Comparison of Mid-term Clinical Experience with Steroid-Eluting Active and Passive Fixation Ventricular Electrodes in Children

NACI CEVIZ, ALPAY ÇELIKER, OSMAN KÜÇÜKOSMANOĞLU, DURSUN ALEHAN, AYHAN KILIÇ, ABDURRAHMAN ÜNER, and SENCAN ÖZME

From the Department of Pediatric Cardiology, Hacettepe University Medical School, Ankara, Turkey

CEVIZ, N., ET AL: Comparison of Mid-term Clinical Experience with Steroid-Eluting Active and Passive Fixation Ventricular Electrodes in Children. Although active fixation ventricular leads seem to have advantages over passive fixation leads, this study compares the follow-up results of active and passive fixation leads in children. We evaluated the implantation and follow-up data of 41 children with active (Accufix II DEC, group 1) (n = 20) or passive (Membrane E, group 2) (n = 21) fixation, steroid-eluting ventricular leads. All but one of the patients in group 1 completed the 12-month follow-up. The mean follow-up period in group 2 was 10.4 ± 2.9 months (range 3–12 months, median 12 months). In both groups the mean pacing threshold was measured as 0.51 ± 0.09 V versus 0.48 ± 0.15 V (P > 0.05) at 0.5-ms pulse width, mean R wave amplitude as 9.9 ± 2.5 mV versus 9.4 ± 3.2 mV (P > 0.05), and mean impedance as 557 ± 92 Ω versus 664 ± 160 Ω (P < 0.05), respectively, at implantation. After the first week of pacing, mean threshold values in group 1 were significantly lower than those of group 2 (P < 0.01 and P < 0.05, respectively). During the follow-up period, lead impedance measurements did not show a significant difference between the two groups. In one patient from group 1, the lead (by unscrewing) was removed easily because of pacemaker pocket infection. No lead dislodgement or helix deformation occurred in group 1. Nevertheless, in one patient from group 2, the lead was extracted at 4-month postimplantation because of lead displacement. We conclude that the steroid-eluting active fixation lead (Accufix II DEC) have advantages of easier implantation and lower acute and chronic stimulation thresholds compared to the passive fixation lead (Membrane E). Therefore, Accufix II DEC is superior to Membrane E, and it is a better first choice in children with an implanted single chamber ventricular pacemaker. (PACE 2000; 23:1245–1249)

steroid elution, active fixation lead, passive fixation lead, children

Introduction

The chronic performance of permanent pacing leads is critically dependent on stable positioning of the electrode. Although early pacing leads were associated with an unacceptably high risk of dislodgement, the development of active and passive fixation mechanisms has dramatically reduced the need for lead repositioning.1 Additionally, development of steroid elution ensured minimal change in stimulation threshold from implantation to a follow-up period of several years.1,2 Pacemaker leads that combine active or passive fixation with steroid elution have become available. Although active fixation ventricular electrodes seem to have advantages over passive fixation electrodes, we are unaware of any study comparing the acute and chronic experiences with active and passive fixation ventricular electrodes in children. We report our midterm experiences with two types of steroid-eluting endocardial electrodes of which one had active and the other passive fixation mechanism.

Patients and Methods

Patients

Forty-eight patients, in whom permanent endocardial pacemakers with active (group 1) or passive (group 2) fixation steroid-eluting ventricular leads were implanted between 1994 and 1997, are evaluated. Accufix II DEC (Telecommuniecs Pacing Systems, Inc. Englewood, CO, USA) leads were used in group I patients and Membrane E (Pacsetter AB, Järffä, Sweden) in group 2 patients. In both groups, patients with atrial (n = 2) electrodes and patients who had < 6-month follow-up periods (n = 5) were excluded. Twenty patients with active fixation and 21 patients with passive fixation steroid-eluting ventricular leads were included in the study.
The group 1 patients had a mean age of 10.6 ± 4.5 years (range 2–18 years, median 10 years) at the time of implantation. Twelve of the 20 group 1 patients were men. The group 2 patients had a mean age of 7.7 ± 4.8 years (range 1–18 years, median 7 years). Fifteen of the 21 group 2 patients were men.

