Temperature-controlled slow pathway ablation for treatment of atrioventricular nodal reentrant tachycardia using a combined anatomical and electrogram guided strategy


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Aims Anatomical and electrogram-guided techniques have been used separately for slow pathway ablation in atrioventricular nodal reentrant tachycardia. The aims of the present study were to analyse electrogram characteristics of target sites and biophysical parameters using a combined anatomical and electrogram-guided technique for temperature-controlled radiofrequency catheter ablation of the slow pathway.

Methods and results Using a temperature-controlled (pre-selected 60 ºC) catheter system, 53 patients with atrioventricular nodal reentrant tachycardia underwent slow pathway radiofrequency ablation. Mapping was started posteroseptally near the coronary sinus ostium and continued towards the midseptal area if needed. The longest and latest atrial electrograms with an atrioventricular ratio of \(<0.5\) were targeted. After a median of two pulses (mean 2.36 ± 1.33), atrioventricular nodal reentrant tachycardia was rendered non-inducible in all patients without complications. Successful sites had longer atrial electrograms (78.8 ± 9.8 vs 67.6 ± 13.3 ms, \(P<0.002\)) and larger ventricular electrogram amplitudes (92.4 ± 51.2 vs 63.1 ± 28.8 mV, \(P<0.05\)) than the failed sites, but had a similar atrioventricular ratio, P-A interval and atrial electrogram amplitude. Overall, an atrial electrogram duration of ≥ 70 ms was associated with effective radiofrequency delivery, with 86% sensitivity and 62% specificity. The achieved temperature maximum was 62.3 ± 9.8 ºC at successful and 58.8 ± 9.0 ºC at unsuccessful sites (ns). There was no significant difference between successful and unsuccessful applications with respect to power output, impedance and total delivery energy. During a pre-discharge study, three patients with inducible atrioventricular nodal reentrant tachycardia underwent a repeat ablation. During 12.3 ± 2.5 (6-15) months of follow-up, three others had a clinical recurrence of atrioventricular nodal reentrant tachycardia.

Conclusions The combined approach for slow pathway ablation is highly effective, requiring a low number of radiofrequency pulses. Long atrial activation time seems to be the most powerful predictor of success. Similar catheter tip temperature levels during successful and unsuccessful radiofrequency applications indicate that suboptimal selection of target sites rather than ineffective heating due to poor catheter tissue coupling is responsible for unsuccessful energy delivery.

Key Words: Catheter ablation, atrioventricular nodal reentrant tachycardia, slow pathway, supraventricular tachycardia, temperature control.

Introduction

Radiofrequency catheter modification of the atrioventricular node targeting the fast or slow pathway has been introduced as a curative approach for atrioventricular nodal reentrant tachycardia. Modification of the posteroseptally located fast pathway has been demonstrated to be effective, but carries the risk of complete atrioventricular block. However, sequential mapping strategies and titration of energy delivery may decrease the incidence of this complication. Slow pathway modification seems to have a lower risk of inadvertent atrioventricular block, since radiofrequency current is applied to posteroseptal or midseptal areas further
away from the central body of the atrophicventricular node[3,5,9-12,15]. To date, strategies for slow pathway ablation have been anatomical or electrogram-guided[3,5,9-12,15]. The electrogram-guided method is primarily oriented on detection of specific slow pathway potentials[9,10]. Nevertheless, several studies have shown that the recording of distinct slow pathway potentials is not a prerequisite for successful ablation sites[5,11,12,15]. In contrast to previous reports, in this study, mapping was oriented on both anatomical landmarks and electrogram criteria, but not focused on the detection of specific slow pathway potentials.

So far, modification of the atrophicventricular node has been performed using power-regulated radiofrequency current[1-15]. Experimental findings have demonstrated that only monitoring of the catheter-tissue interface temperature can accurately predict lesion size[16]. Furthermore, formation of potentially thrombogenic coagula is critically dependent on abrupt changes in impedance and the catheter tip temperature[17,18]. Overheating of the catheter tip and coagulum formation can be prevented by closed loop temperature control and thus might improve the procedure's safety[19].

