

ORIGINAL ARTICLE

Effect of hyperbaric oxygen on oxygen uptake and measurements in the blood and tissues in a normobaric environment

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Br J Sports Med 2003;37:516–520

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Accepted
5 November 2002

Objective: To examine venous partial pressure of oxygen (PvO_2), transcutaneous oxygen tension ($tcPO_2$), and VO_{2MAX} in a normobaric environment after a single hyperbaric oxygen (HBO_2) treatment.

Methods: This was a prospective study of conditions after the intervention compared with baseline. The participants were 10 moderately trained ($VO_{2MAX} = 57.6$ ml/kg/min) men. Two HBO_2 treatments consisting of breathing 95% oxygen at 2.5 atmospheres absolute (ATA) for 90 minutes were administered on non-consecutive days. Baseline testing included measures of VO_{2MAX} , $tcPO_2$, and anthropometry. At 6.0 (1.0) minutes after the first HBO_2 treatment, a VO_{2MAX} test was performed. After the second HBO_2 treatment, leg and chest $tcPO_2$ and PvO_2 were monitored for 60 minutes.

Results: VO_{2MAX} , running time, and peak blood lactate were not altered after the HBO_2 treatment. Leg $tcPO_2$ was lower ($p = 0.003$) and chest $tcPO_2$ was unchanged after the HBO_2 treatment compared with baseline values. PvO_2 was significantly ($p < 0.001$) lower in the first three minutes after treatment than subsequent values, but no other differences were found.

Conclusions: A single HBO_2 treatment at 2.5 ATA for 90 minutes does not raise PvO_2 , $tcPO_2$, or VO_{2MAX} in a normobaric, normoxic environment.

In recent years some professional and college athletic teams have used hyperbaric oxygen (HBO_2) to treat sports injuries, to speed recovery after exercise, and as an ergogenic aid to enhance performance. Because of the importance of oxygen in the aerobic energy system, professional athletes sometimes receive HBO_2 before participation in their sport in the belief that subsequent performance will be improved.¹ HBO_2 therapy is defined as a medical treatment in which the patient breathes 100% oxygen intermittently while inside a chamber at a pressure greater than 1 atmosphere absolute (ATA).² The Undersea and Hyperbaric Medical Society currently approves 13 medical indications for treatment with HBO_2 .²

Physiologists have long debated whether oxygen delivery or use by the skeletal muscles is the limiting factor for VO_{2MAX} . Potential physiological factors limiting VO_{2MAX} include: (a) pulmonary diffusion capacity for O_2 ; (b) maximal cardiac output; (c) the peripheral circulation; (d) the metabolic capacity of skeletal muscle.³ According to Rowell,³ most physiologists believe that the capacity of the central cardiovascular system to transport oxygen to the tissues is the principal determinant of VO_{2MAX} . This concept has been used to justify HBO_2 treatments to enhance the availability of oxygen in an attempt to increase maximal aerobic performance. Also, oxygen stored in the tissues after an HBO_2 treatment may be available to working muscles.

Our reasons for this study are the contrary findings in the research literature^{4–7} and the observation that the athletic community has used HBO_2 before sport performance. Under pressure, oxygen dissolves in all body fluids.⁸ For HBO_2 to be beneficial, increased oxygen must be stored during the treatment and remain raised on return to a normobaric, normoxic environment. Previous studies have not examined the blood or tissues to determine if these compartments would be additional sources of oxygen available to the mitochondria during maximal exercise. Because of potential misuse by the athletic community, there is a need to establish

if there are benefits in using HBO_2 as an ergogenic aid. This study examines the ergogenic potential of HBO_2 . We hypothesised that: (a) venous partial pressure of oxygen (PvO_2) and/or transcutaneous oxygen tension ($tcPO_2$) would be unchanged after a single HBO_2 treatment in a normobaric environment; (b) VO_{2MAX} would be similar after a single HBO_2 treatment in a normobaric environment.

