Hemodynamic Studies of Patients with Myocardial Infarction

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Cardiac output, venous and arterial pressure, total peripheral resistance and blood volume were measured in patients with acute myocardial infarction, with and without shock, and on recovery, and in patients without heart disease. Cardiac output falls to below normal after myocardial infarction, and rises significantly, though not always to normal, on recovery. Cardiac output was very low when myocardial infarction was complicated by shock. Fall of cardiac output to very low levels following myocardial infarction does not necessarily lead to development of shock. Venous pressure tended to be elevated in patients in shock as a result of myocardial infarction. Cardiac output was subnormal in patients with arteriosclerosis but without clinical heart disease: this was not attributable to reduced body oxygen consumption.

The precarious physiologic status of patients with acute myocardial infarction has made it difficult to derive comprehensive hemodynamic data from them. The purpose of this report is to present data from patients with myocardial infarction with and without shock, and from certain control groups, derived by techniques employable at the bedside. The functions studied have been: cardiac output, mean arterial and venous pressure, total peripheral resistance, and circulating plasma and blood volume. It was hoped that the accumulation of these data would disclose circulatory abnormalities peculiar to patients with shock resulting from myocardial infarction, and thus clarify the pathogenesis of this serious complication. Although this hope was not realized, it appears possible to draw some conclusions concerning the hemodynamic changes which occur in patients with myocardial infarction.

Patients

Observations were made on 39 patients grouped in five categories: (1) normal patients, (2) patients with acute myocardial infarction without shock, (3) patients with acute myocardial infarction with shock, (4) patients recovered from myocardial infarction, and (5) patients with arteriosclerosis.

Normal Patients. This group consisted of hospitalized men and women who were considered to have no circulatory abnormality. Their ages ranged from 21 to 55 years; half were 50 to 55 years old. Blood hemoglobin concentration was at least 12.5 Gm. per 100 cc. of blood. Cardiac output and related functions were determined in 10 patients, and blood volume only in four others.

Patients with Arteriosclerosis. This group consisted of 10 individuals of the older age group (62 to 77 years) who had evidence of arteriosclerosis but no clinical evidence of heart disease. Careful examination excluded, as far as possible, diminished cardiac reserve and previous myocardial infarction. All of these patients had normal sinus rhythm and normal heart size. One had essential hypertension. These patients with uncomplicated arteriosclerosis were studied in order to have another control group with which to compare the findings in patients with acute myocardial infarction.

In the patients with myocardial infarction, the diagnosis of myocardial infarction was based on the presence of classic clinical findings, including typical electrocardiographic changes. All of these patients had sinus rhythm at the time of study. With one exception (Ard. *) none had congestive heart failure at the time of study or prior to myocardial infarction. These patients are divided into three groups.

Patients with Acute Myocardial Infarction without Shock. The 10 patients in this group not only fulfilled the criteria just mentioned above, but were included only if they appeared to the observer at the bedside to be in good condition and free from distress. This group thus is composed of patients who appeared to be tolerating their myocardial infarction very well. With one exception (Cass. *) each was studied within 24 hours of clinical myocardial infarction. No

* See Case Summaries.

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medication was given other than 10 to 15 mg. of morphine sulfate administered at the time of admission to the hospital, except for one patient (Ore.*) who received in addition 250 cc. of plasma three hours before study because he was mistakenly believed to be developing shock.

**Patients with Acute Myocardial Infarction and Shock.** The criteria for shock were: prostration, stupor, cold, clammy and cyanotic skin, oliguria and reduced arterial pressure. In most patients blood pressure could not be determined by auscultation. That these patients were critically ill was attested by the fact that all but one died in shock within 12 hours of the time of study. Every patient but one (Ard.*) was studied within two hours of the development of shock. With two exceptions, (Ard., Cass.*) studies were completed before the institution of therapy. Although every patient had a moderate number of scattered pulmonary rales, only one (Ard.*) had overt congestive heart failure either at the time of study or previously.

There were nine patients in this group, in seven of whom the entire series of determinations was performed; in two patients the femoral venous pressure alone is reported.

**Patients Recovered from Myocardial Infarction.** The 12 subjects in this group had recovered to the point of being fully ambulatory without clinical evidence of diminished cardiac reserve. None of the patients had been studied previously, in the acute phase of myocardial infarction. Nine patients were studied six to eight weeks after the occurrence of myocardial infarction; the remaining three, a variable period of time (2, 5, and 13 months respectively) after myocardial infarction.

**Methods**

**Mean arterial pressure** was measured directly, through an inlying arterial needle, with a damped mercury manometer. Preliminary studies disclosed agreement within 5 per cent between values so derived and those determined by integration of blood pressure tracings obtained from the femoral artery.

