Case Report

Combined Epicardial and Transvenous Pacing in an Infant with Operated Complex Congenital Heart Disease and Permanent Epicardial DDD Pacemaker as Treatment of Dysfunction of the Epicardial Ventricular Lead

Sofia N. Loukopoulou, Nikolaos G. Eleftherakis, Antonios N. Kourtesis, Basilios D. Thanopoulos

Department of Paediatric Cardiology and 2nd Department of Paediatric Cardiac Surgery, "Aghia Sophia" Children's Hospital, Athens, Greece

Key words:
Endocarditis, native valves, pacemaker, prosthetic valves.

A male child, 18 months old, with interrupted aortic arch, ventricular septal defect, postoperative complete heart block and an epicardial DDD pacemaker since the age of nine months, was admitted to our department because of episodes of syncope. At first the episodes were considered as epilepsy and the child was given antiepileptic drugs. Twenty-four-hour ambulatory electrocardiographic monitoring revealed dysfunction of the pacemaker due to exit block in the ventricular lead, while the atrial lead was functioning properly. The young patient was treated by preservation of the epicardial atrial lead and implantation of the ventricular lead via the transvenous route. The ventricular lead was then connected through a subcutaneous channel to the pulse generator in an abdominal pocket.

Manuscript received:
February 4, 2008;
Accepted:
July 31, 2008.

Address:
Sofia Loukopoulou

"Aghia Sophia" Children's Hospital
4 Nap. Zerva St.
15341 Agia Paraskevi
Athens, Greece
e-mail:
sofialoukopoulou@hotmail.com

One of the postoperative complications of paediatric cardiac surgery is the appearance of complete atrioventricular block, due to damage to the atrioventricular electrical connection. This complication is more common in paediatric surgical procedures that involve the correction of congenital defects of the interventricular septum (ventricular septal defect, endocardial cushion defect) or other complex cardiac diseases (tetralogy of Fallot, corrected transposition of the great arteries).[1-3] In neonates and young infants pacemakers are implanted in such cases, with leads sutured to the epicardium and the generator placed in a pouch created below the peritonea of the *rectus abdominis* muscle. A disadvantage of the method is the development of fibrosis on the leads.

Case presentation

We describe the case of an infant aged 18 months, with complex congenital heart disease (type B interrupted aortic arch, ventricular septal defect), who was admitted to our department because of syncopal episodes. The infant had had an epicardial DDD pacemaker (Figure 1) implanted at age 9 months because, after repeated surgical procedures for repair of the congenital disease, he exhibited complete atrioventricular block. The parents reported a history of tonic spasms of the upper limbs with pallor lasting 3-4 minutes, upward oculogyration and perioral cyanosis during the previous 2-3 weeks (8 months after pacemaker implantation). Prior to that the infant was free of symptoms and under treatment with diuretics and captopril.

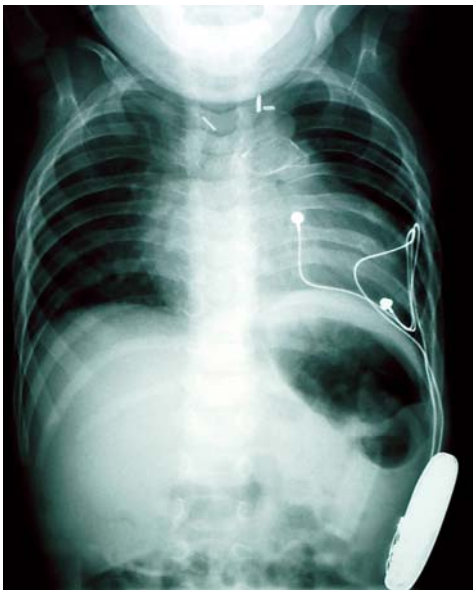

Figure 1. Chest and abdominal X-ray of the infant on his admission for syncopal episodes. There is no mechanical damage to the pacing leads.

