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INFLUENCE OF RESISTANCE EXERCISE INTENSITY ON POST-EXERCISE HYPOTENSION, HEMODYNAMICS AND SYMPATHETIC ACTIVATION.

Rezk Cláudio C[1], Marrache Regina CB[1], Tinucci Taís[1], Mion Jr. Décio[2], Forjaz Cláudia LM[1]
[1]University of São Paulo, School of Physical Education and Sport, Laboratory of Exercise Hemodynamics, São Paulo, Brazil.
[2]University of São Paulo, School of Medicine, General Hospital, Hypertension Laboratory, São Paulo, Brazil.

Introduction
Although extensively demonstrated after aerobic exercise, the occurence of post-exercise hypotension after resistance exercise is still controversial. The aim of this study was to evaluate the effect of different resistance exercise intensities on blood pressure and its hemodynamic and autonomic mechanisms in the post-exercise period.

Methods
Seventeen normotensive subjects were submitted, in a randon order, to 3 experimental sessions: control (C – with no exercise), low-intensity resistance exercise (LI – 40% of 1 repetition maximal – RM), and high intensity resistance exercise (HI – 80% of RM). In all sessions, blood pressure (BP – auscultatory and tonometric methods), heart rate (HR – ECG), and cardiac output (CO – CO2 rebreathing) were measured. Autonomic regulation was evaluated by the spectral analysis of HR and BP variabilities. All measurements were taken at baseline and for 90 minutes after rest (C) or exercise (LI and HI).

Results
Systolic BP decreased after both exercise sessions (LI=-6±1 and HI=-8±1 mmHg, $P<0.05$). Diastolic BP decreased after LI exercise, increased in C session, and did not change after HI session. This response was different between LI and the other sessions (LI=-4±1 vs. C=+5±1 and HI=0±1 mmHg, $P<0.05$). CO decreased similarly in all sessions (marginal value=-0.4±0.2 l/min, $P<0.05$), while systemic vascular resistance (SVR) increased in C (+7±2 U, $P<0.05$), did not change in LI (+1±1 U), and increased in HI (5±2 U, $P<0.05$) session, and this behavior was significantly different between sessions. Stroke volume decreased (LI=-11±2 and HI=-17±2 ml/beat, P,0.05), while HR increased (LI=+17±2 and HI=+21±2 bpm, P,0.05) in both exercise sessions, and these responses were greater after HI exercise. In regard to autonomic regulation of the heart, high-frequency normalized component of HR variability decreased (LI=-22±3, and HI=-23±3%, P,0.05), while low-frequency component (LI=+22±3 and HI=+23±3%, P,0.05) increased after both exercise sessions. Low frequency component of systolic and diastolic BP variability did not change in any session.

Discussion/ Conclusions
Resistance exercise of low and high intensity causes post-exercise hypotension, which is greater for diastolic BP after low-intensity exercise. BP fall was due to CO decrease that is not completely compensated by SVR increase. Moreover, BP fall was accompanied by an increase in sympathetic activation of the heart, with no change in sympathetic vasomotor tone.

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