Noninvasive Assessment of Cardiac Function and Ventricular Dyssynergy by Precordial Q Wave Mapping in Anterior Myocardial Infarction

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SUMMARY To determine whether multiple lead precordial electrocardiographic recordings offer an improved index for noninvasive estimation of left ventricular hemodynamic function and segmental dyssynergy, precordial mapping was performed in patients with anterior myocardial infarction, and the number of pathologic Q waves (≥0.04 sec) was counted (Q-Index). Left ventricular function was determined by cardiac catheterization and angiography and correlated with the Q-Index. The Q-Index correlated well with dyssynergy extent (r = 0.84) and inversely with ejection fraction extent of pathologic precordial Q complex mapping was correlated with left ventricular hemodynamics and angiography obtained by cardiac catheterization.

THE ADVENT OF CORONARY CARE UNITS (CCU) has resulted in a marked decline of dysrhythmic death following acute myocardial infarction. It is now recognized that the majority of remaining CCU mortality is due to myocardial infarction shock. The complication of left ventricular pump dysfunction is directly related to the extent of myocardial necrosis. Improved methods for the assessment of infarct size are necessary for management and prognosis. Myocardial enzymes, radionuclide imaging, echocardiography, standard electrocardiography and right heart catheterization have been utilized to provide important information concerning cardiac function and the extent of necrotic muscle in myocardial infarction. At present, however, left heart catheterization with angiography remains the most accurate means for assessing left ventricular performance and the area of infarction as indicated by abnormal segmental contraction (dyssynergy) unaltered by nitroglycerin. While the multiple precordial ST-segment blanket technique has been employed to estimate the zone of ischemia associated with acute infarction, it occurred to us that evaluation of the number of pathologic Q waves recorded by the multiple precordial blanket method might afford a relatively precise noninvasive modality for judging the actual extent of wall necrosis in patients with anterior myocardial infarction. Thus the present study was carried out to assess the validity and accuracy of this concept of precordial Q wave mapping in assessing infarct size, ventricular segmental dyssynergy and cardiac function in a large group of patients with acute and chronic coronary artery disease. To accomplish the objective of this investigation, the extent of pathologic precordial Q complex mapping was correlated with left ventricular hemodynamics and angiography obtained by cardiac catheterization.

Materials and Methods

Forty-eight patients with clinical, electrocardiographic and angiographic documentation of anterior myocardial infarction form the basis of this study. The study population included 38 males and 10 females, 37-72 years in age (mean 57 years). All patients had Q wave mapping with the 35 lead precordial ECG blanket within 24 hours of cardiac catheterization. The total number of pathologic Q waves (≥0.04 sec) detected by the precordial blanket was designated the Q-Index. In addition, the normal Q-Index was determined in 20 adult individuals without heart disease as documented by cardiac catheterization.

The precordial ECG blanket was constructed such that there were five horizontal rows of seven leads. For orientation, standard lead V1 corresponded to the extreme right lead of the third blanket row; lead V2 to the second lead of the same row; leads V3, V4 and V5 to the fourth, fifth and sixth vertical leads of the fourth blanket row; while lead V6 was between the second vertical column of the third row and the fourth column of the fourth row. Lead I of the blanket (extreme right lead of the first row) was placed in second right intercostal space parasternally, and the blanket was positioned over the left precordium with the remainder of the first row in the second left intercostal space laterally.

All patients were studied within three months of the acute episode. Fifteen of the 48 patients were evaluated within six weeks of their acute infarction, including all eight who were functional class IV by the New York Heart Association classification and seven who were in functional class III. Patients with ECG evidence of inferior myocardial infarction were excluded, as were those with intraventricular conduction disturbances other than right bundle branch block. None of the patients had chronic obstructive lung disease or clinical and electrocardiographic evidence of ventricular hypertrophy.
All patients underwent diagnostic retrograde left heart catheterization which included left ventriculography and selective coronary arteriography. Statham P23Db transducers were utilized for measurements of systemic arterial and intracardiac pressures; the zero reference point was placed at mid-thorax. Cardiac outputs were measured in duplicate by the indicator dilution method. The following hemodynamic parameters were measured or calculated: 1) left ventricular end-diastolic pressure (LVEDP) in mm Hg; 2) cardiac index (CI) in L/min/m²; 3) stroke index (SI) in cc/beat/m²; and 4) stroke work index (SWI) in gm · m/m² by the equation \((\bar{P} - \text{LVEDP}) \times \text{SI} \times 13.6\)/1000 where \(\bar{P}\) = mean systemic arterial pressure.

