Studies on Ventriculo-Atrial Conduction and the Reentry Phenomenon

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SUMMARY

Ventriculo-atrial conduction and the reentry phenomenon was studied during right ventricular and bundle of His pacing in the intact dog heart. Bipolar electrogram recordings were obtained from the sinus node, Bachman's bundle, the right and left atria, the bundle of His, and the ventricle. Retrograde conduction resulted in a specific change in the sequence of atrial activation. The recording of both antegrade and retrograde His deflections provided a more accurate means of comparing A-V and V-A conduction times. In all experiments retrograde conduction time exceeded antegrade conduction time. The A-V nodal region was found to be the zone of retrograde conduction delay and block. Increasing the driving rate of the heart, vagal stimulation, and isoproterenol, affected V-A conduction similar to the way in which these maneuvers affect A-V conduction. Reentry (ventricular echoes) commonly occurred when right ventricular or bundle of His pacing resulted in the reverse Wenckebach phenomenon.

Additional Indexing Words:
Atrial bipolar electrogram Conduction, retrograde, concealed, A-V nodal
His electrograms Vagal stimulation A-V dissociation Isoproterenol
Reverse Wenckebach Reciprocal beating Ventricular echoes
Retrograde block

It has been known for a long time that the conduction system of the heart is capable of transmitting impulses in both an antegrade and retrograde direction. Failure to transmit impulses in one direction does not necessarily mean that the conduction system is unable to transmit them in the opposite direction. A number of clinical reports have demonstrated intact retrograde conduction in the presence of complete or near complete failure of antegrade conduction. The functional properties of atrioventricular conduction have been extensively studied in the experimental animal and man using recordings from specialized conducting fibers. These studies have shown that the major site of delay and block of antegrade impulses is in the A-V node. However, conduction delay and block have also been demonstrated in the His-Purkinje system. Most studies concerned with ventriculo-atrial conduction in the intact heart have utilized single atrial and ventricular electrogram recordings, and the site of retrograde conduction delay has been less precisely defined. Microelectrode experiments on isolated cardiac tissue of the rabbit have demonstrated that in addition to the A-V node, a major location of retrograde conduction delay and block is within the ventricular specialized conduction system.

It has been repeatedly demonstrated in clinical electrocardiograms that the occurrence of retrograde conduction delay and block are important considerations in the understanding of complex cardiac arrhythmias. It will be...
the purpose of this report to describe some of the characteristics of retrograde conduction under various physiologic interventions in the intact dog heart.

Methods

Thirty adult mongrel dogs were anesthetized intravenously with pentobarbital Na (30 mg/kg). Respiration was artificially controlled. A thoracotomy was performed at the level of the fourth right intercostal space and the lateral surface of the right atrium and basal portion of the right ventricle were exposed. Guided by anatomic landmarks as previously described, two fine Teflon-coated wires (0.005 inch in diameter) were threaded into a single 23-gauge needle which was inserted into the region of the bundle of His. The tips of the wires were bent to form small hooks at the distal end of the needle so that they would not be dislodged once inserted. The wires were connected to a distribution switch box from which bipolar leads could be selected. The same method was used to impale the regions of the sinus node, Bachman’s bundle, the right and left atrial appendages, the posterior portion of the left atrium, and the coronary sinus. Each bipolar lead was led into an ECG preamplifier, and frequencies below 40 and above 500 Hz were filtered out. A lead II electrocardiogram was simultaneously recorded with all the bipolar atrial electrograms. Recordings were made on a multi-channel oscilloscopic photographic recorder at paper speeds between 25 and 200 mm/sec. The right cervical vagus nerve was isolated and similarly impaled with bipolar wires. Vagal stimulation was performed using a Grass stimulator (Model S 8) which delivered impulses of 4-msec duration at a frequency of 20 to 40/sec. Bipolar wires were also inserted into the right atrial appendage and the lateral basal portion of the right ventricle so that the heart could be electrically stimulated at various frequencies.