Clinical examination showed a systolic murmur 1/6-2/6 at the left sternal margin. Femoral pulses were symmetrically palpable. The infant was adequately nourished. His psychomotor development was 4-5 months behind his chronological age.

The ECG showed pacing rhythm with a ventricular rate of 120 min^{-1}.

The echocardiographic examination showed good left ventricular contractility with normal internal dimensions. There was no residual flow through the closed ventricular septal defect. In addition, the flow rate in the aortic arch and descending aorta was within normal limits.

Initially, the episodes were treated as epileptic spasms and the infant was put on antiepileptic medication. His sleep electroencephalogram was well organised, without paroxysmal phenomena. Brain computed tomography also found no focal lesions, only a benign widening of the subarachnoid region. The patient underwent 24-hour Holter monitoring, which revealed pacemaker malfunction (Figures 2, 3).

During the pacemaker check a significant increase was observed in the resistance and pacing threshold of the ventricular lead. Resistance had increased from 285 Ω at a check up 1 month before the syncopal episodes to 4000 Ω on the latest examination. Pacing threshold

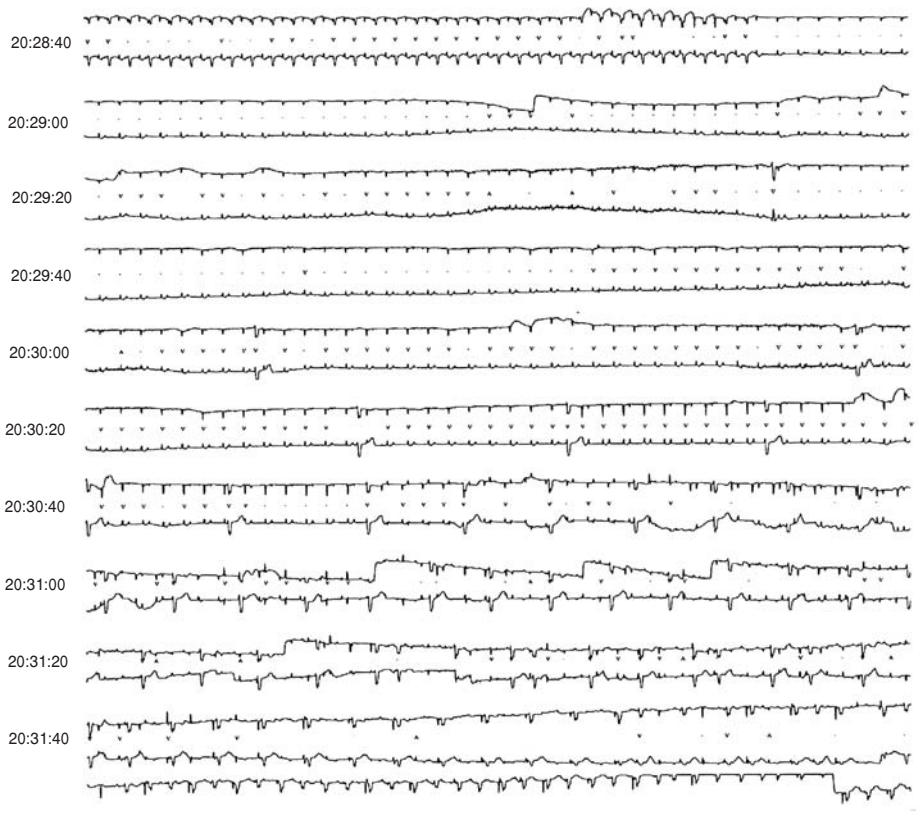

Figure 2. Holter recording during a syncopal episode showing prolonged pacemaker malfunction.

