Management of Shock in Acute Myocardial Infarction

By Abraham Gootnick, M.D., and Frederick H. Knox, Jr., M.D.

Shock complicating acute myocardial infarction contributes materially to the over-all mortality. The wisdom of combating the shock has been a matter of dispute, many clinicians holding that the fall in blood pressure has the salutary effect of reducing the work of an acutely injured heart. This report deals with a four-year experience in active intervention for shock. The data suggest that timely intervention for shock is frequently life-saving, and may be instrumental in halving the mortality of the sickest patients.

The arguments for and against vigorous blood replacement in cases of bleeding peptic ulcer have had their counterpart in the question how best to treat shock associated with myocardial infarction. In the ulcer controversy, misgivings have been voiced lest restoration of normal blood pressure “blow off the clot” and undo the salutary homeostatic effects of a fall in blood pressure; similarly, in the management of myocardial infarction, the fall in blood pressure is regarded by many as an adaptive mechanism for reducing the load on an acutely injured myocardium. Current teaching, as reflected in texts on diseases of the heart, yields no clear answer to the question: Should the shock of acute myocardial infarction be treated or let alone? White, in his chapter on myocardial infarction, does not mention the treatment of shock. Friedberg states: “Therapeutic measures ordinarily employed in other forms of shock may be dangerous, lest they overload the circulation, or strain the injured heart muscle.” Of several widely used textbooks of medicine only one includes in the treatment of acute myocardial infarction a section on management of shock, and advocates the use of vasopressor drugs and blood or plasma.

Stroud states: “... perhaps a drop in blood pressure is an effort of the body’s part to protect the myocardium.” Gilbert states: “... I see no necessity for giving plasma or for transfusion. There are contraindications to the use of peripheral vasoconstrictor drugs. Blood pressure should be left where it is and not tinkered with.”

The issue has been clouded by inadequate definition of terms. A fall in blood pressure of some degree occurs in the large majority of cases of myocardial infarction. In most of these, the drop in blood pressure occurs at onset or shortly thereafter, is of moderate degree, associated with only minor indications of vascular collapse, and (most important) is transitory, with spontaneous recovery in the direction of normal blood pressure within minutes or an hour or two. Experience with patients whose blood pressure behaves in this way has been very favorable and has served as a deterrent against intervention for shock. In contrast, there are other patients whose fall in blood pressure is of marked degree, who show the full-blown picture of shock and who remain in this state for many hours with a steadily deteriorating circulation until death. The gravity of this type of shock is attested to by every observer who has studied a large number of patients with acute myocardial infarction. A fall in systolic blood pressure below 90 mm. Hg, or of the pulse pressure below 25 mm. Hg places the patient in a category in which recovery is uncommon. The experience may be summarized in the conclusion of Master and his co-workers:
“When the blood pressure fell below 80, the patient usually died.”

Although there are numerous recorded observations on the level of blood pressure in myocardial infarction, and on the prognostic implications of various degrees of hypotension, references to duration of shock are few and less explicit. It has been our observation that duration of shock is the more important prognostic criterion. When profound circulatory failure persists for a number of hours, recovery is extremely rare. This is understandable in the light of Wiggers’ experimental studies on hemorrhagic and postreinfusion shock.9 When, after prolonged posthemorrhagic hypotension, blood is reinfused, the animal rallies only temporarily; “... slow spontaneous circulatory failure and eventually death follow. Irreversibility develops during the period of prolonged hypotension ... myocardial depression is indicated by a subminimal stroke volume when venous pressures are elevated to normal levels ...” Other experimental studies on shock show that myocardial lesions were consistently found in dogs who survived longer than eight hours.10 Clinically, moreover, the effect of protracted shock on the coronary circulation has long been recognized, notably in the report of Blumgart and co-workers.11

Early studies by Fishberg, Hitzig and King presented evidence to show that the circulatory failure associated with acute myocardial infarction is not an entity separate from other varieties of shock, and that in common with other types of shock it is characterized by marked decrease in venous return, diminished circulating volume, and lowered venous pressure.12 Later, Stead and Ebert observed in patients with myocardial infarction simultaneous diminution of peripheral blood flow and congestion of the pulmonary or systemic circulation. They concluded that this combination of events was a reflection of primary failure of the heart rather than curtailment of venous return characteristic of other varieties of shock.13 The controversial role of reflex factors in the circulatory collapse, and the probability that abrupt diminution in cardiac output may well be the significant initial event, do not affect the central consideration: Prolonged critical lowering of aortic pressure subjects an already injured myocardium to greatly diminished perfusion with blood. The well established relationship of extensive myocardial necrosis to severe lasting shock has reconciled physicians to the view that such degrees of myocardial injury are incompatible with life, and that exertions against drastic shock are not likely, therefore, to alter a fatal outcome. Nevertheless, it cannot be that mere massiveness of the infarction foredooms a patient; there are patients who have survived a third and fourth authenticated myocardial infarction and who eventually come to autopsy showing that they had lived on with remarkably little intact myocardium. The problem, therefore, is tiding the patient over the acute functional derangement of the circulation precipitated by the myocardial injury.