Results

In this study, retrograde depolarization of the atria was determined to be present when both of the following criteria were met: (1) The sequence of atrial depolarization, as determined by the multiple bipolar electrogram recordings, was altered from that of sinus or antegrade activation. This altered sequence involved a specific pattern in which the lower portion of the atrial septum was depolarized first, followed next by Bachman’s bundle, the coronary sinus, and then the sinus node. The time interval between right and left atrial depolarization always changed from that recorded during antegrade depolarization. During retrograde conduction the left atrium was depolarized before or simultaneously with the right atrium. Less frequently, during retrograde atrial depolarization a minor variation in this pattern occurred with the coronary sinus region being activated after the low atrial septum. Bachman’s bundle again preceded activation of the sinus nodal region. (2) The sequence of retrograde atrial depolarization just described followed the ventricular or junctional impulse.

As will be discussed below, it was possible in this study to record a retrograde His deflection in all of the right ventricular pacing studies. This permitted a more accurate measurement of retrograde conduction time (H-A interval). At times the retrograde His deflection was of opposite polarity to that recorded during sinus rhythm (fig. 1). At other times, the retrograde His deflection exhibited changes in the initial and terminal portions. The morphologic changes of the retrograde His deflection depend upon the relationship of the bipolar wires to the common bundle itself.

Panel A of figure 1 presents a representative tracing obtained during sinus rhythm. The sequence of atrial depolarization proceeds from the region of the sinus node (SN), then to the region of Bachman’s bundle (BB), followed by activation of the right atrial appendage (RA), the left atrial appendage (LAA), the posterior part of the left atrium (LAP), and finally the coronary sinus (CS). The A-H interval (atrial-His) measured 56 msec. During sinus rhythm the A-H and H-V intervals remained constant (±5 msec).

In 26 of 30 experiments, consistent retrograde depolarization of the atria resulted from continuous right ventricular or His bundle pacing. In four experiments retrograde conduction could be induced only intermittently and for a few beats. Most often in these
animals, complete A-V dissociation or A-V dissociation with atrial captures occurred.

Panel B of figure 1 (same experiment) is a representative example of retrograde conduction during right ventricular pacing. Atrial depolarization follows ventricular depolarization at a fixed interval (H-A = 142 msec). Furthermore, the sequence of atrial depolarization is altered from that of sinus rhythm and occurs as described above. The findings illustrated in figure 1, panel B were observed in all 26 experiments involving right ventricular pacing. Similar findings were also obtained during His bundle pacing as shown in figure 2. In panel A of figure 2 the bundle of His was paced at a rate of 170/min. The QRS complex is of the same configuration and duration as during sinus rhythm. The interval between the stimulus artifact and retrograde atrial depolarization (S-A interval) measures 105 msec and is the same as the H-A interval during right ventricular pacing at the same rate (panel B). The equal S-A and H-A intervals of figure 2 prove that the deflection recorded during ventricular pacing is from the bundle of His. Figure 3 depicts a recording of a retrograde His potential during spontaneous premature ventricular contraction. The premature ventricular beat is associated with retrograde atrial depolarization.

In all experiments retrograde conduction time (H-A interval) during right ventricular or His bundle pacing exceeded antegrade conduction time (A-H interval) at comparable right atrial paced rates. Other examples of prolonged retrograde conduction time which were commonly observed included the so-called reverse Wenckeback phenomenon and 2:1 retrograde block. Both of these phenomena could be demonstrated by either increasing the frequency of His bundle or right ventricular stimulation or by applying vagal stimulation during 1:1 retrograde conduction (see below).

An example of 2:1 retrograde block which occurred during right ventricular pacing at a rate of 190/min is shown in figure 4. Every other ventricular impulse is retrogradely con-
Figure 2
Comparison of His bundle pacing (panel A) with right ventricular pacing (panel B) at a rate of 170/min. There is retrograde depolarization of the atria in both panels. In panel A the stimulus to atrial depolarization (S-A interval) is the same as the H-A interval during RV pacing (panel B).
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Figure 3

Recording of retrograde bundle of His (H) activity during a spontaneous premature ventricular contraction (PVC). The third QRS complex is a spontaneous PVC which is associated with retrograde atrial depolarization. The vertical time lines represent 1,000 msec.

