Cardiac Arrhythmias in Acute Myocardial Infarction
I. Complete Heart Block and Its Natural History*

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Complete heart block is recognized as a serious complication of acute myocardial infarction. In their collected series of cases, both Penton, et al7 and Rowe and White,9 found a mortality rate of 43 per cent and 42 per cent respectively within four weeks after the onset of acute myocardial infarction when complicated with complete A-V block. In the small number of cases reported earlier by Master et al,6 Mintz and Katz4 and by Cohen et al,9 the mortality rate was higher, ranging from 67 to 100 per cent. The average mortality of these five studies reported from 1938 to 1958 was 65.8 per cent. In more recent reports,10 the average mortality rate was 46.2 per cent. Of the 100 patients with myocardial infarction monitored by Julian et al,10 three of the eight patients who developed complete A-V block died acutely, a mortality rate of 38 per cent. It is believed that the recent trend toward lower mortality is due to both the early recognition of this arrhythmia and its control.

Review of the records of all patients admitted to Parkland Memorial Hospital in the past eight years with acute myocardial infarction and complete heart block revealed a mortality rate of 20.6 per cent, which is lower than previously reported. The purpose of the present study was to determine the clinical features and the fate of this arrhythmia in patients with acute myocardial infarction.

Materials and Methods

Thirty-two patients suffered 34 episodes of acute myocardial infarction with complete A-V block. The clinical records and the electrocardiographic tracings of these patients were reviewed. The diagnosis of acute myocardial infarction was made clinically and substantiated by the electrocardiographic findings on and following admission of the patient to the hospital. The onset of the myocardial infarction was determined by the time of onset of the chest pain, or by the increased severity of pain in a patient with previous history of angina pectoris.

Admission to the study was limited to those patients who developed the arrhythmia following the onset of an acute myocardial infarction. All patients with previous complete heart block (preceding the myocardial infarction) or that followed cardiac resuscitation were excluded from this study. Similarly, patients with "interference dissociation," first and second degree A-V blocks were not included in this report.

The site of the myocardial lesion, the ventricular and atrial rates and the fate of the arrhythmia were determined electrocardiographically. In 26 patients, the extent of the myocardial lesion was judged by the rise of the serum transaminase level (SGOT), and in five patients at postmortem examination.

Results

This group consisted of 22 men and ten women. Their ages ranged from 33 to 89 years. The average age for men was 60.3 years and that for the women was 72.8 years.

The clinical features of these patients are summarized in Table 1. In 30 of the 34 infarctions (or 88 per cent), the myocardial necrosis was over the diaphragmatic area of the heart. In two patients, the infarction was anterior. The arrhythmia was associated with diffuse subendocardial in-
TABLE 1—CLINICAL FEATURES OF PATIENTS WITH COMPLETE HEART BLOCK COMPLICATING ACUTE MYOCARDIAL INFARCTION

<table>
<thead>
<tr>
<th>A—Site of the Myocardial Lesion</th>
<th>No. cases</th>
<th>Per cent</th>
</tr>
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<tbody>
<tr>
<td>1—Posterior (Diaphragmatic)</td>
<td>30</td>
<td>88</td>
</tr>
<tr>
<td>2—Anterior (septal, lateral)</td>
<td>2</td>
<td>6</td>
</tr>
<tr>
<td>3—Subendocardial</td>
<td>1</td>
<td>3</td>
</tr>
<tr>
<td>4—Atrial</td>
<td>1</td>
<td>3</td>
</tr>
</tbody>
</table>

B—Clinical Severity of the Disease

| 1—Presence of clinical shock   | 16       | 47       |
| 2—Acute left heart failure     | 12       | 35       |
| 3—Presence of ventricular tachycardia | 7 | 21 |
| 4—Adams-Stokes episodes        | 2        | 6        |
| 5—SGOT over 200 units (total 26) | 9 | 35 |

In one case of the myocardial infarction and multiple atrial infarctions demonstrated at postmortem examination in a second patient.

