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 O₂ saturation (Sa_{O₂}) 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 ($\dot{V}O_{2\max}$), interspersed with 2-min recovery period cycling at 50% $\dot{V}O_{2\max}$. During the training, $\dot{V}O_{2\max}$, Sa_{O₂}, the ventilatory equivalent for oxygen ($\dot{V}E/\dot{V}O_2$), and the end-tidal partial pressure of O₂ (PET_{O₂}) 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 $\dot{V}O_{2\max}$, with little change in $\dot{V}E_{\max}$ and pulmonary functions at rest. The training decreased PET_{O₂}, $\dot{V}E/\dot{V}O_2$, and Sa_{O₂} during heavy exercise. Sa_{O₂} is significantly related to $\dot{V}E/\dot{V}O_2$ ($r^2=0.49$). These results suggest that less hyper-

ventilatory 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. PET_{O₂} as well as $\dot{V}E/\dot{V}O_2$ and $\dot{V}O_{2\max}$ did not change anymore after the 6th week, nevertheless Sa_{O₂} kept decreasing up to the last 2 weeks. In addition, when the Sa_{O₂}- $\dot{V}E/\dot{V}O_2$ plot was compared between the two groups, the regression line of the training group was steeper than that of the control groups; i.e., compared at a lower level of $\dot{V}E/\dot{V}O_2$ (~30 ml · ml⁻¹), the Sa_{O₂} of the trained subjects exercising at a higher $\dot{V}O_2$ 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 $\dot{V}O_2$ 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 (Sa_{O₂}) 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: $\dot{V}O_{2\max}$) and Sa_{O₂} during heavy exercise. These findings suggest that the improvement of aerobic work capacity with progress in training must be associated with the reduction of Sa_{O₂} (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 (PA_{O₂}) and less hyperventilation. In contrast, Powers *et al.* [8] and Hopkins and McKenzie [4] con-

Received on February 18, 1999; accepted on July 26, 1999

Correspondence should be addressed to: Motohiko Miyachi, Kawasaki University of Medical Welfare, Kurashiki, 701-0193 Japan. Tel: +81-086-462-1111 ext 4079, Fax: +81-086-464-1109, E-mail: miyachi@mw.kawasaki-m.ac.jp

cluded that an increase in alveolar-arterial PO_2 difference ($A-aDO_2$) 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 $A-aDO_2$. 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 $A-aDO_2$ 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 $A-aDO_2$ 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 $\dot{V}O_{2\max}$, ventilatory parameters at rest, SA_{O_2} 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 $\pm$ 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 $\dot{V}O_{2\max}$ and SA_{O_2} during heavy exercise.

Procedure. One group of six subjects (training group) underwent cycle exercise training for 12 weeks. $\dot{V}O_{2\max}$ and maximal ventilation ($\dot{V}E_{\max}$) were measured every week during the training period. SA_{O_2} and ventilation during exercise at 90% $\dot{V}O_{2\max}$ 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% $\dot{V}O_{2\max}$ (60 rpm). Exercise intervals were interspersed with 2-min recovery periods consisting of cycling at 50% $\dot{V}O_{2\max}$. $\dot{V}O_{2\max}$ was determined every week on Monday to maintain a constant training stimulus, which was achieved by increasing the power output on the cycle ergometer.

$\dot{V}O_{2\max}$ measurement. $\dot{V}O_{2\max}$ (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 $\dot{V}O_2$ 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 gas 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 $\dot{V}O_{2\max}$ was a leveling off or decrease in $\dot{V}O_2$ with an increasing work load. Other criteria were volitional exhaustion, a heart rate of more than $190 \text{ beats} \cdot \text{min}^{-1}$ and a respiratory exchange ratio greater than unity. The peak HR and $\dot{V}E$ (BTPS) values from all rides were established as $HR_{\max}$ and $\dot{V}E_{\max}$, respectively.

