Identification of First Acute Q Wave and Non-Q Wave Myocardial Infarction by Multivariate Analysis of Body Surface Potential Maps

Fred Kornreich, MD; Terrence J. Montague, MD; and Pentti M. Rautaharju, MD, PhD

Background. Patients with acute non-Q wave myocardial infarction (NQMI) appear to have more jeopardized residual myocardium at high risk for subsequent angina, reinfarction, or malignant arrhythmias than patients with acute Q wave myocardial infarction (QMI). Unfortunately, conventional electrocardiographic (ECG) criteria have limited utility in recognizing NQMI.

Methods and Results. The present study combines the increased information content of body surface potential maps (BSPM) over the 12-lead ECG with the power of multivariate statistical procedures to identify a practical subset of leads that would allow improved diagnosis of NQMI. Discriminant analysis was performed on 120-lead data recorded simultaneously in 159 normal subjects and 308 patients with various types of myocardial infarction (MI) by using instantaneous voltages on time-normalized P, PR, QRS, and ST-T waveforms as well as the duration of these waveforms as features. Leads and features for optimal separation of 159 normals from 183 patients with recent or old QMI (group A) were selected. A total of six features from six torso sites accounted for a specificity of 96% and a sensitivity of 94%. All lead positions were outside the conventional electrode sites and selected features were voltages at mid-P, early and mid-QRS, and before and after the peak of the T wave. The discriminant function was then tested on 57 patients with acute NQMI (group B) and 68 patients with acute QMI (group C); rates of correct classification were 91% and 93%, respectively. Because of the possible deterioration of the results caused by ST-T abnormalities also present in other clinical entities, a second classification model including an independent group of 116 patients with left ventricular hypertrophy (LVH) but without MI was developed. Two additional measurements were required, namely, P wave duration and a mid-QRS voltage on a lead located 10 cm below V1. Testing the model on both acute MI groups produced correct classification rates of 88% for acute NQMI and 93% for acute QMI. Group mean BSPM were plotted for the three MI groups at successive instants throughout the PQRST waveform. Typical patterns for each MI group were identified during PQRST by removing the corresponding normal variability at each electrode site from sequential MI maps. These standardized maps or discriminant maps provided information on the capability of each measurement at each electrode site and at each instant to separate each class of MI from the normal group (N). Striking similarities were observed between the three MI groups, particularly at mid-QRS and throughout ST-T. The closest resemblance was between acute NQMI and old QMI. Discriminant analysis was also performed on the 12-lead ECG: The first classification model (N versus MI) produced correct classification rates of 85% for acute QMI and 70% for NQMI. With the second model (MI versus N or LVH), correct rates were 81% and 65%, respectively.

Conclusions. Diagnosis of acute NQMI and QMI (also in the presence of LVH) can be improved substantially by appropriate selection of ECG leads and features. Comparison of discriminant maps from groups A, B, and C does not support the concept of acute NQMI as a distinct ECG entity but rather as a group with infarcts of smaller size. However, pathophysiological and clinical differences between acute NQMI and acute QMI influence long-term risks and may define different therapeutic approaches. (Circulation 1991;84:2442-2453)
The electrocardiographic terms Q wave myocardial infarction (QMI) and non-Q wave myocardial infarction (NQMI) have become accepted clinical designations for classifying acute myocardial infarction (MI), supplanting the use of "transmural" and "subendocardial" infarction. The present terminology, though convenient, has a major limitation. The limitation deals with the lead system used: Because only a fraction of the total available surface potential information is sampled by the standard 12-lead electrocardiogram (ECG), no distinction can be made between "missed" QMI (presence of abnormal Q waves outside the conventional electrode sites) and "true" NQMI (absence of abnormal Q wave anywhere on the thoracic surface).1

Despite some controversy regarding the validity of the present classification as representing distinct pathophysiological and clinical entities, a recent review of the literature by Montague et al2 indicates significant differences between QMI and NQMI populations, particularly regarding the pathophysiology of the infarct-related artery, long-term prognosis, and changing incidence. In view of the higher rate of subsequent coronary events, particularly reinfarctions, patients with acute NQMI may benefit from early interventional therapy. Early recognition of the presence of the condition and development of methods for estimating the site and the extent of the lesions therefore have important clinical value.

The electrocardiogram is the easiest, most commonly available low-cost noninvasive procedure for bedside monitoring in the acute clinical setting; unfortunately, conventional ECG criteria have limited usefulness for identification of NQMI.3 Body surface potential maps (BSPM) have shown promising results in the recognition and quantification of MI.4-10

The present study compared 120-lead BSPM data from 125 patients with acute MI (57 patients with acute NQMI and 68 patients with acute QMI), 183 patients with recent (29 patients) or old QMI (154 patients), and 159 normal control subjects. Multivariate analysis was performed on the 120-lead data to identify a subset of leads and features for optimal classification of acute MI (with or without Q waves) regardless of the site of necrosis. Typical map patterns from NQMI, acute QMI, and old QMI patients were analyzed, and interpretation criteria were developed. Multivariate analysis was also performed on the standard 12-lead ECG in the same groups, and the diagnostic performances were compared with those of BSPM.

