New Catheter Technique for Recording Left Free-Wall Accessory Atrioventricular Pathway Activation
Identification of Pathway Fiber Orientation

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The ability to record accessory atrioventricular (AV) pathway activation consistently may be uniquely beneficial in improving pathway localization, identifying anatomic relations, and providing insight into unusual conduction properties. For the purpose of recording left AV accessory pathway activation, an electrode catheter was specially designed for use in the coronary sinus. The orthogonal catheter has three sets of four electrodes spaced evenly around the circumference. Electrograms were recorded at low gain (<1 cm/mV) between adjacent electrodes on the same set (interelectrode distance, 1.5 mm, center to center). This provides a recording dipole perpendicular to the atrioventricular groove to enhance recording of accessory pathway activation while minimizing overlapping atrial or ventricular potentials. The orthogonal electrode catheter was used in the electrophysiological study of 48 consecutive patients with 59 left AV accessory pathways. The catheter could be advanced along the coronary sinus beyond the site of earliest retrograde atrial activation in 49 of the 59 accessory pathways. Activation potentials were recorded from 45 of the 49 (92%) accessory pathways accessible to the catheter (5 of 5 anterior, 8 of 8 anterolateral, 15 of 16 lateral, 5 of 5 posterolateral, 5 of 5 posterior, and 7 of 10 posteroseptal). Accessory pathway potentials were validated by dissociating them from both atrial and ventricular activation by programmed-stimulation techniques. During surgery, accessory pathway potentials were identified from orthogonal catheter electrodes in the coronary sinus in 14 of 16 accessory pathways (12 patients). Epicardial mapping confirmed the location of the accessory pathway, and direct pressure over the orthogonal catheter electrode that recorded the accessory pathway potential resulted in transient conduction block in nine of the 14 accessory pathways. Orthogonal electrode maps of the coronary sinus identified an oblique course in 39 of 45 recorded accessory pathways. Thirty-two of 38 left-free wall accessory pathways were oriented with atrial insertion 4–30 mm (median, 14 mm) proximal (posterior) to the ventricular insertion. In the remaining six free-wall accessory pathways, the lateral excursion could not be determined because either only the atrial end of the accessory pathway was recorded or activation of multiple pathway fibers prevented tracking of individual strands. The seven recorded posteroseptal pathways exhibited accessory pathway potentials throughout an 8–18-mm (median, 10 mm) length of the proximal coronary sinus, but fiber orientation was difficult to determine. We conclude that left AV accessory pathway activation can be recorded consistently from orthogonal catheter electrodes in the coronary sinus. Direct accessory pathway recordings revealed an oblique fiber orientation and may provide more precise localization for surgical or catheter ablation. (Circulation 1988;78:598–610)

Electrophysiological studies in patients with Wolff-Parkinson-White syndrome have identified many functional characteristics of accessory atrioventricular (AV) pathways by recording activation times of the atrium and ventricle close to the pathway.1,2 The basis for many of

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Supported in part by the American Heart Association, Oklahoma Affiliate, Young Investigator Award OK-83-Y-2, and Oklahoma Health Research Program, Grant HRC-RRP-A-028.

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Received November 30, 1987; revision accepted May 5, 1988.
these findings is not well understood; two such findings, for example, are the marked variability in accessory pathway conduction properties between patients and differences between antegrade and retrograde conduction within the same pathway.3–13 Structural differences may account for much of the physiological variability,14–20 and the ability to record activation of the pathway fibers may help elucidate these relations. Activation potentials of right AV accessory pathways have been recorded intermittently by placing catheter electrodes across the endocardial surface of the tricuspid annulus.21–27 Recordings of left AV accessory pathway activation from conventional catheter electrodes in the coronary sinus have been limited.28

This study describes a new catheter technique that allows consistent recording of activation potentials from left AV accessory pathways. These pathways generally traverse the AV groove on the epicardial side of the anulus fibrosis, relatively close to the coronary sinus or the great cardiac vein.17,18 Attempts to identify accessory pathway potentials from the coronary sinus in previous studies may have been limited by electrode orientation. Conventional bipolar electrodes along the axis of the catheter produce a recording dipole oriented parallel to the coronary sinus. These electrodes amplify the component of the atrial or ventricular impulse propagating parallel to the AV groove. The accessory pathway impulse, propagating at an angle to the recording dipole, is minimized and lost within the larger atrial or ventricular potential. With increasing interelectrode spacing, the bipolar electrogram contains components of atrial and ventricular activation from greater distances, producing wider potentials that further conceal the accessory pathway component. To enhance the accessory pathway potentials and minimize the overlapping atrial or ventricular potentials, we used low-recording amplification and small, closely spaced bipolar electrodes oriented perpendicular to the long axis of the catheter.

