Cardiovagal baroreflex and aortic hemodynamic responses to isometric exercise and post-exercise muscle ischemia in resistance trained men

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Arterial stiffness is associated with reduced baroreflex sensitivity (BRS) and resistance training; thus a potentially increased cardiovascular risk in resistance-trained (RT) individuals. The effects of resistance training on arterial stiffness and BRS have been evaluated at rest, but cardiovascular abnormalities that are not shown at rest may be revealed during recovery after exercise. Aortic systolic (aSBP) and diastolic blood pressure (aDBP), stroke volume (SV), augmentation index (AIx), vagal activity, BRS responses to isometric handgrip (IHG), and post-exercise muscle ischemia (PEMI) were evaluated in 10 RT and 10 untrained (UT) men (21 ± 1 years). Resting aDBP and AIx were lower in RT compared with UT. Heart rate recovery, BRS, and vagal reactivation during PEMI were similar in both groups. Increases in aSBP (13 ± 11 mmHg), AIx (5 ± 10%), and SV (12 ± 12%) during IHG further increased during PEMI (8 ± 14 mmHg, 12 ± 6%, and 10 ± 8%). Increases in aDBP from rest to PEMI were higher in RT (17 ± 9 mmHg) compared with UT (7 ± 8 mmHg). The lower resting aDBP and the enhanced response to PEMI suggest beneficial adaptations in RT men. Wave reflection, aortic SBP, and cardiovagal BRS responses to IHG and PEMI are not affected by resistance training in young healthy men.

Increased arterial stiffness has been associated with reduced baroreflex sensitivity (BRS), a main short-term regulatory mechanism of heart rate (HR) and blood pressure (BP) (Kingwell et al., 1995; Mattace-Raso et al., 2007). Although, increased resting arterial stiffness has been linked to resistance training (Miyachi et al., 2003, 2004), less is known of its effects on cardiac autonomic function. Resting cardiovagal tone is improved (Figueroa et al., 2008) or not affected (Cooke & Carter, 2005) by resistance training. However, autonomic and hemodynamic abnormalities that are not evident at rest may be shown during recovery from isometric handgrip exercise (IHG) (Figueroa et al., 2005).

Post-exercise muscle ischemia (PEMI), induced by circulatory occlusion following IHG, maintains an elevated BP and evokes a baroreflex-mediated increase in vagal tone, which in turn mediates HR recovery to resting levels (Scherrer et al., 1990; Nishiyasu et al., 1994; Iellamo et al., 1999). During PEMI, increased sympathetic stimulation leads to early return of the reflected BP waves from peripheral sites and augmentation of aortic systolic BP (aSBP) (Edwards et al., 2008), defined as augmentation index (AIx) when expressed as a percentage of pulse pressure. AIx, a marker of wave reflection, increases further from IHG to PEMI in healthy individuals (Edwards et al., 2008). Because HR is inversely related to AIx (Wilkinson et al., 2000) and stroke volume (SV), baroreflex-mediated HR recovery may increase SV, AIx and SBP during PEMI in individuals with normal cardiovascular function (Crisafulli et al., 2003; Edwards et al., 2008).

Evaluation of cardiac autonomic and arterial function during stress in resistance-trained (RT) individuals is of potential clinical importance because reduced cardiovagal control of HR and increased AIx are associated with cardiovascular morbidity and mortality (La Rovere et al., 1998; Nichols & Singh, 2002; Weber et al., 2007). A faster post-exercise HR recovery (Otsuki et al., 2007) and an attenuated sympathetic response to PEMI (Sinoway et al., 1992) have been reported in RT compared with untrained (UT) men, suggesting improved autonomic control during recovery after exercise. Based on these findings, we hypothesized that RT men will have improved baroreflex-mediated HR recovery and reduced or comparable AIx and aSBP responses to PEMI compared with UT men. The purpose of this study was to examine cardiovagal BRS modulation of HR and aortic hemodynamic responses to IHG and PEMI in RT men.
Methods

Twenty young men (10 RT) participated in this study (Table 1). RT men had performed whole-body resistance training for 4–7 years, at least 2 days/week at moderate to high intensity. All subjects were performing <20 min of endurance exercise for <3 days/week. None of the UT subjects participated in regular exercise training for at least 6 months before the study. All subjects were apparently healthy, non-smokers and were not taking medications, nutritional supplements or ergogenic substances. The Institutional Review Board approved the procedures and all participants gave written informed consent before data collection.

Experimental protocol

Subjects were tested after an overnight fast and avoiding caffeinated drinks, alcohol, and intense exercise for at least 24 h before testing. Height and weight were measured and body mass index (BMI) was calculated as kg/m². All measurements were performed in a temperature-controlled room (23 °C) in the supine position. Subjects performed a maximal voluntary contraction (MVC) with the dominant arm, calculated from the average of three attempts using an electronic dynamometer (TSDF121C, BioPac System, California, USA). After electrocardiogram (ECG) and BP instrumentation, participants rested for at least 20 min before data collection. The protocol consisted of 4 min of rest followed by 3 min of IHG at 30% of MVC. Five seconds before the cessation of IHG, a cuff on the exercising arm was rapidly inflated to suprasystolic levels (240–250 mmHg) using an automated pneumatic device (Hokanson E20, Bellevue, California, USA) during 4 min of PEMI. Subjects were encouraged to maintain a constant rate and depth of breathing throughout the experiment, but breathing was not paced.

