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Brief Report

Cardiac resynchronization as therapy for congestive cardiac failure in children dependent on chronic cardiac pacing

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Abstract Three patients with heart failure after chronic right ventricular apical pacing were treated with resynchronization. Biventricular pacing was used for two patients, and the other was treated with left univentricular pacing. In all patients, we observed a dramatic improvement of left ventricular dimension, function, and clinical state. We conclude that biventricular or left ventricular pacing is superior to right ventricular apical pacing in children who are pacemaker-dependent.

Keywords: Resynchronization therapy; chronic pacing; cardiac failure; lead position

CARDIAC RESYNCHRONIZATION THERAPY HAS proven its value in adults with congestive heart failure, an ejection fraction of 35% or less, and a QRS interval of 130 milliseconds or more.[1] Exercise tolerance and quality of life improve significantly. Despite several studies regarding the acute effects of pacing in cardiac postoperative children, little information exists on the long-term effects. We report here our experiences in three patients with chronic right-sided double chamber atrial and ventricular pacing who developed heart failure, and were treated with resynchronization therapy with either biventricular or left ventricle pacing.

Case reports

Our first patient is a 1½-year-old girl with congenital atrioventricular block. She received a double chamber pacemaker shortly after birth, with a right atrial and right ventricular apical lead. One year after implantation, her condition was worsening, and she was hardly growing (Fig. 1). She was treated with diuretics, inhibition of angiotensin converting enzyme, and digoxin. Despite this treatment, her condition stayed very poor. Her heart progressively dilated, reaching an end-diastolic diameter of 46 millimetres, the normal maximal value being 30 millimetres, and an ejection fraction of 18%. An additional left ventricular epicardial lead was placed on the left ventricular apex, and she received a biventricular pacemaker. After 2 months pacing, her condition already improved dramatically. Her weight returned to normal, left ventricular dimension decreased, and ejection fraction increased significantly (Fig. 1)

The second patient is a 13-year-old girl who developed complete atrioventricular block after closure of a ventricular septal defect when she was 1-year-old. She received a double chamber pacemaker, with an epicardial right atrial and epicardial right ventricular lead. For the next 8 years, she was in a good condition, but 9 years after implantation, she developed progressive signs of heart failure. Her heart was dilated, and ejection fraction was 21%. In spite of additional pharmacological treatment, she developed progressive heart failure, and she had to move around in a wheelchair. All possible causes of a cardiomyopathy were excluded. After upgrading to a biventricular pacemaker, with a left ventricular lead positioned high and posterolaterally, and a new right ventricular lead in the right ventricular outflow tract, her left ventricular diameter decreased and ejection fraction increased (Table 1). One year after implantation she is walking and riding her bike again.

The third patient is a 1½-year-old boy with discordant ventriculo-arterial connections who developed Mobitz II atrioventricular block after an arterial

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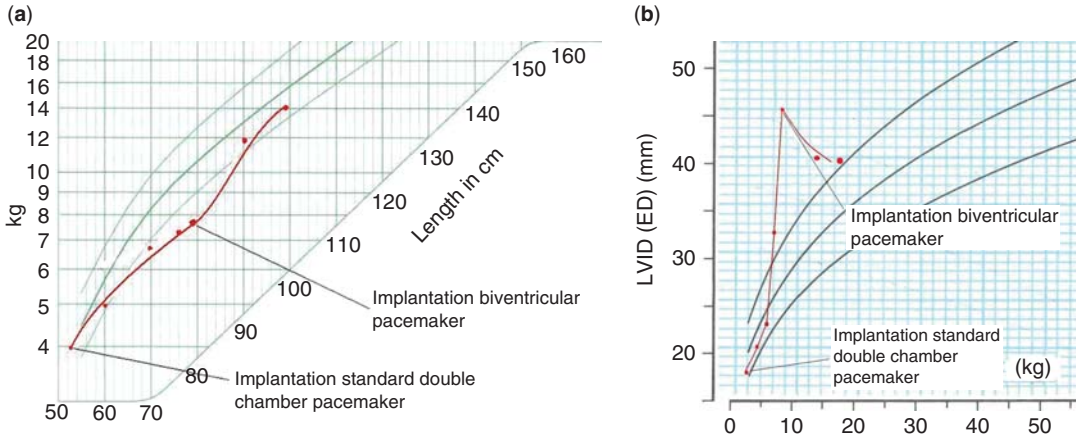

Figure 1.
Growth curve and left ventricular end diastolic diameter of our first patient before and after implantation of a biventricular pacemaker with epicardial leads on right atrium, right ventricle end left ventricle. Abbreviations: LVID(ED): left ventricular internal diameter (end diastolic).

Table 1. Variables before and after resynchronization in our three patients.

