Electrophysiological Evaluation of the Wolff-Parkinson-White Syndrome

Problems in Assessing Antegrade and Retrograde Conduction over the Accessory Pathway

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SUMMARY
The effect of atrial pacing and recording site and ventricular pacing site on assessment of conduction over the accessory pathway (AP) was examined in a group of patients with the Wolff-Parkinson-White syndrome. The importance of initial localization of the AP by recording the sequence of retrograde atrial activation during circus movement tachycardia is demonstrated. Inability to record or pace near the AP may lead to significant errors in the assessment of the antegrade and retrograde conduction properties of the AP. During ventricular pacing, retrograde atrial fusion was consistently demonstrated with laterally located APs.

THE PRESENCE OF AN ACCESSORY ATRIO-VENTRICULAR (A-V) PATHWAY (AP) bypassing the normal A-V node - His-Purkinje pathway (NP) appears to account for the features of the WPW syndrome described during electrical stimulation of the heart, epicardial mapping, and surgical correction. During circus movement tachycardia utilizing the AP for ventriculo-atrial conduction, a sequence of atrial activation beginning at the AP can be anticipated. If the point of earliest atrial activation during circus movement tachycardia is used as the presumptive location of the AP, the influence of atrial pacing site on assessment of antegrade conduction over the AP can be evaluated.

Assessment of retrograde conduction in the WPW syndrome has been limited by the inability to distinguish whether atrial depolarization proceeds over the NP, the AP, or both. Utilizing multiple simultaneous atrial recording sites during ventricular pacing and premature stimulation, atrial fusion has been routinely found in patients with accessory pathways proven to be located laterally. In such cases, assessment of refractoriness in the AP has been possible only when an atrial electrogram was recorded near the AP.

This study is an attempt to evaluate the importance of pacing and recording site on the assessment of the conduction properties of the AP. The technique of study highlights several important problems which can complicate interpretation of electrophysiologic studies in the presence of dual A-V pathways.

Materials and Methods
Between 6/1/73 and 6/1/74 91 patients were evaluated for arrhythmias suspected to reflect pre-excitation syndromes. The presence of an AP was excluded in six. The remaining 25 patients form the basis of this report. Following informed consent, all patients were studied in the fasting basal state. In 23, all medication was discontinued and verified, in most cases, by absence of detectable blood levels of digitalis, quinidine, or procainamide. Propranolol was discontinued at least 48 hours prior to study. Procainamide was continued in two patients to control the ventricular rate during atrial fibrillation.

In all cases three or four catheters were introduced for recording or stimulating. A tri- or hexapolar catheter was positioned across the tricuspid valve for recording the His bundle electrogram and low right atrial electrograms near the NP. A quadripolar or octapolar catheter was positioned in the coronary sinus for recording left atrial (LA) and postero-basilar left ventricular (LV) electrograms and for LA pacing. Rarely, pacing from the coronary sinus results in LV pacing, simulating LV pre-excitation. Left atrial pacing in Type A WPW was confirmed by a stimulus-to-delta interval that varied with different pacing sites in the coronary sinus. In addition, LA premature beats at coupling intervals shorter than the effective refractory period (ERP) of the AP resulted in QRS normalization, a finding which would not occur if the LV were being paced.

A quadripolar catheter was positioned in the right atrium (RA) for stimulating and recording at various sites. A bipolar...
catheter was positioned in the right ventricular (RV) apex for pacing. If, for technical reasons, the study had to be limited to three catheters, the RV catheter was sacrificed and a hexapolar His bundle catheter was advanced sufficiently far to achieve RV pacing from the distal pair, while recording right atrial electrograms near the NP from the proximal electrode pair. The quadripolar atrial recording catheter had 10 mm interelectrode distances. The octapolar atrial recording catheter was especially designed with a 2 mm interelectrode distance. In each case, all atrial electrograms were measured from the onset of the rapid deflection. Following introduction of the catheters, heparin, 100 units/kg, was administered, i.v.

