

Hyperbaric oxygen and muscle performance in maximal sustained muscle contraction

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ABSTRACT

Purpose: The purpose of this study was to examine the effects of hyperbaric oxygenation (HBO₂) on sustained maximal muscle contraction.

Methods: Fifty-two healthy volunteers participated in the study. Thirty-four experimental subjects breathing 100% oxygen at 253kPa (2.5ATA) in a multiplace hyperbaric chamber performed a maximal grip contraction for one minute (initial grip) followed by a 30-second rest period and another one-minute maximal contraction (recovery grip). The protocol was repeated one week later inside the chamber while subjects were breathing normobaric air. A control group of 18 subjects completed the same two-week protocol but breathing normobaric air during both sessions to assess any changes due to learning effect.

Results: Exposure to HBO₂ significantly increased force production for initial maximal grip, recovery maximal grip and total one-minute effort. Time to decrease to 50% of initial contraction was shorter with HBO₂ for both initial grip and recovery grip, but force production remained higher throughout the effort with HBO₂.

Conclusions: These data suggest that when performing sustained maximal contractions during acute exposure to HBO₂, overall contractile force may be significantly increased compared with breathing normobaric air. Initial rate of fatigue is higher with HBO₂, perhaps due to increased extravascular compression with the initial greater force production.

INTRODUCTION

Muscle fatigue may be described as decreased ability to repeat or sustain a muscle contraction [1]. A number of cellular mechanisms are thought to contribute to this decline in muscle performance, including mechanisms related both to increases in oxygen utilization and decreases in oxygen delivery. However, the relative contribution of these factors remains unclear.

When the force of a muscle group in an intact individual exceeds approximately 50% of maximum, the circulation begins to collapse due to extravascular compression, markedly decreasing perfusion of the working muscle. Oxyhemoglobin in that relatively static blood can decrease rapidly during the first 10 seconds, corresponding with a significantly decreased contractile capacity [2,3].

If the contraction is submaximal, the pressor response then causes an increase in mean arterial pressure (MAP), which may restore perfusion and help sustain the muscle contraction [4,5,6,7]. With submaximal contraction of long duration, fatigue occurs relatively slowly, and complete recovery from fatigue may take hours to days [6,7]. However, with maximal muscle contractions the decrease in perfusion is more dramatic. Extravascular compression is such that resultant increases in MAP are insufficient

to restore tissue perfusion, and fatigue typically occurs rapidly, with large decreases in maximal force within the first few seconds. This type of fatigue typically recovers very rapidly as well, with considerable restoration of force occurring within the first several seconds of recovery [8,9].

Mechanisms not directly related to intracellular changes have been found to affect muscle fatigue. For instance, neural changes altering the ability to recruit muscle fibers, perhaps in response to hypoxia or acidosis, appear to play a significant role [3,10,11,12]. However, much muscle research is performed on isolated muscle, and it is generally agreed that a substantial component of muscle fatigue relates to decreased tissue O₂ tension and takes place within the contractile mechanism of the muscle itself [1,3,13,14,15,16,17].

Hemoglobin is essentially fully saturated with oxygen while breathing normal atmospheric with a relatively insignificant amount of O₂ carried in solution in the plasma. When breathing normobaric air, arterial PO₂ (partial pressure of oxygen) is approximately 13.3 kPa, and tissue O₂ tension is approximately 7.3kPa. At this PO₂, the oxygen carrying capacity of blood is relatively stable at 20ml/dL, with almost all oxygen carried as oxyhemoglobin. Hyperbaric oxygenation (HBO₂) can

produce substantial increases in plasma oxygen partial pressures and tissue oxygen tension, as well as increased oxygen-carrying capacity of blood [18,19].

