Effects of Maximal Interval Training on Arterial Oxygen Desaturation and Ventilation during Heavy Exercise

Motohiko MIYACHI and Keisho KATAYAMA*

Department of Health and Sports Sciences, Kawasaki University of Medical Welfare, Kurashiki, 701–0193 Japan; and
Research Center of Health and Sports, Nagoya University, Chikusa-ku, Nagoya, 464–8601 Japan

Abstract: The purpose of the present study was to clarify longitudinally the effects of exercise training on arterial oxygen saturation (SaO₂) and ventilation during heavy exercise. A group of six subjects (training group) volunteered to train four times a week for 12 weeks. Each training session consisted of five 3-min periods of exercise on a cycle ergometer at a power output of 100% maximal O₂ uptake (VO₂max), interspersed with 2-min recovery period cycling at 50% VO₂max. During the training, VO₂max, SaO₂, the ventilatory equivalent for oxygen (VE/VO₂), and the endtidal partial pressure of O₂ (PETO₂) during heavy exercise were measured periodically. The same parameters were measured simultaneously in another group of five subjects (control group) who led normal lives. Maximal interval training increases VO₂max, with little change in VE,max and pulmonary functions at rest. The training decreased PETO₂, VE/VO₂, and SaO₂ during heavy exercise. SaO₂ is significantly related to VE/VO₂ (r²=0.49). These results suggest that less hyperventilatory response to exercise occurs with progress in physical training because the adaptability of ventilatory capacity is less than that of aerobic work capacity, which half induces arterial O₂ desaturation during heavy exercise. PETO₂ as well as VE/VO₂ and VO₂max did not change anymore after the 6th week, nevertheless SaO₂ kept decreasing up to the last 2 weeks. In addition, when the SAO₂-VE/VO₂ plot was compared between the two groups, the regression line of the training group was steeper than that of the control group; i.e., compared at a lower level of VE/VO₂ (−30 ml · ml⁻¹), the SaO₂ of the trained subjects exercising at a higher VO₂ level was lower than that of the control subjects. Predominance of less hyperventilation and another factor, increased A-aDO₂, in the genesis of arterial hypoxemia and O₂ desaturation may be dependent upon VO₂ levels in heavy exercise and the state of training. [Japanese Journal of Physiology, 49, 401–407, 1999]

Key words: arterial oxygen saturation, maximal exercise, physical training, pulse oximetry, ventilation.

Several recent studies [1–10] have demonstrated that a reduction of arterial O₂ saturation (SaO₂) during heavy exercise occurs in trained endurance athletes. Williams et al. [10] demonstrated that there was a negative correlation between aerobic work capacity (maximal O₂ uptake: VO₂max) and SaO₂ during heavy exercise. These findings suggest that the improvement of aerobic work capacity with progress in training must be associated with the reduction of SaO₂ (arterial O₂ desaturation) during heavy exercise. Rowell et al. [11] were concerned with longitudinally assessing the effect of training on arterial O₂ desaturation, and not with investigating an assessment of the mechanisms. Dempsey et al. [2] and Powers et al. [7] reported that arterial O₂ desaturation during heavy exercise was observed in subjects with lower alveolar O₂ partial pressure (PAlO₂) and less hyperventilation. In contrast, Powers et al. [8] and Hopkins and McKenzie [4] con-
cluded that an increase in alveolar-arterial $PO_2$ difference (A-a$DO_2$) plays a major role in the genesis of arterial $O_2$ desaturation during maximal exercise, with ventilation providing a lesser contribution. Recent studies [1, 3, 5, 6] suggest that about half of the arterial hypoxemia and $O_2$ desaturation occurring during maximal exercise may be explained by the less hyperventilation, and the other half due to the increase in A-a$DO_2$. Although the mechanisms of less hyperventilation during heavy exercise are unclear, it is well known that endurance athletes breathe less than non-athletes at comparable exercise intensities [12, 13]. Furthermore, previous studies [2, 10, 14] have hypothesized that the increase in A-a$DO_2$ during heavy exercise is due primarily to diffusion limitations caused by very rapid red blood cell transit time in the pulmonary capillary bed of the trained athlete at the time of maximal cardiac output.

