Is the Clinical Use of a Hyperbaric Chamber as a Modality to Aid in Recovery Supported by Evidence?

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Abstract

Hyperbaric oxygen therapy use has become common practice amongst certain athlete groups. Theoretically, the additional oxygen available from a hyperbaric oxygen treatment aids the body in healing and recovery, creating an optimal environment for the body to heal faster than it normally would. Debate remains as to whether a treatment in a mild hyperbaric oxygen unit (cheaper and portable) can have the same beneficial results of a treatment in a standard hyperbaric oxygen unit. Results observed during the treatment of traumatic brain injury, post-concussion syndrome, delayed onset muscle soreness, and fatigue give reason to believe that potential benefits do exist. The data needed to authenticate the benefits of mild hyperbaric oxygen use as compared to standard hyperbaric oxygen exists in the various settings that use this modality. This data needs to be collected, analyzed, and disseminated to the research community and practitioners who are using these devices.

Keywords: Therapeutic modalities, best practice, injury management
1. Introduction/Background

We live in a day where athletes are continuously looking for the competitive advantage to give them the leg up on their opponent. Faster recovery can be the difference between a long term contract and unemployment. Juxtapose this to individuals who just want to get their lives back after suffering a traumatic brain injury, whether in sport, military service, or as an accident. Interestingly, hyperbaric oxygen therapy (HBOT) has been proposed as a treatment for both, albeit with different levels of evidence. In the paragraphs that follow, we will discuss the theory behind HBOT, as well as outline how HBOT and mild HBOT differ. This distinction is important as we consider whether this treatment is truly beneficial, or a placebo.

2. Theoretical Basis for Hyperbaric Chamber Use

Oxygen is critical for normal cellular function. When the body is in homeostasis, the blood delivers the required amount of oxygen to the cells for them to function properly. Injury removes the body from a homeostatic state and the flow of blood to the area is frequently compromised. This results in less oxygen being delivered to the tissues and additional tissue damage. The inflammatory process works to restore homeostasis quickly. Two circulatory changes occur in the area of tissue damage—arteries dilate and dormant capillaries and venules open. This increases total blood flow to the area, bathing the tissues with the oxygen needed for repair. In time, any damaged blood vessels are repaired as well.¹

A clinician uses HBOT to aid the inflammatory process in restoring the tissue to pre-injury conditions. Under normal circumstances, red blood cells are nearly 100% saturated with oxygen. Interestingly, plasma also has oxygen carrying capacity,² albeit rarely utilized under normal conditions. By breathing 100% oxygen in a pressurized environment (i.e., inside a hyperbaric chamber), the plasma will also carry oxygen, thus enabling the blood to deliver additional oxygen to the damaged
tissues. Theoretically, greater amounts of oxygen help the body to heal faster.

3. Differences between Mild HBOT and HBOT

When one thinks about HBOT they routinely think of the large, stationary units commonly found in the hospital setting. The pressure in these standard units can increase to 2 or 3 atmosphere absolute (ATA). As a point of reference, 1 ATA is equivalent to ambient pressure at sea level or 760 mmHg. Therefore, a standard hyperbaric chamber treatment of “2 or 3 ATA is equivalent to being 45 to 66 feet under the surface of the ocean.” A mild HBOT unit differs from a standard unit in a few key ways. One, they are cheaper and portable. As they expand, the air pressure causes them to inflate to their full size, although they typically have some sort of frame that keeps them open making getting in and out easier. Two, mild HBOT units do not have the ability to obtain the same levels of pressure as a standard unit does. Mild HBOT units are limited to 1.3 ATA.

The significance of treatment pressure is a matter of much debate. For example, in a study by Miller et al.\textsuperscript{4} wherein soldiers were treated for post-concussions symptoms, no difference was noted between those whose treatment parameters were 1.5 ATA with 100% oxygen and the sham treatment of 1.2 ATA with no oxygen supplementation. Both groups did differ from a third group who received no treatment, leading the authors to conclude that all improvements were placebo related. They did not speak to the fact that perhaps the sham treatment parameters could also have a therapeutic effect. If so, this could validate the use of mild HBOT.

The opposite was observed in a more recent systematic review wherein HBOT was used to treat acute severe traumatic brain injury.\textsuperscript{5} Daly et al. concluded that HBOT significantly improves physiological measures while not causing cerebral or pulmonary toxicity. End result, these physiological measures can potentially improve clinical outcomes.\textsuperscript{5} This systematic review was key to
funding a current National Institutes of Health clinical trial wherein the scientists hope to determine the optimal combination of HBOT parameters that are most likely to demonstrate improvement in the outcome of severe traumatic brain injury patients. Of note, all studies reviewed used HBOT parameters of 1.5 ATA or above, which are the same settings used in the clinical trial. The question remains whether mild HBOT would have similar effects.

With respect to recovery, results are similar. It is difficult to find two studies that are similar enough to make a meaningful comparison, let alone find ones completed in the last decade. Babul et al. and Webster et al. both used a sham treatment of HBOT (1.2 ATA and 1.3 ATA respectively) as control for either a 2.0 ATA or 2.5 ATA treatment of delayed onset muscle soreness (DOMS). The DOMS was induced using an eccentric load protocol. Although some of the variables measured differed, the main outcome did not – HBOT did not have an effect on DOMS. In two other studies of clinically induced DOMS, both Germain et al. and Harrison et al. likewise used 2.5 ATA for their treatment, but compared this to no treatment at all as their control. Although both research teams observed changes in the markers they measured, neither group observed changes that were significantly different from the control group.

If treatment happens before DOMS sets in, results may be different. In a paper by Ishii et al. the statement is made that HBOT “treatment has effectively increased recovery from fatigue.” They elaborate by stating that “this was clearly seen at the Nagano Winter Olympics, where sports players experiencing fatigue were successfully treated, enabling the players to continue performing in the games.” Shimoda et al. elaborate on these claims though, comparing the maximal unilateral isometric plantar flexions of individuals treated with either 2.5 ATA or 1.2 ATA HBOT. Those treated with 2.5 ATA HBOT saw a smaller decrease in force production, leading to the conclusion that HBOT helps to maintain force production.
4. Conclusions

The data supporting the practice of HBOT usage is varied depending on the condition being studied. As HBOT increases in prominence, particularly the use of mild HBOT chambers by lay people, efforts need to be made to determine the evidence of usage in these settings. The data to substantiate or refute the use of mild HBOT exists in the various settings it is used, but needs to be collected, analyzed, and discussed, particularly as it relates to the same data from a standard unit.
References


