**Manifest and Concealed Reentry**

**A MECHANISM OF AV NODAL WENCKEBACH PHENOMENON**

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**ABSTRACT**

The mechanism of the AV nodal Wenckebach phenomenon was studied in 25 dogs by multiple atrial and His bundle electrogram (HBE) recordings. During ventricular stimulation with 1:1 retrograde conduction, the interval from the stimulus artifact to the retrograde His deflection (S-H interval) remained constant. Decreasing the cycle length of stimulation (CLS) resulted in retrograde AV nodal Wenckebach cycles. When retrograde AV nodal delay reached a critical value, the Wenckebach cycles were terminated by a reciprocal beat (manifest reentry). The His bundle and ventricles were antegradely depolarized by the reentrant impulse. A further decrease in CLS produced what electrocardiographically looked like ordinary Wenckebach cycles. However, the HBE tracing revealed that reentry was occurring but was concealed by the CLS. This was confirmed by noting that the His deflection of the last ventricular paced beat of the Wenckebach cycle was antegradely depolarized and had a shorter S-H interval than all other beats of that cycle. The reentrant and retrograde impulses collided in the bundle-branch system. Further decreases in CLS masked the reentrant phenomenon on both standard ECG and HBE tracings. The S-H interval remained constant for all ventricular paced beats of the Wenckebach cycle. Under these circumstances, reentry could still be uncovered by turning off the stimulator at an appropriate time during the Wenckebach cycle. This maneuver exposed the reciprocal beat. Thus collision of the reentrant and retrograde impulses occurred within the AV node. These findings provide a satisfactory explanation for why the last beat of the Wenckebach cycle is not conducted retrogradely to the atria.

**KEY WORDS**

- concealed conduction
- type I second-degree AV block
- His bundle electrograms
- reciprocal beats
- AV nodal delay
- retrograde conduction
- echo phenomenon

- Type I second-degree AV block (AV nodal Wenckebach phenomenon) is electrocardiographically characterized by a progressive prolongation in the P-R interval followed by a nonconducted atrial impulse (1). Following the nonconducted atrial beat, the cycle is repeated. This form of AV block can be produced in almost all subjects when the right atrium is stimulated at increasing rates above the sinus rate (2, 3). Electrophysiological studies in man have demonstrated that the conduction delay and block of the nonconducted atrial impulse occurs proximal to the bundle of His (3). The retrograde form of type I AV block can also be produced in human and animal hearts when the ventricles or bundle of His are stimulated at appropriate rates (4). Retrograde conduction delay and block also occur within the AV node. The exact mechanisms for the AV nodal delay and ultimate failure of propagation are not well understood. This report will deal with the role of AV nodal reentry as a mechanism for the antegrade and retrograde AV nodal Wenckebach forms of AV block.
ECG is lead II. Close bipolar atrial electrograms were recorded from the regions of the sinus node (SN), Bachmann's bundle (BB), right atrial appendage (RAA), left atrial appendage (LAA), posterior portion of the left atrium (LAP) and the coronary sinus (CS). The His bundle electrogram tracing (HBE) records a low atrial septum electrogram (A), His bundle deflection (H) and ventricular electrogram (V). Panel A depicts a sinus beat with an A-H interval. Note the antegrade sequence of atrial activation. Panel B represents His bundle stimulation: CL = cycle length; SA = stimulus artifact; note that the interval between the stimulus artifact and the onset of ventricular depolarization (S-V interval) is the same as the H-V interval during sinus rhythm and that the configurations of the QRS complexes are the same. Note the altered sequence of atrial activation during retrograde conduction as compared to sinus rhythm. The same abbreviations will be used for other figures unless otherwise indicated.

