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Significance of the electrocardiogram in the diagnosis of ventricular enlargement

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THE SIGNIFICANCE OF THE ELECTROCARDIOGRAM IN THE DIAGNOSIS OF VENTRICULAR ENLARGEMENT

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The field of cardiac disease and its various manifestations is large and its importance has increased with each decade, until now cardiac evaluation should be one of the major points in any complete physical examination.

In evaluating a heart, one must determine the efficiency with which that organ accomplishes its specific work. The specific work of the heart is to propel blood through the vessels in adequate quantity to meet the needs of the body tissues. Any deviation from the normal should be recognized early and correct diagnoses, by discrimination between the different grades of heart disease, as accepted by the American Heart Association, should be made.

The facilities available for determining the functional capacity of the heart include the basic principles of physical diagnosis: inspection, palpation, percussion, and auscultation; accurate and complete history taking; and laboratory examinations; electrocardiogram, X ray, flouroscopy and blood studies.

In this paper only one of the above mentioned techniques, the electrocardiogram, will be discussed. The discussion will be specifically confined to the electrocardiographic findings in the presence of ventricular enlargement. Ventricular enlargement was chosen because

observed rhythmic contractions in a nerve muscle preparation placed in contact with a beating heart. It wasn't until thirty-one years later (1887) that Waller (58) accomplished the latter. Cardiac curves were perfected soon after by Bayless and Starling (1892) (9). These men used capillary electrometers to record their work. This was quite unsatisfactory because proper contacts were hard to maintain. (13)

Today the string galvinometer remains as the basis for many modern machines, but since the first world war and the development of thermionic vacuum tubes and the mirror osillograph two new types of machines have been developed.

The physical basis of all three machines is similar. The simplest explanation can be made of the string galvanometer. This consists of a finely spun, gold plated quartz string which lies in a strong magnetic field. The string is approximately 3 to 7.5 microns in diameter or the size of a red blood cell. It is suspended on a carrier that is constructed so the tension on the string can be changed. This provides a way of standardizing the amplitude of allowed movement from the point of rest and controls the natural period.

The string is a conductor and the passage of a current through it produces a deviation from the resting position. The degree of movement depends upon the amount of current flowing and the tension of the string. Therefore, the displacement of the string from its resting position will vary in direct proportion to the degree of thrust exerted within elastic limits of the string. The actual movement for a given current is limited only by the string tension.

The explanation for the above is on the basis of a reaction that arises between the constant magnetic field and a field created by the current passing through the conductor. This reaction is oriented at right angles to both the conductor and the steady magnetic field. The direction of movement may therefore be determined by the left hand rule of electricity. (26)

The deflections of the string are optically projected upon positive paper that runs in a vertical position. This projection is brought about by means of a beam of light that passes through two systems of lenses, a condensing set behind the magnets and a projecting set in front of the magnets. The light passes through the magnets by means of a hole drilled in direct line with the string. The standardization scale is projected upon

the film by means of calibrated lines marked on the front lens. The instrument is calibrated so that one millivolt will cause a string displacement of one centimeter. The time intervals are measured by a timer run on an independent motor. This marks a heavy line for each .20 seconds and a thin line for each .04 seconds.

In the electrocardiogram the current is derived from a potential drop that arises between two electrodes placed on the body surface, relative to various aspects of the heart. The current passes from the heart to the body surface through the blood and tissue fluids. These, in reality, are solutions of electrolytes.

The standard points of contact are the right wrist, left wrist, left leg and precordium. These points may be coupled in any one of four combinations, each of which is referred to as a lead. (6)

Lead 1 right arm to left arm
Lead 2 right arm to left leg
Lead 3 left arm to left leg
Lead 4 precordium to left leg

The Normal Electrocardiogram

During each cardiac cycle the electrical changes in the heart cause five different distinct movements of the galvinometer string. These have a wide variation

of normal and give definite pictures with specific pathology. The deflections are either above or below a zero or isopotential line. This is governed by the direction of the electrical axis. The electrical axis is the resultant of the combined electromotive forces of the muscle fibers. Its direction changes constantly throughout the cardiac cycle.

The waves inscribed throughout the cardiac cycle have been arbitrarily designated as P, Q, R, S and T. P, R, and T are usually directed upward, Q and S downward. The P wave represents the spread of the excitation wave through the ventricles. The T wave is derived from the retreat of the impulse from the ventricles.

The configuration of these waves is explained by the theory of limited potential differences. It is an accepted fact that an excitatory impulse doesn't travel through the heart in an organized manner as set forth by the theory of distributed potential differences, as the base apex theory is termed, but follows a semicircular course along the bundle of His and Perkenji network throughout each ventricle. This presents a clockwise movement in the right ventricle and a counterclockwise movement in the left.

As the impulse travels along it excites the underlying muscle. This excitation may occur at any point in a fiber or at several points. The excitation causes a point on the fibers surface to become relatively negative to the adjacent unexcited tissue. This negativity causes the adjoining tissue to become excited and the impulse progresses down the fiber. (7, 59)

To explain this system of progression more thoroughly let us consider a hypothetical cell. The cell membrane is charged positive on its exterior surface and negative on its interior. These charges are paired and referred to as electrical doublets. The excitatory impulse activates any one of these doublets and creates a point of negativity on the exterior surface which is surrounded by positive charges. These, in turn, are discharged and this progression exists throughout the cell. The doublets may be likened to small batteries connected in parallel, all the positive poles connected together and all of the negative poles connected together.

As each fiber is activated in the complex structural arrangement of the myocardium, multiple electrical currents develope such that the direction of electron flow is outward through the heart, at the site of activity. These have different directions in individual cells at a given

moment and from moment to moment. Some currents for any given orientation of pick up electrodes neutralize others, while some will reinforce. It is the algebraic sum of these that determines the potential difference recorded grossly in the electrocardiogram.

To consider this in more detail the changes in potential at the individual electrodes should be followed independently. Suppose to a hypothetical cell we attach two electrodes each of which is connected through a galvinometer with a separate indifferent electrode. As an impulse passes each electrode on the surface of the cell the corresponding galvinometer will move as a potential difference appears and disappears. This change in potential may be recorded against time as a curve. The two curves may be identical and will vary only in the relative time that exists between the activation of the tissue underlying each electrode. Now when the two electrodes on the muscle fiber are connected together through a galvanometer the algebraic sum of these waves is recorded. The complex curve thus produced includes both positive and negative phases and actually represents the summation of all the currents simultaneously developed in the heart. (59, 9, 14)

These waves have definite time intervals in the cardiac cycle. Increase or decrease denotes pathology. The average duration of the P wave is .11 seconds. The upper limit interval that exists between the P wave and the R wave or P-R interval in adults, with an average heart rate, is .20 seconds. This is measured from the beginning of the P wave to the beginning of the R wave. The duration of the QRS complex averages from .04 to .08 seconds, with an upper limit of .10 seconds. The R-T interval varies directly with the heart rate, increasing as the rate decreases and decreasing with an increase in rate. It, at the present time, does not have much significance. The T wave fluctuates with the spread of the QRS complex, usually running .22 seconds or less. (67)

Ventricular Enlargement

Soon after the advent of clinical electrodardiography Einthoven (1906) (18) described characteristic deflections which occurred in the three standard leads in association with ventricular enlargement. (20) These have become well recognized, but the mechanism of the changes remains obscure. Before I begin a discussion of the electrocardiographic changes, let us first briefly review a few

factors concerning ventricular enlargement: physiology, pathology and etiology.

