Concealed Reentry: A Mechanism of Atrioventricular Nodal Alternating Wenckebach Periodicity

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SUMMARY Alternating Wenckebach periodicity is generally believed to reflect bilevel block due to horizontal electrophysiologic dissociation in the conducting tissues. We describe a case of atrial pacing-induced atrioventricular nodal alternating Wenckebach periodicity with concealed reentry in a longitudinally dissociated AV conduction system. Three observations support this unique mechanism: Concealed reentry occurred during the course of alternating Wenckebach periodicity; it could be invoked to explain the peculiar conduction patterns in this patient; and alternative mechanisms did not explain the alternating Wenckebach periods. Our findings also indicate that long conduction times during 2:1 block can be due to concealed reentry rather than concealed antegrade conduction, and participation in the echo process of a retrograde pathway with electrophysiologic characteristics of an atrioventricular nodal bypass tract does not exclude manifest and concealed reentry at a sub atrial level.

ALTERNATING Wenckebach periodicity (AWP) is a conduction disturbance characterized by basic 2:1 block with progressive prolongation of conduction time on each transmitted impulse until the series is terminated by two or three consecutive blocked beats. Horizontal dissociation of the conduction system into a proximal and distal level may be involved in this arrhythmia, one site being responsible for the 2:1 block and the other for the Wenckebach pattern of the conducted beats. We present a case of pacing-induced atrioventricular (AV) nodal AWP with a previously undescribed mechanism: concealed reentry on a longitudinally dissociated AV conduction system.

Case Report

A 29-year-old man was evaluated because of a long history of symptomatic paroxysmal supraventricular tachycardia. The patient gave informed consent for the study.

Catheter electrodes were introduced percutaneously through peripheral veins and were positioned at the right atrium, coronary sinus, His bundle area and right ventricular apex. Three surface ECG leads and three intracardiac leads were displayed on a Siemens 803 ink-jet recorder at a paper speed of 100 mm/sec. Pacing studies were performed by a custom-built programmable pacemaker. The study protocol was similar to that used in other laboratories for evaluation of arrhythmias, and included incremental atrial and ventricular pacing and extrastimulation at various paced cycle lengths (CLs) and during the tachycardia.

Figures 1 and 2 show the evolution of AWP. At atrial CLs of 300–280 msec, there was a basic 2:1 AV nodal block with two distinct populations of AV nodal conduction times of the transmitted impulses. The short AH times ranged from 195–225 msec and the long AH from 285–335 msec; no intermediate values were found. The transitions from short to long and from long to short conduction times always had a similar pattern.

An example at a paced CL of 280 msec is shown in figure 1. Panel A shows 2:1 AV nodal block. AH times of the transmitted impulses were 205–210 msec. A second consecutive beat unexpectedly conducted to the ventricles with a long conduction time (AH, 330 msec), i.e., the 2:1 block was temporarily interrupted by a 3:2 Wenckebach conduction (panel B). Thereafter, 2:1 block resumed but AH intervals of the transmitted impulses were considerably longer than before. Transition from 2:1 block with long conduction times to 2:1 block with shorter conduction times took the form of AWP (fig. 1C). There was 2:1 block with gradual prolongation of AH intervals of the transmitted impulses until two consecutive blocked beats occurred. Then, 2:1 block with short AH intervals reappeared as in figure 1A.

Figure 2 shows similar events at a slightly longer CL. Panel A shows the evolution of 2:1 block with long conduction times; panel B (continuous with panel A) shows AWP terminating in two blocked beats and followed by 2:1 block with shorter conduction times.

During these events, the pacemaker was turned off several times at different portions of the cycles to allow emergence of echo beats (fig. 3). Atrial echo beats appeared whenever the pacer was turned off after a transmitted beat with relatively long conduction. This was seen during stable 2:1 conduction (fig. 3A), just after transition from the short to the long conduction time (fig. 3B), and at different portions of the evolving AWP (fig. 3C). The 2:1 block was never associated with echo beats when relatively short conduction was present (fig. 3D) or when the pacemaker was turned off after a blocked beat. These observations were not surprising; it was clear from the ordinary Wenckebach cycles that the critical AH time required for echo beats during continuous pacing was 245–250 msec (fig. 4). The short conduction times during 2:1 block were always below and the long conduction times were always above this value.

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By further decreasing the CL to 250 and 230 msec, stable 2:1 AV nodal block developed. AH intervals of the transmitted impulses ranged from 200–210 msec. AWP and echo beats were not observed.

