

# Successful cardiac resynchronization with single-site left ventricular pacing in children

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## Abstract

**Background:** Dyssynchronous left ventricular (LV) contraction due to permanent right ventricular apex (RVA) pacing or delayed electrical activation as typically observed in left bundle branch block (LBBB) has a negative impact on LV function. Objective was to evaluate the impact of epicardial single-site LV pacing in children on LV function and resynchronization.

**Patients:** Single-site epicardial LV free wall pacing was established in 6 children with congenital heart disease and echocardiographic signs of LV dyssynchrony. Reasons for dyssynchrony were either long-term RVA pacing ( $n=5$ ; pacing duration:  $7.7\pm 2.4$  years) or LBBB with drug-resistant congestive heart failure ( $n=1$ ).

**Results:** After 1 month of single-site LV pacing, LV ejection fraction increased ( $41\pm 6$  versus  $53\pm 8\%$ ) and LV enddiastolic volume decreased ( $70\pm 22$  versus  $63\pm 18$  ml/m<sup>2</sup>) as compared to pre-implant measurements. Interventricular mechanical delay decreased ( $67\pm 15$  versus  $16\pm 15$  ms) and intraventricular synchrony was restored (septal-to-posterior wall motion delay:  $312\pm 24$  versus  $95\pm 57$  ms). Accordingly, circumferential 2D strain demonstrated a decrease of LV mechanical delay ( $201\pm 35$  versus  $99\pm 23$  ms).

**Conclusion:** After 1 month of single-site LV pacing, conventional and 2D strain derived echocardiographic measurements indicated improved ventricular function and synchronization in children with previous RVA pacing or LBBB. Further studies are needed to evaluate whether single-site LV pacing may be sufficient for resynchronization therapy.

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**Keywords:** Epicardial pacing leads; Cardiac resynchronization; Single-site; Left ventricular pacing; Children

## 1. Introduction

Clinical trials have demonstrated that a dyssynchronous activation pattern of the left ventricle (LV) due to permanent right ventricular apex (RVA) pacing [1,2] or delayed electrical activation as typically observed in left bundle branch block

(LBBB) [3] results in impaired LV haemodynamics. Moreover, it may worsen the signs and symptoms of heart failure. Biventricular pacing, usually performed with transvenous insertion of an LV lead via the coronary sinus, has been introduced to improve inter- and intraventricular conduction delays [4,5].

There are several particular problems emerging in the paediatric population or in patients with complex congenital heart disease facing life long pacemaker-dependency. Small vessel size as well as cardiovascular abnormalities often preclude a transvenous approach and require epicardial pacing [6,7]. With the improved performance indicating stable acute and chronic pacing thresholds and improved lead survival [8,9], epicardial pacing leads are increasingly used.

Objective was to evaluate whether epicardial single-site LV pacing in children improves LV dyssynchrony and performance compared to RVA pacing and LBBB.

**Abbreviations:** BPM, beats per minute; CRT, cardiac resynchronization therapy; FS, fractional shortening; IVMD, interventricular mechanical delay; LBBB, left bundle branch block; LV, left ventricular; LVEDV, left ventricular enddiastolic volume; LVEF, left ventricular ejection fraction; RVA, right ventricular apex; SD, standard deviation; SPWMD, septal-to-posterior wall motion delay; TDI, Tissue Doppler imaging; 2D strain, two-dimensional strain.

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## 2. Patients and methods

### 2.1. Patients and pacing leads

A total of 6 children with congenital heart disease and echocardiographic signs of LV dyssynchrony were enrolled prospectively. Reasons for dyssynchrony were long-term RVA pacing ( $n=5$ ) or post-surgical LBBB ( $n=1$ ) with drug-resistant congestive heart failure. All children were treated with epicardial single-site LV pacing. Access for implantation of the bipolar steroid-eluting epicardial leads (Medtronic CapSure Epi 4968, Medtronic, Inc, Minneapolis, MN, USA) was via a left axillary mini-thoracotomy to reach the LV free wall and the left atrial appendage, as described in detail previously [10]. Standard surgical suture fixation of the leads was used.

