Europace (2003) **5,** 225–229
doi:10.1016/S1099–5129(03)00037-0

Clinical and electrophysiological characteristics in patients with atrioventricular reentrant and atrioventricular nodal reentrant tachycardia

N. Bottoni[1], C. Tomasi[1], P. Donateo[2], G. Lolli[1], N. Muià[1], F. Croci[2], D. Oddone[2], C. Menozzi[1] and M. Brignole[2]

[1]*Unità Operativa di Cardiologia Interventistica, Dipartimento di Cardiologia, Azienda Ospedaliera S. Maria Nuova, Reggio Emilia, Italy; and* [2]*Centro Aritmologico, Dipartimento di Cardiologia, Ospedali del Tigullio, Lavagna, Italy*

Aim To compare clinical, electrophysiological characteristics and transcatheter ablation results between two groups of patients, one with atrioventricular reentrant tachycardia (AVRT) and the other with atrioventricular nodal reentrant tachycardia (AVNRT).

Methods The study population consisted of 94 consecutive patients who underwent endocavitary electrophysiological study and radiofrequency (RF) ablation: 46 patients had AVRT due to an accessory pathway with only retrograde conduction while 48 patients had AVNRT.

Results In relation to general and clinical characteristics, differences between the two groups emerged regarding the age of symptom onset (25 ± 16 vs 37 ± 17 years, $p = 0.001$), the prevalence of heart disease (8 vs 31%, $p = 0.001$) and the correct diagnosis on surface ECG (50 vs 79%, $p = 0.001$). Clinical presentation was quite similar apart from a higher prevalence of fatigue and sweating in the AVNRT group. Transcatheter RF ablation therapy results were similar.

Conclusions Patients with AVRT have a lower mean age at arrhythmia symptom onset compared with those with AVNRT and have fewer associated cardiac abnormalities. Clinical presentation is quite similar as well as their outcome after ablation. A correct diagnosis by standard ECG is more frequent in AVNRT.
(Europace 2003; 5: 225–229)

Key Words: Paroxysmal supraventricular tachycardia, cardiac electrophysiology, transcatheter radiofrequency ablation, atrioventricular reentrant tachycardia, atrioventricular nodal reentrant tachycardia.

Introduction

Atrioventricular nodal reentrant tachycardia (AVNRT) and atrioventricular reentrant tachycardia (AVRT) due to a concealed accessory pathway are the most common forms of paroxysmal tachycardias and together represent about 90% of regular supraventricular tachycardias (SVT)[1]. In patients undergoing endocavitary electrophysiological studies, AVNRT are seen in about 70% of cases, AVRT due to a concealed accessory pathway in 20% and the remainder are atrial tachycardias[2]. Although these arrhythmias have long been known, there are relatively few studies in the literature that compare, in a systematic way, their clinical and electrophysiological characteristics[3–11]. Moreover, the large majority of these studies consider as a single group patients with AVRT due to a concealed accessory pathway and patients with AVRT by manifest accessory pathways; the latter group probably present a more complex symptomatic profile due to a greater tendency to develop atrial fibrillation episodes[12–14].

The aim of the study was to compare clinical, electrophysiological characteristics and transcatheter ablation results in two groups of patients, one with AVRT due to a concealed accessory pathway and the other with AVNRT.

Manuscript submitted 12 June 2002, and accepted after revision 27 March 2003.

Correspondence: Nicola Bottoni, Unità Operativa di Cardiologia Interventistica, Azienda Ospedaliera S. Maria Nuova, V. Risorgimento 80, Reggio Emilia, Italy. Tel: +39-0522-296574; Fax: +39-0522-296288; E-mail: bottoni.nicola@asmn.re.it

1099–5129/03/000225+05 $30.00/0

Methods

Study population

From January 1999 to June 2000, 46 consecutive patients with AVRT due to a concealed accessory pathway underwent electrophysiological study and radiofrequency (RF) catheter ablation at our hospitals. Their clinical and electrophysiological characteristics were compared with a group of 48 consecutive patients with AVNRT who were studied at the same centres during the year 2000. All patients were seen 3 and 6 months after the procedure. They underwent an additional ablation procedure if a tachycardia relapse occurred.

