The University of San Francisco

USF Scholarship: a digital repository @ Gleeson Library | Geschke Center

Kinesiology (Formerly Exercise and Sport Science)   College of Arts and Sciences

2004

Intra-Arterial Blood Pressure Characteristics during Submaximal Cycling and Recovery

Julia Orri
University of San Francisco, jorri@usfca.edu

S E. Griffin

Robert A. Robergs

David S. James

D R. Wagner

See next page for additional authors

Recommended Citation

Authors

Julia Orri, S E. Griffin, Robert A. Robergs, David S. James, D R. Wagner, and R Quintana

Systems Physiology: Cardiopulmonary

INTRA-ARTERIAL BLOOD PRESSURE CHARACTERISTICS DURING SUBMAXIMAL CYCLING AND RECOVERY

ORRI JC[1], GRIFFIN SE[1], ROBERGS RA[1], JAMES DS[1,2], WAGNER DR[1], QUINTANA R[1].

[1]Exercise Physiology Laboratories, University of New Mexico, Albuquerque, NM, USA 87131

[2]Veterans Administration Medical Center and University of New Mexico School of Medicine, Department of Internal Medicine, Albuquerque, NM, USA 87108

ABSTRACT

INTRA-ARTERIAL BLOOD PRESSURE CHARACTERISTICS DURING SUBMAXIMAL CYCLING AND RECOVERY. **Orri JC , Griffin SE, Robergs RA, James DS, Wagner DR, Quintana R**. **JEP**online. 2004;7(2):45-54. The purpose of this study was to measure intra-arterial (IA) blood pressure from rest to steady-state submaximal exercise and immediately post-exercise. Beat-to-beat blood pressure was compared to breath-by-breath VO_2 during steady-state and maximal exercise. Fourteen normotensive subjects volunteered. Systolic (SBP), diastolic (DBP) and mean (mBP) blood pressure was measured from rest to steady state during cycling at 45, 60, and 75% maximal power output (POmax). BP was assessed during recovery from VO_2peak through 2 min of cycling at 50 W. During the rest to exercise transition, mBP decreased from 103.41 ± 9.4 to 90.1 ± 8.9 mmHg after 11.6 ± 6.2 s (p<0.0001). SBP, DBP, and mBP at VO_2peak were 198.2 ± 22.6, 100.5 ± 10.8, and 128.7 ± 14.2 mmHg, respectively. Immediately following exercise, SBP, DBP, and mBP were 156.7 ± 17.2, 62.2 ± 10.4, and 97.4 ± 15.7 mmHg, respectively, which were significantly lower from peak values (p<0.001). The time to the lowest hypotensive response for SBP, DBP, and mBP was 9.2 ± 8.7, 10.2 ± 9.1, and 4.7 ± 1.8 seconds, respectively. Nonlinear relationships between blood pressure and VO_2 were found at all workloads. In summary, the rapidity of a post-exercise hypotension can modify the interpretation of maximal BP immediately post-exercise.

Key Words: radial artery, hypotension, exercise

INTRODUCTION

Although the direct measurement of ascending aortic blood pressures is the "gold standard" or criterion method for measuring blood pressure (BP) at rest and during exercise (1), this procedure involves the insertion and advancement of a catheter from an artery, such as the brachial, into the aorta. Beat-by-beat changes can then be monitored during the transitions from rest to exercise and from exercise to recovery. Manual sphygmomanometry can be used to estimate ascending aortic systolic blood pressure (SBP), but it has been shown to underestimate diastolic blood pressure (DBP) during exercise (2). Peripheral blood

pressure can be measured through intra-arterial (IA) methods using the brachial, radial, or femoral arteries. However, waveform reflection due to the distance of the arterial wall from the central circulation causes amplification of the pulse waveform, resulting in increased SBP, especially with increasing exercise intensities (3).

Little research has been done that specifically examines changes in blood pressure immediately following exercise. Decreases in SBP of 30 mmHg were found within 15 sec after arm crank exercise (4). During clinical exercise testing, immediate post-exercise (IPE) blood pressures are often used to represent maximal systolic and diastolic pressures. Traditionally, the technician measures the "maximal" pressure immediately after the subject completed the test. However, since SBP and DBP can decrease significantly 1 to 10 sec post-exercise (5), this routine clinical practice needs to be re-evaluated.

While the blood pressure response to exercise is documented, there are no previous data examining the relationship between blood pressure and VO_2 during exercise. It has been understood that both parameters increase to maximal values with incremental exercise, but their association has not been established.

The purpose of this study was to evaluate the intra-arterial blood pressure characteristics during the rest to exercise transition, as well as steady-state exercise, and immediately post-exercise. Based on pilot work, we hypothesized that following intense exercise there will be an immediate decrement in SBP, DBP, and mean blood pressure (mBP). In addition, we compared beat-to-beat blood pressure to breath-by-breath VO_2 during steady-state and maximal exercise in order to determine the relationship between these variables.

