CHEST

Official publication of the American C ollege of Chest Physicians



Prognostic significance of the initial electrocardiographic pattern in a first acute anterior wall myocardial infarction.

Y Birnbaum, S Sclarovsky, A Blum, A Mager and U Gabbay

Chest 1993;103;1681-1687 DOI 10.1378/chest.103.6.1681

The online version of this article, along with updated information and services can be found online on the World Wide Web at: http://chestjournal.chestpubs.org/content/103/6/1681

Chest is the official journal of the American College of Chest Physicians. It has been published monthly since 1935. Copyright1993by the American College of Chest Physicians, 3300 Dundee Road, Northbrook, IL 60062. All rights reserved. No part of this article or PDF may be reproduced or distributed without the prior written permission of the copyright holder. (http://chestjournal.chestpubs.org/site/misc/reprints.xhtml) ISSN:0012-3692



Downloaded from chestjournal.chestpubs.org by guest on July 13, 2011 © 1993 American College of Chest Physicians

Prognostic Significance of the Initial Electrocardiographic Pattern in a First Acute Anterior Wall Myocardial Infarction*

Yochai Birnbaum, M.D.; Samuel Sclarovsky, M.D.; Arnon Blum, M.D.; Aviv Mager, M.D.; and Uri Gabbay, M.D.

The availability of potent, but potentially dangerous, types of reperfusion therapy for acute myocardial infarction (AMI) has forced us to refine our tools for early and accurate diagnosis and for early risk stratification of patients with evolving AMI. The estimation of risk has to be made shortly after admission, when only the history, physical examination, and the ECG are available. This study was undertaken to compare in-hospital mortality with different patterns of the ECG obtained at admission in 147 consecutive patients with an evolving first AMI of the anterior wall. By using a new classification of the admission ECG, it was possible to divide the patients into 3 groups: (1) group A contained 12 patients with tall peaked T waves in the involved leads, without ST segment elevation; (2) group B comprised 77 patients with abnormal T waves and ST elevation, but without major changes in the terminal portion of the QRS complex; and (3) group C comprised 58 patients with abnormal T waves, ST elevation, and distortion of the

The availability of potent, but potentially dangerous, types of reperfusion therapy for acute myocardial infarction (AMI) has forced us to refine our tools for early and accurate diagnosis and for early risk stratification of patients with an evolving AMI. The estimation of risk needs to be made shortly after admission, when only the history, physical examination, and the ECG are available.

The ECG is simple and noninvasive and can be obtained at bedside, and its role in the diagnosis of AMI is well established;¹ however, the role of the ECG obtained at admission in predicting the prognosis and in risk stratification is less clear. In the early 1970s, it was found by epicardial and precordial mapping that the magnitude of ST segment elevation is a reflection of the extent of myocardial injury.²⁻⁵ As a result, ST segment elevation was used to define subsets of patients who can benefit most from early thrombolysis^{1,6,7} and even to monitor the effects of reperfusion therapy;8-10 however, the magnitude of ST elevation is influenced not only by the extent and severity of the ischemia, but also by variations of the shape and size of the chest and by localization of the infarction.11

terminal portion of the QRS. The mortality was 0, 3 percent, and 29 percent in groups A, B, and C, respectively $(\chi^{s}=22.91; p=0)$. By using a logistic regression model, it was found that the initial ECG pattern alone is a strong predictor of in-hospital mortality in patients with an evolving anterior wall AMI. The predicted probabilities of death in groups A, B, and C are 0.0016, 0.025, and 0.29, respectively. This simple classification of the initial ECG pattern in patients with a first AMI of the anterior wall may enable the differentiation of patients with low in-hospital mortality (groups A and B) and of those with an in-hospital mortality of almost 30 percent (group C).

(Chest 1993; 103:1681-87)

AMI = acute myocardial infarction; CK = creatine kinase; CK-MB = MB fraction of creatine kinase; rtPa = recombinant tissue-type plasminogen activator; VSD = ventricular septal defect

The purpose of this study is to correlate the different patterns of the admission ECG of patients with an evolving first anterior AMI to in-hospital mortality, in order to define a simple ECG tool that will help the clinician to estimate the risk of a patient with anterior AMI shortly after admission. Instead of using absolute measurements of ST elevation, we divided the patients into three groups according to the pattern of the admission ECG. We considered the relations between the T wave, the ST segment, and the terminal portion of the QRS complex.

