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Concealed Anterograde Accessory Pathway Conduction During the Induction of Orthodromic Reciprocating Tachycardia

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The purpose of this study was to determine whether concealed anterograde accessory pathway conduction occurs during the induction of orthodromic tachycardia by an atrial extrastimulus (S_2). Sixteen patients with an overt (n = 9) or concealed (n = 7) accessory pathway had inducible orthodromic tachycardia by S_2 during an atrial drive (S_1) cycle length of 500 to 650 ms. A ventricular extrastimulus (S_3) was introduced coincident with the His depolarization resulting from S_2 during the longest S_1S_2 interval that reproducibly induced orthodromic tachycardia. The S_1S_3 interval was decreased in 10 ms steps until S_3 reached ventricular refractoriness. Retrograde accessory pathway conduction of S_3 in the presence and absence of S_2 was compared at the same S_1S_3 intervals.

In the absence of S_2 there was retrograde accessory pathway conduction after S_3 in each patient. In the presence of S_2, in patients with overt pre-excitation, retrograde accessory pathway conduction after S_3 was absent in one patient, prolonged in four patients and present only after long S_1S_3 intervals in three patients. Only one patient had unchanged retrograde conduction regardless of the presence or absence of S_2. In patients with a concealed accessory pathway, retrograde accessory pathway conduction after S_3 was absent in five patients and was prolonged in two. Thus, concealed anterograde accessory pathway conduction was present in 15 of 16 patients at the time of orthodromic tachycardia induction.

In conclusion, concealed anterograde accessory pathway conduction occurs in a majority of patients with an overt or a concealed accessory pathway during induction of orthodromic tachycardia by an atrial extrastimulus. In some patients, the initiation of orthodromic tachycardia may depend on a critical interaction between the degree of concealed anterograde accessory pathway conduction and atrioventricular conduction delay after S_2.

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The induction of orthodromic reciprocating tachycardia by a spontaneous or induced atrial premature depolarization in patients with the Wolff-Parkinson-White syndrome is invariably associated with loss of ventricular pre-excitation and, therefore, a relative delay in atrioventricular (AV) conduction (1–3). In contrast, in patients who have a concealed accessory pathway without overt ventricular pre-excitation, the initiation of orthodromic tachycardia is associated only with a critical delay in AV conduction (4). Recent studies (5–8) have demonstrated that concealed conduction may occur in overt and concealed accessory pathways. However, the role of concealed conduction during the initiation of orthodromic tachycardia has not been systematically studied.

The purpose of the present study was to better define the mechanism of induction of orthodromic tachycardia. The following questions were investigated: 1) In patients with an overt accessory pathway, is loss of pre-excitation during induction of orthodromic tachycardia associated with complete block into the accessory pathway, or is there concealed conduction into the accessory pathway by the atrial depolarization that induces the tachycardia? 2) In patients with a concealed accessory pathway, what is the role of concealed conduction when orthodromic tachycardia is induced, and is induction of orthodromic tachycardia dependent on loss of concealed conduction?

Methods

Characteristics of subjects (Table 1). The subjects of the study were 16 consecutive patients who underwent an electrophysiologic test for evaluation of paroxysmal supra-

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Table 1. Clinical Characteristics of 16 Subjects and Evidence of Concealed Accessory Pathway Conduction

Case	Age (yr) & Gender	AP Site	Atrial Pacing Site	V_3A' (ms)	
				A_2 Present	A_2 Absent
1	14M	L	HRA	130	115
2	15M	S	HRA	120*	90
3	14F	R	HRA	No A′	70
4	17F	R	HRA	120*	110
5	32M	L	CS	205	180
6	26M	L	CS	205	160
7	39M	L	HRA	180*	180
8	17M	L	CS	150	120
9	35M	L	CS	155	155
10	30M	S(c)	HRA	No A′	130
11	35M	L(c)	CS	No A′	135
12	30F	L(c)	HRA	No A′	165
13	31F	L(c)	HRA	No A′	140
14	34M	L(c)	HRA	160*	160
15	35F	L(c)	HRA	115*	115
16	79F	L(c)	CS	No A′	145

*A′ only occurred after long S_1S_3 intervals. AP = accessory pathway; c = concealed; CS = coronary sinus; F = female; HRA = high right atrium; L = left free wall; M = male; R = right free wall; S = posterior septum.

ventricular tachycardia and were found to have 1) a single AV accessory pathway; 2) orthodromic reciprocating tachycardia inducible by programmed atrial stimulation with a single extrastimulus; and 3) atrial pre-excitation without a change in the retrograde atrial activation sequence induced by a ventricular depolarization coincident with the His bundle potential during orthodromic tachycardia.

