Electrocardiographic Diagnosis of Postinfarction Regional Pericarditis

Ancillary Observations Regarding the Effect of Reperfusion on the Rapidity and Amplitude of T Wave Inversion After Acute Myocardial Infarction

Philip B. Oliva, MD; Stephen C. Hammill, MD; and William D. Edwards, MD

Background. The ECG recognition of diffuse pericarditis following acute myocardial infarction has been based on changes of the ST segment and, to a lesser extent, alterations of the PQ segment. No ECG criteria exist for the diagnosis of postinfarction regional pericarditis. Recently, it was observed that the T wave evolution follows an atypical pattern before fatal free wall rupture and that this pattern is due to the associated pericarditis. Therefore, this study was conducted on 200 patients with acute myocardial infarction to further elucidate the sensitivity and specificity of the atypical T wave changes in patients with regional postinfarction pericarditis without rupture and to assess the effect of lytic treatment on the rapidity and amplitude of postinfarction T wave evolution.

Methods and Results. An analysis of the clinical courses and serial ECGs of 200 consecutive patients with acute myocardial infarction was performed. Among 43 patients with postinfarction pericarditis, the pattern of T wave evolution consistently differed from the customary postinfection pattern of T wave evolution. This unusual evolutionary course was expressed as either persistently positive T waves or more hours after infarction (67%) or premature, gradual reversal of inverted T waves to positive deflections (33%). The sensitivity and specificity of these T wave alterations were 100% and 77%, respectively. The only other processes identified that caused this type of postinfarction T wave evolution were cardiopulmonary resuscitation, reinfarction, and very small infarcts. Both reperfusion, as judged by the creatine kinase-MB curve, and patency, as assessed by the angiogram, were correlated with the rapidity and depth of T wave inversion. Ninety percent of patients with reperfusion attained a maximum T wave negativity of 3 mm or more within 48 hours after the onset of chest pain in the lead that initially displayed the greatest ST segment elevation. Seventy-six percent of patients without reperfusion attained a maximum negativity of 2 mm or less within 72 hours. Thus, like the ST segment, accelerated evolution and deepening of the T wave may be noninvasive markers of reperfusion.

Conclusions. First, premature concordancy of the ST segment and T wave after acute myocardial infarction is a sensitive, reasonably specific, and easily recognizable ECG manifestation of postinfarction pericarditis. Second, reperfusion is associated with accelerated evolution and deepening of the T waves following acute myocardial infarction. (Circulation. 1993;88:896-904.)

Key Words • myocardial infarction • pericarditis • reperfusion • electrocardiography • waves

The ECG recognition of regional pericarditis following acute transmural myocardial infarction has been said to be "difficult, if not impossible."1-2 Textbooks on ECG3-4 and diseases of the pericardium5-8 describe the development of ST segment elevation in leads uninvolved in the infarction process as indicative of a diffuse pericarditis, but no repolarization changes ascribable to regional pericarditis following infarction have been recognized. Recently, we observed the development of an atypical type of T wave evolution during the first few days after acute myocardial infarction in patients destined to sustain free wall rupture. The T wave alterations were found to be due to associated regional pericarditis in the majority of instances.9 The purposes of this report are to assess the frequency of atypical T wave evolution in patients with early postinfarction regional pericarditis without rupture, to illustrate some ECG nuances of this condition, and to provide certain clinico pathological correlations. In addition, the effect of reperfusion on the rapidity and amplitude of postinfarction T wave evolution is analyzed.

Methods

The medical records and ECGs were reviewed for 100 consecutive patients with acute myocardial infarction...
from January 1985 through March 1986 and for 100 other consecutive patients from December 1990 through January 1992 who fulfilled the following criteria for acute myocardial infarction: characteristic chest pain of at least 30 minutes' duration; initial ST segment elevation of at least 2 mm in two or more contiguous precordial leads or at least 1 mm in 2 or more contiguous bipolar or unipolar limb leads, using the hexaxial reference system of Fumagalli,10 or initial horizontal ST segment depression with a totally or terminally upright T wave in V1-V3 indicative of a strictly posterior infarction, followed by the development of prominent primary R waves in leads V1-V3 (R/S ratio ≥1), as described by Boden and colleagues11; age less than 70 years with no contraindication to lytic therapy in the “1990 to 1992 series”; and a subsequent rise of the myocardial component of creatine kinase (CK-MB) to a peak within 6 to 30 hours with a return to normal within 3 to 4 days. The normal range is 0 to 11 IU/L, and a value of at least 15 IU/L was necessary for inclusion in the study. Patients with previous coronary artery bypass surgery, right bundle branch block, left bundle branch block, left ventricular hypertrophy with strain, or hypokalemia or hyperkalemia and those using digitalis or type IA antiarrhythmic drugs were excluded.

