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Oscillation in O₂ uptake in impulse exercise

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Running title: O₂ uptake and heart rate

The purpose of the present study was to examine 1) whether O_2 uptake ($\dot{\text{V}}\text{O}_2$) oscillates during light exercise and 2) whether the oscillation is enhanced after impulse exercise. After resting for 1 min on a bicycle seat, subjects performed 5-min pre-exercise with 25 watts work load, 10-s impulse exercise with 200 watts work load and 15-min post exercise with 25 watts work load at 80 rpm. $\dot{\text{V}}\text{O}_2$ during pre-exercise significantly increased during impulse exercise and suddenly decreased and re-increased until 23 s after impulse exercise. In the cross correlation between heart rate (HR) and $\dot{\text{V}}\text{O}_2$ after impulse exercise, $\dot{\text{V}}\text{O}_2$ strongly correlated to HR with a time delay of -4 s. Peak of power spectral density (PSD) in HR appeared at 0.0039 Hz and peak of PSD in $\dot{\text{V}}\text{O}_2$ appeared at 0.019 Hz. The peak of the cross power spectrum between $\dot{\text{V}}\text{O}_2$ and HR appeared at 0.0078 Hz. The results suggested that there is an oscillation in O_2 uptake during light exercise that is associated with the oscillation in O_2 consumption in active muscle. The oscillation is enhanced not only by change in O_2 consumption but also by O_2 content transported from active muscle to the lungs.

Key words: O_2 uptake, O_2 consumption, heart rate, impulse exercise, oscillation, power spectral density

Recent studies on O₂ consumption kinetics at the onset of exercise have focused on whether O₂ consumption in active muscle is determined by O₂ supply or by an intramuscular mechanism (O₂ delivery limitation or O₂ availability hypothesis) (2). In examining this hypothesis, a one-way direction from cause to result is assumed as a fundamental principle. However, the principle itself cannot be verified by an experiment. Therefore, it is not always necessary to be restricted by the causality. Recently, system theory has been developed. In the system, interaction of factors induces oscillation. Generally speaking, the following oscillation system is proposed: When factor α is increased by another factor continuously supplied, factor β is increased by factor α . Furthermore, if factor β inhibits factor α and factor β naturally disappears, the factors oscillate with different phases (8). Typical examples of the factors are concentrations of chemical substances. It has been shown that there is a complex dissipative system in the TCA cycle and glycolysis in yeast (1, 4, 8). Thus, in the system approach, it is assumed that factors e. g., O₂ consumption in active muscle and O₂ uptake in the lungs interact with each other and that the interactions lead to self-organization of the system for muscular work. In this system, the interactions are physiological mechanisms.

It is known that heart rate oscillates at rest. The high-frequency band is 0.15-0.5 Hz and the low-frequency band is 0.05-0.15Hz (5). If cardiac pumping affects O₂ uptake, O₂ uptake should oscillate during exercise. Some studies have shown that oxygenation in exercising muscle has slower oscillation (12, 13, 14), and it has been shown that there is an oscillation in the recovery of phosphocreatine from exercise (4). Therefore, it is assumed that O₂ consumption has a rhythm. This rhythm is slower than the lower band of heartbeat. If a slow rhythm of the same level of O₂ consumption is found in O₂ uptake, it is assumed that there is interaction between O₂ uptake and O₂ consumption through the control of cardiac pumping. Since O₂ consumption is also known to be enhanced in impulse exercise (3, 16), temporal enhancement of oscillation in O₂ uptake may occur after the reaction for O₂ consumption for impulse exercise.

The purpose of the present study was therefore to examine 1) whether O₂ uptake oscillates during light exercise and 2) whether the oscillation is enhanced after impulse exercise.

Materials and Methods

A. Subjects

Eight healthy males participated in this study. The subjects' mean age, height and body weight were 21.3 $\pm$ 1.5 (SD) yr, 172.9 $\pm$ 6.2 cm and 67.9 $\pm$ 9.7 kg, respectively.

Each subject signed a statement of informed consent following a full explanation regarding the nature of the experiment. The Ethics Committee of Hokkaido University Graduate School of Education approved the present study.

