Electrocardiographic and Cineangiographic Correlations in Assessment of the Location, Nature and Extent of Abnormal Left Ventricular Segmental Contraction in Coronary Artery Disease

By Richard R. Miller, M.D., Ezra A. Amsterdam, M.D., Hugo G. Bogren, M.D., Rashid A. Massumi, M.D., Robert Zelis, M.D., and Dean T. Mason, M.D.

SUMMARY
The relationship between the resting electrocardiogram and left ventricular contractile pattern, as documented by angiography, was evaluated in 123 patients with coronary artery disease who underwent left ventriculography. Dyssynergy was present in 73/77 (95%) patients with pathologic Q waves on ECG recordings in contrast to 11/46 (24%; P < 0.01) without Q waves. The location of Q waves correlated well with the site of abnormal ventricular motion: antero-apical dyssynergy in 40/40 (100%) patients with anterior myocardial infarction (MI) and infero-apical dyssynergy in 25/28 (89%) with inferior MI. Four contraction patterns were defined: 1) normal motion—39 patients (35 without Q waves, four with inferior or posterior Q waves); 2) segmental hypokinesis—37 patients (six without Q, 31 with Q); 3) segmental akinesis—26 patients (four without Q, 22 with Q); and 4) localized dyskinesis—aneurysm in 21 patients (only one without Q, 20 with Q). The presence of ST elevation and T wave inversion (ST\textsuperscript{T} - Ti) along with Q waves were associated with dyskinesis or akinesis in 18/19 (95%) patients. The Q wave location reflected the type of dyssynergy: 32/40 (80%) patients with anterior MI had akinesis or dyskinesis, while 18/28 (64%) patients with inferior MI exhibited hypokinesis. Lateral extension of the Q wave in an anterior MI was related to the dyssynergy type (average V lead: 4.9 in dyskinesis and 3.3 in hypokinesis; P < 0.05) and extent (dyssynergy area/LV silhouette: 31% with Q to V\textsubscript{5} and 58% to V\textsubscript{6} or V\textsubscript{6}; P < 0.05). Dyssynergy area was larger in isolated anterior than inferior MI (42% and 23% of LV perimeter; P < 0.05) and largest in the anterior-inferior MI (68%; P < 0.05). Dyssynergy was more extensive with Q and ST\textsuperscript{T} - Ti than with Q alone (48% and 33% LV perimeter; P < 0.05). Thus, specific QRS and ST-T wave alterations, when monitoring coronary disease, accurately predict characteristics of LV dyssynergy: Q identifies its presence and location and Q with ST\textsuperscript{T} - Ti estimates its nature and extent.

Additional Indexing Words:
- Akinesis
- Hypokinesis
- Coronary artery disease
- Myocardial infarction
- Dyssynergy
- Ventriculogram
- Dyssynergy
- Ventricular aneurysm
- Electrocardiogram
- Ventricular wall motion

Present Knowledge of the relationship between electrocardiographic (ECG) changes and regional disorders of cardiac wall motion in coronary artery disease has been largely limited to information obtained from postmortem findings of a ventricular aneurysm.\textsuperscript{1-10} Although the initial functional studies of Gorlin and associates established a correlation between the electrocardiogram and the presence and site of ventriculographic abnormalities,\textsuperscript{16, 17} a detailed description of possible interrelations of specific ECG findings to the dynamic characteristics and size of disturbed segmental contraction (ventricular dyssynergy) is

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not available. Therefore, this study was undertaken to evaluate the relationship between ECG alterations and patterns and extent of abnormal left ventricular segmental contraction, defined by ventriculography, in patients with arteriographically documented coronary obstruction. From this analysis the differential criteria are provided for the ECG determination of the location, type and size of left ventricular dyssynergy in coronary heart disease.

