Severity of Contraction Abnormalities
After Acute Myocardial Infarction in Man:
Response to Nitroglycerin

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SUMMARY In order to determine the severity of contraction abnormalities in acute myocardial infarction and their response to sublingual nitroglycerin, 23 patients were studied by radionuclide angiography within 6–24 hours after the onset of chest pain. Radionuclide angiograms were obtained in the right anterior oblique projection before and after nitroglycerin and segmental shortening was determined using a hemiaxis method in the central and lateral chords of the infarcted and nonischemic zones. All 23 patients had asynergy in the zone of acute infarction. Mean hemiaxis shortening of the ischemic zone was depressed after acute myocardial infarction and improved after sublingual nitroglycerin (p < 0.001). Nonischemic zone hemiaxis shortening also improved after nitroglycerin. In another group of 16 patients with documented remote myocardial infarction (more than 1 year old) the response to nitroglycerin of contraction abnormalities in acute and remote myocardial infarcts was studied. Hemiaxis shortening in the zone of infarction was 14.8 ± 11.2% and did not improve after nitroglycerin. Nonischemic zone hemiaxis shortening improved with nitroglycerin from 29.9 ± 20.2% to 37.6 ± 22.8% (p < 0.02). Ejection fraction improved slightly but not significantly in both the acute and remote infarction groups after nitroglycerin.

In the acute group, ejection fraction increased from 33.4 ± 10.8 to 36.4 ± 10.8% and in the remote group from 38.6 ± 15.9 to 41.2 ± 14.3%. Thus, the data indicate that zones of both acute and remote infarction are characterized by a significant reduction in hemiaxis shortening. However, 6–24 hours after acute myocardial infarction, increased hemiaxis shortening is demonstrable in the area of infarction after nitroglycerin administration. No such change occurred in the infarct zone of patients with remote infarction.

RECENTLY interest has focused on the determination of myocardial infarct size. Several indices of ischemic injury have been studied experimentally, including the nature and extent of contraction abnormalities after both complete and varying degrees of coronary obstruction.

Radionuclide angiography has been shown to correlate well with contrast ventriculography in detecting and quantitating regional contraction abnormalities in patients with chronic coronary heart disease and to be useful in evaluating global and segmental abnormalities in patients with acute myocardial infarction. The present study was therefore undertaken using first-pass radionuclide angiography to determine: 1) the severity of contraction abnormalities after acute myocardial infarction in man; 2) the response of these contraction abnormalities to sublingual nitroglycerin; and 3) the difference in response of contraction abnormalities associated with remote (more than 1 year old) infarction.

Methods

Selection of Patients

Thirty-nine patients were studied. The acute study group comprised 23 patients admitted to the coronary care unit with an acute myocardial infarction. They all gave informed consent for radionuclide angiographic studies. Patients were diagnosed as having an acute myocardial infarction if they had significant ST elevations (> 2 mV) and then developed Q waves 2 mm deep and 0.04 second wide with concomitant elevations of MB-CK isoenzymes. Only patients who had the onset of chest pain within 24 hours (range 6–24 hours, mean 17.3 hours) before radionuclide study were included in the study. We also studied a control group of 16 patients, all of whom had a remote myocardial infarction documented by similar Q-wave criteria that were present on the ECG at least 1 year before radionuclide angiography.

Radionuclide Angiograms

Radionuclide angiograms were obtained using a computerized multicrystal scintillation camera (Baird Atomic System Seventy-seven) and a high-sensitivity, parallel-hole collimator. An 18-gauge percutaneous intravenous catheter was placed in an antecubital vein. After administration of 200 mg of potassium perchlorate, the patient was positioned initially in either the left or right anterior oblique view, and a 12–15-mCi dose of technetium-99m pertechnetate in a volume less than 0.8 ml was rapidly administered and flushed with 10–15 ml of 5% dextrose in water to obtain a bolus injection. Counts were recorded at a framing interval of 40 or 50 msec during the first pass of the isotope. Data were recorded on computer disc for processing and magnetic tape for long term storage. After 5–10 minutes the patient was positioned in the opposite view and a background frame collected. Within 30 seconds of obtaining the background frame,
A second injection of pertechnetate was administered and recorded as for the first injection. Acquisition required less than 30 seconds for each view.

