Multiple mechanisms of successful slow-pathway catheter ablation of common atrioventricular nodal re-entrant tachycardia

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Background In patients with atrioventricular nodal re-entrant tachycardia, modifications of the antegrade atrioventricular nodal function curve caused by catheter ablation of the so-called slow pathway are heterogeneous, but have not yet been systematically evaluated.

Aim To test the hypothesis that successful treatment is independent of specific electrophysiological modifications of atrioventricular nodal conducting properties.

Method Standard electrophysiological parameters and comparable antegrade atrioventricular nodal function curves were obtained, before and after successful ablation, in 104 patients (mean age 52 ± 16 years; 69 women) affected by the common form of atrioventricular nodal re-entrant tachycardia.

Results Three different major patterns of antegrade atrioventricular nodal function curve were caused by ablation: downward shift of the curve with disappearance of atrioventricular nodal duality, suggesting the elimination of the slow pathway in 54 (52%) patients (type 1); absence of clear modifications of the curve (and of slow pathway ablation) in 33 (32%) patients (type 2); upward shift of the curve, suggesting a further slowing of conduction velocity through the slow pathway in 17 (16%) patients (type 3). Type-1 pattern was more frequent in patients <45 years, whereas type-2 pattern was more frequent in those >45 years.

Conclusion Successful ablation of atrioventricular nodal re-entrant tachycardia is independent of specific modifications of antegrade atrioventricular conduction and probably depends on critical nodal and perinodal tissue damage at different sites on the re-entrant circuit. The effects of ablation are influenced by patient age.

Key Words: Atrioventricular nodal re-entrant tachycardia, catheter ablation, electrophysiological evaluation.

Introduction
A dual nodal physiology is frequently observed in patients affected by atrioventricular nodal re-entrant tachycardia, but this finding is not specific since it is also frequently present in patients who do not complain of episodes of tachycardia[1-2]. Moreover, it is not completely clear how catheter ablation of the so-called slow pathway works, since ablation results in the suppression of atrioventricular nodal re-entrant tachycardia in virtually all cases[3-6]. However, the slow pathway is not eliminated in 31-69% of cases, and several studies have shown that persistence of the slow pathway does not necessarily portend a bad outcome provided that repetitive re-entrant beats are no longer inducible[3-10]. These observations suggest that factors other than atrioventricular-node duality play a role in the genesis of the tachycardia. Also the modifications of the atrioventricular-nodal function curve caused by catheter ablation of the slow pathway are heterogeneous, although they have not yet been systematically evaluated. In this prospective study, we tested the hypothesis that successful treatment is independent of specific electrophysiological modifications of atrioventricular-nodal conducting properties. We also investigated the mechanism of cure through the systematic analysis of ablation-induced modifications of the atrioventricular-nodal function curve.
Methods

Study population

The study population consisted of 104 consecutive patients with the common form of atrioventricular nodal re-entrant tachycardia who met the following inclusion criteria: pre-ablation inducibility of atrioventricular nodal re-entrant tachycardia during programmed atrial stimulation without isoproterenol administration (or other drugs); successful radiofrequency catheter ablation targeting the slow pathway. There were 35 men and 69 women with a mean age of 52 ± 16 years (range 16–81). The patients had previously been treated with 3 ± 2 antiarrhythmic drugs, without control of their arrhythmia. The mean heart rate of spontaneous atrioventricular node refractory period (calculated at the drive cycle which reproducibly induced clinical atrioventricular nodal re-entrant tachycardia) was 173 ± 30 beats min⁻¹. Associated abnormal cardiac findings were present in 19 patients; no patient had heart failure.

