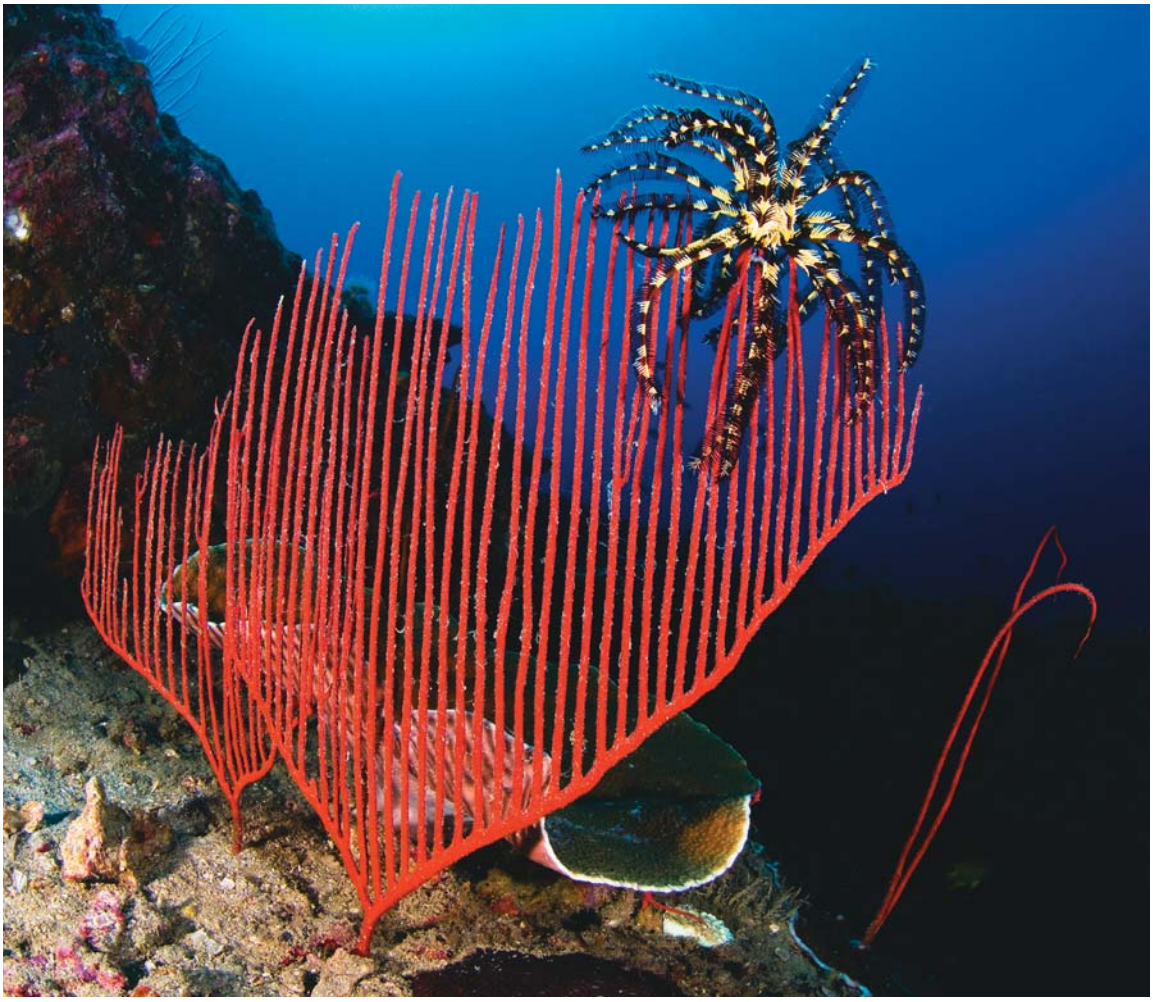
Diving and Hyperbaric Medicine

The Journal of the South Pacific Underwater Medicine Society (Incorporated in Victoria) A0020660B and the European Underwater and Baromedical Society

SPUMS

Volume 40 No. 2 June 2010

EUBS



Hyperbaric oxygen for noise-induced hearing loss

The two faces of Eve: inert gas narcosis and anaesthesia

'Mild hyperbaric therapy' is not the same as HBOT

Tasmanian abalone diving industry

30 years ago: the 'Atlantis' dives

ISSN 1833 - 3516
ABN 29 299 823 713

Print Post Approved
PP 331758/0015

PURPOSES OF THE SOCIETIES

To promote and facilitate the study of all aspects of underwater and hyperbaric medicine
To provide information on underwater and hyperbaric medicine
To publish a journal and to convene members of each Society annually at a scientific conference

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The Journal of SPUMS and EUBS
<www.dhmjournal.com>

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The Editor's offering

There has been controversy for over a decade as to whether hyperbaric oxygen therapy (HBOT) has a role in the treatment of sudden hearing loss, whether of acoustic or idiopathic origin. The paper by Lafère et al, from the Belgian military, sheds new light on this where acoustic trauma from gunfire is concerned.[1] Whilst this is not a randomised controlled trial (RCT), it prospectively compares current best practice with and without HBOT in matched groups and shows a significant hearing recovery benefit from HBOT when combined with steroids and piracetam compared to medical management alone. The writer understands that an Australian multicentre RCT of HBOT in idiopathic hearing loss is about to get under way.

Like Smith and Spiess in their review article,[2] many anaesthetists, from their training, will probably have regarded inert gas narcosis and gaseous anaesthesia as a continuum. There are many theories put forward to explain the mechanisms of obtundation caused by such a diverse groups of molecules, ranging from the noble gases, through the ethers, halogenated and non-halogenated hydrocarbons and modern halogenated ethers. The authors provide a concise overview of these theories, from which they suggest a unifying set of mechanisms for these effects.

The Australia and New Zealand Hyperbaric Medicine Group (ANZHMG) is a standing sub-committee of the South Pacific Underwater Medicine Society and represents the specialist group of medically qualified providers of HBOT in Australasia. Because at times their activities are confused with those of 'alternative' practitioners, the group has prepared a statement on 'mild hyperbaric therapy' in order to allow third parties to accurately identify and characterise the form of therapy being offered to them. That statement is published in this issue. The ANZHMG is not aware of any reliable clinical evidence for therapeutic benefit from 'mild hyperbaric therapy' as defined here, and does not recommend the use of this modality for any medical purpose.

From time to time, we will publish historical articles from the SPUMS Journal archives, hoping to match these with particular themes in the same issue or with current events. The article by John Miller on the Atlantis I and II dives at Duke University, reprinted here with a brief introduction from Richard Moon, fits for several reasons. John was the Guest Speaker at the 1980 Joint Annual Scientific Meeting of SPUMS and the Singapore Navy, whilst this year, 30 years on, SPUMS held only its second joint meeting, this time with the recently-formed Asian Hyperbaric and Diving Medical Association. Whilst not the deepest chamber dive on record (the later Atlantis III at 686 msw and the Comex Hydra 10 with excursion to 701 msw dives were deeper), at the time it was especially exciting to hear about the Atlantis dives only a few months after the completion of Atlantis II from a physician who was a part of the Duke team. These were ground-breaking dives into 'inner space' and very much in the same vein as one of the themes of this year's meeting, with Michael Gernhardt's exciting presentations on physiological aspects of space research. At a personal level, 1980 was my first SPUMS ASM, and I had the pleasure, during the meeting, of sharing a room with John, a colleague whom I had first met in the 1960s at the Royal Naval Physiological Laboratory in Alverstoke, before he moved to the USA, and who later was one of my senior residents in Seattle. It is fitting that Richard Moon has written a brief introduction. For those readers who would like to know more about this era of human deep diving research, the chapter by Peter Bennett and Jean Claude Rostain in Bennett and Elliott's textbook is highly recommended.[3]

Two important changes have occurred recently with the Journal. Firstly we have a new e-mail address, <editor@dhmjournal.com>, and secondly the Journal has its own website, <www.dhmjournal.com>. The old e-mail address, <spumsj@cdhb.govt.nz> will remain active until the end of 2011. The web page is still in its early days of development and we intend to grow this gradually as time permits. We would value any input from EUBS and SPUMS members as to what they would like to see on this site, so please forward your suggestions. Journal issues will not be available here, but rather in the member-access sections of the society websites. The cost of producing a quality journal for society members continues to rise and both EUBS and SPUMS have struggled with this over the past year. As a result, the members attending the Annual General Meeting of SPUMS at Redang voted to increase all types of SPUMS memberships by AUS$20 in 2011. EUBS may need to consider the same. At the same time, both executive committees are looking at ways of expanding the circulation of the Journal without compromising society membership.

References

1 Lafère P, Vanhoutte D, Germonpré P. Hyperbaric oxygen therapy for acute noise-induced hearing loss: evaluation of different treatment regimens. *Diving and Hyperbaric Medicine*. 2010;40(2):63-7.

2 Smith CR, Spiess BD. The two faces of Eve: gaseous anaesthesia and inert gas narcosis. *Diving and Hyperbaric Medicine*. 2010;40(2):68-77.

3 Bennett PB, Rostain C. The high pressure nervous syndrome. In: Brubakk AO, Neuman TS, editors. *Bennett and Elliott's physiology and medicine of diving*, 5th edition. Edinburgh: Saunders; 2003. p. 323-57.

Michael Davis

The front-page photo taken by Dr Simon Mitchell at Redang Island, Federation of Malaysia, is of a red whip (harp) coral, *Ctenocella pectinata*, and the feather star is probably *Stephanometra sp*. (Nikon D300 with Nikkor 10.5 mm fisheye lens in a Nexus housing. 1/60th and f22. ASA 400. Twin Inon Z240 strobes on half power)

The President's page

Peter Germonpré
President, EUBS

Dear friends,

Generally, I am a peaceful and diplomatic person, despite my military occupation. This does not mean that, internally, I never feel any rage or indignation. This happened to me recently when our Editor-in-Chief, Mike Davis, advised the EUBS and SPUMS Executives that the National Library of Medicine Technical Review Committee (who decide whether a journal will be indexed in the Medline database) had judged the quality of this journal insufficient to warrant indexing. I am sure Mike will have elaborated on this matter in his editorial message. Those of us who have been working hard to maintain a top-quality journal and who have prepared and defended its application know all too well that some injustice has been committed here. You, members of our societies and readers of this journal, hopefully agree with me.

Jointly to our next EUBS conference (Istanbul, 14–18 September), the European Committee for Hyperbaric Medicine is planning a workshop on controversial indications for hyperbaric oxygen therapy ('off-label' use). Whilst the exact topics need to be refined (keep an eye on the EUBS and ECHM websites), there is a clear need to organise such a workshop. All of us working in clinical hyperbaric oxygen therapy (HBOT) regularly receive urgent requests from patients, parents and caregivers to treat conditions for which evidence is cruelly lacking or controversial. To name a few: cerebral palsy, autism, stroke, cerebral ischemia, sports injuries, aseptic bone necrosis; by simply 'googling', one can easily see why patients and caregivers assume that there is a place for HBOT in the treatment of their disease or condition. Alas, on the world wide web, it is difficult to distinguish commerce from science, money-making schemes from genuine altruism, and good scientific reports from over-optimistic 'jumping to conclusions' papers. The internet will never replace a good, peer-reviewed independent journal!

What strikes me personally is the ardour with which proponents and opponents face one another, as if one holds the truth and the other is ignorant, unwilling to yield or plain stupid!

Again, the truth is apparently 'flexible' or subject to interpretation. Workshops and consensus conferences should indeed provide an objective way to identify the strengths and weaknesses of currently available research, provide directions for new research, and, most importantly, provide a 'guideline' or at least a motivated point of view as to the acceptance of HBOT treatment for specific conditions.

Anyone wanting to step outside these chalked borders will have to do so in full conscience of the lack of scientific back-up. This does not mean it should be 'forbidden' to treat patients who do not correspond exactly to the accepted indications, only that, when doing so, one should be very cautious and, as much as possible, document the case and the effect of treatment. Whether public or private funds may be requested for such treatments is a question of economics as well as ethics: it is well known that patients are willing to pay huge amounts of money for even the tiniest spark of hope. We are, as a whole, responsible for preventing or stopping any abuse.

Truth does not exist. There is only reason.

I spent three days with family and friends in the French region of Lorraine, known to most of you for its World War One battlefields around Verdun. It is now a beautiful and varied natural reserve and, every year, we spend a long weekend exploring woods, chalk hills, lakes and ponds for the tiny and not-so-tiny living things that make up Nature. Humbly I confess that even after 18 years, I am still largely ignorant of all the creatures that crawl, fly, buzz, and slither beneath or above the earth. I admire those who kneel down in the grass in front of what, at first, looks like a patch of shrub or weed, only to show me the beauty of wild orchid petals, of lizards, frogs, larvae, bugs; with admiration I listen to the sound of birds rarely seen but all the more heard – the songs and names of some of those birds I can already recognise, others still a mystery revealed again every year. Our group consists of individuals of all ages. What I enjoy most is the patience and enthusiasm with which knowledge is passed on from older generations to the new.

The art of observation and constant reference to high-quality field guides and books make these nature walks a lesson in scientific thinking. Is this not also one of our duties in medicine? – to scrutinise every aspect of what we do, even what seems 'evident' or what we have done for many years, to constantly adjust our reference, and patiently teach the younger ones how to observe and evaluate what we see.

Although modern technology allows us to see and appreciate better what is out there (I am talking binoculars, telescopes, digital cameras, etc), beauty is not only in what is aesthetically pleasing, but also in the 'ugly' or 'disgusting' things. Even dead animals are interesting: other creatures feed on them, need them to survive themselves, and so, even after death, organisms are teeming with life. There is no morality here, only the reality of life itself.

Nature knows no emotion.

I hope you enjoy this copy of DHM, and I look forward to seeing you in September!

Original article

Hyperbaric oxygen therapy for acute noise-induced hearing loss: evaluation of different treatment regimens

Pierre Lafère, David Vanhoutte and Peter Germonpré

Key words
Hearing, injury, hyperbaric oxygen therapy, outcome, research

Abstract
(Lafère P, Vanhoutte D, Germonpré P. Hyperbaric oxygen therapy for acute noise-induced hearing loss: evaluation of different treatment regimens. *Diving and Hyperbaric Medicine*. 2010;40(2):63-7.)
Introduction: Impulse noise from firearms is a common cause of acute acoustic trauma (AAT), which is characterized by high-frequency hearing loss and tinnitus. Various treatment modalities have been proposed, some combining medical treatment with hyperbaric oxygen (HBOT) in various ways. We have reviewed the therapeutic effect of primary protocols, with or without HBOT, used in our hospital.
Methods: Sixty-eight soldiers for all of whom pre-AAT audiometry tests were available, were treated with one of three different regimens. Group 1 received oral medication only. Group 2 received HBOT twice a day for 3 days then once a day (7 days), combined with intravenous medication (5 days) followed by oral treatment. Group 3 received HBOT once a day and oral medication for 10 days. Medical treatment consisted of methylprednisolone and piracetam in all groups. Control audiometry was performed after 10 days. Average Hearing Gain (AHG) and Average Residual Hearing Loss (ARHL) were calculated.
Results: The mean AHG in Group 1 was +5.58 ± 3.58 dB (mean ± SD); in Group 2 it was +20.62 ± 17.68 dB; and in Group 3 +17.0 ± 14.0 dB ($P = 0.001$, Kruskal-Wallis test). The mean ARHL without HBOT was -14.7 ± 8.27 dB (Group 1), and respectively -2.36 ± 10.69 dB (Group 2) and -5.0 ± 8.0 dB (Group 3) in the HBOT groups ($P = 0.001$, Kruskal-Wallis test).
Conclusion: These results indicate a significant benefit for the combination of HBOT and medical therapy over medical treatment alone. Which of the two HBOT regimens is the more effective, remains to be determined.

Introduction

Impulse noise from firearms is a common cause of acute acoustic trauma (AAT), which can cause rupture of cell membranes and decreased cochlear blood flow. This leads to decreased oxygen tension in the inner ear and reduction of oxygen-dependent cellular activities.[1] Symptoms are, typically, a high-frequency hearing loss ('noise-induced hearing loss', NIHL) and tinnitus. Different degrees of NIHL are described; in minor cases, the hearing loss is temporary (temporary threshold shift – TTS), recovering within 24 hours of the trauma.[2] In more severe cases, the hearing loss is permanent (permanent threshold shift – PTS).

The optimal treatment for NIHL has not been defined. In some animal models, it has been shown that hyperbaric oxygen treatment (HBOT), combined with corticosteroids, seems to improve the functional and morphological recovery.[3] Only the therapeutic regimens that include HBOT have shown a sustained therapeutic effect on noise-induced cochlear hypoxia.[4] Despite the first reports of HBOT as a treatment for NIHL being published 20 years ago, few human trials have been reported.[5,6]

It was proposed in 1995 that, in AAT, minimal therapy or waiting for spontaneous recovery is not the treatment of choice, because recovery is mostly incomplete, leaving a residual hearing loss and disabling tinnitus.[7] Analogy has been drawn with another acute hearing disorder, sudden idiopathic hearing loss. In human trials in those patients, 65% of poly-pragmatically treated patients demonstrated a hearing improvement independent of the drugs administered, whilst 61% of placebo-treated patients also demonstrated improvement.[8] Whereas the optimal medical therapy, if any, seemed unclear, a large review seemed to confirm that, in patients not responding to medical treatment, HBOT, even if given late, was still of benefit in about 45% of cases.[8]

There is a need to formally evaluate the therapeutic effect of HBOT in hearing loss, both idiopathic and noise-induced. Having at our disposal a unique cohort of patients for whom treatment is standardized and previous pure tone audiogram (PTA) is available, it was possible for us to analyse the results of the treatment protocols for AAT in a military hospital, including those using HBOT.

Subjects and methods

For this study, patient records from all cases with AAT treated between January 2006 and December 2008 at the Queen Astrid Military Hospital in Brussels, Belgium were retrieved. All 121 patients were professional soldiers

employed by the Belgian Armed Forces on active military duty. Their average age was 20.9 ± 4.6 years, average height 176.2 ± 13.4 cm and average weight 75.2 ± 6.9 kg. Ethical approval was obtained from the Military Hospital Bio-Ethical Committee. Each patient was informed and gave consent for use of their data in studies where only group data are reported. Clinical information for each case was loaded into a database that was stripped of individual identifiers.

All soldiers had suffered AAT during practice firing; all firing was done with similar ammunition (NATO [North Atlantic Treaty Organisation] 5.56 mm caliber) either with an FNC assault rifle (FN, Herstal, Belgium) or a Minimi light machine gun (FN, Herstal, Belgium). While appropriate noise protection is provided during military shooting exercises, this failed for a variety of reasons (improper placement of ear plugs, non-adapted ear-plug size, accidental removal or loss of ear plugs). The number of rounds shot before suffering AAT could not be determined with accuracy but, in most cases, AAT was provoked by one or two impulse noises (125 dB at 10 cm).[9] Once the soldiers were symptomatic they were immediately removed from duty and directed to sickbay where the first clinical evaluation took place. When NIHL was suspected, they were immediately transferred to the nearest hospital for audiometry and treatment. All patients thus received treatment within 48 hours after AAT and were formally tested at least 24 hours after the noise exposure. All patients were referred as soon as possible to the Military Hospital in Brussels; depending on logistic and practical considerations, some patients were treated locally with a standard medication schedule including corticosteroids and piracetam (see below).

For this study, only patients with hearing loss of at least -25 dB in at least one frequency (as compared to their baseline PTA) were included. Those with less severe hearing loss, or (to exclude those with only TTS) with improvement in hearing of more than 20 dB in any frequency in the first 24h after AAT, were excluded. Furthermore, patients with a history of previous AAT, even if fully recovered, were also excluded. This left 68 patients with unilateral NIHL, who were available for assessment of the effect of therapy with or without HBOT. They were divided into three groups:

Group 1, 'No HBOT'. Seventeen patients did not receive HBOT because emergency evacuation to the Military Hospital was not possible or practical. Medical treatment was started immediately in the patient's military unit, and consisted of a combination of oral corticosteroids (methylprednisolone) in a decreasing daily dosage (64 mg reducing to 8 mg over 10 days) and piracetam (2400 mg three times a day) for 10 days. This specific treatment regimen has been used in the Belgian Armed Forces since the 1970s; it has been enforced by a military directive in 1994 and has recently been endorsed by the Belgian Society of ENT Physicians after a consensus conference.[10]

Group 2, 'HBOT+IV'. For 32 patients, the delay in transfer to the Military Hospital was less than 36 hours (6 to 36 hours) and they were aggressively treated. They were given HBOT twice daily (pressure 253 kPa, 70 minutes of oxygen breathing) for three consecutive days, followed by once daily sessions for seven days. All patients received daily IV corticosteroids (methylprednisolone 125 mg decreasing to 40 mg) and IV piracetam (12 g over 15 minutes) for 5 days, followed by oral treatment for 5 days (methylprednisolone 32 mg decreasing to 40 mg and piracetam 2400 mg three times a day).

Group 3, 'HBOT+PO'. For 19 patients the delay of transfer was 36 hours or more (36 to 43 hours). They were given daily HBOT (pressure 253 kPa, 70 minutes of O_2 breathing) for 10 days, combined with oral treatment as for Group 1.

All patients were evaluated using PTA from 250 Hz to 8 kHz at the start of the treatment and after 10 days. These PTA curves were compared to the baseline PTA upon their enlistment into the Belgian Armed Forces. The average hearing loss (AHL) at frequencies of 2, 4 and 8 kHz (the only frequencies statistically different from the baseline PTA) was calculated with the enlistment PTA as a baseline. In order to compare the effect of the different treatment regimens, the average hearing gain (AHG) and the average residual hearing loss (ARHL) on these three frequencies were calculated in the same way as for the AHL. AHG was calculated with the initial loss as a basis, ARHL with the enlistment PTA as the baseline.

Results were analysed with GraphPad Prism software (version 5) on a PC, using the Kruskal-Wallis test (one-way ANOVA) and Dunn's multiple comparison tests (the groups failed to pass the Kolmgorov-Smirnov normality test, preventing assumption of a Gaussian distribution).

