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Radiofrequency Catheter Ablation of Right Atriofascicular (Mahaim) Accessory Pathways Guided by Accessory Pathway Activation Potentials

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Background Accessory pathways (APs) exhibiting "Mahaim fiber" physiology (antegrade conduction only, long conduction time, and decremental properties) often connect the lateral right atrium to the right bundle branch (right atriofascicular pathways). Potentials from these pathways have not been recorded previously. The purpose of this study was to determine whether AP activation potentials could be recorded from right atriofascicular APs and to determine whether these potentials could be used to localize a site for catheter ablation.

Methods and Results Of 26 consecutive patients referred for catheter ablation of an AP producing a preexcited (antidromic) atrioventricular (AV) reentrant tachycardia having a left bundle branch block pattern with short ventriculoatrial and long AV intervals, 23 (88.5%) were found to have a right atriofascicular AP. During antidromic AV reentrant tachycardia, (1) right atrial extrastimuli (that did not penetrate the AV node) advanced the timing of the next QRS complex, indicating that the AP was connected to the right atrium; (2) earliest antegrade ventricular activation was recorded at the apical right ventricular free wall; and (3) ventricular activation was preceded by activation of the distal right bundle branch, indicating a fascicular insertion or a ventricular insertion close to the terminus of the right bundle branch. A single, discrete, high-frequency AP potential was recorded at the lateral, anterolateral, or posterolateral tricuspid annulus in 22 of the 23 patients 63 ± 12 milliseconds after the local atrial potential

and 83 ± 23 milliseconds before the local ventricular potential during sinus rhythm. The AP potential was also recorded at sites along the right ventricular free wall between the tricuspid annulus and the site of earliest ventricular activation at the apical region. Programmed atrial stimulation and adenosine each produced prolongation of AP conduction time because of an increase in the A-AP interval and Wenckebach block proximal to the AP potential. Radiofrequency current applied at a site recording the AP potential (tricuspid annulus in 19 patients and right ventricular free wall in 3 patients) eliminated AP conduction in all 22 patients. Tachycardia has not recurred in any patient during 18 ± 13 months of follow-up. AP conduction was absent in all 9 patients who had a follow-up electrophysiological study 3.8 ± 1.7 months after ablation.

Conclusions Right atriofascicular APs consist of two components. The proximal component is located at the lateral, anterolateral, or posterolateral tricuspid annulus, does not generate an AP potential recordable by catheter electrodes, and is responsible for the decremental conduction properties. The "distal" component extends from the tricuspid annulus to the distal right bundle branch at the apical right ventricular free wall and generates a large, high-frequency AP potential that accurately identifies a site for ablation. (*Circulation*. 1994;89:2655-2666.)

Key Words • ablation • Wolff-Parkinson-White syndrome • catheters • Mahaim • atriofascicular

Episodes of preexcited tachycardia without preexcitation during sinus rhythm occur in one distinctive variant of the Wolff-Parkinson-White syndrome. The antidromic atrioventricular (AV) reentrant tachycardia has characteristic features, including a QRS complex with a left bundle branch block pattern, a relatively short ventriculoatrial (VA) interval, and a long AV interval. At electrophysiological study, the accessory pathway (AP) is found to conduct only in the

antegrade direction with a long conduction time that increases further at faster atrial paced rates and exhibits Wenckebach periodicity during conduction block. These decremental (or AV node-like) conduction properties, combined with the observation that retrograde conduction occurs only through the AV node, led early investigators to postulate that these pathways originate in the AV node, connecting the AV node and the right ventricle¹⁻⁷ as described histologically by Mahaim and colleagues,^{8,9} and they were referred to as nodoventricular APs or Mahaim fibers. Most of these APs have since been shown to connect the lateral right atrium to the apical right ventricle or right bundle branch (right atriofascicular APs).¹⁰⁻¹³ Although potentials from typical AV APs have been recorded^{14,15} and used successfully to guide radiofrequency catheter ablation,¹⁶⁻¹⁸ potentials from Mahaim fibers have not been recorded previously. The purpose of this study was to determine whether AP activation potentials could be consistently recorded from

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Mahaim fibers and whether these potentials could be used to localize a site for ablation of these fibers.

Methods

Study Population

The study population consisted of 26 consecutive patients referred for catheter ablation of an AP producing a preexcited tachycardia having a left bundle branch block pattern with short VA and long AV intervals and were found to have an AP exhibiting only antegrade conduction, with long antegrade conduction time and decremental conduction properties. There were 11 men and 15 women, ranging in age from 17 to 58 years (mean, 29.7 ± 9.6 years). One had Ebstein's anomaly; none of the others had underlying cardiac disease. Thirteen of the 26 patients (50%) had previously undergone one or two unsuccessful catheter ablation procedures before referral to our institution. One of these 13 patients (patient 12) had right bundle branch block after DC ablation at the right ventricular apex. Another patient (patient 21) had undergone ablation of the fast AV nodal pathway, resulting in ventricular preexcitation during sinus rhythm. Ventricular preexcitation was not apparent during sinus rhythm in any other patient.

Electrophysiological Study

After written informed consent was obtained, each patient was studied in the postabsorptive state under heavy sedation with fentanyl (25 to 100 mg/h) and midazolam (1 to 4 mg/h). Oxygen saturation was monitored continuously by a pulse oximeter. Five multipolar electrode catheters were inserted percutaneously into the right subclavian vein and right and left femoral veins and were used for programmed atrial and ventricular stimulation and localization of the AP as described below.

Localization of the Distal Insertion of the AP

One deflectable octapolar catheter (2-mm spacing) was positioned to record activation of the His bundle and proximal right bundle branch (Fig 1A and 1B). A second deflectable octapolar or hexapolar catheter was positioned along the right ventricular septum to record activation of the middle portion of the right bundle branch. A deflectable quadripolar electrode catheter (2-mm/5-mm/2-mm spacing) was used to record activation of the distal right bundle branch at the apical region of the right ventricular septum and was then used to map the right ventricle. Right ventricular mapping during fully preexcited complexes (atrial pacing or antidromic AV reentrant tachycardia) identified the site of earliest right ventricular activation and the timing and sequence of activation of the right bundle branch.

Participation of the Right Atrium in the Tachycardia Reentrant Circuit

Participation of the right atrium as an essential component of the reentrant circuit was determined by delivering extrastimuli to the right atrial appendage or lateral right atrium adjacent to the tricuspid annulus during antidromic AV reentrant tachycardia.¹¹ The extrastimuli were introduced sufficiently late to prevent the paced atrial wave front from arriving at the anterior atrial septum (close to the His bundle) or the posterior atrial septum (near the coronary sinus ostium) before retrograde atrial activation was recorded at these sites, indicating that the atrial extrasystole did not penetrate the anterior or posterior inputs to the AV node. Participation of the right atrium in the reentrant circuit was confirmed if the late atrial extrastimulus advanced the timing of the next QRS complex (without changing QRS morphology or ventricular activation sequence) and reset the tachycardia. This finding also confirms the presence of an AP with a right atrial connection. A nodoventricular or nodofascicular pathway would be suggested if the atria could be dissociated from the

tachycardia, either by the inability of the atrial extrastimuli to advance the next QRS complex without penetrating the AV node or by the occurrence of VA block during tachycardia.¹³

Localization of the Atrial End of the AP

Since the right atriofascicular APs did not exhibit retrograde conduction, the approximate location of the atrial end of the AP was determined in the first 5 patients by a form of atrial pace mapping.^{19,20} Late atrial extrastimuli were delivered during antidromic AV reentrant tachycardia (at the same time relative to the QRS complex) at different sites in 2- to 5-mm increments along the atrial margin of the tricuspid annulus. The pacing site at which the extrastimulus caused the greatest advance in the timing of the next tachycardia QRS complex was considered the approximate location of the atrial insertion of the AP. A 7F deflectable quadripolar electrode catheter (2-mm/5-mm/2-mm interelectrode spacing) was inserted through the right subclavian and/or femoral venous sheath and maneuvered along that region of the tricuspid annulus (either above or below the tricuspid leaflet) in an attempt to record an AP activation potential. A potential was considered to represent AP activation if the interval between the AP potential and ventricular activation (AP-V interval) remained constant during AV delay produced by programmed atrial stimulation and adenosine administration and if the loss of the AP potential (during programmed atrial stimulation, adenosine administration, or catheter trauma) was associated with conduction block over the AP.

