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

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Objective: To examine venous partial pressure of oxygen (PvO₂), transcutaneous oxygen tension (tcPO₂), and VO₂MAX in a normobaric environment after a single hyperbaric oxygen (HBO₂) treatment.

Methods: This was a prospective study of conditions after the intervention compared with baseline. The participants were 10 moderately trained (VO₂MAX = 57.6 ml/kg/min) men. Two HBO₂ 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 VO₂MAX, tcPO₂, and anthropometry. At 6.0 (1.0) minutes after the first HBO₂ treatment, a VO₂MAX test was performed. After the second HBO₂ treatment, leg and chest tcPO₂ and PvO₂ were monitored for 60 minutes.

Results: VO₂MAX, running time, and peak blood lactate were not altered after the HBO₂ treatment. Leg tcPO₂ was lower (p = 0.003) and chest tcPO₂ was unchanged after the HBO₂ treatment compared with baseline values. PvO₂ 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 HBO₂ treatment at 2.5 ATA for 90 minutes does not raise PvO₂, tcPO₂, or VO₂MAX in a normobaric, normoxic environment.
treatment followed by nine \( \text{PvO}_2 \) samples and \( \text{tcPO}_2 \) 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 Cleghorn 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 \( \text{PaO}_2 \) 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 \( \text{PaO}_2 \) is predicted to be:

\[
\text{PaO}_2 = \left( \text{PBTPS} \times \text{FiO}_2 \right) - \left( \text{PaCO}_2 / R \right) \\
\text{PaO}_2 = \left( \left(2.5 \text{ ATA} \times 760 \right) - 47 \text{ mm Hg} \right) \times 0.95 - \left(40 \text{ mm Hg} / 0.82 \right) \\
\text{PaO}_2 = 1853 - 49 = 1804 \text{ mm Hg}
\]

where PBTPS = pressure at body temperature pressure saturated (mm Hg), \( \text{FiO}_2 \) = fraction of oxygen in inspired air (%), \( \text{PaCO}_2 \) = partial pressure of \( \text{CO}_2 \) 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.\(^9\)

**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 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. Kaijser44 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.19–21 Using arterial and femoral venous sampling combined with measurement of blood flow, it has been shown that hyperoxia increases VO2MAX of an exercising leg.22 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 findings23–4 and two studies reporting no benefits.25–7

Cabric et alb 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 al4 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 al6 administered 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 al4 and McGavock et al.6 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.28 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.29 Chest tcPO2 values have been recorded at 1312 (112) mm Hg during a HBO2 treatment at 2.4 ATA.22 In our study, tcPO2 was used to 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.22 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

Sheffield26 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 PO2 range from 36 to 40 mm Hg.27 Between 10 and 60 minutes after HBO2, our PO2 data ranged from 31.7 to 38.7 mm Hg indicating that there was no excess oxygen circulating in the blood. Banister et al3 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 PO2 and tcPO2 data indicate that plasma and tissue oxygen levels are not raised after HBO2. After our HBO2 treatment, PO2 was relatively constant from 5 to 60 minutes. The only significant finding occurred at three minutes after treatment with a lower PO2 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 PO2 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 al7, McGavock et al,4 and the Undersea and Hyperbaric Medical Society statement that HBO2 does not have ergogenic properties.

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