ST Segment Shifts Are Poor Predictors of Subsequent Q Wave Evolution in Acute Myocardial Infarction

A Natural History Study of Early Non–Q Wave Infarction

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Acute ST segment elevation is regarded generally as the sine qua non of evolving Q wave myocardial infarction (MI) because such electrocardiographic (ECG) injury is believed to be a marker of transmural ischemia and a forerunner of transmural necrosis. Alternatively, ST segment depression with or without T wave inversion is viewed as the dominant ECG feature of non–Q wave MI. However, this hypothesis has not been assessed prospectively in an acute MI population. We analyzed 2,304 serial ECGs at study entry (admission), day 2, day 3, and predischARGE (mean, 10.2±2 days) from 576 patients with creatine kinase MB confirmed acute non–Q wave MI to determine what percentage of patients with early ST segment elevation culminated in subsequent Q wave development. Of this group, 187 patients (32%) exhibited 1 mm or greater ST segment elevation in two or more contiguous entry ECG leads. Of those patients whose non–Q wave MI could be localized on the basis of diagnostic admission ST segment shifts, the prevalence of early ST segment elevation was 43% (187 of 439). The sum total mean (±SD) peak ST segment elevation by lead group (anterior, inferior, lateral) was 4.0±2.4, 4.5±2.4, and 2.5±0.6 mm, respectively. Despite this, only 20% of patients with ST segment elevation (37 of 187) developed Q waves. Of 252 patients who exhibited early ST segment depression or T wave inversion or both, 39 (15%) evolved subsequent Q waves. Thus, while the prevalence of early ST segment elevation in acute evolving non–Q wave MI was higher than previously reported, 80% of patients with and 85% of patients without ST segment elevation and absent Q waves on the admission ECG did not develop subsequent Q waves during a 2-week period of observation (p=NS). In addition, when patients with ST segment elevation were compared with patients with ST segment depression or T wave inversions or both, there were no between-group differences in log peak creatine kinase (404 vs. 383 IU), reinfarction (6% vs. 8%), postinfarction angina (50% vs. 42%), or early recurrent ischemia (49% vs. 45%), defined as postinfarction angina with transient ECG changes. Thus, in patients who present with initial acute non–Q wave MI, ST segment shifts on admission are unreliable predictors of subsequent Q wave evolution and do not discriminate significant differences in postinfarction outcome. In particular, ST segment elevation during the early hours of evolving infarction is not an invaluable harbinger of subsequent Q wave development. (Circulation 1989;79:537–548)

The terms “transmural” and “nontransmural” myocardial infarction (MI) as detected by an electrocardiogram (ECG) imply a pathologic counterpart, but correlation with the histologic definition of transmural-nontransmural MI at necropsy is poor. Consequently, the more appropriate ECG terms of Q wave and non–Q wave MI have become widely accepted in clinical practice. Non–Q wave MI is not associated with a specific ECG pattern, and patients may present with ST segment depression or

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T wave abnormalities or both without the evolution of Q waves. Typically, these changes are transient (48–72 hours) and nonspecific, requiring confirmation of myocardial necrosis by independent evidence such as elevated plasma MB creatine kinase (CK) activity.4–8

In contrast, there is a well-recognized characteristic ECG pattern in patients evolving Q wave MI consisting of ST segment elevation followed by the development of pathologic Q waves in the leads overlying the area of myocardial necrosis.9–11 Prolonged chest pain and ST segment elevation on the admission ECG are now used as the principal eligibility criteria for prescribing thrombolytic therapy during acute MI12,13 the rationale being that acute ST segment elevation represents transmural ischemia due to a totally occluded infarct related coronary artery and is a forerunner of transmural (Q wave) MI.14

Whether prolonged ST segment elevation in the context of enzymatic evidence of myocardial necrosis is an invariable marker of subsequent Q wave evolution has not been assessed rigorously in a systematic large-scale, prospective study. Until recently, such information was difficult to obtain and was not of vital clinical importance. However, the widespread implementation of CK isoenzymes, the recognition of the distinct prognostic profiles of Q wave and non–Q wave MI,15–21 and the importance of early hospitalization for urgent thrombolytic therapy (the success of which is critically time dependent) make it both relevant and feasible to delineate the predictability of acute ST segment elevation during the early course of evolving MI.