Clinically, 16 patients exhibited evidence of shock, all of whom needed vasopressor drugs for the maintenance of adequate systemic blood pressure. Acute left heart failure was recognized in 12 patients. Digitalis therapy was instituted in these patients in spite of the presence of heart block. In seven patients, ventricular tachycardia became clinically manifest. In three patients of the entire group, the cardiac rhythm was continuously monitored on electrocardiographic tape. In one of these patients, ventricular tachycardia was recorded which was not otherwise recognized. Adams-Stokes attacks were not observed in the hospital, but in two patients, short episodes of these attacks presumably occurred prior to their admission.

Serum enzyme studies were obtained in 26 patients. In nine patients, the SGOT level was over 200 units per ml. In three patients the SGOT level rose over 1000 units per ml. This was ascribed to the presence of clinically recognizable acute hepatic engorgement.

In the majority of cases, the ventricular rate ranged from 40 to 60 beats per minute (Table 2). The rate was under 30 in only one patient, between 30 and 40 in five patients, between 40-50 in ten patients, and above 50 beats per minute in the remaining cases. The average ventricular rate for the entire group was 52 beats per minute.

The time of onset of the complete heart block and its evolution are depicted in Fig. 1. Twenty-seven patients (number shown on the abscissa of Fig. 1) had heart block on admission or within the first 24 hours of the onset of acute myocardial infarction. Two patients had their heart block on the second day, three on the third day and two on the fourth day, and none after the fourth day (or 96 hours after admission). The reversion of the complete heart block to sinus rhythm was rapid (the change is represented by heavy line in Fig. 1). The incidence of complete A-V block in patients who are living falls to 33 per cent on the second day, 21 per cent on the third, 17 per cent on the fourth and to 25 per cent on the fifth day. The slight increase shown on the fifth day was accounted for by the addition of two patients who developed their block at that time. By the end of the fifth day, only 18 per cent showed complete heart block.

Second degree A-V block was seen temporarily in some of these patients and did not persist in any of our patients longer than 48 hours. The incidence of this arrhythmia was represented by the hatched area in Fig. 1. Conversely, the incidence of sinus rhythm, (including those patients with first degree A-V block) rose from 24

<table>
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<tr>
<th>Table 2—VENTRICULAR RATE (BEATS PER MINUTE) IN PATIENTS WITH COMPLETE HEART BLOCK COMPLICATING ACUTE MYOCARDIAL INFARCTION.*</th>
</tr>
</thead>
<tbody>
<tr>
<td>Rate= 30 or 30-40 40-50 50-60 60-70 70-80 80 or</td>
</tr>
<tr>
<td>Number of patients 1 5 10 8 5 4 0</td>
</tr>
</tbody>
</table>

*In one patient, multifocal premature beats were present.
INCIDENCE OF COMPLETE HEART BLOCK

Figure 1: Diagram showing the daily incidence of complete heart block in patients with acute myocardial infarction. The heavy line represents the incidence of patients with complete heart block and the hatched area, that with second degree A-V block. On the first abscissa, the daily number of patients with newly observed complete heart block is shown and on the second abscissa, the number of hospital days.

DAILY INCIDENCE OF SINUS RHYTHM - INCLUDING 1º A.V. BLOCK

Figure 2: Curve representing the daily incidence of sinus rhythm in patients with complete heart block and acute myocardial infarction.
per cent on the first day of admission to 100 per cent at follow-up. The rate of appearance of sinus rhythm is shown in Fig. 2.

Twenty-three patients received no specific therapy aimed at correction of the arrhythmia, as seen in Table 3. Atropine was given to six patients. In two of them, rapid reversion to sinus rhythm occurred, but in one of these the arrhythmia recurred at the end of one hour. In two of the six patients receiving steroid therapy parenterally, the complete A-V block subsided on the second day of therapy. In one patient, quick reversion to second degree A-V block occurred within minutes of isoproterenol (Isuprel) administration and to normal sinus rhythm within 12 hours. In none of our patients was pacing of the heart performed, either externally or with an intracardiac electrode.

