Electrophysiologic Mechanisms of Orthodromic Tachycardia Initiation During Ventricular Pacing in the Wolff-Parkinson-White Syndrome

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Orthodromic tachycardia is the most common arrhythmia in patients with Wolff-Parkinson-White syndrome. It is often initiated during incremental ventricular pacing that requires the onset of retrograde block along the normal pathway (that is, atrioventricular [AV] node-His-Purkinje system) with concomitant retrograde atrial activation by way of the accessory pathway. However, the site of retrograde block, that is, the AV node versus the His-Purkinje system, during incremental ventricular pacing and, hence, the mechanism of orthodromic tachycardia initiation have not been systematically elucidated.

The mechanisms of orthodromic tachycardia induction were studied in 17 patients with Wolff-Parkinson-White syndrome using a specially designed pacing protocol. A beat by beat analysis indicated that the retrograde His-Purkinje system block was the most common initiating mechanism of orthodromic tachycardia in 14 of the 17 cases. In two cases, AV node block preceded the onset of orthodromic tachycardia, whereas the data in the remaining case suggested that both mechanisms were operative but at different pacing cycle lengths. The orthodromic tachycardia induction with His-Purkinje system block occurred within the first two cycles in most cases. When orthodromic tachycardia initiation was delayed beyond the first two cycles of the ventricular train it represented either 1) a 2:1 block in the His-Purkinje system; 2) a linking phenomenon in the His-Purkinje system; or 3) a block in the AV node. These data have methodologic, mechanistic and therapeutic implications for patients with the Wolff-Parkinson-White syndrome.

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Manuscript received April 7, 1986; revised manuscript received July 14, 1986, accepted August 8, 1986.

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systematic manner, utilizing unique pacing protocols that permitted a beat by beat analysis during constant cycle length ventricular pacing.

It is the purpose of this report to provide new insight into the mechanisms of orthodromic tachycardia initiation during ventricular pacing. We also address the complex electrophysiologic phenomena and interplay of various electrophysiologic variables associated with orthodromic tachycardia initiation during ventricular pacing.

**Methods**

**Study patients.** Standard electrophysiologic studies were carried out in 17 patients with previously documented orthodromic tachycardia after informed consent was obtained. The routine conduct of cardiac electrophysiologic studies in our laboratory has been previously described (8) and will not be detailed here. Only two patients in the series had associated significant structural heart disease, that is, one had hypertrophic cardiomyopathy and the other had coronary artery disease.

**Electrophysiology.** Ventricular electrograms from the right or left atrium (from the coronary sinus), or both, His, and right bundle in some cases, were recorded at filtering frequencies of 30 to 500 Hz in all patients. Orthodromic tachycardia initiation was attempted with: 1) incremental atrial pacing; 2) atrial extrastimulus technique; 3) incremental ventricular pacing; and 4) ventricular extrastimulus technique. Included in this study were only those cases in which the orthodromic tachycardia was induced during constant cycle length incremental ventricular pacing.

**Special pacing protocol.** Once it was established that the orthodromic tachycardia could be induced with incremental ventricular pacing, a special pacing protocol was employed to study the mechanism of such an induction (Fig. 1). The method depicted in Figure 1A (A to V train sequence) simulates the routinely used method of orthodromic tachycardia induction, that is, initiation of ventricular pacing during sinus rhythm, except that 1) the sinus rhythm is replaced with atrial paced rhythm (S1, white arrow) to avoid any cycle length variations inherent in spontaneous rhythms; 2) the first beat of the ventricular paced train (S2, black arrow) was introduced after a programmed interval (S1,S2) which could be varied to study the effect of this variable on orthodromic tachycardia induction; and 3) each beat of the rapid ventricular paced train (S3, S4, and so on) was introduced in a stepwise fashion to analyze the influence of prior beats before introduction of additional ones. Although successive beats of the train are labeled S3, S4, S5, and so on, the interval between each complex measures the same. In other words, the S3S4 = S4S5 = S5S6, and successive intervals, therefore, represent a constant cycle length train. This designation of increasing numerals was used to more clearly depict the resultant events from each corresponding paced complex, such as retrograde His deflections (H2, H3 and so on) and atrial potentials (A2, A3, and so on).

