Circulatory Changes in Acute Myocardial Infarction

By Robert P. Gilbert, M.D., Melvin Goldberg, M.D., and Joseph Griffin, M.D.

Using the dye dilution technic of Hamilton the cardiac output was measured in 20 patients with acute myocardial infarction. The blood pressure, circulation time, venous pressure, plasma volume, and hematocrit were also determined. Those patients who developed congestive heart failure had a low cardiac output and a prolonged circulation time. These changes were more pronounced in seven patients with sustained shock. One patient who had just recovered from circulatory collapse was found to have a cardiac output of 9.3 liters per minute.

It is well known that congestive heart failure and collapse after an acute myocardial infarction diminish the chances for survival. Yet our understanding of the accompanying circulatory changes has been meager because of the hazards of carrying out complicated studies in such seriously ill patients.

With the introduction of a reliable and safe technic for measuring the cardiac output without moving the patient,\(^1\)\(^-\)\(^3\) it has become possible to make such determinations at the bedside. The present report concerns our results in 20 patients with recent acute myocardial infarction. Thirteen of these patients manifested congestive heart failure or shock, while the remaining seven patients were in no obvious circulatory embarrassment. The cardiac output was determined by the dye dilution technic of Hamilton.\(^1\) The following measurements were also made: circulation time, peripheral venous pressure, arterial pressure, hematocrit, and Evans blue plasma volume. It is hoped that such information will not only enlighten our understanding of the circulatory defects resulting from acute myocardial infarction but will assist in planning effective treatment.

No attempt will be made to review the reports of animal experiments. These experiments have been cited elsewhere.\(^1\)\(^-\)\(^4\) In general they have shown a lowering of the cardiac output without a corresponding drop in arterial pressure. Clinical reports of individual cases have confirmed this in the main.\(^7\)\(^-\)\(^10\) Data obtained by the ballistocardiograph\(^11\) and by pressure-pulse measurements\(^12\)\(^-\)\(^13\) have also indicated lowering of the cardiac output. Recent studies in this laboratory and elsewhere have shown a reduction of the cardiac index after acute myocardial infarction which is more marked in patients with shock.\(^14\)\(^-\)\(^16\)

Material

Successful determinations were completed in 19 male patients and 1 female patient, aged 33 to 78 years. In all cases the clinical diagnosis of an acute myocardial infarction was confirmed by unequivocal electrocardiographic changes. In all but one case the actual infarction had occurred 6 to 48 hours prior to study, as judged from the history. In this one instance the infarction had apparently happened four days previously, although shock had been present only six hours. The diagnosis of congestive heart failure was based solely on the clinical findings, chiefly, the presence of dyspnea, basal rales, and sometimes venous distention and hepatomegaly. Patients were considered to be in a shock-like condition when they manifested weakness, sweating, a cloudy mental state, thready or impalpable radial pulse, and cold extremities. Severe hypotension was present in two cases. The degree of failure or shock was roughly estimated on a 0 to 4 plus scale and is indicated in table 1 along with the age, sex, and probable time interval after infarction.

Methods and Procedure

The cardiac output was determined with Evans blue dye (T-1824) by the method of Hamilton.\(^1\) Collection times were signalled on an electrocardiograph camera which also served to record arterial pressure from a strain gauge manometer.

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Aided by a grant from the Chicago Heart Association.
TABLE 1.—Results in Twenty Cases Ranked by Cardiac Index