This is further compounded by strong suggestive evidence that patients who evolve non–Q wave MI commonly undergo spontaneous early coronary reperfusion.20,24 There have been few natural history studies of the significance of ECG findings in non–Q wave MI,19,20 and this is important because most infarcts (within the first several hours) begin without Q waves. Hence, the distinction of Q wave compared with non–Q wave MI is more than a semantic one because different therapeutic strategies may be chosen in the initial hours after onset of MI.

Thus, it would seem vital to establish whether the early ECG findings of evolving non–Q wave MI (an infarct subtype characterized by a high prevalence of subsequent patent infarct vessels20,24,25) differ appreciably from what is customarily encountered in Q wave MI and whether ST segment shifts provide any meaningful guide to therapeutic decision making.

Accordingly, the objectives of the present study were to 1) identify the proportion of patients with evolving non–Q wave MI who exhibited initial ST segment elevation and to compare their clinical, enzymatic, and prognostic profiles with patients who exhibited ST segment depression or T wave changes or both on admission; and 2) determine (within the small subgroup of patients who subsequently evolved Q wave infarction) the accuracy of the ECG site, distribution, and extent of early ST segment elevation in predicting subsequent evolution of electrocardiographic Q waves.

Methods

The Diltiazem Reinfarction Study,26 initiated in 1982 and completed in 1985, contains a prospective data bank on 576 patients with acute non–Q wave MI. A total of 1,536 patients from nine enrolling sites (seven in the United States and two in Canada) were screened to assess eligibility for enrollment to the study on presentation to the emergency department with prolonged chest pain (≥30 minutes). All patients were admitted to the coronary care unit, where the initial screening took place. The diagnosis of MI was confirmed by serial elevated CK and plasma MBCK, and after informed written consent, patients were randomly assigned to receive treatment with either diltiazem (n = 287) or placebo (n = 289) in addition to standard care. Thus, 576 of 1,536 unselected screened patients (36%) were subsequently randomized, all of whom had initially absent Q waves. Each patient satisfied specific inclusion and exclusion criteria,26,27 and medication was initiated 53 ± 14 hours (range, 24–72 hours) after onset of infarction, beyond the interval when initial ECG assessments were made. The organization and conduct of the Diltiazem Reinfarction Study, as well as baseline characteristics of patients according to treatment group assignment, were reported previously.26,27

Clinical and Laboratory Evaluation

After randomization, the following evaluations were performed: 1) daily clinical evaluation by the center investigator; 2) 12-lead ECG on enrollment and daily for the first 5 days, followed by every other day thereafter until 14 days or hospital discharge; 3) ECG at the time of recurrent pain and for a minimum of 3 consecutive days if reinfarction was suspected; and 4) serial blood samples for analysis of total CK and MBCK activity. All protocol samples were forwarded to the CK Core Laboratory for quantitative analysis for plasma MBCK activity by the batch absorption glass bead method.28,29 Sampling times consisted of an initial sample at the time of randomization, followed by samples drawn every 12 hours thereafter throughout the 14-day study. Additional samples were obtained at least every 8 hours for the subsequent 72 hours in patients in whom reinfarction was suspected.

Treatment Regimen

Diltiazem hydrochloride or identical placebo was given every 6 hours orally for up to 14 days. Titration began with a 30-mg tablet and was increased by 30 mg every 6 hours up to a maximal dose of 360 mg/day diltiazem or placebo. Maximal tolerated dosing was achieved as soon as clinically possible.

All patients received standard medical therapy in accordance with local guidelines. Concurrent treatment with other calcium-channel blockers was pro-
patients had at

Interpretations

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leads within a given lead group) or the absence

of abnormal R waves (≥0.04 seconds in lead V_{1} and an

R:S ratio ≥1 in lead V_{1}) on admission to the hospital.

A comprehensive new code for the ECG diagnosis

of non–Q wave MI was developed in conjunction

with the Diltiazem Reinfarction Study.27,30 Acute ST
group displacement or T wave inversions or both were

not absolute prerequisites for entry into the study.

