Atrial pacing for suppression of early reinitiation of atrial fibrillation after successful internal cardioversion

H.-F. Tse, C.-P. Lau and G. M. Ayers

Division of Cardiology, Department of Medicine, University of Hong Kong, Queen Mary Hospital, Hong Kong, China; †InControl Inc., Redmond, WA, U.S.A.

Aims To evaluate the efficacy of atrial pacing in the suppression of early reinitiation of atrial fibrillation after successful internal cardioversion.

Methods and Results The efficacy of atrial pacing in suppressing early reinitiation of atrial fibrillation was studied in 12 of 45 (29%) patients with early reinitiation of atrial fibrillation after successful cardioversion. These patients were randomized to undergo either repeated defibrillation alone or repeated defibrillation followed by high right atrial pacing at 500 ms in a crossover fashion. In patients with persistent early reinitiation of atrial fibrillation despite atrial pacing at 500 ms and repeated defibrillation, atrial pacing at 300 ms was tested. Lastly, if early reinitiation of atrial fibrillation persisted, administration of intravenous sotalol (1·5 mg . kg⁻¹) was tested. Atrial pacing at 500 ms after defibrillation prevented early reinitiation of atrial fibrillation in five of 12 (42%) patients, and was significantly more effective than repeated defibrillation (0/9 patients, 0%, P<0·05). During atrial pacing at 500 ms, the density of atrial premature depolarizations (APDs) was significantly decreased (2·4 ± 2·4 APDs . min⁻¹ vs 14·2 ± 4·8 APDs . min⁻¹, P<0·05) and delayed the onset of early reinitiation of atrial fibrillation (33 ± 17s vs 11 ± 11 s, P<0·05). Atrial pacing at 300 ms decreased the coupling interval of atrial premature depolarization as compared to no pacing and during atrial pacing at 500 ms (P<0·05), but without early reinitiation of atrial fibrillation suppression. Administration of intravenous sotalol was effective in preventing early reinitiation of atrial fibrillation in five of seven (71%) patients where pacing failed to suppress early reinitiation of atrial fibrillation.

Conclusion The results of this study suggest that atrial pacing can be useful when combined with transvenous defibrillation in patients with early reinitiation of atrial fibrillation.

(Key Words: Atrial pacing, internal cardioversion, atrial fibrillation.)

See page 1119 for the Editorial comment on this article

Introduction

Early reinitiation of atrial fibrillation is frequently observed after successful cardioversion. Previous studies have reported that early reinitiation of atrial fibrillation occurred in 13–36% of patients shortly (within minutes) after successful internal cardioversion[1–4]. Recent studies in patients with an implantable atrial defibrillator demonstrated that early reinitiation of atrial fibrillation occurred in 51% of these patients and was observed during treatment in 20–27% of episodes[5,6]. Thus, suppression of early reinitiation of atrial fibrillation has important implications in temporary low energy internal cardioversion and decreases the need for therapy with an implantable atrial defibrillator. This prospective study evaluates the efficacy of atrial overdrive pacing in suppressing early reinitiation of atrial fibrillation in patients who underwent internal cardioversion for persistent atrial fibrillation.
Methods

Study population

From December 1995 to December 1997, 45 consecutive patients with persistent atrial fibrillation of at least 1 month's duration underwent successful internal cardioversion in our centre. Patients with early reinitiation of atrial fibrillation after successful internal cardioversion were prospectively enrolled into this study. Early reinitiation of atrial fibrillation was defined as the recurrence of a sustained atrial fibrillation episode within 2 min after a shock that resulted in confirmed sino-atrial rhythm for at least one beat. A detailed clinical examination was performed and a complete medical history was taken. A routine 12-lead ECG, 24 h Holter monitoring, chest X-ray, routine laboratory and thyroid parameters, transthoracic and transoesophageal echocardiography were assessed in all patients. At the time of internal cardioversion, all class I or III antiarrhythmic drugs were stopped for at least five half-lives. None of the patients were treated with amiodarone. All patients were treated with oral anticoagulation for at least 3 weeks prior to the procedure to achieve an international normalized ratio of 2–3. All patients gave written informed consent before the procedure according to a protocol approved by the local ethics committee.

