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ORIGINAL ARTICLE

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Hypotension following mild bouts of resistance exercise and submaximal dynamic exercise

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Abstract Our purposes were (1) to examine resting arterial blood pressure following an acute bout of resistance exercise and submaximal dynamic exercise, (2) to examine the effects of these exercises on the plasma concentrations of atrial natriuretic peptide ([ANP]), and (3) to evaluate the potential relationship between [ANP] and post-exercise blood pressure. Thirteen males [24.3 ± (2.4) years] performed 15 min of unilateral leg press exercise (65% of their one-repetition maximum) and, 1 week later, ≈15 min of cycle ergometry (at 65% of their maximum oxygen consumption). Intra-arterial pressure was monitored during exercise and for 1 h post-exercise. Arterial blood was drawn at rest, during exercise and at intervals up to 60 min post-exercise for analysis of haematocrit and [αANP]. No differences occurred in blood pressure between trials, but significant decrements occurred following exercise in both trials. Systolic pressure was ≈20 mmHg lower than before exercise after 10 min, and mean pressure was ≈7 mmHg lower from 30 min onwards. Only slight (non-significant) elevations in [αANP] were detected immediately following exercise, with the concentrations declining to pre-exercise values by 5 min post-exercise. We conclude that post-exercise hypotension occurs following acute bouts of either resistance or submaximal dynamic exercise and, in this investigation, that this decreased blood pressure was not directly related to the release of αANP.

Key words Post-exercise hypotension · Intra-arterial pressure · Submaximal dynamic exercise · Resistance exercise

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Introduction

It has been estimated that one in five individuals suffer from hypertension (Joffres et al. 1992). Although it has been known for some time that chronic exercise training can result in lower resting blood pressure in hypertensive individuals (Tipton et al. 1991), more recent studies have indicated that even an acute bout of exercise may elicit transient decreases in blood pressure (e.g. Floras and Senn 1991; Floras and Wesche 1992). In individuals with essential hypertension such decrements may average 21 and 12 mmHg for systolic (SBP) and diastolic (DBP) pressure, respectively (Wilcox et al. 1982; Bennett et al. 1984; Pescatello et al. 1991; Floras and Wesche 1992), and may persist for as long as 12–17 h (Pescatello et al. 1991; J. Hagberg personal communication). Such findings suggest that acute bouts of exercise may have the potential to act as a beneficial, non-pharmacological aid in the treatment of hypertension. Although the majority of investigations have focused on endurance exercise as the stimulus for post-exercise hypotension (PEH; Wilcox et al. 1982; Bennett et al. 1984; Floras and Senn 1991; Pescatello et al. 1991; Floras and Wesche 1992), there is also evidence which suggests that resistance exercise yields similar results (Brown et al. 1994).

Unfortunately, studies examining PEH to date have used intermittent auscultatory methods to determine blood pressure. It is well known that respiration causes blood pressure to fluctuate due to changes in intra-thoracic pressure. In addition, large fluctuations in blood pressure are evident approximately 2–5 times per minute. These fluctuations, sometimes known as Mayer waves, are of unknown origin. Given these shifts in blood pressure as well as the changes associated with exercise and movement, continuous beat-by-beat recording is needed to accurately detect the acute changes in blood pressure associated with PEH.

Atrial natriuretic peptide (ANP) is known to possess potent natriuretic and vasodilatory properties which

