Increased arterial desaturation in trained cyclists during maximal exercise at 580 m altitude

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Gore, C. J., A. G. Hahn, G. C. Scroop, D. B. Watson, K. I. Norton, R. J. Wood, D. P. Campbell, and D. L. Emonson. Increased arterial desaturation in trained cyclists during maximal exercise at 580 m altitude. *J. Appl. Physiol.* 80(6): 2204–2210, 1996.—This study utilized a hypobaric chamber to compare the effects of mild hypobaria (MH; 50 mmHg, ~580 m altitude) on blood O_2 status and maximal O_2 consumption ($\dot{V}O_{2\max}$) in 9 untrained and 11 trained (T) cyclists with $\dot{V}O_{2\max}$ values of 51 ± 3 and 77 ± 1 $ml \cdot kg^{-1} \cdot min^{-1}$, respectively. In both groups, arterial O_2 saturation (Sa_{O_2}) decreased significantly during maximal exercise, and this effect was enhanced with MH. Both these responses were significantly greater in the T cyclists in whom the final Sa_{O_2} during MH was 86.5 ± 0.9%. When the group data were combined, ~65% of the variance in Sa_{O_2} could be attributed to a widened alveolar-arterial P_{O_2} difference. The arterial P_{O_2} during maximal exercise at sea level in the T group was on the steeper portion of the hemoglobin-O_2-loading curve (T, 68.3 ± 1.3 Torr; untrained, 89.0 ± 2.9 Torr) such that a similar decrease in arterial P_{O_2} in the two groups in response to MH resulted in a significantly greater fall in both Sa_{O_2} and calculated O_2 content in the T group. As a consequence, the $\dot{V}O_{2\max}$ fell significantly only in the T group (mean change, −6.8 ± 1.5%; range, +1.2 to −12.3%), with ~70% of this decrease being due to a fall in O_2 content. This is the lowest altitude reported to decrease $\dot{V}O_{2\max}$, suggesting that T athletes are more susceptible to a fall in inspired P_{O_2}.

maximal oxygen consumption; hypobaria; hypoxemia

WITH INCREASING ALTITUDE above sea level, there is a progressive decrease in the P_{O_2} of the inspired air (PI_{O_2}), yet, up to ~1,600 m, both the sigmoidal shape of the O_2-loading curve for hemoglobin and an increase in alveolar ventilation are believed to prevent any significant change in either the arterial O_2 saturation (Sa_{O_2}) of hemoglobin or maximal aerobic power [maximal O_2 consumption ($\dot{V}O_{2\max}$)] (16, 28). However, many endurance-trained athletes, even when at sea level with a normal PI_{O_2}, exhibit a decrease in Sa_{O_2} on exercise (18). Although the mechanisms are uncertain (4, 6, 18, 25), it would seem reasonable to hypothesize that, in such individuals, any increase in altitude and consequent fall in PI_{O_2} would exacerbate this effect and reduce aerobic power in such individuals. Within Australia, $\dot{V}O_{2\max}$ testing of endurance athletes in training is done mostly in the coastal cities, which lie at sea level, and before team selection and is repeated at the Australian Institute of Sport in the national capital Canberra where the altitude is ~600 m. A preliminary comparison in a small group of endurance-trained and untrained individuals indicated a fall in $\dot{V}O_{2\max}$ in the trained group on moving from the coast to Canberra despite the modest increase in altitude. The only other study to examine the effect of modest altitude on the aerobic power of endurance-trained athletes reported a 7.5% decrease in $\dot{V}O_{2\max}$ at 900 m (24). There have been no studies at lower altitudes. To substantiate our hypothesis, the present experiments were conducted in a group of endurance-trained and untrained subjects who performed maximal exercise tests on a cycle ergometer at both sea level and ~600 m. All tests were performed in a random order in a hypobaric chamber while measurements were made of arterial blood gases and the common cardiorespiratory variables.

METHODS

Subjects. Twenty healthy men who were lifetime nonsmokers and had no history of asthma gave written consent to participate in the study, which was approved by the Australian Defence Medical Ethics Committee. The subjects were either trained (T) cyclists involved in high-level competition or untrained (UT) physically active men, and their characteristics are presented in Table 1. The respiratory history of all the subjects was screened with an International Union Against Tuberculosis Questionnaire (1), and lung function was assessed with spirometric tests (AS6000 autospirometer, Minato, Osaka, Japan). Spirometry for forced vital capacity and forced expiratory volume in 1 s was conducted with the subjects seated and wearing a noseclip, with all subjects allowed a minimum of three trials. Compared with Australian normal values (5), the mean scores for forced vital capacity and forced expiratory volume in 1 s were >93% of the predicted values, and there were no significant differences between the groups.

