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ORIGINAL ARTICLE

The acute post-exercise response of blood pressure varies with time of day

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Abstract The *reactivity* of ambulatory blood pressure following a given change in everyday physical activities is highest in the morning. Whether the acute response of blood pressure following a *controlled* bout of steady-state exercise is influenced by time of day is examined in this study. After 45 min of supine rest, 12 male normotensives completed 30 min of cycling at 70% $\dot{V}O_{2peak}$ which began at either 0800 or 1600 hours. Arterial blood pressure, cardiac output, total peripheral resistance, cutaneous blood flow and temperature were determined before, and up to 90 min after, exercise. Mean ± SE arterial pressure, averaged over the acute (20-min) period, reduced by 7 ± 2 mmHg following exercise at 1600 hours but increased by 3 ± 3 mmHg following exercise at 0800 hours ($P = 0.03$). Total peripheral resistance fell by 4.2 ± 0.8 mmHg l^{-1} min^{-1} after exercise at 1600 hour, but increased slightly by 0.1 ± 0.5 mmHg l^{-1} min^{-1} after morning exercise ($P = 0.02$). We conclude that the acutely hypotensive effects following 30 min of steady state exercise are less marked in the morning, probably because the exercise-mediated decrease in peripheral resistance is not as apparent at this time of day.

Keywords Cardiac output · Total peripheral resistance · Blood flow · Diurnal variation

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Introduction

In most individuals, resting blood pressure (BP) exhibits circadian variation that is characterised by a nocturnal fall and a rise during the hours after waking known as the 'morning surge' (Kaplan 2003; Millar-Craig et al. 1978). This circadian variation in resting BP is similar to that of the incidence of cardiovascular events. Myocardial infarction, sudden cardiac death and stroke show peak incidences between 0600–1200 hours and the lowest incidences during the night-time hours (Kario et al. 2004; Muller 1999). The morning surge in BP has been implicated in disturbing vulnerable plaques, which in turn can cause an acute cardiac event (Muller et al. 1989).

The mechanism for the early morning surge in BP may be due to an endogenous circadian rhythm, the arousal effects of awakening or the activation of the sympathetic nervous system and other haemodynamic adjustments after arising from bed (Khoury et al. 1992). Waking from sleep, adoption of an upright posture and initiation of physical activity all occur in the morning at the time of the BP surge. Leary et al. (2002) demonstrated that the change in physical activity associated with the act of rising from bed and beginning ambulation within the first 2 h after waking is related to this BP rise in the morning. A relationship between physical activity and changes in BP has also been observed at other times of day, e.g. during the daytime and sleep periods (Kario et al. 1998). A relevant question is whether such relations between physical activity and BP are affected by time of day. To begin to answer this question, a regression-based index of 'BP reactivity' was employed in a recent study (Jones et al. 2006) to describe the rate of change in BP measured 15 min after a change in physical activity for twelve 2-h data bins spaced over a 24 h period. The results of this study, revealed, for the first

time, that the *response* of ambulatory BP to everyday physical activities varies with time of day (Jones et al. 2006). Blood pressure following a unit change in physical activity was found to show the highest reactivity between 0800 and 1000 hours. Nevertheless, it should be noted that in the previous study (Jones et al. 2006), general physical activity was measured using an accelerometer-based device. Participants also went about their everyday lives, sleeping and eating when they desired during data collection and no controlled exercise intervention was administered.

Knowledge about the response of BP following exercise at different times of day is limited. In one of the few relevant studies, Park et al. (2005) examined the responses of ambulatory BP following a bout of exercise in the morning (between 0600 and 0800 hours) and the afternoon (1700–1900 hours). Park et al. (2005) reported that afternoon exercise exhibited a greater reduction in systolic BP during sleep in participants who did not normally exhibit a 10% or greater reduction in average night time BP (i.e. 'non-dippers') compared to participants who did normally exhibit this reduction (i.e. 'dippers'). In the study by Park et al. (2005), medicated hypertensive individuals were recruited and the aim was to investigate how exercise in the morning and afternoon affected the magnitude of BP 'dipping' during sleep. Conversely, the focus of the present study is on the acute reduction in BP that has been found to occur following exercise. This phenomenon, known as post-exercise hypotension (PEH), can occur within a few minutes after cessation of exercise, although it may last for up to 22 h in hypertensive individuals (Pescatello et al. 2004). In view of the recently reported morning increase in BP reactivity to everyday physical activities, (Jones et al. 2006) it would be interesting to examine whether the PEH is affected by time of day for two reasons. First, in terms of chronobiological mechanisms, most controlled experiments on circadian rhythmicity of cardiovascular function have been conducted whilst participants are at rest in 'constant routine' protocols (Kerkhof et al. 1998; Van Dongen et al. 2001). Although masking effects on resting rhythms can be quantified using such protocols, they are not as useful in quantifying the endogeneity of circadian variation in the *response* of a physiological variable to a stimulus (e.g. exercise). Second, from a public health perspective, exercise is often prescribed as a non-pharmacological treatment for hypertension due to its BP lowering effects. Pescatello and Kulikowich (2001) conducted a meta-analysis of studies in which post-exercise BP was examined. They highlighted the fact that many researchers do not disclose the time of day of exercise. Therefore, the aims of the present study were to examine whether the acute post-exercise responses of BP are altered by time of day and to explore the haemodynamic links between this variation and that observed in other variables relevant to cardiovascular function. We hypothesised that the acute post-exercise reduction in BP is less pronounced in the morning than in the afternoon.

