

Multiple AV nodal pathways in patients with AV nodal reentrant tachycardia — more common than expected?

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Aims It was the purpose of this study to determine the incidence of more than two AV nodal pathways in patients with AVNRT.

Methods and results In 78 consecutive patients with AV-nodal reentrant tachycardias (AVNRT) (50 females, 28 males, mean age 52.8 ± 14.6 years), the number of sudden AH increases by 50 ms or more (AH-jump) was analysed during atrial extrastimulation. The incidence of two AV nodal pathways was accepted to be present in patients with AVNRT without an AH-jump ('smooth curve').

The following forms of tachycardia were induced: a typical AVNRT (slow-fast) in 67 patients, an atypical AVNRT (fast-slow) in 12 patients and a slow-slow-AVNRT in 4 patients. Five patients had two forms of AVNRT. 47 patients (60.3%) showed two AV nodal pathways, 27 patients (34.6%) had three AV-nodal pathways and 4 patients (5.1%) exhibited four AV-nodal pathways.

For successful catheter ablation of AVNRT in patients with more than two pathways, more radiofrequency energy applications were required (9.2 ± 6.3) compared with patients with only two pathways (6.7 ± 4.8). Furthermore, in patients with more than two AV-nodal pathways, the catheter intervention resulted more frequently in a modulation of slow pathway conduction than in an ablation of the slow pathway(s).

Conclusion The incidence of more than two AV-nodal pathways in patients with AVNRT was unexpectedly high at about 40%. Thus, these tachycardias require a meticulous electrophysiological evaluation for successful ablation.

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Key Words: AVNRT, multiple AV-nodal pathways, catheter ablation.

Introduction

AV nodal reentrant tachycardia (AVNRT) is the most common form of paroxysmal regular supraventricular tachycardia occurring in about 60% of these patients^[3,5,13]. The presence of at least two AV nodal pathways, characterized by different refrac-

tory periods and conduction properties, is the anatomical prerequisite for the occurrence of these tachycardias^[1,6–9,11,16].

Irrespective of many excellent reviews on AVNRT, there are only few systematic analyses of the incidence of more than two atrioventricular pathways in patients with AVNRT. The purpose of this study was to determine the number of AV nodal pathways in patients with AVNRT.

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Methods

Study population

Seventy-eight consecutive patients referred for catheter ablation with paroxysmal supraventricular tachycardia corresponding to an AVNRT were included in this

study. In 76 of these patients, AVNRT could be induced by programmed stimulation, whereas in 2 patients the diagnosis was confirmed by demonstration of distinctive AV nodal conduction pattern in addition to the event ECG of a regular supraventricular tachycardia^[4].

Electrophysiological study

On the day before the investigation, all patients gave written, informed consent to undergo the electrophysiological study and catheter ablation when needed (in a 16-year-old patient, written consent was given by the parents). All anti-arrhythmic drugs were stopped more than five elimination half-lives before study.

All patients underwent basic electrophysiological study in the unsedated state, the procedure was performed after injection of 5000 IU heparin iv. If necessary, diazepam iv (5–10 mg) and/or morphine iv (2.5–5 mg) were used for sedation during the procedure.

Two 6-French quadripolar electrode catheters (Josephson-curve, Bard Corp., Billerica, MA, U.S.A.) with a 4 mm interelectrode space were introduced through a femoral vein into the right atrial appendage and across the tricuspid valve to record the His bundle potential, and a 5-French bipolar electrode (Cournand-curve, Cordis Corp., MI, U.S.A.) was introduced through the same vein and advanced into the right ventricular apex. Additionally, a 6-French deca-polar electrode (Bard Corp., Billerica, MA, U.S.A.) with 2 mm interelectrode spacing was introduced through a left antecubital vein and placed in the proximal coronary sinus. Intracardiac electrograms (EGMs) and the surface leads I, II, V1 and V6 were displayed on a multichannel screen (Bard Corp., Lab Systems Vs. 2.57) at a maximum paper speed of 200 mm/s, and all EGMs acquired (12 surface and up to 16 intracardiac channels) were stored on an optical disc drive. The bandpasses were set between 30 Hz and 500 Hz.

Programmed pacing at a pulse duration of 2.0 ms was performed using a programmable stimulator (UHS-20, Biotronik Corp., Lörrach, Germany). Atrial and ventricular pacing thresholds were determined before starting programmed stimulation, accepting levels between 0.5–1.5 V, and the output of the stimulator was set at twice diastolic pacing threshold.