**ECG and Enzymatic Analyses**

After the initial pretreatment ECG, serial blood measurements for CK-MB were made every 6 hours for 72 hours. Because patency and reperfusion are not synonymous, reperfusion was defined as a peak CK-MB within 13 hours of onset of symptoms and no reperfusion as a peak at 13 hours or later.12 ECGs were obtained at 12, 24, 48, and 72 hours after infarction and daily thereafter. The ECGs were reviewed blindly by two observers. The amplitude of the T wave in all leads was measured with calipers and a calibrated magnifying glass. An upright T wave was defined as positive, and an inverted T wave was defined as negative.

The former 100 patients served as the control group for a study of patients with free wall cardiac rupture, the group of patients in whom the atypical T wave evolutionary pattern we described was first observed.9 Fifteen percent of the “1985 to 1986 series” received thrombolytic therapy. The more recent 100 patients were examined to assess the effect of reperfusion on the rapidity and amplitude of T wave inversions after acute myocardial infarction. Therefore, all of the patients in the more recent group received thrombolytic therapy. The thrombolytic protocols used were tissue-type plasminogen activator (Activase) (90 mg over 3 hours following a 10-mg loading dose [71%]), anisoylated plasminogen streptokinase activator complex (Eminase) (30 mg over 5 minutes [22%]), or streptokinase (1.5 million units over 1 hour [7%]). Patients with a previous myocardial infarction were not excluded, whereas those patients who failed to evolve CK-MB evidence of infarction were excluded. Echocardiograms were not routinely done. Coronary angiography was performed 2 to 10 days (mean, 4.5 days), after the administration of lytic therapy.

**Definitions**

A diagnosis of pericarditis was made if typical pleuritic-positional chest, left shoulder, or scapular pain and/or a pericardial friction rub occurred during the first week after acute myocardial infarction and was not associated with reerelevation of the CK-MB. Classic symptoms without a rub were accepted as indicative of pericarditis because to rely on a fleeting or overlooked rub would grossly underestimate the frequency of pericarditis.13 Reinfarction was diagnosed when postinfarction chest pain, not pleuritic or positional, was associated with recurrent ST segment elevation and reerelevation of the CK-MB occurred.

**Statistical Analysis**

The sensitivity, specificity, and predictive values for both patency and reperfusion, predicated on the rapidity and amplitude of the T wave inversion, were calculated. The data are given in Table 1. Sensitivity was defined as the probability of a given test result in a group of patients with a disease (ie, number of patients with disease showing a given test result divided by the total number of tested patients with disease), specificity was defined as the probability of not having the given test result in a group of patients without disease (ie, number of disease-free patients not showing the test result divided by the total number of disease-free pa-

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**TABLE 1. Relation of T<sub>max</sub> to the Sensitivity, Specificity, Predictive Value, and Probability of Reperfusion and Coronary Arterial Patency**

<table>
<thead>
<tr>
<th>No. of patients</th>
<th>Sensitivity (%)</th>
<th>Specificity (%)</th>
<th>Predictive value (%)</th>
<th>Probability (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Reperfusion</td>
<td>Patency</td>
<td>Reperfusion</td>
<td>Patency</td>
<td>Reperfusion</td>
</tr>
<tr>
<td>T&lt;sub&gt;max&lt;/sub&gt;</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>≥4 mm†</td>
<td>36</td>
<td>58</td>
<td>100</td>
<td>100</td>
</tr>
<tr>
<td>3 mm</td>
<td>24</td>
<td>87</td>
<td>84</td>
<td>90</td>
</tr>
<tr>
<td>1 to 2 mm</td>
<td>11</td>
<td>86</td>
<td>55</td>
<td>76</td>
</tr>
<tr>
<td>T isoelectric to positive‡</td>
<td>15</td>
<td>...</td>
<td>...</td>
<td>...</td>
</tr>
<tr>
<td>Reversal after inversion§</td>
<td>14</td>
<td>...</td>
<td>...</td>
<td>...</td>
</tr>
</tbody>
</table>

Reperfusion indicates early reperfusion.

