His Bundle Rhythm

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SUMMARY

Traditionally the site of impulse formation in so-called A-V nodal rhythms has been considered to be within the upper, middle, and lower portions of the A-V node. In this study, evidence obtained supports the concept that the His bundle and not the A-V node is the pacemaker site in nodal rhythms. His bundle activity was recorded in clinical cases of nodal rhythms and A-V dissociation by using an electrode catheter positioned at the tricuspid valve. In these studies a single His deflection preceded each QRS complex. From the results of this study, it is suggested that in so-called lower and middle nodal rhythms the pacemaker site is within the His bundle. It is further suggested that so-called upper nodal rhythms may represent a coronary sinus or inferior left atrial rhythm.

Additional Indexing Words:
A-V nodal rhythms Pacemaker site Conduction

Traditionally, A-V nodal rhythms have been described as arising from the upper, middle, or lower portions of the A-V node. In so-called nodal rhythms the P wave is inverted in leads II, III, and aVr and may precede, follow, or be buried within the QRS complex. The P-R or R-P interval has been thought to be dependent upon the region of the A-V node in which the pacemaker is located and also upon the speed of antegrade and retrograde conduction.

The characteristics of the transmembrane action potential of A-V nodal cells, as recorded by micro-electrode technics, suggest that the A-V node itself does not possess properties of automaticity. Accordingly, it has been suggested that the pacemaker in so-called A-V nodal rhythms is located in the N-H region or bundle of His.

The purpose of this study was to obtain evidence supporting the concept that the pacemaker in so-called nodal rhythms is located in the region of the His bundle and not in the A-V node. This was accomplished by use of an electrode catheter technic to record the electrical activity of the A-V node and bundle of His in patients with so-called nodal rhythms and A-V dissociation. The results of this study demonstrate that in these cases the pacemaker was located in the region of the His bundle and not in the A-V node.

Methods

Right heart catheterization was performed in the post-absorptive, nonsedated state. A signed consent was obtained from all of the patients after they were informed of the nature of the study. During local anesthesia, a tripolar electrode catheter was percutaneously introduced into the right femoral vein and fluoroscopically positioned across the tricuspid valve. The electrode catheter was slowly withdrawn until adequate recordings of His bundle and A-V nodal activity were obtained according to methods previously described. His bundle electrograms were recorded simultaneously with standard electrocardiographic leads on a multichannel, oscilloscopic photographic recorder at paper speeds of 100 to 200 mm/sec. Measurements in milliseconds were made of the intervals from the onset of the P wave to the His deflection (P-H interval) and from the His deflection to the onset of the Q wave of the QRS complex (H-Q interval). For retrograde conduction, the interval from the onset of the His
deflection to the end of the P wave was measured (H-P interval). For the right atrial pacing studies a bipolar electrode catheter was percutaneously introduced into an antecubital vein and fluoroscopically positioned against the lateral wall of the right atrium at the junction of the superior vena cava and the right atrium. The right atrium was stimulated by a battery-powered pacemaker* (Model 5837) which delivered impulses of 2-msec duration at approximately twice threshold.

Results

Figure 1 is a record obtained on a 57-year-old male during a "middle nodal" tachycardia and its subsequent spontaneous reversion to normal sinus rhythm (case 1). The top panel (A) represents the tracings obtained at normal sinus rhythm, and the lower panel the records obtained during the nodal tachycardia (labeled His-rhythm). During normal sinus rhythm, the P-H interval measured 102 msec and the H-Q interval 33 msec. In the lower panel, each QRS complex is preceded by a single His deflection. The H-Q interval again measures 33 msec. Atrial activity is not evident. If one assumes that retrograde conduction time during the His rhythm was the same as antegrade conduction time during sinus rhythm, then an interval of 102 msec would cause the P wave to occur within the QRS complex, and it would, therefore, not be visualized.

The second case studied was that of a 62-year-old male with arteriosclerotic heart disease and digitalis toxicity. A standard 12-lead electrocardiogram revealed a slow nodal rhythm (fig. 2). There is absence of sinus P waves which may be due to either sinus arrest or sino-atrial block. Retrograde P waves followed most of the nodal beats. In panel A of figure 3 (labeled His-rhythm) each QRS complex is preceded by a single His deflection. The ventricular electrogram is followed by an

*Medtronic, Inc., Minneapolis, Minnesota.

![Figure 1](http://circ.ahajournals.org/)

(Top panel) Tracings taken at normal sinus rhythm (NSR). (Bottom panel) Tracings taken during a "middle nodal" tachycardia (labeled His rhythm). The H-Q interval during both rhythm strips is the same (33 msec). During the His rhythm the P wave is presumably buried within the QRS complex. ECG = standard electrocardiographic lead. HBE = His bundle electrogram.
A-V nodal potential (N) which in turn is followed by a retrograde P wave (atrial electrogram).