Figure 1
Comparison between the pure tone audiogram on enlistment into the army (baseline) and after acute acoustic trauma in the affected ear (all patients; * *P* < 0.0001)**

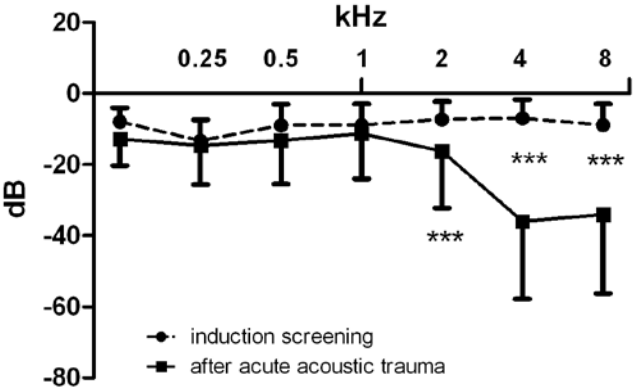


Figure 2
Average hearing loss at presentation for the three treatment groups (see text; $P = 0.66$)

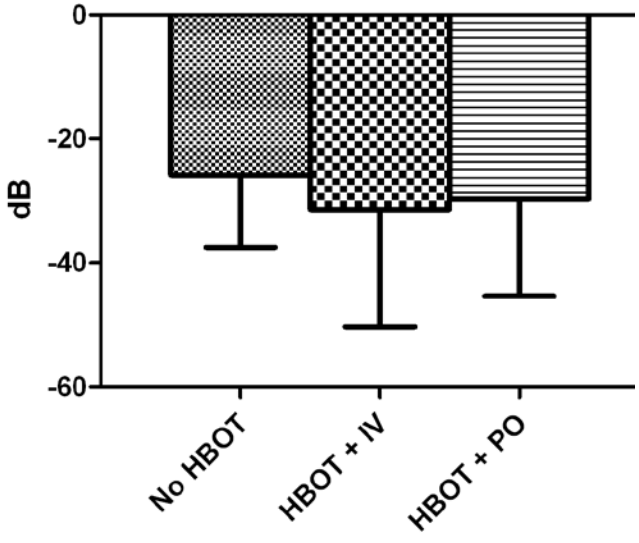
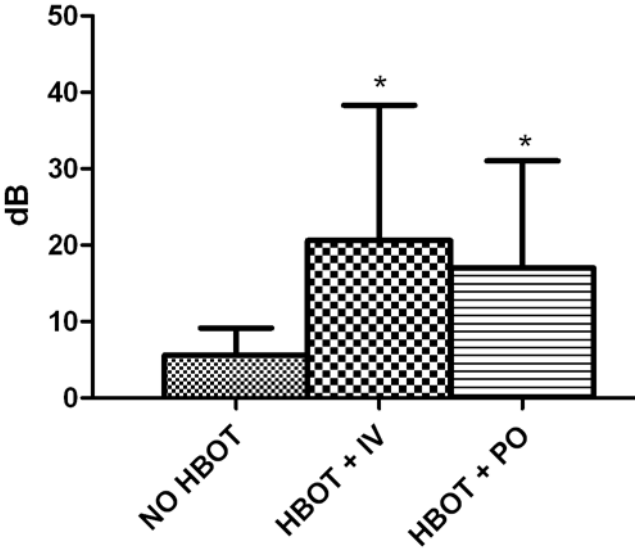


Figure 3
Average hearing gain in the three treatment groups (* $P < 0.05$)



Results

The three treatment groups were comparable as far as age, gender and weight were concerned.

Figure 1 shows the averaged pure tone audiograms of the injured ears compared to the induction PTA for the same ear. These confirm that the primary damage following acoustic trauma occurred at the high frequencies, from 2 kHz to 8 kHz ($P < 0.0001$, Wilcoxon test, two-tailed).

The initial hearing loss is illustrated in Figure 2. The mean (± SD) AHL in Group 1 (No HBOT) was -25.83 ± 11.70 dB; Group 2 (HBOT+IV), -31.35 ± 19.0 dB; and Group 3 (HBOT+PO), -29.68 ± 15.68 dB. There was no statistical difference between the three groups (Kruskal-Wallis test, $P = 0.6603$). The Dunn's multiple comparison test likewise failed to demonstrate statistical significance.

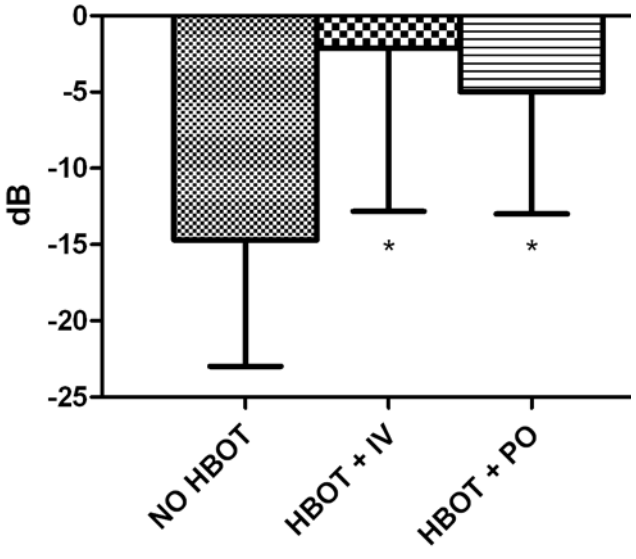
The average hearing gain is shown in Figure 3. Group 1 (No HBOT) had an AHG of +5.58 ± 3.58 dB; Group 2 (HBOT+IV), +20.62 ± 17.68 dB; and Group 3 (HBOT+PO), +17.0 ± 14.0 dB. The difference between the three groups was statistically significant (Kruskal-Wallis test, $P = 0.001$). Dunn's multiple comparison test failed to demonstrate statistical difference between the two HBOT groups but confirmed that both HBOT groups were statistically different from Group 1 ($P < 0.05$).

The average residual hearing loss is shown in Figure 4. For Group 1 (No HBOT), ARHL was -14.7 ± 8.27 dB; for Group 2 (HBOT+IV), -2.36 ± 10.69 dB; and for Group 3 (HBOT+PO), -5.0 ± 8.0 dB. ARHL was statistically significantly different for the three groups (Kruskal-Wallis test, $P = 0.001$). Again, the difference between both HBOT groups and the group without HBOT is significant ($P < 0.05$), but Dunn's multiple comparison test failed to demonstrate a statistical difference between the two HBOT groups.

Discussion

The optimal treatment of NIHL has not been well defined. In analogy with sudden sensorineural hearing loss, various treatment regimens have been proposed. The most common approach to the treatment of SSHL is the use of systemic steroids, which have been deemed by some authors to be the 'gold standard' of treatment.[11,12] However, a recent meta-analysis was unable to definitely support this statement.[13] Some authors recommend pentoxifyllin, and others

Figure 4
The average residual hearing loss in the three treatment groups (see text; * $P < 0.05$)



have reported that 12 g of piracetam administered as an intravenous infusion over 15 minutes significantly increased the chance of complete recovery for patients with SSHL.[14,15] As it is a widely accepted and recommended treatment, the 'standard' approach for AAT in the Belgian Armed Forces is high-dose corticosteroids combined with piracetam, a strategy that has been endorsed recently by the Belgian ENT Society.[10,16]

Whereas for SSHL, scientific understanding of its cause or a rational approach to its treatment is lacking,[13] in NIHL, it has been shown that one of the first effects of AAT is a decrease in the oxygen supply to the organ of Corti.[16,17] It has also been shown that noise can induce hypoxia in the auditory cortex, the hippocampus and the inferior colliculus.[18] The rationale for using HBOT is based on the fact that inhalation of pure oxygen under pressure causes an increase in the arterial partial pressure of oxygen and an increase in the oxygen diffusion distance in tissues. These principles, enhancing tissue oxygenation, are complemented by blood-flow redistribution to hypoxic areas.[19] As a consequence, and in contrast to vasodilatation treatment, HBOT treatment increases oxygen tension in the endo- and perilymph and might in this way help hypoxic cells to survive.[20,21]

An animal study of HBOT for NIHL suggested that HBOT immediately after AAT (one and two hours post exposure) may have an adverse effect, probably by an increase of oxygen free radical production.[22] When HBOT was started later (at 6, 24 or 48 hours post-exposure) this adverse effect seems to be absent, and in these groups hearing was back to the pre-exposure level, as demonstrated by levels of signal-to-noise ratio, within 10 days post exposure.[22] This positive effect has also been suggested in another recent animal study in which only a regimen of combined HBOT and corticosteroids provided significant protection from NIHL, especially when started one day post exposure. Hearing recovery induced by this treatment regimen was about 10–15 dB.[23] These two animal studies support our strategy to use HBOT as a primary tool in association with corticosteroids in the treatment of AAT. Our current treatment protocols adhere to a therapeutic window from 6 to 48 hours, as suggested by Cakir et al.[22]

In this study a significant therapeutic effect on noise-induced hearing loss was only achieved in the HBOT groups. This supports the idea that HBOT therapy is an important therapeutic tool and that medical therapy alone, like minimal therapy or no therapy (waiting for spontaneous recovery), is not the treatment of choice. HBOT was associated with significant improvement in PTA thresholds, although full recovery had not occurred by 10 days post injury. Compared to the baseline PTA at enlistment, even the HBOT groups were left with a residual loss. However, both HBOT groups have gained statistically significant better hearing recovery than the group not receiving HBOT.

When comparing both HBOT groups, a combination of aggressive HBOT and initial treatment with intravenous corticosteroids seems to be the best option. This could be interpreted as a confirmation that HBOT started as early as possible, but not in the first 6 hours post injury to avoid any possible adverse effect of HBOT, produces better results, while therapy started later (after sensory cell death) produces poorer results. The relatively small numbers of patients in the HBOT groups may have been insufficient to demonstrate a significant difference. Alternatively, it is possible that there is indeed no difference between the two HBOT regimens; this would mean that the 'HBOT+IV' group was treated unnecessarily aggressively. From this study, it is not possible to obtain a clear-cut recommendation as to the best HBOT regimen.

This retrospective study on the treatment of AAT has limitations. Despite the fact that clear guidelines for the approach to AAT are available in the Belgian Armed Forces, and that all patients received a standardised emergency treatment, the study was neither prospective nor randomised. Therefore, it is possible that some referral bias played a role in the decision to refer patients acutely for HBOT. Although the differences in AHL in the three groups were statistically not significant, Group 1 had slightly less hearing damage than the other groups. It is possible, although improbable, that the presence or severity of other symptoms (such as tinnitus) may have played a role in the decision to refer patients over an often considerable distance. We tried to minimise the influence of these confounding factors by selecting only those patients who had a severe decrease in their hearing and who failed to improve within the first 24 hours (to exclude patients with TTS only). This, however, reduced the number of patients available for analysis, decreasing the power of the study.

Performing a randomised controlled trial with a placebo group in this disease would probably be inappropriate because several treatments have been shown to have some degree of efficacy. Furthermore, the practical implementation of sham hyperbaric treatment is difficult, and its validity has been questioned.[24] Therefore, in order to obtain evidence regarding the efficacy of a new treatment, it is acceptable that it is tested against the best available treatment.[25] A randomised, prospective study is being conducted in our hyperbaric unit. We hope to open this to multicentre collaboration among military centres in NATO countries and beyond. This study will compare different combinations of HBOT and intravenous or oral corticosteroids, in a similar group of well-defined 'ears' and AAT.

Conclusions

This study demonstrates a clear benefit from the combination of HBOT and medical therapy over medical treatment alone. It suggests that the more aggressive the combined treatment is at an early stage, the better the results. However, at this stage, strong evidence to demonstrate the superiority of one HBOT protocol over another is lacking.

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Submitted: 15 June 2009
Accepted: 10 May 2010

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Review article

The two faces of Eve: gaseous anaesthesia and inert gas narcosis

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Key words
Anaesthesia, nitrogen narcosis, inert gas narcosis, xenon, pharmacology, physiology, review article

Abstract

(Smith CR, Spiess BD. The two faces of Eve: gaseous anaesthesia and inert gas narcosis. *Diving and Hyperbaric Medicine*. 2010;40(2):68-77.)
Gaseous anaesthesia has been a great boon for medicine. These drugs form a foundation from which modern surgery has sprung, yet their mechanism(s) of actions remains poorly understood. Inert gas narcosis is a limitation of deep sea diving, and its mechanisms also remain poorly understood. In this review article we summarise what is known about the mechanisms of both gaseous anaesthesia and inert gas narcosis, including both lipid-based biophysical models and protein-based biochemical models, as well as explore some striking similarities between the two. These two phenomena may, in reality, be gradations of the same underlying mechanism. Recent findings include biochemical evidence suggesting that both gaseous anaesthesia and inert gas narcosis may be mediated by the occupation of minute spaces within the structure of many biologically important proteins, impairing their ability to undergo conformational changes and biological actions. This is exemplified by exploring the effects of the noble gas xenon, which can behave as either a narcotic gas or gaseous anaesthetic, depending on the partial pressure in which it is present.

Introduction

Volatile gas anaesthesia represents a transformational medical advance of the last 200 years. Introduced 160 years ago, these drugs form a foundation from which modern surgery has sprung. Inhaled anaesthetics have the closest lethal dose to effective dose ratio of all drugs used in medicine, requiring an entire specialty to be developed in order to ensure safe utilization. While gas anaesthesia has been a great boon for medicine, inert gas narcosis has been a limitation of deep sea diving. This limitation has impaired our ability to explore the 70% of our planet covered by water, except through indirect means (i.e., submersibles). In fact, it has been remarked that we know more about the surface of Mars than we do about the deep ocean. A discussion of the seemingly far removed topics of general anaesthesia and nitrogen narcosis leads to many questions. What if gaseous anaesthesia and inert gas narcosis are, in reality, different manifestations of the same phenomenon? More basic is the question: are we adapted to a very narrow ambient pressure environment due to inert gas effects? Previously inert gases have been felt to have no physiologic effects but we herein hypothesize that they have complex, little understood effects that indeed modulate many cell membrane and protein functions. It is through a wider understanding and, perhaps, entertaining the notion that inert gases exert very necessary physiologic ordering effects that we accept as 'normal' that we can understand such previously non-investigated phenomena.

Mechanisms

When Behnke et al proposed that nitrogen, or, more broadly, the inert gas fraction of the breathing gas, is responsible for narcosis in 1935, the assertion was based on the Meyer-Overton hypothesis.[1] That hypothesis states that the narcotic potency of an anaesthetic (or an inert gas) is related to its lipid solubility.[2,3] Lipid solubility is the physical property of inert gases that has been found to correlate most consistently with their narcotic/anaesthetic potency.[4] The Meyer-Overton hypothesis, with regards to inert gas narcosis, was found to be tenable by Carpenter when he showed that at iso-narcotic partial pressures (the partial pressure at which each gas shows comparable pharmacologic effects), the inert gas concentration dissolved in the lipid phase is very similar across many gases.[5] The partial pressure of various gases required for narcosis varies from 46 kPa to 16.5 MPa (0.045 to 165 Ata).[5] Table 1 illustrates the lipid solubility to relative narcotic potency correlation properties (Table 1).[4]

Once it was established that the site of action was most likely within the lipid phase, hypotheses began to emerge regarding what was actually taking place that would result in narcosis with identical signs and symptoms being induced by a broad collection of gases with no common structural features.[4] Several hypotheses have evolved, including hypoxia, depression of metabolism, cell membrane stabilization, membrane stiffening causing decreased ion permeability, inhibition of the sodium extrusion pump, increased production of inhibitory neurotransmitters such as gamma aminobutyric acid (GABA), and interference with adenosine triphosphate (ATP) production.[7] These hypotheses fall into two broad categories: biochemical or physical hypotheses. Biochemical hypotheses imply some effect on respiratory enzyme systems, while physical theories imply some interaction with, or within, part of the cell, such as the cell membrane.[8] Until recently, no good

Table 1
Narcotic potencies and physical properties of simple gases[4,6]

Gas	Molecular mass (g/mol)	Van der Waals radius (pm)	Anaesthetic pressure (Ata)	Oil:gas partition coefficient (at 37°C)	Relative narcotic potency
Helium	4	140	190.546	0.016	0.23
Neon	20	154	87.096	0.019	0.28
Hydrogen	2	120	138.038	0.05	0.55
Nitrogen	28	155	33.113	0.069	1.00
Argon	40	188	15.136	0.13	2.33
Krypton	83.7	202	4.467	0.4	7.14
Xenon	131.3	216	0.955	1.8	25.64

evidence had been found to support biochemical changes at pressures relevant to the clinical manifestations of inert gas narcosis. This suggested that the narcotic action is more likely biophysical than pharmacologic, and evolved into the 'unitary hypothesis of narcosis': that the mechanism of narcosis is the same for all anaesthetic gases.[9]

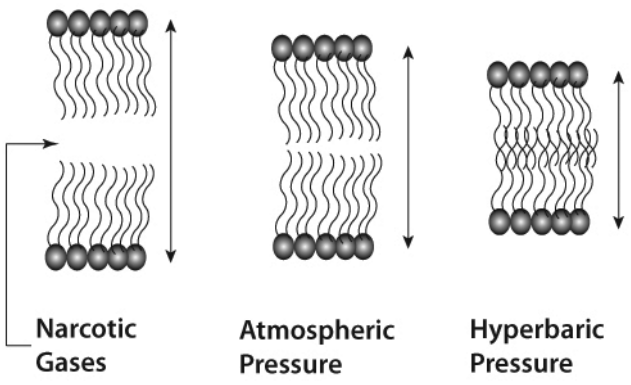
By the late 1950s, the site of action of narcotic gases had been attributed to synapses in the central nervous system. This was deduced largely from the work of Carpenter in the mid 1950s.[5] He demonstrated that 31.4–34.5 MPa (310–340 Ata) of argon, a gas with a narcotic potency more than twice that of nitrogen, were required to effect a block of conduction in isolated peripheral nerve preparations, but argon at a mere 1.8 MPa (18 Ata) of pressure was sufficient to abolish any response to electrical stimulus applied to the foot pad of mice.[5,10] This suggests strongly that higher level functions in the brain are much more susceptible to inert gas narcosis than peripheral nerves. Later work examining reflex inhibition in the spinal cord demonstrated that inhibitory synaptic mechanisms were affected by inert gas narcosis before excitatory mechanisms, and that inert gas narcosis, like general anaesthetics, affects cells in the anterior horn of the spinal cord.[7]

LIPID/MEMBRANE HYPOTHESES

Physical hypotheses, based on the polarization and volume of inert gas molecules are simpler and, perhaps therefore, more likely than biochemical hypotheses.[11] The critical volume hypothesis of general anaesthesia proposes that narcosis occurs when the anaesthetic agent enters the lipid portion of the cell in sufficient quantity to cause, in particular, the plasma membrane to swell.[12] Accordingly, changes in lipid volume ought to differentiate the anaesthetised from the unanaesthetised state. The critical volume hypothesis is supported by observations that anaesthetics and inert gases at increased ambient pressures expand the volume of lipid monolayers and bilayers, bulk phase solvents, oils and even rubber.[13] This hypothesis is further supported by the observation that gas anaesthesia can be reversed with the application of hydrostatic pressure (Figure 1).[12,14] The quantitative aspect of this hypothesis was developed, which suggested that a 0.4% expansion of the membrane would be required to produce anaesthesia.[15]

Although elegant in its simplicity, there are critical elements of this hypothesis that do not agree with observations. The critical volume hypothesis predicts that the percentage change in anaesthetic potency ought to be linearly related to pressure, and that the slope of this relationship ought to be the same for all anaesthetic agents.[16] This has not been found to be true.[17] Quite the opposite, it has been shown that these pressure/anaesthetic interactions are curvilinear, and differ depending on the anaesthetic in question.[17] For instance, the amount of nitrogen required to maintain a given level of anaesthesia increases with pressure to approximately 5.1 MPa (50 Ata), at which point it plateaus and there is no further increase for pressures up to about 13.2 MPa (130 Ata).[17] Conversely the requirements for isoflurane decrease up to pressures of about 811 kPa (8 Ata), after which they increase sharply, and continue to increase up to pressures of about 10.1 MPa (100 Ata).[17] Also, it has been observed that there is no appreciable increase in membrane thickness at narcotic concentrations of various agents.[18]

Figure 1
The critical volume hypothesis. Molecules of narcotic gases dissolve in the cell membrane and cause it to swell. When the membrane reaches a certain volume narcosis is produced. Pressure reverses this narcotic effect by compressing the membrane.



These observations led to the postulation of the multi-site expansion hypothesis.[16] As stated by Halsey *et al*, there are five key elements to this hypothesis:[16]

- *General anaesthesia or narcosis can be produced by the expansion of more than one molecular site and these sites may have different physical properties.*
- *The physical properties of a molecular site may themselves be influenced by the presence of anaesthetics or pressure (i.e., compressibility).*
- *The molecular sites do not behave as if they were bulk solvents but have a finite size and a finite degree of occupancy.*
- *Pressure need not necessarily act at the same site as the anaesthetic in order to reverse anaesthesia. Depending on the anaesthetic, one of the sites may predominate in determining the interaction between anaesthesia and pressure.*
- *The molecular sites for anaesthesia are not perturbed by a decrease in temperature in a manner analogous to an increase in pressure.*

Other work on membrane model systems suggested that anaesthetic gases may also alter the permeability of ions. While examining cation permeability in membrane model systems, it was observed that n-alkyl alcohols, chloroform and ether result in a transient, reversible increase in membrane cation permeability.[19] Inert gases behave the same way, as demonstrated by *in vivo* examination of cerebrospinal fluid (CSF) levels of sodium, potassium and chloride while measuring auditory evoked potentials in cats. A significant decrease in CSF sodium and chloride was found, as well as the amplitude of cortical auditory evoked potentials in animals compressed to 1.1 MPa (11 Ata) breathing a mix of 80% nitrogen/20% oxygen or 80% argon/20% oxygen when compared to breathing 80% helium/20% oxygen at 1.1 MPa (11 Ata) or room air at ambient pressure.[20]

This is further supported by studies by Johnson and Miller as well as by Galey and van Nice.[21,22] In liposomes exposed to butanol, ether or nitrogen in doses that would be just sufficient to abolish the righting reflex in newts, an increase in the permeability for potassium and rubidium was observed, and application of 15.4 MPa (152 Ata) of pressure was required to counterbalance the permeability changes.[21] Pressures of nitrogen up to 8.9 MPa (88 Ata) stimulate active sodium efflux and potassium influx across the red blood cell membrane, and the effect is abolished by ouabain.[22] In addition, hyperbaric pressures of the non-narcotic gas helium, rather than nitrogen, tended to inhibit active sodium and potassium transport.[22] Other work by this group using rat brain synaptosomes showed that hyperbaric pressures of argon would stimulate potassium uptake by the synaptosomes while 7.0 MPa (69 Ata) of helium or hydrostatic pressure inhibited the accumulation of potassium.[23] This suggests that anaesthesia and narcosis may be the downstream product of gases dissolving into the membrane at various sites, altering ion conduction, and thus synaptic conduction, and ultimately consciousness.

More recently, Abraini has modified this multi-site expansion model based on results obtained from human trials using hydrogen, a relatively non-narcotic gas, as the diluent gas.[24] He has proposed theoretical reconsideration of the interaction between inert gases at hyperbaric pressure, and the effects of pressure itself. According to this hypothesis, narcotic gas and pressure act at different hydrophobic sites and narcosis occurs when a critical level of expansion is reached at some cellular hydrophobic site in the central nervous system. With respect to light inert gases, all narcotic gases act at a common hydrophobic region through a non-specific mechanism.

This hypothesis suggests that the psychotic-like symptoms observed in humans at high pressure may be a paroxysmal symptom of narcosis, not simply a manifestation of the high pressure nervous syndrome (HPNS), and are a result of the sum of the individual narcotic potencies of the various inert gases in the breathing mix.[24] This was tested mathematically against various lipid solubility theories of inert gas narcosis and was found to be sound. This suggests that, depending on the environmental parameters (breathing mix, pressure), symptoms of inert gas narcosis or HPNS appear when a critical imbalance is reached between the narcotic actions of inert gas and the actions of pressure, which tend to reverse narcosis, on their respective hydrophobic sites. Accordingly, inert gas narcosis and HPNS can antagonise each other, or can occur simultaneously.[24]

Given the understanding that inert gas narcosis was somehow connected to changes in synaptic conduction in the central nervous system, some researchers began to investigate changes in neurotransmitters with hyperbaric exposure. Changes in levels of dopamine and norepinephrine have been observed by several groups, but whether an increase or decrease is observed seems to depend on what area of the brain is under investigation rather than the pressure applied. For instance, dopamine and norepinephrine were shown to be decreased in response to 10.1 MPa (100 Ata) trimix (helium/nitrogen/oxygen) and to 2.0 MPa (20 Ata) nitrogen/oxygen mixtures in the hypothalamus, but were increased in the caudate nucleus.[25]

Unfortunately these changes may not have anything to do with inert gas narcosis. Rostain and Forni were able to demonstrate a similar increase in striatal dopamine release in response to 9.1 MPa (90 Ata) helium/oxygen mixture, 9.1 MPa (90 Ata) helium/nitrogen/oxygen mixture (5% nitrogen), and 9.1 MPa (90 Ata) helium/hydrogen/oxygen mixture (66% hydrogen).[26] These mixes should have quite different narcotic potencies, but appeared to cause the same change in dopamine levels. These changes were attributed to, and are likely the result of, pressure alone, not narcosis. Balon et al, while also looking at striatal dopamine release, found a 20% decrease in rats exposed to 3.0 MPa (30 Ata) breathing a nitrogen/oxygen mix.[27]

This suggests that neurotransmitter release in response

to hyperbaric exposure and inert gases is quite complex. Exposure to low pressures, breathing a mixture of nitrogen/oxygen, appears to result in a decrease in striatal dopamine release, while exposure to high pressures, breathing a helium/oxygen mixture, increases dopamine release. Unfortunately it is very difficult to separate out the effects of the inert gas from the effects of pressure *per se* because gases with high narcotic potency will result in unconsciousness at high pressures. Thus, in order to expose an *in vivo* preparation to high pressures, helium must be used as a diluent gas if any sort of behavioural observations are to be made. So long as gas pressure is used to generate a hyperbaric exposure, it will remain near impossible to distinguish the effects of high-pressure helium from those of pressure *per se*. Nonetheless, it would appear that there are consistent, reproducible changes in dopamine release in response to hyperbaric exposure. This suggests that neurotransmission is likely altered under hyperbaric conditions, and may offer a partial explanation of inert gas narcosis.

