Diving and Hyperbaric Medicine

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The physiology of apnea diving to depth

Hyperbaric oxygen for mandibular cysts

Pre-dive exercise and intravascular bubbles

Performance and safety of a middle-ear implant

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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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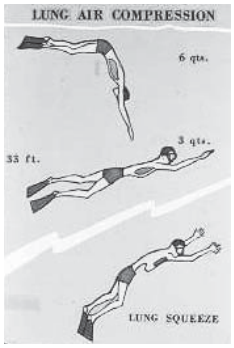
The Editor's offering

As a university student in the early 1960s, I recall breath-hold diving to 30 metres' sea water (msw) depth with a friend (checked on our depth gauges) off the Kyrenia Coast in northern Cyprus; we were very proud of our skills. I have blanked out the depth in a teaching slide from that time (reproduced below), as it is embarrassing to think that our understanding of the physiology was so basic. Professor Schagatay, in three excellent review articles, has brought us up to date and demonstrated the amazing complexity of the physiology of breath-hold diving.[1–3] Before you read Part III in this issue, I strongly urge you to re-read the first two parts in order to understand this complexity better.



Professor Schagatay would be the first to admit that there is still much that we do not understand, and teams such as hers are to be commended for doing the field work necessary with elite divers. Gaining the trust and cooperation of high-performance athletes in any field is a major challenge, so the fact that physiologists undertaking research at international championships is now the norm is very gratifying. It also reflects positively on the participating sportsmen and women who clearly are eager to understand better the physiological challenges they face in what many diving physicians still regard as a sport for suicidal idiots. New technology that will provide in-dive physiological data is being developed and will help to answer some of the outstanding questions relating to human apneic diving.[4] This is very much an endeavour for which the aphorism is 'watch this space'.

I used to run a teaching session on diving and hyperbaric medicine for the residents and teaching staff in the department of anaesthesia. Most times, we ran a demonstration dive to 50 msw with volunteers who were 'diving fit'. On one occasion many years ago, one of my senior colleagues jumped on his bike straight after the dive and pedalled off at his usual furious pace. After a short distance, he became so dyspnoeic that he had to rest for a while, then continued slowly for the remainder of the journey. Gradually the dyspnoea wore off, but he said he felt very tired for the rest of that day. This is a classic description of the 'chokes' and he was fortunate not to get into more serious trouble. The clear message is that vigorous exercise post-dive is not good for you.

The data on the potential impact of pre-dive exercise, however, is conflicting and its impact on decompression sickness (DCS) is by no means clear. Dr Jurd and her colleagues add further diversity to this confusing picture by demonstrating that both the timing and the nature of the exercise may be important in relationship to bubble scores post-dive.[5] As they point out in their discussion, "*the exact mechanism(s) of any protection afforded by exercise is unknown at present, but is likely to be multi-factorial. To have a major impact on diving 'safety', reductions in Doppler grades that represent a substantial reduction in gas load must be demonstrated, as high numbers of VGE* [venous gas embolism] *are associated with an increased risk of DCS. The protective effect may be too small or too variable to allow more stressful dives to be carried out with improved safety*." Again, watch this space.

Oxygen toxicity seizures can be a frightening experience for all involved, including other patients in a multiplace at the time. However, if well managed by trained hyperbaric professionals, this potentially serious side effect of a treatment with a therapeutic ratio of less than one should result in minimal or no morbidity. The incidence is low, but, as more data have been published, the level of risk one needs to advise patients when obtaining informed consent has increased. A decade ago, I used to quote 1:5,000 treatments, but Dr Banham, in his meticulous review of 20 years' events, reports a rate of 1:1,719 at a treatment pressure of 243 kPa.

For 2012, we estimate that the overall cost of the Journal for SPUMS and EUBS will only increase slightly compared to 2011. Because of falling membership numbers, this represents a rise of over 10% as a proportion of your membership dues. The executives ask that, in 2012, every member tries to enrol just one new member into one or other Society; we believe the benefits, particularly a subscription to this Journal, are well worth the modest price of membership.

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Michael Davis

Photo of Lotta Ericson and Linda Paganelli performing a 'dynamic apnea without fins' (DYN) was taken by Jacques de Vos, a professional underwater photographer from South Africa, whilst himself performing a breath-hold.

The President's page

Sorry

"*Peter, are you all right? You are not suffering from depression, are you?*" I apologise for giving this impression – see the September *Presidents' Pages*, and I would like to thank the many friends who expressed their genuine concern for my psychological condition. I am fine, thank you. Maybe I was in a bit of a dark mood at the time I wrote that, but hey, aren't we all entitled to have our ups and downs ? Besides, I was right, wasn't I? Belgium still doesn't have a federal government (as I write this, we have passed the 500-day mark!), new environmental, political and geological disasters have happened since – as they always will, and illness and death of friends and relatives will still occur – that's how life goes...In any case, your concerns were heart-warming, so even if I was unnecessarily pessimistic at the time, I now feel much better because of you. Thanks.

Happy

News: conflicts (especially psychological ones) can be solved. From time to time, it is possible to effectively straighten out differences between people – sometimes dating from a long time back. I clearly state the obvious by saying that this requires an effort from both sides; however, to wait for the other party to take a first step toward reconciliation is not a good idea. Nor is careful strategic planning of such a reconciliation meeting; doing so actually prevents one from listening to the other party's arguments – the main reason why these meetings do not succeed!

For what it's worth, here is a recipe, not foolproof, of course, but it has worked for me (at least once). Before the meeting, analyse what went wrong in the first place. You will find that most of what went wrong can be attributed to the other party. Then, find out (if you do not know already) how the other party sees that same situation (no doubt, he/she will blame you).

Next, prepare to take responsibility. This is no doubt the most difficult phase, but at the start of your reconciliation meeting, you should state "*I am sorry for what has happened and I feel responsible for it. I understand how you feel and I should have handled this situation differently*". Moreover, you should actually be sincere about it (if you cannot be, then question yourself whether you want this conflict solved in the first place). After that, listening again to the arguments of the other party comes much easier, and there will soon be a moment when you can explain your own view of the conflict. Chances are you come to an agreement or at least agree there are still differences of opinion, without this impeding your communication.

Eager

As you read this, the final days of 2011 are nearing. Time to 'wrap up'? I don't know about you, but I already have a lot of projects waiting for 2012. Some of them involve family, some of them have to do with this Society and this Journal. I can't wait for them to start happening, and I sincerely hope you have the same feeling.

Sorry – happy – eager; this seems to me a good summary of 2011. Let's make 2012 an exciting year!

Cheerz

Peter Germonpré
President EUBS

Original articles

The effect of pre-dive exercise timing, intensity and mode on post-decompression venous gas emboli

Karen M Jurd, Julian C Thacker, Fiona M Seddon, Mikael Gennser and Geoffrey AM Loveman

Abstract

(Jurd KM, Thacker JC, Seddon FM, Gennser M, Loveman GAM. The effect of pre-dive exercise timing, intensity and mode on post-decompression venous gas emboli. *Diving Hyperb Med*. 2011 December;41(4):183-188.)

Introduction: The effect of pre-dive exercise on post-decompression venous gas emboli (VGE) remains contentious. The aim of our study was to investigate the effect of timing, intensity and mode of exercise before diving on post-decompression VGE production.

Methods: Fifteen male volunteers performed three identical 100 min chamber dives to 18 metres' sea water. Two of the three dives were conducted with prior exercise at 24 or 2 h; a dive without prior exercise formed the control. Moderate-intensity impact exercise consisted of jogging on the spot for one minute followed by ten star jumps, repeated for a total of 40 min at 70% of maximum heart rate. Post-dive Doppler monitoring began within 2 min of surfacing and was carried out for at least 180 min. VGE were assessed using the Kisman-Masurel (KM) code and the Kisman Integrated Severity Score (KISS).

Results: The median peak KM grade for each condition following the dives was not significantly different. Pre-dive exercise at 2 h resulted in a significant reduction in the mean KISS compared to the control (11.3 versus 17.2, $P < 0.04$, Wilcoxon sign-ranked test). Moderate-intensity jogging/star jump exercise used in this series of dives resulted in significantly lower mean KISS (11.3 versus 21.8, $P < 0.04$) and median KM grade over 180 min ($P < 0.006$, Mann Whitney U test) compared to high-intensity cycling exercise used in our previous study.

Conclusions: This study suggests that moderate-intensity impact exercise reduces VGE production when conducted 2 h prior to diving.

Key words

Doppler, diving, exercise, bubbles, decompression illness

Introduction

The effect of pre-dive exercise on post-decompression venous gas emboli (VGE) remains contentious. It was thought for many years that exercise before, during, or after diving, was an additional risk factor for decompression sickness (DCS).[1] However, a number of studies conducted by two main groups over the last few years have shown that this may not be the case and that exercise prior to diving may actually help to reduce bubble formation and the incidence of DCS. There are, however, differences in the results of these studies with respect to the timing, intensity and mode of the exercise conducted.[2–10]

Wisloff et al. demonstrated that a single bout of high-intensity aerobic exercise performed by rats on a treadmill 20 h before a chamber dive reduced VGE formation and gave protection from lethal DCS.[2] Scheduling appeared to be important, protection occurring only if the interval between exercise and the subsequent dive was 10–20 h. Dujic et al. then demonstrated in man, that a single bout of high-intensity exercise (treadmill running) 24 h before performing a chamber dive to a depth of 18 metres' sea water (msw) significantly reduced the amount of VGE in the pulmonary artery compared to no exercise.[3]

Further studies in rats found the same high-intensity treadmill exercise starting 2 h before a dive either increased or had no effect on VGE formation and eliminated the protection afforded by exercise 20 h prior to diving.[4–6] Contrary to this, a study in military divers found that medium-intensity running starting 2 h before a chamber dive to 30 msw decreased VGE formation.[7] This was repeated using high-intensity running at a controlled heart rate, which resulted in the same outcome.[8] The same dive profile was then performed in open-water, with medium- or high-intensity cycling 2 h prior to diving; both intensities reduced bubble grades.[9] Furthermore, Castagna et al. have recently found that 45 min of treadmill exercise starting just one hour before an open-water dive also reduced bubble grades.[10]

These apparent contradictions in the effect of pre-dive exercise timing led to our study. Our previous series of dives were preceded by exercise which mimicked the high intensity and duration used by Dujic et al., but substituted treadmill running with low-impact cycling.[3,11] The exercise was performed at 24 h or 2 h before a chamber dive to 18 msw, but showed no benefit in reducing VGE compared to the no-exercise controls. As a continuation of our study, the present series of dives examined the effect of reducing the intensity and increasing the impact of the exercise on VGE production following identical dive profiles.

Methods

The study was approved by the UK Ministry of Defence Research Ethics Committee and conducted in accordance with the principles of the Declaration of Helsinki.[12]

SUBJECTS

Fifteen male volunteers, aged 22–53 (mean 36.5) years, participated in the study. They comprised Royal Navy (RN) divers and QinetiQ staff with mixed wet- and dry-diving experience, all of whom had passed their dive medical, involving a fitness test. The purpose of, and procedures and risks associated with the study were explained and the volunteers gave their written consent. Each subject's height and weight were measured and their body mass index (BMI) calculated. Their percentage body fat was measured by bioelectrical impedance analysis using a Bodystat 1500™.

HYPERBARIC EXPOSURES

The study was carried out at the QinetiQ Hyperbaric Medicine Unit, Royal Hospital Haslar, Gosport, UK, a Category 1 facility containing an RN Type A recompression chamber. The chamber air dives were to 18 msw with a bottom time of 100 min. Decompression stops were at 6 msw for 5 min and 3 msw for 15 min, with an ascent rate of 15 msw min^{-1} in accordance with RN Table 11-Mod.[13] Each subject conducted three dives; two were conducted with exercise bouts at 24 or 2 h pre-dive and a dive with no prior exercise formed the control. The order in which the exercise or control dives were conducted was randomly allocated and each dive commenced at exactly the same time each day (1300 h) to avoid any influence of circadian effects. No flying or diving was permitted for at least seven days before commencing the trial and there were at least seven days between the experimental dives. Alcohol and caffeine were prohibited from the evening of the preceding day, but the subjects were free to eat breakfast and lunch on the day of their chamber dives.

