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Differences in Femoral Artery Blood Velocity among Active, Inactive and Passive Recovery Modes Following Knee Extension and Flexion Exercise (膝の伸展と屈曲運動後の活動,非活動及び受動回復モードにおける大腿部大動脈血流速度の違い)

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Differences in Femoral Artery Blood Velocity among Active, Inactive and Passive Recovery Modes Following Knee Extension and Flexion Exercise

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ABSTRACT

The effect of the muscle pump on femoral artery mean blood flow velocity (MBV) was examined in five healthy men during 5 min of recovery following seated knee extension and flexion (KEF) exercise at 0.31±0.03% peak oxygen uptake ($\dot{V}O_{2peak}$) for 5 min. The recovery was evaluated separately during three different activity modes: complete rest (inactive recovery), passively induced KEF (passive recovery), or voluntary KEF (active recovery) at 0.13±0.02% $\dot{V}O_{2peak}$. The superficial MBV was measured beat–by–beat using a Doppler–ultrasound method. After exercise, the MBV temporarily increased within 5 sec followed by a rapid decrease and then a gradual decrease in all three recovery modes, but to different extents. The MBV at the 5th min was significantly higher in the active recovery mode, followed by the passive and inactive recovery mode, respectively. The change in total peripheral vascular resistance (TPR), calculated as the mean arterial blood pressure/cardiac output (CO), was completely opposite to the change in MBV for each recovery mode. Both CO and $\dot{V}O_2$ decreased rapidly during the first minute followed by a gradual decrease, separately reaching either the unloaded exercise levels or the pre–exercise resting levels. The mean CO and $\dot{V}O_2$ at the 5th min were significantly greater for active recovery than for passive and inactive recovery. However, neither CO nor $\dot{V}O_2$ significantly differed between passive and inactive recovery. These data suggest that MBV during recovery from exercise increases in response to passive KEF without extra oxygen expenditure when compared with inactive recovery. This phenomenon is likely mediated by decreased TPR.

INTRODUCTION

We have so far intensively studied that the cardiorespiratory regulations during recovery from

Key words: Heart rate, Stroke volume, Cardiac output, Muscle pump, Hemodynamics, Impedance cardiography

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exercise are mediated by a variety of factors, including distribution of blood flow, the balance of the autonomic nervous system, neural afferents originating from vascular mechanoreceptors, and central commands from the cerebral motor cortex[1–6]. Especially, in an upright position, the function of the muscle pump plays important roles not only in maintaining appropriate blood flow to post–exercise muscles, but also in efficiently promoting venous blood return to the heart[6]. However, when considering the complicated peripheral circulation during muscular activity, a database regarding post–exercise hemodynamics, such as the rate of blood flow to exercising muscles in humans, has been still limited, because of the inability to accurately and noninvasively measure blood flow velocity in the conduit vessels to working muscles without generating artifacts due to body motion.

Recently, a Doppler–ultrasound technique has been developed to noninvasively measure beat–by–beat blood flow velocity in the femoral artery during dynamic knee extensor and flexor exercise[7,8]. Apart from such leg exercise, however, this technique has been often utilized to estimate limb blood flow intermittently between muscle contractions or just after the termination of exercise[9,10]. Even though the Doppler–ultrasound method has offered resistance against motion artifacts, it may still be difficult to measure the blood flow velocity during large–muscle–mass exercise. In this study, in order to accomplish the continuous measurement of femoral artery blood velocity (FABV) during dynamic exercise using the Doppler–ultrasound method, we introduced a newly developed system, with which the subjects performed a two–legged dynamic knee extension and flexion exercise. The both legs movement of knee extension and flexion was transformed to the mode of pedaling using a set of heel harnesses with aluminum rods connected to the pedals of a cycle. Inversely, the subject's legs were moved passively through the link motion by the experimenter pedaling on the cycle ergometer. The knee extension and flexion exercise enabled us to continuously measure the FABV without serious artifacts of Doppler–signal errors.