Electrophysiological study

All patients gave their informed consent and were not under sedation during the procedure. Antiarrhythmic therapy was suspended before the study for a period equal to five half-lives of the drug. Two tetrapolar electrocatheters were introduced via the femoral vein and positioned under fluoroscopic guidance at the top of the right atrium, on the His bundle and successively at the right ventricular apex. A decapolar catheter was placed into the coronary sinus via the antecubital or subclavian vein. Intracardiac signals were amplified and filtered from 30 to 500 Hz and were recorded and analyzed by a digital polygraph system. All patients underwent incremental and programmed atrial–ventricular stimulation protocols in order to evaluate antegrade–retrograde conduction and tachycardia induction. The accessory pathway was defined as concealed if there was no ventricular preexcitation in sinus rhythm, during right and left atrial stimulation, after 20 mg i.v. adenosine infusion and antegrade conduction showed decremental characteristics during incremental atrial pacing. If the tachycardia could not be induced at baseline, we repeated the study after isoprenaline infusion.

Radiofrequency ablation

The ablation procedure was performed using deflectable quadripolar catheters with a distal electrode of 4 mm and an interelectrode space of 2 mm and temperature controlled RF generators. Right concealed accessory pathways were reached by the femoral vein approach while for those on the left we used either the transeptal catheterization or the transaortic retrograde approach. A RF power of 50 W and a temperature of 65°C were set during the ablations for a period of time of 45 s for accessory pathways and 20 s for AVNRT; the power output was titrated during RF application. Procedural endpoints for patients with AVRT were complete disappearance of the accessory pathway during ventricular pacing and inability to induce the tachycardia. In patients with AVNRT we performed catheter ablation targeting the slow nodal pathway, characterized by low amplitude and fragmented potential, located in most cases at the base of Koch's triangle[15]. In this case the appearance of accelerated junctional rhythm during RF pulses was sought, but the endpoint consisted in the inability to restart the AVNRT even after isoprenaline infusion.

Statistical analysis

Obtained results are expressed as mean ± SD. Comparison between continuous variables were performed by the Student t test while proportion comparison was performed by the Fischer exact test.

Results

General and clinical characteristics

According to the general and clinical characteristics (Table 1) patients with AVRT were younger at symptom onset, had a lower prevalence of associated heart disease and a lower percentage of correct diagnosis of the arrhythmia made on surface ECG. The symptomatic profile during the tachycardia episodes (Table 2) and the presence of precipitating factors such as physical activity, emotional stress and postural changes did not differ between the two groups, with the exception of fatigue and sweating.

Electrophysiological characteristics

Significant electrophysiological characteristics differences were seen regarding: AH interval, antegrade Wenckebach point, modality of induction of the tachycardias during electrophysiological study and induced tachycardia cycle length (Table 3). Sustained atrial fibrillation induction did not differ between the two groups. No patient had dual diagnoses (inducibility of both types of tachycardia). We did not evaluate in a systematic way possible differences between the two groups regarding intra–inter atrial conduction properties. The concealed accessory pathways were located on the left side in 93% of the cases and in the remaining 7% on the right side. None of them showed decremental type conduction patterns.

Catheter ablation results, procedural complications, tachycardia recurrence

Using the same number of energy applications, catheter ablation success rate was similar in the two groups. Procedural complications were observed in a similar percentage of patients procedure-related complications were observed 4% in those with a concealed accessory

Table 1 Patients' general and clinical characteristics

	AVRT 46 patients	AVNRT 48 patients	*p*
Gender, males	65%	50%	ns
Heart disease	8%	31%	=0.01
Age of onset of PSVT (years)	25±16	37±17	=0.001
Age at ablation (years)	48±14	53±16	ns
Symptom duration (years)	22±16	17±13	ns
Annual incidence of PSVT	13±21	24±67	ns
PSVT incidence in the past 12 months	38±68	51±100	ns
Correct ECG diagnosis*	50%	79%	=0.001
Minimal PSVT duration (mn.)	16′±28′	16′±21′	ns
Maximal PSVT duration(mn.)	237′±515′	238′±304′	ns
Precipitating factors	30%	35%	ns
Spontaneous atrial fibrillation episodes	11%	8%	ns

AVRT, atrioventricular reentrant tachycardia; AVNRT, atrioventricular nodal reentrant tachycardia; PSVT, paroxysmal supraventricular tachycardia.

