The Relationship between the Anatomic Position of the Heart and the Electrocardiogram

A Criticism of "Unipolar" Electrocardiography

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Using a method for identifying precisely the lie of the ventricular structures in the body at autopsy, the anatomic position of the heart was compared with the directions of the QRS electrical forces in a large number of subjects with and without heart disease. While the mean QRS axis varied through 180 degrees in these subjects, the anatomic position of the left ventricle varied less than 45 degrees. No instances of significant rotation of the heart around its long axis were encountered, and it is shown that the unipolar electrocardiographic criteria for position and rotation of the heart have little validity. An explanation for the axis deviations of ventricular hypertrophy is offered.

That the form of the QRS complex is influenced by the position of the heart in the chest has been known since Einthoven demonstrated changes in the direction of the mean QRS axis with respiration. Later, the introduction of unipolar precordial leads made it possible to study the relationship between the electrocardiogram and position of the heart in three-dimensional space, and Wilson and co-workers devised a method for recognizing five different heart positions from combinations of QRS complexes in the unipolar limb and precordial leads. Subsequent workers have greatly extended this method in order to define in more detail the position of the heart and its components from the electrocardiogram.

Wilson's method was the outgrowth of a particular hypothesis regarding the factors which govern the form of the QRS complex. On the basis of experiments performed by Lewis and in his own laboratory, Wilson concluded that the unipolar lead might be considered to resemble a "direct lead," that is, one placed directly on the heart and recording principally the electrical events of the portion of the heart underlying it. This hypothesis suggested that when a unipolar electrode faced a particular region of the heart it would write a QRS complex with a specific contour for that region, and there would be specific QRS patterns for many different regions of the heart. Thus, when a particular QRS contour was seen in one or another unipolar lead, it could be concluded that the region of the heart known to yield that QRS pattern must be facing that electrode location. This hypothesis has been extensively applied to clinical electrocardiography and detailed systems have been devised for detecting various types and degrees of rotation of the heart, the lie of the septum, anterior and posterior tilting of the apex, the location of sites of infarction in the left ventricle, and similar features from the contours of QRS complexes in the various unipolar limb and precordial leads.

In its clinical application this approach has become known as the "unipolar method" for electrocardiographic interpretation. However, although widely accepted, it has some serious shortcomings. The most important of these is that Wilson recognized, that, only an extremely brief portion of the unipolar lead QRS complex, the "intrinsic deflection," can be considered to be written by the underlying region of the heart. And in a body surface lead even this is written to only a limited extent by the underlying portion of the heart, which is why

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in clinical practice it is called the "intrinsicoid deflection." The portions of the complex before and after the instantaneous intrinsicoid deflection are a much larger portion of the complex and represent the electrical activity of all the other regions of the heart as they undergo activation. To this extent a unipolar lead is not a direct lead registering the electrical events of a single region of the heart, and it was for these reasons that Wilson called it a "semi-direct" lead. The same caution was used in describing the five positions of the heart, for he termed them "electrical positions" of the heart and pointed out that they might or might not be related to the actual anatomic position of the heart.

These uncertainties regarding the validity of the clinical application of this hypothesis have tended to be justified by subsequent experimental studies. These have shown that, for clinical purposes, the contour of the unipolar lead QRS complex is more accurately and more rationally treated by considering it written by all regions of the heart rather than principally by the region facing the electrode. These studies, which are discussed elsewhere, indicate that the validity of the "unipolar" method of electrocardiographic interpretation is open to question.

There is also an important practical shortcoming in the "unipolar" approach to the clinical electrocardiogram. The body surface QRS patterns for the various specific regions of the heart and the electrocardiographic criteria for position and rotation of the heart have been based largely upon conjecture, and there have been no careful anatomic studies in the human subject to test the validity of these criteria. Even the notion that the heart rotates on its long axis with one or another type of ventricular hypertrophy, a basic concept in electrocardiographic as well as x-ray and cardiologic literature, is unproved, for there have been no systematic anatomic studies demonstrating that such rotation occurs. The following studies were undertaken to examine the extent to which the electrocardiogram reflects the anatomic properties of the heart. A method was devised for accurately measuring the position and rotation of the heart at post-mortem examination and this information was compared with the electrocardiographic data obtained shortly before death in a large number of subjects with and without heart disease.