Significant ST segment shifts were defined as 1

mm or more of ST segment elevation or depression,

inhibited. Use of nitroglycerin preparations, β-

blockers, salicylates or dipyridamole, and low-dose

heparin was permitted and equally frequent in the
diltiazem- and placebo-treated patients.26 Initial therapy

for angina included sublingual nitroglycerin or

morphine. The approach to angina refractory to

medical therapy (nitrates and β-blockers) was individualized, and patients could be withdrawn, if

necessary, for surgery or angioplasty.

Electrocardiographic Recordings

and Interpretations

All patients included in the study were defined
electrocardiographically by the absence of pathologic
Q waves (≥0.03 seconds in duration in at least two

leads within a given lead group) or the absence

of abnormal R waves (≥0.04 seconds in lead V_{1} and an

R:S ratio ≥1 in lead V_{1}) on admission to the hospital.

A comprehensive new code for the ECG diagnosis

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group displacement or T wave inversions or both were

not absolute prerequisites for entry into the study.

Significant ST segment shifts were defined as 1

mm or more of ST segment elevation or depression,
Tracings were analyzed, and data were entered onto forms for subsequent computer analysis.

The admission ECGs of all study patients were reviewed by group consensus of five blinded investigators to ascertain the quantitative magnitude of ST segment elevation in respective lead groups. The magnitude of ST segment elevation in each lead group was measured by calipers 80 msec after the J point using the preceding TP segment as baseline. Both mean peak ST segment elevation (by individual lead) and summed ST segment elevation (ΣS-T) for a given lead group were quantified.

**Data Analysis**

To determine whether a specific ECG pattern on admission predicted which patients would evolve subsequent Q waves, the results of the detailed ECG analysis were compared with selected clinical baseline parameters (e.g., age, sex, Killip Class, previous MI), cardiac enzymatic data (peak plasma CK and MBCK, time to plasma CK and MBCK), and outcome variables, (death, reinfarction, postinfarction angina, ventricular tachycardia or fibrillation). Multivariate regression analysis was used to determine specific correlations among the above...
variables and the presence or absence of initial ST segment elevation on the admission ECG.

Statistics

Data were stored with the Washington University IBM mainframe computer system and were analyzed through use of the Statistical Analysis System. Continuous variables are reported as mean ± SD. Data analyses involving means included paired and unpaired t tests and both one-way and repeated measures analyses of variance. Analysis of proportions used χ² tests. While the reporting of means and SDs for peak CK values were for the variable itself, the logarithm of peak CK was used in all data analysis because of the skewed lognormal distribution of that variable. All comparisons of total inferior ST elevation with either anterior or lateral elevation were performed after multiplying the inferior total by a factor of 4/3. This was done to adjust for the difference in the number of leads in the anterior and lateral lead groups (four each) as compared with the inferior location (three leads).

The statistical methods used for ΔST elevation per lead group or millimeters of ST elevation per lead were performed after the values for these variables were ranked from smallest to largest and transformed to normality using the method of Blom. Analyses of variance and t tests were then applied to the transformed ranks. Statistical methods that applied standard parametric analyses to ranked data were used according to the method of Conover and Iman. They were used in this circumstance because the

Figure 2. Serial electrocardiograms from a study patient at entry (top left panel), study day 2 (bottom left panel), and study day 3 (upper panel). In top panel, there is acute ST segment elevation in leads V₂-V₄, with decreased R wave amplitude in lead V₃. Note the ventricular premature beats in a bigeminal pattern. In bottom panel, ST segment elevation has decreased in leads V₂-V₄, but R wave amplitude has not changed. In upper panel, there is residual ST segment elevation in leads V₂-V₅, but Q waves have not evolved.

Figure 3. Flow diagram detailing the electrocardiographic locations of the 187 patients with early ST segment elevation on the prestudy tracing. One hundred sixty-two patients had ST segment elevation isolated to a single electrocardiographic location (anterior, inferior, lateral), while 25 patients had ST segment elevation in more than one electrocardiographic location. For the purpose of assessing the incidence of Q wave evolution by electrocardiographic infarct location (Figures 4 and 5), these 25 patients were censored from analysis.
transformed ranks produced data that approximated the normality and equal variance requirements of $t$ tests and analyses of variance far better than either the original variables or other available transformations such as the logarithm and square root.

