

Pathologic Implications of Restored Positive T Waves and Persistent Negative T Waves After Q Wave Myocardial Infarction

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Objectives. We sought to study the pathologic implications of restored positive T waves and persistent negative T waves in the chronic stage of Q wave myocardial infarction.

Background. Some inverted T waves (coronary T waves) become positive after acute myocardial infarction; others retain their negative T wave component for a long time. The pathologic implications of the difference between restored positive T waves and persistent negative T waves in leads with Q waves has not, until now, been given much careful study.

Methods. Of 17 patients with anterior or anteroapical myocardial infarction confirmed by autopsy, 8 (group P) had positive and 9 (group N) had negative T waves in precordial leads with Q waves ≥ 1 year after the onset of myocardial infarction. The appearance and extent of the infarct area and the degree of coronary artery stenosis were evaluated in both groups.

Results. At autopsy, seven of eight patients in group P had nontransmural fibrotic changes in the anteroapical or anterior wall. However, seven of nine patients in group N had a transmural myocardial infarction consisting of only a thin fibrotic layer in the anteroapical or anterior wall. The left anterior descending coronary artery showed 75% stenosis in 1 patient in each group but $>90\%$ stenosis in the remaining 15 patients.

Conclusions. Persistent negative T waves in leads with Q waves in the chronic stage of myocardial infarction indicate the presence of a transmural infarction with a thin fibrotic layer, whereas positive T waves indicate a nontransmural infarct containing viable myocardium within the layer.

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Many investigators have reported on the relation between electrocardiographic (ECG) findings and the extent of myocardial infarction. Q wave myocardial infarction was thought to imply pathologically transmural myocardial infarction until later reports (1-3) revealed that the presence or absence of Q waves did not permit a distinction between pathologic transmural and nontransmural infarction in autopsy patients. Persistent ST segment elevation after acute myocardial infarction has generally been considered the most helpful indicator of ventricular aneurysm (4-6). In contrast, there have been few reports on the relation between T wave configuration and the extent of myocardial infarction. In patients with myocardial infarction with evolving Q waves after ST elevation, negative T waves (called "coronary T waves") usually develop. Although some negative T waves change gradually and become completely positive, others keep their negative components for a long time. We studied the difference in pathologic findings between restored positive T waves and persistent negative T waves after Q wave myocardial infarction.

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Methods

Case selection. We reviewed all charts and ECGs of 2,000 consecutive autopsy patients and found 17 who had a history of ≥ 1 year of anterior or anteroapical myocardial infarction associated with Q waves in at least three precordial leads, QS waves in at least two leads and myocardial infarction in the territory of the left anterior descending coronary artery. We excluded patients with an ECG showing right or left bundle branch block and those with a history of recurrent myocardial infarction. The patients were classified into two groups according to the characteristics of T waves in leads with Q waves: group P, seven men and one woman, mean age $\pm$ SD 80 ± 8 years, whose ECG showed all T wave components in an upright configuration; and group N, five men and four women, mean age 83 ± 5 years, whose ECG showed a negative T wave fraction.

ECG analysis. We measured amplitude height between the top of the T wave and the ST level (at the J junction) in group P, and the lowest T wave amplitude from the ST level in group N, although these measurements cannot be compared between the two groups. The ST level from the TP segment was also measured in both groups.

Pathologic evaluation. The pathologic findings of myocardial infarction were determined by the presence of macroscopic fibrosis and confirmed by microscopic findings. Exami-

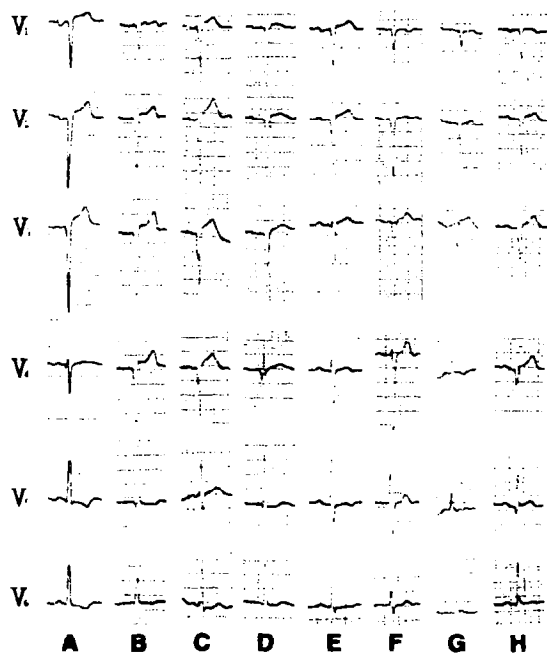


Figure 1. Electrocardiographic leads V_1 to V_6 from Patients A to H (group P), showing positive T waves in leads with Q waves.

nation for coronary artery stenosis was made by cross-sectional slicing at intervals of 3 to 4 mm.

