MAXIMAL INTERVAL TRAINING CONTRIBUTES TO THE DEVELOPMENT OF ARTERIAL OXYGEN DESATURATION AND LESS HYPERVENTILATION DURING HEAVY EXERCISE.

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INTRODUCTION

It is well known that a reduction in arterial \( \text{O}_2 \) hemoglobin saturation (\( \text{SaO}_2 \)) during heavy exercise (arterial \( \text{O}_2 \) desaturation) occurs in trained endurance athletes (Dempsey 1984). Although several mechanisms to explain this finding has been proposed, the precise etiology of arterial \( \text{O}_2 \) desaturation during heavy exercise remains to be determined. There has been considerable debate, especially, whether athletes with the desaturation during exercise do (Miyachi 1994), or do not (Hopkins 1989) exhibit less hyperventilation. Rowell et al. (1964) showed that physical training induces arterial \( \text{O}_2 \) desaturation during heavy exercise. If ventilation during exercise is a major factor in arterial \( \text{O}_2 \) desaturation, physical training should also induce less hyperventilation during heavy exercise. Accordingly, we considered it worthwhile to investigate the longitudinal changes in \( \text{SaO}_2 \) and ventilation during heavy exercise in a program of 12 weeks of maximal interval training.

METHODS

Eleven subjects volunteered to participate in the experiments. All were healthy adult males who were continually active in recreational sports with no history of lung disease. One group of six subjects (trained group) underwent the interval training for 12 weeks (4 days/wk). They underwent five periods of exercise of 3-min duration on a cycle ergometer at a power output of 100% \( \text{Vo2max} \) (60 rpm). Work intervals were interspersed with 2-min recovery periods consisting of cycling at 50% \( \text{Vo2max} \). Maximal \( \text{O}_2 \) uptake (\( \text{Vo2max} \)) and maximal ventilation (\( \text{VEmax} \)) were measured to maintain a constant training stimulus on Monday every week during the training period. \( \text{SaO}_2 \) and ventilation during exercise at 90% \( \text{Vo2max} \) were measured every other week. These parameters were also measured before and immediately after the training program. Pulmonary functions (VC, PF, MVVj2, and DLco) at rest were measured before and immediately after the training. The same parameters were measured simultaneously in the other group of five subjects (control group) who led normal lives. The changes in all parameters during the training period were compared using the two-way analysis of variance with repeated measurements. The relationships among the parameters were determined by simple regression analysis. The level of significance was established at \( p < 0.05 \).

RESULTS

There were no significant differences in \( \text{Vo2max} \) and \( \text{VEmax} \) between the trained group and control group before the training period. \( \text{Vo2max} \) leveled off following a linear increase to the fifth week in the trained group. There was no statistical change in \( \text{Vo2max} \) in the control group. \( \text{Vo2max} \) which was tested by two-way ANOVA with repeated measurements (\( F = 7.14, p < 0.05 \)) was significantly higher in the trained group than that in the control group. The total increase in \( \text{Vo2max} \) for the six trained subjects averaged 10.7 ml/min/Met\(^{-1}\) (19.1%, from 50.9±5.63 to 61.6±4.06 ml/min/Met\(^{-1}\)). \( \text{VEmax} \) in the trained group increased slightly during the early weeks of training. \( \text{VEmax} \) in the trained group was significantly higher than that in the control group (\( F = 5.39, p < 0.05 \)).

Changes in \( \text{SaO}_2, \text{VE}/\text{Vo2} \), and \( \text{PETO}_2 \) during the training period are shown in Fig. 1. \( \text{SaO}_2 \) during heavy exercise decreased with 12 weeks of training in the trained group. \( \text{SaO}_2 \) during heavy exercise in the trained group was significantly lower than that in the control group (\( F = 14.6, p < 0.01 \)). The changes in \( \text{VE}/\text{Vo2} \) and \( \text{PETO}_2 \) during the training corresponded to that in \( \text{SaO}_2 \). \( \text{PETO}_2 \) levels in the trained group were significantly lower than those in the control group (\( F = 5.50, p < 0.05 \); \( F = 5.45, p < 0.05 \), respectively). There were no significant differences in VC, PF, MVVj2, and DLco between the two groups before and after training period. \( \text{SaO}_2 \) during heavy exercise was significantly related to \( \text{VE}/\text{Vo2} \) (\( r = 0.71, r^2 = 0.51, p < 0.001 \), Fig. 2) and \( \text{PETO}_2 \) (\( r = 0.76, r^2 = 0.58, p < 0.001 \)) during heavy exercise.
DISCUSSION

The maximal interval training contributes to the development of arterial $O_2$ desaturation during heavy exercise (Fig. 1). Moreover, the training also induces lower alveolar PO$_2$ and less hyperventilation during heavy exercise. The correlation between Sao2 and VE/Vo2 ($r^2 = 0.51$, Fig. 2) during heavy exercise statistically shows that half of the reduction in Sao2 with the training can be accounted for by the variation in ventilation. Our longitudinal data support the hypothesis that ventilation during heavy exercise is a major factor in arterial $O_2$ desaturation.

Dempsey (1986) has hypothesized that arterial $O_2$ desaturation and less hyperventilation during heavy exercise in the trained athletes imply that critical aspects of the pulmonary control system have not adapted appropriately to the increased metabolic demand. In the present study, Vo2max increased 19.1% due to the training, but Vemax increased only 8.2%. Furthermore, pulmonary functions at rest did not significantly change during the 12 weeks of training. These data suggest that there is little adaptability in pulmonary system to physical training for several months, which indirectly support the hypothesis of Dempsey (1986).

REFERENCES