Methods

Study Population

We retrospectively studied 583 subjects, 159 of whom were normal, 308 patients with first MI, and 116 with pure left ventricular hypertrophy (LVH) (Table 1). None of the normal subjects had evidence of heart disease by history, physical examination, 12-lead ECG, and, when available, echocardiogram. They were all older than 30 years (mean, 43 years). All patients with MI had a typical history of prolonged, ischemic-type cardiac pain and characteristic changes in enzyme levels. In most cases, the diagnosis was further substantiated by coronary angiography and ventriculography, echocardiography, or nuclear angiograms during which multigated, technetium-99m-labeled blood pool imaging was performed. Radionuclide images in the two acute MI groups were collected between the fourth and the sixth hospital day. All patients had at least one 12-lead ECG recorded in the acute phase of the infarction. Patients were excluded if they had ECG evidence of complete left or right bundle branch block, major nonspecific intraventricular conduction delay (QRS, 126 msec or longer), previous MI, or Wolff-Parkinson-White syndrome. The MI population was classified according to the appearance of the QRS complex in the acute phase. Patients were classified as having anterior MI if they had Q waves at least 30 msec in duration in leads I, aVL or V1 to V6, or initial R waves of 0.2 mV or less in V1 and V2 (111 patients). Patients were classified as having inferior (posterior) MI if abnormal Q waves were present in leads II or aVF or if R waves in V1, V2 were exceedingly tall (R to S ratio, 1 or more) or broad (40 msec or more) (140 patients). The NQMI group consisted of 57 patients in which the above described criteria in the 12-lead ECG were absent. Subjects in the MI population were 34–81 years old (mean, 56 years); 68 (22%) patients were women and 240 (78%) were men. Patients with LVH had either pure left-sided valvular disease or sustained hypertension (150/90 mm Hg or higher). The presence of LVH was assessed using ECG-independent information: echo-

<table>
<thead>
<tr>
<th>TABLE 1. Study Population</th>
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<tbody>
<tr>
<td>Normal subjects</td>
</tr>
<tr>
<td>Acute QMI</td>
</tr>
<tr>
<td>Acute QMI</td>
</tr>
<tr>
<td>Anterior</td>
</tr>
<tr>
<td>Inferior</td>
</tr>
<tr>
<td>Recent or old QMI</td>
</tr>
<tr>
<td>Anterior</td>
</tr>
<tr>
<td>Inferior</td>
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</table>

NQMI, non-Q wave myocardial infarction; QMI, Q wave myocardial infarction.
cardiography, cardiac catheterization with coronary angiography and ventriculography, radionuclide angiography, chest radiographs, and cardiac surgery.

Body Surface Mapping

We used previously described methods of recording, processing, and displaying body surface electrocardiographic signals. Briefly, digitized signals were recorded simultaneously from 117 torso and three limb leads with Wilson’s central terminal as reference potential at 500 samples/sec per channel. Tracing quality was monitored visually during the recording; later, the stored data were processed by performing selective averaging and were again carefully inspected and edited. All leads judged invalid were deleted and replaced by interpolation of data from surrounding leads. We time-normalized separately the QRS waveform and the ST-T waveform and represented them by 70 and 180 points, respectively. Patients with MI were studied a mean of 10 months after MI (range, 1 day to 24 months). Further subdivision of the MI population was based on the interval between the acute event and the recording of BSM (Table 1): The interval was less than 1 week (range, day 1–6; mean, day 5) in 125 patients (68 with acute QMI and 57 with acute NQMI), between 1 week and 1 month in 29 (recent MI), and more than 1 month in 154 (old QMI); the latter two subgroups were pooled into one single group (183 patients), referred to as the old QMI group, for further analysis.

Map Display

Isopotential maps. Group mean and individual maps are represented during the PQRT complex as isounit lines, connecting points of equal voltage, and sign at selected time instants. Group mean maps were computed separately for the normal group and each MI group by averaging potentials at each instant at each electrode site from all subjects in each group. Sequential maps were obtained by sampling time-normalized P, PR, QRS, and ST-T waveforms at equal intervals; this resulted in 8, 8, 18, and 18 instants for P, PR, QRS, and ST-T, respectively. Discriminant maps. For each time instant, difference maps were computed by subtracting at each electrode site the normal group mean voltage from each MI group mean voltages; sequential discriminant maps for each pairwise comparison were obtained by further dividing each resulting difference by the corresponding composite standard deviation computed from the pooled groups. The values thus achieved, referred to as discriminant indexes, were strictly proportional to a test statistics and provided, in contrast with difference maps, information on the capability for each measurement at each electrode site and at each instant to separate each class of MI from the normal group. Discriminant maps were also represented as contour lines, connecting discriminant indexes of equal value and sign at each instant. Individual maps were computed in a similar way, subtracting from the patient’s voltage data the normal group mean voltages and dividing the resulting differences by the standard deviation of the normal population at corresponding sites and instants.

