SPECIAL ARTICLE

His Bundle Electrogram

A Critical Appraisal of its Uses and Limitations

By Benjamin J. Scherlag, Ph.D., Philip Samet, M.D.,
and Richard H. Helfant, M.D.

RECENTLY, a simple technic for electrode catheter recording of consistent and stable His bundle activity was described, first in the dog1,2 and then in man.3 In the short time since the introduction of this technic there have been numerous reports utilizing His bundle recordings. These studies have confirmed many of the earlier ingenious deductions made by clinical electrocardiographers utilizing the surface electrocardiogram alone. The His bundle electrogram, in conjunction with the surface electrocardiogram, has provided a more accurate means of localizing the site of conduction abnormality in patients with various forms of heart block.1-10 In addition, this technic has been utilized to study various physiologic20-24 and pharmacologic25-28 interventions, arrhythmias,29-31 and the Wolff-Parkinson-White syndrome.32,33

Published reports have contained a variety of terms applied to the various intervals representing conduction through the atrioventricular transmission system and procedures for quantitating these conduction measurements. In view of this lack of uniformity, this report will suggest standardization of nomenclature and methods of making conduction measurements as well as a rationale for their use. In addition, several methods of validating the His bundle deflection have been suggested, and the importance of such verification has not been widely emphasized. Furthermore, the electrophysiologic interpretations based on His bundle electrograms which have appeared in several recent publications will be critically reviewed.

Methods

His bundle electrograms have been recorded with the use of standard bipolar pacing catheters or special multipolar recording catheters with ring electrodes 5 or 10 mm apart. The catheter is introduced from the femoral vein into the right heart and stabilized at the A-V ring at the base of the posterior tricuspid leaflet. The details of the recording technic, as well as the devices used for recording these electrograms, have been described previously.3

Nomenclature

The use of nomenclature in reports utilizing His bundle electrograms has not been uniform. Even the terminology to describe the technic has not been generally agreed upon by investigators in the field. Two terms have been suggested in the literature to describe the His bundle recording technic. Castellanos et al.34 used the term “His bundle-electrocardiography.” On the other hand, Massumi et al.35 used the term “His bundle-electrography.” Both of these terms are essentially
correct, but it is important here to differentiate between the terms electrocardiogram and electrogram. An electrocardiogram is the electrical recording from the surface of the body or in the proximity of the heart, but not directly in contact with the heart. An electrogram is a recording from the heart surface, either epicardial or endocardial. The bipolar recording of His bundle activity is an electrogram obtained from the endocardial surface of the heart with an electrode catheter. However, its usefulness is dependent upon the simultaneous electrocardiograms displayed with the His bundle electrograms; therefore, we believe the term His bundle-electrocardiography is more appropriate.

Figure 1A shows a recording of His bundle activity with three standard ECG leads in a dog. Another bipolar catheter in the right atrium near the sinus node records atrial activity at the onset of the P wave. The His bundle electrogram
HIS BUNDLE ELECTROGRAM

consists of three distinct deflections. The intervals depicted are identical to those seen in man except for the absolute values shown. The first deflection represents atrial activity (A) which is coincident with the middle portion of the P wave and corresponds to local atrial activity recorded in the area of the A-V node and His bundle. The second deflection which occurs during the P-R segment is the His bundle deflection (H). This is a rapid bi- or triphasic potential, occurring between the P and QRS waves. The third deflection seen in the His bundle electrogram represents ventricular activity (V) as recorded at the A-V junction. The His bundle electrogram, in conjunction with the three standard ECG leads, allows a division of the P-R interval into three components: 1) P-A interval = the time from the earliest onset of the P wave, as seen in any of the surface electrocardiograms, to the onset of atrial activity in the area of the A-V junction as seen in the His bundle electrogram. This interval is taken as a measure of a representative portion of intraatrial conduction, specifically the conduction time from the area of the S-A node to the area of the A-V node during normal sinus rhythm. (It is recognized that the atrial activity recorded on the His bundle electrogram represents local as well as contiguous atrial activation. Therefore, the onset of the A wave is taken as the first rapid excursion from the isoelectric line at an angle of 45° or greater.) 2) A-H interval = the time from the beginning of the A wave to the onset of His bundle activity. This interval is taken as a measure of A-V nodal conduction. 3) H-V interval = the time from the onset of His bundle activity to the earliest onset of ventricular activity as seen on any of the ECG leads or the bipolar His bundle electrogram. This interval includes conduction through the His bundle and bundle branches to the point where the bundle branches activate working ventricular muscle.