**Venous pressure** was measured directly by the technic of Moritz and von Tabora,† usually in a large antecubital vein, occasionally in a femoral vein.

**Cardiac output** was measured by the indicator-dilution technic of Hamilton.‡ About 3 cc. of the dye T-1824,* or 25 to 50 microcuries of P*32-labeled human serum albumin, were injected rapidly into an antecubital or femoral vein from a calibrated syringe, following which serial two- or three-second samples of blood were collected from the femoral artery. Serum concentrations of T-1824 were measured with a Coleman Junior Sceptrophotometer. Measurements of radioactivity in the injected material and the serum were made by conventional technics.†

**Total peripheral resistance** (R) was calculated from the formula:

\[
R = \text{mean arterial pressure} - \text{right atrial pressure (mm. Hg)} \div \text{cardiac output (liters/minute)}
\]

One half of the venous pressure was taken as equal to right atrial pressure, except in the one patient with congestive heart failure. In this patient, venous pressure was regarded as equal to right atrial pressure in view of evidence‡ indicating reduction toward zero of the venoatrial pressure gradient in congestive heart failure. These approximations of right atrial pressure introduce errors of only several millimeters of mercury in total magnitudes of 50 to 140 mm. Hg. The numerical value of peripheral resistance was left in the units of the equation (millimeters Hg per liter per minute), for no advantage is served by conversion to so-called "absolute" units.

"Plasma volume" was measured from the 30-minute disappearance slope of the indicator in arterial blood. "Blood volume" was calculated from the "plasma volume" and the arterial hematocrit. The predicted normal blood volume for a given patient was calculated from his height, weight, and surface area according to the charts of Gibson and Evans.¶

**Hematocrit** was determined on arterial blood which was centrifuged for 30 minutes at 3,000 revolutions per minute. Four per cent of the numerical value of the hematocrit was deducted to correct for trapped plasma.

The equipment to obtain the above observations was mounted on a rolling table so that determinations could be done at the bedside if necessary. When patients were well enough to be brought to the laboratory, the oxygen consumption was also measured. Exhaled gas was collected in a Tissot gasometer, and inhaled and exhaled gas was analyzed for oxygen and carbon dioxide with a Pauling apparatus. In most cases, a respiratory quotient of 0.81 was assumed.

A typical experiment proceeded as follows. The patient was fasting. A needle‡ was introduced into a femoral artery. A no. 18 gauge needle was threaded into an antecubital or femoral vein, attached to a venous manometer and intermittently flushed with heparinized isotonic saline. After a 20-minute delay, during which the patient became accustomed to the needles, the venous manometer was disconnected briefly and a precisely measured volume of indicator was rapidly injected into the intravenous needle from a calibrated syringe. Serial two- or three-

* We are grateful to Warner-Chilcott Laboratories for providing generous quantities of the dye T-1824.

† The use and quantitation of radioactive materials were supervised by Dr. Robert Soberman.

‡ The authors wish to thank Mr. Oscar Schwi-detsky of Becton-Dickinson Co. for his generous cooperation in making available suitable arterial needles.
second samples of arterial blood were then collected in a series of small tubes mounted on a rack, for a period lasting until about 90 seconds after injection. Venous and mean arterial pressures were measured immediately thereafter. During the following half hour, samples of arterial blood were taken every 10 minutes for "plasma volume" determination. After the last of these samples was collected, the entire series of determinations was usually repeated. When the foregoing determinations could be done in the laboratory, oxygen consumption was measured in duplicate or triplicate during the half hour period following the second cardiac output determination.

These procedures appeared not to disturb the patients, and no untoward effects occurred.

Reliability of the Indicator-Dilution technic for Determination of Cardiac Output

In a preliminary study,* cardiac output was measured simultaneously by Fick and Hamilton technics 19 times in 18 resting patients with a variety of cardiovascular disorders. None of these patients was in shock. The values derived by the two technics agreed within 15 per cent in 16 of the 19 determinations (fig. 1).

Several groups* 1, 6, 7 have published comparisons of values derived simultaneously by Fick and Hamilton technics. In order to examine these data for evidence of any systematic differences between the two technics, t scores were calculated for cardiac output derived by the two technics in each laboratory, and in all the laboratories combined. The results of this analysis are presented in table 1.

This analysis indicates a probably significant systematic difference between the two technics in the Werkö series, and a significant systematic difference when the data from all the laboratories are combined. Although the difference between the two technics appears significant, it is important to note that the difference is small, and that the agreement between the two methods in different laboratories is good.

