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 42 No. 2 June 2012

EUBS

Cost analysis of HBOT for radiation cystitis

The normobaric oxygen paradox

HBOT for sickle-cell disease crisis

Brown-Sequard syndrome from DCS

Are dental cements weakened by pressure cycling?

Airways resistance and scuba in divers with asthma

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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Diving and Hyperbaric Medicine **is published jointly by the South Pacific Underwater Medicine Society and the European Underwater and Baromedical Society**

The Editor's offering

As this issue marks ten years as Editor, it is timely for me to reflect on the Journal's journey over that time. At a personal level, it has been an educational and deeply satisfying job which I hope, good health allowing, to continue for years to come. Certainly there are still goals that I hope to achieve before passing this task on to others. Nevertheless, SPUMS and EUBS need to have contingency plans in place as I approach three score and ten all too rapidly!

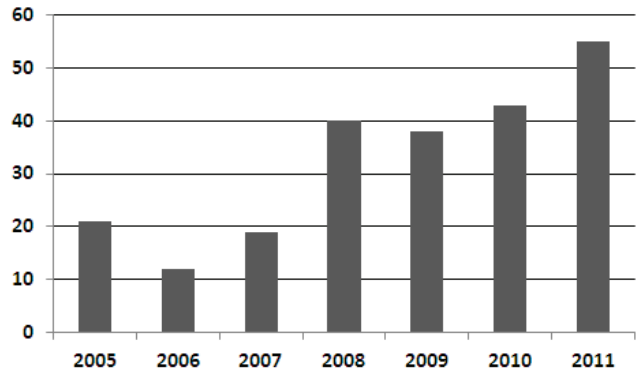
The SPUMS Journal is a remarkable publication which has been inadequately recognised by the international medical community. Thanks to two excellent editors over almost three decades – Douglas Walker and John Knight (JK) – and a policy of basing the Journal's contents primarily on the proceedings of the SPUMS' annual scientific meetings, especially with the fine guest speakers these meetings have attracted, there are many gems in the back catalogue. Some of the most valuable review articles in the literature have appeared within these pages, but have been little appreciated outside of SPUMS. All this has now changed; but first, let's review some of the landmarks over the past decade.

When I took over from JK in 2002, the Journal needed to be taken to the next level; in particular, enhancing its professional appearance, introducing proper peer review, establishing an Editorial Board and creating my own vision for the Journal. This was a very steep learning curve, despite having been on the receiving end of editors' and reviewers' red pens for my entire medical career! Setting up a journal office proved easy, thanks to the help of the New Zealand Medical Association and the Canterbury Area Health Board, but what was especially needed was good professional assistance – I could not physically do this job on my own, run a busy anaesthetic practice and the hyperbaric service in Christchurch.

Murphy was out to lunch the day Sarah Webb, a young history graduate from the UK with medical publishing experience at the Royal College of Physicians in London, turned up. I doubt whether I could have managed without her wonderful, meticulous expertise. I am glad to say that, even though she has moved on to other things, she remains our professional proof reader, in which role she guards the Queen's English tenaciously. SPUMS and EUBS owe a debt of gratitude to Sarah that has largely gone unrecognised.

A very important milestone has been SPUMS and EUBS becoming joint publishers of the Journal in 2008. This had an immediate and radical effect on submissions (Figure 1), not only in their numbers but also in their nature. Before 2008, less than 30% of published articles were of original work, whereas from 2008 onwards this has risen to 60%. Interestingly the ratio of diving medicine to hyperbaric medicine papers has remained constant at 4:1, maintaining the Journal's emphasis on the former. Another major change

Figure 1
Number of papers submitted 2005–2011



has been in the geographical distribution of submitted papers. Prior to 2008, most of the published papers came from Australia (63%) and New Zealand (14%), with the remaining 23% from 11 other countries, mainly the UK and USA. Since 2008, the percentages are Australia 32%, and the UK 12%, with 20 other countries making up the other 56%.

In 2002, the SPUMS Journal was indexed on the Elsevier database Embase/Scopus, but two applications over the years to the National Library of Medicine (NLM) for Medline citation had been unsuccessful. In 2007, I applied to Thomson Reuter's SciSearch®, which would provide the Journal with an Impact Factor, and this was successful. In 2009, the Journal again applied for indexing on Medline, but this was narrowly turned down. We appealed that decision on a number of factual and academic grounds and, after an internal academic review, NLM determined that DHM would appear on Medline from the beginning of 2011. Subsequently we have also been approved for PubMed for abstracts from 2008 onwards. Medline citation is far and away the most important academic achievement of my tenure, but this was only possible because of the quality of the published material with which we had to work. Authors who contributed over the two-year assessment period (2008-09) should be proud of their performance; I am but the ringmaster.

My vision for the Journal has been presented in previous editorials, and is unlikely to change in the foreseeable future. I wish to thank the many people who have helped me, especially Michael Bennett and my partner-in-crime in Europe, Peter Müller. A senior academic recently said that he looked forward to DHM arriving as it was "*the only journal I read from cover to cover!*" – a better compliment could not be had. Keep those submissions coming!

Michael Davis

The new four-compartment recompression chamber, manufactured by Fink Engineering, being craned into its housing at the Prince of Wales Hospital, Sydney. This is currently the largest rectangular therapeutic hyperbaric chamber in the world.

The President's page

Peter Germonpré, President EUBS

It seems like only yesterday since I took over from Alf Brubakk to become the President of this Society. Is it really already three years? During our next Annual Scientific Meeting in Belgrade, I will be handing over to my dear friend Costantino Balestra. A change in presidency should not necessarily mean a change in policy, and I am glad that we will continue to work together for a long time to come, in order to make EUBS even more efficient, more integrated and more attractive to our members.

We have come some way already. The website was developed in 1999 and reworked with new functionalities in 2008. Online membership renewal (by the way, about now is the proper time to do this) is working almost flawlessly; free access is offered to the German Society for Diving and Hyperbaric Medicine (GTUeM) literature database (a full-text database where all previous EUBS Conference Proceedings are also accessible online); a section with research and educational announcements was added.

In 2008, the agreement with SPUMS to jointly develop this journal was 'signed off'. Since then, we have proven together that it has the potential to grow and be recognised as one of the leading journals in the field.

Whilst staying a truly informal society, the active role of our Executive Committee is growing year after year, and we hope to keep this movement going. We have a larger number of candidates for ExCom elections than ever, showing that our enthusiasm is contagious. The voting ballot will be sent electronically to all EUBS members in the next few weeks. Please do not forget to vote. A brief résumé of each of these enthusiastic candidates appears in the EUBS section of this issue.

For our ASM in September, the organising committee is doing an excellent job in cooking up an attractive programme (see <www.eubs2012.org>). Prior to EUBS 2012, the European Committee for Hyperbaric Medicine will hold a Consensus Meeting. Our continuing complementary relationship with ECHM shows that in Europe, despite it being a set of different nations, different cultures and different ideas, we can achieve difficult goals by cooperating with, not opposing each other.

Then, in 2013, our planned joint SPUMS/EUBS meeting will now become a tri-continental meeting, with the South African Undersea and Hyperbaric Medical Association (SAUHMA) joining us in the endeavour. With the support of the Reunion Society for Diving and Hyperbaric Medicine (ARESUB) and of the Scott Haldane Foundation, we are headed for an exciting adventure. Last April, a small party representing all the societies went out to scout the location, and though the going was tough (three days packed with hotel, conference venue and dive centre visits), it was well worth the trip and prospects are looking good. So if there is anything you should do right now, please block 21–28 September 2013 in your calendar and start saving for the trip!

Times do change, and people move on. I could not end this column without thanking our colleague and good friend Willi Welslau. He has been the President of the GTUeM for seven years, before passing the gavel early this year to Karin Hasmiller. Willi has worked hard with GTUeM to promote evidence-based hyperbaric oxygen therapy (HBOT) and diving medicine, to propose structured education programmes and German accreditation and to reach out to other European organisations. His efforts and achievements show once more that, in Europe, national organisations are still an efficient way of advancing HBOT and diving medicine. I am very confident that Karin will do a great job. It is important to realise that people like Willi and Karin, with energy to spare and a vision for the future, are the kind that we need to help advance our branch of medicine. Luckily, I know there are more of those around. If you could now please all show yourselves?

So, to end, here's your 'to do' list for this issue:

- renew your membership to EUBS
- register for the 2012 EUBS Meeting
- cast your vote for EUBS Member at Large 2012 and Vice President
- get involved in an active way: contribute a paper to DHM, promote EUBS membership among your colleagues, participate actively in our meetings and Society!

Key words
Medical society, meetings

The

website is at
<www.eubs.org>

Members are encouraged to log in and to keep their personal details up to date

Original articles

The 'normobaric oxygen paradox': does it increase haemoglobin?

David De Bels, Sigrid Theunissen, Jacques Devriendt, Peter Germonpré, Pierre Lafere, Joseph Valsamis, Thyl Snoeck, Philippe Meeus and Costantino Balestra

Abstract

(De Bels D, Theunissen S, Devriendt J, Germonpré P, Lafere P, Valsamis J, Snoeck T, Meeus P, Balestra C. The 'normobaric oxygen paradox': does it increase haemoglobin? *Diving and Hyperbaric Medicine*. 2012;42(2):67-71.)

Background: A novel approach to increasing erythropoietin (EPO) using oxygen (O_2) (the 'normobaric oxygen paradox') has been reported in healthy volunteers. We investigated whether the EPO increase is sufficient to induce erythropoiesis by comparing two protocols of O_2 administration.

Methods: We compared the effect of daily versus alternate days 100% O_2, breathed for 30 minutes, on haemoglobin concentrations during a 12-day period. Nine subjects underwent the two protocols six weeks apart.

Results: We observed a significant increase in haemoglobin (as a percentage of baseline) in the alternate-days group compared to the daily group and to baseline after four days (105.5 ± 5.7 % vs. 99.6 ± 3.3 % difference from baseline; $P < 0.01$). At the end of the experimental period, haemoglobin values increased significantly compared to baseline in both groups. There was a significant percentage rise in reticulocyte count in the alternate-days group compared to the daily group (182 ± 94 % vs. 93 ± 34 %; $P < 0.001$).

Conclusion: The normobaric oxygen paradox seems effective in increasing haemoglobin in non-anaemic, healthy volunteers, providing sufficient time is allowed between O_2 applications. The exact time interval is not clearly defined by this study but should probably be at least or greater than two days. Further studies are needed to define more precisely clinical applications in the use of O_2 as a pharmaceutical agent.

Key words

Oxygen, haematology, reactive oxygen species (ROS), physiology

Introduction

There has been increasing concern during the last decade about transfusion hazards.[1,2] Furthermore, no clear evidence has arisen about the benefits of transfusion and even questions about increasing mortality have even been raised.[3,4] The use of a red blood cell progenitor enhancer such as exogenous erythropoietin (EPO) is extensively recognised, and a relatively low rate of adverse effects has been reported in patients adequately followed in medical institutions.[5] However, the price of such medications is very high and its availability is limited in some countries.

A recently described phenomenon called the 'normobaric oxygen paradox' (NOP), may show possible clinical applications.[6] The technique consists of the simple application of high-concentration oxygen (O_2) breathing (monitored by transcutaneous oxygen tension) to spontaneously breathing subjects. This has been shown to provoke a significant increase in endogenous erythropoietin production.[7] The purpose of this paper is to report a potential clinical application of NOP to increase haemoglobin (Hb) concentration in healthy humans.

THE NORMOBARIC OXYGEN PARADOX

The mechanism proposed to explain this phenomenon lies deep within the fundamental cellular mechanisms of adaptation to hypoxia. This depends on the availability of oxygen-free radicals (reactive oxygen species, ROS). In fact, in the presence of ROS, the hypoxia-inducible factor alpha, (HIF-1 α), is linked constantly to the tumor-suppressing Von Hippel Lindau protein (VHLp). This formed complex is subsequently bound to ubiquitin ligase and finally recycled in the proteasome (Figure 1).[8] In case of limited availability or absence of ROS, the total amount of HIF-1 α available will not link with VHLp and thus can be dimerised with HIF-1ß. This HIF complex can thus start the cascade of erythropoietin (EPO) gene expression through binding to promoters such as hypoxia-responsive elements, and subsequently lead to EPO de novo synthesis.[9]

Increasing the patient's inspired oxygen (and thus the intracellular availability of ROS) will increase the production of protective agents against ROS, i.e., up-regulation of glutathione synthetase enzyme activity (gamma glutamyl cysteine synthetase). This enhanced activity will increase the glutathione production and subsequently the scavenging of ROS. During the hyperoxic period, the amount of reduced glutathione (GSH) will therefore rapidly increase to overcome the increased oxidative agents. When stopping hyperoxia, this increased amount of GSH together with an ongoing (slow) conversion of oxidised glutathione to GSH will produce an excess of ROS scavenging. The importance

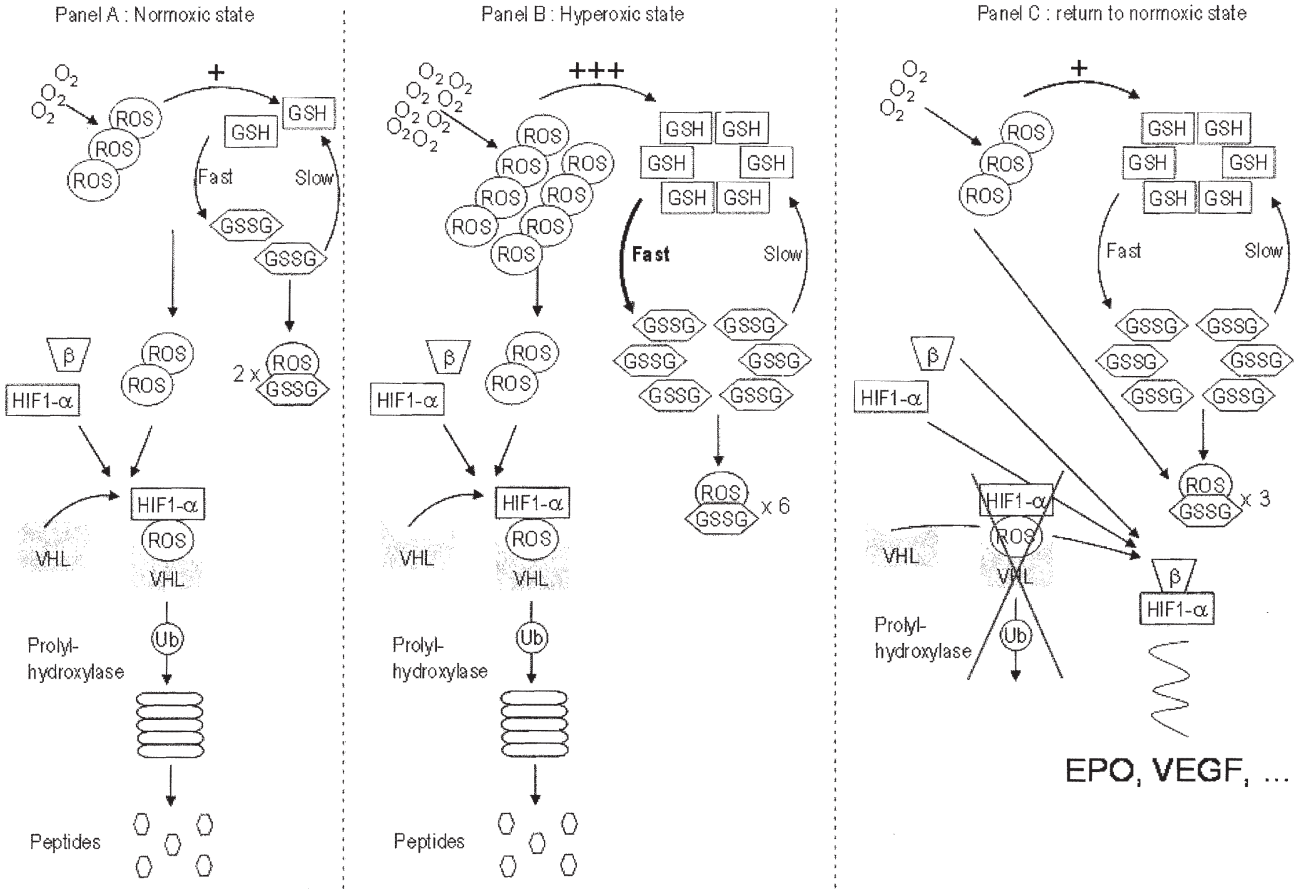
Figure 1

The normobaric oxygen paradox (reproduced with permission)[8]

Panel A: Normoxia – normal intracellular function

Panel B: Hyperoxia – during normobaric hyperoxia, reactive oxygen species (ROS) stimulate glutathione (GSH) production; hypoxia-inducible factor alpha (HIF-1 α) is continuously produced, but continuously inactivated by its binding to another protein, Von Hippel Lindau tumor-suppressor protein (VHL), and by subsequent ubiquitous metabolisation by hydroxylation of proline residues

Panel C: Return to normoxic conditions. All ROS are neutralised by the increased intracellular GSH. This induces exogenous erythropoietin (EPO) gene expression similarly to hypoxia, and this situation could be called the normobaric oxygen paradox; GSSG – glutathione disulphide; VEGF – vascular epithelial growth factor; Ub – ubiquinone (reproduced with permission)



of optimal glutathione availability to increase EPO synthesis in this setting has recently been emphasised by n-acetylcysteine administration (NAC).[10] This phenomenon will last long enough after the O_2 concentration reduction to mimic a 'hypoxic' situation, where the availability of ROS is reduced.[11,12] This complex situation will allow the binding of HIF dimmers, triggering EPO gene expression.

Material and methods

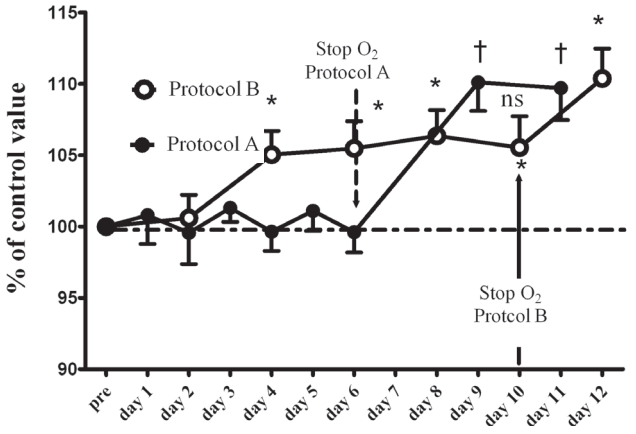
SUBJECTS

Nine healthy volunteers (five men and four women; aged 18 to 41 yrs, mean 30 yrs) participated in this study after Academic Ethics Committee, ISEK, Brussels, Belgium approval and written, informed consent was obtained. The study was performed in accordance with the Declaration of Helsinki Subjects were asked not to smoke or to take any medication (NAC or iron) or perform strenuous physical exercise 24 hours before and during each study period or stay at altitude within two weeks before the experiments.

EXPERIMENTAL PROTOCOL

Subjects underwent two protocols of O_2 breathing. Each one subsequently underwent the two protocols. Protocol A involved 100% O_2 daily and protocol B 100% O_2 on alternate days. The two protocols took place six weeks apart, so that values for the measured parameters had returned to baseline in between. The sequence was not randomised since all subjects underwent both protocols. The subjects breathed 100% normobaric O_2 (15 L min^{-1} via a non-rebreathing facemask) for 30 minutes. Constant monitoring of the following parameters controlled O_2 breathing: mask fit, movement of the three one-way valves on the mask, movement of the reservoir bag and moisture formation on

Figure 2
Comparison baseline haemoglobin after 30 minutes of 100% O_2 breathing daily (protocol A; † $P < 0.01$) or on alternate days (protocol B; * $P < 0.01$); ns – not significant



the transparent mask during expiration. Only 7 ml of blood were withdrawn for analysis each day so that plasma volume contraction did not occur, since this can interfere with EPO production.[13]

ANALYSES

Samples were drawn in EDTA tubes by repeated needle sticks in the antecubital fossa. They were centrifuged within 4 hours of their sampling. Haemoglobin (Hb) and haematocrit (Hct) levels were measured in a routine automated manner using red laser light to measure both volume and haemoglobin concentrations on a cell-by-cell basis (ADVIA 2120, Siemens AG Healthcare Sector, Erlangen, Germany); reticulocyte count was done by colorimetry on the same apparatus.

STATISTICS

Standard statistical analyses were performed, including mean, standard deviation, and a one-sample Student's paired t-tests for between- and within-subject effect after Kolmogorov Smirnov tests for normality of distribution of the data. Taking the initial value as 100%, percentage changes in Hb were calculated, thereby allowing an appreciation of the magnitude of change rather than the absolute values. The raw data Hb are available on Excel™ file from the journal office.

Results

HAEMATOCRIT AND HAEMOGLOBIN

All subjects had baseline haematocrit (41.3 ± 2.8 %) and haemoglobin values (14.8 ± 1.1 g L^{-1}) within the normal population range. No significant increase of haematocrit was seen (43.9 ± 1.2 % and 44.0 ± 1.7 %) after either of the two protocols ($P > 0.05$). This reassured us as to the hydration status of our subjects. The Hb level started to rise after day 4 of protocol B (alternate days; $P < 0.01$) and this rise was maintained throughout that study period. For protocol A (daily O_2) the Hb level did not change from baseline until O_2 administration ceased ($P < 0.01$). Figure 2 shows the percentage changes of Hb during the two study periods. Some Hb values fell transiently before rising, and the rate of rise varied between individuals.

RETICULOCYTE COUNT

Reticulocyte counts were measured at baseline and at day 7, any changes from baseline being expressed as percentage change in count. There was a significant rise in reticulocyte count in the alternate-day group as compared to the daily group (182 ± 94 % vs. 93 ± 34 %, $P < 0.001$), demonstrating enhanced erythrocyte production.

Discussion

At cessation of O_2 breathing, the arterial O_2 partial pressure falls within minutes to a normal baseline level. After this situation, transcription of EPO starts within 4–8 hours.[14] In protocol B, a significant increase in Hb was seen after four days and remained elevated throughout that study period. This is consistent with the NOP hypothesis that permits an increase in EPO synthesis, thus increasing Hb.[7,15] The literature shows a rapid increase in reticulocytes and Hb with high rh-EPO doses in patients with normal bone marrow function.[16] If, during this time lapse, and according to the NOP physiology, one re-introduces O_2, the effect could potentially be cancelled. This could explain the absence of Hb increase until after O_2 cessation in protocol A. The interval between two periods of O_2, on a daily basis is probably too short to permit a sufficient rise in EPO to stimulate an increase in Hb.

Both protocols resulted in a significant increase of Hb after stopping O_2 administration when compared to baseline values. It is important to bear in mind that these people were healthy volunteers. The optimal time between two periods of O_2 is not clearly determined by our study, but we do see an increase in Hb in the alternate-day protocol earlier compared to the daily protocol, proof that every day is too frequent but that when O_2 administration ceases, Hb rises according to the NOP hypothesis. It is interesting to see that Hb remains high for several days, perhaps allowing for a twice-a-week protocol.

A drop from 100% to 21% of O_2 in the breathing gas induces an NOP effect in healthy volunteers.[6] The NOP appears to be an efficient way to increase EPO. This could have clinical potential, as EPO has been shown to be active in both cardio- and neuroprotection and it could reduce costs.[16–18] Repetition of this experimental protocol in anaemic patients could increase circulating EPO levels and

thereby increase erythropoiesis and thus Hb. The important point appears to be to leave a sufficient time interval between two O_2 administrations for EPO synthesis in order to avoid competing against this mechanism reapplication of O_2 too soon.

Even though the exact amount of O_2 required to produce a NOP effect is not known, the minimal concentration of inspired O_2 seems to be around 40–50% providing that glutathione availability is optimal.[19] Increasing the variation of pO_2 shows less consistent results especially when associated with hypoxia, as shown by Debevec.[20] Further investigations to determine the optimal 'dose' are welcomed, and recent publications suggest that 100% O_2 may not be optimal.[21] NOP is a recent protocol and the time frame as well as the precise dose of O_2 needed are crucial to establishing clear recommendations for clinical use.

Whilst the Haldane effect may be present in bed-rest patients with hypoventilation, this seems unlikely in young, healthy adults without pulmonary disease. If this were the case in our study, it would be present in both groups. We also know that NAC by itself has been shown to raise EPO levels and this phenomenon is increased by addition of oxygen.[10,22] Therefore, we asked our volunteers not to undergo strenuous activities or to take any antioxidants such as NAC.

The main limitation of this study is the small sample size. Further clinical investigations are needed to achieve the optimal use of the NOP and to better understand which patients would benefit from the induction of this physiological pathway. Potential clinical applications of the NOP could be twofold. Firstly, a small number of O_2 breathing sessions of limited duration (e.g., 30 minutes) appear to be sufficient to increase circulating endogenous EPO levels, thus leading to a cytoprotective effect on brain and cardiac cells. Secondly, increasing endogenous EPO seems to increase Hb levels of volunteers if sufficient time is allowed between two O_2 sessions.