Biplane left ventricular cineangiograms of the 48 patients with stabilized anterior infarction were evaluated for abnormalities of left ventricular segmental contraction as reported in detail previously from our laboratory.13 Left ventriculography was performed in the 30° right and 60° left anterior oblique projections and recorded on 35 mm film taken at 64 frames/sec using the Philips nine-inch image amplifier system with optimal resolution provided by cesium iodide tubes. The ventricle was opacified with 50 to 75 cc of Hypaque-M 75 or 76% containing sodium and meglumine diatrizoates injected at 300 pounds 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.12, 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. To detect any abnormal wall motion due to ischemia rather than necrosis, a repeat left ventricular angiogram was carried out five minutes after 0.4 mg sublingual nitroglycerin.18

Specific patterns exhibiting localized abnormal left ventricular anterior segmental contraction were defined by the following criteria:19 akinesis — absent systolic movement of a segment of the wall; dyskinesis — paradoxical outward systolic expansion (ventricular aneurysm) in which a portion of the end-systolic silhouette extended outside the end-diastolic perimeter. Dyssnergy is used in this report as a general term signifying any of these localized disorders of wall motion. Combined types of anterior dyssnergy were

![Figure 1](http://circ.ahajournals.org/)

**Figure 1.** Representative tracings of precordial 35-lead Q wave maps obtained from anterior myocardial infarction patients A.W. and S.W. In patient A.W. the Q-Index (11 pathologic Q waves) was accompanied by cardiac index (CI) 2.4 L/min/m², stroke work index (SWI) 34.5 gm · m/m², ejection fraction (EF) 45% and area of left ventricular dyssynergy (LVD) 41%. In contrast, patient S.W. with Q-Index of 26 pathologic Q waves had CI 2.1 L/min/m², SWI 21.2 gm · m/m², EF 21% and LVD 69%.
designated by the quantitatively predominant pattern, and the total area of dyssnergy was included in the measurement of the extent of a disorder in wall motion. For the purpose of quantifying abnormal anterior segmental contractions the perimeter-distance of the anterior left ventricular end-diastolic silhouette showing dyssynergy showing unresponsive to nitroglycerin was expressed as a percentage of the total end-diastolic left ventricular perimeter.

Results

The hemodynamic and angiographic correlations with the pathologic Q-Index in the 48 postinfarction coronary patients are expressed in figures 1 and 2 and tables 1 and 2. In tables 1 and 2, three subsets of coronary patients were identified according to the total number of pathologic Q complexes found on the precordial blanket: group I had less than 15 Q waves, group II had 15–25 Q waves and group III had 26–35 Q waves.

Comparison of Pathologic Q-Index with Stroke Work Index

The Q-Index demonstrated an inverse linear correlation with SWI. Increase in the Q-Index from 6 to 34 was associated with a decline in SWI from 48.8 to 7.3 gm · m/m² (r = –0.79). These data are also expressed in average form in the three groups of coronary patients shown in table 1. Representative precordial blanket recordings in two coronary patients are depicted in figure 1. The Q-Index in normal individuals was found to average 3.2 complexes per patient (range 2 to 5); these values are not expressed in the figures.

Comparison of Q-Index with Cardiac Index

An inverse correlation was also observed between the Q-Index and the cardiac index (fig. 1 and table 1). Thus increase of the Q-Index from 6 to 34 complexes was accompanied by a decrease in cardiac index from 3.80 to 1.30 L/min/m² (r = –0.66).

Comparison of Q-Index with LVEDP and Stroke Index

The findings in this study demonstrated that LVEDP and SI were associated weakly (r = 0.18 and –0.31, respectively) with the total number of pathologic Q waves found by precordial mapping.

