Transcatheter Electrical Ablation of Accessory Pathways in Children

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BROMBERG, B.I., ET AL.: Transcatheter Electrical Ablation of Accessory Pathways in Children. *Supraventricular tachycardia (SVT), the most common sustained symptomatic arrhythmia of childhood, is often supported by a manifest or concealed accessory pathway. Permanent interruption of the accessory pathway usually requires surgical division. Recent experience with electrical ablation of posterior septal pathways in adults prompted us to apply the technique to children. Six children, ages 8 to 15 years, underwent a complete electrophysiological study followed by transcatheter electrical ablation. Five of the 6 children, 3 with a right posterior septal and 2 with a left posterior septal pathway, were approached with the ablation catheter at the os of the coronary sinus. In the remaining patient, a left lateral pathway was mapped with an electrode catheter in the coronary sinus and then approached with the ablation catheter through the patent foramen into the left atrium. Two patients are asymptomatic 18–24 months postablation; one patient had return of anomalous conduction between 7 and 21 days after ablation. Two patients had transient interruption of anomalous conduction, whereas one patient experienced no effect. We conclude that in carefully selected patients, transcatheter electrical ablation offers an alternative to surgery for permanent interruption of an accessory pathway. (PACE, Vol. 12, November 1989)*

electrical ablation, accessory pathway, supraventricular tachycardia

Introduction

Supraventricular tachycardia (SVT) is the most common symptomatic tachyarrhythmia of childhood and adolescence[1] and is often supported by either a manifest or concealed accessory connection (Kent bundle).[2] Definitive therapy for supraventricular tachycardia supported by accessory connections is dependent upon interruption of the pathway. Medical therapy, while often effective, does not permanently interrupt anomalous conduction and is not without complications.[3–7] In addition to the various side effects of the commonly used antiarrhythmic agents, compliance in an otherwise asymptomatic young patient facing a lifetime of drug therapy requires individual and family cooperation. Until recently, definitive therapy required surgical division of the accessory pathway. Recent successful experience in adults with transcatheter electrical ablation of accessory pathways located in the posterior septal region of the atrioventricular junction[8–11] have led us to examine the efficacy of this procedure in children with symptomatic SVT. In this report, results from six children are presented.

Methods/Patients

The clinical data are summarized in Table I. Six patients, ages 8 to 15 years, had electrocardiographic and electrophysiological documentation of SVT due to an accessory pathway and were candidates for definitive therapy. Five of the six patients had received antiarrhythmic medica-

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Table I.

Clinical Data

Patient	Age (Yrs.)	Weight (Kg)	Indications	Preexcitation	Location of Accessory Pathway	Preablation Treatment
1	15	68	Frequent palpitations Rapid ventricular response	Yes	Left posterior septal	Digoxin, Propranolol
2	15	74	Frequent palpitations Syncope Rapid ventricular response	Yes	Left posterior septal	Digoxin, Propranolol
3	17	37	Frequent palpitations	Yes	Left lateral	Propranolol
4	15	101	Frequent palpitations	No	Right posterior septal (concealed)	Digoxin
5	15	69	Frequent palpitations	Yes	Right posterior septal	Digoxin, Quinidine
6	8	31	Frequent palpitations	No	Right posterior septal (concealed)	Digoxin, Quinidine

Abbreviations: kg = kilograms, yrs = years.

tions for at least 1 year, yet continued to experience tachycardia. One patient presented with a wide QRS tachycardia that at electrophysiological study was SVT with rate related right bundle-branch block. Two patients demonstrated at electrophysiological study a short anterograde effective refractory period (with a rapid ventricular response) of the accessory pathway; one of these patients had experienced syncope. None had evidence for atrioventricular nodal reentrant tachycardia at electrophysiological study prior to ablation. The protocol for catheter ablation was approved by the Institutional Review Board of the University of Michigan and informed consent was obtained from both patients and parents.