In our experience with acute myocardial infarction during the years 1948 to 1951, we found 32 instances of acute myocardial infarction marked by severe and prolonged shock, who were treated actively with one or another combination of plasma, blood and a vasoressor drug. For purposes of this study, we have eliminated cases of shock who were treated but in whom there was significant possibility of spontaneous recovery. Those excluded from this study were patients who exhibited no more than moderate clinical manifestations of circulatory collapse, or whose pulse pressure did not fall below 25 mm. Hg; those who, though definitely in shock, were in circumstances which permitted prompt treatment and reversal of the shock; and those whose shock, of whatever duration, was quickly responsive to initial therapeutic measures. We have included only patients whose shock was so profound at the beginning of treatment as to justify no expectation of survival. The data in table 1, and the representative case abstracts which follow, are intended to make clear our basis of selection. This is a group of patients in whom from previous experience we should have expected a mortality close to 100 per cent.

For vasoressor effect we depended on drugs which raise blood pressure without accelerating the heart, namely Paredrine, Neosynephrine...
### TABLE 1.—Clinical Findings, Treatment Used and Results in 32 Cases of Severe Shock Following Myocardial Infarction

<table>
<thead>
<tr>
<th>Name, Age &amp; Diagnosis</th>
<th>Hours In Shock Before Treatment</th>
<th>Clinical Manifestations</th>
<th>Total Hours Treatment of Shock</th>
<th>Treatment and Effects</th>
<th>Remarks</th>
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<tbody>
<tr>
<td>D. E., w. m., 58.</td>
<td>2</td>
<td>Fall of B.P. from 100/80 to 0 with onset of ventricular tachycardia. In coma; incontinent.</td>
<td>26</td>
<td>Sinus rhythm after 1.5 Gm. Pronestyl; shock persisted. After 6 hours of treatment with Neosynephrine and plasma B.P. 92/75; patient improved. Recurrence of ventricular tachycardia and shock. No further response. Total Neosynephrine 80 mg. Total plasma 1500 cc.</td>
<td>Died</td>
</tr>
<tr>
<td>G. J. B., w. m., 74; one previous myocardial infarction.</td>
<td>4</td>
<td>Moribund on admission, B.P. not perceptible.</td>
<td>1</td>
<td>300 cc. of plasma; 26 mg. of Neosynephrine. No response.</td>
<td>Died 1 hour after admission.</td>
</tr>
<tr>
<td>G. J. K., w. m., 55; one previous myocardial infarction.</td>
<td>25</td>
<td>B.P. and pulse not perceptible; deep cyanosis; coma.</td>
<td>2</td>
<td>Plasma 550 cc. Neosynephrine 48 mg. No response. Dead in two hours.</td>
<td>Old and fresh infarctions at autopsy.</td>
</tr>
<tr>
<td>C. H., Negro male, 54.</td>
<td>2</td>
<td>Fall in B.P. from 150/120 to 80/40. Profuse cold sweat, cyanosis, Cheyne-Stokes respiration.</td>
<td>27</td>
<td>Fluctuating B.P. with rise up to 110/80 after each dose of Neosynephrine. Pulmonary edema cleared as B.P. rose. Plasma 1250 cc. Neosynephrine 139 mg.</td>
<td>B.P. on discharge 110/80.</td>
</tr>
<tr>
<td>C. A. J., w. m., 55.</td>
<td>1</td>
<td>Fall in B.P. from 170/110 to 0. Severe chest pain, vomiting, cyanosis.</td>
<td>23</td>
<td>1000 cc. of 10% glucose sol. 500 cc. blood. Total Neosynephrine 108 mg. Moderate pulmonary edema cleared with emergence from shock.</td>
<td>Recovered.</td>
</tr>
<tr>
<td>T. A. E., w. m., 61.</td>
<td>Unknown</td>
<td>Cyanotic, covered with cold sweat, pulmonary edema.</td>
<td>3</td>
<td>Pulmonary edema cleared after I.V. morphine. Highest systolic B.P. 60 mm. Hg. Remained in shock until death. Total Neosynephrine 43 mg. No blood or plasma.</td>
<td>Autopsy: Large area of myocardial necrosis and pericardial hemorrhage.</td>
</tr>
<tr>
<td>B. T. E., w. m., 52; two previous strokes.</td>
<td>2</td>
<td>Fall in B.P. from 190/110 to 0; severe pulmonary edema.</td>
<td>2</td>
<td>35 mg. of Neosynephrine ineffective. Remained in shock until death.</td>
<td>Shock precipitated by third cerebrovascular accident.</td>
</tr>
<tr>
<td>F. J. Q., w. m., 81; two previous infarctions.</td>
<td>5</td>
<td>In coma, incontinent, clammy, cyanotic. Systolic B.P. 50 mm. Hg by palpation.</td>
<td>11</td>
<td>Plasma 750 cc., Neosynephrine 68 mg. Highest B.P. reached 80/65. Remained in shock.</td>
<td>Died</td>
</tr>
<tr>
<td>L. G., w. m., 54; one previous infarction.</td>
<td>2</td>
<td>B.P. 0, cold, cyanotic, vomiting. Increasing pulmonary edema on second day of shock.</td>
<td>26</td>
<td>Plasma 500 cc., blood 500 cc in first 12 hours. Out of shock after 16 hrs., but required Neosynephrine for 10 hrs. more. Total 92 mg.</td>
<td>Discharged Symptom-free with B.P. 115/70.</td>
</tr>
<tr>
<td>S. A. L., w. m., 53.</td>
<td>29</td>
<td>Cyanotic, stuporous, incontinent. B.P. 70/60.</td>
<td>2</td>
<td>Plasma 450 cc. Neosynephrine 28 mg. No response.</td>
<td>Autopsy: Infarction of entire septum and most of anterior wall.</td>
</tr>
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TABLE 1.—Continued

