Permanent Epicardial Pacing in Pediatric Patients
Seventeen Years of Experience and 1200 Outpatient Visits

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**Background**—The purpose of this study was to evaluate the long-term outcome of all pediatric epicardial pacing leads.

**Methods and Results**—All epicardial leads and 1239 outpatient visits between January 1, 1983, and June 30, 2000, were retrospectively reviewed. Pacing and sensing thresholds were reviewed at implant, at 1 month, and at subsequent 6-month intervals. Lead failure was defined as the need for replacement or abandonment due to pacing or sensing problems, lead fracture, or phrenic/muscle stimulation. A total of 123 patients underwent 207 epicardial lead (60 atrial/147 ventricular, 40% steroid) implantations (median age at implant was 4.1 years [range 1 day to 21 years]). Congenital heart disease was present in 103 (84%) of the patients. Epicardial leads were followed for 29 months (range 1 to 207 months). The 1-, 2-, and 5-year lead survival was 96%, 90%, and 74%, respectively. Compared with conventional epicardial leads, both atrial and ventricular steroid leads had better stimulation thresholds 1 month after implantation; however, only ventricular steroid leads had improved chronic pacing thresholds (at 2 years: for steroid leads, 1.9 μJ [from 0.26 to 16 μJ]; for nonsteroid leads, 4.7 μJ [from 0.6 to 25 μJ]; \( P < 0.01 \)). Ventricular sensing was significantly better in steroid leads 1 month after lead implantation (at 2 years: for steroid leads, 8 mV [from 4 to 31 mV]; for nonsteroid leads, 4 mV [from 0.7 to 10 mV]; \( P < 0.01 \)). Neither congenital heart disease, lead implantation with a concomitant cardiac operation, age or weight at implantation, nor the chamber paced was predictive of lead failure.

**Conclusions**—Steroid epicardial leads demonstrated relatively stable acute and chronic pacing and sensing thresholds. In this evaluation of >200 epicardial leads, lead survival was good, with steroid-eluting leads demonstrating results similar to those found with historical conventional endocardial leads. (Circulation. 2001;103:2585-2590.)

**Key Words:** electrocardiography ■ pacemakers ■ pediatrics

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Pacing in the pediatric population typically results from bradycardia produced by sinus node dysfunction or atrioventricular (AV) block.1 Permanent epicardial pacing is often required in children because of their small size, congenital cardiac defects with right-to-left shunts, or lack of access to the chamber requiring pacing. In patients with prior cardiac surgery, the epicardium often has fibrosis and adhesions resulting in higher pacing thresholds and exit block.2–7 The increased pacing thresholds associated with epicardial leads have reduced lead longevity.3,8 Although endocardial pacing systems have demonstrated acceptable midterm sensing and pacing characteristics,9 concern has been raised about long-term vascular and/or valvular integrity with multiple transvenous leads.10,11

Recent advances in lead technology and surgical approach, such as steroid epicardial leads and left atrial leads, have demonstrated encouraging early sensing and pacing qualities when used in children.12,13 The use of these new implant strategies on long-term lead survival have not been well established. The objective of the present study was to report our long-term experience with a large number of epicardial pacemakers and to identify possible predictors of early and late lead-related complications.

**Methods**

A complete search of the cardiac surgical and pacemaker databases identified all patients who underwent permanent epicardial pacemaker implantation between January 1, 1983, and June 30, 2000, at The Children’s Hospital of Philadelphia. Patients aged >21 years at the time of pacemaker implantation were excluded so as to primarily evaluate a large pediatric cohort. All hospital records, operative records, and 1239 outpatient pacemaker clinic visits were retrospectively reviewed. (A portion of these patients were presented in an earlier study.14) These records were abstracted for data on age, sex, cardiac malformation, presence or absence of congenital heart disease, cardiac surgical procedures (type and number), surgical approach, indication for pacing, mode of pacing, chamber placement, lead fixation method, steroid lead properties, pacemaker complications, and all follow-up information available.
Operative Course
Epicardial leads were implanted through a midline sternotomy, lateral thoracotomy, or subxiphoid approach. The surgical approach was based on the patient’s underlying cardiac anatomy, cardiac position in situ, prior operation(s), and/or concurrent operation at the time of lead placement. The ventricular lead was often fixed on the diaphragmatic ventricular surface. The atrial lead was positioned on either the right or left atrium; whichever afforded the best pacing and sensing thresholds. The surplus of lead was addressed by creating loops of the electrode within the pericardium and the pacemaker pocket. Leads were tunneled with care to avoid sites of potential injury along the rib margins to the generator implant site in the abdomen.

