ST-Segment Elevation in Acute Myocardial Infarction

Prognostic Importance

By BENT LYAGER NIELSEN, M.D.

SUMMARY
Four hundred and four patients who were admitted to the hospital with their first myocardial infarction within 24 hours of the onset of symptoms were studied. In 315 patients the ECG findings in 12 standards leads (I, II, aVF, aVR, aVL, V₁–V₆) showed the infarction to be in the anterior wall, the inferior wall, or in multiple areas. The patients in each of these three categories were further subdivided into two groups according to the magnitude of the ST-elevation; those with major ST-elevation and those with minor ST-elevation. Occurrence of the risk factors of diabetes, hypertension, hyperlipidemias, age, and complicating chronic pulmonary and renal diseases were comparable, but since women predominated in one group (anterior wall infarction/minor ST-elevation) the clinical course of the disease in men and women was studied separately. The patients with major ST-elevation had a more unfavorable course: cardiac arrest (P < 0.0005), congestive heart failure (P < 0.0005), death (P < 0.001), atrial fibrillation (P < 0.005), cardiogenic shock (P < 0.005), atrioventricular (A-V) block 2°–3° (P < 0.01), and ventricular extrasystoles and ventricular tachycardia (P < 0.025) all occurred with significant frequency. The magnitude of ST-elevation thus gives valuable prognostic information, useful in selecting patients for prolonged monitoring.

Additional Indexing Words:
Cardiac arrest Congestive heart failure Death in acute myocardial infarction
Atrioventricular block Atrial fibrillation Cardiogenic shock Ventricular extrasystoles
Ventricular tachycardia Thromboembolic complications Pneumonia
Paroxysmal supraventricular tachycardia Risk factors Myocardial ischemia
Serum enzymes Site of infarction Ventricular arrhythmias Mortality rates

PARAMETERS related to the size of infarction are often used when evaluating patients with acute myocardial infarction. A reasonably accurate clinical correlation between the maximum serum enzyme elevation and the extent of the infarction determined at post mortem has been established in earlier studies.1 The correlation between the mortality rate and the serum glutamic oxaloacetic transaminase (SGOT), and lactic dehydrogenase (LDH) is highly significant.2

While serum enzyme values can be used as a rough guide to the extent of the infarction,3, 4 animal experiments5 6 and exercise and arteriographic studies on patients7 have shown that deviation of the ST-segment reflects myocardial ischemia, and that ST-elevation indicates a more severe degree of ischemia than ST-depression.6 7 8

Both the duration and magnitude of the ST-elevation and the number of leads recording ST-elevation have been used for diagnostic and prognostic evaluation. An early reversal of the ST-elevation in standard leads has thus been read as an expression of effective treatment of patients with acute myocardial infarction (AMI).9 With surface mapping of the ST-segment deviations using multiple surface electrodes, it has been possible to demonstrate an abnormal pattern in patients with acute myocardial infarction whose standard leads are abnormal, and in some patients with myocardial infarction when the standard electrocardiogram is normal.10 11 Maroko et al. have shown that the sum of ST-segment elevation in multiple precordial leads (Σ S-T) can be used as an index of tissue injury and the number of sites with ST-elevation (NS-T) as an expression of the extent of myocardial ischemic injury, and that both parameters can be changed by pharmacologic and hemodynamic intervention.12 13 14 If the magnitude of ST-eleva-

338 Circulation, Volume XLVIII, August 1973
tion in standard leads had similar prognostic significance, an important method of predicting the course of myocardial infarction could be introduced into coronary care units with no special demands on time, equipment, or expense. This study asked the question: Of what prognostic importance is the magnitude of the ST-elevation in standard leads?

Material and Methods

Four hundred and four patients admitted to the Coronary Care Unit of the University Hospital of Odense, Denmark, were studied. All of these patients met these criteria: 1) first myocardial infarction; 2) admission within 24 hours after the onset of symptoms; 3) correct calibration of the ECG (1 mV = 10 mm); and 4) at least one standard ECG with 12 leads and determination of at least one series of serum enzymes (SGOT, LDH, and creatine phosphokinase [CPK]) before death or cardiac arrest. Patients with cardiac arrest prior to the first ECG were excluded. Continuous ECG monitoring was carried out on an average of four to five days, and in the first week an ECG was taken daily using the following leads: I, II, III, aVL, aVF, V1–6, after which an ECG was usually recorded two to three times per week or as required.