Test protocol. The experimental protocol required each subject to complete two maximal exercise tests on a geared wind-braked cycle ergometer (B. L. Hayes, Adelaide Superdrome) with a minimum of 24 h of rest between tests. One test was conducted at "sea level," and a second test was conducted at 580 m "altitude," with the order of the tests counterbalanced and double blinded. The T group began their test with a workload of 200 W, whereas the UT group began at 100 W. With both groups, the workload was increased 25 W each minute until volitional exhaustion. The saddle height selected by the subjects for their first trial was measured and

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Table 1. *Physical characteristics of the 9 untrained and 11 trained athletes at normobaria*

	Untrained	Trained
Age, yr	27.1 ± 2.8	23.3 ± 1.5
Height, cm	179.5 ± 1.6	179.4 ± 1.5
Mass, kg	78.05 ± 2.16	71.36 ± 1.09*
FVC, liters	5.32 ± 0.24	5.44 ± 0.21
FEV_1, liters	4.52 ± 0.22	4.49 ± 0.16
$\dot{V}O_{2max}$, $ml \cdot kg^{-1} \cdot min^{-1}$	51 ± 3	77 ± 1*

Values are means ± SE. FVC, forced vital capacity; FEV_1, forced expiratory volume in 1 s; $\dot{V}O_{2max}$, maximal O_2 consumption. *Significant difference from untrained group, $P < 0.05$.

replicated for their second trial. Power output during each test was assessed with Schoberer Rad Messtechnik (SRM) cranks (Ingenieurbüro Schoberer, Jülich, Germany), with the information recorded on a data logger and downloaded into a personal computer. The accuracy of the SRM cranks was verified before the study commenced with a first-principles calibration rig (22). All 20 subjects completed their 2 tests within 5 days. All tests were conducted in a hypobaric chamber (Thompsons, Castlemaine, Australia) located at the Royal Australian Air Force (RAAF) Base Williams, Melbourne, Australia. The chamber was 6.1 m long and 2.4 m in diameter and during operation had an ambient air refresh rate of 2,265 l/min. Barometric conditions in the chamber for the two tests were selected to approximate the lowest barometric pressure that might be encountered either at sea level [745 mmHg; normobaria (N)] or at Canberra [695 mmHg; mild hypobaria (MH)]. The 50-mmHg pressure differential equates to an altitude difference of 580 m (7) because N equals an altitude of 168 m and MH equals an altitude of 748 m. The times of ascent and descent of the chamber from ambient sea-level pressure to N or MH were matched to prevent subjects and experimenters from guessing the chamber pressure. The altimeters used by the RAAF technical staff to set the chamber pressure were calibrated in the week before data collection by Ansett Industries (Melbourne, Australia) in accord with RAAF specifications (19). Records of chamber pressure stability during each test indicated a SD of <0.5 mmHg. Temperature (in °C) and relative humidity (in percent) inside the chamber were monitored electronically with a Testoterm thermohygrometer model 610 (Lenzkirk, Germany) calibrated against National Association of Testing Authorities master instruments. The readings were not significantly different between N and MH for either group (UT: temperature, 25.8°C in N, 24.1°C in MH; humidity, 45.7% in N, 42.8% in MH; T: temperature, 25.1°C in N, 25.2°C in MH; humidity, 42.1% in N, 40.3% in MH). The temperature range for both groups was 21.6–29.0°C in N and 17.7–28.0°C in MH. The relative humidity range for both groups was 32.4–58.0% in N and 33.0–48.0% in MH.

O_2 consumption ($\dot{V}O_2$). $\dot{V}O_2$ was measured each minute with an open-circuit indirect calorimetry system based on the design of Wilmore and Costill (27). Inspired volume was measured with a Morgan ventilometer (P. K. Morgan, Rainham, Kent, UK) attached to a R2700 respiratory valve (Hans Rudolph, Kansas City, MO). The ventilometer was calibrated with a 1.0-liter syringe in accord with the manufacturer's specifications, and this was verified by delivering 60, 120, and 180 l/min with a 3.0-liter Hans Rudolph calibration syringe. Expired gases passed through 1.0 m of tubing (Vacumed, Cleanbor) to a 2.6-liter mixing chamber (Sportech, Australian Capital Territory, Australia) (total system dead space ~3.7 liters) from which mixed expired air was subsampled with a diaphragm pump (model 7530-50, Cole Parker Instruments, Chicago, IL) at a rate of ~2.0 l/min and passed into evacuated 3.0-liter anesthetic bladders. The pump diaphragm was checked for leaks twice a day by passing a sample of α-standard calibration gas (BOC Gases Australia) through the pump from one anesthetic bladder to another. No leaks were detected on any day of testing. Anesthetic bladders filled with expired air were delivered manually to Ametek S-3A O_2 and CD-3A CO_2 analyzers via a 15-cm drying tube filled with $CaCl_2$. These analyzers were located within the chamber and had been calibrated previously with three α-standard gases that spanned the physiological range. All components of the open-circuit indirect calorimetry system were calibrated only after the chamber pressure was signaled as stable, and at the end of each test, the analyzer and ventilometer calibrations were checked before the chamber pressure was returned to ambient. In more than one-half of the experiments, the gas analyses performed within the hypobaric chamber were verified subsequently on analyzers located outside the chamber (MedGraphics Cardiopulmonary Exercise System CPX/D, St. Paul, MN) that were calibrated with MedGraphics calibration gas (7% CO_2-16% O_2-balance N_2). For each of these checks, the CPX/D system was also calibrated with two of the three α-standard gases used to calibrate the Ametek analyzers.

