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Multiple Accessory Pathways in the Young: The Impact of Structural Heart Disease

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Abstract

Background—The presence of multiple accessory pathways (multAP) is described in structural heart disease (SHD) such as Ebstein's anomaly and cardiomyopathies. Structural defects can impact the tolerability of tachyarrhythmia and can complicate both medical management and ablation. In a large cohort of pediatric patients with and without SHD undergoing invasive electrophysiology study, we examined the prevalence of multAP and the effect of both multAP and SHD on ablation outcomes.

Methods—Accessory pathway number and location, presence of SHD, ablation success, and recurrence were analyzed in consecutive patients from our center over a 16 year period.

Results—In 1088 patients, 1228 pathways (36% retrograde only) were mapped to the right side (TV) in 18%, septum(S) in 39%, and left side (MV) in 43%. MultAP were present in 111 pts (10%), involving 250 distinct pathways. SHD tripled the risk of multAP (26% SHD vs 8% no SHD, $p<0.001$). Multivariable adjusted risk factors for multAP included Ebstein's (OR 8.7[4.4–17.5], $p<0.001$) and cardiomyopathy (OR 13.3[5.1–34.5], $p<0.001$). Of 1306 ablation attempts, 94% were acutely successful with an 8% recurrence rate. Ablation success was affected by SHD (85% vs 95% for no SHD, $p<0.01$) but not by multAP (91% vs. 94% for single, $p=0.24$). Recurrence rate was higher for SHD (17% SHD vs 8% no SHD, $p<0.05$) and multAP (19% multAP vs. 8% single, $p<0.001$).

Conclusions—MultAP are found in 10% of pediatric patients, and are more common in SHD compared to those with normal hearts. Both the presence of multAP and SHD negatively influence ablation outcomes.

Introduction

Multiple accessory pathways (multAP) can be identified in 3–13% of patients undergoing electrophysiology study (EPS) for tachycardias mediated by accessory pathways.^{1–10} The presence of multAP can result in higher risk of supraventricular tachycardia,⁷ a higher incidence of antidromic reentry, the potential for more rapid conduction during atrial fibrillation, and ventricular fibrillation.¹¹ Patients with select forms of structural heart

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disease (SHD), such as Ebstein's anomaly of the tricuspid valve, congenitally corrected transposition of the great arteries, and hypertrophic cardiomyopathy, appear to be at higher than average risk for multAP. Because such patients may tolerate tachycardia poorly owing to compromised cardiac reserve, ablation is often required at a young age. The anatomic challenges of SHD, in combination with the presence of multAP, can negatively impact ablation outcome. This study was undertaken to examine the frequency and locations of multAP in young patients with and without SHD, and measure the relative influence of multAP and SHD on ablation success and recurrence.

Methods

This is a retrospective review of all patients who underwent EPS for accessory pathways (with or without ablation) at Children's Hospital Boston from March 1990 until December 2006. Records were reviewed under approval of the hospital's Committee on Clinical Investigation.

Electrophysiology Studies and Ablation

Standard techniques for transcatheter mapping were employed.¹⁸ All forms of accessory connections were included for analysis, including: a) conventional atrioventricular connections (unidirectional or bidirectional), b) decremental retrograde pathways causing the permanent form of junctional reciprocating tachycardia, c) decremental anterograde right free-wall atriofascicular connections, and d) benign septal fasciculoventricular fibers. Pathway locations were ascertained by review of written procedure reports, and were divided into 13 segments: anterior, anterolateral, lateral, posterolateral, and posterior along the tricuspid (TV) or mitral valve (MV) free walls, and anterior, mid, or posterior septum (S). Cases of multAP were defined as: a) during a single EPS, the presence of 2 or more pathways separated by at least 1 cm, or b) during a subsequent EPS, identification of one or more new pathways not located in the same or immediately adjacent segment as a pathway found in a previously acutely successful EPS. Grouping patterns for multAP locations were generated, and included: a) TV free wall, b) septal, c) MV free wall, d) both mitral and tricuspid free walls, e) both septal and MV free wall, f) both septal and TV free wall, g) combined free walls and septal.