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<tr>
<td>L. A. A., w. m., 61; one previous infarction.</td>
<td>5</td>
<td>Fall in B.P. from 190/110 to 0. Pulmonary edema, stuporous.</td>
<td>1½</td>
<td>Initial response to Neosynephrine; B.P. up to 90/70 and pulmonary edema cleared. Chest pain recurred, B.P. fell to 0, with pulmonary edema. No response thereafter. Neosynephrine 25 mg.</td>
<td>Autopsy: Old posterior infarction &amp; fresh extensive anterior infarction.</td>
</tr>
<tr>
<td>W. W. R., w. m., 63; one previous infarction.</td>
<td>48; severe shock 6 hrs.</td>
<td>B.P. 70/60, cold, clammy, cyanotic, in marked congestive failure.</td>
<td>14</td>
<td>Initial improvement with rise in B.P. to 80/50. Severe shock thereafter. Plasma 1000 cc. Neosynephrine 63 mg.</td>
<td>Autopsy: Old posterior, fresh anterior infarction.</td>
</tr>
<tr>
<td>R. J. F., w. m., 51.</td>
<td>6</td>
<td>Stuporous, clammy, cyanotic, vomiting. B.P. 0.</td>
<td>48</td>
<td>B.P. 92/60 after 5 hrs. of treatment but shock recurred when Neosynephrine withheld. After 48 hrs. patient alert, warm, voiding. B.P. 102/54. Plasma 1000 cc. Neosynephrine 105 mg.</td>
<td>Initial response to drug alone inadequate; effect of adding plasma dramatic.</td>
</tr>
<tr>
<td>P. S., w.m., 74; one previous infarction.</td>
<td>½</td>
<td>Markedly cyanotic, cold and wet, vomiting, Cheyne-Stokes breathing. Systolic B.P. 50 mm Hg.</td>
<td>4½</td>
<td>Transient response with peak of B.P. 110/95. Thereafter unresponsive. Plasma 500 cc. Neosynephrine 27 mg.</td>
<td>Autopsy: Old posterior and fresh anterior infarctions; bronchial tree filled with aspirated gastric content.</td>
</tr>
<tr>
<td>R. J. H., w. m., 60; two previous myocardial infarcts.</td>
<td>25</td>
<td>B.P. 0, cold, in coma.</td>
<td>4½</td>
<td>Systolic B.P. reached 60 mm. Hg. briefly. Never out of shock. Plasma 750 cc. Neosynephrine 29 mg.</td>
<td>Autopsy: Two previous infarctions, one fresh infarction.</td>
</tr>
<tr>
<td>P. T. B., w. m., 48.</td>
<td>½</td>
<td>Bilateral bronchopneumonia, marked cyanosis, rapid, shallow respirations, B.P. 80/68.</td>
<td>24</td>
<td>B.P. rose to 88/72, 94/72. Antibiotics, anticoagulants, Neosynephrine 44 mg. No I.V. fluids. Improvement interrupted by cerebrovascular accident.</td>
<td>Autopsy: Extensive anterolateral infarction; cerebral thrombosis.</td>
</tr>
<tr>
<td>C. J., w. m., 51; one previous infarction.</td>
<td>24</td>
<td>B.P. 84/50. In marked pulmonary edema, cyanotic, stuporous.</td>
<td>6</td>
<td>Initial response, B.P. to 98/64 and clearing of pulmonary edema. Later, recurrence of intractable pulmonary edema and death. Neosynephrine 25 mg., no I.V. fluids.</td>
<td>Autopsy: Previous anterior and fresh posterior infarctions. Marked pulmonary edema.</td>
</tr>
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<tr>
<td>G. C. M., w. m., 56; one previous infarction.</td>
<td>9</td>
<td>Moribund; B.P. 0</td>
<td>8½</td>
<td>Plasma 500 cc. Blood 500 cc. Neosynephrine 42 mg. No response.</td>
<td>Died</td>
</tr>
<tr>
<td>G. A. C., w. m., 70; one previous infarction.</td>
<td>5</td>
<td>Stuporous, clammy, cyanotic, vomiting. Fall in B.P. from 210/120 to 60/?.</td>
<td>22</td>
<td>B.P. rose to 80/64, fell again between doses of Neosynephrine. Slow emergence from shock. Plasma 500 cc. Blood 750 cc. Neosynephrine 69 mg. B.P. rose to 96/72. Pulmonary edema cleared. Blood 750 cc. Neosynephrine 48 mg.</td>
<td>Discharged with B.P. 124/90.</td>
</tr>
<tr>
<td>M. D. F., w. m., 55.</td>
<td>4</td>
<td>Cold and sweating, cyanotic. B.P. 78/50.</td>
<td>44</td>
<td>First rise in B.P. to 80/50 only after plasma started. Blood 500 cc. Plasma 500 cc. Neosynephrine 38 mg. Plasma 1500 cc. Neosynephrine 52 mg. No response.</td>
<td>Discharged on discharge 120/80.</td>
</tr>
<tr>
<td>A. B. J., w. m., 60; one previous infarction.</td>
<td>3</td>
<td>Cold, cyanotic, in coma, incontinent. B.P. 60/?.</td>
<td>15</td>
<td>B.P. rose to 80/60 then fell to 0. Increasing pulmonary edema till death. Plasma 500 cc. Neosynephrine 48 mg.</td>
<td>Discharged posterior and fresh anterior infarctions.</td>
</tr>
<tr>
<td>M. R., w. m., 60; two previous infarctions.</td>
<td>29</td>
<td>B.P. 65/50. Cold, wet, anuric, vomiting.</td>
<td>7</td>
<td>Autopsy: Old myocardial infarction and extensive fresh infarction.</td>
<td>Died</td>
</tr>
<tr>
<td>W. E. O., w. m., 60; one previous infarction.</td>
<td>1</td>
<td>B.P. 0. Vomiting, cyanotic and cold, stuporous.</td>
<td>16½</td>
<td></td>
<td></td>
</tr>
<tr>
<td>A. B. J., w. m., 61; three previous infarctions.</td>
<td>½</td>
<td>B.P. 65/58, profuse cold sweat, cyanotic, vomiting.</td>
<td>14½</td>
<td></td>
<td></td>
</tr>
<tr>
<td>R. J. B., w. m., 42; one previous infarction.</td>
<td>8</td>
<td>Coma, marked cyanosis, respirations 8 per minute, B.P. 0.</td>
<td>10½</td>
<td></td>
<td></td>
</tr>
<tr>
<td>S. G., w. m., 62.</td>
<td>3</td>
<td>Fall in B.P. from 200/140 to systolic of 100 by palpation. Cyanosis, intractable hiccup, marked pulmonary edema.</td>
<td>19</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
Oxygen and Demerol or morphine were received by all patients and are not referred to in the table. Most patients received anticoagulants, but in the majority the shock occurred too early in the course of the illness for this treatment to be relevant.

