Some Clinical Features of Complete Heart Block

By George B. Penton, M.D., Harold Miller, M.D., and Samuel A. Levine, M.D.

A review was made of 251 cases of complete heart block. They were divided into the following groups: coronary artery disease (nonacute), 58 cases; myocardial infarction (acute), 49 cases; hypertensive heart disease, 62 cases; rheumatic heart disease, 21 cases; miscellaneous, 16 cases; "etiology undetermined," 18 cases; and digitalis intoxication, 27 cases. Among the various clinical features analyzed were the sex and age factors, the occurrence of syncope, palpitation, congestive failure, anginal pain, and the duration of life after the onset of major symptoms. Methods of treatment are discussed.

The clinical course of patients with complete heart block, with or without attacks of Adams-Stokes syncope, is very variable. Events are often unpredictable. Some live a short time, others may carry on for a great many years. The causes of the underlying defect are numerous and the circumstances under which the major symptoms arise vary greatly. Even the nomenclature is somewhat confusing. A sudden total arrest of the heart when there is no evidence of conduction defect may be called an episode of Adams-Stokes syncope by one observer and not by another. The very mechanism of fainting is also different in different individuals. Although much has been written about this subject, it seemed worth while to analyze in greater detail some of the specific features of complete heart block. An attempt was made particularly to see if there were distinct differences in the course of events and in the findings when heart block developed under one set of circumstances, such as coronary artery disease in contrast to valvular disease, or when no definite etiology could be determined.

In reviewing the very extensive literature on complete heart block comprising over 200 publications, only a few seem to be pertinent for the purposes of this study. One of the first to divide the attacks of syncope into two types was Schwartz. This was more clearly discussed and differentiated by Parkinson, Papp, and Evans. Somewhat later a review was made by White and his associates.

Scope and Materials

Patients who manifested only first or second degree atrioventricular block with or without syncope were not included. Similarly, cases of atrioventricular dissociation typified by "interference dissociation or ventricular capture phenomenon" were excluded. These were all cases in which the ventricular rates were more rapid than the atrial rates. Furthermore, syncopal attacks associated with standstill of the sinus node or atrial mechanism were also excluded if no evidence of atrioventricular block was present. The only cases forming the basis for this study were those which at one time or another had third degree atrioventricular block in which the ventricular rate was slower than the atrial rate. In most instances, the atria were contracting independently of the ventricles, though atrial fibrillation was present in 43 cases, excluding the digitalis intoxication group. In many, first degree or second degree heart block had occurred at one time or another. In 92 per cent of the cases the diagnosis of complete heart block was verified by electrocardiograms. In the other 8 per cent the diagnosis on clinical grounds seemed quite certain. With these

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restrictions, there were 251 cases available for investigation. One hundred fifty-three were obtained from the records of the Peter Bent Brigham Hospital during the years 1913 to 1955, and 98 cases were from the consultation practice of one of us.

**Clinical Picture**

Complete heart block is generally associated with a slow ventricular rate, i.e., 30 or 40 beats per minute. Occasionally, the heart rate may be 50 or 60 beats per minute or a little greater. The bradycardia may be initiated suddenly by an attack of syncope. In others, a slow heart rate is suddenly observed without any significant change in the symptomatology or awareness of the change in rhythm. There are numerous instances in which syncopal attacks occur and with recovery the heart returns rather promptly to a normal sinus rhythm. In such cases, an asystole has taken place, accounting for the spell of unconsciousness, and is followed for only a short time by varying degrees of atrio-ventricular heart block. In a few minutes a normal sinus rhythm is established without evidence of conduction defect. These cases generally display more permanent complete heart block some months later. Before this is apparent, they present puzzling problems and may be misinterpreted as instances of hysteria, benign syncope, or epilepsy. In some of these cases minor clues may be found that direct the observer's attention to the true nature of the condition; these include complete or incomplete bundle branch block or a full P-R interval of 0.20 to 0.22 second. In these puzzling cases, the physician would do well to make every effort to observe the actual episode; for during those brief moments, the diagnosis may be quite obvious. Another clue in this regard is obtained by carotid sinus stimulation. If as a result of this test the heart slows and a temporary transient complete atrioventricular dissociation results, it is very likely that permanent complete heart block may develop at some later date. The inference from the above is that for some period of time, patients may have a normal sinus rhythm after a syncopal attack and yet be suffering from Adams-Stokes episodes, and only later manifest permanent complete heart block.

The symptoms due to complete block itself are very few. The slow ventricular rate of 30 does not necessarily produce pain, dyspnea, or significant prolonged weakness. The patient may suddenly become aware that the heart is beating slowly, and this may be his first complaint to the physician. In a fair number, palpitation may be a primary symptom. More often momentary weakness, dizziness, or actual spells of unconsciousness are the major difficulties. In some, profound weakness, prostration, and even a prolonged state of unconsciousness may result when the heart continues to beat regularly, but extremely slowly, i.e., 15 to 20 times per minute. The duration of actual syncopewill depend upon the length of ventricular asystole. If this lasts two to three seconds the patient may have a feeling of a wave passing over him, with a momentary sense of uncertainty or blankness, just like a petit mal. If it lasts several seconds and the patient is standing or walking, he will drop to the ground, regaining consciousness immediately, having no idea of what has happened. If the asystole continues longer, convulsive movements appear with stertorous breathing. After half a minute or more, if the heart does not resume its beats, the patient may appear to be dead, without either cardiac or respiratory function. Occasionally during such long intervals, the continuing contractions of the atra may still be visibly displayed in faint pulsations of the jugular vein. Finally, if the heart does not resume its beat in several minutes, death occurs. On the other hand, it is amazing how instantaneously consciousness is restored if the heart does start contracting; in fact, after one or two beats the patient may resume the previously interrupted conversation.

The diagnosis of complete heart block is generally a simple matter. Apart from the history of the symptoms mentioned above, physical examination in the great majority of cases reveals distinctive objective findings. On auscultation of the heart the rate is generally slow and regular, 30 to 40 beats per minute. Not infrequently, this regular rhythm may be interrupted by premature beats. The single most valuable and almost pathognomonic sign is the changing intensity of the first heart sound heard best at the apex with a regular slow beat.
In some instances it will be necessary to listen as long as one-half to a whole minute before the loud first sound appears (bruit de canon). With other cycles the first sound may be muffled, faint, or of average intensity. This pathognomonic sign will be absent in cases of complete heart block with atrial fibrillation, as the first sound will then be constant in its intensity. A second sign is the detection of faint aural sounds during the long diastolic intervals. These are more audible in the early than in the late portion of diastole and are heard best between the apex and lower sternum. Occasionally these aural contractions will produce visible, faint pulsations in the jugular pulse. A more distinctive phenomenon to be observed in the neck is the detection of occasional, sudden, large pulsations in the jugular vein, produced by the simultaneous contraction of the atria and ventricles. Finally, it will be difficult in most cases to alter the ventricular rate by carotid sinus stimulation.

Classification of Cases
Among the 251 cases in this study, there were 58 (23 per cent) with coronary artery disease (nonacute), 49 (19 per cent) of acute myocardial infarction, 62 (24 per cent) with hypertensive heart disease, 21 (10 per cent) with rheumatic heart disease, 16 (6 per cent) with rare forms of heart disease (miscellaneous), 18 (7 per cent) in which the etiology was not known, and 27 (11 per cent) with digitalis intoxication.

Those patients who experienced the clinical syndrome of angina pectoris, coronary artery insufficiency, or previous acute myocardial infarction constituted the coronary artery disease group. Those patients who experienced a recent myocardial infarction in association with complete heart block were placed in the acute myocardial infarction group. Those having myocardial infarction, coronary thrombosis, or significant coronary artery sclerosis at autopsy were also included in these sections. Age alone in the absence of any of these criteria was not deemed sufficient for inclusion in this category. Criteria for the hypertensive heart disease group included a systolic blood pressure of 170 mm. Hg or greater; it is appreciated that this demarcation is an arbitrary one. The diastolic pressure in complete heart block is so variable that it was not employed as a useful criterion. All the cases with rheumatic heart disease had aortic, or mitral, or combined aortic and mitral valve disease with or without a past history of rheumatic fever. The miscellaneous or rare forms of heart disease and those with complete heart block of etiology undetermined are discussed later. Those patients were included in the digitalis intoxication group in whom excessive digitalis appeared to be the cause of the complete heart block. All these patients had heart disease due to one cause or another, but had complete heart block as a consequence of excessive digitalis therapy. This group was regarded as unique and sufficiently different from the other cases to merit separate study. They, therefore, will not be included in the statistical analyses of features such as age, sex, and other factors. This type of heart block is induced by the drug and is not a necessary part of the disease process of complete heart block.

Some of the cases had more than one of the above etiologic features. Those who had hypertension and rheumatic valve disease were classified in the rheumatic group. Patients in whom the heart block developed in relationship to coronary artery disease, i.e., directly with acute myocardial infarction, and those in whom angina or myocardial infarction had been present in the past, were put into the appropriate coronary group even if they had hypertension or rheumatic valvular disease. Although there were 9 instances in this study in which there was a positive serologic reaction for syphilis, in only 2 was syphilitic heart disease present and thought to be related to the complete heart block.