Ablation was undertaken at the discretion of the operator using standard techniques for radiofrequency and/or cryoablation. Ablation was not attempted for 17 pathways, in 6 instances because the pathway was found to be a benign fasciculoventricular connection incapable of supporting tachycardia, and for the remaining 11, because the pathway mapped to a location in close proximity to normal conduction tissues where the risk of heart block was judged to be greater than the risk of tachycardia. Most of the latter group (7 of 11) were studied prior to the availability of cryoablation technology.

Ablation outcomes were graded for all cases in which ablation was attempted. "Acute success" was defined as no inducible tachycardia and no evidence of extranodal conduction following targeted energy applications during a single procedure. "Recurrence" was defined as return of tachycardia or preexcitation with a pattern similar to the originally targeted pathway at any point during follow-up. Rare patients in whom a novel delta wave pattern appeared during follow-up and/or whose repeat EPS identified a previously unrecognized pathway distant from the original target site (>1 segment away) were coded as new cases rather than recurrence. "Overall success" was defined as freedom from tachycardia and absence of preexcitation during follow-up after one or more ablation sessions.

Structural Heart Disease

All patients had at least one echocardiogram performed prior to EPS. Patients were classified as having SHD if any of the following abnormalities were present: congenital structural defects (apart from minor atrial septal communications or small patent ductus arteriosus), cardiomyopathy (including hypertrophic, dilated, and noncompaction varieties), or primary cardiac tumor. To distinguish temporary tachycardia-mediated cardiomyopathy from primary dilated cardiomyopathy, depressed left ventricular function had to persist on echocardiogram >3 months following successful ablation.

Statistical Analysis

Comparisons between groups were performed using the Mann-Whitney U test for continuous variables and Fisher's exact test. Sub-analyses were corrected for multiple comparisons using Bonferroni's method. The presence, success, and recurrence of each pathway in multAP patients were treated independently. Multivariable adjusted, stepwise logistic regression analysis was used to identify factors associated with presence of multAP, procedural success, and recurrence in separate analyses, using 0.1 as criteria for model entry and 0.05 as criteria for retention. For the presence of multAP, included covariates were age, and SHD diagnosis. For the multAP ablation success and recurrence analyses, covariates included age, SHD diagnosis and AP location. Significance was set at p values less than 0.05, with adjustment for multiple comparisons when appropriate. Analyses were performed using PASW 17 (SPSS Inc, Chicago IL).

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Results

Pathway and Patient Characteristics

A total of 1228 pathways were identified in 1088 patients [Table 1]. The majority were capable of anterograde or bidirectional conduction resulting in manifest preexcitation, while 36% were "concealed" with exclusively retrograde conduction. Although most connected in the conventional atrioventricular fashion, a subset of 68 pathways (6%) had atypical connections or conduction characteristics, including: 49 with slow decremental retrograde conduction causing the permanent form of junctional reciprocating tachycardia, 13 atriofascicular pathways along the right free wall with decremental anterograde conduction, and 6 benign septal fasciculoventricular fibers.

Of the 1088 study patients, 121 (11%) were diagnosed as having significant SHD. The most common abnormalities included Ebstein's anomaly of the tricuspid valve, congenitally corrected transposition (with atrioventricular discordance and left-sided tricuspid valve), and cardiomyopathy. A variety of "other" congenital defects were seen, ranging from simple ventricular septal defect to complex single ventricle. Patients with SHD were significantly younger (7 years, IQR 2.4–14.2, $p < 0.001$) at time of EPS than those without, possibly reflecting the need for earlier intervention for tachycardia symptoms or in advance of surgical intervention.²

A total of 111 study patients (10%) were identified as having multAP. Patients with SHD were far more likely to have multAP than those with normal hearts. Specifically, Ebstein's anomaly and cardiomyopathy (particularly the hypertrophic and noncompaction varieties) were strongly associated with the finding of multAP. By logistic regression, Ebstein's

anomaly had an odds ratio of 8.7 (95% CI 4.4–17.5, $p < 0.001$), and myopathy an odds ratio of 13.3 (95% CI 5.1–34.5, $p < 0.001$) as multivariable adjusted predictors of multAP.

Accessory Pathway Locations and Patterns

General pathway locations for the entire study group included TV free wall in 18%, septal in 39%, and MV free wall in 43%. Patients with SHD had a much higher likelihood of pathways in the TV location compared with non-SHD patients (35% vs. 16%, $p < 0.001$), driven in large part by those with Ebstein's anomaly or congenitally corrected transposition [Figure 1]. Similarly, patients with multAP were significantly more likely to have locations along the TV compared to those with a single pathway (26% vs. 17%, $p < 0.001$). The occurrence of pathways in a septal location was similar when analyzed according to SHD vs. non-SHD.