This series of chamber dives formed a continuation of our study examining the effect of pre-dive exercise on VGE formation. The same dive profile, period between dives and timing and duration of pre-dive exercise were used throughout. The only difference was in the mode and intensity of exercise conducted: medium-intensity impact exercise (described below) compared to previous high-intensity cycling.

EXERCISE REGIMEN

The exercise regimen for this series of chamber dives consisted of jogging on the spot for 1 min followed by 10 star jumps, repeated for a total of 40 min. No exercise was permitted for 48 h before a dive or exercise bout. Participants were fitted with a Polar™ heart rate monitor and after a brief warm-up period they were asked to aim at 70% of their theoretical maximum heart rate (220 - age (in years) beats min^{-1}) for the exercise period.

DOPPLER MONITORING

Pre-cordial Doppler monitoring of VGE was carried out using a continuous-wave Doppler Bubble Monitor (Techno Scientific Inc., TSIDBM 9008) with the subject standing at rest. Pre-dive baseline monitoring was carried out shortly before the dives. Post-dive monitoring began within 2 min of surfacing and was carried out every 5 min for the first 30 min and every 15 min thereafter, up to 180 min. Subjects were asked not to depart before their Doppler VGE score was declining and so, on a few occasions, monitoring was continued beyond 180 min, but only data collected up to 180 min were used in the analysis. Subjects remained at rest for the whole of the monitoring period. VGE were scored using the Kisman-Masurel (KM) code and the Kisman Integrated Severity Score (KISS) was then calculated to give a linearised measure of VGE.[14,15] Doppler technicians were blinded to the order of the exercise and control dives and were assigned to the monitoring of the same subject for each of their three dives. The Doppler technicians each had several years of experience of audio Doppler monitoring and undertook regular quality assurance assessments. Monitoring sessions were recorded so that they could be re-analysed at a later time if required, using an Archos 605 portable media player, which directly encoded the audio signal to Waveform Audio File Format sampled at 44,100 Hz.

STATISTICAL ANALYSIS

Subject variables are presented as mean and standard deviation (SD). Individual peak Doppler KM grades for

Table 1
Subject demographics

Subject	Age (years)	BMI (kg m^{-2})	Body fat (%)
1	47	28.7	19.1
2	41	26.3	23.2
3	49	25.4	18.7
4	53	25.1	21.1
5	37	25.1	21.1
6	36	28.1	19.4
7	32	24.8	16.5
8	36	28.1	19.4
9	33	24.7	16.4
10	30	25.0	16.4
11	32	27.5	21.1
12	25	25.3	18.5
13	22	24.7	12.6
14	39	26.2	16.1
15	35	29.3	23.6
Mean (SD)	36.5 (8.5)	26.3 (1.6)	18.8 (3.0)

Table 2
Doppler bubble detection

Doppler bubble detection	Control (no exercise)	Exercise at 24 h	Exercise at 2 h
Median peak KM grade (Range)	3 (0–3-)	2 (0–4-)	2 (0–3+)
Time to median peak KM grade (min)	90	82.5	90
Mean KISS (over 180 min period)	17.2	13.1	11.3

Figure 1
KISS for control dives versus pre-dive exercise at 2 h (bars represent mean value); $P < 0.04$

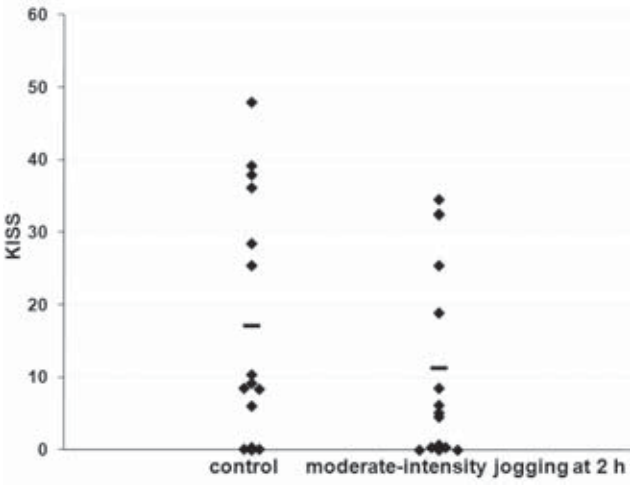
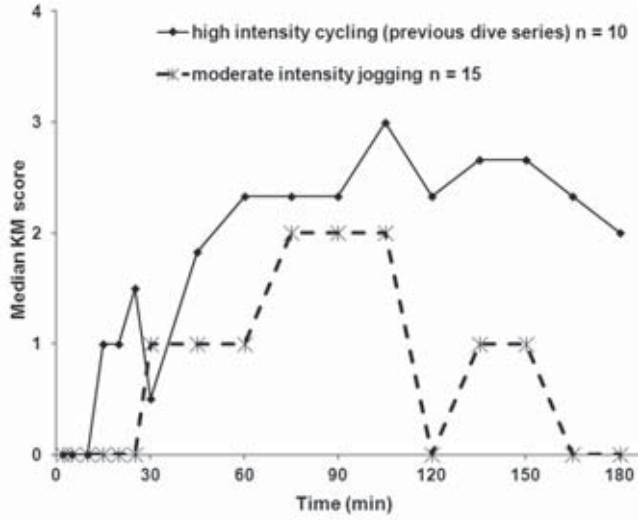


Figure 2
Median KM grades up to 180 min for moderate-intensity jogging versus high-intensity cycling performed 2 h prior to the dive; $P < 0.006$



control and pre-dive exercise dives were compared using the Freidman test. The KISS for pre-dive exercise at 24 or 2 h was compared to the control using the Wilcoxon sign-ranked test. For comparison with our previous series of dives, differences in subject variables were tested using an unpaired t-test. Median KM grades and KISS over the Doppler monitoring period were compared using a Mann Whitney U test. Differences were considered significant if $P < 0.05$.

Results

No DCS occurred in any of the subjects following the dives. Details of individual subject age, BMI and percentage body fat are shown in Table 1.

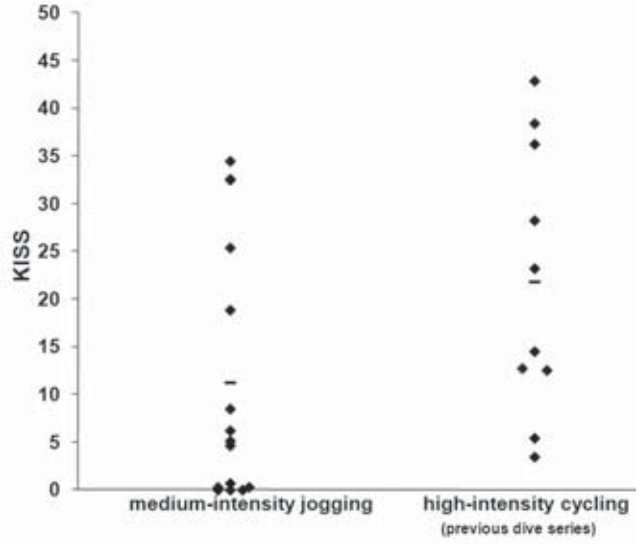
Details of the Doppler measurement of VGE are shown in Table 2. The median peak KM grade decreased from 3 for the control dives to 2 for those dives with pre-dive exercise at either 2 or 24 h, but this decrease was non-significant. The time to reach the median peak KM grade was similar for all dives. The mean KISS up to 180 min post-dive was lower with pre-dive exercise, but was only significantly different from the control when exercise was conducted at 2 h prior to diving (11.3 versus 17.2, $P < 0.04$, Wilcoxon sign-ranked test). The individual KISS following the control dives and pre-dive exercise at 2 h are shown in Figure 1.

This series of dives was a continuation of our study examining the effect of pre-dive exercise on VGE production. There was no significant difference in the median KM grades over the Doppler monitoring period for this and our previous series of control dives. This allowed the effect of the different mode and intensity of pre-dive exercise to be compared. Changing from the high-intensity, low-impact cycling used previously to moderate-intensity impact exercise resulted in a reduction in median KM bubble grades over the monitoring period when conducted at 2 h ($P < 0.006$, Mann Whitney U Test, Figure 2), but not 24 h, prior to diving. Similarly, the KISS was only different between the exercise conditions when performed 2 h prior to the dive (11.3 versus 21.8, $P < 0.04$, Mann Whitney U Test, Figure 3). There were no significant differences in subject variables.

Discussion

Recent studies have demonstrated that prior exercise can reduce the number of VGE and the incidence of DCS following a pressure exposure.[2,3,7–10] The peak Doppler bubble grade is often used for comparing the decompression stress between dives. However, some studies have used limited Doppler monitoring, with measurements at four time

Figure 3
KISS for moderate-intensity jogging versus high-intensity cycling performed 2 h prior to the dive (bars represent mean value); $P < 0.04$



points in some and as few as only two in others, making it difficult (or impossible) to know whether a peak had been reached. Furthermore, the duration of the monitoring has been as short as 60 min, which may have missed VGE produced in those with a long latency of bubble evolution. Indeed, following our present dives the maximum latency to VGE detection was 120 min.

We monitored VGE for at least 180 min, from first appearance until a peak was reached and then until bubbles either declined or disappeared, totalling a minimum of 17 measurements for each subject for each dive. No significant difference was demonstrated between the peak KM grades for the control dives and those with prior exercise at either 24 or 2 h. However, a single peak Doppler grade gives no information as to the grades over the whole of the monitoring period. The KISS integrates VGE over time, providing a more complete picture of bubble activity and gas load than a single peak KM grade.[15] We found no significant difference between the KISS for control dives and those with pre-dive exercise at 24 h. However, the KISS was significantly reduced when exercise was conducted 2 h prior to the dive.

The benefit of this pre-dive exercise at 2 h, but not at 24 h, led us to question whether the results of our previous series of dives could be explained in terms of the intensity/mode of exercise. The only difference we found was a significant reduction in bubbles in changing from the previous high-intensity cycling to moderate-intensity jogging when conducted 2 h prior to a pressure exposure. This suggests that exercise mode/intensity does indeed have an effect if conducted this close to a dive.

The majority of published results on the effect of pre-dive exercise on post-decompression VGE have come from two main groups, showing benefit at either 24 h or 2 h before diving. It is important that independent studies are conducted so that confidence in the results of such studies is robust. Moreover, if there is a common mechanism involved it will not be influenced by factors that may be peculiar to a particular research group. Our previous dive series used the same exercise intensity and duration as that used by Dujic et al. 24 h before diving, but changed the exercise mode from running to cycling and examined its effect at 24 h or 2 h prior to diving.[3,11] This did not result in a reduction in VGE and in our present dive series there was no benefit from medium-intensity jogging on the spot (which can be considered as similar in impact to treadmill running) when conducted 24 h prior to diving. Thus, it may be that only the combination of high-intensity and running (impact) exercise is effective in reducing VGE when conducted 24 h prior to a dive, as reported by Dujic et al.[3]

For pre-dive exercise conducted at 2 h, both medium- and high-intensity running and cycling have been reported to reduce VGE.[7–9] However, in contrast, our previous dive series showed that high-intensity cycling at this time did not reduce VGE formation and others have found no benefit from high-intensity running this close to a dive.[2–6] The results of our present dive series confirm the earlier results of Blatteau et al. by demonstrating that moderate-intensity jogging on the spot 2 h prior to diving reduces VGE formation.[7] An interesting recent finding is that a period of whole-body vibration 1 h before diving decreased VGE formation.[16] Vibration may have a similar action to impact exercise and this may account for the beneficial effect we observed with higher-impact exercise such as jogging and jumping, while we observed no such benefit for low-impact cycling.