Thus, the present study was designed to assess how both active and passive leg movements influence changes in FABV during recovery from dynamic knee extension and flexion exercise in an upright seated position compared to a quiet resting recovery. Even though leg movement in the recovery period following exercise was performed at a very light intensity, an increase in stroke volume with increasing venous return occurred with the assistance of the muscle pump[2,6]. Thus, we hypothesized that FABV during post–exercise recovery would be increased by passive leg movement as well as active leg movement. The knowledge of the difference in hemodynamic responses to passive recovery and to inactive recovery would be important for the understanding of noninvasive human experiments regarding the cardiorespiratory control mechanisms, in which passive muscular contraction has been used to effectively isolate peripheral neurogenic inputs[1,11]. To the best of our knowledge, there have been no studies that have compared the effects of inactive, active and passive recovery modes following exercise on blood velocity in the femoral artery that are directly relevant to the exercising skeletal muscles. It is clinically and scientifically important to know how the three different modes of post–exercise activity affect muscle blood flow velocity during the period of recovery after exercise, because homeostatic changes in the cardiovascular system are not inevitable after exercise or physical activity in daily life and do not always reverse the same process as those after the onset of exercise. Furthermore, information of hemodynamics, blood flow forces, including wall shear stress to the lumen of vessels, has provided insight into the regulation of blood vessel tone or caliber underlying salutary or vicious effects of exercise hyperemia on both the circulation and

vasculatures[12].

I METHODS

1 Subjects

Five healthy young male volunteers participated in the present study. The physical characteristics of the subjects were: mean age 22.2 ± 0.1 years (mean ± SE), with an average height of 1.69 ± 0.02 m, average body mass of 59.2 ± 0.7 kg, and average peak oxygen uptake ($\dot{V}O_{2peak}$) of 2.55 ± 0.11 L・min^{-1}. All subjects were fully informed of the procedures, risks and benefits of the study, and written consent was obtained from all subjects before the study. None of the subjects had a history of circulatory or respiratory disease. Furthermore, none of the subjects were engaged in any regular physical activity or were involved in competitive sports. The $\dot{V}O_{2peak}$ was determined for each subject using a 15 W・min^{-1} incremental ramp–exercise test on an electromagnetically–braked cycle ergometer (Corival 300; Lode, The Netherlands) until the subject was exhausted[2].

2 Measurement of cardiorespiratory variables

$\dot{V}O_2$ was measured breath–by–breath using an on–line automated system[1]. Ventilatory air flow was monitored with a hot–wire–type pneumotachograph (RF–2; Minato, Osaka, Japan). The composition of the expired gas was continuously analyzed using a medical mass spectrometer (WSMR–1400, Westron, Chiba, Japan) that was calibrated with a standard reference gas mixture before each measurement.

Measurements of heart rate (HR), stroke volume (SV) and cardiac output (CO) were obtained using an automated measurement system[2]. Briefly, CO was determined by impedance cardiography (ICG) using a standard constant–current–type ICG unit (RGA–5; Nihon Koden, Tokyo, Japan) with two silver tape electrodes placed on the neck and another two electrodes placed on the torso. The SV was calculated using the standard equation of Kubicek et al[13]. The effects of respiration and body motion artifacts on the impedance signal were eliminated by averaging the ICG waveforms over ten cardiac cycles[14]. Although the absolute values of impedance–derived SV remain to be characterized, there has been general agreement that ICG is suitable for estimating relative changes in SV from rest to exercise and recovery. In particular, to ensure the reproducibility of intra–individual estimates of SV in response to exercise with the least variability of measurements obtained from day to day, special care was taken to maintain the electrode position of the ICG, as the measured SV is known to be a function of the electrode distance[15]. Arterial blood pressure was measured non–invasively using a pneumatic finger cuff (2300 Finapres Blood Pressure Monitor, Ohmeda, NEC San–ei Ltd., Tokyo, Japan). Mean arterial blood pressure (MAP) was calculated as diastolic arterial pressure plus one–third of the pulse pressure. The arm and hand supported by an arm rest of a chair were held at heart level to avoid variations of blood pressure due to changes in hydrostatic pressure. Total peripheral vascular resistance (TPR) was calculated as MAP/CO. All data for respiratory and circulatory variables were stored on diskettes for subsequent analysis using a personal computer (PC–9821Xa13; NEC, Tokyo, Japan).

