Hemodynamic Effects of Artificial Pacing in Complete Heart Block Complicating Acute Myocardial Infarction

By B. W. Lassers, M.B. (Edin.), M.R.C.P., M.R.C.P.E.,
J. L. Anderton, M.B. (Edin.), M.R.C.P.E.,
M. George, M.B. (Edin.), M.R.C.P., M.R.C.P.E.,
A. L. Muir, M.B. (Edin.), M.R.C.P.E., and

SUMMARY

The hemodynamic changes associated with complete heart block complicating acute myocardial infarction and the effects of artificial pacing at various rates on these measurements were studied in 13 patients. Studies were performed on the first day of complete block in all patients and were repeated on subsequent days in six.

With two exceptions cardiac output was increased by pacing. In patients paced at progressively increasing ventricular rates, the rate associated with the maximal cardiac output exceeded 100/min, with one exception. However, an adequate response usually occurred between 80 and 90/min. Stroke volume fell as the rate was increased with pacing in most patients, but rose in some, suggesting improved myocardial performance. Systemic blood pressure increased, and the change paralleled cardiac output changes fairly closely as pacing rates were increased. The blood pressure did not, however, reflect reliably the absolute level of cardiac output either in heart block or after the return of sinus rhythm. Systemic vascular resistance was high in most patients with low cardiac outputs and fell with pacing at maximal output rates. There was no consistent change in mean right atrial or mean pulmonary arterial pressure with pacing during the period of study. Tension time index was low in heart block, and increased considerably with pacing, implying a corresponding increase in myocardial oxygen requirements.

Pacing improved signs of depressed mental function and poor skin circulation which were invariably associated with a severe reduction in cardiac output. These signs were found to be superior to heart rate and blood pressure in clinical evaluation of the adequacy of the cardiac output in heart block and the response to pacing.

Additional Indexing Words: Atrioventricular block Bradycardia Tension time index
Right ventricular failure

COMPLETE HEART BLOCK (CHB) developing after acute myocardial infarction is a complication which carries a high mortality. Although heart block is almost always transient in those who survive, the hemodynamic consequences of bradycardia and the danger of ventricular asystole during the period of disordered atrioventricular conduction jeopardize the patient's chance of survival. With the development of intensive coronary care, temporary transvenous endocardial pacemaking has become a widely used method of treatment of this disturbance of conduction.1-4 Rational management by pacemaking

From the Coronary Care Unit, Royal Infirmary, Edinburgh, Scotland.
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### Clinical Details