Right and left atrial programmed extrastimulation and mapping of the retrograde atrial activation sequence during SVT were performed through right atrial, coronary sinus, and His bundle electrode catheters. Because identification of the coronary sinus os is critical for positioning of the electrode catheter, the coronary sinus electrode catheter was exchanged for a #6 French end-hole catheter for angiographic localization of the os. A previously unused #6 French USCI quadripolar (10-mm interelectrode distance) catheter was then placed in the coronary sinus with the proximal electrode pair at the os. A catheter was advanced to the apex of the right ventricle to ensure a ventricular impulse following the ablation attempt. The patient was then administered general anesthesia. In five patients, the two electrodes at the coronary sinus os were joined to form the cathode and a cardioversion patch was placed between the lower processes of the scapulae (4 patients) or on the chest (1 patient) to serve as the anode. In patient 3, who had a left lateral pathway, the distal electrode pair of the coronary sinus catheter was used to localize the pathway. The ablation catheter was advanced through a patent foramen ovale and positioned adjacent to this distal electrode pair abutting the lateral left atrial wall (Fig. 1). The distal electrode of this catheter served as the cathode. In each patient a synchronized discharge of 3–5 watt-seconds/kg was delivered 1–4 times between the catheter electrodes and patch (Table II).

Thirty minutes following the ablation attempt, electrophysiological studies were repeated. Electrophysiological studies were also

done at 1 week in three patients (patients 4–6) as well as at 6 months in one (patient 4).

Results

Immediate Effects

Electrocardiographic tracings and electrophysiological studies demonstrated preexcitation in patients 1, 2, 3 and 5 and concealed pathways in patients 4 and 6 (Table I). Results immediately following ablation demonstrated significant alteration of the accessory pathway conduction properties in patients 2–6 (Table II). No immediate effect on the accessory pathway was observed in our first patient (#1) who had a left posterior septal pathway confirmed at surgery. In patients 2, 3, and 5 there was transient loss of preexcitation, as well as loss of inducible SVT in the latter two. Of the two children with concealed pathways, patient 4 had loss of both VA conduction and inducible SVT. Although patient 6 had inducible SVT, the cycle length of the SVT was longer, the retrograde atrial activation sequence was altered, and atrial preexcitation by ventricular stimulation during SVT was lost (Fig. 2). These observations suggest that the postablation SVT was not supported by the previously documented accessory pathway but by atrioventricular nodal reentry.

Early (One Week) Effect (Table II)

Because of the return of preexcitation and SVT within 1 week, patients 1, 2, and 3 had surgical division of the accessory pathway. Patients 4–6 were clinically free of recurrent SVT at 1 week. Electrophysiological studies in patients 4 and 6 at 1 week showed interruption of VA conduction across the accessory connection and no inducible SVT. Although patient 5 had inducible SVT, a change in the retrograde atrial activation sequence indicated that the retrograde limb of the reentry circuit had shifted away from the accessory pathway to the atrioventricular node (Fig. 3), suggesting atrioventricular nodal reentry tachycardia.