The reproducibility of the result obtained by the indicator-dilution technic in our hands was tested by performing duplicate determinations of cardiac output by this method within 40 minutes of each other in 34 subjects. In 31 of 34 pairs of determinations (fig. 2) the two values agreed within 15 per cent. According to this series of duplicate determinations, the value of a second single determination of cardiac output will agree within 25 per cent of the value of the first determination in the same patient 95 per cent of the time. If cardiac output is determined in duplicate, the average of the two determinations will agree within 18 per cent of the average of another pair of duplicate determinations on the same patient in the same state. It appears, then, that the Hamilton technic yields acceptably consistent values and seems satisfactory for the comparison of cardiac output magnitudes in patients who are not in shock.†

* Values derived in exercising patients were omitted from this analysis because of questionable validity of methods then in use for the measurement of oxygen consumption in exercising patients (8).
† Werkö and Kopelman injected the indicator into the pulmonary artery, the other two groups, into a peripheral vein.

![Cardiac Output](https://example.com/cardiac_output_image)

**Table 1.** Comparison by t Score Method of Values for Cardiac Output Derived by Hamilton and Fick Technics in Five Laboratories

<table>
<thead>
<tr>
<th>Laboratory</th>
<th>No. paired determinations</th>
<th>Cardiac Output</th>
<th>t</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>Werkö</td>
<td>68</td>
<td>6.11t</td>
<td>2.20</td>
<td>0.03</td>
</tr>
<tr>
<td>Kopelman‡</td>
<td>28</td>
<td>4.43†</td>
<td>0</td>
<td>—</td>
</tr>
<tr>
<td>Cournand,</td>
<td>Hamilton†</td>
<td>39</td>
<td>1.2</td>
<td>0.23</td>
</tr>
<tr>
<td>Smith‡</td>
<td>19</td>
<td>5.46</td>
<td>1.08</td>
<td>0.29</td>
</tr>
<tr>
<td>All Combined.</td>
<td>154</td>
<td>5.68</td>
<td>2.61</td>
<td>0.009</td>
</tr>
</tbody>
</table>

* This part of the study was done in cooperation with the Cardiovascular Hemodynamic Team of New York University College of Medicine.
† The foregoing statistical analyses were done with the guidance of Dr. Donald Mainland and Mrs.
myocardial infarction was not strikingly different from that in patients with arteriosclerosis, who also had significantly \( p < 0.01 \) lower cardiac index than the normal patients (fig. 4).

Cardiac index in the patients with myocardial infarction and shock was very low, significantly lower than in the patients with acute myocardial infarction who were not in shock. However, not all of the patients with acute myocardial infarction who had very low cardiac index were in shock. In 3 of the 10 patients with acute myocardial infarction without shock, the cardiac index was in the range of the shock group, and yet the patients presented no evidence of circulatory embarrassment. That these three patients did not, for some unknown reason, have a fixedly very low cardiac index is indicated by the subsequent rise in cardiac output as they convalesced.

For example, the patient Glaz. (fig. 5) had a cardiac index of 1.8 liters per minute per square meter of body surface area when first studied seven hours after myocardial infarction. Nevertheless his clinical condition was excellent. Nineteen hours later, his cardiac index was 2.4 liters per minute per square meter. This patient, and the two other patients with acute myocardial infarction without shock who also had very low cardiac index, could not be separated clinically from the other patients with acute myocardial infarction who had higher values for cardiac index.

**RESULTS**

**Cardiac Index** (fig. 3). The cardiac index was significantly subnormal in the patients with acute myocardial infarction without shock. Nine patients of this group were studied again on recovery, and cardiac index was noted to have increased significantly \( p = 0.01 \) but not always to normal values. (In one of these nine patients, the value for cardiac index was slightly lower on recovery than immediately after myocardial infarction. This apparent small decrement is within the error of reproducibility of the Hamilton technic and may thus be due to technical errors.) Cardiac index in most patients with acute myocardial infarction without shock and with "healed"

Lee Herrera of the Department of Medical Statistics, New York University College of Medicine. Their attentive cooperation is gratefully acknowledged.
HEMODYNAMIC STUDIES OF PATIENTS WITH MYOCARDIAL INFARCTION