play an integrative role in fluid regulation and the control of blood pressure (deBold 1985). This hormone is released from the atrial granules in response to distension of the atria (Vollmar 1990) and has been shown to increase in the circulation in response to dynamic exercise (Perrault et al. 1991, 1994). Although the half life of ANP is quite short (2–3 min), it has been suggested that its haemodynamic effects persist for several hours (Espiner and Nicholls 1987). Several mechanisms may be responsible for its release during exercise, but it is generally accepted that these are secondary to the ANP release caused by the atrial distension imposed by increase in venous return (e.g. Ray et al. 1990). A number of studies have shown an increase in circulating concentrations of ANP ([ANP]) with endurance exercise (e.g. Perrault et al. 1991, 1994), suggesting that this response is due to the increased atrial distension associated with an enhanced venous return. Significant elevations of [ANP] have been found at peak exercise in the supine, compared to upright position (Ray et al. 1990; Bussieres-Chafe et al. 1994), yet, radionuclide ventriculography has demonstrated that the stroke volume augmentation that occurs in the supine position during cycle ergometry at power outputs above 25 W (including peak exercise) is negligible (Poliner et al. 1980; Steingart et al. 1984). This suggests that atrial distensions not the primary cause for ANP secretion in this instance. In addition we have observed increased circulating [ANP] in healthy young subjects in response to intensive heavy resistance exercise (MacDonald et al. 1995). Since such exercise results in marked elevations in blood pressure (MacDougall et al. 1985; Lentini et al. 1993; MacDougall 1994) but little or no change in end diastolic volume (Lentini et al. 1993), the mechanism for ANP release during weightlifting may involve factors other than atrial distension.

Although there is considerable descriptive literature on the occurrence of PEH, little is known about its underlying mechanism(s). Possibilities include decreased cardiac output (Hagberg et al. 1987) and/or peripheral resistance (Piepoli et al. 1993), increased circulating K^+ (Urata et al. 1987) and opioid-mediated decreases in sympathetic activity (Hoffman and Thoren 1988; Farrell et al. 1991). To our knowledge there have been no investigations of the possible causal relationship between exercise-released ANP and PEH.

This investigation had three purposes. The primary purpose was to evaluate the separate effects of submaximal cycling versus resistance exercise as modulators of PEH using the accuracy of intra-arterial blood pressure monitoring. A secondary purpose was to examine the stimulus for the secretion of αANP during exercise. It was hypothesised that by examining the ANP response to both cycle ergometry and heavy resistance exercise, it would be possible to uncouple the effects of a blood pressure, versus a volume load on the heart as a stimulus for ANP release. The third purpose of this study was to determine whether the occurrence of PEH is related to elevations in circulating [αANP].

Methods

Subjects

Thirteen recreationally active males aged [mean(SD)] 24.3(2.4) years, with a mean(SD) height of 175.9(5.0) cm and a mean (SD) body mass of 74.2(7.9) kg volunteered to participate in the study. After approval by the McMaster University Human Ethics Committee, subjects were advised of the risks associated with the study and provided written informed consent.

Preliminary testing

Prior to beginning the study, the subjects' maximum oxygen uptake ($\dot{V}O_{2\,max}$) was determined using an incremental cycle ergometry test to exhaustion. Using an electrically braked cycle ergometer (Eric Jaeger, Hoechberg, Germany), subjects pedalled at a cadence greater than 60 revolutions per minute (rpm). At the completion of each 2-min interval, the power output was increased by 20–60 W. Volitional exhaustion was deemed to be the point at which subjects could no longer maintain a pedal cadence of 60 rpm. Expired gases were collected using one-way air-flow valves (Hans Rudolph #2700, Hans Rudolph, Kansas City Mo, USA) and analysed on-line via an IBM-compatible computer using the TurboFit software package (Vacumetrics, Ven, Calif., USA) coupled with an AMETEK S3A/1 oxygen analyser (Applied Electrochemistry, Pittsburg, Pa, USA) and a Hewlett Packard 78356A carbon dioxide analyser (Hewlett packard, Mississauga, Ontario, Canada). Both analysers were calibrated prior to and following each test using gases of known oxygen (12.10%) and carbon dioxide (5.10%) content.

The mean $\dot{V}O_{2\,max}$ was found to be 53.9(7.4) $ml \cdot kg^{-1} \cdot min^{-1}$. For each individual, 65% of their $\dot{V}O_{2\,max}$ was calculated as the target oxygen consumption during the submaximal dynamic exercise trial. An average of 180(30) W was required to elicit 65% $\dot{V}O_{2\,max}$. Unilateral leg press strength was determined as the greatest weight that a subject could lift once with the dominant leg, through the entire range of movement (one-repetition maximum; 1RM). This resistance was determined using a progressive incremental protocol on a commercial leg press apparatus (Global Gym, model 3221-168, Global Gym Fitness Equipment, Weston, Ontario, Canada) with at least 3 min between attempts. The average 1RM weight subjects could lift was 111(15) kg. Sixty-five percent of each individual's value was termed the 65% 1 RM, and this value was used in the resistance exercise trial.

