Hemodynamic Changes in Patients with Acute Myocardial Infarction

By Michael Thomas, M.B., M.R.C.P., Raoul Malmcrona, M.D., and John Shillingford, M.D., F.R.C.P.

The hemodynamic changes in patients with acute myocardial infarction have been the subject of several investigations. In most seriously ill patients the usual circulatory abnormality is a low cardiac output with peripheral vasoconstriction. Some cases with a high cardiac output have been reported, however, and recently Malmcrona and Varnauskas have shown a correlation between high cardiac output and a rise of the patient's temperature. The concept of low cardiac output and a state of peripheral vasoconstriction is mainly based on studies of groups of patients on whom single hemodynamic measurements have been made during the first few days following acute myocardial infarction.

The purpose of this paper is to report serial measurements and clinical observations made on patients during each of the first 3 days following acute myocardial infarction, at about 1 week later and, when possible, before the patient left hospital. The results suggest that the hemodynamic state can be influenced to an important extent by factors not directly related to the myocardial contractile defect, and that these factors are not always apparent from the clinical examination.

Material

Nine male patients aged 51 to 73 years were studied. Eight of them were referred to the hospital because of severe chest pain and one because of an attack of unconsciousness. All had electrocardiographic signs of acute myocardial infarction.

The serum transaminase or lactic dehydrogenase was raised in eight patients. In seven patients the oral temperature was abnormally high at some time during the first 3 days.

Three patients had had a previous myocardial infarction. Two patients had had treatment for ventricular fibrillation, including external cardiac massage and direct-current defibrillation, some hours before the first hemodynamic study. Six patients had complained of angina for some time before infarction. Two patients had diabetes mellitus. Clinical features of patients at the times of hemodynamic study are given individually.

Methods

When patients arrived in hospital and the diagnosis of acute myocardial infarction was substantiated, they were transferred to a special intensive care unit permanently equipped with apparatus for observation, investigation, and treatment as described elsewhere.

After clinical assessment, electrocardiography and chest x-rays were completed, polyethylene catheters were inserted percutaneously into the brachial artery and an antecubital vein. The venous catheter was advanced so that the tip lay in the region of the great veins. Between hemodynamic measurements the catheters were flushed every 2 to 4 hours and were kept filled with heparin 5,000 U/ml. The arterial catheter was withdrawn after 24 hours.

Hemodynamic measurements were made as soon as possible after the onset of symptoms and verification of the diagnosis. Usually the first set of measurements was made within 6 hours of the onset of symptoms and in all patients it was done within 24 hours. Measurements were repeated within the next 24-hour period and with one exception in the following 24 hours also. Further investigations were made once or twice between the sixth and fourteenth days in all except one patient. Follow-up studies were made after 23 to

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31 days in four patients and on the fifty-third and sixtieth days in two others.

The cardiac output was determined by a dye-dilution technic\textsuperscript{12} with Coomassie blue as indicator and recording of the curve with a photoelectric-earpiece.\textsuperscript{4} The first curve was calibrated by comparing the height of the tail of the curve 3 minutes after injection with the amount of indicator in a blood sample taken at the same time. Dye was extracted from the plasma and measured by spectrophotometry. The accuracy of the method was checked at intervals by comparisons with \textsuperscript{131} indicator-dilution curves obtained by intermittent arterial sampling. Subsequent relative cardiac outputs were calculated from the areas of dilution curves.\textsuperscript{13} Heart rates were measured from an electrocardiogram over half-minute periods at the same time as the cardiac output was measured. Brachial arterial pressures were measured by strain-gage manometers (P23Gb Statham Transducers, Inc.). A direct-recording system (Devices Ltd.) was used. Some blood pressures measured by sphygmomanometry are specified in the tables and diagrams. Measurements were made throughout the period of study and immediately before each cardiac output determination. Pressures were measured with reference to a point 5 cm. below the sternal angle.