Reviewing our early experience with radiofrequency catheter ablation of the slow pathway and previously described reports regarding the electrogram characteristics of successful and failed sites during sinus rhythm[9,10,15], we hypothesized that electrograms at the successful target sites, at the posteroseptal to midseptal regions, should have the following characteristics: (1) later timing of the atrial electrogram when compared to the onset of the atrial electrogram recorded at the His bundle region or the high right atrium, (2) a prolonged and fractionated atrial electrogram with or without a possible slow pathway potential, since this area presumably behaves like a zone of slow conduction during sinus rhythm and atrophicventricular nodal reentrant tachycardia, and (3) a critical ratio of the atrial to ventricular electrogram amplitude at the target site (atrioventricular ratio of ≤ 0.5).

The present study was conducted to test this hypothesis and to assess the feasibility of a new combined stepwise anatomical and electrogram-guided approach for temperature-controlled catheter ablation of the slow pathway in patients with atrophicventricular nodal reentrant tachycardia.

Methods

Patient data

Fifty-three consecutive patients with a history of recurrent paroxysmal sustained atrophicventricular nodal reentrant tachycardia were prospectively enrolled in the present study. There were 39 females and 14 males, aged 45 ± 2 years (range 15-74). A history of syncope was present in 11 cases. Previous antiarrhythmic therapy (2.5 ± 1.3 agents) was discontinued either due to inefficacy or intolerable side-effects. No patients had other cardiac abnormalities.

Electrophysiological study

Electrophysiological studies were performed in a fasting, lightly sedated state after written informed consent had been obtained. Antiarrhythmic therapy had been discontinued for at least five half-lives. The investigations were performed using a conventional method of intracardiac recording and stimulation[20]. Multipolar electrode catheters with an inter-electrode spacing of 5 or 10 mm were introduced from the femoral veins and positioned into the right atrium, His bundle region and right ventricular apex under fluoroscopic guidance. Continuous anticoagulation was maintained throughout the procedure with an initial bolus injection of 5000 units of heparin, followed by 1000 units i.v. hourly.

When sustained atrophicventricular nodal reentrant tachycardia could not be reproducibly induced, isoproterenol was administered, and titrated to achieve a sinus rate increment of 25%. The diagnosis of atrophicventricular nodal reentrant tachycardia was made according to previously published criteria[21,22].

Endocardial mapping

Either a conventional 7F or a 5F deflectable electrode catheter (Osypka 'Cereblate' Dr P. Osypka, Grenzach-Wyhlen, Germany) with a thermistor embedded into the 4 mm catheter tip was introduced via a femoral vein and used for endocardial mapping and ablation. Bipolar electrograms of the distal pair of the mapping catheter were recorded simultaneously at a paper speed of 100 and 200 mm.s⁻¹. The band pass filter setting was 40 to 500 Hz. Since an additional coronary sinus catheter was not routinely inserted, the coronary sinus ostium was transiently intubated with the ablation catheter for direct localization. Endocardial mapping was guided by both anatomical landmarks and electrophysiological parameters. Initially, the electrode catheter was positioned posteroseptally using biplane fluoroscopic control with LAO 30° and RAO 60° standard views.

At each site, the local bipolar electrograms of five sinus beats prior to energy delivery were analysed. Timing of the atrial electrograms were determined with regard to the onset of surface P, atrial electrogram recorded on the right atrium and/or His catheter, the amplitude and duration of atrial and ventricular electrograms and the atrophicventricular ratio. Because there is no universal definition of slow pathway potentials, we considered both previously described morphologies[3,9] as possible slow pathway potentials. Therefore, either a rapid high frequency signal[9] or a low frequency broad deflection located after or within the terminal portion of the atrial electrogram[10] was labelled as possible slow pathway potential. Distinct slow pathway potentials were not specifically targeted, although all electrograms.
were evaluated for the presence of a possible slow pathway potential. Electrogram stability was assessed during sinus rhythm and constant atrial pacing. The electrograms were categorized as stable if there was less than 10% beat-to-beat variation of the atrial and ventricular potential and no new or loss of major deflection was observed.