**METHODS AND RESULTS**

Twenty-four consecutive cases were studied. Seven of the patients died with clinically manifest hypertensive cardiovascular disease. Two subjects died of mitral stenosis and insufficiency, one of cor pulmonale due to pulmonary emphysema, one of aortic stenosis due to rheumatic heart disease, one of syphilitic aortic insufficiency, and two of arteriosclerotic heart disease. The remaining 10 subjects had no recognized heart disease clinically or at autopsy. Patients with clinical, electrocardiographic, or pathologic evidence of myocardial infarction were excluded from this series as were also those with a QRS interval in excess of .12 second.

Bipolar and unipolar limb leads and precordial V leads were recorded on the Sanborn Visocardiette with the patient in the recumbent position. In all subjects an electrocardiogram had been recorded within three days of death and at least three electrocardiograms were obtained from all subjects during the terminal illness. The tracings were analyzed for the direction of the mean spatial QRS vector, the mean spatial vector for the first .02 second of the QRS interval, and the frontal plane projection of the QRS loop. The method for determining the directions of the spatial vectors and the QRS loops from the conventional electrocardiogram have been described elsewhere.

In accurately defining the anatomic location of the various regions of the heart at autopsy, the following method was used. After removal of the sternal plate and before intrathoracic contents were disturbed, the pericardium was carefully opened and a thin, sharply pointed metal spear was gently thrust anterior-posteriorly through the heart, perpendicular to the autopsy table. Then, another spear was passed from left to right through the chest wall and through the ventricular mass to be parallel with the frontal plane of the body and at right angles to the anterior-posterior spear. The heart was now removed by severing the great vessels. The auricles and the right ventricle were gently packed with formalin-soaked gauze to restore the contour of the heart, and a third spear was inserted perpendicular to the plane defined by the first two, to be parallel with the long axis of the body. The impaled heart was then mounted on a frame to support the spears in their proper rectangular relationships and the whole placed in a jar containing 10 per cent formalin. In certain instances the coronary vessels were injected with formalin to improve fixation.

After 36 to 48 hours of fixation, photographs were made of the anterior, inferior (or diaphragmatic) and lateral surfaces of the intact heart, using
the three spears as precise reference guides to define exactly how the heart lay within the chest at the time of death for each of these views. After these photographs were taken and without disturbing the spears, the auricles, great vessels, epicardial fat and connective tissue, coronary vessels, and valves were carefully removed leaving only the ventricular muscle mass. Photographs were again taken of the heart at this stage of dissection using the spears as reference axes for the three views. Next, the free wall of the right ventricle was cut away where it joins the septum, leaving only the left ventricle and septum; and photographs of the three views were taken at this stage of dissection, using the spears to obtain exactly the same positions as at the earlier stages of dissection. From the photographs at the third stage of dissection it was possible to determine the exact lie of the left ventricle and the interventricular septum in the body at the time of death. From these two measurements the type and degree of rotation of the heart on its long axis, transverse axis and anterior-posterior axis could be precisely determined. The various parts of the heart were weighed and measured at each stage of dissection. Eleven hearts were studied by this method, nine of which are illustrated. In the remaining cases the lie of the left ventricle and the septum were determined by making serial sections through the heart parallel with the transverse plane as defined by the spears.

In some cases the dissection was greatly facilitated by immersing the heart in slightly acidified water and bringing it to a boil. The epicardial fat and connective tissue were much more easily removed.
following this treatment giving a much "cleaner" specimen and one which lent itself more satisfactorily to dehydrating and clearing or "mummifying" for the dissolution of only intracardiac fat and connective tissue is unlikely.

Figure 1 shows the photographs in one case. The demonstration purposes. However, it reduced the weight of the ventricular mass by as much as 30 per cent and reduced some of the measurements by 10 to 20 per cent. That this weight loss represented upper three photographs show anterior views of the heart at the three stages of dissection: first, of the intact heart; second, of the ventricular muscle mass; and, third, of the isolated left ventricle and septum.
The second row of photographs show views of the inferior or diaphragmatic surface of the heart at the same three stages of dissection; and the bottom row shows views of the lateral surface (looked at from the patient’s right side) at the same three stages of dissection.