All catheter electrodes were connected to a junction box to facilitate selection of electrograms. Intracavitary electrograms were filtered at a low pass of 50 Hz and a high pass of 1 KHz. Surface leads together with intracavitary electrograms were stored on magnetic tape at speeds of 1 2/3 or 3 3/4 inches/second and played back on a Siemens 8-channel ink jet recorder at speeds of 100 or 200 mm/sec. Studies were performed with a specially designed stimulator utilizing photoelectric isolation. * Stimulus duration was 2 msec at twice diastolic threshold. Following every eighth driven beat (S1) or every eighth spontaneous beat, programmed premature beats (S2) were delivered at coupling intervals reaching down to the atrial or ventricular refractory period. The antegrade or retrograde effective refractory period (ERP) of the AP was determined as the longest A1A2 or V1V2 which failed to propagate over the AP. Immediate analysis of electrogram sequence was facilitated by using a triggered 8-channel Tektronic storage oscilloscope.

Study Protocol

Following introduction of the catheters, an episode of circus movement tachycardia was initiated with premature stimulation. Lead V1 was recorded together with five simultaneous intra-atrial recording sites or three surface leads and three atrial recording sites to localize the earliest recordable atrial electrogram. The tachycardia was then terminated and retrograde conduction assessed during RV or LV pacing and premature stimulation. Retrograde conduction studies utilized up to five simultaneous atrial recording sites. They were chosen so that one site included the earliest atrial electrogram during tachycardia and one the atrial electrogram near the NP recorded on the HB catheter.

Antegrade conduction was assessed utilizing three surface leads together with an atrial electrogram near the stimulus site, an atrial electrogram near the NP on the His bundle catheter, and an atrial electrogram near the AP as determined by the atrial sequence during circus tachycardia. In most instances the positions of the RA and coronary sinus catheters were the same as during assessment of retrograde conduction. The effect of pacing site on the presence and degree of pre-excitation and on the antegrade ERP of the AP was then determined at several cycle lengths.

Results

Localization of the AP for Electrophysiologic Study

Circus movement tachycardia which utilized the AP for retrograde conduction to the atrium was initiated in 23 of 25 patients. Of the 23, the coronary sinus was successfully catheterized in 21. During circus movement tachycardia the earliest recordable atrial electrogram was localized. This was facilitated by the use of five simultaneous intra-atrial recording sites. The location of the earliest atrial electrogram was considered the site nearest the AP.

Figure 1 illustrates retrograde atrial activation sequences obtained during circus movement tachycardia in four patients. Figure 1A illustrates the sequence in a patient with a surgically-proven right lateral AP. Note that in panel A the atrial electrogram from the low lateral RA (LLRA) preceded atrial activity from the region of the septum near the NP recorded on the HBE catheter and from three LA sites, recorded proximal to distal, in the coronary sinus. In panel B the retrograde atrial sequence is shown in a patient with an AP localized near the septum based on this atrial sequence. The 12-lead ECG during ventricular pre-excitation and the atrial sequence during circus movement tachycardia were similar to a previously proved septal AP. 24 In this case, atrial septal activation recorded from the HBE preceded the LLRA and LA recorded from proximal, mid, and distal coronary sinus.

*Designed by Michael Fieozor, Ph.D., Duke University.

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Figure 1

The sequence of retrograde atrial activation recorded during circus movement tachycardia is shown for four accessory pathway (AP) locations. In panel A for a surgically-proven right lateral AP; panel B for an AP in or near the septum; panel C for a left posterior AP; and in panel D for a surgically-proven left lateral AP. The dotted line denotes the earliest activation from the atrial electrogram.
Although a similar sequence might be anticipated during A-V junctional tachycardias, two observations argue against its presence in this case. First, during atrial pacing, tachycardia was initiated only when a programmed atrial premature beat blocked in the AP. This would be extremely fortuitous if the mechanism was A-V nodal re-entry. Second, during circus movement tachycardia a ventricular paced beat occurring synchronously with the antegrade His deflection conducted retrogradely with the same atrial sequence. Since the His and A-V node were refractory, the impulse had to have traveled over an AP.

In contrast, panel D shows the atrial sequence during tachycardia in a patient with a surgically proven left lateral AP. Notice here that the LA recorded from the distal coronary sinus preceded more proximal LA electrograms, the atrial electrogram near the NP on the HBE catheter, and LLRA. Panel C shows the sequence in a patient with an AP presumed to be near the septum in a left posterior position. In this instance, LA activation recorded from the proximal coronary sinus preceded LA activation from the distal coronary sinus, the atrial electrogram near the NP on the HBE catheter, and the LLRA.