In the final 21 patients, pace mapping was not performed. The atrial end of the AP was localized exclusively by mapping the tricuspid annulus during sinus rhythm or atrial pacing and locating the site generating the AP potential. In 11 of the last 12 patients, the tricuspid annulus was initially mapped by use of a 7F deflectable catheter with 10 close bipolar (2-mm) electrode pairs (Halo catheter, Webster Laboratories). This catheter was inserted through a right femoral venous sheath and positioned around the tricuspid annulus in the counterclockwise direction as viewed in the left anterior oblique projection. The tip of the catheter was placed in the proximal coronary sinus. The catheter was initially positioned along the atrial aspect of the tricuspid annulus and then maneuvered to the ventricular side of the annulus.

All recordings used close bipolar electrodes (2-mm) at relatively low gain (0.5 cm/mV). A filter bandwidth of 30 to 500 Hz was used. The electrograms were recorded on either a Honeywell E for M VR-16 analog recorder or a Bard Lab-System with digital amplifiers.

AP Ablation

For ablation of the AP, a 7F deflectable quadripolar electrode catheter with a 4-mm tip electrode (either cylindrical or grooved²¹) was inserted through the right subclavian or a right femoral venous sheath. The large-tip electrode on the mapping catheter was maneuvered to the site along the tricuspid annulus (either above or below the tricuspid leaflet) at which the distal electrode pair recorded a distinct AP potential. Radiofrequency current (continuous wave, 550 to 750 kHz) was delivered between the large-tip electrode and two standard adhesive electrosurgical dispersive pads applied to the chest wall.²² Radiofrequency current was applied at 45 to 60 V when the ablation electrode was positioned beneath the leaflet and at 50 to 70 V when the electrode was positioned above the leaflet by use of the grooved-tip electrode. Typically, current was applied at lower levels initially and increased in 5-V increments every 10 to 20 seconds. Root mean square (RMS) voltage, current, and impedance were monitored continuously. Energy was applied for 60 to 90 seconds but was terminated immediately in the event of an impedance rise or displacement of the catheter electrode. Energy was usually applied during right atrial pacing to observe the loss of conduction in the AP.

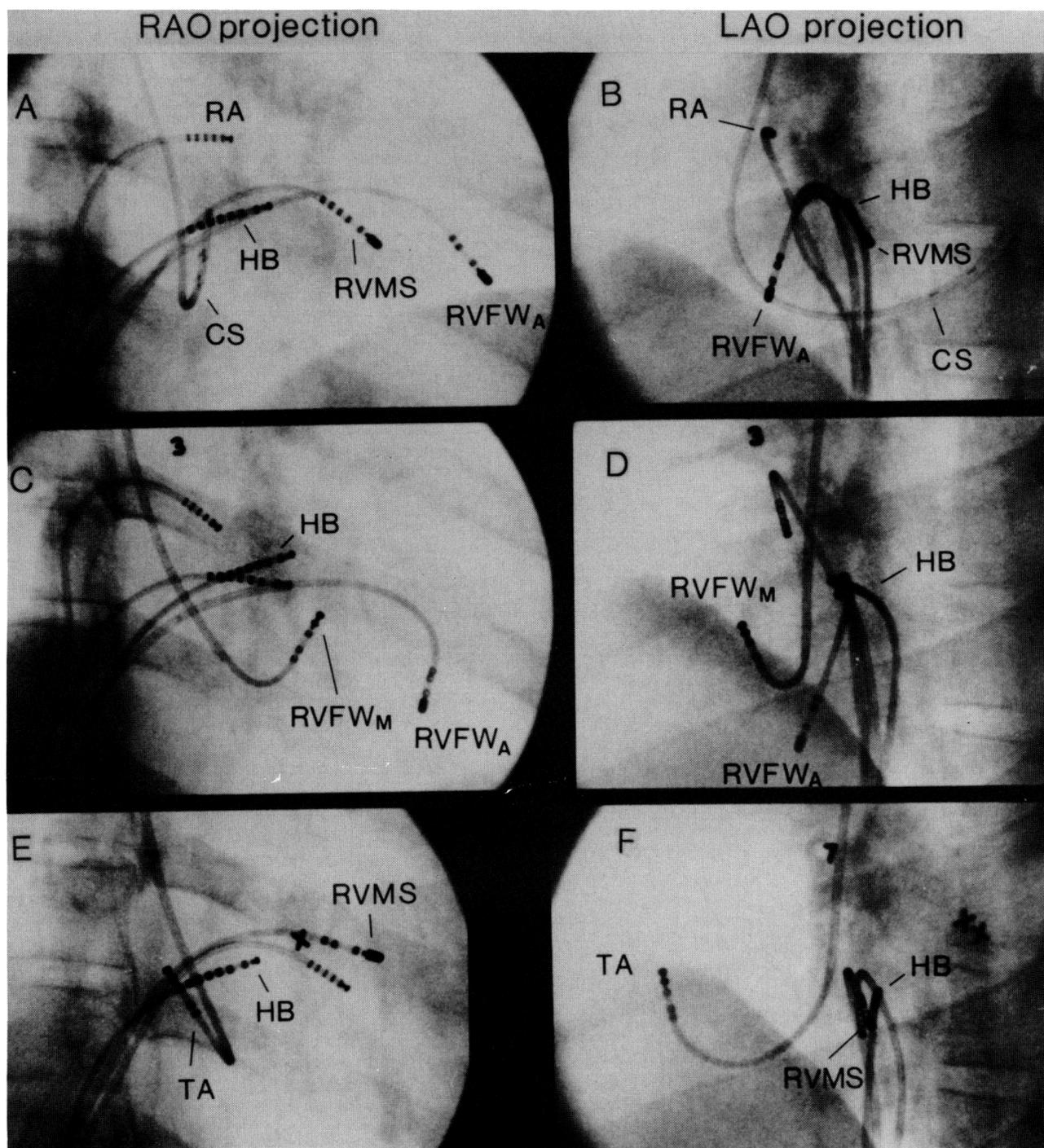


Fig 1. Radiographs in the right anterior oblique (RAO) and left anterior oblique (LAO) projections demonstrating electrode catheters positioned to record activation of the distal, middle, and proximal portion of a right atriofascicular accessory pathway (AP) in patient 6 (electrograms shown in Fig 2). A and B, Electrode catheters were positioned in the apical region of the right ventricular free wall (RVFW_A) at the site of earliest ventricular activation during fully preexcited QRS complexes, the right atrium (RA), His bundle region (HB), coronary sinus (CS), and right ventricular midseptal region (RVMS) to record activation of the mid right bundle branch (Fig 2A and 2B). C and D, A mapping catheter inserted through the right subclavian vein was positioned along the mid right ventricular free wall (RVFW_M) to record activation from the middle of the right atriofascicular AP (Fig 2C). E and F, The subclavian mapping catheter was positioned against the lateral tricuspid annulus (TA) beneath the tricuspid leaflet and recorded activation of the proximal portion of the right atriofascicular AP (Fig 2D).

Beginning 30 to 60 minutes after the final application of energy, programmed atrial and ventricular stimulation was performed with and without isoproterenol infusion (1 to 4 $\mu\text{g}/\text{min}$) to confirm the absence of AP conduction and AV reentrant tachycardia.

Postablation Management

Patients were monitored in an ambulatory unit and discharged on the second day after ablation. Transesophageal echocardiography was performed 18 to 72 hours after ablation.