<table>
<thead>
<tr>
<th>Case§</th>
<th>Age</th>
<th>Hours after onset</th>
<th>Failure</th>
<th>Shock</th>
<th>Out- come</th>
<th>Cardiac index L./min./M.²</th>
<th>Cardiac output L./min.</th>
<th>Femoral B.P. mm. Hg</th>
<th>Mean B.P. mm. Hg</th>
<th>Circ. Time sec</th>
<th>Venous pressure mm. Hg</th>
<th>Rate</th>
<th>TPR dyn. sec. cm.⁻¹</th>
<th>Plasma Volume ml./kg.</th>
<th>Hematocrit</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. LD</td>
<td>53</td>
<td>30</td>
<td>0</td>
<td>0</td>
<td>L</td>
<td>3.3</td>
<td>6.0</td>
<td>133/77</td>
<td>101</td>
<td>15</td>
<td>140</td>
<td>107</td>
<td>1300</td>
<td>28</td>
<td>48</td>
</tr>
<tr>
<td>2. MA</td>
<td>61</td>
<td>13</td>
<td>0</td>
<td>0</td>
<td>L</td>
<td>3.2</td>
<td>5.2</td>
<td>168/71</td>
<td>108</td>
<td>16</td>
<td>75</td>
<td>82</td>
<td>1600</td>
<td>40</td>
<td>41</td>
</tr>
<tr>
<td>3. AU</td>
<td>61</td>
<td>14</td>
<td>0</td>
<td>0</td>
<td>L</td>
<td>3.1</td>
<td>5.7</td>
<td>163/74</td>
<td>107</td>
<td>20</td>
<td>178</td>
<td>105</td>
<td>1400</td>
<td>41</td>
<td>49</td>
</tr>
<tr>
<td>4. RH</td>
<td>66</td>
<td>48</td>
<td>0</td>
<td>0</td>
<td>L</td>
<td>2.5</td>
<td>4.3</td>
<td>124/63</td>
<td>85</td>
<td>23</td>
<td>120</td>
<td>69</td>
<td>1500</td>
<td>40</td>
<td>42</td>
</tr>
<tr>
<td>5. BR</td>
<td>62</td>
<td>24</td>
<td>0</td>
<td>0</td>
<td>D</td>
<td>2.4</td>
<td>4.5</td>
<td>148/93</td>
<td>118</td>
<td>25</td>
<td>235</td>
<td>96</td>
<td>1900</td>
<td>27</td>
<td>45</td>
</tr>
<tr>
<td>6. DU</td>
<td>72</td>
<td>12</td>
<td>+</td>
<td>0</td>
<td>D</td>
<td>2.2</td>
<td>3.8</td>
<td>111/62</td>
<td>86</td>
<td>+</td>
<td>32*</td>
<td>245</td>
<td>120</td>
<td>1600</td>
<td>31</td>
</tr>
<tr>
<td>7. FE</td>
<td>50</td>
<td>19</td>
<td>0</td>
<td>0</td>
<td>L</td>
<td>2.1</td>
<td>3.9</td>
<td>129/72</td>
<td>91</td>
<td>25</td>
<td>94</td>
<td>73</td>
<td>1750</td>
<td>40</td>
<td>46</td>
</tr>
<tr>
<td>8. PO</td>
<td>42</td>
<td>9</td>
<td>2+</td>
<td>0</td>
<td>D</td>
<td>2.1</td>
<td>3.7</td>
<td>117/82</td>
<td>94</td>
<td>28</td>
<td>188</td>
<td>105</td>
<td>1900</td>
<td>38</td>
<td>43</td>
</tr>
<tr>
<td>9. SK</td>
<td>62</td>
<td>8</td>
<td>3+</td>
<td>0</td>
<td>L†</td>
<td>2.0</td>
<td>3.9</td>
<td>170/98</td>
<td>122</td>
<td>25</td>
<td>210</td>
<td>93</td>
<td>2300</td>
