In children <10 kg, transvenous pacing developed in our unit in the 1980s in response to several factors. Epicardial leads at the time were prone to early loss of capture, a high fracture rate and high thresholds leading to short generator survival and the need for early reoperation. Abdominal generators were prone to migration into the peritoneal cavity or bowel. Cardiac stranulation was also an occasional occurrence. In addition, the surgical thrust in our unit was to the repair of structural lesions and reliance on cardiology support for pacemaker implantation. Early reports demonstrated that transvenous systems could be implanted with good short-term results, and this approach became the preferred route for pacemaker implantation in a few centers. Despite the early encouraging results, there has been little in the way of longer term follow-up in the literature. We now present the only long-term report of this strategy in a consecutive series of patients from a single center followed up for a minimum of 11 years over a 27-year period.

### Methods

**Patients**

A transvenous pacemaker system was implanted in 37 neonates and infants <10 kg between April 1987 and July 2003 at the Evelina London Children’s Hospital. The median age was 6.7 months (range, 1 day to 3 years) with a median weight of 4.6 kg (range, 2.7–10 kg). The indications for pacing were congenital complete heart block (CHB) in 22 (isolated in 18—that is without congenital heart disease apart from an atrial septal defect or patent arterial duct), postoperative CHB in 13, reflex anoxic seizures in 1, and long-QT syndrome in 1 patient (Table 1).

**Pacemaker Implantation**

Pacemakers (36 ventricular demand with rate response [VVIR] and 1 dual chamber [DDD]) were implanted under general anesthesia with the same technique used in older patients. The 37 ventricular leads were placed into the right ventricular apex/outflow tract through a subclavian vein puncture with a redundant loop in the atrium. Three patients were lost to follow-up; 4 patients died from complications of cardiac surgery, and 2 patients had their system removed. At long-term follow-up in 28 patients at a median of 17.2 (range, 11.2–27.4) years, 10 patients have a single chamber ventricular pacemaker, 14 a dual chamber pacemaker, 3 a biventricular pacemaker, and 1 has a single chamber implantable cardioverter defibrillator. Subclavian vein patency was assessed in 26 patients. The overall subclavian vein occlusion rate was 10 of 13 (77%) <5 kg and 2 of 13 (15%) >5 kg during long-term follow-up. After a median of 14.3 (range, 13.4–17.6) years of pacing, 7 patients continue with their original lead.

### Conclusions

Transvenous pacing in infants <10 kg results in encouraging short- and long-term clinical outcomes. Subclavian vein occlusion remains an important complication, occurring predominantly in those weighing <5 kg.
WHAT IS KNOWN

- Transvenous pacing has been used in infants weighing <10 kg with good short-term results, but no long-term reports are available.
- Epicardial pacemaker implantation is currently the favored approach for infants weighing <10 kg in many units.

WHAT THE STUDY ADDS

- Transvenous pacing has an encouraging long-term outcome in infants weighing <10 kg, with documented venous patency at up to 25 years and original lead survival up to 17.6 years.
- Weight <5 kg appears to be a risk factor for subclavian vein occlusion, while occlusion in those weighing 5–10 kg was similar to that in older pediatric patients.
- Although epicardial approach is standard, a single-lead transvenous pacing system can be implanted in infants when an open surgical procedure is not optimal.

(4–6.6F; 10 unipolar and 27 bipolar) were inserted via a subclavian vein (SCV) puncture using 4F to 7F sheaths and positioned in the right ventricular apex or outflow tract (Table 2). Initially, passive fixation leads were used (24/37), but after 1997, predominantly active fixation leads were chosen. After obtaining satisfactory pacing and sensing parameters, a redundant lead loop was advanced into the atrium to prevent lead tension during growth and the lead was secured with an absorbable suture. In the single DDD system, the unipolar 4F screw-in atrial lead was implanted through an ipsilateral axillary vein cut down. The generator was placed in a pocket in the anterior abdominal wall in 6 patients, in a prepectoral pocket in 23 patients, and in a subpectoral pocket in 8 patients. An abdominal pocket was the preferred option until 1988 and in the patient with a dual-chamber system; pacing leads were then tunneled subcutaneously to the generator site. Until 1990, there were several different operators, but thereafter all new and follow-on procedures were performed by a single operator (E.R.). SCV patency was assessed with a venogram during subsequent generator or pacemaker system change procedures.