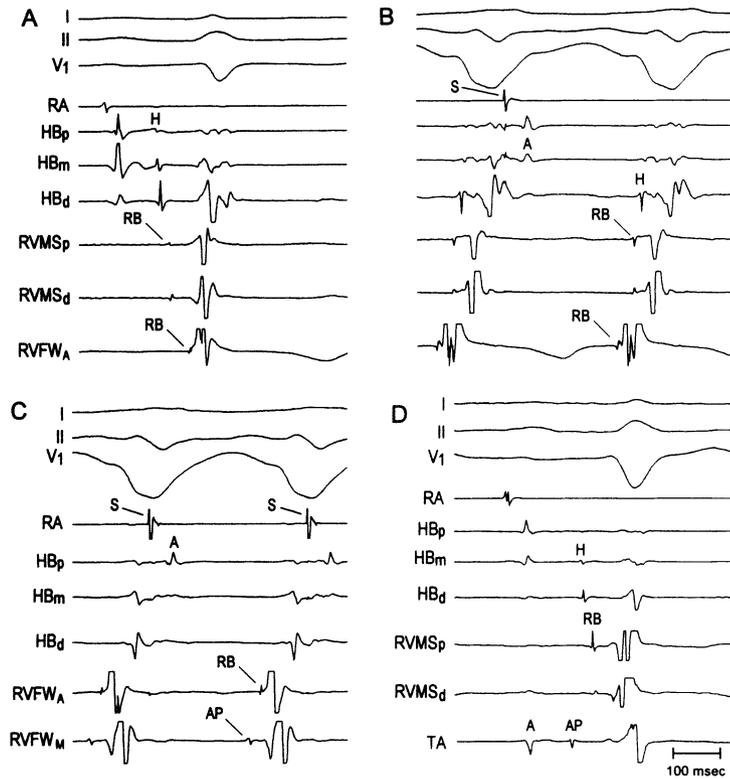


Fig 2. Recordings from the distal, middle, and proximal components of a right atriofascicular accessory pathway (AP) in patient 6. Electrode catheter positions for panels A and B are shown in Fig 1A and 1B. A, During sinus rhythm, right bundle branch activation (RB) precedes ventricular activation at the apical right ventricular free wall (RVFW_A). The His bundle (HB_p, HB_m, HB_d) and right bundle branch are activated antegradely. B, During right atrial (RA) pacing with fully preexcited QRS complexes, right ventricular endocardial mapping identified earliest ventricular activation at the RVFW_A. Ventricular activation was preceded by a distinct high-frequency potential (RB) representing activation of the ventricular end of the AP or the distal end of the right bundle branch. The right bundle (RB) and His bundle (H) were activated retrogradely. These findings suggest that the AP may insert into the distal right bundle branch and that ventricular myocardium may be activated secondarily from the right bundle branch. C, During atrial pacing (selective conduction over the AP), recordings from the distal pair of electrodes on the subclavian mapping catheter positioned against the mid right ventricular free wall (RVFW_M) record activation from the midsection of the right atriofascicular AP. Local ventricular activation occurs later at this site than at the ventricular insertion of the AP at the RVFW_A. Electrode catheter positions are shown in Fig 1C and 1D. D, Recording during sinus rhythm from the distal pair of electrodes on the subclavian mapping catheter positioned against the lateral tricuspid annulus (TA) beneath the tricuspid leaflet. The TA electrogram reveals distinct atrial (A), AP, and ventricular potentials that are separated by isoelectric intervals, producing a pattern very similar to a His bundle electrogram. Electrode catheter positions are shown in Fig 1E and 1F. RVMS_p and RVMS_d indicate proximal and distal right ventricular midseptal regions, respectively.

Patient follow-up was performed by the investigators or by the referring physician. No patient received antiarrhythmic drug therapy after ablation. A follow-up electrophysiological study 2 to 3 months after ablation was recommended. Final follow-up information was obtained by telephone interview with the patient.

Results

Localization of the Distal Insertion of the AP

In 23 of the 26 patients, right ventricular mapping during antegrade AP conduction (ventricular preexcitation) identified earliest ventricular activation at the apical third of the right ventricular free wall, close to the insertion of the distal segment of the right bundle branch (moderator band) and far from the tricuspid annulus (Fig 1A and 1B). The earliest ventricular potential was preceded at that site by a distinct high-frequency potential (Fig 2B). During sinus rhythm, the same potential preceded ventricular activation (Fig 2A), suggesting that this potential represented activation of a distal segment of the right bundle branch. During fully preexcited QRS complexes, the entire length of the right bundle branch and the His bundle were activated retrogradely, and activation of the right bundle branch preceded the onset of the QRS complex (Fig 2B). These observations suggest that the AP may have inserted into the distal right bundle branch and that the ventricular myocardium may have been activated secondarily by the right bundle branch. These patients were considered to have a right atriofascicular AP.

In 9 of these 23 patients, transient right bundle branch block was produced by inadvertent or inten-

tional catheter trauma to the right bundle branch at various sites along the septum, including the apex. In one additional patient (patient 12), permanent right bundle branch block had resulted from a previous DC ablation attempt at the right ventricular apex. Right bundle branch block did not block antegrade activation of the right ventricle via the AP in any of these 10 patients but did increase the tachycardia cycle length because of prolongation of the retrograde (VA) conduction time (Fig 3). The increase in VA interval resulted from delay in retrograde activation of the His bundle (increase in VH interval, Table). The failure of block in the septal right bundle branch to interrupt AP conduction indicates that the pathway inserted into the distal right bundle branch at the right ventricular free wall or into the free wall close to the terminus of the right bundle branch.

In 3 of the 26 patients, earliest ventricular activation during fully preexcited QRS complexes was recorded adjacent to the tricuspid annulus, suggesting an AV AP. The ventricular insertion was located close to the posterolateral tricuspid annulus in 2 patients and the posterior tricuspid annulus in 1. During fully preexcited complexes in these 3 patients, ventricular activation at the right ventricular apex followed the onset of the QRS complex by 22 to 38 milliseconds.

The morphology of the fully preexcited QRS complex did not reliably distinguish the three right AV APs from the 23 right atriofascicular APs, but some trends were evident. The initial forces of the QRS complex in lead V₁ were negative, and the downstroke of the QRS in lead V₁ had a plateau or abrupt change in slope in 2 of

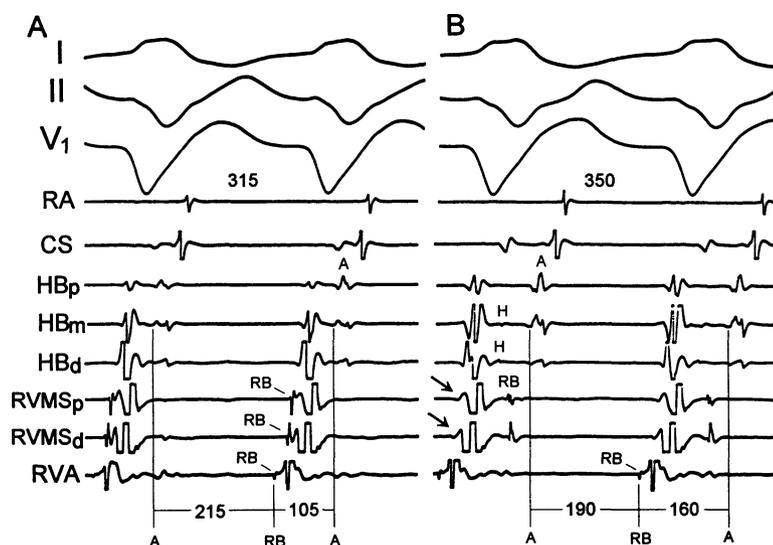


FIG 3. Tracings showing effect of right bundle branch block on right atriofascicular accessory pathway (AP) conduction and antidromic atrioventricular (AV) reentrant tachycardia in patient 4. A, Characteristic features of this tachycardia, including left bundle branch block morphology, retrograde activation of the right bundle branch (RB) preceding ventricular activation, short ventriculoatrial (VA) interval (due to early retrograde activation of the His bundle) and long AV interval (due to long antegrade AP conduction time). B was recorded after manipulation of the right ventricular midseptal (RVMS) catheter, which produced transient right bundle branch block between the mid RVMS and distal right bundle branch recorded at the apical portion of the right ventricular free wall (RVA). Antegrade conduction over the AP was not blocked by block in the mid right bundle branch, and activation of the distal right bundle branch continued to precede earliest ventricular activation (RB potential in RVA electrogram). Retrograde atrial activation (A) is delayed, however (distal RB-A interval is increased to 160 from 105 milliseconds), as a result of delay in the timing of retrograde His bundle activation (H), since the right bundle branch can no longer be used for retrograde conduction. Note that the proximal right bundle branch is activated in the antegrade direction with the right bundle branch potential (RB) in the RVMS electrogram occurring after the retrograde His bundle (H) potential. The increase in VA interval resulted in an increase in the tachycardia cycle length from 315 to 350 milliseconds and a consequent decrease in antegrade AP conduction time (A-RB decreased to 190 from 215 milliseconds).

