CLINICAL VALUE OF UNIPOLAR CHEST AND LIMB LEADS

BY

C. W. CURTIS BAIN AND E. McV. REDFERN

From the Harrogate General Hospital

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Chest leads were first employed in myocardial infarction by Wood and Wolferth in 1932. Before that time their use was limited to the elucidation of the auricular arrhythmias. The original lead IV was an antero-posterior lead since it was hoped that this would register changes in a plane at right angles to the standard leads. In 1933 Wood and others found that the best results were obtained when the exploring electrode was placed either on or just internal to the apex, and they also used a lead in which the remote electrode was placed on the left leg. Later this apical lead came to be known as lead IV F, or IV R when the right arm was used as the remote electrode. In 1934 Wilson and others, seeking to reproduce as nearly as possible the same conditions as in animal experiments when the electrode can be placed directly on the epicardium, devised their central terminal method of obtaining a remote electrode approximately at zero potential. They called leads taken in this way V leads (V for voltage). They also chose six positions on the chest for the exploring electrode, extending from the fourth intercostal space to the right of the sternum (V 1) to the mid-axillary line at the level of the apex (V 6), as subsequently recommended by the American Heart Association (1938). These methods were not generally accepted at first, and leads IV R and F are still widely used. We have tried to determine what advantages may be gained from the use of multiple unipolar chest leads.

The principles underlying the unipolar method are based upon the equilateral triangle hypothesis which was propounded by Einthoven, Fahr, and de Waart in 1913. They stated that, having regard to the comparative remoteness of the extremities, the heart might be regarded as being in the centre of an equilateral triangle, and that, therefore, the algebraic sum of the potentials at the three points of the triangle at any given moment in the cardiac cycle was zero for all forces parallel to the plane of the triangle. So, if the three limbs were used as the remote electrode, instead of one, a remote electrode at zero potential would be obtained, and such a lead would be unipolar since it would record only the changes in potential of the precordial electrode. The Einthoven hypothesis is only applicable to forces parallel to the plane of the triangle, and the cardiac vector moves in three dimensions, but Wilson et al. (1944) have adduced considerable evidence to the effect that the perpendicular forces are small and do not exceed 0.3 mv. For practical purposes these leads can be considered to be unipolar.

UNIPOLAR AND BIPOLAR LEADS

Technique. The apparatus required to take V leads consists of three limb terminals which are brought together at a central terminal. The right arm electrode from the galvanometer is attached to the central terminal: the three limb terminals are attached to the limbs. The left arm electrode from the galvanometer is used in the ordinary way as the exploring electrode on the chest. Wilson et al. (1934) interposed resistances of 5000 ohms on each limb terminal, but Goldberger (1942) published curves taken with and without the resistances and they were identical. We have followed the Goldberger method and have not interposed resistances.

Unipolar Limb Leads. When using a unipolar technique it is possible to obtain the potentials at any point on the surface of the body. The original method of taking unipolar limb leads was to attach the exploring electrode on to the limb to be examined, having two electrodes on that limb. But the deflections by this method were sometimes small and difficult to measure. Goldberger (1942) introduced a modification that increased the size of the deflections by a half while their form was left unaltered. He substituted the exploring electrode for the V terminal on the limb to be examined, allowing that V terminal to hang loose. To take VR (the right arm unipolar lead) the exploring electrode is attached to the right arm and a V
terminal to the left arm and left leg. In taking VL (the left arm lead) and VF (the left leg lead) the exploring electrode is attached to the left arm, and the left leg respectively, with V terminals on the other two limbs.

Bipolar Leads.—In the standard leads the two points are connected and the galvanometer, which is interposed, records the difference in potential between the two points. When the two points are equidistant from the heart, the effect of each upon the cardiogram is approximately equal. In lead I the galvanometer is arranged—or the polarity is such—that a state of relative positivity at the left arm is represented by an upward movement of the fibre. Since it is the difference between the potentials at the two arms which is recorded in lead I, the potentials at the right arm must be subtracted algebraically from those at the left arm. Thus, if the T deflections at the right arm are −2 mm. (which equals a potential of −0.2 mv.) and are +1 mm. at the left arm, the deflections in lead I will be +3 mm. Since the potentials at the right arm are usually negative, the deflections in lead I will generally be more positive than at those at the left arm. This is the reason why an upright T is sometimes found in lead I in anterior infarcts although T is negative in lead VL, the left arm unipolar lead. In lead III a relative state of positivity at the left leg results in an upward movement of the fibre. Thus, if T at the left leg equals +2 mm. and is +1 mm. at the left arm, in lead III it will be +1 mm.

The chest leads CR and CF are also bipolar leads, but since the extremity, or remote, electrode is so much farther from the heart than the chest electrode, the influence it exercises is much less. Wilson (1944) has estimated that the size of the deflections at the precordia is from three to five times that at an extremity. The influence of the extremity electrode is, therefore, about one-quarter that of the chest electrode. But, when multiple chest leads are used, and the potentials at one point of the chest compared with those at another, any influence at all from the remote electrode is undesirable since it may distort the curve.