After the second radionuclide angiogram, the patient was given 0.4-0.8 mg nitroglycerin sublingually to reduce the systolic pressure by 10-15 mm Hg from control. After a decrease in systolic pressure was observed, the right anterior oblique radionuclide angiogram was repeated as described above.

Analysis of Angiographic Data

Radionuclide angiograms were first corrected for nonuniformity of field by automatically correcting each frame of data for crystal bias using a uniform source obtained each day. This resulted in each crystal demonstrating a uniform response to gamma emissions. In addition, a dead-time correction was applied. For the second and third radionuclide angiograms the background frame obtained immediately before this study was automatically used by the computer to correct each frame of data for preexisting counts on a crystal-for-crystal basis.

A region of interest comprising the left ventricle was selected using a zone grid representing the individual crystals. A time-activity curve was generated that displayed counts recorded by the flagged crystals. This resulted in a series of four to eight peaks (diastole) and valleys (systole) that were used to derive a computer-generated representative cycle. This cycle could then be played in cine mode to qualitatively analyze the contraction pattern of the left ventricle. In addition, computer-derived images of end-diastole, end-systole and computer-superimposed perimeters of end-diastole and end-systole were used to provide a quantitative index of wall motion using the hemiasis method. The computer-generated images of end-diastole and end-systole reflect the spatial location of the heart at a given time interval; that is, each image is spatially captured such that any movement represents cardiac movement and not patient or camera motion. The frame representing end-diastole and end-systole as generated are then used to derive and display the computer-added end-diastolic and end-systolic images.

For the contrast ventriculogram, end-diastolic and end-systolic frames were traced and the aortic valve plane was demarcated. A line connecting the midpoint of the aortic plane and the apex was drawn and the end-diastolic and end-systolic frames were superimposed. Hemiases were drawn perpendicular to the long axis and hemiasial shortening was classified as follows: normal contraction, greater than 25% hemiasis shortening; hypokinesis, less than 25% hemiasis shortening; akinesis, less than 5% hemiasis shortening; and dyskinesis, a paradoxical systolic expansion. Corresponding areas on the radionuclide angiographically determined and computer-superimposed perimeters were then determined and used to delineate normal contraction, hypokinesis or akinesis for the radionuclide angiogram.

The ischemic zone (clinically defined by new pathologic Q waves), for the purpose of this study, was the hemiasis chord which most closely bisected the asynergic zone. For example, figure 1 shows a patient with an inferior wall myocardial infarction. A chord was drawn to bisect the inferior wall. In addition, chords adjacent to this hemiasis were drawn at equal distances, bisecting the ischemic zone, and averaged (fig. 1). Each hemiasis was measured and recorded as percent change from end-diastole to ascertain the

**Figure 1.** A) Superimposed angiographically derived end-diastolic and end-systolic perimeters. Dotted lines represent lateral chords and the central chord is in the center of the asynergic area. Control study reveals central chord shortening of 21% and average lateral chord of 17%. Nitroglycerin (TNG) administration increased the central chord to 39% and the lateral chord to 43%. Two solid lines parallel to the long axis represent chords used to assess the apex. B) Computer generated and added images of end-diastolic perimeter and end-systolic image for both the control (C) and nitroglycerin (N) studies for the same patient as in figure 1A. Counts are displayed in a 16-color isocount format normalized to the crystals with the greatest number of counts. Color scale ranges from black, representing the least number of counts per crystal, through red, yellow and white, representing the greatest number of counts.
amount of regional contraction. Hemiaxis shortening for the nonischemic zone was calculated by averaging and comparing the control and nitroglycerin studies.

Ejection fraction was determined from the right anterior oblique projection radionuclide angiograms using the difference in counts method after appropriate background correction of 35%. According to the formula \(\text{ED}_c - \text{ES}_c)/(\text{ED}_c - \text{BA})\), where \(\text{ED}_c\) equals end-diastolic counts, \(\text{ES}_c\) equals end-systolic counts and \(\text{BA}\) equals background activity. This technique has a correlation of 0.801 with contrast ventriculography in a series of 44 patients including 22 patients with coronary artery disease and asynergy.

Statistical analysis was performed using the \(t\) test for determining significance of the change in hemiaxis shortening in a patient before and after nitroglycerin. The \(t\) test was used to compare groups. A \(p\) value < 0.05 was considered significant. All values are given as mean ± SD.

