Different blood flow responses to dynamic exercise between internal carotid and vertebral arteries in women

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Submitted 7 December 2009; accepted in final form 29 June 2010

Sato K, Sadamoto T. Different blood flow responses to dynamic exercise between internal carotid and vertebral arteries in women. J Appl Physiol 109: 864–869, 2010. First published July 1, 2010; doi:10.1152/japplphysiol.01359.2009.—The blood flow regulation in vertebral system during dynamic exercise in humans remains unclear. We examined the blood flow responses in both the internal carotid artery (QICA) and vertebral artery (QVA) simultaneously during graded dynamic exercise by Doppler ultrasound to evaluate whether cerebrovascular responses to exercise were similar. In the semisupine position, 10 young women performed a graded cycling exercise at three loads of 30, 50, and 70% of peak oxygen uptake (VO2peak) for 5 min for each workload. Mean arterial pressure, heart rate, and cardiac output increased progressively with three workloads (P < 0.01). The end-tidal partial pressure of CO2 (PetCO2) in the expired gas increased from the resting level (P < 0.01) at 30 and 50% VO2peak. The PetCO2 at 70% VO2peak (43.2 ± 1.6 Torr) was significantly lower than that at 50% VO2peak (45.3 ± 1.4 Torr). In parallel with the changes in PetCO2, QICA increased from resting level by 11.6 ± 1.5 and 18.4 ± 2.7% at 30 and 50% VO2peak (P < 0.01), respectively, and leveled off at 70% VO2peak. In contrast, QVA did not show a leveling off and increased proportionally with workload: 16.8 ± 3.1, 32.8 ± 3.6, and 39.5 ± 3.4% elevations at the three exercise loads, respectively (P < 0.01). With increasing exercise load, the cerebrovascular resistance in internal carotid artery increased (P < 0.01), while cerebrovascular resistance in vertebral artery remained stable during exercise. The different responses between QICA and QVA in the present study indicate a heterogenous blood flow and cerebrovascular control in the internal carotid and vertebral systems during dynamic exercise in humans.

internal carotid system; vertebral system; Doppler ultrasound; cerebrovascular autoregulation

THE HUMAN BRAIN RECEIVES ~750 ml/min of blood at rest, which represents close to ~15% of cardiac output (CO) (27). Blood supply to the brain originates from two major sources: the internal carotid arteries (ICA) forming the anterior cerebral circulation (ICA system) and the vertebral arteries (VA), which merge into the basilar artery and supply the posterior cerebral circulation (VA system). Although anastomosis between the major arteries via the anterior and posterior communicating arteries completes the Circle of Willis, the ICA system provides blood supply mainly to the cerebral, while the VA system supplies blood to the brain stem, cerebellum, and spinal cord. The relative contribution of the ICA system and VA system to global cerebral blood flow (gCBF) at rest are estimated to be ~75 and ~25%, respectively (32). The regulation of CBF during dynamic exercise has been studied almost exclusively in the ICA system in humans (11, 13, 14, 26, 28). However, there are no data blood flow responses in the VA system to dynamic exercise. Since simultaneous measurements of blood flow in ICA (QICA) and VA (QVA) systems have not yet been done, it remains unclear not only whether the blood flow responses during exercise may differ between ICA and VA systems, but also whether the relative contribution of ICA and VA systems to the gCBF may vary during exercise. We hypothesized that the cerebrovascular responses to dynamic exercise were different between ICA and VA systems and thereby produced different contributions of ICA and VA systems to the gCBF because of the following viewpoints. First, the proportion of blood flow in any single vessel to gCBF may not be constant during exercise, and flow redistribution between major cerebral vessels might occur (11, 19, 26). Second, previous animal studies reported that the cerebrovascular response was not uniformly distributed during dynamic exercise (4, 8). Third, some investigators have reported a difference in cerebral CO2 reactivity (3, 18, 35) and autoregulatory control (10, 21) between the anterior and posterior cerebral circulation. Finally, there are some reports of anatomic and physiological differences in the cerebral vasculature between ICA and VA systems in humans (6, 9).

To verify the aforementioned hypothesis, we compared the QICA and QVA during dynamic exercise. In addition, we estimated the relative contribution of the ICA and VA system to gCBF from the data of QICA and QVA obtained during dynamic exercise.

