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. 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.

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 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, 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, leg and chest tcPO₂ and PvO₂ were monitored for 60 minutes.

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:

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

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

**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

**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 45C 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.

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

<table>
<thead>
<tr>
<th>Variable</th>
<th>Mean (SD)</th>
<th>Range</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (years)</td>
<td>25.7 (5.5)</td>
<td>20–38</td>
</tr>
<tr>
<td>Height (cm)</td>
<td>179.7 (7.5)</td>
<td>165.0–194.9</td>
</tr>
<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 (4.2)</td>
<td>47.5–67.1</td>
</tr>
</tbody>
</table>

**Figure 1** Hyperbaric oxygen monoplace chamber.

**Figure 2** Hyperbaric oxygen protocol. ATA, Atmospheres absolute.
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 Pa O2 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
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.
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