Age and Sex Distribution
Of the 224 cases, 127 (57 per cent) were males and 97 (43 per cent) were females. The age group extended from 10 to 85 years, with 84 per cent occurring in the age group from 41 to 80 years. Table 1 is based upon the age when the patient was first observed by us to have complete heart block and does not necessarily represent the age at which heart block first de-
TABLE 1.—Age Distribution by Decades

<table>
<thead>
<tr>
<th>Age</th>
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<tr>
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<td>11-20</td>
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<tr>
<td>61-70</td>
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TABLE 2.—Age and Sex in Various Groups

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<th>Av. Age yrs.</th>
<th>Age Range yrs.</th>
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<td>30</td>
<td>19</td>
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<td>Acute myocardial infarction</td>
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<td>62.5</td>
<td>40-85</td>
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<tr>
<td>Hypertensive heart disease</td>
<td>62</td>
<td>33</td>
<td>29</td>
<td>63.4</td>
<td>19-82</td>
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<tr>
<td>Rheumatic heart disease</td>
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<td>8</td>
<td>13</td>
<td>52.5</td>
<td>16-79</td>
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<tr>
<td>Etiology undetermined</td>
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<td>9</td>
<td>9</td>
<td>44.5</td>
<td>22-78</td>
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<td>11</td>
<td>5</td>
<td>45.1</td>
<td>10-82</td>
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<td>127</td>
<td>97</td>
<td>59.3</td>
<td>10-85</td>
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</table>

dveloped or was first observed by others. It should be noted that customarily patients under the age of 12 years are not eligible for admission to the Peter Bent Brigham Hospital.

The average age of all patients when first observed with complete heart block was 58.5 years; if the digitalis group is excluded, the average age was 59.2 years. The 3 groups, i.e., coronary artery disease, acute myocardial infarction, and hypertensive heart disease, were the oldest (62.5 to 63.5 years). The miscellaneous group and the group designated "etiology undetermined" were the youngest, about 45 years, and the group with rheumatic heart disease were intermediate (52.5 years) (table 2).

The average age at death in the 126 cases where the time of death was known was 63.2 years. The corresponding figures for the various subgroups of these cases were as follows: Chronic coronary artery disease, 65.9 years; acute myocardial infarction, 65.2 years; hypertensive heart disease, 60.3 years; rheumatic heart disease, 62.7 years; etiology undetermined, 55.0 years; miscellaneous, 55.0 years. The age was generally a little greater in those who had permanent complete heart block than in those who had transient complete heart block. There was a definite tendency for the males to predominate in all groups except those with rheumatic heart disease, in which group there were 13 females and 8 males.

SYNCOPE

Syncope occurred in 137 cases (61 per cent). It was the first evidence of the illness in 48 cases (21.4 per cent). The frequency of these episodes varied greatly. Rarely, only one such spell of unconsciousness occurred. In others, they may have taken place at daily, weekly, monthly, or sporadic intervals, and there were extreme instances in which actual status epilepticus was present with the effective heart action stopping and starting every few minutes. With the exception of the digitalis intoxication group, syncope occurred in all the subgroups in this study with varying frequency: 77.6 per cent of the chronic coronary artery disease group, 43 per cent of the acute myocardial infarction group, 72 per cent of the hypertensive heart disease group, 38 per cent of the rheumatic heart disease group, 56 per cent of the miscellaneous forms of heart disease, and 44 per cent of the etiology undetermined group.

Descriptions of attacks of syncope vary, and the following serves as an example: "Dizziness comes suddenly without warning and not related to exertion; it begins with a feeling of coldness all over the body, and then a sensation of warmth passes quickly up the body and there is a feeling of fullness and heat in the head; everything becomes black before the eyes and then the feeling passes quickly and a feeling of well being exists thereafter except for a sense of slight oppression in the chest and very slight breathlessness for a few minutes, the whole attack lasting five or ten minutes."

Similarly, as the attacks themselves vary, so might the description vary, but the following are illustrative: "The pulse was 45 and suddenly failed to come through; on auscultation
no sounds were heard for 18 seconds; after cessation of the heart beat, the patient began to quake, her arms began to twitch and her body began to straighten out, and her face became ashen gray. After the end of the 18 second period, the same heart sounds were heard followed by other heart sounds at irregular intervals, each sound becoming louder but irregularly spaced. About 15 seconds after the end of asystole, the rate increased and then became regular.” Another type of observation is illustrated in the following:

“I was called to see the patient after several so-called minor attacks in quick succession. The patient spoke to me telling me of the shock-like pain traveling over the right arm to the shoulder; while talking to him and feeling the radial pulse, I felt a period of asystole begin, was able to count ten mentally when the patient announced he was having another spell; with stethoscope over the heart no further heart sounds or radial impulses came through for about 25 more counts (thus 35 counts in all); during this time, the patient’s face was blanched of all color, eyes were rolled upward, and his hands stiffened; then, the heart began to beat, the patient became flushed, his head came forward, and he spoke almost simultaneously. The patient stated that he remembered nothing and that he did not feel well; there were no twitchings of any sort and the heart rate was beating at a rate of about sixty-four and regular after this attack. In this case asystole of 10 seconds with the patient lying flat in bed caused no symptoms. He was aware that a spell was coming only after that interval of 10 seconds and actual syncope ensued a few seconds later.”

There are all degrees of severity of the attacks and there are marked variations in the duration of Adams-Stokes syncope. These range from a brief fleeting aura of faintness to complete unconsciousness, cyanosis, eyes rolled upward, body twitching, face drooping, stertorous breathing, frothing at the mouth, and complete unresponsiveness. With the resumption of the first heart beat there may be sudden awareness of the surroundings and a sense of well being.

The duration of life after the onset of syncope varied greatly among these patients. It was obviously very short in those who had Adams-Stokes attacks for the first time with a fatal acute myocardial infarction. The average duration of life after the first syncopal attack in cases other than those just mentioned was 6.9 years, the range being from several hours to 11 years. In many cases syncope preceded permanently established complete atrioventricular block by a period of months or a few years. Of 39 patients who died in Adams-Stokes attacks, 31 had previous syncope and 8 had no previous syncope. The fact that a patient had previous syncopal attacks with complete heart block would make it seem more likely that he would have a subsequent sudden eventual exitus.

Apart from definite syncope with loss of consciousness, periods of dizziness and weakness were quite common. There were 36 patients (17 per cent) in whom these symptoms occurred without true syncope, 84 (38 per cent) who had both syncope and dizzy spells, and 46 (21 per cent) who had spells of unconsciousness without additional complaints of dizziness.

Palpitation

Palpitation occurred in 43 patients. In comparing the incidence of palpitation with heart rate, it was found that of 55 patients with a slow rate of 30 or less, there were 9 (16 per cent) who had palpitation and 46 who did not have palpitation. Of 169 patients with rates greater than 30 per minute, 42 (25 per cent) had palpitation and 127 did not have palpitation. Although the symptom of palpitation generally connotes a more rapid heart beat, it is significant that the same symptom is not uncommon among those patients who have a very slow heart rate.

The suggestion has been made that in those patients experiencing syncope, the premonitory symptom of palpitation might be useful in predicting syncope due to ventricular acceleration. In this study, 2 of 13 patients with ventricular acceleration (ventricular tachycardia, ventricular fibrillation, ventricular flutter) were aware of palpitation prior to their attack; none of the 21 patients with syncope due to ventricular asystole was aware of palpitation prior to the attack. These cases were all documented by electrocardiograms. There were, however, other cases with syncope not documented by electrocardiograms who experienced palpitation but the subsequent rhythm was not ascertained. Only occasionally, therefore, can the symptoms.
Table 3.—Heart Rate in Patients with Complete Heart Block

<table>
<thead>
<tr>
<th>Heart Rate</th>
<th>No. of Patients</th>
</tr>
</thead>
<tbody>
<tr>
<td>Less than 20</td>
<td>14</td>
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<tr>
<td>21–30</td>
<td>38</td>
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<tr>
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<td>71–80</td>
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<tr>
<td>81 or above</td>
<td>2</td>
</tr>
<tr>
<td>Total</td>
<td>224</td>
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of palpitation preceding the syncopal attack aid the physician in distinguishing attacks that are initiated by an accelerated ventricular rate from those that start abruptly with asystole.

**Congestive Heart Failure and Dyspnea**

Objective evidence of congestive heart failure was present in almost 40 per cent of the patients studied. Failure varied from minimal to severe. In 23 patients, heart failure preceded the onset of complete heart block, in 16 congestive heart failure or dyspnea developed concomitantly with the complete heart block, and in 8 these features appeared after the onset of complete heart block. In the other patients, the relationship of the complete heart block to congestive heart failure was not very clear. Only 10 of 85 patients in whom the mode of death was known died of progressive congestive heart heart failure.

Dyspnea was the presenting feature in about one fourth of the cases, and was present clinically in slightly less than two thirds of all the patients. The dyspnea varied from mild to marked, and there did not appear to be any clinical relationship between the heart rate and the degree of dyspnea. Dyspnea and congestive heart failure appeared in patients having a slow heart rate as well as those having a more rapid heart rate. It was noted in a patient with a heart rate of 18 per minute and in another with a heart rate of 72 per minute.

In the therapy of congestive heart failure, all the usual modes of treatment were employed. When needed, digitalis was administered, unless the heart block was due to overdigitalization and continued digitalis would have led to aggravation of the congestive failure.

Congestive heart failure and dyspnea appear to be manifestations of the underlying heart disease, and the presence of complete heart block is incidental in the course of events. Although we have the general impression that the slow heart rate may actually be beneficial to a certain extent and decrease the degree of congestive heart failure, the observations in this study were neither detailed enough nor sufficiently adequate to offer proof in favor of this concept.

**Heart Rate**

The heart rate varied from a low of 16 to a high of 97, and the over-all average was 38 beats per minute as documented by the electrocardiogram. As shown in table 3, all but 6 patients had heart rates of 60 or less (this does not include the digitalis intoxication group). Rates of 8 and 12 were reported clinically, but not documented by the electrocardiogram. The heart rate was regular in 181 of 224 patients. In the classical description of complete heart block, the rate is usually said to be particularly regular, but there are cases, not too uncommon, in whom the ventricular rhythm is somewhat irregular because of the appearance of ventricular premature beats. It may be mentioned here that there are patients in whom the ventricles as well as the atria are affected by carotid sinus pressure or vagal influence. There were 55 cases of complete heart block having a rate of 30 or less. The average rate recorded in the various groups was as follows: coronary artery disease (nonacute), 32; acute myocardial infarction, 43; hypertensive heart disease, 37; rheumatic heart disease, 45; miscellaneous, 39; etiology undetermined, 43; and digitalis intoxication, 54 beats per minute.

