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ORIGINAL ARTICLE

Is the magnitude of acute post-exercise hypotension mediated by exercise intensity or total work done?

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Abstract The purpose of this study was to investigate the effects of exercise intensity on the magnitude of acute post-exercise hypotension while controlling for total work done over the exercise bout. Seven normotensive physically active males aged 28 ± 6 years (mean ± SD) completed four experimental trials, a no exercise control, 30 min of semi-recumbent cycling at 70% $\dot{V}O_{2peak}$ (INT), cycling for 30 min at 40% $\dot{V}O_{2peak}$ (SMOD) and cycling at 40% $\dot{V}O_{2peak}$ for a time which corresponded to the same total work done as in the intense trial (LMOD). Blood pressure (BP), heart rate, stroke volume, cardiac output, total peripheral resistance, core body temperature and forearm skin and limb blood flow were measured prior to and for 20 min following the exercise bout. Post-exercise summary statistics were compared between trials with a one-factor general linear model. The change in systolic BP, averaged over the 20-min post-exercise period was significantly lower only following the INT (–5 ± 3 mm Hg) and LMOD exercise (–1 ± 7 mm Hg) compared to values in control ($P < 0.04$). The changes in systolic BP and MAP following INT and LMOD were not significantly different from each other ($P > 0.05$). Similar results were obtained when the minimum values of these variables recorded during the post-exercise period were compared. Mean changes in cardiac output (1.9 ± 0.3 l min^{-1}) and total peripheral resistance (–3 ± 1 mm Hg l^{-1} min^{-1}) after INT exercise were also different from those in CON ($P < 0.0005$). The acute post-exercise reduction in BP was clinically similar following high intensity short duration exercise and moderate intensity longer duration exercise that was matched for total work done.

Keywords Blood pressure · Exercise · Blood flow · Temperature

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Introduction

The results of previous research have shown that, following a single bout of exercise, there is a reduction in blood pressure (BP) which has been termed post-exercise hypotension (PEH) (Kenney and Seals 1993). Since PEH begins within the initial minutes immediately following an exercise bout (MacDonald 2002) and can persist for up to 22 h in hypertensive individuals (Pescatello et al. 2004a), it has been suggested that the prolonged hypotensive effect of regular endurance exercise training may be due to repeated instances of PEH (Thompson et al. 2001).

If exercise is to be used as a non-pharmacological intervention in the management of BP, more knowledge is required about different characteristics of the exercise required to evoke PEH, especially the intensity and duration of the bout. Generally, there is no clear consensus within the literature on the exact magnitude of the pressure decrement following exercise or the exact duration of post-exercise BP response. Potential reasons for these between-study variations in outcome could be due to differences in the intensity and/or duration of the selected exercise bout.

A number of researchers have investigated the effect of exercise intensity (Piepoli et al. 1994; Forjaz et al. 1998, 2004; MacDonald et al. 1999) and duration (Forjaz et al. 1998; MacDonald et al. 2000) on PEH. Nevertheless, the effect of such characteristics on PEH is still not clear even in normotensive individuals. For example, varying the

intensity of exercise has resulted in either similar falls in BP post-exercise (Forjaz et al. 1998; MacDonald et al. 1999) or a greater fall following higher intensity exercise (Piepoli et al. 1994; Forjaz et al. 2004). Therefore, whether a longer duration or a higher intensity exercise bout would elicit a greater/lesser PEH is still not clear at this time (Pescatello et al. 2004a). No researcher has attempted to unravel the effects of exercise intensity on the magnitude of PEH whilst controlling for total work done over the exercise bout.

Shephard and Balady (1999) referred to an exercise "dose" in terms of the total amount of energy expended (total work done) during a bout of physical activity. The same total dose of physical activity can be achieved by exercising at a high intensity for a short duration, or at a lower intensity for a longer duration. In a recent study, an attempt was made to unravel the exercise intensity/duration debate in hypertensives. Guidry et al. (2006) compared post-exercise BP changes following exercise that differed in duration and intensity. These researchers employed a parallel design protocol involving separate samples of subjects and were not able to control the exact "total dose" of exercise in each condition. In the current crossover study, we will investigate whether variation in dose and intensity yields differing effects on PEH. In addition, we will consider the underlying variables that contribute to the phenomenon of PEH such as cardiac output (CO) and total peripheral resistance (TPR).

