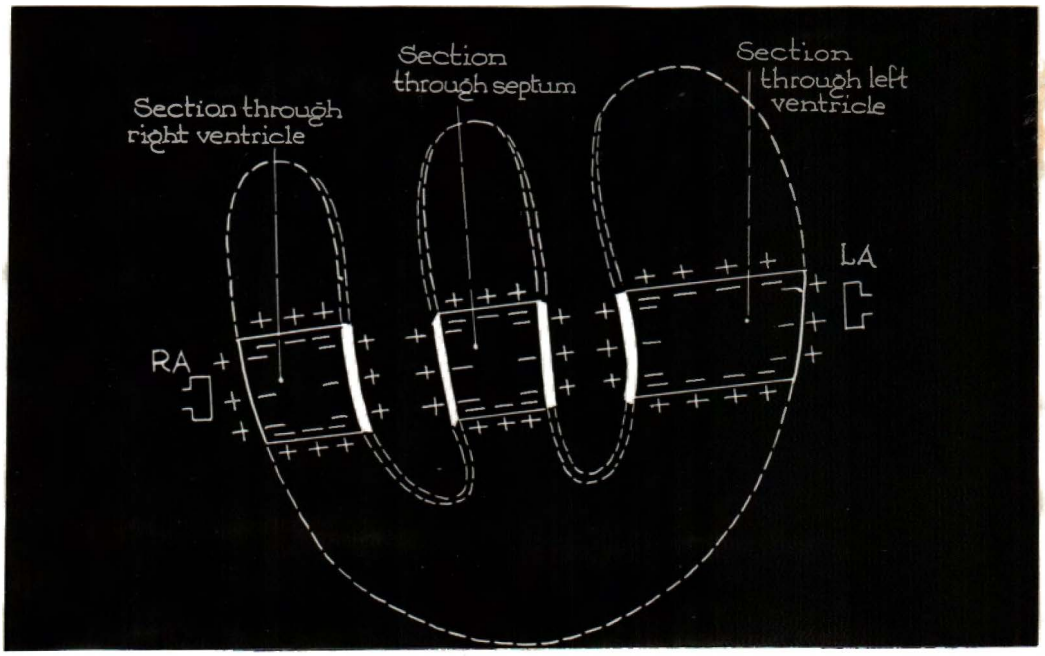


Fig.7.- Transverse section through the entire heart showing relative positions of the segments to each other and to the RA and LA electrode of Lead I. No difference in potential exists between the RA and LA electrodes when the heart is at rest and fully polarized.

Within 0.02 second or less the Purkinje system delivers the impulse to the subendocardial layer of the remainder of the ventricular musculature, thus inaugurating other waves of depolarization (Fig. 8B). The electric forces (band c) created by depolarization on opposite sides of the septum are equal, opposite and cancel out; however, the muscular mass of the left ventricle is greater than that of the right ventricle, and since the magnitude of electric force is dependent on muscle mass, the effective electric forces flowing toward the left arm electrode during depolarization exceeds that flowing toward the right arm. Hence the LA is rendered positive and the RA negative, causing the galvanometer to swing to the positive, inscribing the upstroke of the R wave (Fig. 8B). As the wave of depolarization continues through the walls of the heart, the potential differences decrease until, with completion of the process, there is no difference in potential; the galvonometer returns to the baseline, thus completing the downstroke of the R wave (Fig.8C).

Following mechanical systole a wave of repolarization begins. The process of repolarization begins where depolarization ended, and it begins simultaneously at the epicardial surfaces of both ventricles and