There were 10 male and 6 female patients in this study, had a mean age (±SD) of 30 ± 16 years (range 14 to 79). The accessory pathway was located in the left free wall in 12 patients, in the right free wall in 2 patients and in the posterior septum in 2. Nine patients had overt ventricular pre-excitation and seven had a concealed accessory pathway.

Electrophysiologic study protocol. Studies were performed with patients in the fasting, unsedated state, after they had given informed consent. Four quadripolar electrode catheters were inserted into a femoral and subclavian vein and positioned in the high right atrium, at the AV junction, against the right ventricular apex and within the coronary sinus. Electrocardiographic (ECG) leads V_1 and I and the intracardiac electrograms were displayed simultaneously on an oscilloscope and recorded at a paper speed of 100 mm/s on a Siemens-Elema Mingograf 7 or an Electronics for Medicine VR-12 recorder. Programmed stimulation was performed with a programmable stimulator (Bloom Associates, Ltd.); the stimuli were twice diastolic threshold and 2 ms in duration.

Definitions. The atrial electrogram resulting from the last basic atrial drive stimulus (S_1) was designated as A_1. The atrial, His bundle and ventricular electrograms resulting from the atrial extrastimulus (S_2) were designated as A_2, H_2, and V_2, respectively. The first atrial reentrant beat of an induced orthodromic tachycardia was designated as A_e. The ventricular electrogram resulting from a ventricular extrastimulus (S_3) introduced after S_2 was designated as V_3. The retrograde atrial depolarization resulting from V_3 was designated as A′. In this study, S_1 and S_2 were always introduced in the right atrium or coronary sinus and S_3 was always introduced at the right ventricular apex.

Stimulation protocol. Programmed atrial stimulation with a single right or left atrial (coronary sinus) extrastimulus was performed at a basic drive cycle length of 650 or 500 ms. The longest A_1A_2 interval that reproducibly induced orthodromic tachycardia was identified. S_3 was then introduced at the right ventricular apex such that it was coincident with H_2. The S_1S_3 interval was decreased in steps of 10 ms and the presence or absence of A′ after each V_3 was noted. This was continued until S_3 no longer evoked a V_3. The same S_1S_3 intervals then were repeated in the absence of A_2. The purpose of this maneuver was to determine whether there was concealed anterograde conduction of A_2 into the accessory pathway. If A′ was absent in the presence of A_2, but present with eccentric atrial activation when A_2 was absent, this was considered to be evidence of concealed anterograde conduction into the accessory pathway by A_2 (Fig. 1). In addition, if A′ was present after V_3 but the V_3A' interval in the presence of A_2 was longer than when A_2 was absent, this also was considered to be evidence of concealed anterograde conduction of A_2 into the accessory pathway. The V_3A' interval was measured from the beginning of V_3 on the