All subjects provided typical 4-day diet records (3 weekdays, 1 weekend day). From these, their average daily energy intake was calculated and pre-packaged diets were designed in order to control for caffeine and sodium intake. Since high protein ingestion has been shown to increase ANP secretion (Tam et al. 1990), subjects were asked to refrain from ingesting animal protein for 3 days prior to consumption of the pre-packaged diet. During the test day and the preceding day, subjects consumed the provided diet which contained approximately 75% carbohydrate, 20% fat and 5% protein and was low in sodium.

Experimental protocol

After a 7-h fast, the subjects reported to the laboratory and underwent auscultatory resting blood pressure measurement. Auscultatory readings averaged 130(13)/87(8) mmHg for SBP and DBP, respectively. A 3.8-cm, 20-gauge Angiocath (Becton Dickenson, Sandy, Utah, USA) was then inserted into either the brachial or radial artery ($n = 10$ brachial, $n = 3$ radial). The catheter was coupled to a saline-Heparin (Wyeth-Ayerst, Toronto, Ontario, Canada) drip equipped with a Novotrans pressure transducer (MX 800, Medex, Hilliard, Ohio, USA) for the direct measurement of blood pressure. This transducer was placed at mid-sternal level and coupled to an amplification system (Acudata, model 143, Honeywell, Denver, Colo., USA) and an on-line data acquisition package

(Windaq/200, DataQ Instruments, Akron, Ohio, USA), sampling at a frequency of 300 Hz. Calibration of the pressure-monitoring systems was completed using a mercury manometer prior to each trial. The intra-arterial catheter was calibrated to show a linear response between 0 and 300 mmHg. Reference electrodes, placed in the V5 positions, were affixed to a digital heart rate (HR) recorder which was coupled to a signal-triggered counting device (Lafayette Instrument, Model 54430, Lafayette, Ind., USA) for the determination of summed cardiac cycles. Subjects remained in a seated resting position for a minimum of 20 min prior to the collection of baseline measurements.

The initial testing session required subjects to complete 15 min of unilateral leg press exercise at 65% of their predetermined 1RM. Timing of the lifting, lowering and lockout phases of the exercise was established using a metronome. The metronome emitted an audible stimulus at a frequency of 1 Hz. Subjects were asked to maintain a cadence of 2 during the lifting phase, 1 during the lockout and 3 during the lowering phase, in time with the metronome. During the session, subjects were free to alternate to the contralateral limb as fatigue occurred. At the cessation of exercise, subjects remained seated quietly for 1 h, for continued monitoring.

Intra-arterial blood pressure was monitored continuously throughout the session, with 30-s windows recorded at rest, 5, 10, and 15 min into exercise and then 1.5 3, 5, 10, 15, 30, 45 and 60 min post-exercise for subsequent analysis. Arterial blood was sampled approximately 15 s before and after each pressure-monitoring time point. Aliquots of blood were then mixed to approximate a single sample across each of the time points listed above. The blood was obtained from the arterial catheter site and collected in chilled collection vials which contained ethylenediaminetetraacetic acid (Vacutainer, Becton Dickenson, Rutherford, N.J., USA). Capillary tubes were filled and subsequently centrifuged for haematocrit (Hct) determination in order to calculate possible shifts in plasma volume. Upon completion of the trial, the blood was centrifuged at 4°C and 3000 *g* for 30 min. Plasma was then extracted and stored at −50°C for later analysis.

The second testing session occurred 1 week later, with catheterisation, pressure, heart cycle monitoring and blood collection procedures being identical to the previous session. This trial required subjects to perform a bout of cycle ergometry at a power output which elicited 65% $\dot{V}O_{2\,max}$. Expired gases were collected to ensure that the target of 65% $\dot{V}O_{2\,max}$ was maintained. Collection times for pressures and blood samples in this trial were dependent upon the number of cardiac cycles recorded in the resistance exercise trial (e.g. if during the resistance exercise, blood pressure was taken at 5 min, at which point 800 cardiac cycles had occurred, during the cycle ergometry session pressure measurements would be collected at a time point corresponding to 800 cardiac cycles). This procedure was followed since ANP release is thought to be stimulated by the atrial distension caused by increased atrial filling during diastole. Since one would expect the total amount of ANP released to be a function of both the magnitude of the load and the duration over which it is imposed, summed cardiac cycles were held constant between the two trials in order to hold the number of occasions of diastolic filling constant. At the cessation of exercise, subjects remained quietly seated as in the initial trial.