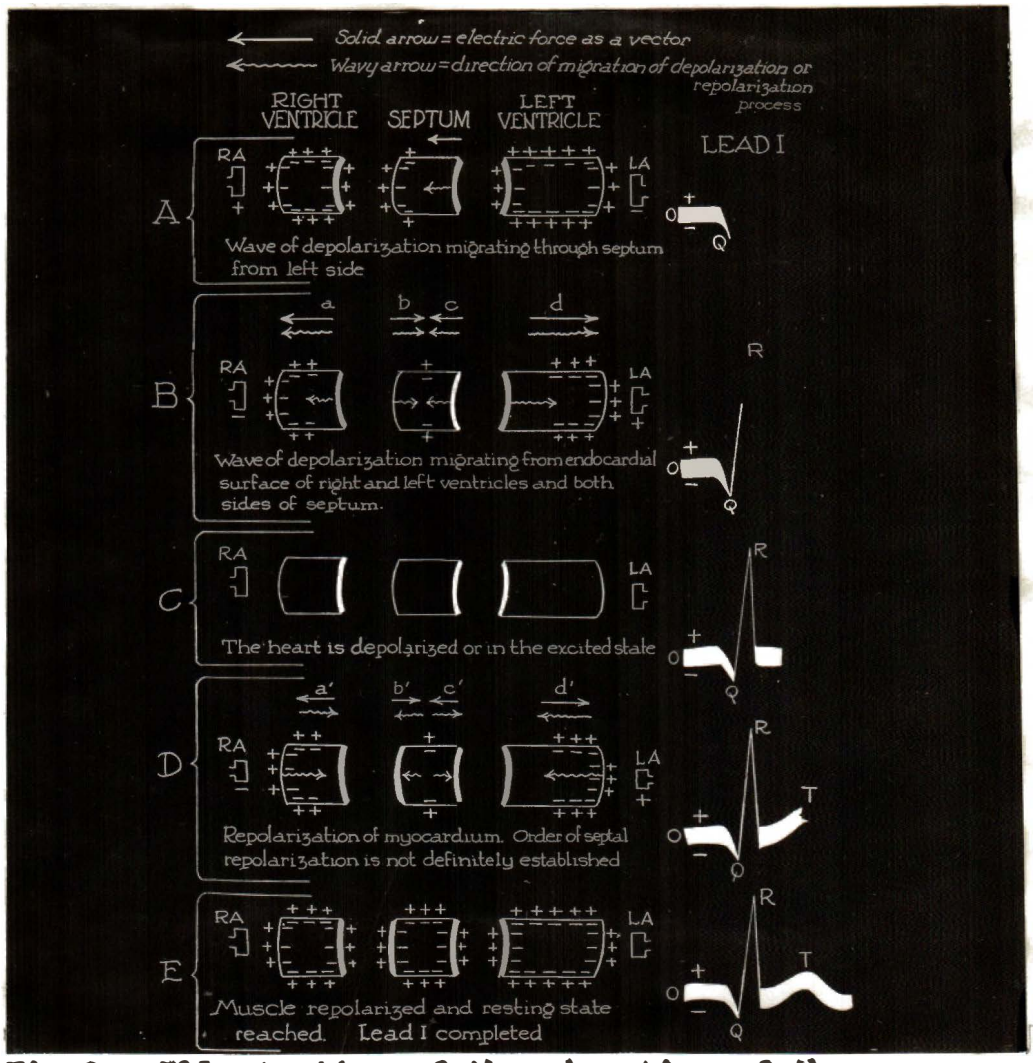


**Fig.8.- Illustration of the migration of the processes of depolarization and repolarization and their resultant forces in the normal heart. The influence of these forces on the RA and LA electrode of Lead I and the inscription of Lead I are shown.**

center of the septum. Because the electric forces (b' and c') produced in the septum act in opposite directions, they cancel each other out. However, the electric force (d') produced in the thick left ventricle is greater than that (a') produced in the thinner right ventricle. The force in the left ventricle is directed toward the LA, whereas the smaller force in the right ventricle is in the opposite direction. Hence the vector addition of the two forces (a' and d') results in a force directed toward the LA, making the LA electrode positive and the RA electrode negative, and a positive T wave is inscribed (Fig. 8D). When repolarization is complete and the resting state achieved no differences in potential exist and the galvanometer swings back to the isopotential line, completing the down stroke of the T wave (Fig. 8E).

Note that during the process of repolarization the direction of migration and the resultant electric forces are in opposite directions.

## Zones in the Region of a Myocardial Infarct

From a physiologic and electrocardiographic viewpoint the area of an infarct may be divided into four zones. (Fig. 9).

1. The dead zone is the central zone which is composed of dead cardiac muscle. This zone has no physiologic or electrocardiographic activity.

2. The zone of injury is a shell of cardiac muscle immediately surrounding the dead zone. The muscle in this zone is composed of fibers, some recovering, some dying. It is this zone that is responsible for the S-T segment changes in the electrocardiogram.

3. The zone of ischemia is a shell of cardiac muscle surrounding the zone of injury. The muscle in this zone is only slightly injured and most of the cells are progressing toward recovery. This zone is responsible for changes in repolarization reflected as T wave changes.