METHODS
Subjects
Fourteen subjects (10 male and 4 female) volunteered for this study. Their mean age was 28.3 ± 5.8 yr. All subjects were normotensive and engaged in aerobic exercise for a minimum of 60 min/week. A standing mercury column sphygmomanometer (KTK, Tokyo, Japan) was used to screen subjects for possible hypertension. All guidelines, as recommended by the American Heart Association for indirect blood pressure measurement, were followed with respect to pre-test instruction and proper cuff size (6). Exclusion criteria included hypertension, cardiovascular disease, and blood pressure differences between left and right arms in excess of 5 mmHg for either SBP or DBP. Informed written consent was obtained from all subjects in accordance with the policy statement of the American College of Sports Medicine prior to testing. The research protocol and informed consent were approved by the Institutional Review Board of the University's School of Medicine and the Veterans Affairs Medical Center.

Procedures
Prior to collection of exercise blood pressures, a maximal power output at VO_2max (POmax) test was administered. For this test, the subjects exercised on a constant-load, electronically-braked, cycle ergometer (Jaeger Ergotest, Hambrecht, Germany). The protocol used incremental workloads of 20, 25, or 30 W/min until the subject reached volitional fatigue. Workload increments were individualized according to the subject's fitness level to ensure a test duration of 10 to 14 minutes. The POmax was used to determine submaximal workloads for subsequent exercise trials involving blood pressure measurements.

In preparation for radial artery cannulation, the skin was cleaned with alcohol and a local anesthetic was administered. A 4.45cm, 18-gauge fluid catheter (Arrow RA-04018, Reading, PA) was placed percutaneously in the radial artery approximately 3 cm proximal to the wrist. The catheter was then connected to a transducer (INTRAFLO®) and drip chamber/pressure infusor setup via low compliance tubing. The system was zeroed and calibrated prior to each trial. The dynamic response of the system was tested using the square wave test to ensure that no air bubbles or clots were present in the tubing.

Data Collection
Due to the invasive nature of IA blood pressure measurements, all data were collected in one, 3-hr session involving a single catheterization. Breath-by-breath indirect calorimetry data were obtained by a Medical Graphics metabolic analyzer (CPX-MAX, St. Paul, MN). Two cycling tests were administered. There was a 20 min rest period between the tests. The first test consisted of 3 min bouts at 45, 60, and 75% POmax. For the second test, a subset of eight subjects cycled for 3 min stages at 45, 60, and 75% POmax. In the ninth

minute, the load was increased 20 Watts/min until the subjects reached VO_2peak. One subject had incomplete VO_2 data during the VO_2peak test. Therefore, her data were excluded from the analysis, bringing the subset size to seven.

Intra-arterial blood pressures were measured continuously from 1 min prior to the start of exercise to the end of each test. Heart rates were monitored beat-to-beat by electrocardiography from a V-5 lead monitoring configuration (Biopac™ Systems Inc., Santa Barbara, CA). Instruments, including an electrocardiograph, were integrated to a data acquisition system (Biopac™ Systems Inc., Santa Barbara, CA) and computer. The analogue signal of the electrocardiogram and blood pressure waveforms was acquired at 200 Hz using commercial software (Acknowledge™, Biopac™ Systems Inc., Santa Barbara, CA). Nonlinear regression was used to obtain BP and VO_2 kinetic response data (half time=$t_{0.5}$), steady-state BP, and peak and low SBP, DBP, and mBP (GraphPad Inplot v. 4.0, San Diego, CA). The $t_{0.5}$ represents the time to reach half of the steady-state or VO_2peak response value.
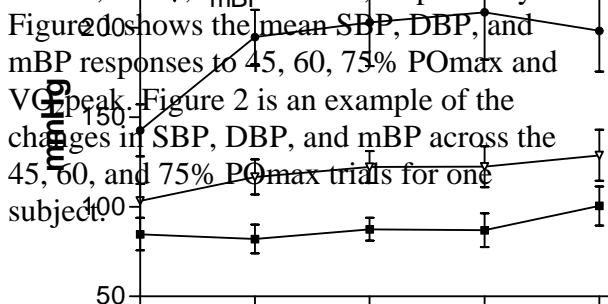
Statistical Analyses

Dependent t-tests were used to compare peak-to-low means in SBP, DBP, and mBP. Peak and low SBP, DBP, and mBP were the maximal and minimal blood pressures during and immediately following the VO_2peak trial. One-way ANOVAs were used to compare differences in the means of SBP, DBP, and mBP for the following variables: start of exercise $t_{0.5}$, recovery $t_{0.5}$, and time to post-exercise hypotensive response (SPSS v. 8.0, Chicago, IL). The alpha was adjusted to 0.017 due to the three ANOVAs that were performed (0.05/3=0.017).

