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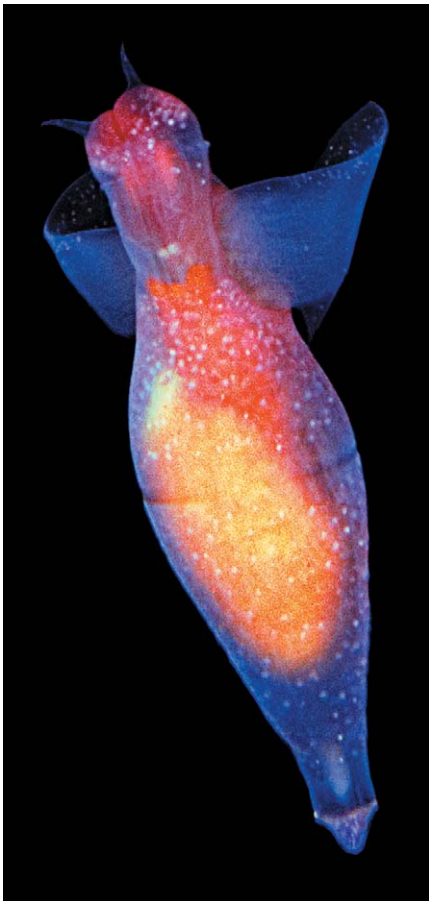

HBOT for malignant otitis externa

Do Western Australian divers dive safely?

'Sea legs' and the sharpened Romberg test

S100B – a biomarker for CNS decompression injury?

Is where a CO_2 sensor is put in a rebreather important?

Antioxidants as an endothelial pre-conditioner for divers

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PURPOSES OF THE SOCIETIES

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

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The Editor's offering

Every year, the Hyperbaric Technicians and Nurses Association (HTNA) collate and publish treatment data from hyperbaric centres in Australia and New Zealand in their magazine *Offgassing*. Thirteen units contribute, only a few small privately owned chambers and one major centre (from which unit the data were obtained independently by the Editor) not doing so. These data, reported unit by unit, are collated by Sue Thurston, the Principal Nursing Officer of the Hyperbaric Medicine Unit, Freemantle Hospital, Western Australia. Table 1 provides a brief summary of some of the data for 2009-2010.

The number of patients treated, the total treatments and the chamber runs are recorded along with the referral diagnoses, which are divided into thirty-six sub-categories in eight broad diagnostic groups (e.g., bubble injury, radiation tissue damage, problem wounds, etc). Staffing levels are recorded and incidents and complications of pressurisation and hyperbaric oxygen treatment (HBOT) are documented for both staff members who undergo pressurisation and for patients. Apart from the overall figures in Table 1, some other aspects, for which precise figures are not possible because some data are missing, are worth mentioning, particularly those related to complications of therapy and patient safety. Where staff safety is concerned, 19 minor middle-ear barotrauma events and two cases of decompression sickness were reported.

The reported rates of middle-ear barotrauma ranged from 10% to 50% of patients, but the incidence per HBOT is well under 2% overall, and of minor severity (grades 0-3 on the Edmonds scale). Only five oxygen convulsions were reported; an incidence of about 1:5,000 HBOT, which is consistent with the published literature. Interestingly one CNS 'hit' occurred at 203 kPa. Hypoglycaemia was a rarely reported occurrence, which likely reflects good management of patients with diabetes during HBOT. Loss of visual acuity was inconsistently reported, and not at all by some units. Since this is a dose-dependent side effect, with individual variation in susceptibility, it is important that this is better documented by all units in future.

The overall impact on the course of HBOT of patient-related problems was small, with only about 1:400 treatments being aborted. It would be useful to know whether the side effects of HBOT reported were mainly minor, or whether there was any longer-term or serious morbidity. This information could be obtained from the Hyperbaric Incident Monitoring Survey to which all units are invited to contribute to voluntarily. Unfortunately, apart from one conference abstract some years ago the HIMS data have never been published. Suffice to say that the oft-quoted criticism of HBOT that it is a dangerous therapy with a high complication rate is completely unfounded in the Australasian environment at this time, based on hard evidence.

There is wide variation from unit to unit in both the workload and the types of conditions treated. For instance, the smallest chamber facility reporting is that in Broome, the home of the Australian sponge-diving industry, which only treated a single case of decompression sickness, compared to the busiest unit, which provided over 4,000 HBOT to 217 patients during 2009-2010. A unit in one major city was responsible for over 80% of all ventilator-dependent patient treatments in the region, as unlike most other units, they have a strong commitment to HBOT in major trauma. There are also differences in the indications used for HBOT, e.g., some centres do not treat acute carbon monoxide poisoning at all whilst others do so.

These differences reflect local medical bias, variations in clinical capability and facilities and the overall lack of high-level evidence medicine to support many aspects of hyperbaric care. Whilst efforts to correct the latter are in progress; e.g., the Australasian multicentre wound care study, individual chamber support for one or two arms of the Hortis trial and a few other clinical trials either in progress or at inception, nevertheless we still have a long way to go before HBOT becomes a routine part of mainstream Western medical thinking. Commitment to such a long-term goal is patchy, with outstanding contributions from some centres and what appears largely to be indifference in others, though I suspect the latter often can be attributed to the limited funding provided for HBOT. Having said that, considerable investment in HBOT has been made recently, or is in process, by health administrations in Queensland, New South Wales, Victoria and Tasmania.

The HTNA, and Sue Thurston in particular, are to be congratulated in making this effort. Such a nation-wide set of data may be unique and is a potentially valuable tool in forward planning of hyperbaric services in Australasia. Whilst some of the data have been incomplete and inaccurate over the years, this has steadily improved with time and now provides a reasonably accurate picture of hyperbaric medicine in the region, perhaps to better than

Table 1
Summary of treatment data from 14 hyperbaric units in Australia and New Zealand, July 2009 – June 2010; only five of the main indications for HBOT are shown

Patients treated	1,173	
Acute	265	
Elective	908	
Number of HBOT	26,352	
Number of chamber runs	10,379	
Diagnosis		
Bubble injury (iatrogenic)	162	(6)
Carbon monoxide poisoning	51	
Necrotising infections	34	
Post-radiation problems (prophylactic)	408	(103)
Problem wounds (diabetic ulcer)	372	(146)

+/-2% reliability. All contributing hyperbaric centres are encouraged to be as accurate and meticulous as possible in providing their returns for future years. What these reports lack at present are patient outcome data, but this is being addressed by the Australian and New Zealand Hyperbaric Medicine Group which is developing a database to which it is hoped all units ultimately will contribute.

Unlike the themed nature of the last issue, this month we provide a variety of short papers which should provide something of interest for everyone amongst our diverse readership. In diving medicine, this ranges from small animal research, through gas monitoring in rebreathers, antioxidants to modify decompression stress and decompression profiles in recreational divers, to the impact on the sharpened Romberg test of being at sea for several days. The report on malignant otitis externa is the largest case series using HBOT yet published, and is accompanied (below) by a brief editorial. The case reports on HBOT in neonatal asphyxia and in the uncommon but highly lethal poisoning with hydrogen sulphide make interesting reading.

The journal's editorial office is now almost back to normal following the Christchurch earthquake, though, ten weeks after the main event, we are still receiving aftershocks up to magnitude 5 on the Richter scale, producing further damage in the Canterbury region. I apologise for not having yet found the time to be online on the journal website for the increasing number of people who have logged on in recent weeks. We wish everyone a safe and healthy 2011.

Michael Davis

Guest editorial

Malignant otitis externa: experience with hyperbaric oxygen therapy

Christian Heiden

With the low incidence of malignant otitis externa (MOE) and its variable manifestations, it is not possible to perform relevant prospective controlled studies in this condition. Therefore, the best evidence on which to base therapeutic decisions is likely to remain retrospective reviews of case series. Even though our knowledge in regard to MOE is not greatly increased by the retrospective case series by Saxby et al in this issue, nevertheless it has value.[1] The larger number of cases here confirms that multimodal therapy, with the inclusion of hyperbaric oxygen therapy (HBOT), enables a reduction in mortality compared to earlier case reports without HBOT. This case series is the largest in the literature. The real value of HBOT for MOE remains unclear, in part because in historical reports, patients were probably treated with less efficient antibiotics and surgical procedures than nowadays.

It would have been interesting to know whether the fatal cases described here had predominantly intracranial propagation of the infection, because a particularly bad prognosis is expected in these patients even without facial nerve palsy. Cures in this high-risk subgroup would have also been worth mentioning. The number of surgical debridements required was noticeably low in this group of patients. One must bear in mind that necrotic tissue cannot be revitalised by HBOT and generally hinders cure. This syndrome of 'necrotizing' infection carries the suffix 'malignant' with good reason, given the high mortality rates reported in the literature.

In summary, multimodal therapy is to be recommended for malignant otitis externa. There is no evidence to justify omitting any of the components of the treatment complex, including HBOT; there is currently no high level evidence-based medicine, just sound common sense, for any of the applied measures.

Reference

1 Saxby A, Barakate M, Kertesz T, James J, Bennett M. Malignant otitis externa: experience with hyperbaric oxygen therapy. *Diving and Hyperbaric Medicine*. 2010;40(4):195-200.

Christian Heiden, MD, PhD, is a consultant in ENT, head & neck and facial plastic surgery at the Klinikum Traunstein, a teaching hospital of the Ludwig-Maximilians University in Munich, Germany.
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Front page photos of an Antarctic 'Christmas tree' and 'fairy':
The feather star *Promachocrinus kerguelensis* on *Perkinsiana* was taken by Dr Neal Pollock.
***Clione limacina*, a shell-less pteropod, found in both Arctic and Antarctic waters, was taken by Dr Martin Sayer.**

The President's page

Peter Germonpré
President, EUBS

Dear friends,

Everyone's life consists of successes and failures. For each accomplishment, other projects fail or remain unfinished. This is so evident, you might say, why make this the opening sentence of a President's address?

The European Underwater and Baromedical Society has just held a very successful Annual Scientific Meeting. Although difficult to please 100% of our membership, our general impression is that most, if not all attendees were positive in their evaluation. There were some points of criticism, of course, and these will be addressed for the next meetings (we have already discussed these items with the upcoming organisers). The extra-scientific activities – discussions during coffee and lunch breaks, dinners, etc – conveyed the impression that our Society retains a sense of 'community', rather than simply of 'concordance', which is obviously one of our most important goals.

On the downside, EUBS still struggles with some 'old sores', and whatever we seem to undertake, these remain painfully present. Our scientific footprint could be considered to be rather isolated to a few laboratories, low numbers of submissions to our Journal remain of constant concern, as our Editors will testify, and even the elaboration of patient registries to document the clinical work that we perform is lagging behind. The Discussion Forum on the EUBS website, a tool to discuss clinical, experimental and technical issues, remains painfully under utilised. Efforts to increase the 'evidence base' and the scientific volume in diving and clinical hyperbaric medicine have been undertaken repeatedly; to name one, the COST B14 action has had a positive impact on community awareness and has produced some very useful documents (see <www.echm.org>) but unfortunately has not had a major and lasting impact on the volume of multicentre clinical research. In many ways, our society is a 'living being', and its successes and concerns reflect those of each of us.

I have noticed that over the last few years these ups and downs of human life seem to have become more extreme. It is easy to see how the media (and more particularly the visual media; television, the internet) play a major role in causing a gradual evolution towards a black-and-white view of success and failure. If a news item is not spectacularly positive or negative, it has little or no journalistic value. Human drama is smeared out night after night, but if not spectacular enough, receives scant attention. It is rare, if not exceptional, to see someone pictured as being 'moderately happy, moderately successful, with the occasional downside, but generally coping reasonably well with life'. Especially amongst the youth of today, this polarisation is gaining more and more ground as the way things should be.

Do the media create expectations that are too high? Are we insidiously becoming afraid to undertake something because we fear we might not live up to expectations? Has the social reward for being good, but not exceptional, become too low? I propose to you that this is the case. Emotional thrills (both negative and positive) have become so important that they influence people's behaviour and actions, rather than resulting from them. Young people especially tend to remain passive unless they can excel at something.

Is excellence not something we should strive for? Do I plead here for plain, dull, emotion-free career and family plans? Not completely. Rather, I encourage you not to back off because of possible failure, not to remain inactive because there seems to be no way you can be 'the best" at something. Not everyone has the resources and an environment that makes research activities easy. Not everyone has the scientific background to design the ultimate experiment. Not everyone has time to spend on the management of a research laboratory, scientific journal or Society. Our Society, the EUBS, is there not only for the 'successful' but is a Society for all professionals in the underwater and hyperbaric fields.

Progress often happens in unspectacular ways. Very recently, a new scientific network has obtained a green light from the European Community 7th Framework Programme. Named "*Phypode*" (Physiopathology of Decompression), it aims at educating young researchers in the field of diving medicine, by coordinating research projects in laboratories throughout Europe, from Poland to Italy, and even extending to South Africa. Dr François Guerrero, EUBS member at the University of Brest, has worked tirelessly towards the success of *Phypode*'s introduction. The basic work undertaken by François is unrewarding in purely scientific (publications) terms, but will undoubtedly be widely recognised to have enhanced the scientific growth of our specialty.

As you read this, the year 2010 is drawing towards its end. May I wish you a peaceful, quiet, uneventful year's end, with little things to make you happy and small steps into a bright future!

Original articles

Acute antioxidant pre-treatment attenuates endothelial microparticle release after decompression

Bryna C R Chrismas, Adrian W Midgley, Lee Taylor, Rebecca V Vince, Gerard Laden and Leigh A Madden

Key words
Decompression, endothelium, antioxidants, treatment, diving research

Abstract
(Chrismas BCR, Midgley AW, Taylor L, Vince RV, Laden G, Madden LA. Acute antioxidant pre-treatment attenuates endothelial microparticle release after decompression. *Diving and Hyperbaric Medicine*. 2010;40(4):184-8.)
Purpose: The hyperbaric and hyperoxic effects of a dive have been demonstrated to elicit changes in oxidative stress, endothelial function and microparticle (MP) release. Endothelial MP, which are small membrane vesicles shed from the endothelium, have been suggested as a valid *in vivo* marker of endothelial function. Furthermore, recent research has shown an increase in CD105 MP post-dive to be associated with a decline in endothelial function. The aim of this study was to ascertain whether antioxidant (AOX) pre-treatment can attenuate increased CD105 MP release post-dive.
Methods: Five healthy, male, pressure-naive subjects completed two simulated dives (control and intervention) breathing compressed air to a depth of 18 metres' sea water for 80 min. For the intervention dive, all subjects received a commercially available AOX pill containing vitamins C and E, selenium and beta-carotene 2 h pre-dive. CD105 MP, total antioxidant capacity (TAC) and thiobarbituric reactive substances assay (TBARS) were determined pre-dive, at depth, immediately and 4 h post-dive.
Results: In the control dive, there was a significant increase in CD105 MP immediately post-dive when compared with at depth ($P < 0.001$) and pre-dive ($P = 0.039$) values. Antioxidant pre-treatment significantly attenuated this release of CD105 MP post-decompression ($P = 0.002$). There were no significant changes in TBARS or TAC.
Conclusion: These results may provide evidence of the potential use of AOX pre-treatment as an effective endothelial pre-conditioner for divers.

Introduction

Decompression illness (DCI) is an inherent risk with scuba diving. While diving with compressed air, nitrogen is taken up by the tissues in proportion to the depth and time spent underwater. During ascent to the surface some of this gas may be released from tissues in the form of bubbles.[1–2] The formation of these bubbles has been previously acknowledged within the literature as the major factor relating to the development of DCI.[3] However, the occurrence of bubbles may be a poor predictor of DCI as divers with no/low bubble scores have developed DCI, whilst divers with high bubble scores have remained asymptomatic.[4] Consequently, recent research has investigated other potential markers of DCI including, but not limited to endothelial function, oxidative stress and increased endothelial microparticle (MP) release.[5–9]

Elevations in reactive oxygen and/or nitrogen species production are thought to reduce nitric oxide bioavailability causing vasoconstriction and subsequently may result in the initiation and progression to endothelial dysfunction.[6] The endothelium is particularly sensitive to changes in oxidative stress.[10] Circulating endothelial MP, shed from the endothelium, have been postulated as a useful *in vivo* measure of endothelial state and a potential marker for DCI.[8,9] Any mechanisms by which the transition to a more oxidising environment can be attenuated may be beneficial in preventing endothelial dysfunction. To date, pre-dive pre-conditioning methods have included the use of hyperbaric oxygen breathing, physical activity, heat exposure and antioxidants (AOX).[11–16]

Endogenous AOX defences may be insufficient to cope during a scuba dive and may lack efficacy in preventing transition to a more oxidative state.[15] Therefore, exogenous AOX may be beneficial in supplementing endogenous AOX.[17] Vitamins C and E have been successfully implemented to maintain endothelial function within the clinical setting following cardiovascular surgery, as well as being used more generally to offer endothelial protection.[18,19] Moreover, exogenous AOX supplementation has been successfully implemented to reduce oxidative stress following simulated dives in rats.[20,21] The use of AOX as a pre-dive pre-conditioning strategy in reducing oxidative stress and/or endothelial dysfunction in humans has yielded contradictory results; however, markers of oxidative stress are often not reported.[15,22] Consequently the usefulness of pre-dive AOX treatment as a potential

endothelial pre-conditioner remains to be elucidated. Therefore, the aim of this study was to ascertain whether AOX pre-treatment can attenuate the increased CD105 MP expression observed post decompression, and the increased oxidative stress that may be associated with diving. CD105 MP was selected as a constitutively expressed endothelial membrane protein (endoglin) and as such is present on MP released from the endothelium.[8]

Methods

SUBJECTS

Five subjects with no prior diving experience (mean age 23 ± 6 years, height 183 ± 8 cm, weight 78 ± 12 kg), volunteered to take part in this study. All subjects were healthy, male non-smokers and recreationally active. They provided written informed consent, and completed a pre-exercise medical questionnaire. Subjects were requested to abstain from alcohol, high-fat food, caffeine, and unaccustomed, vigorous physical activity for 24 hours prior to study commencement, and compliance was monitored via a pre-study questionnaire. Ethical approval for the study was obtained in accordance with departmental and university ethical procedures and followed the principles outlined in the Declaration of Helsinki.

EXPERIMENTAL PROTOCOL

All subjects participated in a control dive (AIR) and an intervention dive (AOX) separated by two weeks. The two dives were identical except that the subjects took an AOX pill prior to one of the dives. Subjects reported to the laboratory at 0930 h and were transported to a hyperbaric chamber via minibus at 1030 h. The simulated dry dives, conducted in a 6-person multiplace chamber and commenced at 1200 h, were to a maximum depth equivalent to 18 metres' sea water (msw, 284 kPa). Compression time was five minutes, time at depth 60 min and ascent time 15 minutes, including two 5-minute stops at 6 msw and 3 msw.

BLOOD SAMPLING

Venous blood samples were obtained pre-dive, at depth, immediately post-dive and 4 h post-dive from an antecubital vein using a standard venipuncture technique with a 21-gauge needle. Preceding each blood collection (not including the 'at depth' sample), subjects rested supine for 10 minutes. Blood was collected into commercially available sodium citrate vacuette tubes (Vacuette®, Greiner, UK) and the first draw was discarded due to the possibility of endothelial damage from the venipuncture.

ANTIOXIDANT PRE-TREATMENT

Subjects consumed a standardised breakfast consisting of shredded wheat (30 g) and semi-skimmed milk (250 ml) and ingested an AOX pill 2 h before the dive (at 1000 h). The commercially available AOX pill (Super antioxidant formula capsules, Holland and Barrett, UK) contained vitamin C 500 mg, vitamin E 268 mg, selenium 50 µg and beta-carotene 15 mg.

MICROPARTICLE QUANTIFICATION

MP were quantified using a BDFACS Calibur flow cytometer (BDBiosciences, UK) and the gating strategy defined in accordance with a previous study.[8] Sodium citrate blood tubes were centrifuged (200 *g*, 10 min) and platelet-rich plasma aspirated and transferred to a microfuge tube and further centrifuged at 1500 *g* for 3 min to prepare platelet-poor plasma (PPP). Anti-CD105:FITC (4 µL, AbD Serotec), and negative control: FITC (4 AbD Serotec) were incubated in the dark with PPP (25 µL) for 30 min prior to addition of Caltag counting beads (25 µL) (Caltag Medsystems, UK) and 200 µL of filtered (0.22 µm) phosphate buffering solution. Samples were subsequently analysed using CellQuest software (BDBiosciences, UK). Forward scatter was set as a trigger determined by the scatter properties of megamix beads (Biocytex, France). MP were quantified as an absolute count per µL PPP in relation to counting beads according to the manufacturer's instructions.

MEASUREMENT OF LIPID PEROXIDATION

Venous blood was alloquated into potassium EDTA tubes (Vacuette®, Greiner, UK) mixed and then spun at 1500 *g* for 10 min. The resulting EDTA plasma was then removed and transferred into 1.5 ml polypropylene tubes and stored at -80^{O}C. Lipid peroxidation was analysed utilising a commercially available thiobarbituric acid reactive substances (TBARS) kit (TBARS Assay Kit, Zeptometrix, USA) according to the manufacturer's instructions. Absorbance was read at 530 nm. Results are expressed as MDA equivalents.

MEASUREMENT OF TOTAL ANTIOXIDANT CAPACITY

Total antioxidant capacity (TAC) was measured using a commercially available kit (TAC Assay Kit, BioVision, CA, USA) according to the manufacturer's instructions. The protein mask included in the kit was not utilised; therefore, measurements include enzyme activity as well as small AOX molecules. Results are expressed as trolox equivalents.