The arrow drawn in the photograph of the left ventricle indicates the direction of the long axis of this ventricle. On the triaxial system below each series of drawings are plotted the mean spatial QRS vector and the frontal plane projection of the QRS loop with .02 and .04 second markers. In addition, the spatial directions of the anatomic long axis of the left ventricle and the long axis of the septum (which was selected as the line from the septal leaflet of the tricuspid valve to the apex of the right ventricle) are drawn on the triaxial system to show the relationship between the directions of the electrical forces and the lie of the heart. The duration of the QRS interval for the given case is indicated in parenthesis.

Only the anterior views of the heart at the three stages of dissection are shown in the illustrations for the other cases (figs. 2, 3, 4). Pen and ink tracings were made from the photographs in order to facilitate reproduction. Since the focal distance was not the same for each heart, the scale of one inch is shown in the upper left drawing in each case. The age, sex, height, weight, postmortem diagnosis, and weights of the heart at various stages of dissection* are recorded beside the triaxial figure. The age, sex, height, weight, postmortem diagnosis, and weights of the heart at various stages of dissection* are recorded.

* The weight of the dissected ventricle is a much more accurate index of the presence and degree of ventricular hypertrophy than is the total weight of the heart. As has been known by pathologists since Muller's classic dissections in 1883, the nonventricular portions of the heart (auricles, epicardial fat, and connective tissue) may represent from 20 to 60 per cent of the total weight of the heart. Average normal weights are: left ventricle and septum, 140 to 180 Gm., free wall of the right ventricle 30 to 50 Gm.
at the lower right side of each figure. Nine cases were selected from the series for illustration. Of the seven cases shown in figures 1, 2, 3 and 4, cases 2162, 2166, 2177, and 2180 represent left ventricular hypertrophy, 2184 represents right ventricular hypertrophy, 2187 is an instance of arteriosclerotic heart failure with little or no ventricular hypertrophy, and 2167 is a normal heart. A relatively large number of cases of left ventricular hypertrophy

QRs cycle, (3) the long axis of the left ventricle, and (4) the long axis of the septum are shown projected on the frontal plane of the body. It can be seen that there were wide variations in direction of the mean QRS vectors, from marked left axis deviation to definite right axis deviation. However, while the direc-

![Diagram of heart and QRS vectors]

are shown because of the wide variation in direction of the mean QRS vector in these cases.

In figure 5 are shown the relevant anatomic and electrocardiographic findings in all cases studied. The range of variation in direction of (1) the mean QRS vector, (2) the mean .02 second vector, which is the mean vector for the earliest electrical forces generated during the

![Diagram of heart and QRS vectors]

...ration of the mean QRS vector varied through 180 degrees, the long axis of the left ventricle varied in direction less than 45 degrees. The .02 vector varied less than the mean QRS vector in direction, and the long axis of the septum varied less than 30 degrees in direction. The implications of these findings will be discussed shortly.

Fig. 4. See text and legend for figure 2.
To measure the rotations of the heart around its long axis, the direction of the axis of the left ventricle was compared with the direction of the septal axis in each case. It was found that these two axes had relatively the same relationship to one another in all cases studied. Thus, although the series included instances of right and left ventricular hypertrophy as well as normal hearts, no instances of significant clockwise or counterclockwise rotation were encountered. This can be seen in the illustrations, for it will be noted that the septum has nearly exactly the same relationship to the body of the left ventricle in all views in all subjects.

Fig. 5. (A) The range in direction of the mean QRS vector and the mean Q2 second vector in the cases studied. (B) The range in direction of the long axis of the left ventricle and of the septum in these cases.

Before discussing the electrocardiographic and anatomic implications of the findings, it is necessary to examine the validity of the method used. Obviously, the characteristics of the heart at post mortem are not identical with those in the living subject. The dying human heart is believed to cease beating in systole, yet the QRS electrical forces, which are the basis of the present study, are written during cardiac diastole. Furthermore, Friedman has shown that the heart is more completely emptied at death than it is by systole in the living subject, and that the volume of the heart (the tissue mass plus the contained blood) as studied by three-dimensional x-ray methods may be as much as 50 per cent less at death than it was in the living state. 13

Accordingly, the over-all size of the hearts shown in the illustrations is not the same as is obtained in the living state when QRS forces were generated. However, the general position of the heart in the chest postmortem examination was probably very nearly the same during life, and the relationships of the various parts of the ventricles to one another shown in the illustrations were unquestionably the same during life. Furthermore, since in all hearts the positions of the right and left ventricle relative to one another and to the three body axes were remarkably similar at postmortem examination, in spite of wide variations in heart size, it is probable that these positions were also remarkably similar during life.