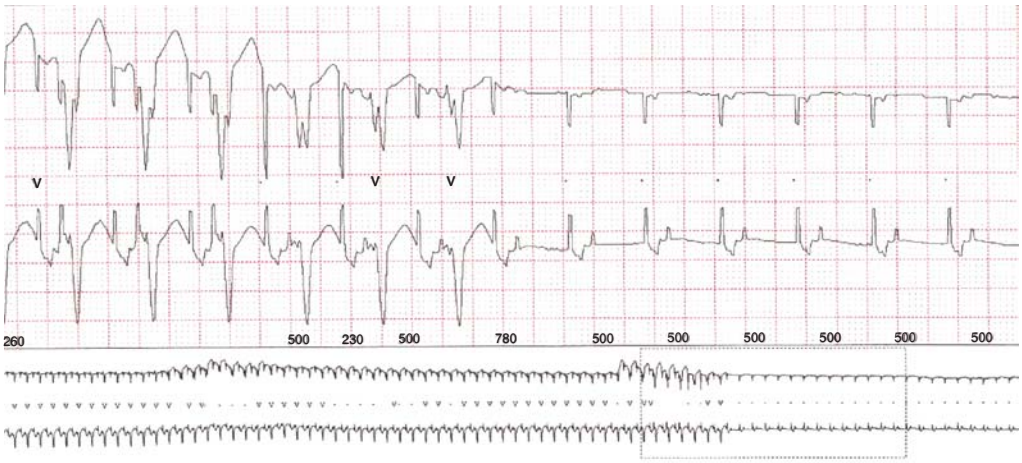

Figure 3. Exit block of the ventricular lead, while the atrial lead functions normally.

had increased from 2.8 V to 4.5 V at 0.5 m/s. In contrast, the atrial lead showed no malfunction, with the resistance increasing from 285 Ω to 294 Ω and the threshold remaining constant at 0.5 V.

In view of these findings, an transvenous ventricular lead was implanted, while the atrial lead was left in place epicardially, as was the pacing generator, which was placed in the existing abdominal pouch after connection with the new ventricular lead (Figure 4). The ventricular lead was introduced through the left subclavian vein and was connected with the generator via a subcutaneous channel that was created from the left subclavian vein to the abdominal pacemaker pouch. Pacemaker function was restored and the infant remains free of symptoms 22 months after the procedure.

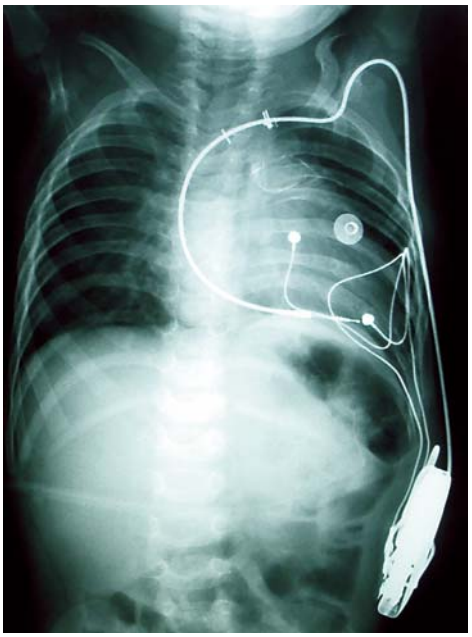

Figure 4. Chest and abdominal X-ray of the infant after placement of the transvenous ventricular electrode.

Discussion

It is well known that in both infants and older children permanent pacemaker leads may be implanted either epicardially or transvenously. In small infants, when permanent pacemaker implantation is necessary epicardial leads are used. The reason for preferring the epicardial route is the patient's small body size. Transvenous lead implantation is hampered by anatomical peculiarities, which are often seen and include anomalous connection of the *venae cavae* as well as other complex endocardial anatomical lesions.[4] Apart from the procedural difficulties, such anomalies also entail a risk of systemic embolism due to endocardial defects.[5] In addition, thrombosis in the superior *vena cava* is a common complication of endocardial lead placement.[6] Thus the transvenous route has lost favour except for possible future lead implantation at a greater age.[7] On the other hand, epicardial lead placement is more invasive. It involves subxiphoid section and possibly a partial sternotomy or thoracotomy. It is often complicated by post-pericardiotomy syndrome.[8]