**Criteria for selection of electrograms at target site**
Electrograms at target sites recorded at the posteroseptal to midseptal regions were accepted for energy delivery if the following characteristics were present: (1) later timing of the atrial electrogram when compared to the onset of the atrial electrogram recorded on the His or high right atrial catheter, (2) fractionated and longer duration of atrial electrograms than those of the His or high right atrial catheter with or without possible slow pathway potential, and (3) an atrial electrogram amplitude of half or less of the ventricular electrogram when recorded on the same mapping catheter (atrioventricular ratio ≤0.5).

**Measurements of timing of local electrograms**
Measurements were based on their onset with the exception of a few cases where the onset of a local electrogram could not be reliably determined, in which case the first rapid deflection crossing the baseline was used. Likewise, the duration of the local atrial electrogram was measured by determining the interval from its onset to the beginning of an isoelectric line located preceding the ventricular electrogram, or to the beginning of the earliest ventricular activity if such an isoelectric line was not present. Any possible slow pathway potential was included for the duration of the atrial electrogram.

Electrogram characteristics of de novo (first) successful target sites were also compared to those of de novo (first of four or more) unsuccessful sites, because even unsuccessful energy delivery may add to tissue damage and thus contribute to the final impulse success. Furthermore, unsuccessful impulses may change local electrograms. To minimize these influences, we have chosen the first failed sites of those patients who received multiple energy applications. In this subanalysis, the cut off of four pulses was selected to minimize the aforementioned influences. Radiofrequency application was initiated during sinus rhythm. However, when ectopic junctional rhythms were observed, in order to unmask radiofrequency-induced atrioventricular block, atrial pacing at cycle lengths of 500 to 400 ms was performed throughout the energy delivery. Impulse delivery was
Combined approach for slow pathway ablation

Figure 1  Radiograms show left anterior oblique (60°) and right anterior oblique (30°) views with the ablation catheter (MAP) and additional intracardiac catheters located in the high right atrium (HRA), His bundle region (HIS) and right ventricular apex (RVA). A representative mapping sequence demonstrates the ablation catheter initially intubating the coronary sinus (panel (a)). The estimated area of the coronary sinus os is indicated by an arrow. The mapping electrode is then positioned posteroseptally (panel (b)) and at lower (panel (c)) and upper midseptal (panel (d)) areas.

started posteroseptally close to the ostium of the coronary sinus where the most suitable local electrogram recording was obtained. The mapping procedure was continued and the catheter was slightly moved towards more midseptally located sites when the first pulse was not successful. The electrode catheter was positioned more superiorly and anteriorly along the inter-atrial septum in a stepwise manner if needed. Figure 1 gives an example of a typical mapping sequence during the ablation procedure, where the left and right anterior oblique views with the mapping catheter positioned in the coronary sinus posteroseptally and at lower and upper mid septal areas, are depicted. It should be emphasized that due to induction of junctional and atrial ectopic beats by energy delivery, occasionally slight dislocation of the catheter tip was observed. Therefore, the presence or absence of slow pathway potentials immediately following application of energy at these sites was not systematically assessed.

