Effect of hyperbaric oxygen therapy on exercise-induced muscle soreness

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The purpose of this study was to examine the effects of HBO₂ therapy on exercise-induced muscle soreness. Subjects (n = 6 male and 10 female university student volunteers) were randomly divided into an experimental group that received HBO₂ therapy and a control group that did not receive any treatments. HBO₂ treatments consisted of 5 sessions of breathing 95% oxygen at 2.5 atm abs for 100 min. Temporary muscle soreness was created using a single-leg eccentric exercise task involving the quadriceps femoris. Over the next 14 days, measurements were obtained on muscle soreness, leg circumference, quadriceps peak torque, quadriceps average power, fatigue and plasma creatine kinase. After eccentric exercise, plasma creatine kinase (CK) levels and perceived muscle soreness were elevated but were not different between HBO₂ and control groups. HBO₂ therapy did not alter leg circumference, quadriceps peak torque, average power or fatigue compared to the control group. Faster recovery was observed in the HBO₂ group on day 3 following the exercise protocol with perceived muscle soreness still elevated for the control group but not different from baseline for the HBO₂ group. The data indicated that five HBO₂ treatments did not speed recovery following eccentric exercise that induced temporary muscle soreness.

delayed onset muscle soreness, muscle damage, eccentric exercise, creatine kinase, hyperbaric oxygen

INTRODUCTION

Many phenomena can lead to muscle degeneration. Degeneration can be brought about by muscular pathology, direct trauma such as a cut or a contusion, an extended ischemia, and strenuous unaccustomed exercise, especially exercise involving eccentric muscle contractions (1-13). Exercise-induced muscle damage (EIMD) can be observed directly at the cellular level or indirectly by the study of alteration in muscular function and other manifestations such as inflammatory response, swelling, muscle soreness, change in enzyme levels and reduction in muscle strength and range of motion.

Regardless of etiology, tissue edema and hypoperfusion are frequently underlying problems associated with exercise-induced muscle damage. Some local hypoxia is a normal and inevitable result of tissue injury (14). It is believed to be, in part, a stimulus for the repair process. Local ischemia is also a factor in the vulnerability of the tissue and a lack of oxygen can lead to inadequate healing and infection.

Hyperbaric oxygen therapy (HBO₂) is beneficial in wound healing and in a number of injuries where edema and ischemia are present, such as compartment syndrome and crush injury (14-20). The benefits from HBO₂ therapy are associated with hyperoxygenation of the tissues,
vasoconstriction which reduces edema, enhancement of white cell function which limits infection, and neovascularization. The decrease in regional blood flow with vasoconstriction more than compensates for an increase in oxygen delivery to damaged tissues. The net effect is decreased tissue inflammation without hypoxia – the mechanism by which HBO\textsubscript{2} therapy is believed to improve crush injuries, thermal burns and compartment syndrome (15-16).

In most cases, muscle soreness (also call delayed-onset muscle soreness or DOMS), caused by exercise is often tolerated rather than treated because symptoms typically dissipate after 7 to 10 days (21). Wage earners as well as athletes have a vested interest in enhancing recovery with full mobility before returning to work or play. The adjunctive use of HBO\textsubscript{2} therapy has the potential to restore a favorable milieu and speed up the recovery process but the limited research with HBO\textsubscript{2} has been inconclusive (22-25).

The purpose of this study was to determine the effects of HBO\textsubscript{2} therapy on exercise-induced muscle soreness. Following eccentric exercise, changes in plasma creatine kinase, perceived muscle soreness, leg circumference, isokinetic quadriceps peak torque, quadriceps average power and fatigue were examined.

METHODS

Subjects: The participants were 16 college-aged male (n = 6) and female (n = 10) volunteers who completed an informed consent document and passed a medical examination. The male subjects were (mean ± standard deviation): age 25 ± 6 years, height 179.3 ± 7.2 cm, weight 78.5 ± 12.9 kg, body mass index 24.3 ± 2.9 kg/m\textsuperscript{2}. The female subjects were: age 23 ± 4 years, height 166.5 ± 7.5 cm, weight 67.0 ± 9.0 kg, body mass index 24.1 ± 2.3 kg/m\textsuperscript{2}. Subjects were free of musculoskeletal injuries, were aware that unaccustomed eccentric exercise can create muscle soreness, and were not involved in weight training using the quadriceps femoris for three months prior to the study. Subjects were randomly divided into two groups, one group received HBO\textsubscript{2} therapy and the other was the control group. All procedures for this study were approved by the appropriate Ethics Committee at McGill University. The timeline of the procedures for the two groups is outlined in Table 1.