Arterial blood gases. Before each test, a catheter (Cathlon iv 20 gauge, 1.5 mm OD, 32 mm length) was inserted into a brachial artery under local anesthesia (2% lignocaine hydrochloride) under standard aseptic procedures. Arterial blood samples (2.5 ml) were taken into heparinized ground-glass syringes at rest and at $\dot{V}O_{2max}$. Samples were capped immediately, stored vertically on ice and, after prior mixing, were analyzed in duplicate for blood gases [arterial PO_2 (Pa_{O_2}), arterial PCO_2 (Pa_{CO_2}), and pH] on a Ciba Corning blood gas system model 278 (Medfield, MA). Triplicate analyses were conducted for Sa_{O_2} and hemoglobin concentration [Hb] on a Ciba Corning CO-oximeter model 270. Two-point calibration of the blood gas analyzer was completed every 2 h with known standards purchased from the manufacturer, with two quality controls (Bio-Rad, Anaheim, CA) assayed approximately every hour. The CO-oximeter was calibrated on the day before the study commenced, and quality controls were assayed on each morning of testing. Both calibrant and control solutions were purchased from Ciba Corning. Blood gas and Sa_{O_2} values were all measured at 37°C. The ideal alveolar gas equation (15) was used to estimate the alveolar PO_2 (PA_{O_2}) and the alveolar-arterial PO_2 difference ($A\text{-}aPO_2$). The O_2 content (CO_2) of arterial blood was calculated according to the method of Siggaard-Andersen et al. (20).

Expression of results and statistical analysis. Unless otherwise stated, the results are expressed as the means ± SE. Repeated-measures three-way analysis of variance (Statistica/W, StatSoft, Tulsa, OK) was used to determine differences among exercise intensity (rest and $\dot{V}O_{2max}$), chamber pressure (N and MH), and athlete group (UT vs. T) for the blood (Sa_{O_2}, Pa_{O_2}, Pa_{CO_2}, PA_{O_2}, $A\text{-}aPO_2$, pH, [Hb] and CO_2), metabolic [$\dot{V}O_2$ and CO_2 production ($\dot{V}CO_2$)] and ventilatory [ventilation ($\dot{V}E$) STPD, $\dot{V}E$ BTPS, the ventilatory equivalent for O_2 ($\dot{V}E$BTPS/$\dot{V}O_2$), and the ventilatory equivalent for CO_2 ($\dot{V}E$BTPS/$\dot{V}CO_2$)] data. Two-way analysis of variance was used when time was not an independent variable. The highest order interactions or main effects were investigated with Tukey's honestly significant difference tests between means. The relationships among a number of the respiratory, saturation, and blood gas variables were also examined with Pearson product moment correlations. Student's *t*-tests for independent samples were used to compare the biometric characteristics of the two groups. The significance level was set at $P < 0.05$ for all analyses.

Table 2. *Metabolic, heart rate, and ventilatory responses at $\dot{V}O_{2max}$ during normobaria (745 mmHg) and hypobaria (695 mmHg)*

	Untrained		Trained	
	Normobaria	Hypobaria	Normobaria	Hypobaria
$\dot{V}O_2$, l/min	3.93 ± 0.18	3.79 ± 0.16	5.48 ± 0.09*	5.10 ± 0.08*†
$\dot{V}CO_2$, l/min	4.66 ± 0.20	4.65 ± 0.20	6.35 ± 0.13*	5.94 ± 0.13*†
Heart rate, beats/min	189 ± 3	189 ± 3	190 ± 3	186 ± 3
$\dot{V}E$ BTPS, l/min	149.4 ± 8.8	151.8 ± 7.8	180.1 ± 5.6*	175.3 ± 6.1*
$\dot{V}E$ STPD, l/min	120.9 ± 7.1	114.0 ± 5.9	145.7 ± 4.5*	131.6 ± 4.6*†
$\dot{V}E$ BTPS/$\dot{V}O_2$	38.2 ± 1.8	40.4 ± 2.0	32.9 ± 0.8*	34.3 ± 0.8*
$\dot{V}E$ BTPS/$\dot{V}CO_2$	31.9 ± 1.3	32.6 ± 1.4	28.2 ± 0.8*	29.3 ± 0.8*

Values are means ± SE. $\dot{V}O_2$, O_2 consumption; $\dot{V}CO_2$, CO_2 production; $\dot{V}E$, ventilation; $\dot{V}E$ BTPS/$\dot{V}O_2$, ventilatory equivalent for O_2; $\dot{V}E$ BTPS/$\dot{V}CO_2$, ventilatory equivalent for CO_2. * Significant difference from untrained group, $P < 0.05$. † Significant difference from normobaria, $P < 0.05$.

RESULTS

$\dot{V}O_2$. Even though the $\dot{V}O_{2max}$ of the UT group was unchanged from N to MH, that of the T group decreased significantly (N, 5.48 ± 0.09 l/min; MH, 5.10 ± 0.08 l/min; Table 2), representing a mean change of −6.8 ± 1.5%, with a range of +1.2 to −12.3%. For either group, there was no significant correlation between sea level $\dot{V}O_{2max}$ and the change in (Δ) $\dot{V}O_{2max}$ from N to MH, and only a weak correlation when the groups were combined ($r = 0.46$; $P = 0.04$; Fig. 1). Thus there was an ~80% unexplained variance in $\Delta\dot{V}O_{2max}$ for a given $\dot{V}O_{2max}$ in N. The exercise time to reach $\dot{V}O_{2max}$ was longer in the T group (T, 11. 0 ± 0.2 min; UT, 9.3 ± 0.3 min; $F_{1,18} = 10.34$; $P < 0.005$), and this time did not change significantly with chamber pressure for either group, although the time to exhaustion in the T group was shorter in MH (N, 11.3 ± 0.3 min; MH, 10.7 ± 0.4 min). When the data from both groups were pooled, the total work completed was lower with MH (N, 174.8 ± 12.4 kJ; MH, 166.5 ± 12.0 kJ; $F_{1,18} = 4.63$; $P = 0.045$). When the data from both normobaria and MH were pooled, the total work was greater for the T group (T, 217.6 ± 5.4 kJ; UT, 123.6 ± 5.7 kJ; $F_{1,18} = 78.9$; $P < 0.0001$). The average power during the minute that the $\dot{V}O_{2max}$ was achieved was also higher for the T group (T, 445 ± 5 W; UT, 306 ± 12 W; $F_{1,18} = 76.0$; $P < 0.0001$), but it was not reduced significantly with MH for either the UT (N, 305 ± 20 W; MH, 306 ± 17 W) or the T group (N, 455 ± 8 W; MH, 435 ± 4 W).