4. The zone of normal muscle.

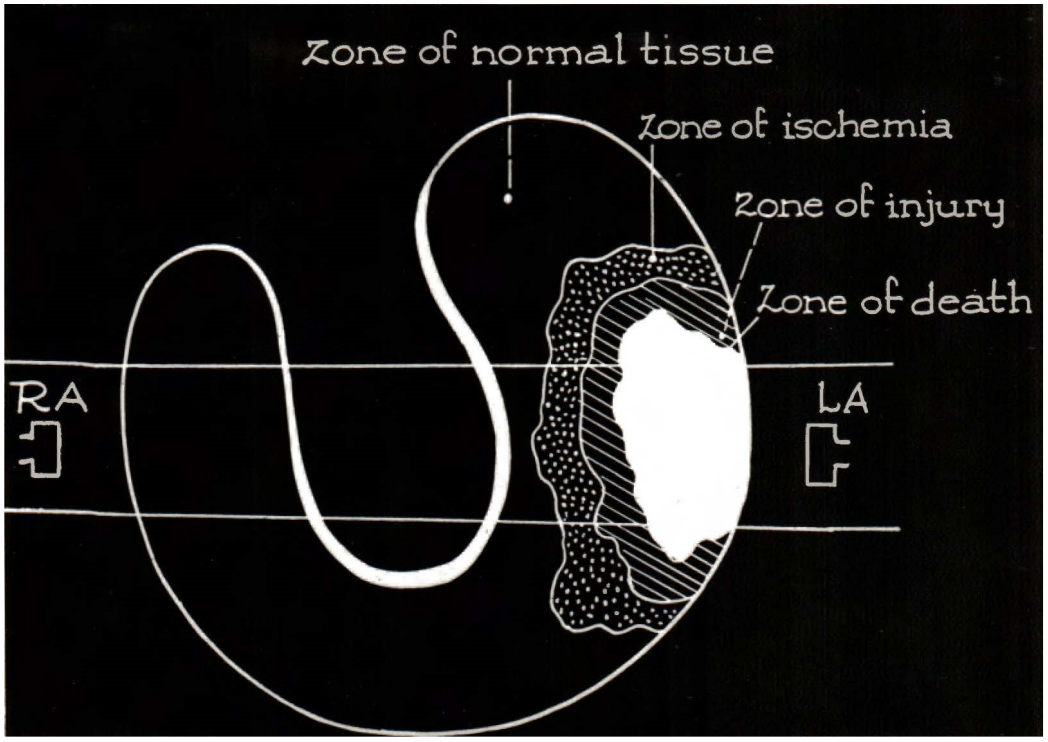


Fig.9.- Illustration of the transverse section of myocardium from the infarcted heart. The septum is eliminated.

## Mechanism of Production of Electrocardiographic Pattern of Anterior Infarction

From the preceeding it can be reasoned that the infarct has disturbed the state of polarization of the resting heart. In the dead zone the myocardium is completely depolarized and devoid of any electrical activity. In the zone of injury where the membrane of the injured cells has become more permeable and the ionic gradient between extra- and intracellular fluids is somewhat reduced, partial depolarization results. Since the subendocardial shell of normal muscle is fully polarized there exists a potential which is directed toward the RA. This potential difference causes the LA electrode to be relatively negative and the RA electrode relatively positive. Therefore the galvanometer swings to the negative or downward by this "current of injury". (Fig. 10A). This would ordinarily place the whole of the tracing to a level below the isopotential line on the graph. However, in recording the electrocardiogram all steady currents (from the body) are neutralized by a source of electomotive force within the circuit of the electrocardiograph. Thus, this anticipated negative displacement does not occur, but manifests itself by the upward displacement of the RS-T segment in the finished records. This RS-T shift will be explained presently (Fig. 10B).