Table 1. Procedure Timeline

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Baseline</th>
<th>Day 0</th>
<th>Day 1</th>
<th>Day 2</th>
<th>Day 3</th>
<th>Day 4</th>
<th>Day 7</th>
<th>Day 14</th>
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<tbody>
<tr>
<td>Eccentric Exercise</td>
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<td></td>
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<tr>
<td>HBO\textsubscript{2} Treatment</td>
<td>XX</td>
<td>X</td>
<td>X</td>
<td>X</td>
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<tr>
<td>(HBO\textsubscript{2} group only)</td>
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<tr>
<td>Blood Sample</td>
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<td>X</td>
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<td></td>
<td>X</td>
<td>X</td>
<td>X</td>
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<tr>
<td>Isokinetic Strength</td>
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<td></td>
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<td></td>
<td></td>
<td>X</td>
<td>X</td>
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<tr>
<td>Rating of Soreness</td>
<td>X</td>
<td>X</td>
<td>X</td>
<td>X</td>
<td>X</td>
<td>X</td>
<td>X</td>
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<td>X</td>
<td>X</td>
<td>X</td>
<td>X</td>
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</table>
**Eccentric Exercise Procedure:** The subjects performed an eccentric exercise task (day 0) involving the quadriceps femoris, which is known to induce temporary muscle soreness. The eccentric exercise task to induce muscle soreness was performed on a dynamic leg extension apparatus (Polaris, Irontco Canada) using one leg. This apparatus is commonly used in weight training programs. The starting weight was equivalent to 120% of the peak force obtained during an isokinetic dynamometer test at 60 °/s. For this study, the investigator lifted the weight, and the subject beginning at full extension lowered the weight slowly (4 s) until the knee was flexed at a 90° angle as described by Lecomte et al. (21). When the subject was no longer capable of lowering the weight in 4 s, the weight was decreased by 7 kg increments. This procedure was repeated with the new weight. The protocol continued until the subject could no longer support a 7 kg weight. Using this protocol, subjects required from 7 to 10 sets and 6 to 15 repetitions per set in order to reach the endpoint of 7 kg. The exercise session was about 20-25 minutes in duration. The exercise caused quadriceps muscle soreness that was monitored by the research team which included sport medicine physicians.

**Hyperbaric Oxygen Procedure:** For the group treated with HBO_2_, there were five HBO_2_ treatments. The treatments were administered in a monoplace hyperbaric chamber (Sigma Plus, Perry Baromedical, Riviera Beach, FL) with the subjects breathing 95% oxygen. The treatment protocol was 2.5 atm abs (253 kPa) for 100 minutes:

- Tx 1 = in the hour following the exercise-induced soreness (Day 0)
- Tx 2 = 6 hours later on the same day (Day 0)
- Tx 3 = one treatment on the next day (Day 1)
- Tx 4 & 5 = two treatments on Day 2, separated by 6 hours

Two 5-minute air breaks, located at the 30-minute and 65-minute mark of the treatment, were administered via a plastic breathing mask with a demand inhalator valve. The purpose of the air breaks was to reduce the risk of oxygen toxicity.

The hyperbaric oxygen treatments were administered by a trained technician. A physician, knowledgeable in hyperbaric emergency procedures was available during the HBO_2_ treatments.

**Assessment:** The dependent variables for this investigation were: plasma creatine kinase, isokinetic peak torque and average power measured at 60, 180 and 300 °/s, rating of muscle soreness, and leg circumference. Table 1 outlines the sequence for these assessments.

Antecubital venous blood samples were drawn for analysis of plasma creatine kinase level. Samples were collected without stasis at baseline and days 0, 1, 4, 7 and 14. Total CK activity was assayed using the Synchron LX Systems (Beckman Coulter, Inc., Fullerton, CA). This system monitors the change in absorbance at 340 nanometers with the change in absorbance proportional to the activity of CK in the sample. Plasma reference values for CK range from 5 to 140 U/L. Plasma CK is frequently used as a marker of muscle cell damage. Peak concentrations are variable and can reach several thousand times the basal value (7). Elevated plasma CK is a consistent finding associated with delayed onset muscle soreness (1, 3, 7), however it may not coincide with peak values of perceived soreness. It is important to consider the quantity and the intensity of physical activity done after eccentric exercise because this can greatly influence the clearance as well as the liberation of this enzyme.

