

DIAGNOSTIC STUDIES

Intracardiac Electrode Catheter Recordings of Atrioventricular Bypass Tracts in Wolff-Parkinson-White Syndrome: Techniques, Electrophysiologic Characteristics and Demonstration of Concealed and Decremental Propagation

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Atrioventricular bypass tract deflections were recorded in five patients with the Wolff-Parkinson-White syndrome using standard, closely spaced (5 mm) electrode catheters. Three right paraseptal and two left-sided Kent bundles were recorded at the level of the tricuspid valve on the His bundle catheter and in the coronary sinus, respectively. Characteristics of the bypass tracts were studied during atrial pacing, programmed premature atrial stimulation, induction of supraventricular tachycardias and programmed ventricular stimulation. During atrial pacing, as pre-excitation increased, the stimulus to bypass tract deflection time remained unchanged. In five patients normalization of the QRS complex coincided with loss of the bypass tract deflection during incremental atrial pacing. Two patients demonstrated fragmentation of the bypass tract deflection before block. In one patient fragmentation of the bypass deflection coincided with normalization of the QRS complex. The effective refractory periods of the bypass tracts coincided with loss of bypass tract deflections in three of the five patients. In one patient, the effective refractory period of the bypass tract at its ventricular insertion preceded

that at its atrial insertion, whereas in the remaining patient, the effective refractory period of the bypass tract was not attained because of atrial refractoriness. During orthodromic supraventricular tachycardia, the bypass tract deflections disappeared in the anterograde limb in all patients. In one patient, the bypass tract deflection was recorded during atrial fibrillation with pre-excitation.

In conclusion: 1) Bypass tract deflections can be recorded with a closely spaced electrode catheter. 2) Right paraseptal bypass tracts are located close to the His bundle. 3) The anterograde effective refractory period of the bypass tract usually reflects its atrial insertion, but concealment through the bypass tract can occur with block at the ventricular insertion. 4) Decremental conduction within the bypass tract can occur before block, suggesting concealed and overt Wenckebach block within the bypass tract. 5) Recordings of bypass tract deflections increase the potential of closed chest ablation of right paraseptal and left-sided bypass tracts.

(J Am Coll Cardiol 1986;7:1392-403)

Various techniques exist to identify and localize anomalous atrioventricular (AV) connections responsible for the Wolff-Parkinson-White syndrome. These include electrocardiographic analysis (1), determination of intracardiac activation sequence (2) and intraoperative epicardial mapping (3). However, the localization of such bypass tracts has been

dependent on deductive analysis, rather than on recordings of the connections themselves.

Recently, electrode catheter recordings of AV bypass tract deflections have been reported in a few patients with the Wolff-Parkinson-White syndrome (4-6). With the exception of two case reports, preliminary studies reported by Jackman et al. (6) have stressed the use of an orthogonal electrode catheter to record bypass tract depolarizations. Utilizing standard quadripolar electrode catheters (USCI) with 5 mm interelectrode spacing, we were able to record bypass tract depolarizations in five patients with the Wolff-Parkinson-White syndrome, two of whom had left-sided and three of whom had right paraseptal AV connections. The electrophysiologic characteristics of these bypass tract depolarizations are reported.

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Manuscript received July 25, 1985; revised manuscript received December 17, 1985, accepted January 21, 1986.

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Methods

Study patients. Five symptomatic patients with the Wolff-Parkinson-White syndrome underwent electrophysiologic studies after informed consent was obtained. They all had experienced recurrent syncope or palpitation, or both. None of the patients had evidence of atherosclerotic or other forms of organic heart disease and none had electrolyte or metabolic imbalances. Cardiac medications were discontinued at least 48 hours before the procedures.

Electrophysiologic studies. All five patients had three or more quadripolar catheters (USCI), with 5 mm interelectrode spacing, inserted percutaneously and positioned fluoroscopically in the high right atrium, at the level of the tricuspid valve for recording of His bundle activity, in the right ventricular apex and in the coronary sinus, as required. In the three patients with electrocardiographic suggestion of a right paraseptal AV bypass tract, the high, mid and low right atrium were mapped along the lateral, septal, posterior and anterior endocardial surfaces. In the other two patients with electrocardiographic evidence of a left-sided bypass tract, the length of the coronary sinus was systematically mapped. In one patient, systematic mapping of the coronary sinus was also done with an orthogonal electrode catheter (Webster Laboratories Inc).

During pacing studies, the proximal poles of the electrode catheter were used for recording and the distal poles for stimulation. Three standard electrocardiographic leads (I, II, V₁) as well as multiple intracardiac electrograms at frequency settings of 30 to 500 Hz and time lines generated at 40, 200 and 1,000 ms were displayed on a multichannel oscilloscope (VR-12, Electronics for Medicine) and reproduced on thermal paper at a speed of 100 to 150 mm/s. Incremental atrial and ventricular pacing was performed up to the onset of anterograde and retrograde block. Standard atrial and ventricular extrastimulus techniques were employed to determine the refractory periods of the AV node, AV bypass connections and ventricular muscle. Induction of supraventricular tachycardias was attempted with rapid atrial pacing and introduction of atrial as well as ventricular premature stimuli during a fixed basic drive.

Definition of terms. *Anterograde conduction intervals and refractory periods* were defined as previously described (7). The A-k interval was measured from the onset of the atrial (A) deflection (on the bipolar recording in which the bypass deflection was seen) to the onset of the bypass tract deflection (k). The S-k interval was measured from the atrial stimulus artifact (S) to the onset of the rapid deflection of the AV bypass tract (k). The k-V interval was measured from the recording of the rapid deflection of the AV bypass tract (k) to the onset of the delta wave (V) on the surface electrogram. In the results described later, only those findings pertinent to recordings of bypass tract deflections and their electrophysiologic characteristics will be described in detail.

Results

High frequency deflections (k) were recorded in the first three patients on the His bundle catheter at the level of the tricuspid valve. These deflections disappeared with normalization of the QRS complexes and probably reflected bypass tract deflections of a right paraseptal Kent bundle. In the other two patients, bypass tract deflections were recorded in the distal coronary sinus. The specific findings in all five patients are discussed later. The anterograde conduction intervals and refractory periods are summarized in Table 1.

Case Reports

Case 1

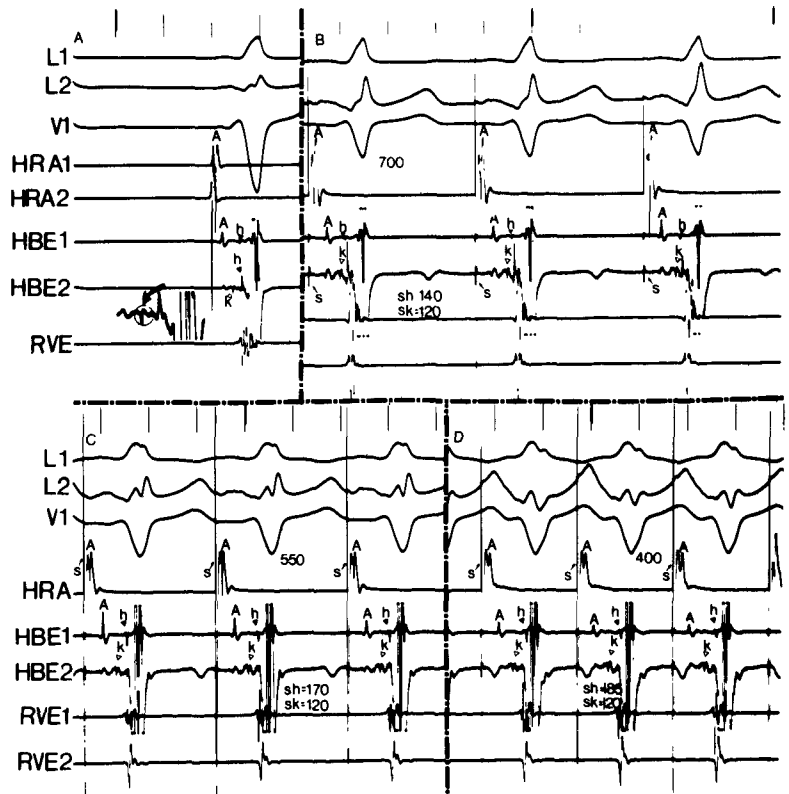
The patient was a 36 year old man who had experienced several syncopal episodes and whose electrocardiogram revealed a pre-excitation pattern consistent with a right posteroseptal AV bypass tract. During normal sinus rhythm, a high frequency, low amplitude deflection (k) was consistently recorded on the distal poles of the His bundle catheter and occurred before the His bundle deflection (Fig. 1A). During incremental atrial pacing (Fig. 1B to D) at cycle lengths of 700, 550 and 400 ms, respectively, the bypass

