Heart Block Complicating Acute Inferior Wall Myocardial Infarction*

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Heart block was noted in 60 (35 complete and 25 second-degree) of 410 patients with acute inferior wall myocardial infarction. This group with heart block was compared to a control group of 30 patients with acute inferior wall infarction without heart block. The incidences of prior myocardial infarction and hypertension, in addition to the highest level of serum creatine phosphokinase and a maximum degree of ST-segment elevation in the inferior leads, were all greater in patients with heart block, as compared to the controls. The incidences of various complications, including dizziness and syncope, transient hypotension, cardiogenic shock, and congestive heart failure, were also higher in the group with heart block, while sinus nodal disturbances and atrial arrhythmias occurred with equal frequency. The mortality in those with heart block was 28 percent compared to 13 percent for the control. It is concluded that patients with heart block complicating acute inferior myocardial infarction have a greater amount of myocardial necrosis, a higher incidence of complications, and a higher mortality. Insertion of a temporary pacemaker should be considered when specific indications are present and not routinely.

Studies concerning heart block complicating acute myocardial infarction have emphasized the appearance of bundle-branch block and the higher mortality associated with anterior wall infarction, and the more benign prognosis of atrioventricular block associated with inferior wall myocardial infarction. The indications for insertion of a temporary pacemaker in patients with heart block complicating acute inferior wall myocardial infarction remain unsettled.

This study reports our experience with 60 patients with heart block complicating acute inferior wall myocardial infarction. Significant clinical features, serum creatine phosphokinase levels, ST-segment elevations, and various complications are compared to a group of control patients with inferior myocardial infarction without heart block. An attempt is made to clarify the indications and value of temporary-pacemaker insertion.

Patients and Methods

Heart block was noted in 60 (35 complete and 25 second-degree) of the 410 patients with acute inferior-wall myocardial infarction seen in our coronary care unit during the period of 1970 to 1974. Most patients were monitored in the coronary care unit for four to six days, except those who required longer periods because of various complications. Daily 12-lead electrocardiograms and determinations of serum creatine phosphokinase levels were performed during this period. In addition to constant electrocardiographic monitoring and hourly rhythm strips, repeat ECGs were obtained whenever changes in atrioventricular conduction were noted. Patients in whom heart block appeared as a terminal event or transiently during cardiac resuscitation were not included in this study.

A history of hypertension was recorded if the patient had previously had more than one blood-pressure determination greater than 160/90 mm Hg or if the patient was receiving antihypertensive medication. Diabetes mellitus was considered to be present if a fasting blood glucose level greater than 120 mg/100 ml had been recorded prior to hospitalization or if the patient was receiving antidiabetic medication. Previous myocardial infarction was diagnosed by the history of typical precordial pain and the presence of significant Q waves on the ECG. Serial 12-lead ECGs were reviewed for the maximum degree of ST-segment elevation in the inferior leads. In addition, rhythm strips were reviewed to determine the time of onset and the disappearance of heart block and to determine the presence of sinus nodal abnormalities and atrial arrhythmias. Maximum serum creatine phosphokinase values were recorded as multiples of the upper limit of normal for our laboratory.

The presence of cardiogenic shock was recorded if the systolic blood pressure was persistently lower than 80 mm Hg and was accompanied by the clinical features of cool moist skin, mental confusion, and a fall in urinary output. A transient fall in blood pressure not accompanied by these clinical features and usually associated with bradyarrhythmias was classified as transient hypotension. Congestive heart failure was considered to be present if bilateral basal rales were heard which were not related to a history of chronic pulmonary disease and were accompanied by radiographic.

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FIGURE 1. Diabetes, hypertension, and old myocardial infarction (MI) in patients with heart block (HB) and controls. Numbers in bars are numbers of patients.

evidence of increasing pulmonary vascular congestion.

Similar historic and clinical data were obtained on a control group of 30 patients with acute inferior wall myocardial infarction who did not show evidence of heart block.