METHODS

Ten healthy young women [22 ± 2 yr (mean ± SD), 164 ± 6 cm, 58 ± 4 kg, and peak oxygen uptake (VO2peak): 38.3 ± 5.1 ml·kg−1·min−1] participated in this study. All procedures and protocols confirmed to Declaration of Helsinki and were approved by the Institutional Review Board at the Japan Women’s College of Physical Education. Following a detailed verbal explanation of the intended experimental measures and procedures, each subject gave informed, written consent before participation. The subjects were not performing endurance training on a regular basis. In addition, they were free of any known cardiovascular and pulmonary disorders and were not using prescribed or over-the-counter medications. Before the experiment, each subject visited the laboratory for familiarization with the CBF measurement by Doppler ultrasound and dynamic exercise protocol.

Aerobic power. The VO2peak was determined by an incremental protocol on a cycle ergometer (Aerobike 800, Combi) 2 wk before the experiments. Subjects were exposed to an initial work rate of 30 W at a pace of 60 cycles/min. The subjects were told to maintain the frequency of pedaling, and work rate was increased 10–15 W every minute until volitional exhaustion. Respiratory variables were determined breath by breath, and gas fractions were analyzed by a mass spectrometer (ARCO-1000, Arco System), while expired gas volume was measured by a Fleisch pneumotachometer (WLCU-5201, We-

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The highest value obtained for oxygen uptake (\(\dot{V}O_2\)) over 30 s was taken as \(\dot{V}O_2\)peak.

**Exercise and experimental protocol.** The subjects were seated on a semisupine cycle ergometer (Cateye-Ergociser EC-3700, Cateye) with a backrest inclination of \(\sim 40 – 50^\circ\). The upper body of the subject was held by shoulder straps and a waist belt to the cycle frame, and head and neck were also held in a stable position by a padded head rest (Fig. 1). The procedure included a 5-min baseline period, followed by exercise with loads of 30, 50, and 70% of \(\dot{V}O_2\)peak, with each stage lasting 5 min. This graded dynamic exercise was followed by a further 3 min of recovery period in a constant position.

**Cerebral blood flow.** The measurements of CBF in this study were carried out during the rest (for 2 min between the 2nd and 4th min), the exercise stage (for 1 min between the 4th and 5th min) and the recovery (for 1 min between the 2nd and 3rd min). The representative values of CBF at each period were the average of three recordings taken.

The \(Q_{ICA}\) was measured with a high-resolution ultrasound system (Vivid 7 Pro, GE Yokogawa Medical Systems) equipped with a 10-MHz linear transducer. Measurements were performed \(\sim 1.0 – 1.5\) cm distal to the carotid bifurcation on the right ICA, while the subject’s chin was slightly elevated (Fig. 2). We first used brightness mode to measure the mean vessel diameter of ICA (\(D_{ICA}\)) in the longitudinal section, and, thereafter, the Doppler velocity spectrum was identified by pulsed wave Doppler mode. The systolic and diastolic diameters of the ICA were measured, and the \(D_{ICA}\) was calculated in relation to the blood pressure curve: \(D_{ICA} = (\text{systolic diameter} \times 1/3) + (\text{diastolic diameter} \times 2/3)\). Moreover, the time-averaged mean flow velocity obtained by the pulsed wave Doppler mode was defined as the mean blood flow velocity (\(V_{ICA}\); m/s). The recordings of the \(V_{ICA}\) were taken from the average of \(\sim 10\) cardiac cycles to eliminate the effects caused by the breathing cycle. In \(V_{ICA}\) measurement, care was taken to ensure that the probe position was stable, that the insonation angle did not vary (in most cases, \(60^\circ\)), and that the sample volume was positioned in the center of the vessel and adjusted to cover the width of the vessel diameter. Three data of \(D_{ICA}\) and \(V_{ICA}\) were obtained for rest and for the last 1 min of exercise with each workload and for the last 1 min of the recovery period, and then the average of three data was defined as the representative value of \(D_{ICA}\) and \(V_{ICA}\) in the individual period. \(Q_{ICA}\) was calculated by multiplying the cross-sectional area \([\pi \times (D_{ICA}/2)^2]\) with \(V_{ICA}\); \(Q_{ICA} = V_{ICA} \times \text{area} \times 60\) (ml/min).

