Muscle pump and central command during recovery from exercise in humans

ROBERT CARTER, III,1,2 DONALD E. WATENPAUGH,1 WENDY L. WASMUND,1 STEPHEN L. WASMUND,2 AND MICHAEL L. SMITH1

1Department of Integrative Physiology, University of North Texas Health Science Center, Fort Worth, Texas 76107; and 2Department of Biology, College of Science, Southern University, Baton Rouge, Louisiana 70813

Carter, Robert, III, Donald E. Watenpaugh, Wendy L. Wasmund, Stephen L. Wasmund, and Michael L. Smith. Muscle pump and central command during recovery from exercise in humans. J. Appl. Physiol. 87(4): 1463–1469, 1999.—We sought to determine the relative contributions of cessation of skeletal muscle pumping and withdrawal of central command to the rapid decrease in arterial pressure during recovery from exercise. Twelve healthy volunteers underwent three exercise sessions, each consisting of a warm-up, 3 min of cycling at 60% of maximal heart rate, and 5 min of one of the following recovery modes: seated (inactive), loadless pedaling (active), and passive cycling. Mean arterial pressure (MAP), cardiac output, thoracic impedance, and heart rate were measured. When measured 15 s after exercise, MAP decreased less (P < 0.05) during the active (−3 ± 1 mmHg) and passive (−6 ± 1 mmHg) recovery modes than during inactive (−18 ± 2 mmHg) recovery. These differences in MAP persisted for the first 4 min of recovery from exercise. Significant maintenance of central blood volume (thoracic impedance), stroke volume, and cardiac output paralleled the maintenance of MAP during active and passive conditions during 5 min of recovery. These data indicate that engaging the skeletal muscle pump by loadless or passive pedaling helps maintain MAP during recovery from submaximal exercise. The lack of differences between loadless and passive pedaling suggests that cessation of central command is not as important.

cardiac output; arterial blood pressure; heart rate; peripheral resistance; stroke volume

DURING SUSTAINED DYNAMIC EXERCISE, mean arterial pressure (MAP) and cardiac output (CO) increase progressively with increasing workload (23). The increase in CO causes an elevation of systolic arterial pressure (SAP) and MAP, whereas diastolic arterial pressure (DAP) changes are minimal (1). Increased arterial pressure during sustained dynamic exercise is mediated by inputs from central command, metaboreflexes, baroreflexes, and the skeletal muscle pump (3, 13, 14, 21, 24, 25). This is accompanied by vasodilatation in the active muscles, leading to a progressive net decrease in total peripheral resistance (TPR) as the workload is increased (14). Therefore, the net increase in MAP is due to the increase in CO.

Inactive recovery from dynamic exercise is associated with cessation of the primary exercise stimulus from the brain (central command from the cerebral motor cortex) and abrupt changes in the stimuli to metaboreceptors and baroreceptors (21, 24). Skeletal muscle pumping, which contributes to increases in venous return during exercise, is also stopped during inactive recovery from exercise. Arterial pressure decreases rapidly during inactive recovery and can be accompanied by decreased cerebral blood flow and associated post-exertional orthostatic hypotension and syncope (3, 4, 11, 28). The skeletal muscle pump and central command each contribute importantly to the elevation and maintenance of arterial pressure during exercise (24), but the relative role of each in arterial pressure regulation during recovery from exercise is unknown. Therefore, the purpose of this study was to assess the relative roles of skeletal muscle pumping and central command during exercise recovery. We hypothesized that withdrawal of skeletal muscle pumping is the primary determinant of rapid decreases in arterial pressure during exercise recovery and that cessation of central command plays a relatively less substantial role.

