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The development of pacing induced ventricular dysfunction is influenced by the underlying structural heart defect in children with congenital heart disease

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ABSTRACT

Background: Right ventricular pacing can cause pacing-induced ventricular dysfunction (PIVD) correctable with biventricular pacing (BiVP). Factors associated with PIVD are poorly understood.
Methods: We reviewed children receiving epicardial dual-chamber pacemakers for complete heart block (CHB) after congenital heart disease (CHD) surgery. PIVD was defined as% fractional shortening <15% improving after BiVP.
Results: Between 2005 and 2014, 47 children <2 years developed CHB after CHD surgery. All had biventricular hearts and underwent epicardial dual chamber pacemaker implantation. Nine of the 47 (19%) developed PIVD. PIVD occurred in 0/10 with ventricular septal defect (VSD), 0/6 with tetralogy of Fallot, 2/6 with double outlet right ventricle, 2/6 with transposition and VSD, 3/9 with atrioventricular canal defect, 1/2 with mitral valve replacement; 1/3 with congenitally corrected TGA repair; and 0/3 with atrioventricular canal plus tetralogy of Fallot and 0/1 with subaortic membrane. QRS duration (QRSD) was 84–170 (median 135 ms) in the non PIVD group and 100–168 (median 124) ms in the PIVD group. Percentage fractional shortening (%FS) while paced was 16–46, median 30% in the non-PIVD group and 6–15 (median 11%) in the PIVD group.%FS post upgrade to BiVP (with an epicardial LV lead) in the 9 patients with PIVD was 23–33 (median 29%).
Conclusions: PIVD occurred in certain CHD but not others. Prolonged QRSD was not associated with PIVD. The predilection for RV pacing to result in PIVD in certain types of CHD needs further study.

1. Introduction

Conventional right ventricular (RV) pacing has been shown to cause pacing-induced ventricular dysfunction (PIVD) in some patients.[1] The cause of PIVD is thought to be pacing induced dyssynchrony and treatment with biventricular pacing has been shown to reverse the PIVD in most patients.[2,3] Complete heart block (CHB) is a potential complication of surgery for congenital heart disease (CHD) and the accepted management for patients with post-surgical CHB is to implant a pacemaker either via the epicardial (in infants and small children) or endocardial (in older children) approach.[4] Children with post-surgical CHB after repair of their CHD have a significant risk of developing PIVD if paced from the RV.[2,3,5] Studies have suggested that PIVD is less likely to occur if the initial pacing employed is CRT or LV pacing rather than RV pacing.[6] It is unclear whether the tendency to develop PIVD from pacing the RV differs between various forms of CHD.

We studied the incidence of PIVD after placement of a conventional dual chamber epicardial pacemaker with right atrial and right ventricular (RV) leads in a cohort of children who developed CHB after surgery for their CHD. This is a report of our findings.

2. Methods

After obtaining institutional ethical committee approval at our institutions, we performed a search of our surgical database to identify children <2 years of age with CHB noted immediately after heart surgery. The medical charts of all such patients were retrospectively reviewed for pertinent information. Patients were excluded if they had a pacemaker with RV lead prior to their heart

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surgery or were paced transiently for CHB after heart surgery but recovered AV nodal conduction and did not receive a permanent pacemaker.

The specific information reviewed included the clinical notes, chest Xray, electrocardiograms (ECG) and echocardiograms. PIVD was defined as% fractional shortening (%FS) of the left ventricle on standard M-mode echocardiography of <15% which improved by at least 5% after upgrade to a biventricular pacemaker (BiVP). An% FS of <15% was considered severe severely depressed function, >16% and <22% was regarded as moderate dysfunction, and >23% and <28% as mild dysfunction.

2.1. Statistical Analysis:

All values are expressed as minimum to maximum with median. Values pre and post biventricular pacing were compared using paired *T* testing.

