CONTENTS

Papers from the five continents !

2.- HOSPITAL BASED CENTRES OF HYPERBARIC MEDICINE

3.- DIVING RESEARCH

4.- BASIC RESEARCH - OXYGEN TOXICITY

5.- HYPERBARIC OXYGEN THERAPY - I

6.- DIVING PHYSIOLOGY

7.- PATHOPHYSIOLOGY OF DYSBARIC INJURIES

8.- HYPERBARIC OXYGEN THERAPY - II

9.- HYPERBARIC OXYGEN THERAPY - III

The Effects of Hyperbaric Oxygen Treatment on the Experimental Atherosclerosis **269**
Tomislav Jovanovic, P. Brkic, Ana Mitrovic, Olga Zaric (Yugoslavia)

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

Bennett MH.

Senior Lecturer in Anaesthesia and Hyperbaric Medicine, Faculty of Medicine, University of NSW and Senior Staff Specialist, Department of Diving and Hyperbaric Medicine, Prince of Wales Hospital, Sydney.

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 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 PO_2 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.

SAMPLE

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

Kiyotaka Kohshi, MD

Division of Hyperbaric Medicine & Department of Neurosurgery, University Hospital of Occupational and Environmental Health, Japan

1-1 Iseigaoka, Yahatanishi-ku, Kitakyushu 807-8555, Japan

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 Tretment in Secondary Brain Damege

Eilam Palzur [a] M.Sc., Eugene Vlodavsky [b] M.D., Ran Arieli [c] Ph.D., Jean F. Soustiel [a] M.D.

([a]) Division of Neurosurgery and Acute Brain Injury Research Laboratory and Department of Pathology.
([b]) Rambam Medical Center, Faculty of Medicine, The Technion, Haifa, Israel.
([c]) Israel Naval Medical Institute[3], Israel Defense Forces Medical Corps, Haifa, Israel.

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

Atochin D-N[a], Demchenko I-T[b], Huang P-L[a],Piantadosi C-A[b]

([a]) Cardiovascular Research Center, Massachusetts General Hospital, Boston, MA 02129.
([b]) Duke University Center for Hyperbaric Medicine and Environmental Physiology, Durham, NC 27710

