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Cardiac resynchronization therapy - Always right for the right ventricle?

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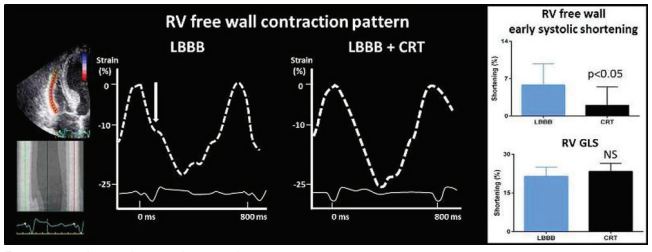
Background: Right ventricular (RV) function influences prognosis in recipients of cardiac resynchronization therapy (CRT). However, direct impact of left bundle branch block (LBBB) and CRT on RV function is not well understood.
Purpose: To study the immediate response of CRT on RV function in LBBB.
Methods: 14 patients with LBBB and non-ischaemic cardiomyopathy (QRS 169±17ms) were studied shortly before and during CRT. RV longitudinal strain was measured by speckle tracking echocardiography. Global RV free wall systolic strain (GLS) was calculated. In 10 anaesthetized dogs we measured RV dimensions by sonomicrometry and pressure by micromanometer and induced LBBB by RF ablation. RV work was calculated from RV pressure-dimension loops.
Results: In patients, LBBB was associated with an abnormal and distinctive early-systolic contraction pattern in the RV free wall, with a steep initial shortening followed by a small plateau before it continued to contract (arrow in left panel of Figure). The abnormal RV free wall shortening coincided with pre-ejection shortening in the septum. This early systolic RV shortening was markedly attenuated by CRT ($p<0.05$). However, RV free wall GLS was unchanged (Figure, right panel). Similar RV free wall contraction pattern as in patients, were observed in the dog model during LBBB. However, with CRT there was a marked increase in RV free wall work from 23±14 to 36±15mm*mmHg ($p<0.01$).



Representative patient and mean data

Conclusions: Patients with LBBB had an abnormal RV contraction pattern occurring in early systole, which was reduced by CRT. The animal model showed that CRT increased workload on the RV free wall despite no improvement in total strain. Therefore, in hearts with intact RV function the RV free wall may compensate well during CRT, whereas hearts with a failing RV may not tolerate the increased workload and may respond poorly to CRT.

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Continuous flow left ventricular assist devices (LVADs) effectively improve pulmonary hemodynamics in bridge-to-transplant patients with end-stage heart failure

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Introduction: Pulmonary hypertension is a frequent sequelae of end-stage heart failure and remains a contraindication for cardiac transplantation. Pulsatile LVADs have been shown to effectively reduce pulmonary hypertension in these patients. However, it remains to be seen if newer continuous flow LVADs have a similar effect on pulmonary hypertension. The objective of this study was to determine if the Heartware (HW), a continuous flow LVAD is effective in improving pulmonary hemodynamics in bridge-to-transplant patients.
Methods: 30 patients with end-stage heart failure underwent placement of Heartware as a bridge-to-transplant (BTT) at our institution.Pulmonary hemodynamics were evaluated with right heart catheterization at baseline, after placement of an intra-aortic balloon pump (IABP), and post-LVAD (prior to heart transplant).
Results: Demographic data of these patients were as follows: mean age 51.6±13.3 years, 70% male, LVEF 14.7±5.11%, 56.6% ischemic etiology and 83.3% received IABP prior to LVAD. Following LVAD support (mean duration of 146. 41±73.83 days), systolic and diastolic pulmonary artery pressures (SPAP and DPAP) decreased significantly (SPAP 56.8±13.55 mmHg, DPAP 28.27±6.23 mmHg to SPAP 35.38±10.23 mmHg, DPAP 15.71±5.36 mmHg; $p<0.001$). Similarly, pulmonary vascular resistance (PVR) decreased significantly from 3.69±2.02 to 2.00±0.85 Woods units ($p=0.004$). Transpulmonary gradient (TPG) also declined significantly post-LVAD from 13.3±5.6 to 9.35±2.98 mmHg ($p=0.02$).

Conclusion: Continuous flow LVADs effectively improve pulmonary hemodynamics associated with end-stage heart failure. Therefore, adequate left ventricular decompression achieved with continuous flow LVAD support can reverse significant pulmonary hypertension in end-stage heart failure patients making them eligible for cardiac transplantation.

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Left ventricular free wall pacing causes excessive work load in septum and right ventricular free wall-a mirror image of left bundle branch block

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Background: Previous studies have shown that ventricular pacing causes non-uniform distribution of work in the left ventricle (LV). This is a potentially deleterious effect since excessive segmental load may be a stimulus to remodelling and may contribute to progression of heart failure.
Purpose: To determine effect of LV free wall pacing on distribution of work within the LV and between the LV and right ventricular (RV) free wall.
Methods: In 16 anaesthetized dogs, LV and RV pressures and dimensions by sonomicrometry were used to assess work as area of ventricular pressure-dimension loops. Longitudinal segment lengths were used for regional work and diameters for LV and RV short axis work. Two different activation patterns were studied, induction of LBBB by RF ablation (n=10) and pacing of the LV lateral wall (n=6) to study early activation from the septum and the LV lateral wall, respectively.
Results: Induction of LBBB caused reduction of RV free wall work from 36±15 to 23±14mm*mmHg ($p<0.01$) and reduction in septal work from 96±52 to 16±61mm*mmHg ($p<0.01$.) There was a simultaneous increase in work in the LV lateral wall from 118±89 to 194±111mm*mmHg ($p<0.01$). Therefore, LBBB caused a shift in workload from the early activated septum and RV free wall to the late activated LV lateral wall (Figure 1). During LV lateral wall pacing there was an opposite shift, with reduction of work in the early activated LV lateral wall from 47±39 to -6±22mm*mmHg ($p<0.05$), and increase of work in the late activated RV free wall from 27±18 to 36±18mm*mmHg ($p<0.05$) and in septum from 72±32 to 141±41mm*mmHg ($p<0.05$).

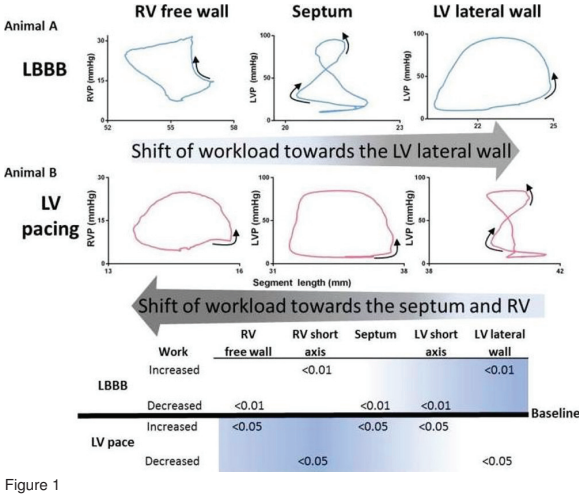

Figure 1

Conclusion: Single lead LV lateral wall pacing shifted ventricular work from the LV lateral wall to septum and RV free wall. This was opposite to effect of inducing LBBB. These results suggest that care should be exerted when placing pacing leads in the left ventricle since work load can become excessive in late activated myocardium in both ventricles. These principles should be explored in clinical studies in patients who receive LV pacing during cardiac resynchronization therapy.