**Table 1**

<table>
<thead>
<tr>
<th>Patient</th>
<th>Sex</th>
<th>Age (yr)</th>
<th>Site of infarct</th>
<th>Rhythm</th>
<th>Lowest rate in CHB</th>
<th>Lowest BP in CHB</th>
<th>Clinical assessment</th>
<th>Duration of advanced heart block (hr)</th>
<th>Outcome and rhythm at discharge</th>
</tr>
</thead>
<tbody>
<tr>
<td>W.H.</td>
<td>M</td>
<td>52</td>
<td>Inferior</td>
<td>CHB</td>
<td>60</td>
<td>110/70</td>
<td>Normal</td>
<td>0</td>
<td>60, Alive—SR</td>
</tr>
<tr>
<td>W.B.</td>
<td>M</td>
<td>59</td>
<td>Inferior</td>
<td>CHB</td>
<td>47</td>
<td>75/60</td>
<td>Confusion, inattention</td>
<td>0</td>
<td>184, Alive—SR</td>
</tr>
<tr>
<td>M.F.</td>
<td>F</td>
<td>70</td>
<td>Inferior</td>
<td>CHB + nodal tachy.</td>
<td>42</td>
<td>85/-</td>
<td>Stokes-Adams</td>
<td>0</td>
<td>108, Alive—SR</td>
</tr>
<tr>
<td>G.R.</td>
<td>F</td>
<td>59</td>
<td>Inferior</td>
<td>CHB</td>
<td>44</td>
<td>115/65</td>
<td>Inattention</td>
<td>+</td>
<td>172, Alive—SR</td>
</tr>
<tr>
<td>J.N.</td>
<td>M</td>
<td>57</td>
<td>Anterior + old inferior</td>
<td>CHB</td>
<td>31</td>
<td>65/-</td>
<td>Stokes-Adams</td>
<td>+</td>
<td>174, Alive—SR</td>
</tr>
<tr>
<td>W.D.</td>
<td>M</td>
<td>77</td>
<td>Inferior</td>
<td>CHB</td>
<td>40</td>
<td>110/60</td>
<td>Mild confusion</td>
<td>0</td>
<td>56, Alive—SR</td>
</tr>
<tr>
<td>C.O.</td>
<td>M</td>
<td>54</td>
<td>Inferior</td>
<td>CHB</td>
<td>52</td>
<td>85/50</td>
<td>Normal</td>
<td>+</td>
<td>27, Alive—SR</td>
</tr>
<tr>
<td>A.S.</td>
<td>M</td>
<td>84</td>
<td>Inferior</td>
<td>CHB + AF</td>
<td>37</td>
<td>75/65</td>
<td>Confusion, inattention</td>
<td>+</td>
<td>15, Died in CHB</td>
</tr>
<tr>
<td>C.C.</td>
<td>F</td>
<td>82</td>
<td>Inferior</td>
<td>CHB</td>
<td>30</td>
<td>80/-</td>
<td>Confusion, inattention</td>
<td>+</td>
<td>153, Alive—SR</td>
</tr>
<tr>
<td>J.R.</td>
<td>M</td>
<td>66</td>
<td>Inferior</td>
<td>CHB to 2*</td>
<td>40</td>
<td>155/75</td>
<td>Faintness, inattention</td>
<td>+</td>
<td>4, Alive—SR</td>
</tr>
<tr>
<td>J.D.</td>
<td>M</td>
<td>48</td>
<td>Inferior</td>
<td>CHB to 2*</td>
<td>30</td>
<td>100/70</td>
<td>Normal</td>
<td>+</td>
<td>3, Alive—SR</td>
</tr>
<tr>
<td>R.C.</td>
<td>M</td>
<td>63</td>
<td>Inferior</td>
<td>CHB</td>
<td>22</td>
<td>80/60*</td>
<td>Restless, confusion</td>
<td>+</td>
<td>34, Alive—AF to SR (cardioverted)</td>
</tr>
<tr>
<td>J.M.</td>
<td>M</td>
<td>71</td>
<td>Inferior</td>
<td>CHB</td>
<td>52</td>
<td>45/-</td>
<td>Semi-conscious</td>
<td>+</td>
<td>19, Died in CHB</td>
</tr>
</tbody>
</table>

*Known hypertensive.

Abbreviations: RVF = right ventricular failure (jugular venous pressure above clavicle at 45°); LVF = left ventricular failure (dyspnea at rest, rales persisting after coughing, radiographic evidence of pulmonary venous congestion); NT = not taken; PM = no radiogram taken, but postmortem evidence of pulmonary congestion; CHB = complete heart block; 2° = second degree heart block; SR = sinus rhythm; and AF = atrial fibrillation.
must be based on knowledge of the circulatory changes associated with heart block and with artificial pacing in the particular circumstances of acute myocardial infarction. Although the hemodynamic effects of pacing in chronic heart block are well documented, there have been no studies on acute myocardial infarction. This report describes the hemodynamic changes associated with advanced heart block complicating acute myocardial infarction and their response to asynchronous ventricular pacing at varying rates.

**Methods**

**Clinical Material**

Thirteen patients with atrioventricular block complicating acute myocardial infarction were studied in the Coronary Care Unit of the Royal Infirmary, Edinburgh (table 1). The clinical diagnosis of myocardial infarction was confirmed in all cases by electrocardiographic and serum enzyme changes. All had developed complete heart block (CHB) within 4.4 days of the onset of symptoms of infarction; the mean duration between the onset of symptoms and the development of CHB was 28 hr. All patients were studied within 24 hr of the onset of CHB and most within 6 hr. In all patients CHB has been present within 30 min of the beginning of the study: 11 remained in CHB throughout the studies on the first day, but two developed second degree blocks immediately prior to study. No patient had received digitalis, a diuretic, a sympathomimetic agent, or an anti-arrhythmic drug. Analgesics had not been given within 3 hr of any study and only one patient had received oxygen within 1 hr of the study.

Six patients were studied with and without pacing on subsequent days while still in heart block, and five of these were studied after return of sinus rhythm. Two patients died while in CHB: one of shock in spite of effective pacing and one of asystole unresponsive to pacing. Normal A-V conduction returned in the remaining 11. These 11 patients survived to be discharged from the hospital.

