A Study of Atrioventricular Conduction in Man Using Premature Atrial Stimulation and His Bundle Recordings

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SUMMARY

In 20 subjects the effects of controlled premature atrial stimulation on atrioventricular conduction was studied using an electrode catheter technic for recording electrical activity of specialized conducting fibers. The least common type of response observed was that in which A-V conduction delay was limited only to the A-V nodal region (type A). This type of response was obtained whenever the conduction delay in the A-V nodal region increased in direct proportion to the prematurity of a propagated atrial impulse. It was characterized by a progressive prolongation in the P-H interval with ultimate failure of conduction occurring proximal to the His bundle. In type B response, conduction delay occurred in both the A-V nodal and His-Purkinje systems. The recording of a characteristic A-V nodal potential permitted the localization of A-V nodal delay to the N-H interval.

Additional Indexing Words:

A-V nodal potential — Right bundle-branch potential — Decremental conduction
His-Purkinje system — Nodal His interval

PREMATURE atrial stimulation and its resultant effects on atrioventricular conduction have provided a useful and commonly employed method of studying the functional properties of the A-V conduction system in the experimental animal.[1-7] Until recently, studies of the effects of premature atrial stimulation on A-V conduction in man have been limited to an analysis of the P-R interval and QRS patterns of the standard electrocardiogram.[8, 9] It was the purpose of this study to define more precisely the effects of premature atrial stimulation on A-V conduction in man using a recently described catheter technic for recording the electrical activity of the specialized conduction system.[10, 11]

Methods

Right heart catheterization was performed on 20 subjects in the post-absorptive, nonsedated state. All subjects were in normal sinus rhythm at the time of study. Using local anesthesia, a tripolar electrode catheter* was percutaneously introduced into the right femoral vein and fluoroscopically positioned across the tricuspid valve. This catheter was used to record A-V nodal, His bundle, and right bundle-branch potentials as described in previous communications.[10, 11] The proximal terminals of the electrode catheter were plugged into a multichannel distribution switch box† which permitted the selection of any two electrodes for bipolar recordings, Each channel of the distribution switch box was led into the AC input of an ECG preamplifier. The filter frequencies of the ECG preamplifier were set at 40

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†Electronics for Medicine, White Plains, New York.

and 500 cps. In most studies three simultaneous bipolar electrograms were recorded using electrodes 1 and 2, 2 and 3, and 1 and 3. A bipolar electrode catheter* was also introduced percutaneously into the right antecubital vein and fluoroscopically positioned against the lateral wall of the right atrium at its junction with the superior vena cava. The right atrium was prematurely stimulated at controlled intervals using an R-wave coupled pulse generator (model 5837)† which delivered impulses of 2-msec duration. Impulses were delivered to the right atrium at a milliamperage sufficient to assure reliable atrial capture (usually 1½ to 2 × threshold). A standard lead electrocardiogram was simultaneously recorded along with the intra-atrial electrograms. All records were taken on a multichannel oscilloscopic photographic recorder‡ at a paper speed of 100 to 200 mm/sec. Measurements (in msec) were made of the intervals between the sinus P wave and the premature stimulus (P-P′ interval), from the P wave to the His bundle deflection (P-H interval), and from the His bundle deflection to the onset of the Q wave of the QRS complex (H-Q interval). A P-P′ coupling interval of less than 300 msec was not attempted in this study because of the risk of inducing atrial fibrillation.[12] Attention was paid to the grounding of all equipment in order to prevent stray currents from entering the system.

Results

Type A Response: Delay in A-V Nodal Region

The least common type of response observed was that in which the atrioventricular conduction delay was limited to the A-V nodal region only over the full range of coupling intervals. It occurred in only three of the 20 subjects studied. The response to progressive shortening of the coupling interval was characterized by a progressive prolongation in the P-H interval (as compared to the sinus beat) with a constant H-Q interval. Ventricular depolarization remained normal. In several studies the conduction delay for the A-V nodal region could be further localized to the N-H interval.