The localization of the AP, to the free wall of the LV, RV, or septum, by recording the earliest atrial electrogram during circus movement tachycardia was verified by epicardial mapping in 12 of 13 patients undergoing attempted surgical correction during the study period. The one failure was related to technical problems in catheterizing the coronary sinus in a patient with a left lateral AP.

Assessment of Antegrade Conduction over the Accessory Pathway

Verifying the Location of the Accessory Pathway. The effect of atrial pacing and recording site on the assessment of antegrade conduction was studied following localization of the AP as illustrated above.

When the atria were paced at the same cycle length from the various recorded atrial sites as illustrated above, the degree of pre-excitation was greatest and the stimulus-to-delta was shortest at the site of earliest atrial activation during circus movement tachycardia. This was true in all patients and supported the initial localization of the AP. This would be true intuitively since the earlier the atrial input to the AP, relative to the NP, the greater the degree of pre-excitation. Frequently, this could be most readily appreciated by inspection of the surface ECG. This is shown in figure 2 for the patient whose atrial sequence during tachycardia was illustrated in figure 1, panel D. Simultaneous leads are shown during atrial pacing (CL = 500 msec) at four of the sites illustrated in figure 1 (LLRA, low medial RA near the NP on the HBE catheter, proximal, and distal coronary sinus). From the four pacing sites the stimulus-to-delta interval was 135, 125, 80, and 30 msec and the delta deflection preceded the His deflection by 15, 30, 70, and 100 msec, respectively. This phenomenon has been observed in a few other cases.

Causes of Apparent Antegrade Block in the Accessory Pathway. Occasionally pre-excitation was neither evident from the surface ECG nor demonstrable by an abnormally short H-V interval when pacing from atrial sites remote from the AP. There were two causes for the apparent absence of pre-excitation: 1) late input into the AP relative to the NP and 2) ineffective input into the AP due presumably to the pattern of atrial activation, resulting in failure of either conduction over or entrance into the AP.

Absence of pre-excitation due to late input into the AP is illustrated in figure 3. The atrial sequence during tachycardia for this patient with a right lateral AP was illustrated in figure 1, panel A. Atrial pacing from the proximal coronary sinus near the NP and remote from the AP resulted in no discernable pre-excitation and an H-V of 40 msec. Note that during atrial pacing (S1) the His bundle was activated over the NP coincident or slightly before the LLRA near the AP. However, on the premature beat (S2), conduction through the A-V node slowed. The LLRA now activated prior to the His bundle and pre-excitation was present, establishing that the absence of pre-excitation was due to late input. The persistence of this pattern during the sinus beat following S2 provided further support for this explanation. Pre-excitation was present with the LLRA near the AP again activates early relative to the His bundle.

Absence of pre-excitation due to ineffective input is
illustrated (fig. 4) in a patient with a left lateral AP verified by epicardial mapping at surgery. During sinus rhythm no evidence of pre-excitation was seen. In addition, the RA was paced, from the appendage, HRA, and LLRA, to heart rates at which Type I block occurred in the A-V node without evidence of pre-excitation. In contrast, pacing the LA anywhere along the coronary sinus or just at the ostium of the coronary sinus resulted in unequivocal pre-excitation.

Figure 4, panel B, shows that during LA pacing from the proximal coronary sinus marked pre-excitation was present. The distal LA electrogram near the AP preceded the delta by 40 msec and the His, presumably obscured in ventricular activation by 225 msec. However, during high RA pacing at the same cycle length but with no pre-excitation (panel A) the LA electrogram from the distal coronary sinus near the proven location of the AP preceded the His bundle deflection by 80 msec. Therefore absence of pre-excitation in this case was probably due to ineffective rather than late input. As a consequence of these observations, we now feel that the finding of unidirectional antegrade block in the AP cannot be made until the possibility of late and/or ineffective input into the AP is excluded by pacing at several CLs at multiple right and left atrial locations.

**Figure 3**

LA pacing (S<sub>1</sub>) produces disappearance of pre-excitation in a patient with a proven right lateral AP due to late input. Delay in A-V nodal conduction with S<sub>2</sub> or in resumed sinus beat permits manifest pre-excitation.