When conducting a series of investigations it is desirable to have a level of confidence in the reproducibility of VGE production following control dives. In our study, there was no significant difference in the level of VGE between our present and previous series of control dives. However, reproducibility has been highlighted by others as being problematical. Studies in rats weighing less than 300 g produced few bubbles on some occasions, but many on others (median Doppler grade 0 versus grade 4).[4,5,17] Similarly in man, control dive profiles which had been chosen to reproducibly produce significant Doppler grades resulted in very low bubble grades (median peak grade 0) in a study which had previously resulted in a median peak grade of 3.[3,18] This lack of reproducibility in VGE following control dives leads to the suspicion that dives with prior exercise may also produce differing results on different occasions. Thus, variability in VGE in studies with relatively small subject numbers may be responsible for some of the differences between studies.

Gas bubbles produced following a pressure exposure are thought to grow from micronuclei present in tissues and crevices on blood vessel walls. Muscle activity can induce micronuclei, and microbubbles have recently been demonstrated in the leg muscles of human subjects after exercise on a cycle ergometer, which decayed over time following cessation of exercise.[19] The half-life of intravascular exercise-induced micronuclei was in the order of one hour in another study.[20] Some forms of exercise may act to dislodge micronuclei from the vessel surface and increased blood flow during exercise may cause 'wash-out' of bubble micronuclei from the endothelial cell surface. Removal of such micronuclei before a pressure exposure would have obvious benefits in terms of VGE production, but the net effect will be the difference between their formation and elimination. For exercise close to a dive, the effect may be different depending on the duration of the dive.

If there is a simple common mechanism for the effect of exercise before diving per se it would seem reasonable that studies would agree on timing and exercise mode and intensity. The exact mechanism(s) of any protection afforded by exercise is unknown at present, but is likely to be multi-factorial.[2,21–23] To have a major impact on diving 'safety', reductions in Doppler grades that represent a substantial reduction in gas load must be demonstrated, as high numbers of VGE are associated with an increased risk of DCS.[24,25] The protective effect may be too small or too variable to allow more stressful dives to be carried out with improved safety. Some apparent disparities between studies in the timing and intensity/mode of exercise can be explained, but others remain unresolved. Perhaps commonality should be sought in developing an approach for larger scale trials using consistent dive profiles and full Doppler monitoring, which may then lead to a consensus on the benefit of exercise before diving.

Conclusion

This study suggests that moderate-intensity impact exercise reduces VGE production when conducted 2 h prior to diving.

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Conflict of interest: none

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Telemedicine in the management of diving accidents: correlation of phone-assessed symptom severity with clinical findings

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Abstract
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Introduction: The object of this study was to evaluate to what extent the severity of decompression illness (DCI) assessed by a diving medicine specialist over the phone correlates with actual clinical findings.
Methods: The phone protocols of calls received by a diving medical hotline between January 2008 and December 2009 were analysed. Each case was followed up after completion of the treatment and categorised into one out of four severity groups according to the same standard protocol used for categorisation at the time of the initial hotline call.
Results: In 47 of 151 calls, DCI was suspected by the hotline experts. The initial estimation was consistent with the clinical findings in 37 cases, 9 were overestimated and one was underestimated. With the 95% bootstrap confidence interval 0.551 to 0.864 and computed weighted Cohen's coefficient = 0.721, the consistency between hotline assessment and clinical assessment can be considered as good. The five divers with minimal symptoms who were categorised as "*no DCI possible*" could not be followed up.
Conclusion: We conclude that, despite some limitations to the study, particularly the limited sample size, a reliable assessment of the severity of DCI can be provided by a specialist-based telephone hotline.

Key words
Diving accidents, decompression illness, clinical audit, underwater medicine

Introduction

Remote management of diving emergencies can be considered appropriate in situations where no on-site professional care by diving medicine experts is available within reasonable time. Phone hotlines offering medical assistance to divers or rescuers are not new, but there are few data published concerning the correlation of phone-assessed symptom severity with actual clinical findings.

Most hyperbaric treatment centres in Switzerland – a nation with almost eight million inhabitants and an estimated 25,000 recreational divers (Binkert H, personal communication, 2011) – have been closed down in recent years on political or economic grounds.[1] Currently the only hospital-based centre treating diving emergencies is situated at the westernmost edge of the country in Geneva. Because of this development, it became necessary to manage the limited resources in cooperation with other bodies to ensure good patient care.

The Swiss diving accident hotline (DAN Suisse), which is part of the European division of the worldwide Divers Alert Network (DAN), works in close cooperation with the dispatch centre of the Swiss airborne rescue service REGA. All divers in need may use REGA's emergency phone number as a relay to an on-call diving medicine specialist. The hotline service is not only available within the country, but can also be contacted from abroad. Incoming hotline calls are referred by the REGA dispatcher to one of six experienced diving medicine physicians (DMP). The on-call physician gathers information about the diver's data, his dive profile, the development of symptoms and signs and the medical history by interviewing the patient, his dive partner or on-site helpers according to a standard protocol. Based on this information, he estimates the severity of the reported case and recommends a course of action which, in a suspected case of DCI, includes administration of normobaric oxygen (NBO_2) and possibly transfer for hyperbaric oxygen treatment (HBOT). He also recommends an appropriate means of transport. In case of a life- or health-threatening emergency, evacuation by air will be initiated simultaneously by the REGA dispatch centre.[2]

The hotline DMP stays in close contact with the treatment centre or, if discharged, the patient to supervise the course of events and intervene if necessary. As soon as the initial treatment has been completed, all medical protocols and reports are transferred to another member of the hotline medical team for review of completeness and collection of missing documents. In case of deficiencies, this expert will contact the patient to obtain more information and offer further advice. Selected cases are subject to biannual case presentation and discussion by the hotline physicians and other DMPs. A high number of hotline calls, especially from abroad, concern injuries or indisposition unrelated to DCI. In these cases, general advice and assistance is provided by a triage centre to which the caller is referred.

The aim of the present study was to evaluate the relationship of severity of decompression illness (DCI), as determined via a telemedicine hotline, with the clinical findings on arrival at a treatment facility.

Methods

The database for this retrospective study was extracted from the original phone protocols recorded by the hotline specialist during or shortly after the emergency call, from the reports following treatment and from follow-up e-mails with the patient. Cases with initial phone calls from 01 January 2008 to 31 December 2009 were used. We excluded all calls concerning events definitely unrelated to DCI (injuries, questions about previous dive accidents or fitness-to-dive issues) but also diving-related lesions without evidence for DCI like isolated barotraumas (Figure 1).

For the comparison of the severity assessments, we had to exclude all cases where the hotline was contacted after the patient had already been evaluated in a clinical centre. However, some of the cases could be used for the analysis of the distribution of symptoms and signs of DCI, so that the number of patients differs for these two investigations.

The standard telephone protocol sheet includes the following information as a minimum:

- personal data, phone number;
- dive/accident site;
- dive profile, equipment and breathing gas used for the last dive (and the five dives before, if there was repetitive diving);
- detailed description of symptoms and signs, including the time lapse to their occurrence in relation to the dive profile or to the time of surfacing;
- measures taken on site (e.g., NBO_2, bystander resuscitation);
- the diver's medical history and evident health disorders prior to the dive;
- the present state of training.

Symptoms and signs were categorised according to a modification of the Francis and Smith classification for description and terminology of DCI (Table 1).[3] There were some modifications made in order to improve usability in a hotline setting, the main aim of these modifications being not to miss potentially severe symptoms.

The main DCI manifestation sites were recorded: cutaneous DCI was assumed in the presence of skin alterations like pruritus associated with macular eruptions, including "*cutis marmorata*"; if pain was mentioned, limb pain was categorised as musculoskeletal DCI, but girdle pain was categorised into the neurological severe group because of the high probability of spinal DCI. Paraesthesiae, with or without hypaesthesia, were categorised as a 'neurological light' manifestation. Cerebral symptoms and signs such as severe headache, dizziness, unconsciousness, convulsion, speech difficulty, impaired vision and mono- or hemiparesis were subsumed with symptoms for spinal DCI like girdle pain, paraparesis, paraplegia, urinary retention as 'neurological severe'. Suspicion of inner ear DCI with symptoms like vertigo, nausea, vomiting, hearing loss and tinnitus was also classed in this group. Soft tissue swelling, especially involving the abdomen, thighs and breasts indicated lymphatic DCI. If the patient was dyspnoeic or showed evidence of pulmonary oedema, pulmonary DCI was assumed. General malaise, anorexia or fatigue were categorised in the group of ambiguous symptoms possibly related to DCI. An actual case with multi-site symptoms/signs was classified into the group of most relevant manifestation but all the other symptoms were documented also.

If the reported symptoms and signs of DCI were improving spontaneously, the course is described accordingly, whereas a spontaneous deterioration of the patient's condition was denoted as progressive DCI. Besides these two dynamic forms, static and relapsing DCI were described as well.[4] With more than five minutes' remaining bottom time to the 'no-stop' time we assumed a low inert gas load. At least moderate gas burden was presumed if there was less than five minutes' bottom time remaining, if a decompression stop had to be respected or in case of repetitive diving. In any violation of a decompression algorithm, we classed the gas load as high. In the case of a rapid ascent with or without clinical signs of barotrauma, the patient was considered to be at high risk for pulmonary over distension and AGE. All details available concerning the breathing gas mixture, the dive profile and ascent rate, the state of physical and dive training, the dive conditions and the activity during the dive (e.g., recreational dive versus construction dive) were also taken into account.

A code for the DCI severity was used which determined the initial telephone decision about therapy:

0 – the case is most probably not related to DCI and can be treated in a regular hospital or by a GP;

+ – mild incident which might be due to DCI; NBO_2 is recommended and medical examination necessary; HBOT is not indicated;

++ – most probably DCI, therapy must be provided in a hyperbaric facility;

+++ – life-threatening DCI which will need hyperbaric and intensive care treatment.

All DCI events which showed severe neurological or pulmonary manifestations came into the '++' category, the same as if the course of DCI symptoms was progressive.

Table 1
Clinical classification of DCI used by the Swiss hotline team

Course	Clinical manifestation	Dive profile
Progressive	Skin	Pulmonary barotruama
Static	Pain	Gas burden
Improving	Neurological light	
Relapsing	Neurological severe	
	Lymphatic	
	Pulmonary	
	Ambiguous	

Patients classed into groups '0' and '+', i.e., where no HBOT was recommended, received a phone call from a hotline DMP the following day at the latest for check-up. The contact details for the non-DMP physician caring for the diver were sought in order to brief them with regard to late-onset symptoms and signs of DCI. The hotline DMP contacted the treatment centre the day after the start of the therapy and requested a preliminary report. A written follow-up consent was obtained from the patient. When the treatment was completed and only if the consent was given, the documents were submitted to the first author of the study who checked them for completeness, gathered further reports and assessed the manifestations and severity of the cases according to the description by the treatment centre. He also received follow-up information by contacting the patients individually from eight weeks to nine months after the incident. For the follow-up assessment the same categorisation as described above was used.