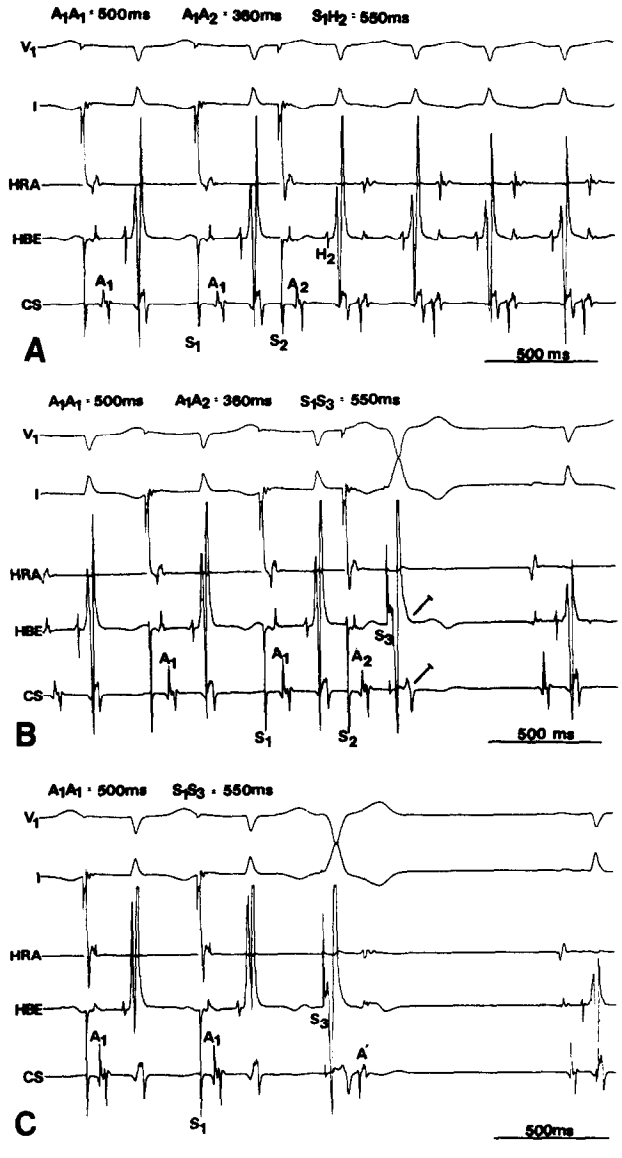

Figure 1. Patient 13. An example of concealed anterograde conduction into a left-sided accessory pathway by the atrial premature depolarization that induces orthodromic tachycardia. **From top to bottom** are leads V_1 and I, the high right atrial electrogram (HRA), His bundle electrogram (HBE) and coronary sinus electrogram (CS). **A,** During high right atrial pacing at a cycle length of 500 ms, the longest A_1A_2 interval that consistently induced orthodromic tachycardia was 360 ms. The coronary sinus electrogram preceded the other atrial electrograms during orthodromic tachycardia, consistent with a left-sided accessory pathway. The S_1H_2 interval is 550 ms. **B,** While the A_1A_2 interval remained unchanged at 360 ms, a ventricular extrastimulus (S_3) was delivered to the right ventricular apex with an S_1S_3 interval of 550 ms, coincident with H_2. There was no retrograde atrial activation after V_3. **C,** In the absence of A_2, a ventricular extrastimulus (S_3) was delivered to the right ventricular apex with a similar coupling interval of 550 ms. This resulted in retrograde atrial activation with a retrograde conduction sequence similar to that during orthodromic tachycardia, indicating retrograde conduction through the accessory pathway. Therefore, the absence of retrograde accessing pathway conduction in **panel B** can be attributed to concealed conduction of A_2 into the accessory pathway.

Table 2. Atrial Functional Refractory Period of A_2, the Accessory Pathway Retrograde Conduction Time (V_3A') and the A_2V_3 Interval of the Blocked V_3*

Case	Functional Refractory Period of A_2 (ms)	$V_3A' + A_2V_3$ (ms)
2	<180	120 + 220
3	170	†70 + 120
4	200	120 + 150
7	<200	180 + 110
10	280	†130 + 160
11	270	†135 + 140
12	235	†165 + 150
13	<270	140 + 130
14	160	160 + 40
15	<220	120 + 100
16	<220	†145 + 150

*Method to exclude atrial refractoriness as a factor in the absence of A′ after V_3 (see text). †Refers to accessory pathway retrograde conduction time in the absence of A_2.

surface ECG to the major deflection of A′ recorded near the atrial insertion of the accessory pathway.

The absence of A′ after V_3 was attributed to retrograde block in the accessory pathway caused by concealed conduction of A_2 into the accessory pathway. To rule out the possibility that the absence of A′ might instead be a result of atrial refractoriness, we determined the atrial functional refractory period after A_2. In every patient in whom V_3 was not followed by A′, the atrial functional refractory period after A_2 was shorter than the sum of the accessory pathway retrograde conduction time and the A_2V_3 intervals (Table 2). Therefore, the absence of A′ after V_3 could not be attributed to atrial refractoriness.

When there was evidence of concealed anterograde conduction of A_2 into a concealed accessory pathway, the shortest S_1S_2 interval that consistently failed to induce orthodromic tachycardia was identified and the pacing maneuvers described were repeated to determine whether the induction of orthodromic tachycardia by a critical S_1S_2 interval was associated with a change in concealed anterograde conduction into the accessory pathway by A_2.

To examine whether a critical delay in the AV node-His-Purkinje system is solely necessary for the induction of orthodromic tachycardia, the longest A_2V_2 interval associated with reproducible induction of orthodromic tachycardia was compared with the shortest A_2V_2 interval that failed to induce orthodromic tachycardia in patients who had a concealed accessory pathway and also in patients with a manifest accessory pathway whose tachycardia induction was not coincident with the accessory pathway refractory period.