Table 2.—Hemodynamic Studies of Patients with Myocardial Infarction

<table>
<thead>
<tr>
<th>Patient (Sex and Age)</th>
<th>Mean Arterial Pressure (mm. Hg)</th>
<th>Venous Pressure (mm. Hg)</th>
<th>Cardiac Index (L/M²/min.)</th>
<th>Total Peripheral Resistance (mm. Hg/L/min.)</th>
<th>Hct (%)</th>
<th>Blood Volume (liters)</th>
<th>Ratio Actual B.V. / Predicted B.V.</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Acute Myocardial Infarction with Shock</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Ard., M, 62</td>
<td>52</td>
<td>20.0*</td>
<td>1.6</td>
<td>15</td>
<td>37</td>
<td>5.7</td>
<td>1.07</td>
</tr>
<tr>
<td>Dam., M, 67</td>
<td>112</td>
<td>6.5*</td>
<td>2.2</td>
<td>28</td>
<td>44</td>
<td>4.4</td>
<td>.88</td>
</tr>
<tr>
<td>Cos., F, 61</td>
<td>58</td>
<td>7</td>
<td>1.9</td>
<td>16</td>
<td>37</td>
<td>5.7</td>
<td>1.4</td>
</tr>
<tr>
<td>Her., M, 50</td>
<td>54</td>
<td>13</td>
<td>1.3</td>
<td>17</td>
<td>42</td>
<td>4.2</td>
<td>.72</td>
</tr>
<tr>
<td>Li., F, 80</td>
<td>60</td>
<td>5.5</td>
<td>1.4</td>
<td>26</td>
<td>41</td>
<td>3.2</td>
<td>.80</td>
</tr>
<tr>
<td>Gri., M, 48</td>
<td>84</td>
<td>6.0*</td>
<td>1.5</td>
<td>31</td>
<td>46</td>
<td>4.5</td>
<td>.87</td>
</tr>
<tr>
<td>Cass., M, 66</td>
<td>70</td>
<td>11.0*</td>
<td>1.3</td>
<td>27</td>
<td>36</td>
<td>5.7</td>
<td>1.02</td>
</tr>
<tr>
<td>(without shock)</td>
<td>66</td>
<td>2.0</td>
<td>2.4</td>
<td>15</td>
<td>38</td>
<td>5.3</td>
<td>.95</td>
</tr>
<tr>
<td>(recovered)</td>
<td>76</td>
<td>4.0*</td>
<td>2.8</td>
<td>14.5</td>
<td>35</td>
<td>5.3</td>
<td>.95</td>
</tr>
</tbody>
</table>

In two other patients venous pressure only was measured and each was 7 mm. Hg.

<table>
<thead>
<tr>
<th><strong>Acute Myocardial Infarction without Shock and on Recovery</strong></th>
</tr>
</thead>
<tbody>
<tr>
<td>Bas., M, 51 (rec.)</td>
</tr>
<tr>
<td>Dae., M, 60 (rec.)</td>
</tr>
<tr>
<td>Glaz., M, 59 (rec.)</td>
</tr>
<tr>
<td>Gal., M, 62 (rec.)</td>
</tr>
<tr>
<td>Ma., M, 51 (rec.)</td>
</tr>
<tr>
<td>Bana., M, 66 (rec.)</td>
</tr>
<tr>
<td>Ung., M, 55 (rec.)</td>
</tr>
<tr>
<td>Belf., F, 60 (rec.)</td>
</tr>
<tr>
<td>Ore., M, 52 (rec.)</td>
</tr>
<tr>
<td>Smi., F, 57 (rec.)</td>
</tr>
<tr>
<td>Cu., M, 47 (rec.)</td>
</tr>
</tbody>
</table>

* Indicates femoral venous pressure; all others are antecubital venous pressures.

rec. = The data on "recovery" when the patient was ambulant and just before discharge from the hospital.

One patient, Cass., (fig. 5) was studied in three different states following myocardial infarction: in shock, after recovery from shock, and prior to discharge from the hospital. Cardiac index on the three occasions was 1.3, 2.4, and 2.8 liters per minute per square meter of surface area, respectively, showing, at least in this patient, increases in cardiac output as he improved clinically.

Venous Pressure (fig. 6). The venous pressure in the patients with acute and "healed" myocardial infarction, as well as in those with arteriosclerosis, was in the normal range of 0 to 10 mm. Hg. In eight patients with myocardial infarction with shock, venous pressure ranged from 5 to 13 mm. Hg; in the ninth patient, the only subject with congestive heart failure, the venous pressure was 20 mm. Hg.

Mean Arterial Pressure (fig. 7). The range of the values for mean pressure of patients

* See Case Summaries.
Table 3.—Hemodynamic Studies of Normal Subjects and Patients with Arteriosclerosis