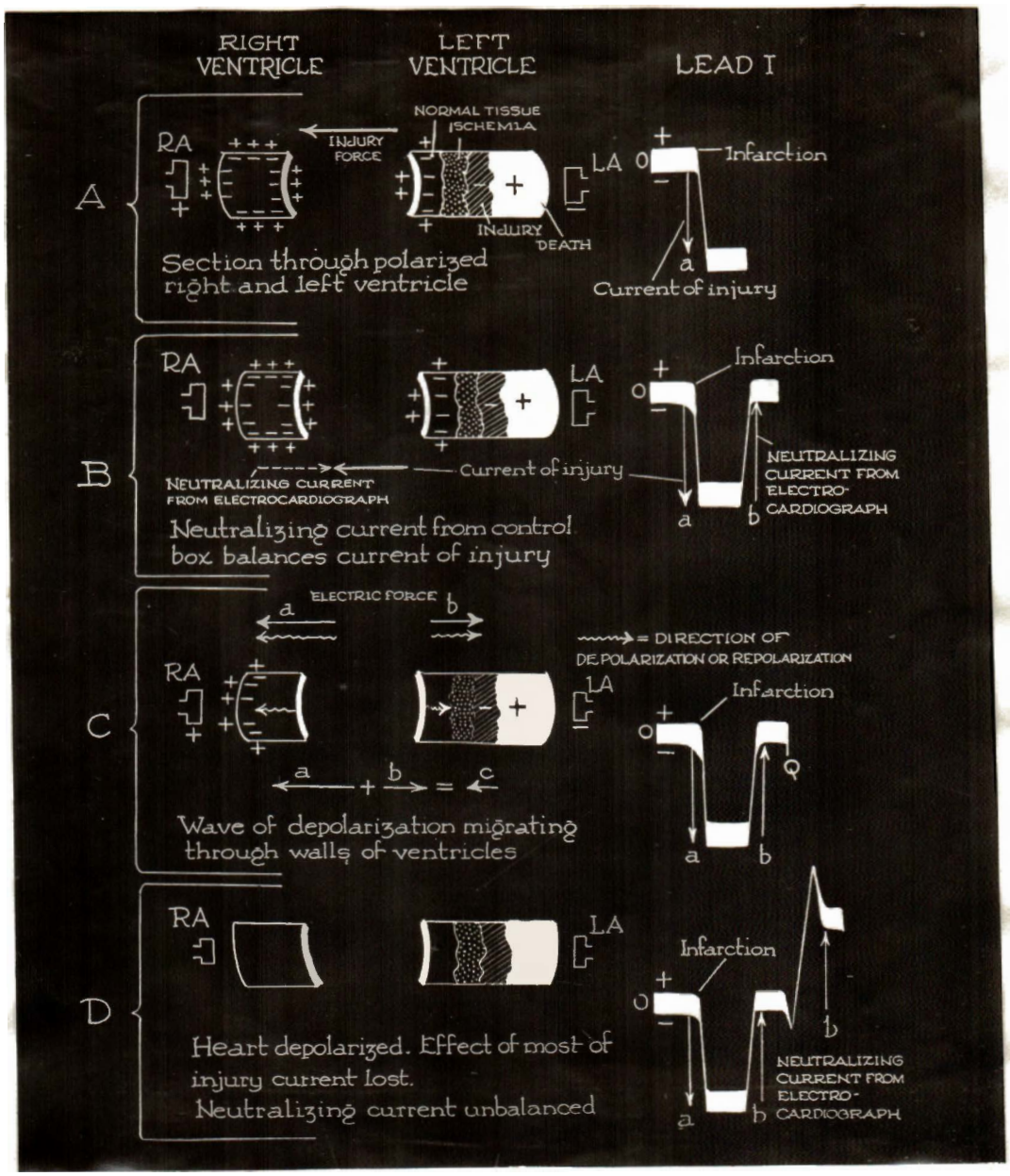


**Fig.10.- Diagram showing the influence of the processes of depolarization, repolarization, current of injury and neutralizing current from the electrocardiograph on the electrocardiogram in the case of an infarct involving the free wall of the left ventricle.**

When an impulse is delivered by the Purkinje system to the subendocardial layer of muscle of the ventricles a wave of depolarization is started in both ventricles and progresses from the endocardium towards the epicardium. In the early phases of this process, since the mass of muscle in the right ventricle is relatively increased, the resultant electric force (a) produced in the wall of the right ventricle exceeds the magnitude of that (b) produced in the noninfarcted wall of the left ventricle. Therefore, the vector addition of forces (a) and (b) yields a force (c), which is directed toward the right arm electrode (Fig.10C). The RA then is relatively positive and the LA is relatively negative, causing the galvanometer to deflect in the negative direction, inscribing the downstroke of the Q wave (Fig. 10C). As the process of depolarization continues, the right ventricle becomes completely depolarized, while living muscle around the infarct in theleft ventricle is still being depolarized. This makes the LA electrode relatively positive terminally with respect to the RA electrode, and the galvanometer swings to the positive,inscribing the R wave. Since the infarct has destroyed much of the left ventricle, a small R is produced,(Fig. 10D). In fact the larger the infarct,

the smaller $R_1$ will be. Therefore it is evident that the dead zone or infarcted area per se is responsible for the QRS changes ( deep $Q_1$, low voltage QRS) seen in myocardial infarction. Without QRS changes a definite diagnosis of infarction cannot be made.

Now, when the depolarization process is completed and no potential differences are present in the heart, the downstroke of the R wave, which normally comes to rest on the isopotential line, levels off at a considerable elevation above this line. This is due to the fact that, as previously mentioned, the current of injury, which would depress the whole of the tracing below the isopotential line, has been neutralized, thereby elevating the recording as a whole, the negative baseline of the tracing now becoming isopotential and the true isopotential level elevated above the isopotential line. Thus the RS-T segment, which is truly isopotential, is elevated and is the only graphic evidence of the current of injury (Fig. 10D). It is evident that a shift of the RS-T segment is indirectly due to a current of injury or zone of injury.

Following ventricular systole the process of repolarization begins. In the right ventricle recovery starts at the epicardial surface, migrating toward the endocardium. In the left ventricle, because the area of ischemia retards the repolarization processes,

recovery begins at the subendocardium where the muscle is more normal, and migrates toward the epicardial surface. The electric force (a') created by the right ventricle is directed toward the RA electrode. The electric force (b') created by the left ventricle is also directed toward the RA electrode. The vector sum of these forces results in a force (c') (Fig. 10E), which causes the RA to be relatively positive and LA relatively negative. Hence the galvanometer swings downward inscribing a negative T wave. When repolarization is complete, the galvanometer swings to the zero level(Fig. 10F). Thus it is evident that the zone of ischemia is responsible for T wave changes in infarction.

