The influence of recovery posture on postexercise hypotension in normotensive men

NEIL M. RAINE, N. TIMOTHY CABLE, KEITH P. GEORGE, and IAN G. CAMPBELL

Research Institute for Sport and Exercise Sciences, Liverpool John Moores University, Liverpool, L3 2ET, Merseyside, UNITED KINGDOM; and Department of Exercise and Sport Science, The Manchester Metropolitan University, Crewe and Alsager Faculty, Stoke-on-Trent, ST7 2HL, UNITED KINGDOM

ABSTRACT

RAINE, N. M., N. T. CABLE, K. P. GEORGE, and I. G. CAMPBELL. The influence of recovery posture on post-exercise hypotension in normotensive men. Med. Sci. Sports Exerc., Vol. 33, No. 3, 2001, pp. 404-412. Purpose: Postexercise hypotension may be the result of an impaired vasoconstrictor response. This hypothesis was investigated by examining the central and peripheral hemodynamic responses during supine and seated recovery after maximal upright exercise. Methods: After supine or seated baseline measurements, seven normotensive male volunteers completed a graded upright cycling protocol to volitional exhaustion. This was immediately followed by either supine or seated recovery. Measurements of pulsatile arterial blood pressure and central and peripheral hemodynamic variables recorded 30 min before exercise were compared with those taken throughout 60 min of recovery. Results: Compared with baseline, mean arterial pressure (MAP) was reduced after exercise (P < 0.05) although the degree of change was not different between the supine $(-9 \pm 4 \text{ mm Hg})$ and seated positions $(-6 \pm 2 \text{ mm Hg})$. This change in MAP was associated with a reduction in diastolic blood pressure (DBP) (P < 0.05) and arterial pulse pressure (APP) (P < 0.01) for the supine and seated positions, respectively. The reduction in APP during seated recovery was accompanied by a decline in stroke volume (SV) (P < 0.05), not seen in the supine position, that limited the contribution of cardiac output (CO) to the maintenance of MAP. This effect of seated recovery was compensated by greater systemic (SVR) and regional vascular resistances in the forearm (FVR) and the forearm skin (SkVR_A). There was also evidence of an augmented return of FVR and SkVR_A to resting levels in the seated position after exercise. Conclusion: The lower peripheral resistance in the supine compared with seated recovery position suggests there is potential for greater vasoconstriction, although this is not evoked to increase blood pressure. This further suggests that the arterial baroreceptor reflex is reset to a lower operating pressure after exercise. Key Words: EXERCISE, BLOOD PRESSURE, SKIN BLOOD FLOW, VASOCONSTRICTION, BARORECEPTOR REFLEX

The moderate rise in mean arterial blood pressure that occurs during dynamic exercise is rapidly reversed after withdrawal of the rhythmic contractions of the skeletal muscles. Previous investigations utilizing a large muscle mass have shown that during this transition from exercise to rest, blood pressure may actually decline to levels below baseline resting values (1,3,4,7–10,20,26, 27,29). For both normotensive and hypertensive volunteers, postexercise hypotension has predominantly been associated with a reduction of systemic vascular resistance (3,4,10,26), whereas in older hypertensive subjects a decline in the cardiac output has been implicated (9).

One of the possible mechanisms underlying postexercise hypotension is some alteration in peripheral vascular regulation. For a given regional circulation, reduced vascular resistance may be associated with the persistence of a vasodilator signal after exercise, an inappropriate compensatory vasoconstrictor stimulus, or impaired vascular responsiveness to a vasoconstrictor stimulus. These mechanisms would not necessarily operate independently of each other

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during the recovery from exercise. The resistance vessels in exercised skeletal muscle clearly remain dilated once physical activity ends and the hyperemia in this region tends to be prolonged (6,16). Coats et al. (4) hypothesized that a metabolic signal is responsible for the continued skeletal muscle vasodilatation after exercise. This is supported by evidence of a relationship between the production of lactate during exercise and total iliac vein blood flow throughout the first 6 min of recovery (5). Other potential mechanisms underlying the reduction of skeletal muscle vascular resistance after exercise include a reduction in muscle sympathetic nerve activity (7,10) and impaired vascular responsiveness to sympathetic stimulation in the exercise limb (10). The latter observation is supported by *in vitro* (13) and in vivo studies (12) that demonstrated attenuated vascular responses to α -adrenergic receptor activation after exercise compared with control conditions. Vasodilation of the cutaneous vessels has also been implicated in the postexercise reductions of regional and systemic vascular resistance (8). This hypothesis was based upon evidence of greater reductions in blood pressure during recovery in a warm compared with a thermoneutral environment when both core and skin temperatures were elevated (8). This, however, has yet to be confirmed through the measurement of skin blood flow before and after exercise.

