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ORIGINAL ARTICLE

Permanent pacing in infants and children: A single center experience in implantation and follow up

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Abstract *Background:* Permanent pacing in pediatric population has been growing in Egypt. The aim of this study is to present one center experience in pediatric pacing including implantation procedures and long-term outcome.

Methods and results: During the period from 1996 to 2010, we collected the data of 32 children (18 males) with a mean age of 5.7 ± 3.8 years that underwent permanent pacemaker (PPM) implantation. Their mean weight was 21.6 ± 13.8 kg, and median body surface area (BSA) was 0.7 m^2. Twenty-five patients (78.1%) had congenital heart disease (CHD). Pacing was done via subclavian vein puncture while epicardial pacing was done via standard surgical techniques. All patients were followed up for 0.25–14 years (median: 2.5 years). Suboptimal pacing parameters were defined by one or more of the following: R/P wave malsensing, pacing threshold >2 V, or battery longevity of <1 year. The first PPM was endocardial in 21 patients (65.6%) and epicardial in 11 patients (34.4%). VVI PPMs were implanted in 8 cases (25%), VVIR in 20 cases (62.5%) and DDD in 4 cases (12.5%). A total of 46 procedures were done during the period of study, and total of 44 pulse generators and 46 leads (31 endocardial) were implanted. Fourteen patients (43.7%) required 2nd ± 3rd procedures. During follow up, suboptimal pacing parameters or pacing system failure were reported in 12 patients (37.5%) who had significantly lower age, weight and BSA

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($P = 0.048$, 0.023, and 0.032, respectively). The overall battery survival was 60% at 125 months, and ventricular lead survival was 63% at 125 months, with no significant difference in survival between epicardial and endocardial leads ($P > 0.05$).

Conclusion: Permanent pacing in pediatric age group is relatively safe. However, there is substantial higher incidence of suboptimal pacing parameters and pacing system failures especially in younger and smaller children. Epicardial steroid eluting leads are comparable to endocardial steroid eluting leads in performance.

1. Introduction

Permanent pacemakers (PPMs) have a growing use in pediatric population due to congenital and surgically acquired rhythm disturbances, but they present unique problems and implications for their implantation and follow-up. The diversity and complexity of pediatric patients and congenital heart disease make device management a highly individualized art. In pediatric pacing there are certain challenges that are not posed in adults like somatic growth, active life style, susceptibility to infection and the generally anticipated long survival.

Permanent cardiac pacing in pediatric patients is performed in few cardiology and cardiothoracic centers in Egypt. The aim of the current study is to present our institute's experience in pediatric and adolescent pacemaker implantation and long-term outcome.

2. Methods

Between 1996 and 2010, thirty-two pediatric patients who underwent permanent pacing were recorded and followed up at outpatient pacemaker follow up clinics of Cardiology Department, Ain Shams University. The techniques used for the first PPM implantation were recorded whether it was endocardial or epicardial. We also recorded any complications during the follow-up and the change of the pacing system or the pacing mode.

2.1. Definitions

Battery replacement was defined as the placement of a new pulse generator only, while PPM replacement was defined as the placement of a new pulse generator and one or more pacing lead(s) with abandonment of old pacing leads. Complications reported during follow up period were divided into two categories: Lead related complications which are related to mechanical or functional failure, and non-lead related complications.

2.2. Implantation techniques

Endocardial or epicardial pacing was chosen according to the presence of complex congenital heart disease, type of corrective surgery and/or the size of the patient. Epicardial pacing was preferred in children of small size, in the presence of intracardiac right-to-left shunt or single ventricle physiology, concomitant heart surgery, or lack of venous access to the heart chambers.[1] The majority of procedures were performed under general anaesthesia. Antibiotic prophylaxis was routinely given perioperatively to all patients.

