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Predictors of left ventricular remodelling and failure in right ventricular pacing in the young

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Aims To identify risk factors for left ventricular (LV) dysfunction in right ventricular (RV) pacing in the young.

Methods and results Left ventricular function was evaluated in 82 paediatric patients with either non-surgical ($n = 41$) or surgical ($n = 41$) complete atrioventricular block who have been 100% RV paced for a mean period of 7.4 years. Left ventricular shortening fraction (SF) decreased from a median (range) of 39 (24–62)% prior to implantation to 32 (8–49)% at last follow-up ($P < 0.05$). Prevalence of a combination of LV dilatation (LV end-diastolic diameter $> +2z$-values) and dysfunction (SF < 0.26) was found to increase from 1.3% prior to pacemaker implantation to 13.4% (11/82 patients) at last follow-up ($P = 0.01$). Ten of these 11 patients had progressive LV remodelling and 8 of 11 were symptomatic. The only significant risk factor for the development of LV dilatation and dysfunction was the presence of epicardial RV free wall pacing (OR = 14.3, $P < 0.001$). Other pre-implantation demographic, diagnostic, and haemodynamic factors including block aetiology, pacing variables, and pacing duration did not show independent significance.

Conclusion Right ventricular pacing leads to pathologic LV remodelling in a significant proportion of paediatric patients. The major independent risk factor is the presence of epicardial RV free wall pacing, which should be avoided whenever possible.

Keywords Permanent cardiac pacing • Heart failure • Cardiac resynchronization therapy • Congenital heart disease • Children

Introduction

Right ventricular (RV) pacing is associated with asynchronous left ventricular (LV) activation,[1] which can lead to deleterious pathologic remodelling and LV failure. Several recent studies have demonstrated that high percentage of RV apical pacing correlates with morbidity and mortality on heart failure in adults,[2–4] however, only limited data are available in case of children. Karpawich *et al.*[5] described histological changes (myofibrillar hypertrophy, fibrosis, fatty deposits) and depressed LV function in paediatric patients with congenital complete atrioventricular block (CAVB) and RV apical pacing. Two multicentre studies reported children with congenital CAVB who developed severe dilated cardiomyopathy.[6,7] Interestingly, all of those patients have been conventionally paced from a very young age (median 1 and 7 days, respectively). No signs of acute inflammation supporting the hypothesis of an immune-mediated process were found in endomyocardial biopsies. Accidental reports and small series could show positive and partially spectacular benefits from upgrades to biventricular pacing in RV pacing-induced heart failure in children.[8–10] Finally, two larger multicentre surveys have found a high percentage of pacing-associated cardiomyopathy among congenital heart disease patients subjected to cardiac resynchronization therapy (CRT).[11,12] Although it is doubtless that a distinct proportion of RV-paced paediatric patients may develop clinically significant LV dysfunction, the incidence of this phenomenon is not known and the risk factors are poorly understood.

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Methods

Patients

A total of 91 consecutive patients with CAVB, systemic left ventricles, and biventricular circulation who underwent pacemaker implantation between 1984 and 2003 in a single tertiary paediatric cardiac surgery centre serving the whole population of the Czech Republic (10.7 millions of inhabitants) have been retrospectively reviewed. Of these 91 patients, 82 have echocardiographic data and were included in the present study. Forty-one patients had non-surgical and 41 patients surgical CAVB. Demographic data relating to block aetiology and pacing site are summarized in *Tables 1* and *2*. Structural heart disease in the non-surgical CAVB group consisted of patent arterial duct in all three cases closed interventionally ($n = 2$) or surgically ($n = 1$). Of these three patients, one had positive, one negative, and one unknown maternal antibody status, respectively.

Pacing

All patients had 100% RV pacing along with complete paced ventricular activation. *Table 1* summarizes last pacing modes and sites. Pacing lead positions were assigned according to implantation protocol data and confirmed by available chest X-rays. All RV apical and septal pacing sites were endocardial, and all epicardial pacing leads were placed on the free wall of the RV. Except for one case (change from VVIR to DDD pacing 20 days before last follow-up), all pacing mode and site changes were performed >3 months before the last echocardiographic examination. Thus only last follow-up pacing variables were used for further analysis.

