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Reduced performance of male and female athletes at 580 m altitude

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Abstract This study examined the effect of mild hypobaria (MH) on the peak oxygen consumption ($\dot{V}O_{2peak}$) and performance of ten trained male athletes [$\bar{x}$ (SEM); $\dot{V}O_{2peak}$ = 72.4 (2.2) ml · kg^{-1} · min^{-1}] and ten trained female athletes [$\dot{V}O_{2peak}$ = 60.8 (2.1) ml · kg^{-1} · min^{-1}]. Subjects performed 5-min maximal work tests on a cycle ergometer within a hypobaric chamber at both normobaria (N, 99.33 kPa) and at MH (92.66 kPa), using a counter-balanced design. MH was equivalent to 580 m altitude. $\dot{V}O_{2peak}$ at MH decreased significantly compared with N in both men [– 5.9 (0.9)%] and women [– 3.7 (1.0)%]. Performance (total kJ) at MH was also reduced significantly in men [– 3.6 (0.8)%] and women [– 3.8 (1.2)%]. Arterial oxyhaemoglobin saturation (S_aO_2) at $\dot{V}O_{2peak}$ was significantly lower at MH compared with N in both men [90.1 (0.6)% versus 92.0 (0.6)%] and women [89.7 (3.1)% versus 92.1 (3.0)%]. While S_aO_2 at $\dot{V}O_{2peak}$ was not different between men and women, it was concluded that relative, rather than absolute, $\dot{V}O_{2peak}$ may be a more appropriate predictor of exercise-induced hypoxaemia. For men and women, it was calculated that 67–76% of the decrease in $\dot{V}O_{2peak}$ could be accounted for by a decrease in O_2 delivery, which indicates that reduced O_2 tension at mild altitude (580 m) leads to impairment of exercise performance in a maximal work bout lasting ≈ 5 min.

Key words Performance · Hypoxaemia · $\dot{V}O_{2peak}$ · Hypobaria · Women

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Introduction

Pulmonary gas exchange is not considered a factor limiting peak oxygen consumption ($\dot{V}O_{2peak}$) in healthy untrained individuals because arterial oxyhaemoglobin saturation (S_aO_2) is maintained near resting values even during maximal exercise (Asmussen and Nielsen 1960; Lawler et al. 1988). However, in approximately 50% of endurance-trained men, exercise-induced hypoxaemia (EIH), characterized by an $S_aO_2 \leq 92\%$ and an arterial oxygen tension (P_aO_2) 2.4 kPa below resting levels (Powers et al. 1992), occurs during maximal exercise at sea level (Dempsey et al. 1984; Powers et al. 1992, 1993). Furthermore, Powers et al. (1989) concluded that an S_aO_2 below ≈ 92–93% caused a measurable reduction of maximal oxygen consumption ($\dot{V}O_{2max}$) with an approximate 1% decrease in $\dot{V}O_{2max}$ for each 1% decrease in S_aO_2. Other studies (Johnson et al. 1992; Lawler et al. 1988; Martin and O'Kroy 1993; Powers et al. 1992) have shown that hypoxic conditions that decrease inspired oxygen tension (P_iO_2) to simulate altitudes of approximately 2000–3500 m accentuate EIH and reduce $\dot{V}O_{2max}$. More recently, Gore et al. (1996) reported that EIH was also significantly greater at 580 m altitude and that $\dot{V}O_{2max}$ was reduced significantly (– 6.8%). These results support the concept that the aerobic power of trained individuals decreases progressively as one begins to ascend from sea level (Squires and Buskirk 1982).

Given the important contribution of maximal aerobic power to cycling performance (Craig et al. 1993; Olds

et al. 1995), a small decrease in $\dot{V}O_{2max}$, as a consequence of decreased S_aO_2 and reduced O_2 delivery, would be expected to compromise performance. However, $\dot{V}O_{2max}$ is not the best predictor of performance in endurance-trained athletes. Studies of runners (Noakes et al. 1990), swimmers (Hawley et al. 1992) and cyclists (Hawley and Noakes 1992) have suggested that measurements of peak speed and peak work output (W_{peak}) are better predictors of performance than is $\dot{V}O_{2max}$. In the only study which has explored the performance consequences of EIH, Koskolou and McKenzie (1994) found a decrease in total work (kJ), but not $\dot{V}O_{2peak}$, during a 5-min performance test when endurance-trained men experienced moderate hypoxaemia ($S_aO_2 \leq 87\%$). However, there remains some doubt as to the minimum level of EIH that will decrease performance, since Koskolou and McKenzie (1994) did not measure S_aO_2 directly from arterial blood. Based on previous observations at 580 m altitude (Gore et al. 1996) we hypothesized that both $\dot{V}O_{2peak}$ and performance (total work) in a 5-min maximal cycle test would be reduced.