*<sup>T</sup> <sub>max</sub> is the maximum depth in millimeters of T wave inversion in the single lead with the greatest inversion.

†Includes the 4 patients with a very small infarct in whom the T wave rapidly inverted but then was succeeded by premature reversal.

‡Includes the 10 patients with pericarditis, 3 with cardiopulmonary resuscitation, and 2 with no apparent cause.

§Includes 5 patients with pericarditis and 9 with reinfarction.
TABLE 2. Electrocardiographic Site of Acute Myocardial Infarction in 43 Patients With Postinfarction Pericarditis

<table>
<thead>
<tr>
<th>Site of infarction</th>
<th>Patients</th>
</tr>
</thead>
<tbody>
<tr>
<td>Anterior</td>
<td>12</td>
</tr>
<tr>
<td>Inferior</td>
<td>9</td>
</tr>
<tr>
<td>Anterolateralosuperior</td>
<td>6</td>
</tr>
<tr>
<td>Inferolateral</td>
<td>6</td>
</tr>
<tr>
<td>Anterolateral</td>
<td>5</td>
</tr>
<tr>
<td>Lateral</td>
<td>2</td>
</tr>
<tr>
<td>Inferolateroposterior</td>
<td>2</td>
</tr>
<tr>
<td>High lateral (superior)</td>
<td>1</td>
</tr>
<tr>
<td>Posterior</td>
<td>0</td>
</tr>
</tbody>
</table>

Tions tested), and predictive value was defined as the proportion of true results among all those patients with a positive test result.

Results

A total of 43 patients were identified with symptoms and/or signs of pericarditis. Twenty-eight of these patients came from the 1985 to 1986 series, 2 of whom received thrombolytic therapy, whereas 15 patients came from the 1990 to 1992 series, all of whom received thrombolytic therapy. Thirty-six patients had pleuropерicardial pain, 20 with a rub, and 7 had a rub without pain. The site of infarction was anterior, anterolateral, or anterolateralosuperior in 53% and inferior, inferolateral, or inferolateroposterior in 40% (Table 2). Signs and symptoms of pericarditis appeared between days 1 and 7 (average, day 3) and lasted from a few hours to 5 days.

Each patient with pericarditis had a pattern of T wave evolution that differed from the pattern of T wave evolution in those without pericarditis (Fig 1). In 29 patients (67%) with pericarditis, the T wave remained persistently positive 48 hours after the onset of symptoms (Fig 2), whereas in 14 patients (33%), initial inversion within 24 to 48 hours was succeeded by gradual reversal of T wave polarity to form an upright or isoelectric deflection (Fig 3). These T wave changes occurred only in the leads that initially displayed ST segment elevation. In one instance, reversal of the T wave polarity to a less-negative deflection was observed. In all except five cases, the T wave, whether persistently positive at 48 hours or becoming positive after being initially inverted, gradually inverted, or reinverted, after resolution of the pericarditis. Three of the five patients who attained and maintained a positive T wave after inversion died, and at autopsy a localized fibrinous pericarditis was present.

Among the 29 patients with persistently positive T waves after 48 hours, the initial ST segment elevation had regressed to 2 mm at that time in 24, whereas persistent ST segment elevation of 3 to 5 mm existed in 4 patients. In one instance, ST segment reelevation accompanied persistently positive T wave at 48 hours. In contrast, when T wave reversal followed initial inversion (14 patients), ST segment reelevation (2 to 6 mm) accompanied the T wave reversal in 9 cases, whereas in 3 patients, the ST segment remained persistently elevated to 3 mm or more, and in only two instances was T wave reversal associated with less than 2 mm of ST segment elevation. Thus, although an unusual pattern of T wave evolution occurred in all patients with pericarditis, the ST segment reced accordingly in 26 instances (60%). In the other 17 patients, ST segment reelevation, in the absence of a secondary increase in CK-MB (10 patients, or 24%), was observed, and in 7 instances (16%), the ST segment remained persistently positive, 2 mm or more at 48 hours.