Implant measurements were obtained by using a pacing system analyzer (Medtronic 5311). Measurements included lead impedances at 0.5 ms, V, sensed P or R waves (if present), amplitude, stimulation threshold (minimum voltage delivered at a fixed pulse width of 0.5 ms that consistently captured the tissue), and the measured current at the threshold voltage and pulse width.

Hospital Course
Acute pacing characteristics were defined as energy threshold (ET), impedance, and sensing at implantation. Hospital charts were reviewed for length of stay, complications, number and duration of antibiotics received, and discharge pacing characteristics. Pacing and sensing thresholds were interrogated in all systems 48 hours before hospital discharge.

Pacemaker Follow-Up
Pacing and sensing thresholds were evaluated at 1 month, at 3 months, and at subsequent 6-month intervals. Outpatient evaluations consisted of real-time telemetry of battery and lead measurements. Sensing and pacing thresholds were determined by using a Medtronic 5300, Pacesetter, Intermedics, or CPI system analyzer. Threshold was determined by decreasing the pulse width until there was failure to capture. Early pacemakers without pulse-width auto-threshold testing capabilities used voltage decrement at a fixed pulse width to assess threshold. The threshold was considered to be the lowest programmable pulse width at which there was consistent capture. For patients with slow ventricular escape complexes or absence of atrial activity, sensing tests could not always be performed.

Pacing and sensing thresholds were compared at implant, at hospital discharge, and at the 1-month, 6-month, 1-year, 2-year, 5-year, 10-year, and most recent pacemaker follow-up visits. For patients whose primary pacemaker care was at a referring institution, phone calls were made requesting a recent clinical summary, and pacemaker interrogation, and any lead- or generator-related complications.

Lead failure was defined as need for replacement or abandonment based on the following: (1) fracture or insulation break, (2) increasing pacing or sensing thresholds, or (3) phrenic or myopotential stimulation. Infections were classified separately into (1) superficial infection (±positive blood culture) or (2) deep infection necessitating generator removal. Lead data were censored for elective change, death, or orthotopic heart transplantation.

Definitions
ET was defined as the least amount of energy producing consistent capture outside the refractory period and was used to facilitate comparison between acute and chronic pacing thresholds. The formula used to calculate ET is as follows: ET (\(\mu J\))=\[\text{voltage (V)} \times \text{pulse duration (ms)} \times 10^6\]/[resistance (\(\Omega\)) \times 1000 ms/s].

Statistical Analysis
Exploratory data analysis was performed by using descriptive measures. Categorical variables were expressed in terms of percent-ages with standard deviations. Continuous variables were expressed as means with standard deviations if the term was normally distrib-uted; skewed variables were expressed as medians (with ranges). The strength of a statistical association was measured by use of the \(\chi^2\) test for categorical variables. When cell numbers were small, the Fisher exact test was used. Statistical significance of the difference between continuous variables was assessed by the Wilcoxon rank sum test for skewed distribution. Logistic regression or the Cox proportional hazards model was used to assess confounding variables. Lead survival was assessed by using Kaplan Meier analysis (STATA 6.0) with significance based on the log-rank test.

Results

Patient Data
A total of 123 patients underwent 158 operations for 207 epicardial lead implantations during the 17-year study period. The age at lead implantation ranged from 1 day to 21 years (median 4.1 years). The average weight at lead implantation was 17 kg (range 1.4 to 87 kg). Indications for pacemaker included postoperative AV block in 50 (40%) of the patients, sinus node dysfunction in 39 (32%) of the patients, congenital complete heart block in 23 (19%) of the patients, and other indications, such as bradycardia secondary to antiarrhythmic medication, in 11 (9%) of the patients.

The majority (84%) of the patients had structurally abnormal hearts and had undergone surgical correction(s) of complex cardiac malformations. Of the 103 patients with congenital heart disease, 37 had single ventricle physiology. The remaining 66 patients with congenital heart disease included those with the following: AV canal (14 patients), dextrotransposition of the great arteries (8 patients), ventricular septal defect (VSD) (8 patients), complex left ventricular outflow tract obstruction (7 patients), tetralogy of Fallot (6 patients), levotransposition of the great arteries with VSD (6 patients), double-outlet right ventricle (5 patients), heterotaxy syndrome and AV canal (4 patients), partial anomalous pulmonary venous return (2 patients), truncus arteriosus (1 patient), cardiomyopathy (2 patients), anomalous left coronary artery (1 patient), and atrial septal defect (2 patients).