**Results**

**Clinical Features**

Of the 39 patients included in the study, 23 had an acute and 16 had a remote myocardial infarction. In the acute group there were 16 males and seven females, while in the remote infarct group there were 11 males and five females. The ages of the patients ranged from 31–90 years. The acute myocardial infarction group included seven patients (four male and three female) with clinical (S₃ gallop and/or basilar rales) and radiologic evidence of cardiac failure. All except one patient (who developed cardiogenic shock and died) recovered sufficiently to leave the coronary care unit. None of the patients in the remote infarct group had clinical or radiologic evidence of cardiac failure. Apart from one patient who was digitalized before the development of chest pain, none of the other patients were receiving cardiotonic drugs before the study. Two patients in the acute and one patient in the remote infarct group were hypertensive (diastolic pressure > 95 mm Hg) before study.

**Electrocardiogram**

In the acute group, 12 had inferior myocardial infarcts and five anterior infarcts. In addition, there were three patients with both inferior myocardial infarctions and lateral extensions (i.e., pathological Q waves in leads V₄₋₅ and one inferoposterior (positive R in V₅) myocardial infarction. There were two patients with anterolateral myocardial infarction (Q waves and ST changes in leads I, aVF and V₄₋₅). Three patients had previous myocardial infarction, two inferior and one anterior. Peak total CK was 1017 ± 488 IU/ml and MB fraction was 104 ± 62 IU/ml. In the remote infarct group 11 patients had inferior Q waves and five had Q waves anteriorly.

**Hemiaxis Shortening in Acute Myocardial Infarction**

**Zone of Ischemic Injury**

In the 23 patients with acute myocardial infarction, all showed asynergy in the zone of the left ventricle corresponding to the electrocardiographic location.

The central hemiaxis chord in the infarct zone showed a mean shortening of 16.1 ± 7.7%. The mean hemiaxis shortening for the lateral chord was 19.3 ± 6.3% (table 1). Twenty of 23 patients showed a positive response to sublingual nitroglycerin (figs. 2 and 3). The mean central hemiaxis for the group as a whole increased from 16.1 ± 7.7 to 25.4 ± 14.7% (\(p < 0.001\)). Of the three patients who did not respond to nitroglycerin, one with an acute inferior myocardial infarction with inferior zone dyskinesis showed paradoxical bulging with nitroglycerin (−5 to −14% shortening) and subsequently developed cardiogenic shock and died. Of the other two patients who did not improve with nitroglycerin, one presented with primary ventricular fibrillation and was defibrillated in the emergency department before admission to the coronary care unit (5 to 3% hemiaxis shortening). He developed an acute inferior myocardial infarction with inferior zone akinesis. After admission to the coronary care unit, he had an uneventful recovery. The third patient was in cardiac failure before developing a myocardial infarction (14 to 9% hemiaxis shortening).

In 21 of 23 patients, the lateral chord improved with nitroglycerin (fig. 3). The segmental shortening for the whole group before nitroglycerin was 19.3 ± 6.3% and this improved significantly after nitroglycerin, to 28.3 ± 10.6% (\(p < 0.001\)). The response to nitroglycerin, however, was not significantly different from the central chord (table 1). There was no relationship between the time interval when the study was performed and the degree of abnormality at rest and after administration of nitroglycerin.

Three of the patients with an acute myocardial infarction had asynergic zones associated with an old myocardial infarction. There was no significant response to nitroglycerin with mean hemiaxis short-

**Table 1.** Hemiaxis Shortening and Effects of Nitroglycerin in the Infarct and Nonischemic Zones in Acute Myocardial Infarction

<table>
<thead>
<tr>
<th>Zone</th>
<th>Central chord</th>
<th>Lateral chord</th>
<th>Nonischemic zone</th>
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<tbody>
<tr>
<td></td>
<td>Before</td>
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<tr>
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<td>nitroglycerin</td>
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<tr>
<td></td>
<td>16.1 ± 7.7%</td>
<td>25.4 ± 14.7%*</td>
<td>19.3 ± 6.3%</td>
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\(p < 0.001\).
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ing of 15.0 ± 4.7% before nitroglycerin and 15.5 ± 3.1% after nitroglycerin.

Nonischemic Zone

The nonischemic zone improved in all 23 patients after nitroglycerin administration. Segmental shortening in the nonischemic zone was 26.6 ± 9.0% and improved to 36.7 ± 11.1% after administration of nitroglycerin (p < 0.001) (table 1).