Discussion

These studies indicate that the QRS axis deviations commonly seen with ventricular hypertrophy are not due to anatomic rotation of the heart. Indeed, the lie of the left and right ventricles in the body was remarkably similar in all hearts, both normal and hypertrophied. To be sure, among normal subjects, those with a more horizontal left ventricle tended to have a QRS axis (or “mean QRS vector” in the nomenclature of vector electrocardiography) which was horizontal to about the same degree. However, in the presence of left ventricular hypertrophy the electrical axis was often markedly rotated leftward while the anatomic position of the left ventricle in these cases was essentially the same as in the normal heart.

Furthermore, in all hearts, both normal and hypertrophied, the long axes of the septum and left ventricle had essentially the same directions relative to one another and to the body axes. Thus, there was no significant clockwise or counterclockwise rotation of the heart in any of the 24 hearts studied. The septum did not rotate with the development of ventricular hypertrophy, and there was little variation in the degree of anterior tilt of the long axis of the heart, as can be seen in the illustrations. The detection of rotation of the heart on its long axis has been a prominent part of the unipolar method of electrocardiographic interpretation, with, in some instances, a rotation of over 90 degrees inferred from the electrocardiogram. From the present studies it is apparent that such rotation rarely if ever occurs, and the “unipolar” electrocardiographic...
criteria for position and rotation of the heart have little validity.

The notion that the heart rotates in one or another direction in various heart diseases seems to have arisen principally in electrocardiographic literature, for no basis for it has been found in anatomic literature. Evidently it arose as the simplest way to explain the rather marked changes in direction of the mean QRS axis with ventricular hypertrophy and from an imperfect picture of the structure of the heart. Although anatomists had clearly defined the relationships of the various parts of the heart to one another, they had not been concerned with the relationship of these structures to various regions of the body surface.

The usual diagram of the ventricular heart in electrocardiographic and other cardiologic literature is of two chambers lying side by side, separated by an anterior-posterior partition, the septum, with the outflow orifices of the two chambers facing superiorly. Schematized in this way as a vertically pedunculated structure, it is easy to see how the idea could arise that the heart might rotate with hypertrophy of one or the other ventricle.

Actually, however, as can be seen in these illustrations, the left ventricle is a conical structure which in the supine subject lies relatively horizontally in the body. Its inflow and outflow orifices face the right side of the body. The septum is structurally a part of the left ventricle, representing that part of the left ventricle to which the free wall of the right ventricle is attached. The septum does not extend anterior-posteriorly but instead is relatively parallel with the frontal plane, the right ventricle lying altogether anteriorly to the left ventricle. The remainder of the left ventricle, the “free wall,” which is considered in electrocardiographic literature to be a lateral structure facing the left side of the body, actually includes nearly 300 degrees of the circumference of the left ventricle, and faces superiorly, posteriorly and inferiorly, a fairly large portion of it resting on the diaphragm.

The inflow and outflow orifices of both ventricles communicate quite directly with relatively fixed intrathoracic structures. While in the left ventricle the inflow and outflow tracts are relatively parallel with one another, in the right ventricle they form a right angle (fig. 6). Thus, the right ventricle lies across the left ventricle, embracing it at its base, and anchoring the heart by its superior and inferior attachments. When these anatomic features are considered, it can be seen that the heart tends to be pyramidal with the base of the pyramid facing the right side of the body and the apices of this base firmly attached to the other intrathoracic structures. Thus, while the heart may shift superiorly or inferiorly, it is prevented from rotation on its long axis by
the fixation of the base. Then, as these studies have shown, when right or left ventricular hypertrophy develops, the involved ventricle simply increases in thickness and length in this same relationship to other parts of the heart.

