

Persistent Negative T Waves in the Infarct-Related Leads as an Independent Predictor of Poor Long-Term Prognosis After Acute Myocardial Infarction

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This study sought to determine the long-term prognostic significance of persistent or transient negative T waves in infarct-related leads. After acute myocardial infarction (AMI), QRS and T wave alterations may resolve. No clinical study has investigated the prognostic importance of persistent versus transient negative T waves. We studied 147 consecutive patients with first AMI and ≥ 2 negative T waves in the infarct-related leads on the electrocardiogram. One hundred twenty patients developed Q waves. Patients were followed clinically for 60 ± 21 months. T-wave normalization was observed early (before hospital discharge) in 34 patients and late (at 4 ± 1 months) in 65. Thirty patients had Q-wave regression. Adverse outcome occurred in 57 patients. There were 23 hard events (cardiac death in 12 patients and

nonfatal AMI in 11). Patients with early or late T-wave normalization had similar event-free survival curves that diverged rapidly from that of patients with persistent negative T waves, who had a worse outcome ($p < 0.0001$). Patients with or without Q-wave regression had similar survival curves. Using multivariate Cox regression analysis, higher end-systolic volume (hazard ratio [HR] 1.01, $p = 0.007$), the presence of multivessel disease (HR 3.33, $p = 0.009$), and persistent negative T waves (HR 2.92, $p = 0.024$) predicted hard events. Persistent negative T waves 4 months after first AMI were independently associated with a worse outcome, whereas Q-wave regression has no long-term prognostic importance. ©2002 by Excerpta Medica, Inc.

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The natural history of electrocardiographic changes after acute myocardial infarction (AMI) is well established. New Q waves at presentation are independently associated with a worse outcome.¹ Q-wave regression is related to a smaller infarct size,² a larger amount of stunned myocardium,³ and less left ventricular dysfunction,⁴ but its prognostic significance remains controversial.^{5,6} The appearance of negative T waves early after AMI is associated with effective thrombolytic treatment, a benign in-hospital course,⁷ and preserved left ventricular function.⁸ Spontaneous normalization of negative T waves in infarct-related leads predicts small infarct size,⁶ more viable myocardium,⁹ and recovery of regional dysfunction.¹⁰ Functional recovery is better when T-wave normalization occurs earlier.¹¹ Persistent negative T waves in the chronic stage of myocardial infarction have been found to be associated with the presence of transmural necrosis with a thin fibrotic layer, whereas restored positive T waves indicate a nontransmural infarct containing viable myocardium within the layer,¹² but these observations were made in a small autopsy study. No clinical study has investigated the prognostic significance of persistent or transient negative T waves after AMI. The aim of this study was to exam-

ine the long-term prognostic importance of persistent versus early or late normalization of negative T waves after inaugural AMI. The significance of Q-wave regression was also investigated.

METHODS

Study patients: This study was a retrospective analysis of prospectively recorded data in 147 consecutive patients with the following inclusion criteria: (1) diagnosis of a first AMI (typical chest pain lasting ≥ 30 minutes with acute ST-segment elevation in ≥ 2 leads and an increase in serum creatine kinase to at least twice the upper limit of normal); (2) normal intraventricular conduction and no electrocardiographic signs of left ventricular hypertrophy; (3) inverted T waves in ≥ 2 infarct-related leads 24 to 36 hours after the acute event; (4) coronary angiography and resting 2-dimensional echocardiography performed during hospitalization; and (5) no cardiac events between inclusion and the 4-month follow-up electrocardiogram.

Electrocardiographic analysis: Standard 12-lead electrocardiograms were recorded on hospital admission, at 24 to 36 hours after AMI, at hospital discharge (13 ± 2 days), and at 4 ± 1 months after AMI. The T wave was considered inverted if it was monophasic and its amplitude was ≥ 0.1 mV. Abnormal Q waves were defined as ≥ 40 ms and/or $> 25\%$ of the R-wave amplitude. T-wave normalization corresponded to abnormal negative T waves becoming positive in ≥ 2 infarct-related leads either at hospital discharge (early) or at 4 months (late). Q-wave regression was defined

