Results: T2 time was lower in diabetic mice (13.8 ± 2.8 ms versus 18.9 ± 2.3 ms in the control group; p < 0.05). This was associated with a significant increase in collagen deposits, as evaluated by picrosirius red staining, in diabetic mice. Morphologic and functional analysis showed no difference in terms of ejection fraction (60.70 ± 5 % versus 60.35 ± 4 %) between the two groups, but end-systolic (1.28 ± 0.26 μL/g versus 1.04 ± 0.24 μL/g) and end-diastolic volumes (3.22 ± 0.60 μL/g versus 2.67 ± 0.65 μL/g) were significantly increased in the diabetic group. During the electrophysiological study, 3 non sustained ventricular tachycardias were induced in diabetic mice (none in the control group) and 4 supra-ventricular arrhythmias (none in the control group).

Conclusion: In diabetic cardiomyopathy, T2 assessment can detect the presence of fibrosis at an early stage. Myocardial fibrosis is a potential substrate for the genesis of (supra-)ventricular arrhythmias in diabetes mellitus.

Does Resting Left Ventricular Longitudinal Function may predict Exercise Pulmonary Hypertension in Organic Mitral Regurgitation?

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Introduction: Exercise pulmonary hypertension (PHT) can develop in patients with organic mitral regurgitation (OMR), even when resting pulmonary arterial pressure (PAP) is normal. However, systolic PAP is not always available during stress echocardiography. The purpose of this study was to identify echocardiographic predictors of exercise PHT in OMR.

Method and results: Resting and exercise transthoracic echocardiography including Doppler and tissue Doppler imaging (TDI) quantification were performed in 66 consecutive patients (61±15 years, 55% of male) with moderate to severe OMR. LV longitudinal and filling functions were quantified by peak and time-to-peak velocities using TDI for Ea- and Sa-wave. PAP was derived from transtricuspid pressure gradient and was available during exercise in 52 patients (79%). Systolic PAP significantly increased during exercise (from 31±10 to 54±17 mmHg; p<0.01) and exercise PHT (exercise PAP ≥60 mmHg) was observed in 24 patients (46%). Patients with exercise PHT were significantly older (69±11 vs. 59±15 yrs, p=0.004) and had higher resting PAP (36±9 vs. 27±7 mmHg, p=0.0004), higher septal E/Ea ratio (16±6 vs. 13±4, p=0.03), slower TP-Sa (127±27 vs. 153±30ms, p=0.002) and TP-Ea (45±68 vs. 48±3±60ms, p=0.03) and lower septal Ea velocity (6.4±2 vs. 7.4±3cm/s, p<0.01). Exercise PAP was correlated with age (r=0.39, p=0.004), resting TP-Sa (r=0.42, p=0.002) and septal E/Ea ratio (r=0.28, p=0.04). On multivariate analysis, after adjustment for age, sex and septal E/Ea ratio, the independent predictors of exercise PHT were resting systolic PAP (Odd-ratio (OR) =1.25, 95% CI: 1.05-1.4, p=0.003) and TP-Sa (OR=1.04, 95% CI: 1.01-1.1, p=0.03).

Conclusion: This study shows that resting impaired LV longitudinal function is associated with exercise PHT in patients with OMR. The presence of resting subclinical LV dysfunction could play an important role in PHT.

Inter-ventricular delay at peak exercise is independently correlated with left ventricular remodeling at three months in heart failure patients selected for cardiac resynchronisation therapy

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Purpose: Cardiac resynchronisation therapy (CRT) is known to improve survival and to induce beneficial left ventricle (LV) remodeling in heart failure (HF) patients presenting with severe LV systolic dysfunction and prolonged QRS interval. Using those selection criteria, about 35 to 40 % of patients remain non-responders to CRT. We assessed whether exercise inter-ventricular dyssynchrony could be a relevant index for the prediction of response to CRT.

Methods: Eighteen HF patients performed a semi-supine symptom-limited exercise echocardiography before device implantation. Inter-ventricular dysynchrony, assessed by inter-ventricular mechanical delay (IVMD) in pulsed wave doppler, was recorded both at rest and peak exercise. We determined correlations between IVMD at exercise and cardiac output, mitral regurgitation, assessed by the effective regurgitant orifice area (ERO), and right ventricular (RV) longitudinal systolic function, assessed by tissue doppler imaging. We looked for correlations between those parameters and the degree of LV reverse remodeling at three months follow-up assessed by the percentage of change in LV end-systolic volume (%ESV) and absolute value of change in ejection fraction (ΔLVEF).

Results: 39 % of patients were non-responders according to LV remodeling. Exercise-induced changes in IVMD only significantly correlated with RV longitudinal systolic function (r= -0.602; p<0.01) at peak exercise. In a multivariate analysis by stepwise regression, IVMD at peak exercise was greater than 120 ms were studied before and after 6-months of CRT. RV function was assessed by tricuspid annulus plane systolic velocity (Vs RV) with a cut-off of 11.5 cm/s. Diastolic LV function was assessed owing to E/A, mean E/e’ (septal and lateral annulus) and left atrial volume (LAV (ml/m²)). Reverse remodelling was defined as reduction in LV end-systolic volume ≥15%.

Results: Eighteen patients had RV dysfunction (mean Vs RV=7.6 ±1.2 cm/s) and 20 had normal RV function (13.6±2.7 cm/s). Ejection fraction and LV volumes were not different according to RV-function. Patients with RV-dysfunction had more advanced diastolic dysfunction (mitral inflow E/A=2.2±1.7 vs. 1.2±0.8, p=0.02; E/e’=22.7±7.8 vs. 14.4±4, p<0.01) and largest LAV indexed (63.7±23.9±ml/m² vs. 41.7±13.6 ml/m², p<0.01). Vs RV was significantly correlated with parameters of LV diastole (fig1). RV dysfunction was associated and correlated with a weak LV-reverse remodelling at 6-month follow-up (75% vs. 44%, p<0.05 and R = 0.46, p<0.05 between delta (pre-6-month post) of LV end systolic volume and Vs RV).