Advanced Atrioventricular Block in Acute Myocardial Infarction

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COMPLETE atrioventricular block that develops during acute myocardial infarction is a significant and disconcerting complication. A knowledge of prognosis for recovery from the block and for life is necessary for the better management of a patient in whom this complication occurs. This is an uncommon occurrence. Previous reports suggest that a severe degree of atrioventricular (AV) block appears in 1 to 4.7 per cent of patients with acute myocardial infarction. Reported mortality rates range from 40 to 100 per cent. Suggested therapy for complete heart block during acute myocardial infarction includes the use of corticosteroids and isoproterenol. In addition, external or internal cardiac pacemakers might be required if the block were permanent. This study was made in order to investigate three aspects of advanced atrioventricular block and acute myocardial infarction. These are the site of the infarction, the duration of the block, and the effect of this complication upon prognosis.

Material and Method of Study

This study includes all patients admitted to the Cincinnati Veterans Hospital during a 30-month period in whom advanced atrioventricular block developed during acute myocardial infarction. These were nine in number. During this 30-month period, 94 patients with electrocardiographic evidence of acute myocardial infarction were admitted to the Cincinnati Veterans Hospital; of these, 35 were classified as inferior or diaphragmatic infarctions. Thus, advanced atrioventricular block was found in 9.6 per cent of all acute infarctions in this group. Further, advanced atrioventricular block appeared in the surprisingly high incidence of 26 per cent of all acute inferior infarctions in the Veterans Hospital group. In addition, nine patients observed elsewhere by the authors, or brought to their attention during the same period, are included. This study then consists of 18 patients: 15 patients who developed complete atrioventricular block and three patients who developed second-degree atrioventricular block during acute myocardial infarction. Electrocardiograms were obtained frequently during the acute conduction disturbance and then periodically until hospital discharge in those who survived. Autopsies were obtained in 6 of 10 patients who died.

Results

The pertinent data of all patients are presented in table 1. This includes 15 patients with complete heart block and three patients with 2:1 AV block.

Sex. Sixteen patients were men. The two women had complete heart block.

Age. Ages were from 38 years to 82 years. The youngest patient with complete heart block was 42 years.

Previous Myocardial Infarction. Three patients had had previous anterior myocardial infarctions, 2 months, 3 years, and 6 years before.

Medication. Two patients were receiving maintenance dosages of digitalis. There were no indications of digitalis intoxication. None had received quinidine.

Onset of Block. Complete atrioventricular block or second-degree block was present on admission or appeared by the fourth hospital day in all patients. Atrioventricular block was present or appeared in the first day of the infarction in 11 patients, on the second day in one patient, on the third day in four patients, and on the fourth day in one patient. Its day of onset was unknown in one patient. The block was discovered when he

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was admitted to the hospital on the third day of his illness.

**Duration of Block.** Complete heart block lasted from 1 hour to 6 days. Four patients died with AV block still present. Three patients died with complete AV block: one 2 hours after admission; one 21 hours after the onset of pain, and one after 3 days of illness. One patient with 2:1 block died 18 hours after admission. In one patient the block was intermittent. No more than first-degree AV block was documented after the seventh day of illness in any patient.

**Location of Infarction.** Each patient showed electrocardiographic changes of acute inferior myocardial infarction in leads II, III, and aVF (figs. 1 and 2). In no case did the duration of the QRS complex exceed 0.10 second.

**Figure 1**

Serial changes in lead II of electrocardiograms of patient R.R. On May 8, 1959, the first day of infarction, there is sinus rhythm with early changes of inferior myocardial infarction. There is a normal P-R interval of 0.17 second. On May 10, 1959, there is a second-degree atrioventricular block with the Wenckebach phenomenon. On May 12, 1959, complete atrioventricular block has developed with no change in the width or configuration of the QRS complex. On May 22, 1959, there is first-degree atrioventricular block, with a P-R interval of 0.24 second. There is deeper inversion of the T wave.
and the pattern of bundle-branch block or of an idioventricular rhythm was seen in only one patient (R.A.). She had the pattern of right bundle-branch block both during complete atrioventricular block and during normal atrioventricular conduction as well. The ventricular pacemaker in all cases of complete block appeared to be either in the AV node or ventricular septum.