Methods

EXPERIMENTAL STUDIES

Thirty adult mongrel dogs (15–40 kg) were anesthetized with pentobarbital sodium, 30 mg/kg body weight, and artificially ventilated. Supplemental anesthesia was given as required. A thoracotomy was performed at the fourth right intercostal space and the pericardium incised. Close bipolar plunge wire electrodes were inserted into the regions of the sinus node, Bachmann’s bundle, the right atrial appendage, the left atrial appendage, the posterior portion of the left atrium, the coronary sinus, and the bundle of His as previously described (4, 5). These multiple atrial electrograms were recorded simultaneously with standard electrocardiographic lead II. Additional bipolar wire electrodes inserted into the bundle of His and right ventricle were used for pacing. Electrical stimulation of the heart was accomplished with a battery-powered pacemaker which delivered impulses of 2-msec duration at approximately twice threshold. The bundle of His and right ventricle were stimulated at rates sufficient to produce 1:1 retrograde conduction. Thereafter, the frequency of stimulation was increased to rates sufficient to produce retrograde AV nodal Wenckebach cycles. Multiple atrial electrograms and bundle of His activity were recorded at 40–500 Hz on a multichannel oscilloscopic photographic recorder at paper speeds of 100–200 mm/sec.

During ventricular pacing, the interval between the retrograde His deflection and the low atrial electrogram recording (H-A interval) was used as a measure of retrograde AV nodal conduction time. During bundle of His pacing, the interval between the stimulus artifact and the low atrial septal electrogram (S-A interval) was used as a measure of retrograde AV nodal conduction time. Validation of the retrograde His deflections was accomplished by pacing the ventricle and thereafter the bundle of His at comparable rates and noting that during 1:1 retrograde conduction the H-A interval equaled the S-A interval.

CLINICAL STUDIES

Electrode catheter recordings of the bundle of His were obtained in patients as previously
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described (6, 7). A quadripolar electrode catheter was percutaneously introduced into an antecubital vein and fluoroscopically positioned against the lateral wall of the right atrium near its junction with the superior vena cava. Two poles of this catheter were used to stimulate the atrium with a battery-powered pacemaker which delivered impulses of 2-msec duration at approximately twice threshold. The other two poles were used to record a high right atrial electrogram. The atrium was stimulated at increasing rates above the sinus rate until AV nodal Wenckebach cycles were produced. The interval from the low atrial electrogram recording to the His bundle deflection (A-H interval) was used as a measure of antegrade AV nodal conduction time. The interval from the His deflection to the point of earliest ventricular depolarization (H-V interval) was used as a measure of antegrade His-Purkinje conduction time. In all clinical studies, the nature of the procedure was explained to the patients and a signed consent obtained.

Results

EXPERIMENTAL STUDIES

In 25 of 30 animal experiments, stable retrograde conduction was achieved by pacing the bundle of His or ventricles at rates above the sinus rate. In the remaining 5 animals, retrograde conduction could be established for only a few beats. Most of the time, these 5 animals exhibited incomplete AV dissociation. In all studies, retrograde AV nodal conduction time during bundle of His and ventricular pacing was longer than antegrade AV nodal conduction at comparable paced atrial rates.

All of the above observations are consistent with our previous findings. (4)

In each experiment, as the cycle length of stimulation was decreased, the pattern of retrograde conduction progressed from 1:1 retrograde conduction, to retrograde Wenckebach cycles with reentry and finally retrograde Wenckebach cycles with concealed reentry. The specific type of retrograde AV nodal Wenckebach cycles obtained was determined by the retrograde AV nodal conduction time and the cycle length of stimulation.

Figure 1 illustrates 1:1 retrograde conduction during bundle of His pacing at a cycle length of 328 msec. When the cycle length was decreased between 312 and 296 msec, retrograde AV nodal Wenckebach cycles with reentry occurred as shown in Figure 2. Under these circumstances, reentry occurred only when retrograde AV nodal conduction time (S-A interval) exceeded 200 msec. In addition, an inverse relationship existed between the retrograde conduction time and the measured A-H interval of the reentrant beat. In Figure 2 the reentrant His deflection (open arrow) occurred only 7 msec before the expected (ineffective) stimulus artifact.

The results shown in Figure 2 suggested that if the cycle length for bundle of His stimulation were decreased by at least 7 msec then the effects of reentry might be masked.