2.3. Endocardial pacing

The endocardial leads were inserted via percutaneous puncture of the right or left (rarely) subclavian vein. Active fixation atrial leads were screwed into the right atrial appendage (RAA) or right atrial (RA) free wall in case of inability to implant in RAA. Ventricular leads were placed in the non-systemic ventricular apex or outflow tract using passive or active fixation leads. An atrial loop (complete or incomplete)[2] was attempted in all patients to allow for somatic growth. Acute pacing thresholds (measured with a pulse width of 0.50 ms at implantation), impedances, and sensing of spontaneous atrial or ventricular electrograms were evaluated during the implantation procedure.

2.4. Epicardial pacing

The PPM pulse generator was placed in the abdominal wall in a subcutaneous or submuscular (generally in infants) pockets. The leads were inserted by standard surgical techniques either through sternotomy or lateral thoracotomy. Epicardial leads were regularly placed on the RA and the right (or left) ventricle.[3]

2.5. Follow up

All patients were followed up every 3–6 months at the outpatient pacemaker follow up clinics. Follow up included clinical examination, interrogation of the device with regular measurement of pacing thresholds, ventricular and atrial spontaneous electrogram amplitudes, lead impedances as well as lead and battery longevities. The type and timing of complications were reported.

2.6. Statistical analysis

Data were analyzed using the SPSS program version 15. Quantitative data were presented using the mean and standard deviation or median, while qualitative data were presented in frequencies and percentages. Correlation of data was analyzed using the Pearson correlation coefficient. The Kaplan–Meier method was used to study the longevity of the batteries and leads.

3. Results

3.1. Demographic and clinical characteristics at first implantation

Between 1996 and 2010, thirty-two patients underwent first PPM implantation including 18 males and 14 females, with

Table 1 Demographic and clinical characteristics at first implantation.

Demographic and clinical data	$n = 32$
Age (years)	
Range, median	0.16–15 (5)
Sex	
Male (no., %)	18 (56.3%)
Female (no., %)	14 (43.7%)
BSA (m^2)	
Range, median	0.3–1.5 (0.7)
Weight (kg)	
Range, mean ± SD	6–74 (21.6 ± 13.8)
Pacing indication (no., %)	
Postoperative CHB	16 (50%)
Congenital AV block	13 (40.6%)
SND	3 (9.4%)
Heart disease (no., %)	25 (78.1%)
Surgically repaired VSD	13 (40.6%)
L-TGA	3 (9.4%)
Valvular PS	1 (3.1%)
Aortic CoA + subaortic membrane	1 (3.1%)
Repaired F4	1 (3.1%)
D-TGA	1 (3.1%)
Single ventricle (fontan)	2 (6.2%)
Repaired AV canal	1 (3.1%)
DCM	2 (6.2%)

BSA: Body surface area, CHB: Complete heart block, AV block: Atrioventricular block, SND: Sinus node dysfunction, VSD: ventricular septal defect, L-TGA: Levo-transposition of great arteries, PS: pulmonary stenosis, CoA: coarctation of the aorta, F4: Fallot's tetralogy, D-TGA: Dextro-transposition of great arteries, DCM: dilated cardiomyopathy.

age range of 0.16–15 years (median: 5 years, mean: 5.7 ± 3.8 years). Weight ranged between 6 and 74 kg (mean: 21.6 ± 13.8 kg). Body surface area (BSA) ranged between 0.3 and 1.5 m^2 (median: 0.7 m^2). Pacing indications were postoperative complete heart block (CHB) in 16 patients (50%), congenital AV block in 13 patients (40.6%), and symptomatic sinus node dysfunction (SND) in three patients (9.4%). Seven patients (21.8%) had structurally normal hearts, while 25 patients (78.1%) had congenital/structural heart disease (Table 1). Total number of procedures was 46 (1.4 per patient).

3.2. First implantation

The first implanted pacing system was a VVI PM in eight cases (25%), VVIR in 20 cases (62.5%), and DDD in four cases (12.5%). Endocardial pacing was performed in 21 patients (65.6%), while epicardial pacing was performed in 11 patients (34.4%). The smallest weight and BSA for endocardial pacing reported were 12 kg and 0.3 m^2, respectively.