Statistics

The SCV occlusion rate was tested for those <5.0 kg and those >5.0 kg in weight at the time of their procedure. Odds ratios and 95% confidence intervals (CI95) for proportions and differences were calculated using the Exact Method of Calculating Confidence Interval for the Difference between 2 proportions based on the binomial distribution (Stata version 13.1; StataCorp. College Station, TX). A Kaplan-Meier lead survival curve was prepared using GraphPad Prism 6.0 software (GraphPad, La Jolla, CA).

Results

Pacemaker implantation was uneventful in all 37 patients with good pacing and sensing characteristics. Follow-up was lost in 3 patients of whom 2 were from abroad. There were 4 patients who died, 3 of whom had postoperative CHB. In 1 withdrawal of care, followed repair of anomalous pulmonary venous drainage, ventricular septal defect, and coarctation of the aorta. Fungal sepsis developed postoperatively, and a transvenous pacemaker was implanted to allow the patient to go home for palliative care and the patient died at 7 months of age. A patient with left atrial isomerism developed suppurative pericarditis at 6 weeks of age because of the temporary epicardial pacing wires after an AV septal defect repair. A dual-chamber transvenous pacemaker system was implanted because of poor cardiac function and failure of the epicardial leads, but the patient died 7 days later from the ongoing sepsis. A patient with double outlet right ventricle, transposition of the great arteries, and subpulmonary stenosis died 18 months after cardiac surgery and endocardial pacemaker implantation because of heart failure. The fourth patient with levo-transposition of the great arteries paced at 3 months of age, died at another center after cardiac surgery at 3 years of age. No deaths were related to pacemaker malfunction or pacemaker-related infections. The pacing system was removed in 2 patients with postoperative CHB: in 1 patient, the system was removed because of infective endocarditis at a time that his conduction system had recovered. The other patient underwent heart transplantation 9 years after repair of transposition and a ventricular septal defect. Of the original cohort, 28 underwent long-term follow-up (Figure 1).

Early Reinterventions

There were 4 early reinterventions within 6 months of the initial system placement. In 1, there was threatened skin erosion with a relatively bulky generator, which was moved to the abdomen. In 1, who had been paced on day 1 of life after several months of in utero steroids, the wound broke down and was resutured at 4 months of age. In 1 patient, the lead was found on echocardiography to have passed through a patent foramen ovale into to the left ventricle and was successfully repositioned 2 days later. In only 1 patient in this series was there a lead displacement (passive fixation lead), and a new lead was placed 3 months after the initial implant.

Table 1. Indications for Pacemaker Implantation

<table>
<thead>
<tr>
<th>Arrhythmia</th>
<th>N</th>
<th>Structural Lesions</th>
</tr>
</thead>
<tbody>
<tr>
<td>Congenital complete heart block</td>
<td>22 (59%)</td>
<td></td>
</tr>
<tr>
<td>Isolated</td>
<td>18</td>
<td>PDA (4), PDA and ASD (1)</td>
</tr>
<tr>
<td>L-TGA</td>
<td>2</td>
<td>...</td>
</tr>
<tr>
<td>CHD</td>
<td>2</td>
<td>TOF (1), VSD* (1)</td>
</tr>
<tr>
<td>Postoperative complete heart block</td>
<td>13 (35%)</td>
<td></td>
</tr>
<tr>
<td>VSD</td>
<td>8</td>
<td>TGA (2), TGA and COA (1), DORV (1), PDA (2), CoA+TAPVD (1), ASD (1)</td>
</tr>
<tr>
<td>AVSD</td>
<td>4</td>
<td>LAI (1), left AV valve replacement (1)</td>
</tr>
<tr>
<td>Subaortic stenosis</td>
<td>1</td>
<td>ASD and VSD</td>
</tr>
<tr>
<td>Reflex anoxic seizures</td>
<td>1</td>
<td>...</td>
</tr>
<tr>
<td>Long-QT syndrome</td>
<td>1</td>
<td>...</td>
</tr>
</tbody>
</table>

*Patient had chickenpox with suspected myocarditis in utero. ASD indicates atrial septal defect; AVSD, atrioventricular septal defect; CHD, congenital heart defect, CoA, coarctation of the aorta; DORV, double outlet right ventricle; LAI, left atrial isomerism; L-TGA, levo-transposition of the great arteries; PDA, patent ductus arteriosus; TAPVD, total anomalous pulmonary venous drainage; TGA, transposition of the great arteries; TOF, tetralogy of fallot; and VSD, ventricular septal defect.
Lead Changes Before End of Life of the First Generator

