The Relation between the Location of Coronary Occlusions and the Occurrence of Shock in Acute Myocardial Infarction

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The mortality rate of shock associated with acute myocardial infarction remains high despite a variety of therapeutic approaches. The relative importance of reduced cardiac output and reflex factors in the pathogenesis of this grave condition requires clarification if more satisfactory therapeutic measures are to be developed. Support for a reflex mechanism may be found in experimental coronary artery embolization in dogs. Microspheres that lodged in the right angle branches of the main stem coronary arteries produced shock, whereas larger or smaller emboli did not. These observations, supported by hemodynamic data, suggested a reflex mechanism with receptors in the right angle branches, which interfered with an adequate compensatory rise in peripheral resistance.

The reflex hypothesis has not been verified in man, however. Hemodynamic data or angiographic studies are sparse because of the hazard in performing these studies on precariously ill patients. The present study, utilizing postmortem coronary angiograms, correlates the location of coronary artery occlusions and the occurrence of shock in acute myocardial infarction.

Method

More than 500 postmortem examinations of patients who died of acute myocardial infarction at the Beth Israel Hospital between 1938 and 1955 were reviewed. All had been studied by the coronary injection and dissection technic of Schleisinger. Cases with an additional disease or other complication of myocardial infarction capable of producing shock were excluded (table 1). Most of the postmortem coronary angiograms obtained in 1949 were also excluded because of a change in the injection technic. About a third of the eligible cases were excluded because the angiograms were not available. The final series consists of 127 carefully studied patients who died following an acute myocardial infarction, with no associated disease capable of producing shock.

The clinical histories were reviewed to determine whether shock attributable to acute myocardial infarction had been present. The criteria for shock were a systolic blood pressure of 80 mm. Hg or less, plus one or more confirmatory signs, including anuria or severe oliguria, pallor, cold sweaty skin, and dulled sensorium. A systolic blood pressure of 100 mm. Hg was accepted for patients with a clear, previous history of hypertension, when other signs of shock were present as well.

The postmortem angiograms and descriptions of coronary artery dissections were examined without knowledge of the clinical histories. The location of fresh occlusions was noted. Occlusions in the right coronary artery, and in the two major branches of the left coronary artery, the left anterior descending and the left circumflex, were

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Table 1

Conditions Associated with Shock. Patients with These Diseases Were Excluded from the Study

1. Rupture of the myocardium
2. Rapid arrhythmias
3. Complete heart block
4. Dissecting aortic aneurysm
5. Pulmonary embolism
6. Mesenteric thrombosis or embolism
7. Cerebral vascular accident
8. Peripheral vascular embolism
9. Gastrointestinal hemorrhage
10. Pneumonia
11. Septicemia
12. Disseminated carcinomatosis

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considered main stem occlusions. Occlusions found in the primary branches of the main stem vessels, or in branches formed after their terminal bifurcations were classified as branch occlusions. The degree of atherosclerotic involvement of the coronary arteries was graded on a scale of zero to nine, with each of the three main stem vessels contributing a maximum score of three, representing complete occlusion. The surface area of the acute infarct in each heart was estimated from the original drawing of the unrolled heart that accompanied each angiogram. The size of the infarct was graded from one to three, one representing minimal and three representing massive infarction.

The occurrence of shock in each case was then correlated with the presence and location of fresh occlusions, the extensiveness of coronary atherosclerosis, and the size of the infarct.

## Results

Forty-six of the 127 patients had shock associated with acute myocardial infarction during their terminal illness. There were no significant differences in age and sex between patients with and without shock (table 2).

The occurrence of shock in patients with fresh main stem occlusions, branch occlusions, or infarction without fresh occlusion is shown in figure 1. Shock occurred in 12 of 16 patients (75 per cent) with branch occlusions as compared to 19 of 54 cases (35 per cent) with main stem occlusions, and 15 of 57 cases (26 per cent) with no fresh occlusion. The difference in the frequency of shock among the three groups is statistically significant (0.005 > p > 0.001).

