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

A N H Hodges, S Delaney, J M Lecomte, V J Lacroix, D L Montgomery

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. 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.

METHODS

Subjects
The subjects were 10 trained (VO2MAX = 57.6 (6.2) ml/kg/min) male volunteers (table 1). They were examined by a doctor and were excluded if contraindications to HBO2 treatment were evident (recent thoracic surgery, repeated ear infections, asthma, cataracts, diabetes, receiving anticonvulsant 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, tcPO2 during normoxic and hyperoxic breathing, and measurement of VO2MAX. Testing on day 2 included a 90 minute HBO2 treatment followed by a VO2MAX test. The time delay from exiting the hyperbaric chamber to the start of the VO2MAX test was 6.0 (1.0) minutes. On day 3, subjects received a 90 minute HBO2 treatment.

Abbreviations: HBO2, hyperbaric oxygen; Pvo2, venous partial pressure of oxygen; tcPO2, transcutaneous oxygen tension; Vo2max, maximum oxygen consumption.
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 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 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:

\[
\text{PaO2} = \left( \left( \text{PBTPS} \times \text{FiO2} \right) - \left( \text{PaCO2} / R \right) \right)
\]

\[
\text{PaO2} = \left( \left( 2.5 \text{ ATA} \times 760 \right) - \left( 47 \text{ mm Hg} / 0.95 \right) \right) - (40 \text{ mm Hg} / 0.82)
\]

\[
\text{PaO2} = 1853 - 49 = 1804 \text{ mm Hg}
\]

where PBTPS = pressure at body temperature pressure saturated (mm Hg), FiO2 = fraction of oxygen in inspired air (%), PaCO2 = 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

VO2MAX was measured on a Quinton Q65 Series 90 treadmill (Quinton Instruments, Seattle, Washington, USA). Subjects began an incremental test at 5 mph (134 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 Ve, 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 P02 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 (IVDSW). 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 IVDSW solution, 5 ml blood was drawn and discarded before every blood sample was collected. Samples were immediately aspirated into a

<table>
<thead>
<tr>
<th>Variable</th>
<th>Mean (SD)</th>
<th>Range</th>
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<tbody>
<tr>
<td>Age (years)</td>
<td>25.7 (5.5)</td>
<td>20–38</td>
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<tr>
<td>Height (cm)</td>
<td>179.7 (7.5)</td>
<td>165.0–194.9</td>
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<tr>
<td>Weight (kg)</td>
<td>76.4 (4.1)</td>
<td>70.9–82.3</td>
</tr>
<tr>
<td>Percentage fat</td>
<td>10.2 (2.0)</td>
<td>5.5–17.4</td>
</tr>
<tr>
<td>VO2MAX (ml/kg/min)</td>
<td>57.6 (6.2)</td>
<td>47.5–67.1</td>
</tr>
</tbody>
</table>

Table 1 Physical characteristics of the subjects (n = 10)
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 tcP02 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 (\(F = 6.61; \text{df 8.40; } p<0.001\)) 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 tcP02 data. In the baseline condition, the start of the oxygen challenge was at 20 minutes. The chest tcP02 increased from about 80 to 290 mm Hg in about five minutes, and the leg tcP02 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 tcP02 returned to baseline values within three minutes. After the HBO2 treatment, the leg tcP02 was significantly (\(F = 11.93; \text{df 1.18; } p = 0.003\)) lower than the baseline values, with a difference of 14 mm Hg. In contrast, the chest tcP02 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
Effect of hyperbaric oxygen

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.

**Take home message**

**REFERENCES**

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