**Figure 4**

In a patient with a surgically-proven left lateral AP, pre-excitation was absent during RA pacing due to ineffective input (panel A). However, marked pre-excitation is present during LA pacing (panel B).
Effect of Pacing and Recording Site on Assessment of the Antegrade Refractory Period of the Accessory Pathway. In 24 of 25 patients the antegrade ERP of the AP was determined by pacing at sites close to the AP. In 20 of these 24, the effect of pacing other atrial sites on the assessment of the antegrade ERP of the AP was also determined. In each instance, whether pacing near the AP or in the contralateral atrium, atrial electrograms were recorded near the AP and stimulating site. The antegrade ERP of the AP was virtually the same regardless of the location of atrial premature stimulation provided one used the A1A2 recorded near the AP site. If the A1A2 near the site of stimulation remote from the AP was used, the interval was occasionally shorter than the A1A2 measured near the AP due to intra-atrial conduction delay of A2 at close coupling intervals. As a consequence apparent differences of 5–50 msec in the antegrade ERP of the AP could result if atrial electrograms were not recorded near the AP.

In three instances the antegrade ERP of the AP was 15–30 msec longer than the ERP of the atrium when stimulating the atrium near the AP. When stimulating the contralateral atrium, the ERP of the atrium was the same, but due to intra-atrial conduction delay of A2, the minimum A1A2's achievable at the AP were longer than the ERP of the AP. As a consequence, the ERP of the AP could only be determined when stimulating the atrium near the AP. These findings suggest that the most reliable determination of the antegrade ERP of the AP resulted from stimulating and recording atrial electrograms near the AP.

Assessment of Retrograde Conduction

The assessment of the antegrade ERP of the AP is facilitated by the relative ease of discerning when an impulse fails to propagate over the AP to the ventricle. The delta wave disappears and the H-V interval becomes normal, or, when block occurs in both the AP and NP, propagation to the ventricles fails completely. The determination of the retrograde ERP of the AP is frequently complicated by uncertainty as to whether the NP, the AP, or both are conducting the impulse retrogradely to the atrium. During ventricular pacing, it is reasonable to anticipate in most circumstances that the impulse will travel retrograde to the atrium over both pathways resulting in atrial fusion analogous to the ventricular fusion that occurs during atrial pacing. In all patients with laterally located APs, except one, who had complete retrograde block in both pathways, retrograde atrial fusion was demonstrated.

During right ventricular pacing, the retrograde conduction time over the NP ought to lengthen in response to a ventricular premature beat that encroaches on the relative refractory period of the NP. In contrast, retrograde conduction should remain relatively constant over the AP, as the coupling interval of the ventricular premature beat shortens down to the retrograde ERP of the AP.28 By recording atrial electrograms near the two pathways during ventricular pacing and the corresponding retrograde conduction time of the premature ventricular beat (V2A2) at each location, one could theoretically analyze retrograde conduction over each pathway.

This hypothesis was tested in 23 of 25 patients. For analysis of retrograde conduction over the AP, the atrial recording site was the earliest recorded atrial electrogram during circus movement tachycardia, as illustrated in figure 1. The atrial electrogram on the His bundle catheter was chosen as a site near the NP. In addition, up to three other atrial recording sites were selected.

This method is illustrated in figures 5 and 6. Figure 5 shows a composite sequence of atrial activation during circus movement tachycardia in a patient with a left lateral AP verified by epicardial mapping. Notice that activation begins in the left atrium recorded from the distal coronary sinus (CS 6, 5, 4), occurs later in the more proximal coronary sinus (CS 3, 2, 1), appears later yet near the NP on the HBE catheter, and is still later in the high RA. In figure 6, V1, together with the LLRA, RA on the HBE catheter near the NP, and the LA (CS 4, 5, 6) near AP, are recorded during RV pacing (CL = 700 msec). During basic drive (S1), the atrial sequence was different from that recorded during tachycardia, with the atrial electrogram on the HBE catheter occurring earliest. In panel A, a premature beat (S1S2 = 440 msec) resulted in prolongation of the retrograde conduction time to the RA sites. As the coupling interval shortened, progressive prolongation of conduction to the RA sites occurred as illustrated in panel B (S1S2 = 550 msec) and panel C (S1S2 = 800 msec). In contrast, the retrograde conduction time to the LA (CS 4, 5, 6) remained essentially unchanged, resulting in a progressive shift in atrial sequence. This suggested that retrograde atrial depolarization resulted from fusion of impulses arriving over both pathways.