To test this hypothesis, we compared responses to three different cycling-exercise recovery modes: 1) inactive recovery, in which the subject stopped exercise and sat completely still; 2) active recovery, during which the subject pedaled against zero resistance following exercise; and 3) passive recovery, during which the subject's legs were passively pedaled for them on a tandem bicycle at the same rate as during active recovery. The principal difference between active and passive recovery is the presence of central command during active recovery; therefore, we attributed any difference in response between these two conditions to the influence of central command. Similarly, the principal difference between inactive and passive recovery is the ongoing skeletal muscle pumping during passive recovery. Therefore, these comparisons allowed us to make assertions about the contributions of changes in skeletal muscle pumping and central command to the net hemodynamic response during recovery from exercise.

METHODS

Subjects. Twelve volunteers (six men and six women) between 21 and 39 yr of age were studied. All subjects were moderately fit, with a maximal oxygen uptake value of 40–60 ml·kg−1·min−1, based on a submaximal graded-exercise test. All subjects were free of any known cardiovascular disease. Female subjects were not tested during menses. Subjects were asked to refrain from exercise and stimulants such as caffeine for 24 h before testing. All experimental procedures were approved by the local institutional review board.
and protocols were approved by the University of North Texas Health Science Center Human Research Review Committee, and each subject gave informed written consent to participate in the study.

Experimental design. Each subject repeatedly performed an exercise protocol that consisted of a 1-min warm-up period on a cycle ergometer (either standard or tandem) with no resistance, followed by 1–3 min of increased workload to elicit ~60% of their individual predicted maximal heart rate (HR) with a constant pedal rate of 70 rpm. Subjects sustained exercise at their peak workload for 3 min. We studied three different cycling-exercise recovery modes: 1) inactive seated; 2) active loadless pedaling on the standard ergometer; or 3) passive cycling on the tandem cycle, with the pedal rate maintained constant (70 rpm) by a tandem partner. On separate days, two protocols were performed in random order: inactive and loadless pedaling recoveries were performed on one day, and inactive and passive pedaling recovery modes were performed on the other day. In every case, recovery was studied for 5 min. During the passive recovery protocol, the subject’s feet were secured to the pedals of the tandem cycle ergometer. Subjects were instructed and reminded to keep their legs fully relaxed so as to neither contribute to nor resist the pedal rotation.

We selected passive pedaling as a recovery mode to examine the role of the skeletal muscle pump during exercise recovery without the concomitant participation of central command. Nobrega and Araujo (15) observed that no muscle contraction was observed by electromyography (EMG) during passive cycling exercise, indicating that central command was absent. The three recovery modes included one mode that did not engage the skeletal muscle pump (inactive, seated) and two that engaged the skeletal muscle pump (loadless and passive pedaling). Because loadless pedaling involves central command, the passive pedaling mode was performed to serve as a control for this central command effect. Ambient temperatures during the studies averaged 24 ± 1°C.

Hemodynamic measurements. Pulsed Doppler ultrasound was used to measure beat-to-beat stroke volume (SV) at the aortic root during rest, exercise, and recovery periods. Doppler-shifted waveforms were obtained with an L-shaped crystal transducer (crystal diameter = 1 cm) with a focal range of 2–8 cm (InterSpec XL, Conshohocken, PA; presently owned by ATL, Bothell, WA). It operated at 3.0 MHz, with a pulse-repetition frequency of 12.6 kHz. This allowed frequency shifts of 6.3 kHz and a maximum velocity detection of 96 cm/s at the assumed Doppler angle of zero. A 400-Hz high-pass filter was used to eliminate low-frequency noise caused by wall motion. Axial resolution (dB) was ~0.5 mm. The Doppler transducer was positioned in the suprasternal notch, and the ultrasound beam was directed inferiorly and posteriorly along the flow stream in the ascending aorta. A measurement of aortic diameter at the aortic root was taken from a two-dimensional parasternal long-axis view of the heart, with the subject in the supine or left lateral recumbent position before experimentation. SV was calculated from the measurement of aortic diameter and the flow velocity of the blood leaving the heart via the aortic root by using the following formula: SV = flow velocity (cm/s) × aortic diameter (cm).

