Postexercise Hemodynamic Responses in Lean and Obese Men

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ABSTRACT

**Purpose:** We assessed resting central/peripheral blood pressure (BP), postexercise BP and hemodynamic responses [stroke volume (SV), cardiac output (CO), systemic vascular resistance (SVR)] after acute exercise and 2 wk of aerobic training in lean and centrally obese men matched for BP.  

**Methods:** Eight lean (body mass index (BMI) <25 kg/m²; visceral fat = 279±224 cm³) and eight centrally obese (BMI>30 kg/m²; visceral fat = 1,471±374 cm³) men performed 6 training sessions (3d/wk for 40 min at 65-70%HRmax). Resting BP and hemodynamic measurements were obtained at baseline, following exercise for 60 min, and at 24h and 48h after the last training session.  

**Results:** Postexercise brachial and central systolic and mean arterial BP decreased 3-4 mmHg below resting in lean (P<0.001), and increased by 3 mmHg in obese (P <0.02). Post-training resting brachial/central systolic BPs were reduced by 3-4 mmHg only in lean men (P <0.05). Pretraining PEH was significantly correlated with the training-induced change in resting brachial SBP at 48h (r=0.58, P =0.02), but not at 24h (r=0.38, P =0.15). Similar correlations were observed between acute reductions in central systolic BP (SBP) and central SBP at 24h (r=0.43, P =0.09) and 48h (r=0.54, P =0.03) post-training.  

**Conclusions:** In contrast to the consistent results for lean men, PEH was not observed in centrally obese men, and resting SBP was not reduced after a short aerobic training program. Considerable individual variation in postexercise BP response among obese men may have implications for design of exercise interventions to lower BP in these individuals.  

**Keywords:** post-exercise hyoptension; systemic vascular resistance; blood pressure regulation;  
post-exercise blood pressure regulation; cardiac output
INTRODUCTION

Hypertension is a leading risk factor for excess cardiovascular morbidity and mortality (1). On the basis of recent changes to blood pressure (BP) guidelines, roughly 46% of Americans are now classified as having hypertension (1). Individuals with systolic BP (SBP) of 120-129 mmHg and diastolic BP (DBP) < 80 mmHg are now classified as having “elevated” BP (1). Those with elevated BP are twice as likely to develop hypertension compared to normotensives and are at increased risk for cardiovascular morbidity and mortality (1). However, evidence suggests that increased levels of physical activity (2) and increased fitness (3) may reduce the conversion from elevated BP to hypertension. A single bout of exercise has been shown to lower BP for up to 12 h (4) and this postexercise hypotension (PEH) contributes to the antihypertensive effects of exercise (5). Thus, investigations examining the mechanisms of BP responses to exercise in those with elevated BP are warranted.

Postexercise hemodynamic responses have been previously characterized in normal weight and overweight individuals matched for BP (5), but data on obese individuals are scarce. A 2014 meta-analysis included only 1 study on obese subjects (6), all women (7). A recent meta-analysis on PEH included no studies of subjects with BMI above 31 kg/m² (8). This meta-analysis reported that higher BMI was associated with a lower PEH, with a regression line that predicted no PEH for individuals with BMI greater than ~31 kg/m². The only study that included exclusively obese subjects showed that PEH was only evident 10 min postexercise, and the subjects were all women (7). Recently, Bunsawat et al. (9) showed that following acute exercise obese subjects increased arterial stiffness compared to normal weight subjects. However, baseline fitness and BP differed between groups, with the lean individuals having a
higher peak oxygen uptake ($\text{VO}_{2\text{peak}}$) and the obese having higher resting BP. Finally, there are no published data in adult males recruited for central obesity, which is an at-risk cardiovascular phenotype (10).

One potential clinical application of PEH is that it has been demonstrated that the BP response to an acute bout of exercise predicts the changes in resting BP after aerobic exercise training in men and women with a mean pre-training BMI of 28-29 kg/m$^2$ (11). However, it is not known whether this can be observed in obese men with significant central adiposity. This may have considerable clinical relevance due to the high prevalence of obesity and elevated BP.

The goals of the present study were twofold: 1) to compare central and peripheral PEH and hemodynamic responses in lean vs obese men with central adiposity matched for BP; 2) to examine exercise training-related changes in BP and resting hemodynamics in lean and obese individuals with central adiposity. It was hypothesized that lean men would experience a greater PEH and subsequently a greater resting BP reduction following training.

**Subjects and Methods**

*Participants*

Twenty inactive (< 60 min of moderate-to-vigorous physical activity per wk) men aged 18-35 year with high-normal BP (SBP 120-139 mmHg or DBP 80-89 mmHg) were recruited and grouped by BMI and waist circumference (WC) into obese (BMI $\geq$ 30 kg/m$^2$ and WC > 94 cm) or lean (BMI < 25 kg/m$^2$ and WC < 94 cm). Screening physical activity levels were ascertained with the international physical activity questionnaire (12). Those with known cardiovascular,
pulmonary, renal or metabolic disease, current smokers or those on vasoactive medications were excluded. The study was approved by the Arizona State University Institutional review board and was carried out in a manner consisted with the Declaration of Helsinki. Written and informed consent was obtained from the subject prior to study enrollment.