During ventricular premature stimulation progressive shifts in atrial sequence could arise in the absence of fusion. This could occur with retrograde conduction exclusively over the AP if the impulse arrived at the atrium and conducted with progressive degrees of intra-atrial conduction delay as V1V2 shortened. In the case illustrated above, and in the following cases illustrating atrial fusion, this was not found to be the case. During assessment of antegrade conduction, all catheters were in similar positions when the atrium was paced near the AP.
duced near the AP conducted without intra-atrial delay to the LLRA. This was true over a range of coupling intervals comparable to the retrograde A1A2 intervals during which atrial fusion was demonstrated.

The effect of shortening the V1V2 interval on the retrograde conduction time (V2A2) of the premature beat is illustrated in figure 7 for a patient without an accessory pathway. During RV pacing (CL=600 msec) atrial activation near the NP on the HBE catheter preceded the LLRA and LA (mid CS). As V1V2 shortened, the relative sequence of atrial electrograms remained unchanged and the V2A2 lengthened until retrograde conduction failed (V1V2 of 365 msec). The sequence of electrograms and the prolongation of retrograde conduction with premature beats was compatible with retrograde conduction exclusively over the NP (unpublished observations). This example is included for comparison with V-A conduction over dual pathways and is not intended to specify the site of conduction delay or imply that V-A conduction always occurs in normal subjects.

In contrast, the effect of shortening V1V2 on V2A2 is shown for the patient whose atrial sequence during circus movement tachycardia was illustrated in figure 1, panel C. During circus movement tachycardia the earliest atrial activation occurred in the LA recorded from the proximal coronary sinus suggesting an atrial location near the AP. The data in figure 8 were obtained during LV pacing (CL=500 msec). Only two of five electrograms were plotted for simplicity. As V1V2 shortened V2A2 near the NP on the His bundle catheter gradually lengthened, compatible with retrograde conduction over the NP. However, V2A2 at the LA near the AP remained constant down to a coupling interval of 310 msec. A 10 msec increase in V2A2 occurred at V1V2 of 300 msec. At 290 msec a sudden increase in V2A2 occurred accompanied by a normalization of atrial sequence. Note that now the RA depolarization near the NP on the His bundle catheter preceded the LA electrogram. Therefore, retrograde fusion was occurring with coupling intervals down to 300 msec. At shorter coupling intervals,
Retrograde block occurred in the AP and the atria were activated exclusively over the NP. Because of retrograde fusion, a single RA recording site could not have detected retrograde conduction over the AP, nor could the retrograde ERP of the AP have been determined.

This phenomenon is not confined exclusively to left-sided APs. Figure 9 illustrates the problem in a patient whose atrial sequence during circus movement tachycardia was shown in panel A of figure 1. A right lateral AP was verified by epicardial mapping. In this case too, V2A2 near the NP on the His bundle catheter lengthened as V1V2 shortened, characteristic of conduction over the NP. However, V2A2 at the LLRA near the AP remained essentially constant to within 15 msec of the ERP of the ventricle (290 msec). As a consequence, the selection of an inappropriate RA recording site in cases of right-sided APs may also lead to erroneous conclusions regarding retrograde conduction over the AP.

As V1V2 shortened, block in the AP was accompanied by a sudden lengthening of V2A2 if conduction continued over the NP. However, the converse was not necessarily true. A sudden increment in V2A2 as
V1V2 shortened could result from failure of retrograde conduction over the NP. This is a logical consequence of dual retrograde atrial activation. The phenomenon is illustrated in figure 10 for the patient whose atrial sequence during tachycardia was illustrated in figure 1, panel D. The sequence suggested a left lateral AP and was verified by epicardial mapping. As V1V2 shortened from 400 to 320 msec V2A2 near the NP showed only a slight increase. At a V1V2 of 315 msec, V2A2 suddenly increased by 125 msec. This was not due to block in the AP. The V2A2 to the LA near the AP remained unchanged. Furthermore, at coupling intervals less than 320 msec, the LA preceded the atrium near the NP typical of the sequence expected from atrial activation over the AP. The explanation for failure of conduction over the NP at coupling intervals less than 320 msec is not clear. Failure of retrograde conduction over the NP was further verified by induction of typical circus movement tachycardia at V1V2 of 280 msec. Therefore, a sudden increment in V2A2 recorded from a site remote from the AP may not indicate block in the AP.