Therefore, we hypothesize that less hyperventilatory response to heavy exercise and increase in A-a$DO_2$ occur with progress in physical training, which induces arterial $O_2$ desaturation during heavy exercise. To assess the propriety of our hypothesis, we clarified the longitudinal changes in $V\dot{O}_{2,\max}$ ventilatory parameters at rest, $Sa_o$, and ventilation during heavy exercise in a 12-week program of maximal interval training.

MATERIALS AND METHODS

Subjects. Eleven subjects volunteered to participate in the experiments. All were healthy adult males with no history of lung disease, who actively participated in recreational sports. Descriptive data (mean ± standard deviation) are as follows: age = 23 ± 4 years, body weight = 68.5 ± 5.5 kg, height = 173.9 ± 3.4 cm. The subjects were informed of the nature of the experiments and gave informed consent before testing. Each subject was assigned to one of two groups based on his $V\dot{O}_{2,\max}$ and $Sa_o$ during heavy exercise.

Procedure. One group of six subjects (training group) underwent cycle exercise training for 12 weeks. $V\dot{O}_{2,\max}$ and maximal ventilation ($V\dot{E}_{\text{max}}$) were measured every week during the training period. $Sa_o$ and ventilation during exercise at 90% $V\dot{O}_{2,\max}$ were measured every other week. These parameters and pulmonary functions at rest were also measured before and immediately after the training program. The same parameters were measured simultaneously in the other group of five subjects (control group) who led normal lives.

Training program. After the completion of initial tests, the training group subjects attended four training sessions per week from Tuesday through Friday for 12 weeks (a total of 48 sessions). They performed five 3-min periods of exercise on a cycle ergometer (Monark 818E, Sweden) at a power output of 100% $V\dot{O}_{2,\max}$ (60 rpm). Exercise intervals were interspersed with 2-min recovery periods consisting of cycling at 50% $V\dot{O}_{2,\max}$. $V\dot{O}_{2,\max}$ was determined every week on Monday to maintain a constant training stimulus, which was achieved by increasing the power output on the cycle ergometer.

$V\dot{O}_{2,\max}$ measurement. $V\dot{O}_{2,\max}$ (STPD) was determined every Monday by an intermittent incremental load test. All subjects underwent several work sessions on a bicycle ergometer (pedaling frequency = 60 rpm) with sufficient rest periods between sessions. The initial work load was 120 W and was increased by 15–60 W each session until $V\dot{O}_2$ had leveled off. At submaximal intensity, the subjects exercised for 10 min. They were considered exhausted (usually after five to six rides) when they could no longer ride for at least 3 min. Subjects breathed through a low-resistance two-way valve (Hans Rudolph #2700, USA), and expired air was collected in Douglas bags during the last minute of each session. Expired $O_2$ and $CO_2$ concentrations were measured by mass spectrometry (Westron MGA1200, Japan), and gas volume was determined using a dry gas meter (Shinagawa Dev. NDS-2A-T, Japan). Heart rate (HR), during the last minute of each session, was recorded with an electrocardiograph (Nihon Kohden WEP 7404, Japan). The main criterion for determining $V\dot{O}_{2,\max}$ was a leveling off or decrease in $V\dot{O}_2$ with an increasing work load. Other criteria were volitional exhaustion, a heart rate of more than 190 beats·min⁻¹ and a respiratory exchange ratio greater than unity. The peak HR and $V\dot{E}$ (BTPS) values from all rides were established as HR$\text{max}$ and $V\dot{E}_{\text{max}}$, respectively.