Echocardiography

Echocardiographic data stored on analogue tapes during evaluations performed prior to and immediately after the pacemaker implantation (before discharge from hospital) and at the end of follow-up were analysed. Parasternal M-mode images were used to measure the LV end-diastolic diameter (LVEDD) and end-systolic diameter (LVESD). Measurements were taken at the point of peak diastolic LV free wall outward motion and peak systolic inward motion, respectively, and LV shortening fraction (SF) was calculated according to the following formula: (LVEDD − LVESD)/LVEDD × 100. Echocardiographic measurements were compared with the normal values of body weight matched individuals,[13] using the *z*-score method. Left ventricular dilatation and dysfunction were defined if both SF<26% and LVEDD>+2*z*-values were present. Colour Doppler echocardiography was performed for quantifying mitral regurgitation using a four-grade scale. In five patients subjected to CRT LV, end-diastolic and end-systolic volumes were prospectively measured using the Simpson's biplane method and ejection fraction was also calculated.

Statistical analysis

Data are presented as mean (SD) or as median (range). The differences between two groups were compared by unpaired *t*-test or Mann–Whitney rank sum test as appropriate according to normality of distribution. Two-sided tests were used in all instances. For categorical variables, the χ^2 or Fisher's exact tests were applied. Multiple comparisons between different patient groups were performed by one-way analysis of variance or the Kruskal–Wallis one-way analysis of variance on ranks followed by pair-wise comparisons using the Holm–Sidak or the Dunn's method, respectively. Repeated measurements within the same group of patients were analysed by the Friedman repeated measures analysis of variance on ranks. When overall significance was found, pair-wise multiple comparisons were performed by the Tukey test. Significance level was accepted at $P < 0.05$. Independent variables showing significant univariate differences related to the development of LV dilatation and dysfunction were entered into a backward stepwise logistic regression analysis. Only pre-implantation and pacing-related variables with a possible causative relationship to the studied endpoint and without potential dependence on it were chosen. The result was finally validated by a bootstrapping method using a BCa approach for the calculation of confidence intervals at a level of significance $\alpha = 0.05$[14] and by the estimate of the shrinkage.[15] SigmaStat for Windows Version 3.11 (Systat Software, Inc., San Jose, CA, USA), SPSS Statistics 17.0 (SPSS, Inc., Chicago, IL, USA), and statistical software R

Table 1 Patient demographics

		All patients	Non-surgical CAVB	Surgical CAVB	*P*-value[a]
Patients	*n*	82	41	41	—
Age at first implantation (years)	Mean (SD)	7.0 (5.4)	8.2 (5.0)	5.8 (5.6)	0.045
Total duration of pacing (years)	Mean (SD)	7.4 (4.5)	6.3 (3.6)	8.5 (5.1)	0.029
Duration of pacing from the last pacing site (years)	Mean (SD)	6.6 (4.1)	6.1 (3.5)	7.1 (4.7)	0.256
Maternal antibody status					
Positive	*n* (%)		12 (29.3)		
Negative	*n* (%)		21 (51.2)		
Unknown	*n* (%)		8 (19.5)		
Structural heart disease	*n* (%)	44 (53.7)	3 (7.3)	41 (100.0)	<0.001
Cardiac surgery	*n* (%)	42 (51.2)	1 (2.4)	41 (100.0)	<0.001
Pacing mode DDD/DDDR	*n* (%)	48 (58.5)	21 (51.2)	27 (65.9)	0.262
Transvenous RV apical pacing	*n* (%)	46 (56.1)	25 (61.0)	21 (51.2)	0.505
Transvenous RV septal pacing	*n* (%)	17 (20.7)	10 (24.4)	7 (17.1)	0.587
Epicardial RV free wall pacing	*n* (%)	19 (23.2)	6 (14.6)	13 (31.7)	0.102

CAVB, complete atrioventricular block; RV, right ventricle; SD, standard deviation.
[a]Non-surgical vs. surgical CAVB.

