Pervenous Pacing of the Specialized Conducting System in Man

His Bundle and A-V Nodal Stimulation

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SUMMARY

His bundle electrograms were recorded and A-V junction pacing was achieved in 30 patients by a pervenous electrode catheter technic. His bundle pacing was achieved in 26, and A-V node pacing in five patients. Conduction time from atrium to the His bundle (A-H time) and His bundle to ventricular activation (H-V time) were measured. During His bundle pacing, the pacing impulse to ventricular activation time (PI-R) was the same as the H-V time during normal sinus rhythm and remained constant at different pacing rates. With A-V node pacing, the PI-R interval was shorter than the conduction time from atrium to the ventricle (sum of the A-H and the H-V time) but longer than the H-V time, and there was progressive lengthening of PI-R interval with increase in pacing rates. The QRS complex remained unchanged in all ECG leads with BH and A-V node pacing from that during normal sinus rhythm. During BH pacing, the retrograde Wenckebach phenomenon was demonstrated. Bundle of His pacing was used to establish a His bundle rhythm in a patient with complete heart block localized within the His bundle. BH pacing is of value for validation of His bundle electrograms and differentiation from that of right bundle branch. Clinically, this technic has proved extremely useful in definitive diagnosis of bilateral bundle-branch block in patients with right bundle-branch block and left axis deviation and infarction.

Additional Indexing Words:
Wenckebach phenomenon Bundle-branch block

ARTIFICIAL electrical pacing of the heart has been employed both experimentally and clinically. However, only two pacing sites have been used in man, ventricular and atrial. Recently, stimulation of specific areas of the specialized conducting system in man has been described.¹ There have been several reports of His bundle recordings in man from this and other laboratories.²⁻⁸ On the basis of our experience with this recording technic in 150 patients, we have found that the usual criteria for differentiating the His bundle (BH) electrogram from that of the right bundle branch (RB) are not always reliable (fig. 1). This is particularly true in patients with a diseased His bundle and bundle-branch system. For quantitative measurements of conduction time in the His-Purkinje system it is essential to differentiate between these two electrograms and thus validate the His bundle recordings.

This study reports our experience with His bundle pacing in 30 patients with normal and abnormal A-V conduction in whom His bundle recordings were validated. During BH pacing, stimulation of the A-V nodal region was also observed in five patients.

Methods

Thirty patients ranging in age from 24 to 72 years were studied during right heart catheteriza-
Simultaneous recordings of bipolar electrogram (BE) from the area of the A-V junction and standard ECG leads I, II and III in a patient with right bundle-branch block and left axis deviation. Note the abnormal durations of RB (15 msec) and BH (30 msec) electrograms. The H-V and RB-V time is 65 and 50 msec, respectively. P-P = the time interval between two consecutive P waves. P-R = the time interval from the P wave to the earliest onset of the QRS complex on either of the three ECG leads. A-H = the time interval from the A wave to the bundle of His (BH) deflection. H-V = the time interval from BH to the onset of ventricular activation (V). A-RB = the time interval from the A wave to the right bundle-branch (RB) deflection. RB-V = the time interval from RB to the onset of ventricular activation. A = bipolar atrial electrogram from the area of the A-V junction. BH = bipolar electrogram of the bundle of His. RB = bipolar electrogram of the right bundle branch. V = bipolar ventricular electrogram from the area of the A-V junction. On this and all subsequent figures, time lines at 1 second; paper speed, 100 mm/sec.

Figure 1

All patients studied had underlying heart disease or chronic lung disease. The patients were studied in the postabsorptive state and premedicated with 100 mg of Nembutal, administered intramuscularly, 1 hour before catheterization.