Measurement of SA_{O_2} and ventilation during heavy exercise. The subjects exercised for 5 min on a cycle ergometer at a power output of 90% $\dot{V}O_{2\max}$ (60 rpm). SA_{O_2} , $\dot{V}O_2$, $\dot{V}E$, the ventilatory equivalent for oxygen ($\dot{V}E/\dot{V}O_2$, BTPS/STPD), and the end-tidal partial pressure of O_2 (PET_{O_2}) were monitored during the last 1 min of the 5-min exercise. SA_{O_2} 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_2} could be monitored as accurately as possible. $\dot{V}O_2$, $\dot{V}E$, and $\dot{V}E/\dot{V}O_2$ were measured by the methods described above. To measure PET_{O_2} , a small amount of expired gas ($60 \text{ ml} \cdot \text{min}^{-1}$) was sampled continuously by the

gas analyzer (Westron MGA1200) through a sampling tube connected to a breathing mask. The quantities of changes in Sa_{O_2} (ΔSa_{O_2}) and $\dot{V}E/\dot{V}O_2$ ($\Delta \dot{V}E/\dot{V}O_2$) during the training period were calculated ($\Delta Sa_{O_2} = Sa_{O_2}$ at the 12th week $- Sa_{O_2}$ at initial test, $\Delta \dot{V}E/\dot{V}O_2 = \dot{V}E/\dot{V}O_2$ 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 SP-300, Japan). The maximal voluntary ventilation for 12 s (MVV_{12}) was measured by the Douglas bag method with the subject in standing position. The pulmonary diffusing capacity for CO (DL_{CO}) was measured by the single-breath method (Sensor Medics MMC 4400 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 $\dot{V}O_{2\max}$ and $\dot{V}E_{\max}$ during the training are shown in Fig. 1. There were no significant differences in $\dot{V}O_{2\max}$ and $\dot{V}E_{\max}$ between the training group and control group before the training. $\dot{V}O_{2\max}$ plateaued following a linear increase to the 5th week in the training group while there was no statistically significant change in the control group. $\dot{V}O_{2\max}$ was significantly higher in the training group than in the control group ($F=7.14$, $p < 0.05$ in ANOVA). The total increase in $\dot{V}O_{2\max}$ for the six subjects of the training group averaged $10.7 \text{ ml} \cdot \text{min}^{-1} \cdot \text{kg}^{-1}$ (19.1%, from 50.9 ± 5.63 to $61.6 \pm 4.06 \text{ ml} \cdot \text{min}^{-1} \cdot \text{kg}^{-1}$). $\dot{V}E_{\max}$ in the training group increased slightly during the early weeks of training and the total increase for the six subjects averaged $0.19 \text{ l} \cdot \text{min}^{-1} \cdot \text{kg}^{-1}$ (8.2%, from 2.33 ± 0.18 to $2.52 \pm 0.27 \text{ l} \cdot \text{min}^{-1} \cdot \text{kg}^{-1}$). $\dot{V}E_{\max}$ in the training group was significantly higher than that in the control group ($F=5.39$, $p < 0.05$). There was no change in $HR_{\max}$ during the training period in either group.

Fig. 1. Changes in $\dot{V}O_{2\max}$ (upper) and $\dot{V}E_{\max}$ (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.

Changes in Sa_{O_2} , $\dot{V}E/\dot{V}O_2$, and PET_{O_2} during the training period are shown in Fig. 2. There were no significant differences in Sa_{O_2} , $\dot{V}E/\dot{V}O_2$, and PET_{O_2} between the training group and control group during heavy exercise prior to the training. Sa_{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 ($F=14.6$, $p < 0.01$). ΔSa_{O_2} in the training group ($-3.1 \pm 0.7\%$) was significantly larger than that in the control group ($0.5 \pm 0.6\%$). The changes in PET_{O_2} and $\dot{V}E/\dot{V}O_2$ were similar to those for Sa_{O_2} . PET_{O_2} as well as $\dot{V}E/\dot{V}O_2$ did not decrease anymore after the 6th week; nevertheless, Sa_{O_2} kept decreasing up to the last week. 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 ($-7.4 \pm 4.6 \text{ ml/ml}$) was significantly larger than that in the control group ($2.1 \pm 2.6 \text{ ml/ml}$).

Table 1 shows pulmonary function test data at rest in both groups. There were no significant differences in VC, PF, MVV_{12} , and DL_{CO} between the two groups before and after the training period.

Fig. 2. Changes in SaO_2 (upper), $P_{ET}O_2$ (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.