*Prescreening and anthropometric assessment*

A two-phase experimental design was carried out to assess the impact of both acute and chronic exercise on central and peripheral hemodynamic outcomes (Figure 1). Participants were selected such that peripheral BP was similar with divergent BMI and adiposity (described below). Participants had their WC measured at the navel, and weight/height measured with a stadiometer to determine if their BMI and WC met inclusion criteria. Following that they were asked to sit quietly for 5 min and then BP was measured on both arms using an automated BP monitor per the protocol described by the World Health Organization (13). The Physical Activity Readiness Questionnaire was used to screen participants for exclusion criteria. Participants completed a whole-body Dual-energy X-ray absorptiometry (DXA) (GE Lunar iDXA GE Healthcare, Little Chalfont, UK) scan to determine body composition following which participants completed a VO$_{2\text{peak}}$ assessment on an electronically-braked cycle ergometer.

*VO$_{2\text{peak}}$ Assessment*

Participants warmed up at 50 W for 5 min on a cycle ergometer. Following this, power output increased by 30 W every min until voluntary exhaustion. Pulmonary ventilation and gas exchange were measured continuously with a Parvo Medics TrueOne 2400 (Parvo Medics, Sandy, UT) (14). Heart rate (HR) was measured with a Polar HR monitor (Polar, Lake Success,
A respiratory exchange ratio > 1.15, plateau in HR and VO$_2$ were used to determine if participants exerted maximal effort. The mean of the two highest consecutive 15-s oxygen uptake averages during the test was taken as VO$_{2peak}$ and maximal heart rate (HRmax) was recorded.

**Experimental Protocol**

Participants were asked to wear an activity monitor for seven days prior to the study and over the course of the two-wk exercise training program (Sensewear$^\text{TM}$ Pro3 armband) (15). Participants recorded wake time, sleep time, and any time the device was removed for longer than 20 min. Daily steps, and time spent in moderate-intensity and vigorous-intensity activity were recorded during each of the three wk.

**Cardiac output and systemic vascular resistance**

Cardiac output and systemic vascular resistance (SVR) were measured during the PEH assessment and at rest during the post-intervention assessment using impedance cardiography (PhysioFlow$^\text{TM}$; Manatec Biomedical, Paris, France) (16). The device uses changes in transthoracic impedance during cardiac ejection to calculate stroke volume (SV). Brachial/peripheral BP was measured in the laboratory at rest and every 5 min following exercise while the subjects were in a supine position. These values were entered into the PhysioFlow software and SVR automatically calculated. The PhysioFlow has been found valid and reliable in both normal weight and obese persons (17). Additionally, cardiac index (CI), stroke volume index (SVi) and systemic vascular resistance index (SVRi) were computed by dividing cardiac output (CO), SV and SVR by the body surface area to relate these variables to body size.
Central/brachial BP, cfPWV, and PWA

Central/brachial BP, carotid-femoral pulse wave velocity (cfPWV), and pulse wave analysis (PWA) measurements were taken using the SphygmoCor XCEL™ (AtCor Medical, Sydney, NSW, Australia) by means of validated methodology (18, 19). Central BP is automatically derived from 10 sequential, high-quality waveforms which undergo a validated generalized transfer function to generate the corresponding central aortic pressure waveform. Augmentation Index (Aix) was calculated as the difference between the first and second systolic peaks of the ascending aortic waveform expressed as a percentage of the central pulse pressure and normalized for a HR of 75 beats/min (Aix @ HR75). This can also be expressed in absolute terms as the Augmentation Pressure (Ap). Ap and Aix@HR75 are surrogate measures of arterial stiffness and provide incremental information regarding the state of the vasculature. Raised cfPWV has been linked to an elevated risk of cardiovascular disease risk and is an important long-term target for therapy.

Carotid-femoral PWV was determined by simultaneously recording carotid artery and femoral artery waveforms. Distances from the carotid sampling site to the suprasternal notch and, from the suprasternal notch to the femoral cuff were measured. The distance from the femoral arterial pulse to the femoral cuff was obtained and subtracted from the total distance (D; in m). The time (t; in s) between the onset of femoral and carotid waveforms was determined as the mean from 10 consecutive cardiac cycles. Carotid-femoral PWV was calculated as follows: PWV = D/t (m/s). Increases in cfPWV have been linked to an elevated risk of cardiovascular disease risk and it is an important long-term target for therapy.
Acute Exercise Response

Since the greatest PEH response occurs in the afternoon compared to the morning (20), the acute exercise session for measurement of PEH was performed at 1300 h (± 30 min). Participants fasted for at least 5 h, refrained from caffeine/alcohol for 24 h and refrained from unaccustomed physical activity for 48 h prior to their visit. Participants were instructed to avoid antihistamines for 5 days and vitamin C supplementation for 3 days prior to each visit (21). Participants ate breakfast no later than 8 am the morning of this visit and the morning of the post intervention measurement. Instructions were given to consume the same dinner and amount of fluids the night before and the morning of the acute PEH assessment and post-intervention measurements. Participants were asked to record their meals to ensure compliance with this requirement. Liquids were tracked by instructing subjects to drink from a bottle of a known quantity, and to maintain that quantity the day of each measurement.