**Results**

**Clinical Characteristics**

Figure 1 shows ECG findings among the 576 patients who were enrolled in the Diltiazem Reinfarction Study. There were 32 patients (5.5%) who, in retrospect, had an acute Q wave MI on entry into the trial; these patients were censored from further analysis. Of the 544 remaining patients with acute non-Q wave MI, 187 (34%) exhibited admission ST segment elevation associated with the MBCK-confirmed event. Because 105 of the 544 patients (19%) had nonlocalizable non-Q wave MI, as defined previously, the true prevalence of ST segment elevation among patients whose infarcts could be localized by diagnostic ST segment shifts on the admission ECG was 43% (187 of 439).

Table 1 compares clinical and laboratory characteristics for the two subgroups of study patients according to the presence or absence of early ST segment elevation. Several between-group differ-

### TABLE 2. Magnitude of ST Elevation per Elevated Lead as a Function of the Number of Elevated Leads in a Given Lead Group

<table>
<thead>
<tr>
<th>Lead Group</th>
<th>Two leads with ST elevation</th>
<th>Three leads with ST elevation</th>
<th>Four leads with ST elevation</th>
<th>Significant differences within each lead</th>
</tr>
</thead>
<tbody>
<tr>
<td>Anterior</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>$V_1$</td>
<td>1.22±0.52 ($n=32$)</td>
<td>1.19±0.63 ($n=55$)</td>
<td>1.39±0.96 ($n=31$)*</td>
<td>2 or 3 vs 4 ($p&lt;0.0001$)</td>
</tr>
<tr>
<td>$V_2$</td>
<td>1.23±0.41 ($n=35$)</td>
<td>1.47±0.63 ($n=61$)</td>
<td>2.65±2.40 ($n=31$)*</td>
<td>2 vs 4 ($p&lt;0.0001$)</td>
</tr>
<tr>
<td>$V_3$</td>
<td>1.19±0.37 ($n=8$)</td>
<td>1.95±0.58 ($n=62$)</td>
<td>2.53±2.32 ($n=31$)*</td>
<td>2 vs 4 ($p=0.0336$)</td>
</tr>
<tr>
<td>$V_4$</td>
<td>1.50±0.61 ($n=5$)</td>
<td>1.75±0.96 ($n=8$)</td>
<td>1.81±2.72 ($n=31$)*</td>
<td>3 vs 4 ($p&lt;0.0001$)</td>
</tr>
<tr>
<td>Lateral</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>I</td>
<td>1.00±0.0 ($n=10$)</td>
<td>0.88±0.25 ($n=4$)</td>
<td>1.38±0.48 ($n=4$)*</td>
<td>3 vs 4 ($p=0.0237$)</td>
</tr>
<tr>
<td>aV_{II}</td>
<td>1.25±0.38 ($n=8$)</td>
<td>1.13±0.25 ($n=4$)</td>
<td>1.50±0.91 ($n=4$)*</td>
<td>3 vs 4 ($p=0.0555$)</td>
</tr>
<tr>
<td>V_{II}</td>
<td>1.04±0.14 ($n=12$)</td>
<td>1.00±0.0 ($n=4$)</td>
<td>4.38±3.15 ($n=4$)*</td>
<td>3 vs 4 ($p&lt;0.001$)</td>
</tr>
<tr>
<td>V_{III}</td>
<td>1.05±0.16 ($n=10$)</td>
<td>0.83±0.29 ($n=31$)</td>
<td>1.88±1.44 ($n=4$)*</td>
<td>3 vs 4 ($p=0.0555$)</td>
</tr>
<tr>
<td>II</td>
<td>1.00±0.0 ($n=2$)</td>
<td>1.01±0.50 ($n=34$)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Inferior</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>III</td>
<td>1.00±0.24 ($n=18$)</td>
<td>1.66±0.95 ($n=34$)*</td>
<td></td>
<td>2 vs 3 ($p=0.0012$)</td>
</tr>
<tr>
<td>aV_{III}</td>
<td>1.00±0.0 ($n=16$)</td>
<td>1.41±0.69 ($n=34$)*</td>
<td></td>
<td>2 vs 3 ($p=0.0014$)</td>
</tr>
</tbody>
</table>

*Significant differences by analysis of variance for the number of elevated leads in any given lead.