It has recently been suggested (Wallace and Grossman, 1946; Hoyos and Tomayo, 1947) that in practice the differences between CR, CF, and V leads are so slight as to be negligible. Since a CR lead equals approximately C−VR/4, and a CF lead equals C−VF/4, the distortion to be expected in any given case can be estimated if the VR and VF leads are available. If the Goldberger augmented method of obtaining the unipolar limb leads has been used, the deflections must be reduced by one-third: the equation then is CR (or CF)=C−VR (or VF)/6.

A series of 300 unipolar limb leads were examined with regard to this point. The T waves were flat in 49 cases in VR and 47 in VF. They were ±1 mm. in 111 and 117 respectively; ±2 in 62 an 65; ±3 in 44 and 40. Thus in 89 per cent the T deflections were 3 mm. or less, which should give a distortion of not more than 0.5 mm., and this is negligible. In the remaining 11 per cent, however, the distortion is appreciable. Fig. 1 was taken from a patient with mitral and aortic disease. Standard leads show left axis deviation; the T waves are

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**Fig. 1.**—V, CR, and CF leads showing effect of distortion on CR and CF leads from the right arm and left leg respectively. For details see text.
in health. In CR it is positive. T is negative in CF 1, 2, and 3, and just upright in CF 4. R waves are more positive and S waves less negative in CR leads than in V leads. This destroys the balance of the series. While the V leads show slight, but definite, left ventricular hypertrophy, the CR series is within normal limits.

Fig. 2 was taken from a patient recovering from an anterior infarct. The position of the heart was semi-vertical. Six months previously there was bowed inversion of T in leads V 1–V 4. Now V 1 and V 2 have negative T waves; T in V 3 is diphasic; T in V 4–V 6 is positive but not much so. In the CR series T is positive from 1–6. In the CF series it is negative from 1–6. The CR leads are, therefore, normal while the CF leads suggest an anterior infarct, and the difference between them is due to distortion from the right arm and left leg respectively.

Fig. 3 shows the type of distortion that may be expected in the different positions of the heart in CR and CF leads. In CR leads the distortion is positive in all positions of the heart except in very position possibly the most accurate of any. With horizontal hearts there is increasing positive distortion, but in general CF leads are good in all these positions. When the heart is vertical, however, large R waves and upright T waves are seen in VF leads, causing a negative distortion, which may lead to the recording of negative T waves in CF leads due only to the influence of the left leg. Nor is it possible to be sure that the heart does not lie vertically unless unipolar limb leads are taken, since standard curves may show left axis deviation in such cases if there is left ventricular hypertrophy, as in Fig. 1.

**Principles underlying Chest Lead Interpretation.**
Active heart muscle is electrically negative to inactive muscle. The impulse for contraction passes down the Purkinje tissue in the subendocardial zone and reaches the ventricular cavities almost at once. The ventricular cavities are, therefore, negative throughout the whole of the QRS. The impulse then spreads outwards through the ventricular muscle, and, as it does so, the muscle which has been activated will be negative, while in front of the
advancing head of the wave there will be a zone of positivity. This zone of positivity is reflected to the surface of body and causes the positive R to be the main initial deflection to be recorded in health. When the impulse reaches the surface of the ventricle, a negative charge is reflected to the surface of the body, and a negative downward deflection, called the intrinsic deflection by Lewis, occurs. Whether or not this downward deflection is prolonged into an S wave depends on whether that part of the heart muscle under the exploring electrode has been activated early or late in relation to other parts of the ventricular muscle. By the time the impulse reaches the surface of the right ventricle, the left ventricle will not be fully activated. The right ventricle will then become negative to the left and an S wave will be recorded in leads to the right of the precordia such as V1 or V2. When, however, the impulse reaches the epicardium over the thickest part of the left ventricle (lead V5 or V6), the whole heart will be in systole. No current flows, since the QRS phase is over, and the fibre comes to rest at zero potential, S being absent. As the electrode is moved over the precordia from right to left, an R followed by a deeper S in leads V1 and V2 gives place to an R without S in V5 and V6. Changes in this normal sequence of events enables the diagnosis of unilateral ventricular hypertrophy to be made, and the side of the lesion in bundle branch block to be determined.

Unipolar Limb Leads and the Position of the Heart. When the position of the heart is normal, the aorta and the pulmonary artery, as they arise, point upwards towards the right shoulder. Lead VR which enters the chest, so to speak, through the right shoulder will face these vessels and so reflect to a great extent the state of the ventricular cavities. Since these are negative throughout the whole of the QRS, lead VR, except in extreme rotation of the heart, has negative deflections. VL, the left arm lead, reflects the potentials on the anterior surface of the left ventricle, while VF, which enters the chest through the left dome of the diaphragm, reflects the potentials of the diaphragmatic or posterior surface of the left ventricle. When the position of the heart is normal, both these leads have positive deflections.