FIGURE 3

Panel A depicts a single sinus beat. In panel B right ventricle stimulation resulted in 1:1 retrograde conduction.

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and the electrocardiographic pattern would resemble ordinary Wenckebach cycles. This concept was explored and demonstrated during ventricular pacing.

Figures 3-6 (same animal experiment) demonstrate the effects of decreasing the cycle length of stimulation. Figure 3 demonstrates 1:1 retrograde conduction during ventricular pacing at a cycle length of 307 msec. The interval between the stimulus artifact and the retrograde His deflection (S-H interval) was constant at 32 msec. When the cycle length for ventricular stimulation was decreased to 286 msec, as shown in Figure 4, retrograde AV nodal Wenckebach cycles with reentry occurred. In this experiment, reentry occurred whenever retrograde AV nodal conduction time exceeded 170 msec. The third retrograde impulse reentered within the AV node and antegradely depolarized the bundle of His (fourth His deflection), producing a fusion QRS complex. Retrograde conduction to the atria did not follow the fourth QRS complex. Figure 5 shows that when the cycle length of stimulation was decreased to 272 msec, the standard electrocardiogram looked like ordinary Wenckebach cycles while reentry could still be demonstrated on the His bundle electrogram recording. The second ventricular impulse reentered within the AV node and antegradely depolarized the bundle of His, as indicated by the fact that the S-H interval of

**FIGURE 4**
Retrograde Wenckebach cycles with reentry during right ventricular pacing. Same animal experiment as Figure 3. Panel A depicts a sinus beat. When cycle length of ventricular stimulation was decreased retrograde Wenckebach cycles occurred. Note the altered sequence of atrial activation as compared to sinus rhythm and the progressive increase in H-A interval. See text for details.

**FIGURE 5**
Shortening cycle length produced 3:2 Wenckebach cycles. Same experiment as shown in Figure 4. See text.
the third stimulated ventricular beat was 21 msec instead of the expected 32 msec. The bundle of His was antegradely depolarized 11 msec in advance of the expected retrograde depolarization. Thus, retrograde conduction of the third ventricular stimulated impulse was blocked within the bundle branch system.

Figure 6 illustrates how a further decrease in the cycle length of stimulation to 249 msec can conceal reentry on both the standard electrocardiogram and the His bundle electrogram recording. The H-A interval of the second ventricular beat measured 174 msec. This retrograde impulse would be expected to reenter as did all other retrograde impulses associated with retrograde conduction times exceeding 170 msec. However, the constant S-H interval of 32 msec on the His bundle electrogram tracing indicates that the His bundle was being consistently depolarized retrogradely by the ventricular impulse. Despite this fact, evidence that reentry within the AV node was occurring is presented in the second Wenckebach cycle. Following the fifth ventricular paced beat, which was also associated with a retrograde conduction time of 174...
msec, the stimulator was turned off and the retrograde impulse allowed to reenter. The A-H interval for this reentrant beat measures 90 msec. Since the interval from the retrograde A deflection to the retrograde His deflection of the third QRS complex measures 70 msec, it is apparent that for the third beat the ventricular stimulus arrived at the bundle of His 20 msec before the reentrant impulse. Thus, both the reentrant and the retrograde impulses were blocked in the AV node. In this example, reentry was concealed by the cycle length of the ventricular pacemaker. Figure 7 depicts another example in which the cycle length of a pacemaker can mask reentry during AV nodal Wenckebach cycles if one considers only the standard electrocardiogram. As in Figure 6, the stimulator was turned off following the fifth ventricular beat and reentry was clearly demonstrated.

**CLINICAL OBSERVATIONS DURING ANTEGRADE AV NODAL WENCKEBACH CYCLES**

The finding that the phenomenon of reentry consistently occurred during retrograde AV nodal Wenckebach cycles suggested that a similar mechanism may be operative during antegrade Wenckebach cycles. Six clinical cases were observed in which antegrade AV nodal Wenckebach cycles induced by atrial pacing were terminated by atrial echoes. None of these patients had a history of paroxysmal atrial tachycardia or other unusual arrhythmia. Also, in none of the patients did atrial pacing procedures produce any arrhythmia other than the type I second-degree AV block. Figure 8 is representative of our findings. In all six patients, single atrial premature depolarizations resulting in a critical AV nodal delay consistently produced single atrial beats.