Figure 1. Schematic representation of the orthogonal electrode catheter. Darkened areas represent electrodes. A, B, and C are the distal, middle, and proximal group or ring of electrodes. Each ring is composed of four separate electrodes spaced equally around the circumference of the catheter (interelectrode distance is 1.5 mm, center to center). Rings of electrodes are spaced at 10-mm intervals, center to center.

Methods
A 6F orthogonal electrode catheter was constructed with 12 small (1-mm²) rectangular electrodes (Webster/Mansfield Catheters, Mansfield Scientific, Mansfield, Massachusetts). The electrodes were arranged in three groups; each group contained four electrodes placed circumferentially around the catheter to form a ring (Figure 1). The distance between adjacent electrodes in the same ring is 1.5 mm, center to center, and the distance between rings is 10 mm. An orthogonal electrode catheter was used in the electrophysiological study of 48 consecutive patients with left AV accessory pathways. Under local anesthesia, the catheter was inserted percutaneously into the left subclavian or right internal jugular vein. It was advanced into the coronary sinus and positioned as anteriorly as possible in the great cardiac vein. Electrograms from each of the four adjacent bipolar electrodes (1–2, 2–3, 3–4, and 4–1; Figure 1) were examined on one or more of the rings to identify the electrodes facing the myocardium. The two contiguous electrode pairs (1–2 and 2–3, 2–3 and 3–4, or 3–4 and 4–1) that exhibited the sharpest atrial and ventricular potentials were selected. Electrograms from the same two electrode pairs on each of the three electrode rings (A, B, and C; Figure 1) were recorded simultaneously with the same low amplification (usually 0.5–1.0 cm/mV) and filter bandwidth of 30–500 Hz.

During AV reentrant tachycardia using the accessory pathway for retrograde conduction (ortho-
dromic reciprocating tachycardia) or ventricular pacing (when retrograde AV nodal conduction was excluded), the catheter was slowly withdrawn until the proximal electrode ring lay outside the coronary sinus ostium. The site(s) of earliest retrograde atrial activation was located by fluoroscopic imaging of the electrodes in the left anterior oblique projection and was classified by the schema illustrated in Figure 2. Posteroseptal pathways in which the earliest retrograde atrial activation was recorded along the tricuspid anulus were excluded from the present study and are described in another report. Any potential that preceded atrial activation within 2–3 cm of the site of earliest retrograde atrial activation was considered to possibly represent retrograde accessory pathway activation. An accessory pathway origin for the potential was considered confirmed only when both atrial and ventricular origins could be excluded by the procedures described below.

The programmed stimulation techniques used to dissociate the retrograde accessory pathway (AP) potential from local atrial and ventricular potentials
are illustrated in Figure 3. During AV reentrant tachycardia, rapid ventricular pacing, or introduction of ventricular extrastimuli, retrograde block may occur near the accessory pathway–atrial interface (AP-A). This would result in loss of the atrial potential while maintaining the AP potential, providing evidence that the AP potential does not result from atrial activation (Figure 3A). The retrograde AP potential can be dissociated from the local atrial potential in the absence of AP-A block by delivering a late atrial extrastimulus. When properly timed, the atrial extrastimulus will advance the timing of the local atrial potential without affecting the retrograde AP potential (Figure 3B).

A ventricular origin for the retrograde AP potential may be excluded by the loss of the accessory pathway potential during retrograde block in those patients in whom retrograde block occurs near the ventricular–accessory pathway (V-AP) interface (Figure 3C). Alternatively, atrial extrastimuli may be used to dissociate the retrograde AP potential from ventricular activation. Critically timed atrial extrastimuli may antegradely activate the accessory pathway, advancing the timing of the AP potential
without altering the timing or morphology of the local ventricular potential or the QRS complex (Figure 3D).