Figure 4

Panel A shows a reference sinus beat. Panel B illustrates 2:1 retrograde conduction during RV pacing.

ducted to the atrium. The recording of a retrograde His deflection and no atrial activity suggests that for alternate beats the level of block was at the A-V node. In this experiment right atrial pacing at a comparable heart rate (not shown) resulted in 1:1 A-V conduction.

Vagal stimulation always caused a delay or block in retrograde conduction. The effects of vagal stimulation during bundle of His pacing are illustrated in figure 5. Panel A is a
The effects of vagal stimulation on retrograde conduction during bundle of His pacing at a rate of 220/min. Panel A is a reference sinus beat. In panel B there is 1:1 retrograde conduction during bundle of His pacing. In panel C at the same paced rate vagal stimulation caused 2:1 retrograde conduction.

Isoproterenol always shortened retrograde conduction time. Figure 6 illustrates that at the same paced ventricular rate isoproterenol shortened the H-A interval from 112 msec (panel A) to 81 msec (panel B). In addition, during isoproterenol infusion the stimulus artifact to H interval (S-H) decreased from 35 msec to 30 msec.

The reentry phenomenon (ventricular echoes) was consistently and repeatedly demonstrated in 20 different experiments in which a significant degree of retrograde conduction delay could be achieved during His bundle...
(Panel A) During right ventricular pacing, retrograde conduction time (H-A interval) is 112 msec. The interval between the stimulus artifact and the retrograde His deflection (S-H interval) is 35 msec. (Panel B) At the same paced ventricular rate isoproterenol shortened the H-A interval to 81 msec and the S-H interval to 30 msec.
Figure 7

(Panels A) Two reference sinus beats. The sequence of atrial activation should be noted. An A-V nodal (N) and right bundle-branch potential (RB) are also recorded during antegrade conduction. The vertical time lines (panel C) represent 1,000 msec.

(Panels B) Bundle of His pacing (212/min). The R-R interval is 283 msec. The S-V interval is the same as the H-V interval of panel A. An antegrade RB potential is again recorded. Note the retrograde sequence of atrial activation and a retrograde nodal (N) potential. The S-A interval is 166 msec.

(Panels C) Bundle of His pacing at the same rate as panel B. Vagal stimulation now causes reverse Wenckebach block with reentry. The fourth QRS complex is a reentrant beat. It is preceded by an antegrade H and RB potential and occurs 31 msec before the next driving stimulus which is ineffective and falls within the ventricular electrogram. The cycle is repeated starting with the fifth QRS complex.

and right ventricular pacing. An example of reentry which occurred during His pacing is illustrated in figure 7. Panel A depicts two sinus beats. The His bundle electrogram...
Reentry during bundle of His pacing. The sinus node had been crushed. There is a reverse Wenckebach. The fifth QRS complex is a reentrant beat which occurs only 5 msec before the next drive stimulus.

Figure 8

(RBE) records an A-V nodal (N), His bundle (H), and right bundle branch (RB) potential. In panel B the bundle of His was paced at a rate of 212/min. The stimulus artifact is followed by a RB potential. There is retrograde depolarization of the atria (compare sequence of atrial depolarization with panel A) at a fixed H-A interval of 166 msec. In panel C vagal stimulation was applied while the bundle of His was paced at the same rate as shown in panel B. With vagal stimulation retrograde conduction progressively lengthened (reverse Wenckebach). The longest retrograde conduction time followed the third QRS complex. This retrograde impulse reentered the A-V conduction system antegrade, depolarized the bundle of His, and was conducted to the ventricles producing the fourth QRS complex. The fourth QRS complex is of normal configuration and occurs 31 msec before the next ineffective stimulus artifact. The cycle is repeated starting with the fifth QRS complex.

Of interest is a comparison of the findings illustrated in figures 7 and 8. Figure 8 demonstrates the reverse Wenckebach and reentry which are consequent to the rate of His bundle pacing rather than to vagal stimulation. The R-R intervals of the first four QRS complexes are 311 msec. The fifth QRS complex occurs prematurely by 5 msec. It is a reentrant beat which is preceded by an antegrade His deflection (open arrow). There is no retrograde depolarization following this QRS complex. The fifth stimulus artifact (S) is ineffective. The degree of prematurity of the reentrant beat in figure 8 as compared to figure 7 has direct bearing on the proper recognition of this phenomenon in clinical electrocardiograms.