To maintain consistency, samples were labelled corresponding to the resistance exercise trial with the following coding: BL, D5, D10, D15, 1:30P, 3P, 5P, 10P, 15P, 30P, 45P, 60P, where BL = baseline, D = during exercise, and P = post-exercise, and the number refers to the number of minutes during or post-exercise.

Analysis

Blood pressure waveforms were analysed using a Windaq data analysis program (DataQ Instruments). SBP and DBP were calculated as the highest point in the waveform and the lowest point in the waveform, respectively. Mean arterial pressure (MAP) was determined as the quotient of the integrated pressure and the duration of the time interval. The rate-pressure product (RPP) was calculated as the product of SBP and HR divided by 1000.

The plasma was analysed for human αANP using a commercially available human αANP [^{125}I] radioimmunoassay system (Amersham Chemicals, Oakville, Ontario, Canada). The intra-assay coefficient of variation was found to be ≈4.9%.

Subsequent testing

In order to assess the effects of 75 min of sitting on its own (e.g. without being preceded by exercise) on blood pressure, following the two sessions described above, two subjects returned to the laboratory for additional testing. Following arterial catheterisation, subjects remained seated in an identical manner to the previous two trials. Blood pressure and Hct were monitored as above for 75 min in order to determine the extent to which possible reductions in plasma volume due to a static seated position may have contributed to the response.

Statistical analysis

Each variable (as described above), with the exception of the subsequent non-exercise data for blood pressure and Hct influenced by the seated posture, were assessed using a single two-factor, repeated-measures analysis of variance (ANOVA) with trial (leg press and cycle ergometry) and time of measurement as the repeated measures. The subsequent non-exercise data were assessed using a single-factor, repeated-measures ANOVA with time (as above) as the repeated measure. The Tukey Honestly Significant Differences method was used to assess the location of any significant differences. A probability level of $P \leq 0.05$ was considered to be statistically significant. Unless otherwise stated, all values are expressed as the mean (SD).

Results

Matching the duration of the two exercise modes to total cardiac cycles resulted in a significantly different total exercise time [$F(1,12) = 20.69$, $P = 0.0007$] for cycling [(min:s) 13:47 (1:10)] compared to the resistance exercise (constant at 15:15).

Because of faulty preservative reagents, ANP analysis was confounded for seven subjects and it was therefore necessary to exclude these data. As shown in Fig. 1, in the remaining six subjects, no statistically significant differences in plasma [αANP] were found between the resistance and submaximal cycling trials [$F(1,5) = 1.11$, $P = 0.34$]. In addition plasma [αANP] failed to increase significantly with exercise [$F(11,55) = 1.05$, $P = 0.42$]. However, it should be noted that one subject (who exercised at the highest power output) did exhibit a more than five-fold rise in [αANP] during submaximal dynamic exercise.

Analysis of SBP (Fig. 2) produced a significant main effect [$F(11,132) = 101.74$, $P < 0.00001$] for time, indicating an increase in pressure at all time points during exercise as well as a reduction from baseline at the 10P, 15P, 30P, 45P and 60P time points. Collapsed across modality, maximal SBP was ≈225(31) mmHg and occurred during the initial 5 min of exercise. Conversely, the greatest decrement in SBP was evident at the 30P time point and was ≈20 mmHg below resting values. In addition, the SBP recorded 5 min into the submaximal dynamic exercise was significantly [$F(11,132) = 2.71$,