Electrodes
Accufix II DEC (model 033–212) is a ventricular active fixation, bipolar, endocardial pacing lead with 55D polyurethane inner and outer insulation, an IS-1 connector, and a drug-eluting collar. The lead is a porous platinum and/or iridium electrode with a distal surface area of 6 mm². The distal tip diameter is 2.6 mm, and has an electrically inert active fixation screw with a helix penetration of 1.7 mm. The screw is extended and retracted by rotating a fixation stylet. The silicon rubber drug-eluting collar, impregnated with approximately 0.7 mg of the steroid dexamethasone sodium phosphate, is situated adjacent to the distal electrode.³

Membrane E (1400 T or 1450 T models) is a ventricular passive fixation, bipolar, straight, endocardial pacing lead with a silicone rubber outer insulation and an IS-1 UNI connector. The electrode tip is made of titanium nitride on titanium covered by a copolymer membrane that is used as a steroid (13 μg of dexamethasone) carrier. The distal tip diameter and surface area are 3 mm² and 3.5 mm², respectively. The passive fixation mechanism comprises four silicone tines. This electrode was used with threshold tracking pacemakers that need membrane electrodes to evaluate the presence of evoked response signals.⁴

Implantation Procedure
The leads were implanted in the ventricle under ketamine anesthesia via percutaneous puncture of the subclavian vein, and Meta III (Telecommunications PACing Systems, Inc.) pacemakers in group 1 and Microny SR+ or Regency SR+ (Pacesetter AB) pacemakers in group 2 were placed in the subpectoral area. The voltage stimulation threshold at 0.5-ms pulse width, pacing impedance, and R wave amplitude were assessed at the time of implantation. Measurements were done with a pacing system analyzer (PSA); Dual Chamber PSA, model 2401 (manufactured for Telecommunications PACing Systems Inc.) for active fixation leads and Dual Chamber PSA, model 3100 (SEAMED Corporation, Redmond, WA, USA) for the passive fixation leads. Following satisfactory testing, the leads were implanted in the optimal place that the lowest threshold values were measured. In group 2, evoked response and polarization signal measurements were done (APS-2 programmer, Pacesetter) in the catheterization room, in addition to threshold measurement. The electrodes were implanted into the ventricular site at which a sufficient evoked response signal for autodaptic function and an acceptable threshold value were measured.

Patients were discharged between the fifth and seventh day of implantation.

Follow-Up
Follow-up testing was performed at the seventh day, first month, third month, sixth month, and every six months thereafter. Evaluation included routine clinical examination, electrocardiogram, chest X ray, and a full analysis of the pacing system using the specific analyzers provided by the manufacturers (Telecommunications PACing System Analyzer, model 5603 or 9602 for active fixation electrodes, and Pacesetter, APS-2 programmer for passive fixation electrodes). Each patient also underwent 24-hour ambulatory electrocardiographic monitoring (Holter), and if the patient was suitable, treadmill testing was performed when needed.

Pacing thresholds were determined in volts at 0.5-ms pulse width in group 1 and 0.49 ms in group 2. The lead impedance was measured at output settings of 5 V and 0.5 ms. Only in a few patients could R wave amplitude be measured in follow-up visits, because most of the patients were completely pacemaker dependent and had slow ventricular escape rates.

Statistical Analysis
Patients with an active fixation lead completed the 24-month follow-up period, however, only eight of the patients with a passive fixation electrode completed the 18-month follow-up period. Therefore, only the results obtained at the end of the first year were compared for each group. Data are expressed as mean ± SD. A t-test was used to compare the threshold and impedance data of group 1 and group 2 for each follow-up, and one-way analysis of variance was used to compare data over time for each group. A P value < 0.05 was considered significant.

Results
Table I shows the indications for permanent pacemaker implantation and the clinical features of the patients in the study groups.

All but one of the patients in group 1 completed the 12-month follow-up period. The remaining patient developed pacemaker pocket infection at month 9 of implantation and the electrode was extracted. In one patient with a passive fixation electrode, the lead was extracted due to electrode dislodgement at month 4 of implantation. Hence, the mean follow-up period in group 2
was 10.4 ± 2.9 months (range 3–12 months, median 12 months).

In group 1 and group 2, pacing threshold values (0.51 ± 0.09 V vs 0.48 ± 0.15 V, respectively, P > 0.05), sensing characteristics (R wave) (9.9 ± 2.5 mV vs 9.4 ± 3.2 mV, respectively, P > 0.05) and impedance values (557 ± 92 Ω vs 664 ± 160 Ω, respectively, P < 0.05) were good at implantation.

In both groups, pacing thresholds increased in the acute period, and reached maximum values at the first month after implantation. Mean pacing threshold at the first month versus that at implantation was not significantly different in group 1 (0.70 ± 0.45 V vs 0.51 ± 0.09 V, P > 0.05). However, in group 2, there was a significant increase in mean pacing threshold (1.09 ± 0.68 V vs 0.48 ± 0.15 V, P < 0.001) in the same period. A slight decrease was observed in the following evaluations in both groups (Fig. 1). In all follow-up measurements, mean pacing threshold values of group 2 were significantly lower than the values of group 1 (P < 0.01 and P < 0.05, respectively) (Table II).