B. Experimental protocol

Each subject performed a pre-test and main test consisting of one impulse exercise by a bicycle ergometer (Ergometer 232 CXL, Combi, Tokyo, Japan). After resting for 1 min on a bicycle seat, subjects performed 5-min pre-exercise with 25 watts work load, 10-s impulse exercise with 200 watts work load and 15-min post exercise with 25 watts work load at 80 rpm.

C. Measurements and determinations

In the pre-test, we checked whether blood lactate level (La) is increased by the impulse exercise used in the present study. Blood was sampled from fingertips at rest and after 1 min and 5 min during post exercise after the impulse exercise. La in blood samples was measured by using Lactate Pro LT-1710 (ARKRAY Corp.). Each subject's hand was pre-warmed in 40-45°C water prior to each test in order to arterialize capillary blood (11). There was no increase observed in the pre-test.

Data on respiration gas exchange were obtained using a respiratory gas analyzer by breath-by-breath mode (AE-280S, Minato Medical Science, Osaka, Japan). Ventilation (VE) was measured by a hot-wire flow meter, and the flow meter was calibrated with a syringe of known volume (2 liters). O₂ and CO₂ concentrations were measured by a zirconium sensor and infrared absorption analyzer, respectively. The gas analyzer was calibrated by known standard gas (O₂: 15.17%, CO₂: 4.9%). Respiration gas exchange was measured continuously during rest, exercise, and recovery periods. Heart rate (HR) was recorded using a heart rate monitor installed in the respiratory gas analyzer. O₂ uptake (VO₂) and HR were obtained breath-by-breath.

D. Calculation and statistical analysis

In a previous study, in order to obtain 1-s data, breath-by-breath data obtained in repeated exercise with a time interval were converted to 1-s data in each exercise, and the data obtained in each exercise were summated (10). However, in this method, the oscillation of measured data is eliminated by the summation. In order to avoid this effect, breath-by-breath data were interpolated into 1-s data using three-dimensional spline in the present study. However, there is also a problem in this method. Higher

frequency of oscillation than respiration rate has no meaning.

The 1-s data were analyzed by fast Fourier transform (FFT) for the period from 500 s to 1000 s from the start of the test (See Fig. 1. Since $\dot{V}O_2$ and HR each show a steady state from 450 s, data in this range were used for FFT analysis.). Power spectral density (PSD) was calculated using five rectangular windows with an overlap of 50%. In order to visualize the data of low frequency, a low pass filter was used. The pass frequency was set below 0.05 Hz. A cross correlation was obtained using average data from 375 s (5 s after impulse exercise) to 650 s from the start of the test. A cross power spectrum (CPS) between $\dot{V}O_2$ and HR was obtained using average data from 375 s to 650 s from the start of the test. Results are presented as means $\pm$ standard deviations. The paired t-test was used to examine significant difference between two values.

Results

Blood lactate (La) was 1.15 ± 0.29 mM at rest. La after impulse exercise was 1.23 ± 0.43 mM at 1 min and 1.0 ± 0.13 mM at 5 min during post exercise. There was no significant difference between La at rest and that during post exercise.

As shown in Figure 1, $\dot{V}O_2$ during pre-exercise (0.8 ± 0.13 l/min) significantly increased during impulse exercise to 1.05 ± 0.12 l/min. $\dot{V}O_2$ suddenly decreased and re-increased until 23 s after impulse exercise. The peak value was 1.27 ± 0.20 l/min. $\dot{V}O_2$ during post exercise returned to the pre-exercise value. HR during pre-exercise (89 ± 9.9 beats/min) showed a rapid significant increase during impulse exercise and then decreased during post exercise. Peak HR appeared at the end of impulse exercise. Peak HR was 115 ± 8.91 beats/min.

Figure 2 shows the relationship between average HR and $\dot{V}O_2$. There was a significant correlation coefficient ($r=0.878$).

Figure 3 shows the cross correlation between HR and $\dot{V}O_2$ after impulse exercise. $\dot{V}O_2$ strongly correlated to HR with a time delay of -4 s.