Grouping patterns for multAP in a given patient were examined [Table 2]. The combination of a tricuspid valve pathway with a septal pathway (TV + S) was especially common in the SHD group (41% of patients), particularly those with Ebstein's anomaly. In contrast, patients with multAP and structurally normal hearts appeared far more likely to have the pathways cluster towards the mitral valve ring (57% of patients with either MV or MV + S).

Ablation Outcomes

Ablation was attempted during 1170 procedures on 1312 individual pathways (including repeat procedures for recurrence). Median follow-up time was 6.2 years (interquartile range 2.7–11.5 years). Overall success rate was 94% (92% TV, 92% S, and 97% MV), with a recurrence rate of 8%. Acute and overall success rates were not different between single pathways and multAP as a whole [Table 3], though multAP patients had higher recurrence rate than single pathways, and SHD had higher recurrence than patients without SHD. Examining SHD by subclass, patients with myopathy, and those with “other” SHD, had lowest acute success rates (80% and 88% respectively, $p < 0.01$ for both), and Ebstein's patients a much high recurrence rate (22%, $p < 0.001$), compared to patients with normal hearts. The advent of cryoablation had no significant effect on overall recurrence rates (pre 9%, post 7%, $p = 0.2$).

Multivariable-adjusted predictors of ablation success were absence of SHD (OR 2.7, 95% CI 1.5–4.8, $p < 0.001$), and MV location (OR 3.8, 95% CI 2.0–7.2, $p < 0.001$). Multivariable predictors of recurrence were age less than 2 years (OR 2.4, 95% CI 1.2–5.2, $p = 0.02$) and the existence of multAP (OR 3.2, 95% CI 1.9–5.6, $p < 0.001$).

Major complications from these 1170 procedures included 2 cerebrovascular events, 3 instances of high-grade atrioventricular block, and 3 cases of significant bleeding (1 pericardial effusion, 2 hemothorax). The risk of major complications did not differ according to the presence of either SHD or multAP.

Discussion

This single-center study describes the relationship between structural heart disease and multAP, and measures the relative impact of each on ablation outcome. While some of the findings have been noted previously in smaller series, this large experience provides a firm degree of quantitation that highlights the challenges of accessory pathway ablation in young patients with abnormal hearts.

In this series, 8% of patients with structurally normal hearts had multAP, a number that is comparable to several prior reports. However, study patients with structural disease appeared three times as likely to have multAP as those with structurally normal hearts. The

present data confirm previous findings of a high proportion of multAP in Ebstein's and highlight for the first time that cardiomyopathy patients appear to be prone to multAP. The fact that multAP can be linked to a higher incidence of serious arrhythmic events, coupled with the hemodynamic vulnerability in certain forms of structural heart disease, heightens the importance of effective ablation despite the technical challenges in such patients. Appreciation for this heightened risk of multAP should encourage a compulsive search for secondary and tertiary pathways whenever ablation is undertaken in patients with congenital defects or cardiomyopathy.

The general distribution of accessory pathways in patients with structural defects followed expected patterns, with TV locations predominating in congenital anatomic malformations such as Ebstein's anomaly and congenitally corrected transposition, but more MV locations in patients with cardiomyopathy. These patterns persisted even among patients with multAP. There were also certain preferred groupings of pathway locations in structural disease patients with multAP, most notably a TV + S combination in Ebstein's malformation.²² A genetic mutation affecting AV valve development could explain these observations. For example, the *Nkx2.5/Alk3/BMPR* pathway is involved in AV patterning and electrically-insulating annulus fibrosis formation. *Nkx 2.5* mutations have been shown to be associated with Ebstein's anomaly. Aberrant *Alk3/BMPR* signaling can lead to gaps in the annulus fibrosis and the presence of AP.