STATISTICAL ANALYSIS

Statistical analysis was completed using IBM SPSS Statistics 18 (SPSS Inc., Chicago, IL). No power analysis was performed. The changes in CD105 MP, fluorescence (FL), TBARS, and TAC across condition (control and AOX) and time (pre-dive, at depth, immediately post-dive and 4 h post-dive) were analysed using linear mixed models for repeated

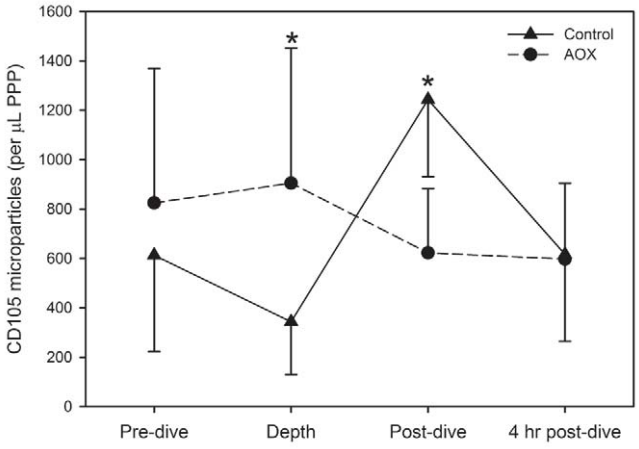
measures. Different covariance structures were assumed and the one that minimised the Hurvich and Tsai's criterion was chosen for the final model. Quantile-quantile plots showed the models for CD105 MP and TBARS exhibited right-skewed distributed residuals, which were corrected using natural log transformations of the observed data. Where a significant F ratio was observed, *post hoc* comparisons with Sidak-adjusted *P* values were used to identify which pairs of means were significantly different. Two-tailed statistical significance was accepted as $P < 0.05$.

Results

The simulated dives were both well tolerated, with no subjects reporting symptoms of DCI. The mean CD105 MP are shown in Figure 1. There were no significant main effects for condition (AIR or AOX, F = 0.2, $P = 0.66$) or time (F = 3.0, $P = 0.051$) for CD105 MP. However, there was a significant condition and time interaction (F = 6.8, $P = 0.002$). In the AIR (control) condition there was a significant increase in CD105 MP from pre-dive ($P = 0.039$) and at depth ($P < 0.001$) to immediately post-dive. The decrease in CD105 MP from immediately post-dive to 4 h post-dive did not reach statistical significance ($P = 0.058$). No significant changes across time were observed in the AOX condition ($P \geq 0.82$).

The mean FL, TBARS, and TAC for each time point in the two experimental conditions are shown in Table 1. No significant main effects for condition (F = 0.08, $P = 0.78$) or time (F = 2.1, $P = 0.13$), or an interaction effect (F = 0.4, $P = 0.77$), were observed for FL. Similarly, no significant main effects for condition (F = 2.5, $P = 0.16$) or time (F = 0.4, $P = 0.68$), or an interaction effect (F = 0.7, $P = 0.53$), were observed for TBARS. A main effect for condition for TAC was observed, where, on average, the TAC was higher in the control condition than in the AOX condition (F = 8.4, $P = 0.013$). A significant main effect also was observed for time (F = 4.6, $P = 0.039$), where TAC decreased from pre-dive to depth ($P = 0.036$), but no significant change was observed thereafter ($P = 0.44$). The interaction between condition and time was not statistically significant (F = 0.7, $P = 0.53$).

Figure 1
Mean (SEM) CD105 microparticle concentrations observed before, during and after simulated dives with antioxidant (AOX) and without (Control) pre-treatment; *n* = 5; * *P* < 0.05



Discussion

The techniques used in this study are robust. Flow cytometry is currently the only viable method for measuring microparticles, supported by a recent study.[23] The TAC and TBARS kits are commercially available kits that are certified and validated for use with human serum/plasma samples.

The main finding in the present study was that CD105 MP within the circulation were significantly lower post-dive ($P = 0.002$) in the AOX group compared to the control dive. A return to CD105 MP levels observed pre-dive was seen after 4 h, showing that the MP generated are removed within this time frame and, therefore, a return to a homeostatic condition was observed. Increases in both CD105 MP and CD106 MP have previously been observed following a dry chamber dive breathing air.[8,9] TAC was shown to be significantly decreased at depth when compared to both pre- and post-dive. This suggests AOX present within the circulation may have been utilised to deal with increased reactive oxygen species (ROS) at depth. The temporary reduction of TAC observed at depth

Table 1
Mean fluorescence intensity (MFI) of CD105 MP, thiobarbituric acid reactive substances (TBARS) and total antioxidant capacity (TAC) before, during and after a simulated dive in two groups (*n* = 5) with (AOX) or without (Control) antioxidant pre-treatment (see text for statistical analysis)

		Pre-dive		At depth		Immediately post-dive	
CD105 MP MFI	Control	33.2	(9.0)	31.7	(6.9)	36.3	(9.8)
(arbitrary units)	AOX	36.0	(4.3)	28.5	(11.0)	33.6	(4.7)
TBARS (MDA equivalents)	Control	17.3	(10.6)	25.0	(11.4)	31.4	(15.7)
(nmol mL^{-1})	AOX	25.6	(22.2)	17.5	(6.1)	18.8	(10.0)
Total antioxidant capacity	Control	30.1	(2.0)	28.1	(1.9)	30.7	(4.0)
(mmol mL^{-1})	AOX	27.7	(2.8)	25.4	(2.4)	27.0	(5.2)

may have a role within our previous hypothesis, in which it was suggested that there may be temporary endothelial dysfunction.[23] Although TBARS appears to increase at depth and post-dive, an effect that may be countered by exogenous AOX, these changes were not statistically significant, most likely due to the small number of subjects and low power of the study.

Previous, yet similar, work found that the increase in forearm vascular resistance (28 ± 10%) usually associated with hyperoxia was significantly attenuated following vitamin C administration.[24] Additionally acute endothelial dysfunction can be attenuated following both acute and chronic AOX pre-treatment, suggesting that AOX may have a prophylactic effect on endothelial dysfunction after diving.[15,16] Notably during the chronic protocol, the last dose of AOX was administered 3–4 h prior to the dive; therefore, the observed effect may have been an acute effect of the AOX and not a result of chronic treatment. Acute pre-treatment with AOX has been suggested to be more beneficial than chronic treatment.[25,26] In light of these findings, AOX pre-treatment was administered 2 h pre-dive in the present study and significantly attenuated subsequent CD105 MP release compared to the control dive. Furthermore, recent work within our laboratory has demonstrated a significant increase in CD105 MP post-dive, which was associated with a decline in endothelial function.[8] Hyperoxia-induced ROS production rapidly inactivates nitric oxide production, potentially causing vasoconstriction and ultimately contributing to the initiation and progression to endothelial dysfunction.[17]

The present study failed to demonstrate any significant changes in oxidative stress. Furthermore, chronic (four weeks) AOX pre-treatment with vitamins C and E has also failed to prevent hyperoxia-induced oxidative stress following a 2 h hyperbaric, hyperoxic exposure.[22] These results may suggest that hyperbaric hyperoxia is insufficient to cause any significant changes in oxidative stress within the circulation. However, it is more likely that other mechanisms, such as gas-bubble formation, are responsible for endothelial dysfunction and increased MP release following a dive.[9] Although gas bubbles *per se* are perhaps not the sole mechanism responsible for the development of DCI, *de novo* formation of bubbles may cause vascular endothelial cell stripping through mechanical interaction with the endothelium, and consequently cause an increase in MP release.[8,23,27]

In conclusion, a single dose of AOX 2 h prior to a dry chamber dive attenuated the release of CD105 MP from the endothelium. The mechanism(s) responsible for this endothelial protection remain to be elucidated and further research is required in order to ascertain whether AOX pre-treatment is an effective endothelial pre-conditioner for divers.

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'Sea legs': sharpened Romberg test after three days on a live-aboard dive boat

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Key words
Ear barotrauma, decompression illness, decompression sickness, sharpened Romberg test, motion sickness, postural control

Abstract
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Introduction: The sharpened Romberg test (SRT) is commonly used by diving and hyperbaric physicians as an indicator of neurological decompression illness (DCI). People who spend a prolonged time on a boat at sea experience impairment in their balance on returning to shore, a condition known as *mal de debarquement* ('sea legs'). This conditioning of the vestibular system to the rocking motion of a boat at sea may impact on the utility of the SRT in assessing a diver with potential DCI after a live-aboard dive trip.
Aim: To assess the impact 'sea legs' has on the SRT after three days on a live-aboard dive trip.
Methods: Thirty-nine staff and passengers of a three-day, live-aboard dive trip performed a SRT before and after their journey, with assessment of potential variables, including middle ear barotrauma, alcohol consumption, sea-sickness and occult DCI.
Results: There was no statistically significant impact on SRT performance, with 100% completion pre-trip and 35 out of 36 divers (97.2%) post-trip. There were trends towards more attempts being required and time needed for successful SRT post-trip, but these were not statistically significant. There was a small, but noteworthy incidence of middle-ear barotrauma, with seven people affected pre-trip, and 13 post-trip. There was a higher incidence in student divers. Middle-ear barotrauma did not appear to have a direct impact on SRT performance.
Conclusion: There was no significant impact on SRT performance resulting from 'sea legs' after three days at sea. Recreational divers, especially dive students, have a substantial incidence of mild middle ear barotrauma.

Introduction

The sharpened Romberg test (SRT) is commonly used by physicians as an indicator of neurological decompression illness (DCI).[1,2] People who spend a prolonged time on a boat at sea often experience impairment in their balance on returning to shore, a disorder known as *mal de debarquement* or 'sea legs'. This conditioning to the rocking motion of a boat at sea may impact on the utility of the SRT in assessing a diver with potential DCI after a live-aboard dive trip.

SHARPENED ROMBERG TEST

The Romberg test (RT), from which the SRT is derived, was first described by Dr Moritz Romberg in 1846 as an indicator of tabes dorsalis in patients with neurosyphilis.[3] This reflected the loss of lower limb proprioception through destruction of the dorsal columns of the spinal cord. Modifications of the RT were first described by Edmond Barbey in 1944, with Alfred Fregly introducing the 'sharpened' RT in 1966 with his research into vestibular disease. 'Sharpening' of the RT permits assessment of the cerebellum and vestibular apparatus, as well as lower limb proprioception.[4–7] Its use has since expanded, and the SRT was introduced into diving medicine by Dr Carl Edmonds in 1974.[5]

In hyperbaric and diving medicine, the SRT is commonly used in the objective assessment of neurological DCI, and as a marker of recovery following treatment with hyperbaric oxygen.[2,5,8,9] Research has shown the SRT has a sensitivity between 46–49% and a specificity of around 95% for DCI, and is "*a useful marker of decompression illness*", especially in "*a patient where the disease process was in question*".[5,9] Additionally, dive medicals in Australia and Europe include a baseline assessment of the SRT.[7,10–12] Known independent confounders of the SRT include alcohol intoxication, advancing age, and female gender.[4,6,13–15] Any 'learning effect' present is minimal and generally not clinically relevant.[5,6,16]

MAL DE DEBARQUEMENT

Mal de debarquement, or 'sea legs', is the sensation of a persistent rocking or swaying motion, with associated unsteadiness and dysequilibrium, experienced by people who spend time on a boat at sea. It occurs upon returning to shore and is usually transient, lasting hours to days, and resolving spontaneously.[17–20] The symptoms of 'sea legs' are believed to be secondary to central nervous system adaptation to the rocking motion of a boat at sea, but can also occur after rail, air and car travel.[18,19] It is unclear how long it takes for 'sea legs' to develop, with as little as five hours at sea producing symptoms.[17] However, most reports of 'sea legs' document it occuring after a prolonged time

at sea.[17–20] Nearly everyone adapts to the rocking motion of a boat within two to three days, as seen in the resolution of sea sickness.[2]

Single-day dive-boat trips are common, but the SRT after such trips is not affected.[5] Live-aboard trips permit divers to stay out at dive sites for a number of days, commonly 2–7 days, but increases the likelihood and severity of 'sea legs'.[1] This might, in turn, independently affect SRT performance. Our study aimed to determine whether 'sea legs' experienced by divers and snorkellers spending three days on a live-aboard dive boat results in compromise of their SRT. The null hypothesis was that spending three days on a live-aboard dive boat would not impair subjects' SRT performance.

Methods

Ethics approval for this study was granted by the Human Research Ethics Committee of Townsville Health Service District. Thirty-nine people participating in a three-day live-aboard dive trip, including five crew members and two of the researchers (CG and KC), were enrolled into the study, and underwent a SRT prior to boarding, and within one hour of disembarking. Seventeen participants were completing their Professional Association of Diving Instructors (PADI) 'Open Water Diver' certificate at the time of the study, and had finished two days of pool work prior to their initial assessments. All participants were given a study information sheet and informed consent was obtained. All collected data were de-identified and recorded onto a pre-formatted worksheet. This information was, in turn, entered onto a Microsoft Excel spreadsheet, and analysed using SPSS Version 17.0.

The SRT was performed on a flat, hard surface, barefoot or in hard-soled shoes, with one foot in front of the other. Subjects could decide for themselves which foot they placed in front. Arms were then crossed in front of the body, with the palms rested on the contralateral shoulder. Once settled, the subject was asked to close their eyes and keep their balance for 60 seconds (Figure 1). Each attempt was timed using a stop watch, which was stopped if the subject opened their eyes, shifted their feet, moved their arms, or fell over. The time of each attempt was recorded, and subjects were given four attempts to reach 60 seconds, adhering to Fregly's original description of the test and allowing comparison to other SRT research.[4,5,9] If they remained steady for 60 seconds, subsequent attempts were not required.

Subjects also underwent otoscopic examination of both tympanic membranes using a Welch Allyn Diagnostic Otoscope before and after the trip, with any signs of middle-ear barotrauma (MEBT) graded by two researchers (CG and KC) using the Edmonds classification system:[2]
Grade 0 – Symptoms without signs
Grade I – Injection of the tympanic membrane (TM)
Grade II – Injection, plus slight haemorrhage in the TM

Figure 1
The stance to be adopted by a subject performing the sharpened Romberg test[4,5,9]

Grade III – Gross haemorrhage within the TM
Grade IV – Free blood in the middle ear
Grade V – Perforation of the TM.

Signs of otitis externa were also documented. A digital image was kept of the post-trip findings, recorded with a Welch Allyn Digital MacroView Otoscope (Welch Allyn Australia, Radylmere NSW), to enable external validation by hyperbaric physicians blinded to the subjects' SRT results.

Basic demographic data were collected on each participant, as well as the use of any medications and incidences of sea-sickness. The dive profiles (maximum depth, underwater time and surface interval) of each participant were recorded by obtaining copies of the dive logs completed by the dive supervisors. Notably, there were two non-divers on the trip, who snorkelled a number of times. A record of alcoholic drinks consumed by passengers and crew was to be noted, but, because of changes in the company's alcohol licence, this was not possible. Timing of the SRT assessments ensured no subject was intoxicated whilst being tested.

STATISTICAL ANALYSIS

Our *a priori* plan for primary analysis was to use McNemar's Chi-square to evaluate the proportion of subjects passing SRT pre- and post-live aboard while accounting for the paired nature of the data, as well as comparing the exact 95% confidence intervals. The number of attempts required to achieve success pre- and post-live aboard were compared using the Wilcoxon Signed Rank Test, and differences in the cumulative time score for the SRT were compared using a

paired t-test. The cumulative time score is calculated from the sum of the times of the four attempts, with a maximum score of 240 seconds. If a subject maintains the SRT stance for 60 seconds, subsequent attempts are allocated 60 seconds and not performed. For all analyses, an alpha value of 0.05 was used to establish statistical significance.

Results

SRT

All 39 people on the boat were enrolled in the study; 22 (56.4%) male and 17 (43.6%) female. Ages ranged from 18 to 55 years, with a mean age of 26.4 +/- 7.1 years. The participants in the study were demographically similar to divers treated at The Townsville Hospital Hyperbaric Medicine Unit in the past three years (n = 38; mean age 31 +/- 8.7 years, range 14–52, 54.5% male).

All 39 subjects successfully completed the pre-trip SRT; 30 on the first attempt, six on the second attempt, two on the third attempt and one on the fourth attempt. The median number of attempts required to achieve success was 1 (interquartile range: 1 – 1), and the average cumulative time score was 226.5 +/- 10.0 seconds.

Two subjects departed immediately after returning to shore and were thus lost to follow-up. One participant declined to perform subsequent attempts after not obtaining 60 seconds on their first post-trip SRT attempt and, therefore, was also considered as lost to follow-up. Of the 36 subjects completing the post-trip assessment, 35 (97.2%; 95% CI 85.5% to 99.9%) were successful; 25 on the first attempt, five on the second attempt, three on the third attempt and two on the fourth attempt. The median number of attempts required to achieve success was 1 (IQR: 1 – 2), with an average cumulative time score of 217 ± 14.0 seconds.

The absence of any failures at baseline precluded analysis using McNemar's Chi-square; the confidence intervals around the proportions for successful SRT pre- and post-trip, however, do overlap. This remains true even when the three subjects lost to follow-up are allocated as 'failures' (35/39 = 89.7%; 95% CI 75.8, 97.1).

There was no significant difference in the number of attempts required to achieve success pre-trip versus post-trip (Wilcoxon Signed Rank Test, P = 0.563). When conservatively allocating the one subject who failed SRT and the three subjects lost to follow-up as requiring five attempts, there remained no significant difference, although a trend towards more attempts in post-trip SRT became more apparent (Wilcoxon Signed Rank Test, P = 0.064) (Figure 2). This general trend of poorer performance is also seen in the cumulative time score, with a difference of 9.4 seconds, but again this was not of statistical significance (P = 0.358) (Figure 3). Removing data for the two subjects who only snorkelled had no impact on the results.

OTOSCOPY

Seven of the 17 'Open Water' students had otoscopic

Figure 2
Pre- and post-trip cumulative percentage of successful SRT performance with each attempt (P = 0.563); allocating those lost to follow-up as requiring five attempts post-trip, the difference was not statistically significant (P = 0.064)

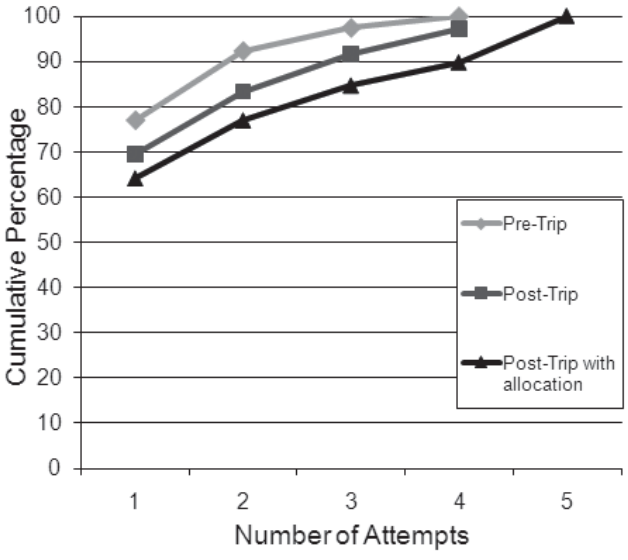


Figure 3
The 'cumulative time score' for SRT performance pre- and post-trip with 95% confidence intervals; difference 9.4 seconds (P = 0.358)

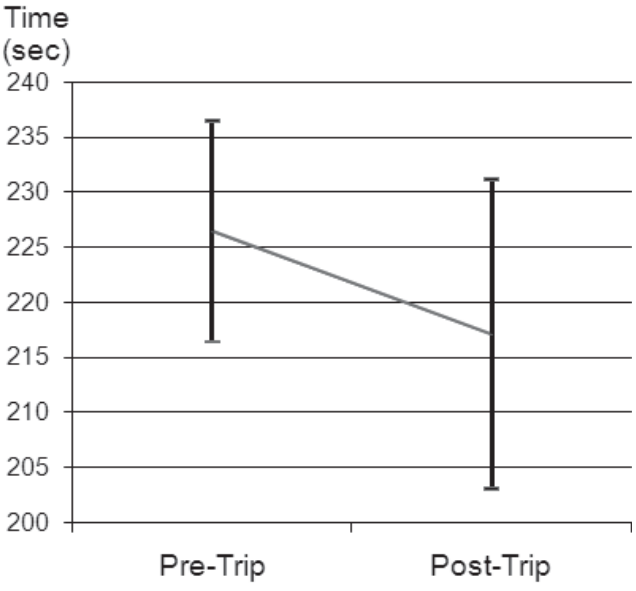
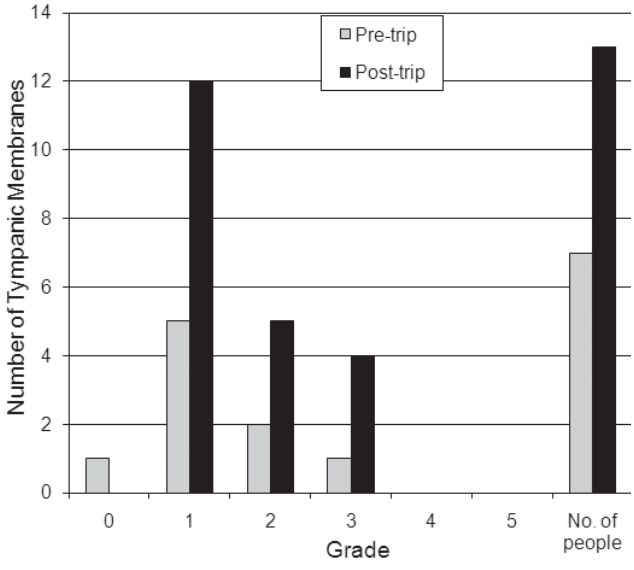


Figure 4
The incidence and grade of middle-ear barotrauma pre- and post-trip, graded using the Edmond's classification system[2]



evidence of MEBT prior to the live-aboard trip (41.2%), resulting from pool training dives. Most had Grade I changes (n = 5), with the worst being Grade III (n = 1), affecting nine tympanic membranes. None of the certified divers (n = 22) had MEBT prior to the trip, giving an overall pre-trip MEBT incidence of 17.9%. After completion of the trip, thirteen participants (33%) had MEBT, a significant increase (P = 0.002), with 21 tympanic membranes affected. Of the 32 with no MEBT prior to the trip, seven had developed MEBT (21.9%). Again, most only had Grade I changes (n = 12), but two participants had Grade III changes involving both tympanic membranes (Figure 4).