No patient at the time of hospital discharge was left with a disturbance of atrioventricular conduction. One patient had atrial fibrillation of unknown duration; this persisted until his sudden death on the thirty-fourth day. Complete heart block had lasted 6 days. First-degree heart block (P-R interval = 0.24 second) persisted for 24 days in one patient.

Syncope. Stokes-Adams seizures were not common in these patients. Only one had such an attack after hospitalization and the seizure was attributed to ventricular standstill. This patient died 18 hours after admission. Two other patients were said to have had a period of loss of consciousness prior to admission. Two patients tolerated ventricular rates of 30 and 37 per minute without untoward symptoms.

Outcome. There were seven deaths during hospitalization, giving an immediate mortality of 39 per cent. Four deaths occurred while atrioventricular block was present at 2 hours, 18 hours, 21 hours, and 2 days after admission. Three deaths occurred, although complete heart block had subsided, after 11 days, 18 days, and 34 days.

Ten patients were discharged from the hospital for convalescence. Of these, two died of recurrent myocardial infarction 4 months, and 30 months later. A third patient died of terminal bronchogenic carcinoma 6 months after discharge. Nine of the 18 patients were critically ill and in shock at the time of admission. Three of these eventually recovered. Of the patients in shock, one died after 2 hours, one after 18 hours, one after 21 hours, and one after 2 days, with atrioventricular block persisting to death. Two patients recovered from shock, and the atrioventricular block subsided. Nevertheless, these two patients died after 11 days, and 34 days. One patient has been observed only 5 weeks in the hospital, and is recovering satisfactorily.

Autopsy. Autopsies of six patients were performed (table 1). In three autopsies of patients who died during the acute stage of their illness, a recent thrombus in the proximal portion of the right coronary artery was present in each, and there was fresh infarction of the posterior wall of the left ventricle and interventricular septum, which in one patient extended to the AV node. In one patient, (R.A.), who died 21 hours after symptoms began, there was marked sclerotic narrowing of the main right coronary artery 2 cm. from its ostium. There was no gross evidence of myocardial infarction. Unfortunately, histologic sections of the myocardium were technically unsatisfactory. In two cases in which autopsy was performed, 34 days and 2 years respectively, after the occurrence of infarction associated with heart block, old occlusions of the left circumflex arteries were found.

Discussion

Anatomy. An advanced degree of heart block occurs most frequently when the infarction is of the inferior wall and results from interference to the blood supply of the AV node or upper part of the bundle of His. The blood supply to this region is derived as a rule from the right coronary artery, and it is occlusion of this artery that is responsible for 90 per cent of inferior myocardal infarctions. In the remaining 10 per cent of cases, it is occlusion of the left circumflex artery that is responsible.

The artery to the AV node (sometimes more

Figure 2

Serial changes in leads II and III from electrocardiograms of patient L.W. On May 9, 1962, the first day of infarction, complete atrioventricular block is present. There is no increased width of the QRS complex. On May 10, 1962, 2:1 atrioventricular block has developed and on May 11, 1962, atrioventricular conduction is normal. The T wave has become negative in both leads.
### Table 1