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	Early T-wave Normalization (n = 34)	Late T-wave Normalization (n = 65)	Persistent Negative T Waves (n = 48)	Q-wave Regression (120 Q-wave AMI)	
				Yes (n = 30)	No (n = 90)
Revascularization before hospital discharge	17 (50%)	52 (80%) [†]	24 (50%)	21 (70%)	52 (58%)
Hard events	3 (9%)*	5 (7%) [†]	15 (31%)	6 (20%)	16 (18%)
Death	1 (3%)	3 (5%)	8 (16%)	3 (10%)	8 (9%)
Nonfatal AMI	2 (6%)	2 (2%)*	7 (15%)	3 (10%)	8 (9%)
Adverse events	9 (26%)*	21 (32%)*	27 (56%)	11 (37%)	40 (44%)
Unstable angina	6 (17%)	11 (17%)	8 (17%)	4 (13%)	18 (20%)
Heart failure	0 (0%)	5 (8%)	4 (8%)	1 (4%)	6 (8%)

*p <0.04; [†]p <0.003 versus persistent negative T waves.

as Q-wave disappearance and reappearance of R waves ≥ 0.1 mV in ≥ 1 of the infarcted-related leads.

Coronary angiography: Quantitative angiography and left ventriculography were performed in all patients within 1 week after the acute event. Quantitative measurements of coronary stenoses were performed with the use of the Cardiovascular Angiography Analysis System (CAAS, Philips Integris-Medical System, Eindhoven, The Netherlands).

Follow-up data: Outcome was determined from patient interviews, hospital chart reviews, telephone interviews, and/or personal communications with the patient's physician. Four types of cardiac events were identified as adverse events: cardiac death, nonfatal AMI, unstable angina, and congestive heart failure requiring hospitalization. Hard events were considered to be cardiac death and nonfatal AMI. If a patient had >1 event, only the most severe was considered as an end point. Cardiac death was defined as sudden death or death related to AMI, congestive heart failure, or cardiac arrhythmias. Nonfatal AMI was defined as hospital admission for prolonged chest pain with new electrocardiographic changes and a significant increase in serum levels of cardiac enzymes. Unstable angina was defined as accelerating anginal symptoms requiring hospital readmission or progression of symptoms requiring revascularization. Coronary artery bypass surgery and coronary angioplasty during the follow-up period were also recorded.

Statistical analysis: Student's *t* test was used to assess differences between mean values; categorical variables were compared with chi-square test and Fisher's exact test when appropriate. The individual effects of variables on event-free survival were evaluated with the use of the Cox regression model. To detect independent predictors of hard and adverse events, a multivariate Cox regression procedure was performed according to the unmodified forward-selection stepwise analysis. The Kaplan-Meier method was used for cumulative survival analysis with the log-rank test for assessing statistical differences between curves. Cumulative incidence and interval estimates were calculated using the lifetable method. Results are expressed as mean $\pm$ SD. A *p* value of <0.05 was considered statistically significant.

RESULTS

Clinical characteristics: The study population comprised 124 men and 23 women (aged 58 ± 11 years). Thrombolytic therapy was administered to 103 patients. No patient was treated by primary angioplasty. Q-wave myocardial infarction developed in 120 patients. At angiography, persistent occlusion of the infarct-related artery was observed in 23 patients. Mean residual stenosis of the infarct-related artery was $74 \pm 11\%$. Elective angioplasty of the infarct-related lesion was performed in 82 patients. Eleven patients underwent coronary artery bypass graft surgery. Fifty patients had multivessel disease: 34 patients had 2-vessel and 16 had 3-vessel disease.

Electrocardiographic changes: T-wave normalization was observed early in 34 patients and late in 65 patients. T waves remained inverted at 4 months in 48 patients (Table 1). Thirty patients had Q-wave regression at 4 months. Twenty-eight patients had both T-wave normalization (late in 23) and Q-wave regression. Peak level of creatine kinase was lower in patients with early T-wave normalization than in patients with late normalization or persistent negative T waves ($1,333 \pm 659$ vs $1,912 \pm 1,128$ vs $2,272 \pm 1,082$ IU/L, respectively; $p < 0.001$). Residual stenosis of the infarct-related artery was greater in patients with persistent inverted T waves ($80 \pm 20\%$) or late T-wave normalization ($74 \pm 27\%$) than in patients with early T-wave normalization ($59 \pm 23\%$; $p < 0.01$). The use of a revascularization procedure during hospital stay was more frequent in patients with late T-wave normalization (52 patients) than in patients with early normalization (17 patients; $p = 0.0029$) and patients with persistent negative T waves (24 patients; $p = 0.0011$). The incidence of revascularization was not different in patients with or without Q-wave regression (21 of 30 vs 52 of 90; $p = \text{NS}$).