*Diagnosis was based on RP′ interval evaluation: tachycardia with RP′<70 ms was classified as AVNRT; tachycardia with RP′≥70 ms was classified as AVRT.

pathway (one transient cerebral ischaemic attack and one episode of ventricular fibrillation, induced by a burst of premature ventricular complexes in a patient with ischaemic heart disease during catheter manipulation) and 2% in patients with AVNRT (one transient atrioventricular block). There were no late complications. During the 12±6 month follow-up period tachycardia recurrence was similar in the two groups; these patients underwent a successful additional procedure (Table 3).

Discussion

The aim of the present study was to compare clinical and electrophysiological characteristics between two groups of consecutive patients, one with AVRT due to concealed accessory pathway and the other with AVNRT. The main results of the study can be summarized as follows.

Table 2 PSVT symptom profile

	AVRT 46 patients	AVNRT 48 patients	*p*
Palpitation	100%	100%	ns
Fatigue	24%	59%	=0.001
Dyspnoea	26%	46%	ns
Presyncope	50%	60%	ns
Syncope	8.7%	8.3%	ns
Headache	2%	6%	ns
Feeling warm	4.3%	6.2%	ns
Confusion	6.5%	4.1%	ns
Sweating	10.8%	37.5%	=0.006
Chest discomfort	21.7%	39.6%	ns

AVRT, atrioventricular reentrant tachycardia; AVNRT, atrioventricular nodal reentrant tachycardia; PSVT, paroxysmal supraventricular tachycardia.

Patients with AVRT had a lower mean age at the arrhythmia symptom onset compared with those with AVNRT and this finding is similar to that found by other authors[3,5,8–10,16]. The reason is not precisely known but it seems to be due to the fact that AVRT has a well defined circuit tachycardia, which is congenital and, therefore, has a greater probability of earlier activation, while AVNRT may require more time before the circuit pathways (slow nodal pathway, fast nodal pathway and perinodal atrium) acquire the electrophysiological properties necessary to allow sustained reentry.

We observed a greater prevalence of organic heart disease (especially the ischaemic-hypertensive type) in patients with AVNRT. This finding was confirmed by other studies where, on average, 45% of the patients with AVNRT and 18% with AVRT had heart disease[3,8,10]. This can possibly be explained by the fact that patients with AVNRT were older.

A greater number of AVNRT were recognized on the surface ECG compared with AVRT. To distinguish the two types of tachycardia we did not use any particular algorithms[17] but only the RP′ interval duration (a value <70 ms was indicative of nodal tachycardia and ≥70 ms of concealed tachycardia). In our study population, many cases of AVRT had a RP′ interval of nearly 70 ms and were erroneously classified as AVNRT. The difficulty in correctly identifying the retrograde P wave could have contributed to this diagnostic error.

Although fatigue and sweating were seen more frequently in patients with AVNRT, the symptomatic profiles of the tachycardia were similar in the two groups. In particular we did not see any significant difference in the incidence of the episodes associated with haemodynamic instability and syncope. Haemodynamic deterioration induced by the tachycardia was correlated with a higher heart rate and atrial contraction with atrioventricular valves closed which are more commonly seen in tachycardias with VA <200 ms and