Digitalis was administered to the following patients: R. J. F. (second admission), L. G., C. J. J., S. G., G. J. K., B. T. E. and W. W. R.

The following received mercurial diuretics: R. J. F. (second admission), H. J. M., A. B. J., and W. W. R.

Most patients received some 5 per cent or 10 per cent glucose solution. Only C. A. J. received an effective quantity.

* B.P. imperceptible.

and norepinephrine. However, none of the patients treated with Paredrine was sufficiently in shock to qualify for inclusion in this study. The patients treated with norepinephrine are the subjects of a separate investigation to be reported in the future. The present group, therefore, were all treated with Neosynephrine, administered intravenously or intramuscularly, in doses varying from 2 mg. to 7 mg., and at intervals of 15 minutes to an hour. The size of the dose, route of administration, and intervals between doses were determined by the initial status of the patient, the presence or absence of sufficient circulation to absorb the drug from the tissues, and adequate or inadequate response to previous doses of the drug. Plasma or whole blood was infused in quantities varying from 250 cc. to 1500 cc. Only outright pulmonary edema complicating the shock was considered a contraindication to the use of blood or plasma.

Treatment in other respects was that accorded to most patients with myocardial infarction. Morphine or Demerol was given in doses sufficient to control pain. All patients included in this study received oxygen, either in a tent or by nasal catheter. Some of the patients, whose course was marked by increasing pulmonary congestion, received a mercurial diuretic, and several were digitalized. With the exception of those who presented a decisive contraindication, all received adequately controlled anticoagulant therapy.

**Case Abstracts**

R. J. F. was a 51 year old white man who had collapsed abruptly and was found three hours later barely conscious and complaining of agonizing anterior midchest pain. When brought to the hospital six and one-half hours after onset, the patient appeared moribund. Heart rate was 40 and regular; blood pressure could not be determined; temperature was 96.6 F. The heart sounds were barely audible. The patient was cyanotic and semistuporous; his skin was covered with perspiration and cold. He had received no medication prior to admission. The electrocardiogram showed a pattern typical of acute posterior wall infarction.

The patient was placed in an oxygen tent. An infusion of 5 per cent glucose in water was started at once and replaced by plasma within 15 minutes. Five milligrams of Neosynephrine were administered intravenously and another 5 mg. intramuscularly. Fifty milligrams of Demerol were given subcutaneously. Within an hour his blood pressure was 60/40. Repeated 5 mg. doses of Neosynephrine were given intravenously at intervals varying from 20 to 40 minutes until the blood pressure rose to 86/72.
At the end of five hours of treatment, in the course of which 55 mg. of Neosynephrine and 1000 cc. of plasma had been administered, the blood pressure was 92/60. The skin was less cyanotic, he was conscious and no longer in pain. Treatment with Neosynephrine and plasma was discontinued. An hour later he was again covered with cold perspiration, blue, and blood pressure had dropped to 88/72. Neosynephrine was resumed and the blood pressure again rose to 96/66 within 15 minutes of the first 5 mg. dose. Thereafter, the drug was continued in 3 mg. to 5 mg. doses at progressively lengthening intervals for a total of 48 hours. At the end of this time the patient's blood pressure was 102/54, the first time systolic pressure had exceeded 100 mm. Hg. One thousand cubic centimeters of plasma was administered, all of it within the first five hours after admission. The total quantity of Neosynephrine given was 105 mg.