The \(Q_{VA}\) was measured with a similar Doppler ultrasound systems (Vivid e, GE Yokogawa Medical Systems) equipped with a 10-MHz linear transducer. Measurements were mainly performed between the transverse processes of the C4 and C5 vertebrae on the left side, and the \(Q_{VA}\) was calculated as described for \(Q_{ICA}\). To avoid the ultrasound interference, we chose the right ICA and left VA for CBF measurement. In a pilot study, we confirmed no significant differences in blood flow volume in the left and right side of ICA, whereas the left VA tended to have a larger blood flow than the right VA (31). All of CBF measurements were performed by the same two experienced operators (28, 29).

The coefficients of variation (CV) in \(Q_{ICA}\) and \(Q_{VA}\) were 5.3 \(\pm 1.2\) and 5.8 \(\pm 1.0\) at rest, 6.1 \(\pm 0.9\) and 6.3 \(\pm 1.2\) at 30% \(\dot{V}O_2\)peak, 5.9 \(\pm 0.7\) and 5.8 \(\pm 1.0\) at 50% \(\dot{V}O_2\)peak, and 5.3 \(\pm 0.7\) and 6.3 \(\pm 0.6\) at 70% \(\dot{V}O_2\)peak, respectively. Moreover, we carried out a test-retest experiment to confirm the reproducibility of \(Q_{ICA}\) and \(Q_{VA}\) measurement at rest and during dynamic exercise.

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Fig. 1. Position of the subject while semisupine cycle exercising (A), and the head position for cerebral blood flow measurement (B).

Fig. 2. Ultrasound Doppler screen while the internal carotid (ICA; A) and vertebral artery (VA; C) blood flow are measured. B: probe and head placement.
exercise in the pilot study (n = 6). Such determinations of Q˙ICA and Q˙VA were made with an average CV of 4.6 ± 10% for Q˙ICA and 5.3 ± 13% for Q˙VA at rest, 6.3 ± 0.9 and 6.2 ± 1.3% at 30% V˙O2peak, 5.0 ± 0.9 and 4.9 ± 1.2% at 50% V˙O2peak, and 6.4 ± 0.8 and 6.5 ± 1.1% at 70% V˙O2peak, respectively. The intraclass correlation coefficient of the repeated measurements of Q˙ICA was 0.94, and that in Q˙VA was 0.96, respectively. The CVs in Q˙ICA and Q˙VA at rest and during dynamic exercise were within the range of reported values at rest and all workloads (11, 13, 14, 31).

Cardiorespiratory responses. Heart rate was continuously monitored using a three-lead electrocardiogram (OEC-6401, Nihon Koden). Beat-to-beat blood pressure was measured using finger photoplethysmography obtained from the middle or index finger of the nondominant hand (Finometer, Finapres Medical Systems). These methods of blood pressure measurement have been validated for use both at rest and during low to moderate level of exercise (5). Furthermore, stroke volume and CO were determined from the blood pressure waveform by the Modelflow software program that incorporates sex, age, height, and weight (Beat Scope 1.1, Finapres Medical Systems). This method provides a reliable estimate of change in stroke volume and CO in healthy humans at rest and during moderate exercise (15, 16, 36). Respiratory variables were determined as described for V˙O2peak, and end-tidal partial pressure of CO2 (PETCO2) was measured. The cardiorespiratory responses at rest were analyzed over 2 min that ended 1 min before the onset of exercise. During exercise, these parameters were analyzed within the last 1 min of each workload. In addition, the data of the last 1 min of the recovery period were analyzed.

Data processing and statistics. The ratio of mean arterial pressure (MAP) at ICA level to Q˙ICA and the ratio of MAP at VA level to Q˙VA were, respectively, taken as indexes of cerebrovascular resistance (CVRICA and CVRVA). The MAP at ICA level or VA level took into consideration the vertical distance from the fourth intercostal space in the midclavicular line (heart level) to the Doppler probe (i.e., hydrostatic pressure = the vertical distance × 0.77 mmHg/cm) (25). The gCBF was calculated as the sum of volume flow in ICA and VA [(Q˙ICA + Q˙VA) × 2 (ml/min)] (7). The distribution of CO to brain was expressed as gCBF/CO × 100 (%). The relative contribution of Q˙ICA to gCBF and Q˙VA to gCBF was estimated as Q˙ICA/gCBF × 100 (%) and Q˙VA/gCBF × 100 (%), respectively.