During the 5-month period in which these patients were investigated, eight other patients with CHB complicating acute infarction were managed in the coronary care unit. Seven of these eight patients died, however, before a pacing

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**Table 2**

<table>
<thead>
<tr>
<th>Patient</th>
<th>Day of HB</th>
<th>Rate HB</th>
<th>Rate paced*</th>
<th>Cardiac index (L/min/m²)</th>
<th>Stroke index (ml/m²)</th>
<th>Mean art. BP (mm Hg)</th>
</tr>
</thead>
<tbody>
<tr>
<td>W.H.</td>
<td>1</td>
<td>60</td>
<td>75</td>
<td>2.22</td>
<td>3.45</td>
<td>37</td>
</tr>
<tr>
<td>W.H.</td>
<td>2</td>
<td>49</td>
<td>75</td>
<td>2.10</td>
<td>3.10</td>
<td>43</td>
</tr>
<tr>
<td>W.H.</td>
<td>3</td>
<td>59</td>
<td>92</td>
<td>4.13</td>
<td>4.42</td>
<td>70</td>
</tr>
<tr>
<td>W.B.</td>
<td>1</td>
<td>47</td>
<td>75</td>
<td>1.26</td>
<td>1.95</td>
<td>27</td>
</tr>
<tr>
<td>W.B.</td>
<td>2</td>
<td>53</td>
<td>81</td>
<td>1.51</td>
<td>1.80</td>
<td>29</td>
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<tr>
<td>M.F.</td>
<td>1</td>
<td>98</td>
<td>120</td>
<td>1.48</td>
<td>2.52</td>
<td>15</td>
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<tr>
<td>G.R.</td>
<td>1</td>
<td>44</td>
<td>80</td>
<td>1.29</td>
<td>2.47</td>
<td>30</td>
</tr>
<tr>
<td>G.R.</td>
<td>2</td>
<td>52</td>
<td>104</td>
<td>2.98</td>
<td>2.67</td>
<td>57</td>
</tr>
<tr>
<td>G.R.</td>
<td>3</td>
<td>62</td>
<td>115</td>
<td>3.17</td>
<td>3.41</td>
<td>51</td>
</tr>
<tr>
<td>J.N.</td>
<td>1</td>
<td>31</td>
<td>103</td>
<td>—</td>
<td>(2.57)</td>
<td>—</td>
</tr>
<tr>
<td>W.D.</td>
<td>1</td>
<td>51</td>
<td>102</td>
<td>2.92</td>
<td>3.33</td>
<td>57</td>
</tr>
<tr>
<td>C.O.</td>
<td>1</td>
<td>52</td>
<td>104</td>
<td>3.13</td>
<td>3.89</td>
<td>57</td>
</tr>
<tr>
<td>A.S.</td>
<td>1</td>
<td>38</td>
<td>106</td>
<td>—</td>
<td>(1.64)</td>
<td>—</td>
</tr>
<tr>
<td>C.C.</td>
<td>1</td>
<td>61</td>
<td>107</td>
<td>1.91</td>
<td>2.96</td>
<td>21</td>
</tr>
<tr>
<td>J.R.</td>
<td>1</td>
<td>50</td>
<td>90</td>
<td>1.81</td>
<td>1.85</td>
<td>36</td>
</tr>
<tr>
<td>J.D.</td>
<td>1</td>
<td>41</td>
<td>115</td>
<td>2.31</td>
<td>3.70</td>
<td>50</td>
</tr>
<tr>
<td>R.C.</td>
<td>1</td>
<td>40</td>
<td>83</td>
<td>2.03</td>
<td>2.81</td>
<td>51</td>
</tr>
<tr>
<td>J.M.</td>
<td>1</td>
<td>52</td>
<td>83</td>
<td>1.21</td>
<td>1.44</td>
<td>24</td>
</tr>
<tr>
<td>Mean</td>
<td></td>
<td>52</td>
<td>95</td>
<td>2.22</td>
<td>2.85</td>
<td>41</td>
</tr>
<tr>
<td>sp</td>
<td></td>
<td>14</td>
<td>15</td>
<td>0.84</td>
<td>0.83</td>
<td>15.8</td>
</tr>
<tr>
<td>P</td>
<td></td>
<td></td>
<td></td>
<td>&lt;0.001</td>
<td>&lt;0.005</td>
<td>&lt;0.001</td>
</tr>
</tbody>
</table>

*Rate associated with maximal cardiac index.
Abbreviations: HB = heart block; RA = right atrial; Pac = paced; 0 = not elevated clinically; NS = not significant; measurements were not made.
Maximal Cardiac Index