If the heart rotates clockwise, becoming more vertical, the aorta and pulmonary artery, as they arise, will tend to point directly upward or midway between the two shoulders. Lead VL will then become similar to VR, or to leads to the right of the precordia, and have negative deflections. An S wave in lead VL shows that the heart is vertical. When R and S are small and equal, the position is semi-vertical. A vertical position of the heart occurs in long narrow chests, in emphysema, and in right-sided hypertrophy.

If an anti-clockwise rotation occurs, the heart becomes horizontal. A horizontal position is found in left-sided hypertrophy or when the left diaphragm is high as in obesity or in sthenic types. An S wave then appears in lead VF, or, if the position is semi-horizontal, the deflections are small. For this there are two explanations. According to Wilson et al. (1944) the aorta, issuing more horizontally to the right, comes to face almost midway between the right shoulder and the diaphragm, and consequently lead VF will resemble VR, or V1 and V2, and have negative deflections. In this view if the
deflections in VF resemble those on the right side of the precordia (V1 and V2) while VL resembles those on the left (V5 and V6) the position of the heart is horizontal; if VF resembles V5 and V6, while VL is like V1 and V2, the heart is vertical. In this interpretation an S wave in lead VF means that the heart is horizontal except in certain cases of right branch block (see Fig. 11).

Goldberger (1944) in his explanation points out that the voltage of the negative potentials in each ventricular cavity will depend on the mass of muscle involved. Since the left ventricle has nearly always a greater mass than the right, the potentials in the cavity of the left ventricle will be more negative than those on the right, and, should the two sets of potentials oppose each other, the stronger potentials on the left side will overcome those on the right.

When the heart is normal in position, lead VF, which enters the chest through the left diaphragm, will face about equally the advancing wave in each ventricle. As a result the potentials will be positive. When the heart is placed vertically lead VF will face more of the left ventricle than of the right, and the potentials will become correspondingly more positive still. When, however, the heart is horizontal lead VF will face more of the right ventricle, but it will also face the negative potentials at the tail of the wave in the left ventricle which has now come to lie more superiorly. These stronger negative potentials from the left ventricle overcome the positive potentials in the head of the wave in the right ventricle, with the result that small but negative waves occur in lead VF. In this view an S wave in lead VF always means that the heart is horizontal.

**Present Series**

Cases with cardiac enlargement were specially selected at first since the intention was to try to establish more satisfactory criteria for unilateral ventricular hypertrophy than was afforded by axis deviation. Normal subjects of differing habitus

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**Fig. 4.**—Normal chest leads. (A) Heart in normal position. Chest leads show S to have twice the amplitude of R in V1 and V2 while S is diminutive in V5 and V6. VL and VF are positive. (B) Heart horizontal. Left axis deviation. Note S in VF. (C) Heart vertical. Right axis deviation. Note S in VL. T is inverted in V1 and V2.
were also examined to find the effect of varying positions of the heart upon the electrocardiogram. Later complete 12 lead electrocardiograms were taken on all patients with infarcts and bundle branch block.

NORMAL PRECORDBAL LEADS

In leads V1 and V2 an R wave is followed by an S wave of about double the amplitude (Fig. 4A). In leads to the left of the precordia (V5 and V6) an R only is seen, S being absent or diminutive. The point where the complexes change from a predominant S to a predominant R is called the transitional point, and is situated about the level of V3, which is placed approximately over the interventricular septum.

The T wave in V1 is frequently inverted in health, and this has no significance. It may also be inverted in V2 if the heart is placed vertically. In children inversion of T may also involve V3, but this is exceedingly rare in adults.

Normal precordial leads were found in 49 cases in whom ventricular hypertrophy was judged to be absent on clinical grounds.

In 33 cases the heart was normal. In these the heart was in normal position in 9 (Fig. 4A), horizontal or semi-horizontal in 9 (Fig. 4B), and vertical or semi-vertical in 15 (Fig. 4C). Both left and right axis deviation were frequent, the electrical axes varying from +110 to −70. The high proportion of abnormal positions was due in part to the selection of cases for the purpose of the investigation, but it is undoubtedly more common to find a vertical heart in a normal subject than a horizontal.

In 16 cases some clinical abnormality was present. In 9 there was moderate elevation of the blood pressure but no cardiac enlargement could be made out on screen examination. The remainder were made up of patients with angina pectoris (3), heart block (2), auricular fibrillation (1), goitre (1).

The position of the heart in these patients was normal in 11, horizontal in 4, and semi-vertical in 1.

LEFT VENTRICULAR HYPERTROPHY

There are five criteria of left ventricular hypertrophy (Fig. 5).

1. The R wave in leads to the right (V1, V2, and V3 and occasionally V4) becomes diminutive. We have adopted as a minimum requirement that the R wave should have an amplitude of no more than one quarter that of the S wave which should measure at least 12 mm. in any one of these leads.

