COMPLETE HEART BLOCK

COMPPLICATING ACUTE MYOCARDIAL INFARCTION

AND

THE ROLE OF ARTIFICIAL PACING IN ITS MANAGEMENT

by

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The haemodynamic investigations which are reported were carried out in collaboration with Drs. J. L. Anderton, M. George, and A. Muir, and I wish to thank them for their assistance. The British Heart Foundation and the Scottish Hospitals Endowment Research Trust provided funds for technical assistance and for equipment used in this study. I also wish to thank Dr. M. J. Godman for permission to use certain data from his study of bundle branch block in which I took part.

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APPENDIX  102
To determine the effect of artificial pacing on the natural history of complete heart block (CHB) complicating acute myocardial infarction, 100 patients with this disorder of conduction were studied under conditions of continuous electrocardiographic monitoring and intensive care with pacing immediately available. The clinical and electrocardiographic features in these patients and the necropsy findings in those patients who died were examined and related to the need for and effects of pacing. To determine the circulatory alterations associated with heart block in acute infarction and their response to pacing, haemodynamic measurements were carried out in 13 consecutive patients soon after the onset of CHB and were repeated on the second and subsequent days of heart block and after the return of normal atrioventricular conduction in five. In order to assess the long-term outlook of patients who had required pacing during the course of an acute infarct complicated by CHB, the first 27 of the 100 patients who survived to be discharged from hospital were reviewed at the end of one year.

CHB occurred early in the course of acute myocardial infarction and had developed prior to admission to hospital in many patients. However, when CHB was observed to develop, it was usually preceded by a premonitory disorder of conduction: second degree atrio-ventricular block in the case of inferior or inferolateral infarction, and complete bundle branch block in the case of anterior infarction. The most unfavourable immediate prognosis occurred in patients with anterior/
anterior infarction or a QRS complex of greater than 0.12 second duration during CHB. These patients usually had severe myocardial damage and pacing made little impact on their outlook. Inferior infarction with a QRS complex of less than 0.12 second duration was associated with a mortality little higher than that of patients with acute infarction uncomplicated by heart block. In the majority of these patients pacing was not required, but in a proportion of those with attacks of asystole or evidence of a low cardiac output, it produced striking improvements in clinical and haemodynamic features.

The prognosis during the remainder of the first year after discharge from hospital was no worse for patients who had been benefited by pacing during the acute attack than the outlook for patients with complete block in whom pacing had not been required. Moreover, the long-term prognosis for patients who survived an acute myocardial infarction complicated by CHB was no worse than that of patients with infarcts of similar severity but without heart block.

Because patients in whom bradycardia or asystole had been corrected by pacing and who survived the acute attack had a relatively good long-term prognosis, it was concluded that pacing does have a role to play in the management of CHB complicating acute myocardial infarction. Since 50% of patients with second degree heart block progressed to CHB and because it was not possible to predict which patients with block would eventually require pacing or when they would require it, it was also concluded that electrodes should be introduced in all patients with either second degree of complete heart block. On the other hand, although 30% of patients with complete bundle/
bundle branch block progressed to CHB and most subsequently required pacing, electrodes should not be inserted prophylactically in this group of patients because pacing did not reduce mortality and because electrode insertion was associated with a high incidence of serious arrhythmias.

Because of the dangers associated with electrode insertion and pacing, special facilities including continuous electrocardiographic monitoring and intensive care are required when this form of treatment is used. When such facilities are available, it was concluded that pacing is superior to drug therapy in treating patients with ventricular asystole and patients in whom bradycardia is responsible for clinical deterioration.
HISTORICAL REVIEW

AND

INTRODUCTION
Complete heart block (CHB) has been recognised as a complication of acute myocardial infarction since the case report by Levine and Tanter in 1918. In fact, however, it is probable that CHB was present in the very first instance of acute myocardial infarction to be recognised clinically and confirmed at necropsy (Hammer 1876). In Hammer's case the pulse rate was observed to drop from 80/min to 8/min; the patient survived a further 30 hours and at necropsy occlusion of the right coronary artery was demonstrated.

Following the report by Levine and Tanter (1916) there were numerous reports of single cases or small series of CHB occurring in the context of ischaemic heart disease. In most of these reports, however, the criteria used in the diagnosis of myocardial infarction and complete heart block were not stated and it is clear from the clinical descriptions that many of these were in fact cases of chronic CHB with Stokes-Adams attacks, acute myocardial infarction superimposed on chronic heart block, or CHB occurring in patients with long standing ischaemic heart disease or old myocardial infarction and attributed on this basis to coronary artery occlusion. Thus, for example, electrocardiographic evidence of acute infarction was present in only two of the 15 cases reported by Schwartz (1936) under the title "Auriculoventricular/"

---

1 See Kerr (1937) and Master et al (1938) for extensive bibliographies of the early literature.
"Auriculoventricular dissociation and the Adams-Stokes syndrome in acute coronary vessel closure". However, there were a considerable number of single case reports which clearly do describe CHB occurring during an acute myocardial infarction and also a few small series in which the diagnostic criteria appear to be adequate.¹

In these studies the detection of CHB during an episode of acute infarction depended either on the conduction disturbance being discovered on routine electrocardiograms, or on the observation of clinical deterioration and/or a slow pulse rate. However, in the context of acute myocardial infarction, CHB may, on the one hand, be very transient and not accompanied by clinical deterioration, or, on the other hand, be abrupt in onset and rapidly fatal. Accordingly, without continuous electrocardiographic monitoring, many cases will remain undiagnosed and studies such as those mentioned above were not representative of the complete spectrum of the condition. However, the introduction of artificial pacing for the management of CHB coincided with the advent of continuous electrocardiographic monitoring of patients with acute myocardial infarction. Thus, although monitoring permitted the detection of most cases of heart block, the widespread use of pacing prevented the acquisition of information about the natural, untreated history of heart block after acute myocardial infarction.

Nevertheless, monitored series have increased understanding of the course of heart block following infarction. They have made it evident/

¹ See Bibliography A, page 97
evident that GHB is a more common occurrence in acute myocardial infarction than was previously thought and have shown that despite treatment it continues to be associated with a high mortality. In some patients death is undoubtedly due to coexistent severe myocardial damage rather than to the heart block itself, but in others it has been attributed to ventricular asystole or the haemodynamic consequences of severe bradycardia. Specific therapy has, for this reason, been directed at preventing or abolishing the asystole and correcting the bradycardia. And over the past 15 years temporary transvenous pacemaking has become increasingly used for this purpose.

But despite the extensive use of pacing, a number of questions about its application to the management of heart block in acute myocardial infarction still remain unanswered. Neither the exact indication for the insertion or removal of electrodes nor the precise circumstances in which pacing should be initiated have been defined. Furthermore, although clinical observations have suggested that pacing may result in improvement in the circulatory state, there have been no measurements of the haemodynamic changes associated with heart block and the effects of artificial pacing on these changes in the particular situation of acute myocardial infarction. In addition, the true effect of pacing on immediate mortality is not known and its advantages over drug therapy continue to be debated. And finally, there have been no long-term follow-up studies of patients who have survived/

1 See Bibliography B, page 99
survived acute myocardial infarction complicated by CHB and it has been suggested that resuscitation of these patients may be of little value as it is possible that they have such severe underlying myocardial damage that their long-term outlook is very poor (Shillingford, 1968; Sloman, 1968). Because of uncertainty about these points, there continues to be considerable controversy about the management of heart block in acute myocardial infarction (Friedberg et al., 1968; McNally and Benchimol, 1968; Lown et al., 1969).

In view of this, the present investigations were undertaken to attempt to answer these questions and in doing so to help to clarify the role of artificial pacing in the management of CHB complicating acute infarction. In the absence of a controlled study it seemed that the best way of achieving this would be to study a large number of patients with heart block and its precursors under conditions of continuous monitoring and intensive care and to ensure that pacing was always immediately available. In such an environment the indications for electrode insertion and removal and for initiation of pacing could be determined and the haemodynamic effects of pacing measured. In addition it should be possible to establish to what extent death in these patients was due to factors other than heart block itself and, thereby, gain some idea of the benefits of pacing although its absolute effect on mortality might remain uncertain. Those patients who survived could subsequently be followed up and their long-term progress related to their clinical behaviour during the acute attack.

To accomplish these aims, the clinical and electrocardiographic features and the course of CHB in 100 patients with acute myocardial infarction/
infarction were studied in detail and the pathological findings in those patients who died were examined (Section One). In 13 consecutive patients measurements were made of the haemodynamic alterations associated with the early hours of advanced heart block and of their response to ventricular pacing at various rates; to assess the changes in circulatory status on the second and subsequent days of heart block and after return to normal atrioventricular conduction, measurements were also repeated in five patients (Section Two). Pacing electrodes were introduced in 95 of the 100 patients and the indications for and techniques, benefits and complications of pacing were examined and related to the clinical, haemodynamic and pathological findings (Section Three). Finally, to determine the long-term prognosis, the first 27 patients who survived to be discharged from hospital were reviewed at the end of one year and the mortality and degree of functional recovery assessed (Section Four).
SECTION ONE

CLINICAL STUDY
METHODS

Material

The clinical study is based on the first 100 patients with CHB complicating acute myocardial infarction managed in the coronary care unit of the Royal Infirmary, Edinburgh, between May, 1966 and May, 1969. The author was personally involved in the management and the case documentation of the first 80 of these patients. Information on the remaining 20 patients was obtained from their coronary care unit case records.¹

Selection of Patients: Coronary Care Unit Admission Policy

Routine admission to the coronary care unit was restricted to patients under 70 years of age in whom the onset of symptoms suggestive of acute infarction had occurred within 48 hours before admission. In addition, patients of any age who had developed dangerous arrhythmias or second or third degree atrioventricular block were also admitted (Lawrie et al, 1967). Furthermore, during a period of one year, pacing electrodes were inserted as a prophylactic measure in patients with complete bundle branch block complicating acute myocardial infarction and such patients were also admitted irrespective of the time of onset of symptoms or of age. Therefore, over the three-year/

¹ Records were not complete in two further cases of CHB, which have not been included in the study.
year period patients with CHB were derived from three sources:

(1) Routine admissions who developed CHB while in the coronary care unit,

(2) Patients admitted to the unit because second or third degree AV block had been detected in a general medical ward or prior to admission to hospital, and

(3) During a period of one year only, patients admitted to the unit because complete bundle branch block had been discovered in a general medical ward or prior to admission to hospital and who progressed to CHB.

**Electrocardiographic Monitoring**

Each patient's electrocardiogram (ECG) was continuously monitored by a Sanborn Visomonitor Unit with a rate-activated alarm. A ten-second sample ECG was automatically recorded every 15, 30 or 60 minutes as required and also whenever an alarm occurred. In addition, the ECG was continuously displayed on an oscilloscope at the central nursing station. The unit was staffed by nurses specially trained in the detection of arrhythmias and disorders of conduction and a doctor was in the unit 24 hours a day (Lawrie et al, 1967).

**DIAGNOSTIC CRITERIA AND DEFINITIONS**
1. **Acute myocardial infarction**

A clinical diagnosis of acute myocardial infarction was considered established if:

(a) pathological Q waves appeared on the ECG accompanied by an elevation of the ST segment and subsequent inversion of the T wave (World Health Organisation, 1959);

(b) there were changes in the ST segment and T wave suggestive of infarction accompanied by a significant rise in the serum aspartate aminotransferase (>50 Reitman and Frankel units) and serum creatine kinase (>80 i.u.);

or

(c) there was left bundle branch block accompanied by similar enzyme changes.

The diagnosis was also confirmed at necropsy in 41 of these patients and in two others in whom full electrocardiograms had not been recorded.

2. **Site of infarction**

The ECG site of acute infarction was classified as follows:

(a) Inferior, if there were Q waves and ST segment changes occurring in leads III and aVF;

(b) Anterior, if these changes occurred in the chest leads.
leads with or without changes in leads I and aVL;

(c) Inferolateral, if the changes in leads III and aVF were accompanied by changes in leads I and aVL and/or V5-V6 without changes in the other chest leads.

Four patients with evidence of combined anterior and inferior infarction were classified as anterior for purposes of analysis. Two patients in whom the pattern of complete left bundle branch block obscured the site of infarction on the ECG were classified as anterior on the basis of necropsy findings.

3. **Complete Heart block (CHB)**

CHB, or third degree AV block, was diagnosed when there was complete interruption of AV conduction with independent atrial activity, or if atrial fibrillation or flutter was present with a regular ventricular rate of less than 60/min in the absence of digitalis therapy. Patients with AV dissociation of the interference type in which there is usually a slow atrial rate (McNally and Benchimol, 1968), patients with transient CHB after direct current shock for ventricular tachyarrhythmias, and patients with atrial as well as ventricular asystole have not been included.

4. **First and second degree AV block**

First degree heart block was defined as a PR interval exceeding 0.20 sec. Second degree block was diagnosed when there was intermittent failure of AV conduction. This included:

(a) AV block with 2:1, 3:1, 4:1 or varying ventricular response;
response;
(b) the Wenckebach phenomenon;
(c) Mobitz Type II block (second degree block with a fixed PR interval); and
(d) atrial fibrillation or flutter with an irregular ventricular response at a rate of less than 60/min in the absence of digitalis therapy.

The term "advanced heart block" includes both second and third degree block and the term "partial heart block" includes first and second degree block.

5. Complete bundle branch block (BBB)

Complete BBB was diagnosed when it was reasonably certain that the ventricles were responding to a pacemaker situated above the AV node and the longest QRS interval in any one of the three standard ECG leads measured 0.12 sec or greater.

Complete right bundle branch block (RBBB) was said to be present when the above criteria were fulfilled and in addition there were RSR' or rSR' complexes in V1-V2 with a wide S wave in V5-V6. Complete left bundle branch block (LBBB) was diagnosed when the criteria were met and there were broad slurred R waves or rSR' or RaR' complexes in V4-V6.

The following ECG criteria were used for the diagnosis of impaired conduction in both right and left bundle branches - bilateral bundle branch block (Wilson et al, 1934; Rosenbaum and Lepeschkin, 1954; Lepeschkin, 1964):

(a)/
(a) RBBB with marked left axis deviation;
(b) alternating RBBB and LBBB;
(c) LBBB in the limb leads with RBBB in the precordial leads;
(d) BBB with, in addition, prolongation of the PR interval to 0.21 sec or greater.

6. **Asystole**

The term "asystole" is restricted to the situation in which there was neither atrial nor ventricular activity evident on the ECG - i.e. total cardiac standstill. "Ventricular asystole" or "ventricular standstill" is used to describe the ECG finding of absence of ventricular complexes but continuing atrial activity.

7. **Cardiogenic shock**

The clinical diagnosis of cardiogenic shock was made when all of the following features had been present for longer than 30 minutes:

(a) a systolic blood pressure measured by sphygmononometer of less than 90 mm Hg;
(b) marked restlessness or a diminished level of consciousness; and
(c) intense cutaneous vasoconstriction.