Femoral artery blood flow velocity (FABV) was continuously measured using a pulsed Doppler ultrasound velocitymeter (DVM–4000; Nihon–Koden Co., Tokyo, Japan). A flat transducer with an operating frequency of 5 MHz was fixed to the skin over the superficial branch after the bifurcation of the common femoral artery distal to the inguinal ligament. The position was chosen to minimize

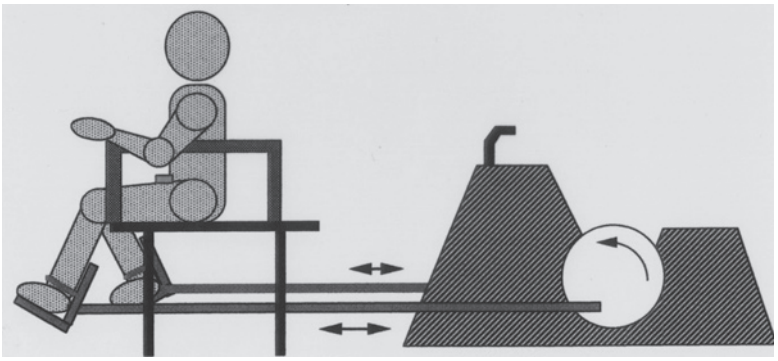

Fig. 1 Diagram of experimental setup with subject seated in a chair in front of a cycle ergometer

interference of blood flow by leg exercise. The angle of the transducer of the piezoelectric ceramic relative to the skin was approximately 60 degrees, and the ultrasound gate was maintained at full width to facilitate insonication of the total width of the artery with approximately constant intensity. Optimal positioning of the Doppler equipment was verified by observing the velocity spectra on a PC monitor, resulting in clear Doppler signals at rest and during exercise and recovery. The velocity signal was recorded at 500 Hz on a computer system along with the electrocardiograph to allow beat-by-beat analysis of the data. The beat-by-beat mean blood flow velocity (MBV) was calculated by integrating the total area under the instantaneous FABV profile, with the marked QRS complex of the electrocardiogram signaling the end of one heartbeat and the beginning of the next. The FABV, MBV and R-R interval data were recorded sequentially on a personal computer (PC-9801FS; NEC, Tokyo, Japan) via an RS232C interface. Unfortunately, the volumetric flow rate of muscle blood could not be obtained, as the Doppler equipment was unable to measure the caliber of the femoral artery or the cross-sectional area of the target vessel.

3 A novel system

Dynamic exercise with knee extensor and flexor muscles was performed at a rate of 60 times/min (60 rpm), using the experimental set-up illustrated in **Fig. 1**. Briefly, the subjects were instructed to sit in a chair in front of the cycle ergometer. The chair was set at an appropriate high to allow the subject's legs to move freely. The trunk was vertical, and both thighs were horizontal. The chair was adjusted so that both the legs were in a neutral vertical position (i.e., the knee-joint angle between the thigh and leg was approximately 90 degrees). The movement of knee extension and flexion was transformed to the mode of pedaling using a set of heel harnesses with aluminum rods connected to the pedals of the cycle. The harness and rod for each leg weighed a total of 2 kg; in practical usage, however, the weight loaded on the subject's leg was reduced by about 1 kg to balance the support of the pedal of cycle ergometer. The forward and backward movements of the legs were conducted within a space 15 degrees from the vertical line, and the two legs moved inversely from one another.

Without any effort on the part of the subject, passive leg exercise was induced by the experimenter, who performed pedaling behind the cycle ergometer. The degree of knee joint motion was adjusted to be the same as that achieved during the voluntary exercise, and the knee extend-flex movements were performed smoothly and continuously in a manner analogous to stationary cycling.