Although the characteristic of complete heart block is a slow, regular rate, slight irregularities were not uncommon, and the actual ventricular rate varied from under 20 to 60 or more. Variability in the heart rate occurred in the same individual at various times.

**Blood Pressure and Pulse Pressure**

Systolic blood pressure averaged 172 mm. Hg and diastolic blood pressure averaged 79
mm. The highest systolic blood pressure was 280 mm. Hg and the lowest systolic blood pressure was 60 mm. The highest diastolic blood pressure was 150 mm. Hg and the lowest was zero. Pulse pressure averaged 93 mm. and ranged from a low of 28 mm. to a high of 180 mm. Hg.

The accompanying table 4 demonstrates the relationship of heart rate to blood pressure and the average of these readings in each subgroup. It is obvious that with well-marked hypertension, the pulse pressure will necessarily be large. In cases without hypertension, however, greater blood pressure readings were more likely to accompany slower heart rates. In following the same individual who goes in and out of complete heart block, it was often found that as the heart rate slowed the systolic pressure rose and the diastolic fell. In that way it was possible for the heart to maintain as great an output, and even a normal one, at the rate of 30 beats per minute as it would with a rate of 70.7

**Heart Size**

The heart was considered to be enlarged in 140 of 224 cases. In 52 the enlargement was slight, in 49 moderate, and in 32 marked. The estimate of the increased heart size was based upon x-ray examination or physical findings. Absence of cardiac enlargement in 47 cases of complete heart block was confirmed by fluoroscopic examination or x-ray films. In a patient with rheumatic heart disease and complete heart block, who was followed for a period of over 15 years, cardiac enlargement was never demonstrable. Cardiac enlargement need not necessarily be associated with complete heart block; the degree of heart enlargement for the most part went, pari passu with the accompanying type of heart disease.

**OTHER CONDUCTION DEFECTS**

Of the 224 patients, there were 62 in whom the electrocardiograms were indicative of right bundle branch block and 60 in whom tracings were of the type generally designated as left bundle branch block. It is evident that in these instances of so-called bundle branch block the impulse may have actually arisen in one of the branches rather than in the common junctional tissue or node. First or second degree block, or both, was present prior to or subsequent to the complete heart block pattern in 67 patients. Two patients had documentary evidence of both right and left bundle
branch block at varying periods while they had complete heart block. Thus, third degree block is frequently preceded by a lesser degree of atrioventricular block and is often accompanied or preceded by bundle branch block.

**Other Associated Rhythms**

There were 83 patients in whom the atrial rate was under 100 beats per minute. In 41 the rate was between 100 and 120, and in 12 the range was from 120 to 200. There were 2 documented cases of paroxysmal atrial tachycardia with complete heart block. In 57 cases atrial fibrillation was associated with complete heart block, 14 of these patients were in the “digitalis intoxication” group. Atrial flutter concomitant with complete heart block occurred in 4 patients. The atrial rate was fairly constant in some patients with complete heart block and in others the atrial rate varied slightly from day to day. In still others, there were sharp increases in the atrial rate when an abnormal form of atrial rapid heart action developed. The atrial and ventricular rates may simultaneously but independently increase or decrease. In other instances, the atrial and ventricular rates may simultaneously but also independently be altered in opposite directions. Thus, the atrial rate may increase when the ventricular rate decreases or the atrial rate decrease when the ventricular rate increases.

Ventricular tachycardia and ventricular fibrillation were present in 15 cases documented by the electrocardiogram. In some, there followed an appreciable interval of electric inactivity and, in others, the ventricular complexes of complete heart block were quickly resumed after the rapid ventricular rhythm. Ventricular asystole was documented by the electrocardiogram in 21 cases; 10 were in the coronary artery disease group (nonacute), 6 were in the acute myocardial infarction group, 2 in the hypertensive heart disease group, 1 in the rheumatic heart disease group, and 2 in the miscellaneous group.

Observations in this study support the conclusions made by Parkinson, Papp, and Evans, in 1941, that the syncopal attacks may be due to asystole associated with complete heart block or to ventricular acceleration such as ventricular tachycardia or ventricular fibrillation.

**Permanence of Complete Heart Block**

There were 176 patients with permanent complete heart block, 29 others had transient complete heart block, and 19 additional patients had repeated transient complete heart block. Those in whom complete heart block was recorded at every examination from its inception to death or to the time of last follow-up were considered to have permanent complete heart block. Included in this group also were those who had transient or intermittent block, or both, but who then progressed to a permanent block of a fixed nature and of long duration. The group with transient complete heart block included those patients who had a single period of complete heart block preceded and followed by a lesser degree of block or normal rhythm. The number of the latter who returned to a normal sinus rhythm was 17 (coronary artery disease, nonacute, 1; acute myocardial infarction, 12; miscellaneous, 1; etiology undetermined, 1; rheumatic heart disease, 1; and hypertensive heart disease, 1). The number of those who returned to partial heart block was 12 (coronary artery disease, nonacute, 2; acute myocardial infarction, 6; hypertensive heart disease, 1; rheumatic heart disease, 1; and miscellaneous, 2) (table 5). Repeated transient block included those patients who had complete heart block on more than one occasion; 7 so classified returned to

<table>
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<th>Permanent block</th>
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<tr>
<td>Acute myocardial infarction</td>
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<td>16</td>
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<td>Etiology undetermined</td>
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<td>1</td>
<td>18</td>
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<tr>
<td><strong>Total</strong></td>
<td><strong>176</strong></td>
<td><strong>29</strong></td>
<td><strong>19</strong></td>
<td><strong>224</strong></td>
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normal sinus rhythm (coronary artery disease, nonacute, 1; acute myocardial infarction, 1; hypertensive heart disease, 2; rheumatic heart disease, 2; and etiology undetermined, 1) and 12 returned to partial heart block (coronary artery disease, nonacute, 2; acute myocardial infarction, 3; hypertensive heart disease, 5; and miscellaneous, 2).

In the majority of patients in this study, complete heart block was of a permanent nature. However, there were some in whom the complete heart block was not permanent but of a transitory nature. In this latter group, it is of interest that the largest number occurred in association with acute myocardial infarction.

**Duration of Complete Heart Block**

The longest time interval of a patient having complete heart block, documented by the electrocardiogram, was 21 years. The longest duration of complete heart block on a clinical basis was 47 years. There were 4 cases of over 15 years' duration documented by the electrocardiogram, 2 cases of from 20 to 25 years, and 3 from 15 to 19 years' duration on the basis of the clinical picture and history. Aside from those cases in the digitalis group, in which there were some with complete heart block for only a few hours, there were others in whom the duration was less than one day, particularly among those who died of acute coronary thrombosis. In table 6 are recorded the data concerning those cases in whom the diagnosis of permanent complete block was proved by electrocardiogram. The duration of life after the first occurrence of syncope and after the first development of complete heart block, where the time of death was known, varied somewhat in the different subgroups. It was generally about a year longer after the initial syncope (average 35.2 months) than after the first appearance of heart block (aver-

<p>| Table 6.—Comparative Observations of Patients with Complete Heart Block (C.H.B.) |
|----------------------------------|----------------|----------------|----------------|----------------|----------------|</p>
<table>
<thead>
<tr>
<th>Group</th>
<th>Age first observed</th>
<th>Length of life after first observed</th>
<th>Age at onset first syncope</th>
<th>Length of life after first syncope</th>
<th>Age onset of C.H.B.</th>
<th>Length of life after onset of C.H.B.</th>
<th>Age at death C.H.B.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Acute myocardial infarction</td>
<td>62.4 y. (40–85 y)</td>
<td>11 m. (4–7 y)</td>
<td>63.1 y. (30–77 y)</td>
<td>4 m. (hrs–1 y)</td>
<td>62.4 y. (40–85 y)</td>
<td>5.5 m. (hr–5 y)</td>
<td>65.2 y</td>
</tr>
<tr>
<td>(49 cases)</td>
<td>34 pts.</td>
<td>21 pts.</td>
<td>14 pts.</td>
<td>49 pts.</td>
<td>21 pts.</td>
<td>33 pts.</td>
<td></td>
</tr>
<tr>
<td>Coronary artery disease nonacute</td>
<td>63.5 y. (40–84 y)</td>
<td>31 m. (1–18 m)</td>
<td>63.0 y. (42–85 y)</td>
<td>39 m. (1–15 y)</td>
<td>63.5 y. (40–84 y)</td>
<td>29.4 m. (1–18 y)</td>
<td>65.9 y</td>
</tr>
<tr>
<td>(58 cases)</td>
<td>40 pts.</td>
<td>42 pts.</td>
<td>28 pts.</td>
<td>58 pts.</td>
<td>35 pts.</td>
<td>41 pts.</td>
<td></td>
</tr>
<tr>
<td>Hypertensive heart disease</td>
<td>63.4 y. (19–82 y)</td>
<td>28.5 m. (7–9 y)</td>
<td>61.0 y. (42–82 y)</td>
<td>57 m. (15–11 y)</td>
<td>63.1 y. (19–82 y)</td>
<td>29.6 m. (1–9 y)</td>
<td>60.3 y</td>
</tr>
<tr>
<td>(62 cases)</td>
<td>27 pts.</td>
<td>42 pts.</td>
<td>19 pts.</td>
<td>62 pts.</td>
<td>28 pts.</td>
<td>29 pts.</td>
<td></td>
</tr>
<tr>
<td>Rheumatic heart disease</td>
<td>52.5 y. (16–79 y)</td>
<td>14.5 m. (1–7 y)</td>
<td>56.3 y. (42–66 y)</td>
<td>62.1 m. (1–8–5 y)</td>
<td>52.0 y. (16–79 y)</td>
<td>40.7 m. (12–7 y)</td>
<td>62.7 y</td>
</tr>
<tr>
<td>(21 cases)</td>
<td>11 pts.</td>
<td>8 pts.</td>
<td>5 pts.</td>
<td>21 pts.</td>
<td>10 pts.</td>
<td>11 pts.</td>
<td></td>
</tr>
<tr>
<td>Etiology undetermined</td>
<td>44.5 y. (22–78 y)</td>
<td>6.4 m. (6–1 y)</td>
<td>40.8 y. (22–78 y)</td>
<td>7.3 m. (1–13 m)</td>
<td>43.5 y. (22–78 y)</td>
<td>5.6 m. (6–1 y)</td>
<td>55.0 y</td>
</tr>
<tr>
<td>(18 cases)</td>
<td>9 pts.</td>
<td>3 pts.</td>
<td>18 pts.</td>
<td>4 pts.</td>
<td>5 pts.</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Miscellaneous</td>
<td>44.9 y. (12–82 y)</td>
<td>68 m. (5–24 y)</td>
<td>60.3 y. (32–82 y)</td>
<td>16.2 m. (5–2 y)</td>
<td>44.9 y. (10–82 y)</td>
<td>84.8 m. (5–24 y)</td>
<td>55.0 y</td>
</tr>
<tr>
<td>(16 cases)</td>
<td>5 pts.</td>
<td>10 pts.</td>
<td>4 pts.</td>
<td>16 pts.</td>
<td>4 pts.</td>
<td>7 pts.</td>
<td></td>
</tr>
<tr>
<td>Average</td>
<td>59.2 y.</td>
<td>25.3 m. (4–24 y)</td>
<td>60.2 y. (22–82 y)</td>
<td>35.2 m. (24–11 y)</td>
<td>59.2 y. (10–85 y)</td>
<td>26.2 m. (8–24 y)</td>
<td>63.2 y</td>
</tr>
<tr>
<td>(224 cases)</td>
<td>132 pts.</td>
<td>73 pts.</td>
<td>224 pts.</td>
<td>104 pts.</td>
<td>126 pts.</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