Figure 3 shows relationships among $\dot{V}O_{2max}$, $\dot{V}_E/\dot{V}O_2$, and SaO_2 . SaO_2 and $\dot{V}_E/\dot{V}O_2$ during heavy exercise were negatively related to $\dot{V}O_{2max}$: for SaO_2 , $r = -0.70$, $p < 0.001$ ($n = 77$ based on 11 subjects $\times$ 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 SaO_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 ΔSaO_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 $\dot{V}O_{2max}$, $\dot{V}_E/\dot{V}O_2$, and SaO_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 $\dot{V}O_{2max}$, with little change in $\dot{V}_{E_{max}}$ and ventilatory parameters at rest; 2) training decreased $P_{ET}O_2$, $\dot{V}_E/\dot{V}O_2$, and SaO_2 during heavy exercise; 3) $P_{ET}O_2$ as well as $\dot{V}_E/\dot{V}O_2$ did not decrease anymore after the 6th week, nevertheless, SaO_2 kept decreasing up to the last 2 weeks; 4) SaO_2 or ΔSaO_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 SaO_2 and $\dot{V}_E/\dot{V}O_2$ of the training group was larger than that of the control group.

Training state, $\dot{V}O_{2max}$ and SaO_2 . $\dot{V}O_{2max}$ and the absolute intensity of exercise at 90% $\dot{V}O_{2max}$ significantly increased with training, while SaO_2 during heavy exercise decreased. SaO_2 after training ($92.8 \pm 1.1\%$) 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 $\dot{V}O_{2max}$ of the subjects. If the $\dot{V}O_{2max}$ of our subjects reached a higher level (e.g., $\geq 65 \text{ ml} \cdot \text{min}^{-1} \cdot \text{kg}^{-1}$), the SaO_2 during heavy exercise could have decreased to less than 92%. There was a negative relationship between $\dot{V}O_{2max}$ and SaO_2 during

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

		Before training	After training
VC (l)	Training group	4.87 $\pm$ 0.51	5.06 $\pm$ 0.58
	Control group	5.12 $\pm$ 0.42	4.96 $\pm$ 0.21
PF (l $\cdot$ s $^{-1}$)	Training group	9.96 $\pm$ 1.11	10.25 $\pm$ 1.02
	Control group	10.69 $\pm$ 1.63	10.71 $\pm$ 1.43
MVV ₁₂ /BW (l $\cdot$ min $^{-1}$ $\cdot$ kg $^{-1}$)	Training group	2.93 $\pm$ 0.30	2.81 $\pm$ 0.36
	Control group	2.75 $\pm$ 0.33	2.88 $\pm$ 0.25
D_{LCO} (ml $\cdot$ min $^{-1}$ $\cdot$ mm Hg $^{-1}$)	Training group	36.2 $\pm$ 4.0	33.7 $\pm$ 3.3
	Control group	35.7 $\pm$ 3.3	33.7 $\pm$ 4.6

VC, vital capacity; PF, peak flow; MVV₁₂/BW, maximal voluntary ventilation for 12 s per body weight; D_{LCO} , pulmonary diffusing capacity for carbon monoxide.

Fig. 3. Relationships among $\dot{V}_E/\dot{V}O_2$, and SaO_2 during heavy exercise and $\dot{V}O_{2max}$.

heavy exercise, which corresponds with the results of Williams *et al.* [10]. The relationship suggests dependence of the $\dot{V}O_2$ level on arterial O_2 desaturation during heavy exercise. However, when the relationship was compared between the control and training groups, the slope of the regression line between $\dot{V}O_{2max}$ and SaO_2 in the training group was larger than that in the control group. Therefore, the arterial O_2 desaturation during heavy exercise is considered to be dependent on not only the $\dot{V}O_2$ level attained during exercise but also other adaptations with progress in training, described as follows.

Less hyperventilation and arterial O_2 desaturation. During heavy exercise, PET_{O_2} , which reflected alveolar PO_2 and $\dot{V}_E/\dot{V}O_2$, 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 PO_2 and less hyperventilation during heavy exercise. The present study showed significant relationships between $\dot{V}_E/\dot{V}O_2$ and SaO_2 ($r^2=0.51, p<0.001$, Fig. 3 lower), and ΔSaO_2 and $\Delta \dot{V}_E/\dot{V}O_2$ ($r^2=0.63, p<0.01$). The relationship indicates that about half of the arterial O_2 desaturation during heavy exercise can be accounted for by the less hyperventilatory response. Previous cross-sectional studies demonstrated similar correlations among SaO_2 and $\dot{V}_E/\dot{V}O_2$ [1, 3, 5, 6].