Upon arrival at the research facility participants lay supine for 20 min to achieve hemodynamic stability. This was verified with repeated BP measurements. Baseline measurements of central and brachial BP, cfPWV, PWA, CO and SVR (measured non-invasively via impedance cardiography) were carried out. Following this, participants performed 40 min of exercise on a cycle ergometer at 65-70% HRmax. Heart rate max was derived from the highest HR value achieved during VO₂peak test. This intensity has been shown to induce a PEH response (22). Following exercise, participants remained in the laboratory for an additional h in a supine position. Central and brachial BP and PWA were measured every 5 min following exercise for one h. Postexercise cfPWV was assessed 30 min and 60 min after exercise cessation. CO and SVR were continuously measured throughout with data collected in 10-s
averages. The time frame of measurement was chosen due to previous research illustrating the greatest hypotensive response to acute exercise is within the first h (23, 24).

*Aerobic Exercise Training*

Participants returned to the laboratory 5 additional times for a total of 6 exercise sessions. This amount of training has been shown to reduce resting BP (25-27). The acute exercise bout served as the first training session and subsequent exercise bouts were performed at the same intensity. At 24 and 48 h following the last exercise session, participants were asked to return to the laboratory for final hemodynamic measurements which were performed as previously described.

*Statistics*

Data were analyzed using SPSS software (version 23). Descriptive data are expressed as mean ($M$) ± standard deviation (SD). Alpha was set at 0.05 for two-tailed hypotheses. Data were analyzed for normality and transformed if necessary. An independent t-test was used to determine if any baseline group demographic differences existed (Table 1). ANOVA was used to detect any mean differences between baseline, 24h, and 48h resting hemodynamic values (Table 2). The physical activity data, BP (brachial and central), arterial stiffness indices, and hemodynamic outcomes between the 2 groups over the 1-h measurement period following exercise, were analyzed using linear mixed models in a hierarchical fashion using restricted maximum likelihood model and ‘variance components’ covariance error structure. Group, time (in h), and age were used as fixed effects. Time was also used as a random effect to account for both inter-individual and diurnal variation in hemodynamic outcomes.
RESULTS

Baseline characteristics

Twenty participants (10 obese, 10 lean) started the study and two subjects dropped out from each group. Subject characteristics are provided in Table 1. There were no significant baseline differences for age or VO_{2peak}, but obese had higher BMI, waist circumference, total body fat, and visceral fat. There were no between-group baseline differences in resting HR, brachial and central BP, cfPWV, Ci, SVi or SVRi. Obese had higher Ap, Aix @ HR 75, CO and SV, and lower SVR at rest.

There were no between-group differences for daily steps at baseline (lean = 6517 ± 3823; 6239 ± 2853 steps/d) or during the two weeks of exercise training (lean = 6235 ± 3483; obese = 6390 ± 2689 steps/d). Lean subjects recorded significantly more time performing moderate-intensity activity (72 ± 29 min/d vs. 47 ± 33 min/d, 95% CI [15, 35], \( P < 0.001 \)) and vigorous-intensity activity (3 ± 3 min/d vs. 1 ± 1 min/d, 95% CI [1 to 3], \( P < 0.001 \)) over the three wk of measurement. It is important to note that there were no within-group changes in daily steps, moderate-intensity activity or vigorous-intensity activity during the 2-wk training period.

Acute Exercise Response

Postexercise BP was initially elevated in both groups, after which brachial SBP/MAP (95% CI’s [-1, -9] (\( P = 0.014 \)), [-.1, -.6] (\( P = 0.002 \)), respectively), and central SBP/MAP (95% CI’s [-2, -7] (\( P <0.001 \)), [-.2, -.5] (\( P = 0.014 \)), respectively), decreased to levels 3-4 mmHg below rest in lean but not in obese (Figure 2A (\( P = 0.196 \)), 2B (\( P = 0.831 \)), 2E (\( P = 0.928 \)), 2F (\( P = 0.831 \))). In obese, sustained elevations in brachial (+3 ± 7 mmHg, 95% CI, [.9, 5]) and central
(+3 ± 8 mmHg, 95% CI [2, 6]) SBP were evident throughout the 60-min postexercise period (both $P = 0.02$; Figure 2A, 2B). This resulted in higher average brachial SBP (5 ± 8 mmHg, 95% CI [.2, 11]) and central SBP (8 ± 8 mmHg, 95% CI [3, 13]) in obese compared to lean during the entire 60-minute postexercise period (both $P < 0.02$). There were no significant postexercise changes over time in brachial or central DBP (both $P = 0.16$; Figure 2C, 2D).