Methods

Participants

Following an estimation of the sample size required for the primary comparisons (see "Statistical analysis"); 12 normotensive physically active males participated in the study. Participants were aged 26 ± 5 years, had a body mass of 74.5 ± 6.2 kg, were 1.78 ± 0.10 m in height and had a $\dot{V}O_{2peak}$ of 47.5 ± 7.3 ml kg^{-1} min^{-1} (mean ± SD). All participants were non smokers, had no history of cardiovascular disease, were not taking any medication and engaged in regular physical activity (defined as greater than 2 h per week). 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.

Research design

Participants attended the laboratory on four separate occasions with the first visit for familiarisation purposes, the second visit for measurement of peak oxygen uptake and the final two visits for completion of the main experimental trials; morning and afternoon exercise. These two trials were counterbalanced in order, and were separated by 7–10 days. Exercise was performed after a 4-h fast, 12-h abstinence from caffeine, 24-h abstinence from alcohol and strenuous exercise.

Familiarisation

Participants first attended the laboratory for familiarisation and anthropometric measurements. During this session, height (m), body mass (kg) and resting BP (mercury sphygmomanometer) were determined. Resting BP was determined from the average of three measurements.

Measurement of peak oxygen uptake

On the second visit to the laboratory, peak oxygen uptake was determined using a progressive continuous protocol (Bird and Davison 1997). Participants performed 10 min of submaximal cycling (Kettler Sport, Worcestershire, UK) as a standard warm-up. Power output was set initially at 50 W and increased in increments of 25 W every 2 min until volitional exhaustion or 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 rate (i.e. watts) corresponding to 70% $\dot{V}O_{2peak}$ was calculated using a linear regression equation.

The experimental protocol

Participants reported to the laboratory at 0700 hours ready to begin exercise at 0800 hours in the morning exercise condition, and at 1500 hours ready to begin exercise at 1600 hours in the afternoon exercise condition. The experimental protocol at each time of day consisted of two phases, a laboratory phase consisting of 45-min of supine rest, 5-min of seated rest on the cycle ergometer, 30-min of semi-supine cycling at 70% $\dot{V}O_{2peak}$ and 20-min of seated post-exercise rest on the cycle ergometer; and a subsequent ambulatory phase consisting of a 1 h ambulatory BP recording period after leaving the laboratory and going about everyday activities.

The laboratory phase began with a 45-min period of rest in the laboratory. During this time period, resting BP was measured using a TM-2430 ambulatory BP monitor (A&D Company Ltd, UK). The ambulatory BP monitor was fitted to the upper arm according to practical guidelines outlined by the European Society of Hypertension (O'Brien et al. 2005) and, during this rest period, was calibrated according to British Hypertension Society guidelines (O'Brien et al. 1990). Following successful calibration, the average of three BP measurements was recorded as the baseline measurement for the later ambulatory phase of the protocol.

Measurement procedures during the laboratory phase

The ambulatory monitor was removed following the baseline measurements and participants moved to the cycle ergometer and were fitted with the Portapres device, laser Doppler probes and the temperature recording equipment. Baseline measurements from this equipment were recorded for 5-min before exercise began (baseline measurements for the laboratory phase). The 30-min of semi-supine cycling at 70% $\dot{V}O_{2peak}$ was identical to that previously described (Jones et al. 2007) and to ensure the participants were cycling at the correct intensity the mechanical resistance (Watts) was kept constant during the exercise bout. Following cessation of exercise, participants remained on the cycle ergometer (seated upright position) for a 20-min post-exercise recording period.

During the exercise protocol, 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 surface attached to an electrically driven screw-thread platform. This device in combination with the semi supine cycle ergometer allowed the arms to be positioned either side of the cycle ergometer to limit arm movement during the exercise and to enable the same arm positioning for the portapres and skin blood flow measurement devices.