An alternative explanation for this observation would postulate the presence of a "concealed" septal accessory pathway that conducted only in the retrograde direction. This may be suggested by the relatively small increment in V2A2 near the NP until block occurred with coupling intervals less than 320 msec. However, several observations argue against this interpretation. A circus movement tachycardia was only induced with the atrial activation sequence illustrated in figure 1, panel D. During circus movement tachycardia utilizing the left lateral AP for retrograde conduction, paced ventricular beats during the diastolic interval were not observed to propagate to the atrium over a septal pathway. A postoperative study following successful division of the left lateral pathway would have been conclusive.

It is quite clear that retrograde atrial fusion occurs and when present necessitates recording atrial sites near the AP and NP in order to interpret the responses to ventricular pacing and premature stimulation. Every right or left lateral AP proven by epicardial mapping or suggested by atrial activation sequence during circus movement tachycardia has demonstrated retrograde atrial fusion. In each case, the retrograde ERP of the AP could not be reliably determined.
employing an atrial electrogram remote from the AP.

Our experience with patients who have had septal APs either proven at surgery or suggested by catheter recording techniques differs somewhat. To date, retrograde atrial fusion has not been detectable utilizing these techniques. This is most likely due to the proximity of these two pathways, permitting the atrial impulse from the AP to antegradely penetrate the A-V node and block the retrograde impulse from reaching the atrium over the NP. In these cases, atrial recording site was not critical in assessing the retrograde ERP of the AP. However, the need for multiple simultaneous atrial recording sites remains since they are a requisite for verifying a septal location.24, 28

Discussion

The use of multiple catheter recording sites along the coronary sinus for analysis of atrial and ventricular activation during sinus rhythm in Type A WPW has been described by Torresani et al.29 However, the potential of this technique has not been widely appreciated for the study of retrograde conduction. Early activation of the LA during circus movement tachycardia in Type A WPW has been reported by Wells et al. who recorded through a patent foramen ovale. Using an endocavitary RA recording and an esophageal electrode, Grolleau et al. were able to demonstrate differences in atrial sequences during sinus rhythm, junctional escape beats, ventricular premature beat conducted retrogradely to the atria, and in tachycardia in a patient with Type A WPW.30

The coronary sinus is a particularly advantageous site for the analysis of atrial activation in left-sided APs. Its location along the posterior and lateral left A-V groove allows one to record adjacent LV and LA activity on the same bipolar electrogram. Near the AP a close temporal sequence is recorded between LA and LV electrograms during sinus rhythm and retrograde conduction.

During a circus movement tachycardia conducting retrogradely to the LA over the AP we have consistently demonstrated LA activation prior to atrial activation near the NP on the HBE catheter or other RA sites, and a short LV-to-LA interval (30–40 msec) on the coronary sinus electrogram. This technique for localizing the AP is limited by the inability to know the location of the recording electrode precisely and the inability to record along the entire left A-V groove.

An additional problem is encountered with interpretation of atrial electrograms at or near the ostium of the coronary sinus. It is difficult with our present information to decide whether the atrial activation recorded there represents right or left paraseptal or true atrial septal activation. In the RA, the problem of the precise location is more difficult since no fixed anatomic structure analogous to the coronary sinus is available to record along the right A-V groove.

These important limitations prevent precise anatomic localization of the AP, but we have consistently identified whether the AP was in the free wall of the RV, the LV, or was near the septum. A new electrode catheter technique developed by one of us (JJG) promises to resolve the problem of more precise anatomic localization (to be published elsewhere).

Since the atrial sequence during tachycardia utilizing a septal AP may resemble that seen during retrograde atrial activation over the NP or during a-V nodal re-entry, further evidence is needed to confirm that a septal AP is functional. This would include: 1) mode of induction of tachycardia, mentioned above; 2) effect of ventricular premature beats on the AA interval during tachycardia and propagation of ventricular premature beat retrogradely to the atrium when the NP is refractory,31, 32 and 3) a constant V-A interval at the onset of the tachycardia independent of alterations in the A-V interval, in contrast to the pattern typical in A-V nodal re-entry.33, 34

From a therapeutic point of view the precise localization of the AP is less important than the ability to exclude an AP located in or near the septum. Surgical intervention in this location is more difficult.25 In spite of these limitations, we have correctly predicted the approximate location of the AP prior to surgery in 12 of 13 patients during this study period. The one failure occurred in a patient with a left lateral AP in whom the coronary sinus could not be catheterized.24