Other work has suggested that nitric oxide may play a role in narcosis. Vjotosh et al found that when rats were compressed to 4.2 MPa (41 Ata) breathing air, they showed alterations in motor activity at 0.5–1.2 MPa (5–12 Ata), ataxia at 1.0–3.4 MPa (10–34 Ata), and side body position at 2.6–4.2 MPa (26–41 Ata).[28] These were taken as signs of nitrogen narcosis. When treated with the nitric oxide synthase inhibitors L-NAME or 7-NI, the above mentioned signs were abolished or attenuated. While interesting, these results must be taken with a grain of salt. Air breathing at pressures greater than 1.0 MPa (10 Ata) makes acute oxygen toxicity a serious risk. Since seizures are one of the symptoms of acute oxygen toxicity the indicators this group used as signs of narcosis may make narcosis and oxygen toxicity difficult to distinguish.

PROTEIN/METABOLIC HYPOTHESES

Research attention is now focusing on the possibility of direct interactions between inert/anaesthetic gases and proteins, lipoproteins, and other hydrophobic sites within the cell.[29] Much of this evidence comes from the anaesthesia community and the study of volatile, inhaled anaesthetics in general. If inert gas narcosis was solely a matter of gas dissolving in lipid membranes, it would be expected that the onset of narcotic effects would be linearly related to the rate of increase in pressure.[23] That this relationship is, in fact, sigmoidal suggests that the inert gas molecules are interacting with protein receptors directly and act as allosteric modulators.[29]

The idea that the mechanism underlying anaesthesia involves an interaction with proteins is not new. This was first proposed in 1875 by Claude Bernard.[30] He based this conclusion on the way some anaesthetic potencies deviated from that which would be predicted from their lipid solubility alone, combined with the understanding that many proteins contain small hydrophobic domains that would allow for interactions with small, hydrophobic compounds.[30] Unfortunately the interactions between proteins and narcotic compounds appear to be very short-lived (a millisecond or less).[31] Conventional binding assays are simply unable to measure such low-affinity binding.[31]

Since direct measurements of binding are not possible, those interested in studying protein-based mechanisms of anaesthesia and narcosis have resorted to molecular pharmacology and assays of protein activity in various *in vitro* preparations. These techniques have evaluated possible protein/anaesthetic interactions based on two criteria: 'plausibility' and 'sensitivity'. Plausibility refers to the degree to which changes in protein activity observed in the preparation line up with our preconceptions of anaesthetic mechanisms.[32] For instance, it is believed that anaesthesia and narcosis are products of CNS depression; therefore, the observation that an anaesthetic inhibits proteins involved in excitatory synaptic transmission, or activates proteins involved in inhibitory synaptic transmission would fit the 'plausibility' criterion. The sensitivity criterion would come into play in order to evaluate the dose-dependence of the observed changes in protein activity. This criterion would be satisfied if the observed *in vitro* EC_{50} (effective concentration for 50% of the effect) were similar to the observed clinical EC_{50}. Plausibility, in this sense, is certainly a very fuzzy concept. Our understanding of the neurophysiology of consciousness is very limited. Consequently our understanding of altered states of consciousness is even more limited, so plausibility is very much open to the subjective interpretation of the investigator.

Inhalational anaesthetics including inert gases have been investigated in several different *in vitro* systems, and have been found to alter the functions of many enzymes, receptors, transporters, ligand- and voltage-gated ion channels as well as structural proteins.[31] A growing body of evidence suggests that inhalational general anaesthetics work through interactions with proteins, particularly post-synaptic ligand-gated ion channels.[33] These interactions fit well, not only with the plausibility criterion, but with the sensitivity criterion; the doses observed to have appropriate *in vitro* effects are very similar to clinical doses used to produce general anaesthesia.[34]

The idea that inhaled anaesthetics exert their effects through modulation of inhibitory post-synaptic ligand-gated ion channels is interesting. When activated by the binding of the appropriate ligand (e.g., gamma aminobutyric acid, GABA) these chloride channels open and chloride flows into the cell causing a hyperpolarisation and decreasing the likelihood of action potential propagation.[34] Clinically effective concentrations of several inhaled anaesthetics, as well as high partial pressures of nitrogen, have been demonstrated to potentiate both GABA- and glycine-modulated chloride currents *in vitro*.[35] Induction of the GABA receptor system is not the only ligand-gated ion channel that anaesthetic and narcotic gases have been demonstrated to interact with,

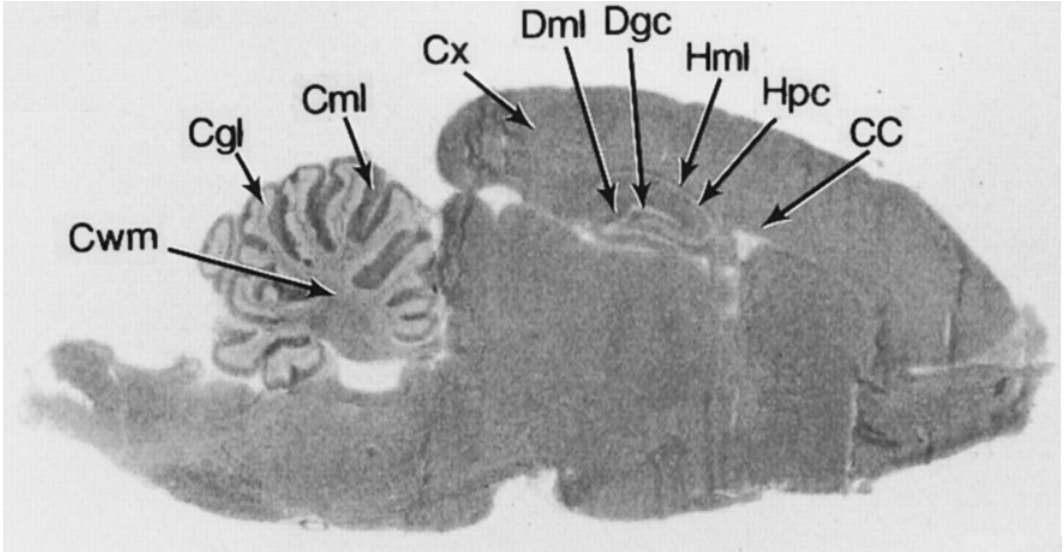
and alter the function of. Specifically, it has been elegantly demonstrated by Balon et al that the inhaled anaesthetic nitrous oxide exerts its anaesthetic effects via inhibition of the NMDA receptor.[36]

Several problems still exist with the hypothesis that inhaled anaesthetics operate through the modulation of ligand-gated ion channels such as the GABA receptor. First, *in vitro*, it has been shown that at high doses of anaesthetic drugs (above 1 mM), $GABA_A$ activity tends to be inhibited, yet clinically, increasing anaesthetic doses lead to deeper anaesthesia, not reversal.[35] Second, in neonatal rodents, chloride gradients are reversed, thus GABA acts as an excitatory neurotransmitter, but inhaled anaesthetics are still effective in these animals, although slightly higher doses are required.[37] Third, if the anxiolytic effect of benzodiazepines is a result of potentiation of the $GABA_A$ receptor, inhaled anaesthetics must act through a different mechanism since their effect is decidedly non-anxiolytic.[37] The early stages of general anaesthesia induced with inhaled anaesthetics produces an excitatory phase, which can produce seizure-like activity, whereas benzodiazepines prevent seizures. Fourth, chloride channel blockers and $GABA_A$ antagonists have only minimal effects on the potency of inhaled anaesthetics.[38,39] This evidence suggests that, although volatile anaesthetics can modulate ligand-gated chloride channel activity at clinically relevant concentrations *in vitro* (and possibly *in vivo*), it is unclear how this effect is related to anaesthesia.

Anaesthesia may be a product not of interaction with a single protein, but of interaction with multiple molecular targets. This is suggested by the observations that inhaled anaesthetic agents affect multiple proteins, as well as the fact that multiple anaesthetics with diverse molecular structures all produce the same end result: anaesthesia.[40] There is evidence for unique binding sites for several inhaled anaesthetics on a single target, but it seems unlikely that a single molecular target would have specific binding sites for diverse molecular structures ranging from xenon to nitrous oxide to sevoflurane.[41] Other evidence suggests that there may be a single, selective target for each anaesthetic, but it seems unlikely that all these molecular targets would lead to the same end result of anaesthesia.[42] However, this finding does provide reasonable grounds to believe that interactions with multiple molecular targets may converge to produce the single effect of anaesthesia.[40]

It should be remembered that specific interaction with a protein target and multiple sites of action are not mutually exclusive. It is well understood that adenosine triphosphate (ATP), oxygen and calcium all bind selectively with multiple different targets. Volatile anaesthetics have also been shown to bind selectively with multiple different targets, including firefly luciferase, serum albumin, myoglobin, adenylate kinase, haloalkane dehalogenase and T4 lysozyme.[43,44] Even more compelling evidence comes from autoradiograms of rat brain slices probed with a radiolabelled halothane derivative. The radiographs revealed widespread binding of the labelled halothane derivative throughout the brain. The distribution of the labelled halothane derivative did not match that of any known receptor or channel (Figure 2).[45] Furthermore, the binding was reduced to background levels in the presence of a 10-fold excess of unlabelled halothane. When extracted and separated, multiple brain proteins were found to be specifically labelled in a saturable and stoichiometric manner with estimated affinities near the

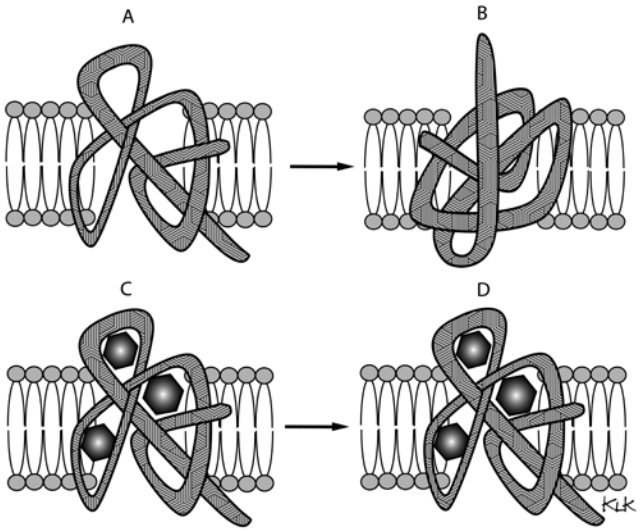
Figure 2
Autoradiogram of rat brain section photoaffinity-labelled with radioactive halothane. Degree of halothane binding is indicated by level of darkness; no other staining has been applied to the section. The binding shows little regional preference and is reduced to near-background levels in the presence of a tenfold excess of unlabeled halothane; labels indicate various brain regions.[45] (with permission)



clinical EC_{50}.[40] The dramatic inhibition of labelling by non-radioactive halothane indicates that most halothane binding is saturable and specific, showing that many proteins could be involved in anaesthetic action.[45]

The nature of the interaction between anaesthetics and proteins may lie in the structure of proteins themselves. It is understood that proteins fold into complex 3-dimensional structures that are not solid, but rather contain cavities. These cavities are believed to be critical structural elements in protein function as they introduce areas of instability that allow conformational changes to take place.[46] These cavities within the hydrophobic core of proteins provide plausible binding sites consistent with the observation that anaesthetic potency is correlated with lipid solubility (Figure 3). That potent anaesthetics exhibit weak polarity is also consistent with the hypothesis that they bind in protein cavities, as most cavities are also weakly polar.[46,47] The elements of protein secondary structure that form the surface of these cavities could also provide an explanation for the weak stereo selectivity observed with some anaesthetics such as isoflurane.[48] Occupancy of these cavities by anaesthetic molecules could affect anaesthesia by limiting the motion that underlies protein activity.[47,49] Studies have indicated that occupancy of cavities by small, hydrophobic molecules does reduce protein motion.[46] This is clearly a multiple-target hypothesis, and nicely reconciles results from binding and functional studies.

Figure 3
Anaesthetic gases may exert their effects through the occupation of small cavities in proteins. A depicts a protein in its resting, inactive state. When activated (B), the protein undergoes a conformational change. C depicts a protein in its inactive state with molecules of an anaesthetic or narcotic gas occupying cavities within the tertiary structure of the protein. In D, the protein is unable to undergo conformational change, and thus remains inactive.



Xenon

Thus far, the terms 'inert gas narcosis' and 'anaesthesia' have been, to a degree, used interchangeably. This is based on the hypothesis that all anaesthetics act through the same basic mechanisms, and the observations that inert gas narcosis resembles early stages of anaesthesia and that inert gases can produce general anaesthesia if delivered at sufficiently high partial pressures. The inert gas xenon exemplifies much of this overlap. Xenon is an inert gas with a narcotic potency sufficiently high that it can be used as a general anaesthetic at 101.3 KPa (1 bar).[50] If it can be assumed that inert gas narcosis exists as a single condition regardless of the inert gas in question (to a large extent the research community has already done so in order to try to separate narcotic effects from pressure effects), what is understood about the mechanism of xenon anaesthesia can be extended to all inert gas narcosis and gaseous anaesthesia.

Investigations into the mechanisms of action of xenon gas anaesthesia present a microcosm of investigations into the mechanism of action of the entire scope of inhalational anaesthesia. Hypotheses abound regarding both lipid (membrane) and direct interaction with protein. In support of the membrane hypothesis, there is evidence that xenon can interact with lipid bilayers and change surface tension, bilayer volume, and pressure within the bilayer.[51] There is also evidence to suggest that the xenon dissolved in the membrane, despite its high lipid solubility, may not reside within the central core, or tail region of the membrane, but may preferentially migrate to the amphiphilic region around the head groups after dissolving in the membrane.[51] This accumulation of xenon molecules in the membrane is believed to affect the structure and function of proteins embedded within the membrane.[52] In particular, it is believed that changes in surface tension and pressure within the membrane result in conformational changes in ion channels within the synaptic terminals of neurons, resulting in decreased conduction leading to anaesthesia.[52]

The greatest criticism levelled against much of the work done on the basis of the lipid hypothesis of narcosis, be that *in vivo* or *in silico*, is the fact that most of these studies are done using dipalmitoylphosphatidylcholine (DPPC) bilayers as a membrane system.[32] DPPC is an easily obtained egg yolk phospholipid that contains no double bonds to oxidise and readily forms into a biologically relevant liquid crystalline bilayer. Unfortunately it is also far too simple a system to accurately model a biological membrane. Living plasma membranes, especially those of neurons, contain a highly heterogeneous mixture of lipids, some saturated, some unsaturated, some charged, some neutral, as well as cholesterol.[53] Biological membranes also contain various proteins, which make up approximately 50% of the mass of the membrane.[54] In addition, it is now understood that the inner and outer leaflets of biological membranes are composed of different phospholipids, and that the membrane

is arranged laterally into distinct areas known as lipid rafts, which are composed of distinct lipids and proteins.[54] For example, the nicotinic acetylcholine receptors in nerve and muscle cells require the presence of cholesterol and anionic phospholipids in their immediate vicinity in order to function properly.[55] The simple membrane systems often used experimentally cannot approximate well the behaviour of biological membranes and thus their findings for the mechanism of narcosis are questionable. On the other hand, these models do demonstrate that it is plausible that the membrane is, at least in part, responsible for the mechanism of narcosis.

There is evidence that xenon anaesthesia/narcosis may be a product of interaction with proteins. Whereas most general anaesthetics have been shown to enhance the inhibitory activity of $GABA_A$ receptors, xenon seems to have little or no effect on them.[56] Instead, it appears that xenon inhibits the excitatory action of the *N*-methyl-D-aspartate (NMDA) receptor and, to lesser extents, the neuronal nicotinic receptor and the TREK-1 two-pore K^+ channel.[57,58] Interestingly the NMDA receptor, a subtype of the glutamate receptor, is believed to be involved in learning, memory and the perception of pain, which could explain the attractive pharmacodynamic properties of xenon.[59] This inhibition of the NMDA receptor by xenon goes a long way toward explaining the analgesic and amnesic effects of xenon.

X-ray crystallographic studies have also been performed in order to examine how and where xenon is interacting with various proteins. Unfortunately, membrane-bound proteins, such as the NMDA receptor, are difficult to crystallise, so xenon has not been crystallised with the protein actually believed to be its target, but with soluble surrogate proteins. Xenon has been crystallized with urate oxidase, a prototype of various intracellular globular proteins, and with annexin V, a protein with structural and functional characteristics that allow it to be considered a prototype of the NMDA receptor.[60,61] A single xenon molecule was found to bind to both these proteins in a flexible, hydrophobic cavity within the structure of the protein. This is consistent with both the hydrophobicity of xenon, and with the previous hypothesis that anaesthetic molecules exert their effect by binding proteins in these hydrophobic cavities.[46,47] This suggests that it is plausible that xenon binds with the NMDA receptor, but that it is also capable of binding to a wide range of soluble intracellular proteins, consistent with the hypothesis that anaesthesia/narcosis is likely the ultimate product of multiple drug/protein interactions.[40]

Other anaesthetic compounds that are believed to operate primarily by inhibiting NMDA-receptor signalling, such as nitrous oxide and ketamine, have been observed to increase both global and regional cerebral metabolism in humans.[62] Thus it would be expected that, if the anaesthetic action of xenon also operates primarily by inhibiting the NMDA-receptor, it should also increase both global and regional cerebral metabolism.[63] Cerebral metabolic rate, which is depressed relative to the conscious state by most general anaesthetics, can be examined using positron emission tomography and such studies have been performed.[62,63] Contrary to expectations, xenon anaesthesia depressed cerebral metabolism both globally and in multiple regions of the brain.[63] This suggests strongly that the mechanism of xenon anaesthesia/narcosis is not simply the inhibition of NMDA receptors.

Conclusion

Inert gases clearly have physiological effects. The fact that the inert gas xenon fills voids in proteins is itself provocative. Work has shown inert gases act on neurons, nerve conduction and consciousness. Today, a picture of how inert gases function as anaesthetics is beginning to emerge. The idea that inert gas narcosis can be reduced to a single cause is likely incorrect. The symptoms displayed vary widely, not only between different individuals, but also between different exposures for the same individual. The conditions required to bring about the onset of inert gas narcosis (ambient pressure, gas mix being breathed, temperature, psychological factors) also appear to vary widely. Hypotheses focusing exclusively on either cell membranes or protein interactions do not appear to tell the whole story. At this point in the research it would appear that there are elements of truth in both of these theories. Continued research into both inert gas narcosis, and the mechanisms of general anaesthesia, particularly mechanisms pertaining to inhaled anaesthetics, is likely to further understanding of both conditions. The time may be nearing when, in order to truly understand the mechanisms of inert gas narcosis and general anaesthesia, hypotheses will need to be able to bring together understanding gained from both lipid- and protein-based models in order to construct a single model that can explain all the observations.

It is clear that pressure, combined with inert gases, changes cell functions. From such an observation, a series of conclusions tumble: membranes, proteins and other bio-active molecules have evolved to their functions on Earth's surface perhaps in a selected 'pressure/inert gas window of normal activity'. Therefore, inert gases may not be truly 'inert', as they exert ordering effects upon membranes, proteins and cell signalling. They clearly do not react by ionic bonding or undergo metabolism like oxygen and carbon dioxide but neither are they non-participators, the inert gases provide order. The proposed mechanisms discussed in this article are summarised briefly in the Appendix.

For readers who would like further background information on inert gas narcosis or mechanisms of anaesthesia, please refer to the chapter by Bennett and Rostain on inert gas narcosis in *Bennett and Elliott's physiology and medicine of diving*,[64] or, for mechanisms of anaesthesia, a 2001 article by RG Eckenhoff in *Molecular interventions*.[65]

Appendix
Summary table of the major hypotheses as to the mechanism(s) of gas anaesthesia and inert gas narcosis

Hypothesis	Mechanism of anaesthesia	Evidence
Physical models		
Critical volume	Physical expansion of plasma membrane	Increasing partial pressures of anaesthetic gases increase the volume of various lipids.[13] Gas anaesthesia can be reversed by hydrostatic pressure.[12,14]
Multi-site expansion	Physical expansion of discrete sites in the plasma membrane	Slope of anaesthetic potency vs. pressure found to be non-linear and different for various agents.[16,17]
Changes in ion permeabilities	Anaesthetic agents alter plasma membrane permeabilities to various ions	Application of inert and anaesthetic gases alters ion content on either side of various lipid membranes.[19–23]
Modified multi-site expansion	Anaesthetic gases and pressure have separate actions at separate membrane sites	All light anaesthetic gases interact with the membrane at a common hydrophobic site.[24] Narcosis and HPNS can coexist in the same individual at the same time.[24]
Biochemical models		
Altered synaptic conduction	Changes in neurotransmitter release at CNS synapses affect consciousness	Hyperbaric exposure and breathing gas mix alter the release of various CNS neurotransmitters.[25–28]
Single-protein interactions	Anaesthesia results from the interaction of anaesthetic molecules with certain specific functional proteins	Various anaesthetics alter conduction through specific post-synaptic inotropic and metabotropic ion channels.[31–35]
Multi-protein interactions	Anaesthesia results from the specific interaction of anaesthetic molecules with multiple functional proteins	Many anaesthetic gases have been found to bind selectively with multiple different target proteins.[42,43] Halothane binds in a competitive, saturable, stoichiometric manner to multiple proteins throughout the brain.[44] Protein tertiary structures contain small hydrophobic cavities with weak polarity. Occupation of these cavities by small molecules reduces protein motion.[45–47]

Acknowledgements

The authors would like to thank Kandice L Klepper for her illustrations.

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Submitted: 03 September 2009
Accepted: 03 February 2010

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The world as it is

ANZHMG statement on the administration of mild hyperbaric oxygen therapy

David Smart and Michael Bennett

Key words
Mild hyperbaric therapy, hyperbaric oxygen therapy, hyperbaric facilities, medical conditions and problems, evidence, medical society, policy

Executive summary

(ANZHMG statement on the administration of mild hyperbaric oxygen therapy. *Diving and Hyperbaric Medicine*. 2010;40(2):78-82.)
'Mild' hyperbaric therapy (MHT) and hyperbaric oxygen therapy are easily confused. Essentially the difference lies in the effective oxygen dose. Oxygen is an extremely useful and efficacious drug in a wide range of medical conditions. MHT does not typically provide more available oxygen to the body than is possible with oxygen administration at one atmosphere (sea level), and there is no known therapeutic benefit of mild compression alone. There is, therefore, no documented, biologically plausible evidence for the use of MHT over delivery of oxygen by a simple facemask at one atmosphere of pressure. MHT is advocated for a wide range of clinical conditions, in particular for chronic neurological conditions and as part of a suite of 'wellbeing' therapies. The Australia and New Zealand Hyperbaric Medicine Group, a standing sub-committee of the South Pacific Underwater Medicine Society, is not aware of any reliable clinical evidence for therapeutic benefit from mild hyperbaric therapy and does not recommend the use of this modality for any medical purpose.