Blood pressure was measured continuously from the middle and/or index finger on the left hand using a Portapres device (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 BP 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, heart rate and cardiac output 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, using 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. 2002) and cardiac output (Nieminen et al. 2002; Stok et al. 1993). Total peripheral resistance was calculated by the ratio of mean arterial pressure (MAP) to cardiac output (total peripheral resistance = MAP/cardiac output).

Red blood cell flux was measured as an index of cutaneous blood flow using laser-Doppler flowmetry (Periflux System 5001, Perimed Instruments, Jarfalla, Sweden). Participants were seated on the cycle ergometer for at least 10-min prior to any measurements being recorded; this was performed to allow sufficient time for equilibration of blood volume throughout the body. Laser-Doppler probes (455 probes, Perimed, Suffolk, UK) were attached to the skin sites using adhesive discs. One probe was attached to the mid-anterior ventral aspect of the left forearm (forearm cutaneous blood flow) and another probe was attached to the mid anterior thigh, midline, halfway between the inguinal line and the patella (thigh cutaneous blood flow). 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 skin blood flow data are presented as CVC increase relative to baseline.

Core temperature was measured using a thermometric temperature sensor (CorTemp™ Disposable Temperature

Sensor HQInc, FL, USA), which was ingested prior to sleep the night before morning exercise and a minimum of 4 h prior to afternoon exercise. Core temperature was monitored continuously throughout the exercise protocol and data were recorded every minute.

The post-exercise ambulatory phase

The equipment used for the laboratory phase was removed and participants were allowed ~10 min to shower and change clothing before being fitted with the ambulatory BP monitor once again, to begin the post-exercise ambulatory phase. During the ambulatory phase, participants were instructed to leave the laboratory and follow their normal daily routine. The ambulatory BP monitor was programmed to obtain a reading every 15-min, which began ~30 min and ended ~90 min after exercise (i.e. 1-h of ambulatory BP data). These additional measurements of BP were made in order to cross-validate the readings from the portapres device as well as examine whether any changes in BP are maintained during 'everyday' activities following a controlled exercise bout (MacDonald et al. 2001).

Statistical analysis

The primary outcome variables were systolic, diastolic and MAP averaged over the 20-min post-exercise period and subtracted from their respective baseline values. The primary comparison for all dependent variables was between the 0800 hours and 1600 hours exercise bouts. For estimation of sample size, it was deemed that a mean 20-min post-exercise difference of 5 mmHg in systolic and/or diastolic BP between the two times of day would be clinically important and it was estimated that seven participants would allow this difference to be deemed statistically significant (statistical power = 80%, SD of differences ≤4 mmHg using a one tailed paired *t*-test). The BP lowering effects of an isolated exercise session (acute) in hypertensive individuals is 5–7 mmHg (Pescatello et al. 2004). Therefore, we deemed a 5 mmHg difference in normotensive individuals an important difference, given that BP reductions are generally more pronounced in people with hypertension (Pescatello et al. 2004).

Absolute baseline data were averaged over a 5-min period and paired t-tests were performed to explore time of day differences. Post-exercise measures were averaged into 5-min periods and delta changes from the pre-exercise baseline were calculated. These data were analysed using a two factor (time of day vs post-exercise time) general linear model with repeated measures. The three post-exercise ambulatory BP readings were averaged and subtracted from the baseline values. Paired *t*-tests were employed to explore any differences in the change in ambulatory blood pressure between the two times of day. All data were analysed using Statistical Package for the Social Sciences (version 14). Data are presented in the text as mean ± SE and 90% confidence intervals (Sterne et al. 2001). Exact *P* values are cited (values of *P* of "0.000" provided by the statistics package are reported as "<0.0005"). Statistical significance was delimited at $P < 0.05$. Nevertheless, *P* values between 0.05 and 0.10 were also considered to be suggestive of null hypothesis rejection on the basis of Fisher's original treatise (Sterne et al. 2001).

Results

Baseline (pre-exercise) values for all measured variables in the laboratory phase were averaged of the 5-min recording period and differences between times of day are presented in Table 1.