Analysis of the reentry process revealed that atrial echo beats (Ae) occurred over a "concealed AV nodal bypass tract."7 The HAe intervals (as measured from the onset of the respective deflections) were constant at 30 msec during single atrial echoes and during induced paroxysmal supraventricular tachycardia, irrespective of the length of the preceding AH intervals. Rapid ventricular pacing and ventricular extrastimulation disclosed fast and all-or-none retrograde His–atrium conduction (fig. 5). The HA intervals during ventricular pacing were similar to the HAe intervals of echo beats (figs. 3–5). The fast pathway was probably not available for antegrade conduction; the AV nodal conduction curves as tested by the atrial

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

**Figure 1.** Two-to-one atrioventricular nodal block with short (A) and long (B) conduction times of the transmitted impulses. (C) Atrioventricular nodal alternating Wenckebach periodicity. CL = cycle length of stimulation; HRA = high right atrial lead; HBE = His bundle lead; S = stimulus artifact; A and H = low right atrial and His bundle deflections in the His lead. V denotes the onset of ventricular activation. Time intervals are in milliseconds. Time lines are substituted by pacemaker artifacts in each figure. Paper speed is 100 mm/sec.
extrastimulus method were smooth at different basic CLs and two nonoverlapping ranges of AH intervals were not found during the ordinary Wenckebach cycles.8

During right atrial extrastimulation, critical A2H intervals were associated with single atrial echo beats. With right atrial pacing at critical CLs of 380–310 msec, an AV nodal reentrant paroxysmal supraventricular tachycardia could be induced. The tachycardia was sustained only if the last paced beat of the Wenckebach sequence that initiated reentry displayed sufficient AV nodal delay.8

Discussion

AWP in this case probably evolved as follows (fig. 6): At atrial CLs ranging from 300–280 msec, there was a basic 2:1 block. AV nodal conduction times of the transmitted beats ranged from 195–225 msec, and this AH interval was not long enough for echo to occur (figs. 1A, 3D and 6A). The 2:1 block was sometimes interrupted by 3:2 Wenckebach conduction (figs. 1B, 2A and 6A). Because AV nodal conduction time of the second consecutively transmitted beat exceeded the critical delay, reentry occurred to the proximal junction of the two pathways within the AV node (fig. 6A). The echo process was not manifest because the coupling time of the expected reentrant beat at the right atrium was longer than the CL of stimulation. The next impulse blocked proximal to this site. The next conducted beat also had a long conduction time due to relative refractoriness in the antegrade limb of the circuit created by the concealed penetration of the reentrant impulse. The presence of the concealed echoes perpetuated the longer conduction times of transmitted beats during 2:1 block. If then for any reason the antegrade conduction time of a single beat became somewhat longer during 2:1 block with concealed reentry, this inevitably initiated events that eventually led to two consecutive blocked beats (figs. 1C, 2 and 6B). The longer antegrade conduction time postponed arrival of the reentrant beat at the proximal junction. Therefore, the next atrial impulse came earlier during the relative refractoriness of the AV node. Antegrade conduction was even more depressed and the echo was even more postponed. The later the echo arrived at the initial common pathway after its discharge by the blocked beat, the higher the reentrant impulse could penetrate. This self-

![Figure 2](https://circ.ahajournals.org/doi/fig/10.1161/01.cir.68.4.1271)

**Figure 2.** Transitions from short to long (A) and from long to short (B) conduction times during 2:1 block. The latter takes the form of alternating Wenckebach periodicity. Abbreviations are as in figure 1.
perpetuating sequence of events was finally terminated when an echo eventually discharged the proximal AV node within the initial common pathway and created effective refractoriness for a second consecutive impulse (fig. 6B). Since refractoriness at this site was "peeled back" by the concealed echo, the next atrial beat could travel down to the ventricles with a short conduction time. An alternating Wenckebach period was terminated by two consecutively blocked beats:

The first block occurred at a more distal and the second at a more proximal site within the initial common pathway of the proximal AV node.

Four mechanisms could explain the AWP in this patient. The long and short conduction times during 2:1 block could have been due to dual AV nodal pathways. However, this patient had smooth, continuous AV nodal conduction curves during antegrade conduction studies using the extrastimulus technique at several paced CLs, and AH intervals during ordinary Wenckebach cycles did not fall into a long and short population. By analyzing several simple Wenckebach cycles, the apparent gap between 225 and 285 msec, i.e., between the postulated upper limit of fast and lower limit of slow pathway conduction, was completely filled in by intermediate AH intervals (fig. 4). In contrast, during AWP, the gap between 225 and 285 msec was never filled in, despite pacing at several CLs. These observations suggest that two pathways with different electrophysiologic properties were not available for antegrade conduction. Nevertheless, the possibility of dual antegrade pathways cannot be eliminated. In this event, concealed reentry would still be responsible for maintaining slow-pathway conduction and for evolution of the alternating Wenckebach periods.

Deeper concealed penetration of nonconducted impulses during 2:1 block could have produced AWP and the longer population of conduction times. If so, the last (nonconducted) atrial beat of the 3:2 Wenckebach cycle should have blocked high in the AV node, and the next beat should have conducted with a short rather than a long AH interval. Furthermore, during 2:1 block with long conduction, alternate nonconducted impulses should have blocked high in the AV node, resulting in shorter conduction of the next transmitted impulse. In figures 1B and 2A, this was not the case.