Radiofrequency catheter ablation

Using the radiofrequency generator HAT 200S (Dr Osypka, Grenzach-Whylen, Germany), energy (500 kHz unmodulated current) was delivered during sinus rhythm or atrial pacing between the tip electrode of the ablation catheter and the indifferent patch electrode positioned at the mid-posterior chest wall. Actual power output, impedance, energy delivery and catheter tip temperature were continuously measured and displayed via an interface with a microcomputer. Catheter-tip-tissue interface temperature was pre-selected to 60 °C.
Power output was adjusted automatically by the generator (0-50 watt) in order to achieve and maintain the pre-selected tip temperature. Energy delivery (pre-set time 60-90 s) was discontinued automatically when sudden rises in impedance occurred. A sudden impedance rise of more than 50 Ohm was considered a significant impedance 'jump'. Subsequently, the catheter was withdrawn to exclude coagulum formation at the electrode tip. Additionally, energy application was stopped immediately on catheter dislocation, atrioventricular nodal reentrant tachycardia, Wenckebach periodicity or atrioventricular block. Inducibility of atrioventricular nodal reentrant tachycardia and anterograde and retrograde atrioventricular conduction capabilities were assessed following the delivery of each impulse. Non-inducibility of atrioventricular nodal reentrant tachycardia at baseline and during isoproterenol infusion was defined as the endpoint of the ablation session. Persisting longitudinal dissociation of the atrioventricular node or the inducibility of only one atrioventricular nodal reentrant beat was an acceptable endpoint. Anticoagulation with i.v. heparin was continued for 24 h. All patients underwent echocardiographic investigation within 48 h to exclude thrombus formation or pericardial effusion.

Follow-up

All patients underwent a pre-discharge electrophysiological study 2-4 days after the ablation procedure and were regularly followed in the outpatient clinic. Aspirin (100 mg daily) was prescribed for 3 months.

Data analysis

Continuous data are expressed as mean ± SD or as median. Variables were compared by Student’s t-test or Mann-Whitney U test as appropriate. P values <0.05 were considered significant. Statistical analysis was performed using a commercially available computer software (SPSS; SPSS Inc., U.S.A.). Multivariate analysis was performed to compare electrogram characteristics of effective and ineffective catheter ablation sites. A two-tailed Fisher’s exact test was used for two by two contingency table measurements (GraphPAD InStat Inc. 1990, U.S.A.) when required.

Results

Immediate outcome

Atrioventricular nodal reentrant tachycardia was reproducibly inducible with or without additional application of isoproterenol in all patients prior to the initial ablation attempt. The cycle length of the tachycardia was 326 ± 61 ms. The common type of atrioventricular nodal reentrant tachycardia was rendered non-inducible in all but one patient with the uncommon form. Both types were present in another patient.

A median of two radiofrequency pulses (mean 2.36 ± 1.3) was required to eliminate tachycardia in all 53 patients. In 23 patients, only a single energy delivery was necessary for slow pathway modification. One to five pulses, delivered to the right posteroseptal to midseptal area, were required in all but one patient who received 14 pulses. In this patient, 12 ineffective pulses were delivered to the posteroseptal and midseptal region. Thereafter, two pulses were applied to the anterior aspect of the inter-atrial septum. Subsequently, the tachycardia was no longer inducible. The electrophysiological findings (unaltered PR interval, persistent dual atrioventricular nodal physiology) were consistent with selective modulation of the slow pathway. The mean fluoroscopy time was 27.0 ± 19 min. No complete atrioventricular block or other procedure-related complications occurred.

Electrophysiological findings

Electrophysiological parameters at the baseline study and after the ablation session are summarized in Table 1. The cycle length at which anterograde atrioventricular block occurred during incremental atrial pacing increased significantly from 308 ± 60 to 373 ± 94 ms following the ablation procedure (P<0.01). Retrograde ventriculo-atrial Wenckebach point was not influenced by radiofrequency delivery (284 ± 56 vs 298 ± 63 ms; ns). The duration of PR, AH, and HV interval did not change significantly after slow pathway ablation. In 36 patients (64%), longitudinal dissociation of the atrioventricular node, as evidenced by sudden AH prolongation during premature atrial stimulation, was found before ablation. This finding persisted in 24 patients (43%) after the procedure. Atrioventricular nodal reentrant tachycardia was rendered non-inducible in all patients. A single retrograde atrioventricular nodal echo beat via the presumed fast pathway with subsequent block in the

