Laser catheter coagulation of atrial myocardium for ablation of atrioventricular nodal reentrant tachycardia

First clinical experience

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A new technique for ablation of atrioventricular nodal reentrant tachycardia, using catheter-directed continuous wave Nd-YAG laser light, 1064 nm, via a novel pin-electrode laser catheter, was applied in 10 patients aged 15–63 years (mean 43 years). A total of 22 laser pulses, 1–5 per patient, at 20 or 30 W, of 10–45 s (mean 27 s) were aimed at the postero-inferior aspect of the tricuspid annulus.

In all patients the tachycardia was rendered non-inducible at baseline as well as during orciprenaline administration. The amplitudes of the local atrial potentials diminished from 20 ± 0-5 mV before to 0-4 ± 0-4 mV after ablation, atrio-His intervals increased from 73 ± 7 ms to 157 ± 36 ms. Anterograde atrioventricular nodal refractory periods (212 ± 31 vs 238 ± 31 ms) and Wenckebach rate (174 ± 8 vs 167 ± 8 beats min⁻¹) did not change significantly (P>0.05). There were no complications or recurrent arrhythmias in a follow-up of 12–35 (mean 27) months.

Anatomically guided laser catheter coagulation of the postero-inferior aspect of the tricuspid valve ring is a safe and effective method for the cure of patients with common atrioventricular reentrant tachycardia.

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Key Words: laser, catheter ablation, atrioventricular nodal reentrant tachycardia, supraventricular tachycardia.

Introduction

Modification of the atrioventricular node without creating complete heart block can be successfully accomplished by catheter-directed radiofrequency current application resulting in cure of patients with atrioventricular nodal reentrant tachycardia. However, in some patients radiofrequency modification of the atrioventricular node requires repeated procedures with numerous radiofrequency current applications and long X-ray exposure times. Major complications including pericarditis, tamponade, atrioventricular block and pneumothorax have been reported. In addition, other arrhythmias may be induced.

Recently we have demonstrated the feasibility of transcatheter laser coagulation of atrial myocardium in dogs by using a novel electrode–laser catheter. Histopathologically, acute lesions showed intramural bleeding and coagulation necrosis, but there was no tissue vaporization or crater formation. Transmural lesions up to 1·8 cm in diameter were produced without perforation of the atrial walls. Chronic lesions showed clear-cut oval-shaped areas of transmural fibrosis. By directing the laser pulse towards the peri-nodal area, long-term modification of antegrade atrioventricular node conduction properties was achieved without the risk of heart block. Histological examination showed fibrosis of the peri-nodal atrial septum including the atrial approaches to the atrioventricular node, while the compact atrioventricular node itself was spared. Mapping guided laser catheter ablation of the atrioventricular junction was also tested and chronic atrioventricular block was induced selectively in the targeted segment of the conduction system. Laser energy could be applied in a controllable manner, and the method proved to be safe and effective.

Based on our experimental experience with laser irradiation of atrial myocardium, we have applied...
## Methods

### Patient population

The study group consists of 10 unselected patients aged 43 ± 14 years with the common type of drug-refractory symptomatic atrioventricular nodal re-entrant tachycardia. Their clinical characteristics are summarized in Table 1. Symptomatic tachycardia had been present for 20 ± 8 years and resulted in syncope in eight patients and sudden death with successful resuscitation in five patients. The mean tachycardia rate was 186 ± 17 beats.min⁻¹. Patients had been unsuccessfully treated with antiarrhythmic drugs. Four patients had mild idiopathic dilated cardiomyopathy and two had mild mitral valve prolapse. The remaining three had no structural heart disease and, when in sinus rhythm, their chest X-ray showed normal hearts in size and configuration.

### Laser and catheter system

A continuous-wave Nd-YAG laser, 1064 nm, with an integrated pilot laser was used in the study. The laser light was transmitted via a custom made laser catheter system which consists of a guiding catheter (Fig. 1) and a pin-electrode mapping and ablation laser catheter as described elsewhere. The optical characteristics of the fibre tip allowed for irradiation of an endocardial spot of 2.0–2.5 mm in diameter. The laser catheter was continuously flushed with saline throughout the procedure at a rate of 6 ml.min⁻¹, which was automatically augmented to 30–60 ml.min⁻¹ during the laser pulses. Three bipolar ECGs were recorded via the pin-electrodes and were simultaneously displayed with other intracardiac and surface-lead ECGs on a six-channel monitor and a 12-lead recorder at paper speeds of 25–200 mm.s⁻¹.