Measurements of SAP and DAP were performed noninvasively by using a pneumatic finger cuff (Finapres blood pressure monitor, Ohmeda) based on the method of Penaz (18). MAP was calculated as DAP plus one-third pulse pressure (SAP – DAP). CO was calculated as SV × HR, and TPR was calculated as MAP/CO. A tetrapolar Minnesota impedance cardiograph model 304A (Surcom, Minneapolis, MN) was used to measure changes in thoracic impedance (TI). This measurement involved placement of four Mylar band electrodes circumferentially around the neck and thorax, application of alternating current to the outer two electrodes, and measurement of impedance between the inner two electrodes. Decreases in thoracic blood volume are associated with increases in basal TI (5). Therefore, we used changes in TI as an indirect measure of changes in central blood volume.

Data analyses. Comparisons of responses during the different exercise recovery modes were performed with repeated-measures analyses of variance. The main factors were exercise recovery mode and time. When significant main effects were observed, a post hoc analysis was performed by using Student-Newman-Keuls multiple-comparisons test. Statistical significance was set at an α level of 0.05. All data are presented as means ± SE.

RESULTS

No significant differences existed between baseline conditions before either active, passive, or inactive recovery for HR, SV, TPR, TI, and CO (P < 0.05). Exercise intensity was set at ~60% of each subject’s predicted maximal HR. The mean HR at peak exercise was 121 ± 2 beats/min. There were no differences in HR, SV, CO, or TI at peak exercise among exercise bouts followed by active, passive, or inactive recovery (Table 1). At peak exercise before active recovery, MAP was slightly less (5–7%) than MAP at peak exercise before inactive recovery, but exercise increased MAP similarly before the three recovery conditions.

Responses of MAP, HR, CO, and TI were not different between the two inactive recovery sessions performed on different days (P ≥ 0.89). Therefore, these responses were averaged and used for comparisons with responses to the loadless pedaling and passive pedaling recovery modes.

During inactive recovery from submaximal upright exercise, MAP showed a marked decrease during the

Table 1. Hemodynamic data for resting baseline and peak exercise

<table>
<thead>
<tr>
<th></th>
<th>Inactive Baseline</th>
<th>Inactive Peak</th>
<th>Active Baseline</th>
<th>Active Peak</th>
<th>Passive Baseline</th>
<th>Passive Peak</th>
</tr>
</thead>
<tbody>
<tr>
<td>HR, beats/min</td>
<td>75 ± 3</td>
<td>121 ± 2</td>
<td>78 ± 3</td>
<td>120 ± 2</td>
<td>78 ± 7</td>
<td>121 ± 2</td>
</tr>
<tr>
<td>SV, ml</td>
<td>56 ± 10</td>
<td>80 ± 6</td>
<td>53 ± 4</td>
<td>81 ± 11</td>
<td>54 ± 7</td>
<td>75 ± 10</td>
</tr>
<tr>
<td>CO, l/min</td>
<td>4.6 ± 0.4</td>
<td>9.7 ± 1.4</td>
<td>4.0 ± 0.2</td>
<td>9.6 ± 1.3</td>
<td>4.0 ± 0.4</td>
<td>8.9 ± 1.1</td>
</tr>
<tr>
<td>MAP, mmHg</td>
<td>98 ± 4</td>
<td>120 ± 3</td>
<td>93 ± 4</td>
<td>112 ± 4*</td>
<td>96 ± 3</td>
<td>118 ± 3</td>
</tr>
<tr>
<td>TPR, mmHg·l⁻¹·min</td>
<td>23 ± 2</td>
<td>14 ± 2</td>
<td>24 ± 2</td>
<td>13 ± 2</td>
<td>25 ± 2</td>
<td>13 ± 1</td>
</tr>
</tbody>
</table>