Methods

Biplane left ventricular cineangiograms of 123 consecutive patients with an arteriographically proven coronary obstruction (≥75% stenosis of at least one of the three major coronary arteries) were evaluated for abnormalities of left ventricular segmental contraction. Prior to coronary arteriography, biplane left ventriculography was performed in the 30° right and 60° left anterior oblique projections on 35 mm film taken at 64 frames/sec using a Philips nine-inch image-amplifier system. The ventricle was opacified with 50 to 75 cc of Hypaque-M 75 or 76% containing sodium and meglumine diatrizoates injected at 300–400 per square inch through an angiography catheter. Tracings of left ventricular end-diastolic and end-systolic endocardial silhouettes were obtained in the right anterior oblique position from which qualitative and quantitative determinations of segmental contraction were performed.16,17 The first complete cardiac cycle in which the left ventricular cavity was completely opacified by contrast material, and which was at least two beats following any premature ventricular contractions, was utilized for the end-systolic and end-diastolic images.

Specific patterns exhibiting localized abnormal left ventricular segmental contraction were defined by the following criteria: hypokinesia—diminished regional systolic shortening in which there was less inward excursion of the disturbed segment (<15–30% decrease of end-diastolic long and short diameters) than the remaining unaffected areas;18,19 akinesia—absent systolic movement of a segment of the wall; dyskinesia—paradoxical outward systolic expansion (ventricular aneurysm) in which a portion of the end-systolic silhouette extended outside the end-diastolic perimeter. Dyssynergy is used in this report as a general term signifying any of these localized disorders of wall motion. Combined types of dyssynergy were designated by the quantitatively predominant pattern, and the total area of dyssynergy was included in the measurement of the extent of a disorder in wall motion. For the purpose of localizing dyssynergy, the perimeter-distance of the left ventricular end-diastolic silhouette was measured and divided into three segments of equal length: anterior, apical and inferior walls. Resting standard 12 lead electrocardiograms were obtained prior to cardiac catheterization. The electrocardiographic and angiographic interpretations were performed independently to eliminate observer bias. Patients with a history of a myocardial infarction occurring within three months prior to cardiac catheterization were excluded from this study. Patients with left bundle branch block, left anterior or posterior hemiblock, Wolff-Parkinson-White syndrome, or ECG evidence of left or right ventricular hypertrophy were excluded. The electrocardiographic criteria utilized in this study were as follows: antero-septal infarction, QS deflection or Q ≥ 0.04 sec in leads V1 to V6; antero-apical infarction, QS wave or Q ≥ 0.04 sec in V6 to V4; and antero-lateral infarction, Q wave ≥ 0.04 sec in leads I, V5 and V6. The general term anterior myocardial infarction includes antero-septal, antero-apical or antero-lateral infarctions. Inferior wall myocardial infarction was identified by Q wave ≥ 0.04 sec in lead aVF and posterior infarction by R wave ≥ 0.04 sec in lead V1 in absence of right ventricular hypertrophy or right bundle branch block. The designation of ST segment elevation with T wave inversion indicates the specific combination of convex upward ST with negative T deflection in the leads with pathologic Q waves diagnostic of myocardial infarction. Non-specific ST and T wave changes are defined as ST segment depression in any leads and/or abnormal isoelectric or inverted T waves in the absence of combined ST segment elevation with T wave inversion in the leads diagnostic of myocardial infarction.

Results

Of the 123 patients with coronary stenosis, 77 (63%) exhibited the ECG Q wave criteria diagnostic of a transmural myocardial infarction. Of these 77 patients with infarction, 73 (95%) demonstrated localized disorders of segmental contraction (P < 0.01; table 1). Of the 46 patients without ECG evidence of transmural myocardial infarction, 35 (76%) had normal ventricular motion (P < 0.05; table 1). Of the 11 patients with dyssynergy in the absence of diagnostic Q waves, 10 had non-specific ST-T wave changes, leaving only one patient with dyssynergy in the presence of a normal ECG.