In this study, exposure to 100% oxygen at 2.5 atmospheres absolute pressure (ATA) produced PO₂ in the breathing gas of 253kPa. This can produce arterial PO₂ (PaO₂) 19 times as high as when breathing normobaric air. Not only is PaO₂ greatly increased, but the oxygen-carrying capacity due to O₂ dissolved in plasma may increase from 0.3ml/dL to 5.0ml/dL, and tissue O₂ tension may exceed 50kPa, resulting in “superoxygenation” of the muscle fiber [18,20,21]. Both O₂ loading and intracellular stores will be increased. High tissue O₂ will fully saturate myoglobin, normally saturated to about 90% under normobaric conditions, creating an increase in intracellular O₂ stores. This could potentially improve muscle performance for the first few seconds of maximal contraction [22].

We did not directly measure muscle O₂ tension in this study. However, based on the studies cited, we assume that the hyperbaric exposure our subjects experienced should have produced approximately the same level of “superoxygenation” as was reported by those previous studies under the same HBO₂ exposures.

Hyperbaric oxygenation has been touted, by some as a means for enhancing muscle performance and athletic performance [23,24,25]. For this reason, it is being used by some as an ergogenic aid in an attempt to improve athletic performance or to promote recovery from exercise-induced muscle damage or fatigue. Many claims are based on studies that rely on anecdotal evidence or studies with limited study design [23]. Most of the well-designed studies have reported little improvement with the application of HBO₂ in most variables related to athletic performance, reduction in soreness, or recovery from exercise-induced muscle injury [26,27, 28,29,30,31].

A primary limitation on research to date is that studies have typically examined the effects of HBO₂ after, rather than during, exposure to HBO₂, and may have missed the acute effects of superoxygenation of the tissues during hyperbaric exposure. We hypothesize that superoxygenation during acute HBO₂ exposure may enhance the ability of a muscle to generate force. Therefore, the purpose of this study is to examine the acute effects, during exposure to hyperbaric oxygenation, on maximal muscle tension, muscle fatigue, and on recovery from fatigue after maximal sustained muscle contraction.

MATERIALS AND METHODS

Subjects

A total of 55 healthy volunteers were recruited for the study and asked to refrain from exercise for at least 48 hours prior to any data-collection session. Subjects were excluded if they had any contraindications to hyperbaric oxygen treatment, had any musculoskeletal injury or were taking any medications that might affect muscle performance. The study was approved by the Human Subjects Review Board of Saba University, and all subjects gave written informed consent. Three subjects were unable to complete the protocol due to the inability to equalize their ears during pressurization, resulting in a final *N* of 52, with 34 subjects assigned to the experimental group and 18 subjects to a control group.

The 34 experimental subjects were the first subjects recruited, and the first to complete the protocol. The control subjects were recruited afterward and completed the protocol on two subsequent weeks to assess any possible changes due to training effect. This was felt to be necessary since all subjects in the experimental group performed the HBO protocol first and the normobaric protocol second. Subject characteristics were:

- Subjects were all students at the Saba University School of Medicine; mean age =26 years, range 23-30 years.
- Experimental group; male *n*=25, female *n*=9.
- Control group; male *n*=10, female *n*=8.

Protocol

On their first test day, experimental subjects were pressurized to 253kPa in a four-person multiplace 5,500-square-foot Class I hyperbaric chamber (Lucas Diving, Ltd.). Two subjects were placed in the chamber at a time, as there were two complete oxygen delivery systems in the chamber. Subjects were placed at the opposite ends of the bench inside the chamber and instructed not to talk or otherwise interact with each other during the protocol.

Descent to 50 feet of sea water (fsw) followed normally accepted diving descent rates and was completed in four to five minutes, or approximately 0.5ATM/minute. After pressurization, subjects were asked to breathe 100% O₂ for five minutes via tight-fitting oxygen masks to allow substantial oxygen loading into the plasma, and to allow time for this blood to perfuse the muscles of the forearm.