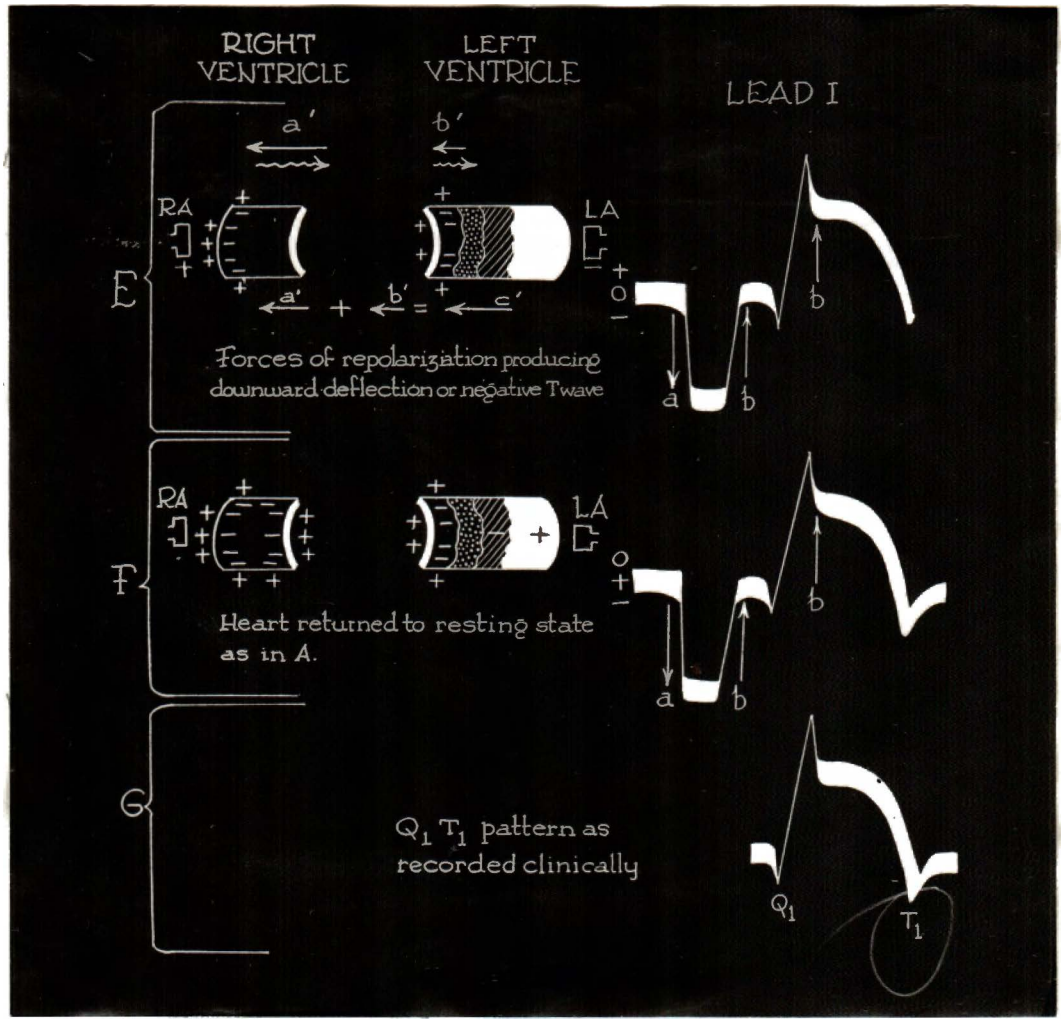


**Fig.10.- Continued.**

## Coronary Circulation and Site of Infarction

The most important branch of the left coronary artery is its anterior descending branch. This branch supplies the adjacent third of the anterior portion of the right ventricle, the anterior portion of the left ventricle, the anterior two-thirds of the basal portion of the interventricular septum, the apex of both ventricles, and a variable part of the posterior portion of the left ventricle adjacent to the apex. Barnes,[7] Rose,[30] and others[33, 11, 35] conclude that it is the most common offender in coronary occlusion. Occlusion of this anterior descending branch results in infarction which includes a varying amount of the anterior portion of the left ventricle, the adjacent interventricular septum, the apex, apical one-third to one-half of the lateral wall, and such portions of the posterior apical aspect of the left ventricle as are supplied by this artery, but seldom crosses over into the right ventricle.[40] This viewpoint is substantiated by Myers.[25]

The right ventricle is supplied by the right coronary artery. Infarction which follows occlusion of this vessel or its branches most commonly is found in the posterior portion of the left ventricle and septum, and at times in the apex, but almost never in the right ventricle.[3]

It is therefore concluded that acute anterior infarction most commonly involves the anterior aspect of the left ventricle, the apex,the adjacent interventricular septum and lateral apical wall.

Sequence of Changes Following Acute Anterior Infarction

The time element in development of the characteristic changes in the electrocardiographic pattern is of vast importance, because it is the sequence of changes which aids the clinician in determining the stage of infarction or its healing process, and thereby guides him in his program of therapy.

Patients with myocardial infarction due to coronary thrombosis will show abnormalities of the electrocardiogram in practically every case if more than one record can be obtained during the first week.[27] A single record will occasionally fail to show the diagnostic changes for these are sometimes late in their onset and sometimes transient. Whitten,[35] Barnes[4,6] and others[12,25] have shown that changes are present within thirty minutes to forty-eight hours after a coronary accident, and these changes persist for several weeks or months- or even years.