The ventricles are the two lower heart chambers. Their specific function is to propel the blood into the pulmonary circulation (right ventricle) and systemic circulation (left ventricle). When their efficiency is impaired the heart must compensate. This can be done by increasing the rate or the size. The former acts with a very narrow physiological limit; therefore the latter has to be relied upon. Heart size can be increased by dilatation of the chambers, hypertrophy of the fibers or both. Dilatation compensates by increasing the volume of blood that can be held within the heart. The normal capacity for the right ventricle is 137 c.c. and that of the left ventricle 121 c.c. Hypertrophy brings about a change in the thickness of the walls and total weight of the heart. The left ventricular wall, in the adult heart, is normally of greater thickness than the right. Normal value for the right ventricular wall is 3-4 millimeters at a point midway between the apex of the right ventricle and the pulmonary valve, and 10-14 millimeters for the left ventricle when measured between the papillary muscles and the mitral valves. These valves may

be distorted by dilatation. The only way to determine the degree of hypertrophy is to determine the total heart weight or the weight of each ventricle and compare the findings with a normal ratio.

The normal weight of the heart has long been a disputed question. Smith (52) presents a plan that can be easily accepted. He considers the heart weight to be directly proportional to the size of an individual. He presents the following formula:

Heart weight =.43 X mans weight in grams
=.40 X womans weight in grams

To actually determine the degree of hypertrophy of the two ventricles, they have to be weighed separately and the ratio of their weights determined. This can be done by means of Lewis' dissection technique. The normal ventricular ratio is 1.85:1 with a normal range of 1.7:1 to 1.95:1. A ratio of 2:1 or more indicates left ventricular hypertrophy and a ratio of 1.5:1 or less indicates right ventricular hypertrophy.

Ventricular enlargement is caused by strain exerted on the muscle fibers. This strain may be intrinsic or extrinsic. The former refers to localized pathology

within the heart such as valvular disease, myocarditis or myocardial infarction. The latter refers to systemic pathology as hypertension, anemia, myxedema and chronic thyrotoxicoses. A more complete classification is by means of the involved chambers. This is shown by table I. (2, 12, 48, 56)

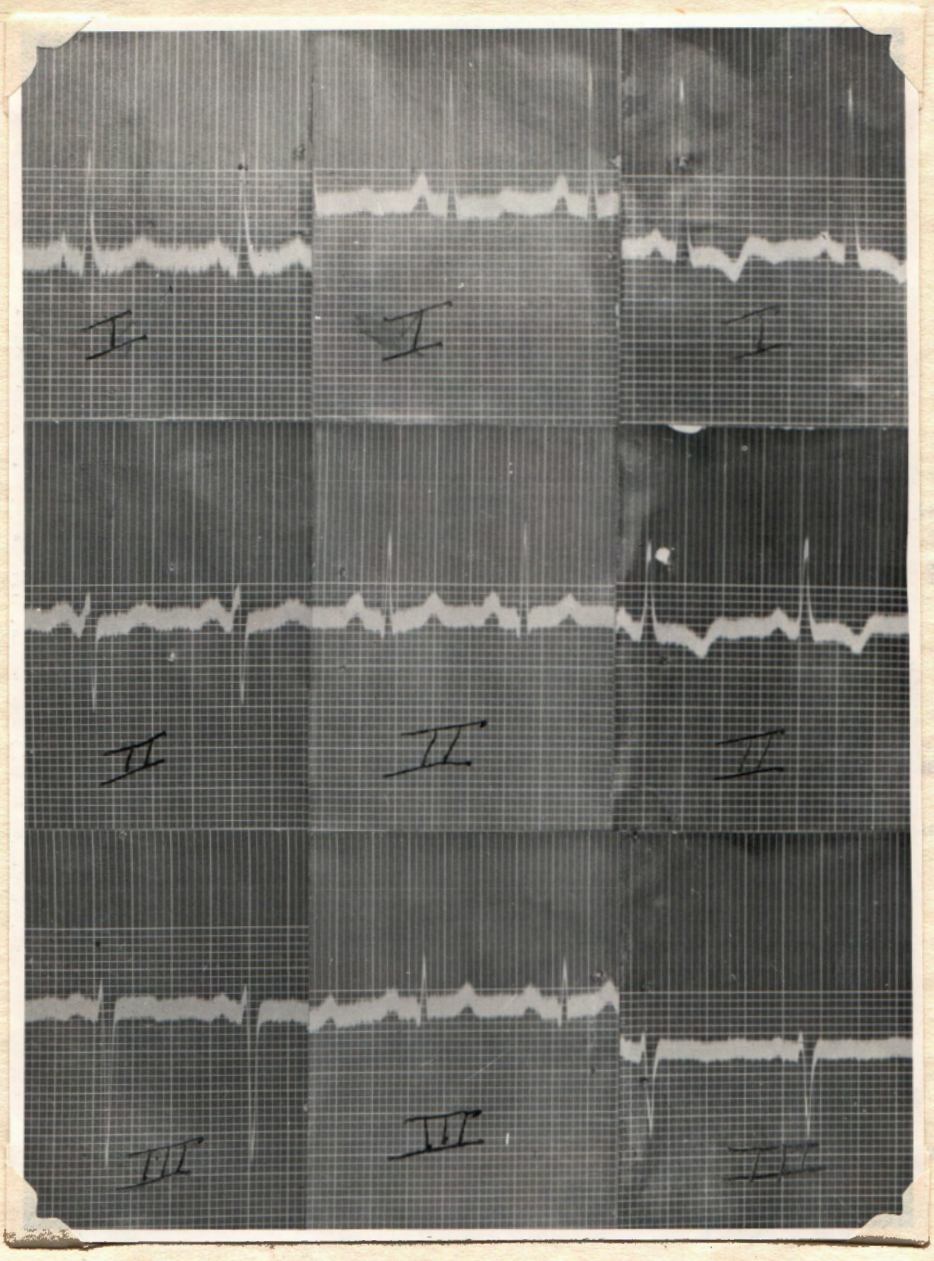
There are two explanations for ventricular enlargement. One is on the basis of changes in hydrostatic pressure and the other is from direct insult to the muscle fibers by disease. They both cause injury to the muscle fibers. The former first causes a stretching of the fibers and thus dissipation of the normal reserve. The latter produces a direct injury. The reaction of the tissue to either type of injury is an increase in the size of the muscle fibers and thus a relative increase in the reserve. (67)

The gross pathological picture depends upon whether the greater part of the enlargement is due to dilatation or hypertrophy. When a preponderant muscle weight increase is present the so called concentric type of enlargement is present. When dilatation is in the greater balance, an eccentric type of enlargement is present. The micro-pathological picture shows an equal amount