Since anatomic rotation does not account for the marked axis deviation of left ventricular hypertrophy, what is its explanation? In order to answer this, it is first necessary to review the factors governing the sequence in which the various parts of the heart are excited. During the first .03 to .04 second of the QRS interval, endocardial surfaces of the right and left ventricles undergo activation. Since these surfaces are not changed in position by hypertrophy, the vectors during this early part of the QRS interval should be relatively normally directed in the presence of ventricular hypertrophy. This proved to be the case in the present series, for the direction of the mean .02 vector varied much less than did that of the mean QRS vector and had a more fixed relationship with the lie of the heart in all cases.

The forces after the first .03 second of the QRS interval are effectively generated from the epicardial surfaces of the ventricles and tend to be directed perpendicularly to the generating surface. Since the epicardial area is greater than the endocardial area, these vectors are much greater in magnitude than the endocardial vectors. Furthermore, the epicardial forces from the right ventricle are normally smaller in magnitude and are generated earlier than those of the left ventricle. Accordingly, the direction of the mean QRS vector in the normal subject and in the subject with left ventricular hypertrophy depends principally upon the characteristics of epicardial depolarization of the left ventricle. There are two factors which determine these characteristics: (1) The conduction system, that is the sequence in which the excitation process is distributed to the various regions of the left ventricle, and (2) the thickness of the wall of the left ventricle, for it takes approximately 10 times longer for excitation to traverse the wall than to spread an equal distance along the conduction pathways of the endocardium.

The characteristics of left ventricular conduction have been recently reviewed by Murray. For present purposes it need only be pointed out that left ventricular excitation extends first down the endocardial surface of the anterior and diaphragmatic walls of the left ventricle toward the apex, and then spreads toward the base on the superior and posterior endocardial surfaces, ending usually at the remotest posterobasal region of the free wall of this ventricle. With normal wall thickness, epicardial excitation tends to follow this same sequence, but more slowly. The terminal QRS vectors in the normal subject tend to be resultants of vectors from the last epicardial regions to be depolarized, and therefore these terminal forces tend to be directed posteriorly and somewhat leftward. This is the explanation for the direction of the mean QRS vector in the normal subject.

To be sure minor variations in this sequence of left ventricular depolarization are extremely common and, indeed, the fact that everyone has a slightly different QRS loop is probably primarily due to the fact that this sequence of endocardial and epicardial depolarization varies slightly from person to person. When the loop becomes prolonged in duration or abnormal in contour it is often possible to determine the location of the region of disturbed intraventricular conduction by studying the direction of the initial QRS forces. And, since the epicardial vectors tend to be directed perpendicularly to the surface where they are generated, the direction of terminal vectors will also reveal the delayed region, for, when plotted from the electrocardiogram as vectors, they tend to point toward the blocked region.

Increases in left ventricular wall thickness as in left ventricular hypertrophy delay the transmyocardial progression of excitation. This tends to extend the period of epicardial excitation and more clearly separate in time the vectors generated from the very last region of the left ventricle from those generated from more intermediate regions. Because of this the terminal vectors are more exclusively contributed from the superior and posterior regions of the left ventricle in the hypertrophied than in the normal heart, and hence have a more superior and posterior direction than
normally. In addition, because of the increased surface area which accompanies the hypertrophy, the vectors generated from the hypertrophied region are increased in magnitude. Accordingly, in left ventricular hypertrophy the terminal QRS vectors are more leftward and posterior in direction and are greater in magnitude than in the normal subject, even though the heart has essentially the same anatomic position in the two instances. This then is the explanation for the marked leftward QRS axis in left ventricular hypertrophy, with present views of ventricular excitation.

There are other factors which, no doubt, contribute to the leftward direction of mean QRS vector in left ventricular hypertrophy. The anatomic studies of the hearts in this series of cases have shown that with hypertrophy the diameter and length of the wall of the left ventricle are increased, but the diameter of the mitral-aortic orifice usually remains normal. This causes the contour of the free wall of the left ventricle to become considerably more bowed superiorly and posteriorly than normally (fig. 7). As a consequence, the epicardial surface of the base of the left ventricle comes to face more superiorly, and, in some cases, even rightward. This also causes the terminal vectors of the left ventricle to be directed more superiorly and posteriorly than in the normal subject.

Still another factor has been suggested by Wilson. The wave of excitation travels much faster along the endocardial surface than it does across the wall of the left ventricle. When the wall is increased in thickness, this difference in rate of movement causes the wave of excitation advancing across the myocardial wall to become more and more oblique to the surface of the heart. As a consequence, the epicardial QRS vectors tend to be oblique to the surface rather than perpendicular as in the normal subject. This would cause the direction of mean QRS vectors to be more leftward and posterior in the presence of left ventricular hypertrophy than in the normal heart.