†Bonferroni adjusted in anterior and lateral location where three groups are compared, in contrast to the two inferior groups.

Magnitude of ST segment elevation per elevated lead, within the three electrocardiogram lead groups, as a function of the number of elevated leads for anterior, lateral, and inferior non-Q wave MI.

![Figure 4](http://circ.ahajournals.org/)

**Figure 4.** Bar graph detailing mean peak creatine kinase (CK) values for each electrocardiographic non-Q wave infarction location (anterior, inferior, lateral) as a function of the number of leads within a given lead group showing ST segment elevation. Values are reported as the logarithm of peak CK because of the skewed lognormal distribution of that variable (see text).
ences were observed. Compared with patients without initial ST segment elevation, patients with ST segment elevation were younger (59.6±10.4 years compared with 62.0±10.0 years, \( p = 0.0098 \)), more likely to be cigarette smokers (64\% compared with 50\%, \( p = 0.0022 \)), exhibit subsequent T-wave inversion (64\% compared with 54\%, \( p = 0.0374 \)), develop ventricular tachycardia or fibrillation during hospitalization (5\% compared with 2\%, \( p = 0.054 \)), and progress to Q wave MI during the subsequent course of their hospitalization (20\% compared with 11\%, \( p = 0.0046 \)). However, 80\% of all non-Q wave MI patients who exhibited early ST segment elevation (150 of 187) did not evolve Q waves on review of serial ECGs.

Figure 2 is a representative sequence of tracings obtained from one patient on admission, on study day 2, and on study day 3.

**Location and Magnitude of ST Segment Elevation**

Of the 187 patients with initial ST segment elevation, 111 (59\%) had elevations that were anterior only, 12 (6\%) were lateral only, 39 (21\%) were inferior only, and 25 (13\%) had multiple location ST segment elevation (Figure 3). To determine whether the magnitude of acute ST segment elevation cor-

**TABLE 3. ST Segment Elevation Compared with Depression**

<table>
<thead>
<tr>
<th></th>
<th>ST segment elevation</th>
<th></th>
<th>ST segment depression</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Anterior MI</td>
<td>Inferior MI</td>
<td>Anterior MI</td>
</tr>
<tr>
<td><strong>Sensitivity</strong></td>
<td>21/37 (57%)</td>
<td>16/37 (43%)</td>
<td>16/37 (43%)</td>
</tr>
<tr>
<td><strong>Specificity</strong></td>
<td>38/150 (25%)</td>
<td>114/150 (76%)</td>
<td>38/150 (25%)</td>
</tr>
<tr>
<td><strong>Predictive value</strong></td>
<td>21/21+112 (16%)</td>
<td>16/16+36 (31%)</td>
<td>16/16+38 (30%)</td>
</tr>
</tbody>
</table>

MI, myocardial infarction.
Sensitivity, specificity, and predictive value of ST segment elevation or depression for the subsequent development of Q waves.
related with a higher rate of Q wave development and, if so, whether this was a function of the ECG site or enzymatic "size" of infarction, as assessed by comparative log CK values, a detailed analysis of the summed total ST segment elevation by respective lead group was performed, over a range of ST segment elevation, in 1-mm increments. To compare the prevalence of Σ ST in one lead group with ΣST in another, those patients with ST segment elevation in more than one electrocardiographic location (n=25) were censored for the purpose of this analysis.

Among the remaining 162 patients with single-lead group early ST segment elevation, Σ ST segment elevation was 4.0±2.4 mm for the anterior infarction subgroup, 3.5±1.8 mm for the inferior infarction subgroup, and 2.5±0.6 mm for the lateral infarction subgroup. Analysis after multiplication by a weighting factor of 4/3 for the smaller number of leads in the inferior group resulted in a total adjusted inferior Σ ST of 4.5±2.4 mm (p=NS).