Isokinetic strength and power were measured using a Biodex dynamometer. Subjects performed knee extension-flexion movements at three velocities with 5 reps at 60 °/s, 20 reps at 180 °/s and 10 reps at 300 °/s. Peak torque (Nm) at each velocity was calculated as the highest value of torque developed throughout the range of motion. Average power (watts) was calculated as the total extension work divided by the time to complete the work. Results from the isokinetic
test at 180 °/s were used to obtain fatigue scores. Fatigue was calculated as the ratio of the difference, expressed as a percentage, between work in the first third (reps 1 to 6) to work in the last third (reps 15 to 20) of the test. The denominator in calculating the ratio of differences was the work completed in the first third of the test. Strength and power were recorded for the exercise leg and reference leg at baseline and days 2, 4 and 14.

Leg circumference measurements were taken at the mid-point between the greater trochanter of the femur and the middle of the patella with the subject in a standing position with weight distributed equally on both legs. Leg circumferences were obtained at baseline and days 0, 1, 2, 3, 4, 7 and 14. A mean of three measurements was used for the exercised and reference leg.

Rating of perceived soreness was scored using a visual analogue scale (VAS) (26). The VAS measures the intensity or magnitude of pain along a continuous scale. It consists of a straight line of 100 mm in length, where the ends are defined in terms of the extreme limits of the pain experience, going from no pain to the worst pain ever experienced. Pain ratings were obtained at baseline and days 0, 1, 2, 3, 4, 7 and 14.

Statistics. A 2-way repeated analysis of variance [group (HBO$_2$ vs control) and time (days 0, 1, 2, 3, 4, 7, 14)] was used to analyze the dependent variables (plasma creatine kinase, isokinetic peak torque, average power, fatigue, perceived soreness, and leg circumference). Following the ANOVA, significant F values were examined using planned comparisons ($p \leq 0.05$). Data analyses were performed with SYSTAT 9.0 (SPSS, Inc., Chicago, IL). No attempt was made to examine potential gender differences.

RESULTS

Plasma Creatine Kinase: Figure 1 illustrates the plasma CK results. There was no significant difference between the HBO$_2$ and control groups ($F = 1.13; P = 0.31$). There was a significant main effect for time ($F = 2.62; P = 0.03$) with the eccentric exercise elevating the plasma CK values on days 0, 1 and 4 compared to the baseline value. While CK peaked faster in the control group, there was large variability associated with the response. Mean values were similar by day 4. The ANOVA analysis revealed no significant interaction between time and group ($F = 1.07; P = 0.38$).

Muscle Soreness: Figure 2 illustrates the rating of soreness results. As expected, there was a significant main effect for time ($F = 16.82; P < 0.01$). Compared to baseline, perceived muscle soreness was higher on days 0, 1, and 2. On day 3, perceived muscle soreness was still elevated for the control group but not different from baseline for the HBO$_2$ group. Before the eccentric exercise, muscle soreness was rated at 5.6 mm on the 100 mm scale with peak values of 34.4 mm on day 1 and 30.0 mm on day 2. There were no significant differences between groups ($F = 1.60; P = 0.23$) and the interaction of groups X time ($F = 1.38; P = 0.22$).

Leg Circumference: There were no significant differences between groups ($F = 0.17; P = 0.68$), between legs ($F = 0.06; P = 0.80$), or across time ($F = 0.96; P = 0.46$).

Quadriceps Strength: Table 2 summarizes the isokinetic peak torque results. There were no significant differences between groups at 60 °/s ($F = 0.19; P = 0.66$), at 180 °/s ($F = 0.75; P = 0.39$), and 300 °/s ($F = 1.46; P = 0.24$). Similarly, there were no significant differences between the exercised and reference legs at 60 °/s ($F = 0.33; P = 0.57$), at 180 °/s ($F = 0.00; P = 0.96$), and 300 °/s ($F = 0.00; P = 0.99$). When assessed across time, there were no changes in peak torque at 60 °/s ($F = 1.37; P = 0.26$) and 180 °/s ($F = 0.33; P = 0.81$), however peak torque was different.
across time when measured at 300 °/s (F = 3.85; P = 0.01). These changes were unrelated to group or leg.

