Tachycardia-induced Incomplete Relaxation and Left Ventricular Volume Loss in Concentric Hypertrophy

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Introduction

Concentric Left Ventricular hypertrophy (LVH) is a precursor to heart failure with normal ejection fraction. When isolated human LV myocardium from patients with LVH is electrically stimulated at rates typical for exertion or tachycardia, incomplete relaxation (IR) develops. This is due a reduced sarcolemmal calcium extrusion reserve. We hypothesized IR should translate clinically in a more pronounced reduction in LV end-diastolic volumes (LVEDV) in patients with LVH.

Method

LV parameters including dimensions and volumetric measurements were obtained from 48 dobutamine stress echocardiograms. Heart rate-dependent LV volume changes were recorded and their relationship to LVH investigated.

Result

Patients with LVH had a more pronounced decrease in LVEDV with tachycardia (non-LVH: -16±13 mL, LVH: -26±18 mL, $p<0.02$). This was also evident when the patients were categorized by septal wall thickness. The associated decrease in stroke volume leads to an inability to significantly increase the cardiac output with higher heart rates in patients with moderate levels of LVH (baseline: 3.78±1.28 l/min, peak: 3.74±0.99 l/min, $p=0.94$). In stark contrast, patients without LVH almost doubled their cardiac output (baseline 2.34±0.65 l/min, peak: 4.40±2.56 l/min, $p<0.01$).

Conclusion

Patients with concentric LVH display tachycardia-induced incomplete relaxation that results in a pronounced LV volume loss. This volume loss eliminates the tachycardia-induced increase in cardiac output and may play a central role in symptom development in patients with LVH and diastolic dysfunction.