Recording of Specialized Conducting Fibers (A-V Nodal, His Bundle, and Right Bundle Branch) in Man Using an Electrode Catheter Technic


SUMMARY
Extracellular action potentials of the A-V node (N), His bundle (H), and right bundle branch (RB) were recorded in subjects with an electrode cardiac catheter which was fluoroscopically positioned across the tricuspid valve. The N potential is a slow diphasic wave occurring between the atrial electrogram (P) and the H potential. It is characterized by slurring or notching on the upstroke. The H potential, as previously described, is a biphasic or triphasic wave of 15 to 20-msec duration. The RB potential is a fast biphasic wave of 10 to 20-msec duration occurring between the H potential and the Q wave. During single atrial pacing and premature atrial stimulation, the A-V conduction delay could be localized to the N-H interval. Evidence suggests that during aberrant ventricular conduction of the right bundle-branch block type the impulse was blocked proximally in the right bundle. During concealed conduction the nonconducted impulse was completely blocked within the A-V node or the N-H interval.

Additional Indexing Words:
Premature atrial stimulation
Temporal relationships of electrograms
Atrial electrogram
Ventricular electrogram
Heart rate

In the experimental animal, the recording of electrical activity of the specialized conducting tissues (A-V node, His bundle, right and left bundle branches, and Purkinje fibers) has been achieved with a variety of technics. These studies have contributed greatly to our understanding of the nature of the transmission of impulses from the atrium to the ventricle. Until recently, most observations concerning atrioventricular (A-V) conduction in man have been limited to an analysis of the P-R interval of the standard electrocardiogram. A method for the systematic recording of His bundle electrograms in man has recently been reported from this laboratory. The purpose of this present report is to describe the application of this technic for recording the electrical activity of the A-V node and right bundle branch, as well as the His bundle in man.

Methods
Right heart catheterization was performed on 15 subjects who were in the post-absorptive, nonsedated resting state. Under local anesthesia, an electrode catheter (6 or 7F) was introduced...
Figure 1

The top three tracings are simultaneously recorded standard ECG leads. The bottom tracing is a His bundle electrogram (HBE). The arrow points to a small biphasic deflection (N) which appears between the atrial (P) and His bundle (H) electrograms. The paper speed is 200 mm/sec.

Figure 2

Simultaneous ECG and His bundle electrogram (HBE) recordings at a paced atrial rate of 100/min. PI marks the stimulus artifact; P is the atrial electrogram, N is the nodal potential, H is the His bundle potential, QRS is the ventricular electrogram. The N potential measures 30 msec and is separated from the H potential by 21 msec.

*Elecath Corp., Linden, New Jersey.
†Electronics for Medicine, White Plains, New York.
the tricuspid valve and then slowly withdrawing it until a biphasic or triphasic spike appears between the atrial and ventricular electrograms and within the P-R segment. For the simultaneous recording of right bundle-branch activity and His bundle activity, the best results were obtained by using a six-pole electrode catheter. The design of the catheter was such that each electrode was 2 mm wide, and the electrodes were placed 1 cm apart starting at the distal tip of the catheter. Simultaneous His and right bundle activities were recorded by advancing the catheter 1 to 2 cm into the ventricular cavity. The preformed curved tip of the catheter was directed caudad. A few premature ventricular contractions usually occurred when the catheter was first introduced into the ventricular cavity. When the catheter was properly positioned, the premature contractions disappeared and stable recordings could be obtained for periods of 2 hours. The following measurements were made in milliseconds from tracings obtained at a paper speed of 150 to 200 mm/sec:

1. P-N interval—the interval between the P wave and the nodal potential.
2. P-H interval—the interval between the P wave and the His bundle potential.
3. N-H interval—the interval between the nodal potential and His bundle potential.
4. H-Q interval—the interval between the His

**Figure 3**

*Tracing obtained at a paced atrial rate of 110/min. Labeling is the same as figure 2. There is definite slurring of the upstroke of the N potential. The N-H interval has increased to 27 msec.*

**Figure 4**

*Records obtained at a paced atrial rate of 140/min. Slurring of the upstroke of the N potential is evident. The duration of the N potential measures 40 msec, and the N-H interval has increased to 32 msec.*

*Circulation, Volume XXXIX, April 1969*
His bundle recordings have been obtained in all but one of 80 subjects thus far studied in our laboratory. Right bundle-branch activity was recorded in 12 of 15 subjects, and A-V nodal activity, in 10 of 15 subjects in whom this was attempted.

