Ventilatory Capacity and Exercise-induced Arterial Desaturation of Highly Trained Endurance Athletes

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Recent evidence suggested that exercise-induced arterial O\(_2\) desaturation may occur in highly trained endurance athletes. So, Dempsey brought the hypothesis that the pulmonary capacity for oxygen transport cannot meet superior demands imposed by cardiovascular system in highly trained endurance athletes, and endurance training primarily causes adaptation in the skeletal muscles and in the systemic cardiovascular system, with little change in the pulmonary system. In the present study, we determined the propriety of the hypothesis due to measure the ventilatory capacity of endurance athletes. Sixteen highly trained endurance athletes (ET) and thirteen untrained subjects (UT) volunteered to participate in these experiments. All subjects performed the four experiments, 1) the highest oxygen uptake (peak \(\dot{V}\text{O}_2\)) during incremental cycle exercise and ventilation (\(V_e\)), ventilatory equivalent for O\(_2\) (\(\dot{V}_e/\dot{V}\text{O}_2\)) and arterial O\(_2\) saturation (SaO\(_2\)) at which peak \(\dot{V}\text{O}_2\) was observed, 2) the maximal voluntary ventilation for 30 sec at rest (MVV), 3) the pulmonary diffusing capacity for CO (DLCO) and expressed per unit of alveolar lung volume (KCO) at rest by the single breath method, and 4) the ventilatory response to hypercapnia (S) at rest by rebreathing method, were measured. The peak \(\dot{V}\text{O}_2\) of ET (66.7 ml \(\cdot\) min\(^{-1}\) \(\cdot\) kg\(^{-1}\)) was significantly (30.8\%) higher than UT (52.4 ml \(\cdot\) min\(^{-1}\) \(\cdot\) kg\(^{-1}\)), and \(\dot{V}_e/\dot{V}\text{O}_2\) and SaO\(_2\) of ET (29.3 and 93.7\%, respectively) were significantly lower than UT (34.6 and 95.8\%). There were no differences in \(\dot{V}_e\), MVV, DLCO, and S between two groups. Although, KCO of ET (6.02 ml \(\cdot\) min\(^{-1}\) \(\cdot\) mmHg\(^{-1}\) \(\cdot\) l\(^{-1}\)) was significantly higher than UT (5.45 ml \(\cdot\) min\(^{-1}\) \(\cdot\) mmHg\(^{-1}\) \(\cdot\) l\(^{-1}\)), the relative difference was only 10.5\%. Therefore, the arterial oxygen desaturation and less hyperventilation during maximal exercise were observed in ET, and the ventilatory capacity of ET may not be so much superior to that of untrained subjects. These findings supported the hypothesis of Dempsey.


**Key words**: Arterial oxygen saturation (SaO\(_2\)), Maximal voluntary ventilation, Pulmonary diffusion capacity, Ventilatory response to CO\(_2\)

Traditionally, pulmonary capacity has been dismissed as a potentially limiting factor to endurance performance or aerobic capacity in healthy individuals exercising at sea level, since arterial oxygen pressure (PaO\(_2\)) and arterial oxygen saturation (SaO\(_2\)) remains high during exercise (Asmussen and Nielsen 1960, Hesser and Matell 1965). However, recent evidences suggest that exercise-induced hypoxemia or arterial oxygen desaturation may occur in highly trained endurance athletes (Dempsey et al. 1984, Hopkins and McKenzie 1989, Lawler et al. 1988, Powers et al. 1984, Powers et al. 1988, Williams et al. 1986). It may be due to lower alveolar oxygen partial pressure (P\(_a\)O\(_2\)) by less hyper-
ventilation (Dempsey et al. 1984, Powers et al. 1984) and diffusion limitation by short red blood cell transit time through lung capillaries caused by large cardiac output (Dempsey et al. 1984, Torre-Bueno et al. 1985, Williams et al. 1986). Therefore, Dempsey (1986) has brought the working hypothesis that the pulmonary capacity for oxygen transport cannot meet superior demands imposed by limbs and cardiovascular system in highly trained endurance athletes. In the present study we tested the hypothesis of Dempsey by measuring the pulmonary capacity of endurance athletes.