MEBT was recorded as an indicator of possible inner ear barotrauma, a known confounder of SRT performance. Despite a statistically significant increase in the incidence of MEBT after the trip, there appeared to be no direct link between it and SRT performance. The one subject who failed the post-trip SRT assessment had bilateral Grade II MEBT, but the two subjects with more severe injuries (bilateral Grade III) passed their post-trip SRT on the first attempt. Also, the two subjects who required four attempts to pass the post-trip SRT (i.e., 'nearly failed') had normal middle ear otoscopy. As any MEBT present did not affect the SRT, the barotrauma grades were not externally validated.

DIVE PROFILES

It was not possible to correlate subjects' dive profiles with SRT performance because we did not have access to subjects' dive computers. Depth and time information was available; however, the divers performed multi-level dives. When compared to DCIEM and PADI tables, some of the divers may have missed recommended decompression stops; however, without the raw data from computers we were unable to undertake any meaningful analysis. All divers, except one who only made one attempt, passed the SRT after the trip, and no divers complained of symptoms that were consistent with DCI.

SEA CONDITIONS

Wind and sea conditions were regularly estimated by the company's dive supervisor and recorded on the dive logs. Wind speeds ranged from calm to 15–20 knots, with most records (9 of 11) noting speeds over 10 knots. Sea conditions ranged from 'calm' to 'choppy' over the trip, with it being 'choppy' most of the time (7 of 11 records).

Discussion

The SRT is routinely used by physicians in the assessment of divers with potential neurological DCI, and in monitoring the response to treatment. The persistent swaying and rocking sensation of 'sea legs' could be expected to produce an abnormal SRT. Lee found a half-day, open-water boat trip had no effect on SRT performance.[5] Our aim was to evaluate whether 'sea legs' produced by a longer period at sea would affect SRT performance. The 39 subjects spent over 60 hours on a boat in open waters. All 39 successfully completed the SRT prior to the trip, and 35 of 36 subjects (97.2%) successfully performed the post-trip SRT, three subjects being lost to follow-up. This difference in pre- and post-trip SRT success rates was not statistically significant, even if those lost to follow-up were labelled as having failed. In an attempt to unmask subtle changes in SRT performance, we examined the number attempts required and the cumulative time score. Neither of these measures showed a statistically significant difference in performance.

Only two previous studies into the utility of the SRT in diving medicine have been published. The first paper prospectively assessed SRT performance in 60 control subjects, and retrospectively reviewed 35 cases of DCI.[9] Nearly half of the divers with DCI had a markedly abnormal SRT performance (less than 30 seconds). The rest had a near-normal performance, with times comparable to the control group. The majority of those with an abnormal SRT prior to treatment had normal SRT performance following recompression therapy. Fitzgerald deduced "*the SRT could be used as a 'marker' for DCI*".[9]

In a similar study, SRT performance was assessed prospectively in a control group, and retrospectively in 50 cases of DCI.[5] This showed that a single-day boat-diving trip did not adversely impact on SRT performance, and that roughly half of the divers with DCI had a markedly abnormal SRT performance (less than 35 seconds). All of the DCI cases improved to a normal SRT after treatment, supporting

the earlier findings. Lee concluded that "*the sharpened Romberg test is a useful marker of decompression illness*" and is "*resistant to several potentially confounding factors"* such as *"post-dive fatigue, decompression stress, vestibular disturbance resulting from the swaying motion of a dive boat and improvements due to practice or learning effect*".[5]

Our study looked exclusively at what Lee called *"vestibular disturbance"*, more commonly known as 'sea legs'. We hypothesised that spending three days at sea on a boat would amplify the development of sea legs, resulting in SRT failure. Our hypothesis was disproved; the SRT was not affected.

'Sea legs' is a diagnosis of exclusion, with no single diagnostic test. There is little consensus about the underlying pathogenesis of the condition, with traditional theories generally revolving around the concept of neuroplasticity of the vestibular apparatus. However, published reports of 'sea legs' commonly document normal examination, specifically with normal or equivocal vestibular testing.[17,18,20] Newer theories still involve the vestibular apparatus, but focus on neuroplasticity of higher centres in the cerebral cortex and hippocampus. These may then provide ongoing stimulation of the vestibular apparatus, and generate the ongoing sensation of swaying.[17,18,20]

The SRT assesses the cerebellum, the vestibular apparatus and lower limb proprioception. The failure of our hypothesis supports earlier research into 'sea legs' and the lack of evidence of abnormal vestibular function. If the pathogenesis of 'sea legs' truly lies 'higher' than the vestibular apparatus, then the SRT would not be affected, as seen with our results.

We attempted to account for, and record the presence of, potential confounders of SRT performance, including alcohol intoxication, provocative dives, and inner ear barotrauma (IEBT). None of the subjects were under the influence of alcohol at the time of the SRT assessment, controlled by timing of the testing. It was not possible to draw any conclusions from the dive profiles because computer dive records were not available.

The mechanism between IEBT and poor SRT performance is clear and logical, but there is no published research examining a direct correlation between MEBT per se and the SRT. Analysis of our limited data did not reveal any obvious link between MEBT and SRT performance, but further directed research into this should be performed before a solid conclusion can be made.

Our study has a number of limitations. Firstly, the sample size was constrained by the capacity of the dive boat; however a *post hoc* power analysis reveals the study to be adequately powered to statistically detect small differences in the SRT success rate. With either 36 or 39 subjects, the power to detect a decrease in SRT success from 99.9% to 96.0% exceeded 0.80. Thus, it is unlikely that this study failed to identify any clinically meaningful change in SRT performance.

Secondly, our hypothesis was based on the premise that spending three days at sea would result in the subject developing 'sea legs'. Being a diagnosis of exclusion, we were reliant on subjective descriptions of symptoms consistent with 'sea legs'. The majority of subjects stated they felt unsteady during their post-trip SRT, "*like* [they were] *back on the boat*", and almost certainly had developed 'sea legs'. 'Sea legs' severity is likely to increase with even more prolonged durations at sea and/or more severe sea conditions, and may still influence SRT performance. Correlation with varying sea conditions may also be of importance.

Conclusions

The effects of 'sea legs' on people spending three days on a live-aboard dive trip does not impair their ability to perform a SRT. Therefore, in a patient with suspected DCI, who has spent some days at sea, SRT failure should not be attributed to 'sea legs' and should be considered a confirmatory marker of DCI.

MEBT was common. Further studies of the SRT in divers who remain at sea for many days is warranted, and any correlation between MEBT and SRT performance should be examined.

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This paper is based on a dissertation submitted by Dr Gibbs towards the SPUMS Diploma in Diving and Hyperbaric Medicine.

Malignant otitis externa: experience with hyperbaric oxygen therapy

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Key words
Hyperbaric oxygen therapy, malignant otitis externa, ENT, clinical audit, outcome

Abstract
(Saxby A, Barakate M, Kertesz T, James J, Bennett M. Malignant otitis externa: experience with hyperbaric oxygen therapy. *Diving and Hyperbaric Medicine*. 2010;40(4):195-200.)
Introduction: The treatment of malignant otitis externa (MOE) with hyperbaric oxygen therapy (HBOT) remains controversial. The rarity of MOE, combined with poor access to hyperbaric facilities, explains the paucity of existing data.
Methods: We retrospectively reviewed all patients with a diagnosis of MOE referred to the Prince of Wales Hospital hyperbaric unit over a period of six years, and report one of the largest case series to date.
Results: From August 2001 to October 2007, 17 patients with MOE were referred, of whom 15 (88%) completed therapy, one did not tolerate HBOT and one was withdrawn due to pulmonary complications. Length of admission averaged 48 days (range 8–93 days) and three received outpatient care. Five patients had complications attributable to HBOT: acute pulmonary oedema (n = 2), seizure (n = 1), tympanic membrane perforation (n = 1) and claustrophobia (n = 1). Average time to follow up was 47 months (range 1–94 months). Twelve patients (70%) were considered cured of their disease, being disease-free at follow up, including four patients who had died of other causes but were symptom-free at the time of death. Three patients died directly from MOE (18%), one after a recurrence of their disease. Two further patients had recurrent disease, both successfully treated with a second cycle of HBOT and antibiotics. Nine patients (53%) had facial nerve palsy before commencement of HBOT, of whom four died, three from MOE, four had ongoing facial paralysis, and one resolved.
Conclusions: HBOT confers minimal morbidity, but its role in MOE remains uncertain. The high mortality of MOE despite maximal therapeutic intervention highlights the need for more effective treatment protocols.

Introduction

Malignant otitis externa (MOE) refers to a severe infection involving both the external auditory canal and the surrounding skull base (temporal, occipital or sphenoid bones).[1] MOE usually arises secondary to inadequately treated chronic otitis externa, although it may also originate from chronic otitis media, sphenoid sinusitis or any inadequately treated infection in close proximity to the skull base.[1] Previous series have found the most common causative agent to be *Pseudomonas aeruginosa*.[2]

Afflicted patients generally tend to be elderly and have some form of systemic immunocompromise, most often diabetes mellitus, or a history of radiotherapy for a head and neck malignancy in the region of the skull base.[3] Patients frequently present with vague symptoms running a long and insidious course. More severe symptoms include unilateral severe otalgia of prolonged duration and out of proportion for routine external otitis, unremitting headache mainly over the temporal and parietal regions, recurrent and relapsing otorrhoea, hearing impairment, vertigo and a sensation of aural fullness.

Treatment protocols for MOE are controversial but usually include long periods of both intravenous and subsequent oral antibiotics with various adjuvant therapies including antifungal medications, surgical debridement and hyperbaric oxygen therapy (HBOT).[4–12] A typical protocol for HBOT is 90–100 minutes daily at between 203 and 243 kPa repeated 20 to 60 times.[8,12–14]

Since 2001 the department of Otolaryngology, Head and Neck Surgery at Prince of Wales Hospital in Sydney has routinely referred patients with established MOE for consideration of HBOT and this case series documents our experience to date.

Materials and methods

Following approval from the South East Sydney Health Area (Northern Network) Human Research Ethics Committee (ref: 07/07), we conducted a retrospective review of all patients who had received HBOT at the Prince of Wales Hospital Department of Diving and Hyperbaric Medicine (DDHM) to treat MOE. Long-term outcomes were determined by chart review, patient assessment and telephone interview.

We identified suitable cases by examination of the DDHM database (FileMaker Pro 5.1, FileMaker Inc, 1999) using 'acute necrotising infections', 'osteomyelitis', 'other infections' and 'audiovestibular' as non-exclusive search terms. Where insufficient information was available on the database, we retrieved the full medical record and conducted a telephone interview with the patient or a relative as appropriate. All facial nerve palsies were graded on the House Brackman system from II (slight) to VI (total).[15]

Table 1
Summary table of 17 patients, pathogens grown and treatment outcomes; MRSA – Methicillin-resistant *Staphylococcus aureus*; MOE – malignant otitis externa; HBOT – hyperbaric oxygen therapy; TM – tympanic membrane; o/p – out-patient

Patient	Age/sex	Diabetes	Cranial neuropathy	Pathogen	Admission (days)	HBOT (number)	HBOT problem	Outcome	Follow up (months)
1	75 M	Yes	No	*Bacteroides*	53	29		Ongoing otorrhoea, died of other disease	62
2	79 M	No	No	MRSA	32	30		Disease free, died of other disease	90
3	72 M	Yes	VII	*P. aeruginosa*, *Candida* spp	36	30		Disease free, ongoing VII palsy	51
4	81 M	No	No	Unspecified yeast	57	30		Disease free	37
5	66 M	Yes	VII	Negative cultures	59	40		Died of MOE (2 months)	2
6	65 F	Yes	VII	*P. aeruginosa*, *S. aureus*	71	37		Seizure, died of MOE (1 month)	1
7	84 M	Yes	VII	Negative cultures	17	5	Pulmonary oedema	Disease free, VII palsy resolved	72
8	78 M	Yes	XII	*P. aeruginosa*	45	30		Disease free, XII palsy resolved	94
9	65 M	Yes	No	Negative cultures	8	1	Claustrophobia	Otorrhoea and otalgia	80
10	71 F	No	No	Negative cultures	o/p	30		Disease free, died of other disease	26
11	75 F	No	No	*P. aeruginosa*	9+o/p	30		Disease free, died of other disease	38
12	84 M	Yes	VII	*P. aeruginosa*, *S. aureus*	43	29	Perforated TM	Recurrence (11 m), VII palsy, died of other disease	37
13	69 M	Yes	No	*S. epidermidis*, *S. apiospermum*	42	30		Disease free	43
14	76 M	Yes	VII	*P. aeruginosa*, *Candida spp*	o/p	30		Disease free, ongoing VII palsy	49
15	71 M	Yes	VII (X)	*P. aeruginosa*, *Candida* spp *S. apiospermum*	74	39		Recurrence (4 m), new X palsy, died of MOE	6
16	77 F	Yes	VII, IX, XII	*P. aeruginosa*	39	30	Pulmonary oedema	Disease free, ongoing VII palsy, IX & XII resolved	77
17	77 M	Yes	VII	MRSA, *Corynebacterium*, *Aspergillus flavus*	93	38		Recurrence (5 weeks), disease free, ongoing VII palsy	35

Results

Between August 2001 and October 2007, 17 patients were accepted for HBOT therapy for confirmed MOE (Table 1). The average age was 75 years (range 65–84 years), and there were 13 males. Eight cases involved the right ear alone, eight the left and one patient had bilateral disease. A complete course of HBOT was achieved in 15 of the 17 patients (88%). Those who underwent a complete course had between 29 and 40 treatment sessions, with a median of 34.

All patients received broad-spectrum and culture-directed intravenous or oral antibiotics under the direction of the infectious diseases department. The area was debrided surgically as clinically indicated or based on imaging evidence. All but two patients were treated as in-patients, depending on their physical ability. Diagnostic and progress imaging was undertaken as clinically necessary.

All patients were treated in a multiplace chamber once daily from Monday to Friday on a standard 90-minutes schedule at

243 kPa breathing 100% oxygen. All patients were initially scheduled to receive 30 sessions over six weeks, but this course could be shortened or extended depending on the clinical and/or imaging response.

The average duration of symptoms prior to presentation at POWH was 4 weeks (range 2–6 weeks). The most common symptom was otalgia (94%), followed by otorrhoea (75%), headache (50%), facial or jaw pain (19%) and tinnitus (13%). Examination revealed the typical findings associated with otitis externa, including purulent discharge in the external auditory canal and tenderness on manipulation of the pinna. Only two patients were febrile on admission. Nine patients (53%) had a facial nerve palsy on admission with documented grading of House Brackman IV in one case, V in another and 'abnormal' in the remainder. Involvement of other cranial nerves was rare. Two patients had hypoglossal neuropathy, one of whom had additional glossopharyngeal nerve involvement.

Thirteen patients (76%) had a previous diagnosis of type II diabetes mellitus, of whom three were insulin-dependent. The average HbA1C at the time of diagnosis of MOE was 7.0%. Other co-morbidities included ischaemic heart disease ($n = 7$), chronic renal failure ($n = 2$), alcoholic liver cirrhosis ($n = 1$) and one patient who had undergone successful renal transplantation and was immunosupressed. Three patients had a history of previous ear surgery temporally unrelated to the current infection; modified radical mastoidectomy in two and tympanoplasty in one. Eleven patients had a history of cigarette usage but only four were currently smoking. Four patients had significant regular alcohol intake.

INVESTIGATIONS

Imaging

All patients were investigated with a computed tomogram (CT) scan of the petrous temporal bones and Gallium 67 scintigraphy. Four patients had magnetic resonance imaging (MRI) of the area.

Microbiology

A pathogen was isolated in thirteen cases (76%). *Pseudomonas aeruginosa* was cultured in nine (56%), multiple pathogens in eight cases (47%) and fungal cultures were positive in six cases (35%). Table 1 details the microbiology.

TREATMENT

Three patients received HBOT as outpatients (two for the entire course, one after nine in-patient sessions). The remaining patients required admission either because of their health status or because they were geographically removed from their usual residence. Admission periods ranged from 8 to 93 days with an average of 48 days.

A variety of different antibiotic regimens were employed with varying time courses. The most common intravenous antibiotic was timentin, used in thirteen cases (76%). Systemic and ototopical ciprofloxacillin were also frequently prescribed. A systemic antifungal agent was added in four cases. On discharge, all patients received oral ciprofloxacillin, with or without ototopical drops, for an average of four months (range 1–9 months). In addition, culture-directed antibiotics were prescribed; oral flucloxacillin (two patients) and oral clindamycin, oral augmentin, oral voriconazole and IV timentin via a long line, each in a single patient.

In two cases, microscopic and diagnostic evaluation under general anesthesia with curette debridement of necrotic tissue in the external auditory canal was undertaken within the first few days of presentation. Histology demonstrated granulation tissue with necrosis and evidence of acute inflammation consistent with MOE. Two further patients underwent cortical mastoidectomy, one in conjunction with external ear canal debridement (prior to HBOT), and in the other, mastoidectomy was undertaken at the time of MOE recurrence (after HBOT).

ADVERSE EFFECTS

In addition to the cranial neuropathies described above, two patients had disease-related complications: sigmoid sinus thrombosis in one and temporomandibular joint septic arthritis requiring aspiration in another. One other patient developed a brachial vein thrombosis associated with antibiotic administration. No long-term sequelae from these complications ensued.

Complications that might be attributable to HBOT were encountered in five patients (29%): acute pulmonary oedema in two and seizure, tympanic membrane perforation and claustrophobia, each in a single patient. HBOT was stopped early in the course of two patients. One refused further sessions after the first, due to claustrophobia in the chamber, and the second was withdrawn due to worsening lung function following an episode of acute pulmonary oedema during the fifth HBOT on a background of chronic obstructive pulmonary disease. No long-term sequelae associated with these events occurred.

DISEASE PROGRESSION AND OUTCOME

Routine blood tests including white cell count (WCC), C-reactive protein (CRP) and erythrocyte sedimentation rate (ESR) were used in an attempt to monitor disease progression. WCC was almost entirely within the normal range throughout every patient's admission. A sustained moderate rise in CRP was found in most patients with little fluctuation as treatment progressed. ESR was more indicative of disease progression and trended down during the first month of HBOT in most patients.

All patients were followed up to June 2010. The average time of follow up was 47 months from diagnosis (range 1–94 months). Twelve patients (70%) were considered cured of their disease, being disease-free at follow up, including four patients who had died of other causes but were symptom-free for MOE at the time of death. Two patients died within two months of diagnosis despite a full course of HBOT and antibiotics and a third despite a second course of antibiotics and HBOT for a recurrence after four symptom-free months. Three patients had a recurrence of MOE; one died as outlined above. The second recurred at 11 months, and was successfully treated with a second course of antibiotics and HBOT. He died of an unrelated illness 26 months later, symptom-free for MOE. The third was readmitted only five weeks after discharge from hospital, with a new onset of facial nerve palsy. He went on to have a cortical mastoidectomy and commencement of systemic antifungal therapy after which he has remained disease free for 35 months.

Of the nine patients to have facial nerve involvement, four died, three from MOE, four had ongoing facial paralysis, and only one resolved. The one resolution occurred within one year after discharge. All lower cranial nerve neuropathies resolved during the time the patient was in hospital.

Discussion

The use of HBOT for MOE was first described by Mader in 1982 in a single case report.[9] Subsequently a number of case series have been reported (Table 2) but no randomised, controlled trials (RCTs) have been undertaken. Given the rarity of the disease and its variable presentation, this is not surprising.[12]

The treatment of MOE remains a formidable challenge despite improvements in antibiotic choice and increasing availability of new therapies such as HBOT. With the absence of RCTs studying the benefit of any therapeutic measures, including HBOT, we must rely on lower levels of evidence to guide the choice of therapy. At the present time with regard to HBOT, case series such as this are the best evidence we have upon which to base decisions. The rarity of MOE coupled with the relative paucity of available HBOT facilities makes this report an important summary of how MOE is treated in a tertiary referral centre with HBOT capabilities. The study size is one of the largest published (Table 2).[6–10,16–19]

Table 2
Published reports on hyperbaric oxygen therapy to treat malignant otitis externa; NR – not reported

Year	Author	Patients	Cure Rate
Case series			
1982	Lucente[17]	3	NR
1989	Shupak[7]	2	100 %
1992	Davis[6]	16	100 %
2004	Narozny[10]	8	88 %
2010	Present study	17	70 %
Case reports			
1982	Mader[9]	1	Cured
1990	Schweitzer[8]	1	Cured
1998	Bath[18]	1	Cured
2000	Lancaster[19]	1	Died
2005	Singh[16]	1	Cured

Pseudomonas remains the commonest pathogen, isolated in half our cases. However, multiple pathogens were cultured in a number of patients and nearly a third of patients had evidence of fungi present. Antibiotic choice should be culture-directed but, in the absence of positive cultures, a reasonable choice would be IV timentin combined with oral and topical ciprofloxacillin. An antifungal agent should be added if there are any features suggestive of fungal involvement.

The role of surgical debridement in the treatment of MOE remains controversial. A small proportion of patients in this series had surgical debridement of necrotic tissue. In terms of outcome, there is no clear evidence that debridement improves cure rate, cranial nerve palsy resolution or speed of recovery. In our series, both patients who underwent debridement were symptom-free at over a year of follow up with no recurrence, but neither had a resolution of their facial nerve palsy. Their length of stay was not significantly different from those who did not undergo surgery (37 versus 42 days respectively). With such small patient numbers, no solid conclusions can be drawn in regards to these findings. In our opinion, surgical debridement should be reserved for selected patients with disease progression despite maximal medical management and HBOT.

