Chapter 20
O_2 Saturation in the Intercostal Space During Moderate and Heavy Constant-Load Exercise

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Abstract To examine the hypothesis that the relationship between minute ventilation (V_E) and deoxygenation from the intercostal space (IC) would be steady regardless of exercise protocols, if an increase in O_2 consumption of the accessory respiratory muscles with an increase of V_E brings about deoxygenation in IC, we measured the relationship between V_E and O_2 saturation in IC (SO_{2IC}) during a constant-load exercise test (CET), and the relationship was compared with that during a ramp incremental exercise test (RIET). Six male subjects performed RIET. On a different day, the subjects performed a moderate and heavy CET (CET_MOD and CET_HVY, respectively). SO_{2IC} decreased from the start of both CET_MOD and CET_HVY and changed little from 2 min. Moreover, SO_{2IC} was significantly lower during CET_HVY than during CET_MOD. In comparison between RIET and CET_HVY at the similar V_E level, SO_{2IC} was significantly higher during CET_HVY than RIET. These results suggest that the decrease in SO_{2IC} was caused not only by an increase in O_2 consumption in IC with an increase in V_E but also by a decrease in O_2 supply.

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20.1 Introduction

During incremental cycling exercise, deoxygenation, measured by near-infrared spectroscopy (NIRS), in the working muscle (the vastus lateralis) was attenuated at high intensity [1–4], and in previous studies, it was thought to be related to metabolism of the respiratory and accessory respiratory muscles [1]. Deoxygenation from the intercostal space (IC) hyperbolically increased until exhaustion. The deoxygenation was thought to be because the recruitment of the accessory respiratory muscles increased the O_2 consumption. However, since the NIRS data reflect the balance of O_2 supply and consumption [5, 6], deoxygenation was not always induced by an increase of O_2 consumption but also by a decrease of O_2 supply.

During constant-load exercise, minute ventilation (V_E) increases gradually from the start of exercise [7]. However, little is known about deoxygenation in IC. If deoxygenation in IC was led by an increase in O_2 consumption, it could be hypothesized that deoxygenation was controlled by the amplitude of V_E, independent of exercise protocols. However, if deoxygenation was different between protocols despite the same V_E, deoxygenation would be influenced not only by the change of O_2 consumption but also by O_2 supply.

The purpose of this study was to examine deoxygenation in IC during constant-load exercise and to compare between deoxygenation during constant-load exercise with that during incremental exercise at the similar V_E level.

20.2 Methods

20.2.1 Subjects

Six active male volunteers participated in this study (age, 22 ± 1 years; height, 170.5 ± 5.7 cm; body mass, 67.3 ± 12.0 kg; mean ± S.D.). Before participation in the experiment, all procedures and any potential risks were explained to each subject and an informed consent document was signed. This study was approved by the local ethics committee, and all work was performed in accordance with the Declaration of Helsinki.

20.2.2 Experiment Design

The subjects performed a ramp incremental exercise test (RIET). The protocol followed 0-W exercise for 1 min and increased at a ramp rate of 20 $W \cdot min^{-1}$ to exhaustion. Ventilatory threshold (VT) calculated by a V-slope method [8] and peak O_2 uptake (VO_{2peak}) were evaluated. On a different day, the subjects performed a moderate (CET_MOD; the intensity was 90 % of VT) and heavy (CET_HVY; the

intensity was the middle between VT and VO_{2peak}) constant-load exercise test. The protocol followed a warm-up exercise (the same intensity as CET_MOD) for 3 min and 0-W exercise for 6 min and performed CET_MOD and CET_HVY exercise for 6 min separated by 0-W exercise for 6 min. The warm-up exercise was performed to meet the metabolic balance before CET_MOD and CET_HVY exercise. An electromagnetically braked cycle ergometer (Strength Ergo 8, Fukuda Denshi, Japan) was utilized; the seat and handle height remained constant for each subject, and the pedal frequency was maintained at 60 rpm.

20.2.3 Measurements

V_E and O_2 uptake (VO_2) were determined breath by breath by a computerized metabolic cart (AE300S, Minato, Japan). Expiratory flow measurements were performed by a mass flow sensor. V_E was normalized by the peak of V_E during RIET ($\%V_{Epeak-RIET}$). O_2 saturation in IC (SO_{2IC}) was measured with near-infrared spatially resolved spectroscopy (NIRO-200, Hamamatsu photonics, Japan). The optode was placed on the interion between the seventh intercostal space and the anterior axillary line. The source-detector distance was 4 cm.

20.2.4 Statistical Analyses

All data represented as means ± S.D. Statistical analyses were performed using the statistical package SPSS for Windows (version 19.0; SPSS, Chicago, IL). SO_{2IC} during CET_MOD and CET_HVY were compared by two-way ANOVA, with time and intensity as main effects. The Dunnett post hoc test was performed to examine time changes, and the paired *t*-test was used to compare between CET_MOD and CET_HVY (Fig. 20.1). SO_{2IC} during RIET, CET_MOD, and CET_HVY were compared by the paired *t*-test, at the similar $\%V_{Epeak-RIET}$ (Fig. 20.2). Values of $P<0.05$ were considered significant.

20.3 Results

At VO_{2peak} during RIET, V_E, VO_2, and SO_{2IC} were 126 ± 22 $L \cdot min^{-1}$, 51.7 ± 9.1 $mL \cdot kg^{-1} \cdot min^{-1}$, and 54.6 ± 14.0 %, respectively.