**REQUIREMENT OF THE ATRIUM FOR REENTRY**

During the course of our studies it was noted in four different experiments that reentry was associated with fusion activation of the atria. An example of this is illustrated in Figure 9. Panel A illustrates reentry associated with retrograde activation of the atria, and panel B demonstrates reentry associated with fusion activation of the atria. These findings suggest that retrograde activation of the entire atrial muscle is not required for reentry.

Panel A of Figure 10 depicts a single sinus beat, occurring at a cycle length of 500 msec, in which the normal A-H interval was 65 msec. In panel B, reentry is manifest. The

**FIGURE 8**

Atrial echo terminating an antegrade Wenckebach cycle. The recordings are: Electrocardiographic leads I, II, III, high right atrial electrogram (HRA) and His bundle electrogram (HBE) recordings. Note that for the first three stimulated atrial beats the atrial depolarization on the HRA preceded the low atrial electrogram (A) as recorded on the HBE. At an A-H interval of 220 msec the impulse reentered within the AV node and retrogradely depolarized the atria (A') prior to the delivery of the next stimulus artifact. The atrial echo beat (A') produces an inverted P wave (I) on lead II. In addition, the sequence of atrial depolarization was low to high. The diagonal bars have been inserted for illustrative purposes and do not indicate at what point during the A-H interval the reentry occurred.
Panel A: The fourth QRS complex is a reentrant beat. The S-H interval for the first three beats is constant at 30 msec. The first atrial beat is a fusion beat with the low atrial septal (A) and coronary sinus (CS) electrograms preceding the SN and BB recordings. The sequence of the second and third atrial beats is that of retrograde activation of the atria. Note the early activation of the A and CS electrograms. The BB recording precedes SN and the LAA and LAP electrogram recordings precede RAA. Reentry occurred following the third atrial beat. The fourth atrial beat depicts the sinus sequence of activation. Panel B: The first atrial beat is a sinus beat and the second and third atrial beats are fusion beats. Note the altered sequence of atrial activation of the fusion beats as compared to the normal antegrade sequence (first and fourth atrial beats). The sequence of atrial activation for the fusion beats differs also from the completely retrogradely activated beats (second and third beats of panel A). The SN-SN interval remains constant while the CS-CS interval varies. Reentry follows the third atrial (fusion) beat.

Discussion

The phenomenon of AV nodal reentry is an accepted electrophysiological event which has been extensively studied in the experimental animal and man (8-20). Moe has stated that reciprocal responses can be so readily induced in normal animal hearts that their occurrence must be considered a normal physiological response (10). Our own observations are in agreement with this statement and further indicate that reentry during retrograde Wenckebach cycles is the rule rather than the exception. Our findings are also in agreement with the results of investigators who used microelectrode techniques to study AV nodal reentry (14-17).

The single most important determinant of atrial and ventricular reciprocal beats is a critical AV nodal delay (20). Thus turning off the ventricular stimulator during earlier cycles of the retrograde Wenckebach cycle did not produce reciprocal beats because the requisite AV nodal delay had not yet been achieved.

The results of this study satisfactorily explain why the last beat of a typical retrograde Wenckebach cycle is not conducted to the atria. The AV node (Fig. 6) or part of the bundle branch system (Figs. 5 and 7) is antegradely depolarized by the reentrant impulse. In one experiment, evidence was obtained for the simultaneous activation of the bundle of His by the reentrant and retrograde impulses. Thus retrograde block during Wenckebach cycles may also occur within the common bundle.