The mortality rate due to MOE in this series was 3/17 (18%). Previous case series involving the use of HBOT have reported mortality rates as low as zero and up to 12%.[6,11] On the other hand, Chandler described a rate of around a third in a series of 22 patients, only four of whom received HBOT.[2] Within our series, two-thirds of the patients were symptom-free at follow up. The part that HBOT played in such recoveries is hard to quantify. An argument against the use of HBOT would be that the one patient who withdrew from HBOT due to a complication made a full recovery including facial palsy resolution whilst three other patients succumbed to their disease despite a full HBOT course. In the group of patients with ongoing symptoms including otalgia and otorrhoea, and in the three patients who had recurrence, there were no obvious reasons for treatment failure. Their presentations and management protocols were not appreciably different from any of the disease-free group. The incidence of recurrence in the absence of HBOT with antibiotic therapy alone is reported to be in the order of 15–20%, so the finding of three cases in the 17 presented (18%) is within expectations.[16]

In this series the facial nerve was the most common cranial nerve to be involved, and was the least likely to resolve with treatment. Seven of nine patients presenting with a facial nerve palsy and who survived the disease had ongoing facial paralysis at long-term follow up (mean of 53 months in this subgroup). Involvement of the lower cranial nerves was rare. The journey of the facial nerve directly through the infected temporal bone as opposed to the paths of the lower cranial nerves which merely pass adjacent to the area affected by osteomyelitis is likely to be the explanation for this difference. Previous studies without HBOT have described a grave prognosis if lower cranial nerves are involved with mortality rates of 80–100%.[20] We did not find this, and the two patients with lower cranial neuropathies on their first presentation responded well to treatment, with complete resolution by the time of discharge from hospital. One patient who developed a tenth cranial neuropathy during a recurrence of his MOE did ultimately die. This suggests the development of lower cranial neuropathies later in the disease progression may be more significant than in the early stages of treatment.

The potential benefits of HBOT must be weighed against the associated complications. In our series almost one in three patients encountered some form of morbidity attributable to HBOT, which is contrary to the experience of previous authors.[6] Whilst the majority of problems, such as middle ear barotrauma, were relatively minor and did not preclude further therapy, a small proportion of patients did have significant problems with longer-term implications. Complications such as oxygen toxic seizures and acute pulmonary oedema are directly related to high intra-arterial oxygen tensions, and are well documented in the literature.[8,14] A recent review of 240 patients receiving a combined total of 4,638 HBOT sessions reported a complication rate of 20% of which 94% were 'mild to moderate'.[18] The incidence of serious complications including seizures and pulmonary oedema was 1.7% and there were no deaths.[21] In our series, one patient had to be withdrawn from further HBOT due to exacerbation of pre-existing chest disease. The other occurrence of pulmonary oedema was after the thirteenth treatment and resolved with diuretic therapy alone. The patient with HBOT-related seizures experienced these around 30 minutes into the first two sessions. Her blood sugars at the time of the seizures were recorded as 2.3 and 2.8 mmol L^{-1} respectively and resolved with injection of 50% glucose. Following a change to the protocol, incorporating two air breaks and an alteration to her insulin dose prior to commencing each session, she completed a full course of HBOT with no further complications.

It is interesting that, within the same institution, the treatment protocol for the 17 patients presented was different in every case, whether it be through antibiotic choice, length of treatment or number of HBOT sessions. Precise treatment protocols will always be tailored to specific patient need; however, a more unified approach to treating this disease is warranted. How to decide on such 'best practice' is more challenging. Access to hyperbaric facilities will always be relatively difficult, however, most countries have referral centres available and, given the rarity of this disorder, it seems appropriate that such cases are treated at a tertiary referral centre with a more unified approach.

Conclusion

Further research is required to fully understand the advantages, if any, HBOT offers patients with MOE. Our experience suggests HBOT, where available, is an appropriate adjunct to antibiotic regimens, with only minor associated morbidity. Despite this, a proportion of patients will succumb to their disease, which highlights the need to search for more effective treatment protocols.

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This case series was presented by Dr Saxby at the Australasian Society of Otolaryngology, Head and Neck Surgery 2008 Annual Scientific Conference, Perth, Australia.

Exceeding the limits - estimated tissue pressures among Western Australian recreational divers

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Key words
Scuba diving, recreational diving, decompression, models, diving tables, research

Abstract
(Buzzacott P, Pikora T, Heyworth J, Rosenberg M. Exceeding the limits - estimated tissue pressures among Western Australian recreational divers. *Diving and Hyperbaric Medicine*. 2010;40(4):201-5.)
Introduction: In Western Australia (WA), approximately 40 divers suffer decompression sickness per year, many after exceeding accepted safe time and depth limits.
Methods: Divers on organised recreational scuba dives wore depth/time loggers. Dives ('case' dives) exceeding the Diving Science and Technology gas-content limits (M-values) were matched to control dives made at the same dive site at the same time during which no M-values were exceeded. Potential risk factors for decompression sickness were evaluated using a conditional logistic regression model.
Results: A total of 1,032 organised recreational dives were recorded. Case dives ($n = 38$) were more likely made by females, deeper than other divers in the water at the same time. They were also made by divers less likely to have previously dived as deep.
Conclusions: One in 27 recreational dives studied exceeded an M-value during the dive, but none on surfacing. We recommend that dive organisers in WA continue to encourage recreational dive groups to watch their displayed remaining no-stop time and to dive within the limits of their training and experience. This study successfully utilised periodic depth/time dive profile analysis using freely available software.

Introduction

The first reported experimental attempt to reduce the incidence of decompression sickness (DCS) was conducted for the Royal Navy by physiologist JS Haldane.[1] Dive tables were developed based on a gas-content model, where theoretical compartments of varying (parallel) blood perfusion and inert gas solubility were defined by the respective times they would take to half-fill with nitrogen, known as 'half-times'.[2] In 1965, Workman, in developing tables for a wider range of diving exposures for the US Navy, changed the limits of tolerable decompression stress from supersaturation ratios to maximum pressure differentials, known as 'M-values'.[2,3] During ascent, divers are advised not to ascend above the depth at which any half-time compartment reaches its M-value. Once the pressure differential between tissue compartment and environment is reduced, the diver may recommence ascending. By definition, in recreational no-stop diving, a direct ascent to the surface may be made at any time during the dive without exceeding an M-value.[4]

Gas-content limited models based upon M-values have been validated for recreational no-stop dives to 40 metres' sea water (msw) depth with an ascent rate no greater than 18 m min^{-1}.[2,3,5–8] One of these in popular use in Western Australia (WA) is the Diving Science and Technology (DSAT) model, used by the Professional Association of Diving Instructors, responsible for the majority of diver certifications in WA, and incorporated into at least one brand of recreational dive computer. Since their release in 1988, millions of divers have made millions of dives using the DSAT tables.[9] The majority of recreational dives using the DSAT tables are probably made well within prescribed depth/time limits. After two decades, the DSAT model appears to adequately limit no-stop recreational divers to an acceptably low probability of DCS.[10] The model was adapted for dive computer use by reducing the M-values to account for real-time depth estimation and remaining 'no-stop' time calculation, and by an increasingly severe threshold level for reducing surface interval credit to account for multi-day, repetitive diving.[11]

Between 2000 and 2009 inclusive, a mean of 39 divers per year were treated for DCS at the Fremantle Hospital hyperbaric facility in WA (Sakar K, personal communication, 2010). As not all divers carry downloadable personal dive computers, it remains uncertain which injured divers have exceeded accepted, safe time and depth limits. Recreational dives made in WA can be divided into two groups; self-organised dives, where a small group or pair of divers select a site suited to their intended purpose (e.g., to catch crayfish), and organised group dives, where a dive supervisor selects the site to accommodate the range of abilities within a larger group (e.g., a dive club or charter boat). The present study examined organised group dives to determine which potential factors increase the risk of a dive exceeding safe depth-time limits.

Methods

Eighteen dive businesses or dive clubs were invited to participate in the study and to allow a researcher (PB) to

accompany their dives to collect data. One dive business declined to participate. The study was approved by the Human Research Ethics Committee of the University of Western Australia.

Between February 2008 and March 2009, the researcher met these organised dive groups at popular dive sites in WA and invited divers to participate prior to the commencement of 35 separate dive series. Divers were allowed to participate or decline on multiple occasions. Participants were provided an information sheet, asked to sign a consent form and completed the Diver's Alert Network Project Dive Exploration (PDE) diver data form. A Sensus Ultra data-logger (ReefNet, Ontario) was attached to the front of each participating diver's buoyancy control device and a dive record listing the site details, entry/exit times and cylinder pressures was completed for each dive group. The loggers had a pressure resolution to 1 mbar, with an accuracy equivalent to 30 cm change in depth in seawater. Sampling was at 10-second intervals. Starting and returning air pressure and cylinder volumes were recorded on the dive record. Post-dive recollections regarding warmth and workload were recorded on the PDE dive-data form as the diver exited the water. Lastly, 15 additional questions addressing potential risk factors were asked by the researcher. These recorded familiarity with the dive site and scuba unit worn, pre-dive depth/time planning, previous dives to the same depth and/or with the same dive buddy and personal dive computer use.

After each dive, data from the loggers were downloaded onto a laptop computer. Segmented dives, such as if a diver surfaced momentarily during a dive, were manually cross-checked to the dive record and joined into single dives where appropriate. Once data entry was completed, every twentieth dive ($n = 52$ dives, made by 47 divers) was physically checked for accuracy against the original paper data-collection forms. Of 3,380 items checked, 3,362 (99.5%) were found to have been entered and coded correctly.

To control for environmental conditions, dives in which a diver exceeded a DSAT M-value were classed as 'case' dives, and dives made at the same dive site and at the same time by another diver who did not exceed a DSAT M-value were classed as 'control' dives. Group dives where no diver exceeded an M-value were not considered for further analysis.

Analysis

Dive profile data were exported to the statistics program R and analysed using the package *SCUBA*.[12] For recreational no-stop diving, DSAT uses a 60-minute half-time as the off-gassing rate when estimating the penalty to be applied to no-stop limits for repetitive diving.[3] In this study, however, Henry's law and Fick's first law of diffusion were used to predict gas washout between repetitive dives, with no limiting minimum half time (Baddely A, personal communication, 2008). Theoretical tissue tensions for the original eight DSAT compartments, (5, 10, 20, 30, 40, 60, 80 and 120 minute half-times), were estimated and re-imported into the database. Both ending and maximum compartment pressures were compared with M-values for the DSAT model, using the same conversion factor used by the package *SCUBA* (1 bar = 10.00 msw = 32.646 fsw).[3,12]

Maximum rate of ascent per dive in m min^{-1} was generated by multiplying the largest negative difference in depth over a 10-second interval by six. Diver certification was categorised arbitrarily as low (<10 open-water dives), medium (10–20 dives) and high (≥20 dives).[13]

The data were imported into SAS version 9.1 (Cary, North Carolina) and the distribution of variables tested for normality. Bivariate analyses were conducted for each factor, variables with expected cell counts of less than five were excluded from further analysis. These included rare events such as cigarette use (1/38 cases), ascending faster than 18m min^{-1} (4/38 cases), reported panic (1/38 cases), not making a safety stop (3/38 cases), and not using a dive computer (2/38 cases). Four significant factors were fitted to a conditional logistic regression model.[14] This was achieved by numbering each organised dive consecutively and stratifying the regression by dive number. Non-significant differences ($P > 0.05$) between case-dives and control-dives were removed by backwards elimination.

Results

A total of 1,032 organised recreational dive profiles were collected from sites throughout WA, involving 137 organised group dives with a mean of 7.5 divers per organised group. The opportunity to participate was presented to 503 potential dive-series participants on the first dives of organised dive series and 321 of these 503 dive series (64%) were recorded. Figure 1 illustrates the relationship between dive series, individual divers and group dives.

Figure 1
Relationship between dive series, divers and group dives

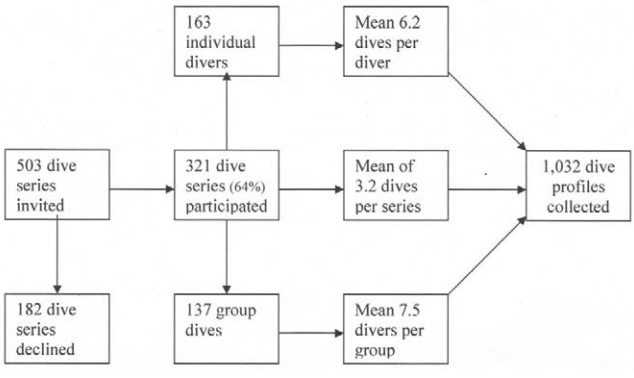
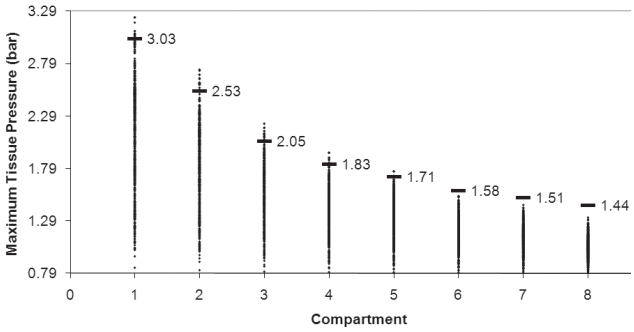


Figure 2
Maximum tissue pressure estimates during 1,032 dives; M-values for each DSAT tisue are shown by the horizontal bars



Recorded depths ranged from one to 45 msw, water temperatures ranged from 15°C to 29°C and vertical visibility was to the bottom or at least 20 m at the start of 792 (77%) of the dive profiles. Of the 163 individual divers who participated in one or more dive series, 117 (72%) were male and 46 (28%) female, with mean ages of 42.6 years (range 21–65, sd 10.0) for males and 38.7 years (range 24–58, sd 9.4) for females (Wilcoxon rank sum test $P < 0.01$). As the majority of dives were collected from live-aboard dive boats, bivariate tests were conducted to assess diver heterogeneity across dive platforms. Overall, there were no significant differences between the divers who dived from live-aboard boats (656 dives) and those who made day-trips (376 dives).

Figure 2 shows that the majority of dives resulted in maximum tissue pressures lower than the corresponding recommended M-values. Of the 1,032 recorded dive profiles, at least one DSAT M-value was exceeded during 38 dives (3.7%). These 38 dives were matched to 152 control dives in which divers at the same site on the same occasion recorded depth-time profiles that were estimated to have not exceeded any of the eight DSAT compartment M-values. Table 1 summarises half-times and M-values for the case dives for each of the eight DSAT compartments in the *SCUBA* package, the estimated maximum value reached based on the profiles recorded and the frequency with which each M-value was exceeded.

No shore dives exceeded an M-value. The proportion of dives exceeding an M-value was the same (4%) for both day-boat and live-aboard dives. When comparing case dives with control dives, the difference between the two groups on univariate analysis reached a P-value ≤ 0.01 in 10 of the many variables considered. (Table 2).

On surfacing, none of the case dives exceeded an M-value. As their maximum rates of ascent were actually higher, this suggests decompression was completed in-water before the final ascent.

MULTIVARIATE ANALYSIS

Nine of the 190 dives in the case/control set studied were not considered due to missing variables, leaving 181 dives for analysis. Adjusted odds ratios and confidence intervals for divers exceeding an M-value are given in Table 3. The three main risk factors for exceeding at least one M-value were female divers, diving to deeper average depths and being less likely to have been as deep as the maximum depth before.

Discussion

In this study, it was found that 38 of 1,032 dives exceeded an M-value. After taking into account the stratified nature of the data, these 'case' dives, when compared with 152 'control' dives were more likely made by females; this may have been due to the effect of clustering, as two divers accounted for 21 of the 38 (55%) cases. However, case dives were matched to control dives made in the same location at the same time and not to other dives made by individual divers, as in another recent study.[15] The relatively small sample size should be taken into account when interpreting these results.

The most commonly exceeded M-values were for the 10- and 20-minute half-time compartments (Table 1), which

Table 1
Theoretical compartment M-values and frequency and maximum values exceeded on 1,032 dives

Compartment	Half-time (mins)	Max. DSAT M-value (bar)	Divers exceeding M-value	Maximum estimated tissue pressure (bar)	Difference
1	5	3.03	14 (1.4%)	3.23	(+0.19)
2	10	2.53	34 (3.3%)	2.73	(+0.20)
3	20	2.05	29 (2.8%)	2.21	(+0.17)
4	30	1.83	14 (1.4%)	1.94	(+0.11)
5	40	1.71	6 (0.6%)	1.76	(+0.05)
6	60	1.58	0	1.52	(-0.05)
7	80	1.51	0	1.44	(-0.07)
8	120	1.44	0	1.32	(-0.12)

Table 2
Univariate differences between case and control dives

Variable	Case dives (n = 38)	Control dives (n = 152)	P-value
Sex ratio (female:male)	23:15	46:106	<0.01
Median weeks since previous dive	1.0	2.6	<0.01
Median years since certification	10.0	5.0	0.08
Number of dives in own scuba unit	80	48	<0.01
Deeper maximum depth (msw)	30.0	25.1	<0.01
Deeper average depth (msw)	16.0	12.3	<0.01
Not been as deep before	16/38 (42%)	23/152 (15%)	<0.01
Maximum depth planned pre-dive	6/38 (14%)	86/152 (53%)	<0.01
End dive with <50 bar gas	2/38 (5%)	36/152 (24%)	0.01
Felt cold during dive	13/38 (34%)	12/152 (8%)	<0.01
Maximum ascent rate (m min^{-1})	12.6	11.3	0.07
Able to state a 'safe' ascent rate	18/38 (47%)	110/152 (73%)	<0.01

would be relatively fast to equalise if they off-gassed at an exponential rate, as predicted by Haldane and assumed in the package *SCUBA*.[1,12] These tissues have also been found to be associated with higher post-dive bubble scores.[16] The exponential off-gassing rate used in this study to predict repetitive dive penalties may explain why no cases were estimated to have surfaced with tissue pressures in excess of an M-value (i.e., with omitted decompression). If a 60-minute limiting half-time had been used in our study, as is used by DSAT, then it is possible more of the repetitive case dives may have exceeded an M-value at the surface. There is, however, no evidence that any diver ignored the advice offered by a dive computer worn during this study.

That case dives were made by divers who had not been as deep before was a novel finding. No shore dives exceeded an M-value, which may have been due to the shallower depths associated with diving close to shore. This suggests dive organisers may be effective in preventing divers exceeding an M-value by carefully selecting dive sites.

Table 3
Overall risk factors for exceeding an M-value

Risk factor	Adjusted OR	(95% CI)	P-value
Female vs. male	5.63	(1.86, 17.03)	<0.01
Deeper av. depth	10.45	(3.02, 36.12)	<0.01
Not as deep before	14.49	(3.11, 66.67)	<0.01

The limitations of this nested case-control study include that no data were collected from non-participants. Therefore, it is unknown if non-participants differed to participants, nor if they declined (or participated) at more than one dive series. How organised recreational dives might differ from self-organised dives was also not explored, and caution is advised before generalising these findings to all recreational divers.

In conclusion, the majority of recreational divers in this study appeared to stay within accepted recreational diving depth/time limits and none are thought to have surfaced with omitted decompression obligations. This should be reassuring to the recreational diving industry in WA. Naturally, we nevertheless recommend dive organisers in WA encourage recreational dive groups to watch their displayed remaining no-stop time and to dive within the limits of their training and experience, even though this study did not find evidence of divers doing otherwise. Furthermore, this study successfully utilised stepwise depth/time dive profile analysis software with finer resolution than traditional table-based depth/time analysis methods, which may be of interest to other researchers.

Acknowledgements

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Analyser position for end-tidal carbon dioxide monitoring in a rebreather circuit

Alastair Ineson, Kaylene Henderson, David Teubner and Simon Mitchell

Key words
Carbon dioxide, hypercapnia, rebreathing, rebreathers/semi-closed circuit, rebreathers/closed circuit, physiology, simulation

Abstract
(Ineson A, Henderson K, Teubner D, Mitchell S. Analyser position for end-tidal carbon dioxide monitoring in a rebreather circuit. *Diving and Hyperbaric Medicine*. 2010;40(4):206-9.)
Introduction: A diving rebreather currently nearing release incorporates an infra-red CO_2 analyser at the end of the exhale hose and uses the expired gas CO_2 measurement made at this position to detect hypercapnia. This configuration may allow exhaled anatomic and mouthpiece dead space gas to mix with alveolar gas in the exhale hose thus falsely lowering the CO_2 measurement, especially at low tidal volumes.
Methods: A test circuit was constructed using a typical rebreather mouthpiece and exhale hose connected into an anaesthetic machine breathing loop. True end-tidal PCO_2 was measured in gas sampled from the mouth and compared breath-by-breath to the PCO_2 measured in gas sampled at the end of the exhale hose. Two subjects each completed 60 breaths at tidal volumes of 500, 750, 1000, 1500 and 2000 ml.
Results: There was a small (≤ 0.21 kPa) mean difference between true end-tidal CO_2 and end-of-hose CO_2 at tidal volumes of 1000 ml or more. However, at lower tidal volumes, the mean difference increased and, at 500 ml, it was 1.04 kPa and 0.70 kPa in subjects 1 and 2 respectively.
Conclusion: Measurement of the peak exhaled PCO_2 at the end of a rebreather exhale hose may provide a reasonable estimation of the true end-tidal CO_2 at large tidal volumes, but may significantly underestimate the true end-tidal CO_2 at low tidal volumes.