Although the mean data revealed a PEH in lean but not obese, there was considerable heterogeneity of postexercise BP responses in the obese that were not evident in the lean (Figure 3). Whereas every lean subject experienced a PEH, the lack of a mean reduction in brachial SBP in the obese subjects was primarily due to three subjects whose brachial SBP increased postexercise, two by ~10 mmHg.

Postexercise Ap was reduced by an average of 1.9 ± 5 mmHg in lean (95% CI [-.05, -3], $P = 0.02$), whereas a trend for an increase in Ap was observed in obese ($P = 0.055$). Postexercise Aix @ HR 75 was elevated almost two-fold in obese ($P = 0.013$), but unchanged in lean, resulting in a significant group-by-time interaction ($P = 0.004$). Postexercise cfPWV was unchanged in both groups.

Stroke volume was higher in obese compared with lean at rest and throughout the postexercise period ($P = 0.02$), but did not change significantly from rest in either group (Figure 4A). Stroke volume index did not differ between groups, and did not change during the postexercise period (Figure 4B). Cardiac output was higher in obese compared with lean ($P < 0.001$) and was elevated throughout the postexercise period in both groups ($P < 0.001$) (Figure
Cardiac index was similar in both groups and was elevated postexercise \( (P < 0.001) \) (Figure 4D). Systemic vascular resistance was higher in lean compared to obese \( (P = 0.01) \), and decreased postexercise in lean \( (P <0.001) \) but not in obese (Figure 4E). Although no between-group differences were observed in SVRi, postexercise SVRi decreased significantly in lean \( (P < 0.001) \) but not in obese (Figure 4F). There were no between-group differences in HR at rest, during exercise, or postexercise.

**Training adaptations**

The effects of training on hemodynamic variables are presented in Table 2. Brachial and central SBP were reduced after training in lean but not in obese. Brachial DBP was unchanged in both groups. Central DBP was reduced in obese only 24 h after the last training session. Training had no effect on Ap, Aix @ HR 75, or cfPWV, but Ap and Aix @ HR 75 were higher in obese compared to lean. At both 24h and 48h after the last training session, CO and SV were decreased in obese and increased in lean. Heart rate was lower in both lean and obese 48 h after the last training session. At both 24 h and 48 h after the last training session, SVR was significantly lower in lean and higher in obese.

For all subjects combined, there was a significant correlation between the PEH during the acute exercise bout prior to training and the training-induced change in resting brachial SBP at 48h \( (r=0.58, 95\% \ CI [.07, .78], \ P =0.02) \), but not at 24h \( (r=0.38, 95\% \ CI [-.09, .71], \ P=0.15) \) post-training. Similar correlations were observed between acute reductions in central SBP and central SBP at 24h \( (r=0.43, 95\% \ CI [-.03, .74], \ P =0.09) \) and 48h \( (r=0.54, 95\% \ CI [.11, .80], \ P \)
DISCUSSION

The primary finding of this study was that PEH and reduced resting brachial and central SBP after 2 wk of moderate-intensity training were only observed in lean men. In fact, brachial and central SBP were elevated throughout the 60-min postexercise period in centrally obese men. Apart from a transient reduction in central DBP 24h after the last training session, centrally obese men had no acute or training-induced changes in BP. To our knowledge, these data are the first to compare PEH responses between lean and centrally obese adult males.

Acute Exercise Responses

While PEH has been well documented (6, 28), characterization of the postexercise BP response in obese populations is limited. Although it has been reported that BMI is not significantly associated with PEH, that study did not include obese participants (5). A recent meta-analysis reported that PEH was significantly correlated with BMI ($r = 0.26$), with a regression line indicating that PEH would not be expected in individuals with BMI higher than $\sim 31 \text{ kg/m}^2$ (8). A 2014 meta-analysis included only one study of PEH in obese adults (6). In that study (7), obese women with only slightly elevated resting SBP (mean = 122 mmHg) demonstrated a decrease in SBP for only 10-20 min postexercise, with a return to pre-exercise SBP by 30 min after the 20-min walk at 65% VO$_2$peak. The magnitude and duration of the PEH was similar in obese and lean women, despite the lean women having lower resting SBP (mean = 114 mmHg). PEH was also observed in young overweight and obese women (mean SBP = 119
mmHg) after a graded maximal exercise test (29), but the PEH was not observed until the final 30 min of a 60-min postexercise measurement period. Our data are the first to show that, on average, obese men with high-normal SBP do not exhibit PEH following a moderate-intensity aerobic exercise bout. Although sex does not appear to influence PEH (6), no direct comparison of obese men and women in the same study has been published.