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<th>Before ablation</th>
<th>2-4 days after ablation</th>
<th>P value</th>
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<tr>
<td>PR interval</td>
<td>152 ± 20</td>
<td>152 ± 18</td>
<td>ns</td>
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<tr>
<td>AH interval</td>
<td>79 ± 21</td>
<td>78 ± 21</td>
<td>ns</td>
</tr>
<tr>
<td>HV interval</td>
<td>40 ± 7</td>
<td>41 ± 8</td>
<td>ns</td>
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<tr>
<td>IAP-CL</td>
<td>308 ± 60</td>
<td>373 ± 94</td>
<td>P&lt;0.01</td>
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<td>IVP-CL</td>
<td>284 ± 56</td>
<td>298 ± 63</td>
<td>ns</td>
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IAP=Wenckebach point during incremental atrial pacing. IVP=Wenckebach point during incremental ventricular pacing.

Values are expressed as mean values ± standard deviation. All intervals in msec.
slow pathway was inducible with or without isoproterenol infusion in 22 cases (39%) following the ablation session.

**Local electrogram characteristics**

*Successful versus unsuccessful sites*

Table 2 depicts local electrogram characteristics of all successful and all unsuccessful ablation sites. The duration of the local atrial electrogram at effective sites (78.8 ± 9.8 ms) was significantly longer than at ineffective sites (67.6 ± 13.3 ms, P < 0.003). There was a higher amplitude of the ventricular electrogram at successful sites (92.4 ± 51.2 mV vs 63.1 ± 28.8 mV, P < 0.05). Possible slow pathway potentials were more frequently observed at effective (30%) than at ineffective (12%) target sites (P < 0.02). At effective sites electrograms were more frequently classified as stable than at ineffective sites (79% vs 49%). There was no significant difference between successful and unsuccessful ablation sites with respect to the amplitude of the atrial electrogram, the interval between surface P-wave and the onset of local atrial activation, and the atrioventricular ratio (Table 2). Multivariate analysis demonstrated that the atrial electrogram duration was the only variable with a significant difference between effective and ineffective target sites. A local atrial electrogram duration of 70 ms or more was predictive of successful ablation site, with 86% sensitivity and 62% specificity.

*Successful first versus failed first site*

The electrogram characteristics of 23 patients with only a single successful radiofrequency application (group 1) were compared to the first unsuccessful target site in 10 patients who required four or more pulses (group 2) for slow pathway ablation (Table 3). Local atrial electrogram duration at target sites of group 1 was significantly longer when compared to group 2 patients (76.3 ± 9.2 ms vs 60.0 ± 11.6 ms, P < 0.006). The local atrial electrogram measured 70 ms or more in 90% of group 1 patients, but in only 25% of group 2. Figure 2 demonstrates a local electrogram recorded at an ablation site with a single successful delivery of energy (group 1). The recording shows a sharp positive deflection within the terminal portion of the atrial segment. This distinct potential was classified as a possible slow pathway potential. After delivery of the successful first impulse this possible slow pathway potential disappeared. Possible slow pathway potentials were present in 22% of group 1 ablation sites and 10% of group 2 (ns). Figure 3 gives an example of the first unsuccessful site of a group 2 patient. Energy delivery was ineffective despite relatively long atrial activation with a low amplitude 'hump' within the terminal portion of the atrial electrogram as a possible slow pathway potential. There were no significant differences in the amplitude of the atrial and ventricular electrogram, the interval between the surface P-wave and onset of local atrial activation or the atrioventricular ratio between the two groups as depicted in Table 3.
Figure 2 Surface ECG leads I, II, V₁ and simultaneous intracardiac recordings of the distal and proximal pair of electrodes of the mapping catheter (MAP), His-bundle electrogram (HIS) and high right atrium (HRA) are shown. Depicted is the local electrogram recording during sinus rhythm with a possible slow pathway potential (SP) at the target site of a single successful pulse prior to energy delivery (left panel). Note the distinct sharp potential within the terminal portion of the atrial electrogram which occurred slightly before the His bundle electrogram. Following energy delivery (right panel), the presumed slow pathway potential disappeared and atrioventricular nodal reentrant tachycardia was rendered non-inducible. The characteristic of the ventricular portion of the local electrogram remained unchanged, suggesting a stable catheter position.