The hematocrit level was measured by centrifugation in a Wintrobe tube at 3,000 r.p.m. for 30 minutes. The radius to the end of the tube was 15 cm.

Pressure/flow ratio (systemic peripheral resistance) was calculated as the mean pressure in the brachial artery expressed in millimeters of mercury, divided by cardiac output expressed in liters per minute. In the case of sphygmonanometric measurements, diastolic pressures plus one third of pulse pressure were used.

During the acute illness initial measurements were made with the patient breathing oxygen. For comparison, therefore, later measurements were also made with the patient breathing oxygen.

\textbf{Results}

Results of the hemodynamic measurements are summarized in table 1 and in figures 1 to 8. In the figures, continuous lines between points are used as an aid to identification of individual patients.

\textbf{Hemodynamic Changes}

\textit{Heart Rate}

The range of initial heart rates varied between 51-96 beats/min. and showed no special subsequent trend (fig. 1). Of particular note are patients 1 and 5, who had low heart rates despite a low cardiac output. In case 9, both heart rate and stroke volume rose during the first 3 days. The resultant increase in cardiac output, however, was as-

\begin{figure}
\centering
\includegraphics[width=\textwidth]{heart_rates.png}
\caption{Heart rates of each patient at the time of hemodynamic investigations.}
\end{figure}

\textsuperscript{4}Cambridge Instrument Co. Dye Dilution Recorder Mk II modified as reported.
### Table 1

**Hemodynamic Data of Patients with Myocardial Infarction**

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<th>Patient no.</th>
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*Circulation, Volume XXXI, June 1965*
associated with a fall in mean arterial pressure due to a fall in peripheral resistance.

Of four patients (nos. 1, 2, 3, and 4) in whom heart rate decreased from the first to the second day, two (nos. 2 and 3) showed a fall in cardiac output and a fall in mean arterial pressure. Cases 2 and 3 thus showed a fall in heart rate over the first 3 days despite a large fall in blood pressure. A previously hypertensive patient (no. 6) was remarkable in that the heart rate on the first day was unusually low in the presence of a markedly lowered blood pressure.

From the third day onward the heart rate tended to remain constant.

Cardiac Output

The initial cardiac output varied from 2.1 to 7.1 L./min. (fig. 2). The lowest outputs were seen in patient 1, who had had two previous myocardial infarctions, and in patient 9, who had had one previous myocardial infarction. In the latter case, clinical improvement paralleled increase in cardiac output from 2.1 to 4.4 L./min. The highest outputs measured were in patient 8, who had a marked febrile reaction, and in patient 7, who was known to have been hypertensive previously.

In patient 7, a progressive fall in cardiac output during the first 3 days was associated with a fall in arterial pressure such that the calculated peripheral resistance increased. In patient 6, another previously hypertensive patient, a progressive rise in cardiac output over the first 3 days was associated with a rise in arterial pressure and an increase in peripheral resistance.

In the other patients the cardiac output was in the lower range of normal for this age group for the first 3 days and during the following 2 weeks, but then increased.

The low cardiac output on the sixth and seventh days in patient 4 may be due to an extension of myocardial infarction, as suggested by the development of atrial fibrillation and new ST elevation in lead V6 on the electrocardiogram. The cardiac outputs shown on days 6 and 7 are those recorded after conversion to sinus rhythm. Immediately before conversion to sinus rhythm the cardiac output was of the same order although the heart rate was much higher.
HEMODYNAMIC CHANGES IN INFARCTION

Stroke Volume

Stroke volume varied even more widely than the cardiac output on the first day, there being a five-fold difference between highest and lowest levels (fig. 3). The three patients (nos. 1, 2, and 9) with very low stroke volume on the first few days had a progressive increase during the hospital stay.