Results

Electrical Activity of the A-V Node

The identification of the A-V nodal potential and its relationship to the His bundle potential are illustrated in figures 1 to 7.

Figure 1 is a record obtained at a sinus rate of 72/min. A small positive-negative deflection (marked N) appears between the atrial and His bundle electrograms. From this tracing alone, a positive identification of a nodal potential cannot be made. However, at an atrial paced heart rate of 100/min (fig. 2), the positive-negative deflection becomes evident.

Figure 5

A magnified reproduction of the first beat of tracing depicted in figure 3. There is slurring of the upstroke of the N potential.

5. RB-Q-interval between the right bundle-branch potential and the Q wave of the QRS complex.

Figure 6

A magnified reproduction of the first beat of tracing depicted in figure 4. Slurring of the upstroke of N is most obvious.

Figure 7

The effect of premature atrial stimulation on A-V nodal conduction. At a coupling interval of 360 msec the premature atrial beat increases the P-H interval from 81 msec to 97 msec. The major portion of this delay occurred in the N-H interval.
and a finite period now separates it from the H potential. The N-H interval measures 21 msec. The duration of the N potential at this paced heart rate is 30 msec, and there is a suggestion of slurring or notching on the upstroke of the N potential. Definite slurring or notching on the upstroke of the N potential can be seen in figure 3 which represents a tracing obtained at a paced heart rate of 110/min. The duration of the N potential has not changed, but the N-H interval has increased to 27 msec at this higher heart rate.

A tracing obtained at a paced rate of 140/min is shown in figure 4. The slurring of the upstroke is most evident at this heart rate. The upstroke of the N potential appears to be flattening out or becoming more iso-electric. The duration of the N potential now is 40 msec, and the N-H interval has increased to 32 msec. Magnified reproductions of the N potential, recorded at rates of 110/min and 140/min, are depicted in figures 5 and 6, respectively. The recording of the A-V nodal potential, during premature atrial stimulation is illustrated in figure 7. The first beat is a sinus beat. A small deflection (marked N) can be seen between the atrial (P) and His-bundle (H) electrograms. The N potential with its slurred upstroke is more easily identified following the premature atrial beat. The increase in the P-H interval during the premature atrial beat is accounted for by an increase in the N-H interval.

Table 1 lists the intervals between the P wave and the nodal potential (P-N interval) and between the nodal potential and the His-bundle deflection (N-H interval) at various heart rates. As shown in this example, the greater delay in transmission of impulses occurs between the nodal and His potentials as the heart rate is increased. From a paced atrial rate of 90/min to 160/min, the P-N interval increased 11%, whereas the N-H interval increased 237%. The H-Q intervals remained constant over the range of heart rates studied. Similar findings were obtained in nine other cases in which the nodal and the His potentials were simultaneously recorded.

An example of concealed conduction is depicted in figure 8. In the top panel the right atrium is being electrically stimulated at a rate of 100/min. For each atrial impulse there is a ventricular response. The P-H interval measures 104 msec, and the H-Q interval 47 msec. In the bottom panel the right atrium is being stimulated at a rate of 200/min. There is a 2:1 block with a ventricular response of 100/min. A pacemaker impulse (PI) also occurs in the downstroke of the QRS complex. Every other P wave is blocked. The blocked P waves are followed by a widened nodal potential (N), indicating that the level of block was either within or just distal to the A-V node. The blocked P waves affect subsequent conduction as evidenced by the fact that the P-H interval of the conducted beat is 197 msec.

**Electrical Activity of the Right Bundle Branch**

Recordings of the electrical activity of the right bundle branch using a tripolar electrode catheter are shown in figures 9 and 10. The right bundle-branch potential (RB) is a sharp biphasic spike of 10 to 12-msec duration. In comparison, the His bundle spike is usually of 15 to 20-msec duration. In all of our recordings the interval between the right bundle potential and the Q wave of the QRS complex (RB-Q interval) was between 12 and 18 msec, whereas the H-Q interval varied between 30 and 60 msec. In figure 9 the RB potential is recorded at a sinus rate of 72/min. The RB-Q interval is 18 msec. In figure 10 the His and right
Figure 8

Demonstration of concealed conduction. In the top panel the right atrium is being paced at a rate of 100/min. The P-H interval measures 104 msec. In the bottom panel, the atrium is being paced at a rate of 200/min. There is 2:1 block, and the ventricular rate is 100/min. There is a pacer impulse (PI) on the downstroke of each QRS complex. The nonconducted beats are blocked within or just distal to the A-V node (N) and cause the subsequent P-H intervals to be increased to 197 msec.