Average power of the quadriceps at 60, 180 and 300 °/s was not significantly different between the HBO₂ and control groups. Statistical analyses of these data reflected the analyses for quadriceps peak torque. Results from the isokinetic test at 180 °/s were used to obtain fatigue scores. There were no significant differences between groups, between legs, and across time in the fatigue patterns at 180 °/s.

**Figure 1.** Plasma Creatine Kinase over Time

![Plasma Creatine Kinase over Time](http://rubicon-foundation.org)

**Figure 2.** Rating of Soreness over Time

![Rating of Soreness over Time](http://rubicon-foundation.org)
Table 2. Quadriceps Peak Torque (Nm) for Control and HBO₂ Groups (mean ± SD)

<table>
<thead>
<tr>
<th></th>
<th>Control Group</th>
<th>HBO₂ Group</th>
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<tbody>
<tr>
<td></td>
<td>Exercised Leg</td>
<td>Reference Leg</td>
</tr>
<tr>
<td>Day</td>
<td></td>
<td></td>
</tr>
<tr>
<td>60 °/s</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Baseline</td>
<td>140.9 ± 32.6</td>
<td>149.5 ± 41.0</td>
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<tr>
<td>2</td>
<td>140.5 ± 32.1</td>
<td>145.4 ± 30.3</td>
</tr>
<tr>
<td>4</td>
<td>138.9 ± 45.9</td>
<td>145.8 ± 45.9</td>
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<tr>
<td>14</td>
<td>133.4 ± 40.8</td>
<td>138.8 ± 44.7</td>
</tr>
<tr>
<td>180 °/s</td>
<td>107.3 ± 26.6</td>
<td>108.7 ± 31.4</td>
</tr>
<tr>
<td>Baseline</td>
<td>110.2 ± 34.1</td>
<td>104.2 ± 26.6</td>
</tr>
<tr>
<td>2</td>
<td>107.1 ± 24.4</td>
<td>106.3 ± 31.5</td>
</tr>
<tr>
<td>4</td>
<td>106.1 ± 23.9</td>
<td>104.3 ± 27.7</td>
</tr>
<tr>
<td>14</td>
<td>83.5 ± 36.8</td>
<td>83.2 ± 26.2</td>
</tr>
<tr>
<td>300 °/s</td>
<td>90.8 ± 28.7</td>
<td>94.4 ± 32.7</td>
</tr>
<tr>
<td>Baseline</td>
<td>94.3 ± 29.8</td>
<td>86.1 ± 23.5</td>
</tr>
<tr>
<td>14</td>
<td>88.0 ± 23.9</td>
<td>87.0 ± 28.4</td>
</tr>
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</table>
DISCUSSION

Acute bouts of high force eccentric exercise cause DOMS (3,7,23). Four other studies (22-25) have used HBO2 in DOMS. The protocols for their HBO2 and placebo treatments were different. Only the study by Staples et al. (24) suggested that HBO2 treatments were beneficial in the treatment of DOMS by enhancing recovery of eccentric torque. Our results are in agreement with the conclusions by Mekjavik et al. (23), Harrison et al. (22), and Webster et al. (25) in that HBO2 is not an effective therapy for the treatment of DOMS.

The mode of exercise to induce temporary muscle soreness may have influenced the recovery of functional capacity in these studies. The exercise protocol would vary the amount of muscle damage (biochemical, contractile, functional and sensory properties) between subjects. Another factor determining this response would be the recent exercise history of the subjects. This important characteristic has not been clearly described in some of the studies. Previous investigations (22-25) used only male subjects. Our sample included 10 females and 6 males who were not involved in weight training using the quadriceps femoris for three months prior to the study. In the other studies, the subjects were described as healthy male university students (n = 12) who had not performed weight training or strenuous eccentric training of the lower leg during the previous 3-month period (25), 21 college-aged males (22), 24 healthy male subjects (23), and 66 male subjects randomly assigned to 4 groups described as control, immediate HBO2, delayed HBO2, and sham (24). Athletic subjects with previous exposure to resistance exercise may have pre-adapted to muscle damage with a faster recovery to baseline muscular performance levels (27-28) whereas sedentary subjects would be more susceptible to muscle damage.

It is believed that the extent of muscle injury due to eccentric exercise is related more to the muscle's change in length rather than the amount of force generated by the muscle (8). Tension and strain may produce damage, and eccentric exercise performed at a longer muscle length seems to cause more damage than if the same exercise is done at a shorter muscle length.