3. Results

Between 2005 and 2014, 5081 children underwent cardiac surgery and, in 3732, the operations were open heart surgeries with cardiopulmonary bypass at the two institutions. Of them, 47 children developed CHB after CHD surgery. All were <2 years of age (5 days–7 months, median 3.5 months in the PIVD group and 7 days–23 months, median 5.5 months in the non-PIVD group) and all had biventricular forms of CHD. All underwent epicardial pacemaker placement with right atrial and ventricular leads through a xiphoid approach (Fig. 1 a& b). Nine of the 47 (21%) developed PIVD. PIVD was noted in 0/10 with ventricular septal defect (VSD), 0/6 with tetralogy of Fallot (TOF), 2/6 7 with double outlet RV, 2/6 with transposition of the great arteries (TGA) and VSD, 3/9 with atrioventricular (AV) canal defect, one of two with mitral valve replacement; one of three with congenitally corrected TGA with repair; and none of three with AV canal plus TOF and none of one with subaortic membrane. Ventricular function prior to placement of the initial DDD pacemaker was normal (FS > 15%) in all but one patient, whose %FS was 10%. This patient showed an acute worsening of function within 5 days of placement of the dual chamber (DDD) pacemaker with a % FS of 3%. This patient was excluded from the study due to the poor function prior to placement of the conventional pacemaker and was not included in the numbers given above.

The QRS duration (QRSD) with conventional pacing was 84–170 (median 135 ms) in the non PIVD group and 100–168 (median 124) ms in the PIVD group (Fig. 2). The FS% while paced was 16–46, median 30% in the non-PIVD group and 6–15 (median 11%) in the PIVD group. The FS post upgrade to biventricular pacing (BiVP) (with an epicardial left ventricular (LV) lead) in the patients with PIVD was 23–33 (median 29%) ($p < 0.001$) (Fig. 2a).

All patients with %FS <15% were upgraded to BiVP.

The time delay between placement of the initial dual chamber pacemaker and detection of PIVD ranged from nine days to 29 months (median 5 months).

The left ventricular end-diastolic dimension prior to placement of a BiVP (in the PIVD group) was 31–44 mm (median 41 mm) and changed to 36–41 mm (median 34 mm) after upgrade to a BiVP ($p < 0.01$) (Fig. 2b).

We did not have any patients who did not respond to BiVP.

4. Discussion

Right Ventricular pacing appears more likely to cause PIVD in certain forms of CHD such as double outlet RV, TGA and AV canal defect and less likely to do so in certain other defects such as VSD and TOF.