Table 2 Pacing site-specific patients' data

		RVA	RVS	RVFW	*P*-value (overall)	*P*-value (RVA vs. RVS)	*P*-value (RVS vs. RVFW)	*P*-value (RVA vs. RVFW)
Patients	*n*	46	17	19	—			
Age at first implantation (years)	Median (range)	8.1 (0.1–24.3)	10.1 (0.4–18.8)	1.1 (0.0–7.2)	<0.001	NS	<0.05	<0.05
Total duration of pacing (years)	Mean (SD)	8.7 (4.2)	6.2 (5.0)	5.5 (3.9)	0.014	NS	NS	<0.05
Duration of pacing from the last pacing site (years)	Mean (SD)	7.8 (4.2)	5.0 (3.5)	5.5 (3.9)	0.012	<0.05	NS	<0.05
Structural heart disease	*n* (%)	22 (47.8)	9 (52.9)	13 (68.4)	0.317			
VSD	*n*	3	1	3				
AVSD	*n*	3	1	4				
ToF	*n*	5	—	1				
d-TGA/DORV	*n*	4	—	2				
AS subvalvular	*n*	3	2	—				
Other	*n*	3	2	3				
Cardiac surgery for SHD	*n* (%)	22 (47.8)	7 (41.2)	13 (68.4)	0.207			
Surgical AV block	*n* (%)	21 (45.7)	7 (41.2)	13 (68.4)	0.178			
Maternal antibody status								
Positive	*n* (%)	7 (15.2)	2 (11.8)	3 (15.8)	0.930			
Negative	*n* (%)	34 (73.9)	13 (76.5)	15 (78.9)	0.908			
Unknown	*n* (%)	5 (10.9)	2 (11.8)	1 (5.3)	0.732			
Pacing mode DDD/DDDR	*n* (%)	21 (45.7)	11 (64.7)	16 (84.2)	0.014	0.257	0.255	0.006

AS, aortic stenosis; AV, atrioventricular; AVSD, atrioventricular septal defect; DORV, double outlet right ventricle; d-TGA, d-transposition of great arteries; NS, non-significant; RVA, right ventricular apical pacing; RVFW, right ventricular free wall pacing; RVS, right ventricular septal pacing; ToF, tetralogy of Fallot; SD, standard deviation; VSD, ventricular septal defect.

2.8.0 (R Foundation for Statistical Computing, Vienna, Austria, http://www.r-project.org/foundation/) were used for statistical workup.

Results

Left ventricular size and function

There was a significant decrease in LV SF and increase in the LVEDD at the end of follow-up (*Table 3*). LV SF tends to worsen more in the surgical vs. non-surgical CAVB group between the post-implantation measurement and last follow-up [mean −9 (11) vs. −4 (8)%, respectively, $P = 0.052$]. Similar tendency has, however, not been observed for the change in LVEDD. The incidence of LV dilatation and dysfunction of patients with available data was found to increase significantly from 1.3% prior to pacemaker implantation and 1.6% immediately after implantation to 13.4% (11 of 82) patients at last follow-up (*Table 3*) and differed depending on the pacing site (*Figure 1*). Although complete echocardiographic data were not available for all follow-up points, all patients with LV dilatation and dysfunction at last follow-up had both pre- and early post-implantation echocardiographic measurements included in the analysis. Detailed clinical and echocardiographic data of the 11 patients with late LV failure are shown in *Table 4*. All of these 11 patients had progressive deterioration of LV SF as well as LV dilatation except Patient 2, in whom LV size decreased (but did not normalize) after mitral valve replacement for severe regurgitation. In six of 11 patients, LVEDD increased by >2z-values and SF decreased by >10% from early post-implantation evaluation to last follow-up. No significant haemodynamic or structural abnormalities other than the influence of pacing explaining late LV dysfunctions were identified. Myocardial biopsies were available in two of three patients with non-surgical AV block who reached the defined endpoint of LV dilatation and dysfunction and did not reveal any signs of myocarditis. The findings were, however, consistent with those reported by Karpawich *et al.*[5] in patients with chronic RV pacing.