Introduction

Hyperbaric oxygen therapy (HBOT) is an established treatment for a number of health conditions. It is available at approximately 18 centres around Australia and New Zealand, including both public hospitals and private facilities. More recently, a number of centres have opened that offer an apparently similar therapy using low-pressure treatments. The term 'mild hyperbaric therapy' (MHT) is the one most often used to describe this form of treatment. For the purpose of this document, we suggest this term is more or less synonymous with 'mild hyperbaric oxygenation', 'low-pressure hyperbaric therapy' and related terms. These terms all signify a form of therapy that differs substantially from conventional HBOT. The purpose of this document is to clearly define the relative places of these two therapies within the context of medical practice in general.

The Australia and New Zealand Hyperbaric Medicine Group (ANZHMG), a standing sub-committee of the South Pacific Underwater Medicine Society, is the local specialist group of the medically qualified providers of HBOT. Because, at times, our activities are confused with those of 'alternative' practitioners, this statement is issued in order to allow third parties to accurately identify and characterise the form of therapy being offered to them.

Definitions

Whilst not universally accepted in the current confusion of terms in this area, the definitions below will serve as a practical and workable means by which to distinguish the practice of HBOT from the various forms taken under the umbrella term 'mild hyperbaric therapy'.

HYPERBARIC OXYGEN THERAPY (HBOT)

The Undersea and Hyperbaric Medical Society (UHMS) is the leading world body representing practitioners in this area and defines HBOT as: "*A treatment in which a patient breathes 100% oxygen while inside a treatment chamber at a pressure higher than sea level pressure (i.e., >1 atmosphere absolute or Ata)*".[1]

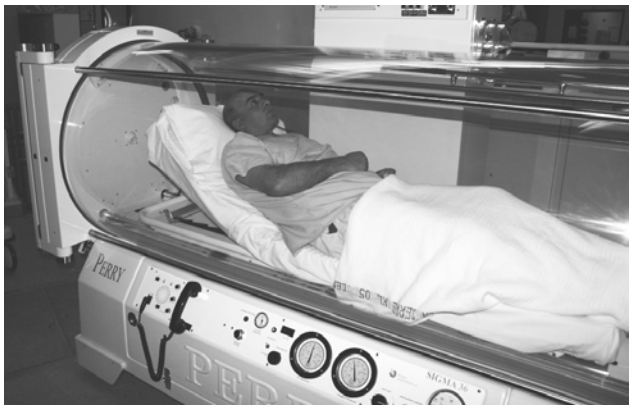
The treatment chamber referred to is an air-tight vessel variously called a hyperbaric chamber, recompression chamber or decompression chamber, depending on the clinical and historical context. Such chambers may be capable of compressing a single patient (a monoplace chamber) or multiple patients and attendants as required (a multiplace chamber) (Figures 1 and 2). These chambers typically operate at pressures above 202.6 kPa (2 Ata) for periods of 60 to 120 minutes for each session of treatment, with the patient breathing 100% oxygen.

MILD HYPERBARIC THERAPY (MHT)

There are many definitions and each individual practitioner or retailer tends to develop their own variant. One compromise definition that covers almost all of this activity is "*a*

Footnote: For a simple pressure conversion chart to assist with interpretation of different pressure measurements in this document, please refer to the appendix.

Figure 1
A monoplace chamber (Prince of Wales Hospital)



treatment, usually administered in an inflatable portable chamber, in which a patient breathes air or oxygen-enriched air at pressures between 1.2 and 1.5 Ata (slightly higher than sea level pressure)".

MHT is often delivered in a 'shop-front' facility, usually under the supervision of a non-medical person, but the chambers can be hired or purchased for use at home. While any hyperbaric chamber is capable of delivering MHT, most MHT is delivered in vessels constructed specifically for this purpose. These chambers are usually built of pliable material and are easily transported and inflated at the point of treatment. One such vessel is illustrated in Figure 3.

What are the important differences between HBOT and MHT?

While constructed of different materials, the differences in the type of compression vessel are less important than the pressure that can safely be generated inside.

MHT implies low pressure therapy: almost always less than 151 kPa (1.5 Ata), while HBOT, although possible at any pressure above 101.3 kPa (1 Ata), is almost universally

Figure 3
An inflatable chamber suitable for the administration of MHT (photo by Bruce McKeeman)

Figure 2
A chamber designed to treat multiple patients (The Karolinska Institute, Stockholm; photo by Peter Kronlund)

delivered at between 203 and 304 kPa (2.0–3.0 Ata).

The most important difference, however, is that during HBOT the patient breathes 100% oxygen in order to deliver greatly increased oxygen pressure to the target tissues in the body; far more oxygen than can be delivered in any other way. On the other hand, MHT is delivered with air, or air mixed with added oxygen at low pressures such that, although oxygen pressures are higher than breathing air alone at sea level pressure, they do not exceed the pressure of oxygen that can be given by the administration of 100% oxygen at 101.3 kPa (1 Ata). For example, in most Australian hospital-based hyperbaric facilities, the standard treatment for a chronic, non-healing foot ulcer in a diabetic patient involves breathing 100% oxygen at 243 kPa (2.4 Ata). Therefore, each breath taken contains oxygen at a partial pressure approaching 243 kPa (1,824 mmHg) and the arterial oxygen pressure will reach something around 203 kPa (1,500 mmHg).

In contrast, a typical MHT session will involve pressurisation to 131 kPa (1.3 Ata) breathing 30% oxygen for about one hour. Under these conditions, each breath has an inspired oxygen pressure of 40 kPa (296 mmHg) and the arterial pressure is likely to reach a more modest 30 kPa (230 mmHg). This is the same oxygen pressure that can be attained by breathing about 35% oxygen at sea level. To put it another way: this amount of oxygen can easily be achieved without the use of the chamber at all.

There are many well-proven effects of increased oxygen levels in the blood and tissues. The administration of oxygen outside a chamber is a very common and familiar treatment in any healthcare system. There is, however, very little evidence indeed that mild compression while breathing oxygen-enriched (to a modest degree) air is any more useful than oxygen alone in a slightly higher concentration at ambient pressure; as in the example above. The latter is certainly much cheaper and more widely available. The

Table 1
ANZHMG accepted indications for hyperbaric oxygen therapy; these indications are reviewed annually. At the time of writing, the ANZHMG proposes that, after review of the evidence, the indications below are appropriate.

Broad indication	Specific indication
Bubble injury	Decompression illness
	Arterial gas embolism (diving/iatrogenic/misadventure)
Acute ischaemic conditions	Compromised flaps/grafts
	Crush injury/compartment syndrome
	Reperfusion injuries
	Sudden sensorineural hearing loss
	Avascular necrosis
Infective conditions	Clostridial myonecrosis
	Necrotizing fasciitis non clostridial
	Myonecrosis necrotizing cellulitis
	Malignant otitis externa
	Refractory mycoses
	Refractory osteomyelitis
	Intracranial abscess
Radiation tissue injury	Osteoradionecrosis
	established
	prophylactic
	Soft tissue radiation injury
	established
	prophylactic
Problem wounds	Chronic ischaemic problem wounds
	Diabetic: ulcers/gangrene/post surgical
	Non-diabetic problem wounds:
	pyoderma gangrenosum
	refractory venous ulcers
	post-surgical problem wounds
Toxic gas poisoning	Carbon monoxide poisoning:
	moderate/severe
	delayed sequelae
Ocular ischaemic pathology	Cystoid macular oedema
	Retinal artery/vein occlusion
Miscellaneous	Thermal burns
	Bells palsy
	Frostbite
Adjuvant to radiotherapy	Adjunct to radiotherapy in treatment of solid tumours

proponents of MHT claim that in addition to the extra oxygen, the mild compression has some benefit in oxygen delivery that remains unexplained and unproven.

What are HBOT and MHT used for?

As suggested above, HBOT is a legitimate therapy prescribed and administered in a hospital or specialised clinic setting under the direction of a medical doctor. There is an increasing body of evidence to support the use of HBOT in a range of serious medical conditions. Those for which the ANZHMG believes there is sufficient evidence to justify routine clinical use are summarized in Table 1.

A useful publication on the evidence for the major indications for HBOT can be purchased from the UHMS web site (<www.uhms.org>).[1] Much information is freely available on the internet. For example, all the randomised trial evidence is summarised at <www.hboevidence.com>, and a detailed examination of many of the indications listed in Table 1 may be found in a doctoral thesis linked from the front page of the same site.[2–12]

The uses for which MHT has been advocated are much wider and this therapy is often offered along with a suite of 'natural' therapies, massage and lifestyle advice. It would be a very difficult task to locate all the claims made for MHT, but Table 2 lists some of those offered in a collection of several internet web site advertisements.

There is very little if any evidence that MHT (or indeed HBOT) has meaningful beneficial effects for the great majority of these indications. For many indications (see Table 2), there has simply been no objective investigation of potential benefit and any such claim is either entirely speculative or based on personal experience. For others, there is good evidence that HBOT and MHT do not positively affect these conditions. For the remainder, the clinical evidence is unclear.

The ANZHMG is not aware of convincing evidence for the effectiveness of MHT for any indication listed in Table 2 and, therefore, does not agree that MHT has any place as a therapeutic modality. Medical science is a process of

Table 2
Summary of proposed indications and evidence for mild hyperbaric therapy
(RCT = randomized controlled trial; Cochrane review = formal systematic analysis of all randomized trials)

Broad indication	Specific indication	Notes on evidence
Paediatric neurological disorders	Cerebral palsy	RCT evidence indicates no difference MHT versus HBOT Not tested against oxygen alone Wide agreement there is no therapeutic effect[2,3]
	Autism spectrum disorder	RCT evidence of more improvement in MHT group (but actual outcome measured was not different) Not tested against oxygen alone Benefit unlikely but possible[4,5]
	ADHD/ADD	No formal evidence
Injury healing	Surgical trauma	No formal evidence
	Traumatic brain injury	Acute – Cochrane review suggests no established benefit for HBOT[6] No formal evidence for MHT Chronic – RCT underway for HBOT
Nervous system dysfunction	Multiple sclerosis	Cochrane review shows no benefit for HBOT[7] No formal evidence MHT
	Parkinson's disease	No formal evidence
	Chronic fatigue syndrome	No formal evidence
	Stroke	Cochrane review suggests no benefit in acute stroke from HBOT[8]
	Altzheimer's disease	No formal evidence
	Optic neuritis	No formal evidence
	Headache and migraine	Cochrane review suggests benefit from HBOT and 100% oxygen at 1 ATA[9]
Infections	Sinusitis	No formal evidence
	Osteomyelitis	Some poor comparative evidence for HBOT, nil for MHT
	Human immunovirus (HIV)	Poor evidence from case series for HBOT
	Lyme disease	Poor evidence for HBOT only
Enhanced immunity	No specific claim	
Skin disorders	No specific claim	
Athletic performance	Enhanced performance	Conflicting evidence for HBOT
	Muscle stiffness	Cochrane review shows that HBOT does not improve post-exercise stiffness[10]
	Improved strength	Low-grade evidence is conflicting for HBOT
	Improved recovery	
	Strengthened heart and lungs	Not tested
Arthritis	(type not specified)	No evidence
Cancer	Basal cell carcinoma	No formal evidence
	Various unspecified	HBOT may enhance radiotherapy[11]
Wellbeing	Relieving tension and stress	No formal evidence for any claims
	Improving cognitive function	
	Detoxifying the blood	
	Retard aging	
	Improving sleep pattern	
	Improving digestion	

hypothesis testing and modification of our understanding. The use of HBOT for many of these indications is under active investigation and it is likely that some individual indications will be shown to be appropriate at some future date whilst others will not.

Conclusion

Oxygen is a very useful and efficacious drug in a wide range of medical conditions. MHT does not typically provide more available oxygen to the body than oxygen administration at one atmosphere, and there is no known therapeutic benefit of mild compression alone. It is therefore difficult to understand how MHT might have therapeutic benefits.

MHT is advocated for a wide range of clinical conditions, in particular for chronic neurological conditions and as part of a suite of 'wellbeing' therapies.

The ANZHMG is not aware of any reliable clinical evidence for therapeutic benefit from mild hyperbaric therapy and does not recommend its use for any medical purpose.

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Submitted: 24 April 2010
Accepted: 25 April 2010

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Appendix

Pressure conversion chart; note the term 'atmospheres absolute' is used to emphasise the use of total pressures including the air pressure at sea level: thus 1 Ata = 760 mmHg = 101.3 kPa = sea level pressure

Atmospheres (Ata)	Kilopascals (kPa)	mmHg	Metres' seawater (msw)
1.0	101	760	10.07
1.2	121	912	12.08
1.4	141	1604	14.10
1.6	162	1216	16.11
1.8	183	1368	18.13
2.0	203	1520	20.14
2.2	223	1672	22.15
2.4	243	1824	24.17
2.6	263	1976	26.18
2.8	284	2128	28.20
3.0	304	2280	30.21

Health risk management in the Tasmanian abalone diving industry

David Smart

Key words
Occupational diving, occupational health, risk management, health, diving industry, abalone

Abstract

(Smart D. Health risk management in the Tasmanian abalone diving industry. *Diving and Hyperbaric Medicine*. 2010;40(2):83-7.)
Risk management is a systematic process applied to all aspects of diving operations. The process aims to reduce accidents and adverse outcomes to a minimum. Risk results from a combination of probability and consequence, and where this combination has major or extreme impact, the risk should not be tolerated. Over the four years 2001–2004, the incidence of decompression illness amongst abalone divers in Tasmania was 1.4 cases per 100 divers per year. Risk management in diving encompasses medical fitness, education and training, dive planning, equipment and maintenance, emergency procedures and equipment, and continual vigilance to remedy new risks as they are identified. There is still much to achieve in the Tasmanian abalone diving industry in all areas of risk management.

Introduction

Contrary to popular belief, diving is a remarkably safe occupation and this level of safety is improving with enhanced levels of training of participants in the activity. In figures derived from amalgamated data from the professional and recreational industries, serious incidents occur approximately 1:10,000 to 1:20,000 dives, and the death rates have been estimated at 1:95,000 to 1:200,000 dives.[1] Between 10 and 20 divers die each year in Australia, and this compares with a national annual death toll due to road trauma of nearly 3,000.[2,3] In the year to 30 June 2003, there were 262 cases of decompression illness treated in Australia and 17 (6.5%) of these were in Tasmania.[4]

Over the four years 2001–2004, 56 divers were treated for decompression illness (DCI) at Royal Hobart Hospital. Of these 56, 16 were recreational scuba divers (all with training), 17 were recreational 'hookah' surface-supplied breathing apparatus divers (10 untrained, five trained and two unknown), 12 were employed divers from the aquaculture industry, seven were professional abalone divers and four others. Thus, abalone divers make up 12.5% of all divers treated. The Tasmanian Government restricts the total number of abalone licences to 125. Based on this restriction, the incidence of DCI is, therefore, 1.4 cases per 100 divers per year. The relative incidence for other groups is unknown, because the total numbers of divers and dives are unknown, and have not been studied formally. It is not possible to completely eliminate risk from diving; we are dealing with a biological animal (the diver) in a hostile and frequently changing environment.

Risk management

Risk management is a systematic approach to improving safety and reducing adverse incidents, and the principles can be applied to almost any process or activity.[5] Risk management is covered by Australian and New Zealand Standard 4360.[6] The process of risk management identifies the risks specific to an industry and assesses their potential impact; the risks are then mitigated. As part of the process, systems are needed to ensure that previously treated risks do not return, and that further risks are monitored.

Risk is a product of probability and consequence. Probability is the chance that an adverse event will occur. Consequence is the impact of the adverse event on the diver. The higher the probability and the worse the consequence, the greater the health risk to the diver. Risk management aims to reduce adverse health events from diving to as low as possible whilst maintaining productivity. In particular, divers should aim to completely prevent events that have catastrophic short- or long-term consequences.

This report provides a medical perspective of risk management in abalone diving, focusing on how risk management principles may be applied to improve diving safety and maintain health of divers. By applying the basic principles of risk management to diving practice, the majority of abalone divers should be able to complete a 30–40 year career in the industry and retire from diving in good health without disability.

A medical perspective of risk management in diving

Based on the experience of diver morbidity treated at the Royal Hobart Hospital (RHH), a medical perspective is provided below under eight broad headings.

1. MEDICAL FITNESS TO DIVE

There is no doubt that occupational divers need to maintain optimum physical health. It is a physically demanding occupation in a potentially hostile environment. Annual medical assessment of fitness is required under Australian Standard 2299.1.[7] Unfortunately, based on the author's observations, only a fraction of abalone divers comply with

the AS2299.1 recommendation for annual diving medical assessments. An equally important principle is that divers take responsibility for their own day-to-day fitness to dive. It goes without saying that many long-term health issues result from individual choices regarding consumption of alcohol, tobacco and other drugs. In abalone divers, long-term health problems from ear and sinus barotrauma are commonly encountered by diving physicians. Time spent in the short term recovering from such conditions is well spent, rather than 'soldiering on', thus causing permanent hearing impairment or sinus injury.

Divers are encouraged to seek early advice from a diving medicine specialist if they experience health problems after diving. The most common clinical syndrome of DCI resembles a bout of influenza: tiredness and lethergy, inability to concentrate, headache and non-specific migratory muscle and joint pains. Occasionally there may be nausea and vomiting. Musculoskeletal pains are common and may be restricted to one joint, most frequently the shoulder, or develop in multiple joints. Skin rashes occur on rare occasions. Other non-neurological symptoms include chest pain, shortness of breath and abdominal pain. Neurological syndromes can range from minor paraesthesiae, numbness and slight unsteadiness, through to paraplegia, hemiplegia, severe cognitive deficits and even loss of consciousness and seizures. Any of these symptoms and signs may be worsened by ascent to altitude (>300 m) after diving; a significant issue in Tasmania (see below).

Early treatment of diving-related illness results in faster and more complete recovery. It is recognised that earlier treatment of DCI results in better outcomes for the diver. For serious neurological DCI, recompression treatment is even more time-critical. In Tasmania, there is a 24-hour diving emergency contact via the Ambulance Tasmania 000 number. The diving medicine specialist is contacted once the alarm is raised, and provides input at the earliest stage to management and transport of the diving casualty. In the majority of cases, divers are treated in the hyperbaric chamber within four hours of an emergency call. Early treatment also prevents long-term sequelae of diving, such as bone necrosis.

2. EDUCATION AND TRAINING

Industry-specific education and training is an essential process supporting diving safety. Well-trained divers have the skills and knowledge to recognise and prevent hazards, and respond to emergencies. In Tasmania, all abalone divers undergo training in accordance with the Tasmanian Abalone Industry Code of Practice, and this code outlines many risk management procedures.[8] This training constitutes a minimum entry platform from which to launch an abalone-diving career. From a medical perspective, additional training beyond the basic minimum is always an advantage, as is the revision of skills, particularly in the area of diver rescue and management of emergencies. Because diving accidents are infrequent, divers and their tenders are at risk of deskilling if emergency procedures are not revised and practised regularly. The divers' tender is an integral part of the diving team, and has great responsibility in supporting the diver. The current code of practice requires that tenders possess an up-to-date first-aid certificate that includes an oxygen therapy course. However, there is no clear process by which currency in first-aid skills is monitored. In addition, there does not appear to be any requirement for rescue training for divers or tenders, or training regarding the specific aspects of administration of 100% oxygen to the injured diver. In many situations, the tender is alone on board the dive boat. Whilst the probability of needing to rescue an incapacitated or unconscious diver from the water is low, the consequence of a delay in rescue, or rescue in a vertical position could be catastrophic. It is doubtful whether, currently within the industry, rescue drills and oxygen administration are practised regularly.

3. DIVE PLANNING AND EMERGENCY PROCEDURES

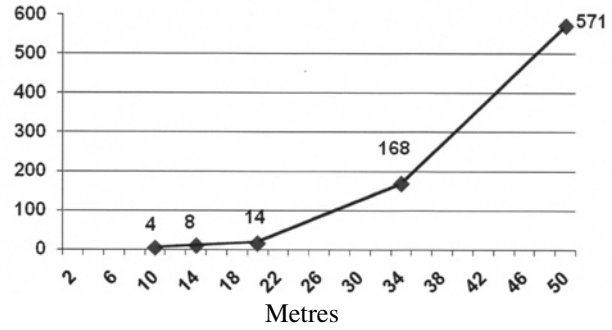
Planning of the dive is an essential part of risk management. There are several areas that have impacted on the health of Tasmanian abalone divers in recent years. One of the most common problems experienced by abalone divers requiring recompression at RHH is failure of the surface air supply, resulting from compressor malfunction or severance of air hoses (usually due to boat propellers). This forces the diver to undertake an emergency ascent to the dive boat, leading to DCI. At present, emergency bail-out air cylinders with regulators and contents gauges are mandated only for dives deeper than 15 metres' seawater (msw).[8] It is the author's opinion that bail-out air supply should be required during all abalone diving, regardless of depth. In an out-of-air situation, this simple risk-management procedure allows the diver to undertake a controlled ascent, thus preventing a potentially fatal rapid ascent in a state of extreme stress. Gas embolism with neurological deficit has resulted from depths as shallow as 2 msw.

The planning process must also consider the remoteness of the dive location, since greater degrees of self-sufficiency will be required for remote locations. Divers should be in peak physical health when diving in remote areas. Emergency equipment, procedures and links to emergency assistance and recompression facilities must be checked and tested prior to departure. Supplies of oxygen must be sufficient to provide continuous treatment of an injured diver for the full return distance from the most remote site, with a 50% reserve. Emergency contact numbers should be checked. Remote diving also mandates greater conservatism in diving practice to reduce the risk of accidents.

4. DIVE PROFILES

Deep diving poses an independent health hazard for all divers (Figure 1). Where possible, abalone divers should maintain

Figure 1
Risk of decompression sickness per 10,000 dives versus depth of dive for controlled dives in hyperbaric chambers.[9]



depths shallower than 20 msw. The no-decompression line is not an equal risk line and risk increases as divers descend deeper than 20 msw. The data in Figure 1 are based on 25,164 chamber dives at the no-decompression limit; and the risk of decompression sickness increased significantly with depth.[9] Deeper diving has also been associated with higher risk of dysbaric osteonecrosis. The effect of depth is compounded by repetitive dives and short surface intervals, due to greater nitrogen loads in the 'fast' tissues such as the brain and circulation, and higher bubble loads in the body. Hookah diving at depths greater than 20 msw creates potential problems of adequate air volume delivery, because of the increased ambient pressure.

