Background—Many patients with congenital heart disease require permanent pacing for rhythm management but cannot undergo transvenous lead placement. In others, epicardial scarring prohibits adequate sensing and pacing thresholds using epicardial leads. This study describes long-term lead performance using a transmural atrial (epicardial to endocardial) pacing approach in patients with congenital heart disease.

Methods and Results—For transmural atrial (TMA) lead access, a bipolar, steroid-eluting transvenous lead was placed from the epicardium via purse-string incision or atriotomy and affixed to atrial endocardium. Records were reviewed for patient data and acute and long-term lead performance for TMA leads placed 1998 to 2004. Forty-two of 48 TMA leads remain active at last follow-up. Two leads fractured, 4 were functional at >5-year follow-up but no longer active. Freedom from lead failure 98% (95% confidence interval, 86%–100%) at mean follow-up 7.8 years. TMA leads gave excellent sensing and pacing characteristics at implant and chronically. Median acute and chronic sensing thresholds were 3 and 2.8 mV, respectively; median acute and chronic pacing thresholds at 0.5 ms were 0.9 and 0.7 V, respectively. TMA leads performed similarly in Fontan patients. Overdrive pacing for intra-atrial re-entrant tachycardia was successful in 7 of 8 patients. One patient with high baseline risk died of stroke 7 years after implant. No lead-associated thrombi were observed.

Conclusions—TMA pacing leads had excellent longevity, initial, and chronic functional properties and provide an alternative to epicardial leads in patients with congenital heart disease. Patients who cannot receive transvenous leads, have epicardial scarring or have intra-atrial re-entrant tachycardia could benefit most from routine use of this technique. (Circ Arrhythm Electrophysiol. 2014;7:652-657.)

Key Words: cardiac pacing, artificial ▪ Fontan procedure ▪ heart defects, congenital
underlying cardiac diagnoses, indication for pacing, and specific patient characteristics prompting a transmural approach to atrial lead placement. Operative reports were reviewed for concomitant procedures and lead manufacturer and model. Follow-up data were reviewed for each patient, including acute and chronic lead performance data, such as sensing and capture thresholds, echocardiography to evaluate the presence of possible lead-associated thrombus, and incidence and treatment of subsequent atrial tachyarrhythmias. Study procedures were approved by the appropriate institutional review board.

Lead Implantation

Transmural placement of atrial pacing leads has been described previously. Figure 1 illustrates the technique used to implant the TMA pacing leads, including representative chest radiographs. Leads were implanted using a purse-string incision in the atrial wall or during closure of the atriotomy incision done as part of concurrent surgical procedure. The site of endocardial implantation was targeted away from obvious scar or known surgical patch locations and was otherwise determined by underlying atrial anatomy and concurrent surgical procedure. A bipolar, steroid-eluting transvenous lead and stylet were used in all cases and advanced transmurally from the epicardium to the endocardial surface. The lead was then affixed to position the tip and bipolar ring against the atrial endocardium. The lead was then secured at the epicardial surface using the tie-down sleeve and suture at a location that allowed for neutral orientation of the lead toward the pacemaker generator.

Implantation sites and procedural characteristics are described in Table 1. Forty-three of the 48 leads were placed in the morphological right atrium. Of the 5 patients with lead placement in the morphological left atrium, 1 had a history of d-transposition of the great arteries and had undergone a Senning procedure. The lead was then secured at the epicardial surface using the tie-down sleeve and suture at a location that allowed for neutral orientation of the lead toward the pacemaker generator.