Internal cardioversion procedure

Details of the internal cardioversion procedure used in our laboratory have been reported previously[7]. In brief, two custom-built 6 Fr defibrillation catheters (Elecath, Rahway, NJ, U.S.A.) were positioned, one in the coronary sinus and one in the anterolateral right atrium. The right atrial catheter served as the cathode and the coronary sinus catheter as the anode for shock delivery. A third 6 Fr catheter was positioned in the right ventricular apex for shock synchronization and post-shock ventricular pacing. The defibrillation catheters were connected to an external atrial defibrillator (XAD, In-Control Inc.) capable of delivering R wave synchronized biphasic (3/3 ms) shocks with a leading-edge voltage that could be programmed between 10 and 400 V. Patients were sedated with intravenous midazolam (0-05 mg, kg⁻¹) and pethidine (0-5 mg, kg⁻¹), and additional doses were given as required. Beginning with a 20 V test shock, R wave synchronized shocks were delivered starting at 180 V, increasing in steps of 40 V until sinus rhythm was restored or the highest output of 400 V was delivered. Between unsuccessful defibrillation attempts, at least 2 min was permitted to elapse before the next shock was applied.

Study protocol (Fig. 1)

In patients with early reinitiation of atrial fibrillation, when energy was successfully delivered, the defibrillation shock was repeated to confirm the reproducibility of early reinitiation of atrial fibrillation. Patients in whom atrial fibrillation recurred, were randomized to undergo atrial pacing at a drive cycle length of 500 ms immediately after defibrillation or repeated defibrillation without pacing in a cross-over fashion. Pacing was performed in the high right lateral atrium using the distal pair of electrodes on the defibrillation catheter. Pacing was performed using a 10 mA pulse of 2 ms duration using a programmable stimulator. In case there was a failure in capture during pacing, the study protocol was repeated using a higher output to ensure consistent capture. Atrial pacing was continued for up to 5 min after defibrillation or until early reinitiation of atrial fibrillation occurred. In patients who failed both interventions after cross-over, atrial defibrillation and post-shock pacing at a cycle length of 300 ms was tested. If this intervention also failed to suppress early reinitiation of atrial fibrillation, intravenous sotalol was administered (at a dose of 1·5 mg, kg⁻¹ over 30 min) prior to repeated internal defibrillation.

During the procedure, two electrocardiographic leads (leads II and V1) and the bipolar intracardiac electrograms from the anterolateral right atrium and coronary sinus were recorded on paper or optical disk. The filter setting for the intracardiac electrograms were 30 to 500 Hz. Following successful defibrillation, the rhythm was monitored for up to 5 min to determine: (1) the time
of onset of early reinitiation of atrial fibrillation after defibrillation; (2) the density of atrial premature depolarizations; (3) the coupling interval of atrial premature depolarization and (4) the mean atrial cycle length of 3–5 preceding beats, using the high right atrial electrogram recorded from the distal pair of electrodes on the defibrillation catheter.

was significantly shorter than those atrial premature depolarization without early reinitiation of atrial fibrillation (426 ± 38, P=0.02). The mean cycle length of the preceding atrial rhythm of the APD was 879 ± 382 ms (range: 460–1320 ms).

Effects of atrial pacing

High right atrial pacing at a cycle length of 500 ms, started immediately after defibrillation, prevented early reinitiation of atrial fibrillation in five of 12 patients and was significantly more effective than repeated defibrillation in preventing early reinitiation of atrial fibrillation (42% vs 0%, P<0.05) (Fig. 3). Atrial pacing significantly decreased the density of the atrial premature depolarization (3.4 ± 2.4 APDs·min⁻¹ vs 16.4 ± 9.8 APDs·min⁻¹, P<0.05) and increased the mean coupling interval of the atrial premature depolarization (420 ± 32 ms vs 398 ± 19 ms, P<0.05) as compared to no pacing (Fig. 2b). None of the patients in whom atrial pacing at 500 ms prevented early reinitiation of atrial fibrillation had a recurrence of atrial fibrillation after stopping atrial pacing.

