Time course of blood pressure changes immediately after maximal exercise

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Aim. The aim of this study was to investigate the effect of exhaustive exercise on the time course of arterial blood pressure (BP) and heart rate (HR) during upright resting (inactive) and loadless pedaling (active) recovery from a bicycle exercise to exhaustion.

Methods. The subjects were 11 healthy normotensive males. Systolic, diastolic and mean BP, and HR were recorded every 20 s for the initial 6 min of the recovery period.

Results. The time course of all BP measures during inactive and active recovery was characterized by a marked and sudden drop during the initial 20-s period, followed by a quick rise. This was followed by a gradual decline till the end of the recovery period. The time course of HR recovery, on the other hand, exhibited a smooth decline without the initial drop. With active recovery, the initial drop of diastolic and mean BP was less than the inactive recovery. After the 20-s period, the diastolic BP and HR were kept slightly higher with the active recovery than the inactive recovery.

Conclusions. A sudden drop of the BP occurred at the initial recovery period of postcycle exercise to exhaustion even though HR did not show such a change. The initial BP drop could be attenuated by the actively pedaling the cycle without load.

Key words: Blood pressure - Heart rate - Exhaustive exercise - Inactive recovery - Active recovery.

Although much has been studied regarding the systemic and regional hemodynamic responses to dynamic exercise, less attention has been directed toward understanding these responses at the conclusion of the recovery period of exercise. On the other hand, a postexercise hypotension and syncope after competitive exercise and maximal exercise in the upright exercise have frequently been reported. 1-3 To diminish the risk of syncope or hypotension after strenuous exercise, therefore, the use of active or supine recovery is recommended. 4-6 This postexercise period after performing strenuous exercise may be of extreme importance considering the fact that there are rapid and large cardiovascular changes which may lead to syncope, hypotension or arrhythmias. 7-9 Carter et al. used mean BP measured for every minute to compare three recovery methods used for 5 min following a cycle-exercise at 60% of maximal HR. The three methods were inactive seated rest, active pedaling against 0-W resistance, and passive cycling on a tandem cycle. They found that declines in mean BP, stroke volume and cardiac output were slower with the active and passive methods than the inactive rest. In addition, the decline in HR was also attenuated during the active pedaling more than the inactive and passive methods. 4

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When exercise severity approaches maximal levels, the chemo-reflex can become much more powerful due to accumulated muscle metabolite concentration, especially lactic acid. Muscle vasodilation also resulted in depressed systemic vascular resistance despite any compensatory vasoconstriction of splanchnic and renal vasculature. Crisafulli et al. also studied the difference in mean BP, HR, stroke volume and cardiac output between active pedaling at 40-W and inactive seated rest from a short duration of supramaximal exercise, which consisted of pedaling against a resistance equivalent to 150% of maximal workload. They reported that, although HR, stroke volume and cardiac output were lower during passive recovery than during active recovery, no difference was found between mean BP between the two recovery modes.\(^\text{3,4}\) In the effect of active leg movement following exercise, however, limited information is available since most of the recovery studies have observed on the various exercise intensity, duration and body position.\(^\text{3,4,6}\) Therefore, to our best knowledge, little is known about the details of time course of BP and the effect of active recovery movement immediately after the exertional exercise. The aim of this study was to investigate the effect of exhaustive exercise on the time course of BP and HR during inactive and active recovery from a bicycle exercise to exhaustion.

Materials and methods

Eleven young males who enjoy sports activities such as soccer, baseball, volleyball and basketball few times a week volunteered to serve as subjects in this study. Their age ranged between 18 and 39 years (mean±SD: 23.2±6.4 years). Their VO\(_{2}\text{max}\) measured prior to the experiment ranged from 2.70 to 4.066 mL/min (mean±SD: 3.344±0.408 mL/min). The subjects were all normotensive individuals with free of antihypertensive treatment. The subjects gave their written consent to participate in the study. The experimental protocol and consent form were reviewed and approved by the Human Subjects Committee of Osaka University. The experimental set-up consisted of an indirect automatic manometer using auscultatory technique (model EB-300, Minato Med. Sci. Co., Osaka, Japan), a three-lead electrocardiogram (model DS-2150, Fukuda Denshi Co., Tokyo, Japan), an automatic breath-by-breath respiratory gas analyzing system (model AE-280S, Minato Med. Sci. Co.), and a bicycle ergometer (model 232CXL, Minato Med. Sci. Co.). The work rate of the ergometer was modifiable from 10 to 400 W within a 5% error margin, which was controlled by a computer (model PC9821-xa20, NEC co. Tokyo, Japan). The autonomic manometer was validated through the auscultatory method every time prior to the experiment.