3.3. Second procedure

Eleven patients (34.3%) required a 2nd procedure within 0.5–11 years from 1st implantation. Six patients underwent battery replacement as a 2nd procedure due to reaching battery elective replacement indicator (ERI) or end of life (EOL) in four patients, and for impending skin erosion in the remaining two patients. PPM replacement was performed in two patients (for battery ERI plus ventricular and atrial lead fractures which necessitated replacement in one patient and for skin erosion in the other patient). One patient had ventricular lead repositioning after early lead dislodgement. Switch from epicardial to endocardial pacing was performed as a 2nd procedure in two patients (due to battery depletion and lead insulation break in one patient and due to battery ERI with system upgrade from VVIR to DDD in the other patient).

3.4. Third procedure

Three patients (9.3%) required 3rd procedure within 0.5–8 years from 2nd procedure. One patient needed pacing system upgrade from VVI to DDD with ventricular lead replacement due to lead fracture. One patient required ventricular lead replacement due to failure of capture. The third patient had battery replacement due to battery ERI (Table 2).

3.5. Implantable generators

A total of 44 batteries were implanted during the period of study. Table 3 shows battery manufacturers and models used in the study population.

3.6. Pacing leads

A total of 46 leads were implanted during the period of the study (thirty-six were implanted during 1st procedure); thirty-one leads were endocardial (15 passive and 16 active fixation), and 15 leads were epicardial.

All epicardial and endocardial leads were steroid eluting. Failures that necessitated re-intervention occurred in six leads (13%); three endocardial and three epicardial leads. Mechanical failure (fracture/insulation break/dislodgement) occurred in two endocardial leads and three epicardial leads, while functional failure (pacing malfunction) occurred in one endocardial lead. The proportions of lead failure in endocardial versus epicardial leads were not statistically significant ($p = 0.37$).

Furthermore, three more epicardial leads showed mechanical and functional problems including insulation break in one lead and sensing malfunction (R wave < 5 mV) in two leads. These problems did not require re-intervention.

3.7. Follow up

Follow up duration ranged from 0.25 to 14 years (Median: 2.5 years). Patients with suboptimal pacing parameters or pacing system failure were defined by one or more of the following: (1) Pacing failure: Failure of capture at the highest pulse amplitude and/or pulse width. (2) Pacing threshold > 2 V ± lead impedance > 1500 Ohm. (3) P/R wave malsensing (P wave < 1.5 mV, R wave < 5 mV). (4) Battery longevity < 1 year.

Patients' group with suboptimal pacing parameters/pacing system failure during follow up included 12 patients (37.5%), with median age of 4 years (range: 0.16–8 years), mean weight of 14.5 ± 5.8 kg, and median BSA of 0.62 m^2 (range: 0.31–0.93 m^2) at 1st implantation. Patients' group with optimal pacing parameters included 20 patients, with median age of 6.2 years (range: 0.75–15 years), mean weight of

Table 2 Types and indications of second and third procedures.

Type of procedure (n = number of patients)	Indications of procedure	Endocardial pacing (n, %)	Epicardial pacing (n, %)	Duration (years) from the previous implantation
Battery replacement $n = 7$	Battery ERI/EOL.	2 (6.2%)	3 (9.3%)	2–8
	Impending skin erosion	2 (6.2%)		2,5
PM upgrading (VVI to DDD) with ventricular lead replacement $n = 1$	Lead fracture	1 (3.1%)		6
Pacing system replacement $n = 2$	Battery depletion + lead fracture (atrial and ventricular). Skin erosion	1 (3.1%)	1 (3.1%)	5 3
Lead repositioning $n = 1$	Lead dislodgement	1 (3.1%)		0.5
Lead replacement $n = 1$	Lead failure (failure of capture)	1 (3.1%)		0.5
Switch from epicardial to endocardial pacing $n = 2$	Battery depletion + insulation break. Battery ERI with PM upgrade to DDD		1 (3.1%) 1 (3.1%)	2 11

ERI: elective replacement indicator, EOL: end of life.