Figure 4 shows PSD of HR and $\dot{V}O_2$ from 500 s to 1000 s from the start of the test. Peak of PSD in HR appeared at 0.0039 Hz and the second peak of heart rate (HR) appeared at 0.085 Hz. Peak of PSD in $\dot{V}O_2$ appeared at 0.019 Hz and the second peak appeared at 0.035 Hz. Peak of the CPS appeared at 0.0078 Hz.

Figure 5 shows $\dot{V}O_2$ treated by a low pass filter. $\dot{V}O_2$ kinetics in relation to impulse exercise is distorted by this filtering. $\dot{V}O_2$ after 500 s clearly showed oscillations by 100 ml/min in all subjects.

Discussion

Oscillations of O_2 uptake and O_2 consumption

We divided the frequency band into three periods according to previous reports (6, 7): Endothelium-mediated vasodilation (0.005-0.0095 and 0.0095-0.02 Hz) was termed as phase Ia and phase Ib, neurogenic activity on the vessel wall (0.02-0.06 Hz) was termed as phase II, and intrinsic myogenic activity of vascular smooth muscle (0.06-0.16 Hz) was termed as phase III. A frequency band below phase III was not detected due to low frequency of sampling. Therefore, we did not use data above phase III. This division was based on analysis using cutaneous blood perfusion. It has been confirmed that the first peak of deoxygenation determined by near-infrared spectroscopy (NIRS) in the vastus lateralis muscle during light exercise was within phase I and the second peak was within phase II (15). In the present study, O_2 uptake was within phase I. This correspondence suggests that there may be interaction between O_2 consumption in active muscle and O_2 uptake affected by cardiac pumping.

In the present study, frequencies of oscillations between HR and $\dot{V}O_2$ after impulse exercise did not coincide, but peak of the CPS appeared at 0.0078 Hz. Iotti et al. (4) found oscillation of phosphocreatine (PCr) re-synthesis during recovery from exercise in humans. PCr can recover by consuming O_2 . This oscillation reflects O_2 consumption by mitochondria. They also found that that oscillations depend on cytosolic pH. The highest frequency occurs at cytosolic pH>7 and the lowest occurs at pH=6.8. The frequency ranged from 0.002 to 0.025 Hz. It has also been reported that oxygenation in active muscle including venous blood oscillates with very slow frequency (0.01 Hz) with a lower level in light exercise than at rest (12, 13). This frequency coincides with the present peak of the CPS. However, there are cautions about oscillation due to blood transportation as follows. In the venous side, the O_2 content in mixed venous blood could oscillate because the oscillation from active muscle is enlarged more than that at rest and amount of blood from active muscle is large. In the arterial side, O_2 content may oscillate slightly corresponding to fluctuation of alveolar O_2 pressure. A more important factor is arterial CO_2 pressure ($Paco_2$). $Paco_2$ is known to oscillate at the same frequency as oxygenation in active muscle and could affect the oxygenation (14). Therefore, the oscillation of oxygenation might affect the oscillation of $\dot{V}O_2$ probably through cardiac pumping, and oscillation of arterial O_2 content may affect oscillation of oxygen consumption.

When the condition is transformed from a resting state to exercise, the changed direction of the cardiorespiratory system is given by an upper system such as the cerebral system. The direction may be a set point of arterial blood pressure. The operating point is reset in exercise (9). During exercise, substances produced can reduce the peripheral resistance. In order to maintain the set point of arterial blood pressure, cardiac output should be increased. Therefore, in order to maintain the set point, many factors concerning exercise are self-organized through interactions of factors into the direction of constant arterial blood pressure. In the interactions, if the oscillation of oxygenation in active muscle affects cardiac pumping, oxygenation in active muscle could indirectly affect O_2 uptake in the lungs through the change in cardiac pumping.

Enhancement of O_2 uptake after impulse exercise

Average $\dot{V}O_2$ was significantly related to average HR during impulse exercise. This result confirms a relationship between average $\dot{V}O_2$ and HR at the cardiodynamic phase in constant moderate exercise (10). In the present study, $\dot{V}O_2$ was also strongly correlated to HR after impulse exercise. These results suggest that HR affects $\dot{V}O_2$.

