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Brief Report

Emergency trans-oesophageal ventricular pacing in a child

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Abstract We report our experience with an 8-year-old boy with complete atrioventricular block and syncopal bradycardia who required urgent pacing. Each attempt to cross the tricuspid valve with a femoral lead triggered ventricular standstill, followed by fibrillation, and pacing through the coronary sinus failed. Successful ventricular pacing was finally achieved through the oesophagus, allowing subsequent implantation of a transvenous pacemaker.

Keywords: Oesophageal ventricular pacing; atrioventricular block

EMERGENCY PACING THROUGH A FEMORAL LEAD may sometimes be needed prior to implantation of a pacemaker. We report our experience with a young boy with complete atrioventricular block and prolonged ventricular standstill, in whom emergency trans-oesophageal ventricular pacing was successfully performed after failure of the transvenous approach. This technique may be life saving when it is not possible immediately to implant a pacemaker transvenously.

Case report

An 8-year-old boy with no previous medical or family history was brought to the emergency room for repeated syncope occurring over a period of 40 h. There was no other symptom, and clinical examination was normal except for bradycardia. The 12-lead electrocardiogram showed complete atrioventricular block, with a ventricular rate of 35 beats/min (Fig. 1) and repeated ventricular pauses. The QT interval was normal. Echocardiography showed normal cardiac structures and left ventricular contraction.

The patient was immediately taken to the catheterisation laboratory in order to achieve right ventricular pacing. The right atrium was easily reached, but three attempts to cross the tricuspid valve with a femoral pacing lead triggered prolonged ventricular standstill, followed by torsade de pointes and ventricular fibrillation (Fig. 2a), requiring three episodes of cardiac resuscitation and cardioversion using a current of 30 J. The child was resuscitated, and received intravenous amiodarone given at a dose of 1 mg/kg over 1 min, followed by a second dose of 4 mg/kg over 5 min, but another attempt to introduce the lead into the right ventricle was again followed by ventricular fibrillation and cardioversion. An infusion of 1 mg/kg of lidocaine also failed to prevent a further episode of ventricular fibrillation requiring a 50 J electric shock. Stimulation through the coronary sinus and the coronary veins did not capture the left ventricle, despite high outputs, and steady temporary external pacing could not be achieved.

Finally, we introduced an oesophageal bipolar pacing catheter (Esosoft 2S, Fiab®) through the nose of the child, and positioned it so that the maximal ventricular potential was obtained. Pacing using a Fiab 2007® was begun at a rate of 80 beats/min, and the amplitude was increased until ventricular capture was obtained with an energy of 40 mA, and a pulse width duration of 20 ms (Fig. 2b). After 10 min of stable trans-oesophageal ventricular pacing, the femoral lead was advanced across the tricuspid valve and positioned in the right ventricular apex without any ventricular dysrhythmia. Temporary transvenous ventricular pacing (Fig. 2c) was achieved with a low threshold, and allowed uneventful implantation of a dual chamber permanent pacemaker. Subsequent to implantation, the pacing threshold was 0.5 V with a pulse width of

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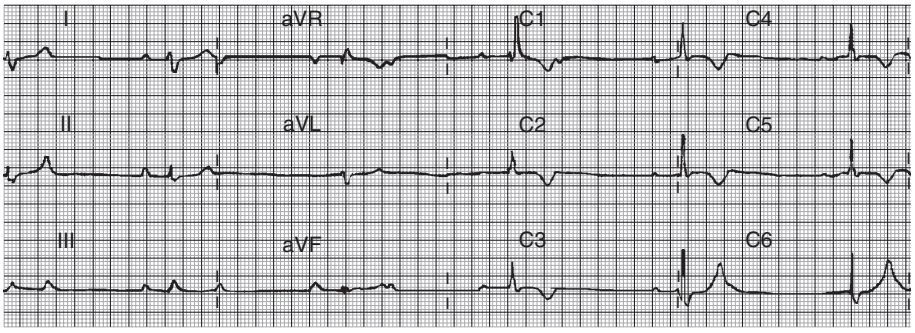

Figure 1.
The 12-lead electrocardiogram of the patient at admission, showing complete atrioventricular block.

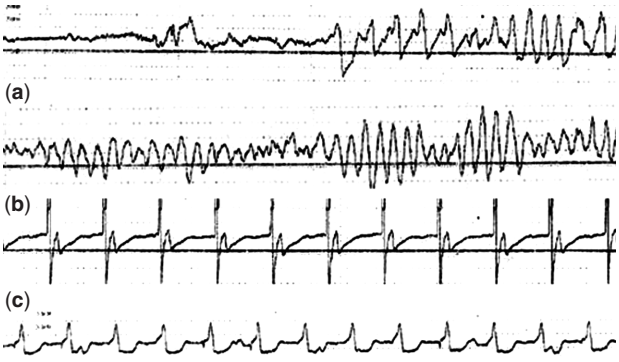

Figure 2.
*We recorded electrocardiographic tracings successively during the period of catheterisation and implantation of the pacemaker. The selected strips show (**a**) a long pause followed by prolonged torsade de pointes, (**b**) trans-oesophageal ventricular pacing, and (**c**) transvenous ventricular pacing.*

0.5 ms, and P wave detection was 3 mV. One year after implantation, the patient is doing well, echocardiography is normal, but transient inhibition of the pacemaker shows permanent complete atrioventricular block, with a low escape rhythm. A maternal immune pathology was excluded, the family has a normal electrocardiogram, and no infectious aetiology has been found. Routine biochemical tests at the time of hospitalisation were normal.

Discussion

Trans-oesophageal pacing usually provides a safe and relatively non-invasive method for pacing the atriums without fluoroscopy.[1] As the ventricular mass is not in close relation with the site of stimulation, variable success has been reported for ventricular pacing,[1,2] and oesophageal pacing has been thought to be of limited value in the investigation of patients with paroxysmal ventricular tachycardia.[2,3] The use of the trans-oesophageal approach for emergency ventricular pacing, however, has been reported in adults,[4] and also in one infant of which we are aware.[5] In our case, this approach permitted us to pace the heart without entering the right ventricle, as each previous attempt to cross the tricuspid valve had mechanically triggered a long ventricular standstill followed by torsade de pointes. In typical sequences of "pause-dependent" torsade de pointes, the first paced or spontaneous ventricular beat may induce ventricular arrhythmias because the pause destabilises and lengthens the ventricular repolarisation. Longer pauses are followed by longer and faster runs of torsade de pointes.[6] In most cases, torsade de pointes is not sustained, but in our patient, it degenerated into ventricular fibrillation, and required cardioversion. Fast transvenous pacing is the treatment of choice for bradycardia-induced torsade de pointes, but in our case, it was in itself dysrhythmogenic. In such very rare cases, and when transvenous pacing is not immediately available, we suggest that trans-oesophageal ventricular pacing may be life saving.

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