TABLE I

ETIOLOGY OF VENTRICULAR ENLARGEMENT

Primary Left Ventricular Strain	Primary Right Ventricular Strain	Primary Strain of Both Ventricles
1. Arterial Hypertension a. Essential b. Secondary to kidney disease 2. Aortic Stenoses 3. Aortic Regurgertation 4. Infarction of left ventricle myocardium 5. Congenital defects a. Coarctation of the aorta b. A. V. Septal defects c. Ductus Arteriosus	1. Mitral Stenoses 2. Pulmonary Hypertension a. Bronchiectases b. Emphysema c. Chronic interstitial pneumonites d. Silicoses 3. Pulmonary valve disease a. congenital b. acquired 4. Organic tricuspid disease 5. Beri Beri 6. Myocardial infarction 7. Congenital lesions a. Tetralogy of Fallot b. A. V. Septal defects with or without associated mitral stenoses 8. Primary left heart failure	1. Multiple valvular disease 2. Large myocardial infarcts 3. Chronic pericarditis with external adhesions 4. Severe Anemia 5. Myxedema 6. Thoracic and spinal defects 7. Renal disease 8. A. V. Aneurysms 9. Acute Myocarditis 10. Von Girkes disease 11. Cardiac neoplasms 12. Idiopathic congenital enlargement 13. Severe paroxysmal tachycardia in infants



A B C

FIGURE I

A. Left heart strain; left axis deviation (-40°)-QRS duration .08 to .09 , T waves and S-T segments normal.

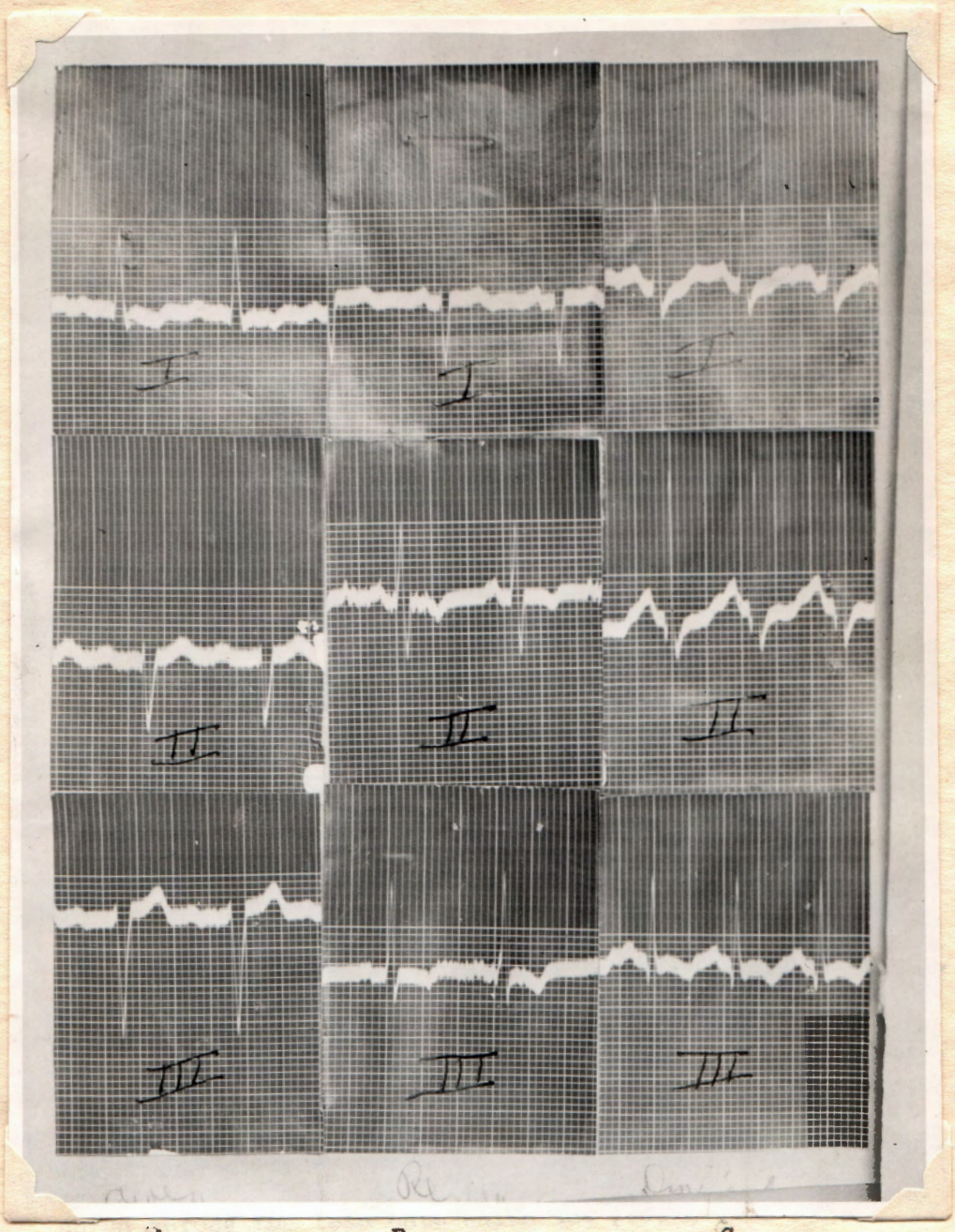
B. Left heart strain; no axis deviation, T of less amplitude then T_3 S-T, depressed one millivolt and S-T_3 elevated one millivolt.

C. Left heart strain; slight left axis deviation, QRS_1 amplitude increased, $T_{1,2}$ inverted. T_3 flat, S-$T_{1,2}$ depressed.

of enlargement of all the fibers and not an increase in the number of fibers. (28)

Electrocardiographic Findings in the Standard Leads

The electrocardiographic changes first noted by Eithoven were (18) a deviation of the electrical axis. He wrote that when the right heart was enlarged the deviation of the electrical axis occurred in lead one (figure IIB) and when hypertrophy or dilatation of the left heart was present the changes were in lead three (figure IA) (10) He also noted that physiological displacement could account for changes in the anatomical axis and thus changes in the direction of the electrical axis. Therefore changes in the electrical axis on a physiological basis must be differentiated from changes brought about by increase in the size of the ventricles. When this is done, evaluation of the tracing can be made. To do this the position of the heart, whether it lies in a vertical, normal or horizontal position must be obtained. One of the ways that this can be done is by determining the direction of the electrical axis, and if it lies within certain limits that are considered normal, the shift is on a physiological basis. If the electrical axis lies above the horizontal



A B C

FIGURE II

A. Left heart strain; left axis deviation (-35°), T, inverted, S-T, depressed one millivolt, S-T elevated 2 millivolts, discordant type of S-T segment.

B. Chronic right heart strain; right axis deviation (+ 110), $T_{2,3}$ inverted, and S-T_3 depressed one millivolt.

C. Acute right heart strain; S_1 present with small Q_3, T_2 flat T_3 inverted and S-T is depressed.

or past the vertical, abnormal displacement is present.

