Long term management of atrial arrhythmias in young patients with sick sinus syndrome undergoing early operation to correct congenital heart disease

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Aims The objective of our study was to evaluate the clinical outcome of patients with operated congenital heart disease (CHD), post-operative sinus node dysfunction and atrial tachyarrhythmias (AT) who had a new generation of DDDR pacemakers (Model AT501, Medtronic Inc., MN, USA) able to deliver preventive atrial pacing and antitachycardia pacing (ATP) therapies.

Methods and results Fifteen CHD patients (mean age 17 ± 9 years, eight after Mustard operation, five after extracardiac Fontan operation and two after atrial septum repair) received a dual-chamber pacemaker with transvenous (eight patients) or epicardial leads (seven patients). In the year before implantation, all patients had symptomatic AT (palpitations), eight patients required hospitalization and five required electrical cardioversion. Pacing prevention algorithms were enabled in all patients, and ATP therapies in six patients. During a mean follow-up of 30 months (range 24–44), three patients (two Fontan, one Mustard) died of CHF, whereas AT required hospitalization in three patients (two Fontan, one atrial septum repair). Only seven patients had symptomatic AT. One hundred and twenty-five AT episodes were treated by ATP in three patients, with an overall termination efficacy of 43.2%. In one patient, atrial lead noise induced inappropriate AT detection that resulted in ATP delivery. Several AT episodes were not treated owing to their very short duration, atrial undersensing, or 1:1 atrioventricular conduction.

Conclusions Our experience with antitachycardia pacemakers in CHD patients with post-operative sick sinus syndrome after biventricular correction or palliation shows that these devices are safe and that atrial pacing may play a role in AT prevention and treatment.

Introduction

Patients with repaired congenital heart disease (CHD) are prone to sudden death or pump failure.1–5 Frequently, their prognosis may be adversely affected by complications such as loss of sinus rhythm and atrial tachyarrhythmias (AT).6–8 Arrhythmic death and heart failure may be secondary to AT.6–11 In particular, a strong association between AT and congestive heart failure (CHF) has been demonstrated in CHD patients,9 suggesting that AT may worsen CHF or that AT may result from mechanical–electrical interactions in heart failure.

Radiofrequency catheter ablation (RFCA) is now the treatment of choice for post-operative AT. However, despite the high acute success rate of this therapy, AT tends to recur during long-term follow-up, because of the severe and progressively increasing atrial dilatation. Moreover, RFCA is not feasible in patients with single ventricle physiology and an unapproachable right atrium, for instance, in patients who have undergone an extracardiac Fontan operation.

In the past, we proposed the use of permanent overdrive pacing in order to prevent AT recurrence in patients with CHD and post-operative sinus node dysfunction (SND).6,12 a strategy, that in our experience, has yielded a satisfactory success rate. Antitachycardia pacing (ATP) therapies delivered by a pacemaker have already been used for the treatment of supraventricular re-entrant tachycardias13–16 and, very recently, ATP has been applied in the treatment of AT in general, with some promising results.17–23

In the light of all these considerations, we aimed to evaluate the effectiveness of a dual-chamber pacemaker able to deliver overdrive atrial pacing and ATP therapies in the prevention and treatment of AT in patients with CHD and post-operative SND.

KEYWORDS
Congenital heart disease; Mustard; Fontan; Atrial tachyarrhythmia; Pacemaker

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Methods

Recruitment
We recruited consecutive patients with SND, operated CHD, class I or IIa pacemaker indications, and at least two AT episodes during the year before implantation, or at least one symptomatic AT episode requiring either cardioversion or hospitalization within 1 month before implantation, observed at our institution between December 2000 and August 2003. In accordance with our clinical practice, based on previous experience, all patients received a permanent pacemaker with a specific function designed to prevent, detect and, if necessary, treat atrial arrhythmias. For the whole period, we prospectively collected data on the patients’ baseline conditions, the performance of the implanted system, and follow-up outcome, in order to evaluate our practice through a post hoc analysis. All patients—or their legal representatives, when appropriate—gave written informed consent to the anonymous use of health information for scientific research purposes, by signing a form approved by the ethical board responsible.