A leg-movement frequency of 60 rpm was maintained throughout exercise with the aid of an audible metronome, and was monitored using an inductive revolution meter fitted to the ergometer. Each subject was allowed a practice period to become accustomed to knee extension and flexion exercise on separate days about one week prior to commencement of the formal study.

4 Experimental protocol

The subjects rested for 2 min in the upright seated position in the chair (**Fig. 1**) in order to obtain baseline cardiorespiratory data. The subject then performed knee extension and flexion exercise at a work rate of 0 W for 3 min of a warm-up period and then at a work rate of 60 W for 5 min. The recovery phase during a 5-min period after the exercise was studied in each of the following three different modes: 1) complete seated rest with no leg movement (inactive recovery), 2) passively induced knee extend-flex movements (passive recovery), or 3) voluntary knee extend-flex movements at 0 W (active recovery). The subjects were in the sitting position throughout the experiments. Subjects were informed that the test could consist of either no leg movement, passive leg movement or voluntary leg movement during recovery following exercise, and that the order of the trials would be randomized. The absolute intensities of 0 W and 60 W were equivalent to the relative intensities of $0.13 \pm 0.02\%$ and $0.31 \pm 0.03\%$ $\dot{V}O_{2peak}$, respectively. The changes in the work rate were automatically controlled by a computer program.

Tests were initiated at approximately 10:00 A.M. in all subjects, and the order of testing (e.g., inactive, active or passive recovery) was randomly assigned. Each subject completed two repetitions of the test for each of the three experiments, and each trial was performed only once a week to eliminate the influence of exercise training. Subjects were instructed to abstain from any beverages containing caffeine or alcohol beginning at 9:00 P.M. on the night before exercise trials and to abstain from vigorous exercise for the 36 h preceding the exercise trial. Subjects were allowed a small breakfast no later than 7:00 A.M. on the test day. All experiments were conducted at an ambient temperature of 21-22°C .

5 Data analysis

All breath-by-breath data and beat-by-beat data from two repetitions under each test condition for each subject were arranged separately with a 5-s interval time base using a Lagrange interpolation. These data were then averaged to yield a single data set for each subject. Group mean values were obtained from the five individual averaged data sets. Average values for all variables were determined from the last minute of averaged data at rest before exercise, during exercise and during recovery.

6 Statistical analyses

The differences in mean values for each variable at the 5th min of recovery among the three recovery conditions were tested with the Friedman repeated measures analysis of variance on rank and a post-hoc Wilcoxon's signed-ranks test (two-sided). The changes in average values for each variable in the transition during the first-minute period of recovery were evaluated using one way repeated-measures analysis of variance, and the peak of MBV and the bottom of TPR during the early phase of recovery was analyzed with Dunnett's multiple comparison test. A least-squares regression line was fitted to the mean MBV plots for the first run versus those for the second run using the individual average data together at rest and during exercise at both 0 W and 60 W. The correlation between the mean MBV for the two runs was statistically examined using the Pearson correlation

Table 1 Cardiorespiratory variables before, during and after dynamic knee extension and flextion exercise