Hemodynamic and autonomic measurements

Continuous ECG and BP recordings were obtained from a bipolar lead sampled by a data acquisition system (Biopac, Santa Barbara, California, USA) and from the middle finger of the nondominant hand using a Finometer device (TNO Biomedical Instrumentation, Amsterdam, The Netherlands), respectively. The ECG and BP signals were sampled and stored in a computer. SV was assessed from the Finometer’s BP waveform using the Modelflow method (Wesseling et al., 1993).

The R-to-R (RR) interval was inspected and edited for ectopic beats and artifacts using linear interpolation. Fast Fourier transformation was used to obtain power spectrums of the RR interval using the WinCPRS software (Absolute Aliens, Turku, Finland). High frequency (HF, 0.15–0.40 Hz) was derived from power spectral analysis (Malliani et al., 1991). Vagal activity was evaluated by the HF power (Malliani et al., 1991; Pagani et al., 1988). Spontaneous BRS was evaluated by the sequence method. The time series of pulse intervals (PI) and SBP were scanned with the WinCPRS software to identify sequences in which PI and SBP concurrently increased (up sequences) or decreased (down sequences) for three or more consecutive beats (Iellamo et al., 1997, 1999). For each sequence, a regression line is computed from changes in SBP and PI at least 1 mmHg and 5 ms per beat, respectively. Only sequences with a correlation >0.80 were considered and the mean slope of all sequences in a period was accepted as an index of BRS.

Pulse wave analysis

A high fidelity transducer (SPT-301B; Millar Instruments, Houston, Texas, USA) was used to obtain BP waveforms from the radial artery of the inactive arm by applanation tonometry during rest, IHG and PEMI. Aortic pressure waveform was synthesized from the radial pressure waveform using a generalized transfer function (SphygmoCor, AtCor Medical, Sydney, Australia). AIx was defined as the difference between the second and first systolic peaks of the aortic BP waveform, expressed as a percentage of pulse pressure. Transit time of the reflected wave (TR) indicates the round-trip travel of the forward wave to the peripheral reflecting sites and its return to the ascending aorta (Nichols & Singh, 2002). Both AIx and TR were accepted as markers of wave reflection and systemic arterial stiffness (McEniery et al., 2005). The average of three measurements recorded during each condition was used in the analysis.

Body composition

Total body lean soft tissue (LST), bone mineral density (BMD), and body fat were determined from a whole-body scan using dual-energy x-ray absorptiometry (iDXA, GE Lunar, Madison, Wisconsin, USA).

Statistics

Autonomic parameters were transformed to their natural logarithm (Ln) before statistical analysis due to skewed distribution. Subject’s characteristics and resting values were compared for group differences using Student unpaired t-tests. A 2 (trained vs UT) × 3 (rest, IHG, and PEMI) analysis of variance with repeated measures was used to assess differences in all dependent variables over time. If a significant group-by-time interaction was observed, the Fisher LSD test was used for post hoc comparisons. Statistical significance was set at P ≤ 0.05. Statistical software SPSS v. 15.0 (Chicago, Illinois, USA) was used for the analysis. Data are presented as mean ± SD.

Results

Age, height, weight, body fat percentage, and fat mass were similar in both groups. The significantly (P < 0.05) higher BMI, LST, and BMD indicated that the overweight was attributed to greater fat-free mass in RT compared with UT men.
Aortic diastolic BP (aDBP) and AIx at baseline were lower \((P<0.05)\) in RT than in UT men. All cardiovascular responses to IHG and PEMI were similar in both groups, except for aDBP (Table 2). The increase \((P<0.001)\) in HR \((20 \pm 10 \text{ beats/min})\), and decreases in total power \((-0.9 \pm 0.9 \text{ Lm}^2\text{s})\), HF power \((-1.1 \pm 1.3 \text{ Lm}^2\text{s})\), and BRS \((-0.5 \pm 0.4 \text{ Lm}^2\text{mmHg})\) during IHG returned to baseline levels during PEMI. Brachial SBP \((22 \pm 9 \text{ mmHg})\) and DBP \((14 \pm 5 \text{ mmHg})\) increased \((P<0.001)\), while \(T_R\) decreased \((-23 \pm 15 \text{ ms}, P<0.001)\) during IHG and the change was maintained during PEMI. The increase from baseline to IHG in SV \((12 \pm 12\%\), \(P<0.001)\), aSBP \((13 \pm 11 \text{ mmHg}, P<0.001)\), AIx \((5 \pm 10\%, P=0.05)\) further increased \((P<0.05)\) during PEMI \((SV 10 \pm 8\%\), aSBP \(8 \pm 14 \text{ mmHg}, \text{AIx} 12 \pm 6\%\)). Although there was no difference in the increase \((P<0.001)\) in aDBP during IHG between the RT \((14 \pm 8 \text{ mmHg})\) and UT \((8 \pm 8 \text{ mmHg})\) men, a group-by-time interaction \((P<0.05)\) was detected such as the increase from baseline to PEMI was greater in RT men \((17 \pm 9 \text{ mmHg})\) compared with UT men \((7 \pm 8 \text{ mmHg})\).