Statistical analysis. Values are expressed as mean value $\pm$ SD. Statistical comparisons between the two groups were performed with use of the Student *t* test. A *p* value < 0.05 was considered significant.

Results

ECG findings. ECGs of chest leads in each autopsy patient are shown in Figures 1 (group P) and 2 (group N). The maximal height of the top of the T waves from the ST line in leads with Q waves was 4.0, 5.0, 4.5, 2.0, 2.0, 2.0, 2.0 and 3.5 mm, respectively, in the eight patients in group P. The minimal height of the T waves from the ST line in leads with Q waves was -5.5, -7.5, -4.5, -6.0, -5.0, -3.0, -2.0, -2.5 and -6.0 mm, respectively, in the nine patients in group N. The mean ST level was 1.8 ± 0.8 mm in group P and 2.1 ± 1.0 mm in group N (*p* = NS).

In group P, six patients had already had a positive T wave tracing on their first visit to our hospital. The polarity of the T waves in these six patients never changed. We were able to observe the other two patients in group P from the onset of infarction. These patients' negative (coronary) T wave tracings became positive within 13 months (at 13 months in one patient and at 12 months in the other). After becoming positive, these T waves were never negative again.

In group N, six patients had already had a negative T wave tracing on their first visit to our hospital. The T wave polarity in these six patients never changed, except in one patient (Patient O) whose T waves became positive for a short time immediately after an exercise ECG-stress test. The other three

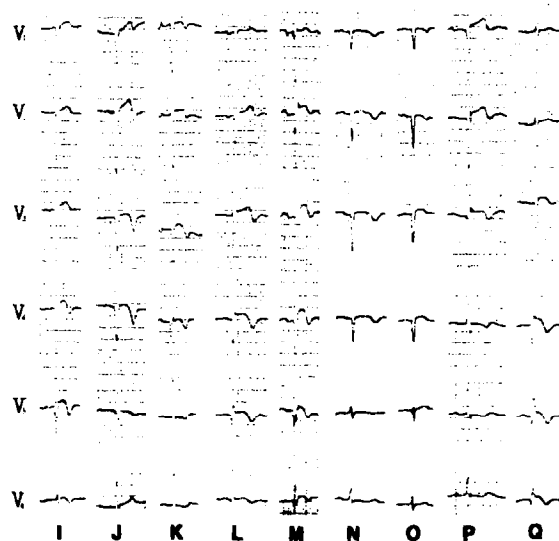


Figure 2. Electrocardiographic leads V_1 to V_6 from Patients I to Q (group N), showing negative T waves in leads with Q waves.

patients in group N were observed from the onset of infarction. These patients' negative (coronary) T waves remained negative, with two exceptions: In Patient I, T wave polarity became positive for a period while the patient was experiencing dyspnea due to pneumonia. In the other, Patient M, T wave

Table 1. Clinical Findings in the Two Patient Groups

Pt. No.	Age (yr)/Gender	History of MI (mo)	T Level (mm)	ST Level (mm)	Cause of Death
Group P: Positive T Waves					
A	86/F	89	4.0	3.0	Sudden death
B	92/M	>108	5.0	2.5	CHF
C	71/M	>36	4.5	2.5	ALS
D	83/M	13	2.0	1.0	Lung cancer
E	72/M	48	2.0	1.5	Sudden death
F	86/M	>60	2.0	1.0	Pneumonia
G	75/M	77	2.0	1.0	Prostate cancer
H	73/M	>88	3.5	1.5	Rupture of TAA
Mean	80	>65		1.8	
$\pm$ SD	± 8	± 32		± 0.8	
Group N: Negative T Waves					
I	90/F	53	-5.5	2.0	Pneumonia
J	77/M	432	-7.5	2.0	Gastric cancer
K	89/F	108	-4.5	1.5	Aortic embolism
L	88/F	75	-6.0	2.0	Pneumonia
M	81/M	20	-5.0	4.0	COPD
N	83/M	72	-3.0	0.5	Liver failure
O	75/M	84	-2.0	1.5	Hodgkins disease
P	85/M	>60	-2.5	2.5	Pneumonia
Q	82/F	19	-6.0	3.0	Acute pancreatitis
Mean	83	>103		2.1	
$\pm$ SD	± 5	127		± 1.0	

ALS = amyotrophic lateral sclerosis; CHF = congestive heart failure; COPD = chronic obstructive pulmonary disease; F = female; M = male; MI = myocardial infarction; Pt = patient; TAA = thoracic aortic aneurysm.