Nonlinear regression was used to obtain the best-fit equation for VO_2 versus SBP, DBP, and mBP (GraphPad Prism, v. 2.0, San Diego, CA). Dependent t-tests were used to compare means between the linear and curvilinear equations for absolute sum of squares and standard deviation of the residuals ($S_{y.x}$). Statistical power was calculated using GraphPad Statmate (v. 3.0, San Diego, CA). Power for the initial and post-exercise hypotension was 0.8 for a mean difference of 10.48 and 19.59 mmHg, respectively. Effect sizes were 1.8 and 3.53 for rest-exercise and VO_2peak-recovery transitions, respectively. For blood pressure and VO_2 kinetics, power was 0.8 for a mean $t_{0.5}$ difference of 57.93 s. The effect size for the comparison of $t_{0.5}$ values between VO_2 and SBP was 2.89. Significance was set at $p< 0.05$. All measures are presented as mean ± SD. Blood pressure data are presented for the 45, 60, and 75% POmax trial, as well as VO_2peak and 2 min of active recovery following VO_2peak.

RESULTS
Rest to 45% POmax

Table 1 shows the demographic data for the 14 subjects. Blood pressure data for the rest to exercise transition are presented in Table 2. Steady state SBP, DBP, and mBP were 194.69 ± 15.13, 82.05 ± 8.02, and 116.56 ± 9.77 mmHg, respectively. The time to reach steady state for SBP, DBP, and mBP was 104.32, 99.48, and 102.66 s, respectively. Figure 2 shows the mean SBP, DBP, and mBP responses to 45, 60, 75% POmax and VO_2peak. Figure 2 is an example of the changes in SBP, DBP, and mBP across the 45, 60, and 75% POmax trials for one subject.

Table 1. Subject demographics (N=14)

VARIABLE	MEAN ± SD	RANGE
Age (yr)	28.3 ± 5.8	21 - 42
Body Mass (kg)	72.3 ± 12.4	52.7- 89.5
POmax (W)	294.5 ± 63.2	200 - 450
Resting BP*		
systolic	111.0 ± 12.6	90 - 126
diastolic	78.6 ± 8.1	64 - 92
Mean	89.4 ± 9.0	73 - 103
ng IA BP		
systolic	142.7 ± 14.5	122.8 - 166.4
diastolic	84.7 ± 9.0	71.7 - 97.5
mean	103.4 ± 9.4	90.5 - 114.3

*...l sphygmomanometry (mmHg).

Figure 1. Mean SBP, DBP, and mBP at rest, 45, 60, 75% POmax and VO₂peak.

The one-way ANOVAs resulted in no significant differences in the $t_{0.5}$ between SBP, DBP, and mBP (p=0.153). There was a significant difference between the $t_{0.5}$ values for VO_2 and SBP (p=0.008) (Table 3).

Dependent t-tests showed significant differences between the absolute sums of squares (SS) for the linear and curvilinear equations, respectively, for SBP (8562.5 ± 10279.69 to 6677.7 ± 9997.17) (p=0.015), and DBP (1582.57 ± 1170.4 to 1327.31 ± 1128.67) (p=0.006). For SBP and DBP, the nonlinear equation gave the least sums of squares. Table 4 shows the goodness of fit comparisons between the linear and curvilinear equations for VO_2 and blood pressure for each subject. The smaller sums of squares and larger R^2 for the nonlinear equations quantified goodness of fit. Figure 3 represents the nonlinear relationship between blood pressure and VO_2 for one subject.

Table 3. $t_{0.5}$ values for VO_2 and SBP, DBP, mBP

	REST-45%	*45-60%*	*60-75%*
VO_2 $t_{0.5}$	18.50 ± 10.36	142.80 ± 74.53	253.17 ± 70.1
SBP $t_{0.5}$	49.03 ± 30.68*	61.55 ± 27.92**	50.44 ± 16.83†
DBP $t_{0.5}$	4455.61 ± 10291.94	89.72 ± 51.67	53.14 ± 71.69†
mBP $t_{0.5}$	957.03 ± 2872.81	69.63 ± 26.78	38.30 ± 51.04†

† p <0.001 vs VO_2 $t_{0.5}$ (60-75%), * p =0.008 vs. VO_2 $t_{0.5}$ (Rest-45%),
** p =0.013 vs. VO_2 $t_{0.5}$ (45-60%)

Table 4. Goodness of fit of linear and curvilinear models for describing relationship between BP and VO_2 (45% POmax trial, N=14)

R^2 I^a SBP	R^2 II^b SBP	R^2 I^a DBP	R^2 II^b DBP	R^2 I^a mBP	R^2 II^b mBP
0.49 ± 0.27	0.61 ± 0.26	0.15 ± 0.14	0.30 ± 0.19	0.31 ± 0.23	0.45 ± 0.24

