A Comparison of Transmural and Nontransmural Acute Myocardial Infarction

By John E. Madias, M.D., Robert A. Chahine, M.D., Richard Gorlin, M.D., and Daniel J. Blacklow, M.D.

SUMMARY
The records of one hundred and four patients who had enzyme curves diagnostic for acute myocardial infarction (MI) were analyzed to determine what differences, if any, existed between the clinical course of patients with transmural myocardial infarction (TMI) and patients with nontransmural myocardial infarction (NTMI). The patients were subdivided into the two groups on the basis of accepted electrocardiographic criteria. There were no significant differences in prevalence or type of arrhythmias, occurrence of cardiogenic shock, or mortality in the hospital between the two groups. Enzymes tended to be somewhat lower in those with NTMI than in those with TMI. Clinical congestive heart failure (CHF) occurred more frequently in patients with TMI than in those with NTMI. Therefore, determining by electrocardiographic criteria whether or not a MI is transmural or nontransmural does not make it possible to predict the outcome or pattern of complications in a patient with acute myocardial infarction.

Additional Indexing Words: Arrhythmias   Congestive heart failure   Coronary care unit   Electrocardiogram

Acute ischemic injury to the myocardium can express itself in many forms. The clinical pattern can range from prolonged episodes of chest pain without a change in the enzyme levels or the electrocardiogram (ECG) to the full blown transmural myocardial infarction (TMI).\textsuperscript{1} Even when there is clear clinical evidence of necrosis, cardiologists usually divide the patients, on the basis of ECG criteria, into those with a transmural and those with a nontransmural myocardial infarction (NTMI). Diagnosis of NTMI has generally been considered indicative of a more favorable clinical course and final outcome.\textsuperscript{2, 3, 4} This prospective study of the two above named types of myocardial infarction (MI) was carried out in the Coronary Care Unit (CCU) of The Waltham Hospital (community hospital) to re-examine the hypothesis that the clinical outcome of patients with a NTMI was different from those with a TMI.

Material and Methods
Selection of Patients
One-hundred and four patients admitted to the CCU between July 1970 and March 1972 who were diagnosed as having had a MI were included in this study. Selection was carried out according to the following criteria:

1) Chest pain of over one hour in duration, occurring within the 48 hours preceding admission.\textsuperscript{2}

2) Curves of enzyme activity indicative of acute myocardial infarctions. Patients with elevated enzymes exhibiting unchanging levels were excluded from the study.\textsuperscript{1} Enzyme levels—creatine phosphokinase (CPK), serum glutamic oxaloacetic transaminase (SGOT) and lactic dehydrogenase (LDH)—were obtained serially for the first three days and subsequently as clinically warranted, except for the three patients who died within the first 48 hours.

3) Electrocardiographic considerations: ECGs, using standard 12 lead scalar tracings, were obtained on a daily basis in the CCU and every 2–3 days after discharge from the CCU. The precordium of the patients was marked to assure accurate daily repositioning of the "V" electrode. The following defines the criteria for both types of MI.

Transmural Myocardial Infarction (TMI): Recordings of Q waves with a duration \( \geq 0.04 \) sec and \( \geq 25\% \) of amplitude of the following R wave in the same lead,\textsuperscript{5, 6} accompanied by transient ST elevation and followed "temporarily" by T wave inversion (fig. 1).
Nontransmural Myocardial Infarction (NTMI): Patients with ECG results showing ST segments and T waves displaying a sequential pattern of change persisting throughout hospitalization. The following ECG alterations were noted:

1) ST segment depression of the ischemic "square wave" type, in the limb and precordial leads (figs. 2, 3, 4, 8–11).
2) Deep symmetrical inversion of T waves in some or all precordial leads with smaller similar changes in limb leads, changing in amplitude during the course of hospitalization (figs. 3, 4, 5, 6, 11, 12).
3) Transient reduction in R wave amplitude in the precordial leads (figs. 2, 4, 6).
4) Two patients had small transient nonpathologic Q waves (<0.02 sec in duration as observed in figure 3).
5) Slight ST segment elevation in aVR was observed in several but not all patients (figs. 2–6).

Twenty-two patients in the TMI group were known to have had a previous MI, although only 15 showed evidence by ECG recordings on admission. Sixteen patients of the NTMI group had a prior history of MI. Eight had persisting ECG evidence of an old inferior or infero-lateral TMI. Eight showed no signs of an old infarction by current ECG criteria.

Patients with chronic or acute, persistent left bundle branch block (LBBB) and right bundle branch block (RBBB) were excluded.