In all patients with Fontan physiology, in whom the leads were placed in the atrial chamber receiving pulmonary venous return, sutures were placed to imbricate atrial tissue around the intra-atrial portion of the pacemaker lead. This was done to reduce or eliminate the exposed surface of the pacemaker lead within the atrium to ameliorate thrombogenic risk. The precise technique to draw-in and imbricate the atrial tissue over the lead was determined by each patient’s operative procedure, underlying atrial anatomy, lead location, and the quality of surrounding atrial tissue. In the 18 Fontan patients undergoing lead placement as part of a larger procedure, an open approach was used to target the lead tip into viable myocardium under direct visualization to ensure best results. Once the lead was satisfactorily tested, sutures were placed to draw-in and wrap atrial tissue around the intra-atrial portion of the lead. The atriotomy was then closed using standard technique. In the 4 Fontan patients receiving leads as the primary procedure (and thus off of cardiopulmonary bypass), the leads were placed via purse-string incision. External sutures were then placed to draw atrial tissue into a tunnel around the intra-atrial portion of the lead. In all other patients, leads were placed in the atrial chamber receiving systemic venous return. Five patients with Fontan physiology were maintained on warfarin for indications not related to the TMA lead. Most other patients with Fontan physiology were placed on antiplatelet therapy with aspirin (n=16) or aspirin and clopidogrel (n=1) as determined by their primary cardiologist.

Leads used were Medtronic 5072 (n=43; Minneapolis, MN), Guidant 4469 (n=4; Indianapolis, IN), and Medtronic 4068 (n=1). Specific lead selection was based on the preference of the individual surgeon, related primarily to ease of use with the stylet. Each of the leads used uses a fixed endocardial helical fixation system.

Table 1. Patient Demographics and Procedure Characteristics

<table>
<thead>
<tr>
<th></th>
<th>Non-Fontan</th>
<th>Fontan</th>
<th>All Patients</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Patient demographics</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Total implants</td>
<td>34</td>
<td>24</td>
<td>58</td>
</tr>
<tr>
<td>Patients with long-term follow-up</td>
<td>26</td>
<td>22</td>
<td>48</td>
</tr>
<tr>
<td>Median age at TMA, y</td>
<td>16.4 (1.7–39)</td>
<td>7.3 (0.7–34)</td>
<td>12.3 (0.7–39)</td>
</tr>
<tr>
<td>Women (%)</td>
<td>11 (42%)</td>
<td>11 (50%)</td>
<td>22 (45%)</td>
</tr>
</tbody>
</table>

**Pacing indication**

- Sinus node dysfunction: 18, 20, 38
- Congenital AV block: 3, 0, 3
- Acquired AV block: 7, 3, 10

**Procedure characteristics**

- Placed as part of larger procedure: 16, 18, 34
- Replacement of existing lead: 7, 9, 16
- Initial Fontan procedure: n/a, 3, ...
- Fontan revision+Maze procedure: n/a, 13, ...

**Implantation site**

- Right atrial appendage: 15, 9, 24
- Right atrial wall: 11, 8, 19
- Left atrial appendage: 0, 1, 1
- Dome of left atrium: 1 (systemic venous) | 3 | 4 |

Patients were excluded who did not have sufficient chronic follow-up data as detailed in text. Age range is reported in parentheses following median age. TMA indicates transmural atrial.
Study Exclusions
Ten of the 58 patients were excluded from analysis because long-term data were unavailable: 6 patients moved; 1 was lost to follow-up; 1 patient expired 2 years after lead placement while awaiting orthotopic cardiac transplant; 1 patient presented with pallor during right atrial tachycardia 1 year after lead placement (due to a rare form of primary arrhythmia) and was converted to a transvenous implantable defibrillator. One patient underwent reoperation for underlying congenital heart disease 2 years after lead placement and a functional TMA lead was removed. The remaining 48 patients for whom long-term data were available were all included in analysis.

Statistical Methods
Categorical variables are reported as number of cases and frequency or percentage. Continuous variables are reported as mean±SD or as median with range and interquartile range as noted. The Mann–Whitney U test was used to compare initial and chronic lead characteristics in non-Fontan and Fontan groups separately. A Kaplan–Meier curve was used for event rate estimation of lead failure. Lead failure was defined as a fractured or otherwise inoperable lead. Leads with satisfactory sensing and pacing characteristics at last follow-up but currently inactive were considered as functional leads although they were censored at the time they were made inactive. All 58 patients who underwent lead implantation were included, with data censored at time of last follow-up. 95% confidence interval also plotted. Statistical analysis was done using Stata (Stata/IC for Mac version 12.1 revision 20; StataCorp, College Station, TX).