h = hours, d = days, m = months, y = years.
age 26.2 months). When these major groups were compared it was found that the length of life after the first syncope or heart block was greatest in the rheumatic, least in the coronary, and intermediate in the hypertensive group.

**Diphtheria**

This review throws further light on the concept that was presented over 25 years ago, and which has since been discussed, that diphtheria plays some role in the development of complete heart block.8 There were 41 (16 per cent) cases in which there was a past history of diphtheria; this seems to us a great deal higher than the incidence in the general population during the time that this group of patients was studied. The exact mechanism is difficult to evaluate, but increased vulnerability to conduction defects apparently exists after infection with diphtheria. A long lapse of time may intervene between the actual infection and occurrence of the complete heart block, perhaps at a time when further stress, insult, or injury is encountered. In regard to diphtheria, it will no doubt play less of a role in the future with decreasing incidence of the infection. However, we would like to emphasize that there is reason to believe that some of the unexplained cases of complete heart block may be due to previous apparently minor infections from which the patient recovers, as a result of which the patient is left more vulnerable, just as occurs after recovery from diphtheria. Among the 18 patients with heart block due to unknown cause, there were 7 who gave a history of previous diphtheria, whereas in all the remaining 206 cases, there were only 33 cases of diphtheria. The distribution of the 41 cases of diphtheria was as follows: coronary artery disease, nonacute group, 13 of 58 patients; acute myocardial infarction group, 2 of 49 patients; hypertensive heart disease group, 11 of 62; rheumatic heart disease group, 4 of 21; miscellaneous group, 3 of 16; etiology undetermined group, 7 of 18; digitalis intoxication group, 1 of 27 patients.

It seems to be more than a matter of coincidence that a past history of diphtheria should be so much more prevalent among those cases in which there is no known cause of heart disease than among those in which definite etiologic factors such as rheumatic heart disease or coronary artery disease are involved. This adds validity to the concept that early diphtheria was related to the subsequent development of heart block.

**Seroology**

There was a total of 9 cases who showed a positive serologic reaction for syphilis. Cases were not considered to have complete heart block on a syphilitic basis unless there was some other evidence of syphilitic heart disease. There were 2 such cases included in the miscellaneous group.

**Gallbladder Disease**

Because of some suspicion of the relationship between gallbladder disease and Adams-Stokes attacks, its occurrence was investigated. Unfortunately, until very recent years no systematic search for gallbladder disease was made in these cases. Not infrequently, one discovers the presence of gallstones on routine x-ray examination, when there has been very little if any clinical evidence of biliary disease. However, there were 26 patients who demonstrated evidence of cholelithiasis or cholecystitis during the time that they were known to have had complete heart block. Eleven of 58 patients with nonacute coronary artery disease were found to have gallbladder disease as well as 3 of 49 patients in the acute myocardial infarction group, 10 of 62 in the hypertensive heart disease group, 1 of 21 in the rheumatic heart disease group, and 1 of 18 in the etiology undetermined group. Gallbladder disease occurred in no patients in the miscellaneous or digitalis group.

Most instances of gallbladder disease were detected during the past 10 years when interest was aroused concerning the relationship of gallbladder disease and syncopal attacks.9 In 6 instances a cholecystectomy was performed, and in 4 of these there was striking improvement in the incidence of Adams-Stokes attacks. There was no operative mortality in this small group.
Operative Risk

There were 19 patients who underwent 24 surgical procedures. The following operations were performed with no surgical mortality; cholecystectomy in 6, hysterectomy and salpingo-oophorectomy in 1, spinal fusion in 1, tonsillectomy in 1, thyroideectomy in 2, amputation of toes in 2, anal fistula in 1, hemorrhoidectomy in 1, low-thigh amputation in 1, hernia repair in 1, abdomino-perineal resection for adenocarcinoma of the sigmoid in 1, appendectomy in 1, and multiple pregnancies in 2 patients, one of whom also had an appendectomy and removal of left ovarian cyst subsequent to her pregnancies.

Anesthetics employed in these procedures included ether, nitrous oxide, intravenous Pentothal, spinal anesthesia with procaine, local infiltration with procaine, and field block with xylocaine. It might be pointed out that in the administration of a local anesthetic such as procaine and its derivatives in amounts evoking a systemic effect, a myocardial depressant action might ensue. In general, intravenous procaine is contraindicated in complete heart block.10

Two of the patients undergoing cholecystectomy had multiple episodes of asystole during anesthesia and surgery, but with careful observation and management both survived the procedures and are still living.

Complete heart block is not a contraindication for necessary surgical procedures or for pregnancy. It would appear that in anesthesia it is not the anesthetic agent employed during the surgery but the manner and method of administration of this that may determine the success of the procedure.

Follow-up Studies and End Results

Of the 224 patients included in this study, 127 were known to have died, 36 were reported to be living, and 61 did not respond to attempted follow-up. The modes of death in the 127 cases were as follows: sudden death, 39; progressive heart failure, 10; acute coronary occlusion, 22; miscellaneous causes, 18, which included deaths due to uremia, pulmonary infarction, pneumonia, cerebral hemorrhage, and other causes; the actual cause of death was unknown in 38 cases.

One hundred thirty-seven of these cases had syncope and 87 did not. In the former group, 76 were known to have died and in 53 the type of death was also known. Thirty-one deaths occurred suddenly. Among the 87 cases who did not have syncope, 51 were known to have died and the manner of death was known in 36 instances. Only 8 of these died suddenly. It would appear that instant death occurs about 3 times as frequently in those who have syncope as in those who do not. However, sudden death takes place not only because of the conduction defect but also in a manner common to the various types of heart disease.

Coronary Artery Disease

Patients with coronary artery disease constituted the largest of all groups studied (107 cases). Of these, 63 had definite myocardial infarction, about half of which also had clear-cut anginal episodes as well. An additional 44 had angina pectoris without clear-cut evidence of myocardial infarction.

It is of interest that among the cases that were most carefully observed, there were none with anginal manifestations after the development of complete heart block, though many had this symptom before the block appeared. With such a large number of cases having coronary artery disease, it was impressive how rare angina was in those who had a slow ventricular rate.

Males predominated over females, 65 over 42, respectively, as would be expected in any study of coronary artery disease. The age of the patients in this group ranged from 40 to 85 with an average of 64 years.

The various symptoms such as syncope, dyspnea, and palpitation occurred in patients in the coronary artery disease group with about the same frequency as patients in the other groups of the study. The average blood pressure was 176 mm. Hg systolic and 76 mm. Hg diastolic. For the most part, the other clinical features, such as heart rate, heart size, congestive failure, and various arrhythmias, were not significantly different in patients with
coronary artery disease than in patients in the other groups. However, episodes of paroxysmal ventricular acceleration were recorded electrocardiographically in 10 of 107 patients with coronary artery disease in contrast to only 4 of the remaining 117 patients in this study. There was also a difference in the incidence of gallstones in this study (14 in 107 patients in the coronary group, 10 in 62 in the hypertensive group, 2 in the other 55 patients). To be sure, the average age of the coronary and hypertensive heart disease patients was about 10 years greater than that of the others.

Cases of acute coronary thrombosis made up a distinct group with certain peculiar characteristics. Here it is quite clear that the heart block was precipitated by a specific event. In most of the other types of complete heart block, there was no direct cause either for syncopal attacks or for the initial development of the conduction defect, the process being insidious in nature. There was a total of 49 patients with acute myocardial infarction who showed complete heart block. In 2 of these, the block preceded the coronary attack and, in all the others, complete heart block developed during the early days of the acute infarction. It is of interest that heart block occurs about twice as frequently with posterior as with anterior infarctions.