	Rest	Exercise	Recovery
VO_2 (L・min^{-1})			
Inactive	0.23 ± 0.02	0.78 ± 0.06	0.22 ± 0.01
Passive	0.21 ± 0.03	0.77 ± 0.06	0.23 ± 0.01
Active	0.25 ± 0.01	0.84 ± 0.13	0.31 ± 0.02 * †
MBV (cm・sec^{-1})			
Inactive	4.5 ± 0.6	13.1 ± 0.5	5.0 ± 0.6
Passive	4.2 ± 0.5	14.3 ± 0.7	7.7 ± 0.5 *
Active	4.7 ± 0.5	15.8 ± 1.4	9.7 ± 1.1 * †
CO (L・min^{-1})			
Inactive	5.1 ± 0.4	9.3 ± 0.6	6.1 ± 0.3
Passive	5.3 ± 0.2	9.5 ± 0.5	6.5 ± 0.3
Active	5.3 ± 0.3	9.4 ± 0.4	7.2 ± 0.4 * †
HR (beats・min^{-1})			
Inactive	68.5 ± 5.1	98.7 ± 7.6	68.1 ± 5.6
Passive	67.2 ± 4.3	96.4 ± 6.3	67.8 ± 5.2
Active	64.4 ± 4.9	96.5 ± 7.5	73.5 ± 6.9 * †
SV (mL)			
Inactive	74.6 ± 5.4	94.4 ± 3.6	91.0 ± 6.6
Passive	80.1 ± 6.3	99.1 ± 3.8	96.8 ± 4.5
Active	82.7 ± 3.4	98.8 ± 6.3	99.9 ± 4.4
MAP (mmHg)			
Inactive	86.8 ± 3.1	100.7 ± 3.4	83.0 ± 3.4
Passive	88.0 ± 3.4	96.2 ± 3.4	82.6 ± 2.3
Active	84.6 ± 2.3	99.9 ± 4.7	85.5 ± 2.6
TPR (mmHg・L^{-1}・min)			
Inactive	17.9 ± 1.8	11.3 ± 1.0	13.9 ± 0.8
Passive	16.9 ± 1.2	10.5 ± 0.9	13.0 ± 0.8 *
Active	16.5 ± 1.5	11.0 ± 0.9	12.1 ± 0.9 * †

* $p < 0.05$, compared to inactive recovery; † $p < 0.05$, compared to passive recovery
Values are mean ± SE for 10 trials of five subjects.
VO_2: oxygen uptake, MBV: mean femoral artery blood velocity, CO: cardiac output, HR: heart rate, SV: stroke volume, MAP: mean arterial blood pressure, TPR: total peripheral vascular resistance. Inactive: inactive recovery experiment, Passive: passive recovery experiment, Active: active recovery experiment.
Values for rest, exercise and recovery were obtained at the 2nd min of rest, the 5th min of exercise, and the 5th min of recovery, respectively.

coefficient. The differences were considered significant for all statistical analyses at $p < 0.05$. All values are expressed as means ± SE.

Ⅱ RESULTS

Table 1 shows the average values for all variables obtained from the five subjects at rest before exercise and during steady-state exercise at 60 W, corresponding to a relative intensity of 0.31 ± 0.03% $\dot{V}O_{2peak}$, and those during the three different recovery modes. No significant differences in the resting or exercise values were detected when comparing the three experiments.

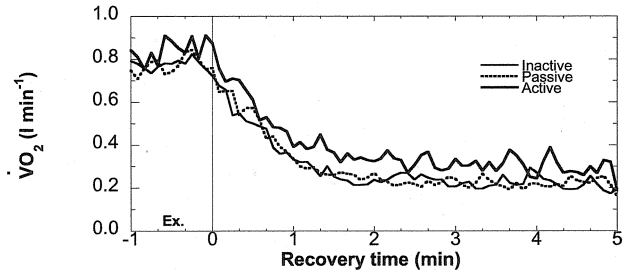

Fig. 2 Oxygen uptake ($\dot{V}O_2$) during recovery after two-legged knee extension and flexion exercise (Ex.) at 60 W with complete rest (Inactive, thin line), passive leg movement (Passive, dashed line) and active leg movement at 0 W (Active, thick line)

Fig. 2 shows the time courses of $\dot{V}O_2$ during inactive, passive and active recovery modes. $\dot{V}O_2$ for all three recovery modes decreased rapidly during the first minute and declined slowly thereafter. The mean $\dot{V}O_2$ at the 5th min was significantly ($p < 0.05$) greater during active recovery when compared with inactive and passive recovery modes, whereas the later two were not significantly different from each other.