The study protocol was performed with institutional ethical committee approval and parental written informed consent was obtained.

### 2.2. Electrocardiogram

Electrocardiogram and echocardiographic studies were performed one day before LV lead implant, at hospital discharge and 1 month thereafter.

Surface 12-lead electrocardiograms were recorded using a Mac®5000 System (GE Medical Systems, Milwaukee, WI, USA). Measurements of QRS intervals and axis were performed by computed electrocardiographic system analysis of intervals and validated manually.

### 2.3. Echocardiography

Echocardiography was obtained from standard precordial views with a 4-MHz phase array transducer using a Vivid 7 digital ultrasound system (GE VingMed Ultrasound, Horten, Norway). Mono- and two-dimensional transthoracic echocardiography and Doppler evaluation was performed according to recent guidelines [11] and by a single, experienced observer. Cineloops for 3 consecutive heartbeats were stored digitally. All recordings were made after patients had been resting for at least 10 min in the supine position. Measurements were taken during continuous monitoring to confirm stable heart rate. Analyses of the digitalized loops were performed offline using the Echopac version 4.0.2 (GE VingMed Ultrasound, Horton, Norway) after blinding for the ventricular pacing site.

### 2.4. Conventional parameters

Enddiastolic and endsystolic LV diameters were derived from parasternal long-axis M-mode. Enddiastolic and endsystolic LV volumes (LVEDV and LVESV) were determined from the apical 4-chamber view using the monoplan modified Simpson's rule. LVEDV was corrected for the individual body surface area. Fractional shortening (FS) and ejection fraction (LVEF) were calculated to assess global LV function. The

delay between peak septal and left posterior wall systolic motion (septal-to-posterior wall motion delay, SPWMD) was determined as a measure of intraventricular dyssynchrony [12]. This was accomplished by taking into account the maximum wall thickening as the expression of active contraction [13]. Furthermore, the interval between the onset of electrical systole (QRS complex) and the opening of the pulmonic and aortic valves was measured. The difference between the left and right ventricular pre-ejection periods (interventricular mechanical delay, IVMD) was calculated and regarded as a measure of interventricular dyssynchrony [14].

### 2.5. Tissue Doppler imaging (TDI)

Two-dimensional colour-coded TDI was performed in the apical 4-chamber view as described previously [15]. Two basal and two mid-segmental areas were analyzed to evaluate the long-axis motion of the LV. Time to peak myocardial systolic velocity (with reference to the QRS complex) was measured prior and after single-site LV pacing. The maximum time difference between the initial and latest peak myocardial systolic velocity prior and during single-site LV pacing was determined ( $\Delta$ Time TDI-s).

### 2.6. 2D strain imaging

Myocardial circumferential 2D strain analysis was performed based on frame-to-frame tracking of acoustic tissue pixels within 2D echocardiographic images, as described in detail previously [16–18]. In the parasternal short axis view, a 12 segment LV model including the mitral valve and papillary muscle level was evaluated. Frame rates were 60 to 120 frames/s. Visual control of tracking quality was performed to ensure accurate automatic tracking. Negative deflections of strain were interpreted as segmental contraction representing circumferential myocardial shortening, and positive deflections as relaxation, respectively. For each segment, the time from the QRS complex to maximal peak negative circumferential strain was determined. To define the severity of LV dyssynchrony, the maximum difference between any two segments for circumferential strain ( $\Delta$ Time 2Dstrain) prior and during single-site LV pacing was reported. Moreover, the standard deviation of the time to peak deformation of all 12 segments was determined (SD  $\Delta$ Time 2Dstrain).

### 2.7. Interobserver agreement

To assess interobserver agreement, a second experienced paediatric cardiologist who was blinded for the ventricular pacing site and study purpose, performed independent measurements of the echocardiographic data.

### 2.8. Statistics

Data are expressed as mean ( $\pm$ standard deviation). Differences between measurements prior and during LV pacing

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