Table 1. Anterograde Conduction Intervals and Refractory Periods in Five Cases

Case	Location of AVBT	A-k Interval (ms)	k-V Interval (ms)	AVN-ERP (ms)	BT-ERP (ms) Defined	
					By Norm. of QRS	By Loss of k
1	RPS	40	20	NA	320	320
2	RPS	115	15	NA	300	300
3	RPS	55	45	320	320	280
4	L	60	15	NA	300	300
5	L	47	20	NA	NA	NA

AVBT = atrioventricular (AV) bypass tract; AVN-ERP = effective refractory period of the AV node; BT-ERP = effective refractory period of the bypass tract, k = bypass tract deflection; L = left-sided; NA = not attained; Norm. of QRS = normalization of QRS complex; RPS = right paraseptal.

Figure 1. Case 1. Recordings during normal sinus rhythm and incremental atrial pacing. From **top to bottom**, recordings represent electrographic leads I, II and V₁ (L₁, L₂, V₁), as well as intracardiac recordings from one or two sites in the high right atrium (HRA), proximal and distal His bundle electrograms (HBE₁, HBE₂) and one or two sites at the right ventricular apex (RVE). **A** highlights a recording during normal sinus rhythm. An enlargement of the k deflection is designated in the lower portion of this panel with a **curved arrow**. **B to D** reveal maintenance of the S-k interval at 120 ms, despite an increase in the S-h interval during incremental atrial pacing and increasing pre-excitation. See text for further details. A = atrial electrogram; h = His bundle electrogram; k = atrioventricular bypass tract deflection; s = atrial stimulus artifact.

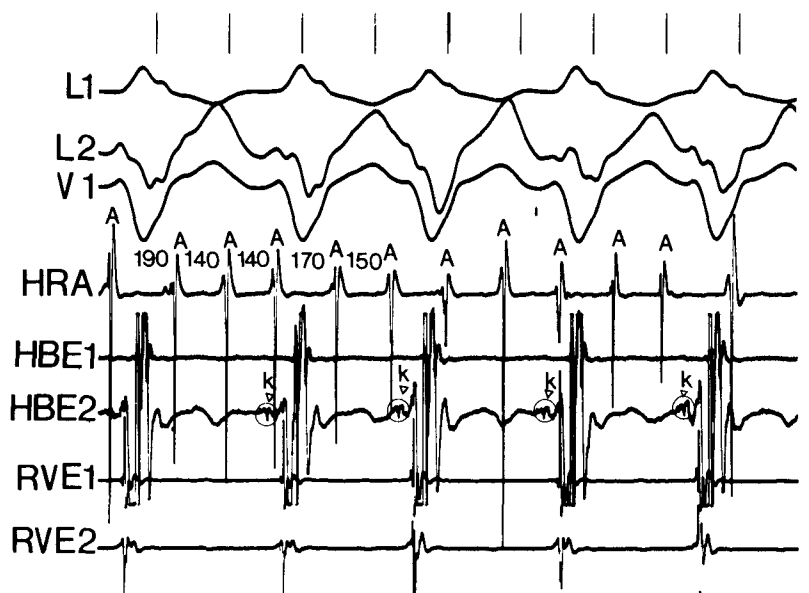


tract deflection (k) remained interposed between the atrial and ventricular deflections. Simultaneously, the His bundle deflection (h) moved further away from the atrial deflection and into the ventricular complex with increasing pre-excitation. Note that as the pacing cycle lengths decreased, the S-h intervals increased, whereas the S-k intervals and the k-V intervals remained constant. During atrial fibrillation, associated with a wide QRS complex pattern (Fig. 2), the

k deflection was consistently noted before the onset of the QRS complex.

Introduction of programmed premature atrial stimuli resulted in increasing ventricular pre-excitation with loss of the His bundle depolarization (h) within the ventricular complex, but persistence of the k deflection before the QRS complex (Fig. 3A). The effective refractory period of the bypass tract (that is, normalization of the QRS complex)

Figure 2. Case 1. Recordings during atrial fibrillation associated with a wide QRS complex. The atrioventricular bypass tract deflection (k) is noted before each ventricular complex. Other abbreviations as in Figure 1.



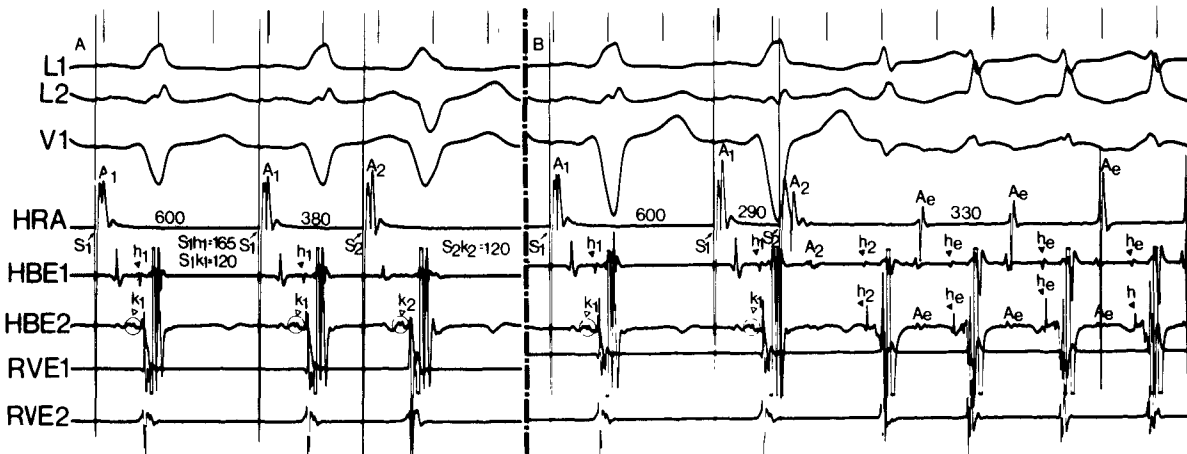


Figure 3. Case 1. Recordings during introduction of programmed premature atrial stimuli. In A, the k deflection remains interposed between A and the ventricular complex, with the S_2k_2 interval equal to the S_1k_1 interval. In B, at a critical coupling interval (290 ms), induction of a sustained orthodromic tachycardia is associated with loss of recording of the k deflection and normalization of the QRS complex. See text for discussion. Abbreviations as in Figure 1.

was 320 ms and was associated with loss of the k deflection. Introduction of a premature atrial stimulus at a coupling interval of 290 ms (Fig. 3B) resulted in normalization of the QRS complex in association with loss of the k deflection and induction of a sustained orthodromic tachycardia.