The coronary sinus and great cardiac vein were also examined during sinus rhythm or atrial pacing. Near the site(s) of earliest ventricular activation or previously identified retrogradely conducting pathways, potentials that preceded ventricular activation were evaluated as possibly representing antegrade accessory pathway activation. Programmed-stimulation techniques used to exclude atrial and ventricular origins for the antegrade AP potentials are similar to those described for validating the retrograde AP potential and are illustrated in Figure 4. An atrial origin can be excluded by the occurrence of block near the atrial–accessory pathway interface (A–AP), resulting in loss of the AP potential while maintaining local atrial activation (Figure 4A). Atrial activation can also be excluded by delivering critically timed ventricular extrastimuli, which retrogradely activate the accessory pathway and advance the AP potential without changing the timing or morphology of the local atrial potential (Figure 4B). A ventricular origin for the antegrade AP potential may be excluded in some patients by the occurrence of block near the accessory pathway–ventricular interface (AP–V), resulting in the loss of ventricular activation while maintaining the AP potential (Figure 4C). In the absence of AP–V block, a late ventricular extrastimulus may be used to dissociate the antegrade AP potential from ventricular activation. The ventricular extrastimulus advances the local ventricular potential without affecting the timing or morphology of the AP potential (Figure 4D).

To further validate the accessory pathway origin of the AP potentials, the orthogonal electrode catheter was used intraoperatively in 12 patients undergoing surgical ablation of 16 left AV accessory pathways. During normothermic cardiopulmonary bypass, the catheter was inserted through a right atriotomy into the coronary sinus and advanced to the great cardiac vein. The coronary sinus and great cardiac vein were mapped during AV reentrant tachycardia or ventricular pacing and during sinus rhythm or atrial pacing as described above. The catheter was positioned to record the AP potential from orthogonal electrodes on the distal ring, close to the catheter tip. In this position, the catheter tip provided a palpable reference to the location of the AP potential. Epicardial mapping of the AV groove was performed with a hand-held bipolar electrode oriented perpendicular to the axis of the coronary sinus and parallel to the orthogonal catheter electrodes. The epicardial electrode was then positioned over the catheter tip electrode, and light pressure was applied to determine the effect on the AP potential and accessory pathway conduction.

Results

Orthogonal electrode maps of the coronary sinus and great cardiac vein in the 48 patients identified the presence of 59 separate left AV accessory pathways. The regional distribution of the pathways is illustrated in Figure 5. The catheter could not be advanced far enough into the great cardiac vein to cross four lateral and six anterior or anterolateral pathways. Of the 49 pathways accessible to the catheter electrode, AP potentials were recorded and dissociated from both atrial and ventricular activation in 45 (92%). The ability to record acces-

Figure 5. Schematic representation of the coronary sinus and great cardiac vein in the left anterior oblique projection, which illustrates the distribution of the 59 accessory pathways in the 48 patients studied. Number in parentheses indicates all pathways, including those that were not crossed by the catheter. In the fraction below, the numerator represents the number of pathways in which the accessory pathway potential was recorded, and the denominator indicates the number of pathways accessible to the catheter electrode (number crossed by the catheter).