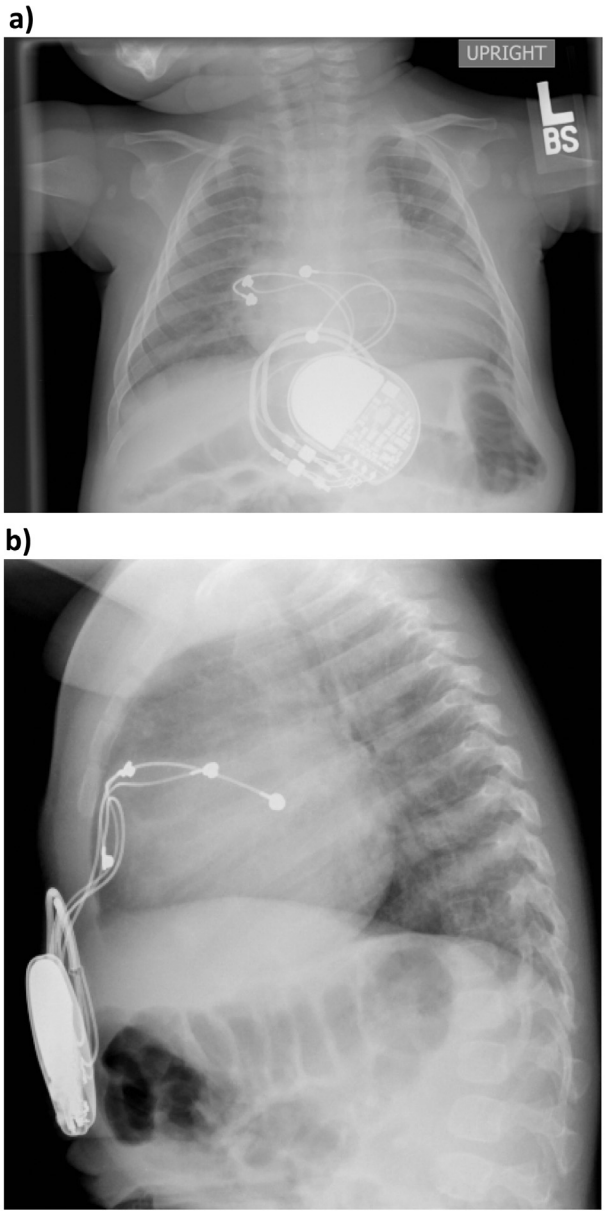

Fig 1. X-rays show typical lead placement in the antero-posterior (a) and lateral view (b) on the anterior aspect of the right atrium (RA) and RV in an infant with DDD pacemaker.

Pacing induced ventricular dysfunction is thought to be the result of dyssynchrony and has been demonstrated in many cardiac situations such as left bundle branch block, preexcitation, frequent premature ventricular contractions and also pacing.[7] In patients with PIVD, conversion to BiVP (i.e. cardiac resynchronization therapy (CRT)) or minimization of pacing with restoration of the native conduction (even though at slower heart rates) has been shown to restore ventricular function back to normal.[2,3,5,8] Certain pacing sites such as the RV free wall and RV outflow tract appear to be more prone to PIVD while pacing at the apex or the LV appears to be less likely to cause dyssynchrony and PIVD (6).

The phenomenon we have observed, namely of different types of CHD having greater or lesser predilection to develop PIVD has not, to our knowledge, been described before. Different forms of CHD are associated with significant differences in myocardial architecture which may influence the conduction of depolarization