Conclusion

Alternate-day O_2 breathing for 30 minutes stimulated an early increase in Hb, whereas a rise did not occur with daily O_2 until administration ceased. Likewise, the reticulocyte count was elevated more by the alternate-day protocol than the daily protocol. Daily O_2 is too frequent an exposure in a non-anaemic population, but the exact best time course for the use of the normobaric oxygen paradox to stimulate erythropoiesis is not clearly established in this study.

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Submitted: 15 September 2011
Accepted: 09 April 2012

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Editor's note:
All nine authors contributed to several components of the planning, conduct, analysis and reporting of this study..

Effect of a single pool dive on pulmonary function in asthmatic and non-asthmatic divers

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Abstract

(Ivkovic D, Markovic M, Todorovic BS, Balestra C, Marroni A, Zarkovic M. Effect of a single pool dive on pulmonary function in asthmatic and non-asthmatic divers. *Diving and Hyperbaric Medicine*. 2012;42(2):72-77.)

Introduction: The aim of this study was to evaluate the effect of a single, shallow, swimming pool scuba dive on pulmonary function in divers with asthma as compared to controls. Opinions concerning the risks of diving with asthma are still contradictory and inconclusive in the diving community.

Methods: Baseline pulmonary function tests (PFTs) were performed on a group of 22 divers with asthma and on a control group of 15 healthy divers. The same PFTs were repeated within 10 minutes after a single pool dive, at 5 metres' depth for 10 minutes. PFTs were measured using a portable Jaeger SpiroPro™ device. Student's paired t-tests and linear mixed effects model comparisons and interactions within the groups were used in the data analysis.

Results: Divers with asthma initially presented significantly lower values of FEV_1/FVC%* ($P < 0.01$), FEF_{25}* ($P < 0.01$), FEF_{50}* ($P < 0.001$), FEF_{75}* ($P < 0.01$) and $FEF_{25–75}$* ($P < 0.001$) compared to controls. There were significant reductions in FEV_1 ($P < 0.01$), FEV_1/FVC% ($P < 0.05$), FEF_{50}* ($P < 0.01$), FEF_{75}* ($P < 0.05$) and $FEF_{25–75}$* ($P < 0.001$), in the asthma group after the dive as compared to the control group. PEF was initially lower, although not significantly, in the asthma group and did not change significantly after the dive in either groups ($P > 0.05$).

Conclusions: A single, shallow, pool scuba dive to 5 metres' depth may impair function of small airways in asthmatic divers. More studies are necessary to estimate the risks when divers with asthma practise scuba diving. PFT results should be analysed after replicated dives in deeper pools and controlled open-water conditions.

Key words

Lung function, pulmonary function, scuba diving, asthma, research

Introduction

The population of divers with asthma is growing and many asthmatics dive regularly, even though some diving physicians do not give them approval to dive. A problem is that many divers with asthma deny any history of asthma while answering medical questionnaires. It is known that 10% to 15% of children have some history of recurrent wheezing, while 5% to 8% of adults are diagnosed as 'asthmatics'. Survey data show a similar prevalence of asthma in recreational divers as in the general population.[1]

People with asthma represent a heterogeneous group of patients who may experience a wide range of both frequency and severity of symptoms, such as coughing, wheezing, chest tightness and/or shortness of breath. The most common type of diving, with open circuit scuba, can expose the participant to several important asthma triggers. The diver is exposed to cold and physical stress, with possible exertion and anxiety The breathing gas is cold, dry and dense, which increases respiratory resistance. Diving equipment imposes both increased inspiratory and expiratory resistance, resulting in increased work of breathing. Theoretically it might be expected that all these factors would provoke bronchospasm, which might increase the risk of pulmonary barotrauma and reduce exercise capability.

Asthma remains the most controversial medical condition affecting recreational divers. Current criteria for pulmonary fitness to dive in people with asthma are also inconsistent and controversial. Asthma has long been considered a strong contra-indication to scuba diving. However, concensus in the diving medical community has changed radically since the 1990s, especially after the UHMS Meeting in 1995, at which a more liberal attitude was adopted that some asthmatics could be certified for scuba diving under certain circumstances.[2,3]

There are still many different medical opinions with recommendations ranging from 'never – once asthmatic, always asthmatic', to 'no diving with a history of asthma over the previous five years', to 'no diving within two days of wheezing'.[4] The most consistent consensus among diving experts is that people whose episodes of bronchospasm are associated with exercise, anxiety or the inhalation of cold air should not dive. Despite the theoretical objections

*** Footnote:** VC_{in} – inspired vital capacity; FEV_1 – forced expiratory volume in 1 sec; FVC – forced vital capacity; FEF_{25} – forced expiratory flow rate at 25% of FVC; FEF_{50} – forced expiratory flow rate at 50% of FVC; FEF_{75} – forced expiratory flow rate at 75% of FVC; $FEF_{25–75}$ – mean forced expiratory flow rate between 25 and 75% FVC; PEF – peak expiratory flow rate.

and speculations related to increased risk of pulmonary barotrauma, there is no solid evidence that asthma carries an increased accident rate. Epidemiology is also inconclusive and data are mainly based on surveys of active divers and the retrospective compilation of accident information.

A Divers Alert Network (DAN) retrospective review (reported only as a meeting abstract), aimed at assessing the risk of asthma inducing arterial gas embolism (AGE) and type II decompression sickness, suggested an approximately twofold increase in risk for divers with asthma, but this did not reach statistical significance. Other surveys have shown that scuba diving deaths linked to asthma were infrequent.[5,6] DAN reported 96 recreational scuba diving fatalities in 1992 and concluded that diabetes mellitus and bronchial asthma did not appear prominently in these series.[7] Recent annual reports have offered similar conclusions.[8] In contrast to the USA and UK experiences, asthma was reported as a contributing factor in 8% of 124 scuba diving deaths in Australia and New Zealand.[9] In a study of 17,386 dives, there were no reported cases of serious diving injury.[10] Another study (again only reported as a meeting abstract) did not indicate any statistically significant decrease in the peak expiratory flow rates in people with asthma after a single introductory open-water dive to 5 metres' depth for 30 minutes.

In order to better assess the possible risks of asthma when scuba diving, we decided to evaluate lung function in a real-life situation, immediately after a single, swimming pool scuba dive, a diving pattern experienced by all prospective divers, in a group of divers with asthma and a control group with no history of bronchospasm.

Methods

The subjects were 22 divers with asthma and 15 healthy control divers, who were members of various diving clubs in Belgrade. All divers completed a questionnaire relating to their medical history and diving habits, and signed an informed consent form. Tests were conducted in accordance with the Declaration of Helsinki and were approved by the Ethics Committee of the Faculty of Medicine, the University of Belgrade, Serbia.

STUDY GROUPS

The control group comprised healthy divers without any serious respiratory disease in their medical history. The group of divers with asthma was relatively heterogeneous, and none had ever experienced any breathing difficulty while diving. All asthmatics declared that their previous episodes of wheezing were not associated with exercise, anxiety or the inhalation of cold air. For at least 15 days before the dives, all asthmatics were in a stable condition, without presenting any breathing difficulties. There were no smokers in either of the groups.

On the basis of their medical history and documentation, the divers with asthma were classified into three categories. There were eight participants classified as 'childhood asthma', all free of any symptoms during the previous 5–10 years. There were 10 participants classified as 'mild intermittent asthma', with symptoms less than twice a week and with a normal level of activity between brief exacerbations. There were four divers classified as 'mild persistent asthma', with symptoms twice or more times a week, but less than once a day, whose activity might be affected by exacerbations.[11] Eight of these 22 asthmatics (two with a history of childhood asthma, two with mild intermittent asthma and four with mild persistent asthma) had undergone medical assessment of fitness to dive in our facility before the commencement of their first diving course. Another six asthmatic divers (two childhood asthma and four mild intermittent asthma) requested our medical advice during their diving career, while the remaining eight asthmatics (four childhood asthma and four mild intermittent asthma) never requested any medical opinion on their fitness to dive.

Thus 14 of the divers with asthma had previously been assessed in our facility, when they performed a 43 cm step test for 3 minutes (according to the UK Sports Diving Medical Committee guidelines), followed by pulmonary function tests (PFTs) at 10 minutes after exercise.[12] There was no decrease in FEV_1 by more than 10% from the baseline value or in other respiratory parameters measured in these divers. Coincidentally, we recorded an improvement in small airways function and a statistically significant increase in $FEF_{25–75}$, with the result that these asthmatics had been cleared at that assessment to practise scuba diving.

PULMONARY FUNCTION TESTS

Baseline PFTs were measured in both groups 10 minutes before the study dive. Spirometry and flow-volume loop measurements, including PEF, VC_{in}, FVC, FEV_1, FEV_1 / FVC ratio, FEF_{25}, FEF_{50}, FEF_{75}, and $FEF_{25–75}$ (see footnote p. 72 for definitions), were performed according to the 1993 European Respiratory Society (ERS) recommendations, and updated according to the 2005 ATS/ERS standardisation of spirometry.[13,14] PFTs were assessed by means of a portable Jaeger SpiroPro™ device and the results were corrected to BTPS. Testing was undertaken with the diver in a standing position, and the results of at least three acceptable flow-volume measurements were used for analysis. The same PFTs were repeated 10 minutes after the dive.

DIVE PROTOCOL

Test dives were performed in an indoor, chlorinated swimming pool. Chlorine concentration was set to 0.4 mg L^{-1}, the pH value to 7.3 and water temperature was 25°C. The depth of the dive was 5 metres and duration was 10 minutes. Diving was performed in a relaxed manner, with minimal

underwater work. The approximate speed of underwater swimming, mainly in a horizontal position, was 15–20 m min^{-1}, which corresponded to mild physical activity.

STATISTICAL ANALYSIS

Student's paired t-tests were applied after normality testing of the samples by means of the Kolmogorov Smirnov test. All data are expressed as mean (SD). A value of $P < 0.05$ was considered significant. A linear mixed-effects model was also used, allowing for different variances within the levels of a group factor. In statistics, the term "*covariance components models*" is often used, alluding to the fact that in linear mixed-effects models, we may decompose the covariance into components attributable to within-groups vs. between-groups effects. This was useful for better insight into how the two groups behaved before and after the dive, and what the differences were between the groups. Data are presented as mean and 95% confidence intervals (CI).

Results

There were no statistical differences between the two groups relating to age, anthropometric data and diving habits. Data related to diving habits were not normally distributed and are reported as median and range (Table 1). Categorisation of the asthma group and details of their medications are shown in Table 2.

Table 1
Anthropometric data and diving habits of the two groups of divers sthma group (n = 22, 1 female) and control group (n = 15, 3 females). Anthropometric data are presented as mean (SD); diving data are presented as median (range)

	Asthma group		Control group	
Age (yr)	28.9	(8.2)	30.8	(8.1)
Height (cm)	180.6	(5.7)	179.2	(7.5)
Weight (kg)	82.9	(12.2)	80.1	(13.9)
Diving history (yr)	4	(1–11)	5	(2–10)
Number of dives	80	(10–220)	90	(25–300)

Table 2
Asthma types and medications in the asthma group (n = 22); 14 suffered from 'allergic asthma'

Asthma type	**No of subjects**
Childhood asthma	8
Mild intermittent	10
Mild persistent	4
Medications	
No medication	12
On medication	10
Inhaled short-acting ß-2 stimulants	4
Inhaled short- and long-acting ß-2 stimulants	2
Inhaled short- and long-acting ß-2 stimulants + inhaled corticosteroids	4
No ß-2 stimulants used before dive	All

All asthmatic divers were free of respiratory symptoms and without any wheeze on lung auscultation in the period 10 to 60 minutes after the dive. The asthma group had statistically significant reductions in FEV_1, FEV_1/FVC%, FEF_{50}; FEF_{75} and $FEF_{25–75}$ after the dive. In the control group, we found significant increases in VC, FVC and FEV_1, while other parameters did not change after the dive. As these data were normally distributed, they are reported as mean (SD) (Table 3).

Using the linear mixed-effects model, we estimated that the asthma group had lower pre-dive results of VC_{in}, FVC, FEV_1 and PEF, but not significantly when compared to the control group. The asthma group had significantly lower pre-dive values of FEV_1/FVC% ($P < 0.01$), FEF_{25} ($P < 0.01$), FEF_{50} ($P < 0.001$), FEF_{75} ($P < 0.01$) and $FEF_{25–75}$ ($P < 0.001$) as compared to the control group. A statistically significant reduction in FEV_1 ($P < 0.01$, Figure 2); FEV_1/FVC% ($P < 0.05$, Figure 3); FEF_{25} ($P < 0.01$), FEF_{50} ($P < 0.01$), FEF_{75} ($P < 0.05$) and $FEF_{25–75}$ ($P < 0.001$) were apparent after the dive in the asthma group as compared to the control group.

The control group demonstrated an increase in VC_{in}, FVC and FEV_1 after the dive, although not significantly as compared to the asthma group. The PEF was initially lower, but not significantly, in the asthma group and did not change significantly after the dive in either group ($P > 0.05$).

When considering the individual changes post-dive in the asthma group, one diver with mild intermittent asthma had a reduction in FVC of 26%, in FEV_1 of 22% and PEF of 23%, as well as a reduction in expiratory flow rates at low lung volumes. The other divers in the asthma group had reductions in the range 3–10% of pre-dive values in FVC and FEV_1 as well as in the other respiratory parameters.

Discussion

Significant reductions in expiratory flows at low lung volumes in divers with asthma were observed in this study after a short pool dive. Theoretically, dysfunction of the small airways may cause an underwater asthma attack, as well as air trapping with possible lung barotrauma, but no diver in the asthma group showed any signs or symptoms of bronchoconstriction after the dive. However, one diver with marked decrease in his pulmonary function after the dive was strongly advised against diving in the future. Before participation in the study, this diver had not requested a medical assessment of his fitness to dive. He had logged only 10 air dives, to depths up to 20 metres over one year and had experienced no breathing difficulties during these dives. More importantly, since other divers in the asthma group did not show such dramatic changes, this single diver could

Table 3

Lung volumes and flows, before and after a single pool scuba dive (mean (SD)); the asthma group had statistically significant reductions in FEV_1, FEV_1/FVC% and expiratory flow rates at low lung volumes after the dive; the control group had significant increases in VC, FVC and FEV_1 after the dive (sse footnote p. 72 for definitions)

	Asthma group			Control group		
Variable	**Pre-dive**	**Post-dive**	***P* value**	**Pre-dive**	**Post-dive**	***P* value**
VC_{in} (L)	5.98 (0.89)	5.92 (0.95)	> 0.05	5.99 (1.34)	6.26 (1.35)	< 0.01
FVC (L)	5.93 (0.91)	5.85 (0.92)	> 0.05	5.96 (1.18)	6.22 (1.21)	< 0.01
FEV_1 (L)	4.44 (0.66)	4.29 (0.77)	< 0.05	5.00 (0.99)	5.17 (1.00)	< 0.05
FEV_1/FVC %	75.27 (6.89)	73.10 (6.63)	< 0.001	84.00 (4.95)	83.20 (4.76)	> 0.05
PEF (L s^{-1})	10.99 (1.87)	11.04 (2.45)	> 0.05	11.43 (2.56)	11.63 (2.51)	> 0.05
FEF_{25} (L s^{-1})	7.18 (2.00)	6.81 (2.13)	< 0.01	9.90 (2.66)	10.29 (2.95)	>0.05
FEF_{50} (L s^{-1})	4.00 (0.93)	3.69 (1.04)	< 0.001	5.82 (1.51)	6.08 (1.40)	> 0.05
FEF_{75} (L s^{-1})	1.66 (0.35)	1.52 (0.40)	< 0.001	2.48 (1.01)	2.52 (0.93)	> 0.05
FEF_{25-75} (L s^{-1})	3.53 (0.82)	3.26 (0.88)	< 0.001	5.09 (1.40)	5.24 (1.49)	> 0.05

have weighted to a small degree the differences observed between the asthma and control groups.

Previous studies have indicated acute as well as long-term changes in pulmonary function amongst saturation and professional divers who had used air or oxygen as the breathing gas.[15] Our findings fit some others, which found that divers had larger lung volumes than a standard reference population and that small airways function might be disturbed in healthy divers after compressed air dives.[16,17] The long-term changes included a reduction in expiratory flows at low lung volumes and a greater increase in FVC than in FEV_1, with an associated reduction in the FEV_1/FVC ratio.[18,19]

It has been speculated that diving itself might induce bronchial hyperresponsiveness by affecting small airways function due to breathing cold, dense gas, which increases airway resistance. The work of breathing is further increased during immersion by the increase in intrathoracic blood volume and consequent small airways closure.[20] In a cross-sectional study of 28 divers and a control group of 31, a higher prevalence of bronchial hyperresponsiveness to histamine among divers than in non-diving matched controls has been reported.[21] In participants with asymptomatic respiratory atopy, diving caused a decrease in airway conductivity.[22] It was also found that scuba diving was associated with the development of early airway hyperresponsiveness in atopic subjects.[23,24]

In another recent study, it was demonstrated that compressed air breathing via a scuba regulator on land in laboratory conditions increased the severity of exercise-induced bronchoconstriction (EIB) in susceptible individuals. These results have implications for those individuals with EIB wishing to dive.[25]

A mechanism that would explain some of the changes seen post-dive in the asthma group might be an inflammatory reaction based on pre-existing airway hyperresponsiveness. Raised exhaled NO and endothelin 1 plasma concentrations, even after short hyperbaric air exposures commonly practised by recreational divers, provide some evidence that an inflammatory reaction and/or repeated oxidative stress or capillary stress failure might cause small airways dysfunction, which needs further investigation.[26,27] However, as these dives were performed in a chlorinated swimming pool, we could not exclude the possibility that the inhalation of toxic chlorination products might have provoked bronchial hyperresponsiveness. Swimming pool chlorine is reported to increase childhood asthma in industrial countries with attendance at a chlorinated swimming pool being associated with higher risks of asthma, airway inflammation and some respiratory allergies.[28,29] Contrary to this, a recent, prospective, longitudinal study suggested that swimming does not increase the risk of asthma or allergic symptoms in British children but rather was associated with increased lung function and a lower risk of asthma symptoms, especially among children with pre-existing respiratory conditions.[30]

According to interviews with divers from the asthma group, none of them noticed that swimming or diving in indoor chlorinated swimming pools provoked any discomfort or triggered asthma attacks. Further studies are needed and dives should be performed in confined open water in order to exclude potential allergic effects from the inhalation of toxic chlorine products. We did not monitor the time needed for the changes in variables to normalise. In future studies, it would be important to perform repeated PFTs at several time intervals after the dives and assess the dynamics of possible changes in variables.

The PFT changes observed in this study were completely different both within and between the two groups. While the asthma group exhibited a reduction in dynamic parameters, the control group showed a slight increase in static parameters, without any impairment of dynamic ones. The increases in VC and FVC in the control group post-dive might be the result of increased muscle strength due to repetitive resistive breathing during the diving activity.

The increase in FEV_1 is less easy to explain. It might be that the increase in VC and total lung volume, with a resultant increase in large airways diameters could compensate for the expected increase in airway resistance. This might increase FEV_1 when compared to pre-dive values, in healthy, well-trained divers.

PEF was initially lower, although not significantly so, in the asthma group and did not change significantly after the dive in either of the groups. This questions the utility of this measure in estimating the status of the respiratory system after a dive. Those parameters that it would be useful to follow up in order to reveal who might be prone to an asthma attack during a dive are not clear. One might well speculate whether such a brief 'diving exercise test' as performed in this study might usefully be included in a 'fitness-to-dive' assessment protocol to assist in improving the estimate of risks of diving in candidates who have asthma.

Some authors do not support the idea that asthma-provocation tests, or exercise on a treadmill are particularly useful for assessing the fitness to dive of people with asthma.[21] More recently, some dive physicians have begun to take a more liberal, informed consent approach in assessing previous or mild asthmatics for diving. It seems reasonable that decisions must be made on an individual basis and involve the patient through informed, shared decision making. We would agree with Dr Neuman's opinion that: "*In contrast to many earlier recommendations, the importance of an open mind and individual assessment are becoming increasingly recognised.*"[3]

There is no 'absolute truth' here. However, more research is necessary and suggested further investigations would be to test divers with asthma in different diving conditions, adding a depth component first in controlled, confined waters, and then adding both depth and temperature variables in controlled open-water dives. This is achievable within the DAN Europe model of participated research and the DAN–UWATEC Diving Safety Laboratory that creates and maintains permanent laboratory research on the safety of recreational diving. We intend to replicate the dives in the deep pools that are available in Belgium and France, followed by exposure in lake-diving conditions.

Conclusions

A single pool scuba dive to 5 metres' depth may impair small airways function in divers with asthma. All divers in the study were free of symptoms after the dive. In one diver in the asthma group, we found reductions of 22–26% in FVC, FEV_1 and PEF of the pre-dive values, and he was advised against further diving. In the other divers in the asthma group, reductions in these parameters were 3–10% of the pre-dive values. More research is necessary in deeper pools and in controlled open-water conditions, adding depth and temperature variables in such controlled dives.

Acknowledgements

We would like to thank diving instructors Sinisa Karalic and Aleksandra Karalic (Nautilus Diving Club, Belgrade), for providing diving equipment and acting as safety divers during the the tests. We also thank diving instructors Zoran Rubinjoni and Bratislav Markovic (Amfora Diving Club, Belgrade), for providing us with access to an appropriate swimming pool.

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Submitted: 19 October 2011
Accepted: 08 April 2012

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The effect of environmental pressure changes on the retentive strength of cements for orthodontic bands

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Abstract

(Gulve MN, Gulve ND, Shinde R, Kolhe SJ. The effect of environmental pressure changes on the retentive strength of cements for orthodontic bands. *Diving and Hyperbaric Medicine*. 2012;42(2):78-81.)

Objectives: The purpose of this study was to evaluate the effect of environmental pressure changes on the retentive strength of orthodontic bands cemented with conventional glass ionomer cement or resin-modified glass ionomer cement.

Materials and methods: Stainless steel bands were cemented to 80 extracted first and second molars in two equal groups comprising conventional glass ionomer cement and resin-modified glass ionomer cement. Each group was randomly divided into two sub-groups of 20 samples each, one sub-group to act as a control, and the other to be used experimentally. After seven days of storage, the experimental groups were subjected to simulated dives to 304 kPa for 3 minutes, 15 times in a pressure pot, after which the force required to deband was tested using a universal testing machine. The data were statistically analysed using Student's t-tests, significance being assumed at $P < 0.001$.

Results: The retentive strength of bands cemented with conventional glass ionomer in the pressure-cycled group was statistically significantly less than that in the control group. No statistically significant difference in strength was found between the two groups cemented with resin-modified glass ionomer.

Conclusions: This study showed that the retentive strength of bands cemented with conventional glass ionomer is reduced after pressure cycling. We suggest that dentists should consider using resin-modified glass ionomer cement for cementing orthodontic bands for patients who are divers and thus likely to be exposed to raised-pressure cycling.

Key words

Barotrauma, diving, scuba diving, dental, teeth

Introduction

'Aerodontalgia' was reported for the first time as an in-flight problem early in the 20th Century. In the 1940s, with the advent of scuba, many in-flight manifestations caused by barometric changes were found to be associated with diving as well. Consequently, the prefix was changed to 'baro-'.[1] The term barotrauma is used to describe a physical injury caused by rapid or extreme changes in barometric pressure. Enclosed areas within the body, such as the middle ear, sinuses and lungs, are particularly affected by barotrauma.[2] Barotrauma is associated closely with Boyle's law, which states that, at a given temperature, the volume of a gas is inversely proportional to the ambient pressure, hence its association with both diving and altitude exposure. Problems arise when the enclosed spaces containing gases cannot expand or contract to adjust the internal pressure to correspond to the outer pressure.[3] Carious, inflamed or necrotic teeth and teeth with inadequate restorations develop barotrauma more readily, which manifests itself as tooth or restoration fracture and reduced retention of restorations.[4,5]

In recent years, it has become increasingly popular to holiday at a tropical destination, often with the opportunity to dive, and recreational sports diving generally has become very popular.[6] The importance of sports dentistry has gained increasing recognition from all members of the sports medical team, thus enabling individual sports people to obtain the latest advice on prevention and treatment of orofacial injuries and related topics.[7]

It is inevitable that the dental practitioner will have patients who participate in sports diving and they should be aware of a number of problems that a diver can experience that are associated with the teeth and related structures. There has been a great increase in patients seeking orthodontic treatment in recent years.[8] Most orthodontic treatment is carried out before or during adolescent years, but with an increasing number of options for orthodontic treatment, adult patients now form a significant segment of practice.[9] When a scuba diver is wearing an orthodontic appliance, a potential danger may result from dislodgement of any component during diving. Although this subject is rarely and only briefly discussed in dental textbooks, it is important for a dentist to be aware of the effect of pressure changes on certain dental materials in terms of their retentive strength.[10]

Orthodontics is the branch of dentistry that specialises in the diagnosis, prevention and treatment of dental and facial irregularities. By placing a constant, gentle force in a carefully controlled direction, an orthodontic appliance can slowly move teeth through their supporting bone to a new, more desirable position. Orthodontic appliances usually consist of attached brackets on anterior teeth and bands on molars. Bands are made from stainless steel and are very similar to a ring. Bands are available in different sizes. They are cemented on molars using luting cement, the most popular of which is glass ionomer cement.[11,12] Newly developed resin-modified glass ionomer cements are currently the subject of consideration.[13]

Table 1
Summary characteristics of the two luting cements studied

Cement type	Composition
Conventional glass ionomer cement	Powder: glass powder, pigments, polycarboxylic acid Liquid: tartaric acid, water, conservation agents
Resin-modified glass ionomer cement	Paste A: fluoroaluminosilicate glass, hydroxyethyl methacrylate, dimethacrylate, pigment, initiator Paste B: Polyacrylic acid, distilled water, silica powder, initiator

What effect pressure variations have on the retention of orthodontic bands is still largely unknown. The present study aimed to clarify whether repeated exposure to the pressure variations typically experienced by divers could have a negative influence on the retentive strength of orthodontic bands cemented with different cements, particularly because luting cements easily trap micro-bubbles of air during manual mixing.[14]

Materials and methods

Two types of cements were studied: a conventional glass ionomer cement (Ketac-Cem™, 3M ESPE, Germany) and a resin-modified glass ionomer cement (GC Fuji Ortho Band Paste Pak™, GC Corporation, Japan) (Table 1).