<table>
<thead>
<tr>
<th>Patient</th>
<th>Arterial Pressure (mm Hg)</th>
<th>Venous Pressure (mm Hg)</th>
<th>Cardiac Index (L/min/m²)</th>
<th>Peripheral Resistance (mm Hg/L/min)</th>
<th>Hct (%)</th>
<th>Blood Volume (liters)</th>
<th>Ratio Actual B.V./Predicted B.V.</th>
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</thead>
<tbody>
<tr>
<td>Arteriosclerosis</td>
<td></td>
<td></td>
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<td></td>
</tr>
<tr>
<td>Go., M, 77</td>
<td>75</td>
<td>0</td>
<td>2.8</td>
<td>17</td>
<td>33</td>
<td>4.0</td>
<td>1</td>
</tr>
<tr>
<td>La., M, 69</td>
<td>94</td>
<td>3.5</td>
<td>2.8</td>
<td>20</td>
<td>43</td>
<td>3.8</td>
<td>.9</td>
</tr>
<tr>
<td>Ir., M, 74</td>
<td>88</td>
<td>2</td>
<td>2.3</td>
<td>21</td>
<td>44</td>
<td>5.3</td>
<td>1</td>
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<tr>
<td>Ga., M, 70</td>
<td>73</td>
<td>0</td>
<td>2.4</td>
<td>17</td>
<td>41</td>
<td>4.7</td>
<td>.89</td>
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<tr>
<td>Ha., M, 62</td>
<td>83</td>
<td>2</td>
<td>3.2</td>
<td>15</td>
<td>39</td>
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<td>—</td>
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<td>Su., M, 62</td>
<td>85</td>
<td>2</td>
<td>2.7</td>
<td>17</td>
<td>37</td>
<td>—</td>
<td>—</td>
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<td>Lip., M, 77</td>
<td>114</td>
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<td>2.4</td>
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<td>3.8</td>
<td>.79</td>
</tr>
<tr>
<td>M., M, 73</td>
<td>80</td>
<td>0</td>
<td>2.2</td>
<td>24</td>
<td>38</td>
<td>3.2</td>
<td>.75</td>
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<tr>
<td>An., M, 72</td>
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<td>16</td>
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<td>4.6</td>
<td>.94</td>
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<tr>
<td>Ya., M, 74</td>
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<td>0.5</td>
<td>2.7</td>
<td>28</td>
<td>40</td>
<td>4.0</td>
<td>1.0</td>
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<td>Ni., M, 22</td>
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<td>3.0</td>
<td>20</td>
<td>46</td>
<td>3.4</td>
<td>.87</td>
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<td>Fi., M, 55</td>
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<td>2</td>
<td>3.4</td>
<td>16</td>
<td>34</td>
<td>—</td>
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<td>Gi., F, 38</td>
<td>79</td>
<td>3</td>
<td>4.8</td>
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<td>3.0</td>
<td>.83</td>
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<td>Sec., M, 52</td>
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<td>2.9</td>
<td>17</td>
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<td>4.6</td>
<td>.96</td>
</tr>
<tr>
<td>MeA., M, 43</td>
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<td>—</td>
<td>3.1</td>
<td>—</td>
<td>40</td>
<td>—</td>
<td>—</td>
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<tr>
<td>Gra., M, 38</td>
<td>—</td>
<td>—</td>
<td>4.0</td>
<td>—</td>
<td>40</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>Rot., M, 53</td>
<td>86</td>
<td>8</td>
<td>3.5</td>
<td>15</td>
<td>34</td>
<td>4.7</td>
<td>.98</td>
</tr>
<tr>
<td>Mo., M, 52</td>
<td>88</td>
<td>4</td>
<td>3.8</td>
<td>13</td>
<td>41</td>
<td>5.6</td>
<td>1.08</td>
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<td>Ros., M, 50</td>
<td>87</td>
<td>6</td>
<td>3.5</td>
<td>13</td>
<td>36</td>
<td>7.3</td>
<td>1.3</td>
</tr>
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<td>Wri., F, 21</td>
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<td>3.1</td>
<td>—</td>
<td>40</td>
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<td>—</td>
</tr>
<tr>
<td>Fa., M, 44</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>44</td>
<td>4.4</td>
<td>.8</td>
</tr>
<tr>
<td>Cru., M, 42</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>40</td>
<td>3.8</td>
<td>.81</td>
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<td>Sta., M, 39</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>41</td>
<td>6.1</td>
<td>1.1</td>
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<tr>
<td>Pha., M, 38</td>
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<td>—</td>
<td>—</td>
<td>—</td>
<td>39</td>
<td>6.8</td>
<td>1.1</td>
</tr>
</tbody>
</table>

* In antecubital veins.

Fig. 4. Oxygen consumption per unit area of body surface and cardiac index in normal patients contrasted with those with uncomplicated arteriosclerosis.

Fig. 5. Summary of studies done on three different patients described in the text. Note that blood flow is given in terms of cardiac output (liters per minute) rather than cardiac index. Blood volume is recorded as liters. The filled area designates cell volume, the open, plasma volume.
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Fig. 6. Venous pressure in the various categories. A vertical bar through an entry indicates that only venous pressure was measured in that patient. A horizontal bar through an entry indicates that venous pressure was measured in the femoral, rather than antecubital, vein.

with acute myocardial infarction without shock who did not have hypertensive vascular disease was slightly lower than the range of normals here reported. The data are too few to state whether this apparent difference is significant. However, among the nine patients studied in the acute phase and on recovery, the mean arterial pressure with recovery rose definitely in five, and probably significantly in two more. The rise was greatest in the patients with hypertensive vascular disease. The values for mean arterial pressure among nonhypertensive patients recovered from myocardial infarction were the same as those of the normals.