Under fluoroscopic control and intracardiac electrographic monitoring, a bipolar electrode catheter with ring electrodes 2 mm wide and 5 mm apart was introduced percutaneously from the right femoral vein and advanced into the right ventricle. The catheter was slowly withdrawn from the right ventricle until the electrodes were in proximity to the septal leaflet of the tricuspid valve, and a His bundle electrogram was obtained. Sometimes a slight rotational movement of the catheter toward the septum was used to enhance the BH recording. Bipolar and unipolar electrograms from the area of the A-V junction were recorded by the connection of the catheter leads to an electrode distribution box, which was then connected to a multichannel oscilloscopic photographic recorder. Three standard ECG leads, I, II, and III, or I, aVF, and aVL were recorded simultaneously with the intracardiac recordings. The unipolar electrogram and standard lead electrocardiogram were recorded at filter settings of 0 to 20 cycles/sec. The bipolar electrogram was recorded at filter settings of 40 to 200 cycles/sec. All the recordings were made at paper speed of 100 mm/sec with 1-second time lines. A second bipolar pacing catheter was introduced into the right atrium through a surgically exposed antecubital vein and connected to a Medtronic paired pulse generator model 5734. The right atrium was paced at progressively increasing rates up to 170 per minute or until Wenckebach cycles developed. His bundle electrograms were recorded at each atrial pacing rate.

The same bipolar catheter used for BH recording was then connected to an external Medtronic pacemaker for BH pacing. The pacemaker was used in a fixed-rate pacing mode at 15 MA. The BH could be paced at lower current levels, but the pacemaker output of 15 MA was arbitrarily chosen to avoid failure of BH excitation due to inadequate stimulus strength rather than poor catheter position. BH pacing was usually achieved from its recording site except for slight rotation or withdrawal of the catheter. Occasionally, reversing the pacemaker polarity was also helpful. The bundle of His was paced at three different heart rates above the control levels. During the course of BH pacing in several patients, A-V nodal pacing was observed as the catheter was withdrawn slightly (1 to 2 mm). The criteria for validating the A-V nodal pacing are discussed below. The atrial bipolar catheter was used to record the atrial activity during BH and A-V nodal pacing. In addition, three standard ECG leads were simultaneously recorded.

The P-R interval was measured from the onset of the P wave to the beginning of the QRS complex. A-H time is the interval from the first rapid deflection of the A wave to the first rapid deflection of the BH bipolar electrogram. Since the A wave is recorded from the area adjacent to the A-V node, the A-H time represents the conduction time through the A-V node. The His bundle to ventricular activation time (H-V) was measured from the first rapid BH deflection to the earliest ventricular depolarization recorded in either the intracardiac electrogram or standard ECG leads. The PI-R interval is the time from the pacemaker impulse to the earliest ventricular depolarization seen on either of the three standard electrocardiographic leads during atrial, A-V nodal, or BH pacing.

Results

Bundle of His and A-V nodal pacing were done in 26 and 5 patients, respectively. During
Figure 2

A. Simultaneous recordings of bipolar electrogram (BE) from the area of the A-V junction and standard ECG lead I (L-1). Normal sinus rhythm = 65 per minute. The A-H and H-V times are 110 and 60 msec, respectively. B. Atrial pacing at 90 per minute resulted in a prolongation of A-H time to 140 msec with an unchanged H-V time = 60 msec. C and D. The bundle of His is paced at rates of 90 and 110 per minute. The pacing impulse (PI) to the onset of the QRS complex time (PI-R) remained constant at 60 msec during both levels of BH pacing rates. 

BH pacing, the pacing impulse to QRS (PI-R) interval was the same as the H-V time during normal sinus rhythm or atrial pacing. There was no change in the amplitude, configuration, and duration of the QRS complex in all ECG leads from that seen during normal sinus rhythm. With BH pacing, the conduction time (PI-R) remained constant at all heart rates (fig. 2). During pacing of the A-V nodal region, the PI-R interval was shorter than the sum of the A-H and H-V time, but longer than the H-V time alone during atrial pacing at the same heart rate (fig. 3). During A-V nodal pacing with progressive increases in heart rate, there was progressive prolongation of the PI-R interval. In most of the instances the
atrium and the His bundle were depolarized simultaneously during BH pacing, whereas in other instances retrograde conduction through the A-V node was seen (fig. 5).