The ST-elevation was measured on the ECG using the T-P-segment as the isoelectric line. The deviation was measured in mm to the nearest 0.5 mm at 0.06 sec after the nadir of the S wave, when this was present. In most patients without an S wave, the deviation was measured 0.06 sec after the nadir of the QS-complex, or from the Q wave. In a very few patients without well-defined S, QS, or Q waves, the R wave was used as the starting point. Under this definition ST-elevation in patients with broad QRS complexes (0.12 sec or more) and bundle branch block (BBB) could not be measured. Patients with such findings were not studied further and are incorporated in the group of patients in whom the site of the infarction is uncertain.

Ventricular tachycardia is defined as a run of six or more ventricular ectopic beats in sequence at a rate greater than 100 beats/min. Internationally accepted definitions were used for the diagnosis of arrhythmias in all other cases. E-leads have been recorded in cases where the arrhythmia was difficult to classify.

Thromboembolic complications include deep lower extremity phlebitis, pulmonary embolism, cerebral embolism, and emboli in the extremities, but excluded splenic or renal emboli, which often are difficult to diagnose with certainty. Cardiogenic shock is defined as a systolic blood pressure (BP) of less than 90 mm Hg in patients with clinical symptoms of shock. All these patients had either pulmonary congestion/pulmonary edema (31 patients) or pronounced cardiomegaly (three patients).

Results

Grouping of Patients

Anterior wall infarction was present in 147 patients, inferior wall infarction in 103, and multiple infarctions in 65. Three hundred and fifteen patients out of 404 were included in these 3 groups. Of the remaining 89 patients, ten presumably had a strictly posterior infarction. In 79 patients the site of infarction was uncertain. All patients with broadened QRS and/or BBB were included in this group. No further analysis was made of these 89 patients.

In the group of patients with anterior wall infarction, 79 patients had major ST-elevation (≥ 5.0 mm) in one or more leads (I, aVL, V1–6), while the remaining 68 patients had minor ST-elevation. Fifty-four patients in the group with inferior wall infarction had major ST-elevation (≥ 2.0 mm) in one or more leads (II, III, aVF); the remaining 49 had minor ST-elevation. In the group with multiple infarctions, 25 patients had ST-elevation ≥ 3.0 mm in one or more leads (I, aVL, V1–6) and ST-elevation of ≥ 2.0 mm (major elevation) in leads II, III, and aVF. The remaining 40 had minor ST-elevation.

Thus in the total group, 158 patients had major ST-elevation and 157 patients, minor ST-elevation. Definitions for major and minor ST-elevation were selected which would result in groups of almost equal size.

Comparability

Risk factors (diabetes mellitus, hypertension, and hyperlipidemia), complicating diseases (chronic pulmonary and renal diseases) and patients 60 years and older had the same frequency when the patients were grouped according to the location of the infarct. The distribution of these factors remained unchanged when these groups were further subdivided into those with major and minor ST-elevations.

A significant predominance of women was demonstrated in the group with anterior wall infarction and minor ST-elevation (χ² = 5.28, P < 0.025), which is also reflected in the composition of the total group with minor ST-elevation (χ² = 4.386, P < 0.05). As a result of this difference men and women were studied separately.

Occurrence of Arrhythmias, Complications, and Death

Atrial fibrillation, A-V block of 2° to 3°, ventricular extrasystoles, ventricular tachycardia, cardiac arrest, shock, and death occurred with significantly greater frequency in patients with major ST-elevation than in patients with minor ST-elevation (table 1).
Table 1

Comparison of ST-elevation and Occurrence of Arrhythmias, Cardiac Arrest, Shock, and Death in Patients Suffering Their First Acute Myocardial Infarction

<table>
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<tr>
<th></th>
<th>No. of patients</th>
<th>Atrial fibrillation</th>
<th>A-V block</th>
<th>Ventricular extrasystoles</th>
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Frequency of other complications was also studied. Supraventricular tachycardia (atrial tachycardia, paroxysmal atrial tachycardia [PAT] with block and nodal tachycardia) took place with the same frequency in patients with minor ST-elevation as in those with major elevation of this segment (6%). More patients with multiple infarctions (15%) experienced supraventricular tachycardia, but those with inferior wall infarction had this complication only rarely (1%). Congestive heart failure was demonstrated in 49% of all patients with minor ST-elevation but in 70% of the patients with major ST-elevation ($\chi^2 = 13.0107$, $P < 0.0005$). The correlation between congestive heart failure and major ST-elevation is statistically significant in patients with anterior wall infarction ($\chi^2 = 11.8255$, $P < 0.001$), and particularly in male patients ($\chi^2 = 14.8251$, $P < 0.0005$).