Table 3 Electrophysiological characteristics and catheter ablation results

	AVRT 46 patients	AVNRT 48 patients	*p*
AH interval (ms)	77 ± 16	85 ± 24	= 0.04
HV interval (ms)	38.7 ± 7.6	42.8 ± 6.2	ns
Antegrade AV node ERP (ms)	287 ± 97	273 ± 78	ns
Atrial ERP (ms)	207 ± 28	213 ± 27	ns
Antegrade WP (ms)	330 ± 79	365 ± 77	= 0.03
Retrograde AV node ERP (ms)	302 ± 79	280 ± 85	ns
Atrial stimulation induction	81%	94%	ns
Ventricular stimulation induction	66%	9%	= 0.001
Baseline induction	91%	62%	= 0.002
Isoprenaline induction	9%	38%	= 0.001
Inducted PSVT cycle length (ms)	329 ± 46	363 ± 55	= 0.002
Atrial fibrillation induction	26%	25%	ns
Number of RF applications	7.5 ± 7.4	8.2 ± 6.6	ns
Ablation success	91%	98%	ns
Procedural complications	4%	2%	ns
PSVT relapse	6%	6%	ns

AVRT, atrioventricular reentrant tachycardia; AVNRT, atrioventricular nodal reentrant tachycardia; PSVT, paroxysmal supraventricular tachycardia; AH, atrial potential–His potential interval; HV, His potential–ventricular potential interval; AV, atrioventricular; ERP, effective refractory period; WP, Wenckebach point; RF, radiofrequency.

therefore in patients with AVNRT[18]. However, this was not confirmed in our study population. In accordance with our findings, Wood *et al.*[10] noticed that the incidence of syncope was similar in the two types of tachycardia (16% in patients with AVNRT and 27% in patients with AVRT; p = ns) and was significantly correlated with a heart rate $\geq$170 bpm. It is, however, probable, as recently suggested[19], that syncope during tachycardia is due to vasovagal activation and that an altered cardiovascular reflex status plays a more important role in determining episodes of syncope during tachycardia than the heart rate and the tachycardia mechanism.

Regarding the electrophysiological characteristics, some differences between the two groups were seen in AH intervals, antegrade Wenckebach cycle and induced tachycardia cycle length. A multivariate analysis was not performed, but it is possible that these differences derived from the different age of the two groups of patients and were not correlated with the mechanism of the arrhythmia. There were no significant differences regarding other characteristics, such as gender, annual incidence, incidence during the year prior to ablation (a significant increase was observed in both groups), duration of the tachycardia episodes, precipitating factors, association with spontaneous episodes of atrial fibrillation, rate of catheter ablation success, procedural complication and late recurrence of the tachycardia. These findings are consistent with those reported in the literature[3,5,7,10,13,20,21].

Study limitation

This study was conducted with a relatively small number of patients and this could have rendered some differences insignificant between the two groups. Patient history was taken in some cases years after the beginning of the symptoms and might have been incorrectly given by the patient. The study was conducted in tertiary arrhythmological centres where the majority of patients are more symptomatic and refractory to pharmacological therapy and, therefore, may not reflect the true natural history of patients with these types of tachycardia in the general population.

References

[1] Josephson M, Buxton A, Marchlinski F. The tachyarrhythmias. In: Isselbacher K, Braunwald E, Wilson JD, eds. Harrison's Principles of Internal Medicine. 13th edn. New York: McGraw-ill 1994: 1024–9.
[2] Calkins H, Sousa J, El-Atassi R, *et al.* Diagnosis and cure of the Wolff–Parkinson–White Syndrome or paroxysmal supraventricular tachycardias during a single electrophysiologic test. N Engl J Med 1991; 324: 1612–8.
[3] Wu D, Denes P, Amat-y-Leon F, *et al.* Clinical, electrocardiographic and electrophysiologic observations in patients with paroxysmal supraventricular tachycardia. Am J Cardiol 1978; 41: 1041–51.
[4] Sintetos AL, Roark SF, Smith MS, McCarthy EA, Lee KL, Pritchett EL. Incidence of symptomatic tachycardia in untreated patients with paroxysmal supraventricular tachycardia. Arch Intern Med 1986; 146: 2205–9.
[5] Rodriguez LM, de Chillou C, Schlapfer J, *et al.* Age at onset and gender of patients with different types of supraventricular tachycardias. Am J Cardiol 1992; 70: 1213–5.
[6] Ko JK, Deal BJ, Strasburger JF, Benson DW Jr. Supraventricular tachycardia mechanisms and their age distribution in pediatric patients. Am J Cardiol 1992; 69: 1028–32.
[7] Clair WK, Wilkinson WE, McCarthy EA, Page RL, Pritchett EL. Spontaneous occurrence of symptomatic paroxysmal atrial fibrillation and paroxysmal supraventricular tachycardia in untreated patients. Circulation 1993; 87: 1114–22.
[8] Hamer ME, Wilkinson WE, Clair WK, Page RL, McCarthy EA, Pritchett ELC. Incidence of symptomatic atrial fibrillation in patients with paroxysmal supraventricular tachycardia. J Am Coll Cardiol 1995; 25: 984–8.
[9] Goyal R, Zivin A, Souza J, *et al.* Comparison of the ages of tachycardia onset in patients with atrioventricular nodal