A lead intervention was required in 6 patients before pacemaker generator battery depletion after a median 10.6 (range, 2.5–11.5) pacing years. In 1, the lead was advanced at 2.5 years because of straightening of the lead (loss of the atrial loop). In 2 patients with increased pacing thresholds, the leads were extracted—through the femoral vein in 1 at 4.1 years and through the SCV in 1 at 9.2 years. An ipsilateral dual-chamber pacemaker was implanted in 1 and an ipsilateral single-chamber pacemaker in 1. In 2 patients with reduced exercise tolerance at 11.0 and 11.5 years after the initial procedure, the pacemakers were upgraded to dual-chamber systems and the ventricular leads replaced—an insulation break was found in 1 and a new lead was placed in a septal position in 1. The old ventricular lead was removed completely in 1 and partially in the other with powered sheaths, allowing placement of atrial and ventricular leads through the original SCV. In 1 patient with good pacing and sensing characteristics, the pacemaker was upgraded to an epicardial DDD system during cardiac surgery for prosthetic mitral valve replacement at 10.9 years after the initial pacemaker.

Battery End of Life as the First Intervention

The first reintervention was battery end of life in 16 patients at a median 5.9 (range, 4.1–12.9) years after the initial implantation. In 11 patients, only the generator was changed at a median 6.0 (range, 4.1–12.9) years. In 1, the lead was advanced at the generator change because of straightening of the ventricular lead (loss of the atrial loop) and in 1 the system was upgraded to a dual-chamber system by adding an atrial lead at 4.2 pacing years.

In 5 patients, there were concomitant ventricular lead changes at 4.7 to 6.2 pacing years. There was loss of the atrial loop and inability to advance the lead in 3 abdominally sited generators, which were then moved to a pectoral position. The lead was changed from unipolar to bipolar to allow submuscular position of the generator in 1. In 1 patient, a well-functioning ventricular lead was damaged during the generator replacement. In 3, a new ventricular lead was placed through the ipsilateral SCV after a lead extraction. The ventricular lead could not be extracted in the fourth, but the SCV was patent and new ventricular and atrial leads were placed via the ipsilateral SCV. In 1, the lead was extracted through the femoral vein, allowing recanalization of the occluded SCV and ipsilateral lead replacement.

Lead Survival

During long-term follow-up, ventricular pacing lead revision was required in 21 of 28 patients at the first or subsequent procedures. In 6 patients (21%), lead revision was not related to lead malfunction or lead straightening (lead displacement at 3 months in 1, pocket infection in 2 at 1.7 and 6.9 years, skin erosion in 1 at 2.0 years, lead damage during elective generator replacement in 1 at 4.7 years, and concomitant heart surgery in 1 at 10.9 years).