The pacing technique shown in Figure 1B is essentially similar to that in Figure 1A except that the atrial pacing during the basic drive (S1,S2) is replaced by ventricular pacing. This method, which will be referred to as V to V train sequence, was tried to compare it with the technique shown in Figure 1A and to test its relative usefulness in orthodromic tachycardia initiation. The protocol depicted in Figure 1B does not require a sequential train from the two sites (A and V) and may be more widely applicable because many of the available stimulators are not capable of delivering programmable sequential trains from different sites as depicted in Figure 1A.

The S1S1 duration was chosen to be shorter than the sinus cycle length to allow reliable and consistent pacing from the atria as well as the ventricles at the same cycle length. In most cases the S1,S2 interval was programmed to equal S1,S1. In addition, however, S1,S2 intervals measuring longer or shorter than the S1,S1 were also tried in some patients. The cycle length duration of the ventricular paced train (S2S3, S3S4 interval, and so on) was initially set so that orthodromic tachycardia was not induced, that is, it exceeded values at which the effective refractory period of the His-Purkinje system was encountered (usually 70% of the

![Figure 1](image-url)
The train cycle length was gradually shortened by 10 to 20 ms until the orthodromic tachycardia was initiated at a given S1S1 and S1S2. A maximum of eight beats were used in the paced ventricular train (up to V9) in cases in which orthodromic tachycardia was not induced with a lesser number of programmed complexes.

**Definition of terms.** The definition of terms for the routine measurement of cardiac conduction times for our laboratory have been previously published (4,9) and will not be detailed here. During pacing from a given site (atrium (A) or ventricle (V)), the measurements were made from the corresponding stimulus artifact. Therefore, the terms S1 or A1 during atrial pacing or S1 or V1 (also S2 or V2, and so forth) during ventricular pacing are used interchangeably in the text. The effective refractory period of the His-Purkinje system was defined as the longest V1V2 interval in which V2 retrogradely blocked below the His bundle recording site.

**Results**

**Electrophysiologic Data**

The pertinent electrophysiologic data are summarized in Table 1. At the time of study all patients had sinus rhythm and all antiarrhythmic drugs had been discontinued at least five half-lives before the electrophysiologic evaluation. Ventricular pre-excitation was overt in 13 of 17 cases and the remaining four (Cases 5, 14, 15 and 17) had so-called concealed Wolff-Parkinson-White syndrome with only intact retrograde conduction.

In all patients, the orthodromic tachycardia was initiated in the laboratory by one of the pacing techniques described in Figure 1. As depicted in Table 1, the orthodromic tachycardia induction was attempted with only an A to V train sequence in 14 of 17 (Cases 1 to 14) and a V to V train sequence in three (Cases 15 to 17). Both methods were employed in four cases (Cases 2, 3, 5 and 8). The A to V train sequence was tried in the majority of cases, primarily because it most closely simulates 1) the routine method of orthodromic tachycardia induction with ventricular pacing; and 2) the onset of spontaneous orthodromic tachycardia following ventricular premature beats during sinus rhythm. Therefore, data and illustrations pertaining to the A to V train sequence will be presented first.

**Mechanisms of Orthodromic Tachycardia Initiation**

**Orthodromic tachycardia induction with A to V train sequence.** Figure 2 (Case 1) displays the typical application of the pacing protocol and the sequence of events during induction of orthodromic tachycardia. The basic atrial paced cycle length (S1S1) measures 600 ms in all panels. The process of ventricular train initiation is depicted in Figure 2A. The pause between the last beat of the atrial drive and first beat of the ventricular train (S2S3) measures 800 ms. During an A to V train sequence the corresponding V1V2 interval measures less than the programmed S1S2 interval by the duration of the AV interval. This difference between S1S2 and corresponding V1V2 intervals (not labeled in Fig. 2) is constant (105 ms in this case) at all cycle lengths tested in a given patient.