Seven patients died during their hospitalization as shown in Table 4. Six of these patients died in the first 96 hours of their illness and in one patient, death occurred on the 23rd day. In three patients, the complete heart block had reverted to sinus rhythm prior to death. In none of the 21 patients followed-up for a period of two months or more did complete heart block persist or reappear. In one patient, short episodes of Wenckebach's phenomenon were observed by continuous monitoring following heavy meals and moderate exercise.

The following case report illustrates the evolutionary changes that occurred in the cardiac rhythm when complete heart block complicates acute myocardial infarction. The cardiac rhythm was continuously monitored on an electrocardiographic tape. The two electrodes of the monitor were placed at the second right intercostal space and the left fifth intercostal space in the mid-axillary region, respectively.

**Case Report**

A 53-year-old mechanic was admitted to Parkland Memorial Hospital on December 17, 1964, with substernal chest pain of two hours' duration. For three days prior to admission, he had experienced recurrent episodes of substernal pain that lasted from one to two minutes each. The day of admission, the pain became worse, radiating to the neck and the medial aspect of the left arm. It was associated with nausea, vomiting and shortness of breath. On admission, at 2:00 p.m., the patient was still complaining of moderately severe pain. He was pale and clammy. The pulse was irregular, 45-50 beats per minute. The blood pressure was 100/70 mm of mercury. The heart sounds were distant, no murmurs or gallops were heard. On admission, he received morphine/atropine for chest pain and was placed on 100 per cent O2 breathing from December 17 to December 25, 1964.

The pertinent laboratory findings were: moderate leukocytosis, sedimentation rate 30 mm per hour which rose to 100 mm per hour. SGOT rose to 194 units per ml. The electrocardiogram revealed complete auriculoventricular block (ventricular rate 36 per min), marked ST elevation in II, III and aVF. The following day (December 18), evolutionary changes of acute inferior (posterior) myocardial infarction were observed. His course was uncomplicated, and recovery was uneventful. The patient was discharged January 29, 1965.

Continuous monitoring of the cardiac rhythm on tape revealed the following sequential

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**Table 3—Treatment Used in Patients with Complete Heart Block Complicating Acute Myocardial Infarction**

<table>
<thead>
<tr>
<th></th>
<th>Atropine</th>
<th>Steroids</th>
<th>Isoproterenol</th>
<th>No Treatment</th>
</tr>
</thead>
<tbody>
<tr>
<td>Treated</td>
<td>6</td>
<td>6</td>
<td>2</td>
<td>23</td>
</tr>
<tr>
<td>Reverted to Sinus Rhythm</td>
<td>2</td>
<td>2</td>
<td>1</td>
<td></td>
</tr>
</tbody>
</table>

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**Table 4—Mortality Rate in Patients with Complete Heart Block Complicating Acute Myocardial Infarction**

<table>
<thead>
<tr>
<th></th>
<th>Died Within 96 hours</th>
<th>Over 96 hours</th>
<th>Total</th>
<th>Over 2 months</th>
<th>Under 2 months</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>6(17.7%)</td>
<td>1(2.9%)</td>
<td>7(20.6%)</td>
<td>21(61.8%)</td>
<td>6(7.6%)</td>
</tr>
<tr>
<td>In 3° block</td>
<td>4</td>
<td>3</td>
<td>7</td>
<td>0</td>
<td>27</td>
</tr>
<tr>
<td>In sinus</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
changes (Fig. 3): Approximately ten hours after admission (12:17 a.m. of December 18, 1964) first degree A-V block (PR interval=0.34 sec.) appeared. At 4:07, a decrease in the PR interval (0.26 sec.) was observed, and at 5:40, the PR interval returned to normal level (0.20 sec.).