<table>
<thead>
<tr>
<th>Systemic vasc. resist. (dyne·sec·cm⁻²/m²)</th>
<th>Mean pressure (mm Hg)</th>
<th>Left vent. work (kg·m/min/m²)</th>
<th>Tension time index (mm Hg·sec/min)</th>
</tr>
</thead>
<tbody>
<tr>
<td>HB</td>
<td>Pac</td>
<td>HB</td>
<td>Pac</td>
</tr>
<tr>
<td>2634</td>
<td>1922</td>
<td>10</td>
<td>9</td>
</tr>
<tr>
<td>3186</td>
<td>2296</td>
<td>12</td>
<td>11</td>
</tr>
<tr>
<td>1735</td>
<td>1513</td>
<td>14</td>
<td>13</td>
</tr>
<tr>
<td>3651</td>
<td>3405</td>
<td>21</td>
<td>20</td>
</tr>
<tr>
<td>3761</td>
<td>3734</td>
<td>20</td>
<td>18</td>
</tr>
<tr>
<td>5243</td>
<td>2984</td>
<td>16</td>
<td>17</td>
</tr>
<tr>
<td>4775</td>
<td>3174</td>
<td>16</td>
<td>20</td>
</tr>
<tr>
<td>1986</td>
<td>2966</td>
<td>17</td>
<td>18</td>
</tr>
<tr>
<td>1994</td>
<td>2322</td>
<td>16</td>
<td>17</td>
</tr>
</tbody>
</table>

— (2432) | 24 | 25 | 10 | 8 | — | (1.64) |
| 1712 | 2126 | 11 | 12 | 7 | 7 | 1.54 | 3.87 |
| 1256 | 1504 | 24 | 25 | 19 | 15 | 1.04 | 3.24 |

— (4728) | — (25) | + | (13) | — | (1.55) |
| 2765 | 2963 | 20 | 20 | 8 | 6 | 0.82 | 3.27 |
| 3530 | 3806 | 15 | 17 | 7 | 10 | 0.75 | 1.22 |
| 3061 | 2604 | 16 | 22 | 14 | 11 | 1.31 | 6.17 |
| 3114 | 3455 | 29 | 31 | 12 | 9 | 0.91 | 2.24 |
| 2426 | 2147 | 21 | 23 | 10 | 12 | 0.19 | 0.35 |
| 2927 | 2684 | 18 | 19 | 10 | 9 | 0.94 | 2.91 |
| 1104 | 740 | 5.1 | 5.6 | 4.6 | 5.8 | 0.43 | 1.90 |

NS | NS | NS | <0.02 | <0.001

PA = pulmonary artery; and + = clinically raised. Figures in parentheses not included in mean values as complete

Techniques and Measurements

In nine patients, cardiac outputs were calculated by the Fick principle. In the remaining four patients, cardiac outputs were determined by dye-dilution methods. Bipolar pacing electrodes (U. S. Catheter Co.) were inserted percutaneously into the right subclavian vein and manipulated under fluoroscopic control to the apex of the right ventricle. This procedure, as well as all other studies, were carried out at the bedside in the coronary care unit. The electrode was connected to a variable rate, variable current, continuously discharging external pulse generator incorporated in the Sanborn Visomonitor unit. A single lumen, or in the case of the dye-dilution studies, a triple lumen catheter was then advanced from an antecubital vein until its tip lay in the main pulmonary artery. Right atrial pressures were recorded through the proximal lumen of the triple lumen catheter or by means of a third catheter passed via an arm vein or the subclavian vein. Systemic arterial pressures and blood samples were obtained through a nylon catheter inserted percutaneously into a brachial artery and advanced centrally. Pressures were transduced with Sanborn 267B manometers or Bell and Howell 4-327-L221 strain-gauge manometers and recorded either on a Mingograf 24 B ink-jet recorder or on a Devices M4 direct-writing recorder. Zero reference level for the manometers was taken as 5 cm below the level of the manubrium sterni.

Expired air was breathed through a dry gas meter, the volume recorded in liters on a direct-writing recorder, and the oxygen and carbon dioxide content analyzed by the Lloyd-Haldane method. During a 5-min expired air collection period, arterial and mixed venous blood samples were withdrawn continuously and analyzed for oxygen tension, carbon dioxide tension, and pH. The oxygen saturation of the blood was derived from Dill's desaturation table and the oxygen content was calculated from the saturation and the hemoglobin capacity.

For the dye-dilution studies, indocyanine green was used as indicator and injected into the right ventricle via the middle lumen of the triple-lumen catheter. Blood was sampled from the arterial catheter through a Waters Xc-302 cuvette-densitometer and curves were recorded on a Servoscribe direct-writing ink recorder. Calibration
was carried out at the end of each study by drawing known concentrations of dye in samples of the patient's blood through the cuvette.