Many hypotheses have been presented to determine the electrical axis. The first was Einthoven's which may be briefly described as follows: (1) The body is a homogenous conducting medium, which can be considered a sphere; (2) The electrical activity of the heart, at a given instant, may be considered as a dipole; (3) The dipole is located in the center of the sphere; (4) The right arm and left arm and left leg may be considered as linear extensions of three points on the periphery of the sphere. They lie in the same plane as the dipole and are located so that if they were joined together by straight lines, they would form the sides of an equalateral triangle and represent the three standard leads of the electrocardiogram. Now, no matter how the dipole is situated in the sphere, perpendiculars drawn from the dipole to the sides of the triangle will be proportional to the potential drop measured across the lead onto which the perpendiculars are dropped. These projections will be positive or negative depending on their relationship to a perpendicular bisecting each side of the triangle. (21) Now if the projections of leads one and three are added together, their sum will always equal lead two.

The dipole or electrical axis occuring at any instant in the cardiac cycle may be plotted by determining the sum of $R_1 - S_1$ and $R_3 - S_3$. These sums are then measured off in millimeters on the corresponding lead sides of the triangle. The dipole then lies on a line constructed between a point in the center of the triangle and a point formed by the intersection of perpendiculars dropped from the sites plotted on the lead sides.

The direction of this dipole or the electrical axis is determined by the angle formed between the dipole and the horizontal. This can be done in two ways, either by formula or extension onto a circle surrounding the triangle. The lower half of this circle is considered positive and the upper half negative. Any dipole forming an angle between 0° and $+90^\circ$ is considered normal axis. If it lies between 0° and -20° normal left axis deviation is present and if it lies between $+90^\circ$ and $+110^\circ$ normal right axis is present. If the dipole has an angle of greater than -20° it lies above the horizontal, and abnormal left axis deviation is present and if the angle is greater than $+110^\circ$ it lies past the verticle, and abnormal right axis deviation is present. (67)

This theory is valid except in curves where the values of correspondingly named peaks in the three leads do not occur at synchronous points. Here simulaneous leads must be had. Except for this one instance this method is accurate enough for general use. (20, 46)

Other methods used to obtain the electrical axis graphically are by means of Dieuaide's chart (16) and Bayley's triaxial referance system. (9) Several formulas have been presented for the same determination. Two of the most common are White's (67) and the one presented by the Criteria Committee of the New York Heart Association. (15) White's formula concerns the relationship of the R wave in lead one to the S wave of lead three and the R wave of lead three to the S wave of lead one. Mathematically it is expressed $(R_1 + S_3)-(R_3 + S_1)$. With this, the normal range is from -10 or -15 to +20 or +30. Anything under this is right axis and over this is a left axis.

The Criteria Committee suggested that the deviation of the electrical axis be determined by finding the algebraic sum of the QRS deflections of each of the three standard leads. Then when the value is zero or positive in leads one and three and positive in lead two, there is

considered to be no deviation of the electrical axis. Left axis deviation is considered present when the sum of the QRS deflections is positive in leads one and two and negative in lead three. If it is negative in leads two and three the degree of left axis is to be considered pathological. Right deviation of the electrical axis is considered to be present when the sum of the QRS deflections is negative in lead one and positive in two and three. This is pathological if negative in leads one and two and positive in only lead three.

Another method by which the position of the heart may be determined and after which the pathology evaluated is through a comparison of the precordial leads with the limb leads. Wilson and Associates (62) have presented a plan using the unipolar precordial leads and comparing them with the unipolar limb leads. By this method exact position may be estimated without confusion of axis deviation. The theory of unipolar leads will be discussed later in the paper.

This plan is based upon the fact that the potential variations of the right arm (V_R) is similar to the potential variations of those parts of the heart's surface that are nearest the right shoulder. The potential variations

of the left arm (V_L) resemble the potential variations of those parts of the heart's surface that are nearest the left shoulder; and the potential variations of the left leg (V_F) are like the potential variations of the heart's diaphragmatic surface.

The potential of the right arm (V_R) is usually negative throughout the greater part of the QRS complex. The reason lies in the relation of the right shoulder to the great valvular orifices at the base of the heart and the negativity of the ventricle cavities is transmitted through these orifices to the adjacent parts of the body, including the right shoulder.

If the heart rotates into a horizontal position the right ventricle will lie below the left ventricle. Now the left leg lead will be exposed to a greater amount of right ventricular influence and the left arm to greater amount of left ventricular. The latter will resemble the precordial leads of the left chest. If the reverse is had the left leg will be exposed to more left ventricular influence than right and the left arm will be exposed to more right ventricular influence than previously. Now the latter will resemble the precordial leads from the right side of the chest. (61) Different degrees of either

type of rotation exist. Due to the correlation existing between the limb leads and precordial leads, Wilson and Associates present the following chart to determine the relative position of the heart.

Vertical Position (figure III)

a. The ventricular complexes of lead V_L resemble those of leads V_1 and V_2.

b. The ventricular complexes of lead V_F resemble those of leads V_5 and V_6.

Semivertical Position

a. The ventricular complexes of lead V_F resemble the ventricular complexes of leads V_5 and V_6.

b. The QRS deflections of lead V_L are small.

Intermediate Position

a. The ventricular complexes of leads V_L and V_F are similar in form and size and like those of leads V_5 and V_6.

Semihorizontal Position

a. The ventricular complexes of lead V_L resemble those of leads V_5 and V_6.

b. The QRS deflections of lead V_F are small.

Horizontal Position (figure IV)

a. The ventricular complexes of lead V_L resemble those of leads V_5 and V_6.

b. The ventricular complexes of lead V_F resemble those of leads V_1 and V_2.