23.2 ± 11.6 kg, and median BSA of 0.82 m^2 (range: 0.33–1.4 m^2). Age, weight, and BSA were significantly lower in patients with suboptimal pacing parameters/pacing system failure than that of patients with optimal pacing parameters ($p = 0.048$, 0.023, and 0.032, respectively). No otherwise difference between the two groups as regards gender, presence of heart disease, pacing approach, passive or active-fixation leads, and ventricular lead looping (Table 4).

3.8. Pacemaker dependency

At 3–6 months follow up; 15 patients (46.8%) were PPM dependent with complete absence of intrinsic R waves. Among these patients, 14 patients (93.3%) had CHD while one patient (6.6%) had structurally normal heart, and 10 patients (66.6%) had post operative CHB while 5 patients (33.3%) had congenital AV block.

3.9. First battery longevity

The overall longevity of PPM batteries, independently from pacing mode and battery manufacturer is presented by Kaplan–Meier survival estimate curve (Fig. 1). At 50 months ~75% of batteries were still functioning, and the proportion decreased to ~60% at 125 months.

3.10. First ventricular lead longevity

The overall longevity of RV leads, independently from pacing approach is represented by Kaplan–Meier survival estimate curve (Fig. 2A). At 50 months ~85% of leads were still functioning and the proportion decreased to ~63% at 125 months. No significant difference in ventricular lead longevity between endocardial and epicardial leads ($p = 0.38$, Fig. 2B).

3.11. Complications

A total of 12 patients (37.5%) developed complications during the period of the study. Lead related complications or failures occurred in six patients (18.7%), while non-lead related complications occurred in six patients (18.7%). Non-lead related complications included skin erosion in three patients at 2.92 ± 1.82 years follow up. One patient had pectoral muscle twitching that was managed by decreasing pacing amplitude and pulse width from 3.5 V at 0.49 ms to 2.5 V at 0.43 ms. One patient (with VVI PPM) developed atrial flutter at 1.7 years from implantation that was managed by electrical cardioversion. One patient had keloid formation at healing site.

Table 3 Implantable generators used in study population.

Manufacturer	Model	Number
St. Jude Medical	MICRONY SR+ 2425T	1
	MICRONY II SR 2525T	12
	Verity ADx XL SR 5156	1
	Regency SCX 2408L	1
Medtronic	SIGMA SSR 303	7
	SIGMA SSR 203	4
	SIGMA TM SWI 103	2
	Relio	1
	PRODIGY S 8164	1
	En pulse E2DR01	2
Biotronic	Pikos 01	1
	Talos SR	2
	Axios SR	1
	Talos D	1
	Talos DR	1
Vitatron	Vita2	1
	TOPAZ 3 SSIR	1
Telectronics	Reflex model 8218	1
Ela-Sorin	SORIN Diapason	1
	NEWAY DR	1
Guidant	INSIGNIA I AVT	1

4. Discussion

Children represent a unique subset of patients with special characteristics including anatomical variations depending on

Table 4 Patients with suboptimal pacing parameters/pacing system failure compared to patients with optimal pacing parameters during follow up.

	Suboptimal pacing parameters/ pacing system failure (n = 12)	Optimal pacing parameters (n = 20)	P-value
Median age (yrs)	4	6.2	0.048
Mean weight (kg)	14.5 ± 5.8	23.2 ± 11.6	0.023
Median BSA (m^2)	0.62	0.82	0.032
Sex (M/F)	9,3	9,11	0.147
Heart disease (no.)	11	14	0.16
Pacing approach (no.) (endocardial/epicardial)	6, 6	15, 5	0.25
Atrial lead (no.) (passive, active, epicardial)	0, 0, 1	0,2,1	1.00
Ventricular lead (no.) (passive, active, epicardial)	4, 2, 6	11, 4, 5	0.39
Looping of ventricular lead (no.)	12	17	0.48

BSA: body surface area, M: males, F: females.

the type of CHD, small vascular access, somatic growth and higher frequency of infections and traumatic events making them more prone to complications. Still, pacemaker implantation in children and adolescents is a procedure with a generally favorable outcome.