Biophysical findings

Biophysical parameters during energy current applications at effective and ineffective sites were compared (Table 4). There was no significant difference between effective and ineffective sites regarding the achieved catheter tip temperature or power output after 5 or 10 s. Also, the maximal values were not significantly different, although the achieved temperature maximum of 62.3 ± 10.1 °C during effective pulses tended to be higher as compared to 58.8 ± 9.0 during ineffective applications. Furthermore, there were no differences with respect to impedance and total delivered energy. Overall, sudden rises in impedance were observed during six (4.8%) radiofrequency applications with coagulum formation adherent to the catheter tip in three cases (2.4%).

Follow-up

Atrioventricular nodal reentrant tachycardia was inducible without clinical recurrence in three patients during the pre-discharge study. These patients underwent successful repeat ablation during the same session. During a follow-up period of 12.3 ± 2.5 (range of 6–15 months, recurrence of the sustained tachycardia was observed in three patients who underwent a second successful procedure. The local electrogram characteristics and the number of radiofrequency pulses were not significantly different in patients with tachycardia recurrence when compared to those without recurrence. Persistent atrioventricular longitudinal dissociation and a single atrioventricular echo beat were present following the first ablation session in three of these six patients. No procedure-related complications were observed during follow-up.

Discussion

Main findings

The results of the present study demonstrate that atrioventricular nodal reentrant tachycardia can be successfully abolished with high efficacy and a low number of radiofrequency applications when endocardial mapping is performed using a sequential, combined anatomical and electrogram-oriented strategy. Concordant to other reports, the site of successful radiofrequency delivery was located within the posterior or midseptal area in most patients. It was not necessary to deliver radiofrequency current to target sites within the
coronary sinus, as previously described\cite{9,15}. In one patient 14 impulses were applied to the anterior aspect of the inter-atrial septum to achieve elimination of slow pathway conduction. This location outside the common boundary demonstrates the potential anatomical variability of slow pathway conduction\cite{29}.

Previous strategies for slow pathway ablation were mainly based on either local electrogram criteria\cite{9,10} or anatomical landmarks\cite{3,11,23}. When the anatomical-guided strategy was applied, radiofrequency current was usually delivered unguided to the posterior aspect of the inter-atrial septum in a stepwise manner. Several reports have shown that this technique is effective in eradicating atrioventricular nodal reentrant tachycardia\cite{3,11,23}. The electrogram-guided technique uses endocardial mapping for detection of distinct slow pathway potentials. Thus, this method is dependent on a high incidence of slow pathway potentials at successful target sites, which has not been observed uniformly in other studies\cite{3,11}.

Using both anatomical- and electrogram-guided strategies, tachycardia can be abolished with a high success rate. However, unintended ablation of the fast pathway and induction of complete heart block has been observed with both techniques\cite{3,9,11}. In some instances, multiple radiofrequency deliveries were necessary. When the anatomical approach was applied, the mean number of required radiofrequency pulses ranked between 6 and 20, with a maximum of up to 56 delivered pulses\cite{3,5,11,23}. Occasionally, slow pathway conduction could not be abolished and the fast pathway had to be targeted subsequently\cite{3,4}. A relatively broad region or network of fibres capable of slow conduction and participation in reentrant tachycardia may contribute to these findings.