Methods

Participants

Following an estimation of the sample size required for the primary comparisons in our study (see Statistical analysis), seven normotensive (systolic BP < 130 and diastolic BP < 85 mm Hg) physically active males participated in the study. All participants were non smokers, had no history of cardiovascular disease, were not taking any medication and engaged in regular physical activity <2 h per week. Participants were instructed to refrain from exercise and the consumption of alcohol for 24 h, caffeine for 12 h and food overnight prior a test session. The study conformed to the declaration of Helsinki and was approved by the Institutional Ethics Committee; all participants were informed of the methods before giving written informed consent. The characteristics of the participants are shown in Table 1.

Research design

All exercise was performed on a semi-supine cycle ergometer (Kettler Sport, Worcestershire, UK). Prior to any exercise, participants attended the laboratory for familiarisation and anthropometric measurements. During this session, height (m), weight (kg) and resting BP (mercury sphygmomanometer) were determined. Resting BP was determined from the average of three measurements. Participants also completed a bout of cycling on the semi-supine ergometer to familiarise themselves with this exercise. Peak oxygen uptake was determined using a progressive continuous protocol (Bird and Davison 1997). Participants performed 10 min of submaximal exercise as a standard warm-up. Work rate began at 50 W and increased in increments of 25 W every 2 min until volitional exhaustion was reached. Volitional exhaustion was defined as the point at which the subject could no longer maintain the required work rate (≥60 rev min^{-1}). Expired gases were collected using an on-line collection system that sampled every 10 s (MetaMax 1, Cortex Biophysic GmbH, Leipzig, Germany). Oxygen uptake was then plotted against work rate and the exercise work rates (i.e. watts) corresponding to 40 and 70% $\dot{V}O_{2peak}$ were calculated using a linear regression equation.

Table 1 Characteristics of participants

Participant characteristics	Number (n = 7)
Age (years)	28 ± 6
Weight (kg)	73 ± 4.4
Height (m)	1.78 ± 0.4
$\dot{V}O_{2peak}$ (ml kg^{-1} min^{-1})	52.1 ± 6.6
Resting systolic BP (mm Hg)	120 ± 10
Resting diastolic BP (mm Hg)	74 ± 8

Experimental protocol

All participants completed four experimental trials which all began at 0800 hours in a thermoneutral environment (23°C). During each trial, central and peripheral hemodynamic measurements were recorded at rest, and during the 20 min period immediately following the exercise bout. Each trial was completed in a random order and separated by 7–10 days. The intense (INT) trial consisted of 30-min of cycling at 70% $\dot{V}O_{2peak}$. The moderate intensity longer duration trial (LMOD) involved cycling at a lower intensity 40% $\dot{V}O_{2peak}$ for a time (50 ± 8 min, mean ± SD) that corresponded to an equivalent dose (work-rate (W) × time (s)) as in the intense trial. The moderate intensity shorter duration trial (SMOD) consisted of cycling at 40% $\dot{V}O_{2peak}$ (the same intensity as LMOD) for 30 min (the same duration as INT) and the control (CON) trial consisted of resting for 30 min on the cycle ergometer. To ensure the participants were cycling at the correct intensity the

mechanical resistance (watts) was constant during each exercise bout.

A control trial involving collection of data during a period of no exercise was used as the comparator condition, since BP has been shown to change over time when resting in the same body position (Harrison et al. 2000). The authors deemed it clinically important that post-exercise BP was not only lower than pre-exercise values, but that post-exercise BP was lower than values obtained during a period of semi-supine rest.

Measurements

During each trial, participants sat on the cycle ergometer with both arms supported at the same height (i.e. level with the heart). This was achieved using an adjustable arm rest device which consisted of a U shaped flat wooden top surface attached to an electrically driven screw-thread platform. This device allowed two flat wooden surfaces to be positioned either side of the cycle ergometer to limit arm movement during the exercise and to enable the correct arm positioning for the measurement devices. All measurements, with the exception of forearm blood flow, were recorded for 5 min before and 20 min after exercise.

Core body temperature was measured using a thermometric temperature sensor (CorTemp^{TM} Disposable Temperature Sensor HQInc, Florida USA), which was ingested prior to sleep the night before the test. Core temperature was monitored continuously throughout each trial and data were recorded every minute.