The indications for pacing in children and patients with CHD are slightly different than in adults, mainly reflecting the broad range of ages and concomitant structural heart disease involved.[4] The natural history of bradyarrhythmias in these palliated or repaired CHD patients and the specifics of the surgical approach are major determinants influencing the need for pacing.

In our series, a big proportion of patients (≈72%) had CHD and 50% of patients had postoperative CHB. These results are in line with the observations of other studies.[3,5]

Epicardial pacing is usually established because of either cardiac anatomy or small body size.[6,7] In our series, epicardial pacing was only limited to small children (< 12 kg body weight, < 0.3 m^2 BSA), single ventricle physiology, or early postoperative CHB. The smallest size for uncomplicated endocardial pacing was a body weight of 12 kg and BSA of 0.3 m^2 due to our concern about the development of venous thrombosis, which results from disproportion between vessel and lead size in smaller patients. Because of this consideration also, we used purely ventricular pacing mode in transvenous pacing in our series.

Despite recent technical progress, pacing leads remain the 'weakest link' of the permanent pacing system,[8] especially in a growing patient. Moreover, in the era of modern PPM technology, the reported complication rate in pediatric patients is still high being around 10–30%[2,3,9–11] compared to a 14% reported complication rate in adults.[12] Therefore, concerns are raised about the long-term efficacy and safety of endocardial and epicardial pacing leads in children in terms of high rate of lead abandonment.[13]

In our series the complication rate was 37.5% and half of which were lead related complications or failures that necessitated re-intervention and lead abandonment in the majority of cases. In lead related complications or failures, 83.3% were mechanical lead complications including lead dislodgement, fractures and insulation breaks. Lead failures occurred in 9.6% of endocardial leads and in 20% of epicardial leads. Other authors have described almost similar rates of lead failures.[1,3,5,14]

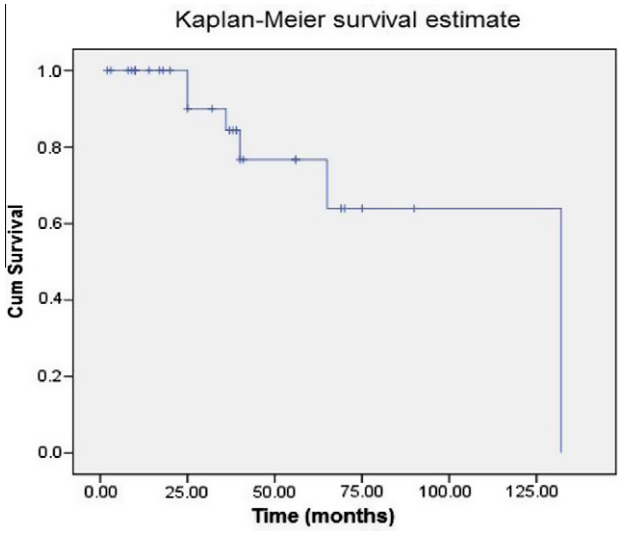

Figure 1 Cumulative survival of PPM batteries in overall study population.

The relatively high failure rate of epicardial leads is related not only to fracture due to somatic growth or traction imposed on epicardial leads by thoraco-abdominal movement but also to insulation break. Although all epicardial leads in our series were steroid eluting still a rather high failure rate was reported. The difference in failure rates between endocardial and epicardial leads did not reach statistical significance in our series. Although it almost matches the rates reported by other authors, this could be explained by the small total number of both endocardial and epicardial leads.

No endocardial lead insulation break or mal-sensing were detected in our experience whereas, insulation break and sensing mal-function were detected in 26.6% of epicardial leads. Non-lead related complications were only seen in endocardial devices; late skin erosions (at 2.92 ± 1.82 years follow up) had an incidence of 9.4% in our series compared to ~2% incidence reported by other authors,[3,5] which may be due to the small sample size of our study. Neither PPM pocket infection nor deaths were reported in our series.