Although data investigating the prevalence of EIH in men are extensive (Dempsey et al. 1984; Powers et al. 1992, 1993), only two studies have examined this phenomenon in endurance-trained females (McCusker and Brilla 1992; Pedersen et al. 1992). However, the $\dot{V}O_{2peak}$ values (57 and 52 $ml \cdot kg^{-1} \cdot min^{-1}$) and the training status of their subjects were relatively modest. Given the high relative $\dot{V}O_{2max}$ and cardiac output of endurance-trained female athletes, combined with reduced haemoglobin concentration ([Hb]) (Kjellberg et al. 1949), we hypothesized that EIH may be more prevalent in female athletes than in male athletes. Moreover, in response to increased altitude and lower oxygen tension (P_iO_2), the $\dot{V}O_{2peak}$, S_aO_2 and performance of women may be even more susceptible than that of men. Therefore, the aim of this study was to determine whether 580 m altitude reduced 5-min cycling performance in male or female endurance-trained athletes.

Methods

Subjects

Ten men and ten women (Table 1) gave written, informed consent to participate in the study. All subjects were either cyclists or triathletes and were life-time non-smokers with no history of asthma. The International Union Against Tuberculosis Questionnaire (Burney and Chinn 1987) was completed by all subjects to obtain a respiratory history, and spirometry was conducted using a Vitalograph (Med. Industr., Hamburg, Germany), with the subjects performing a minimum of three trials whilst standing and wearing a nose clip. Stretch height and mass [Toledo Digital scales, 120 (0.02)kg capacity] were measured with the subject in light clothing and barefoot. Normal lung function was confirmed because the mean forced vital capacity (FVC) and forced expiratory volume in one second (FEV_1) were ≥ 100% of Australian normative data (Gore et al. 1995) predicted for appropriate sex, age and height.

Table 1 Physical characteristics of the men (n = 10) and women (n = 10) at normobaria. (FVC Forced vital capacity, FEV_1 forced expiratory volume in 1s). Values are means (SEM)

	Males	Females
Age (years)	23.8 (1.9)	26.7 (1.7)
Height (cm)	177.7 (1.3)	*166.3 (2.0)
Mass (kg)	72.38 (2.13)	*58.56 (1.35)
FVC (l)	6.08 (0.25)	*4.53 (0.23)
FEV_1 (l)	4.66 (0.24)	*3.65 (0.19)
$\dot{V}O_{2peak}$ ($l \cdot min^{-1}$)	5.21 (0.13)	*3.56 (0.13)
$\dot{V}O_{2peak}$ ($ml \cdot kg^{-1} \cdot min^{-1}$)	72.3 (2.0)	*60.8 (2.0)

*Significant difference from men, $P < 0.05$

Test protocol

The research techniques and protocol were approved by the Australian Defence Medical Ethics Committee. Each subject completed two 5-min maximal work tests on a geared, air-braked cycle ergometer (Hayes, Adelaide, Australia) with a minimum of 24 h between tests. One test was conducted at normobaria (N = 99.33 kPa) and the other at mild hypobaria (MH = 92.66 kPa), with the order of testing counter-balanced and double-blinded. The 6.67kPa difference between tests is equivalent to 580 m altitude (International Civil Aviation Organisation 1954). All subjects completed a 5-min maximal work test at sea level, as a habituation trial, 1 week prior to the test protocol. Prior to the 5-min performance test, all subjects warmed up for 5 min with standardized work rates of 200 and 125 W for men and women, respectively. Total work (kJ) and power output ($\dot{W}$) during each test was assessed using SRM cranks (Ingenieurbüro Schoberer, Jülich, Germany) with information recorded on a data logger and analyzed using SRM software. The accuracy of the SRM cranks was verified before the study commenced using a first principles calibration rig (Stanef 1988). Cardiac frequency (f_c) was recorded with a Polar PE4000 receiver (Polar Electro, Kempele, Finland) and transmitter belt.

All tests were conducted in a hypobaric chamber (Thompsons, Castlemaine, Australia) located at the Royal Australian Air Force (RAAF) Base Edinburgh, Adelaide, Australia. The chamber dimensions, operating conditions, altimeter calibration and ascent/descent rates replicated those of our previous study (Gore et al. 1996). Normobaria of 99.3 kPa rather than 101.3 kPa was chosen since the chamber did not have hyperbaric capability and since 99.3 kPa represents the lowest barometric pressure that might occur at sea level in Adelaide. Temperature (°C) and relative humidity (%) within the chamber were monitored with a Solomat MPM 200 Environmental Analyser (UK), calibrated by Telstra Products and Services to National Association of Testing Authorities standards. Mean (SEM) chamber temperature and relative humidity during normobaria tests were 25.0 (0.4)°C and 48.6 (1.8)% respectively. Mean chamber temperature and relatively humidity during the mild hypobaric trials were 25.1 (0.3)°C and 43.7 (1.1)%, respectively.

Oxygen consumption

Oxygen consumption ($\dot{V}O_2$) was measured every 30 s with a custom-built open-circuit indirect calorimetry system interfaced to an Apple IIe computer. The inspired volume of air was measured with a Morgan ventilometer (PK Morgan, Rainham, Kent, UK) connected to a R2700 two-way respiratory valve (Hans Rudolph, Kansas City, Miss., USA). The ventilometer was calibrated according to the manufacturer's specifications with 10 l of air administered with a 1.0-l calibration syringe (PK Morgan). This was then verified by administrations of 60, 120, 180 $l \cdot min^{-1}$ with a 3.0-l calibration syringe (Hans Rudolph). The precision and accuracy of Morgan ventilometers have been established previously (Hart et al.