Introduction

In a new recreational diving rebreather, an infrared carbon dioxide (CO_2) analyser has been placed at the distal end of the exhale hose to measure "end-of-breath CO_2" and to warn of hypercapnia. We undertook this simple study specifically to investigate the potential for exhaled anatomic dead space gas to mix with alveolar gas during passage along an exhale hose, thus diluting the CO_2 measured at the analyser.

Methods

This study was approved by the Northern X Regional Ethics Committee of the New Zealand Ministry of Health. A test circuit (Figure 1) was constructed using a rebreather mouthpiece (Jetsam Technologies, Port Moody, Canada) with an internal volume of 40 ml, connected to a rebreather exhale hose (Carleton Technologies, New York, USA) with an internal volume of 340 ml. The inhale port of the mouthpiece and the end of the rebreather exhale hose were connected to the inhale and exhale hoses (respectively) on an Aestiva 7900 anaesthetic machine (GE Healthcare, Madison WI, USA) to establish a breathing circuit in which exhaled CO_2 was removed by an absorbent canister and tidal volume was measured by the circuit ventilator.

Gas sampling lines were plumbed into the rebreather mouthpiece to measure the 'true' end-tidal CO_2, and into the end of the rebreather exhale hose to approximate the CO_2 analyser position on the new rebreather (Figure 2). These lines were connected to identical infra-red CO_2 analysers (Datex-Ohmeda, Helsinki, Finland) on separate Aestiva anaesthetic machines. The analysers continuously draw gas through the sampling lines (200 ml min^{-1}) with a 2.5 s sampling delay and <400 ms delay in measurement, accurate to ± 0.2 vol%. The analysers are programmed to automatically zero every 60 minutes. Fresh gas flow into the circuit was nitrox 50 at 1 L min^{-1}. The anaesthetic machines and analysers were set up and checked by a qualified anaesthetic technician.

A resting subject breathed on the circuit with the nose pinched shut, using the digital readout on the ventilator to volitionally control tidal volume. Breath-by-breath peak CO_2 measurements were recorded simultaneously from the two positions for tidal volumes 500 ml, 750 ml, 1000 ml, 1500 ml and 2000 ml. Breaths were 'accepted' for each of these tidal volumes if they were between 450–550, 700–800, 900–1100, 1400–1600 and 1900–2100 ml respectively. Recordings were undertaken for each tidal volume in three groups of 20 breaths, giving 60 breaths per tidal volume per subject. The breathing rate was determined by subject comfort and no attempt was made to influence this. Two subjects completed this protocol on separate days.

The mean (± standard deviation) differences between 'true' end-tidal PCO_2 and the 'end-of-hose' PCO_2 were calculated for each tidal volume and each subject. Between-

Figure 1
Stylised layout of the experimental breathing circuit and monitoring devices

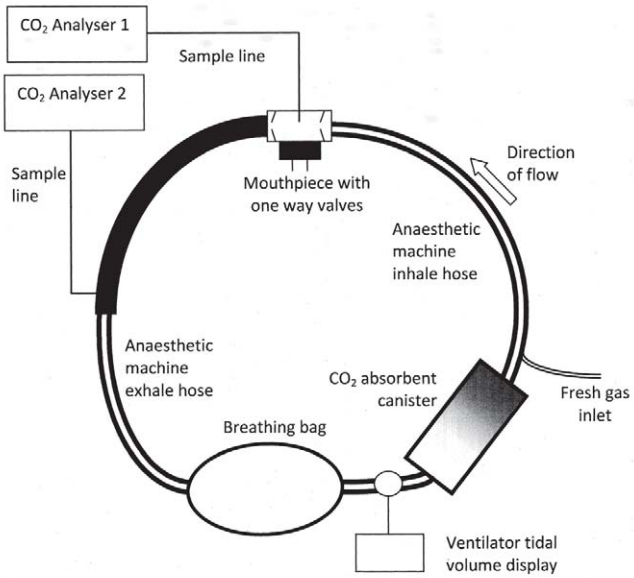
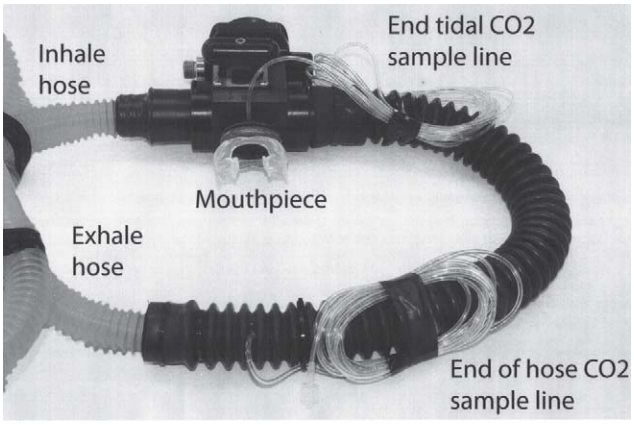


Figure 2
Rebreather mouthpiece and exhale hose showing sampling line positions for end-tidal and end-of-hose CO_2 sampling and connections to anaesthetic circuit



subject comparisons of these mean differences were made using a Student t-test (STATA 10SE, Statacorp, Texas). A *P*-value of less than 0.05 was taken to indicate statistical significance.

Results

Subject characteristics are reported in Table 1. The mean differences between true end-tidal PCO_2 and the PCO_2 measured at the 'end-of-hose' position were inversely related to tidal volume (Table 2), though at tidal volumes 1000 ml and above they were small and relatively constant. There were subtle but significant inter-subject differences at all tidal volumes except 750 ml. The subjects are reported separately.

Discussion

Hypercapnia is an important hazard in diving. A predisposition to CO_2 retention in divers has been documented in multiple studies which were reviewed recently.[1] This may result from perturbation of the respiratory response to rising arterial PCO_2 under conditions of increased work of breathing caused by breathing dense gas or the use of scuba equipment that imposes high breathing resistance or significant static lung loads. The resulting tendency to relative hypoventilation can lead to hypercapnia. Divers using rebreathers, in which gas is recycled around a closed-circle circuit, face the unique risks of failure in the valves which ensure unidirectional flow and of CO_2 absorbent failure. The CO_2 absorbent has a finite capacity which may become exhausted. If the absorbent canister fails, then the diver will rebreathe CO_2 and may become hypercapnic. Although there are no reliable data to describe the relative importance of hypercapnia as a disabling agent in accidents, there is abundant anecdote describing 'near misses', and there are examples of carefully investigated injuries or deaths that were strongly linked to hypercapnia.[2,3]

Until recently, attempts to detect impending hypercapnia were limited to the use of a temperature measurement device in the absorbent canister to monitor progress of the exothermic CO_2 absorbent reaction through the canister. These devices, colloquially referred to as 'temp sticks', are now deployed in a number of rebreathers and their efficacy in predicting scrubber exhaustion and CO_2 break-though has been reported.[4] However, although a temp stick may predict absorbent failure, it will not warn a diver that they are becoming hypercapnic. This would require direct measurement of CO_2 in the expired gas in a manner analogous to that used in anaesthesia.

Devices to measure PCO_2 in a mix of gases by using its unique absorbance of infra-red light are incorporated into anaesthetic monitoring systems. The measurement can be made directly by an analyser device placed on the breathing circuit at the entrance to the airway, or by sampling gas from

Table 1
Subject characteristics
(% of predicted forced vital capacity in brackets)

	Subject 1	Subject 2
Gender	Male	Female
Age (yr)	52	48
Height (cm)	186	164
Weight (kg)	87	66
Body mass index	25.1	24.5
Forced vital capacity (L)	5.97 (107%)	3.9 (107%)

Table 2
Mean (sd) 'true' end-tidal CO_2, 'end-of-hose' CO_2 ,the difference between them (kPa) and the 95% CI for the difference for the two subjects over the range of tidal volumes; *n* per subject for each tidal volume = 60; means rounded to one decimal place

Tidal volume (ml)	500	750	1000	1500	2000
Subject 1					
True end-tidal CO_2	6.0 (0.23)	6.0 (0.16)	6.1 (0.23)	5.8 (0.27)	5.6 (0.21)
End-of-hose CO_2	5.0 (0.51)	5.7 (0.20)	5.9 (0.23)	5.6 (0.23)	5.4 (0.07)
Difference	1.0 (0.53)	0.3 (0.08)	0.2 (0.08)	0.2 (0.07)	0.2 (0.07)
95% CI	0.90–1.18	0.28–0.34	0.19–0.23	0.18–0.22	0.19–0.23
Subject 2					
True end-tidal CO_2	4.8 (0.07)	4.5 (0.10)	4.8 (0.26)	3.7 (0.037)	3.6 (0.37)
End-of-hose CO_2	4.1 (0.12)	4.2 (0.11)	4.6 (0.26)	3.5 (0.36)	3.4 (0.32)
Difference	0.7 (0.10)	0.3 (0.07)	0.2 (0.06)	0.1 (0.05)	0.2 (0.10)
95% CI	0.67–0.73	0.28–0.32	0.16–0.20	0.11–0.13	0.61–0.19

the same point and carrying it back to an analyser remote from the circuit. Either strategy provides a continuous measurement of the PCO_2 in gas entering and leaving the airway.

Selecting a sampling point at the entrance to the airway imparts several crucial advantages. First, inspired gas can be analysed to ensure it is free of CO_2, thus warning of any CO_2 rebreathing. Second, the anatomical dead space gas will pass distally in the circuit early during exhalation, and the end-tidal gas can therefore be assumed to represent mixed alveolar gas. Since alveolar gas is in equilibrium with dissolved arterial gases, the PCO_2 measured in the end-tidal gas can be used as an approximation of arterial PCO_2. In fact, the end-tidal CO_2 usually underestimates the true arterial PCO_2 by a small amount due to a contribution to mixed alveolar gas by well-ventilated, poorly perfused alveolar units that contain little CO_2, but in healthy individuals the difference is small enough that it is typically ignored for most purposes.

Infra-red CO_2 detection devices are now sufficiently small, moisture-resistant, and economical of power to allow deployment in rebreather circuits. For the reasons described above, such a device would ideally be deployed at the entrance to the airway (in the rebreather mouthpiece) to allow measurement of both the inspired CO_2 and the end-tidal CO_2. Unfortunately with current devices, this would render the mouthpiece too bulky. Moreover, drawing a sample from the mouthpiece to a remote analyser would require a pump with high power consumption.

Placement of the analyser at the distal end of the exhalation hose avoids these problems, but may allow mixing of anatomical and mouthpiece dead-space gas with the alveolar gas within the exhale hose prior to measurement of the 'end-of-hose' PCO_2. Since the total dead-space volume is approximately 200 ml (150 ml anatomical dead space and 50 ml mouthpiece dead-space), this would have little impact on the measured end-of-hose PCO_2 after a large tidal volume exhalation. However, at smaller tidal volumes the dilution effect might become significant.

Our study confirmed these suspicions. There was a small and physiologically insignificant difference between the true end-tidal CO_2 and the end-of-hose CO_2 during respiration at larger tidal volumes (≥ 1000 ml), but this difference became larger at lower tidal volumes; averaging 1 kPa (7.5 mmHg) in one subject and 0.7 kPa (5.3 mmHg) in the other at 500 ml tidal volume. This between-subject difference may be accounted for by a larger dead space volume in the larger, male subject. Although a relatively small difference in gas partial pressure, 1 kPa (7.5 mmHg) is potentially a physiologically significant difference in arterial PCO_2. If a warning device underestimated the end-tidal CO_2 by 1 kPa (7.5 mmHg) during an episode of hypercapnia, then this could delay an appropriate response and make incapacitation more likely.

Interpretation of this finding is difficult because little is known about tidal volumes in typical diving situations. A number of studies of exercise in controlled underwater laboratories have reported tidal volumes as part of their datasets.[5–7] These data suggested that tidal volumes less than 1000 ml are not often encountered under the conditions imposed by the various experiments, at rest or during exercise.

Taken together with our findings, this suggests that an end-of-hose CO_2 measurement may be an adequate indicator of hypercapnia in the majority of circumstances. Nevertheless, an event has been reported in which an exercising, immersed diver was subjected to an increase in breathing resistance and responded by increasing respiratory rate and reducing tidal volumes (to approximately 600 ml).[8] The diver appeared completely unaware of a consequent rapid rise in the end-tidal CO_2 and became incapacitated almost without warning. A similar event was described in a second diver

but respiratory volumes were not reported. It is in this type of 'low tidal volume, progressive hypercapnia' scenario that inaccuracy in end-of-hose CO_2 measurement might be important. It is plausible that such scenarios are more common than we realise as part of the fatal pathway in hypercapnic events during diving.

It must be acknowledged that this simple study has several limitations and must be interpreted cautiously. Firstly, we did not utilise the mouthpiece and exhale hose from the new rebreather in which end-of-hose CO_2 monitoring will occur. Differences in volume and geometry may result in less mixing in this device's exhale hose, but it is also possible there may actually be more mixing, especially since it appears to have a greater diameter than the components used in this study. Secondly, the study was conducted with normocapnic subjects and we did not replicate the hypercapnic conditions that the end-of-hose analyser is in place to detect. We believe our method was adequate for the purpose of demonstrating a difference between true end-tidal CO_2 and end-of-hose CO_2, but we do acknowledge that no hypercapnic test was performed. Thirdly the study was not performed under real diving conditions. Diving may increase anatomical dead space if tidal breathing is conducted at higher lung volumes, a ventilation pattern which is known to be adopted during respiration with dense gas.[9,10] If this occurred, the dilution effect may be greater than we have demonstrated. Finally only two subjects were studied. Nevertheless, all humans have anatomical dead space and all rebreathers have mouthpiece dead space. Our findings are thus predictable from physiological first principles. Studying a larger number of subjects possessing a wider range of physical characteristics might further define the magnitude of effect but it would be very unlikely to change our fundamental conclusion.

Conclusions

Our results confirm that dilution of alveolar gas by dead-space gas does occur in the exhale hose of a rebreather during low tidal volume breathing. In respect of estimating end-tidal CO_2 by analysing gas at the end of the hose, this dilution is likely to be insignificant during breathing at tidal volumes that are thought to be typical of many diving scenarios. Nevertheless, it is possible that significant under-estimation of end-tidal CO_2 could occur when CO_2 measurements are made at the end-of-hose position during low tidal volume breathing unless some form of compensation is built into the interpretation algorithm.

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S100B and its relation to intravascular bubbles following decompression

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Key words
Biomarkers, decompression sickness, bubbles, Doppler, diving research

Abstract
(Havnes MB, Hjelde A, Brubakk AO, Møllerløkken A. S100B and its relation to intravascular bubbles following decompression. *Diving and Hyperbaric Medicine*. 2010;40(4):210-2.)
Introduction: When neurological damage occurs in divers, it is considered to be caused by gas bubbles. Entrapment of these bubbles may lead to cellular injury and cerebral oedema. S100B is a protein biomarker that is released in CNS injuries and the concentration is related to the amount of brain damage.
Methods: A total of 27 rats were randomly assigned to one of three groups. Group I served as controls (n = 9). Group II (n = 7) underwent a simulated dive to 400 kPa and Group III to 700 kPa (n = 11). In groups II and III, venous gas bubble scores were evaluated by ultrasound during the first hour after surfacing. The amount of S100B in serum after the dives was tested using a commercial ELISA kit. Bubble grades were compared to S100B protein concentrations.
Results: The average level of S100B was significantly higher in rats compressed to 700 kPa compared to the control rats, (P = 0.038) and the rats compressed to 400 kPa, (P = 0.003). There was no difference in S100B concentration between groups I and II. Following the dive to 700 kPa, there were significantly higher bubble grades observed than following the dive to 400 kPa (P = 0.001).
Conclusion: The correlation between bubble grade and an increase in serum protein level of S100B indicates that this protein may be useful as a biomarker for neurological damage caused by decompression.

Introduction

When neurological damage occurs in divers, the prime suspects are vascular gas bubbles. Most bubbles are filtered out by the pulmonary capillaries but may also break through the lung filter or enter the arterial circulation via shunts to mediate bubble-induced tissue injury.[1,2] Entrapment of these bubbles may lead to cellular injury and cerebral oedema.[3] Under normal conditions, central nervous system (CNS) tissue is separated from plasma by the blood-brain-barrier (BBB), formed by endothelial cells of the brain microvessels and their underlying basement membrane.[4] The BBB acts as a sieve to restrict passage of large molecules, including most plasma proteins, into the CNS. Increased permeability of the BBB has been demonstrated after decompression, resulting in oedema.[3,5,6] Increased permeability of the cerebral vasculature could be due to changes induced by chemical factors released or activated by microbubbles or to direct mechanical injury of blood vessels from these bubbles.[7]

S100B is a protein biomarker that is released in CNS injury, the concentration being related to the amount of brain damage.[8] Elevated S100B concentrations have been reported in patients with stroke compared to control subjects and also in elite breath-hold divers after prolonged apnea.[9,10]

Signs and symptoms of decompression sickness (DCS) differ with the pressure profile and the breathing gas, but have a common first step, namely the formation of gas bubbles. The emerging evidence of the effects of venous gas emboli on the endothelium has led to the hypothesis that these are a main cause for neurologic DCS through its adverse effects on the CNS.[11] This experiment examines whether S100B could be used as a biological marker of decompression stress in the CNS and to examine a possible correlation between bubble grade and serum S100B concentration.

Methods

All experimental procedures and the care of experimental animals conformed to the European Convention for the Protection of Vertebrate Animals Used for Experimental and Other Scientific Purposes, and the protocol was approved by the Norwegian Council for Animal Research.

A total of 27 rats (female Sprague Dawley, Scanbur, Denmark) were randomly divided into three groups. Group I was a surface control group (100 kPa) breathing air (n = 9). Groups II and III underwent simulated dives for 45 min to 400 kPa (group II, n = 7) or 700 kPa (group III, n = 11) in a 20 L hyperbaric chamber. The compression and decompression rates were similar in both groups, 200 kPa min^{-1} and 50 kPa min^{-1} respectively. During the simulated dive, the rats were awake and observed through a window in the hyperbaric chamber.

Immediately after surfacing, the rats were anaesthetised with a subcutaneous injection of a mixture of haloperidol 0.33 mg, fentanyl 0.05 mg and midazolam 0.5 mg; 0.4 ml per 100 g body weight. The pulmonary artery was monitored at discrete intervals (15, 30, 45 and 60 min after surfacing) for gas bubbles using a 10 MHz transducer connected to a FiVe ultrasound scanner (GE Vingmed Ultrasound AS, Norway). Bubbles are seen on the monitor screen as bright

spots in the pulmonary artery, and verified with Doppler. Bubble quantity was graded on a 0 to 5 scale according to a previously described method.[12] Results are presented as maximum bubble grade during the observation period.

S100B levels in serum drawn one hour after the dives were tested using a commercial ELISA kit (BioVendor-Laboratorní medicína, Brno-Modice, Czech Republic). Bubble grades were compared to S100B protein concentrations.

STATISTICAL ANALYSIS

Because the S100B data in the control group were not normally distributed (confirmed by the Kolmogorov-Smirnov test and Q-Q plot) and the small number of animals studied, the Mann-Whitney test was used to assess differences between the groups. Statistical significance was set at $P < 0.05$. All statistical analyses were performed using SPSS 16.0 (SPSS Inc., Chicago, Illinois, USA). The results are presented as medians (25th and 75th percentiles).

Results

There was a significant difference in S100B concentration between the rats compressed to 700 kPa (Group III) and the rats in the control group (Group 1, $P = 0.038$) (Figure 1). There was also a significantly higher S100B concentration in the rats compressed to 700 kPa (Group III) compared to those compressed to 400 kPa (Group II, $P = 0.003$) (Figure 1). There were no significant differences between Groups I and II. Following the dive to 700 kPa, there were significantly higher bubble grades observed than following the dive to 400 kPa ($P = 0.001$) (Table 1).

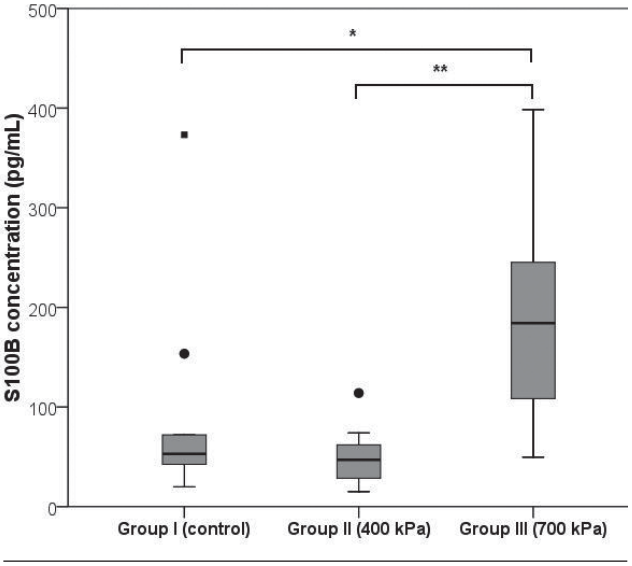
Discussion

We showed an increased concentration of S100B and a higher occurrence and grade of bubbles in rats compressed to 700 kPa compared to both normobaric controls and rats compressed to 400 kPa. The adverse effects of decompression have been discussed for decades, but markers for decompression stress other than the detection of vascular gas bubbles are still lacking. Although intravascular gas bubble scores are related to the risk of DCS and, higher grades with an increased incidence of DCS, there are large inter- and intra-individual differences in the response to bubbles. Thus, the search for other markers of decompression stress has continued. This inter-individual susceptibility to DCS and the fact that

Table 1
Bubble grades after dives to 700kPa and 400kPa (number of animals in each grade; *P = 0.001*)

	Bubble grade					
	0	**1**	**2**	**3**	**4**	**5**
Group II (400 kPa; $n = 7$)	4	3	0	0	0	0
Group III (700 kPa; $n = 11$)	2	0	1	0	0	8

Figure 1
Box plots of serum S100B concentration in undived rats and rats compressed to 400 kPa and 700 kPa, median, 25th and 75th percentiles shown; vertical lines represent the largest and smallest values except:
• outlier values > 1.5 box-lengths away;
■ extreme outlier > three box-lengths away;
(differences between groups: * P <0.05; ** P <0.005)



repetitive dives appear to result in greater tolerance to DCS due to acclimatisation, have given rise to the hypothesis that DCS might have an inflammatory basis.[13,14]

To our knowledge, there are no published data relating bubble formation and S100B concentration. However, S100B has been shown to be increased in goats after deep dives with rapid decompression.[15] These data are supported by our study in rats. On the other hand, a pilot study on S100B in human divers diagnosed and treated for acute DCS did not show an increased concentration of S100B.[16] However, a major difference between that study and ours is that the blood samples were drawn two to three days after the dives, while ours were drawn one hour after the dive. The half-life of S100B in relation to other diseases is estimated to be about 30 minutes, which might explain this difference.[17] In studies of patients with Alzheimer's disease or traumatic head injury, S100B appears to be a useful marker for brain function and, in ischaemic stroke patients, S100B concentrations correlate with infarct volume.[18–20]

Whether a high bubble grade produces brain injury in rats is not known, but it is reasonable to believe so. Injected microbubbles have been shown to affect the BBB in guinea pigs.[21] This method might give better control of the amount of bubbles than in decompressed rats, but is highly invasive and, thus, might alter other variables that could affect biomarker production.