The following formulae were employed:

\[
\begin{align*}
\text{Systemic vascular resistance} & = \frac{\text{pressure}}{\text{flow}} \\
\text{Left ventricular external work} & = \frac{\text{pressure}}{\text{flow}} \\
\text{Tension time index} & = \frac{\text{pressure} \times \text{flow}}{\text{pressure}}
\end{align*}
\]

where MSEP is mean systolic ejection pressure obtained by planimetry. When direct right atrial pressure was not recorded, it was considered for the purpose of calculation of systemic vascular resistance to be 6 mm Hg if the jugular venous pressure was not elevated clinically. If the venous pressure was elevated, a clinical estimate was made and this figure was substituted.

Statistical analysis was carried out employing Student's t-test with the method of paired comparisons.

**Plan of Investigation**

**Fick Principle Cardiac Output Studies**

Whenever possible, control measurements were made in heart block and pacing was then performed for 20 to 30 min before the measurements were repeated. Serial studies were performed at three or four different rates, that is, in the range: (1) 60 to 80/min; (2) 81 to 100/min; (3) 101 to 120/min; and (4) 121 to 140/min. In each instance, observations were repeated after 20 to 30-min stabilization at the new rate of pacing.

**Dye-Dilution Studies**

In the four patients in whom the cardiac output was measured by dye-dilution, continuous intravascular pressures were recorded and dye-dilution curves were performed at 5-min intervals throughout the study. Initial control observations were made for 15 min. The pacemaker was then turned on and measurements continued for a further 15 min when the rate was increased.

**Results**

**Cardiac Index**

In two cases (J.N. and A.S.) base-line measurements in CHB were not made because the patients required pacing urgently. The remaining 11 patients were studied in heart block on the first day. Three of these were studied again while still in heart block on subsequent days. The mean cardiac index in these 11 patients studied on 16 occasions in heart block was \(2.22 \pm 0.84\) L/min/m². This increased by 28% with pacing at the rate associated with the highest cardiac index (maximal output rate) to \(2.85 \pm 0.83\) L/min/m² \((P < 0.001)\) (table 2, fig. 1). In nine patients the cardiac index in heart block was less than \(2.50\) L/min/m². In these nine patients pacing at maximal output rates resulted in an increase of 46% from a mean value of \(1.74 \pm 0.40\) L/min/m² to \(2.54 \pm 0.72\) L/min/m² \((P < 0.001)\). The cardiac index was greater than \(2.50\) L/min/m² on the first day of CHB in two patients and had risen above \(2.50\) L/min/m² in two others by the second and third days, respectively. In these patients the increase in cardiac index from a mean level in heart block of \(3.27 \pm 0.49\) to \(3.54 \pm 0.65\) L/min/m² with pacing at maximal output rates was not statistically significant.

In the 11 patients studied on 16 occasions on the first and subsequent days of heart block, cardiac index increased with pacing in 14 instances, fell in one instance, and did not change in another. The single patient (G.R.) whose cardiac index fell with pacing on the second day of CHB had an index of \(2.98\) L/min/m² with a ventricular rate of 52/min. This fell with pacing at a single rate of 104/min to \(2.67\) L/min/m². However, this patient had previously had an index of \(1.29\) L/min/m² in CHB with a ventricular rate of 44/min on the first day of CHB which had increased to \(2.47\) L/min/m² with pacing at 80/min. The patient (J.R.) whose index failed to increase significantly was in second degree heart block at the time of study with a cardiac index of \(1.81\) L/min/m² and a ventricular rate of 50/min.

Patient R.C. was studied for a control period of 15 min in heart block and then paced at 83/min for 65 min with repeated cardiac
output measurements throughout the 80-min period of study (fig. 2). The maximal response to pacing at this rate was reached within 10 min and outputs remained steady over the 65-min period.

Figure 3A shows the effect of increasing ventricular rate on the cardiac index. It includes only the eight patients paced at three or more rates. In two of these (J.N. and A.S.) measurements were not made in heart block. In six patients there was a progressive rise in cardiac index with increasing ventricular rate. The maximal cardiac output response occurred at rates above 100/min in all patients who were paced at rates above this level. The cardiac index fell as the rate was increased in two patients: from 107/min to 125/min in C.C. and from 115/min to 130/min in J.D. Although the maximal response occurred above 100/min, an adequate increase in cardiac index had occurred with rates between 80 to 90/min in most patients.

Figure 1

Effect of pacing at the rate associated with the maximal cardiac index on hemodynamic measurements in complete heart block complicating acute myocardial infarction (mean and standard deviation).

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In one patient (J.M.) who had severe cardiogenic shock and whose clinical condition appeared to be deteriorating steadily, the cardiac index increased slightly from 1.21 L/min/m² to 1.44 L/min/m², with pacing at 83/min, but fell to 0.99 L/min/m² when the rate was increased to 104/min (fig. 4).