The patients with the highest values (patients 6 and 7) had low heart rates but patient 8, with the next highest stroke volume, had a heart rate of 80/min. Patient 2, with a low cardiac output, stroke volume, and normal range of blood pressure associated with a high peripheral resistance, failed to show a rise in stroke volume when mean pressure, heart rate, and calculated peripheral resistance had fallen. Patient 9 showed only a small increase in stroke volume when there was a considerable fall in mean arterial pressure.

Patient 1, who also had a low stroke volume initially, showed a fall in heart rate from the first o the second day and a corresponding increase in stroke volume. There was no fall in mean pressure. During this period the patient had been digitalized.

Blood Pressure

Brachial arterial systolic pressure was normal or high in six patients (nos. 2 to 7) on admission and subsequently decreased over the first 3 days in five of the six (fig. 4A). The pressure remained low in the three patients (nos. 1, 8, and 9) with an initially low blood pressure. The systolic pressure was low also during the following 2 weeks but was higher at the final investigations.

Brachial arterial diastolic pressures changed proportionately much less than systolic pressure (fig. 4B). The four patients with the largest fall in diastolic pressure in the first and second days (patients 2, 3, 7, and 9) all had large falls in mean and systolic pressure also. The diastolic pressure showed no major change over the period of study.

Brachial arterial mean pressure showed the same trend as the systolic pressure (fig. 4C). In patient 1, 8, and 9, with an initially low blood pressure, it remained low throughout the first 3 days. Patient 8, however, was in marked distinction from patients 1 and 9 in that the cardiac output was more than three times the level of the other two patients.
on the first as well as the second day. On the third day the cardiac output in patient 8 was twice the level seen in the other two patients. The peripheral resistance was accordingly very low in patient 8 and high in patients 1 and 9.

Of six patients with a mean arterial pressure within the normal or high range on the first day (nos. 2 to 7) all except one (no. 6) had a lower blood pressure on the second day. This fall in mean pressure was associated with a fall in cardiac output in four patients but with a rise in one (patient 5). Patient 6 showed a progressive rise in pressure over the first 3 days and a rise in cardiac output due to a rise in heart rate.

**Figure 4A**

Systolic arterial blood pressure measurements made at intervals throughout the period of study. Direct intravascular pressure measurements are indicated by small circles. Sphygmomanometric readings are indicated by large circles.

**Figure 4B**

Diastolic arterial blood pressure measurements made at intervals throughout the period of study. Direct intravascular pressure measurements are indicated by small circles. Sphygmomanometric readings are indicated by large circles.
HEMODYNAMIC CHANGES IN INFARCTION

Of the four patients (nos. 2, 3, 4, and 7) who had a fall in blood pressure associated with a fall in cardiac output, two had no change in peripheral resistance, one had a fall, and one had a rise.

From the second week to the final study there tended to be a rise in mean arterial pressure.

Pulse Pressure

Pulse pressure changes essentially reflected the fall in systolic arterial pressure over the first few days (fig. 5). Of the three patients (nos. 1, 8, and 9) with the lowest pulse pressure only two had a low stroke volume and the third (patient 8) a high one. Pulse pressure from the first to the second day showed no relation to stroke volume change over the same period. Pulse pressure remained low over the following 2 weeks and had risen by the time of the final investigation.

Peripheral Resistance

On the first day the range of peripheral resistance in the different patients was wide and varied from 10 to 38 units (fig. 6). The brachial arterial systolic pressure of the two patients (nos. 8 and 9) at the extremes of the range of peripheral resistance was the same. The peripheral resistance of the four patients (nos. 1, 2, 5, and 9) with the highest values on the first day decreased within the following day. The general tendency was for abnormal peripheral resistance to approach the normal over the acute period.