The subjects were requested to refrain from exercise other than domestic duties including walking, bicycling and house keeping, alcohol and caffeine for 24 h before the day of the experiment. After explaining the experimental procedures to each subject, to accustom them to the stationary cycle exercise, each subject practiced for 3 min while the work-rate was set at 20 W. After an adequate resting period, the experiment was started. The subject first rested for 10 min in a seated position during which baseline measurements were made, and then cranked the ergometer at 20 W for 6 min which was followed by an incremental work rate of 10 W per minute until exhaustion. During this cycle exercise, the subjects pedaled at a constant rate of 60 rpm as paced by a metronome. The criteria to determine the exhaustion were to meet 1) an oxygen uptake (VO\(_2\)) plateau despite increasing exercise intensity (≤150 mL/min), 2) highest gas exchange ratio value during the final increment of the exercise test being equal to or greater than 1.1, 3) attainment of the age-predicted maximal HR, and 4) the subject was unable to maintain a pedaling rate of at least 50 rpm. The exhaustion was considered to have occurred only if the subjects met at least three of these criteria. All of the subjects participated in this experiment fulfilled these criteria. A 6-min recovery period was started while the subject remained in an upright seated position on the cycle. Two recovery modes were examined. One was the inactive mode in which the subjects rested while their legs fully relaxed, and in the other the subjects maintained cranking at 0-W. These recovery modes were selected in random order and were performed on alternate days. The ambient temperature ranged from 22 to 24 °C.

Systolic and diastolic BP were measured every 20 s. Mean BP was (systolic BP-diastolic BP)/3 + diastolic BP. HR was continuously monitored and stored in a computer.

The average HR every 20 s was then computed.

Statistical analysis

Student’s paired t-tests with P-value of 0.05 were performed for some of the group data comparisons.
Results

Their mean body height and weight were 177.5±4.9 cm and 69.1±8 kg, respectively. Mean values of cardiovascular measures at rest and maximal exercise, and maximal work rate are presented in Table I.

Figures 1A-D show the time course of averaged systolic, mean and diastolic BP, and HR values for all subjects during the 6 min of inactivity recovery and active recovery. In inactive recovery during the first 20 s period from the cessation of exercise, all BP measures fell rapidly from 212.9±12.4 mmHg (mean±SD) to 156.7±30.4 mmHg for the systolic BP, 124.6±8.9 mmHg to 98±11.6 mmHg for the mean BP, and 83.8±10.9 mmHg to 67.5±6.2 mmHg for the diastolic BP. The drops in BP thus amounted to 56.2±33.8 mmHg for the systolic, 26.6±13.2 mmHg for the mean, and 16.3±11.3 mmHg for the diastolic measures. The BP then quickly rose following 20 s. Between 60 and 360 s, the systolic and mean BP gradually fell, while the diastolic BP remained at a similar value. HR remained at a high level during the initial 20 s post-exercise period, and then decreased gradually for the subsequent 360 s period.

During active recovery, a clear drop in systolic and mean BP values during the first 20 s, as observed during inactive recovery, was also present. After the first 20 s period, as in the case of the inactive recovery, all BP measures and HR with the active recovery decreased gradually until around 360 s following the cessation of exercise.

The point-by-point difference between the active and inactive recovery modes was computed for BP and HR for each subject. The mean value of these differences for all subjects was then computed. The mean differences in all BP measures during the initial 20 s recovery period were positive values, indicating that the active recovery produced higher BP levels. After the 20 s period, the differences in systolic and mean BP were close to zero. The diastolic BP and HR were maintained to slight higher level. A statistically significant difference was found only for the mean and diastolic BPs for the initial 20 s data (Figure 2).

Discussion and conclusions

Characteristic changes in BP and HR after exhaustive exercise

The findings of the present study indicated that the time courses of systolic, mean and diastolic BP during 6 min of upright inactive recovery from a cycling exercise to exhaustion were characterized by two distinct phases. The first was a marked and sudden drop in all BP measures during the initial 20 s, followed by an abrupt increase. The second was a gradual decrease in systolic and mean BP during the subsequent period. The initial drop of the BP was contrasted with HR recovery, which declined relatively smoothly during the corresponding period.