During the midterm follow-up, in the majority of patients in group 1 (18/20 patients 90%), ventricular pacing could be performed using a pulse amplitude of 2.5 V at a 0.5-ms pulse width while maintaining at least a two-fold voltage threshold safety margin. Patients in group 2 had a pulse generator with a threshold tracking feature that ad-

### Table I.

<table>
<thead>
<tr>
<th>Patient Clinical Characteristics</th>
<th>Group 1 (Accufix II DEC) (n = 20)</th>
<th>Group 2 (Membrane E) (n = 21)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (years) (mean ± SD)</td>
<td>10.6 ± 4.5</td>
<td>7.7 ± 4.8</td>
</tr>
<tr>
<td>Rhythm diagnosis</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Congenital complete heart block (n)</td>
<td>5</td>
<td>7</td>
</tr>
<tr>
<td>Surgical complete heart block (n)</td>
<td>12</td>
<td>12</td>
</tr>
<tr>
<td>Sick sinus syndrome (n)*</td>
<td>2</td>
<td>2</td>
</tr>
<tr>
<td>Acquired complete heart block (n)</td>
<td>1</td>
<td>0</td>
</tr>
<tr>
<td>Pacemaker mode (n)</td>
<td>DDD</td>
<td>—</td>
</tr>
<tr>
<td></td>
<td>VViR</td>
<td>16</td>
</tr>
<tr>
<td></td>
<td>Implantation site (anatomic RV/anatomic LV)</td>
<td>17/3</td>
</tr>
</tbody>
</table>

*Patients with ventricular lead implantation; RV = right ventricle; LV = left ventricle.

### Table II.

<table>
<thead>
<tr>
<th>Electrode</th>
<th>Implantation</th>
<th>1st Week</th>
<th>1st Month</th>
<th>3rd Month</th>
<th>6th Month</th>
<th>12th Month</th>
</tr>
</thead>
<tbody>
<tr>
<td>Accufix II DEC</td>
<td>0.51 ± 0.09</td>
<td>0.51 ± 0.20</td>
<td>0.70 ± 0.45</td>
<td>0.67 ± 0.42</td>
<td>0.67 ± 0.47</td>
<td>0.66 ± 0.46</td>
</tr>
<tr>
<td></td>
<td>(20)*</td>
<td>(20)</td>
<td>(20)</td>
<td>(20)</td>
<td>(20)</td>
<td>(19)</td>
</tr>
<tr>
<td>Membrane E</td>
<td>0.48 ± 0.15</td>
<td>0.50 ± 0.23</td>
<td>1.09 ± 0.68</td>
<td>0.98 ± 0.61</td>
<td>0.94 ± 0.5</td>
<td>0.93 ± 0.54</td>
</tr>
<tr>
<td></td>
<td>(21)</td>
<td>(21)</td>
<td>(21)</td>
<td>(21)</td>
<td>(20)</td>
<td>(16)</td>
</tr>
<tr>
<td>P</td>
<td>&lt; 0.05</td>
<td>&gt; 0.05</td>
<td>&lt; 0.05</td>
<td>&lt; 0.05</td>
<td>&lt; 0.01</td>
<td>&lt; 0.05</td>
</tr>
</tbody>
</table>

* Values in parenthesis reflect the number of observations.

Figure 1. Mean pacing threshold values in group 1 (Accufix II DEC) and group 2 (Membrane E) at implantation and during follow-up. Imp = implantation; W = week; M = month.
justs the pulse amplitude automatically by measuring the pacing threshold every 8 hours or after two consecutive losses of capture, and adding an additional 0.3-V “working margin” for additional safety. During the same period, the evoked response signal measurements were sufficient for autocalibration function in all but three patients.

Mean pacing impedance did not differ significantly throughout the follow-up (P > 0.05 for both electrodes) in both groups. However, in group 2, mean pacing impedance decreased at week 1 of implantation. In the following period, pacing impedance increased and reached a maximum level at month 3, and a subsequent slight decrease was observed (Table III, Fig. 2). After the first week of implantation, mean pacing impedance values did not differ significantly between the two groups.

In both groups, no patient developed syncope or presyncope. Perforation and exit block were not observed. One patient with an active fixation lead developed pacemaker pocket infection, and the pulse generator and the lead (by unscrewing) were removed easily. Among group 1 patients, no lead dislodgement or helix deformation occurred. Nevertheless, in one (4.7%) patient with a passive fixation lead, the lead was extracted due to lead displacement, and an active fixation one was implanted. In both groups, no sensing or capture problem was observed during follow-up.

**Discussion**

Although it has been suggested that active fixation endocardial leads offer some advantages over passive fixation leads in pediatric patients, this has not been demonstrated by a comparative study between two groups with active and passive fixation electrodes. Our study enables us to see the positive impact of an active fixation mechanism on the implantation site and threshold values.