Our population consisted of 15 patients, whose anatomy, surgical procedures and pacing lead types are described in Table 1. Eight patients were treated with antiarrhythmic drugs. Pre-implantation electrophysiological examination was not performed in any patient. All patients underwent implantation of a dual-chamber pacemaker (Model AT501, Medtronic Inc., MN, USA) with transvenous (eight patients) or epicardial leads (seven patients). The method of transvenous implantation in our institution has already been described by Silvetti and Drago. The atrial transvenous leads used (Model 5076, Medtronic Inc., MN, USA) are steroid-eluting, bipolar, with a helix fixation mechanism (extracardiac Fontan) with intraoperative modified Maze procedure in two.

In three patients, the epicardial implantation was performed at the time of the following surgical procedures: re-Mustard for a stenosis of the superior cava vein baffle in one, and conversion from an atrio-pulmonary Fontan to a total cavopulmonary con-nection (extracardiac Fontan) with intraoperative modified Maze procedure in two.

The atrial transvenous leads used (Model 5076, Medtronic Inc., MN, USA) are steroid-eluting, bipolar, with a helix fixation mechanism, 10 mm tip-to-ring spacing and 6.0 French diameter. The epicardial leads (Model 4968, Medtronic Inc., MN, USA) are steroid-eluting, bipolar, hemispherical platiniAZed porous electrodes with a suture fixation mechanism. The patients’ characteristics are listed in Table 2.

Device characteristics and programming
The Medtronic AT501 is a dual-chamber pacemaker with advanced algorithms designed for rhythm classification, and AT prevention and treatment, and has already been described. Rhythm classification tests conducted on this device have shown 100% sensitivity for AT detection and 97% specificity. According to the AT cycle length (ATCL) and regularity, every AT episode is classified by the device either as a fast and/or irregular tachyarrhythmia, or as a slow and/or regular tachyarrhythmia. The AT detection was programmed in a simplified way that did not take into account all the parameters listed in Table 1. Eight ramp sequences were programmed. The second ramp consists of a programmable drive of atrial pulses with a rate proportional to the current ATCL, a mean coupling interval of 88 ± 6% of ATCL, a mean coupling interval of the first extrastimulus equal to 84 ± 8% of the cycle of previous pulses and the second extrastimulus delayed by 17 ± 5 ms. On an average, 6 ± 3 burst sequences were programmed. The second therapy was always a burst, with a mean coupling interval of 85 ± 8% of ATCL, a mean coupling interval of the first extrastimulus equal to 80 ± 5% of the cycle of previous pulses and the second extrastimulus delayed by 18 ± 4 ms. On an average, 7 ± 3 burst sequences were programmed. The third therapy was always a ramp, with a mean coupling interval of 91 ± 4% of ATCL. On an average, 7 ± 3 ramp sequences were programmed.

Programming of ATP therapies
The device automatically delivers ATP therapies for any episode detected as slow atrial arrhythmia and lasting longer than a programmable ‘time to first therapy’ value. Every atrial arrhythmia episode, regardless of its classification at onset, is continuously monitored and can be treated as soon as it is classified as a slow atrial arrhythmia. The device classifies the effects of ATP therapy as successful if it detects five consecutive beats of sinus rhythm within the first 32 ventricular cycles that follow a previously delivered ATP.

The device enables two types of ATP therapies to be delivered: burst- therapy consists of a programmable drive of atrial pulses with a rate proportional to the current ATCL, followed by two extrastimuli; ramp therapy consists of a decremental drive of a programmable number of pulses, starting at a rate proportional to the current ATCL.

ATP therapies were enabled only in patients who suffered appropriate episodes of arrhythmia, and were programmed to treat atrial arrhythmia episodes longer than 1 min. ATP was tailored to individual patients. The first therapy was always a burst-, with a mean coupling interval of 88 ± 6% of ATCL, a mean coupling interval of the first extrastimulus equal to 84 ± 8% of the cycle of previous pulses and the second extrastimulus delayed by 17 ± 5 ms. On an average, 6 ± 3 burst- sequences were programmed. The second therapy was always a burst-, with a mean coupling interval of 85 ± 8% of ATCL, a mean coupling interval of the first extrastimulus equal to 80 ± 5% of the cycle of previous pulses and the second extrastimulus delayed by 18 ± 4 ms. On an average, 7 ± 3 burst sequences were programmed. The third therapy was always a ramp, with a mean coupling interval of 91 ± 4% of ATCL. On an average, 7 ± 3 ramp sequences were programmed.

