Comparison of a Good Orthogonal Lead System and One Additional Chest Lead with the Conventional 12-Lead Electrocardiogram

By Oscar Tannenbaum, M.D., Harry Vesell, M.D., and Jerome A. Schack, M.D.

SUMMARY
In a study of the McFee-Parungao system of orthogonal lead electrocardiography, 452 electrocardiograms were recorded on 111 normal subjects and 201 patients with cardiovascular disease and abnormalities in the conventional 12-lead electrocardiogram. Comparison of the two lead systems and evaluation of criteria for the electrocardiographic diagnosis of normal, right ventricular hypertrophy, left ventricular hypertrophy, right and left bundle-branch block, and several types of myocardial infarction revealed good correspondence between the two systems. Because of deficient representation of the precordial midzone potential, a preferential unipolar lead resembling lead V4 was introduced. This ancillary lead contributed important information to the electrocardiographic diagnosis of localized apical myocardial infarction and also indicated ventricular hypertrophy in cases in which it was not evident in the three orthogonal leads.

Additional Indexing Words:
Ventricular hypertrophy
Myocardial infarction
Bundle-branch block

Under the impetus of vectorcardiographic investigation, new and improved orthogonal lead systems have been developed. At the outset of this search for better leads that were mutually orthogonal, a geometric pattern based on electrode placements that were anatomically orthogonal was used.1-8 These leads obviously were subject to some of the same factors of distortion in recording the electromotive force of the heart as the ordinary bipolar and unipolar leads of the routine electrocardiogram. By means of torso models and other methods corrected orthogonal leads based on sound biophysical principles were developed.4-7 Thus, the effect of the direction and strength of the leads should be relatively constant regardless of the location of voltages in the heart, conducting media, and body build. The ability of these new corrected lead systems to record accurately the electrical events of the cardiac cycle, using only three leads as compared to 12 or more leads of the present system of clinical electrocardiography, is an important advance, providing there is no loss of important clinical information. The reduction in the number of leads lends itself to computer analysis and display as Lissajous figures on the face of the cathode-ray screen to determine phase differences. Simplicity and accuracy are most important factors in utilization of any new lead system for clinical electrocardiography.

The corrected orthogonal leads, although based on sound physical concepts, have also demonstrated certain shortcomings when applied to clinical practice. Studies8-12 comparing various corrected orthogonal lead systems with conventional electrocardiograms and clinical observations have revealed the frequent failure of these systems to show certain important diagnostic information which was...
present in the usual 12-lead electrocardiogram. To correct for this deficiency, it has been suggested that either an ancillary or preferential precordial lead be introduced\textsuperscript{11,13,14} or an electronic resolver\textsuperscript{9} be utilized.

In the present study the axial lead system of McFee and Parungao\textsuperscript{15} was used. This choice was made because it satisfies clinical requirements of simplicity, reasonable accuracy, relative few electrodes, less sensitivity to errors in electrode placement, and also the fact that the sagittal lead consisted of three anterior electrodes making it simple to record a preferential precordial lead. Several comparative studies of corrected orthogonal leads in torso models and on the living human chest\textsuperscript{16,17} have shown that the axial leads of McFee and Parungao are well corrected.

The present study was undertaken to ascertain the accuracy of this orthogonal lead system when compared with clinical studies and the conventional electrocardiogram and to introduce an additional unipolar precordial lead that would record preferentially electromotive forces of midzone potentials that are occasionally not well recorded or even completely missed\textsuperscript{11,12} with the corrected orthogonal leads. As clinical experience was gained with these corrected orthogonal leads, it became more apparent that such an ancillary lead was necessary. In fact, Frank\textsuperscript{7} in describing a clinical orthogonal lead mentioned two alternative central terminal leads representing the dipole midpoint for use with his lead system though he did not think they were necessary.