Our data illustrate a high heterogeneity in PEH response in obese men (Figure 3). The high degree of individual variability in response to exercise has been documented (30). Adverse responses to chronic training have also been documented (31), but it is unknown if obesity increases the risk for a non- or adverse response. Although we did not have a sufficient sample size to perform correlations within each subgroup, it is worth noting that the two obese subjects who had the greatest increases in postexercise brachial SBP after the acute exercise bout (Figure 3) also had the greatest increases in resting brachial SBP after training (5-10 mmHg). Although we did not observe reduced BP in the initial 1 h postexercise in obese men, it is possible that PEH might be delayed in this population.

In most instances, indices of SVR are reduced below pre-exercise values during the PEH period (32) while CO is most often elevated (33). Consequently, PEH is thought to be principally due to a decrease in SVR that is not completely offset by an increase in CO (22). Data from our lean subjects are consistent with previous findings (7, 22), as a sustained reduction in SVR throughout the 60-minute postexercise period was relatively greater than the elevated postexercise CO. By contrast, the obese men had elevated CO postexercise during which time SVR was not significantly reduced.
The lack of change in SVR in obese men could be due in part to the significantly lower resting SVR compared to lean men (1218 vs 1604 dyn*s/cm^5), thus limiting the extent to which this variable could be further reduced. Although SVR was lower in the obese group, obese SVR was still higher than what is considered a normal range of 800 – 1200 dyn*s/cm^5. We obtained measurements in a supine position, which increases the likelihood of observing decreases in SVR (6). In obese women, PEH was achieved by decreases in SVR that more than offset increases in CO, and these responses were like those observed in lean women (7). In that study, resting SVR of obese women was only 11% lower than in lean women. In our study, resting SVR of obese men was 24% lower than that of the lean men. This could contribute a “floor effect” that limited the capacity for the obese men to further reduce SVR postexercise.

The reductions in AP and Aix@HR75 following exercise are consistent with previous findings in individuals with elevated BP although the exact mechanisms driving these improvements remain unclear (34). Finally, cfPWV did not change following exercise in the present study. Although reductions in cfPWV have been reported following a single bout of exercise it is important to note that these reductions were observed in lean, normotensive individuals (35). Finally, there is evidence for reductions in cfPWV following acute maximal exercise in lean individuals with non-response observed in obese individuals (9).

**Exercise Training Adaptations**

Similar to what was observed acutely following exercise, differences in resting SBP after training were likely driven due to opposite adaptations in CO and SVR. In lean men, the reduction in SBP was due to a decrease in SVR that was relatively greater than the increase in
CO, whereas in obese men a reduction in resting CO was offset by an increase in SVR. Differences in CO adaptations were accounted for by opposite changes in SV.

The observed reductions of 3-4 mmHg in brachial and central SBP in lean men after training are consistent with previous findings (28) and would be predicted on the basis of the PEH response (11). Liu et al. (11) reported that the magnitude of PEH was significantly correlated with the reduction in resting SBP ($r = 0.89$) after aerobic exercise training. Similarly, Hecksteden et al. (36) reported that pre-training PEH was significantly correlated ($r = 0.77$) with the post-training reduction in resting SBP. Both studies included overweight and obese men and women, but any effect of BMI was not reported. In view of these findings, the unchanged SBP in our obese men was not unexpected because they exhibited no PEH prior to the training program. For central SBP in our study, correlations between the PEH observed during the initial acute bout of exercise and changes in resting central SBP at 24 h ($r=0.43; P=0.09$) and 48 h ($r=0.54; P=0.03$) after the last training session were not as strong as previously reported. Nonetheless, the positive correlations support the previous findings and suggest that the central SBP blood pressure response to a single exercise bout might be useful in identifying individuals who could be classified as potential responders or non-responders to aerobic exercise training for purposes of reducing resting SBP. Approximately 25% of individuals who engage in an exercise program with the goal of decreasing BP are considered non-responders (30).

Despite the lack of reduction in SBP after training in obese men, central DBP (but not brachial DBP) was reduced by 5 mmHg 24 h (but not 48 h) after the last training session. The fact that there was no PEH for central DBP suggests that changes in central DBP in response to a
single aerobic exercise session may not be useful in predicting changes in resting central DBP after exercise training.

The lack of reduction in cfPWV, AP and Aix@HR75 in response to aerobic training in either group is in accordance with prior literature. A recent meta-analysis (37) established that aerobic training caused greater improvements in peripheral arteries (brachial-ankle PWV) than with central arteries (cfPWV) and those with stiffer arteries (PWV > 8 m/s) saw greatest reductions in PWV. The PWV of our subjects (6.0 m/s and 6.6 m/s for lean and obese, respectively) may have limited our ability to detect changes in arterial stiffness, especially with an exercise program lasting only 2 wk. It is thus plausible that subjects in our study needed a stimulus of a greater duration to observe effects on cfPWV.