Examples of reentry during right ventricular pacing with the reverse Wenckebach phenomenon were also observed. Comparison of figure 9 with figures 7 and 8 demonstrates the importance of precisely defining retrograde conduction as it relates to the reentry phenomenon. Panel A of figure 9 shows two reference sinus beats with A-H intervals of 46 msec. In panel B there is A-V dissociation during right ventricular pacing. The antegrade sequence of atrial depolarization should be noted. The sinus P waves are occurring progressively later after each QRS complex. This R-P relationship simulates a reverse Wenckebach phenomenon. The third sinus P wave captures the ventricles. It is antegradeley conducted with an A-H interval of 66 msec. The prolonged A-H interval reflects the
retrograde concealed conduction of the previous ventricular beat. The next sinus P wave (4th) is also antegradeely conducted to the bundle of His with a normal A-H interval of 46 msec. The resultant QRS complex is a fusion beat. It should also be pointed out that retrograde concealed conduction is the reason why the first two atrial beats of sinus origin in panel B are not followed by an H deflection. Figures 7 and 8 present a true reverse Wenckebach phenomenon with reentry while figure 9 is an example of A-V dissociation with ventricular capture beats.

Experiments were also performed to determine whether during premature ventricular stimulation retrograde delay and block could be elicited within the Purkinje-bundle branch system as well as the A-V node. Two types of studies were performed. In one, the ventricles were driven at a regular rate and after each sixth ventricular beat a premature stimulus was introduced at varying coupling intervals. In the other studies, a single premature ventricular beat was intermittently coupled to the sinus beat. During ventricular premature stimulation retrograde conduction delay was manifested by a prolonged S-H interval (not shown). Figure 10 illustrates retrograde conduction delay in the myocardial-bundle branch system. Panel A depicts a reference sinus beat in which simultaneous recordings of His bundle and right bundle-branch activity were recorded. In panel B the right ventricle was stimulated at a regular rate and the S-RB interval measures 55 msec. In panels C, D, and E the right ventricle is prematurely stimulated after a sinus beat at progressively shorter coupling intervals. In panel C, at an R-S coupling interval of 316 msec, the S-RB interval remains at 55 msec. As the R-S coupling interval is shortened (panels D and E) the S-RB interval progressively increases to 75 and 110 msec, respectively.

Discussion

The results of the present study are in agreement with previously reported observations concerning the transmission of impulses within the intact heart. These observations are as follows: (1) The atrioventricular conduction system is capable of transmitting impulses in both an antegrade and retrograde direction, 

Figure 9

(Panels A) Two reference sinus beats. The A-H interval is 46 msec. (Panel B) Right ventricular pacing. There is A-V dissociation. Note the sinus activation of the atria. A retrograde H deflection is recorded which is of opposite polarity to that in panel A. The sinus P waves occur progressively later after each QRS complex simulating a reverse Wenckebach phenomenon. The fourth QRS complex is an atrial capture beat. There is an antegrade H deflection. The A-H interval of 66 msec reflects concealed conduction. The fifth QRS complex is a fusion beat. The sinus impulse, however, did travel unimpeded to the bundle of His. The A-H interval is normal at 46 msec.
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(Panel A) A reference sinus beat is shown. The middle HBE tracing records a right bundle-
branch potential (RB). In the bottom HBE an H and RB potential are recorded. (Panel B) The
ventricles are being paced at a regular rate. The S-RB interval is constant at 55 msec.
(Panels C) The first beat is a sinus beat. The second beat results from a premature ventricular
stimulus (S) applied to the right ventricle at an R-S coupling interval of 316 msec. The S-RB
interval remains the same at 55 msec. As the coupling interval is decreased to 214 msec (panel D)
and 178 msec (panel E) the S-RB intervals increase to 75 msec and 110 msec, respectively.

(2) at comparable heart rates retrograde conduction time is longer than antegrade conduction time, and (3) retrograde transmission occurs via the A-V node.