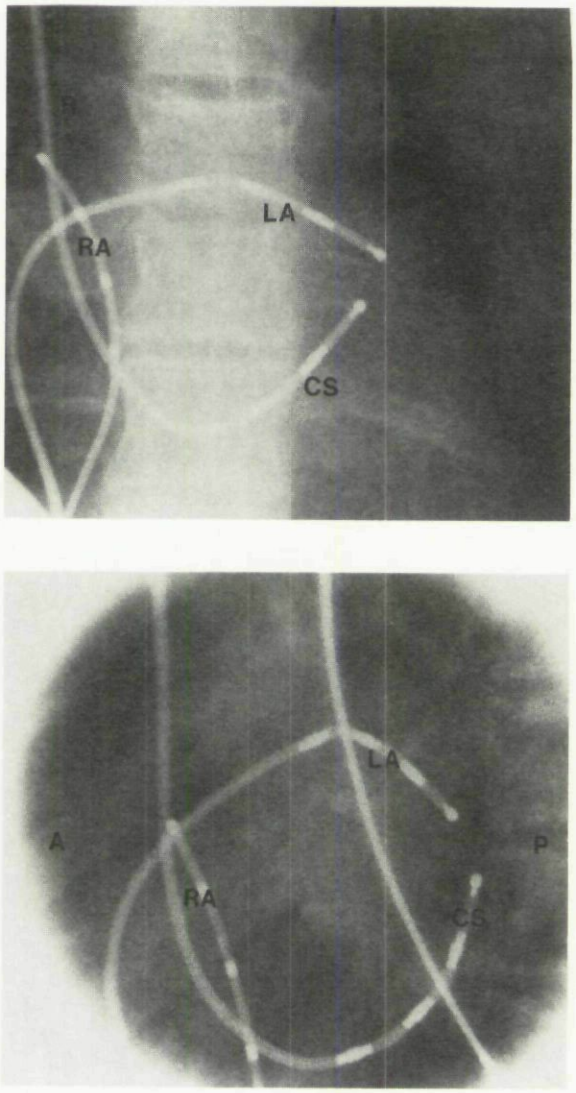


Figure 1. *Catheter positions for ablation of the left lateral pathway in patient 3. Top panel: PA projection; Bottom panel: left lateral projection. The mapping catheter was advanced into the coronary sinus (CS) from the left subclavian vein. The ablating catheter was passed across the patent foramen ovale into the left atrium (LA). The pacing catheter is shown in the high right atrium (RA). A = anterior, lateral projection; L = left, anteroposterior projection; P = posterior, lateral projection; R = right, anteroposterior projection.*

Late Effects

The late follow-up data (Table II) pertain only to patients 4–6 who did not have surgical division of the accessory connection. A repeat electrophysiological study on patient 4 6 months later showed no ventriculoatrial conduction and no in-

Table II.

Follow-up Data

Patient	Location	Voltage	Immediate Effect	One Week Follow-up	One Year Follow-up
1	Left posterior septal	300 ws	None (poor position in CS)	Surgical division	S/P surgical division; no SVT; no preexcitation
2	Left posterior septal	300 ws × 3	3 minute loss of pre-excitation	Surgical division	S/P surgical division; no SVT; no preexcitation
3	Left lateral	20 ws 100 ws 150 ws	Loss of pre-excitation X 24 hour and no inducible SVT	Surgical division	S/P surgical division; no SVT; no preexcitation
4	Right posterior septal (concealed)	300 ws × 2	Loss of V-A conduction No inducible SVT	EPS: Loss of V-A conduction No inducible SVT No clinical SVT	No clinical SVT; EPS:-No V-A conduction -No inducible SVT
5	Right posterior septal	300 ws × 3	Loss of pre-excitation Decreased V-A conduction Non-inducible SVT	EPS: SVT due to AVN reentry No preexcitation No atrial preexcitation during SVT	Preexcitation (returned 7-21 days after ablation) Clinical SVT
6	Right posterior septal (concealed)	100 ws × 2	SVT CL increased from 260–320 to 420 ms due to increase in AV conduction time; No atrial preexcitation during SVT	Repeat EPS: No inducible SVT No AC retrograde conduction by PVES	No clinical SVT on Quinidine and Digoxin

Abbreviations: AC = Accessory connection, AV = Atrioventricular, AVN = Atrioventricular nodal, CL = Cycle length, CS = Coronary sinus, EPS = Electrophysiologic study, PVES = Programmed ventricular extrastimulation, S/P = Status post, SVT = Supraventricular tachycardia, V-A = Ventriculoatrial, WS = Watt seconds.

ducible SVT. This patient remains well and arrhythmia-free 2 years after ablation. Preexcitation and clinical SVT returned in patient 5 between 7 and 21 days postablation attempt, but at slower rates and more responsive to termination by Valsalva maneuvers. SVT at the longer cycle length returned in patient 6. He resumed his medications and remains free of clinical tachycardia; he has declined follow-up electrophysiological study.

Complications

There were no major complications associated with the procedure. As expected, all pa-