Blood pressure was measured continuously from the finger on the left hand (Portapres Model 2, TNO Biomedical Instrumentation, Amsterdam), and was obtained from the electrical integration of the continuous pressure signal (Wesseling et al. 1993). Comparisons of the portapres and intra-arterial blood pressure have shown that finger arterial pressure gives a satisfactory representation of central arterial pressure (Imholz et al. 1998) and BP can be determined reliably and accurately (Eckert and Horstkotte 2002). The Portapres device also provided indirect measurements of stroke volume (SV), heart rate (HR) and cardiac output (CO) based on the same three-element model of aortic input impedance as for arterial BP (Wesseling et al. 1993). The continuous finger arterial pressure wave data were analysed on a beat to beat basis and averaged every minute, by BeatScope pulse contour analysis software (TNO Biomedical Instrumentation, Amsterdam). The pulse contour analysis has been shown to be a reliable method to track changes in stroke volume (Jellema et al. 1999; Nieminen et al. 2000) and cardiac output (Stok et al. 1993; Nieminen et al. 2000). Total peripheral resistance (TPR) was calculated by the ratio of mean arterial pressure (MAP) to cardiac output (TPR = MAP/CO).

Two laser Doppler probes (Perimed, Suffolk, UK) were attached to the ventral aspect of the left forearm using adhesive discs for the measurement of forearm cutaneous blood flow (Periflux System 5001, Perimed Instruments, Jarfalla, Sweden). The arm was then secured to limit movement and elevated to the level of the heart using foam supports. The laser Doppler Flowmetry data were converted from perfusion units to cutaneous vascular conductance (CVC) by the ratio of laser Doppler flux (PU) to MAP (CVC = laser Doppler flux/MAP). All cutaneous blood flow data are presented as CVC increase relative to baseline.

Forearm blood flow (FBF) measurements were obtained immediately prior to, and at the end of, the exercise bout and were recorded from the right arm using venous occlusion plethysmography. The arm was elevated above heart level using foam supports to ensure adequate venous drainage. A double-stranded Silastic Indium–gallium filled strain gauge was placed around the forearm and connected to a plethysmograph (EC-4, Hokanson, Washington, USA). A collecting cuff was secured on the upper arm (immediately above the antecubital fossa) and an occlusion cuff was attached to the wrist. Both cuffs were attached to a rapid cuff inflator device. Circulation to the hand was occluded by inflating the wrist cuff to a supra-systolic pressure of 215 mm Hg for 1 min before the occlusion cycle commenced. Forearm blood flow was assessed by the cyclical inflation (10 s inflation and 5 s deflation repeated five times) of the upper arm cuff to a pressure of 50 mm Hg. The calculation of the slope relating to the change in voltage (i.e. change in gauge length) and time during venous occlusion was performed using Powerlab 5 (AID, Australia) analysis package to enable blood flow to be expressed as per unit of mass, per unit of time (ml·100 ml tissue^{-1}·min^{-1}). An average of the five readings were then used to calculate forearm vascular conductance (FVC) by dividing forearm blood flow by MAP.

Statistical analysis

All measures during the 20-min post-exercise period, with the exception of FVC (since only one post-exercise measurement was recorded) were averaged every minute and subtracted from baseline (pre-exercise) values. Various summary statistics of these post-exercise changes were then calculated and compared between experimental trials with a one-factor repeated measures general linear model (GLM). Newman Kuels multiple comparisons were

employed to examine differences between pairs of trials. This analysis of summary statistics approach was chosen as an alternative to a complicated multifatorial GLM, due to the large number of levels for both the trial (4) and post-exercise time (20) factors. The approach affectively reduces the time factor to important summary statistic(s) thus simplifying the interpretation of interactive effects between factors with so many levels (Atkinson 2001).

The calculated summary statistics were (1) the overall 20 min mean change between post-exercise and baseline, (2) the difference between the 20 min post-exercise minimum value and baseline, and (3) the time that the minimum value occurred during the 20 min post-exercise. The single post-exercise measurement of FVC was subtracted from baseline and recorded as a summary statistic.