During the period of follow up, patients with suboptimal pacing parameters when added to patients with pacing system failures represented 37.5% of our patient's population. When

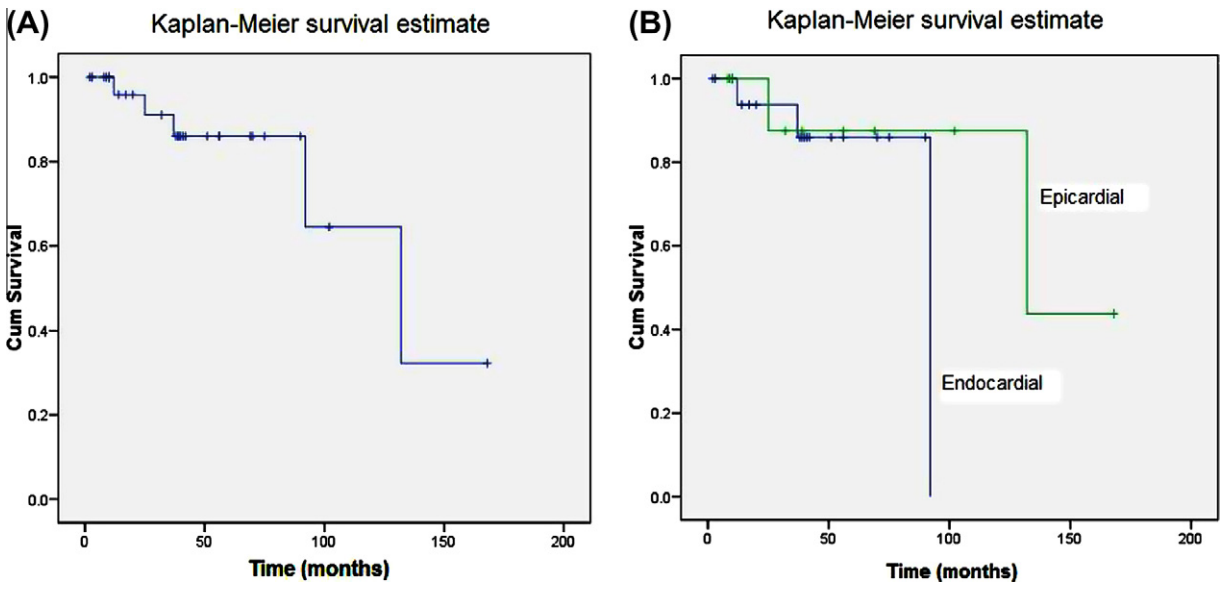

Figure 2 (A) Ventricular lead survival in overall study population, (B) Endocardial versus epicardial ventricular lead survival (log-rank test $\chi^2 = 0.76$, $p = 0.38$).

compared to patients with optimal pacing parameters and smooth follow up, only younger and smaller children had higher incidence of suboptimal pacing parameters or pacing system failures with no difference between endocardial and epicardial implants. This suggests that age and size of the child play a pivotal role in the long-term outcome of the device whether it is endocardial or epicardial.

At follow up, 15 patients were PPM dependent, the majority of whom (14 patients) had heart disease and two-thirds had post operative CHB. This may indicate that the presence of heart disease and operative trauma resulted in distal block that is associated with more pacing dependency.

In our series, the longevity of steroid eluting epicardial leads was comparable to that of endocardial leads which is different from what is reported by some other authors, who reported better survival of endocardial leads over epicardial leads.[3,5,10] This could be explained by the fact that the majority of epicardial leads used in these studies were non-steroid eluting leads, which were proved to have worse outcomes, and when they compared steroid eluting epicardial leads to conventional endocardial leads in one of these studies they showed comparable survival[3] as shown in our study.

From this study we have learnt that careful and complete follow-up evaluation is extremely useful, because the experience acquired and the knowledge of the complications that have occurred may help in the understanding of how to pace this population and help in new decision-making processes. The continuing technical innovations can make accumulated experience in a particular procedure quickly redundant and demand new experience perhaps on a better basis. This makes pediatric pacing more difficult, but also more interesting and challenging.