Another factor of arterial O_2 desaturation. It is interesting that PET_{O_2} as well as $\dot{V}_E/\dot{V}O_2$ did not decrease anymore after the 6th week; nevertheless, SaO_2 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 O_2 desaturation may be associated with another factor (e.g., an increase in A-a DO_2). Hopkins and McKenzie [4] reported that Pa_{O_2} was not significantly related to PA_{O_2} ($r=0.51, p=0.08$) but was closely related to A-a DO_2 ($r=0.98, p<0.001$) during heavy exercise. Furthermore, Powers *et al.* [8] confirmed this and concluded that an increase in A-a DO_2 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 elite endurance athletes as subjects, with $\dot{V}O_{2max} \geq 65 ml \cdot min^{-1} \cdot kg^{-1}$ [4, 8], whereas our subjects' $\dot{V}O_{2max}$ finally reached $61.6 \pm 4.1 ml \cdot min^{-1} \cdot kg^{-1}$ after training for 12 weeks ($50.9 \pm 5.63 ml \cdot min^{-1} \cdot kg^{-1}$ at initial test). Rice *et al.* [22] suggested that Pa_{O_2} is strongly associated with inadequate hyperventilation when exercising at a lower $\dot{V}O_2$ level, while an increased A-a DO_2 was the greater determinant of hypoxemia at a higher $\dot{V}O_2$ level. The predominance of less hyperventilation and increased A-a DO_2 in the genesis of arterial hypoxemia and O_2 desaturation may be dependent upon $\dot{V}O_2$ levels during exercise. In the present study, 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 (Fig. 3 lower); i.e., compared at lower level of $\dot{V}_E/\dot{V}O_2$ ($\sim 30 ml \cdot min^{-1}$), SaO_2 of the trained subjects exercising at a higher $\dot{V}O_2$ level was lower than that of the control subjects. Furthermore, $\dot{V}_E/\dot{V}O_2$ of the trained subjects exercising at a lower $\dot{V}O_2$ level ($45\text{--}55 ml \cdot min^{-1} \cdot kg^{-1}$) was lower than that of the control subjects exer-

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-a $\dot{D}O_2$ 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, PE, MVV₁₂, and DL_{CO} 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 DL_{CO} 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 Sa_{O_2} . Accurate measurement of Sa_{O_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 Sa_{O_2} of arterial blood samples determined by a CO-oximeter (Corning 2500, USA) and Sa_{O_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 Sa_{O_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 Sa_{O_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 Sa_{O_2} between the two groups after the 6th 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 Sa_{O_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_{O_2} , $\dot{V}E/\dot{V}O_2$, and Sa_{O_2} during heavy exercise. The decreases in Sa_{O_2} or ΔSa_{O_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_{O_2} as well as $\dot{V}E/\dot{V}O_2$ did not decrease anymore after the 6th week; nevertheless, Sa_{O_2} kept decreasing up to the last 2 weeks. In addition, the regression line between Sa_{O_2} and $\dot{V}E/\dot{V}O_2$ of the training group was steeper than that of the control group: i.e., compared at a lower level of $\dot{V}E/\dot{V}O_2$ ($\sim 30 \text{ ml} \cdot \text{ml}^{-1}$), Sa_{O_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-a $\dot{D}O_2$, 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. Miharū Miyamura (Nagoya University) for his advise, 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