Soon after occlusion the part of the curve most strikingly affected is the RS-T segment[4,28,31] although QRS and T may also be changed. The typical features of this stage are elevation of the R-T interval in leads one and two and depression of the S-T component in lead three.[29] Usually the interval exceeds the limit of normal variation by from one to five millimeters. From the S-T segment the curve passes to the peak of the T

by a straight line or by a curve that is concave toward the baseline.[35,19] Pardee,[26] in his first report on infarction, stated that records taken shortly after a coronary occlusion may have as typical features "the extreme height of the T wave" in addition to the RS-T deviation. In 1947 Dressler[16] reported a series of 27 instances of high T waves found in tracings taken from one and one-half hours to twelve hours after onset of the occlusion. However, in no instances were the T waves in excess of the upper normal limits in lead one. Many current cardiologic textbooks[3,4,2,23] make no mention of these early high T waves. Therefore, it is concluded that if an early elevation of T occurs, it is not in excess of normal limits and is transient. Smith,[32] Willius[36] and others have shown that this RS-T deviation and upright T wave occur soon after infarction.

The typical RS-T deviation of the acute phase may appear within an hour or two of the time when the infarction occurs or may be delayed until the third day or even later; it may persist in typical form for a period which varies from 24 hours to as much as fourteen days, though usually from five to ten days.

As healing of the infarct progresses a characteristic series of changes develop in the T waves. The

descending limb of T begins to pass slightly below the zero level so that an inverted sharp peak follows the broad upward deflection which still remains.[26] The RS-T junction gradually approaches the zero level,[28] and as it does so, the downward peak becomes deeper until eventually the typical diphasic form (coronary T wave) is produced. This consists of an upward convexity of the S-T segment followed by a downward T wave. This is usually most pronounced in lead one. Herrick[21] was among the first to describe these changes (1919) (Fig.11).

While this sequence of changes is taking place in lead one (and two), the reciprocal lead (three) shows a gradual return of the S-T segment to normal so that when the stage of the coronary T wave has been reached in lead one, it is usual for the T wave in lead three to have a normal appearance. The typical S-T deviation of the acute stage thus gives place to the coronary T wave in a variable time. While this latter peculiarity may occasionally be established as soon as 24 hours after the attack, it more often appears between the fifth and tenth day.

The QRS changes, though not usually an early sign, almost always are manifest before the coronary T wave

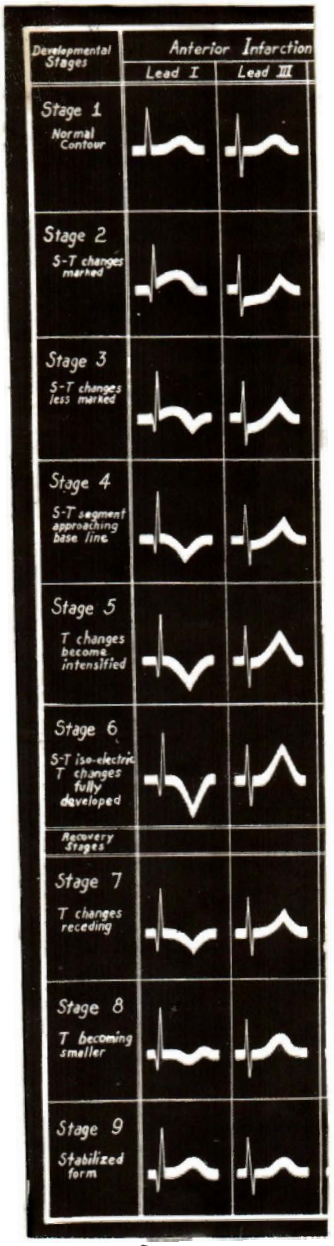


Fig.II.- Developmental changes in anterior myocardial infarction.

stage.[17] These changes consist of a deep $Q_1$, low voltage R in all leads, and deep $S_2$ and $S_3$.[22]

To recapitulate, the characteristic graphic changes of acute anterior infarction are:

1. Early stage, one to fourteen days after infarction:
   a) Elevated RS-T segment in leads one and (usually) two.
   b) Rounded convex or sloping RS-T segment in leads on and (usually) two.
   c) Depressed RS-T segment in lead three.
   d) Low voltage QRS with prominent $Q_1$ and deep $S_2$ and $S_3$.
2. Later stage, fourteen days to six months after infarction:
   a) Inverted T wave in lead one and (usually) two.
   b) Rounded or convex RS-T segment in leads one and (usually) two.
   c) High positive sharply peaked T wave in lead three.
   d) Low voltage QRS, with prominent $Q_1$ and deep $S_2$ and $S_3$.

## Substantiation of Diagnosis by Other Investigators

Correlation of typical electrocardiographic changes of anterior infarction with cases proved clinically or at autopsy began with Herrick[21] in 1919. He noted the typical T wave changes. Subsequently Pardee[26] (1920) reported a case showing left axis deviation, low voltage QRS, elevated S-T segment and inverted $T_1$. Smith[32] in 1923, in reporting on eleven cases, observed the same changes. In 1925 Willius,[36] reviewing 33 cases, concluded the most constant finding was a negative T wave in lead one. Clarke[14] at the same time was able to corroborate Pardee's findings.

