A Study of Heart Block in Man Using His Bundle Recordings

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SUMMARY
The technique of recording His bundle electrograms in man by a tripolar electrode catheter was applied in a study of patients with first, second, and third-degree heart block. In all patients with first-degree heart block (congenital, acquired, or induced by atrial pacing), the delay in A-V conduction occurred proximal to the His bundle as evidenced by a prolonged P-H interval. The enhancement of A-V conduction (shortening the P-H interval) due to isoproterenol was qualitatively similar in the three forms of first-degree block. In cases of Wenckebach phenomenon the P-H interval progressively increased until a dropped beat occurred. The nonconducted P wave was not followed by a His deflection, indicating block proximal to the His bundle. Cases of 2:1 and 3:1 block occurring proximal to the His bundle were also studied. The usefulness of His-bundle recordings in the diagnosis of cases of complete and incomplete bilateral bundle-branch block is also demonstrated.

Additional Indexing Words:
Wenckebach phenomenon     Bilateral bundle-branch block     A-V conduction
Atrial pacing

CLINICALLY, atrioventricular block is classified into three varieties: first-degree, second-degree, and third-degree heart block. The clinical electrocardiographic criteria for these three forms of heart block have been established.

The P-R interval represents the delay in transmission of an atrial impulse to the ventricles. Unfortunately, the electrical activity of the specialized conducting tissue (A-V node, His bundle, right and left bundle branches and part of the Purkinje fibers) through which an atrial impulse passes is not recorded in the P-R interval of the standard electrocardiographic leads. Consequently, our knowledge concerning the precise location(s) of delay and block during A-V transmission in man has been limited. In this regard, much of our information has been extrapolated from animal experiments in which the electrical activity of the specialized conducting tissues has been recorded by a variety of techniques.

A method for the safe and systematic recording of His-bundle electrograms in man has recently been developed in our laboratory. The purpose of this study was to define more precisely in man the areas of delay or block in the three varieties of heart block encountered clinically by means of the technique of His bundle recording.

Methods
Right heart catheterization was performed in the postabsorptive, nonsedated state. Under local anesthesia, a tripolar electrode catheter was percutaneously introduced into the right femoral vein and fluoroscopically positioned across the tricuspid valve. The proximal terminals of the electrodes were connected to a distribution
switch box,* which allowed for the selection of two bipolar leads (electrodes 1 and 2 and electrodes 2 and 3). Each bipolar lead was connected to the A-C input of an ECG preamplifier. The filter frequencies of the ECG amplifiers were set at 40 and 500 cps. A standard ECG lead was simultaneously monitored and recorded. The electrode catheter was withdrawn slowly across the tricuspid valve until a rapid biphasic or triphasic deflection appeared between the atrial and the ventricular electrograms and within the P-R interval. All records were made on a multichannel oscilloscopic photographic recorder* at paper speeds of 100 to 200 mm/sec. In most patients an additional bipolar electrode catheter was percutaneously introduced into an antecubital vein and fluoroscopically positioned against the lateral wall of the right atrium. This catheter was used to pace the right atrium up to 150 beats/min. Right atrial pacing was accomplished by using a battery-powered pacemaker that delivered impulses of 2-msec duration at an adjusted milliamperage to insure reliable “atrial capture.” Measurements in milliseconds were made of the interval from the P wave (or pacer impulse) to the His bundle deflection (P-H interval) and from the His deflection to the onset of the ventricular electrogram (H-Q interval). Careful attention was paid to the grounding of all equipment to avoid the introduction of random currents into the system. Nearly 100 cardiac catheterizations have been performed in our laboratory, in which His bundle recordings have been obtained without any untoward effects. An occasional premature ventricular contraction has been noted when the electrode catheter first enters the right ventricle. Once the electrode

*Electronics for Medicine, White Plains, New York.