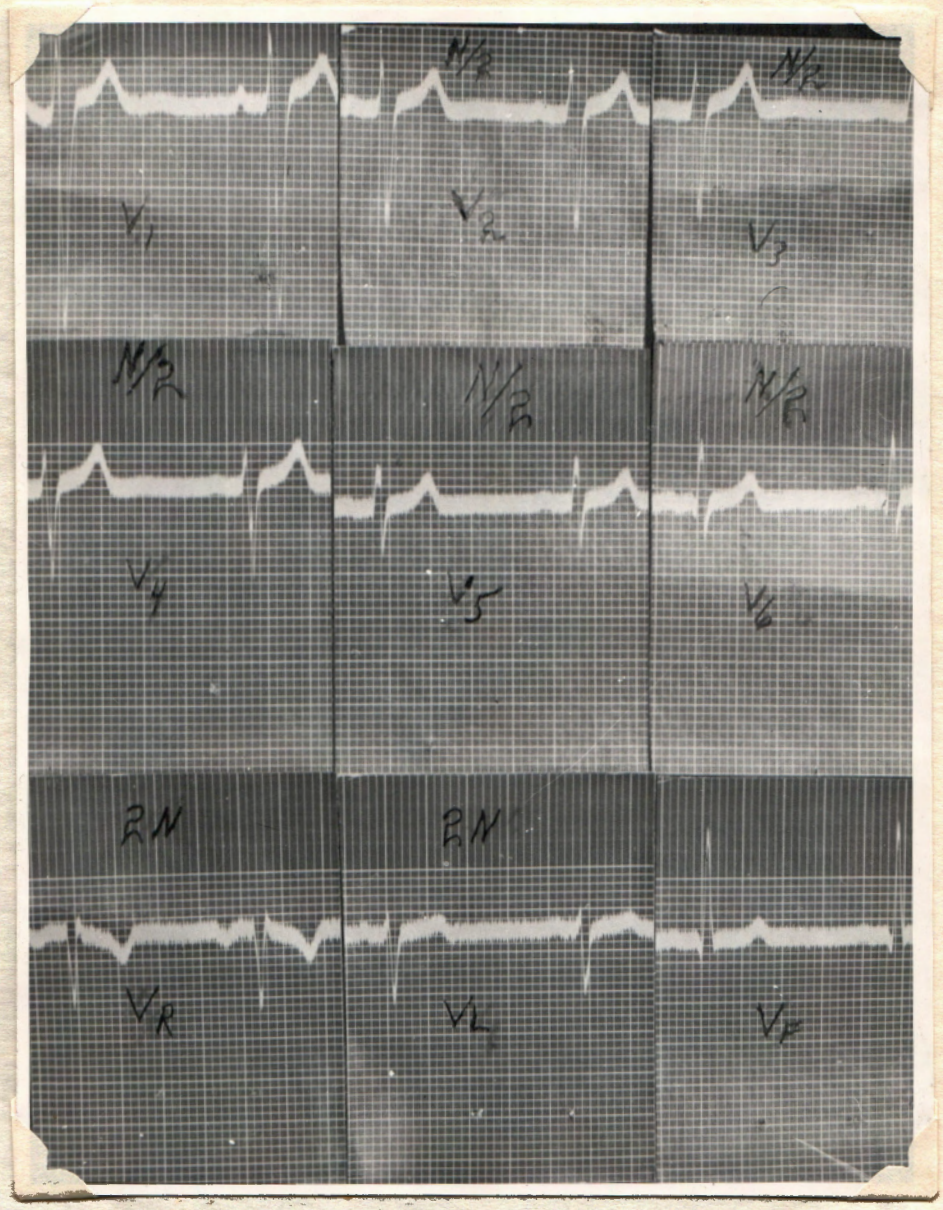


FIGURE III

Normal unipolar precordial curve; V_L resembles V_1 and V_2, V_F resembles V_5 and V_6; therefore a vertical position of the heart is present.

Indeterminate Position

a. There is no obvious relationship between the ventricular compleses of the limb leads and those of the precordial leads.

To summarize, it can be said that the axis deviation seen in ventricular enlargement is due to two factors. Whether these work alone or together remains to be determined.

The first is relative rotation of the heart or a change of relationship due to the enlargement of a ventricle. This causes a change of the normal relationship between the heart mass and the paired electrodes. This distorts the normal picture in such a way that one lead, which was positive, will now be negative.

The other factor is the change of time relationship that is created by the enlarged muscle mass. As the fibers increase in size the currents arising at each electrode will create a phase relationship that causes one wave to be more negative than the other at a point later than normal. Therefore, negative deflection exists. This also explains the negativity seen in the leads taken from the involved ventricles in bundle branch block. It also explains why the electrical axis is negative in leads taken from the ventricle opposite the one containing an

ectopic point which creates extrasystoles.

Besides the axis deviation there are other changes in the QRS complex in ventricular enlargement. These are increase of the amplitude and duration of the QRS complex. (figure IA) (60) Any amplitude that is equal to or greater than the allowed standardization can be taken as increased, and any duration of .09 to .11 seconds can be taken as an indication of ventricular hypertrophy. If the duration is longer, the various forms of bundle branch block must be eliminated. (66)

The explanation for the above two changes has not been as conclusively proved as that for axis deviation. It is thought both are due to the increase in muscle mass. This would cause an increase in the period of activation, leading to an increase in amplitude. The increased duration is explained by two theories. The first is that the increased muscle mass causes a prolongation of the conduction time; the second, anoxemia of the muscle will increase the conduction time. The latter has been proved in dogs, by clamping of the coronary arteries. This seems as if it would be quite possible in man because it is an accepted fact that, as the muscle mass increases, the blood supply remains the same. Therefore an increased

muscle mass places more strain upon the vessels present and a relative anoxemia exists. (49)

The Q wave is of some significance by itself. (45,22) With hypertrophy of the left ventricle a small Q in lead one is associated with a deep S in lead three. If a large Q exists in lead three associated myocardial disease is usually present, but not always. The direct opposite holds for right ventricular hypertrophy. The only explanation is that strain on the myocardium causes a decrease in the conduction time of the involved muscle. This muscle becomes activated later and this accounts for the initial downstroke of the QRS complex.

Other changes, often associated with the above, are seen in the T waves and S-T segments. These changes usually do not occur until advanced stages of myocardial strain are present. Then they appear in 88 per cent. (57)

In marked left ventricular strain T_1 and T_2 will be inverted and T_3 upright. (figure IC) Different gradations of this exist down to T_1 being upright but of less amplitude than T_3. (figure IB) These findings are usually associated with marked left axis deviation but it is not uncommon to find QRS_3 positive and of increased amplitude. (4,11)

The S-T segment in a left heart is below the isoe-

lectric line in lead one and slightly elevated in lead three. (figure IIA) This can be easily differentiated from the depression and elevation seen in acute coronary disease because it is two millimeters or under here, while acute coronary elevation or depression is over two millimeters.

Right ventricular strain gives a comparable picture of T wave and S-T segment changes. However, there are present here two different types of changes, those associated with chronic strain and those manifested by an acute condition, such as seen with an embolism of a pulmonary artery. (4,30)

The changes seen in the chronic type are not as marked as those of left ventricular enlargement. (figure IIB) Usually some elevation of the S-T segment in lead one occurs with slight depression in lead three. T_1 is always upright, T_2 and T_3 show different degrees of changes, varying from a decrease in amplitude to a marked inversion. The latter carries a graver prognosis. These are usually associated with a change in the direction of the electrical axis.

In acute right heart strain, changes in the electrical axis are rare. (figure IIC) An S_1 may develope and a small Q_3 is often had. The S-T segment begins below

the isoelectric line in lead one. T_2 and T_3 may be flat to inverted. The S-T segments in these leads are convex toward the base line and may or may not show the diphasic appearance seen in coronary involvement. (46)

The S-T segment changes of hypertrophy may be differentiated from those found in hypertrophied hearts with coronary sclerosis. In the former the convexity of the S-T segments are toward the base line or concordant (figure IC) while in the latter it is upward or discordant. (figure IIA) This point can be of much help in evaluating the prognosis. The latter is indicative of a pathological process that is irreversable. (28) Explanation is emperical and has been based on correlative findings between the electrocardiogram and autopsy.