Measurement of $Sa_o$ and ventilation during heavy exercise. The subjects exercised for 5 min on a cycle ergometer at a power output of 90% $V\dot{O}_{2,\max}$ (60 rpm). $Sa_o$, $V\dot{O}_2$, $V\dot{E}$, the ventilatory equivalent for oxygen ($V\dot{E}/V\dot{O}_2$, BTPS/STPD), and the end-tidal partial pressure of $O_2$ ($PETO_2$) were monitored during the last 1 min of the 5-min exercise. $Sa_o$ was estimated with a pulse oximeter (Nihon Kohden OLV1200) placed on the tip of the right forefinger. The accuracy of pulse oximetry has been proven by several studies [15–19]. The subjects were advised not to strain while moving their right hands so that pulse waves and $Sa_o$ could be monitored as accurately as possible. $V\dot{O}_2$, $V\dot{E}$, and $V\dot{E}/V\dot{O}_2$ were measured by the methods described above. To measure $PETO_2$, a small amount of expired gas (60 ml·min⁻¹) was sampled continuously by the
gas analyzer (Westron MGA1200) through a sampling tube connected to a breathing mask. The quantities of changes in \( S_A O_2 \) (\( \Delta S_A O_2 \)) and \( V E / V O_2 \) (\( \Delta V E / V O_2 \)) during the training period were calculated (\( \Delta S_A O_2 = S_A O_2 \) at the 12th week - \( S_A O_2 \) at initial test, \( \Delta V E / V O_2 = \dot{V} E / \dot{V} O_2 \) at the 12th week - \( \dot{V} E / \dot{V} O_2 \) at initial test).

**Measurement of pulmonary functions.** Vital capacity (VC) and peak flow (PF) were measured by pneumotachography (Fukuda Denshi SP300, Japan). The maximal voluntary ventilation for 12s (MVV12) was measured by the Douglas bag method with the subject in standing position. The pulmonary diffusing capacity for CO (DL\(\text{CO} \)) was measured by the single-breath method (Sensor Medics MMC4400 tc, USA) [20].

**Statistical analysis.** The values were expressed as the mean and standard deviation (SD). The changes in all parameters during the training period were compared using two-way analysis of variance (ANOVA) with repeated measurements. The differences between the two groups were analyzed by Sheffe’s F-test. The differences in each group were analyzed by the paired t-test. The relationships among several parameters of each group were determined by simple regression analysis. The differences of two regression slopes and intercepts were tested using t-statistics [21]. The level of significance was established at \( p<0.05 \).

**RESULTS**

There were no significant changes in body weight in any of the subjects during the 12-week training period (from 68.5±5.2 to 67.6±4.2 kg). Changes in \( \dot{V} O_2 \text{max} \) and \( \dot{V} E \text{max} \) during the training are shown in Fig. 1. There were no significant differences in \( \dot{V} O_2 \text{max} \) and \( \dot{V} E \text{max} \) between the training group and control group before the training. \( \dot{V} O_2 \text{max} \) plateaued following a linear increase to the 5th week in the training group while there was no statistically significant change in the control group. \( \dot{V} O_2 \text{max} \) was significantly higher in the training group than in the control group (\( F=7.14, p<0.05 \) in ANOVA). The total increase in \( \dot{V} O_2 \text{max} \) for the six subjects of the training group averaged 10.7 ml·min\(^{-1} \)·kg\(^{-1} \) (19.1%, from 50.9±5.63 to 61.6±4.06 ml·min\(^{-1} \)·kg\(^{-1} \)). \( \dot{V} E \text{max} \) in the training group increased slightly during the early weeks of training and the total increase for the six subjects averaged 0.19 l·min\(^{-1} \)·kg\(^{-1} \) (8.2%, from 2.33±0.18 to 2.52±0.27 l·min\(^{-1} \)·kg\(^{-1} \)). \( \dot{V} E \text{max} \) in the training group was significantly higher than that in the control group (\( F=5.39, p<0.05 \)). There was no change in HR\text{max} during the training period in either group.