We cannot say for sure that the concentration of S100B in serum is related to neurological DCS since we do not have

a neurological examination of the rats. However, recent experiments suggest that bubbles caused by diving can affect the BBB, and that there is an increased concentration of S100B in human subjects after traumatic brain injury.[22,23] Our preliminary results indicate a correlation between exposure to pressure and the expression of S100B.

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Case reports

Severe hydrogen sulphide poisoning treated with 4-dimethylaminophenol and hyperbaric oxygen

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Key words
Hyperbaric oxygenation, hyperbaric oxygen therapy, clinical toxicology, exogenous poison, resuscitation, outcome

Abstract
Lindemann J, Matzi V, Neuboeck N, Ratzenhofer-Komenda B, Maier A, Smolle-Juettner F-M. Severe hydrogen sulphide poisoning treated with 4-dimethylaminophenol and hyperbaric oxygen. *Diving and Hyperbaric Medicine*. 2010;40:213-7.)

Introduction: Hydrogen sulphide (H_2S) is a highly toxic gas which originates mainly during breakdown of organic matter under anaerobic conditions. After inhalation, H_2S binds to mitochondrial respiratory enzymes preventing oxidative phosphorylation, thereby causing reversible inhibition of aerobic metabolism and cellular anoxia. The use of hyperbaric oxygen therapy (HBOT) for H_2S poisoning remains controversial, but has a similar underlying rationale to that in carbon monoxide poisoning.

Methods: A retrospective review of patients with severe H_2S intoxication who presented during 2006 and 2007 was carried out. Ten victims of severe occupational H_2S poisoning were identified, of whom four died at the site of the accident. Two further patients required cardiopulmonary resuscitation at the site of the accident and the remaining four all received 100% oxygen followed by endotracheal intubation and artificial ventilation prior to hospital admission. In these six cases, 4-dimethylaminophenol was administered on admission as an antidote, followed immediately by HBOT using the schedule otherwise used in carbon monoxide intoxication.

Clinical outcome: The two patients who required cardiopulmonary resuscitation at the site of exposure died of cerebral ischaemia or pulmonary oedema on the first and seventh days after the accident respectively. The remaining four patients recovered without any neurological sequelae and were discharged for outpatient care after a median of nine days (range 8–12 days). No antidote-related adverse effects could be detected. Acid-base status and oxygenation improved and methaemoglobin fell with the first HBOT in all six cases.

Conclusion: In severe H_2S intoxication, supportive HBOT may play a useful role in improving oxygenation and acid-base status quickly and counteracting the decrement in oxygen carriage caused by methaemoglobinaemia due to antidote administration.

Introduction

Hydrogen sulphide (H_2S) is a colourless, flammable, highly toxic, irritant gas with a characteristic odour of 'rotten eggs'. This potentially life-threatening gas usually results from the breakdown of organic matter in the absence of oxygen and is released as a by-product of industry and agriculture. After inhalation, H_2S is rapidly absorbed through the respiratory mucosa, distributing mainly into the lungs and brain. The mechanism of toxicity, with inhibition of oxidative phosphorylation, seems to be similar to that of cyanide and carbon monoxide (CO) poisoning.[1] The severity of clinical symptoms of H_2S poisoning varies with the concentration and duration of exposure to H_2S; low concentrations cause respiratory tract irritation resulting in cough, dyspnoea and local mucosal soreness. With increasing concentrations of H_2S, neurological symptoms develop, high concentrations causing severe cerebral and pulmonary oedema which may lead to brain death, asphyxia and cardiopulmonary arrest (Table 1).[2]

The management of patients suffering from H_2S intoxication remains a therapeutic challenge. The mainstay of treatment is rapid rescue from the site of exposure, immediate 100% oxygen, resuscitation as clinically indicated and administration of antidotes as early as possible. The use of hyperbaric oxygen therapy (HBOT) has been reported, but remains controversial.[3,4] We undertook a retrospective study of our experience at the Medical University Hospital, Graz, in managing acute severe H_2S poisoning.

Methods

We did a retrospective analysis of patients treated with HBOT in combination with 4-dimethylaminophenol therapy for severe H_2S intoxication occurring in two industrial accidents in October 2006 and July 2007. The study was approved by the local ethics committee and was conducted in accordance with the precepts of the Declaration of Helsinki. Six out of 10 victims with severe intoxication (two males and four females, aged 26 to 60 years, mean 39.8 years) were

Table 1
Health effects of hydrogen sulphide at various exposure levels

Concentration (ppm)	Effects
2	Chronic exposure: fatigue, headache, loss of appetite, weight loss, diarrhoea
100	Mild eye and lung irritation: 'gas eye', coughing, dyspnoea, sore throat
200	Olfactory paralysis
300	Pulmonary oedema, nausea, vomiting
500	Cerebral oedema, vertigo, dizzyness, somnolence, convulsions, loss of consciousness
800	Unconsciousness; death within 5 min
1000	Immediate collapse; asphyxia with cardiac arrest after inhalation of a single breath

referred to our centre and are documented in this report. In fact, a total of 25 victims, of whom four died at the site, were involved in these two accidents. Twenty-one patients underwent HBOT at our department. Fifteen patients who suffered only mild intoxication are not included in this report. They were conscious and had no need of ventilatory assistance or circulatory support, but complained of mild symptoms such as headache, dizziness, nausea, emesis and dyspnoea, or had signs of mucosal irritation with cough, sore throat and/or eye strain ('gas eye'). The six severe cases who are the subject of this report had respiratory and circulatory shock, were unconscious and needed artificial ventilation and intensive circulatory support, including cardiopulmonary resuscitation in two patients.

Case reports

PRE-HOSPITAL COURSE

The accidents occurred in large pelt-processing factories and tanneries during cleaning of or due to gas leakage from tanks containing rests of chromium sludge and sulphuric acid. In one case, an above-ground pipeline had leaked. None of the victims had worn protective equipment or clothing while working within endangered areas. Six workers collapsed immediately after taking a few breaths, fell unconscious and required cardiopulmonary resuscitation (CPR), performed by the local emergency doctors, after having been moved away from the site of H_2S exposure by their co-workers. Two regained a circulation after 20 and 25 minutes of resuscitation respectively, but four died at the site of the accident. Four further victims were able to leave the site of exposure but then became unconscious immediately afterwards and developed acute respiratory failure and hypoxaemia, in three cases combined with arterial hypotension.

INITIAL HOSPITAL MANAGEMENT

The six critically-ill, intubated and ventilated survivors were evacuated by helicopter to our centre. On admission, all remained comatose and in critical condition, with mid-dilated pupils and a Glasgow Coma Scale score of three. Mean systolic blood pressure was 13 kPa (range 11–15 kPa, 80–110 mmHg) and diastolic 8 kPa (range 4–9.3 kPa, 30–70 mmHg). Mean heart rate was 88 beats per minute (range 85–90 bpm). As first-line antidote, 3 mg per kg body weight of 4-dimethylaminophenol (4-DMAP) was administered intravenously. The time interval between accident and administration of 4-DMAP ranged from 60 to 90 minutes. In addition, 250 mg prednisolone was given intravenously. An emergency chest X-ray, electrocardiogram (ECG) and laboratory tests including carboxyhaemoglobin, methaemoglobin (MetHb), arterial blood gases and lactate were performed.

Table 2
Biochemical and blood-gas measurements before and after the first HBOT

Patient	1	2	3	4	5	6
Methaemoglobin (%)						
Pre-HBOT	11.2	17.8	0.8	8.6	7.5	5.0
Post-HBOT	6.8	8.9	0.2	2.5	5.1	2.0
Lactate (mmol L^{-1})						
Pre-HBOT	4.3	2.0	1.3	2.9	1.3	12.4
Post-HBOT	3.5	1.5	0.5	1.7	1.3	8.4
F_IO_2 (%)						
Pre-HBOT	100	100	100	100	100	100
Post-HBOT	100	40	50	70	90	40
P_aO_2 (mmHg)						
Pre-HBOT	320	250	248	125	471	350
Post-HBOT	310	158	185	131	344	207

Table 3
Patient characteristics and clinical course
(AF - atrial fibrillation; VF - ventricular fibrillation; ARDS - Adult respiratory distress syndrome)

Patient	Sex	Age (yrs)	CPR	Time to DMAP	Time to HBOT	Number HBOT	Diagnosis	Intubation (days)	ICU (days)	Hospital (days)	Outcome
1	F	39	Yes	90	100	2	VF, cerebral ischaemia & oedema pulmonary oedema, malignant hyperthermia	1	1	1	Death (day 2)
2	F	40	No	60	100	6	Respiratory shock, pleural effusions	1	2	8	Full recovery
3	F	26	No	60	100	6	Respiratory & circulatory shock, cerebral oedema	1	2	8	Full recovery
4	F	46	No	60	100	11	Respiratory & circulatory shock, pulmonary oedema, ARDS	4	5	12	Full recovery
5	M	28	No	60	100	8	Respiratory & circulatory shock, 'gas eye'	1	2	8	Full recovery
6	M	60	Yes	90	125	1	Cardio-respiratory arrest, AF, cerebral ischaemia & oedema pulmonary oedema, ARDS, artificial hypothermia	7	7	7	Death (day 8)

HBOT

Bilateral myringotomies were performed, and the intubated, ventilated patients proceeded immediately to HBOT in the largest multiplace hyperbaric chamber in central Europe, which enables simultaneous treatment of up to five intensive care patients. HBOT was administered according to the protocol used in CO intoxication: first treatment; 304 kPa for 60 min then 223 kPa for 30 min; subsequent treatments were at 223 kPa for 90 min. Two HBOT were given within the first 24 hours, followed by a single daily HBOT until symptoms had subsided and the levels of MetHb were within the normal range. After HBOT, the patients were transferred to the intensive care unit for ongoing management.

The mean time interval between accident and the start of HBOT was 104 minutes (100–125 min). The median number of HBOT was 5.7 for all patients (range 1–11 treatments). HBOT was well tolerated. In these critically-ill patients, there was a short-term need for intensified circulatory assistance (enhancement of catecholamines intravenously), but after about ten minutes, all patients had stable cardiorespiratory parameters.

Arterial blood gas analysis was performed in every patient before and after the first HBOT (Table 2). The mean MetHb level fell by about 49%, from 8.5% to 4.3% (normal range 0.4–1.0% of total haemoglobin) and mean lactate decreased about 30%, from 4.0 mmol L^{-1} to 2.8 mmol L^{-1} (normal range 0.5–2.2 mmol L, whilst the mean pH increased slightly from 7.36 to 7.40. Furthermore, the inspired oxygen (F_iO_2) was lowered in five patients (mean post-HBOT F_iO_2 65%). These data are summarised in Table 2.

CLINICAL COURSE AND OUTCOME

The two patients who required on-site CPR both had elevated cardiac biomarkers (myoglobin, creatine kinase-MB, lactate dehydrogenase and troponin T). The ECG in the other four patients revealed sinus rhythm without ST-elevation or any other signs of myocardial ischemia and no increase in cardiac biomarkers. Toxic lung oedema and adult respiratory distress syndrome were observed in three and two cases respectively, one of whom had required on-site CPR. Cerebral and thoracic CT-scans were performed to evaluate the severity of cerebral ischemia and cerebral or pulmonary oedema. Cerebral oedema and cerebral ischemia occurred in three patients, whilst one patient suffered from kerato-conjunctivitis and another victim developed small pleural effusions. One of the two CPR patients underwent artificial hypothermia (33^{O}C body temperature for 24 hours) after the first HBO treatment and received no further HBOT. The other post-resuscitation patient developed malignant hyperthermia (42^{O}C body temperature) after the second HBOT, and was given additional 4-DMAP, 3 mg per kg body weight. Other treatments comprised administration of mucolytic agents and bronchodilators, alone or in combination with corticosteroids. In all six patients, prophylactic antibiotic therapy was initiated in order to avoid pulmonary bacterial superinfection. The clinical courses of the six patients are summarised in Table 3.

Discussion

Despite similarities to both cyanide and CO poisoning, the detailed pathogenic mechanisms of H_2S poisoning are not fully understood.[5–7] Several antidotes, such as amyl or sodium nitrite and 4-DMAP, have been advocated.[2,8]

The main therapeutic goal of antidote administration is to inhibit sulphide binding to intracellular respiratory enzymes enabling sulphide detoxification and restoration of function of the oxidative chain. Both nitrites and 4-DMAP support the conversion of haemoglobin to methaemoglobin which readily binds to the toxic hydrosulphide anion until the latter is detoxified by haeme-catalyzed oxidation. Aerobic metabolism is enhanced by re-activation and protection of cytochrome oxidase. However, there is no clear evidence of their efficacy, and the emergency administration of antidotes that induce methaemoglobinaemia is not without risk.[2,8–10]

To be effective, nitrite therapy should be initiated within the first few minutes after H_2S poisoning.[2,8] It is postulated that nitrite-induced methaemoglobinaemia occurs preferentially under poor oxygen conditions.[11] An oxygen-enriched environment, such as during resuscitation and/or ventilation with oxygen, may favour sulphide depletion, and nitrite administration may actually slow sulphide removal under these circumstances. Other adverse effects of nitrites are hypotension, tachycardia, vomiting, headache and biochemical interaction with haemoglobin-oxygen dissociation due to excessive nitrite-induced methaemoglobinaemia. In contrast to most other reports, we used DMAP as the initial antidote, and adverse effects of methaemoglobinaemia were not observed.

Isolated case reports have suggested that HBOT may be effective in the treatment of excessive methaemoglobinaemia.[8,12–15] The biochemical mechanism for this is based on enhanced reduction of methaemoglobin levels due to prevention of the oxidation of haemoglobin. In our series, considerable methaemoglobinaemia was caused by inhalation of high concentrations of H_2S and not due to administration of the antidote 4-DMAP, which caused no apparent side effects in our patients.

Further useful effects of HBOT are increased tissue oxygen tension, vasoconstriction, reduction in cerebral oedema, a decrease in leucocyte adhesion, down-regulation of inflammatory mediators and enhancement of nerve cell regeneration.[16–19] HBOT at 304 kPa has been shown in an animal model to be effective in treating H_2S poisoning, particularly in combination with early sodium nitrite therapy.[20] Case reports in human victims also appear promising.[2,8,12,21–23] Neurological symptoms appear to respond readily to HBOT, and neurological sequelae may be prevented, even if HBOT is delayed.[21–26] These reports and our own experience would suggest that the role of HBOT in severe H_2S poisoning is probably supportive rather than as an antidote.[8,12] In addition, in cases of antidote-related side effects, e.g., respiratory insufficiency, or failure of antidote therapy, HBOT may be the treatment of choice. Moreover, the extent of concomitant cerebral and/or pulmonary oedema, which is often observed in severe cases, can be decreased significantly by HBOT, possibly resulting in reduction of the mortality rate.[22] Objective benefits from HBOT in the present patient series were the reduction in MetHb and an improvement in acid-base status and oxygenation, as documented with the first therapy. Despite this, the mortality in severe H_2S intoxication remains high.

Despite successful use in both experimental and clinical H_2S intoxication, an optimal dose or duration of HBOT has not been established.[2,8,12,21–23] Given the somewhat similar pathogenic mechanisms to CO intoxication, it would seem justified to treat H_2S intoxication using regimens similar to those described for CO poisoning.[27]

Conclusions

Supportive HBOT in severe H_2S intoxication ensures rapid correction of hypoxia and counteracts any antidote-induced decrement in oxygen transport capacity caused by MetHb. HBOT not only enables and/or supports efficient emergency treatment but appears to help avoid neurological sequelae. Even when first-line antidote therapy has failed and/or side effects have occurred, a benefit from secondary HBOT can be achieved. Controlled clinical studies of HBOT for H_2S poisoning are needed, but will be difficult to achieve given its rarity and the limited access to hyperbaric facilities.[28] In the meantime, cases series such as this are the best evidence available.

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Hyperbaric oxygen in the treatment of asphyxia in two newborn infants

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Key words
Hyperbaric oxygen, hyperbaric oxygen therapy, neuroprotection, brain injury, children, case reports

Abstract

(Orozco-Gutierrez A, Rojas-Cerda L, Estrada RM, Gil-Rosales C. Hyperbaric oxygen in the treatment of asphyxia in two newborn infants. *Diving and Hyperbaric Medicine*. 2010;40(4):218-20.)
Hypoxic-ischaemic encephalopathy (HIE) is a common cause of brain damage in the neonatal period. Approximately 10% of births involve some degree of asphyxia, and 1% of these are severe. Current treatment has been limited to supportive measures and the recent use of hypothermia. Beneficial effects of hyperbaric oxygen treatment (HBOT) in neonatal asphyxia have been reported in the Chinese literature. We report the use of HBOT to treat two term neonates with moderate HIE according to Sarnat's classification. Clinical improvement occurred following HBOT. A 50% decrease in the total creatine phosphokinase (CPK) level and a 40% decrease in the CPK myocardial fraction were observed within 24 hours of the first treatment. The decline in CPK levels may be related to a reduction in the overall systemic inflammatory process and cannot be attributed solely to a reduction in brain damage. HBOT may have a role in HIE.

Introduction

Perinatal asphyxia occurs at disruption or cessation of gas exchange at the placenta or lung, causing progressive hypoxaemia and hypercapnia. Approximately 10% of births involve some degree of asphyxia, and 1% of these are severe.[1] Hypoxic-ischaemic encephalopathy (HIE) is a common cause of brain damage in the neonatal period and remains an important cause of neonatal morbidity and mortality.[1,2] Current therapeutic strategies involve mainly supportive measures, e.g., adequate oxygenation and ventilation, blood pressure support, maintenance of a normoglycaemic state, and fluid management. Recently, hypothermia has been used to decrease the neurological sequelae of HIE.[2]

Theoretical and anecdotal evidence exists for the beneficial effects of hyperbaric oxygen therapy (HBOT) in neonatal asphyxia.[3–5] Meta-analysis of 20 Chinese trials demonstrated a significant reduction in mortality in neonates with HIE treated with HBOT compared to those not receiving HBOT (odds ratio (OR) 0.26, 95% confidence interval (CI) 0.14, 0.46). Neurological damage was also reduced in infants treated with HBOT (OR 0.41, 95% CI 0.27, 0.61).[6] However, these studies were not performed in a blinded fashion, and the results should be interpreted with caution.

Based on these data, HBOT was used for two newborns with HIE. Permission to report these cases was obtained from the parents. Clinical evaluation, using the Sarnat Scale,[7] and measurements of creatine phosphokinase (CPK) and lactate dehydrogenase (LDH) levels were used to assess patient progress. Breathing 100% oxygen, each patient was compressed to 203 kPa for 30 minutes, followed by decompression over five minutes, followed by a further 30 minutes of oxygen breathing at room pressure. Both patients received standard care, including body temperature control, early nutritional support and glucose monitoring. Anticonvulsants and diuretics were not used.

Case 1

Following a traumatic vaginal delivery with a prolonged second stage of labour, Kristeller manoeuvres were performed for a cord round the neck. The child, weighing 4.2 kg, was diagnosed with perinatal asphyxia, ultrasound-documented subarachnoid hemorrhage and diffuse left renal injury. At 36 hours of age, the patient became lethargic, and the sucking reflex slowed. Examination revealed an incomplete Moro reflex, caput succedaneum, and a right parietal cephalohaematoma (Sarnat scale II); the head circumference was 37.5 cm. Total creatine phosphokinase (CPK), creatine kinase-myocardial fraction (CK-MB) and lactate dehydrogenase (LDH) values are shown in Table 1.

After obtaining informed consent from the family, three HBOT sessions were initiated, commencing at 42 hours of age. At 48 hours, after the first HBOT, clinical improvement had occurred and head circumference had decreased by 0.5 cm. A second HBOT was given at 68 hours of age and a third at 96 hours. Neurologic examination had normalised, and a significant decrease in the size of the cephalohaematoma was noted. CPK, CPK-MB, and LDH levels improved (Table 1). The patient was discharged without further complications. Auditory evoked potentials, cerebral ultrasound, and an electroencephalogram were normal.

Case 2

A newborn, weighing 2.9 kg, experienced moderate asphyxia secondary to uterine rupture and Caesarean delivery. Apgar

scores were 3, 4, 5, and 8 at 1, 5, 10, and 15 minutes, respectively. On admission to the neonatal intensive care unit, the newborn demonstrated a slow sucking reflex, increased muscle tone, decreased reflexes, and an incomplete Moro reflex (Sarnat scale II). Initial CPK, CPK MB and LDH are shown in Table 1.