METHODS

Sixteen highly trained endurance athletes (ET) and thirteen untrained subjects (UT) volunteered to participate in these experiments. All were healthy, nonsmoking, adult males with no history of lung diseases. For several years, ET had been performing endurance training for competition. The UT was composed of nonathletes who performed exercise several times/week on a regular basis. Descriptive data for each group are as follows: age (ET: 23.5 yr., UT: 23.5 yr.), Body wt. (ET: 55.9 kg, UT: 63.3 kg), height (ET: 167.0 cm, UT: 171.2 cm).

The present study was divided into four tests that were 1) the incremental cycle exercise test, 2) the maximal voluntary ventilation (MVV) test, 3) the pulmonary diffusion test, and 4) the ventilatory response to CO2 test. All tests were performed under a uniform environment (temperature: 20-25 °C, barometric pressure: 740-755 mmHg).

1) The incremental cycle exercise test. The incremental cycle exercise began at a work rate of 103 watts (70 rpm), and power output was increased by 17 watts/min until the subject could not maintain the fixed pedalling frequency. SaO2 was monitored minute by minute. Oxygen uptake (VO2), expired ventilation (VE) and ventilatory equivalent for oxygen (VE/VO2) were monitored during the last 30 sec of each work rate. The highest value of VO2 during the exercise test was called peak VO2. The values of other parameters at which peak VO2 was observed were used as the typical data for the exercise test. SaO2 was estimated with a pulse oxymeter (Nihon Koden OLV1200, Japan) which was positioned on the tip of the right forefinger. The accuracy of pulse oxymetry was proven by several studies (Ries et al. 1985, Taylor and Whitman 1988, Yelderman and New 1983). Subjects breathed through a low-resistance two-way valve (Hance Rudolph #2700, USA), and the expired air was collected in Douglas bags. Concentrations of expired O2 and CO2 gases were measured by mass spectrometry (Perkin Elmer MGA 1100, USA), and gas volume was determined using a dry gas meter (Shinagawa Dev. NDS-2A-T, Japan).

2) MVV test. The subjects were asked to breathe as rapidly and as deeply as possible at rest in standing position for 30 seconds. The expired air was collected in Douglas bags through a low-resistance two-way valve (Hance Rudolph #2700, USA), and expired gas volume was determined using a dry gas meter (Shinagawa Dev. NDS-2A-T, Japan).

3) Pulmonary diffusion test. The pulmonary diffusing capacity for CO (DLCO) and the diffusion per unit of alveolar volume (KCO) were measured with the single-breath method (Sensor Medics MMC 4400 tc, USA) (Cotton et al. 1979, Krogh 1914). They were recorded by measuring the rate of uptake of CO on a bag with continuous monitoring of mixed expired gas. It provided from a single breath of sample gas a continuous concentration profile of measured gases. CO was used as the sample gas for DLCO; CH4 was used as an insoluble reference gas, providing the lung dilution normalization factor. A fast responding multigas analyzer operates on nondispersive infrared absorption principle. The single-breath method is less demanding on a subject's cooperation and the test itself is much simpler to perform. Before test, as the subject ends expiration, then maximally inhaled the mixture from the bag. After maximum inhalation, the subject held his
breath for 10 seconds, then he exhaled maximally. During both inhalation and exhalation, the sample was measured and analyzed 100 times per seconds.