STATISTICS

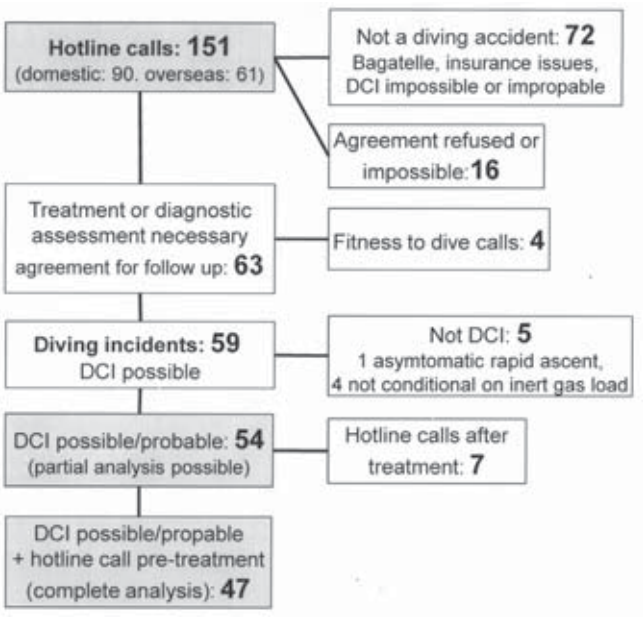
For demographic analysis, Microsoft Excel® was used. For the comparison of severity assessments, we computed the weighted Cohen's coefficient (with absolute weights).[5] The weighting ensures that differences between assessments that are "*close*" (adjacent categories) are considered to be "*better*" than those that are "*not close*". In general, confidence intervals based on large-sample theory do not perform well for Cohen's . Typically, therefore, a 'bootstrap' is used to compute confidence intervals (where M = 10,000 bootstrap samples were drawn); > 0.75 can be considered excellent agreement, values of k between 0.4 and 0.75 as fair to good agreement.[6,7]

All confidence intervals were computed using $\alpha = 0.05$. All analyses were performed using R (R Development Core Team, 2010).[8] Cohen's was computed using the package *psy* (Falissard B. 2005. *psy*: Various procedures used in psychometry. R package version 0.7), and bootstrap confidence intervals using the package *boot* (Canty A and Ripley B. 2009. *Boot*: Bootstrap R (S-Plus) Functions. R package version 1.2-35). The tables of descriptive statistics were generated using the package *report tools*.[9]

Table 2
Gender and age distribution, number of dives and depths

	Women (n = 13)	**Men** (n = 50)	**All** (n = 63)
Age (years) median (range)	35.8 (22–52)	40.5 (23–70)	39.5 (22–70)
Diving experience			
< 20 dives	-	1	1
21–100 dives	1	7	8
101–500 dives	7	29	36
> 500	5	13	18
Max. depth (msw)			
mean (SD)	30.1 (20.4)	42.9 (19.6)	40.2 (20.3)
range	6–82	10–101	6–101

Figure 1
Overview of hotline calls to DAN Suisse over a two-year period (2008–2009)



Results

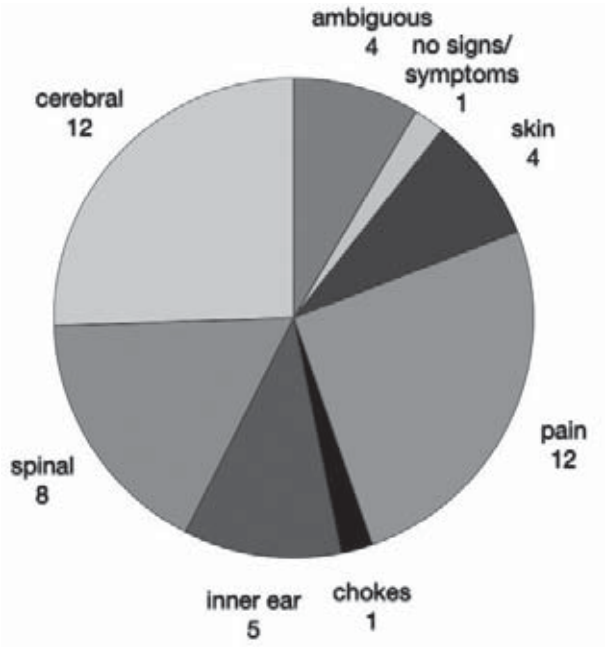
Figure 1 shows an overview of the hotline calls. 47 calls were related to diving accidents and occurred before the initiation of definitive treatment so that a complete analysis was possible in these cases. In addition, as much information as possible was extracted from the data available on the remaining calls. Therefore, the total case numbers reported may vary. None of the cases which were initially suspected as non-bubble related turned out to be DCI later.

DIVING BEHAVIOUR AND DIVE PROFILES

The gender and age distribution, the number of dives and the depths of the dives are shown in Table 2. Only 17 of the 59 included dives were shallower than 30 metres' water depth (mw).

For 55 of the 59 dives, there were sufficient data on the dive profile, circumstances of the dive, number of repetitive dives and the breathing gas, so that we were able to estimate the gas load and the probability for pulmonary barotrauma. High gas load was assumed in 20 cases, moderate or low in 35 cases. High probability for pulmonary barotrauma was expected after 14 dives, moderate or low after 41 dives. Looking for cases with moderate or low gas load as well as low or moderate probability for pulmonary barotrauma, we found 23 profiles. In most of these cases of 'undeserved' DCI

Figure 2
Main site of symptoms of DCI (hotline assessment), *n* = 47



other risk factors like patent foramen ovale (PFO, three), transpulmonary shunt (one), PFO and transpulmonary shunt (one), dehydration (one), repeated diving to minor depths (three), obesity (one) and former episodes of DCI (three) were identified.[10–12] In nine of the affected divers, no follow up was possible.

MANIFESTATIONS OF DCI

As shown in Figure 2, in about half of the 47 reported cases, DCI initially manifested with pain or with cerebral symptoms. In 26 cases, the symptoms were described as distressing (cerebral, spinal, pulmonary or inner ear). In one case, the hotline was called after an emergency ascent from 35 mw. While this diver remained symptom free, his buddy died. In seven cases, there was more than one manifestation site. Three were relatively minor (joint bends and ambiguous symptoms or skin bends), whereas in four cases cerebral symptoms were combined with pulmonary, inner-ear or spinal lesions.

ASSESSMENT OF SEVERITY

Table 3 shows the correlation of the estimated grade at the time of the call compared to clinical assessment. Clinically there were five cases categorised as, '0', 18 as, '+', 19 as, '++' and 1 as, '+++'. The initial estimation was consistent with the clinical findings in 37 of 47 cases, nine were overestimated and one was underestimated. Cohen's = 0.721 with 95% bootstrap confidence interval 0.551 to 0.864. Therefore, the agreement between the telephone and subsequent clinical assessment can be considered as 'good'.

Table 3
Comparison between DAN Suisse phone hotline DCI severity scores and subsequent clinical grading of DCI severity (see text for explanation of grading)

Telephone category	Clinical category			
	0	+	++	+++
0 (*n* = 5)	5	–	–	–
+ (*n* = 19)	4	14	1	–
++ (*n* = 20)	–	3	17	–
+++ (*n* = 3)	–	1	1	1

DELAY TO NORMOBARIC AND HYPERBARIC OXYGEN TREATMENT

NBO_2 was used in 40 divers and 29 required HBOT. The median time from surfacing to NBO_2 was 0.875 h (immediately to 48 h) and the median time to HBOT was 11 h (2–170 h). By comparison in Europe, the median time to NBO_2 was reported as 0.75 h (immediately to 48 h) and to HBOT a median of 5 h (2–170 h), while the corresponding intervals for remote areas outside Europe reached a median of 4 h (0.75–24 h) for NBO_2 and a median of 36 h (9–72 h) for HBOT.[13,14]

Discussion

Many recreational divers are aware of the existence of diving medical hotlines in various parts of the world. Information concerning the assistance and rescue networks is now included in the basic training of a majority of novice divers. According to some publications, hotlines are used frequently.[15] On the other hand, there are no publications to our knowledge relating the remote phone assessment with the clinical findings during the primary clinical assessment. Comparable studies exist only for clinical disciplines other than diving medicine.[16–18] We intended to fill this gap at least partly for a small national hotline. We used mostly handwritten notes for data analysis. Audio recording was not technically possible and might have breached privacy laws. It cannot be excluded that some information that was potentially useful for evaluation was lost. As the accident assistance is given the first priority during a hotline call, the data completeness required for a later evaluation may suffer. Nevertheless, thanks to the hotline DMPs' experience, adequate record keeping was fulfilled in most of the cases. Inter-investigator variability was not evaluated. The case numbers were sufficient for a statistical analysis.

Our decision to categorise the presumed DCI cases into four severity groups might seem arbitrary. But this approach simplified the workflow by implication of a therapeutic decision in each triage group and allowed re-evaluation based on the clinical presentation. In nine cases, the clinical findings were less severe than initially graded by the hotline specialist. We did not assess if these patients

were consequently over treated. In view of possible severe sequelae, it is common practice in diving medicine to pre-emptively treat patients in the presence of serious, or rapidly developing initial symptoms. The improvement in symptoms during transportation as a result of the administration of NBO_2 and other supportive measures may contribute to this apparent over estimation.[19]

The one case that was underestimated is worth discussing. A young woman presenting with a discontinuous course of cerebral symptoms after repetitive diving called the hotline late, after consulting a general practitioner without any diving medical training, who found her psychosomatically ill. She was referred to a local DMP who found a slight sensorimotor deficit caused possibly by a radicular irritation and denied any relation to DCI. Because of the persistence of fluctuating symptoms, an MRI of the neck and head was performed several weeks later, with a diagnosis of a vertebral artery dissection.[20,21] In view of this diagnosis, we interpreted this case as a symptom underestimation although the underlying pathology was not related to DCI.

It remains questionable whether minor DCI was missed among the divers categorised as 'no DCI possible' by the hotline DMP. We are trying to prevent misinterpretations by contacting the patients again shortly after their initial call and encouraging them to call the hotline if there are any residual or new health issues up to 48 hours after diving.

Failure to contact the hotline in the presence of symptoms might be because of non-recognition or denial by the diver or associated others.[22,23] Skin manifestations of cutaneous DCI are easily misinterpreted by divers or by physicians not trained in diving medicine as an allergic reaction, jellyfish sting or sunburn and classical 'bends' as musculoskeletal injury. Missed hotline contact in such minor cases could explain the relative over-representation of severe cases in our sample in comparison to other publications.[24]

For an experienced DMP it might be straightforward to diagnose the type and severity of a diving emergency based on medical history and clinical findings. In the telemedicine setting, the latter are missing. It is not surprising, therefore, that a hotline physician tends to attribute symptoms described by callers into a more severe category. Besides these difficulties, medicolegal aspects also have to be considered.[25] The times elapsed from the onset of possible manifestations of DCI to a hotline call and to the initiation of treatment in this investigation are also noteworthy. Various factors contribute to this, such as diver delay to make a hotline call and the availability of transport and of hyperbaric facilities

A diving medical hotline may not only actually shorten the time to recompression treatment by organising the transfer and HBOT facility information but alleviate the symptoms and signs by advising immediate initiation of NBO_2 therapy. This reflects our own observations and is worthy of further investigation. Diving medical hotlines are widely believed to be able to shorten the time to recompression. Our data showed that recompression treatment was provided more quickly inside Europe than in remote areas. Whether or not this observation correlates with the greater availability of hyperbaric treatment centres, with better transportation or with the use of a DMP hotline cannot be determined based on this small sample. By encouraging the hotline callers to use NBO_2 if there is a possibility of DCI, symptoms and signs can be alleviated. Further prospective studies with large numbers of cases are needed to investigate these factors effectively.