RESULTS

The incidence of heart block (second-degree and third-degree) in patients with acute inferior wall myocardial infarction was 15 percent. The average age of the patients with heart block (61 years) was similar to that of the control group (62 years), as was the male/female ratio (2:1). The incidences of previous myocardial infarction, hypertension, and diabetes mellitus in the patients with heart block and the control group are shown in Figure 1. The maximum ST-segment elevation measured in millimeters in the inferior leads and the maximum serum creatine phosphokinase level expressed in multiples of the upper limit of normal are shown in Figure 2.

Time of Onset and Duration of Heart Block

Heart block was present on admission or developed within 24 hours in 35 patients (early heart block) and appeared late (two to seven days after admission) in 25 patients (late heart block). Of the group with early heart block, the block was noted at the time of admission in 26 patients and appeared within 24 hours in nine patients. There were no deaths in ten of the patients with early heart block in whom the defect was transient (a few minutes to one hour), while there were ten deaths in the remaining 25 patients with early heart block patients in whom the block lasted from two hours to 14 days. In the 25 patients with late block, the block lasted from two hours to 16 days, and seven deaths occurred in this group. The duration of block varied and was noted to be intermittent and unstable in 11 patients and present for more than six days in nine others. In four patients in whom the block was present transiently on admission, it reappeared on the third and fourth days of hospitalization.

The incidences of various complications for those with heart block and the controls are shown in Figure 3. The greater incidences of transient hypotension and cardiogenic shock in the group with heart block were statistically significant ($P < 0.05$). The mortality of 28 percent (17/60) in the patients with heart block was higher than the 13 percent (4/30) noted in the controls and was not affected by the degree of heart block. The 56-percent incidence (34/60) atrial arrhythmias and sinus nodal abnormalities was similar to the 53-percent incidence (16/30) noted in the control group.

Rate and Morphologic Appearance of Escape Rhythm

The average rate of the escape rhythm in those patients with complete heart block was 45 beats per minute, with a range of 24 to 70 beats per minute. There was no relationship between the rate of this escape pacemaker and the morphologic appearance of the escape beats, the time of occurrence of heart block, the associated complications, or the mortality.

The escape beats were wide in seven patients (Table 1). In three of these patients, bundle-branch block was not present in the conducted beats, and the morphologic appearance of the escape beats was consistent with right bundle-branch block and left anterior hemiblock, suggesting an escape focus in the posterior division of the left bundle branch (Fig 4). A His-bundle electrogram recorded in one of these patients showed a markedly prolonged atrio-His (A-H) interval, in addition to block distal to the
His-bundle potential (Fig 5). One of these patients died, and two survived. The four remaining patients with wide escape beats all had evidence of bundle-branch block during periods of normal conduction. In one patient, who ultimately survived, a pattern of right bundle-branch block was present during normal conduction and in the escape beats. In the remaining three patients, the morphologic appearance of the escape complex was different from that of the normally conducted beats, and all three died, despite pacemaker insertion. An additional patient developed trifascicular block manifested by periods of right bundle-branch block and left posterior hemiblock and of left bundle-branch block. Ventricular asystole occurred, requiring insertion of a temporary pacemaker. A permanent pacemaker was inserted at four weeks after hospitalization, but the patient died suddenly of a possible arrhythmia at seven weeks.

### Table 1—Characteristics of Patients Showing Wide Escape Beats*

<table>
<thead>
<tr>
<th>Patient</th>
<th>Sinus- Conducted QRS</th>
<th>Escape QRS</th>
<th>Outcome</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Normal</td>
<td>RBBB and LAH</td>
<td>Survived (shock)</td>
</tr>
<tr>
<td>2</td>
<td>Normal</td>
<td>RBBB and LAH</td>
<td>Died (shock)</td>
</tr>
<tr>
<td>3</td>
<td>Normal</td>
<td>RBBB and LAH</td>
<td>Survived</td>
</tr>
<tr>
<td>4</td>
<td>RBBB</td>
<td>RBBB</td>
<td>Survived</td>
</tr>
<tr>
<td>5</td>
<td>RBBB</td>
<td>LBBB</td>
<td>Died (CHF)</td>
</tr>
<tr>
<td>6</td>
<td>RBBB and LAH</td>
<td>RBBB and LPH; RBBB and LAH; Died (shock)</td>
<td></td>
</tr>
<tr>
<td>7</td>
<td>RBBB and LAH; Wide LPH</td>
<td>Died (shock)</td>
<td></td>
</tr>
</tbody>
</table>

*RBBB, Right bundle-branch block; LAH, left anterior hemiblock; LBBB, left bundle-branch block; LPH, left posterior hemiblock; and CHF, congestive heart failure.