**Methods**

Four hundred and fifty-two electrocardiograms were taken on 312 patients: 145 females and 167 males, 21 to 85 years of age. In 111 patients the cardiovascular system was considered normal by the usual clinical standards as previously described.\textsuperscript{11} Any patient having a condition known to affect the electrocardiogram directly or indirectly was excluded from the normal group. The 12-lead electrocardiogram was considered normal by usually accepted clinical electrocardiographic criteria.\textsuperscript{18} The normal group consisted of 50 males and 61 females, 21 to 65 years of age. Cardiac abnormalities were present in 201 patients. One hundred and nineteen were men and 82 women, 17 to 85 years of age. Conditions selected for study were left ventricular hypertrophy, right ventricular hypertrophy, left bundle-branch block, right bundle-branch block, anterior wall myocardial infarction, inferior wall myocardial infarction, and nontransmural infarction with T-wave abnormalities alone.

The corrected orthogonal lead electrocardiogram was recorded by the method of McFee and Parungao.\textsuperscript{15} The X lead (transverse) is recorded from two electrodes 11 cm apart in the left axilla located one third of the way from the anterior chest wall and the back. The midpoint between these electrodes lies at the fifth interspace at the left axilla. The third electrode for the X lead is a single electrode placed at this level at the right axilla. The potential of the two left axillary electrodes is averaged with two matched, precision (\( \pm 1\% \)) 65,000 ohm resistors. The Y lead is a simple bipolar lead obtained by pairing an electrode from the left leg with one placed on the left side of the neck. The Z lead is obtained by pairing three electrodes in the form of a triangle over the precordium with a single electrode on the back directly behind the heart. The base of the triangle is oriented toward the feet. The precordial electrodes are equalized with three matched precision resistors (\( \pm 1\% \)) of 100,000 ohms. The preferential precordial lead to record the midzone potential is unipolar and is obtained with the orthogonal lead electrodes left in situ. By connecting the right axillary electrode, the upper electrode of the left axilla, and the left leg electrode through equal matched 10,000 ohm resistors, this becomes the central terminal; the active, exploring electrode is the one at the outer aspect of the base of the triangle located on the precordium (figs. 1 and 2).\textsuperscript{*} The polarities of the leads were selected as follows: X axis, right negative, left positive; Y axis, superior negative, inferior positive; and Z axis, posterior negative, anterior positive. Polarity of the modified unipolar precordial (MUP) lead is that used in conventional clinical electrocardiography. All tracings were recorded on a single channel direct writing instrument at a paper speed of 25 mm per second with the patient in the recumbent position. The conventional 12-lead electrocardiogram was taken immediately before the orthogonal and MUP lead tracing. Standardization of both the conventional and axial-MUP lead electrocardiogram was at 1 mv equal to 1-cm

\*This electrode arrangement will give a central terminal similar to, but not identical with, the one usually employed for V leads. It obviated repositioning of the electrodes and permitted the use of a single switch to record this potential.
Location of the electrodes. The left axillary electrodes are permanently affixed to a Plexiglass plate 11 cm apart. The precordial electrodes are also permanently attached to a triangular Plexiglass plate located 6 cm from the center of the triangle. The center point of this triangle is placed at the fifth interspace 2 cm to the left of the sternal margin.

deflection. The conventional 12-lead electrocardiogram was interpreted by the customary standards. The axial and MUP leads were described in the same manner as used in clinical electrocardiography. The conventional electrocardiogram was compared with the corrected orthogonal lead and ancillary lead for the presence or absence of findings that may be clinically useful. All deflections and intervals were measured with the aid of a 5× magnifying lens; amplitude was estimated to 0.2 mm, and duration was read to 0.01 per second.

**Results**

The normal orthogonal lead and MUP lead electrocardiogram, durations, amplitudes, and amplitude ratios are shown in table 1 for