Strategies to reduce risk in the dive-planning phase include:

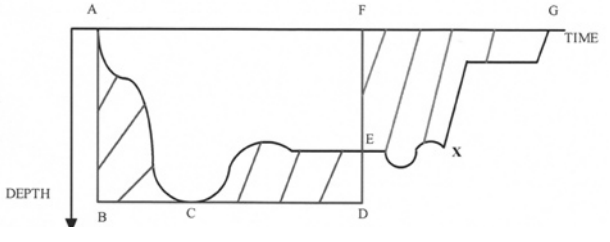
- *Table limits*: Ensure that the tables or the computer schedules are adhered to, and keep inside table limits. US Navy tables dived to the limit have a predicted 5.6% decompression illness rate, whilst that of the DCIEM tables is approximately 0.5%.[10–12] The DCIEM tables are now backed by thousands of hours of human diving data, measuring decompression stress using Doppler ultrasound, and are used by most professional diving operations in Australia, including the Royal Australian Navy, and all hyperbaric facilities.
- *Ascent rates*: In many studies, rapid decompression is associated with greater bubble formation.
- *Surface intervals*: Plan for surface intervals of at least two hours. This allows significant off-gassing of nitrogen from the body, because of its exponential removal from tissues. Repetitive dives at closer intervals have been shown to increase the risk of DCI, as demonstrated with dives on the *HMAS Swan* in Western Australia.[13]
- *Dive computers*: Computers have become very useful tools to assist recreational and professional divers.[14] The advantages of computers are that they travel with the diver and are able to precisely monitor multi-level dive profiles. Many Tasmanian abalone divers now use a computer to track their dives. Computers provide immediate feedback on ascent rates using alarms, and also guidance on repetitive dive schedules. Computers have limitations, in that the models under which they operate have not been researched as thoroughly as 'square dive profiles' (e.g., DCIEM tables).

With multi-level diving, computers provide credit for time not spent at the deepest depth, permitting longer dives. This is demonstrated in Figure 2. The areas enclosed by ABCDE represent a safety margin created by not following a precise square dive profile. In this dive profile, the computer allows extra dive time EXG by the credit given for not spending time at maximum depth, ABCDE. Hence, if the computer is dived to the limit, there is no safety factor left in the dive time. If something goes wrong at point X (e.g., a rapid ascent), then the diver is placed at greater risk than they would be with a dive time limit based on a square dive profile for the deepest point of the dive.

In hyperbaric chamber tests with repetitive diving, dive computers appear to operate less conservatively than dive tables.[15] Divers also need a backup plan using easily accessible, printed dive tables should their computer fail. It goes without saying that the same computer should be used for the same diver, every dive, day after day, so that it accurately tracks all of the diver's in-water activities. The situation is potentially more risky if decompression diving is undertaken because this deliberately exceeds the no-stop limits determined by the tables. Dive computer algorithms are largely untested in terms of risk for decompression diving. Decompression diving carries an exponential increase in risk, and an advanced knowledge of dive tables is needed. Decompression procedures are referred to in the code of practice but lack sufficient detail to be workable. The author has observed that diving for longer than recommended table limits still occurs without the use of appropriate decompression schedules. This is associated with an excessive degree of risk and is not recommended.

- *Bounce diving*: Multiple ascents at rates exceeding 18 msw per minute pose an independent risk factor for DCI. When limits for bounce diving were placed upon Tasmania's aquaculture industry, there was a significant reduction in decompression illness.[5,16]
- *Ascent to altitude after diving*: Based on the diving exposures regularly undertaken by abalone divers, flying

Figure 2
Hypothetical dive profile showing square dive limits ABCDF versus multi-level computer dive ABCEXG



after abalone diving should be avoided for a period of 48 hours. Ascent to altitudes less than 2,400 m after diving should also be limited in accordance with the Australian Standard 2299.1 (2007) (Table 1).[7] Because of the extreme nature of abalone diving, ascent to altitudes greater than 300 m should be avoided for 12 hours. This is of serious practical importance in Tasmania where many of the roads traverse hills or mountainous regions (Figure 3). There are limited data on the safety of ascents to altitudes of 300–2,400 m, and a conservative approach is advised.

- *Nitrox diving*: Nitrox diving using oxygen concentrations greater than air (e.g., 32% or 40%), may reduce the risk of DCI, but only if dived using air tables. When dived to the limits of the equivalent air depths, it is unlikely to be safer. In practice, given the cost and logistic issues of remote area diving, it is unlikely to be useful in the abalone industry.

5. DIVING EQUIPMENT AND MAINTENANCE

The Tasmanian Abalone Industry Code of Practice outlines recommended maintenance schedules, but not in accordance with AS 2299.1, and there are no recommendations regarding frequency of maintenance. Unfortunately the code of practice does not even refer to the Australian Standard 2299.1, instead referring simply to "Australian Standards".[8] In the author's opinion, this is a major omission and constitutes a significant area of risk for the industry. Australian Standard 2299.1 (2007) is the default reference for all professional diving operations, and abalone divers should be fully conversant with its contents. The need to carry functioning, well-maintained bail-out cylinders while diving, and rescue/oxygen equipment in the boat is emphasised again.

Table 1
Recommended time intervals after diving before ascent to altitude (Australian Standard 2299.1)[7]

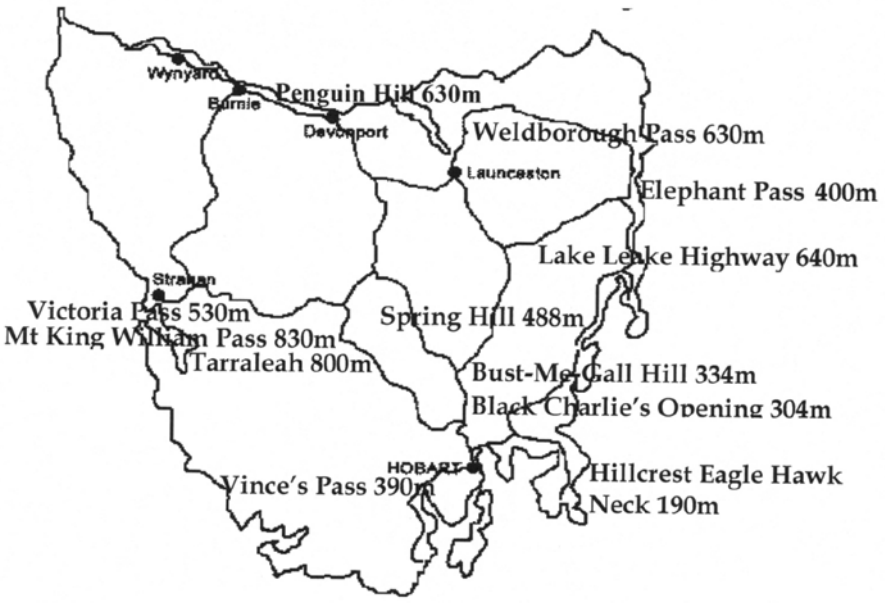
Altitude (m)	Time after last dive (h) Category of dive		
	1	2	3
0–150	Nil	Nil	2
150–600	Nil	2	12
600–2,400	12	24	48
>2,400	24	48	72

Category 1: Single dive to 50% of no-decompression limits, with no decompression or repetitive dives in previous few days.
Category 2: Routine no-decompression diving; single decompression dives
Category 3: Multiple decompression dives; extreme exposures; omitted decompression

6. EMERGENCY EQUIPMENT

Administration of 100% oxygen is essential for all diving accidents. Abalone diving is frequently undertaken in remote areas, considerable distances away from assistance. The average diver breathes up to 15 litres per minute when receiving 100% oxygen. The Australian D-sized oxygen cylinder contains approximately 1,400 litres, providing just over 90 minutes' endurance at this rate. In remote-area diving risk assessment, quantities of oxygen should be carried to ensure an injured diver can receive 100% oxygen until rescued, allowing for a worst-case scenario. Sufficient oxygen should be carried for all diving, because an episode of gas embolism is a possibility from any depth.

Figure 3
Maximum elevations on roads from dived areas of Tasmania



7. TRANSPORT OF THE INJURED DIVER

The goals of pre-hospital management are to provide treatment with 100% oxygen and to transport the diver to a hyperbaric chamber for recompression as quickly as possible without causing deterioration in their condition. The mode of transport of patients with serious diving illness needs to take into account factors such as the distance to the nearest chamber, available resources such as transportable recompression chambers, aircraft and helicopters, road ambulance and access to the sick diver. For road transport, detailed knowledge of road routes from the dive locations to the chamber is also required, because even hills higher than 300 m may result in worsening of the diver's condition (Figure 3). Air transport should not be used unless the aircraft can be pressurised to sea level. The choice of systems depends on the severity of the injury and consideration of local resources and geography. Once a call is made for emergency assistance, this is best left to medical specialists and paramedics directly involved in the incident to determine what is needed.

8. RECORDING OF INCIDENTS AND 'NEAR MISSES'

Industry-wide anonymous incident reporting has proven useful in identifying risks in other diving industries, and allows a systematic approach to remedying any problems identified. The opportunity exists for the Tasmanian abalone industry to set up an incident-reporting system to assist with risk management.

Summary

Risk management is a systematic process applied to all aspects of diving operations. The process aims to reduce accidents and adverse outcomes to a minimum. Risk results from a combination of probability and consequence, and where this combination has major or extreme impact, the risk should not be tolerated. Risk management in diving encompasses medical fitness, education and training, dive planning, equipment and maintenance, emergency procedures and equipment, and continual vigilance to remedy new risks as they are identified. There is still much to achieve in the Tasmanian abalone diving industry in all these areas.

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Submitted: 24 May 2010
Accepted: 01 June 2010

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Historical article (SPUMS Journal. 1981;11(Suppl):4-7.)

John Miller's article brings back fond memories of my first year at Duke, when the FG Hall Lab was abuzz with preparations for the Atlantis II dive. John generously listed me as a 'Principal Investigator', although, along with Bret Stolp, I was very much a trainee, under the mentorship of John Salzano and Enrico Camporesi. We four planned the assessment of the divers' cardiorespiratory responses to exercise. We were able to obtain the first ever blood gas measurements at such great depths, revealing reasonable gas exchange despite a 17-fold increase in gas density. Under such conditions, I am still in awe that the human lung works at all!

Richard Moon, Duke University, Durham, North Carolina

THE DUKE UNIVERSITY 2132 FOOT DIVE, 1980

John N. Miller

Duke University Medical Center, Durham, North Carolina USA

The story of ultra-deep diving is also the story of the High Pressure Nervous Syndrome. Before 1970, the High Pressure Nervous Syndrome was known as the helium tremors or the helium shakes. Some people believed in it, notably in the Royal Navy. Others disbelieved its existence, notably in the US Navy. Since then, the High Pressure Nervous

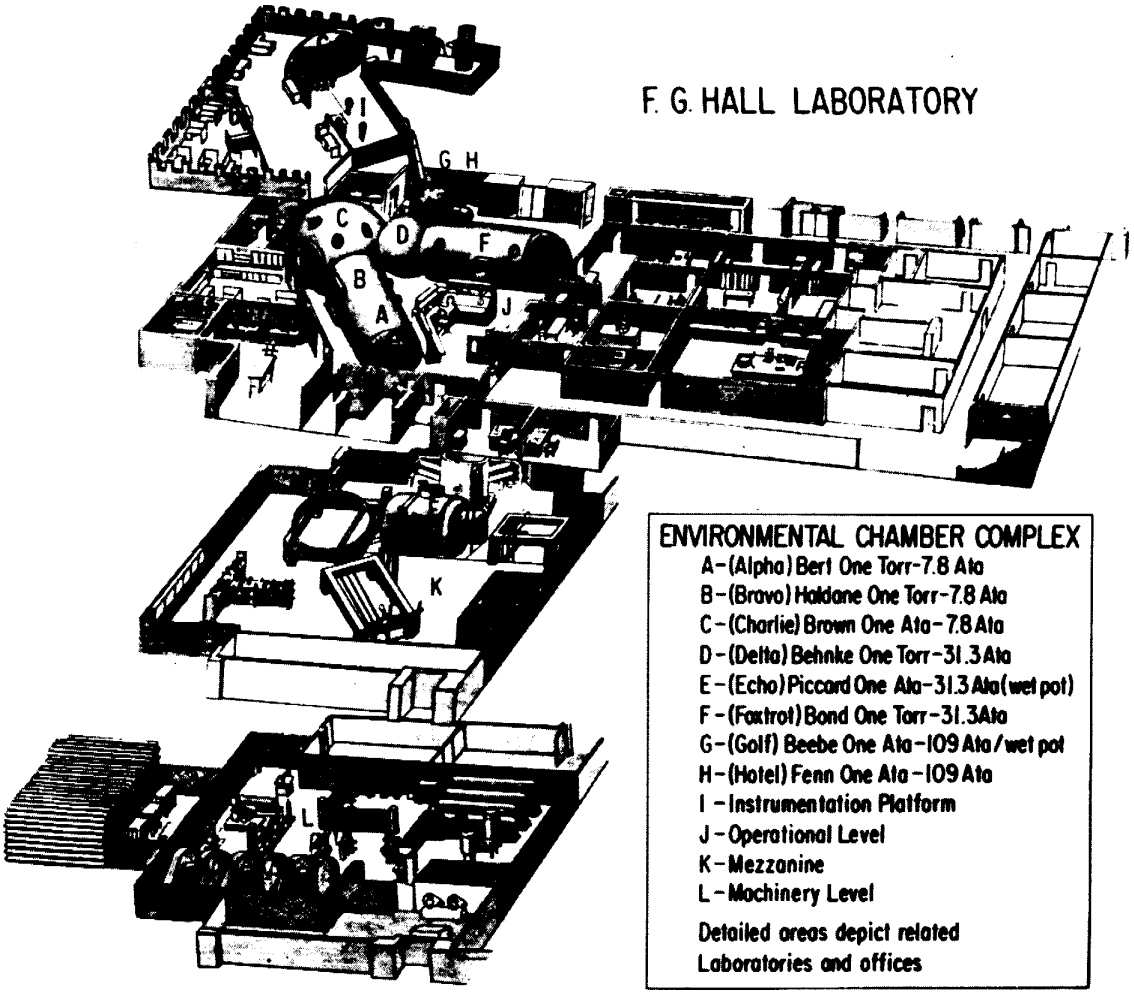

FIGURE 1

EXPLODED VIEW F.G.HALL LABORATORY, DUKE UNIVERSITY MEDICAL CENTER, DURHAM, NORTH CAROLINA

Syndrome has been widely described in all aspects of deep heliumoxygen diving. It is associated with gross abnormalities of the electro-encephalogram, severe tremors, micro-sleep (when divers appear to drift off to sleep and wake up a few moments later), gross discoordination, nausea, vomiting and a marked incapacity to work. All dives deeper than about 1300 feet have been largely limited by the High Pressure Nervous Syndrome. The Atlantis series of dives at the F.G.Hall Environmental Laboratory at Duke University was designed to explore the effects of adding nitrogen to the helium-oxygen mixture to offset the effects of the High Pressure Nervous Syndrome.

Before describing the Atlantis series of dives, I will take you on a brief tour of the F.G.Hall Laboratory (Fig. 1). The facility consists of four levels. The main deck being where the chamber complex itself lies. A small study area and computer bay is on the top. Surrounding the main deck are office space and further laboratories. In the basement are the compressors, gas mixing manifolds, machine shop and electronic workshop. A large helium store is located outside the building. The chambers form a rather massive V-shaped complex around the main control console. A smaller deep chamber mated to one corner of the V has its own control console. Underneath is a cylinder that can be filled with water for wet dives, and a vertical cylinder attached to a sphere that can also be filled with water. The A and B chambers are rated to a maximum pressure of 7.8 atmospheres absolute (ATA) or 230 feet. The large sphere C, also rated to 7.8 ATA and 20 feet in diameter, was originally built as a surgical chamber. Chambers D and K are rated to 1000 feet. The G and H chambers, including the vertical cylinder are rated to 3600 feet.

The facility is used for a wide variety of different purposes. At the present time in A chamber, a wind tunnel is used for studying the metabolism of birds flying at altitude. Chambers B and C are currently used for patient treatments with hyperbaric oxygen. Chamber D and the wet compartment K, are used to develop operational decompression procedures for a new closed circuit, mixed gas, under-water breathing apparatus under a US Navy contract. The G and H chambers are used primarily for deep dives. During decompression on very deep dives when we get back to 1000 feet, the subjects transfer to the D and F chambers where there are showers and toilets, and much more space.

Fig. 2 shows the pressure-time profile for the first of the Atlantis series. It was a dive to 460 metres, 1509 feet, breathing a mixture that consisted of 0.5 ATA oxygen, 5% nitrogen and the balance helium. The 5% nitrogen was designed to offset the effects

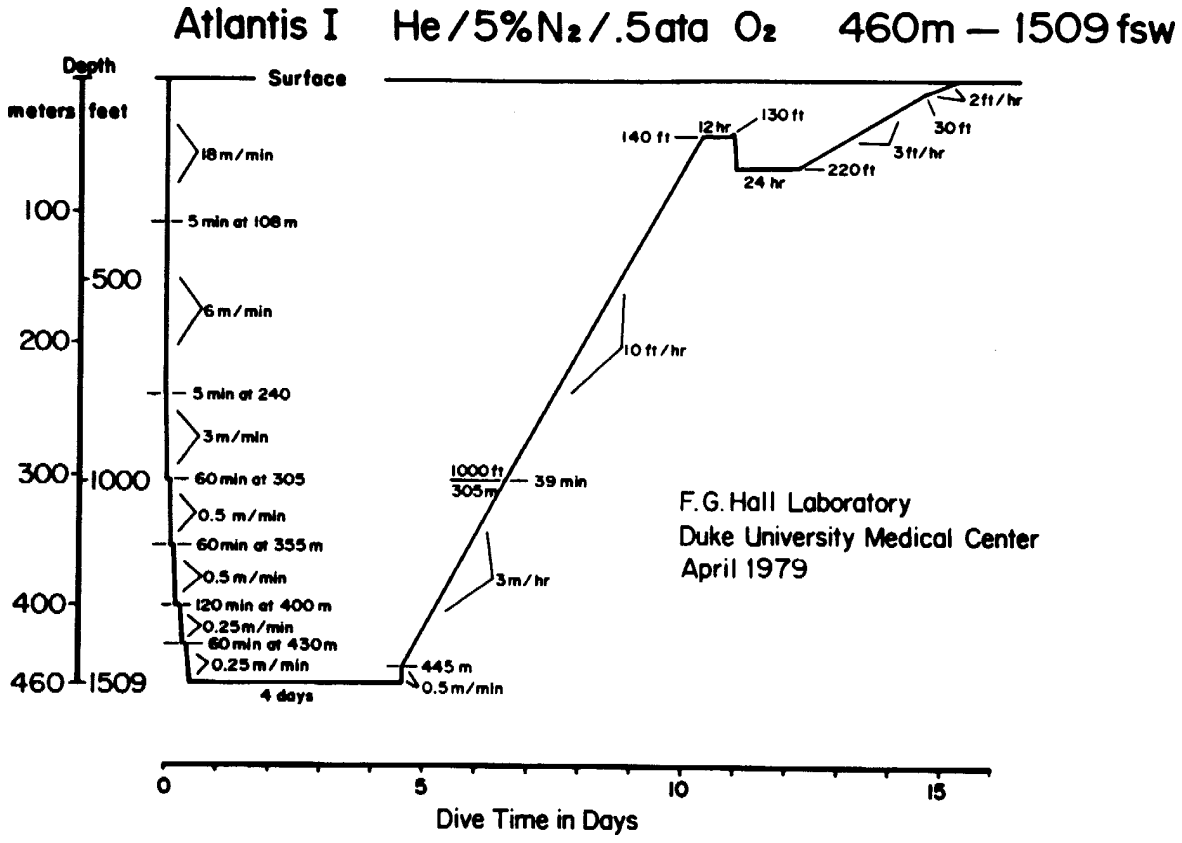

FIGURE 2

PRESSURE/TIM PROFILE ATLANTIS I: 460 METRE DIVE IN F.G.HALL LABORATORY CHAMBERS

of the High Pressure Nervous Syndrome. Over a large series of dives it has been found necessary to decrease the rate of compression as depth increases. One can make a very rapid descent to 1000 feet, and then a progressive slowing of the compression rate is required in each of a number of steps with a smaller bite taken at each pressure increment as the depth increases. This is more obvious in the pressure-time profile for the second dive of the Atlantis series. The total compression rate was 12 hours and 20 minutes. In the Comex 2001 feet dive, heliox with no nitrogen added was used and hence the compression rate was very much slower. Our divers spent 4 days on the bottom at 460 metres doing a wide variety of studies. These included motor and coordinated performance studies, significant physical exercise riding a bicycle ergometer and gas exchange studies, which included the placement of arterial catheters for the measurement of arterial blood gases. In the early stage of the decompression, we made a fundamental error by a relatively rapid ascent from 460 to 445 metres. That was a sufficient pressure decrease to generate enough gas bubbles so that during the continuing stages of the decompression, the subjects rode on the edge of decompression sickness. Ultimately, all three subjects developed "bends" symptoms at around 140 feet. It is quite customary for divers to have fleeting niggling pains all over their bodies during saturation decompressions. However, we felt that this was a little more severe than usual, and yet there was nothing that we could effectively treat without significantly modifying the decompression profile. It is important to note that the whole dive was experimental: not only were we studying compression rate, gas mixture and performance capability on the bottom, we were also studying the decompression approaches necessary to return from extreme depths. Therefore, we decided not to treat until frank symptoms developed, which they did at 140 feet. We initially held at 140 feet until the symptoms disappeared. With another 10 feet of pressure reduction, there was a return of symptoms. These were then treated by minimal recompression and a 24 hour hold. Finally, with a slower decompression rate there was an uneventful decompression to the surface.

How effective was 5% nitrogen in ameliorating the High Pressure Nervous Syndrome? Because of the fairly rapid compression rate, symptoms were marked during the first 24 hours at 460 metres. It was apparent that the 5% nitrogen was insufficient. Nevertheless, there was blocking of the symptoms, and the subjects were sufficiently coordinated particularly after the first 24 hours on the bottom, to place arterial catheters. They were able to perform moderately heavy exercise and all of the performance studies but at a significantly reduced level: there were changes in their electro-encephalograms, some transient nausea and vomiting, some minor tremors, and short periods of micro-sleep, particularly in the first 24 hours during adaptation. However, there was no marked incapacity. Although there was evidence of High Pressure Nervous Syndrome present, there was also evidence that some of it had been blocked with 5% nitrogen.

Atlantis 2 was designed to study a 10% nitrogen mixture with no change in the other gases at a similar bottom depth of 460 metres. Our subjects at 460 metres, in contrast to those in Atlantis 1, breathing a mixture of 0.5 ATA P02, 10% nitrogen, and the balance helium showed absolutely no signs of the High Pressure Nervous Syndrome. The situation at 460 metres looked as though we had simply closed the chamber door and pressurised the gauge, with the chamber remaining at surface pressure.

In the Atlantis 1 dive, another limiting factor was a curious form of breathlessness. It was not density related, nor related to exercise. It was seen mainly at rest, and particularly during eating. All subjects noted a degree of anxiety about taking mouthfuls of food for fear that their breathing would become uncoordinated. They ate less and lost weight. This observation has been made on other dives, and it was true also on the 2001 feet Comex dive. With 10% nitrogen at the same depth, the subjects' appetites remained normal with no apparent dyspnoea, either during exercise or at rest.

Because of the total lack of abnormality, we decided, with the concurrence of our subjects, and of course with the concurrence of our Clinical Investigation Committee, to go deeper. The divers had spent 5 days at 460 metres and were enthusiastic to go deeper. We did so in a series of steps. At 2000 feet our subjects, in excellent condition, wanted to go deeper still. We decided to stop at 650 metres (2132 feet). At 650 metres, there was no evidence of impaired appetite. To mark the occasion, it being Spring, we sent in some daffodils from outside. Again, the divers performed magnificently.

Inside a sphere 8 feet in diameter it is cramped. With the bunks folded in the corners, a table almost occupies the floor space. The table also has to be folded when they raise the hatch to drop down into the vertical chamber below. Despite these inconveniences, they were able to perform a reasonable level of useful work between 100 and 150 watts on the bicycle ergometer in the vertical chamber. They operated a complex array of equipment, taps, bags and hoses, the whole thing being very carefully orchestrated, the bag filling and emptying timed correctly in order to get very accurate gas analysis of the expired gas mixture. Again, blood samples were taken for analysis.