Eighty extracted human first and second molars without caries were collected and cleaned of large debris. All the teeth were decontaminated in 10% formalin. Then they were promptly rinsed and stored in deionized water at room temperature till used for the study. A hole was drilled through the centre of each tooth near the root furcation area and a 0.9 mm stainless steel wire was placed in the hole to resist pulling of the tooth out of embedded medium at debanding. All samples were then mounted in a block of self-curing acrylic resin, below the amelocementum junction with the long axis vertical. The exposed crowns were polished with fluoride-free dental prophylactic paste to remove any fine debris.

Stainless steel orthodontic bands with micro-etched fitting surfaces (3M Unitek) were selected and adapted for best fit to the crown of each tooth. Orthodontic lingual sheets were welded on the buccal and lingual surfaces of the bands. Samples were randomly divided into two equal groups of 40 each according to the type of orthodontic cement used:
Group 1: Conventional glass ionomer cement;
Group 2: Resin-modified glass ionomer cement.

The preparation of the cements was conducted exactly according to the manufacturers' instructions. The cement mix was applied directly to the band-fitting surface, and the bands were seated by using a band pusher with hand pressure. Excess cement was removed from the occlusal and cervical margins of the band so that it would not influence the test results. All the specimens were allowed to bench cure for 5 minutes, and then stored in 0.9% NaCl solution for 7 days at 37^{O}C. Each group was randomly divided into two sub-groups of 20 samples, one sub-group to act as a control and the other to be used experimentally.

All the experimental samples, in open glass containers, were placed in a pressure pot (Puneet Industries, India). During the simulated dives, pressure was changed at a rate of 101 kPa min^{-1} during compression and decompression. The maximum pressure applied was 304 kPa, equivalent to a depth of 30 metres' sea water (msw), and was maintained for 3 minutes. Fifteen pressure cycles were repeated one after the other. This basically corresponds to the activity of a typical recreational diver during a 10-day holiday.[15]

The force necessary to dislodge the orthodontic bands was evaluated using a universal testing machine (Star Testing System, model no STS248). Each specimen was loaded into the inferior vice grip of the universal testing machine and secured in place by tightening adjustable screws to fix the acrylic block in position. Stainless steel loops that engaged through the welded lingual sheaths on the buccal and lingual side of the bands were attached to the superior self-tightening wedge action grip of the universal testing

Figure 1
Retentive strength testing on universal testing machine

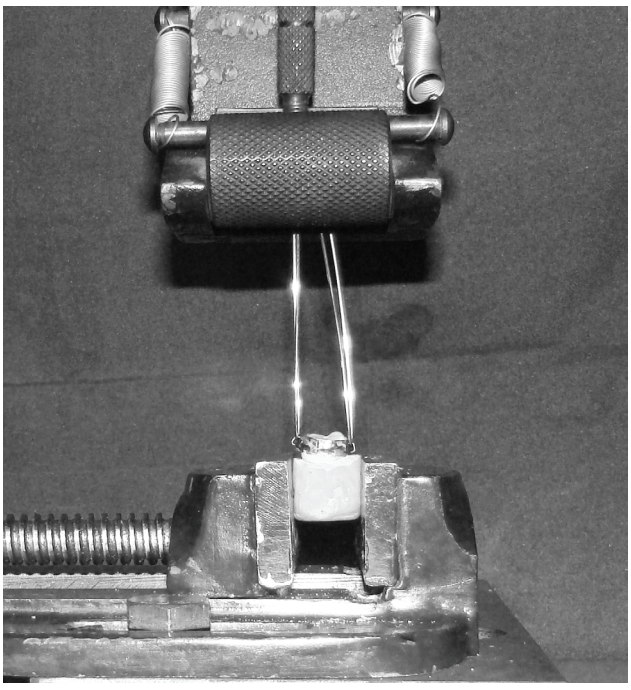

Table 2
Retentive strength of bands cemented with two types of luting cements, for pressure-cycled and control groups (mean (SD))

Cement type	Control group	Pressure-cycled group	*P* value
Conventional glass ionomer cement	1.496 (0.313)	0.975 (0.348)	< 0.001
Resin-modified glass ionomer cement	1.810 (0.392)	1.764 (0.419)	0.722

machine (Figure 1). The universal testing machine was programmed to perform at a crosshead speed of 1 mm min^{-1} and testing proceeded for each sample until the band was removed from the tooth. The maximum debanding force in newtons (N) was interpreted from the stress-strain curve as the maximum force recorded during debanding.

The band was cleaned with a sickle scaler and pumice, cut with a scissor, and laid out flat. Its length and width were measured using vernier calipers and, thus, its area was determined in mm^2. Dividing the debanding force (N) by band area (mm^2) resulted in the band strength expressed in megapascals (MPa).

The statistical analysis was performed using a commercially available software programme SPSS version 1.5. Descriptive statistics that included mean and standard deviation were calculated. Student's t-test was applied to determine whether significant differences existed among the control and experimental sub-groups for both cements. Significance for all statistical tests was predetermined at $P < 0.001$.

Results

A summary of the results of retentive strength of orthodontic bands is presented in Table 2. The retentive strength of bands cemented with conventional glass ionomer in the pressure-cycled group was significantly less than that of the control group ($P < 0.001$), while no significant difference was found between the pressure-cycled group and the control group for bands cemented with resin-modified glass ionomer ($P = 0.722$).

Discussion

It is important for an orthodontist to be aware of the effect of pressure changes on certain dental materials in terms of retentive strength, as the potential danger resulting from dislodgement of orthodontic components during a dive is obvious. This is particularly important for materials that are mixed by hand, such as band cements, as during the mixing process, air may become incorporated into the mixture, forming voids which would be subject to expansion and contraction with environmental pressure changes.[14] No study has examined previously the effect of environmental pressure changes during diving on the retentive strength of orthodontic bands.

In a study examining the long-term effects of barometric pressure changes on the dental health status of German naval personnel, the teeth of personnel working under changed atmospheric pressure (navy divers and frogmen) deteriorated over 10 years at significantly higher rates than did those of personnel working under normal atmospheric pressure (submariners).[16] These findings suggest that sustained exposure to barometric pressure plays a role in dental deterioration.

Previous in vitro studies have shown that pressure changes can affect the retention of restorations and crowns.[15,17,18] Exposure to pressure cycling of full-cast crowns resulted in decreased retentive strength in those cemented with zinc phosphate and conventional glass ionomer cements, whereas no significant effect was found in crowns cemented with resin cement.[19] The authors linked the failure associated with zinc phosphate and conventional glass ionomer cements to porosities that had been generated during mixing and expansion or contraction of these micro-bubbles during pressure cycling, which eventually led to disruption and weakening of the cement layer.[19] In another study, micro-cracks appeared as a result of volumetric contraction in luting cements.[20] When subjected to the effect of pressure cycling, such cracks may produce tensile stresses that exceed the cohesive and adhesive strength of the material, resulting in significant reduction in tensile bond strength.

Although the exact mechanisms of barodontalgia and barotrauma are not known, the air trapped beneath a restoration or in luting cement may be a factor.[3] During diving, dental barotrauma usually occurs while ascending. Upon returning to the surface after completing the dive, the diver may report fracture of teeth or dislodgement of restorations.[4] Differences in the physical properties of the breathing gas mixture used during deep sea diving may contribute to barodontalgia.[17] However, dislodgement of restorations can occur while descending.[3]

Other authors have concentrated on the possible variations in volume in micro-bubbles within insufficiently filled restorations, cements or root canals caused by rapid pressure changes. Trapped air micro-bubbles will be compressed on descent and will expand on ascent.[15,19] Teeth may actually implode during descent or explode upon ascent.[21] It is possible that dislodgement of restorations may develop as a result of micro-leakage following mechanical failure of the luting cement.[18] As military and professional divers are more likely to be subjected to rapid manoeuvres and extreme situations than are civilian divers, it can be assumed that they are more vulnerable to pathological consequences of rapid pressure changes.[4]

In the present study, the retentive strength of bands cemented with resin-modified glass ionomer cement was not significantly affected by pressure cycling to 304 kPa, whereas conventional glass ionomer cement was weakened. This could be attributed to the higher tensile strength, lower elastic modulus and greater amount of plastic deformation that can be sustained before fracture occurs with resin-modified glass ionomer cement compared to conventional glass ionomer cement.[22] The pressure chosen (304 kPa) is equivalent to that to which a scuba diver is subjected at a depth of 30 msw. However, the pressure was held for only 3 minutes, whereas in real life a diver would spend a much longer time underwater. It would be interesting to investigate the behaviour of cements beyond 304 kPa and for longer durations of time.

What effect the pressure variations that divers are exposed to have on the retention of other dental components where luting cements are used is still largely unknown. Also only one brand of each type of cement was tested in this study; further studies with other materials are recommended. Surface details of dislodged bands under scanning electron microscopy could provide better insight into the findings in the present study. Finally, it must be noted that in vitro studies are limited in predicting the success of a material or technique in clinical use.

Conclusions

Resin-modified glass ionomer cement for orthodontic bands retains its strength after pressure cycling to 304 kPa better than conventional glass ionomer cement. Therefore, this type of cement should be used for patients who are exposed to marked variations in environmental pressure, such as recreational and professional divers, balloonists, aviators who fly in unpressurised cabins and mountaineers.

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Submitted: 26 October 2011
Accepted: 12 January 2012

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Hyperbaric oxygen therapy for vaso-occlusive crises in nine patients with sickle-cell disease

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Abstract

(Stirnemann J, Letellier E, Aras N, Borne M, Brinquin L, Fain O. Hyperbaric oxygen therapy for vaso-occlusive crises in nine patients with sickle-cell disease. *Diving and Hyperbaric Medicine*. 2012;42(2):82-84.)

Introduction: Vaso-occlusive crisis (VOC) is the most frequent complication of sickle-cell disease and is associated with significant acute bone pain.

Objective: To evaluate the feasibility and efficacy of hyperbaric oxygen therapy (HBOT) for severe VOC.

Methods: We report our retrospective experience with HBOT in VOC in nine patients and 15 HBOT sessions.

Results: All nine patients had received appropriate conventional treatment prior to HBOT. Pain scores using a Visual Analog Scale (0 to 10) determined whether HBOT was effective or not in improving symptoms. While no change in pain score occurred before the HBOT session, pain scores fell significantly from 3.3 prior to HBOT to 1.9 24 hours after HBOT ($P = 0.002$). While morphine dosage increased before HBOT (median morphine dose 23 mg per day and 35.95 mg per day respectively on Day -2 and Day -1, $P = 0.04$), the median morphine dose one day after HBOT (Day +1 23 mg per day) tended to be lower than Day -1 ($P = 0.08$), and decreased to zero 2 days after HBOT ($P = 0.004$). Two patients had ear pain during compression, requiring rapid interruption of the HBOT session, although neither patient had any sequelae.

Conclusion: HBOT is feasible in sickle cell disease and appears to be effective in reducing the pain of VOC rapidly.

Key words

Haematology, hyperbaric oxygen therapy, pain, medical conditions and problems

Introduction

Vaso-occlusive crisis (VOC) is the most frequent complication of sickle-cell disease (SCD) and is associated with severe bone, thoracic and/or abdominal pain. It is the leading cause of hospitalisation among patients with SCD, as well as the leading cause of acute chest syndrome and of death.[1] It is linked to sickling disorders and to vaso-occlusion and can be triggered by environmental factors such as acidosis, cold, dehydration, hyperthermia, infection and hypoxia.[1]

Hyperbaric oxygen therapy (HBOT) has previously been used to manage vaso-occlusive crises associated with SCD, but while some authors found it to be effective others did not.[2–5] Unfortunately these studies are very dated even though HBOT continues to be used in certain strongly endemic regions.[5] In a recent study, Medahoui related his experience of HBOT in 15 patients with VOC, in which a decrease in the degree of pain was obtained in 12 cases after the first session of HBOT; patients had an average of four successive sessions.[5]

We report our retrospective experience with HBOT for VOC in nine patients followed in a French internal medicine department.

Methods

Patients were eligible for HBOT if they had VOC that necessitated hospitalisation and their symptoms were refractory to other conventional therapy (hydration, normobaric oxygen therapy, analgesics). The research project was reviewed and approved by the local Institutional Review Board of Ile-de France (Comité de Protection des Personnes IdF X) and informed consent of the patients was obtained prior to HBOT. The HBOT sessions were conducted at the Val de Grace Hospital, Paris. Patients were treated at a pressure of 253.3 kPa (2.5 atmosphere absolute), each session lasting a total of 90 minutes, including a 15-minute compression and decompression, and a 60-minute treatment phase. Each patient could have up to four successive HBOT sessions. Each session was analysed independently. When an adverse event occurred, the HBOT session was interrupted, but data were still analysed as if the patient had been treated (intention-to-treat). Visual Analog Scale (VAS) score (0–10), daily morphine dose, C-reactive protein (CRP), haemoglobin and lactic dehydrogenase (LDH) levels were compared two days (Day -2) and one day (Day -1) before HBOT and one and two days (Day +1 and Day +2) afterward. VAS scores, morphine doses, CRP and LDH were compared using a non-parametric test (Mann-Whitney-Wilcoxon test). Statistical analysis was performed using R software <http://www.R-project.org/>.

Results

Nine patients presenting between March 2006 and July 2007 and receiving a total of 15 HBOT sessions were analysed, . No further patients have been referred for HBOT since that time. The characteristics of the patients and HBOT sessions are shown in Table 1. Patients were hospitalized with a median stay of 3 days (range 0–11).

Table 1
Patient characteristics and hyperbaric oxygen therapy (HBOT) details; ACS – acute chest syndrome; SS – homozygous SS sickle cell disease

Patient	Age (yr)	Sex	Type	ACS	Hydroxyurea	Haemoglobin (g L^{-1})	HBOT sessions	Complications of HBOT
1	24	M	SS	Yes	No	80	1	Barotraumatic otitis
2	18	M	SS	No	No	80	2	None
3	19	M	SS	Yes	No	80	1	None
4	19	M	SS	No	No	100	2	None
5	24	M	SS	No	No	100	2	None
6	22	F	SS	No	Yes	80	4	Barotraumatic otitis 4th HBOT
7	19	M	SS	No	No	100	1	None
8	18	M	SS	No	Yes	90	1	Paraesthesiae
9	21	M	SS	Yes	No	75	1	None

The median VAS score fell significantly (Figure 1) from 3.3 at Day -1 to 1.9 at Day +1 (P = 0.002) and 1.4 at Day +2 (P = 0.002; data not shown). Before HBOT, median VAS score did not change significantly between Day -2 and Day -1 (VAS of 3 and 3.3 respectively, P = 0.08).

Before HBOT, the median dose of morphine increased between Day -2 and Day -1 (23 mg per day and 35.95 mg per day respectively, P = 0.04). The median morphine dose received during the first 24 h after HBOT (Day +1, 23 mg per day) tended to be lower than the dose received during Day -1 (P = 0.08), and decreased to a median of zero after 48 h (Day +2) (P = 0.004, Figure 2).

No significant changes were noted in CRP, haemoglobin or LDH levels.

Two patients had ear pain during compression, requiring rapid interruption of the HBOT session, with no sequelae in either patient. One patient had paraesthesiae at the end of a session, although these disappeared spontaneously. No adverse events occurred in the other six patients. No transfusions, including exchange transfusions, were necessary after HBOT, except for one patient whose session had been interrupted by ear pain.

Discussion

The present results suggest that HBOT appears effective against pain associated with VOC, as witnessed by the drop in median VAS score from 3.3 at baseline to 1.9 at 24 hours and 1.4 at 48 hrs post HBOT, whereas this score did not change prior to HBOT. The patients had serious

Figure 1
Changes in mean pain score in patients before and after hyperbaric oxygen therapy (HBOT); median scores are shown for two days prior to HBOT (Day -2), the day before the session (Day -1) and the day after the session (Day +1)

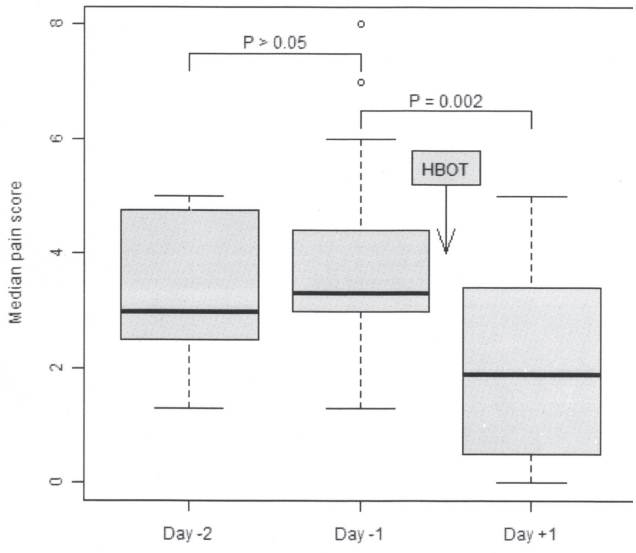
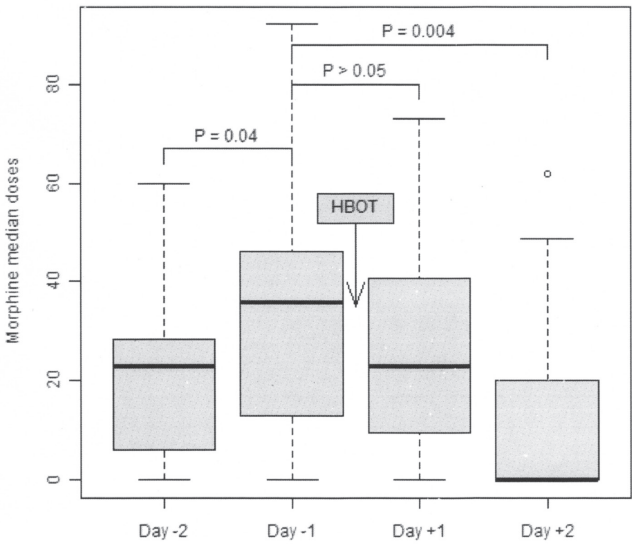


Figure 2
Changes in morphine dose in patients before and after hyperbaric oxygen therapy (HBOT); median doses are shown for two days prior to HBOT (Day -2), the day before the session (Day -1) and the day after the session (Day +1)



VOC episodes and, in most cases, had been hospitalised for several days, during which conventional treatments had proven inadequate. While morphine remains the predominant treatment for pain associated with VOC, it is sometimes difficult to increase the dose sufficiently without risking alveolar hypoventilation and chest syndrome. Initial treatment of the nine patients proved insufficiently effective, as shown by the lack of improvement (Figure 1) and increasing doses of morphine (Figure 2) before HBOT.

Few data have been published on the use of HBOT in this setting, although one series (only reported in a textbook) has shown some efficacy.[5] In the present series, HBOT was well tolerated. Barotraumatic otitis was the main complication and was successfully managed by decompression. No serious adverse events occurred in these nine patients, although the safety of HBOT cannot be fully ascertained given the small size of the series. Serious adverse effects of HBOT in other indications include rare cases of seizures, pneumothorax, and asthma attacks. These adverse effects necessitate close monitoring during HBOT sessions, and resuscitation equipment must be on hand.

HBOT could be beneficial in several ways during VOC. Although haemoglobin S polymerisation and red cell sickling under deoxygenated conditions are central to the pathophysiology of this disease, emerging evidence indicates that initial events may involve sickle red cell–endothelial interaction as one of the major potential initiating mechanisms in vaso-occlusion with implication of adhesion proteins.[6] HBOT would tend to limit hypoxaemia and, thus, sickling. Hyperbaric therapy also appears to reduce cell stickiness by down-regulating adhesion proteins.[7–10] Finally, HBOT seems to increase the release of nitric oxide (NO) and NO synthetase, which in turn could compensate for the reduction in NO that promotes vaso-occlusive complications in SCD.[5,11–13] HBOT also reduces neutrophil adhesion to bovine aortic endothelial cells.[7]

While the present study does not formally establish the efficacy of HBOT in VOC associated with SCD, it does, nonetheless, show the feasibility of this treatment. These preliminary observations warrant a prospective, randomised trial. Indeed, HBOT may be indicated for patients with refractory VOC, especially when transfusion therapies are not possible, such as in cases of allo-immunisation.

Acknowledgements

We thank Saad Rouaghe and Anne-Sophie Morin for their assistance in conducting this study.

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Submitted: 01 September 2011
Accepted: 20 March 2012

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Technical report

Hyperbaric critical care patient data management system

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Abstract

(Kronlund P, Olsson D, Lind F. Hyperbaric critical care patient data management system. *Diving and Hyperbaric Medicine*. 2012;42(2):85-87.)

A patient data management system (PDMS) has been used for years in the intensive care unit (ICU) at the Karolinska University Hospital to provide bedside or remote clinical patient documentation and information. Data from monitors, mechanical ventilators and syringe pumps are fed into a central clinical information management system to monitor, display trends and record data of vital parameters, ventilator settings and drugs. In order to continue routine critical care monitoring and recording during hyperbaric oxygen therapy (HBOT), without endangering the safety demands of hyperbaric procedures, we have modified the PDMS system for hyperbaric use. Via an ethernet box placed inside the chamber, data is transmitted to the Clinisoft™ system through the local area network. By standardised risk-analysis procedures, in close cooperation between the hyperbaric and biomedical engineering departments, the chamber producer and the notifying body (Germanischer Lloyd), the ethernet box was modified to receive full safety approval by all parties. The PDMS is now functioning routinely during HBOT for intensive care patients so that data can be seen bedside and followed on-line in the ICU. Data are also continuously stored on the clinical information management system for later clinical or research purposes. Work continues to obtain CE approval for hyperbaric use for modern syringe pumps and mechanical ventilators connected to the PDMS system. Improved documentation of ICU care will improve quality of care during HBOT and facilitate research and development in hyperbaric medicine.

Key words

Hyperbaric oxygen therapy, patient monitoring, equipment, computers, data, risk assessment, review article

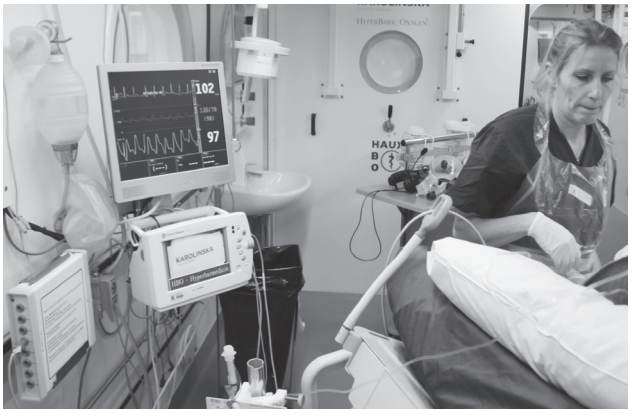
Introduction

Treatment of critically-ill victims of carbon monoxide poisoning, air embolism, traumatic crush injuries and severe, necrotizing soft-tissue infections with hyperbaric oxygen therapy (HBOT) calls for an array of technical medical equipment to be modified for hyperbaric use.[1–3] Intubated, septic patients with multi-organ failure require not only experienced intensive care staffing but also a number of technical and equipment considerations. Mechanical ventilation in the hyperbaric environment requires close monitoring of blood gases and end-tidal CO_2. Vital parameter monitoring includes electrocardiogram, intravascular pressures, pulse oximetry and urinary output. Critically ill patients have need for continuous fluid, electrolyte and drug infusions. Blood products are frequently given postoperatively. The intubated patient has need of sedation and suctioning. Syringe pumps are required for accurate delivery of potent drugs such as vasopressors and insulin.