The results of hemodynamic measurements repeated between 2 and 10 days after the onset of CHB are presented in figure 5.

**Mean Systemic Arterial Blood Pressure**

The mean systemic blood pressure was measured in heart block in all 13 patients. It increased with pacing by 23% from an average level in heart block of 80 ± 14.7 mm Hg to 98 ± 17.3 mm Hg at maximal output rates ($P < 0.001$) (table 2, fig. 1). The average mean systemic blood pressure in heart block was 82 mm Hg in both the group of patients with cardiac indices of less than 2.50 L/min/
Figure 3

Effect of increasing ventricular rate by pacing on: (A) cardiac index; (B) mean systemic arterial pressure; (C) stroke index; and (D) systemic vascular resistance in complete heart block complicating acute myocardial infarction. See key in D.

m² and the group with higher indices. With pacing at maximal output rates, the group with indices of less than 2.50 L/min/m² increased the mean blood pressure to an average level of 98 mm Hg, and the group with higher indices increased it to 97 mm Hg. However, if the initial pressure was low, there was a proportionally greater increase with pacing than if it was initially high. The average increase for patients with pressures of 80 mm Hg or less during heart block was 32% compared with 18% for those with higher initial pressures. Blood pressure fell with pacing at maximal measured output rates despite increases in cardiac index in two patients. Patient W.H. studied again on the third day of heart block had developed 2:1 block with a relatively high output, while patient M.F. was paced at 120/min to suppress a nodal tachycardia which was alternating with CHB (table 2).

In eight patients blood pressure was measured in heart block and during pacing at three or more rates (fig. 3B). Blood pressure rose progressively in all but two patients (C.O. and G.R.). In these two patients there was an initial fall with pacing at 65/min and 75/min, respectively, before it rose with higher rates. In G.R. this was associated with a fall in cardiac index, but in C.O., cardiac index had risen. In patient J.D. (fig. 6) blood pressure rose when the rate was increased from 115 to 130/min although cardiac index fell. With these two exceptions changes in
blood pressure tended to parallel the changes in cardiac index produced by pacing. There was no consistent relationship, however, between the absolute levels of cardiac index and the patient's blood pressure on the first or subsequent days of heart block or after return to sinus rhythm (fig. 5).

**Stroke Index**

The mean stroke index in the 11 patients in whom measurements were made in heart
Hemodynamic changes on the first and subsequent days of heart block and after return to sinus rhythm in six patients with acute myocardial infarction.

block was $41 \pm 15.8$ ml/m$^2$. With pacing at maximal output levels this decreased by 25% to $31 \pm 8.9$ ml/m$^2$ ($P<0.005$) (table 2, fig. 1). In six of the 16 instances in which measurements were made in both heart block and during pacing in these 11 patients, the stroke index increased or fell only slightly with pacing despite increases in heart rate of 15 to 46/min (table 2). Patient M.F. represents a special case. She had CHB with an idioventricular rate of 42/min alternating with brief periods of nodal tachycardia at 115/min and periods of ventricular asystole. Her average ventricular rate was 98/min. Pacing at 120/min suppressed the nodal tachycardia and resulted in an increase in stroke index of 6 ml/m$^2$.

In seven of the eight patients paced at three or more rates, stroke index fell progressively with increases of heart rate. In the case of patient C.C., however, stroke index increased with pacing at 87/min and 107/min and only fell when the rate was increased to 125/min when the cardiac index also fell (fig. 3C).

Figure 5 shows the changes in stroke index on subsequent days. Despite relatively large increases in heart rate, stroke index either had changed very little or had increased.
Hemodynamic effect of increasing ventricular rate in patient J.D. with complete heart block complicating acute myocardial infarction.

Systemic Vascular Resistance

The average systemic vascular resistance for the group as a whole did not change significantly with pacing at maximal output rates (table 2). However the patients with cardiac indices of less than 2.50 L/min/m² had an average resistance of 3,468 ± 873 dyne-sec cm⁻¹/m² which fell significantly with pacing at maximal output rates to 2,956 ± 641 dyne-sec cm⁻¹/m² (P < 0.05). In contrast, the pa-
PACING IN COMPLETE HEART BLOCK

Figure 7

Effect of increasing ventricular rate by pacing on the tension time index in complete heart block complicating acute myocardial infarction.

tients with cardiac indices of greater than 2.50 L/min/m² had a much lower resistance in heart block of 1,737 ± 300 dyne-sec cm⁻²/m² (P < 0.005), and pacing at maximal output rates increased this insignificantly.