### Table 2. Pacemaker Leads Used at the First Pacemaker Implantation

<table>
<thead>
<tr>
<th>Lead Model</th>
<th>N</th>
<th>Size (F)</th>
<th>Fixation</th>
<th>Polarity</th>
<th>Steroid Eluting</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ventricular</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Biotronic PE69/4DNP</td>
<td>3</td>
<td>5</td>
<td>P</td>
<td>U</td>
<td>No</td>
</tr>
<tr>
<td>Siemens 424M/60</td>
<td>3</td>
<td>4.8</td>
<td>P</td>
<td>U</td>
<td>No</td>
</tr>
<tr>
<td>Biotronic TIR-60-BP</td>
<td>1</td>
<td>6.6</td>
<td>P</td>
<td>B</td>
<td>No</td>
</tr>
<tr>
<td>Siemens1050T</td>
<td>1</td>
<td>6</td>
<td>P</td>
<td>B</td>
<td>No</td>
</tr>
<tr>
<td>Medtronic CapSure SP 4024</td>
<td>2</td>
<td>5.8</td>
<td>P</td>
<td>B</td>
<td>Yes</td>
</tr>
<tr>
<td>Vitatron Slimline</td>
<td>2</td>
<td>5</td>
<td>P</td>
<td>U</td>
<td>No</td>
</tr>
<tr>
<td>Vitatron Excellence+</td>
<td>8</td>
<td>5.7</td>
<td>P</td>
<td>B</td>
<td>Yes</td>
</tr>
<tr>
<td>Biotronik Y60-BP</td>
<td>2</td>
<td>6.6</td>
<td>A</td>
<td>B</td>
<td>No</td>
</tr>
<tr>
<td>Osympka K467-40</td>
<td>1</td>
<td>4</td>
<td>A</td>
<td>U</td>
<td>No</td>
</tr>
<tr>
<td>St Jude Tendril SDX 1488T</td>
<td>9</td>
<td>6.1</td>
<td>A</td>
<td>B</td>
<td>Yes</td>
</tr>
<tr>
<td>Medtronic CapSureFix Novus 5076</td>
<td>1</td>
<td>6.2</td>
<td>A</td>
<td>B</td>
<td>Yes</td>
</tr>
<tr>
<td>Medtronic CapSure Sense 4074</td>
<td>3</td>
<td>5.3</td>
<td>P</td>
<td>B</td>
<td>Yes</td>
</tr>
<tr>
<td>Intermedics Thinline EZ 438-10</td>
<td>1</td>
<td>5/7</td>
<td>A</td>
<td>B</td>
<td>No</td>
</tr>
<tr>
<td>Atrial</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Osympka K467-40</td>
<td>1</td>
<td>4</td>
<td>A</td>
<td>U</td>
<td>No</td>
</tr>
</tbody>
</table>

### Figure 1. Indication for the first reintervention after pacemaker implantation.

DDD indicates dual chamber; EOL, end of life; and LV, left ventricle.
In 15 patients (54%), lead revision caused by lead malfunction (n=8), lead straightening (n=4) at generator replacement or system upgrade, the need to change from a unipolar to bipolar lead to allow a submuscular implant (n=2) or the need to move the apical pacing lead to a septal position (n=1) occurred at a median of 9.2 (range, 4.1–14.3) years after the initial pacemaker insertion. Particularly with abdominally sited generators in the earlier years, loss of all the atrial loop raised concerns that tension might contribute to eventual lead failure during further growth and if advancement was not possible during a generator change, lead replacement was undertaken. The Kaplan–Meier survival rate of the original lead excluding non–lead-related problems was 99.5% at 5 years, 72.7% at 8 years, and 59.1% at 10 years (Figure 2).

The original lead continues to function in 7 patients (25%) at a median of 14.3 years of pacing (range, 13.4–17.6 years). The pacing leads include 2 Vitatron Excellence+, 3 St Jude Tendril, 1 Biotronik Y60-BP, and 1 Medtronic Capsurefix Novus. The indications for pacing were congenital CHB in 5 and postoperative CHB in 2. A pacemaker generator change was performed in 5 of these patients at a median 6.8 (range, 5.1–12.9) years of pacing, whereas 2 have not required any intervention despite 100% pacing at 14.3 and 13.4 years of pacing (both Regency generators; St Jude).

SCV Occlusion

There were no clinical symptoms of SCV occlusion. Venography was used to assess SCV patency in 26 patients during generator changes or lead revisions. In 2 patients who continue with their original systems (see above), SCV patency has not been formally assessed.

Subclinical SCV occlusion was documented in 10 patients at a median 9.2 (range, 0.25–12.9) years of pacing at their first reintervention (Figure 3). A new pacemaker system was implanted using the contralateral SCV in 2 patients; the transvenous pacing system was changed to an epicardial system in 2 (1 because of concomitant heart surgery and in 1 for a pacemaker site infection); 3 had lead extraction (Figure 4) and lead/s placed via the original SCV; 2 had only a generator change with lead advancement in 1 (Figure 5), and the pacemaker system was removed and a new pacemaker system deferred in 1 patient. Nine of these 10 patients were <5 kg at the initial pacemaker implantation. Of the 16 patients with a patent SCV at their first generator or lead change at a median 6 (range, 2.1–14.4) years, 4 were <5 kg and 12 were >5 kg at the initial pacemaker implantation. The SCV occlusion rate was therefore 9 of 13 (69%) <5 kg (CI95, 42.4%–87.3%) and 1 of 13 (8%) >5 kg (CI95, 1.4%–33.3%) at the time of the first intervention (Figure 3). The difference of the proportion of SCV occlusions between the 2 groups is 61% (CI95, 24.4%–80.7%).