Clinical and Hemodynamic Features of Individual Patients

Patient 1 was 64 years old and had had two previous myocardial infarctions. He was admitted with a severe chest pain and in cardiac failure. He looked ill, pale, cyanotic, had a moist skin, and was orthopneic. He was in sinus rhythm, heart rate was 100 beats/min., systolic blood pressure 80 mm. Hg, and jugular venous pressure was elevated 6 cm. The heart was enlarged. A third heart sound was present. The liver was enlarged but there was no peripheral edema. Rales were audible over the whole chest. An electrocardiogram showed ST-segment elevation in V2 and V4. Chest x-ray confirmed cardiac enlargement. The pulmonary veins were dilated and there was extensive pulmonary edema.

He was given digitalis and diuretics and improved progressively. Oral temperature was normal (below 98.4 F.) on the first 2 days and 100.8 F. on the third day.

When the first hemodynamic measurement was made he had improved slightly. Heart rate was 68 beats/min., and blood pressure was 110/70 mm. Hg. Cardiac output was 2.5 L./min. and stroke volume 37 ml.

![Chart](chart.png)

**Figure 4C**

Mean arterial blood pressure measurements made at intervals throughout the period of study. Direct intracardiac pressure measurements are indicated by small circles. Sphygmomanometric readings are indicated by large circles.

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He improved further over the following 2 days. Jugular venous pressure had fallen to normal during the first 24 hours. On the second day the third heart sound was absent but was just audible on the third day. Chest x-ray revealed almost complete resolution of the pulmonary edema by the second day. Heart size was unchanged. In this time the cardiac output had risen slightly, heart rate had fallen, and stroke output had almost doubled. Blood pressure also remained unchanged. At the time of follow-up there had been another small rise in cardiac output, which was

**Figure 5**

Pulse pressure measurements made at intervals throughout the period of study. Direct intravascular pressure measurements are indicated by small circles. Sphygmomanometric readings are indicated by large circles.

**Figure 6**

Peripheral resistance calculated from measurements of cardiac output and mean arterial blood pressure. Small circles represent calculations from direct intravascular pressure measurements. Large circles represent calculations based on sphygmomanometric readings.
associated with a further increase in stroke volume. Blood pressure also showed a slight rise.

The notable feature in this patient recovering from acute myocardial infarction in a previously damaged heart was that the clinical improvement was associated with only a small rise in cardiac output but a marked lowering of the heart rate and increase in stroke volume.

**Patient 2** was 67 years old and was admitted with severe chest pain, looking pale and anxious. The skin was moist. He was in sinus rhythm, blood pressure was 170/90 mm. Hg. Jugular venous pressure was not elevated. The apex beat was normal in position. An atrial sound and a third heart sound were audible. In the lungs a few rales were heard. No peripheral edema was present. Temperature was normal.

Electrocardiogram showed ST elevation in V₂ to V₄. Chest x-ray showed that the heart was slightly enlarged; lung fields were normal.

When the first hemodynamic measurement was made he had improved slightly. Heart rate was 91 beats/min. and brachial arterial pressure was 138/85, with a mean of 105 mm. Hg. and mean central venous pressure was +3 mm. Hg. Cardiac output was 3.6 L./min., stroke volume was 40 ml., and pressure/flow ratio was 29.

In the following day he was free from pain, more composed, and his skin appeared normal. The presystolic heart sound remained and a pericardial friction rub was audible. Chest x-ray showed some abnormal shadowing in the right lower zone. Oral temperature remained normal. Cardiac output had fallen slightly with a fall in heart rate, and stroke volume remained the same. Blood pressure had fallen considerably and pressure/flow ratio had fallen to normal. At the follow-up study, cardiac output was considerably higher. Heart rate was lower and stroke volume was raised. Blood pressure had also risen.

In this patient clinical improvement and loss of pain were associated with a small fall in cardiac output, lower blood pressure, and a decrease of pressure/flow ratio to normal value.

**Patient 3** was 63 years old and had diabetes mellitus. He was admitted with severe chest pain and looked ill, pale and anxious. The skin was moist. Oral temperature was 99.2 F. He was in sinus rhythm and blood pressure was 115/70 mm. Hg; jugular venous pressure was normal. No added sounds were present. The lungs were normal to auscultation. There was no peripheral edema.