Medical technical devices in the Karolinska Central Intensive Care Unit (ICU) such as ventilators, infusion/syringe pumps and patient monitoring systems are nowadays connected to a clinical information management system (Clinisoft; Centricity™ Critical Care, GE Healthcare). The ethernet box (E-box) placed bedside in the intensive care unit converts the serial communication output from the connected medical technical equipment to a TCP/IP protocol and transmits the data to the Clinisoft system through the normal computer network. The E-box has a total of eight available connectors for input of data from mechanical ventilator, syringe pumps, etc. Data can be seen bedside as well as in other, more distant settings, for example, during ward rounds and, in a later phase, as stored data for trend analyses or research. The nurse and doctor can also manually enter dispensed medications, etc., into the system from their computer station. In the past, a blank period with two or

Figure 1

Critical care nurse and intubated patient in the hyperbaric chamber. ethernetbox (lower left) connected to a Datex monitor (GE healthcare); the slave screen above the monitor displays ECG, arterial blood pressure and oxygen saturation; E-box has seven vacant connectors for future use.



more hours' loss of information occurred due to disconnected PDMS monitoring and recording as the patient went to the operating theatre, to the radiology department or for HBOT.

We have now modified an E-box for use under hyperbaric conditions in the Karolinska hyperbaric chamber (HAUX 3500) to allow continuous routine monitoring and recording with the PDMS during critical care HBOT (Figure 1).

Risk assessment

A risk-analysis group was created in cooperation between the hyperbaric and biomedical engineering departments of the Karolinska University Hospital to investigate whether the E-box could be safely used under hyperbaric conditions. The participants were chosen from different professions with the aim of combining hyperbaric knowledge with that of biomedical engineering and quality assessment. A risk analysis was performed according to the ISO 14971 standard with pressure test protocols, technical documentation of modifications for its intended use and an evaluation of possible consequences which may occur when using the E-box in the hyperbaric environment. Several European Community directives and norms were considered by the group in this process.[4–7]

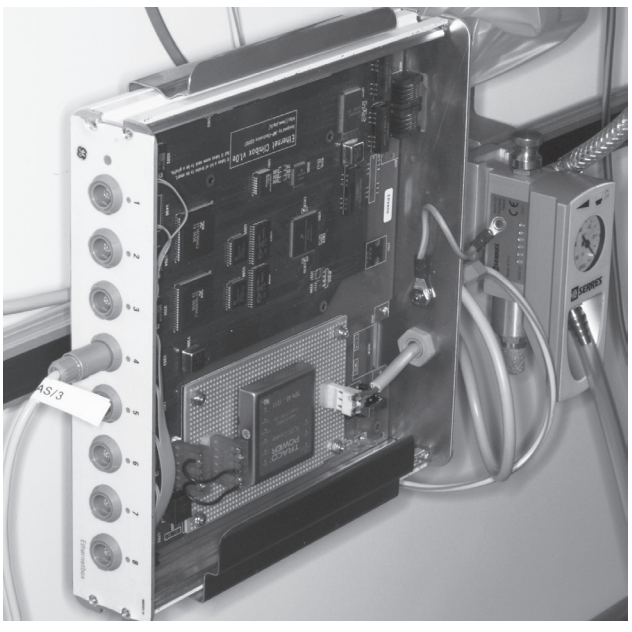
Modifications and tests

The power supply unit was modified to work with the 12V current in the chamber (Figure 2). A standard DC 12V/DC 5V reducer was chosen to function in the hyperbaric environment with a shielded metal case, over-temperature protection and an extended operating temperature range (-40^{O}C to +85^{O}C). No increased risk of spark formation from the power supply unit was recognised by the risk-analysis group. The normal pressure for HBOT in ICU patients is 1.8 bar gauge pressure (284 kPa) with control of temperature and humidity through the air conditioning system in the chamber. The intended use inside our hyperbaric chamber could possibly involve larger changes in environmental conditions, i.e., pressure, humidity, temperature and oxygen levels. The maximum pressure in our ICU chamber is 3.0 bar gauge pressure (405 kPa, corresponding to 30 metres' fresh water).

The E-box was modified for its intended use by removing the circuit breaker and moulding the power supply cable into the chassis with a DC 12V/DC 5V reducer in order to avoid sparks (Figure 2). The power cord that is connected to the E-box is itself modified to suit the standard conduit in the chamber system. All cables are only attached and detached during normobaric ambient conditions, before and after HBOT. Functional tests of the E-box were performed electrically by the Biomedical Engineering Department during repeated, prolonged (60 minutes) and rapid bounce test pressurisations to 608 kPa as well as during 405 kPa pressurisations in the regular ICU chamber setting without any equipment problems or failures. An abbreviated Swedish text manual for hyperbaric use of the E-box, is pasted on its lid as shown in Figure 1.

Figure 2

Ethernet mainboard (lid removed); the DC 12V/DC 5V reducer is the rectangular device in the lower part of the box connected to the 12V cable at right and flat 5V cable at left



Risk acceptance

Risk acceptance was thus accomplished and the advantages of the intended use of the E-box in the hyperbaric chamber were judged to exceed the identified risks. The major risk found was that the E-box would transfer corrupt or distorted information to Clinisoft, which is a risk taken by all ICU departments with the use of a PDMS system. The E-box can be used in the intended environment in the Karolinska ICU chamber without having to change the user manual, with the exception that all cables should be connected before HBOT. Deviations in PDMS function are reported to the Karolinska Biomedical Engineering Department, who reports to the producer who in turn would inform other users. So far this has not occurred during the three-year period Clinisoft has been in use at the Karolinska.

Discussion

Hyperbaric oxygen treatment of critically ill patients necessitates special considerations regarding technical solutions. By having close cooperation between the hyperbaric and biomedical engineering departments at the Karolinska, the chamber producer (HAUX Life Support) and the notifying body (Germanischer Lloyd), we have been able to gain full approval for a modified E-box for hyperbaric use. We have chosen to bring the equipment into the hyperbaric treatment room to allow best possible ICU care by the accompanying doctor and nurse, who can also

Figure 3
Nurse inside tender at the PDMS computer workstation

access the PDMS system through their respective computer workstations inside the hyperbaric ICU chamber (Figure 3). At present, ventilator settings and given drugs are entered manually into the PDMS system. Most of these data will be automatically recorded in the future via the bedside chamber workstation. Blood gases taken and sent out of the chamber for analysis are automatically entered into the PDMS system.

The E-box is CE marked as a registered medical device and approved for its intended use by the manufacturer (General Electric Healthcare) according to the directive for medical products (MDD) 93/42 EC.[4] CE-marking indicates that a product conforms to a European technical specification called a 'Harmonised European Norm'. Once a manufacturer has demonstrated that the product complies with the requirements of the relevant norm, he can affix the CE marking to the product, its packaging or delivery documentation. According to EU and Swedish regulations (SOSFS 2008:1[7]), any modification for hyperbaric use will alter the product so that it is no longer CE marked, but is to be looked upon as an 'in-house production'. As such, the modified E-box has received full safety approval for hyperbaric use at the Karolinska by the notifiying body Germanischer Lloyd. However, because the work was carried out in cooperation with the chamber manufacturer and the notifying body, the chamber and E-box in use at the Karolinska remain approved CE-marked medical devices.

Work is in progress with other manufacturers to achieve hyperbaric compatibility and, if possible, full CE approval for syringe pumps and ventilators. This will allow routine intensive care monitoring to continue without interruption during HBOT, improving the quality of patient care, and facilitate research and development in hyperbaric medicine.

Acknowledgements

The authors wish to thank Farhang Naderi and Niklas Blomberg, Biomedical Engineering Department and Bengt Eriksson, MD, Medical Director of Hyperbaric Medicine, Department of Anaesthesia, Surgical Services and Intensive Care, Karolinska University Hospital, Stockholm, Sweden.

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Submitted: 30 November 2011
Accepted: 25 January 2012

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Case reports

MRI features of spinal cord decompression sickness presenting as a Brown-Sequard syndrome

Pierre Louge, Emmanuel Gempp and Michel Hugon

Abstract

(Louge P, Gempp E, Hugon M. MRI features of spinal cord decompression sickness presenting as a Brown-Sequard syndrome. *Diving and Hyperbaric Medicine*. 2012;42(2):88-91.)
Decompression sickness often manifests as central nervous system impairment. We report a 49-year-old woman who developed an unusual case of spinal cord decompression sickness presenting as complete Brown-Sequard syndrome. Initial MRI revealed increased signal intensity in the left side of the cervical cord at the level of C2–C3. A second MRI at 10 days post-injury showed signal abnormalities corresponding to an infarction in the posterior spinal artery territory. After two weeks of intensive treatment with various HBOT regimens, the clinical outcome was still poor, but at six months after the injury her neurological condition was greatly improved, with only slight impairment of proprioception on the left when walking remaining.

Key words
Decompression sickness, decompression illness, central nervous system, radiological imaging, case reports

Introduction

Decompression sickness (DCS) is an acute disorder caused by the development, during decompression, of bubbles formed by inert gas (usually nitrogen) previously dissolved in the tissues. The spinal cord is frequently involved and the clinical presentation varies according to the affected site.[1–3] Neurological symptoms may vary considerably from subjective sensory to complete motor deficiency with sphincter dysfunction. The pathophysiological mechanisms of spinal cord DCS have not yet been fully identified; several hypotheses have been raised, such as venous infarction generated by bubbles, development of autochthonous bubbles within nervous tissue of the spinal cord or embolisation of arterial bubbles.[4] The first hypothesis is the best documented, since the alteration of spinal cord venous drainage that results from the obstruction of the epidural venous system has been shown experimentally.[5] Secondary immuno-inflammatory processes on endothelial activation, as well as the start of blood platelet aggregation and coagulation, would contribute to worsening of the phenomenon.[4] In the present report, we describe a severe case of spinal cord DCS presenting as a Brown-Sequard syndrome with MRI findings suggestive of an occlusion of the posterior spinal artery.

Case report

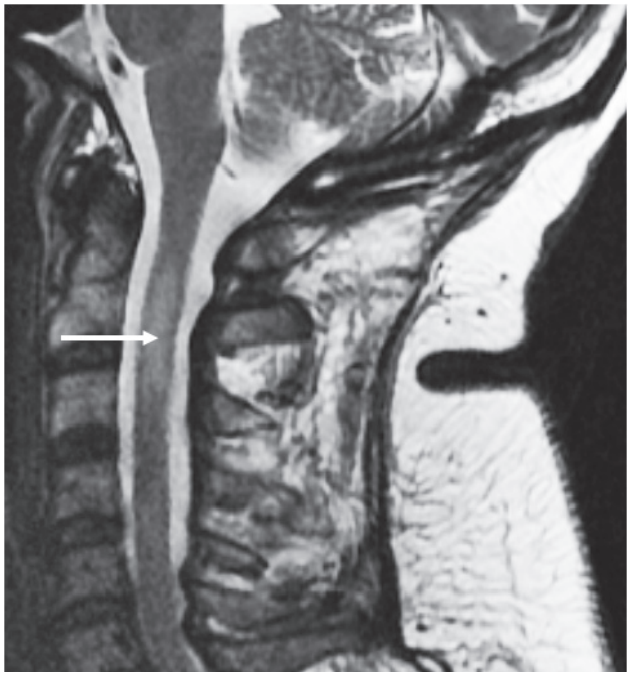
A 49-year-old female, experienced recreational diver was referred to our hyperbaric facility with motor weakness affecting the left lower limb that developed after an uneventful scuba dive to a maximum depth of 55 metres' sea water (msw) for 20 minutes, without violation of the decompression procedure given by her dive computer. The day before, she had performed two repetitive scuba dives to a maximum depth of 50 msw. The patient was on medication for hypertension and was a non-smoker.

Close questioning revealed that, after the dive in question, she felt faint and had difficulty climbing into the boat. This was followed by an episode of vomiting. One hour later, she complained of transient cervical pain with tingling sensations in her left arm accompanied by muscle weakness gradually developing in the ipsilateral lower limb. She was placed on normobaric oxygen and transferred by helicopter for clinical evaluation and initiation of recompression therapy.

At presentation, her level of consciousness and cognition and cranial nerves were normal. Neurological examination revealed incomplete paralysis of the left lower limb for L2–L5 myotomes (scored 3/5 according to the American Spinal Injury Association) with impaired proprioception at the same level. Paraesthesiae in her left arm persisted without objective sensory signs. Pin-prick and temperature sensations were altered on the right side below the T5 level. The left Babinski reflex was positive while knee and ankle deep tendons reflexes were increased but symetrical. The remainder of her physical examination was normal.

Four hours after surfacing, she underwent a COMEX 30 hyperbaric treatment (total 300 minutes). She received adjunctive therapy consisting of methylprednisolone (80 mg intravenously), aspirin (250 mg orally) and lignocaine (1 mg kg^{-1} intravenously). However, the abnormalities persisted, and during the night her condition deteriorated. Repeat examination revealed increased weakness of the left arm

Figure 1
Sagittal T2-weighted magnetic resonance image at 24 hr post-injury showing a hyperintense intramedullary lesion at the level of C2–C3 in a diver with decompression sickness



and leg (2/5), with hypoaesthesia to light touch below the T3 dermatome and anaesthesia to pain and temperature on the right side at the C7 level. Anal sphincter tone and micturition were also disturbed. These findings were consistent with a typical Brown-Sequard syndrome on the left. Laboratory data were unremarkable. A transcranial ultrasonography Doppler with agitated saline was performed and did not detect a right-to-left shunt.

According to our protocol for the treatment of serious DCS, hyperbaric therapy was continued the following day with a US Navy Treatment Table 6 (extended). On the third day she underwent another COMEX 30 treatment table, followed by 10 daily sessions of hyperbaric oxygen therapy at 254 kPa for 90 min in combination with intensive physiotherapy.

An initial MRI examination (3.0 T system, GE Medical system, HD TX) of the whole spinal cord was undertaken within 24 hours of the injury. This showed an area of hyperintense signal in the left lateral cervical spinal cord on sagittal and axial T2 imaging at level C2–C3 consistent with an ischaemic lesion (Figures 1 and 2). A repeat MRI performed one week later, revealed bilateral signal abnormalities in the posterior spinal horn, corresponding to a spinal cord infarction in the posterior vertebral artery territory (Figures 3 and 4). After two weeks, the clinical outcome remained poor and she was transferred to a functional rehabilitation centre. However, when reviewed six months after the accident, her neurological condition was greatly improved with complete motor recovery, resolution of sphincter dysfunction and persistence of pain sensation and with only a slight impairment of proprioception on the left when walking. The follow-up MRI did not to show any residual abnormalities.

Discussion

Spinal cord DCS is not unexpected, but our case is unusual for the following reasons:

- the clinical picture of Brown-Sequard syndrome has rarely been described after scuba diving;
- the repeated MRI scans demonstrated imaging features of diving-related spinal myelopathy that correspond to a variety of pathophysiological possibilities, and

Figure 2
Axial T2-weighted magnetic resonance image at 24 hr post-injury showing a hyperintense intramedullary lesion at the level of C2–C3 in a diver with decompression sickness

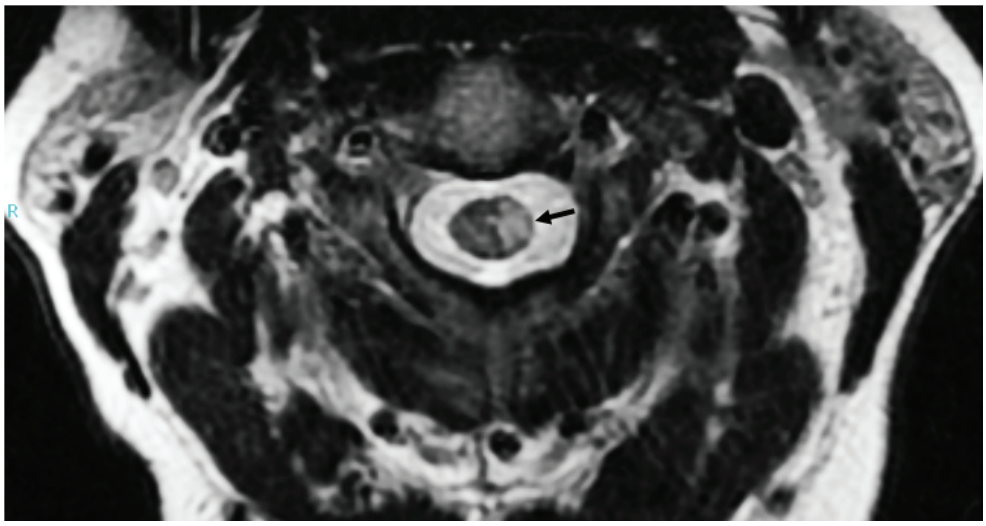
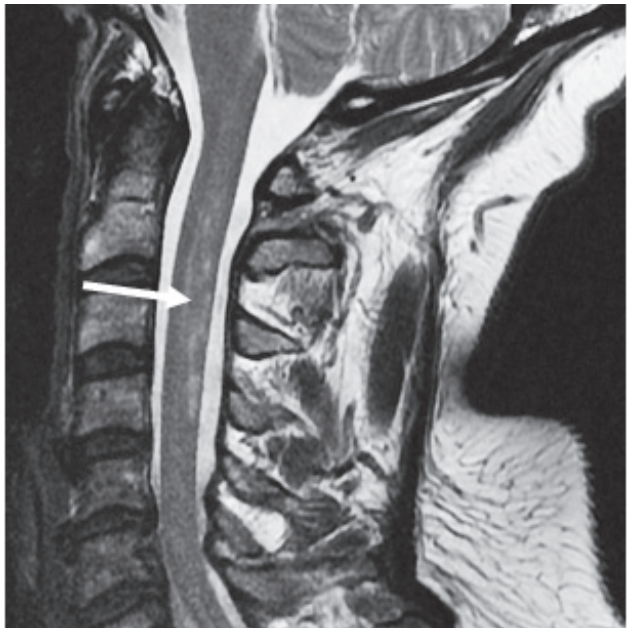


Figure 3
Sagittal T2-weighted MRI in the same diver a week later showing high intensity oedema of the gray matter of the C3 spinal cord with minimal enhancement at the level of C2–C3



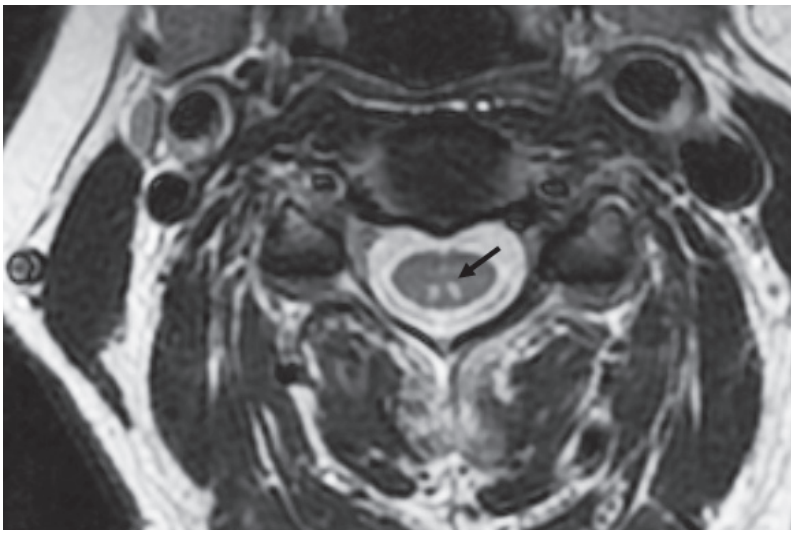
- the clinical outcome was ultimately favourable despite severe initial presentation and apparent poor response to HBOT.

To our knowledge, there are only three previously reported cases of Brown-Sequard syndrome attributed to DCS.[6–8] Although the distribution of symptoms was quite different with partial presentation in these cases, MRI findings were consistent with the level of neurological deficit observed by the authors. In these previous cases, spinal cord lesions were described as focal hyperintense signals on T2-weighted images localised in the lateral and posterior white matter columns of thoracic cord segment as generally observed in past spinal cord DCS imaging studies.[6–12]

In the present report, the first MRI examination showed a diffuse enlargement and increased water content in the left lateral cervical white matter, suggesting the predominant role of oedema and inflammation in the initial part of the pathophysiological process. The most likely mechanism responsible for this myelopathy appears to be spinal cord injury resulting from congestion of the epidural vertebral venous system by nitrogen gas bubbles.[5,13] On the other hand, the repeat MRI depicted a predominance of gray matter involvement, a radiological appearance suggesting infarction in the posterior spinal artery territory.[14,15] This localisation has been described rarely in spinal cord DCS presentation and may be associated with a poor prognosis.[12,16]

Thus, we might assume that the arterial nature and extent of ischaemic damage have two possible origins: either an initial appearance of oedema reflective of ischaemia-reperfusion and cellular dysfunction following arterial occlusion, or a secondary arterial impairment caused by the vasogenic oedema initiated by venous infarction. The severe initial presentation of our patient reflected a likely worse prognosis than the general trend in spinal cord DCS, as evidenced in previous reports.[2,17] However, clinical functional at six months was surprisingly good after treatment encompassing several hyperbaric sessions using heliox mixture, intravenous lignocaine and early intensive physiotherapy and rehabilitation. In practice, the identification of the most important therapeutic factors in preventing severe disability in divers with severe DCS

Figure 4
Axial T2-weighted MRI in the same diver a week later demonstrating high intensity oedema of the "H" gray matter of the C3 spinal cord



remains difficult. Nevertheless, this report does suggest that an intensive multidisciplinary treatment programme in the management of such divers holds some promise in the treatment of spinal cord DCS.

Acknowledgement

We thank our patient for providing written consent to report her case history.

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Submitted: 24 November 2011
Accepted: 10 April 2012

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A cost-analysis case study of radiation cystitis treatment including hyperbaric oxygen therapy

David Smart and Margaret Wallington

Abstract

(Smart D, Wallington M. A cost-analysis case study of radiation cystitis treatment including hyperbaric oxygen therapy. *Diving and Hyperbaric Medicine*. 2012;42(2):92-97.)

Aim: To undertake an economic analysis of the direct costs of treating radiation cystitis from a purchaser perspective, comparing conservative, non-operative and surgical interventions with hyperbaric oxygen treatment (HBOT).

Methods: A male in his 60s with prostatic carcinoma consented to this study. Full details of treatment costs in AUD were obtained (AUD 1.0, approx. EUR 0.6). A detailed patient diary accurately cross-referenced the consultations, investigations, admissions and treatment. Costs were recorded on a spreadsheet, dated and grouped under eight major headings related to treatment. Costs were compared for radiation cystitis treatment pre- and post-HBOT, to calculate savings or losses.

Results: The study covered three years (including 2.5 years post successful HBOT). Costs prior to HBOT (139 days) were AUD32,571.42 at an average of AUD231.09 per day, 70% from inpatient fees. Direct HBOT costs were AUD12,014.95 for 38 treatments, AUD316.18 per treatment. Post-HBOT (897 days), healthcare costs were AUD17,113.42 (AUD19.08 per day), with no emergency admissions. HBOT reduced costs of inpatient admissions, consultations, investigations and procedures and provided a projected healthcare saving of AUD187,483.96 over a 2.5 year follow up.

Conclusions: The cost of HBOT compared favourably against other costs, and HBOT may provide major health cost savings in this condition. There are significant hidden costs associated with radiation cystitis, not apparent to health funders, because the reasons for admissions and procedures are not easily captured with current information systems.

Key words

Hyperbaric oxygen therapy, soft-tissue radionecrosis, irradiation, injuries, economics, case reports

Introduction

With an ageing population and finite resources, the cost of health care has been very much in the spotlight and hyperbaric oxygen treatment (HBOT) has not escaped scrutiny.[1–3] Health technology assessments (HTAs) undertaken by the Federal Government's Medical Services Advisory Committee have reported on the costs of HBOT in a negative way, indicating that there was little evidence that HBOT was cost-effective for two conditions: soft tissue radiation injury and refractory non-diabetic problem wounds. The report (MSAC 1054) concluded: "*The clinical evidence was inadequate to substantiate claims that HBOT was cost-effective in the treatment of refractory soft tissue radiation injuries or non-diabetic refractory wounds*".[4]

The costs of HBOT in the Australian setting were documented from a provider perspective by Gomez-Castillo and Bennett.[5] Despite detailed analysis of HBOT costs, the costs of 'standard care' for many of the conditions referred for HBOT is unknown. Referrals may occur after months or years of standard care. Standard care may involve dressings, bandages, lotions or antibiotics for problem wounds, and may include inpatient and more complex treatments (including surgical procedures) for wounds and radiation injury. To undertake a cost study of a population receiving HBOT compared to standard care is challenging, particularly when standard care may be spread around multiple providers and multiple geographic locations, in the community and as both outpatients and inpatients. In addition, it is not possible to search the Medicare schedule to identify costs of treating a specific disease, because very few Medicare item numbers are linked by detailed description to a disease process or diagnosis.