Values are means ± SE for data from 12 subjects. HR, heart rate; SV, stroke volume; CO, cardiac output; MAP, mean arterial pressure; TPR, total peripheral resistance. *Significant difference between active peak and inactive peak (P < 0.05).
first minute, followed by a gradual decline to preexercise values. Furthermore, MAP during the active and passive recoveries showed mild decreases during the first minute, followed by modest further decreases during the 5 min of recovery. No significant differences existed between active and passive recovery MAP responses (Fig. 1). Significant differences in the reduction of MAP existed between inactive and pedaling (loadless and passive) recoveries. The decreases in MAP from peak exercise to 15 s of recovery were significantly less during the active loadless recovery (−3 ± 1 mmHg) and passive recovery (−6 ± 1 mmHg) than during inactive recovery (−18 ± 2 mmHg), as illustrated in Fig. 2. These data also illustrate that the decreases in MAP from peak exercise to the end of the first minute of recovery were significantly less during the active loadless (−6 ± 1 mmHg) and passive recovery (−7 ± 1 mmHg) than during the inactive recovery mode (−17 ± 2 mmHg).

Figure 2 demonstrates the responses of TI as an inverse index of central blood volume and MAP during the first minute of recovery from exercise. The initial decrease in MAP occurs abruptly during inactive recovery and more gradually during passive or active recovery. During inactive recovery, TI increased, whereas during the active loadless and passive pedaling recovery modes TI did not change, relative to peak exercise values. TI plateaued after the first minute in all conditions.

The changes in HR during each recovery mode are illustrated in Fig. 3. These data illustrate that during all recovery modes, the greatest decreases in HR occurred during the first minute of recovery, after which a gradual decrease was observed. HR decreased significantly less during active recovery (−20 ± 4 beats/min) than during the inactive (−32 ± 4 beats/min) and the passive (−31 ± 4 beats/min) recovery modes by the end of the first minute of recovery. Throughout the 5 min of recovery from exercise, HR decreased less on average during the active loadless recovery than during the passive pedaling and the inactive seated recovery modes.

The changes in CO during exercise recovery, the decreases in CO in inactive conditions (−4.47 ± 0.23 l/min) were significantly greater than the passive (−2.55 ± 0.46 l/min) and active (−2.23 ± 0.26 l/min) recovery modes (Fig. 4). SV decreased less during the active and passive pedaling recovery modes than during inactive recovery. The net decrease in SV after 5 min of active recovery (−14 ± 1 ml) was significantly less than the decrease in SV during the inactive (−26 ± 2 ml) or passive (−18 ± 1 ml) recovery modes (Fig. 4).

TPR increased during the inactive recovery mode to baseline TPR levels during the first minute of recovery from exercise (Fig. 5). TPR increased substantially less during the active and passive recovery modes, and a trend appeared for passive TPR responses to fall between inactive and active responses over the 5-min
DISCUSSION

The results of this study show that the initial decrease in MAP during inactive recovery from moderate exercise is primarily due to cessation of skeletal muscle pumping. This conclusion is supported by the following observations: 1) MAP was better maintained during pedaling (loadless or passive) recovery, in which the exercise stimulus was minimal, but dynamic skeletal muscle pumping continued; 2) central blood volume was maintained near exercise levels during both pedaling recovery modes; and 3) the decrease in SV during pedaling recovery was modest compared with that during the inactive recovery. Thus maintenance of arterial pressure during loadless or passive pedaling was accomplished through maintenance of CO by the effect of skeletal muscle pumping to minimize the postexercise reduction in venous return and SV.

The role of the skeletal muscle pump during recovery from exercise. The skeletal muscle pump is an important mechanism in the maintenance of MAP and CO during dynamic exercise (14). The pumping facilitates the return of blood from the lower extremities back to the heart (23, 25). Rhythmic muscle contractions create intramuscular pressure oscillations (2), which facilitate venous blood flow to the heart during dynamic exercise (19, 26). Modest muscle contractions can effectively maintain central venous pressure and SV in the upright individual (22, 27). Therefore, the skeletal muscle pump contributes importantly to the maintenance of CO and, thus, oxygen delivery during upright dynamic exercise.