Figure 1 presents the differences in acute post-exercise changes in BP with times of day. Significantly greater post-exercise reductions were evident at 1600 hours for systolic BP ($P = 0.01$; 90% CI = −22 to −5 mmHg), diastolic BP

Table 1 Differences between mean baseline values (laboratory phase) measured prior to exercise at 0800 hours and 1600 hours

Variable	Mean ± SE		90% CI of the difference	*P*-value
	0800 hours	1600 hours		
Systolic BP (mmHg)	124 ± 5	141 ± 5	−26 to −8	0.005*
Diastolic BP (mmHg)	66 ± 2	73 ± 3	−12 to −2	0.03*
MAP (mmHg)	83 ± 2	92 ± 3	−15 to −3	0.01*
Heart rate (beats min^{-1})	69 ± 4	62 ± 3	2 to 13	0.03*
Stroke volume (ml $beat^{-1}$)	96 ± 6	99 ± 5	−7.6 to 2.1	0.33
Cardiac output (l min^{-1})	6.4 ± 0.3	6.1 ± 0.3	0.1 to 0.7	0.04*
Total peripheral resistance (mmHg l min^{-1})	13.3 ± 0.7	15.8 ± 1.1	−3.8 to −2.1	0.005*
Core body temperature (°C)	36.9 ± 0.1	37.1 ± 0.1	−0.3 to 0.0	0.09
Forearm CVC (PU)	0.09 ± 0.01	0.09 ± 0.01	−0.01 to 0.02	0.39
Thigh CVC (PU)	0.08 ± 0.01	0.09 ± 0.01	−0.03 to 0.02	0.55

* Indicates significant difference between time of day

($P = 0.03$; 90% CI = −22 to −14 mmHg) and MAP ($P = 0.03$; 90% CI = −16 to −2 mmHg) compared to after 0800 hours exercise. There was also evidence of interactions between time of day and post-exercise time for both diastolic BP ($P = 0.06$) and MAP ($P = 0.05$). Both diastolic BP and MAP remained below baseline levels for the entire duration of the post-exercise period following exercise at 1600 hours. After the cessation of morning exercise diastolic BP and MAP were initially lower than baseline, and then 10-min post-exercise increased to higher than baseline levels (Fig. 1). The mean ± SE difference in diastolic BP was 6 ± 3 and 11 ± 15 mmHg for 5 and 20-min post-exercise time points. The respective mean ± SE difference in MAP was 7 ± 3 and 12 ± 4 mmHg.

The acute post-exercise changes in cardiac output and total peripheral resistance are presented in Fig. 2. A greater post-exercise change was found following exercise at 1600 hours compared to 0800 hours in cardiac output ($P = 0.01$; 90% CI = 0.4–1.7 l.min^{-1}) and a greater reduction in total peripheral resistance at 1600 hours compared to 0800 hours ($P = 0.02$: 90% CI = −7.3 to −1.4 mmHg l^{-1} min^{-1}). Post-exercise cardiac output was elevated above baseline but the profile was a gradual decline throughout the 20-min post-exercise period at both times of day. Total peripheral resistance remained below baseline for the entire duration of the post-exercise period following exercise at 1600 hours. Following exercise at 0800 hours total peripheral resistance was lower than baseline, and then 10-min post-exercise increased to higher than baseline. There was no evidence of an interaction in time course between time of day and post-exercise time ($P = 0.16$).

The acute post-exercise changes in both heart rate, stroke volume and forearm cutaneous vascular conductance were not different between the two times of day ($P > 0.16$). The acute changes for thigh cutaneous vascular conductance were 40.0 ± 7.6 and 52.5 ± 14.5% for morning and afternoon exercise, respectively ($P = 0.56$).