MATERIALS AND METHODS

Patients

We studied the records of all patients admitted to the coronary care unit from January 1988 to March 1991 who were in the early stages of an evolving first AMI involving the anterior wall. We studied only those patients who presented before inversion of the T wave or evolution of an abnormal Q wave had occurred. Patients with a history or ECG evidence of a previous AMI were excluded. Anterior AMI was diagnosed when the following criteria were met: prolonged chest pain of more than 30-min duration, suggestive of AMI; diagnostic increase and decrease of the serum creatine kinase (CK); and evolution of serial ECG changes in 2 or more adjacent precordial leads (tall peaked T waves that become inverted on subsequent recordings, ST elevation, and shortening of the R wave or evolution of an abnormal Q wave on subsequent recordings). The records of 228 patients were examined. Eighty-one patients were excluded from the study because the admission ECG showed inverted T waves (29 patients, 5 of them with abnormal Q waves), ST segment depression in the involved leads without ST elevation (9 patients), abnormal Q waves (31 patients), and bundle-branch

^{*}From the Israel and Ione Massada Center for Heart Diseases (Drs. Birnbaum, Sclarovsky, Blum, and Mager), and the Epidemiology Unit (Dr. Gabbay), Beilinson Medical Center, Petah Tikva, Israel. Manuscript received July 13; revision accepted November 4. Reprint requests: Dr. Birnbaum, Cardiac Intensive Care Unit, Beilinson Medical Center, Petah-Tikva, Israel 49100



block (17 patients). The studied group consisted of 147 patients (111 men and 36 women) aged 37 to 86 years (mean, 64 years); 98 were treated with thrombolytic therapy (87 with streptokinase [750,000 to 3,000,000 units in 1 h] and 11 with recombinant tissue-type plasminogen activator [rtPa; 100 mg in 1 h]). All of the patients were treated with intravenous heparin (1,000 units/h) and isosorbide dinitrate (0.03 to 0.07 mg/min).

Electrocardiographic Evaluation

Definition of the initial ECG pattern (Fig 1) was as follows: (1) group A, tall symmetric abnormal T waves in the involved leads, without ST elevation or major changes in the terminal portion of the QRS; (2) group B, abnormal T waves and ST elevation (>0.1 mV) in 2 or more adjacent leads, without major changes in the morphology of the terminal portion of the QRS; and (3) group C, abnormal T waves and ST elevation (>0.1 mV), accompanied by distortion of the terminal portion of the QRS complex in 2 or more adjacent leads (emergence of the J point at a level above the lower half of the R wave or disappearance of the S wave in leads with an Rs configuration).¹²

All of the admission ECGs done in the emergency room and on arrival to the coronary care unit were evaluated by two cardiologists separately. In case of disagreement, a third examiner was consulted. The ST elevation was measured manually to the nearest 0.05 mV in each lead at 0.08 s from the J point. The number of leads with ST elevation (>1 mm in limb leads and >2 mm in precordial leads) was recorded.¹³ The patients were divided into three groups according to the definitions mentioned previously.

Clinical Evaluation

We studied the charts of the patients for clinical data such as age, sex, history of diabetes mellitus, hypertension, and angina pectoris, Killip functional class on admission, thrombolytic therapy, and death during the index admission. FIGURE 1. Initial ECG patterns. Examples of different patterns are demonstrated in three different patients with anterior AMI. Pattern A, tall symmetric abnormal T waves in involved leads (V_2 to V_3), without ST elevation or major changes in terminal portion of QRS complex; pattern B, abnormal T waves and ST elevation (>0.1 mV) in leads V_1 to V_3 , without major changes in terminal portion of QRS; and pattern C, abnormal T waves and ST elevation (>0.1 mV), accompanied by distortion of terminal portion of QRS complex (disappearance of S wave in leads V_1 to V_3 and emergence of J point at level above lower half of R wave in leads V_4 to V_9).

Statistical Analysis

The χ^2 test was applied to see if there is a difference in the inhospital mortality among the three groups of initial ECG patterns. We used the Spearman correlation coefficient to measure the strength of association between in-hospital mortality (1=death; 2=survival) and each of the following parameters: age; sex; Killip functional class on admission; thrombolytic therapy; number of leads with ST elevation; and the three initial ECG patterns.