<p>| Table 1 |
| Sensitivity and Specificity of ECG to Dyssynergy in Coronary Disease |</p>
<table>
<thead>
<tr>
<th>Total patients</th>
<th>Percent of total</th>
</tr>
</thead>
<tbody>
<tr>
<td>ECG infarction</td>
<td>77</td>
</tr>
<tr>
<td>Dyssynergy</td>
<td>73</td>
</tr>
<tr>
<td>False positive ECG*</td>
<td>4</td>
</tr>
<tr>
<td>No ECG infarction</td>
<td>46</td>
</tr>
<tr>
<td>Normal synergy</td>
<td>35</td>
</tr>
<tr>
<td>False negative ECG†</td>
<td>11</td>
</tr>
<tr>
<td>Correct prediction +</td>
<td>108</td>
</tr>
</tbody>
</table>

*Pathologic Q wave incorrectly predicting dyssynergy.
†Absence of pathologic Q wave incorrectly predicting normal synergy.
+Presence or absence of dyssynergy.

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ECG ASSESSMENT OF VENTRICULAR DYSSYNERGY

Location of Dyssynergy

Anterior Myocardial Infarction

Anterior myocardial infarction was documented by ECG changes in 40 patients, all demonstrating dyssynergy. Out of the 21 patients with isolated anterior infarction, two showing antero-septal necrosis exhibited localized dyssynergy by ventriculography confined to the anterior one-third of the left ventricular perimeter, while the remaining 19 patients with antero-septal, antero-apical or anterolateral infarctions had abnormal contraction patterns involving portions of both the anterior and apical thirds of the ventricular silhouette.

Inferior Myocardial Infarction

Isolated inferior myocardial infarction was defined by ECG changes in 28 patients. Dyssynergy was documented by ventriculography in 25 of these patients. Fifteen areas of dyssynergy were located in the inferior one-third, six were positioned in the apical one-third and four included portions of both the inferior and apical segments. Three patients demonstrated normal ventricular motion.

Anterior-Inferior Myocardial Infarction

Of the 19 patients with combined anterior-inferior myocardial infarctions by ECG changes, dyssynergy was demonstrated in the anterior-apical-inferior walls in 10 and the anterior-apical segments in seven, and was limited to the inferior border in two.

Posterior Myocardial Infarction

True isolated posterior infarction evidenced by ECG changes occurred in five patients. Dyssynergy was present in four of these patients (two apical, one anterior and one inferior).

Posterior-Inferior Myocardial Infarction

In the four patients with combined posterior-inferior infarctions, abnormal wall motion occurred in the inferior border in each case.

Nature of Dyssynergy

The correlation of ECG determination of the site of infarction to the pattern of dyssynergy is shown in table 2. Of the 77 patients with ECG documented transmural infarction, 31 patients (40%) had left ventricular segmental hypokinesis by ventriculography, 22 patients (29%) exhibited localized akinesis and 20 patients (26%) showed regional dyskinesis (fig. 1). In contrast, six of the 46 patients (13%) without ECG determination of transmural infarction had segmental hypokinesis, four (9%) demonstrated localized akinesis and only one (2%) showed regional dyskinesis (fig. 1).

Normal Motion

Normal left ventricular wall motion was observed by ventriculogram in 39 patients (fig. 2A). Of these patients, there was no ECG evidence of infarction in 35: 21 (54%) had normal electrocardiograms, 14 (36%) exhibited non-specific ST-T wave changes and four (10%) demonstrated ECG infarction patterns (three inferior and one true posterior). The four patients with ECG evidence of infarction without dyssynergy had infarctions of the inferior or posterior wall and all patients with ECG criteria for anterior infarction, isolated or combined with inferior necrosis, exhibited abnormal segmental contractile patterns.