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**Figure 1**

His bundle recordings in a case of congenital first-degree heart block. P.I. represents the pacer impulse. At sinus rate (top panel) the interval from the P wave to the His bundle (P-B.H.) is 360 msec. At a paced atrial rate of 85/min the P-B.H. interval increased to 415 msec. At a paced atrial rate of 90/min (bottom panel) second-degree A-V block occurred. The third pacer impulse depolarizes the atrium but is not conducted to the ventricles.

Circulation, Volume XXXIX, March 1969
catheter is positioned at the tricuspid valve no ventricular irritability occurs, and stable recordings may be obtained for several hours.

Results

First-Degree Heart Block

Congenital First-Degree Heart Block

Figure 1 represents a case of congenital first-degree heart block in a 21-year-old asymptomatic man. In each panel the top tracing represents a His-bundle electrogram (HBE) and the bottom tracing a standard lead II electrocardiogram (L2). At sinus rhythm the P-H interval was 360 msec, and at a paced heart rate of 85/min the P-H interval increased to 415 msec. At a paced rate of 90/min, second-degree A-V block occurred. The H-Q interval remained constant (38 msec) throughout the pacing procedure. In this patient the infusion of isoproterenol (0.15 μg/kg/min) or atropine (1 mg) enhanced A-V conduction. The right atrium could be paced up to rates of 132/min without the occurrence of dropped beats. Following isoproterenol the P-H interval at a paced heart rate of 132/min was 331 msec and the H-Q interval remained constant.

Acquired First-Degree Heart Block

A case of first-degree heart block occurring in a 67-year-old man with arteriosclerotic heart disease is illustrated in figures 2 and 3. At sinus rhythm (70/min) the P-H interval was 330 msec and the H-Q interval 46 msec. At a paced rate of 110/min the P-H interval increased to 495 msec, but the H-Q remained constant. It is to be noted that at a paced heart rate of 110/min, the atrial impulse (P.I.), which is responsible for each QRS response, is located just prior to the Q wave of the preceding QRS complex. Isoproterenol (0.05 μg/kg/min) shortened the P-H interval to 237 msec but did not alter the H-Q interval. Similar results were observed after the infusion of atropine (1 mg).

First-Degree Heart Block Induced by Atrial Pacing

Figure 4 depicts a case of first-degree heart block induced by atrial pacing in a
normal 32-year-old man. The P-H interval at a paced rate of 80/min was 167 msec, and at a paced heart rate of 110/min the P-H interval increased to 281 msec. At the higher paced heart rate the P-R interval measured 334 msec (281 + 53 msec).

Second-Degree Heart Block

Type I–Wenckebach Phenomenon

Several cases of spontaneous and induced (by right atrial pacing) Wenckebach phenomena were studied. A case of spontaneous Wenckebach is shown in figure 5. Starting with the third beat, the P-H interval progressively increased from 200 to 600 msec before the dropped beat occurred. The second QRS complex in this tracing occurs prematurely and has a right bundle-branch-block pattern. It is preceded by an inverted His bundle deflection. One possibility regarding the origin of this beat is that it is an A-V junctional escape rhythm arising in the common bundle downstream from the sensing electrodes, which recorded a His bundle deflection of opposite polarity to the impulses that were propagated in an antegrade direction. The prematurity of the impulse caused aberrant ventricular depolarization (RBBB). Another possibility is that the catheter caused a transient distal block in the right bundle branch while concomitantly producing retrograde conduction into the main bundle and then down the left bundle.

A case of Wenckebach phenomenon with a 4:3 conduction ratio resulting from atrial pacing is shown in figure 6. Again the P-H interval progressively increased until a dropped beat occurred. The nonconducted P wave is blocked proximal to the His bundle.
wave was not followed by a His-bundle deflection. During the Wenckebach phenomenon the H-Q interval remained constant.