This sequence of events confirms that the recorded P waves are retrogradely conducted since a fortuitously recorded P wave of sinus or ectopic atrial origin would have been followed by an N potential and not preceded by one. In addition, the absence of an N potential preceding the His deflection lends further support to the concept that the origin of impulse formation is in the His bundle and not in the A-V node. The H-Q interval during the His rhythm measures 50 msec while the interval from the His deflection to the end of the retrograde P wave measures 320 msec. Retrograde conduction time varied between 250 and 320 msec. Although this patient had sinus arrest due to digitalis toxicity, antegrade conduction could be easily demonstrated during right atrial pacing. This is illustrated in panel B of figure 3. The right atrium was paced at a rate of 80/min and the P-H interval during antegrade conduction was 135 msec. The H-Q interval during the paced heart rate is the same as during the His rhythm (50 msec).

The recording of an antegrade A-V nodal potential in this same patient during right atrial pacing is illustrated in figure 4.

Figure 5 is a continuous strip from a 52-year-old patient with A-V dissociation (case 3).
Figure 3
His bundle recordings in case 2. (Panel A) His rhythm with retrograde conduction to the atria. Each QRS complex is preceded by a single His deflection (H). The retrogradely conducted P wave is preceded by an A-V nodal potential (N). See text for further discussion. (Panel B) Antegrade conduction during right atrial pacing. H-Q time same as in panel A.

Figure 4
Case 2. Recording of antegrade A-V nodal potential (N) during right atrial pacing. PI = pacer impulse.
HIS BUNDLE RHYTHM

The patient was not taking digitalis, and the exact cause of this temporary rhythm disturbance could not be determined. The atria and ventricles were beating independently. There was right bundle-branch block. Each QRS complex is preceded by a single His deflection. The N potential follows each nonconducted P wave. The level of block for antegrade conduction is proximal to the His bundle, and the pacemaker site for the ventricles is located in the His bundle.

Discussion

The terminology of upper, middle, and lower A-V nodal rhythms was introduced by Zahn. This descriptive terminology is based on the relationship of an inverted P wave (leads II, III, aVF) to the QRS. In upper nodal rhythms the inverted P wave precedes the QRS complex while in lower A-V nodal rhythms the inverted P wave follows the QRS. In middle nodal rhythms the P wave is presumed to be buried in the QRS complex. It was recognized, however, that the relationship of the P wave to the QRS complex is also dependent upon the speed of antegrade and retrograde conduction. Thus, it was assumed that an impulse arising in the upper nodal region, might result in a P wave which is located within the QRS complex or after it, providing retrograde conduction to the atria is sufficiently prolonged. Conversely, a lower nodal rhythm could result in a P wave which precedes the QRS complex if antegrade conduction is prolonged. Since the standard electrocardiogram provides no accurate measurement of the relationship of antegrade conduction to retrograde conduction with regard to the pacemaker site, the term "A-V rhythms" has been used.

One of the electrophysiologic characteristics of cells which function as pacemakers of the heart (actual or latent) is the phenomenon of slow diastolic depolarization (phase 4 depolarization). The transmembrane action potentials of single fibers of the A-V node have been recorded by Matsuda, Sano, and Hoffman and their associates. Some investigators have described a very slow diastolic depolarization phase of A-V nodal action potentials while others state that it is more apparent than real. Ultrastructure studies of the heart have revealed that the A-V node contains a relatively small number of cells which are

Figure 5

His bundle recordings during A-V dissociation. Atrial and ventricular activity appear independent of each other. However, the last beat in the upper strip and the third beat in the lower strip may represent ventricular captures by sinus impulses. Each nonconducted P wave is followed by an A-V nodal potential (N) indicating that the block occurred proximal to the region of the His bundle. Each QRS complex is preceded by a single His deflection (H) indicating that the pacemaker for the ventricles was in the region of the His bundle.
identical to the P cells of the sinus node. The P cells of the sinus node have been postulated to be sites of pacemaker impulse formation. However, pacemaker activity of A-V nodal cells has not been conclusively proven but has only been inferred. On the other hand, automaticity of the His-Purkinje network has been demonstrated by in-vitro studies.

In man, the electrical activity of the His bundle, as recorded by the electrode catheter technic, appears as a rapid biphasic spike occurring within the P-R segment. The A-V nodal potential is a slow biphasic wave, appearing immediately after the atrial electrogram and prior to the His deflection. Positive identification of the N potential is facilitated by procedures which increase the degree of A-V delay, such as single atrial pacing or premature atrial stimulation. Under these circumstances the duration of the N potential increases and slurring or notching of both the ascending and descending limbs becomes more evident.

From the results of the present study it is concluded that the pacemaker site in so-called middle and lower nodal rhythms is located in the region of the His bundle and not in the A-V node itself. This appears certain in at least two of the three cases herein reported (cases 2 and 3). Our conclusion is based on the following: (1) During so-called nodal rhythms a single His deflection preceded each QRS (cases 1, 2 and 3). (2) Retrograde conduction from the His bundle through the A-V node and ultimately to the atria was demonstrated (case 2). (3) During A-V dissociation, nodal and His activity were independent of each other, while His and ventricular activities bore a constant temporal relationship to each other.