Five weeks after admission anticoagulant therapy had been discontinued and the patient was discharged, symptom-free, with a blood pressure averaging 105/70.

Six months after his first myocardial infarction, this patient was admitted a second time approximately 12 hours following onset of severe chest pain. The patient was in coma, cold and blue, pulseless, and exhibiting Cheyne-Stokes respiration. Blood pressure was unobtainable. Heart sounds were faintly audible, regular, approximately 140 per minute. The electrocardiogram showed a pattern of acute anterolateral infarction.

The patient was placed in an oxygen tent, 7 mg. of Neosynephrine were administered intravenously and an infusion of 5 per cent glucose in water was started. Plasma was substituted for the glucose solution 10 minutes later. Within 90 minutes blood pressure was 86/74. Neosynephrine was again given, 5 mg. intravenously and 5 mg. intramuscularly, and 5 mg. doses of the drug were repeated at intervals averaging one-half hour. In the course of the succeeding 12 hours, blood pressure readings were 86/74, 80/62, 88/70, and 90/74. On two occasions, the systolic blood pressure remained above 90 mm. Hg for 90 minutes before dropping again. The patient's color improved gradually and periodic breathing became regular. He regained consciousness 12 hours after admission and complained of anterior mid-chest pain; this was controllable with 10 mg. of morphine sulfate given subcutaneously. The blood pressure at this time was 102/75. During this 12 hour period he had received 102 mg. of Neosynephrine and 750 cc. of plasma.

The patient's course from this point on was marked by steadily increasing pulmonary congestion which was controlled with some difficulty by the use of mercurial diuretics, augmented later by digitalis. Eventually, he was free of congestive failure on a regimen including low-sodium diet, maintenance doses of digitalis, and a weekly dose of a mercurial diuretic. Blood pressure at the time of discharge was 100/76.

C. A. J., a 55 year old white male known to be hypertensive, was admitted with complaints of epigastric and midchest pain of seven days' duration, associated with much eructation of gas, anorexia and abdominal distention. Acute myocardial injury was suspected and confirmed by an electrocardiogram which showed the pattern of posterior infarction. Treatment, including anticoagulant therapy, was begun. During the succeeding five days the patient complained of intermittent pain of considerable severity in the anterior chest. Low-grade fever continued, and the blood pressure which had been 170/100 on admission, now averaged 120/85.

On the morning of the sixth hospital day, the patient was seized with severe retrosternal pain, was nauseated and vomited once. Within an hour, blood pressure had become imperceptible, heart sounds were feeble, and heart rate was 140 and regular. The patient had become ashen-gray; profuse, cold perspiration covered the entire body; he was restless and markedly apprehensive. He was placed in an oxygen tent and 10 mg. of morphine sulfate were given intravenously. A slow drip of 10 per cent glucose in water was started and a first dose of 5 mg. of Neosynephrine was given intravenously. Additional 5 mg. doses of Neosynephrine were given before a systolic blood pressure level of approximately 70 mm. Hg. could be detected by palpation. An electrocardiogram taken at this point, two and one-half hours after onset of the acute pain, showed changes consistent with fresh extension of the myocardial infarction. The patient remained conscious, but was cyanosed, cold, vomited repeatedly, and was clearly in deep shock. During the succeeding 10 hours, 1000 cc. of 5 per cent glucose in water, given intravenously, 500 cc. of whole blood, and repeated intravenous doses of Neosynephrine had the effect of maintaining a blood pressure averaging 70/50. Several observers who left notes on the patient's chart unanimously considered his prognosis hopeless.

Rales could now be heard over the lung bases which had been clear initially, but there was no overt pulmonary edema. Treatment continued with only repeated doses of Neosynephrine; intravenous fluids were omitted. Intermittent peaks of systolic blood pressure were recorded at 88, 94 and 92 mm. Hg, shortly after a dose of Neosynephrine, but these levels were not sustained. During the next 12 hours the patient very slowly emerged from shock. The blood pressure rose to 95 systolic and finally, 24 hours after onset of his circulatory collapse, blood pressure was 110/90 and the patient was out of shock. A total of 108 mg. of Neosynephrine had been administered. Serial electrocardiograms were characteristic of fresh extension of the posterior myocardial infarction, showing reappearance of positive S-T
deviation in leads which previously had shown only the T-wave changes of a healing injury.

C. H., a 54 year old Negro, had been a known hypertensive for eight years. The background included a stroke six years previously and an intermittently active duodenal ulcer first diagnosed four years previously. The patient had been on maintenance doses of digitalis for congestive failure which had begun one and one-half years prior to admission. The day before admission, the patient was seized with severe pain in the left chest radiating from the left axilla to mid sternum. With this there was marked exacerbation of his dyspnea, and great weakness. Physical examination 24 hours after onset of the pain showed an acutely ill, dyspneic, heavily perspiring Negro male. Blood pressure was 150/120, temperature 99, respiration 24; heart rate was 90 and regular. No signs of pulmonary or visceral congestion were evident, and the only other notable finding was old right hemiplegia. Electrocardiogram showed a typical pattern of acute posterior infarction and an occasional ventricular premature contraction.