Figure 2A shows the first cycle length of the ventricular train with the V2V3 interval of 320 ms. No retrograde His bundle deflection is seen after V2 but it clearly follows the V1. Retrograde atrial activation during both V2 and V3 occurs by way of the accessory pathway because the corresponding A2A1 interval = V2V3, despite marked delays along the bundle branches within the normal pathway (S1Hs = 125 ms). Orthodromic tachycardia is not initiated and sinus rhythm follows. Another beat (S4) is added to the ventricular train in Figure 2B and all preceding events are left undisturbed. V4 now blocks in the His-Purkinje system, conducts to the atria through the accessory pathway (A4) and anterogradely reaches the His bundle through the AV node. There is a sudden jump in the VH interval after V4 compared with V3, suggesting a switch of His bundle activation from retrograde (with V3) to anterograde depolarization (4). It is important to note that the A4 impulse encounters no intranode delay such that the A4-H (85 ms, not labeled) equals the AH interval of sinus beats and is much shorter than the AH interval of all subsequent beats. This lack of concealed conduction of V4 in the AV node supports the theory that V4 blocked in the His-Purkinje system. The orthodromic tachycardia is initiated with V4 and the first beat of tachycardia conducts with a left bundle branch block pattern due to concealed penetration of the left bundle branch by V4 during its block below the His bundle. This is the primary reason for the high incidence of left bundle branch block during right ventricular pacing at the onset of orthodromic tachycardia in patients with Wolff-Parkinson-White syndrome (10). The longer ventriculoatrial interval (by >60 ms) after the beat with left bundle branch block, as compared with subsequent narrow QRS complexes during the induced orthodromic tachycardia, suggests the left free wall location of the accessory pathway (11).

Although the ventricular train cycle length is kept constant at 320 ms, a shortening in the S1S2 interval to 600 ms (Fig. 2C) results in the disappearance of retrograde His-Purkinje system delay and no retrograde His bundle deflections can be identified. Consequently, the lack of retrograde His-Purkinje system block results in abolition of orthodromic tachycardia initiation. At the same S1S2, however, with a decrease in the ventricular train cycle length to 300 ms (Fig. 1D), retrograde His-Purkinje system delay and block resume and events similar to those seen in Figure 1B are encountered. The S3 is ineffective because ventricular activation has already begun. The sequence of events presented in Figure 2A to D indicates that: 1) the onset of
Table 1. Electrophysiologic Findings in 17 Cases

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All electrophysiologic data are in ms. *The longest train cycle length associated with orthodromic tachycardia initiation; † values less than ERP-ventricular ERP. AP = accessory pathway; AVN = atrioventricular node; BCL = basic cycle length; CL = cycle length; ERP-HPS = effective refractory period of His-Purkinje system; HBE = His bundle electrogram; LFW = left free wall; Loc = location; OT = orthodromic tachycardia; PS = posteroseptal; RFW = right free wall; RVP = right ventricular pacing; VA = ventriculoatrial interval.
orthodromic tachycardia is secondary to progressive delay (with \( V_3 \)) and block (with \( V_4 \)) in the His-Purkinje system; 2) His-Purkinje system conduction delay and block can be abolished by a decrease in the preceding cycle length (\( S_1S_2 \) interval); and 3) at shorter preceding cycle lengths, such delays can be reestablished at shorter ventricular train cycle length.

The mechanism whereby retrograde His-Purkinje system block during one of the beats of ventricular train initiated orthodromic tachycardia was observed in 13 of 14 patients during \( A \) to \( V \) sequence of stimulation. However, the manner with which such a block was achieved varied considerably and will be presented later. Further shortening in the \( S_1S_2 \) interval to 500 ms (the corresponding \( V_1V_2 \) being <400 ms) no longer allowed retrograde His-Purkinje system block to occur at any cycle length of the ventricular train. However, another mechanism of orthodromic tachycardia initiation emerged after a prolonged ventricular train duration and these results are displayed in Figure 3. Panels A to C show progressive increases in the number of beats within the ventricular train at a cycle length of 320 ms. None of the paced beats (in any of the panels) are accompanied by retrograde block in the His-Purkinje system, thus permitting intranodal penetration of each paced impulse. Such a turn of events would make it difficult to initiate orthodromic tachycardia because anterograde intranodal propagation of the impulse reaching the atria through the accessory pathway will be impeded, which is caused by retrograde impulse.
conduction across (or concealment within) the AV node. However, continued pacing at the same cycle length but for a longer duration ($V_a$) initiates orthodromic tachycardia (Fig. 3C). Intranodal penetration of $V_a$ is suggested by the longer AH ($A_V-H$) interval compared with the sinus or subsequent beats of orthodromic tachycardia. This can be contrasted with Figure 2B and 2D in which the site of retrograde block is in the His-Purkinje system and hence there is no evidence of intranodal concealed conduction.

Within the range of cycle lengths tested (which are typical of those utilized during the conduct of routine clinical electrophysiologic studies), intranodal block of the retrograde impulse was responsible for the initiation of orthodromic tachycardia in one other case (Case 13) during an A to V train sequence. In both instances a prolonged duration of the ventricular train ($V_b$ or $V_c$) was needed to start the orthodromic tachycardia with this mechanism.