2. Inverted T waves are seen in V4, V5, or V6. Similar changes occur in lead I, but not so frequently as in the chest leads. They must be distinguished from inversion due to digitalis (Fig. 6A).

3. The transitional point swings to the left. Minor changes in the transitional point are without significance. This criterion was judged to be present when the transitional point reached V4 or further to the left.

4. An increase in voltage of the deflections may be seen.

5. The QRS increases to 0:09 sec. or more.

Left ventricular hypertrophy was judged to be present on clinical grounds in 120 cases. Most of the patients had hypertension, and cardiac enlargement was seen on screen examination. Others had aortic disease. Congenital heart lesions were present in a few. Those showing bundle branch block or evidence of infarction are not included in this group.

Diminution in the R wave and increase in the S wave in leads over the right side. The R waves had an amplitude of not more than one quarter that of the S wave, which measured 12 mm. or more in either V1, 2, 3, or 4. The R waves were not present in V1 or V2, but the S waves were less than 12 mm.; 2 of these subsequently showed characteristic deep S waves. One other patient had kypho-scoliosis with considerable distortion of the chest. In the remaining five cases no cause could be found for the absence of the first criterion.
Taking the two criteria together, evidence of left ventricular hypertrophy was present in 112 of the 120 cases or 93 per cent. Excluding those who had received digitalis they were both present together in 30 cardiograms or 25 per cent.

Shift in the Transitional Point to V4 or further to the left. This occurred in 69 records, but it was also found in 14 patients in whom there was no clinical evidence of left-sided hypertrophy. More than any of the other criteria, the transitional point depends upon the correct positioning of the electrodes. A transitional point to the left of V4, which occurred in 26 cases is almost always evidence of considerable left ventricular hypertrophy, but other criteria will then be present. By itself a shift in the transitional point is unreliable, and is not acceptable alone as evidence of left-sided hypertrophy.

Increase in Voltage of the Complexes. In left-sided hypertrophy the voltage of the S wave in leads to the right and of the R wave in leads to the left may be very large. But this sign is variable, and depends upon many factors, which affect the
Fig. 7.—Right ventricular hypertrophy. (A) Diminutive primary R and primary S in V1, followed by large secondary R from the down-stroke of which arises the T wave. S waves in V4, V5, and V6. T is inverted in V1, V3, V4, V5, and V6. The heart is vertical. From a case of auricular septal defect. (B) Small Q waves in V1, V2, V3, and V4 followed by a large R. An S wave seen in V6. T is inverted from V1–V6 due to digitalis. The heart is vertical. From a patient with Lutembacher's disease and congestive failure.

Fig. 8.—Right ventricular hypertrophy. (A) Small primary R followed by large secondary R in V1 and V2. S waves appear in V3 and V4 and are diminutive in V5 and V6. T is inverted in V1, V2, V3, and V4. A digitalis effect is seen in lead II, III, V5, and V6. The heart is vertical. From a case of mitral and tricuspid disease with auricular fibrillation and congestive failure. (B) Small Q followed by large R with a small S in V1 only. T is inverted in V1. The heart is vertical. From a girl of 10 with recurrent rheumatic fever and mitral stenosis.
distance between the epicardium and the electrode. Anasarca, pulmonary edema, or a thick chest wall will tend to diminish the size of the deflections (Lapin, 1947). Dilatation of the ventricle, bringing the epicardium nearer to the chest wall, may increase them (Bayley, 1947). Large complexes may occur in health, and as a criterion of ventricular hypertrophy we have found it of no value.

Increase in the QRS Breadth. The QRS was increased to 0.09 second, or more, in 9 advanced cases only, where it was also prolonged in the standard leads (Fig. 5).

In 9 patients with left ventricular enlargement no criteria of hypertrophy were found in the chest leads. In one the record was taken during a paroxysm of auricular tachycardia. Although the R waves were absent in V1, the S waves were only 9 mm. Two days later the paroxysm had stopped and deep S waves were then present. Two more cases developed characteristic changes in a later record. Four had hypertension with slight to moderate cardiac enlargement, and no reason could be found why the chest leads were normal. In all, the R waves were small in leads V1 and V2, varying from 1 to 2 mm, but the S waves were from 9 to 11 mm. In two patients the chest leads were quite normal, but both had considerable displacement of the heart, one from an old empyema, one from kypho-scoliosis.

In 9 cases the first criterion was present without clinical evidence of left ventricular enlargement. In 5 of these some hypertrophy may in fact have been present. One was a young woman admitted to hospital with acute pulmonary edema, who gave a history of two similar attacks for which she had been kept in bed a month and 6 weeks respectively: her lung roots were prominent but the heart appeared to be normal in size. Another was a soldier with a history of acute nephritis three years previously and a relapse a month before when the blood urea was 72. The third had myxœdema with a B.M.R. of -35 per cent; the heart did not seem to be enlarged, but the pulsations were feeble. Two cases were seen in surgical wards and skigrams were not taken. The first had gangrene of a toe with moderate elevation of the blood pressure, and the second was very obese and had had a pulmonary embolism after the removal of an umbilical hernia.