Figure 9

Recording of the right bundle (RB) potential during sinus rhythm. The interval between the RB and the onset of the O wave of the QRS complex is 18 msec.
bundle potentials were recorded sequentially at a paced atrial rate of 100/min. In the top panel the PI-H interval measures 162 msec while in the bottom panel the PI-RB interval measures 174 msec. The H-RB interval measures 18 msec.

The simultaneous recording of His-bundle and right bundle-branch activities has been more easily achieved with a six-pole electrode catheter than with a tripolar catheter. A representative tracing is presented in figure 11. The middle tracing records a His bundle deflection, and the bottom tracing records a right bundle-branch deflection. The P-H interval measures 114 msec, and the P-RB interval is 126 msec. The H-Q and RB-Q intervals are
30 and 18 msec, respectively. The H-RB interval is 12 msec.

The electrical activity of the right bundle branch was also studied during coupled premature atrial stimulation. This is illustrated in figure 12. The first and third beats are sinus beats, and the second beat (marked PI) is induced by a premature atrial stimulus.

The premature stimulus produces an aberrant right bundle-branch block pattern. The electrical activity of the right bundle is recorded during the sinus beats (first and third beats) but is not recorded during right bundle-branch block.

**Electrical Activity of the His Bundle**

The recording of the electrical activity of the His bundle in man using the catheter technique has been previously reported. Briefly, it appears as a diphasic or triphasic wave occurring between the atrial and ventricular electrograms. The duration of the His potential averages 15 to 20 msec. The interval from the P wave to the His deflection (that is, the P-H interval) is fairly constant at any given heart rate (+5 msec). The P-H interval increases as the heart rate is increased by electrical stimulation of the right atrium. At any given heart rate stimulation of the carotid sinus...
Figure 13

Effect of increasing the heart rate by atrial pacing on the P-H interval. PI denotes pacer impulse.
and digitalis caused an increase in the P-H interval, while isoproterenol and atropine shortened the P-H interval. The interval from the His deflection to the onset of the Q wave of the QRS complex (H-Q interval) remains constant during all of these interventions. Representative tracings are shown in figure 13.

Discussions

It must be stated at the outset that for obvious practical reasons it was not possible to obtain anatomic correlation of the electrical activity of the various specialized conducting tissues contained in this report. Nonetheless, the similarities between the previously reported experimental data and our recordings lend strong support to the validity of our findings.

In 1958, Alanis and associates recorded the electrical activity of the bundle of His (designated the H potential) in the isolated perfused hearts of dogs. The H potential was described as a diphasic wave appearing between the atrial and ventricular electrograms. These investigators used a variety of interventions to demonstrate that the H potential was not part of either the atrial or ventricular electrograms (increasing the atrial rate by electrical stimulation, stimulation of the vagus nerve, asphyxia, and injections of acetylcholine and epinephrine). Other investigators have recorded the electrical activity of the bundle of His in experimental animals using a variety of techniques. In these studies, the temporal relationships of the H potential to the atrial and ventricular electrograms were demonstrated to be similar to those reported by Alanis and associates, although some slight configurational differences may be noted. The configurational differences are in all likelihood due to the different filter frequencies and recording electrodes used. Recent reports concerning the systematic recording of His bundle activity in man have confirmed the previously reported experimental data.