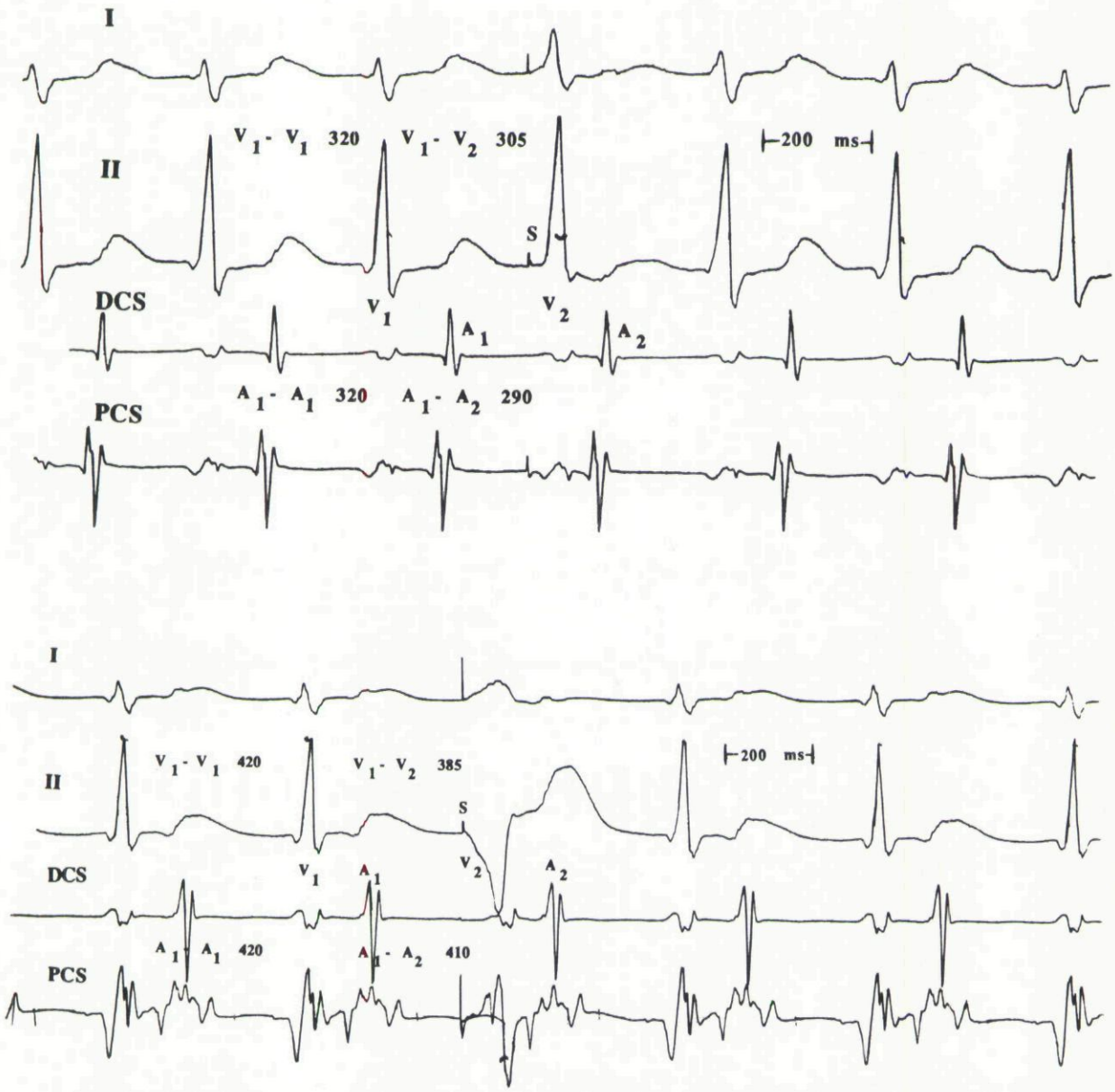
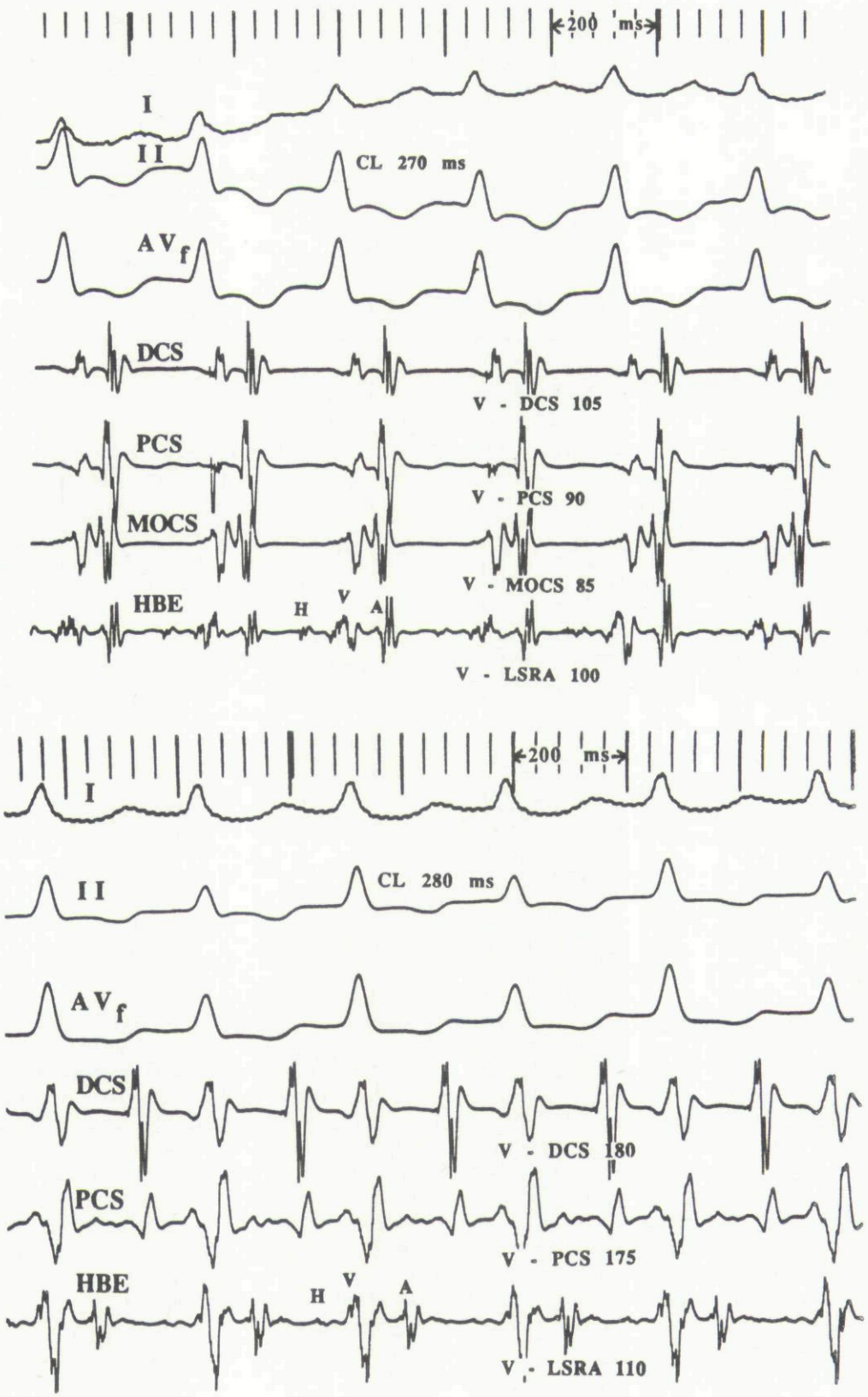