Study design and data analysis
Primary endpoints were AT-related hospitalizations, the occurrence of any AT episode, occurrence of symptomatic AT episodes, occurrence of AT episodes treated by ATP therapies, ATP therapy efficacy, and ventricular arrhythmias detected by the device.

Patients were defined as ‘non-responders’ if during follow-up they underwent AT-related hospitalization or complained of symptomatic AT recurrences. The baseline characteristics of responders were compared with those of non-responders in order to pick out the predictors of positive clinical response.

Data on clinical outcomes were obtained during follow-up visits, which were scheduled one, four, and seven months after implantation, and at 6-month intervals thereafter.

For each patient, device diagnostics enabled long-term daily data on AT frequency and duration to be collected, together with information on ATP use and efficacy. ATP efficacy was calculated as the ratio between successfully treated and terminated episodes.

Detailed data on up to 35 newly detected AT episodes were collected from device diagnostics at each follow-up visit. For each episode, data were stored at the onset, detection, and termination of AT, including: time of day and date, 48 atrial and ventricular cycles with marker annotations and cycle lengths plus 4 s atrial electrograms (EGMs) (only at detection or at first ATP, optionally at onset) and, before detection, a flashback memory with duration and type (i.e. paced or sensed) of the last 650 atrial cycles.

All device-detected AT episodes were manually reviewed by studying device-stored atrial EGMs and marker channels, in order to evaluate the appropriateness of detection and termination.

Statistical analysis
Descriptive statistics were reported by using mean and standard deviation for normally distributed continuous variables, or median with 25th-75th interquartile range in the case of skewed distributions. Absolute and relative frequencies were reported for categorical variables.
Table 1  Anatomy, surgery, atrial pacing lead type, number of true AT episodes, noise-induced episodes, and FFRW-induced episodes among device-detected atrial episodes in enrolled patients

<table>
<thead>
<tr>
<th>Patient</th>
<th>Anatomy</th>
<th>Surgery</th>
<th>Lead</th>
<th>Age (years)</th>
<th>FU (months)</th>
<th>Death H</th>
<th>AT episodes</th>
<th>Noise-induced episodes</th>
<th>FFRW-induced episodes</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Left isomerism + CAVSD + AV discordance</td>
<td>Mustard</td>
<td>Endocardial</td>
<td>13</td>
<td>29</td>
<td>Yes</td>
<td>36</td>
<td>35 (19 treated)</td>
<td>1</td>
</tr>
<tr>
<td>2</td>
<td>TGA</td>
<td>Mustard</td>
<td>Endocardial</td>
<td>11</td>
<td>32</td>
<td></td>
<td>1</td>
<td></td>
<td></td>
</tr>
<tr>
<td>3</td>
<td>TGA</td>
<td>Mustard</td>
<td>Endocardial</td>
<td>17</td>
<td>27</td>
<td></td>
<td>1</td>
<td></td>
<td></td>
</tr>
<tr>
<td>4</td>
<td>TGA</td>
<td>Mustard</td>
<td>Epicardial</td>
<td>15</td>
<td>27</td>
<td></td>
<td>1</td>
<td></td>
<td></td>
</tr>
<tr>
<td>5</td>
<td>CAVSD + multiple VSD</td>
<td>Ex. Fontan</td>
<td>Epicardial</td>
<td>13</td>
<td>1</td>
<td>Yes</td>
<td>1</td>
<td></td>
<td></td>
</tr>
<tr>
<td>6</td>
<td>TGA</td>
<td>Mustard</td>
<td>Endocardial</td>
<td>15</td>
<td>16</td>
<td></td>
<td>Yes</td>
<td></td>
<td></td>
</tr>
<tr>
<td>7</td>
<td>UVH</td>
<td>Ex. Fontan</td>
<td>Epicardial</td>
<td>19</td>
<td>5</td>
<td>Yes</td>
<td>Yes</td>
<td>3</td>
<td></td>
</tr>
<tr>
<td>8</td>
<td>TGA + UVH</td>
<td>Ex. Fontan</td>
<td>Epicardial</td>
<td>10</td>
<td>18</td>
<td>Yes</td>
<td>23 (22 treated, and 19 successfully terminated)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>9</td>
<td>Left isomerism + CAVSD</td>
<td>Atrial septal repair</td>
<td>Endocardial</td>
<td>48</td>
<td>13</td>
<td>Yes</td>
<td>5</td>
<td></td>
<td>1</td>
</tr>
<tr>
<td>10</td>
<td>TGA</td>
<td>Mustard</td>
<td>Endocardial</td>
<td>15</td>
<td>12</td>
<td></td>
<td>35</td>
<td></td>
<td></td>
</tr>
<tr>
<td>11</td>
<td>TGA + UVH</td>
<td>Fontan</td>
<td>Epicardial</td>
<td>8</td>
<td>16</td>
<td></td>
<td>175 (86 treated and 19 successfully terminated)</td>
<td>2</td>
<td></td>
</tr>
<tr>
<td>12</td>
<td>TA</td>
<td>Ex. Fontan + Maze</td>
<td>Epicardial</td>
<td>24</td>
<td>12</td>
<td>Yes</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>13</td>
<td>Left isomerism + CA + Dextrocardia + Situs inversus</td>
<td>Atrial septal repair</td>
<td>Epicardial</td>
<td>12</td>
<td>13</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>14</td>
<td>TGA</td>
<td>Mustard</td>
<td>Endocardial</td>
<td>14</td>
<td>15</td>
<td></td>
<td>15</td>
<td></td>
<td></td>
</tr>
<tr>
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<td>TGA</td>
<td>Mustard</td>
<td>Endocardial</td>
<td>15</td>
<td>16</td>
<td></td>
<td>15</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