BACKGROUND

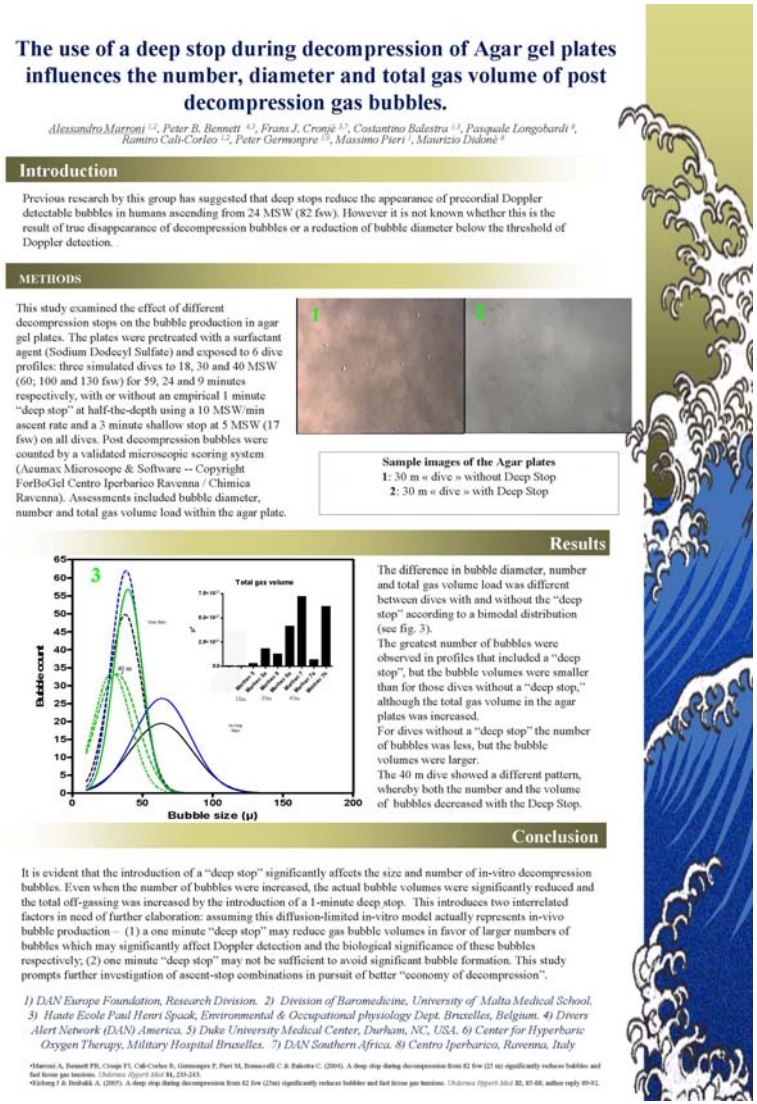
Hyperbaric oxygen (HBO_2) produces O_2 toxicity involving primarily two organs: the brain and the lungs. CNS O_2 toxicity is manifested by the appearance of electrical discharges on EEG, tremor, jerks and tonic-clonic convulsions [1]. The lung's susceptibility to O_2 toxicity differs from the brain's 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 PO_2-dependent reactive oxygen and nitrogen species (ROS and RNS) generation that overwhelms biological anti-oxidant defenses and injures the lung. Prolonged exposure to 100% O_2 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% O_2, dies of respiratory failure after about three days [3]. HBO_2, however, accelerates pulmonary O_2 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 O_2 toxicity are poorly understood. In a preliminary study we have shown that HBO_2-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 HBO_2 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 HBO_2 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. Cronjé [3,7], Costantino Balestra [3,6], Pasquale Longobardi [8], Ramiro Cali-Corleo [1,2], Peter Germonpre [3,6], Massimo Pieri [1], Maurizio Didone [8]

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.

Bubble count
Bubble size (µ)
Total gas volume

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".

1) DAN Europe Foundation, Research Division. 2) Division of Baromedicine, University of Malta Medical School. 3) Haute Ecole Paul Henri Spaak, Environmental & Occupational physiology Dept. Bruxelles, Belgium. 4) Divers Alert Network (DAN) America. 5) Duke University Medical Center, Durham, NC, USA. 6) Center for Hyperbaric Oxygen Therapy, Military Hospital Bruxelles. 7) DAN Southern Africa. 8) Centro Iperbarico, Ravenna, Italy

Hyperbaric Medicine Practice in China
Present and Future Prospective and Development

Gao Chunjin

Chairman of Chinese Association of Hyperbaric Oxygen Medicine

HBO medicine research has been developing rapidly in China, though it started later than in Europe and America. In 1964, Professor Li Wenren built China's first medical hyperbaric chamber in Fuzhou, Fu Jian Province, and practiced open-heart-surgery successfully in it.

October 1992 marked the start of a new era of HBO medicine in China, when Chinese Association of Hyperbaric Oxygen Medicine (CAHOM) was established in Lanzhou City. Professor Li Wenren was the first Chairman of the association, who held the post from 1992 to 1995. In 1993, the 11th International Congress on Hyperbaric Medicine was held in Fuzhou, Fujian Province, and Professor Li Wenren was the executive chairman of the meeting.

Since 2001, Professor Gao Chunjin has been the fourth Chairman of the association. In 2001, CAHOM won the sponsorship of the 16th International Conference on Diving and Hyperbaric Medicine.