Increasing the paced atrial rate in this patient produced higher degrees of A-V block. Figure 7 is an example of 2:1 A-V block that occurred at an atrial paced rate of 120/min. The ventricular response is 60/min. In this example, the nonconducted atrial impulses were not followed by a His deflection, indicating block proximal to the His bundle.

When the atrial rate was further increased, a 3:1 block occurred (fig. 8). Again, the nonconducted beats are not followed by a His-bundle deflection.

**Type II—Second-Degree A-V Block**

Figure 9 represents a case of spontaneous 2:1 block with left bundle-branch block. The atrial rate is 80/min, and the ventricular response is 40/min. The configuration of the P waves from the standard ECG lead and the atrial electrograms indicates that all the P waves are conducted in an antegrade direction. All the P waves are followed by a His-bundle deflection but only every other P wave is conducted to the ventricles. In this case of 2:1 block the level of block is distal to the His bundle. The left bundle was completely and permanently blocked. Since only one of every two impulses is conducted down the right bundle branch, second-degree right bundle-branch block may be diagnosed. Thus, this case represents an example of incomplete

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**Figure 7**

A 2:1 block during right atrial pacing. Every other P wave is blocked proximal to the His bundle.

**Figure 8**

A 3:1 block induced by right atrial pacing. The block occurs proximal to the His bundle.

**Figure 9**

A case of incomplete bilateral bundle-branch block. The standard ECG lead shows 2:1 block with left bundle-branch block. The HBE reveals that the nonconducted P waves are blocked distal to the His bundle. There is complete (third-degree) block of the left bundle branch and second-degree block of the right bundle branch.
A case of complete heart block due to bilateral bundle-branch block. See text for discussion.

bilateral bundle-branch block. However, it is not possible from our data to exclude the possibility that the level of block was in the lowest part of the His bundle.

Third-Degree Heart Block
A case of complete heart block due to bilateral bundle-branch block occurring in a 66-year-old man is shown in figure 10. In the top panel, the atrial rate is 81/min and is independent of the ventricular rate, which is 49/min. The ventricular complexes are of 0.12-sec duration and arise from an idioventricular focus. The His bundle electrogram

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**Figure 10**

A case of complete heart block due to bilateral bundle-branch block. See text for discussion.

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**Figure 11**

Atrial flutter. In the top panel each QRS is preceded by P wave and His bundle deflection. In the bottom panel carotid sinus stimulation increases the degree of block and reveals that the nonconducted P waves are blocked proximal to the His bundle.
reveals that each of the P waves is followed by a His bundle deflection, indicating that the area of block was below the common bundle. In the bottom panel, the atrial rate is increased by pacing, and each P wave is followed by a His deflection.

**Atrial Flutter**

His-bundle recordings were also made in a case of atrial flutter (fig. 11). The atrial flutter rate is 320/min, and the ventricular response is 160/min. Each QRS complex is preceded by a single His deflection, and the H-Q interval is constant from beat to beat. The level of block for the nonconducted P waves is revealed during carotid sinus stimulation, depicted in the lower panel. None of the nonconducted P waves are followed by a His deflection, indicating that block occurred proximal to the His bundle.

**Discussion**

The electrocardiographic manifestation of first-degree heart block in adults is a P-R interval that exceeds 0.20 seconds. This prolongation in the P-R interval is the result of a decrease in the conduction velocity within the atroioventricular conduction system. The findings in the present study clearly demonstrate that in all forms of first-degree heart block, congenital, acquired, and induced, the delay in A-V conduction occurs proximal to the His bundle. The values for the H-Q intervals were the same as those obtained in subjects without first-degree heart block. Furthermore, the enhancement of A-V conduction due to isoproterenol and atropine was found to be qualitatively similar in the three forms of first-degree heart block. Whether the greater delay in A-V conduction occurs at the atrionodal junction as suggested by Hoffman and associates or between the nodal-His region as suggested by Alanis and co-workers could not be determined from the results of this study.