Fourteen patients in the TMI and four in the NTMI groups were on digitalis. A changing ST-T pattern helped in distinguishing pathologic patterns from stabilizing effects produced in the pre-infarction ECG by the cardiac glycoside.

All patients were examined by one of the authors. Continuous electrocardiographic monitoring was immediately instituted upon admission to the CCU or Intensive Care Unit (ICU) and maintained throughout hospitalization. Pointers for selection of alarm triggering levels were set at 60 and 100 beats/min. A magnetic tape "memory loop" recorder provided recordings of
ECG data 30 sec before the alarm activation (presumed arrhythmic) and continuously thereafter.

Four electrode chest placement was used. Multiple ECG lead combinations were available but an equivalent of lead V₁ was routinely employed, except in cases of an unsatisfactory tracing, at which time another lead was selected which displayed a more reliable or desirable tracing.

Oxygen was given to all patients at 6 liters/min by nasal prongs or face mask for one to two days. Sedation with diazepam and diazepoxide was used in all patients and morphine or meperidine was used for pain as indicated. Anticoagulation with heparin and/or coumarin was used in 23 patients with a TMI and 17 with a NTMI.

The Chi-square method was used for statistical analyses and P values of less than 0.05 were considered significant.

Clinical Features

Ninety-seven patients included in this study had chest pain. Seven patients (two with TMI and five with NTMI) having a presentation other than chest pain were also included (table 1). Their enzyme curves and ECG changes were comparable to that of the other 97 patients. The lowest maximal SGOT value observed in both groups (TMI and NTMI) in this study was 60 mIU/mL (milliInternational unit per milliliter).

Transmural Myocardial Infarctions: Sixty-one patients were diagnosed as having a TMI. Antero-septal, strictly anterior, and antero-lateral TMIs were combined as anterior and comprised 39% of

Results

Nontransmural MI. Sixty year old man. Note the marked ST depression in V₅-V₆ (3/14/71). Comparison of the ECGs of 2/3/67 and 3/30/71 shows that the tall R in V₆, observed on the admission ECG (3/14/71) is not related to a true posterior transmural MI since the patient had a persistent V₃ R/S ratio of >1 since 1967. Note the tiny Q waves (< 0.02 sec in duration) in V₅-V₆, which lasted for one day only. Symmetrically inverted T waves in I, II, aV₅₆, and V₅-V₆ were present two weeks after admission.

Figure 3

Nontransmural MI. Sixty-eight year old woman. A routine ECG on 9/18/70 was consistent with myocardial ischemia and/or the digitalis effect (patient on digitalis). On admission (1/21/71) for chest pain, the ECG exhibited ST and T changes in I, II, III, aV₅₆ and precordial leads. T waves were symmetrically inverted across the precordium. ECG changes persisted (1/26/71). Of interest is the transient decrease in R wave deflection in V₁ and V₂.

Figure 4

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the TMI group (24 cases). Twenty-three patients (38%) had an inferior wall TMI.1 Twelve patients (20%) had an infero-lateral wall TMI.1 One patient had a lateral wall MI,5 and one a lateral and true posterior wall MI.7 Figure 1 depicts two typical cases of TMI.

Nontransmural Myocardial Infarction (NTMI): Forty-three patients were diagnosed according to our criteria as having NTMI. One patient in this group manifesting a NTMI on two separate occasions two months apart (anterior and inferior ST-T changes respectively) has been included twice in this study.

Out of the five ECG alterations listed in Methods, no systematic or isolated patterns were detected among the patients, but multiple ECG abnormalities were seen during the clinical course. Because such changes occur without true infarction (fig. 7), final diagnosis depended on the accompanying enzyme level patterns.

The age of patients with a TMI ranged from 39 to 77 (average 60.5) years, and those with a NTMI from 45 to 86 (average 63.7) years. The average age of seven patients with atypical presentation (table 1) was 65.0 years (P = not significant). The average stay in the CCU was comparable (TMI–6.3 days, NTMI–5.0 days). The clinical features of all patients are summarized in table 2.

Although males predominated in both groups, there were more females in the NTMI group than in the TMI group. Seventeen women sustained a TMI and 19 a NTMI.

The duration of angina reported in the study by the patients ranged from seven years to one month prior to admission. No correlation was found between the prior duration of angina and the type of infarction.