Figure 6. Electrograms depicting effects of coronary sinus catheter electrode orientation and amplification on recordings of accessory pathway activation. Top two tracings are electrocardiogram lead V1 and a right atrial electrogram (RA). Catheter symbol in the left margin indicates the electrogram was recorded from the coronary sinus with an orthogonal electrode catheter, and the solid electrode symbols identify the recording electrodes. See text for discussion.
FIGURE 7. Use of atrial extraextrastimuli during atrioventricular reentrant tachycardia to dissociate the retrograde accessory pathway (AP) potential from atrial activation in a patient with a left lateral accessory atroventricular pathway. Top Panel: Retrograde AP potential was recorded earliest in the distal orthogonal electrogram (bottom tracing, left arrow) and latest in the proximal orthogonal electrogram (third tracing, left arrow), whereas retrograde atrial activation (A) was recorded earliest from the proximal electrode and latest from the distal electrode (right arrows). Two extrastimuli ($S_2$ and $S_3$) were delivered to the right atrium (RA). The second extrastimulus ($S_3$) advanced the timing of atrial activation in the proximal orthogonal electrogram without altering the timing of the retrograde AP potential (ventricular (V)-AP remains fixed at 60 msec). Atrial potential was masked by the larger AP potential, and the open arrow marks the time the retrograde atrial potential would have occurred in the absence of $S_3$. Middle Panel: Earlier $S_3$ advanced the timing of atrial activation in all three orthogonal electrograms. In the proximal electrogram, recorded near the atrial insertion of the accessory pathway, atrial activation was advanced sufficiently (V-A=45 msec) to produce antegrade activation of the accessory pathway (AP). In the middle electrogram, atrial activation was advanced by 10 msec (V-A decreased from 70 to 60 msec) without changing the timing or morphology of the retrograde AP potential (V-AP=50 msec). Lower Panel: Earlier $S_3$ further advanced atrial activation so that the antegrade atrial potential in the middle orthogonal electrocardiogram (V-A=40 msec) preceded the unaffected retrograde AP potential (V-AP=50 msec). In the distal electrogram, the atrial potential was advanced by 20 msec (V-A decreased from 75 to 55 msec) without altering the timing or morphology of the retrograde AP potential (V-AP=40 msec). Ability to advance the atrial potential without affecting the AP potential is strong evidence the AP potential does not result from atrial activation.
sory pathway activation was independent of pathway location (Figure 5).

The effects of electrode orientation and recording gain on identification of AP potentials are illustrated in Figure 6. The fourth tracing from the top was recorded at low gain from an orthogonal electrode in the lateral region of the great cardiac vein. During sinus rhythm, the atrial wavefront propagated quickly through the small recording range of the electrode, producing a single, narrow triphasic atrial potential. Atrial activation was no longer being recorded 20 msec later when the AP potential was inscribed. The result is two discrete potentials. As little as a sixfold increase in gain (bottom tracing) caused the atrial and AP potentials to blend together, making identification of the AP potential difficult. The third tracing from the top was recorded at low gain between one of the same orthogonal electrodes and an electrode 10 mm proximally, which mimicked the conventional electrode configuration. The atrial potential is wider and increased in amplitude, and the accessory pathway potential cannot be discerned.

Examples of the pacing techniques used to dissociate the AP potential from atrial or ventricular activation are illustrated in Figures 7–11.

**Intraoperative Validation of Accessory Pathway Potentials**

During surgery, the orthogonal electrode catheter in the coronary sinus recorded AP potentials from 14 of the 16 left AV accessory pathways. The two unrecorded pathways also could not be recorded during the preoperative study. With the catheter electrode as a guide to both pathway location and timing of the AP potential, the hand-held epicardial

**FIGURE 8.** Electrograms depicting evidence against a ventricular origin for the antegrade and retrograde accessory pathway (AP) potentials in a patient with a left posterolateral accessory pathway. Panel A: During sinus rhythm, the orthogonal electrogram recorded the AP potential 30 msec after the local atrial potential (left). Local ventricular potential (V) began 20 msec later and preceded the onset of the delta wave in the surface electrocardiographic leads (long dotted line) by 15 msec. Late diastolic extrasinus (S) delivered at the right ventricular (RV) apex advanced the timing of the local ventricular potential (V) by approximately 20 msec (curved arrow) without altering the timing or morphology of the antegrade AP potential (A-AP remained 30 msec). Panel B: Spontaneous termination of atrioventricular reentrant tachycardia due to retrograde block occurring before accessory pathway activation. Loss of the AP potential (unfilled arrow) without change in the timing or morphology of the local ventricular potential (V) is strong evidence the AP potential does not represent activation of ventricular myocardium. Time scale in Panels A and B are different.
electrode recorded an AP potential (Figure 12) in 10 of the 14 pathways. Typically, the AP potential recorded from the epicardial electrode was lower in amplitude and frequency than the potential recorded from the catheter electrode. Manipulation of the epicardial electrode over the catheter electrode recording the AP potential resulted in transient loss of the AP potential and conduction block in nine of the 14 pathways (Figure 12), including two pathways in which the AP potential was recorded from the catheter electrode but not from the epicardial electrode. Epicardial pressure failed to produce conduction block in the two accessory pathways in which activation was not recorded from the coronary sinus catheter electrode.