the 3 patients with AV pathways, whereas lead V_1 showed a small brief positive initial component with a relatively straight downstroke (similar to left bundle branch block) in 22 of the 23 patients with right atriofascicular pathways. The initial forces in lead II were negative in 2 of the 3 patients with right AV pathways, whereas the initial forces were positive or isoelectric in lead II in all but 1 patient with a right atriofascicular pathway. The two types of APs could be readily differentiated by examining the timing of ventricular activation at the right ventricular apex and the timing of retrograde activation of the right bundle branch during fully preexcited complexes, both occurring before the onset of the delta wave in patients with a right atriofascicular AP and both occurring after the onset of the delta wave in patients with a right AV AP.

The remainder of this report is limited to the 23 patients with a right atriofascicular AP.

Induction of AV Reentrant Tachycardia

Antidromic AV reentrant tachycardia was induced in all 23 patients with a right atriofascicular AP. Tachycardia was most commonly induced by short periods of right ventricular pacing, due to retrograde conduction block in the AP at long pacing cycle lengths. In 21 of the 23 patients, the AV node formed the retrograde limb of the reentrant circuit. Retrograde conduction occurred over the fast AV nodal pathway in 17 patients and the slow AV nodal pathway in 4 patients (Table). In the 2 remaining patients, a concealed left anterolateral (patient 1) and a right midseptal (patient 22) accessory AV pathway initially formed the retrograde limb of the reentrant circuit. After ablation of the AP, antidromic

AV reentrant tachycardia remained inducible using the slow AV nodal pathway for retrograde conduction in both patients.

The induction of sustained tachycardia required isoproterenol 0.5 to 1.5 $\mu\text{g}/\text{min}$ to facilitate retrograde AV nodal conduction in 4 patients. In the absence of right bundle branch block (22 patients), the antidromic AV reentrant tachycardia had a cycle length that ranged from 275 to 445 milliseconds (mean, 350 ± 53 milliseconds), with an AV interval of 165 to 355 milliseconds (mean, 241 ± 49 milliseconds), VH interval of 5 to 55 milliseconds (mean, 18 ± 10 milliseconds), and HA interval of 35 to 205 milliseconds (mean, 92 ± 51 milliseconds). In the presence of transient (9 patients) or permanent (1 patient) right bundle branch block, the tachycardia cycle length ranged from 340 to 480 milliseconds (mean, 371 ± 41 milliseconds) with an AV interval of 190 to 255 milliseconds (mean, 213 ± 20 milliseconds), VH interval of 60 to 125 milliseconds (mean, 85 ± 19 milliseconds), and HA interval of 35 to 175 milliseconds (mean, 74 ± 40 milliseconds).

Participation of the Right Atrium in the Tachycardia Reentrant Circuit and Localization of the Atrial End of the Right Atriofascicular AP

During antidromic AV reentrant tachycardia, single late right atrial extrastimuli (that did not penetrate the AV node) advanced the timing of the next tachycardia QRS complex and reset the tachycardia in all 23 patients, indicating that the right atrium formed part of the reentrant circuit. This also indicated that the AP was connected to the right atrium rather than the AV node. Late atrial extrastimuli delivered around the

Conduction Times in AV Reentrant Tachycardia and Sinus Rhythm in Patients With Right Atriofascicular Accessory Pathways

Patient	AVRT				AVRT With Right Bundle Branch Block				Shortest 1:1 AP CL	Sinus Rhythm		
	CL	A-V	V-H	H-A	CL	A-V	V-H	H-A		A-AP	AP-Local V	H-V
1	445	230	25	190	480	195	110	175*	330	65	150	45
2	420	335	15	70	≤420	†	†	55
3	320	250	10	60	340	190	90	60	270	55	110	50
4	315	230	25	60	350	215	75	60	≤320	60	80	50
5	320	240	15	65	310	70	85	50
6	365	205	20	140*	280	80	125	60
7	275	210	15	50	200	40	50	35
8	375	300	25	50	≤375	80	80	45
9	275	185	15	75	340	210	70	60	240	70	75	55
10	340	245	15	80	440	60	80	55
11	275	200	20	55	330	50	90	50
12	‡	‡	‡	‡	360	215	85	60	240	75	75	45
13	405	260	20	125*	355	205	75	75	310	50	75	45
14	360	300	15	45	380	255	80	45	370	70	100	45
15	345	260	10	75	370	85	55	40
16	320	210	10	100	355	190	60	105	250	70	60	55
17	445	355	5	85	370	55	75	45
18	290	220	10	60	270	55	80	20
19	325	250	15	60	360	220	80	60	310	60	75	50
20	345	175	5	165*	330	75	90	50
21	360	165	20	175*	250	50	55	30
Mean±SD	346±52	241±51	16±6	89±45	369±43	211±20	81±14	78±40	314±63	64±12	83±24	46±9

AV indicates atrioventricular; AVRT, antidromic AV reentrant tachycardia; CL, tachycardia cycle length; A-V, interval between the atrial potential recorded in the His bundle electrogram and the onset of ventricular activation; V-H, interval between onset of ventricular activation and retrograde His bundle potential; H-A, interval between retrograde His bundle potential and retrograde atrial potential recorded in the His bundle electrogram; Shortest 1:1 AP CL, shortest atrial pacing cycle length maintaining 1:1 antegrade conduction over the right atriofascicular accessory pathway; A-AP, interval measured from the lateral tricuspid annulus electrogram between the local atrial potential and the accessory pathway (AP) potential; AP-Local V, interval measured from the lateral tricuspid annulus electrogram from the AP potential to the local ventricular potential; and H-V, interval measured from the onset of the His bundle potential to the onset of ventricular activation in any electrogram.

*Slow AV nodal pathway used for retrograde limb of reentrant circuit.

†AP potential not recorded in this patient.

‡Permanent right bundle branch block.

tricuspid annulus (2- to 5-mm intervals) in the first 5 patients identified the approximate location of the atrial insertion of the AP at the lateral or anterolateral tricuspid annulus. Recordings at the tricuspid annulus close to those sites (Fig 1E and 1F) identified a distinct potential, consistent with AP activation (Fig 2D), in 4 of the 5 patients. In the remaining 18 patients, atrial pace mapping was not used. The atrial end of the AP was located exclusively by recording an AP activation potential along the tricuspid annulus (Fig 4).