During programmed ventricular stimulation, a k deflection was not identified at long coupling intervals and was probably obscured in the ventricular complex. As the S_2A_2 interval increased, a k deflection was recorded between the ventricular and atrial complexes. The retrograde k-A interval of 30 ms was similar to the anterograde A-k interval. The retrograde effective refractory period of the AV bypass tract was not obtained in this patient because the effective refractory period of the ventricular myocardium was achieved at an S_1S_2 interval which preceded that of the bypass tract.

Case 2

A 57 year old woman with recurrent palpitation and syncope in association with the Wolff-Parkinson-White syndrome was referred for electrophysiologic evaluation. Analysis of the early ventricular forces suggested a right paraseptal location of the AV bypass connection. During sinus rhythm (Fig. 4), a high frequency, low amplitude deflection (k) was consistently recorded between the His bundle (h) and ventricular deflections on the distal poles of the His bundle catheter (Fig. 4A). Slight advancement of the His bundle catheter (Fig. 4B) resulted in recording of the k deflection on the proximal as well as the distal poles, and clear separation of the k deflection from the His bundle recording

(h). In addition, it was possible to record the same deflection by repositioning the right ventricular catheter in the same position (HBE₃ and HBE₄) as the original His bundle catheter (HBE₁ and HBE₂) (Fig. 4B). The occurrence of two spontaneous sinus echo beats (Fig. 4A) was associated with total ventricular pre-excitation and loss of His bundle deflections but preservation of the baseline A-k interval. Because the k deflection became dissociated from the His deflection (h), it is unlikely that the former could reflect a bundle branch depolarization. The effective refractory period of the AV bypass tract (k) was 300 ms. This was associated with loss of the k deflection, normalization of the QRS complex and induction of an orthodromic tachycardia with a cycle length of 310 ms. No k deflection was visualized during the tachycardia.

During programmed ventricular stimulation, a distinct, low amplitude deflection was noted at the end of the ventricular deflection. The interval from this deflection to the atrial deflection (k-A) was similar to that of the anterograde A-k interval. The retrograde effective refractory period of the bypass tract was 330 ms and was associated with loss of retrograde k deflections.

Case 3

This patient was a 30 year old man with a history of supraventricular tachycardia and near syncope. The surface electrogram suggested the presence of the Wolff-Parkinson-White syndrome with a right paraseptal AV bypass connection.

Recordings of the AV bypass tract (k) and its characteristics are demonstrated in Figure 5. Figure 5A shows a low amplitude deflection (k) recorded before the His deflection (h) in association with pre-excitation during sinus rhythm. The A-k interval was 55 ms and the k-V interval was 45 ms on the proximal poles of the His bundle catheter. Figure 5B demonstrates that atrial pacing up to a cycle length of 350 ms resulted in prolongation of the S-h interval but not of the S-k interval. Rapid atrial pacing at a cycle length of

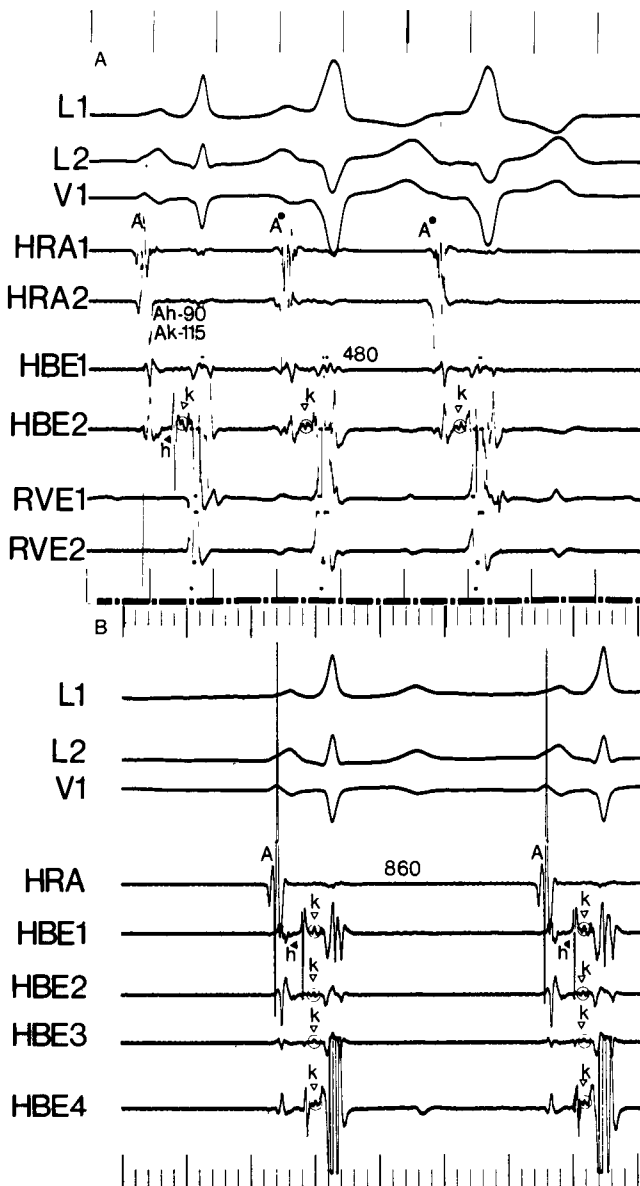


Figure 4. Case 2. In A, tracings during sinus rhythm show an initial sinus beat followed by two sinus echo beats (A●). During these latter two beats, the His bundle deflection (h) moves into the ventricular complex; however, the bypass tract recordings (k) remain stationary and precede the ventricular complexes. In B, two more distal His bundle sites are recorded (HBE₃, HBE₄) and clear separation of k and the ventricular complex during sinus rhythm is demonstrated. See text for details. Other abbreviations as in Figure 1.

320 ms (Fig. 5C) was associated with block within the bypass tract and 2:1 block at the level of the AV node. Note that normalization of the QRS complex was associated with loss of the k deflection (Fig. 5C).

Figure 6 shows introduction of premature atrial stimuli during a basic drive cycle length of 600 ms, at progressively shorter coupling intervals. At an interval of 320 ms (Fig. 6C), ventricular and His bundle deflections (h) were no

longer detected; however, k deflections were still recorded. Shortening of the coupling interval to 280 ms resulted in loss of k deflections (Fig. 6D). This suggests that block occurred distally in the bypass tract before reaching the effective refractory period at its atrial insertion.

During ventricular pacing, an orthodromic tachycardia with a cycle length of 400 ms, associated with normalization of the QRS complexes, was initiated. However, a retrograde k deflection was identified before recording of the atrial deflection (Fig. 7). Ventricular pacing was associated with recording of the k deflection after the ventricular complex, but before the atrial complex (Fig. 8). The effective refractory period of the retrograde AV bypass tract and the AV node was 350 ms. At this coupling interval, recording of the k deflection as well as atrial activity was lost.

Case 4

The patient was a 30 year old woman with a history of recurrent palpitation and syncope. The electrocardiogram at rest revealed the Wolff-Parkinson-White syndrome with a suggestion of a left-sided AV bypass tract. Mapping of the coronary sinus (Fig. 9A and B) revealed a distinct high amplitude deflection (k) interposed between the atrial and ventricular deflections in the distal coronary sinus. The A-k interval was 60 ms and the k-V interval measured 15 ms. Incremental atrial pacing resulted in movement of the His bundle deflection (h) into the ventricular complex and maintenance of a constant S-k interval (Fig. 10). At a pacing cycle length of 360 ms (Fig. 11), varying degrees of pre-excitation and normalization of the QRS complexes occurred. The second and third beats in Figure 11 demonstrate maximal pre-excitation with the S-k interval maintained at 120 ms. In the fourth and fifth paced beats the delta waves were lost and normalization of the QRS complexes occurred. This was associated with fragmentation and diminution in amplitude of the bypass tract deflections (d-k), as further highlighted in the insert above the electrogram in Figure 11. These deflections were subsequently lost. Further atrial pacing at a cycle length of 320 ms resulted in a sustained orthodromic tachycardia without visualization of bypass tract deflections.

