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

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

Br. J. Sports Med. 2003;37;516-520
doi:10.1136/bjsm.37.6.516

Updated information and services can be found at:
http://bjsm.bmj.com/cgi/content/full/37/6/516

These include:

References
This article cites 23 articles, 6 of which can be accessed free at:
http://bjsm.bmj.com/cgi/content/full/37/6/516#BIBL

1 online articles that cite this article can be accessed at:
http://bjsm.bmj.com/cgi/content/full/37/6/516#otherarticles

Rapid responses
You can respond to this article at:
http://bjsm.bmj.com/cgi/eletter-submit/37/6/516

Email alerting service
Receive free email alerts when new articles cite this article - sign up in the box at the top right corner of the article

Notes

To order reprints of this article go to:
http://journals.bmj.com/cgi/reprintform

To subscribe to British Journal of Sports Medicine go to:
http://journals.bmj.com/subscriptions/
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 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 and were retested on the day and day after HBO2.

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:

\[
\text{PaO}_2 = \left( \beta \text{PBP}_{\text{TPS}} \times \text{FIO}_2 \right) - \left( \text{PaCO}_2 / R \right)
\]

where \( \beta \text{PBP}\) = pressure at body temperature pressure saturated (mm Hg), \( \text{FIO}_2 \) = fraction of oxygen in inspired air (%), \( \text{PaCO}_2 \) = 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.

**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 PV02 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 PV02 results. There was a significant change in PV02 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 PV02 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. Quadriiceps muscle soreness was induced by eccentric exercise.13 HBO2 treatments improved recovery of eccentric strength compared with placebo treatments. The effect of a
single HB0₂ treatment on subsequent exercise performance has also been examined. Kaijser⁴ 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.⁵⁻¹¹ Using arterial and femoral venous sampling combined with measurement of blood flow, it has been shown that hyperoxia increases VO₂MAX of an exercising leg.⁶ As it is unclear if HB0₂ treatment before exercise alters performance, we examined VO₂, tcPO₂, and VO₂MAX in a normobaric environment after a single HB0₂ treatment.

Four studies have investigated maximal aerobic performance in a normobaric environment after HB0₂ treatments, with two studies showing positive findings⁵ ⁶ and two studies reporting no benefits.⁷ ⁸

Cabric et al.⁹ administered 100% oxygen at 2.8 ATA for 60 minutes. Eighteen female students were randomly divided into three groups (six per group). After the HB0₂ 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 HB0₂ 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 HB0₂.¹⁰ This study included only two subjects and therefore it is difficult to generalise their findings.

Webster et al.¹¹ questioned the ergogenic effect of HB0₂. 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 HB0₂ 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, V̇EMAX, 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 HB0₂ treatment, muscle tissue oxygenation during rest and recovery were similar to control values.

McGavock et al.¹² examined the acute effects of a single HB0₂ treatment on aerobic performance in a normobaric environment. Subjects (n = 12) performed four exercise-HB0₂ conditions designated as: (a) control; (b) exercise-non-HB0₂; (c) no exercise-HB0₂; (d) exercise-HB0₂. Exercise was a 90 minute run to produce fatigue. The HB0₂ 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 HB0₂ treatment nor did it alter submaximal or maximal running performance.

Our findings support the results of Webster et al.¹¹ and McGavock et al.¹². Baseline conditions and those after HB0₂ were similar for VO₂MAX, treadmill running time, and peak blood lactate, indicating that the single HB0₂ treatment was not ergogenic.

tcPO₂ is a reliable assessment of oxygen available to tissues.¹³ It is traditionally used to predict if HB0₂ treatment will be beneficial for wound healing and to maintain tissue oxygen values within an appropriate range.ⁱ⁴ Chest tcPO₂ values have been recorded at 1312 (112) mm Hg during a HB0₂ treatment at 2.4 ATA.²² In our study, tcPO₂ was used to assess oxygen levels in muscle tissue after the HB0₂ treatment. It appears that the excess oxygen that is physically dissolved in plasma during HB0₂ is rapidly consumed upon exiting the HB0₂ chamber. Upon application of the tcPO₂ electrode, it takes about 10 minutes to obtain a reliable value as the electrode warms the skin.¹⁵ 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.⁶ The vasoconstrictive effect occurs in both arterial and venous vascular beds.²⁵

Sheffield²⁶ presents normal values for blood and tissue PO₂ 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.²⁷ Between 10 and 60 minutes after HB0₂, 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.²⁸ examined PaO₂ and PacO₂ after an HB0₂ 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 HB0₂. After our HB0₂ 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 HB0₂ treatment. Tissue autoregulation reduces O₂ levels upon return to a normobaric, normoxic environment.²⁹

In summary, the results of this study show that a single HB0₂ 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 HB0₂ 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.,³ McGavock et al.,³ and the Undersea and Hyperbaric Medical Society statement that HB0₂ does not have ergogenic properties.

Take home message
A single HB0₂ 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 HB0₂ treatment support the statement that HB0₂ does not have ergogenic benefits for the athletic community.

Authors’ affiliations
A N H Hodges, J S Delaney, J M Lecomte, V J Lacroix, D L Montgomery, McGill University, Montreal, Canada

REFERENCES