Fig. 3 shows the dynamic profiles of MBV, CO, MAP and TPR obtained during recovery with the three different modes. Immediately after exercise, MBV showed an initial rapid increase (58%), regardless of the recovery mode, and then a gradual decrease, but to different extents. The peak values of MBV at 5 sec after the end of exercise were significantly greater than the steady-state values during exercise (for inactive, 21.1±2.4 vs. 13.1±0.8 cm・sec^{-1}; for passive 23.7±2.6 vs. 14.3±1.2 cm・sec^{-1}; for active, 21.9±1.7 vs. 15.8±1.3 cm・sec^{-1}; each $p < 0.05$). The mean MBV at the 5th min of recovery was significantly ($p < 0.05$) greatest in the active recovery mode, followed by the passive recovery mode and then inactive recovery mode (**Table 1**).

Post-exercise CO in all recovery modes decreased gradually after an initial delay of about 20 sec. The mean CO at the 5th min was significantly ($p < 0.05$) greater for active recovery than for inactive and passive recovery, whereas there was no significant difference between the later two. The greater CO at the 5th min for active recovery was predominantly due to an increase in HR rather than an increase in SV (**Table 1**).

MAP decreased suddenly ($p < 0.05$) and subsequently remained relatively stable during the inactive and passive recovery modes. Similarly, MAP during active recovery showed a significant, but small, drop. However, there was no significant difference between the three recovery modes in the mean MAP at the 5th min.

TPR showed an initial rapid fall within the first 10 sec and a rapid rise in the next 20 sec ($p < 0.05$) followed by a gradual increase, regardless of the recovery mode. However, the mean TPR at the 5th min was significantly less during active recovery when compared with inactive and passive recovery modes, whereas the later two were not significantly different from each other.

The Doppler measurements of MBV at rest and during exercise at 0 W and 60 W were highly reproducible in the two exercise tests performed one week apart (**Fig. 4**). Pearson's correlation coefficient (r) and the p value for the correlation were $r = 0.984$ and $p < 0.001$, respectively.

Ⅲ DISCUSSION

After the end of exercise, MBV showed a transient increase, reaching the peak within 5 sec, followed by a rapid decrease and a gradual decrease thereafter in all three recovery modes. The peak