**Advanced Atrioventricular Block in Acute Myocardial Infarction**

<table>
<thead>
<tr>
<th>Patient</th>
<th>Age</th>
<th>Sex</th>
<th>Day of onset of atrioventricular block</th>
<th>Duration of atrioventricular block</th>
<th>Location of infarct on ECG</th>
<th>Ventricular rate during atrioventricular block</th>
<th>Outcome</th>
<th>Autopsy</th>
</tr>
</thead>
<tbody>
<tr>
<td>AJ</td>
<td>52</td>
<td>M</td>
<td>1st</td>
<td>1 da.</td>
<td>Inf.</td>
<td>65</td>
<td>Uneventful recovery</td>
<td>—</td>
</tr>
<tr>
<td>WJ</td>
<td>60</td>
<td>M</td>
<td>1st</td>
<td>1 hr.</td>
<td>Inf.</td>
<td>40</td>
<td>Recurrent chest pain and sudden death 11th day</td>
<td>No</td>
</tr>
<tr>
<td>GS</td>
<td>61</td>
<td>M</td>
<td>1st</td>
<td>2 hr. (died with AV block)</td>
<td>Inf.</td>
<td>40</td>
<td>Died 2 hr. after admission</td>
<td>Coronary arteries moderately atherosclerotic. Rt. coronary occluded by thrombus 4 cm. from its origin. Red and yellow mottling of posterior wall of left ventricle</td>
</tr>
<tr>
<td>WG</td>
<td>58</td>
<td>M</td>
<td>3rd</td>
<td>3 da. or less</td>
<td>Inf.</td>
<td>60</td>
<td>Uneventful recovery</td>
<td>—</td>
</tr>
<tr>
<td>RR</td>
<td>48</td>
<td>M</td>
<td>3rd</td>
<td>5 da. or less</td>
<td>Inf.</td>
<td>50</td>
<td>Uneventful recovery. Died 2 yr. later with true post. infarct</td>
<td>Old and new occlusion of left circumflex artery. No occlusion of rt. coronary. Thinning and scarring of apical region</td>
</tr>
<tr>
<td>AC</td>
<td>64</td>
<td>M</td>
<td>1st</td>
<td>3 da. (died with AV block)</td>
<td>Inf.</td>
<td>54</td>
<td>Died on 3rd day</td>
<td>No</td>
</tr>
<tr>
<td>JW</td>
<td>38</td>
<td>M</td>
<td>1st</td>
<td>18 hr. (died with 2:1 AV block)</td>
<td>Inf.</td>
<td>70</td>
<td>Died after 18 hr.</td>
<td>Recent occlusion of rt. coronary artery. Old occlusion of left circumflex and anterior left descending coronary artery. Acute infarct of posterior and lateral wall of left ventricle and ventricular septum</td>
</tr>
<tr>
<td>EB</td>
<td>82</td>
<td>M</td>
<td>2nd</td>
<td>5 da.</td>
<td>Inf.</td>
<td>30-50</td>
<td>Doing well until sudden death on 18th day</td>
<td>Thrombus in rt. coronary 2 cm. from its origin. Infarction of posterior aspect of left ventricular wall and ventricular septum</td>
</tr>
<tr>
<td>LW</td>
<td>57</td>
<td>M</td>
<td>1st</td>
<td>2 da.</td>
<td>Inf.</td>
<td>45-52</td>
<td>Uneventful recovery</td>
<td>—</td>
</tr>
<tr>
<td>TP</td>
<td>71</td>
<td>M</td>
<td>1st</td>
<td>1 da.</td>
<td>Inf.</td>
<td>43</td>
<td>Uneventful recovery</td>
<td>—</td>
</tr>
<tr>
<td>CL</td>
<td>42</td>
<td>M</td>
<td>3rd</td>
<td>4 da.</td>
<td>Inf.</td>
<td>44-50</td>
<td>Uneventful recovery but died 4 mo. later with acute anterior infarct</td>
<td>No</td>
</tr>
</tbody>
</table>
Recent thrombus in anterior descending and left circumflex arteries. Extensive healed infarction of left ventricular wall and ventricular septum.


Gross, is given off at the posterior junction of the interatrial and interventricular septa of the crux of the heart. The AV nodal artery originates from the artery that crosses the coronary sinus and rises deep to the base of the heart, whether it be the right coronary artery or left circumflex coronary artery.

James and associates, 27 in their studies of the arterial coronary arteries, demonstrated that the AV nodal artery arises from the right coronary artery in 88 per cent of persons, from the left circumflex coronary artery in 7 per cent, and from both left and right coronary arteries in 5 per cent. The AV nodal artery passes anteriorly from its origin, travels deep to the base of the coronary sinus and rises deep to the base of the heart. It is the greater of the two, named the ramus septi fibrosi by Gross.
sumably the anastomotic vessels were adequate to maintain the blood supply to this area.

**Prognosis for Life.** First-degree heart block which develops during acute myocardial infarction is not a threat to life. Master reported⁹ that simple prolongation of the P-R interval exerted no adverse effect on the clinical course or prognosis of acute myocardial infarction.

More advanced degrees of AV block carry a graver prognosis. In the present series seven of 18 patients with complete AV block died within 34 days while hospitalized with acute myocardial infarction, a mortality of 39 per cent. Other reported mortality rates are from 40 to 100 per cent.³ ⁴ ⁶ ⁷ ¹² ²⁵-²⁷

**Prognosis for Recovery from Block.** In this series no patient who recovered had a residual atrioventricular conduction disturbance. Penton et al.¹ in a study of complete heart block, found that there was not a single instance in which a patient who had previously had normal sinus rhythm and who then developed complete AV block during acute myocardial infarction continued thereafter in complete heart block.