Our primary outcome variables were systolic and diastolic BP averaged over the 20-min post-exercise period and subtracted from their respective baseline values. The primary comparison was between the intense and moderate (LMOD) exercise trials, which were equated for total work done. We deemed that a difference between these trials of 5 mm Hg in systolic and/or diastolic BP was clinically important and estimated that seven subjects would allow this difference to be deemed statistically significant (statistical power = 80%, SD of differences ≤4 mm Hg).

Results

Post-exercise BP responses

It can be seen in Fig. 1 that the post-exercise responses of systolic BP and MAP were similar between the INT and LMOD exercise doses. These BP responses were generally greater than in the SMOD dose, which were in turn generally greater than control values. Comparison of the post-exercise summary statistics of mean and minimum BP values confirm these observations ($P < 0.05$). Following INT, LMOD and SMOD exercise, the mean ± SE changes in systolic BP were –5 ± 3, –1 ± 7 and 5 ± 6 mm Hg, respectively. The respective changes in MAP were 2 ± 3, 1 ± 3 and 5 ± 2 mm Hg. Only the changes in systolic BP and MAP following INT and LMOD were different to control values ($P < 0.04$, Fig. 1). The changes following INT and LMOD were not different to each other for both the systolic BP and MAP variables ($P > 0.5$). Mean post-exercise responses for diastolic BP (DBP) were not significantly different from CON following all three doses of exercise ($P = 0.147$).

The minimum post-exercise values of systolic BP following INT, LMOD and SMOD were –15 ± 4, –12 ± 6 and –6 ± 5 mm Hg. All these changes were significantly different from CON ($P < 0.02$), although it can be seen that the reduction in systolic BP was smallest following SMOD. Similarly, the minimum values in MAP following all of the exercise doses were significantly different to CON ($P < 0.02$). The minimum values of diastolic BP were –5 ± 1, –5 ± 2 and –6 ± 1 mm Hg for INT, LMOD and SMOD, respectively. These values were all significantly lower than CON ($P = 0.02$) but were not different to each other.

The time the minimum value occurred for systolic BP and MAP following exercise was not significantly different between exercise doses and generally occurred between 2 and 4 min following cessation of exercise (Fig. 1, $P > 0.15$). The minimum value of diastolic BP generally

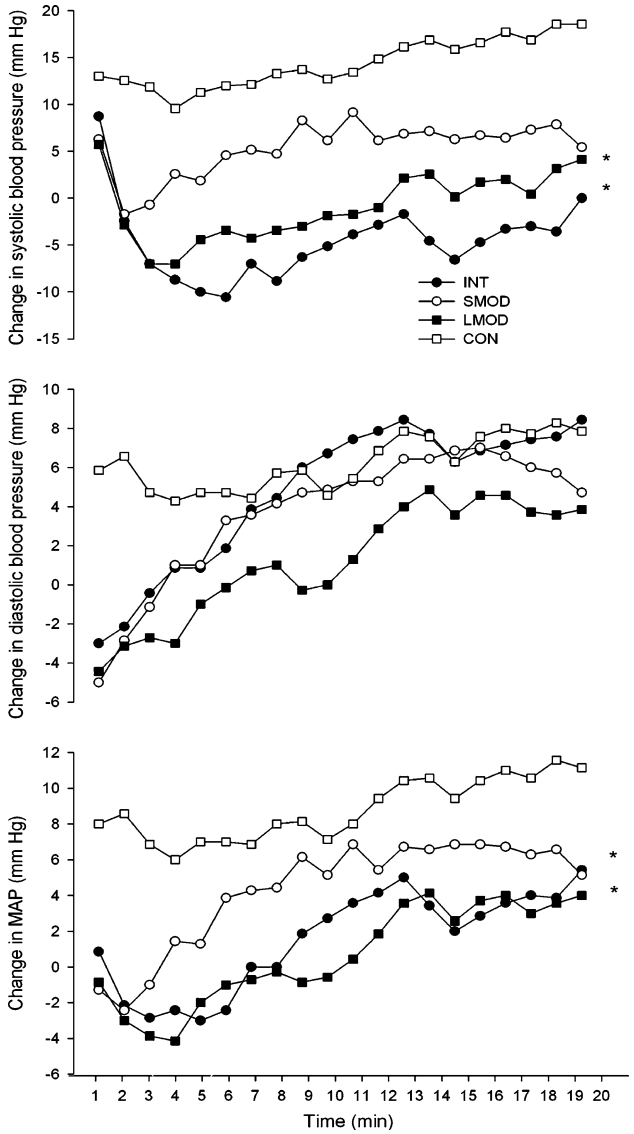

Fig. 1 Post-exercise systolic, diastolic and mean arterial blood pressure responses following INT, SMOD, LMOD and CON exercise trials. *Significantly different to CON when averaged over whole post-exercise period ($P < 0.05$)

occurred earlier, close to the cessation of exercise. No significant difference between the exercise trials in time of diastolic BP minimum was found ($P > 0.68$).