Diving medicine hotlines can be considered as a form of telemedicine, in spite of missing some aspects such as electronic imaging or laboratory data transfer, both primarily non-essential in the initial management of diving accidents.[26] The existing infrastructures, e.g., mobile phone networks, are sufficient for diving medical purposes even if the calls arrive from very remote areas. Future technical development might permit the submission of data about dive profiles by mobile phone. PDAs have been shown to be useable as dive computers if enclosed in an underwater housing and equipped with a compatible sensor device.[27]

Besides the comparison of the hotline diagnosis to the final clinical diagnosis, our limited study has served a quality assurance purpose and helped us to better adapt the hotline to developing international telemedicine standards.[28] We have demonstrated that accurate assessment of the severity of DCI can be achieved by a specialist-manned telephone hotline. Overestimation of severity occurred in less than one fifth of the cases, and the hotline provides divers with a specialist DMP's prompt advice, enabling them to receive specialised treatment. A standardised international reporting system similar to existing cancer registries would be helpful to evaluate indications and treatment options. Further prospective studies with larger numbers of patients are necessary to evaluate the consistency of decisions provided by diving medical hotlines. These studies should also aim at the introduction of a benchmarking system in order to enhance the quality of these commonly used services.

Conclusion

In a small group of divers contacting a diving emergency hotline manned by diving medical specialists, reliable estimates of the severity of the condition were possible in the majority of cases.

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Effect of hyperbaric oxygen on bone healing after enucleation of mandibular cysts: a modified case-control study

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Abstract

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Introduction: Mandibular cysts may require enucleation, resulting in large cavities that compromise mandibular strength and functions. We investigated the effects of hyperbaric oxygen therapy (HBOT) on healing after enucleation of mandibular cysts.

Patients and methods: Fourteen healthy individuals in whom no modifiers of wound healing were present received a median of 20 post-operative HBOT. The rate of filling of the defect was derived from the number of pixels in the residual cavity after transformation of the area of the lesion in orthopantomograms taken immediately after surgery and at six months post operation. Modifications in bone density, detected on panoramic radiographs, were defined through a gray scale of 256 tonalities. The radiolucency of a healthy tooth was used as a reference to control for differences between radiographs taken at different times in the same patient. Both the rate of filling and changes in bone density were compared with corresponding data from a previous study of 27 healthy subjects who were allowed to heal spontaneously without HBOT.

Results: At six months post operation, the HBOT group showed 55 ± 9% reduction in the size of the residual cavity and 55 ± 17% increase in bone density compared to immediate postoperative values. Corresponding values in the control study were 12 ± 4% and 37 ± 23%, respectively. These differences were significant (t = -16.95; P = 1.21E-11 [x 10^{-11}] for the reduction in cavity size and t = -2.39; P = 0.029 for bone density).

Conclusion: HBOT merits a place as a useful adjunct in the surgical management of defects of the mandible.

Key words

Hyperbaric oxygen therapy, dental, radiological imaging, clinical audit, outcome

Introduction

The mandible is one of the important bones of the facial skeleton. It provides a framework for many of the structures involved in mastication, phonation, deglutination, facial expression and aesthetics. Several lesions, such as cysts of the mandible, may need to be enucleated as a part of treatment, resulting in large cavities that compromise its strength and functions. Additionally, this may lead to a pathological fracture and result in protracted morbidity, treatment and sometimes hospitalisation. The natural healing process takes up to twelve months for 43% of bone filling to occur with a 48% increase in bone density.[1]

Accelerating the rate of filling of these mandibular defects without compromising the quality of the regenerated bone is a much desired innovation. Various methods used include bone grafts (autogenous, allograft or xenograft) alone or in combination with alloplastic materials (e.g., bovine-derived collagen paste), hydroxyapatite granules (proposed with the aim of providing a scaffold for enhanced bone tissue repair) and guided bone regeneration with the use of semi-permeable barriers. However, all these methods may present some limitations, and are reviewed elsewhere.[1]

In the last few decades, hyperbaric oxygen therapy (HBOT) has been found to be an important adjuvant in the healing of wounds and fractures. A substantial literature is available to support the benefit of HBOT in cases such as osteoradionecrosis and osteomyelitis.[2–4] Even in mandibles which neither have been irradiated nor are infected, HBOT has been found to be of value in restoring bone defects.[5–7]

Nonetheless, the inferences of the latter cannot be translated to mandibular defects arising after enucleation of cysts because most of such studies are confined to animals. Healing in a human mandible is conceived to be different from that in rat or rabbit due to variation in structural morphology which includes distribution as well as local modifications of compact bone and arrangement and development of spongy bone trabeculae.[8] Some recent studies and reviews have raised doubts on the usefulness of HBOT in osteoradionecrosis or delayed union/non-union of bony fractures.[9–12]

The present study investigated the effects of HBOT on the healing process and rate of bone regeneration subsequent to enucleation of mandibular cysts in a group of individuals in whom no other modifier of wound healing was found to be present. The above objective was achieved by quantifying the rate of filling and changes in the bone density of the lesion in these patients over a period of six months and then comparing these variables with the data reported in a previous study of 27 subjects who were allowed to heal spontaneously (i.e., without HBOT).[1]

Table 1
Patient details and summary of presenting pathology and surgical management of mandibular cysts

Case	Age (yr)	Sex	Diagnosis	Site	Treatment
1	11	M	Radicular cyst	Lt body, from canine to 1st molar	Enucleation
2	16	F	Odontogenic keratocyst	Rt ramus	Enucleation and chemical cauterization
3	12	M	Odontogenic keratocyst	Rt parasymphysis, from canine to Lt ramus	Enucleation and chemical cauterization
4	56	M	Odontogenic keratocyst	Rt angle and ramus	Enucleation and chemical cauterization
5	58	M	Multiple radicular cysts	Lt 1st molar, Rt lateral incisor and premolar	Enucleation and curettage
6	29	M	Odontogenic keratocyst	Lt body to ramus	Enucleation and chemical cauterization
7	14	M	Dentigerous cyst	Rt body	Enucleation
8	31	M	Odontogenic keratocyst	Rt body to Lt parasymphysis	Enucleation
9	18	M	Odontogenic keratocyst	Lt angle and ramus	Enucleation and chemical cauterization
10	26	M	Dentigerous cyst	Lt angle	Enucleation
11	12	M	Radicular cyst	Lt body	Enucleation
12	72	F	Radicular cyst	Rt body	Enucleation
13	34	M	Multiple odontogenic keratocyst	Rami bilaterally	Enucleation and chemical cauterization
14	36	F	Radicular cyst	Symphysis	Enucleation and curettage
15*	13	M	Dentigerous cyst	Lt body	Enucleation
16**	32	F	Odontogenic keratocyst	Rt angle	Enucleation and chemical cauterization
17**	48	F	Radicular cyst	Lt body	Enucleation

* Refused HBOT
** Lost to follow up

Methods

PATIENT SELECTION

In all, we received 17 patients in whom the inclusion and exclusion criteria (*vide infra*) were met. Of these, one (case 15) refused to undergo treatment with HBOT and two (cases 16 and 17) were lost to follow up. The remaining 14 patients ranged between 11 and 72 years old. Such a varied age group was admitted to the study because cysts and cystic lesions of the mandible are uncommon (confinement of subjects to a narrower age group, e.g., 20–40 years old, would have entailed rejection of nine patients, precluding a statistically viable sample size). Also the 'control' group study to which we compared our data varied between 6 and 63 years in age.[1] Age, sex, diagnosis and the site of lesion(s) and the surgical treatment given for individual subjects are shown in Table 1. All the subjects were of middle socioeconomic status and apparently well nourished.

Inclusion criteria included 1) cysts and cystic lesions of the mandible treated surgically and 2) defects not less than 30 mm in a single lesion and not adding up to 30 mm in multiple lesions. These inclusion criteria were to make the subjects comparable to those studied by Chiapasco et al.[1] Exclusion criteria included patients with any intercurrent illness and/or medication that would have affected healing and patients with bone grafts or osteopromotive membrane.

The patients were informed about the nature of the study and all possible risks involved, and consent was obtained. The protocol was approved by the Ethics Committee of the Institute of Aerospace Medicine (IAM), Bangalore, India. All the patients were subjected to thorough pre-operative and pre-anaesthetic evaluation. The investigations included routine haematology, urinalysis, bleeding and clotting times, fasting and postprandial blood sugar, serum urea, a resting ECG, pulmonary function tests and a chest X-ray. The last was especially to identify Gorlin-Goltz syndrome associated with odontogenic keratocyst. Pre-operative orthopantomograms (OPG) were taken to calculate baseline values of the area of bony defect and density.

SURGERY

All the surgical procedures were performed by an intraoral approach under local and general anaesthesia. Appropriate antibiotics were administered at induction. Intra-operative corticosteroids were administered to reduce oedema. For cysts involving the dento-alveolar region, a sulcular approach was used in most cases. Whenever tooth preservation was possible, a crevicular incision was used. The cyst wall was enucleated. In cases of odontogenic keratocyst, peripheral ostectomy was performed to destroy the daughter cysts.

Patients with odontogenic keratocysts also underwent, after enucleation, chemical cauterization with Carnoy's solution (6 parts ethyl alcohol (absolute or 95%), 3 parts chloroform and 1 part glacial acetic acid). This kills epithelial remnants and dental lamina in the osseous margin. The cystic cavity was irrigated with saline and hydrogen peroxide and haemostasis was secured. In all cases, the wound was closed primarily with 3-0 Vicryl. All patients received perioperative antibiotic cover. In-patients received intravenous antibiotics whereas patients treated on an outpatient basis were administered oral antibiotics.

HYPERBARIC TREATMENT

Within a week of surgery, HBOT was commenced daily, five days per week, in a multiplace chamber at a pressure of 253 kPa with the patients breathing 100% O_2 via an oronasal mask and demand regulator (British MK 17E). Duration of each treatment session was about 90 min, inclusive of compression and decompression, the rate of which was approximately 10 kPa min^{-1}. The median number of HBOT sessions was 20 (18 and 21 being the lower and upper quartiles, respectively). Before HBOT, mobility of tympanic membranes and the ability to ventilate middle ears was ascertained by Valsalva procedures.

FOLLOW UP AND RADIOGRAPHIC ANALYSIS

Patients were followed up clinically and radiologically. This included postoperative assessment, after HBOT and then on a monthly basis for a period of six months. Clinical evaluation included an examination of the operation site for inflammation, wound dehiscence, evidence of infection, the amount of healthy granulation tissue, pathologic fractures and the general condition of the patient.

OPGs were taken immediately after surgery and at six months postoperatively to evaluate the reduction in size of the residual cavity and changes in bone density. Radiographs were also assessed for trabecular pattern, amount of radiological bone filling and evidence of infection and fracture. The size of the residual surgical defect and the bone density were calculated by the technique described by Chiapasco et al.[1] Accordingly, the area of the residual defect was transformed into pixels using Corel Photo-Paint 5™ (Corel Corporation, Canada). The number of pixels in the residual cavity was calculated and the variation in the number of pixels in the remaining surgical defect across time was interpreted as bone filling in the area of the lesion.

Similarly, modifications in bone density, detected on the panoramic radiographs, were defined through a gray scale of 256 tonalities. Put simply, it was an average of all the 256 tonalities weighted for their numbers. The grayscale value thus obtained was normalised to the corresponding value derived from a healthy tooth, the radiolucency of which served as a reference to control for differences between the radiographs taken at different times in the same patient. MATLAB®, version 7.2.0.232 (Release 2006a), was used for this analysis. The selection of the area of the lesion at six months was kept corresponding to that seen immediately after surgery. For bone density, a smaller number of cases (n = 8) was available as bone density estimates could not be made in the others due to artefact shadows from tongue or cervical vertebrae.