R^2 I^a= goodness of fit for linear equation; R^2 II^b= goodness of fit for nonlinear equation

45-60% POmax

Due to system errors, two subjects were unable to complete the 45-60% trial, resulting in the analysis of data on 12 subjects. The steady state SBP, DBP, and mBP were 202.88 ± 24.35, 87.47 ± 6.37, and 122.19 ± 8.9 mm Hg, respectively. The one-way ANOVA's showed no significant differences in the $t_{0.5}$ between SBP, DBP, and mBP (p=0.184). Dependent t-tests again showed significant differences between the absolute SS for the linear and curvilinear equations, respectively, for SBP (5525.25 ± 3118.37 to 5178.68 ±3029.98) (p=0.003), and mBP (5521.31 ± 4187.86 to 5249.88 ±3978.48) (p=0.005). For both SBP and mBP, the nonlinear equation gave the least sums of squares. There were no significant differences between the standard deviation of the residuals ($S_{y.x}$) for the linear and curvilinear equations (p>0.0167). There was a significant difference (p=0.013) between the $t_{0.5}$ values between SBP and VO_2. The large $t_{0.5}$ values for DBP and mBP were due to extremely slow rates of change in DBP and mBP for some subjects (Table 3).
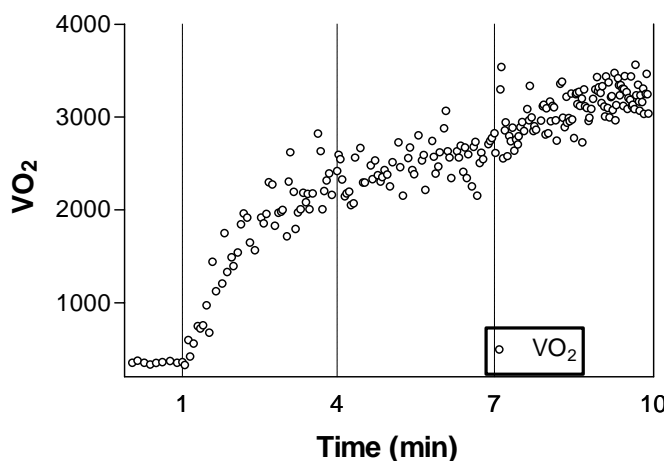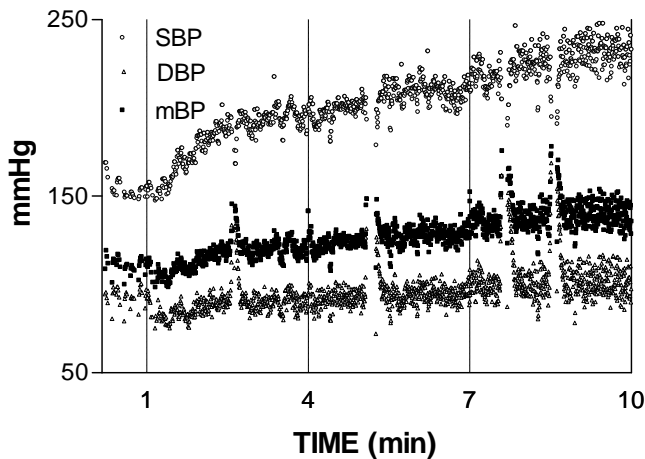
Figure 2. VO$_2$, SBP, DBP, and mBP responses at rest, 45, 60, and 75% POmax for one subject. Note: min 0 = rest, min 1= 45% POmax, min 4= 60% POmax, min 7= 75% Pomax. VO$_2$ data are ml/min.

60-75% POmax

The steady state SBP, DBP, and mBP were 208.54 ± 26.42, 86.98 ± 9.45, 122.38 ± 11.43 mm Hg, respectively. There were no significant differences between the three pressures for $t_{0.5}$ (p=0.736). Significant differences were found between the absolute sums of squares for the linear and curvilinear equations, respectively, for SBP (6010.17 ± 2945.94 to 5724.58 ±3003.47) (p=0.005), and DBP (9197.17 ± 10272.6 to 8956.5 ± 10096.58) (p=0.014). In addition, there were significant differences between the VO$_2$ $t_{0.5}$ and SBP, DBP, and mBP $t_{0.5}$ values (p<0001 for all) (Table 3).

75% POmax through VO$_2$peak and active recovery

Table 5 shows the mean decrement in blood pressure immediately following maximal exercise. Dependent t-tests showed significant differences in peak-to- low SBP, DBP, and mBP preceding active recovery (p<0001). Figure 4 is an example of the post-exercise hypotensive response for one subject. The one-way ANOVA showed no significant differences between SBP, DBP, and mBP for the amount of time required to reach the lowest pressure prior to active recovery (p=0.6). There were also no significant differences for the $t_{.05}$ between SBP, DBP, and mBP from VO$_2$peak to recovery (p=0.403).

