Hyperbaric oxygen and muscle performance in maximal sustained muscle contraction

Stewart J, Gosine D, Kaleel M, Kurtev A

The Saba University School of Medicine, Saba, Dutch Caribbean

CORRESPONDENCE EMAIL: James A. Stewart, Ph.D. – *jstewart@saba.edu*

ABSTRACT

Purpose: The purpose of this study was to examine the effects of hyperbaric oxygenation (HBO_2) 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 HBO_2 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 HBO_2 for both initial grip and recovery grip, but force production remained higher throughout the effort with HBO_2.

Conclusions: These data suggest that when performing sustained maximal contractions during acute exposure to HBO_2, overall contractile force may be significantly increased compared with breathing normobaric air. Initial rate of fatigue is higher with HBO_2, 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 O_2 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 O_2 carried in solution in the plasma. When breathing normobaric air, arterial PO_2 (partial pressure of oxygen) is approximately 13.3 kPa, and tissue O_2 tension is approximately 7.3kPa. At this PO_2, the oxygen carrying capacity of blood is relatively stable at 20ml/dL, with almost all oxygen carried as oxyhemoglobin. Hyperbaric oxygenation (HBO_2) can