DISCUSSION
The occurrence of complete auriculoventricular block in the course of acute myocardial infarction denotes severe disease and carries a serious prognosis. In this study, the clinical finding of left heart failure and shock were more commonly observed in these patients than in the uncomplicated acute myocardial infarction (without heart block). The appearance of these complications, namely left heart failure and shock, is probably the result of the extent of the myocardial necrosis, as attested by the marked elevation of SGOT levels in these patients and was not solely caused by the complete A-V block.

Adams-Stokes attacks were less frequent than anticipated in spite of the critical condition of these patients. In earlier reports, the incidence of these attacks ranged from 16 to 40 per cent of these patients. In the present study, only two patients gave history of short episodes of Adams-Stokes. The relatively low attack rate was due to the transient nature of the arrhythmia and maintenance of an adequate ventricular rate. In this study, one patient had a ventricular rate below 30 beats per minute, but in the majority of instances the ventricular rate ranged between 40 and 60 beats per minute.

In agreement with previous reports, most acute complete heart blocks were associated with diaphragmatic (inferior) myocardial infarction. In other reports, however, the arrhythmia was equally seen in patients with acute infarction of the anterior wall of the heart. The interventricular septum receives most of its blood supply from the left anterior descending coronary artery. The right coronary artery supplies the diaphragmatic area of the heart and in 83-92 per cent in individuals it gives an arterial branch to the auriculoventricular node, and in the remaining instances, the left circumflex artery supplies the node. The site of occlusion of the right coronary artery in acute diaphragmatic infarctions was proximal to the origin of the A-V nodal artery in 84 per cent of patients, but only in 1-26 per cent of these patients complete heart block occurred. The surprisingly low in-

Figure 3: ECG strips obtained by the continuous recording system in the reported case.
The occurrence of complete heart block following occlusion of the right coronary artery was ascribed to the rich arterial anastomoses in the region of the auriculoventricular node (vide infra)."

The appearance of complete heart block and its subsidence were both rapid. It was observed previously that the heart block appears early, usually within the first few days of acute myocardial infarction and in rare instances, the heart block occurred as late as the 14th day. In these latter patients, an extension of the myocardial process might have been responsible for the development of the heart block. In our study, heart block became manifest in the first 24 hours of admission in 27 of the 34 patients, and none after the fourth day. The subsidence of the heart block was equally rapid. Reversion to sinus rhythm with or without prolonged PR interval was completed by the fifth day in the majority of patients in this series. In two patients reversion to sinus rhythm occurred on the sixth day and ninth day, respectively and in one patient by the end of the fourth week. The exact duration of the arrhythmia could not be determined, but in the three patients who were continuously monitored, the arrhythmia lasted from three to ten hours. The process of reversion of this heart block to sinus rhythm was, in most instances, a stepwise decrease in the magnitude of the heart block. As diagrammed in Fig. 4, the arrhythmia passed from a complete heart block to first degree A-V block and to sinus rhythm with normal PR interval, but in some patients this process passed through an intermediate step of a second degree A-V block. Short of continuous monitoring of the cardiac rhythm, it is not possible to determine the frequency of a second degree A-V block in the recovery of the sinus mechanism. The rapid resumption of the normal A-V conduction and the relatively low incidence of complete heart block in acute myocardial infarction are primarily due to the rich arterial network and anastomotic vessels that supply the A-V node. As stated earlier, the A-V node is supplied by an artery which arises in most instances from the right coronary artery, and in some patients from the left circumflex artery. In 1927, Kugel" described wide anastomotic vessels between the left circumflex and the right coronary arteries in the region of the A-V node. Following occlusion of the A-V nodal artery, these anastomotic vessels are expected to provide a sufficient quantity of arterial blood to the A-V node to restore its normal function. In rare instances where these channels are inefficient and/or when both the right coronary and left circumflex arteries are obliterated, complete heart block becomes permanent.