### Experimental protocol

In all patients, antiarrhythmic medication was discontinued before the study, and all patients gave written, informed consent for the laser ablation procedure. Standard 6-French quadripolar electrode catheters were introduced into the femoral (Seldinger technique) or brachial veins and placed in the coronary sinus, the high right atrium, the His bundle region and the right ventricular apex. Over a guide-wire, the 7-French pre-shaped guide catheter was advanced through the femoral vein into the right atrium and positioned, under X-ray control, on the postero-inferior aspect of the

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### Table 1: Clinical characteristics of patients undergoing laser catheter ablation of atrioventricular nodal re-entrant tachycardia

<table>
<thead>
<tr>
<th>Patient No.</th>
<th>Age (years)</th>
<th>Sex</th>
<th>Symptoms</th>
<th>Duration (years)</th>
<th>Number of drugs used</th>
<th>Max. heart rate AVNRT (beats . min⁻¹)</th>
</tr>
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<tbody>
<tr>
<td>1</td>
<td>59</td>
<td>F</td>
<td>SCD, S, D, P</td>
<td>30</td>
<td>4</td>
<td>180</td>
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<tr>
<td>2</td>
<td>35</td>
<td>M</td>
<td>SCD, S, P</td>
<td>11</td>
<td>5</td>
<td>200</td>
</tr>
<tr>
<td>3</td>
<td>53</td>
<td>M</td>
<td>S, D, P, L</td>
<td>17</td>
<td>3</td>
<td>170</td>
</tr>
<tr>
<td>4</td>
<td>32</td>
<td>F</td>
<td>SCD, S, P</td>
<td>9</td>
<td>6</td>
<td>200</td>
</tr>
<tr>
<td>5</td>
<td>44</td>
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<td>P, S, L</td>
<td>27</td>
<td>4</td>
<td>160</td>
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<tr>
<td>6</td>
<td>15</td>
<td>M</td>
<td>SCD, P, D</td>
<td>10</td>
<td>7</td>
<td>210</td>
</tr>
<tr>
<td>7</td>
<td>49</td>
<td>F</td>
<td>SCD, S, P, D</td>
<td>28</td>
<td>3</td>
<td>180</td>
</tr>
<tr>
<td>8</td>
<td>39</td>
<td>F</td>
<td>S, P, L</td>
<td>19</td>
<td>5</td>
<td>200</td>
</tr>
<tr>
<td>9</td>
<td>41</td>
<td>F</td>
<td>P, D, L</td>
<td>23</td>
<td>3</td>
<td>190</td>
</tr>
<tr>
<td>10</td>
<td>63</td>
<td>F</td>
<td>S, P, D, L</td>
<td>30</td>
<td>4</td>
<td>160</td>
</tr>
</tbody>
</table>

Mean ± SD 43 ± 14

AVNRT = atrioventricular nodal reentrant tachycardia; L = lightheadedness; P = palpitations; S = syncope; D = dyspnoea; SCD = sudden cardiac death; SD = standard deviation.

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**Figure 1** Distal intracardiac ends of the 7-French guide catheters as used in this study. The catheters were provided with two curves shaped as follows: one curve with a radius of 3 cm and an angle of 180° (type A) or of 90° (type B) followed in a vertical plane by the second curve with an angle of 90°.
cardia was inducible a second laser pulse was applied. 

extrastimuli, and by decremental and burst stimulation. 

The endpoint of the study was non-inducibility of the 

and during orciprenaline infusion 1-5—2-0 ug . kg^{-1}. 

Whenever the atrioventricular nodal re-entrant tachy- 

during atrioventricular nodal re-entrant tachycardia at baseline 

atrioventricular nodal re-entrant tachycardia at baseline 

during orciprenaline infusion 1-5–2-0 µg . kg^{-1}. 

In the first patient, a laser pulse of 30 s was applied 

during atrioventricular nodal re-entrant tachycardia. 

The immediate laser effects are summarized in Table 2. 

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A total of 22 laser pulses, 1–5 per patient, were applied: 

12 pulses during sinus rhythm and 10 pulses during the 

atrioventricular nodal re-entrant tachycardia.

Early effects

In the first patient, a laser pulse of 30 s was applied 

during atrioventricular nodal re-entrant tachycardia. 