1994). Expired gases passed through 1 m of Clean-bor tubing (Vacumed, Ventura, Calif., USA) to a 2.6-l mixing chamber (Sportech, ACT, Australia), with the total system dead space being ≈ 3.7 l. Gas samples from the mixing chamber passed through a 15-cm $CaCl_2$ drying tube before reaching O_2 (Ametek S-3 AI) and CO_2 (Ametek CD-3A) gas analysers. The metabolic gas analysers were calibrated immediately before and after each test with three alpha-standard gas mixtures (BOC Gases, Australia), with O_2 and CO_2 concentrations spanning the physiological range. All components of the open-circuit calorimetry system were calibrated when chamber pressure was confirmed as stable. Post-test checks of both the ventilometer and gas analysers were completed before chamber pressure was returned to ambient conditions. $\dot{V}O_{2peak}$ was calculated as the mean of the two highest consecutive 30-s $\dot{V}O_2$ samples during exercise.

Prior to the experiments, the precision or technical error of measurement [TEM, (Denegar and Ball 1993)] of the indirect calorimetry system was assessed using duplicate tests on a similar group of ten well-trained male cyclists. The TEM for an incremental cycling $\dot{V}O_{2max}$ was 2.6%. A TEM of 1.3% for both total work and average power output for a 5-min test was established in five male high-performance cyclists. Preliminary (unpublished) tests on 16 male cyclists also demonstrated that the $\dot{V}O_{2peak}$ during 5-min maximal work tests ($\bar{x}$ = 5.23 $l \cdot min^{-1}$) was not significantly different (t = – 1.66, P = 0.12) from the $\dot{V}O_{2max}$ obtained during incremental tests ($\bar{x}$ = 5.16 $l \cdot min^{-1}$).

Arterial blood sampling

An indwelling catheter (Cathlon IV 20 gauge, 1.5 mm OD × 32 mm length, Johnson and Johnson, USA) was inserted with the tip upstream into the brachial artery, which was anaesthetized locally (lignocaine hydrochloride, 2%, Astra Pharmaceuticals, Australia) immediately prior to each test. Arterial blood samples (2.5 ml) were drawn into heparinized glass syringes at rest and after 1, 3 and 5 min of exercise. Samples were capped immediately and stored vertically on ice and, after thorough mixing, were analyzed in triplicate for blood gases [P_aO_2; arterial carbon dioxide tension (P_aCO_2)] and pH using a Ciba Corning Blood Gas Analyser (model 860, Medfield, Mass., USA). Quadruplicate analyses of functional oxyhaemoglobin saturation (HbO_2 = HbO_2/HbO_2 + reduced Hb), fractional oxyhaemoglobin saturation [S_aO_2 = $HbO_2/(HbO_2$ + reduced Hb + metHb + COHb)] and [Hb] were conducted using a Ciba Corning CO-oximeter (model 270 Medfield). The blood gas analyser was calibrated every 2h using standards purchased from the manufacturer, with two quality controls (BioRad, Anaheim, Calif., USA) assayed every hour. The CO-oximeter was calibrated prior to the first test, and quality controls were assayed on each morning of testing. Calibration and control solutions were purchased from Ciba Corning. Blood gas and S_aO_2 values were measured at 37°C with no temperature correction factor applied, since in a repeated measures design each subject acts as their own control and results are directly comparable. The ideal alveolar gas equation was used to calculate alveolar oxygen tension (P_AO_2) and the alveolar – arterial oxygen tension difference ($AaDO_2$) (Otis 1964). The arterial oxygen content (c_aO_2) values were calculated using functional HbO_2, according to the method of Siggaard-Andersen et al. (1988).

Expression of results and statistical analysis

A three-way repeated measures analysis of variance (ANOVA) (Statistica/W – StatSoft, Tulsa, Okla., USA) was used to determine the interactions and main effects of time, chamber pressure and gender on blood (P_aO_2, P_aCO_2, P_AO_2, $AaDO_2$, S_aO_2, pH, [Hb], c_aO_2), metabolic [$\dot{V}O_2$, expired carbon dioxide production ($\dot{V}CO_2$)], and ventilation data [expired minute volume of gas at standard temperature and pressure dry ($\dot{V}_{E\ STPD}$), expired minute volume of gas at body temperature and pressure saturated ($\dot{V}_{E\ BTPS}$), ventilatory equivalent for O_2 ($\dot{V}_{E\ BTPS}/\dot{V}O_2$), ventilatory equivalent for CO_2 ($\dot{V}_{E\ BTPS}/\dot{V}CO_2$)]. A two-way ANOVA was used when time was not an independent variable ($\dot{V}O_{2peak}$, total work). The Tukey HSD post-hoc test was performed to locate specific mean differences. Student's t-tests for independent samples were used to compare the physical characteristics of the men and women. The significance level was set at $P < 0.05$, with results expressed as mean (SEM), unless otherwise stated.