In some patients, A′ was present after V_3 only at long S_1S_3 intervals and A′ was absent at shorter S_1S_3 intervals. In two of these patients, the S_1S_3 interval not associated with

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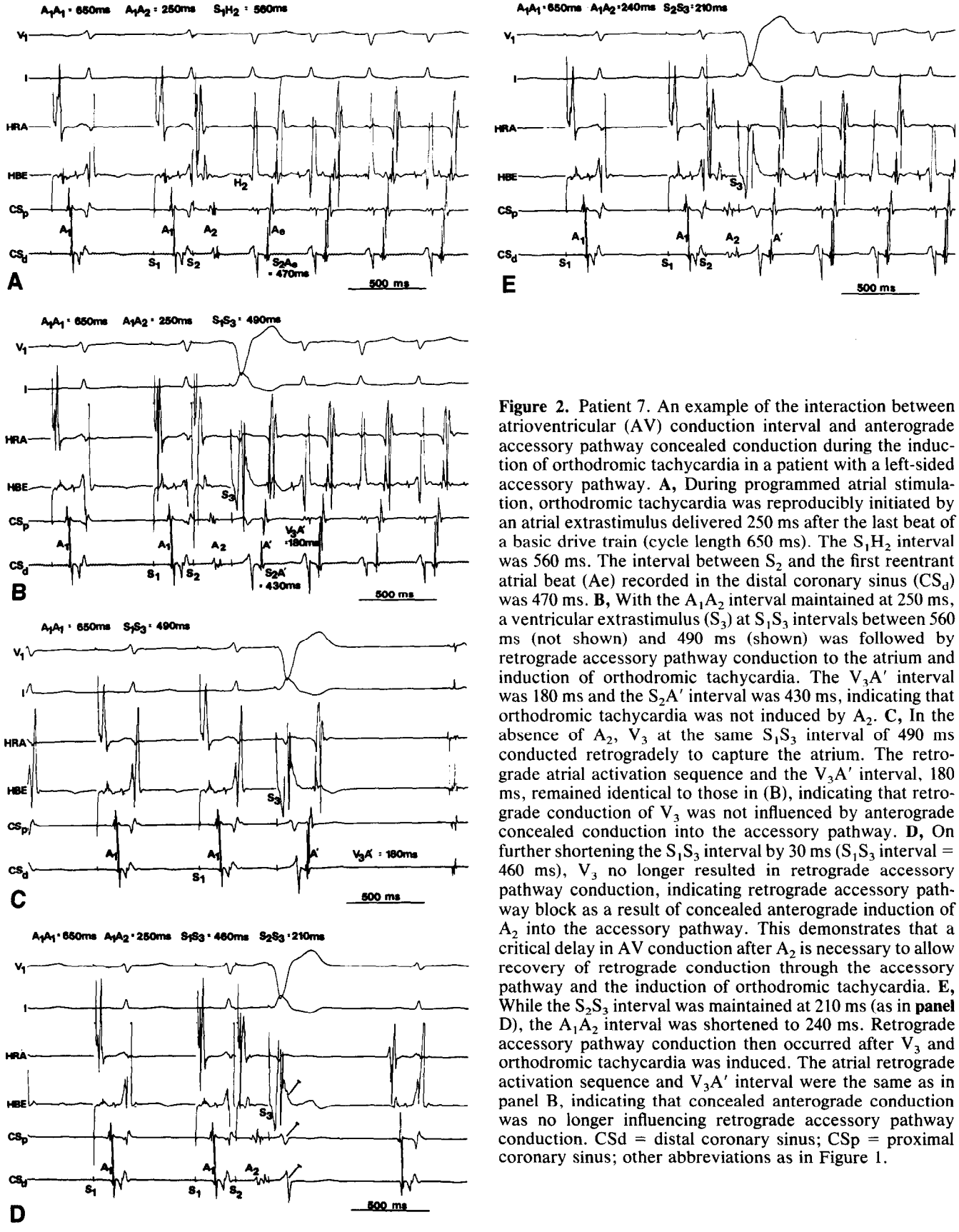