We found that resting AIx and aDBP were lower in RT than in UT men. A lower resting AIx in endurance-trained than UT individuals has been attributed to reduced wave reflection associated with improved endothelial function (Edwards & Lang, 2005). Resistance training also has proven to improve endothelial-dependent vasodilation (Olson et al., 2006). Resistance training decreased resting aDBP (Taaffe et al., 2007), which may have been influenced by improved vasodilatory capacity. Thus, our finding of reduced resting AIx and aDBP in RT men would support the contention that chronic resistance training improves vascular function.

The increase in HR during IHG was associated with decreases in BRS and HF power (vagal activity). The mechanism for the increase in HR during low-intensity IHG is decreased BRS and concurrent vagal withdrawal (Figueroa et al., 2005; Heffernan et al., 2005). During PEMI, HR, vagal activity, and BRS returned to resting levels in both groups. HR recovery after exercise is due to the reactivation of cardio-vagal BRS and the loss of inhibitory signals from central command (Nishiyasu et al., 1994; Iellamo et al., 1999). Arterial baroreceptors are activated by the distending effect of high BP during PEMI (Scherrer et al., 1990; Nishiyasu et al., 1994). Although reduced BRS has been associated with increased arterial stiffness (Kingwell et al., 1995; Mattace-Raso et al., 2007), our findings provide evidence of an unaffected baroreflex-mediated cardiovagal regulation during PEMI in healthy RT men.

We found that IHG increased aSBP, wave reflection (reduced \(T_R\)), AIx, and SV in both groups.

### Discussion

The major findings of the present study were that (1) resting aDBP and AIx are lower in young RT men compared with UT men, (2) aDBP response to PEMI is higher in RT than in UT men, and (3) cardiac autonomic, SV, aSBP, and wave reflection responses to IHG and PEMI are similar in RT and UT men.
Increased AIx and reduced \( T_R \) during IHG at 40% MVC have been related to sympathetic activation (Lydakis et al., 2008). Vasodilation induces a faster return of the reflected wave to the aorta and its fusion with the forward wave during systole leads to increases in aSBP and AIx (Wilkinson et al., 2001; Nichols & Singh, 2002). In addition, left ventricular contractility influences AIx by affecting SV (Weber et al., 2007). Thus, the increases in aSBP and AIx during IHG are likely due to the combined effect of increased wave reflection and SV.

A progressive increase in SV, aSBP and AIx from IHG to PEMI was observed. Although a further increase in AIx during PEMI has been reported in young healthy individuals (Edwards et al., 2008), the SV and aSBP response is a new finding. Because wave reflection (\( T_R \)) did not change from IHG to PEMI, it is likely that the increases in aSBP and AIx during PEMI were associated with changes in SV influenced by cardiac autonomic regulation. HR recovery, which increases diastolic time, in combination with increased myocardial contractility evoke an increase in SV during PEMI (Crisafulli et al., 2003). Moreover, reduced HR is also associated with increased AIx due to a longer ejection duration (Wilkinson et al., 2000). Thus, prolonged ejection of a higher SV may explain the further increase in aSBP and AIx during PEMI.

Aortic DBP increased during IHG and PEMI, but the change from IHG to PEMI was not significant. A greater increase in aDBP from baseline to PEMI was observed in RT compared with UT men, suggesting that the lower resting aDBP may have influenced this response. External compression of inactive muscles increases DBP, which is associated with the size of the muscle mass (Williamson et al., 1994). Moreover, the increase in BP during PEMI is also muscle-mass dependent (Freund et al., 1978). The increased aDBP response during compression of a larger muscle mass in RT men may be due to a greater mechanical vascular compression. Because myocardial perfusion mostly occurs during diastole, high DBP would have a protective effect against myocardial ischemia during exercise (Yamagishi et al., 2005) and post-exercise recovery.

The present study is limited by its cross-sectional design and a small sample size. Longitudinal studies are needed to confirm the effects of resistance training on vascular and autonomic regulation in populations with chronic diseases and physical disabilities. Nevertheless, our results are contributing to clarify the effects of resistance training on arterial function and autonomic control during recovery after exercise.

In conclusion, lower resting aDBP and the enhanced response to PEMI indicate beneficial adaptations to resistance training in young healthy men. Our results also suggest that resistance training has no effect on cardiac autonomic function, wave reflection, and aSBP at rest and in response to exercise and metaboreflex activation.

**Perspectives**

Because resistance training is an intervention for the prevention of sarcopenia, osteopenia, and insulin resistance, it is interesting to know that cardiovagal BRS and pulse wave reflection are not affected in RT individuals. Our results may have important implications for the use of this exercise modality in clinical populations.

**Key words:** aortic blood pressure, wave reflection, heart rate variability, metaboreflex

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**References**


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