Measurement of healthcare costs can be indirect or direct. Indirect measurement of costs may be derived from administrative databases or other published healthcare studies. More precise data may be obtained from direct measurement or observation.[6] When assessing healthcare cost, it is important to document from which viewpoint the cost is being assessed. This is known as the 'perspective'.[7] The perspective dictates the range of cost elements included in a cost analysis. Various economic perspectives have been documented: those of the provider (clinicians), the purchaser (Medicare and insurance companies), the patient and society. Provider costs involve detailed analysis of all costs associated with delivery of a service to calculate a 'per patient' or 'per treatment' cost. Purchaser costs are calculated by the amount the purchaser pays for treatment of a clinical condition during an episode or episodes of care. Patient costs may involve additional costs to those paid by the purchaser such as medications, incontinence pads, dressings, transport and loss of wages. In addition there is often a significant non-financial cost to the individual patient owing to lost quality of life.[8] Finally, costs to society may include the need to replace or retrain individuals if they suffer poor health and are no longer able to perform their work. This may also include payment of sickness benefits.

Aim

This paper reports on a detailed economic analysis of the direct costs from the purchaser's perspective over a three-year period of treatment of a single patient with radiation cystitis, comparing conservative, non-operative and surgical interventions with HBOT.

Case report

Mr X, a man in his 60s, was diagnosed with an early, localised carcinoma of the prostate in September 2003. He was initially treated with a radical prostatectomy in October 2003. There was evidence of slowly progressive biochemical recurrence during 2004, thought due to early local microscopic recurrence. In November 2004, radical external beam radiotherapy to the tumour bed was initiated (66 Gray in 37 fractions). Recovery from this treatment was uneventful, resulting in a complete biochemical response.

There were no other health issues until early February 2006, when Mr X required two inpatient admissions for clot retention and urethral obstruction totalling seven days. Cystoscopy demonstrated radiation cystitis, and bleeding areas were photocoagulated. Further clot obstruction occurring in late February necessitating a bladder washout and diathermy (single-day admission) and long-term urethral catheter insertion. He was referred for HBOT treatment in early March, but was unable to pressurise because of long-standing sinus blockages. This was assessed with a CT scan, then ENT referral for remedy, pending return for HBOT. While waiting for sinus surgery, he had two further admissions for haematuria with clot obstruction, two admissions for systemic sepsis (total 10 days as inpatient), and one extended emergency department visit for sepsis. Septoplasty, medial meatal antrostomies and intranasal ethmoidostomies were performed in mid-May (three inpatient days). After discharge and until mid-June, Mr X had five further visits to hospital with haematuria and urological complications, including two admissions (and one episode of sepsis for seven days). Hence, prior to HBOT, Mr X had nine inpatient admissions to treat complications from the radiation cystitis including one ICU admission. The non-HBOT admissions resulted in 26 inpatient days, and four prolonged emergency attendances out of a total of 139 days. During the active period of haematuria, Mr X's haemoglobin fell from 150 g L^{-1} to 130 g L^{-1}; transfusion was not required.

In mid-June 2006, HBOT was commenced when Mr X still had macroscopic haematuria. Thirty-eight treatments were administered over 60 days. By Day 21, macroscopic haematuria was no longer discernible. By Day 23, the urinary catheter was removed. HBOT was completed in mid-August 2006. Admission for a check cystoscopy two weeks later demonstrated all bleeding had stopped. Post-cystoscopy, there was no further bleeding. Routine medical follow up continued to occur, and a check cystoscopy was performed in October 2008. This showed no signs of cancer and minor remaining evidence of radiation cystitis. From October 2008 to 31 January 2009, Mr X received consultations, routine blood tests and had no admissions to hospital.

QUALITY OF LIFE ISSUES

During the 897 days of follow up on Mr X post-HBOT, he remained well with no episodes of recurrent haematuria or cystitis. His personal medical diary demonstrated that the intervention of HBOT resulted in many positive health outcomes and improved quality of life including:

- prolonged (2.5 years) freedom from haematuria;
- no admissions to hospital for complications of radiation cystitis during 2.5 years' follow up;
- no further requirement for a urinary catheter, which was possibly a permanent requirement prior to commencing HBOT, having remained in situ for 134 out of a possible 155 days from commencement of haematuria;
- no further distress with urinary retention or catheter blockage;
- no further emergency presentations or surgery with its associated risks.

Methods

The study period covered from 01 February 2006 to 31 January 2009. It commenced one week prior to onset of macroscopic haematuria, covered the duration of the illness, and extended for almost 2.5 years after completion of HBOT. Mr X provided full consent for his case history to be published including analysis of the costs of his health care. He assisted with data collection by obtaining printouts of treatment costs from Medicare and his private health fund for the study period. These records also documented gap payments made by or on behalf of the patient. As part of a life-time habit, Mr X kept a detailed diary of his symptoms and the reasons for all medical care. This diary allowed cross-referencing of his clinical status with reimbursements paid by Medicare and health funds (including gap fees), and ensured accurate assignment of treatment costs to appropriate category headings. All non-HBO treatments were delivered in the private sector in the State of Tasmania, Australia. Mr X was treated as a private patient at the Royal Hobart Hospital hyperbaric facility. The study did not include patient transport costs, medication costs or consumables purchased by the patient in the community. Actual costs directly paid by Medicare and his private health fund were included. Costs were entered into a Microsoft Excel® spreadsheet, by date and grouped under eight major headings:

- hyperbaric oxygen consultations and treatment costs;
- hyperbaric gap payments;
- hyperbaric other costs (e.g., procedures/treatments to support HBOT);
- consultations not related to HBOT;
- procedures not related to HBOT;

Table 1
Costs for cancer-related and non-cancer-related care excluding HBOT-related costs (all figures in AUD)

Time period	Consults	Procedures	Private GAP	Private pathology + X ray	Private hospital (incl. OR fees)	TOTAL
Start of study to completion of HBOT (199 days)	2,406.40	2,347.76	3,845.65	1,408.55	22,563.06	32,571.42
Post–HBOT to end of study (897 days)						
Cancer-related	2,543.75	827.65	4,337.25	2,460.89	4,662.48	14,832.22
Non-cancer-related	734.60	226.45	440.30	121.05	759.00	2,281.40

- private gap fees not related to HBOT;
- private X-ray and pathology costs not related to HBOT;
- private hospital inpatient costs.

METHOD OF CALCULATION OF COSTS

Costs were calculated in Australian Dollars (AUD); for the period of the study, AUD1.0 was approximately €0.6. Costs were split into three time periods: before HBOT, during HBOT and after HBOT. Costs were further subdivided into non-HBOT (cancer and non-cancer related), and HBOT-associated costs. This enabled any additional therapy for the radiation cystitis administered during HBOT to be detected, and costs unrelated to cancer to be separated. All costs of other treatments (for example ENT surgery) to support HBOT were included in the HBOT costs.

A further calculation was made of the cost of all treatment delivered for the period after HBOT. The point at which HBOT was completed (18 August 2006) was defined as the index date, for the purposes of calculating pre- and post-HBOT costs. For the two time periods, pre- and post-HBOT, the medical costs per day were calculated by dividing the total cost by the number of days. The predicted post-HBOT cost was calculated by multiplying the daily cost pre-HBOT by the number of days post-HBOT. Daily costs were then compared pre-and post-HBOT.

NON-HBOT MEDICARE PROCEDURE CODES

During the course of the study, procedural services were provided under 17 Medicare Procedural Item Numbers.* None of these item numbers were directly traceable to an episode of radiation cystitis, unless the admission episode and the diagnosis-related group (DRG) were also searched simultaneously. In addition, procedures required to allow the patient to receive HBOT (to correct previously undiagnosed paranasal sinus disease) were provided under six more Medicare codes.

Results

During the period before HBOT (139 days), Mr X had nine admissions totalling 26 inpatient days (one day spent in hospital every 5.4 days) as a result of his radiation cystitis. HBOT was administered as 38 treatments over 60 days. The time period from study commencement to completion of HBOT was 199 days. The study time period post-HBOT was 897 days.

COSTS PRE-HBOT

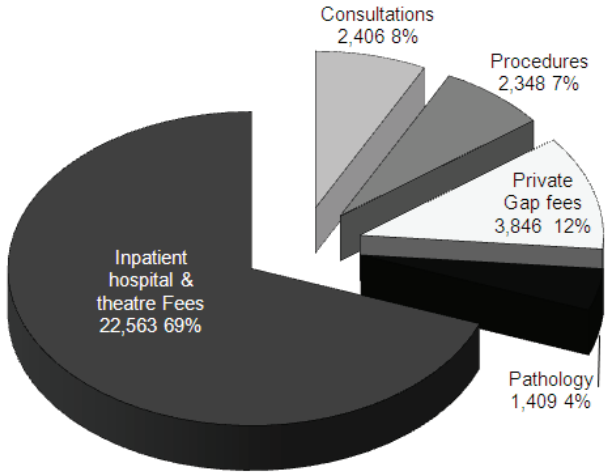
Prior to commencing HBOT, Mr X incurred AUD32,120.82 non-HBOT treatment costs (AUD231.09 per day over 139 days). While receiving HBOT, there was an additional AUD450.60 in non-HBOT related consultations (AUD157.85), pathology fees (AUD100.45) and gap fees (AUD192.30), which totalled only 1.4% of the non-HBOT costs. Total non-HBOT costs at the completion of HBOT were AUD32,571.42 (AUD163.68 per day over 199 days). Table 1 shows the non-HBOT patient treatment costs incurred before and until completion of HBOT. During this period, all costs were for treatment of radiation cystitis or cancer follow up. Figure 1 summarises the percentage breakdown by cost category. Prior to commencing HBOT, hospital admission fees made up 69% of all medical costs.

COSTS OF HBOT

HBOT costs were AUD12,014.95 in 38 treatments, spread over 60 days or AUD316.18 per treatment (Medicare AUD216.15 with private gap AUD100.03). The cost of sinus surgery (AUD7,788.82) was added to the HBOT costs, (a pre-existing condition but required in order to undertake HBOT). This increased the total cost to enable the patient to receive HBOT to AUD19,803.77. When receiving HBOT, there were no further admissions to hospital for radiation cystitis complications. Hence for the 199 days from start of study to completion of HBOT, the treatment costs for Mr X were:

*** Footnote:** Medicare Procedural Item Numbers used: 20120H, 20160H, 20810H, 20910H, 31210, 32090H, 34528H, 36800, 36812H, 36840H, 37318H, 55113H, 56507, 56507H, 58503, 58503H, 58706H.

Figure 1
Actual and percentage non-HBOT costs (AUD) over 199 days in a patient with post-radiation haemorrhagic cystitis



- All alternative treatments (unsuccessful), including 9 hospital admissions, one ICU admission, long-term urinary catheterisation, multiple procedures (including surgical and diathermy), and investigations: Pre-HBOT cost = AUD32,571.42.
- HBOT (successful), including admission for sinus surgery prior to HBOT, and associated investigations and consultations: HBOT cost = AUD19,803.77.

COSTS POST-HBOT

Table 1 summarises the costs of non-HBOT care, split by cancer-related costs and other medical costs. For the purposes of the study, all treatment costs (including those not related to radiation cystitis) were included, to ensure there was no bias in data collection. The other medical costs included minor surgery for a sebaceous cyst, and routine general practice consultations. The only HBOT-related cost after completion of the course was a single review consultation a year later.

The total cost post-HBOT of AUD17,113.62 was 53% of the pre-HBOT cost for a period 6.45 times as long (897 days vs. 139 days), at an average of AUD19.08 per day, or 8.3% of the pre-HBOT cost per day. Of the post-treatment costs, AUD14,832.22 (86.7%) was cancer related. Table 2 shows the numbers of consultations, procedures, and investigations before and after HBOT, with a significant reduction per 100 days post-HBOT ($P < 0.0001$ for all).

Discussion

These data have been derived directly from a detailed study of actual costs incurred by a single patient with radiation cystitis in an Australian setting. The authors are confident that the costs have been accurately assigned to the correct disease category, because the patient documented his life events over the course of the illness. Printouts from Medicare and his private health fund were also classified by date and provider, and contained a description of the service that had been reimbursed. Direct measurement of these raw data provided more accurate assessment of costs from a purchaser perspective than if modelling had occurred based on DRG, or per occasion of service. Previous HBOT cost-effectiveness studies have been based on modelling and hypothetical patients, or administrative databases, rather than prime source data, and they have not been linked to comparator treatments of the relevant clinical conditions.[2,9,10]

This case study demonstrates that there may be significant hidden costs in the treatment of radiation cystitis, none of which are easily identified. The reasons for admissions and procedures are not easily captured and linked to the Commonwealth Medicare Benefits Schedule item numbers with current information systems. An admission diagnosis of haematuria or urinary retention may have multiple aetiologies. Hence it may be difficult to track a patient over the time course of their specific clinical condition. The analysis of this case was assisted considerably by the patient's detailed personal diary.

The cost of HBOT compared favourably against costs of preceding unsuccessful treatments, which required multiple admissions. Standard treatments, including surgical diathermy, had not been successful in arresting this patient's haematuria prior to commencement of HBOT. There was a clear cause-and-effect relationship between onset of HBOT and the cessation of bleeding (and catheter removal). This is consistent with available knowledge of the beneficial

Table 2
Comparison of pre- and post-HBOT numbers of consultations, procedures and investigations per 100 days in a patient with post-radiation haemorrhagic cystitis; * $P < 0.0001$ for pre-/post-HBOT items

Time period	Consultations	Procedures	Investigations X-rays + pathology
Before HBOT (all cancer related)	59	14	61
Number per 100 days*	29	7	31
After HBOT	64	14	67
Cancer related	49	13	59
Number per 100 days*	7	2	7

effect of HBOT in late soft-tissue radiation injury, which suggests upward of three quarters of such patients are likely to benefit from HBOT.[11–14] Most other treatments (surgical or non-surgical) are directed at symptom control and do not influence the underlying pathophysiology of radiation cystitis. In contrast, HBOT has been shown to reverse the pathophysiology of radiation tissue injury.[11] A Cochrane review has investigated non-surgical interventions such as alum, formalin and placental extract bladder instillations, and systemic therapies such as pentosan polysulphate, tetrachlorodecaoxide, and oestrogens and pentoxyfilline.[15] The review was inconclusive regarding the efficacy of any of these treatments.

This case also demonstrates the positive impact on patient wellbeing and quality of life after successful HBOT, to a follow-up of 2.5 years. However, there are some limitations to the report. The economic perspective is that of a purchaser of healthcare services (Medicare and private insurers + gap fees), and does not include patient-related costs such as transport, medications and other consumables. Despite the detailed analysis, this is a single case, and the results cannot be generalised. However, the methodology should provide the basis to undertake more detailed study of a larger series in a prospective manner. Mr X suffered a high number of complications from his disease, and may have experienced a severe form of radiation cystitis. However, this story is fairly typical of such patients referred for HBOT, and who have often failed a variety of other treatments before their referral.[13] Radiation cystitis may affect 5 to 10 per cent of individuals receiving pelvic radiotherapy for cancer and, once established, tends to be progressive.[15]

The patient's HBOT course was also atypical. Sinus barotrauma occurred in only 11 cases in 24,731 Australian hyperbaric patient treatments in 2008 (unpublished data). The need to undertake surgery for his pre-existing problem is even less common. Even inclusive of sinus surgery, the cost of HBOT was 60.8% of all other preceding treatment for radiation cystitis, and it succeeded in stopping the bleeding when other treatments had failed.

Before HBOT was instituted, Mr X was incurring an average daily treatment cost of AUD231.09. There was no sign that his disease process was being controlled. If this had continued, the total cost for the 897 days of follow up this patient received after cessation of HBOT would have been AUD207,287.73. Therefore, the expenditure of AUD19,803.77 for HBOT provided a potential (theoretical) saving of AUD187,483.96, and resulted in successful remission of manifest disease symptoms for a 2.5 year period, with a leveraged cost-advantage factor of 9.5. The success of HBOT was supported by a significant reduction in the number of consultations, procedures and investigations required post treatment. Without the successful intervention provided by HBOT, it is likely that Mr X may have suffered major complications from radiation cystitis, or required radical surgical intervention such as total cystectomy, with a severe negative impact on his quality of life. Although cost-effectiveness cannot be calculated from a single case, there was a cost saving of AUD12,767.65 when comparing cost of HBOT with preceding treatments, which had been ineffective. The average cost of HBOT of AUD316.18 per treatment, compares closely to that of AUD311.00 calculated from a provider as opposed to a funder perspective using a comprehensive modelling technique.[5]

The major costs of standard treatment for Mr X prior to HBOT resulted from hospital bed fees and theatre charges (69%, Figure 1). Post-HBOT no further hospital admissions were required for complications of radiation cystitis, and costs of inpatient cancer-related follow up fell to 31% of all costs (Table 1). Despite the considerable reduction in healthcare costs post-HBOT following successful remission of radiation cystitis, 87.1% of all Mr X's healthcare costs, and the majority of consultations, procedures and investigations were still attributable to his original cancer. This demonstrates that even in remission, cancer has a major impact on healthcare costs. It is also noteworthy that, with a greater amount of care delivered in the outpatient setting, Mr X had a higher percentage of out-of-pocket gap fees (29%) in the follow-up period.

In 2009–10, there were 1,355 separations in Australia for diagnosis N30.4-irradiation cystitis, occupying just under two bed days per separation.[16] This may be an underestimate because presentations due to urosepsis, haematuria and bladder obstruction due to clots may not be linked to radiation cystitis. HBOT has potential to reduce requirements for hospital admission, leading to major cost savings for health services and improvement in quality of life for patients.

Conclusions

This study demonstrates the complexity of calculating healthcare costs for late soft-tissue radiation injury. The cost of standard treatment for soft-tissue radiation injury has not been previously studied, and may be much higher than generally appreciated. In this patient, HBOT was clinically effective in resolving complications from radiation cystitis, and had lower costs than other unsuccessful treatments. Further investigation in a prospective study of multiple patients is warranted.

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Submitted: 01 September 2011
Accepted: 10 April 2012

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A case of spinal epidural haematoma during breath-hold diving

Lucio Tremolizzo, Mirko Patassini, Massimo Malpieri, Carlo Ferrarese and Ildebrando Appollonio

Abstract

(Tremolizzo L, Patassini M, Malpieri M, Ferrarese C, Appollonio I. A case of spinal epidural haematoma during breath-hold spearfishing. *Diving and Hyperbaric Medicine*. 2012;42(2):98-100.)
Spinal epidural haematoma (SEH) is a rare condition usually the result of bleeding of the epidural venous plexus that might present with acute spinal cord compression. It is often due to traumatic events, but 'spontaneous' cases have been described, usually related to different predisposing conditions, such as coagulopathies. A 47-year-old male presented with severe frontal headache and intense cervical pain which developed during a protracted breath-hold spearfishing session. A cervical spine MRI performed 12 days after symptom onset showed a small epidural blood collection on the left side of the spinal canal, at the C7–T1 level. One week later, blood was no longer present and the asymptomatic patient was discharged. Protracted minor trauma (neck flexion) and repeated Valsalva manoeuvres might have played a role in the genesis of this event. The role of decompression sickness is discussed as well.

Key words
Breath-hold diving, spearfishing, central nervous system, Valsalva manoeuvre, injuries, decompression sickness, case reports

Introduction

Spinal epidural haematoma (SEH) is a rare condition usually owing to bleeding from the epidural venous plexus and presenting with local and radicular pain associated with acute spinal cord compression that may require urgent surgical decompression. It is often the result of traumatic events, including surgery, intervertebral disc herniation and lumbar puncture.[1–3] However, 'spontaneous' cases of SEH (SSEH) have been described, usually related to coagulopathies or anti-coagulation therapy, vascular malformations, drug abuse, plasma cell myeloma, and non-Hodgkin's lymphoma, among other causes.[3,4] Here, we report a case of a spontaneously recovered SEH that presumably developed during an intense and protracted period of breath-hold spearfishing.

Case report

A 47-year-old male presented with severe frontal headache and intense cervical pain. The onset came one week earlier, during an intense (operating depth between 15 and 25 metres' sea water, msw) and protracted (repetitive dives over more than 4 h) breath-hold spearfishing session. Past medical history only documented migraine without aura and the patient denied taking any medications during the days before. Neurological examination was unremarkable, although severe tenderness in the cervical region was noted. Headache and cervical tenderness progressively subsided following several days of oral diazepam and paracetamol and intravenous fluid administration. Considering the intensity of the pain, a brain and cervical spine MRI scan was performed 12 days after symptom onset, showing a small epidural blood collection on the left side of the spinal canal, at the C7–T1 level (Figure 1). A bulging disk at the C5–C6 level was also noted. One week later, blood was no longer present on repeat scanning (Figure 2). The patient was asymptomatic and was discharged with advice to have his blood pressure monitored, since borderline hypertensive values were noted during hospitalisation.

Discussion

Scuba diving-related haemorrhages have been reported previously, mainly involving tissues classically prone to barotrauma, such as the lungs, the orbital region (mask squeeze) and the inner ear.[5–7] Middle-ear barotrauma, caused by failure to equalise the pressure between the middle ear and ambient pressure during descent (or ascent), is common in diving, and such events can result in pneumocephalus associated with parenchymal and extra-axial haemorrhage.[8] Fatal epidural haematoma overlaying the tegmen tympani has also been reported following air insufflation via a Siegler speculum.[9] A similar case of pneumocephalus with disruption of the tegmen tympani due to barotrauma during scuba diving was subsequently shown on MRI performed 16 days after the injury.[10] Epidural blood near the base of the skull, and in both mastoids was seen. Rarely, haemorrhagic events following scuba diving have been reported in predisposed subjects in tissues that are not classically targets of barotrauma, e.g., oesophageal variceal bleeding in a patient with a history of cryptogenic liver cirrhosis.[11] Moreover, a case of spontaneous, multiple, albeit subdural, spinal haemorrhages (from C7 to T11) has been reported, occurring in the absence of apparent pre-existing abnormalities.[12]

Decompression sickness-related myelopathy might present with perivascular haemorrhages, although in this case it occurred within the spinal cord parenchyma, possibly related

Figure 1
Spinal MRI at 12 days following symptom onset (A) sagittal T1WI showing a poorly defined faint epidural hyperintensity at C7–T1 (white arrow), and (B) axial T2WI showing a small inhomogeneous epidural hyperintense collection (white arrow) inducing mild left postero-lateral cord surface compression; no intramedullary hyperintensity was present

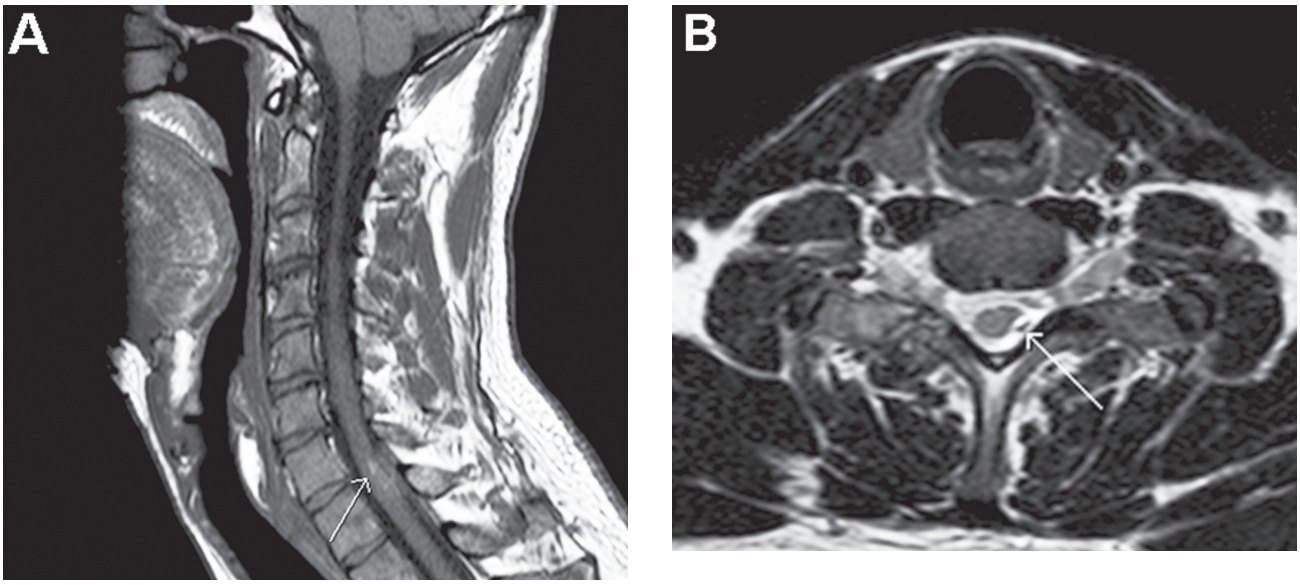
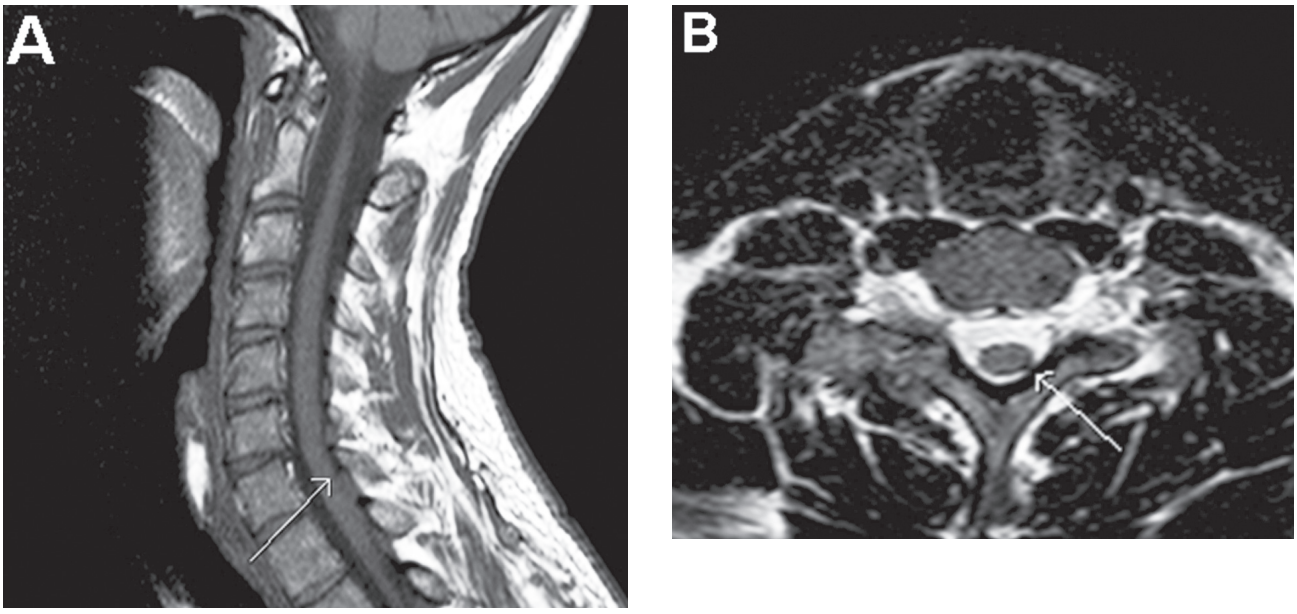


Figure 2
One week later, both sagittal T1WI (A), and axial T2WI (B) evidenced a complete recovery



to venous infarction.[13] Interestingly, simulated chamber dives in dogs demonstrated that the epidural vertebral venous system became obstructed during spinal cord damage due to decompression sickness.[14] Even accepting the existence of decompression sickness from breath-hold diving, involving protracted apnoeas with short surface intervals,[15,16] we did not find evidence of spinal cord parenchymal damage, and the haemorrhagic event was confined to the epidural space. Even when spearfishing in shallow waters, many free divers need to repeat the Valsalva manoeuvre frequently to equalise pressure in the middle ear with each descent. Such transient venous hypertension as a result of sudden Valsalva manoeuvres, including coughing and sneezing, is thought to play a role in SSEH.[17] This might be considered as a contributing factor in this diver.