Electrophysiological protocol

Complete electrophysiological assessment, including atrioventricular-nodal function curve before and after ablation, was performed in all patients. The electrophysiological evaluation and catheter ablation procedures were performed during a single session. Antiarrhythmic drugs had been discontinued at least five half-lives before the study. Four quadripolar electrode catheters were introduced via femoral and jugular veins and positioned in the high right atrium, the His-bundle region, the right ventricular apex and the coronary sinus. The stimulation protocol consisted of atrial and ventricular incremental pacing and extrastimulation. The following parameters were considered, before and after ablation: AH interval, effective atrioventricular-node refractory period (calculated at the drive cycle which induced atrioventricular nodal re-entrant tachycardia, antegrade and retrograde maximum 1:1 atrioventricular conduction rate during incremental pacing and antegrade atrioventricular-nodal function assessment. In order to minimize the potential for autonomic variability for valid comparison of pre-ablation and post-ablation values, we performed the post-ablation evaluation at least 30 min after the energy application.

Antegrade atrioventricular-nodal function curve

Programmed atrial stimulation was performed at the high right atrium at increasing steps of an 8-beat drive at a cycle of 750, 600, 500 and 430 ms. The extrastimulus coupling interval was stepped down by 10 ms until atrial refractoriness occurred. Protocol was stopped at the drive cycle which reproducibly induced clinical atrioventricular nodal re-entrant tachycardia. For that drive cycle, the A1-H1 duration was plotted against the A-A1 interval (measured on the His bundle electrocardiogram), and the resulting antegrade atrioventricular-nodal function curve was drawn and considered for analysis. An increase of >50 ms in the A1-H1 interval in response to a 10 ms decrease in the A-A1 coupling interval was defined as a discontinuous curve and taken as evidence of a dual antegrade atrioventricular pathway.

For each patient, the curve obtained before ablation was compared with that obtained, using the same drive, after ablation. Our preliminary study ¹ and previous studies from other authors ²,³ had shown that the modifications of the atrioventricular-nodal function curve induced by ablation are heterogeneous and that the post-ablation function curve may shift downward, upward or remain unchanged. Accordingly, patients were assigned to one of the following patterns: the type-1 pattern was defined when ablation caused a downward shift of the left side of the curve, whether or not a discontinuous curve had been present before ablation; in the type-2 pattern the atrioventricular-nodal function curves were substantially overlapping before and after ablation whether or not a discontinuous curve had been present before ablation; the type-3 pattern was defined when ablation caused an upward shift of the left side of the atrioventricular-nodal function curve, whether or not discontinuity was present before ablation.

Radiofrequency ablation

The ablation site was chosen on the basis of electrocardiogram recordings along the tricuspid annulus near the ostium of the coronary sinus, in the zone of low-frequency, fractionated, slow-potential recording. The catheter was initially positioned in the right anterior oblique view along the tricuspid annulus anterior to the ostium of the coronary sinus. At this site, the bipolar electrocardiogram from the distal pair of electrodes recorded a larger ventricular than atrial deflection and showed an atrioventricular ratio of less than 1:2. If application was not successful, the catheter was repositioned higher along the tricuspid annulus (more cephalad) in a continued attempt to ablate the slow pathway. Radiofrequency energy was delivered at 15–30 W for 20–40 s. The ablation procedure was ended and considered successful if clinical atrioventricular nodal re-entrant tachycardia and more than a single atrioventricular-node re-entrant echo beat were rendered no longer inducible with the complete stimulation protocol (even with stimulation during isoproterenol facilitation).

Patients were generally discharged from hospital the day after the procedure and were seen for follow-up 3 months later. Late follow-up was obtained for each patient by a direct telephone interview.
Table 1  Electrophysiological data in the patients with the three types of patterns