Figure 2. *Electrocardiographic leads I and II and intracardiac electrograms recorded during supraventricular tachycardia before (top panel) and after (bottom panel) ablation in patient 6. Top Panel: The SVT cycle length (V_1–V_1) is 320 msec. Programmed ventricular extrastimulation delivered when the His bundle is refractory shortens the cycle length (V_1–V_2) by 15 msec. However, the A, A_2 interval is shortened by 30 msec demonstrating atrial preexcitation and indicating retrograde conduction over an accessory pathway. Bottom panel: The SVT cycle length (V_1–V_2) has slowed to 420 msec. Ventricular extrastimulation shortens the cycle length 35 msec at a time when the His bundle is refractory. However, there is no atrial preexcitation, A_1–A_2 interval shortening only 10 msec, indicating interruption of retrograde conduction over the accessory pathway and relatively slow conduction (170 msec) through the postablation atrioventricular node-His Purkinje system. DCS = distal coronary sinus; Msec = millisecond; PCS = proximal coronary sinus; S = stimulus artifact.*


200 ms
I
II
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DCS
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V - PCS 90
MOCS
V - MOCS 85
HBE
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A
V - LSRA 100
200 ms
I
II
CL 280 ms
AVf
DCS
V - DCS 180
PCS
V - PCS 175
HBE
H
V
A
V - LSRA 110