Pacing Characteristics
The initial pacing mode used was AAI in 6 patients, VVI in 75 patients, and DDD in 42 patients. Of the 207 epicardial leads, 177 were implanted during 138 operations at a remote time from reparative or palliative cardiac surgery. Thirty leads were implanted at the completion of 20 open-heart surgical cases. Over the last 2 decades, numerous leads (Table 1) and generators (Table 2) were used, reflecting the technological advances.

<table>
<thead>
<tr>
<th>Manufacturer/Lead</th>
<th>n (%, A/V)</th>
<th>Steroid</th>
<th>Fixation</th>
</tr>
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<tbody>
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<td>Medtronic 4965 Capture</td>
<td>82 (40, 33/49)</td>
<td>Yes</td>
<td>Myocardial</td>
</tr>
<tr>
<td>Medtronic 6917 AT</td>
<td>36 (17, 1/35)</td>
<td>No</td>
<td>Screw in</td>
</tr>
<tr>
<td>Medtronic 5071</td>
<td>26 (13, 8/18)</td>
<td>No</td>
<td>Screw in</td>
</tr>
<tr>
<td>Medtronic 4951</td>
<td>38 (18, 17/21)</td>
<td>No</td>
<td>Fishhook</td>
</tr>
<tr>
<td>Medtronic 5069</td>
<td>9 (4, 0/9)</td>
<td>No</td>
<td>Myocardial</td>
</tr>
<tr>
<td>Medtronic 1029A</td>
<td>2 (1, 1/1)</td>
<td>No</td>
<td>Suture</td>
</tr>
<tr>
<td>Cordis 325</td>
<td>2 (1, 0/2)</td>
<td>No</td>
<td>Screw in</td>
</tr>
<tr>
<td>Pacesetter 1043K</td>
<td>12 (6, 0/12)</td>
<td>No</td>
<td>Screw in</td>
</tr>
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</table>

A indicates atrial lead; V, ventricular lead.
Operative and Perioperative Course
Epacardial leads were implanted by a subxiphoid approach (14%, 4 atrial/25 ventricular), a lateral thoracotomy (29%, 22 atrial/38 ventricular), or a sternotomy (57%, 34 atrial/84 ventricular). Fifty-eight patients had a chest tube placed after pacemaker implantation for a period of 1.4±1.7 days. The average length of stay after an isolated pacemaker implantation was 3 days (range 1 day to 69 days). Antibiotics were used in all patients after lead implantation for an average of 48 hours. Four patients developed a superficial cellulitis with negative blood cultures before discharge and received intravenous antibiotics for 7 days. Two other patients with congenital heart disease had a fever and positive blood culture after generator implantation. Antibiotics were given for 4 weeks, with prompt resolution of the bacteremia allowing for continued use of the pacing system. One patient developed a significant pacemaker pocket infection necessitating removal of the leads and generator.

There were 3 hospital deaths after epicardial pacemaker implantation. Two patients with prematurity, congenital complete heart block, and hydrops fetalis died secondary to lung disease of prematurity. One 6-month-old with trisomy 21 underwent pacemaker implantation for third-degree AV block 10 days after repair of an AV canal defect and suffered a cardiac arrest 3 days later.

Implant Data
The atrial ETs were 1.4 μJ (from 0.01 to 10.6 μJ) (steroid, 1.13 μJ [from 0.2 to 6.5 μJ]; nonsteroid, 2.2 μJ [from 0.01 to 8.3 μJ]; P=NS). Atrial sensing at implant was obtainable from 44 leads (2.9 mV [from 0.6 to 7.9 mV]). The atrial lead impedance was 335 Ω (from 223 to 748 Ω). No significant difference was observed in the acute ventricular stimulation thresholds between steroid (0.9 μJ [from 0.05 to 6.6 μJ]) and nonsteroid (1.1 μJ [from 0.05 to 25 μJ]) leads. Acute R-wave sensing was performed in 125 leads (11 mV [from 2 to 25 mV]). The ventricular lead impedance was 403 Ω (from 163 to 1000 Ω). No significant differences in implant stimulation thresholds were observed between atrial and ventricular epicardial leads.