At present, CAHOM has established branches in every province in China. Many medical universities in China now offer subjects on HBO medicine to produce HBO talents with high academic degree. In 1992, an academic journal on HBO---*Journal of Hyperbaric Oxygen Medicine*---was firstly published in China. In 2001, the journal merged with *Chinese Journal of Nautical Medicine* and was renamed "*Chinese Journal of Nautical Medicine and Hyperbaric Medicine*". Meanwhile, a professional website on HBO medicine information was set up, which greatly promoted the informatization of HBO medicine in China. www.chinahbo.org.cn

Until now, CAHOM has organized 13 annual academic meetings on HBO medicine. On these meetings, HBO professionals from around the country gathered together to exchange their ideas on clinical experience, scientific research, new development in HBO theory and technology.

Since 1995, CAHOM has sponsored 14 training programs on new development of HBO medicine, 43 training programs for maintenance and operation personnel of HBO chamber and 70 training programs for medical staff of HBO medicine. So far, more than 10000 HBO medical professionals and technicians have attended these training programs. With more than 40 years of development, presently China has a total of 3892 HBO Chambers and more than 21000 HBO professionals in HBO departments around the country.

China's clinical and experimental study in HBO medicine has gained certain position in world HBO medical community. Rough statistics show that many diseases involving most clinical subjects have been treated with HBO therapy (HBO) in China.