In the remaining seven patients, atrial pacing at 500 ms did not prevent early reinitiation of atrial fibrillation (Fig. 4a), however, it significantly delayed the onset of early reinitiation of atrial fibrillation (33 ± 16 s vs 11 ± 11 s, P<0.05), decreased the density of atrial premature depolarization (3.5 ± 2.0 APDs·min⁻¹ vs 14.2 ± 4.8 APDs·min⁻¹, P<0.05) and increased the mean coupling interval of atrial premature depolarization as compared to no pacing (404 ± 30 ms vs 386 ± 18 ms, P<0.05) (Fig. 5).

On comparing the baseline clinical parameters in patients with or without atrial pacing, suppression of early reinitiation of atrial fibrillation showed no significant difference with respect to age, sex, duration of atrial fibrillation, atrial defibrillation threshold, left atrial size

### Table 1 Clinical characteristics of patients with early reinitiation of atrial fibrillation

<table>
<thead>
<tr>
<th>Patient no.</th>
<th>Sex</th>
<th>Age (years)</th>
<th>Heart disease</th>
<th>LVEF</th>
<th>LA size (cm)</th>
<th>AF duration (months)</th>
<th>ADFT (volts)</th>
<th>Suppression of ERAF by AP</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>M</td>
<td>49</td>
<td>Nil</td>
<td>0.69</td>
<td>3.9</td>
<td>9</td>
<td>340</td>
<td>Yes</td>
</tr>
<tr>
<td>2</td>
<td>M</td>
<td>36</td>
<td>Nil</td>
<td>0.63</td>
<td>4.3</td>
<td>7</td>
<td>340</td>
<td>No</td>
</tr>
<tr>
<td>3</td>
<td>F</td>
<td>59</td>
<td>Hypertension</td>
<td>0.53</td>
<td>4.4</td>
<td>18</td>
<td>260</td>
<td>Yes</td>
</tr>
<tr>
<td>4</td>
<td>M</td>
<td>58</td>
<td>Hypertension</td>
<td>0.63</td>
<td>5.4</td>
<td>8</td>
<td>380</td>
<td>No</td>
</tr>
<tr>
<td>5</td>
<td>M</td>
<td>60</td>
<td>Nil</td>
<td>0.54</td>
<td>4.5</td>
<td>3</td>
<td>380</td>
<td>No</td>
</tr>
<tr>
<td>6</td>
<td>M</td>
<td>57</td>
<td>Dilated cardiomyopathy</td>
<td>0.35</td>
<td>4.0</td>
<td>36</td>
<td>340</td>
<td>Yes</td>
</tr>
<tr>
<td>7</td>
<td>M</td>
<td>72</td>
<td>Nil</td>
<td>0.61</td>
<td>4.3</td>
<td>60</td>
<td>260</td>
<td>No</td>
</tr>
<tr>
<td>8</td>
<td>M</td>
<td>51</td>
<td>Nil</td>
<td>0.75</td>
<td>3.9</td>
<td>4</td>
<td>340</td>
<td>Yes</td>
</tr>
<tr>
<td>9</td>
<td>M</td>
<td>32</td>
<td>Nil</td>
<td>0.63</td>
<td>4.3</td>
<td>24</td>
<td>380</td>
<td>No</td>
</tr>
<tr>
<td>10</td>
<td>M</td>
<td>64</td>
<td>Hypertension</td>
<td>0.43</td>
<td>3.4</td>
<td>48</td>
<td>260</td>
<td>No</td>
</tr>
<tr>
<td>11</td>
<td>M</td>
<td>49</td>
<td>Nil</td>
<td>0.56</td>
<td>3.8</td>
<td>36</td>
<td>220</td>
<td>No</td>
</tr>
<tr>
<td>12</td>
<td>M</td>
<td>30</td>
<td>Nil</td>
<td>0.52</td>
<td>3.6</td>
<td>72</td>
<td>220</td>
<td>Yes</td>
</tr>
</tbody>
</table>

Mean ± SD

51 ± 13

0.56 ± 0.1

4.2 ± 0.5

27 ± 23

310 ± 62

ADFT=atrial defibrillation therapy; AF=atrial fibrillation; AP=atrial pacing; ERAF=early reinitiation of atrial fibrillation; LA=left atrial; LVEF=left ventricular ejection fraction.