4) The ventilatory response to CO₂ test. The ventilatory response to CO₂ was measured with the rebreathing method modified from Ohkuwa et al. (1980) and Ohyabu et al. (1990). The subjects rebreathed hyperoxic and hypercapnic gas mixture from the bag through the mouthpiece for 3.0-3.5 min. The expired ventilation volume (Vₑ) during rebreathing was measured with a pneumotachograph (Minato Med. Sci. RM 200, Japan) attached between the mouthpiece and rebreathing bag. To measure end-tidal carbon dioxide partial pressure (PₑCO₂), the expired gas was continuously withdrawn through the mouthpiece from a sampling capillary tube connected to the gas analyzer (Perkin Elmer MGA 1100, USA). Vₑ and PₑCO₂ data were stored on a breath-by-breath basis and averaged every 30 sec. Each rebreathing bag was filled with 5 l of gas mixture of 5 % CO₂ and 95 % O₂. The slope of the Vₑ-PₑCO₂ line was calculated by least-squares regression method. The equation Vₑ = S (PₑCO₂ - B) was used for determination of ventilatory response to CO₂ (S).

All gas volumes were corrected to STPD condition. Differences between trained and untrained were analyzed using unpaired t-test. The level of significance was established at p < 0.05.

RESULTS

The mean data and SD are shown in Table 1. 1) The incremental exercise test. Peak VO₂ of ET was significantly (30.8 %) higher than UT, and Vₑ/VO₂ and SaO₂ of ET were significantly lower than UT. There was no difference in Vₑ between the two groups. Arterial oxygen desaturation and less hyperventilation were observed in the ET during maximal exercise. 2) MVV test. There was no difference in MVV between the groups. 3) Diffusion capacity test. There was not difference in DLCO between the groups. Although KCO of trained was significantly higher than untrained, the relative difference was only 10.5 %. 4) The ventilatory response to CO₂ test. There was not significant difference in S between the groups.

DISCUSSION

Arterial oxygen desaturation. Traditionally, pulmonary capacity has been dismissed as a potentially limiting factor to endurance performance or aerobic capacity in healthy individuals exercising at sea level, since PaO₂, SaO₂ and PₐO₂ remain high during exercise (Asmussen and Nielsen 1960, Hesser and Matell 1965). However, several recent studies (Dempsey et al. 1984, Hopkins and McKenzie 1989, Lawler et al. 1988, Powers et al. 1984, 1988, Williams et al. 1986) reported that the arterial oxygen desaturation was observed in endurance trained subjects during maximal exercise. The SaO₂ and
of ET were significantly lower than UT during maximal exercise in the present study. The present results also suggest that the pulmonary capacity for oxygen transport cannot meet the superior demands imposed by cardiovascular system in highly trained endurance athletes.

Ventilatory capacity. The peak \( \dot{V}_{\text{O}_2} \) of ET was significantly higher than UT, and the relative difference was 30.8\%. However, there were no differences in \( \dot{V}_E \), MVV, DLCO, and S. Although KCO of ET was significantly higher than UT, the relative difference was only 10.5\%. Therefore, the ventilatory capacity of endurance trained athletes may not be so much superior to that of untrained subjects. It has been generally accepted that there is no difference in MVV between endurance athletes and control subjects (Marler et al. 1982, Reuschlein et al. 1968). The previous studies, further, (Marler et al. 1982, Newman et al. 1962, Reuschlein et al. 1968) reported that there are no differences in DLCO and KCO at rest between athletes and nonathletes. It is not clear that S of endurance trained athletes are different from S of untrained (Marler et al. 1982, Ohyabu et al. 1990, Martin et al. 1978, 1979, Ohkuwa et al. 1980). However, at least, these findings suggest that endurance training primarily induces adaptation in skeletal muscle and in cardiovascular system, but not in pulmonary system.

Conclusion. The arterial oxygen desaturation and less hyperventilation during maximal exercise were observed in the trained athletes. Further, there was not so much difference in ventilatory capacity between the trained athletes and untrained subjects. These results support the Dempsey's hypothesis that the pulmonary capacity for oxygen transport cannot meet superior demands imposed by cardiovascular system in highly trained endurance athletes.

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