Feature Extraction and Statistical Analysis

We used instantaneous voltage measurements obtained by sampling the time-normalized waveforms at equal intervals; this resulted in 8, 8, 18, and 18 samples for P wave, PR segment, QRS, and ST-T waveforms, respectively (i.e., a 52×120-measurement matrix per subject). Added to these variables were P, PR, QRS, and ST-T durations measured before time normalization. Programs of the Biomedical Program Library were used for each bigroup classification (BMD7M). The statistical procedure involved two steps: First, the best classifiers were selected from the total number of available features by a stepwise selection; second, the selected classifiers were combined into a properly weighted linear discriminant function. For each group considered in the discriminant procedure, a group mean discriminant score was also computed; each group mean score was obtained by averaging individual scores (calculated from the sum of products of the standardized values of the selected measurements times the corresponding discriminant coefficients) of all patients in the group. The difference between group mean scores represented the multivariate distance, expressed in standard deviation units, separating these groups. To test the robustness of the discriminant function, the jackknife option, available in the BMD7M package, was also used. With this procedure, each case is eliminated in turn from the computation and subsequently assigned to one of the groups formed by the remaining cases. Because of the very large number of variables (6,244 per subject), the program processed a maximum of 80 input variables at a time and selected the 20 best from each batch, repeating the procedure until it acquired a final set of 20 optimal classifiers. Varying the composition of the batches and their order of entry did not affect the first six classifiers selected.

Results

Identification of Acute NQMI

Multivariate analysis was performed for optimal separation of 57 patients with acute NQMI from 159 normal subjects. Table 2 lists the results of the discriminant analysis. Only the first five variables are tabulated because little improvement was noted beyond this point. The lead positions from which the listed variables originate are shown in Figure 1. The bulk of the separation (92% correctly classified subjects) is achieved with the first three variables, namely, a voltage measurement on the downslope of the T wave in the left flank (lead 86), the duration of the ST-T segment (the mean ST-T duration is 346 msec in the NQMI group versus 310 msec in the normal group; p<0.001), and a mid-QRS voltage measurement in the lower right anterior torso (lead 4). Figure 2 depicts the discrimi-
TABLE 2. Differentiation Between 159 Normal Subjects and 57 Patients With NQMI

<table>
<thead>
<tr>
<th>Stepwise selection of variable type</th>
<th>Specificity (%)</th>
<th>Sensitivity (%)</th>
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</thead>
<tbody>
<tr>
<td>L 86, ST-T 13/18</td>
<td>92</td>
<td>75</td>
</tr>
<tr>
<td>ST-T duration</td>
<td>93</td>
<td>81</td>
</tr>
<tr>
<td>L 4, QRS 9/18</td>
<td>94</td>
<td>87</td>
</tr>
<tr>
<td>L 15, P 5/8</td>
<td>94</td>
<td>89</td>
</tr>
<tr>
<td>L 6, ST-T 13/18</td>
<td>95</td>
<td>91</td>
</tr>
</tbody>
</table>

NQMI, non-Q wave myocardial infarction; L, lead.

Numbers after name of waveform identify the instantaneous measurement on that waveform.

Multivariate classifier derived from measurements on 120-lead data.

nant maps corresponding to the time instants at which optimal measurements were obtained. At 13/18 ST-T, negative discriminant indexes exceeding two standard deviations are observed in the left flank where lead 86 is measured and correspond to reduced positive T voltages in the NQMI group; the area encompassing these indexes fails to include the standard lead positions. Only V₄ to V₆ have indexes between one and two standard deviations. A region of significant reciprocal positive indexes and images, corresponding to negative T voltages of smaller amplitude in the NQMI group, is located in the upper right anterior and posterior chest. Lead 6, which is measured in this area, contributes independent information to the discriminant function despite its apparent symmetry with lead 86. Voltage measurements at 5/8 P in and around lead 15 at the right of V₁ also contribute to the discriminant model but do not produce indexes equal to or greater than 1. Discriminant maps from early QRS (5/18 QRS) and early T (9/18 ST-T) are also depicted although they do not contribute to the NQMI versus normal separation. For comparative purposes, BSPM and discriminant maps are also displayed for the old QMI group (Figure 3) and the acute QMI group (Figure 4) and will be referred to in the next paragraph.