All the performance studies were a little reduced from normal, but no more than 8 to 10% and not as much as they had been with 5% nitrogen at 460 metres on the previous dive. Two of the subjects on this dive in fact, had been subjects on the first dive. The third subject was provided by Oceaneering International as had been the third subject on the first dive.

The profile of the Atlantis 2, 650 metre dive (Figure 3) had the same rapid compression rate to 1509 feet (460 metres) with a 5 day stay at that depth for the detailed series of studies. Then the progressive

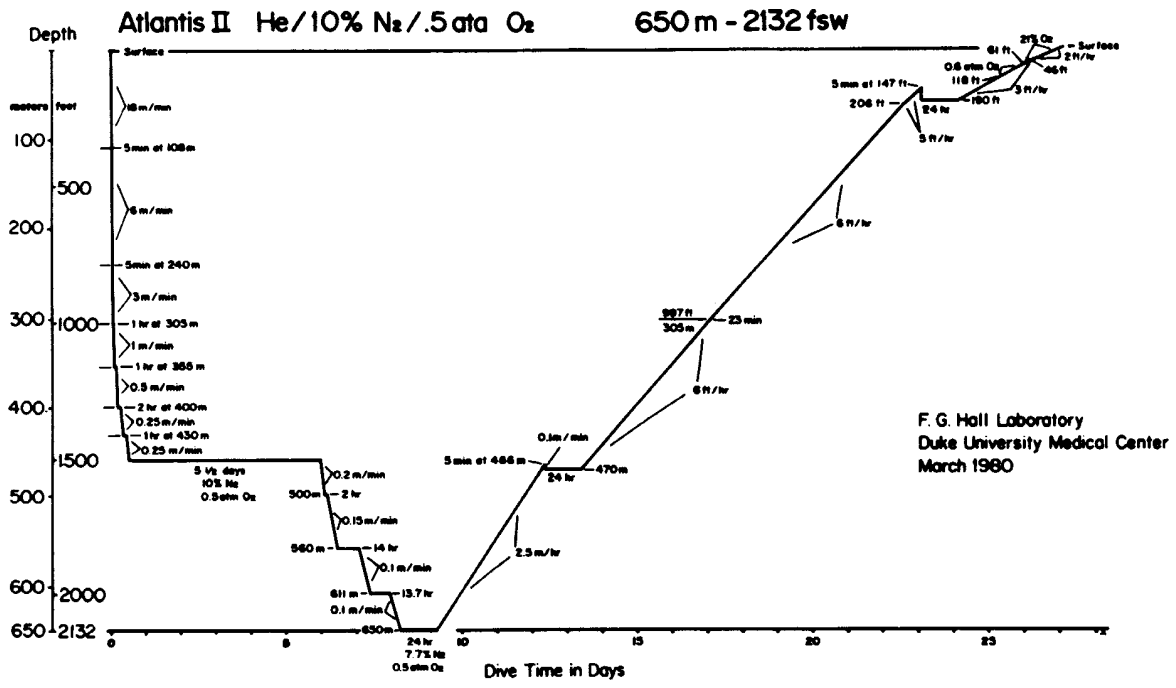

FIGURE 3

PRESSURE/TIME PROFILE ATLANTIS II: 650 METRE DIVE IN F.G.HALL LABORATORY CHAMBERS

BEEBE (C), FENN (H), BEHNKE (D) AND BOND (F)

feeling out of the increments in depth as we gradually compressed further to a maximum of 650 metres. We

elected to go no deeper than 650 metres largely for logistical reasons. We only had a limited amount of helium. Certainly, we could have obtained more, but if we had we would have overspent the budget by far too much. We were concerned about the increase in density with a
10% nitrogen mixture at 650 metres, so the same nitrogen partial pressure was maintained as at 460 metres rather than percentage. Consequently there was a fall to 7.7% nitrogen from 460 metres to 650 metres thus maintaining approximately the same gas density of 16.8 grams per litre as at 460 metres.

The same decompression rate without the initial pull that we had used on the previous dive, was employed with the expectation that at some point it would give trouble. Unfortunately, causing decompression sickness to some extent is inherent in the development of decompression tables. At 470 metres one of our divers developed the deepest decompression sickness in the world. It was adequately treated with a hold for 24 hours and an augmentation of the oxygen partial pressure for a short period of time. The rest of the decompression continued at much the same rate as it had previously, until again reaching the 150 to 200 feet region. Again, there were similar problems to those previously encountered on the decompression in Atlantis 1. Luckily also, the “bends” were easily treated.
This decompression gave us enough information to plan a slower more conservative decompression rate in future dives, and have now (1981) been successfully carried out without such problems in our most recent dive to 2250 feet, Atlantis 3.

Dives of this magnitude cannot be performed other than as a highly coordinated team effort. Therefore it is entirely fitting to list the primary personnel involved in the planning and execution of these dives.

Principal Investigators
Judith Andersen, M.D. Peter Bennett, Ph.D
Enrico Camporese, M.D. David Harris, Ph.D.
Richard Moon, M.D. John Salzano, Ph.D.
Richard Vann, Ph.D.

Dive Physicians
Arthur Dick, M.D. John Miller, M.D.

Divers
William Bell M.D.
Stephen Porter
Delmar Shelton

Dive Supervisors
Owen Doar
William Greeman
Robert Schumacher

Continuing professional development

CME activity 2010/2

Inert gas narcosis and gaseous anaesthesia

Michael Bennett

Accreditation statement
To complete a course successfully, 80% of questions in each quiz must be answered correctly. Activities published in association with *Diving and Hyperbaric Medicine* (DHM) are accredited by the Australia and New Zealand College of Anaesthetists Continuing Professional Development Programme for members of the ANZCA Diving and Hyperbaric Medicine Special Interest Group under Learning Projects: Category 2 / Level 2: 2 Credits per hour.

Intended audience
The intended audience consists of anaesthetists and other specialists who are members of the ANZCA SIG in Diving and Hyperbaric Medicine. However, all subscribers to DHM may apply to their respective CPD programme co-ordinator or specialty college for approval of participation.

Objectives
The questions are designed to affirm the takers' knowledge of the topics covered, and participants should be able to evaluate the appropriateness of the clinical information as it applies to the provision of patient care.

Faculty disclosure
Authors of these activities are required to disclose activities and relationships that, if known to others, might be viewed as a conflict of interest. Any such author disclosures will be published with each relevant CPD activity.

Do I have to pay?
All activities are free to subscribers.

Practitioners are referred to the article in this issue (Smith CR, Spiess BD. The two faces of Eve: gaseous anaesthesia and inert gas narcosis) and the relevant chapter (9.2) in *Bennett and Elliott's physiology and medicine of diving*, 5th edition, for a discussion relevant to the exercise below.

Answers should be posted by e-mail to the nominated CPD co-ordinator (for members of both SPUMS and the ANZCA Diving and Hyperbaric Medicine Special Interest Group, this will be Assoc. Prof. Mike Bennett, <M.Bennett@unsw.edu.au>. On submission of your answers, you will receive a set of correct answers with a brief explanation of why each response is correct or incorrect. Successful undertaking of the activity will require a correct response rate of 80% or more. Each task will expire within 24 months of its publication to ensure that additional, more recent data have not superceded the activity.

Key words
MOPS (maintenance of professional standards), inert gas narcosis, nitrogen narcosis, anaesthesia

Question 1: Which of the following is not a gas that displays anaesthetic or narcotic properties?

A. Nitrogen
B. Nitrous oxide
C. Nitric oxide
D. Isoflurane
E. Xenon

Question 2: Concerning the possible mechanisms involved in the anaesthetic action of gases...

A. The critical membrane hypothesis proposes that gases get into and contract the lipid components of the cell membrane such that signalling in the central nervous system is impaired.
B. The most likely site of action is on nerve conduction along the axon.
C. The high pressure neervous syndrome (HPNS) is a common manifestation of impending anaesthesia because of disinhibition of higher centres.
D. The phenomenon of pressure reversal of anaesthesia supports the lipid phase of the cell membrane as the site of action.
E. The demonstrated linear relationship between anaesthetic potency and pressure supports the lipid/membrane as the site of action.

Question 3: Concerning the multisite expansion hypothesis...

A. Anaesthesia may be produced by expansion of more than one molecular site and each site may have a different set of physical properties.
B. The hypothesis is supported by the similarity of the response to low temperatures and high pressures.
C. The hypothesis begins with the assumption that pressure and anaesthetic agents are acting at the same molecular site.
D. The protein phase of the cell membrane is unlikely to be involved because there is no evidence that anaesthetic agents alter membrane permeability to ions.
E. This hypothesis is incompatible with the hypothesis that effects on a protein target are crucial to the onset of anaesthesia.

Question 4: Concerning xenon gas...

A. In common with most general anaesthetics, xenon has been shown to enhance the inhibitory activity of $GABA_A$ receptors.
B. The MAC of xenon is 20%, making it five times more potent than N_2O as an anaesthetic.
C. Xenon is insufficiently potent to act as a sole anaesthetic agent at 1 ATA.
D. Xenon is a greenhouse gas and should be recycled to keep it out of the atmosphere.
E. Inhibition of the NMDA receptor by xenon may explain the analgesic and amnesic effects of this gas.

Question 5: Which of the following has not been reported as sign or symptom of inert gas narcosis?

A. Increased risk-taking behaviour
B. Unconsciousness
C. Engorgement of the corpus cavernosum
D. Amnesia
E. Impairment of the ability to perform mathematical calculations

Critical appraisal

HBOT did not improve exercise performance immediately following hyperbaric oxygen exposure

Bottom line
No evidence found to suggest improved athletic performance after hyperbaric oxygen therapy.

Citation
Rozenek R, Brennan FF, Banks JC, Russo AC, Lacourse MG, Strauss MB. Does hyperbaric oxygen exposure affect high-intensity, short-duration exercise performance? *J Strength Cond Res*. 2007;21(4):1037-41.

Lead author's name and e-mail:
R Rozenek, < rrozenek@csulb.edu>

Three-part clinical question
Does hyperbaric oxygen exposure acutely improve exercise performance?

Search terms
Exercise, sport

The study
Double-blinded, concealed, randomised, controlled trial with intention-to-treat.

The study patients
Physically active males

CONTROL GROUP
(*n* = 9 analysed) Normobaric air for 1 h at 1.2 ATA.
EXPERIMENTAL GROUP
(*n* = 9 analysed) 100% O_2 for 1 hr at 2.0 ATA.

The evidence
See Table 1.

Comments
1 Small study with low power to show important differences.
2 Subjects were not high-performance athletes.
3 Paper actually describes two small trials with no benefit shown in treadmill or bench-press performance.

Appraised by: Michael Bennett, Adam Perczuk; Tuesday 17 June 2008
E-mail*: <m.bennett@unsw.edu.au>*

Key words
Hyperbaric oxygen, exercise, performance, research, critical appraisal

Source
<www.hboevidence.com>

Table 1
Exercise performance outcome for hyperbaric oxygen exposure versus sham treatment

Outcome	Control group		HBOT group		Difference	95% CI
	Mean	SD	Mean	SD		
Time to fatigue (s) (treadmill)	84.4	20.0	77.3	14.7	7.1	-10.4 to 24.6
Heart rate post exercise (treadmill)	173	12	172	16	1.0	-12.9 to 14.9
Rating of exertion (/20) (treadmill)	18.3	0.9	17.9	1.2	0.4	-0.6 to 1.5

Abstracts reprinted from other sources

An experimental study of the use of hyperbaric oxygen to reduce the side effects of radiation treatment for malignant disease

Williamson RA

Hyperbaric oxygen (HBO) has been used for more than 20 years to assist wound healing in the treatment of the more severe complications associated with the side effects of therapeutic radiation treatment. A prospective study was performed in an irradiated rat model to determine whether HBO is effective in reducing the long-term side effects of therapeutic radiation treatment. The experimental model was designed to simulate a fractionated course of therapeutic radiation that is commonly used in the treatment of cancer of the mandible. One week following completion of the radiotherapy, the animals underwent a four-week course of HBO treatment and two animals from each group were killed at eight-week intervals until the end of the experiment at 36 weeks. Histological sections of tissue clearly showed continued growth of teeth and maintenance of specialised tissues, such as salivary gland and bone, in the treated group compared to the non-treated group. This experiment model demonstrated that HBO is effective in reducing the long-term side effects of therapeutic radiation treatment in normal tissue, when given one week after the completion of the radiation treatment.

Reproduced with kind permission from the International Association of Oral and Maxillofacial Surgeons: Williamson RA. An experimental study of the use of hyperbaric oxygen to reduce the side effects of radiation treatment for malignant disease. *Int J Oral Maxillofac Surg*. 2007;36:533-40.

Key words
Hyperbaric oxygen, hyperbaric research, radiation, side effects, reprinted from

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Early hyperbaric oxygen therapy for reducing radiotherapy side effects: Early results of a randomized trial in oropharyngeal and nasopharyngeal cancer

David N Teguh, Peter C Levendag, Inge Noever, Peter Voet, Henrie van Der Est, Peter van Rooij, et al

Departments of Radiation Oncology, Maxillofacial Surgery, Otorhinolaryngology and Head and Neck Surgery, Biostatistics of Erasmus Medical Centre - Daniel den Hoed Cancer Centre, Rotterdam, The Netherlands; and The Institute for Hyperbaric Medicine, Rotterdam, The Netherlands

Purpose: Comparison of quality of life (QoL) and side effects in a randomised trial for early hyperbaric oxygen therapy (HBOT) after radiotherapy (RT).

Methods and materials: From 2006,19 patients with tumours originating from the tonsillar fossa and/or soft palate (15), base of tongue (1), and nasopharynx (3) were randomised to receive HBOT or not. HBOT consisted of 30 sessions at 2.5 ATA (15 msw) with oxygen breathing for 90 minutes daily, five days per week, applied shortly after the RT treatment was completed. As of 2005, all patients received validated questionnaires (i.e., The European Organisation for Research and Treatment of Cancer [EORTC] QLQ-C30, EORTC QLQ Head and Neck Cancer Module (H&N35), performance status scale): before treatment; at the start of RT treatment; after 46 Gy; at the end of RT treatment; and two, four and six weeks and three, six, 12, and 18 months after follow up.

Results: On all QoL items, better scores were obtained in patients treated with hyperbaric oxygen. The difference between HBOT versus non-HBOT was significant for all parameters: EORTC H&N35 swallowing ($P = 0.011$), EORTC H&N35 dry mouth ($P = 0.009$), EORTC H&N35 sticky saliva ($P = 0.01$), PSS eating in public ($P = 0.027$), and pain in mouth (visual analogue scale; $P < 0.0001$).

Conclusions: Patients randomised for receiving hyperbaric oxygen after the RT had better QoL scores for swallowing, sticky saliva, xerostomia and pain in mouth.

Reprinted with kind permission from Elsevier: Teguh DN, Levendag PC, Noever I, Voet P, van Der Est H, van Rooji P, et al. Early hyperbaric oxygen therapy for reducing radiotherapy side effects: Early results of a randomized trial in oropharyngeal and nasopharyngeal cancer. *Int J Radiat Oncol Biol Phys*. 2009;75(3):711-6.

Key words
Hyperbaric oxygen, radiotherapy, malignancy, head & neck, side effects, reprinted from

Does hyperbaric oxygen therapy reduce radiotherapy side effects and improve quality of life in oral and oropharyngeal cancer?

The effects of radiation treatment on the hard and soft tissues in the management of malignancies of the head and neck are well known and often produce distressing symptoms, such as xerostomia, dysphagia, pain and particularly the problem of osteoradionecrosis (ORN) of the jaws. Patients having hyperbaric oxygen therapy (HBOT) in the management of ORN of the jaws often report an improvement in saliva flow and taste perception, which has led to the suggestion that HBOT given after initial radiotherapy might have some protective effect and, therefore, improve quality of life (QoL) for such patients.

An animal study by Williamson demonstrated that HBOT is effective in reducing the long-term side effects of therapeutic radiation treatment in normal tissues, when given one week after the completion of the radiation therapy.[1] Histology showed maintenance of specialised tissues such as salivary gland and bone, as well as continued growth of teeth, in the HBO-treated group compared to the non-treated group.

In a randomised trial for early HBOT after radiotherapy for oropharyngeal cancer, Teguh et al showed better scores for QoL in patients treated with hyperbaric oxygen, and significant differences for all parameters of swallowing, dry mouth, sticky saliva, eating in public, and pain in the mouth, in patients treated with HBOT versus non-HBOT.[2] Similarly, Gerlach et al observed a reduction of swallowing problems, a subjective improvement of xerostomia and an improvement in sense of taste in 21 patients receiving HBO after radiotherapy for oral or oropharyngeal carcinoma.[3]

Whilst these studies are limited by relatively small numbers and it is yet to be determined the optimal commencement of HBOT after radiation therapy and the ideal numbers of treatments, results are very encouraging in relation to QoL and should demand a large randomised trial to answer these questions.

In the meantime, our patients are our best measure of the success of this modality of treatment.

References

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2 Teguh DN, Levendag PC, Noever I, Voet P, van Der Est H, van Rooij P, et al. Early hyperbaric oxygen therapy for reducing radiotherapy side effects: Early results of a randomized trial in oropharyngeal and nasopharyngeal cancer. *Int J Radiat Oncol Biol Phys.* 2009:75(3):711-6.
3 Gerlach NL, Barkhuysen R, Kaanders JHAM, Janssens GORJ, Sterk W, Merkx MAW. The effect of hyperbaric oxygen therapy on quality of life in oral and oropharyngeal cancer patients treated with radiotherapy. *Int J Oral Maxillofac Surg.* 2008:37:255-9.

Mr Leslie Snape
Maxillofacial Surgeon
Christchurch Hospital, New Zealand

Key words
Hyperbaric oxygen, radiotherapy, malignancy, head & neck, side effects

Review of diver noise exposure

Anthony TG, Wright NA, Evans MA
QinetiQ Ltd, Hampshire, UK

Divers are exposed to high noise levels from a variety of sources both above and below water. The noise exposure should comply with *The Control of Noise at Work Regulations 2005* (CoNaWR05, 2005). A detailed review of diver noise exposure is presented, encompassing diver hearing, noise sources, exposure levels and control measures. Divers are routinely exposed to a range of noise sources of sufficiently high intensity to cause auditory damage, and audiometric studies indicate that diver hearing is impaired by exposure to factors associated with diving. Human hearing underwater, in cases where the diver's ear is wet, is less sensitive than in air and should be assessed using an underwater weighting scale. Manufacturers of diving equipment and employers of divers have a joint responsibility to ensure compliance with the exposure values in the CoNaWR05, although noise is only one hazard to a diver, and a balanced risk assessment must be applied to the whole diving operation. A diver noise-reduction strategy is proposed, and a health surveillance programme involving audiometric tests for divers should be established.

Reprinted with kind permission from Anthony TG, Wright NA, Evans MA. Review of diver noise exposure. *Underwater Technology*. 2010;29(1):21-39.

Key words
Occupational diving, occupational health, hearing, injuries, environment, reprinted from

Editor's comment

This report is part of a larger research report (*RR735 - Review of diver noise exposure*) funded by the United Kingdom Health and Safety Executive (the full report available at <http://www.hse.gov.uk/research/rrhtm/rr735.htm>). This paper is available from Ingenta at: <http://www.ingentaconnect.com/content/sut/unwt/2010/00000029/00000001/art00003>. Physicians undertaking commercial diving medical assessments should familiarise themselves with this article since hearing loss is one of the commonest medical problems that present in employed divers.

Letter to the Editor

Continuing Professional Development Programme (ANZCA)

Dear Editor,

Here are the details of the Continuing Professional Development Programme (CPD) points that have been approved by the Australian and New Zealand College of Anaesthetists (ANZCA) for the SPUMS 39th Annual Scientific Meeting combined with the Asian Hyperbaric and Diving Medicine Association 6th Annual Scientific Meeting held at Redang Island, Malaysia, 23–28 May 2010 (Approval number 1721).

Lecture Type Session: Category 1 / Level 1: 1 credit per hour

Practical Workshop: Category 3 / Level 2: 3 credits per hour

Jan Lehm
Senior Specialist, Department of Diving and Hyperbaric Medicine, Prince of Wales Hospital, Randwick, NSW 2031

Key words
Letters (to the Editor), meetings, MOPS, (maintenance of professional standards)

Book reviews

The future of diving: 100 years of Haldane and beyond

A Smithsonian contribution to knowledge
MA Laing, AO Brubakk, editors

Softcover, 286 pages
ISBN-13:978-0-9788460-5-3
ISBN-10:0-9788460-5-2
Smithsonian Institute of Scholarly Press
Download a PDF at: <www.scholarlypress.si.edu>
Print copies: <schol_press@si.edu>
Print copies of this publication are free on request while supplies last.

When I was asked to review this book, I took it as a welcome opportunity to virtually participate in the meeting from which it originated, a meeting that unfortunately I missed because of unexpected, last-minute complications. The positive expectations I had had about the meeting were fully confirmed by reading the book, which well deserves its title. It is pleasant and informative reading for any specialist or, for that matter, anyone interested in diving medicine.

The book is well structured, in three independent but linked sections, taking the reader along a path that, starting from the origins of the modern scientific basis of decompression theory, leads to its possible future. The list of international contributors reads like a 'Who's Who' of diving and diving physiology – Costantino Balestra, Jean-Eric Blatteau, Alf Brubakk, David Doolette, Zeljko Dujic, Cliff Effedal, David Elliott, Susan Kayer, Michael Lang, Lassie Loevstakken, Andreas Møllerløkken, Richard Moon, George Perdrizet and Russell Richardson.

The foreword, by David Elliott, and the introductory papers offer a different perspective on the John Scott Haldane we in diving medicine are used to thinking of, placing him in a more complex and complete frame as an all-round scientist of his time and an initiator of what is today known as environmental physiology, not simply the 'inventor' of the decompression theories and modalities still in use today. The introductory section of the book closes with an article that bridges the past and future of decompression research, highlighting its environmental physiology nature and the need to monitor and properly measure not only "*phenomena of immediate interest, but of physiology in general*", going beyond the traditional steady-state methods "*to include techniques that can detect and measure transients*".

The second section starts by covering 'classic' aspects of decompression physiology, with comprehensive, though concise, papers on the clinical aspects of decompression illness, and on decompression algorithms, both tissue-gas content and bubble-model based. After these, the reader is led through other essential, current and possible future aspects of decompression physiology and pathophysiology, illustrating the complexities of the biological and functional responses to decompression stress. The possibilities of *"biochemical decompression"* using *"gas-metabolizing"* bacterial strains, endothelial and cellular responses to decompression stress, the possibility that exposure to pressure and decompression cause genetic variation and the role of physical and pharmacological *"preconditioning"* methods to reduce decompression stress are covered in articles by this international range of authors. Another interesting paper in this section was that dealing with the much discussed and still controversial topic of long-term, diving-induced brain damage. In all, this was a very interesting and informative section.

The next section covers (in two articles) the current methods of monitoring decompression and its risks, mainly using ultrasound bubble-detection methods. How to turn all this into a 'diver-friendly' tool is illustrated in an article on the future of dive computers. The subsequent chapters contain lively and interesting discussions on the future of decompression physiology and methodology and the need for more effective recruitment of young researchers into diving and environmental physiology – a topic that, in particular, has concerned Alf Brubakk for a number of years.

The 'pearl' in this book, however, or the 'cherry on the cake' if you wish, and what makes it really worth having, if for no other reason, is a complete reprint of the original 1908 article by Boycott, Damant and Haldane on the prevention of decompression sickness.[1] After more than 100 years, this is still enlightening reading and a 'must know' for every diving medicine specialist.

This is definitely a book to have, not only on the library shelf, but on our desks.