In those cases of acute coronary occlusion in which complete heart block occurred, the immediate mortality was significantly high. Of the 49 cases, 21 died in the brief period of a few days (4 hours to 18 days, average 5.5 days). Of these 21, 6 had had a previous coronary occlusion. Of the 28 who survived the immediate coronary insult, it is of interest that 23 returned to their previous atrial rhythm, 21 to normal sinus rhythm and 2 to atrial fibrillation. Five patients continued in complete heart block; 2 of these had previously had complete heart block, 1 had previously had atrial fibrillation, and the other 2 were undocumented as to their previous rhythm. There was one instance in which the patient had an acute coronary episode with complete heart block and recovered with the return of a normal sinus rhythm. Sometime later he had a second myocardial infarction with a stormy course but survived and maintained a normal sinus rhythm throughout. Of these 28 who survived their acute coronary occlusion, 5 had had a previous similar episode.

It is very impressive that there was not a single instance in which it was known that the patient previously had a normal sinus rhythm and then developed complete heart block with an acute infarction and thereafter continued in complete heart block. This observation corresponds with the general cardiological experience in thousands of cases of acute coronary thrombosis, for it is extremely rare to see permanent complete heart block result as a direct sequel of an acute attack of myocardial infarction. Permanent complete heart block, as shown in this study, must be an extremely rare immediate sequel of acute myocardial infarction. This is of interest when it is appreciated that about 2.5 per cent of cases of acute myocardial infarction do develop temporary complete heart block.11

Another peculiarity of complete heart block occurring with acute myocardial infarction is that the ventricular rate is not infrequently more rapid than is customarily seen in other types of third degree heart block. Whereas the average ventricular rate of all the other cases in this study was 36, the average rate of heart block when it occurs with acute myocardial infarction was 43. Although slow rates of 28 to 35 did occur, there were 8 in which complete atrioventricular dissociation was present with ventricular rates of 50 to 60; in fact there were 2 with ventricular rates over 70, 1 with a rate of 75 and another with a rate of 97. In all these cases there was electrocardiographic confirmation that the atria and ventricles were contracting independently of each other.

**Hypertensive Heart Disease**

The average age, sex distribution, incidence of syncope and dizziness, dyspnea, and palpitation were not strikingly different in this group of 62 cases with hypertension from the over-all distribution. The pulse pressure average for this group was 117, whereas the average for the remainder was 81. As would be expected because of the classification of these cases, the systolic and diastolic blood pressures were
significantly higher. For the most part there were only slight differences in the incidence of other features, some of which are presented in table 7.

Systolic hypertension was a general feature of complete heart block and the diastolic pressure was, as an over-all average, within normal limits. Thus, systolic hypertension might bear some relation to the fact that the average age of all patients studied was 59.3 years. It is not necessary that hypertension be present in every patient having complete heart block. A patient may be quite fit, with a slow heart rate of 30 to 35, and still have the systolic not over 130 mm. Hg. In one patient in this study the systolic pressure was as low as 110 mm. Hg. It is of interest that, in general, there was a fall of systolic and diastolic blood pressure as well as pulse pressure with heart rates over 40. However, in the hypertensive group the heart rate had no effect on the systolic or diastolic pressure levels.

The length of life after the first syncopal attack, in general, was longer in the hypertensive patients than in patients in the coronary artery disease group, and the age at death was about 5 years less than in this latter group.

It would appear that although hypertension is a not infrequent accompaniment of complete heart block, it is not a necessary part of the condition, and, when present, has only a slight effect on the various features and complication of heart block.

Rheumatic Heart Disease

The average age of patients in the rheumatic heart disease group (21 cases) was 52.5 years, in comparison with an average age of 59.8 years for the other 203 patients. In the 21 patients in this group, 13 were females and 9 were males, which is consistent with the general predominance of rheumatic mitral stenosis in females. Syncope occurred in 39 per cent of the rheumatic patients in contrast to 60 per cent of the remaining groups. Palpitation occurred in 57 per cent as compared with 15 per cent in the remaining 203 patients; atrial fibrillation occurred in 8 of the 21 rheumatic patients (35 per cent) in contrast to only 24 of the 203 patients (12 per cent) in the other groups.

There were differences in the occurrence of some of the clinical features in patients in the rheumatic group as compared with all the others; syncope 39 per cent and 60 per cent, palpitation 57 per cent and 15 per cent, atrial fibrillation 35 per cent and 12 per cent, past history of rheumatic fever 66 per cent and 1 per cent, bundle branch block 54 per cent and 59 per cent. The average ventricular rate in the rheumatic group was 45, while in the others it was 37; the length of life after the onset of syncope was a good deal longer in the rheumatic group, 5.2 years in contrast to 2.8 years. The length of life after the onset of complete heart block was 3.2 years as compared to 2.4 years. The average age at death was 62.7 years while in the others it was 63.2 years.

There were 12 patients with mitral valve disease, 4 with aortic valve disease, and 5 with both. Complete heart block was not common in rheumatic heart disease in general, for there were only 21 such cases out of the many hundreds of valvular cases that formed the background for this study. When it did occur, however, it was a little surprising that mitral stenosis or some involvement of the mitral

<table>
<thead>
<tr>
<th>Table 7.—Miscellaneous Features in Complete Heart Block</th>
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<tr>
<td></td>
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<tr>
<td>No. Cases</td>
</tr>
<tr>
<td>Acute coronary occlusion</td>
</tr>
<tr>
<td>Coronary artery disease (nonacute)</td>
</tr>
<tr>
<td>Hypertensive heart disease</td>
</tr>
<tr>
<td>Rheumatic heart disease</td>
</tr>
<tr>
<td>Etiology undetermined</td>
</tr>
<tr>
<td>Misc. cases</td>
</tr>
<tr>
<td>Total</td>
</tr>
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valve was present more frequently than aortic valvular disease. It is of interest that complete heart block associated with acute rheumatic myocarditis is rare, none having been observed in this study. A case of transient complete heart block associated with an acute rheumatic flare-up was recently called to our attention.12

It is obvious that there should be a much greater frequency of a past history of rheumatic fever and a greater incidence of atrial fibrillation in the rheumatic group than in the others. It is also of interest that the patients with rheumatic heart disease had a higher ventricular rate and lived longer after the onset of syncope than other patients with complete heart block. Although in this study the patients with rheumatic heart disease were younger than those in the other groups, they were still a good deal older than the average patient with rheumatic valvular disease, from which one could infer that heart block in these rheumatic cases required many years for its development.

"ETIOLOGY UNDETERMINED"

There were 18 patients with complete heart block in whom no definite etiologic diagnosis could be made. Possible causes that might be invoked were an infection early in life accompanied by a transient myocarditis, a congenital cardiac defect of conduction unknown to the patient or family, trauma to the heart at a previous occasion, a toxic myocarditis, allergic or some other cause, or some specific response of the conduction tissue to noxious agents.

Nine were males and 9 were females. The average age was 44.5 years, the youngest age group. Syncope occurred in 8 of the 18 patients (44 per cent) and in 122 of the 206 remaining patients (59 per cent). Seven of the 18 patients (39 per cent) in this group had diphtheria, whereas the over-all figure was 33 in 206 patients (16 per cent). This makes it very likely that early diphtheria was related to the later development of heart block in some of these cases.

It is of interest that in this group without coronary artery disease, valvular disease, or hypertension, the average blood pressure was 132/67 mm. Hg (pulse pressure of 65) whereas the average blood pressure in the others was 171/78 mm. Hg (pulse pressure of 93). Thus it is pertinent to point out again that hypertensive levels are not a necessary concomitant part of complete heart block. Alteration of heart rates below and above 40 beats per minute had very little effect on comparative systolic and diastolic blood pressures. Atrial fibrillation was present in 4 of the 18 patients.

The age at onset of syncope, the age at onset of complete heart block, and the duration of life after the development of complete heart block in the two groups, namely those with etiology undetermined and all others in this study, were as follows: 40.8 years and 61.5 years, 53.5 years and 60.6 years, and 5.6 months (4 patients) and 27 months (100 patients), respectively. The average age of death was 35.0 years (5 patients) as compared with 63.5 years (121 patients). The youngest patient in this entire study having syncope was 22 years of age and was among this subgroup “etiologic undetermined.”

This group obviously contained cases of complete heart block without major organic cardiac defects, such as valvular or coronary artery disease. Here the heart block appears in a purer form. It is of interest that such cases may be normotensive, are younger, and have slightly higher ventricular rates. The greater incidence of a past history of diphtheria is also of significance and lends interest to the concept that an early infection may be conducive to the later development of complete heart block. Finally, it is our distinct impression that patients in this group have a better prognosis as far as the duration of life is concerned. Although there were only 4 cases that were known to have died after an average interval of only 5.6 months after the first onset of complete heart block, there are at least 3 others who are still alive and well 15, 10, and 9 years after the complete heart block was first observed.

MISCELLANEOUS CASES

There were 16 patients in this study in which there were unusual types of heart disease associated with complete heart block. For purposes of description we have further sub-
divided this group into those (a) with complete heart block produced by pressure on the carotid sinus, (b) with complete heart block due to syphilitic heart disease, and (c) other cases with rare etiologic causes of complete heart block.

**Carotid Sinus Group.** There were 6 cases in this group. These six did not have complete heart block prior to carotid sinus stimulation. Two had normal sinus rhythm and 4 had second degree atrioventricular block. Both of the first 2 and 2 of the latter 4 had ventricular asystole during carotid sinus stimulation at a time when the atria were contracting though a little more slowly than before. In all instances the rhythm returned to the state that preceded the carotid sinus test. Two of the cases that showed partial block manifested transient complete atrioventricular dissociation during the test. A follow-up observation in these 6 cases revealed that 2 subsequently had transient complete heart block, 2 developed permanent complete heart block, and 1 still showed the same partial heart block that was previously present 4 years later; in the subsequent course was not ascertained.