Finally, the subject reported that his operating depths, duration and intensity of the exercise were possibly somewhat excessive for his physical status, and that he was over-weighted for the conditions, necessitating extra effort to maintain buoyancy during surface recovery periods. The effort of continuous flexion-extension of the neck for respiration might have played a role in the genesis of the haemorrhagic event. In fact, minor traumas, such as falling to the ground, protracted crawling, change of posture during sleep, coughing or the Valsalva manoeuvre are all proposed or recorded as possible predisposing factors in published case series of SSEH patients.[18] Analogously, disk herniation is considered in the list of risk factors since dorsal displacement of the annulus or nucleus during acute disk disruption might produce a tear within the venous plexus.[17] However, our patient did not present evidence of complete disk herniation, as he did not have any other known predisposing factors. Interestingly, the involved site, thoracic spine, is the most common for those SEHs that are often referred to as 'spontaneous'. However, only about 40% of the cases remain truly idiopathic, whilst in the rest a precipitating factor could be found.[3,4]

Therefore, considering the aforementioned evidence, we conclude that the most likely predisposing conditions for this event were the overlap of repetitive minor traumas, i.e., the incessant movements of neck flexion, and the frequent Valsalva manoeuvres during breath-hold diving.

Acknowledgements

We wish to thank Drs E Susani, D Cereda and G Costantino for the clinical follow-up of the patient.

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Submitted: 01 December 2011
Accepted: 11 April 2012

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Cerebral venous air embolism treated with hyperbaric oxygen: a case report

Pieter A Bothma, Andreas E Brodbeck and Bruce A Smith

Abstract

(Bothma PA, Brodbeck AE, Smith BA. Cerebral venous air embolism treated with hyperbaric oxygen: a case report. *Diving and Hyperbaric Medicine*. 2012;42(2):101-103.)

We present a case of cerebral venous gas embolism. Our patient made a complete neurological recovery after hyperbaric oxygen therapy (HBOT). The principles of HBOT, compressing and eliminating air bubbles and decreasing ß-2 integrin function, thus improving microcirculation, can only be beneficial in a situation where neurological damage is likely. Retrograde cerebral venous gas embolism is a less well recognised variant of gas embolism than the arterial variant. Its existence as a different entity is better recognised in the forensic medicine and radiology literature than in other disciplines. There is evidence in the literature of patients dying from this complication and others seemingly experiencing very little effect. This case report highlights this condition, to encourage others to look out for it and report outcomes, and to serve as a reminder that peripheral lines may be a potential cause of gas embolism, although the portal of air entry in our case remains uncertain.

Key words

Venous gas embolism, hyperbaric oxygen therapy, medical conditions and problems, radiological imaging, right-to-left shunt, case reports

Introduction

Gas embolism is a serious complication of diving and is nowadays more commonly encountered as an iatrogenic complication of invasive medical procedures.[1–3] In most instances air is the gas involved.[1] Gas can be introduced into either the venous or arterial circulation. Hyperbaric oxygen therapy (HBOT) is recognised as the most effective treatment for cerebral arterial gas embolism.[3] The role of HBOT in cerebral venous gas embolism (VGE) is still to be established. VGE is well known to occur as a result of central venous line insertion, or its accidental disconnection or removal when incorrect techniques are used.[1] A less well recognised source of VGE is peripheral venous access.[4,5]

Paradoxical gas embolism occurs when gas passes from the right-sided circulation to the left side via an intra-cardiac shunt, e.g., most commonly a patent foramen ovale (PFO) or, in the absence of that, a presumed physiological or pathological arterio-venous pulmonary shunt.[2,6] Such paradoxical emboli would be distributed throughout the systemic circulation but would preferentially circulate to areas of high blood supply, e.g., the cerebral and coronary vessels with life-threatening ischaemia if cardiovascular collapse is not fatal in itself.[1]

We present a case of cerebral VGE of uncertain aetiology, discussing possible ports of entry of gas into the circulation as well as the diagnosis and management of this case. We will emphasise the awareness of retrograde cerebral venous air embolism as a yet poorly recognised variant of a well-known phenomenon.

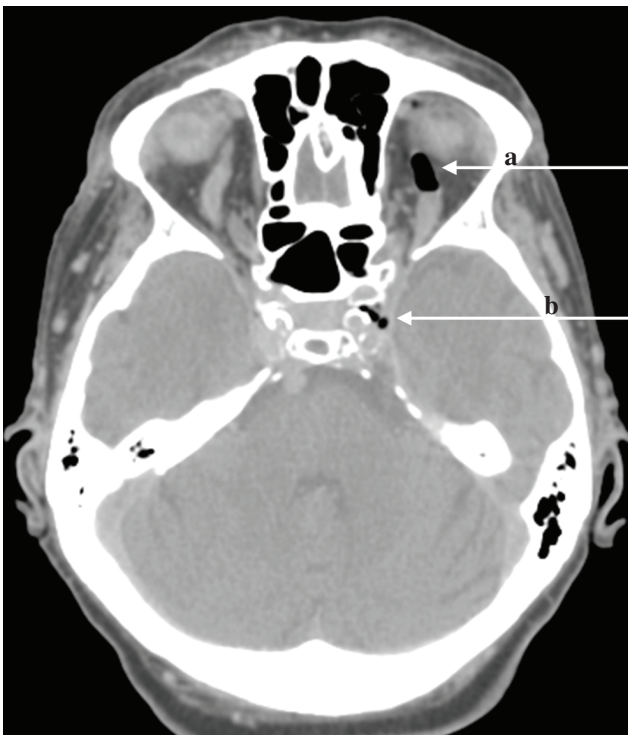
Case report

A 74-year-old female patient was admitted to the Emergency Department with suspected septic shock as a result of pneumonia. She had been found in a semi-comatose state on the floor of her flat. She had apparent renal failure as a result of rhabdomyolysis from lying on the floor for a long time. She had no lateralising neurological signs at that stage.

She was fluid resuscitated via two large-bore peripheral cannulae. When she was referred to ICU, she remained hypotensive and dyspnoeic with obviously distended neck veins. Initially her breathing was supported with non-invasive ventilation by face mask. An inspiratory pressure of 10 mmHg and positive end expiratory pressure of 5 mmHg was used. An arterial cannula was inserted in the right radial artery and a left internal jugular central line inserted with the patient supine. Both procedures were straightforward. The central line was inserted with ultrasound guidance, showing a dilated central venous system confirming the clinical impression of high venous pressure. The first measurement of central venous pressure was 18 mmHg. An infusion of noradrenaline was started immediately through the central line and tracheal intubation was performed to be able to control ventilation. A nasogastric tube was inserted, as well as a haemocath in the left femoral vein under ultrasound guidance for haemofiltration and a PiCCO® line for cardiac output studies in the right femoral artery.

The patient's condition stabilised and she started passing urine once her blood pressure improved, obviating the need for immediate renal replacement therapy. She became

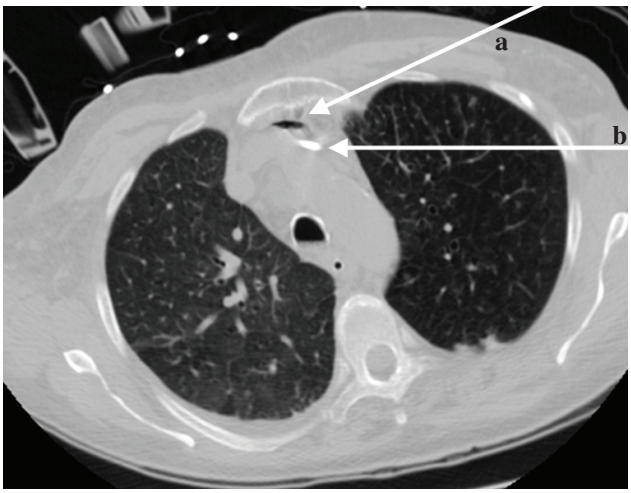
Figure 1
Air in the ophthalmic vein (a) and central venous sinus (b)



more stable haemodynamically and could be ventilated in the 45^{O} head-up position to decrease the risk of aspiration pneumonia.[7,8] The next morning, her sedation was stopped for neurological assessment. It was then clear that she had a right hemiparesis. She also had bilateral extensor plantar reflexes. A computerised tomographic (CT) brain scan ruled out an intracerebral bleed, but gas emboli could be detected in the intracranial blood vessels (Figure 1) and after contrast was given, gas emboli were clearly seen in the cerebral venous system as well as the left subclavian, internal jugular and brachiocephalic veins (Figure 2).

A CT scan of the chest excluded occult pneumothorax. The patient was then taken to the hyperbaric unit where she was treated with a US Navy Table 6 treatment protocol with full ICU monitoring and still on a noradrenalin infusion. This was followed by three further hyperbaric sessions using a US Navy Table 5 protocol, at which time the patient's neurological signs had apparently resolved. Her further progress was slow as a result of chronic lung disease (bronchiectasis and chronic obstructive pulmonary disease). She required a tracheostomy and prolonged weaning with separation from the ventilator after 41 days and was discharged from ICU after 49 days to the rehabilitation ward, being neurologically completely intact but very weak. She was eventually discharged from hospital to suitable nursing-home accommodation.

Figure 2
Air in the brachiocephalic vein (a) just anterior to a central venous line (white, b)



Discussion

The portal of gas entry into the venous system of our patient is uncertain. The central line insertion technique was impeccable and the patient had high venous pressure at that stage. This could be related to vigorous fluid resuscitation or it reflected right heart strain from air embolism. Partial disconnection of venous lines at a later stage could not be ruled out. Malfunction of the needle-free connectors attached to the central line was an unlikely possibility, but was excluded in view of previous warnings by the Medicines and Healthcare Products Regulatory Agency.[9] Peripheral lines could be a portal of entry of air into the venous system, even when injecting contrast during CT imaging, and is often overlooked.[4,5] The CT scans of the brain and chest ruled out the possibility of head trauma or a pneumothorax as the cause of air embolism, leaving the various vascular access ports and their connections as the alternative and most likely cause. Ongoing air entrainment from an underlying pulmonary condition is extremely unlikely as the patient remained on positive-pressure ventilation for a long time without recurrence of any neurological abnormality.

Cerebral gas embolism with neurological damage is usually assumed to be caused by obstruction of the arterial blood supply to parts of the brain. It is now evident that cerebral VGE could also cause significant morbidity and even mortality.[10–12] Cerebral air embolism has traditionally been assumed to result from direct arterial access or paradoxical embolism from the venous side through a PFO or pulmonary capillary filtration overload or arterio-venous malformation. Retrograde access of gas to the cerebral venous system has been ignored, possibly being regarded as innocuous.[1–3] Since the first description of cerebral VGE in 1991, this has been

recognised more frequently, and published, but not taught or described routinely .[1–4,10,12–15]

Differentiating retrograde cerebral VGE from paradoxical cerebral arterial gas embolism is not only of academic importance and the former is certainly not innocuous.[10–12] With arterial gas embolism, small quantities of gas could theoretically immediately cause ischaemia in an area of brain distal to obstruction of small arteries. This necessitates urgent treatment with HBOT as soon as the patient's condition has been stabilised, despite the fact that some reported cases had good outcomes even after 30 hours' delay in treatment.[1,2] With retrograde cerebral VGE there is no immediate arterial obstruction causing ischaemia. This may be a slow collection of gas depending on differential pressures in the cerebral venous and thoracic systems, blood flow rate and position of the patient's head above the heart.[13–15] The inflammatory response between gas bubbles and the endothelium may lead to activation of neutrophils with ß-2 integrin adhesion to endothelial cells, resulting in stasis and venous infarction. This biochemical process, however, may allow more time to arrange HBOT. Lack of early symptoms should not be regarded as a sign of lack of severity, as several cases with poor outcomes have been reported.[10–12,15] Lastly, the importance of accurate diagnosis for medicolegal purposes cannot be overemphasised.[14]

Conclusions

Venous gas embolism is a well-known phenomenon. Gas emboli accumulating in the cerebral venous system and its consequences are fairly unknown and poorly understood variants. It is clear from the literature that patients have died from this. When a patient presents with cerebral VGE and neurological signs, it would be foolhardy not to offer HBOT. When the diagnosis is made in an asymptomatic patient, the management is uncertain. Careful neurological follow up may be adequate, but if available, HBOT may avoid late complications. Documenting and reporting such cases may help with future decision making.

Acknowledgements

Written consent was obtained from the patient to publish details of her case. No external funding was received for this work and no conflict of interest is declared.

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Submitted: 08 December 2011
Accepted: 09 February 2012

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Critical appraisal

The addition of hyperbaric oxygenation to specialised wound care for chronic diabetic foot ulcers improved healing and quality of life

Clinical bottom line:

- The addition of hyperbaric oxygen therapy (HBOT) improved the proportion of diabetic wounds that healed at one year.
- The adverse event rate was low.

Citations:

1 Londahl M, Katzman P, Nilsson A, Hammarlund C. Hyperbaric oxygen therapy facilitates healing of chronic foot ulcers in patients with diabetes. *Diabetes Care*. 2010;33:998-1003.

2 Londahl M, Katzman M, Nilsson A, Hammarlund C, Sellman A, Wykman A, et al. A prospective study: hyperbaric oxygen therapy in diabetics with chronic foot ulcers. *J Wound Care*. 2006;10:457-9.

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Three-part clinical question:
For diabetic patients with chronic lower limb ulcers, does the addition of hyperbaric oxygenation to a specialised wound care protocol, result in an improved rate of healing?

Search terms:
Diabetes, chronic wound, skin ulcer

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

The study patients:
Adult diabetic patients with ankle or foot ulcers for more than three months (including two months of specialised wound care) and where there is no major vessel disease requiring surgical intervention.
Control group (n = 45; 42 analysed)
Specialised, comprehensive wound care plus 40 hyperbaric treatments breathing air at 2.5 ATA for 90 minutes daily Monday to Friday over 8–10 weeks.
Experimental group (n = 49; 48 analysed):
As above, but breathing 100% oxygen at 254 kPa for 90 minutes daily on same schedule as the control group.

The evidence: See Table 1.

Comments:

- Well-conducted study with high methodological rigour and a low risk of bias.
- Quality of life assessed at one year using the SF-36 questionnaire over eight dimensions. Those receiving HBOT improved statistically significantly in two dimensions (physical functioning and emotional state), while those receiving sham had not.
- Baseline transcutaneous oxygen levels correlated positively with the chance of healing in those who received HBOT. Neither toe blood pressures nor ankle-brachial index at baseline predicted healing.
- A per protocol analysis of those who received at least 35 treatment or sham sessions confirmed a benefit from HBOT (61% healing versus 27% healing at one year, P = 0.009).

Appraised by: *Michael Bennett, 19 April 2012*
E-mail: *< m.bennett@unsw.edu.au>*

Key words
Hyperbaric oxygen therapy, wounds, diabetes, critical appraisal

Table 1
Major clinical outcomes

Outcome	Time to outcome	Sham rate	HBOT rate	Relative risk reduction (%)	Absolute risk reduction	Number needed to treat (harm)
Wound healed	1 year	0.27	0.51	91	-0.243	4
95% CI				20 to 162%	0.05 to 0.43	2 to 19
Death	1 year	0.07	0.02	70	0.05	21
95% CI				54 to 100%	0.04 to 0.13	8 to INF
						NNH = 28 to INF
Major amputation	1 year	0.02	0.06	184	-0.041	25
95% CI				100 to 549%	0.04 to 1.21	25 to INF
						NNH = 8 to INF

Letters to the Editor

HBO Evidence website

Dear Editor,

As many of your readers may be aware, I have been having trouble maintaining the HBO Evidence site <www.hboevidence.com> over the last 18 months. This is partly because of the inevitable evolution of page-writing software and partly to the equally inevitable problems generated by hospital firewalls that seem designed to prevent employees from posting useful information on the internet. Our pages were also rather too often under cyber attack, making the discussion forums unworkable.

While we have moved the site behind the University of New South Wales (UNSW) defences in order to prevent its corruption, it is unfortunately no longer possible to update the contents – rendering it rather purposeless.

To this end, I have been working over the last 15 months to re-write the site as a wiki under the auspices of the UNSW 'wikispaces' group of sites. This has several advantages, including affording a high level of protection, an enhanced ability to quickly and easily update the contents, the ability to allow others to easily update contents if required and a more secure discussion facility.

I am pleased to advise, therefore, that the new site is now open for business. The new address is:
<http://hboevidence.unsw.wikispaces.net/>.

Interested readers should reset their favourites list to this address instead of the old <www.hboevidence.com>. The site remains dedicated to presenting useful summaries of all the randomised trials in both diving and hyperbaric medicine.

When you first visit the site, you will be asked if you wish to become a member. Membership is not required to view the pages, but anyone interested in assisting us with adding to the content of the site and keeping it up to date, can apply to join us by submitting the membership request.

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Key words
Hyperbaric medicine, world wide web, research, letters (to the Editor)

Safety of deep apneic diving

Dear Editor,

We agree with Dr Walker's concerns about risks associated with breath-hold diving, voiced after Professor Schagatay's first review in 2009 in this journal.[1,2] We thank Professor Schagatay for her very thorough reviews, but only agree in part with her view that reporting increases safety, as breath-hold deep diving per se is unsafe.[2,3,4] To weave a scientific lifebelt for this high-risk activity is inappropriate. We also doubt that uncritical reporting increases safety. We also believe that it is scientifically unsound to recommend so-called 'proper techniques for preparation and performance' to achieve 'maximal performance'. We list below some of the serious pitfalls that could evolve from reading parts of the most recent review.[4]

Competitors in static/dynamic apnea experience extended hypoxia. While acutely elevated levels of a marker of brain damage may not be of major relevance, long-term, possibly cumulative effects must be suspected.[5]

If extended breath-holding alone poses serious risks for unconsciousness, brain injury and death,[6] then breath-hold deep diving adds risks associated with the effect of increased ambient pressure on gas volumes and increased partial pressures.

If a coach advises the use of new hydrodynamic goggles, almost frictionless dolphin-skinned swim suits and more efficient power fins, then he does not harm the athlete. If the ambitious breath-hold deep diving athlete reads about 'tricks' on how to fool physics, then he is seriously endangered. After reading *Training, preparation and equalization to avoid barotrauma* (p. 220ff.) he feels encouraged to perfect his glossopharyngeal insufflation (GI) and exsufflation (GE) to prevent descent barotrauma,[4] but he would thus go from bad to worse, as such techniques can do harm. Describing techniques without describing possible deleterious consequences seems too short-sighted.

GI might considerably increase intrathoracic pressures (up to 80 cmH_2O) with an increased risk of pulmonary barotraumas and arterial gas embolism.[7] In turn, increased intrathoracic pressures will likely impede venous return, inducing hypotension with consequences varying from dizziness to fainting just prior to diving.

Submersion shifts blood towards the chest, and more blood is shifted as ambient pressure increases. Thus, all thoracic structures with a high compliance are considerably enlarged. In consequence, chest sonography frequently documents pulmonary oedema after immersion,[8] and great depths are associated with the risk of pulmonary barotrauma (lung squeeze). GE can seemingly increase the risk of lung squeeze by taking some mouth-fills of air from the lungs

and should not be presented by a coach without its serious hazards being explained. While haemoptysis is the visible consequence of acute pulmonary barotrauma, any less severe damage might remain subclinical. Hence, regular competitive apnea diving over a few seasons might carry a chronic cardiopulmonary risk leading from early functional changes to the manifestation of pulmonary hypertension.[9]

Regarding lung squeeze, it should be noted that involuntary contractions of the thorax and diaphragm can produce waves of negative pressure.[10] Once intrathoracic pressure is already negative at great depth, additional negative pressure waves might well damage the pulmonary capillaries.

Finally, the risk of decompression sickness (DCS) after breath-hold dives has been considered by Dr Schagatay. After a breath-hold diver has suffered from cerebral DCS, such athletes should only perform extensive breath-hold activities near a treatment chamber.

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Reply: Safety will increase with knowledge

Dear Editor,

Thank you for the opportunity to respond to Dr Schipke and his colleagues. It seems these writers are responding to a paper promoting extreme breath-hold diving. This is not the case; I am simply describing what people do and attempting to understand their physiology. Since part of the scientific community decided to stop reporting on the physiology behind deep diving in the 1990s, record setting has continued evolving at a tremendous pace. Thus, the lack of involvement of scientists has had little relevance to recruitment to these sports. Turning a blind eye to these activities simply does not work. My target audiences for the reviews were both advanced freedivers and researchers, and my aim to make diving safer. Many divers have appreciated the papers for telling them more about the risks they face. Finally, there is a response from some researchers, albeit a negative one: I was hoping to stimulate renewed interest in researching these factors and potential risks, not a recommendation of 'non-reporting' and neglect.

I share with the writers their concern that these activities are potentially dangerous, but believe they could be made safer by a better understanding of the real and imagined risks. I have also come to realise when studying these divers (after initially sharing the view of the writers that these divers must be careless daredevils) that these sports men and women are not there to take risks but to limit them. Just like climbers using advanced safety systems, they try to reduce the risks to a minimum. The safety routines of these sports can be learned at serious climbing or diving clubs. But how could divers avoid risks unknown to them? This was well put recently by a world record holder in deep diving when thanking me after reading my last paper:

> "*Freediving, just like climbing, is not about taking risks, but on the contrary about how to avoid risk. That's why we need researchers who find out and tell us which the major risks are when we dive, and what isn't a risk. If nobody does – then we are exposed to risk!*"

Many sports and other activities are 'inherently dangerous', but this does not deter us from trying to make them safer.