Hypokinesis

Localized hypokinesis was demonstrated in 37 patients (fig. 2B). Of these patients, 31 (84%) had ECG evidence of myocardial infarction of whom 23

Table 2

<table>
<thead>
<tr>
<th>ECG-Ventriculographic Correlations in Coronary Disease</th>
<th>Contractile pattern</th>
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<tbody>
<tr>
<td></td>
<td>Normal</td>
</tr>
<tr>
<td>No ECG infarction</td>
<td>35</td>
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<tr>
<td>ECG infarction</td>
<td></td>
</tr>
<tr>
<td>Anterior</td>
<td>0</td>
</tr>
<tr>
<td>Anterior-inferior</td>
<td>0</td>
</tr>
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<td>Inferior</td>
<td>3</td>
</tr>
<tr>
<td>Posterior</td>
<td>1</td>
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<tr>
<td>Posterior-inferior</td>
<td>0</td>
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<tr>
<td>Total infarction patients</td>
<td>4</td>
</tr>
<tr>
<td>Total patients</td>
<td>39</td>
</tr>
</tbody>
</table>
Prevalence and nature of dyssynergy in 46 patients (pts) without ECG infarction (No Q) and 77 patients with infarction by ECG (Path Q). Dyssynergy was more frequent with path Q than no Q (P < .02). Each pattern of dyssynergy was more common in patients with path Q than no Q.

(74%) exhibited involvement of the inferior and/or posterior walls only (table 2). In the remaining six patients without a diagnostic infarction pattern by electrocardiogram, five had nonspecific ST-T abnormalities, leaving only one patient with a normal electrocardiogram.

Akinetic
Ventriculographic evidence of localized akinesis was observed in 26 patients (fig. 2C). Of these patients, 22 (85%) had ECG infarction patterns, 14 (64%) of which were anterior. The remaining four patients without diagnostic infarctions by ECG changes had nonspecific ST-T changes. Therefore, all patients with akinesis had an abnormal electrocardiogram (table 2).

Dyskinesis
Segmental dyskinesis was demonstrated by ventriculogram in 21 patients, all but one of whom (95%) showed ECG evidence of transmural infarction (fig. 2D). In seven of the 21 patients (33%), the infarction was confined to the anterior wall by ECG; 11 (52%) were combined anterior-inferior infarctions, one was isolated to the inferior wall and one was a posterior-inferior infarction. The single patient without transmural infarction by ECG criteria had nonspecific ST-T wave changes. Therefore, all patients with dyskinesis exhibited an abnormal electrocardiogram (table 2). Furthermore, 18 patients (85%) with dyskinesis had anterior infarction either isolated or in combination with inferior infarction.

Precordial Q Waves
In the 19 patients with isolated anterior infarction with QS deflections beginning in leads V₁ and V₂, the most lateral precordial lead to which pathologic Q waves extended was related to the qualitative severity of dyssynergy (fig. 3). The average chest lead to which the most lateral pathologic Q wave deflection extended was determined in each group of patients by the type of dyssynergy: hypokinesis 3.3, akinesis 4.0 and dyskinesis 4.9. The extension of precordial pathologic Q waves is significantly greater (P < .05) in patients with dyskinesis than in those with hypokinesis.

Figure 1
Prevalence and nature of dyssynergy in 46 patients (pts) without ECG infarction (No Q) and 77 patients with infarction by ECG (Path Q). Dyssynergy was more frequent with path Q than no Q (P < .02). Each pattern of dyssynergy was more common in patients with path Q than no Q.

Figure 2
Nature of dyssynergy related to the presence (path Q) or absence (no Q) of ECG myocardial infarction. Normal contraction pattern (A) was associated with no Q (P < .01) while in hypokinesis (B) (P < .02), akinesis (C) (P < .05) and dyskinesis (D) (P < .01) path Q was more common.