The importance of the peripheral circulation to blood pressure regulation was realized through the study of regional vasoconstrictor responses to upright posture and simulated gravitational stress (28). To compensate for the decline in stroke volume and cardiac output that occurs under these conditions, vasoconstrictor outflow is directed toward the skeletal muscle, splanchnic (18) and cutaneous circulations (17,21). Peripheral vasoconstriction is also particularly important after exercise when the tolerance to gravitational stress is diminished (2) leading to postural hypotension (11). Despite this evidence, blood pressure and cardiovascular responses after exercise have yet to be examined in relation to the recovery posture adopted. This model could provide important information concerning the significance of any alteration in peripheral vascular regulation to postexercise hypotension. Furthermore, posture may be the confounding variable underlying the conflicting reports of predominant changes in diastolic or arterial pulse pressure after exercise. This problem has also not been addressed in the current literature.

In the present study, measurements of blood pressure and central and peripheral hemodynamic variables were compared before and after exercise under the two experimental conditions of supine and seated posture. The hypothesis developed was that arterial hypotension will be exacerbated under the combined influence of postexercise vasodilation and gravitational stress compared to a supine recovery condition.

METHODS

Subjects

Seven male subjects (mean \pm SEM: age, 24 \pm 1 yr; height, 177 \pm 2 cm; and body mass, 74 \pm 3 kg) gave written informed consent to participate in the investigation after an explanation of procedures that were formally approved by the University Human Ethics Review Committee. All participants were normotensive nonsmokers taking no medication. Before the first experiment each volunteer was familiarized with the exercise testing protocol and the laboratory setting.

Experimental Protocol

Baseline measurements of pulsatile arterial blood pressure, heart rate, stroke volume, cardiac output, and peripheral blood flow were compared with those taken at intervals after exercise under the two experimental conditions of supine and seated posture. The experiments were separated by a 1-wk interval and controlled for a potential time of day effect. Each evaluation lasted approximately 2.5 h.

Subjects attended the laboratory after an overnight fast and a 24-h abstinence from alcohol and exercise. The order of experimental conditions (supine or seated posture) was randomly assigned and the subjects were not informed until the first visit to the laboratory. The laboratory environment was kept comfortably warm [mean (\pm SEM) temperature: supine condition, 23.9 \pm 0.2°C; seated condition, 24.3 \pm 0.4°C; relative humidity: supine condition, $52 \pm 3\%$; seated condition, $55 \pm 4\%$], quiet, and dimly lit. The initial 30 min of each experiment, necessary to position monitoring devices, allowed the subjects time to adjust to the controlled laboratory setting and the required posture. Participants then rested for 30 min on a bed in the supine posture or sat in an armchair while baseline measurements were taken. Pulsatile arterial blood pressure and heart rate were recorded continuously and the data sampled at 30-s intervals. Skin blood flow was measured continuously and forearm blood flow and cardiac output at 15-min intervals before and after exercise. All of the monitoring probes remained in place during the exercise bout, and subjects were asked not to grip the handlebars with the hand of the arm used for blood flow measurements. Immediately after exercise, subjects recovered in the supine or seated posture under identical laboratory conditions for a further 60-min period of measurement.

Upright cycling exercise was performed on an electrically braked isokinetic cycle ergometer (Cybex, Ronkonkoma, NY) and began with a 2-min warm-up at a work load of 100 W. Subjects then progressed to a graded exercise protocol that commenced at a work load of 120 W and increased by 30-W increments every 2 min until volitional exhaustion was reached. To determine the point of volitional exhaustion for each subject, the following criteria were used: 1) an inability to maintain the desired work load, 2) heart rate in excess of 180 beats min⁻¹, and 3) a respiratory exchange ratio above the value of 1.1. Expired respiratory gases were collected continuously throughout exercise and sampled at 20-s intervals by an open-circuit spirometry gas analysis system (Sensor Medics, Anaheim, CA). This equipment was first calibrated to known concentrations of O₂ and CO₂, a given volume of air, and to the atmospheric pressure. Respiratory gas concentrations and the ventilatory volume were relayed to an on-line microcomputer that calculated the volume of oxygen consumed ($\dot{V}O_2$). The peak oxygen uptake ($\dot{V}O_2$ peak) was determined as the highest $\dot{V}O_2$ value attained. Heart rate was measured continuously throughout the exercise protocol using an ambulatory monitor (Polar Sports Tester, Kempele, Finland).