From the standpoint of electrophysiologic evalu-
tion, the precise knowledge of the anatomic location of the AP along the right or left A-V groove is less important than the ability to locate the earliest retrograde atrial electrogram during circus movement tachycardia. This recording site is considered to be located near the AP in the atrium. Systematic evaluation of these 25 patients has shown how errors in assessing antegrade and retrograde conduction over the AP may arise. During assessment of the antegrade ERP of the AP significant errors may arise unless the A1A2 near the AP is recorded, due to intra-atrial conduction delay between the site of atrial stimulus and the AP. This may be particularly important when assessing the effect of drugs on the antegrade ERP of the AP. Drugs which affect atrial refractoriness and/or intra-atrial conduction time may cause apparent changes in the antegrade ERP of the AP which are due solely or in part to changes in the intra-atrial conduction time of the premature beat to the AP. The only way this error can be avoided is by being sure the A1A2 intervals obtained reflect electrograms near the AP when block occurs.

More serious errors in the assessment of retrograde conduction will arise unless atrial activation near the NP and the AP is recorded. For an AP to participate in the patient’s tachycardia, the retrograde ERP of the AP must be shorter in msec than the CL of the tachycardia. However, the presence of two A-V pathways may result in retrograde atrial fusion during ventricular pacing. In every laterally-located right or left AP, analysis of retrograde conduction with an atrial recording site remote from the AP has failed to show retrograde conduction characteristic of the AP. At these remote locations, the increasing retrograde conduction time of ventricular premature beats with decreasing coupling intervals was compatible with conduction over the NP. Only when recording near the AP was the retrograde conduction time relatively independent of ventricular coupling interval down to the retrograde ERP of the AP. This characteristic of conduction over an AP was verified by Wellens in two patients in whom the His bundle was divided to terminate refractory circus movement tachycardia.

Occasionally the retrograde conduction time over the AP is not completely independent of coupling interval. As V1V2 shortens V2A2 may be shorter than V1A1, suggesting supernormal conduction over the AP. This phenomenon, though not commented on, is seen in figure 4 of a study by Wellens and Durrer, and has been observed in several of our patients with left-sided APs. The phenomenon is illustrated in figure 6, panel A, where the retrograde conduction time from S2 to the LA over the AP shortened by 15 to 20 msec. However, careful inspection of the record will show that the V-A interval on the coronary sinus electrogram did not change, suggesting that activation of the postero-basilar LV occurred earlier with S2. This earlier activation of the postero-basilar LV with right ventricular premature beat was observed by Castellanos et al. in a study of impulse propagation during RV pacing employing coronary sinus catheters for LV recordings.

In addition to apparent shortening of V2A2 at some coupling intervals V2A2 may lengthen 15 to 20 msec at coupling intervals that approach the ERP of the ventricle or the retrograde ERP of the AP. In the former case, latency and/or intraventricular conduction delay can be demonstrated. In the latter case, slight slowing of conduction in the AP has been demonstrated in a few patients with left lateral APs. This is evident by a 5–20 msec increase in the V-A interval recorded on the CS electrogram at coupling intervals 5–20 msec longer than the retrograde ERP of the AP. This phenomenon was responsible for the slight increase in V2A2 over the AP shown in figure 7.

In an occasional patient, the site of ventricular pacing may also be a critical determinant in assessing retrograde conduction over the AP. In spite of appropriate atrial recording sites, analysis of retrograde conduction during RV pacing could not establish with certainty that the impulse traversed the left-sided AP in the patient illustrated in figures 1, panel C, and 7. Fortunately, we were able to pace the LV through a patent foramen ovale and establish the retrograde conduction properties of the AP as shown in figure 7.

Similarly, our results with multiple atrial pacing sites show that some cases of antegrade block in the AP may result from late or ineffective input into the AP. Therefore, one must pace from both right and left atrial sites in cases of tachycardia suspected of having unidirectional antegrade block in an AP. The application of these techniques to the identification of patients with true antegrade block in the AP is discussed in detail elsewhere (submitted for publication).

In summary, localization of the AP by recording the sequence of retrograde atrial activation during circus movement tachycardia is critical for assessing the antegrade and retrograde conduction properties of the AP. Utilizing the techniques described, retrograde atrial fusion has been consistently found to date in every laterally located AP, but not in those APs located in or near the septum. The atria and ventricles may have to be paced from many sites to demonstrate antegrade and retrograde conduction over the AP and to accurately determine the conduction properties of the AP.

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References
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