Figure 3
Relationship of nature of dyssynergy to left precordial extension of pathologic Q waves in 19 patients with isolated anterior infarction. QS deflections began in lead V₁ or V₂, in each individual. Patients with dyskinesis had abnormal Q waves extending farther laterally than those with hypokinesis (P < .05).
**ST Segment Elevation with T Wave Inversion**

Of the 77 patients with ECG documented infarction, 19 exhibited ST segment elevation combined with T wave inversion in the leads with pathologic Q waves. All of these 19 patients had segmental dyssynergy: one (5%) hypokinesis, 5 (26%) akinesis and 13 (69%) dyskinesis (fig. 4). In the 58 patients with infarction documented by electrocardiogram, with nonspecific ST-T wave changes or without ST-T wave alterations, abnormal regional wall motion occurred in 54 (93%): 30 (52%) hypokinesis; 17 (29%) akinesis and seven (12%) dyskinesis. Four patients (7%) exhibited normal motion. Dyskinesis occurred more frequently (> 0.02) in patients with the specific combination of ST segment elevation with T inversion on leads signifying infarction than in patients with ECG evidence of infarction without these ST-T changes (fig. 4).

**Extent of Dyssynergy**

**Precordial Q Waves**

In the 19 patients with isolated anterior infarction with QS waves starting in leads V1 and V2, the lateral precordial extension of pathologic Q wave deflections was compared with the extent of dyssynergy defined as the percent of total left ventricular inner perimeter demonstrating abnormal wall motion (fig. 5A). The quantity of dyssynergy was 31% in the five patients with QS deflections from V2-V8, 40% in 10 patients with pathologic Q waves extending to V4, and 58% in four patients with pathologic Q wave deflections extending to V6 or V8. The area of dyssynergy in patients with abnormal Q waves reaching V6 or V8 was significantly greater (P < 0.05) than in patients with QS deflections limited to V1 to V3.

**Anterior and Inferior Infarction**

The area of dyssynergy of the left ventricle in the 21 patients with isolated anterior infarction determined by ECG changes was 42% (fig. 5B). In 28 patients with infarction limited to the inferior wall, determined by ECG changes, the extent of dyssynergy was 23% of the left ventricular silhouette, an area which was significantly (P < 0.05) less than in anterior infarction (fig. 5B). In 19 patients with ECG evidence of combined anterior and inferior infarctions the area of dyssynergy was 68%, which was significantly greater than that of isolated anterior (P < 0.05) or inferior (P < 0.02) infarction.

**ST Segment Elevation With T Wave Inversion**

In 15 patients with anterior infarction and pathologic Q waves extending to V6 or V8, the extent of dyssynergy was 48% of the left ventricular perimeter in the four patients with persistent convex upward ST segment and T wave inversion. In the remaining 11 patients without these specific ST-T alterations, the quantity of dyssynergy was

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**Figure 4**

The relation of persistent convex upward ST segment with T wave inversion in leads with pathologic Q waves (ST*T+) to pattern of dyssynergy in 77 patients with ECG infarction. Dyskinesis was more frequent with ST*T+ (P < .02) while hypokinesis was more common (P < .01) without these specific ECG changes (no ST-T).

**Figure 5**

A. Relation of the percent of left ventricular perimeter demonstrating dyssynergy (dyssynergy/LV silhouette) to the extent of pathologic Q waves in 19 patients with isolated anterior infarction and QS deflections beginning in V1 or V2. With abnormal Q waves extending to V6 or V8, a greater quantity of left ventricle demonstrated abnormal motion than when pathologic Q reached only V3 (P < .05).

B. Relation of the area of dyssynergy to ECG location of infarction. The percent of left ventricular perimeter with dyssynergy was greater with anterior infarction (AMI) than inferior (IMI) (P < .05), and larger with combined anterior and inferior infarction (A+IMI) compared to AMI (P < .05).
33%, an area significantly less \( P < 0.05 \) than that found in the patients with upward ST segments combined with T negativity. In the 19 patients with combined anterior-inferior infarction, the quantity of dyssnergy was 76% in 11 patients with upward ST and T negativity compared to 56% \( P < 0.05 \) in the remaining eight patients. The frequency of ST elevation with T inversion in all patients with isolated anterior infarction was 33% compared to 4% \( P < 0.05 \) in patients with isolated inferior infarction, a finding which was consistent with the larger area of dyssnergy observed in anterior relative to inferior infarctions.