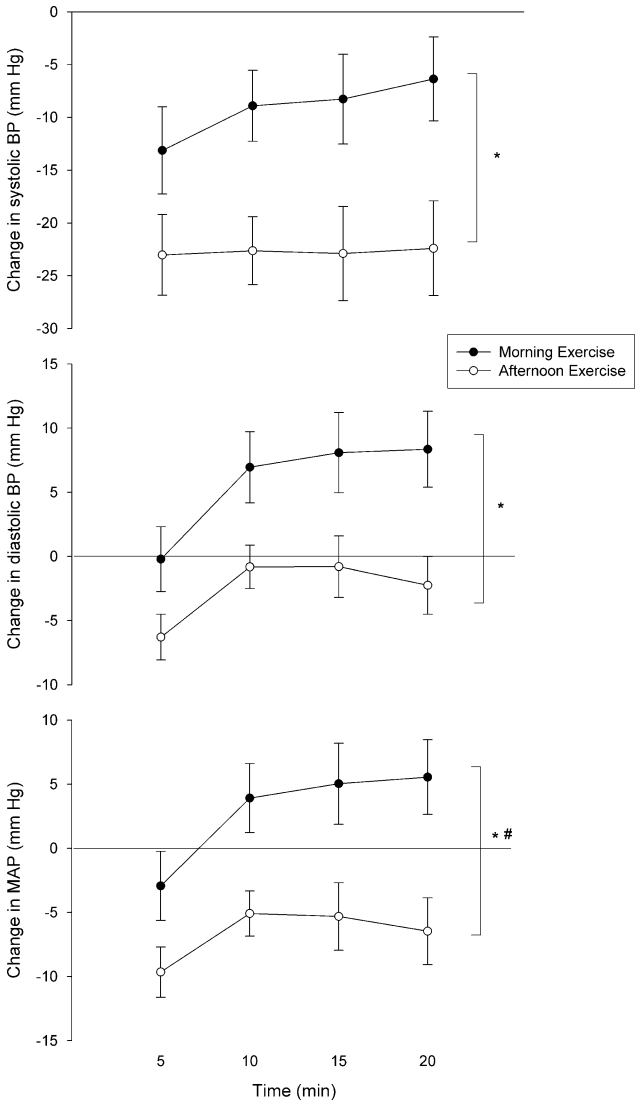

Fig. 1 Mean post-exercise changes in systolic BP (*top*), diastolic BP (*middle*) and MAP (*bottom*) between time of day. *Indicates significant difference between time of day when averaged over the whole 20-min period. #Indicates significant interaction between time of day and post-exercise time

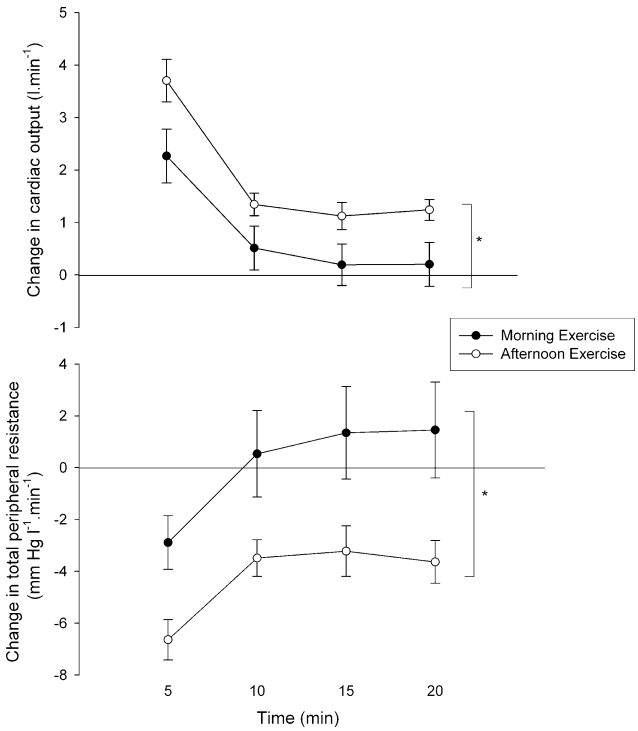

Fig. 2 Mean post-exercise changes in cardiac output (*top*) and total peripheral resistance (*bottom*) between time of day. *Indicates significant difference between time of day when averaged over the whole 20-min period

However, there was evidence of a significant interaction between time of day and post-exercise time ($P = 0.02$, Fig. 3). The difference in thigh cutaneous vascular conductance between morning and afternoon exercise was greater at 20-min (mean ± SE difference 1.0 ± 11.5%) compared to 5-min (mean ± SE difference −22.8 ± 13.9%) post-exercise. There was also evidence of a greater reduction in core temperature following exercise at 1600 hours compared to at 0800 hour ($P = 0.04$; 90% CI = −4.4 to −0.1°C) and of an interaction between time of day and post-exercise time ($P = 0.09$, Fig. 4).

As a cross-validation of the readings from the Portapres device, ambulatory BP was measured for 1-h, which equates to 60–90 min after exercise cessation in the laboratory phase. Figure 5 presents the ambulatory data averaged over this 1 h period. Similar to the BP readings from the portapres device pre exercise baseline systolic (90% CI = −12 to −1 mmHg, $P = 0.06$), diastolic (90% CI = −9 to −1 mmHg, $P = 0.07$) and MAP (90% CI = −10 to −1 mmHg, $P = 0.05$) were generally lower in the morning compared to the afternoon. In addition, the trends for ambulatory BP following exercise were also similar to the portapres device with diastolic BP (90% CI = 7–28 mmHg) and MAP (90% CI = 6–25 mmHg) significantly greater in the morning compared to the afternoon ($P < 0.02$). Post-exercise ambulatory systolic BP was greater in the morning compared to the afternoon but was not statistically different (90% CI = −2 to 25 mmHg, $P = 0.16$).