Among patients with multisegment infarcts, the T wave often inverted normally in one region but not in others. The two patients with multisegment infarcts who died had pericarditis overlying the region with "atypical" T wave evolution and a normal pericardium overlying the region with expected T wave evolution (Fig 4).

Within the total study population of 200 patients, all 43 patients with pericarditis had either persistently positive T waves for 48 hours or more of inverted T waves. Among the 157 patients without pericarditis, persistently positive T waves were seen in 10 instances (6%) and reversal of inverted T waves in 25 cases (16%). Five of the 10 patients without pericarditis with persistently positive T waves had had cardiopulmonary resuscitation performed. Twenty-one of the 25 patients with reversal of inverted T waves had reinfection, and the other 4 had small, initial infarcts. In these four instances, the T wave rapidly inverted within 12 to 24 hours in association with reperfusion, but then the T wave became prematurely upright over the ensuing 2 to 5 days, unaccompanied by an adverse clinical event (Fig 5). These 4 patients had very small infarcts as manifested by a peak CK-MB value of 15 to 40 IU/L and failure to develop permanent Q waves. Thus, the sensitivity and specificity of these T wave changes for postinfarction pericarditis are 100% and 77%, respectively, inclusive of patients with cardiopulmonary resuscitation, reinfection, and small infarcts. If these three easily identifiable clinical events are excluded, specificity becomes 96%.

CK-MB Data in Lytic Therapy Group

All patients subsequently developed an increase and a decrease in CK-MB, confirming myocardial necrosis.

FIG 1. Schematic diagram depicting the course of T wave evolution after myocardial infarction with and without pericarditis. T represents the customary course without pericarditis. T1 displays the course of those patients with pericarditis expressed as persistently positive T waves. T2 illustrates the course of those patients with postinfarction pericarditis expressed as reversal after inversion of the T waves. Adapted from Lepeshkin E. Modern Electrocardiography. Baltimore, Md: Williams & Wilkins, 1951:413.
Wave patography by regeneration, cases mum 40 minutes Four the leadsthat also developed persistent T waves, in 48 hours and often within two days later, after resolution of the pleuropertencardial pain and a friction rub. d, Recorded 5 days later, after administration of lytic therapy occurred, implying that reperfusion was probably achieved but was succeeded by asymptomatic reocclusion.

The T wave inverted progressively, but slowly, to an amplitude of only 1 to 2 mm within 72 hours in the lead that initially displayed the greatest ST segment elevation in 11% of patients (Fig 6, c and d). In nine (82%) of these instances, angiography disclosed an occluded infarct-related artery, and the CK-MB curve indicated no reperfusion. In the two other patients, the infarct-related artery was patent, whereas the CK-MB curve also did not show an early peak, implying that patency was due to spontaneous late reperfusion.

The T wave inverted to an intermediate value of 3 mm in the lead initially displaying the maximum ST segment elevation in 24 patients, 21 (90%) of whom had a patent infarct-related artery, and 18 of whom had early reperfusion. If a maximum T wave negativity (Tmax) of 3 mm or more is used to separate patients with a patent artery from those without, the sensitivity, specificity, and predictive value of a Tmax of 3 mm or more are 89%, 87%, and 92%, respectively. If a Tmax of 4 mm or more is used, the sensitivity is, predictably, reduced, whereas the specificity and predictive value are enhanced (Table 1). The probability of a given Tmax indicating a patent artery also is given in Table 1.
FIG 3. Serial ECGs of a patient with an acute anterior infarction followed by pericarditis between days 3 and 6. a, ST segment elevation and tall peaked T waves characteristic of the hyperacute phase of myocardial infarction. b, Recorded 48 hours later, after reperfusion with lytic therapy, discloses the expected T wave inversions with the maximum T wave negativity existing in lead V2 (6 mm), the lead that showed the greatest ST segment elevation initially. On January 10, the patient developed pleuropericardial pain, which persisted for 3 days, accompanied by an intermittent friction rub. c, d, e, f, show gradual reversal of the T wave polarity and a gradual reelevation of the ST segment, without reelevation of the creatine kinase-MB. Maximum reversal of the T wave was attained on January 12. The pain remitted over the ensuing 24 to 36 hours. The ECGs on January 14 and 17 show a gradual reduction of the accentuated T waves, finally again becoming inverted.