METHODS

Subjects

The subjects were 10 trained ($VO_{2MAX} = 57.6$ (6.2) ml/kg/min) male volunteers (table 1). They were examined by a doctor and were excluded if contraindications to HBO_2 treatment were evident (recent thoracic surgery, repeated ear infections, asthma, cataracts, diabetes, receiving anti-convulsant medication, hereditary spherocytosis, and recent upper respiratory tract infections). All experimental procedures were evaluated and approved by the McGill University Faculty of Medicine institutional review board. Subjects gave written consent to participate after the design and risks of the study had been described to them.

Experimental design

Subjects underwent tests on three non-consecutive days within a two week period. Baseline testing on day 1 included assessment of physical characteristics, $tcPO_2$ during normoxic and hyperoxic breathing, and measurement of VO_{2MAX} . Testing on day 2 included a 90 minute HBO_2 treatment followed by a VO_{2MAX} test. The time delay from exiting the hyperbaric chamber to the start of the VO_{2MAX} test was 6.0 (1.0) minutes. On day 3, subjects received a 90 minute HBO_2

Abbreviations: HBO_2 , hyperbaric oxygen; PvO_2 , venous partial pressure of oxygen; $tcPO_2$, transcutaneous oxygen tension; Vo_{2max} , maximum oxygen consumption

Table 1 Physical characteristics of the subjects (n = 10)

Variable	Mean (SD)	Range
Age (years)	25.7 (5.5)	20–38
Height (cm)	179.7 (7.5)	165.0–194.9
Weight (kg)	76.4 (4.1)	70.9–82.3
Percentage fat	10.2 (2.0)	5.5–17.4
VO ₂ MAX (ml/kg/min)	57.6 (6.2)	47.5–67.1

treatment followed by nine Pvo₂ samples and tcPo₂ measurements for 60 minutes.

Hyperbaric oxygen protocol

Figures 1 and 2 illustrate the HBO₂ chamber and protocol. The HBO₂ treatment was administered in a Sigma Plus monoplace hyperbaric chamber (Perry Baromedical Corporation, Riviera, Florida, USA) under the supervision of a certified chamber operator at the Clegghorn Hyperbaric Laboratory, McGill University. It took about 10 minutes to pressurise the chamber to 2.5 ATA with 95% oxygen. At 25 and 55 minutes into the 90 minute treatment, subjects were given a five minute air break through an oronasal mask to reduce the risk of oxygen toxicity. After 90 minutes, the chamber was decompressed from 2.5 to 1.0 ATA in about eight minutes.

Under normal sea level conditions, barometric pressure is 1 ATA or 760 mm Hg, and oxygen content in the air is 20.9%. In these conditions the PaO₂ is 100 mm Hg. During our HBO₂ treatment, the combination of increased pressure (2.5 ATA) and increased oxygen concentration (95%) results in additional oxygen dissolved in plasma. During the hyperbaric treatment at these conditions the PaO₂ is predicted to be:

$$PaO_2 = ((P_{BTPS} \times F_{IO_2}) - (P_{ACO_2}/R))$$

$$PaO_2 = (((2.5 \text{ ATA} \times 760) - 47 \text{ mm Hg}) \times 0.95) - (40 \text{ mm Hg}/0.82)$$

$$PaO_2 = 1853 - 49 = 1804 \text{ mm Hg}$$

where P_{BTPS} = pressure at body temperature pressure saturated (mm Hg), F_{IO₂} = fraction of oxygen in inspired air (%), P_{ACO₂} = partial pressure of CO₂ in arterial blood (mm Hg), and R = respiratory quotient.

Exercise test procedure

Before the exercise test, physical characteristics (height, weight, and body composition) were measured. Percentage body fat was estimated from skinfold measurements and the regression equation of Jackson and Pollock.⁹



Figure 1 Hyperbaric oxygen monoplace chamber.