Changes in \( S_A O_2 \), \( \dot{V} E / \dot{V} O_2 \), and \( \text{PET}_O_2 \) during the training period are shown in Fig. 2. There were no significant differences in \( S_A O_2 \), \( \dot{V} E / \dot{V} O_2 \), and \( \text{PET}_O_2 \) between the training group and control group during heavy exercise prior to the training. \( S_A O_2 \) during heavy exercise decreased with the 12 weeks of training (3.1%, from 95.9 to 92.8%) and was significantly lower in the training group than in the control group (\( F=14.6, p<0.01 \)). \( \Delta S_A O_2 \) in the training group (−3.1±0.7%) was significantly larger than that in the control group (0.5±0.6%). The changes in \( \text{PET}_O_2 \) and \( \dot{V} E / \dot{V} O_2 \) were similar to those for \( S_A O_2 \), \( \text{PET}_O_2 \) as well as \( \dot{V} E / \dot{V} O_2 \) did not decrease anymore after the 6th week; nevertheless, \( S_A O_2 \) kept decreasing up to the last week. \( \text{PET}_O_2 \) and \( \dot{V} E / \dot{V} O_2 \) levels in the training group were significantly lower than those in the control group (\( F=5.50, p<0.05; F=5.45, p<0.05 \), respectively), while \( \Delta \dot{V} E / \dot{V} O_2 \) in the training group (−7.4±4.6 ml/ml) was significantly larger than that in the control group (2.1±2.6 ml/ml).

Table 1 shows pulmonary function test data at rest in both groups. There were no significant differences in VC, PF, MVV12, and DL\(\text{CO} \) between the two groups before and after the training period.

**Fig. 1. Changes in \( \dot{V} O_2 \text{max} \) (upper) and \( \dot{V} E \text{max} \) (lower) with 12 weeks of maximal interval training. * Shows significant difference from the value of the control group. † Shows significant difference from the preceding value in each group.**
Figure 3 shows relationships among $\dot{V}O_2\text{max}$, $\dot{V}E/\dot{V}O_2$, and $\text{Sa}_O_2$, $\text{So}_2$, and $\dot{V}E/\dot{V}O_2$ during heavy exercise were negatively related to $\dot{V}O_2\text{max}$. For $\text{Sa}_O_2$, $r = -0.70$, $p<0.001$ ($n=77$ based on 11 subjects×7 measurements, Fig. 3 upper); and for $\dot{V}E/\dot{V}O_2$, $r = -0.57$, $p<0.001$ (Fig. 3 middle). There were significant correlations between $\text{Sa}_O_2$ and $\dot{V}E/\dot{V}O_2$ ($n=77$, $r=0.71$, $p<0.001$, Fig. 3 lower) during heavy exercise, and between $\Delta \text{Sa}_O_2$ and $\Delta \dot{V}E/\dot{V}O_2$ ($n=11$, $r=0.79$, $r^2=0.63$, $p<0.01$). When these plots among $\dot{V}O_2\text{max}$, $\dot{V}E/\dot{V}O_2$, and $\text{Sa}_O_2$ were compared between groups, regression slopes of the training group significantly differed from those of the control group (Fig. 3).

**DISCUSSION**

The principal findings of the present study are: 1) maximal interval training increased $\dot{V}O_2\text{max}$, with little change in $\dot{V}E\text{max}$ and ventilatory parameters at rest; 2) training decreased $\text{PET}_O_2$, $\dot{V}E/\dot{V}O_2$, and $\text{Sa}_O_2$ during heavy exercise; 3) $\text{PET}_O_2$ as well as $\dot{V}E/\dot{V}O_2$ did not decrease anymore after the 6th week, nevertheless, $\text{Sa}_O_2$ kept decreasing up to the last 2 weeks; 4) $\text{Sa}_O_2$ or $\Delta \text{Sa}_O_2$ was significantly related to $\dot{V}E/\dot{V}O_2$ ($r^2=0.49$) or $\Delta \dot{V}E/\dot{V}O_2$ ($r^2=0.63$), respectively; and 5) the regression slope between $\text{Sa}_O_2$ and $\dot{V}E/\dot{V}O_2$ of the training group was larger than that of the control group.

**Training state, $\dot{V}O_2\text{max}$, and $\text{Sa}_O_2$** $\dot{V}O_2\text{max}$ and the absolute intensity of exercise at 90% $\dot{V}O_2\text{max}$ significantly increased with training, while $\text{Sa}_O_2$ during heavy exercise decreased. $\text{Sa}_O_2$ after training (92.8±1.1%) in the present study was slightly higher than that of endurance athletes (88–92%) described in previous studies [2, 4, 7, 9, 10] because there were differences in the training state or $\dot{V}O_2\text{max}$ of the subjects. If the $\dot{V}O_2\text{max}$ of our subjects reached a higher level (e.g., ≥65 ml·min⁻¹·kg⁻¹), $\text{Sa}_O_2$ during heavy exercise could have decreased to less than 92%. There was a negative relationship between $\dot{V}O_2\text{max}$ and $\text{Sa}_O_2$ during heavy exercise.