Post-exercise central responses

The post-exercise responses of CO and HR were generally greater (and SV lower) following the INT dose of exercise as shown in Fig. 2. Comparison of the post-exercise summary statistics for mean and minimum confirm these observations. Mean ± SE responses following the INT dose of exercise were significantly greater in magnitude for HR (33 ± 4 beats min^{-1}) and CO (1.9 ± 0.3 l min^{-1}) and significantly lower for SV (–15.4 ± 3.2 ml) compared to other doses of exercise and CON ($P < 0.0005$). Mean changes in HR and CO following LMOD and SMOD doses of exercise were significantly greater than control ($P < 0.0005$) but not to each other. The responses of SV following SMOD and LMOD doses of exercise however, did not differ from control or each other ($P < 0.10$).

The minimum post-exercise values following the INT dose of exercise for CO, HR and SV were 0.6 ± 0.3 l min^{-1}, 25 ± 5 beats min^{-1} and –26.1 ± 3.2 ml, respectively. All these values were significantly different from CON ($P < 0.04$). The time the minimum value occurred for CO was 11 min following the INT dose of exercise. The time the minimum value occurred in the post-exercise period was not different between all exercise trials for HR, CO and SV ($P > 0.07$).

Post-exercise peripheral responses

It can be seen in Figs. 3, 4, 5 that the post-exercise responses of TPR, CVC and FVC were significantly different following the INT dose of exercise. Comparison of the post-exercise summary statistics for mean and minimum confirm these observations. Mean ± SE changes following the INT dose of exercise was significantly lower for TPR (–3 ± 1 mm Hg l^{-1} min^{-1}) and significantly greater in magnitude for CVC (16 ± 4%) and FVC (0.07 ± 0.03 ml·100 ml $tissue^{-1}$ min^{-1} mm Hg) compared to all other doses of exercise and CON ($P < 0.04$).

The minimum value of TPR following the INT dose of exercise was significantly lower than all trials ($P < 0.0005$). There was no difference in the time the post-exercise minimum value occurred for TPR ($P = 1.141$) all values occurred immediately following the cessation of exercise. The minimum values of CVC in the post-exercise period generally occurred later (18–20 min) than those of other variables. There was no difference in the time of minimal CVC between exercise trials ($P > 0.27$).