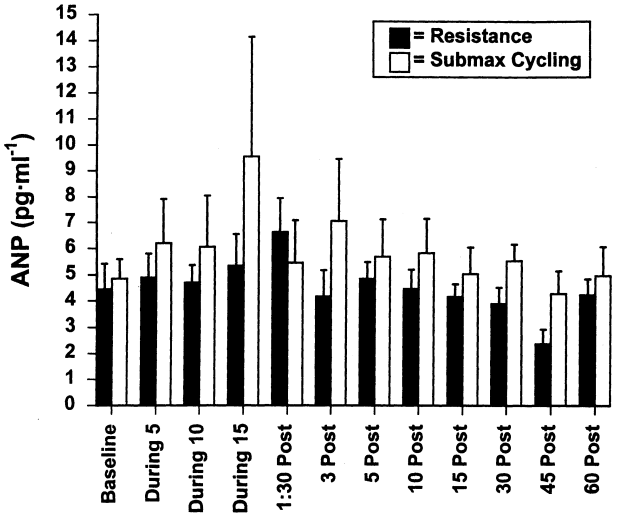

Fig. 1 The response of atrial natriuretic peptide *ANP*: [mean(SEM)] to resistance (*black bars*) and submaximal dynamic (*clear bars*) exercise ($n = 6$). The time scale on the abscissa is given as min (or min:s) during or post-exercise (*Post*)

$P < 0.00001$] elevated from the corresponding time point during the resistance trial.

Analysis of DBP (Fig. 2) indicated a significant main effect for time [$F(11,132) = 22.35$, $P < 0.00001$], but unlike SBP it did not decrease below baseline during recovery. Across trials, increases in DBP were found only during the exercise period, with maximal values reaching 93(15) mmHg at the D5 reading. The difference between exercise modalities failed to reach significance. Post hoc analysis of the significant [$F(11,132) = 10.02$, $P < 0.00001$] interaction did indicate that the average DBP that were incurred with resistance exercise were elevated above both the resting values and those reached during the cycle ergometry. Submaximal dynamic exercise failed to increase DBP.

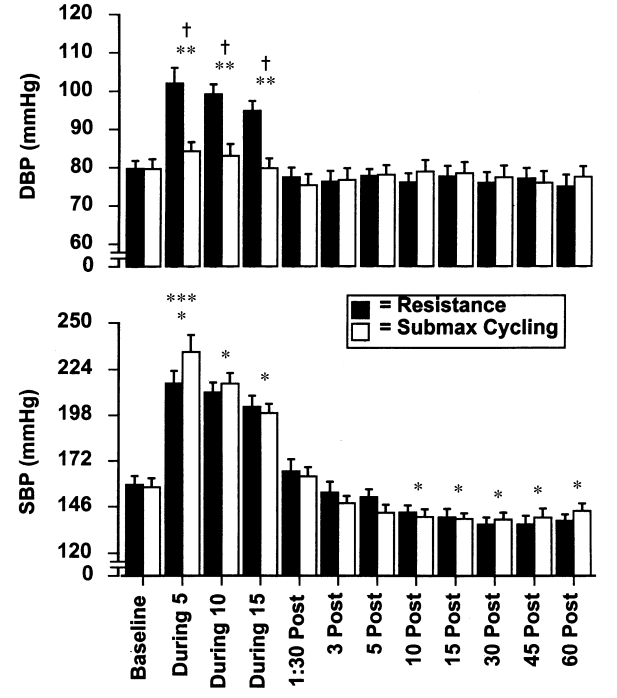

Fig. 2 The response of blood pressure [mean(SEM)] to resistance (*black bars*) and submaximal dynamic (*clear bars*) exercise *SBP* systolic blood pressure, *DBP* diastolic blood pressure pooled systolic data is significantly different from baseline; **systolic pressure of the resistance trial is significantly different from that of the submaximal cycling trial; ***pooled diastolic data is significantly different from baseline; †diastolic pressure of the resistance trial is significantly different from that of the submaximal cycling trial

MAP increased [F(11, 132) = 79.44, $P < 0.00001$] from 103(8) mmHg to a peak value of 127(13) mmHg during exercise (D5). The increases noted at the D10 and D15 time points were also significant. MAP was reduced at 30, 45 and 60 min post-exercise, with a maximal decrement of approximately 7 mmHg (at 45P) below baseline. Significant interactions [$F(11,132) = 8.72$), $P < 0.00001$] confirmed that resistance exercise elicited greater MAP increases than cycling exercise. During the 15 min of exercise, MAP averaged 129(12) mmHg during the resistance trial and 116(9) during the submaximal cycling trial.