Recording of Right Atriofascicular AP Activation Potentials

Potentials consistent with AP activation were recorded in 22 of the 23 patients with right atriofascicular APs. An AP origin was confirmed by demonstrating a constant relationship between the AP potential and the

subsequent QRS complex during antidromic AV reentrant tachycardia (Fig 4) and during AP conduction delay produced by programmed atrial stimulation (Fig 5) and adenosine administration (Fig 6). The electrograms recorded at the tricuspid annulus close to the AP were similar in appearance to a His bundle electrogram (Figs 2D and 4 through 6). There was an isoelectric interval between the atrial potential and the AP potential at all sites along the tricuspid annulus, suggesting the presence of a proximal component of the AP that did not generate a potential that could be recorded with conventional catheter electrodes and a "distal" component (beginning at the tricuspid annulus) that generates the AP potential. During sinus rhythm, the AP potential at the tricuspid annulus was recorded 40 to 85 milliseconds (mean, 63 ± 12 milliseconds) after the local atrial potential and 50 to 150 milliseconds (mean, 83 ± 23

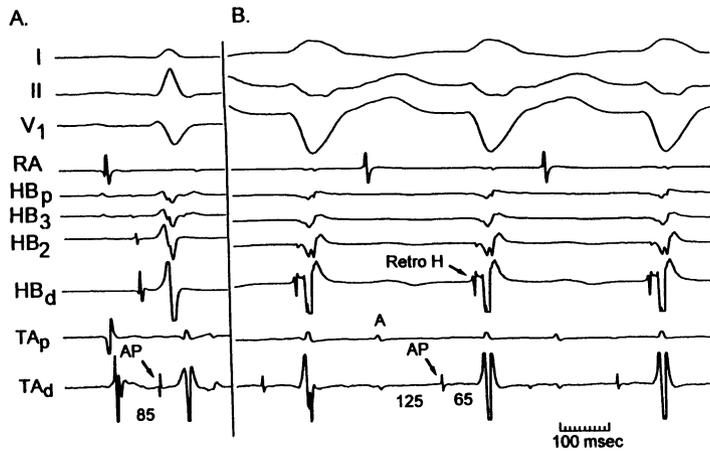


FIG 4. Direct recordings of activation of the proximal end of a right atriofascicular accessory pathway (AP) during sinus rhythm (A) and during antidromic atrioventricular (AV) reentrant tachycardia (B) in patient 15. Recordings from the tricuspid annulus (TA₄) using a 2-mm spaced bipolar electrode show distinct atrial, AP, and ventricular potentials. During sinus rhythm, the AP potential followed the local atrial potential (A-AP interval) by 85 milliseconds. During tachycardia, the A-AP interval increased to 125 milliseconds, and the AP potential preceded the onset of the QRS complex by 65 milliseconds. Note that during tachycardia, the ventricular potential at the tricuspid annulus was recorded 25 milliseconds after the onset of the QRS complex, indicating that ventricular activation began far from the atrial end of the atriofascicular pathway. Retrograde His bundle activation (Retro H) occurs coincidentally with the onset of the QRS complex, which rapidly distinguishes this arrhythmia from antidromic AV reentrant tachycardia using a typical accessory AV pathway for antegrade conduction, whereas retrograde His bundle activation is recorded well after the onset of the QRS complex.

milliseconds) before the local ventricular potential (Table). During fully preexcited QRS complexes, the local ventricular potential at the tricuspid annulus followed the onset of the QRS complex by 10 to 38 milliseconds (mean, 21 ± 7 milliseconds), consistent with earliest ventricular activation occurring far from the tricuspid annulus at the apical region of the right ventricular free wall.

In 20 patients, the AP potential was recorded at one or more sites along the right ventricular free wall, between the tricuspid annulus and the distal insertion near the right ventricular apex. At progressively more apical sites, the AP potential was recorded later, whereas the local ventricular potential was recorded earlier (Figs 1 and 2). This suggests a long AP extending between the tricuspid annulus and the right ventricular free wall close to the apex, without intermediate connections to the right ventricle.

Site of Antegrade Conduction Block in Right Atriofascicular APs

Recordings of AP activation during programmed atrial stimulation or AV reentrant tachycardia in 17 patients demonstrated that prolongation of the antegrade AP conduction time resulted from a delay in the onset of the AP potential at the tricuspid annulus (increase in A-AP interval), whereas the interval between the AP potential and the QRS complex of fully preexcited complexes remained relatively constant (Fig 5). Stable recordings of AP potentials were obtained during pacing-induced AP conduction block in 9 patients; block consistently occurred proximal to the AP potential (Fig 5D). AP recordings were obtained after adenosine (6 to 12 mg IV) was administered during AV reentrant tachycardia or during atrial pacing in 9 patients. AP conduction delay and then block occurred proximal to the AP potential in all 9 patients (Fig 6).

Catheter-Induced AP Extrasystoles and Conduction Block in Right Atriofascicular APs

AP extrasystoles were observed at electrophysiological study in 5 patients. The QRS complexes were identical to the fully preexcited complexes and were preceded by an AP potential with similar AP-V interval

(Fig 7). In 3 patients, the extrasystoles appear to have been produced by catheter contact with the AP, since extrasystoles with this morphology were observed only when the mapping catheter was close enough to the AP to record the AP potential (Fig 7). In the other 2 patients, spontaneous AP extrasystoles were also present on monitor recordings before electrophysiological study.

Conduction block over the right atriofascicular AP was produced by light catheter trauma in 13 of the 23 patients (57%) when the mapping catheter was positioned to record the AP potential close to the tricuspid annulus or along the basal half of the right ventricular free wall. Loss of AP conduction was consistently associated with loss of the AP potential at the block site and apical to that site (Fig 8A) but not proximal to the block site (Fig 8B). In 8 of the 13 patients, AP conduction block was transient. Recurrence of AP conduction was associated with the return of the AP potential. In the remaining 5 patients, AP conduction block persisted through the remainder of the procedure, and ablation was targeted at the AP potential recorded proximal to the block site (Fig 8B).

Radiofrequency Catheter Ablation of Right Atriofascicular APs

The right atriofascicular AP was successfully ablated in a single session in all 23 patients. The successful application of radiofrequency current was delivered close to the tricuspid annulus in 20 patients and at the right ventricular free wall (one fourth to one third of the distance from the tricuspid annulus to the apex) in 3 patients. Radiofrequency current was applied during right atrial pacing or antidromic AV reentrant tachycardia to verify the loss of AP conduction (Fig 9). AP potentials were recorded and used to guide ablation in 22 of the 23 patients. In the 17 patients with AP potentials recorded and intact AP conduction (not blocked by catheter trauma), AP conduction was eliminated by 1 to 9 (median, 2; mean, 3.2 ± 2.9) applications of radiofrequency current. Only 1 application was required in 8 patients (including 6 of the last 7 patients). In the single patient (patient 2) in whom an AP potential was not recorded, radiofrequency current was

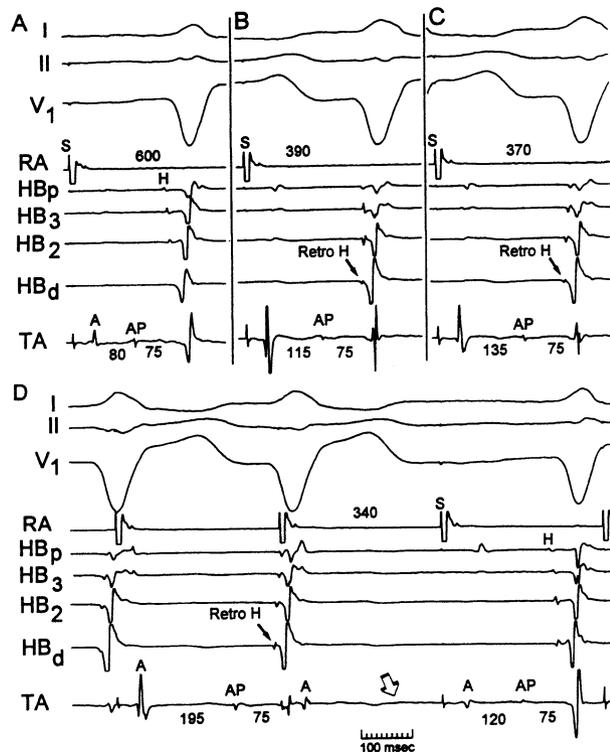


FIG 5. Tracings showing site of conduction delay over a right atriofascicular accessory pathway (AP) during decremental atrial pacing in patient 14. A, During right atrial pacing at cycle length 600 milliseconds, the electrogram recorded at the tricuspid annulus (TA) showed a distinct high-frequency AP potential beginning 80 milliseconds after the local atrial potential (A) and 75 milliseconds before the onset of the QRS complex. B and C, As the atrial pacing cycle length was shortened to 390 milliseconds and subsequently 370 milliseconds, the A-AP interval increased progressively to 115 and 135 milliseconds, respectively, while the AP-QRS interval remained constant at 75 milliseconds. D, At atrial pacing cycle length of 340 milliseconds, Wenckebach block in AP conduction occurred proximal to the AP potential. The markedly prolonged A-AP interval of 195 milliseconds was followed by AP conduction block (open arrow marks the absent AP potential). The A-AP interval shortened to 120 milliseconds after the blocked impulse. The AP-QRS interval remained fixed at 75 milliseconds. The constant AP-QRS interval provides strong evidence for an AP origin for the AP potential. S indicates stimulus; A, atrial activation; and Retro H, retrograde His bundle activation.