Table 5. Decreases in IA blood pressure preceding active recovery (n=7)

	PEAK (mmHg)	LOW (mmHg)	CHANGE (mm Hg)	TIME TO LOW (s)
SBP	198.17 ± 22.62	156.65 ± 17.20 *	41.52 ± 21.29	9.16 ± 8.71
mBP	128.73 ± 14.21	97.38 ± 15.72 *	31.36 ± 10.78	4.65 ± 1.81
DBP	100.48 ± 10.84	62.2 ± 10.39 *	38.28 ± 9.72	10.2 ± 9.15

*= significantly different from peak, p<0.001

Significant differences were found between the absolute sums of squares for the linear and curvilinear equations, respectively, for SBP (2748.4 ± 1984.34 to 2635.96 ± 1983.18), DBP (2894.29 ± 1025.08 to 2632.14 ±1051.12), and mBP (1775.23 ± 979.91 to 1644.59 ± 948.79) versus VO$_2$ (p<0.0167). Again, the nonlinear equation provided the best fit to the data due to the least sums of squares. Dependent t-tests showed significant differences between the VO$_2$ $t_{.05}$ and the SBP $t_{0.5}$ (p=0.008). Figure 5 illustrates the nonlinear relationship between blood pressure and VO$_2$ for one subject. Finally, there were no significant

differences between the standard deviation of the residuals ($S_{y.x}$) for the linear and nonlinear equations ($p > 0.0167$).

DISCUSSION

Rest to exercise transition

We found significant differences between the time constants ($t_{0.5}$) of VO_2 and BP during the rest to exercise transition, steady state, and VO_2peak, suggesting their independent rates of change. During the rest to exercise transition, VO_2 changed more rapidly than BP, and the opposite response occurred at exercise intensities greater than 45% POmax. Since blood pressure and flow can be influenced by many factors, a linear relationship may not always be expected with VO_2. According to Poiseuille's law, changes in pressure are directly proportional to blood viscosity, but inversely related to radius raised to the fourth power. Therefore, any changes in flow or diameter would influence pressure and the relationship to VO_2.

Sheriff and colleagues reported that arterial pressure reached a nadir within 2-5 s after exercise onset (7). They concluded that arterial pressure fell due to activation of the muscle pump, which propelled a volume of blood from the arterial system. Subsequently, the rhythmic contractions of exercise increase venous return and further perfusion of skeletal muscle. Increased flow can plateau after only 5-7 s of exercise, followed by a slower velocity at 15-20 s (8). Blood flow is also dependent on arm position. Tschakovsky (9) reported a 60% increase in blood flow after 2 s of rhythmic exercise with the arm below heart level. In the present study, the hand position on the cycle ergometer was below heart level of the subjects. Even though they were not performing arm exercise, it is possible that the strength of grip on the handlebars changed blood flow.

Factors contributing to vasodilation such as increased temperature (10, 11, 12), carbon dioxide, acidity, magnesium, potassium (K^+) (13), acetylcholine and nitric oxide (NO) (14) can disproportionately lower blood pressure compared to VO_2. However, some researchers have stated that the K^+ and adenosine relationship to vasodilation is unclear (8). Adenosine may act jointly with other vascular determinants, or increases in flow may be non-metabolic (15). Shoemaker (8) suggested that trained individuals produce lower muscle metabolites at exercise onset, and subsequent vascular influences result from sympatho-inhibition rather than vasodilation.
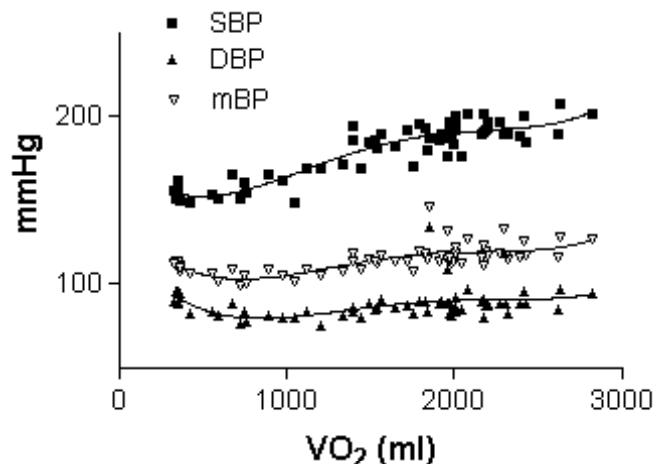