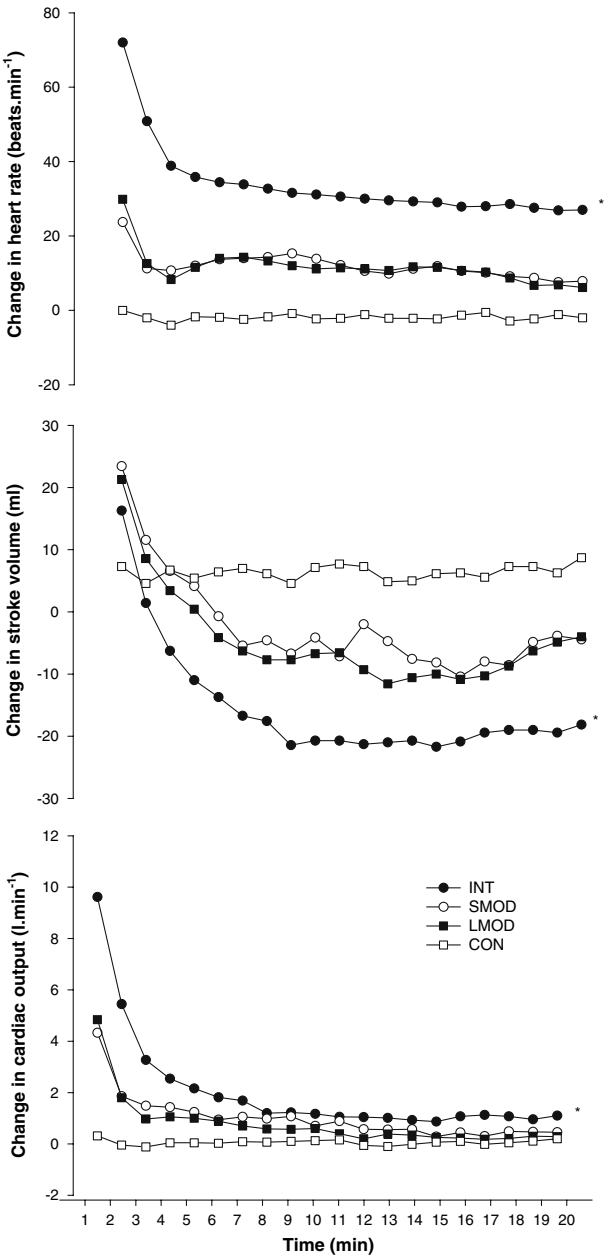

Fig. 2 Post-exercise heart rate, stroke volume and cardiac output responses following INT, SMOD, LMOD and CON control exercise trials. *Significantly different to CON when averaged over whole post-exercise period ($P < 0.05$)

Post-exercise thermoregulatory responses

The post-exercise response of core body temperature was generally greater following all exercise doses compared to control as shown in Fig. 6. This is confirmed by the comparison of the mean and minimum summary statistics. Mean ± SE changes in core body temperature were 0.6 ± 0.2, 0.7 ± 0.5, 0.5 ± 0.1°C for INT, LMOD and SMOD, respectively. These values were all significantly greater than CON ($P < 0.0005$) but were not different to

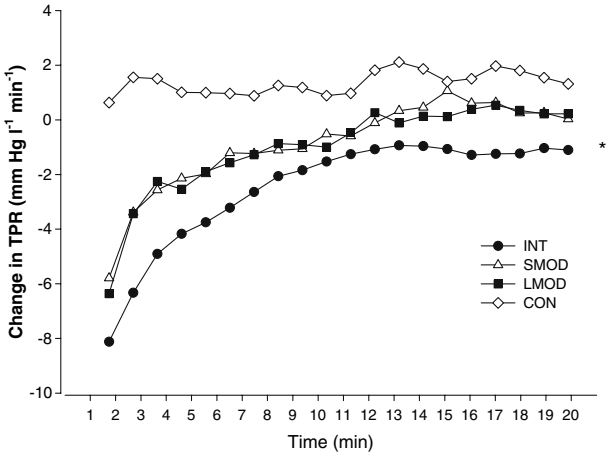

Fig. 3 Post-exercise total peripheral resistance following INT, SMOD, LMOD and CON trials. *Significantly different to CON when averaged over whole post-exercise period ($P < 0.05$)

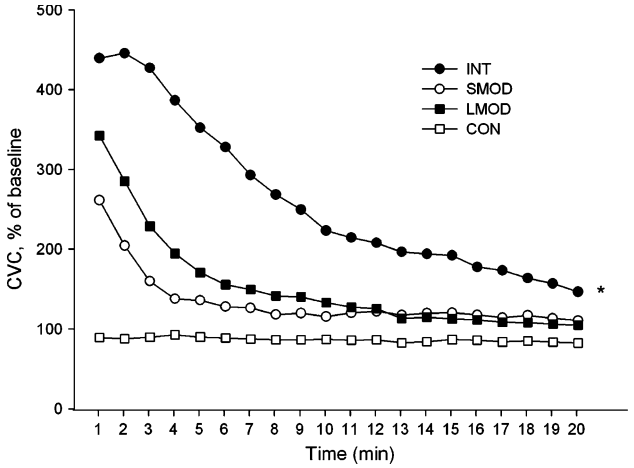

Fig. 4 Post-exercise cutaneous vascular conductance responses following INT, SMOD, LMOD and CON trials. *Significantly different to CON when averaged over whole post-exercise period ($P < 0.05$)