**Table 1**

*Measurements of P, QRS, and T in Leads X, Y, Z and MUP in 111 Normal Patients*

<table>
<thead>
<tr>
<th></th>
<th>Mean X</th>
<th>SD</th>
<th>Mean Y</th>
<th>SD</th>
<th>Mean Z</th>
<th>SD</th>
<th>Mean MUP</th>
<th>SD</th>
</tr>
</thead>
<tbody>
<tr>
<td>P amplitude (mm)</td>
<td>0.54</td>
<td>0.13</td>
<td>0.91</td>
<td>0.62</td>
<td>0.17</td>
<td>0.11</td>
<td>0.16</td>
<td>0.10</td>
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<td>QRS duration (sec)</td>
<td>0.06</td>
<td>0.012</td>
<td>0.06</td>
<td>0.014</td>
<td>0.07</td>
<td>0.014</td>
<td>0.07</td>
<td>0.012</td>
</tr>
<tr>
<td>Q duration (sec)</td>
<td>0.003</td>
<td>0.005</td>
<td>0.001</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>Q amplitude (mm)</td>
<td>0.25</td>
<td>0.48</td>
<td>0.05</td>
<td>0.01</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>0.08</td>
</tr>
<tr>
<td>R amplitude (mm)</td>
<td>12.53</td>
<td>3.22</td>
<td>5.52</td>
<td>3.32</td>
<td>4.82</td>
<td>2.92</td>
<td>9.72</td>
<td>8.25</td>
</tr>
<tr>
<td>S amplitude (mm)</td>
<td>0.48</td>
<td>0.22</td>
<td>1.31</td>
<td>1.99</td>
<td>8.40</td>
<td>3.94</td>
<td>3.26</td>
<td>2.61</td>
</tr>
<tr>
<td>R/S amplitude (ratio)</td>
<td>12.46</td>
<td>9.94</td>
<td>2.21</td>
<td>3.80</td>
<td>0.72</td>
<td>0.69</td>
<td>4.73</td>
<td>4.86</td>
</tr>
<tr>
<td>T amplitude (mm)</td>
<td>2.54</td>
<td>1.28</td>
<td>1.71</td>
<td>0.94</td>
<td>2.51</td>
<td>1.43</td>
<td>2.18</td>
<td>1.88</td>
</tr>
</tbody>
</table>
Figure 2

Circuit diagram. All lead cables to the patient should be shielded as well as those to the electrocardiograph. All components are enclosed in a shielded box, which should be grounded suitably. The right leg lead from the electrocardiograph should be connected to the shielded box.

the leads X, Y, Z, and MUP in the group of 111 normal patients.

P Waves

In lead X, the P wave was upright in all cases except for two instances of diphasic P waves. In lead Y, the P wave was upright except for three instances, in one of which it was inverted, in one isoelectric, and in one diphasic. In lead Z, there were 72 instances of upright P waves, 11 of isoelectric, 27 of diphasic, and one of inverted P waves. In the MUP lead, there were upright P waves in 82 cases; in 24 cases P waves were isoelectric and in five diphasic but in none were they inverted. The mean P vector pointed to the left anteriorly and inferiorly.

QRS Complex

The QRS duration for leads X and Y was 0.06 ± 0.01 second and for lead Z and the MUP lead 0.07 ± 0.01 second. In 30 cases a small q wave measuring 0.30 to 1.50 mm and lasting 0.01 to 0.02 second was present in lead X. A small q wave measuring no more than 1.0 mm with a duration of 0.01 second was also noted in 11 patients in lead Y; and in the MUP lead such small q waves were observed in 10 patients; here too the amplitude never exceeded 2.50 mm with a duration of 0.01 second. In lead Z an RS was
present in all cases except one with a QS. However, an S wave, measuring 0.50 to 14.50 mm, was present in the MUP lead in 102 instances. Small s waves were occasionally noted in lead X, and more frequently in lead Y. The range of R was 4.0 to 23 mm in lead X, 0.50 to 16.0 mm in lead Y, 1 to 17 mm in lead Z, and 1 to 30 mm in the MUP lead. The range of S was 0.25 to 3.50 mm in lead X, 0.40 to 16 mm in lead Y, 1.80 to 20 mm in lead Z, and 0.50 to 34 mm in the MUP lead.

The combinations of the components of the QRS complex measured are given in table 2.

The S-T segment was isoelectric in all leads except on a rare occasion when it was depressed up to 0.2 mm in leads X and MUP. The T wave was upright in all cases in lead X, isoelectric in two cases in leads Y and Z, diphasic in four cases in the MUP lead, and inverted in three instances in lead Z, and once in the MUP lead. Mean amplitudes of T are given in table 1.