Follow-Up Data
Of the 120 patients discharged after epicardial pacemaker implantation, 9 patients relocated and were lost to follow-up. Early readmission (<30 days) occurred in 13 patients. The major indication for early readmission was superficial cellulitis (n=7). All 7 patients had negative blood cultures, responded to intravenous antibiotics, and did not require removal of the lead(s) and/or generator. Three patients were readmitted with postperiadriectomy syndrome. Two other patients were readmitted with pleural effusions, and 1 Fontan patient had an exacerbation of protein-losing enteropathy after a thoracotomy for pacemaker implantation.

Late pacemaker-related readmission (>30 days) occurred in 6 patients (superficial cellulitis [n=3] and deep pacemaker pocket infection necessitating removal of leads and/or generator [n=3]). There were 5 late deaths in patients with pacemakers. Two Fontan patients died at 7 and 9 years, respectively, from pacemaker implantation secondary to low cardiac output and ventricular dysfunction. One Fontan patient with known atrial flutter died suddenly from a presumed arrhythmic death. There were 2 late deaths that may have been related to the pacemaker system. A newborn with double-outlet right ventricle, VSD, and PS developed complete heart block after a Damus-Kaye-Stansel procedure. A VVI pacemaker was implanted 10 days later. The child, who had been doing well at home, died suddenly 5 weeks later. Another newborn with transposition of the great arteries (levotransposition), VSD, PS, and congenital heart block died suddenly 7 weeks after VSD closure, left ventricle–to–pulmonary artery conduit, and pacemaker implantation. In both situations, nonsteroid-eluting epicardial ventricular leads were used.

The average length of follow-up was 29 months (range 1 to 207 months) for all epicardial lead and 19 months (range 3 to 61 months) for steroid-eluting leads. Forty-two leads had follow-up periods exceeding 5 years. Twenty-six patients had at least 1 reoperation for generator replacement with use of the original epicardial leads. An elective lead change at the time of reparative cardiac surgery occurred in 3 patients. Four other patients whose battery life had ended were changed to an endocardial system.

Lead Pacing and Sensing Characteristics
Atrial stimulation thresholds were significantly better for steroid-eluting leads at 1 month (steroid, 1.7 μJ [from 0.2 to 16.1 μJ]; nonsteroid, 4.1 μJ [from 1.2 to 18.1 μJ]; P=0.02). Atrial ETs remained relatively constant over 5 years (1.5 μJ [from 1.1 to 8.5 μJ]) without any further significant differ-

TABLE 2. Implantable Generators Used Between 1983 and 2000

<table>
<thead>
<tr>
<th>Manufacturer</th>
<th>Model</th>
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<tr>
<td></td>
<td>Symbion 7001/05</td>
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<td></td>
<td>Minuet 7108</td>
<td>1</td>
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<td></td>
<td>Pasys 8320/29</td>
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<tr>
<td></td>
<td>Minx 8341</td>
<td>2</td>
</tr>
<tr>
<td></td>
<td>Actitrix 8403/13</td>
<td>11</td>
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<tr>
<td></td>
<td>Legend 8417/19</td>
<td>12</td>
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<tr>
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<td>Spectra 8420/22/23</td>
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<td>Jewell II</td>
<td>1</td>
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<td></td>
<td>Thera 7940/50/60/8960</td>
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<td></td>
<td>Kappa 700</td>
<td>13</td>
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<td></td>
<td>Sigma 300</td>
<td>4</td>
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<tr>
<td>Pacesetter</td>
<td>Solus 2002/06</td>
<td>3</td>
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<tr>
<td></td>
<td>Phoenix-II 2008</td>
<td>9</td>
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<td></td>
<td>Synchro II 2028</td>
<td>4</td>
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<tr>
<td>Cordis (St. Jude)</td>
<td>Multicor Gamma 337</td>
<td>1</td>
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<tr>
<td>CPI (Guidant)</td>
<td>Vista T 445</td>
<td>3</td>
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<tr>
<td></td>
<td>Discovery 1174</td>
<td>1</td>
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<tr>
<td>Intermedics</td>
<td>Intertach</td>
<td>1</td>
</tr>
</tbody>
</table>
ence between steroid- and nonsteroid-eluting leads (Figure 1). The sensed P-wave amplitudes at 1 month and 2 years were 2.5 ± 1.8 mV (n = 17) and 3.2 ± 2.5 mV (n = 17), respectively (P = NS). There were no observable differences in atrial sensing between steroid and nonsteroid leads at any follow-up interval. Similarly, no acute or chronic differences in atrial lead impedance were observed between steroid and nonsteroid leads (at 1 month, 339 ± 82 V; at 1 year, 364 ± 82 V; and at 2 years, 372 ± 87 V). No acute or chronic difference in stimulation thresholds was observed between left atrial (discharge ET [n = 14], 1.7 μJ [from 0.6 to 10 μJ]; 2-year ET [n = 11], 2.3 μJ [from 0.39 to 12 μJ]) and right atrial (discharge ET [n = 27], 1.9 μJ [from 0.03 to 14 μJ]; 2-year ET [n = 21], 1.5 μJ [from 0.4 to 6 μJ]) epicardial leads (P = NS).