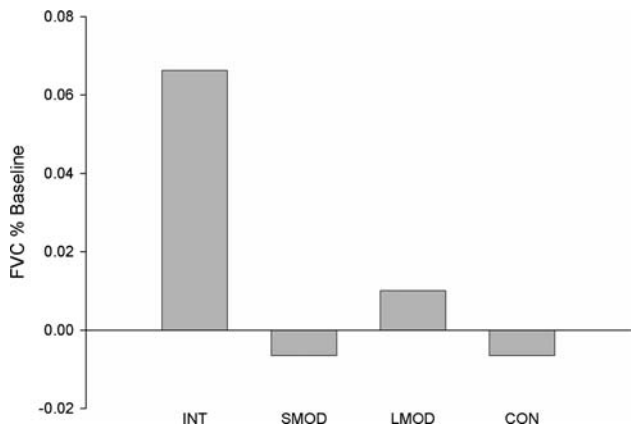

Fig. 5 Post-exercise forearm vascular conductance responses following INT, SMOD, LMOD and CON trials

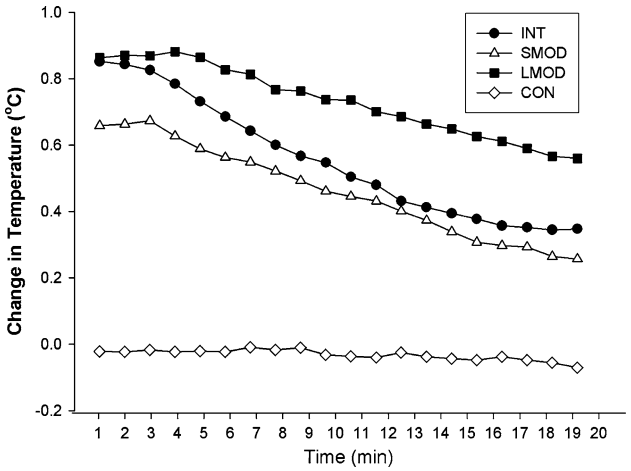

Fig. 6 Post-exercise core body temperature following INT, SMOD, LMOD and CON trials

each other. The post-exercise minimum values of core body temperature were significantly greater following all exercise doses compared to CON ($P < 0.0005$) but not to each other. The time the minimum value occurred following exercise was not significantly different between exercise trials.

Discussion

In the present study, BP and hemodynamic variables were compared before and immediately after exercise in four experimental trials to examine if exercise intensity (INT vs. SMOD), duration (LMOD vs. SMOD) or total work done (INT vs. LMOD) was the most important factor determining the extent of PEH in normotensive individuals. The major finding was that the acute hypotensive response during the 20 min after exercise is different for bouts that differ in terms of intensity and duration, but not in terms of total work completed. This finding suggests that total work done is the most important factor in determining the degree of acute PEH for normotensive individuals. This BP response was apparent despite other cardiovascular adjustments, including CO and TPR, being greater after the bout of higher intensity, shorter duration exercise (INT) compared to LMOD. This finding is unique and may mean that BP can be lowered by a bout of relatively low intensity exercise, as long as it is prolonged in nature. As a result of this finding, exercise could be prescribed at a lower intensity, for a longer duration and would elicit a beneficial effect of lowering BP immediately following exercise. Although, the current data is specific to normotensive individuals, such guidelines may be relevant to a hypertensive population, since post-exercise BP reductions are greater in magnitude in hypertensive compared to normotensive individuals (Kenney and Seals

1993; Thompson et al. 2001). Furthermore, lower intensity exercise would be advantageous as it would reduce the risk of an adverse cardiac event associated with high intensity exercise in vulnerable populations including hypertensive individuals (Lemaitre et al. 1999).

The results of this study showed that the overall PEH response was similar following exercise bouts of equivalent dose (INT and LMOD). The pattern of hypotension was such that BP dropped immediately after the cessation of exercise and either transiently increased towards baseline levels (in the case of systolic BP and MAP) or transiently increased above the baseline level (in the case of diastolic BP) in all trials. The fall in BP is similar between exercise bouts of varying intensities, but the overall reduction (i.e. taking the time course into consideration) is greater with a higher dose of exercise. The systolic BP and MAP data suggest that the overall magnitude of PEH differs due to exercise intensity (Piepoli et al. 1994; Forjaz et al. 2004) and exercise duration (Forjaz et al. 1998), which supports previous research work. Furthermore, the lower intensity, short duration bout of exercise (SMOD) did not show a similar overall reduction in BP, which supports the higher intensity, greater PEH debate.