Univariate risk factors for late left ventricular dilatation and dysfunction

Patients with late LV dysfunction were significantly younger at pacemaker implantation, had higher degree of baseline mitral regurgitation, and had higher proportion of dual-chamber and epicardial RV free wall pacing. The pre-implantation, early post-implantation, and last follow-up parameter differences are listed in *Table 5*.

Table 3 Changes in left ventricular size and function

Parameter		Preimp (n = 77)	Postimp (n = 62)	Last (n = 82)	*P*-value (overall)	*P*-value (preimp vs. postimp)	*P*-value (postimp vs. last)	*P*-value (preimp vs. last)
LVEDD (z-score)	Median (range)	+0.9 (−6.3 to +8.5)	+0.3 (−4.0 to +6.9)	+0.9 (−3.4 to +11.3)	0.017	NS	<0.05	NS
LV SF (%)	Median (range)	39 (24–62)	37 (15–58)	32 (8–49)	<0.001	NS	<0.05	<0.05
Left ventricular dilatation and dysfunction[a]	*n* (%)	1 (1.3)	1 (1.6)	11 (13.4)	0.001	0.574	0.026	0.010

Last, last follow-up; LVEDD, left ventricular end-diastolic diameter; LV SF, left ventricular shortening fraction; Preimp, pre-implantation; Postimp, post-implantation.
[a]LVEDD >+2z, SF <26%.

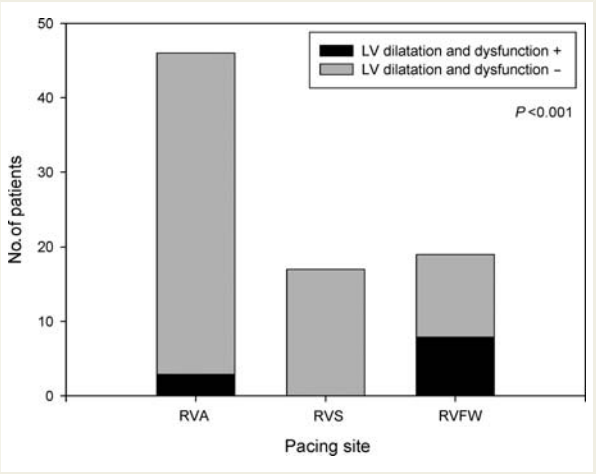

Figure 1 Proportion of patients developing LV dilatation and dysfunction (SF<26% and LVEDD >+2z-values) according to the pacing site. RVA, endocardial RV apical pacing; RVS, endocardial RV septal pacing; RVFW, epicardial RV free wall pacing.

Multivariable risk factors for late left ventricular dilatation and dysfunction

Four independent variables showing univariate significance and lack of cross-correlation (denoted by footnote in *Table 5*) were entered into a backward stepwise logistic regression model that identified the epicardial RV free wall pacing (OR = 14.3, 95% CI = 2.3–78.2, $P < 0.001$) as the only significant predictor of the development of late LV dilatation and dysfunction (standard error of the regression coefficient $b_1 = 3.85$ and the estimate of shrinkage = 1.005).

Outcome of patients with late left ventricular dilatation and dysfunction

Eight of the 11 patients were in NYHA Class II–IV. One of the 11 patients had to be placed on extracorporeal membrane oxygenation for refractory heart failure and died (Patient 11, *Table 4*). Another patient was successfully transplanted (Patient 3, *Table 4*). Five patients were resynchronized (Patients 1, 4, 5, 7, and 9, *Table 4*). Four of them by an upgrade to biventricular pacing and the remaining one (Patient 1, *Table 4*) by programming the pacemaker to low intervention rate to allow a narrow QRS escape rhythm to prevail. These five patients showed major reverse remodelling of the LV within 1–18 months of therapy (*Table 6*). Two of these five patients (Patients 1 and 9) had already been reported in the previous publication.[8] The remaining four of 11 patients are compensated on heart failure medication (*Table 4*).

