

Proceedings of the Fifteenth International Congress on Hyperbaric Medicine

Organized by CRIS-UTH
Editor: Jordi Desola, MD, PhD

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Papers on all fields of Diving and Hyperbaric Medicine !

Hyperbaric Oxygen Therapy And The Cochrane Collaboration – Hope Or Despair?

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INTRODUCTION:

Hyperbaric oxygen therapy (HBO) has been defined in a surprising number of ways. For the purposes of this address, I shall define HBO as:

“the therapeutic administration of oxygen at pressures greater than one atmosphere absolute (1 ATA)”.

Among physicians trained in the western tradition, HBO is a relatively poorly understood therapeutic modality. Often consigned to a basket including alternative therapies with no apparent physiologic basis, HBO remains on the fringe of accepted medical practice despite 50 years of clinical experience. In Australia and New Zealand there are only 12 comprehensive hyperbaric facilities located within hospitals, although there are a number of small, free-standing facilities that tend to concentrate on a narrow spectrum of disease.

One recurrent criticism that has been made of this field is that treatment is based on little or no good clinical evidence. The recently improved awareness of the importance of evidence for all medical interventions has highlighted this perception. Hyperbaric practitioners are divided about the appropriate response to this criticism. While some confine themselves to clinical practice and the generation of informal clinical evidence in the form of case series and individual reports, others have attempted to prosecute more formal, high level clinical studies, while others still have stepped up the efforts to understand the basic mechanisms involved.

It has been similarly difficult to justify our choice of treatment tables and duration. Hyperbaric physicians regard oxygen as a drug, much like any other. It follows then, that for any particular condition there should exist a sub-therapeutic dose, a therapeutic dose range and a toxic dose. Treatment tables should be designed to reflect this reality. Total oxygen doses to produce these effects are likely to vary between individuals, but it is equally likely that there is a target tissue PO₂ that will produce a predictable effect – analogous to a target concentration of a pharmaceutical agent. For each putative condition therefore, it should be possible to devise a regimen that achieves the most efficacious dose with an acceptable safety profile. In HBO, of course, this dose is described in a pressure and time profile for each individual exposure, as well as a total dose over time.

New Strategies for Cancer Treatments Using Hyperbaric Oxygenation: Radiotherapy, Chemotherapy and Treatment for Brain Radionecrosis

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SUMMARY

We have applied hyperbaric oxygen (HBO) therapy for the treatment of cancer, especially for malignant brain tumors. Based on the result of persistence of high oxygen pressure in tissues after HBO therapy, we have performed HBO exposure preceding radiotherapy. Recently a few clinical reports have shown prolonged survivals of patients with high-grade gliomas, despite small non-randomized series. We confirmed that this new approach improved radiation response in a tumor model with hypoxic cells. In addition, some types of chemotherapeutic agents showed enhancement by HBO in experimental studies. A recent clinical trial shows that HBO enhances the therapeutic effects of carboplatin, a platinum complex, for the patients with recurrent high-grade gliomas. In the treatment of radiation-induced brain injury after radiosurgery, some investigators note that HBO therapy is effective for the treatment of this condition. Moreover, our preliminary clinical trial suggests that HBO therapy after radiosurgery protects the progression of radiation injury. HBO therapy is becoming an important strategy in the field of oncology.

PREFACE

Hyperbaric oxygen (HBO) therapy, which is mainly used for the treatments of hypoxic tissue damage, has also therapeutic effects of enhancement of tissue damage. One of them is cancer treatment such as radiotherapy and/or chemotherapy. The presence of hypoxic tumor cells is widely regarded as one of the major reasons for failure to control the malignant tumors with radiotherapy and/or chemotherapy [1,2]. To control the hypoxic cells is the most important approach to cancer treatments. Since HBO therapy improves oxygen supply to hypoxic cells, a pilot study of radiotherapy combined with HBO was published in 1950's [3]. Then some clinical trials were performed, and this adjunctive treatment was effective for a few types of cancer. However, the previous combined method, radiotherapy during HBO exposure, was hazardous to patients and was a complex technique, and as a result HBO therapy has not been routinely adopted with radiotherapy to treat cancers [3].