On the third night after the infarction, following an initially favorable course, the patient grew increasingly dyspneic, had recurrence of chest pain, and perspired profusely. When seen two hours later, his blood pressure had fallen to 80/40 and he was in pulmonary edema. The patient was placed in an oxygen tent, a single 10 mg. dose of morphine sulfate was given intravenously and Neosynephrine was given in repeated 5 mg. doses intravenously at 15 to 20 minute intervals. The blood pressure rose intermittently in response to the drug, and the pulmonary edema cleared within an hour. Blood pressure thereafter fluctuated between 76/56 and 100/70, with one briefly sustained peak of 160/110. Twenty-four hours after onset of the shock the patient was still in a state of circulatory collapse, cyanotic, bathed in clammy perspiration and showed Cheyne-Stokes breathing. The pulmonary edema had not recurred. His blood pressure was sustained at the levels indicated but lapsed within one-half hour after each intravenous dose of Neosynephrine. A slow infusion of plasma was started and from this point on effectiveness of Neosynephrine was much more lasting. Twenty-nine hours after onset of shock, following a total of 139 mg. of Neosynephrine and 1250 cc. of plasma, the patient was warm, voiding, breathing easily, and able to take food. Blood pressure was 106/80 and remained stable without further medication.

Careful study failed to reveal evidence of a fresh cerebrovascular accident, of bleeding from his ulcer or any other source, or of any extracardiac reason for the shock. The patient was discharged with a blood pressure of 110/80 and did not thereafter regain hypertensive levels. Two years later a follow-up examination showed a blood pressure 140/90.

G. J. K. was a 55 year old white man whose past history included diabetes mellitus of eight years' duration and a myocardial infarction one year previously. He was on a diabetic regimen which included neutral-protamin Hagedorn insulin, 40 units daily, and he took a daily maintenance dose of Digoxin. Five days prior to admission he was seized with severe retrosternal pain, became very weak and vomited. The pain recurred intermittently thereafter, and on the day of admission the pain grew much worse and it was associated with great weakness and dyspnea. On admission blood pressure was 120/80, the heart was moderately enlarged and a grade II systolic murmur was heard over the entire precordium. The only other finding of note was moderate dependent edema of the lower extremities. The electrocardiogram showed a pattern of recent extensive anterior infarction, superimposed on a previous posterior infarction.

The patient's course was uneventful until two weeks after onset of his infarction, when he again complained of severe chest pain. He developed a protodiastolic gallop rhythm, and the electrocardiogram showed changes consistent with extension of the myocardial injury. The patient was weak and vomited repeatedly. The skin was cyanotic and clammy. Blood pressure was 106/90. Despite the marked deterioration of the patient, antishock therapy was withheld, apparently because the systolic blood pressure exceeded 100 mm. of Hg. A check of the blood sugar showed no hypoglycemia. The following morning, 24 hours after his abrupt change for the worse, the patient was moribund. He was stuporous, cold, without perceptible pulse or blood pressure, and was blue despite the administration of oxygen. Plasma and intravenous Neosynephrine were administered but with no effect, and the patient died after two hours of intensive treatment which included 48 mg. of Neosynephrine and 550 cc. of plasma.

Autopsy confirmed the electrocardiographic interpretation of an old posterior and extensive acute anterior myocardial infarction.

P. S., a senile, incoherent, 74 year old white man, was admitted seven days after the onset of severe chest pain. He had vomited repeatedly following the onset of his pain. Except for moderate dyspnea the patient was not in distress at the time of admission. Significant physical findings included rales over the left chest posteriorly and faint heart sounds. Blood pressure was 110/80; temperature 100.2 F. and respirations 24. Electrocardiogram showed complete atrioventricular block, a ventricular rate of 55 and a pattern of recent extensive anterior infarction.

Twelve hours after admission the patient went into shock abruptly. Blood pressure fell to 80/60, he became stuporous, and developed Cheyne-Stokes respiration. Rales and rhonchi were audible over both lung bases posteriorly. Repeated intravenous
doses of Neosynephrine and 500 cc. of plasma resulted in an intermittent rise in blood pressure to 90/70 and at one point to 110/85, but the patient remained cyanotic, stumpy and incontinent. He received 500 cc. of plasma and 27 mg. of Neosynephrine but failed to rally and died five hours after lapsing into shock. At autopsy, an old posterior infarction and an extensive anterior infarction were found. The entire bronchial system on the left was filled with aspirated gastric contents, and there were many areas of pulmonary atelectasis.

