Selecting pacing sites in children with complete heart block: is it time to avoid the right ventricular free wall?

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This editorial refers to ‘Predictors of left ventricular remodelling and failure in right ventricular pacing in the young’, by R.A. Gebauer et al., on page 1097

Patients with congenital or acquired complete heart block (CHB) require chronic right ventricular (RV) pacing when bradycardia causes symptoms or exercise intolerance. Indications for pacing are summarized in the American College of Cardiology/American Heart Association Task Force Report.1 Patient age and individual centre preference will determine whether an epicardial or endocardial pacing system is placed. Epicardial leads are usually placed on the RV free wall, which is readily exposed at surgery, while endocardial leads are usually implanted at the interventricular septum, or, more commonly, close to the RV apex. However, electrical stimulation of the heart from the RV apex and especially from the RV anterior wall differs significantly from normal electrical activation, which originates at the apical interventricular septum and propagates to the basal septal and lateral left ventricular (LV) walls. Asynchronous electrical activation may lead to changes in cardiac structure and function. In adults, there are considerable data to suggest that electromechanical dyssynchrony associated with chronic RV apical pacing may result in asymptomatic unfavourable LV remodelling and dysfunction in up to 50% of patients and ultimately to LV failure in ~10% of patients.2–4 Data pertaining to children are far more limited. Tantengco et al.5 found that LV function, measured by LV fractional area change and the myocardial performance index, was reduced in asymptomatic unfavourable LV remodelling following chronic RV pacing in children.9 All patients with a systemic left ventricle and biventricular circulation who underwent RV pacing at the Heart Centre in Prague, and in whom serial echocardiographic data were available, were included. This retrospective review used M-mode measurements for evaluation of LV function and remodelling. Despite these important limitations, the study presents several interesting findings. The first is a higher incidence than previously reported of unfavourable LV remodelling and LV dysfunction (13.4%). This may be partially attributable to the relatively high number of post-operative patients as well as to the exclusion of nine patients from the study because of lack of follow-up data. This finding, which is similar to those of previous adult studies, demonstrates that development of LV dilatation and dysfunction following RV pacing are not rare events. The implication of this is that all pacemaker patients require serial echocardiographic evaluation for timely detection of unfavourable remodelling. Interestingly, unfavourable remodelling was more common in patients with surgical heart block than congenital CHB, indicating perhaps that patients with CHB in the setting of congenital heart disease are at higher risk for developing LV dysfunction. Conversely, congenital atrioventricular (AV) block may not lead to unfavourable LV remodelling. This further supports the late-onset ‘cardiomyopathy’, which occurs in ~10% of patients with congenital AV block, may be related to RV pacing rather than to inflammatory-mediated myocardial damage.10,11

Perhaps more important is the finding that RV free wall pacing was strongly associated with unfavourable LV remodelling and development of LV dysfunction. Patients paced from the RV free wall were at significantly higher risk of developing LV dilatation and dysfunction as compared with patients paced from the RV

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apex or interventricular septum. In contrast, favourable reverse remodelling was observed in those patients upgraded to biventricular pacing. This is the first time that chronic RV free wall pacing has been shown to be associated with a higher risk of development of LV dysfunction in children. Unlike previous studies, RV apical pacing was not a significant risk factor for LV dysfunction, while RV septal pacing seemed to be protective as none of the children with RV septal pacing developed LV dysfunction. Based on these results, it would seem preferable to avoid epicardial pacing from the RV free wall. With endocardial pacing, this study, as well as previous adult studies,12 support RV septal pacing as the preferred pacing mode, in that none of the children with RV septal pacing developed LV dysfunction. The site of pacing can significantly influence haemodynamics, and, although RV apical or outflow pacing may not acutely affect ventricular haemodynamics in children with normal ventricular function, despite induction of electromechanical dyssynchrony,13 acute studies in children early after cardiac surgery have suggested that LV apical or biventricular pacing result in better haemodynamics compared with RV pacing.14,15 It should be noted, however, that acute haemodynamics during pacing may not necessarily predict long-term outcomes.

The study by Gebauer et al. has important limitations related predominantly to its retrospective nature. Furthermore, the echocardiographic data are limited and do not provide insight into the mechanism or degree of electromechanical dyssynchrony caused by the various pacing modes. Despite the recent limitations found by the PROSPECT study16 in using echocardiographic indices to define electromechanical dyssynchrony, it would be interesting to compare electromechanical dyssynchrony caused by different pacing modes, as this may be related to unfavourable haemodynamics and the risk of unfavourable outcome.

In this respect, defining echocardiographic criteria to assess dyssynchrony which may predict unfavourable remodelling will be important for echocardiographic guidance of optimal pacing site selection.16 The QRS duration is not a reliable index of mechanical dyssynchrony and does not predict pacemaker-related ventricular dysfunction. In the current study, there was only a small mean difference and significant overlap in QRS duration between patients who developed LV dysfunction vs. those who did not. It remains to be shown which pattern of electrical activation is more likely to cause LV dysfunction. This may have more to do with the activation sequence than with QRS duration alone. Further research, using modern echocardiographic techniques, may potentially identify the underlying mechanisms explaining electromechanical dyssynchrony and help to predict the development of pacemaker-related ventricular dysfunction.

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References