Statistics

Continuous variables are presented as mean value ± 1 standard deviation and were compared with an unpaired Student’s t-test or by analysis of variance, as appropriate. Discrete variables were compared using the Fisher’s exact test. P-values <0.05 were considered statistically significant.

Results

Early reinitiation of atrial fibrillation after successful internal cardioversion

Fourteen of 45 patients (31%) developed early reinitiation of atrial fibrillation after successful internal cardioversion. Two of 14 (14%) patients had only one episode of early reinitiation of atrial fibrillation and remained in stable sinus rhythm after the repeated shock. Twelve patients with early reinitiation of atrial fibrillation despite repeated successful internal cardioversion, were enrolled into this study. Their clinical features are summarized in Table 1. For patients with early reinitiation of atrial fibrillation, atrial fibrillation was reinitiated by an APD with a mean coupling interval of 380 ± 43 ms (range: 260–420 ms), at an average of 16 ± 22 s (range: 2–80 s) after successful cardioversion (Fig. 2a). The coupling interval of atrial premature depolarization with early reinitiation of atrial fibrillation...
and ejection fraction. After successful defibrillation, the time of onset of early reinitiation of atrial fibrillation, the mean cycle length of the preceding atrial rhythm and the mean coupling interval of the atrial premature depolarization that initiated atrial fibrillation were not significantly different between patients with or without early reinitiation of atrial fibrillation suppression. Of four patients with early reinitiation of atrial fibrillation who had a significant sinus pause (>1000 ms) after internal cardioversion, atrial pacing only prevented early reinitiation of atrial fibrillation suppression. Of four patients with early reinitiation of atrial fibrillation who had a significant sinus pause (>1000 ms) after internal cardioversion, atrial pacing only prevented early reinitiation of atrial fibrillation during atrial pacing (Table 2).

**Effects of changes in the rate of pacing**

In the seven patients with failure of suppression of early reinitiation of atrial fibrillation by repeated defibrillation and atrial pacing at 500 ms, atrial pacing at 300 ms immediately after defibrillation also did not prevent early reinitiation of atrial fibrillation (Fig. 4b). The time of onset of early reinitiation of atrial fibrillation after defibrillation (35 ± 31 s vs 33 ± 27 s, *P*=0.8) and the density of atrial premature depolarization (4.2 ± 1.8 APDs . min⁻¹ vs 3.5 ± 2.0 APDs . min⁻¹, *P*=0.4) were similar when pacing at 300 ms as compared to pacing at 500 ms. During atrial pacing at 300 ms, the coupling interval of the atrial premature depolarization that reinitiated atrial fibrillation was significantly shorter than the coupling interval without pacing or with atrial pacing at 500 ms (Fig. 5).

**Effect of intravenous sotalol**

Administration of intravenous sotalol followed by repeated defibrillation was performed in the seven of 12 patients (58%) with refractory early reinitiation of atrial fibrillation. After sotalol infusion, there were no significant changes in the atrial defibrillation threshold, as compared to baseline (293 ± 35 V vs 310 ± 42 V, *P*>0.05); however, five of seven patients (71%) had no early reinitiation of atrial fibrillation after sotalol administration and repeated defibrillation. In these two patients with refractory early reinitiation of atrial fibrillation, although sotalol increased the mean coupling interval of their atrial premature depolarization from 350 ms to 410 ms, early reinitiation of atrial fibrillation still occurred.