Hemiasis Shortening in Remote Myocardial Infarction

Dysynergic Zone

Hemiasis shortening in the zone of remote infarct was 14.8 ± 11.2% (table 2). In contrast to the acute group, nitroglycerin had no significant effect on ischemic zone segmental shortening in this group of patients (fig. 4). Of the 16 patients, four improved slightly, three deteriorated and the rest did not change. Thus, mean hemiasis shortening was 14.8 ± 11.2% before and 15.3 ± 13.5% after nitroglycerin (table 2). Seven patients with remote infarction also underwent cardiac catheterization, including contrast ventriculography at rest and after administration of nitroglycerin. Of these seven, one had hypokinesis, four had akinesis and two had dyskinesis in the zone demarcated by the pathologic Q waves. None demonstrated improvement with nitroglycerin. Radionuclide angiography revealed similar resting abnormalities, with hemiasis shortening in the asynergic zone of 5.8 ± 8.0 at rest and 7.2 ± 9.8% after nitroglycerin (NS).

Nonischemic Zone

Segmental shortening in this zone before nitroglycerin was 29.9 ± 20.2% and this improved after nitroglycerin to 37.6 ± 22.6% (p < 0.02).

Ejection Fraction

In the acute infarct group the mean ejection fraction was 33.4 ± 10.8%. This increased insignificantly to 36.4 ± 10.8% after nitroglycerin administration. In the group of patients with anterior infarction, the ejection fraction improved from 31.6 ± 9.8 to 35.0 ± 9.1% (table 3). In the patients with inferior infarction the ejection fraction improved similarly, from 35.3 ± 13.9% to 37.7 ± 14.3%. In the remote infarct group, the mean ejection fraction was 38.6 ± 15.9% and 41.1 ± 14.3% after nitroglycerin. In the group of

<table>
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<tr>
<th>Infarct zone</th>
<th>Nonischemic zone</th>
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<tr>
<td>Before nitroglycerin</td>
<td>Before nitroglycerin</td>
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<tr>
<td>After nitroglycerin</td>
<td>After nitroglycerin</td>
</tr>
<tr>
<td>14.8 ± 11.6%</td>
<td>29.9 ± 20.2%</td>
</tr>
<tr>
<td>15.3 ± 14.0%</td>
<td>37.6 ± 23.6*</td>
</tr>
</tbody>
</table>

*p < 0.02.
patients with anterior infarction, the ejection fraction improved from $41.7 \pm 11.6\%$ to $45.0 \pm 10.5\%$. In the group with inferior infarction, the ejection fraction improved similarly from $38.3 \pm 23.5$ to $41.1 \pm 21.6\%$ (table 3).

**Heart Rate**

In the acute infarct group, the mean heart rate was $80.1 \pm 17.3$ beats/min before nitroglycerin. The rate increased insignificantly to $87.3 \pm 20.6$ beats/min (table 4). Similarly, in the chronic infarct group, mean heart rate increased from $69.4 \pm 6.2$ to $73.3 \pm 10.8$ beats/min.

**Blood Pressure**

In patients with acute infarction, systolic pressure was $123.4 \pm 16.9$ mm Hg and decreased after nitroglycerin to $111.7 \pm 16.9$ mm Hg ($p < 0.025$) (table 4). Similarly, in patients with chronic infarction, the systolic pressure decreased from $126.0 \pm 20.1$ mm Hg to $116.3 \pm 22.1$ mm Hg ($p < 0.001$).

**Discussion**

Several experimental studies have indicated that the extent and severity of myocardial ischemic injury and resultant contraction abnormality can be altered substantially by various interventions. However, the time constraints required for effective preservation of ischemic and potentially reversibly damaged myocardium may be quite stringent. Ischemic injury might become irreversible in time frames encompassing only a few hours after the onset of the infarction depending, in part, on whether the occlusion is complete or partial.

Both the size and evolution of a myocardial infarction are complex and variable, depending on a variety of interrelated factors. Flow reduction with resultant ischemia or infarction is dependent on the extent and severity of the coronary artery lesion and the mechanism and time course of flow reduction. Other factors also may modify the rate of extent of an evolving infarction in the area of left ventricle subserved by the occluded vessel, including the functional status of the other major coronary vessels and the status of coronary collateral circulation as well as myocardial oxygen requirements.