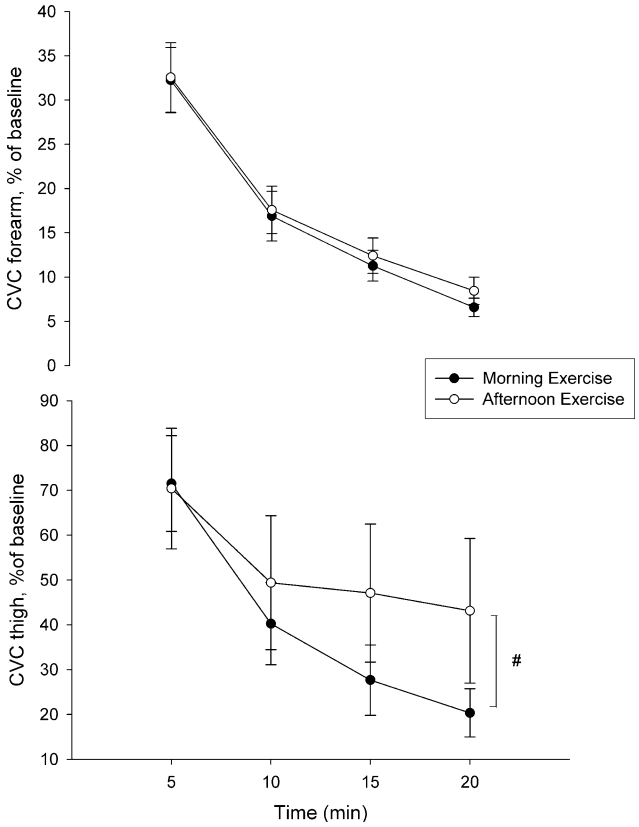

Fig. 3 Mean post-exercise changes in forearm (*top*) and thigh (*bottom*) cutaneous vascular conductance between time of day. #Indicates significant interaction between time of day and post-exercise time

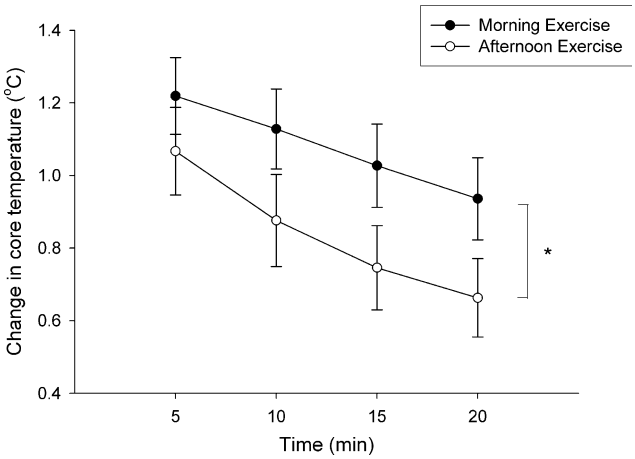

Fig. 4 Mean post-exercise changes in core temperature between time of day. *Indicates significant difference between time of day when averaged over the whole 20-min period

Discussion

The present study is the first to examine, in detail, whether time of day alters cardiovascular function during the acute post-exercise period. Our aim was to compare the exercise-mediated change in BP and other cardiovascular variables between two times of day. Both steady-state exercise bouts in the present study were performed following a period of supine rest scheduled early in the morning and in the afternoon. While we found the conventional PEH phenomenon in the afternoon, the BP changes following exercise in the morning were much less marked. Indeed, we found evidence that diastolic BP and MAP increased rather than decreased during this period following exercise in the morning; a change which persisted during 60 min of subsequent everyday physical activities. This observation is in accord with the results of our previous large scale descriptive study involving ambulatory BP measurements obtained from hypertensive individuals (Jones et al. 2006). The present study also provides haemodynamic insight in that the differences in BP responses between times of day are mirrored by post-exercise changes in total peripheral resistance.

The baseline BP data in the laboratory phase, collected following a prolonged period of supine rest was lowest in the morning. This observation is consistent with the results of studies involving constant routines (Kerkhof et al. 1998; Van Dongen et al. 2001). The early morning increase in BP