**Strengths and Limitations**

Our study is the first to compare both brachial and central hemodynamic responses to acute and chronic exercise in lean and obese men. Measurements were made in the supine position which minimizes orthostatic stress (6). Activity monitors allowed us to confirm that PA outside the supervised exercise training was not likely to have influenced our results, as daily energy expenditure, daily steps and time spent in moderate and vigorous intensity PA did not change in either group during the 2 wk of exercise training. In addition to BMI and WC, DXA was used to characterize subjects by total body and visceral fat since obese individuals are likely to have increased arterial stiffness independent of peripheral BP levels (38), due primarily to central adiposity rather than obesity *per se* (39). We did not provide food to the participants. However, subjects recorded food and beverage consumption and were instructed to replicate the
same for dinner and breakfast prior to testing sessions. Beverage consumption during visits was kept constant. The fact that we only used men in our study is a limitation but also allowed for a focused study design that eliminated the potential confounding due to sex differences in cardiovascular hemodynamic responses to exercise (40). Given the lack of PEH data on individuals with central obesity, which is an at-risk phenotype regarding blood pressure, we chose to examine centrally obese men (10). Additional research is required to determine whether our findings extend to obese women. It is possible that the relatively short 2-wk training program may have diminished our chances of observing a reduction in resting SBP in the obese men. Meredith et al. (25) and Murray et al. (26) demonstrated BP reductions following 2 wk of training in lean subjects using moderate intensities comparable to the current study. Whyte et al. (27) demonstrated that 2 wk of sprint-training reduced BP in the obese. It is possible that obese individuals may need a more intense dose of exercise, and detailed dose-response studies are needed. However, sprint-interval training utilizing repeated Wingate tests tend to be poorly tolerated even among lean, recreationally active adults and the generalizability of this protocol is suspect (23). Liu et al. (11) reported that decreases in resting SBP were evident only after wk 5 of an 8-wk training program so it is possible that a longer exercise training duration was required. However, subjects in this study lacked significant central obesity (mean waist circumference in Liu et al. 97 cm vs 133 cm in present study) (11). It is plausible that longer duration of exercise training is required to bring about a response in centrally obese individuals. However, our data still underscore the early divergence of post-exercise blood pressure responses between lean and centrally obese men. Although statistically significant differences were observed, the relatively small sample size and large inter-individual variability within groups indicates a need for larger studies to confirm blood pressure differences between obese
and lean subjects. Finally, although we did not observe reductions in blood pressure in obese men in the first hour after exercise cessation, it is possible that post-exercise blood pressure reductions may require more time to occur in this population.

Conclusions

Our results demonstrate that PEH is observed in lean men but not obese men, and that the absence of a PEH in obese men is due to a lack of a decrease in SVR along with an elevated CO during the initial 60-min postexercise. In fact, obese men exhibited a significant postexercise elevation in brachial and central SBP. Whereas all lean men experienced a PEH, there was considerable individual variability in the post-exercise response in obese mean, with PEH evident in half the subject but an elevation in postexercise BP occurring in 3 of the 8 obese subjects. Like the response to acute exercise, only lean men reduced resting brachial and central SBP after exercise training. This was achieved by a decrease in SVR that more than offset an increase in CO. By contrast, in obese men a decrease in resting CO was compensated for by an increase in SVR. Additional research is necessary to establish whether the centrally obese phenotype increases risk of being a non-responder (30) or adverse responder (31) to exercise training.
Acknowledgments:

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Conflicts of Interest:

The authors declare no conflicts of interest.
References


Figure 1: Schematic of study design

Figure 2: Mean brachial SBP (A), central SBP (B), brachial DBP (C), central DBP (D), brachial MAP (E), and central MAP (F) for 60 min following exercise on Day 1 for lean and obese. Horizontal lines represent pre-exercise resting value. The first BP measurement was taken 5 minutes following exercise cessation. Error bar +/- 1 SE

Figure 3: Central systolic BP at rest prior to exercise and the lowest BP value 60 minutes following exercise in lean and obese individuals with slightly elevated BP.

Figure 4: Mean Stroke volume (ml) (A), Stroke volume index (ml/min/m²) (B), Cardiac output (lit/min) (C), Cardiac index (lit/min/m²) (D), Systemic vascular resistance (dyn s/cm²) (E) and Systemic vascular resistance index (dyn s/cm²/m²) 60 min following exercise on Day 1 for lean and obese. Data averaged every 5 minutes. Horizontal lines represent pre-exercise resting values. Error bar +/- 1 SE.
Figure 1

Exercise Training protocol

-4 -2 0 1 3 5 8 10 12 13 14

Days

Exercise sessions

Baseline assessments Acute Exercise Chronic Exercise 2-week (6 sessions) Supervised Exercise Training 24 h post assessment 48 h post assessment

Anthropometrics CV Assessments VO\textsubscript{peak} Acute PEH CV Assessments

40-min cycle ergometer exercise at 65-70% HR max
Figure 2
Figure 3

[Graph showing changes in central systolic blood pressure before and after exercise for lean and obese groups, with statistical significance indicated by P-values 0.001 and 0.948.]
Table 1. Demographic mean values ± SD (range) for subjects at rest by group.