8. **Left ventricular failure**

Left ventricular failure was diagnosed if there was radiographic evidence of pulmonary venous dilation or oedema or if widespread pulmonary crepitations were accompanied by breathlessness at rest.
Left ventricular failure was not diagnosed on the bases of crepitations or added heart sounds alone (Lassers et al, 1969).

RESULTS

Age, Sex and Mortality

There were 71 male and 29 female patients. The average age was 61.6 ± 10.5 years with a range of 31 to 87 years (Figure 1:1). The female patients were significantly older than the male patients (Table 1:1).

Fifty-two patients died and 48 survived. There was no significant difference between the average age of the patients who died and those who survived (Table 1:1). The male patients who died were significantly older than the male survivors, but this was not true in the case of the female patients. The female survivors were significantly older than the male survivors, but the difference between the ages of the male and female patients who died was not significant (Table 1:2).

Site of Infarction

CHB was associated with inferior infarction in 53 patients, with anterior infarction in 38 and with inferolateral infarction in nine. Although CHB was more common in inferior infarction, when it did complicate anterior infarction, the mortality was significantly higher despite the fact that there was no significant difference in the average ages of patients with infarcts in different sites (Tables 1:1 and 1:3).
Figure 1:1 Age distribution of patients with CHB complicating acute myocardial infarction.
<table>
<thead>
<tr>
<th>Characteristic</th>
<th>Number of Patients</th>
<th>Age ± SD</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>Male</td>
<td>71</td>
<td>59.8 ± 11.4</td>
<td>&lt;0.02</td>
</tr>
<tr>
<td>Female</td>
<td>29</td>
<td>65.8 ± 6.4</td>
<td></td>
</tr>
<tr>
<td>Alive</td>
<td>48</td>
<td>59.8 ± 11.2</td>
<td>n.s.</td>
</tr>
<tr>
<td>Dead</td>
<td>52</td>
<td>63.2 ± 9.7</td>
<td></td>
</tr>
<tr>
<td>Inferior Infarct</td>
<td>53</td>
<td>61.6 ± 10.9</td>
<td></td>
</tr>
<tr>
<td>Anterior Infarct</td>
<td>38</td>
<td>61.3 ± 9.1</td>
<td>n.s.</td>
</tr>
<tr>
<td>Inferolateral Infarct</td>
<td>9</td>
<td>62.6 ± 14.6</td>
<td></td>
</tr>
</tbody>
</table>

* Mean ± SD

Table 1: Relationship between age and various characteristics of patients with CHB complicating acute myocardial infarction. The last column gives the P value for the significance of the difference between the ages in the previous column.
<table>
<thead>
<tr>
<th></th>
<th>Age of Patients Dying</th>
<th>Age of Patients Surviving</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>Males (71 patients)</td>
<td>62.0 ± 11.0</td>
<td>57.7 ± 11.6</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>Females (29 patients)</td>
<td>66.1 ± 5.1</td>
<td>65.4 ± 7.8</td>
<td>n.s.</td>
</tr>
<tr>
<td>P</td>
<td>&gt;0.10</td>
<td>&lt;0.05</td>
<td></td>
</tr>
</tbody>
</table>

* Mean ± SD

Table 1:2 Relationship between sex, age and mortality in patients with CHB complicating acute myocardial infarction. The significance levels given at the end of the rows refer to a comparison (Student's t-test) between the age of the groups in the two columns of that row; similarly the significance levels at the bottom of the columns refer to a comparison between the age of the groups in the two rows of that column.

Comparison of males dying vs males surviving: t = 2.679, 69 d.f.; p <0.01
<table>
<thead>
<tr>
<th>Site</th>
<th>Number of Patients</th>
<th>Number of Deaths</th>
<th>Percent</th>
</tr>
</thead>
<tbody>
<tr>
<td>Anterior</td>
<td>38</td>
<td>33</td>
<td>87%</td>
</tr>
<tr>
<td>Inferior</td>
<td>53</td>
<td>15</td>
<td>28%</td>
</tr>
<tr>
<td>Inferolateral</td>
<td>9</td>
<td>4</td>
<td>44%</td>
</tr>
<tr>
<td>Total</td>
<td>100</td>
<td>52</td>
<td>52%</td>
</tr>
</tbody>
</table>

Table 1:3 Relationship between site of infarction and mortality in patients with CHB complicating acute myocardial infarction.

Comparison of mortality in anterior v. inferior infarction: $X^2 = 30.43$; p < 0.0005; in anterior v. inferolateral: $X^2 = 7.81$; p < 0.01; in inferior v. inferolateral: n.s.
Time of Onset of CHB

Both the time of onset of symptoms of infarction and the time of onset of CHB were known with reasonable accuracy in 98 patients. 52% of cases of CHB had developed within 24 hours of the onset of symptoms of infarction, 85% within 48 hours and no case later than seven days (Figure 1:2). Although the majority of cases of CHB in inferior and inferolateral infarction developed on day one, a slightly higher percentage of cases of CHB complicating anterior infarction occurred on day two than on day one (Figure 1:3).

Preceding Disorders of Conduction

Forty-four patients had CHB on admission to the coronary care unit. In 52 of the remaining 56 patients some disorder of conduction was detected before CHB developed. With inferior or inferolateral infarction this was almost always second degree AV block, but with anterior infarction it was invariably complete BBB occasionally accompanied by second degree AV block (Table 1:4). The average length of time between the detection of second degree heart block and the onset of CHB was $9.7 \pm 11.3$ hours and ranged from minutes to as much as 48 hours. ECG patterns associated with bilateral BBB were detected in 19 of the 30 patients with BBB preceding CHB (Table 1:5). Of the four patients in whom neither second degree AV nor complete BBB had been detected prior to CHB, two had had episodes of ventricular tachycardia and ventricular fibrillation for which they had been receiving large doses of lignocaine and procainamide which may have contributed to the development/
Figure 1:2  Time of onset of CHB after onset of symptoms of acute myocardial infarction. (Time of onset of infarction not known in one patient.)
Figure 1:3  Relationship between time of onset of CHB after onset of symptoms of acute myocardial infarction and site of infarction. Each bar represents the percentage of patients with infarcts in the particular site indicated who developed CHB on that day.
### Table 1:4 Relationship between site of infarction and type of disturbance of conduction detected preceding the development of CHB.

#### Inferior Infarction

<table>
<thead>
<tr>
<th>Preceding AV Conduction</th>
<th>Preceding Bundle Branch Conduction</th>
<th>Number of Patients</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Second Degree Block</strong></td>
<td>BBB Present</td>
<td>3</td>
<td>21</td>
</tr>
<tr>
<td></td>
<td>BBB Absent</td>
<td>18</td>
<td></td>
</tr>
<tr>
<td><strong>Normal</strong></td>
<td>BBB Present</td>
<td>2</td>
<td>5</td>
</tr>
<tr>
<td></td>
<td>BBB Absent</td>
<td>3</td>
<td></td>
</tr>
<tr>
<td><strong>Total</strong></td>
<td></td>
<td></td>
<td>26</td>
</tr>
</tbody>
</table>

#### Inferolateral Infarction

<table>
<thead>
<tr>
<th>Preceding AV Conduction</th>
<th>Preceding Bundle Branch Conduction</th>
<th>Number of Patients</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Second Degree Block</strong></td>
<td>BBB Present</td>
<td>0</td>
<td>3</td>
</tr>
<tr>
<td></td>
<td>BBB Absent</td>
<td>3</td>
<td></td>
</tr>
<tr>
<td><strong>Normal</strong></td>
<td>BBB Present</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td></td>
<td>BBB Absent</td>
<td>0</td>
<td></td>
</tr>
<tr>
<td><strong>Total</strong></td>
<td></td>
<td></td>
<td>3</td>
</tr>
</tbody>
</table>

#### Anterior Infarction

<table>
<thead>
<tr>
<th>Preceding AV Conduction</th>
<th>Preceding Bundle Branch Conduction</th>
<th>Number of Patients</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Second Degree Block</strong></td>
<td>BBB Present</td>
<td>3</td>
<td>3</td>
</tr>
<tr>
<td></td>
<td>BBB Absent</td>
<td>0</td>
<td></td>
</tr>
<tr>
<td><strong>Normal</strong></td>
<td>BBB Present</td>
<td>23</td>
<td>24</td>
</tr>
<tr>
<td></td>
<td>BBB Absent</td>
<td>1</td>
<td></td>
</tr>
<tr>
<td><strong>Total</strong></td>
<td></td>
<td></td>
<td>27</td>
</tr>
<tr>
<td>Type of Bundle Branch Block</td>
<td>Anterior Infarcts</td>
<td>Inferior Infarcts</td>
<td>Total</td>
</tr>
<tr>
<td>-----------------------------</td>
<td>-------------------</td>
<td>-------------------</td>
<td>-------</td>
</tr>
<tr>
<td><strong>A. Bilateral</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>REBB + LAD</td>
<td>8</td>
<td>1</td>
<td>9</td>
</tr>
<tr>
<td>Alternating REBB and LBBB</td>
<td>2</td>
<td>2</td>
<td>4</td>
</tr>
<tr>
<td>LBBB in Limbs, REBB in Chest Leads</td>
<td>2</td>
<td>0</td>
<td>2</td>
</tr>
<tr>
<td>BBB + PR &gt;0.20 sec</td>
<td>3</td>
<td>1</td>
<td>4</td>
</tr>
<tr>
<td><strong>Total</strong></td>
<td>15</td>
<td>4</td>
<td>19</td>
</tr>
<tr>
<td><strong>B. Unilateral</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>11</td>
<td>0</td>
<td>11</td>
</tr>
<tr>
<td><strong>Total</strong></td>
<td>26</td>
<td>4</td>
<td>30</td>
</tr>
</tbody>
</table>

**Table 1:5** Type of bundle branch block detected before the onset of CHB.
development of block.

During the three year period of the study there were 26 additional patients with second degree AV block detected who did not progress to CHB. Thus the incidence of CHB in patients with second degree block was 51%.

The total number of patients with complete BBB during this period was not known. However, pacing electrodes were introduced as a prophylactic measure in 31 consecutive patients with complete BBB complicating acute myocardial infarction, and nine of these subsequently developed CHB. This gives an estimate of the incidence of CHB in patients with preceding complete BBB of 29%.

QRS Duration in CHB

When the QRS complex was greater than 0.12 sec in duration prior to CHB it invariably remained so when CHB developed. In addition it was uncommon for a QRS of normal duration before CHB to become prolonged with the onset of CHB. Thus, only two patients with a QRS complex of less than 0.12 sec before CHB progressed to CHB with a QRS complex of 0.12 sec or greater (Table 1:6).

A prolonged QRS complex in CHB was associated with a mortality of 82% compared with 22% in patients with a normal QRS duration. However, since all patients with anterior infarction and no patient with inferolateral infarction had prolonged QRS complexes, this feature was of prognostic value only in patients with inferior infarction. In these patients the mortality was 67% when the QRS complex was prolonged and 17% when it was less than 0.12 sec (Table 1:7). The mortality/
Table 1: Relationship between mortality and duration of QRS complex before and during CHB. (Duration QRS not known in one anterior infarct.)
<table>
<thead>
<tr>
<th>QRS Duration in CHB</th>
<th>Number of Patients</th>
<th>Number Dying</th>
<th>Percent Dying</th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt;0.12 sec</td>
<td>41</td>
<td>7</td>
<td>17%</td>
</tr>
<tr>
<td>≥0.12 sec</td>
<td>12</td>
<td>8</td>
<td>67%</td>
</tr>
<tr>
<td>Total</td>
<td>53</td>
<td>15</td>
<td>28%</td>
</tr>
</tbody>
</table>

Table 1:7 Relationship between mortality and duration of QRS complex during CHB in patients with inferior infarction.

Comparison of mortality in patients with QRS <0.12 sec v. patients with QRS ≥ 0.12 sec: $X^2 = 4.63$; $p < 0.05$. 
mortality associated with ECG evidence of bilateral BBB was not significantly different from that associated with unilateral BBB (Table 1:5).

Duration of Advanced Heart Block

Normal AV conduction returned in all 48 survivors. Figure 1:4 shows the time course of disappearance of advanced heart block after the onset of CHB in the survivors. Advanced heart block persisted for more than one week in only 6% of cases and normal conduction had returned in all patients by the end of the second week. The median duration of advanced block was 48 hours. The median time between onset of symptoms of infarction and disappearance of advanced block was 5.2 days and in no case did block persist for more than 17 days after the onset of symptoms.

Time of Death

When death occurred in CHB complicating acute infarction, it usually did so within the first few days after the onset of symptoms of infarction. Thus 31% of deaths occurred within 24 hours of the onset of symptoms and 73% by the end of the third day (Figure 1:5). There was no significant difference with respect to time of death between patients with anterior or inferior infarcts.

Mode of Death

The commonest mode of death was cardiogenic shock and 35 patients died in this manner (Table 1:8). At necropsy three of these patients were/
Figure 1: Time course of disappearance of advanced heart block after onset of CHB.
Figure 1:5  Time of death after onset of symptoms of acute myocardial infarction in patients with CHB complicating the infarct. (Time of onset of infarction not known in one patient.)
<table>
<thead>
<tr>
<th>Mode of Death</th>
<th>Number of Patients</th>
<th>Percent of Deaths</th>
</tr>
</thead>
<tbody>
<tr>
<td>Shock</td>
<td>35</td>
<td>65%</td>
</tr>
<tr>
<td>Asystole</td>
<td>6</td>
<td>12%</td>
</tr>
<tr>
<td>Asystole after DC shock for VF</td>
<td>5</td>
<td>10%</td>
</tr>
<tr>
<td>VF</td>
<td>1</td>
<td>2%</td>
</tr>
<tr>
<td>Sudden, late death in general ward</td>
<td>5</td>
<td>10%</td>
</tr>
</tbody>
</table>

 Patients with left ventricular failure, ventricular fibrillation (VF) or asystole who were also shocked, classified as shocked.

Table 1:8  Mode of death in patients dying from acute myocardial infarction complicated by CHB.
were found to have ventricular rupture. Six patients died in ventricular or in total cardiac asystole for which pacing was unsuccessful. At necropsy a pulmonary embolus was present in one of these patients. A further five patients died in asystole following direct current shock for ventricular fibrillation and one patient died of recurrent ventricular fibrillation unresponsive to treatment. Five patients died suddenly after transfer from the coronary care unit to general medical wards. In only one of these patients was the rhythm at the time of death - ventricular fibrillation - known.

**NECROPSIES**

Necropsies were performed in 43 of the 52 patients who died. Detailed results of these examinations are presented in Table A:1 in the Appendix.

**Coronary Artery Occlusion**

Acute coronary artery occlusions were demonstrated in 34 patients. In 13 patients more than one artery was occluded. Each of the nine patients in whom acute occlusions were not found, had severe stenosis of all three major coronary arteries.

Eleven of the 14 patients with ECG evidence of acute inferior infarction had occlusion of the right coronary artery and in five cases this was combined with occlusion of either the left anterior descending, the left circumflex or both left anterior descending and left circumflex arteries. Two further patients had severe stenosis of all three major/
major arteries and the remaining patient had left circumflex occlusion with severe stenosis of the right coronary artery. The only patient with ECG evidence of inferolateral infarction who came to necropsy had severe stenosis of all three major arteries.