**Discussion**

This investigation clearly demonstrates that a close relationship exists between specific findings on the standard 12 lead electrocardiogram, and the presence of abnormal left ventricular segmental wall motion and its characteristic features in patients with previous transmural myocardial infarction. When considering detection of dyssnergy, the electrocardiogram provides a highly sensitive means for its identification since 95% of patients with ECG evidence of transmural infarction demonstrated localized abnormalities of segmental contraction (table 1). Also, Q waves pathognomonic of infarction predicted disorder in regional wall motion in 87% of the patients with ventricular dyssnergy and coronary stenosis. The electrocardiogram is also relatively selective in identifying segmental dyssnergy since 76% of the patients with coronary obstruction without ECG evidence of infarction had normal wall motion. From these observations, the ECG reliably predicts the presence or absence of dyssnergy in 88% of patients with coronary disease (table 1).

In addition to pathologic Q deflections which relate to the presence of dyssnergy, the electrocardiogram contains characteristic alterations which allow description of the location, nature and extent of abnormal regional wall motion. Thus, the ECG location of pathologic Q waves correlates reliably with the site of ventriculographic dyssnergy. Most accurate in this regard is the ECG determination of anterior infarction which was associated with antero-apical dyssnergy in all patients. Nearly as specific is the ECG determination of inferior infarction which was accompanied by infero-apical dyssnergy in 89% of patients. In anterior-inferior infarction, abnormal wall motion was present in the antero-apical region in 89% of patients and antero-apical or inferior dyssnergy occurred in all patients. The angiographic findings in true posterior infarction were substantially less specific than in anterior or inferior infarctions: 60% of the patients exhibited disorders of inferior or apical wall motion. In posterior infarction the left anterior oblique view improved localization of the area of dyssnergy to the abnormal site as evidenced by electrocardiogram; this projection demonstrated a disorder in the contraction pattern of the posterior-lateral wall in three of the five patients, while the right anterior oblique view detected dyssnergy of the inferior segment in only one of these patients. However, in all 123 patients studied, dyssnergy was not observed in the left anterior oblique view when it was not observed in the right anterior oblique projection which is consistent with the experience of other authors.20, 21

In addition to identifying the site of dyssnergy, the nature of abnormal segmental contraction is also indicated by the ECG evidence of location of infarction (table 2), as well as by other characteristic changes on the electrocardiogram. Thus, the majority of patients with dyskinesis or akinesis exhibited ECG evidence of anterior infarction. Furthermore, 80% of the patients with ECG evidence of an anterior infarction demonstrated dyskinesis or akinesis. In contrast, ECG evidence of inferior or posterior infarction occurred in most patients with hypokinesis, and in all four patients with normal ventricular motion and pathologic Q waves. Also, 64% of patients with ECG evidence of inferior necrosis had hypokinesis. Thus, the qualitative severity of dyssnergy correlated with the site of infarction evidenced by electrocardiogram. Dyskinesis and akinesis usually result from anterior infarction while inferior infarction most frequently produces hypokinesis.

The dyssnergy pattern is also related to the extent of pathologic Q deflections in individual patients. Thus, in patients with anterior infarction, dyskinesis was more frequent in patients with the greatest lateral extension of abnormal Q waves (fig. 3). In addition, dyskinesis is essentially always associated with ECG evidence of transmural necrosis, whereas akinesis and hypokinesis are occasionally observed in the absence of transmural infarction (fig. 1 and 2).