Discussion
The diagnosis of regional postinfarction pericarditis has remained elusive. During the course of a recent study attempting to identify any clinical markers allowing prediction of cardiac rupture, it was observed that the T wave evolution after infarction and before rupture...
followed an atypical pattern. This pattern was found to be due to associated regional pericarditis in 99% of cases. The serial ECGs of 100 patients with acute myocardial infarction without rupture were reviewed to determine if this unusual pattern was distinctive of rupture. It was learned that the same pattern existed in patients with early postinfarction pericarditis and rarely in its absence. It also was noted that T wave evolution in those patients without pericarditis, who received thrombolytic therapy with resultant reperfusion, appeared to follow an ordinary but accelerated course.

Therefore, the serial ECGs of a separate 100 patients with acute myocardial infarction, all of whom received thrombolytic therapy, were reviewed to further assess the effect of reperfusion on the rapidity and amplitude of postinfarction T wave inversion.

**Effect of Reperfusion on Rapidity and Amplitude of T Wave Evolution**

Both the rapidity and depth of T wave inversion depend on whether reperfusion of the infarcted myocardium occurs. When reperfusion exists, a maximum T
Effect of Regional Pericarditis on T Wave Evolution

The foremost finding of this study is that when pericarditis succeeds infarction, a different pattern of T wave evolution emerges. This departure of the pattern of T wave evolution from the customary course occurred in all instances of clinically recognized pericardi-

TABLE 3. Relation of T<sub>max</sub> to Time to T<sub>max</sub>

<table>
<thead>
<tr>
<th>T&lt;sub&gt;max&lt;/sub&gt;</th>
<th>No. of patients</th>
<th>Peak creatine kinase–MB (IU/L)</th>
<th>Time to T&lt;sub&gt;max&lt;/sub&gt; (h)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>Range</td>
<td>Mean</td>
</tr>
<tr>
<td>≥4 mm†</td>
<td>36</td>
<td>15-378</td>
<td>196</td>
</tr>
<tr>
<td>3 mm</td>
<td>24</td>
<td>50-374</td>
<td>178</td>
</tr>
<tr>
<td>1 to 2 mm</td>
<td>11</td>
<td>55-348</td>
<td>214</td>
</tr>
<tr>
<td>T isoelectric to positive‡</td>
<td>15</td>
<td>65-366</td>
<td>224</td>
</tr>
<tr>
<td>Reversal after inversion§</td>
<td>14</td>
<td>60-354</td>
<td>218</td>
</tr>
</tbody>
</table>

*T<sub>max</sub> is the maximum depth in millimeters of T wave inversion in the single lead with the greatest inversion.
†Includes the 4 patients with a very small infarct in whom the T wave rapidly inverted but then was succeeded by premature reversal.
‡Includes 10 patients with pericarditis, 3 with cardiopulmonary resuscitation, and 2 with no apparent cause.
§Includes 5 patients with pericarditis and 9 with reinfarction in whom early T wave reversal was followed by the development of a positive deflection.
The T wave changes consisted of persistently positive deflections after 48 to 72 hours or gradual, premature reversal of initially inverted T waves. The T wave changes were misleading and deceptive because they ostensibly rendered the ECG more "normal" in appearance. Among patients with multisegment infarcts (e.g., those involving the anterior, lateral, and superior walls), the T wave often inverted normally in one region but not in others. We believe that the region in which the T wave did not invert or in which reversal followed inversion was the site of pericarditis. This notion is supported by the pathological observations in the patient whose ECGs are displayed in Fig 4.