The cerebrovascular and cardiorespiratory responses at rest were analyzed over 2 min that ended 1 min before the onset of exercise. During exercise, these parameters were analyzed from the last 1 min of each workload and also expressed relative to rest. In addition, the data of the last 1 min of recovery period were analyzed.

Values are expressed as means ± SE, and differences between values at rest, exercise, and recovery were evaluated by ANOVA with repeated measures and Dunnett post hoc test. To compare differences between changes in the cerebrovascular responses in the ICA and VA, two-way repeated-measures ANOVA were used. If the data were normally distributed, a two sample t-test was performed. Otherwise, Wilcoxon signed-rank test was used (SPSS12.0, SPSS), and P < 0.05 was considered to indicate a significant difference.

RESULTS

The resting values, the change in the cardio-respiratory and cerebrovascular responses to graded exercise, and the recovery are shown in Table 1. V˙O2, MAP, heart rate, and CO increased with workload (P < 0.01) and also PETCO2 was higher than at rest (P < 0.01). Yet, at the 70% V˙O2peak workload, PETCO2 was lower than at 50% V˙O2peak (from 45.3 ± 1.4 to 43.2 ± 1.6 Torr; P < 0.05). All cardio-respiratory variables in the recovery were lower than during exercise, but they remained higher than at rest.

We confirmed whether metabolism had reached steady state at each workload by difference in V˙O2 between the 4th or 5th min of each exercise stage. According to this results, there were no significant differences in V˙O2 between the 4th or 5th min of exercise during 30 and 50% V˙O2peak, suggesting that a steady-state V˙O2 was achieved during 30 and 50% V˙O2peak. However, 70% V˙O2peak did not show a steady-state V˙O2.

With exercise, Q˙ICA increased (P < 0.01: Table 1 and Fig. 3A). At 30% V˙O2peak, the increase was by 11.6 ± 15.5%, and it then

| Table 1. Cardiorespiratory and cerebrovascular variables at rest, during dynamic exercise, and at recovery |
|-------------|-------------|-------------|-------------|-------------|-------------|
| V˙O2, ml/min | 236 ± 9     | 904 ± 32*   | 1335 ± 68*  | 1728 ± 77*  | 377 ± 27*   |
| MAP, mmHg    | 77 ± 2      | 90 ± 2*     | 102 ± 3*    | 114 ± 2*    | 83 ± 2      |
| HR, beats/min| 60 ± 2      | 93 ± 2*     | 127 ± 3*    | 159 ± 3*    | 79 ± 3*     |
| CO, 1/min    | 4.4 ± 0.5   | 8.7 ± 0.4*  | 11.5 ± 0.4* | 16.1 ± 0.6* | 6.5 ± 0.2*  |
| PetCO2, Torr | 40.3 ± 0.9  | 44.7 ± 1.3* | 45.3 ± 1.4* | 43.2 ± 1.6* | 42.7 ± 2.6* |
| Q˙ICA, ml/min| 295 ± 20    | 330 ± 24*   | 349 ± 25*   | 344 ± 21*   | 311 ± 23*   |
| Change from rest, % | 0     | 11.6 ± 1.5  | 18.4 ± 2.7  | 17.2 ± 2.0  | 5.1 ± 2.2   |
| DICA, cm     | 0.48 ± 0.02 | 0.49 ± 0.02 | 0.49 ± 0.02 | 0.49 ± 0.02 | 0.48 ± 0.02 |
| V˙ICA, cm/s  | 28.0 ± 2.5  | 30.9 ± 3.2* | 31.1 ± 2.8* | 31.7 ± 2.7* | 29.6 ± 2.8  |
| Q˙VA, ml/min | 95 ± 5      | 111 ± 8*    | 126 ± 7*    | 132 ± 8*    | 109 ± 7*    |
| Change from rest, % | 0      | 16.8 ± 3.1  | 32.8 ± 3.6  | 39.5 ± 3.4  | 15.8 ± 4.0  |
| DVA, cm      | 0.32 ± 0.01*| 0.33 ± 0.01*| 0.33 ± 0.01*| 0.33 ± 0.01*| 0.33 ± 0.01*|
| V˙VA, cm/s   | 19.9 ± 0.9  | 21.9 ± 1.1* | 24.5 ± 1.0* | 25.3 ± 1.0* | 21.2 ± 0.7  |
| CVRICA, mmHg·ml⁻¹·min⁻¹ | 0.26 ± 0.02 | 0.28 ± 0.03 | 0.30 ± 0.03*| 0.34 ± 0.03*| 0.27 ± 0.02 |
| CVRVA, mmHg·ml⁻¹·min⁻¹ | 0.81 ± 0.05 | 0.83 ± 0.07 | 0.82 ± 0.05 | 0.87 ± 0.05 | 0.76 ± 0.05 |
| gCBF, ml/min | 779 ± 40    | 883 ± 51*   | 950 ± 52*   | 952 ± 45*   | 840 ± 47*   |
| Change from rest, % | 0      | 12.9 ± 1.5  | 21.9 ± 2.5  | 22.5 ± 2.0  | 7.6 ± 1.6   |
| Q˙ICA/gCBF, % | 17.7 ± 1.2  | 10.3 ± 0.7* | 8.4 ± 0.6*  | 6.4 ± 0.4*  | 13.0 ± 0.8* |
| Q˙VA/gCBF, % | 75.2 ± 1.8  | 74.3 ± 2.0  | 73.0 ± 2.0  | 71.9 ± 1.8* | 73.4 ± 2.0  |
| gCBF/CO × 100, % | 24.8 ± 1.8  | 25.7 ± 2.0  | 27.0 ± 2.0  | 28.1 ± 1.8* | 26.2 ± 2.0  |