Neuroprotective Anti-Apoptosis Effect of Hyperbaric Oxygen Treatment in Secondary Brain Damage

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Traumatic brain injury (TBI) is a major health problem in all developed countries, with cerebral contusions been the most common consequence of TBI. Recent evidence has clearly demonstrated, that TBI, may give rise to the development of the delayed secondary brain damage and that the apoptotic cell death is involved in the secondary brain damage.

The goal of the present study is to evaluate the expression of apoptosis-related proteins of bcl-2 family (bcl-2, bcl-xL and bax) in the traumatic penumbra area in correlation with the extent of apoptosis in the rat model of dynamic cortical deformation (DCD), treated by HBOT. four groups of 5 Sprague-Dawley rats each were included in this study. The study protocol was as follows: group 1-DCD, group 2-DCD and HBOT; group 3-DCD and perioperative hypoxia ; group 4-DCD, perioperative hypoxia and HBOT. The bcl-2 family of proto-oncogenes was revealed by Immunohistochemical staining for bcl-2, bcl-xL and bax. The expression of bcl-2 in the penumbra area was lower in the animals, which underwent hypoxemia before the treatment, than in non-hypoxemic rats. The decrease in the expression of bcl-2 includes both the intensity of staining and its extent (the area). After the HBOT we observed statistically significant increase in the intensity and the extent of bcl-2 expression in both groups of animals (hypoxemic and non-hypoxemic) with hypoxemic animals showing still lower expression, but the difference was not significant.

The changes in the expression of bcl-xL were generally parallel to those of bcl-2, but differences between the groups were not statistically significant.

Bax protein expression, increase insignificantly after posttraumatic hypoxemia. After the HBOT there was some decrease in bax staining intensity and extent, but the measurement revealed marked variability of staining pattern and the differences between the groups were statistically significant ($p > 0.1$).

Our results provide more evidence of the importance of apoptotic mechanisms in delayed cell death in traumatic penumbra area of brain injury. We also demonstrate the

Neuronal and Endothelial Nitric Oxide are involved in Hyperbaric Pulmonary Oxygen Toxicity

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BACKGROUND

Hyperbaric oxygen (HBO₂) produces O₂ toxicity involving primarily two organs: the brain and the lungs. CNS O₂ toxicity is manifested by the appearance of electrical discharges on EEG, tremor, jerks and tonic-clonic convulsions [1]. The lung's susceptibility to O₂ toxicity differs from the brain's not only in dose threshold but in the manner of damage. At 0.6 to 1 ATA, the lung's responses are characterized by pulmonary inflammation, which has been attributed to PO₂-dependent reactive oxygen and nitrogen species (ROS and RNS) generation that overwhelms biological anti-oxidant defenses and injures the lung. Prolonged exposure to 100% O₂ damages lung epithelium and capillary endothelium diffusely and causes extensive inflammatory cell infiltration and interstitial and intra-alveolar edema [2]. The adult rat, exposed continuously to 100% O₂, dies of respiratory failure after about three days [3]. HBO₂, however, accelerates pulmonary O₂ toxicity and greatly shortens this survival interval, to just about six hours, at 3 ATA [4].

The mechanisms that cause such dramatic shortening of survival in hyperbaric pulmonary O₂ toxicity are poorly understood. In a preliminary study we have shown that HBO₂-induced lung injury is attenuated after non-specific inhibition of both neuronal and endothelial NO synthases (NOS) with L-NAME [5]. The current study was designed to examine specific roles for neuronal or endothelial NOS in the development of pulmonary HBO₂ toxicity.