**Accessory Pathway Fiber Orientation**

Distances along the coronary sinus during catheter pullback were estimated by interpolating the timing and morphology of the potentials with those recorded from the proximal, middle, and distal electrode groups at some reference position. The resolution of this interpolation technique is estimated at 2–3 mm because at least three (usually four) discrete, nonoverlapping electrogram patterns are recorded from an electrode during catheter pullback before the electrogram mimics the electrogram that was originally recorded, 10 mm proximally (Figure 13). In 32 of the 38 recorded free-wall pathways, the retrograde AP potential was recorded earliest distally (anteriorly) along the coronary sinus.

**FIGURE 9.** Electrograms depicting use of atrial pacing to dissociate the atrial and antegrade accessory pathway (AP) potentials by producing intermittent conduction block at the atrial-accessory pathway interface. Tracings were recorded during right atrial pacing (S-stimulus artifact) at cycle length 480 msec in a patient with a left anterolateral accessory atrioventricular pathway. Solid arrows below orthogonal electrogram in first and third complexes identify the antegrade AP potential. Local ventricular potential began approximately 10 msec after the AP potential and preceded the onset of the delta wave (dotted line) by 15 msec. Loss of preexcitation in second and fourth complexes was associated with loss of the AP potential (open arrow) without change in the timing or morphology of the atrial potential, which shows the AP potential does not represent atrial activation. Because antegrade block occurs at the atrial insertion of the accessory pathway, the accessory pathway was activated retrogradely producing the retrograde AP potential (AP retro).

**FIGURE 10.** Use of atrial pacing to dissociate the antegrade accessory pathway (AP) and ventricular potentials in same patient as Figure 7. Antegrade accessory pathway conduction was present during right atrial pacing at cycle length 350 msec (left). The AP potential (arrows) was recorded 20 msec after the local atrial potential and 30 msec before the onset of the delta wave (left long dotted line). Shortening the atrial pacing cycle length to 340 msec produced antegrade accessory pathway conduction block, which was associated with loss of the delta wave and increase in the interval between the AP potential and onset of ventricular activation (65 msec). Persistence of the AP potential despite the loss of local ventricular activation is strong evidence that the AP potential does not represent ventricular activation.
FIGURE 11. Electrograms depicting use of ventricular extrastimuli to dissociate the antegrade accessory pathway (AP) potential from atrial and ventricular activation. Tracings were recorded during surgery. Orthogonal electrode catheter was inserted into the coronary sinus through a right atriotomy and was positioned with the tip electrode near the atrial insertion of a left lateral accessory pathway. Reference atrial electrogram was recorded from the anterior atria, near the septum (AS). Panel A: Extrastimulus (S) delivered to the left ventricular apex (LV) during sinus rhythm advanced the local ventricular potential (V) by 110 msec without changing the timing or morphology of the AP potential (short arrow), which shows that the AP potential does not result from ventricular activation. Panels B and C: Progressively earlier extrastimuli advanced the AP potential without changing the timing or morphology of the local atrial potential, which shows that the AP potential does not represent atrial activation. The AP potential was advanced 10 msec in Panel B and is superimposed on the end of the local atrial potential. In Panel C, the AP potential was advanced 20 msec, and it was superimposed on the beginning of the atrial potential. Open arrows mark the time the AP potential would have been inscribed in the absence of the ventricular extrastimuli.

The retrograde AP potential propagated proximally (posteriorly) along the coronary sinus, 4–30 mm (median, 14 mm), before giving rise to the earliest retrograde atrial potential (Figures 14 and 15). In the remaining six free-wall pathways, the magnitude and direction of lateral excursion could not be determined. Only the atrial end of the accessory pathway was recorded (long V-AP and short AP-A intervals) in three accessory pathways, and multiple accessory pathway potentials in the other three pathways prevented identification of individual fiber orientation. In the seven left posteroseptal or para-
septal accessory pathways, AP potentials were recorded throughout an 8–18 mm (median, 10 mm) length of the proximal coronary sinus, but the fiber orientation was unclear. In some, the ventricular insertion appeared to be located distal to the atrial insertion as in the free-wall pathways. The length of lateral excursion along the coronary sinus for each of the 39 pathways is plotted as a function of pathway location in Figure 16. There is a trend toward longer lateral excursion in more anterior locations, but this is not statistically significant.