Measurements

Arterial blood pressure and heart rate. Continuous pulsatile systolic blood pressure (SBP), diastolic blood pressure (DBP), and heart rate (HR) were measured noninvasively from the finger using a photoplethysmographic device (Ohmeda Finapres 2300, Englewood, U.S.). This instrument has previously been validated against intra-arterial pressure measurements (15) and provides an accurate measure of blood pressure changes during various laboratory tests (25). Allowing a 30-min period of adjustment to laboratory temperature controlled for the influence of digital artery constriction on Finapres blood pressure measurements (22). Blood pressure was measured from the left hand that was comfortably elevated to the level of the heart using foam supports. Postexercise measures were averaged over 5-min intervals and compared with mean values recorded

during the baseline evaluation period. Arterial pulse pressure (APP) was calculated as SBP – DBP. MAP was calculated according to the formula; MAP = DBP + 1/3 APP.

Stroke volume and cardiac output. Subjects were considered "echogenic" allowing clear delineation of major cardiac structures. Stroke volume (SV) was determined echocardiographically using a HP Sonos 100 Ultrasound System (Hewlett-Packard, Andover, MA) with a 2.5-MHz transducer. A 2-D sector scan of the left ventricle outflow tract was gained from a parasternal long axis view. Placement of a cursor line at the level of the aortic valve allowed an M-mode trace of the aortic tract and delineation of the opening and closure of the aortic valve leaflets. Placement of the ultrasound transducer in the suprasternal notch allowed a 2-D-sector scan of the ascending aorta. The sample volume was orientated parallel to flow in the aorta to facilitate pulsed-wave Doppler echocardiographic representation of the aortic flow waveform. Using software inherent to the Sonos 100, videotaped recordings of 3-5 consecutive cardiac cycles were later digitized by an experienced echocardiographer. From the M-mode trace, the diameter (D) of the annulus of the aortic valve was measured (14). The crosssectional area (CSA) of the open valve was then determined using the following equation: $CSA = (\pi D^2)/4$. From the Doppler trace, the outline of the waveform was digitized to calculate flow velocity integral (FVI, cm). The FVI and CSA measurements permitted the calculation of stroke volume according to the following equation: $SV = CSA \times$ FVI. Cardiac output (CO) was calculated from simultaneous heart rate and stroke volume measurements. Systemic vascular resistance (SVR) was derived according to the formula: $SVR = MAP \div CO$.

From the M-mode trace of the left ventricle, the ventricular internal dimension during diastole (LVIDd) was measured at the peak of the ECG R-wave. As an index of ventricular contractility, ventricular ejection fraction percentage was then calculated according to the formula: (SV \div LVIDd³) \times 100 (23).

Forearm blood flow. Forearm blood flow (FBF) was measured by venous occlusion plethysmography using 17.5 cm mercury-in-Silastic strain gauges. The output voltage from the plethysmograph (Parks Medical Electronics, Oregon) was relayed to a personal computer comprising an eight-channel 12-bit analog-to-digital converter, sampled at a frequency of 128 Hz and displayed in real time on a chart recorder (Perimed, Jarfalla, Sweden). The data for forearm blood flow was stored on diskette and later analyzed manually by the same experimenter using the chart recorder software to determine the slope of the relationship between time and plethysmograph output voltage. The increase in forearm circumference during venous occlusion was then determined using the linear regression equation calculated for each individually calibrated strain gauge. To perform measurements, the mercury strain gauge was first mounted on a lightweight adjustable frame and passed around the circumference of the forearm at a standard position 5 cm below the antecubital crease. The collecting cuff on the

upper arm was then rapidly inflated (Hokanson, Washington) to a pressure of 50 mm Hg in a cycle of 10 s inflation–5 s deflation for a total of 3 min. Venous drainage was encouraged by supporting the wrist slightly above the height of the elbow. Circulation to the hand was occluded at a pressure of 200 mm Hg for 1 min before each venous occlusion cycle commenced. Measurements taken during the last 1 min of each 3-min occlusion cycle were used in the subsequent analysis. Forearm vascular resistance (FVR) was calculated from simultaneous records of pulsatile MAP and forearm blood flow and is presented as peripheral resistance units (P.R. units). In this laboratory we have calculated the day-to-day reliability of the venous occlusion technique to be within satisfactory limits, 95% limits of agreement = -0.81 to 1.1 mL·100 mL tissue⁻¹·min⁻¹.