The configuration of the QRS complexes in the orthogonal leads and the MUP lead was compared with that in the conventional 12 leads (fig. 3). There was close similarity in the appearance of this complex in the following leads: Lead X with leads I, V5, and V6; less so with aV1, and only occasionally with leads V3 and V4; lead Y and aVF and

![Figure 3](http://circ.ahajournals.org/)

Comparison of the configuration of the QRS in the orthogonal and modified unipolar precordial leads with that in the 12-lead conventional electrocardiogram of normal patients (111).

### Table 2

<table>
<thead>
<tr>
<th>Type of Wave</th>
<th>X</th>
<th>Y</th>
<th>Z</th>
<th>MUP</th>
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</thead>
<tbody>
<tr>
<td>Isoelectric P wave</td>
<td>0</td>
<td>1</td>
<td>11</td>
<td>24</td>
</tr>
<tr>
<td>Diphasic P wave</td>
<td>2</td>
<td>1</td>
<td>27</td>
<td>5</td>
</tr>
<tr>
<td>Inverted P wave</td>
<td>0</td>
<td>1</td>
<td>1</td>
<td>0</td>
</tr>
<tr>
<td>Q wave</td>
<td>30</td>
<td>11</td>
<td>1</td>
<td>10</td>
</tr>
<tr>
<td>S wave</td>
<td>32</td>
<td>64</td>
<td>111</td>
<td>102</td>
</tr>
<tr>
<td>Isoelectric T wave</td>
<td>0</td>
<td>2</td>
<td>2</td>
<td>0</td>
</tr>
<tr>
<td>Diphasic T wave</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>4</td>
</tr>
<tr>
<td>Inverted T wave</td>
<td>0</td>
<td>0</td>
<td>3</td>
<td>1</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Amplitude (mm) of</th>
<th>Mean</th>
<th>SD</th>
<th>Range</th>
</tr>
</thead>
<tbody>
<tr>
<td>Rₓ + Sₓ</td>
<td>20.95</td>
<td>5.37</td>
<td>9.50-36.50</td>
</tr>
<tr>
<td>Rₓ + Rₓ</td>
<td>17.35</td>
<td>4.71</td>
<td>4.50-33.50</td>
</tr>
<tr>
<td>Rₓ + Rₘᵤₙ</td>
<td>22.21</td>
<td>7.31</td>
<td>6.00-49.50</td>
</tr>
<tr>
<td>Rₓ + S₂</td>
<td>5.26</td>
<td>3.15</td>
<td>0.50-17.00</td>
</tr>
</tbody>
</table>
lead II, rarely with lead III, and only two instances with aV1; lead Z often with V2 and V3, rarely with V1, and in four instances with V4. Lead Z did not resemble any of the conventional leads in two cases; the MUP lead resembled V4 in most instances, occasionally V5, and rarely V3 and V6. In isolated instances this lead resembled leads I, V1, and no conventional lead.

The S-T segment of the axial and the MUP lead closely followed those observed in the conventional 12 leads. However, the T corresponded well in both the axial and the MUP lead with the conventional leads in all cases except in three instances in the MUP lead where these were diphasic and in the corresponding conventional lead were distinctly upright.

Figure 4 illustrates the orthogonal leads (X, Y, and Z) and the additional chest lead taken from a patient with a normal heart and from patients with the abnormalities discussed below.

Left Ventricular Hypertrophy

Sixty patients ranging in age from 47 to 85 years had readily recognizable clinical cardiac disease capable of producing hypertrophy of the left ventricle. In addition, these patients had definite enlargement of the left ventricle on radiographic examination. In 50 of these cases the 12-lead electrocardiogram fulfilled the criteria of Sokolow and Lyons19 for left ventricular hypertrophy, in QRS voltage and S-T and T changes. In the remaining 10 cases the conventional electrocardiogram disclosed the abnormal voltage criteria of left ventricular hypertrophy without the S-T and T-wave abnormalities. None of these 60 patients had a previous history of known myocardial infarction or of intraventricular conduction defects (QRS interval exceeding 0.12 second) in the 12-lead electrocardiogram.