<table>
<thead>
<tr>
<th></th>
<th>Lean (n = 8)</th>
<th>Obese (n = 8)</th>
<th>P-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Brachial SBP (mm Hg)</td>
<td>126 ± 7 (115-137)</td>
<td>126 ± 4 (117-133)</td>
<td>0.976</td>
</tr>
<tr>
<td>Brachial DBP (mm Hg)</td>
<td>76 ± 4 (68-83)</td>
<td>78 ± 7 (69-92)</td>
<td>0.567</td>
</tr>
<tr>
<td>Age (yr)</td>
<td>24 ± 4 (19-31)</td>
<td>25 ± 4 (19-29)</td>
<td>0.547</td>
</tr>
<tr>
<td>VO\textsubscript{2}peak (l.min\textsuperscript{-1})</td>
<td>2.9 ± 4 (2-4)</td>
<td>3.2 ± 7 (2-4)</td>
<td>0.248</td>
</tr>
<tr>
<td>BMI (kg/m\textsuperscript{2})</td>
<td>23 ± 2 (20-25)</td>
<td>34 ± 3 (30-38)</td>
<td>&lt; 0.001</td>
</tr>
<tr>
<td>Weight (kg)</td>
<td>72 ± 19 (64-85)</td>
<td>104 ± 20 (56-120)</td>
<td>&lt; 0.001</td>
</tr>
<tr>
<td>Waist (cm)</td>
<td>81 ± 3 (75-84)</td>
<td>133 ± 40 (99-201)</td>
<td>0.002</td>
</tr>
<tr>
<td>Body fat (%)</td>
<td>23 ± 7 (14-31)</td>
<td>35 ± 2 (33-38)</td>
<td>&lt; 0.001</td>
</tr>
<tr>
<td>Visceral fat (cm\textsuperscript{3})</td>
<td>279 ± 224 (56-661)</td>
<td>1471 ± 374 (843-1903)</td>
<td>&lt; 0.001</td>
</tr>
<tr>
<td>Central SBP (mm Hg)</td>
<td>110 ± 5 (102-119)</td>
<td>113 ± 6 (106-125)</td>
<td>0.123</td>
</tr>
<tr>
<td>Central DBP (mm Hg)</td>
<td>77 ± 4 (69-83)</td>
<td>80 ± 7 (70-94)</td>
<td>0.316</td>
</tr>
<tr>
<td>Ap (mm Hg)</td>
<td>4.63 ± 2 (-4 - 4)</td>
<td>4.5 ± 4 (-3 – 10)</td>
<td>0.001</td>
</tr>
<tr>
<td>Aix @ HR 75 (%)</td>
<td>-2.1 ± 7 (-11 - 6)</td>
<td>6.1 ± 12 (-16 - 23)</td>
<td>0.02</td>
</tr>
<tr>
<td>HR (bpm)</td>
<td>64 ± 10 (51-78)</td>
<td>66 ± 7 (60-72)</td>
<td>0.729</td>
</tr>
<tr>
<td>CO (l/min)</td>
<td>4.7 ± 1 (4-6)</td>
<td>6.3 ± 1 (4-9)</td>
<td>0.005</td>
</tr>
<tr>
<td>Ci (l/min/m\textsuperscript{2})</td>
<td>2.5 ± .4 (2-3)</td>
<td>2.7 ± .6 (2-4)</td>
<td>0.387</td>
</tr>
<tr>
<td>SV (ml)</td>
<td>75 ± 15 (56-92)</td>
<td>101 ± 21 (75-132)</td>
<td>0.013</td>
</tr>
<tr>
<td>Svi (ml/m\textsuperscript{2})</td>
<td>40 ± 7 (33-50)</td>
<td>42 ± 8 (30-56)</td>
<td>0.6</td>
</tr>
<tr>
<td>SVR (Dyn.s/cm\textsuperscript{5})</td>
<td>1604 ± 444 (1141-1830)</td>
<td>1218 ± 263 (839-1520)</td>
<td>0.003</td>
</tr>
<tr>
<td>SVRi (Dyn.s/cm².m²)</td>
<td>2956 ± 867 (2190-3371)</td>
<td>2783 ± 547 (1990-3463)</td>
<td>0.429</td>
</tr>
</tbody>
</table>

Ci (cardiac index), Svi (Stroke volume index), SVRi (systemic vascular resistance index)
Table 2. Mean (± SD) resting values pre-training, 24 and 48 hours following the last training session between group.