Twenty-one of the 28 patients with ECG evidence of acute anterior infarction had occlusion of either the anterior descending or left circumflex artery or both. Six other patients had severe stenosis, without occlusion, of all three arteries. The remaining patient had occlusion of the right coronary artery with severe stenosis of the left circumflex and anterior descending arteries.

**Site of Infarction**

There was agreement between the ECG and necropsy location of the site of infarction in all but one patient. This patient (Table A:1, patient No. 27) whose ECG on admission showed Q waves with marked ST segment elevation from V1 to V4 without evidence of inferior involvement, had necropsy evidence of acute infarction of the inferior surface with extension to the anterolateral surface only. The right coronary artery was occluded and there was severe stenosis of the left anterior descending and left circumflex arteries.

Infarction was transmural in all cases that came to necropsy. In most instances there was extensive myocardial damage. Thus 27 patients had necropsy evidence of combined involvement of anterior and inferior walls of the heart by either old and/or recent infarction although this was detected on the ECG in only four.
QRS Duration and Necropsy Findings

All 28 patients with ECG evidence of anterior infarction and six patients with evidence of inferior infarction had prolonged QRS complexes in CHB. Twenty-six of the 28 patients with ECG evidence of anterior infarction had extensive anterior wall involvement with damage to the septum. In addition to the patient with ECG evidence of anterior infarction but necropsy evidence of inferior involvement only (mentioned in the previous paragraph), a further patient with anterior involvement on the ECG had damage to the anterolateral wall of the heart without apparent infarction of the septum demonstrated at necropsy. On the other hand, only two of the six patients with ECG evidence of inferior infarction plus a prolonged QRS complex, had detectable infarction of the anterior septum. The remaining four patients did, however, have occlusion or severe stenosis of the left anterior descending artery combined with right coronary artery occlusion and there was, therefore, a pathological basis for poor perfusion of the anterior septum in these patients.

Additional Necropsy Findings

Three patients were found at necropsy to have rupture of the free wall of the left ventricle. Despite the high incidence of septal infarction, there were no cases of rupture of the interventricular septum. One patient had a pulmonary embolus which probably contributed to death.

DISCUSSION/
DISCUSSION

Time of Onset, Duration and Incidence of CHB

CHB was a complication of the early hours of an acute myocardial infarction. Thus 52% of patients in the present series had developed CHB within 24 hours of the onset of symptoms of infarction, 85% within two days and 94% by the end of the third day (Figure 1: 2). This experience is similar to that of other authors (Master et al, 1938; Courter et al, 1963; Julian et al, 1964; Scott et al, 1967; Sutton et al, 1968; Beregovich et al, 1969; Norris, 1969).

In addition, heart block was a transient phenomenon following acute infarction and could be of very brief duration. It could, at one extreme, lead to sudden death within minutes of its onset, or, at the other, be accompanied by little or no change in the patient's clinical condition. In those patients who survived, normal AV conduction almost invariably returned within seven to 14 days (Figure 1: 4). In most reported cases of CHB persisting after acute infarction (Master et al, 1938; Cohen et al, 1958; Ourd et al, 1963; Bruce et al, 1965; Harris and Bluestone, 1966; Parsonnet et al, 1967; Sutton et al, 1968; Beregovich et al, 1969), either it had been recognised before the infarction or the previous state of AV conduction was unknown (Penton et al, 1956; Gilchrist, 1958; Fiedberg et al, 1964; Beregovich et al, 1969). Sudden late deaths do occur up to three or four weeks after the return of normal AV conduction (Epstein et al, 1966; Harris and Bluestone, 1966; Sutton et al, 1968; Norris, 1969) and although these deaths may have been due to recurrence of heart block, the mode of death/
death has usually not been established. In only one of the five late deaths in the present series was the rhythm at the time of death known and in this case it was ventricular fibrillation.

Because CHB is both an early and a transient complication of acute myocardial infarction, the incidence of this disorder in a series will depend on the average time between onset of symptoms of acute infarction and the establishment of ECG monitoring. Thus Adgey et al (1968) in their study with a mobile coronary care unit found that 32% of patients seen within two hours had, or subsequently developed, advanced heart block compared with only 10% of those first seen later than this ($X^2 = 35.12, P < 0.0005$). These authors do not, however, distinguish between second and third degree block in their report.

Other factors which affect the incidence of CHB in any particular series of acute myocardial infarction are the admission policy, which determines the selection of cases, and the particular diagnostic criteria which are used to define CHB. Thus, a high incidence of CHB will be obtained if patients with CHB or complete BBB are selectively admitted to a unit, as they were in the present study. Similarly, if patients are included who have AV dissociation of the interference type or transient CHB after direct current shock for ventricular arrhythmias, the incidence of CHB will be over-estimated (McNally and Benchimol, 1968).

None of the reports of CHB complicating acute myocardial infarction provide adequate information on which an accurate estimate of the incidence of CHB in this circumstance can be based. Probably the/
the best series is that of Julian et al (1964) in which an incidence of 8% was found in patients who had developed symptoms of infarction within the preceding 48 hours. However, the series was relatively small and the mean duration between onset of symptoms and admission was not reported. Furthermore, in their classification of cause of death these authors did not distinguish between complete cardiac standstill and ventricular asystole with continuing atrial activity. It is possible, therefore, that two of their patients who died in asystole may have had CHB (D. G. Julian, personal communication).

Site of Infarction

In the present series 38% of the cases of CHB followed anterior infarction. A similar incidence of between 41 to 47% has been found in other recent studies employing continuous electrocardiographic monitoring (Scott et al, 1967; Stock and Macken, 1968; Beregovich et al, 1969; Norris, 1969). However, in early, unmonitored series, CHB was invariably found to complicate inferior infarction very much more commonly than anterior infarction. For example, in the series of Master et al (1938) and of Courter et al (1963) all patients with CHB had had inferior infarctions. This finding is due in part, however, to the fact that the onset of heart block in anterior infarction is often sudden and unexpected and frequently an idioventricular pacemaker fails to emerge (Godman, M.J., Lassers, B.W., and Julian, D.G., unpublished observations). Therefore, many patients with anterior infarction and CHB die suddenly without any disturbance of conduction having been detected unless they have continuous/
continuous ECG monitoring.

Prognostic Features

The site of infarction and the duration of the QRS complex in CHB were found to be of considerable prognostic significance. Involvement of the anterior wall of the heart was associated with a high mortality, while both inferior and inferolateral infarction carried a better prognosis. Thus the mortality of 87% in anterior infarction was considerably greater than the mortality of 28% in inferior infarction (p < 0.0005) and significantly greater than the mortality of 44% in inferolateral infarction (p < 0.01). The somewhat higher mortality in inferolateral compared with isolated inferior infarction was not significant (Table 1:3).

All patients with anterior infarction had prolonged QRS complexes in CHB, but this feature was found to be of prognostic value in patients with isolated inferior infarction. Thus in inferior wall infarction with a prolonged QRS complex, 67% of patients died compared with only 17% of patients with a complex of less than 0.12 sec (Table 1:7).

Pathogenesis of CHB and Preceding Disorders of Conduction

The mechanism responsible for the interruption of AV conduction in inferior infarction is probably different from that producing heart block in anterior infarction. In the present study 12 of the 14 patients with ECG evidence of inferior infarction who came to necropsy were found to have occlusion of either the right coronary artery or the/
In the remaining two patients there was severe stenosis of all three major arteries without occlusion but with definite evidence of infarction of the inferior surface of the heart. In approximately 90% of hearts the AV node and main bundle receive their blood supply from a branch of the right coronary artery (Gross, 1921; James, 1968). In the remaining 10% of hearts these structures are supplied by a branch of the left circumflex artery. In either case the main artery from which the blood supply to the node derives is also the major source of blood to the inferior surface of the heart. Detailed necropsy studies have shown that when AV block complicates inferior infarction the coronary artery occluded is the one that supplies the node, but that the nodal branch itself is rarely involved (Sutton and Davies, 1968).

In inferior or inferolateral infarction, when AV block results from diminished perfusion of the AV node and bundle, it is common for complete block to be preceded by a lesser degree of AV block. Thus, 81% of the patients in the present series who developed CHB after admission had second degree AV block detected earlier (Table 1:4). Although many of these patients also had first degree block, only two patients developed CHB without interceding second degree block having been detected. This finding is important in determining the indications for pacing electrode insertion (see page 64).

Although diminished perfusion of the AV node is almost certainly the cause of disturbed AV conduction in inferior infarction, the exact mechanism whereby it produces block is not known. In most cases which have come to necropsy and in which the conduction tissue has been/
been carefully examined, major structural damage to the AV node and bundle has not been present (Blondeau, 1961; Lenegre, 1964; James, 1968; Sutton and Davies, 1968). In some cases these structures are histologically normal and in others there are small foci of damage which have been judged insufficient in themselves to produce interruption of conduction (Sutton and Davies, 1968). In view of this and the fact that block is almost always a transient event in those who survive, a variety of other factors have been invoked to explain heart block in inferior infarction. James (1958) has attributed it to oedema and inflammation of the node, but these features are difficult to evaluate and were not observed in the study of Sutton and Davies (1968). Hypoxia of the node by itself might be expected to produce block within the first few minutes of an occlusion as it does in animals (Hashiba, 1966), but it is difficult to account for block developing 12 to 48 hours later on this basis. Excessive vagotonia due to stimulation of cholinergic nerves and ganglia in the region of the posterior septum may contribute to heart block and may also explain the partial response of some patients to atropine (see page 80) (James, 1968). Depression of nodal function by substances released from infarcted tissue adjacent to the node has also been postulated, although no such substance has yet been identified (Sutton and Davies, 1968).

When CHB accompanied ECG evidence of anterior infarction in the present series, occlusion of the anterior descending coronary artery was demonstrated in 64% of cases that came to necropsy and severe stenosis of this artery was found in the remaining 36%. Extensive infarction/
infarction of the anterior wall and anteroseptal region was evident in 93% of these patients. This experience is similar to that of Blondeau et al (1961) and Sutton and Davies (1968).

In these patients with anterior infarction CHB is almost invariably preceded by complete BBB: 96% of the patients with anterior infarcts in the present series who developed CHB after admission had ECG evidence of block of one or other bundle, and 58% of these had ECG patterns suggestive of bilateral BBB detected prior to the onset of CHB (Table 1:5).

In view of this association with ECG evidence of BBB and the extensive anteroseptal damage which is usually found at necropsy in patients with CHB following anterior infarction, it has been postulated that the cause of the disruption of conduction in infarction in this site, is damage to both bundle branches (Master et al, 1938; Blondeau, 1961; Lenegre, 1964; Friedberg et al, 1968; McNally and Benchimol, 1968). Sutton and Davies (1968) examined the conduction tissue in five cases of isolated anterior infarction with CHB and found severe damage to both bundles in each case. On the other hand, James (1968) believes that the region of the AV node and bundle of His must also be ischaemic to produce CHB in these patients, although the lesion responsible for this may be either old or recent, and he states that all examples which he has studied postmortem had appropriate lesions which could produce such ischaemia. Sutton and Davies (1968) found this to be the case in their patients with ECG evidence of combined anterior and inferior infarction, but not in those with isolated anterior involvement present on the ECG. In the present study 77%
77% of the patients with the ECG appearance of isolated anterior infarction had necropsy evidence of old or recent infarction of the inferior surface of the heart as well. 18% had recent right coronary artery occlusions and a further 55% had severe stenosis of this artery. Although this high incidence of associated involvement of the inferior aspect of the heart and the right coronary artery might support the contention that there must be associated ischaemia of the AV node and bundle, histological examination of the conduction tissue was not performed, and the morphological state of these structures and of the bundle branches is not known. However, even with such knowledge, it is not possible to infer the functional capacity of the conducting tissues from their histological appearance alone.

In view of the high incidence of uni- and bilateral BBB before CHB in anterior infarction, the rarity of preceding partial AV block in these cases and the observations of severe damage to both bundles in a number of carefully studied cases, it does seem likely that at least some cases of CHB complicating acute anterior infarction are due to interruption of both bundle branches rather than primarily to associated ischaemia of the AV node.

There is little information on the pathological findings in patients with the ECG pattern of inferolateral infarction and CHB. However, most patients with infarction in this site have occlusion of the left circumflex coronary artery (James, 1968). Since this artery supplies the AV node in approximately 10% of people, it is likely that the mechanism of production of AV block in these cases is similar to that of isolated inferior infarction. In the present series/
series the clinical behaviour of the nine patients with infarcts in this site was similar to that of the patients with inferior infarction in that those patients with inferolateral infarction who developed CHB while being monitored had preceding second degree AV block (Table 1:4) and none of them had complete BBB patterns before CHB or prolonged QRS complexes during CHB (Table 1:6). In addition, their mortality did not differ significantly from that found in inferior infarction but was significantly lower than the mortality in anterior infarction (Table 1:3). However, the pathological findings in the present study are inconclusive in that only one patient with ECG evidence of inferolateral infarction came to necropsy and he had severe stenosis of all three coronary arteries with infarction of both surfaces of the heart.
SECTION TWO

HAEMODYNAMIC STUDIES
In order to determine the haemodynamic alterations associated with the early hours of advanced heart block complicating acute myocardial infarction and their response to ventricular pacing at various rates, 13 consecutive patients were studied. In five of these patients measurements were repeated on the second and subsequent days of heart block and after return to normal AV conduction to assess changes in circulatory status. During the five-month period in which these investigations were carried out, eight other patients with CHB following acute infarction were admitted to the coronary care unit. Studies were initiated in these patients, but seven of them died before electrodes could be inserted or studies completed and measurements were not obtained in the eighth patient for technical reasons.

METHODS

Patients

The clinical detail of the 13 patients who were studied are given in Table 2:1. All had developed CHB within 4.4 days of the onset of symptoms of infarction and the mean duration between onset of symptoms of infarction and the development of CHB was 28 hours. All patients were studied within 24 hours of the onset of CHB and most within six hours. In all patients CHB had been present at the beginning of the study: 11 remained in CHB throughout the studies on the first day, but two developed second degree block immediately after commencing the study. No patient had received digitalis, a diuretic, a/
<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>Mental State</th>
<th>RVF</th>
<th>Dyspnoea</th>
<th>Creps</th>
<th>X-ray</th>
<th>Duration of advanced heart block (hr)</th>
<th>Outcome and rhythm at discharge from hospital</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>0</td>
<td>+</td>
<td>O</td>
<td>60</td>
<td>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>Confused, inattentive</td>
<td>0</td>
<td>0</td>
<td>+</td>
<td>NT</td>
<td>184</td>
<td>Alive - SR</td>
</tr>
<tr>
<td>H.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>0</td>
<td>+</td>
<td>NT</td>
<td>108</td>
<td>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>Inattentive</td>
<td>+</td>
<td>0</td>
<td>+</td>
<td>O</td>
<td>172</td>
<td>Alive - SR</td>
</tr>
<tr>
<td>J.N.</td>
<td>M</td>
<td>57</td>
<td>Inferior + old inferior</td>
<td>CHB</td>
<td>31</td>
<td>65/-*</td>
<td>Stokes-Adams</td>
<td>+</td>
<td>0</td>
<td>+</td>
<td>+</td>
<td>174</td>
<td>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>Slightly inattentive</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>56</td>
<td>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>0</td>
<td>0</td>
<td>+</td>
<td>27</td>
<td>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>Confused, inattentive</td>
<td>+</td>
<td>0</td>
<td>0</td>
<td>NBC</td>
<td>15</td>
<td>Died in CHB</td>
</tr>
<tr>
<td>C.C.</td>
<td>F</td>
<td>82</td>
<td>Inferior</td>
<td>CHB</td>
<td>40</td>
<td>80/-</td>
<td>Inattentive</td>
<td>+</td>
<td>0</td>
<td>+</td>
<td>O</td>
<td>153</td>
<td>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>Faint, inattentive</td>
<td>+</td>
<td>0</td>
<td>0</td>
<td>O</td>
<td>4</td>
<td>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>0</td>
<td>0</td>
<td>0</td>
<td>3</td>
<td>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, confused</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>34</td>
<td>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>+</td>
<td>+</td>
<td>NBC</td>
<td>19</td>
<td>Died in CHB</td>
</tr>
</tbody>
</table>

Table 2:1 - Clinical details of patients in haemodynamic study.