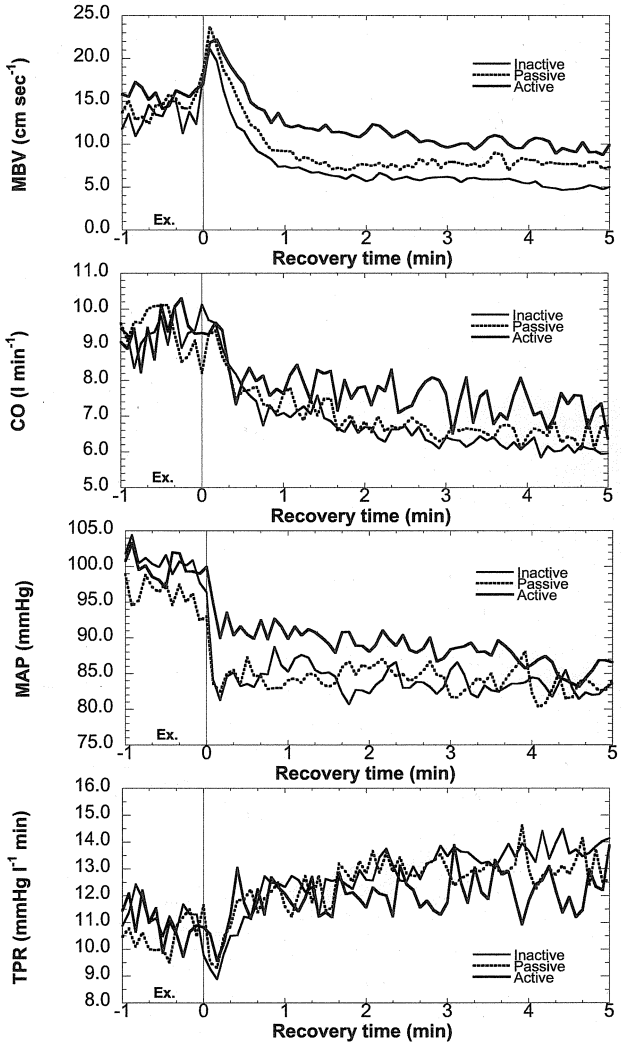

Fig. 3 Mean femoral artery blood velocity (MBV), cardiac output (CO), mean arterial blood pressure (MAP), and total peripheral vascular resistance (TPR) during recovery after two-legged knee extension and flexion exercise (Ex.) at 60 W with complete rest (Inactive, thin line), passive leg movement (Passive, dashed line), and active leg movement at 0 W (Active, thick line)

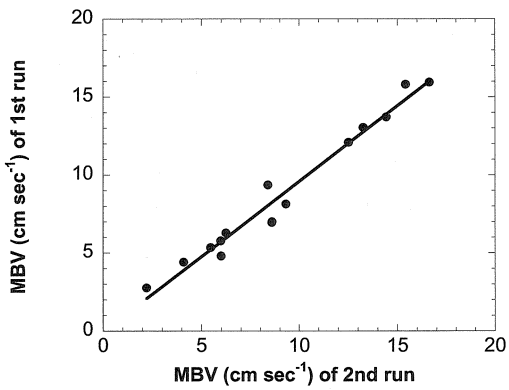

Fig. 4 Reproducibility of Doppler measurements of femoral artery blood velocity (MBV) at rest and during knee extension and flexion exercise both at 0 W and 60 W

Data were obtained from five subjects who performed two runs at an interval of one week. The equation of the regression line: $Y = -0.078 + 0.968X$ ($r = 0.984$, $n = 15$, $p < 0.001$).

MBV was similar among the three recovery modes. However, MBV at the 5th min was significantly greatest in the active recovery, followed by the passive recovery and then the inactive recovery. Conversely, TPR decreased temporarily and then increased slowly. The $\dot{V}O_2$ at the 5th min in passive and inactive recovery modes did not significantly differ, whereas each of them was significantly less than that during active recovery. These data support our hypothesis that FABV during recovery from exercise was increased by passive leg movement. Further, this phenomenon was likely related to a decrease in TPR without extra oxygen expenditure.

The rapid, initial increase in MBV seen immediately after exercise, regardless of the recovery mode, appeared to be independent of the changes in CO and MAP, since the initial increase of MBV occurred despite a sudden decrease in MAP or no increase in CO. In addition, the initial transient decrease in TPR and subsequent gradual increase were observed when intensive muscular contraction was terminated. These data suggest that blood flow to the exercising muscles is partly restricted by the mechanical compression of vasculatures due to intensive muscular contraction, and that the resulting blood flow is not sufficient to meet the metabolic demands of the muscle during intensive exercise.

MBV values during exercise in the present study (13–16 $cm \cdot sec^{-1}$) were approximately half of those reported in previous studies[8, 10, 16, 17]. There are several possible reasons for this discrepancy. First, there may be differences in the type of exercise, such as cycling, knee extension, calf muscle contraction, or differences in the size of the exercising muscles between the present study and previous studies. Second, FABV measurements were obtained during different time periods (e.g., between and after muscular contractions, or during prolonged muscle relaxation), at different sites (e.g., 2 cm distal from the inguinal ligament and before the bifurcation into the superficial and profound vessels vs. about 5–8 cm distal from the inguinal ligament), and with subjects in different positions (e.g., a supine or semi-supine position vs. an upright seated position). Preliminary experiments by our group failed to obtain an adequate Doppler signal when FABV measurements were performed 2 cm distal from the inguinal ligament, most likely secondary to the artifact associated with leg movement in the upright seated position. Thus, the maintenance of a supine or semi-supine posture would be required to successfully measure the blood flow velocity in the femoral artery in the proximity of the inguinal ligament during intensive dynamic leg exercise, although such a supine posture is not natural for usual physical activity.