Three selected cases are described below:

J. K., is a 69-year-old male patient whose ECG showed right bundle-branch block, left axis deviation, and a myocardial infarction pattern (fig. 4). His bundle recordings showed an H-V time of 50 msec. The patient developed antegrade Wenckebach cycles with atrial pacing at a rate of 130 per minute. BH pacing was achieved at three different heart rates ranging up to 150 beats per minute, with 1:1 conduction to the ventricles, which was 20 beats faster than the rate at which antegrade A-V block developed. The PI-R interval during BH pacing was 50 msec, equal to the control H-V time. The same ECG pattern of RBBB as during control recordings and atrial pacing was maintained without any change in the shape of the QRS complex during BH pacing.

W. A. is a 76-year-old male patient who
showed alternating right and left bundle-branch block (partial bilateral bundle-branch block) with first-degree A-V block during the study. He had abnormal A-H (175 msec) and H-V (95 msec) times and developed Wenckebach cycles during atrial pacing at a rate of 90 per minute. His bundle pacing was achieved during each of the alternating conduction patterns that were seen during normal sinus rhythm (figs. 5 and 6) at a rate of 100 beats per minute. With BH pacing at this heart rate, the retrograde Wenckebach phenomenon was seen (fig. 5). The pacing impulse to retrograde atrial (A) depolarization (P1-A) interval progressively lengthened from 200 msec to 540 msec until finally the retrograde impulse was blocked.

J. R., a 48-year-old female patient with a heart rate of 37 beats per minute, had complete heart block with narrow QRS complex (0.08 sec) in all ECG leads (fig. 7). Recordings from the area of the His bundle revealed two separate His bundle deflections BH and BH' during sinus rhythm with complete heart block. One BH deflection was paired with the atrial activity (A-H time = 90 msec) and the second BH' deflection preceded the ventricular depolarization with H'-V time of 40 msec. The first BH deflection associated with the atrial activity (A) was demonstrated to move away from the A-wave (A-H time increased from 90 to 150 msec) as the A-A time was shortened from 940 to 440 msec by atrial pacing. During BH' pacing at rates of 57 and 85 beats per minute, the PI-R interval was 40 msec, which was identical to the BH' to ventricular activation interval (H'-V) during the control complete heart block. The QRS pattern during BH' pacing again remained unchanged from that seen with complete heart block, that is, narrow QRS complex of 0.08 sec in duration.
Case W.A. Twelve standard ECG leads (top) show first-degree block with right bundle-branch block and left axis deviation. A. Simultaneous recordings of bipolar electrogram (BE) from the area of the A-V junction and standard ECG leads L-1, L-2, and aVF, during normal sinus rhythm. The A-H time varies between 150 and 175 msec, with variations in P-P interval. The H-V time of 95 msec remained constant. B. Simultaneous recordings of bipolar electrogram (BE) from the right atrium (RA) with standard ECG leads L-1, L-2, and aVF, during BH pacing at a rate of 100 per minute (PI-PI = 600 msec). PI-R interval of 95 msec is the same as the H-V time during normal sinus rhythm and the shape of the QRS complex remained unaltered throughout. Note that there is a retrograde Wenckebach phenomenon exhibited as the PI to the A wave (PI-A) time progressively lengthens from 200 to 540 msec.

The BH pacing was stable in some instances and was maintained for up to one-half hour. In most instances, pacing could be maintained for four to six consecutive beats before the catheter...
Case W.A. Twelve standard ECG leads (top) from the same patient as in figure 5, whose pattern changed during study from right bundle-branch block to left bundle-branch block. A. Simultaneous bipolar electrograms (BE) recorded from the area of the A-V junction and standard ECG leads L-1, L-2, and aVF. The A-H time at P-P interval of 850 msec was 180 msec. The induced (PI) premature atrial systole (A) at P-P interval of 640 msec was either blocked and followed by an A-V junctional escape beat or conducted with an extremely prolonged A-H time. The H-V time of 90 msec was constant throughout. B. Bundle of His pacing at a rate of 100 per minute (PI-PI = 600 msec). The PI-R interval of 90 msec is equal to the H-V time during normal sinus rhythm. The shape of the QRS complex remains unaltered throughout.