*Known hypertensive.

Abbreviations: RVF = right ventricular failure (jugular venous pressure above clavicle at 45°); LVF = left ventricular failure (dyspnoea at rest, or radiographic evidence of pulmonary venous congestion); NT = not taken; NBC = no radiograph taken, but necropsy evidence of pulmonary congestion; CHB = complete heart block; 2° = second degree heart block; SR = sinus rhythm; and AF = atrial fibrillation.
a sympathomimetic agent or an antiarrhythmic drug. Analgesics had not been given within three hours of any study and only one patient had received oxygen within an hour of the study.

Two patients died while in CHB: one of shock in spite of effective electrical pacing and one of asystole unresponsive to pacing. Normal AV conduction returned in the remaining 11. These 11 patients survived to be discharged from hospital.

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 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/
artery and advanced centrally. Pressures were transduced with Sanborn 267B manometers or Bell and Howell 4-327-L221 strain-gage manometers and recorded either on a Mingograf 24B ink-jet recorder or on a Devices MI4 direct-writing recorder. Zero reference level for the manometers was taken as five centimeters below the level of the manubrium sterni. Mean pressures were obtained by electrical integration. The pulmonary arterial diastolic pressure was used as an estimate of the pulmonary artery wedge or left atrial mean pressure (Lassers et al, 1969).

Expired air was breathed through a dry gas meter, the volume recorded in litres on a direct-writing recorder, and the oxygen and carbon dioxide content analysed by the Lloyd-Haldane method. During a five minute expired air collection period, arterial and mixed venous blood samples were withdrawn continuously and analysed for oxygen tension, carbon dioxide tension and pH using Radiometer equipment. The oxygen saturation of the blood was derived from Dill's desaturation table and the oxygen content was calculated from the saturation and the haemoglobin 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 used to derive the systemic vascular resistance/
resistance and the tension time index:

\[
\text{SVR} = \frac{(BP_A - BP_{RA}) \times 80}{CI}
\]

\[
\text{TTI} = \frac{MSEP \times T_s \times HR}{CI}
\]

Where:

\( \text{SVR} \) = Systemic vascular resistance (dyne-sec cm \(^{-5}\)/m\(^2\))

\( BP_A \) = Mean systemic arterial blood pressure (mm Hg)

\( BP_{RA} \) = Mean right atrial blood pressure (mm Hg)

\( CI \) = Cardiac Index (L/min/m\(^2\))

\( TTI \) = Tension time index (mm Hg sec/min)

\( MSEP \) = Mean systolic ejection pressure (mm Hg)

\( T_s \) = Duration of systole (sec)

\( HR \) = Heart rate (beats/min)

Mean systolic ejection pressure was 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 substituted.

Results are presented in the text and the tables as the mean value ± one standard deviation (SD), but in the figures they are presented as the mean value ± one standard error of the mean (SEM) unless otherwise stated/
<table>
<thead>
<tr>
<th>Patient</th>
<th>Clinical Features</th>
<th>Haemodynamics</th>
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<tbody>
<tr>
<td></td>
<td>Mental Function</td>
<td>Cardiac Index</td>
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<td></td>
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<td>1. J.M.</td>
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<td>2. W.B.</td>
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</tr>
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<td>3. G.R.</td>
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<td>4. M.F.</td>
<td>Confused, Stokes-</td>
<td>42</td>
</tr>
<tr>
<td></td>
<td>Adams</td>
<td></td>
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<td>5. J.R.</td>
<td>Inattentive, Faint</td>
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</tr>
<tr>
<td>6. C.C.</td>
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<td>7. R.C.</td>
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<td>8. W.H.</td>
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<td>9. J.D.</td>
<td>Normal</td>
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</tr>
<tr>
<td>10. W.D.</td>
<td>Slightly inattentive</td>
<td>51</td>
</tr>
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<td>11. C.O.</td>
<td>Normal</td>
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<td></td>
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<td>49</td>
</tr>
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<td>SD</td>
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</table>

* Mean pressure in brackets.

Table 2:2 - Clinical and haemodynamic features in 11 patients on the first day of complete heart block complicating acute myocardial infarction arranged in order of increasing cardiac index.
(the "higher output group"), had cardiac indices above 2.00 L/min/m² and their average cardiac index of 2.64 ± 0.45 L/min/m² was significantly higher than the average cardiac index in the low output group (p < 0.005). In contrast to the low output group, these four patients had normal or nearly normal mental function and good skin circulation.

Heart Rate

The average heart rate was 49 ± 7/min (Table 2:2). The average rate of 48 ± 7/min in the low output group was not significantly different from the average rate of 51 ± 8/min in the higher output group.

Stroke Index

The average stroke index in the 11 patients was 40 ± 13 ml/m² (Table 2:2). In the first six patients in Table 2:2, small stroke indices together with relatively slow heart rates produced the low cardiac indices. In the seventh patient, although the stroke index was within normal limits, the heart rate was only 40/min, and this resulted in a low cardiac index. Patient eight (W.H.) had a low stroke index but a heart rate high enough to maintain an adequate flow. In the last three patients in Table 2:2 the stroke indices were well maintained and the cardiac indices adequate.

Mean Systemic Arterial Blood Pressure

The average arterial blood pressure for the 11 patients was below normal at 80 ± 16 mm Hg (Table 2: 2). The average pressure of 78/
78 ± 18 mm Hg in the low output group was not significantly different from the average pressure of 83 ± 15 mm Hg in the higher output group.

**Systemic Vascular Resistance**

The mean systemic vascular resistance for the 11 patients was 3140 ± 1160 dyne-sec cm⁻⁵/m² (Table 2:2). The average resistance of 3660 ± 1040 dyne-sec cm⁻⁵/m² in the low output group was significantly higher than the average resistance of 2240 ± 780 dyne-sec cm⁻⁵/m² in the higher output group (p < 0.05). Thus an increase in systemic vascular resistance in the patients with the lower cardiac indices was responsible for maintaining the arterial blood pressure within a range similar to that found in the patients with higher indices.

**Pulmonary Arterial Diastolic Pressure**

The average pulmonary arterial diastolic pressure for the 11 patients of 12 ± 5 mm Hg was within normal limits (Table 2:2). The average pressure of 13 ± 2 mm Hg in the low output group was not significantly different from the average pressure of 10 ± 8 mm Hg in the higher output group.

**Right Atrial Mean Pressure**

The average right atrial mean pressure for the 11 patients in heart block was elevated at 9 ± 4 mm Hg (Table 2:2). The average pressure in the higher output group of 12 ± 6 mm Hg was greater than that of the low output group (8 ± 3 mm Hg), but this difference was not/
not significant.

**Effects of Pacing**

**Cardiac Index**

In eight patients measurements were made with pacing at three or more rates. In six of the eight, control measurements in heart block were also made (Table A:2, Appendix). Figure 2:1 shows the effect of increasing ventricular rate on the cardiac index. 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. In only two patients was an increase in rate accompanied by a fall in cardiac index: in patient C.C. the cardiac index fell from 2.86 to 2.20 L/min/m² when the rate was increased from 107 to 125/min, and in patient J.D. it fell from 3.70 to 3.34 L/min/m² when the rate was increased from 115 to 130/min (Figure 2:1, Figure 2: 6 and Table A:3). Although the maximal cardiac output response occurred above 100/min, an adequate increase in cardiac output had occurred with rates between 80 to 90/min in most patients.

In one patient (J.M.) who was semi-conscious with intense cutaneous vasoconstriction and severe acidosis (pH 7.03), and whose clinical condition appeared to be deteriorating steadily, the cardiac index increased slightly from 1.21 to 1.44 L/min/m² with pacing at 83/min, but fell to 0.86 L/min/m² when the rate was increased to 104/min (Figure 2:7 and Table A:4, Appendix).
Figure 2:1  Effect of increasing ventricular rate by pacing on the cardiac index.
Figure 2.2 Effect of increasing ventricular rate by pacing on the stroke index.
Figure 2: Effect of increasing ventricular rate by pacing on the mean systemic arterial blood pressure.
**Figure 2.4** Effect of increasing ventricular rate by pacing on the systemic vascular resistance.
Figure 2:5 Effect of increasing ventricular rate by pacing on the pulmonary arterial diastolic blood pressure.
Figure 2:6 Haemodynamic effects of increasing ventricular rate by pacing in the patient J.D. with CHB complicating acute myocardial infarction.
Figure 2:7 Haemodynamic effects of increasing ventricular rate by pacing in patient J.M. with CHB and cardiogenic shock complicating acute myocardial infarction.
**Figure 2:8** Effect of pacing at the rate associated with the maximal cardiac index on haemodynamic measurements in CHB complicating acute myocardial infarction (mean and standard error of the mean).
Figure 2:9  Effect of increasing ventricular rate by pacing on the tension time index in CHB complicating acute myocardial infarction. Dashed line is exponential regression line.
In Figure 2:8A the average increase in cardiac index produced by pacing at the rate associated with the highest measured cardiac index (maximal output rate) in the low output group is compared with the average increase in the higher output group. The actual measurements for the individual patients are presented in Table A: 5 in the Appendix. The mean maximal output rate of the low output group was 91 ± 16/min and was not significantly different from the mean maximal output rate of 99 ± 17/min in the higher output group. There was a significant increase in the cardiac index with pacing at the maximal output rate in both groups of patients. Thus in the low output group it increased by 47% from 1.56 ± 0.13 to 2.27 ± 0.20 L/min/m² (p < 0.005) and in the higher output group by 36% from 2.64 ± 0.45 to 3.59 ± 0.25 L/min/m² (p < 0.01). However, the average increase in the lower output group was not significantly greater than the increase in the higher output group.

**Stroke Index**

In most patients paced at three or more rates there was a progressive fall in stroke index as ventricular rate was increased. (Figure 2:2). However, in patient J.D., increasing ventricular rate from 69 to 87/min reduced stroke index from 40 to only 39 ml/m², and in patient C.C., increasing the rate from 87 to 107/min produced no change in stroke index (Figure 2:2 and Figure 2:6).

Pacing at maximal output rates was associated with a fall in stroke index in all but two cases. In patient G.R., the stroke index changed insignificantly from 29 to 31 ml/m² when the heart rate was/
was increased from 44 to 80/min and in patient W.H. it rose from 37 to 46 ml/m² when the rate was increased from 60 to 75/min (Table A:4, Appendix). The average stroke index of the low output group decreased by 24% from 33 ± 9 to 25 ± 6 ml/m², and of the higher output group by 30% from 53 ± 11 to 37 ± 6 ml/m² (Figure 2:8B).

Mean Systemic Arterial Blood Pressure

Blood pressure was measured in heart block and during pacing at three or more rates in all eight patients (Figure 2:3). 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 and 75/min respectively, before it rose with higher rates. In patient G.R. this fall in blood pressure was associated with a fall in cardiac index, but in patient C.O. cardiac index had risen. In patient J.D. blood pressure rose when the rate was increased from 115 to 130/min although the cardiac index fell (Figure 2:6). With these exceptions changes in blood pressure tended to parallel the changes in cardiac index produced by pacing.

In the low output group pacing increased the average mean arterial blood pressure by 23% from 78 ± 18 mm Hg to 96 ± 23 mm Hg (p < 0.01), and in the higher output group it also increased it by 23% from 82 ± 15 mm Hg to 101 ± 20 mm Hg (p < 0.01) (Figure 2:8C).

Systemic Vascular Resistance

In seven of the eight patients paced at three or more rates the systemic vascular resistance increased slightly with increasing ventricular/
ventricular rates (Figure 2:4). In the eighth patient (J.D.), who had the highest blood pressure and the largest increments in cardiac index, the resistance fell slightly as the rate was increased (Figures 2:1, 2:4, and 2:6).

Pacing at the maximal output rate decreased the average systemic vascular resistance of the low output group of patients by 16% from 3660 ± 1040 to 3090 ± 540 dyne-sec cm⁻⁵/m² and of the higher output group by 9% from 2240 ± 390 to 2030 ± 230 dyne-sec cm⁻⁵/m², but these small decreases were not significant (Figure 2:8D).

Tension Time Index

Increasing ventricular rate by pacing produced an exponential rise in the tension time index (Figure 2:9).

Pulmonary Arterial Diastolic Pressure

The pulmonary arterial diastolic pressure rose with increase in ventricular rate in all eight patients paced at three or more rates, but the change was small and in no case did it exceed 4 mm Hg (Figure 2:5). Pacing at the maximal output rate increased the average pulmonary arterial diastolic pressure in the low output group of patients from 13 to 15 mm Hg (p < 0.05), and the average pressure in the higher output group from 10 to 12 mm Hg, but this change was not significant (p < 0.10).

Mean Right Atrial Pressure

Increasing ventricular rate by pacing had no significant effect
on right atrial pressure in the eight patients paced at three or more rates (Table A:2, Appendix) and did not significantly affect the pressure in either the low or higher output groups.

**Duration of Haemodynamic Effects of Pacing**

In order to determine the time course of the response to pacing, patient R.C. was studied for a control period of 15 minutes in heart block and then paced at 83/min for 60 minutes with repeated measurements throughout the period of study (Figure 2:10 and Table A:6, Appendix). The maximal cardiac index was reached within 10 minutes and the cardiac index, stroke index, mean systemic arterial blood pressure, systemic vascular resistance, pulmonary arterial diastolic pressure and right atrial mean pressure all remained relatively steady over the period of pacing.