In the 50 patients whose orthogonal lead electrocardiograms exhibited the voltage criteria of left ventricular hypertrophy in association with S-T and T-wave abnormalities, the amplitude of the R wave in lead X measured 22.40 ± 5.60. This was larger than 12.53 mm (normal mean) in 49 patients and greater than the maximum Rx amplitude of 23 mm in the normal control in 21 cases. Although the amplitude of the S wave in lead Z (13.76 ± 8.42 mm) was similarly increased, the R/S ratio was not uniformly changed when compared with the normal controls. However, in 22 patients (44%) there was a substantial reduction (less than 0.3) of the R/S ratio. In the remaining 26 cases in which the R/S ratio was not reduced to less than 0.30, the R-wave amplitude in the MUP lead was considerably increased above the normal control. On the other hand when the R/S ratio in lead Z was reduced (less than 0.30), the amplitude of the R wave in the MUP lead did not exceed the R-wave amplitude of the normal control in 11 patients. In two patients a QS was present in lead Z. The sum of the amplitude of the R wave in lead X and the S wave in lead Z measured 34.75 ± 4.33 mm and exceeded 20.95 mm (the normal mean) in 47 cases. In 24 instances the sum of Rx and Rx was greater than 36 mm, the maximum observed in the normal control. On the other hand the sum of amplitudes of Rx and Sx was 27.47 ± 7.63 mm and was greater than 17.35 mm (the normal mean) in 48 patients and in 24 cases exceeded 33.50 mm, the maximum of the normal control. The sum of the amplitude of the R wave in lead X and the MUP lead measured a mean of 38.42 ± 11.07 and exceeded the mean (22.21 mm) of the normal control in 48 cases (96%); and in nine instances (18.0%) exceeded the maximum measurement (49.50 mm) in the normal series. When the R/S ratio of lead Y was utilized as a measure of left axis deviation in the frontal plane of the orthogonal leads, there was no significant difference in this parameter in the group of left ventricular hypertrophy when compared with the normal controls. An S wave was present in lead Y in 62.0% (31 cases) of tracings with left ventricular hypertrophy, and similarly in 57.7% (64 cases) of the normal control series. Obviously, cardiac position is a limiting factor in the use of this measurement. The mean
Figure 4

The orthogonal and modified unipolar precordial lead electrocardiograms. (A) Normal heart. (B) Left ventricular hypertrophy. (C) Right ventricular hypertrophy due to Eisenmenger's syndrome. (D) Right bundle-branch block. (E) Left bundle-branch block. (F) Extensive anterior wall myocardial infarction. (G) Anteroseptal myocardial infarction. (H) Localized apical myocardial infarction. Note the absence of any significant abnormality in the orthogonal leads X, Y, and Z and the presence of a significant Q wave and S-T elevation in the modified unipolar precordial lead, thus making the diagnosis possible. (I) Inferior wall myocardial infarction. (J) “T-wave infarction.”

QRS interval was increased slightly with the maximum 0.12 second.

In lead X the S-T segment was depressed in all cases and the T wave was inverted in this group. In lead Y the S-T was isoelectric in 32 cases, depressed in 16, and slightly elevated in two. The T wave in this lead was inverted in 12 instances, diphasic in seven, isoelectric in two, and definitely upright in the remaining 29 cases. In lead Z the S-T segment was isoelectric in 41 cases, depressed in four, and slightly elevated in five. The T
wave was upright in 30 instances, inverted in 11, and diphasic in the remaining nine cases; in no case was the T wave isoelectric. In the MUP lead the S-T segment was depressed in 24 cases, isoelectric in 22, and slightly elevated in the remaining four. The S-T and T-wave changes corresponded closely with their counterparts in the conventional 12-lead electrocardiogram.

Seven patients with diagnostic high QRS voltage criteria of left ventricular hypertrophy in conventional leads and without S-T and T-wave abnormalities, also failed to demonstrate the S-T and T abnormalities in the regular orthogonal leads. However, in two of these seven cases S-T and T-wave changes did appear in MUP lead with definite inversion of the T wave.