**Mode of Death**

Seventeen patients in the group with heart block died. The cause of death was cardiogenic shock in 11 patients; death occurred within the first 24 hours in seven of these and occurred between the third and fourth day in the other four.

**Figure 3.** Incidences of various complications in patients with heart block (HB) and controls. Numbers in bars are numbers of patients. CHF, congestive heart failure.

**Figure 4.** Selected leads from ECGs (case 1, Table 1). A. At time of admission. Note sinus rhythm with changes of acute inferior-wall myocardial infarction. B. Recorded soon after admission. Note complete heart block with QRS morphologic appearance suggestive of right bundle-branch block and left anterior hemiblock.
If I A-H = 190-220 msec

**Figure 5.** His-bundle electrograms (HBE) (case 1, Table 1). Complete heart block is present, and block occurs distal to His-bundle potential. Atrio-His interval (A-H) is prolonged and ranges from 190 to 220 msec.

the seventh days of hospitalization in the remaining four. All of these 11 patients had heart block, and six had temporary pacemakers at the time of their death. In the remaining five, there was not adequate time for pacemaker insertion. Cardiac rupture was the cause of death in one additional patient, who died on the eighth day of hospitalization. In the remaining five patients, death occurred from other causes between the fifth and 35th days of hospitalization. In four of these patients, heart block was not present when the patients were discharged from the coronary care unit, but in the three instances in which death was sudden, it is not known whether the mechanism was recurrent block or ventricular fibrillation.

**Indications for Pacemaker Insertion**

Temporary transvenous pacemakers were inserted in 31 patients (eight with second-degree and 23 with complete heart block). The various indications for pacemaker insertion were as follows: atropine-resistant symptomatic bradyarrhythmia, 19 patients; shock, 11 patients; hypotension, eight patients; congestive heart failure, 17 patients; prolonged asystole, two patients; control of atrial and ventricular arrhythmia, five patients; and wide escape beats, seven patients. In many patients, two or more of these indications existed simultaneously. Ventricular fibrillation occurred in three patients during pacemaker insertion, and in each case, successful defibrillation was achieved.

**Discussion**

The reported incidence of heart block complicating acute myocardial infarction ranges from 1.5 percent to 18 percent.\(^5,9,11,15\) This variation is due in part to inclusion of both anterior and inferior wall infarctions in some of the reports and the lack of constant monitoring in earlier series. The incidence of 15 percent in our series is less than the incidences of 26 percent and 27 percent reported by others.\(^5,11\) Our finding of a higher incidence of prior myocardial infarction in the group with heart block, as compared to the controls, suggests more extensive coronary arterial disease and is similar to other reported results.\(^4,5,9\)

The magnitude of ST-segment elevation and the levels of serum creatine phosphokinase have been correlated with the extent of myocardial necrosis.\(^23,24\) The elevation of both of these parameters in our group of patients with heart block is in agreement with the concept that patients with heart block complicating acute inferior-wall myocardial infarction have a greater amount of myocardial damage. This may also explain the higher incidences of congestive heart failure and shock and the higher mortality in patients with heart block.