Skin blood flow. Skin blood flow changes were measured noninvasively from the lateral calf (SkBF_C) and contralateral forearm (SkBF_A) to blood pressure measurements by laser-Doppler flowmetry using a laser Doppler perfusion monitor (Periflux 4001, Perimed, Jarfalla, Sweden). Laser-Doppler probes remained attached to the skin during exercise to eliminate the error of repositioning over a different skin site. Although this technique does not derive an actual value for skin blood flow, the Doppler shift in laser generated light is proportional to the changes in skin blood flow (28). Skin blood flow after exercise was averaged over 10-min periods and compared with values measured during the control period. All skin blood flow data are reported as perfusion units (P.U.). Skin vascular resistances in the calf (SkVR_C) and forearm (SkVR_A) were calculated from simultaneous skin blood flow and MAP determinations and are presented as peripheral resistance units (P.R. units).

Statistical Analysis

All results are expressed as mean values \pm standard error of the mean (SEM). Baseline measurements recorded for the supine and seated conditions were compared using a dependent *t*-test. Comparisons between baseline and postexercise measures between the two experimental conditions were made using a two-way analysis of variance (ANOVA) design with repeated measures on both factors (time and posture). Any violations to the assumption of sphericity were controlled by correcting the degrees of freedom by the Huynh-Feldt epsilon (30). Significance was accepted at a probability level of P < 0.05. All statistical analysis was performed using the Statistical Package for the Social Sciences (SPSS).

RESULTS

Preexercise Resting Variables and Exercise Variables

Resting variables. Arterial blood pressure variables were not different between the supine and seated positions throughout the 30-min period of baseline rest. Stroke volume and cardiac output were lower and systemic and

forearm vascular resistances were greater throughout seated compared to supine rest (Table 1).

Exercise variables. Subjects had reached a work load 339 ± 18 W after 15.9 ± 1.1 min of upright cycling exercise to exhaustion during both experimental conditions. At the end of exercise, maximal heart rate (187 ± 3 beats·min⁻¹—supine condition; 183 ± 3 beats·min⁻¹—seated condition) and the maximal oxygen consumption (3608 ± 181 mL [48.9 ± 3.1 mL·kg⁻¹·min⁻¹]—supine condition; 3577 ± 156 mL [48.4 ± 2.3 mL·kg⁻¹·min⁻¹]—seated condition) were not different (P > 0.05).

Postexercise variables. Arterial blood pressure and heart rate. Pulsatile SBP (P < 0.01), DBP (P < 0.05), MAP (P < 0.05) (Fig. 1) and APP (P < 0.05) (Fig. 2) were all reduced after exercise compared with baseline values. Throughout the entire 60 min recovery period, the mean reductions in SBP ($-8 \pm 3 \text{ mm Hg}$ —supine condition; $-11 \pm 3 \text{ mm Hg}$ —seated condition) (P = 0.32), and MAP ($-9 \pm 4 \text{ mm Hg}$ —supine condition; $-6 \pm 2 \text{ mm Hg}$ —seated condition) (P = 0.51) were not influenced by the posture adopted. In contrast, for the seated recovery condition the change in DBP after exercise was markedly less (P < 0.05) and the absolute DBP was greater compared with the supine condition ($59 \pm 2 \text{ mm Hg}$ —supine condition; $68 \pm 2 \text{ mm Hg}$ —seated condition) (P < 0.01) (Fig. 1).

In the supine posture APP returned back to the baseline level following exercise but remained markedly depressed in the seated position (P < 0.01) (Fig. 2). The maximum change in APP occurred between 20–25 min of seated recovery from exercise ($-14 \pm 2 \text{ mm Hg}$) and remained reduced after 60 min of rest ($-8 \pm 2 \text{ mm Hg}$). Heart rate was elevated after exercise and remained 13 ± 2 beats·min⁻¹ and 17 ± 2 beats·min⁻¹ above baseline values after 60 min of supine and seated recovery, respectively (P < 0.01). Although absolute heart rate was greater in the seated compared with the supine position (P < 0.05) the heart rate change compared to baseline was not different (P = 0.66) (Figure 2).