Of the remaining 4 cases, one had angina pectoris; 2 had moderate hypertension, but the heart was not enlarged. In the last patient it is possible that an error in standardization may have been made. Coronary occlusion had been suspected but the pain was due to gall stones and the heart was normal. Two years later the chest leads were normal.

RIGHT VENTRICULAR HYPERTROPHY

The characteristic changes of right ventricular hypertrophy are seen in lead V1. The intrinsic deflection is delayed owing to the time taken by the impulse to reach the surface of the thickened right ventricle. A late R wave is seen. S is either absent or diminutive. There may be a small primary R followed by a primary S (Fig. 7A) or else a small Q (Fig. 7B). Sometimes these small primary deflections appear as a notch on the upstroke of R (Fig. 8A). These features may be limited to V1 (Fig. 8B) or may be seen also in V2, V3, and V4 (Fig. 7B). In leads to the left of the precordia S waves are usually seen, but there is no abrupt transitional point as occurs in left ventricular hypertrophy. The T waves may be inverted in any of the precordial leads.

Although the changes in V1 are characteristic, a considerable amount of right ventricular hypertrophy must be present before they appear, since they indicate that the thickness of the right ventricular wall approaches that of the left. The position of the heart is always vertical when the precordial leads show right ventricular hypertrophy.

In some cases where V1 was normal, evidence of right ventricular hypertrophy was found in lead V3R, in which the electrode is placed on a point midway between the right sternal border and the right mid-clavicular line (Myers, Klein, and Stofer, 1948).

Nineteen cases showed the changes of right ventricular hypertrophy: 10 had advanced mitral stenosis; 8 had congenital heart disease, comprising 6 cases of auricular septal defect, and 2 of pulmonary stenosis. One case with old standing Pott's disease is included since the R wave in V1 was greater than the S, but the curve was bizarre from the gross distortion of the chest.

Eight cases had clinical evidence of right ventricular hypertrophy but the precordial leads were normal: 3 of these had mitral stenosis; 4 had asthma or severe bronchitis; 1 had an auricular septal defect, but the heart was displaced to the left, and probably rotated, by scoliosis. In two patients with mitral stenosis, the heart was normal in position. In the remainder it was vertical.

BUNDLE BRANCH BLOCK

In bundle branch block the intrinsic deflection is delayed on the side of the lesion, but occurs early on the healthy side. The QRS has usually a slightly longer duration than in standard leads.

In left branch block R is diminutive or absent in V1 and V2 and a deep broad S wave occurs (Fig. 9A). In leads over the left side such as V5 and V6 a
large broad notched R is seen. The position of the transitional point is very variable and the R wave may not appear until V6 (Fig. 9B). This seems to happen particularly when the heart is vertical. Discordant types of standard leads occur when the heart is normal or horizontal: concordant when the heart is vertical. These terms no longer serve any purpose since they signify changes that are confined to the standard leads and are due merely to differing positions of the heart. Occasionally standard leads may suggest a right branch block, when the chest leads are characteristic of a left-sided lesion (Fig. 10).

Out of 17 cases of left branch block, the heart was horizontal in 14 (Fig. 9A) and was vertical in 3 (Fig. 9B).

In right branch block a broad notched R occurs in V1, and often in V2 as well (Fig. 11A). Occasionally the R is preceded by a small Q; more often there is a diminutive primary R with a succeeding S which is followed by a large secondary R (Fig. 11B). A deep Q in V1 and V2 in right branch block is due usually to the involvement of the septum in an antero-septal infarct, and will be described later. In leads to the left of the praecordia a slender R wave is followed by a broad S (Fig. 11B). The R wave is not small or absent as in leads over the right side in left branch block because the impulse takes longer to pass through the thicker left ventricular wall to reach the epicardium.

Some curves do not conform either to a right- or left-sided lesion. In these cases the disease is probably bilateral (Fig. 12).

Right branch block was present in 14 patients. Of these the heart was in a normal or horizontal position in 6 and in a vertical position in 8 according

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**Fig. 9.—Left bundle branch block. (A) Discordant. QRS 0·18 second. Small R with deep S in V1, V2, and V3. Large notched R in V5 and V6. Transitional point seen at V4. The heart is horizontal. (B) Concordant. QRS 0·12 second. V1, V2, V3 diminutive R waves with deep S. At V6 small but notched R: S absent. The heart is vertical.**

**Fig. 10.—Left bundle branch block. QRS 0·16 second. Standard leads suggest right branch block. V1, V2, V3 have small R waves with very deep S waves. V4-V5 are transitional. V6 small but notched R wave: S diminutive. The heart is vertical.**
In patients with right bundle branch block, the QRS complex is prolonged, and the R wave in V1 is often small. The S wave in V6 is usually prominent. The heart may appear vertical according to Wilson but horizontal according to Goldberger. In patients with left bundle branch block, the QRS complex is also prolonged, and the R wave in V6 is often small. The S wave in V1 is usually prominent. The heart may appear horizontal according to Wilson but vertical according to Goldberger.