In the patients with shock, mean arterial pressure ranged from 52 to 112 mm. Hg. The two highest values of 84 and 112 mm. Hg were in patients with hypertensive vascular disease, both of whom had values for blood pressure approximating 180/130 mm. Hg prior to the occurrence of myocardial infarction.

Total Peripheral Resistance (fig. 8). The peripheral resistance was increased in two patients with acute myocardial infarction without shock. With recovery, although cardiac output had risen significantly in the whole group, resistance changed significantly in only three of the nine patients (increased in one, decreased in two) for whom values during the stage of acute infarction were available for comparison.

The calculated peripheral resistance was increased in four of the seven patients with shock and was normal in the other three. The patients with arteriosclerosis also had increased total peripheral resistance, in a range similar to that of the patients with myocardial infarction.

Blood Volume. When the individual values are expressed as decimal fractions of the predicted normal (fig. 9) there appears to be little

Fig. 7. Mean arterial pressure in the various categories.

Fig. 8. Total peripheral resistance in the various categories.

Fig. 9. The blood volume, given as a decimal fraction of the predicted normal, of the various patients. A vertical bar through an entry indicates that blood volume only was measured in that patient.
difference between the groups. Blood volume 
values in the patients with uncomplicated 
arteriosclerosis ranged slightly lower than in 
the normals, but the number of observations 
is too small to state whether this is significant. 
The wide range covered by the normal group 
illustrates the deficiencies in predicting blood 
 volume by its quantitative relation to body 
size.

**Discussion**

The data indicate that in patients with acute 
myocardial infarction without shock, the car-
diac output is subnormal, and rises signifi-
cantly with recovery, but not always to normal values. 
When shock is associated with the infarction, 
the cardiac output is very low, significantly 
lower than in patients with acute myocardial 
infarction who are not in shock. However, 3 
of the 10 patients with acute myocardial 
infarction without shock had values for cardiac 
index which fell into the range of the shock 
group, even though the patients without shock 
were selected to exclude those with cardio-
vascular complications (antecedent or con-
comitant congestive heart failure and dys-
rhythmias).

It is, of course, not certain that the reduced 
cardiac output in patients with acute myo-
 cardial infarction (without shock) was due to the 
infarction since the patients were not 
studied prior to their illness. One cannot state 
whether these patients before their myocardial 
infarction had normal cardiac output, or a sub-
normal cardiac output like the arteriosclerotic 
group. However, since cardiac output rose 
significantly on recovery from myocardial in-
 farction, it is a reasonable inference that this 
rise in cardiac output is a measure of the decre-
ment that attended acute myocardial infar-
tion. Thus the occasional finding of a tran-
 siently very low cardiac index in the patient 
with acute myocardial infarction but without 
shock means that following infarction the 
cardiac output may decrease markedly without 
necessarily causing shock.

The subnormal cardiac output found in the 
aged patients with uncomplicated arteri-
sclerosis was unexpected. It does not appear to 
be due to reduced metabolic demand because 
oxygen consumption per unit area of body 
surface averaged the same in both the arterio-
sclerotic and the normal patients (fig. 4).

Variations in the magnitude of cardiac out-
put do not necessarily parallel clinical recovery. 
In two patients cardiac output was measured 
during convalescence from acute myocardial 
infarction (Glaz., Bar., fig. 5). Both had sig-
nificantly higher cardiac output at some time 
during convalescence than on recovery. That 
the level of resting cardiac output does not 
necessarily parallel the apparent adequacy of 
circulation—nor increases in cardiac output, 
clinical improvement—is in accord with an in-
creasing body of observations made on various 
forms of cardiovascular disease.

The values for total peripheral resistance in 
the shocked patients were unexpected in that 
three of the seven were in the normal range. 
Shock, other than the temporary collapse as-
associated with fainting, has been found to be 
associated with increased peripheral resistance. 
It is possible that these three normal values 
are erroneous, due presumably to error in the 
calculated value for cardiac output. However, 
the most important expected source of error in 
the Hamilton method when used in shocked 
patients would be undetected recirculation of the 
indicator. This would yield a falsely low 
value for cardiac output, and thus an errone-
osely high value for calculated peripheral re-
sistance, not low values as reported here. The 
question of the validity of the values for periph-
eral resistance cannot be settled with the data 
available at present.