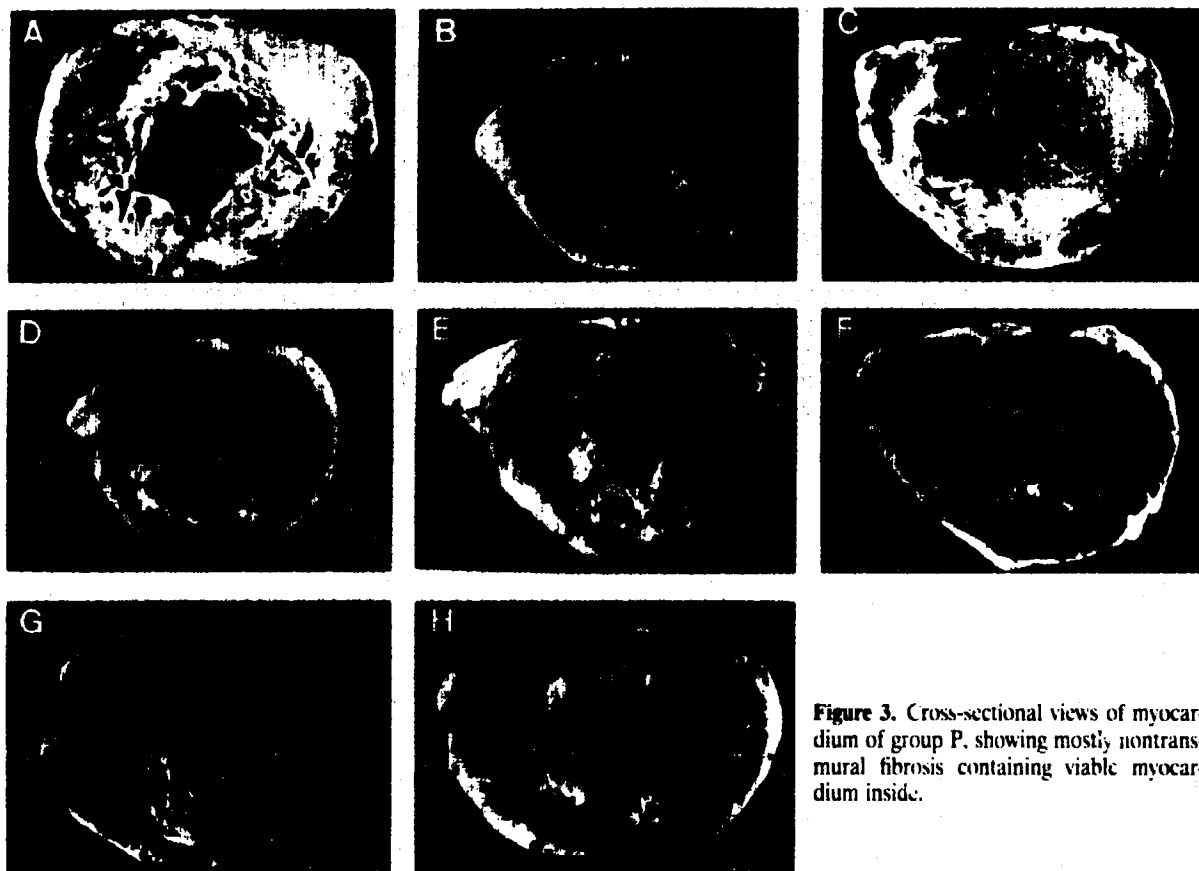


Figure 3. Cross-sectional views of myocardium of group P, showing mostly nontransmural fibrosis containing viable myocardium inside.

polarity became positive just before the patient's death. In all three patients in group N, whose T waves changed from negative to positive (Patients I, M and O), the change coincided with an increased heart rate.