reentrant tachycardia and accessory pathway-mediated tachycardia. Am Heart J 1996; 132: 765–7.
[10] Wood KA, Drew BJ, Scheinman MM. Frequency of disabling symptoms in supraventricular tachycardia. Am J Cardiol 1997; 79: 145–9.
[11] Tada H, Oral H, Greenstein R, *et al*. Analysis of age of onset of accessory pathway-mediated tachycardia in men and women. Am J Cardiol 2002; 89: 470–1.
[12] Della Bella P, Brugada P, Talajic M, *et al*. Atrial fibrillation in patients with an accessory pathway: importance of the conduction properties of the accessory pathway. J Am Coll Cardiol 1991; 17: 1352–6.
[13] Chen SA, Chiang CE, Tai CT, *et al*. Longitudinal clinical and electrophysiological assessment of patients with symptomatic Wolff–Parkinson–White syndrome and atrioventricular node reentrant tachycardia. Circulation 1996; 93: 2023–32.
[14] Bottoni N, Donateo P, Tomasi C, *et al*. Confronto delle caratteristiche cliniche ed elettrofisiologiche dei pazienti con via accessoria atrioventricolare occulta e manifesta. Ital Heart J Suppl 2001; 2: 888–93.
[15] Haïssaguerre M, Gaita F, Fischer B, *et al*. Elimination of atrioventricular nodal reentrant tachycardia using discrete slow potentials to guide application of radiofrequency energy/ contribution > . . Circulation 1992; 85: 2162–75.
[16] Akhtar M, Jazayeri MR, Sra J, Blanck Z, Deshpande S, Dhala A. Atrioventricular nodal reentry. Clinical, electrophysiological, and therapeutic considerations. Circulation 1993; 88: 282–95.
[17] Tai CT, Chen SA, Chiang CE, *et al*. A new electrocardiographic algorithm using retrograde P waves for differentiating atrioventricular node reentrant tachycardia from atrioventricular reciprocating tachycardia mediated by concealed accessory pathway. J Am Coll Cardiol 1997; 29: 394–402.
[18] Goldreyer BN, Kastor JA, Kershbaum KL. The hemodynamic effects of induced supraventricular tachycardia in man. Circulation 1976; 54: 783–9.
[19] Brembilla-Perrot B, Beurrier D, Houriez P, Claudon O, Wertheimer J. Incidence and mechanism of presyncope and/ or syncope associated with paroxysmal junctional tachycardia. Am J Cardiol 2001; 88: 134–8.
[20] Jazayeri MR, Hempe SL, Sra JS, *et al*. Selective transcatheter ablation of the fast and slow pathways using radiofrequency energy in patients with atrioventricular nodal reentrant tachycardia. Circulation 1992; 85: 1318–28.
[21] Calkins H, Yong P, Miller JM, *et al*. Catheter ablation of accessory pathways, atrioventricular nodal reentrant tachycardia, and the atrioventricular junction final results of a prospective, multicenter clinical trial. Circulation 1999; 99: 262–70.