<td>29</td>
<td>53</td>
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<tr>
<td>10. ZA</td>
<td>63</td>
<td>14</td>
<td>0</td>
<td>0</td>
<td>D</td>
<td>1.8</td>
<td>3.1</td>
<td>117/77</td>
<td>91</td>
<td>13</td>
<td>190</td>
<td>101</td>
<td>2100</td>
<td>22</td>
<td>56</td>
</tr>
<tr>
<td>11. CA</td>
<td>61</td>
<td>24</td>
<td>0</td>
<td>0</td>
<td>L</td>
<td>1.7</td>
<td>3.1</td>
<td>118/68</td>
<td>91</td>
<td>15</td>
<td>78</td>
<td>94</td>
<td>2300</td>
<td>25</td>
<td>47</td>
</tr>
<tr>
<td>12. OH</td>
<td>59</td>
<td>8</td>
<td>0</td>
<td>0</td>
<td>D</td>
<td>1.5</td>
<td>2.4</td>
<td>141/88</td>
<td>111</td>
<td>33*</td>
<td>110</td>
<td>105</td>
<td>3600</td>
<td>35</td>
<td>46</td>
</tr>
<tr>
<td>13. HA</td>
<td>59</td>
<td>18</td>
<td>2+</td>
<td>0</td>
<td>+</td>
<td>1.4</td>
<td>2.6</td>
<td>124/88</td>
<td>100</td>
<td>38</td>
<td>374</td>
<td>133</td>
<td>3000</td>
<td>29</td>
<td>55</td>
</tr>
<tr>
<td>14. HE</td>
<td>50</td>
<td>46</td>
<td>0</td>
<td>0</td>
<td>+</td>
<td>1.2</td>
<td>2.1</td>
<td>102/90†</td>
<td>96†</td>
<td>40</td>
<td>270</td>
<td>320</td>
<td>590</td>
<td>30</td>
<td>59</td>
</tr>
<tr>
<td>15. MA</td>
<td>64</td>
<td>96</td>
<td>0</td>
<td>0</td>
<td>+</td>
<td>1.1</td>
<td>2.2</td>
<td>114/57</td>
<td>70</td>
<td>30</td>
<td>223</td>
<td>51</td>
<td>2900</td>
<td>46</td>
<td>46</td>
</tr>
<tr>
<td>16. RO</td>
<td>52</td>
<td>6</td>
<td>3+</td>
<td>2+</td>
<td>D</td>
<td>1.1</td>
<td>1.9</td>
<td>111/67</td>
<td>87</td>
<td>45</td>
<td>179</td>
<td>77</td>
<td>3300</td>
<td>35</td>
<td>54</td>
</tr>
<tr>
<td>17. WO</td>
<td>58</td>
<td>49</td>
<td>3+</td>
<td>3+</td>
<td>D</td>
<td>0.8</td>
<td>1.5</td>
<td>102/52</td>
<td>81</td>
<td>42</td>
<td>220</td>
<td>114</td>
<td>3900</td>
<td>35</td>
<td>47</td>
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<tr>
<td>18. GR</td>
<td>75</td>
<td>16</td>
<td>3+</td>
<td>3+</td>
<td>D</td>
<td>0.8</td>
<td>1.2</td>
<td>65/43</td>
<td>54</td>
<td>60</td>
<td>65</td>
<td>88</td>
<td>3400</td>
<td>47</td>
<td>44</td>
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<td>19. LO</td>
<td>78</td>
<td>6</td>
<td>4+</td>
<td>0</td>
<td>D</td>
<td>0.6</td>
<td>0.9</td>
<td>45/18</td>
<td>25</td>
<td>77</td>
<td>175</td>
<td>53</td>
<td>1600</td>
<td>44</td>
<td>44</td>
</tr>
</tbody>
</table>