5. Limitations

The study is a single centre study and the number of patients is relatively small.

6. Conclusion

Permanent pacing in pediatric patients is generally safe and has a favorable long-term outcome, but there remains a high rate of complications, mainly related to leads. This is of particular concern in children who need a lifetime of pacing. With modern technology,[15] transvenous and epicardial pacing are initially comparable. In the older child or in the adolescent, endocardial pacing should be considered to be the first choice.

References

1. Cohen MI, Bush DM, Vetter VL, et al. Permanent epicardial pacing in pediatric patients. Seventeen years of experience and 1200 outpatient visits. *Circulation* 2001;**103**:2585–90.
2. Kammeraad JAE, Rosenthal E, Bostock J, Rogers J, et al. Endocardial pacemaker implantation in infants weighing < 10 k. *Pace* 2004;**27**:1466–74.
3. Silvetti MS, Drago F, Grutter G, et al. Twenty years of cardiac pacing in pediatric age: 515 pacemakers and 480 leads implanted in 292 patients. *Europace* 2006;**8**:530–6.
4. Epstein AE, DiMarco JP, Ellenbogen KA, et al. ACC/AHA/HRS: Guidelines for Device-Based Therapy of Cardiac Rhythm Abnormalities: a report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines (Writing Committee to Revise the ACC/AHA/NASPE 2002 Guideline Update for Implantation of Cardiac Pacemakers and Antiarrhythmia Devices): developed in collaboration with the American Association for Thoracic Surgery and Society of Thoracic Surgeons. *Circulation* 2008;**117**(21):E350–408 (2008).
5. Silvetti MS, Drago F, De Santis A, et al. Single-centre experience on endocardial and epicardial pacemaker system function in neonates and infants. *Europace* 2007;**9**:426–31.
6. Kerstjens-Fredrikse MWS, Bink-Boelkens MTE, de Jongste MJL, et al. Permanent cardiac pacing in children: morbidity and efficacy of follow-up. *Int J Cardiol* 1991;**33**:207–14.
7. Beaufort-Krol GC, Mulder H, Nagelkerke D, et al. Comparison of longevity, pacing, and sensing characteristics of steroid-eluting epicardial versus conventional endocardial pacing leads in children. *J Thorac Cardiovasc Surg* 1999;**117**(3):523–8.

8. De Voogt WG. Pacemaker leads: performance and progress. *Am J Cardiol* 1999;**83**:187D–91D.
9. Fortescue EB, Berul CI, Cecchin F, et al. Patient, procedural, and hardware factors associated with pacemaker lead failures in pediatrics and congenital heart disease. *Heart Rhythm* 2004;**1**:150–9.
10. Sachweh JS, Vazquez-Jimenez JF, Schondube FA, et al. Twenty years experience with pediatric pacing: epicardial and transvenous stimulation. *Eur J Cardiothorac Surg* 2000;**17**:455–61.
11. Udinkten Cate F, Breur J, Boramanand N, et al. Endocardial and epicardial steroid lead pacing in the neonatal and paediatric age group. *Heart* 2002;**88**:392–6.
12. Kiviniemi MS, Pirnes MA, Eranen HJK, et al. Complications related to permanent pacemaker therapy. *Pacing Clin Electrophysiol* 1999;**22**:711–20.
13. Lau YR, Gillette PC, Buckles DS, et al. Actuarial survival of trans-venous pacing leads in a pediatric population. *Pacing Clin Electrophysiol* 1993;**16**:1363–7.
14. Villain E, Martelli H, Bonnet D, et al. Characteristics and results of epicardial pacing in neonates and infants. *Pacing Clin Electrophysiol* 2000;**23**:2052–6.
15. Bauersfeld U, Nowak B, Molinari L, et al. Low-energy epicardial pacing in children: the benefit of autocapture. *Ann Thorac Surg* 1999;**68**:1380–3.