Why should it in this case? To achieve maximal performance is the goal in most sports, there is nothing surprising or wrong with that. My series of review papers was aimed to provide a state-of-the-art update of our knowledge of the physiology of competitive free diving and, with appropriate rather than negative responses from others, I believe my goal of making diving safer can be achieved. Apart from active divers, who will dive safer when given some serious fact-based information, the target audience for these papers is the scientific community. New data has to be added, we cannot simply parrot old 'facts' when these have been proven wrong by actual performance; more research is needed!

I am grateful for Schipke et al's response as this hopefully might stimulate new research from physiologists who do not turn a blind eye to these extreme sports. To simply dismiss the entire field of research by saying "*freediving is dangerous*" seems to be a very unscientific attitude. If ignorance in any field would make things safer, I would be very surprised. With that said, there are points in the letter that deserve direct comment as they are somewhat surprising, and in some cases make me wonder if they have read my papers carefully enough, as all the points made and the associated risks have been dealt with in my reviews.[1–3]

Extended apnea and hypoxic brain injury

Schipke et al. dismiss the lack of alarming increases of markers of brain damage as of little informative value.[4] Another study looked at cognitive function in divers who have experienced syncope often, without evidence of damage,[5] and both studies were cited in my first review.[1] Negative effects from dive-related hypoxia cannot be excluded, but similar periods and levels of hypoxia are experienced by climbers, sleep apnea and COPD patients and by many highly cerebral diving mammals. While there may be other consequences in the patient groups, it seems the brain tolerates this surprisingly well. Perhaps we do have an ability to cope with this type of hypoxia, at least after training? Hypoxic preconditioning in rats is neuroprotective.[6] If repeated exposure reduces risk, this could be interesting also in other groups, e.g., limiting the impact of stroke. I have studied many competition divers directly after a syncope, and found that they generally recover completely within 3–4 min. Syncope in a competition leads to disqualification, but the situation is quickly resolved by rescue divers performing the blow/tap/talk manoeuvre.[2] To say that long term effects must be suspected is not supported by present scientific evidence, but hypoxia in apnea clearly deserves further study. After years in the field, I know personally some 10 of the 20 best deep divers in the world, and there is nothing wrong with their brains: the most merited man in deep diving is an airline pilot, and the most merited woman has just finished her doctoral degree.

I agree completely that people can die from breath-hold diving and that deep divers are potentially at greater risk. Only, competition diving is not dangerous per se. In the approximately 40,000 competition dives in the six existing competition disciplines organised by AIDA International, the leading diving competition federation, there have been no fatalities to date. Diving alone is, however, dangerous; diving on a sled as in non-competition record attempts has indeed involved lethal accidents. Spearfishing also causes casualties, as does training without the proper safety measures in place. To learn from competition diving is the best we can do to increase safety in freediving. If all freedivers were using the safety systems used in diving competitions, fatalities would fall to a minimum. That is why these safety systems have been described in detail in my reviews.[2,3]

Barotrauma

This was not a training recommendation – I simply report what people do. I also report the possible side effects in my review: that this may lead to capillary rupture and pulmonary oedema. Surprisingly often it does not. Why? Are there protective mechanisms? How about researching this? Scuba divers are told how to avoid barotrauma, drowning and decompression sickness (DCS); why not give freedivers the same information? Many scuba divers die each year, but this activity is not categorically advised against, yet scuba diving is physiologically much more unnatural than freediving, and there are no mammalian counterparts to compare our responses with to see whether they are protective or pathological. Competing athletes in all sports try to fool, if not physics, at least physiology. That is an inherent quality of sport. In the case of the described diving and training methods, however, all competition divers already know these 'tricks', but they may not be aware of the possible side effects.

Glossopharyngeal insufflation (GI) and exsufflation (GE)

Again, risks and effects pointed out by the authors have been dealt with in my review.[3] Trumpeters will likely be at twice the risk according to their own reference, with lung pressures of 150 mm Hg.[7] Our group was the first to describe GI in divers scientifically, including pointing out (but not proving) the potential risks.[8,9] The study by Chung et al used maximal GI, which is not normally used by divers, and also, as noted by these authors, the supporting pressure from water may counteract the development of pneumomediastinum.[7] Total lung capacity (TLC) being lower in an immersed diver, it may be that an immersed diver can use GI to reach normal 'dry' TLC before diving, without side effects. The conclusions by Chung et al. are more tentative and nuanced than Schipke et al would have us believe, and these matters clearly deserve further study.

Lung squeeze and diaphragmatic contractions

As known to all divers, not only can diaphragmatic contractions produce waves of negative pressure, but they will during every extended dive.[10] However, at least 90% of all deep divers present back at the surface without any

signs of side effects. This is good news. Why is this so? And why not in the remaining, perhaps 10%, of divers? Are there mechanisms that could be protective in other groups as well? Again, a problem worthy of deeper scientific study.

Decompression sickness

Although the possibility of DCS after repeated apnea diving has been pointed out by others, we reported the first Doppler bubble score grade 1 in a freediver after a single deep dive.[3] This is alarming, especially as diving gets progressively deeper. I recommend strongly that chamber facilities should always be provided to deep freedivers – not only to those with previous DCS – and that freediving tables should be developed. Post-dive oxygen is already being used as divers become more aware of the potential DCS risk.[3]

Whilst the deepest diving in the variable weight and no-limits disciplines is certainly not something I would recommend, the people doing this nevertheless provide a model for studying extreme survival. From a natural science perspective, it is interesting to try to understand how this is at all possible. This may also be useful for other survival situations, not the least in emergency medical situations. I believe better understanding of how our bodies work is a good thing, and that it will increase survival in many different situations. I remain uncertain in what ways my reviews could be considered "*uncritical*" and how these writers suggest we go about our common goal, to make diving safer, without trying to better understand how our bodies work when we dive. To ignore good news and report only the bad is unscientific. The only way to make the divers listen is by gaining their trust. This is not done by banning their sport, but by providing solid facts. That is, I believe, our job as scientists.

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Key words
Breath-hold diving, hypoxia, physiology, decompression sickness, barotrauma, safety, letters (to the Editor)

Diving medicine for scuba divers

Dear Editor,

Diving medicine for scuba divers by Edmonds, Thomas, McKenzie and Pennefather is a web-based book available as a free, downloadable text. Recently it has had to move to a different web host. Thus, it and all bookmarked subdirectories will no longer be available at the old address, which one needs to delete and replace with a new one: <www.divingmedicine.info>.

Since the text was made available almost three years ago, there have been over 30,000 downloads. Because we do not apply copyright restrictions, dive instructors and clubs are encouraged to supply copies to their clients and members; diving physicians have supplied specific chapters to their diver patients. Thus we have no idea of the actual number of copies distributed.

We have upgraded the text, and so even those with downloaded copies should now replace them with the 2012 4th edition. Our appreciation goes to all those who have made suggestions for corrections and modifications.

Carl Edmonds
Phone: *+61-(0)2-9976-5556*
E-mail: *<puddle@bigpond.net.au>*
Website: *<www.divingmedicine.info>*

Key words
Textbook, world wide web, recreational diving, letters (to the Editor)

Continuing professional development

CME activity 2012/2

Breath-hold diving

David Cooper and Margaret Walker

Accreditation statement

Intended audience

The intended audience consists of all physicians subscribing to *Diving and Hyperbaric Medicine* (DHM), including anaesthetists and other specialists, who are members of the Australia and New Zealand College of Anaesthetists (ANZCA) Diving and Hyperbaric Medicine Special Interest Group (DHM SIG). However, all subscribers to DHM may apply to their respective CPD programme coordinator or specialty college for approval of participation. This activity, published in association with DHM, is accredited by the ANZCA Continuing Professional Development Programme for members of the ANZCA DHM SIG under Learning Projects: Category 2 / Level 2: 2 credits per hour.

Objectives

The questions are designed to affirm the participant's 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.

Background reading

Practitioners are referred to the following background references and reading.

1 Balestra C, Levenez M, Lafère P, Dachy B, Ezquer M, Germonpré P. Respiratory rate can be modulated by long-loop muscular reflexes, a possible factor in involuntary cessation of apnea. *Diving Hyperb Med*. 2011;41(1):3-8.

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How to answer the questions

Please answer all responses (A to E or F) as True or False. Answers should be posted by e-mail to the nominated CPD coordinator.

For EUBS members for this CPD issue this will be Peter Müller.

E-mail: *<peter.mueller@eubs.org>*

For ANZCA DHM SIG members, this will be David Cooper.

E-mail: *<david.cooper@dhhs.tas.gov.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.

Successfully undertaking 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

Breath-hold diving, exercise, hypoxia, physiology, safety, MOPS (maintenance of professional standards)

Questions on next page

Question 1. Physiology of apnea diving 1:

A. Respiratory muscle spasms can, with time, build up enough metabolites to trigger a long-loop reflex and induce the cessation of apnea even in the absence of pronounced hypercapnia and/or hypoxia.

B. Although the spleen is an important erythrocyte storage site in diving mammals, studies in splenectomised subjects confirm this is of little relevance in increasing human apneic duration.

C. Yoga-breathing hyperventilation reduces the large, 'slow' tissue stores of CO_2, maximizes O_2 storage and may reduce O_2 consumption by >30%, allowing longer apneas before asphyxia develops.

D. Lactate accumulation decreases the affinity of myoglobin for O_2, thus facilitating diffusion of O_2 to mitochondria for sustained oxidative phosphorylation during apnea – prolonging aerobic metabolism in parallel with anaerobic metabolism.

E. In deep apnea diving, passive flooding of the paranasal sinuses and middle ears with seawater during descent avoids barotrauma whilst circumventing the need for active insufflation with precious air.

Question 2. Physiology of apnea diving 2:

A. Fasting does not prolong apnea duration because, during fasting, the body relies mainly on fat metabolism – which requires significantly more O_2 per unit energy produced than does metabolism of carbohydrate alone.

B. Muscle fatigue is primarily the result of disruption of contractile processes by acidification.

C. Vital capacity correlates with diving performance.

D. A high ratio of compressible air spaces to tissues in the body will lead to a more rapid switch to negative buoyancy during descent.

E. Aside from a small contribution from energy-rich phosphates, the production of lactate from muscle glycogen is the most important anaerobic process for energy production.

Question 3. The physiological diving response:

A. A full diving response is only developed with simultaneous face chilling, mainly of the forehead and eye region.

B. The main effects are a selective vasoconstriction in areas tolerant to hypoxia, and sympathetically-mediated tachycardia developing during the initial 30 seconds of apnea, with a corresponding increase in cardiac output.

C. The diving response does not occur in warm water even if there is a temperature gradient between the air and water.

D. Is a reflex that occurs only in air-breathing mammals.

E. Protects the breath-hold diver from decompression sickness.

F. Splenic contraction is augmented by hypercapnia.

Question 4. Lung packing:

A. Using the oral cavity and tongue to repeatedly press down small volumes of additional air into lungs already filled to total lung capacity (TLC) ('lung packing' a.k.a. 'glossopharyngeal breathing') allows a breath-hold diver to increase total lung capacity by several litres.

B. Autoinflating the lungs in this manner increases oxygen stores at the start of a dive, increases alveolar surface area and reduces respiratory membrane thickness.

C. Lung packing increases apnea performance in trained divers by up to 40%.

D. High intrathoracic pressures from lung packing reduce venous return, and can cause syncope if the diver does not submerge quickly or deeply enough.

E. The depth which can be reached without risk of barotrauma is set by the relation between the inspired lung volume at the surface (classically assumed to be the TLC reached by maximal inspiration) and the diver's residual lung volume.

Question 5. Complications of breath-hold diving:

A. Violent diaphragmatic contraction during the 'struggle phase' of apnea may trigger pulmonary capillary stress failure.

B. Although uncommon in breath-hold divers, pneumomediastinum due to pulmonary barotrauma of ascent may occur because of shear forces between lung compartments (owing to non-uniform compliance reduction), and airway closure and gas-trapping (causing some areas to become overdistended to the point of rupture).

C. Cerebral air embolism is a risk of pulmonary hyperinflation by glossopharyngeal insufflation.

D. Ascent syncope is a direct effect of the change in ambient (and therefore gas partial) pressures and is a different entity from 'shallow water blackout'.

E. Repetitive dives even to moderate depths with short intervals may cause an excess nitrogen load over time, leading to decompression sickness in apnea divers.

The world as it is

New hyperbaric facilities for the Prince of Wales Hospital, Sydney

Rob Turner

The Department of Diving and Hyperbaric Medicine located at the Prince of Wales Hospital, Sydney officially opened its new facility on 07 February 2012. This houses a four-compartment multiplace recompression chamber which is currently the largest rectangular chamber in the world. Designed and built in Australia by Fink Engineering, the chamber is 17.2 metres long by 4.1 metres wide. Three compartments are rated to a working pressure of 335 kPa with the fourth, 'divers' compartment rated to 557 kPa. The main treatment compartment is designed to treat up to 12 'routine' patients comfortably (Figure 1) and a second roomy compartment to treat two intensive care patients. There are two smaller compartments, one used primarily as an air lock and the other higher-pressure compartment used to recompress divers. Both smaller compartments are equipped with flushing toilet facilities.

The chamber is equipped with several unique features including adjustable LED lighting throughout and an ICU gas pendant/work station in the critical care compartment. Other features include 'fly-by-wire' operation, a video entertainment system and touch-screen operation (Figure 2). Advanced safety features include a fire deluge system which, in the event of an emergency, simultaneously cuts power and oxygen to the chamber, commences emergency depressurisation and floods the chamber with 2,000 litres of water. We also have a recently commissioned Perry monoplace chamber supplied by Hyperbaric Health.

One of the unique and complex aspects of this project was the requirement to maintain a fully functioning facility during the upgrade works. In order to achieve this, the new chamber (including plant) was installed in an area previously used for offices, whilst the original chamber continued 'normal' operations. Once the new chamber was commissioned, the old chamber was removed through the roof and the clinical and non-clinical areas were rebuilt. This period of time was challenging as the staff had to work between two chambers located in a construction zone.

The clinical areas and office space provide new consultation rooms, wound treatment areas and a procedure/critical care room for intensive care patients. The non-clinical area includes a 25-person conference room which was recently used for the ANZHMG Introductory Course in Diving and Hyperbaric Medicine which is convened by A/Prof Michael Bennett and hosted annually at the Prince of Wales Hospital.

The Department of Diving and Hyperbaric Medicine is staffed by six staff specialists (two full-time equivalents), one training registrar, five full-time nursing staff, two full-time technicians and an office manager. The Department is accredited for registrar training by the Australian and New Zealand College of Anaesthetists and the Australian College of Emergency Physicians. Currently, the unit is treating an average of 25 'routine' patients a day. The monoplace chamber, once commissioned, will increase our treatment capacity by a further four patients daily.

We welcome visitors or enquiries about training positions; requests to visit the facility should be directed to Gabrielle Janik, e-mail: <Gabrielle.janik@sesiahs.health.nsw.gov.au>

Rob Turner, Medical Director, Department of Diving and Hyperbaric Medicine, The Prince of Wales Hospital, Sydney
E-mail: *<robert.turner@unsw.edu.au>*

Key words
Hyperbaric facilities, equipment, hyperbaric medicine

Figure 1
Interior of part of the 12-person 'routine' chamber

Figure 2
View of control panel and three chamber entry doors

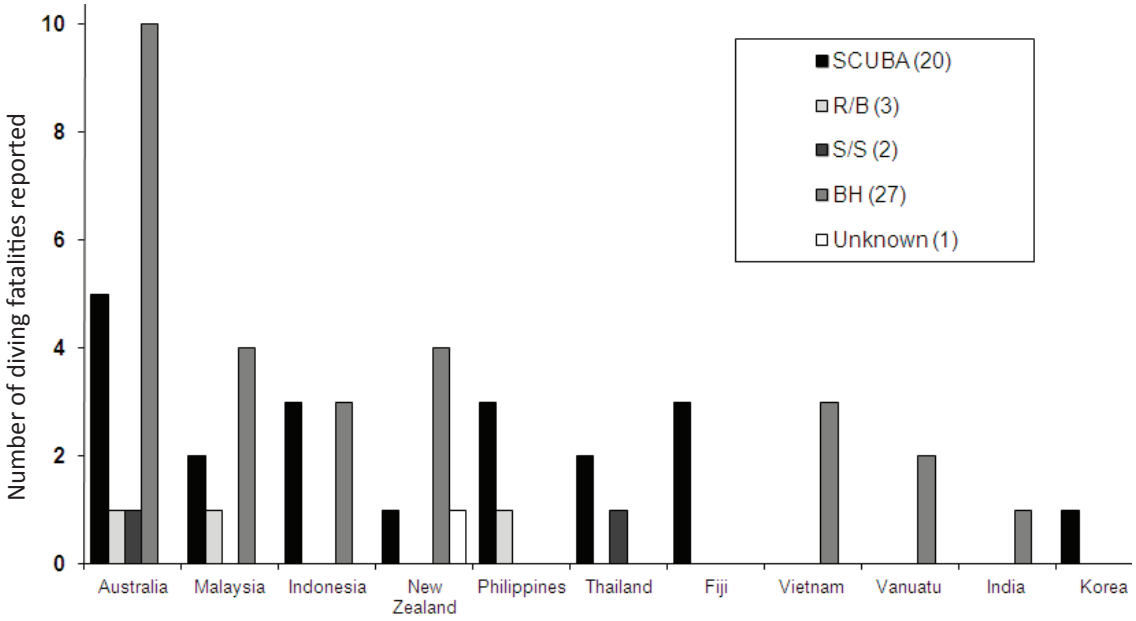
Diving-related fatalities and decompression illness in the Asia-Pacific region 2010 as reported to the Diver Alert Network (provisional)

John Lippmann

The data as reported to DAN AP for the number of diving-related deaths in this region during the 2010 calendar year are shown in Figure 1. We believe that the numbers from Australia and New Zealand are reasonably accurate. However, we suspect that the numbers in most of the other countries are understated as a result of poor reporting from these countries. We strongly encourage divers and physicians to notify DAN if they hear about a diving fatality so that we can better understand where and how these problems occur.

Figure 1

Diving-related fatalities in the Asia-Pacific in 2010 (provisional); R/B – rebreather; S/S – surface supply; BH – breath-hold



The data as reported to DAN AP for the number of recreational divers treated for decompression illness in this region during the 2010 calendar year are shown in Figure 2.

The numbers for Thailand and Indonesia are estimates and/or minimums as data were not provided by all of the chambers.

Once again, the data from Korea appear to be high and may indicate a safety issue for Korean divers, a low threshold for treatment and/or possibly an issue with reporting. Fiji, Palau, Yap, and China each reported one case of treated DCI.

John Lippmann
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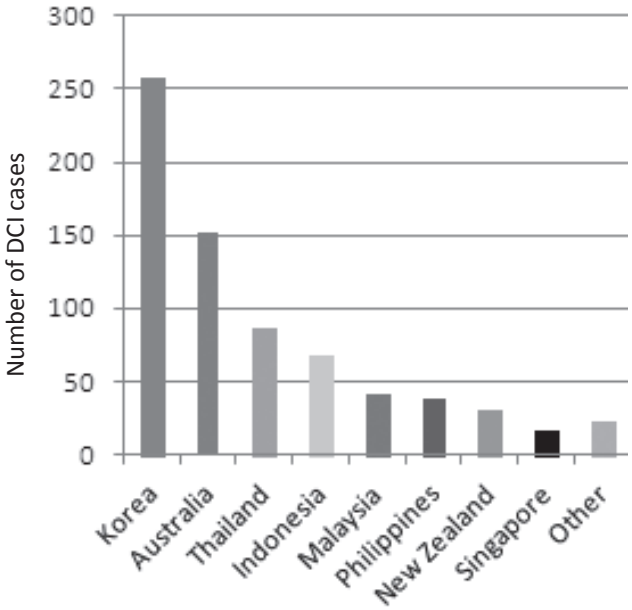
Key words
Recreational diving, deaths, decompression illness, decompression sickness, DAN – Divers Alert Network

Figure 2

Decompression illness in the Asia-Pacific region in 2010 as reported to DAN Asia Pacific



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 submitted to, or accepted by other journals should 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 2011, the SPUMS Academic Board consists of:
Associate Professor David Smart, Education Officer;
Associate Professor Simon Mitchell;
Associate Professor (retired) Mike Davis.

All enquiries and applications should be sent 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

Summary of the Minutes of the SPUMS Executive Committee Meeting 12 November 2011 at the Prince of Wales Hospital, Randwick

Opened: 0925 h

Present

M Bennett, K Richardson, J Lehm, D Smart, P Smith, G Williams, G Hawkins, M Davis and S Lockley (invited guest as ASM 2011 Convenor)

Apologies

C Acott and A Fock

An unedited copy of the minutes is available by application to the Secretary.

1 Minutes of previous meeting

1.1 Minutes accepted for Executive Committee Meeting held in Guam May 2011 with clarification that the DHM finances are the joint responsibility of the Executive Committees of SPUMS and EUBS.

1.2 Minutes for the Executive Meeting held at Prince of Wales Hospital November 2010 have been previously accepted by e-mail consensus and published in DHM.

2 Matters arising from previous minutes

2.1 There are no dive training courses for people with diabetes in Australia or NZ. The possibility is the subject of ongoing study by MB and others.

2.2 A UK position paper on diving with epilepsy is in draft and has input from SPUMS. The Society will be updated on developments.

2.3 MD continues work on the Editor's Handbook for handover to the next editor when that arises.

2.4 A website hot key has been added to enable members to access their membership renewal and edit details.

2.5 The assets list has been updated.

KPI satisfied: Minuted actions addressed in a timely fashion.

3 Annual Scientific Meetings

3.1 ASM 2011:

Final budget shows an overall profit of AUD3,924.23. Registrant numbers – 58 full registrants including convener and speakers, 8 accompanying adults with 4 children.

3.2 ASM 2012:

Update provided by Cathy Meehan. Scheduled for Madang, PNG 20–26 May. See website for link to booking sites. The cost of conference registration will be kept as low as possible.

3.3 ASM 2013:

Strong support expressed for a joint EUBS, SPUMS and SAUHMA ASM to be held on Reunion Island in August–September 2013; planning under way.

3.4 Future Meetings Steering Committee: Cathy Meehan (Chair), Sue Paton and Janine Gregson. Three destinations are under consideration: Saipan, Micronesia; Wakatobi, Indonesia and the Maldives.

3.5 Update to Convenor's Handbook: A significant update is required and under way.

4. Journal matters

4.1 Editor's Report November 2011: MD gave a detailed report. Medline citation, combined with the amalgamation with EUBS, has increased submissions to the Journal by at least 150% over the past 3 years. This increase has added considerably to the Editor's and his Assistant's workload. Abstracts from all three of the 2012 issues are now on Medline but funding is required to go back three years. The Journal is in budget, but falling numbers continue to pose a threat. SPUMS and the EUBS have been formally established as the publishers of the journal. Guidelines for commercial advertising in the Journal were published in the September 2011 issue.

4.2 Financial independence of the Journal: Following e-mail correspondence from the EUBS, the financial reporting in matters relating to the Journal are under active review. The principle is open and full financial dealings for the Journal. P Smith as Assistant Treasurer will undertake oversight on matching journal budget to actual expenditure and conduct an internal audit beginning with the 2011 financial year.

4.3 EBSCO and Infotrieve search engines: The committee recommend against accepting these proposals.

5. Website update

5.1 Updating SPUMS website: Updated 'Purposes and Rules' to be posted on the website.

5.2 Proposal for Honorarium for the Webmaster to cover costs of maintaining the website: The Webmaster will be allowed an honorarium of AUD2,000 to commence 2012 financial year to be used as he/she sees fit in relation to developing the website.

KPI satisfied: Request for change to website addressed in a timely fashion.

6. Education Officer's report

6.1 Progress of Stellenbosch University DHM diploma alignment: DS reports the ANZCA SIG certificate lines up well with international requirements with the addition of DMAC IIa gap course run in conjunction with AHDMA. Consideration should be given to running an Australian or New Zealand based DMAC IIa course every 2–3 years.

6.2 RCC facility accreditation update: DS reports this should be the role of the ANZHMG representative in conjunction with HTNA using Australian Standard 4774.2 once updated.

6.3 RACGP Accreditation progress report: Gaining CPD

accreditation from the RACGP remains problematic and may involve significant costs. S Lockley undertakes to further investigate and establish options for accrediting SPUMS ASM for RACGP CPD points.

KPI Satisfied: SPUMS Diploma has been awarded and issued to Dr Geoff Frawley.

7. Treasurer's report

7.1 Additional signatories: K Richardson and P Smith completed paperwork for their inclusion on the electronic signatory list. SPUMS Sec credit card transfer documentation was initiated for K Richardson.

7.2 Publishing of annual SPUMS accounts on the website rather than in the Journal: approved.

7.3 Budget 2010–2011 to date: on target for increased profit margins compared with last year.

7.4 Barrett, Baxter, Bye: Offer from BBB to cover up to AUD20,000 of the costs associated with auditing by the ATO at charge of AUD350 per year. Offer discussed but rejected.

KPI satisfied: Counter-signing responsibilities have to date been generously continued by S Lockley.