The standardized stimulation protocol included:

- (1) evaluation of sinus node refractory time at different cycle lengths,
- (2) determination of atrial and antegrade atrioventricular refractory periods using single atrial extrastimuli with decremental shortening of coupling interval in 10-ms steps during sinus rhythm and at 600, 500 and 400 ms basic drive cycle lengths,
- (3) incremental atrial and ventricular pacing for determination of antegrade and retrograde AV nodal conduction characteristics,

- (4) determination of ventricular and retrograde atrioventricular refractory periods using single ventricular extrastimuli shortened in 10-ms decrements at 500 ms basic drive cycle length.

If dual AV nodal conduction behaviour could not be shown, or if an AVNRT was not induced using single atrial stimuli, double stimuli were applied likewise in 10-ms decrements. Furthermore, orciprenaline 0.25–0.5 mg (metaproterenol, Alupent[®], Boehringer Corp., Ingelheim, Germany) was used at dosages which are able to increase spontaneous heart rate by 50% of basic rate, and the stimulation protocol was repeated.

Differentiation from other forms of tachycardias

The differentiation of suspected AVNRT from other forms of supraventricular tachycardias (AV reentry, atrial tachycardia) was performed by the classic differential stimulation as previously described^[7,8,17,24].

Mapping

Primary target for ablation was the atrial insertion of the slow pathways^[7,9,10]. A deflectable 7-French quadripolar catheter with 4 mm tip size (Cordis-Webster Corp., Watertown, MA, U.S.A.) was placed through a long 8F sheath (DAIG Corp., Minnetonka, MN, U.S.A.) at the base of the triangle of Koch at the assumed slow pathway insertion. Initially, the electrode was placed in a posterior position, achieving a ratio of atrial to ventricular potential of 0.1–0.5. We looked for a local late high frequency signal representing the potential of the slow pathway^[12].

Ablation

Radiofrequency current was generated from an EP-Shuttle system (Stockert, Cordis-Webster Corp., Watertown, MA, U.S.A.). Ablation was performed in a temperature-guided mode (preselected maximum temperature at 70°C) applying a stepwise upward approach. Energy was limited at 30 W with a maximum time period of 60 s.

The occurrence of accelerated junctional rhythms during energy delivery was considered as an indicator of possible successful ablation^[2,20]. In case of prolongation of PR-interval, occurrence of AV or VA conduction block, dislocation of the catheter, sudden rise of impedance^[14] or atrioventricular dissociation during accelerated junctional rhythm, energy delivery was stopped immediately to avoid permanent AV conduction block^[14]. Noninducibility of the AVNRT was set as endpoint for ablation. The persistence of AH jumps or single echo beats during control stimulation however, was accepted as successful therapy of AVNRT^[15,19].

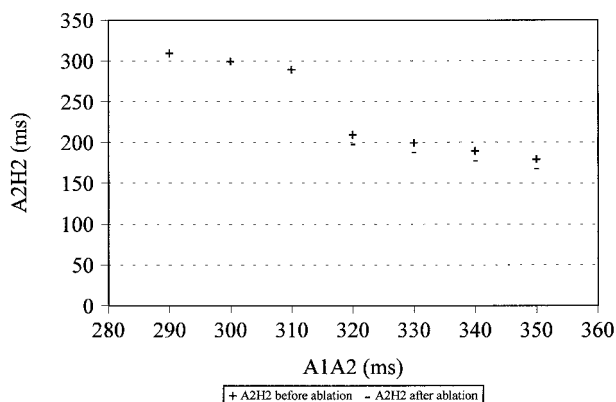


Figure 1 AV nodal conduction curve A1A2 vs A2H2 in a patient with two AV nodal pathways. At the effective refractoriness of the fast pathway at 310 ms, a sudden increase in A2H2 indicates further conduction via the slow pathway. After ablation, there is no slow pathway conduction.

After successful ablation, an electrophysiological study was repeated 30 min after the last radiofrequency energy application without and with administration of orciprenaline (0.5–1 mg) to prove noninducibility of AVNRT^[23].

Definitions

Dual atrioventricular conduction was defined by a sudden increase in A2–H2 interval (>50 ms, AH jump) at a critical coupling interval A1–A2 applying atrial extrastimuli shortened in 10-ms decrements, leading to a discontinuity in the atrioventricular conduction curve A1–A2 vs A2–H2 (Fig. 1). The longest A1–A2 interval after the AH jump characterized the effective refractory period of the fast pathway.