**Initiation of orthodromic tachycardia with V to V train sequence.** This method of pacing was tried in seven patients (Cases 2, 3, 5, 8, 15–17) and in four of these cases (Cases 2, 3, 5 and 8) an A to V train sequence was also tested at the same cycle lengths (Table 1). The site of retrograde block starting the orthodromic tachycardia was in the His-Purkinje system in six of seven cases (Fig. 4) and in the AV node in one (Case 15). Where both A to V and V to V trains were tested in the same patient, the site of retrograde block remained the same, that is, the His-Purkinje system in all four cases with both methods of stimulation. However, it was noted that at the same basic cycle length and $S_5S_6$ pause, retrograde block was achieved at a longer cycle length of the paced ventricular train during a V to V sequence (compare Figures 4 and 5 for Patient 2). This difference is readily explained by realizing that during the A to V sequence the corresponding $V_1V_2$ intervals always

![Figure 3. Case 1. Induction of orthodromic tachycardia with A to V train sequence. The general format is the same as in Figure 2. The $S_5S_6$ interval is further decreased to 500 ms in A to C. At a ventricular train cycle length of 320 ms, a progressive number of programmed beats is depicted (two in A, six in B and eight in C). Orthodromic tachycardia onset is seen in C. The first AH ($A_V-H$) interval of the tachycardia is significantly longer than the AH interval of sinus beats (A and B), suggesting retrograde concealed conduction of $V_a$ in the AV node. See text for explanation. Abbreviations as in Figure 2.](image-url)
Figure 4. Case 2. Initiation of orthodromic tachycardia with V to V train sequence. A single cycle of the V train is introduced (A) during a basic drive of 500 ms after a pause of 600 ms. The S₁ produces an A₁ response through the accessory pathway and His bundle activation by way of the left bundle branch (S₁H₁ < S₁RB₁). S₂ is added in B and no His bundle deflection follows the V₄. This suggests either accommodation of the His-Purkinje system conduction (His activation obscured by the local V electrogram) or block of V₄ in the His-Purkinje system. The latter is unlikely because A₄ blocks anterogradely proximal to the His bundle. This accommodation of His-Purkinje system conduction and consequent intranodal concealment prevents orthodromic tachycardia induction even during extended duration of the train (C). When the cycle length of the V train is shortened from 340 to 330 ms (D), V₄ blocks in the His-Purkinje system, conducts retrogradely through the accessory pathway and the orthodromic tachycardia is initiated. Note again that the A₄-H interval measures less than subsequent AH intervals and is only slightly longer than the AH at the sinus cycle length (last beat in A and B). The same sequence of events would be expected if V₄ in B had blocked in the His-Purkinje system.

CS = coronary sinus electrogram; RB = right bundle branch electrogram; other abbreviations as in Figure 2.

Patterns of His-Purkinje System Block Preceding Orthodromic Tachycardia Onset

The introduction of a progressive number of beats in the ventricular train revealed a variety of patterns of His-Purkinje system delay and block before orthodromic tachycardia started. In 11 cases (Cases 3–6, 8–12, 16 and 17) the second beat of the ventricular train (V₂) abruptly blocked in the His-Purkinje system and was not preceded by any detectable delay in the His-Purkinje system. Four cases (Cases 1–3, 14) demonstrated a delay of V₁ and a block of V₄ in the His-Purkinje system (Fig. 2 and 4). In all of the above instances retrograde block in the His-Purkinje system was associated with concomitant atrial activation through the accessory pathway. Orthodromic tachycardia would obviously not start if a simultaneous block of the ventricular impulse occurred in the His-Purkinje system and accessory pathway. In such instances an extended train was necessary to start the orthodromic tachycardia (Fig. 5).

In three cases (Cases 7, 9 and 11) another phenomenon was observed in which either the V₃ or V₄ did block in the His-Purkinje system (with concomitant atrial activation through the accessory pathway), and yet the next atrial impulse failed to start the orthodromic tachycardia and anterogradely blocked in the His-Purkinje system (Fig. 6). This in turn resulted in the block of the next beat of the ventricular train in the His-Purkinje system as well, while reaching the atria through the accessory pathway (12). This type of linking phenomenon illustrated in Figure 6 could continue in-