Discussion

During A-V nodal or BH rhythms, the QRS complex is identical to that during normal sinus rhythm in all ECG leads. Spontaneous or induced impulses arising from the sinus node, A-V node or the His bundle are normally transmitted through the same pathways to the ventricular musculature producing similar pattern of ventricular depolarization. However, direct stimulation of the ventricular
Case J.R. Twelve standard ECG leads (top) show narrow QRS complexes (0.08 sec) with complete heart block. A. Simultaneous recordings of bipolar electrogram from the area of the A-V junction and standard ECG leads L-1, L-2, and L-3 during complete heart block, with a ventricular rate of 37 beats per minute. The control A-A time was 940 msec, with A-H time of 90 msec. As the A-A time was shortened to 440 msec with an induced atrial extrasystole (PI) the A-H time lengthened to 150 msec. The BH' deflection precedes each ventricular activation (V) by 40 msec (H'-V). B and C. Show BH pacing at heart rates (H-H) of 57 and 85 per minute, respectively. The PI to ventricular activation interval (PI-R) of 40 msec remained constant at both pacing levels and is equal to the H'-V time during His bundle rhythm (A). Note that the shape and duration of the QRS complex are maintained throughout.

Musculature or the right bundle branch produces aberrant QRS complex due to alteration in ventricular activation pattern from that during normal sinus rhythm.

Further evidence indicating that the His bundle is the structure being stimulated is shown in figure 4 (case J. K.) by pacing the BH at a higher rate than the atrial pacing rate at which the patient developed antegrade Wenckebach cycles. The pattern of right bundle-branch block present during atrial pacing was also maintained during BH pacing, thus excluding the possibility of RB or ventricular pacing. Pacing the BH at a higher rate with 1:1 conduction to the ventricle than the rate at which Wenckebach developed during atrial
pacing rules out the possibility of stimulation proximal to the BH. In W. A. (fig. 5) the exhibition of retrograde Wenckebach during BH pacing demonstrates clearly the retrograde impulse transmission through A-V node. At the same time antegrade ventricular activation was maintained identical to that during normal sinus rhythm. Also, the BH pacing rate was higher than the rate at which antegrade Wenckebach cycles developed during atrial pacing.

In the patient (J. R., fig. 7) with complete heart block with a narrow QRS complex, this technic was used to provide evidence for a focal His bundle block and the existence of BH rhythm. The recording of a rapid deflection (BH) after each A wave indicated that the block was distal to the BH. Furthermore, the BH deflection was validated by demonstrating the prolongation of A-H time by shortening of the P-P interval with atrial pacing. The "ventricular" pacemaker in this patient, because of the narrow QRS complex, must be situated in the His bundle proximal to the bifurcation into the right and left bundle branches. The second BH' deflection was validated as arising from the bundle of His because the PI-R interval during BH' pacing was the same as BH' to ventricular activation time (H'-V = 40 msec). This could not have been the deflection arising from the right bundle branch because the conduction time from the RB to ventricular activation is shorter than 40 msec in patients with normal A-V conduction and a narrow QRS complex. Also, pacing of the right bundle would produce a left bundle-branch block pattern, thus abolishing the narrow QRS complex.

Several recent reports describe the utilization of BH recordings for analysis of A-V conduction. This recording technic has been used in patients to analyze quantitatively the pattern of right bundle-branch block (RBBB) and left axis deviation (LAD). Our data show that RBBB and LAD may represent bilateral bundle-branch block by demonstrating abnormal H-V time. These patients consistently showed H-V times ranging from 55 to 90 msec whereas the patients with normal A-V conduction show H-V times ranging from 35 to 45 msec. In the presence of old myocardial infarction, the pattern of RBBB and LAD may not represent true bilateral bundle-branch block if the H-V time is normal (fig. 4). Therefore, the use of BH recordings for analysis of these and other conduction defects and for precise quantitative measurements of conduction time through the His-Purkinje system requires that the BH electrogram be validated.