A third explanation would require the presence of stressed 2:1 conduction. Under these circumstances, a beat slowly conducting through the AV node would cause the next transmitted beat to fall within the relative refractory period of the AV node. A gradual increase in AV nodal refractoriness would then result in progressive prolongation of conduction times of the alternate impulses, with an eventual block of two consecutive atrial beats. This mechanism, previously suggested to explain AWP, probably did not occur in our patient. First, the 2:1 conduction at CLs of 300–280 msec was not at all stressed. At a slightly longer CL of 305 msec, there was AV nodal Wenckebach conduction (fig. 4B), and even at CLs of 300–270 msec, the 2:1 block was sometimes interrupted by a temporary 3:2 Wenckebach (figs. 1B and 2A). Moreover, 2:1 conduction with fairly short AH intervals of the transmitted beats was maintained at considerably shorter CLs, down to 230 msec. In any case, had the above mechanism been valid, slow conduction and a Wenckebach sequence of alternate beats should have reappeared after perturbation of the 2:1

\[ \text{Figure 3. Atrioventricular nodal echo beats (Ae) occur whenever the pacing is terminated after relatively slow atrioventricular nodal conduction (A-C). Relatively short conduction times are not associated with echo beats (D). Abbreviations are as in figure 1.} \]
sequence by the two consecutively blocked beats. In our case, however, despite the continuous pacing at the same rate, 2:1 block with short conduction times of the transmitted beats was maintained for long periods after two consecutively blocked beats.

The most widely accepted explanation suggests that two levels of block are responsible for the typical conduction patterns of AWP. In our case, however, such a mechanism does not explain the phenomenon. Wenckebach conduction at a proximal site with 2:1 block at a more distal level does not hold. In brief, there is no reason why 1:1 conduction with short conduction times at a proximal structure should suddenly switch to slower conduction and Wenckebach periodicity, with short conduction times reappearing after the blocked beat and remaining stable for long periods. The reversed pattern, i.e., 2:1 block at a proximal site and Wenckebach conduction at a more distal site, does not explain AWP terminating in two consecutively blocked beats.

Concealed reentry had been clearly shown to occur during AV nodal retrograde Wenckebach cycles. But, as reported by Simson et al., the presence of concealed reentry may not mean that it contributed to the type I block. Figure 4 in our case was an example of a simple Wenckebach conduction coexisting with manifest and concealed reentry where a cause-and-effect relationship between the two phenomena could not be proved. Alternative mechanisms explaining Wenckebach periodicity could not be ignored; concealed reentry may well have been uninvolved during the ordinary Wenckebach cycles. On the other hand, concealed echoes were not only easily invoked to explain AWP in this case, but alternative mechanisms did not explain AWP. Both reentry and the alternating Wenckebach were critically related to a certain degree of AV nodal delay, and other factors, such as rate and RP intervals, did not seem critical for the evolution of AWP.

Certain features of the reentry process in this case are of theoretical interest. Although the retrograde pathway had all the characteristics of an AV nodal bypass bundle, both manifest and concealed reentry occurred at a subatrial level. This assumption was based on two observations: The CL of the sustained tachycardia was much shorter than the longest atrial pacing CL associated with AV nodal Wenckebach periodicity, and the AWP could only be explained by subatral concealed reentry. The functional anatomy of the AV junction in patients who exhibit antegrade or retrograde conduction characteristics of an AV nodal bypass tract is a widely debated question.

Our observations strongly suggest that the presence of an apparent retrograde AV nodal "bypass" tract capable of very rapid and all-or-none conduction may not imply that these fibers are completely isolated from the main body of the AV node. The anatomic substrate of this type of conduction is not known; thus,
The term "AV nodal bypass tract" should be used in a descriptive rather than in a morphologic sense.

References

FIGURE 5. Retrograde conduction over an atrioventricular nodal bypass tract. (A and B [continuous]): One-to-one retrograde conduction followed by a type II second-degree retrograde His-atrium block. (C, D, and E) Retrograde refractory study at a driven cycle length of 700 msec. The shortest attainable $H_2$, $H_3$, interval (i.e., the functional refractory period of the His-Purkinje system in the retrograde direction, 380 msec) exceeded the effective and relative refractory periods of the retrograde His-atrium pathway. $S_1$, $H_1$, and $L_1$, denote the stimulus artifacts, His deflections, and left atrial deflections of the basic drive beats. $H_2$, $A_2$, and $L_2$, represent the His, low right atrial, and left atrial deflections of the premature impulses ($S_2$). $H_1$ and $H_2$ are validated by the constant $H$-$L_A$ intervals on all ventricular beats. The $V_1$ in panel E is most likely due to His-Purkinje reentry. CS = coronary sinus; other abbreviations as in figure 1.

FIGURE 6. The proposed mechanism of evolution of 2:1 block with long conduction times of the transmitted impulses (A) and alternating Wenckebach periodicity (B). Dotted horizontal bars represent effective refractoriness and open bars represent relative refractoriness in the antegrade limb of the atrioventricular node.
CONCEALED REENTRY IN ALTERNATING WENCKEBACH/LITTMANN AND SVENSON

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L Littmann and R H Svenson

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