METHODS

Adult wild type (WT) mice and mice deficient in extracellular SOD (EC-SOD^{-/-}), glutathione peroxidase (GPx^{-/-}), neuronal NOS (nNOS^{-/-}), endothelial NOS (eNOS^{-/-}) and inducible NOS (iNOS^{-/-}) were exposed to HBO₂ at 2.5 ATA for 6 hours. Immediately after exposure, bronchoalveolar lavage (BAL) was performed to determine total cell count (macrophages, neutrophils, lymphocytes), lactate dehydrogenase (LDH) activity and total protein content in BAL fluid as indicators of lung injury and alveolar-capillary permeability.

Full page colour reproduction of Posters like they were exhibited in the Conference

The use of a deep stop during decompression of Agar gel plates influences the number, diameter and total gas volume of post decompression gas bubbles.

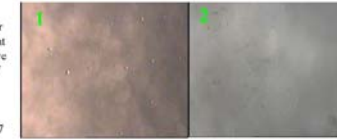
Alessandro Marroni^{1,2}, Peter B. Bennett^{4,5}, Frans J. Cronje^{6,7}, Costantino Balestra^{1,3}, Pasquale Longobardi⁸, Ramiro Coll-Corleto⁹, Peter Gernsgore¹⁰, Massimo Fleri¹, Maurizio DiGennaro¹

Introduction

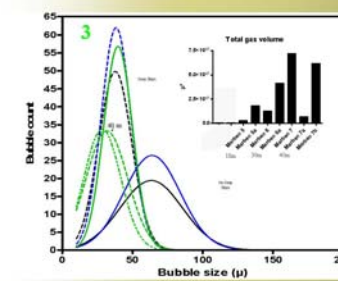
Previous research by this group has suggested that deep stops reduce the appearance of precordial Doppler detectable bubbles in humans ascending from 24 MSW (82 fsw). However it is not known whether this is the result of true disappearance of decompression bubbles or a reduction of bubble diameter below the threshold of Doppler detection.

METHODS

This study examined the effect of different decompression stops on the bubble production in agar gel plates. The plates were pretreated with a surfactant agent (Sodium Dodecyl Sulfate) and exposed to 6 dive profiles: three simulated dives to 18, 30 and 40 MSW (60, 100 and 130 fsw) for 59, 24 and 9 minutes respectively, with or without an empirical 1 minute "deep stop" at half-the-depth using a 10 MSW/min ascent rate and a 3 minute shallow stop at 5 MSW (17 fsw) on all dives. Post decompression bubbles were counted by a validated microscopic scoring system (Acumax Microscope & Software -- Copyright ForBoGel Centro Iperbarico Ravenna / Chimica Ravenna). Assessments included bubble diameter, number and total gas volume load within the agar plate.



Sample images of the Agar plates
1: 30 m « dive » without Deep Stop
2: 30 m « dive » with Deep Stop



Results

The difference in bubble diameter, number and total gas volume load was different between dives with and without the "deep stop" according to a bimodal distribution (see fig. 3). The greatest number of bubbles were observed in profiles that included a "deep stop", but the bubble volumes were smaller than for those dives without a "deep stop," although the total gas volume in the agar plates was increased. For dives without a "deep stop" the number of bubbles was less, but the bubble volumes were larger. The 40 m dive showed a different pattern, whereby both the number and the volume of bubbles decreased with the Deep Stop.