Introduction of premature atrial stimuli resulted in lengthening of the S-h interval but no change in the S-k interval. Figure 12A shows that the S-k interval remained 120 ms and the k-V interval remained 15 ms at an S₁S₂ cycle length of 310 ms; however, the His bundle deflection (h) disappeared into the ventricular complex as the degree of pre-excitation increased. The effective refractory period of the AV node was limited by atrial refractoriness, and that of the AV bypass tract was 300 ms. Figure 12B demonstrates that at a coupling interval of 290 ms, atrial echo beats (A_e) were induced and revealed early left atrial activation, thus confirming retrograde conduction across a left-sided AV

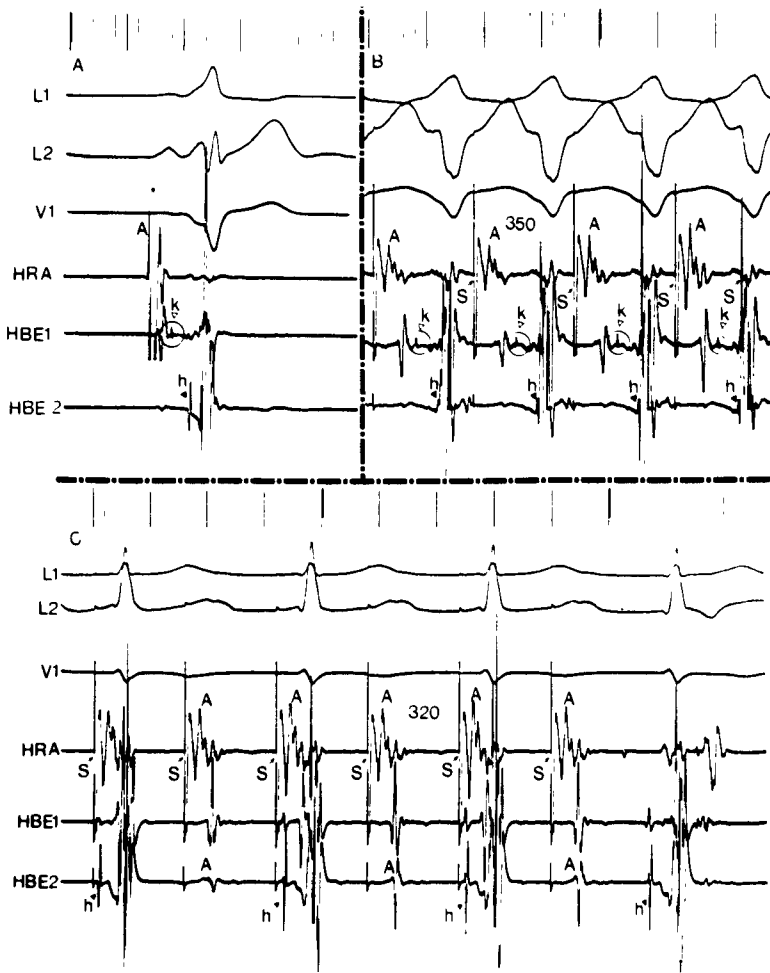
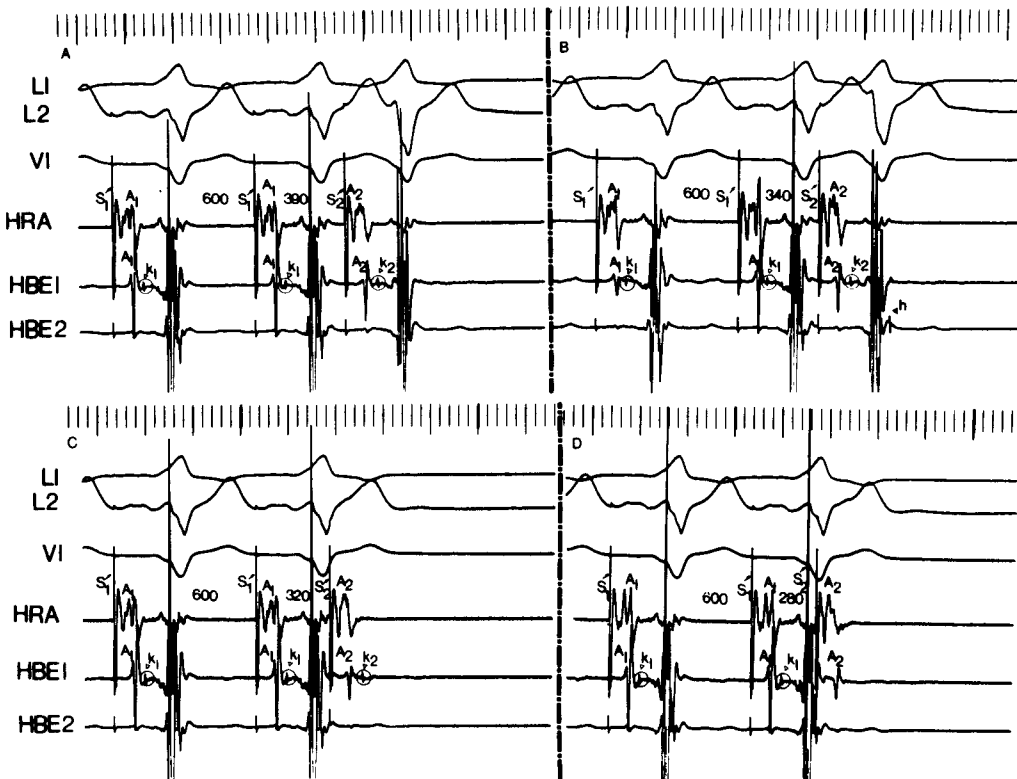


Figure 5. Case 3. Recordings of atrioventricular bypass tracts (k) and their characteristics during sinus rhythm (A), as well as during atrial pacing at cycle lengths of 350 ms and 320 ms (B and C). The A-k interval remains unchanged despite incremental pacing and the k deflection precedes the ventricular complex despite increasing pre-excitation (B). At a cycle length of 320 ms (C) block occurred within the bypass tract and k was no longer recorded; however, 2:1 block at the level of the AV node was noted. See text for further discussion. Other abbreviations as in Figure 1.

Figure 6. Case 3. Introduction of premature atrial stimuli at closer coupling intervals. A and B demonstrate a fixed A_2k_2 interval. In C, at an S_1S_2 interval of 320 ms, the atrioventricular bypass tract is still recorded (k_2); however, ventricular activation is lost. Further

decrease in the S_1S_2 interval to 280 ms (D) is associated with loss of the bypass tract recording. Thus, concealed conduction within the bypass tract is suggested. See text for further discussion. Abbreviations as in Figure 1.