Discussion

Our data have confirmed the previously published reports on the development of pathologic LV remodelling in a significant proportion of young patients with RV pacing. The incidence of

Table 4 Patients with left ventricular dilatation and dysfunction at follow-up

No.	Age (years)	Diagnosis	Surgical procedure	Block	AB	P. mode	P. site	FUP (yrs)	NYHA	Outcome	LVEDD z-score			LV SF%			MR grade		
											Pre	Post	Last	Pre	Post	Last	Pre	Post	Last
1[a]	0.24	TAPVD	Redirection	Surg.		DDD	RVFW	0.66	III	CRT[b]	−0.9	−1.0	8.5	36	33	12	2	2	2
2	1.34	AVSD/I	Patch MV replacement	Surg.		DDD	RVFW	13.21	I	HFx	6.6	5.1	2.5	45	29	24	4	0	0
3[a]	8.13	CCAVB	—	Cong.	NA	DDD	RVA	4.13	III	HTx	2.3	2.3	6.0	24	30	16	0.5	0	3
4	5.18	PA/IVS	Biventricular repair	Surg.		DDD	RVFW	4.79	II	CRT	−0.3	0.6	4.3	34	24	4	0	2	1
5[a]	6.99	CoA, MV AN, VSD	CoA repair VSD closure MV replacement	Surg.		DDD	RVA	7.64	II	CRT	1.8	0.1	3.5	32	44	19	3	0	0
6	1.41	AVSD/I	Patch MV replacement	Surg.		DDD	RVFW	13.83	I	HFx	−0.5	2.0	3.6	38	24	20	0	0	0
7[a]	0.44	VSD	Patch	Surg.		DDD	RVFW	2.16	II	CRT	3.2	0.1	11.3	40	35	8	1	1	1.5
8	0.14	CCAVB	—	Cong.	+	DDD	RVFW	3.21	I	HFx	2.6	3.7	4.5	35	15	20	0	0	0.5
9[a]	0.00	CCAVB	—	Cong.	+	DDD	RVFW	3.36	IV	CRT	4.0	0.1	10.2	30	26	10	2	0	3
10	6.86	IE	TV replacement	Surg.		VVI	RVFW	9.53	II	HFx	1.2	0.5	4.6	41	31	24	1	0	0
11[a]	1.13	ToF	Repair	Surg.		DDD	RVA	6.68	IV	died	0.7	−1.8	7.0	36	39	12	0	0	3

AB, specific maternal antibodies; Age, age at first pacemaker implantation; AN, anomaly; AVSD/I, incomplete atrioventricular septal defect; CCAVB, congenital complete atrioventricular block; CoA, coarctation of aorta; cong., congenital; CRT, cardiac resynchronization therapy; FUP, length of follow-up in years; HFx, heart failure medication; HTx, heart transplantation; IE, infectious endocarditis; last, last follow-up; LV, left ventricle; LVEDD, left ventricular end-diastolic diameter; MR, mitral regurgitation; MV, mitral valve; NA, not available; P., pacing; PA/IVS, pulmonal atresia with intact ventricular septum; preimp, pre-implantation; postimp, post-implantation; RVA, endocardial right ventricular apex; RVFW, epicardial right ventricular free wall; surg., surgical; ToF, tetralogy of Fallot; TV, tricuspid valve; VSD, ventricular septal defect.

[a]Patients, whose LVEDD increased by >2z-values and SF decreased by >10% from early post-implantation evaluation to last follow-up.

[b]Resynchronization by spontaneous narrow QRS escape rhythm.

