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# Oxygen Kinetics in Response to Impulse Work

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## Introduction

There have been several studies on the kinetics of oxygen uptake ($\dot{V}O_2$) responding to impulse work (Bakker et al., 1980; Hughson et al., 1988; Swanson, 1990; Whipp and Ward, 1991), and it has been demonstrated that $\dot{V}O_2$ is composed of two components (phase I and phase II). With regard to these components, Hughson et al. (1988) have suggested that phase I reflects an immediate increase in pulmonary blood flow and phase II reflects tissue $\dot{V}O_2$. However, this inference is derived only from an analysis of $\dot{V}O_2$ kinetics and not from correspondence to the oxygen consumption in working muscles.

It has recently become possible to noninvasively and continuously measure the kinetics of $O_2$ in working muscles due to the development of near-infrared spectroscopy (NIRS) (Chance et al., 1992; Kuwamori et al., 1995; Homma et al., 1992). In this study, by comparing the change in deoxygenated hemoglobin (deoxy Hb) in the working muscle using NIRS during impulse work with the change in $\dot{V}O_2$, we reexamined the hypothesis proposed by Hughson et al. (1988).

## Methods

Four healthy male subjects (means ± S.D.: age, 31.3 ± 10.2 years; body mass, 67.1 ± 6.8 kg; height 174.5 ± 0.6 cm) participated in this study. All exercise was performed in the upright position on a bicycle ergometer (Ergometer 232C: Combi) at 80 rpm.

The subjects were kept in a resting state on the ergometer for 5 min. Then, a work rate of 25 W was performed for 5 min followed by impulse work of 300 W for 20 sec, and a work rate of 25 W was loaded again as a recovery exercise for 7 min. An impulse work load of 300 W was used for the following reasons. First, the maximum work rate of the ergometer used in this study was 300 W. Although this work rate can be set for a duration of 10 sec, as it was in a former study (Hughson et al., 1988), it takes a few seconds to attain 300 W. Thus, in order to obtain a clear effect of 300 W, we set the period of impulse work at 20 sec. The experiment was performed once a day and repeated three times on separate days.

Deoxy Hb was measured in the rectus femoris in the first and third experiments and in the vastus lateralis in the second experiment by NIRS (HEO-200: Omron) during exercise. Data of deoxy Hb was obtained every 3 sec. Parameters of pulmonary gas exchange and heart rate (HR) were obtained breath-by-breath using a computerized system (Aeromoniter AE-280S: Minato).

Breath-by-breath data, $\dot{V}O_2$ and HR were interpolated every second to sum up the data of all subjects. The 12 data values (4 subjects × 3 repetitions) were averaged every second. The data were averaged over 5 sec and are plotted in Fig. 1 A and B. The data of deoxy Hb in all subjects were also averaged every 3 sec and are shown in Fig. 1-C-a and b.

## Results

The $\dot{V}O_2$ response to impulse work can be separated into two phases (Fig. 1-A). $\dot{V}O_2$-phase I started to increase instantaneously at the onset of impulse work and reached a peak at about 20 sec, i.e., at the end of the impulse work period. $\dot{V}O_2$-phase II started to increase at 25–30 sec after the start of impulse work and reached a peak at about 20 sec after the end of impulse work, followed by a stable level for about 10 sec. HR rose a little before the start of impulse work and increased rapidly after impulse work started. HR peaked at the end of impulse work and then decreased (Fig. 1-B).

Changes in deoxy Hb in the rectus femoris and vastus lateralis are presented in Fig. 1-C-a and b. In both muscles, deoxy Hb decreased after a delay of a few seconds at the onset of impulse work. Changes in deoxy Hb expresses an approximate value for changes in oxygenated hemoglobin (oxy Hb). Therefore, the decrease in deoxy Hb in Fig. 1 implies a decrease in oxy Hb. In the rectus femoris, deoxy Hb decreased until the end of impulse work and then maintained a stable level for about 10 sec. In the vastus lateralis, it also peaked at the end of impulse work and then maintained a stable level for about 5 sec. The recovery phase was about 50 sec in the rectus femoris and 30 sec in the vastus lateralis.