Ten percent of all patients developed thromboembolic complications, with a comparable rate for those in minor and in major ST-elevation groups. The distribution did not follow the same pattern as congestive heart failure. Pneumonia was diagnosed in 24% of all patients, most frequently in the groups with a high incidence of congestive heart failure, a fact which would suggest that many of these cases were hypostatic pneumonia. Comparing the subgroups within the major breakdown according to ST-elevation, the study found that major ST-elevation was significantly related to a higher frequency of development of pneumonia only in the group of patients with anterior wall infarction ($\chi^2 = 5.1229$, $P < 0.025$).

The Number of Leads with ST-Elevation and the Mortality Rate

In order to ascertain whether the magnitude of the ST-elevation was a better parameter for evaluating the risk of death than the number of leads in which ST-elevation could be shown, the mortality rate was analyzed by the number of leads with ST-elevation without regard to the magnitude of ST-elevation. Of 147 patients with anterior wall infarction, 82 had ST-elevation in five or fewer leads and 12 died (15%). The same mortality rate was demonstrated in the remaining 65 patients, who had ST-elevation in more than five leads, as 10 patients died (15%). Only 38 of the patients with inferior wall infarction had ST-elevation in one or more leads, and four (11%) died. The remaining 65 had ST-elevation in three leads. Of these, eight died (13%). Forty-five of the patients with multiple infarctions had ST-elevation in less than five leads. Of these, eight died (18%), as compared to three (15%) of 20 patients with ST-elevation in five leads or more.

Of the total, 165 patients had ST-elevation in “a few leads,” and 24 died (14.5%). A total of 150 patients had ST-elevation in “many leads,” and of these, 21 died (14.0%). As can be seen, the magnitude of the ST-elevation, and not the number of leads with ST-elevation, is a helpful predictive tool.

Discussion

Why ST-elevation occurs is uncertain. Some have suggested that the true shift in the ST-segment is a manifestation of both earlier repolarization of myocardial cells in the injured area and a current of injury which deviates the TQ-segment.5, 11

Recordings in epicardial leads have been used to define the boundaries of experimentally-induced myocardial infarction in animals10, 16 and to study the alterations in the area of ischemic tissue.17 Electrograms recorded from the surface of the heart usually cannot be taken without considerable risk to the patient. However, Maroko et al.12, 13, 14 showed that a noninvasive technique with multiple surface electrodes provides the same information in animals as insertion of epicardial electrodes and that the sum of ST-elevation in precordial leads ($\Sigma S-T$) is an index of tissue injury and the number of leads in which ST-elevation exceeds 0.1 mV (NS-T) reflects the extent of myocardial ischemic injury.

The duration of the ST-elevation has not been the subject of further analysis here, although previous studies have suggested that this parameter is of clinical value.9 Maroko et al. have used precordial ST-segment elevation mapping with 35 surface electrodes in 19 patients with acute myocardial infarction. In three patients in whom ventricular fibrillation, arterial hypotension, and further ischemic pain occurred, $\Sigma S-T$, and NS-T increased, whereas in one patient propranolol decreased the same parameters. The study suggests that this atraumatic method enables evaluation to be made of acute changes of ischemic injury caused by the hemodynamics of pharmacological intervention, which alter myocardial oxygen consumption or coronary perfusion.14

The importance of the number of leads recording ST-elevation has not been discussed in detail here either. In a previous investigation the magnitude of ST-elevation and the number of leads in which ST-elevation was seen were significantly related. A pronounced ST-elevation usually meant that there was ST-elevation in many leads (unpublished data). The number of leads with ST-elevation is not
helpful in determining the risk of death, as stated above.