**Clinical Effects of Pacing**

Those patients in whom the initial cardiac index was low showed considerable improvement in mental state and skin circulation when the cardiac index was increased by pacing. These signs were found to be superior to blood pressure in clinical evaluation of the adequacy of the cardiac output response to pacing. With continued pacing, the improvement in these signs persisted for many hours.
Figure 2:10  Haemodynamic effect of pacing at 83/min for 60 minutes in patient R.C. with CHB complicating acute myocardial infarction.
Measurements on Subsequent Days of Heart Block and After Return of Normal AV Conduction

Figure 2:11 and Table A:7 in the Appendix show the findings in the five patients who were studied on subsequent days while still in heart block and after return to normal AV conduction with sinus rhythm. In all patients except W.B., stroke index had either changed very little or had increased despite relatively large increases in heart rate. This resulted in a substantial rise in cardiac index on subsequent days. In patient W.H. who remained in CHB for two days, the rise in cardiac index occurred on the third day with the development of second degree heart block and there was little further change after the return of normal AV conduction on the fifth day. In the case of patient G.R., who was studied in CHB on three successive days, there was a considerable increase in cardiac index on the second day of CHB due both to an increase in stroke index and an acceleration of the idioventricular rate. Patient M.F. returned to sinus rhythm with a tachycardia of 103/min and first degree heart block on the second day. This was accompanied by a fall in stroke index, but an increase in cardiac index. The stroke index and cardiac index were well maintained in CHB in patient C.O., but there was a further rise in cardiac index with the increase in heart rate associated with the return of normal AV conduction on the third day. Patient W.B. had a low stroke index and a low cardiac index in CHB which persisted for eight days and there had been little improvement when measurements were repeated after the return of normal AV conduction on the tenth day.

Although/
CARDIAC INDEX
1./min./sq.m

STROKE INDEX
ml./sq.m

MEAN SYSTEMIC ARTERIAL B.P.
mm. Hg.

SYSTEMIC VASCULAR RESISTANCE
dyne. sec. cm.\(^5\)/sq.m

Figure 2:11 Haemodynamic changes on the first and subsequent days of heart block and after return to sinus rhythm in five patients with acute myocardial infarction.
Although systemic arterial blood pressure changes tended to parallel the changes in cardiac index produced by pacing, there was no consistent relationship between the absolute levels of cardiac index and the patients' blood pressure on the first or subsequent days of heart block or after return of normal AV conduction. Thus in patient W.H., the mean blood pressure rose from 80 mm Hg on the first day in CHB to 90 mm Hg on the second day in CHB despite a small decrease in cardiac index. Similarly, patient W.B. showed a progressive rise in blood pressure with virtually no change in cardiac index. On the other hand, patient G.R. showed little change in blood pressure despite a very large increase in cardiac index, and in patient M.F., the blood pressure decreased from 100 to 85 mm Hg on the second day with the return of sinus rhythm although the cardiac index increased 64%. These variable changes in the blood pressure with alterations in cardiac index were due to the inconsistent changes in systemic vascular resistance.

There was no uniform change in pulmonary arterial diastolic or right atrial mean pressure on subsequent days of heart block or after return of normal AV conduction (Table A:7, Appendix).

DISCUSSION

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. Although CHB complicates anterior infarction/
infarction almost as frequently as inferior infarction, all 13 patients in the present study had infarcts involving the inferior surface of the heart (Table 2: 1). The reason for this is probably that when CHB follows anterior infarction it is usually associated with extensive myocardial damage, the onset of block is frequently sudden and commonly an idioventricular pacemaker fails to emerge. Because of this such patients frequently die soon after the onset of CHB or despite pacing. Thus, seven of the eight other patients with CHB complicating acute infarction who were managed in the coronary care unit during the period of the study had anterior infarcts and all died before pacing electrodes and catheters could be inserted or complete measurements made. In view of this, although the observations which were made are probably representative of the spectrum of circulatory changes found in CHB complicating inferior infarction, the changes found following anterior infarction with CHB are probably dissimilar. Furthermore, it is likely that the haemodynamic response to pacing differs considerably in patients with anterior infarction.

**Findings in Heart Block**

The cardiac output in patients in the present study with CHB complicating acute myocardial infarction was usually, but not invariably, severely reduced. Most patients were unable to increase stroke volume to compensate for the bradycardia and maintain an adequate flow. Since pulmonary arterial diastolic and, therefore, left atrial pressures were normal or elevated in all patients, venous return was sufficient and the failure to increase stroke volume probably reflects depression/
depression of myocardial contractility.

Since the stroke volume in heart block varied considerably among patients, heart rate was not a reliable guide to the adequacy of the cardiac output. Patients with rates of less than 45/min usually had low cardiac outputs; but in several patients myocardial function was obviously severely depressed with very low stroke volumes and low cardiac outputs despite heart rates as high as 50 to 61/min. In contrast, however, patient J.D. maintained an adequate stroke volume and, therefore, a satisfactory cardiac output despite a bradycardia of 41/min.

Similarly, arterial blood pressure was unreliable in predicting cardiac output because of considerable variations in systemic vascular resistance. There were, on the one hand, patients in whom a low systemic vascular resistance resulted in low blood pressures despite well-maintained cardiac outputs, and on the other, patients in whom a high vascular resistance produced relatively well-maintained blood pressures in the face of very low cardiac outputs.

In the clinical assessment of the adequacy of the cardiac output, the patient's mental state and skin circulation provided the most useful information. Thus seven patients who had varying degrees of inattention, confusion, restlessness and poor skin circulation had cardiac indices of less than 2.00 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.
Effects of Pacing

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. There was little difference between the group of patients with cardiac indices in heart block below 2.00 l/min/m² and the group with cardiac indices above this value in the response of stroke output, cardiac output or blood pressures to an increase in ventricular rate by pacing. However, those patients in whom the initial cardiac output 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 haemodynamic 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 severe cardiogenic shock, cardiac index fell when the rate was increased to 104/min. The rate associated with the maximal cardiac output 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 maximal output rates is considerably higher than the range of 60 to 80/min which has been reported in chronic CHB (Benchimol et al, 1964; Escher et al, 1964; Judge et al, 1964; Segel et al, 1964; Sowton, 1964; Samet and Bernstein, 1966).

The/
The effect on myocardial function of increasing cardiac output and systemic blood pressure in CHB complicating acute myocardial infarction has not been established. In the patients in whom the stroke output changed very little or actually increased 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 output and blood pressure. In most patients, however, stroke output fell in response to pacing in spite of still subnormal cardiac outputs and in these patients myocardial performance was probably abnormal. In most instances in which measurements were repeated after the first day of CHB, stroke output 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 response with increasing rates. In the present study a regular increase and decrease in systemic arterial pressure was observed 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 abolished by sequential atrio-ventricular pacing (Chamberlain et al, 1969). However, the extent of the contribution of atrial systole to ventricular filling in the context of ischaemic heart disease is not clear. Gillespie and associates (1967) have demonstrated that the augmentation of stroke volume due to optimally timed/
timed atrial systole is of much less importance in the diseased than in the normal heart. On the other hand, a recent study of sequential atrio-ventricular pacing in CHB complicating acute myocardial infarction demonstrated an additional 24% increase in cardiac output when atrial transport was restored by pacing the right atrium as well as the right ventricle (Chamberlain et al, 1969).

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 (Katz et al, 1958; Sarnoff et al, 1958; Monroe and French, 1961; Rolett et al, 1965). In the normal heart a rise in oxygen demand is met by an increase in coronary flow (Katz and Shaffer, 1966). This is also true in most cases of chronic CHB when the ventricular rate is increased by pacing (Gregory et al, 1967; Hedworth-Whitty et al, 1968). However, when coronary flow is restricted following coronary thrombosis, this may limit myocardial oxygen uptake (Kahler et al, 1963; Weisberg et al, 1963; Bacaner et al, 1965) and "set a limit to the ability of the heart to speed up its rate without suffering a further decline in work capacity" (Katz and Shaffer, 1966). This is evidenced clinically by the occasional patient who develops angina or dyspnoea 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 favourable effect on myocardial oxygen requirements by decreasing ventricular end-diastolic volume and, thus, reducing ventricular wall tension (Sarnoff/
(Sarnoff et al., 1958; Rodbard et al., 1969).
SECTION THREE

ARTIFICIAL PACING
Pacing electrodes were inserted in 95 of the 100 patients described in Section One (Table 3:1). Three of the five patients in whom electrodes were not introduced died soon after admission and before electrodes could be inserted. Electrodes were not inserted electively in the remaining two patients early in the series. One of these, who survived, had CHB for only a few seconds during a period of vomiting. In the other, who died, an electrode was not inserted because severe cardiogenic shock had been present for some hours.

In this section the methods of pacemaking that were employed in the present series will be described. The benefits produced by its use and the complications encountered will also be outlined. On the basis of both these results and the clinical and haemodynamic observations previously described, the techniques of artificial pacing in acute infarction and the indications for electrode insertion and removal and for the initiation of pacing will be discussed.

**METHODS**

**Electrode Insertion**

Pacing electrodes were inserted routinely in all patients with second or third degree AV block apart from the exceptions mentioned above. In addition, for a period of one year, transvenous electrodes were also inserted into patients with complete BBB irrespective of the state of AV conduction (Goedman, M.J., Lassers, B.W., and Julian, D.G., unpublished/
<table>
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<tr>
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Table 3:1  Type and site of electrode insertion in patients with CHB complicating acute myocardial infarction.
unpublished).

Of the 95 patients with CHB in whom electrodes were inserted, 87 had bipolar electrodes (USCI, size 5Fr) inserted by the transvenous route. In the remaining eight patients percutaneous transthoracic electrodes (Elecath) were introduced directly into the heart. Transvenous electrodes were passed from an antecubital vein in the first 18 patients, but subsequently they were inserted percutaneously from the subclavian vein by either the supraclavicular (Mobin-Uddin et al, 1967) or infraclavicular (Vellani et al, 1969) techniques.

At first patients were transferred to the cardiac laboratory for insertion of electrodes under fluoroscopic control, but later a portable image intensifier was designed for use in the coronary care unit at the patient's bedside (Samuel, 1968). Right ventricular outflow tract pacing was not used and all electrodes were positioned with the tip at the apex of the right ventricle. The electrode position was accepted as satisfactory only if the contraction threshold was less than 1.0 volt.

**Pulse Generators**

Continuously discharging variable-rate, variable-current pulse generators incorporated in the Sanborn Visomonitor were originally used for pacing. The minimum current of 5 milliamps obtainable with this instrument was found to be above the contraction threshold in all patients in whom the electrode was properly/
properly positioned. Because of the risk of pacemaker-induced ventricular fibrillation under these circumstances, this apparatus was abandoned. Devices battery-operated, continuously discharging variable-voltage units were substituted and used until ventricular demand pacemakers became available. These demand units were employed in the last 52 patients in the series.

The pulse generator was set to discharge at a voltage twice that of the contraction threshold at a pulse width of 2 milli-seconds. When continuously discharging pacemakers were used, the presence of CHB was not regarded as an indication for instituting pacing unless ventricular asystole occurred or a low cardiac output was suggested by clinical evidence of diminished cerebral and skin blood flow. When these features were present, the patient was paced at the slowest rate which produced an increase in blood pressure accompanied by clinical evidence of improved cerebral and skin circulation. Pacing was discontinued as soon as a return of AV conduction was detected. When ventricular demand pacemakers were used, the unit was set to inhibit if the patient's rate was above 70/min.

Drug Therapy

Atropine or isoprenaline were not used routinely in patients with second or third degree AV block either before or after electrode insertion and only a few patients received these drugs. Corticosteroids were not given. After electrodes had been positioned/
positioned, digoxin and diuretics were used when indicated
(Lawrie et al, 1967) and antiarrhythmic drugs were employed if
ventricular arrhythmias persisted after correction of bradycardia.

Removal of Electrodes

Electrodes were withdrawn after 72 hours had elapsed without
heart block of greater than first degree and after a further 24
hours the patient was transferred to a general medical ward.

RESULTS

Benefits of Pacing

The results of pacing were classified as follows:

(1) "Beneficial" if repeated attacks of ventricular
asystole were abolished or if the clinical
features associated with a low cardiac output
(see page 35) were reversed and the patient
survived to be discharged from hospital.

(2) "Equivocal" if demand pacing was employed for
periods of more than half hour without
previous evidence of asystole or a low output,
or if pacing had been used for marked brady-
cardia (less than 35/min) not accompanied by
obvious evidence of clinical deterioration.

(3)/
(3) "Not Beneficial" if it had no effect on the clinical state or if the patient died despite pacing.

(4) "Pacing Not Used" if:

(a) the clinical indications for pacing had not been present and, therefore, continuously discharging pacemakers had not been used; or

(b) demand pacing had occurred for only brief periods in patients with this type of instrument.

It can be seen from Table 3:2 that pacing was thought to have been of definite benefit in only 13 patients and of equivocal benefit in another 11. In the 13 patients categorised as "benefited", pacing had abolished ventricular asystole in four and corrected a low cardiac output state in nine. Of the 49 patients not benefited by pacing, 48 died; one patient survived despite the fact that pacing had to be discontinued because it produced angina and dyspnoea. Of the 48 patients who died, 15 had been temporarily benefited by pacing: in 10 pacing had abolished attacks of asystole and in five it had produced distinct, but transient, improvement in the circulatory state. In 22 patients electrodes were inserted, but pacing was not necessary.

Pacing was thought to have been of definite benefit in ten patients with inferior infarction and only two patients with anterior infarction/
<table>
<thead>
<tr>
<th>Site of Infarct</th>
<th>Pacing</th>
<th>Electrode</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Beneficial</td>
<td>Equivocal</td>
</tr>
<tr>
<td>Inferior</td>
<td>10 (19%)</td>
<td>10 (19%)</td>
</tr>
<tr>
<td>Anterior</td>
<td>2 (5%)</td>
<td>1 (3%)</td>
</tr>
<tr>
<td>Inferolateral</td>
<td>1 (11%)</td>
<td>0</td>
</tr>
<tr>
<td>Total</td>
<td>13</td>
<td>11</td>
</tr>
</tbody>
</table>

Percentages in brackets are percent of patients with infarcts in that site in that category.

Table 3:2 Benefits of pacing in CHB complicating acute myocardial infarction. For definition of categories, see text, page 54.
infarction; but the number of patients is small and this difference is not significant \(X^2 = 3.58, \ p > 0.05\). In both of the patients with anterior infarction, pacing had abolished attacks of ventricular asystole. Pacing was beneficial in eight of the ten patients with inferior infarction and the single patient with inferolateral infarction by correcting a low cardiac output state. The remaining two patients with inferior infarction had had attacks of ventricular asystole controlled by pacing.