The SCV was noted to be occluded in 2 more patients, during further revisions to their pacemaker system at 13.7 and 17.1 years of pacing: a new ventricular lead was implanted from the contralateral side in 1, and 3 new leads were implanted through the occluded SCV in 1 for biventricular pacing (Figure 6). One patient was <5 kg and 1 was >5 kg at the initial pacemaker implantation. Overall, the SCV occlusion rate was 10 of 13 (77%) <5 kg (CI95, 49.7%–91.8%), and 2 of 13 (15%) >5 kg (CI95, 4.3%–42.2%; Figure 3). The difference between the 2 groups is 62% (CI95, 23.3%–80.1%). The odds ratio of SCV occlusion in the <5 kg group are 18 times those in the >5 kg group. In 1 patient at 25 years, the SCV remains patent with 3 functioning and 2 partially extracted redundant leads (Figure 7).

Long-Term Outcome

The 28 patients with long-term follow-up underwent a total of 88 procedures including the initial pacemaker implantations, pacemaker generator changes, lead advancements, lead changes, lead extraction, pacemaker upgrades to dual chamber, biventricular, and implantable cardioverter defibrillator systems. At a follow-up of 11.2 to 27.4 (median, 17.2) years, 10 patients are paced VVI, 14 DDD, 3 biventricular, and 1 has a VVI-implantable cardioverter defibrillator.

Discussion

Despite early encouraging reports of transvenous pacemaker implantation in small children,3–5 concerns raised by SCV occlusion and the need for many years of pacing resulted in epicardial pacemaker implantation becoming the favored approach in children <10 to 15 kg in many units.6,12–14 Transvenous pacing was, therefore, only practiced in a small number of centers.6–11 To date, ours is the only single-center series of transvenous pacing in children <10 kg with systematic follow-up and is therefore unique in documenting the long-term follow-up over a minimum of 11 years ≥27 years.

Ward et al5 first reported the outcome of transvenous pacing via a SCV puncture in 5 children weighing 5.4 to 10 kg. During follow-up of 12 to 30 months, there was 1 erosion and 1 threshold rise requiring reintervention. Stojanov et al10 subsequently reported experience with transvenous pacing in 12 children weighing 2.25 to 10 kg between 1986 and 2003. There was an excellent outcome with no lead malfunction, infection, or venous obstruction over 3 months to 13 years. Robledo-Nolasco et al11 also reported their experience with transvenous pacing in 12 children <10 kg (5 patients <5 kg) between 2001 and 2007. There was 1 lead dislodgement but no venous occlusion over 4.6 years of follow-up.

In older children (mean ages, 5.3±3.9 and 9.2±4.7 years), early- and intermediate-term complication rates after transvenous pacemaker implantation (after a median of 3–8 years)
Pocket infections and erosions were seen in 3% to 8%. Lead dislodgements, exit block, and fractures were seen in 8.5% to 19%. Deaths were reported in 5% in a series of 155 children with pacemaker-related deaths in one third of those. In a mixed series of 71 patients (mean age, 5.3±4.2 years) followed up for 3.2±4 years with epicardial systems in 49 and transvenous systems in 22, there were 8 deaths, of which 2 were probably related to the pacing system. Pocket complications occurred in 6 (8.5%) patients and lead complications in 6 (8.5%) patients. In a series of 119 epicardial pacemakers implanted at a mean age of 1.8 years with a median follow-up of 6.4 years, there were pocket complications in 6.7% and lead fractures or exit block in 20%. Other procedural complications included hemotheraces and peri-cardial effusions. We had 4 patients who required early reintervention because of complications (skin erosion/infection in 2 and lead displacement/malposition in 2), 1 late pocket infection, and 1 late endocarditis. We have not experienced any lead fracture, exit block, or pacing-related mortality. The overall complication rate in our cohort was 16% (6/37), which is similar to or less than in older children followed up for a much shorter period.