Central hemodynamic variables. During recovery in the seated posture, SV was reduced compared to baseline (P < 0.05), whereas in the supine posture SV was nonsignificantly elevated. The maximum changes in SV occurred

TABLE 1. Baseline blood pressure and central and peripheral hemodynamic variables. Heart rate, systemic, and peripheral vascular resistance were greater during seated rest. In the seated position these responses maintained mean arterial pressure close to supine values.

	Supine Posture	Seated Posture	
SBP	124 \pm 4 mmHg	125 \pm 3 mmHg	NS
DBP	65 ± 4 mmHg	$69 \pm 3 \text{ mmHg}$	NS
MAP	$84 \pm 4 \text{ mmHg}$	$88 \pm 3 \text{ mmHg}$	NS
APP	$59 \pm 4 \text{ mmHg}$	$55 \pm 4 \text{ mmHg}$	NS
HR	$55 \pm 3 \text{ beats-min}^{-1}$	$62 \pm 3 \text{ beats} \cdot \text{min}^{-1}$	* *
SV	$128 \pm 8 \text{ ml}$	$83 \pm 7 \text{ ml}$	* *
CO	7.1 ± 0.6 1·min ⁻¹	5.1 ± 0.6 1·min ⁻¹	*
SVR	12.7 ± 1.3 P.R.units	18.4 \pm 1.6 P.R.units	* *
FVR	26 ± 2 P.R.units	45 ± 3 P.R.units	**
SkVR₄	15 ± 2 P.R.units	22 \pm 3 P.R.units	NS
SkVR	6 ± 1 P.R.units	20 ± 2 P.R.units	**

Variables are expressed as mean values (\pm S.E.M). Significant difference for dependent *t*-test; * P < 0.05; **P < 0.01.



FIGURE 1—Mean (±SEM) change in systolic blood pressure (Δ SBP), diastolic blood pressure (Δ DBP), and MAP (Δ MAP) during 60 min of supine (\bullet) and seated recovery (\square) from exhaustive upright cycling exercise. Marked reductions in all blood pressure variables were observed after exercise. The change in diastolic blood pressure from baseline was greater in the supine compared to the seated recovery position (P < 0.05).

after 30 min of supine (14.5 ± 8.8 mL, 11%) and 45 min of seated recovery (-14.9 ± 3.7 mL, -18%). Absolute SV remained greater in the supine compared with the seated posture throughout the 60-min recovery period (P < 0.01) (Fig. 3). After exercise CO was markedly elevated above resting values (P < 0.01). Both the absolute values for CO (P < 0.001) and the change in CO from baseline were greater in the supine compared with the seated position (P < 0.05). These changes in CO amounted to 5.1 ± 1.0 L·min⁻¹



FIGURE 2—Mean (±SEM) change in arterial pulse pressure (Δ APP) and heart rate (Δ HR) throughout 60 min of supine (\bullet) and seated recovery (\Box) from exhaustive upright cycling exercise. The change in APP was markedly greater during recovery in the seated position (P < 0.01) and remained depressed for the entire postexercise observation period. Heart rate remained elevated after 60 min of rest (P < 0.01), and the return to baseline was attenuated in the seated posture (P < 0.05).

(71%) and 1.6 \pm 0.5 L·min⁻¹ (30%) after 15 min of supine and seated recovery, respectively (Fig. 3). Throughout recovery, calculated SVR was reduced compared with baseline (P < 0.01) but remained markedly greater in the seated compared with the supine position (P < 0.01) (Fig. 3). The changes in SVR in the supine (-6.0 ± 0.9 P.R. units, -47%at 15-min recovery) and seated recovery positions ($-4.7 \pm$ 1.0, -25%, at 15-min recovery) after exercise were not different (P = 0.4) (Fig. 3).

Compared with resting baseline measurements (65.0 \pm 2.0%—supine condition; 61.1 \pm 1.9%—seated condition), calculated ventricular ejection fraction percentage was increased after exercise (P < 0.001). After 15 min of recovery, the values for ejection fraction percentage amounted to 72.2 \pm 0.7% and 71.1 \pm 1.5% for the supine and seated positions, respectively. These changes in ventricular ejection fraction percentage after exercise were not influenced by the recovery posture adopted (P = 0.23).