![Fig. 2. Changes in $\text{Sa}_O_2$ (upper), $\text{PET}_O_2$ (middle), and $\dot{V}E/\dot{V}O_2$ (lower) during heavy exercise over 12 weeks of maximal interval training. * Shows significant difference from the value of the control group. † Shows significant difference from the preceding value in each group.](image)

**Table 1. Resting pulmonary functions before and after the training period.**

<table>
<thead>
<tr>
<th></th>
<th>Before training</th>
<th>After training</th>
</tr>
</thead>
<tbody>
<tr>
<td>VC (l)</td>
<td>Training group</td>
<td>4.87±0.51</td>
</tr>
<tr>
<td></td>
<td>Control group</td>
<td>5.12±0.42</td>
</tr>
<tr>
<td>PF (l·s⁻¹)</td>
<td>Training group</td>
<td>9.96±1.11</td>
</tr>
<tr>
<td></td>
<td>Control group</td>
<td>9.89±1.63</td>
</tr>
<tr>
<td>MVV₁₅/BW (l·min⁻¹·kg⁻¹)</td>
<td>Training group</td>
<td>2.93±0.30</td>
</tr>
<tr>
<td></td>
<td>Control group</td>
<td>2.75±0.33</td>
</tr>
<tr>
<td>DLCO (ml·min⁻¹·mmHg⁻¹)</td>
<td>Training group</td>
<td>36.7±3.3</td>
</tr>
<tr>
<td></td>
<td>Control group</td>
<td>35.7±3.3</td>
</tr>
</tbody>
</table>

VC, vital capacity; PF, peak flow; MVV₁₅/BW, maximal voluntary ventilation for 15 s per body weight; DLCO, pulmonary diffusing capacity for carbon monoxide.
Less hyperventilation and arterial O₂ desaturation. During heavy exercise, $P_{ETO₂}$, which reflected alveolar $PO₂$ and $\dot{V}E/\dot{V}O₂$, decreased with training. Several studies have reported that endurance athletes breathe less at a given metabolic rate than untrained subjects during exercise [12, 13]. These results suggest that endurance training induces a lower alveolar $PO₂$ and less hyperventilation during heavy exercise. The present study showed significant relationships between $\dot{V}E/\dot{V}O₂$ and $S_{A0₂}$ ($r^2=0.51$, $p<0.001$, Fig. 3 lower), and $D\dot{S}O₂$ and $D\dot{V}E/\dot{V}O₂$ ($r^2=0.63$, $p<0.01$). The relationship indicates that about half of the arterial $O₂$ desaturation during heavy exercise can be accounted for by the less hyperventilatory response. Previous cross-sectional studies demonstrated similar correlations among $S_{A0₂}$ and $\dot{V}E/\dot{V}O₂$ [1, 3, 5, 6].