Table III.
Complications

Patient	Location	CK (normal 30-225 IU/L)	Myocardial Band (normal 0–3%)	Observation at Surgery
1	Left posterior septal	255	6	No lesion (3 days)
2	Left posterior septal	—	—	No lesion (5 days)
3	Left lateral	427	5	Endocardial 8 mm lesion, raised and hemorrhagic (not transmural) (3 days)
4	Right posterior septal (concealed)	214	9	—
5	Right posterior septal	639	8	—
6	Right posterior septal (concealed)	298	6	—
	Mean	367	6.8	

Abbreviations: CK = Creatinine phosphokinase, IU/L = International units per liter.

tients experienced transient atrioventricular block and all recovered within 5 minutes. The serum creatinine kinase ranged from 214 to 639 IU/L (normal 30 to 225 IU/L) and the myocardial band fraction from 6% to 9% (normal 0% to 3% if CK > 300 IU/L). All returned to normal by the third day (Table III). The two operated patients with left posterior septal had no visible lesions observed at surgery 3–7 days following ablation. The patient with the left lateral pathway, operated on 3 days after ablation attempt, exhibited an 8-mm raised petechial lesion on the lateral left atrial endocardial surface, probably related to electrode abutment on the left atrial wall during

Figure 3. *Electrocardiographic leads AVF and V_1 and intracardiac recordings before (top panel) and after (bottom panel) electrical ablation in patient 5. Top panel: During SVT the cycle length is 270 msec and the initial atrial activation is at the mouth of coronary sinus, preceding activation in the low septal right atrium by 15 msec, indicating retrograde activation of the atria through a right posterior septal pathway. Bottom panel: During SVT after ablation the activation of the low septal right atrium precedes atrial activation at the proximal coronary sinus by 65 msec and at the anterior right atrium by 10 msec. This indicates a shift in the origin of retrograde atrial activation during SVT from the right posterior septal pathway to the region of the atrioventricular node. Decremental retrograde conduction during right ventricular extrastimulation was also present, making the presence of an additional concealed accessory connection unlikely. A = atrial electrogram; I = electrocardiogram lead I; II = electrocardiogram lead II; AV_F = electrocardiogram lead AV_F; DCS = distal coronary sinus; H = His bundle; HBE = His bundle electrogram; LSRA = low septal right atrium; MOCS = mouth of coronary sinus; msec = milliseconds; PCS = proximal coronary sinus; V = ventricular electrogram; V–DSC = ventricular-distal coronary sinus conduction time (msec); V–LSRA = ventricular-low septal right atrium conduction time (msec); V–MOCS = ventricular-mouth of coronary sinus conduction time (msec); V–PCS = ventricular-proximal coronary sinus conduction time (msec).*

electrical discharge. Two patients had atrioventricular (AV) nodal reentrant tachycardia following ablation.

Discussion

The recent development of closed-chest catheter ablation of conduction pathways rests upon several clinical and experimental observations. Initial reports of successful interruption of atrioventricular conduction by transcatheter His bundle ablation dogs[12] were followed by application of the technique to humans with rapid ventricular response to atrial flutter/fibrillation or refractory SVT in WPW syndrome.[13,14] Following initial case reports of posterior septal pathway ablation,[8,9] Morady and Scheinman summarized their initial experience in eight patients using closed-chest catheter ablation.[11] Their current experience of ablation of posterior septal pathways includes 42 patients; the success rate is 74% (Morady, personal communication). Less success has accompanied electrical ablation of automatic ectopic foci and accessory pathways with persistent junctional reciprocating tachycardia in children.[15,16] Although the results of surgery are excellent for Wolff-Parkinson-White syndrome (86%–99% successful interruption of the pathway), the procedure requires thoracotomy, cardiopulmonary bypass, prolonged hospital stay (5–7 days at our institution) and the associated patient morbidity.[17,18]

Two of our six patients received long-term benefits from the electrical ablation procedure. Conduction across the concealed accessory pathway was permanently interrupted in patient 4. In patient 6, no evidence of conduction across the pathway was present following ablation; however, pharmacologically controlled atrioventricular nodal reentrant tachycardia appeared after the ablation, suggesting either the unmasking or the induction of another tachycardia. Because of the known coexistence of AV nodal reentrant tachycardia in the setting of an accessory pathway,[19–22] we speculate that the substrate for this arrhythmia probably existed prior to ablation attempt.