Peripheral blood flow variables. There was an increase in FBF after exercise (P < 0.01), but the absolute level remained markedly greater in the supine compared with the seated posture (P < 0.01). After 5-min recovery,



Time before and after exercise (min)

FIGURE 3—Mean (±SEM) stroke volume, cardiac output and calculated systemic vascular resistance (SVR) 30 min before and during 60 min of supine (•) and seated recovery (_) from exhaustive upright cycling exercise. Stroke volume was below baseline in the seated position after exercise (P < 0.05) but was unchanged during supine recovery. The rise in cardiac output after exercise was less in the seated compared with the supine posture (P < 0.05) although this effect was counteracted by a greater absolute SVR in the upright position (P < 0.01).

the peak change in FBF was not different between the supine $(1.7 \pm 0.5 \text{ mL} \cdot 100 \text{ mL tissue}^{-1} \cdot \text{min}^{-1})$ and seated conditions $(1.5 \pm 0.1 \text{ mL} \cdot 100 \text{ mL tissue}^{-1} \cdot \text{min}^{-1})$ (P = 0.41) (Fig. 4). FVR was reduced after exercise (P < 0.01) but remained markedly greater throughout the seated compared with the supine recovery condition (P < 0.01). After 5 min of rest, the mean changes in FVR amounted to -11.0 ± 2.0 P.R. units (-42%)–supine recovery, and -21.6 ± 2.9 P.R.

units (-48%)—seated recovery. Although the change in FVR from baseline was not influenced by the recovery posture adopted (P = 0.47), the return to the preexercise level was augmented in the seated position (P < 0.01) (Fig. 4).

After exercise there was a nonsignificant increase in both $SkBF_{C}$ (P = 0.19) and $SkBF_{A}$ (P = 0.13). Although $SkBF_{C}$ remained greater in the supine (24 ± 4 P.U.—10 min postexercise) compared with the seated posture (8 \pm 2 P.U.—10 min postexercise) after exercise (P < 0.05), no such difference was observed for SkBF_A (29 \pm 10 P.U.—supine condition; 16 \pm 3 P.U.—seated condition, at 10 min postexercise) (P = 0.21). After exercise both calculated SkVR_C (P < 0.05) and SkVR_A (P < 0.01) were reduced compared with baseline levels. Although absolute SkVR_C remained greater in the seated (13.1 \pm 2.0 P.R. units-10 min postexercise) compared with the supine posture (3.3 \pm 0.4 P.R. units—10 min postexercise) after exercise (P < 0.01), no postural effect was identified for $SkVR_A$ (P = 0.11). The change in $SkVR_C$ from the resting level was, however, not different between the supine and seated recovery conditions (P = 0.98). There was also evidence of an augmented return of SkVRA toward baseline for the seated compared with the supine condition (P < 0.05) (Fig.

5), and although a similar pattern was observed for SkVR_C, this interaction effect was not statistically significant (P = 0.067).

DISCUSSION

In the present investigation, blood pressure and central and peripheral hemodynamic variables were compared before and after maximal upright cycling exercise under the two experimental conditions of supine and seated posture. The results revealed the presence of a marked but transient reduction in mean arterial blood pressure after exercise that had returned to baseline resting values after 60 min of recovery. In contrast to the original hypothesis of the study based upon evidence of altered peripheral vascular regulation following exercise (10,12,13), these changes in MAP were similar whether recovery was undertaken in the supine or the seated position. This indicated that the regulation of blood pressure was not further compromised by superimposing gravitational stress onto the postexercise situation.

Changes in the gross hemodynamic profile after exercise were similar between the two recovery conditions and were characterized by a decline in calculated total peripheral resistance (TPR) and an elevation in the cardiac output.



FIGURE 4—Mean (±SEM) forearm blood flow (FBF), forearm vascular resistance (FVR) at rest, and throughout 60 min of supine (\oplus) and seated recovery (\square) from exhaustive upright cycling exercise. Compared with baseline, the changes in FBF and FVR after exercise were not influenced by the recovery posture adopted. FVR remained markedly greater throughout seated compared to supine recovery (P < 0.01), and there was evidence of an augmented return to the resting level.

FIGURE 5—Mean (±SEM) calculated skin vascular resistance in the calf (SkVR_C) and the forearm (SkVR_A) at rest and throughout 60 min of supine (•) and seated recovery (□) from exhaustive upright cycling exercise. Skin vascular resistance was reduced after exercise (P < 0.05). A postural effect was observed for SkVR_C before and after exercise (P < 0.01), but this was not seen for SkVR_A.