Following this period of oxygen loading, and while continuing to breathe hyperbaric oxygen, test subjects were asked to perform the grip strength test described

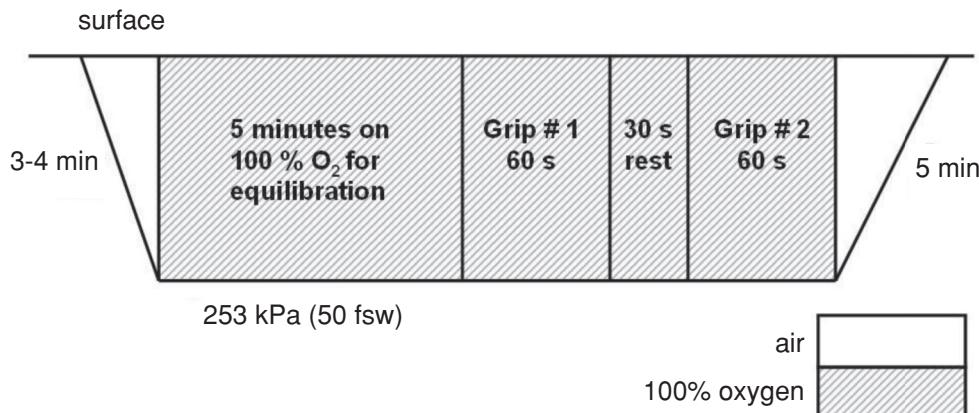
FIGURE 1

FIGURE 1: Dive profile showing times for descent and ascent breathing normobaric air, and the grip and rest sessions during exposure to hyperbaric oxygen.

below (the time-course of the protocol is shown in *Figure 1, above*). Seven days later each subject returned and repeated the grip strength test in the same position inside the hyperbaric chamber with the door sealed and air running into the chamber but without being pressurized, and while breathing atmospheric air through a “sham” mask. Subjects in the control group later performed the two protocols, also one week apart, under identical conditions inside the hyperbaric chamber, but without pressurization and breathing normobaric air through the sham mask.

Temperature inside the chamber was maintained via cooling of the compressed air prior to introduction into the chamber, and then by continuous mild flow during the protocol. Chamber temperature prior to pressurization was approximately 25.5°C, and after pressurization at 253kPa was approximately 26.5°C.

Grip strength test

Each subject was instructed to sit upright and lean forward slightly in the chamber so that his or her back did not contact the hull of the chamber. Using the dominant hand, the subject was asked to grip the Dynapac Systems Digital Hand Dynamometer, Model SD121C, connected via a cable through a penetrator in the chamber hull to a DA100 transducer amplifier and MP100 multimodular system (Biopac Systems, Inc.) located outside the chamber.

The subject held the hand slightly to the outside of the thigh, palm facing upward, elbow flexed to a 90° angle. At the signal, the subject was told to grip maximally and sustain that same grip for one minute without regripping or adjusting the hand (*Figure 2 Max-I, Page 486*). After one minute the subject was told to relax only the grip for 30 seconds without readjusting the hand or arm. Following the 30-second rest period, the subject was instructed to repeat the maximal contraction, again for one minute. Subjects were given a 10-second warning prior to each grip and received no interaction or encouragement during the tests.

Data processing

Each effort was recorded at a sampling rate of 200/s. Force curves for initial (I) grip and recovery (R) grip were analyzed for maximal grip (Max-I and Max-R respectively), time to decrease to 50% of maximal grip (50%-I and 50%-R respectively), and minimal contraction at the end of 60-second effort (Min-I and Min-R respectively), which was the average of the last five seconds of the contraction. Total effort (TE-I and TE-R) was calculated by measuring the total area under the curve and is expressed as total kilograms \times seconds (kg \cdot s). Percent of initial contraction, as a measure of recovery, was calculated as the maximal recovery grip divided by the maximal initial grip.