8. Public Officer's report

KPI satisfied: Lodging of documents with authorities before due date achieved, with difficulties noted.

9. Secretary's report

9.1 Use of generic e-mail addresses: All SPUMS ExCom are issued with a generic <spums.org.au> e-mail address as their official point of contact as advertised in DHM.

9.2 SPUMS membership group e-mail protocol: Group e-mails will be sent only at the request of the SPUMS President and will be submitted to the Webmaster for distribution. Each request must specify target audience, for example: the diving doctors list, current members, ASM registrants, all SPUMS contacts past and present.

9.3 Dive Med Course details approval for website: Information on diving medicine courses in Australasia should be available on the SPUMS website.

9.4 Distribution of SPUMS and DHM intellectual property: Protocols for distribution of documents were discussed and confirmed as follows: SPUMS Diving Medical: available on website under [HOME] then [Position Papers]; SPUMS Purposes and Rules: available on website under [HOME] then [Constitution]; Requests for hard copies of DHM: Refer to SPUMS administrator Steve Goble with cost to enquirer; Requests for soft copies individual DHM article since 2006: DHM Editor to consider individual requests; Requests for soft copies individual DHM article up to 2002 (soon to 2006): Refer to Rubicon database.

9.5 SPUMS policy for handling medical enquiries via electronic media: Patients making enquiries will be advised to seek review with a medical officer trained in diving medicine and referred to the Diving Doctors List. Queries from medical professionals will be forwarded for response to the SPUMS President as voice of the Society. All statements lodged on the SPUMS online forums are individual opinions, not those of the Society.

KPI satisfied: All Minutes circulated in draft form within four weeks of ExCom meetings.

10. Membership matters

10.1 Update on Membership status: Current membership: 462 full, 58 associate, 22 retired, 13 corporate, total membership 555 for 2011. The Website is now able to track membership by financial year and is logging many new electronic members.

10.2 Membership Database: It is noted that the SPUMS membership database is currently out of date and requires significant editing. MD will publish notice in next edition DHM requesting members log on and update their details. SPUMS Administrator S Goble will be asked to review database and clean files.

10.3 Request from AHDMA for electronic journal access: AHDMA President Tony Lee's proposal rejected at this stage as a secure electronic format of the Journal is not yet available.

10.4 Request from EUBS for global membership: E-mail correspondence received from EUBS asking us to consider the concept of global membership between EUBS and SPUMS. We are discussing the concept, but there are many issues remaining.

10.5 SIG and SPUMS at the NSC ASA: Unfortunately no desk space was available this year but enquiries will be made for all coming ANZCA events.

10.6 Report from ODEX 2011: A SPUMS team, headed by Cathy Meehan, had a successful weekend at the ODEX event in Brisbane. A position has been secured for SPUMS at ODEX in Sydney on 01–02 September 2012.

10.7 SPUMS Facebook page: The Facebook page is operating at 'South Pacific Underwater Medicine Society'.

10.8 Other initiatives: Discussed.

11. Other business

11.1 MSAC review process 2011: MB and DS reported that recently finalised documentation supports treatment for soft tissue-radiation injury, particularly proctitis, but is negative with regard to non-diabetic chronic wounds. A dissenting report has been submitted with respect to the latter.

11.2 Proposed SafeWork Legislation and Codes of Practice: Despite two rounds of submissions by DS on behalf of SPUMS to appeals processes in March and August 2011, the Government has failed to address the

deficiencies in the proposed legislation. He will write a letter to the appropriate Minister outlining our concerns. K Richardson will attempt to organise a sit-down meeting with the government representatives that can influence editing of legislation still under consideration.

11.3 SPUMS' relationship with HTNA: MB proposed an enhanced formal relationship between SPUMS and HTNA. Correspondence is under way.

12. Correspondence

15.1 Always e-mail to M Bennett and M Davis: E-mail correspondence tabled and discussed.

15.2 Workplace Health and Safety Queensland: A web address for the position statement on recreational diving medical has been notified to SPUMS: <http://www.deir.qld.gov.au/workplace/resources/pdfs/alert-advice-cert-divers.pdf>

15.3 ICHM to SPUMS: SPUMS has nominated MB to sit as their representative on the ICHM committee.

15.4 UHMS to SPUMS Treasurer: Arrangements to continue the exchange of journals are under way.

13. Next meeting

SPUMS Exec teleconference roughly scheduled for February/March 2012 with precise date to be arranged by e-mail closer to time.

Closed: 1706 h

Key words
Medical society, meetings

Australian and New Zealand Hyperbaric Medicine Group (ANZHMG) Chairman's Report, April 2012

Medical Services Advisory Committee

In 2010, the ANZHMG (a sub-committee of SPUMS) supported by the Australian Healthcare and Hospitals Association and the Australian Society of Anaesthetists, forwarded a detailed submission regarding Medicare funding for hyperbaric oxygen treatment of late soft-tissue radiation injury and non-diabetic hypoxic problem wounds. This submission was accepted by the Medical Services Advisory Committee. Associate Professors David Smart and Mike Bennett were invited to join the Committee to undertake evaluation of the submission. The process was concluded in October 2011. At present there has been no further news from the Federal Government regarding the outcome of the MSAC review and whether or not funding has been recommended. There were a number of areas within the report produced by MSAC with which ANZHMG was not happy and a very vigorous criticism of the report was sent to MSAC, endorsed by the four organisations.

Australian Federal Government Model Work, Health and Safety Regulations

In 2010, Safety Work Australia was tasked by the Federal Government to develop a model set of legislation to be universally applied to all industries including the diving industry. Despite two submissions totalling 25 pages, which outlined significant risks to safety inherent in the legislation, the Federal Government did not even acknowledge SPUMS' input. Some of the risks were outlined in my report last year. Most of the risks relate to inadequate training of divers and a massive gap in detail of safety processes covering a construction diver compared to those covering a general diver, and abandonment of the diver training standards AS2815 series. Divers performing high-risk work (very poorly defined) are stated to be governed by AS2299.1, however 'general divers' are not. None of the issues raised in SPUMS' first submission were addressed, and a Federal Government *Draft code of practice: managing risks associated with diving work* was released in March 2011. Whilst this document had improved detail relevant to diving operations, it did not correct the significant errors that had been enshrined in the legislation.

The Australian Model Work Health and Safety Regulations also had the effect of quarantining divers in Australia from those in New Zealand because, as national legislation, it overrides the Australian and New Zealand Standards. It also requires the medical practitioner providing certification of divers to be registered in Australia.

A copy of the regulations can be accessed at:
<http://www.safeworkaustralia.gov.au/AboutSafeWorkAustralia/WhatWeDo/Publications/Pages/Model-WHS-Regulations.aspx>
The section on diving work commences on page 177, section 4.8.

The code of practice supporting diving is not currently accessible; it appears to have been removed from the website after the public comment phase.

The legislation mandates that all occupational divers receive a current certificate of medical fitness to dive by a doctor with appropriate training in underwater medicine. By the legislated reference to AS2299.1:2007, SPUMS is referred to as the appropriate body to provide information on training courses in diving medicine for medical practitioners. The linkages for this mandate are as follows:
Definition of *appropriate* training in underwater medicine (Page 4):
Appropriate training in underwater medicine means training that results in knowledge of the matters specified in clause M3 of Appendix M to AS/NZS 2299.1:2007 (Occupational diving operations–Standard operational practice).
The requirement for workers to hold a current certificate of

medical fitness (page 177, clause 168):
Division 2 General diving work–Fitness and competence of worker
168 Person conducting business or undertaking must ensure fitness of workers
(1) A person conducting a business or undertaking at a workplace must not direct or allow a worker to carry out general diving work or undergo training for general diving work unless the worker holds a current certificate of medical fitness.
Definition of current (page 15):
Current certificate of medical fitness means a certificate of medical fitness that:
(a) was issued within the past 12 months, and (b) has not expired or been revoked.
Requirement that the certificate is issued by a registered medical practitioner with *appropriate training in underwater medicine* (page 178, clause 169):
169 Certificate of medical fitness
A certificate of medical fitness must:
be issued by a registered medical practitioner with appropriate training in underwater medicine
and (E) Definition of registered medical practitioner (page 39)
Registered medical practitioner means a person registered under the Health Practitioner Regulation National Law to practise in the medical profession (other than as a student).

Prior to this legislation, the professional diving industry of Australia referred to a series of Australian/New Zealand Standards for guidance that covered (1) diving training (2815 series), (2) operational diving in different industries such as construction, onshore and offshore diving, scientific diving, recreational instructors, film and photographic diving (2299 series), and (3) work in compressed air and hyperbaric facilities (4474 series). These standards have developed and evolved with industry participation and cooperation since 1965. Only AS/NZS 2299.1:2007 has been referred to in the Australian Model Work Health and Safety Regulations. Even more strangely, recreational standard AS4005.2 has been referenced in clause 171 competence of worker – general diving work. This clearly demonstrates that those who drafted the legislation did not consult appropriately. Now that the legislation has been set in concrete, it may be regarded as a potentially serious threat to diving safety.

Diving Medicine Courses

The international collaborative to develop a curriculum and learning objectives for training in diving medicine, hosted by the University of Stellenbosch in South Africa, has mainly been focused on producing a course based at Stellenbosch University. The course will be accessible from all over the globe, in due course, through the Stellenbosch University web site. A process has not been developed yet to allow recognition of prior learning. The Stellenbosch course will form a template against which all diving medicine courses can be compared. I will produce a discussion paper regarding international parallels in diving medicine training for a future edition of *Diving and Hyperbaric Medicine*.

David Smart
Chairman ANZHMG

Key words
Occupational diving, working in compressed air,operations–diving, diving at work, safety, standards, medicals–diving

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SPUMS Diploma in Diving and Hyperbaric Medicine

Dr Geoff Frawley (Melbourne) was awarded the Diploma in August 2011. The topic of his project was:

Paediatric hyperbaric oxygen therapy in Victoria 1998–2010: case series and review

Dr Si Jack Chong (Singapore) completed a project entitled:

Characterization of early thermal burns and the effect of hyperbaric oxygen treatment: A randomized controlled pilot trial

This was accepted towards the Diploma in March 2012.

David Smart
Education Officer

ANZCA 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 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 Suzy Szekely, FANZCA
Chair, ANZCA/ASA Special Interest Group in Diving and Hyperbaric Medicine
E-mail: *<Suzy.Szekely@health.sa.gov.au>*

Advertising in *Diving and Hyperbaric Medicine*

Commercial advertising is now welcomed within the pages of Diving and Hyperbaric Medicine. Companies and organisations within the diving, hyperbaric medicine and wound-care communities who might wish to advertise their equipment and services are welcome. The advertising policy of the parent societies – EUBS and SPUMS – appears on the journal website: <www.dhmjournal.com>.

Details of advertising rates and formatting requirements are available on request from:
E-mail: <editor@dhmjournal.com>
Fax: +64-(0)3-329-6810

Tri-Continental Scientific Meeting on Diving and Hyperbaric Medicine

Reunion 2013

For the first time ever
EUBS, SPUMS and SAUHMA will hold their ASMs as a joint scientific meeting

Location: Reunion Island, Indian Ocean

A full week of science, fun and social interaction on this most exotic island

Save the date!
21–29 September, 2013

Full details will appear later in the year on the Society websites

EUBS European Underwater and Baromedical Society

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EUBS European Underwater and Baromedical Society

38th EUBS Annual Scientific Meeting 2012

Final Announcement

Date: 11–16 September 2012
Venue: Sava Centre, Belgrade, Serbia

11–12 September: ECHM Consensus Conference
12–15 September: EUBS Annual Conference
12 September: EUBS Workshop
16 September: DAN Divers Day

Hosts: The Centre for Hyperbaric Medicine and the University of Belgrade School of Medicine

Chairman of the Organising Committee: Miodrag Zaric
General Secretary of the Organising Committee: Mariana Sedlar
Executive Secretary of the Organising Committee: Alessandro Marroni

Scientific Committee: Alessandro Marroni (IT)(Chairman), Costantino Balestra (BE), Vladimir Bumbasirevic (SRB), Jordi Desola (ES), Peter Germonpre (BE), Tomislav Jovanovic (SRB), Jacek Kot (PL), Yehuda Melamed (IL), Djordje Radak (SRB)

Conference main topics

Fundamentals of hyperbaric oxygen and pressure-related physiology and pathophysiology
Pressure physiology and medicine
Clinical diving and hyperbaric medicine
Basic and applied research in diving and hyperbaric medicine
Occupational Health issues in diving and hyperbaric medicine
Cost-benefit in HBOT

EUBS Workshop

What is the point of research in hyperbaric medicine – if there is a point, how can we do it better?

ECHM 9th Consensus Conference

Organisation of a clinical hyperbaric therapy centre and related health management issues

Working group meetings during conference:
EUBS Executive Committee, ECHM Executive Board,
ECHM Board of Representatives, EDTC Medical Committee

Language: The official language of the conference will be English.

Contact details

Centre for Hyperbaric Medicine
Mackov kamen 24a
11040 Belgrade, Serbia
Phone: *+381-(0)11-3670-158*
Fax: *+381-(0)11-2650-823*
E-mail: *<office@eubs2012.org>*
or *<chm@scnet.rs>*
Website: *<www.EUBS2012.org>*

EUBS General Assembly 2012

All EUBS Members are invited to attend the EUBS General Assembly, which will take place during the Annual Scientific Meeting.

Date: Friday 14 September 2012
Time: 1700 h (approx., following scientific session)

Agenda:

1 Approval of minutes of previous GA (published in *Diving and Hyperbaric Medicine*. 2011;41:244-5.)

2 Awards and Grants

3 Financial Report

4 Journal Report

5 Website Report

6 Elections for Vice-President and Member at Large

7 Next EUBS Meetings

8 Any other business

Candidates for Vice-President and Member at Large 2012

According to the EUBS Constitution and Bylaws, it is time to elect a new Member-at-Large and, this year, also a new Vice-President. All regular EUBS members will receive an online ballot sheet by e-mail, which must be filled in and submitted before 12 August 2012.

As of 01 May 2012 the following candidates have been proposed (in alphabetical order):

For Vice-President:

Jean-Eric Blatteau is a 43-year-old senior diving medical officer in the French Armed Forces. As well as being a medical hyperbaric doctor, he has extensive research experience on the effects and mechanisms of bubble formation in decompression sickness, and has, among others, developed a clinically relevant animal model for DCS. He is a member of NATO submarine rescue and diving working groups. He has been the scientific advisor for scientific expeditions to the Clipperton Atoll (2005) and the North Pole (Deep Sea Under the Pole 2010), and he is involved currently in a humanitarian programme for preventing DCS among diving fishermen in Vietnam. He is a long-time member of EUBS.

Jacek Kot is a 47-year-old Polish anaesthetist and intensive care specialist, working at the National Centre for Hyperbaric Medicine in Gdynia. He is a Professional Diver 2nd Class, CMAS diving instructor, nitrox and semi-closed rebreather instructor and a trimix diver. He was a member of the Management Committee and of the Working Group "Safety" of the COST B14 Action, and was Member at Large of EUBS from 2004–2007. He is currently the General Secretary of the ECHM and a member of the Editorial Board of *Diving and Hyperbaric Medicine*. His professional interests span hyperbaric medicine and hyperbaric oxygen physiology, critical care, diving operations and safety, saturation decompression and sea rescue.

For Member at Large:

Samantha Lesley Blogg has been a decompression physiology researcher at the UK Defense Evaluation Research Agency (DERA, now QinetiQ), and later at the Karolinska Institute in Stockholm. She now has her own consulting company in scientific writing, but the bulk of her work is still experimental in hyperbaric and diving physiology. She is also a snowboarding, dog and horse enthusiast who likes to move around the globe.

Ingrid Eftedal is currently a research scientist at the Department of Circulation and Medical Imaging at the NTNU Faculty of Medicine in Trondheim, Norway. She was leader of the Medical Genetics Section at St Olav's Hospital in Trondheim from 2000 to 2009. She has a particular interest in gene expression changes induced by hyperbaric exposure, and leads an ongoing research project on this topic.

Pierre Lafère is a Belgian anaesthetist and hyperbaric medicine specialist, working at the Hyperbaric Centre of the Military Hospital in Brussels. He is an avid CCR and trimix diver and instructor, and has published research on diving physiology (nitrogen narcosis), hyperbaric oxygen therapy (acoustic trauma) and anaesthesiology (erythropoiesis and transfusion alternatives). He also has an interest in martial arts, photography, woodworking and mechanics.

Albert van den Brink is a Dutch cardio-thoracic surgeon, practising at the Academic Medical Centre in Amsterdam, and is also currently Head of the Department of Hyperbaric Medicine of the AMC. He trained as a diving medicine physician whilst in the Royal Dutch Navy

You will receive the ballot sheet containing full CVs and pictures of all candidates by the end of June 2012. Please make sure your e-mail address on file is correct; if needed, check your data by logging onto the EUBS website. As the ballot sheet may occasionally be blocked by a (company or institution) firewall, it may be advisable to change to your personal e-mail address. If you have not received the 2012 ballot sheet by 15 July, please contact the EUBS Secretary <secretary@eubs.org>

Royal Australian Navy Medical Officers' Underwater Medicine Course 2012

Dates: 05–16 November 2012
Venue: HMAS PENGUIN, Sydney

The MOUM 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 workshops covering the key components of the diving medical.

Cost: AUD705 without accommodation
(AUD2,100 with accommodation at HMAS PENGUIN)

For information and application forms contact:
Rajeev Karekar, for Officer in Charge,
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HMAS PENGUIN
Middle Head Rd, Mosman
NSW 2088, Australia
Phone: +61-(0)2-9647 5572
Fax: +61-(0)2-9960 4435
E-mail: <Rajeev.Karekar@defence.gov.au>

Asian Hyperbaric & Diving Medical Association 8th Annual Scientific Meeting 2012

Dates: 26–28 July 2012
Venue: Phuket, Thailand

Guest Speakers
Professor Alf Brubakk and Assoc. Professor David Smart

Post-conference course
Medical support of commercial diving
(equivalent to EDTC Level IIa)

Dates: 29–31 July 2012
Faculty: Professors Alf Brubakk and David Elliott

For all enquiries visit: <www.ahdma.org>

Royal Adelaide Hospital
Hyperbaric Medicine Unit Courses 2012

Medical Officers Course
December 2012
Unit 1 03–07 December
Unit 2 10–14 December

Diving Medical Technician (DMT) – Full Course
July/August 2012
Unit 1 30 July–03 August
Unit 2 06–10 August (lecture week)
Unit 3 13–17 August

For further information, please contact:
E-mail: <Lorna.Mirabelli@health.sa.gov.au >
Phone: +61-(0)8-8222-5116
Fax: +61-(0)8-8232-4207

Hyperbaric Technicians and Nurses Association 20th Annual Scientific Meeting 2012

Dates: 23–25 August 2012
Venue: Chateau on the Park
Christchurch

Guest Speakers
Richard Moon
Cathy Hammond,
with others to be advised

For information and registration go to:
Website: <www.htna.com.au>
or E-mail: <yvonne.denny@cdhb.health.nz>

DIVING HISTORICAL SOCIETY AUSTRALIA, SE ASIA

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

Scott Haldane Foundation

The Scott Haldane Foundation is dedicated to education in diving medicine, and has organised more than 100 courses over the past few years, both in the Netherlands and abroad. Below is a list of courses planned for the remainder of 2012. More information can be found at: <www.scotthaldane.nl>.

The new basic course (Part I plus Part II) fully complies with the current EDTC/ECHM curriculum for Level I (Diving Medical Examiner), and the different advanced courses offer a modular way to achieve Level IIa competence according to the EDTC/ECHM guidelines.

Course details for second half of 2012

22 September: Refresher Course Diving Medical Examiner, (Amsterdam, NL)
19–20 October In-depth course "*Diving accidents*" (Driebergen, The Netherlands)
09–17 November: Basic Course Part I (Maldives)
16–24 November: 20th In-depth Course "*Challenges in diving medicine*" (Maldives)
23 November–01 December: 20th In-depth Course "*Challenges in diving medicine*" (Maldives)

For further information: <www.scotthaldane.nl>

British Hyperbaric Association Annual Scientific Meeting 2012

Dates: 09–11 November 2012

Venue: Sheraton Skyline Hotel, Heathrow Airport, UK

This is a Joint BHA meeting with the Association of Aviation Medical Examiners
Meeting theme: Medicine in extreme environments
More details will be available soon.
Website: <http://www.hyperbaric.org.uk>

EUBS ASM 2014

Preliminary notice

Dates: 24–27 September 2014
Venue: Wiesbaden

The German Society for Diving and Hyperbaric Medicine (GTUeM) has appointed Dr Peter Müller to serve as the Secretary General for the EUBS ASM 2014. This will be held in conjunction with the 2014 congress of GTUeM.

For further information at this early stage contact:
E-mail: <peter.mueller@eubs.org>

Hyperbaric Oxygen, Karolinska

Welcome to: <http://www.hyperbaricoxygen.se/>. This site, supported by the Karolinska University Hospital, Stockholm, Sweden, offers publications and free, high-quality video lectures from leading authorities and principal investigators in the field of hyperbaric medicine.

You need to register to obtain a password via e-mail. Once registered, watch the lectures on-line, or download them to your iPhone or computer for later viewing.

We offer video lectures from:
- The 5th Karolinska PG course in clinical hyperbaric oxygen therapy, 07 May 2009
- The European Committee for Hyperbaric Medicine '*Oxygen and infection'* Conference, 08–09 May 2009
- The 17th International Congress on Hyperbaric Medicine, Cape Town, 17–18 March 2011

Also available is the 2011 Stockholm County Council report: *Treatment with hyperbaric oxygen (HBO) at the Karolinska University Hospital*

For further information contact:
Folke Lind, MD, PhD
E-mail: <folke.lind@karolinska.se>
Website: Editor <www.hyperbaricoxygen.se>

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>

Undersea and Hyperbaric Medical Society 46th Annual Scientific Meeting

Dates: 13–15 June, 2013
Venue: Lowes Royal Pacific Resort @ Universal Studios Orlando, Florida, USA
Phone: +1-(0)877-533-UHMS (8467)
E-mail: <lisa@uhms.org>
Web site: <www.uhms.org>

Inter-university Diploma in Diving and Hyperbaric Medicine, France

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

Instructions to authors

(Short version, updated December 2011)

Diving and Hyperbaric Medicine welcomes contributions (including letters to the Editor) on all aspects of diving and hyperbaric medicine. Manuscripts must be offered exclusively to *Diving and Hyperbaric Medicine*, unless clearly authenticated copyright exemption accompanies the manuscript. All manuscripts will be subject to peer review. Accepted contributions will also be subject to editing. An accompanying letter signed by all authors should be sent.
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
Documents should be submitted electronically. The preferred format is Microsoft® Office Word or rich text format (RTF). Paper submissions will not be accepted. All articles should include a title page, giving the title of the paper and the full names and qualifications of the authors, and the positions they held when doing the work being reported. Identify one author as correspondent, with their full postal address, telephone and fax numbers, and e-mail address supplied. The text should generally be subdivided into the following sections: a structured Abstract of no more than 250 words, Introduction, Methods, Results, Discussion, Conclusion(s), Acknowledgements and References. Acknowledgements should be brief. Legends for tables and figures should appear at the end of the text file after the references. Conflicts of interest and funding sources should be identified.

The text should be 1.5 lines spaced, using both upper and lower case. Headings should conform to the current format in *Diving and Hyperbaric Medicine*. All pages should be numbered. Underlining should not be used. SI units are to be used (mmHg is acceptable for blood pressure measurements; bar for cylinder pressures); normal ranges should be shown. Abbreviations may be used after being shown in brackets after the complete expression, e.g., decompression illness (DCI) can thereafter be referred to as DCI.

Preferred length for **Original Articles** is up to 3,000 words. Inclusion of more than five authors requires justification, as does that of more than 30 references. **Case Reports** should not exceed 1,500 words, and a maximum of 15 references. Abstracts are required for all articles. **Letters to the Editor** should not exceed 500 words and a maximum of five references. Legends for figures and tables should generally be shorter than 40 words in length.

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References
The Journal reference style is based closely on the the *International Committee of Medical Journal Editors (ICMJE) Uniform Requirements for Manuscripts*. Examples are given in detail at:
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Examples of the exact format for a standard paper and a book are given below:

1 Freeman P, Edmonds C. Inner ear barotrauma. *Arch Otolaryngol*. 1972;95:556-63.
2 Hunter SE, Farmer JC. Ear and sinus problems in diving. In: Bove AA, editor. *Bove and Davis' diving medicine*, 4th ed. Philadelphia: Saunders; 2003. p. 431-59.

Accuracy of references is the responsibility of the authors.

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Enquiries to: <research@danasiapacific.org>

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The Diving Incident Report Form can be downloaded from, or an on-line form accessed at the DAN AP website: <www.danasiapacific.org>

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Diving and Hyperbaric Medicine is indexed on MEDLINE, SciSearch® and Embase/Scopus

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