Steroid-eluting epicardial leads improved the effectiveness of epicardial pacing significantly. Cohen et al showed that steroid-eluting epicardial leads had better chronic pacing thresholds than nonsteroid epicardial leads in 123 children (<21 years) over a mean follow-up of 2.4 years. The 5-year lead survival was 83% for steroid-eluting leads. Tomaske et al showed excellent sensing and pacing thresholds with bipolar steroid-eluting epicardial leads in children (<18.5 years) followed up to 12 years with ventricular lead survival of 85% at 5 years. Paech et al recently reported a single-center experience of 158 steroid-eluting bipolar epicardial leads in 82 children and adults with congenital heart disease—median follow-up of 3.3 years. Lead survival at 5 years was reported to be 93%. However, despite the improvement in epicardial lead characteristics, transvenous pacemaker leads continue to have significantly lower thresholds and better longevity. Fortescu et al implanted 256 epicardial leads and 265 transvenous leads in pediatric patients and patients with congenital heart disease (median age, 16.7 years). The estimated freedom from lead failure was 85% at 5 years with transvenous leads and 58% with steroid-eluting epicardial leads.

There are few studies of epicardial lead outcomes in children <10 kg. The majority of studies include this subset in the much larger pediatric/congenital population making comparisons difficult. Villain et al reported 34 neonates and infants undergoing epicardial pacing in 2000. There were 4 lead failures in the first year and 2 steroid-eluting leads failed within 1 month of implant. They concluded that epicardial leads should be replaced by transvenous leads at

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**Figure 3.** Subclavian vein patency documented at the first reintervention and weight at the initial pacemaker (PM) implantation. In 2 patients with an initially patent subclavian vein (SCV), occlusion was demonstrated at their third procedure.

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**Figure 4.** A patient with congenital complete heart block received a transvenous pacemaker (PM) system at 3.6 kg (A). A patent arterial duct plug was also placed. At 6 y of age, a threshold rise necessitated a pacing system revision. A venogram shows the occluded subclavian vein (SCV; B). The pacing lead was successfully removed with an evolution extraction system (C) through which 2 new guide wires were inserted (D). New atrial and ventricular pacing leads were implanted through the occluded SCV (E).
the end of life of the generator. A more favorable outcome with steroid-eluting epicardial leads was described by Aellig et al in 2007 in 22 patients. One ventricular lead displaced at day 7 and 1 was replaced at 3.2 years. One pacemaker migrated into the abdomen and required repositioning after a year to prevent bowel complications. In another patient, the pacemaker eroded into the bowel necessitating a laparotomy. Silvetti et al showed in a nonrandomized study that epicardial pacing leads had a worse survival rate when compared with transvenous pacing leads in children <1 year of age with a median weight of 3.7 (range, 1.5–7.5) kg. Epicardial leads failed in 24% of 37 patients, whereas transvenous leads failed in only 5% of 19 patients between 1984 and 1991. Cardiac strangulation by epicardial leads continues to be reported.

In our study using a variety of leads, the Kaplan–Meier estimates for freedom from lead failure (excluding patients with nonlead complications) was 95.5% at 5 years, 72.7% at 8 years, and 59.1% at 10 years of pacing. The 5-year lead survival is similar to transvenous pacing in older children, and similar or better than epicardial steroid-eluting leads in older children. Thus, lead survival even in small children—a challenging setting with a rapidly growing child—is encouraging. At a median 14.3 years of pacing, 7 patients continued with their original leads, suggesting that the atrial loop at the initial implant allowed somatic growth and contributed to the long pacing lead survival.

SCV Occlusion

Ours is the first study to document angiographically SCV patency in a large consecutive series of pacemaker implants in small children followed up over a long period. Bar-Cohen et al performed contrast venography in 85 children and young adults (median age, 15 years at implant) before a repeat pacemaker/implantable cardioverter defibrillator procedure. There was a 13% SCV occlusion at a median interval of 6.5 years. They were unable to implicate age, size, growth, or lead factors in this older population. We found that SCV occlusion after multiple procedures during long-term follow-up was more frequent in those weighing <5 kg at the time of the pacemaker implantation—10 of 13 (77%) <5 kg and 2 of 13 (15%) >5 kg. The rate of SCV occlusion in those weighing 5 to 10 kg was similar to that in considerably older patients.