To avoid any bias and consequent imprecision in the above analysis, the two computations were performed by a resident from the Department of Oral and Maxillofacial Surgery, RV Dental College and Hospital, Bangalore, who was blinded to identity of panoramic radiographs and was not associated with the study. Such assessments of size of the residual surgical defect and of bone density are reported to have good agreement with the corresponding measurements derived from computed tomography.[1] A similar/comparable procedure has been used by others.[13,14]

STATISTICAL ANALYSIS

An unpaired Student t test, as applicable to data samples with unequal variance, was employed to examine the significance of differences in the reduction in the size of the residual cavity and increase in bone density in the two groups (viz, one which healed spontaneously, and the other with HBOT). Such a comparison of cases with rather unrelated (but not un-matched) controls which have been studied by others, is regarded as an acceptable procedure employed by others.[15,16] It was also employed to see if the healing was different after chemical cauterization of the defect, as part of the standard treatment, in a subset of patients in the present study. Single factor ANOVA was used to examine variation in healing in cystic defects arising after enucleation of different types of cysts because it was, essentially, a multi-sample hypothesis. Pearsonian product moment correlations were calculated (between age and rate of filling/increase in bone density) to find out if bone healing was influenced by age. The level of significance was kept at $P < 0.05$. However, exact significance with the associated degrees of freedom is annotated in the results.

Results

Cases in the present study were comparable in age with the 'controls' studied by Chiapasco et al[1] (cases: 30 ± 19 yr versus 'controls': 35 ± 13 yr; t = 0.96, P = 0.341 for df = 39). Except for two patients, who developed wound dehiscence, all experienced uneventful primary healing. These two cases were allowed to granulate and heal secondarily. One of them developed infection at the operated site that resolved with antibiotic therapy. Clinically, the degree of bone regeneration after six months was sufficient for the dental surgeons to consider implant rehabilitation of the patients in the following few months. In fact, the healing was so good that, in two of the initial cases, implants were placed and osseous integration occurred without any problem.

Table 2
Reduction in size of residual cavities (expressed as number of pixels) and increase in bone density (expressed as normalised grayscale values) in cases (*n* = 14 or 7; present study) and 'controls' (*n* = 27; derived from Chiapasco et al, 2000[1])

	Present study			'Control' study			
	Immed. post-op	6 months post-op	% change	Immed. post-op	6 months post-op	% change	*P*-value
No. of pixels	137,143 ± 84,252	60,303 ± 40,215	55 ± 9	14,832 ± 6,238	12,999 ± 5,589	12 ± 4	1.21E-11 for df = 15.65
Bone density	39 ± 12	60 ± 17	55 ± 17	82 ± 59	103 ± 61	37 ± 23	0.029 for df = 15.95

Note: Degrees of freedom (df) are for unpaired Student t tests for data samples with unequal variance and, in the case of bone density, also fewer cases (*n* = 7) as density estimates could not be made in all patients because of artefact shadows from tongue or cervical vertebrae.

Table 2 shows the reduction in size of the residual cavities (expressed as number of pixels) and increase in bone density (expressed as normalised grayscale values) in cases in the present study and in the 'controls' study.

As can be seen from Table 2, the number of pixels amongst the patients in our study was 137,143 ± 84,252 in the immediate post-operative period. This reduced to 60,303 ± 40,215 at six months. Corresponding values in the control study were 14,832 ± 6,238 and 12,999 ± 5,589. Therefore, in normalised terms, a reduction in the size of the defect of 55 ± 9% was seen in the HBOT group (present study) at six months, compared to 12 ± 4% in the 'control' study. The difference in the reduction in the size of defect was highly significant (t = -16.95, *P* = 1.21 E-11 [x 10^{-11}], df = 15.65). The above values of 't' and degrees of freedom are calculated considering unequal variance in the two groups, hence degrees of freedom are not integers (see Table 2).

It is to be appreciated that the large difference in the values in the number of the pixels/grayscale values between the two studies is because the images were enlarged in the present study to facilitate easy computation. However, this does not affect the analysis because it influenced the number of pixels/grayscale values in the films taken immediately and at six months post-operatively in a similar manner.

Weighted grayscale histograms of the area of interest (cavity), along with those of the reference (healthy) tooth are given in Figure 1. Figure 2 shows serial orthopantomograms in a representative case (Case 3).

Bone density, expressed as the average grayscale value of 256 tonalities and normalised to a healthy tooth, increased from 39 ± 12 in the immediate post-operative period to 60 ± 17 at six months in our subjects. Corresponding values in the 'control' study were 82 ± 59 and 103 ± 61. Thus, an increase of 55 ± 17% in bone density was seen in the HBOT group (present study) at six months; whereas the increase in the 'control' study was 37 ± 23%. The above difference was, again, statistically significant (t = -2.39, *P* = 0.029, df = 15.95). Both the reduction in the size of defects and the increase in bone density correlated poorly with age (r = 0.026, t = 0.09, *P* = 0.928 for reduction in size of defect and r = 0.554, t = 1.629, *P* = 0.154 for increase in bone density).

In the present study, chemical cauterization (employed as part of the treatment of odontogenic keratocyst after enucleation to prevent recurrence) did not modify healing. Reduction in size of defect was 57 ± 8% in the group treated with chemical cauterization and 52 ± 10% in the group not so treated. The two values were not significantly different (t = 1.05, *P* = 0.314, df = 9.31). Similarly, increase in bone density (50 ± 19% in the group treated with chemical cauterization and 63 ± 8% in the group not so treated) was statistically comparable (t = -1.12, *P* = 0.306, df = 5.70).

No significant variation in healing of the defect arising after enucleation of different type of cysts was observed, both in terms of reduction in the size of defect (56 ± 6% for radicular cysts, 52 ± 9% for odontogenic keratocysts and 60 ± 12% for dentigerous cysts; F = 1.01, *P* = 0.395) and an increase in bone density (45 ± 26% for radicular cysts, 59 ± 11% for odontogenic keratocysts and 57 ± 24% for dentigerous cysts; F = 0.42, *P* = 0.680).

Discussion

Bone healing occurs in three distinct but overlapping stages: an early inflammatory stage, the repair stage and the late remodelling stage. In the inflammatory stage, a haematoma develops within the defect during the first few hours and days. Inflammatory cells (macrophages, monocytes, lymphocytes, and polymorphonuclear (PMN) cells) and fibroblasts infiltrate the bone under prostaglandin mediation. This results in the formation of granulation tissue, in-growth of vascular tissue, and migration of mesenchymal cells. Once a haematoma forms at a fracture site, it must be invaded by a vascular spindle. A continuously occurring state of bone deposition, resorption, and remodelling facilitates the healing process.

Control of angiogenesis in wound healing is shown to be

Figure 1
'x' axis represents pixels with grayscale values from 0 to 256. Each bar singularly represents a grayscale tonality weighted for its number averaged across subjects. Weighted values, represented on 'y' axis, have no units.

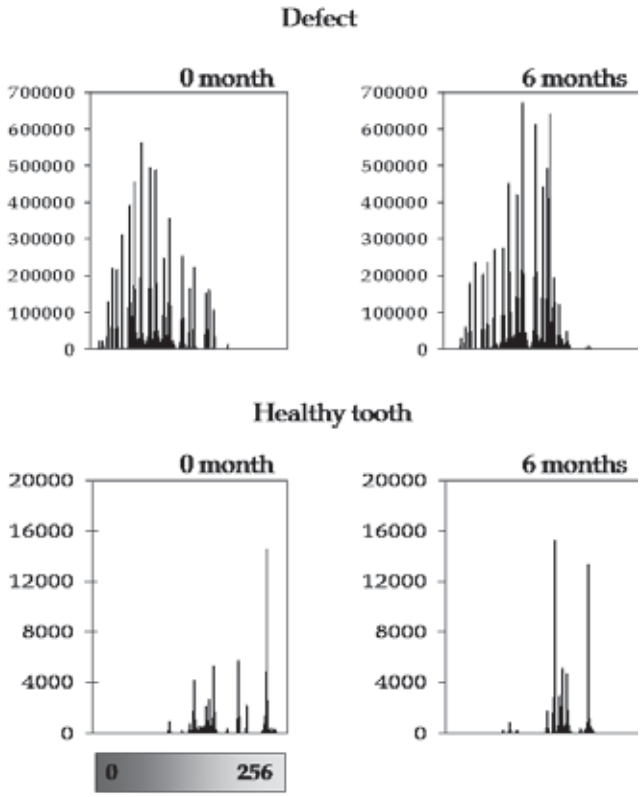
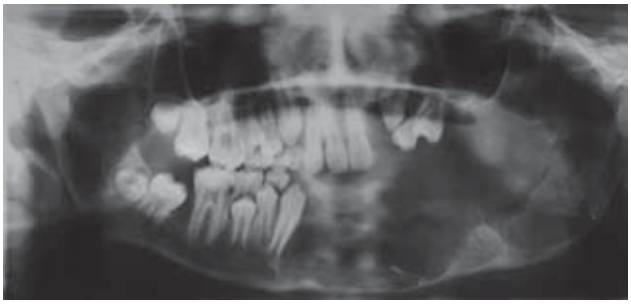
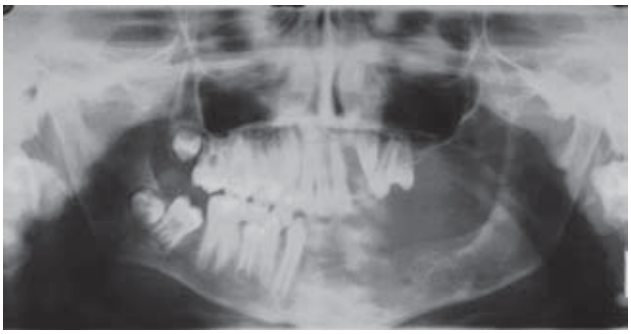


Figure 2
Serial orthopantomograms from Case 3
immediate post-op



6 months post-op



the result of macrophages responding to tissue oxygen (O_2) tension without the necessity of interacting with other cell types or biochemical signals.[17] Hyperbaric oxygen has been shown to increase the expression of vascular endothelial growth factor, a potent stimulus for endothelial cell activity, in calvarial critical-sized defects in rabbit.[18] With the progression of vascular in-growth, a collagen matrix is laid down, while osteoid is secreted and mineralised, and leads to formation of a soft callus. O_2 is essential to promote fibroblast proliferation and collagen production. Hydroxylation and cross-bonding of the collagen precursors also require O_2. Collagen cannot be synthesised by the fibroblast unless adequate amounts of both proline and lysine are hydroxylated with O_2. Synthesis requires one atom of O_2 for every three amino acids in sequence. O_2 is also required in increased amounts during the repair process to provide energy for protein synthesis.[19] A high rate of energy metabolism, evident from adenosine triphosphate content of callus is observed in the early phase of fracture healing, which persists until the callus is corticalised and remodelling starts.[20] Several animal studies have shown that hyperbaric O_2 speeds up callus formation.[21,22]

Resistance to infection is extremely dependent on local O_2 tension. The bactericidal action of polymorphonuclear leukocytes uses O_2. Thus, HBOT acts as an adjuvant to antibiotic therapy.[23] PMN cells require O_2 to kill organisms by producing superoxide, hydrogen peroxide, singlet O_2, and other products via the respiratory burst phenomenon.[24] The PMN is protected by detoxifying free radicals with superoxide dismutase, catalase, and glutathione. It has been shown that the degree of PMN cell function in killing of bacteria is directly dependent on O_2 tension.[25] There is also O_2-independent killing with lysozymes, acidic vacuoles, and lactoferrins.[26] However, they are less efficient and vary significantly according to the organism.

Subsequent bone formation is also O_2 dependent, and variations in O_2 supply may affect the type of tissue that differentiates in a culture of multipotential cells.[27,28] Hyperoxia causes differentiation to osseous tissues, whereas hypoxia resulted in cartilage formation. HBOT has been shown to increase the mineralisation and density of bone.[29] Finally, hyperbaric O_2 also affects osteoclastic activity.[30] Thus, HBOT influences all the stages of bone healing.

Results of the present study also suggest that, in the absence of a pathological condition which can adversely affect tissue regeneration and healing, the latter is not affected by age. This is apparent from the poor correlation between age and both reduction in the size of defects and the increase in bone density.