Venous pressure was measured in these 
patients in order to derive an index of right 
atrial pressure. The increase in the range of 
values for venous pressure found in the patients 
with shock is consistent with increased right 
atrial pressure in these shocked patients, but 
is not conclusive. Although data are lacking, 
it is probable that the considerable pressure 
gradient from the antecubital vein to the atrium 
occasionally noted in shock a may not be so 
exaggerated in the case of the femoral vein 
since the large femoral vein and inferior vena 
cava would probably undergo little reduction 
of cross-sectional area even during marked 
general vascular constriction. Unfortunately,
venous pressure was measured in the femoral vein in only five shocked patients.

Although the validity of measurements of blood volume from T-1824 dilution and the hematocrit is open to serious criticism, the distribution space of this indicator is probably a rough index of the circulating plasma volume. The results of the "blood volume" determinations in this series indicate no apparent systematic abnormality in acute myocardial infarction with or without shock, or on recovery. These findings are not surprising inasmuch as the patients were studied soon after they became ill and there was little time for them to accumulate fluid or become dehydrated.

Determinations of circulating blood volume based on a dilution technic, when done on patients in shock associated with myocardial infarction, probably should be based on several points of the 30-minute indicator disappearance slope, despite inferences drawn from work on patients with traumatic shock. If a single 10-minute sample is used, the indicator may be overly concentrated because the inefficient circulation has retarded its dissemination throughout the vascular compartment, and the calculated value for blood volume may thus be falsely low. This source of error may have contributed to the subnormality of values for blood volume in patients with shock resulting from myocardial infarction reported by Agress.

In view of the trends among the various groups of patients described above, it is possible to propose certain hemodynamic characteristics of the patient with uncomplicated acute myocardial infarction. Cardiac output is subnormal and arterial pressure is usually slightly reduced, generally pari passu. Infrequently there is significant increase in peripheral resistance. Venous pressure and circulating blood volume are normal. With recovery to the point where the patient is permitted full ambulation, there is a significant increase in cardiac output which, however, often remains subnormal. Total peripheral resistance changes inconsistently or not at all. Mean arterial pressure rises slightly or considerably, the increment chiefly being due to the increase in cardiac output. Larger increments in arterial pressure on recovery may be characteristic of the patients with hypertensive vascular disease.

In contrast, the typical patient with myocardial infarction and shock has a cardiac output that is extremely low, significantly lower than in the patient with uncomplicated acute myocardial infarction. Total peripheral resistance as calculated by the technics here used is elevated in some patients, normal in others. Mean arterial pressure is reduced, but not necessarily below the range of normal values: the arterial pressure of a patient with hypertensive vascular disease may drop sharply and yet still be in the normal range. The typical patient with shock resulting from myocardial infarction has a venous pressure that is slightly elevated when compared with the normal range. Circulating blood volume is normal.

The extremely low values for cardiac index noted in the patients with shock suggests that the degree of reduction of cardiac output following myocardial infarction determines whether or not the patient will develop shock, and this has been proposed by some investigators. However, the data reported here indicate that following myocardial infarction cardiac output may fall to very low levels and yet shock need not necessarily develop.

Furthermore, it appears that cardiac output does not necessarily increase progressively in the patient recovering from myocardial infarction. In two patients who were studied during convalescence (fig. 5), values for cardiac output were found during convalescence that were significantly higher than values found after recovery. It seems clear, then, that the magnitude of cardiac output is only one of the factors that determines the clinical appearance and course of the patient with myocardial infarction.

Summary and Conclusions

Statistical analysis of the results of 154 simultaneous determinations of cardiac output by Fick and Hamilton technics, done in this and other laboratories, indicates that the latter yields values that are systematically slightly higher than those yielded by the Fick method. Nevertheless the reproducibility of the Hamilton technic is satisfactory, being within 25 per cent at the 95 per cent confidence level.
By means of technics based on the Hamilton method for determination of cardiac output, hemodynamic studies were made at the bedside on patients with myocardial infarction, on patients with uncomplicated arteriosclerosis, and on patients without cardiovascular disease.

Patients with acute myocardial infarction without shock had a subnormal cardiac output. Following recovery cardiac output was noted to have increased significantly, but not to normal values. Some of these patients with acute myocardial infarction had a very low cardiac output, which appeared to be temporary, but nevertheless they did not appear acutely ill, and were not in shock. The patients with acute myocardial infarction and shock had very low cardiac output, and either normal or elevated peripheral resistance.

Cardiac index in aged patients with arteriosclerosis but without clinical heart disease was significantly less than in the normal patients. These subnormal values were not accountable to a reduced total oxygen consumption.

Circulating “blood volume” was not abnormal in any of the phases of myocardial infarction studied.

Venous pressure tended to be elevated in shock which followed myocardial infarction.