Decremental conduction, which means loss of propagation velocity, is a descriptive term which has been applied to describe the conduction characteristics of the AV nodal Wenckebach phenomenon (21, 22). This term, however, does not explain why there is progressive prolongation in AV nodal conduction followed by block. The fact that reentry is responsible for failure of the last impulse to conduct raises the interesting possibility that a
FIGURE 10
Panel A: Single sinus beat with an antegrade sequence of atrial activation and an A-H interval of 65 msec. The sinus cycle length was 500 msec. Panel B: The CS electrogram recording was removed and the stimulus artifact (S) monitored. Retrograde AV nodal Wenckebach cycles occurred (H-A = 120 to 230 msec) and manifest reentry occurred both on the ECG and HBE tracing. The interval from the low atrial electrogram, as recorded on the HBE, to the antegrade H deflection measures 45 msec.

Panel A: Single sinus beat with an antegrade sequence of atrial activation and an A-H interval of 65 msec. The sinus cycle length was 500 msec. Panel B: The CS electrogram recording was removed and the stimulus artifact (S) monitored. Retrograde AV nodal Wenckebach cycles occurred (H-A = 120 to 230 msec) and manifest reentry occurred both on the ECG and HBE tracing. The interval from the low atrial electrogram, as recorded on the HBE, to the antegrade H deflection measures 45 msec.

concealed (abortive) form of reentry may occur continuously throughout the Wenckebach cycle and that this concealed reentry may account for the progressive prolongation of the P-R interval. In this regard, the transmembrane action potential recordings of AV nodal cells are of interest.

During antegrade AV nodal Wenckebach cycles induced by vagal stimulation or acetylcholine, there occurs a characteristic “notch” during the repolarization phase of the action potential (23-27). Mendez and Moe provided convincing evidence that such notches may be due to activity of elements adjacent to the nodal recording site (26). Figures 6 and 8 of their article demonstrate that the notch was due to retrograde conduction.

Since notching is characteristically recorded throughout almost all of the Wenckebach cycle, this may represent continuous concealed reentry, which in turn may be the cause of progressive conduction delay.

Except for quantitative aspects, antegrade and retrograde AV nodal conduction are quite similar (4). It therefore appears reasonable to extrapolate our findings during retrograde Wenckebach cycles to the antegrade counterpart of this phenomenon. Our six clinical examples of atrial echoes terminating antegrade Wenckebach cycles lend support to this hypothesis. During ordinary antegrade Wenckebach cycles, the last atrial impulse may be blocked in the AV node by the reentrant impulse. Reciprocal beats are less commonly seen terminating antegrade AV nodal Wenckebach cycles than during retrograde Wenckebach cycles. One possible explanation centers about the relationship of antegrade and retrograde AV nodal conduction to cycle length of stimulation. At any given heart rate, retrograde AV nodal conduction time is generally longer than antegrade conduction time (4). Therefore the requisite conduction delay for reentry is obtainable at a longer cycle length of stimulation during retrograde Wenckebach cycles than the antegrade form. Consequently, concealed reentry would be more common in antegrade Wenckebach cycles because the atrial pacemaker could depolarize the atria before the arrival of the reentrant impulse. Also important in this consideration is the total transit time (antegrade and retrograde) of the reentrant impulse. In atrial reentry, the atrial impulse is subjected to an initial antegrade AV nodal delay and as it becomes a reentrant impulse it must travel in a retrograde direction, which is slower than antegrade conduction. In ventricular echoes, the return
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pathway is in an antegrade direction which is generally faster than retrograde conduction. In the latter situation, manifest reentry is more likely.

There is a divergence of opinion concerning the question whether the atrium is a necessary link in the reentrant process (10, 18, 27). As illustrated in Figure 9, the occurrence of reentry during fusion activation of the atria indicates that the entire atrial muscle is not required. Furthermore, as illustrated in Figure 10, the occurrence of a significantly shorter retrograde A to antegrade H interval (45 msec) as compared to the normal A-H interval of 65 msec during sinus rhythm suggests that the reversal occurs in the AV node and that the atria were not required. Thus, the A-H interval of 45 msec is spuriously short. An alternative possibility to explain the shorter A-H interval of the reentrant beat would require invoking the concept of super-normal conduction. This latter possibility appears less attractive, since faster than normal AV conduction has not been demonstrated.

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