Classification of Acute MI

Selection of optimal leads and features for identifying patients with acute NQMI has only limited value in a clinical setting where the immediate concern is recognition of the acute condition whether Q waves are present or not. Moreover, the computed discriminant function, based on a relatively modest sample size of 57 patients, may lack the robustness required for reliable classification of new patients. A similar consideration precludes the use of the acute QMI group (68 patients) for the computation of a classification model. We therefore turned to the larger group of predominantly old QMI (183 patients) as a training set and computed a discriminant function separating these patients from normals. Both acute MI groups were then used as testing sets. The results of the analysis are shown in Table 3 and the lead positions from which the classifiers originate are depicted in Figure 1. BSPM and discriminant maps at instants corresponding to the selected measurements are shown in Figure 3. Several observations are in order: 1) The lead set and measurements selected for optimal separation of old QMI from normals include the lead positions and features (except ST-T duration) previously identified for the acute NQMI group; this explains why similar rates of correctly classified NQMI patients were found with both models. The second model contains two additional measurements, namely, an early QRS voltage in the dorsal region and a late ST or early T voltage in the left flank slightly above and to the left of V₆. 2) At a specificity level of 96%, 94% of old QMI are recognized using measurements from six lead positions; at the same time, 91% of acute NQMI are correctly classified with this same classifier as well as 93% of acute QMI. 3) Only features measured at the end of the T wave (ST-T 13/18) in the left flank (lead 86) and in the right subclavicular area (lead 6) produce discriminant indexes greater than two standard deviations and are potent classifiers in the univariate sense; other measurements have corresponding indexes of smaller magnitude (e.g., mid-P and mid-QRS voltages) and contain less diagnostic information. 4) Interestingly, similar patterns are observed in discriminant maps from comparable instants in the three MI groups (Figures 2, 3, and 4). The resemblance is particularly striking between the NQMI and the old QMI groups with nearly identical
BSPM and discriminant map patterns in mid-P and mid-QRS and throughout ST-T. Although map patterns from the acute QMI group also bear a close resemblance to those from the old QMI group, they differ mainly quantitatively as evidenced by the increased magnitude of corresponding discriminant indexes (two or greater) in early and mid-QRS; P wave indexes are also increased in acute QMI, whereas ST-T indexes remain practically unchanged in all three MI groups.

**ST-T Abnormalities and Left Ventricular Hypertrophy**

Three of six classifiers are T wave measurements (Table 3). Moreover, a relatively large number of patients from both acute MI groups had coexisting LVH (Table 4). In a general clinical setting where several diagnostic categories have to be considered at a time, the risk of wrongly calling patients with ST-T abnormalities but no MI, "MI," or labeling MI patients as "LVH without MI" was particularly high. We therefore performed multivariate analysis by computing discriminant functions at each of the two nodes of a decision-tree structure, considering normals versus old QMI in the first node and old QMI versus a group of 116 patients with pure LVH in the second node: 96% of the normals, 93% of the infarctions, and 85% of the hypertrophic patients were correctly classified. The discriminant function in the latter node required two new measurements reported earlier as important markers of LVH,\(^{14}\) namely, a mid-QRS voltage in lead 24 (Figure 1) and the P wave duration; aVF was found better suited for LVH than lead 4 and could also be substituted for that lead in the normal versus old QMI separation without deterioration of the classification. Patients from both acute MI groups were then directed through the two nodes of the classification model: Despite some loss at each node, group assignment rates were 93% for acute QMI and 88% for NQMI. It thus seems that the multivariate classifiers developed for the MI versus normal separation retain their stability and classification accuracy also in the presence of LVH with ST-T "strain" patterns.

**The Standard 12-Lead ECG**

Discriminant analysis was similarly performed on the 12-lead ECG. Variables again consisted of 8, 8, 18, and 18 instantaneous voltage measurements on the P, PR, QRS, and ST-T waveforms from each of the 12 leads plus the durations of these waveforms. In
Presence or Absence of Q Waves

Myocardial necrosis is accompanied by loss of electrical forces previously generated by the intact tissue. Negative voltages are recorded in electrodes facing the infarcted area when the loss is complete; when viable cells are still present in the injured area, reduced voltages will be recorded.\textsuperscript{15,16} The new balance of electrical forces will also produce reciprocal increases in potential in leads spatially opposite from the infarcted area.\textsuperscript{17} However, the complexity of the conducting medium, the variability of the heart and torso geometry in relation to electrode positions, the alterations in the activation sequence, and the size, degree, and location of the lesion are factors that greatly influence transmission of abnormal epicardial potential distributions to the body surface.\textsuperscript{18,19} As a consequence, the classical electrical signs of necrosis may not be visible in surface ECG recordings (true NQMI) or visible only in thoracic regions not explored by the conventional precordial leads (missed QMI). In a study relating the presence of abnormal Q waves in the 12-lead ECG with postmortem evidence of myocardial infarction or scar, Horan et al\textsuperscript{20} observed a sensitivity of only 61\% with a specificity of 89\%. Abnormal Q waves could also be generated by slowed intramural conduction and late activation of surviving epicardial tissue overlying subendocardial infarction.\textsuperscript{21} Moreover, the pathway of ventricular depolarization determines the type of QRS changes caused by infarction: Infarctions located in those parts of the ventricular walls where activation starts later than 30 msec will not produce early QRS abnormalities.\textsuperscript{22,23} This is particularly true for posterior and lateral lesions. In a recent study, Mirvis et al\textsuperscript{24} produced transmural as well as nontransmural necrosis by occluding the left circumflex artery in dogs and concluded that both types of infarction