FU, follow-up (months); H, AT related hospitalizations; CAVSD, complete AV septal defect; VSD, ventricular septal defect; CA, common atrium; TA, tricuspid atresia; TGA, transposition of the great arteries; UVH, univentricular heart; Ex. Fontan, extracardiac Fontan.
Comparison was performed by means of the $\chi^2$ or Fisher’s exact test, as appropriate, for categorical variables, and by $t$-test or Mann–Whitney test, as appropriate, for continuous variables.

A two-tailed probability ($P$) value $< 0.05$ was considered significant.

For all the statistical analyses, the SPSS (SPSS Inc., Chicago, IL, USA) software, version 11.5, was used.

**Results**

Mean follow-up duration was 30 ± 8 months, ranging between 24 and 44.

Device diagnostics detected 10 episodes of non-sustained ventricular tachycardia (VT) in one Mustard patient. In this patient, an electrophysiological examination was performed, which did not show VT inducibility. For this reason, and owing to the shortness of the VT episodes, upgrading of the system to implantable cardioverter defibrillator (ICD) was not considered. Sotalol proved effective in eliminating VT recurrences.

Three patients died of CHF. All three died in our institution. No autopsy was performed in these patients.

After implantation, five hospitalizations were necessary in four patients (26.7%). The causes of hospitalization were atrial flutter, atrial flutter associated with CHF, sinus tachycardia associated with fast ventricular rate and CHF, and atrial fibrillation. The patient first hospitalized for atrial flutter was again hospitalized for CHF. In two patients, atrial cardioversion was also performed during their hospital stay.

Of the five Fontan patients, two died of CHF, and another two were hospitalized for AT; only one survivor was free from cardiac-related hospitalizations. Of the eight Mustard patients, one died of CHF, while none of the others was hospitalized.

In the first 12 months of follow-up, 3/15 (20%) patients were hospitalized for AT [$P = 0.12$, compared with 8/15 (53%) who were hospitalized in the 12 months before pacemaker implantation].

During post-implantation follow-up, 7/15 (46.7%) patients had symptomatic AT [$P = 0.002$, compared with 15/15 (100%) who had AT-related symptoms in the 12 months before pacemaker implantation].

Using AT-related hospitalization and symptomatic AT recurrence as criteria for negative clinical outcomes, patients were divided into eight non-responders and seven responders. No significant differences were found among the baseline characteristics of the two groups; nevertheless, the non-responders were older ($20 \pm 12$ years vs. $14 \pm 4$ years; $P = 0.13$) and had a higher incidence of pre-implantation hospitalizations ($75\%$ vs. $29\%$, $P = 0.13$), cardioversions ($50\%$ vs. $14\%$, $P = 0.28$), and AT-related symptoms ($100\%$ vs. $57\%$, $P = 0.08$).