Results

Patient and Procedure Characteristics

Patient Demographics
Patient data are shown in Table 1. Twenty-two of the 48 late follow-up patients had Fontan procedures and had TMA lead placement at an average age of 7.3 years (0.7–34 years) when compared with the non-Fontan patients who averaged 16.4 years (1.7–39 years).

Indication for Pacing
Pacing with a permanent pacemaker was initiated for patients because of sinus node dysfunction in 38 and complete AV block in 13 (3 patients had >1 indication).

Indication for Transmural Lead Placement
This technique was not used in any patient who was a candidate for transvenous lead placement. Contraindications to transvenous placement included venous anatomy status after Glenn or Fontan procedure (n=24), discontinuous superior vena cava (n=1), too young or small for transvenous placement (n=12), leads placed as part of concurrent operative procedure (n=12), or a combination of the above.

The following concerns with epicardial lead placement prompted the decision to proceed with lead implantation via a transmural approach: extensive epicardial scar or limited surgical access to epicardium (n=12), limited size and surface area of possible epicardial sites (n=8), Fontan revisions with grossly abnormal atrial tissue (n=13), extensive scarring and thinning of atrial tissue (n=22), or a combination thereof. Nine patients initially had traditional epicardial atrial leads placed but had immediate concerns with pacing performance of these leads. Each of these patients then underwent successful transmural lead placement. All patients had epicardial ventricular leads placed at the time of the procedure. Two of these patients had intraoperative concerns for poor lead sensing and pacing performance; epicardial ventricular pacing leads functioned well in the remainder.

Underlying Anatomy in Fontan Patients
Patients with Fontan physiology represented various forms of underlying structural heart disease: tricuspid atresia (n=8), double-inlet left ventricle (n=4), unbalanced AV canal (n=3), double-outlet right ventricle (n=3), hypoplastic left heart syndrome (n=2), and other (n=2).

Lead Performance

Active Leads, Lead Failure, and Functional Leads That Are No Longer Active
Lead performance characteristics are shown in Table 2. For the 48 patients with long-term data available, 42 leads remained active at the time of last follow-up. 95% confidence interval also plotted. Statistical analysis was done using Stata (Stata/IC for Mac version 12.1 revision 20; StataCorp, College Station, TX).

Table 2. Lead Performance Characteristics

<table>
<thead>
<tr>
<th>Lead Performance</th>
<th>Non-Fontan</th>
<th>Fontan</th>
<th>All Patients</th>
</tr>
</thead>
<tbody>
<tr>
<td>Follow-up duration (months±SD)</td>
<td>86.7±24.2</td>
<td>102.7±28.5</td>
<td>93.7±27.1</td>
</tr>
<tr>
<td>Active leads</td>
<td>24</td>
<td>18</td>
<td>42</td>
</tr>
<tr>
<td>Functional lead now inactive</td>
<td>2 (at 5, 6 y)</td>
<td>1 (at 6 y)</td>
<td>3</td>
</tr>
<tr>
<td>Patient deceased</td>
<td>0</td>
<td>1 (at 7 y)</td>
<td>1</td>
</tr>
<tr>
<td>Lead failure</td>
<td>Lead fracture</td>
<td>0</td>
<td>2 (at 4, 8 y)</td>
</tr>
<tr>
<td>Lead dislodgment</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Lead pacing performance</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Initial pacing threshold at 0.5 ms, V</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Median (range)</td>
<td>0.9 (0.3–1.8)</td>
<td>0.9 (0.4–1.7)</td>
<td>0.9 (0.3–1.8)</td>
</tr>
<tr>
<td>Interquartile range (25–75)</td>
<td>0.7–1.0</td>
<td>0.5–1.0</td>
<td>0.6–1.0</td>
</tr>
<tr>
<td>Most recent pacing threshold at 0.5 ms, V</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Median (range)</td>
<td>0.7 (0.2–2.4)</td>
<td>0.7 (0.4–2.2)</td>
<td>0.7 (0.2–2.4)</td>
</tr>
<tr>
<td>Interquartile range (25–75)</td>
<td>0.5–1.0</td>
<td>0.6–0.9</td>
<td>0.6–1.0</td>
</tr>
<tr>
<td>Lead sensing performance</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Initial sensing threshold, mV</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Median (range)</td>
<td>3 (0.5–8)</td>
<td>2.9 (0.8–10.4)</td>
<td>3 (0.5–10.4)</td>
</tr>
<tr>
<td>Interquartile range (25–75)</td>
<td>2.1–4.0</td>
<td>2.2–4.4</td>
<td>2.1–4.1</td>
</tr>
<tr>
<td>Most recent sensing threshold, mV</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Median (range)</td>
<td>2.8 (1–5.6)</td>
<td>2.8 (0.3–5.6)</td>
<td>2.8 (0.3–5.6)</td>
</tr>
<tr>
<td>Interquartile range (25–75)</td>
<td>1.4–3.7</td>
<td>2.7–3.3</td>
<td>2.0–3.6</td>
</tr>
<tr>
<td>IART</td>
<td>Preoperative IART</td>
<td>7</td>
<td>13</td>
</tr>
<tr>
<td>IART resolved with overdrive pacing</td>
<td>3/3</td>
<td>4/5</td>
<td>7/8</td>
</tr>
</tbody>
</table>