![Diagram of ECG tracings and surface potential maps](http://circ.ahajournals.org/)

**FIGURE 3.** Group mean body surface potential maps of patients with old Q wave myocardial infarction (old MI) and normal subjects and corresponding discriminant maps for the separation of normal subjects from patients with old Q wave myocardial infarction (DM old MI). Maps are selected at instants 5/8 on the P wave, 5/18 and 9/18 on the QRS waveform, and 9/18 and 13/18 on the ST-T waveform. Left half of each map represents anterior torso and right half represents the back. Small circles correspond to \( V_1 \) through \( V_6 \) standard electrode positions. Top of the map is at the level of the sternal notch and bottom is at the umbilical level. Contour lines in surface maps are drawn for voltages \( \pm 20, 40, 80, 160, 320, 640, \) and \( 1,280 \) \( \mu V \). Contour lines in discriminant maps are drawn with increments of 0.5 SD (see text). Solid lines indicate positive values; dashed lines, negative values. In discriminant maps, the negative area encompassing discriminant indexes \( \pm 2 \) SD are blackened for emphasis.
altered R and S wave potentials and that the major determinant of these QRS changes was the size of the necrotic lesion. In a related study, Mirvis et al.\textsuperscript{25} investigated the anatomic determinants of abnormal Q waves in dogs. They found no difference in incidence of Q wave changes in dogs with induced nontransmural and those with transmural lesions. They demonstrated, however, that infarct size and depth of necrosis were significantly greater in dogs with than in those without Q wave changes.

In the present study, 56% of the NQMI patients had abnormal negative or reduced voltages or excessive positive voltages during the first half of QRS, inscribing abnormal Q waves, reduced R waves, or abnormally tall R waves in leads located outside the conventional electrode positions (missed QMI); the remaining patients had either early QRS changes of short duration, mid- or late QRS changes, or exclusively ST-T abnormalities (true NQMI). Because of the wide spatial scatter of abnormal patterns in NQMI and the resulting cancellation when averaging was performed, group mean maps failed to demonstrate a common, group-related abnormal pattern during the first half of QRS. In contrast, the more homogeneous distribution of larger and often overlapping areas of excessive loss of voltages in the acute QMI patients produced significant discriminant patterns in early to mid-QRS similar to those previously.

\begin{table}[h]
\centering
\caption{Differentiation Between 159 Normal Subjects and 183 Patients With Recent or Old QMI}
\begin{tabular}{|c|c|c|c|c|}
\hline
Stepwise selection of variable type & Specificity (%) & Sensitivity (%) & Correctly classified acute MI & NQMI (%) & QMI (%) \\
\hline
L 6, ST-T 13/18 & 86 & 88 & 83 & 86 & \\
L 99, QRS 5/18 & 91 & 90 & 83 & 88 & \\
L 4, QRS 9/18 & 94 & 92 & 84 & 90 & \\
L 15, P 5/8 & 95 & 91 & 86 & 90 & \\
L 78, ST-T 9/18 & 95 & 94 & 88 & 91 & \\
L 86, ST-T 13/18 & 96 & 94 & 91 & 93 & \\
\hline
\end{tabular}
\end{table}

QMI, Q wave myocardial infarction; MI, myocardial infarction; NQMI, non-Q wave myocardial infarction; L, lead. Numbers after name of waveform identify the instantaneous measurement on that waveform. Multivariate classifier derived from measurements on 120-lead data; accuracy of the same classifier in correct identification of 57 patients with acute NQMI and 68 patients with acute QMI.


Table 4. Comparison Between Clinical Variables in Acute NQMI and Acute QMI

<table>
<thead>
<tr>
<th>Comparison</th>
<th>Acute QMI</th>
<th>Acute NQMI</th>
</tr>
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<tbody>
<tr>
<td>Age (years)</td>
<td>54 (39–78)</td>
<td>57 (34 –81)</td>
</tr>
<tr>
<td>n</td>
<td>68</td>
<td>57</td>
</tr>
<tr>
<td>Coronary angiography</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Stenosis≥70%</td>
<td></td>
<td></td>
</tr>
<tr>
<td>1 Vessel</td>
<td>50%</td>
<td>27%</td>
</tr>
<tr>
<td>2 Vessels</td>
<td>42%</td>
<td>33%</td>
</tr>
<tr>
<td>&gt;2 Vessels</td>
<td>8%</td>
<td>40%</td>
</tr>
<tr>
<td>Vessel involved</td>
<td></td>
<td></td>
</tr>
<tr>
<td>LAD</td>
<td>58%</td>
<td>53%</td>
</tr>
<tr>
<td>RCA</td>
<td>50%</td>
<td>86%</td>
</tr>
<tr>
<td>LCx</td>
<td>25%</td>
<td>74%</td>
</tr>
<tr>
<td>Wall motion abnormality</td>
<td></td>
<td></td>
</tr>
<tr>
<td>0 Segments</td>
<td>0%</td>
<td>26%</td>
</tr>
<tr>
<td>1 Segment</td>
<td>12%</td>
<td>32%</td>
</tr>
<tr>
<td>&gt;1 Segment</td>
<td>88%</td>
<td>42%</td>
</tr>
<tr>
<td>Ejection fraction</td>
<td>37±16</td>
<td>57±19</td>
</tr>
<tr>
<td>First year</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Interventions</td>
<td>26%</td>
<td>16%</td>
</tr>
<tr>
<td>Deaths</td>
<td>18%</td>
<td>7%</td>
</tr>
<tr>
<td>Hypertension</td>
<td>(≥150/90 mm Hg)</td>
<td>29%</td>
</tr>
<tr>
<td>Increased echocardiographic LV mass</td>
<td>37%</td>
<td>58%</td>
</tr>
</tbody>
</table>