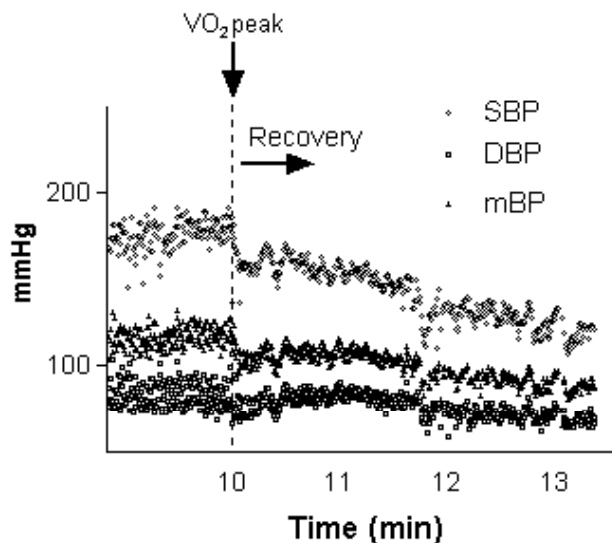**Figure 3. BP-VO_2 relationship at rest- 45% POmax for one subject. VO_2 data are ml/min.**

Figure 4. Immediate post-exercise hypotensive response for one subject. Note: 10 min = VO_2peak, followed by an active recovery through min 13.
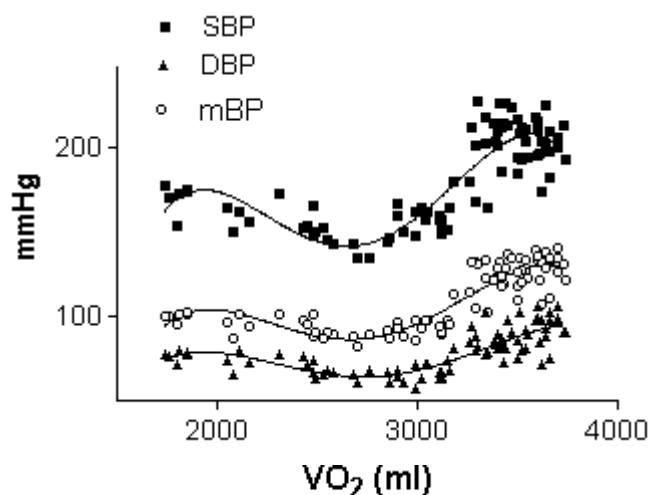

Figure 5. BP-VO_2 relationship at 75% POmax-VO_2peak POmax for one subject. VO_2 data are ml/min.

VO_2 kinetics are dependent, in part, on fitness level. Ebfeld et al. (16) reported an association between VO_2max and VO_2 dynamics through spectral analysis. Subjects with higher relative VO_2max values had augmented amplitude ratios, compared to untrained. In the present study, our subjects' mean maximal workload was nearly 295 Watts, representing a moderate to highly-trained fitness level. It is possible that specific VO_2 kinetics, reflecting the training status of our subjects', contributed to the nonlinear relationship between VO_2 and blood pressure.

VO_2 kinetics can also be influenced by previous exercise. Yoshida et al. (17) showed accelerated changes in the VO_2 time constant when one-legged cycling was repeated following a 5 min rest. Slower VO_2 kinetics were observed when the other leg was used. In the present study, since no warm-up period was given, it can be speculated that the VO_2 kinetics may have been slowed in relation to blood pressure changes. Additional research is needed to fully explain the transient development of a hypotensive response during the initial seconds after commencement of lower body dynamic exercise.

Steady-State

Steady state was reached by the end of minute 2 for SBP, DBP, and mBP. This was not surprising, given the low intensity of the exercise (45% POmax), and the average maximal workload (294 W) of the subjects. Furthermore, the rapid attainment of steady state could be explained by the use of upright, rather than supine cycling (18).

The increases in SBP and mBP, and the slight decrease in DBP during exercise were expected. As cardiac output increases diastolic blood pressure is maintained through reduced peripheral vascular resistance. Following the initial 5-10 s of exercise, the increased blood flow is modulated through a combination of neural, metabolic, and endothelial factors, with a competitive association between sympathetic and dilatory factors (15). As exercise continues toward steady state, metabolites play an enhanced role in regulating vascular tone compared to exercise onset (8).

VO_2peak to recovery transition

This study confirmed the hypothesis of an immediate decrement in SBP, DBP, and mBP following maximal exercise. Hollingsworth et al. (4) also reported an immediate and significant fall in SBP after arm-crank exercise. Our data suggest the influence of an immediate intramuscular peripheral vasodilation preceding active recovery. Post-exercise peripheral vasodilation has been shown to persist for 1 hr following maximal cycling in untrained subjects (10). In addition, a reduction in total peripheral resistance can be expected post-exercise (11).