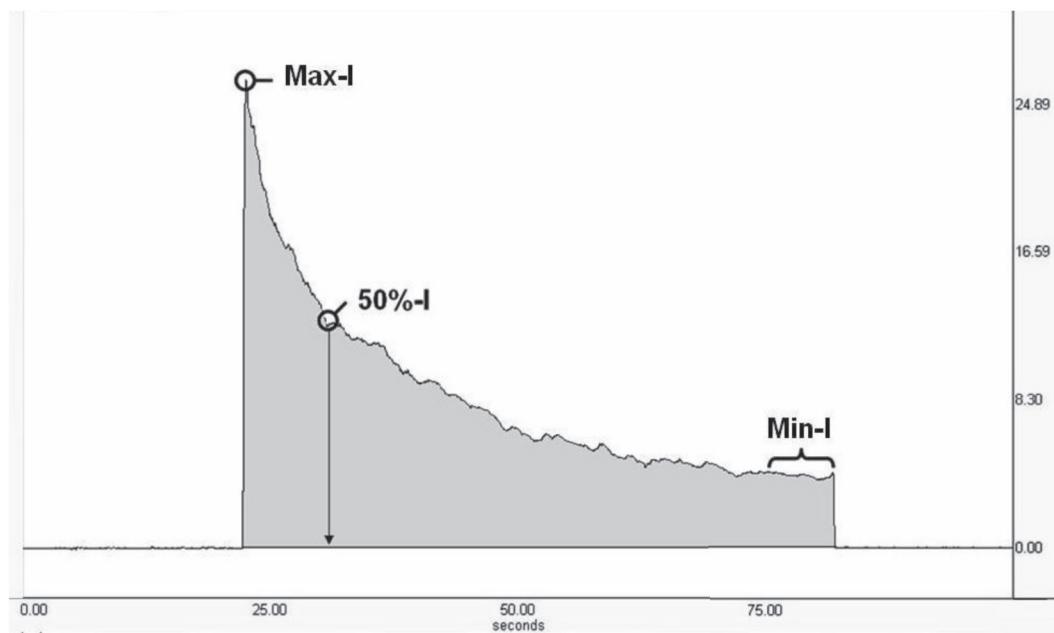
FIGURE 2

FIGURE 2: Representative individual subject force curve. Typical individual subject force change during the initial grip effort. The curve represents the raw data points obtained at sampling rate of 200/s without further data processing or curve fitting.

Statistical analysis

Data were analyzed utilizing GraphPad InStat software (San Diego, Calif.) repeated measures analysis of variance (ANOVA) $\alpha=0.05$, followed by a Tukey-Kramer multiple comparisons test. The experimental subjects served as their own controls in this test-retest protocol, and were not actually compared to control subjects. Therefore the repeated measures ANOVA was utilized to evaluate the two conditions for the experimental group. The control group was utilized separately to reveal any possible learning effect, as all experimental subjects performed the HBO₂ protocol first, followed by the normobaric test one week later. Data from the control group therefore were separately analyzed by a repeated measures ANOVA. Normality test was passed for all parameters.

RESULTS

Exposure to hyperbaric oxygen in the experimental group significantly increased maximal initial force of contraction for both initial grip (Max-I $p<0.001$) and recovery grip (Max-R $p<0.001$). Total effort was greater with hyperbaric oxygenation for both initial grip (TE-I $p<0.01$) and recovery grip (TE-R $p<0.001$). Exposure to HBO₂ decreased the time it took for force to drop to 50 percent of initial force for both initial grip (50%-I $p<0.01$) and recovery grip (50%-R $p<0.01$). However, as the initial HBO₂ force was so much higher, force remained higher with HBO₂ throughout the entire effort. Minimal grip at the end of each contraction tended to be higher with HBO₂ but just failed to achieve significance ($p>0.05$). There was no difference in the recovery parameter calculated as the maximal recovery grip as a percentage of the initial grip, though again it tended to be higher with HBO₂ ($p>0.08$) (Tables 1-2, facing page).