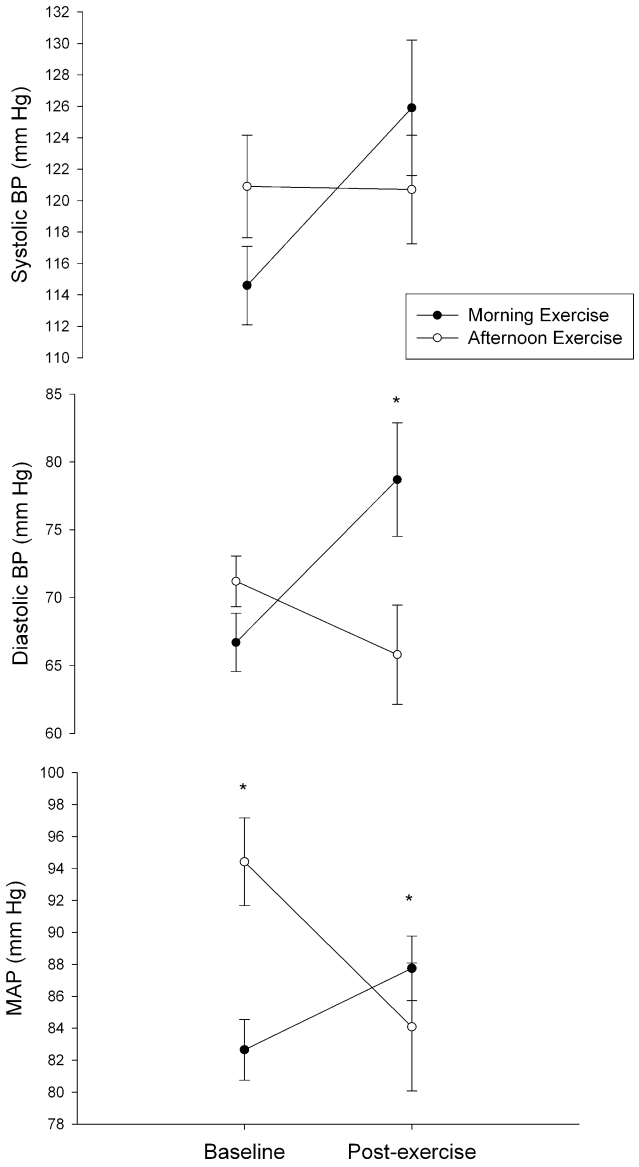

Fig. 5 Mean ambulatory systolic BP (*top*), diastolic BP (*middle*) and MAP (*bottom*) measured at baseline and between 30 and 90 min post-exercise. *Indicates significant difference between time of day

has been shown to persist for three or more hours after waking from nocturnal sleep and initiating everyday physical activity (Kario et al. 2004). Despite baseline values being higher in the afternoon, the post-exercise diastolic and MAP profiles were higher in the morning. The post-exercise change of diastolic and MAP in the afternoon was characterised by a hypotensive response (i.e. reduction), whilst in the morning an increase in diastolic and MAP above baseline values was evident. It could be argued that the greater reduction in BP in the afternoon is due to higher baseline values, i.e. post-exercise BP reduction is greater when individuals have higher initial BP (Pescatello et al. 2004; Pescatello and Kulikowich 2001). However, this is only true for systolic BP as the post-exercise change in systolic BP in the morning displayed a hypotensive response but of a lesser magnitude than that seen in the afternoon. The results of the current study indicate that exercise in the morning was followed by an increase in diastolic BP and MAP compared to baseline. Furthermore, in absolute terms the rise in diastolic and MAP in the morning was not only above baseline levels but also above the post-exercise BP values observed in the afternoon. Interestingly, this trend persisted for the duration of the ambulatory period and therefore during everyday physical activity which supports the findings of MacDonald et al. (MacDonald et al. 2001). These data provide strong evidence that the acute post-exercise response in BP varies with time of day.

Higher values of heart rate and cardiac output were observed at baseline in the morning compared to the afternoon. Circadian variation in resting heart rate and cardiac output has been reported previously and variation has been attributed to the sleep-wake cycle with an increase observed after waking and commencing activity (Veerman et al. 1995). However, the post-exercise change in cardiac output was greater in the afternoon. A possible explanation for the differences in cardiac output and the trends in heart rate and stroke volume could be due to a greater fall in central venous pressure as a consequence reduced cardiac filling and reduced end diastolic volume in the morning. Another important mediating variable for the observed post-exercise rise in morning BP is total peripheral resistance. A sustained post-exercise decrease in total peripheral resistance was evident in the afternoon which remained below baseline levels. Nevertheless, in the morning, total peripheral resistance transiently increased above baseline levels and, in absolute terms, resistance values were higher in the morning compared to the afternoon. These responses were evident despite greater baseline values being observed in the afternoon. Resting values of total peripheral resistance have also been found to be higher in the afternoon in a previous study using the same measurement device (Veerman et al. 1995). Furthermore, it is clear that the changes in total peripheral resistance found in the current study mirrored those of MAP. Since the PEH response is generally mediated by a persistent reduction in vascular resistance (Pescatello et al. 2004), our data suggest that the typical PEH response did not occur in the morning because of greater vascular resistance. Nevertheless, further research is necessary to confirm this notion, preferably using a *direct* measure of vasculature resistance rather than the estimation we employed that is derived from an algorithm based on assumptions underlying the human arterial tree.