**Device-collected AT episodes**

Device diagnostics detected 331 AT episodes in eight patients. In seven of these eight patients, 317 AT detections gave EGM information and were analysed in order to study detection appropriateness (Table 1). Overall inappropriate detections occurred in five patients owing to atrial lead noise and/or far field R wave (FFRW) oversensing. In two patients, only inappropriate AT detections were registered due to atrial lead noise. In five patients with appropriate AT detection, AT episodes were always self-terminating, in particular, in three patients AT duration was always shorter than 12 h, whereas, two patients of the Fontan group were characterized by persistent AT episodes lasting longer than 7 days and were hospitalized for AT treatment.

**Treatment of AT episodes**

One hundred and twenty-five appropriately detected AT episodes were treated in three patients (Table 1). Overall ATP efficacy was 54/125 (43.2%), while ATP efficacy on individual patients was 16/17 (94.1%), 19/22 (86.4%), and 19/86 (22.1%), respectively.
In one patient, 19 episodes of AT induced by atrial lead noise were treated. No adverse event was associated with ATP therapy. Very short AT duration or atrial undersensing were the causes of the untreated AT episodes.

In two patients, AT episodes conducted 1:1 via the AV node were inappropriately classified as VT by the device.

Electrical data

On implantation, the mean atrial threshold was 0.9 ± 0.8 V at 0.5 ms, the mean P-wave amplitude was 3.7 ± 2.3 mV, and the mean atrial impedance was 942 ± 441 Ω. At the last follow-up examination, the mean atrial threshold was 0.2 ± 0.1 ms at 1.7 ± 0.3 V, mean P-wave amplitude was 4.0 ± 2.2 mV, and mean atrial impedance was 820 ± 365 Ω. During the follow-up, no adjustment of pacing or sensing was necessary.

Discussion

About 85% of the children born with CHD survive to adulthood, thanks to surgical repair or palliative operations. An indication for permanent pacing in these patients may directly result from surgery or may arise later in life. The choice of the best device depends on the patient’s characteristics: dual-chamber pacemakers are appropriate for AV block, for example, after repair of septal defects and Tetralogy of Fallot; biventricular pacemakers may provide haemodynamic optimization in patients with CHF, pacemakers able to deliver ATP may suit patients suffering from AT, and finally, ICDs may be indicated in patients at risk of ventricular arrhythmias.

In patients with surgically corrected or palliated CHD, permanent pacing is also often indicated, owing to the presence of bradycardia due to SND, which increases proportionally with the duration of follow-up. In this patient population, ATs very often occur and RFCA has therefore been proposed. However, while the acute success rate of this therapy is high, AT tends to recur during long-term follow-up. This phenomenon, which has also been observed in our experience, was the reason why ablation procedures were not considered in any of our patients in whom an endocardial approach was deemed possible.

In 1997, our group studied the effectiveness of permanent overdrive pacing in the prevention of AT unresponsive to conventional treatment in 18 children with CHD and post-operative sick sinus syndrome. The pacemaker was programmed at a lower pacing rate: 20% faster than the spontaneous mean daily rate, previously determined by means of 24-h Holter monitoring. One patient died suddenly 10 months after pacemaker implantation. At the end of the follow-up, 15 patients (83%) were arrhythmia-free and only two were still on antiarrhythmic drugs. These important results suggest that AV synchrony and permanent atrial overdrive pacing could be useful tools in the management of patients with atrial re-entrant tachycardia following repair of CHD. During the study observation period, no changes were made in the antiarrhythmic drug regimens that patients were taking before pacemaker implantation. Therefore, our results were presumably uninfluenced by pharmacological therapy, apart from a synergistic action between antiarrhythmic drugs and atrial pacing algorithms.

In this particular setting, a pacemaker able to deliver preventive overdrive atrial pacing and ATP therapies (AT501, Medtronic Inc., MN, USA) has recently been introduced. In 2003, Stephenson et al. reported a multi-centre experience in which the incidence of atrial and ventricular arrhythmias, the appropriateness of device detection, and ATP efficacy were studied in patients with operated CHD and post-operative AT who had undergone AT501 implantation. The results showed that almost half of the enrolled patients experienced AT during follow-up, and that inappropriate AT detection was mainly due to noise, which is consistent with myopotential oversensing and/or lead integrity, or else to sinus tachycardia with FFRW oversensing.