TABLE 1

INITIAL GRIP	Normobaric air mean ± SD	Hyperbaric oxygen mean ± SD	% change with HBO₂	p
Maximal initial grip (kg)	24.40 ± 9.12	30.27 ± 10.87	24%	<0.001
Time to 50% of max (sec)	17.6 ± 11.58	12.4 ± 7.76	-41%	<0.001
Minimal grip, avg. last 5 sec (kg)	4.83 ± 3.42	5.75 ± 3.92	20%	.06
Total effort (kg•s)	635.3 ± 330.5	718.3 ± 328.4	13%	<0.01

This table displays the results for the initial grip effort for the experimental group.

TABLE 2

RECOVERY GRIP	Normobaric air mean ± SD	Hyperbaric oxygen mean ± SD	% change with HBO₂	p
Maximal recovery grip (kg)	18.23 ± 1.26	23.77 ± 1.80	30%	<0.001
% of maximal initial grip	75.2% ± 0.02	79.8% ± 0.04	4.6%	0.08
Time to 50% of max (sec)	11.72 ± 1.28	9.45 ± 1.79	-24%	<0.01
Minimal grip, avg. last 5 sec (kg)	2.82 ± 0.41	3.61 ± 0.49	28%	0.05
Total effort (kg•s)	411.0 ± 209.3	508.2 ± 605.4	24%	<0.001

This table displays the results for the recovery grip effort following a 30-second rest period.

No significant differences were seen in the control subjects between Weeks 1 and 2 for any parameters, thus reducing the possibility of a significant learning effect or fatigue effect.

The averaged time-course of the grip strength for all subjects under hyperbaric and normobaric conditions is shown in *Figure 3*, (Page 488). The hyperbaric curves are higher, for both initial and recovery grip, than the corresponding curves for normobaric air.

DISCUSSION

The most dramatic effect of exposure to HBO₂ revealed by this study is the increase in maximal grip, both at the beginning of the initial grip (24% increase) and the recovery grip (30% increase) compared with breathing normobaric air. The presumed superoxygenation of the muscle fibers appears to enhance the potential for crossbridge cycling and force production, particularly during the first 5-10 seconds of contraction.

A possible mechanism for fatigue during decreased muscle oxygenation may be an increase in intracellular ADP, which has been implicated in inhibiting cross-bridge detachment, and thus inhibiting the movement of actin during muscle contraction [15]. Westerblad *et al.* (2002) suggests that increases in intracellular inorganic phosphate (Pi) during muscle contraction may contribute to muscle fatigue by limiting Ca⁺⁺ release from the sarcoplasmic reticulum [32]. In our subjects, increases in tissue oxygen tensions prior to muscle contraction may have depressed the intracellular ADP and/or Pi concentrations, driving the reactions in the direction of ATP and PCr production, allowing for more successful cross-bridge cycling. Increased intracellular O₂ stores (full saturation of myoglobin) may have helped sustain the ATP/PCr production and may account for the ability to maintain force at a somewhat higher level throughout the effort.