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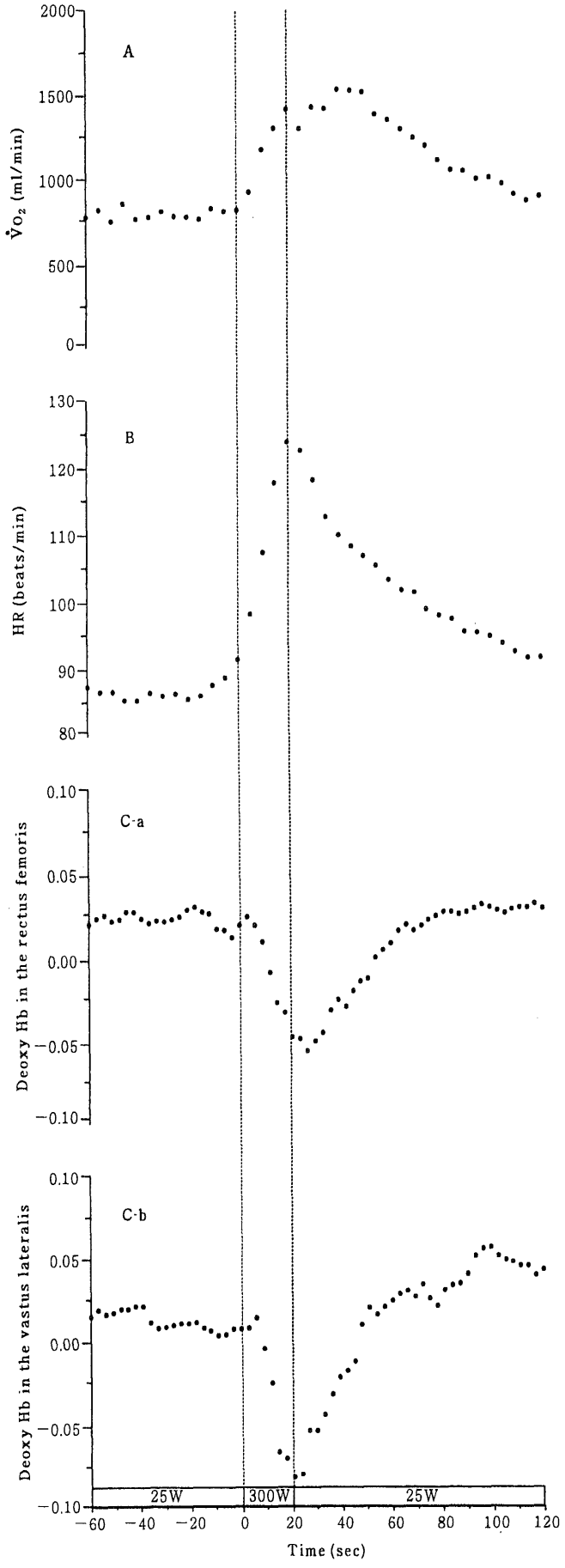


## Discussion

The $\dot{V}O_2$ response to impulse work can be separated into two phases (phases I and II), and the kinetics of deoxy Hb in the rectus femoris and vastus lateralis in the recovery phase are different. The significance of each of these phases is discussed below.

(1) Phase I

Deoxy Hb showed a delay at the onset of impulse work, while $\dot{V}O_2$-phase I showed no delay. Although this delay may be due to the effect of the drift of deoxy Hb appearing before the start of impulse work, it is difficult to account for $\dot{V}O_2$-phase I by the changes in deoxy Hb. Thus, the origin of $\dot{V}O_2$-phase I does not appear to be $O_2$ kinetics in the working muscle. Hughson et al. (1988) suggested that $\dot{V}O_2$-phase I is related to the increase in pulmonary blood flow. In the present study, although HR increased a little just before the start of impulse work, the main increase in HR was after the start of impulse work and HR reached a peak at the same time as $\dot{V}O_2$-phase I did. Since stroke volume is also reported to increase immediately after the onset of exercise (Loeppky et al., 1981), the hypothesis of Hughson et al. (1988) appears to be valid.

(2) Phase II

Since it has been suggested that there is transport lag from the working muscle to the lung at the onset of exercise (Linnarsson, 1974), $\dot{V}O_2$-phase II is thought to reflect $O_2$ consumption of the working muscles. Therefore, we investigated the time delay from the difference between the peak time of deoxy Hb and that of $\dot{V}O_2$-phase II, and we found the delay to be about 20 sec. If blood volume is assumed to be 4L, we must regard cardiac output as 12L/min to account for this time delay. According to a former study (Åstrand, 1964), this value of cardiac output corresponds to 1.0–1.5 L/min of $\dot{V}O_2$. This value of $\dot{V}O_2$ is close to the present result. Although deoxy Hb shows a similar change to that of $\dot{V}O_2$, deoxy Hb is considered to reflect the difference between $O_2$ transport and $O_2$ consumption in tissue. Furthermore, it is suggested that changes in oxyhemoglobin + oxymyoglobin saturation by NIRS is close to changes in venous $O_2$ content (Belardinelli et al., 1995). Consequently, deoxy Hb could be considered to be an indirect index for $VO_2$. Therefore, reexamination of the hypothesis of Hughson et al. on $\dot{V}O_2$-phase II (1988) by using changes in deoxy Hb would require a further approach.

**Fig. 1** Group mean responses of oxygen uptake (A), heart rate (B), and deoxygenated hemoglobin in the rectus femoris (C-a) and the vastus lateralis (C-b) during impulse test.

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(3) Recovery phase

Kushmerick et al. (1992) investigated $O_2$ kinetics of the muscle during and after isometric contraction using animals, and they demonstrated that the recovery time of creatine phosphate (CP) resynthesis was faster in type I muscle than in type II muscle. Since it is thought that this resynthesis of CP requires $O_2$ consumption, the kinetics of recovery $O_2$ consumption would differ according to the difference in muscle fiber type. As fast twitch fiber is richer in the rectus femoris than in the vastus lateralis (Weineck, 1984), the difference in the recovery time for deoxy Hb in both muscles observed in this study may be associated with the process of recovery for CP resynthesis.

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