Figure 1 illustrates this type of response. In panel A, at a coupling interval of 518 msec, the P-H interval increased from 102 msec to 153 msec. The H-Q interval remained constant at 43 msec. Shortening the coupling interval to 372 msec (panel B) resulted in prolongation of the P-H interval to 255 msec. Further shortening of the coupling interval to 313 msec (panel C) resulted in prolongation of the P-H interval to 401 msec. Finally, at a coupling interval of 311 msec the premature atrial impulse is blocked proximal to the His bundle (panel D).

On the bottom electrogram tracing of panels B and C, an A-V nodal potential (N) was recorded along with the His bundle potential. The N potential of the sinus beats cannot be identified with certainty since it is contiguous with the atrial electrogram and the His potential. However, for the premature beat the N potential is more readily identified. It is a diphasic wave with slurring of the upstroke and downstroke. Both the duration of the N potential and the interval between the N and H potentials increase during premature atrial stimulation, thereby localizing the A-V conduction delay to the N-H interval.

Type B Response: Delay in Both the A-V Nodal and the His-Purkinje Region

Atrioventricular conduction delay occurring in both the A-V nodal and His-Purkinje systems was the more common type of response observed. It occurred in 17 of the 20 subjects studied. This type of response to progressive shortening of the coupling interval was characterized by a progressive prolongation of the P-H interval until at a critical coupling interval a prolongation of the H-Q interval occurred. The prolongation of the H-Q interval was always associated with aberrant ventricular conduction. The most common aberrant ventricular conduction pattern noted was a right bundle-branch block. By shortening the coupling interval even further, a left bundle-branch block pattern could be elicited in seven of the 17 subjects. Shortening the coupling interval beyond the point which produced aberrant ventricular depolarization resulted in complete block of the premature atrial stimulus. Complete block occurred in

*U. S. Catheter Corporation, Glens Falls, New York.
†Medtronics, Inc., Minneapolis, Minnesota.
‡Electronics for Medicine, White Plains, New York.