Given that BP is a product of CO and TPR and the general consensus within the literature is that PEH is due to a persistent drop in systemic vascular resistance that is not completely offset by increases in CO (Halliwill 2001) we also examined the contribution of these variables. The results from the central hemodynamic responses indicate that exercise intensity elicits a greater change post-exercise in and CO, HR and SV and exercise duration has no effect on these variables post-exercise. All three variables showed a significantly greater overall change following the INT exercise bout. The HR and SV findings support previous research examining the effect of exercise intensity on PEH (Forjaz et al. 2004). However, Forjaz et al. (2004) reported no effect of intensity and a rise in CO during the post-exercise period. Nevertheless, contradictory findings, when measuring CO post-exercise, are not unusual and could be attributed to differing measurement techniques.

The reduction in SV post-exercise likely indicates a reduction in venous return due to the lack of a muscle pump post-exercise suggesting a decrease in end-diastolic volume due to peripheral pooling. This reduction in venous return could have caused an unloading of the cardiopulmonary baroreceptors, which in turn would cause greater HR values. Such a response supports previous research suggesting post-exercise hypotension is associated with a desensitization of barorecptors (Somers et al. 1985), which is possibly linked to hydration status (Charkoudian et al. 2003). Although baroreflex sensitivity or hydration status were not measured in the current study it is interesting to note that both HR and SV showed greater responses following the INT exercise bout. This could be suggestive of an influence of exercise intensity on baroreflex sensitivity. There is limited data examining cardiovascular control following exercise. Therefore, further research into post-exercise effects of exercise intensity on baroreflex sensitivity is warranted.

The changes in peripheral variables followed the same pattern as the central variables. The results indicate post-exercise responses in TPR and FVC are more pronounced with higher intensity exercise bouts irrespective of duration or total work done. Therefore, greater vasodilation occurs and as a consequence greater peripheral pooling with higher intensity exercise in both the previously active and non-active skeletal muscle sites during the post-exercise period. This finding also supports the theory of unloading of the cardiopulmonary baroreceptors post-exercise. Furthermore, the decline in TPR occurred following all exercise bouts within this study, which indicates the dose of exercise in all of the trials in this study was enough to evoke immediate neural and vascular components that have been associated with sympathetic vascular regulation; which has been proposed as a mechanism for PEH (Halliwill 2001).

Elevated core body temperature was present following all exercise doses and did not show any differences. This finding suggests that temperature is not simply intensity dependent, which contrasts previous research (Kenny and Niedre 2002). However, the exercise intensity of 93% $\dot{V}O_{2\,max}$ used in that study was much higher than that used in our current study or other research by the same group. Core body temperature remained elevated throughout the 20 min post-exercise period. This is in contrast to the response of skin blood flow that is usually a mechanism for heat loss. Cutaneous vascular conductance showed a gradual decline throughout the post-exercise period and a significant effect of exercise intensity with a greater response following the INT exercise bout. These responses in skin blood flow and temperature have been reported previously by Wilkins et al. (2004) who found that skin blood flow does not play and obligatory role in PEH. Furthermore, the data in this study provides support for a cardiopulmonary baroreceptor-mediated influence on post-exercise skin blood flow in order to increase TPR, and suggests that post-exercise temperature regulation may be preceded by the need to regulate post-exercise blood pressure as proposed by Kenny and Niedre (2002).

In summary the unique finding from this study is that the magnitude of acute PEH is clinically similar when the "dose" (intensity × duration) of exercise is the same for normotensive individuals. Therefore, a lower intensity longer duration dose of exercise may yield the same beneficial BP lowering effect as an equivalent dose of higher intensity exercise. It should be noted that, in the present

study, PEH was examined under controlled conditions of posture for 20 min after exercise in order to track the changes in several other cardiovascular variables besides blood pressure. Other researchers have adopted a more longitudinal approach to investigations of PEH (Pescatello et al. 1991, 2004b). These researchers were not able to measure the range of haemodynamic variables that we have examined, but they were able to measure ambulatory blood pressure during everyday living for 24 h. Future research work might examine the influence of exercise intensity, whilst controlling for total work done, on these more long-term post-exercise responses of BP.

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