Exercise-induced oxyhaemoglobin desaturation, ventilatory limitation and lung diffusing capacity in women during and after exercise

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Abstract Arterial haemoglobin saturation during exercise in healthy young women [eight subjects mean (SEM) age 20.8 (1.8) years] was measured to confirm the theory that young women experience exercise-induced arterial hypoxaemia (EIAH) at a lower relative percentage of maximal oxygen uptake ($V_{O2max}$) than has been documented in their male counterparts. To determine if flow limitation [the percentage of the tidal volume ($V_T$) that met or exceeded the boundary established by multiple maximal expiratory manoeuvres] and/or post-exercise lung diffusing capacity are linked to EIAH in women, and to investigate the influence of exercise intensity and duration on post-exercise carbon monoxide lung diffusing capacity ($D_{L, CO}$), these parameters were measured during and after three exercise tests (incremental test until exhaustion, 5 km run and 5 km run with sprint). All subjects experienced physiologically significant EIAH (a fall of more than 3% in oxygen saturation of arterial blood from levels at rest) and seven subjects experienced flow limitation during the $V_{O2max}$ protocol [mean (SD) 12.2 (8.8)% of $V_T$]. Even though there was no significant relationship between aerobic capacity and the degree of flow limitation ($r = 0.33, P > 0.05$), the flow limitation was related to absolute ventilation in the subjects studied ($r = 0.82, P < 0.05$). There was no significant relationship between decrements in post exercise $D_{L, CO}$ and EIAH ($r = 0.05, P > 0.05$), however there was a strong correlation between the extent of flow limitation (% of $V_T$) and EIAH ($r = 0.71$). Significant decreases in $D_{L, CO}$ lasted for up to 16 h after each of the exercise tests ($P < 0.05$) and lasted for a further 8 h after the maximal test ($P < 0.05$). Exercise intensity was the main contributing factor to the observed decreases in post-exercise $D_{L, CO}$ with the percentage of $VO_2max$ attained during the various tests being significantly related to the fall in $D_{L, CO}$ for 1, 2, 3, 16 and 24 h after exercise ($P < 0.05$). As the appearance of flow limitation closely coincided with the appearance of EIAH, the results from the present study suggest that flow limitation is a contributing factor to EIAH in women although the exact mechanism remains unclear.

Keywords Lung diffusing capacity · Exercise induced arterial hypoxaemia · Flow limitation · Oxyhaemoglobin desaturation · Exercise

Introduction

The incidence of hypoxaemia and/or arterial oxyhaemoglobin desaturation in male athletes is well documented (Dempsey et al. 1984; Dempsey and Wagner 1999; Johnson et al. 1992; Powers et al. 1988) and appears to occur in approximately 40%–50% of highly trained (68–70 ml·kg$^{-1}$·min$^{-1}$) male endurance athletes in response to maximal exercise. This inability of the pulmonary system to maintain homeostasis of the arterial blood $O_2$ content during exercise is termed exercise-induced arterial hypoxaemia (EIAH). As the threshold for a fall in oxygen saturation of arterial blood ($S_aO_2$) measurably influencing systemic $O_2$ transport and maximal oxygen uptake ($VO_2max$) is a decrease of 3% from levels at rest (Dempsey and Wagner 1999), a 3% reduction can be used as an indication of physiologically significant EIAH.

This failure of homeostasis may occur due to transient mechanisms that only come into play during exercise such as a relative hypoventilation during exercise, physiological shunting in the setting of a falling mixed venous partial pressure of oxygen and/or a mismatch between alveolar ventilation and pulmonary capillary
flow ($V_{A}/Q_{m}$). Another possibility is that EIAH is caused by physical changes occurring at the alveolar-capillary interface induced by heavy exercise that may result in diffusion impairment and a failure of diffusion equilibration in the alveolar capillaries.

In many endurance-trained men almost all of the flow-volume envelope may be used during maximal exercise (Johnson et al. 1992). This group of subjects would be likely to experience some form of flow limitation during maximal exercise because of their high ventilatory requirements. Inadequate hyperventilatory compensation caused by true flow limitation, decreased chemosensitivity or a reflex termination of expiratory effort brought about by impending flow limitation could contribute to EIAH in the male population (Chapman et al. 1998). Flow limitation may also influence gas exchange through other mechanisms, such as an altered distribution of ventilation (Dempsey and Wagner 1999).