In the other three patients with such criteria the T wave was borderline abnormal, isoelectric, or small and diphasic. In the MUP lead these T waves were definitely inverted.

**Right Ventricular Hypertrophy**

Eleven patients, ranging in age from 17 years to 72 years, had clinically evident cardiopulmonary disease capable of producing right ventricular hypertrophy. In all 11, the conventional 12-lead electrocardiogram disclosed a pattern of right ventricular hypertrophy according to the criteria of Sokolow and Lyons as modified by Milnor and Roman and associates. In six patients in this group the diagnosis of congenital heart disease was substantiated by clinical findings, catheterization studies, and angiocardiography. The diagnoses were pulmonary stenosis in four cases, atrial septal defect in one, and Eisenmenger's syndrome in one; there were two cases of rheumatic heart disease with tight mitral stenosis and three of pulmonary emphysema.

The QRS complex in the orthogonal lead did not exceed 0.1 second in duration in this group. In lead X the mean amplitude of the R wave, measuring 8.09 ± 4.52 mm, was reduced with a marked increase in the amplitude of the S wave (9.96 ± 3.68). The R/S ratio in lead X was reduced to 1.04 ± 0.86 compared to 12.46 ± 9.9 in the control. Lead Y presented little change from the normal control. In lead Z there were differences among the etiological groups. The six patients with congenital lesions had a substantial increase of the R amplitude compared to the normal controls. R measured 9.28 ± 2.39 mm with an increase of the mean R/S ratio to 2.28 ± 2.35, from 0.72 ± 0.69. On the other hand in the three patients with cor pulmonale, there was no significant increase of the R amplitude or R/S ratio in lead Z. In the MUP lead there was a considerable increase in the amplitude of the S wave (10.15 ± 4.43 mm) for the entire group of 11 regardless of the etiology of the right ventricular enlargement; also there was a considerable decrease in the R/S ratio to 0.88 ± 0.52 as compared to the normal (4.73 ± 4.86). It is noteworthy that in the case with the intraventricular septal defect the characteristic rSR' in V1 was not evident in any of the orthogonal leads or in the MUP lead. However, the R/S ratio in lead Z approached unity with a marked reduction of the R/S ratio in the MUP lead. The sum of the amplitude of R and S was considerably increased (18.23 ± 7.06 mm) for the entire group of right ventricular hypertrophy.

Lead Y best demonstrated the P wave of right atrial strain. The mean amplitude was 1.70 mm (range, 0.25 to 5.0 mm) compared with a normal mean amplitude of 0.71 mm (range, 0.20 to 2.0 mm).

**Bundle-Branch Block**

In 26 patients the conventional 12-lead electrocardiogram demonstrated QRS intervals of 0.12 second or more and the usual criteria of right bundle-branch block. Six had had myocardial infarction that will be considered later. The remaining 20 were considered to have uncomplicated right bundle-branch block, with absence of myocardial infarction, congenital heart disease, cor pulmonale, or valvular disease known to produce right ventricular hypertrophy.

In 16 of these 20 patients the orthogonal leads showed a maximum QRS duration of 0.12 second or more. In four, however, the longest QRS interval did not exceed 0.10
second. These four also did not have the characteristic RSR' (just a tall R and a slurred S) in lead Z though RSR' was present in this lead in the other 16 patients and in leads V_1 and V_2 in all 20.

Such RS findings in lead Z (McFee and Parungao) were observed experimentally in the canine heart after severance of the false tendon, and believed to be due to incomplete right bundle-branch block. Similar findings in our four cases might suggest incomplete right bundle-branch block rather than more complete right bundle-branch block as manifested in the conventional 12-lead electrocardiogram.

A broad S wave of 0.05 to 0.08-second duration (mean, 0.07 ± 0.01 second) was present in lead X in the 20 patients and in the MUP lead with a mean duration of 0.075 ± 0.03 second.