Table 5 Univariate analysis of differences between patients with left ventricular dysfunction and dilatation at the last follow-up and the rest of the group

Late LV dysfunction		Yes (n = 11)	No (n = 71)	P-value
Pre-implantation parameters				
Structural heart disease	n (%)	8 (72.7)	36 (50.7)	0.208
Surgical AV block	n (%)	8 (72.7)	33 (46.5)	0.121
Age at first implantation (years)	Mean (SD)	2.9 (3.2)	7.6 (5.4)	0.006
Age at first implantation <2 years[a]	n (%)	7 (63.6)	13 (18.3)	0.003
QRS duration (ms)	Median (range)	80 (60–100)	80 (60–160)	0.857
LVEDD (z-score)	Mean (SD)	+1.2 (2.4)	+1.9 (2.2)	0.403
LV SF (%)	Mean (SD)	40 (8)	35 (6)	0.064
Mitral regurgitation ≥grade 2/4[a]	n (%)	4/11 (36.4)	4/70 (5.7)	0.010
NYHA class	Median (range)	1 (1–2)	1 (1–2)	0.747
Early post-implantation parameters				
Paced QRS duration (ms)	Median (range)	130 (100–160)	135 (80–180)	0.665
LVEDD (z-score)	Median (range)	+0.32 (−1.80 to +5.07)	+0.46 (−4.02 to +6.92)	0.617
LV SF (%)	Mean (SD)	37 (8)	31 (8)	0.032
Mitral regurgitation ≥grade 2/4	n (%)	2/11 (18.2)	2/60 (3.3)	0.111
NYHA class	Median (range)	1 (1–2)	1 (1–2)	0.775
Last follow-up parameters				
Age at last follow-up	Mean (SD)	9.2 (5.8)	15.2 (6.1)	0.003
Total duration of pacing (years)	Median (range)	4.8 (0.7–13.8)	7.2 (0.1–20.1)	0.301
Duration of pacing from the last pacing site (years)	Median (range)	4.1 (0.7–13.8)	6.1 (0.1–17.3)	0.327
DDD pacing[a]	n (%)	10/11 (90.9)	38/71 (53.5)	0.022
Transvenous RV apical pacing	n (%)	3/11 (27.3)	43/71 (60.6)	0.052
Transvenous RV septal pacing	n (%)	0/11 (0.0)	17/71 (23.9)	0.109
Epicardial RV free wall pacing[a]	n (%)	8/11 (72.7)	11/71 (15.5)	<0.001
Paced QRS duration (ms)	Median (range)	160 (140–200)	155 (120–200)	0.014
LVEDD (z-score)	Median (range)	+4.6 (+2.5 to +11.3)	+0.6 (−3.4 to +5.5)	<0.001
LV SF (%)	Mean (SD)	17 (6)	33 (5)	<0.001
Mitral regurgitation ≥grade 2/4	n (%)	4/11 (36.4)	4/68 (5.9)	0.011
NYHA class	Median (range)	2 (1–4)	1 (1–2)	0.009

AV, atrioventricular; DDD, dual-chamber pacing; LV, left ventricular; LVEDD, left ventricular end-diastolic diameter; NS, non-significant; RV, right ventricular; SF, shortening fraction.
[a]Variables entered into multivariable analysis.

Table 6 Patients who underwent CRT

No.[a]	Age at CRT years	Follow-up on CRT months	SPWMD (ms)		IVMD (ms)		SF%		LVEDVi (mL/sqm BSA)		EF%	
			Before	After	Before	After	Before	After	Before	After	Before	After
1[b]	0.90	1.0	250	−10	50	10	12	38	119	83	32	76
4	11.59	7.5	337	−126	126	59	4	18	141	98	9	31
5	14.74	11.2	309	−111	45	11	19	31	144	67	48	68
7	2.62	13.0	270	−40	50	15	8	33	249	58	22	70
9	3.40	17.5	230	−22	40	22	10	27	163	67	28	62

Data immediately before CRT and after given follow-up on CRT are displayed. BSA, body surface area; CRT, cardiac resynchronization therapy; IVMD, interventricular mechanical delay; SF, shortening fraction; SPWMD, septal to posterior wall motion delay; LVEDVi, left ventricular end-diastolic volume index.
[a]Numbers identify patients according to *Table 3*.
[b]Resynchronization by spontaneous narrow QRS escape rhythm.