applied to the tricuspid annulus close to the atrial insertion of the AP as indicated by atrial pace mapping. Eight applications of radiofrequency current were required to eliminate AP conduction in that patient. The successful application of radiofrequency current was delivered for a mean of 51 ± 38 seconds at a voltage of 65 ± 0 V, current of 0.69 ± 0.11 A, and power of 46 ± 14 W. Extrasystoles or a brief accelerated rhythm that probably originated in the AP (QRS morphology identical to fully preexcited QRS complexes, early ventricular activation at the right ventricular apex, and early retrograde activation of the right bundle branch and His bundle) occurred at the onset of the successful application of radiofrequency current in 11 patients (Fig 9). In 5 of the 22 patients with AP potentials, catheter trauma during mapping eliminated AP conduction. Three to seven (mean, 4.8 ± 1.6) applications of radiofrequency current were delivered to the mapping site, which

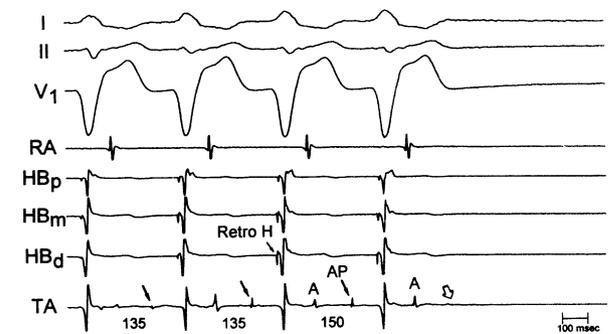


FIG 6. Tracings showing site of accessory pathway (AP) conduction block during adenosine administration in patient 14. Adenosine (12 mg IV) was administered during antidromic atrioventricular reentrant tachycardia. Recordings from the tricuspid annulus (TA) close to the AP show that adenosine resulted in prolongation of the atrial-AP interval from 135 to 150 milliseconds, followed by block proximal to the AP potential (open arrow marks loss of AP potential), terminating tachycardia. Retro H indicates retrograde His bundle activation.

resulted in loss of AP conduction, and to sites proximal to the block, which continued to generate an AP potential (Fig 8B).

Of the 20 atriofascicular APs that were ablated close to the tricuspid annulus, radiofrequency current was delivered below the tricuspid leaflet via the right subclavian venous approach in 7 patients, above the tricuspid leaflet via the subclavian approach in 7 patients, above the tricuspid leaflet via the femoral approach in 5 patients (Fig 10), and below the leaflet via the femoral approach in 1 patient. The most stable ablation electrode position was obtained by the right subclavian venous approach, maneuvering the 4-mm-tip electrode beneath the tricuspid leaflet high against the annulus (Fig 1E and 1F). A somewhat less stable but more readily attained subclavian approach was to position the electrode above the tricuspid leaflet, either by forming a loop in the right atrium and advancing the loop into the ventricle until the tip electrode rested on the lateral tricuspid annulus or by positioning the tip of the catheter directly against the annulus without forming a loop. The femoral approach was better suited to patients with a small heart (Fig 10), whereas the subclavian approach was more effective in patients with a large heart. In the

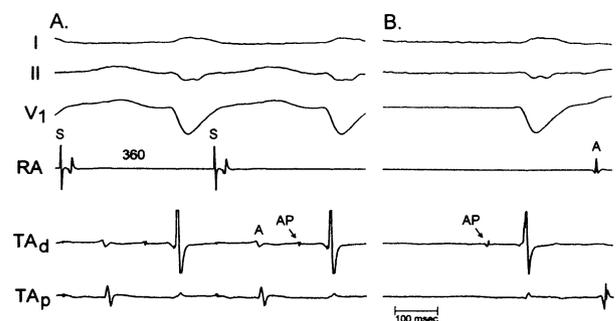


FIG 7. Tracings showing a right atriofascicular accessory pathway (AP) extrasystole in patient 13. A, Right atrial pacing at cycle length of 360 milliseconds resulted in fully preexcited QRS complexes preceded by an AP potential. B, An AP extrasystole (QRS morphology identical to the preexcited QRS complex and preceded by a distinct AP potential) occurred 570 milliseconds after termination of atrial pacing. A indicates atrial; S, stimulus.

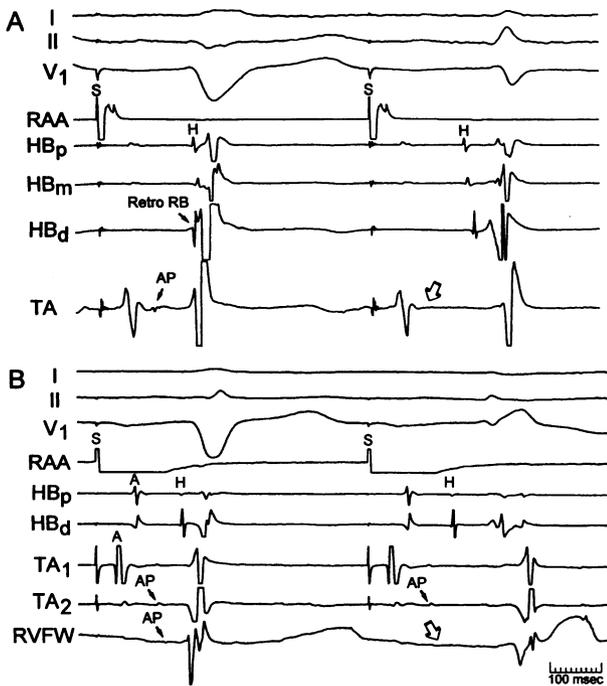


Fig 8. Tracings showing transient conduction block in two right atriofascicular accessory pathways (APs) due to catheter trauma, resulting in loss of the AP potential at and distal to the site of block. Both panels were recorded during right atrial pacing, which was used to enhance preexcitation during mapping. A, Manipulation of the mapping catheter close to the tricuspid annulus (TA) was associated with the loss of the AP potential (open arrow) and simultaneous loss of ventricular preexcitation (patient 13). B, In patient 12, a "Halo" catheter was positioned around the tricuspid annulus (TA₁ and TA₂) to record the AP potential and provide a reference for the ablation catheter. Manipulation of the ablation catheter at the lateral right ventricular free wall (RVFW) resulted in AP conduction block with loss of the AP potential in the RVFW electrogram (open arrow). Note that the AP potential was still recorded at TA₁ and TA₂ proximal to the site of block. AP conduction did not return, and ablation was directed at the AP potential recorded at the tricuspid annulus. S indicates stimulus; H, His bundle; A, atrial; and Retro RB, retrograde right bundle branch activation.

3 patients in whom the AP was ablated along the right ventricular free wall, the femoral approach was used in 2 patients and the subclavian approach in the other.

Neither AV reentrant tachycardia nor even single ventricular echo complexes were induced by programmed atrial or ventricular stimulation after ablation, either in the baseline state or during isoproterenol administration (0.5 to 4 µg/min) in any of the 23 patients. The location around the tricuspid annulus of the successful ablation site or the site recording the AP potential is illustrated in Fig 11.