Distribution of ST Segment Elevation and Peak Creatine Kinase Levels

The quantitative ST segment analysis was repeated on a lead-by-lead basis for each of the 11 leads (aV₆ excluded). This analysis examined the relation between the amount of ST segment elevation per elevated lead and the number of elevated leads in the associated lead group. The pattern that emerged was that as the number of elevated leads in a given lead group increased, the magnitude of ST segment elevation per elevated lead likewise increased. Table 2 demonstrates that for all 11 leads, the maximum mean ST segment elevation per elevated lead occurred when all leads in the associated lead group were elevated.

Figure 4 summarizes peak CK values for each ECG MI location (anterior, inferior, lateral) as a function of the number of leads within a given lead group showing ST segment elevation. There was a trend toward larger enzymatic infarcts as the number of leads exhibiting ST segment elevation increased from two to three and from three to four, but there were no significant differences in log peak CK values within respective lead groups.

Site of Initial ST Segment Elevation and Subsequent Q Wave Evolution

Figures 5 and 6 examine the relation of initial ST segment elevation for anterior and inferior non-Q wave MI and the likelihood of evolving a subsequent Q wave infarction, based on a review of serial ECGs. Of note, of the 111 patients with isolated anterior non-Q wave MI with at least 1 mm ST segment elevation per lead in two or more leads, 45 (41%) exhibited 2.0 mm or greater ST segment elevation per lead. Of these, only seven (16%) progressed to Q wave MI, including just two of 13 patients with 3.0 mm or greater ST segment elevation (Figure 5). Conversely, six of 11 patients (55%) with inferior ST segment elevation of 2.0 mm or more per lead progressed to subsequent Q wave MI (Figure 5). The p value associated with progression to Q wave infarction (anterior compared with inferior location) was 0.013 (two-tailed Fisher’s exact test).

ST Segment Depression or T Wave Inversion

Of the 439 patients with localizable ST segment shifts, 252 (57%) exhibited ST segment depression or T wave inversion or both in two or more leads on the entry ECG (Table 1). Seventy-seven patients (31%) had isolated ST segment depression, whereas 175 patients (69%) had a combination of ST segment
depression and T wave inversion. Of this entire group, 39 patients (15%) progressed to Q wave MI, compared with 37/187 patients (20%) with initial ST segment elevation (p = NS).

There were no differences in the site of qualifying infarct by ECG, peak CK, or the time to peak CK, nor was the incidence of post-MI angina and reinfarction in patients with ST segment depression or T wave inversion or both different compared with those patients with early ST segment elevation.

**Sensitivity, Specificity, and Predictive Value of ST Segment Shifts in Evolving Non-Q Wave MI**

As noted above, analysis for site of MI among the 187 patients with ST segment elevation showed only 16% of the patients (21/133) with anterior ST segment elevation progressed to Q wave MI (Figure 6). There was a significant negative association for developing Q wave MI if the initial ECG showed anterior ST segment elevation (p = 0.031). Conversely, of the 52 patients with inferior ST segment elevation, 16 (31%) progressed to Q wave MI leading to a significant positive association of developing Q wave MI (p = 0.019). There were too few lateral non-Q wave infarctions with isolated ST segment elevation to permit a statistical analysis in this group.

Table 3 summarizes the sensitivity, specificity, and predictive value of ST segment shifts for the subsequent development of Q waves in the Diltiazem Reinfarction Study population. ST segment elevation for both anterior and inferior MI was poorly predictive of subsequent Q wave evolution. Somewhat surprisingly, early ST segment depression was more predictive of subsequent Q wave evolution (30%) than early ST segment elevation (16%) for anterior non-Q wave infarction, but the reverse was true for inferior infarction.

**Discussion**

In the Diltiazem Reinfarction Study of early non-Q wave MI, 576 patients were enrolled and followed prospectively with serial analysis of ECGs and plasma CK and MBCK determinations. Paradoxically, one third of the patients (187 of 576), despite initial non-Q wave MI only, exhibited ST segment elevation on admission to the hospital. Subgroup analysis showed that of patients presenting with ST segment elevation, 80% did not develop new Q waves; in the group presenting with ST segment depression or T wave inversion or both, 85% of the patients did not develop new Q waves.