Analysis of exercise oxygen consumption during exercise revealed both a main effect for trial [$F(1,12) = 73.50$, $P < 0.00001$] and time [$F(3,36) = 268.12$, $P < 0.00001$]. The trial effect was indicative of greater oxygen consumption during the submaximal cycling (which was ≈65% of $\dot{V}O_{2\,max}$). Submaximal cycling elicited an oxygen consumption of 35.2(6.8) versus 19.8(4.5) $ml \cdot kg^{-1} \cdot min^{-1}$ for the resistance exercise. Post hoc analysis of time (irrespective of trial) revealed a significant increase from baseline to D5. This increase was further heightened at the D15 time point.

As shown in Fig. 3, changes in HR were consistent between conditions, both during and following exercise, with the exception of the D5 point where it was significantly [$F(11,132) = 4.91$, $P < 0.00001$] higher during the submaximal cycling trial. Resting HR was 69(12) beats per minute (bpm). This value increased significantly [$F(11,132) = 271.69$, $P < 0.00001$] with exercise at the D5 time point [131(15) bpm] and increased further to 152(16) bpm immediately prior to the cessation of exercise. In recovery, this value immediately dropped below exercise values, yet remained significantly elevated above baseline values for ≈15 min.

Analysis of RPP indicated that there was no main effect for trial. The RPP was significantly elevated from baseline at all points during exercise [$F(11,132) = 220.20$, $P < 0.00001$] and returned to resting values by approximately 3 min post-exercise (collapsed across trial). Results of the interaction [$F(11,132) = 7.20$, $P < 0.00001$] indicated that the RPP of the initial two time points during exercise (i.e. D5 and D10) were significantly greater during the resistance exercise session.

A main effect for trial indicated a significantly increased [$F(11,121) = 24.65$, $P < 0.00004$] Hct during the resistance exercise (Fig. 4). There was an additional main effect for time [$F(11,121) = 23.72$, $P < 0.00001$],

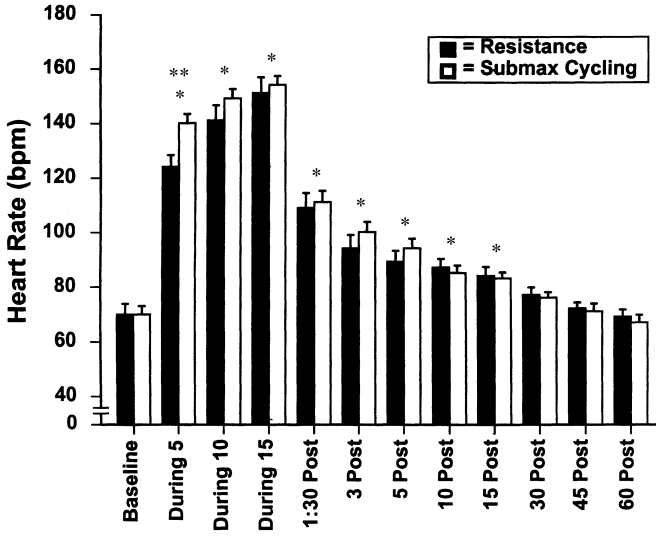

Fig. 3 The response of heart rate [mean(SEM)] to resistance (*black bars*) and submaximal dynamic (*clear bars*) exercise. *pooled data is significantly different from baseline, **data of resistance trial is significantly different from that of the submaximal cycling trial

indicating that Hct was significantly elevated from the onset of exercise until the 5P time point. There was a continued decrement after 5P, but it failed to reach significance. The significant interaction [$F(11,121) = 2.40$, $P = 0.01$] is indicative of Hct being higher in the resistance trial than in the submaximal cycling trial at D10, D15, 1:30P, 3P and 5P.

There were no observed changes for any blood pressure or Hct measures in the two individuals who were tested during the non-exercise condition.