The four patients in whom interruption was only transient illustrate the limitations of the technique. A number of factors probably may influence the results of the procedure. The microanatomy of the accessory connection (multiple vs. single, broad vs. narrow), and its course through the atrioventricular groove (endocardial, epicardial, or within the fat pad) determine its proximity to the coronary sinus os and cannot be altered. On the other hand, certain variables can be altered, including the amplitude of energy delivered and the electrode placement. Given the small, but probably decisive variation in the distance between the discharge source and the conduction pathway target, as well as the variable injury resulting from the discharge, we postulate that in patients 1, 2, 3, and 5, the lack of proximity between the ablating cathode at the mouth of the coronary sinus and the accessory connection contributed to the failure of the technique. This hypothesis is supported in patients 1 and 2 by epicardial and endocardial mapping at surgery demonstrating that the accessory pathway was further to the left and more epicardial and thus, more distant from the mouth of the coronary sinus than was predicted by electrophysiological study prior to ablation attempt.

The approach to patient 3 with the left lateral pathway was unique. Because experimental delivery of an ablative electrical discharge into the coronary sinus of dogs has produced perforation of the coronary sinus wall,[23,24] we approached the left lateral pathway through the patent foramen ovale. Although we were able to achieve close proximity (Fig. 2) between the left atrial ablating and the coronary sinus mapping catheters, the transient effect (loss of preexcitation and inducible SVT for 12 hours) as well as the raised oval shaped petechial lesion found at surgery 3 days later suggest that the effective proximity between the target pathway and energy source is difficult to achieve by this technique. This patient, as did all the patients, received a joules per kilogram dose equivalent to the reported adult dose; in view of the observed lesion at surgery, an increased or repeated dose would appear to be imprudent. It should be noted, however, that one recent report suggests that localization of the pathway is more precise and that ablation of posterior septal pathways as well as left lateral and right-sided free-wall pathways can be achieved if

either the accessory pathway potential is directly recorded during SVT or ablation is delivered at the site (electrode) of earliest activation recorded during SVT.[25]

While it is tempting to consider manipulating the ablation catheter more distally into the coronary sinus or to increase the energy delivered when an attempt is unsuccessful, caution must be exercised. Rupture of the coronary sinus has been demonstrated in the canine model using 10 joules/kg discharge, as well in adult patients.[23,26] Furthermore, rupture of the coronary sinus has been reported in a woman in whom a left lateral pathway ablation was attempted from the distal coronary sinus, and has been observed in attempted ablation of a posterior septal pathway even when the proximal pair of electrodes were thought to be properly placed. In the latter case, an observed defect in insulation of the catheter perhaps resulted in a leakage of current to the distal electrodes in the coronary sinus with resultant perforation (Morady, personal communication). Thus, it is imperative that the paired discharging electrode be no more distal than at the os of the coronary sinus where atrial myocardium provides a support posteriorly and where the discharge energy is not confined by a closed space. We are also hesitant to attempt the procedure in small children (≤30 kg), in whom the energy per kilogram generated by most direct current cardioversion devices cannot be precisely controlled at lower energy settings, and the electrode position relative to the coronary sinus os as well as relative to the accessory connection may be difficult to determine.

Based upon our limited success, the 74% success rate in adults, and the absence of any unexpected complications, we believe that further attempts of transcatheter electrical ablation in older children with symptomatic SVT supported by the appropriately located accessory connection are warranted. While the overall success of surgery is excellent, the concomitant morbidity warrants further investigation of this technique. It is clear from our experience that patient selection is crucial. Complete electrophysiological study, with the use of unipolar electrodes to record localized electrograms, including perhaps recording of the accessory pathway potential, should be performed to locate as precisely as possible the site of the accessory connection. It should be noted, however, that there exists a subgroup of patients in whom the microanatomy of the accessory pathway may preclude successful interruption by electrical discharge. Furthermore, coexisting mechanisms of tachycardia (such as multiple pathways or atrioventricular nodal reentry) should be carefully excluded prior to ablation. With these caveats in mind, transcatheter electrical ablation of accessory connections supporting SVT may prove to be useful in older children with recurrent, symptomatic, or life-threatening tachyarrhythmia who would otherwise be candidates for surgical treatment of this disorder.

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