Figure 2. Patient 7. An example of the interaction between atrioventricular (AV) conduction interval and anterograde accessory pathway concealed conduction during the induction of orthodromic tachycardia in a patient with a left-sided accessory pathway. **A,** During programmed atrial stimulation, orthodromic tachycardia was reproducibly initiated by an atrial extrastimulus delivered 250 ms after the last beat of a basic drive train (cycle length 650 ms). The S_1H_2 interval was 560 ms. The interval between S_2 and the first reentrant atrial beat (Ae) recorded in the distal coronary sinus (CS_d) was 470 ms. **B,** With the A_1A_2 interval maintained at 250 ms, a ventricular extrastimulus (S_3) at S_1S_3 intervals between 560 ms (not shown) and 490 ms (shown) was followed by retrograde accessory pathway conduction to the atrium and induction of orthodromic tachycardia. The V_3A' interval was 180 ms and the S_2A' interval was 430 ms, indicating that orthodromic tachycardia was not induced by A_2. **C,** In the absence of A_2, V_3 at the same S_1S_3 interval of 490 ms conducted retrogradely to capture the atrium. The retrograde atrial activation sequence and the V_3A' interval, 180 ms, remained identical to those in (B), indicating that retrograde conduction of V_3 was not influenced by anterograde concealed conduction into the accessory pathway. **D,** On further shortening the S_1S_3 interval by 30 ms (S_1S_3 interval = 460 ms), V_3 no longer resulted in retrograde accessory pathway conduction, indicating retrograde accessory pathway block as a result of concealed anterograde induction of A_2 into the accessory pathway. This demonstrates that a critical delay in AV conduction after A_2 is necessary to allow recovery of retrograde conduction through the accessory pathway and the induction of orthodromic tachycardia. **E,** While the S_2S_3 interval was maintained at 210 ms (as in **panel** D), the A_1A_2 interval was shortened to 240 ms. Retrograde accessory pathway conduction then occurred after V_3 and orthodromic tachycardia was induced. The atrial retrograde activation sequence and V_3A' interval were the same as in panel B, indicating that concealed anterograde conduction was no longer influencing retrograde accessory pathway conduction. CSd = distal coronary sinus; CSp = proximal coronary sinus; other abbreviations as in Figure 1.

an A′ was held constant and the S_1S_2 interval was decreased in steps of 10 ms to determine whether there was an S_1S_2 interval at which A′ reappeared, suggesting either less penetration or loss of concealed anterograde conduction of A_2 into the accessory pathway when the coupling interval was shortened (Fig. 2).

Results

Patients with overt ventricular pre-excitation. Among the nine patients (Cases 1 to 9) who had overt ventricular pre-excitation, the longest A_1A_2 interval associated with loss of ventricular pre-excitation was 278 ± 57 ms and the longest A_1A_2 interval resulting in the reproducible induction of orthodromic tachycardia was 271 ± 43 ms. The longest A_1A_2 interval that reproducibly induced orthodromic tachycardia was equivalent to the longest A_1A_2 interval associated with loss of ventricular pre-excitation in eight patients. However, in one patient (Case 2), the longest A_1A_2 interval that induced orthodromic tachycardia was 160 ms shorter than the longest A_1A_2 interval associated with loss of ventricular pre-excitation.

There was evidence of concealed anterograde conduction into the accessory pathway by the atrial depolarization that induced orthodromic tachycardia in eight patients (Cases 1 to 8). In one of these patients (Case 3), no A′ followed V_3 in the presence of A_2, but A′ was present when A_2 was omitted. In four of these patients (Cases 1, 5, 6 and 8), A′ was present after the V_3 of all S_1S_3 intervals tested; however, the V_3A' interval was longer than that obtained at comparable S_1S_3 intervals in the absence of A_2. In the remaining three patients (Cases 2, 4 and 7) concealed anterograde accessory pathway conduction was evident by the absence of A′ after short S_1S_3 intervals. In each of these three patients, A′ was present after the initial V_3 that was coincident with H_2. On shortening the S_1S_3 interval by 30, 40 and 100 ms, respectively, A′ was lost indicating retrograde accessory pathway block.

There was only one patient (Case 9) in whom the induction of orthodromic tachycardia was not associated with concealed anterograde accessory pathway conduction (Fig. 3). In this patient, A′ was present after all S_1S_3 intervals tested and the V_3A' interval was the same regardless of the presence or absence of A_2.

Patients with a concealed accessory pathway. Among the seven patients (Cases 10 to 16) who had a concealed accessory pathway, the longest A_1A_2 interval that reproducibly induced orthodromic tachycardia was 357 ± 72 ms. There was evidence of concealed anterograde conduction into the accessory pathway by the atrial depolarization that induced orthodromic tachycardia in each of the seven patients. In five patients (Cases 10 to 13 and 16), A′ was absent after all S_1S_3 intervals but was present when A_2 was omitted (Fig. 1). In two patients (Cases 14 and 15), A′ was present after the initial V_3 that was coincident with H_2. On reduction of the S_1S_3 interval by 20 and 50 ms, respectively, A′ no longer was present.

Among three (Cases 14 to 16) of the seven patients who had a concealed accessory pathway, the same pacing protocol was repeated at the shortest A_1A_2 interval that consistently failed to induce orthodromic tachycardia. In these three patients, the longest A_1A_2 interval resulting in consistent induction of orthodromic tachycardia was 307 ± 38 ms and the shortest A_1A_2 interval that consistently failed to induce the orthodromic tachycardia was 327 ± 38 ms. At the A_1A_2 interval that did not induce orthodromic tachycardia each of the three patients also showed evidence of anterograde concealed conduction. The response to V_3 was the same as at the longest A_1A_2 interval that did induce tachycardia in two patients (Cases 14 and 16). In Patient 15, A′ was absent after all S_1S_3 intervals tested, suggesting a greater degree of concealed conduction at the longest A_1A_2 that failed to induce tachycardia.