Complications Associated with Pacing

(1) Arrhythmias

The most serious complications associated with pacing were ventricular tachycardia or fibrillation and ventricular asystole. In four patients, ventricular tachyarrhythmias requiring direct current shock were mechanically induced during electrode insertion (Table 3:3). In eight patients ventricular asystole occurred during manipulation of the electrode within the right ventricle. None of these 12 patients died directly as a result of the mechanically induced arrhythmia. In contrast to ventricular tachycardia and fibrillation which were more frequent in patients with QRS complexes of less than 0.12 sec, mechanically induced asystole occurred only in patients with prolonged QRS complexes. This was true irrespective of the site of infarction and occurred only in patients with a complete RBBB pattern. In three further patients atrial fibrillation was induced during electrode insertion or withdrawal and in one of these cases direct current cardioversion was/
<table>
<thead>
<tr>
<th>Site of Infarction</th>
<th>QRS Complex at time of electrode insertion</th>
<th>Complication</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>Asystole</td>
</tr>
<tr>
<td>Anterior</td>
<td>QRS &lt; 0.12 sec</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Unilateral BBB</td>
<td>3</td>
</tr>
<tr>
<td></td>
<td>Bilateral BBB</td>
<td>2</td>
</tr>
<tr>
<td>Inferior</td>
<td>QRS &lt; 0.12 sec</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Unilateral BBB</td>
<td>0</td>
</tr>
<tr>
<td></td>
<td>Bilateral BBB</td>
<td>3</td>
</tr>
<tr>
<td>Inferolateral</td>
<td>QRS &lt; 0.12 sec</td>
<td>0</td>
</tr>
<tr>
<td>Total</td>
<td></td>
<td>8</td>
</tr>
</tbody>
</table>

\* VT/VF: Ventricular tachycardia or fibrillation requiring DC shock

\*\* AF: Atrial fibrillation

**Table 3.3** Mechanical risks of electrode insertion and their relationship to the type of QRS complex present at the time of electrode insertion.

Comparison of incidence of asystole and ventricular tachyarrhythmia in patients with QRS complexes <0.12 sec v. patients with QRS complexes ≥0.12 sec: Exact $X^2$, $p = 0.02$.

Comparison of incidence of ventricular asystole and tachyarrhythmia in patients with inferior infarction v. patients with anterior infarction: Exact $X^2$, $P = 0.24$ (n.s.).
was required.

Seven additional cases of ventricular tachycardia or fibrillation requiring immediate direct current shock were associated with pacing (Table 3:4). In two of these patients recurrent episodes of ventricular fibrillation occurred during pacing. The first, who died, had asystole when pacing was discontinued and ventricular fibrillation whenever pacing was attempted. The second, who survived, continued to have recurrent ventricular fibrillation after AV conduction had returned and the electrode had been removed. In the remaining five patients, ventricular fibrillation occurred after AV conduction had returned and competition had developed between the patient's own rhythm and that of the pacemaker. Two of these patients died of ventricular fibrillation and their deaths were probably pacemaker-induced. Both patients had, however, required pacing for asystole and severe hypotension and would probably have died without it. Three of the instances of ventricular fibrillation associated with competition occurred with the Sanborn instrument, one with the Devices continuously discharging pulse generator, and one with the Devices demand pacemaker. In the four cases which occurred with continuously discharging units, competition had not been recognised or had been misinterpreted as a failure of electrical capture and the pacing stimulus voltage or current had been increased to a relatively high level. In the single case which occurred with the demand unit, failure of inhibition had developed probably due to a low intra-cardiac/
<table>
<thead>
<tr>
<th>Type of Pulse Generator</th>
<th>Number of Patients with Electrodes</th>
<th>Electrically Induced VT/VF</th>
<th>Deaths</th>
</tr>
</thead>
<tbody>
<tr>
<td>Set-Rate</td>
<td>43\textsuperscript{***}</td>
<td>6</td>
<td>3</td>
</tr>
<tr>
<td>Demand</td>
<td>52</td>
<td>1</td>
<td>0</td>
</tr>
<tr>
<td>Total</td>
<td>95\textsuperscript{***}</td>
<td>7</td>
<td>3</td>
</tr>
</tbody>
</table>

\textsuperscript{*} VT/VF: Ventricular tachycardia or fibrillation
\textsuperscript{**} Pacing actually employed in 31 patients
\textsuperscript{***} Electrodes not inserted in 5 patients

Table 3:4. Occurrence of electrically induced ventricular tachycardia or fibrillation requiring direct current shock in patients with set-rate or demand pacemakers. In patients with set-rate units, the generator was turned-on only if pacing was required (31 patients).
cardiac potential (see page 63).

(2) **Complications associated with the route of electrode insertion**

A number of complications were specifically related to the route of electrode insertion. Thus, displacement of the electrode tip requiring remanipulation occurred in 83% of the patients in whom the electrode was passed from an antecubital vein, despite the fact that the arm was usually splinted and bound (Table 3:5). In contrast, remanipulation for electrode displacement was necessary in only 6% of the patients in whom the electrode was introduced from the subclavian vein. Small pneumothoraces, which did not require treatment, occurred in two patients in whom the subclavian vein route was used.

(3) **Myocardial perforation**

Perforation of the myocardium by the electrode tip was thought to have occurred in ten patients (Table 3:5). This was invariably associated with a rise in contraction threshold and intermittent pacing or with a failure of a demand pacemaker to inhibit without displacement of the electrode evident on fluoroscopy. In a number of cases these changes were accompanied by pericardial pain and localised pericardial friction. Bleeding into the pericardial sac was not detected. Occasionally perforation was corrected by withdrawing the electrode slightly, but in most cases it required repositioning under fluoroscopio control.

(4) **Electrical failures**

Fractured/
<table>
<thead>
<tr>
<th>Complication</th>
<th>Number</th>
</tr>
</thead>
<tbody>
<tr>
<td>Electrode Displacement</td>
<td></td>
</tr>
<tr>
<td>Antecubital vein (18)</td>
<td>15 (83%)</td>
</tr>
<tr>
<td>Subclavian vein (69)</td>
<td>4 (6%)</td>
</tr>
<tr>
<td>Pneumothorax</td>
<td>2 (3%)</td>
</tr>
<tr>
<td>Perforation</td>
<td>10 (11%)</td>
</tr>
<tr>
<td>Electrical Faults</td>
<td></td>
</tr>
<tr>
<td>Pulse generators</td>
<td>5 (5%)</td>
</tr>
<tr>
<td>Fractured electrodes</td>
<td>3 (3%)</td>
</tr>
</tbody>
</table>

Displacement requiring remanipulation, but not including remanipulation for perforation

Figure in brackets is number of patients in whom electrode was inserted by that route

Percentage in brackets is percentage of patients at risk who developed this complication

All 5 were demand units

**Figure 3:5** Complications - other than electrically or mechanically induced arrhythmias - associated with pacing in CHB complicating acute myocardial infarction.
(4) **Electrical failures**

Fractured electrodes occurred in three cases and may have contributed to death in one of these. In five cases of failure of demand pulse generators to inhibit, this was not associated with a rise in the contraction threshold. In one of these cases this was attributed to battery failure with a voltage being produced which was adequate to activate the pulse generator but insufficient to operate the inhibit mechanism. In the remaining four cases failure of inhibition was thought to be due to low intracardiac potentials.

(5) **Infection**

Sepsis at the site of electrode insertion was not uncommon, but septicaemia did not occur.

**DISCUSSION**

**Techniques of Pacing in Acute Myocardial Infarction**

External transcutaneous pacemaking, which has been used by Zoll (1952) and Levowitz et al (1960) is usually ineffective in the treatment of asystole in myocardial infarction (Lown, 1967), and is unsuitable for the management of bradycardia because of pain and skin burns (Escher, 1967). The direct percutaneous insertion of an electrode into the heart may prove effective as an emergency measure. In the present series, this technique allowed control of the/
the heart rate in two patients who had developed sudden onset ventricular asystole. Transvenous electrodes were then inserted, but both patients subsequently died. It is difficult, however, to assess the potential of this method since it is usually employed only when the patient is in extremis and there is insufficient time to use another route (Gregory and Grace, 1968). Thus, the other six patients in whom transthoracic electrodes were inserted (Table 3:1), died although electrical capture of the heart was achieved, but without apparent mechanical effect. With direct percutaneous insertion there is also a risk of producing pneumothoraces, pericardial tamponade and coronary artery damage. However, few patients in whom this technique has been employed survive long enough to permit an accurate assessment of these dangers.

The most widely used technique of pacing has been that of introducing an electrode through a peripheral vein. The use of this technique in acute myocardial infarction was first described by Hurwitt (1960) and later by Delman et al (1963), De Sanctis (1963) and Zucker et al (1963). Various sites have been utilised including arm veins (Siddons and Sowton, 1967), external jugular veins (Paulk and Hurst, 1966; Parsommet et al, 1967), femoral veins (Sloman, 1968), and percutaneously into the subclavian vein (Mobin-Uddin et al, 1967). When an arm vein is used, the electrode position is unstable and repositioning is frequently required. Thus there was electrode displacement requiring remanipulation in 83% of the patients in the present series in whom an antecubital vein/
vein was used (Table 3:5). It is claimed that displacement of the electrode tip can be prevented if arm movement is restricted with an arm board and bandages or a special harness placed around the chest and enclosing the arm (Harris, 1969). However, this is a cumbersome arrangement, causes discomfort to the patient and impedes nursing. The external jugular route which provides electrode stability, requires a cut-down and is often time-consuming and technically difficult (Faulk and Hurst, 1966; Parsonnet et al., 1967). The percutaneous subclavian vein route, like the external jugular route, provides a stable electrode position, but is a much more rapid and simple technique. Its principle hazard is the production of a pneumothorax, but this is infrequent and any pneumothorax is usually small.

A stable and effective electrode position can be achieved reliably only by fluoroscopy. In most hospitals this requires the potentially hazardous transfer of the patient to and from a cardiac laboratory. Because of this danger, techniques have been evolved for the blind insertion of electrodes with intracardiac electrographic control using either USCI 5F electrodes or special float-in electrodes (Kimball and Killip, 1965; Siddons and Sowton, 1967; Dalle, 1968; Rosenberg et al., 1969). However, even in skilled hands, difficulty may be encountered in getting these electrodes to enter the right ventricle from the right atrium and failure to pace consistently, requiring repositioning, is relatively frequent (Kimball and Killip, 1965; Frei et al., 1966; Rosenberg et al., 1969).
By employing a portable image intensifier specially designed for use in coronary care units, these difficulties can be avoided and the electrode can be positioned rapidly and accurately at the patient's bedside (Samuel, 1968).

* * *

Ventricular tachycardia or fibrillation is a major risk associated with pacing in acute myocardial infarction and occurs in from five to 24% of cases (Paulk and Hurst, 1966; Parsons et al, 1967; Scott et al, 1967). Bradycardia itself appears to increase the risk of the development of ventricular arrhythmias. It has been postulated that pacing, by accelerating the heart rate, may reduce this risk (Han et al, 1966; Lown, 1968), but at present there is no conclusive clinical evidence that this is so. Moreover, ventricular tachyarrhythmias may be mechanically induced by electrode insertion as occurred in 4% of the present series and 27% of the series of Paulk and Hurst (1966). The patients in Paulk and Hurst's series (1966) had been receiving isoprenaline infusions prior to electrode insertion. There is evidence that isoprenaline alone can provoke ventricular fibrillation in CHB complicating acute myocardial infarction (Scott et al, 1967), and it is possible that it increases the risk of mechanically induced ventricular arrhythmias accounting for the very high incidence in the series of Paulk and Hurst (1966). Accordingly, it is probably advisable to avoid isoprenaline if possible if a pacing electrode is to be inserted.

Serious ventricular arrhythmias may also result from competition between the patient's spontaneous rhythm and that of the pacemaker/
pacemaker as occurred in five cases in the present series. Competition is particularly dangerous in acute myocardial infarction for two reasons. Firstly, the vulnerable period of the cardiac cycle during which a stimulus can provoke ventricular tachycardia or fibrillation is prolonged; and secondly, there is a considerable decrease in the energy required to produce these repetitive ventricular responses during the vulnerable period (Wolff et al, 1968). For this reason pulse generators, such as the Sanborn instrument, which deliver a high energy stimulus relative to the contraction threshold are particularly dangerous if competition arises.

With continuously discharging pacemakers, competition is liable to occur especially during the period when AV conduction is returning when episodes of varying degrees of block frequently alternate with normal conduction (Figure 3:1). The introduction of ventricular demand or inhibit instruments has largely, but not entirely, eliminated this danger (Sowton, 1967). Although a properly functioning demand pacemaker will not deliver a stimulus during the vulnerable period, the intracardiac QRS potential which is detected by the pacing electrode and which is the signal activating the inhibit mechanism, may be of inadequate amplitude following infarction (Chatterjee et al, 1968). This phenomenon of a low intracardiac potential occurred in four cases in the present series (Figure 3:2) producing ventricular fibrillation in one. In addition, demand units do not eliminate the danger of a ventricular ectopic beat occurring during the vulnerable period of a paced/
Competition associated with pacing in complete heart block complicating acute myocardial infarction. The upper tracing shows two mechanisms of production of ventricular tachyarrhythmias by competition. The first paced beat is followed by a normally conducted beat. The second pacing stimulus falls during the vulnerable period of this beat and produces a short run of ventricular tachycardia. Six paced beats then occur followed by a ventricular ectopic beat falling during the vulnerable period of the sixth paced beat and this initiates ventricular fibrillation which continues in the lower tracing.
Figure 3:2 Competition due to failure of the inhibit mechanism in a demand pacemaker. The arrows mark pacing stimulus artifacts. In the first three beats on the tracing, there is inhibition of the demand pacemaker and no stimulus artifact appears. However, following the third beat there is failure of inhibition and a pacing impulse is produced which falls on the T wave of the conducted beat initiating a run of three ventricular ectopic beats.
paced beat and this can also provoke ventricular arrhythmias (Figure 3:1). Despite these potential hazards, ventricular fibrillation due to competition is rare when demand pacemakers are employed and there seems little doubt that this type of instrument should always be used following acute myocardial infarction.

**Indications for Electrode Insertion**

In patients with CHB complicating acute infarction asystole or bradycardia requiring pacing may develop abruptly and death may ensue before an electrode can be introduced. This situation occurs most commonly in heart block associated with anterior infarction and a complete BBB pattern, but it may also occur in isolated inferior infarction with a normal QRS duration in which case treatment is usually more effective. It is advisable, therefore, to insert electrodes in all patients who have developed CHB since it is not possible to predict which of them will require treatment for these complications.

Furthermore, since bradycardia or asystole may occur at the very onset of CHB, it would be advantageous to be able to predict which patients were going to develop CHB and to insert electrodes prophylactically before it develops.

First degree AV block and sinus bradycardia are common disturbances following acute infarction and may proceed to CHB. However, they rarely progress to CHB without intervening second degree block (Julian et al, 1964; George and Greenwood, 1967). Thus/
Thus, sinus bradycardia did not proceed directly to CHB in the present series and CHB followed first degree block without intervening second degree block being detected in only two patients. Sinus bradycardia and first degree AV block are not, therefore, indications for prophylactic electrode insertion.

By contrast, in the present series approximately 50% of patients with second degree block and 30% of those with complete BBB and normal AV conduction progressed to CHB. However, although the insertion of pacing electrodes in patients with second degree AV block was accompanied by a low incidence of complications, this was not the case when complete BBB, sinus rhythm and normal AV conduction were present. Thus, the prophylactic introduction of pacing electrodes in 31 patients with complete BBB and normal AV conduction was associated with asystole or ventricular tachyarrhythmias requiring direct current shock in nine (Godman, M. J., Lassers, B. W. and Julian, D. G., unpublished observations).