The mode of exercise used to create muscle soreness varied among the studies that have investigated the effects of HBO2. Staples et al. (24) used an isokinetic dynamometer with subjects exerting maximum resistance to the downward force of the dynamometer arm (30 °/s). A total of 30 sets of 10 repetitions each were performed in 30 minutes with the quadriceps muscles. Mekjavik et al. (23) created soreness in the elbow flexor muscles using a modified preacher curl bench. Subjects performed 6 sets of 12 maximal repetitions. Harrison et al. (22) also used a preacher curl bench with subjects completing 6 sets of 10 repetitions at 120% of maximum concentric strength. Webster et al (25) used an eccentric exercise protocol designed to elicit muscle damage within the medial gastrocnemius muscle. The load was 80% of 1-RM with 5 sets of calf raises to failure with only 2-minutes of recovery between sets. This protocol differed from the other studies in that it contained a substantial concentric (shortening) as well as an eccentric (lengthening) component. In our protocol, subjects required from 7 to 10 sets and 6 to 15 repetitions per set in order to complete the exercise. The session was about 20-25 minutes in duration. After these eccentric exercise protocols, plasma CK levels and perceived muscle soreness were elevated and muscle force was reduced.

The HBO2 interventions have also varied and may have influenced the final conclusions. These interventions have included 5 HBO2 sessions for 100-min at 2.5 atm with one group receiving the first treatment with-in 2 hours of eccentric exercise and another group receiving
delayed HBO$_2$ that was initiated 24 hours after the exercise (22). The protocol of Mekavic et al (23) involved 7 HBO$_2$ sessions for 60-min at 2.5 atm abs. The protocol of Webster et al (25) included only 3 HBO$_2$ sessions for 60-min at 2.5 atm abs. The first treatment was administered 3-4 h after damage, with the second and third treatments at 24 and 48 h after the first treatment, respectively. After the induction of muscle soreness, Staples et al (24) randomly assigned their subjects into four groups for the first phase (control, HBO$_2$, delayed treatment, and sham treatment) and into three groups for the second phase (3 days of treatment, 5 days of treatment, and sham treatment). Subjects were treated over a 5-day period. The HBO$_2$ treatment involved 100% oxygen (60 min at 2.0 atm) and the sham treatment involved 21% oxygen (60 min at 1.2 atm). The subjects in the Staples et al (24) study were probably not truly blind to the conditions since chamber pressures were different for control and HBO$_2$ groups and would likely be evident in a study involving repeated trials. This blinding strategy is likely less effective than an experimental design with pressurization to 2.0 atm abs with a hypoxic breathing mixture (0.11 atm abs) to provide normoxic conditions.

The eccentric exercise utilized in this study brought about a significant rise in plasma CK and muscle soreness, but five HBO$_2$ treatments applied immediately post-exercise and spread over three days did not alter the response. Plasma CK, quadriceps strength and power, perceived soreness, and leg circumference were not affected by HBO$_2$. Staples et al. (24) also studied HBO$_2$ treatments following eccentric exercise. They conducted a randomized, controlled, double blind, prospective study with 66 subjects. After the induction of quadriceps muscle soreness, subjects were randomly assigned into four groups (control, HBO$_2$, delayed treatment, and sham treatment). Eccentric quadriceps peak torque was lowered following the exercise that created DOMS. In contrast to our study, five HBO$_2$ treatments benefited the recovery of eccentric torque. Their HBO$_2$ treatments were also administered immediately following the eccentric exercise. Their findings were attributed to a combination of the edema-reducing properties of HBO$_2$, reduced neutrophil adhesion to injured-muscle cells and free-radical-quenching ability associated with HBO$_2$. They cautioned about attributing the beneficial torque results to the absolute efficacy of HBO$_2$. The differences between studies cannot be attributed to muscle pain as their muscle soreness rating were similar to our values at 48 hours post-exercise. Possibly the differences between the present study and that of Staples et al. were due to the mode of evaluation. We measured concentric peak torque with extension-flexion movements using a Biodex dynamometer while Staples et al. (24) measured the eccentric peak torque using an isokinetic dynamometer with resistance measured against downward force of the dynamometer arm (30 °/s). This difference in methodology with measurements recorded during muscle shortening (concentric) versus lengthening (eccentric) must be acknowledged since the extent of muscle injury due to eccentric exercise relates more to the muscle's change in length rather than the amount of force generated by the muscle (8).