One of the 6 cases presented some interesting features. This patient was a physician 46 years of age. He had had previous syncope and also complained of dizziness. During carotid sinus stimulation ventricular asystole occurred and the atrial rate was little disturbed; he also had similar spontaneous episodes with the return to normal sinus rhythm. At other times he developed brief episodes of spontaneous complete atrioventricular dissociation. During this time following a brief exercise test the atrial rate would accelerate and independent ventricular rhythm would result temporarily; as the atrial rate returned to the pre-exercise level, normal sinus rhythm would return. Finally, two years later, permanent stable complete heart block developed and has persisted for some years. He is at present quite fit and carrying on an active practice.

It would appear that in patients who have had syncope, if carotid sinus stimulation results in temporary ventricular asystole with continuation of atrial activity or in transient complete atrioventricular dissociation, the subsequent development of complete heart block is likely to occur.

**Syphilitic Heart Disease.** There were only 2 cases of complete heart block in this group. Both were males and both had syncope, enlarged hearts, and bundle branch block. One had a blood pressure of 205/50 and had congestive heart failure, with a ventricular rate of 51 beats per minute. The other had a blood pressure of 140/55 and a heart rate of 20 but did not have congestive heart failure. Post-mortem examination of the former patient showed a gumma in the region of the septum. Inasmuch as syphilitic heart disease in general is becoming increasingly infrequent, this unusual form of heart block is likely to become extremely rare in the future.

**Rare Etiologic Causes of Complete Heart Block.** Because of the peculiar nature of each of these cases, a brief description follows:

**Case Reports**

**Case 1.** J.A., a 21-year-old man who, after having an infection of the foot, developed partial block and then permanent complete heart block; he had no syncope. He showed a blood pressure of 115/60, a heart rate of 32, and left bundle branch block. He lived for 24 years after the onset of complete heart block. Inasmuch as there are rare instances of localized pyogenic abscesses in the heart muscle, it is not unlikely that such an infection occurred in this case, which healed and resulted in a permanent complete heart block.

**Case 2.** L.S., a 28-year-old man who had been told that he had a slow heart during an attack of diphtheria as a child; it was not known what his rate was during the remaining years of childhood but from the ages of 18 to 28 he knew that his heart was slow. When examined at the age of 28 he had complete heart block and a ventricular rate of 44; the blood pressure was 115/60. He had never had syncope. The relationship of diphtheria and complete heart block has been discussed in greater detail earlier in this paper.

**Case 3.** J.V., an 82-year-old man, was a patient who had syncope three weeks prior to admission. His blood pressure was 160/90 and the heart rate was 37. He had a calcified mitral annulus by x-ray examination. Such calcification as a concomitant of heart block is probably not infrequent.

**Case 4.** J.D., a 12-year-old boy, was a patient in whom heart block had been noted at the age of two weeks and had subsequent heart rates varying from 53 to 135 during the 12 years prior to entry. He had
a murmur suggestive of ventricular septal defect. His blood pressure was 125/75 and the heart rate when last seen was 47. He has had no syncope. He is now 33 years old and employed as a laborer. This probably is an instance of congenital heart block.

Case 5. R.K., a 10 year old boy, was a patient who was born with a slow heart and a "leak valve," had pneumonia two weeks prior to admission, and had an irregular pulse with the rate of 21; the first heart sound varied in intensity. Years later he had a cerebral episode at the age of 27 with a heart rate reported as 45 beats per minute. This is another instance of congenital heart block.

Case 6. M.C., a 28 year old woman, had had no syncope but had had renal shutdown after trauma and hematuria followed by bilateral staghorn calculi. She developed oliguria with no benefit from nephrostomy, and later had uremia. She developed complete heart block during the last six days of life. Post-mortem findings included deposits of calcium crystals in the myocardium. One may speculate that the calcium deposits in the heart muscle may have impaired conduction in the A-V node or bundle of His.

Case 7. C.R., a 50 year old man, for three weeks prior to admission had had a chest cold, lassitude, generalized aching, chills, fever, and night sweats. Three days after admission he developed complete heart block and syncope and died 11 days later. Post-mortem findings revealed vegetations in the right ventricle and a necrotic cavity in the interventricular septum extending to the bundle of His.

Case 8. H.T., a 35 year old man, had experienced blurred vision and syncope four years prior to admission and for two months prior to admission had had recurrent syncope. This patient had a precordial systolic murmur and was regarded as having probable congenital interventricular septal defect.

A review of the above 8 cases would indicate that complete heart block may result on rare occasions from a variety of causes. Unusual mechanisms should therefore be sought for in any case of complete heart block where the more common causes, such as hypertensive heart disease, valvular disease, or coronary artery disease, are lacking.

Digitalis Intoxication

In addition to nausea, vomiting, and yellow vision, it is well known that cardiac arrhythmias are manifestations of overdosage of digitalis and its glycosides. In this study there were 27 cases of complete heart block due to digitalis. There were 15 males and 12 females; the average age of patients in this group was 52 years with a range of 13 years to 80 years. Those in the digitalis group represent 11 per cent of all patients in this study. Two patients had hypertensive heart disease, 4 had myocarditis of undetermined cause, and of the 21 remaining, 17 had rheumatic valvular disease and 4 had a history of rheumatic fever without valvular disease. In the 27 patients placed in this group, there was an obvious relation between digitalis administration and complete heart block. Therefore, the block was of a transient or intermittent type in 17 of the 21 patients in this group who died with complete heart block. Features such as palpitation, blood pressure (the average was 148/78), heart size, and partial heart block were no different in this group than in the others. It was of interest that syncope did not occur in any patient during the period of complete heart block due to digitalis intoxication. This is unusual in that syncope occurred in 137 (61 per cent) of the other 224 cases and was about equally frequent in each of the various groups. The heart rate was higher in this group (54 beats per minute) than in any other group (over-all average 38 beats per minute). The range of the heart rate was 32 to 98 beats per minute. Atrial fibrillation was present in 17 patients (63 per cent) in contrast to 43 of the other 224 patients (19 per cent). This would be expected because of the nature of the cases making up this group. Other arrhythmias were present prior to the development of complete block. Ventricular premature beats were recorded in only 4 cases and transient ventricular fibrillation in 1 case. Bundle branch block was noted in 4 of the 27 cases (15 per cent) but occurred in 122 of the other 224 cases (55 per cent). All the patients in this group received digitalis because of congestive heart failure. Five patients had received intravenous strophanthrin during hospital admissions prior to 1920.

In general, it can be stated that excessive doses of digitalis are occasional causes of complete heart block. One can add that it is a very rare occurrence considering the fact that so few of the thousands of cardiac patients who received digitalis therapy in this hospital developed complete heart block. The impression
is gained that this complication arises in very sick patients who are not responding to medication, and in whom one is tempted to push the drug in a last hope of improving the circulation. In these patients with complete heart block the ventricular rate is apt to be more rapid than in other patients with complete heart block and syncope is extremely rare. The block will disappear when the drug is discontinued and the patient survives. This group differs from some of the other groups in that bundle branch block is less common and a past history of diphtheria is very rare (1 out of 27).

**TREATMENT**

Before embarking on the therapy for complete heart block, one naturally would prefer to have the diagnosis well established. Actual recognition of complete heart block, if it persists, is a fairly simple matter. A more difficult problem is presented when patients have fainting spells of short duration, but when seen by the physician, show little or no evidence of conduction defect. They may then be readily confused with, or actually misdiagnosed as, cases of hysteria, major or minor epilepsy, or other disease entities. Occasionally no satisfactory diagnosis can be made until the case is carefully observed during an actual spell.

Treatment of complete heart block consists of two parts. The first and generally the most urgent is the treatment, immediate or preventive, of the syncopal attacks. The second is the treatment or the management of the more static slow heart rate that generally accompanies this condition. When syncopal attacks come rarely, i.e., at intervals of many months or years, it is obviously difficult to outline a program of therapy to prevent recurrences. One will have to administer a specific medication or other mode of therapy daily and constantly for a very long time before one can be convinced that attacks are being prevented. However, if the attacks recur frequently, every few minutes, hours, or days, one can quickly determine whether or not any given procedure is effective. One can readily see, therefore, that under certain circumstances heroic measures might be needed when syncopal seizures appear to be imminent and critical, while at other times no constant therapy might be indicated.

A further therapeutic consideration is the set of circumstances under which heart block with or without periods of asystole may arise. The therapy of a case of Adams-Stokes disease occurring acutely in the midst of an attack of acute myocardial infarction with accompanying prolonged shock may demand one method of treatment while similar asystole coming as an isolated event in a patient with well compensated hypertensive heart disease, or with no other important cardiac disability, might demand an entirely different therapeutic approach.

Before undertaking the type of therapy for an attack of syncope, it would be very helpful if we could establish the exact physiologic nature of the episode. As has been discussed before, in general, there are two possible mechanisms involved: (1) a sudden ventricular arrest not preceded by any ventricular irritability and (2) a condition in which the initial events are ventricular extrasystoles or ventricular tachycardia ending in ventricular flutter or fibrillation. Electric inactivity with the arrest of the heart in cases showing the latter disturbance may take place after a few premature beats or after a more lengthy interval of ventricular irritability and acceleration. It is generally impossible to distinguish one type from the other because electrocardiograms are rarely available the moment these sudden events occur. However, one may suspect that ventricular acceleration precipitates attacks if it is possible to obtain evidence that ventricular extrasystoles or tachycardia have been occurring during the intervals between major episodes. Likewise, the detection of short pauses without ventricular acceleration at different times would favor the first of the two mechanisms as a cause.