Electrocardiogram showed ST elevation in aV₁ and III. Chest x-ray showed the cardiothoracic ratio to be 0.59; the lung fields were normal.

When the first hemodynamic measurement was made, heart rate was 74 beats/min., brachial arterial pressure was 143/74; the mean was 100 mm. Hg. Mean central venous pressure was +5 mm. Hg. Cardiac output was 4.4 L./min., stroke volume was 59 ml., and pressure/flow ratio was 23.

On the second day the patient was free from pain but remained pale. Cardiovascular signs were essentially the same but rales were heard in the lungs. Oral temperature was 100.0 F. on the second and third days. Cardiac output had fallen in association with a fall in heart rate and stroke volume. Blood pressure had fallen considerably and pressure/flow ratio was essentially unchanged.

In this patient subjective improvement and loss of pain were associated with a fall in cardiac output and in blood pressure. Neither, however, reached a very low level.

**Patient 4** was 72 years old and had had a myocardial infarction 4 years previously. He was admitted following sudden loss of consciousness. On recovering consciousness he had vomited. He was very pale, sweating, and looked ill. He had no pain. He was in sinus rhythm, blood pressure was 130/73, and jugular venous pressure was normal. The cardiac signs were normal. There were no added sounds. Rales were audible at both lung bases. There was no peripheral edema. Oral temperature was 99.5 F.

Electrocardiogram showed ST elevation in V₂ to V₇. Chest x-ray showed the cardiothoracic ratio to be 0.51; shadows at the lung bases suggested pulmonary edema.

When the first hemodynamic measurement was made, heart rate was 77 beats/min., brachial arterial pressure was 131/72, mean was 94 mm. Hg. and mean central venous pressure was +2 mm. Hg. Cardiac output was 4.9 L./min., stroke volume was 64 ml., and pressure/flow ratio was 19.

On the second day the patient continued to look pale. A third heart sound was heard but no other change in physical signs had occurred. Oral temperature was 99.4 and 99.8 F. on the second and third days. Cardiac output had fallen in association with a fall in heart rate, and stroke volume was the same. Blood pressure was of the same order as well as the pressure/flow ratio.

The clinical course of this patient with a painless myocardial infarction was otherwise unremarkable and the hemodynamic feature was a small fall in cardiac output when the third heart sound became audible.

**Patient 5** was 73 years old and was admitted with chest pain extending down both arms. He was pale and felt weak and nauseated. He was in sinus rhythm, blood pressure was 140/80 mm. Hg. and jugular venous pressure was +3 cm. No cardiac enlargement was detectable clinically. An atrial sound was present but no third sound.
The lungs were normal to auscultation. The liver was not abnormally palpable and there was no edema. Temperature was normal.

Electrocardiogram showed ST elevation and T inversion in aVF and III. Chest x-ray revealed an enlarged heart (cardiothoracic ratio 0.59), slightly full hilar shadows, but no evidence of edema.

When the first hemodynamic measurement was made, heart rate was 51 beats/min., and brachial arterial pressure was 137/68, with a mean of 100 mm. Hg. Cardiac output was 3.5 L./min., stroke volume was 68 ml., and pressure/flow ratio was 29. During the following 2 days a retrosternal ache remained. Skin appeared normal. An atrial sound was still present and temperature was normal. Only 200 ml. of urine had been passed in the first 24 hours. In this time cardiac output had risen in association with a rise in heart rate and a small rise in stroke volume. Brachial arterial pressure had fallen as had pressure/flow ratio.

At the follow-up, cardiac output had risen to normal value associated with a rise in heart rate, stroke volume, and arterial pressure.

Notable features in this patient were a large fall in pulse pressure over the first few days despite a rise in the stroke volume and an initial decrease of arterial pressure associated with a rise in heart rate.