P-A Interval

Several reports have not considered the P-A or intraatrial conduction between the upper and lower right atrium in studying A-V conduction, whereas others have stressed its importance. It has been found that the separation of intraatrial conduction from conduction through the A-V node can occasionally be of critical importance in the localization of ativoventricular conduction disturbances. In figure 2, electrical recordings from a patient studied in the clinical labora-
tory* reveal a normal A-H time (88 msec) and a moderate delay in the H-V time (63 msec). Adding these to a normal P-A time (50 msec, upper limits of normal) would yield a P-R interval of 200 msec, which is within the normal limit. Instead the P-R interval, which is variable, ranges from 286 to 301 msec due mainly to an intraatrial conduction defect. Note that the P-A interval ranges from 135 to 150 msec seen during sinus rhythm. In figure 2B, atrial pacing validates both the prolonged and variable intraatrial conduction interval as well as an A-H time which is still within the normal range despite the stress of atrial pacing. This patient did not show Wenckebach cycles until the atrial pacing rate was increased to 150 beats/min. Figure 2C shows validation of the His bundle potential by His bundle pacing. Note that with low atrial pacing there is a prolonged pacing impulse PI-A interval of 195 msec, again indicative of an intraatrial conduction defect.

A-H Interval

This interval can only be an approximation of A-V nodal conduction because the actual onset of A-V nodal activity cannot be accurately determined as yet. In our initial study, and in subsequent reports the term P-H interval has been used as a measure of A-V nodal conduction. We now believe that the A-H interval represents a more accurate measure of transmission through the A-V node, since the A wave of the His bundle electrogram represents local atrial activity in the immediate vicinity of the A-V node and, therefore, eliminates the intraatrial conduction between the SA node area and low right atrium. In addition, the A-H interval allows a comparison of A-V nodal conduction time during both sinus rhythm and atrial pacing because the atrial deflection used in the measurement is not ordinarily altered by the pacing site. This is in contrast to the P-H measurement, which cannot be accurately compared under these

*Clyde D. Schoenfeld, M.D., at the Mount Sinai Hospital, Miami Beach, Florida.
The contribution of an intraatrial (P-A) conduction defect to the A-V conduction disturbance in a patient with first-degree heart block. Each panel shows a bipolar His bundle and/or right bundle electrogram (BE, Hb/Rb) and simultaneously recorded ECG leads I (L-1), aVF, and V1. (A) During sinus rhythm the onset of the P wave (dotted line) was dissociated from the preceding T wave by an early ventricular premature beat (VPC). The estimated P-R interval varied from 286 to 301 msec due to a variable P-A interval (135–150 msec); A-H = 88 msec and H-V = 63 msec in each of the last three beats. (B) Atrial pacing (100 beats/min) from a midatrial site produced a variable interval from the pacer impulse (PI) to A wave (PI-A = 150 msec); A-H = 95 msec; and H-V = 63 msec. (C) Pacing from the His bundle electrodes while recording a bipolar atrial electrogram (BE, RA) produced low atrial or A-V nodal pacing (first three beats) and effective His bundle pacing in the last two beats. Note that the pacer impulse (PI) to R-wave interval in the latter beats is 65 msec, the same as the measured H-V time (A, B). Again, note the prolonged latency from PI to
circumstances, i.e. the pacer impulse is not usually applied at the site of spontaneous impulse formation, the sinus node, but at a different site in the right atrium.