Another factor of arterial O₂ desaturation. It is interesting that $P_{ETO₂}$ as well as $\dot{V}E/\dot{V}O₂$ did not decrease anymore after the 6th week; nevertheless, $S_{A0₂}$ kept decreasing up to the last 2 weeks (Fig. 2). These changes over the course of time imply that, specifically in the latter half of the training period, training-induced arterial O₂ desaturation may be associated with another factor (e.g., an increase in A-aDO₂). Hopkins and McKenzie [4] reported that $P_{A0₂}$ was not significantly related to $P_{A0₂}$ ($r=0.51$, $p=0.08$) but was closely related to A-aDO₂ ($r=0.98$, $p<0.001$) during heavy exercise. Furthermore, Powers et al. [8] confirmed this and concluded that an increase in A-aDO₂ plays a major role in the genesis of arterial hypoxemia and desaturation during maximal exercise, while a less hyperventilatory response has a minor role. They used only endurance athletes as subjects, with $\dot{V}O₂_{max} \geq 65 \text{ ml \cdot min}^{-1} \cdot \text{kg}^{-1}$ [4, 8], whereas our subjects’ $\dot{V}O₂_{max}$ finally reached $61.6 \pm 4.1 \text{ ml \cdot min}^{-1} \cdot \text{kg}^{-1}$ after training for 12 weeks ($50.9 \pm 5.63 \text{ ml \cdot min}^{-1} \cdot \text{kg}^{-1}$ at initial test). Rice et al. [22] suggested that $P_{A0₂}$ is strongly associated with inadequate hyperventilation when exercising at a lower $\dot{V}O₂$ level, while an increased A-aDO₂ was the greater determinant of hypoxemia at a higher $\dot{V}O₂$ level. The predominance of less hyperventilation and increased A-aDO₂ in the genesis of arterial hypoxemia and $O₂$ desaturation may be dependent upon $\dot{V}O₂$ levels during exercise. In the present study, the regression line between $S_{A0₂}$ and $\dot{V}E/\dot{V}O₂$ of the training group was steeper than that of the control group (Fig. 3 lower); i.e., compared at lower level of $\dot{V}E/\dot{V}O₂$ (30 ml · min⁻¹), $S_{A0₂}$ of the trained subjects exercising at a higher $\dot{V}O₂$ level was lower than that of the control subjects. Furthermore, $\dot{V}E/\dot{V}O₂$ of the trained subjects exercising at a lower $\dot{V}O₂$ level (45–55 ml · min⁻¹ · kg⁻¹) was lower than that of the control subjects exer-

---

Fig. 3. Relationships among $\dot{V}E/\dot{V}O₂$ and $S_{A0₂}$ during heavy exercise and $\dot{V}O₂_{max}$.

heavy exercise, which corresponds with the results of Williams et al. [10]. The relationship suggests dependence of the $\dot{V}O₂$ level on arterial $O₂$ desaturation during heavy exercise. However, when the relationship was compared between the control and training groups, the slope of the regression line between $\dot{V}O₂_{max}$ and $S_{A0₂}$ in the training group was larger than that in the control group. Therefore, the arterial $O₂$ desaturation during heavy exercise is considered to be dependent on not only the $\dot{V}O₂$ level attained during exercise but also other adaptations with progress in training, described as follows.
cising at the same $\dot{V}O_2$ level (Fig. 3 middle). In subjects exercising at a higher $\dot{V}O_2$, arterial $O_2$ desaturation, if it occurs, might be due more to increased $A-aDO_2$ than to reduced ventilatory response, while in subjects exercising at a lower $\dot{V}O_2$, the latter factor might be a greater determinant of desaturation.

**Mechanisms of less hyperventilation.** The interval training resulted in reductions of $\dot{V}E/\dot{V}O_2$ during heavy exercise, while its mechanisms are still unclear. Casaburi et al. [23] showed that the decrease in ventilation after training is due to a decrease in the metabolic production of $CO_2$ resulting from a reduction in blood lactate concentration. Other studies [12, 13] suggested that endurance athletes, endowed with low ventilatory chemoresponses, breathe less than non-athletes at comparable exercise intensities. Recently, it was shown that low ventilatory chemoresponsiveness contributes to less hyperventilation, hypoxemia and arterial $O_2$ desaturation during heavy exercise [1, 3, 5]. Therefore, the less hyperventilation during heavy exercise may be partly associated to adaptations in ventilatory chemoresponses in the present training.

Dempsey [14] has hypothesized that arterial desaturation and less hyperventilation during heavy exercise in trained athletes are an indication that critical aspects of the pulmonary system have not adapted appropriately to the increased metabolic demand. In the present study, $\dot{V}O_2\max$ increased 19.1% due to the training, while $\dot{V}E\max$ increased only 8.2%. VC, PF, MVV, and DLCO did not significantly change during the 12 weeks of training. Reuschlein et al. [24] observed that 5 months of endurance exercise training has no effect on lung diffusion capacity or other ventilatory parameters. These findings support the hypothesis of Dempsey [14], and suggest that reduced hyperventilatory response to heavy exercise may be related to lower adaptation of the ventilatory system to physical training.