A_2V_2 intervals associated with tachycardia induction. The longest A_2V_2 interval associated with the reproducible induction of orthodromic tachycardia was compared with the shortest A_2V_2 interval that failed to induce orthodromic tachycardia in all patients who had a concealed accessory pathway and in Patient 2, in whom the A_1A_2 interval that induced orthodromic tachycardia was 160 ms shorter than the accessory pathway effective refractory period. There was no change in one (Case 15) and a 5 to 45 ms increment in AV conduction time in the other seven patients (Cases 2, 10 to 14 and 16) when orthodromic tachycardia was induced.

Effect of S_1S_2 on concealed conduction. In two patients (Cases 7 and 15), an S_1S_3 interval at which there was no A′ after V_3 was identified and the S_2S_3 interval was held constant. The initial S_1S_2 interval was the same as the S_1S_2 interval that induced orthodromic tachycardia. This interval then was shortened in 10 ms steps. On shortening the S_1S_2 interval by 10 ms and 30 ms respectively, A′ was observed after V_3 (Fig. 2D and E). The retrograde atrial activation sequence remained the same as during orthodromic tachycardia and the V_3A' interval was the same as that obtained in the absence of A_2 in each case.

Discussion

Demonstration of concealed conduction. The results of this study demonstrate that there almost always is concealed anterograde accessory pathway conduction by the atrial premature depolarization that induces orthodromic reciprocating tachycardia. Concealed anterograde conduction was identified by the occurrence of retrograde block or decremental conduction in the accessory pathway after a ventricular premature depolarization coincident with the His bundle potential that followed the atrial premature depolarization that induced tachycardia.