Piepoli et al. (19) noted that during recovery from maximal exercise on a cycle ergometer, there is a significant decrease in systematic vascular resistance compared to control values. The sharpest decrease (36.5%) was at 5 min into recovery. Results by Sprangers et al. (20) showed a 41% fall in total peripheral resistance immediately after a 3-s bout of cycling. Control values were reached 20 s after the start of exercise. A decreased limb vascular resistance has been suggested by others to be the mechanism responsible for the immediate post-exercise hypotension following moderate (21) and maximal cycling exercise (10). Since our subjects had to briefly stop pedaling to allow for workload adjustment following VO_2peak, it is also possible that muscle pooling and decreased venous return resulted in the post-exercise hypotension.

Another mechanism responsible for vasodilation during exercise recovery is increased core temperature. Kaufman et al. (12) showed decreases of 20 mmHg in SBP following 50 min of treadmill walking at 67% age-predicted maximal heart rate. They speculated that elevated body temperature promotes vasodilation for thermoregulation. As a result, central blood volume, stroke volume, and cardiac output decrease. Intensities over 50% VO_2max would be expected to result in increased vasodilation and improved thermoregulation (19). Coats et al. (10) stated that increased body heat is a possible mechanism for marked vasodilation, but they argued against relative hypovolemia. Although we did not measure temperature, the maximal exercise intensities used in the present study would be expected to elicit this temperature regulation response.

It is also possible that circulating metabolites contributed to the peripheral vasodilation. Adenosine, prostaglandins, opioids (22), H^+, potassium (19), and lactate (10) have been mentioned as potential factors although their relationship to post-exercise hypotension remains unclear (21). Given that blood samples were not collected in the present study, the relative influence of circulating metabolites on peripheral dilation could not be assessed.

Lastly, hormone release may have played a role in the observed vasodilation. Since exercise causes an increase in atrial natriuretic peptide (ANP) due to increased venous return and filling pressures in the right atrium, its release may induce vasodilation (22). Piepoli et al. (19) noted that DBP remains depressed during 1 hr of recovery, despite a three-fold increase in renin, which converts angiotensinogen to angiotensin I. Nitric oxide (NO) may also be involved due to its potent vasodilatory effect, although it may enhance postexercise blood flow only (14).

Halliwill et al. (23) reported lower mBP and peripheral resistance after 60 min of cycling at 60% VO_2peak despite elevated norepinephrine during recovery. It was concluded that the augmented baroreflex heart rate gain attenuates the post-exercise hypotension. Again, vasodilation was suggested as the contributing factor. In another study, Halliwill et al. (24) reported reduced SBP and mBP 60 min-post-exercise versus rest, and muscle sympathetic nerve activity was also lower than rest. These authors concluded that post-exercise vasodilation may be the result of modified baroreflex control of sympathetic outflow. This team also found impedance to the nerve impulses that control vascular resistance, suggesting a neural-vascular factor in post-exercise hypotension.

On the other hand, increases in VO_2max can occur independently of blood pressure. Spriet et al. (25) infused 3 units of red blood cells into highly-trained, endurance runners and found significant increases in VO_2max, while SBP decreased.

Franklin et al. (11) reported lower SBP and mBP during recovery in a warm environment compared to baseline values, although there were no corresponding differences in VO_2. In addition, they reported that VO_2 during recovery was higher than baseline values in a neutral temperature (21.4 °C) while blood pressure remained constant.

RECOMMENDATIONS AND CONCLUSIONS

This study reports new findings in the study of exercise blood pressure assessment. First, there is a significant initial hypotension after 11.6 s of low-intensity cycling (45%POmax). Second, the relationship between BP and VO_2 is nonlinear, suggesting that their association may be more complex than previously thought. Additionally, during the rest to exercise transition, the $t_{0.5}$ values showed that VO_2 changed more rapidly than BP, while the opposite response occurred after 45% POmax.

We also found a significant immediate decrease in SBP, DBP, and mBP post-exercise, which questions the use of IPE blood pressure measurement as an indication of maximal BP. Since direct measurement of blood pressure is not practical in the clinical setting, manual sphygmomanometry is often used. The findings of this study suggest the need to obtain maximal blood pressure recordings during the last 30 s of exercise. In conclusion, these results demonstrated that the observed post-exercise hypotension can modify the interpretation of maximal blood pressure during exercise. The attainment of actual maximal blood pressure appears to coincide with POpeak, not termination of exercise.

Address for correspondence: Robergs, RA, Ph.D., Exercise Physiology Laboratories, Johnson Center, Room B143, University of New Mexico, Albuquerque, NM, USA. 87131-1251. Phone: (505) 277-2658; FAX: (505) 277-9742; Email: **rrobergs@unm.edu**

REFERENCES

1.   Griffin, S. E., Robergs, R. A., and Heyward, V. H. Blood pressure measurement during exercise: a review. *Med Sci Sports Exerc* 1997;29:149-159.

2.   Karlefors, T., Nilsen, R., and Westling, H. On the accuracy of indirect auscultatory blood pressure measurements during exercise. *Acta Med Scand* 1966;180:81-87.