Anatomical studies have suggested that fibres covering the area near the coronary ostium in the direction of the compact node represent the posterior input to the atroventricular node\cite{24}. In addition clinical observations during intra-operative ice mapping of atrioventricular nodal reentrant tachycardia have pointed to the possible variation of fast and slow pathway locations. In that study, slow pathway conduction was interrupted in two patients when cryolesions were applied not to the posterior or inferior inter-atrial septum but more superiorly along the tendon of Tordaro\cite{25}.

Table 4  Biophysical parameters of effective and ineffective ablation sites

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<tr>
<th>All effective sites (n=53)</th>
<th>All ineffective sites (n=72)</th>
<th>P value</th>
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<tr>
<td>Temp 5 s (°C)</td>
<td>45.1 ± 3.3</td>
<td>45.3 ± 4.6</td>
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<tr>
<td>Temp 10 s (°C)</td>
<td>51.0 ± 4.5</td>
<td>50.3 ± 5.7</td>
</tr>
<tr>
<td>Temp max (°C)</td>
<td>62.3 ± 9.8</td>
<td>58.8 ± 9.0</td>
</tr>
<tr>
<td>Power 5 s (W)</td>
<td>22.4 ± 7.9</td>
<td>22.6 ± 8.6</td>
</tr>
<tr>
<td>Power 10 s (W)</td>
<td>36.2 ± 12.2</td>
<td>34.3 ± 13.5</td>
</tr>
<tr>
<td>Power max (W)</td>
<td>45.3 ± 9.2</td>
<td>43.5 ± 13.2</td>
</tr>
<tr>
<td>Total energy (J)</td>
<td>2145 ± 1521</td>
<td>2195 ± 1510</td>
</tr>
<tr>
<td>Impedance max (Ω)</td>
<td>113 ± 36</td>
<td>108 ± 16</td>
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On the basis of these findings, anatomical- and electrogram-guided strategies were combined in the present study to avoid the possible disadvantages described above. The local electrogram during sinus rhythm was characterized by its detection of the longest and latest fractionated atrial activation. Sequential mapping was started posteroseptally near the coronary sinus ostium, targeting an ablation site characterized by the above mentioned criteria and proceeding stepwise to more midseptal sites if required. Recently, the anatomical- and electrogram-guided technique has been compared in a randomized study\cite{13}. Four of 25 patients assigned for the anatomical approach subsequently underwent successful electrogram-guided ablation after the initial attempt had been unsuccessful. The cross-over from the anatomical to the electrogram-guided approach in these patients and the average six to seven radiofrequency applications, respectively, may signify the value of both criteria for slow pathway ablation.

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Electrophysiological findings

The electrophysiological findings after the ablation procedure were suggestive of a selective slow pathway modification in all of our patients. Prolongation of the PR interval and unintended ablation of the fast pathway could be avoided with the combined stepwise approach. In previous studies, inadvertent ablation of the fast pathway has been observed with an incidence of 1-3% up to 14% using either the anatomical or electrogram-guided technique. This might be due to the previously described anatomical variability of the location of the fast and slow pathways. The atrioventricular Wenckebach point was prolonged after ablation. This finding, and the unaltered retrograde conduction capacity, is consistent with previous results following slow pathway ablation.

Following ablation, dual atrioventricular nodal physiology and single echo beats were observed in 43% and 39% of all patients, respectively. This was accepted as an endpoint of the procedure if no more than one echo beat or sustained tachycardia was inducible with or without isoproterenol. This concept is concordant to previous studies with a comparable incidence of persistent slow pathway conduction following the procedure.

Local electrogram characteristics

Analysis of all local bipolar electrograms during sinus rhythm demonstrated a significantly longer atrial electrogram duration at successful compared to unsuccessful target sites. A long duration of local atrial activation of \( \geq 70 \text{ ms} \) was associated with success with 86% sensitivity and 62% specificity. Long or fractionated local atrial activation may also be due previous applications of radiofrequency energy at this site. To exclude this possibility, we also assessed the first target site in patients with a single successful pulse and the first failed site in patients who required four or more radiofrequency pulses. Atrial electrogram duration was also significantly longer at effective compared to ineffective sites when only the initial ablation attempts were compared in these patients. These data strongly support the notion that prolonged atrial electrogram duration is indeed an important criterion for identifying the target site, as suggested by our approach. The findings are consistent with results reported by Kalbfeisch and coworkers. In their study, local atrial electrograms were significantly longer and more fractionated at successful compared to unsuccessful target sites. Interestingly, this difference was also present when target sites were exclusively selected on the basis of anatomical criteria.