Moreover, the presence or absence of admission ST segment elevation was not associated with significant differences in the incidence of subsequent reinfarction or post-MI angina. In patients with early anterior ST segment elevation, there was no significant correlation between the magnitude or extent of ST segment elevation and the subsequent development of Q waves. However, early inferior ST segment elevation was more frequently associated with subsequent Q wave evolution, although the explanation for this finding is uncertain. One possibility is that collateral blood flow to the anterior wall was less jeopardized than to the inferior wall in this study population, which might explain the higher rate of Q wave development in the latter group.

Feldman and coworkers34 have shown that, compared with patients with reversible ST segment depression, those patients who exhibited reversible ST segment elevation during left anterior descending (LAD) coronary artery occlusion had less severe degrees of LAD stenosis and uniformly absent collaterals to the distal LAD bed. Persistent ST segment depression complicating early non-Q wave MI, however, may be a marker of more severe ischemia and subsequent Q wave evolution, particularly in those patients with initial anterior non-Q wave MI.

**Electrocardiographic Pattern of Q Wave and Non-Q Wave Infarction**

Non-Q wave MI generally is defined electrocardiographically by the absence of abnormal Q waves (≥0.03 seconds in two or more leads) or “Q wave equivalent” (i.e., R wave ≥0.04 seconds in lead V₁ and R:S ≥1 in lead V₂) and is usually characterized by ST segment depression, with or without an abnormal T wave.35 It is curious that evolving acute non-Q wave infarction is not typically regarded as one of the clinical settings in which acute ST segment elevation may occur.

The fact that 43% (187 of 439) of all non-Q wave MI patients with initially “localizable” infarcts by ECG in our study (and 32% of all non-Q wave MI patients) exhibited ST segment elevation is a noteworthy observation. Of importance is that of the 162 patients who exhibited ST segment elevation confined to a single ECG lead group, 57 (35%) exhibited more than 2 mm ST segment elevation per lead (Figure 3), yet only 13 of 57 (23%) developed new Q waves (Figure 5). Thus, it is apparent that the specificity of early ST segment elevation in the diagnosis of evolving Q wave MI is much less than previously suspected and that ECG ST segment elevation, while clearly a marker of transmural ischemia, may not invariably culminate in transmural infarction and subsequent Q wave evolution.

Finally, we observed that an equivalent number of patients with initial ST segment depression or T wave inversions or both went on to evolve Q waves. These observations would indicate that ST segment shifts (either elevation or depression) are of limited predictive value in determining the likelihood of subsequent Q wave evolution (due to a presumably occluded infarct-related coronary artery) in patients who initially presented with early repolarization changes only.

**Comparison With Other Studies**

Four recent studies on acute non-Q wave MI are relevant to the findings we report. Huey et al19 examined the electrocardiographic, enzymatic, thallium scintigraphic, and angiographic findings in 150 consecutive patients with acute MI who, on admis-
sion, exhibited 1 mm or greater ST segment elevation in two or more contiguous ECG leads. Among the group without Q waves on admission, analysis of serial ECGs revealed that 35 of 95 patients (37%) evolved non-Q wave MI, a prevalence very similar to our own findings. Spontaneous coronary reperfusion as reflected by early peaking of serum enzyme levels and a high prevalence of patent infarct vessels characterized this subgroup of patients, compared with those with ST segment elevation culminating in Q wave MI.19 A recent report from the Myocardial Infarction Limitation of Infarct Size (MILIS) Study also showed a high prevalence of acute ST segment elevation in acute non-Q wave MI.36 Of 304 patients with isoenzyme-confirmed acute non-Q wave MI, 207 patients (68%) exhibited ST segment elevation on admission.

A recent study by DeWood et al,24 which examined the coronary arteriographic findings soon after non-Q wave MI, showed that in contrast to Q wave MI, total coronary occlusion of the infarct-related vessel was infrequent in the early hours of non-Q wave MI. These data, along with other published studies detailing the subsequent high patency rate of the infarct-related coronary artery,19,20,24,25 support the concept that acute non-Q wave MI represents an aborted Q wave MI. Thus, acute ST segment elevation that abates without subsequent Q wave evolution may represent spontaneous reperfusion,19–25 and in our present experience, 80% of patients without initial Q waves exhibited this electrocardiographic pattern.