Wenckebach phenomenon, or type I, is the more commonly encountered form of second-degree heart block. In this form of block the P-R interval progressively widens, with a decreasing increment of delay, until an atrial impulse is not conducted to the ventricles. The P-R interval following the dropped beat is short and then progressively widens as the cycle is repeated. In both the spontaneous and the induced forms of Wenckebach phenomenon herein reported, the area of block was located proximal to the His bundle. These findings are in agreement with the experimental studies in which microelectrode techniques have been used. Hoffman and Cranefield have proposed that the primary change in nodal activity causing delay and failure of transmission in the Wenckebach phenomenon occurs between the mid-node and the atrium. Watanabe and Dreifus have concluded from their studies that the major conduction delay in the Wenckebach form of block occurs in the nodal (N) region of the A-V junction. However, additional delay may be noted in the nodal-His (N-H) region. Those investigators have also shown that a gradual shortening of the ventricular interval need not be considered a prerequisite for the diagnosis of Mobitz type I heart block. In some of their experiments, a progressive prolongation of A-V transmission time with increasing increments occurred, resulting in a gradual slowing of the ventricular rate prior to the dropped beat. They found that any combination of ventricular cycle lengths may be expected, depending on the relative degree of delay in the various portions of the A-V transmission system. These investigators concluded that inhomogeneous conduction of impulses in the A-V junctional tissue may be responsible for this type of block. Our cases of 2:1 and 3:1 A-V block (figs. 7 and 8) are a form of type I Mobitz block. Failure of impulse transmission always occurred proximal to the His bundle. The mechanism of block in these cases is similar to that observed in the induced Wenckebach phenomenon, since cases of the latter could be made to demonstrate 2:1 and 3:1 conduction by increasing the paced atrial rate.

Watanabe and Dreifus have shown that in contrast to Mobitz type I block, where the greatest delay occurred in the N region, the
major delay in Mobitz type 2 block is in or below the His bundle. Our case of Mobitz type 2 block (fig. 9) is in agreement with these findings. In this example, failure of impulse transmission occurred distal to the His bundle.

Lepeschkin\textsuperscript{16} has proposed several electrocardiographic criteria for the differential diagnosis of various degrees of bilateral bundle-branch block. According to Lepeschkin's criteria, our case of 2:1 block with left bundle-branch block (fig. 9) represents an example of incomplete bilateral bundle-branch block. There is third-degree or complete block in the left bundle and second-degree block in the right bundle branch, which in its simplest form presents itself in the standard ECG as 2:1 A-V block with a left bundle-branch-block pattern. In our case, the atrial impulses depolarized the His bundle as evidenced by the fact that each P wave was followed by a His deflection. Furthermore, the P-H intervals of the conducted and non-conducted beats were the same (P-H 90 msec). The left bundle branch could conduct none of the atrial impulses (third-degree block) whereas the right bundle could conduct only every other impulse (second-degree block). It has been previously emphasized that bilateral bundle-branch block should always be suspected whenever second-degree A-V block is associated with either a right or left bundle-branch-block pattern.\textsuperscript{16-18}

The histopathological studies of Mahaim,\textsuperscript{19} Yater,\textsuperscript{20} and Lenegre\textsuperscript{21} have demonstrated that bilateral bundle-branch block rather than block at the A-V node or His bundle is a frequent cause of complete heart block. The value of analyzing the sequential electrocardiographic changes in diagnosing complete heart block due to bilateral bundle-branch block has been demonstrated.\textsuperscript{16, 17} In our case, electrocardiograms antedating the onset of complete heart block were not available. Nonetheless, it appears that the diagnosis of complete heart block due to bilateral bundle branch block can be made with reasonable certainty by the technique of His-bundle recordings. The diagnosis is based on the fact that the level of block was demonstrated to be below the bundle of His and the ventricular pacemaker was of an idioventricular origin rather than one of the bundle branches.

References

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Circulation. 1969;39:297-305
doi: 10.1161/01.CIR.39.3.297

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