The results of the present clinical study indicate that 6-24 hours after the symptomatic onset of acute myocardial infarction, there is a significant abnormality in hemiasis shortening in the zone of myocardium corresponding to the ECG location of the infarct. Central hemiasis chord shortening was reduced

![Control and TNG Radionuclide angiogram](image)
to 16.1 ± 7.7% while lateral chord shortening was 19.3 ± 6.3%. Variability in severity of the contraction abnormality was present (fig. 3). One patient with dyskinesia on the initial study with no improvement after nitroglycerin developed cardiogenic shock 2 days later and died. This initial variability is consistent with a recent study by Reduto et al.9

In addition, experimental data have demonstrated that the progression and severity of changes within the ischemic area after coronary occlusion are not uniform.27, 28 Thus, electrographic, biochemical and histopathologic parameters indicate that after coronary occlusion, the endocardium is the most severely affected area, with a temporal progression of abnormalities toward the epicardium. In addition, recent studies from our laboratory using a 50% reduction in coronary blood flow have shown that within the ischemic area, epicardial tissue can maintain active systolic shortening while the endocardium manifests evidence of systolic bulging.29 Thus, ischemic myocardium is composed of zones of myocardium that are variably affected by coronary flow reduction. The clinical findings of the present study are consistent with the experimental data indicating variable progression of ischemic damage.

An important consideration in this study is the accuracy of radionuclide angiography. Correlations performed in our own laboratory with contrast ventriculography have shown a good overall correlation for ejection fraction whether obtained in the right anterior oblique view or in left anterior oblique view.8 In addition, our own experience, as well as that of Marshall et al., has been that ejection fraction determination is repeatable.30 Assessment of wall motion using radionuclide angiography is quite accurate, as previously reported,6 although interpretation of inferior wall motion may be difficult.5 In that study, anterior and apical zones were accurately delineated with the inferior segment asynergy detected in 13 of 17 patients. However, it remains clear that improvements in frame and count rates would further enhance delineation of wall motion and aid in detection of subtle changes from end-diastole to end-systole that are particularly important in this study. It would also be of considerable value if assessment of transmural involvement would be possible. Clearly, the limitations of this technique must be kept in mind when interpreting these data.

The response of the contraction abnormalities that result from acute myocardial infarction to sublingual nitroglycerin was unexpected in light of previous studies that demonstrated the absence of an improvement in the contractile performance of a central ischemic zone after administration of nitroglycerin or nitroprusside to dogs subjected to coronary artery ligation.13-15 Twenty of the 23 patients demonstrated a positive response to sublingual nitroglycerin. Both central and lateral hemi-axes chords demonstrated improvement for the group as a whole from 16.1 to 25.4% (p < 0.001) and 19.3 to 28.3% (p < 0.001), respectively. In contrast, nitroglycerin had no significant effect on segmental shortening in patients with Q-wave infarctions that occurred at least 1 year before study. The hemi-axial shortening in this group of patients was 14.8% before and 15.3% after nitroglycerin administration. Similarly, the three patients with both acute and remote myocardial infarction showed no change in the remote areas after nitroglycerin.

These data may be interpreted in several ways. Perhaps the infarct zone improved as a result of remaining areas of still functionally viable myocardium possessing functional contractile reserve and capable of responding to nitroglycerin administration.31 This would also explain the difference in response to nitroglycerin in patients with acute and remote in-

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TABLE 3. Changes in Ejection Fraction Before and After Nitroglycerin in Acute and Chronic Myocardial Infarction

<table>
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<tr>
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<th>Acute infarction</th>
<th>Chronic infarction</th>
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<tbody>
<tr>
<td></td>
<td>Before nitroglycerin</td>
<td>After nitroglycerin</td>
</tr>
<tr>
<td>Anterior</td>
<td>31.6 ± 9.8%</td>
<td>35.0 ± 9.1%</td>
</tr>
<tr>
<td>Inferior</td>
<td>35.3 ± 13.9%</td>
<td>37.7 ± 14.3%</td>
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</table>

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TABLE 4. Changes in Heart Rate and Blood Pressure Before and After Nitroglycerin in Acute and Chronic Myocardial Infarction