Scar tissue formation due to cardiac surgery and anatomic variations due to congenital heart disease may limit available sites for implantation of passive fixation leads. We were able to implant Accufix II DEC into the anatomic left ventricle in three patients with congenitally corrected transposition of the great arteries. This does not seem possible with passive fixation electrodes.

Although dislodgement may occur with active and passive fixation leads, current data show that active fixation reduces the incidence of lead dislodgement. Lead migration did not occur in group 1. Whereas, in 1 (4.7%) of 21 patients of group 2, the lead was changed to an active fixation electrode due to dislodgement at month 4 of implantation.

Lower stimulation threshold would allow confident use of lower amplitude outputs, and thus would increase the pacemaker longevity. This is a clear advantage in children. New developments in lead technology provided marked improvements in chronic stimulation thresholds compared with their predecessors. The rise in stimulation threshold that normally occurs after lead implantation is a direct result of inflammation at the electrode-tissue interface on which steroid elution has a revolutionary role. Among limited reports about steroid-eluting active fixation ven-

---

**Table III.**

<table>
<thead>
<tr>
<th>Electrode</th>
<th>Implantation</th>
<th>1st Week</th>
<th>1st Month</th>
<th>3rd Month</th>
<th>6th Month</th>
<th>12th Month</th>
</tr>
</thead>
<tbody>
<tr>
<td>Accufix II DEC</td>
<td>557 ± 92</td>
<td>558 ± 83</td>
<td>614 ± 124</td>
<td>605 ± 105</td>
<td>601 ± 93</td>
<td>570 ± 104</td>
</tr>
<tr>
<td></td>
<td>(20)</td>
<td>(20)</td>
<td>(20)</td>
<td>(20)</td>
<td>(20)</td>
<td>(19)</td>
</tr>
<tr>
<td>Membrane E</td>
<td>664 ± 160</td>
<td>588 ± 144</td>
<td>591 ± 104</td>
<td>677 ± 127</td>
<td>664 ± 121</td>
<td>640 ± 84</td>
</tr>
<tr>
<td></td>
<td>(21)</td>
<td>(21)</td>
<td>(21)</td>
<td>(21)</td>
<td>(20)</td>
<td>(16)</td>
</tr>
<tr>
<td>P</td>
<td>&lt; 0.05</td>
<td>&gt; 0.05</td>
<td>&gt; 0.05</td>
<td>&gt; 0.05</td>
<td>&gt; 0.05</td>
<td>0.05</td>
</tr>
</tbody>
</table>

*Values in parenthesis reflect the number of observations.*

---

*Figure 2. Mean impedances in group 1 (Accufix II DEC) and group 2 (Membrane E) over time. Imp = implantation; W = week; M = month.*
tricular electrodes, our previous report was the sole study performed in children. Although they were low when compared to group 1 (patients with an active fixation lead), the mean stimulation thresholds in group 2 (patients with a passive fixation lead) were higher at follow-up measurements, and the difference was significant after the first week of implantation (P < 0.01 and P < 0.05, respectively). However, the passive fixation electrode used in our study was specially produced for threshold tracking pacemakers, and we are not sure if this special configuration is the reason for this relatively high stimulation threshold. Despite the higher threshold values, with the use of threshold tracking pacemakers, energy consumption can be decreased with Membrane E lead.

Because of different indications (increased thresholds, muscle stimulation, lead fracture, lead dislodgement, infection), lead repositioning or explantation may be required. The passive fixation devices are rapidly covered by fibrous tissue, making later removal of the lead by simple traction difficult or impossible in as short a time as 3–6 months. However, active fixation endocardial leads can be safely repositioned or explanted. Although it cannot be generalized to all patients who need lead explantation, in one of our cases with Accufix II DEC, the lead was easily removed by unscrewing at 9-month postimplantation. None of the patients in group 2 required lead removal. We do not know what surprises are awaiting us. However, new lead extraction techniques make extraction of passive fixation leads as successful as active fixation electrodes. Data from the Cook Pacemaker Corporation multicenter lead extraction registry indicates high complete extraction rates for active (91.9%) and passive (88.8%) fixation leads.

In conclusion, our data show that a steroid-eluting active fixation electrode (Accufix II DEC) has some advantages (easier implantation, lower acute and chronic stimulation thresholds) over the passive fixation electrode (Membrane E). Therefore, Accufix II DEC is superior to Membrane E, and it is a better first choice in children with implanted single chamber ventricular pacemakers.

References
This document is a scanned copy of a printed document. No warranty is given about the accuracy of the copy. Users should refer to the original published version of the material.