There are some good reasons to ascribe little clinical prognostic value to the magnitude of the ST-elevation in 12 standard leads in daily recordings, and these factors should be discussed in the light of our findings of the value of this measure. The leads do not cover the posterior wall of the heart and some parts of the septum and lateral wall, and the number of leads is so small that it is possible that the area with the most intense ischemia is missed by this method. In addition, if continuous monitoring of the ECG had been followed, ST-elevations more pronounced than those found in daily ECG recordings might have been detected. Even though patients with strictly posterior infarction (no cases of broadened QRS or BBB) were excluded, it must be presumed that in a number of cases that were included submaximal ST-elevation was registered. For these reasons—and because myocardial ischemia, reflected electrocardiographically as ST-elevation—has an adverse affect on ventricular performance,18, 19, 20, 21 it must be presumed that continuous monitoring and recordings taken from multiple electrodes would have demonstrated that the ECGs of many patients in our study who had an unfavorable clinical course, classified here as minor ST-elevations, in reality ought to have been included in the major ST-elevation groups.

A number of patients included in the group with major ST-elevation could have been included on the basis of only brief periods of pronounced ST-elevation, even though for most of the time only a slight elevation was noted.22 Patients were included in the group, major ST-elevation, regardless of whether the episode of ischemia recorded was brief, caused by the natural course of the disease, or brought about by the treatment.

Drugs did not greatly affect division of patients into groups with minor and major ST-elevation. The study included only patients with their first myocardial infarction and the majority of these patients had not been under treatment prior to admission. The maximum ST-elevation in the majority of cases was registered on the first ECG, i.e., before treatment was commenced (unpublished data).

Even though the study demonstrates that many complications occur more frequently in patients with major ST-elevation than in patients with minor ST-elevation, many factors other than myocardial ischemia—for example, the site of the infarction and the treatment given—may have brought on the complication. These additional possible contributing factors are discussed here in relation to each complication.

**Atrial Fibrillation and Paroxysmal Supraventricular Tachycardia**

The cause of atrial fibrillation in patients with acute myocardial infarction, often in paroxysms, is unknown, but it is generally considered that there are multiple etiological factors, partly primary (diseases of the sinus node or atrial muscle, distention of right atrium, metabolic disturbances), and partly precipitating (toxic, hypoxic, neurogenic, mechanical).23 In addition, atrial fibrillation/flutter occurs most often in patients with occlusion of the right coronary artery, which in the majority of patients supplies both the sinus node and A-V node.24

This study confirms that atrial fibrillation/flutter occurs most frequently in patients with occlusion of the right coronary artery (patients with inferior wall infarction and multiple infarctions), but this type of arrhythmia is not uncommon in patients with anterior wall infarction. Irrespective of the site of the infarction atrial fibrillation is seen most frequently in patients with major ST-elevation, most significantly in patients with inferior wall infarction. A pronounced localized myocardial ischemia therefore appears to be among the factors that can give rise to atrial fibrillation.

James25 found in a series including 11 necropsied patients with myocardial infarction and atrial arrhythmias (including atrial tachycardia) that all had a coronary occlusion proximal to the origin of the sinus node artery and there was infarction of the sinus node.

Our results, however, do not support the assumption that atrial fibrillation/flutter and paroxysmal supraventricular tachycardia (atrial tachycardia, atrial tachycardia with block, nodal tachycardia) have the same etiology. Supraventricular tachycardia is demonstrated significantly more rarely in patients with inferior wall infarction than in patients with anterior wall infarction ($X^2 = 4.186$, $P < 0.05$) and those with multiple infarctions, and in all the groups studied it is clear that the magnitude of the ST-elevation has no influence on the incidence. The frequency of paroxysmal supraventricular tachycardia is greatest in patients with multiple infarctions.

*Circulation, Volume XLVIII, August 1973*
A-V Block

A-V block, like atrial fibrillation, occurs most frequently in patients with involvement of the right coronary artery, and this is confirmed by our results.

It has been suggested that the cause of A-V block in patients with inferior wall infarction is reversible ischemia in the A-V node and bundle, while A-V block in patients with anterior wall infarction is caused by total or subtotal destruction of both bundle branches, with little or no involvement of the A-V node and the main bundle. From this one would expect that patients with inferior wall infarction would show A-V block most often combined with pronounced ST-elevation (caused by ischemia); that patients with multiple infarctions and presumed involvement of several coronary arteries would show both mechanisms (ischemia and destruction of both bundle branches); and that the patients with anterior wall infarction would not demonstrate any connection between the ST-elevation and the occurrence of A-V block.

This presumption is found to be correct. In patients with inferior wall infarction A-V block is demonstrated more than ten times as frequently in patients with pronounced ST-elevation as in patients with minor ST-elevation, and in patients with multiple infarctions A-V block is found to be non-significantly increased in patients with major ST-elevation over those with minor ST-elevation. In patients with anterior wall infarction A-V block occurs with no greater frequency in patients with major ST-elevation as in those with minor ST-elevation.