H. J. M., a 47 year old white man, was admitted two weeks after onset of severe mid-chest pain, diagnosed at another hospital as acute anterior myocardial infarction. Electrocardiograms were in keeping with this diagnosis. The patient was asymptomatic on admission, presented no significant abnormalities on physical examination, and did well on a regimen of restricted activity and anticoagulant therapy. During the fourth week in hospital, increasing dyspnea developed, the heart rose to 120 per minute, the liver became enlarged and tender, and congestive changes were detectable in the basins of the lungs. Response to a mercurial diuretic was satisfactory. The blood pressure was 105/85. Despite the absence of pain, the reappearance of leukocytosis, of low grade fever, and characteristic S-T segment changes in the precordial leads led to the diagnosis of fresh infarction six weeks after the initial one. The following day the patient was seized with severe chest pain which responded to a single 10 mg. dose of morphine. His course for a week thereafter was uneventful, and serial electrocardiograms showed the expected evolution of an acute anterior myocardial infarction. On the eighth day after onset of the fresh infarction, the patient vomited his breakfast and complained of sudden weakness. Ventricular tachycardia at a rate of 190 was found. This responded readily to a total dose of 2 Gm. of quinidine sulfate, but the patient soon lapsed into deep shock. He had reverted to sinus rhythm, and the heart rate varied between 100 and 120. The blood pressure, which had been 105/75, was now imperceptible. Repeated 5 mg. doses of Neosynephrine intravenously and intramuscularly, and 500 cc. of plasma resulted in a rise of blood pressure to 90/80, but 18 hours after onset the patient was still in marked circulatory collapse, cyanotic, oliguric, exhibiting Cheyne-Stokes respiration and increasing pulmonary congestion. Prognosis at this point was considered practically hopeless. Within the next six hours, he gradually improved on a regimen of plasma and Neosynephrine, the lung cleared progressively, his color became normal, he stopped vomiting and began to void. Blood pressure now was 104/86, 90/86. The total duration of shock was 34 hours. He had received a total of 108 mg. of Neosynephrine and 1250 cc. of plasma.

The patient had a slow convalescence, but finally did well and was returned to his sedentary job on a maintenance dosage of digitalis. The blood pressure on discharge was 125/80.

**Results**

The abstracted case histories presented are representative of the histories of the entire group of 32 patients. Fourteen of these 32 patients recovered from their shock, and 18 died.

The patients with myocardial infarction associated with severe shock were of the same age distribution as the entire group with acute myocardial infarctions. The average age of the 14 survivors was 55 years, of the 18 who died, 60 years. Only 11 of our 32 patients were undergoing their first myocardial infarction. Twenty had one or two previous infarctions, and one had survived three well-documented previous infarctions. Of the 11 with shock complicating an initial infarction, two out of three survived the acute attack; of the 21 who had had previous infarctions, only one out of three survived the acute attack.

An important factor bearing on the outcome of shock therapy was the length of time the patient had remained in deep shock before treatment was instituted. None of our patients survived whose shock had lasted more than eight hours without treatment.

Half of those who failed to recover went into shock at the onset of the acute attack; the rest went into shock at a later time within the first eight days following infarction. Among the survivors, all but two developed shock after a delay of one or several days, so that most were already in the hospital and within reach of prompt intervention.

In the large majority (26 of 32 cases), shock was precipitated by the myocardial infarction proper. In these 26 patients, there was no manifestation of complicating thromboembolic developments, of acute blood loss, of infection, or of significant change in the cardiac mechanism. In two, shock was precipitated by ventricular tachycardia. In one of these two the shock persisted for many hours after the rhythm had reverted to normal; he emerged from shock after 34 hours. In the other, ventricular tachycardia could not be controlled and this patient's shock persisted until death.
Only two developed shock from thromboembolic complications; a stroke in one patient who had had two strokes previously, and multiple cerebral and renal infarctions in another. One patient went into shock from hemopericardium and tamponade, and one from massive pulmonary atelectasis and infection.

In all 32 patients, evidence of acute myocardial infarction was unequivocal electrocardiographically, and in the 15 patients who came to autopsy the predicted infarctions, recent and old, were found without exception.

The use of Neosynephrine was not associated with detectable alteration of the cardiac mechanism. In those patients whose infarction was manifested by premature contractions or runs of ectopic rhythm, or by altered atrioventricular conduction, the addition of Neosynephrine to the regimen appeared to have no effect on the duration or severity of the disturbance. In only one patient with initially regular sinus rhythm did several supraventricular premature contractions appear in the course of Neosynephrine therapy; however, these premature contractions disappeared within 15 minutes despite continued administration of the drug, and it was felt that Neosynephrine was not the cause.

Except for terminal unresponsiveness in the patients who died, the vasopressor effect of Neosynephrine persisted with repeated doses given over the course of many hours. However, neither the drug alone nor intravenous infusion alone, whether of blood or plasma, was as effective as the two used together. It was repeatedly observed that plasma intravenously might fail to raise the blood pressure appreciably until followed by Neosynephrine. In the same patient, when the blood pressure had again fallen, the pressor effect of a dose of Neosynephrine alone might be negligible but would be augmented noticeably when followed by blood or plasma.

We were unable to detect any differences between the effects of plasma and whole blood. In several cases hematocrit determinations were made, and these were uniformly above normal. The significant advantage of plasma was its prompt availability, without the time lag involved in typing and cross matching blood.

Survival occurred, in some of our patients, after a remarkably long period of profound circulatory collapse. It was not unusual for a patient who had been in deep shock for a number of hours, and moribund on admission, to respond to treatment partially, and then continue, barely alive, at a blood pressure level of perhaps 80/65, for 24 or 36 hours. One required 48 hours of treatment before definitely emerging from shock. The effect of shock treatment in these patients was made evident in many instances by the repeated fall of blood pressure to imperceptible levels shortly after an infusion of blood or plasma had ended or when a dose of Neosynephrine was too long withheld. Among those who died, the majority showed no response at all to treatment, lapsed into progressively severe shock and died in a short time. Several, however, responded partially and lived many hours in a state of shock before they died. We have come to regard patients with acute myocardial infarction, however severe their shock, as having a chance for life. The chance is less the longer they have been in shock before treatment is begun, and it improves progressively the longer they remain alive under treatment.