**H-V Interval**

The terms H-V and H-Q have been used to describe the conduction time through the His-Purkinje system. The term H-V would appear more appropriate since the measurement is actually taken from the onset of His bundle activity to the earliest recordable onset of ventricular activity (V) whether or not a Q wave is present in the ventricular deflection utilized. The earliest onset of ventricular activity may be in the electrogram itself, which has no recognizable Q wave, or from an electrocardiogram with no Q wave in the lead suggested, i.e. LBBB.

**Validation of His Bundle Recordings**

Since the precise anatomic location of the recording electrode on a catheter cannot be ascertained by fluoroscopy, a recorded deflection within the P-R segment must be verified as truly representing His bundle activity. The methodology and rationale for this validation have been previously published.\(^1\), \(^2\), \(^3\), \(^4\) Several criteria must be met to validate the recording as truly originating from the His bundle. In order to demonstrate this specificity stimulation of the His bundle using the recording electrode catheter should be performed.\(^2\), \(^9\) Standard safeguard procedures routinely utilized during temporary transvenous pacing are employed during His bundle pacing. Under these circumstances His bundle pacing is no more difficult or hazardous than pacing from any other portion of the right or left ventricle. In practice His bundle pacing has been performed in scores of animal studies, as well as in several hundred patients, without any untoward effects, i.e. repetitive firing or ventricular fibrillation. Pacing the His bundle from the right heart in man or, as seen in figure 1B, from the aortic root in the dog,\(^40\) produces capture of the ventricles with the same QRS morphology in all ECG leads as that seen during sinus rhythm or atrial pacing. This indicates that the point of stimulation was proximal to the right or left bundle branch and must therefore be located in the His bundle or the A-V node. Note also in figure 1B that the interval from the pacer impulse (PI) to the onset of ventricular activity (PI-R) is the same as the interval from the recorded His bundle spike to ventricular activation during sinus rhythm or paced atrial rhythm. If this interval is constant over a wide range of heart rates, a pacing site in the A-V node can be eliminated\(^39\) since conduction velocity in any part of the A-V node decreases with increasing rates. On the other hand, conduction in the His-Purkinje system is virtually unaffected by heart rate.

That these criteria apply in patients with severe disease of the His-Purkinje system, as well as in normal patients, is indicated in a recent study by Narula et al.\(^39\) In particular, His bundle pacing was performed in a patient showing right bundle-branch block and left-axis deviation, and an H-V time of 95 msec. The QRS complexes in leads I, II, and aV\(_F\) were reproduced, and the pacer impulse to ventricular activation time exactly duplicated the H-V time of 95 msec (their fig. 5\(^{39}\)). During this study there were periods of left bundle-branch block (their fig. 6), again with the appropriate QRS complexes in all three leads and an H-V time of 90 msec. Pacing from the His bundle at this time reproduced the same QRS configuration and duration in all three leads as seen during sinus rhythm.

It has been suggested that the production of functional right bundle-branch block by coupled atrial pacing allows differentiation of the His and right bundle potential.\(^41\) Although this approach may be theoretically valid, one cannot be certain that the induced block is not in the distal right bundle and that the first recorded potential arises from the more proximal portion of the right bundle branch.

---

RA electrogram of 195 msec in all leads indicative of an intraatrial conduction defect. Interval between time lines = 1 sec.

*Circulation, Volume XLVI, September 1972*
Table 1

Normal Values of A-V Conduction Intervals in Man

<table>
<thead>
<tr>
<th>Reference</th>
<th>Basal HR (beats/min)</th>
<th>Conduction time (msec) (mean ± SD)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Narula et al.</td>
<td>67 ± 10</td>
<td>172 ± 11 43 ± 14 88 ± 21 41 ± 4</td>
</tr>
<tr>
<td>Damato et al.</td>
<td>71 ± 8</td>
<td>167 ± 16 116 ± 14* 51 ± 6</td>
</tr>
<tr>
<td>Bekheit et al.</td>
<td>64 ± 15</td>
<td>154 ± 19 37 ± 11 78 ± 18 37 ± 5</td>
</tr>
</tbody>
</table>

Abbreviations: Basal HR = normal sinus rate; P-R = intraatrial, A-V nodal, and His-Purkinje conduction time; P-A, A-H, H-V: see text for discussion.