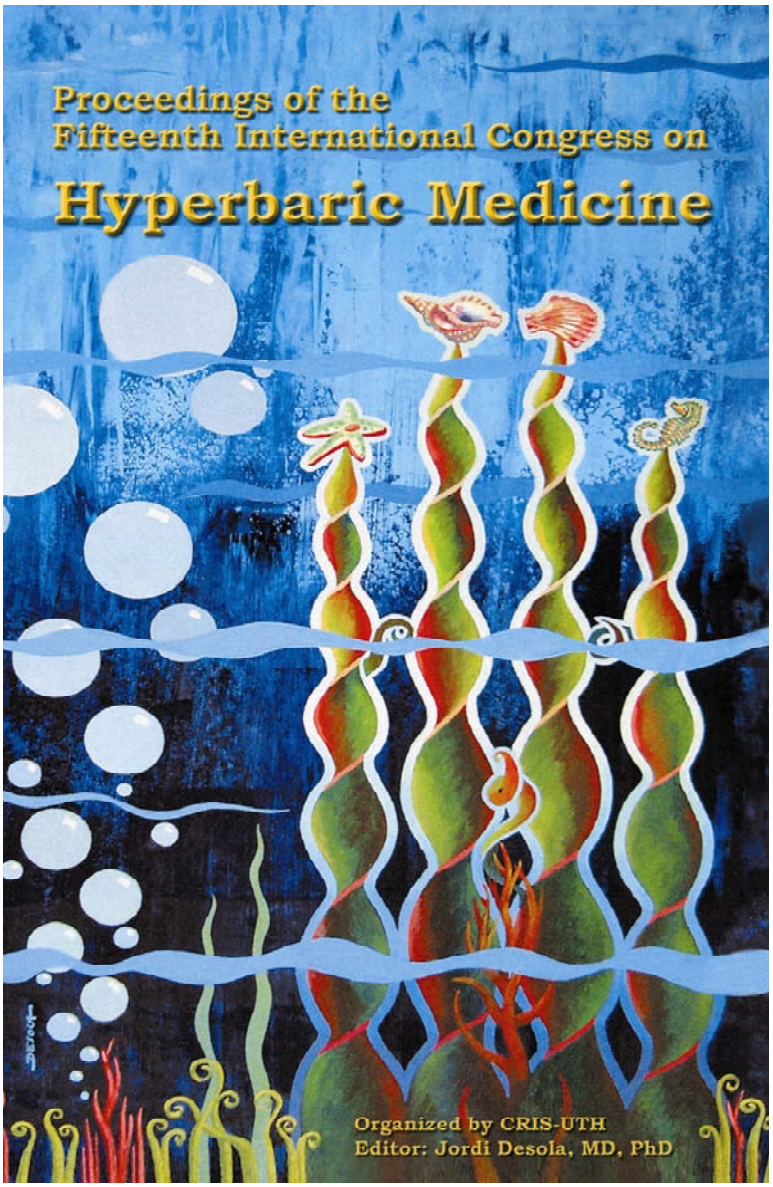
Indication of HBO Therapy

1. Acute carbon monoxide poisoning
2. Delayed encephalopathy after acute CO poisoning
3. Harmful gas poisoning
4. Head injury
5. Ischemic cerebrovascular disease
6. Sudden deafness
7. Trauma
8. Burn
9. Avascular necrosis of the femoral head
10. Diabetic foot
11. Gas gangrene tetanus and other anaerobic infection
12. Decompression sickness
13. Gas embolism
14. Osteomyelitis
15. Radiation injury
16. Aseptic osteonecrosis
17. Anesthesia accident CPR
18. Phlebitis
19. Tibia Osteonecrosis

Experimental Study

Experimental research of HBO medicine has also developed rapidly in China. Many hospitals and institutes applied molecular biological, immunological and other techniques

Proceedings of the Barcelona-2005 ICHM Already Available !



A 300 pages book with more than 50 colour charts.
Full text of the Invited lectures, Oral communications, Posters,
and full page colour reproduction of posters like they were exhibited in the Conference.
Statistics of delegates, attendants, countries, committees, exhibitors, and sponsors.
Download a sample with the list of contents and some examples from :
<http://www.sub-help.com> or ask it at <sub-help@comb.es>

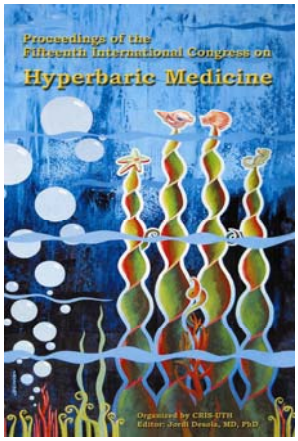
Order it now !

Retail price : 60 € (Euro)
Registered attendants will benefit from a 10% discount
See other discounts in the order form

SUB-HELP

FAX: +34-934-503-736 - E-Mail: sub-help@comb.es
Ap.Correos 4033 - 08080 Barcelona - NIF: B60418282

Proceedings of the Fifteenth International Congress on Hyperbaric Medicine Barcelona 2005



ORDER FORM

Retail price : 60 € (Euro)

Registered attendants will benefit from a 10% Discount.

Name of the attendant exactly like he/she was registered in the Conference :
……..……..

Shipping and Handling :

Spain : 5 €
Europe :15 €
Rest of the world : 24 €
(*Cost per unit. Especial conditions for multiple orders*)

Other Discounts :

Number of units	% Discount	Price per unit (Euro)
1-4	0	60
5-9	5	57
10-24	10	54
25-49	15	51
50-75	20	48
75-99	25	45
more than > 100	30	42
Distributors, book-shops		Consult

Family name ..... Name: .....
Company : ..... N.I.F.: .....
Invoicing Address: ……..... .....
ZIP : ..... City: ..... Country .....
Phone: ..... FAX: ..... E-mail: .....
Sending Addres (if different from above): ……..
ZIP: ..... City: ..... Country ….....

No. of units ……..
Discount ……..... %
Price per unit ……..... €
Subtotal ……..... €
Shipping and handling ……..... €

TOTAL DUE ……..... €

PAYMENT:

☐ Banc Transfer to SUB-HELP
Caja de Ahorros del Mediterráneo
IBAN: ES73-2090-6871-86-0041615871
SWIFT: CAAMES2A

☐ VISA-Mastercard

☐ American Express

Credit card number : ……

Expiry date : ……. / …….

Name of the card holder : ……

Signature: ……

Send this order to :

SUB-HELP - E-Mail: sub-help@comb.es - FAX: +34-934-503-736 - Apartado de correos 4033 - E-08080 BARCELONA