Results

Oxygen consumption

$\dot{V}O_{2peak}$ decreased significantly in response to hypobaria in both the men [– 5.9 (0.9)%: N, 5.21 (0.13) $l \cdot min^{-1}$; MH, 4.90 (0.12) $l \cdot min^{-1}$] and women [– 3.7 (1.0)%: N, 3.56 (0.13) $l \cdot min^{-1}$; MH, 3.43 (0.13) $l \cdot min^{-1}$], and the decrease was significantly greater in men than women, $F_{(1,18)}$ = 8.4, P = 0.01. During exercise, $\dot{V}O_2$ of the men at MH was reduced after 1, 2, 3, 4 and 5 min of exercise compared with the matched times at N (Fig. 1), while that of women was reduced after 3 and 4 min. The correlation between normobaric $\dot{V}O_{2peak}$ and $\%\Delta\dot{V}O_{2peak}$ from N to MH failed to reach statistical significance for the pooled data of the men and women (r^2 = 0.15, P = 0.09).

Performance

Women produced significantly less total work and power than men at both N and MH. When assessed for each minute of exercise, $\dot{W}$ of the men was reduced significantly after 1, 2 and 3 min, while that of the women was not changed with hypobaria (Fig. 1). However, the total work for the entire 5 min of exercise decreased significantly from N to MH in both men [– 3.9 (0.8)%: N, 114.8 (2.8) kJ; MH, 110.3 (2.5) kJ] and women [– 3.8 (1.2)%: N, 80.8 (1.8) kJ; MH, 77.7 (2.1) kJ], respectively, $F_{(1,18)}$ = 31.4, $P < 0.0001$. When the data of men and women were pooled there was no significant correlation between %Δ total work from N to MH and either the corresponding $\Delta\dot{V}O_{2peak}$ (r^2 = 0.05) or ΔS_aO_{2peak} (r^2 = 0.04).

Oxygen saturation of haemoglobin

There was no significant difference in S_aO_2 between men and women, with both showing a progressive decrease from rest (≈ 98%) to mean values after 5 min of exercise of 92.1 (0.6)% (N) and 89.9 (0.6)% (MH) (Fig. 2). When the data of men and women were pooled, S_aO_2 was significantly lower at MH than at N, at rest and throughout exercise, $F_{(1,18)}$ = 79.1, $P < 0.0001$. For pooled data of men and women, the correlation between $\Delta\%S_aO_2$ during the final minute of exercise at N and MH and the corresponding $\Delta\dot{V}O_{2peak}$ was not significant (r^2 = 0.10). Functional HbO_2 was similar to fractional S_aO_2, with no significant difference between men and women at N or MH. HbO_2 was significantly lower at N

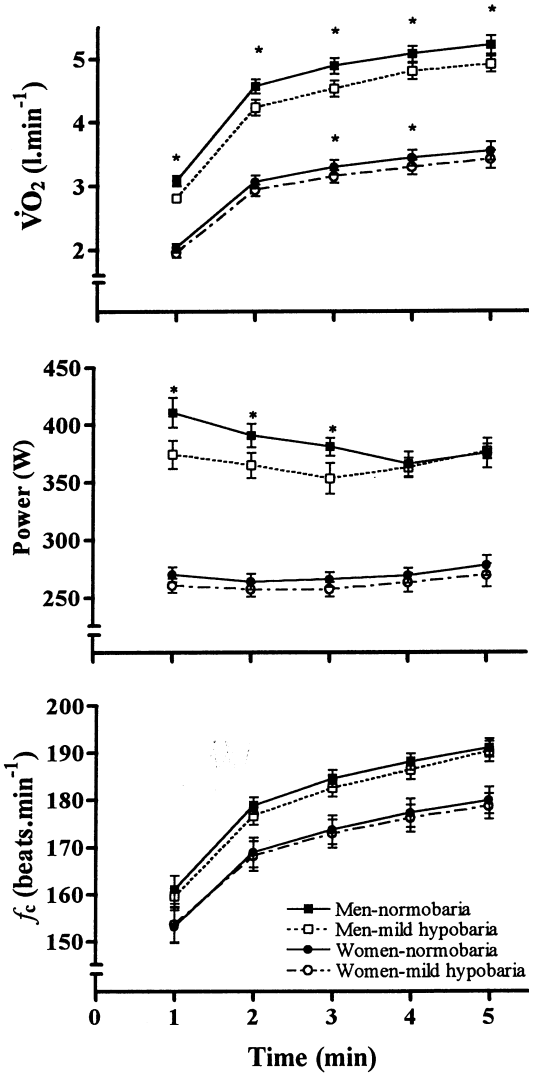

Fig. 1 Oxygen consumption ($\dot{V}O_2$) power output (in watts, W) and cardiac frequency (f_c) of endurance-trained male (n = 10) and female (n = 10) athletes during 5-min all-out performance tests on a cycle ergometer at normobaria (99.33 kPa) and at mild hypobaria (92.66 kPa). This hypobaria is equivalent to 580 m altitude referenced to sea level. Values are means (SEM). *Significantly different from normobaria, within-group comparison. Note that, at all times, the values for women are significantly different from those for the men at the matched chamber pressure