The relationship between HR and $\dot{V}O_2$ after impulse exercise indicated a time delay. The time delay expresses the time from the heart to lungs (4 s). $\dot{V}O_2$ is affected not only by cardiac pumping but also by O_2 content of mixed venous blood that comes from active muscle with a time delay. The time delay would be about 20 s judging from the $\dot{V}O_2$ peak after impulse exercise. Although the re-increase in $\dot{V}O_2$ may partly reflect O_2 consumption during impulse exercise, due to the decrease in HR immediately after impulse exercise and the time delay from the muscle to lungs, $\dot{V}O_2$ after impulse exercise should be transformed as a cardiodynamic phase. In this case, $\dot{V}O_2$ is not completely equal to O_2 consumption.

It has been reported that oxygenation in muscle in impulse exercise decreases during impulse exercise and increases during recovery (Yunoki *et al.* 1998). Since the reported data are averaged data, there is no oscillation. This kinetics is likely to average HR kinetics in the present study. However, since there is oscillation in oxygenation during light exercise, the oscillation could be enlarged during impulse exercise. In this case, $\dot{V}O_2$ is enlarged during impulse exercise and decreased during recovery if the interaction is carried out corresponding to the change in oxygenation in impulse exercise. The delayed appearance in $\dot{V}O_2$ from impulse exercise may be strongly affected by the delayed attainment of O_2 content from the active muscle to lungs.

During recovery, $\dot{V}O_2$ is also affected by a decrease in cardiac output. Therefore, there would be two interactions: an interaction between active muscle and the heart and an interaction by the transportation of blood from active muscle and from the lungs.

Conclusions

The results suggested that there is an oscillation in O_2 uptake during light exercise that is associated with the oscillation in O_2 consumption in active muscle. The oscillation is enhanced not only by change in O_2 consumption but also by O_2 content transported from active muscle to the lungs.

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Legends of figures

Fig. 1. $\dot{V}O_2$ uptake ($\dot{V}O_2$) and heart rate (HR) kinetics. The straight lines show the duration of impulse exercise. Before and after impulse exercise, light exercises (25 watts) were performed.

Fig. 2. Relationship between average heart rate (HR) and average $\dot{V}O_2$ uptake ($\dot{V}O_2$) during impulse exercise.

Fig. 3. Cross correlation between average $\dot{V}O_2$ uptake ($\dot{V}O_2$) and average heart rate (HR) 5 s after impulse exercise.

Fig. 4. Power spectra density (PSD) in heart rate (HR: upper panel) and $\dot{V}O_2$ uptake ($\dot{V}O_2$: middle panel) obtained from 500 s to 1000 s after the starting point of a test. Cross power spectrum (CPS) is shown in the lower panel. A dotted line shows the division of each phase determined by Yano et al. (2013).

Fig. 5. $\dot{V}O_2$ uptake ($\dot{V}O_2$) treated by a low pass filter is shown in all subjects. A straight line shows impulse exercise. Before and after impulse exercise, light exercises (25 watts) were performed.