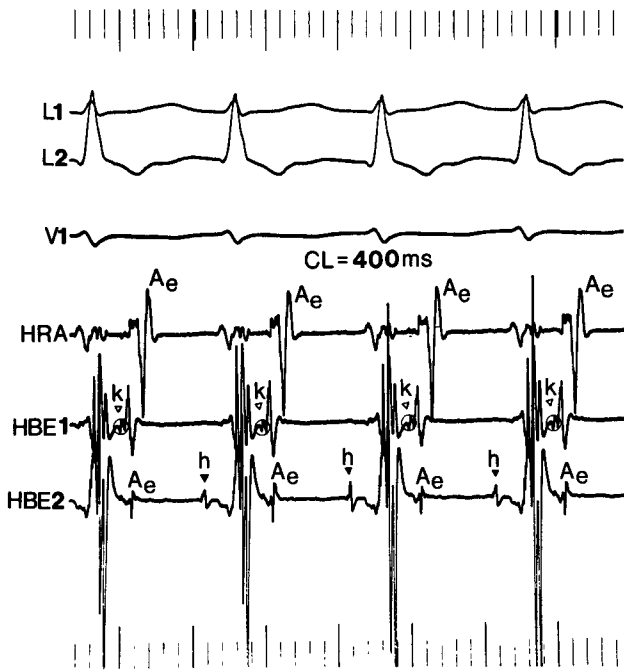


Figure 7. Case 3. Orthodromic tachycardia demonstrating bypass tract deflection in the retrograde limb. From **top** to **bottom** are electrocardiographic leads I, II and V₁ followed by intracardiac recordings from the same locations as in Figure 6. There is a clear k deflection (**arrowhead**) interposed between each ventricular and atrial echo (A_e) beat. His bundle deflections (h) follow the atrial depolarizations. CL = cycle length. Other abbreviations as in Figure 1.

Figure 8. Case 3. Ventricular pacing at a cycle length of 600 ms (recordings as in Figure 7). Whereas the His bundle deflection (h₁) appears before the ventricular complex on the HBE₂ recording, distinct k deflections follow the ventricular electrograms on HBE₁. The atrial depolarization (A₁) most likely occurs through retrograde bypass tract activation. Abbreviations as in Figure 1.

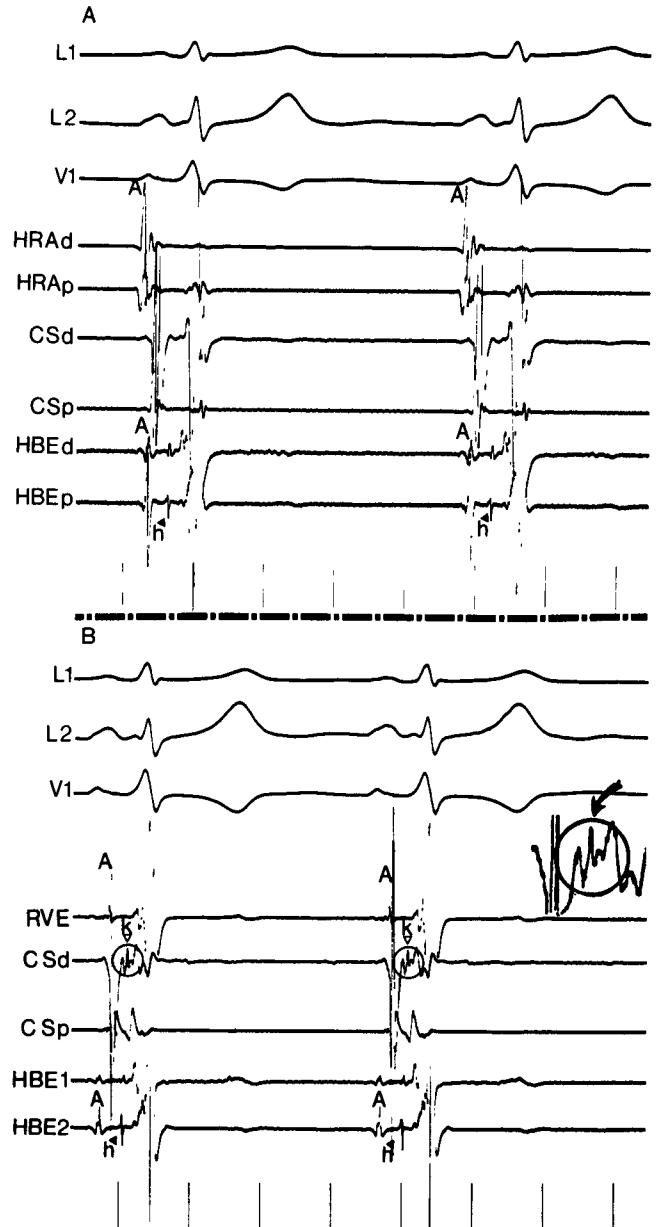
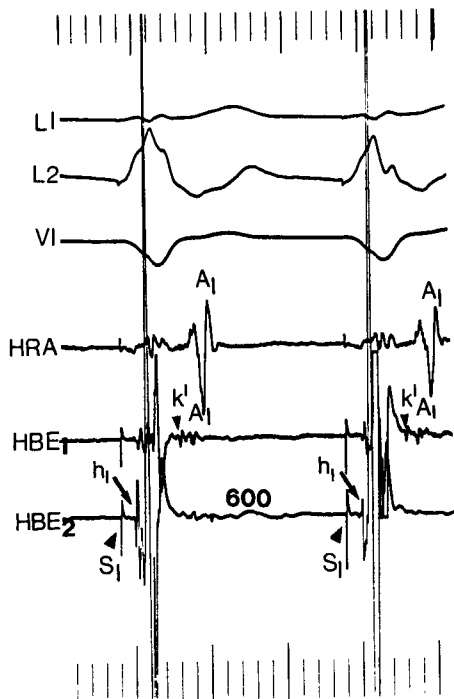


Figure 9. Case 4. Intracardiac recordings of a left-sided atrioventricular bypass tract. HRAd and HRAP refer, respectively, to the distal and proximal poles of the catheter situated within the high right atrium and CSd and CSp refer to the distal and proximal sites of recording within the coronary sinus. HBE₁ and HBE_d refer to distal and HBE₂ and HBE_p to proximal sites of recording from the His bundle catheter. Whereas no bypass tract deflection is noted in **A**, it becomes visible in **B** as the coronary sinus catheter is advanced deeper within the coronary sinus. This k deflection (**arrow**) is enlarged in the insert shown in the **right** portion of **B**. Other abbreviations as in Figure 1.

bypass connection. A retrograde bypass tract deflection was not recorded and was probably obscured in the ventricular complexes.

Case 5

The patient was a 26 year old man with a history of recurrent palpitation associated with presyncopal attacks.