Most experimental protocols are relegated to using intermittent static or dynamic contractions to prevent deterioration of Doppler-ultrasound signals due to body motion. Despite the difficulty of the Doppler method in the present study, the sensor remained optimally positioned at the center of the target vessel in sitting subjects performing leg extension and flexion movement. Further, the relative changes in MBV in inactive recovery above the resting baseline level were similar to those reported in subjects following heel lifting[16]. In addition, the mean values of MBV at rest, during exercise, and during inactive recovery in this study were consistent with results reported from subjects performing one-legged knee extension[7]. Finally, the correlation between MBV at rest and during exercise on separate days suggested that this is a highly reproducible method for measuring dynamic changes in FABV during and after dynamic knee extension and flexion exercise, as well as at rest.

Characterization of the femoral artery MVB allows determination of the Reynolds number (Re), wall shear rate ($\dot{\gamma}_w$), and wall shear stress (τ_w). The τ_w, the flow-oriented stress acting as the

viscous drag on the vascular endothelial surface, was given by the product of the blood viscosity (μ) and the shear rate of blood on the endothelial surface assuming a laminar, Newtonian flow through a cylindrical vessel:

$$\tau_w = \mu\ \dot{\gamma}_w = \mu\ \frac{8V}{D} = 0.032 \times 154 = 4.9\,(\mathrm{dyn \cdot cm^{-2}})$$

where the mean blood flow velocity $V = 15$ cm・sec^{-1}, superficial femoral artery diameter $D = 0.78$ cm, and blood viscosity $\mu = 3.2$ cP. In this calculation, vessel diameter and blood viscosity were assumed to be constant, since the diameter of the femoral artery before the bifurcation into the superficial and profound branches has not been changed[8,17]. Further, according to Fåhraeus and Rindqvist[18], the blood viscosity has been asymptotically increased as the vessel caliber increases to be constant above 1 mm in diameter[19]. The Re ($= \rho VD/\mu$; blood density $\rho = 1.00$ g・cm^{-1}) was estimated to be 293, indicating that flow was occurring in a laminar pattern[20]. The vascular morphology has been characterized by a diameter exponent linking the relationship of a mother branch (D_0) and its daughter branches (D_1, D_2):

$$D_0^{\,m} = D_1^{\,m} + D_2^{\,m}$$

where m is estimated to be 3.0 by the minimum work model[21] and the minimum volume model[22]. Uylings[23] argued that the diameter exponent should be dependent on whether laminar ($m = 3.0$) or turbulent flow ($m = 2.33$) is present. However, the diameters of the common femoral artery and its superficial branch have been reported to be 1.05 cm and 0.78 cm, respectively[8,17,24]. In this case, an exponent of 2.33 can be obtained from the above equation, assuming that the diameter of the profound branch is identical to that of the superficial branch. According to Uylings[23], the blood flow in the superficial branch after the bifurcation of the common femoral artery may be turbulent, despite the lower Re suggesting a laminar flow state. This discrepancy would be attributed to the flow pattern after the bifurcation, since a certain distance is required to re-establish the stability of blood flow after a junction[25].

Electromyogram recordings were not obtained in this study, making it difficult to confirm that no voluntary contractile effort was made during passive activity. However, there was no significant difference in $\dot{V}O_2$ when comparing the inactive and passive recovery modes, which does suggest the absence of voluntary contractile effort during passive recovery accomplished through the leg-pedal link system developed in this study. In addition, without the extra expenditure of energy for muscular contraction, the greater MBV was maintained by passive leg movements as compared to inactive recovery. Other investigators have suggested that increased muscle blood flow during active recovery following exercise facilitates restoration of the cardiovascular and respiratory systems through the removal or reuse of metabolic substances in active tissues and the circulation[2,26,27]. Accordingly, the increased muscle blood flow induced by passive leg movement may also facilitate this process efficiently with respect to lower energy expenditure (oxygen consumption). Further studies to explore the physiological significance of passive recovery would be of benefit.

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