Single A-V Lead Pacing in Dilated Cardiomyopathy

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Summary

Single Lead VDD pacing systems with back-up OLBI atrial stimulation capability are routinely implanted in our center in patients (pts) with symptomatic AV block II° and III° and uncompromised sinus node function. Supported by the satisfactory results obtained in 61 pts, we decided to verify if the same pacing approach can be also used in pts with dilated cardiomyopathy and acceptable chronotropic competence. In 5 pts, all male, with mean age of 65.9 years, all affected by LV severe dilated cardiomyopathy, with mean ejection fraction of 26% and normally sized RV, were implanted with a single A-V lead VDD/DDD pacing system (BIOTRONIK mod. EIKOS SLD) allowing OLBI atrial stimulation. Fluoroscopic observation showed that atrial dipole was positioned in high atrium in 2 pts and in mid atrium in 3 pts. Inconstant atrial pacing was achieved in 2/5 pts with OLBI pulse amplitudes over 3.8 V during the entire year of follow-up. In remaining 3 pts, it was impossible to induce atrial pacing even at the maximum OLBI output (4.8 V), both at implant and follow-up. VDD pacing, with atrial synchronization >98%, was constantly maintained in all pts. The mean value of minimum P-wave sensing at one year was 0.7 mV. Single-lead DDD pacing, even using atrial OLBI stimulation, can not be performed in pts with dilated cardiomyopathy. In these pts atrial myocardium seems to have a reduced excitability and, to be depolarized, it requires a pacing pulse with an high specific energy density, like that delivered by most conventional contact electrodes.

Key Words

Dilated cardiomyopathy, single lead DDD pacing, OLBI atrial stimulation, single lead VDD pacing

Introduction
discharge and at 1, 3 and 6 months follow-up, when DDD-OLBI pacing was temporarily programmed. Two leads were implanted by cephalic (Pt. 1 and Pt. 3) and two by subclavian access (Pt. 2 and Pt. 4).

Results

Data collected at implant were: Atrial minimum P-wave amplitude 1.15 mV (range 0.4-2.2 mV); R-wave amplitude 15.8 mV (range 9.8-22 mV); ventricular pacing threshold 0.72 V (range 0.6-1.0 V) and ventricular pacing impedance 585 $\Omega$ (range 420-778 $\Omega$).

During the entire follow-up the minimum P-wave amplitude, detected in the worst condition (expiration, lateral decubitus), showed a substantial stability in pt 2, pt 3 and pt 5, an unexpected improvement at the 3rd and 6th month in pt 1, while in pt 4 the signal became gradually worse. The course of P-wave amplitude in the four patients is shown in Figure 3.

Atrial capture with OLBI pacing thresholds included within 3.8 and 4.5 V were showed by pt 1 and pt 2 only. Pt 3, pt 4 and pt 5 did not show any atrial capture, even not with OLBI pulse amplitude of 4.8 V (see Figure 4).

In addition, the atrial capture in pt 1 and pt 2 was never constant, only pt 1 showed a percentage of capture included between 50 and 95% during the first two follow-ups, but in the followings the occurrence of capture dropped below the 50% (see Table 2). At discharge and at the 3rd month follow-ups, the position of atrial ring electrodes was assessed by X-ray fluoroscopy. At discharge, electrode pairs were posi-

Materials and Methods

In 5 pts, all male, with mean age of 65.9 years (range 43-75), all affected by advanced and symptomatic AVB with uncompromised sinus node function, were implanted with a VDD/DDD cardiac pulse generator (BIOTRONIK mod. EIKOS SLD), allowing unsupervised OLBI atrial stimulation, and a single AV lead (BIOTRONIK model SLD 60/13) with 13 cm A-V inter-electrode distance. In all pts the A-V conduction pathology was associated to a severe left ventricular dilated cardiomyopathy, normally sized right chambers, with mean ejection fraction of 26% (range 23-30%). In Table 1, all data of the echocardiographic and Doppler analysis of left ventricular function of each pt are reported, and in Figures 1 and 2, the typical four chambers and Doppler frames common to all pts are depicted. The lead implantation was performed in according to the standard procedure for single lead VDD systems and the positioning of the atrial dipole was based exclusively on P-wave optimal amplitude and stability. Atrial pacing was only performed during follow-up at:

<table>
<thead>
<tr>
<th>Pt.1</th>
<th>Pt.2</th>
<th>Pt.3</th>
<th>Pt.4</th>
<th>Pt.5</th>
</tr>
</thead>
<tbody>
<tr>
<td>LV diastolic diam. (mm)</td>
<td>85</td>
<td>90</td>
<td>85</td>
<td>73</td>
</tr>
<tr>
<td>LV systolic diam. (mm)</td>
<td>76</td>
<td>78</td>
<td>75</td>
<td>62</td>
</tr>
<tr>
<td>shortening (%)</td>
<td>11</td>
<td>13</td>
<td>12</td>
<td>15</td>
</tr>
<tr>
<td>ejection fraction (%)</td>
<td>25</td>
<td>25</td>
<td>25</td>
<td>30</td>
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Table 1. Echocardiographic and Doppler analysis of LV function of the five pts in the study.
tioned in mid atrium in 2 pts and in mid-low atrium in 3 pts, all in quite good proximity of atrial wall. At the 3rd month the situation was substantially unchanged. At least two 24 h ECG Holter monitoring were performed in each pt during the six months follow-up. All recordings confirmed a correct and reliable pacing in VDD mode, with the same sporadic loss of AV synchrony shown by pts without dilated cardiomyopathy (mean synchronization: 97.3%).

Discussion

Definitive conclusions can not be achieved since the limited population of patients enrolled in this study, but the substantially equivalent results assessed in all subjects allow to suggest a preliminary guideline.

Single-lead DDD pacing seems not to be effective in those patients affected by severe dilated cardiomyopathy, even when the OLBI atrial pacing method, the most performed non-contact stimulation approach available today, is used.

In those patients the atrial myocardium shows a reduced excitability and it needs pacing pulses with a highest specific energy density to be stimulated, like those generated by contact electrodes. While single-lead VDD pacing is effective and safe, when dilated pathology occurs in left side only and sino-atrial function remains unaltered.

References


Table 2. Constancy of atrial capture during follow-up. (N.C. = no capture).

<table>
<thead>
<tr>
<th></th>
<th>7 days</th>
<th>1 month</th>
<th>3 months</th>
<th>6 months</th>
</tr>
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<tbody>
<tr>
<td>Pt 1</td>
<td>50-95%</td>
<td>50-95%</td>
<td>&lt;50%</td>
<td>&lt;50%</td>
</tr>
<tr>
<td>Pt 2</td>
<td>&lt;50%</td>
<td>&lt;50%</td>
<td>&lt;50%</td>
<td>&lt;50%</td>
</tr>
<tr>
<td>Pt 3</td>
<td>N.C.</td>
<td>N.C.</td>
<td>N.C.</td>
<td>N.C.</td>
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<tr>
<td>Pt 4</td>
<td>N.C.</td>
<td>N.C.</td>
<td>N.C.</td>
<td>N.C.</td>
</tr>
<tr>
<td>Pt 5</td>
<td>N.C.</td>
<td>N.C.</td>
<td>N.C.</td>
<td>N.C.</td>
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Figure 3. Mean value of minimum P wave amplitude during follow-up of the four patients.

Figure 4. OLBI atrial pacing threshold during follow-up of the four patient.