In 2004, we reported successful DDDRPM pacemaker treatment in a 13-year-old boy with tachy-brady syndrome and CHF after a Mustard procedure for a very complex CHD. On the basis of this successful preliminary experience, we began to implant DDDRPM pacemakers in every patient with operated complex CHD, SND, and AT. As far as we know, this is the first single-centre experience reported.

Unlike the study by Stephenson et al., our study evaluated the clinical outcome of CHD patients who had received an AT501 pacemaker. Our data show that Fontan patients seem to have a worse clinical outcome. Indeed, only one Fontan patient of five neither underwent hospitalization nor had AT recurrences. A possible explanation for this result is that Fontan patients had more scars (surgical: atriotomy and linear lesions caused by modified Maze procedure, spontaneous due to the extreme dilatation of the right atrium) than others, and these surgically acquired features could result in AT circuits that are less amenable to ATP termination. Moreover, all Fontan patients in our population had epicardial leads, which might have conditioned the ability of AT501 to treat ATs. This experience prompts us to think that Fontan patients with AT and SND need a hybrid therapeutic approach, consisting of a switch to extracardiac Fontan, modified Maze procedure, DDDRPM, antiarrhythmic therapy; in the event of failure of all these treatments, heart transplantation should be considered.

Another finding in our study was that AT-related symptoms decreased after AT501 implantation. When responders and non-responders were differentiated on the basis of post-implantation AT hospitalizations or symptomatic episodes, we were not able to discern any statistically significant difference in baseline characteristics that could predict the individual response to the therapy applied; this was probably due to the extremely small size of our population. Nevertheless, in view of the extreme rarity of the condition examined, and the probable difficulty in recruiting larger single-centre cohorts, the observed trends toward higher average age and incidence of pre-implantation AT hospitalizations, cardioversions, and AT-related symptoms may be tentatively interpreted as useful indications for this therapy. Indeed, such differences could suggest reduced ATP effectiveness in young adult CHD patients with a history of more severe AT.

It could be interesting to evaluate the impact of DDD pacemaker (PM) therapy alone in reducing AT recurrence and symptoms in a multi-center randomized study comparing the use of a DDD PM and a DDDRPM PM in these particular patients.

In our study population, AT was less frequent than in the multi-centre pacemaker (PM) study reported by...
Stephenson et al., as AT episodes recurred and were appropriately detected in five patients (33%). In contrast, ATP efficacy was lower (43 vs. 54%) and no difference in ATP efficacy was observed between Mustard patients and the others, as reported by Stephenson.

In our patient population, ATP efficacy and inappropriate AT detection due to myopotential oversensing and/or lead integrity (five out of seven patients with AT detection) were similar to Stephenson’s data, but FFRW oversensing was very rare. Moreover, in our study too, no adverse events were associated with ATP therapy or false detection.

Device diagnostics should also be considered when choosing the best device. In our patient sample, device memory collected detailed information and EGM recordings for several ventricular tachyarrhythmia episodes in one patient. In addition, AT episodes conducted 1:1 via the AV node were detected as VT by the device in two patients. The AT501 device does not detect 1:1 conducted AT. Therefore, in these particular patients, the use of beta-blocking agents may be advisable even in the case of appropriate arrhythmia detection and prompt ATP therapy. The implantation of a dual-chamber defibrillator with atrial prevention and ATP therapies and ventricular defibrillation could be considered as a valid therapeutic option in CHD patients with AT and VT.

Study limitations

Our conclusions should be limited to the population we studied. Our findings are based on a retrospective analysis of prospectively collected observational data. Assessment of the clinical impact of preventive and ATP therapies would require a randomized trial that cannot be undertaken in this patient population, as the sample sizes needed for such a study would not be obtainable. No post-mortem device interrogation was performed.

Conclusions

Our long-term experience of the implantation of pacemakers with AT prevention algorithms and atrial ATP therapies in patients with CHD, with biventricular correction or palliation and post-operative sick sinus syndrome showed that these devices are safe. Our data also suggest that AV synchrony and/or atrial pacing algorithms may play a role in AT prevention, above all in patients in whom an endocardial approach is possible. However, in this particular group of patients, a hybrid medical and surgical approach is needed in order to avoid AT recurrence and sudden death.

References