The usual epicardial leads are associated with a high incidence of rapidly increasing sensing and pacing thresholds after lead placement, necessitating the early

replacement of lead and generator. Recent studies have shown that steroid-eluting epicardial leads appear to have encouraging results at mid-term follow up[9,10] and over long-term follow up they behave like conventional endocardial leads.[11] Endocardial leads are favoured in older infants with a body weight >8 kg,[12] or preferably 15-20 kg,[13] in order to avoid vascular damage and thrombosis as well as lead dysfunction from excessive strain caused by the infant's development. Moreover, in small infants the small dimensions of the atrium[13] are insufficient for successful placement of the preformed atrial lead.

The elevated pacing thresholds and the high incidence of exit block associated with conventional epicardial leads are caused by a combination of epicardial fibrosis with scar formation, and/or pericardial adhesions following the surgical procedure. Cases have been reported of exit block due to lead fracture caused by the infant's muscular activity.[14] The five-year survival of the conventional epicardial lead is 40-70%.[15,16] Five-year survival of a steroid-eluting epicardial lead is 74%,[11] comparable with conventional endocardial leads[17,18] and modern, thin, transvenous leads.[19] As regards steroid-eluting atrial leads, no improvement has been seen in pacing threshold compared to conventional leads. This is attributed to exaggerated scarring of the atrium that is not affected by the simple addition of dexamethasone.[11]

In all paediatric cardiac surgical procedures leads are placed for temporary epicardial pacing. These can safely be removed if no disturbances of atrioventricular conduction are manifested during the first 24 hours after surgery.[20] If atrioventricular block is observed and persists for more than 14 days postoperatively, permanent pacing is indicated.[1-3]

In neonates and infants with a permanent pacemaker the occurrence of episodes of loss of consciousness may be due to pacemaker malfunction.[21] Follow-up checks should be performed every 6 months in those without symptoms; parameters of pacemaker function should be measured, mainly the resistance and threshold of the atrial and ventricular leads. If symptoms occur as a result of pacemaker malfunction, 24-hour Holter monitoring is useful for their detection.[23,24]

Pacemaker malfunction due to the development of fibrosis around one or both of the epicardial leads can be treated by the substitution of endocardial leads. At the same time, the generator can be left in its abdominal site and the electrodes can be connected via a subcutaneous channel.

In conclusion, the choice of lead type during pacemaker implantation should aim at achieving optimum cardiac function and maximum battery and lead life, while taking account of the risks of lead placement as well as the future surgical treatment of the patient.