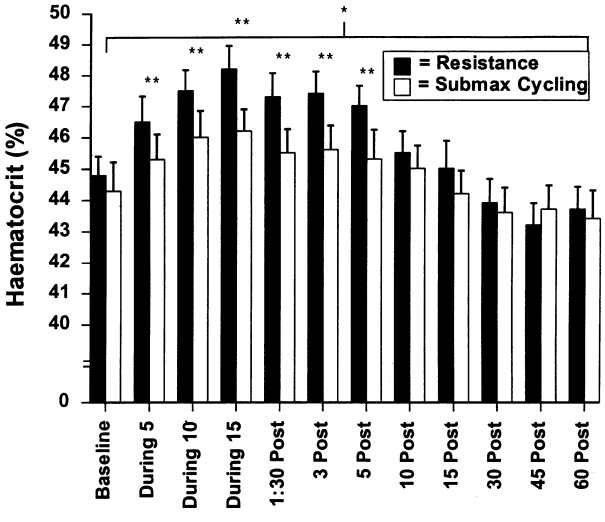

Fig. 4 The response of haematocrit [mean(SEM)] to resistance (*black bars*) and submaximal dynamic (*clear bars*) exercise, (*main effect for trial, **pooled data is significantly different from baseline

Discussion

The data indicate that when healthy young normotensive subjects perform ≈15 min of mild-intensity (65% $\dot{V}O_{2\,max}$) cycle ergometry or continuous weightlifting (65% 1RM) exercise, no changes occur in circulating [ANP]. Ten minutes following exercise, SBP decreased significantly below baseline, and 30 min following exercise MAP was also significantly lower than baseline. This post-exercise "hypotensive effect" was similar for both exercise modalities until measurements were terminated at 60 min. Follow-up studies using the identical methodology in a sub-sample of subjects who simply remained in the same seated position (without performing exercise), revealed no changes in resting blood pressure (Table 1), thus indicating that the observed hypotensive response was due to the exercise intervention.

Considerable intra- and intersubject variability was evident in the αANP response to exercise. The 89% increase in [αANP] at the 15-min point of the cycle ergometry exercise was largely due to the exaggerated response of one subject and was not statistically significant. Similarly, the 49% elevation in [αANP] at (min:s) 1:30 following the resistance exercise, the result taken from six subjects, was not statistically significant. Lentini et al. (1993) previously found pronounced ventricular dimension changes with resistance and submaximal dynamic exercise. A sub-sample of subjects in the current study underwent echocardiography to confirm these volume changes in the right atrium. The volume of the right atrium was found to be statistically significantly augmented during the submaximal cycling trial and statistically significantly reduced during the resistance trial. The failure to detect an increase in αANP release with either form of exercise was unexpected, in light of these changes in cardiac volumes and the heightened blood pressures associated with the resistance exercise. It is possible that the duration of exercise was insufficient to elicit αANP release during the submaximal cycling trial. In addition, blood pressure did not increase as much in this study as in our previous study where a significant release of αANP was observed with resistance exercise (MacDonald et al. 1995).

The significant increase in Hct which was detected during the resistance exercise, and which persisted until 5 min following exercise, has been well documented (Goodman et al. 1993; Ploutz-Snyder et al. 1995). Loss of plasma to the extravascular space in muscle is thought to be caused by the increased hydrostatic capillary pressures that accompany the dramatic increases in blood pressure which occur with each lift (MacDougall et al. 1985; Ploutz-Snyder et al. 1995). Haemoconcentration is also known to occur with endurance exercise, but the magnitude is considerably less (Novosadova 1977). The finding that Hct returned to normal after 5 min of recovery indicates that shifts in plasma volume were only transient and thus could not have been the cause for the PEH which occurred.