Three patients (2–TMI, 1–NTMI) were found to be hypertensive on admission, while a portion of patients in both groups had had evidence in the past of hypertension. One patient with diabetes mellitus had peripheral neuropathy. He presented

Figure 5
Nontransmural MI. Fifty-nine year old woman. Note T wave inversion in the right precordial leads (8/4/71). Eight hours later an increase of R wave amplitude was recorded in V2, but without R, ST and T wave changes of a true posterior MI. Symmetrically inverted deep T waves across the precordium with ST depression in I, aVL and V5-V6 were present one week following admission with evolutionary changes recorded 18 days post admission (8/22/71).

Figure 6
Nontransmural MI. Seventy-four year old woman. Note persistent T wave changes in I, aVL and V5-V6. Also note the initial loss and subsequent return of R wave amplitude in V2 and V3.
Table 1

Summary of Admission Histories

<table>
<thead>
<tr>
<th>Mode of presentation</th>
<th>TMI</th>
<th>NTMI</th>
</tr>
</thead>
<tbody>
<tr>
<td>Typical</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Chest pain</td>
<td>59</td>
<td>38</td>
</tr>
<tr>
<td>Atypical</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Dyspnea</td>
<td></td>
<td>1</td>
</tr>
<tr>
<td>Pulmonary edema</td>
<td>2</td>
<td></td>
</tr>
<tr>
<td>Persistent left shoulder pain</td>
<td>1</td>
<td></td>
</tr>
<tr>
<td>Dizziness — weakness spells</td>
<td>1</td>
<td></td>
</tr>
<tr>
<td>Collapse (conscious on admission)</td>
<td>1</td>
<td></td>
</tr>
<tr>
<td>Unconscious on admission</td>
<td>1</td>
<td></td>
</tr>
<tr>
<td>(Remained in coma)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Total</td>
<td>61</td>
<td>43</td>
</tr>
</tbody>
</table>

Abbreviations: TMI = transmural myocardial infarction; NTMI = nontransmural myocardial infarction.

with pulmonary edema without chest pain (table 1); ECG results showed an inferior wall TMI (table 1).

Enzyme Changes

By definition, a rising and falling pattern of the curve of enzyme activity was required for inclusion of a case in the study. The NTMI group exhibited approximately half the maximal elevation in enzyme activity compared to the TMI group (table 3).

Mortality—Autopsy Findings

The rate of mortality was comparable in the two types of MI (TMI = 9.8% and NTMI = 9.3%) as depicted in figure 8. Nine of the ten deaths occurred in either the CCU or ICU (fig. 8). One patient with a TMI succumbed in the ward on the 48th day after admission. The causes of death are summarized in table 4. Autopsy was performed in three of the four fatalities with NTMI and none with TMI.

The average age of patients who died of a TMI was similar to the age of the total TMI group. The average age of patients who died of a NTMI was 7.17 (higher than the average age of the total NTMI group). Three of the six fatalities with TMI were men, and three of the four deaths in the NTMI group were men.

Localization of the fatal TMI by ECG criteria

![Figure 7](http://circ.ahajournals.org/)

Preinfarction angina—transmural MI. Sixty year old woman. History of hypertension of several years and angina for the two years prior to admission. The initial ECG shows possible old inferior wall TMI. T wave changes were indistinguishable from the ones observed in NTMI, but no changes in enzyme levels occurred. The patient was kept in the CCU for 12 days, because of daily short episodes of chest pain. She finally improved and was transferred to the ward only to return five days later with severe chest pain and ECG evidence of anterior wall TMI. Hyperacute and evolutionary phases are seen in the ECG patterns on 4/7/72 and 4/9/72. Enzyme levels rose in a pathognomonic pattern. The site of maximum T wave inversion was the site of greatest subsequent current of injury.
**Table 2**

<table>
<thead>
<tr>
<th>Clinical Features</th>
<th>Numbers and percentages</th>
<th>TMI (61)</th>
<th>NTMI (43)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Number of patients</td>
<td>61</td>
<td>43</td>
<td></td>
</tr>
<tr>
<td>Average age (years)</td>
<td>60.5</td>
<td>63.7</td>
<td></td>
</tr>
<tr>
<td>Sex — male</td>
<td>44 (72%)</td>
<td>24 (56%)</td>
<td></td>
</tr>
<tr>
<td>History of angina pectoris</td>
<td>28 (46%)</td>
<td>22 (51%)</td>
<td></td>
</tr>
<tr>
<td>History of previous myocardial infarction</td>
<td>22 (36%)</td>
<td>16 (37%)</td>
<td></td>
</tr>
<tr>
<td>History of hypertension</td>
<td>17 (28%)</td>
<td>16 (37%)</td>
<td></td>
</tr>
<tr>
<td>Incidence of diabetes mellitus</td>
<td>13 (21%)</td>
<td>6 (14%)</td>
<td></td>
</tr>
</tbody>
</table>

Abbreviations: TMI = transmural myocardial infarction; NTMI = nontransmural myocardial infarction.

was anterior in four cases and inferior in two. Only one fatal case with TMI had had a previous myocardial infarction. In comparison, all patients with fatal NTMI had had a prior MI.