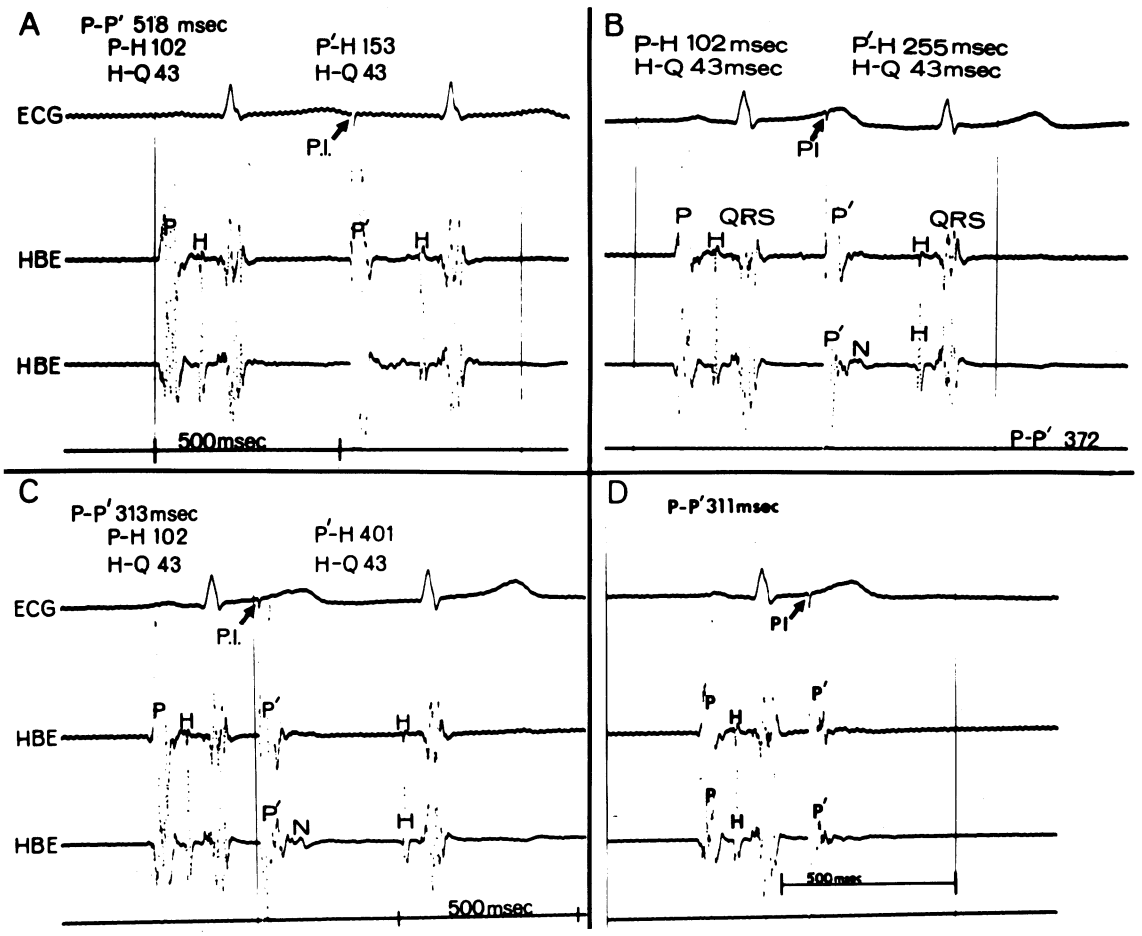


Figure 1

Panels A through D demonstrate the effects of shortening the coupling interval of the premature atrial stimulus on the P-H and H-Q intervals. ECG = standard electrocardiographic lead; HBE = His bundle electrograms; H = His bundle potential; N = A-V nodal potential; PI = the pacer impulse. The atrial deflections are designated P and P'. In panels B and C the A-V delay can be localized to the N-H interval. In panel D complete block proximal to the His bundle occurred.