**CARDIAC INFARCTION**

Since precordial leads face the part of the advancing wave that is activating the anterior surface of the left ventricle, they show characteristic changes only in anterior infarcts. In posterior infarction they face the tail of the wave and may have some depression of the S-T interval. Otherwise they are normal, although signs of left ventricular hypertrophy may be seen in cases of hypertension.

In anterior infarcts involving the whole thickness of the ventricular wall, deep QS waves are seen in the precordial leads. When the muscle is dead or

**FIG. 11.—Right bundle branch block.** (A) QRS 0.15 second. Complete heart block. V1 and V2 have a large notched R without an S; V5 and V6 a slender R followed by a broad S. The heart is vertical according to Wilson but horizontal according to Goldberger. (B) QRS 0.14 second. V1 has small R with S followed by large secondary R. V2 has notching of upstroke of R. V5 and V6 have tall but slender R followed by broad S. The heart is semi-horizontal.
irrespective there is nothing to prevent the initial negativity of the ventricular cavity passing straight through to the chest electrode. As Wilson et al. (1944) points out, it is as if a window had been cut in the ventricular wall. Elevation of the RS-T junction and sharply pointed negative T waves are also present. In antero-septal infarction the precordial leads chiefly affected are those to the right—V1, V2 and V3. In this type lead I is often normal. If the infarct involves the septum, the bundle branches may be cut. The combination of the changes due to anterior infarction and to right branch block gives a characteristic picture. If the left branch is cut, signs of infarction seldom appear.

This is partly because the large diphasic complexes of left branch block engulf the RS-T deviation and negative T waves, and partly because in left branch block the left ventricular cavity does not become negative until the impulse crosses the septum. Only if the whole thickness of the septum is involved in the infarct, will the negative potentials of the right ventricular cavity be transmitted through the dead muscle to the left, and allow Q waves to appear.

If the infarct is situated towards the lateral wall—the antero-lateral infarct—leads V4, V5, and V6 will show the maximal changes. In small sub-endocardial infarcts not involving the whole thickness of the wall, Q waves may be absent. Bowed
inversion of T may be the only evidence found. This must be distinguished from inversion due to hypertrophy or digitalis. The height of the R wave in the precordial leads may help. In health the R increases as the electrode is moved from right to left, and this tendency is more pronounced in left ventricular hypertrophy. A diminution in the height of the R as the electrode is moved to the left is valuable corroborative evidence of infarction. Or in some cases of left ventricular hypertrophy, the R wave may remain diminutive in V5 and V6 (Fig. 14). Occasionally the chest leads may be normal, and yet leads I and VL may be typical of infarction. In cases of this kind Wilson (1946) has found that the precordial lead changes were present at a higher level of the chest, and he places the electrode at the usual positions but along the third interspace.

**Extensive Anterior Infarction.** In extensive anterior infarction QS waves with elevation of the RS-T junction and deep inversion of T appear in all the chest leads (Fig. 15). In 7 cases of this type 2 died and 2 developed a cardiac aneurysm. In all T was inverted in lead VL and in all but 1 in lead I, when it was flat. In 3 cases T was negative in lead II as well as in lead I.

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**Fig. 14.**

Fig. 14.—Anterior infarction. Q waves, elevation of the RS-T junction and bowed inversion of T in leads I and VL. Chest leads show only left ventricular hypertrophy, but the R wave is unusually small in V5 and V6. The heart is horizontal.

**Fig. 15.**

Fig. 15.—Extensive anterior infarction. QS waves are seen from V1–V6, with bowed inversion of T.
Antero-septal Infarct. The changes affect especially leads to the right, such as V1, V2 and V3. T is upright in V6, and usually in V5 (Fig. 16). There were 10 cases of this type. In 8 there was bowed inversion of T in V1, V2, V3, and V4: in the others V3-V5 were affected. In 7 of these cases T was also just inverted in VL, but in two only was it inverted in lead I though in two more it was flat.

Antero-septal Infarct with Right Bundle Branch Block. When an antero-septal infarct involves the septum, the right branch may be cut. A deep Q is then seen in V1, V2, and V3 (Fig. 17A). This is followed by a late R, from the descending limb of which arises the bowed T wave, with considerable elevation of the RS-T junction. The QRS is widened. In leads to the left a slender R wave is seen and the T waves may be normal (Fig. 17B). Standard leads may show little more than widening of the QRS (Fig. 17A), though the T wave may be bowed in lead I.