The differences between antegrade and retrograde conduction times were present in all experiments in which comparisons were made during right atrial, His bundle, and right ventricular pacing. Similar prolongations of V-A conduction in the dog heart have been noted by Rosenblueth and Rubio and Moe and co-workers. It appears that, as in antegrade conduction, the major site of retrograde delay occurs within the A-V node. Furthermore, the functional characteristics of retrograde conduction through the A-V node were qualitatively similar to those recorded for antegrade conduction. The effects of vagal stimulation and isoproterenol on retrograde conduction were similar to their known effects on antegrade conduction.

It has been demonstrated in both the experimental animal and man that progressive increases in the rate of atrial stimulation result in progressive prolongation in the A-H interval (first degree heart block), the Wenckebach phenomenon, and finally higher degrees of A-V block (2:1 and 3:1). Likewise, during 1:1 retrograde conduction increasing the rate of ventricular or His bundle stimulation produced (in order) the reverse Wenckebach phenomenon, 2:1 retrograde block, and finally A-V dissociation. Not infrequently, however, the transition from 1:1 retrograde conduction to A-V dissociation could be elicited over a relatively narrow range of paced heart rates (5 to 20 beats). This finding is in contrast with antegrade conduction in which the transition from 1:1 A-V conduction to high degree A-V block generally occurs at appreciably greater increases in heart rate.

In none of the studies in which measure-
ments were made at comparable heart rates, was retrograde conduction time (H-A interval) found to be less than antegrade conduction time (A-H interval). These findings in the experimental animal differ from those obtained in clinical studies in which 1:1 V-A conduction was occasionally associated with retrograde conduction times which were equal to or less than antegrade conduction times.28 This discrepancy may be related to the fact that in these clinical studies the onset of the ventricular arrhythmias may have been associated with changes in autonomic tone which are known to have a significant effect on conduction within the A-V node.

Our findings indicate that when the dominant pacemaker of the heart is located in the ventricle or His bundle, the zone of retrograde concealment is within the A-V node. This conclusion is based on the fact that a retrograde His bundle deflection was recorded within the ventricular electrogram during high degree V-A block (figs. 4 and 5), complete retrograde block, and premature ventricular stimulation. The prolongation of the S-H and S-RB intervals (fig. 10) indicates that retrograde conduction delay does occur within the ventricular muscle-Purkinje-bundle branch systems. In this regard our findings are in agreement with those of Moore18 who studied retrograde concealment of multiple premature ventricular responses in isolated cardiac tissue. The retrograde propagation of the ventricular impulses to the His bundle and A-V node provide a satisfactory explanation and support for the interpretation of many clinical arrhythmias based on concealed conduction.19, 20, 24

The reentry phenomenon (also called reciprocal rhythm, reciprocal beating, echoes, and return extrasystoles) has until recently been considered a rare phenomenon.24–30 It has been demonstrated experimentally and clinically to occur whenever there is prolonged retrograde conduction following impulses arising in either the A-V junctional region (that is, His bundle) or ventricles.3–5, 25, 27, 28, 31 In this study reentrant beats could be readily induced in the normal animal which is constant with the findings of Moe and Mendez.27 The recording of His bundle activity and right bundle activity (figs. 7 and 8) confirms the fact that the reentrant impulse depolarizes the ventricle through the normal His-Purkinje network.

Comparison of figures 7, 8, and 9 illustrates the difficulty which may be encountered clinically in distinguishing a reentrant beat from atrial capture beats. This is especially true when analysis is limited to short electrocardiographic strips. That both phenomena (reciprocal beating and ventricular captures in certain forms of incomplete A-V dissociation) may occur in the same electrocardiographic record is well illustrated by Pick and Langendorf (fig. 17 of reference 24).

Another aspect of the clinical recognition of reentrant beats (ventricular echoes) is demonstrated in figures 7 and 8. One might conclude that in a junctional tachycardia, as simulated by figure 7, the phenomenon of reentry could be appreciated by the degree of prematurity of the reentrant beat (that is, 31 msec). On the other hand a prematurity of 5 msec (fig. 8) might conceivably be overlooked.

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