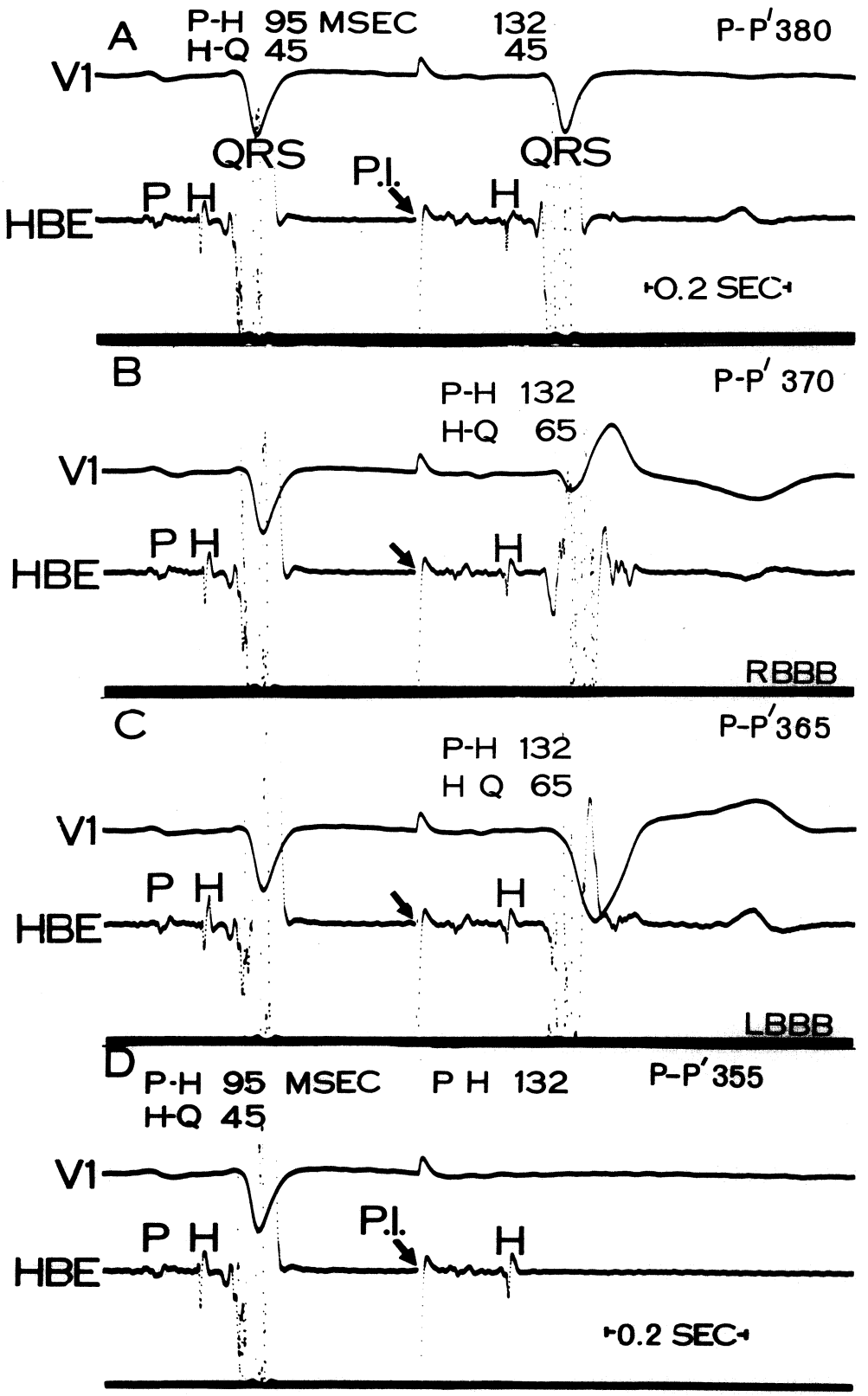
either the N-H region (in other words, proximal to the His bundle) or in the His-Purkinje system (distal to the His bundle).

This type of response to premature atrial stimulation is illustrated in figures 2 to 5.

In figure 2, panels A through D demonstrate the sequential changes which occur as the P-P' interval is progressively shortened. In panel A, at a P-P' interval of 380 msec the premature atrial stimulus results in prolongation of the P-H interval, a constant H-Q interval, and normal ventricular depolarization.

Shortening the coupling interval to 370 msec (panel B) and 365 msec (panel C) produces RBBB and LBBB, respectively. Both aberrant beats are associated with an increase in the H-Q intervals from 45 msec to 65 msec. Panel D demonstrates that with the shortest coupling interval (355 msec) block occurs distal to the His bundle.

An example of delay in the N-H interval and complete block below the His bundle is shown in figure 3. In the bottom electrographic tracing, both the N and H potentials