Values are means ± SE. V˙O2, oxygen uptake; V˙O2peak, peak V˙O2; MAP, mean arterial pressure; HR, heart rate; CO, cardiac output; PetCO2, end-tidal partial pressure of CO2; Q˙ICA and Q˙VA, blood flow in internal carotid arteries (ICA) and vertebral arteries (VA), respectively; V˙ICA and V˙VA, mean blood flow velocities in the ICA and VA, respectively; DICA and DVA, mean diameter in ICA and VA, respectively; CVRICA and CVRVA, index of cerebrovascular resistance in the ICA and VA, respectively; gCBF, global cerebral blood flow [(Q˙ICA + Q˙VA) × 2]; gCBF/CO × 100 (%), the distribution of CO to brain; Q˙ICA/gCBF (%), the relative contribution of Q˙ICA to gCBF; Q˙VA/gCBF (%), the relative contribution of Q˙VA to gCBF. *Different from rest (P < 0.05).
Differential responses between the QICA and QVA during dynamic exercise. The increase in QICA leveled off over an intensity of 50% \(\dot{V}_\text{O}_2\text{peak}\), whereas the continuous increase in QVA occurred until an intensity of 70% \(\dot{V}_\text{O}_2\text{peak}\), and that the CVRICA increased with increasing exercise load, whereas CVRVA remained stable throughout the graded intensities. These results confirmed our hypothesis that the cerebrovascular responses to dynamic exercise are different between ICA and VA systems. Furthermore, we found that gCBF was elevated \(\sim 20\%\) during dynamic exercise, and that the relative contribution of ICA and VA systems to the gCBF varied during dynamic exercise.

**DISCUSSION**

Using Doppler ultrasound, we examined the simultaneous blood flow responses in ICA and VA during graded dynamic exercise. The major findings of the present study were that the increase in QICA leveled off over the intensity of 50% \(\dot{V}_\text{O}_2\text{peak}\), whereas the continuous increase in QVA occurred until an intensity of 70% \(\dot{V}_\text{O}_2\text{peak}\), and that the CVRICA increased with increasing exercise load, whereas CVRVA remained stable throughout the graded intensities. These results confirmed our hypothesis that the cerebrovascular responses to dynamic exercise are different between ICA and VA systems. Furthermore, we found that gCBF was elevated \(\sim 20\%\) during dynamic exercise, and that the relative contribution of ICA and VA systems to the gCBF varied during dynamic exercise.