NQMI, non–Q wave myocardial infarction; QMI, Q wave myocardial infarction; LAD, left anterior descending artery; RCA, right coronary artery; LCx, left circumflex artery; LV, left ventricle.

described in both anterior and inferior MI and accounting for the inscription of Q waves or reduced R waves in corresponding leads. These changes also appear, albeit to a lesser degree, in the old QMI group: Q waves may indeed regress or even disappear in the weeks or months after the actual infarction as a consequence of the healing process, so the fibrotic infarctional scar has a smaller area than the original necrosis. In the present old QMI population, 18% of the patients (33 of 183) with Q waves in the acute phase no longer revealed typical Q waves at the time of BSPM recording. The reduced size and extent of anatomical necrosis in old QMI and the disappearance of transient Q waves consecutive to severe and prolonged ischemia in areas surrounding irreversibly necrotic tissue resulted in map abnormalities of smaller perimeter and magnitude than those observed in acute QMI but resembling those seen in NQMI patients.

Optimal Leads and Features

The leads selected for the recognition of NQMI are located in areas not interrogated by the standard V1 to V6 ECG leads. Interestingly, these leads constitute a subset of the leads found optimal for the diagnosis of recent or old QMI and are perfectly suited for acute QMI as well. The best measurements for NQMI are a mid-QRS voltage and a late T voltage. The former corresponds to reduced amplitude of S waves in the upper anterior torso and of R waves in the inferior border around the chest; the latter indicates reduced voltages of the T wave in the left flank and in the upper right anterior and posterior chest. As noted earlier, abnormal Q waves or Q wave equivalents were observed over a wide range of areas on the torso in a majority of NQMI patients; similarly, late abnormal QRS patterns were scattered around the thoracic surface in 35% of the NQMI patients. As a consequence of this heterogeneity, no common classifiers could account for abnormalities occurring during either the first or the last third of QRS. In QMI, both acute and old, early, and mid-QRS measurements were selected for classification purposes: Despite a certain amount of cancellation produced by pooling patients with anterior and inferior infarction, patterns characteristic of both groups could still be observed. Discriminant maps from NQMI patients showed predominantly inferior (posterior) MI patterns although anterior and apical wall motion abnormalities were found in individual patients; however, abnormal wall motion (present in 74% of the subjects) was noted more frequently and consistently in inferoapical and posterolateral segments than in anterior segments. At mid-QRS, all three MI groups displayed a loss of potentials in the anterior inferior torso with reciprocal changes in the upper chest: These features were suggestive of apical involvement that occurred frequently regardless of the type of infarct. These regions are poorly sensed by the conventional leads, which explains the low sensitivity of the 12-lead ECG in recognizing apical infarction. Mid-to-late depolarization patterns may also be ascribed to delayed activation caused by alterations in the pathway of activation or changes in conduction velocity. Several investigators have shown the diagnostic usefulness of voltage measurements from the second half of QRS in the 12-lead ECG and in the XYZ leads and have incorporated these measurements in their diagnostic and/or quantitative model. Patterns throughout ST-T were practically identical in the three MI groups and reflect reduced ST-T voltages in the left flank. Schechtman et al demonstrated recently that ST segment depression was an independent predictor of mortality between hospital discharge to 3 months after NQMI; they attributed, in part, ST depression to ischemia because it could not be accounted for by mechanical damage alone. The single best measurement for recognizing any type of MI was a late T voltage in leads located either in the right subclavicular area or, symmetrically, in the left flank (Table 3 and Figures 2, 3, and 4). Recently, Ishikawa et al studied 48 patients with angina pectoris at rest (without MI) and various types of one-vessel disease and concluded that T wave maps were able to localize the involved coronary artery and its associated area of ischemia in one half of their patients; moreover, all T map abnormalities were different from those in patients with left ventricular overload. Mid-P wave changes,
indirectly reflecting mechanical dysfunction of the left ventricle, which, in turn, may cause left atrial conduction disturbances without left atrial enlargement, were also similar in the three groups. Most of the relatively small differences observed between NQMI and other MI groups were quantitative rather than qualitative and could be explained by the smaller size and the location of the lesions.