When dynamic exercise is stopped and inactive recovery occurs in upright posture, arterial pressure decreases rapidly in the first 1–2 min. Similarly, SV and CO decrease in parallel, with the majority of the decrease occurring in the first 2 min (8, 12). In contrast, systemic vascular resistance increases progressively toward preexercise levels. Our results are consistent with these findings and show that the hemodynamics of inactive recovery from exercise are similar, even when the exercise is mild and does not produce a substantial thermal load, as in our experimental conditions.

During inactive recovery from exercise, without the engagement of the skeletal muscle pump, we observed an increase in TI relative to both active exercise recovery modes. This is consistent with a relatively greater decrease in central blood volume during inactive recovery, since TI increases when central blood volume decreases (5). These results suggest that the combined effects of upright posture and the lack of muscle pump activity throughout inactive recovery
lead to decreases in venous return, presumably due to accumulation of blood in the leg veins.

What role does the skeletal muscle pump play in this response? We sought to determine whether the rapid decrease in MAP during the early phases of inactive recovery was due, in part, to the lack of the skeletal muscle pump. This was accomplished by comparing the MAP response during inactive vs. passive and loadless active recovery modes after a bout of submaximal exercise. With the active loadless pedaling recovery, central command may have contributed to the maintenance of arterial pressure. Therefore, it was necessary to include the passive recovery to eliminate the possibility of central command contributing to the hemodynamic responses. The rapid decrease in MAP was significantly attenuated with either loadless or passive pedaling following the exercise bout, relative to inactive recovery. Therefore, our data indicate that lack of muscle pumping is a major determinant in the rapid decreases in CO and MAP during inactive recovery from exercise. The maintenance of MAP by skeletal muscle pumping was accomplished by minimizing the decreases in venous return (assessed by TI), SV, and CO. Moreover, this was accomplished despite a persistent vasodilatation.

Role of central command during recovery from exercise. Another possible mechanism for the maintenance of MAP during active recovery from exercise may be the central command. Central command acts as a feedforward neural controller of the circulation during exercise to increase CO (21, 29). At the onset of exercise, the increase in HR is caused by vagal withdrawal, which results from the central command signals (6, 15, 16). Therefore, cessation of central command at the onset of exercise recovery hypothetically contributes to the postexercise reduction of CO and arterial pressure. Previous studies suggest that HR is a linear measure of central command during exercise (23). During inactive and passive pedaling recovery conditions following submaximal exercise, the decrease in HR was greater than during active recovery from exercise, indicating that central command was contributing to the maintenance of HR during active recovery. However, this HR effect did not translate into significant differences in CO or MAP. In addition, we found that passive pedaling maintained CO and MAP essentially the same as active loadless pedaling during recovery. These observations suggest that MAP and CO were being supported solely by the skeletal muscle pump during passive pedaling conditions, because the HR during this recovery condition equaled values seen during inactive recovery, in which central command was near resting values. Furthermore, Nobrega and Araujo (15) found that EMG, oxygen consumption, and perceived exertion were higher during loadless pedaling than during passive pedaling. They further noted that CO increased during both modes of exercise: during active exercise, it resulted from a HR increase with no change in SV, whereas passive exercise increased SV with no change in HR (15, 16). These data support our finding that the maintenance of CO and SV during passive pedaling recovery from exercise was due to the muscle pump maintaining central blood volume.

Our results indicate that TPR increases immediately after submaximal exercise, regardless of the recovery mode (active, passive, or inactive). During inactive recovery, there was a relatively greater vasoconstrictor response. Despite this vasoconstrictor response, the decreased CO led to substantially decreased arterial pressure from peak exercise values. During inactive recovery, MAP decreased to preexercise baseline levels within 2 min postexercise, whereas passive and active recovery MAP remained above preexercise levels. Passive cycling produced postexercise TPR elevation that was intermediate between inactive and active recovery. However, this observation was statistically inconsistent, and, therefore, any conclusions regarding control of vascular resistance would be equivocal.