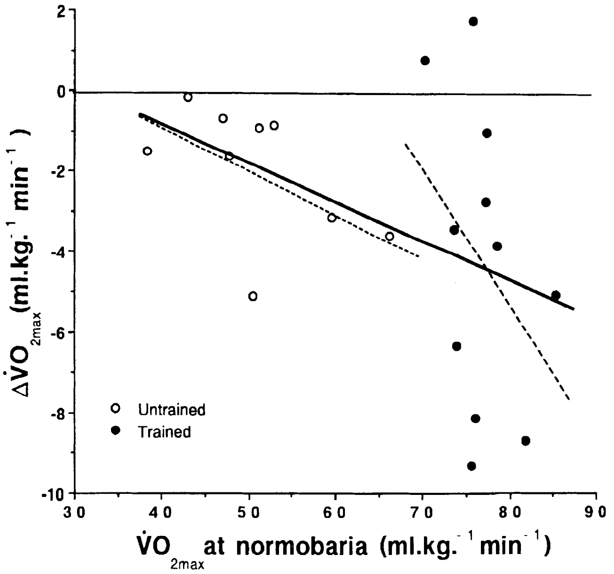

Fig. 1. Relationship between maximal aerobic power [maximal O_2 consumption ($\dot{V}O_{2max}$)] at normobaria and change in $\dot{V}O_{2max}$ ($\Delta\dot{V}O_{2max}$) from normobaria (745 mmHg) to mild hypobaria (695 mmHg) in untrained cyclists ($r = 0.52$; $P = 0.15$; short-dashed line), trained cyclists ($r = 0.35$; $P = 0.29$; long-dashed line), and all subjects ($r = 0.46$; $P = 0.04$; solid line).

Sa_{O_2}. At rest, the mean Sa_{O_2} was not significantly different between groups at either chamber pressure (Table 3). At $\dot{V}O_{2max}$, Sa_{O_2} decreased significantly in both subject groups, although in the T group this effect was more marked and more pronounced under hypobaric conditions (Fig. 2, Table 3). The three-way interaction for Sa_{O_2} among exercise intensity, athlete group, and chamber pressure was significant ($F_{1,18} = 10.63$; $P = 0.004$). When the groups were considered individually, there was no significant correlation between Sa_{O_2} and A-aP_{O_2} at either altitude, but when the groups were combined, the correlations were significant at both altitudes (N, $r = -0.82$; MH, $r = -0.86$). There was no significant correlation between ΔSa_{O_2} from N to MH and the corresponding $\Delta\dot{V}O_{2max}$ whether the groups were analyzed individually (T, $r = 0.11$, $P = 0.79$; UT, $r = 0.09$, $P = 0.82$) or combined ($r = 0.25$; $P = 0.29$).

C_{O_2}. The hemoconcentration from rest to $\dot{V}O_{2max}$ was similar (~8.5%) for both groups and independent of altitude (Fig. 2). However, although the C_{O_2} of the UT group increased significantly from rest to maximal exercise in both N (7.0 ± 0.9%) and MH (5.4 ± 1.3%), that of the T group did not change in N and decreased significantly in MH (−3.8 ± 0. 8%; (Table 3, Fig. 2). The relative change in C_{O_2} at $\dot{V}O_{2max}$ for the T group was 4.5% lower in MH (Table 3) than in N. The three-way interaction for C_{O_2} among exercise intensity, athlete group, and chamber pressure was significant ($F_{1,18} = 4.63$; $P = 0.0454$; Fig. 2). Maximal heart rate was unaffected by altitude (Table 2), and if a maximal cardiac output of 30 l/min for the T subjects at both chamber pressures is assumed (23), O_2 delivery can be calculated as 5.972 l/min in N and 5.704 l/min in MH. The measured mean decrease in $\dot{V}O_{2max}$ from N to MH was 380 ml/min, whereas the predicted decrease in O_2 delivery was 268 ml/min. Thus an average of 70.5% in the decrease in $\dot{V}O_{2max}$ could be accounted for by a decrease in O_2 delivery.