**Therapy for the Acute Attack.** Sympathomimetic amines have been regarded as having the most potent effect on the rhythmic function of the heart. In complete heart block our attention is focused on the fundamental properties of the heart, particularly those of rhythm and conduction. Sympathomimetic amines such as epinephrine, N-isopropyl-
norepinephrine (Isuprel), and ephedrine appear to have the effect of stimulating the rhythmic function of the heart. Other sympathomimetic amines such as Neosynephrine, norepinephrine, methoxamine (Vasoxyl), and Wyamine have little or no effect on increasing the rhythmic function of the human heart and, therefore, serve little or no purpose in increasing the heart rate in complete heart block.

For the acute attack of asystole when heroic measures are indicated, epinephrine 0.3 ml. to 0.5 ml. of 1:1000 aqueous solution should be administered by intracardiac injection if the circulation has ceased; this is imperative as standoff affords no means of transportation of the medication to the coronary arteries and thence to the rhythmic foci of the heart itself. If the peripheral circulation is effective, then the epinephrine can be administered parenterally. This may be repeated at frequent intervals as the apparent duration of the accelerating effect is about 8 to 15 minutes. Constant intravenous drip may also be effectively employed. Subcutaneous doses of 0.5 ml. of 1:1000 aqueous solution or 0.5 to 1.0 ml. of 1:500 epinephrine in oil solution intramuscularly are also effective. The time interval of administration is dependent upon the degree of responsiveness; one patient may require an injection every two hours while another patient may require an injection every hour.

More recently isoproterenol (isopropyl-norepinephrine, Isuprel) has been introduced and found to be effective in increasing the rhythmic function of the heart.\textsuperscript{15, 16} This may be administered in doses of 0.02 mg. intravenously or by intracardiac injection. It may be repeated every 10 to 15 minutes. The subcutaneous dose is 0.2 mg. every 40 to 60 minutes. A constant intravenous infusion at the rate of 2 to 6 \textmu g./min. has been administered. Constant attention must be given to the cardiac mechanism as the infusion should be titrated to the desired heart rate and should be discontinued if the patient unblocks and the normally conducted impulse is initiated.

On recent theoretic grounds it is possible that adrenal steroids may be of value in heart block. The rationale for such therapy is based on the following observations.\textsuperscript{19} In a group of 50 patients with Addison's disease the mean P-R interval measured 0.18 second, and 20 per cent exhibited heart block during some phase of their disease. In a group of 34 patients with Cushing's syndrome, the mean P-R interval duration was 0.14 second and A-V conduction was consistently shortened when the underlying disease was adrenal hyperplasia. The abbreviation of the P-R interval appeared to correlate with the urinary 17-ketosteroid excretion. Administration of cortisone to patients with Addison's disease and resection of the hyperplastic adrenal gland in patients with Cushing's syndrome respectively shortened and lengthened the P-R interval. When cortisone is given to patients without endocrine disorders, there is likewise a decrease in this interval. It may be that adrenal steroids participate in facilitating A-V conduction.

In the acute attack, irritation of the heart with a direct needle prick may stimulate systole. The external pacemaker may also serve a useful purpose\textsuperscript{17} and direct massage of the heart can be a lifesaving procedure. Recently, sodium lactate intravenously has been suggested as useful in therapy.\textsuperscript{18}

In patients having Adams-Stokes attacks associated with acute myocardial infarction, the shock state is an added threat. The practical importance of maintaining a pacemaker as well as the peripheral circulation is obvious. Theoretic considerations and data accumulated thus far would lend support to the belief that the cardiogenic factor rather than the peripheral vascular factor is the important feature of preventing the shock state. Intravenous isoproterenol not only increases the rhythmic function but also increases the peripheral systolic and diastolic blood pressures, if shock has been previously present.

If the acute attack is identified as ventricular acceleration it would seem that certain cardiac depressants might be preferred, but the effects are not altogether clear. It is a common experience that in patients who have ventricular tachycardia or ventricular fibrillation but do not have complete heart block, procaine amide and quinidine can be beneficial and epinephrine harmful. But the situation is not so simple when the patient has complete heart block. Here the
same sequence of events does not prevail as when complete heart block is not present, and it would seem that procaine amide, quinidine, and possibly potassium should not be used.\textsuperscript{19, 20}

If the acute attacks occur and one is unable to identify their mechanism, the recommended course to be followed is one in which asystole is assumed to be the underlying cause.

\textit{Therapy for Prevention of Subsequent Attacks.} The administration of therapeutic agents will depend upon the frequency of the attacks. If the attacks are rare and very infrequent, perhaps no therapy is indicated. However, if they are frequent, sublingual isoproterenol in doses of 15 to 30 mg. every four hours or ephedrine sulfate, 25 to 50 mg. 4 times daily may be of value. Occasionally barium chloride, 30 mg. in 4.0 ml. solution 4 times daily, will be found useful. Aqueous epinephrine, 1:100 as a nebulizer spray; epinephrine in oil, 1:500 intramuscularly once or twice daily; isoproterenol, 1:200 as a nebulizer spray, or atropine in isolated circumstances may all be measures worthy of trial. When the heart rate is slow, 18 to 22 beats per minute, and the patient is in a semistupor, any possible means of increasing the rate should be employed.

Thyroid gland has been recommended for complete heart block, but its value is uncertain. Ammonium chloride by its acidifying effect may theoretically be beneficial. In 1 patient who had syncopal attacks in which the heart would stop regularly on holding a deep inspiration, the condition was immediately controlled on large doses of ammonium chloride. This patient also had occasional spontaneous attacks that did not appear to be related to respiration. In this series, one patient received 20 mg. of corticotrophin over a 12 hour period intravenously and the heart rate decreased, the patient appeared critical, pale, moist, and weak. More complete evaluation of the role and effect of adrenal steroids is necessary. We believe strychnine, Metrazol, picrotoxin, Coramine, and caffeine are of little or no value in the treatment of Adams-Stokes disease. (If complete heart block is a result of quinidine therapy, however, caffeine, oxygen, and artificial respiration would be indicated.)

An attempt was made to evaluate the role of cardiac depressants such as quinidine, Pronestyl (procaine amide), and potassium in ventricular acceleration—ventricular tachycardia, ventricular flutter, ventricular fibrillation. It has been thought that these compounds might be of value in Adams-Stokes attacks when these abnormal ventricular rhythms are present. These drugs suppress the rhythm function of the heart but prolong conduction and suppress myocardial contractility. Paradoxically, the administration of procaine amide and quinidine may actually result in the production of ventricular accelerated rhythms when given to patients with complete heart block. Although it is not simple and clear-cut, these drugs should probably not be employed at any time if complete heart block is present.

In those patients who have complete heart block and in whom congestive heart failure develops, digitalis therapy should be employed with somewhat more careful supervision. Other usual treatment for decompensation such as ammonium chloride, aminophyllin, and diuretics should be used as necessary. It is not altogether clear whether digitalis has a beneficial effect in those patients who do not have objective signs of failure. It is controversial whether there is any value in trying to slow the atrial rate so as to enhance a 2:1 partial block in patients having complete heart block.

\textbf{Discussion}

There is some vagueness concerning the definition of Adams-Stokes disease. By some it is confined only to those who have a slow ventricular rate, the atria beating independently and more rapidly, and who are also subject to attacks of syncope. Others will include patients with syncopal attacks who display periods of asystole but at other times do not show atrioventricular dissociation. Heart block of varying degree may be present in such cases during the short periods of the major episodes, but with recovery, little evidence of conduction defect such as P-R interval of 0.22 second or incomplete bundle branch block, may remain. In this present review we have included all cases that had complete atrioventricular dissociation with an independent ventricular rate slower than the atrial whether the atria
were contracting or fibrillating, and also whether or not there had been syncopal attacks.

It is evident that complete heart block occurs in a great variety of conditions; in the young and in the old, with valvular or non-valvular disease, with or without hypertension, in a transient or permanent form, as a direct result of excessive digitalis administration, or as a consequence of a great variety of etiologic causes. The most common cause is coronary artery disease with resultant myocardial fibrosis. There is reason to believe that some otherwise benign infections, from which patients apparently make a complete recovery, can be responsible for the development of heart block many years later in some instances. In such cases a transient myocarditis may have involved the conduction apparatus, only to manifest its dire consequences many years later. Among these early infectious diphtheria formerly was a prominent one. Many other simple infections such as measles, mumps, scrub typhus, undulant fever, infectious mononucleosis, poliomyelitis, and others may also be in this category. It is difficult to have pathologic proof of this because these patients obviously recover from the early infection. Indirect evidence to support this view would be afforded if some of these rare cases of transient conduction defects occurring in benign infections were followed and years later found to have heart block. We know of no such fortuitous recorded cases. An example of a well-documented fatal form of myocarditis of undetermined origin that did manifest complete heart block with syncopal attacks has been published.21 If such a case had recovered and eventually developed Adams-Stokes attacks, this would have been proof of the above concept.

The clinical picture presented by patients with complete heart block also varies greatly from case to case. There are instances where syncope never occurs and the patient feels fit and able to carry on normal or even strenuous activities. In others, syncopal attacks may be frequent, occasionally presenting the picture of status epilepticus. It is obvious that heroic therapeutic measures might be necessary under the latter circumstances, whereas with the former no medication whatever would be needed for years at a time.

The actual cause for ventricular arrest or acceleration remains a mystery. The structural disease that is present in these patients is for the most part unalterable, except in acute cases such as those with acute myocardial infarction. The accompanying heart disease is the same before, during, and after the actual syncope. What, therefore, could be the trigger mechanism that produces the asystole and what is the mechanism that restores the heart beat? The two factors that can act instantaneously are neurogenic or changes in the caliber of vessels, or both. A nerve reflex may suddenly prevent or augment the formation of impulses. A sudden constriction in the caliber of the minute arteriole supplying the conduction tissue can conceivably produce an arrest of the heart. With these mechanisms, the chemical state of the involved tissue may be sufficiently altered to impair its function. Furthermore, the process must be reversible as the attacks so often cease spontaneously. Is there some chemical substance that is released within the body or accumulates in the involved tissue that finally reverses the deleterious process that is going on? These are questions that at present are unanswered.