1. Benoit H, Busso T, Castells J, Denis C, and Geysant A: Influence of hypoxic ventilatory response on arterial O₂ saturation during maximal exercise in acute hypoxia. *Eur J Appl Physiol* 72: 101–105, 1995
2. Dempsey JA, Hanson PG, and Henderson KS: Exercise-induced arterial hypoxemia in healthy human subjects at sea level. *J Physiol (Lond)* 355: 161–175, 1984
3. Harms CA and Stager JM: Low chemoresponsiveness and inadequate hyperventilation contribute to exercise-induced hypoxemia. *J Appl Physiol* 79: 575–580, 1995
4. Hopkins SR and McKenzie DC: Hypoxic ventilatory response and arterial desaturation during heavy exercise. *J Appl Physiol* 67: 1119–1124, 1989
5. Miyachi M and Tabata I: The relationship between arterial oxygen desaturation and ventilation during maximal exercise. *J Appl Physiol* 73: 2588–2591, 1992
6. Norton KI, Squires B, Norton LH, Craig NP, McGrath P, and Olds TS: Exercise stimulus increases ventilation from maximal to supramaximal intensity. *Eur J Appl Physiol* 70: 115–125, 1995.
7. Powers SK, Dodd S, Woodyard J, Beadle RE, and Church G: Hemoglobin saturation during incremental arm and leg exercise. *Br J Sports Med* 18: 212–216, 1984
8. Powers SK, Martin D, Cicale M, Collop N, Huang D, and Criswell D: Exercise-induced hypoxemia in athletes: role of inadequate hyperventilation. *Eur J Appl Physiol* 65: 37–42, 1992
9. Powers SK, Dodd S, Lawler J, Kirtley M, McKnight K, and Grinton S: Incidence of exercise induced hypoxemia in elite endurance athletes at sea level. *Eur J Appl Physiol* 58: 298–302, 1988
10. Williams JH, Powers SK, and Stuart MK: Hemoglobin desaturation in highly trained athletes during heavy exercise. *Med Sci Sports Exerc* 18: 168–173, 1986
11. Rowell LB, Taylor HL, Wang Y, and Carlson WS: Saturation of arterial blood with oxygen during maximal exercise. *J Appl Physiol* 19: 284–286, 1964
12. Byrne-Quinn E, Weil JV, Sodal IE, Filley GF, and Grover RF: Ventilatory control in athlete. *J Appl Physiol* 30: 91–98, 1971
13. Martin BJ, Sparks KE, Zwillich CW, and Weil JV: Low exercise ventilation in endurance athletes. *Med Sci Sports* 11: 181–185, 1979
14. Dempsey JA: Is the lung built for exercise? *Med Sci Sports Exerc* 18: 143–155, 1986
15. Martin D, Powers SK, Cicale M, Collop N, Huang D, and Criswell D: Validity of pulse oximetry during exercise in elite endurance athletes. *J Appl Physiol* 72: 455–458, 1992
16. Ries A, Farrow J, and Clausen J: Accuracy of two ear oxymeters at rest and during exercise in pulmonary patients. *Am Rev Respir Dis* 132: 685–689, 1985
17. Taylor MB and Whitman JG: The accuracy of pulse oximeters. *Anesthesia* 43: 229–232, 1988
18. Yelderman M and New W: Evaluation of pulse oxymetry. *Anesthesiology* 59: 349–352, 1983
19. Aoyagi T: Pulse oximetry. *Kokyu* 11: 560–567, 1992 (in Japanese)
20. Cotton DJ, Newth CJL, Portner PM, and Nadel JA: Measurement of single-breath CO diffusion capacity by continuous rapid CO analysis in man. *J Appl Physiol* 46: 1149–1156, 1979
21. Ichihara K: Statistics for Bioscience—Practical Technique and Theory—Nankodo, Tokyo, pp 218–222, 1990 (in Japanese)
22. Rice A, Scroop GC, Gore CJ, Thornton AT, Chapman MA, Greville HW, Holmes MD, and Scicchitano R: Exercise-induced hypoxaemia in highly trained cyclists at 40% peak oxygen uptake. *Eur J Appl Physiol* 79: 353–359, 1999
23. Casaburi R, Storer TW, and Wasserman K: Mediation of reduced ventilatory response to exercise after endurance training. *J Appl Physiol*, 63: 1533–1538, 1987
24. Reuschlein P, Radden WG, Burpee J, Gee JBL, and Rankin J: Effect of physical training on the pulmonary diffusing capacity during submaximal work. *J Appl Physiol* 24: 152–158, 1968
25. Belman MJ and Gaesser GA: Ventilatory muscle training in the elderly. *J Appl Physiol* 64: 899–905, 1988
26. Morgan DW, Kohrt WM, Bates BJ, and Skinner JS: Effect of respiratory muscle endurance training on ventilatory and endurance performance of moderately trained cyclist. *Int J Sports Med* 8: 88–93, 1987
27. Dempsey JA, Gledhill N, Reddan WG, Forster HV, Hanson PG, and Claremont AD: Pulmonary adaptation to exercise: effect of exercise type and duration, chronic hypoxia, and physical training. *Ann NY Acad Sci* 301: 243, 1977