The tachycardia converted to sinus rhythm in the 16th 

second of the pulse but was still inducible. Ventriculo- 

atrial interval during atrioventricular nodal re-entrant 

tachycardia was now 120 ms as compared to 40 ms prior 

to the laser pulse. Atrioventricular nodal re-entrant 
tachycardia ceased spontaneously after a maximum of 

13 cycles (Fig. 3, top). A second pulse of 30 s was 

applied, now during sinus rhythm, to an area infero- 

posterior and adjacent to that of the first pulse. Five 

cycles were inducible after the second pulse (Fig. 3, 

bottom).

In the second patient, a laser pulse of 30 s was 

applied during atrioventricular nodal re-entrant tachy- 
cardia. An accelerated junctional rhythm occurred in the 

4th second which was followed by sinus rhythm in the 

18th second of the pulse. Subsequent intra-cardiac 

stimulation produced two echo beats. Atrioventricular 
nodal re-entrant tachycardia was not inducible. In the 

third patient, three consecutive pulses of 10–15 s each 

showed no effects. A defect of the optical fibre was 
disclosed and the catheter was exchanged. The fourth 

cycle, applied during sinus rhythm, produced five atrial 

extrasystoles. Atrioventricular nodal re-entrant tachy- 
cardia was still inducible, the ventriculo-atrial interval 

was now 150 ms as compared to 45 ms measured before 

the pulse. A fifth pulse of 30 s applied infero-posterior 

and adjacent to the site of the preceding one, again 

during sinus rhythm, produced an accelerated junctional 
rhythm lasting from the 7th to the 26th second when 
sinus rhythm resumed. Atrioventricular nodal re-entrant 

Follow-up

Subsequent ECG monitoring was performed for 48 h, 
followed by daily 12-lead ECGs at rest for at least 3 
days. Creatine phosphokinase and creatine kinase MB 

isoenzyme concentrations were measured before and 
after laser application. Chest X-ray, Holter monitoring, 
echocardiography and exercise tests were performed 3–5 
days and 6–22 weeks after the ablation procedure. Follow-up electrophysiological study was carried out 
after 6–22 weeks, except in the last patient in whom only 
event-monitoring by means of a wrist watch recorder 

was performed.

Data were analysed using Student's test. All data 
are expressed as mean ± standard deviation. Probability 
values of P<0.05 were considered significant.

Results

The immediate laser effects are summarized in Table 2. 

A total of 22 laser pulses, 1–5 per patient, were applied: 

12 pulses during sinus rhythm and 10 pulses during the 

atrioventricular nodal re-entrant tachycardia.

Figure 3 Diagram of the right atrial cavity and the 
pin-electrode laser catheter directed towards the area of 

interest by means of a type A guide catheter. EV= eustachian valve; IVC=inferior vena cava, CT=crista 

terminalis, SVC=superior vena cava, TT=tendon of 

Todaro, FO=foramen ovale, CS=coronary sinus ostium, 

L=assumed area of lesion induced.
tachycardia was not inducible thereafter. In the fourth patient, a laser pulse of 30 s was delivered during atrioventricular nodal re-entrant tachycardia which was converted to sinus rhythm in the 12th second. Only short runs of atypical atrial flutter of up to 10 s duration were inducible after the pulse. In the fifth patient, four consecutive pulses of up to 45 s were without effect because it was difficult to maintain a stable position on the targeted area with the type B guide catheter. Therefore, it was replaced by a type A guide catheter. Eventually, a fifth pulse of 26 s abolished the atrioventricular nodal re-entrant tachycardia. In the sixth patient, similar difficulties were encountered with the type B guide catheter. Again, by using a type A guide, the ablation procedure was successful. In the following patients only type A guide catheters were used and pulses at a power of 20 instead of 30 W were applied. In the seventh patient, the laser pulse converted the tachycardia to stable sinus rhythm in the 9th second. In the eighth patient, a sharp spike suggesting a fast pathway potential was recorded from the targeted area remote from the His bundle region. The first pulse was without effect. After a second pulse the sharp spike was abolished and atrioventricular nodal re-entrant tachycardia was non-inducible (Fig. 4). In the ninth patient, a pulse of 10 s at 20 W abolished the atrioventricular nodal re-entrant tachycardia (Fig. 5). In the 10th patients, the laser pulse was applied during sinus rhythm. The fractionated low amplitude local atrial potential diminished gradually during laser application (Fig. 6). Only short episodes of atrial fibrillation up to 4 s were inducible after the pulse.