A logistic regression model was fitted, using the "SAS Procedure Logistic for P.C.," to evaluate the prediction of in-hospital mortality for each subgroup of the parameters. Decisions concerning potential explanatory variables, to be included in the model, were based on the p value of the Wald χ^2 statistic. The dependent or response variable was in-hospital mortality (denoted for convenience by 1 = death and 2= survival). The potential prognostic factors to be included in the model were the initial ECG pattern (1 to 3), the number of leads with ST elevation, Killip functional class, age, and sex. The estimates of the logistic regression model were used to calculate the predicted probabilities of death in the different groups of initial ECG patterns.

RESULTS

One hundred forty-seven patients were enrolled in the study. There were 12, 77, and 58 patients included in initial ECG pattern groups A, B, and C, respectively. Ninety-nine patients received thrombolytic therapy: 3 (25 percent), 53 (69 percent), and 42 (72 percent) in groups A, B, and C, respectively. Nineteen patients died during hospitalization (in-hospital mortality of 13 percent). The age, sex, initial ECG pattern, time of death after admission, and the presumed cause of death are shown in Table 1. Most of the patients

Significance of Initial ECG in Acute Anterior Wall MI (Birnbaum et al)

Table	1-Data	From	19	Patients	Who	Died
	Duri	ıg Ho	spi	talization	1	

Age, Sex	Initial ECG Pattern	Time of Death after Admission	Cause of Death
73, F	В	12 d	After operation for VSD
86, F	В	8 h	Cardiogenic shock
80, M	С	10 h	Cardiogenic shock
69, F	С	25 h	Cardiogenic shock
63, M	С	30 h	Cardiogenic shock
70, F	С	8 h	Cardiogenic shock
75, M	С	5 h	Cardiogenic shock
			(after rescue PTCA*)
74, F	С	6 h	Cardiogenic shock
75, M	С	2 h	Cardiogenic shock
63, M	С	5 h	Cardiogenic shock
75, F	С	3 h	Arrhythmia
75, F	С	24 h	Cardiogenic shock
80, M	С	8 h	Cardiogenic shock
66, M	С	10 h	Cardiogenic shock
69, F	С	18 h	Cardiogenic shock
70, M	С	21 d	Reinfarction
68, F	С	13 d	Cardiogenic shock
67, M	С	2 h	Cardiogenic shock
81, F	С	2 d	Cardiogenic shock

*PTCA, Percutaneous transluminal coronary angioplasty.

who died did so during the first hours after admission. The cause of death was pump failure in 17 patients, one of them after reinfarction on the 21st day. One patient died after operation for a ventricular septal defect (VSD) on the 12th day, and one died because of ventricular fibrillation. In all of the patients, the ECG pattern was stable from arrival at the emergency room until the initiation of specific therapy (approximately 15 to 30 min later).

There was no significant difference between the mortality of patients with a previous history of diabetes mellitus, hypertension, and angina pectoris and patients without such a history. The mortality in 48 patients who did not receive thrombolytic therapy was 19 percent (9 patients). Ten of the 99 patients who received thrombolytic therapy died, but the difference is not significant ($\chi^2 = 2.15$; p = 0.14). The mortality in groups A, B, and C was 0, 3 percent (2 patients), and 29 percent (17 patients), respectively. The mortality among the patients who received thrombolytic therapy in groups A, B, and C was 0, 2 percent (1 patient), and 21 percent (9 patients), respectively. The relationship between mortality and the ECG patterns was

Tal	ble	e 2—Morta	dity b	y Initia	l ECG	Pattern*
-----	-----	-----------	--------	----------	-------	----------

ECG Pattern	Died	Survived	Total	Mortality, %
Group A	0	12	12	0
Group B	2	75	77	3
Group C	17	41	58	29
Total	19	128	147	13

 $\chi^2 = 22.91; p = 0.00.$

Table 3—Correlation Coefficients and Significance Between Several Parameters and Mortality*

Variable	Correlation Coefficient	Significance
Initial ECG pattern	-0.38	0.0001
Age	-0.29	0.0003
Sex	-0.25	0.0021
Number of leads	-0.24	0.0029
Killip class	-0.16	0.0571
Thrombolysis	+0.12	0.1447

*Death was defined as 1 and survival as 2. That is cause for negative value in most of correlations.

statistically significant (p=0) (Table 2).

Table 3 shows the correlation coefficient and the p value of each parameter and the in-hospital mortality. The initial ECG pattern was the most significant parameter (correlation coefficient = -0.38; p = 0.0001). Other variables (age, sex, number of leads with ST elevation, and Killip class at presentation) were also statistically significant.