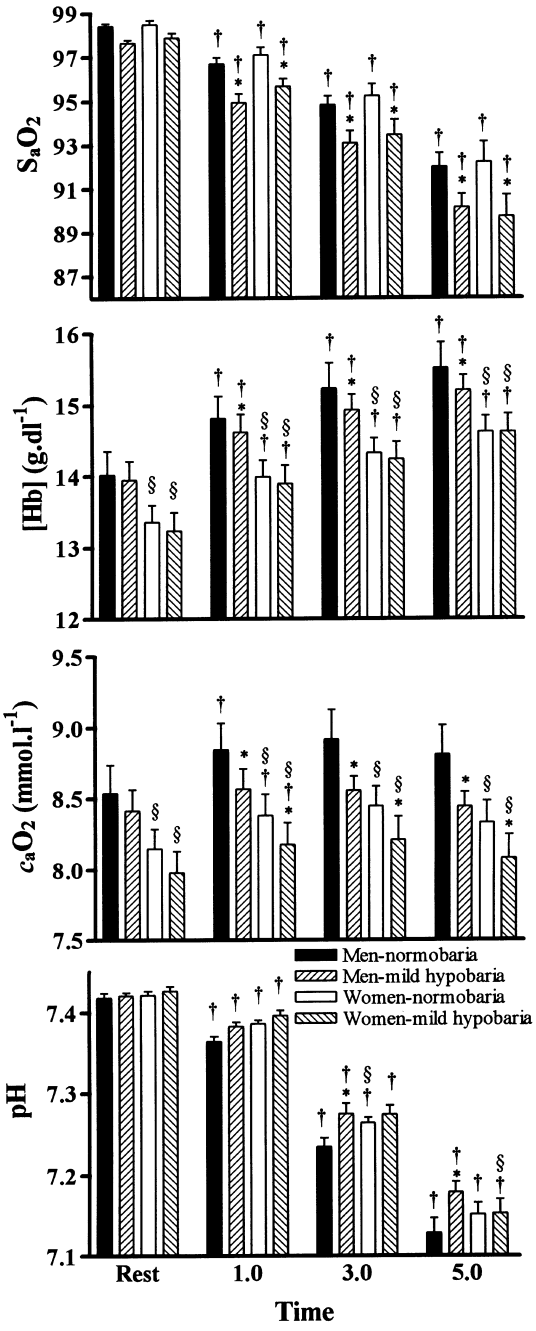

Fig. 2 Changes in arterial oxyhaemoglobin saturation (S_aO_2), haemoglobin concentration ([Hb]), arterial oxygen content (c_aO_2) and pH during 5-min performance tests on a cycle ergometer at normobaria (99.33 kPa) and at mild hypobaria (92.66 kPa). Values are means (SEM) for ten male and ten female endurance-trained athletes. *Significantly different from normobaria, within group comparison; † significantly different from previous time, within group comparison; § significantly different from men at matched chamber pressure

than at MH, at rest and throughout exercise and, furthermore, decreased significantly between successive measurement times.

Blood gases and associated variables

There was no significant difference in P_aO_2, P_aCO_2, P_AO_2 and $AaDO_2$ between men and women and, therefore, group data were pooled for each of these variables. At both N and MH, there was a significant decrease in P_aO_2 from resting values after 1 min of exercise, after which P_aO_2 was generally maintained throughout the remainder of exercise (Fig. 3). P_AO_2 and $AaDO_2$ increased significantly and progressively from rest values during exercise at both N and MH (Fig. 3). When data from all four measurement times were pooled, P_AO_2 was significantly lower at N than MH,