**Onset and Duration of Heart Block**

The time of onset and the duration of block in acute inferior wall myocardial infarction have no uniform pattern.\(^5,9,11,12,20\) We observed block within the first 24 hours following admission in 58 percent (35/60) of our cases, which is similar to the experience of others.\(^5,9,12,20\) The duration of heart block varied markedly in our series and lasted from a few minutes to 16 days. Persistence of heart block to this degree was noted by Norris\(^11\) and by Kostuk and Beanlands,\(^12\) but not by Simon et al.\(^20\)

It appears that different mechanisms may be in operation, producing heart block with different times of onset and different periods of duration. Heart block occurring early and transiently during periods of chest pain might be related to inappropriate vagal tone; or transient ischemia of the atrioventricular node might occur, as is seen in patients...
with Prinzmetal's angina and heart block. In those patients with heart block appearing later and lasting for longer periods of time, edema and inflammation of the atrioventricular node is likely. Massive necrosis of the atrioventricular node has been demonstrated in patients with block who died of cardiogenic shock. This concept of different mechanisms may explain the reappearance of block on the third and fourth days of hospitalization in four of our patients who showed transient block at the time of admission. Kostuk and Beanlands found, as we did, that patients with early and transient block had a more benign prognosis.

Significance of Wide Escape Beats

The site of block in acute inferior wall myocardial infarction has been localized to the region of the atrioventricular node or within the His bundle. Although the majority of these patients show narrow escape beats, a small percentage does show an escape rhythm with a wide QRS complex. In the study by Lie et al., 12 out of 35 patients admitted with heart block complicating acute inferior-wall infarction showed a wide QRS escape complex. Kostuk and Beanlands noted four patients with a wide QRS escape rhythm and observed that their mortality was similar to those with narrow escape beats. Earlier studies by Lassers and Julian and by Friedberg et al. found a higher mortality in those patients with wide escape beats. We believe that the mechanisms producing a wide QRS complex relate to mortality rather than the QRS width alone. Prognosis in patients with prior bundle-branch block who develop block at the atrioventricular node is similar to those with narrow escape beats; however, if the escape beats are wide as a result of acute trifascicular block, the mortality increases significantly and resembles those with anterior myocardial infarction and trifascicular block. Those patients who do not have bundle-branch block in sinus beats but show wide escape beats during heart block are most likely to have block in the area of the His bundle and show a variable prognosis.

The benefit of routine pacemaker therapy for patients with heart block complicating inferior wall infarction has not been established. There does not appear to be a direct relationship between a policy of pacemaker insertion and mortality. The death rates of 40 percent and 45 percent were noted by groups who implanted pacemakers in all patients with block, while mortalities of 37 percent, 39 percent, and 44 percent were reported by groups who did not use prophylactic pacemakers. Further examples of this discrepancy are the exceptionally low mortality of 20 percent without pacing described by Jackson and Bashour, while Rotman et al. reported a 30 percent mortality despite pacing. In the series of Friedberg et al., three deaths were related to Stokes-Adams attacks, and it was thought that pacemaker therapy might have been useful. Other investigators have reported beneficial effects of pacing in patients who had experienced syncope.

It is our policy to insert a temporary pacemaker in a patient with heart block only if symptoms are present. These symptoms include dizziness or syncope, hypotension, cardiogenic shock, and congestive heart failure. In addition, we insert a pacemaker if significant ventricular arrhythmias requiring overdrive are present, or if the patient is completely asymptomatic and there is complete heart block with a wide QRS escape complex.

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Modern Wood Engraving

Apart from lithography and etching, the post-war period saw the rebirth of wood engraving, especially in Switzerland. In 1953, an international society of wood engravers was founded in Zurich, under the name of Xylon. This group looks back to fifteenth-century engraving, especially colored wood engraving, for its members do not want just “grim black and white.” In the introduction of the catalogue at the exhibition at Vienna in October 1961, they pointed out that wood engraving should draw its inspiration from the Gargas cave in the Pyrenees, “where you can see imprints of mutilated hands done with a primitive paint made of iron oxide and animal fat.” In spite of their manifesto, several artists hesitated between nineteenth-century tradition-alism and the influence of Gauguin, but there are among them marked personalities like HR Bosshard (born in 1929 in Zurich), Werner Hoffmann (born in 1935 in Affoltern), and Heinz Keller (born in 1928 in Wintertthur). Nearly all of them are Expressionists, for wood does not lend itself well to Abstract art, and moreover, they say their forebears of the fifteenth century had such power of expression that they were the ancestors of modern Expressionism.


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