References

1. Kratz JM, Gillette PC, Crawford FA, et al. Atrioventricular pacing in congenital heart disease. Ann Thorac Surg. 1992; 54: 485-489.
2. Weindling SN, Saul JP, Gamble WJ, Mayer JE, Wessel D, Walsh EP. Duration of complete atrioventricular block after congenital heart disease surgery. Am J Cardiol. 1998; 82: 525-527.
3. Kertesz N, McQuinn T, Collins E, et al. Surgical atrioventricular block in 888 heart operations. New implications for early implantation of a permanent pacemaker. Pacing Clin Electrophysiol. 1996; 19: 613.
4. Udink ten Cate F, Breur J, Boramanand N, et al. Endocardial and epicardial steroid lead pacing in the neonatal and pediatric age group. Heart. 2002; 88: 392-396.
5. Noiseaux N, Khairy P, Fournier A, Vobecky SJ. Thirty years of experience with epicardial pacing in children. Cardiol Young. 2004; 14: 512-519.
6. Aellig NC, Balmer C, Dodge-Khatami A, Rahn M, Prêtre R, Bauersfeld U. Long-term follow-up after pacemaker implantation in neonates and infants. Ann Thorac Surg 2007: 83; 1420-1423.
7. Beaufort-Krol GC, Mulder H, Nagelkerke D, Waterbolk TW, Bink-Boelkens MT. Comparison of longevity, pacing and sensing characteristics of steroid eluting epicardial versus conventional endocardial pacing leads in children. J Thorac Cardiovasc Surg. 1999; 117: 523-528.
8. Gillette PC, Shannon C, Blair H, et al. Transvenous pacing in pediatric patients. Am Heart J. 1983; 10: 843-847.
9. Hamilton R, Gow R, Bahoric B, et al. Steroid-eluting epicardial leads in pediatrics: improved epicardial thresholds in the first year. Pacing Clin Electrophysiol. 1991; 14: 2066-2072.
10. Johns JA, Fish FA, Burger JD, Hammon JW Jr. Steroid-eluting epicardial pacing leads in pediatric patients: encouraging early results. J Am Coll Cardial. 1992; 20: 395-401.
11. Cohen MI, Bush DM, Vetter VL, et al. Permanent epicardial pacing in pediatric patients: seventeen years of experience and 1,200 outpatient visits. Circulation. 2001; 103: 2585-2590.
12. Silveti MS, Drago E. Upgrade of single chamber pacemakers with transvenous leads to dual chamber pacemaker in pediatric and young adult patients. Pacing Clin Electrophysiol. 2004; 27: 1094-1098.
13. Silvetti MS, Drago F, Grutter G, et al. Twenty years of paediatric cardiac pacing: 515 pacemakers and 480 leads implanted in 292 patients. Europace. 2006; 8: 530-536.
14. Bakhtiary F, Dzemali O, Bastanier CK, et al. Medium-term follow-up and modes of failure following epicardial pacemaker implantation in young children. Europace. 2007; 9: 94-97.
15. Sachweh JS, Vazquez-Jimenec JF, Schöndube FA, et al. Twenty years experience with pediatric pacing: epicardial and transvenous stimulation. Eur J Cardiothorac. Surg. 2000; 17: 455-461.
16. Kratz JM, Gillette PC, Crawford FA, et al. Atrioventricular pacing in congenital heart disease. Ann Thorac Surg. 1992; 54: 485-489.

17. Lan YR, Gillette PC, Buckles DS, et al. Actuarial survival of transvenous pacing leads in a pediatric population. Pacing Clin Electrophysiol. 1993; 16: 1363-1367.
18. Esperer HD, Sirger LL, Riede FT, et al. Permanent epicardial and transvenous single and dual chamber cardiac pacing in children. Thorac Cardiovasc Surgeon. 1993; 41: 21-27.
19. Fortescue EB, Berul CI, Cecchin F, et al. Comparison of modern steroid eluting epicardial and thin transvenous pacemaker leads in pediatric and congenital heart disease patients. J Interv Card Electrophysiol. 2005; 14: 27-36.
20. Jowett V, Hayes N, Sridharan S, et al. Timing of removal of pacing wires following paediatric cardiac surgery. Cardiol Young. 2007; 17: 512-516.
21. Serner GA, Dorostkar PC. Paediatric Pacing. In: Ellenbogen KA, Kay GN, Wilkott BL, editors. Clinical Cardiac Pacing. Philadelphia: WB Saunders; 1995. p. 706-734.
22. Gillette PC, Heinle JS, Zeigler VL. Cardiac Pacing. In: Gillette PC, Garson A Jr, editors. Clinical Pediatric Arrhythmias. Philadelphia: WB Saunders; 1999. p. 190-220.
23. Strathmore NF, Nond HG. Noninvasive monitoring and testing of pacemaker function. Pacing Clin Electophysiol. 1987; 10: 1030-1035.
24. Janosik DL, Redd RM, Buckingham TA, et al. Utility of ambulatory electrocardiography in detecting dysfunction in the early postimplantation period. Am J Cardiol. 1987; 60: 1030-1035.