Accessory pathway fiber orientation was also examined at surgery in 10 patients with bidirectional accessory pathway conduction. In all six patients with a single pathway and in one patient with two left AV pathways, earliest retrograde atrial epicardial activation was recorded proximal (posterior) to the site that recorded earliest antegrade ventricular epicardial activation. In three patients, each with two close left free-wall accessory pathways, earliest retrograde atrial activation was recorded distal (anterior) to the site of earliest

**Figure 12.** Intraoperative validation of the accessory pathway (AP) potential. Tracings were recorded during left ventricular (LV) pacing at cycle length 400 msec. Orthogonal catheter (inserted into the coronary sinus through a right atriotomy) was positioned to record the retrograde AP potential from the tip electrode. A hand-held bipolar epicardial electrode (Epi) was placed directly over the catheter tip and similarly recorded the AP potential (arrows). Gentle pressure on the epicardial electrode resulted in loss of the accessory pathway potential in both electrograms (open arrows) and retrograde accessory pathway conduction block, which shows that the accessory pathway is located very close to the epicardial and catheter tip electrodes. Ventricular origin for the AP potential can be excluded by the loss of the AP potential without change in the timing or morphology of the local ventricular potential (V). LA, epicardial electrocardiogram recorded from the left atrial appendage.

**Figure 13.** Electrograms illustrating the resolution of low-gain recordings from an orthogonal electrode. Left panel: Electrograms recorded simultaneously from the middle electrode (top tracing) and distal electrode (bottom tracing) at the initial catheter position. Right panel: Electrograms recorded from the distal electrode as the catheter was slowly withdrawn (bottom to top). Four distinct patterns were recorded before the electrogram simulated the pattern originally recorded from the middle electrode (left panel, top tracing), signifying a pullback of 10 mm. Complete change in pattern as the electrode is withdrawn 3–4 mm, without overlap of potentials between sites, suggests that only events occurring within several millimeters of the electrode are recorded. Same patient as Figures 7 and 10.
antegrade ventricular activation. In all three patients, the two pathways were oriented with the atrial insertion proximal, but the proximal pathway conducted only antegradely, and the distal pathway conducted only retrogradely.

**Discussion**

Closely spaced orthogonal catheter electrodes in the coronary sinus and low-recording gain allowed relatively consistent recording of activation from left AV accessory pathways. Fusion of the atrial and AP potentials (Figure 14, electrode position B), presumably representing the atrial insertion site, was recorded in most patients. However, fusion of the AP and ventricular potentials (Figures 9 and 14, position F) often was not recorded from any location in the coronary sinus. The coronary sinus and great cardiac vein generally lie on the atrial side of the anulus, placing the catheter electrode relatively close to the atrial insertion. Ventricular insertions of left AV accessory pathways are often displaced apically along the epicardial surface and lie outside the field of view of the closely spaced, orthogonal electrode. This limitation is greatest near the coronary sinus ostium, where the vessel is farthest from the anulus. This may account for the inability to record activation from three of 10 left posteroseptal or posterior paraseptal pathways, although differences in the anatomic course of these pathways cannot be excluded.