One patient also had a concealed left anterolateral accessory AV pathway. This pathway was successfully ablated by two applications of radiofrequency current delivered to the mitral annulus, beneath the mitral leaflet, via the retrograde approach.¹⁶ One patient also had a right midseptal accessory AV pathway, which was successfully ablated by a single application of radiofrequency current delivered to the ventricular side of the tricuspid annulus just anterior to the anterior margin of the coronary sinus ostium.

Sustained AV nodal reentrant tachycardia (slow pathway antegrade/fast pathway retrograde) was induced by programmed atrial stimulation before and after ablation of the right atriofascicular pathway in 2 patients and, in 1 additional patient, nonsustained episodes (up to 24 beats) were induced during isoproterenol administration. AV nodal reentrant tachycardia was eliminated in each of the 2 former patients by a single application of radiofrequency current delivered to the posteroseptal right atrium between the coronary sinus ostium and the tricuspid annulus.²³ Slow pathway ablation was not attempted in the third patient, who had only nonsustained episodes of AV nodal reentrant tachycardia during isoproterenol administration.

The mean fluoroscopy time for the procedure was 99±39 minutes. Pulsed fluoroscopy at 7.5 pulses per second (25% of the radiation exposure of continuous fluoroscopy) was used in the last 11 patients.

Complications

There were no acute complications. Transesophageal echocardiography was performed on the day after the procedure in 21 of the 23 patients, and transthoracic echocardiography was performed in the remaining 2 patients. Transesophageal echocardiography identified a small thrombus (3×7 mm) in the superior vena cava, just above the right atrial junction, presumably from

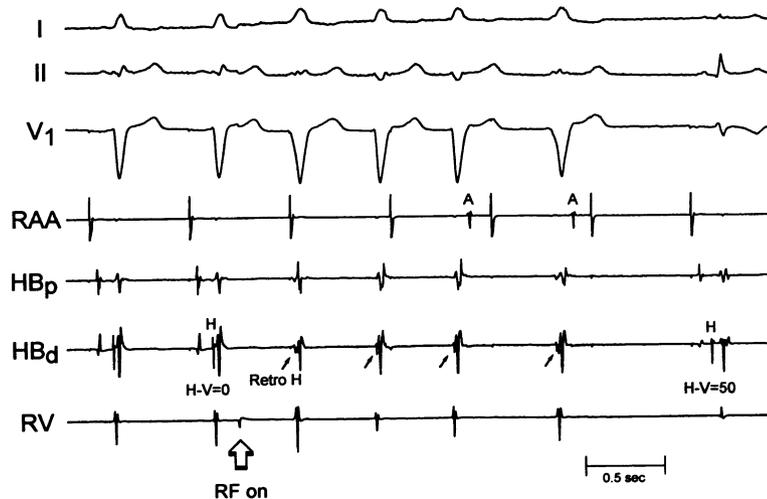


Fig 9. Tracings showing radiofrequency (RF) catheter ablation of the right atriofascicular accessory pathway (AP) in patient 18. Radiofrequency current (53 V, 0.56 A, 30 W) was applied to the lateral tricuspid annulus (beneath the tricuspid leaflet) during right atrial pacing (open arrow, RF on). An accelerated rhythm (four complexes) began immediately after the onset of energy delivery. The QRS morphology of the accelerated complexes was identical to fully preexcited complexes, with early ventricular activation at the right ventricular apex (RV) and early retrograde His bundle activation (Retro H), suggesting an automatic rhythm originating from the AP produced by RF energy. After the accelerated rhythm, antegrade conduction over the AP was absent.

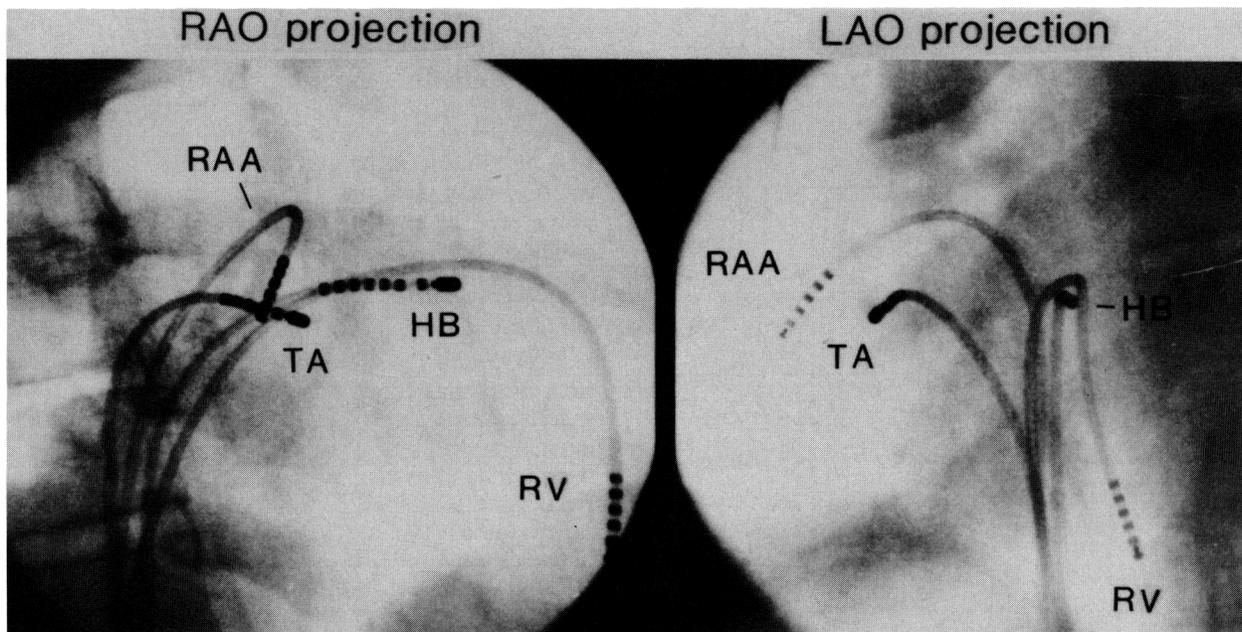


FIG 10. Radiographs in the right anterior oblique (RAO) and left anterior oblique (LAO) projections during ablation of a right atriofascicular accessory pathway via the femoral venous approach. The ablation catheter with large-tip electrode was advanced from the right femoral vein and positioned at the lateral tricuspid annulus above the valve leaflet. TA indicates tricuspid annulus; HB, His bundle; and RV, right ventricular free wall.

trauma associated with manipulation of the right subclavian venous catheter. This was treated with a short course of warfarin. No intracardiac thrombi, pericardial effusion, or other abnormality was identified by transesophageal echocardiography with the exception of Ebstein's anomaly, known to be preexisting in one patient.

Clinical Follow-up

Long-term follow-up ranged from 1.4 to 43 months (mean, 18 ± 13 months). There were no further episodes of tachycardia, and none of the patients received antiarrhythmic therapy. Nine patients (including 3 who had catheter-induced AP conduction block before ablation) underwent electrophysiological study 1.9 to 7.1 months (mean, 3.8 ± 1.7 months) after ablation. These studies

confirmed the absence of AP conduction and AV reentrant tachycardia in all 9 patients.