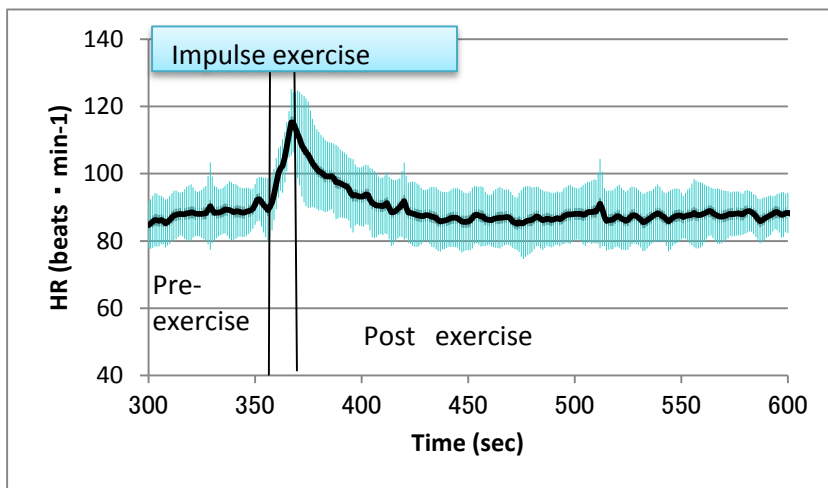
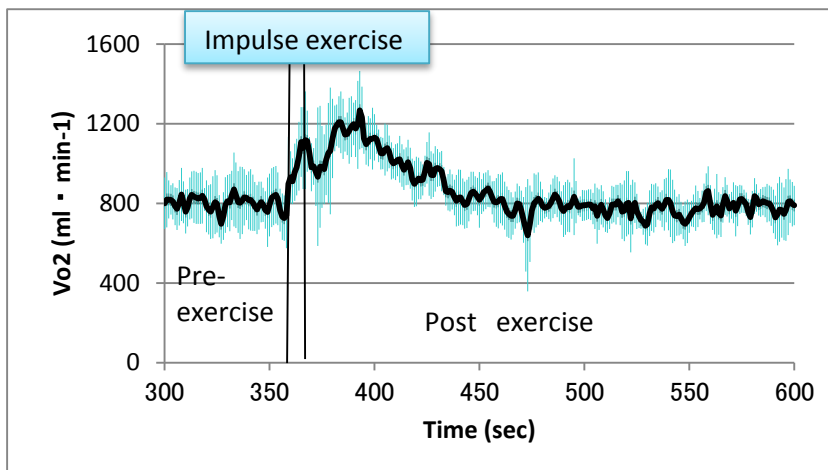


Fig. 1.