Analysis of ST-T wave alterations further enhances determination of the nature of abnormal regional wall motion. In patients with ECG determination of transmural infarction, the finding of concomitant ST segment elevation with T wave inversion on leads with pathologic Q waves always

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designated dyssynergy. Moreover, the combination of convex upward ST with inverted T deflection in the presence of infarction indicated dyskinesis in 68% of patients with these characteristic ECG alterations (fig. 4). This finding is consonant with those of Gorlin and co-authors. Also consistent is our observation that infarction without ST elevation and T inversion as evidenced by electrocardiogram is usually associated with hypokinesis and only occasionally with dyssynergy (fig. 4).

The results of previous studies have been inconsistent concerning the ECG alterations associated with necropsy and angiographic evidence of aneurysm. Early reports described a relation between ventricular aneurysm found at postmortem examination and myocardial infarction evidenced by electrocardiogram with deep S waves in leads II and III. Other authors have noted a relation between ventricular aneurysm found at autopsy and ECG evidence of anterior wall myocardial infarction, intraventricular conduction disturbances, persistent precordial ST depression in the presence of inferior wall infarction, low QRS voltage and multiple pathologic Q waves. A recent study indicated a close correlation between ST segment elevation of 2 mm or more persisting in two or more ECG leads three weeks following acute myocardial infarction and angiographic evidence of left ventricular akinesis or dyskinesis. The frequency of persistent ST-T wave changes has differed widely from 1.5 to 79 percent of instances. Although certain reports have emphasized persistent ST segment elevation with infarction as pathognomonic of left ventricular aneurysm, the largest autopsy series concluded that there is no characteristic ECG pattern, a belief which has also been demonstrated by other studies. In contrast, our investigation correlates ECG findings with the dynamic features of abnormal segmental wall motion during life; dyssynergy is considered herein as a functional inadequacy of regional contraction and not necessarily limited to the fibrotic area of aneurysm seen by the pathologist.

In addition to determination of the presence, location and nature of abnormal regional contraction, the extent of left ventricular involvement with dyssynergy can also be predicted from the electrocardiogram. In patients with anterior myocardial infarction, the quantity of dysynergy was directly related to the precordial extent of pathologic Q waves (fig. 5A). This finding is consistent with postmortem studies in which abnormal Q waves in V5 and V6 correctly predicted infarction in more instances (96%) than in any other ECG zone. Furthermore, the ECG evidence of location of infarction is related to the size of dyssynergy. In anterior infarction, the area of abnormal motion was nearly twice the size of that observed in inferior infarctions, while the largest extent of dyssynergy occurred in combined anterior-inferior infarction (fig. 5B). Also, the quantity of dyssynergy was increased when ECG evidence of infarction was accompanied by ST segment elevation and T inversion. The greater frequency of upward ST with T negativity with anterior infarctions when compared to inferior infarctions is in agreement with the more extensive segment of disordered contraction in infarction of the anterior wall. In this regard, persistent ST segment elevation after myocardial infarction has been related to twice the mortality over a subsequent three-year period when compared to survivors of myocardial infarction without this specific ECG alteration. The decreased survival may result in part from a greater extent of muscle loss which is manifested electrocardiographically as persistent ST elevation.

Since the principal purpose of this study was to define the ECG correlates of segmental dysynergy in coronary artery disease, all patients included in the study had documented coronary obstruction. Therefore, our study was not designed to specifically evaluate the ability of the electrocardiogram to detect the presence or site of coronary artery stenosis. Other studies have shown that the resting electrocardiogram is an inaccurate predictor of coronary disease in individual patients. However, in the special circumstance of infarction evidenced by electrocardiogram, the resting electrocardiogram does indicate the location of coronary obstruction. Since the electrocardiogram relates to the status of the myocardium and not directly to the coronary arteries, it is not surprising that ECG disturbances indicating infarction correlate well with other properties of cardiac muscle, such as characteristics of ventricular wall abnormalities demonstrated in this study and cardiac pump function as indicated in a previous study.

Acknowledgments

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