The presence of structural disease adversely affected ablation success and increased the recurrence risk. There are many potential explanations for this finding, including younger patient age / small size²⁵, distorted anatomic landmarks, reduced certainty about location for normal conduction tissue (and hence increased caution at septal locations), and the potential for limited vascular access in patients who may have undergone multiple prior catheterizations. Furthermore, in Ebstein's patients where the recurrence risk was particularly high, there can be difficulty identifying the true AV groove due to TV displacement and highly abnormal tissue in the region of interest. Likewise in the case of cardiomyopathy, lower success may be due to broader disorganized accessory pathways²¹ and/or thicker ventricular myocardium.²⁶ *PRKAG2*, located on chromosome 7, has been associated with familial cases of HCM with WPW.²⁷ The high proportion of our cohort of unrelated HCM patients with multiple AP may suggest a need to genetically screen these patients and may have implications for future progeny. The presence of multAP in itself did not affect acute success of the ablation procedure, though recurrence was higher than single pathway patients, perhaps owing to an additive risk of recovery for each individual pathway.

Limitations

As a referral center for complex patients, the institutional case mix may have been biased towards more abnormal anatomic substrates, which could affect observations on the prevalence of multAP. Also, the ablation experience described here spanned a period of 16 years, during which multiple technological advances have occurred to refine anatomic resolution and increase safety and efficacy of ablative lesion creation. Success and recurrence rates may be improved in the current era. It is possible that pathways we have noted as multiple due to AP separated by at least 1cm may rarely represent a single oblique pathway or some other complex pathway geometry. This would overestimate the number of multAP. However the overwhelming number of multAP occurring in nonadjacent segments and rarity of AP diameters or length exceeding 1cm gives credence to the overall findings herein.

Conclusions

In a large cohort of young patients with accessory pathways, multAP were more common in those with structural disease, particularly Ebstein's anomaly and cardiomyopathy. Certain distribution patterns are apparent for both isolated and multiple pathway locations in this population. Ablation success is lower, and recurrence rate is higher, among patients with structural defects. The presence of multiple accessory pathways may further increase this recurrence risk.

References

1. Bromberg BI, Lindsay BD, Cain ME, Cox JL. Impact of clinical history and electrophysiologic characterization of accessory pathways on management strategies to reduce sudden death among children with Wolff-Parkinson-White syndrome. *J Am Coll Cardiol.* 1996; 27(3):690–695. [PubMed: 8606283]
2. Chetaille P, Walsh EP, Triedman JK. Outcomes of radiofrequency catheter ablation of atrioventricular reciprocating tachycardia in patients with congenital heart disease. *Heart Rhythm.* 2004; 1(2):168–173. [PubMed: 15851148]
3. Chen SA, Hsia CP, Chiang CE, Chiou CW, Yang CJ, Cheng CC, et al. Reappraisal of radiofrequency ablation of multiple accessory pathways. *Am Heart J.* 1993; 125(3):760–771. [PubMed: 8438705]
4. Iturralde P, Guevara-Valdivia M, Rodriguez-Chavez L, Medeiros A, Colin L. Radiofrequency ablation of multiple accessory pathways. *Europace.* 2002; 4(3):273–280. [PubMed: 12134973]
5. Kugler JD, Danford DA, Deal BJ, Gillette PC, Perry JC, Silka MJ, et al. Radiofrequency catheter ablation for tachyarrhythmias in children and adolescents. The Pediatric Electrophysiology Society. *N Engl J Med.* 1994; 330(21):1481–1487. [PubMed: 8164700]
6. Kugler JD, Danford DA, Houston KA, Felix G. Pediatric radiofrequency catheter ablation registry success, fluoroscopy time, and complication rate for supraventricular tachycardia: comparison of early and recent eras. *J Cardiovasc Electrophysiol.* 2002; 13(4):336–341. [PubMed: 12033349]
7. Pappone C, Manguso F, Santinelli R, Vicedomini G, Sala S, Paglino G, et al. Radiofrequency ablation in children with asymptomatic Wolff-Parkinson-White syndrome. *N Engl J Med.* 2004; 351(12):1197–1205. [PubMed: 15371577]
8. Weng KP, Wolff GS, Young ML. Multiple accessory pathways in pediatric patients with Wolff-Parkinson-White syndrome. *Am J Cardiol.* 2003; 91(10):1178–1183. [PubMed: 12745099]
9. Yeh SJ, Wang CC, Wen MS, Lin FC, Wu D. Radiofrequency ablation in multiple accessory pathways and the physiologic implications. *Am J Cardiol.* 1993; 71(13):1174–1180. [PubMed: 8480643]
10. Arya A, Haghjoo M, Jafari A, Emkanjoo Z, Fazelifar AF, Dehghani MR, et al. Effect of conduction mode and location on electrophysiologic characteristics of accessory pathways. *Am J Cardiol.* 2005; 95(10):1250–1252. [PubMed: 15878005]
11. Klein GJ, Bashore TM, Sellers TD, Pritchett EL, Smith WM, Gallagher JJ. Ventricular fibrillation in the Wolff-Parkinson-White syndrome. *N Engl J Med.* 1979; 301(20):1080–1085. [PubMed: 492252]
12. Benson DW Jr, Gallagher JJ, Oldham HN, Sealy WC, Sterba R, Spach MS. Corrected transposition with severe intracardiac deformities with Wolff-Parkinson-White syndrome in a child. Electrophysiologic investigation and surgical correction. *Circulation.* 1980; 61(6):1256–1261. [PubMed: 7371140]
13. Perosio AM, Suarez LD, Bunster AM, Locreille A, Apkarian OA, Vallazza MA, et al. Pre-excitation syndrome and hypertrophic cardiomyopathy. *J Electrocardiol.* 1983; 16(1):29–40. [PubMed: 6682136]
14. Deal BJ, Keane JF, Gillette PC, Garson A Jr. Wolff-Parkinson-White syndrome and supraventricular tachycardia during infancy: management and follow-up. *J Am Coll Cardiol.* 1985; 5(1):130–135. [PubMed: 3964800]