Averages.................................................. 1.8  3.3
S.D..................................................... 0.8  1.6

* Circulation time from peak of dye curve. † Patient left hospital against advice on tenth day. ‡ Blood pressures by auscultory method. § All patients males except 20. LO.

In order to estimate the appropriate rate of arterial sampling, the arm-to-tongue circulation time was first measured with Decholin through a calibrated needle in the antecubital vein. This needle was kept open by periodic flushing from the venous pressure manometer and was used later for the dye injection. A 15 or 17 gauge needle was inserted in the femoral artery after procainization. A three-way stopcock connected this needle to the strain gauge or to a curved metal tube for delivering blood samples. Two ml. of 0.5 per cent Evans blue dye (T-1824) were injected from a calibrated 2 ml. tuberculin type syringe. Arterial samples were then collected at 2 to 5 second intervals with a straight rack of tubes containing heparin powder. A single 10-minute sample was taken for estimation of the plasma volume. The plasma dye concentrations were read in the Evelyn photoelectric microcolorimeter. Hematocrits were measured in duplicate in Wintrobe tubes spun for 30 minutes at 3500 revolutions per minute at a 15-cm. radius (axis to tube tip).

Surface area was estimated by nomogram from the height and weight values furnished by the patient. Mean arterial pressures were measured either by planimeter or by averaging pressure values read from the tracing at 0.04 second intervals. The total peripheral resistance was calculated by the formula:

\[
\text{Total peripheral resistance} = \frac{(\text{Mean arterial pressure} - \frac{1}{2} \text{venous pressure}) \times 1832}{\text{cc. of cardiac output/second}}
\]

The peripheral venous pressure was halved to approximate the right atrial pressure, although it was recognized that the pressure gradient may be diminished in congestive failure.\textsuperscript{18}

**Results**

The results are ranged in table 1 in order of descending cardiac index. It is evident that with decreasing values for the cardiac index, the following changes occur: (1) increasing mortality rate, (2) appearance of failure and then shock, (3) increasing circulation time, and (4) usually increasing total peripheral resistance. The average cardiac index for all cases was 1.8 ± 0.8 liters per minute per square meter of body surface area. The mean venous pressure was elevated and the average circulation time prolonged.

In figure 1 the mean femoral arterial pressures are plotted against the corresponding
cardiac indices. The arterial pressures are seen to lie within the normal range despite low indices except in three patients with extreme lowering of the cardiac index. Accordingly, the calculated values for the total peripheral resistance are increased in most instances. Figure 2 illustrates the expected lengthening of the Decholin circulation time in patients with low cardiac indices (r = -.765, p < .01). The patients who survived had shorter circulation times. No correlation was found between the cardiac index and the venous pressure (r = -.20, p > .40). The venous pressure was elevated in two patients without clinically apparent heart failure and was normal in three patients with obvious cardiac insufficiency. Pressure on the right upper quadrant of the abdomen caused the venous pressure to rise in some patients without signs of failure and usually produced a rise in those with obvious failure.

Three subgroups are compared in table 2. The first group comprises those patients with no clinical evidence of either failure or shock. Those in the second group showed signs of heart failure. The cases in the third group were in a shock-like condition, and all had signs of congestive failure. In addition to significantly lower cardiac indices in the patients with failure and shock, the circulation times are also seen to be lengthened. No differences are apparent for the average hematocrits or plasma volumes, and the differences in mean venous pressure values are not significant because of the large deviations

### Table 2.—Average Figures for the Three Groups

<table>
<thead>
<tr>
<th>Group</th>
<th>No. Pets</th>
<th>Cardiac Index L/min./M$^2$</th>
<th>Circ. Time sec</th>
<th>Venous Pressure mm. HgO</th>
<th>Hematocrit</th>
<th>Plasma Volume ml./Kg</th>
</tr>
</thead>
<tbody>
<tr>
<td>Group I</td>
<td>No fail.</td>
<td>7</td>
<td>2.61</td>
<td>19.0</td>
<td>130</td>
<td>45.</td>
</tr>
<tr>
<td></td>
<td>No shock</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Group II</td>
<td>Failure</td>
<td>6</td>
<td>1.9</td>
<td>24.8</td>
<td>167</td>
<td>50.</td>
</tr>
<tr>
<td></td>
<td>2-S.E.</td>
<td></td>
<td>0.46</td>
<td>3.4</td>
<td>44.3</td>
<td>2.3</td>
</tr>
<tr>
<td>Group III</td>
<td>Shock</td>
<td>7</td>
<td>1.0</td>
<td>47.4</td>
<td>215</td>
<td>47.1</td>
</tr>
<tr>
<td></td>
<td>2-S.E.</td>
<td></td>
<td>0.21</td>
<td>12.2</td>
<td>83.4</td>
<td>4.4</td>
</tr>
</tbody>
</table>

Fig. 1. Cardiac index (liters/min./M$^2$) and mean femoral arterial pressure in 20 patients with acute myocardial infarction.