Although DeWood and coworkers did not quantify the proportion of individuals with non–Q wave MI who exhibited ST segment elevation before cardiac catheterization, their observation that spontaneous reperfusion occurred commonly during the 1st week (especially within the first 72 hours) after non–Q wave MI supports the hypothesis that acute ST segment elevation without progression to Q waves is indicative of coronary obstruction with subsequent early recanalization and attendant myocardial salvage.

Finally, a recent Diltiazem Reinfarction Study data bank analysis showed that a minority of non–Q wave MI patients with acute precordial ST segment depression, interpreted at study entry as classic anterior subendocardial necrosis, developed pathologic R waves in leads V1–V6 on subsequent review of serial ECGs.37 A detailed review of these tracings showed a characteristic pattern of horizontal or downsloping ST segment depression with upright T waves in the precordial leads, indicative of posterior wall injury. Because of the reciprocal nature of these acute repolarization changes, early transmural ischemia of the posterior wall may be manifest initially as precordial ST segment depression, which later evolves into a Q wave equivalent posterior transmural MI.36

Clinical Implications

Our observations may have important clinical implications, particularly in deciding which infarct patients should be selected for acute thrombolytic therapy. Most current guidelines for patient selection involving streptokinase or recombinant tissue plasminogen activator require the presence of only 1 mm of ST segment elevation in two or more ECG leads.12–14 ST segment depression is generally regarded as an exclusion criterion. Because it is not possible to differentiate with precision those patients with acute ST segment elevation indicative of transmural ischemia who will evolve Q waves from those with transmural ischemia who will not, some patients (particularly non–Q wave MI patients) may receive thrombolytic therapy but would likely have undergone spontaneous coronary reperfusion anyway. Whether there would be additional myocardial salvage in such non–Q wave MI patients who receive thrombolytic therapy has not been addressed scientifically and must await future study.

Conversely, many patients who exhibit ST segment depression (particularly patients with occluded or subtotally occluded circumflex or posterior descending coronary arteries37,38) and would be appropriate candidates otherwise for thrombolytic therapy are presently excluded from receiving such potentially life-saving thrombolytic therapy. A priori, there is no scientific basis for excluding patients with ST segment depression, especially when such occurrences reflect transmural ischemia of the posterior or lateral wall, and not trivial subendocardial ischemia.

Related to this issue of interpreting the proper significance of ST segment depression during prolonged myocardial ischemia is the recent observation that ST segment depression complicating acute non–Q wave MI is a major predictor of adverse long-term prognosis.39,40 We have observed that ST depression, whether measured on the admission ECG or on the discharge ECG, was associated with a two-fold higher incidence of reinfarction and death at 1-year, compared with patients with either ST segment elevation or T wave inversion or both.39,40 Moreover, we have shown that persistent ST segment depression that is seen on both the admission and discharge ECG is an independent risk factor for increased late mortality.41 The mortality rate among 515 non–Q wave MI patients without any ST segment depression was 5.5%, compared with 10.1% in patients with ST segment depression on either the admission or discharge ECG, and 22.2% in patients with ST segment depression on both ECGs.

Investigators for the MILIS Study have also reported that non–Q wave MI patients with ST segment depression are a high-risk group of patients with a poor long-term outcome,36 similar to our own findings. Why ST segment depression appears to be a marker of adverse long-term prognosis is not presently clear, but studies suggest that impaired, or threatened, collaterals may be a prominent feature in those non–Q wave MI patients who are prone to repetitive ischemic events.34,42,43
Summary

We believe our data show that ST segment elevation is a prevalent finding in early acute non-Q wave MI and does not necessarily prease the development of Q waves. Moreover, these data indicate that ST segment shifts (elevation or depression) may indicate transmural ischemia that may not invariably culminate in transmural infarction and that ST segment depression should not be considered inherently more “benign” than ST segment elevation. Finally, decisions to proceed to early thrombolytic therapy should not be predicated solely on the arbitrary adoption of prespecified thresholds of ST segment deviation because either ST segment elevation or depression may be associated with comparable degrees of coronary luminal narrowing. The role of thrombolytic therapy in patients with evolving MI characterized chiefly by ST segment depression remains to be elucidated.

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