*This value represents the P-H interval consisting of P-A + A-H. See text for discussion.

Normal Values for A-V Conduction

At the time of this writing there have been three published studies in which recordings from the His bundle have been obtained in 51 adult patients with normal A-V conduction, i.e., a P-R interval of 200 msec or less. In table 1 the statistical analyses of the values from the various studies are shown. It can be seen that the values from the study by Narula et al. as well as those from the studies of Bekheit et al. are closely comparable. In addition, if one assumes an average P-A time of 40 msec for the data for heart rate, P-R, P-A, and A-H intervals in all three studies also show good agreement. The average value for the H-V interval and the upper and lower limits (± 2 standard deviations) in the last two studies also are in close accord: 41 ± 8 msec vs 37 ± 10 msec (range 33–49 msec vs 27–47 msec, respectively). In the study by Damato et al., the H-V intervals averaged 51 ± 12 with a range of 39–63 msec. Other laboratories have reported normal values of 35–55 msec and in a more recent report Damato and his group have indicated an average value of 45 msec for the normal H-V time in patients. However, the tabulated data used to determine these values have not been published.

It is probable that the difference in reported normal values for the H-V time may be due, at least in part, to the various methods employed to measure the H-V interval. In the study by Narula et al. the H-V was measured from the onset of the His bundle deflection, which is validated by His bundle pacing, to the earliest ventricular activation as seen on the His bundle electrogram or any of the standard ECG leads. In the study by Bekheit all of the various intervals are measured on the His bundle electrogram except for the beginning of P-wave activity which is taken from the one standard ECG lead recorded. In the study by Damato et al., the H-V interval or the H-Q interval was measured “from the His deflection to the Q wave of the QRS complex.” It has been our experience that the use of one lead may not indicate the earliest ventricular activity and therefore the H-V measurements are usually overestimated. When the QRS is narrow, the earliest ventricular activation usually can be observed on the His bundle electrogram (fig. 1A). However, when intraventricular conduction defects are seen, the measurement of the H-V time from the His bundle electrogram may not indicate earliest ventricular activity.

The determination of normal limits is of more than academic interest. Basic studies as well as recent clinical results indicate the close relationship between prolongation of the H-V time and the existence of partial or complete bilateral bundle-branch block. Other reports have shown a correspondence between abbreviated A-H or H-V intervals and anomalous A-V conduction.

Ventriculoatrial Conduction

When ventriculoatrial conduction is studied identification and validation of His bundle recordings during retrograde transmission must take certain physiologic principles into account. Figure 3 shows a recording of a bipolar His bundle electrogram from a dog as well as bipolar recordings from the sinus node area and the standard ECG leads, I, II, and aV_R. The H-V time during sinus rhythm was
Identification and validation of the retrograde His bundle deflection recorded with an electrode catheter in the aortic root in the dog. Traces from above: Three ECG leads I (L-1), II (L-2), and aV_R, and bipolar catheter recordings from the area of the sinus node (BE, SA) and His bundle (BE, Hb). The latter shows atrial (A), His bundle (Hb), and ventricular (V) activity. (A) During normal sinus rhythm at a heart rate of 110 beats/min the A-H interval = 50 msec and H-V interval = 30 msec. (B) Pacing at a rate of 160 beats/min from the recording catheter at the His bundle (Stimulus, Hb) produced normal ventricular activation in all three leads and the pacer impulse (PI) to onset of the R wave was 30 msec, identical to the previously measured H-V time in A. Retrograde conduction to the sinus node area electrogram was constant in all cardiac cycles (PI-SA = 125 msec). (C) Pacing from the posterior left ventricular epicardium at the same rate as in B (160 beats/min) allowed the identification of two deflections on the His bundle electrogram which resembled the antegrade His deflection seen in A. See text for discussion.