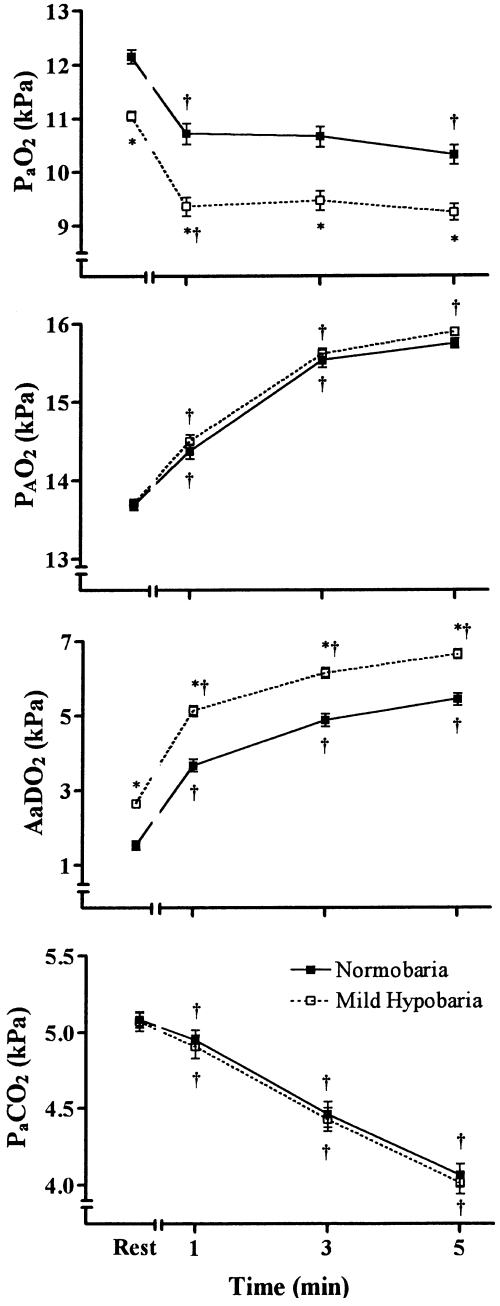

Fig. 3 Time course of arterial O_2 tension (P_aO_2), alveolar O_2 tension (P_AO_2), alveolar-arterial O_2 tension difference ($AaDO_2$) and arterial CO_2 tension (P_aCO_2) during 5-min performance tests on a cycle ergometer at normobaria (99.33 kPa) and at mild hypobaria (92.66 kPa). Values are means (SEM) for ten male and ten female endurance-trained athletes. The data for the men and women are pooled since they are not significantly different. *Significantly different from normobaria; †significantly different from previous time, within-condition comparison

$F_{(1,18)} = 5.6$, $P < 0.03$, while the $AaDO_2$ was significantly widened with hypobaria $F_{(1,18)} = 168.9$, $P < 0.0001$. P_aCO_2 exhibited a significant, progressive decrease from rest until after 5 min of exercise, with no significant difference between N and MH (Fig. 3). The pH response during exercise was different for men and women, depending upon chamber pressure, $F_{(3,54)} = 4.31$, $P < 0.01$. Overall, pH decreased with exercise time but was significantly higher at MH than N for men only after 3 and 5 min of exercise. While in the early stages of exercise at both N and MH pH was not different between men and women; the pH of the women was higher after 3 min of exercise at N, and lower after 5 min of exercise at MH (Fig. 2).

Oxygen content and haemoglobin

While there was no difference in resting values, c_aO_2 during exercise was significantly lower at MH than at N, $F_{(1,18)} = 11.93$, $P < 0.01$. At N for the pooled data of men and women, c_aO_2 increased significantly [≈ 3.3 (0.3)%] above resting values after 1 min and remained elevated throughout exercise (Fig. 2). At MH, however, men failed to increase c_aO_2 above resting levels at any time during exercise, while women exhibited an increase after 1 min but not after 3 and 5 min. Overall, men had a significantly higher [Hb] than women at rest and during exercise at both N and MH. There was a significant haemoconcentration from rest to 5 min of exercise in both men [N, 10.6 (0.6)%; MH, 8.9 (0.9)%] and women [N, 9.4 (0.5)%; MH 10.4 (0.7)%], although, the haemoconcentration of men was significantly greater during exercise at N than at MH (Fig. 2).

f_c and O_2 delivery

Although significantly lower in women than men, f_c was not significantly different between N and MH at each minute of exercise for both men and women (Fig. 1). Cardiac output is apparently unaffected by acute hypobaria (Stenberg et al. 1966) and, assuming cardiac outputs during 5 min of exercise of 30 $l \cdot min^{-1}$ for men and 25 $l \cdot min^{-1}$ for women, it can be calculated that there was a corresponding reduction in mean O_2 delivery of 198 $ml \cdot min^{-1}$ for men and 100 $ml \cdot min^{-1}$ for women from N to MH. The reduction in mean $\dot{V}O_2$ with hypobaria at min 5 was 297 $ml \cdot min^{-1}$ in men and 131 $ml \cdot min^{-1}$ in women, such that 76% of this reduction in men and 67% in women could be accounted for by a decrease in O_2 delivery.

Ventilation

Both $\dot{V}_{E\,STPD}$ and $\dot{V}_{E\,BTPS}$ increased significantly and progressively after 1 min of exercise in both the men and women at N and MH (Fig. 4) and, throughout exercise, men had significantly higher ventilation than women. While $\dot{V}_{E\,BTPS}$ at any minute of exercise was not significantly different between N and MH, $\dot{V}_{E\,STPD}$ was significantly decreased with hypobaria in both men and women. During exercise both $\dot{V}_{E\,BTPS}/\dot{V}O_2$ and $\dot{V}_{E\,BTPS}/\dot{V}CO_2$ of the women were significantly higher than those of the men. Within each group, the response