	Age (years)		
	1½	13	1½
Male/Female	Female	Female	Male
Diagnosis	Congenital AV-block	VSD closure	TGA, VSD, arterial switch
Conduction	Complete AV-block	Complete AV-block	Mobitz II AV-block
Conventional pacemaker	Yes	Yes, DDD	Yes, VVIR
Weight (kg)	6.9	33	9.5
Length (cm)	77	137	?
Clinical status before	Very poor	Poor	Poor
Clinical status after	Very good	Good	Good
Medication before	Aspirin, digoxin, diuretics, ACE-inhibition	Aspirin, digoxin, diuretics, ACE-inhibition	Aspirin ACE-inhibition
Medication after	Aspirin, ACE-inhibition, beta-blocker	Aspirin, digoxin, diuretics, ACE-inhibition, beta-blocker	Aspirin, ACE-inhibition, beta-blocker
LVEDD before (mm)	46	70	45
LVEDD after (mm)	39	52	35
EF before (%)	18	21	19
EF after (%)	50	42	64
QRS width before (ms)	160	200	180
QRS width after (ms)	100	160	140
Pacing site	RA, RV, LV	RA, RV, LV	LA, LV
Lead position	Epicardial	Endocardial + epicardial	Epicardial
Follow-up (months)	25	35	21

Abbreviations: AV-block: atrioventricular block; VSD: ventricular septal defect; TGA: discordant ventriculoarterial connections; ACE: angiotensin converting enzyme; LVEDD: left ventricular end diastolic diameter; EF: ejection fraction; RA: right atrium; RV: right ventricle; LA: left atrium; LV: left ventricle

switch operation. One month after birth, he received a univentricular pacemaker, with the epicardial lead positioned at the right ventricular apex. One year later, his condition was very poor. His left ventricle was dilated, with an end-diastolic diameter of 45 millimetres, and ejection fraction was 19%. He received a double-chamber pacemaker, with a left atrial and a left ventricular epicardial lead on the mid-anterior wall. His condition, left ventricular dimension, and QRS width gradually improved, and were almost normal 1 year after replacement of the pacemaker (Table 1).

Discussion

In young adults with a complete congenital atrioventricular block, chronic transvenous dual chamber pacing of the right atrium and right ventricle can dramatically change the cardiac function. Dilation of the left ventricle and remodelling is seen, with

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hypotrophy in the early-activated segments, and hypertrophy in the late-activated segments. Furthermore, there is dyssynchrony of the myocardial contraction. The systolic and diastolic cardiac function decreases, and therefore the cardiac output and exercise tolerance decreases.[2] Recent cases of two children also show that the dyssynchrony associated with right ventricular pacing is at least partly responsible for the severe dilation of the left ventricle and heart failure. Furthermore, this seems to be reversible, with resynchronisation of the contraction by biventricular pacing or spontaneous junctional narrow QRS escape rhythm.[3]

In our three patients, the left ventricular function had deteriorated to such a degree that they all developed signs of progressive cardiac failure despite optimal medical therapy. Assessment of dyssynchrony was performed by M-mode and cross-sectional; echocardiography, and justified resynchronisation. In two patients, we used a biventricular configuration, employing a bifocal configuration in the other patient, with the epicardial atrial lead at the left atrium, and the ventricular lead at the left posterolateral region.

In the two youngest patients, the left ventricular leads were positioned via a left lateral thoracotomy. In these patients, the optimal lead position and pacing configuration was defined by optimizing the synchrony of the left ventricle with per-operative transoesophageal echocardiographic guidance in short and long axis views, along with electrocardiographic monitoring. The oldest patient received her left ventricular lead transvenously, and optimal positioning and timing were defined by transthoracic echocardiography in cross-sectional and motion modes together with electrocardiographic monitoring. During follow-up, QRS duration and ventricular synchrony improved in all three patients as judged by echocardiography. More importantly, the clinical state improved dramatically (Table 1). Cardiac resynchronization, therefore, seems an effective therapy for congestive cardiac failure in children.

Vanagt et al.[4] recently studied the optimal site for pacing in animals, and in eight children undergoing cardiac surgery. They found that pacing from the right ventricular apex or left ventricular free wall both resulted in an asynchronic activation, producing a significant decrease in haemodynamic values compared to sinus rhythm and pacing at the left ventricular apex. Another recent review[5] also showed that other sites of pacing the right ventricle, such as the septum, outflow tract, or bundle of His, can potentially prevent systolic and diastolic dysfunction. In our opinion, individual definition of the optimal site for pacing is necessary, but at least it is recommended to position the ventricular lead in the left ventricle, or sites other than the apex of the right ventricle, in children depending on chronic ventricular pacing. We submit that our experience shows that resynchronization must be considered when treating children who develop cardiac failure after chronic right ventricular pacing.

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