**Discussion**

This study suggests that single-site atrial pacing can prevent or delay early reinitiation of atrial fibrillation after successful internal cardioversion. There are no
clinical parameters predictive of which patients may benefit from atrial pacing after defibrillation to prevent early reinitiation of atrial fibrillation. Atrial pacing at a shorter cycle length (300 ms) does not have any incremental benefit in preventing early reinitiation of atrial fibrillation. In patients with persistent early reinitiation of atrial fibrillation despite repeated defibrillation and atrial pacing, intravenous sotalol may be useful in its suppression.

Early reinitiation of atrial fibrillation

Early reinitiation of atrial fibrillation can occur in patients with atrial fibrillation following successful electrical cardioversion, using either internal\(^1\)\(^{-}\)\(^4\) or external\(^5\) methods. The incidence of early reinitiation of atrial fibrillation after external cardioversion is unknown. Recent studies have demonstrated that early reinitiation of atrial fibrillation occurs in a significant proportion of patients after internal cardioversion\(^1\)\(^{-}\)\(^4\). These studies suggest that the occurrence of this phenomenon was not related to the arrhythmia duration or to any of the evaluated clinical parameters (age, gender, left atrial size, left ventricular ejection fraction, defibrillation threshold and type of underlying heart disease). Furthermore, early reinitiation of atrial fibrillation can complicate the treatment of atrial fibrillation episodes with an implantable atrial defibrillator, resulting in the need for additional shock delivery and reducing the clinical efficacy of this treatment modality\(^5\)\(^{-}\)\(^6\). Prior studies\(^8\) have demonstrated that repeated cardioversion was only effective in preventing early reinitiation of atrial fibrillation in 10% of patients, and early reinitiation of atrial fibrillation could not be prevented by increasing the energy of the defibrillation shock. In this study, we found that 30% of our patients with persistent atrial fibrillation had early reinitiation of atrial fibrillation after internal cardioversion. Only 15% of these patients with early reinitiation of atrial fibrillation could be successfully treated with repeated cardioversion. Therefore, early reinitiation of atrial fibrillation is indeed a significant clinical problem in patients with atrial fibrillation treated by internal cardioversion and an additional therapy is needed to prevent early reinitiation of atrial fibrillation.

Atrial pacing for prevention of atrial fibrillation recurrence

In patients with sick sinus syndrome, atrial pacing has been shown to be associated with a lower incidence of atrial fibrillation than with ventricular pacing\(^9\)\(^{-}\)\(^{13}\). In a subset of patients with bradycardia-dependent atrial fibrillation, atrial pacing was shown to reduce the frequency of recurrent atrial fibrillation\(^14\)\(^{-}\)\(^{15}\). Furthermore, a recent clinical study has demonstrated that chronic atrial pacing can prevent recurrence of atrial fibrillation in patients with drug-refractory atrial fibrillation who had an indication for pacing\(^16\). However, there is no data on the effect of atrial pacing in preventing atrial fibrillation recurrence after electrical cardioversion. Acute suppression of early reinitiation of atrial fibrillation by atrial pacing has important implications for the use of temporary low energy internal cardioversion and implantable atrial defibrillator in the treatment of atrial fibrillation.

This is the first systematic study to investigate the effects of atrial pacing for the prevention of early reinitiation of atrial fibrillation after successful internal cardioversion. Our results have demonstrated that single-site atrial overdrive pacing can prevent early reinitiation of atrial fibrillation in about 42% of patients. Furthermore, in those patients in whom early reinitiation of atrial fibrillation could not be prevented by atrial pacing, the time of onset of early reinitiation of atrial fibrillation was significantly delayed by atrial pacing. In this study, the clinical characteristics of the patients, the coupling interval of the atrial premature depolarization that triggered the atrial fibrillation and the cycle length of the atrial rhythm preceding the atrial premature depolarization were found not to be useful in predicting whether atrial pacing was effective in preventing early reinitiation of atrial fibrillation. Furthermore, more aggressive and higher-rate atrial pacing did not have an incremental benefit in preventing early reinitiation of atrial fibrillation.