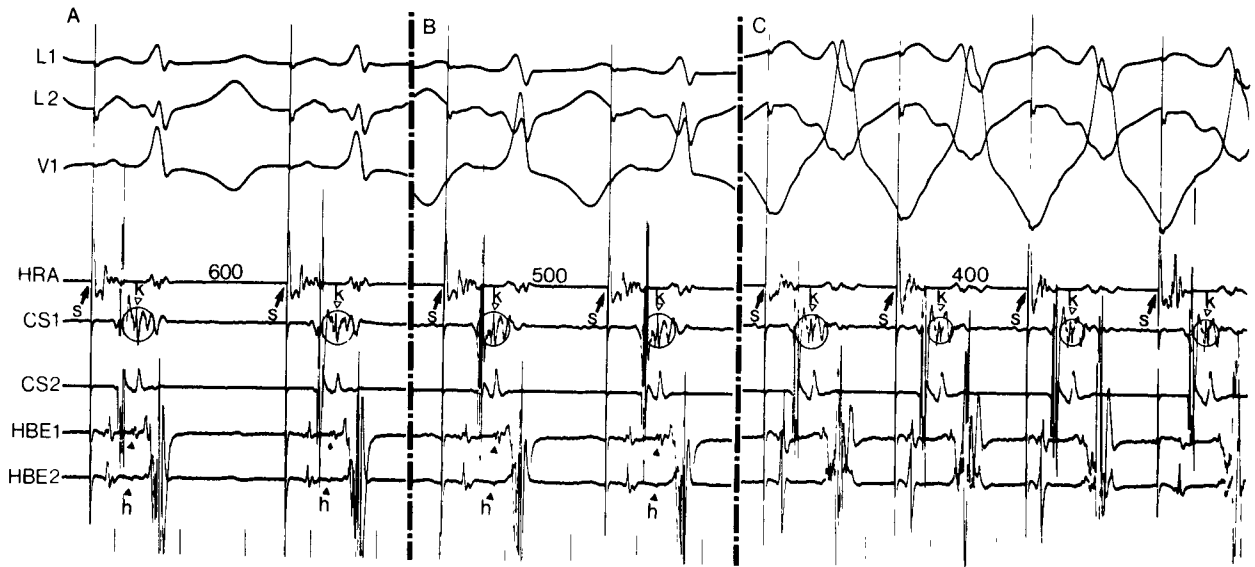
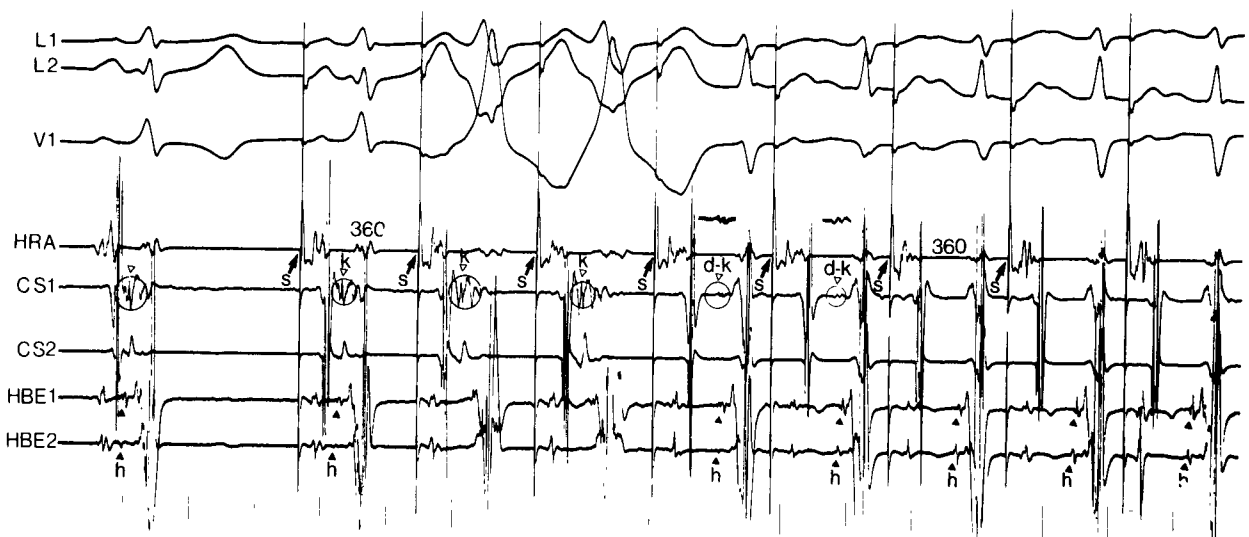


Figure 10. Case 4. Recordings of incremental atrial pacing. CS₁ and CS₂ refer to the distal and proximal poles of the coronary sinus catheter, respectively; HBE₁ and HBE₂ similarly refer to the distal and proximal poles of the His bundle catheter. A to C demonstrate a stable A-k interval despite increasing degrees of pre-excitation at shorter cycle lengths. Other abbreviations as in Figure 1.

Physical examination was unremarkable and the electrocardiogram was suggestive of the Wolff-Parkinson-White syndrome with a left-sided AV bypass tract. During normal sinus rhythm, a high frequency deflection, interposed between the atrial and ventricular electrograms, was recorded from the distal poles of a standard quadripolar electrode

Figure 11. Case 4. Rapid atrial pacing at a cycle length of 360 ms results in decremental propagation within the bypass tract (d-k), an enlarged recording of which is shown above the HRA recording after the fourth and fifth stimuli. See text for further discussion. Abbreviations as in Figures 1 and 10.



catheter with 5 mm interelectrode spacing positioned in the distal portion of the coronary sinus. Pacing from this site resulted in maximal pre-excitation. Furthermore, this deflection was also recorded when the standard catheter was replaced with an orthogonal electrode catheter (Webster Laboratories Inc.) (Fig. 13).

During incremental atrial pacing, the k deflection remained fixed in its location, although the His bundle recording progressed into the ventricular electrogram in association with increasing degrees of pre-excitation. At a coupling interval of 270 ms, the A-k interval gradually lengthened in association with diminution of the amplitude of the k deflection before block within the bypass tract (Fig. 14). Repeated pacing at this cycle length resulted in induction of orthodromic supraventricular tachycardia. During the tachycardia, k deflections were not seen in the anterograde or retrograde limbs. The k deflections were most likely obscured in the ventricular deflections. Introduction of atrial

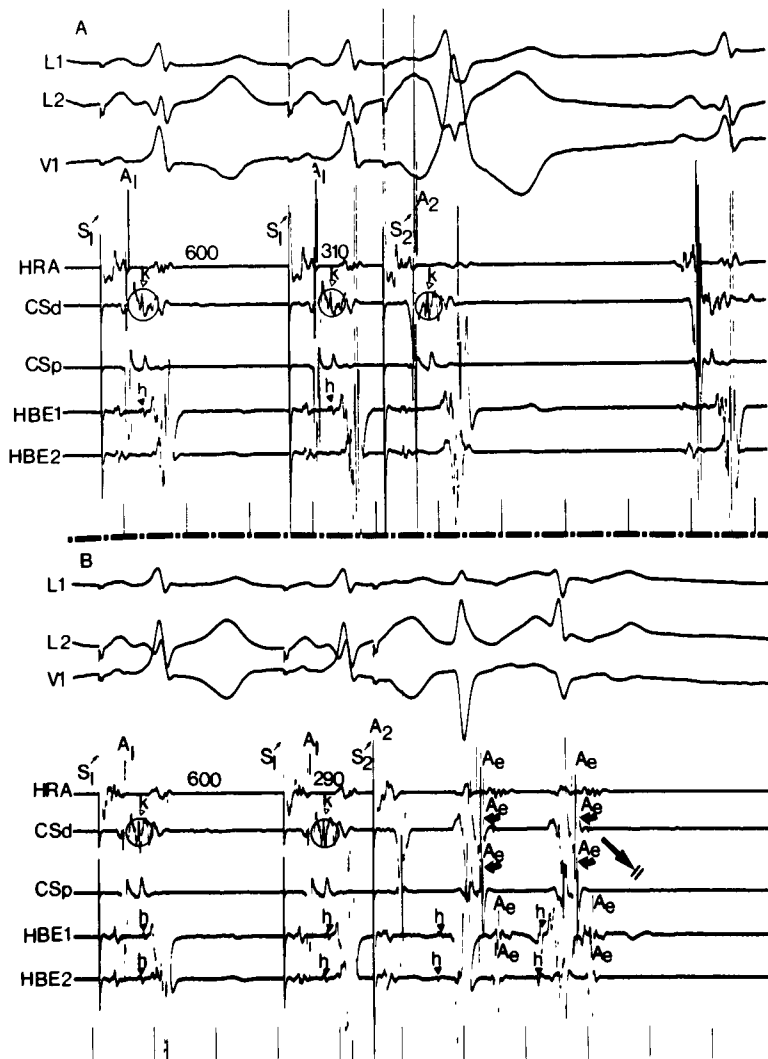


Figure 12. Case 4. Introduction of programmed premature atrial stimuli. Whereas the A₁-k interval and the A₂-k interval remain stable in **A**, a premature stimulus at a coupling interval of 290 ms results in two atrial echo beats (A_e) in **B**. Thus, early left atrial activation is confirmed in **B** and the retrograde bypass tract deflection was probably obscured within the ventricular complexes. See text for details. Abbreviations as in Figures 1 and 9.

premature stimuli resulted in lengthening of the S-h interval but maintenance of a constant S-k interval. Achievement of the effective refractory periods of the AV node and the bypass tract was limited by refractoriness of the atrium.