In three of the six patients studied on subsequent days systemic vascular resistance fell as cardiac index increased (fig. 5).

Pulmonary Arterial Pressure

The mean pulmonary arterial pressure was elevated above 20 mm Hg in six of the 12 patients in whom it was measured in heart block (table 2). There was no significant difference in pressures between the patients with cardiac indices in heart block of less than 2.50 L/min/m² and those with higher indices. Pacing had no significant effect on the pulmonary arterial pressure, whether normal or elevated, and there was no significant change in pressure with pacing at different rates.

Right Atrial Pressure

The mean right atrial pressure was elevated above 6 mm Hg in eight of the nine patients in whom it was measured in heart block (table 2). The mean atrial pressure of five patients with elevated pulmonary arterial pressure averaged 12 ± 4.3 mm Hg and that of four with normal pulmonary arterial pressures averaged 8 ± 4.6 mm Hg. The elevation of right atrial pressure was always associated with a corresponding elevation in right ventricular end-diastolic pressure. Pacing had no significant effect on right atrial pressure (table 2).

Left Ventricular External Work

Left ventricular external work averaged 0.94 ± 0.43 kg-m/min/m² in the seven patients in whom it was measured in heart block and increased with pacing at maximal output rates to 2.91 ± 1.90 kg-m/min/m² (P < 0.02) (table 2).

Tension Time Index

The tension time index was measured in nine patients. It increased from a mean of
1,308 ± 528 mm Hg sec/min in heart block to 6,782 ± 3,613 mm Hg sec/min with pacing at maximal output rates (P < 0.001) (table 2). The progressive rise in tension time index with increasing rates is shown in figure 7.

Discussion

There is general agreement that ventricular asystole is an indication for pacing in CHB complicating acute myocardial infarction. The role of pacing in the management of bradycardia, however, is less easy to define partly because an alternative treatment in the form of isoproterenol is available, and partly because there have been no objective measurements of the hemodynamic effects of pacing. The present study was undertaken to determine the circulatory changes associated with CHB in acute infarction and to evaluate the effect of pacing on these changes.

The cardiac output in CHB complicating acute myocardial infarction in our cases was usually, but not invariably, severely reduced. In contrast to chronic CHB, most patients were unable to increase stroke volume because of marked depression of myocardial function and could not, therefore, compensate for the bradycardia and maintain an adequate flow. The patient's mental state and skin circulation provided the most useful clinical information about the adequacy of the cardiac output in heart block. Thus seven patients who had varying degrees of inattention, confusion, restlessness, and poor skin circulation had cardiac indices of less than 2.03 L/min/m². In contrast, four patients who had nearly normal mental function and good skin circulation had cardiac indices between 2.22 and 3.13 L/min/m². Heart rate was not a reliable guide to the adequacy of the cardiac output. Although patients with rates of less than 45/min usually had low cardiac indices, low values were also found in several patients with rates between 50 and 61/min. Similarly, blood pressure was unreliable in predicting cardiac output. There were, on the one hand, patients with low blood pressures but well-maintained cardiac indices, and on the other, patients with relatively well-maintained pressures but very low cardiac indices.

The present study demonstrates that cardiac output can be increased in almost all instances by increasing rate by artificial pacing. This response was accompanied by an increase in systemic blood pressure which was progressive with increasing pacing rates. Those patients in whom the initial cardiac index was low had considerable improvement in mental state and skin circulation. The fact that the clinical benefit achieved by pacing continued for many hours suggests that the hemodynamic improvements demonstrated in the acute studies were also maintained. When pacing rates were increased, cardiac index continued to rise until rates between 102 and 120/min were reached. In two patients who were studied at 125 and 130/min, cardiac index had begun to fall. In one patient with cardiogenic shock, cardiac index fell when the rate was increased to 104/min. The rate associated with the maximal cardiac output (optimum rate) was found, therefore, with one exception to be between 102 and 120/min, but as faster rates were not tested in most patients, it might have been higher. This range of optimum rates is considerably higher than the range of 60 to 80/min which has been reported in chronic CHB.

The effect on myocardial function of increasing cardiac output and systemic blood pressure by pacing has not been established. In the patients in whom the stroke index increased or stayed the same in spite of increased rate, it is reasonable to assume that myocardial function was good or had been improved by pacing and that the bradycardia per se had been the major factor in the previously low cardiac index and blood pressure. In most patients, however, stroke index fell in response to pacing in spite of still subnormal cardiac indices and in these patients myocardial performance was probably abnormal. In all instances in which measurements were repeated after the first day of CHB, stroke index had either increased or changed only slightly despite relatively large increases.
in heart rate, suggesting that myocardial function was improving with recovery from the infarct.