In a preliminary step for prediction of in-hospital mortality, we included in the logistic regression model the following prognostic factors: age; sex; Killip functional class; number of leads with ST elevation; and the initial ECG pattern.

Neither the Killip class nor the number of leads with ST elevation was statistically significant (Table 4). When the initial ECG pattern was included in the model, the contribution of the other prognostic factors was very small, so in our final logistic regression model, the only explanatory variable included was the initial ECG pattern. The odds ratio between the initial ECG patterns B and A is 15.6. The odds ratio between the initial ECG patterns C and B is 11.6, while the odds ratio between patterns C and A comes up to 181 (Table 5). Thus, for a patient presenting with ECG pattern A, the predicted probability of death is 0.0016. For a patient with pattern B, the predicted probability of death is 0.025, and for a patient with pattern C, the probability is 0.29 (Table 6).

DISCUSSION

In this study, we attempted to correlate the different

Table 4—Estimated Parameters From Multivariate Logistic Regression With In-Hospital Mortality as Dependent Variable

Variable	Parameter Estimate (β)	Standard Error of β	p Value*
Intercept	- 16.1187	3.4766	0.0001
Initial ECG pattern	2.6623	0.8941	0.0029
Age	0.0652	3.6681	0.0555
Sex	1.5138	5.3838	0.0203
Number of leads	0.0219	0.0122	0.9121
Killip class	0.3357	1.1693	0.2795

p value was based on Wald χ^{} statistic.

Data	Variable			
	Intercept	ECG Pattern		
Parameter estimate (β)	-9.1768	2.766		
Standard error of B	2.1993	0.7632		
p value*	0.0001	0.0003		

 Table 5—Estimated Parameters from Logistic Regression With In-Hospital Mortality as

 Dependent Variable and Only ECG Pattern as Predictor Variable

*p value is based on Wald χ^2 statistic.

patterns of the ECG on admission in patients having evolving anterior AMI with the in-hospital mortality.

Previous Studies

Previous studies concerning the ability of the admission ECG to predict either infarct size or prognosis have concentrated on absolute ST segment measurements. Cohen et al¹⁴ found that the number of leads with ST elevation observed during angioplasty of the left coronary artery correlated with the angiographic estimate of the size of the ischemic hypocontractile zone. Aldrich et al⁸ found a formula relating the number of leads with ST elevation at the J point to the infarct size in anterior AMI, with the validity of the formula proven in a prospective study.¹⁵ Mauri et al,¹³ by assessing the patients enrolled in the Gruppo Italiano Per Lo Studio della Streptochimasi Nell'infarcto Miocardico (GISSI) trial, found that the mortality of patients with an anterior AMI increased from 8 percent in patients with ST elevation in 2 to 3 leads to 23.8 percent in those with 8 to 9 involved leads; however, Murray et al¹⁶ found only a weak correlation between the number of leads with ST elevation (measured by precordial 35-lead maps) and infarct size (estimated by imaging with^{99m}Tc-labeled stannous pyrophosphate [99mTc-PYP]). Yusuf et al¹¹ and Nielsen¹⁷ did not find any correlation between the number of leads with ST elevation and cumulative CK-MB¹¹ or mortality.17

Estimation of the magnitude of ST elevation has been correlated with mortality. Nielsen¹⁷ found that the death rate of patients with "major ST elevation" was 23 percent, while in patients with "minor ST elevation," the mortality was 6 percent (p<0.005). Marik et al¹⁸ stated that the mortality in 42 patients with maximal ST elevation of 0 to 1 mm in a single lead was 0, while this value rose to 54 percent in 70 patients with an ST elevation of 5 mm or more in a

 Table 6—Estimated Probabilities* of Death in Relation to ECG Pattern

ECG	Predicted Probability	95% Confidence
Pattern	of Mortality	Limits
Group A	0.0016	0.0001-0.027
Group B	0.025	0.006-0.95
Group C	0.29	0.19-0.42

*Probabilities were calculated based on logistic regression estimates.

single lead.

The sum of ST elevations, measured by 35-lead precordial mapping^{11,19,20} or in a standard 12-lead ECG^{7,21,22} has been correlated with infarct size and also with mortality;²² however, other investigators did not find such a correlation.^{4,16,23,24} In a recent study,²⁵ only a weak correlation between the sum of ST elevations and the myocardial area at risk assessed by ^{99m}Tc-sestamibi imaging, was found.