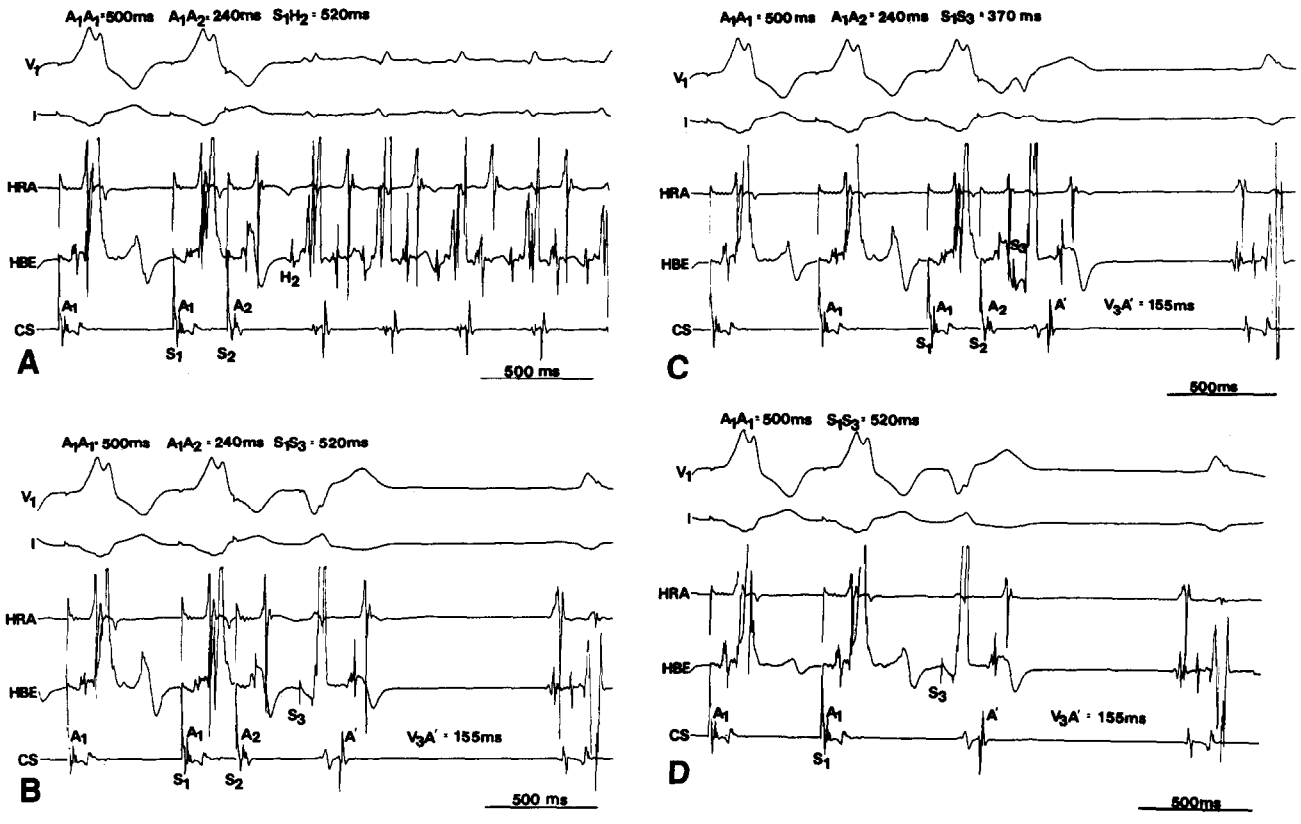

Figure 3. Patient 9. An example of absence of anterograde accessory pathway concealed conduction during orthodromic tachycardia induction. **A,** At a basic drive cycle length of 500 ms, an atrial extrastimulus at a coupling interval of 240 ms initiated orthodromic tachycardia utilizing a left-sided accessory pathway for retrograde conduction. The S_1H_2 interval was 520 ms. **B,** After the same A_1A_2 interval of 240 ms, V_3 was introduced at the right ventricular apex, coincident with the His bundle depolarization. This was followed by retrograde atrial activation, utilizing the accessory pathway for retrograde conduction. The V_3A' interval was 155 ms. **C,** Retrograde accessory pathway conduction was present after each V_3 delivered between H_2 and ventricular refractoriness. V_3 with a coupling interval of 370 ms was still able to propagate to the atrium through the accessory pathway with a similar V_3A' interval (155 ms). S_3 with a coupling interval of 360 ms (not shown) was not able to evoke a ventricular response because of ventricular refractoriness. **D,** In the absence of A_2, V_3 after an S_1S_3 interval of 520 ms was again introduced at the right ventricular apex. This was followed by retrograde conduction to the atrium through the accessory pathway. The V_3A' interval was again 155 ms suggesting the absence of concealed anterograde accessory pathway conduction at the time of orthodromic tachycardia induction. Abbreviation as in Figures 1 and 2.

The absence of an atrial depolarization after V_3 theoretically could be caused by atrial refractoriness rather than retrograde block within the accessory pathway. However, this possibility was ruled out, because determination of the refractory period of A_2 indicated that the atria were capable of being depolarized had there not been retrograde block in the accessory pathway.

The proof that retrograde accessory pathway block was caused by concealed anterograde conduction is twofold. First, intact retrograde accessory pathway conduction to the atrium could be demonstrated after the ventricular premature depolarization (V_3) when the atrial premature depolarization (A_2) that induced tachycardia was omitted. Second, a ventricular depolarization coincident with the His bundle potential during orthodromic tachycardia was followed by retrograde accessory pathway conduction to the atrium in each patient. Therefore, retrograde accessory pathway block during the pacing protocol could be explained only by concealed anterograde conduction of A_2 into the accessory pathway.

The occurrence of accessory pathway concealed conduction has been described in previous reports (5,6,10). Klein et al. (5) demonstrated that there may be concealed conduction into an accessory pathway after an atrial extrastimulus that results in the loss of ventricular pre-excitation. Kuck et al. (16) recorded accessory pathway potentials and demonstrated the presence of accessory pathway anterograde concealed conduction during sinus rhythm or atrial pacing in 8 of 10 patients whose ECG did not show ventricular pre-excitation. Winters et al. (10) also demonstrated the existence of accessory pathway anterograde concealed conduction by recording an accessory pathway potential during atrial extrastimulation. However, in these studies, the role of accessory pathway anterograde concealed conduction in the initiation of orthodromic tachycardia was not examined.

Concealed conduction in overt accessory pathways. In the case of overt accessory pathways, concealed conduction usually was not a limiting factor in the induction of orthodromic tachycardia, because orthodromic tachycardia in almost every patient was induced by the A_1A_2 interval that defined the accessory pathway effective refractory period. In these patients, the induction of orthodromic tachycardia was dependent only on block of A_2 somewhere within the accessory pathway.

However, in one of the nine patients (Case 2) who had an overt accessory pathway, the A_1A_2 interval that induced orthodromic tachycardia was 160 ms shorter than the effective refractory period of the accessory pathway. In this patient, concealed anterograde conduction at the A_1A_2 interval that defined the accessory pathway effective refractory period prevented the generation of an atrial echo. Further shortening of the A_1A_2 interval may have resulted in a lesser degree of concealed conduction of A_2 into the accessory pathway. In addition, shortening of the A_1A_2 interval also resulted in sufficient lengthening of the A_2V_2 interval to allow recovery from the effects of concealed conduction. This would explain why retrograde conduction block in Patient 2 was not demonstrable at the A_1A_2 interval that induced orthodromic tachycardia unless V_3 was introduced at least 30 ms earlier than H_2.