The ventricular stimulation thresholds were significantly better for steroid-eluting leads at acute follow-up (at 1 month: steroid, 2.4 μJ [from 0.05 to 12.7 μJ]; nonsteroid, 6.1 μJ [from 0.4 to 27 μJ]; P < 0.01) and chronic follow-up (at 2 years: steroid, 1.9 μJ [from 0.26 to 16 μJ]; nonsteroid, 4.7 μJ [from 0.6 to 25 μJ]; P < 0.01) (Figure 2). The intrinsic R waves were greater in the steroid-eluting leads at 1 month after implant (steroid, 8 mV [from 4 to 31 mV]; nonsteroid, 4 mV [from 0.7 to 10 mV]; P = 0.02) without a chronic difference (at 2-year follow-up, 5.6 mV [from 2.8 to 31 mV]).

Ventricular impedances were relatively constant over the acute and chronic period (at 1 month, 356 ± 84 Ω; at 1 year, 382 ± 94 Ω; and at 2 years, 389 ± 97 Ω). There were no significant differences in acute or chronic atrial and/or ventricular pacing or sensing between leads implanted in children with congenital heart disease and those with structurally normal hearts.

**Lead Survival**

Epicardial lead failure occurred 16% (in 34 of 207 implantations) of the time (Table 3). The mean time to lead failure was 2.4 ± 2.3 years. Increasing threshold was the most common cause of lead failure. Only 2 (2.4%) steroid-eluting leads had to be abandoned for exit block. The 1-, 2-, and 5-year epicardial lead survival was 96%, 90%, and 74%, respectively. No differences in lead survival were noted between atrial and ventricular epicardial leads (Figure 3). The 5-year freedom from survival for steroid-eluting leads was 83%, whereas for nonsteroid-eluting leads, the survival was 73% (Figure 4). The surgical approach significantly correlated with lead failure. None of the 29 subxiphoid-implanted leads failed during the present study (20 months [range 1 to 162 months]) (Figure 5). ETs ≥3.0 μJ at hospital discharge predicted early lead failure (relative risk 2.8 [95% CI 1.2 to 6.6], P = 0.02). There was no significant difference in the failure rate between epicardial leads that were redone and the remaining cohort of epicardial leads used. Neither congenital heart disease, lead implantation with a concomitant cardiac

**TABLE 3. Cause of Lead Failure or Abandonment**

<table>
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<th></th>
<th>Atrial (n = 60)</th>
<th>Ventricular (n = 147)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Increasing pacing thresholds, n (%)</td>
<td>1 (1.6)</td>
<td>14 (9.5)</td>
</tr>
<tr>
<td>Fracture, n (%)</td>
<td>3 (5)</td>
<td>8 (5.5)</td>
</tr>
<tr>
<td>Phrenic or muscle stimulation, n (%)</td>
<td>2 (3.3)</td>
<td>1 (0.7)</td>
</tr>
<tr>
<td>Inappropriate sensing, n (%)</td>
<td>2 (3.3)</td>
<td>1 (0.7)</td>
</tr>
</tbody>
</table>

**Figure 1.** Atrial ETs stratified by lead type. Nonsteroid-eluting leads (n = 23, solid bars) and steroid-eluting leads (n = 32, open bars) are shown.

**Figure 2.** Ventricular ETs stratified by lead type. Nonsteroid-eluting leads (n = 89, solid bars) and steroid-eluting leads (n = 48, open bars) are shown.