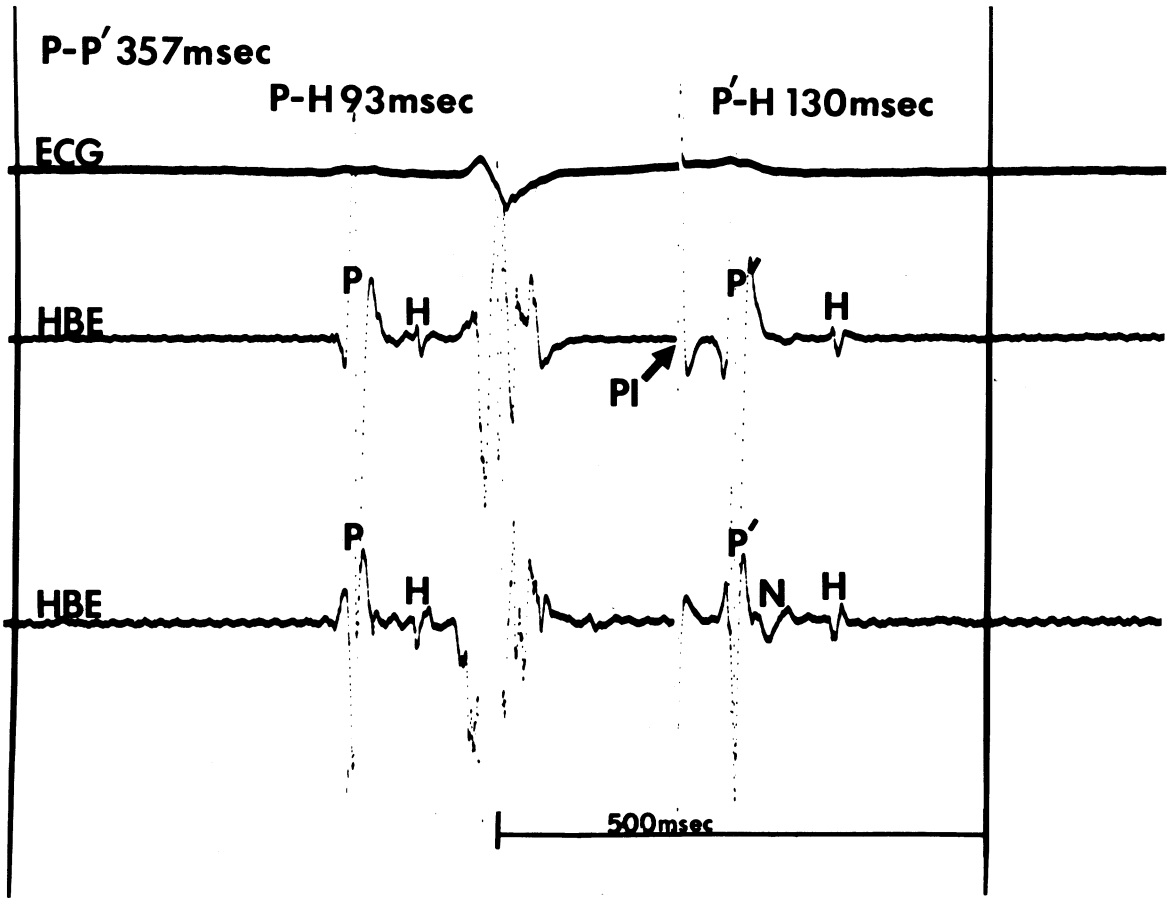


Figure 3

Delay in the N-H interval and complete block below the His bundle. It is also noted that the nodal potential (N) is characterized by slurring on the downstroke and upstroke.

were simultaneously recorded. The N potential is characterized by slurring of both limbs of the biphasic wave.

The effect of concealed conduction below the His bundle on conduction of a subsequent impulse was also observed. In figure 4 the first beat is a sinus beat with a P-H interval of 100 msec. The second beat results from a premature atrial stimulus which is coupled at an interval of 395 msec. This premature beat has a P-H interval of 120 msec and is blocked below the His bundle. The third beat is a sinus beat, and the P-H interval is prolonged to 140 msec. This prolongation is the effect of the

Figure 2

In panels A through D, the top tracing is a lead V_1 and the bottom tracing a His bundle electrogram (HBE). When the atrium was prematurely stimulated at a coupling interval of 380 msec (panel A), a delay in A-V conduction occurred as manifested by an increase in the P-H interval to 132 msec. The H-Q interval remained constant, and ventricular depolarization was unaltered. Shortening the coupling interval to 370 msec (panel B) and 365 msec (panel C) resulted in aberrant ventricular depolarization (RBBB and LBBB) with an associated prolongation of the H-Q interval. At a coupling interval of 355 msec (panel D) complete block below the His bundle resulted.