V_E increased progressively as exercise time increased and was significantly higher during CET_HVY than CET_MOD. In contrast, SO_{2IC} decreased from the start of both CET_MOD and CET_HVY and changed little from 2 to 6 min (Fig. 20.1). The amplitude of SO_{2IC} was significantly higher during CET_HVY than CET_MOD. The final values of V_E, $\%V_{Epeak-RIET}$, VO_2, and SO_{2IC} were

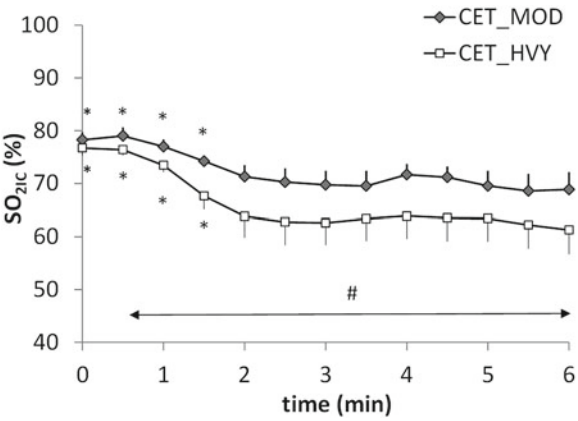

Fig. 20.1 SO_{2IC} kinetics during CET_MOD and CET_HVY. * versus 6 min, # versus MOD, $P < 0.05$

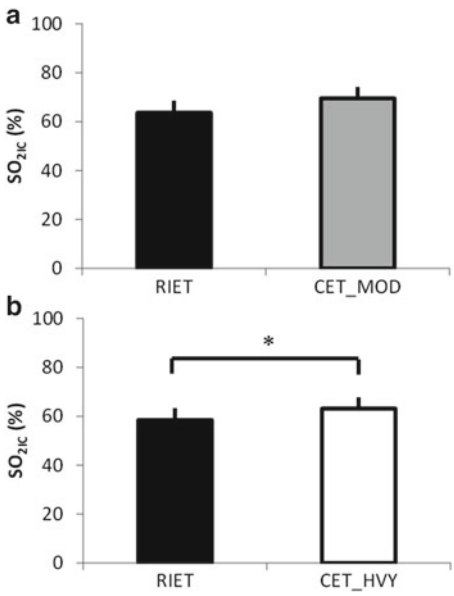

Fig. 20.2 SO_{2IC} at the similar $\%VE_{peak-RIET}$ during RIET and CET_MOD (**a**) and during RIET and CET_HVY (**b**). * RIET versus CET_HVY

59.1 ± 4.6 L · min^{-1}, 46.1 ± 6.9 %, 32.3 ± 6.1 mL · kg^{-1} · min^{-1}, and 68.9 ± 7.3 % during CET_MOD and 103 ± 19 L · min^{-1}, 79.3 ± 11.5 %, 47.9 ± 6.9 mL · kg^{-1} · min^{-1}, and 61.3 ± 10.2 % during CET_HVY, respectively.

In comparison with the similar $\%V_{Epeak-RIET}$ between RIET (48.3 ± 3.3 %) and CET_MOD at the peak of $\%V_{Epeak-RIET}$ (48.4 ± 3.2 %), SO_{2IC} tend to be higher during CET_MOD than RIET, but not significant (Fig. 20.2a). On the other hand, in comparison between RIET (81.0 ± 12.2 %) and CET_MOD at the peak of $\%V_{Epeak-RIET}$ (81.0 ± 11.7 %), SO_{2IC} was significantly higher during CET_HVY than RIET (Fig. 20.2b).

20.4 Discussion

We examined SO_{2IC} kinetics during constant-load exercise and compared SO_{2IC} at similar $\%V_{Epeak-RIET}$ between RIET and CET. The main findings of this study are that SO_{2IC} was little changed over 2 min during both CET_MOD and CET_HVY and that the level of SO_{2IC} was different between RIET and CET_HVY despite similar $\%V_{Epeak-RIET}$. These findings suggest that the decrease of SO_{2IC} is not induced by increased O_2 consumption of the accessory respiratory muscles under the measurement area. In this study, neither blood flow nor O_2 supply in IC was measured, but a decrease in them would mainly result in a decrease in SO_{2IC}.

O_2 saturation is thought to represent the balance between O_2 supply and consumption. We believe that O_2 consumption in the respiratory and accessory respiratory muscles was increased because a rise of V_E led to high work of breathing [9]. However, it would be unlikely that the increase in O_2 consumption caused deoxygenation in these muscles, at least the intercostal muscles. Rather, we assume that SO_{2IC} was more related to O_2 supply changes, which could be effected by sympathetic nerve activity-induced vasoconstriction, which was seen in organs and resting muscles during high-intensity exercise [10, 11]. Blood flow in working muscle was relatively maintained or increased by vasodilation effects such as NO [12], but blood flow and O_2 supply in resting muscles were decreased [11]. Although neither autonomic nerve activity nor blood flow in the resting muscle was measured in this study, previous studies have reported that during high-intensity exercise, the resting muscle deoxygenation increased gradually [13], similar to SO_{2IC} in this study. Hence, in our study where O_2 saturation in IC and the resting muscle were simultaneously monitored, these values were similar during both incremental and decremental exercise (unpublished data). Furthermore, an isocapnic hyperpnea at rest did not deoxygenate IC [14]. Therefore, it was unclear why SO_{2IC} showed different kinetics between protocols, but it was likely that the change was more related to the O_2 supply than to the O_2 consumption.

In summary, the present study demonstrated that SO_{2IC} during CET_HVY did not change despite V_E increasing and that SO_{2IC} during CET_HVY was different from that during RIET in the same V_E. These results imply that a decrease in SO_{2IC} is caused by a decrease of the O_2 supply, rather than an increase of the O_2 consumption.

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