As is true in many aspects of heart disease, some of the events that occur in heart block are entirely unpredictable. This is particularly true of the attacks of faintness and actual syncope. Some cases of complete block need the customary treatment for congestive failure; others are completely free of congestive symptoms and remain so indefinitely and therefore require no such treatment. In the main, therapy is directed at the actual attacks of syncope.

**Summary**

A review was made of 251 cases of complete heart block. These were divided into the following groups: coronary artery disease (non-acute), 58 cases; myocardial infarction (acute), 49 cases; hypertensive heart disease, 62 cases; rheumatic heart disease, 21 cases; “etiology undetermined,” 18 cases; miscellaneous, 16 cases; and digitalis intoxication, 27 cases. The
main analysis concerned the 224 cases not related to digitalis.

Of the 224 cases, 127 (57 per cent) were males and 97 (43 per cent) were females. The age when first observed by us varied from 10 to 85 years (average 59.2 years) with 84 per cent occurring between 41 and 80 years. The youngest group was the one designated “etiology undetermined.” The average age at death in 126 fatal cases was 63.2 years.

Syncope occurred in 137 cases (61 per cent). There were no instances of syncope in the “digitalis group.” The average duration of life after the first syncopal attack, excluding those who died of acute myocardial infarction, was 6.9 years (range, several hours to 11 years). In many instances syncope preceded complete heart block by a few months to a few years.

Palpitation was a definite symptom in 43 cases. It was a little more common in those with a more rapid ventricular rate. Only occasionally was this symptom helpful in deciding whether attacks of syncope were initiated by ventricular acceleration.

Congestive heart failure was present in about 40 per cent of the cases. This could precede, accompany, or follow the onset of complete heart block. On the other hand, anginal pain was extremely rare during established complete block with slow ventricular rates.

The ventricular rate during complete heart block varied from 16 to 97 (average 38). Six patients had rates over 60 (excluding the digitalis group). Although the ventricular rate is generally regular, in many patients slight irregularities were present.

Although cardiac enlargement of some degree was found in 140 of 224 cases, there were 47 in which no enlargement was detected by x-ray examination. In one instance known to have had complete heart block for 15 years, cardiac enlargement was never demonstrable.

Bundle branch block was noted in 122 cases, 62 of the left and 60 of the right type. First or second degree atrioventricular block was present before or after complete heart block in 67 instances. In 57 cases atrial fibrillation was present, 14 of these were in the “digitalis intoxication” group. Ventricular tachycardia or ventricular fibrillation, either transient or terminal, was observed in 15 cases.

In 176 cases the block was permanent, in 29 it was transient, and in 19 there were repeated bouts of transient complete block. Single episodes were most common in the group of acute myocardial infarction.

The longest duration of heart block proved by electrocardiogram was 21 years, although one patient was known, on clinical examination, to have had a block for 47 years. The average duration of life after the first syncopal attack, including those who died within a few days of acute myocardial infarction, was 35.2 months, and after the first appearance of complete heart block it was 26.2 months.

As to etiologic causes, statistical evidence was presented to support the view that early diphtheria was related in some cases to latent heart block. Syphilis was a cause in only two instances. There was reason to believe that minor infections early in life might well have been the cause in some cases. In the majority of instances, disease of the coronary arteries was the main cause.

Gallbladder disease, generally with stones, was a frequent finding, and cholecystectomy appeared to alter the condition favorably. Nineteen patients underwent 24 major surgical operations without any operative mortality.

One hundred twenty-seven of these cases were known to have died. The mode of death was known in 89 instances. Sudden death occurred in 39 instances and was about three times as common in those who had had syncope than in those who had not.

Of particular interest was the group who had complete heart block during acute myocardial infarction. The immediate mortality was high, 21 of 49 cases. In all those who survived, the rhythm returned to the one which prevailed before the myocardial infarction. Another peculiarity was the ventricular rate in this group; often it was more rapid than is customarily seen in complete block.

The hypertensive group was large (62 cases). Hypertension was also present in many of the cases classified in the other groups, particularly those with coronary artery disease. However,
an elevated blood pressure was by no means a necessary accompaniment of complete heart block.

There were only 21 cases of rheumatic heart disease; 12 had mitral valve disease, 4 had aortic valve disease, and 5 had both. It is clear that complete heart block is an uncommon complication of rheumatic valvular disease.

There were 18 cases with “etiologia undetermined.” In these, some otherwise innocent infection early in life might have been the cause. In general, patients in this classification had a better prognosis.

There were 16 cases that made up the miscellaneous group. In these cases sensitive carotid sinus, syphilitic infection, and other interesting and peculiar factors such as an infected foot, calcium deposits in the heart associated with uremia, and congenital heart disease, formed the background for the heart block.

Digitalis intoxication was a direct cause of complete heart block in 27 instances. Here the condition was transient or terminal. Syncope did not occur in this group, and the ventricular rate was somewhat higher than in the other cases.

Various methods of treatment both for the acute episode and for the prevention of recurrences were analyzed. Therapeutic agents which increase the rhythmic property of the heart, such as epinephrine, isoproterenol, ephedrine, and a mechanical cardiac pacemaker were found to be useful.

**SUMARIO IN INTERLINGUA**

Esseva facite un revista de 251 casos de complete bloco cardiac. Le serie total esseva dividite in le sequente gruppos: 58 casos (non-acute) de morbo de arteria coronari, 49 casos (acute) de infarcimento myocardial, 62 casos de morbo cardiac hypertensive, 21 casos de morbo cardiac rheumatic, 18 casos a “etiologia indeterminate,” 16 casos miscellaneous, e 27 casos de intoxication a digitalis. Le analyse principal se restringeva al 224 casos non relationate a digitalis.

Le 224 casos includeva 127 masculos (57 pro cento) e 97 femininas (43 pro cento). Le etate del patientes quando illes esseva primo obser-
“intoxication a digitalis.” Tachycardia ventricular o fibrillation ventricular, transiente o terminal, esseva observate in 15 casos.

In 176 casos le bloco esseva permanente. In 29 illo esseva transiente. E in 19 casos il habeva repetitive episodios de transiente bloco complete. Episodios unic esseva le plus frequente in le gruppo de acute infarimenti myocardial.

Le plus extense duration de bloco cardiae, electrocardiographicamente demonstrate, esseva 21 annos, sed in un paciente evidenteria clinica esseva disponibile que indicava bloco cardiac de un duration de 47 annos. Le supervivencia median post le prime attacco syncopic (includente le patientes qui moriva intra aliquen dies in consequentia de infarimento myocardial) esseva 35,2 menses; post le prime manifestation de complete bloco cardiac, illo esseva 26,2 menses.

Quanto al question etiologic, nos ha presentate datos in supporto del conclusion que le precoce occurrentia de diphtheria esseva relationate in certe casos con le disveloppamento de un latente bloco cardiac. Syphilis esseva le causa in solmente duo casos. In un serie de casos il habeva justificationes a vider le causa in minor infectiones in juveme annos del vita. In le majoritate del casos morbo del arterias coronari esseva le causa principal.

Morbo del vesica biliari, generalmente con calculos, esseva un observation frequente, e cholecystectomy pareva influentar le condition favorabilemente. Dece-nove patientes esseva subjicite a 24 major operationes chirurgic sin ulla mortalitate operatori.

In 127 casos il esseva cognoscite que le patientes esseva morte. Le modo del morte esseva cognoscite in 89 casos. Mortes subitanee esseva characteristic de 39 casos; illos esseva circa tres vices plus commun in patientes con syncope que in patientes qui non habeva habite synopes.

De interesse special esseva le gruppo de patientes qui habeva complete bloco cardiac durante acute infarimento myocardial. Le mortalitate immediate esseva alte: 21 inter 49 casos. In omne le superviventes le rhythmio retornava al forma prevalente ante le infarimento myocardial. Un altere peculiaritate in iste gruppo esseva le frequentia ventricular: frequentemente illo esseva plus alte que lo que es commun in casos de bloco complete.

Le gruppo hypertensive esseva grande. Illo includeva 62 casos. Hypertension esseva etiam presente in multes del casos classificate in le altere gruppos, specialmente in le gruppo de morbo del arterias coronari. Tamen, elevation del pression sanguine esseva non del toto un accompaniamento necessari de complete bloco cardiac.

Il habeva solmente 21 casos de rheumatic morbo cardiac: 12 con morbo de valvula mitral, 4 con morbo de valvula aortica, 5 con ambes. Il es clar que complete bloco cardiae es un complicatation non commun de rheumatic morbo valvar.

In 18 casos le etiologia esseva “indeterminata.” In illos un alteremente innocue infection durante epocas precoce del vita esseva possibilemente le causa. In general, patientes in iste gruppo habeva un melior prognose.

Le gruppo miscellanee consisteva de 16 casos. In iste cases le bloco cardiac esseva associate con sensibile sinus carotide, infection syphilitic, e altere interessante e specialisate factores, como per exemplo infection del pede, depositos de calcium in le corde in combination con uremia, e congenite morbo cardiac.

Intoxication a digitalis esseva le causa directe de complete bloco cardiac in 27 casos. In illos le condition esseva transiente o terminal. Syncope non occurreva in iste gruppo, e le frequentia ventricular esseva alique plus alte que in le altere cases.

Es analysate varie methodos de tractamento, tanto pro le episodio acute como etiam pro le prevention de recurrentias. Agentes therapeutic que augmenta le rhythmicitate del corde (p.ex. epinephrina, isoproterenol, e ephedrina) e le uso de un mechanic pacemaker cardiac se monstравa utile.

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