measured less than the programmed S₁S₂ interval by an amount equal to the duration of the AV interval of the basic drive cycles. This would shorten the His-Purkinje system cycle length preceding the V train and would necessitate the use of a shorter cycle length during the train to achieve the same degree of His-Purkinje system delay and block.
**Figure 5.** Case 2. Unusual pattern of His-Purkinje system block before orthodromic tachycardia initiation. During an A to V train sequence the V1 blocks in the accessory pathway (A) but activates the H through the left bundle branch (S1H1 < S1RB3). When V4 is added in B, it conducts to the atria over the accessory pathway. The orthodromic tachycardia is not initiated because V4 also reaches the AV node by way of the His-Purkinje system (the H4 deflection is obscured by the local V electrogram). This rapid retrograde His-Purkinje system conduction is related to the fact that V3 had blocked in the His-Purkinje system rather than propagate through the His-Purkinje system as in A. V4 cannot initiate the orthodromic tachycardia because there is no concomitant atrial activation along the accessory pathway (no A3). C. With continuation of 2:1 block in the His-Purkinje system, however, conduction of V5 to the atria through the accessory pathway initiates the orthodromic tachycardia. The onset of tachycardia after V5 is not related to a corresponding long ventriculoatrial time but is due to block of V5 in the His-Purkinje system itself. This can be deduced from the orthodromic tachycardia onset in D of Figure 4 taken from the same patient where VA (V4A4) is not prolonged and yet the orthodromic tachycardia is initiated concurrent with retrograde His-Purkinje system block. Abbreviations as in Figures 2 and 4.

**Factors Influencing the Site of Retrograde Block and Consequently the Mechanism of Orthodromic Tachycardia Initiation**

**Cycle length.** Within the ranges of S1S1 or S1S2 cycle lengths utilized, retrograde block in the His-Purkinje system was overwhelmingly more common than in the AV node using either the A to V or the V to V sequence of stimulation. Shortening the S1S2 interval to less than 450 ms during the A to V sequence invariably shortened the corresponding V1V2 to less than 350 ms. This degree of abbreviation in the cycle length produced a critical amount of shortening in the effective refractory period of the His-Purkinje system and was not associated with retrograde block in the His-Purkinje system during the ventricular train. Whenever orthodromic tachycardia was initiated with V1V2 intervals measuring less than 400 ms, the site of retrograde impulse block was always the AV node, unless the first beat of the train (the S2) blocked in the His-Purkinje system and started the orthodromic tachycardia. However, the orthodromic tachycardia initiation was seldom observed at S1S1 or S1S2 intervals of less than 400 ms in this series, even though such cycle lengths were tested. Table I lists only those cycle lengths during which the orthodromic tachycardia was actually induced, regardless of the underlying mechanism. The cycle length of the ventricular train was obviously critical. The cycle lengths depicted in the table represent the longest values associated with the onset of orthodromic tachycardia. Two of the three largest values (360 and 400 ms) after a relatively short S1S2 interval of 500 ms were seen in the two cases in which the site of block was the AV node (Cases 13 and 15). On the other hand, His-Purkinje system block occurred at a cycle length of less than 350 ms in all cases except one (Case 11) where the His-Purkinje system block occurred at a train cycle length of 380 ms but followed a relatively long S1S2 interval of 800 ms.

**Train duration.** In the majority of cases when His-Purkinje system block resulted in orthodromic tachycardia, the tachycardia was initiated by V3 or V4 (Fig. 2 and 4). Further continuation of pacing was unnecessary and, in fact,
Figure 6. Case 7. Unusual pattern of His-Purkinje system block before orthodromic tachycardia initiation. **A,** During an A to V train sequence there is a 3:2 retrograde His-Purkinje system block, that is, delay with $V_3$ ($A_1$ and $H_3$ activated simultaneously) and block with $V_4$. Subsequently a linking phenomenon is established in which the anterograde impulse blocks in the His-Purkinje system and forces the block of the next paced ventricular impulse (ventricular muscle refractoriness is an unlikely reason for failure of anterograde impulse prolongation because the effective refractory period of the ventricular muscle in this patient was 230 ms at a basic cycle length of 600 ms). Note that after discontinuation of ventricular pacing no orthodromic tachycardia is initiated. **B,** At a shorter ventricular train cycle length of 290 ms, the anterograde site of block and therefore the linking migrates distally as the supraventricular impulse captures the ventricle ($V_5$). The $V_5$ is the last paced impulse. Subsequent stimuli ($S_6$ to $S_9$) are ineffective. See text for explanation. The tracings in each panel from top to bottom are leads I, $V_1$ and right atrial and His bundle electrograms; time lines at 10 and 100 ms. $A_3 =$ retrograde atrial deflection.