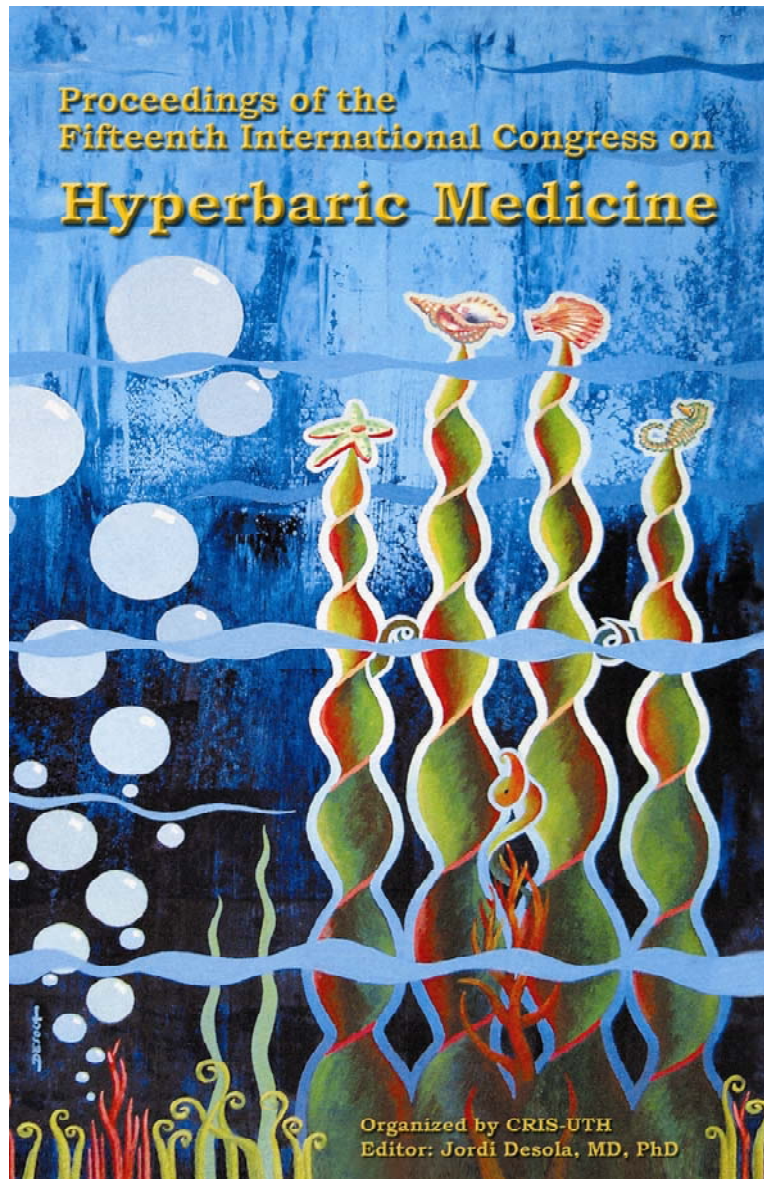
Conclusion

It is evident that the introduction of a "deep stop" significantly affects the size and number of in-vitro decompression bubbles. Even when the number of bubbles were increased, the actual bubble volumes were significantly reduced and the total off-gassing was increased by the introduction of a 1-minute deep stop. This introduces two interrelated factors in need of further elaboration: assuming this diffusion-limited in-vitro model actually represents in-vivo bubble production -- (1) a one minute "deep stop" may reduce gas bubble volumes in favor of larger numbers of bubbles which may significantly affect Doppler detection and the biological significance of these bubbles respectively; (2) one minute "deep stop" may not be sufficient to avoid significant bubble formation. This study prompts further investigation of ascent-stop combinations in pursuit of better "economy of decompression".

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*Marroni A, Bennett PB, Cronje FJ, Coll-Corleto R, Gernsgore P, Fleri M, Balestra C & Balestra C (2004). A deep stop during decompression from 82 fsw (24 m) significantly reduces bubble and total tissue gas tensions. *Undersea Hyperbaric Med* 31, 220-224.
*Marroni A, DiGennaro M (2007). A deep stop during decompression from 82 fsw (24m) significantly reduces bubble and total tissue gas tensions. *Undersea Hyperbaric Med* 34, 47-48, author reply 49-52.

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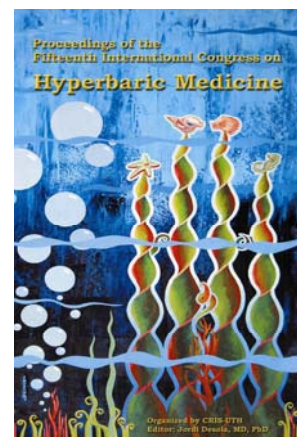
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