Comparison of Q-Index with Angiographic Ejection Fraction

As shown in figures 1 and 2A and table 1, an increase in the Q-Index correlated closely with decline in the left ventricular ejection fraction. Thus as the Q-Index rose from 6 to 34 the left ventricular ejection fraction was reduced from 69% to 12% (r = –0.87).

Table 1. Correlation of Q-Index to Left Ventricular Function

<table>
<thead>
<tr>
<th>Group</th>
<th>Number of patients</th>
<th>Q-Index</th>
<th>Cardiac index</th>
<th>Stroke work index</th>
<th>Ejection fraction</th>
<th>Percent LV dyssynergy</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>22</td>
<td>&lt;15</td>
<td>2.8 ± 0.1</td>
<td>36.6 ± 1.2†</td>
<td>52 ± 2.1†</td>
<td>39 ± 1.9†</td>
</tr>
<tr>
<td>II</td>
<td>16</td>
<td>15–25</td>
<td>2.4 ± 0.2</td>
<td>24.0 ± 1.8</td>
<td>36 ± 2.9</td>
<td>55 ± 3.3</td>
</tr>
<tr>
<td>III</td>
<td>10</td>
<td>26–35</td>
<td>1.8 ± 0.1††</td>
<td>16.8 ± 2.4**</td>
<td>19 ± 2.0††</td>
<td>72 ± 2.3††</td>
</tr>
</tbody>
</table>

Values represent mean ± standard error of mean.
††P < 0.001 group I to II.
‡‡P < 0.001 group II to III.
### FIGURE 2. Relation between Q-Index and ejection fraction (panel A) and left ventricular (LV) segmental dyssynergy unaltered by nitroglycerin (panel B).
Comparison of Q-Index with Angiographic Extent of Dyssynergy

Figure 2B demonstrates the close relationship between the Q-Index and quantity of abnormal segmental wall motion unresponsive to nitroglycerin. Thus the Q-Index rose proportionately with the extent of left ventricular dyssynergy from 24% to 82% of the chamber perimeter ($r = 0.84$). Thirty-six patients had predominant akinesis while the remaining 12 patients had principally dyskinesia. These data are consonant with the representative coronary patients shown in figure 1 and in the group values given in table 1.

Correlation of Q-Index with Clinical Status

The three groups (I, <15 pathologic Q waves; II, 15-25 Q waves; III, 26-35 Q waves) were compared according to their NYHA cardiac functional classification (table 2). The group III patients were found to have the worst degree of symptomatology (table 2) as well as the greatest impairment of cardiac function (table 1). Thus the 10 patients comprising group III were all severely limited in physical activity as indicated by their class III or IV functional status (table 2).

Correlation of Q-Index with Subsequent Survival

The relation of the three Q-Index coronary groups to subsequent survival was also evaluated in this study (table 2). Eleven patients died; their survival averaged 3.9 months (range 0.2–18 months). The 37 survivors were observed an average of 12.2 months (range 6–20 months).

The nature of death in each group and the functional status of the survivors were examined relative to their Q-Index. Of the 22 patients in group I, only two died (9%); one patient died suddenly after 11 months while the other death occurred in the early postoperative period following coronary artery bypass surgery. Thus in the group I patients in whom left ventricular function was least disturbed and in whom the size of segmental dyssynergy was the smallest (table 1), 20 of 22 individuals survived the period of observation. Ten of the survivors were asymptomatic and ten had mild functional impairment (table 2). Sudden death was defined herein as that occurring within six hours after the onset of symptoms and unrelated to cardiac failure and known noncardiac diseases. The diagnosis of sudden death was established in each instance by interviews with the patient’s relatives.

Six of the ten patients in group III died from cardiac pump failure in cardiogenic shock; survival averaged 19.8 days (range 6 to 52 days) (table 2). Therefore, in these patients with greatest disturbance of ventricular performance and the largest area of abnormal segmental motion (table 1), only four of ten patients survived the period of observation. Further, there were marked symptoms due to pump dysfunction in the four survivors.