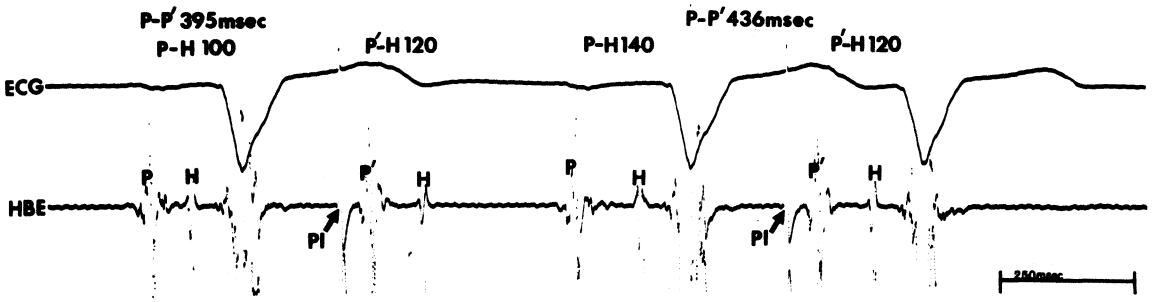


Figure 4

The effect of concealed conduction on conduction of subsequent impulses. See text for discussion.

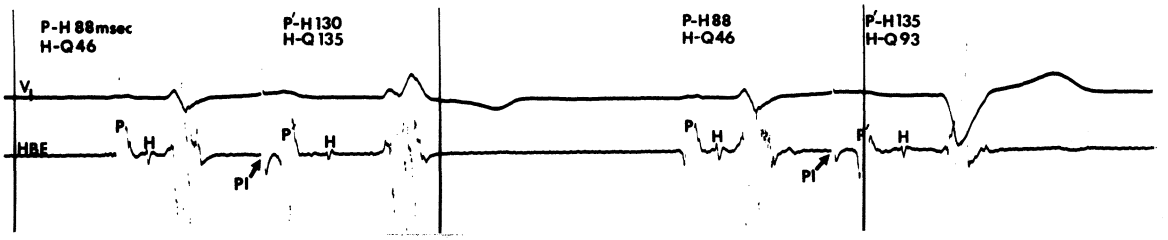


Figure 5

Alternating RBBB and LBBB when premature stimuli were coupled to successive sinus beats at an interval of 348 msec.

previously concealed conduction. The fourth beat is a premature beat coupled at an interval of 436 msec and is conducted to the ventricles.

In three patients alternating RBBB and LBBB occurred during premature atrial stimulation at the same coupling interval. In figure 5, at a coupling interval of 348 msec, alternating RBBB and LBBB occurred.

Figure 6 illustrates a recording of the right bundle-branch potential in a human subject. The first and third beats are sinus beats and the right bundle-branch potential is labeled RB. The second beat results from a premature atrial stimulus which is coupled to the preceding sinus beat at an interval of 310 msec. The premature beat results in a RBBB pattern as reflected in V_1. The RB potential is not recorded for the premature beat, indicating that the site of propagation failure occurred in the proximal portion of the right bundle.

Discussion

In 1956, Moe and associates[1] made a detailed study of the spread of premature atrial impulses from the atrium to the ventricles in dog hearts. On the basis of their findings, it was postulated that a dual A-V transmission system existed. Subsequently, Hoffman and associates[2], performed similar studies in which direct recordings were made from selected areas of the specialized conducting system. From their findings, these later investigators concluded that it was unnecessary to postulate a dual A-V transmission system in the mammalian heart to account for the variations in propagation of premature atrial impulses. They stated that these variations in propagation could be explained in terms of the level of membrane potential of the individual fibers constituting the A-V conduction system and their response to fiber stimulation.