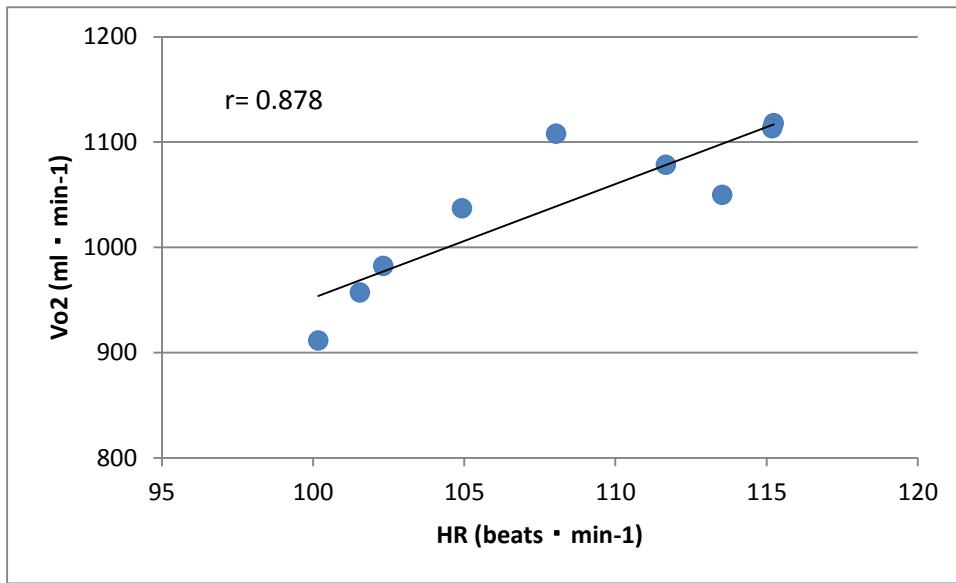


Fig. 2

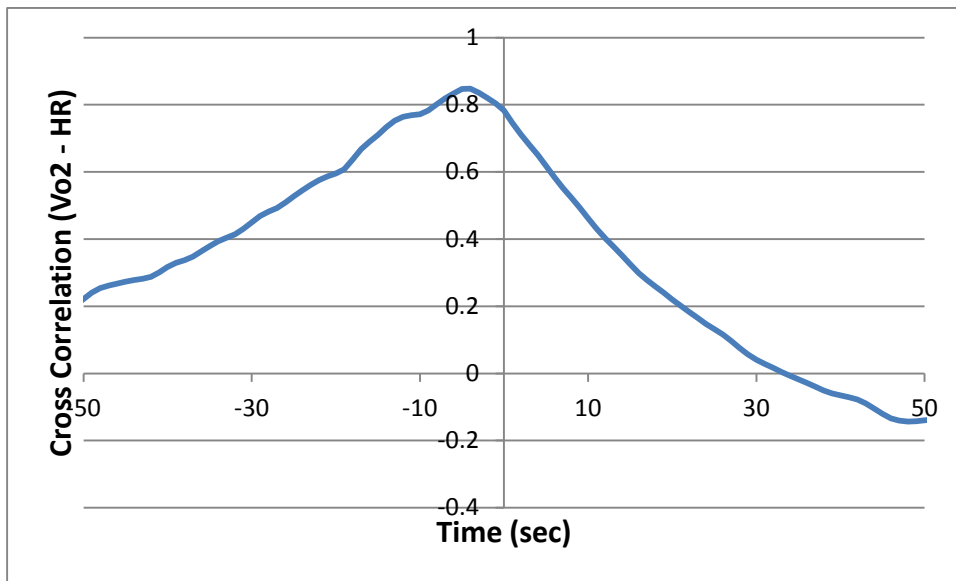


Fig. 3.