<table>
<thead>
<tr>
<th>Parameters</th>
<th>Type 1</th>
<th>Type 2</th>
<th>Type 3</th>
</tr>
</thead>
<tbody>
<tr>
<td>Number of patients</td>
<td>54 (52)</td>
<td>33 (32)</td>
<td>17 (16)</td>
</tr>
<tr>
<td>AVNRT cycle length (ms)</td>
<td>368 ± 62</td>
<td>385 ± 75</td>
<td>354 ± 68</td>
</tr>
<tr>
<td>AH interval (ms)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Before ablation</td>
<td>77 ± 16</td>
<td>77 ± 18</td>
<td>86 ± 29</td>
</tr>
<tr>
<td>After ablation</td>
<td>76 ± 19</td>
<td>75 ± 19</td>
<td>89 ± 33</td>
</tr>
<tr>
<td>Effective AV node refractory period (ms)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Before ablation</td>
<td>260 ± 45*</td>
<td>265 ± 57</td>
<td>253 ± 53*</td>
</tr>
<tr>
<td>After ablation</td>
<td>280 ± 52*</td>
<td>257 ± 63</td>
<td>280 ± 68*</td>
</tr>
<tr>
<td>Maximum AH interval during atrial extrastimulation (ms)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Before ablation</td>
<td>318 ± 80**</td>
<td>275 ± 108</td>
<td>268 ± 120</td>
</tr>
<tr>
<td>After ablation</td>
<td>195 ± 69**</td>
<td>266 ± 86</td>
<td>268 ± 102</td>
</tr>
<tr>
<td>Maximum 1:1 antegrade AV conduction (beats . min −1)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Before ablation</td>
<td>178 ± 32</td>
<td>175 ± 38</td>
<td>178 ± 35</td>
</tr>
<tr>
<td>After ablation</td>
<td>172 ± 30</td>
<td>171 ± 27</td>
<td>165 ± 35</td>
</tr>
<tr>
<td>Maximum 1:1 retrograde AV conduction (beats . min −1)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Before ablation</td>
<td>173 ± 32</td>
<td>179 ± 32</td>
<td>173 ± 29</td>
</tr>
<tr>
<td>After ablation</td>
<td>182 ± 36</td>
<td>180 ± 25</td>
<td>174 ± 39</td>
</tr>
<tr>
<td>Presence of discontinuous curve before ablation</td>
<td>39 (72)</td>
<td>18 (55)</td>
<td>8 (47)</td>
</tr>
<tr>
<td>Persistence of AV-nodal echo beat after ablation</td>
<td>2 (4)</td>
<td>10 (30)</td>
<td>6 (35)</td>
</tr>
</tbody>
</table>

AV = atrioventricular; AVNRT = AV node re-entrant tachycardia. Numbers in brackets are percentages.

*Intrapatient comparison before and after ablation:  P=0.05

**Intrapatient comparison before and after ablation:  P=0.0001.

Statistical analysis

Statistical comparisons of electrophysiological data were performed using the analysis of variance. Comparisons of patients' characteristics or proportions were done by means of chi-square test. Comparisons of data before and after ablation were carried out by means of the two-tailed paired Student’s t test. Pearson’s correlation coefficient was used for correlation between continuous variables.

Results

Overall modifications of atrioventricular-nodal properties induced by ablation (Table 1)

The antegrade atrioventricular-nodal function curve used for analysis (and reproducible inducibility of clinical atrioventricular nodal re-entrant tachycardia) was obtained at a drive cycle of 750 ms in 21 patients, 600 ms in 50 patients, 500 ms in 27 patients and 430 ms in six patients.

The type-1 pattern was observed in 54 (52%) patients. Before ablation, atrioventricular-nodal duality was present in 39 of these patients (Fig. 1(a)) and absent in 15 (Fig. 1(b)). After ablation, atrioventricular nodal duality was always absent. Moreover, a marked decrease in the maximum AH interval during incremental atrial pacing (−223 ± 79 ms,  P<0.0001) and a slight increase in the effective atrioventricular-node refractory period (+20 ± 51 ms,  P=0.04) were observed after ablation. Thus, these changes suggest that the ablation of slow-conducting atrioventricular-nodal pathways was successful.

The type-2 pattern was observed in 33 (32%) patients, of whom atrioventricular-nodal duality was present both before and after ablation in 18 (Fig. 1(c)) and was absent both before and after ablation in 15 (Fig. 1(d)). In these 33 patients, the effective atrioventricular-nodal refractory period remained unchanged after ablation (−8 ± 53 ms, not significant). Thus, evidence of successful slow-pathway ablation was lacking in this group.