During ventricular pacing, retrograde atrial activity at the site of bypass recording (in the coronary sinus) was recorded very close to the ventricular electrograms. Whereas the bypass tract deflection was most likely obscured within the ventricular electrogram at a pacing cycle length of 250 ms (Fig. 15), a distinct k deflection became apparent as the SA interval increased. Retrograde conduction block occurred between the retrograde bypass deflection and atrial activity. The fourth beat was associated with VA conduction and the k deflection was most likely obscured within the ventricular electrogram.

Discussion

Localization of AV bypass tracts. Until now the following techniques have been utilized to localize the site of AV bypass tracts: electrocardiographic analysis (1), intra-

cardiac electrophysiologic studies (2) and intraoperative mapping (3). Gallagher et al. (1) correlated the initial delta vector with intraoperative mapping findings and established criteria for electrocardiographic localization of AV bypass tracts. However, electrographic analysis can be inaccurate in the setting of prior myocardial infarction, drug therapy, electrolyte imbalance or intraventricular conduction disturbances (8). Intracardiac electrophysiologic studies for recognition and localization of AV bypass tracts are based on deductive analysis (9) which includes: 1) association of onset of the delta wave with the earliest site of endocardial activation; 2) pacing from the atrial location that results in the greatest degree of pre-excitation; and 3) atrial mapping for the earliest site of activation during retrograde ventricular atrial conduction and orthodromic tachycardia. Furthermore, prolongation of the tachycardia cycle length and the VA_e interval during supraventricular tachycardia can aid in localization of a concealed or overt bypass tract ipsilateral to the site of bundle branch block. As the five cases described illustrate, direct recordings of these bypass connections are possible with closely spaced (5 mm) electrode

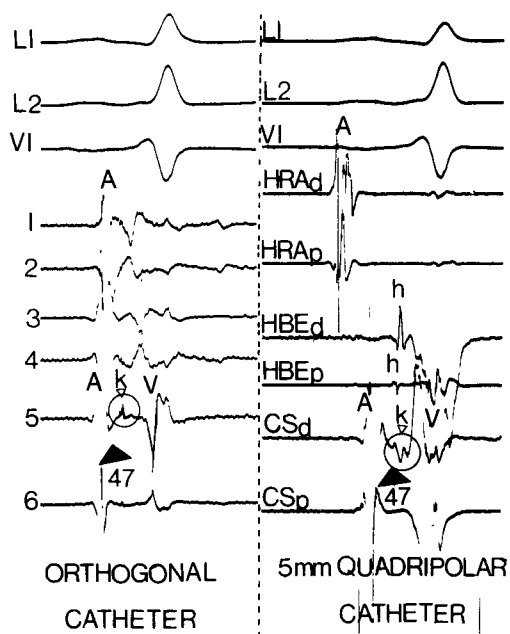


Figure 13. Case 5. Sinus beat recorded using a quadripolar catheter with 5 mm interelectrode spacing (right) and a special orthogonal catheter (left). Left, Six bipolar recordings (1 to 6) from the orthogonal catheter are shown. Right, Recordings from distal and proximal poles of the high right atrium (HRAd, HRAp), distal and proximal poles of the His bundle catheter (HBE d, HBE p) along with distal and proximal poles of the coronary sinus (CSd, CSp) are shown (paper speed = 150 mm/s). The a-k interval (47 ms) (arrowhead) was the same in both methods of recording. Other abbreviations as in Figure 1.

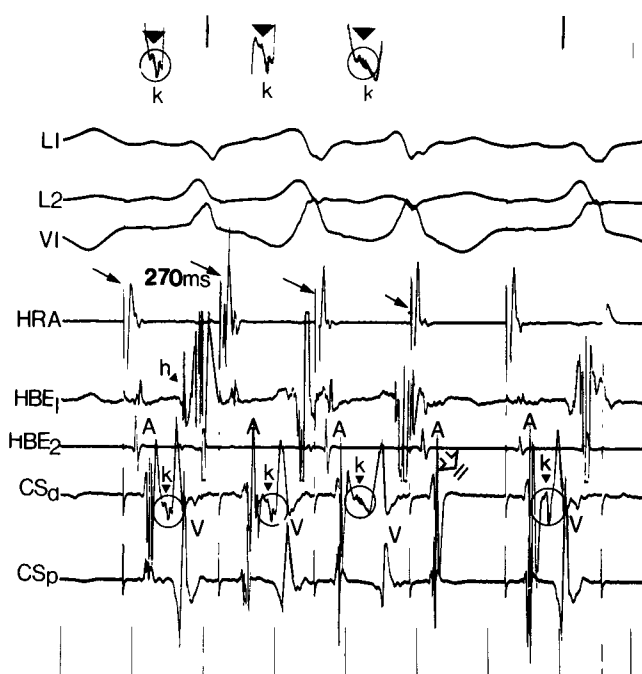


Figure 14. Case 5. Atrial pacing at a cycle length of 270 ms (recordings as in Figure 9). The first three paced beats result in prolongation of the s-k intervals and diminution in the amplitude of the k deflections as recorded on the CSd channel (see enlargement at top of figure). The fourth paced beat blocks at the proximal end of the bypass tract; thus, no k deflection or ventriculogram is recorded. This most likely represents overt Wenckebach block in the bypass tract. See text for further details. Abbreviations as in Figure 1 and 9.

catheters. The deflections recorded are of high frequency, precede the onset of the delta wave and disappear with normalization of the QRS complex.

Identification of the AV bypass tract deflections. The following points are presented to support that these k deflections indeed represent AV bypass connections: 1) The deflections preceded the delta wave on surface electrographic leads and disappeared with normalization of the QRS complex. 2) As the His bundle deflection moved into the ventricular complex with incremental atrial pacing or introduction of premature atrial stimuli at closer coupling intervals, the k deflections remained fixed in their location. Similar observations were made in two case reports of patients with an AV bypass tract (5,6). 3) During orthodromic tachycardia the k deflections were not recorded in the anterograde limb in any of the patients. 4) In the one patient who developed atrial fibrillation with a wide QRS tachycardia, the k deflections were still present before ventricular depolarization.

Further support can be gained through exclusion of other causes of the k deflections. First, these complexes are unlikely to represent split His bundle potentials as there was clear dissociation of the two deflections and only the His deflection (h) progressed through the ventricular recordings. Second, these deflections are unlikely to represent atrial

activity for the following reasons: 1) They were seen on the His bundle or the coronary sinus catheters corresponding to the electrocardiographically suggested locations and not in other regions of the right atrium. 2) They were seen during sinus rhythm with varying degrees of pre-excitation before recording of the ventricular electrogram. 3) They followed recordings of ventricular activity during ventricular pacing in those patients in whom retrograde k deflections could be identified. 4) They disappeared during atrial pacing and premature atrial stimulation in association with normalization of the QRS complex. 5) The a-k and k-a intervals were similar when the retrograde intervals could be measured. Furthermore, if these were atrial depolarizations, the k deflections should have become more fragmented and delayed with closely coupled premature atrial stimulation. Finally, block of the retrograde k deflections in three patients was associated with loss of retrograde activity to the atrium.