Twenty-one patients had typical electrocardiographic features of complete left bundle-branch block in the conventional 12-lead electrocardiogram and no clinical or usual electrocardiographic evidence of myocardial infarction. In the orthogonal leads, the mean QRS interval was prolonged and measured 0.14 second ± 0.01 second in lead X, 0.14 ± 0.01 second in lead Y, 0.14 ± 0.02 second in lead Z, and 0.14 ± 0.02 second in the MUP lead. Q waves were absent in lead X in all cases. The R wave in lead X was tall and broad and either notched or bifid. It was associated with a depressed S-T segment and a sharply inverted T wave in 19 cases. In two instances the T wave was diphasic. The QRS, S-T and T in lead X closely resembled their counterparts in leads I, aV_L, and V_6. In lead Y there was either a broad R, or an rS, a tall R with a broad S, or a QS (four cases). There were no S-T or T-wave alterations in this lead. Lead Y closely resembled lead aV_F. Lead Z revealed an rS or QS pattern with upright T waves and resembled the right unipolar precordial leads of the conventional electrocardiogram. The MUP lead resembled the conventional left precordial leads in this condition. The correlation of all of the orthogonal leads with the conventional electrocardiogram in left bundle-branch block was excellent.

**Myocardial Infarction**

There were 83 patients with clinical evidence of myocardial infarction and characteristic electrocardiographic changes in the conventional electrocardiogram. In 56 of these patients electrocardiographic changes were suggestive of “transmural infarction,” and in 27 patients, of probable subendocardial infarction without definite abnormalities in the QRS complexes. Localization of the myocardial infarcts, on the basis of the conventional electrocardiograms, and their distribution in this series are shown in Table 3. A myocardial infarct which involved an extensive area of the anterior wall of the left ventricle, according to the conventional 12-lead electrocardiogram, was represented by a deep, or broad (0.04 second or more), or both, Q

<table>
<thead>
<tr>
<th>Area of infarction</th>
<th>Total patients</th>
<th>Patients presenting Q waves in leads</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>X</td>
</tr>
<tr>
<td>Extensive anterior</td>
<td>7</td>
<td>7</td>
</tr>
<tr>
<td>Anteroseptal</td>
<td>12</td>
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<tr>
<td>Antero-apical</td>
<td>4</td>
<td>1</td>
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<tr>
<td>Lateral</td>
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<td>2</td>
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<tr>
<td>Inferior</td>
<td>18</td>
<td>0</td>
</tr>
<tr>
<td>Inferolateral</td>
<td>7</td>
<td>7</td>
</tr>
<tr>
<td>Combined anterior and inferior “T-wave infarct”</td>
<td>6</td>
<td>3</td>
</tr>
<tr>
<td>Right bundle-branch block with subsequent myocardial infarction</td>
<td>27</td>
<td>0</td>
</tr>
</tbody>
</table>

Table 3

*Distribution of Pathological Q Waves in Myocardial Infarction in the Orthogonal and Modified Unipolar Precordial Leads*

*Circulation, Volume XXXV, January 1967*
wave in leads X, Z, and the MUP; S-T and T-wave changes depended upon the stage of the infarction.

In 12 cases electrocardiographic changes in the conventional 12-lead tracing suggested the presence of an anteroseptal infarction. Diagnostic Q and T-wave changes were present in lead Z in 11. In four of the 11 cases these Q and T-wave abnormalities also were present in the MUP lead. Two of the 11 patients showed variations in T-wave recordings. In these two a negative T wave was registered by the MUP lead but not in its counterpart V₄.

In the remaining case with the electrocardiographic pattern of anteroseptal infarction in the conventional leads, the orthogonal leads failed to reveal any diagnostic changes of myocardial infarction. Postmortem examination in this case confirmed the presence of an anteroseptal infarction. Left ventricular hypertrophy was also present.

There were four cases of antero-apical myocardial infarction according to the conventional 12-lead electrocardiogram. In only one of these four cases were significant and diagnostic Q and T-wave changes found in one of the axial leads, lead X. In the remaining three cases no pathological Q or T-wave changes were recorded in any of the three orthogonal leads. However, in all four of the cases of antero-apical infarction the unipolar MUP lead did reveal the characteristic Q and T-wave changes of myocardial infarction.