Further observation in 1928 led Parkinson[28] to conclude that inverted $T_1$, positive $T_3$, in addition to early elevated RS-T and low voltage QRS was characteristic of the disorder. Rose[30] substantiated this in his investigation. Gilchrist,[18] in 1930, recognized all the above changes in a study of 148 cases, and stated that the changes, if they are progressive over a short period of time, are strong evidence of infarction. In two reports comprising a total of 122 cases Wilson[37,38] concluded that the typical findings (as previously described) in $Q_1$, $S_2$, and $S_3$, RS-T segment and T wave were present in the majority of his cases of pure anterior infarction. Since that

time many investigators--Barnes,[5] Bohning,[9,10] Dressler,[15] Sprague,[33] Winternitz[39] and Appelbaum,[1] to mention but a few-- have made similar observations and have corroborated these findings. Figure twelve illustrates admirably the usual changes in pattern observed in typical pure anterior infarction.

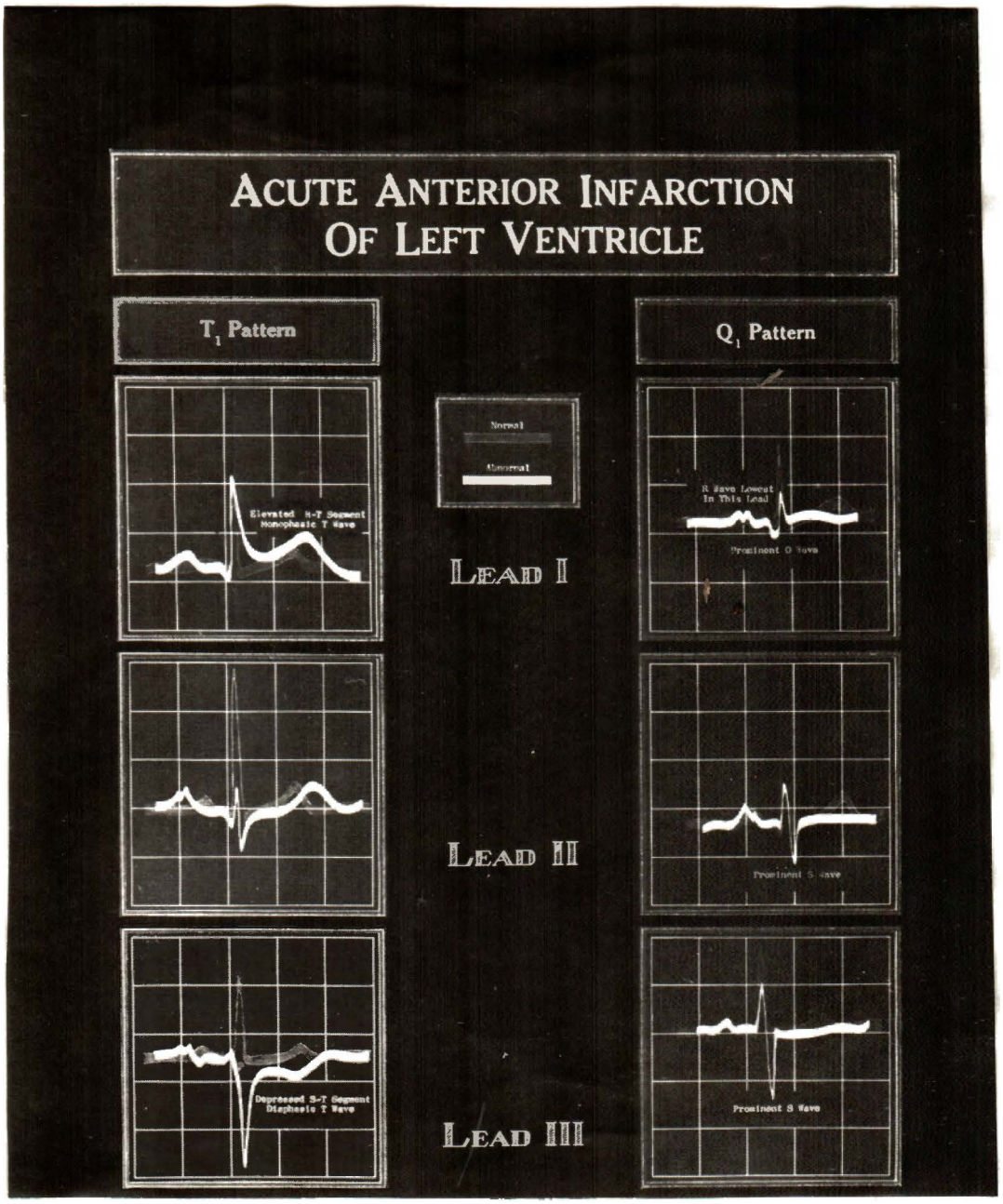
ACUTE ANTERIOR INFARCTION
OF LEFT VENTRICLE
T1 Pattern
Q1 Pattern
Normal
Abnormal
Elevated S-T Segment
Monophasic T Wave
R Wave Lowest
In This Lead
Prominent Q Wave
LEAD I
Prominent S Wave
LEAD II
Depressed S-T Segment
Diphasic T Wave
Prominent S Wave
LEAD III

Fig.I2.