The occurrence of multiple atrioventricular conduction pathways was defined as being present, if more than one sudden increase in A2–H2 interval during atrial extrastimulus testing was detected, resulting in two or three discontinuities in the atrioventricular conduction curve (Fig. 2).

The target sites for catheter ablation was or were the slow pathway(s) of the AV node. Noninducibility of AV nodal reentrant tachycardia was the primary endpoint. Differentiation between ablation versus modulation of the target pathway was performed according to the result of the control stimulation 30 min after the last energy application to describe correctly the result of the ablation procedure.

Modulation of the pathway

Noninducibility of the AVNRT, but persistence of discontinuous atrioventricular conduction (persistent AH jump) and/or appearance of only one echo beat. The

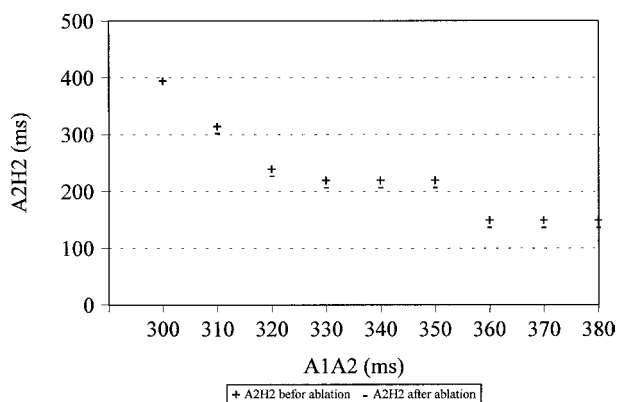


Figure 2 AV nodal conduction curve A1A2 vs A2H2 in a patient with four AV nodal pathways. At the effective refractoriness of the fast pathway at 360 ms, a sudden increase in A2H2 indicates further conduction via the first slow pathway. The following sudden increases in A2H2 indicate the existence of a second and third slow pathway. After ablation, there is a persistent slow pathway conduction in slow pathways one and two (which were modulated), whereas slow pathway three shows no conduction.

result of the catheter intervention was regarded as a modification of AV nodal conduction by modulation of slow pathway conduction.

Ablation of the pathway

Noninducibility of the AVNRT, no evidence for slow pathway conduction: no discontinuity in atrioventricular conduction (no AH-jump), no AV nodal echo beats; increase in the effective refractory period of the AV node.

Statistics

Data are presented as means \pm SD. Statistical analysis was performed using Student's t-test for unpaired variables including F-test for analysis of variance to compare two sets of data, multiple analysis of variance was used to compare more than two sets of data. A *P*-value of <0.05 was considered to be significant.

Results

Patients

The study population consisted of 78 patients with AVNRT who underwent electrophysiological study and catheter ablation using radiofrequency current. There were 50 women (64.1%) and 28 men (35.9%), ranging in age from 16–82 years (52.8 ± 14.6 years).

All these patients presented paroxysmal supra-ventricular tachycardias documented by ECG. Before electrophysiological study, all patients underwent clinical investigation, surface-ECG, echocardiography and chest X-ray before electrophysiological study to evaluate

Table 1 Size of sudden AH increases (AH-jump) in patients with 2, 3 and 4 antegrade AV nodal pathways

Number of AV nodal pathways	AH-jump 1 (ms)	AH-jump 2 (ms)	AH-jump 3 (ms)
2	97 ± 58		
3	82 ± 44	94 ± 65	
4	117 ± 42	87 ± 36	147 ± 129
All patients	92 ± 52	93 ± 52	147 ± 129

potential structural heart disease. None of these patients had structural heart disease where treatment was necessary, and all patients showed normal left ventricular function on echocardiography.

Multiple AV nodal conduction: sudden increase (AH jump) and smooth-curve conduction

Programmed atrial stimulation using atrial extrastimuli with a coupling interval shortened in 10-ms decrements in sinus rhythm and at basic drive cycle lengths set to 600, 500 and 400 ms revealed distinctive antegrade AV nodal conduction in 67 (85.9%) patients (Table 1). The existence of at least two antegrade AV nodal pathways without demonstration of a sudden increase in AV nodal conduction ('smooth curve') was proven by induction of AVNRT^[18] in 11 patients (14.1% of the patients). In these patients, the existence of two antegrade AV nodal pathways was assumed (in two of them, a sudden increase in AV nodal conduction could be shown during control stimulation after radiofrequency ablation).