In 1958, Matsuda and Sano and their associates recorded intracellular action potentials of A-V nodal cells using microelectrode techniques. Sano and co-workers used a marking technic with the intracellular electrodes to demonstrate histologically that the recorded action potentials did arise from the A-V nodal region. The characteristic features of the cellular action potential of the A-V node are described as follows: (1) a slightly lowered resting potential; (2) a slow rise of depolarization; (3) a pre-potential or one or more notches in the upstroke; (4) small or absent overshoot; and (5) slight undershooting following repolarization. These characteristic features of single A-V nodal cells have been contrasted with simultaneous recordings from adjacent atrial and His bundle fibers. Recordings of extracellular action potentials from the A-V node have been described by Van der Kooi and co-workers, Scher and co-workers, Alanis and associates, Sodi-Pollares and associates and Pruitt and Essex. Scher and co-workers have pointed out the difficulties encountered in the interpretation of these various reports. In a study of atrioventricular conduction, Scher's group used multipolar extracellular electrodes to record from the A-V conducting system. From their unipolar recordings, they described a slowly changing potential which was negative in the upper A-V node, positive-negative in the mid-nodal region, and positive in the lower node. The A-V nodal potential was described as a nodal hump which occupied a period of about 20 msec between the excitation of atrial fibers and the common bundle. The A-V nodal recordings presented by Pruitt and Essex are similar to those of Scher and co-workers.

Using different recording technics, Alanis and co-workers described the nodal potential (N) as a diphasic wave of slow temporal course appearing between the atrial electrogram and the His potential. These investigators described two areas of latency or delay during the transmission of impulses along the A-V conducting system. One area of latency occurred between the atrium and the A-V node and the greater latency occurred between the nodal (N) and His (H) potentials.

One of the difficulties concerning the interpretation of A-V nodal potentials is the degree of cellular injury which results when electrodes are either directly impaled into or sewn over...
this region. We believe that the electrode catheter technic, as used in this study, produces no significant cellular injury. In support of this is the fact that His bundle activity has been successfully recorded in 79 of 80 patients thus far. The temporal relationships, configuration, and response to various physiologic interventions of the His potential have been the same in all cases. Such consistency would not be expected if the recorded electrical activity resulted from a nonspecific injury potential. The same applies to the 12 of 15 instances in which the electrical activity of the right bundle branch has been recorded.

The configuration and temporal relationships of our recorded N potential lend additional support to its being a valid reflection of the true A-V nodal potentials. Our extracellular recordings indicate that the N potential is a diphasic wave with steplike notches on the upstroke. Others have also reported that the extracellular N potential is a diphasic wave.

Intracellular action potential recordings from single nodal fibers have demonstrated a “slow foot” or “pre-potential” occurring before the fast rise phase. Under certain conditions a progressive increase in slurring and notching of the upstroke has been demonstrated. In figures 2, 3, and 9 in an article by Hoffman,16 slurring and notching of the transmembrane action potential is depicted in records obtained following the infusion of acetylcholine and premature atrial stimulation. Paes de Carvalho17 (fig. 8 of his article) has demonstrated how the “slow foot” of the nodal action potential may develop into definite steps occurring in the upstroke as a result of decremental conduction. Likewise, Watanabe and Dreifus18 have shown notching of the upstroke of nodal-fiber action potentials during delayed A-V conduction. The slowing and notching depicted in these references appear identical to the notches of our own extracellular recordings. We have come to rely heavily on this characteristic for identifying the extracellular N potential in our clinical recordings.

The exact cause of the notching of the nodal potential is not known. Watanabe and Dreifus18 have suggested that inhomogeneous conduction or fragmentation of the excitation front within the A-V junctional tissue may in part explain this phenomenon. The complexity of the A-V junctional region as determined histologically by James19 and Truex20 lend support to the hypothesis of inhomogeneous conduction. It has been suggested that under normal conditions minor degrees of inhomogeneity may not be significant. However, marked degrees of fractionation of the wavefront may occur once conduction through the A-V node is slowed. In our tracings a greater degree of notching occurred on the upstroke of the N potential as A-V conduction was progressively slowed by increasing the paced atrial rate.

It might be argued that the diphasic wave which we have identified as the N potential is the atrial repolarization wave. This consideration appears unlikely for the following reasons: The atrial T wave is normally located within or soon after the inscription of the QRS. Our recordings clearly indicate that at sinus rhythm the N potential is contiguous with the atrial electrogram and separates from it during atrial pacing. Furthermore, notching is not a characteristic of the atrial repolarization wave. Finally, if the N potential were due to atrial repolarization, then one would expect our recordings to demonstrate the larger ventricular repolarization wave (T wave), but they do not.