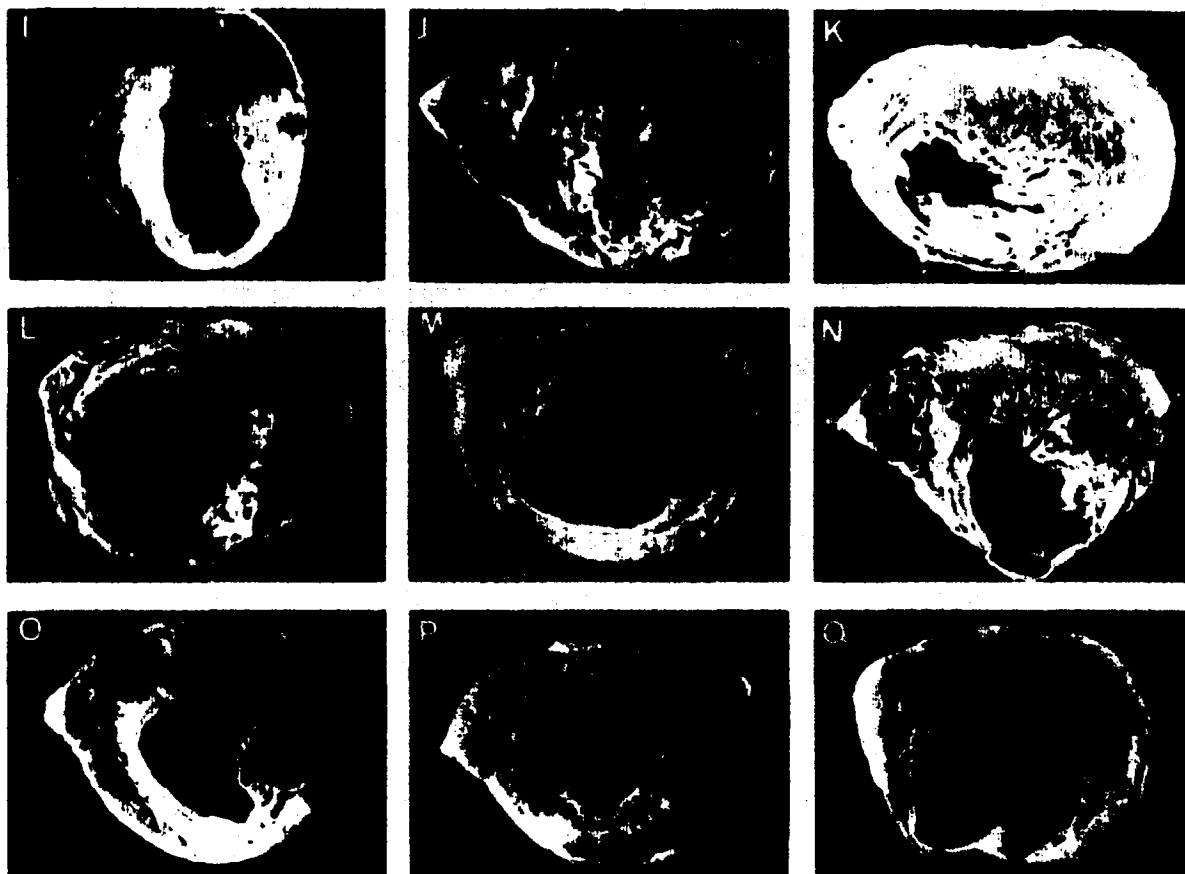
Time to death. Clinical findings are shown in Table 1. The interval between onset of myocardial infarction and death was $\geq 65 \pm 32$ months in group P and $\geq 103 \pm 127$ months in group N. Two patients in group P (Patients A and E) died within 30 min after the onset of dyspnea and back pain, respectively; one patient (Patient B) died of heart failure. No patient in group N died of cardiac causes.

Characteristics of infarction. Pathologic findings showed $\geq 90\%$ stenosis of the left anterior descending coronary artery in all patients except one patient in each group with 75% stenosis. Heart weight did not differ between the two groups (group P, 419 ± 71 g; group N, 359 ± 102 g). Although one patient in group P (Patient C) showed transmural fibrosis at the septum of the anterior side, the other 7 patients in group P showed nontransmural fibrotic changes in the anteroseptal or anterior wall (Fig. 3). In contrast, seven of the nine patients in group N showed transmural myocardial infarction with only a very thin fibrotic layer in the anteroseptal or anterior wall. Another patient in group N (Patient P) showed transmural fibrosis 1 cm in length, but viable myocardium in the trabeculae and mainly subendocardial fibrosis in the anterior wall. An-

other (Patient J) showed near transmural infarction including scattered viable myocardium inside (Fig. 4).