That the conduction disturbance is usually transient may be explained by the resolution of inflammatory and congestive changes in the region of the AV node when anastomotic channels become effective. Anoxemia is another factor that may account for the transient nature of the block. In clinical studies, Greene and Gilbert³¹ demonstrated that the AV node is very sensitive to oxygen lack. Shock or pulmonary edema accompanying acute infarction may produce sufficient anoxemia to cause temporary malfunction of the conduction system. Conduction disturbances occurring in the second, third, or fourth day after infarction probably result from extension of inflammation or edema from adjacent infarction and disappear when such local changes have subsided. Corticosteroid therapy may be of value in such situations.¹⁰-¹²

The brief duration of advanced AV block in acute infarction, however, makes it difficult to evaluate the results of such treatment.

**Comparison with Long-Term Block.** Permanent complete heart block as a sequel to acute myocardial infarction must be rare. However, many instances of persistent complete heart block are attributed to coronary or hypertensive heart disease,¹ and only a minority of such patients have a history of acute myocardial infarction.³²

The pathologic changes in long-term heart block are variable. The most common lesion is fibrosis of the bundle of His, but sometimes there is no obvious lesion. That the pathologic changes may be so minimal is attributed to clinically unrecognized occlusions in small arteries supplying the AV node and bundle, a vital part of the conduction system. On the other hand, marked changes in the region of the AV node and the bundle of His may occur without clinical disturbance in AV conduction.³³

**Conclusions**

Data are presented on 18 patients with advanced atrioventricular block during acute myocardial infarction. Fifteen of these had complete atrioventricular block; three had second-degree atrioventricular block.

Each patient demonstrated electrocardiographic evidence of acute inferior myocardial infarction.

In patients who recovered, heart block during acute myocardial infarction was transient and lasted not over 6 days. The brief duration of the conduction disturbance makes it difficult to evaluate the effect of adrenal steroid therapy.

Of 15 patients with complete atrioventricular block, six died during hospitalization. Of three patients with second-degree atrioventricular block, one died during hospitalization.

Three of four autopsies, done following death during the acute illness, demonstrated a recent thrombus in the proximal portion of the right coronary artery with recent infarction of the posterior wall of the left ventricle. The remaining one showed marked sclerotic narrowing of the right coronary artery 2 cm. from its ostium without gross evidence of myocardial infarction. In two autopsies done, 1 and 30 months after acute
infarction, there was old occlusion of the left circumflex artery. One of these patients had a scar at the left ventricular apex; the other had extensive healed infarction of the free wall of the left ventricle and of the interventricular septum.

Only one of the 15 patients with complete atrioventricular block had syncope during hospitalization. It would appear that a cardiac pacemaker or intravenous isoproterenol is usually not required in the treatment of patients with this complication of acute myocardial infarction.

Acknowledgment

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References


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Les troubles de la conduction auriculo-ventriculaire dans l'infarctus myocardique recent.

Amyl Nitrite and Angina Pectoris

During the past winter there has been in the clinical wards one case in which the anginal pain was very severe, lasted from an hour to an hour and a half, and recurred every night, generally between two and four A.M.; besides several others in whom the affection, though present, was less frequent and less severe...

When chloroform was given so as to produce partial stupefaction, it relieved the pain for the time; but whenever the senses again became clear, the pain was as bad as before. Small bleedings of three or four ounces, whether by cupping or venesection, were, however, always beneficial; the pain being completely absent for one night after the operation, but generally returning on the second. As I believed the relief produced by the bleeding to be due to the diminution it occasioned in the arterial tension, it occurred to me that a substance which possesses the power of lessening it in such an eminent degree as nitrite of amyl would probably produce the same effect, and might be repeated as often as necessary without detriment to the patient's health. On application to my friend Dr. Gangee, he kindly furnished me with a supply of pure nitrite which he himself had made; and on proceeding to try it in the wards, with the sanction of the visiting physician, Dr. J. Hughes Bennett, my hopes were completely fulfilled. On pouring from five to ten drops of the nitrite on a cloth and giving it to the patient to inhale, the physiological action took place in from thirty to sixty seconds; and simultaneously with the flushing of the face the pain completely disappeared, and generally did not return till its wonted time next night.—T. L. BRUNTON. "Use of Nitrite of Amyl in Angina Pectoris." The Lancet, 2: 97, 1867.
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