30 msec. Using an electrode catheter positioned in the aortic root, validation of the His bundle recording is shown in panel B during His bundle pacing at 160 beats/min. Selective depolarization of the His bundle produced retrograde conduction to the sinus node area. The three standard ECG leads show the same QRS morphology when the His bundle was depolarized as they did during sinus rhythm. The interval from the pacer impulse to the onset of earliest ventricular activity (PI-R) is 30 msec, the same as the H-V time measured previously. Note, in addition, that the time from the His bundle stimulus to the sinus node electrogram is 125 msec. In panel C ventricular pacing is performed from a posterior left ventricular site, midway between the base and apex of the heart. On the basis of the morphology alone, there are two deflections in the His bundle electrogram which are
similar to the His bundle deflection recorded during sinus rhythm in panel A. However, the time from the first deflection, retrograde, to the sinus node electrogram is 125 msec, the same retrograde conduction time as seen during stimulation of the His bundle itself in panel B. This evidence would indicate that only the first deflection is, in fact, the His bundle deflection during retrograde ventriculoatrial conduction. The other deflection recorded after ventricular activity is retrograde atrial activity recorded in the His bundle-A-V node area.

In contrast to this quantitative validation of the His bundle recording, recently reported studies dealing with the phenomenon of ventricular conduction identified deflections as His bundle activity without substantial validation. It should be emphasized that recordings of retrograde His bundle potentials with standard electrode catheters do not ordinarily allow easy separation of H potentials from ventricular activity. Therefore, the similar shape and duration of the deflection as it appears during ventricular pacing is an unreliable means of identification of the His bundle potential. In figure 4, from a recent report on ventriculoatrial conduction, panels are shown in which ventricular beats with varying prematurity are induced in patients during sinus rhythm. Recorded His bundle electrograms revealed a P-H time of 75 msec and an H-V time of 45 msec during antegrade conduction. In panel C, during retrograde conduction, the His bundle is identified as a
deflection which occurs well after the ventricular pacemaker stimulus and the ventricular deflection (145 msec from the preceding pacing impulse) with a retrograde H-P time of 50 msec. Since the atrial activity was measured high in the right atrium, the H-A time or retrograde conduction time through the A-V node was less than 50 msec. This is inconsistent with the fact that the beat in question was premature and the antegrade P-H time was 75 msec. Since artifactual deflections resembling the His potential are often seen and confusion of His bundle and low atrial activity is possible (fig. 3C), the identification of the His bundle potential during ventriculoatrial conduction should be quantitatively validated. Quantitative validation on the basis of known physiologic properties of the tissues involved in ventriculoatrial conduction makes proper identification more certain whereas the use of the appearance of the recorded deflection alone is unreliable.

Differentiation of Ventricular Beats from Supraventricular Beats with Aberrant Conduction

The differentiation of ventricular beats from aberrant supraventricular QRS complexes has been reported, based in large part on whether or not a His bundle deflection precedes the ventricular complex being analyzed. In addition to this criterion, however, the temporal relationship of the preceding His bundle deflection to the subsequent ventricular complex must be taken into consideration. The normal response of the His-Purkinje system to premature atrial stimulation has been extensively studied. Damato et al., in figure 5, showed the response of the His-Purkinje system to atrial premature beats delivered at varying intervals after the previous cardiac cycle. Slight degrees of aberration can be seen even with no prolongation of the H-V or H-Q times. However, more marked aberration occurs with the prolongation of the His-Purkinje conduction time from 45 to 65 msec. Finally, block is shown distal to the recorded His bundle deflection. This property of the His-Purkinje system during premature activation was confirmed in another study by Narula et al. (their fig. 8). Therefore, the normal response to atrial premature or His premature beats, which result in aberrant ventricular conduction, is either an unchanged or prolonged H-V time.