Cardiac events: Patients were followed for 60 ± 21 months. Of the 147 patients studied, 90 had an uneventful clinical course and 57 experienced a cardiac event. These comprised 23 (16%) hard events, including cardiac death in 12 patients and nonfatal AMI in 11. Unstable angina occurred in 25 patients and congestive heart failure required hospitalization in 9. Seventeen patients underwent percutaneous transluminal

Characteristics	Hard Events		Univariate Cox Analysis		Multivariate Cox Analysis	
	0 (n = 124)	+ (n = 23)	Chi-square	p Value	Chi-square	p Value (HR)
Age (yrs)	58 ± 25	61 ± 10	—	NS	—	NS
Men	84%	87%	—	NS	—	NS
Diabetes mellitus	22 (18%)	5 (2%)	—	NS	—	NS
Thrombolytic therapy	84 (68%)	19 (83%)	—	NS	—	NS
Peak creatine kinase (IU/L)	1,795 ± 1,998	2,438 ± 871	11.2	0.0008	—	NS
Peak creatine kinase MB(IU/L)	172 ± 155	206 ± 85	4.8	0.028	—	NS
Anterior infarction	54 (43%)	16 (69%)	5.3	0.022	—	NS
Q-wave infarction	98 (79%)	22 (96%)	—	NS	—	NS
Echo score index	1.38 ± 0.56	1.57 ± 0.38	10.6	0.0011	—	NS
Multivessel CAD	35 (28%)	15 (65%)	11.5	0.0007	7.1	0.009 (3.33)
LV ejection fraction (%)	59 ± 24	52 ± 16	7.8	0.005	—	NS
End-diastolic volume (ml/m ²)	95 ± 51	110 ± 38	7.1	0.009	—	NS
End-systolic volume (ml/m ²)	41 ± 44	61 ± 41	10.1	0.0012	7.5	0.007 (1.01)
Revascularization	83 (67%)	10 (43%)	4.9	0.026	—	NS
Treatment						
ACE inhibitor	30 (24%)	8 (35%)	—	NS	—	NS
β blocker	64 (52%)	9 (39%)	—	NS	—	NS
Persistent negative T waves	33 (27%)	15 (65%)	13.6	0.0002	5.2	0.024 (2.92)
Q-wave regression	24 (19%)	6 (26%)	—	NS	—	NS

ACE = angiotensin-converting enzyme; CAD = coronary artery disease; HR = hazard ratio; LV = left ventricular.

coronary angioplasty during follow-up and 7 were referred for coronary artery bypass graft surgery.

Predictors of hard events: The clinical characteristics of the patients with or without hard events are listed in Table 2. Two electrocardiographic parameters, persistent negative T waves and anterior infarct location, were more frequent in patients who experienced a hard cardiac event. With multivariate Cox regression analysis, 3 variables were found to be independent predictors of hard cardiac events: a high end-systolic volume, the presence of multivessel disease, and persistent negative T waves in the infarct-related leads at 4 months.

The event-free cumulative survival curves for hard events in patients with or without persistent negative T-waves are depicted in Figure 1. Patients with either early or late T-wave normalization had similar hard event-free survival, whereas patients with persistent negative T waves in the infarct-related leads had a worse outcome, with an event-free curve differing rapidly from those of the 2 other groups ($p < 0.0001$). Patients with or without Q-wave regression had similar hard event-free survival curves ($p = 0.45$) (Figure 1).

Determinants of adverse events: In multivariate analysis, the independent predictors of adverse events were persistent negative T waves, high echocardiographic score index, and the presence of multivessel disease (Table 3). The Kaplan-Meier adverse event-free survival curves revealed that patients with persistent negative T waves in the infarct-related leads had a highly significant ($p < 0.0001$) lower adverse event-free survival rate than patients with either early or late T-wave normalization (Figure 1). Cumulative survival rates were similar in patients with or without Q-wave regression ($p = 0.15$) (Figure 1).

DISCUSSION

Hitherto, studies addressing the significance of changes in negative T waves over time after AMI have

concentrated on in-hospital outcome¹³ and on recovery of ventricular dysfunction.¹¹ The results of this study indicate that patients with persistent negative T waves have a poorer long-term prognosis than patients with either early or late T-wave normalization. This easily obtainable finding provides independent prognostic information in addition to variables such as end-systolic volume, presence of multivessel disease, and echocardiographic score index. T-wave normalization was associated with excellent 5-year outcome; only 8 of 99 patients died ($n = 4$) or had a recurrent AMI ($n = 4$). In contrast, approximately 1/3 of the patients with persistent negative T waves died or experienced recurrent AMI with cardiac events occurring during the first 2 years of follow-up. Only 44% of patients with persistent negative T waves had a 5-year survival without reinfarction, unstable angina, or congestive heart failure requiring hospitalization.