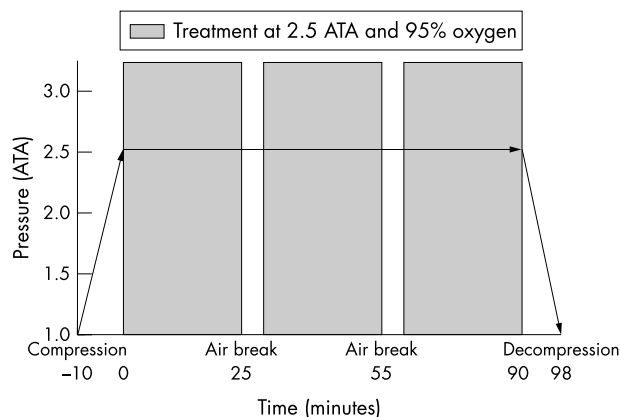


Figure 2 Hyperbaric oxygen protocol. ATA, Atmospheres absolute.

VO₂MAX was measured on a Quinton Q65 Series 90 treadmill (Quinton Instruments, Seattle, Washington, USA). Subjects began an incremental test at 5 mph (13.4 m/min) and 5% grade with speed increased by 0.5 mph (13.4 m/min) every minute until volitional exhaustion. Expired gases were collected with V_E, VO₂, and VCO₂ averaged every 20 seconds using a SensorMedics 2900 Metabolic Measurement Cart (SensorMedics, Yorba Linda, California, USA). Subjects were verbally encouraged to continue exercising until volitional exhaustion. Criteria for reaching VO₂MAX were attainment of age predicted heart rate maximum, a respiratory exchange ratio of 1.10, or a plateau in VO₂ with increased workload. VO₂MAX was calculated by averaging the highest values over one minute. Heart rate was measured using a Polar Accurex Heart Rate Monitor (Polar Electro, Kempele, Finland) and averaged every five seconds. Four minutes after the VO₂MAX test, a finger prick blood sample was taken to determine peak blood lactate concentration. The blood samples were analysed with an Accusport Portable Lactate Analyzer (Behringer Mannheim, Mannheim, Germany).

tcPo₂ measurement

tcPo₂ was measured at two sites: chest (second intracostal) and leg (mid-thigh over the rectus femoris). The sites were prepared by removal of hair, cleaning with alcohol, and denuding the skin by repeated application and removal of adhesive tape.¹⁰ A calibrated TCM 30 Transcutaneous PO₂ Monitoring System (Radiometer, Copenhagen, Denmark) was used to measure tcPo₂ continuously. The electrodes were warmed to 45°C as recommended for use in hyperbaric operations.^{11,12} There was a lag of about 10 minutes after application of the electrodes before stable values were achieved. Values were recorded every minute for 60 minutes. The baseline tcPo₂ assessment included a 20 minute oxygen challenge in which the subjects breathed 100% oxygen through an oronasal mask from minute 20 to minute 40 to demonstrate their tcPo₂ responsiveness to high concentrations of oxygen.

Pvo₂ measurement

When each subject left the hyperbaric chamber on day 3, a 14 gauge intravenous catheter was inserted into an antecubital vein. The line was kept patent between samples with 5% dextrose solution (IVD5W). Blood samples (3–5 ml) were drawn 3, 5, 10, 15, 20, 30, 40, 50, and 60 minutes after the subject had left the chamber. To ensure blood samples were not contaminated with IVD5W solution, 5 ml blood was drawn and discarded before every blood sample was collected. Samples were immediately aspirated into a

Radiometer ABL5 blood analyser, which was calibrated with known samples provided by the manufacturer. Every 30 minutes, the blood analyser performed a barometric pressure and a 1 point calibration of the PO₂ electrode using gas of 19.8% O₂. Every two hours the blood analyser performed a 2 point calibration of the PO₂ electrode using gases of 0% and 19.8% O₂.

With regard to blood sampling, our preference was to obtain arterial PO₂ (PaO₂) measurements because it is unclear how long PaO₂ remains raised after an HBO₂ treatment. The ethics institutional review board did not approve arterial sampling for this study, and requested that an intravenous catheter be used to obtain blood samples.

Statistical analysis

Paired *t* tests were used to compare baseline conditions with those after treatment for VO₂MAX and peak blood lactate data. A one way repeated measures analysis of variance was used to compare PvO₂ data for the two conditions. A two way repeated measures analysis of variance was used to compare tcPO₂ data at two sites (chest and leg) and two conditions (baseline and after HBO₂). Analysis of variance was followed by post hoc comparisons using Tukey's HSD (honestly significant difference) test. For all statistical analyses, was set at *p*<0.05.