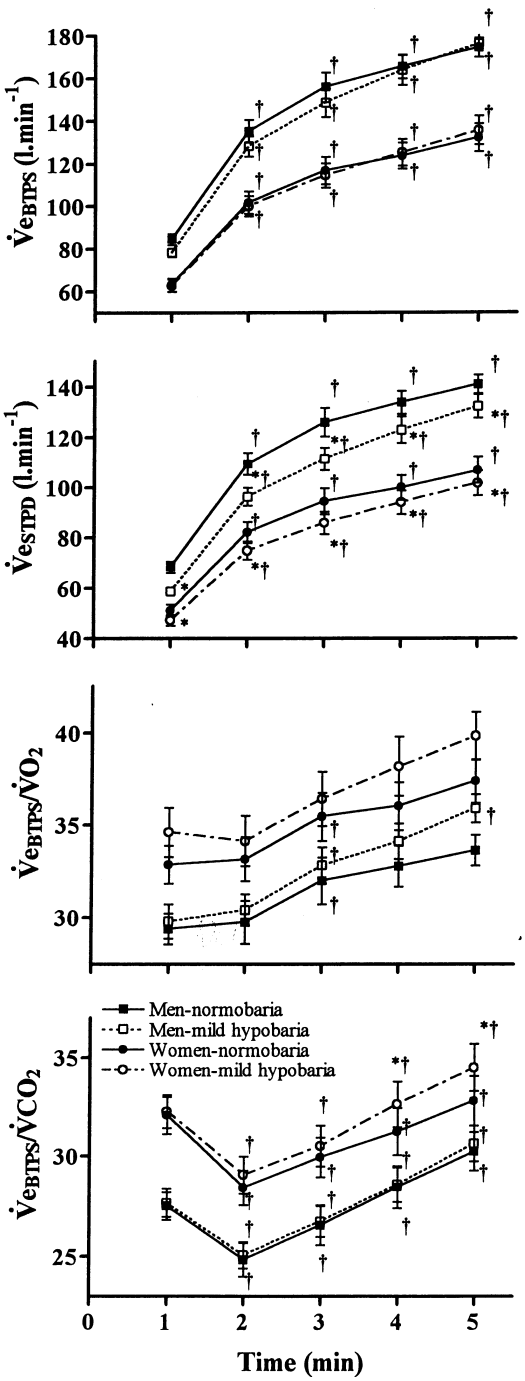

Fig. 4 Ventilatory response of endurance-trained male (n = 10) and female (n = 10) athletes during 5-min all-out performance tests on a cycle ergometer at normobaria (99.33 kPa) and at mild hypobaria (92.66 kPa). Ventilation is expressed as expired gas at standard temperature and pressure dry ($\dot{V}_{E STPD}$), expired gas at body temperature and pressue saturated ($\dot{V}_{E BTPS}$), ventilatory equivalent for O_2 ($\dot{V}_{E BTPS}/\dot{V}O_2$), and ventilatory equivalent for CO_2 ($\dot{V}_{E BTPS}/\dot{V}CO_2$). Values are means (SEM) *Significantly different from normobaria, within group comparison; †significantly different from previous time, winthin-group comparison. Note that, at all times, the values for women are significantly different from those for the men at the matched chamber pressure

of men and women was generally not different between N and MH, although the $\dot{V}_{E BTPS}/\dot{V}CO_2$ of women was significantly elevated after 4 and 5 min of exercise at MH compared with N (Fig. 4).

Discussion

This is the first study to demonstrate that a significant decrease in performance (total work) was concurrent with both reduced S_aO_2 and $\dot{V}O_{2peak}$. However, the strength of association of these relationships failed to reach significance because of the small and relatively homogeneous decrease in total work [– 3.9 (0.9)%]. Additionally, the decrease in performance at hypobaria equivalent to 580 m was not different for men or women, even though men experienced a significantly greater decrement in $\dot{V}O_{2peak}$. Other studies (Johnson et al. 1992; Lawler et al. 1988; Martin and O'Kroy 1993; Powers et al. 1992) have shown that hypoxic conditions that simulate altitudes of approximately 2000–3500 m accentuate EIH and reduce $\dot{V}O_{2max}$. However, $\dot{V}O_{2max}$ has limitations as an adequate assessment of performance (Snell and Mitchell 1984) and the only other study which has directly assessed the performance consequences of EIH (Koskolou and McKenzie 1994) reported that moderate hypoxaemia, induced by 16.8% inspired oxygen, was associated with reduced performance (≈ 4.5%) but did not alter $\dot{V}O_{2max}$.

Koskolou and McKenzie (1994) suggested that their result may be a consequence of enhanced muscle blood flow as well as enhanced O_2 extraction at the tissue. On the other hand, the present data support the proposal of Warren et al. (1991) that c_aO_2 should decrease if exercise-induced hypoxemia is to have a negative impact on exercise performance. The normal response to exercise is haemoconcentration (Harrison 1985) with a consequent increase in c_aO_2 above resting levels (Warren et al. 1991). This response was seen in both men and women at normobaria; however, both sexes had significantly lower c_aO_2 after 1, 3 and 5 min of exercise at MH compared with N. At the corresponding times, the $\dot{V}O_2$ of both men and women was reduced and $\dot{W}$ was lower, although only significantly so after 1 and 3 min for men. After 5 min of exercise at MH, the women were unable to increase c_aO_2 above resting levels, despite a similar degree of haemoconcentration to that at N, while the men experienced both lower haemoconcentration than at N and c_aO_2 that was not different from that at rest. It is likely that the decrease in S_aO_2, c_aO_2 and $\dot{V}O_2$ with hypobaria may be attributed to a significantly lower P_aO_2 during exercise at MH, since the P_aO_2 after 1 min of exercise in both men and women fell much closer to the steeper portion of the O_2 loading curve for haemoglobin [N, 10.7 (0.2) kPa; MH, 9.4 (0.2) kPa]. Compared with N conditions, the reduction in c_aO_2 and calculated oxygen delivery after 5 min of exercise in both the men and women when exercising under hypobaric conditions accounted for 67–76% of the decrease in $\dot{V}O_2$.