Reference

1 Boycott AE, Damant GC, Haldane JS. The prevention of compressed-air illness. *J Hyg.* 1908;8:342-443.

Alessandro Marroni,
President, Divers Alert Network (DAN) Europe and International DAN

Key words
Diving, decompression, decompression illness, decompression sickness, diving tables, diving theory (see Physiology), history, book reviews

Diving medicine for scuba divers, 3rd edition

Carl Edmonds, Bart McKenzie, Robert Thomas, John Pennefather

e-Book, 347 pages
ISBN: 978-0-646-52726-0
Carl Edmonds; 2010
Available as a free copy at: <www.divingmedicine.info>

Diving medicine for scuba divers was downloaded and reviewed on screen. It was reviewed by two non-medical but DMT-trained, ex-commercial divers now involved in hospital-based diving and hyperbaric medicine.

The authors, three physicians and a physiologist, all worked at the Royal Australian Navy's School of Underwater Medicine in its heyday and have decades of experience in teaching diving medicine to medical practitioners and divers. The third edition of this book is a comprehensive and useful resource.

With enough physics and physiology to provide a sound introduction, the medical aspects of diving are covered in a thorough, interesting and well-explained way. Many of the chapters would surpass information currently available in more specialised DMT manuals. The highlighting of key words in important paragraphs is certainly helpful in absorbing information.

The text is broken into 43 short chapters and several appendices. Background chapters 1–8 deal with history, physics, physiology, breath-hold diving, diving equipment and environments, stress disorders, and female divers. Specific diving diseases (pressure-related diseases) are fully covered in the next 16 chapters. Aquatic diseases, covering drowning, salt-water aspiration, hypothermia, infections and dangerous marine animals, are chapters 25–29. General diving-related medical problems are covered in chapters 30–37, whilst chapters 38–42 look at treatment and prevention, covering medical examination, the first-aid kit, oxygen therapy, training and safety, and a review of resuscitation. The final chapter is a brief look at technical diving. The appendices consist of a diving medical library, emergency contact numbers, an in-water oxygen recompression therapy table and diving emergency (DAN) contacts.

"If a diver were to pass wind in a confined room, all the occupants of the room would soon be aware of the fact but, fortunately, not necessarily the source." On the topic of gas diffusion, this is just an example of the touches of humour that abound, making the book so readable for a lay audience. However, whilst not incorrect, there were several areas in the book about which we would like to offer our comments. Whilst flying and altitude exposure after diving is discussed, we felt this was somewhat vague. We are often posed with the same questions at the hyperbaric unit. Although there are many different situations, and all of them open to conjecture, we would follow the DCIEM recommendation of "whenever possible it is inadvisable to fly above 600 metres in any aircraft within 48 hours of completing any dive. Travelling by vehicle over mountain ranges or hills can expose divers to the same dangers as flying and should be avoided in the same way for 24 hours. If flying after diving is considered essential, flying may be carried out after 24 hours but the increased risk of DCS must be borne in mind."

When dealing with deaths in professional divers, from experience, the highly regulated and safety conscious offshore sector has a relatively low incidence of diving disease fatality compared to traumatic accidents. The authors' quote of 48 deaths per 10,000 divers per year is long out of date; the Health and Safety Executive in the UK safety strategy to 2010 quotes approximately three deaths per 10,000 divers per year for the offshore and inshore sectors.

The authors seem rather sceptical of anything electronic or technical, ranging from dive computers to closed-circuit rebreathers. While we can understand this reluctance to a certain extent, modern computers, when used with the authors' own ten commandments, will prove very safe, particularly for today's multi-level recreational diving. Computers must surely have contributed to the reduction in the incidence of decompression sickness seen throughout Australasia in recent decades.

Nitrox has distinct bottom-time advantages in the 15–30 metre depth range and is widely used in scientific, military, and commercial diving, including saturation. Recreational use of oxygen rebreathers should be limited to an oxygen partial pressure of 1.6 Ata, i.e., 6 metres' sea water maximum. Rebreathers are not for everyone; however, it is another interesting facet of the sport that has made advances in recent years. Reliable oxygen and more recently carbon dioxide monitoring, are enhancing the safety aspects of rebreather diving.

The chapter on ear barotrauma was one of our favourites, with a concise explanation of an often ill-informed subject. The four chapters involving decompression sickness tackle the age-old subject clearly in lay person's terms. The chapter on dangerous marine animals provides a quick reference for the nastiest of the nasty. Oxygen therapy techniques and the resuscitation review provided a good reminder of what we should know.

To provide a free, downloadable copy of this comprehensive resource to the general diving public, in this day and age, is a tremendous educational gift to the diving community from Dr Edmonds and his colleagues. Not all divers are good at reading from a screen and to read the entire text in this way is quite a task. For quick reference to a particular situation, where a computer is available, it will prove very

useful. Even in the field these days, a computer is never far away and a downloaded disc carried on a dive trip could prove very useful.

Astute divers, dive instructors, DMTs and doctors new to diving will find this text worthy of a regular read. Nursing staff at hyperbaric units would find it a great introduction or refresher in the diving medicine field. We found it to be a humorous and well-informed read. We will leave you with the mental image of a heavily over-weighted gentleman sitting on the gunwale of a resort vessel and the accompanying comment: "*Lead poisoning is a common contributor to recreational scuba diving deaths*."

Warren Harper and Trevor Carson
Hyperbaric Technologists, Christchurch Hospital Hyperbaric Medicine Unit, New Zealand

Key words
Scuba diving, physiology, underwater medicine, general interest, book reviews

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Conference time

It was conference time again, time to update and dive.
The gang flocked to Redang to see who's still alive,
For the regulars are aging, less able but more patient
But new faces and new cases prevent them dying ancient.

Ex sat dive and astronaut, Mike Gernhardt, led the speaking
With underwater lessons that guide Moon and Mars' seeking
And Swedish Dr Folke Lind with a chamber extraordinaire
Gave lectures of pictures of intensive hyperbaric care.

Other presentations were varied in their range;
Spinal bends and recent trends, new ideas to interchange;
Shark bites and rebreather sets, lung function, Romberg's sign
Were revisited and elicited along fresh and novel lines.

The great debate was a huge success, 'The Diving Doctor's Dead'.
Speakers posed, weakness exposed, no fault or fact unsaid.
Once the blood was mopped up and handshakes healed the scars,
The count confirmed a large amount still lived and breathe the bars.

The warm Malaysian waters were tropical and clear
So the diving was thriving with the minimum of gear.
Flashes flashed and bubbles blew in each underwater group,
A mismatch of limbs and fins like an acrobatic troupe.

'The convenor' Glen was tireless, a hawkeyed overseer,
Meeting, greeting, seating and always ever near.
All delegates were overjoyed for a conference free of stress.
The gala ball was fun for all to conclude this huge success.

John Parker
<drjohnparker@hotmail.com>

EUBS European Underwater and Baromedical Society

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The EUBS website is at
www.eubs.org
Members are encouraged to log in

EUBS European Underwater and Baromedical Society

EUBS Annual Scientific Meeting 2010

14–18 September 2010
Istanbul (European Cultural Capital 2010)

Venue: The Point Hotel-Barbaros, Istanbul, Turkey

Prof. Maide Cimsit, Istanbul University
Secretary General, EUBS ASM 2010
E-mail: <mcimsit@istanbul.edu.tr>

Istanbul is a centuries-old city, located on the Bosphorus Strait connecting Asia and Europe. It was the capital of three empires: Roman, East Roman (Byzantine), and the Ottoman Empire. Many historic areas are on the UNESCO World Heritage List. Istanbul is unique with its location, cultural and historical heritage, palaces, and monuments, museums and bazaars, blending with modern architecture, shopping centres, and all sorts of restaurants, clubs and friendly wine houses.

The scientific programme will cover a broad spectrum of topics in diving and hyperbaric medicine.
An ECHM Workshop and EDTC meeting will also take place during the meeting.
Full details of the scientific programme and workshops are available on the meeting website:
<http://www.eubs2010.org>

Main Topics

Diving Medicine: Diving physiology, fitness to dive standards, breath-hold diving, handicapped diving, medical aspects of underwater archaeology, diving pathologies, diving technologies.
Hyperbaric Medicine: Infection, wound healing, HBOT in traumatology, burns injury, HBOT in ICU patients, ophthalmologic disorders, sudden hearing loss and HBOT, hyperbaric safety and organization.

For important dates, registration and accommodation and other details please visit the meeting website:
<http://www.eubs2010.org>

The official language of the conference will be English.

Social programme

Details of the many optional activities, including a diving programme, tours and excursions, in the Social Programme may be found on the website or are available from the Congress Secretariat.
We hope that you will enjoy the meeting, and the unique ambience and hospitality of Istanbul.

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Nominations for election as EUBS Executive Committee Member-at-Large 2010

For the term 2010–2013, the following nomination has been accepted by the EUBS Executive Committee. Voting will be done via Internet Ballot; all members of EUBS will receive an e-mail with a voting 'link'.

Jean-Michel Pontier

Born 28 February1968 in Aix-en-Provence (France)

Since 2002 Licensed physician in diving medicine with the French Armed Forces, and working at the Diving School (Ecole de Plongée) in St Mandrier.
2002-2004 St. Anne Hospital hyperbaric centre (Toulon, France). During that period, participated as on-call physician for the hyperbaric centre of the Font-Pré Hospital.
2005 Master's Degree in physiology and extreme environments.
Certified specialist in sports medicine and emergency medicine.
Member, French Society of Diving and Hyperbaric Medicine (MedSubHyp).

Author or co-author of eight peer-reviewed scientific papers and 15 scientific conference presentations on the specific aspects of military diving (more particularly navy 'seals'), military rebreather diving (epidemiological study) and factors increasing the risks for decompression pathology (clinical study).

Participated in the experimental studies demonstrating the protective effect of physical activity on decompression bubble formation.

Currently investigating, for a Master Thesis, the mechanisms of platelet activation during decompression, using an animal model of decompression sickness.

He is a professional diver and mine-clearance diver, certified sports medicine and emergency medicine specialist. He worked at the St. Anne Hospital hyperbaric centre (Toulon, France) from 2002 to 2004 and during that time also acted as on-call physician for the hyperbaric centre of the Font-Pré Hospital.

In 2005 he served as on-board physician for the Clipperton Atoll expedition, together with Dr Jean-Louis Etienne. For this mission, he participated in the elaboration of a therapeutic re-immersion protocol for treating decompression disorders in remote areas.

EUBS General Assembly: invitation and agenda

All EUBS Members are invited to attend the EUBS General Assembly, which will take place during the Annual Scientific Meeting, on 18 September at 1400 h.

Agenda:

1 Approval of minutes of previous GA (see the December 2009 issue of DHM: 239-41.)
2 Status of current Meeting
3 Awards and Grants
4 Financial Report
5 Website Report
6 Journal Report
7 Next EUBS Meetings
8 Miscellaneous

Members who wish to place an item on the agenda, are kindly requested to notify the Honorary Secretary, Joerg Schmutz, and/or another ExCom member in writing (paper or e-mail).

EUBS website news

The EUBS website provides information about the Society and its Executive Committee, the Annual Scientific Meeting, the Corporate Members, and lists research, courses and conferences in hyperbaric and diving medicine. In addititon, on the 'Members Area' pages, EUBS members will find access to:

- the full-text literature database on diving and hyperbaric medicine, provided courtesy of GTUEM
- the EUBS Members Directory
- full text of the *Diving and Hyperbaric Medicine* Journal
- their own membership information and status
- a dedicated private discussion forum

Log in at: <www.eubs.org>

SPUMS notices and news

South Pacific Underwater Medicine Society Diploma of Diving and Hyperbaric Medicine

Requirements for candidates (updated October 2008)

In order for the Diploma of Diving and Hyperbaric Medicine to be awarded by the Society, the candidate must comply with the following conditions:

1 The candidate must be medically qualified, and be a current financial member of the Society.
2 The candidate must supply evidence of satisfactory completion of an examined two-week full-time course in Diving and Hyperbaric Medicine at an approved facility. The list of approved facilities providing two-week courses may be found on the SPUMS website.
3 The candidate must have completed the equivalent (as determined by the Education Officer) of at least six months' full-time clinical training in an approved Hyperbaric Medicine Unit.
4 The candidate must submit a written proposal for research in a relevant area of underwater or hyperbaric medicine, in a standard format, for approval *before* commencing their research project.
5 The candidate must produce, to the satisfaction of the Academic Board, a written report on the approved research project, in the form of a scientific paper suitable for publication. Accompanying this written report should be a request to be considered for the SPUMS Diploma and supporting documentation for 1–4 above.
6 In the absence of documentation otherwise, it will be assumed that the paper is submitted for publication in *Diving and Hyperbaric Medicine*. As such, the structure of the paper needs to broadly comply with the 'Instructions to Authors' – full version, published in *Diving and Hyperbaric Medicine* 2010; 40(2):110-2.
7 The paper may be submitted to journals other than *Diving and Hyperbaric Medicine*; however, even if published in another journal, the completed paper must be submitted to the Education Officer for assessment as a diploma paper. If the paper has been accepted for publication or published in another journal, then evidence of this should be provided.
8 The diploma paper will be assessed, and changes may be requested, before it is regarded to be of the standard required for award of the Diploma. Once completed to the reviewers' satisfaction, papers not already accepted or published in other journals will be forwarded to the Editor of *Diving and Hyperbaric Medicine* for consideration. At this point the Diploma will be awarded, provided all other requirements are satisfied. Diploma projects submitted to *Diving and Hyperbaric Medicine* for consideration of publication will be subject to the Journal's own peer review process.

Additional information – prospective approval of projects is required

The candidate must contact the Education Officer in writing (e-mail is acceptable) to advise of their intended candidacy, and to discuss the proposed subject matter of their research. A written research proposal must be submitted before commencing the research project.

All research reports must clearly test a hypothesis. Original basic or clinical research is acceptable. Case series reports may be acceptable if thoroughly documented, subject to quantitative analysis, and the subject is extensively researched and discussed in detail. Reports of a single case are insufficient. Review articles may be acceptable if the world literature is thoroughly analysed and discussed, and the subject has not recently been similarly reviewed. Previously published material will not be considered.

It is expected that all research will be conducted in accordance with the joint NHMRC/AVCC statement and guidelines on research practice (available at <http://www.health.gov.au/nhmrc/research/general/nhmrcavc.htm>) or the equivalent requirement of the country in which the research is conducted. All research involving humans or animals must be accompanied by documented evidence of approval by an appropriate research ethics committee. It is expected that the research project and the written report will be primarily the work of the candidate, and that the candidate is the first author, where there are more than one.

The SPUMS Diploma will not be awarded until all requirements are completed. The individual components do not necessarily need to be completed in the order outlined above. However, it is mandatory that the research project is approved prior to commencing research.

The Academic Board reserves the right to modify any of these requirements from time to time. As of October 2008, the SPUMS Academic Board consists of:
Associate Professor David Smart, Education Officer
Associate Professor (ret'd) Mike Davis
Associate Professor Simon Mitchell.

All enquiries and applications to the Education Officer:
Associate Professor David Smart
GPO Box 463, Hobart, Tasmania 7001
E-mail: *<david.smart@dhhs.tas.gov.au>*

Key words
Qualifications, underwater medicine, hyperbaric oxygen, research, medical society

Minutes of the SPUMS Executive Committee Meeting 21 November 2009 at Prince of Wales Hospital Hyperbaric Unit, Randwick

Opened: 0930h

Present: M Bennett, S Lockley, J Lehm, G Hawkins, M Davis, D Smart and G Williams

Apologies: S Squires, C Acott and V Haller

1 Minutes of previous meeting

Minutes accepted for Executive Committee Meeting, Snorkelers' Cove Resort, Iririki Island, Vanuatu held 29 May 2009. Proposed Dr M Bennett, seconded Dr G Hawkins, carried.

2 Matters arising from previous minutes

Reviewed.

3 Annual Scientific Meetings

3.1 ASM 2010

3.1.1 Berjaya Resort (Redang) has postponed renovations so can accommodate conference. Some issues that have arisen were discussed including weight restriction of 10 kg on flights to Redang. Resort to arrange transport of all luggage for registrants. In addition, passenger limit of 90 on Berjaya Air flights from Kuala Lumpur and Singapore. Timing of flights to Redang require an overnight stay in Singapore or Kuala Lumpur, with luggage collection from the Berjaya Hotels in these locations. Other option is ferry to Redang.

3.1.2 Delegates to book three components online: conference registration, accommodation at Berjaya Redang Resort with Berjaya Airline flight, then international flights.

3.1.3 Academic programme in development and Dr Hawkins shared current plan. Includes AGM on 27 May 2010. Diving from 0800–1300h. Poster presentations and free papers also included.

3.1.4 Welcome dinner, cocktail party and gala theme dinner are all covered by the conference registration.

3.1.5 Diving and hyperbaric medicine refresher courses on 25 and 27 May, during diving programme.

3.1.6 Budget presented to the Committee – AUD23,920 (fixed costs) and AUD17,995 variable costs. Dives at a cost of AUD28 per dive. Booking for 60 registrants, 50 accompanied and 20 children.

3.2 ASM 2011

Dr Lockley to convene ASM 2011. Discussed theme will cover technical diving, including military and occupational diving medicine. Suggestions for speakers requested and discussed. Intention to actively invite and involve military divers and military diving doctors. Location options being explored. Action: Dr Lockley to report progress planning arrangements to President and Committee.

3.3 ASM 2012

Dr Bennett called for volunteers to comvene ASM 2012. Committee members to direct expressions of interest to the President or Secretary. Action: Committee members to report expressions of interest to the President.

4 Journal matters

4.1 EUBS/SPUMS arrangements discussed. Committee agrees quality of the Journal has improved significantly due to the efforts of the Editor DHM and amalgamation of EUBS/SPUMS.

4.2 EUBS to be formally requested to provide details regarding two-year agreement (EUBS proposal).

4.3 Proposed advertising policy has been sent out to the Committee by the DHM Editor and is accepted. This includes free advertising of "not for profit" organisations, institutions and for courses and meetings. Policy needs further development and the letter will be recirculated by the Editor, with points from Dr Smart.

4.4 Discussed SPUMS/EUBS could endorse that one of two editors be funded to attend EUBS and SPUMS ASMs and this should be factored into journal costs. Committee unanimously agreed.

4.5 Dr Bennett and Neal Pollock were mentioned for their ongoing support of the Journal. The number of manuscripts is now double previous years with a pool of articles awaiting publication.

4.6 Editor suggested the future of the DHM hangs in the balance of approval for Medline citation.

4.7 Recommended by the Editor that EUBS and SPUMS provide the same amount of money for each copy of the DHM. Proposed Dr M Davis, seconded Dr J Lehm, carried.

4.8 Ownership and publisher: current situation is that SPUMS does not own journal due to statement in the Constitution. SPUMS incorporated in Victoria Articles states that we will produce a Journal. Publisher should be through the "Journal Entity" for example "DHM incorporated". Likely that the incorporation journal currently belongs to SPUMS and that it may require a change of ownership to the two societies. Actions: President to explore legal opinion regarding ownership/publisher. Editor to explore options for legal advice.

4.9 Journal costs include: meeting attendances, publication, staffing. Estimation of AUD70,400 equates to AUD20 per issue to cover distribution numbers. Costs outlined for 2009. Action: Dr Lehm to calculate cost per journal and each Society charged per copy received. In Annual Financial Report, true cost of Journal needs to be outlined with costs separated out.

4.10 Discussed possibility of book-keeper being employed, with financial independence of Journal. Await further legal advice as above.

4.11 DHM Editor contract was discussed and has been forwarded via e-mail to the President and Secretary. Action: Executive Committee to resolve contract before end December. President to forward the proposed contracts to the Committee for an opinion.
4.12 Discussed DHM Editorial Board newsletter (e-mail correspondence has been forwarded to the Committee).

5 Website update

5.1 Demonstration of new website by Dr Hawkins. All members will require new passwords for access. Almost completed new website and total cost to date is just over AUD8,000.

6 Education Officer's report

6.1 Accreditation of courses discussed, including SPUMS role in this. Committee agrees SPUMS should have a leading role in accreditation of diving medicine courses.
6.2 Dr Smart recommended a tiered structure to the course accreditation system, similar to the current European system. For example, a candidate must complete requirements for Level 1 prior to progressing to Level 2 and Level 2 prior to progression to Level 3. Action: Dr Smart to further investigate European system.
6.3 Discussed proposal to change requirement of SPUMS Diploma from "must be a financial member of SPUMS for 2 years" to "must be a current financial member of SPUMS".

7 Treasurer's report

7.1 The Treasurer has recommended that the SPUMS Administrator Mr Steve Goble be given approval for purchase of a new laptop and software for the purpose of SPUMS administration including the ASM up to AUD3,000. Proposed Dr J Lehm, seconded Dr M Bennett, carried.
7.2 DHM Editor requires a new printer and will obtain quotations on purchase of a laser printer and provide to the Treasurer. The old printer used by the Editor and the old printer/scanner used by the Secretary are both to be written off as these items are well over five years old and now outdated. Dr Lockley will use her own printer and purchase consumables as required. Proposed Dr M Davis and Dr S Lockley, seconded Dr J Lehm, carried.
7.3 Dr Lehm has suggested opening an on-line savings account at St George Bank, with a higher interest rate, so that a reasonable rate of interest can be earned on SPUMS funds. Proposed Dr J Lehm, seconded Dr M Bennett, carried.
7.4 Proposed that Dr G Hawkins be added as a signatory to all SPUMS accounts. Other current signatories on the accounts will remain unchanged. Proposed Dr J Lehm, seconded Dr M Bennett, carried.

8 Secretary's report

8.1 Update all committee member contact details to <spums.org.au> addresses, and previous e-mail accounts will or have been redirected.
8.2 The training requirements to perform occupational dive medicals in Australia and New Zealand, including current recognised courses, were discussed because of apparent confusion and request from members via e-mail for clarification on this issue. Education Officer confirmed that doctors performing occupational diving medicals must complete one of the SPUMS approved courses or a course accredited by the Education Committee. Currently SPUMS recommend if five years or more has lapsed since a medical practitioner has performed an occupational dive medical or since completion of an approved course, the medical practitioner should re-attend a course prior to performing an occupational dive medical.

9 Other business

9.1 Option for tele/video conferencing committee meetings was discussed. Is a cost-effective option; however, at present not all committee members have access to the technology and this arrangement would be difficult given current circumstances. At present, two meetings per year have been adequate.
9.2 DHM Editor was asked to leave the room while Committee discussed SPUMS funding part of attendance costs of a member of the ANZHMG sub-committee at MSAC. Last meeting attendance in Canberra was approximately AUD1,000. Two further meetings expected in the coming months. Committee has agreed to contribute funds to support attendance. Proposed Dr M Bennett, seconded Dr D Smart, carried.
9.3 Committee to consider formation and be involved in supporting proposition of an international diving and hyperbaric medicine federation as proposed initially by Dr A Brubakk, President EUBS. Proposed Dr M Bennett, seconded Dr D Smart, carried.
9.4 Committee informed that Gordon Bingham received the SPUMS Award of a Book Prize at HTNA.

10 Correspondence

Submission written to Medical Council of New Zealand by Dr M Davis regarding qualifications for practising hyperbaric medicine in New Zealand. Correspondence received in response from Dr Philip Pigon (CEO Medical Council of New Zealand).

11 Next meeting

The next meeting is scheduled on 23 May 2010 at Berjaya Resort, Redang Island, Malaysia.