The initial and fairly time-locked (<20 s) drop in the systolic and mean BPs amounted to 56 and 27 mmHg, respectively. The diastolic BP also dropped by 16 mmHg. Recently, Takahashi et al. also showed an apparent drop in the mean BP of around 30 mmHg immediately after the cessation of cycling exercise at 80% maximum oxygen uptake.6 Pokan et al. also reported that left ventricular dimension during first 15 s after maximal upright exercise was lower than resting value. Their data indicate that preload is markedly reduced due to a reduced venous return immediately after maximal exercise.10 It is known that skeletal muscles can receive up to 88% of systemic blood flow during exercise and changes in skeletal muscle blood flow at rest can profoundly affect systemic arterial pressure.11,12 The initial and marked drop in BP may thus be caused by extensive blood pooling with vasodi-
Figure 1.—Time course of systolic (A), mean (B) and diastolic (C) BPs, and HR (D) during the inactive recovery and active recovery. The mean values for the pre-exercise resting and exercise periods are also presented. *P<0.05.

lulation in the vascular beds of the leg muscles and internal organs with their vasodilation. During the inactive recovery with no muscle pumping action, therefore, blood pooling was more facilitated than during the active recovery, producing a greater drop in BP. The abrupt (<20 s) and the large increase in BP following the initial drop is most likely caused by reflex activity of the arterial and carotid baroreceptors, by which strong vasoconstriction can be initiated.13 While the enhancement of parasympathetic nerve activity and withdrawal of sympathetic nerve activity force a rapid decrease in arterial BP after the cessation of intense exercise, accumulation of metabolites within the active skeletal muscle, increased plasma nor-
Epinephrine, and exercise-induced heat accumulation keep sympathetic drive elevated for some time. The net effect is expected to be a slow decline in BP toward the resting level. As expected, we found a gradual decrease in systolic and mean BP, which lasted at least about 6 min after the cessation of exercise, and this was in agreement with the findings of Johnson et al. and Takahashi et al.

Several studies suggested that to function at the prevailing arterial BP for exercise, the arterial baroreflexes are reset and/or their sensitivity can be adjusted prior to and during exercise by the central command, and to some extent by cardiopulmonary afferents. At the end of exercise, then, the arterial baroreflexes should be reset to a lower operating point. The present findings may indicate that activation of the baroreflexes during recovery from a single bout of maximal exercise occurs at a level higher than that during the pre-exercise resting period, with the operating range returning gradually over the initial 6 min of the postexercise period.

**Effects of active recovery**

With the use of an indirect automatic manometer, we were able to compare the time course of changes in arterial BP and HR during active and inactive recovery following maximal exercise with a relatively small temporal resolution. Earlier, Carter et al. presented data describing temporal changes in BP, HR and stroke volume during active recovery with 0-W pedaling after 3 min of cycling at 60% maximal HR. Stroke volume was measured every minute for 5 min using a pulse Doppler ultrasound method. All of these variables, and subsequently computed cardiac output, were maintained at a higher level during the entire period of active recovery compared with the inactive recovery. Interestingly, during the recovery with passive cycling with the help of a tandem partner, HR recovered by a time course similar to that of the inactive mode, whereas recovery of other variables followed a similar decline path to that of active recovery. They thus concluded that the skeletal muscle pump, not the central command, is the most important mechanism in the attenuation of initial postexercise decreases in BP and cardiac output. Our results concerning the higher BP during the initial 20-s period of the active recovery agree with their findings. Active use of the leg muscles, therefore, alleviated the sudden drop in BP following the cessation of exercise. Almost no active-inactive difference in BP after 1 min, and HR throughout the entire period of this study, was most likely related to the higher intensity level and longer duration of the preceding exercise performed. It is reasonable to postulate that levels of blood lactate concentration, thermal load and plasma norepinephrine were near maximum at the end of the exhaustive exercise. This enhances the muscle chemoreflex, and thus sympathetic activity, by which HR and arte-
rrial BP are augmented. These appear to be strong enough to mask the positive effects of the muscle pump and central command with 0-W pedaling on BP and HR. Indeed, Bangsbo et al. reported that the effect of 10-W pedaling recovery on blood lactate-clearence after high-intensity exercise was minimal for the first 10-min of the recovery period. 18

References