Table 3. *Sa_{O_2}, blood gases, [Hb], and C_{O_2} at rest and $\dot{V}O_{2max}$ under normobaric (745 mmHg) and hypobaric (695 mmHg) conditions*

	Rest				$\dot{V}O_{2max}$			
	Untrained		Trained		Untrained		Trained	
	Normobaria	Hypobaria	Normobaria	Hypobaria	Normobaria	Hypobaria	Normobaria	Hypobaria
Sa_{O_2}, %	97.3 ± 0.2	96.5 ± 0.2	97.7 ± 0.1	97.1 ± 0.2	94.7 ± 0.7†	93.7 ± 0.6†	90.4 ± 0.5*†	86.5 ± 0.9*†‡
Pa_{O_2}, Torr	91.3 ± 1.5	82.2 ± 1.6‡	97.7 ± 1.5*	88.5 ± 1.2*‡	89.0 ± 2.9	81.3 ± 2.8‡	68.3 ± 1.3*†	61.5 ± 0.8*†‡
Pa_{CO_2}, Torr	40.2 ± 0.8	39.9 ± 0.8	40.5 ± 0.7	40.1 ± 0.6	32.4 ± 1.0†	32.5 ± 1.0†	36.7 ± 1.1*†	34.9 ± 0.8*†
$P_{A_{O_2}}$, Torr	99.1 ± 1.8	91.3 ± 1.7‡	99.9 ± 0.9	89.9 ± 0.7‡	115.3 ± 1.3†	108.7 ± 1.5†‡	112.3 ± 1.2†	103.9 ± 0.7*†‡
A-aP_{O_2}, Torr	7.8 ± 1.6	9.1 ± 2.5	2.2 ± 1.2	1.4 ± 0.9*	26.2 ± 2.8†	27.4 ± 1.9†	44.0 ± 1.4*†	42.4 ± 1.1*†
pH	7.414 ± 0.007	7.424 ± 0.002	7.414 ± 0.006	7.415 ± 0.004	7.228 ± 0.021†	7.250 ± 0.018†	7.242 ± 0.019†	7.225 ± 0.016†
[Hb], g/dl	15.2 ± 0.4	15.5 ± 0.4	14.8 ± 0.2	14.8 ± 0.2*	16.7 ± 0.4†	16.7 ± 0.5†	16.1 ± 0.2*†	16.0 ± 0.3*†
C_{O_2}, mmol/l	9.14 ± 0.22	9.20 ± 0.23	8.98 ± 0.11	8.93 ± 0.15*	9.77 ± 0.23†	9.70 ± 0.28†	8.99 ± 0.13*	8.59 ± 0.15*†‡

Values are means ± SE. Sa_{O_2}, arterial O_2 saturation; Pa_{O_2}, arterial P_{O_2}; Pa_{CO_2}, arterial P_{CO_2}; $P_{A_{O_2}}$, alveolar P_{O_2}; A-aP_{O_2}, alveolar-arterial P_{O_2} difference; [Hb], hemoglobin concentration; C_{O_2}, O_2 content. *Significant difference from untrained group at matched intensity and chamber pressure, $P < 0.05$. †Significant difference from rest (within group comparison, $P < 0.05$). ‡Significant difference from normobaria (within group comparison, $P < 0.05$).

Blood gases and associated variables. In the present study, which used a repeated-measures design, all blood gas measurements were made at 37°C and no temperature correction was made. However, it is likely that within each group the core temperatures at $\dot{V}O_{2max}$ were similar in both N and MH because the time to exhaustion and average power output at $\dot{V}O_{2max}$ were not different. Furthermore, because the pH at $\dot{V}O_{2max}$ in both N and MH also was not different, it is likely that the blood gas data at both altitudes are directly comparable. Comparisons between groups without correcting for temperature must be considered more cautiously because the total work was significantly greater in the T group and the blood temperatures achieved may have been different. A similar limitation applies to the interpretation of pooled group data.

The trends in Pa_{O_2}, $P_{A_{O_2}}$, A-aP_{O_2}, and Pa_{CO_2} with exercise intensity were group specific and were not affected by altitude. Therefore, the N and MH data for each group have been pooled, and the interactions between group and intensity are illustrated in Fig. 3. At $\dot{V}O_{2max}$, the Pa_{O_2} and $P_{A_{O_2}}$ were significantly lower in the T group, whereas the A-aP_{O_2} and Pa_{CO_2} were significantly higher. In addition to the interactions between group and intensity, the Pa_{O_2} pooled for both groups and exercise intensities was lower under MH conditions (N, 86.6 Torr; MH, 78.4 Torr; $F_{1,18} = 68.63$; $P < 0.0001$), as was the $P_{A_{O_2}}$ (N, 106.6 Torr; MH, 98.5 Torr; $F_{1,18} = 43.31$; $P < 0.0001$). With regard to pH, the highest order effect was the main effect of exercise intensity ($F_{1,18} = 271.43$; $P < 0.0001$), with pH decreasing throughout exercise from rest (7.417) to $\dot{V}O_{2max}$ (7.236). Summary data for the blood gas, pH, and concentration variables are presented in Table 3.