Functional leads that are now inactive are detailed in text. Sensing thresholds were obtained in 44/48 patients who had intrinsic P waves. No statistically significant differences (P<0.05) were observed between initial and chronic pacing and sensing characteristics in each group. IART indicates intra-atrial reentrant tachycardia.
active at time of last follow-up, with mean follow-up duration 94±27 months (7.8±2.2 years). Of the 6 inactive leads, 2 patients experienced lead fractures, at 4.2 and 8.7 years after implant, respectively. Neither of these leads had fractures that were evident radiographically. Fracture was inferred from lead characteristics consistent with fracture on device interrogation.

In 4 other patients, currently inactive leads were included in analysis as functional leads. One patient expired because of massive stroke 7 years after lead implantation, with circumstances detailed below. The lead remained fully functional before the event. Two patients underwent late reoperation for their underlying structural congenital heart disease with excision of TMA lead at 5.4 and 6 years after lead implantation. One patient developed a severe mediastinal infection at 6.6 years after implantation, and though it did not seem to be involved, the lead was excised without complication as part of her management. Each of these leads was fully functional at time of excision. Thus, 46 of 48 leads were considered functional in our analysis. There were no lead dislodgments. Kaplan–Meier estimate of freedom from lead failure at mean follow-up time of 7.8 years is 98% (95% confidence interval, 86%–100%). Freedom from lead failure is plotted in Figure 2.

**Lead Sensing and Pacing Performance**

Acute and chronic sensing and pacing performance are detailed in Table 2. Lead pacing characteristics were good at time of implant, with a median pacing threshold at 0.5 ms of 0.9 V (range, 0.3–1.8 V). At the time of most recent follow-up, pacing threshold at 0.5 ms remained stable, with median of 0.7 V (range, 0.2–2.4 V). There was no statistically significant difference between initial and chronic sensing characteristics in either group of patients (non-Fontan, \( P=0.39 \); Fontan, \( P=0.81 \)).

In the 44 patients with intrinsic P waves, sensing thresholds were also good and stable. At the time of implant, median sensing threshold was 3 mV (range, 0.5–10.4 mV). At latest follow-up, median sensing threshold was 2.8 mV (range, 0.3–5.6 mV). Of 14 Fontan patients with an intrinsic atrial rhythm, 13 had sensing thresholds >3.0 mV at their latest follow-up. There was no statistically significant difference between initial and chronic sensing characteristics in either group of patients (non-Fontan, \( P=0.50 \); Fontan, \( P=0.72 \)).

**Intra-Atrial Re-entrant Tachyarrhythmias and Lead Performance**

Thirteen Fontan and 7 non-Fontan patients had documented episodes of intra-atrial re-entrant tachycardia (IART) before TMA lead placement. Each of the 13 patients undergoing Fontan revision at time of lead implantation had an atrial Maze procedure performed concurrently. Four non-Fontan patients underwent an atrial Maze procedure at time of lead placement.