There were 6 cases with this combination. In 5 there was complete right branch block, the duration of the QRS varying from 0-12 to 0-14 second. Three of these patients died; in another the block was temporary only, disappearing in a week. The right branch block was incomplete in 1 case, the QRS being 0-11 second (Fig. 16).

Antero- and Postero-lateral Infarct. Here the changes are seen in leads to the left of the praecordia.

**Fig. 16.** Antero-septal infarction with incomplete right branch block. Bowed inversion of T in V1, V2, and V3. The T wave in V4 and lead I is normal. QRS 0-11 second. Diminutive primary and secondary R waves in V1. The patient gave a history of recent short attacks of angina at rest.

**Fig. 17.** Antero-septal infarction with right bundle branch block. (A) Deep Q waves present in V1, V2, and V3 with elevation of the RS-T junction and bowed inversion of T from V1-V5. QRS 0-12 second. Delayed intrinsic deflection in V1, with slender R and broad S in V6. (B) Q waves present with inversion of T in V1, V2, and V3. QRS 0-14 second. V1 and V2 have broad R waves with a delayed intrinsic deflection. The R wave is small in V5 and V6. At autopsy the infarct was anterior and apical, and involved the upper part of the septum.
(V 5 and V 6) and also in lead I and in VL. There were 12 patients in this group of whom 6 had inversion of T in V 4, V 5, and V 6, and 6 in V 5 and V 6 only. In 11 of these 12 patients T was inverted in lead I and in VL: in the twelfth it was flat in both. In 6 of the 12 T was also inverted in lead II and in 5 it was inverted in VF, being flat in a sixth.

Posterior Infarct. In posterior infarction the counterpart of the precordial leads is the cesophageal lead since this lead faces the wave as it advances through the infarcted area. But lead VF also reflects the changes over the diaphragmatic or posterior surface of the left ventricle. Lead III is VF–VL, and VL faces the tail of the wave in posterior infarction. To the depth of Q in VF will be added in lead III the reversed R of VL; to the upward deviation of the RS–T junction, the reversed depression of the RS–T junction in VL; to the negative T of VF, the reversed positive T of VL. Lead III, therefore, always shows more pronounced changes than VF but in a sense these are spurious, being due to the subtraction of opposite values in VL (Fig. 18A).

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**Fig. 18.**—Posterior infarction. (A) Q waves and negative T waves in leads II, III, and VF. Chest leads are normal. (B) Chest leads show left ventricular hypertrophy, with deep QS in V 2.

**Fig. 19.**—Left ventricular hypertrophy with right axis deviation and a vertical heart. Concave depression of S–T interval due to digitalis seen in leads I, II, III, VF, and V 6. Convex elevation of S–T interval seen in leads VR, VL, V 1, V 2, V 3, and V 4. From a patient with mitral stenosis, aortic incompetence, hypertension, auricular fibrillation, and congestive failure.
Precordial leads may show some depression of the RS-T junction. Sometimes the infarct may encroach upon the lateral wall of the left ventricle, and inversion of T may be present in V6. In other cases inversion of T in the precordial leads may be due to left ventricular hypertrophy, or to a previous anterior infarct.

There were only 8 patients in this group. All had Q waves and deep inversion of T in VF as well as in lead III, and to a less degree in lead II. In one case T was inverted in V6. One patient with considerable hypertension and another with aortic incompetence had evidence of left ventricular hypertrophy in the chest leads (Fig. 18B). Inversion of T in V4, V5, and V6 in one case was probably due to a previous anterior infarct.

Congenital Heart Disease

In a small miscellaneous group of congenital heart lesions comprising cases of dextrocardia (4), pulmonary stenosis (1), and aortic stenosis (1), the curves were either bizarre or within normal limits.

Effect of Digitalis

Digitalis causes the same depression of the RS-T junction, and negative T waves, in leads to the left of the precordia such as V5 and V6, as it does in the standard leads. The inversion due to digitalis may be difficult to distinguish from that of left ventricular hypertrophy and both may be present in the same record. The negative T due to hypertrophy is usually convex, while the T wave of digitalis saturation is concave. A considerable digitalis effect may involve all the precordial leads. When this occurs in advanced left ventricular hypertrophy with deep QS waves from V1-V4, convex elevation of the S-T interval takes the place of depression in these leads (Fig. 19). This is due to the fact that leads to the right of the precordia have the same characteristics as lead VR. Since VR is in effect an intracardiac lead, the deflections are altered by digitalis, as also by anterior infarction (Fig. 17B), in an opposite direction to those of lead I, VL, and the leads to the left of the precordia. In right ventricular hypertrophy, inversion of T can occur in all the precordial leads in the absence of digitalis, and a digitalis effect can seldom be distinguished.