Mekjavic et al. (23) also tested their subjects using a maximal strength test. Male subjects were randomly assigned to either a placebo group or a HBO$_2$ group in a double-blind, randomised controlled study. Subjects were tested for maximal isometric strength (pre-exercise) of the elbow flexors. Muscle soreness was created by a high-force eccentric workout involving the elbow flexor muscles. On the seven successive days after this workout, subjects were exposed to a hyperbaric environment of 2.5 atm abs for 60 minutes, inspiring either a normoxic mixture (PiO$_2$ = 0.2 atm abs; placebo group) or a hyperoxic gas mixture (PiO$_2$ = 2.5 atm abs; HBO$_2$ group). They found no difference in the rate of recovery of muscle strength, upper arm circumference and rating of perceived muscle soreness between the two groups. They also found
that perceived soreness peaked at about 48 hours after exercise. They concluded that HBO₂ was not an effective therapy for the treatment of DOMS.

Harrison et al. (22) also examined the role of HBO₂ in the treatment of exercise-induced muscle injury to the forearm flexor muscles. After the eccentric exercise, the HBO₂ treatment was given daily through day 4 post-exercise. The HBO₂ treatments were administered at the same pressure and duration as our study. Their results suggested that HBO₂ was not effective in treating exercise-induced muscle injury as indicated by isometric strength, serum CK levels, rating of perceived soreness, forearm flexor cross-sectional area, and T2 relaxation time via magnetic resonance imaging. Possibly the HBO₂ treatment protocol was not optimal for this type of muscle injury. They suggested that modifying the HBO₂ treatment by reducing the delay between injury and exposure to less than 2 hours or by manipulating the number of treatments per 24-h period may prove to be beneficial in reducing the initial inflammatory response. Following our eccentric exercise to create muscle soreness, we administered 2 HBO₂ treatments within 12 hours with the first treatment immediately post-exercise and arrived at the same conclusion.

Webster et al (25) examined if HBO₂ therapy could accelerate recovery from exercise-induced muscle damage in humans. The first HBO₂ treatment was administered 3-4 hours after muscle damage. There was little evidence of a difference in recovery rate between the HBO₂ and sham groups, however faster recovery was observed in the HBO₂ group for isometric peak torque, pain sensation and unpleasantness. On day 2 following the exercise protocol, the isometric peak torque of the HBO₂ group was not significantly different from baseline whereas the sham group was significantly lower than baseline. It was stated that HBO₂ appeared to confer a protective effect against the loss of isometric strength after muscle damage. HBO₂ was reported to promote a quicker recovery from pain and unpleasantness from days 2 to 5 post-damage. Our data also suggest a faster recovery for the HBO₂ group. On day 3 following the exercise protocol, perceived muscle soreness was still elevated for the control group but not different from baseline for the HBO₂ group.

The response to eccentric exercise can vary. It is well known that subjects who are unaccustomed to eccentric exercise will experience greater muscle soreness (9, 10). The preponderance of findings suggests that HBO₂ therapy has no effect on the course of DOMS. It should be noted, however, that there is a wide variation in the DOMS response. Harrison et al. (22) labelled two subjects as “high CK responders”. Staples et al. (24) rejected four subjects who got stronger after induction of DOMS. We had one subject who was dropped from the protocol because of a quadriiceps muscle compartment syndrome. It is known that in mutations in the dystrophin gene increases the susceptibility of muscle cells to oxidative stress (29; 30). It is not known if any of the rejected subjects had dystrophin mutations, or whether HBO₂ played a provocative or palliative role in these subjects. The role of genetic variability in susceptibility to DOMS, and possible interaction with HBO₂, deserves further study.

In summary, this study examined the effects of HBO₂ therapy on exercise-induced muscle soreness. Following DOMS induced by eccentric exercise, changes in plasma CK, perceived muscle soreness, leg circumference, quadriiceps peak torque, quadriiceps average power and fatigue were not altered by HBO₂. After eccentric exercise, plasma CK and perceived soreness were elevated but were not different between HBO₂ and control groups. HBO₂ therapy did not alter leg circumference, quadriiceps peak torque, average power or fatigue compared to a control group. Faster recovery was observed in the HBO₂ group on day 3 following the exercise
protocol with perceived muscle soreness elevated for the control group but not different from baseline for the HBO₂ group.

REFERENCES