<table>
<thead>
<tr>
<th></th>
<th>Pre-training</th>
<th>24-h post-training</th>
<th>48-h post-training</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Brachial SBP (mm Hg)</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Lean</td>
<td>126 ± 7</td>
<td>122 ± 6*</td>
<td>122 ± 6*</td>
</tr>
<tr>
<td>Obese</td>
<td>126 ± 5</td>
<td>127 ± 6</td>
<td>127 ± 4a</td>
</tr>
<tr>
<td><strong>Brachial DBP (mm Hg)</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Lean</td>
<td>76 ± 4</td>
<td>76 ± 6</td>
<td>76 ± 6</td>
</tr>
<tr>
<td>Obese</td>
<td>78 ± 7</td>
<td>74 ± 7</td>
<td>75 ± 8</td>
</tr>
<tr>
<td><strong>Central SBP (mm Hg)</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Lean</td>
<td>110 ± 5</td>
<td>108 ± 5</td>
<td>107 ± 5*</td>
</tr>
<tr>
<td>Obese</td>
<td>113 ± 6</td>
<td>112 ± 6a</td>
<td>113 ± 6a</td>
</tr>
<tr>
<td><strong>Central DBP (mm Hg)</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Lean</td>
<td>77 ± 4</td>
<td>77 ± 4</td>
<td>77 ± 4</td>
</tr>
<tr>
<td>Obese</td>
<td>80 ± 7</td>
<td>75 ± 7*</td>
<td>76 ± 7</td>
</tr>
<tr>
<td><strong>AP (mm Hg)</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Lean</td>
<td>.63 ± 2</td>
<td>.45 ± 2</td>
<td>.12 ± 2</td>
</tr>
<tr>
<td>Obese</td>
<td>4.5 ± 4a</td>
<td>4.9 ± 3a</td>
<td>4.3 ± 3a</td>
</tr>
<tr>
<td><strong>Aix @ HR 75 (%)</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Lean</td>
<td>-2.1 ± 7</td>
<td>-4.9 ± 7</td>
<td>-5.0 ± 7</td>
</tr>
<tr>
<td>Obese</td>
<td>6.1 ± 12a</td>
<td>7.4 ± 9a</td>
<td>6.1 ± 11a</td>
</tr>
<tr>
<td><strong>cfPWV (m/s)</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Lean</td>
<td>Obese</td>
<td>Obese</td>
</tr>
<tr>
<td>-------</td>
<td>--------------</td>
<td>--------------</td>
<td>--------------</td>
</tr>
<tr>
<td></td>
<td>6.0 ± 0.7</td>
<td>6.1 ± 0.9</td>
<td>6.0 ± 0.7</td>
</tr>
<tr>
<td>Obese</td>
<td>6.6 ± 0.8</td>
<td>6.6 ± 0.8</td>
<td>6.7 ± 0.8</td>
</tr>
</tbody>
</table>

SV (ml)

<table>
<thead>
<tr>
<th></th>
<th>Lean</th>
<th>Obese</th>
<th>Obese</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>75.1 ± 15</td>
<td>80.5 ± 13*</td>
<td>81.2 ± 14*</td>
</tr>
<tr>
<td>Obese</td>
<td>100.8 ± 21*a</td>
<td>89.2 ± 19*a</td>
<td>92.2 ± 21*a</td>
</tr>
</tbody>
</table>

HR (bpm)

<table>
<thead>
<tr>
<th></th>
<th>Lean</th>
<th>Obese</th>
<th>Obese</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>64 ± 10</td>
<td>63 ± 11</td>
<td>63 ± 10*</td>
</tr>
<tr>
<td>Obese</td>
<td>66 ± 7</td>
<td>66 ± 9</td>
<td>64 ± 7*</td>
</tr>
</tbody>
</table>

Resting CO (L/min)

<table>
<thead>
<tr>
<th></th>
<th>Lean</th>
<th>Obese</th>
<th>Obese</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>4.7 ± 1</td>
<td>5.0 ± 1*</td>
<td>5.0 ± 1*</td>
</tr>
<tr>
<td>Obese</td>
<td>6.3 ± 1*a</td>
<td>5.8 ± 1<em>a</em></td>
<td>5.8 ± 1<em>a</em></td>
</tr>
</tbody>
</table>

SVR (Dyn∙s/cm5)

<table>
<thead>
<tr>
<th></th>
<th>Lean</th>
<th>Obese</th>
<th>Obese</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>1604.3 ± 444</td>
<td>1435.5 ± 267*</td>
<td>1467.6 ± 296*</td>
</tr>
<tr>
<td>Obese</td>
<td>1218.2 ± 263*a</td>
<td>1265.3 ± 270*a</td>
<td>1313.3 ± 303*a</td>
</tr>
</tbody>
</table>

*Significant difference from baseline
^Significant difference from 24 h following
*Significant between group differences (all P < 0.05)