All the procedures were successful and without complication. The electrophysiological effects of laser pulses are summarized in Table 3. Normal 1:1 atrioventricular conduction was preserved in all patients after laser application. No patient showed atrioventricular duality or retrograde atrioventricular block. Total duration of studies, from the insertion until removal of catheters, was 1-7-3-5 (mean 2-4 ± 0-7) h and X-ray exposure time totalled 4-7-33-7 (18-5 ±9-1) min. Peak creatine phosphokinase was elevated in three patients at values of 92, 111 and 134 U.l⁻¹.

### Table 2 Immediate effects of laser pulses aimed at the postero-inferior aspect of the tricuspid annulus in patients with the common type of atrioventricular nodal re-entrant tachycardia

<table>
<thead>
<tr>
<th>Patient</th>
<th>Laser pulse</th>
<th>Applied during</th>
<th>Laser effects</th>
</tr>
</thead>
<tbody>
<tr>
<td>No.</td>
<td>TCL (ms)</td>
<td>Length (s)</td>
<td></td>
</tr>
<tr>
<td>1</td>
<td>335</td>
<td>1</td>
<td>30</td>
</tr>
<tr>
<td>2</td>
<td>295</td>
<td>1</td>
<td>30</td>
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<td>3</td>
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<td>4</td>
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</tr>
<tr>
<td>5</td>
<td>375</td>
<td>1-4</td>
<td>30, 45, 45, 42</td>
</tr>
<tr>
<td>6</td>
<td>285</td>
<td>1-2</td>
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<tr>
<td>7</td>
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</tr>
<tr>
<td>10</td>
<td>365</td>
<td>1</td>
<td>15</td>
</tr>
</tbody>
</table>

TCL=tachycardia cycle length; AVNRT=atrioventricular nodal reentrant tachycardia; SR=sinus rhythm; AES=atrial extrasystole; AJR=accelerated junctional rhythm; AFl=atrial flutter; AFlb=atrial fibrillation; A=atrial potential amplitude (prior to–after laser pulse); AH=atrio-His conduction interval; VA=ventriculoatrial conduction interval; SVT=supraventricular tachyarhythmia inducible by intracardiac stimulation; ¥=no inducible SVT; Laser power: 30 W (patient 1–7); 20 W (patient 8–10).

### Long-term results

The effects of laser ablation were followed for 12 to 35 (mean 27 ± 7) months. PR intervals had slightly decreased at 1 month but had not returned to pre-ablation values. Chronic first-degree atrioventricular block was present in only one patient. All patients demonstrated normal exercise capacity. Chest X-rays showed the hearts were of normal size, including the four patients with dilated cardiomyopathy prior to the laser treatment. They were off medication and without recurrent atrioventricular nodal re-entrant tachycardia. During follow-up invasive studies in nine patients, atrioventricular nodal re-entrant tachycardia remained non-inducible. In the 10th patient, the event monitoring performed by means of a wrist-watch recorder showed sinus rhythm with normal heart rate throughout the follow-up and she remained asymptomatic.

In four patients coronary angiography showed normal coronary arteries and normal coronary venous drainage with normal morphology of the coronary sinus ostium. An enlarged coronary sinus ostium was disclosed in two patients (6 and 8).
Ablation of AVNRT

Discussion

This study demonstrates that transcatheter application of Nd-YAG laser light can abolish atrioventricular nodal re-entrant tachycardia without complications and with a high success rate. Anatomically guided laser coagulation of the postero-inferior atrioventricular nodal region between the tricuspid valve ring and coronary sinus ostium caused loss of antegrade atrioventricular duality with preservation of atrioventricular conduction and rendered atrioventricular nodal re-entrant tachycardia non-inducible in all patients.

Two approaches have been developed for catheter ablation of atrioventricular nodal reentry: radiofrequency lesions placed anteriorly near the apex of the triangle of Koch, and lesions applied posteriorly, adjacent to the ostium of the coronary sinus. Anterior lesions produce an increase in the AH interval without changing the mean atrioventricular nodal Wenckebach block cycle length. The efficacy of the posterior radiofrequency approach is comparable to or greater than the anterior approach and it does not produce first degree atrioventricular block. However, laser pulses aimed at the postero-inferior area of the atrioventricular nodal region produced lengthening of the AH interval in all patients. This finding conforms to our experience with laser irradiation of peri-nodal tissue in dogs. Recently, reversible increases of the atrioventricular interval were reported after the posterior radiofrequency approach. The increase in the AH interval can best be explained by interruption of the approaches to the atrioventricular node located in the irradiated region. Second- and third-degree atrioventricular block caused by total depletion of atrial muscle at the approaches to the atrioventricular node has been described.