Closed: 1849h

South Pacific Underwater Medicine Society
40TH Annual Scientific Meeting
Preliminary Notification

24–28 May 2011

Venue: Palau Pacific Resort

Further details will be on the SPUMS website soon:
<www.spums.org.au>

Theme

What's so technical about diving? Medical aspects of military, occupational and recreational technical diving

Guest speakers

Dr David Doolette, PhD
Associate Professor Simon Mitchell, PhD
Dr Andrew Fock, FANZCA

palau pacific resort

Dive & Travel 2008 Winner
BEST RESORT HOTEL
BEST DIVING RESORT HOTEL
Reader's Choice

SPUMS 2011 ASM Convenor:
Dr Sarah Lockley
Secretary SPUMS
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46-50 Kent Rd, Mascot
NSW 2020

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Australian and New Zealand College of Anaesthetists

Certificate in Diving and Hyperbaric Medicine

Eligible candidates are invited to present for the examination for the Certificate in Diving and Hyperbaric Medicine of the Australian and New Zealand College of Anaesthetists.

Eligibility criteria are:

1 Fellowship of a Specialist College in Australia or New Zealand. This includes all specialties, and the Royal Australian College of General Practitioners.

2 Completion of training courses in Diving Medicine and in Hyperbaric Medicine of at least four weeks' total duration. For example, one of:
   a ANZHMG course at Prince of Wales Hospital Sydney, **and** Royal Adelaide Hospital or HMAS Penguin diving medical officers course **OR**
   b Auckland University Diploma in Diving and Hyperbaric Medicine.

3 **EITHER:**
   a Completion of the Diploma of the South Pacific Underwater Medicine Society, including six months' full-time equivalent experience in a hyperbaric unit and successful completion of a thesis or research project approved by the Assessor, SPUMS
   b **and** Completion of a further 12 months' full-time equivalent clinical experience in a hospital-based hyperbaric unit which is approved for training in Diving and Hyperbaric Medicine by the ANZCA.

   OR:
   c Completion of 18 months' full-time equivalent experience in a hospital-based hyperbaric unit which is approved for training in Diving and Hyperbaric Medicine by the ANZCA
   d **and** Completion of a formal project in accordance with ANZCA Professional Document TE11 "Formal Project Guidelines". The formal project must be constructed around a topic which is relevant to the practice of Diving and Hyperbaric Medicine, and must be approved by the ANZCA Assessor prior to commencement.

4 Completion of a workbook documenting the details of clinical exposure attained during the training period.

5 Candidates who do not hold an Australian or New Zealand specialist qualification in Anaesthesia, Intensive Care or Emergency Medicine are required to demonstrate airway skills competency as specified by ANZCA in the document "Airway skills requirement for training in Diving and Hyperbaric Medicine".

All details are available on the ANZCA website at:
<www.anzca.edu.au/edutraining/DHM/index.htm>

Dr Margaret Walker, FANZCA, Cert DHM (ANZCA)
Chair, ANZCA/ASA Special Interest Group in Diving and Hyperbaric Medicine

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The Hyperbaric Research Prize

The Hyperbaric Research Prize encourages the scientific advancement of hyperbaric medicine and is awarded annually whenever a suitable nominee is identified. It will recognise a scholarly published work or body of work(s) either as original research or as a significant advancement in the understanding of earlier published science. The scope of this work includes doctoral and post-doctoral dissertations. The Hyperbaric Research Prize is international in scope. However, the research must be available in English. The Hyperbaric Research Prize takes the form of commissioned art piece and US$10,000 honorarium.

For detailed information please contact:
Baromedical Research Foundation
5 Medical Park, Columbia, SC 29203, USA
Phone: +1-803-434-7101
Fax: +1-803-434-4354
E-mail: <samir.desai@palmettohealth.org>

Erratum

Hyperbaric medicine practice, 3rd edition

Kindwall EP, Whelan HT, editors

The review by Dr Karen Richardson of this textbook was not accompanied by key words for searches. These are listed below.

Key words
Diving medicine, physiology, diving, textbook, book reviews

The Environmental Physiology Group, NNTU, Norway
Man in extreme environments − applied physiology from subsea to space
A symposium to honour Professor Alf O Brubakk and his long research career

Dates: 16–17 December 2010

More information will be available at:
<www.ntnu.no/diving>
or contact <andreas.mollerlokken@ntnu.no>

Scott Haldane Foundation, The Netherlands

The Scott Haldane Foundation is dedicated to education in diving medicine, and has organised over 100 courses in the past few years, both in the Netherlands and abroad.
More information can be found at:
Website: <www.scotthaldane.nl>
E-mail: <info@scotthaldane.nl>

2–9 October: Basic course "Diving medicine for pneumologists"
14–15 October: Advanced course "Evidence-based diving medicine" (Doorn, NL)
6–13 November: Basic course in diving medicine (Zanzibar, Tanzania)
13–20 and 20–27 November: 17th Advanced course in diving medicine (Zanzibar, then Mafia Island, Tanzania)
11 December: Refresher course "Neurology and diving"

Inter-university Diploma in Diving and Hyperbaric Medicine, France

University course (1-year duration) in diving and hyperbaric medicine, organised concurrently by 13 French universities (Angers, Antilles-Guyane, Besançon, Bordeaux II, Lille II, Lyon II, La Réunion, Marseille, Nancy, Nice, Paris XIII, Strasbourg, Toulouse).

For further information go to:
<http://www.medsubhyp.org> or
<http://medecine.univ-lille2.fr/format/diu/hyperbar.htm>

17th International Congress of Hyperbaric Medicine

Dates: 16–19 March 2011
Venue: Cape Town International Convention Centre, Cape Town, South Africa
Link to ICHM: <www.ichm.org>
For further details go to SAUHMA website:
<www.sauhma.co.za>

German Society for Diving and Hyperbaric Medicine (GTUeM)

An overview of basic and refresher courses in diving and hyperbaric medicine, accredited by the German Society for Diving and Hyperbaric Medicine (GTUeM) according to EDTC/ECHM curricula, can be found on the website:
<http://www.gtuem.org/212/Kurse_/_Termine/Kurse.html>

British Hyperbaric Association 2010 Annual Conference

Dates: 18–21 November
Host: East of England Hyperbaric Unit
James Paget University Hospitals NHS
Lowestoft Road
Gorleston Great Yarmouth
Norfolk NR31 6LA

For further information contact:
Karen Turner <karen.turner@jpaget.nhs.uk> or
Maxine Palmer <maxine.palmer@jpaget.nhs.uk>
Phone: +44-(0)1493-453526
Fax: +44-(0)1493-453261

Diving Diseases Research Centre (DDRC), Plymouth, UK

Diving medicine courses for 2010

- Introduction to Hyperbaric Medicine Course for Physicians (UHMS): 13–17 September
- Combined Introduction to Hyperbaric Medicine Course for Physicians (UHMS) and Level I (Medical Examiner of Divers) Course: 13–19 September
- Level I (Medical Examiner of Divers) Course: 17–19 September
- Level IIa (Diving Medical Physician): 20–24 September
- Medical Examiner of Divers, Refresher Course: 25–26 November

For further information: <www.ddrc.org>

The Royal Swedish Navy in cooperation with Sahlgrenska University Hospital, Gothenburg University

Basic course in diving medicine and HBO

Dates: 20 September – 1 October 2010
Venue: Gothenburg, Sweden

For further information:
E-mail: Lena Fridman <lena.fridman@mil.se>

Royal Adelaide Hospital Diving Medicine Medical Officers Course 2010

Week 1, 29 November – 3 December
Week 2, 6 – 10 December

Full DMT Courses:
2nd DMT course in November t.b.d.

For more information contact:
Lorna Mirabelli
Senior Administrative Assistant
Hyperbaric Medicine Unit, Royal Adelaide Hospital
Phone: +61-(0)8-8222-5116
Fax: +61-(0)8-8232-4207
E-mail: <Lmirabel@mail.rah.sa.gov.au>

2010 Royal Australian Navy Medical Officers Underwater Medicine Course

Dates: 25 October – 5 November 2010
Venue: HMAS PENGUIN, Sydney
Cost: to be advised

The course seeks to provide the medical practitioner with an understanding of the range of potential medical problems faced by divers. Considerable emphasis is placed on the contra-indications to diving and the diving medical, together with the pathophysiology, diagnosis and management of the more common diving-related illnesses. The course includes scenario-based simulation focusing on management of diving emergencies and workshop covering the key components of the diving medical.

For information and application forms contact:
Mr Rajeev Karekar for Officer in Charge,
Submarine and Underwater Medicine Unit
HMAS PENGUIN
Middle Head Rd, Mosman, 2088 NSW, Australia
Phone: +61-(0)2-99600572
Fax: +61-(0)2-99604435
E-mail: <Rajeev.Karekar@defence.gov.au>

DIVING HISTORICAL SOCIETY AUSTRALIA, SE ASIA

P O Box 347, Dingley Village
Victoria, 3172, Australia
E-mail:
<deswill@dingley.net>
Website:
<www.classicdiver.org>

Introductory Course in Diving Medicine New Zealand

Dates: 24–27 September 2010
Venue: Navy Hospital, Devonport, Auckland

This course is designed to provide GPs who have an interest in diving medicine with a basic understanding of the principles involved.

RNZCGP approved for 20 hours' CME.

For details and application form please see the website:
<www.navyhyperbaric.mil.nz>

The Australia and New Zealand Hyperbaric Medicine Group Introductory Course in Diving and Hyperbaric Medicine

Dates: 21 February – 4 March 2011
Venue: Prince of Wales Hospital, Sydney, Australia

This course is approved as a CPD Learning Project by ANZCA – Cat 2, Level 2 – 2 credits per hour (Approval No. 1191)

For more information contact:
Ms Gabrielle Janik, Course Administrator
Phone: +61 (0)2-9382-3880
Fax: +61 (0)2-9382-3882
E-mail: <Gabrielle.Janik@sesiahs.health.nsw.gov.au>

Conference proceedings available The future of diving: 100 years of Haldane and beyond

Michael A Laing and Alf O Brubakk, editors
Smithsonian Institution Scholarly Press

The proceedings of "*The Future of Diving: 100 Years of Haldane and Beyond*" symposium, convened 18–19 December 2008 in Trondheim, Norway, by the Baromedical and Environmental Physiology Group of the Norwegian University of Science and Technology, are reported in 28 papers and three discussion sessions.

Download a PDF of this publication through:
<www.scholarlypress.si.edu>
To request a print copy, e-mail SISP at:
<schol_press@si.edu>
Print copies of this publication are free upon request, while supplies last; limit five (5) copies.

Instructions to authors (full version)

(revised May 2010)

Diving and Hyperbaric Medicine, as the combined journal of the South Pacific Underwater Medicine Society and the European Underwater and Baromedical Society, seeks to publish papers of high quality on all aspects of diving and hyperbaric medicine of interest to diving medical professionals, physicians of all specialties, and members of the diving and hyperbaric industries.

Contributions should be sent to:
The Editor, Diving and Hyperbaric Medicine
C/o Hyperbaric Medicine Unit, Christchurch Hospital
Private Bag 4710, Christchurch, New Zealand
E-mail: <editor@dhmjournal.com>

Requirements for manuscripts

Diving and Hyperbaric Medicine welcomes contributions that meet the following requirements:

Original articles and **technical reports** (maximum 3,000 words, plus 30 references)
These articles should be subdivided into the following sections: an **Abstract** of no more than 250 words, **Introduction**, **Methods**, **Results**, **Discussion**, **Conclusions**, **Acknowledgements** and **References**. Acknowledgements should be brief.

Review articles (maximum 5,000 words, plus 60 references); include an **Abstract** of no more than 250 words.

Case reports, **brief reports** and **work-in-progress reports** (maximum 1,500 words, plus 15 references); include an **Abstract** of no more than 200 words.

Educational articles, **commentaries** and **case reports** for '**The diving doctor's diary**', '**World as it is**', '**Opinion**' or **'Historical**' occasional sections may vary in format and length, but should be a maximum of 3,000 words and generally comply with the requirements below.

Letters to the Editor (maximum 500 words, plus 5 references)

Articles not conforming to these instructions will be considered, but require detailed justification for any increase in word count, references cited or change in format.

Inclusion of more than five authors in any one manuscript requires justification.

Documents should be submitted electronically on CD-R or as file attachments to e-mail. The preferred format is Microsoft Office Word 2003. Paper submissions will no longer be accepted.

All articles should include a **title page**, giving the title of the paper and the full names of all authors, their principal qualifications and the positions they held when doing the work being reported. One author must be identified as correspondent, with their full postal address, telephone and fax numbers, and e-mail address supplied.

A **covering letter** from the principal author, acknowledging this as the authors' own work, that they have permission from all co-authors for submission to DHM, that the manuscript is not being submitted to another journal concurrently and declaring financial, commercial, academic or any other conflicts of interest (including potential ones), must be included .

A maximum of five **key words or terms** best describing the paper should be chosen from the list on the journal <www.dhmjournal.com>, SPUMS <www.spums.org.au> or EUBS <www.eubs.org> websites. New key words must be justified and will be used at the discretion of the Editor. Key words should be placed at the bottom of the title page.

Text should be single or 1.5 line-spaced using both upper and lower case. Headings should conform to the current format in *Diving and Hyperbaric Medicine*:
Section heading
SUB-SECTION HEADING 1
Sub-section heading 2

All pages should be numbered, but no other text should appear in the header and footer space of the document. Do not use underlining. No running title is required.

English spelling will be in accordance with the *Concise Oxford Dictionary*, 11th edition revised. Oxford: Oxford University Press; 2006. Editorial assistance will be provided to authors for whom English is not their first language. However, adequate English is a prerequisite for acceptance of the paper.

Measurements are to be in SI units (mmHg are acceptable for blood pressure measurements) and normal ranges should be included where appropriate. Authors are referred to the on-line BIPM brochure, International Bureau of Weights and Measures (2006), *The International System of Units* (SI), 8th ed, available at ISBN 92-822-2213-6 : <http://www.bipm.org/utils/common/pdf/si_brochure_8_en.pdf>, or Baron DN, McKenzie Clarke H, editors. *Units, symbols and abbreviations. A guide for biological and medical editors and authors*, 6th edition. London: Royal Society of Medicine; 2008. Atmospheric and gas partial pressures should be presented in kPa rather than Ata or bar (Ata/bar/mmHg may be provided in parenthesis on the first occasion). Water depths should be presented in metres' sea (or fresh)

water (msw or mfw).

Abbreviations may be used once they have been shown in parenthesis after the complete expression. For example, decompression illness (DCI) can thereafter be referred to as DCI.

References

The Journal reference style is that of the *International Committee of Medical Journal Editors (ICMJE) Uniform requirements for manuscripts submitted to biomedical journals*. Examples of the exact formats for different types of references (journal articles, books, monographs, electronic material, etc) are given in detail on the website <http://www,nlm.nih.gov/bsd/uniform_requirements.html> (updated August 2009).
Specific requirements for *Diving and Hyperbaric Medicine* are:
References should be numbered consecutively in the order in which they are first mentioned in the text, tables or figures as superscript numbers preferably at the end of the sentence **after** the full stop.[1,2]
Up to six authors' names followed by "et al" should be given.
Use MEDLINE abbreviations for journal names. The *List of Journals Indexed for MEDLINE* publication ceased with the 2008 edition. The Journals database: <http://www.ncbi.nlm.nih.gov/sites/entrez?Db=journals&Cmd=DetailsSearch&Term=currentlyindexed[All]> can be used to obtain a list of currently indexed MEDLINE titles.
Titles of quoted books and journals should be in italics.
Capital letters are used only at the start of the article or book title and for proper names.
Avoid using abstracts from meeting proceedings as references.
Verifying the accuracy of references against the original documents is the responsibility of authors.
Personal communications should appear as such in the text and not be included in the reference list, e.g., (Other AN, personal communication, year).

An example journal reference is shown below:
de Bruijn R, Richardson M, Schagatay E. Oxygen-conserving effect of the diving response in the immersed human. *Diving and Hyperbaric Medicine*. 2009 Dec;39(4):193-9.
If a journal carries continuous pagination throughout a volume (as many medical journals do) then the month and issue number may be omitted:
de Bruijn R, Richardson M, Schagatay E. Oxygen-conserving effect of the diving response in the immersed human. *Diving and Hyperbaric Medicine*. 2009;39:193-9.

Illustrations, figures and tables

These should **NOT** be embedded in the word processor document, but submitted as individual, separate electronic files. Each figure and table must be mentioned within the text of the article, e.g., 'Rates of decompression illness by demographic are presented in Table 1…', 'Differences in rates of decompression illness were not significant (Table 1)', etc.

The approximate positions of tables and figures should also be identified in the text. No captions or definition of symbols used should appear within the body of the table or image, but should be placed in the legend. Legends should generally contain fewer than 40 words and must be listed on a separate page at the end of the main text file.

Table data should be presented either as tab-spaced normal text or using table format, with tab-separated columns auto-formatted to fit content. No gridlines, borders or shading should be used. References appearing in table or figure legends should continue the sequence of references in the main text of the article in accordance with the position of citing the table/figure in the text .

Illustrations and figures should be submitted as separate electronic files in TIFF, high-resolution JPEG or BMP format. JPEGs must be saved at their maximum size and compression avoided. Files of 10 Mb or larger should be submitted on CD-R or in a zip file. There is also a free, large-file send service available on the internet at <https://www.wetransfer.com>. Authors are advised to view their illustrations converted to grayscale to ensure that contrast within the image is sufficient for clarity when printed. Any graphs or histograms created in Excel should be sent within their original Excel file, including the data table(s) from which they were produced.

Special attention should be given to ensuring that font sizes within a diagram are sufficiently large to be legible should the diagram be resized for single-column representation. The preferred font is Times New Roman.

Posted photographs should be glossy and can be either black-and-white or colour. Magnification should be indicated for photomicrographs, and consideration given to the positioning of labels on diagnostic material as this can greatly influence the size of reproduction that can be achieved in the published article.

Colour is available only when it is essential and will be at the authors' expense (approximately Aus $750 for a single A4 page).

Consent and ethical approval

Studies on human subjects must comply with the Helsinki Declaration of 1975, as revised in 2000, and those using animals must comply with National Health and Medical Research Council Guidelines or their equivalent in the country in which the work was conducted. A statement affirming Ethics Committee (Institutional Review Board)

approval should be included in the text. A copy of that approval should be available if requested. Patient details must be removed and photographs made unrecognizable unless written consent for their publication has been obtained from the patient(s). When informed consent has been obtained, this should be indicated in the article. When considering clinical trials, preference will be given to submissions registered at a recognised trial registry site such as the Australia and New Zealand Clinical Trials Registry <http://www.anzctr.org.au/>.

Conflict(s) of interest and funding sources

Any conflicts (including potential ones) of interest, financial, academic or otherwise should be identified.
All sources of funding for a submitted piece of research should be identified.

Copyright

Manuscripts must be offered exclusively to *Diving and Hyperbaric Medicine*, unless clearly authenticated copyright exemption accompanies the manuscript. Obtaining permission to use tables or figures, etc, from other published work is the responsibility of the author(s), and evidence of such permission is required before publication.

Authors must agree to accept the standard conditions of publication. These grant *Diving and Hyperbaric Medicine* a non-exclusive licence to publish the article in printed form in *Diving and Hyperbaric Medicine* and in other media, including electronic form; also granting the right to sub-license third parties to exercise all or any of these rights. *Diving and Hyperbaric Medicine* agrees that in publishing the article(s) and exercising this non-exclusive publishing sub-licence, the author(s) will always be acknowledged as the copyright owner(s) of the article.

SPUMS and EUBS Annual Scientific Meetings

Diving and Hyperbaric Medicine has published articles based on many of the presentations from SPUMS ASMs. Presenters, including the Guest Speaker(s), are reminded that this is an explicit condition of their participation in the SPUMS meetings, but recognizing that not all presentations are suitable for publication. Speakers at EUBS meetings, both those giving keynote addresses and those presenting previously unpublished research are strongly encouraged to submit manuscripts to DHM. All such articles are subject to the above requirements of standards, presentation and peer review.

Zetterström Award

The author(s) of the scientific poster winning the Zetterström Award at each EUBS ASM explicitly agrees to submit an article based on their poster to *Diving and Hyperbaric Medicine*. This paper is subject to the above requirements of standards and presentation and will be subject to the standard review process.

SPUMS Diploma dissertations

It is the policy of SPUMS that diploma candidates are encouraged to publish their dissertation in *Diving and Hyperbaric Medicine*. Advice on preparing a dissertation for submission to DHM is available from the SPUMS Education Officer or the Editor on request.

Synopses or summaries of master's or doctoral theses will also be considered in order to draw the diving and hyperbaric medical and scientific community's attention to the work of young researchers.

Any manuscript not complying with the above requirements will be returned to the author for revision before it will be considered for publication.

Publication schedule

All submitted manuscripts will be subject to open peer review by a member of the Editorial Board and at least one other reviewer. Reviewer comments will be provided to authors with any recommendations for improvement before publication, or if the article is rejected. *Diving and Hyperbaric Medicine* believes that a transparent review process is indicated in such a small specialty. Reviewers are often able to identify the origin of manuscripts and, in the interests of fairness, the authors are therefore provided the names of reviewers of their articles.

The review process typically takes 4–8 weeks and papers are generally scheduled for publication in order of final acceptance. The Editor retains the right to delay publication in the interests of the Journal. Accepted contributions will be subject to editing.

Proofs of articles to be published will be sent to authors in PDF format by e-mail close to the time of publication. Authors are expected to check the proofs very carefully and inform the editorial office **within five days** of any minor corrections they require. Corrections should be listed in an e-mail sent to the journal address <editor@dhmjournal.com>, or annotated electronically in the pdf file.

Following publication, one complimentary copy of *Diving and Hyperbaric Medicine* will be sent to the corresponding author of each contributory paper (except to members of SPUMS/EUBS). A PDF copy of their article will be forwarded to the corresponding author. A limited number of additional print copies of the journal issue containing the article are available for purchase from the SPUMS Administrator, <admin@spums.org.au>.

DIVER EMERGENCY SERVICES PHONE NUMBERS

AUSTRALIA
1800-088200 (in Australia, toll-free)
+61-8-8212-9242 (International)

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0800-4DES-111 (in New Zealand, toll-free)
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+852-3611-7326 (China)
010-4500-9113 (Korea)
+81-3-3812-4999 (Japan)

SOUTHERN AFRICA
0800-020111 (in South Africa, toll-free)
+27-10-209-8112 (international, call collect)

EUROPE
+39-06-4211-8685 (24-hour hotline)

UNITED KINGDOM
+44-07740-251-635

USA
+1-919-684-9111

The DES numbers (except UK) are generously supported by DAN

DAN Asia-Pacific DIVE ACCIDENT REPORTING PROJECT

This project is an ongoing investigation seeking to document all types and severities of diving-related accidents. Information, all of which is treated as being confidential in regard to identifying details, is utilised in reports on fatal and non-fatal cases.

Such reports can be used by interested people or organisations to increase diving safety through better awareness of critical factors.

Information may be sent (in confidence unless otherwise agreed) to:

DAN Research
Divers Alert Network Asia Pacific
PO Box 384, Ashburton VIC 3147, Australia
Enquiries to: <research@danasiapacific.org>

DIVING INCIDENT MONITORING STUDY (DIMS)

DIMS is an ongoing study of diving incidents. An incident is any error or occurrence which could, or did, reduce the safety margin for a diver on a particular dive. Please report anonymously any incident occurring in your dive party. Most incidents cause no harm but reporting them will give valuable information about which incidents are common and which tend to lead to diver injury. Using this information to alter diver behaviour will make diving safer.

Diving Incident Report Forms (Recreational or Cave and Technical)
can be downloaded from the DAN-AP website: <www.danasiapacific.org>
They should be returned to:
DIMS, 30 Park Ave, Rosslyn Park, South Australia 5072, Australia.

CONTENTS

Diving and Hyperbaric Medicine Volume 40 No. 2 June 2010

Diving and Hyperbaric Medicine is indexed on SCIE and Embase/Scopus

Printed by Snap Printing, 166 Burwood Road, Hawthorn, Victoria 3122
hawthorn@snap.com.au