It frequently either terminated or entrained the orthodromic tachycardia. In only two cases with His-Purkinje system block (Cases 2 and 7) was a $V_3$ needed to start orthodromic tachycardia. When the orthodromic tachycardia onset was delayed beyond the $V_3$, it either represented block in the AV node or resolution of the linking phenomenon in the His-Purkinje system. The two modes of orthodromic tachycardia initiation could be readily separated though by identification of the His bundle deflection sandwiched between the two successive paced complexes with a $V \rightarrow A \rightarrow H \rightarrow$ block sequence during linking.

**Location of the accessory pathway.** The majority of cases in this series had a left free wall accessory pathway and the His-Purkinje system was the most common site of block, regardless of the accessory pathway location. However, none of the six patients who had either a right free wall or posteroseptal accessory pathway demonstrated retrograde block in the AV node as the mechanism for orthodromic tachycardia initiation. The ventriculoatrial intervals on the His bundle electrogram during conduction through the accessory pathway ranged from 150 to 205 ms (mean $181.8 \pm 18.1$) in patients with a left free wall accessory pathway compared with 90 to 170 ms (mean $136.6 \pm 39.8$) in patients with either a right free wall or posteroseptal accessory pathway ($p < 0.005$). In the three patients (Cases 1, 13 and 15) in whom orthodromic tachycardia was initiated with retrograde intranodal block, the ventriculoatrial intervals through the accessory pathway are among the longest and measured 205, 185 and 200 ms, respectively.

**Comparison of responses during the current pacing protocol versus conventional extrastimulus technique.** Table 1 depicts the salient findings obtained after introduction of a single extrastimulus during a paced ventricular drive at the cycle lengths listed. Orthodromic tachycardia was induced in only 10 of the 17 cases with the conventional ventricular extrastimulus technique. In all of these cases and at all cycle lengths tested, orthodromic tachycardia was only induced when the $V_3$ blocked retrogradely in the His-Purkinje system. The orthodromic tachycardia was not initiated in the remaining seven cases because of onset of block in the accessory pathway in two cases (Cases 4 and 17) or continued propagation to the His bundle ($H_2$) and, therefore, to the AV node in the remaining five cases (Cases 1, 8, 12, 14 and 16).

**Discussion**

His-Purkinje system block during constant cycle length pacing. The results of this study clearly indicate that during so-called constant cycle length incremental ventricular...
pacing the onset of retrograde block in the His-Purkinje system is responsible for orthodromic tachycardia initiation in most cases. Within the range of cycle lengths tested in this study, the onset of ventricular pacing represents the introduction of a series of successive short cycles that closely simulate the pacing method routinely applied. It is, therefore, not unexpected that His-Purkinje system conduction delay and block were often encountered during the first few cycles, because the latter represent a series of premature beats although the technique is often referred to as constant cycle length pacing. If His-Purkinje system block does not occur early, accommodation of His-Purkinje system conduction takes place and orthodromic tachycardia onset with continuation of pacing is unlikely (13). This is because an absence of His-Purkinje system block permits retrograde penetration of the AV node, and the consequent concealment is an impediment to subsequent anterograde propagation through the AV node.

In exceptional situations, orthodromic tachycardia may be initiated despite retrograde concealed penetration of ventricular impulses into the AV node. The latter mechanism of orthodromic tachycardia initiation could be favored by the following circumstances.

**Distal site of retrograde intranodal block.** Distal intranodal block would facilitate AV node recovery of excitability. It is tempting to postulate that patients who demonstrate orthodromic tachycardia onset despite intranodal penetration may have no effective retrograde propagation along the AV node. In patients without an accessory pathway and with no demonstratable ventriculoatrial conduction through the AV node, we have identified a relatively distal site of intranodal block in most cases (14). Absence of ventriculoatrial conduction has been demonstrated in almost 40% of patients with the Wolff-Parkinson-White syndrome after surgical resection of the accessory pathway (15). In other patients with intact retrograde conduction through the AV node, successive impulses may be required to achieve a distal block and such an occurrence is also possible in patients with Wolff-Parkinson-White syndrome. Some support for this hypothesis can be marshalled from the present data. In none of the cases was the orthodromic tachycardia initiated with retrograde block in the AV node using a single extrastimulus, and yet in three cases such a mechanism of orthodromic tachycardia onset was seen with an A to V or V to V train. Furthermore, orthodromic tachycardia after intranodal block rarely occurred after the first or second cycle and needed an extended number of beats in the ventricular train. The retrograde conduction characteristics of the AV node and His-Purkinje system in patients with Wolff-Parkinson-White syndrome have not been sufficiently explored. The only existing data dealing with patterns of ventriculoatrial conduction along the nodal pathway in these cases do not separate AV node versus His-Purkinje system conduction and are, therefore, of limited value for application toward our hypothesis (15). From the data available, however, it seems reasonable to conclude that patients with rapid retrograde AV node conduction are unlikely to have orthodromic tachycardia initiation with retrograde AV node block because the latter is difficult to achieve in this population with relatively short (≤50 ms) retrograde HA intervals (16).