The type-3 pattern was observed in 17 (16%) patients. The upward shift of the atrioventricular-nodal function curve observed after ablation suggests a worsening of conduction velocity through the slow pathway. Atrioventricular-nodal duality was present in eight patients before ablation (Fig. 1(e)) and in seven after ablation. Of these latter, a discontinuous curve was already present before ablation in two, whereas a new discontinuity, not been present before appeared after ablation in the other five patients (Fig. 1(f)). The effective atrioventricular-node refractory period increased by +27 ± 51 ms (  P=0.05) after ablation; this increase is similar to that observed in type-1 pattern patients, but significantly different from that of type-2 pattern patients (  P=0.03). The AH interval was longer in this group than in the others, both before (  P=0.08, not significant) and after ablation (  P=0.03). The mean value of the maximum AH interval during atrial
Figure 1  Different patterns of antegrade atrioventricular-nodal function curves in individual patients. In each panel the A1-H1 duration was plotted against the A-A1 interval. For explanation see text. + = before radiofrequency; O = after radiofrequency. (a) and (b) = type 1; (c) and (d) = type 2; (e) and (f) = type 3.
Table 2  The influence of age on electrophysiological parameters

<table>
<thead>
<tr>
<th>Parameters</th>
<th>Age ≤45 years</th>
<th>Age &gt;45 years</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Number of patients</td>
<td>29 (28)</td>
<td>75 (72)</td>
<td>—</td>
</tr>
<tr>
<td>Age at time of ablations (years)</td>
<td>31 ± 9</td>
<td>61 ± 9</td>
<td>—</td>
</tr>
<tr>
<td>Age at the onset of arrhythmia (years)</td>
<td>17 ± 10</td>
<td>42 ± 16</td>
<td>—</td>
</tr>
<tr>
<td>Duration of clinical AVNRT (years)</td>
<td>14 ± 11</td>
<td>18 ± 13</td>
<td>ns</td>
</tr>
<tr>
<td>Presence of discontinuous curve</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Before ablation</td>
<td>20 (69)</td>
<td>45 (60)</td>
<td>ns</td>
</tr>
<tr>
<td>After ablation</td>
<td>8 (28)</td>
<td>17 (23)</td>
<td>ns</td>
</tr>
<tr>
<td>AH interval (ms)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Before ablation</td>
<td>70 ± 14</td>
<td>82 ± 20</td>
<td>P=0.005</td>
</tr>
<tr>
<td>After ablation</td>
<td>68 ± 14</td>
<td>83 ± 23</td>
<td>P=0.001</td>
</tr>
<tr>
<td>Effective AV node refractory period (ms)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Before ablation</td>
<td>242 ± 40*</td>
<td>268 ± 52</td>
<td>P=0.02</td>
</tr>
<tr>
<td>After ablation</td>
<td>277 ± 55*</td>
<td>272 ± 62</td>
<td>ns</td>
</tr>
<tr>
<td>Maximum 1:1 antegrade AV conduction (beats.min⁻¹)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Before ablation</td>
<td>195 ± 36*</td>
<td>170 ± 29</td>
<td>P=0.001</td>
</tr>
<tr>
<td>After ablation</td>
<td>180 ± 35*</td>
<td>167 ± 26</td>
<td>P=0.001</td>
</tr>
<tr>
<td>Maximum 1:1 retrograde AV conduction (beats.min⁻¹)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Before ablation</td>
<td>177 ± 34</td>
<td>174 ± 29</td>
<td>ns</td>
</tr>
<tr>
<td>After ablation</td>
<td>190 ± 35</td>
<td>178 ± 30</td>
<td>P=0.001</td>
</tr>
<tr>
<td>AVNRT cycle length (ms)</td>
<td>346 ± 49</td>
<td>379 ± 71</td>
<td>P=0.04</td>
</tr>
<tr>
<td>Modifications of AV node function curve induced by ablation</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Type 1 pattern</td>
<td>20 (69)</td>
<td>34 (45)</td>
<td>P=0.02</td>
</tr>
<tr>
<td>Type 2 pattern</td>
<td>5 (17)</td>
<td>28 (37)</td>
<td>P=0.04</td>
</tr>
<tr>
<td>Type 3 pattern</td>
<td>4 (14)</td>
<td>13 (17)</td>
<td>ns</td>
</tr>
</tbody>
</table>

AV=atrioventricular; AVNRT=AV node re-entrant tachycardia. Numbers in brackets are percentages.