15. Weindling SN, Saul JP, Walsh EP. Efficacy and risks of medical therapy for supraventricular tachycardia in neonates and infants. *Am Heart J.* 1996; 131(1):66–72. [PubMed: 8554021]
16. Bae EJ, Ban JE, Lee JA, Jin SM, Noh CI, Choi JY, et al. Pediatric radiofrequency catheter ablation: results of initial 100 consecutive cases including congenital heart anomalies. *J Korean Med Sci.* 2005; 20(5):740–746. [PubMed: 16224145]
17. Wu MH, Lin JL, Lai LP, Young ML, Lu CW, Chang YC, et al. Radiofrequency catheter ablation of tachycardia in children with and without congenital heart disease: indications and limitations. *Int J Cardiol.* 2000; 72(3):221–227. [PubMed: 10716130]
18. Saul, JP. Ablation of Accessory Pathways. In: Walsh, EP.; Saul, JP.; Triedman, JK., editors. *Cardiac arrhythmias in children and young adults with congenital heart disease.* Philadelphia: Lippincott Williams & Wilkins; 2000.
19. Bar-Cohen Y, Cecchin F, Alexander ME, Berul CI, Triedman JK, Walsh EP. Cryoablation for accessory pathways located near normal conduction tissues or within the coronary venous system in children and young adults. *Heart Rhythm.* 2006; 3(3):253–258. [PubMed: 16500293]
20. Cox JL, Gallagher JJ, Cain ME. Experience with 118 consecutive patients undergoing operation for the Wolff-Parkinson-White syndrome. *J Thorac Cardiovasc Surg.* 1985; 90(4):490–501. [PubMed: 4046618]
21. Lev M, Kennamer R, Prinzmetal M, De Mesquita QH. A histopathologic study of the atrioventricular communications in two hearts with the Wolff-Parkinson-White syndrome. *Circulation.* 1961; 24:41–50. [PubMed: 13761305]
22. Colavita PG, Packer DL, Pressley JC, Ellenbogen KA, O'Callaghan WG, Gilbert MR, et al. Frequency, diagnosis and clinical characteristics of patients with multiple accessory atrioventricular pathways. *Am J Cardiol.* 1987; 59(6):601–6. [PubMed: 3825901]
23. Stroud DM, Gaussin V, Burch JB, Yu C, Mishina Y, Schneider MD, et al. Abnormal conduction and morphology in the atrioventricular node of mice with atrioventricular canal targeted deletion of *Alk3/Bmpr1a* receptor. *Circulation.* 2007; 116(22):2535–2543. [PubMed: 17998461]
24. Gioli-Pereira L, Pereira AC, Mesquita SM, Xavier-Neto J, Lopes AA, Krieger JE. NKX2.5 mutations in patients with non-syndromic congenital heart disease. *Int J Cardiol.* 2010; 138(3): 261–265. [PubMed: 19073351]
25. Langberg JJ, Calkins H, Kim YN, Sousa J, el-Atassi R, Leon A, et al. Recurrence of conduction in accessory atrioventricular connections after initially successful radiofrequency catheter ablation. *J Am Coll Cardiol.* 1992; 19(7):1588–1592. [PubMed: 1593055]
26. Bharati S, Strasberg B, Bilitch M, Salibi H, Mandel W, Rosen KM, et al. Anatomic substrate for preexcitation in idiopathic myocardial hypertrophy with fibroelastosis of the left ventricle. *Am J Cardiol.* 1981; 48(1):47–58. [PubMed: 6454339]
27. Mehdirdad AA, Fatkin D, DiMarco JP, MacRae CA, Wase A, Seidman JG, et al. Electrophysiologic characteristics of accessory atrioventricular connections in an inherited form of Wolff-Parkinson-White syndrome. *J Cardiovasc Electrophysiol.* 1999; 10(5):629–35. [PubMed: 10355918]
28. Anderson RH, Becker AE. Stanley Kent and accessory atrioventricular connections. *J Thorac Cardiovasc Surg.* 1981; 81(5):649–58. [PubMed: 7012454]
29. Becker AE, Anderson RH, Durrer D, Wellens HJ. The anatomical substrates of wolff-parkinson-white syndrome. A clinicopathologic correlation in seven patients. *Circulation.* 1978; 57(5):870–9.