**Long ventriculoatrial intervals through the accessory pathway.** After a retrograde intranodal block of a paced impulse, delayed arrival of anterograde impulse at the AV node could facilitate orthodromic tachycardia initiation. With the right ventricle as the site of stimulation the ventriculoatrial interval (measured at a site close to the AV node, that is, the His bundle electrogram) tends to be longer with a left free wall accessory pathway as compared with a posteroseptal or even right free wall accessory pathway. All patients in this series who had orthodromic tachycardia onset with intranodal block did indeed have a left free wall accessory pathway and in all cases the ventriculoatrial interval was 185 ms or more, among the longest in this group (Table I). However, the presence of long ventriculoatrial times over the accessory pathway, as such, was not sufficient to start orthodromic tachycardia after retrograde AV node block, because in only three of seven cases with a ventriculoatrial interval of 185 ms or more was the orthodromic tachycardia initiated after intranodal block. Other factors, such as the site of retrograde block within the AV node and recovery of nodal excitability, would therefore seem to be important considerations. However, the data presented suggest that orthodromic tachycardia initiation with intranodal block will be less likely in patients with a septal accessory pathway.

**Limitations of the study.** One might argue that intranodal block as a mechanism of orthodromic tachycardia initiation may be more often observed if different cycle lengths were scanned. In the present study the S_S1 and S_S2 cycle lengths were selected to represent ranges routinely utilized in clinical laboratories. Application of long cycle lengths will be limited by spontaneous sinus rates and with a relatively short S_S1 cycle length (<400 ms) the first few beats of the drive could produce retrograde His-Purkinje system block and, hence, limit its utility. Longer cycle length of the ventricular train than those listed in Table 1 were tested in all patients but did not produce orthodromic tachycardia. We cannot be sure, however, whether a longer duration of the V train (beyond V_d) would have made a difference in some cases. The present data also do not address the pacing sequences whereby the cycle length during incremental ventricular pacing is continually and gradually reduced without interruption. Because His-Purkinje system accommodation can be accomplished by this technique, one could postulate that orthodromic tachycardia initiation will be uncommon by this method. If orthodromic tachycardia were induced by such a method, it would most probably be due to intranodal block, unless the paced cycle length were
relatively short (≤300 ms). At those latter cycle lengths, retrograde His-Purkinje system block is not uncommon regardless of preceding interval (13).

A possible concern with the pacing method employed might be the use of S_1 S_2 cycle lengths that often exceeded the S_1 S_1 cycle lengths. This sudden cycle length change is known to produce an increase in the His-Purkinje system refractoriness and may have artificially increased the incidence of retrograde block in the His-Purkinje system as compared with intranodal block (17–19). This argument can be summarily dismissed because S_1 S_2 = S_1 S_1 cycle length was tried in 15 of the 17 cases with essentially the same result (Fig. 2). At an S_1 S_2 cycle length that exceeds the S_1 S_1 interval, the retrograde His-Purkinje system block can, however, be achieved at a longer cycle length of the ventricular train and therefore an S_1 S_2 greater than S_1 S_1 might be an effective and practical way to increase the incidence of orthodromic tachycardia initiation in the laboratory.