The literature discussing the direct cause of T wave and S-T changes is controversial. Robb and Robb (45) found the wave changes depended upon which layer or layers of muscle were involved. They concluded that when only the superficial sinospiral muscle was damaged changes would occur causing elevation of the S-T segment. If the superficial bulbospiral muscle was involved S-T depression would be had in one lead with elevation in another. Also, if the

deep sinospiral and bulbospiral muscles were involved R and T wave changes would occur. The fallacy in this proposal lies in the fact that other workers have been unable to produce the same changes, but have produced the opposite. Gubner and Ungerleider (23) demonstrated that if the interventricular pressure was raised the S-T and T waves showed changes. This places the strain on the deeper muscle layers. These men also showed that such conditions as chronic anemia, carbon monoxide poisoning, shock, overexertion, anoxemia and mechanical trauma could produce this same picture. These all cause changes to occur in the subendocardial muscle layers. Conclusive proof of the fact that embarrasment of the subendocardial region will first show electrocardiographic changes is found in work done by White. He demonstrated that subendocardial infarcts heal less rapidly than those in the center of the myocardium or beneath the endocardium. (65)

Another theory is that metobolic strain caused by inadequate coronary circulation will bring about these changes. (1, 53) Inadequate coronary circulation causes a collection of acid metabolites. This is explained by two factors, a decrease in capillary count per square millimeter of heart muscle is present and an increase in

thickness of the hypertrophied muscle cells exists. The former causes a relative decrease in the amount of nutriment for each cell. The latter makes rapid diffusion of oxygen, nutriments and metabolites difficult. The same electrocardiographic changes are seen in cases with coronary sclerosis where a known decrease of blood to the tissues exists. Further evidence that the changes are due to a metabolic strain is that S-T and T wave changes may be reversible. This has been observed to occur in cases in which marked lowering of blood pressure has attended therapy of hypertension by various measures, such as sympathectomy and administration of thiocyanates, renal extracts and tyrosinase. (23)

This same theory of metabolic strain can be used to explain the change seen in serial tracings in which slight left heart strain first appears and then with increasing fibroses of anoxemia, shows progressive grades of embarrasment. This process may continue to completely involve the bundle of His and produce a bundle branch block.

Significance of Precordial Leads

Precordial leads are contacts made between the chest wall and a distant point on the body surface. They

have been standardized by the American Heart Association so that a deflection above the isoelectric line is considered relatively positive and one below is relatively negative.

In keeping with this standardization it is necessary that the precordial lead must be the ground point of the connection. For example, let us consider use of the regular third lead position on the electrocardiogram gang switch. Here the electrical activity travels from the right arm to the left leg. Therefore, the left leg contact must be placed over the precordium where it acts as the exploring electrode and the right arm contact over the left leg to form the indifferent electrode. (46, 9)

Precordial leads may be used either singularly or in multiple. The accepted positioning is in a line across the chest descending with the mass of the heart. (15) Lead one is found in the fourth intercostal space to the right of the sternum, lead two in the fourth intercostal space left of the sternum, lead three is located midway between two and four and placed on the fifth rib, four is in the fifth intercostal space at the left midclavicular line. Lead five is at the anterior axillary line at the same level, six is in the left midaxillary line, at the level

of four and five. Numerous other body leads are used. The above are employed more frequently. With the setup as explained the leads are referred to as CF leads. These letters stand for chest-foot. When a single precordial lead is referred to, in this paper, it is CF_4 because this is the one in standard use at the University Hospital.

In CF_4 left ventricular strain causes a depression of the S-T segment and also a flattening to inversion of the T wave. Changes in the QRS complex vary with the position of the heart. In a vertical position the R wave will be of increased amplitude and duration, if horizontal a deep S wave will be present. Here confusion may arise again with differentiation from an anterior coronary. The absence of a Q wave and past history of an acute attack are the main points of differentiation. Right strain manifests itself in much the opposite way. The S-T segment is slightly elevated. (.5-1 mm.) The T wave is upright and with a verticle position one will have a deep S wave and with a horizontal position a large R wave. (57)

The advantage of a single precordial lead in ventricular enlargement is realized when you consider that standard leads pick up cardiac changes in 64 per cent of

patients with hypertension and precordial leads 83%. (57) Pathology such as notching of the QRS complex, resulting from injury of the myocardium, will alter axis changes in the standard leads, but not to as great an extent as in the precordial leads. Coronary sclerosis masks the pictures of strain by producing signs quite similar and a Pardee Q in lead three will confuse the picture in like manner.

A recent contribution has been made in the field of precordial leads by Wilson and Associates.(62) Their technique is to connect through a central terminal, composed of three 5000 ohm resisters, leads from the three extremities. This equalizes the potential variations found in the extremities and gives you an indifferent pole whose theoretical potential is relatively zero. Thus you have a unipolar lead rather than a standard bipolar lead. These leads are referred to as V leads. The advantage of this type of connection is that an equalized potential gives more accurate recording of electrical changes in the cardiac cycle. The explanation is made on the basis that the amplitude of the precordium is approximately thirty times as great as in any extremity. This increase

in amplitude is appreciated by anyone who has taken precordial leads and had to cut their standardization into half to control the string. Explanation lies in the fact that as the location recedes from the heart, the amplitude will decrease with the square root of the distance. (3)

Even though CF leads are more commonly employed, my discussion of multiple chest leads will be confined to Wilson or V leads. For general purpose what is said for V leads may be applied to CF leads. With the latter standard limb leads are used as comparison, to obtain the heart's position. In this way CF_5 and CF_6 will resemble lead one. In the former the unipolar limb leads are used.

The picture of a normal precordial curve (figures III and IV), is characterized by very small R deflections and deep and relative broad S waves in leads from the right side of the chest. In these the peak of the R occurs early in the QRS interval and is followed by a large, abrupt downward movement which obviously corresponds to the intrinsic deflections of direct leads. The ventricular complexes of the leads from the left side of the precordium have a tall and relatively wide R component. This is often preceded by a small Q wave and followed by an S deflection.

The peak of the R is on the average about 0.02 seconds later, in leads from the left side of the chest than in leads from the right side. The transitional zone of these two different types of complexes varies from person to person and even within the same individual. It is usually found in leads from the third and fourth positions. (62)

The value of multiple precordial leads in the diagnosis of ventricular strain is to correctly evaluate the exact heart position and at the same time determine the degree of strain. Since the method of determining position has been previously explained, the following discussion will deal only with strain.

In uncomplicated cardiac strain the findings are mainly an exaggeration of the normal picture. The changes seen are not different from those of the standard leads. The leads affected are those that lie directly over the involved ventricles.

In strain of the left ventricle (figure V) increased amplitude and widening of the QRS is seen in all leads, but is most pronounced in those from the left chest (V_4, V_5 and V_6). Other changes are the decrease in the amplitude to complete absence of an R wave from the right chest leads