In all likelihood, the reason for the low number of radiofrequency impulses required for slow pathway modification in this study may not only be the use of the combined method of mapping but also in the pre-selection and initial screening of local electrograms. The fact that only very late (later than atrial electrograms recorded on the high right atrium and His catheter), prolonged and fractionated atrial electrograms with low atrioventricular ratio were targeted, may have markedly facilitated the ablation procedures. There has been a wide range in the correlation between successful radiofrequency ablation and the recording of slow pathway potentials using a slow pathway-guided mapping approach. The range extends from a 100% incidence to studies that have reported no evidence for any specific slow pathway potentials despite effective radiofrequency application. This might be due to different electrogram recording techniques and the fact that mapping was not focused solely on the detection of slow pathway potentials in all studies. Nevertheless, previous reports and the present study have shown that this is not a prerequisite for the selection of successful sites for slow pathway ablation.

Biophysical parameters

In the present study, primarily the temperature-controlled mode of radiofrequency current application for transcatheter modulation of the atrioventricular node was introduced and analysed. Temperature response, power output level, impedance and the overall delivered energy did not differ between effective and ineffective radiofrequency applications. The almost similar temperature response at effective compared to ineffective sites indicates that suboptimal target site selection rather than poor tissue heating is responsible for failed radiofrequency application. The catheter tip temperature levels determined were concordant to results recently reported by Calkins and coworkers. In their multicentre study, the average temperature level measured 59 °C. However, the comparison with the present data might be influenced by the fact that fast and slow pathway ablation were not assessed separately in their study.

In our study, sudden rises in impedance occurred in 4-8% of all pulses. Experimental in vivo studies have demonstrated that temperature control but not regulation of power output alone accurately predicts lesion size. Abrupt changes in impedance are associated with overheating and formation of a carbonized layer adherent to the catheter tip. Consequently, sudden rises in impedance are potentially hazardous and may cause thromboembolic complications. In the clinical setting, thromboembolic events represent a substantial amount of procedure-related complications. Furthermore, procedure duration and fluoroscopy exposure are prolonged because following each impedance rise, the ablation catheter has to be withdrawn in order to exclude carbonization and thrombus formation at the catheter tip. It has been shown that the incidence of sudden rises in impedance is markedly reduced using a closed-loop feedback temperature-controlled mode compared to power-regulated radiofrequency delivery.
Limitations

The criteria used to select target sites were only tested in one group as all patients underwent the same approach for mapping and ablation of slow pathways. The method tested was not compared to other methods such as the anatomical approach alone. Since endocardial mapping was not focused on presence or absence of possible slow pathway potentials, no further pacing interventions or drug administrations, i.e. application of adenosine, were performed to confirm the evidence for suspected slow pathway potentials. There was no systematic attempt to record local electrograms at target sites immediately following energy delivery to assess the presence or absence of possible slow pathway potentials because the recording of target electrograms was influenced by catheter movements caused by junctional escape beats or extrasystoles.

Conclusion

The present study demonstrates that anatomical and electrogram criteria are helpful to guide catheter ablation of the slow pathway. The combined approach using prolonged, fractionated and late atrial activation to guide endocardial mapping is highly effective and selective in eliminating atrioventricular nodal reentrant tachycardia with a low number of radiofrequency pulses. Successful target sites are characterized by a longer atrial activation compared to unsuccessful slow pathway ablation sites. Strategies for slow pathway ablation should be based on both criteria using a temperature-controlled mode of radiofrequency delivery.

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