Concealed conduction in concealed accessory pathways. Concealed anterograde conduction appeared to be more often an important factor limiting the induction of orthodromic tachycardia when the accessory pathway was not capable of overt conduction to the ventricle. In five of seven patients, a ventricular depolarization coincident with H_2 at the A_1A_2 interval that induced orthodromic tachycardia was not able to conduct to the atrium over the accessory pathway. This observation implies that, if the A_2V_2 interval that had induced the orthodromic tachycardia had been only a little shorter, an atrial echo or the orthodromic tachycardia would not have been generated because of retrograde accessory pathway block caused by concealed anterograde conduction into the accessory pathway by A_2. Therefore, the induction of orthodromic tachycardia in these patients was dependent on a critical delay in A_2V_2 that allowed recovery of the accessory pathway from the effects of concealed conduction.

In the other two patients who had a concealed accessory pathway, the effects of concealed conduction were not apparent at the A_1A_2 interval that induced orthodromic tachycardia until V_3 was 20 to 50 ms earlier than H_2. In these patients, the A_2V_2 interval at the A_1A_2 interval that induced orthodromic tachycardia was longer than the critical delay necessary for induction of orthodromic tachycardia. This difference suggests that there may have been a lesser degree of concealed conduction in these patients.

Effects of the atrioventricular interval. When programmed atrial stimulation with a single extrastimulus is performed in a patient who has a concealed accessory pathway, orthodromic tachycardia may be induced when a critical A_2V_2 interval is reached. The factors responsible for the induction of orthodromic tachycardia at a critical A_2V_2 interval include one or both of the following: 1) a decrease in the degree of concealed anterograde conduction in the accessory pathway as the A_1A_2 interval is shortened; and 2) an increase in the AV conduction time as the A_1A_2 interval is shortened, allowing more time for the accessory pathway to recover from the effects of anterograde concealed conduction. The results of this study suggest that, at least in some patients, a decrease in the degree of concealed conduction may be the more critical factor. For example, in Patient 15, concealed anterograde conduction was more readily demonstrable at the shortest A_1A_2 interval that did not induce tachycardia than at the longest A_1A_2 interval that did induce tachycardia. In addition, in this patient, the AV interval at the shortest A_1A_2 interval that did not induce tachycardia was the same as the AV interval at the longest A_1A_2 that did induce tachycardia. Therefore, in Patient 15, induction of orthodromic tachycardia clearly was dependent on a decrease in the degree of concealed conduction as the A_1A_2 interval was shortened.

In other patients who had a concealed accessory pathway, the induction of orthodromic tachycardia was not associated with any discernible change in concealed anterograde conduction, but was associated with an increase in the AV interval. In these patients, it appears that orthodromic tachycardia was induced when the AV interval became long enough to allow recovery from a given degree of concealed anterograde conduction.

Interplay between atrioventricular interval and concealed conduction. The critical interplay between the AV interval and the degree of concealed anterograde accessory pathway conduction during the induction of orthodromic tachycardia was further demonstrated by the response to changes in the A_1A_2 interval. In two patients, concealed conduction was manifest as retrograde accessory pathway block when V_3 was delivered 100 to 50 ms earlier than H_2. When the A_1A_2 interval then was shortened while maintaining a constant A_2V_3 interval, retrograde accessory pathway block after V_3 resolved, indicating a lesser degree of concealed anterograde conduction in the accessory pathway at the shorter A_1A_2 interval. Therefore, depending on the degree of concealed conduction into the accessory pathway, a particular AV interval may or may not provide sufficient time for recovery of retrograde conduction through the accessory pathway.

Conclusions. Anterograde concealed conduction into the accessory pathway occurs commonly at the time of induction of orthodromic tachycardia by an atrial premature depolarization. In patients who have an overt accessory pathway, there is often concealed anterograde conduction at A_1A_2 intervals shorter than the accessory pathway effective refractory period; however, the induction of orthodromic

tachycardia by an atrial premature depolarization usually is not limited by concealed anterograde conduction in the accessory pathway. In contrast, in patients who have a concealed accessory pathway, the degree of concealed conduction into the accessory pathway often may be one of the important factors limiting the induction of orthodromic tachycardia. In these patients, the initiation of orthodromic tachycardia more often requires a critical interplay between the degree of concealed anterograde accessory pathway conduction and the atrioventricular conduction delay after the atrial premature depolarization that induces the tachycardia.

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