The P-R interval of the standard electrocardiogram may be divided into the interval from the onset of the P wave to the His deflection (P-H interval) and the interval from the His deflection to the onset of the Q wave of the QRS complex (H-Q interval). The former interval is a measure of atrioventricular conduction time while the latter is a measure of intraventricular conduction. Studies to date have demonstrated that when A-V conduction is delayed, there is a prolongation of the P-H interval. The H-Q interval remains fairly constant during antegrade propagated responses. Exceptions to the latter statement include aberrant ventricular conduction and the pharmacologic effect of drugs. Therefore, during His rhythms, the H-Q interval would be expected to be comparable to that observed when the pacemaker was located in the atrium. This expected finding was observed in cases in which recordings were obtained during both sinus and His rhythms (fig. 1) and during His and right atrial paced rhythms (fig. 3).

Retrograde conduction from the ventricles to the atria has been demonstrated in a number of clinical studies including situations where permanent antegrade block exists. Retrograde or V-A conduction time may be longer, shorter, or the same as antegrade conduction. In the 23 cases of retrograde conduction reviewed by Winternitz and Langendorf, the R-P intervals ranged from 0.10 to 0.23 sec. None of these 23 cases was due to digitalis administration. Wilson and Robinson found an R-P interval of 0.20 to 0.34 sec, and Kline and associates reported an R-P interval of 0.38 sec. In a report of 15 cases by Kistin and Landown, retrograde conduction time (taken as the interval from the QRS to the intrinsicoid of the retrograde P) was found to range from 0.09 to 0.48 sec. Thus in our case 2, the maximum retrograde conduction time observed (H-P interval of 320 msec) is not unusual. The exact reasons why retrograde conduction is at times longer than antegrade conduction in the same patient is not known.

Upper A-V nodal rhythms (with inverted P waves preceding the QRS) require additional considerations. As stated previously, during normal antegrade conduction the H-Q interval is fairly constant and in normal subjects studied previously this interval did not exceed 60 msec at all heart rates tested. The average H-Q interval for 11 normal subjects was 51 msec (range, 44 to 60 msec). Likewise, during normal A-V conduction the P-H interval at any given heart rate is constant. At
sinus rhythm, the P-H interval averaged 117 msec (range for normal subjects, 96 to 140 msec). Consequently, for an inverted P wave to precede the QRS complex from an impulse arising in the His bundle would require consistently enhanced retrograde conduction from the His bundle to the atria. Although enhanced retrograde conduction could explain the P-QRS relationship, it appears more likely that the pacemaker site in upper A-V nodal rhythms is not located in the His region. One must then ask, "Does the electrocardiographic finding of an inverted P wave preceding the QRS complex represent a true A-V nodal rhythm with the pacemaker site located in some region of the upper A-V node or at the atrionodal junction?" Under these circumstances the atria would be depolarized retrogradely and the ventricles antegrade without the need to invoke significant changes in conduction times. However, the lack of demonstrated automaticity (diastolic depolarization) in atrionodal and nodal cells makes this hypothesis less attractive. In this regard it is important to note that Watanabe and Dreibus reported findings which suggested that, in the isolated rabbit heart, the N H and less frequently the A N regions may serve as pacemaker sites. However, these authors did not discuss the possibility that the points of earliest activation may have resulted from a pacemaker site located in the adjacent coronary sinus. Hence, an alternate, and more attractive possibility is that the so-called upper A-V nodal rhythms are in fact coronary sinus rhythms or inferior left atrial rhythms.

Coronary sinus rhythm is characterized by P waves which are inverted in leads II, III, and aVF and which precede the QRS complex. Lancaster and associates, experimentally produced coronary sinus rhythm in eight subjects by electrically stimulating the coronary sinus os through an electrode catheter. In our laboratory, we have electrically stimulated various parts of the coronary sinus vein (os, middle, and distal areas) in 20 subjects and have always produced electrocardiographic findings which are indistinguishable from clinical tracings of coronary sinus rhythm or so-called upper nodal rhythm. In addition, in all of our cases coronary sinus pacing resulted in an inverted P wave in lead V6. Thus, the combination of inverted P waves in leads II, III, aVF, and V6 fulfill the criteria for either a coronary sinus rhythm or an inferior left atrial rhythm.

A few recent reports have questioned the validity of the polarity of the P wave in coronary sinus rhythm. Moore and associates and Massumi and Tawakkol failed to demonstrate consistent inversion of P waves in leads II, III, and aVF in their studies of coronary sinus rhythm, while Lancaster and associates and we have consistently found P wave inversions in these leads. This discrepancy appears related to the differences in methodology employed. The former groups of investigators electrically stimulated the region of the coronary sinus while the latter investigators stimulated from within the os of the coronary sinus. It would appear therefore that the pacemaker site for a coronary sinus rhythm is somewhere within the os and not in the floor of the right atrium. Since the os of the coronary sinus was not entered, Moore and associates produced P-wave polarity which was not significantly different from normal sinus rhythm. From the results of this study, it is concluded that the pacemaker site in so-called middle and lower nodal rhythms is located in the bundle of His. It is further suggested that the so-called upper nodal rhythm may represent a coronary sinus or inferior left atrial rhythm.

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