Although only a small percentage of patients with partial AV block who proceed to CHB will actually require pacing, it is not possible to predict beforehand those that will. Since electrode insertion does not appear to be particularly hazardous in these patients, it is probably advisable to insert electrodes in all patients with second degree block. In contrast, however, although a large proportion of patients with complete BBB and normal AV conduction eventually require pacing, electrode insertion is extremely dangerous and pacing appears to be of little or no value in preventing/
preventing death. In view of this, routine prophylactic electrode insertion is not indicated in this group of patients.

**Indications for Pacing**

There is general agreement that asystole is an indication for pacing, although it may not always be effective under this circumstance (Epstein et al, 1966; Paulk and Hurst, 1966; Parsonnet et al, 1967). However, the role of pacing in the management of bradycardia is less easy to define. As has been pointed out in Section Two of this thesis, the value of improving the systemic circulation by pacing must be balanced against the increased work - and, therefore, the increased oxygen requirement - it demands of a damaged heart. This must be taken into account both in deciding which patients should be paced and the best rate for pacing.

As shown in Table 2:2, patients with normal mental function and good skin circulation usually have adequate cardiac outputs. Furthermore, their prognosis without pacing appears to be excellent. All 22 patients in the present series in whom electrodes were inserted but who were thought on clinical grounds not to require pacing, survived (Table 3:2). However, the presence of depressed mental function and poor skin circulation is almost invariably associated with severe reduction of the cardiac output and pacing at 80 to 90/min may produce a significant increase in cardiac output and substantial clinical improvement. These clinical features, therefore, provide a valuable guide in deciding which patients require pacing.

In/
In determining the rate at which to pace, it is reasonable to increase the cardiac output to the lower limits of normal when perfusion should be adequate and the increase in work insufficient to compromise the damaged heart. The cardiac output can not usually be measured at the bedside, but with pacing at increasing rates the blood pressure usually rises pari passu with cardiac output at least until the rate has exceeded 100/min. It may be assumed that the cardiac output has reached adequate levels when the systolic blood pressure has reached 100 mm Hg or more if mental function and skin circulation have also improved. On the other hand, the provocation of angina or dyspnoea is an obvious indication that the optimum pacing rate has been exceeded. In practice this rate can be determined by clinical trial - in most cases, however, it will prove to be between 80 to 90/min.

Duration of Heart Block and Removal of Electrodes

As was pointed out on page 18, heart block following acute myocardial infarction is a transient phenomenon in survivors, and normal AV conduction almost invariably returns within seven to 14 days. During the period of recovery, AV conduction is often unstable and electrodes should be left in situ since pacing may be required at any time. However, once normal AV conduction (or first degree block only) has been present for 72 hours, recurrence of CHB is rare. In the present series there was only one documented late recurrence. This happened eight days after the return of normal AV conduction; an electrode was reinserted and the patient survived. Sudden late/
Late deaths do occur and may be caused by recurrence of CHB, but the mode of death has seldom been documented in these cases. Therefore, the risk of removing electrodes after 72 hours has elapsed without heart block of greater than first degree is not great and the long-term monitoring of such patients is probably unjustified if there is a shortage of intensive care facilities (Oliver et al, 1967).
SECTION FOUR

FIRST-YEAR FOLLOW-UP STUDY
In the previous sections, it has been shown that acute myocardial infarction complicated by CHB is associated with a high mortality in the first weeks after the acute attack and that in the majority of cases it is the severity of the underlying myocardial damage rather than the heart block itself which determines the immediate prognosis. In this section, the mortality in the remainder of the first year after discharge from hospital is analysed; the degree of functional recovery and evidence of residual cardiac damage is described; and the work status of those patients who were alive at the end of the follow-up period is discussed. In view of the suggestion that the resuscitation of these patients may be of little value since their long-term prognosis may be poor (Shillingford, 1968; Sloman, 1968), the prognosis in those patients who required pacing during the acute attack is compared with the outlook for those patients in whom pacing was not needed.

**PATIENTS AND METHODS**

Of the first 51 of the 100 patients with CHB complicating acute myocardial infarction described in Section One, 27 survived to be discharged from hospital and form the basis of the follow-up study reported in this section. Twenty-six of the 27 patients had had electrodes inserted and pacing had been instituted in 12
(Table 4:1). Pacing was thought to have been beneficial or of equivocal benefit in 11 of the 12. In one patient it had to be discontinued because of angina and dyspnoea.

The patient's functional and work status in the year prior to infarction was evaluated and recorded at the time of the acute episode. Exercise tolerance was graded according to the New York Heart Association classification. The following clinical features of the acute attack were also recorded:

(1) The lowest recorded systolic blood pressure;
(2) The presence of pulmonary venous dilatation or oedema on antero-posterior chest radiographs taken in the coronary care unit;
(3) The occurrence of atrial fibrillation or ventricular tachycardia or fibrillation;
(4) The maximum serum creatine phosphokinase (SCPK) value; and
(5) The duration of advanced heart block.

The 27 patients were reviewed 12 months after the date of their original hospital admission. Twenty-two patients were still alive at this time and 19 of them were seen personally. For the remaining three patients, who had moved from the area, questionnaires were completed by the patient's general practitioner or by a consultant physician at another hospital. The patient's exercise tolerance and work status were re-evaluated and their drug treatment noted. In two survivors the cardiac rate and rhythm were determined/
<table>
<thead>
<tr>
<th>Pacing</th>
<th>Number of patients</th>
<th>Deaths</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Required</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Beneficial</td>
<td>7</td>
<td>1</td>
</tr>
<tr>
<td>Equivocal benefit</td>
<td>4</td>
<td>1</td>
</tr>
<tr>
<td>Not benefited</td>
<td>1*</td>
<td>1</td>
</tr>
<tr>
<td><strong>Not required</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Electrode not inserted</td>
<td>1</td>
<td>0</td>
</tr>
<tr>
<td>Inserted - not used</td>
<td>14</td>
<td>2</td>
</tr>
<tr>
<td><strong>Total</strong></td>
<td>27</td>
<td>5</td>
</tr>
</tbody>
</table>

* Pacing produced angina and dyspnoea

**Table 4:1** Relationship between first-year mortality and the use of pacing during an acute attack of myocardial infarction complicated by CHB.
determined clinically, but in the remaining 20, ECG's were recorded. Posteroanterior chest radiographs were taken and assessed independently in the 19 patients seen personally and were compared with postero-anterior radiographs taken at the time of discharge from hospital in 16 patients. All five patients who died had been readmitted to the Royal Infirmary, Edinburgh, prior to death. Detailed records were available for these patients and in three cases necropsies had been performed.

**RESULTS**

**Mortality and Factors Affecting Prognosis**

Of the 27 patients who survived to be discharged from hospital, five (18.5%) died during the remainder of the first year. Death was due either to a complication of the infarction or to reinfarction in four of these five (Table 4:2).

The only three patients with anterior infarcts who survived to be discharged, died during the remainder of the first year. In contrast, only two of the 24 patients with inferior or inferolateral infarction who were discharged subsequently died ($p = 0.003$) (Figure 4:1, Table 4:3).

At the time of discharge complete RBBB was present in five patients of whom two had anterior and three inferior infarcts. Both patients with anterior infarcts and one patient with an inferior infarct/
<table>
<thead>
<tr>
<th>Patient</th>
<th>Time of Death after Initial Admission</th>
<th>Cause of Death</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>9 months</td>
<td>LVF</td>
</tr>
<tr>
<td>2</td>
<td>4 months</td>
<td>LVF; Ruptured Papillary Muscle&lt;br&gt;<strong>&lt;br&gt;</strong>*</td>
</tr>
<tr>
<td>3</td>
<td>5 months</td>
<td>LVF; Re-infarction**&lt;br&gt;***</td>
</tr>
<tr>
<td>4</td>
<td>4 months</td>
<td>Re-infarction: sudden death**&lt;br&gt;***</td>
</tr>
<tr>
<td>5</td>
<td>12 months</td>
<td>CVA; Pulmonary embolus**&lt;br&gt;***</td>
</tr>
</tbody>
</table>

LVF: Left ventricular failure  
CVA: Cerebrovascular accident  
* Occurred in general medical ward and cause not determined  
** Necropsy evidence  
*** Table 4:2 Time and cause of death in patients surviving to be discharged from hospital after acute myocardial infarction complicated by complete heart block.
Figure 4:1  Relationship between one-year survival and site of infarction in 51 patients with CHB complicating acute myocardial infarction.
<table>
<thead>
<tr>
<th>Site of Infarct</th>
<th>CRBBB</th>
<th>Alive</th>
<th>Dead</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>Anterior</td>
<td>Present</td>
<td>0</td>
<td>3</td>
<td>3</td>
</tr>
<tr>
<td></td>
<td>Absent</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Infero-lateral</td>
<td>Present</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td></td>
<td>Absent</td>
<td>3</td>
<td>1</td>
<td>4</td>
</tr>
<tr>
<td>Inferior</td>
<td>Present</td>
<td>2</td>
<td>1</td>
<td>3</td>
</tr>
<tr>
<td></td>
<td>Absent</td>
<td>17</td>
<td>0</td>
<td>17</td>
</tr>
</tbody>
</table>

CRBBB: Complete right bundle branch block

Table 4:3  
Association between site of infarct, presence of complete right bundle branch block at time of discharge, and mortality in the remainder of the first year following discharge from hospital after acute myocardial infarction complicated by complete heart block.

Comparison of mortality in inferior v. infero-lateral plus anterior infarction:  
Exact $X^2$: $p = 0.003$.

Comparison of mortality in patients with and without bundle branch block:  
Exact $X^2$: $p = 0.03$.  

infarct subsequently died. In one of the two remaining patients who were alive at the end of the year, the BBB had resolved. Seventeen patients with isolated inferior infarction and without BBB were discharged from hospital and all were alive at the end of the year (Table 4:3).

The patients who died did not differ significantly from those who survived the remainder of the first year in age, previous history of infarction, angina, hypertension or diabetes, or in their exercise tolerance prior to admission. Similarly, the two groups did not differ significantly in the severity of their infarcts as assessed by the lowest recorded systolic blood pressure, the presence of radiographic evidence of pulmonary venous dilatation or oedema, the occurrence of other serious arrhythmias, the maximum SCPK value recorded, or the duration of advanced heart block (Table 4:4).

The subgroup of 12 patients who required pacing during CHB differed from the other hospital survivors in that they had had ventricular asystole, severe bradycardia, or bradycardia accompanied by signs of a low cardiac output (see page 54). In two cases the clinical picture of cardiogenic shock had been present. The subgroups did not differ significantly, however, in age, previous history and functional status, radiological evidence of left ventricular failure, the occurrence of ventricular tachycardia, ventricular fibrillation or atrial fibrillation, the maximum SCPK level, or the duration of advanced heart block. Furthermore, there was/
<table>
<thead>
<tr>
<th>Feature</th>
<th>Alive (22 patients)</th>
<th>Dead (5 patients)</th>
<th>P</th>
</tr>
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<tr>
<td>Age</td>
<td>60 ± 13 years*</td>
<td>58 ± 5 years</td>
<td>n.s.</td>
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<td>Lowest Systolic Blood Pressure</td>
<td>88 ± 13 mm Hg</td>
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<td>Maximum SCK Level</td>
<td>392 ± 321 units</td>
<td>447 ± 167 units</td>
<td>n.s.</td>
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<tr>
<td>Duration of advanced heart block</td>
<td>64 ± 59 hours</td>
<td>35 ± 11 hours</td>
<td>n.s.</td>
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<tr>
<td>X-ray Evidence of Pulmonary Venous Dilatation or Oedema</td>
<td>6 patients</td>
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<td>n.s.</td>
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<tr>
<td>Ventricular Tachycardia or Fibrillation</td>
<td>7 patients</td>
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<td>Atrial Fibrillation</td>
<td>7 patients</td>
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<td>n.s.</td>
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</tbody>
</table>

n.s.: not significant

* Mean ± 1SD

**Table 4.4** Comparison of clinical features during acute attack between patients who died during the remainder of the first year following discharge from hospital after acute myocardial infarction complicated by complete heart block and those who survived.
was no significant difference in mortality over the remainder of the first year between the two groups (Table 4:1). In both patients with cardiogenic shock, pacing had produced marked improvement in their clinical condition and both were alive at the end of the follow-up period. The patient in whom pacing had induced angina and dyspnoea died of a further infarct four months later.

Clinical Status of Survivors

Twenty-six of the 27 patients were in sinus rhythm with normal AV conduction at the time of discharge from hospital and one patient had atrial fibrillation. This latter patient died during the course of the year and one of the one-year survivors had developed atrial fibrillation. The remaining 21 one-year survivors were all in sinus rhythm and in none had there been clinical or ECG evidence of recurrence of AV block.

Thirteen of the 22 patients alive at the end of the follow-up period had been breathless on exertion during the year prior to the acute infarct, but only one patient had been receiving treatment with digitalis or diuretics. At the end of the follow-up period, 11 patients were receiving such treatment. With treatment, two patients were less breathless than they had been in the year preceding infarction, 14 were in the same functional grade, four had deteriorated one grade, one had deteriorated two grades and one patient, three grades.

Seven/
Seven of the 22 survivors had had angina in the year prior to the acute infarct. At the end of the follow-up period, two of these patients no longer complained of angina, angina remained unchanged in five and had developed for the first time in two other patients.

At the end of the year the cardiothoracic ratio was greater than 50% in ten of the 19 patients in whom radiographs were taken. In the 16 patients in whom postero-anterior films had been taken, both at the time of discharge from hospital and at the end of the follow-up period, nine showed no change in the cardiothoracic ratio, five showed a decrease of 5% or more, and two showed an increase of 10%. In the other three patients in whom radiographs were taken at follow-up but not at the time of discharge, the cardiothoracic ratio was within normal limits.

Of the nine one-year survivors who had been benefited or equivocally benefited by pacing, six were receiving diuretics and/or digoxin at the end of the follow-up period. Exercise tolerance was unchanged in six, and had deteriorated in three. The cardiothoracic ratio was unchanged in five, had decreased by more than 5% in two and had increased by 10% in one. Radiographs were not available in the ninth patient. Of the 13 one-year survivors who had not required pacing, five were receiving diuretics and/or digoxin at the end of the period. Exercise tolerance was unchanged in eight of these patients, had deteriorated in three and had improved with treatment in two. Radiographs taken both at discharge and/
and follow-up were available in eight of the 13 survivors who had not required pacing. The cardiothoracic ratio was unchanged in four, had decreased by more than 5% in three and had increased by 10% in one.

**Work Status of Survivors**

Fourteen of the 22 patients alive at the end of one year had been working prior to their infarction. At the end of the year three patients had retired, but only one because of symptoms of ischaemic heart disease. Eleven patients had returned to work after an average period of 7.4 months convalescence (Median period = 7 months). Seven of the 11 returned to their original job and four were doing less active work. Five of the nine survivors who had been paced had been employed prior to infarction and all had returned to work.

**DISCUSSION**

**Mortality**

The hospital mortality in the original 51 patients from whom the patients in the follow-up study were derived was high (4.7%). A further five patients representing 10% of the original 51 patients or 18.5% of the hospital survivors died during the remainder of the first year. There are many factors such as age, sex, socio-economic status, the presence of complicating diseases, the severity/
severity of the acute attack and the degree of recovery which profoundly affect the long term survival of patients who have had acute myocardial infarction and render the comparison of mortality figures among different series drawn from different populations difficult and often unreliable (McMichael and Parry, 1960; Lew, 1967).