$\dot{V}_E$. When pooled across both groups, $\dot{V}_E$ STPD at $\dot{V}O_{2max}$ was significantly lower under MH conditions (N, 133.3 l/min; MH, 122.8 l/min; $F_{1,18} = 14.78$; $P = 0.001$). When pooled across the two chamber pressures, $\dot{V}_E$ STPD was significantly higher for the T group (T, 138.6 l/min; UT, 117.4 l/min; $F_{1,18} = 8.59$; $P = 0.009$) and similarly for $\dot{V}_E$ BTPS (T, 177.7 l/min; UT, 150.6 l/min; $F_{1,18} = 8.51$; $P = 0.009$). However, the main effect for chamber pressure was not significant for $\dot{V}_E$ BTPS. The $\dot{V}_E$ BTPS/$\dot{V}O_2$ was lower for the T group (T, 33.6 ± 0.4; UT, 39.3 ± 1.0; $F_{1,18} = 9.60$; $P = 0.006$) and lower under N conditions (N, 35.2 ± 0.8; MH, 37.1 ± 0.8; $F_{1,18} = 9.11$; $P = 0.007$). These main effects for group and chamber pressure were the highest order effects. When the data from all subjects were pooled, there was a significant correlation between $\dot{V}_E$ BTPS/$\dot{V}O_2$ and Sa_{O_2} in both N ($r^2 = 0.46$; $P < 0.005$) and MH ($r^2 = 0.36$; $P < 0.01$) and between $\dot{V}_E$ BTPS/$\dot{V}O_2$ and Pa_{O_2} in both N ($r^2 = 0.50$; $P < 0.001$) and MH ($r^2 = 0.51$; $P < 0.0005$). Thus ~40% of the variability in both Sa_{O_2} and Pa_{O_2} can be explained by the variability in $\dot{V}_E$ BTPS/$\dot{V}O_2$. The $\dot{V}_E$ BTPS/$\dot{V}CO_2$ also was lower for the T group (T, 28.8 ± 0.4; UT, 32.3 ± 0.7; $F_{1,18} = 5.51$; $P = 0.03$) and lower in N (N, 29.9 ± 0.6; MH, 30.8 ± 0.6; $F_{1,18} = 5.54$; $P = 0.03$). When the data from all subjects were pooled, there was a significant correlation between $\dot{V}_E$ BTPS/$\dot{V}CO_2$ and Sa_{O_2} in both N ($r^2 = 0.45$; $P < 0.005$) and MH ($r^2 = 0.32$; $P < 0.01$) and between $\dot{V}_E$ BTPS/$\dot{V}CO_2$ and Pa_{O_2} in both N ($r^2 = 0.45$; $P < 0.05$) and MH ($r^2 = 0.36$; $P < 0.01$). Thus ~40% of the variability in both Sa_{O_2} and Pa_{O_2} can be explained by the variability in $\dot{V}_E$ BTPS/$\dot{V}CO_2$.

DISCUSSION

This is the first study to report a significant decrease (6.8%) in $\dot{V}O_{2max}$ in T subjects at 580 m altitude (50 mmHg hypobaria), although a 7.5% decrease has been reported at 900 m (24). Both studies used T subjects of similar aerobic power and reported no significant effect of such MH in their UT control subjects. These results suggest that the concept of a "threshold" altitude for aerobic impairment (16, 28) may be misleading in the case of the T athletes and support the proposal of Squires and Buskirk (21) that the aerobic power of T individuals decreases progressively as one begins to ascend from sea level. Although previous research has suggested a strong correlation between sea-level $\dot{V}O_{2max}$ and the altitude-induced decrement (10, 11), no such