Patient 6 was 47 years old and previously had been noted to be hypertensive. He was admitted following sudden severe chest pain in the night. He had got up and felt faint, sweated profusely, and had vomited. On admission to hospital pain had regressed. He looked ill; the skin was pale, cold, and moist. He was in sinus rhythm, heart rate was 60 beats/min., and blood pressure was 150/105 mm. Hg. Jugular venous pressure was not raised. The heart was of normal size as judged clinically. Atrial and third heart sounds were heard alternately. Lungs were normal to auscultation. There was no peripheral edema. Oral temperature was normal.

Electrocardiogram showed slight ST elevation in V4 and subsequently development of a pathologic Q wave in aVF, II, and III. Chest x-ray showed slight cardiac enlargement to the left and normal lung fields.

One hour before measurements were made he had a sinus bradycardia associated with a fall in systolic blood pressure to 80 mm. Hg, increased pallor, and mental deterioration. Spontaneous increase in heart rate resulted in general improvement. When the first hemodynamic measurement was made, the heart rate was 51 beats/min. and brachial arterial pressure was 131/72, with a mean of 94 mm. Hg. Mean central venous pressure was +7 mm. Hg. Cardiac output was 4.7 L./min., stroke volume was 94 ml., and pressure/flow ratio was 20.

For the first and second days the patient remained pale. Atrial and third sounds were present. He was oliguric. On the third day he was generally improved and the heart sounds were normal. Oral temperature was 98.2 and 99.1 F. on the second and third days. Cardiac output rose progressively associated with a rise in heart rate. Stroke volume fell slightly. Brachial arterial pressure rose to hypertensive levels. There was a small rise in pressure/flow ratio. Pulse pressure remained of the same order as on the first day. At the follow-up cardiac output had increased somewhat further. Blood pressure and pressure/flow ratio had not materially altered.

The notable feature in this previously hypertensive patient was a low initial blood pressure with low heart rate. When cardiac output rose with an increase in heart rate, blood pressure rose considerably.

Patient 7 was 60 years old and previously hypertensive. He had a mild form of diabetes mellitus controlled by diet. He was admitted with severe chest pain extending to the neck which had not responded to pethidine (Meperidine) given several hours previously. He looked distressed and ill but the skin appearance was normal. He was in sinus rhythm, blood pressure was 200/120. Jugular venous pressure was elevated 4 cm. The heart was enlarged as judged by an apical impulse displaced to the left. An atrial sound was present. No third sound was heard. The lungs were normal to auscultation and there was no peripheral edema. Oral temperature was normal.

Electrocardiogram showed a pathologic Q wave in III and aVF together with ST elevation and T-wave inversion.

When the first hemodynamic measurement was made, heart rate was 60 beats/min., brachial arterial pressure was 196/85 mm. Hg, and 120 mm. Hg mean. Mean central venous pressure was –2 mm. Hg. Cardiac output was 6.3 L./min., stroke volume was 105 ml., and pressure/flow ratio was 19. After the first set of observations and before he was given morphia, the patient vomited. This was associated with a tachycardia and rise in cardiac output to twice the previous value though the stroke volume remained the same. Pressure increased considerably and there was a small fall in pressure/flow ratio.

On the second and third days he was free from pain and in good general condition. Skin appearance was normal. Oral temperature was normal. An atrial heart sound was heard throughout this period. Cardiac output fell considerably over the first 3 days. Heart rate rose to some
extent and stroke output fell markedly. Systolic arterial pressure fell, although diastolic pressure remained constant. Pressure/flow ratio was essentially unchanged. Pulse pressure fell to half the initial value.

At follow-up investigation, the cardiac output and stroke volume had risen. Systolic blood pressure and pulse pressure had risen also.

This previously hypertensive patient showed a fall in blood pressure on the first 3 days associated with a fall in stroke output.