Present Study

We have studied the initial ECG patterns (the terminal portion of the QRS, the ST segment, and the T wave) in patients presenting in the early stages of an evolving anterior AMI, before inversion of the T wave and before appearance of an abnormal Q wave. We found that it is possible to recognize three different patterns of the admission ECG (Fig 1).12 The first pattern is characterized by tall peaked abnormal T wave in the involved leads, without ST elevation or major changes in the terminal portion of the ORS. Recently, we described a subgroup of patients with anterior AMI compatible with this pattern.²⁶ In this subgroup, there is a high incidence of total or near total obstruction of the left anterior descending coronary artery and retrograde filling of the artery by collateral vessels. In these patients the prognosis is relatively good. The second pattern is recognized by abnormal T waves and ST elevation (>0.1 mV) in 2 or more leads, without major changes in the morphology of the terminal portion of the QRS. The third pattern is characterized by abnormal T waves and ST elevation accompanied by distortion of the terminal portion of the QRS. Recently, changes in the terminal portion of the QRS were described early in the course of AMI,²⁷ but the prognostic significance of these changes have never been previously studied.

We found this classification to be simple and highly reproducible. The ECG pattern was stable in all of our patients from arrival in the emergency room until the initiation of reperfusion therapy.

The in-hospital mortality in our patients was 13 percent and is comparable with other series of patients with first anterior AMIs.^{13,18,28-30} In-hospital mortality in groups A, B, and C was 0, 3 percent and 29 percent, respectively. The correlation between the initial ECG pattern and mortality is highly significant (correlation

coefficient = 0.38; p = 0.0001). The number of leads with ST elevation also had a good correlation with inhospital mortality, but the correlation coefficient was less than that of the ECG pattern (Table 3).

Mortality in Relation to Clinical Data

Our data demonstrate that age and female sex are associated with increased mortality (Table 3). This is in agreement with other studies.^{18,29-36}

Our data do not support an association between a history of previous angina pectoris, hypertension, and diabetes mellitus and mortality. Evidence of heart failure or hypotension, as classified by Killip and Kimball,37 has a strong association with mortality.^{18,20,30,33,38-40} Our data are in concordance with those studies (Table 3). The mortality in 99 patients who received thrombolytic therapy was 10 percent versus 19 percent in 48 patients who were treated conservatively. Even though the two groups are not comparable because of selection bias (there was no randomization between the two groups; those patients who survived cardiopulmonary resuscitation and most of the patients with no ST elevation were not treated by thrombolysis), the relationship between thrombolysis and in-hospital mortality was not statistically significant ($\chi^2 = 2.15$; p = 0.14).

Multivariant Regression Analysis

Among the different parameters that were examined, the initial ECG pattern seems to be the one with the strongest association with mortality. Correlations of in-hospital mortality with age, sex, Killip functional class on admission, and the number of leads with ST elevation were also significant. When we separated the effect of the ECG pattern on the inhospital mortality from the additional effects produced by age and sex, the odds ratio was almost unchanged.

The ECG is thus a simple and an immediately available noninvasive tool that can help the clinician in the diagnosis and risk stratification of patients with AMI, shortly after admission. By using simple ECG criteria, such as the initial ECG pattern, we were able to divide patients shortly after admission into three subgroups that differ significantly in the risk of death, from negligible mortality to almost 30 percent.

Electrophysiologic Explanation of Initial Pattern

It is accepted that ST elevation during myocardial ischemia is caused by a "current of injury" between the normal region and the ischemic zone.^{41,42} ST elevation is a manifestation of segmental damage. Good correlation was found between regional wall motion abnormalities and ST elevation during coronary angioplasty¹⁴ and between the grade of collateral flow during transient coronary occlusion during angioplasty and the sum of ST elevation.^{43,44} Distortion of the QRS complex during myocardial ischemia is caused by alterations in the conduction velocity of the activation wave in the Purkinje fibers as it travels through the ischemic region.⁴⁵ The delayed conduction decreases the degree of cancellation and, by so doing, increases the amplitude of the R wave and causes disappearance of the S wave. The Purkinje system is less sensitive to ischemia than the contracting myocardial cells.^{46,47} For an alteration in the terminal portion of the QRS to occur, there must be a more severe degree of ischemia that would injure the Purkinje system.⁴⁵

From these data, it can be concluded that in patients in group A, probably because of a good collateral circulation, the ischemia is not severe enough to cause an "injury current." The damage is minimal, and the prognosis is good.³⁶ One may postulate that in group B the ischemia is more severe, causing an "injury current," but the Purkinje fibers are not injured enough to cause major changes in the terminal portion of the QRS. Group C is characterized by ST elevation, accompanied by alteration of the terminal portion of the QRS. This may be because of a lack of collateral circulation, causing an even more severe degree of myocardial ischemia that affects the Purkinje system, and this may explain the increased mortality in group C.