There were two patients whose conventional lead tracing revealed significant Q waves in leads I, aV₁, and V₆, considered to represent lateral wall infarctions. Both showed an abnormal Q wave in lead X.

Infarctions of the inferior or diaphragmatic wall were manifested by deep and broad Q waves in lead Y in 18 cases. Associated T-wave changes were present in lead Y in 16 of the 18 cases. In one, the T was upright, and in another, it was small and diphasic.

In seven cases electrocardiographic evidence of inferolateral infarction was found in the appropriate conventional leads. Diagnostic Q and T-wave changes were present in both leads X and Y in all seven. In addition, pathological Q and T waves were present in the MUP lead in one patient.

In six cases electrocardiographic changes of both anterior and inferior wall myocardial infarction were clearly discernible in the conventional leads. All six had characteristic Q and T-wave changes in leads Y and Z. Five also had these diagnostic changes in the MUP lead and three in lead X as well.

There were six examples of myocardial infarction with accompanying right bundle-branch block, not considered in the above categories but mentioned in the discussion on right bundle-branch block. Two were anteroseptal and four were inferior wall infarcts according to the conventional leads. All six had diagnostic Q waves and associated T-wave changes in the corresponding orthogonal leads.

There were 27 records of so-called coronary T-wave pattern of coveplane, deeply inverted T waves in the conventional unipolar precordial leads without pathological Q waves. In 22 of the 27 the T waves were similarly inverted in the corresponding orthogonal and MUP leads. In the other five cases diphasic T waves or upright T waves were present in leads X and Z; however, in these five instances the T wave was distinctly inverted in the MUP lead as in the conventional chest leads. In none of these instances did the orthogonal or MUP lead demonstrate a pathological Q wave.

Discussion

The application of corrected orthogonal leads to clinical electrocardiography has been made by many investigators in the attempt to elicit more accurate and useful information than is found in the conventional 12-lead electrocardiogram. On purely theoretical grounds (substantiated by biophysical investigation) the corrected orthogonal leads appear to provide a more accurate representation of the cardiac electromotive force at the body surface, especially if vectorcardiograms are to be recorded.
The discrepancies between torso model studies and clinical application raise the possibility of considering the equivalent cardiac generator as a multiple.26–28 Sodi-Pallares and co-workers29,30 on the basis of both clinical and experimental studies believe that the more remote the electrodes are from the heart the more valid is the concept of a single dipole generator, and conversely the closer the exploring electrode approaches the heart the more the influence of multipoles must be considered. It thus appears that certain clinically important electrical activity of the heart might not be adequately recorded by any one particular orthogonal lead system, and therefore that it may be necessary to introduce an ancillary lead regardless of its theoretical shortcomings.

Abildskov and Wilkinson31 by means of an electronic resolver determined the percentage contribution of the X, Y, and Z components of the heart vector to each precordial lead. Their results indicated that information obtained from lead V4 is likely to influence leads X and Z approximately equally. This makes it unlikely that a preferential recording of the apical area comparable to V4 is obtained by any of the orthogonal leads.

Fischmann and Barber32 have described a method of “aimed electrocardiographic leads” selectively sensitive to dipoles in a limited cardiac area but insensitive to electromotive forces in other regions of the heart. They offer this method theoretically to make up for shortcomings of orthogonal lead systems.

Our own clinical studies comparing an orthogonal lead system with the conventional 12-lead electrocardiogram have shown that most but not all of the important clinical information was recognized. In spite of the advantages of simplicity of application and distortionless recording of corrected orthogonal leads, there remains a clinical need for a preferential precordial lead. The MUP lead employed in this study is such a lead, especially useful in demonstrating the presence of localized apical myocardial infarction. It has also furnished corroborative evidence of left ventricular hypertrophy in instances in which the orthogonal leads gave equivocal results.

The simplicity of recording and of interpreting the electrocardiogram with fewer leads is desirable. The data presented suggest that this system may be applicable to routine clinical electrocardiography. The reduced number of leads also is more useful for analysis by computers.

References
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Comparison of a Good Orthogonal Lead System and One Additional Chest Lead with the Conventional 12-Lead Electrocardiogram

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