Fig. 2. Cardiac index (liters/min./M$^2$) and circulation time in 20 patients with acute myocardial infarction.

Fig. 3. Comparison of cardiac index (liters/min./M$^2$) in three subgroups. Presence of failure or shock determined on clinical grounds. Other data in table 2.
from the mean. The cardiac indexes for these three groups are portrayed in figure 3. While there is some overlapping, the cardiac index is usually lower when there is heart failure and still lower in shock. The open circles indicate patients who survived the hospitalization period, usually those patients who had fairly normal outputs. The strikingly high mortality rate can be explained by the fact that there was a tendency to select for study those patients who were more seriously affected.

The individual findings in shock are shown in more detail by the last seven cases in table 1, which can be compared with the average values given in table 2. While the femoral arterial pressures were severely depressed in only two cases, the auscultatory pressures obtained in the arm were often lower than those obtained by direct recording from the femoral artery. In other respects these patients showed the clinical picture of shock described above, and differed from each other only quantitatively. Three patients with normal pressure had histories of hypertension. All had been in a shock-like condition for at least six hours prior to study. Only the first of the patients with shock survived longer than 12 hours. The third patient was studied four days after the onset of pain, five to six hours after going into shock. They all were dyspneic and most had rales. The extreme reduction of their cardiac indices may have been exaggerated by the hidden recirculation error, particularly in the second, fourth and sixth cases. Curves in these cases had overly rounded peaks and slow downslopes. However in one case which was not included in the table because the data were incomplete, the cardiac output was 9.3 liters per minute and the blood pressure 138/78 some 12 hours after recovery from circulatory collapse. On admission the blood pressure was unobtainable. No pressor therapy was used.

The circulation time in the shock group was uniformly prolonged, and the venous pressure was normal in only one instance. The hematocrits were not remarkable. The plasma volume was not found to be especially low in those patients with shock despite the fact that slow mixing in such cases might be expected to give higher dye concentrations at ten minutes. Despite the presence of shock and low cardiac indices, two cases showed no increase in heart rate. Auriculoventricular block accounted for the slow rates in cases 16 and 20.

Discussion

These results agree with those reported by Freis and Smith and described less completely by others. Following acute myocardial infarction the cardiac index is usually low. The degree of reduction is roughly proportional to the clinical severity of the attack and is most marked in patients with sustained shock. The relationship is by no means an absolute one, as exemplified by patient 12, who appeared to be in good condition in spite of a cardiac index of only 1.7. Smith reported two similar cases. Except in those cases with very low cardiac indexes the blood pressure tends to be well sustained, indicating overall vasoconstriction. There is no significant correlation between a high venous pressure and a low cardiac index, nor between a high venous pressure and a long circulation time. The longer circulation times were associated with lower cardiac indices. Quite possibly this association accounts for the usefulness of the circulation time as a guide to the severity of myocardial infarction. The patients with shock were not found to have particularly low plasma volumes, contrary to the data shown by Agress and co-workers and hinted at by others. Resolution of this question awaits a more precise method for determining plasma volumes. It is quite possible that the lower plasma volume found in some cases is the result not only of dehydration but of a high venous pressure. We found no significant correlation between the venous pressure and the plasma volume (r = -.259, p > .30) but there was a correlation coefficient of 0.40 (p < .10 .05) between the venous pressure and the hematocrit and a significant correlation between high hematocrit and low plasma volume (r = -.533, p < .01). In two cases followed serially the hematocrit dropped from 55 to 44, and from 44 to 39 in several days, while the venous pressure changed from 384 to 215 and from 205 to 210 mm. saline.