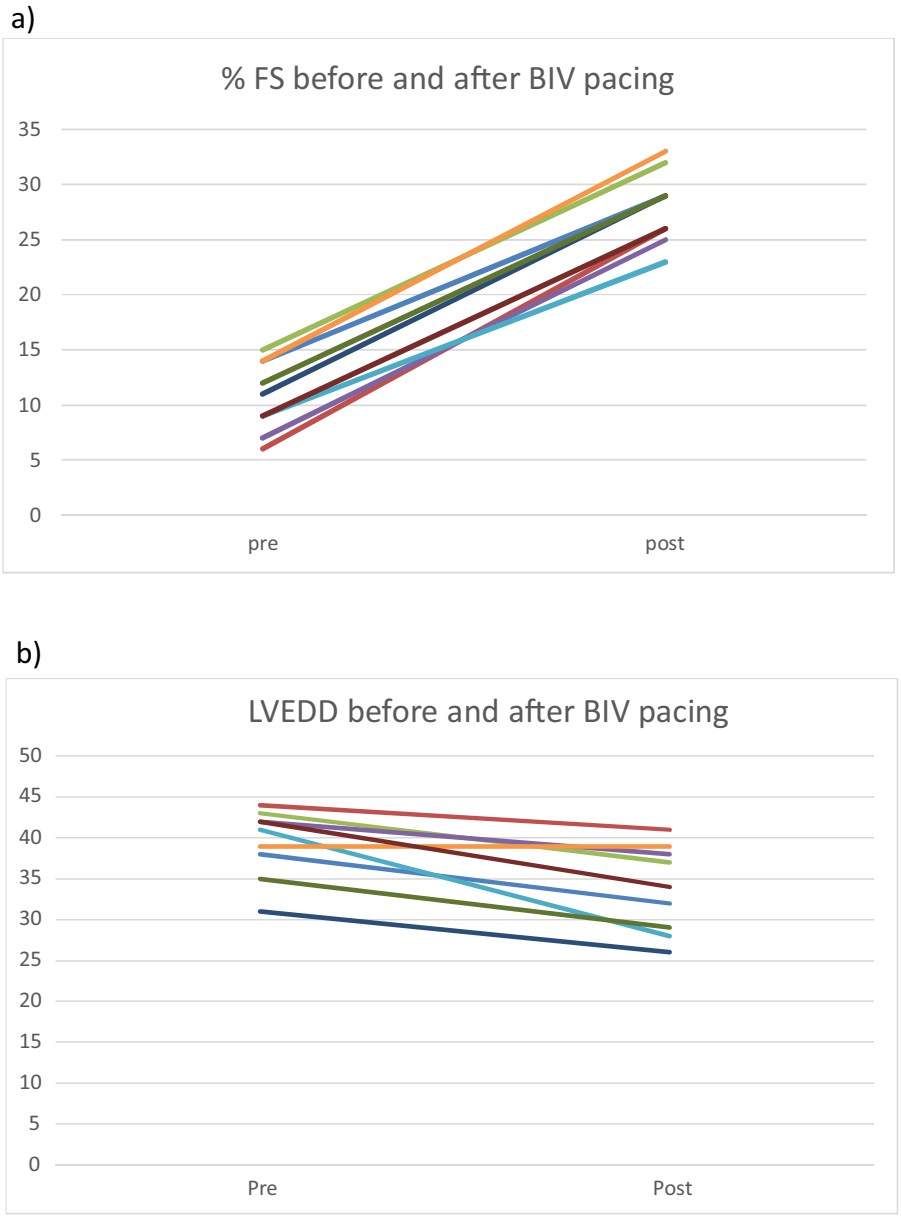

Fig. 2. compares the%FS (a) and LV end diastolic dimension (b) between RV only pacing (DDD mode) versus biventricular (BiV) pacing in the PIVD group.

from the pacing site with consequent differences in the extent of dyssynchrony.

One of the potential markers of the extent of dyssynchrony is the QRS duration. A longer QRS duration is thought to imply a longer depolarization time of the myocardium and is considered a marker of dyssynchrony.[9] However, in our cohort, we were unable to see a difference in the QRSD during RV pacing between patients who developed PIVD compared to those who did not.

Another possible influence could be the pacing rate. All the patients in this study had implantation of a dual chamber pacemaker for CHB. In children, this usually results in the rhythm usually being one of atrial sensing (of sinus rhythm) with ventricular pacing. In such a situation the ventricular rate is similar to a normal person and highly variable. We would not expect there to have been any difference in sinus rates between the two sub-groups. Therefore it would be hard to surmise that the difference we saw was secondary to the ventricular rate.

Pacing the LV as the preferred site may prevent the development of PIVD in most patients.[6,10] Indeed, conversion to LV apex pacing has been shown to be an attractive alternative in patients who develop RV pacing-induced dysfunction.[10] However, during the pacemaker implantation procedure, surgical exposure may be limited due to extensive scarring with consequent need for more dissection. In such a situation, the surgeon has to balance the immediate intra-operative risks of further dissection against potential future benefit from decreased dyssynchrony. Our study may help guide surgeons facing such a dilemma. Based on our findings, we feel that greater effort to improve exposure and place leads on the LV (rather than the RV) is warranted in certain conditions and probably not warranted in others.

Our current strategy in children with post-surgical complete heart block has evolved to placement of the ventricular lead at the LV apex unless it is deemed to be of much higher risk at the time of surgery, in which case, the leads are placed at the safest and most accessible location.

5. Limitations

This was a retrospective study with small patient numbers and therefore limited in the strength of the conclusions that may be drawn. Furthermore, the numbers within each sub-group were even smaller and could have impacted our findings. For instance, it is possible that certain kinds of VSDs not included in our study are indeed at risk of developing PIVD, but this aspect was not noted because of the small numbers in our cohort.

The congenital heart operations and the pacemaker procedures were performed by different surgeons at different centers.

The left ventricular%FS only uses septal and posterior wall movement to determine function and can be an unreliable measure of ventricular function in patients with dyssynchrony. However, the advantage of%FS is its simplicity and in our cohort we could see improvement in%FS in all patients upgraded tom biventricular pacing.

6. Conclusions

Our study appears to suggest that the tendency to develop PIVD may differ based on the underlying structural cardiac abnormality in children with CHD. Further studies are needed to confirm or deny the validity of our findings.

Disclosure

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