Until recently, recordings from the specialized fibers of the atrioventricular conduction system in man have been unavailable. Much of our information concerning the functional properties of the A-V conduction system in man has been extrapolated from animal experiments and obtained from observations

made on the various components of clinical electrocardiograms. It is now possible to extend our observations in man by recording extracellular action potentials from several specialized areas along the A-V conduction system.[11]

The results of the present study relate closely to the reports of Moe and co-workers,[3] Hoffman and associates,[2] and Moore,[4] despite the fact that obvious differences in methodology exist between this clinical report and their animal experiments. Among these differences is the fact that in the aforementioned studies the basic driving rate of the atrium was controlled while in this study premature atrial stimulation was performed during sinus rhythm. Nonetheless, it would appear that these differences do not significantly alter the relevance of our findings and that pertinent correlations related to the electrophysiology of the human heart can be made.

The findings in the present study are in general agreement with those of Hoffman and associates[2] who emphasized that delay in propagation of premature atrial activity to the ventricles may occur at more than one of several regions arranged in series. Using microelectrode technics, Moore[4] also demonstrated that block of premature atrial responses could result from decremental conduction within the A-V nodal region as well as in the ventricular specialized conduction system. Type A response was obtained whenever the conduction delay occurring in the A-V nodal region increased in direct proportion to the prematurity of the propagated atrial impulse. This was manifested by a prolongation of the P-H interval (fig. 1). Conduction along the His-Purkinje system remained normal as reflected in this study by a constant H-Q interval and an unaltered QRS configuration. Presumably, the progressive A-V nodal delay (increasing P-H interval) allows sufficient time for repolarization of the His-Purkinje fibers so that the premature impulse is normally conducted throughout this latter system. Further shortening of the P-P′ interval resulted in complete block of the premature atrial impulse within the A-V node or nodal His interval (figs. 1 and 3). Progressive A-V nodal delay with normal His-Purkinje conduction has also been noted when the heart rate of human subjects was increased by single atrial pacing.[13] Similarly under these conditions, the localization of A-V conduction delay to the N-H interval has been reported.[11]

Type B was by far the more common response observed in this study (17 of 20 patients). Premature atrial stimulation first produced a prolongation of the P-H interval until at a critical coupling interval prolongation of conduction along the His-Purkinje system resulted. This was manifested by a prolonged H-Q interval which was associated with altered ventricular depolarization. The most common aberrant ventricular depolarization pattern observed was an RBBB. The vulnerability of the right bundle branch to block during premature atrial stimulation as compared to the left bundle has been noted by others.[3, 8, 14-16] Moe and associates[3] observed RBBB in 17 of 24 experiments. Cohen and associates[8] found a 100% incidence of RBBB aberration in patients demonstrating aberrant conduction during induced premature atrial stimulation. The electrode catheter technic has also been used to record right bundle-branch potentials in the human heart.[11]

Our results (fig. 6) are in agreement with the experimental findings of others. Moore and associates demonstrated in the dog heart that transection of the distal right bundle branch results in incomplete right bundle-branch block while transection of the proximal right bundle branch results in complete right bundle-branch block.[16] Moe and associates[3] concluded from their studies in which RBBB occurred during premature atrial stimulation that "the site of propagation failure was within or at the termination of the main trunk of the right bundle. . . ." The exact reasons for the greater frequency of block in the right bundle as compared to the left bundle are not known, although differences in the effective refractory periods of the two bundle branches have been suggested. In seven subjects premature atrial stimulation produced an LBBB at the same