Ventricular Arrhythmias and Cardiac Arrest

Ventricular extrasystoles and ventricular tachycardia occur relatively frequently in patients with minor ST-elevation, but the risk of developing these arrhythmias appears to increase with increasing ST-elevation.

Patients with cardiac arrest (ventricular fibrillation, asystolia/bradycardia, or primary pump failure) during admission are included in the survey. Terminal rhythm disturbances, which did not require treatment, are not included. Only the number of patients and not the number of cases of cardiac arrest are given here.

If the magnitude of the ST-elevation is of any practical prognostic importance, for example as a guide to selecting patients for prolonged monitoring, then the frequency of cardiac arrest in patients with major ST-elevation is one of the more important correlations and must be considerably higher than in patients with minor ST-elevation. Cardiac arrest is one of the most feared complications and it often occurs after the patient has been taken off continuous ECG monitoring. It can be seen from table 1 that patients with major ST-elevation are subject to cardiac arrest two to five times more often than patients with minor ST-elevation. The difference is statistically significant in patients with anterior wall infarction, inferior wall infarction, in men, and in the total group.

Cardiogenic Shock

A recent quantitative study of infarcted myocardium in cardiogenic shock by Harnarayan shows that extensive myocardial injury, predominantly affecting the left ventricle and septum, accompanies cardiogenic shock, and that the mean total area of ventricular infarction is > 40%.

In our own study the highest incidence of shock is seen in patients with multiple infarctions (19%), while 9% of the patients in the other groups developed shock. This supports to some degree the presumed correlation between shock and the amount of myocardial tissue affected by the infarction.

Shock is found significantly more frequently in patients with major ST-elevation than in those with minor ST-elevation, and in patients with anterior wall infarction even 15 times more frequently. A possible explanation may be that myocardial ischemia is aggravated by hypotension and the accompanying systemic hypoxia. In addition, these critically ill patients are often given a drug which must be presumed to increase the magnitude of the ST-elevation (digoxin, glucagon, isoproterenol). Aramine could possibly have the opposite effect.

Only three ECGs of the 22 patients with anterior or inferior wall infarction who developed shock had minor ST-elevation, while six of the recordings of 12 patients with multiple infarctions had minor ST-elevation despite the presence of shock. This finding suggests that patients with multiple infarctions can possibly develop cardiogenic shock with a lower degree of acute myocardial ischemia than is the case with the other groups, or that patients with multiple infarctions causing extensive destruction are limited in their capacity to manifest ST-segment elevation.

Of 34 patients with shock 29 had the maximum ST-elevation during the first day. In 14 patients the maximum ST-elevation was demonstrated before
the symptoms of shock occurred, while in 16 patients the maximum ST-elevation was measured during the shock-syndrome. In four the maximum values of the ST-elevation were recorded after the symptoms of shock had disappeared.

**Mortality Rate**

The risk of death in patients treated in Coronary Care Units is generally 10–25%, depending upon the diagnostic criteria, the time of admission, distribution according to age and sex, and possibly on the various types of treatment. In this survey the mortality rate was 14%, but the figure is of no great interest as the survey does not include either patients with cardiac arrest on admission or patients admitted after the first 24 hours of the onset of symptoms.

In the groups studied the death rate is two to four times higher in patients with major ST-elevation than in patients with minor ST-elevation. The difference is statistically significant in patients with anterior wall infarction, in men, and in the total group. This is not surprising as the study has also shown a significantly greater frequency of the complications which most often lead to death in patients with major ST-elevation (cardiac arrest, congestive heart failure, A-V block 2°–3°, cardiogenic shock).

While all the results, which have been obtained in this study by using maximum values of the ST-elevation in standard leads, are in agreement with conclusions obtained by Maroko et al. using S-T, it does not appear that the number of standard leads with ST-elevation (NS-T) alone can be used to evaluate the risk of death. It is, however, possible that the ST-elevation and the number of leads with elevation together might result in a more reliable prognostic evaluation.

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BENT LYAGER NIELSEN

Circulation. 1973;48:338-345
doi: 10.1161/01.CIR.48.2.338

Circulation is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 1973 American Heart Association, Inc. All rights reserved.
Print ISSN: 0009-7322. Online ISSN: 1524-4539

The online version of this article, along with updated information and services, is located on the World Wide Web at:
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