Elevated septal wall stress - a driver of left ventricular dysfunction in left bundle branch block?

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Background: Septal dysfunction is a main feature of left bundle branch block (LBBB), and increasing wall stress is a proposed mechanism of heart failure development in LBBB patients. To try to reveal the pathophysiologic pathway from dyssynchrony to heart failure, we investigated the relationship between septal and left ventricular (LV) lateral wall stress in patients with LBBB.

Hypothesis: Increased septal wall stress causes septal dysfunction in LBBB.

Methods: We included 24 LBBB-patients (65 ± 11 years, 11 males) with LV ejection fraction (EF) ranging from 18 to 67%, and 8 healthy controls (58 ± 10 years, 4 males). Wall stress was calculated at peak LV pressure (LVP) according to the law of La Place ([LVP x radius]/[wall thickness]). Wall thickness was measured using M-mode, and regional curvature was measured in mid-ventricular shortaxis from 2D echocardiographic images. We used a previously validated non-invasive method to estimate LVP from brachial blood pressure and adjusted for valvular events. Myocardial scar was ruled out by late gadolinium enhancement cardiac magnetic resonance imaging.

Results: Wall stress was significantly higher in septum than LV lateral wall at peak LVP (48 ± 12 vs 37 ± 11 kPa, $p < 0.01$) in LBBB patients, while no difference was seen in the controls (Figure A). In patients, septal wall thickening showed a strong correlation with LVEF ($r = 0.77$, $p < 0.01$) (Figure B). Similar correlation was not significant for the LV lateral wall ($r = 0.13$, NS). Attenuation of septal wall thickening in LBBB-patients correlated well with increasing septal wall stress ($r=-0.60$, $p < 0.01$). Wall thickening and stress did not correlate in the LV lateral wall ($r=-0.14$, NS).

Conclusion: Increased septal wall stress is associated with reduced systolic thickening in patients with LBBB. Septal wall thickening, in contrast to LV lateral wall thickening, was correlated to global LV function. These findings suggest that septal remodeling which could have normalized septal wall stress, was not achieved and heart failure may develop.

Abstract Figure.