In follow-up after TMA lead implantation, 3 Fontan and 2 non-Fontan patients had nonsustained episodes of IART, which were treated medically. Five Fontan and 3 non-Fontan patients had sustained episodes of IART recalcitrant to medical therapy alone. There were no cases of atrial fibrillation observed. Each of these patients underwent atrial overdrive pacing using the TMA lead. Overdrive pacing successfully terminated the episode of IART in 3/3 non-Fontan and 4/5 Fontan patients. IART cycle length ranged from 200 to 380 ms, with a median of 255 ms. Overdrive pacing was successful at cycle lengths ranging from 160 to 320 ms, with a median of 190 ms; overdrive:IART cycle length ratios ranged from 0.57 to 0.9, with a median of 0.72. One patient with unsuccessful overdrive pacing of an IART cycle length of 200 ms had attempted overdrive at cycle lengths from 150 to 190 ms. Lead proximity to the re-entrant circuit in this case and all others was unknown. Lead locations for successful overdrive pacing were right atrial wall (n=4), right atrial appendage (n=2), and dome of left atrium (n=1). The 1 patient for whom overdrive pacing was unsuccessful had an underlying diagnosis of tricuspid atresia status after Fontan revision and had a lead implanted in the right atrial appendage.

Three patients had IART postoperatively that was not documented preoperatively—2 Fontan patients and 1 non-Fontan. Each of these patients had leads placed as part of a larger procedure that would be expected to alter the underlying anatomic substrate for arrhythmias.

**Lead-Associated Complications**

Echocardiography was reviewed for each patient. There were no thrombi attached to, or in the vicinity of, the TMA lead for any patient, either acutely after implant or chronically. One patient expired because of stroke 7 years after lead implantation. The patient was 35 years old and had undergone an extracardiac Fontan procedure for double-inlet left ventricle. She had a residual pulmonary arterial stump and was taking aspirin. Echocardiography had no evidence of any thrombus related to the TMA lead or any other location. The lead was implanted in the right atrium although the patient did have an unrestricted atrial communication. On autopsy, there were findings about primary carotid artery intimal abnormality. On inspection of the interior surface of the heart, no exposed portion of the TMA lead was evident, and there was no evidence of an intracardiac thrombus. Histopathology of the brain, kidneys, liver, and other organs demonstrated no findings consistent with chronic microembolization. Although the patient...
was at high risk of stroke from multiple causes, a lead-associated thromboembolic event cannot conclusively be excluded.

There were no other clinical findings consistent with thromboembolic events in other patients. Routine surveillance for subclinical microembolization was not performed because noninvasive modalities for assessment (eg, MRI and angiography) can be problematic in patients with pacemaker.

**Discussion**

This retrospective review demonstrates that TMA pacing leads have excellent early and late functional properties as an alternative to problematic epicardial leads in patients with complex congenital heart disease potentially. These TMA leads maintained consistently low atrial pacing thresholds throughout long-term follow-up. Atrial sensing thresholds were also favorable and remained stable over time. Lead longevity was good, with 42 of 48 leads active at last follow-up and excellent freedom from lead failure. In addition, TMA leads were effectively used to terminate IART using overdrive pacing, thus avoiding the need to arrange administration and sedation to use direct current cardioversion in those patients. These lead performance properties held true in patients with a variety of Fontan anatomies.

Many patients with complex congenital heart disease require permanent pacing and are not candidates for transvenous leads. Epicardial lead sites can also be problematic because patients frequently have extensive epicardial scarring because of multiple re-entry stenotomies and may have surgical patches or extensive atrial suture lines limiting availability of an appropriate epicardial site for lead implantation. Transmural placement of atrial pacing leads has been previously described and has been performed on selected patients at our institution since 1998. The present analysis of chronic TMA lead performance validates the use of this approach as a practical and appropriate treatment option. Patients at risk for postoperative intra-atrial re-entrant tachyarrhythmias may also benefit from TMA leads to facilitate overdrive pacing as a safe alternative to direct current cardioversion.