However, despite the persistent tachycardia after exercise, the increase in cardiac output in the seated position was limited by a sustained depression of stroke volume. This observation was reflected by a reduction in arterial pulse pressure that remained depressed throughout the 60 min of recovery. In contrast to seated recovery, no such changes in stroke volume and arterial pulse pressure were observed for the supine condition, indicating that venous return of blood to the heart was not compromised in this position after exercise. The likely mechanism underlying the decline in stroke volume during seated recovery was a decrease in cardiac filling given that afterload pressure was actually reduced after exercise. This change in stroke volume also occurred at a time when ventricular contractility was enhanced although this appears to have had little effect given the low ventricular volumes. In contrast to the results of the present study, Bjurstedt et al. (2) concluded that reduced cardiac filling was not the primary mechanism responsible for impaired orthostatic tolerance after exhaustive exercise. This was based upon evidence of similar decrements in central venous pressure under orthostatic stress before and after exercise. Rather, the primary mechanism was considered to be some impairment to peripheral vasoconstriction (2). Central venous pressure was, however, below zero in these subjects before the institution of orthostatic stress after exercise. Furthermore, arterial pulse pressure was the primary variable to change during passive tilting after exercise and may have been related to a greater decline in stroke volume (2). Indeed, any alteration to peripheral vascular regulation and cardiac output under orthostatic stress after exercise would not necessarily occur independently. Any increase in peripheral blood flow leading to increased venous pressure and volume in the dependent limbs would be predicted to impair venous return in the absence of muscle pumping action (28).

The cutaneous vasodilatation that occurs during exercise (19) extended into the periods of both supine and seated recovery. This has previously been inferred from changes in hand vascular resistance (3); however, we have provided direct evidence of a postexercise cutaneous vasodilation for both the active and nonactive limbs. The observed increase in total forearm blood flow also presumably reflected this cutaneous vasodilation. When prolonged gravitational stress was imposed after exercise, vascular resistance in the calf skin and the total forearm remained markedly greater than that observed in the supine position. Forearm skin vascular resistance was also greater in the seated position after exercise, although this did not approach the required level of probability. This disparity between skin sites in the cutaneous vascular response to upright posture (at rest and postexercise) may have been related to the position of the respective limbs. Although the leg was made dependent in upright posture, the forearm remained horizontal in both body positions. For the calf skin, such a positional change would be predicted to activate a local sympathetic venoarteriolar reflex that initiates vasoconstriction in addition to that provided by central sympathetic outflow (28).

c stress after ndently. Any increased venbs would be the of muscle ring exercise e and seated m changes in ave provided sodilation for rved increase reflected this the intensity of exercise in the seated posture, for the regions examined here, it is clear that there was a large capacity for enhanced vasoconstriction in the supine recovery condition but this was not utilized to restore arterial pressure back to preexercise levels. As both the degree and pattern of change for MAP was similar for the recovery postures and the vasoconstrictor activity different, these results do not support the hypothesis of impaired vascular regulation as an important

whether a proportion of the cutaneous vasoconstriction seen after exercise was initiated by cardiopulmonary or arterial baroreceptor populations. Reductions in ventricular filling and arterial pulse pressure in the seated recovery position would be predicted to inhibit both of these receptor populations, respectively. With regard to the responsiveness of the cutaneous circulation, similar observations to those reported here have been made in hyperthermic humans during whole body heat stress. Under these conditions, simulated gravitational stress results in a marked cutaneous vasoconstriction. However, baroreflexes do not completely override thermoregulatory reflexes and the skin remains relatively vasodilated compared to thermoneutral conditions (17,21). The present study has provided evidence that this competition between thermoregulatory and baroreflex control of skin blood flow (17,21) also applies to the postexercise situation. Although skin vascular resistance remained greater during seated compared to supine recovery, values were below those recorded during seated rest and were indicative of a relative cutaneous vasodilation after exercise.