We used a range of leads, the majority of which were >5F in diameter. Since then 4.1F catheter delivered bipolar leads have been developed, which are likely to have suited the small patients described here, potentially reducing the rate of SCV occlusion. The initial drawback was the need for an 8.4F steerable catheter to allow lead positioning. Despite favorable pacing parameters and SCV patency in older children, the large introducer sheath seemed to preclude its use in small children. Subsequently, Kenny and Walsh was able to deliver these leads through a 5F peel-away sheath, and more recently fixed curve 7F delivery catheters have become available. We now feel comfortable using these leads in small

Figure 5. A transvenous pacemaker was implanted in a 4.3 kg infant for congenital complete heart block (A). At 8 y of age, at the time of generator battery end of life, the chest x-ray shows the lead has straightened out (B). A venogram showed the subclavian vein was occluded (C). As the original ventricular lead had good sensing and pacing characteristics, it was advanced through the occluded subclavian vein to form a new atrial loop to allow continued growth without tension on the lead (D).

Figure 6. A patient with congenital complete heart block received a pacemaker system at 6.9 kg. The initial ventricular lead was replaced with a single pass lead through the original subclavian vein at the age of 6.5 y (A). At the age of 18 y, he developed left ventricular dysfunction and was upgraded to biventricular pacing. The venogram showed an occluded subclavian vein (B). After extracting the single pass pacing lead (apart from a tip in the right ventricle [RV]) 3 new leads (right atrium [RA], RV, and coronary sinus [CS]) were implanted (C).
children, but our short experience does not allow comparison with this report.

**Lead Extraction**

In earlier years, traction alone was used, whereas now telescoping or powered sheaths are used. The decision to extract depends on the need to recanalize an SCV and the parent/patient’s choice. If the SCV is widely patent, a failed lead may simply be abandoned and a new lead/s placed or the lead may be extracted. When the SCV is occluded, there is a lower threshold for extraction of the lead although if this fails or when the patient does not wish for an extraction, the contralateral side will be used for lead placement. A combined subclavian and femoral approach allows extraction with smaller sheaths in the SCV.32 In the presence of infection, leads are always removed and followed by contralateral or epicardial lead placement.

**Pacing Mode**

We initially subscribed to the “one lead good – two leads better” philosophy upgrading to a dual-chamber system at the earliest opportunity. With evidence that pacing could induce ventricular dysfunction when used early and at rapid rates33,34 we changed strategy. Dual-chamber pacing was generally delayed until the teens or for a clear clinical indication. Indeed, even when 2 leads were placed through a previously occluded SCV, the initial mode was usually VVI with rate response until the patient needed dual-chamber function. Pacemakers were set to VVI mode up to 90 beats per minute in neonates, reducing to 70 beats per minute by 1 year of age and subsequently as low as 50 beats per minute over the next few years. Rate response was introduced when the children became active from 3 to 4 years of age—initially VVI with rate response, ≤140 beats per minute, and subsequently ≤170 beats per minute when older. The rationale for this strategy was 3-fold. Single-chamber systems are simpler to implant and program; battery usage is considerably lower without any noticeable effect on exercise tolerance and the greatly reduced number of paced beats per 24 hours may preserve left ventricular function.

**Current Approach**

More recently, the benefits of left ventricular pacing on left ventricular function have become increasingly appreciated.35 Together with the higher venous occlusion rate in the smallest of infants, our approach has been modified such that, transvenous pacing is no longer first line for patients <5 kg. We now favor placement of steroid-eluting epicardial leads on the left ventricle reserving transvenous pacing with a 4.1F lead for failure of the epicardial system or complications. Above 5 kg, a left ventricular epicardial approach is preferred if concomitant surgery is being performed or has recently been performed. For transvenous pacing, we use 4.1F pacing leads placing the lead on the right ventricular septum or apex. Above 15 kg, we use transvenous magnetic resonance imaging conditional leads.36

**Conclusions**

Our policy of transvenous pacing in children <10 kg, using mainly 5 to 6.6F leads, has produced encouraging long-term clinical benefit, with good lead survival, albeit with a high venous occlusion rate in those <5 kg. Nevertheless, venous patency at up to 25 years has been documented, in some with multiple leads in the original vein. Even when the SCV has been occluded, recanalization, especially using modern extraction tools, has enabled ipsilateral pacemaker implantation by placing new leads through the occlusion in a proportion. Although the development of 4.1F transvenous leads may further improve the patency rate of the SCV, in those <5 kg, we would currently advocate epicardial lead implantation onto the left ventricle in the first instance.
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Disclosures
None.

References


Twenty-Seven Years Experience With Transvenous Pacemaker Implantation in Children Weighing <10 kg
Laura Konta, Mark Henry Chubb, Julian Bostock, Jan Rogers and Eric Rosenthal

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