Since histopathological characteristics of the three cystic lesions (radicular cysts, odontogenic keratocysts and

dentigerous cysts) examined in the present study are different, it was of interest to explore whether the rate of healing was different across the three groups. It was especially applicable to odontogenic keratocysts which are well circumscribed by a thin shell rich in calcium. We did not find any significant difference in healing across the three groups. Nonetheless, this inference is to be viewed with caution because of the small sample size (four radicular cysts, seven odontogenic keratocysts and three dentigerous cysts).

As mentioned earlier, some recent studies and reviews have lent support to scepticism regarding the usefulness of HBOT in osteoradionecrosis of mandible or healing of bony fractures.[9–12] In sharp contradistinction to these studies, our results have demonstrated spectacular improvement in bone healing with HBOT, given after mandibular cyst surgery. We believe there are several reasons for this. The study of Annane et al. was under-powered (as admitted in the accompanying editorial) and was terminated prematurely with a less than required number of subjects.[9,31] It is also questionable if employment of 'intention-to-treat' analysis was appropriate in such a study with a large Type-II error. The other study, by Maier et al. employed a different treatment strategy that was overtly out of phase.[10] In any case, these two studies investigated the effect of HBOT in a pathological condition and are not comparable to the healing of cystic defects after enucleation of mandibular cyst(s) in healthy humans. On the other hand, two systematic reviews, per se, do not refute the appropriateness of HBOT in fracture healing.[11,12] What these reviews stress is the necessity of well-controlled trials to endorse the usefulness of HBOT in such instances. Non-availability of controls is the most commonly felt deficiency in the research related to HBOT involving human subjects. Ours is an investigation which overcomes this, albeit imperfectly, comparing cases with unrelated controls. Thus, in real terms, our results do not contradict these reviews.

The present study demonstrates that the rate of filling of the residual cavity and increase in bone density in surgical defects of the mandible were significantly greater in subjects given HBOT than in subjects in a previous study who healed spontaneously. The present study, with certain limitations, is a case-control study. Nonetheless, the 'controls' did not form part of our study. Even though the two groups were comparable in age, it could not be ascertained whether the 'control' patients were comparable to the our patients with respect to nutritional status and absence of other modifiers of healing.[1] However, we have no reason to presume that the two groups are not comparable, even though other factors which influence tissue regeneration and wound healing were not reported by Chiapasco et al.[1] Their subjects were from a well-developed Western country so it is unlikely that they were undernourished, receiving drugs or had less time for rest and recuperation, or were suffering from illness which could have influenced healing.

Conclusion

We conclude that HBOT significantly improved bone healing and merits a place as an effective adjuvant therapy in the post-operative management of bony defects of the mandible arising from enucleation of large cysts.

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Conflict of interest: none

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Editor's note:
The M file used to calculate the grayscale values is available from the corresponding author or from the journal office. It can be read in MSWORD™ or NOTEPAD, and provides a method to import a file in MATLAB®, select a portion of the image and calculate the grayscale values.

Oxygen toxicity seizures: 20 years' experience from a single hyperbaric unit

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Abstract

(Banham NDG. Oxygen toxicity seizures: 20 years' experience from a single hyperbaric unit. *Diving Hyperb Med*. 2011 December;41(4):202-210.)

Introduction: Oxygen toxicity seizures (OTS) are a known complication of hyperbaric oxygen therapy (HBOT). The incidence of OTS has been variously reported and appears to be related to the duration and pressure of exposure in addition to individual susceptibility factors.

Method: All OTS occurring in patients undergoing HBOT during the first 20 years of operation of the Fremantle Hospital Hyperbaric Medicine Unit were reviewed.

Results: During 41,273 HBOT exposures in 3,737 patients, 25 OTS occurred; a rate of 0.06% (1/1,651 or 6 per 10,000 HBOT exposures). For the initial treatment of dysbarism with United States Navy Treatment Table 6, the rate was 0.56% (4/714) and for the treatment of carbon monoxide (CO) poisoning it was 0.18% overall but 0.49% for the first HBOT. There was an increasing OTS rate with increasing pressure with a statistically significant difference ($P < 0.001$) in OTS rate at 203 kPa or less versus > 203 kPa (odds ratio (OR) 8.5, 95% confidence intervals (CI) 2.0 to 36.1), and for comparison of two commonly used pressures of 203 kPa versus 243 kPa ($P = 0.028$, OR 5.1, 95% CI 1.1 to 22.8), but not with first versus follow-up HBOT at 284 kPa for dysbarism ($P = 0.061$) nor CO poisoning ($P = 0.142$).

Conclusions: This study reports all OTS in a single hyperbaric unit over a 20-year period; the longest observational study period yet reported for OTS during HBOT for all indications. The incidence of OTS in this study compares favourably to previously reported rates, and shows an increasing OTS rate with increasing pressure.

Key words

Central nervous system, oxygen, toxicity, seizures, hyperbaric oxygen, hyperbaric oxygen therapy

Introduction

Oxygen toxicity seizures (OTS) are a well recognised but uncommon complication of exposure to oxygen at pressures greater than 1 atmosphere (101.3 kPa, hyperbaric oxygen). OTS (in animals) was first described by Paul Bert in 1878; CNS oxygen toxicity is now known as the Paul Bert effect.[1] OTS occurring in humans was first observed by Damant and Phillips in 1933, who breathed 100% oxygen in compressed air at 405 kPa and had convulsive symptoms at 16 and 13 minutes respectively.[2] Experiments by Donald during World War II resulted in oxygen toxicity seizures in experimental divers – at that time "*no experimental dives where men had breathed pure oxygen at toxic tensions under water had yet been reported*".[2] Donald's experiments found a large variation in oxygen tolerance in his group of human subjects, as well as an "*individual variation, which is found to be over an enormous range*".[2]

A review of OTS was recently published by Bitterman, briefly presenting the features, dosing, risk factors, mechanisms of and protection against hyperoxic seizures.[3] Risk factors included 'wet' diving compared to 'dry' chamber diving, elevated concentrations of carbon dioxide and exercise. The mechanism of OTS, however, remains poorly understood. The rate of OTS in patients undergoing hyperbaric oxygen therapy (HBOT) has been variously reported and seems dependent on the exposure table (time and pressure), the condition treated and perhaps even the method of oxygen delivery (face mask versus head hood versus monoplace chamber).[4–15] *

The aim of this study was to determine the rate of OTS in all patients treated with HBOT during the first 20 years of operation of the Fremantle Hospital Hyperbaric Medicine Unit (HMU). The HMU provides a 24/7 365 days a year emergency service covering all of Western Australia as well as providing an elective hyperbaric service predominantly for the metropolitan areas of Perth and Fremantle. We also wished to examine OTS rates for different treatment pressures, indications and chamber types. A synopsis of the peer-reviewed published literature appears in Table 1.

Methods

Written approval for data extraction and review was obtained from the Human Research Ethics Committee of the South Metropolitan Area Health Service, Perth, Western Australia. A prospective database of all OTS has been maintained in the HMU since the opening of the unit in November 1989. A manual (book) database including OTS patient clinical details, treatment profile and OTS description was checked

*** Editor's footnote:**

Many of the reports of OTS are in abstract form only. As per this Journal's policy, these are not included in Table 1 or the references list since there are sufficient data published in the peer-reviewed literature. A full list is available on request from the author.

Table 1
Oxygen seizures during HBOT as reported in previous peer-reviewed publications (O_2 – oxygen; DCI – decompression illness; CO – carbon monoxide; CAGE – cerebral arterial gas embolism; USN TT6 – United States Navy Treatment Table 6)

Author	Treatment pressure (kPa)	Location	Chamber type	Method of O_2 delivery	Patient type	Incidence
Sanders et al[4] 2009	243 + 284	Single unit	Monoplace	Not stated	All	0.03%, 2/5,972 = 1/2,986 CO: 2/171 (1.17%)
Weaver[5] 2006	284	Single unit	Monoplace	O_2-filled monoplace; 7 ventilated	Divers + CAGE	1.11% 1/90
Smerz[6] 2004	PO_2 263–294 Hawaiian Tables	Single unit	Multiplace	Not stated	Divers	0.65%, 14/2,166 = 1/155
Wilkinson et al[7] 2005	284	Single unit	Multiplace	Not stated	Divers/CO/ wounds/other	0.28%, 17/6,084 = 1/358
Yildiz et al[8] 2004	203–284	2 units	Monoplace + Multiplace	Mask usually	Routine HBOT	0.002%, 2/80,679 = 1/40,339
Yildiz et al[9] 2004	239–284	Single unit	Multiplace	Mask usually	Routine and emergency	0.008%, 3/36,500 = 1/12,166
Hampson & Atik[10] 2003	239	Single unit	Multiplace	Head hoods	Routine HBOT	0.03%, 6/20,328 = 1/3,388
Plafki et al[11] 2000	243–253	2 units	Multiplace	Masks or hoods	All (? not CO)	0.04%, 4/11,376 = 1/2,844
Hampson et al[12] 1996	248/284/304 (300 at each)	2 units	Multiplace	? Head hoods but 15% ventilated	CO poisoning	1.8%, 16/900 = 1/56 0.3% at 248 kPa 3.0% at 284 kPa 2.0% at 304 kPa
Weslau & Almeling[13] 1996	243–304	19 units via survey	18 Multiplace 1 Monoplace	Not stated	Routine + divers	0.015%, 16/107,264 = 1/6,704
Sloan et al[14] 1989	304	Single unit	Multiplace	Mask; 18% ventilated	CO poisoning	4.71%, 14/297 = 1/21
Davis[15] 1989	243	2 units	Multiplace	Hoods	All	0.01%, 5/52,758 = 1/10,552
Banham 2011	193–405	Single unit	Multiplace + Monoplace (from 2001)	Hoods in multiplace	All	0.06%, 25/41,273 = 1/1,651

Notes:
1. Published pressures in Ata or depths of seawater equivalent have been converted to kilopascals (kPa).
2. Percentages rounded to two decimal places where possible.

with our electronic database from 27 November 1989 to 26 November 2009, a period of exactly 20 years. Further details of each OTS were cross-checked with the treating physicians' and chamber attendants' documentation in the patients' medical records and also with the HMU technicians' dive logs that are recorded for every chamber compression. Records from each OTS patient pertinent to this study were copied and collated for manual data extraction. No patient identifying information was recorded. Statistical analysis of data was via Fisher's Exact Test. Statistical significance was assumed where $P < 0.05$.

Results

The presence of prodromal symptoms was documented in 10 out of 25 cases of OTS. Prodromal features included twitching, staring gaze, auditory hallucinations, anxiety and irritability. Despite cessation of oxygen (O_2) with prodrome, these cases still proceeded to a convulsion almost immediately. The remaining 15 cases had a convulsion as their first overt evidence of cerebral O_2 toxicity. In total, 25 OTS in 21 patients occurred in 41,273 HBOT exposures (3,737 patients), a rate of 1/1,651 (0.06%) or 6 per 10,000 HBO sessions. Details of the individual cases are shown in Table 2. All of the OTS were generalised in nature. Episodes of OTS prodrome that did not progress to a seizure were not included in this study. The OTS rate per chamber type and treatment pressure, including initial treatment of dysbarism is shown in Table 3.