Pulmonary edema complicating the shock occurred in 11 patients, and of these four survived. This combination is a particularly unfavorable one. For one thing, it reflects myocardial injury of extreme severity. For another, much of the therapy directed against pulmonary edema is intended to reduce venous return to the pulmonary circulation, and whatever reduces venous return reduces cardiac output also, and aggravates the shock. The number of patients treated is obviously too small for final conclusions, but our experience suggests that in choosing between the two coexisting problems it is more rewarding to place emphasis on combating the shock. The four survivors received no therapy directed toward the pulmonary edema other than small doses of morphine. They showed impressive clearing of the lungs as the blood pressure rose and they emerged from shock. In other instances tourniquets to the extremities, oxygen
by pressure mask, and rapid digitalization failed to influence the pulmonary edema favorably. The patients remained in shock and died in a short time. None of the patients in this group were among those we have treated with ethyl alcohol inhalation. Added to the favorable results we have observed with this method is the theoretical consideration that the improvement in respiratory function is not bought at the price of reduction in venous return.

Repeatedly, we encountered the sequence of severe shock leading to pulmonary edema as the terminal event. It would appear that pulmonary edema in such cases may not be simply a matter of "backward failure" but a late effect, reflexly induced, of extreme myocardial and cerebral anoxia. The favorable effect of correcting shock on the pulmonary edema suggests that the decisive factor is increased perfusion of the coronary and cerebral circulations.

The relationship of blood pressure levels to clinical status was only crudely consistent. While it is true that generally a patient whose systolic blood pressure rose to 100 mm. Hg could be expected to recover, there were exceptions in whom a systolic blood pressure of 100 mm. Hg or slightly higher was recorded while all the clinical manifestations of critical shock continued and the patient went on to die. The pulse pressure was more consistently accurate as an index of the circulatory status of the patient. A patient emerging from shock, with warm extremities, improving color, and functioning kidneys, might have a blood pressure of 85/50 when another, deep in shock, might show a blood pressure of 95/80. Clinical recovery from circulatory failure in all those who survived was associated with attainment of a pulse pressure of at least 25 mm. Hg.

COMMENT

This experience with 32 instances of severe shock complicating myocardial infarction is notable more for the 14 who survived than for the 18 who did not. As may be seen from the examples described, the subjects of this study were the kind who contribute a substantial segment of the total mortality in any large series of acute myocardial infarctions. If one patient with hemopericardium and the 74 year old patient with massive aspiration of gastric contents are excluded as having been beyond help, our results would indicate that active intervention for shock offers the hope of reducing by half fatalities from this complication.

Of the 32 patients, 26 went into lasting shock without any detectable reason other than the effects of acute myocardial infarction. However meticulous the preventive measures against thromboembolism, rhythm disturbances, pulmonary infection and other complications, the adverse course of these patients could not have been forestalled. Their survival depended on sustaining a minimal circulation compatible with life during a critical period, until such hearts as were capable of recovery resumed adequate function. The contribution of shock therapy in these patients would seem to be prevention or limitation of progressive myocardial deterioration from the effects of shock, per se.

The adverse experience with patients who have been long in shock before treatment is begun suggests that it may be beneficial not to defer shock therapy until circulatory failure is manifest and well established. In the hope of encroaching further on the mortality from acute myocardial infarction, we have extended the use of vasopressor drugs, and also of blood or plasma where pulmonary edema did not threaten, to patients whose overt manifestations of shock are minimal or absent, but whose blood pressure falls from normal or hypertensive levels to a systolic level under 100 mm. Hg or to a pulse pressure under 25 mm. Hg. We find these patients much more promptly responsive than the critically ill group which is the subject of the present study; however, an undetermined proportion of these patients would be expected to recover spontaneously. How many are saved by prompt treatment from progressive and fatal shock it may be possible to evaluate eventually from analysis of a large and statistically valid experience.

Review of our records brings to light the great importance of systematic and well re-
hearsed teamwork in the management of the desperately ill patient in shock. A nurse cannot be given responsibility for the many decisions required by the fluctuating balance of the patient. The emergency situation requires a hospital environment. It may last for 48 hours or longer and require the close attention of a succession of resident physicians. A single experience of seeing a hopelessly shocked, moribund patient respond and later walk out of the hospital is generally sufficient to inspire physicians and nurses with the high morale and persistence necessary for the management of this type of emergency.

SUMMARY

Acute myocardial infarction accompanied by deep and lasting shock was encountered in 32 patients in a three-year period. Fourteen of the 32 survived following vigorous management of the shock with vasopressor drugs, blood and plasma.

Shock, per se, contributes materially to the mortality from acute myocardial infarction. Active intervention against shock may spare the injured heart a period of greatly reduced coronary blood flow and appears to improve the chances for survival.

SUMARIO ESPAÑOL

El “shock” complicando infartos agudos del miocardio contribuye materialmente al total de la mortalidad. El raciocinio de combatir el shock ha sido discutible, muchos clínicos opinando que la caída en presión arterial tiene un efecto saludable al reducir el trabajo de un corazón agudamente averiado. Este informe relata la experiencia de cuatro años en la intervención activa en el shock. Los datos sugieren que la intervención temprana en el shock es frecuentemente una salvadora de vidas, y puede ser instrumental en disminuir la mortalidad de la mitad de los pacientes más graves.

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