Discussion

Extent of infarction and T wave configuration. Many investigators have reported on the relation between ECG findings and the extent of myocardial infarction. One area of study was the relation between the presence or absence of Q waves and transmural or nontransmural myocardial infarction, although it has been proved (1-3) that Q waves do not always imply transmural infarction. In our study 9 of 17 patients with Q wave infarction had nontransmural infarction and the remaining 8 transmural infarction by autopsy, thus demonstrating that the presence of Q waves does not distinguish between transmural and nontransmural infarction. It has also been reported (4-6) that persistent ST elevation after acute myocardial infarction indicates ventricular aneurysm, whose presence has generally been confirmed by ventriculography or echocardiography. In contrast, relatively little attention has been given to the relation between the configuration of T waves and the extent of myocardial infarction. In the present study, we evaluated the relation between restored positive T waves or persistent negative T waves in leads with Q waves and the extent of myocardial infarction.



Persistent negative T waves were related to the presence of pathologically confirmed transmurular myocardial infarction, that is, the absence of viable myocardium at the left ventricular wall under the surface of leads with Q waves. However, positive T waves were related to nontransmurular myocardial infarction containing viable myocardium inside the layer.

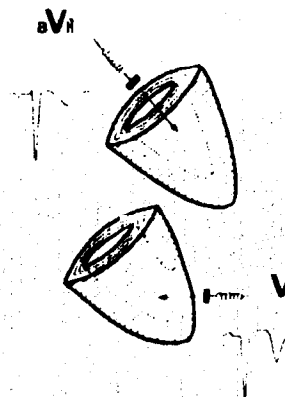
Negative T waves in the setting of an ischemic event are considered to be caused by prolongation of repolarization of injured myocardial cells (7,8). As all patients in our series had a long history of myocardial infarction and those in group N had a clearer distinction between the lesion of myocardial infarction and viable myocardium than did patients in group P, the presence of unstable myocardial cells does not seem to be a cause of negative T waves in group N. As shown in Figure 4, the myocardial lesions in group N consisted of only a fibrotic thin layer, which does not have electrical activity. ECG findings in the leads on the surface of these lesions seem to reflect those of the subendocardial leads of the opposite site of the myocardial wall, such as lead aVR, which always shows negative T waves in normal subjects (Fig. 5). The lack of viable myocardium with electrical activity in pathologically assessed transmurular infarction seems to be a cause of persistent negative T waves in leads with Q waves.

ST elevation and ventricular aneurysm. Although there is no precise definition for "persistent ST elevation" and "ven-

Figure 4. Cross-sectional views of myocardium of group N, showing transmurular infarction with only a thin fibrotic layer.

tricular aneurysm." ST elevation is commonly cited as a sign of left ventricular aneurysm. However, several investigators (9,10) have reported less association between these findings. If

Figure 5. Schematic representation of negative T waves in lead aVR of a normal subject (top) and a patient with transmurular infarction (bottom). Both leads reflect the endocardial leads of the opposite side. V = precordial leads.



we define ST elevation as an ST level ≥ 2.0 mm (0.2 mV) in amplitude from the baseline and ventricular aneurysm as external protrusion of fibrotic tissue, three of eight patients in group P and six of nine patients in group N showed ST elevation, and at least four patients in group N (Patients 1, L, N, O) appeared to have ventricular aneurysm. However, not only all nine patients in group N—but also these four patients with apparent ventricular aneurysm (who had a mean ST level of 1.9 ± 1.0 mm)—showed no difference in ST level from that in group P. We could not distinguish between nontransmural infarction and transmural infarction or ventricular aneurysm from the presence or absence of ST elevation.

The configuration of T waves may be more helpful in distinguishing between nontransmural infarction and transmural infarction or ventricular aneurysm than is the presence or absence of ST elevation in the chronic stage of Q wave myocardial infarction.

Conclusions. Persistent negative T waves in leads with Q waves in the chronic stage of myocardial infarction indicate the presence of pathologically transmural infarction consisting of a thin fibrotic layer, whereas positive T waves indicate nontransmural infarction containing viable myocardium within the layer of the ventricular wall under the electrodes.

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