Concerning the group II patients in whom left ventricular function and segmental dyssynergy were intermediate in abnormality between the group I and III patients (table 1), three of 16 patients (19%) died after 3.3 months (range 0.5 to 7 months) (table 2). Two deaths occurred because of chronic congestive heart failure, while one patient died suddenly. Six survivors had symptoms of cardiac disturbance with more than ordinary activity (class II) while six had heart failure symptoms with ordinary physical effort (class III). The remaining patient is asymptomatic (class I). Therefore, the Q-Index correlated with heart failure and severity of symptoms due to pump impairment.

Discussion

This investigation demonstrates that preordial multiple lead electrocardiographic mapping of pathologic Q waves in patients with anterior myocardial infarction is a valuable and reliable means for the noninvasive evaluation of hemodynamic status, quantity of abnormal segmental ventricular motion and extent of muscle damage. Thus, the Q-Index in these patients correlated closely with stroke work index, cardiac index and ejection fraction, and the area of segmental ventricular akinesia-dyskinesia. In addition, the Q-Index was found to accurately reflect the patients’ functional classification of physical activity. The aforementioned variables of cardiac function and clinical course were observed to have the same close correlation with the Q-Index in the settings of both acute and chronic coronary heart disease.

Cardiac catheterization with left ventriculography constitutes the most definitive method for assessing ventricular hemodynamic function and for identifying and quantifying the extent of abnormal chamber wall motion. In turn, the region of localized dyssynergy not responsive to nitroglycerin is known to closely reflect the area of infarcted myocardium. Furthermore, in coronary patients, intraoperative myocardial biopsies and epicardial electrograms have shown that nitroglycerin-irreversible areas of abnormal ventricular wall motion are comprised of replacement fibrosis and associated with local pathologic Q waves, whereas reversible segments are histologically normal without Q waves.

It is also important to delineate the pathophysiology of preordial Q wave mapping in the quantification of myocardial infarction. Thus studies in dogs following coronary occlusion have shown that the number of epicardial

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**Table 2. Correlation of Q-Index with Clinical Functional Status and Survival**

<table>
<thead>
<tr>
<th>Group</th>
<th>Number of patients</th>
<th>Q-Index</th>
<th>NYHA functional class</th>
<th>Mean follow-up</th>
<th>Mortality</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>22</td>
<td>&lt;15</td>
<td>I = 10</td>
<td>14.5 mo</td>
<td>2</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>I = 12</td>
<td>11.4 mo</td>
<td></td>
</tr>
<tr>
<td>II</td>
<td>16</td>
<td>15-25</td>
<td>I = 1</td>
<td>3.3 mo</td>
<td>3</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>I = 7</td>
<td>13.2 mo</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>III = 8</td>
<td>19.8 days</td>
<td>6</td>
</tr>
<tr>
<td>III</td>
<td>10</td>
<td>26-35</td>
<td>III = 2</td>
<td>12.3 mo</td>
<td>(60%)</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>IV = 8</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

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No
Pathologic Q waves reliably indicates the area of myocardial necrosis, as determined by biochemical (reduction of tissue creatine phosphokinase activity) and histologic criteria. Further, these experimental findings have been extended to use of the 35-electrocardiographic lead precordial blanket to evaluate the area of necrosis in patients with acute myocardial infarction undergoing interventions designed to limit the extent of infarct size. In addition, recent clinical studies have demonstrated the general correlation between the number of pathologic Q waves recorded by standard six-lead precordial electrocardiography and the extent of abnormal left ventricular segmental contraction by angiography in coronary patients with previous myocardial infarction. Since clinical investigations have demonstrated that the standard 12-lead electrocardiogram substantially understimates the presence of epicardial pathologic Q waves, the 35-lead electrocardiographic precordial blanket employed in the present study constitutes a more thorough approach for the detection of the extent of the infarcted area as well as identification of zones of necrosis unrecognized by conventional electrocardiography.