However, in a reported case of atrial fibrillation in which the second beat is preceded by a His bundle deflection (fig. 6) it
In this clinical case of atrial fibrillation the first, fourth, and sixth complexes represent the normally conducted beats, each of which is preceded by a His deflection. The second complex, which is also preceded by a His deflection, represents an aberrant beat. The third complex is a premature ventricular contraction. No His potential precedes this beat.\(^7\)

is stated that “aberrantly conducted beats were easily distinguished from premature ventricular beats by the His bundle deflection which preceded the former.”\(^7\) The second complex in figure 6 is designated as a supraventricular beat conducted from above with aberrant ventricular activation. However, the H-V time is clearly shorter in this beat than in the normally conducted beats 1, 4, and 6. Note that the H-V time is measured from the H deflection to the earliest ventricular activation, which in this case is in the standard ECG lead; in the normally conducted beats, the earliest ventricular activation occurs in the His bundle electrogram. It should be noted that, as Lau et al.\(^7\) indicate, the recording of His bundle activity is a “valid method for diagnosing aberrant conduction,” particularly in differentiating supraventricular beats from ventricular premature contractions or ventricular tachycardia. However, the differentiation must be made on a quantitative as well as a

Simultaneous recording of leads I, II, and V\(_1\), together with His bundle potentials recorded from the main His bundle. Note that the His bundle potentials (arrows) precede not only the QRS of the sinus beats (1, 2, 4, 6), but also the interpolated extrasystoles (3, 5, 7). The aberrant intraventricular conduction of the first interpolated extrasystole (no. 3) suggests impaired conduction through the right bundle branch and the superior division of the left bundle branch. Our localization of the extrasystolic focus within the His bundle is based on the observation that extrasystoles 3, 5, and 7 are preceded by His bundle potentials. (Reprinted from Amer J Med, by permission.\(^29\))
HIS BUNDLE ELECTROGRAM

qualitative basis. Thus in figure 7, in a recent report by Massumi,29 the first two beats are sinus beats, showing atrial, His bundle, and ventricular activity. The third beat shows a QRS complex which indicates "incomplete right bundle branch block and left axis deviation suggesting impaired conduction through the right bundle branch and superior division of the left bundle branch. Our localization of the extrasystolic focus within the His bundle is based on the observation that this extrasystole is preceded by a His bundle potential."29 However, the H-V time for this third beat is markedly shortened compared to the H-V time seen during sinus beats. Another interpretation of these observations can be determined from the recording made in a patient by Narula et al.17 in the clinical laboratory, at Mount Sinai Hospital in Miami Beach, during simultaneous His bundle and left bundle recording. Figure 8 shows a simultaneous recording of His bundle and left bundle deflections during sinus rhythm in a patient with a normal QRS complex. The second beat in panel A shows left bundle activity preceding His bundle activity during a ventricular premature contraction, probably arising in the area of the proximal left bundle. Panel B shows a series of beats arising in the same region with left bundle activity preceding His bundle activity. Ordinarily, His bundle activity precedes ventricular activation by 50 msec when the patient is in sinus rhythm. Without the left bundle recording, one could interpret the aberrant beats with a shortened H-V time as beats arising in the His bundle which were conducted through the ventricles aberrantly.

Acknowledgment

We thank the Mount Sinai Medical Staff for referral of patients for study, and especially Dr. Clyde D. Schoenfeld for permission to use the recordings shown in figure 2. In addition, we are grateful to those authors cited for their permission to reproduce the selected figures herein, as well as the permission of the American Heart Association, Inc., and the American

Figure 8

Retrograde activation of the BH by premature "ventricular" beats (probably left bundle beats).17

Circulation, Volume XLVI, September 1972
References


Circulation, Volume XLVI, September 1972