Response to exercise training is not predicted by exercise oxygen pulse profile

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Introduction: The gain of peak VO2 after exercise training has prognostic value in CHF patients. The predictive factors influencing this response remain debated. As oxygen pulse is correlated with the stroke volume, it could reflect cardiac adaptation during exercise. The aim of this study is to evaluate the cardiac participation in benefits of exercise training (ET).

Methods: We included prospectively 53 CHF patients (mean age: 57±12 years, LVEF 29±6%) who underwent two cardiopulmonary exercises (CPX): before and after 20 endurance exercise training sessions. We analysed for the two CPX heart rate, workload, VO2, oxygen pulse and calculated stroke volume and cardiac output at rest, anaerobic threshold (AT) and peak exercise. We measured BNP levels before and after the exercise training program. Patients were considered as responders if the gain of peak VO2 was >10%.

Results: Mean change in peak VO2 after exercise training is 17% for global population, 36% for responders (n=27) and 4% for non responders (n=26). At baseline, responder patients have a lower workload at AT and at peak exercise (52.6±19 vs 62.2±17 watts p: 0.035 and 68.9±28 vs 86.6±27 watts p:0.035); a lower oxygen uptake (14.8±4.8 vs 17.5±4.6 ml/kg/min p: 0.04), a chronotropic incompetence (max heart rate 99.8±19.8 vs 116±25 b/min p:0.01), a lower maximal cardiac output (6.6±2.5 vs 8.0±2.3/min p:0.019) and a higher BNP serum level (median 506 vs 279 pg/l p:0.019). However oxygen pulse trends or left ventricular function do not influence changes in peak VO2 at any time and regardless of the training type (continuous vs interval training).

Conclusion: Cardiac adaptations in CHF patients do not seem to play major role in the response to exercise. Benefits of exercise training on exercise tolerance in CHF patients are mainly due to peripheral improvements.

Role of endoplasmic reticulum stress in the deleterious cardiovascular consequences of chronic intermittent hypoxia. Beneficial effects of high-intensity interval training

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Background: Chronic intermittent hypoxia (IH) is described as the major detrimental factor leading to cardiovascular morbi-mortality in obstructive sleep apnea (OSA) patients. Previous animal studies demonstrated that IH increases myocardial susceptibility to ischemia-reperfusion (IR). Among mechanisms involved in the pathophysiology of IR, modulation of endoplasmic reticulum (ER) stress seems to play a major role. Exercise is known to exert beneficial effects on the cardiovascular system and a growing body of evidence demonstrates a particular efficacy of high-intensity interval training (HI). The aim of the present study was 1) to evaluate the effects of HI on IH-induced increased susceptibility to IR; 2) to determine whether ER stress was involved.

Methods: Wistar male rats were exposed to 21 days of IH (21-5% FiO2, 60s cycle, 8h/day) or normoxia (N). After one week of IH alone, rats were daily submitted to both IH and HI (2*24min, 15 to 30min). Rat hearts were either submitted to an IR protocol ex-vivo (30min-global ischemia followed by 120 min-reperfusion, Langendorff technic) or rapidly frozen to evaluate ER stress by Western Blot.

Results: IH induced a significant increase in infarct size (35.4±3.2% vs 22.7±1.7% of ventricles, in IH and N respectively, p<0.05) that was corrected by HI (28.8±3.9 vs 21.0±5.1% of ventricles, in IH and N respectively). This was accompanied by a myocardial ER stress IH-dependent, characterized by the increased expression of the ER stress sensor (i.e. GRP78) and the activation of the proapoptotic ER stress pathway. HIT could represent a good preventive strategy to limit myocardial ischemia reperfusion-related damages in OSA patients.

A new animal model of induced DT2 and its application in evaluating exercise benefits

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Type 2 diabetes (DT2) is a major risk factor of atherosclerosis. As Hypertension or dyslipidemia, DT2 can occur in patients with metabolic syndrome and may result in endothelial dysfunction. Physical activity is a part of the preventive treatment of endothelial dysfunction. The aims of this study were to develop a new animal model of induced DT2 and to investigate the impact of exercise on the DT2 development. Male Wistar rats were supplemented with a fructose enriched diet.