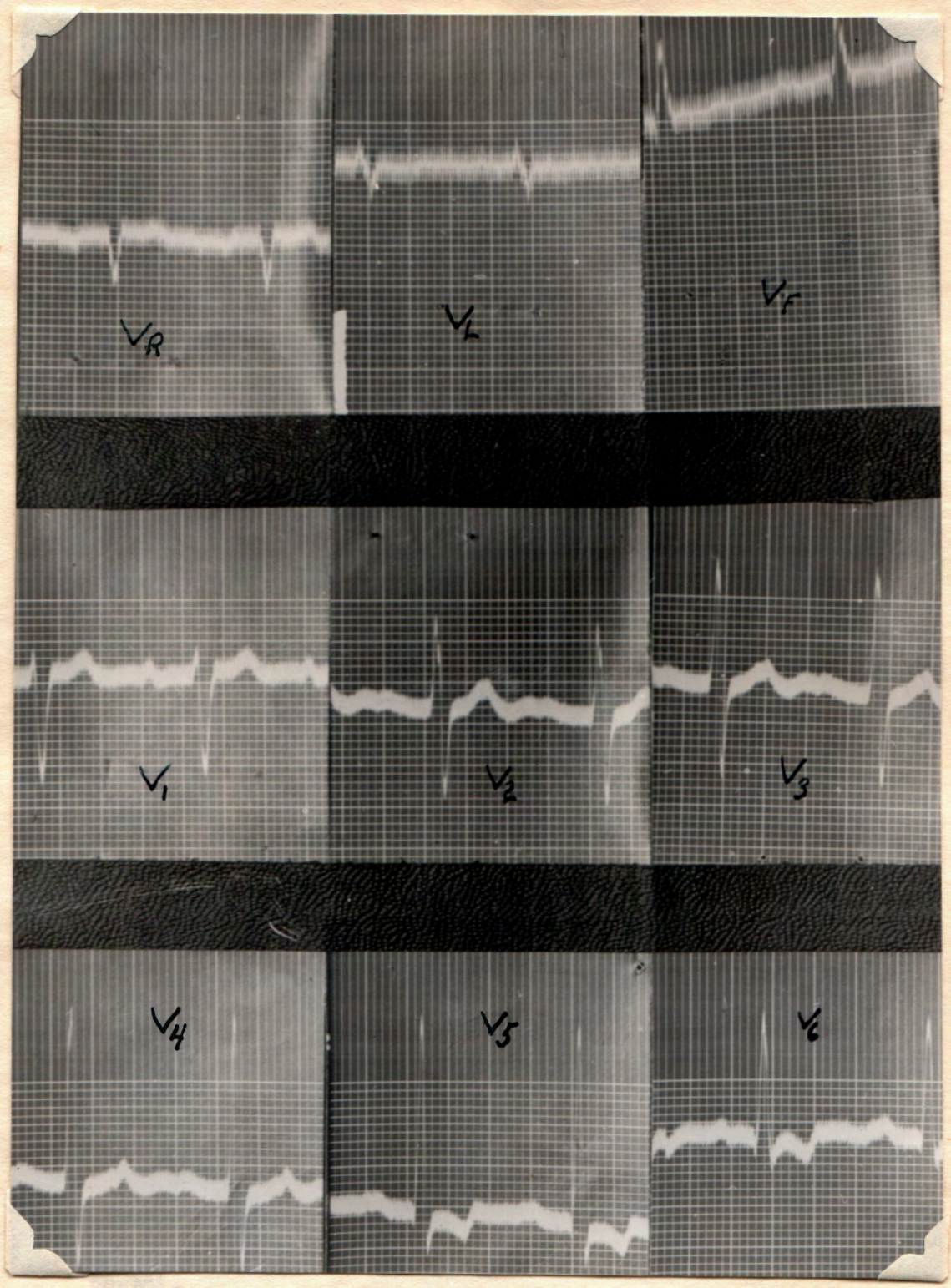


FIGURE V

V_L resembles V_1 and V_2; V_F resembles V_5 and V_6. Therefore vertical position of heart is present. -QRS duration .09 seconds, Transition in V_4 - T waves in V_5 and V_6 inverted; therefore left ventricular strain is present.

(V_1, V_2 and V_3). The transitional zone is shifted to the left. The R waves of the left side leads are increased. Often a prominent Q wave is present that increases in depth toward the left side of the chest. The peak of R comes abnormally late in the QRS interval and the T deflections are inverted.

In a high degree of right ventricular enlargement (figure VI), the picture appears much like that of bundle branch block, differing in that the interval is under .12 seconds and is not slurred or notched. As a rule the voltage of the chief QRS deflections are above normal although not as large as in left ventricular strain. In the leads from the right precordium the R wave is very large, the Q is frequently present, an S wave is usually absent and the T deflections are commonly inverted. In some instances a small initial deflection precedes the first downward deflection of the QRS group. In the leads from the left side of the precordium the R deflections are abnormally large. In other words, the precordial curves are opposite in type to those obtained when the heart is normal.

When the heart is enlarged it is usually in a horizontal or semihorizontal position. In either case the

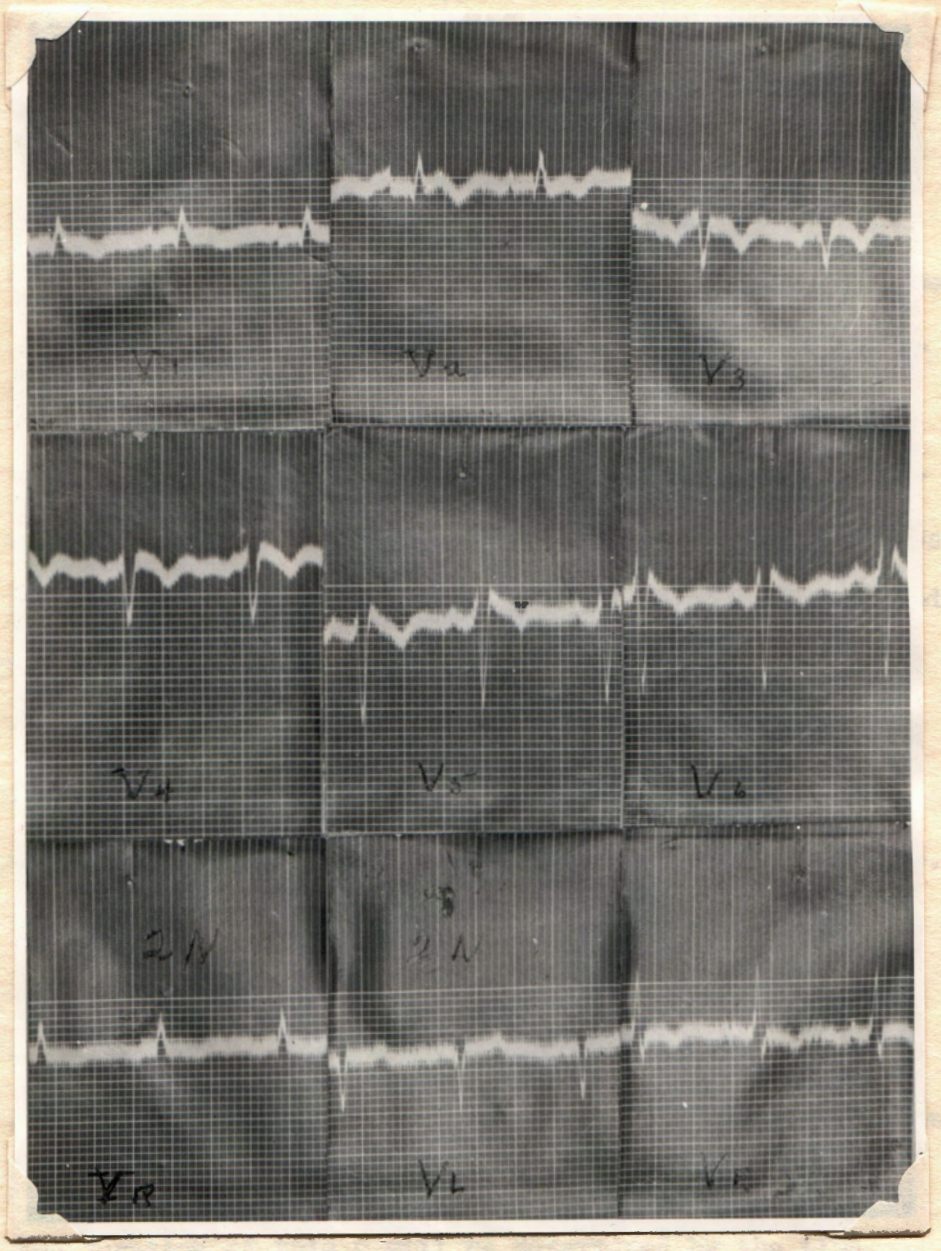


FIGURE VI

V_L resembles V_5 and V_6; V_F resembles V_1 and V_2; therefore heart is in horizontal position. V_1 and V_2 are positive. V_5 and V_6 are negative. The normal picture is reversed; therefore right heart strain is present.