As a third point, these deflections cannot represent right bundle branch depolarizations because in four of the patients they occurred before the His bundle deflection during normal sinus rhythm. Fourth, during incremental atrial pacing in these patients, the S-k intervals remained constant, while the His deflections (h) moved into the ventricular complexes. Furthermore, the appearance of the k deflections

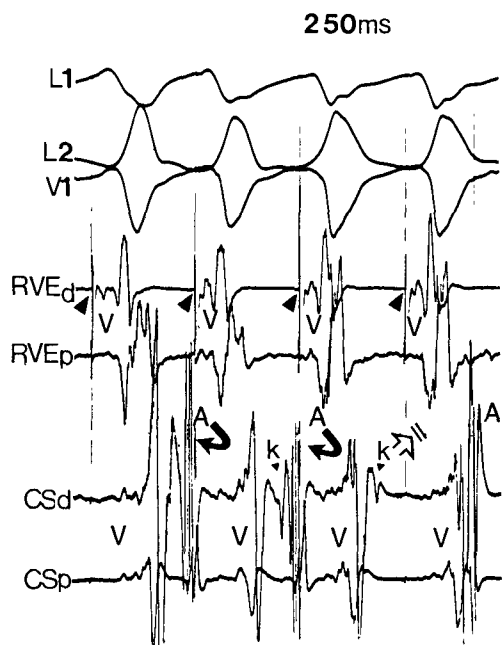


Figure 15. Case 5. Demonstration of retrograde bypass tract (k) recordings. RVEd and RVEp refer to recordings from the distal and proximal poles of the right ventricular catheter, respectively. Whereas a k deflection is distinctly recorded in the coronary sinus recordings after the second and third paced ventricular beats, there is no conduction to the atrium after the second k deflection. Thus block has occurred between the bypass tract recording (k) and the atrium (A). After the fourth ventricular beat, conduction to the atrium resumes and the k deflection is most likely obscured in the ventriculogram. Other abbreviations as in Figure 9.

before the ventricular electrograms on anterograde stimulation and after the ventricular electrogram in four of the patients during ventricular stimulation suggests that the recordings are neither of ventricular nor of atrial activity. Thus, the k deflections recorded represent AV connections responsible for pre-excitation in the Wolff-Parkinson-White syndrome.

Previous reports. It is unclear why AV bypass tracts have not been recorded until recently. Perhaps this is because of utilization of catheters with interelectrode distances of 10 mm or more in the past during intracardiac studies in patients with the Wolff-Parkinson-White syndrome. However, there have been two case reports (4,5) of intracardiac recordings of AV bypass tracts with the use of standard electrode catheters. In one, the patient had Ebstein's anomaly and the authors (4) suggest that recordings may have been facilitated by the unusually low amplitudes of the adjacent atrial and ventricular potentials due to the associated thin, fibrotic atrial and ventricular walls. In this case, the interelectrode spacing was 10 mm; however, the interelectrode spacing is not mentioned in the other case report (5). In this regard none of our patients had cardiomegaly or Ebstein's anomaly. Thus, the close interelectrode spacing used (5 mm) may have been responsible for our successful recordings. To our knowledge, recordings of left-sided AV

bypass tracts within the coronary sinus have not been reported with the use of a standard, closely spaced electrode catheter.

Effective refractory period of bypass tract. Normalization of the QRS complex associated with loss of the delta wave during atrial premature stimulation and incremental atrial pacing has been taken to reflect the effective refractory period of the AV bypass tract and to reflect block of conduction within the bypass tract. However, it has remained unclear whether the effective refractory period or conduction block or both, occur at the atrial or the ventricular end of the bypass tract insertion. The anterograde refractory period of the bypass tract (that is, normalization of the QRS complex and loss of the delta wave) in Cases 1, 2, 4 and 5 occurred in association with loss of recordings of the k deflection suggestive of block at its atrial insertion. In contrast, during atrial premature stimulation in Case 3, normalization of the QRS complex, associated with loss of the delta wave, and persistent recording of the k deflection occurred at a coupling interval of 320 ms, suggesting initial block of the bypass tract distal to the atrial insertion or at the ventricular insertion. However, at a coupling interval of 280 ms, the k deflection was lost, implying block of the bypass tract at the atrial insertion. These findings suggest that the effective refractory period of the bypass tract at the ventricular insertion (320 ms) may precede the effective refractory period at the atrial insertion (280 ms).

Mechanism of block in the bypass tract. In an elegant series of experiments, de la Fuente et al. (10) proposed a model to explain pre-excitation. They adjoined two segments of canine myocardium with a narrow isthmus of atrial muscle. When the proximal segment was electrically stimulated in the presence of an elevated potassium concentration, conduction across the narrow strip was recorded, but transmission of action potentials ceased at its junction with the distal myocardial segment. Thus, the wave front was of an inadequate degree to excite the distal segment. They suggested that the abrupt insertion of the isthmus into the relatively large, geometric mass represented a low "margin of safety" for conductivity. In other words, if the voltage and slew rate of the action potential traversing the isthmus as well as the extent of "excitability and conductivity" at its insertion were low, an "impedance mismatch" would develop and lead to failure of propagation. According to this theory, block would be expected to occur at the site of ventricular insertion rather than at the site of atrial insertion of an AV bypass tract. However, the findings in the majority of our patients (Cases 1, 2, 4 and 5) argue against this theory because block occurred at the atrial insertion of the bypass tract. Only Case 3 would support the theory of "impedance mismatch." Thus, our findings suggest that block of the bypass tract in the Wolff-Parkinson-White syndrome may occur at either the atrial or ventricular insertion. In addition, our findings in Case 3 suggest that concealment in the bypass tract can be demonstrated directly and that

block may occur distally in the bypass tract before achievement of the effective refractory period at its atrial insertion. This might be one mechanism of concealed AV bypass tract conduction.

Our observations during atrial pacing in Cases 4 and 5 are of considerable interest. In Case 4, at a pacing cycle length of 320 ms, the QRS complexes normalized; however, the k deflections were still evident but were fragmented for two beats before the occurrence of block. In Case 5, there was prolongation of the S-k interval along with fragmentation of the k deflection during maximal pre-excitation before block in the bypass tract. Block of the bypass tract was associated with loss of k deflections. This suggests that decremental conduction occurs before block at the atrial insertion of the bypass tract. To our knowledge these are the first direct demonstrations of concealed as well as overt Wenckebach block in AV bypass tracts.

Clinical implications. Direct recordings of AV bypass tracts during electrophysiologic studies are possible with standard, closely spaced electrode catheters. In addition to confirming the presence and location of such pathways, direct recordings may have therapeutic implications. Much interest has developed in the area of catheter fulguration to ablate AV bypass tracts (11,12). However, blind attempts at ablation through the coronary sinus in patients with left-sided bypass tracts have not yielded optimal results (11). Thus attempts at electrode catheter ablation guided by direct recordings of bypass tract deflections may improve the success rate. Our findings suggest that right paraseptal bypass tracts are located in close proximity to the bundle of His, as the former were recorded on the His bundle catheter. Thus it is possible that attempted ablation at this site may also result in disturbance of normal His bundle activity. In fact, attempts at surgical ablation of posteroseptal bypass tracts are associated with a 10% risk of complete AV block (13). Electrode catheter ablation of posteroseptal bypass tracts has also been associated with transient injury to His bundle conduction (12). Therefore, a temporary pacemaker should be in place before catheter ablation of the right paraseptal AV bypass tract is attempted.

Addendum

After submission of this manuscript for publication, a case of a right-sided retrograde bypass tract was recorded by O'Callaghan et al. (14). Interestingly, the deflection was

identified on the atrial electrode catheter, which was positioned close to the bypass tract's insertion.

We acknowledge Maxine Wright for secretarial assistance, Philip Barreca, BS, MTA for technical assistance and Valentin Fuster, MD, FACC for encouragement and support

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