Other potential factors that may be involved in the regulation of arterial pressure during recovery from exercise are mechanoreflexes, chemoreflexes, and arterial baroreflex resetting. Such resetting may, in turn, contribute to or modulate the postexercise reduction of CO and MAP. These possibilities remain to be investigated.
Assumptions and limitations of this work. Our use of Doppler ultrasonography for measurement of CO required the assumption that the diameter of the aorta did not change during and after exercise (7). In support of this assumption, aortic diameters of six subjects were measured at peak exercise and during recovery, and no significant differences were observed. Recent data from Hadjis and co-workers (7) indicate that Doppler-derived aortic blood flow (CO) measurements and the Fick indirect carbon dioxide rebreathing CO method correlate well. Our interpretations assume that loadless and passive pedaling produce similar leg muscle pumping (intramuscular pressure oscillations). Had a substantial difference in muscle pumping existed between loadless and passive pedaling conditions, we would have expected differences in postexercise TI or arterial pressure responses between the two conditions, yet none were seen. We did not use EMG to confirm lack of leg muscle activity during passive pedaling. This gives us confidence that no central command signals existed during passive pedaling.

Previous studies suggest that short-duration exercise at moderate workloads for <5 min does not cause thermoregulatory reflex-mediated responses such as cutaneous vasodilation and sweating (10). Therefore, our workload of 60% maximal HR for 3 min probably elicited minimal cutaneous vasodilation and sweating, although we did not measure those variables. Our conclusions do not necessarily extrapolate to recovery from longer, more intense exercise.

We speculate that the skeletal muscle pump may be even more important for blood pressure maintenance after more intense or prolonged work. Postexertional orthostatic hypotension is characterized by precipitous decreases in systolic and MAP, venous return, and left ventricular filling pressure (9, 11). MAP may decrease excessively, due to the ongoing circulatory demands of exercise-induced heat accumulation and metabolic debt (11). The resulting hypotension can lead to dizziness and, ultimately, syncope or fainting (3, 28). Therefore, an important benefit of an active recovery may be a reduced risk of syncope, presumably by engagement of the skeletal muscle pump.

For the present study, our rationale focused on the possible consequences of acute postexertional hypotension in normotensive individuals, such as risk of syncope. Other investigators have studied the potential benefit of chronic postexercise hypotension as a therapeutic approach to reduce MAP in hypertensive individuals. We did not study hypertensive individuals, nor did we focus on effects beyond the first few minutes of recovery; therefore, we cannot speculate on the potential benefits of our results in hypertensive individuals.

Conclusions. In summary, this study suggests that the skeletal muscle pump is important in the maintenance of MAP during recovery from submaximal exercise. Central command appears to be relatively less important in these hemodynamic responses. Although during the active loadless recovery HR decreased less than during the passive pedaling and inactive seated recovery, it is predominately SV, and not HR, that contributes to the maintenance of CO and MAP. In conclusion, these data suggest that the maintenance of blood pressure and CO during active recovery was primarily mediated by the actions of the skeletal muscle pump.

We acknowledge Nicolette Muenzer for technical support and our subjects for their participation. We thank Dr. Jere Mitchell for the use of the tandem bicycle.

This study was supported, in part, by National Heart, Lung, and Blood Institute Grant HL-49266.

Address for reprint requests: R. Carter III, Dept. of Integrative Physiology, Univ. of North Texas Health Science Center, 3500 Camp Bowie Blvd., Fort Worth, TX 76107 (E-mail: rcarter@hsc.unt.edu).

Received 4J January 1999; accepted in final form 18J une 1999.

REFERENCES