The numbers of antegrade AV nodal pathways in the 78 patients were distributed as follows: 47 patients (60.3%) showed two AV nodal pathways, 27 patients (34.6%) had three AV-nodal pathways and 4 patients (5.1%) exhibited four AV-nodal pathways (Table 1).

The corresponding refractory periods of the fast and slow pathways, ascertained by the atrial extrastimulus technique with 10-ms decrements in sinus rhythm and basic drive cycle lengths of 600, 500 and 400 ms, are summarized in Table 2a–2c.

Statistical analysis could not be performed in patients with four AV nodal pathways because of the occurrence of the third 'AH jump' at different basic drive cycle lengths at 600, 500 and 400 ms, respectively.

Different forms of tachycardia

The following forms of tachycardia were induced: a typical AVNRT (slow-fast) in 67 patients, an atypical AVNRT (fast-slow) in 12 patients and a slow-slow-AVNRT in 4 patients. In 5 patients, two forms of

AVNRT could be induced. Separated by the number of pathways, the distribution of the different forms of AVNRT was as follows:

Of the 47 patients with two AV nodal pathways, 39 patients (83.0%) showed a typical AVNRT and 8 patients (17.0%) an atypical AVNRT. Actually, these patients presented only one form of tachycardia.

Of the 27 patients displaying three AV nodal pathways, the majority (24 patients, 88.9%) exhibited the slow-fast form of AVNRT, whereas the fast-slow form occurred just as often as the slow-slow form in 4 patients, respectively. In 22 patients however, only one single form of AVNRT could be induced: in 19 patients a slow-fast form, in only 1 patient a fast-slow form and in 2 patients a slow-slow form of AVNRT. In the 5 patients with two different forms of AVNRT, 3 patients showed both a slow-fast and a fast-slow form of AVNRT, and 2 patients had a slow-fast and a slow-slow form of AVNRT.

Surprisingly, all 4 patients with four pathways had only the slow-fast form of AVNRT.

Relation between the number of pathways and the different forms of AVNRT

Table 3 shows the number of AV nodal pathways in the different forms of tachycardia. According to its definition, the slow-slow form of AVNRT is limited to the presence of at least two slow AV nodal pathways which are used for antegrade and retrograde conduction during tachycardia.

Comparison of cycle lengths of the different forms of AVNRT

There was no significant difference in the cycle lengths of the typical and the atypical form of AVNRT ($P=0.35$, Table 4), whereas the cycle lengths of slow-slow form tachycardias were expectedly longer compared with the other forms of AVNRT ($P=0.02$, Table 4). The cycle lengths of the atypical form of AVNRT in patients with three AV nodal pathways (3 patients, in 1 patient, there was no documented cycle length of tachycardia) were significantly longer than in patients with only two pathways (8 patients, Table 4).

Results of catheter ablation

In 39 of the 47 patients with two AV nodal pathways (83.0%) radiofrequency ablation was performed, resulting in ablation of the slow pathway in 18 patients (46%) and in modulation of slow pathway conduction in 21 patients. 24 (89.8%) of the 27 patients with three AV nodal pathways underwent catheter ablation. No evidence of slow pathway conduction (i.e. ablation) was observed in seven patients (29.1%), and residual slow

Table 2a Effective refractory periods of the fast pathway at different cycles lengths

Number of AV nodal pathways	ERP-fast pathway (ms) at 400 ms CL	ERP-fast pathway (ms) at 500 ms CL	ERP-fast pathway (ms) at 600 ms CL
2	310 ± 46	328 ± 57	337 ± 49
3	302 ± 40	322 ± 44	339 ± 76
4	330 ± 40	365 ± 81	345 ± 49

Abbreviations: ERP=Effective refractory period, CL=cycle length S1S1.

Table 2b Effective refractory periods of the first slow pathway at different cycle lengths

Number of AV nodal pathways	ERP-slow pathway 1 (ms) at 400 ms CL	ERP-slow pathway 1 (ms) at 500 ms CL	ERP-slow pathway 1 (ms) at 600 ms CL
2	270 ± 39	302 ± 46	278 ± 28
3	251 ± 37	290 ± 45	302 ± 52
4	316 ± 45	300 ± 42	320 ± 28

Abbreviations: ERP=effective refractory period, CL=cycle length S1S1.