changes previously described will be found. When a verticle or semiverticle position is associated with ventricular enlargement, the picture is somewhat different. The semivertical position produces abnormally large R waves and usually inverted T waves in left ventricular enlargement. Right ventricular enlargement produces large S waves in the standard precordial leads. A verticle position of the heart with left ventricular enlargement causes a right axis deviation. Right ventricular enlargement produces a left axis deviation. (62)

The explanation for changes here are the same as previously outlined for the standard leads. The only addition to make is in speaking of chest leads as a whole. Here the specific lead records the activity of the heart muscle lying directly under it. If the entire heart is activated at the same time a normal deflection occurs, but if some part of the heart becomes activated before that underlying the electrode, a Q wave is formed. The depth and duration of this Q will be determined by the time element involved. If muscle becomes activated after the tissue underlying the electrode, an S wave will be inscribed upon the record, the depth and duration of which will

be determined by the time element involved. This is not true in the case of normal leads V_1 and V_R because, as stated previously, the relative negativity of the ventricular cavities determine these deflections. (61)

Discussion

Electrocardiographic interpretation is based upon emperical knowledge gained by correlation of data obtained from electrocardiograms and clinical or post-mortem observations, and well constructed hypotheses. This information has provided the physical and physiological foundations of the science. At present, well established criteria are available so that electrocardiography is now an invaluable clinical aid. Its value in diagnosis of ventricular enlargement has increased as the science has developed. Today accurate evaluation of ventricular enlargement can be made early and it is possible to differentiate whether the right or left ventricle is involved.

The former statement is borne out by case 89034 at the University of Nebraska Hospital. Here the heart showed no signs of enlargement by X ray, but in the presense of an electrical axis of -60°, and a flat T_1 with

no deviation of the S-T segment, definite signs of left heart strain were demonstrated on the electrocardiogram. This diagnosis was borne out by the finding of a heart that weighed 400 grams with a left ventricular wall that measured 2 cm. in thickness at autopsy.

The value in knowing the latter plays an important role in helping to determine the etiology of the ventricular enlargement. This is especially important in certain cases where auscultation presents a confusing picture of multiple murmurs. This was well brought out in case 88683. The patient had an old rheumatic fever history and was diagnosed on the ward as a mitral stenoses. Because he was not reacting to digitalis theropy, an electrocardiogram was run. Instead of finding signs of right heart strain and fibrillation as would be expected with a mitral stenoses the opposite picture was present. The electrical axis formed an angle of -30°, T_1 was flat, $S-T_1$ was depressed one millivolt and $S-T_3$ was elevated one millivolt. The rate was regular. Autopsy showed a dilated heart, with a calcified, typical fishmouth aortic valve and only minimal involvement of the mitral valve.

The prognostic value of the electrocardiogram in

ventricular enlargement is difficult to evaluate because it presents a picture showing only that such a condition exists. It does not tell you the degree of involvement. Therefore a single tracing only contributes to the prognoses when it is coupled with other laboratory tests and clinical observations. It is true that on an emperical basis tracings showing marked T wave changes indicate more extensive pathology and should carry a graver prognosis. Still, no correlation can be made between the tracing and the amount of pathology existing.

Serial tracings have definite prognostic value for they trace the progression of changes as the disease increases in severity.

The electrocardiogram plays an important role in checking improvement and recovery of cases of hypertension that respond to treatment. In these cases that respond to sympathectomy, thyocyanate treatment and so forth, the T waves in leads one and two can be seen to reverse their direction and even the direction of the electrical axis has been seen to revert back to normal limits. (7)

The only instance where the electrocardiogram fails to be of any importance in ventricular enlargement is in cases where an equal or near equal enlargement of both

ventricles exists. Here the changes of electrical axis cancel each other. T wave changes may exist and when they do the balance is in leads one and two.

A question often presented is whether or not the electrocardiogram is of any significance in determining whether enlargement is due to dilatation. Hartog and von Niewenhuezen (45) maintain the electrical axis changes are due to dilatation, while hypertrophy causes S-T segment and T wave abnormalities. Their opinion is based upon the fact that left axis deviation is most common in old age groups and this group of people show a greater incidence of dilatation. Also, they claim left axis deviation is less common in hypertensive patients. This is opposite from the findings of Smithwick and Associates and from information gained from the files of the Cardiology Department of the University of Nebraska Hospital. In both cases one of the earliest signs of hypertension noted was a shift of the electrical axis. The former group has proven this by demonstrating that the electrical axis as well as the T waves and S-T segments may reverse their direction after sympathectomy.

The confusion arising over this point lies in the

basic fact causing axis change--relative rotation of the heart on its longitudinal axis and a shift of time relationship of the currents at each electrode. These changes can be caused by either dilatation or hypertrophy or both. The differentiation then lies upon facts obtained from the patient's history and other clinical data, rather than upon electrocardiogram.

Summary

The electrocardiogram is of diagnostic importance in ventricular enlargement. Accurate interpretation can readily determine certain changes that have been proven by autopsy to be characteristic. These changes are an abnormal shift of the electrical axis, depression to inversion of the T waves and elevation or depression of the S-T segments in specific leads.

It is possible to determine, with accuracy, whether the right ventricle or left ventricle is involved. This accuracy increases in proportion to the degree of dissociation of the weight ratios of the two ventricles.

In diagnosis of ventricular enlargement the position of the heart must be determined because a marked verticle or horizontal position may distort or confuse the evaluation.

The significance of the electrocardiogram in ventricular enlargement lies in its value in early recognition and helping to establish a diagnosis as well as to follow what progress is being made in the therapy of hypertension.

Conclusions

1. The electrocardiogram has been proven to be of a clinical aid in diagnosis of ventricular enlargement.

2. It demonstrates that ventricular enlargement is present, but at this time no direct correlation can be made between the curve recorded and the amount of pathology.

3. The electrocardiogram is of no practical significance in determining whether the strain is due to dilatation, hypertropyy or both.

4. The relative position of the heart may be determined by correlating findings of the limb leads with the precordial leads. This is significant because it alleviates one of the greatest sources of confusion in the diagnosis of ventricular enlargement.

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