Fatal, irreversible, primary ventricular fibrillation occurred in two patients with TMI five hours after admission and on the ninth day after admission, respectively. Neither exhibited a prior MI. Ventricular fibrillation occurred in one patient with NTMI who died on the first day of admission. Autopsy was not obtained, but there had been a prior MI by history and ECG changes. The patient who died in asystole had a transmural inferior MI and no prior MI; death occurred the fourth day after admission. The remaining fatalities are described in table 4.

Two patients in each group died of cardiogenic shock (class IV of Killip). The location of the MI by ECG criteria was anterior in the two patients with TMI, one of whom had a prior MI. Death occurred at the fifth hour and fourth day after admission, respectively. The two patients with NTMI and cardiogenic shock died on the fifth and sixth days after admission. In each instance disease of three coronary arteries and fresh NTMI were present at autopsy. The old MI was transmural in one, and in the other the old MI was extensive, nontransmural and subendocardial in location. In these two patients, while the acute MI was limited in range, its contribution to the amount of previously destroyed myocardium was probably incompatible with adequate pump function.18

**Arrhythmias**

We have elected to use the physiological instead of anatomical classification of arrhythmias19 which divides disturbances of rate, rhythm and conduction in three groups:

1) Arrhythmias of electrical instability.
2) Bradyarrhythmias or arrhythmias of potential electrical instability.
3) Arrhythmias of pump failure.

Fifty-seven (93.6%) in the TMI and 42 (97.6%) in the NTMI groups were detected as having one or more types of arrhythmias during their CCU course. Most of the arrhythmias were observed during the initial 72 hours after admission.20

**Arrhythmias of Electrical Instability**

Ventricular premature contractions (VPCs), fast ventricular tachycardia (FVT),21 and primary ventricular fibrillation were included in this group. Even a single recorded VPC was included in the analysis.

The ECGs of all patients who developed primary ventricular fibrillation showed VPCs or short runs of FVT only one to ten minutes prior to the onset of ventricular fibrillation.19, 22 As a result, only one patient had been placed on therapeutic doses of lidocaine prior to the onset of ventricular fibrillation.23, 24 Ventricular premature contractions recorded before onset of ventricular fibrillation exhibited the R-on-T phenomenon in two patients.25 DC defibrillation was applied repeatedly to the eight patients with primary ventricular fibrillation. Four of them died, three of irreversible ventricular fibrillation and one of prolonged coma following

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**Table 3**

<table>
<thead>
<tr>
<th>Enzyme Data</th>
</tr>
</thead>
<tbody>
<tr>
<td>Methodology</td>
</tr>
<tr>
<td>Technicon</td>
</tr>
<tr>
<td>Autoanalyzer</td>
</tr>
<tr>
<td></td>
</tr>
</tbody>
</table>

Abbreviations: TMI = transmural myocardial infarction; NTMI = nontransmural myocardial infarction; CPK = creatine phosphokinase; SGOT = serum glutamic oxaloacetic transaminase; LDH = lactic dehydrogenase; mIU/mL = milliInternational unit per milliliter; <sub>max</sub> = mean maximal enzyme value.

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Table 4
Causes of Death in Patients with TMI and NTMI

<table>
<thead>
<tr>
<th></th>
<th>TMI Total</th>
<th>TMI No. with prior MI</th>
<th>NTMI Total</th>
<th>NTMI No. with prior MI</th>
</tr>
</thead>
<tbody>
<tr>
<td>V.F.</td>
<td>2</td>
<td>0</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>Asystole</td>
<td>1</td>
<td>0</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>Post V.F. coma and pneumonia</td>
<td>—</td>
<td>—</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>CVA: ? 2° to MI and pneumonia</td>
<td>1</td>
<td>0</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>Cardiogenic shock</td>
<td>2</td>
<td>1</td>
<td>2</td>
<td>2</td>
</tr>
</tbody>
</table>

Abbreviations: TMI = transmural myocardial infarction; NTMI = nontransmural myocardial infarction; V.F. = ventricular fibrillation; CVA = cerebrovascular accident.

defibrillation. Ventricular fibrillation occurred within eight hours of admission in all cases except one patient.26 No correlation between the type of arrhythmia of electrical instability and the type of MI was found. No statistically significant differences were found in the incidence of various arrhythmias in the two types of MI (fig. 9), although ventricular tachycardia was somewhat more prevalent in those patients with TMI.