Ease of lead removal is an important factor when considering implantation technique. Not including 3 patients in whom leads were excised as part of de-reoperation for their underlying structural heart disease, only 1 patient in our series underwent lead removal. This patient had a severe mediastinal infection 6 years after implant, and although the pacing system did not seem to be involved, it was removed as part of her management. The procedure was done on cardiopulmonary bypass for considerations not related to the pacing system. The transmural lead, including lead tip, was successfully removed without complication. In general, lead extraction of a transmural lead may be expected to be similar to epicardial leads. As with epicardial leads, one may expect to encounter difficulty excising the lead tip occasionally although this is frequently unnecessary unless it is grossly infected.

This retrospective review supports the further use and analysis of TMA pacing leads, especially in patients with complex congenital heart disease. Given the present findings of excellent lead longevity and performance, a future logical extension, already underway, is to investigate using a similar transmural approach for ventricular lead placement in dual-chamber or biventricular pacing systems in patients for whom epicardial or transvenous lead placement may be impractical. Two patients included in this analysis had ventricular leads concurrently placed using a transmural approach (through the tricuspid valve to subpulmonic right ventricle) after multiple epicardial ventricular leads exhibited poor sensing and pacing characteristics intraoperatively.

**Limitations**

Although this study represents the largest report on the use of this technique, and the only published long-term analysis, there are several limitations. The data originate from a single center, and patient numbers are somewhat limited. Thus, results may not be generalizable to other settings. The informative censoring of the event times of 10 patients with no follow-up data beyond 2 years and 4 patients with inactive leads may bias the results. For the 10 patients, no long-term follow-up data were available for the following reasons: lost to follow-up (including moving), reoperation for structural heart disease, defibrillator placement, and death while awaiting heart transplant (detailed above).

The patients were not randomized to different treatment arms of TMA versus standard epicardial leads, and the data were not blinded. This study does not account for multiple potential confounding variables that may affect lead performance although these would largely tend to have a negative effect on lead performance, which was not observed in this study. Finally, it is conceivable that placement of the TMA lead, while relatively focal, could contribute to postoperative IART, as might be the case for any surgical scar/barrier.

**Conclusions**

TMA pacing leads exhibit excellent early and late functional properties as an alternative to problematic epicardial pacing leads in patients with complex congenital heart disease potentially, including patients with Fontan anatomies. TMA leads had good longevity for the overwhelming majority of these patients and maintained low atrial pacing thresholds and favorable sensing thresholds. Transmural leads had the additional benefit of effective atrial overdrive pacing in the treatment of IART. Routine use of TMA pacing leads may be considered for patients with extensive epicardial scarring or contraindications to transvenous lead placement.

**Disclosures**

Dr Perry has received speaker fees from Biotronik. The other authors report no conflicts.

**References**

CLINICAL PERSPECTIVE

Many patients with complex congenital heart disease require permanent pacing as part of their management and are not candidates for transvenous leads. Epicardial lead sites can be also be problematic because patients frequently have extensive epicardial scarring because of multiple re-entry sternotomies and may have surgical patches or extensive atrial suture lines limiting availability of an appropriate epicardial site for lead implantation. Placement of atrial pacing leads using a transmural approach can ameliorate these concerns, particularly when placed as part of a larger procedure, which allows direct visualization and targeting of the lead tip into viable myocardium. The present analysis of chronic transmural atrial lead performance demonstrates excellent lead longevity and performance and validates the use of this approach as a practical and appropriate treatment option. Patients at risk for postoperative intra-atrial re-entrant tachyarrhythmias may also benefit from transmural atrial leads to facilitate overdrive pacing as a safe alternative to direct current cardioversion.
Long-Term Follow-Up Shows Excellent Transmural Atrial Lead Performance in Patients With Complex Congenital Heart Disease
Matthew R. Williams, Suzanne M. Shepard, Nicole K. Boramanand, John J. Lamberti and James C. Perry

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