However, the mortality in the remainder of the first year after a first or subsequent infarct in the studies reviewed by McMichael and Parry (1960) in which the mean age of the patients was similar to that of the present study, ranged from 14 to 20%, and mortality tended to be higher in so-called "poor risk" patients or patients with cardiac failure during the acute attack. Thus the mortality in the remainder of the first year following discharge from hospital after an infarct complicated by CHB does not appear to differ greatly from that which has been found in patients with infarcts of similar severity but without heart block. Furthermore, those patients in the present series who required artificial pacing during the acute attack and who survived to be discharged from hospital, fared no worse than those patients in whom CHB had not required pacing despite the fact that they appeared to be more severely ill during the acute attack. Anterior myocardial infarction, which was associated with a mortality of 87% in the acute attack, was also associated with a poor long term prognosis. In contrast, isolated inferior infarction, without BBB, which carried a good prognosis in the acute attack, was also associated with a good prognosis in the remainder of the first year.
Functional Recovery

Many of the patients who were alive at the end of the year had evidence of significant residual cardiac damage. Thus, 11 of the 22 survivors were receiving treatment with diuretics and/or digoxin. Despite treatment, six patients were more breathless on exertion than they had been prior to infarction and in nine patients the cardiothoracic ratio was greater than 50%. In none of the patients, however, had there been any evidence of recurrence of AV block. In spite of the high incidence of residual disability, only one patient who had been working prior to infarction had had to retire as a result of it, although four other patients were employed in less active jobs.
DISCUSSION

The Role of Artificial Pacing in the Management of Heart Block Complicating Acute Myocardial Infarction
In a number of recent, uncontrolled studies, the mortality observed in a small number of cases of CHB complicating acute myocardial infarction managed with artificial pacing has been compared with that found in other unpaced studies. On the basis of these comparisons, it has been claimed that pacing has resulted in a reduction in mortality and is superior to drug therapy in the management of heart block in acute infarction (Bruce et al, 1965; Paulk and Hurst, 1966; Scott et al, 1967). However, the clinical setting in which CHB following acute myocardial infarction may occur is so heterogeneous and there are so many variable factors such as intensity of monitoring, average time from onset of symptoms to admission, site of infarction, duration of QRS complex and severity of underlying myocardial damage which affect the comparability of patients, that a valid comparison between small samples can almost certainly not be made. Only a properly controlled clinical trial could adequately evaluate the impact of pacing on the mortality of acute myocardial infarction complicated by CHB or provide a sound comparison between pacing and drugs in its management. There has been no such trial and, indeed, a study of this kind would be extremely difficult to design and conduct.

In the absence of a controlled study, any assessment of the benefits of pacing must be based on clinical impressions which may often be incorrect. Thus, for example, the patient who has had repeated Stokes-Adams attacks abolished by pacing, or the patient who has had the clinical features of a low cardiac output reversed by/
by pacing, is usually regarded as having been benefited by this form of treatment with the implication that mortality has been reduced (Bruce et al, 1965; Beregovich et al, 1968). However, there is no doubt that patients with CHB following acute myocardial infarction may survive many syncopal attacks without treatment of any kind. Similarly, although a low cardiac output following acute infarction may not be desirable, it does not necessarily lead to death. For this reason, even a striking clinical improvement following an increase in heart rate by pacing, does not of necessity mean that pacing has prevented death. It is even more difficult to evaluate the benefits of pacing when it has been employed to correct marked bradycardia not associated with clinical deterioration or when a demand pacemaker has been activated intermittently. Although pacing may appear to be life-saving in some individual cases, there is no clear-cut evidence that is has significantly reduced the mortality in acute myocardial infarction complicated by CHB (Friedberg et al, 1968). Moreover, it is clear from the present study that in approximately 25 to 40% of patients (most of whom have inferior or inferolateral infarction with a normal QRS duration) artificial pacing is unnecessary because the disturbance of conduction does not lead to deterioration; and in a further 50% of patients (who usually have anterior infarction and/or a prolonged QRS complex) pacing is unsuccessful because death is due to severe underlying myocardial damage rather than to heart block itself.

On the other hand, pacing does provide an effective method of abolishing Stokes-Adams attacks and correcting a low cardiac output state/
state in 10 to 25% of patients, most of whom have inferior or inferolateral infarction. These patients who have been benefited by pacing have just as good a long-term prognosis as the patients with CHB who have not required pacing, despite the fact that they have appeared to be more severely ill during the acute attack. It would seem reasonable, therefore, to treat asystole and bradycardia, if the treatment itself does not worsen the prognosis. In evaluating the role of artificial pacing as a method of managing asystole and bradycardia, the effectiveness and limitations of other forms of treatment which have been used must also be considered.

A variety of drugs including atropine, corticosteroids, a solution containing a mixture of potassium, glucose and insulin, and the sympathomimetic agents, particularly isoprenaline, have been used in an attempt to restore AV conduction, abolish ventricular asystole and increase ventricular rate.

Atropine has been used in heart block following acute infarction to restore AV conduction (Master et al, 1938; Adgey et al, 1968; Kimball and Killip, 1968). The rationale behind this form of therapy is that myocardial ischaemia may lead to excessive vagal discharge which contributes to the block in AV conduction and that this may be reversed by atropine (Master et al, 1938; James, 1968). Atropine is undoubtedly effective in accelerating the atrial rate in sinus and nodal bradycardia (Julian et al, 1964; Adgey et al, 1968) and may reduce the PR interval in first degree AV block (Master et al, 1938). A reduction in the degree of second or third degree block immediately/
immediately after the intravenous injection of atropine has also been observed in some patients (Master et al, 1938; Jackson and Bashour, 1967; Adgey et al, 1968). On the other hand, an acceleration in atrial rate following atropine administration may actually increase AV block and the drug can, therefore, occasionally be dangerous (Master et al, 1938).

Corticosteroids have also been advocated as a means of restoring AV conduction. Following the intravenous administration of steroids to patients with heart block after acute infarction, Dall and Buchanan (1962) observed a reduction in the PR interval and the disappearance of block. This occurred within periods varying from less than two hours up to 26 hours and on this basis Dall claimed that steroids had been instrumental in restoring AV conduction. A number of workers have postulated that the anti-inflammatory action of steroids could reduce oedema and inflammation produced by ischaemia in the region surrounding the AV node and thereby improve conduction (Printzmetal and Kennamer, 1954; Dall, 1964); and Lowet al (1955) have demonstrated an acceleration of AV conduction by steroids in normal subjects and also in patients with both adrenal insufficiency and Cushing's syndrome. However, Sievers et al (1964) in a double blind clinical trial of oral corticosteroids, failed to show any significant reduction in the incidence of conduction disturbances.

Mitra (1966) has claimed that a solution of potassium, glucose and insulin reduces both the duration of CHB and the mortality associated/
associated with it. He postulated that this solution promotes the movement of potassium from the extracellular space to the intracellular space in the specialised cells of the AV conducting tissue and thereby restores the resting membrane potential. However, Malach (1967) has shown a higher incidence of increasing heart block in patients who received this solution than in a control group, and both Sievers et al (1966) and the Medical Research Council Working Party (1968) were unable to demonstrate any significant effect upon conduction in clinical trials of potassium, glucose, and insulin therapy.

All three methods of treatment - atropine, steroids and potassium, glucose and insulin therapy - attempt to manage heart block by restoring AV conduction. However, it is difficult to evaluate the efficacy of these drugs because of the rapid, spontaneous variations which frequently occur in heart block following acute myocardial infarction. The clinical trials which have been carried out have involved small samples of patients with CHB and have been uncontrolled or have used treatment and control groups which were either not randomly selected or not properly matched. For this reason the value of these drugs in the management of heart block in infarction is not clear. Furthermore, although it seems probable that both atropine and steroids can reduce the degree of block in some cases, they are of limited value in the management of CHB after acute infarction since they may take some time to alter AV conduction and are unpredictable in their effect in an individual patient. They are, therefore, of little use in the patient who requires treatment for asystole or severe bradycardia.
There is little doubt that the sympathomimetic amines—particularly isoprenaline—have been the most effective drugs in the treatment of heart block after infarction (Vogel and Bramnell, 1967; Zoll, 1967; Gregory and Grace, 1968). Isoprenaline may protect against repeated attacks of ventricular asystole by arousing or accelerating idioventricular pacemakers (Gale and Enfroy, 1959), and may in this way also correct a low cardiac output and hypotension when these are due to bradycardia. However, the drug must be given parenterally since oral and sublingual routes are not regularly effective (Gregory and Grace, 1968) and dosage may be difficult to control without producing serious ventricular arrhythmias (Beregovich et al, 1968; Friedberg et al, 1968). Moreover, ventricular asystole is not consistently prevented (Friedberg et al, 1968), and the drug is of little value once ventricular standstill has developed. A further limitation of isoprenaline is that the drug can not be used to prevent asystole in patients with conduction defects not associated with bradycardia.

Pacing has the advantage over drug therapy in CHB following acute myocardial infarction in that it is immediately effective in controlling the electrical activity of the heart at any given rate. In addition, it permits the use of digitalis and anti-arrhythmic drugs which depress AV conduction and are, therefore, otherwise contraindicated when heart block is already present (Friedberg et al, 1968; Gregory and Grace, 1968; Beregovich et al, 1969; Norris, 1969). However, because of the dangers associated with electrode/
electrode introduction and pacing - particularly ventricular tachyarrhythmias - specially trained staff and special facilities including continuous monitoring and intensive care should be available when this form of treatment is used. When such facilities are available, pacing is no more hazardous than management with isoprenaline and is probably superior to drug therapy in treating patients with asystole and patients in whom bradycardia is responsible for clinical deterioration.
CONCLUSIONS

(1) Artificial pacing is unnecessary in 25 to 40% of patients with complete heart block complicating acute myocardial infarction because the disturbance of conduction does not lead to deterioration. Most of these patients have inferior or inferolateral myocardial infarction with a QRS complex of less than 0.12 second duration.

In a further 50% of patients pacing is not successful because death is due to severe underlying myocardial damage rather than to heart block itself. The majority of these patients have anterior infarction and/or a QRS complex of greater than 0.12 second duration.

(2) In a minority, comprising between 10 to 25% of patients with complete block, most of whom have inferior or inferolateral infarction, pacing may lead to obvious clinical improvement by abolishing ventricular asystole or correcting the haemodynamic disturbances produced by bradycardia. This improvement is associated with a rise in cardiac output and systemic blood pressure.

Although pacing appears to be life-saving in some individual cases, there is no clear-cut evidence that it produces a significant reduction in mortality in acute myocardial infarction complicated by complete heart block.

(3) The long-term prognosis of patients who have been benefited by pacing is no worse than the outlook of patients with complete heart/
heart block in whom pacing has not been required - in spite of the fact that they appear to be more severely ill during the acute attack. Furthermore, the prognosis in patients who survive an acute infarction complicated by complete heart block is no worse than that of patients with infarcts of similar severity but without heart block.

(4) Since patients who develop ventricular asystole or a low cardiac output state associated with bradycardia have a good long-term prognosis if they survive the acute attack, it is reasonable to treat these complications despite the lack of objective evidence of a reduction in hospital mortality.

(5) Pacing provides an effective method of managing asystole and bradycardia and is probably superior to drug therapy when trained staff and special facilities including continuous monitoring and intensive care are available.

(6) 50\% of patients with second degree heart block progress to complete heart block. Almost all of these patients have inferior or inferolateral infarctions and pacing will, therefore, be of benefit in some. Since it is not possible to predict which patients will require pacing, or when they will require it, pacing electrodes should be inserted in all patients with second degree or complete heart block.

30\%
30% of patients with complete bundle branch block in acute infarction progress to complete heart block and most subsequently develop asystole or bradycardia with a low cardiac output state. However, the prophylactic introduction of pacing electrodes is not indicated in this group of patients because pacing does not reduce the mortality which is due to severe underlying myocardial damage, and because electrode insertion is associated with a high incidence of serious arrhythmias.
The work described in this Thesis has been reported in part in the following publications:


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Hammer/


Katz/


Lown/


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Schwartz/


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of

ters on

Complete Heart Block

Complicating Acute Myocardial Infarction
97

A. Bibliography covering case reports and studies on complete heart block complicating acute myocardial infarction in which continuous electrocardiographic monitoring was not employed in the detection of cases.


Kerr, J.D.O/


B. Bibliography of principal clinical studies of acute myocardial infarction in which electrocardiographic monitoring was employed and complete heart block considered.


Scott/


C. Bibliography of principal studies on artificial pacing in the management of heart block complicating acute myocardial infarction.


D. Recent review articles on complete heart block complicating acute myocardial infarction and its management.


APPENDIX
<table>
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<th>Coronary Arteries</th>
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Legend: A = Anterior; I = Inferior; IL = Inferolateral; A + I = Anterior + inferior; LBBB = Left bundle branch block; B = Bilateral BBB pattern; R = Right BBB pattern; L = Left BBB pattern; P = QRS duration not known; >0.12 = Prolonged but not known if L or R BBB pattern; 0 = occluded; SS = Severe stenosis; SS0 = Severe stenosis and old occlusion; CS = Congenitally small; - = Not involved; + = Recent infarction; +0 = Old infarction.

Table A1: Necropsy findings.
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<th>Mean RA BP</th>
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Legend: Day of HB = Day after onset of CHB; PA = Pulmonary arterial; RA = Right atrial; SVR = Systemic vascular resistance (dyne sec. cm⁻⁵/m²); TTI = Tension time index (mm Hg sec/min)

Table A:2 Haemodynamic effects of increasing ventricular rate by pacing.
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Legend: See Table A:2

Table A3: Haemodynamic effects of increasing ventricular rate in patient J.D.
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Legend: See Table A:2

**Table A:4** Haemodynamic effects of increasing ventricular rate by pacing in patient J.M. with CHB and cardiogenic shock.
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<th>PA Diastolic BP $\text{mm Hg}$</th>
<th>RA Mean BP $\text{mm Hg}$</th>
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| P       | <0.001    | <0.005  | <0.001        | N.S.                            | N.S.                      | N.S.                        | <0.001                     |

Legend: HB = Heart block; Max. Op. Rate = Maximal output rate; M.O.R. = Maximal output rate; ↓ = clinically not elevated; ↑ = clinically elevated

Figures in brackets not included in mean values as complete measurements not made.

Table A:5.
Haemodynamic measurements made in heart block and with pacing at the rate associated with the maximal cardiac index.
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<th>Time</th>
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<th>Rate</th>
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<th>SVR</th>
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Legend: See Table A:2

**Table A:5.** Haemodynamic effects of pacing at 83/min for 60 minutes in patient R.C.
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*Day after onset of CHB

**Drugs with a possible haemodynamic effect.

Table A:7- Subsequent Days.

Haemodynamic measurements on first and subsequent days of heart block and after return of normal AV conduction.