The distribution of fresh occlusions in each of the three main stem coronary arteries in patients without shock followed the pattern described in previous studies.5, 6 In the patients with shock, however, there were fewer left anterior descending occlusions, and the relative frequency of left circumflex occlusions was increased (fig. 2). This difference is not so marked as that between patients with

### Table 2

<table>
<thead>
<tr>
<th>Shock</th>
<th>Number</th>
<th>Mean Age</th>
</tr>
</thead>
<tbody>
<tr>
<td>Men</td>
<td>(32)</td>
<td>(63.9)</td>
</tr>
<tr>
<td>Main stem</td>
<td>13</td>
<td>68.6</td>
</tr>
<tr>
<td>Branch</td>
<td>7</td>
<td>62.1</td>
</tr>
<tr>
<td>No fresh occlusion</td>
<td>12</td>
<td>57.2</td>
</tr>
<tr>
<td>Women</td>
<td>(14)</td>
<td>(62.9)</td>
</tr>
<tr>
<td>Main stem</td>
<td>6</td>
<td>65.8</td>
</tr>
<tr>
<td>Branch</td>
<td>5</td>
<td>61.4</td>
</tr>
<tr>
<td>No fresh occlusion</td>
<td>3</td>
<td>60.0</td>
</tr>
<tr>
<td>No Shock</td>
<td>(59)</td>
<td>(63.0)</td>
</tr>
<tr>
<td>Men</td>
<td>26</td>
<td>62.3</td>
</tr>
<tr>
<td>Main stem</td>
<td>3</td>
<td>63.3</td>
</tr>
<tr>
<td>No fresh occlusion</td>
<td>30</td>
<td>63.5</td>
</tr>
<tr>
<td>Women</td>
<td>(22)</td>
<td>(63.3)</td>
</tr>
<tr>
<td>Main stem</td>
<td>9</td>
<td>64.8</td>
</tr>
<tr>
<td>Branch</td>
<td>1</td>
<td>53.0</td>
</tr>
<tr>
<td>No fresh occlusion</td>
<td>12</td>
<td>63.0</td>
</tr>
</tbody>
</table>

*Figure 1*

The incidence of shock in myocardial infarction in relation to the presence of main stem or branch occlusions.

*Figure 2*

The relation between the occurrence of shock and the anatomic location of coronary artery occlusion.
branch occlusions and others, but it is significant \((0.05 > p > 0.02)\). Furthermore, 10 of the 12 branch occlusions in patients with shock occurred in the distribution of the left circumflex coronary artery.

On the other hand, neither the size of the infarct nor the extent of involvement of the coronary arteries with arteriosclerotic plaques or old occlusions was significantly different when patients with and without shock were compared, as shown in figures 3 and 4. The group of patients with shock and branch occlusions is notable, however, in that seven of 12 (58 per cent) had grade-III infarcts, and 10 of 12 (84 per cent) were scored as 7 to 9 plus coronary artery involvement. Grade-III infarcts were found in 18 of 46 (39 per cent) patients with shock, and 25 of 46 (54 per cent) had 7 to 9 plus coronary involvement with atherosclerosis.

**Discussion**

Hemodynamic studies in patients with shock associated with acute myocardial infarction have failed to provide a clear picture of the relative roles of cardiac output and peripheral resistance in the hypotensive state.\(^{7-10}\) The number of patients in each study has been small, and the lack of uniform criteria for shock makes them difficult to evaluate. Freis et al.\(^7\) studied 11 patients with acute myocardial infarction in whom the lowest values for cardiac output and stroke volume and the highest peripheral resistances were found in the four patients with cardiogenic shock. Smith, Wilker, and Fox,\(^8\) however, found cardiac output reduced as much in three of 10 patients with acute myocardial infarction without shock as in seven patients with shock. In the latter group, total peripheral resistance was elevated in four subjects and unchanged in three. Similar variability is found in the studies of Gammel et al.\(^9\) and Gilbert et al.\(^10\)

Experimental studies of cardiogenic shock have been hampered by the lack of an adequate model. Ligation of coronary arteries in dogs is associated with a drop in cardiac output and an increased total peripheral resistance, but blood pressure is maintained above shock levels.\(^11\) The use of graded microspheres for coronary embolization by Agress et al.\(^3\) produced the first consistent model of experimental cardiogenic shock. Microspheres with a diameter of 190 \(\mu\), which occluded primary right angle branches rather than main stem vessels or more peripheral branches, were essential to the production of shock. Cardiac output and stroke volume were comparably reduced in those dogs that remained normotensive after embolization and in those that became hypotensive. Total peripheral resistance, however, rose markedly in the normotensive ones, but failed to rise in the animals with shock.\(^12\)
Certain animals, with low initial cardiac output and high total peripheral resistance, were more susceptible to hypotension induced by embolization than others. In some dogs, the peripheral resistance and blood pressure could be increased by thoracic spinal epidural block following coronary embolization. In this model, therefore, there is good evidence to support the hypothesis of a reflex mechanism for shock associated with acute myocardial infarction.