Table 1 Data from subsequent non-exercise trials. The time points are labelled to correspond to the exercise trials, although no exercise occurred. (*During* During exercise, *Post* post-exercise)

Subject	Baseline	During 5	During 10	During 15	1:30 Post	3 Post	5 Post	10 Post	15 Post	30 Post	45 Post	60 Post
						Haematocrit (%)						
1	47	48	44	46	45	46	46	46	46	48	44	46
4	41	42	42	41	41	42	41	42	41	42	41	42
						Mean blood pressure (mmHg)						
1	91	94	92	92	96	96	94	93	92	88	88	88
4	88	95	92	90	92	89	91	90	85	91	96	93

PEH has been documented to occur to a greater extent in hypertensive subjects than in normotensive subjects. In addition, many investigations of PEH in normotensive subjects have yielded contradictory results (Floras and Senn 1991; Pescatello et al. 1991; Floras and Wesche 1992; Hara and Floras 1992). In previous studies, however, blood pressure was measured by indirect methods that may have contributed to these discrepancies. In addition, no standardised body posture has been accepted for the post-exercise measurement period. In the present study, decrements in average SBP that were observed following exercise exceed those normally reported for a normotensive population. This may be partially because in the present study intra-arterial pressure was monitored directly. This method of sampling is more apt to detect small differences in pressure and is less affected by motion and limb position than the auscultatory methods used in the majority of studies (Kiyonaga et al. 1985; Landry et al. 1992). Unlike a number of previous reports, the average DBP observed following exercise in the present study did not parallel the change in systolic pressure (e.g. Wilcox et al. 1982; Bennett et al. 1984; Kaufman et al. 1987; Hara and Floras 1992). A possible explanation for this may be that some studies which report a decrease in DBP following exercise involved measurement of subjects in the supine position (Piepoli et al. 1993), whereas the present study maintained subjects in the sitting position. Bennett et al. (1984) suggested that the decreases in DBP which are detected post-exercise differ by approximately 6 mmHg between the sitting and standing position, with the greatest reductions occurring in the sitting position. It is possible that similar differences exist between the sitting and supine position, and thus, changes in DBP might not be detected in seated, normotensive subjects.

With the exception of maximal exercise tests (which generally last ≈8–12 min), the majority of endurance exercise protocols which have resulted in PEH have ranged from 20 to 60 min in duration (Bennett et al. 1984; Floras and Senn 1991; Floras and Wesche 1992; Hara and Floras 1992). The exercise intensities used in those studies were ≈75% of maximal HR. Assuming that subjects in the present study exercised at 65% $\dot{V}O_{2\,max}$ for approximately 13.5 min, this study has documented the occurrence of PEH after a mild bout of exercise of shorter duration than has been previously reported. This may have significant clinical implications, since this is a readily obtainable target intensity and duration, even for the hypertensive and elderly population.

The present study has also documented the occurrence of PEH after a bout of resistance exercise. Previous work in this area is sparse and contradictory. Brown et al. (1994) observed decreases in blood pressure after a bout of resistance exercise, whereas O'Connor et al. (1993) reported elevations in blood pressure following resistance exercise. The present study confirms that PEH occurs in response to resistance exercise. Moreover, the significantly decreased SBP and MAP values recorded at the termination of measurement (after 1 h) in the rest phase concur with reports of prolonged hypotension (Pescatello et al. 1991; J. Hagberg, personal communication).

In the present study, αANP appears not be a significant modulator of PEH since both the resistance and submaximal dynamic exercise modalities had a minimal effect on circulating levels of αANP. Moreover, any changes in [ANP] had disappeared following 5 min of recovery, and significant declines in MAP did not occur until 30 min following the cessation of exercise. This is in agreement with previous research (Perrault et al. 1991; Bussieres-Chafe et al. 1994; Perrault et al. 1994) which has found a return to baseline [ANP] by 30 min following upright exercise. However, the possibility of a delayed effect of ANP on the cardiovascular control centres modulated by the earlier ANP release cannot be discounted, nor can the potential contribution of ANP to PEH during longer, more intense bouts of exercise.

Although the catheter provided a saline drip, the volume infused over the testing session was minimal [62.3(30.2) ml], and is similar to that of the blood withdrawn for analysis (≈72 ml). This would therefore argue against a hypotensive effect due to decreased total blood volume.

Finally, the fact that Hct did not change during the 75 min non-exercise control trial (Table 1), nor during the recovery portion of the exercise trials in which PEH occurred, argues against vascular pooling as a causal mechanism. One might postulate that maintaining a relatively static body position for 60 min caused a shift of plasma from the vascular space and thus a decreased the total blood volume resulting in a subsequent drop in blood pressure. It is apparent that this possibility can also be dismissed as a mechanism.

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