The overall incidence of orthodromic tachycardia induction was higher using an A to V or a V to V train sequence compared with the single extrastimulus technique. This difference could not be secondary to the duration of either S_1 S_1 cycle lengths (the same in both situations), S_1 S_2 intervals (discussed earlier) or even the first cycle length of the ventricular train. The V_1 V_2 interval tested with the extrastimulus method encompassed all the ventricular train cycle lengths used in individual cases during this study. Higher yield during an A to V or V to V sequence in terms of orthodromic tachycardia initiation must therefore be ascribed primarily to additional beats introduced during the ventricular train. In 7 of the 17 cases in which more than two cycles of a ventricular train (V_2 or beyond) were needed to induce orthodromic tachycardia, the arrhythmia was initiated in only two of seven during the extrastimulus technique that provided a single ventricular premature beat (V_2). These observations suggest that a series of ventricular premature beats will often be more successful in inducing orthodromic tachycardia when a single premature beat fails. Such a series could be added to an atrial or a ventricular paced drive. The latter method is universally employed for induction of ventricular tachyarrhythmias, can be accomplished with most conventional stimulators and can be employed for orthodromic tachycardia induction as well.

Electrophysiologic implications. The present study represents the first systematic demonstration of the retrograde site of block preceding the onset of orthodromic tachycardia and, hence, the mechanism of orthodromic tachycardia initiation during so-called constant cycle length incremented ventricular pacing. The data indicate that onset of orthodromic tachycardia with His-Purkinje system block is more likely when 1) the tachycardia starts early (within one or two cycles); 2) it is initiated at short ventricular train cycle lengths (<350 ms); 3) there is an absence of prior retrograde concealed conduction in the AV node as indicated by lack of AH interval prolongation with the first beat of orthodromic tachycardia; and 4) left bundle branch block occurs at the onset of orthodromic tachycardia after ventricular pacing (Fig. 2 and 6). This latter aspect of orthodromic tachycardia initiation has been systematically explored by Lehmann et al. (10) and will not be detailed here. The AV node as the site of retrograde block is indicated by the induction of orthodromic tachycardia following: 1) longer ventricular train cycle lengths (>400 ms); 2) several paced cycles without discernable His-Purkinje system delay; and 3) evidence of retrograde intranodal concealed conduction at the onset of orthodromic tachycardia.

It is not unreasonable to suggest that with the pacing method utilized, orthodromic tachycardia can be initiated in virtually every patient in whom this tachycardia is clinically documented. Knowledge of this may be particularly useful for patients with short effective refractory period accessory pathway in whom anterograde accessory pathway block cannot be achieved during atrial stimulation. In these cases effective retrograde propagation along the accessory pathway and achievement of block in the His-Purkinje system (which can be accomplished with single or double premature beats) provides an ideal milieu for orthodromic tachycardia initiation using the technique described here.

The mode of spontaneous initiation of orthodromic tachycardia in the clinical setting is uncertain, but it is generally assumed that atrial or ventricular premature beats, or both, are the usual triggering mechanisms. The induction of orthodromic tachycardia with the ventricular extrastimulus method in which the premature beats are introduced during a paced rhythm represents a somewhat artificial situation because such circumstances would seldom be replicated in spontaneous clinical settings. The A to V train sequence closely simulates the spontaneous occurrence of ventricular premature beats with preceding supraventricular (or sinus) rhythm. Single or double premature beats occur often on a spontaneous basis and therefore, orthodromic tachycardia induction with retrograde His-Purkinje system block could be a likely mechanism of orthodromic tachycardia initiation during sinus rhythm. In fact, the requirement of a series of ventricular premature complexes to start orthodromic tachycardia due to AV node block may make it an unlikely mechanism of orthodromic tachycardia initiation in the clinical setting.

Clinical implications. The aforementioned observations may have important clinical implications in the prevention of spontaneous initiation of orthodromic tachycardia with antiarrhythmic drugs. Based on the results of this study, several points can be made: 1) in patients whose orthodromic tachycardia is initiated with retrograde block in the His-Purkinje system triggered by spontaneous ventricular ectopic beats, reduction of the triggering mechanism as well as prevention of block in the His-Purkinje system may be possible with lidocaine-like agents such as mexiletine or
tocainide. Lidocaine is known to shorten the refractoriness of the His-Purkinje system in humans (19). For prevention of orthodromic tachycardia, these agents have seldom been systematically tested. 2) Coincidental with a retrograde block in the His-Purkinje system, an effective propagation along the accessory pathway is essential for orthodromic tachycardia initiation. If retrograde accessory pathway block can be achieved with any of the drugs, it would represent the most effective pharmacologic prophylaxis. This, in essence, will constitute prevention of orthodromic tachycardia by direct drug action on the accessory pathway, in contrast to other pharmacologic approaches that target other components of the circuit, such as the AV node, His-Purkinje system and atrial or ventricular myocardium.

References