Not all hearts with left ventricular hypertrophy show a left axis deviation. For example, in case 2180 the mean QRS vector is somewhat vertical in direction in the frontal plane, in 2170 and 2206 it is horizontal, while in 2166 and 2162 it is markedly leftward. Yet the left ventricle had essentially the same position in all these cases, and no systematic feature of wall thickness, length of inflow or outflow tract, or left ventricular weight could be identified to account for this variation. It has been suggested that a normally directed QRS axis in the presence of ventricular hypertrophy indi-

![Fig. 7. Comparison of a normal and an hypertrophied left ventricle, right lateral view above, inferior or diaphragmatic view below. The free wall of the right ventricle has been removed. Larger heart: case 2227, mitral stenosis and aortic insufficiency due to chronic rheumatic fever; weight of the left ventricle and septum: 360 Gm. Smaller heart: case 2209, bronchogenic carcinoma; weight of the left ventricle and septum: 130 Gm.](http://circ.ahajournals.org/)

icates the presence of combined right and left ventricular hypertrophy. This was not the case in the present series as can be seen by comparing the right ventricular weights. It must be concluded that the mean QRS vector is vertical or horizontal in these cases because of anomalous intraventricular conduction. Perhaps delay in the posterior subdivisions of the left bundle branch is responsible for the relatively vertical axis in some of these cases.
When left ventricular hypertrophy is marked, the directions of the early QRS forces are often changed, becoming more or less parallel with the mean QRS vector (and manifested by absence of an initial R wave in V1 and V2 and lead III). Although no instance of this type with a QRS interval measuring less than .12 second was encountered in the present series, there are two possible explanations for this abnormality, either of which may be responsible in a given case: (1) it may represent selective delay in the anterior divisions of the left bundle branch,16 or, (2) it may represent the presence of significant dilatation in addition to the hypertrophy. The anatomic studies done in connection with these series of cases have confirmed the observation of Kirch that in left ventricular dilatation the inflow tract and superior wall of the left ventricle may often become more markedly stretched and bowed than the outflow tract and inferior wall. Under these circumstances the vectors generated from the endocardium of the inflow tract tend to be increased in magnitude. The resultant vectors during the first .03 second of the QRS loop would be dominated by these larger posteriorly directed vectors and point leftward and posteriorly, thus producing Q waves in V1, V2 and V3.

There was a single instance of right ventricular hypertrophy in the present series, a case of cor pulmonale due to pulmonary emphysema (case 2184), and the mean QRS vector was relatively vertical in this case. Although the descent of the diaphragms in pulmonary emphysema may often cause the heart to rotate somewhat rightward producing right axis deviation, this proved not to be the case in the present instance. As can be seen in the illustration, the right ventricle became more prominent but did not produce significant rotation of the heart as a whole. Accordingly, the vertical axis in this case must be due, not to rotation of the heart, but to the larger epicardial surface area and increased wall thickness of the right ventricle. This would cause the epicardial vectors from the right ventricle to be increased in magnitude and slightly delayed in time, causing the mean QRS vector to have a more rightward direction than normally. That the mean QRS vector was directed inferiorly rather than anteriorly may be due to the fact that the inflow tract of the right ventricle was more hypertrophied than the outflow tract.21 The measurements of the right ventricle in this heart indicated that while the length of the inflow and outflow tracts were about equally increased, the wall of the inflow tract was significantly more increased in thickness than was the wall of the outflow tract. As can be seen in the protocol in the illustration, the free wall of the right ventricle was nearly three times normal weight in this case.

There are two other aspects of these studies which are of electrocardiographic interest. One of these concerns the electrical forces generated by the septum. It has been widely accepted that the earliest region of the ventricular myocardium from which QRS forces are generated is the interventricular septum, producing a force traveling from the left to the right side of the septum.7 22 If this force were perpendicular to the septum it would be significantly different in direction from the remainder of the resultant forces during the QRS interval. On this supposition, the appearance of Q waves in leads on the left lateral chest have been attributed to a septal electrical force, and variations in the distribution of these Q waves in the various unipolar leads have been used to identify rotation of the septum.