Prognostic value of the electrocardiogram after AMI: Although the independent risk factors for early and late mortality are substantially different,¹⁴ short- and long-term prognosis after AMI are strongly related to early establishment of patency of the infarct-related artery and preserved left ventricular function.¹⁵ The role of the electrocardiogram in identifying successful thrombolysis has been emphasized. Rapid resolution of ST-segment elevation is a surrogate for both infarct-vessel patency and microvascular integrity.¹⁶ Early T-wave inversion in the infarct-related leads is also a reliable marker of coronary reperfusion¹⁷ and is associated with greater in-hospital¹³ and 1-month¹⁸ survival rates. Although QRS and T wave alterations usually resolve, the long-term prognostic importance of persistent or transient electrocardiographic abnormalities is controversial. The persistence of Q waves has been found to be associated with a higher mortality rate than Q-wave disappearance,¹⁹ but other investigators

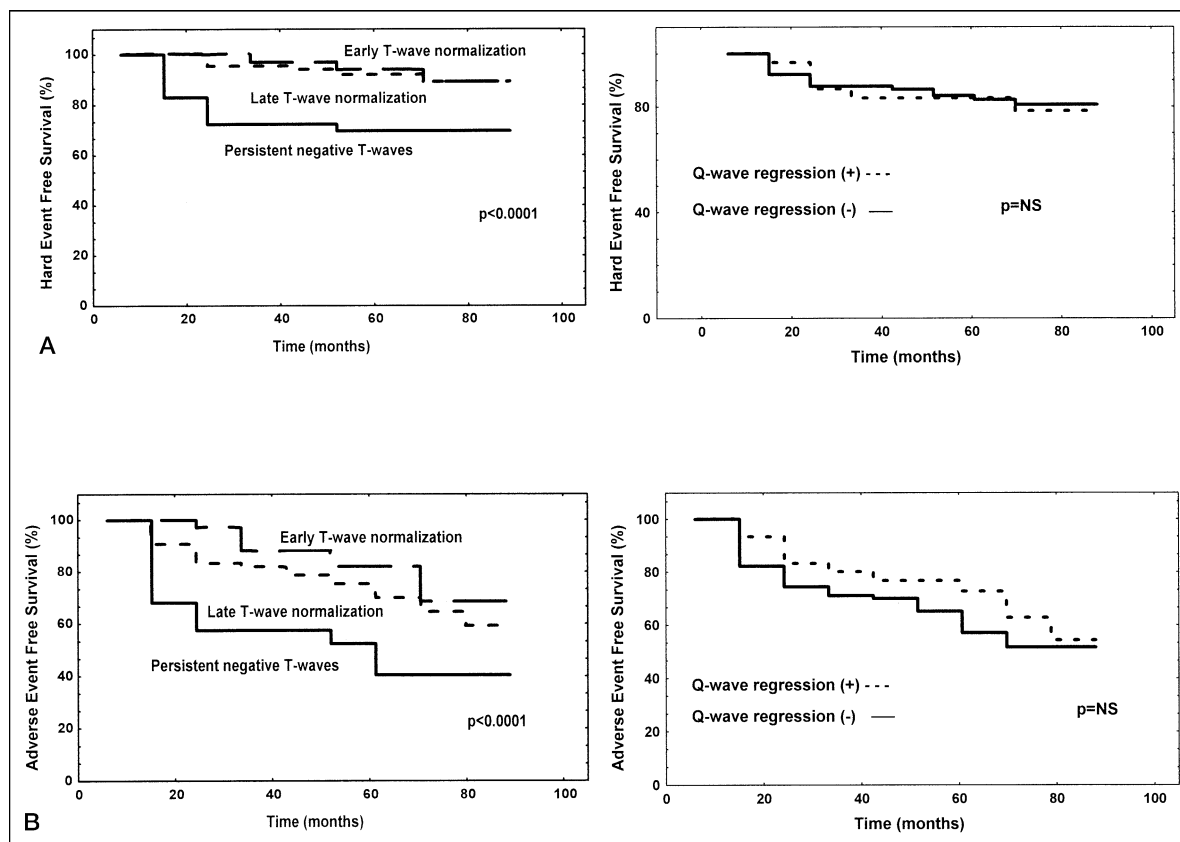


FIGURE 1. Kaplan-Meier survival curves drawn for hard (A) and adverse (B) cardiac events in patients with persistent negative T waves, early and late T-wave normalization (left panels), and with or without Q-wave regression (right panels) in the infarct-related leads.