Limitations

This is a historical prospective study of patients with a confirmed first anterior AMI. There was no randomization between patients who received thrombolytic therapy and those who did not. There is a need for a larger prospective study to verify our results and to examine, by randomized study, the role of more aggressive reperfusion measures in patients with AMI presenting with initial ECG pattern C.

Clinical Implication

It is most important to estimate the prognosis of a patient with an evolving AMI shortly after admission, because a potential poor prognosis would justify more aggressive reperfusion measures such as "primary" or "salvage" angioplasty or emergency coronary artery bypass grafting following angiography, especially in cases where thrombolytic therapy is contraindicated. By using the admission ECG, we were able to differentiate between three subgroups of patients with a first anterior AMI. The in-hospital mortality differs significantly among these subgroups. Categorizing the admission ECG according to the three patterns may aid the clinician in risk stratification before other data such as left ventricular ejection fraction and serial CK-MB serum levels are available.

References

1 Bern GB, Wasserman AG, Ross AM. The electrocardiogram in

patients undergoing thrombolysis for myocardial infarction. Circulation 1987; 76:11-18-24

- 2 Maroko PR, Libby P, Covell JW, Sobel BE, Ross JJ, Braunwald E. Precordial S-T segment elevation mapping: an atraumatic method for assessing alternations in the extent of myocardial ischemic injury. Am J Cardiol 1972; 29:223-30
- 3 Muller JE, Maroko PR, Braunwald E. Evaluation of precordial electrocardiographic mapping as a means of assessing changes in myocardial ischemic injury. Circulation 1975; 52:16-27
- 4 Madias JE, Venkataraman K, Hood WB Jr. Precordial STsegment mapping: 1. clinical studies in the coronary care unit. Circulation 1975; 52:799-809
- 5 Muller JE, Maroko PR, Braunwald E. Precordial electrocardiographic mapping: a technique to assess the efficacy of interventions designed to limit infarct size. Circulation 1978; 57:1-18
- 6 Vermeer F, Simoons ML, Bar FW, Tijssen JGP, Van Domburg RT, Serruys PW, et al. Which patients benefit most from early thrombolytic therapy with intracoronary streptokinase? Circulation 1986; 74:1379-89
- 7 Bar FW, Vermeer F, de Zwaan C, Ramental M, Braat S, Simoons ML, et al. Value of admission electrocardiogram in predicting outcome of thrombolytic therapy in acute myocardial infarction: a randomized trial conducted by the Netherlands Interuniversity Cardiology Institute. Am J Cardiol 1987; 59:6-13
- 8 Aldrich HR, Wagner NB, Boswick J, Corsa AT, Jones MG, Grande P, et al. Use of initial ST-segment deviation for prediction of final electrocardiographic size of acute myocardial infarcts. An. J Cardiol 1988; 61:749-53
- 9 Krucoff MW, Green CE, Satler LF, Miller FC, Pallas RS, Kent KM. et al. Noninvasive detection of coronary artery patency using continuous ST-segment monitoring. Am J Cardiol 1986; 57:916-22
- 10 Clemmensen P, Ohman M, Sevilla DC, Peck S, Wagner NB, Quigley PS, et al. Changes in standard electrocardiographic STsegment elevation predictive of successful reperfusion in acute myocardial infarction. Am J Cardiol 1990; 66:1407-11
- 11 Yusuf S, Lopez R, Maddison A, Maw P, Ray N, McMillan S, et al. Value of electrocardiogram in predicting and estimating infarct size in man. Br Heart J 1979; 42:286-93
- 12 Sclarovsky S, Strasberg B, Lewin RF, Arditi A, Klainman E, Agmon J. Effects of isosorbide dinitrate intravenously in high dose over a short period in anterior acute myocardial infarction. Am J Cardiol 1988; 61:78E-80E
- 13 Mauri F, Gasparini M, Barbonaglia L, Santoro E, Franzosi MG, Tognoni G, et al. Prognostic significance of the extent of myocardial injury in acute myocardial infarction treated by streptokinase (the GISSI trial). Am J Cardiol 1989; 63:1291-95
- 14 Cohen M, Scharpf SJ, Rentrop KP. Prospective analysis of electrocardiographic variables as markers for extent and location of acute wall motion abnormalities observed during coronary angioplasty in human subjects. J Am Coll Cardiol 1987; 10:17-24
- 15 Clemmensen P, Grande P, Hindman NB, Aldrich H, Wagner GS. Initial ST-segment deviation can predict final electrocardiographic myocardial infarct size. J Am Coll Cardiol 1988; 11:68A
- 16 Murray RG, Peshock RM, Parkey RW, Bonte FJ, Willerson JT, Blomqvist CG. ST isopotential precordial surface maps in patients with acute myocardial infarction. J Electrocardiography 1979; 12:55-64
- 17 Nielsen BL. ST-segment elevation in acute myocardial infarction: prognostic importance. Circulation 1973; 48:338-45
- 18 Marik PE, Lipman J, Eidelman IJ, Erskine PJ. Clinical prediction of early death in acute myocardial infarction: a prospective study of 233 patients. S Afr Med J 1990; 77:179-82
- 19 Selwyn AF, Ogunro EA, Shillingford JP. Natural history and evaluation of ST segment changes and MB CK release in acute myocardial infarction. Br Heart J 1977; 39:988-94