Previous workers have established some of the criteria for validating the His bundle deflection. Since the BH deflection is recorded during the P-R interval, it could represent the electrical activity of the A-V node, BH, or the right bundle branch (RB). The BH and RB deflections can easily be differentiated from that of the A-V node. The A-V node deflection is of longer duration (50 msec), inscribed slowly, and of low amplitude. As the P-P interval is shortened with atrial pacing, there is a progressive and marked lengthening of the interval between the A wave and the BH or RB deflections. However, the increase in the time from the A wave to the A-V nodal deflection is insignificant as compared to the prolongation of the A-H time. During shortening of the P-P interval, BH and RB electrograms both will respond in the same manner. The means for differentiating these two deflections have not been previously elucidated in man. The criteria for distinguishing between these on the basis of shape, duration, and amplitude are not always dependable. The normal duration for RB electrogram is about 10 msec. The right bundle deflection may be of longer duration, especially in abnormal cases with conduction defects. Note that in figure 1 the duration of RB electrogram is 15 msec, which is usually the duration of the BH electrogram in patients with normal A-V conduction. Also, the RB-V time of 50 msec is abnormally long. If this were the only deflection recorded between the atrial and ventricular deflections, it could be readily interpreted for a BH electrogram because of its duration and the
timing from the deflection to the ventricular activation.

This study demonstrates that during BH pacing, PI-R interval is equal to the H-V time during normal sinus rhythm but longer than RB to ventricular activation time by 10 to 15 msec. The shape of the QRS complex with BH pacing is the same, as during normal sinus rhythm irrespective of its shape and duration. This is not the case with RB pacing, which produces a pattern of left bundle-branch block. These findings are in agreement with previous experimental work in dogs. With A-V nodal pacing, the PI-R interval is longer than during BH pacing or the control H-V time, but shorter than the sum of A-H and H-V time, that is, the conduction time from the atrium to the ventricle.

Previous work from this laboratory had shown that the H-V time remained constant during atrial pacing at different heart rates ranging up to 180 beats per minute. The present findings of a constant PI-R interval at different heart rates with BH pacing are in agreement with the same observations and further corroborate the work originally done by Alanis in animals. Alanis measured the conduction velocity through the His-Purkinje system at different pacing rates and showed that it was virtually constant.

A theoretical possibility does exist that during pacing of the distalmost region of the BH, the PI-R interval may be shorter than during pacing of the proximal BH and shorter than the control H-V time. But this possibility is unlikely for the following reasons: (1) The chances of achieving BH stimulation at its lowermost end, with electrodes 5 mm apart, without adjacent ventricular septal depolarization are very rare since the BH itself is usually only 1 cm long. (2) Conduction time through the BH itself is normally very short (about 15 msec), thus reducing the range of error to half this time if the lower end of BH was stimulated as compared to its uppermost end. (3) The latency from the pacing stimulus to BH activation partly compensates for this range of error.

Stable BH pacing could not be achieved in all patients because of anatomical variations and catheter movement caused by cardiac and respiratory movements. However, bundle of His pacing is the only definite means of validating the His bundle electrogram, especially for accurate diagnosis of bilateral bundle-branch block in patients with RBBB and LAD and old myocardial infarction.

References


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Early American Assessment of Digitalis

There has been much inquiry into the manner in which foxglove acts in curing dropsies. It has been supposed to exert a specific action on the kidneys as a diuretic; but I am rather disposed to believe, that it acts only by lessening the action of the arterial system by a sedative quality which appears to reside in it. . . .

. . . There are different opinions concerning the efficacy of this medicine in dropsies. From the cases related by Dr. Withering, it appears to have done good; but from those related by Dr. Lettsom it seems to have done harm. I suspect the different accounts of those two gentlemen have arisen from their having given it in different states of the system. In dropsies of too much action, I believe it has sometimes been used with success, but in atonic dropsies, I am satisfied that it is not only an useless but a dangerous medicine. I am sorry to add further, that after many trials of this medicine I have failed in most of the cases in which I have given it. I have discharged the water in three instances by it, but the disease returned, and my patients finally died.

. . . Where medicines have once been in use, and afterwards fall into disrepute, as was the case with the Foxglove, I suspect the cases in which they were useful, to have been either few or doubtful, and that the cases in which they had done harm, were so much more numerous and unequivocal, as justly to banish them from the materia medica.—Benjamin Rush: Medical Inquiries and Observations. Philadelphia, T. Dobson, 1797, p. 173.
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