

Comparative investigation of in vivo hemodynamics in rat models of physiological and pathological left ventricular hypertrophy

Authors:

A. Olah¹, B.T. Nemeth¹, C. Matyas¹, A. Lux¹, M. Ruppert¹, D. Kellermayer¹, L. Szabo¹, M. Torok¹, B. Merkely¹, T. Radovits¹, ¹Semmelweis University, Heart and Vascular Center - Budapest - Hungary,

Topic(s):

Ventricular function / haemodynamics

Citation:

European Heart Journal (2015) 36 (Abstract Supplement), 184-185

Background: Left ventricular (LV) hypertrophy is a physiological (PhyH) or pathological (PaH) response of LV myocardium to increased cardiac load and is associated with characteristic molecular changes. To date, a direct comparison of functional consequences of PhyH and PaH is missing.

Purpose: We aimed at investigating and comparing in vivo hemodynamic alterations in rat models of PhyH and PaH by using LV pressure-volume (P-V) analysis.

Methods: PhyH and PaH were induced in rats by swim training (athlete's heart) and by suprarenal abdominal aortic banding (AB), respectively. Morphology of the heart was investigated by echocardiography. A detailed characterization of cardiac function was completed by LV P-V analysis using a pressure-conductance microcatheter. In addition to in vivo experiments, histological and molecular biological measurements were performed. All data were normalized to the corresponding control group.

Results: As detected by echocardiography, myocardial hypertrophy was more pronounced in PaH (LV mass: $+14.3 \pm 1.5\%$ PhyH vs. $+25.6 \pm 3.2\%$ PaH, $p < 0.01$), which was further confirmed by post mortem heart weight data. In AB we detected subendocardial fibrosis, while no fibrotic remodeling was present in athlete's heart. Reactivation of the fetal gene program could be observed only in the PaH model. PhyH was associated with unchanged LV pressure relations along with increased stroke volume and ejection fraction. In contrast, markedly higher LV end-systolic pressure, as well as unaltered stroke volume and ejection fraction were detected in PaH, indicating the compensated nature of the observed hypertrophy. Active relaxation was ameliorated in athlete's heart, while it showed a marked impairment in AB (Tau: $-7.7 \pm 2.6\%$ PhyH vs. $+42.1 \pm 11.1\%$ PaH, $p < 0.01$). Load-independent, sensitive indices of LV contractility were increased in both models, in parallel with the degree of hypertrophy. Stroke work increased in both models, whereas mechanical efficiency of LV was improved in PhyH and remained unchanged in PaH ($+20.8 \pm 4.7\%$ PhyH vs. $+4.7 \pm 4.9\%$ PaH, $p < 0.05$).

Conclusions: In this project we provided the first detailed, comparative hemodynamic characterization of PhyH and PaH in relevant rodent models. Increased LV contractility could be observed in both types of myocardial hypertrophy, characteristic differences were detected in diastolic function and LV mechanoenergetics.