Bradyarrhythmias (Or Arrhythmias of Potential Electrical Instability)

Sinus bradycardia, complete heart block (CHB), slow ventricular tachycardia, and nodal rhythm were included together, because of their slow ventricular rates, predisposition to ectopic ventricular irritability, and their response to atropine-induced cardioacceleration.19, 22, 27, 28 The incidence of these arrhythmias is also depicted in figure 9. Nineteen of 25 cases with sinus bradycardia in the TMI group had inferior or infero-lateral infarction.29 Such a relationship could not be determined for the NTMI group because repolarization changes exhibited no consistent localized pattern. Complete heart block was observed in two patients in the TMI group, one with an inferior and one with an anterior MI. Four patients in the NTMI group developed CHB as a terminal event. For this reason they are not plotted in figure 9.

Slow ventricular tachycardia with rates ranging from 60-90 beats/min occurred in both types of MI (fig. 9). This arrhythmia was observed in a setting of sinus bradycardia.28

Arrhythmias of “Pump Failure”

The type of rhythm disorders depicted in figure 10 have been considered to occur with myocardial infarction-induced left ventricular dysfunction (see criteria below), although there is no substantial objective support for this belief as yet.19, 22, 30 The prevalence of sinus tachycardia and atrial flutter—atrial fibrillation was similar in both groups. There was an insignificantly higher prevalence of atrial premature beats and supraventricular tachycardia31 in the recordings from patients with NTMI. These latter two arrhythmias occurred against a background of sinus bradycardia.32

All but three patients who eventually developed clinical congestive heart failure (CHF) in both groups had had ECG evidence of one or more arrhythmias associated with pump failure before congestive heart failure was recognized. Conversely, however, only 45% of the TMI and 13% of the NTMI patients whose recordings showed one or more of the above mentioned arrhythmias supposedly associated with pump failure developed clinical CHF (P < 0.03), as depicted in figure 10.

Other Arrhythmias

Arrhythmias not included in the physiologic classification and atrioventricular and intraventricular conduction defects are shown in table 5.

Congestive Heart Failure

The diagnosis of CHF was based on clinical criteria: moist rales heard at lung bases persisting after vigorous coughing, an S3 gallop heard transiently or continuously, dyspnea, tachycardia, and a chest X-ray showing vascular redistribution or Kerley “B” lines. These patients were in classes II and III of CHF classification.17 No correlation be-
between acute congestive heart failure and previous myocardial infarction was noted in the patients with a TMI. On the other hand, all patients who developed CHF as a result of an acute NTMI had had a previous TMI. The incidence of CHF was higher in the TMI group (P < 0.02), as depicted in figure 11.

**Discussion**

**Diagnosis of Myocardial Infarction**

Acute myocardial infarction is best recognized when an orderly sequence of repolarization changes in the electrocardiogram is followed by development of a Q wave or loss of pre-existing R wave potential. While acute anterior or inferior MI is relatively easy to identify, this is not always the case for true posterior, high lateral or high anterior MIs. The more remote in time an ECG is taken, relative to the acute infarct, the less assurance there is that diagnosis is definitive. The situation is further compounded when previous MIs, which may possibly cancel out some electrical forces, are present.

While recognition of TMI can be difficult, NTMI presents even more diagnostic uncertainties. The findings in the repolarization stages are not diagnostic of necrosis, and may also be global rather than segmental in distribution. Thus, diagnosis in this category must rest on ancillary evidence which is usually derived from sequential changes in serum enzyme levels occurring *pari passu* with changes in the ECG pattern.