Discussion

The initial study population consisted of 26 patients with "Mahaim fiber" physiology, in which the AP exhibited conduction only in the antegrade direction with long conduction times and decremental conduction properties. Each of the patients presented with antidromic (preexcited) AV reentrant tachycardia, in which the QRS complex exhibited a left bundle branch block pattern. In all 26 patients, the AP originated from the right atrium rather than the AV node. In 3 patients, the AP inserted directly into the ventricle, adjacent to the tricuspid annulus; these patients were considered to have an AV AP. The ventricular insertion of these three AV pathways was located adjacent to the posterolateral or posterior tricuspid annulus (Fig 11). In 23 patients (88%), antegrade conduction over the AP resulted in earliest ventricular activation at the apical third of the right ventricular free wall with early retrograde activation of the right bundle branch. The earliest ventricular potential during fully preexcited complexes was preceded by a short high-frequency potential (Figs 2B, 2C, and 3). This high-frequency potential also preceded the local ventricular potential during sinus rhythm, when conduction occurred exclusively over the normal conduction system (Fig 2A), suggesting that the potential represented activation of a distal segment of the right bundle branch. Catheter-induced right bundle branch block at any site along the septum, including close to the apex, failed to affect antegrade conduction over the AP, suggesting that the AP inserted into the distal right bundle branch at the apical region of the right ventricular free wall (moderator band). These observations are consistent with the hypotheses of earlier investigators suggesting that these APs may insert directly into the

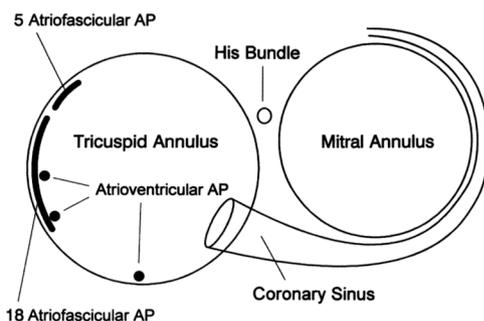


FIG 11. Schematic representation of the tricuspid annulus and mitral annulus, as viewed in the left anterior oblique projection, illustrating the location along the tricuspid annulus of the successful ablation site or, in the five patients in whom accessory pathway (AP) conduction block occurred from catheter manipulation before ablation, the site recording the AP potential. All of the atriofascicular APs were located at the lateral, anterolateral, or posterolateral tricuspid annulus. The AV APs were located at the lateral or posterior tricuspid annulus.

right bundle branch (right atriofascicular APs) and that the right ventricle may be activated secondarily.¹¹

A distinct AP activation potential was recorded close to the lateral, anterolateral, or posterolateral tricuspid annulus in 22 of 23 patients with right atriofascicular pathways. The AP potential was a high-frequency potential of short duration, similar to that recorded from the His bundle or right bundle branch (Figs 2D and 4 through 8). An isoelectric interval separated the local atrial potential and the AP potential (mean A-AP interval, 63 milliseconds). Conduction delay and block produced by programmed atrial stimulation and adenosine administration occurred between the atrial and AP potentials (Figs 5 and 6), suggesting a proximal component that is responsible for the decremental conduction properties of right atriofascicular APs but does not generate an AP potential and a “distal” component that generates the AP potential.

The AP potential was also recorded between the tricuspid annulus and the apical portion of the right ventricular free wall. Earliest ventricular activation was recorded at the apical region of the right ventricular free wall, suggesting that the “distal” component of the AP was a single, long fiber without an intermediate insertion into the right ventricle, similar to the right bundle branch. As with the right bundle branch, light pressure from the recording electrodes resulted in loss of the AP activation potential and AP conduction (Fig 8).

These observations are consistent with the hypothesis that right atriofascicular APs represent an “accessory” AV conduction system consisting of a proximal component (similar to the AV node) positioned at or above the tricuspid annulus, which connects to a “distal” component (similar to the right bundle branch) that generates the AP potential. The right bundle branch–like component courses along the endocardial surface of the right ventricular free wall from the tricuspid annulus to the apical region (Fig 12). This fiber may insert into the apical portion of the right ventricular free wall, close to the distal components of the right bundle branch (Fig 12A), or fuse with the distal right bundle branch (Fig 12B). The concept that right atriofascicular APs may represent an “accessory” AV conduction system is supported by the observations of Guiraudon and coworkers.²⁴ Histological examination of a block of right atrium encompassing a right atriofascicular AP showed a node of cells morphologically similar to the cell types generally seen in and around the AV node, including transitional cells, nodal cells, and P cells. Kent's description^{25,26} of a collection of specialized conduction fibers at the anterolateral tricuspid annulus may be more consistent with the AV node–like proximal portion of a right atriofascicular pathway than the common type of AP, which is often referred to as a “Kent bundle.” More recently, Becker and Anderson and coworkers^{27,28} and Bharati and Lev and coworkers^{29,30} have described AV node–like APs at the anterolateral tricuspid annulus that might have been right atriofascicular APs.

None of the 26 patients in this study had an AP extending from the AV node to the right bundle branch or right ventricle. The AP originated from the right atrial free wall in all patients. This is consistent with the findings of earlier studies of surgical ablation of “Mahaim fibers” reported by Gillette et al¹⁰ and Klein et al.¹² These observations do not exclude the existence of

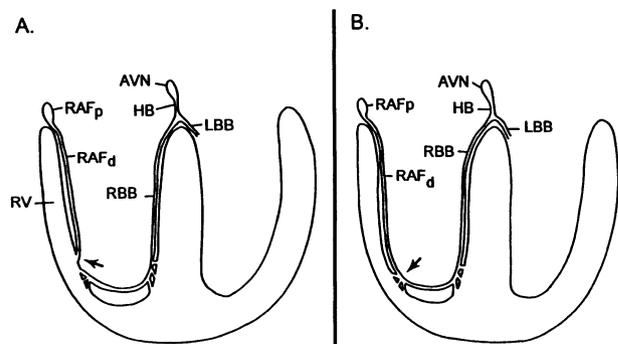


FIG 12. Schematic representation of a right atriofascicular accessory pathway (AP) containing a proximal portion (RAF_p) at and above the tricuspid annulus that is responsible for the conduction delay and decremental conduction properties, and a long distal segment (RAF_d) that has electrophysiological properties similar to those of the right bundle branch (RBB), traveling along the endocardial surface of the right ventricular free wall (RV) beginning at the tricuspid annulus. The distal end of the right atriofascicular AP might insert into the apical region of the right ventricular free wall close to the distal components of the right bundle branch (arrow, panel A) or might fuse with the distal components of the right bundle branch (arrow, panel B). AVN indicates AV node; HB, His bundle; and LBB, left bundle branch.

nodoventricular or nodofascicular APs. Patients with preexcited tachycardia associated with VA block (and AV dissociation) have been described by Gallagher et al.¹³ It appears, however, that nodoventricular and nodofascicular APs produce a preexcited tachycardia far less commonly than right atriofascicular APs.

Radiofrequency catheter ablation of AV APs has become first-line therapy in patients with Wolff-Parkinson-White syndrome.¹⁶⁻¹⁸ When AP potentials are recorded by the ablation electrode, the procedure is highly successful,¹⁶⁻¹⁸ and recurrence of AP conduction after ablation is uncommon.³¹ Catheter ablation of right atriofascicular APs by delivery of high-energy DC shocks near the ventricular apex has been shown to be effective.³² Care must be taken to ensure that the AP is ablated proximal to its first connection with the ventricle or right bundle branch (basal to arrows in Figs 12A and 12B, respectively). Ablation of the distal septal right bundle branch will not eliminate AP conduction and, paradoxically, may increase episodes of AV reentrant tachycardia by prolonging the VA conduction time (Fig 3). Ablation close to the atrial end of the AP should be highly successful, with a lower risk of proarrhythmia. The absence of retrograde AP conduction prevents traditional means of locating the atrial end of the pathway. Ablation of right atriofascicular fibers at the tricuspid annulus at sites identified by delivering atrial extrastimuli around the tricuspid annulus (atrial pace mapping) has been described.^{19,20} Localizing the atrial insertion of the AP by atrial pace mapping is cumbersome and limited in accuracy, as reflected by the large number of applications of radiofrequency current that were required.^{19,20} The results of this study show that the location of a right atriofascicular AP can be accurately identified by recording the AP potential along the lateral, anterolateral, or posterolateral tricuspid annulus and along the right ventricular free wall and that these potentials identify sites for successful ablation of these unique APs.

Acknowledgments

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