*Intrapatient comparison before and after ablation: P<0.05.

extrastimulation did not change after ablation. However, it should be noted that three patients in this group showed a marked prolongation of the maximum AH interval >100 ms, suggesting a further slowing of atrioventricular-nodal conduction.

At baseline evaluation, the electrophysiological parameters did not differ among the patients with the three patterns, except for a higher percentage of discontinuous curves among type 1 pattern patients (P=0.05). A higher number of radiofrequency pulses were delivered in type 1 pattern patients than in type 2 pattern patients (8 ± 7 vs 5 ± 4, P=0.02). Type 3 pattern patients received 7 ± 9 pulses (not significantly different from the other two groups). Post-ablation atrioventricular node echo beats were less frequent in type 1 pattern patients than in the others (P=0.001). The mean age was, respectively, 49 ± 17, 56 ± 14 and 57 ± 17 for the patients with the three patterns (P=0.05). Sex, duration of clinical arrhythmia and associated cardiac abnormalities were similar.

**Age-related differences**

A weak, even though significant, correlation was observed between age and the following parameters measured during the baseline electrophysiological evaluation: AH interval (r=0.22, P=0.01), effective atrioventricular-node refractory period (r=0.20, P=0.04), maximum 1:1 antegrade atrioventricular conduction (r=0.25, P=0.01).

Patients were subdivided into two groups, according to their age, (≤45 or >45 years), at the time of ablation, (mean age: 31 ± 9 and 61 ± 9 respectively) (Table 2). In both groups, the duration of arrhythmia (14 ± 11 and 18 ± 13 years, respectively) was similar; the mean age at the time of onset of the arrhythmia was therefore 17 ± 10 and 42 ± 16 years respectively. Type-1 pattern was more frequent in the younger patients, while type-2 pattern was more frequent in the older patients. After ablation, the effective atrioventricular-node refractory period increased to a greater extent in the younger than in the older patients (+3 ± 9 ms vs +4 ± 50 ms, P=0.006) and maximum 1:1 antegrade atrioventricular conduction decreased by a greater magnitude in the younger than in the older patients (−15 ± 35 beats per min vs −3 ± 27 beats per min, P=0.06).

**Follow-up**

During follow-up (minimum 6 months), clinical atrioventricular nodal re-entrant tachycardia spontaneously recurred (always within 3 months) in one patient with
the type-1 pattern, in one patient with the type-2 pattern and in one patient with the type-3 pattern (not statistically different). The mean duration of follow-up was similar in the three groups (18 ± 6, 20 ± 7 and 18 ± 6 months respectively).

Discussion

Previous studies on the effects of catheter ablation of the slow pathway in atrioventricular nodal re-entrant tachycardia have focused on the modifications of refractoriness of the atrioventricular node and have given partly discordant results. To our knowledge, this is the first study to focus on a systematic evaluation of atrioventricular-nodal properties by means of comparing pre- and post-ablation atrioventricular-nodal function curves.

Modifications of atrioventricular-nodal properties induced by ablation

This study shows that successful ablation of common atrioventricular nodal re-entrant tachycardia results in a wide spectrum of changes in atrioventricular-nodal function curves which are partly age-dependent. The results of the electrophysiological evaluation suggested that elimination of the antegrade slow pathway of the re-entry circuit occurred in only half of the patients (type-1 pattern group). The downward shift of the left side of the function curve (and the disappearance of atrioventricular-nodal duality when previously present) suggested that antegrade atrioventricular conduction could occur only through fast-conducting fibres. Downward shift of the function curve has been demonstrated to be due to the shortening of refractoriness of the fast pathway after slow-pathway ablation, and its effect is independent of autonomic influences; it is transient. Moreover, it has been shown that a possible autonomic variability induced by ablation is minimized if the electrophysiological evaluation is performed >10 min after ablation. The hypothetical model is shown in Fig. 2 and Fig. 3(a).