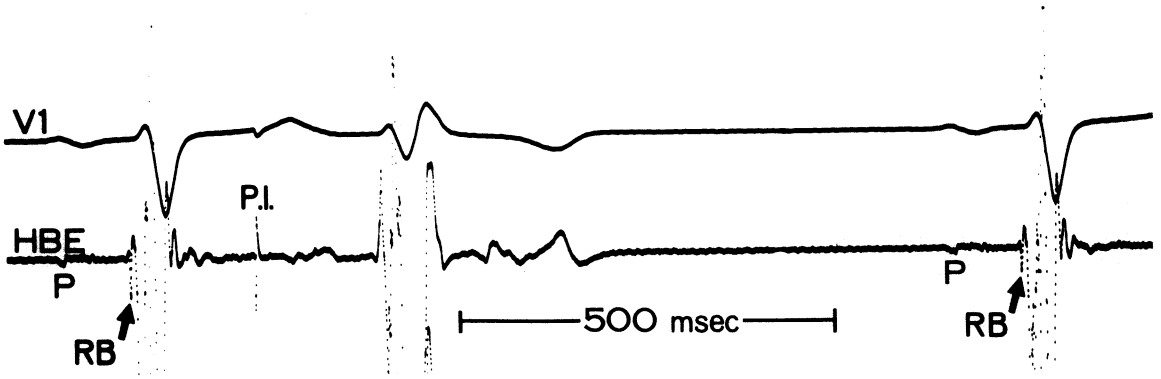


Figure 6

Right bundle-branch block induced by premature atrial stimulation. The first and third beats are sinus beats and the right bundle potential is labeled RB. The second beat is induced by a premature atrial stimulus. The premature beat results in an RBBB pattern and the RB potential is not recorded, indicating that failure of impulse propagation occurred in the proximal portion of the right bundle.

(two patients) or shorter (five patients) coupling intervals than those which produced RBBB. Moe and associates[3] observed only one incidence of LBBB although in most experiments some depression of conduction on the left side was noted when propagation failed on the right. The exact reasons for the occurrence of alternating RBBB and LBBB at the same coupling interval are not known.

Further shortening of the coupling interval beyond the point of bundle-branch block produced complete decrement of the premature stimulus in either the His-Purkinje system or in the nodal His interval. The response demonstrated in figures 2 and 3, in which the block occurred distal to the His bundle, can be interpreted as a functional, bilateral bundle-branch block, although the exact location of the block could not be determined. Complete failure of impulse transmission within the His-Purkinje system has been demonstrated by Hoffman and associates,[2] Moe and associates,[3] and Moore.[4]

The recording of A-V nodal potentials using an electrode catheter has been previously reported.[11] In that report, the identification of A-V nodal potentials was based largely on the slurring or notching which occurred on the ascending limb of the biphasic wave. Further experience as herein reported, has demonstrated that during A-V conduction delay, the A-V nodal potentials demonstrate slurring or notching on both the upstroke and downstroke. This finding in man is consistent with the microelectrode studies which demonstrate notching on both the depolarization and repolarization limbs of the transmembrane action potentials of the single A-V nodal fibers.[7, 17] During A-V conduction delay, the duration of the N potential and the interval between the N and H potentials increased, thus localizing the A-V nodal delay to the N-H interval. Our conclusions are not in keeping with the generally held concept that the greatest delay occurs at the atrio-nodal junction.[17] Instead our findings are consistent with those of Alanis and associates[18] who proposed that the area of greater vulnerability to A-V conduction is in the N-H interval.

In the present study, no evidence of re-excitation of the atrium (echo phenomenon) was observed despite the wide range of coupling intervals employed. Moe and associates[1, 6] have postulated that re-excitation occurs as a result of two functionally different pathways (α and β) in the upper part of the A-V node. Atrial echo beats have been recorded by others.[4, 9] Schuilenburg and Durrer[9] reported a clinical case in which atrial echo beats were induced by controlled premature atrial stimulation. In all these reports concerning atrial echoes, the basic driving rate

was controlled. In the present study the basic driving rate was not controlled, which may in part account for the fact that this phenomenon was not observed in any of the 20 patients studied.

The recording of extracellular action potentials from selected sites along the A-V conduction system in man using an electrode catheter technic, has provided a unique opportunity to confirm much of the previously reported experimental data concerning the functional properties of A-V conduction. The results of the present study confirm that the most common site of delay in A-V conduction is in the nodal-His interval although delay and block of impulse transmission can occur in the His-Purkinje system.

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