Mechanisms of prevention of early reinitiation of atrial fibrillation

Although the mechanism of early reinitiation of atrial fibrillation after internal cardioversion remains unclear, it is usually preceded by atrial premature depolarization with a short sinus-APD coupling interval and occurs within minutes after cardioversion\(^1\)\(^{-}\)\(^4\). It is uncertain whether early reinitiation of atrial fibrillation is due to atrial premature depolarization that serve as a trigger for atrial fibrillation, whether transient changes in atrial electrophysiological properties following defibrillation renders the atrial substrate more vulnerable to an APD reinitiation of atrial fibrillation, or whether both mechanisms are operative\(^6\). There may be multiple mechanisms by which atrial pacing could prevent atrial fibrillation.

**Figure 4** An example of early recurrence of atrial fibrillation during high right atrial pacing. (a) During atrial pacing at 500 ms, atrial fibrillation is reinitiated by an atrial premature depolarization with a coupling interval of 350 ms. (b) During atrial pacing at 300 ms, atrial fibrillation is also reinitiated by an atrial premature depolarization, however, the coupling interval of the atrial premature depolarization is only 220 ms, which is shorter than during atrial pacing at 500 ms.

Eur Heart J, Vol. 21, issue 14, July 2000
fibrillation\textsuperscript{[17]}. Theoretically, atrial pacing can prevent atrial fibrillation either by suppressing the atrial premature depolarization or by modifying the atrial activation pattern and atrial electrical recovery. Atrial pacing at a faster rate can suppress automaticity from ectopic sites and reduce the frequency of atrial premature depolarization\textsuperscript{[17,18]}. Furthermore, pre-excitation of an abnormal atrial substrate, where reentry is initiated by atrial pacing can, prolong the coupling interval of atrial premature depolarization\textsuperscript{[17]}. Both of these mechanisms can reduce the likelihood of an APD and therefore may reduce early reinitiation of atrial fibrillation.

The results of the present study suggest that atrial pacing after defibrillation could suppress early reinitiation of atrial fibrillation by both reducing the numbers of atrial premature depolarization and prolonging their coupling interval. In this study, atrial pacing was not more effective in preventing early reinitiation of atrial fibrillation in patients with bradycardia, as there was no significant difference in the cycle length of the preceding atrial rhythm between patients with or without suppression of early reinitiation of atrial fibrillation by pacing. However, there were also no significant differences in the coupling intervals of atrial premature depolarization and their density during atrial pacing between patients with or without suppression of early reinitiation of atrial fibrillation. This suggests that suppression of atrial premature depolarization and prolongation of their coupling interval by atrial pacing may not be sufficient to prevent early reinitiation of atrial fibrillation. Electrical remodelling of the atrium with shortening and maladaptation of the atrial refractory period may also contribute to the occurrence of early reinitiation of atrial fibrillation after defibrillation\textsuperscript{[19,20]}.

Furthermore, during more rapid atrial pacing, we observed a significant shortening of the coupling interval of atrial premature depolarization. This may offset the benefit of atrial pacing in suppressing atrial premature depolarization and explain why pacing at a higher rate was not more effective in preventing early reinitiation of atrial fibrillation. The mechanism of this phenomenon is unclear. One possible explanation is that rapid atrial pacing triggered spontaneous atrial activity by delayed afterdepolarizations\textsuperscript{[21]}. Another possible explanation is the suppression of atrial premature depolarization with a longer coupling interval by rapid atrial pacing, therefore, only those atrial premature depolarization with a shorter coupling interval was observed.

**Pharmacological therapy for early reinitiation of atrial fibrillation**

Previous studies\textsuperscript{[3,8]} demonstrated that administering antiarrhythmic drugs before cardioversion might prevent early reinitiation of atrial fibrillation. Preliminary clinical data suggested that intravenous flecainide and intravenous sotalol could suppress early reinitiation of atrial fibrillation\textsuperscript{[1,3]}. In the present study, we have
confirmed that intravenous sotalol could prevent early reinitiation of atrial fibrillation in up to 70% of patients with pacing-refractory early reinitiation of atrial fibrillation. However, the optimal antiarrhythmic agents for treating early reinitiation of atrial fibrillation remains unclear and requires future randomized, prospective investigations.