Our data indicate that cardiac function can be evaluated noninvasively by application of precordial multiple lead Q wave mapping. Previous studies of coronary patients with myocardial infarction from our laboratories have shown only a general correlation between the number of pathologic Q waves, obtained by the standard 12-lead electrocardiogram and the conventional six-lead precordial electrocardiogram, and the extent of abnormal left ventricular segmental contraction and abnormal variables of ventricular function. In addition, our previous investigations have shown that persistent ST-segment abnormalities following infarction indicate a more severe nature and a generally more extensive area of localized segmental dyssynergy. The resulting pathologic Q wave index obtained from the 35-electrocardiographic lead precordial blanket in the present study correlated considerably better than standard electrocardiography with left ventricular stroke work index, cardiac index, ejection fraction and extent of segmental dyssynergy, parameters which have been shown by a number of investigators to relate accurately to patient prognosis in coronary heart disease.

Concerning clinical prognosis in coronary disease, Swan and colleagues observed that stroke work greater than 66 gm · m/m² was associated with survival in acute infarction, while a value less than 36 gm · m/m² occurred in non-survivors. Scheidt and co-workers found that following myocardial infarction the mean cardiac index was reduced to 1.1 L/min/m² in patients with pump failure and shock, whereas patients without ventricular dysfunction had cardiac indices averaging 2.5 L/min/m². Our experience has been consistent with these observations. Thus, in a series of 287 patients with acute myocardial infarction we have observed that the clinical course and prognosis can be classified according to initial hemodynamics into four groups: 1) SWI > 40 gm · m/m² indicated an uncomplicated course with patients remaining essentially functional class I; 2) SWI between 28–40 gm · m/m² was found in patients having mild to moderate functional limitations who were class II; 3) SWI between 18–28 gm · m/m² occurred in functional class III or IV patients who had severe congestive heart failure; and 4) SWI less than 18 gm · m/m² was observed in patients with marked pump dysfunction and cardiogenic shock.

In addition to demonstrating the close correlation between the number of pathologic Q waves by multiple lead precordial mapping and the important variables of cardiac function (fig. 2), to translate our findings for more convenient clinical usefulness, the Q-Index was divided into three groups which were related to hemodynamic function (table 1) and to clinical status and prognostic outcome (table 2). Group I (<15 pathologic Q complexes) patients had a normal cardiac index, normal ejection fraction, mild reduction of stroke work index and approximately 40% localized dyssynergy in the right anterior oblique view. Group II patients (15 to 25 pathologic Q waves) had 55% abnormal segmental wall motion and moderate depression of stroke work index and ejection fraction while cardiac index was slightly decreased. The group III patients, who had the largest number of pathologic Q waves (26 to 35 such complexes), demonstrated more than 70% segmental akinesis dyskinesia accompanied by markedly diminished stroke work index, ejection fraction and cardiac index.

From these observations it is readily apparent that a progressively greater Q-Index group was associated with steady worsening of cardiac performance and increasing area of abnormal wall motion. Therefore, the Q-Index group provides a valuable noninvasive means for the estimation of the degree of impairment of ventricular function as well as the extent of segmental dyssynergy indicating infarct size in anterior infarction patients with acute and chronic coronary artery disease. An additional role of the clinical usefulness of the Q-Index is that in group I or II patients any wide disparity between the individual's status and Q-Index should arouse suspicion of complicating factors relative to pump function other than ventricular necrosis per se, such as mitral regurgitation, septal rupture, pericardial tamponade, aortic dissection, pulmonary embolism and blood volume depletion.

Besides the ability of Q-Index grouping to afford valuable information concerning ventricular function and estimation of infarct size in anterior myocardial infarction, it was found herein that the Q-Index groups correlated closely with patient clinical status and future complications relative to pump integrity (table 2). Thus, group I patients' physical activity functional classification was normal (class I) or mildly compromised (class II), and none of them died of pump failure. Contrary to group I, nearly all of the group II patients were impaired in physical effort, the majority experiencing symptoms of pump dysfunction even with ordinary activity (class III). Two group II patients died from chronic congestive heart failure while the remaining death was sudden.

All group III individuals were severely impaired in physical activity with most having heart failure symptoms at rest (class IV). More than one-half of these patients died within a short period of time because of refractory pump failure or cardiogenic shock. From these data the higher the Q-Index group classification, the greater was the propensity for functional clinical deterioration and mortality. Thereby the Q-Index allowed prediction of both immediate functional status as well as eventual demise from pump dysfunction in coronary patients with acute and chronic anterior myocardial infarction.
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References
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