- 20 Henning H, Hardarson T, Francis G, O'rourke RA, Ryan W, Ross J. Approach to the estimation of myocardial infarct size by analysis of precordial S-T segment and R wave maps. Am J Cardiol 1978; 41:1-8
- 21 Hackworthy RA, Vogel MB, Harris PJ. Influence of infarct artery patency on the relation between initial ST segment elevation and final infarct size. Br Heart J 1986; 56:222-25
- 22 Willems JL, Willems RJ, Willems GM, Arnold AER, Van de Werf F, Verstraete M. Significance of initial ST segment elevation and depression for the management of thrombolytic therapy in acute myocardial infarction. Circulation 1990; 82:1147-58
- 23 Norris RM, Barratt-Boyes C, Heng MK, Singh BN. Failure of ST segment elevation to predict severity of acute myocardial infarction. Br Heart J 1976; 38:85-92
- 24 Thompson PL, Katavatis V. Acute myocardial infarction: evaluation of praecordial ST segment mapping. Br Heart J 1976; 38:1020-24
- 25 Clements IP, Kaufmann UP, Bailey KR, Pellikka PA, Behrenbeck T, Gibbons RJ. Electrocardiographic prediction of myocardial area at risk. Mayo Clin Proc 1991; 66:985-90
- 26 Sagie A, Sclarovsky S, Strasberg B, Kracoff O, Rechavia E, Bassevich R, et al. Acute anterior wall myocardial infarction presenting with positive T waves and without ST segment shift: electrocardiographic features and angiographic correlation. Chest 1989; 95:1211-15
- 27 Barnhill JE, Tendera M, Cade H, Campbell WB, Smith RF. Depolarization changes early in the course of myocardial infarction: significance of changes in the terminal portion of the QRS complex. J Am Coll Cardiol 1989; 14:143-49
- 28 Stone PH, Raabe DS, Jaffe AS, Gustafson N, Muller JE, Turi ZG, et al. Prognostic significance of location and type of myocardial infarction: independent adverse outcome associated with anterior location. J Am Coll Cardiol 1988; 11:453-63
- 29 Sahasakul Y, Chaithiraphan S, Panchavinnin P, Jootar P, Thongtang V, Srivanasont N, et al. Multivariate analysis in the prediction of death in hospital after acute myocardial infarction. Br Heart J 1990; 64:182-85
- 30 Kitchin AH, Pocock SJ. Prognosis of patients with acute myocardial infarction admitted to a coronary care unit: survival in hospital. Br Heart J 1977; 39:1163-66
- 31 Goldberg RJ, Gore JM, Gurwitz JH, Alpert JS, Brady P, Strohsnitter W, et al. The impact of age on the incidence and prognosis of initial acute myocardial infarction: the Worcester heart attack study. Am Heart J 1989; 117:543-49
- 32 Dubois C, Pie'rard LA, Albert A, Smeets JP, Demoulin JC, Boland J, et al. Short-term risk stratification at admission based on simple clinical data in acute myocardial infarction. Am J Cardiol 1988; 61:216-19
- 33 Hillis LD, Forman S, Braunwald E. Risk stratification before thrombolytic therapy in patients with acute myocardial infarction. J Am Coll Cardiol 1990; 16:313-15
- 34 Puletti M, Sunseri L, Curione M, Erba SM, Borgia C. Acute myocardial infarction: sex-related differences in prognosis. Am Heart J 1984; 108:63-6
- 35 Tofler GH, Stone PH, Muller JE, Willich SN, Davis VG, Poole WK, et al. Effect of gender and race on prognosis for women, particularly black women. J Am Coll Cardiol 1987; 9:473-82
- 36 Greenland P, Reicher-Reiss H, Coldbourt U, Behar S. Inhospital and 1-year mortality in 1524 women after myocardial infarction: comparison with 4315 men. Circulation 1991; 83:484-91
- 37 Killip T, Kimball JT. Treatment of myocardial infarction in a coronary care unit: a two-year experience with 250 patients. Am J Cardiol 1967; 20:457-64
- 38 Norris RM, Brandt PWT, Caughey DE, Lee AJ, Scott PJ. A new coronary prognostic index. Lancet 1969; 1:274-78