<table>
<thead>
<tr>
<th></th>
<th>Acute infarction</th>
<th>Chronic infarction</th>
</tr>
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<tbody>
<tr>
<td></td>
<td>Before nitroglycerin</td>
<td>After nitroglycerin</td>
</tr>
<tr>
<td>Heart rate</td>
<td>80.1 ± 17.3 beats/min</td>
<td>87.3 ± 20.6 beats/min</td>
</tr>
<tr>
<td>Systolic</td>
<td>123.4 ± 16.9 mm Hg</td>
<td>111.7 ± 16.9 mm Hg</td>
</tr>
<tr>
<td>Diastolic</td>
<td>81.3 ± 11.3 mm Hg</td>
<td>77.8 ± 11.7 mm Hg</td>
</tr>
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* p < 0.025.
† p < 0.001.
‡ p < 0.05.
farcts. The experimental demonstration that there is a progression of electrographic, biochemical, histologic and functional abnormalities from endocardium to epicardium as a function of time would support this conclusion.27-29

However, changes in compliance of the zone of acute myocardial infarction may be involved. Tennant and Wiggers28 first showed bulging (an increase in compliance) of the ischemic zone of left ventricle after acute coronary occlusion, which is associated with an early increase in compliance.30 However, the duration of these compliance changes are unclear. Hood et al.31 demonstrated that increased stiffness appears in the infarcted area of animals 3–5 days after the infarct. Parmley et al.32 noted in man that paradoxical expansion was still present 3 and 6 days after infarction. Diamond and Forrester33 in a study of patients 2–24 hours after acute myocardial infarction found an increase in wall stiffness. If compliance of the infarct areas is reduced, facilitation of contraction of nonischemic myocardium that resulted from nitroglycerin administration would allow for a passive movement of otherwise nonfunctional myocardium. It has been shown experimentally that areas remote from the acute infarct exhibit depressed contraction and are, therefore, not truly normal.37 In the present study hemias axis shortening was reduced in the nonischemic areas35 and was improved after administration of nitroglycerin. These depressed areas may have been able to augment significantly their contraction in the early stage of the acute infarct, facilitating the passive inward movement of the infarct zone. Thus, changes in compliance of the ischemic or the normal zone during the acute phase may differ enough from that of a 1-year-old infarct so as to change their apparent responses to nitroglycerin. Also, the changes in compliance of the infarcted region or some geometrical changes in the ventricles’ response to the healing process might have occurred. In view of the three-dimensional nature of the images and the uncertainties mentioned above, it is not surprising that lateral and central chords showed no significant differences.

The issue of whether a change in wall motion after nitroglycerin is a nonspecific result of changes in hemodynamic parameters or is related to a true improvement in contraction of the involved segment was examined in a previous study from our laboratory.38 We noted a similar reduction in left ventricular volume regardless of the presence of reversible or irreversible asynergy.39 In addition, in the presence of marked asynergy, ejection fraction improved only if large areas of asynergy improved.39 Thus, in patients with a lower ejection fraction and marked asynergy, ejection fraction improved only if multiple segments improved. More recently, similar findings have been reported.40 While those studies were carried out in chronic patients, segmental asynergy can probably improve without improvement in global left ventricular function as measured by ejection fraction. The fact that ejection fraction did not significantly improve in the present study may relate to the size of the asynergic zone involved and/or the concomitant change in end-diastolic volume. Although it could be argued that it relates to measurement technique, the excellent correlation between radionuclide angiography and contrast ventriculography make this unlikely.5,7

The observations concerning remote infarction are consistent with our previous findings that the presence of Q waves in patients with chronic coronary heart disease significantly decreases the likelihood of the asynergic zone improving after nitroglycerin.15 At the remote states of the infarct, a good correlation was found between the results of nitroglycerin contrast ventriculography and histopathologic evidence of myocardial necrosis and/or fibrosis.40 However, these findings cannot be extrapolated to acute infarction.

Further studies are important to better understand these findings. In particular, the time course of the differential response to nitroglycerin and the effect of the degree and duration of the lowering of systemic pressure in patients with acute myocardial infarction require evaluation. Although this type of intervention will result in reduced afterload, and thus decrease oxygen demand, excessive lowering of systemic pressure could adversely affect coronary flow and myocardial perfusion, with a net negative balance between oxygen supply and myocardial demand. Thus, an appropriate degree of pressure reduction must be determined to result in optimal improvement of the ischemic zone. Perhaps most important, the effect of an intervention such as afterload reduction on the natural history of the contractile abnormalities after an acute myocardial infarction must be clarified. These studies should allow a better understanding of the mechanism and clinical importance of the observations made in the present study.

Acknowledgment

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