Discussion

Multiple precordial leads have given an accurate picture of left ventricular hypertrophy in 90 per cent of the cases in whom it was judged to be present on other grounds. The most satisfactory criterion has been a diminution in the height of the R wave and an increase in the depth of the S wave in leads to the right of the precordia. Left axis deviation was present in only half those who showed this change. In some of the cases with a normal electrical axis, and in all those with right axis deviation, unipolar limb leads showed that the heart was vertical. In autopsies performed on such cases the right ventricle has always been found to be hypertrophied as well as the left. The combination of signs of left ventricular hypertrophy in the chest leads and a vertical heart would seem to indicate hypertrophy of both ventricles.

In right ventricular hypertrophy chest leads are not so successful since lesser grades of hypertrophy do not alter the curves. The heart is always in a vertical or semi-vertical position when the characteristic changes are present in lead V1.

In bundle branch block it is nearly always possible to determine the side of the lesion. The form of the standard leads varies greatly with the position of the heart and curves are concordant when the position is vertical. Occasionally the standard leads may suggest a right branch block, when in fact the lesion is on the left side. The reverse has not been seen.

Although in general the maximal changes in infarction are usually to be found in the region of the apex, localization is more exact when multiple leads are taken. A clearer picture is given of the extent of infarction. Antero-septal and antero-lateral types can be distinguished. The combination of anterior infarction and right bundle branch block, due to involvement of the septum, is clearly seen.

The information given by the apical lead IV is inadequate in many respects. Ventricular hypertrophy is not shown by it at all. Indeed, it often lies in the transitional zone which is under the influence of both ventricles. The lead does not assist in determining the side of the lesion in bundle branch block. In antero-septal infarction the changes may be limited to leads to the right of the precordia and the apical lead may be normal.

When several points on the chest wall are to be explored, it is important that all extraneous influence should be eliminated as far as possible. Since CR and CF leads are bipolar, the extremity used has some effect upon the curve. Although in 90 per cent of cases these leads were found to be sufficiently accurate as regards the T waves, distortion was appreciable in the remaining 10 per cent. Inverted T waves may be recorded in CF leads, if the heart is vertical, owing to negative distortion from the left leg. Negative T waves may be made positive in CR leads by distortion from the right arm. The distortion is not, however, confined to the T waves. R waves will be more positive in CR leads and S waves will be more negative in CF leads, with the result that the balance of a series may be altered.
and left ventricular hypertrophy may be missed or diagnosed wrongly. The unipolar method of Wilson appears to be accurate within narrow limits. The leads are simple and easy to apply. If they become, as we believe they will become, the standard method of taking chest leads, instrument makers will have no difficulty in introducing a switch that will obviate the need for any additional connections.

Unipolar limb leads enable the position of the heart to be ascertained. Axis deviation is a compound of positional changes and hypertrophy, whereas the unipolar limb leads vary, as regards the R and S waves, with position only. When the position is normal, lead VL reflects the potentials of the anterior surface of the left ventricle, and lead VF of the posterior surface, more accurately than do leads I and III. In lead I a state of negativity at the left arm may be obscured by the subtraction of a greater state of negativity at the right arm. The T wave is, therefore, inverted in lead VL in anterior infarction more often than in lead I. In lead III the reverse occurs. VL, facing the tail of the wave, will have positive deflections and this, when subtracted from the negative deflections obtained at the left leg, will cause lead III to have more pronounced changes than VF.

The difficulty with the unipolar method is the time required to take a twelve lead electrocardiogram. It is hardly practicable for general use. We have found that three chest leads give a reasonably accurate picture if they are varied according to circumstances. Thus, V2, V3, and V4 will register the changes of infarction if the heart is not enlarged. If enlargement is present, it is better to bracket the apex: thus V4 and V5 may be used or, if enlargement is gross, V5 and V6. When right-sided hypertrophy or right branch block is suspected, V1 is more appropriate than V2. In left-sided hypertrophy the S wave is usually deeper in V2 than in V1. Further experience will probably suggest better combinations of leads. It has been suggested (Goldberger, 1942) that unipolar limb leads may come to supplant the standard leads. Although this is possible, it seems certain that it must be a long time before the knowledge which has been accumulated regarding the standard leads can be safely discarded.

CONCLUSIONS

A series of 300 twelve lead electrocardiograms taken with the unipolar method devised by Wilson have been analysed.

Left ventricular hypertrophy has been diagnosed from the precordial leads in 90 per cent of those in whom it was judged to be present on other grounds. Left axis deviation was present in half of these cases.

Right ventricular hypertrophy causes characteristic changes in leads to the right of the precordia, but a considerable amount of hypertrophy is needed to produce them.

In bundle branch block it is almost always possible to determine the side of the lesion. Left branch block has sometimes been shown to be present by the precordial leads, although the standard leads suggested a right-sided lesion.

Anterior infarction can be divided into antero-septal and antero-lateral types. The combination of anterior infarction and a right branch block, due to involvement of the septum, is clearly shown.

The unipolar method avoids the distortion that occurs in a proportion of cases when bipolar leads such as CR and CF are used. Unipolar leads are preferable when multiple precordial leads are employed.

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REFERENCES

Hoyos, Tomayo (1947). Ibid., 33, 698.