Patient 8 was 51 years old. Following acute chest pain he collapsed while being admitted to hospital. Ventricular fibrillation was diagnosed and treated by external cardiac massage and direct-current defibrillation. Eighteen hours afterwards the first hemodynamic measurements were made. At this time he was free from pain but felt very weak and looked ill. Rib fractures were present. The skin was of normal temperature and color but very moist. Body temperature was normal. He was in sinus rhythm, blood pressure was 90/50 mm. Hg, and jugular venous pressure was not raised. The heart was of normal size as judged by the position of the apical impulse but this was abnormally sustained throughout systole. Heart sounds were normal. There were no added sounds. In the lungs, crepitations were audible at both bases. No peripheral edema was present.

Electrocardiogram showed ST elevation and T inversion in V1 to V4 and pathologic Q waves were present in V1 to V4. Chest x-ray showed slight cardiac enlargement, pulmonary venous congestion, and generalized pulmonary edema. A rib fracture was visible.

When the first hemodynamic measurement was made, heart rate was 82 beats/min., and brachial arterial pressure was 98/53, with a mean of 70 mm. Hg. Central venous pressure was +2 mm. Hg. Cardiac output was 7.1 L./min., stroke volume was 87 ml. Pressure/flow ratio was 10.

The patient’s general condition remained poor, but he had no pain, although ribs had been fractured during resuscitation. Heart sounds were normal. The skin was excessively moist but warm and of normal color at all times. On the first day the temperature was normal, on the following 2 days it was 100.4 and 100.5 F. Cardiac output remained high, heart rate rose, and stroke volume fell. Brachial arterial pressure remained of the same order. At follow-up cardiac output was still high and blood pressure had risen.

The notable feature of this patient was hypotension in association with a high cardiac output and low pressure/flow ratio. This was present before, during, and after a rise in the patient’s temperature.

Patient 9 was 56 years old and had suffered myocardial infarction 2 weeks before study. Fourteen hours earlier he had ventricular fibrillation and was resuscitated by external cardiac massage and direct-current defibrillation. An electrocardiogram taken subsequently showed extension of the ST-segment elevations in precordial leads. A metaraminol drip was administered but discontinued 2 hours before the first hemodynamic investigation, at which time the patient was very pale, cyanotic, perspiring, and mentally blunted. He was in sinus rhythm at 60/min., with ventricular extrasystoles every third beat; blood pressure was 95/60 mm. Hg and jugular venous pressure was elevated 4 cm. The apex beat was not palpable. A third heart sound was audible. No peripheral edema was present. The chest wall had been damaged during resuscitation such that part of the lower sternum showed paradoxical movement during respiration. Rales were present in the lungs and a pleural rub was heard. Rectal temperature was 99.0 F. He was anuric.

Electrocardiogram showed pathologic Q waves in V2 to V4, and ST elevation in V2 to V4 was seen after the episode of ventricular fibrillation. Chest x-ray showed gross pulmonary edema. The heart was enlarged.

When the first hemodynamic measurement was made, heart rate was 96 beats/min., and brachial arterial pressure was 100/68, with mean of 80 mm. Hg. Mean central venous pressure was +5 mm. Hg. Cardiac output was 2.1 L./min., stroke volume was 22 ml., and pressure/flow ratio was 38.

During the following 2 days temperature was normal and some general improvement occurred on the second day and more on the third day. The patient remained pale. Diuresis began on the second day. Mean central venous pressure fell but a third heart sound remained. Rectal temperature was 98.6 and 100.0 F. on the second and third days. Cardiac output rose to some extent by the second day and was up to 3.5 L./min. by the third day. The heart rate remained the same and the stroke volume had risen. Brachial arterial pressure was even lower on the second day and pressure flow/ratio had fallen to normal.

The notable features of this patient were hypotension associated with low cardiac output and high pressure flow ratio. Obvious clinical improvement occurred when stroke volume and cardiac output rose although pressure remained low.