## Presentation of a Typical Early Tracing

Here is presented a case of proved anterior infarction. The tracing was taken shortly after onset of symptoms. The characteristic form should be studied. (Fig. 13).

Interpretation: Normal rhythm is present at a rate of seventy-five to eighty. The P waves and P-R intervals are normal. The QRS complexes are rather low in voltage, their duration is 0.10 second which is the upper limit of normal, and there is left axis deviation. In lead one the RS-T segments are upwardly displaced and merge into the T waves which are upright. The RS-T segments are displaced slightly upwards in lead two and slightly downwards in lead three. The T waves which follow are normal. These findings indicate fresh myocardial infarction involving the anterior wall of the heart.

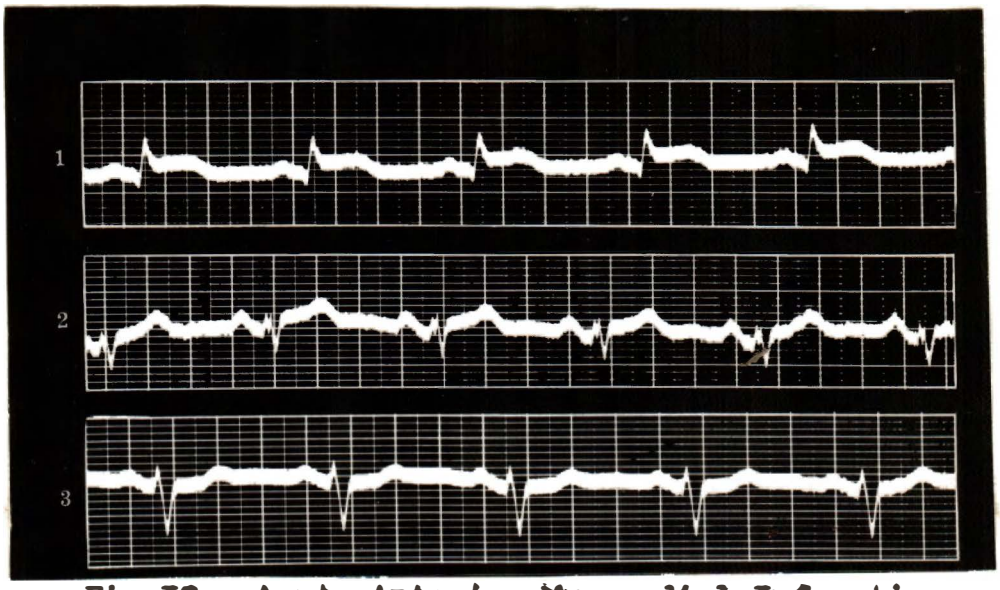


Fig.I3.- Acute Anterior Myocardial Infarction.

## Summary

An attempt has been made to give to the busy practitioner an understanding of the basic fundamentals of electrocardiography and a working knowledge in interpretation of the simpler types of cardiac disorders, especially acute anterior coronary infarction.

A brief review of the instrument used, its mechanics and the physiologic basis of electrocardiography was undertaken. A popular theory of the mechanism of production of the P, QRS and T waves was presented, and it was applied to the formation of both the normal tracing and the tracing seen in anterior infarction. For the sake of brevity the mechanism was applied to the formation of the waves in lead one only, but with understanding of the theories involved the formation of the waves in the other leads can be readily understood.

In this paper only the three standard limb leads were considered.

The tracings of two normal and one typical early anterior infarction were presented with interpretation of each.

The theoretical concepts used in explanation of the formation of the waves of infarction were substantiated by a large number of investigators who, from 1919 to the present, have correlated and corroborated electrocardiographic findings with cases proved both clinically

and at necropsy. A summary of the changes in pattern in anterior infarction as observed by these and other investigators was presented, sequence of change in the typical pattern was pointed out, and its value in diagnosis emphasized.

## Conclusion

The diagnosis of acute anterior coronary infarction by use of the standard leads resolves itself into two major findings: 1) typical changes in pattern, 2) orderly sequence of changes in pattern.

The typical changes in pattern are:

1. Elevated RS-T segment in lead one and (usually) two.
2. Depressed RS-T segment in lead three.
3. Coronary T wave in lead one and (usually) two.
4. Prominent $Q_1$.
5. Low voltage of QRS complex
6. Deep $S_2$ and $S_3$ .

The sequence of changes in pattern are:

1. RS-T changes are observed early, lasting usually from five to ten days.
2. T wave changes are observed somewhat later though they may be present in the early phases.
3. QRS changes precede, usually, the T wave changes and succeed the RS-T deviations, although QRS abnormalities may occur concurrently with the RST changes.

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