Figure 1.

Depiction of each pathway in SHD patients by 13 location segments: anterior, anterolateral, lateral, posterolateral, and posterior locations along the tricuspid (TV) or mitral valve (MV); or along anterior, mid, or posterior septum. Panel a) Ebstein's anomaly; b) AV Discordance with left-sided tricuspid valve; c) Myopathy; and d) "Other" structural heart disease. Solid circles indicate pathway locations in multAP patients while open circles indicate single pathways.

Table 1

Baseline Characteristics comparing Single Pathway to Multiple Pathway patients

	All	Single	MultAP	P value
Patients	1088	977	111	
Accessory Pathways	1228	977	251	
Age	12.9	12.9	13.0	0.40
(median,interquartile range; maximum)	(8.1,15.9;60)	(8.2,15.9;60)	(7.5,15.2;41)	
Female	426 (39%)	384 (39%)	42 (38%)	0.38
Structural Heart Disease	122 (11%)	90 (9%)	32 (29%)	<0.0001
Ebstein's	37 (3%)	20 (2%)	17 (15%)	<0.0001
AV Discordance	22 (2%)	18 (2%)	4 (4%)	0.18
Myopathy	18 (2%)	8 (1%)	10 (10%)	<0.0001
<i>Hypertrophic</i>	6	2	4	
<i>Dilated</i>	6	4	2	
<i>LV Noncompaction</i>	4	1	3	
<i>Rhabdomyoma</i>	2	1	1	
Other SHD	73 (7%)	64 (7%)	9 (8%)	0.31

Data listed as number of patients and proportion of total patients in respective column except where otherwise noted. Some SHD patients had more than one diagnosis making the subclass total exceed the number of patients with structural heart disease. P value denotes comparison between single and MultAP

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Table 2
Grouping pattern for pathway location in patients with multiple accessory pathways

		TV	TV + S	S	TV + MV + S	MV + S	MV	TV + MV
MultAP (111 pts)	No SHD (79 pts)	7	9	12	1	26	19	5
	SHD (32 pts)	5	13	3	2	5	3	1

Abbreviations: mult/AP = multiple accessory pathways, MV = mitral valve, S = septal, SHD = structural heart disease, TV = tricuspid valve

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Table 3

Comparison of Success and Recurrence

	Single	MultAP	No SHD	SHD
Procedures	1051	119	1031	139
Overall Success per patient	94%	91%	95%	85%
p value	0.17 <0.001			
Acute Success per pathway	94%	93%	94%	89%
p value	0.66 0.002			
Recurrence per patient	7%	18%	7%	15%
p value	<0.001 0.003			

Abbreviations: Single- single accessory patients; MultAP- multiple accessory pathway patients; SHD- structural heart disease.

Overall Success per patient is the proportion of patients successfully ablated. Acute Success per pathway is the proportion of pathways successfully ablated. Recurrence is the proportion of pathways that returned for EPS. P values pertain to comparisons of Single vs. MultAP or No SHD vs. SHD.