Table 2c Effective refractory periods of the second slow pathway at different cycle lengths

Number of AV nodal pathways	ERP-slow pathway 2 (ms) at 400 ms CL	ERP-slow pathway 2 (ms) at 500 ms CL	ERP-slow pathway 2 (ms) at 600 ms CL
3	265 ± 7	273 ± 34	280 ± 30
4	305 ± 64	290 ± 52	

Abbreviations: ERP=effective refractory period, CL=cycle length S1S1.

Table 3 Occurrence of the different forms of AVNRT corresponding to the number of AV nodal pathways

Number of AV nodal pathways	Typical AVNRT (n=67)	Atypical AVNRT (n=12)	'Slow-slow' AVNRT (n=4)
2	39	8	0
3	24	4	4
4	4	0	0

pathway conduction (i.e. modulation) was found in 17 patients (70.9%). Residual slow pathway conduction was found as follows: modulation of slow pathway 1 and ablation of slow pathway 2 in 8 patients, ablation of slow pathway 1 and modulation of slow pathway 2 in 2 patients and modulation of both slow pathway 1 and slow pathway 2 in 7 patients. Catheter ablation in 2 patients with four AV nodal pathways could not eliminate residual slow pathway conduction (i.e. modulation): in 1 patient modulation of slow pathway 1 and ablation of slow pathway 2 and 3 was obtained; in the other patient slow pathway 1 and 2 were modulated and slow pathway 3 was ablated.

Persistent total AV conduction block occurred in only one case, a patient with two AV nodal pathways.

Radiofrequency ablation was avoided in some cases due to a planned second session (patient's request), non-sustained tachycardia or non-reproducibility of tachycardia, occurrence of atrial flutter or fibrillation or because of the patient's decision in favour of medical treatment.

Number of energy applications

The number of RF energy deliveries required to achieve the endpoint of ablation (noninducibility of tachycardia) was higher with a greater number of AV nodal pathways: 6.7 ± 4.8 RF energy applications in patients with two AV nodal pathways, 8.9 ± 6.5 RF energy

Table 4 Cycle length of tachycardias

Number of AV nodal pathways	Typical AVNRT cycle length (ms)	Atypical AVNRT cycle length (ms)	'Slow-slow' AVNRT cycle length (ms)
2	357 ± 68	353 ± 69	
3	381 ± 83	476 ± 50*	482 ± 28
4	337 ± 64		
All patients	364 ± 73	387 ± 85	482 ± 28#

* $P=0.008$ (cycle length of atypical AVNRT in patients with two AV nodal pathways vs cycle length of atypical AVNRT in patients with three AV nodal pathways).

$P=0.02$ (cycle length of 'slow-slow' AVNRT vs cycle length of typical and atypical form of AVNRT, all patients).

Table 5 Number of AV nodal pathways in relation to the result of RF ablation

Number of AV nodal pathways	Number of RF energy deliveries		
	Ablation	Modulation	
2	5.8 ± 5.2	7.5 ± 4.5	$P=0.28$
3	8.1 ± 4.3	9.3 ± 7.4	$P=0.71$
4		13 ± 4.2	

applications in patients with three AV nodal pathways and 13.0 ± 4.2 RF energy applications in patients with four AV nodal pathways. This was not statistically significant ($P=0.12$).

Comparing the number of energy deliveries required for successful ablation in patients with only two AV nodal pathways (6.7 ± 4.8) versus patients with more than two AV nodal pathways (9.2 ± 6.3), there was a stronger tendency ($P=0.07$) supporting the assumption that the existence of more pathways would require more energy deliveries for successful treatment of AV nodal reentrant tachycardias. In contrast to our expectations, 'ablation' was achieved with fewer energy deliveries than 'modulation', in patients with two as well as in patients with more than two AV nodal pathways. These data are shown in Table 5.

Discussion

Incidence of multiple AV nodal pathways in patients with AVNRT

The results of this study in 78 patients with AV nodal reentrant tachycardia revealed an incidence of more than two AV nodal pathways of almost 40%, and is unexpectedly high.

These data are in contrast to data previously reported by Tai *et al.*^[21] with only 5.2%. In the study of Tai *et al.*, the atrial stimulation protocol included only two basic drive cycle lengths for atrial extrastimulus testing, whereas in the present study usually three — but up to four — basic cycle lengths were used (sinus rhythm: 600, 500 and 400 ms as basic drive cycle lengths).

Furthermore, Tai *et al.* used atropine or isoprenaline to provoke critical conduction properties without mentioning any parameter to control adequate dosage. In the present study, orciprenaline (metaproterenol) was used in such a dosage that sinus rate was increased by at least 50%.