The loss of atrial transport function which accompanies CHB could be another factor responsible for the inadequate stroke output with increasing rates. In the present study a regular increase and decrease in systemic arterial pressure occurred as atrial systole moved in and out of phase with ventricular diastole. This is almost certainly attributable to an increase and decrease in stroke volume as the result of varying ventricular filling and is also often seen in chronic CHB.15,18 In the patients with acute myocardial infarction, these pressure changes were of relatively small magnitude. Moreover, Gillespie and associates18 have shown that the augmentation of stroke volume due to optimally timed atrial systole is of much less importance in the diseased than in the normal heart. Although the temporary value of atrial synchronized pacing has been demonstrated in chronic CHB,16 this remains to be demonstrated for CHB complicating acute myocardial infarction.

Five patients in the present study had mild elevation of the pulmonary arterial pressure (20 to 31 mm Hg) with marked elevation of the right atrial pressure (12 to 19 mm Hg) and corresponding elevations of the right ventricular end-diastolic pressures. These levels could not be due to volume overload or to the mild pressure overload of the right ventricle and were, therefore, probably due to failure of the right ventricular myocardium secondary to ischemia. These patients all had inferior myocardial infarctions which are usually due to right coronary artery occlusion.19 Since the right coronary artery supplies most of the right ventricle as well as part of the left in most hearts,20 the predominant involvement of right ventricular myocardium by the occlusion could account for the predominant right ventricular failure.

Although pacing may result in substantial improvement in cardiac output and blood pressure, this is accompanied by a considerable rise in the tension time index implying a corresponding increase in myocardial oxygen requirements.21,22 In the normal heart a rise in oxygen demand is met by an increase in coronary flow.23 However, when coronary flow is restricted, this may limit myocardial oxygen uptake24–26 and “set a limit to the ability of the heart to speed up its rate without suffering a further decline in work capacity.”23 This contention is supported by the observation in chronic CHB that tachycardia induced by pacing, whether or not it results in an increase in cardiac output, may produce a decrease in actual myocardial oxygen consumption and an increase in excess myocardial lactate production, indicating an increase in anaerobic myocardial metabolism.27 In addition it is evidenced clinically by the occasional patient who develops angina with pacing and may explain in part the inadequate stroke output response seen in most cases. On the other hand, it is possible that in some patients pacing may have a favorable effect on myocardial oxygen requirements by decreasing ventricular end-diastolic volume and, therefore, reducing myocardial tension.21,28

In clinical practice, therefore, the value of improving the systemic circulation by pacing must be balanced against this increased work and oxygen requirement in deciding which patients should be paced and the best rate for pacing. Patients with normal mental function and good skin circulation usually have adequately maintained cardiac outputs and do not require pacing.3 When there is depressed mental function and poor skin circulation, however, severe reduction of the cardiac output is invariable and pacing is indicated. In determining the rate at which to pace, it is reasonable to aim for the lower limits of normal for cardiac output. It may be assumed that the cardiac output has reached adequate levels when the blood pressure has done so if this is accompanied by improved mental function and by evidence of increased skin circulation. The provocation of angina or dyspnea is an indication that the optimum rate has been exceeded. In practice this rate can be determined by clinical trial—in most cases it will prove to be between 80 and 90/min.
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50 Years Ago
Auto-Regulation of Blood Flow

"My own first contribution to the problem . . . was published in Danish in 1918 . . . and appeared in the British Journal of Physiology (1919). . . . I found it possible to observe at least the superficial capillaries of muscles both in the frog and in mammals through a binocular microscope. . . . Resting muscles observed in this way are usually quite pale, and the microscope reveals only a few capillaries at fairly regular intervals. These capillaries are so narrow that red corpuscles can pass through only at a slow rate and with a change of form from the ordinary flat discs to elongated sausages. When the muscle . . . is stimulated to contractions a large number of capillaries become visible and dilated. . . . Since capillaries, even in a group fed by the same arteriole, do not all behave in the same way, the changes obviously cannot be due to arterial pressure changes. . . ."—AUGUST KROGH: Description of the Prize-Winning Work. In SOURKES, THEODORE L.: Nobel Prize Winners in Medicine and Physiology 1901-1965. London, Abelard-Schuman, 1966, pp. 98-99.
Hemodynamic Effects of Artificial Pacing in Complete Heart Block Complicating Acute Myocardial Infarction
B. W. LASSERS, J. L. ANDERTON, M. GEORGE, A. L. MUIR and D. G. JULIAN

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