**Study limitations**

Due to the small size of the study population, the effects of different or multiple sites for pacing could not be tested. A recent study by Prakash et al.\[22\] has demonstrated that different or multiple sites of atrial pacing have different effects on atrial electrophysiological properties. Multisite atrial pacing is more likely to improve local excitability by preventing regional atrial conduction delay, and to reduce the inducibility of atrial fibrillation.\[22–24\] The effect of multisite atrial pacing on the prevention of early reinitiation of atrial fibrillation merits further study.

Since only a limited number of atrial sites were recorded and detailed atrial mapping was not performed during the study, the site of origin of the APD could not be determined. Although our observations suggested that sotalol might be effective in preventing early reinitiation of atrial fibrillation, it is impossible to draw firm conclusions because the present study was not randomized had no control group. Nevertheless, before the sotalol infusion, patients with refractory early reinitiation of atrial fibrillation had failed repeated defibrillation with and without atrial pacing. This suggests that the observed effect of sotalol can be attributed to treatment with some confidence.

**Conclusions**

The results of this study suggest that single-site atrial pacing is effective in preventing early reinitiation of atrial fibrillation in 40% of patients with early reinitiation of atrial fibrillation after internal cardioversion and prolonged the period of post cardioversion sinus rhythm in 60% of the remaining cases. These findings have important implications for the use of atrial pacing to prevent early reinitiation of atrial fibrillation after defibrillation when using temporary catheters or implantable atrial defibrillators. In patients with persistent early reinitiation of atrial fibrillation despite atrial pacing, antiarrhythmic therapy may be required to prevent their early reinitiation of atrial fibrillation episodes. The role of combination therapy with atrial pacing and antiarrhythmic therapy in the treatment of early reinitiation of atrial fibrillation need to be explored. Furthermore, multisite atrial pacing might be more effective than single-site atrial pacing in preventing early reinitiation of atrial fibrillation and requires further investigation.

**Table 2 Comparison of patients with or without suppression of early reinitiation of atrial fibrillation by atrial pacing**

<table>
<thead>
<tr>
<th>Variable</th>
<th>ERAF (n=7)</th>
<th>No ERAF (n=5)</th>
<th>P values</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (years)</td>
<td>53 ± 15</td>
<td>49 ± 11</td>
<td>0.64</td>
</tr>
<tr>
<td>Duration of atrial fibrillation (months)</td>
<td>27 ± 22</td>
<td>28 ± 28</td>
<td>0.93</td>
</tr>
<tr>
<td>Left atrial size (cm)</td>
<td>4.4 ± 0.48</td>
<td>4.0 ± 0.29</td>
<td>0.11</td>
</tr>
<tr>
<td>Left ventricular ejection fraction (%)</td>
<td>58 ± 7</td>
<td>55 ± 13</td>
<td>0.65</td>
</tr>
<tr>
<td>Atrial defibrillation threshold (V)</td>
<td>317 ± 69</td>
<td>300 ± 57</td>
<td>0.53</td>
</tr>
<tr>
<td>APD coupling interval (ms)</td>
<td>379 ± 27</td>
<td>380 ± 63</td>
<td>0.97</td>
</tr>
<tr>
<td>Preceding rhythm cycle length (ms)</td>
<td>953 ± 331</td>
<td>849 ± 213</td>
<td>0.52</td>
</tr>
<tr>
<td>Duration of onset of ERAF (s)</td>
<td>11 ± 11</td>
<td>21 ± 27</td>
<td>0.53</td>
</tr>
<tr>
<td>During atrial pacing at 500 ms</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>APDs density (APDs·min⁻¹)</td>
<td>3.5 ± 2.0</td>
<td>3.1 ± 1.4</td>
<td>0.67</td>
</tr>
<tr>
<td>APD coupling interval (ms)</td>
<td>404 ± 30</td>
<td>432 ± 20</td>
<td>0.18</td>
</tr>
</tbody>
</table>

APD=atrial premature depolarization; ERAF=early reinitiation of atrial fibrillation.

References