It is generally believed that the greatest delay in A-V conduction occurs in a narrow zone at the junction of the atrium and atrioventricular node. Alanis and co-workers2 have suggested that two areas of delay are encountered during transmission of impulses between the atrium and ventricle. One area of delay is between the atrium and the A-V node while the other and greater delay occurs between the A-V node and the His bundle. Our findings are in agreement with those of Alanis and co-workers. The P-N interval increased only 11% from a paced atrial rate of 90/min to 160/min, whereas the N-H interval increased 237% over the same range of heart rates. Part of the delay in N-H conduction is accounted
for by an increase in the duration of the N potential.

The electrical activity of the bundle branches and Purkinje network have been recorded in the experimental animal by a number of investigators. In 1953, Burchell and associates recorded the electrical activity of either the bundle branches or Purkinje network using plunge needle electrodes. Sodi-Pollares and co-workers recorded from various portions of the specialized conducting tissue by using bipolar plunge electrodes in the in situ dog heart preparation. Hoffman and co-workers used endocardial plaque electrodes sewn over the ventricular specialized conducting system and obtained bipolar electrograms of the His bundle, right and left bundle branches, and peripheral Purkinje fibers. As stated previously, an anatomic correlation for our right bundle-branch recordings was not practically possible. We are unaware of any previous reports in which right bundle-branch potentials have been recorded in man. Our recordings in man are similar to those obtained in the experimental animal. The validity of the right bundle-branch recordings is further supported by the inability to record such potentials during induced right bundle-branch block (fig. 12). Our recordings of the RB potentials were obtained from that portion of the right bundle which travels in the right ventricular cavity: that is, a few centimeters distal to the common bundle; this portion in turn accounts for a period of several milliseconds separating the H from the RB potential. On three occasions, during the review of our His bundle tracings it was noted that the H potential was composed of a double deflection (fig. 14). In figure 14 simultaneous bipolar tracings were obtained from a tripolar electrode catheter. The most distal pair of electrodes (nos. 1 and 2) were positioned approximately 1 cm into the right ventricular cavity. These electrodes recorded an H and an RB potential which were separated by 2 mm (10 msec) as depicted on the bottom tracing. The second and more proximal bipolar pair of electrodes (nos. 2 and 3) recorded a double deflection as

**Figure 14**

Top tracing is lead V₁ of the ECG. The other two tracings are simultaneously recorded His bundle electrograms. The most distal electrode (no. 1) was positioned 1 cm within the ventricular cavity. The bottom tracing was recorded from electrodes 1 and 2 and demonstrates an RB and an H potential which are separated by 10 msec. The middle tracing was obtained from electrodes 2 and 3 which record a double deflection composed of the H and RB potentials. These potentials are separated by > 5 msec. The RB of the middle tracing represents recordings from the main trunk of the right bundle while the RB of the bottom tracing is recorded at a more distal site along the bundle.
illustrated on the middle tracing. These two deflections (H and RB) are separated by less than 1 mm (> 5 msec). It is believed that the second deflection represents the electrical activity of the most proximal portion of the main trunk of the right bundle. The separation of the His and right bundle potentials as depicted in the bottom tracing could be distinguished during oscilloscopic monitoring while the double deflection as depicted on the middle tracing could not be.

Acknowledgment

The authors gratefully acknowledge the assistance of Anne Mazzella, Theresa Halloran, Audrey Pedersen, Joan Cumming, Michael Morretti, and David Berry.

References

Recording of Specialized Conducting Fibers (A-V Nodal, His Bundle, and Right Bundle Branch) in Man Using an Electrode Catheter Technic

ANTHONY N. DAMATO, SUN H. LAU, WALTER D. BERKOWITZ, KENNETH M. ROSEN and KENNETH R. LISI

doi: 10.1161/01.CIR.39.4.435

Circulation is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 1969 American Heart Association, Inc. All rights reserved.
Print ISSN: 0009-7322. Online ISSN: 1524-4539

The online version of this article, along with updated information and services, is located on the World Wide Web at:
http://circ.ahajournals.org/content/39/4/435

Permissions: Requests for permissions to reproduce figures, tables, or portions of articles originally published in Circulation can be obtained via RightsLink, a service of the Copyright Clearance Center, not the Editorial Office. Once the online version of the published article for which permission is being requested is located, click Request Permissions in the middle column of the Web page under Services. Further information about this process is available in the Permissions and Rights Question and Answer document.

Reprints: Information about reprints can be found online at:
http://www.lww.com/reprints

Subscriptions: Information about subscribing to Circulation is online at:
http://circ.ahajournals.org//subscriptions/