The cutaneous vascular conductance supports the total peripheral resistance data. Cutaneous vascular conductance is a measure of peripheral flow and there was evidence of

increased displacement of blood to the thigh cutaneous vasculature in the afternoon. It is possible that greater vasodilation in the cutaneous vasculature of the exercising limbs caused a greater reduction in total peripheral resistance in the afternoon. In turn this could have contributed to the reduction in MAP in the afternoon. This pattern was not observed in the forearm, nevertheless, the forearm was relatively inactive during the exercise bout and post-exercise differences in cutaneous vascular conductance measured at sites of active and inactive limbs have been reported previously (Wilkins et al. 2004).

Potentially the time of day differences in post-exercise thigh cutaneous vasculature and thus total peripheral resistance could be influenced by the circadian rhythm in core temperature. Heat production during exercise causes increased body temperature and the thermoregulatory response of cooling via sweating and increased cutaneous blood flow is initiated which continues post-exercise. An increase in cutaneous blood flow causes dilation of the cutaneous vascular beds. However, previous research has shown a reduction in heat loss in the post-exercise period due to declining cutaneous vascular conductance when PEH is evident, (Wilkins et al. 2004) although the specific time of day for exercise was not reported. Nevertheless, peripheral vasodilation has a circadian component (Waterhouse et al. 2005) and the time of day differences in post-exercise thigh cutaneous vascular conductance suggests that the post-exercise decline in cutaneous vascular conductance is greater in the morning when the circadian rhythm of core temperature in the heat gain phase which agrees with previous research (Aldemir et al. 2000). In the afternoon, when the circadian rhythm of core temperature has reached its plateau, the post-exercise cutaneous vascular conductance decline was not as rapid and thus the decay in core temperature was greater.

The techniques utilised in this investigation were limited to non invasive measurements of cardiovascular function mostly derived from using finger plethysmography (i.e. portapres). There are a number of reports suggesting that the portapres is not a reliable measurement tool to monitor absolute BP (Imholz et al. 1998; Stok et al. 2006). Therefore, in the current study the portapres device was used to track short-term minute by minute changes (20 min) as reports suggests the portapres it is acceptable for monitoring such changes (Imholz et al. 1998). Furthermore, to corroborate the BP data from the portapres additional BP measurements were recorded using the TM-2430 ambulatory BP monitor. Although, the TM-2340 has not been directly assessed in the post-exercise period nor the data correlated with data from the Portapres device, it has been validated previously (Palatini et al. 1998). Additionally, there are concerns with non-invasive estimates of stroke volume, cardiac output and total peripheral resistance determined via mathematical models in Modelflow. Nevertheless, measurements of variables such as these are generally difficult and we felt the haemodynamic measurements from Modelflow potentially provided important insight about BP regulation in the post-exercise period. These measurements were not directly cross validated with a 'gold standard' technique during the current study. Although a recent investigation in our laboratory has shown that stroke volume estimated from Modelflow to track changes in the pre and post-exercise period are not different to stroke volume changes measured using pulse Doppler echocardiography (Harriss et al. 2007). Furthermore, Pitt et al. (2004) reported that cardiac output estimated from the portapres is useful when assessing changes in individual participants.

Regular exercise is advocated because there is substantial evidence suggesting that exercise can reduce BP (Pescatello et al. 2004). Therefore, exercise has the potential to be a key non-pharmacological tool in the management of BP. However, the findings from this current study suggest that the scheduling of exercise is important, given that time of day variation in post-exercise BP is evident. Thus, scheduling exercise in the afternoon would offer the greatest acute BP lowering effects whereas scheduling exercise in the morning may in fact cause a post-exercise rise in diastolic and mean arterial BP. Nonetheless, the findings of this study are specific to normotensive individuals performing an acute bout of aerobic exercise; it is unknown whether this pattern would persist if exercise was taken regularly in the morning over a number of weeks. Future research work could centre on a longer-term study of BP responses to a period of exercise training in the morning and afternoon.

In addition, from a clinical perspective the post-exercise rise in BP in the morning may be a cause for concern for hypertensive individuals who are attempting to lower BP via exercise. Vigorous exercise can acutely and transiently increase the risk of sudden cardiac death and acute myocardial infarction in susceptible individuals (Thompson et al. 2007). Hypertensive individuals are at greater risk of acute cardiovascular events (Pescatello and Kulikowich 2001), and BP surges have been implicated in disturbing vulnerable plaques, which in turn can cause an acute cardiac event (Muller et al. 1989). Therefore, a higher post-exercise BP would be unfavourable for those individuals at greater risk. Further research is required to confirm a post-exercise time of day difference in hypertensive individuals.

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