While tracing activation along the accessory pathway, we found that the majority of left free-wall accessory pathway fibers traverse the AV groove obliquely, with the atrial insertion proximal (posterior) to the ventricular insertion. The oblique course greatly facilitates separation of accessory pathway potentials from potentials generated by the adjacent atrial and ventricular myocardium as shown in Figure 15. If the accessory pathways traversed the AV groove exactly perpendicular to the coronary sinus, the potentials generated by activation of the atrial, accessory pathway, and ventricle would tend to fuse, and distinguishing the individual components would be more difficult. Bipolar electrodes oriented parallel to the coronary sinus are able to record the lateral component of the oblique course. The major limitation is the large, overlapping atrial or ventricular potential during antegrade or retrograde accessory pathway conduction, respectively. The atrial and ventricular potentials can be shortened by very closely spaced electrodes (2 mm
center to center), but the orthogonal orientation is usually more effective. The occurrence of left bundle branch block during AV reentrant tachycardia in patients with left free-wall accessory pathways is associated with an increase in the ventriculo-atrial interval of at least 35 msec. The ventriculo-atrial interval is prolonged as a result of the delay in activation of the left ventricular free wall. An increase in the ventriculo-atrial interval of up to 25 msec has also been reported by Kerr et al in some patients with posteroseptal accessory pathways. These investigators believed their data suggested that the posterior septum was activated by the left bundle branch system. An additional factor may be the oblique fiber course. Although the atrial insertion of a fiber may lie in the left posteroseptal region, the ventricular end of the strand may insert into the left ventricular free wall. Some patients with posteroseptal pathways fail to show an increase in the ventriculo-atrial interval during left bundle branch block. The absence of an increase in the ventriculo-atrial interval may be explained by the observation that, in almost half of the patients with "posteroseptal" accessory pathways, accessory pathway potentials are recorded across the tricuspid anulus rather than from the coronary sinus. The demonstration of significant lateral excursion in left free-wall accessory pathways seems consistent with what is known of the embryological development of the heart at the AV boundary. In the primitive, fused cardiac tube, circumferential rings of histologically distinct specialized tissue have been described that later contribute variously to the cardiac fibrous skeleton and the several divisions of the specialized cardiac conduction system. In the course of looping of the primitive cardiac tube, the common atrial region migrates upward and posteriorly, coming to lie above and behind the primitive ventricle and bulbus cordis. As the right atrium is relatively fixed by continuity with the sinus venous and its attendant vessels, the resultant rotational tension produced by upward movement and bending will tend to center upon the AV ring area (atrioventricular flange). Viable islets of anatomic continuity along with developing left AV groove would then presumably be subject to the effects of torsion upon growth and would exhibit counterclockwise rotation from atrium to ventricle as viewed from the apex. As part of the developmental expansion of the primitive ventricle and bulbus cordis at the time of bulboventricular folding, the previously circumferential myogenic fibers are pulled diagonally toward the vortex, which in turn migrates distally and comes to form the apical
insertion of the bulbospiral and sinospiral muscle bands. This same process of diagonal downward growth may explain apical displacement of the ventricular insertion in many left free-wall accessory pathways. In contrast, the right AV conduit is formed by apposition of the bulbus cordis and primitive right atrium, accompanied by anterior and rightward excursion of the left sided AV ring. Rotational forces are not prominent here; therefore, accessory AV connections arising from and around the right AV boundary may not be expected to course laterally. With respect to septal and paraseptal pathways, many factors such as initial torsion, rightward AV ring migration, central fusion of the AV ring for partitioning of left and right AV conduits, and migration within endocardial cushion tissues may modify both the length and course of accessory pathway fibers in various ways. This latter supposition is supported by the observed heterogeneity of these connections.

Little information is available regarding the influence of anatomic variations on conduction properties of the accessory pathway. Direct recordings of accessory pathway activation may greatly facilitate the study of structural and functional relations by allowing a correlation between sites of conduction block and factors such as pathway location, magnitude of lateral excursion, and the presence of multiple, closely spaced strands or interconnecting networks of fibers. Direct recordings also provide greater accuracy in identifying accessory pathway location, which may be useful in the development of catheter ablative techniques.

Acknowledgment

We acknowledge the invaluable contribution of Will Webster for the innovative and skilled construction of the orthogonal electrode catheters.

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KEY WORDS • catheter electrode • electrophysiology • accessory atrioventricular pathway • preexcitation syndromes • Wolff-Parkinson-White syndrome
New catheter technique for recording left free-wall accessory atrioventricular pathway activation. Identification of pathway fiber orientation.


Circulation. 1988;78:598-611
doi: 10.1161/01.CIR.78.3.598

Circulation is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 1988 American Heart Association, Inc. All rights reserved.
Print ISSN: 0009-7322. Online ISSN: 1524-4539

The online version of this article, along with updated information and services, is located on the World Wide Web at:
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