Analysis of the current published data show a similar success rate with anatomically and electrophysiologically guided radiofrequency approaches. Both

Figure 3 Electrocardiograms recorded at electrophysiological study showing, Top: an incessant common type atrioventricular nodal reentrant tachycardia at a cycle length (TCL) of 335 ms prior to (left) and after the first laser pulse of 30 s (right). Atrial burst stimulation, S1S2 = 300 ms, still induced atrioventricular nodal reentrant tachycardia but the ventriculo-atrial interval lengthened from 40 to 120 ms and sinus rhythm resumed after 13 cycles (A1-A13). Bottom: after a second pulse adjacent to the area of the first, but postero-inferior to the coronary sinus ostium, only seven consecutive retrograde atrial activations (A1-A7) were induced by atrial stimulation (S1/S2/S3 = 500/300/240 ms). Ventriculo-atrial intervals increased gradually from 80 to 150 ms followed by sinus rhythm with normal atrioventricular conduction. II and V1 are surface leads, RAE and RVE are intracardiac right atrial and ventricular electrograms, A=atrial, V=ventricular, and H=His bundle potentials, S=stimulus artifact; paper speed: 100 mm.s⁻¹.
Figure 4  Electrocardiograms recorded at invasive electrophysiological study, showing local atrial potentials recorded via the mapping and ablation pin-electrode laser catheter (MAP 1,2,3) from the postero-inferior atrial septum in a patient with atrioventricular nodal reentrant tachycardia. A sharp spike, suggesting a fast pathway potential (arrows), is conspicuous between the atrial (A) and the ventricular (V) potentials (left), which is abolished by laser irradiation (arrows, right). The atrioventricular nodal re-entrant tachycardia was not inducible after the laser pulse. Note: weakening of local atrial potentials A, and lengthening of atrioventricular intervals A^/P, after the pulse as compared to A and AV/PR prior to that pulse. II=surface lead, LVE = intracardiac left ventricular electrograms; paper speed: 50 mm. s~’.

approaches were comparable in efficacy and duration. However, more radiofrequency applications are required using the anatomical approach: 5–24 vs 1–2.

Recently, it has been demonstrated that the atrial ECG characteristics that have been found to be useful in identifying effective posteroseptal slow pathway ablation sites in patients with atrioventricular nodal re-entrant tachycardia are equally prevalent in patients without atrioventricular nodal re-entrant tachycardia or dual nodal physiology[21]. Atrial ECGs in the posteroseptal area are broader and contain more deflections than at other areas in the right atrium, possibly because of conduction properties of the posterior transitional zone that are independent of the presence of atrioventricular nodal re-entrant tachycardia. Histologically, a recent study demonstrated that slow potentials originate in subendocardially located transitional fibres[22]. However, it is still unclear whether the slow pathway originates at the postero-inferior atrial inputs of the transitional zone or whether it involves the junction of both tissues. Histopathological study following successful radiofrequency current ablation of slow pathways in a patient with atrioventricular nodal re-entrant tachycardia demonstrated a sharply demarcated atrial lesion extending from the septal portion of the tricuspid annulus to the posterior border of the atrioventricular node. The lesion did not encompass the compact atrioventricular node, supporting the hypothesis that the slow pathway is comprised of atrial approaches to the
Figure 5  Electrograms recorded at invasive electrophysiological study showing atrioventricular nodal reentrant tachycardia (AVNRT) which is interrupted by laser irradiation. In the 3rd second of the laser pulse, retrograde atrial activation is abolished (0) and sinus rhythm (SR) resumes. Noise during the laser pulse is caused by a defective pin-electrode in the mapping and ablation catheter. I=Surface lead, 1,2,3=local intracardiac and HRA=high right atrial electrograms, A and V=atrial and ventricular potentials; paper speed: 100 mm . s⁻¹.