Significance of Initial ECG in Acute Anterior Wall MI (Birnbaum et al)

- 39 Pie'rard LA, Albert A, Chapelle JP, Carlier J, Kulbertus HE. Relative prognostic value of clinical, biochemical, echocardiographic and haemodynamic variables in predicting in-hospital and one-year cardiac mortality after acute myocardial infarction. Eur Heart J 1989; 10:24-31
- 40 Pie'rard LA, Chapelle JP, Albert A, Dubois C, Kulbertus HE. Characteristics associated with early (<3 month) versus late (>3 month to <3 years) mortality after acute myocardial infarction. Am J Cardiol 1989; 64:315-18
- 41 Braunwald E, Maroko PR. ST-segment mapping: realistic and unrealistic expectations. Circulation 1976; 54:529-32
- 42 Fozzard HA, DasCupta DS. ST-segment potentials and mapping: theory and experiments. Circulation 1976; 54:533-37
- 43 Cohen M, Rentrop KP. Limitation of myocardial ischemia by collateral circulation during sudden controlled coronary artery

occlusion in human subjects: a prospective study. Circulation 1986; 74:469-76

- 44 Mizuno K, Horiuchi K, Matui H, Miyamoto A, Arakawa K, Shibuya T, et al. Role of coronary collateral vessels during transient coronary occlusion during angioplasty assessed by hemodynamic, electrocardiographic and metabolic changes. J Am Coll Cardiol 1988; 12:624-28
- 45 Holland RP, Brooks H. The QRS complex during myocardial ischemia: an experimental analysis in the porcine heart. J Clin Invest 1976; 57:541-50
- 46 DeHaan RL. Differentiation of the atrioventricular conducting system of the heart. Circulation 1961; 24:458-70
- 47 Schiebler TH, Stark M, Caesar R. Die Stoffwechselsitation des Reizleitungssystems. Klin Wochenschr 1956; 34:181-83



Prognostic significance of the initial electrocardiographic pattern in a first acute anterior wall myocardial infarction.

Y Birnbaum, S Sclarovsky, A Blum, A Mager and U Gabbay Chest 1993;103; 1681-1687 DOI 10.1378/chest.103.6.1681

This information is current as of July 13, 2011

Updated Information & Services

Updated Information and services can be found at: http://chestjournal.chestpubs.org/content/103/6/1681

Cited Bys

This article has been cited by 2 HighWire-hosted articles: http://chestjournal.chestpubs.org/content/103/6/1681#related-urls

Permissions & Licensing

Information about reproducing this article in parts (figures, tables) or in its entirety can be found online at: http://www.chestpubs.org/site/misc/reprints.xhtml

Reprints

Information about ordering reprints can be found online: http://www.chestpubs.org/site/misc/reprints.xhtml

Citation Alerts

Receive free e-mail alerts when new articles cite this article. To sign up, select the "Services" link to the right of the online article.

Images in PowerPoint format

Figures that appear in *CHEST* articles can be downloaded for teaching purposes in PowerPoint slide format. See any online figure for directions.