After parental permission, two HBOT sessions were given, commencing at 12 hours of age. After the first, decreased irritability and improved reflexes were observed. After the second, completed by 36 hours of age, the newborn continued to demonstrate clinical improvement. Changes in CPK, CPK-MB, and CPK-BB levels are shown in Table 1. The patient was discharged without complications.

In both cases, ophthalmologic studies were performed subsequently to detect retrolental fibroplasia, and no evidence of retinal damage was found. Clinical and radiological studies also showed no lung damage as a consequence of HBOT. At six months of age, psychometric development in both babies was assessed by external evaluators and declared normal. Neither patient experienced seizure activity nor evidence of residual neurological damage.

Discussion

HBOT involves the administration of 100% oxygen at greater than atmospheric pressure to increase dissolved oxygen and improve overall oxygenation within tissues. HBOT can reverse local hypoxia by inhibiting post-ischaemic vasoconstriction, thereby decreasing reperfusion injury.[8] Neutrophils have been implicated as the primary culprit in reperfusion injury. Adhering to ischaemic vessel walls, they release proteases and produce free radicals, leading to pathologic vasoconstriction and extensive tissue destruction.[3,4] HBOT not only inhibits neutrophil adherence and post-ischaemic vasoconstriction, but also promotes collagen matrix formation, which is essential for angiogenesis and restoration of blood flow to injured tissue. HBOT will also reduce cerebral oedema.[3,8]

Typically, patients with HIE exhibit a progressive clinical deterioration. However in these two neonates, we identified an unexpected clinical course, with clinical improvement immediately after the first HBOT session, as measured by the Sarnat scale, and their enzyme levels decreased markedly. Normal serum CPK levels in the healthy newborn range from 10–200 U L^{-1}. Levels usually rise within the first six hours after ischaemic injury. If hypoxia is not sustained, CPK levels peak 18 hours after injury and return to normal within two to three days. A serum CK-MB level >92.6 U L^{-1} at eight hours, or >60 U L^{-1} at 24 hours is considered abnormal. Newborns with elevated serum CPK levels within the first six hours of birth should be closely monitored for the development of HIE.[9] The increased levels of enzymes in these two cases decreased more rapidly than expected. This drop in enzyme levels may actually reflect a decrease in a systemic inflammatory process rather than being a specific indication of neurological improvement.

Retinopathy of prematurity is similar to an ischaemic/reperfusion injury. HBOT has been used to manage and prevent ischaemic/reperfusion injury, and short exposures to oxygen pressures of 203 kPa for 45 minutes once or twice a day are unlikely to cause harm. Acute central nervous system oxygen toxicity is rare at 203 kPa or lower, and pulmonary oxygen toxicity is generally not seen with HBOT. The HBOT management scheme used in these two cases, combining both hyperbaric and normobaric oxygen was intended to minimise any risk of toxic side effects. This strategy has been used successfully in newborns with a history of hyaline membrane disease and/or bronchopulmonary dysplasia.[9] No evidence of lung damage or eye damage was identified in these two newborns following HBOT.

Promising clinical evidence of benefit exists for treatment of HIE using mild to moderate hypothermia (33–34ºC) using total body or selective head cooling applied within six hours of birth.[2,10] There are no studies reported comparing controlled hypothermia with HBOT or reports of their combined use.

Conclusion

Two neonates with HIE improved with HBOT consistent with previous reports of its potential benefit. HBOT may represent a viable alternative to, or be combined with controlled hypothermia.

Table 1
Enzyme levels in two neonates suffering from neonatal asphyxia before, during and after a short course of hyperbaric oxygen therapy (HBOT)

		Pre-HBOT	Post-HBOT 1	Post-HBOT 2	Post-HBOT 3
Case 1	CPK total (units)	3069	795	342	493
	CK-MB (units)	58	35	29	41
	LDH (units)	1036	730	645	657
Case 2	CPK total (units)	2156	1230	539	
	CK-MB (units)	77	43	30	

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CME ACTIVITY 2010/4

HBOT AND MALIGNANT OTITIS EXTERNA

Michael Bennett

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

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

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

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

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Background reading
Practitioners are referred to the article in this journal dealing with the POWH experience with malignant otitis externa (Saxby A et al. Malignant otitis externa: experience with hyperbaric oxygen therapy. *Diving and Hyperbaric Medicine*. 2010;4:195-200) and the relevant chapter in Harrison's online (Rubin MA, Gonzales R, Sande MA, "Chapter 31. Pharyngitis, Sinusitis, Otitis, and Other Upper Respiratory Tract Infections" (Chapter). Fauci AS, Braunwald E, Kasper DL, Hauser SL, Longo DL, Jameson JL, Loscalzo J, editors. *Harrison's principles of internal medicine*, 17e: <http://accessmedicine.net/content.aspx?aID=2883531&searchStr=otitis+externa#2883531>. This link may not be free to readers outside of Australia.

How to answer the questions
Please answer all responses (A to E) as true or false.

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

Question 1: Which of the following statements are true in relation to the diagnosis of malignant otitis externa (MOE)?

A. MOE may arise from a chronic infection of the respiratory sinuses.
B. The diagnosis of MOE always implies inadequate treatment of an initial infection in or near the external auditory canal.
C. The onset of MOE is often characterised by a sudden deterioration in general wellbeing and severe otalgia with hearing loss.
D. As implied in the name, MOE is always associated with an underlying malignancy.
E. MOE is often associated with pre-existing conditions that might affect immune function and healing capacity of the tissue.

Question 2: Concerning the organisms associated with MOE...

A. As with other necrotising infections, Clostridial species are commonly isolated in tissue samples.
B. Multiple organisms are quite commonly cultured from tissue samples and wound swabs, and it may be unclear what the primary pathogen is.
C. MOE is readily distinguished from invasive fungal infections because fungal species are never isolated in MOE.
D. In the absence of positive cultures, a reasonable choice of antibiotics would be IV penicillin combined with an oral antifungal agent.
E. The most commonly isolated organism is *Pseudomonas aeruginosa*.

Question 3: Hyperbaric oxygen therapy for MOE…

A. Remains the only treatment modality for which we have randomised evidence of effectiveness.
B. Was first described in a single case report by John Mader in 1952.
C. Has now been reported in more than 50 human cases, although no formal comparative studies have been published.
D. Needs to be given in maximal doses (typically 284 kPa for two hours each session) because the infection is in bone and there is poor delivery of oxygen to the affected area.
E. Is an adjunctive therapy and not a substitute for appropriate antibiotics and surgical debridement as indicated.

Question 4: Concerning the assessment and treatment of MOE…

A. Surgical debridement should be performed at regular intervals to reduce the bacterial load in the tissues.
B. The routine use of timentin and ciprofloxacin in combination has been shown to improve the case fatality rate in pseudo-randomised trials (sequential assignment to therapy).
C. The extent of disease may be assessed by using gallium and technetium-99 scintigraphy studies.
D. The characteristic finding on examination is granulation tissue in the posteroinferior wall of the external canal, near the junction of bone and cartilage.
E. Cranial nerve involvement is sometimes seen, with the sixth nerve most often involved because of the close anatomical relationship between the course of this nerve and the external auditory canal.

Question 5: With regard to the complications and outcome of therapy…

A. Thrombosis of the sigmoid sinus had never been described before it was noted in one case of the POWH series.
B. Complications due to hyperbaric therapy are very uncommon in this patient group.
C. Around two thirds of patients were symptom free at final follow-up in the POWH series, and this clearly indicates that the addition of HBOT is of major benefit.
D. The development of lower (ninth to twelfth) cranial nerve lesions during appropriate antibiotic therapy may be a grave sign.
E. Seizures during HBOT for this group of patients are unlikely to be due to hypoglycaemia because many are diabetics and will have high blood sugars.

Key words
MOPS (maintenance of professional standards), malignant otitis externa

Breathe easy

John Parker

My name is Carbon Die-Oxide
I'm a molecule of fame.
I can keep you healthy or kill you at will
So you'd best not forget my name.
Some say I am heartless
For I can cause terror,
But really I'm a blameless, gameless pawn
For it is usually your error.
I am the one who reminds you to breathe.
Not to do so is dire.
If you doubt it and are bold to hold your breath
I will make you inspire.
When you are really stupid and hyperventilate
My blood levels plummet.
When you free dive your oxygen can drop till you drown
Before I can overcome it.
Diving to great depth does not make sense.
It never seems enough
And at depth your air and commonsense are dense
And breathing is tough.
For I cannot escape and you cannot get enough air
No matter how hard you try.
Your airways collapse, consciousness lapses
And you die.
So treat me with great respect.
Don't hyperventilate or get wheezy,
And don't dive deep on air. Take care
To keep breathing free and easy.

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EUBS European Underwater and Baromedical Society

EUBS 37th ANNUAL SCIENTIFIC MEETING 2011

24–27 August 2011
Gdansk, Poland
First Announcement

Hosts: The National Centre for Hyperbaric Medicine, Gdynia
Venue: The Medical University of Gdansk
Zdzislaw Sicko, Chairman of the Organising Committee
Jacek Kot, General Secretary of the Organising Committee

Main topics:

Diving physiology and medicine, non-dysbaric disorders
Research in deep diving and dysbaric diving disorders
Basic research and clinical hyperbaric medicine
Hyperbaric safety, technology and organisation

There are also plans to have several satellite meetings including:
ECHM Workshop on "HBO in Emergency Medicine"
EBAss meeting
EDTCmed meeting
DAN Divers' Day

Contact the organising Committee at:

EUBS2011, National Centre for Hyperbaric Medicine
Ul. Powstania Styczniowego 9B
81-519 Gdynia, POLAND
Phone: +48-(0)58-699-8650
Fax: +48 58 699-8641
E-mail: <office@eubs2011.org>

Visit our website:

<www.EUBS2011.org>
and register today for EUBS 2011 in Gdansk!

Minutes of EUBS Executive Committee Meeting, 17 September 2010, Point Hotel, Istanbul

Opened: 1100 h

Present: C Balestra, A Brubakk, P Bryson, P Germonpré, P Knessl, A Möllerlökken, P Müller, J Schmutz, P Wooding
Invited: M Cimsit, J Kot, M Sedlar, M Zaric

1 Minutes of previous meeting
Accepted without comments

2 Status of current and future EUBS meetings
2.1 Secretary General of EUBS2010: M Cimsit
2.1.1 The meeting is considered a success, with 262 registrants including accompanying persons, from 32 countries, four continents.
2.1.2 The congress budget is even. The cost of invited speakers and printing is sponsored by a grant from the Office of Naval Research (ONR), USA. Reimbursement to Secretary General will be delayed till October, pending funding availability within ONR. Formal reimbursement request from M Cimsit needs to be obtained for our own book-keeping.
2.2 Presentation of EUBS2011: J Kot
2.2.1 J Kot informs ExCom that the Meeting will be held from 24–27 August 2011 in the University Aula of Gdansk, meaning that organisational costs

will be kept low.

2.2.2 ExCom stresses the importance of accessibility to as many participants as possible, meaning keeping registration, banquet and hotel fees as low as possible; J Kot agrees.

2.2.3 There will be three satellite meetings: DAN Divers Day, EBAss and an ECHM workshop: "HBO in Emergency Medicine".

2.2.4 J Kot agrees that ExCom participates in the organisation of the sessions, with the aim to improve interaction; two members of ExCom will be members of the (international) Scientific Committee. Exchange with these members can be done via the Procom debate communication system, J Kot will send details.

2.2.5 For now, one Keynote lecturer has been confirmed: David Elliott

2.2.6 The 1st announcement has been handed out at this meeting. The Congress Website <www.eubs2011.org> is on line. J Kot will give a five-minute presentation at the GA.

2.3 Presentation of EUBS 2012: M Sedlar, M Zaric

2.3.1 M Zaric (Belgrade) gives ExCom his full assurance that the congress will be well organised; the Hyperbaric Centre in Belgrade, as local organisers, will strive to have as many attendees as possible, with a good scientific as well as social programme.

2.3.2 ECHM will have a Consensus Conference in direct conjunction with the EUBS Annual Meeting. Care will be taken to ensure there will be no conflict between these two meetings. Chronologically ECHM will hold its meeting first and then EUBS.

2.3.3 It is accepted that ExCom plays a greater role in the scientific organization and planning of the congress in order to improve quality. Invited Speakers will be suggested by ExCom, and the local organisers will submit to ExCom their own proposals for invited speakers.

2.3.4 Congress fees will be kept low (350 Euro for early, 400 Euro for late registration). Organisers are ready to assume losses in order to increase participation. A Brubakk stresses the importance of participants' discussion and interaction during the meeting. First announcement to be published in March 2011.

3 Travel grant

3.1 One application for travel grant was received, which was accepted: P Buzzacott (Australia); P. Wooding to action.

3.2 After a discussion on a possible maximum amount to be allocated, ExCom proposes to limit the Travel Grant in the future to a maximum of three grants each year, each for a maximum amount of 800 Euro. It will also be specified that the Travel Grant is to be used for economy airfare (or other transportation), and accommodation only, not other costs (such as poster printing, etc).

4 Zetterström Award 2010

4.1 Committee Members: J Schmutz (EUBS ExCom), Samil Aktas, Günalp Uzun (local scientific committee)

4.2 Only two posters agreed to participate in the Zetterström Award competition.

4.3 P. Germonpre proposes that as a 'statement' and expression of our concern, both posters should be selected. Accepted by ExCom.

4.4 Registration fee will be waived by local organising committee at the next meeting, who should make a formal reimbursement request to EUBS ExCom, for transparency.

4.5 Posters can compete only if they have not been published or submitted previously elsewhere. The winner must have his final paper submitted within 12 months to DHM for consideration of publication. This should be clearly stated in the contest rules. In order to avoid confusion (there was a small tick box on the Abstract Submission form, to be ticked if authors wanted to compete), the Abstract Submission Form will be modified so that everybody competes for the Zetterström award, unless they tick a box if they do not wish to compete. A clear footnote will be placed on the abstract form to explain this procedure. Accepted by ExCom.

5 EUBS ballot

5.1 The internet-based voting system was generally very well accepted as easy and quick, in spite of this, only 91 members responded (35%). The reasons for this are not entirely clear, possibly some members did not receive the ballot form because of their e-mail spam filters. A query will be made during the GA.

5.2 Election Member at Large 2010: one candidate – Jean-Michel Pontier, elected.

Proposal: P. Wooding: check by mail/telephone why people did not vote.

6 Diving and Hyperbaric Medicine Journal

6.1 As from this year on, only abstracts will be published in the EUBS Proceedings Book. This had been proposed by ExCom in 2008, but now applied for the first time. This fulfills the requirements of DHM Editors in order to avoid 'duplication of publication' issues.

6.2 DHM has an Impact Factor (ISI) of 0.49, which is only slightly below that of the UHMS journal (which has been listed for a much longer time). P Mueller will make a separate point of that during his presentation at the GA.

6.3 There was a debate as to the costs of producing the DHM Journal, with annual print runs lower than a few years back, reflecting a slowly declining membership base.

6.4 **Actions:**

6.4.1 Inform membership of high cost of DHM Journal, as well as ecological concerns (printing and sending paper around the world).

6.4.2 Consider an electronic version only – there could be unexpected complications involved in

this method of publication. We need to consider carefully the pros and cons.

6.5 **ICHM:** a letter was received from Mike Bennett (ICHM secretary) requesting the possibility of free advertisement in DHM for the next ICHM meeting in Cape Town, South Africa, in March 2011. Accepted by ExCom.

7 Website

7.1 Not many changes to the website at this time.

7.2 The financial report will be published on the Members-only pages for a limited time.

7.3 The full e-version of *Diving and Hyperbaric Medicine* is also posted on the members-only pages, only the cover and content pages are 'public'.

8 Membership

8.1 EUBS: overall membership is stable; as of today, 91 members have not paid their membership dues. P Bryson proposes that the Membership Secretary contact the non-renewers in order to increase the number of members renewing in time. P Germonpré will ensure Patricia Wooding receives all the automatic renewal messages so she knows when to expect members paying.

8.2 Some members have asked for a discount of EUBS Membership because they would like to keep the 'double' membership of EUBS and SPUMS but want to only receive the Journal once. Although this is a justified concern, there is as yet no real solution, because EUBS does not have a membership category 'without Journal'. It is proposed that EUBS ExCom will discuss this matter with SPUMS Committee to find a common solution.

8.3 Retired members: the Membership Secretary will approach retiring members to inform them that the EUBS is willing to offer them free membership as a retiring member. They should, however, apply again each year for renewal in order to avoid costs due to 'automatically continued invalid memberships' (e.g., death of the retired member).

9 EUBS finances

9.1 The financial report is presented by P Wooding. Auditing of the books has been done by Ms Anna Stillman from DDRC, and has been accepted. ExCom will express its gratitude to Ms Stillman at the GA.

9.2 The financial report reflects the fact that a substantial portion of the EUBS membership fee serves to produce the DHM Journal, a fact that is known and has been addressed above.

9.3 The full financial statements will not be published in DHM but will be available for a limited time on the EUBS website (Members-only section).

10 Miscellaneous

10.1 It is proposed that an official display banner be produced, to be used by the Membership Secretary when present at EUBS Meetings, so she is readily recognisable as such, rather than displaying printed-out A4 pages with the EUBS logo. Such a banner does not represent a major cost and could be designed internally.

Action: A Möllerlökken to examine production possibilities, P Knessl to propose graphic layout to ExCom.

10.2 Proposals to enhance and improve the efficiency and activities of the Society were discussed:

10.2.1 To increase the size of the Society by incorporating new categories of members would need a change in the ByLaws

10.2.2 It is proposed to give more specific tasks to Members at Large:

Member at Large year 3 – responsible for scientific committee and scientific programme of the next EUBS Meeting

Member at Large year 2 – responsible for the discussion forum of the website

Member at Large year 1 – observation.

10.2.3 Create specific Subcommittees: this possibility is explicitly present under the ByLaws of the Society. P Germonpré proposes the creation of a 'Liaison Committee' to be chaired by Member at Large 2007 (P Bryson) for the term of at least one year, with the specific task of interconnecting between organisations and individuals in order to advance scientific work in the fields of diving and hyperbaric medicine. The continuation and possible expansion of this Liaison Committee will be evaluated at the 2011 ExCom meeting.

10.3 The US Office of Naval Research (ONR) sponsoring: ONR granted sponsoring for this year's EUBS meeting but there are considerable delays in payment. P Germonpré will ask for the grant earlier so that it can be received before the meeting actually takes place. EUBS should try to have continuing sponsorship from ONR for future meetings.

11 Proposals to (local meeting) Secretary General:

11.1 Scientific programme, including timing and distribution of posters/oral presentations to be approved by ExCom beforehand

11.2 Choice of invited speakers to be approved by ExCom

11.3 Condition for invited speakers to submit their presentation(s) for publication in DHM

11.4 Work needs to be done to try and ensure ExCom 'assists' those present at meetings to publish their data in an appropriate format. A debate was had as to whether one of the specific invited speakers at the annual meetings could be an 'educational person' to explain the differences between different levels of evidence and how to do work and publish at each level. Not everyone can do RCTs' but there is a lot of information out there which could and should be assessed appropriately and published. This could possibly be done in conjunction with the Journal team.

Closed: 1355 h

Minutes of EUBS General Assembly 2010 Istanbul, 18 September 2010

Opened: 1400 h

1 Welcome

1.1 The President, Peter Germonpré (PG), welcomes all the participants with a promise of a short session, as there are not many controversial issues on the agenda.

1.2 The minutes from the General Assembly 2009 are accepted.

2 Status of the 2010 Annual Scientific Meeting.

2.1 PG thanks the organisers for a well-conducted meeting with excellent, professional technical staff.

2.2 Professor Maide Cimsit, Secretary General of the Annual Meeting, gives her report: there were attendants from four continents, 32 countries, 262 registrants – 92 EUBS members, 62 non-members, 35 nurses and technicians, 19 commercial registrants, 19 accompanying persons and 35 free registrations. There were 70 oral presentations. Of 69 posters submitted, 65 were presented. Prof. Cimsit thanks all her staff for their hard work. PG hands over beautiful flowers to Prof. Cimsit and the rest of the staff.

3 Awards and grants

3.1 Zetterström Award:

There were only two posters submitted for the Zetterström Award competition, which rewards the best poster. The Zetterström Committee (J Schmutz, member EUBS ExCom, Samil Aktas and Günap Uzun, members of the local scientific committee) rewarded both competing posters:

3.1.1 Serkan Ergozen, Senol Yildiz, Hakan Ay and Recai Ogur. *The effects of hyperbaric oxygen treatment on hypoxia inducible factor-1alpha, inducible nitric oxide synthetase and vascular endothelial growth factor levels with diabetic foot*.

3.1.2 Umut Akgun, Maide Cimsit, Baris Kocaoglu, Onur Basci, Selva Zeren, Yesim Saglican, Gulcin Basdemir. *Effect of hyperbaric oxygen therapy (HBOT) combined with microfracture technique on healing of full thickness cartilage lesions*.

3.1.3 The Award (free registration for next EUBS meeting) is basically a research stimulus and represents the possibility to submit to *Diving and Hyperbaric Medicine* (DHM) for peer review.

3.1.4 There are remarks from the audience. People were surprised that not all posters had the chance to compete. The reason is that, because the prize involves submission to DHM, the poster cannot have been submitted to another journal. Two years ago, the Zetterström prize winner was unable to accept his prize because he had submitted it to another journal than DHM. This is the reason why it was explicitly stated on the abstract submission form, and only posters indicating they were willing to compete for the prize were part of the competition. The format of the abstract submission form may have caused some confusion; however, the rules were very clear. Next year, the abstract form will be modified and will allow the authors very clearly to state whether they want to compete or not.