An alternative explanation for the recovery of the sinus mechanism lies in the subsidence of the inflammatory process that follows acute myocardial infarction. It would be expected in these instances that the inflammatory process would take a few
days to subside and it would be difficult therefore to explain the rapid regression of the complete heart block on this basis. As observed in the patient reported above, the complete heart block lasted a short period of time, approximately ten hours, to be followed by a gradual decrease in the duration of the PR interval. These changes in the A-V conduction could best be explained by the return of the arterial blood supply of the A-V node to normal.

From such observation and from the therapeutic measures employed in our patients, it became clear that the anatomic nature of the vessels in the area of the A-V node rather than therapy was the single major factor responsible for the restoration of the A-V node to its normal function in the post-myocardial infarction period, as 23 patients in this series received no specific therapy aimed at correction of the arrhythmia.

The possibility that excessive stimulation of the vagal neuroreceptor centers at the opening of the coronary sinus occurs in acute posterior myocardial infarction, induced us to try atropine in six patients. In two of these patients, rapid reversion of the heart block to sinus rhythm occurred and in one of these two, the heart block recurred at the end of one hour, suggesting, perhaps, that such a mechanism may play an additive role in the production of heart block.

The rationale for steroid therapy in complete heart block was based on the anti-inflammatory properties of these agents and on the finding of an accelerated auriculoventricular conduction in Cushing's disease. In our patients, the role that these drugs played in the reversion of the arrhythmia in the post-myocardial infarction period was difficult to assess because of the transient nature of the arrhythmia in the untreated group. The quickly beneficial effects of intravenous steroids noted by Dall and Buchanan were not seen in any of our patients. Isoproterenol (Isuprel) was not given an adequate trial in this series. In one patient, isoproterenol reverted the cardiac rhythm to second degree A-V block within minutes and to sinus rhythm thereafter. It was surmised that the beneficial effect of this drug might have been due to improvement of the marginal blood supply to the A-V node.

The routine use of intracardiac pacemaker in these patients is probably not indicated, and we agree with Julian et al. that ventricular arrhythmia and fibrillation, venous thrombosis and puncture of the myocardium are hazards which make their use dangerous. However, in patients with critically low heart rates and/or recurrent Adams-Stokes attacks, intracardiac pacemaker may prove life-saving.

**Summary**

Complete heart block complicating an acute myocardial infarction frequently follows lesions of the diaphragmatic wall. This arrhythmia is a temporary phenomenon. It subsides by a stepwise decrease in the magnitude of the A-V block, from complete heart block to second heart block in some patients and then to first degree A-V block and finally to normal A-V conduction. No permanency of the heart block was observed in our patients.

The rich vascular anastomosis in the region of the A-V node is responsible for restoring the normal conduction to the A-V node. The effectiveness of therapeutic drugs is difficult to assess because of the transient nature of the arrhythmia.

**Resumen**

Las lesiones del área diafragmática del miocardio consecutivas al infarto cardíaco son con frecuencia seguidas de bloqueo cardíaco total. Este regresa por etapas sucesivas de bloqueo completo, bloqueo A-V de primer grado y finalmente conducción A-V normal. En ninguno de nuestros pacientes se constituyó un bloqueo permanente.

La extensa red vascular de la región del nódulo A-V facilita la restauración del funcionamiento normal del mismo. La efectividad de la medicación es difícil de valorar, debido a la naturaleza transitoria de la arritmia.

**Resumé**

Le bloc auriculo-ventriculaire complet compliquant un infarctus myocardique aigu s'ob-

Les riches anastomoses vasculaires dans la région du nœud auriculo-ventriculaire sont responsables de la restauration d'une conduction normale dans ce nœud. L'efficacité des médications thérapeutique est difficile à prouver, en raison de la nature transitoire de l'arythmie.

References
10 Julian, D., Valentine, P. and Miller, G.: "Disturbances of rate, rhythm and conduc-


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