**Figure 3.** Freedom from lead failure stratified by paced chamber: for atrial lead (solid line) at 1 year, 96% (CI 84.8% to 99%); at 2 years, 90.8% (CI 77 to 96.8%); at 3 years, 83.3% (CI 66.6% to 92.6%); at 5 years, 72% (CI 48.6% to 86.1%); and at 10 years, 72% (CI 48.6% to 86.1%); for ventricular lead (dotted line) at 1 year, 94.4% (CI 88.5% to 97.3%); at 2 years, 88.6% (CI 80.6% to 93.5%); at 3 years, 81.1% (CI 71.3% to 87.8%); at 5 years, 73.8% (CI 62.7% to 82%); and at 10 years, 69.2% (CI 54.7% to 79.8%).
operation, age or weight at implantation, nor the chamber paced was predictive of lead failure.

Discussion

Although epicardial pacing is often required in small children, in patients with residual right-to-left shunts, and in patients with chambers that cannot be accessed by the transvenous route, most children can have either an endocardial or epicardial system. Although endocardial pacing requires less extensive surgery than does epicardial lead implantation, there is concern about vascular obstruction, AV valve integrity, and the limitations of lead accommodation with somatic growth. However, these risks have generally been outweighed in children by the higher acute and chronic stimulation thresholds of conventional epicardial leads, resulting in premature battery depletion and the need for subsequent operations. Recent advances in epicardial leads and surgical approaches have demonstrated improved early pacing and sensing thresholds. Prior studies evaluating epicardial leads in children have had low patient numbers or have involved series that predated both the introduction of lithium iodide batteries and steroid-eluting leads.

The excessive pacing thresholds and high incidence of exit block with conventional epicardial leads presumably arise from a combination of epicardial fibrosis, scar formation, and/or pericardial adhesions after cardiac surgery. Conventional nonsteroid epicardial leads are associated with a 45% risk of exit block when implantation thresholds exceed 0.9 V at 0.5 ms. The addition of dexamethasone to the lead delivery system reduces the inflammatory response and the formation of the fibrous capsule.

Atrial ETs were stable for both steroid- and nonsteroid-eluting leads at implantation through 5-year follow-up. The only significant drop in ET occurred 1 month after implantation in the steroid-eluting leads. This is similar to the study of Johns et al, who reported a significant decrease in atrial pulse width threshold 1 week after implantation, with little change thereafter. In contrast to the atrial leads, the nonsteroid ventricular stimulation thresholds were significantly worse than the steroid-eluting leads at acute and 2-year follow-up. The lack of significance at 5-year follow-up between steroid- and nonsteroid-eluting leads likely reflects some selection bias, in that leads with very high pacing thresholds were removed and replaced with lower ET leads. The relative consistency of our ventricular steroid-eluting leads regarding pacing thresholds over time is similar to previously published smaller epicardial series.

The absence of any significant long-term improvement in atrial pacing thresholds with steroid-eluting leads compared with ventricular leads is somewhat interesting. Most of the patients in this cohort had structural heart disease and had undergone numerous cardiac operations. It is possible that the atria in these children have significant “scarring” that cannot be overcome by the simple addition of dexamethasone. Greater benefit may be achieved by reducing the pacing lead–epicardial interface and minimizing the battery drain.
ment. However, it is generally more difficult to implant atrial leads from a subxiphoid incision, and this usually requires a limited sternotomy or thoracotomy. The observation that discharge ETs ≥3.0 μJ predicted lead failure reflects our early experience when intraoperative lead assessment was not routinely performed. In the last 5 years, our approach has been to test all pacing leads in the operating room.

Because surgical repair of complex cardiac malformations is being performed at younger ages, a growing percentage of children is expected to require pacemakers. The primary goal of pacemaker implantation is to achieve the lowest possible ET that can safely pace and sense the tissue appropriately. The present study reported acceptable pacing and sensing thresholds and a low lead-failure rate in a large cohort of epicardial leads implanted over 17 years. Steroid leads, especially ventricular leads, significantly reduced battery drain and the potential for subsequent surgery. Most children requiring a pacemaker will need one for the rest of their lives. As pediatric cardiologists, we should consider not only the immediate result of a particular lead/implant but also (and more important) how best to achieve a lifetime of pacing. Although it is technically feasible to implant endocardial leads in most children weighing ≥15 kg, use of epicardial leads when the child is older and the innominate/cephalic veins are more developed may be a more prudent approach and may minimize long-term vascular and/or valvular injury.

References
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