In a third of our patients, no changes in the atrioventricular-nodal function curve were observed after successful ablation, and dual atrioventricular-nodal physiology persisted in those patients in whom it had been present before ablation. This pattern was observed more frequently in older patients. Admittedly, the ability to distinguish slow-pathway conduction from fast-pathway conduction might have been enhanced by the extensive use of pharmacological manipulations or by the introduction of a double extrastimulus. Nevertheless, it must be noted that atrioventricular nodal re-entrant tachycardia was initiated by the same drive that was unable to show modifications of atrioventricular-nodal properties induced by ablation, even though this intervention rendered atrioventricular nodal re-entrant tachycardia no longer inducible. Therefore, demonstration of atrioventricular-nodal duality and its suppression were not essential to achieving successful ablation, as has been reported in the literature. Recently, Anselme et al. performed a simultaneous multisite catheter mapping of Koch’s triangle during both atrioventricular nodal re-entrant tachycardia and ventricular pacing, at a similar cycle length to the tachycardia. They showed that the retrograde fast pathway is heterogeneous within the area of Koch’s triangle in patients with atrioventricular nodal re-entrant tachycardia both during tachycardia and during ventricular pacing. Multiple sites of early breakthrough in the triangle of Koch as well as a broad wave front of activation (which were inconsistent with a sequential activation from the apex of the triangle) were present in

Figure 2 Hypothetical models of the re-entrant circuit. The tinted area indicates the limits of the compact atrioventricular node. (a) Sinus rhythm. Impulses enter Koch’s triangle from the anterior limbus of the fossa ovalis (FO). Impulses penetrate all atrioventricular nodal connections but the preferred route of conduction is via the anterior connections because this route of conduction traverses less atrioventricular nodal tissue. (b) When atrioventricular nodal re-entrant tachycardia is induced, the circuit is likely to be functional, not anatomical, and any two of the pathways may complete a re-entrant circuit, providing they possess the appropriate properties of refractoriness and conduction velocity. Modified from McGuire et al.
two-thirds of the patients; moreover, in a few patients
the earliest site of retrograde activation was in the
coronal sinus or at the base of the triangle. Furthermore,
since a discordance in the retrograde atrial activ-
ation sequence was present between ventricular pacing
and atrioventricular nodal re-entrant tachycardia in
about half of the patients, the authors supposed that the
retrograde fast pathway was not an anatomically dis-
crete structure, but dynamic for each individual. There-
fore, in several patients, the anatomical retrograde limb
of the tachycardia circuit may be at the base of the
triangle of Koch, close to the site of the antegrade slow
pathway. In our patients with the type 2 pattern, it is
speculated that the application of radiofrequency energy
at the base of the Koch's triangle might have resulted in
the modification of the retrograde hidden limb of the
circuit or the destruction of a critical mass of peri-
nodal and nodal connecting fibres without affecting the
antegrade slow pathway.

Residual connections might have an inadequate
balance between refractoriness and conduction velocity
or simply be anatomically too close together to allow
recovery of excitability of either pathway; thus, re-entry
is no longer sustainable, even if the persistence, in some
patients, of single atrioventricular-node echo beats after
ablation showed that a re-entry circuit is still present
(Fig. 3(b)). Since in type 2 pattern patients successful
ablation could be achieved with a slightly lower number
of pulses of radiofrequency energy, we can speculate
that the circuit was smaller or that minor functional
damage was effective for suppression. Type 2 and type 3
patterns may actually represent an incomplete ablation
of the slow pathway. However, there is growing apprecia-
tion that so-called slow pathway ablation is neither
routinely achieved nor necessary for a successful out-
come. Indeed, follow-up data from the present study
and from others in the literature show that the
recurrence rate of atrioventricular nodal re-entrant
tachycardia is low and similar both in patients who had
ablation of the slow pathway and in those who did not;
recurrences of atrioventricular nodal re-entrant
tachycardia usually occur within a few months.