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atrioventricular node and is distinct from the compact node. Although the electrophysiological criteria of the dual atrioventricular nodal pathway seems to be well established, the anatomical substrate is still unclear. Histological studies of the atrioventricular conduction system in patients with electrophysiologically proven dual atrioventricular pathways prior to cardiac transplantation showed the atrioventricular node to be of normal structure. The anatomical substrate for conduction over dual pathways may be too subtle to be detected by histological studies or it may be that the potential for dual pathways is ubiquitous. Thus, atrioventricular nodal re-entrant tachycardia could represent functional anomalies of an otherwise anatomically normal structure. Pathophysiological studies suggest that the atrioventricular node is an anisotropic three-dimensional structure and its function is still unknown.

We conclude that regardless of the presence or absence of 'slow' or 'fast' potentials in the local electrocardiograms, a sufficiently large lesion produced by anatomically guided Nd-YAG laser application in the postero-inferior aspect of the atrioventricular node, between the coronary sinus ostium and the tricuspid annulus, will abolish atrioventricular nodal re-entrant tachycardia while maintaining intact atrioventricular conduction.

The special A-type guide catheter facilitates the stable orthogonal end-on position of the electrode laser catheter on the atrioventricular nodal area during laser application. By means of this self-guiding system, catheterization of the atrioventricular nodal area was achieved quickly, with relative ease, without demanding high expertise in catheter manipulation, thereby reducing the procedure duration and X-ray exposure times and contributing to the safety and efficacy of the procedure. As the laser lesion produced by a single pulse can measure more than 1 cm in diameter, it can encompass the entire target region, extending from the atrial margins of the tricuspid annulus to the ostium of the coronary sinus. Furthermore, the lesion reaches deep intramurally into the triangle below the inter-atrial septum between the tricuspid and mitral valve rings.

Anomalies of the coronary sinus region that are relatively frequently encountered in patients with supraventricular tachyarrhythmias and that may be present in patients in this study, may cause difficulties in
Figure 6 electrocardiograms recorded at invasive electrophysiological study showing the weakening of a fragmented atrial potential with a 'slow' potential component which is abolished by laser irradiation (arrows). The local atrial electrocardiogram is recorded via the mapping and ablation pin-electrode laser catheter (MAP) prior to (left), during (mid), and after a laser pulse (right) of 16 s at 20 W aimed at the postero-inferior area of the inter-atrial septum during sinus rhythm, in a patient with atrioventricular nodal reentrant tachycardia. I and II are surface leads, HRA=high right atrial electrocardiograms, A=atrial and V=ventricular potentials; paper speed: 100 mm . s$^{-1}$.

Table 3 Atrioventricular nodal function after successful slow pathway laser catheter ablation of atrioventricular nodal re-entrant tachycardia

<table>
<thead>
<tr>
<th>Patient No.</th>
<th>Effective laser pulses</th>
<th>Effective refractory period (ms)</th>
<th>Wenckebach rate (beats . min$^{-1}$)</th>
</tr>
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<tbody>
<tr>
<td></td>
<td>No.</td>
<td>Total energy (J)</td>
<td>Anterograde pre/post</td>
</tr>
<tr>
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<td>2</td>
<td>1800</td>
<td>250/270</td>
</tr>
<tr>
<td>2</td>
<td>1</td>
<td>900</td>
<td>250/250</td>
</tr>
<tr>
<td>3</td>
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<td>212 ± 31/238 ± 31</td>
<td>320 ± 25/384 ± 29</td>
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<tr>
<td>pre vs post</td>
<td>$P&lt;0.05$</td>
<td>$P&lt;0.001$</td>
<td>$P&gt;0.05$</td>
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</table>

stable positioning of the ablation catheter. A gradual decrease in local atrial potentials and an increase in the AH interval present in all patients reflect the spreading coagulation zone in the irradiated atrial myocardium and document the effectiveness of the laser light. The onset of a junctional escape rhythm can be considered as a sensitive marker of successful ablation$^{26,27}$. Regardless of the energy source used, an accurate targeted lesion of sufficient size will ablate arrhythmogenic substrates in the heart. In our experience, by using the novel electrode laser catheter system, the Nd-YAG laser produces larger and better controllable lesions than radiofrequency current application, without untoward effects on the atrial walls$^{28}$. Based on the results of this study, we have extended the laser method to all arrhythmogenic substrates amenable to ablative procedures, regardless of their location in the heart. Since the last procedure in this study we have not performed routine follow-up electrophysiological studies. Invasive procedures with the option of ablation attempts are only carried out if symptomatic arrhythmias were documented during event monitoring$^{29}$.

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References