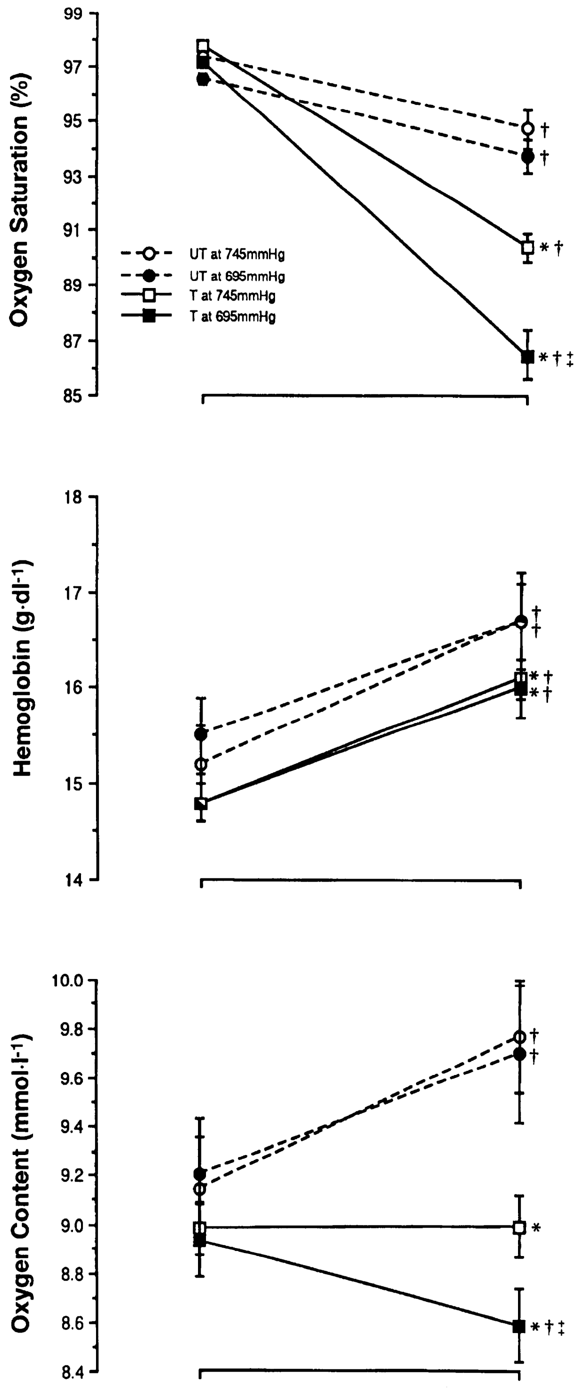

Fig. 2. Changes in arterial O_2 saturation (Sa_{O_2}), hemoglobin concentration, and calculated O_2 content (Co_2) at rest and $\dot{V}o_{2max}$ in both untrained (UT) and trained (T) cyclists in normobaria (745 mmHg; open symbols) and hypobaria (695 mmHg, solid symbols). Values are means ± SE. *Significantly different from UT group at matched intensity and chamber pressure, $P < 0.05$. †Significantly different from rest, $P < 0.05$ (within group comparison). ‡Significantly different from normobaria, $P < 0.05$ (within group comparison). Three-way interactions among exercise intensity, athlete group, and chamber pressure were significant for Sa_{O_2} ($F_{1,18} = 10.63$; $P = 0.004$) and Co_2 ($F_{1,18} = 4.63$; $P = 0.0454$).

relationship was found in the present study. This is probably explained by the coupling of a homogeneous sea-level $\dot{V}o_{2max}$ in the T group with a broad range of altitude-induced $\Delta\dot{V}o_{2max}$ (+1.2 to −12.3%).

Given the important contribution of maximal aerobic power to cycling performance (3, 14), a 6.8% decrease in $\dot{V}o_{2max}$ would be expected to compromise performance. Indeed, with a similar degree of desaturation to that found in the present study, Koskolou and McKenzie (9) reported a significant decline in performance during a 5-min cycle ergometer test. In the T subjects in the

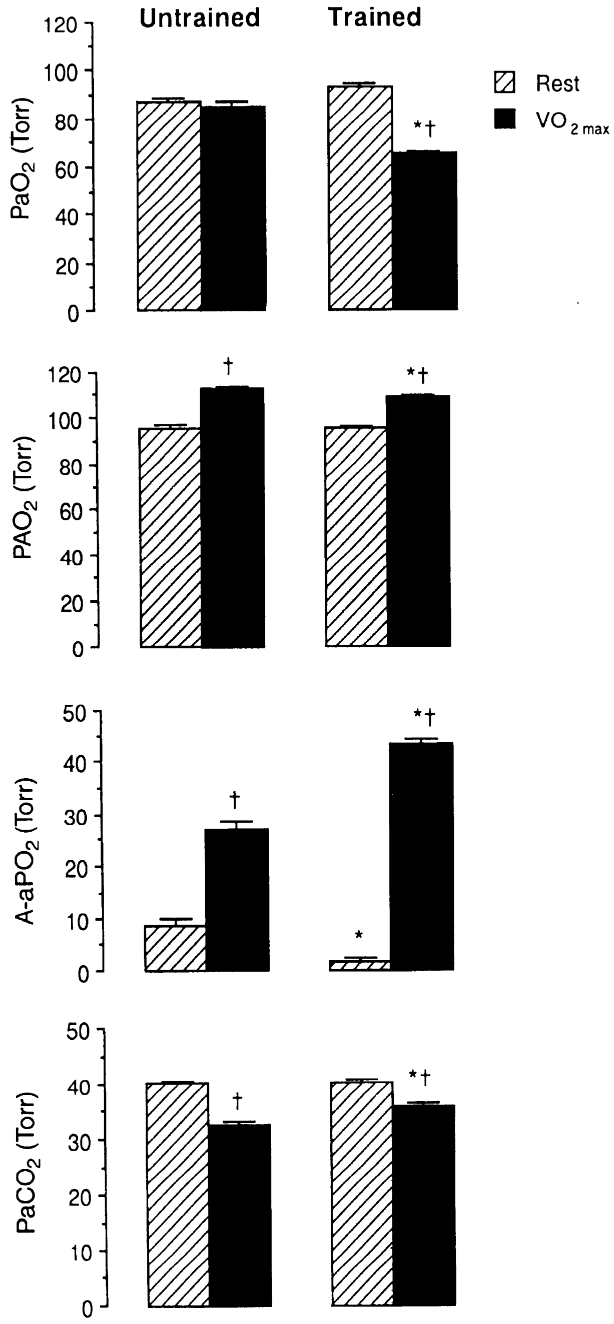

Fig. 3. Mean blood gas data pooled across the 2 chamber pressures during maximal exercise tests in UT and T cyclists. Values are means ± SE. *Significantly different from UT group at matched exercise intensity and chamber pressure, $P < 0.05$. †Significantly different from rest within a group, $P < 0.05$. Interactions between group and exercise intensity were significant for arterial Po_2 (Pa_{O_2}; $F_{1,18} = 58.1$; $P < 0.0001$), alveolar Po_2 (PA_{O_2}; $F_{1,18} = 7.68$; $P = 0.01$), alveolar-arterial Po_2 difference (A-aPo_2; $F_{1,18} = 52.92$; $P < 0.0001$), arterial Pco_2 (Pa_{CO_2}; $F_{1,18} = 6.45$; $P = 0.02$), and Co_2 ($F_{1,18} = 8.96$; $P < 0.005$).