RESULTS

Table 2 shows the exercise test results. No significant differences were found for VO₂MAX or peak blood lactate concentration between the baseline condition and after HBO₂ treatment. The mean (SD) VO₂MAX values were 57.6 (6.2) and 57.3 (5.8) ml/kg/min in the two conditions. The time from exiting the chamber and initiation of the exercise test was 6.0 (1.0) minute. The HBO₂ treatment did not enhance exercise performance, as run times were identical in both conditions (10.1 (1.9) min). Peak lactate concentrations were similar (8.9 (2.8) and 10.0 (1.9) mmol/l) in the two conditions.

Table 3 summarises and fig 3 illustrates the PvO₂ results. There was a significant change in PvO₂ over time (*F* = 6.61; *df* 8.40; *p*<0.001) after the HBO₂ treatment, with a lower PO₂ value at three minutes than at 5–60 minutes. The tourniquet on the upper arm was in place for about one minute before drawing of the initial blood sample. We attribute the significantly lower PO₂ at three minutes to altered blood flow in the arm. The PvO₂ data suggest that there was no excess oxygen circulating in the blood after the HBO₂ treatment.

Figure 4 summarises the tcPO₂ data. In the baseline condition, the start of the oxygen challenge was at 20 minutes. The chest tcPO₂ increased from about 80 to 290 mm Hg in about five minutes, and the leg tcPO₂ increased from 70 to 230 mm Hg in the same time frame. Upon completion of the oxygen challenge at 40 minutes, both the chest and leg tcPO₂ returned to baseline values within three minutes. After the HBO₂ treatment, the leg tcPO₂ was

Table 2 Physiological responses during exercise tests

Variable	Baseline	After HBO ₂
VO ₂ MAX (ml/kg/min)	57.6 (6.2)	57.3 (5.8)
VO ₂ MAX (litre/min)	4.38 (0.5)	4.38 (0.5)
Run time (min)	10.1 (1.9)	10.1 (1.9)
HRMAX (beats/min)	191 (10.3)	189 (11.3)
LaMAX (mmol/l)	8.9 (2.8)	10.0 (1.9)

Values are mean (SD).

VO₂MAX, Maximum oxygen consumption; HRMAX, maximum heart rate; LaMAX, maximum blood lactate concentration.

Table 3 Venous PO₂ after hyperbaric treatment

Time (min)	PO ₂ (mm Hg)
3	18.0 (4.3)*
5	31.3 (7.9)
10	37.1 (10.6)
15	36.3 (6.8)
20	37.3 (9.1)
30	37.0 (10.2)
40	36.8 (9.6)
50	38.7 (15.6)
60	31.7 (9.4)

Values are mean (SD).

*Significantly different from other readings (*p*<0.001).

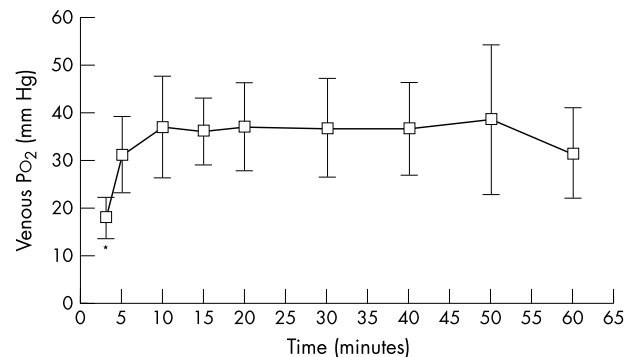


Figure 3 Venous PO₂ plotted against time after hyperbaric treatment. **p*<0.001 compared with all other values.