Discussion

Myocardial infarction often results in a severe circulatory disturbance. Attempts to define the exact hemodynamic changes in the circulation have been few in relation to the importance of the subject. Early investiga-
tors used ballistography\textsuperscript{14} or pulse wave analysis\textsuperscript{1} to estimate cardiac output; one used the Fick principle.\textsuperscript{2} The application of indicator-dilution technic made bedside measurement of cardiac output in myocardial infarction a more convenient procedure and resulted in several reports.\textsuperscript{3-9} The conclusion drawn from these studies was that cardiac output was reduced according to the severity of the illness as assessed on clinical grounds. Groups of patients were compared, and individual differences were not emphasized. Two authors, however,\textsuperscript{4, 9} commented that not all cases with a low cardiac output were in clinical shock; another\textsuperscript{7} investigated two patients with a raised cardiac output on admission and suggested that this was due to alarm.

A rise in the patient’s temperature has been shown to be one factor promoting a fall in peripheral resistance and, in some, a rise in cardiac output.\textsuperscript{10}

By studying serial measurements of the hemodynamic changes we have shown that there may be a variety of circulatory disturbances that are incompletely explained by the low-output theory. In some cases the cardiac output is high while the blood pressure is low. Low peripheral resistance can be due to a fever but other factors appear to exist. Peripheral resistance can progressively fall while cardiac output is falling. In some cases heart rate is low in the presence of a low cardiac output. Some hypotensive patients have a low heart rate which, when subsequently increased, is accompanied by a rise in blood pressure.

Experimental work in animals\textsuperscript{15} demonstrated that reflexes initiated by receptors in the left ventricle can produce a slowing of the heart and hypotension. The importance of such reflexes in the physiologic changes that follow occlusion of a major coronary artery in dogs and cats was recently pointed out.\textsuperscript{16} Measurements made in our patients show that the hemodynamic changes that follow acute myocardial infarction in man are sometimes of a pattern that could be explained by reflex activity of this type.

The clinical picture does not always appear to follow the hemodynamic changes but a larger series is necessary for substantiation. Severely ill patients, as judged by a pale cold sweating skin, mental blunting, and immobility, were found to have in some cases a low cardiac output and high peripheral resistance; in others, however, who had similar clinical findings, the cardiac output was only slightly decreased and the peripheral resistance was normal. In one patient in whom the cardiac output was high and the peripheral resistance low, the skin was moist but warm.

The presence of a rapid pulse has been related to the severity of the circulatory disturbance in myocardial infarction;\textsuperscript{9} while this may be true in some cases, in our series very rapid heart rates were not encountered and in some patients the rate was remarkably low. The blood pressures were within a comparatively narrow range and gave little indication of the cardiac output or peripheral resistance; this was especially so if the patient had previously been hypertensive.

It has been suggested\textsuperscript{9} that the presence of a third heart sound in patients with acute myocardial infarction is related to a low stroke output. In our series there was no clear relationship between stroke output or change in stroke output and presence or absence of a third heart sound.

A common feature in the initial recovery of patients with acute myocardial infarction is the symptomatic improvement that can occur without much change in the cardiac output and blood pressure; in fact, this improvement sometimes occurred despite a fall in cardiac output; thus there may be factors quite apart from the circulatory disturbance that contribute to the patient’s clinical condition. Respiratory and metabolic changes could be important in this respect. The mechanisms underlying the circulatory and other changes are not clear and require further study, especially in relation to the need for a rational treatment of patients with acute myocardial infarction.
Summary

The hemodynamic changes following acute myocardial infarction have been studied in patients in an intensive care unit. Serial determinations of cardiac output and blood pressure have shown a wide range of hemodynamic patterns in severely ill patients varying from a low cardiac output and high peripheral resistance to a high cardiac output and low peripheral resistance. The progression of changes during the illness and their relation to the clinical findings have been discussed.

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Vision and Fulfillment

Genius begins great works, labor alone finishes them.—Joubert.
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