A distinctive feature of NQMI was the prolonged ST-T duration. The difference in QT duration between normals and patients with acute MI would have been even more pronounced had six patients from the NQMI group and four patients from the acute QMI group, who received digoxin, been excluded from the analysis. The QRS durations in NQMI were practically identical to those measured in normals and in the other MI groups whether acute or not. Prolonged QT has been reported in acute MI and acute NQMI79-39; although the mechanism of prolonged action potential duration is not clear, ischemia is believed to be the main determinant. No adjustment of QT duration to heart rate was performed but no significant differences in heart frequencies were observed between the two acute MI groups. We have no explanation as to why ST-T was more prolonged in NQMI than in acute QMI (346 msec versus 323 msec; p=0.017): It could be related to persistent and widespread ischemia in patients with smaller areas of necrosis but larger areas of myocardium at risk of future events.40-42

NQMI: A Distinct Entity?

Despite some controversy regarding the validity of NQMI as a distinct clinical entity, numerous studies have reported the increasing incidence of acute MI without typical Q waves (or Q wave equivalents)43 and stressed the differences between acute QMI and NQMI in terms of physiopathology and prognosis.44-46 The most consistent differences were the lower incidence in total occlusion in NQMI, the earlier reperfusion or better collateral supply, the better short-term but deteriorating long-term prognosis, and the larger amounts of residual jeopardized myocardium at risk of recurrent ischemic events, particularly reinfarctions. In the present investigation, we were unable to demonstrate clear qualitative differences in map patterns between various types of MI; the differences between acute QMI and NQMI or between acute MI and old QMI were mainly quantitative and related to the size and the extent of the lesion, much less to the location. The recognition rates of acute QMI and NQMI achieved with classifiers derived from old QMI further established the link between closely related ECG entities. Note also that the group mean discriminant scores were 1.95 for normals, -1.54 for acute QMI, -1.12 for old QMI, and -0.75 for acute NQMI, indicating the intermediate position of old QMI.

Several non-ECG differences (listed in Table 4) between the two groups of acute MI (with and without Q wave) were observed in the present study. Due to the retrospective character of the report, no in-depth study could be performed of the various clinical variables. No difference in clinical variables was observed in the NQMI group between true NQMI or missed QMI subjects. The most striking features were the higher incidence of multiple-vessel disease in the NQMI group, the more frequent involvement of the right coronary and left circumflex arteries, the lower frequency of multiple segment wall motion abnormalities, the lower incidence of first-year mortality and intervention, and the higher ejection fraction and higher frequency of left ventricular hypertrophy. Left ventricular hypertrophy is frequently associated with ischemic heart disease, particularly in elderly subjects.46 The presence of more frequently increased echocardiographic left ventricular mass in NQMI, which cannot be accounted for by the number of hypertensive patients in both groups, may reflect compensatory hypertrophy consequent to the more diffuse and persistent areas of residual ischemia accompanying nonocclusive, multiple-vessel disease and producing a small, incomplete, or "aborted" myocardial necrosis but leaving large amounts of myocardium in a critical situation.

Relation to Previous Work

Several clinical investigations have demonstrated that surface maps could reveal the presence of myocardial necrosis undetected by the standard 12-lead ECG. In most cases, however, the study population consisted of patients in whom maps were recorded several weeks to months after the acute event. Few studies have correlated surface potential map patterns with acute QMI. In a recent study, Montague et al compared day 5 Q wave integral maps and radionuclide angiographic patterns among patients with first NQMI (27 patients) or QMI (29 patients). These patients were included in the present study population. The authors found a relation between the degree of wall motion abnormality (WMA) and the presence of visually abnormal Q wave integral maps: In the group with two or more segment WMA, all nine subjects had abnormal Q wave torso patterns. This was in contrast with only two of eight subjects without WMA and two of 10 subjects with one abnormal segment. Comparing these findings with those observed in 17 patients with anterior QMI and in 12 patients with inferior QMI, they demonstrated the presence of a wider spectrum of acute-phase ventriculographic and electrocardiographic spatial patterns in QMI than in QMI, including a large portion of patients with little or no LV systolic dysfunction and no spatially obvious depolarization abnormalities. Because of this greater heterogeneity among patients with NQMI, they suggested that, despite a close resemblance in map patterns, wall motion abnormality score, and ejection fraction between NQMI and inferior QMI, NQMI could be viewed as a distinct pathophysiological entity.

In the present report, we used quantitative analysis of sequential maps throughout the PQRST waveform...
rather than visual inspection of integral maps; although this offers the advantage of achieving considerable data reduction, integral values tend to dilute changes of small amplitude or of short duration that may contain significant diagnostic information. In particular, small changes often result from the time-varying locations of abnormal patterns, staying for too short a time at a particular lead site to significantly alter the integral value. The present investigation uses information collected at each instant on all leads. This information was analyzed in two different yet complementary ways. On one hand, discriminant maps were calculated and displayed: By subtracting the range of normal variability at each instant and at each site, sequential surface map abnormalities could be extracted and highlighted, facilitating the interpretation of BSPM in terms of underlying electro-physiological events. On the other hand, multivariate analysis was performed and discriminant functions computed from all the data: Identification of specific locations of ECG leads on the torso and selection of features for optimal classification of both acute QMI and NQMI were achieved.