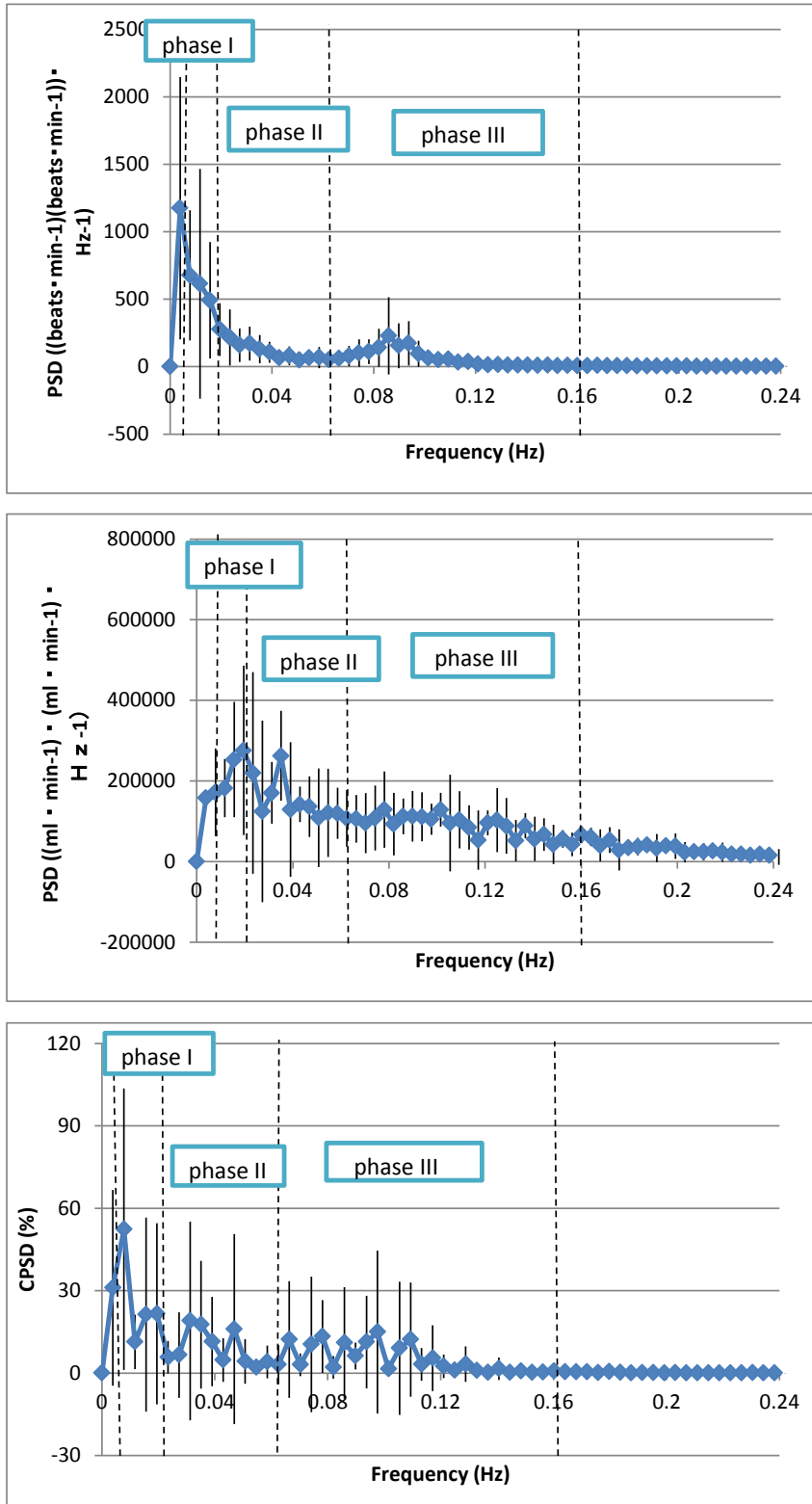


Fig. 4.

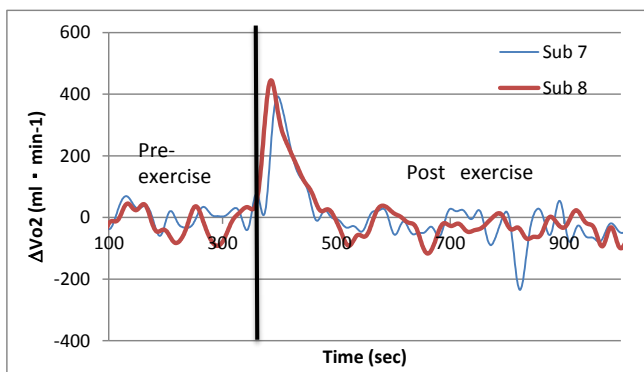
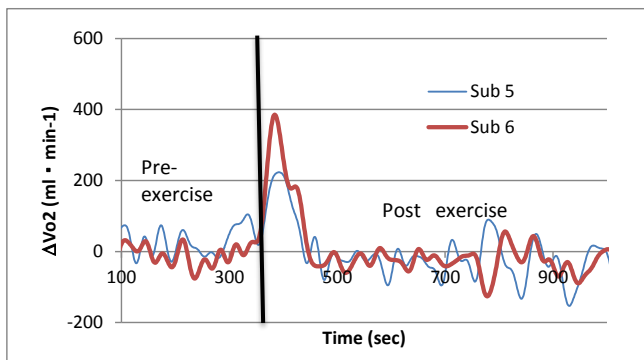
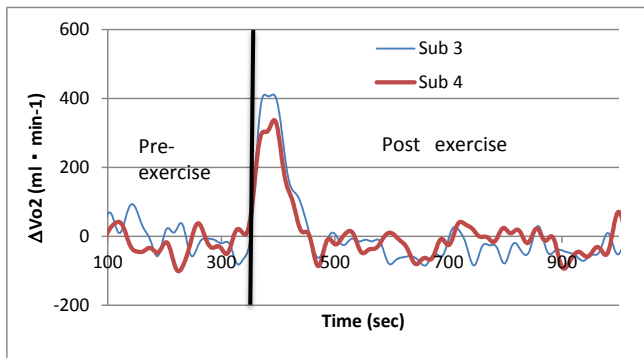
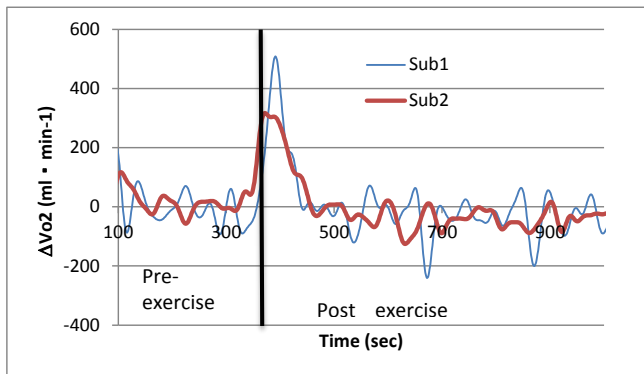


Fig. 5.