Some factor or factors in addition to the development of a very low cardiac output appears necessary for the development of shock following myocardial infarction.

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Case Summaries

The patients in each group were fairly homogeneous with regard to clinical status, therapy prior to study, and interval between development of illness and time of study. There were certain exceptional patients, however, and because their respective histories are somewhat complex, they are summarized separately here.

Ard. This 62 year old Negro male was first admitted in February 1950, with hypertension and congestive heart failure. He was digitalized, but discontinued digitalis after discharge. He was admitted to another hospital in October 1950, again with hypertension and severe congestive heart failure. He left against advice shortly after admission without having received medication. On Dec. 13, 1950, he was admitted to Bellevue Hospital complaining of severe dyspnea, and of retrosternal pain which had commenced suddenly 18 hours before admission. When first examined at 11 p.m., he was severely dyspneic and orthopneic, apprehensive, and complaining of chest pain. He was in moderate shock, and the blood pressure was unobtainable. There was pulmonary edema and anasarca. Electrocardiogram disclosed evidence of acute anterior wall infarction. The patient was given continuous positive pressure oxygen by inhalation, and morphine and aminophyllin. Between midnight and 11 a.m., December 14, the patient received 750 cc. of plasma and 500 cc. of blood without apparent effect. The patient’s general status gradually deteriorated to that of severe shock without other change. Hemodynamic studies were done at 4 p.m., December 14. The patient expired at 5:30 p.m.

Ore. This 52 year old Costa Rican male, a known hypertensive, had had recent onset of angina after overeating, but no symptoms of cardiac decompensation. At 3 a.m. on Sept. 13, 1951, he noted sudden onset of chest pain, weakness, diaphoresis and syncope. When admitted to the hospital two hours later he had a blood pressure of 80/54, and looked acutely ill. He was sedated with morphine. During the remainder of the night, the blood pressure remained about 80/50, but there was no other evidence of shock, and the patient, having been sedated, looked rather well. A house officer was concerned about the subnormal blood pressure, and administered 250 cc. of plasma at 7 a.m. The patient had a mild pyrogen reaction an hour later, and with this developed some extrasystoles. When hemodynamic studies were done at 10 a.m., September 13, patient appeared to be in good condition, being afebrile and having evidence of neither congestive failure nor of shock. The heart rate was 80 to 100 per minute, with sinus arrhythmia. However, after hemodynamic study he developed ventricular tachycardia. During the next 13 hours the rhythm varied between wandering atrial pacemaker and ventricular tachycardia. During this time the patient was treated with oral doses of procaine amide. However, he died suddenly on September 14.

Case. This 66 year old Italian male sustained clinical myocardial infarction on March 1, 1952, and entered the hospital one day later. He had no symptoms of cardiac decompensation. Examination was negative except for scattered pulmonary basilar rales which were interpreted as hypostatic. Electrocardiogram disclosed right bundle branch block, normal sinus rhythm, and subsequently disclosed progressive changes indicative of myocardial infarction. Moderate fever was present during the first three hospital days. On the morning of March 21, he was found to be in moderate shock. Electrocardiogram then disclosed what probably was a
ventricular tachycardia. This was converted to sinus rhythm shortly after the intravenous administration of 0.5 Gm. procaine amide. Nevertheless the patient remained in moderate shock. Hemodynamic determinations were made for the first time one hour after conversion of the arrhythmia. Subsequently the patient gradually recovered from shock without special treatment. Because the venous pressure was found to be somewhat elevated at the time of the initial hemodynamic study, although the patient had no other evidence of congestive heart failure, at the conclusion of study he was given Digoxin (1.5 mg. within 24 hours, 0.5 mg. daily thereafter, by mouth). Digitalis was discontinued on March 27. Hemodynamic studies were repeated on March 27, and again on May 7, when the patient was ready for discharge.

**Sumario Español**

Producción total cardíaca, presión venosa y arterial, resistencia periférica total y volumen sanguíneo fueron determinados en pacientes con infartos agudos del miocardio, en choque o sin choque, luego de convalecer y en pacientes sin enfermedad cardíaca. La producción cardíaca cae bajo lo normal luego de un infarto del miocardio y aumenta significativamente, aunque no siempre hasta lo normal, al recobrar. Producción cardíaca fué muy baja cuando el infarto fué complicado con choque. Decremento en la producción cardíaca a niveles muy bajos no necesariamente conlleva al desarrollo de choque. La presión venosa tendió ha estar elevada en pacientes en choque como resultado de infartos del miocardio. La producción cardíaca fué subnormal en pacientes con arteriopatía avanzada cardíaca: esto no se pudo atribuir a una consunción disminuida de oxígeno por el cuerpo.

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