**Study Limitations**

The first limitation deals with the sample sizes. Therefore, no breakdown of the QMI populations according to infarct location was attempted for statistical analysis. In addition, we had to limit the number of classification variables to achieve repeatable results. The anterior-inferior designations were based on QRS in the 12-lead ECG recorded in the acute phase. We could have used ECG-independent information (e.g., radionuclide imaging) but, due to the retrospective nature of the study, these images were not available in all MI subjects. Moreover, wall motion abnormalities could lead to ambiguous group assignment, particularly when multiple abnormalities coexist in various parts of the left ventricle and septum. For obvious reasons, stratification of NQMI could not occur on the basis of QRS in the 12-lead ECG; on the other hand, wall motion was normal in 26% of the subjects and infarct-related areas could not be reliably identified in the presence of multiple-vessel stenosis (40% of the subjects), given the rare occurrence of totally occluded arteries. Finally and foremost, the purpose of this investigation was to correctly identify acute MI regardless of the site of necrosis.

Another limitation is the proposed classification model itself. Indeed, the discriminant function used for group assignment arose from the pairwise comparison of normals versus recent and old QMI. The model recognizes well over 90% of acute, recent, and old MI at a high level of specificity with respect to a normal population. However, several other non-MI entities produce ST-T changes and have to be ruled out before the classifier is considered specific of MI. Because of the high prevalence of LVH in our MI groups, we were prompted to test the possible deterioration of the classification when assignment of MI patients to an LVH class without MI was made available: Very little misclassification occurred (none in the acute QMI group and 3% in the NQMI group). Other cardiac states affecting ST-T need to be considered similarly; this is of particular interest in a clinical setting of acute chest pain in which urgent therapeutic decisions are required and a diagnostic classifier must recognize acute MI as early as possible and rule out non-MI subjects.

**Clinical Implications**

The present study identified six electrode positions from which ECG signals could be recorded for optimal recognition of different types of myocardial infarction. The sensitivities at a specificity level of 96% were indeed 94% for recent or old Q wave MI, 93% for acute QMI, and 91% for acute NQMI. In a coronary care unit, such a lead system that is both practical and tailored to acute MI could be used for monitoring the evolution of the lesion and the effects of interventional procedures. In a more general setting, the two-step classification model, which considered the possibility for patients with acute MI to be misclassified as LVH, used a total of seven lead sites and yielded comparable diagnostic results; indeed, despite the large number of patients with combined LVH and MI in both acute MI groups, very few are lost to the pure LVH group. The present lead set is open-ended: Increasing the sample sizes allows for more variables to be selected, which, in turn, may require more electrodes; also, ruling out additional groups with confounding ST-T changes will most likely use supplementary recording sites. Another typical aspect of the various classification models is the combination of nonredundant measurements from all parts of the ECG waveform to produce stable and specific discriminant functions from different lead systems with minimum misclassification. We observed a similar pattern in a recent study on LVH in which P, QRS, and ST-T voltages and durations had to be combined to produce a stable and performing regression model for predicting LV mass with high accuracy. In addition to discriminant functions, group mean discriminant scores provide useful quantitative information regarding the distance between groups: They confirm the close proximity of NQMI to old QMI and justify a posteriori the choice of the latter for classification purposes. If discriminant scores, which reflect the distance of a group or an individual to normals, are indicative of the size and extent of an infarct, quantification of necrosis by BSPM becomes feasible. These are important steps in determining which patients are at highest risk and may benefit from early intervention. Impact of intervention could also be quantitatively assessed.

Interestingly, classification rates achieved in NQMI with the 12-lead ECG were surprisingly high despite the absence of Q waves; 70% of the patients were correctly classified with the single bigroup model and 65% with the two-step model. These
results stress two aspects of the study: the presence of diagnostic information outside the initial part of QRS and the power of multivariate statistical procedures that combine measurements which, considered separately, do not seem to contain significant diagnostic information.

References


29. Williams RA, Cohn PF, Yokonas PS, Young E, Herman MV, Gorlin R: Electrocardiographic, arteriographic and ventriculographic correlations in transmural myocardial infarction. Am J Cardiol 1973;31:595–599
36. Josephson ME, Kastor JA, Morganroth J: Electrocardiographic left atrial enlargement: Electrophysiologic, echocar-


44. Krone RJ, Friedman E, Thanavaro S, Miller JP, Kleiger RE, Oliver GC: Long-term prognosis after first Q-wave (transmural) or non-Q-wave (nontransmural) myocardial infarction: Analysis of 593 patients. Am J Cardiol 1983;52:234–239


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