3.   Rowell, L. B., Brengelmann, G. L., Blackmon, J. R., Bruce, R. A., and Murray, J. A. Disparities between aortic and peripheral pulse pressures induced by upright exercise and vasomotor changes in man. *Circulation* 1968;37:954-964.

4.   Hollingsworth, V., Bendick, P., Franklin, B., Gordon, S., and Timmis, G. C. Validity of arm ergometer blood pressures immediately after exercise. *Am J Cardio* 1990;65:1358-1360.

5.   Holmgren, A. Circulatory changes during muscular work in man. *Scand J Clin Lab Invest* 1956;8:29-47.

6.   The sixth report of the Joint National Committee on prevention, detection, evaluation and treatment of high blood pressure. *Arch In. Med* 1997;157:2413-2446.

7.   Sheriff, D. D., Rowell, L. B., and Scher, A. M. Is rapid rise in vascular conductance at onset of dynamic exercise due to muscle pump? *Am J Physiol* 1993;265:H1227-H1234.

8.   Shoemaker, J. K. and Hughson, R. L. Adaptation of blood flow during the rest to work transition in humans. *Med Sci Sports Exerc* 1999;31:1019-1026.

9.   Tschakovsky, M. E., Shoemaker, J. K., and Hughson, R. L. Vasodilation and muscle pump contribution to immediate exercise hyperemia. *Am J Physiol* 1996;271:H1697-H1701.

10.   Coats, A. J. S., Conway, J., Isea, J. E., Pannarale, G., Sleight, P., and Somers, V. K. Systemic and forearm vascular resistance changes after upright exercise in man. *J Physiol* 1989;413:289-298.

11.   Franklin, P. J., Green, D. J., and Cable, N. T. The influence of thermoregulatory mechanisms on post-exercise hypotension in humans. *J Physiol* 1993;470:231-241.

12.   Kaufman, F. L., Hughson, R. L., and Schaman, J. P. Effect of exercise on recovery blood pressure in normotensive and hypertensive subjects. *Med Sci Sports Exerc* 1987;19:17-20.

13.   Kingwell, B. A., Berry, K. L., Cameron, J. D., Jennings, G. L., and Dart, A. M. Arterial compliance increases after moderate-intensity cycling. *Am J Physiol* 1997;273:H2186-H2191.

14.   Shoemaker, J. K., Halliwill, J. R., Hughson, R. L., and Joyner, M. J. Contributions of acetycholine and nitric oxide to forearm blood flow at exercise onset and recovery. *Am J Physiol* 1997;273:H2388-H2395.

15.   Delp, M. D. Control of skeletal muscle perfusion at the onset of dynamic exercise. *Med Sci Sports Exerc* 1999;31:1011-1018.

16.   Ebfeld, D., Hoffmann, U., and Stegemann, J. VO2 kinetics in subjects differing in aerobic capacity: investigation by spectral analysis. *Eur J Appl Physiol* 1987;56:508-515.

17.   Yoshida, T., Kamiya, J., and Hishimoto, K. Are oxygen uptake kinetics at the onset of exercise speeded up by local metabolic status in active muscles? *Eur J Appl Physiol* 1995;70:482-486.

18.   Grucza, R., Miyamoto, Y., and Nakazono, Y. Kinetics of cardiorespiratory response to dynamic and rhythmic-static exercise in men. *Eur J Appl Physiol* 1990;61:230-236.

19.   Piepoli, M., Coats, A. J. S., Adamopoulos, S., Bernardi, L., Feng, Y. H., Conway, J.et al. Persistent peripheral vasodilation and sympathetic activity in hypotension after maximal exercise. *J Appl Physiol* 1993; 75:1807-1814.

20.   Sprangers, R. L. J., Wesseling, A. L. T., Imholz, B. P. M., and Wieling, W. Initial blood pressure fall on stand up and exercise explained by changes in total peripheral resistance. *J Appl Physiol* 1991;70:523-530.

21.   Kenney, M. J. and Seals, D. R. Postexercise hypotension: key figures, mechanisms, and clinical significance. *Hypertension* 1993;22:53-664.

22.   Isea, J. E., Piepoli, M., Adamopoulos, S., Pannarale, G., Sleight, P., and Coats, A. J. S. Time course of haemodynamic changes after maximal exercise. *Eur J Clin Invest* 1994;24:824-829.

23.   Halliwill, J. R., Taylor, J. A., Hartwig, T. D., and Eckberg, D. L. Augmented baroreflex heart rate gain after moderate-intensity, dynamic exercise. *Am J Physiol* 1996;270:R420-R426.

24.   Halliwill, J. R., Taylor, A., and Eckberg, D. L. Impaired sympathetic vascular regulation in humans after acute dynamic exercise. *J. Physiol* 1996;495:279-288.