TABLE 3 Determinants of Adverse Events

Variable	No Adverse Events (n = 90)	Adverse Events (n = 57)	Univariate Cox Analysis		Multivariate Cox Analysis	
			Chi-square	p Value	Chi-square	p Value (HR)
Anterior infarction	40 (44%)	30 (53%)	—	NS	—	NS
Peak creatine kinase (IU/L)	1,769 ± 1,006	2,095 ± 965	4.41	0.036	—	NS
Echocardiographic score index	1.35 ± 0.26	1.49 ± 0.34	13.9	0.0002	10.4	0.0016 (1.00)
Multivessel CAD	22 (24%)	28 (49%)	10.2	0.0014	6.1	0.015 (1.96)
Persistent negative T waves	21 (23%)	27 (47%)	12.1	0.0004	7.7	0.006 (2.13)
Q wave regression	19 (21%)	11 (19%)	—	NS	—	NS

Abbreviations as in Table 2.

have not found a difference in survival.^{2,20} The conflicting results may be related to different definitions of Q-wave regression and to patient selection. In our study, which included patients with evolving ST-segment elevation, the presence or absence of Q-wave regression had no influence on long-term prognosis. The mortality rate and the distribution of cardiac events were similar in both groups. In contrast to QRS changes, persistent negative T waves had a significant influence on long-term survival.

Prognostic factors after AMI: The results of the present study confirm the predictive value of several parameters. In addition to T-wave evolution, 3 other independent variables were found to predict hard and adverse events: end-systolic volume,²¹ multivessel

disease,²² and echocardiographic score index.²³ The prognostic value of end-systolic volume has already been emphasized in the prethrombolytic era.²¹ The echocardiographic wall motion score index integrates the extent of necrotic, stunned, and/or hibernating regions; it has been found to be predictive of adverse outcome.²⁴ Persistent negative T waves 6 months after AMI has been associated with poor recovery and with left ventricular remodeling suggestive of the presence of transmural necrosis and irreversible injury in the affected area.⁶ In contrast, QRS changes did not predict recovery or ventricular enlargement.⁶ The number of diseased vessels has previously been found to be comparable in patients with and without early T-wave inversion.⁷ In our study, the proportion of patients

with multivessel disease was higher in those with persistent negative T waves (24 of 48) than in those with T-wave normalization (26 of 96) ($p = 0.017$). These findings indicate that negative T waves on the electrocardiogram, 4 months after AMI, are associated with left ventricular dysfunction and more extensive coronary artery disease.

Mechanisms of QRS and T-wave changes: The mechanisms underlying normalization of the QRS complex are not completely understood. Loss of Q waves might be due to the combination of retraction of scarred tissue and hypertrophy of the adjacent myocardium.²⁵ After AMI, negative T waves occasionally become completely positive. A recent study has shown that the earlier the negative T waves reverted to positive, the greater the improvement of left ventricular function.¹¹ In our study, patients with early T-wave reversion had smaller infarct size and less severe residual infarct artery stenosis. Preservation of coronary flow reserve associated with greater amount of salvaged myocardium argues for the presence of stunned myocardium in these patients. In contrast, persistent T-wave inversions at hospital discharge may be related to the presence of jeopardized myocardium or extensive necrosis. In these patients, poor residual perfusion or blunted flow reserve may result in hibernating myocardium. Restoration of blood flow by coronary revascularization, which may allow occurrence of electrical stability, was obtained in 80% of patients with late T-wave normalization compared with only 50% of patients with persistent T-wave inversion. Recent data suggest that the hibernating myocardial state represents a progressive and dynamic phenomenon.²⁶ Absent or incomplete revascularization may facilitate apoptosis and increase the proportion of fibrotic tissue. Negative T waves that persist after 4 months of electrocardiographic follow-up might be associated with either extensive necrosis or unstable hibernating myocardium. In the chronic stage of myocardial infarction, persistent negative T waves were associated with the presence of a thin fibrotic layer corresponding to transmural necrosis.¹²

Study limitations: Our observations pertain only to patients admitted to the hospital with AMI, who developed ST-segment elevation and negative T waves in the infarct-related leads and who survived ≥ 4 months after the acute event. We did not evaluate the time course of T-wave normalization before and after the 4-month electrocardiographic follow-up. The possible prognostic impact of later T-wave changes could not be assessed. The decision to perform coronary revascularization in the acute stage was made by the physician in charge of the patient. This may have influenced prognosis. Coronary angiography was not repeated at follow-up and a relation between restenosis or reocclusion and persistent negative T waves could not be excluded. The validity of our observations should be prospectively tested in a larger series of patients.

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