The T wave changes seen in postinfarction pericarditis were sometimes (40% of instances) accompanied by persistent, progressive, or recurrent ST segment elevation in the leads revealing the T wave alterations, presumably also reflecting the overlying regional pericarditis. The T wave changes were more consistent and much easier to discern than the ST segment changes because the T wave alterations were directional, whereas the ST segment alterations were magnitudinal. We, like Kranin and colleagues, found widespread ST segment elevation, a time-honored sign of diffuse pericarditis, in only 2 of 43 patients (5%).

The only other identified conditions that caused a deviation of the T wave evolution from the customary course were, not surprisingly, cardiopulmonary resuscitation, reinfarction, and, interestingly, very small infarcts. Differentiation of the latter from pericarditis is not difficult because small infarcts are associated with minimal or no QRS changes of infarction and a minimal CK-MB increase, whereas postinfarction pericarditis is usually associated with definite QRS changes of infarction and a peak CK-MB of more than 50 IU/L. The cause of the persistently positive T waves in the five patients without clinically evident pericarditis who did not receive cardiopulmonary resuscitation or experience any other apparent adverse clinical event could have been asymptomatic and acoustically silent pericarditis or painless reinfarction. Conversely, pericarditis might have existed but not been detected by the T wave criteria described as a result of other forces and factors that may also affect T wave polarity, such as conduction defects and potassium abnormalities, which constituted exclusions from analysis.
Pathophysiological and Biochemical Considerations

We believe that both the persistently positive T waves and the reversal of inverted T waves are the ECG expressions of the same morphological and biochemical processes at different times. During the first few days of pericarditis associated with various other etiologies, the ECG initially discloses ST segment elevation with upward, if not accentuated, T waves.5 These changes have been attributed to subepicardial inflammation.5,6,8,16 Thus, morphologically, when these ECG changes occur after myocardial infarction, they most likely indicate that the necrotic front has reached the subepicardial myocardium. Inverted T waves overlying the infarct zone reflect subepicardial ischemia and are ascribed to asynchronous repolarization created by delayed repolarization within the ischemic subepicardial zone.17,18 If this layer becomes necrotic, more synchronous repolarization may ensue, resulting in either persistently positive T waves or reversal of inverted T waves, depending on how quickly the necrotic process reaches the subepicardial myocardium. Furthermore, the T wave alterations observed in this clinical study of postinfarction regional pericarditis were very similar to those observed experimentally by Burchell and colleagues,19 when acute myocardial infarction was accompanied by localized pericarditis in the dog.

Biochemically, the basis for the alteration of T wave polarity may be considered the result of multiple factors, including hypoxia, acidosis, and, probably most important, regional hyperkalemia due to the accumulation of potassium in the extracellular space from intracellular sources as a consequence of the loss of subepicardial myocardial cell membrane integrity as ischemia advances to necrosis.16 Experimentally, the production of subepicardial injury by various physical and chemical stimuli, including the application of potassium to the pericardium, results in elevation of the T wave and ST segment.20

Historical Background and Practical Implication

Although neither the failure of early ST segment and T wave discordancy to occur nor the premature concordancy of the ST segment and T wave that we described have been recognized previously as signs of regional pericarditis following infarction, they have, interestingly and unwittingly, been reported. In 1934, Barnes21 emphasized that when pericarditis follows infarction, the ST segment is elevated in "all leads" on a four-lead ECG. Scrutiny of the ECGs in the eight cases reported also reveals persistently positive T wave (or perhaps reversal may have followed inversion) 2 to 8 days after infarction and delayed inversion between days 8 and 21, after resolution of the pericarditis. Why 58 years elapsed before the significance of this observation was appreciated is unclear. The explanation may merely reside in the fact that the T wave normally is upright, except in leads aVR and V1, on the 12-lead ECG that became standard in 1944,22 and thus when T waves remain or become upright after infarction, we have tended to view this auspiciously rather than ominously. In fact, T wave inversion following infarction is the norm and implies sparing of the subepicardial myocardium, whereas persistently positive T waves or premature reversal after inversion indicates that the infarct has traversed this region and reached the visceral pericardium, irrespective of the development or absence of Q waves.

Acknowledgments

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