The present study offers no definitive explanations as to why the training had little effect on pulmonary functions, but it may be related to the training stimulus. Previous studies [25, 26] have shown that specific respiratory muscle training increases ventilatory functions. Furthermore, it is well known that the DLCO of permanent residents at high altitude is higher than that of persons living at sea level [27]. These findings suggest that respiratory muscle training and long-term exposure to high altitudes (hypoxia) may stimulate the pulmonary system. The stimulus provided by the maximal interval training in this study may not have been sufficient to change the pulmonary function since the $\dot{V}E$ during a training session barely reached 70–80% of $\dot{V}E\max$ or MVV at the fifth interval period, and alveolar $PO_2$ was more than 100 mm Hg.

**Methodology.** One methodological concern is the use of the pulse oximeter to measure $SaO_2$. Accurate measurement of $SaO_2$ or arterial gases has required invasive sampling from the artery. On the other hand, the accuracy of pulse oximetry has been proven by several studies [15–19]. A 23 subject study by Aoyagi showed that there was a high correlation between the $SaO_2$ of arterial blood samples determined by a CO-oximeter (Corning 2500, USA) and $SaO_2$ estimated by OLV1200 ($r=0.97$, $p<0.001$, $n=73$), and that the standard error of estimate (SEE) of the OLV 1200 at rest was 1.58% in $SaO_2$ values from 70 to 100% ([19] and personal communication). To evaluate the validity of OLV 1200 during exercise at 90% $\dot{V}O_2\max$, and confirm Aoyagi's study, the $SaO_2$ of five subjects (10 blood samples) was estimated by an OLV1200 and compared with that determined by a CO-oximeter (Corning 2500). The correlation was significant ($r=0.89$, $p<0.001$), and the SEE was 0.82%. The difference in $SaO_2$ between the two groups after the 4th week (2–3%) was larger than the 2 SEE (1.64%). These results show that the validity of the pulse oximeter meets the level to assess the propriety of our hypothesis. Furthermore, it was necessary to avoid the risk to subjects of repeated invasive measurements of $SaO_2$ for 12 weeks. Therefore, pulse oximetry was the most suitable method for the present study.

**Summary.** Maximal interval training increases $\dot{V}O_2\max$ with little change in $\dot{V}E\max$ and pulmonary functions at rest. Training decreases $PET_{CO_2}$, $\dot{V}E/\dot{V}O_2$, and $SaO_2$ during heavy exercise. The decreases in $SaO_2$, or $\Delta SaO_2$, are significantly related to the decreases in $\dot{V}E/\dot{V}O_2$ ($r^2=0.49$) and $\Delta \dot{V}E/\dot{V}O_2$ ($r^2=0.63$), respectively. On the other hand, $PET_{CO_2}$, as well as $\dot{V}E/\dot{V}O_2$ did not decrease anymore after the 4th week; nevertheless, $SaO_2$ kept decreasing up to the last 2 weeks. In addition, the regression line between $SaO_2$ and $\dot{V}E/\dot{V}O_2$ of the training group was steeper than that of the control group: i.e., compared to a lower level of $\dot{V}E/\dot{V}O_2$ (~30 ml·min⁻¹), $SaO_2$ of the trained subjects exercising at a higher $\dot{V}O_2$ level was lower than that of the control subjects. These results suggest that less hyperventilatory response to exercise and another factor, likely occurring with progress in physical training, are involved in the development of arterial $O_2$ desaturation during heavy exercise. The predominance of less hyperventilation and another factor, probably increased $A-aDO_2$, in the genesis of arterial hypoxemia and $O_2$ desaturation may be dependent upon the $\dot{V}O_2$ levels in heavy exercise and the state of training.
We are grateful to Dr. Miharu Miyamura (Nagoya University) for his advice, and Mr. Fred Furuta and Michael Kremenik (Kawasaki University of Medical Welfare) for their generous help in writing the English manuscript. This study was supported by Grants-in-Aid for Scientific Research from the Japanese Ministry of Education, Science, Sports, and Culture (Nos. 08780106 and 09308002).

REFERENCES