3.2 Travel Grants:

3.2.1 There was one application for the travel grant:

Peter Lee Buzzacott from Australia: *Risk factors for running low on gas in Western Australia*

3.2.2 PG informs the GA that ExCom has proposed that the number of travel grants will be limited to a maximum of three, with a limitation to a maximum of 800 Euros per grant, to be used for economy air fare and lodging only. Applicants will have to submit their presentation to DHM in order to receive the grant. There is no geographical limitation as to the country of origin of applicants.

4 EUBS publications (Journal and Website).

4.1 Peter Müller (PM), European editor of DHM, informs the GA that our Journal has received an Institute of Scientific Information (ISI) Impact Factor of 0.49. As a comparison, the UHMS journal (UHM) has an Impact Factor of 1.04. This is very encouraging. The application for indexing in PubMed has been missed only by a narrow margin this year. Our Journal has been asked to apply again this year. All EUBS members are, therefore, even more vigorously encouraged to submit their research to DHM.

4.2 The DHM journal has its own website (<www.dhmjournal.com>) which is still in a primary phase. It will be expanded soon.

4.3 The EUBS website has not encountered big changes. The full text issue of DHM is available as PDF in the members section, whereas only the cover and contents pages are 'public'.

4.4 Ongoing or recruiting RCTs, with the address of the responsible person, will be accessible in the 'research, courses and events' section of the website. Investigators are encouraged to 'advertise' their ongoing research in this section, by simple e-mail to the webmaster <webmaster@eubs.org>.

5 Financial report

5.1 The financial report was prepared by our membership secretary, Patricia Wooding (PW) and projected to the GA. The audit was done by Anna Stillman from DDRC in Plymouth, who is thanked by ExCom for her generous efforts. The report will be accessible for a limited period of time on the society's website (members-only section)

5.2 The financial report is accepted by the General Assembly.

5.3 As a comment to the report, PG states that the biggest part of the Society's budget goes to the journal.

He informs the GA of the discussion in ExCom on the option to produce only an electronic 'e-journal' and to suppress the paper copy. From the floor, Alf Brubakk explains that in his view, almost 80% of the Society's income is used to "*produce paper and to send it around the world*". He would prefer to use this money to support young scientists, to give them free access to the meetings and to invite high level speakers to help better understand our field. He asks the General Assembly to support the 'e-version only' option. Phil Bryson notes that the Society spends approximately £16,000 per year only for printing and shipping costs and that this also has a negative ecological impact. E-books are now widely accessible, although, as remarked from the floor, not everybody has an e-book reader at hand, and some prefer still to read paper versions rather than PDFs. Alf Brubakk replies that anyone wishing a paper version could very well print out his PDF himself. Jacek Kot asks if members could not choose if they want to have a hard copy instead of the e-journal. Peter Mueller answers that the price of producing only a few copies would be barely affordable. A remark is made that having only an e-version may have an influence on the process of indexing in PubMed, because one of the criteria is the "quality of print".

5.4 PG proposes to organise an electronic ballot on the matter and informs the GA that EUBS ExCom will approach the SPUMS Committee, co-owner of the DHM Journal, with these concerns.

The General Assembly supports this motion.

5.5 As a comment from the floor, it is asked why EUBS still has its accounts in UK pounds sterling, and not in Euro as was announced last year. PG answers that changing the main account to Euros was not possible since EUBS is a UK-based charity; the Euro account is and will still be used, however, and transaction and exchange costs are minimal with the PayPal system.

6 Votes and elections

6.1 The results of the voting ballot were as follows: Member-at-Large 2010, one candidate: Jean-Michel Pontier, elected.

6.2 This year, the voting process was done electronically; this resulted in only a slight increase in returned ballots. 258 ballots were sent out, only 35% were returned. It seems that only a few of the members present did not receive the ballot. In general, the electronic voting process was appreciated.

6.3 Leaving the Committee as Member-at-Large is Phil Bryson. The President expresses many thanks for services rendered with applause from the floor.

7 Future meetings

7.1 Jacek Kot presents the 37th EUBS Meeting 2011 in Gdansk, Poland (PL) and the National Hyperbaric Centre in Gdynia. The Meeting will take place from 24–27 August 2011. There will be three satellite meetings: DAN Europe Divers Day, European Committee for Hyperbaric Medicine (ECHM) and European Baromedical Association for Nurses, Operators and Technicians (EBAss) will be held. The website is already available at <www.eubs2011.org>.

7.2 The 2012 meeting venue in Belgrade, Serbia, has been presented by the local organising committee to ExCom. They left a very good impression.

7.3 The 2013 meeting will be held in La Réunion, it will be aimed at being a joint meeting with SPUMS. ExCom is still awaiting an official reply from the SPUMS President, who expressed his enthusiasm for such a joint meeting previously.

8 Miscellaneous

8.1 PG informs that the ExCom wishes to participate more actively in the preparation of the annual scientific meetings.

8.2 There is a need for the Society to better communicate with the outside. Several scopes of action are proposed: increase the possibilities to join existing multicentric research, increase the world awareness of EUBS, stimulate networking and interaction outside of the meetings. ExCom proposes the creation of a Liaison Committee; Phil Bryson, having finished his term as Member-at-Large is appointed as a liaison officer to ExCom and will be responsible for this new development. His term will be one year, renewable. This proposal is accepted by the General Assembly.

8.3 Questions from the floor:

8.3.1 Eric Janssen asks if it would not be good for the Society to have meetings with other specialties, for instance radiotherapy. PG answers that this was previously done by ECHM, not EUBS, during a consensus conference organised on purpose.

8.3.2 Why are the EUBS Proceedings shrinking over the years? More and more papers are published only as abstracts, not as "mini-papers" anymore.

PG answers that having a full-length paper published in a proceedings book can hinder its subsequent publication in a peer-reviewed journal. Therefore, it was decided two years ago that the proceedings book would be transformed to an abstract and posters book only.

Closed: 1456 h

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 2008, the SPUMS Academic Board consists of:
Associate Professor David Smart, Education Officer
Associate Professor Simon Mitchell
Associate Professor (ret'd) 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

Minutes of the SPUMS Executive Committee Meeting, 23 May 2010 at Berjaya Resort, Redang Island, Malaysia

Opened: 1435 h

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

Apologies: J Lehm, S Squires, C Acott and V Haller

1 Minutes of previous meeting:
Minutes accepted for Executive Committee Meeting, held 21 November 2009. Proposed M Bennett, seconded G Hawkins, carried.

2 Matters arising from previous minutes:
Reviewed action list.

3 Annual Scientific Meetings:

3.1 ASM 2010

3.1.1 Registrant numbers - 93 SPUMS, 59 full registrants, 115 total registrants (Including AHDMA), meeting total 80. To date, AUD9,800 profit. Resort has offered free lunch for conference attendees. G Hawkins thanked Committee for support to amend the Hyperbaric Update component from Thursday to Friday.

3.2 ASM 2011

3.2.1 S Lockley to convene ASM 2011. Quotations discussed. Decision to make hotel booking arrangements independent of travel agent after cost analysis and quote comparison. Venue currently planned is Palau Pacific Resort with Pacific Flier as major carrier. Expected costs were outlined, including airline quote, accommodation quotes and dive operator quotes. Compared with travel agent quotes. Decision supported by the committee for the convenor to make arrangements directly with providers. Website, organised through Cvent and already paid for, will allow delegates to register and pay and book flights and accommodation on line.

3.3 ASM 2012

3.3.1 Discussed possible locations for ASM 2012. Suggestions included Marshall Islands and Palau Tioman. As yet no convenor identified.

Action: Committee members to continue to seek expressions of interest from SPUMS members for convenor.

3.4 ASM 2013

3.4.1 Combined meeting with EUBS is planned, possible locations discussed.

4 Journal matters:

4.1 NLM Technical Committee Report - DHM Editor suggests letter be sent from the Presidents of EUBS and SPUMS expressing concerns about report and addressing specific issues.

Action: M Davis to draft letter for feedback from both ExComs.

4.2 Addition to the Editor's contract.

Action: M Davis to forward wording for approval.

4.3 DHM Journal web site now operating.

4.4 Mail-out problem with March issue. Partial mail out sent again in April 2010. Discussed seeking alternative distribution method. Proposed: M Davis, seconded M Bennett, carried.

Action: M Davis to explore option of sending out electronic journal to SNAP in Europe, for mail out from Europe.

4.5 Advertising in the Journal dependent on setting up secure payment options for advertisers - best method of depositing advertiser's payments into SPUMS account needs to be determined.

Action: M Davis to discuss with Treasurer who is to advise best method.

4.6 Problems with June issue content - more papers required.

4.7 Policy re: ASM papers and difficulties with 2009 ASM presenters submitting - four still have not been submitted. Professor Bruce Spiess has provided three high-quality papers.

5 Website update

5.1 Costs have been reduced from AUD500 per fortnight to AUD100 per month. Journal will not be added to DHM website at this stage as DHM website does not check membership.

5.2 DHM website - pdf articles, core contents and how to subscribe were discussed.

6 President's report

6.1 Discussed formation of International Society of Baromedicine. Item is now closed.

6.2 Diabetic diving courses discussed.

Action: M Bennett to draft policy statement re diabetic diving courses.

6.3 Discussed Australian Standards for diving medicals and recent meeting attended by D Smart. As a result of ISO Standards coming in, and lack of objection from training organisations within Australia and New Zealand, concern expressed by D Smart that the introduced ISO standards, which have lower qualitymay over-ride current ANZ Standards. Three options were proposed as a way forward:

i) Sponsor our own standard through Australian Standards; however, submission would cost AUD13,000 and minimal dive industry support expected;

ii) Develop a separate medical standard for the recreational dive medical; and

iii) Forward our medical to relevant government OHS departments stating this is the new SPUMS Dive Medical and we recommend legislation reflects this. The medical could then be placed on our website and into the DHM Journal.

Action: D Smart to continue exploring options and possible solutions.

6.4 Diving Doctors List –- Current concerns include that the list is not being up-dated. Suggested that the requirement that members must update details before renewing registration be introduced. In addition, a disclaimer should be placed on the list stating that SPUMS is not responsible for quality control of the doctors listed.

6.5 Mailing list has been updated and is now correct.

6.6 AUHMA - no further correspondence received at this stage to further develop the relationship between the SPUMS and SAUHMA.

6.7 Discussed invitation to SPUMS to have a promotion booth at ODEX,at no charge. A link to their website through the SPUMS website has been requested. Proposed: M Bennett, seconded G Hawkins, carried. It was agreed not to link to commercial providers of equipment/travel, etc.

6.8 Discussed separating membership dues from ASM registrations. With new Cvent website, this will not be an issue, as secure pay link is provided.

6.9 Discussed weekly hours for SPUMS Administrator, currently average of 2.5 days per week.

Action: Letter to Administrator requesting outline of costs for EUBS and SPUMS contribution calculations.

6.10 Managing specific complaints about a SPUMS diving doctor. Correspondence sent to a concerned patient with advice provided on mechanism of contacting Medical Complaints Commission in relevant state.

6.11 Discussed possibility of developing reciprocal website links with commercial enterprises - minimal benefit to our society.

6.12 Donation to Rubicon of AUD1,000 to be made. Proposed M Bennett, seconded G Hawkins, carried

6.13 Epilepsy position paper - currently being drafted.

Action: M Bennett

6.14 ICHM request to contribute to DHM Journal. Possibly room for a one-page newsletter.

Action: M Bennett and M Davis

6.15 Declining membership numbers - discussed multiple concerns including loss of EUBS members of SPUMS.

6.16 Web conferencing <http://www.umeeting.com/> - to trial as a meeting tool when enough committee members are available.

7 Education officer's report

7.1 EUBS and SPUMS Diving Doctors list –- discussed above.

7.2 Australian Standards for Dive Medicals –- discussed above.

7.3 Report re Medline and Australasian College for Emergency Medicine – registrar papers in *Diving and Hyperbaric Medicine* will be recognised by the College for training purposes without Medline citation.

8 Treasurer's report

8.1 In absence of Treasurer, discussed concern with discrepancy between income generated by subscriptions, and expected income based on member numbers provided by SPUMS Administrator. A special meeting was held to discuss this and the SPUMS Administrator was invited in to further explore the discrepancy. Subsequently, the President requested a full investigation of the current membership for this year and the preceding years, through accessing information from the previous website provider and from Secure Pay, as well as SPUMS Membership databases.

Action: M Bennett to liaise with S Goble, G Hawkins and J Lehm to locate the relevant information and undertake an investigation. A report on this matter will be generated and provided to the SPUMS membership in the DHM Journal.

8.2 Recommendation that DHM Journal costs be fully ascertained and members charged appropriately for the actual costs of the production of the Journal. Treasurer has expressed concern that currently SPUMS is not covering the costs of *Diving and Hyperbaric Medicine* fully with subscription charges.

9 Secretary's report

9.1 Contact details for the committee members are currently correct.

10 Other business

Nil

11 Correspondence

Nil

12 Next meeting

To be scheduled around RAN MOUM Course, November 2010

Closed: 1936 h

South Pacific Underwater Medicine Society 40th Annual Scientific Meeting

22–27 May 2011

Venue: Hilton Resort and Spa, Tumon Bay, Guam

Call for Abstracts, Conference Information and Registration Forms

Themes:
Medical aspects of military, occupational and recreational technical diving
Head injury and diving workshop – review of clinical cases and guidelines
Management of acute diving injuries

The Head Injury and Diving Workshop will include medical risk assessment for diving post-head injury and post-seizure. Current guidelines will be examined and specific cases reviewed. Management of acute diving injuries will also be covered in the workshop forum.

Keynote speakers:
David Doolette, PhD, US Navy Experimental Diving Unit, Panama City, USA
Simon Mitchell, PhD, FANZCA, The University of Auckland
Andrew Fock, FANZCA, The Alfred Hospital, Melbourne

Abstracts:
Abstracts for presentations should be submitted before 31 March 2011 as a Word file of up to 250 words (excluding references – 4 only) and with only one figure.

Intending speakers are reminded that it is SPUMS policy that, wherever possible, their presentation should be submitted for consideration of publication in *Diving and Hyperbaric Medicine*.
The Editor will contact speakers prior to the meeting.

Papers should preferably reflect the themes of the conference. However, all free papers relevant to diving and hyperbaric medicine will be considered.

If you wish to present a paper please contact:
SPUMS ASM 2011 Convenor:
Dr Sarah Lockley
E-mail: <spumssecretary@gmail.com> or <secretary@spums.org.au>
Mobile: +61-(0)4-3114-4817

Register via the SPUMS website <www.spums.org.au>
or contact the Convenor for a Registration Brochure
Registrations not done via the website will incur a handling fee

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 4 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 6 months' full-time equivalent experience in a hyperbaric unit and successful completion of a thesis or research project approved by the Assessor, SPUMS
   b **and** Completion of a further 12 months' full-time equivalent clinical experience in a hospital-based hyperbaric unit which is approved for training in Diving and Hyperbaric Medicine by the ANZCA.

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

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

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

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

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

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Important notice: New Continuing Professional Development Coordinator needed for 2011

Associate Professor Michael Bennett has recently been appointed as the Chief Examiner for the ANZCA Certificate in Diving and Hyperbaric Medicine. He is advised by the College that he will not be able to continue to produce the CPD exercises in future issues of *Diving and Hyperbaric Medicine* due to a potential conflict of interest.

Therefore, we are seeking a volunteer to continue these CPD exercises. Those interested should contact the Editor.

ANZCA SIG members: Time to front up and support your College, this Journal and your colleagues!

Michael Davis
E-mail: <Editor@dhmjournal.com>

International Congress on Hyperbaric Medicine

President: *Dr Frans Cronje*
Executive Director *Dr Alessandro Marroni*
Secretary: *Associate Professor Michael Bennett*

The ICHM is a world-wide organisation, with minimal formal structure, entirely dedicated to hosting an international scientific congress every three years with the purpose of improving understanding among the international hyperbaric community. The *First International Congress on Hyperbaric Medicine* was held in Amsterdam in 1963, under the auspices of the Founding President, Professor Boerema. Since then, the Congress has been held all over the world and the 17th Congress in 2011 will be our first visit to Africa.

Communication among members is through the publication of a newsletter *Oxygen* and a website at <www.ICHM.net>.

With the endorsement of SPUMS and EUBS, a regular page of news and information about the ICHM, of which this is the first, will appear in *Diving and Hyperbaric Medicine* (DHM). We welcome comments from readers, whether or not they are members of the ICHM.

Also under negotiation is the possibility of publishing the Proceedings of each Congress under the *Diving and Hyperbaric Medicine* banner – watch this space. Members of the ICHM who wish to publish scholarly articles are encouraged to consider submission to DHM. All submissions will be subject to the Journal's peer review process.

For all enquiries contact Mike Bennett:
E-mail: <m.bennett@unsw.edu.au>

The 17th International Congress on Hyperbaric Medicine
Cape Town, South Africa
16–19 March 2011

On behalf of the Organising Committee of the 17th International Congress of Hyperbaric Medicine, we would like to invite you to attend the congress in Cape Town, South Africa

We believe we have crafted an exciting and dynamic programme which will include:

- Critical reviews on and identification of shortcomings of the current evidence base for the practice of hyperbaric medicine
- Update on the absolute and relative contra-indications to hyperbaric oxygen therapy
- Overview of essential occupational medical principles and practice
- Diving medical examinations
- Hyperbaric staff and patient issues in hyperbaric centres
- Update on saturation diving, including a review of current occupational medical evidence
- Hyperbaric centre medical director's safety responsibilities
- Practical issues surrounding wound care
- Acrylics - the facts and fallacies about viewports and windows
- Chamber safety and risk management for hyperbaric facilities

Exhibitors: Sponsorship opportunities and exhibition packages are still available

Welcome to Cape Town: Cape Town ranks as one of the world's most beautiful cities, and on our doorstep are a wide array of attractions. We believe that a truly unique and wonderful experience awaits you and your partner. A wide variety of accompanying persons tours and sightseeing tours will be available to showcase the city and surrounding attractions.

Airfares: Specially rebated airfares have been negotiated for the conference

Accommodation: The organisers have arranged a range of discounted accommodation, with many hotels just a short walk from the conference venue.

For all enquiries and registration, visit the Conference website:
www.acitravel.co.za/ichm2011/
Frans Cronje, Convenor
<info@ichm.co.za>

Inter-university Diploma in Diving and Hyperbaric Medicine, France

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

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

Oxygen and Infection; European Committee for Hyperbaric Medicine (ECHM) Conference Proceedings

Free video lectures from the May 2009 Stockholm meeting are available for your iPhone or computer
<www.hyperbaricoxygen.se>

5th Karolinska Postgraduate Course in Clinical Hyperbaric Oxygen Therapy
14 lectures on fundamental concepts and front-line knowledge in the clinical use of HBO.

ECHM Conference 'Oxygen and Infection'
22 lectures and three panel discussions are available on topics such as necrotizing fasciitis and the diabetic foot.

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

The Hyperbaric Research Prize

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

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

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

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

Course content includes:
- History of hyperbaric oxygen
- Physics and physiology of compression
- Accepted indications of hyperbaric oxygen (including necrotising infections, acute CO poisoning, osteoradionecrosis and problem wound healing)
- Wound assessment including transcutaneous oximetry
- Visit to HMAS Penguin
- Marine envenomation
- Practical sessions including assessment of fitness to dive

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

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

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>

DIVING HISTORICAL SOCIETY AUSTRALIA, SE ASIA

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

Instructions to authors

(Short version, updated November 2010)

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 on disk or as attachments to e-mail. The preferred format is Microsoft® Office Word 2003. Paper submissions will also 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: an 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 or single-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. Including more than five authors requires justification, as does 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 less than 40 words in length.

Illustrations, figures and tables must NOT be embedded in the wordprocessor document, only their position indicated. No captions or symbol definitions should appear in the body of the table or image.

Table data may be presented either as normal text with tab-separated columns (preferred) or in table format. No gridlines, borders or shading should be used.

Illustrations and figures should be submitted as separate electronic files in TIFF, high resolution JPG or BMP format. If figures are created in Excel, submit the complete Excel file. Large files (> 10 Mb) should be submitted on disk.

Photographs should be glossy, black-and-white or colour. Colour is available only when it is essential and will be at the authors' expense. Indicate magnification for photomicrographs.

References

The Journal reference style is the 'Vancouver' style (Uniform requirements for manuscripts submitted to biomedical journals, updated August 2009. Website for details: <http://www.nlm.nih.gov/bsd/uniform_requirements.html>). References must appear in the text as superscript numbers at the end of the sentence after the full stop.[1,2] The references are numbered in order of quoting. Index Medicus abbreviations for journal names are to be used (<http://www.nlm.nih.gov/tsd/serials/lji.html>). 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.

Manuscripts not complying with the above requirements will be returned to the author before being considered for publication.

Consent

Studies on human subjects must comply with the Helsinki Declaration of 1975 and those using animals must comply with National Health and Medical Research Council Guidelines or their equivalent. A statement affirming Ethics Committee (Institutional Review Board) approval should be included in the text. A copy of that approval (and consent forms) should be available if requested.

Copyright

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

Full instructions to authors (revised June 2010) may be found on the DHM Jounal, EUBS and SPUMS websites.

DIVER EMERGENCY SERVICES PHONE NUMBERS

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

NEW ZEALAND
0800-4DES-111 (in New Zealand, toll-free)
+64-9-445-8454 (International)

SOUTH-EAST ASIA
+852-3611-7326 (China)
010-4500-9113 (Korea)
+81-3-3812-4999 (Japan)

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

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

UNITED KINGDOM
+44-07740-251-635

USA
+1-919-684-9111

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

DAN Asia-Pacific DIVE ACCIDENT REPORTING PROJECT

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

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

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

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

DIVING INCIDENT MONITORING STUDY (DIMS)

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

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

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