Postmortem studies in man have also suggested that size of the infarct cannot be the sole factor in the pathogenesis of cardiogenic shock. Gootnick and Knox emphasized that many hearts have been seen at autopsy with little functional myocardium left after successive infarcts, and yet shock was not part of the clinical picture. In a clinicopathologic study of a large series of patients with acute myocardial infarction, Malach and Rosenberg could find no difference between the groups with and without shock, with respect to the size or location of the acute infarct. A positive correlation however, was noted between shock and the presence of old infarction.

Our data indicate a high incidence of shock in patients dying of acute myocardial infarction with a fresh occlusion of a branch coronary artery. On the other hand, only a quarter of the patients with shock had branch occlusions. Clearly, if occlusion of a branch coronary artery plays a role in the pathogenesis of shock associated with acute myocardial infarction, it is not the only mechanism. Patients with main stem occlusions, or even with infarction without occlusion may also develop shock. Nevertheless, in the smaller group with branch occlusions, shock is a more frequent complication.

The argument for a reflex mechanism would be stronger if the patients with shock and branch occlusions were found to have had smaller infarcts than the others with shock. In fact, these patients had a preponderance of extensive infarcts. Large infarcts, however, are to be expected in a study limited to fatal myocardial infarctions. Patients with shock and branch occlusions also had a high degree of old coronary occlusive disease. One might speculate that these patients are physiologically similar to the dogs with low initial cardiac outputs and high total peripheral resistances, which Agress found to be more susceptible to shock induced by coronary embolization.

The association of shock with occlusions in the distribution of the left branch of the circumflex coronary artery is striking. If a shock producing reflex is involved, the receptor site may be located in primary branches of this area of the coronary circulation. Experiments in which microspheres were introduced into all three main stem coronary arteries at once have not shed light on this possibility. Guzman, Swenson, and Mitchell have recently studied the hemodynamic effects of selective embolization of the left anterior descending coronary artery with glass beads and lycopodium spores of 30 to 40 μ. They reported that embolization uniformly produced a drop in cardiac output and blood pressure, and a marked rise in total peripheral resistance. The hypotension was transient, however, and may not have been shock as defined in the studies of Agress. Similar studies, injecting microspheres of various sizes, after selective catheterization of other main stem coronary arteries are needed to clarify the role of local reflex receptors in cardiogenic shock.

The use of postmortem data to relate the location of fresh coronary occlusions to shock in acute myocardial infarction may be criticized on the basis that the branch occlusions might have been the result rather than the cause of shock. Blumgart et al. demonstrated that patients dying of shock caused by noncardiac conditions frequently had multiple coronary thromboses at autopsy. This finding was attributed to stasis, hypercoagulability, hypoxia, and subintimal hemorrhages induced by shock itself. Although other conditions capable of causing shock were excluded from the present study, it is conceivable that the branch occlusions were secondary to shock caused by infarction with

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main stem occlusion, or infarction without occlusion. Only three, however, of the branch occlusions in patients with shock had concomitant main stem occlusions, and in almost every instance the location of the branch occlusion correlated well with the location of the infarct. Nevertheless, this consideration cannot be entirely excluded from a postmortem study, and emphasizes the need for further experimental confirmation of our observations.

Summary and Conclusions
Postmortem and clinical data for 127 patients dying of acute myocardial infarction were correlated to determine the relationship between primary branch occlusions and cardiogenic shock. The relative frequency of shock was significantly greater in patients with branch occlusions than in those with main stem occlusions or infarction without fresh occlusion. Branch occlusions in the distribution of the left circumflex coronary artery had a particularly high association with shock. Patients with and without shock were not significantly different with respect to the size of the infarct and the extent of coronary atherosclerosis. Most of the patients with shock and branch occlusions had large infarcts and extensive coronary atherosclerosis.

A reflex mechanism, with receptor sites in the primary branches of the coronary arteries, may be implicated in the pathogenesis of shock in certain cases of acute myocardial infarction, as has been demonstrated in the experimental animal. Shock also occurs in patients with myocardial infarction who have had main stem occlusion, or no occlusion at all. Further studies of reflex factors in shock associated with myocardial infarction are suggested. A clearer understanding of these factors may have important therapeutic implications.

References

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