The additional vasoconstriction in the total forearm and

calf skin circulations during seated compared with supine recovery provided evidence that these regions retained the

ability to vasoconstrict after the initiation of a baroreflex after exercise. From the present results it remains uncertain

In light of the present results, the influence of recovery posture on mechanisms of thermoregulation after exercise warrants investigation. Any reduction in cutaneous blood volume in the seated compared to the supine recovery position would be predicted to impair the degree of convective heat loss during the recovery from exercise. This may have important implications regarding the management of individuals presenting with postevercise hyperthermia

after exercise. Given the markedly greater regional vascular resistance in the seated posture, for the regions examined here, it is clear that there was a large capacity for enhanced vasoconstriction in the supine recovery condition but this was not utilized to restore arterial pressure back to preexercise levels. As both the degree and pattern of change for MAP was similar for the recovery postures and the vasoconstrictor activity different, these results do not support the hypothesis of impaired vascular regulation as an important mechanism underlying postexercise hypotension (12,13,10). However, we did not measure blood flow in the exercised limb and could not directly evaluate the influence of posture on vascular resistance in this region. Nevertheless, impaired vasoconstriction in exercised muscle after exercise would not necessarily alter the regulation of blood pressure given that under conditions of high vascular conductance a given increase in vascular resistance leads to a greater change in blood flow (according to Poiseuille's Law). In this study during seated recovery, there was also evidence of an augmented return of vascular resistance toward resting levels in the total forearm and the forearm skin circulations. This supports the findings of Bennett et al. (1) of enhanced forearm vasoconstrictor responses to simulated gravitational stress after leg exercise. Thus, vasoconstriction in regions other than exercised muscle may potentially compensate for the impaired vascular regulation in this region during the recovery period.

The baroreceptor mechanism would be predicted to respond to arterial hypotension after exercise by increasing sympathetic nerve activity both to the heart and peripheral vasculature. On the contrary, Bennett et al. (1) hypothesized that baroreflex control of sympathetic outflow may be altered by exercise. This was based upon evidence of altered cardiopulmonary baroreflex control of forearm vascular resistance after exercise assessed using lower body negative pressure (1). The above hypothesis is also supported by the observations of reduced muscle sympathetic nerve activity in recovery (7,10). In the present study, the observation of an unutilized capacity for vasoconstrictor activity (i.e., greater peripheral resistance measured in the seated versus supine posture) supports these previous reports. However, given that the degree of arterial hypotension was the same in both postures, a further intriguing possibility is that the arterial baroreceptor reflex is reset to a lower operating pressure immediately postexercise. In this regard, there is recent evidence that during dynamic exercise up to maximal levels the carotid arterial baroreflex is reset to a higher operating pressure with no change in the maximal reflex gain compared to the resting situation (24). Given this dynamic feature of the arterial baroreflex during exercise and in light of the above observations of reduced muscle sympathetic nerve activity in recovery, the hypothesis of arterial baroreflex resetting after exercise deserves investigation.

Based upon the findings of this study there are some practical implications for the recovery of individuals after dynamic exercise up to maximal levels. Although the postexercise reductions in MAP were similar, in the seated compared with supine recovery position venous return and

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stroke volume were compromised, heart rate was elevated and there was a greater reliance on mechanisms of peripheral vasoconstriction. This pattern of response indicated that the demand placed upon the circulation to maintain arterial pressure was greater when the seated position was adopted after exercise. Assuming an upright standing rather than a sitting position into recovery would theoretically exacerbate this problem further. We therefore suggest that recovery in the supine position would reduce the likelihood of developing hypotension to levels sufficient to induce syncope after exercise. It is also worthy to note that the subjects in this study were young and generally physically fit. For older hypertensive patients, postexercise hypotension has been explained by a different physiological mechanism to that described here, a reduction in cardiac output subsequent to impaired left ventricular function (9). Whether the recovery posture alters the pattern of the postexercise cardiovascular response in this population remains to be determined experimentally.

In conclusion, the results of this investigation have provided additional evidence of a marked but transient reduction in mean arterial blood pressure after a single bout of upright cycling exercise to exhaustion. In the seated recovery position, there was a prolonged reduction in arterial pulse pressure, whereas for the supine condition there were corresponding changes in systolic and diastolic blood pressure. Stroke volume was also reduced throughout seated recovery from exercise and this limited the contribution of cardiac output to the regulation of blood pressure. The capacity for peripheral vasoconstriction clearly was not utilized to return blood pressure back to baseline values in the supine position raising the possibility that blood pressure is regulated about a lower value after exercise.

Address for correspondence: Dr. N. T. Cable, Research Institute for Sport and Exercise Sciences, Liverpool John Moores University, Trueman Building, Webster Street, Liverpool L3 2ET, Merseyside, United Kingdom; E-mail: t.cable@livjm.ac.uk.

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