**DIFFERENTIAL RESPONSES BETWEEN THE QICA AND QVA DURING DYNAMIC EXERCISE**

The increase in QICA leveled off over an intensity of 50% \(\dot{V}_\text{O}_2\text{peak}\) during cycling exercise, which was consistent with the previous report (11). However, the QVA increased progressively with graded intensities of exercise up to 70% \(\dot{V}_\text{O}_2\text{peak}\). These different cerebrovascular responses between ICA and VA during dynamic exercise are probably mediated by several factors and/or mechanisms. The first possible explanation is that the neurovascular responses in the brain were regionally different between the territories covered by the ICA system and the territories covered by the VA system and thereby resulted in different cerebrovascular responses between ICA and VA systems. The previous animal studies support this explanation (3, 8). Delp et al. (4) reported that the blood flow in the cortical areas showed less increase to maximal exercise than that in the brain stem, spinal cord, and the cerebellum. A second possibility is that anatomic differences might exist between ICA and VA systems. In line with this explanation, the histological studies of Edvinson et al. (6) have shown regional differences concerning density of \(\beta\)-adrenergic, cholinergic, and serotonergic innervation of the intracerebral vessels, which may have different influences on the cerebrovascular resistance. In addition, pharmacological studies have suggested regional differences in the sensitivity to vasoactive substances, e.g., noradrenaline (9). The third factor is the difference in cerebral CO2 reactivity (3, 18, 35) and/or autoregulatory control (10, 21) between ICA and VA systems in humans. The present study indicates that cerebral CO2 reactivity during moderate exercise is reduced in the VA system compared with the ICA system (35). In addition, previous studies have demonstrated impaired autoregulation in the VA system compared with the ICA system (10). Thus several factors probably contributed to the different cerebrovascular responses observed between the ICA and VA systems during dynamic exercise in the present study. Further research is required to clarify the detail mechanisms.

**GCBF RESPONSES DURING DYNAMIC EXERCISE**

We found that gCBF, calculated as the sum of QICA and QVA, elevated \(\sim 20\%\) during dynamic exercise, which was consistent with the previous report (11). However, the QVA increased progressively with graded intensities of exercise up to 70% \(\dot{V}_\text{O}_2\text{peak}\). These different cerebrovascular responses between ICA and VA during dynamic exercise are probably mediated by several factors and/or mechanisms. The first possible explanation is that the neurovascular responses in the brain were regionally different between the territories covered by the ICA system and the territories covered by the VA system and thereby resulted in different cerebrovascular responses between ICA and VA systems. The previous animal studies support this explanation (3, 8). Delp et al. (4) reported that the blood flow in the cortical areas showed less increase to maximal exercise than that in the brain stem, spinal cord, and the cerebellum. A second possibility is that anatomic differences might exist between ICA and VA systems. In line with this explanation, the histological studies of Edvinson et al. (6) have shown regional differences concerning density of \(\beta\)-adrenergic, cholinergic, and serotonergic innervation of the intracerebral vessels, which may have different influences on the cerebrovascular resistance. In addition, pharmacological studies have suggested regional differences in the sensitivity to vasoactive substances, e.g., noradrenaline (9). The third factor is the difference in cerebral CO2 reactivity (3, 18, 35) and/or autoregulatory control (10, 21) between ICA and VA systems in humans. The present study indicates that cerebral CO2 reactivity during moderate exercise is reduced in the VA system compared with the ICA system (35). In addition, previous studies have demonstrated impaired autoregulation in the VA system compared with the ICA system (10). Thus several factors probably contributed to the different cerebrovascular responses observed between the ICA and VA systems during dynamic exercise in the present study. Further research is required to clarify the detail mechanisms.
期间的动态运动，尽管各系统间存在差异，但ICA和VA的血流在静息时有所不同（30, 32）。进一步研究发现，使用Kety-Schmidt法测得的CBF在不同系统的动态运动中不一致。然而，如果测量来自男性受试者的数据，这种情况可能发生变化。

### Limitations

研究的主要限制包括：
- 动态运动期间，CBF可能不会达到最大值（100% VO2peak），因为CBF测量值在最大运动时可能不一致。
- 可能由于运动的不对称性而导致静脉回流的改变。
- 动态运动期间，CBF对脑血流的相对贡献在静息时大于运动期间。
- 在裤底运动中，CBF的变化可能大于运动期间。
- CBV变化可能取决于运动强度。
- CBF的测量可能受到不同系统的影响。
- 研究结果可能随性别而变化。

### Acknowledgments

我们特别感谢Hiroyuki Yamamoto (GE Yokogawa Medical Systems, Tokyo, Japan)和Ai Hirasawa (Research Institute of Physical Fitness, Japan Women’s College of Physical Education)为我们提供了宝贵的指导。