In the present study, it was found that the septum has very nearly the same position in the chest in all hearts, both normal and abnormal as can be seen in the illustrations. It is relatively parallel with the frontal plane, its apex somewhat anterior to its base, and in none of the 24 cases studied was significant rotation of the septum encountered. When the lie of the septum was compared with the direction of the mean force for the first .02 second of the QRS interval, it was found that this vector tended to be more tangential than perpendicular to the septum (fig. 5). Thus, there appears to be little validity in the notion of a “septal Q wave.” As others have suggested, if there is a measurable QRS force due to exclusive septal activation, it is likely that it is
of considerably shorter duration than .02 and may be too small in magnitude to be recorded from body surface leads.14

Another implication of these anatomic studies concerns the nomenclature of myocardial infarctions. In electrocardiography, "posterior" myocardial infarction was so designated because of the notion that the septum extended anterior-posteriorly, defining an anterior and posterior region of the left ventricle. From this reasoning, infarcts of the left ventricle adjacent to the "posterior" attachment of the septum were called "posterior" infarcts. However, as can be seen in the illustrations and as has been long recognized by anatomists, the interventricular septum is not an anterior-posterior structure, but more nearly lies parallel with the frontal plane. The sulcus for the descending branch of the right coronary artery and the adjacent region of the left ventricle where "posterior" infarcts occur, rests on the diaphragm. Hence infarcts in this region are more accurately termed "diaphragmatic" or "inferior" than posterior.

The notion that "posterior" infarcts lie on the anatomically posterior wall of the left ventricle has been a source of confusion in clinical electrocardiography as well. The QRS and T wave deformities of myocardial infarction are due to abnormal QRS and T forces pointing away from the location of the infarct.9, 18 Therefore, if a "posterior" infarct were actually due to a lesion on the anatomically posterior wall of the heart, the QRS and T abnormalities should be most conspicuous in the precordial leads, for these leads effectively measure anterior-posterior forces, while the limb leads measure forces parallel to the frontal plane of the body. Actually, however, the most conspicuous QRS and T abnormalities of what is called "posterior" infarction are seen in the limb leads (Q2, T3, with Q waves in leads II and V1, and relatively normal precordial leads in most cases). When the directions of the abnormal QRS and T forces of such "posterior" myocardial infarctions are measured, they prove to point away from the diaphragmatic and not the posterior region of the heart. Thus, the electrocardiographic evidence confirms the point of view expressed in the previous paragraph, that "posterior" myocardial infarctions actually lie in the diaphragmatic and not in the anatomically posterior wall of the left ventricle.

**Summary**

1. A method was devised for accurately defining the postmortem position of the left ventricle and the interventricular septum in terms of three body axes. These anatomic findings were compared with the characteristics of the mean QRS and mean .02 second vectors of the premortem electrocardiogram in 24 subjects, 14 of whom had varying degrees of right or left ventricular hypertrophy and/or dilatation, and 10 of whom had no heart disease.

2. It was found that the left ventricle and the interventricular septum have remarkably similar positions in the body in all subjects regardless of the presence or type of heart disease, and no instances of significant clockwise or counterclockwise rotation of the heart were encountered. On the basis of these findings it is concluded that the criteria in "unipolar" electrocardiography for identifying the position and rotation of the heart and its components have little validity.

3. In view of the fact that rotation of the heart is not responsible for the QRS axis deviation of the ventricular hypertrophy an alternative explanation is offered which is consistent with prevailing concepts of ventricular excitation.

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**Summary Español**

La posición anatómica del corazón se comparó con las direcciones de las fuerzas eléctricas QRS en un número de sujetos con y sin enfermedad cardíaca, usando un método para precisar la localización de las estructuras ventriculares en el cuerpo durante la autopsia. Aunque el QRS promedio varió por 180° en
estos sujetos, la posición anatómica del ventrículo izquierdo varió menos de 45 grados. En ningún instante se encontró rotación significativa del corazón alrededor de su eje, y se demuestra que el criterio electrocardiográfico unipolar para posición y rotación del corazón tiene poco valor. Una explicación para las desviaciones del eje en hipertrofia ventricular se ofrece.

REFERENCES


16. Murray, R. H.: To be published.


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