In a minority of cases, an 'unusual' upward shift
of the atrioventricular-nodal function curve suggested a
further slowing of conduction velocity through the
slow-pathway network. From a theoretical point of
view, a slowing of conduction velocity should enhance

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**Figure 3** Possible mechanisms of cure based on the
hypothetical model of the re-entrant circuit shown in Fig.
2. If such a model is correct, interruption of the circuit, in
any area, must result in successful cure of atrioventricular
nodal re-entrant tachycardia. (a) Type 1 pattern. A radio-
frequency lesion (hatched area) has destroyed the pos-
terior connections (slow pathway). The anterior atrionodal
connections (antegrade fast pathway) remain. (b) Type 2
pattern. Both antegrade fast and slow pathways remain
intact after the ablative procedure, but the radiofrequency
lesion has destroyed the retrograde fast limb or a critical
mass of perinodal and nodal connecting fibres in the
re-entrant circuit. Residual connections have an inad-
quate balance of refactoriness and conduction velocity
or are simply anatomically too close together to allow
recovery of excitability of either pathway and re-entry is
no longer sustainable. (c) Type 3 pattern. A radio-
frequency lesion has only partly destroyed the slow path-
way, so that conduction still occurs at a slower velocity
(ultraslow connections). Moreover, the lesion has de-
stroyed the retrograde fast limb or a critical mass of
connecting fibres. Residual connections have an in-
adquate balance of refactorines and conduction velocity
or are simply anatomically too close together to allow
recovery of excitability of either pathway and re-entry is
no longer sustainable.
the inducibility of atrioventricular nodal re-entrant tachycardia by disrupting the balance in velocity between the antegrade slow pathway and the retrograde fast pathway. Whether this very slow conduction was the result of damage to the slow pathway, which further slowed conduction velocity down through the same pathway, or to the presence of another, more slowly-conducting pathway which had previously been hidden, is not clear (Fig. 3c). An upward shift of the atrioventricular-node function curve is usually observed with ablation of the so-called fast pathway from an anterior approach. This was unlikely to have occurred in our patients. Indeed, with the anterior approach, successful ablation is associated with the appearance of retrograde atrioventricular block, marked lengthening of the AH interval and disappearance of atrioventricular nodal duality. This was not the case in our patients, in whom conduction through the antegrade fast pathway remained unchanged after ablation and a discontinuous curve was present after ablation in several cases.

Role of age

We observed a weak, even though significant, correlation between age and some electrophysiological parameters. In old age, baseline electrophysiological characteristics revealed poorer properties of atrioventricular-nodal function, with slower conduction velocity and longer time to recover excitability; consequently, on tachycardia onset, its cycle length was longer. Similar differences in baseline electrophysiological characteristics have been observed by Wu et al. This implies that atrioventricular nodal re-entrant tachycardia can occur late in the life of a patient, when these parameters become critical. The modification of electrophysiological parameters induced by ablation were also more evident in younger than in older patients. For example, the type 2 pattern was more frequent in older patients and effective refractory period increased after ablation in younger but not in older patients. Our study group had a higher mean age, at time of ablation, than that usually reported in the literature, which varies from 37 to 48 years. This higher age explains the slightly lower percentage of slow pathway elimination observed by us in respect to that usually reported in the literature.

Therefore, it is likely that the electrophysiological (and possibly anatomical) substrate of the re-entry circuit differs between younger and older patients. The exact reason for these differences is unknown. We can hypothetically that in older patients a lower degree of atrioventricular nodal duality is required for the activation of the circuit of the tachycardia (which is more unstable) and that a minor functional damage is effective for suppression of the arrhythmia.

Conclusion

The main result of this study is that successful elimination of atrioventricular nodal re-entrant tachycardia is independent of the modification of specific electrophysiological parameters and probably depends on critical nodal and perinodal tissue damage at different sites on the re-entrant circuit. The effects of ablation are influenced by the age of the patients.

References

Catheter ablation of re-entrant tachycardia