The genesis of shock in acute myocardial infarction cannot yet be precisely described
from the data at hand. Harrison, Stead, Boyer, and others have held that it results from inability of the heart to deliver enough blood despite a more than adequate venous supply. This view is strongly supported by our data and that of others. Various workers have suggested that shock after infarction may also result from peripheral vasodilatation. The one patient with a cardiac output of 9.3 liters per minute after recovery from circulatory collapse could represent such an occurrence. So could the one with a cardiac output of 5.3 liters per minute and a blood pressure of 66/44 reported by Stead. One of Freis' patients was in shock despite a cardiac index of 2.4. It is quite possible that some of the patients who were found to have a low cardiac output several hours after the onset of shock had a considerably higher output at the beginning. Prolonged hypotension is well known to seriously affect the myocardium of previously normal animals, not to mention the hearts of patients with generalized coronary artery disease. It is likely that many patients who recover spontaneously from brief periods of collapse shortly after the onset of infarction would be found to have a normal or even elevated cardiac output at the time of collapse. We have no data from such cases. It does not seem likely that there is enough reduction of plasma volume to contribute significantly to shock. While both central and peripheral factors may operate initially to produce shock in acute myocardial infarction, it seems clear that in sustained shock there is a low output because of a failing heart.

Insofar as reduction in the cardiac index is the chief circulatory defect in sustained shock due to acute myocardial infarction, it seems logical that the output should be supported as well as possible. The obvious drugs for this purpose are the digitalis glycosides and the xanthines. Aminophylline in acute myocardial infarction seems to have such an effect. Measures to restore aortic pressure might increase the cardiac output by improving coronary perfusion of uninvolved and marginally infarcted areas. Some of the reports concerning pressor agents and infusions are encouraging. Elucidation of this problem requires more objective data concerning the dynamic effects of such measures in myocardial infarction.

SUMMARY

1. Cardiac index, blood pressure, venous pressure, circulation time, plasma volume, and hematocrit were determined in 20 patients soon after acute myocardial infarction.

2. Lowering of the cardiac index and prolongation of the circulation time were roughly proportional to the clinical severity of the attack.

3. Vasoconstriction tended to sustain the blood pressure as evidenced by increased values for the peripheral resistance.

4. In patients with sustained shock the cardiac index was particularly low, though no major plasma volume changes were evident in this group. One patient who had recovered from profound circulatory collapse was found to have a cardiac output of 9.3 liters per minute.

5. The possibility of normal or high output at the onset of shock is discussed.

ACKNOWLEDGMENTS

The authors gratefully acknowledge the work of Stephen Aldrich, M.D. and Lynn Anderson, M.D. who participated in the early phases of this project.

SUMARIO ESPAÑOL

1. En 20 pacientes con infarto del miocardio reciente se determinó el índice cardíaco, la presión arterial, presión venosa, tiempo de circulación, volumen plasmático y hematocrito.

2. Decremento del índice cardíaco y prolongación del tiempo de circulación fueron aproximadamente proporcionales a la severidad clínica del ataque.

3. La vasoconstricción tendió a sostener la presión arterial como fué comprobado por los valores aumentados en resistencia periférica.

4. En pacientes en choque sostenido el índice cardíaco fué particularmente bajo, aunque cambios significativos en el volumen plasmático no fueron evidentes en este grupo. Un paciente que había recobrado de un colapso circulatorio profundo se encontró tener una producción cardíaca de 9.13 litros por minuto.

5. La posibilidad de producción normal o alta al comienzo del choque se discute.
REFERENCES


Circulatory Changes in Acute Myocardial Infarction
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Circulation. 1954;9:847-852
doi: 10.1161/01.CIR.9.6.847

Circulation is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
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Print ISSN: 0009-7322. Online ISSN: 1524-4539

The online version of this article, along with updated information and services, is located on the World Wide Web at:
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