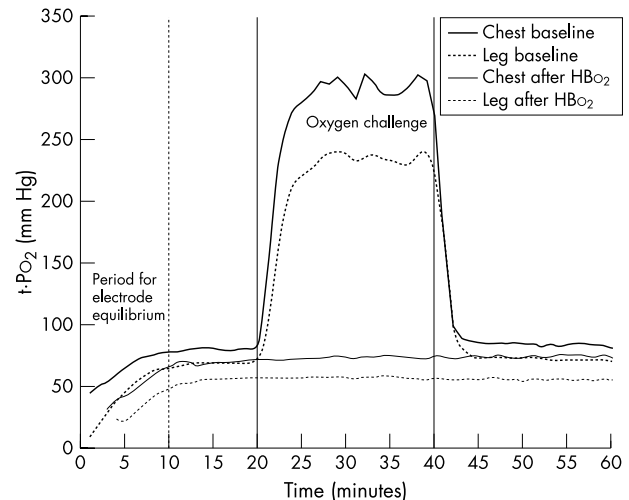


Figure 4 Chest and leg transcutaneous oxygen tension (tcPO₂) during the baseline and hyperbaric treatment (HBO₂) conditions versus time.

significantly (*F* = 11.93; *df* 1.18; *p* = 0.003) lower than the baseline values, with a difference of 14 mm Hg. In contrast, the chest tcPO₂ values were similar at baseline and after HBO₂ treatment.

DISCUSSION

Intermittent HBO₂ treatments have been used to speed recovery of muscle strength after exercise induced injury. Quadriceps muscle soreness was induced by eccentric exercise.¹³ HBO₂ treatments improved recovery of eccentric strength compared with placebo treatments. The effect of a

single HBO₂ treatment on subsequent exercise performance has also been examined. Kaijser¹⁴ compared dynamic forearm exercise under hyperbaric (3.0 ATA) and normobaric conditions. The performance time to exhaustion was increased in three subjects and unchanged in three subjects.

There is evidence that breathing hyperoxic gas during exercise enhances performance.^{15–21} Using arterial and femoral venous sampling combined with measurement of blood flow, it has been shown that hyperoxia increases VO₂MAX of an exercising leg.¹⁸ As it is unclear if HBO₂ treatment before exercise alters performance, we examined PVO₂, tcPO₂, and VO₂MAX in a normobaric environment after a single HBO₂ treatment.

Four studies have investigated maximal aerobic performance in a normobaric environment after HBO₂ treatments, with two studies showing positive findings^{5, 6} and two studies reporting no benefits.^{4, 7}

Cabric *et al*⁶ administered 100% oxygen at 2.8 ATA for 60 minutes. Eighteen female students were randomly divided into three groups (six per group). After the HBO₂ treatment, the first group performed a VO₂MAX test at 30 minutes, the second at three hours, and the third at six hours. Both VO₂MAX and treadmill run time to exhaustion had increased significantly 30 minutes and three hours after treatment. After the HBO₂ treatment, VO₂MAX had increased by 15% at 30 minutes ($p < 0.05$), 10% at three hours ($p < 0.05$), and 7% at six hours (non-significant). The improved performance was attributed to oxygen stored within skeletal muscle tissue. It has also been reported that blood lactate levels, VO₂, and VCO₂ were lower during submaximal exercise in a normobaric environment after HBO₂.¹⁴ This study included only two subjects and therefore it is difficult to generalise their findings.

Webster *et al*⁷ questioned the ergogenic effect of HBO₂. Their subjects performed three exercise tests on a cycle ergometer. These tests were performed on separate days with the first two exercise tests designed to establish baseline data, and the third test after an HBO₂ treatment at 2.0 ATA for 60 minutes. The mean time from exiting the chamber to cycling was 22.5 minutes. No significant differences were found for VO₂MAX, ventilatory threshold, lactate threshold, VEMAX, or HRMAX for the three tests. Near infrared spectroscopy was used to examine tissue oxygenation of the vastus lateralis muscle at rest, throughout exercise, and during recovery. After the HBO₂ treatment, muscle tissue oxygenation during rest and recovery were similar to control values.