present study, neither total work nor average power during the minute when $\dot{V}O_{2max}$ was attained was affected by MH, although there was a trend toward a lower power output with MH (N, 455 W; MH, 435 W). However, such incremental tests are not an ideal performance test because, with strong subject motivation, $\dot{V}O_2$ can plateau while work continues to increase as a consequence of greater anaerobic work. Warren et al. (26) have argued that C_{O_2} should decrease if exercise-induced hypoxemia is to have a negative impact on exercise performance. The normal response to high-intensity exercise is hemoconcentration, with a consequent increase in C_{O_2}, and this was seen in the UT subjects in the present study. However, despite a similar degree of hemoconcentration in the T subjects, C_{O_2} was unchanged during sea-level exercise and decreased significantly during MH, suggesting that performance might be impaired at mild altitude. At sea level, the Pa_{O_2} at $\dot{V}O_{2max}$ in the T group fell much closer to the steeper portion of the O_2-loading curve for hemoglobin (T, 68.3 ± 1.3 Torr; UT, 89.0 ± 2.9 Torr), such that, despite a similar degree of hemoconcentration in the two subject groups during maximal exercise and an equivalent differential in Pa_{O_2} between N and MH conditions (~7 Torr), a much greater decrease in both Sa_{O_2} and C_{O_2} was seen in the T group under MH conditions. Compared with N conditions, the reduction in C_{O_2} at $\dot{V}O_{2max}$ in the T group when exercising under MH conditions accounted for ~70% of the decrease in O_2 delivery. This value is similar to the 71.2% reported by Lawler et al. (10), who used hypoxia (14% O_2) to reduce C_{O_2} and $\dot{V}O_{2max}$.

An "inadequate ventilatory response" has been suggested as a mechanism for the exercise-induced hypoxemia seen at sea level (4). In the present study, the ventilatory response to maximum exercise was different between the two groups. Compared with the UT group, the $\dot{V}E$ BTPS, $\dot{V}E$ STPD, and Pa_{CO_2} were significantly higher in the T group at maximal exercise, and the T group had significantly lower PA_{O_2} and ventilatory equivalents for both O_2 and CO_2. Collectively, these data indicate that the T group had a lesser hyperventilation. The same conclusion was reached by Caillard et al. (2), but whether the reduced hyperventilation represents a ventilatory limitation or a ventilatory constraint is uncertain. Johnson et al. (8) concluded that $\dot{V}O_{2max}$ was not constrained by a mechanical failure to achieve maximal alveolar ventilation because the two were achieved simultaneously. Furthermore, they reported that the mean ventilatory response during maximal exercise was not increased by either hypoxic or hypercapnic stimuli. However, Norton et al. (13) showed that T subjects exercising at supramaximal intensity could increase $\dot{V}E$ BTPS significantly beyond that attained during maximal exercise and attenuate the desaturation seen at $\dot{V}O_{2max}$. Miyachi and Tabata (12) suggested that 50% of the variability in Sa_{O_2} could be explained by less hyperventilation, and in the present study, ~40% of the variability in Sa_{O_2}, and also Pa_{O_2}, could be explained by the variability in the $\dot{V}E/\dot{V}O_2$. Despite these studies suggesting a significant relationship between the degree of hypoxemia and the ventilatory response, between 50 and 60% of the variance for the relationship between Sa_{O_2} and $\dot{V}E/\dot{V}O_2$ remains unexplained.

Most contemporary studies have shown that a widened A-aP_{O_2} accompanies arterial desaturation (4, 6, 8, 17, 25). Wagner et al. (25), using subjects of modest aerobic power, concluded that the impairment of pulmonary gas exchange was due principally to an alveolar end-capillary diffusion limitation, perhaps based on interstitial edema. More recently, Hopkins et al. (6), using athletes with high aerobic power, concluded that a ventilation-perfusion mismatch could explain >60% of the wide A-aP_{O_2} at $\dot{V}O_{2max}$. Although in our study ~65% of the variance in Sa_{O_2} was associated with a widened A-aP_{O_2}, this was only the case when the data from all subjects were pooled. Even so, in the T group, where $\dot{V}O_{2max}$ was reduced with MH, the A-aP_{O_2} could account for <28% of the variance in Sa_{O_2}. The works of Wagner et al. (25) and Hopkins et al. (6) provide somewhat conflicting evidence with respect to the importance of diffusion limitations to explain the widened A-aP_{O_2}, which may be a reflection of the subject populations but clearly requires further study.

Summary. These results indicate that $\dot{V}O_{2max}$ of T but not UT cyclists was reduced significantly with 50-mmHg hypobaria, equivalent to an altitude of 580 m. This is the lowest altitude reported to decrease $\dot{V}O_{2max}$, illustrating that T athletes are more sensitive to a decrease in PI_{O_2} and that this reduction in aerobic power is compounded by a blunted hyperpneic response. Approximately 70% of this decrease in $\dot{V}O_{2max}$ could be explained by decreased O_2 delivery as a consequence of a reduced Pa_{O_2} and Sa_{O_2} despite an increased hemoglobin concentration. At sea level, the Pa_{O_2} of the T group at $\dot{V}O_{2max}$ was much closer to the steeper portion of the O_2-loading curve, and, as such, an equivalent fall in Pa_{O_2} with hypobaria resulted in a much greater desaturation, with a consequent reduction in C_{O_2}. When the group data were combined, ~65% of the variance in Sa_{O_2} could be attributed to a widened A-aP_{O_2}, which may reflect either a ventilation-perfusion mismatch or an alveolar end-capillary diffusion limitation.

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