This range is similar to the 71.2% reported by Lawler et al. (1988) who used hypoxia (14% O_2) to reduce c_aO_2 and $\dot{V}O_{2peak}$, and similar to the 70.5% reported by Gore et al. (1996) when using identical hypobaria for a progressive $\dot{V}O_{2max}$ test.

This is also the first study to demonstrate that EIH at $\dot{V}O_{2peak}$ is not significantly different between endurance-trained male and female athletes, either at N (92.0 versus 92.1%, respectively) or at MH (90.1 versus 89.9%, respectively). Although McCusker and Brilla (1992) reported an S_aO_2 of 85% at $\dot{V}O_{2max}$ in trained female athletes, their results were obtained indirectly using ear oximetry, which may overestimate the incidence of EIH (Brown et al. 1993; Norton et al. 1992). In contrast, Pedersen et al. (1992) reported that five well-trained female athletes did not experience EIH at $\dot{V}O_{2max}$, with a radial artery S_aO_2 of ≈ 96%. The present findings challenge the proposal of Powers and Williams (1987) of an absolute $\dot{V}O_{2max}$ of 4–5 $l \cdot min^{-1}$ for development of EIH, since the normobaric $\dot{V}O_{2peak}$ of women was 3.6 $l \cdot min^{-1}$. Relative, rather than absolute, $\dot{V}O_{2peak}$ may be a more appropriate predictor since the female athletes had a mean $\dot{V}O_{2peak}$ of 61 $ml \cdot kg^{-1} \cdot min^{-1}$.

While the precise mechanism of EIH remains equivocal (Powers et al. 1993), one theory suggests an inadequate hyperventilatory response (Dempsey et al. 1984) for which the cardinal marker is a rise in P_aCO_2 above resting values. In the present study, P_aCO_2 during exercise fell progressively below resting values at N and MH in both men and women, and $\dot{V}_{E\ BTPS}$ was not significantly different between N and MH. These results are consistent with the findings of Powers et al. (1992). Also consistent with the work of others (Johnson et al. 1992; Powers et al. 1992; Wagner et al. 1986) is the widened $AaDO_2$ that accompanied EIH. Although the P_AO_2 was not different between N and MH, $AaDO_2$ was significantly increased at rest and throughout exercise with hypobaria, but this was consequent to the 1.3-kPa fall in P_aO_2 at rest, and was not exacerbated by exercise. The pH is an indicator of the effectiveness of alveolar ventilation ($\dot{V}_A$) to reduce P_aCO_2 relative to the high $\dot{V}CO_2$ during exercise (Whipp and Pardy 1986). In the current study, pH of the women was not different between N and MH, while the men had a significantly higher pH at MH than N, after 3 and 5 min of exercise. While the higher pH at MH may be partially a function of lower absolute work intensity, enhanced EIH at MH has the "benefit" of improved buffering capacity of reduced haemoglobin. Although the pH of men and women was somewhat different (possibly related to different total Hb mass and [Hb]), metabolic acidosis was not increased with greater hypoxaemia, as speculated by Koskolou and McKenzie (1994). Rather than incurring the metabolic cost to the respiratory muscles of greater $\dot{V}_A$ to reduce further P_aCO_2 and limit the fall in pH, the considerable buffering capacity of reduced Hb as a consequence lower S_aO_2 may be energetically more efficient. Hopkins and McKenzie 1993) and Otis (1964) have also speculated that increasing ventilation in an attempt to lessen metabolic acidosis may actually worsen it by increasing the O_2 consumption of the respiratory muscles.

In conclusion, this is the first study to show that a significant decrease in performance (≈ 3.7% decrease in total work for both men and women) was concurrent with both reduced S_aO_2 and $\dot{V}O_{2peak}$ at hypobaria equivalent to 580 m altitude. This study also demonstrate that hypoxaemia at $\dot{V}O_{2peak}$ was not significantly different between endurance-trained male and female athletes. Therefore, relative rather than absolute, $\dot{V}O_{2peak}$ may be a more appropriate predictor of exercise-induced hypoxaemia. For men and women, it was calculated that 67–76% of the decrease in $\dot{V}O_{2peak}$ could be accounted for by a decrease in O_2 delivery, which indicates that reduced P_iO_2 at even mild altitude may have ramifications for performance in events with high aerobic energy demands.

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