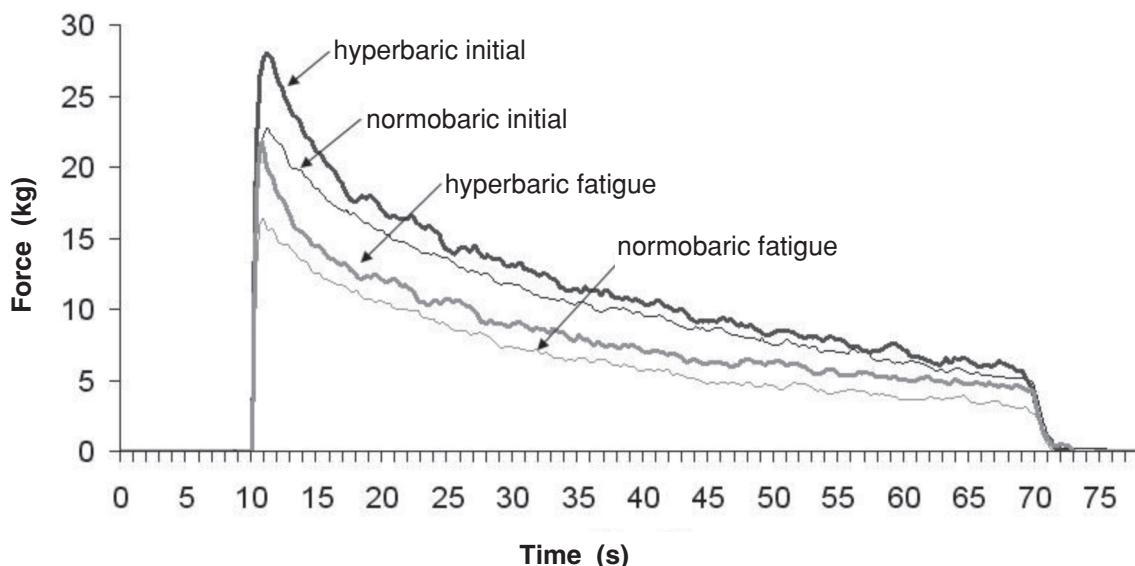
FIGURE 3

FIGURE 3: Averaged curves from the 34 experimental subject recordings for each of the four grip efforts. The curves represent the mean values at each data point with no curve fitting or smoothing applied.

Force among our subjects also consistently decreased more rapidly to 50% with HBO₂ than with normobaric air. This could be related to higher metabolism and utilization of O₂ with HBO₂, or to the fact that initial contractions were considerably higher, producing greater extravascular compression and more complete occlusion of blood flow. So, the increased ability to produce initial force may have been directly responsible for the more rapid drop in force seen with higher initial grip. Even though force dropped more rapidly to 50% of maximum under hyperbaric conditions, the starting point was high enough that force was still somewhat higher at all points along the curve compared to normobaric conditions. That difference decreased during the effort so that at the very end minimal sustained force tended to be higher with HBO₂ only. Higher extravascular compression with HBO₂ may have prevented reperfusion to the extent that the advantage of higher O₂ stores, and therefore ATP/PCr stores, gradually decreased during the one-minute effort. The generally sustained higher force throughout the grip effort is further reflected by the increase in total effort (both TE-I and TE-R) which was higher with exposure to hyperbaric oxygen.

A slight increase in Max-R as a percentage of Max-I, although not statistically significant, shows a trend toward more rapid recovery with HBO₂. The actual grip forces, including the recovery grip forces were all higher with HBO₂, but the percentages of recovery grip compared with initial grip only tended toward being higher. Furthermore, despite stronger grip initially and during most of the effort, the minimal grip force at the end of the effort was not significantly improved with HBO₂, just missing significance at $p=0.05$ (Min-I) and $p=0.06$ (Min-F). This suggests that with a maximal grip effort HBO₂ had somewhat less effect on fatigue and recovery from fatigue than on the initial contractile capabilities of the muscle.

It is possible that additional equilibration time breathing hyperbaric oxygen might have produced an even greater effect than seen with the five-minute HBO₂ exposure. However, during the first five minutes of exposure to hyperbaric oxygen, considerable oxygen loading takes place [33]. Even with significant vasoconstriction, as is the case in the tightly autoregulated brain, tissue oxygen tension can increase to 400% of normobaric

values during five minutes of exposure to HBO₂. The rate of oxygen loading levels off considerably after the first five minutes, though it may continue for another 15-20 minutes before complete equilibration is achieved [33,34]. Therefore, the five-minute oxygen loading period employed by this study likely produced most of the potential oxygen saturation possible in the skeletal muscle under investigation.