There is also considerable circumstantial evidence for a role of a limitation of lung diffusion in widening the alveolar to arterial oxygen pressure difference during exercise and therefore contributing to EIAH (Powers et al. 1988; Rice et al. 1999). Indirect evidence for a change in fluid regulation at the alveolar-capillary membrane in humans includes reductions in carbon monoxide lung diffusing capacity ($D_{L, CO}$) after strenuous exercise (Caillaud et al. 1995; Turcotte et al. 1997) and $V_{A}/Q_{m}$ inequalities that have persisted after exercise had ceased (Schaffartzik et al. 1992). Fluid accumulation in the lung may contribute to the fall in $D_{L, CO}$, which contributes to an impairment of lung diffusion (Hopkins et al. 1998). Post exercise changes to $D_{L, CO}$ have been linked with a decrease in post-exercise pulmonary capillary blood volume (Sheel et al. 1998) as well as a decrease in the membrane diffusing capacity.

The effect of exercise duration and intensity on lung diffusing capacity has not been documented to any great extent for women. However, significant changes in lung diffusing capacity have been shown in men after both short-duration high-intensity exercise (Sheel et al. 1998) and long-duration moderate-intensity exercise (Manier et al. 1991; Miles et al. 1983). To determine the effect of exercise duration and intensity on $D_{L, CO}$ in the female population, $D_{L, CO}$ was measured after three different exercise protocols of varying intensity and duration. We hypothesized that exercise intensity would influence $D_{L, CO}$ to a greater degree than exercise duration, as the high pulmonary pressures associated with intense exercise have been associated with the largest falls in $D_{L, CO}$ in men (Sheel et al. 1998).

There are several studies investigating EIAH in the female population (Harms et al. 1998, 2000; Hopkins et al. 2000; McClaran et al. 1998; St Croix et al. 1998). The work by Harms et al. (1998) suggests that many young women experience significant EIAH at a $VO_{2\text{max}}$ that is substantially less than those in their active male counterparts. The onset of EIAH during sub-maximal exercise, and its occurrence at a relatively low $VO_{2\text{max}}$, implies that the lung structure/function involved in alveolar to arterial transport of oxygen may be abnormally compromised. However, Hopkins et al. (2000) only documented EIAH in a subject that had a $VO_{2\text{max}}$ of more than 50 ml·kg$^{-1}$·min$^{-1}$ (average of 180% of predicted). The authors were uncertain why the women in the two study populations experienced EIAH at differing percentages of $VO_{2\text{max}}$.

In the present study the incidence of arterial haemoglobin desaturation during exercise in healthy young women, with widely varying fitness levels was measured to confirm the theory that young women experience EIAH at a lower relative percentage of $VO_{2\text{max}}$ than has been documented in their male counterparts. The study also sought to determine if the extent of EIAH in women during incremental exercise until exhaustion was related to the degree of limitation of expiratory flow and/or to post exercise $D_{L, CO}$.

### Methods

#### Subjects

A total of eight healthy women ranging in age from 19 to 25 years participated in the study. All subjects participated in amateur sport (field hockey, netball and running) only at a social level and therefore could not be considered to be endurance trained to any great extent. The subjects were [mean (SEM)] age 20.8 (1.8) years, height 165.5 (2.6) cm, body mass 62.4 (5.5) kg. Institutional consent forms were completed by all subjects before participating in the study.

#### Lung function at rest

At 1 week prior to the tests the subjects completed a range of lung function tests at rest using a Medgraphics 1085/D body plethysmograph (Edward Keller Australia Pty. Ltd., Victoria, Australia) connected to an Acer Power PC computerised spirometer. Measurements included total lung capacity (TLC), forced vital capacity (FVC), forced expiratory volume in 1 s (FEV$_1$), (FEV$_1$/FVC), functional residual capacity (FRC), and maximal expiratory flow from 50% of TLC (MEF$_{50}$). Lung diffusing capacity ($D_{L, CO}$, in millilitres of carbon monoxide per minute per millimetre of mercury) was measured using a modified Krogh single-breath technique (Ruppel 1998).

#### Exercise protocols

Subjects participating in the study completed three different exercise tests on a motor-driven treadmill with an interval of 1 week between each test. The test protocols were designed so that changes in lung function in response to differing exercise intensities and durations could be investigated. The tests included:

1. An incremental treadmill test to $VO_{2\text{max}}$. The test protocol was designed to exercise to maximal intensity and to be of short duration.
2. A free-paced 5 km time-trial with a finishing sprint. The protocol was designed to produce an exercise intensity similar to test 3 until the final sprint when the exercise intensity approached that of test 1. The test was of moderate duration.
3. A steady-state 5 km time trial without a sprint finish. The exercise intensity was close to the individual’s anaerobic threshold (blood lactate concentration of 4.0 mmol·l$^{-1}$). The test was of moderate duration.

Before each test the subjects completed a 5 min warm-up on the treadmill at a gradient of 0% and at a speed of 7 km·h$^{-1}$.