McGavock *et al*⁴ examined the acute effects of a single HBO₂ treatment on aerobic performance in a normobaric environment. Subjects ($n = 12$) performed four exercise-HBO₂ conditions designated as: (a) control; (b) exercise-non-HBO₂; (c) no exercise-HBO₂; (d) exercise-HBO₂. Exercise was a 90 minute run to produce fatigue. The HBO₂ treatments were at 2.5 ATA for 90 minutes. At the end of each condition, aerobic performance was assessed using running economy tests and a VO₂MAX test. The time between exiting the chamber and running on the treadmill averaged 40 minutes. Recovery was not enhanced after a single HBO₂ treatment nor did it alter submaximal or maximal running performance.

Our findings support the results of Webster *et al*⁷ and McGavock *et al*.⁴ Baseline conditions and those after HBO₂ were similar for VO₂MAX, treadmill running time, and peak blood lactate, indicating that the single HBO₂ treatment was not ergogenic.

tcPO₂ is a reliable assessment of oxygen available to tissues.¹⁰ It is traditionally used to predict if HBO₂ treatment will be beneficial for wound healing and to maintain tissue oxygen values within an appropriate range.²² Chest tcPO₂ values have been recorded at 1312 (112) mm Hg during a HBO₂ treatment at 2.4 ATA.²³ In our study, tcPO₂ was used to

Take home message

A single HBO₂ treatment at 2.5 ATA for 90 minutes does not raise VO₂max in a normobaric, normoxic environment. Transcutaneous tissue and blood PO₂ measurements after the HBO₂ treatment support the statement that HBO₂ does not have ergogenic benefits for the athletic community.

assess oxygen levels in muscle tissue after the HBO₂ treatment. It appears that the excess oxygen that is physically dissolved in plasma during HBO₂ is rapidly consumed upon exiting the HBO₂ chamber. Upon application of the tcPO₂ electrode, it takes about 10 minutes to obtain a reliable value as the electrode warms the skin.¹⁰ In our study, 10 minutes after exiting the chamber tcPO₂ values had returned to baseline and leg tcPO₂ values were lower than baseline. The lower tcPO₂ values in the leg may be attributed to vasoconstriction. It has been shown both in vivo and in vitro that blood flow is decreased when inspired PO₂ increases above 500 mm Hg.²⁴ The vasoconstrictive effect occurs in both arterial and venous vascular beds.²⁵

Sheffield¹⁰ presents normal values for blood and tissue O₂ measured by blood gas analyser, mass spectrometer, tissue tonometer, implanted polarographic electrode, and tcPO₂ at pressures of 1–3 ATA. Normal mean values for PVO₂ range from 36 to 40 mm Hg.^{26, 27} Between 10 and 60 minutes after HBO₂, our PVO₂ data ranged from 31.7 to 38.7 mm Hg indicating that there was no excess oxygen circulating in the blood. Banister *et al*⁵ examined PaO₂ and PaCO₂ after an HBO₂ treatment in two subjects. The PaO₂ and PaCO₂ remained unchanged. The time from the end of treatment to drawing blood samples was not stated. Our PVO₂ and tcPO₂ data indicate that plasma and tissue oxygen levels are not raised after HBO₂. After our HBO₂ treatment, PVO₂ was relatively constant from 5 to 60 minutes. The only significant finding occurred at three minutes after treatment with a lower PVO₂ value. We attribute the significantly lower PO₂ at three minutes to altered blood flow in the arm, as a tourniquet was placed on the upper arm for about one minute before drawing of the initial blood sample. The PVO₂ data suggest that there was no excess oxygen circulating in the blood after the HBO₂ treatment. Tissue autoregulation reduces O₂ levels upon return to a normobaric, normoxic environment.²⁸

In summary, the results of this study show that a single HBO₂ treatment at 2.5 ATA for 90 minutes does not raise VO₂MAX in a normobaric, normoxic environment. Oxygen measurements in the venous blood (PVO₂) and in the tissues (tcPO₂) provide new data to support the rationale that HBO₂ treatments do not enhance performance. This message needs to be conveyed by doctors and sport scientists to the athletic community. Our findings support the work of Webster *et al*,⁷ McGavock *et al*,⁴ and the Undersea and Hyperbaric Medical Society statement that HBO₂ does not have ergogenic properties.

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