In the experimental group all HBO₂ results were obtained first, with the subjects returning for normobaric assessments a week later. This was done because any subject who could not complete the hyperbaric portion of the study would, therefore, be dropped from the study. This could possibly have introduced a training effect or fatigue effect to be seen in Week 2, following the subjects' single initial experience with the dynamometer. Therefore, we utilized an external control group to reveal any changes that might take place simply because of repeated protocols one week apart. The control group demonstrated no significant difference in any of the measured values between Weeks 1 and 2, leading to the conclusion that there was no significant learning effect or fatigue effect between weeks.

There is a possibility that a placebo effect may be responsible for some of the improvement seen under hyperbaric conditions. All conditions were controlled as strictly as possible, including the use of a sham mask for all normobaric sessions. However the researchers felt it would not be possible to reliably blind a large group of medical students to the fact that they were being pressurized to a depth of 50 feet. Subjects were told that the researchers did not expect any particular effect, were instructed not to expect any difference one way or the other, and to simply grip their hardest each time.

Research reports that muscle fatigue is different in males vs. females, with females showing a greater resistance to fatigue. Suggested mechanisms include differences in substrate utilization, muscle morphology and neurological stimulation [35] and a difference in muscle fiber type distribution [36]. However, in this study subjects were compared to themselves, not to each other. Therefore we felt it was acceptable to include both genders in the study, for an evaluation of HBO₂ effects on humans in general. A future look at gender differences in response to HBO₂ would be warranted.

Temperature increase due to adiabatic compression was not a factor in this study, as the increase during pressurization was restricted to approximately 1°C. Furthermore, while isolated muscle cells *in vitro* show considerable contractile changes with modest temperature changes, *in vitro* contractile properties in mammals have been found to remain relatively stable at temperatures ranging from 24-40°C [37].

The time course of fatigue agreed with previously published research, with large initial losses in the first 15 seconds (64% of total decrease) and a stabilization after that [12,38]. Little drop in force was observed during the final 15 seconds to the end of each contraction (11% of total decrease – *Figure 3*). This contractile stabilization may be due to the force of contraction having dropped below the critical 50% level, thus reducing extravascular compression and allowing some resumption of tissue perfusion [1,38].

Previous work in our laboratory revealed the ability of subjects to increase force of contraction, late in a “maximal” contraction, if they were given additional verbal encouragement. This may be due to an ability to recruit additional muscle fibers not initially recruited, particularly after force has decreased and some perfusion has resumed [12]. We wished to evaluate changes only in the original group of fibers recruited at the onset of the grip effort. Therefore, in this study our protocol instructed them to grip maximally and “lock” or “sustain” that exact contraction for as long as possible without “regripping,” and subjects were given no additional encouragement during the grip effort.

Subjects reported that it felt to them as though they were producing the exact same force throughout the entire minute of contraction, even though we could see the rapid loss in force on the external computer display. This produced very consistent and predictable decreases in force that appear related to the original muscle fibers recruited at the onset of the contraction.

This study produced consistently higher force production with HBO₂, but only allowed us to postulate as to the mechanisms. A follow-up study currently under way is utilizing EMG along with maximal grip in an attempt to determine if the effects are due to intracellular mechanisms as opposed to alterations in fiber recruitment patterns with exposure to hyperbaric oxygenation.

CONCLUSIONS

A short, sustained exposure to hyperbaric oxygen can enhance the ability of forearm muscles to produce force during maximal, sustained contractions, producing 25–30% higher maximal contractions both initially and after a brief period of recovery, and increased total force production of 13–24% during a one-minute effort. Drop in force to 50% of initial grip is more rapid with HBO₂. However, overall force remains higher throughout the one-minute maximal effort, with the effect steadily decreasing toward the end of the effort. This suggests that the advantage gained by increased oxygen tensions and phosphagen stores may be gradually lost during maximal grip effort. Recovery from fatigue has a non-significant tendency to improve with HBO₂.

ACKNOWLEDGEMENTS

The authors would like to thank the students of Saba University who volunteered to participate in this study.

The study was supported by a research grant from the Saba University Department of Research.

The results of the present study do not constitute endorsement by the Undersea and Hyperbaric Medical Society.



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