LV dilatation and dysfunction (13.4%) is somewhat higher when compared with data published recently (7.4 and 6.0%, respectively).[10,16] This may be caused by different definitions used to specify LV dysfunction and influenced by a bias introduced through lack of information in nine of 91 originally eligible consecutive patients, who could not be included in our analysis because of insufficient echocardiographic data but are likely not to have developed clinically significant heart failure because not coming to clinical attention.

For the first time, our data point to a pacing site-specific risk for LV dysfunction development. The presence of epicardial RV free wall pacing was the only significant and independent multivariable predictor of adverse outcome. One may speculate that epicardial RV free wall pacing may carry more LV dyssynchrony than commonly used endocardial pacing sites. The quality of the retrospectively analysed echocardiographic data was unfortunately not sufficient to prove this hypothesis by measuring mechanical LV dyssynchrony indices and further studies specifically looking at this issue are needed. The results of the analysis may point to additional contributing factors for the development of pacing-induced LV cardiomyopathy based on the presence of high sinus-driven pacing rates associated with young age and a DDD pacing mode. When combined, these risk factors may constitute a vicious circle of a tachycardia–dyssynchrony-mediated cardiomyopathy.[6,8]

Our findings also confirm the benefit of LV resynchronization in symptomatic RV pacing-induced LV dysfunction in children as has been reported previously.[8,10] Upgrading to biventricular pacing or just switching the pacemaker off to allow an escape narrow QRS rhythm to prevail may lead to successful reverse LV remodelling. The importance of conventional pacing-induced systemic ventricular dysfunction has been recently reported by two published retrospective multicentre studies on CRT in paediatric and congenital heart disease. Pacing-induced ventricular failure was a major indication for CRT ranging from 44.7%[11] to 77.1%.[12]

The present report has several limitations. First, the retrospective study design did not allow for evaluation of complete echocardiographic data sets. This may have influenced the results of univariate and multivariate analyses. However, serial echocardiographic data were available in all patients reaching the composite endpoint of LV dilatation and dysfunction, thus excluding at least the possibility that already existing pre-implantation dysfunction could have been missed and interpreted as a consequence of pacing during later follow-up. Secondly, measurement of LV SF may not be the best method to reflect the degree of systolic LV dysfunction in the presence of dyssynchrony. However, a decrease in SF along with progressive LV dilatation is, in our opinion, a relatively solid marker of pathologic LV remodelling, and changes in SF were well correlated with changes in LV ejection fraction as measured by the biplane Simpson's method in patients undergoing CRT. Thirdly, LV dysfunction may have been potentially caused by operation in the surgical AV block patients. However, development of LV dilatation and dysfunction has not been significantly different between the non-surgical and surgical AV block groups and aetiology of AV block has not been identified as a significant risk factor in the statistical analysis. Furthermore, in the published reports[8,10] as well as in all our patients, LV function was found to improve regardless of AV block aetiology after CRT.

Clinical implications

The results of this study once again stress the need for prosynchronization strategies in conventional cardiac pacing in the young. Several reports have evaluated optimal pacing lead positions in both adults and children. Karpawich *et al*. have been the first to show preserved LV function and myocardial ultrastructure during pacing from RV septum (proximal His–Purkinje conduction system) when compared with RV apex pacing.[17–19] Tse *et al*. have mapped the RV septum to select a pacing site with the shortest QRS duration. After 18 months of pacing, LV ejection fraction in those patients was significantly higher than in a control group paced from the RV apex.[20] Finally, epicardial LV apical pacing has been shown to carry minimal LV dyssynchrony and the lowest decrease in maximum LV $+dP/dt$ in both an acute animal and human studies[21,22] and may be a promising substitute to RV epicardial pacing in the young. Thus, there are alternative pacing strategies available that should be studied prospectively in order to better define optimal ventricular lead placement in the paediatric age group. In the presence of a systemic LV, epicardial RV free wall pacing should, however, be avoided whenever possible.

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