However, this might not be sufficient to explain the marked difference in AV nodal pathway count. It is known to us that there is no 'gold standard' for the proof of presence of an AV nodal pathway using the AH conduction curve. We also know that a 'jump' at the end of the curve is discussed as not reflecting the presence of an additional pathway. However, for this study, we used the definition of dual AV-nodal physiology as a sudden increase in AH interval of ≥ 50 ms, also used by Tai *et al.*^[21], so as not to overlook an AV nodal pathway. This definition for the presence of multiple AV nodal pathways may lead to overestimation of the number of AV nodal pathways. However, there is evidence that the findings of Tai *et al.* (which show only 5.2% of patients with multiple AV nodal reentrant tachycardias) is an underestimation of the presence of more than two AV nodal pathways in those patients. This may be due to the different stimulation protocol Tai *et al.* used. There are findings in another patient cohort studied by our group, which support our data. Of 250 patients with atrial fibrillation, 97 patients gave evidence of more than one AV nodal pathway in the RR interval histogram of the Holter ECG^[22]. A distinct RR interval peak identified each AV nodal pathway. In 17 (18%) of these 97 patients, the findings were in accordance to the presence of more than two AV nodal pathways, which is far more than in the patient group of Tai *et al.*. Thus, the results of our study support the assumption, that the presence of multiple AV nodal pathways in patients with AV nodal reentrant tachycardias is higher than expected.

More pathways — more forms of AV nodal reentrant tachycardias?

Tai *et al.*^[21] reported an incidence of more than one form of AV nodal reentrant tachycardia in the same patient in 36 of 550 cases, representing 6.5% of these patients. In the present study, the percentage of

two forms of tachycardia was comparable at 6.4%. Interestingly, since the occurrence of two forms of AV nodal reentrant tachycardia was found only in patients with more than two AV nodal pathways, it can be suggested that in patients with two different forms of AV nodal reentrant tachycardia more than two AV nodal pathways might exist. On the other hand, the existence of more than two AV nodal pathways does not necessarily lead to the occurrence of more than one form of AV nodal reentrant tachycardia. In our patient population, there were only five of the 27 patients (18.5%) with three AV nodal pathways or five of 31 patients (16.1%) with three or more AV nodal pathways, who showed two different forms of AV nodal reentrant tachycardia. This can lead to the conclusion that most additional slow pathways may be considered to be 'innocent bystanders'. Further investigations are necessary to evaluate the importance of these additional pathways with regard to the recurrence rate of AV nodal reentrant tachycardias after RF ablation.

Differences in cycle length in different types of AV nodal reentrant tachycardia

Similar mean cycle lengths were found in patients with the typical as well as with the atypical form of AVNRT, which can be explained by a reentrant circuit consisting of the fast and the slow AV nodal pathway, irrespective of the direction in conduction in each pathway. However, the mean tachycardia cycle lengths of slow-slow AV nodal reentrant tachycardias were longer, due to the slower conduction velocity of the two slow pathways forming the reentrant circuit.

The difference of the mean cycle lengths of the atypical form of AVNRT in patients with two versus patients with three AV nodal pathways may be due to the participation of the slower of the two slow pathways within the reentrant circuit in the latter patient group.

Ablation vs modulation: is the number of pathways relevant?

RF current applications targeting the slow pathway resulted in a relatively low rate of ablation, compared with the results of Jazayeri *et al.*^[10], who could eliminate slow pathway conduction in 35 of 60 patients (58.4%) with AV nodal reentrant tachycardia.

In the present study, in patients with two AV nodal pathways the rate of ablation was 46%, whereas in patients with three AV nodal pathways the complete elimination of slow pathway conduction was achieved in only 29.1%, and in none of the patients with four AV nodal pathways. Since these data show a marked decrease in ablation rate in patients with more than two AV nodal pathways, the number of slow pathways seems to be relevant for catheter ablation of AV nodal reentrant tachycardias.

Conclusions

In this study, the incidence of multiple AV nodal pathways in patients with AVNRT was unexpectedly high, almost 40%. The presence of multiple AV nodal pathways in patients with AVNRT can be revealed by subtle electrophysiological evaluation using a standard stimulation protocol before and after catheter ablation. To ascertain the real number of pathways in each case is clinically relevant, since catheter ablation in patients with multiple pathways is more difficult and may require more energy applications.

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