**Table 5**

<table>
<thead>
<tr>
<th>Type of arrhythmia, Atioventricular and Intraventricular Conduction Defects</th>
<th>TMI</th>
<th>NTMI</th>
</tr>
</thead>
<tbody>
<tr>
<td>Paroxysmal sinus arrest</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>A-V conduction defects</td>
<td></td>
<td></td>
</tr>
<tr>
<td>1° A-V block</td>
<td>9</td>
<td>3</td>
</tr>
<tr>
<td>2° A-V block</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mobitz I</td>
<td>3</td>
<td>0</td>
</tr>
<tr>
<td>Mobitz II</td>
<td>2</td>
<td>2</td>
</tr>
<tr>
<td>Intraventricular bundle branch defects</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Complete RBBB (transient)</td>
<td>3</td>
<td>3</td>
</tr>
<tr>
<td>Complete LBBB (transient)</td>
<td>3</td>
<td>1</td>
</tr>
<tr>
<td>Incomplete RBBB (transient)</td>
<td>0</td>
<td>1</td>
</tr>
<tr>
<td>Incomplete LBBB (transient)</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>Left anterior hemiblock</td>
<td>3</td>
<td>1</td>
</tr>
</tbody>
</table>

Abbreviations: TMI = transmural myocardial infarction; NTMI = nontransmural myocardial infarction; A-V = atrioventricular; RBBB = right bundle branch block; LBBB = left bundle branch block.

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*Circulation, Volume XLI, March 1974*
Differences in the Pathology of the TMI and NTMI

Transmural myocardial infarction can usually, although not always, be attributed to acute coronary occlusion, most often caused by fresh local arterial thrombosis superimposed on an old atherosclerotic plaque. A nontransmural infarction, on the other hand, does not exhibit thrombotic occlusion as frequently, and is often layered within the heart. It is presumably the lack of involvement of the outer layer of myocardium that inhibits development of a Q wave. Whether the two types of infarctions have similar natural histories is not certain, particularly in view of the difference in their pathogenesis.

Differences in Clinical Manifestations Between TMI and NTMI

Arrhythmias

The two groups could not be separated on the basis of arrhythmias. This observation suggested that either necrotic tissue per se or the deep layer of the myocardium itself is the site of arrhythmogenesis. There was no difference between the two groups in the arrhythmias attributed by other authors to pump failure (fig. 10), although the incidence of heart failure varied markedly depending on the type of MI (fig. 11). This lack of association between supraventricular tachycardias and heart failure challenges the validity of the functional classification which associates particular arrhythmias with heart failure. Nevertheless, when heart failure occurred it was usually preceded by a supraventricular tachyarrhythmia complicating the MI. Pericarditis may occasionally be the activating mechanism of these arrhythmias.

Heart Failure and Shock

As mentioned before, clinical heart failure was found more frequently in association with a TMI than with a NTMI. This may relate in part to the size as well as the location of the infarction, and is evidenced by the generally higher level of enzyme activity seen with the TMI group when compared to the NTMI group. Furthermore, only when there had been a prior MI did heart failure occur with a NTMI. As reported by others, congestive heart failure may occur with the first TMI.

Fatal cardiogenic shock occurred as frequently in both the TMI group as in the NTMI group; however, there was one difference. In the two patients with TMI a massive area of acute necrosis developed in the absence of any prior old myocardial damage. However, in the two cases with NTMIs, the area of new necrosis was relatively small, but extensive areas of old infarction and scar tissue were present. Thus, a new minor injury apparently tipped the balance in a previously damaged heart, decreasing the amount of functioning muscle below the level necessary for survival. This phenomenon may well explain the occasional patient who develops shock with an apparently minor acute MI.

Mortality

Mortality, as well as causes of death, were similar in both groups of patients. Wolk et al. reported a very wide range of mortality rates with acute MI, from 8 to 44%. Generally, mortality tended to be higher than reported in our findings and possibly was related to the slightly lower prevalence of heart failure in this study as opposed to the results of others. Wolk and co-authors further elaborated on the significance of congestive heart failure present in 49% of 112 consecutive patients. Fifteen percent of the heart failure group died, while only 5% of the patients who improved or continued in class II died during hospitalization. Selection of patients with specific diagnostic ECG recordings or enzyme level patterns, and inclusion of only those patients who survived the first few hours following admission, may also have influenced the composition of the groups finally studied. It is probably significant that all four patients with fatal NTMIs had had a prior MI when compared to only one in the six patients who died with a TMI. The presence of this variable, which may be unknown on the basis of medical history alone of a patient with ischemic heart disease, suggests that prognosis cannot be decided solely on the basis of an acute current event. Unless further research finds clear differences between the clinical course of patients with TMI and those with NTMI, both types of infarction should be observed with equal attention.

References


Circulation, Volume XLIX, March 1974

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Circulation. 1974;49:498-507
doi: 10.1161/01.CIR.49.3.498

Circulation is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
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Print ISSN: 0009-7322. Online ISSN: 1524-4539

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