A further seizure that occurred in this 20-year period was almost certainly a hypoglycaemic event as the blood glucose at the time of seizure was 1.4 mmol L^{-1}. This patient was an insulin-dependent diabetic who had very labile blood sugars during HBOT with frequent drops requiring intervention. This case as such, was excluded from OTS rate

Table 2
Details for 25 oxygen toxicity seizures (OTS) in 21 patients over a 20-year period at (DCI – decompression illness; CO – carbon monoxide; CAGE – cerebral arterial gas embolism; ORN – osteoradionecrosis; STRN – soft-tissue radiation necrosis;

Case	Age	Sex	Indication for HBOT	kPa of OTS	Chamber type	HBOT No.	Total HBOT
1	22	M	Crush injury	284	Multi	1	6
2	58	M	Venous ulcers	284	Multi	3	20
3	28	M	CO poisoning	284	Multi	1	2
4	20	F	CO poisoning	284	Multi	1	3
5	31	M	CAGE (diver)	193	Multi	1	11
6	27	M	Gas gangrene	284	Multi	1	2
7	24	M	CAGE (diver)	284	Multi	1	2
8	36	F	DCI	284	Multi	1	2
9	36	F	DCI	284	Multi	1	2
10	28	F	Crush injury	203	Multi	1	1
11	23	M	Crush injury	284	Multi	2	3
12	68	F	Ischaemic foot	243	Multi	1	1
13	22	M	Compartment syndrome	284	Multi	1	3
14	67	M	Venous ulcers	243	Multi	12	13
15	58	M	ORN prophylaxis	243	Multi	26	27
16	14	M	Crush injury	243	Mono	1	8
17	65	M	ORN prophylaxis	203	Mono	27	27
18	81	F	Non-healing wound	193 (243 kPa table)	Multi	22	28
19	57	M	ORN	243	Multi	15	40
20	52	F	ORN	243	Multi	6	9
21	57	M	ORN prophylaxis	243	Multi	28	30
22	78	F	Diabetic ulcer	243	Multi	11	37
23	78	F	Diabetic ulcer	243	Multi	15	37
24	74	M	STRN	243	Multi	13	30
25	78	F	Diabetic ulcer	193 (243 kPa table)	Multi	37	37

Table 2 (cont.)
Fremantle Hyperbaric Medicine Unit; three patients had more than one OTS; see text for details of treatment tables used USN TT6 – United States Navy Treatment Table 6; EEG – electro-encephalogram; CT – computerised tomography; MRI – magnetic resonance imaging; "End of" implies OTS occurred close to end of an O_2 period)

O_2 Period	Time on O_2 (min)	BSL (mmol L^{-1})	Risk factors	Comments
End of 2nd	48	-	?Nil	Pethidine 75 mg several h prior
End of 2nd	48	-	Nil	Usually at 203 kPa; this HBOT at 284 kPa to fit with other patients; same patient as 14
End of 1st	23	-	Nil	—
End of 2nd	48	-	Nil	—
Start of 4th	125	-	Salt water aspiration	P_aCO_2 48 mmHg several h prior; USN TT6
End of 2nd	48	-	Nil	Afebrile at the time
End of 3rd	59	-	Nil	USN TT6
End of 1st	16	-	Nil	USN TT6; EEG post-HBOT epileptiform; CT brain normal; same patient as 9
End of 3rd	55	-	Nil	USN TT6; same patient as 8
1st	17	-	approx. 1,300 mg pethidine in 30 h	HBOT aborted
End of 1st	24	-	Nil	—
2nd	68	-	Acetazolamide; pethidine	HBOT aborted
End of 1st	25	-	Morphine IV pre-HBOT	Drowsy
End of 2nd	77	-	Prior OTS	Same patient as 2
End of 2nd	84	-	Nil	—
End of 2nd	77	-	?Nil	Flucloxacillin 1g 6 hourly IV
12 mins prior to decompression	108	4.8	Nil	Final post-op HBOT not given
5 mins into 10 min stop at 193 kPa	90	8.1	Prednisolone 10 mg daily; amitriptylline 10 mg nocte	—
1st	34	4.2	Alcohol abuse	—
1st	32	7.4	Nil	Pre-syncope during 9th HBOT; HBOT ceased; EEG + MRI brain normal
End of 2nd	85	-	Nil	Diazepam 10mg pre-HBOT for claustrophobia
End of 2nd	87	4.9	Insulin	Same patient as 23 and 25; morbid obesity
End of 1st	40	6.7	Insulin; prior OTS	Same patient as 22 and 25
End of 1st	44	5.2	Nil	—
On decompression	90	8.6	Insulin; prior OTS	Same patient as 22 and 23

Table 3
Oxygen toxicity seizures (OTS) rate per chamber type and treatment pressure including initial treatment of dysbarism; an OTS that occurred during decompression in a table is included in the data for that table (USN TT6 – United States Navy Treatment Table 6)

Treatment table/pressure (kPa)	HBOT sessions	OTS number	OTS %	OTS rate
Multiplace chamber	36,068	23	0.06	1/1,568
COMEX 30	7	0	0	0
USN TT6	593	4	0.67	1/148
203	15,732	1	0.01	1/15,732
243	17,847	11	0.06	1/1,622
284	1,889	7	0.37	1/270
Monoplace chamber	5,205	2	0.04	1/2,602
USN TT6	121	0	0	0
193	36	0	0	0
203	1,780	1	0.06	1/1,780
243	2,786	1	0.04	1/2,786
284	482	0	0	0
Totals for treatment type				
COMEX 30	7	0	0	0
USN TT6	714	4	0.56	1/178
193	36	0	0	0
203	17,512	2	0.01	1/8,756
243	20,633	12	0.06	1/1,719
284	2,371	7	0.30	1/339
TOTAL	41,273	25	0.06	1/1,651

analysis, although it is possible that the seizure was due to a combination of hypoglycaemia and hyperoxia.

PRESSURE

The rate of OTS at 203 kPa or less (2 /17,548) versus OTS occurring at all treatment pressures greater than 203 kPa (23/23,725) shows a significant difference in OTS rate between the two treatment pressures ($P < 0.001$, odds ratio (OR) 8.5, 95% confidence intervals (CI) 2.0 to 36.1). Comparison of the OTS rate at 203 kPa versus 243 kPa shows a significantly lower rate at 203 kPa ($P = 0.028$, OR 5.1, 95% CI 1.1 to 22.8). Exclusion of those with their first HBOT for dysbarism (4/721) from the above data still shows statistical significance with 2/17,548 versus 19/23,004 ($P = 0.001$, OR 7.3, 95% CI 1.7 to 31.1).

MULTIPLACE VERSUS MONOPLACE CHAMBER

Overall, 36,068 of 41,273 cases were treated in a multiplace chamber; these cases comprised 23 of the 25 OTS that occurred (difference not significant, $P = 0.762$).

OTS TIMING

The most frequent timing for OTS (9/25 cases) was in the final third of the second O_2 period, followed by OTS occurring at the end of the first O_2 period (6/25 cases). In divers being treated for dysbarism, four OTS occurred after the second O_2 period (one during the third O_2 period, one during a table extension in their fourth 20-minute O_2 period at 284 kPa and two at 193 kPa during the decompression phase of HBOT). There were three cases that occurred in the first two thirds of the first O_2 period and one in the same period of the second O_2 period. The other OTS in our series was in the monoplace chamber at 203 kPa, occurring near the end of this table. The accrued time on oxygen to the time of the OTS (excluding air breaks) is listed in Table 2. Analysis of these times shows a mean of 58 min, a mode and median of 48 min and a range of 16 to 125 min.

CO POISONING

There were 1,088 HBOT for acute CO poisoning, 409 first treatments and 679 follow-up HBOT, all at 284 kPa. Two OTS occurred during a first treatment, 0.49%. The overall OTS rate was 0.18%. The different between first and subsequent HBOT was not statistically significant ($P = 0.142$, 95% CI 0.09 to 1.95).

DYSBARISM

Dysbaric injuries comprised decompression illness (DCI) in divers and iatrogenic cerebral arterial gas embolism (CAGE). The OTS rate for the initial treatment of dysbarism (4/721) with either USN TT6 or COMEX 30 (Compagnie Maritime

d'Expertises) table was not different to that for follow up with United States Navy Treatment Table 5 (USN TT5) (0/731) (P = 0.061). The profile for the first two oxygen periods in a USN TT5 is identical to that of a USN TT6.

RECURRENT OTS

Three of the 21 OTS patients had a recurrent OTS, with one of these having a third convulsion. The first patient (listed as events 2 and 14 in Table 2) was receiving HBOT for non-healing venous leg ulcers. His first OTS occurred in the third of a course of 20 HBOT in 1992, without prodrome. His recurrent seizure was in 2001 at 243 kPa during his twelfth HBOT session for recurrence of non-healing venous leg ulceration. This OTS was immediately preceded by facial grimacing. His next treatment was modified to reduce the likelihood of recurrent OTS by increasing the duration of the air break from 5 to 10 minutes, but the patient became extremely anxious about the possibility of a further OTS and declined further HBOT.

The second patient (8 and 9 in Table 2) was a 36-year-old, experienced female scuba diver who had onset of constitutional and musculoskeletal symptoms suggestive of DCI. She was treated with a USN TT6 in the multiplace chamber. An OTS occurred near the end of the first 20-minute O_2 period and again towards the end of the third, despite the administration of diazepam 4 mg IV during the first OTS. Further diazepam was administered and the table completed without incident. Both OTS were preceded by a brief prodrome (anxiety and auditory hallucinations) and occurred despite immediate O_2 delivery cessation. The patient had a 203 kPa HBO treatment the next day without incident. In view of these seizures, she was investigated for an underlying seizure disorder. A computerised tomography scan of her brain was normal but an electroencephalogram (EEG) performed 12 days post seizure was reported as showing findings typical of generalised epilepsy. However, a repeat EEG eight years later was normal. Recent contact with the patient revealed that she had had no further seizures, had resumed diving and had also been recompressed again on a USN TT6 in 2005 without incident.

The third patient (22, 23 and 25 in Table 2) with recurrent OTS was an obese Type-2 insulin-dependent diabetic with a chronic non-healing leg ulcer having HBOT at 243 kPa. Her three OTS all occurred without prodrome and with a normal BSL. Following the second, the decision as to whether to continue with HBOT was discussed with the patient, and as she was very keen to continue, it was decided to give an extra 5-minute air break in the middle of each of the two scheduled 45-minute oxygen periods at 243 kPa. Despite this, the patient had a further OTS at 193 kPa (on decompression from 243 kPa) during HBOT session number 37, and HBOT was discontinued. No further investigation of her seizures was undertaken. Of the 18 patients with a single episode of OTS, all but two had further HBOT ranging from 1 to 25 sessions.

OTS TREATMENT

Pharmacological treatment with benzodiazepines was given in eight cases, and a further patient who was an insulin-dependent diabetic was given glucagon empirically pending the result of a finger-prick blood glucose, which was within the normal range.

Discussion

OTS PRODROME

Prodromal symptoms of OTS were described in detail by Donald and included lip twitching, visual changes, nausea, vertigo, auditory hallucinations and spasmodic breathing.[2] Damant and Phillips in 1933 in their self-experimentation breathing O_2 at 405 kPa both had tremor of the lips, which resolved in one by immediately reverting to air breathing; the other, however, progressing to convulsions and unconsciousness despite reverting to air breathing.[16] Less than half (10/25) of OTS patients in this series were noted to have warning signs of an impending OTS. Prodromal features included twitching, vacant or staring look, anxiety, auditory hallucinations and nausea. Despite immediate cessation of O_2 upon recognition of an impending OTS, there was a rapid progression to a seizure. However, the majority of patients in this series had an OTS as their first manifestation of cerebral O_2 toxicity.

OTS TREATMENT PRESSURE

The rate of OTS at 203 kPa or less (2/17,548) was statistically significantly less than that at treatment pressures greater than 203 kPa (23/23,725). This is consistent with many reports, dating back to the time of Donald, showing an increased OTS rate with increasing pressure.[2] Seizures occurred at all treatment pressures, the lowest being 193 kPa; but only during depressurisation from a higher treatment pressure, (203, 243 and 284 kPa). The only exception was during the COMEX 30 table, used only seven times during this period. That no OTS occurred with COMEX 30 treatments is likely due to the small number of patients treated with this table in this series. At 405 kPa and until depressurisation to 284 kPa, patients breathe 50:50 Heliox, with an FiO_2 of only 203 kPa. The rest of the COMEX table is, however, completed with periods of 100% O_2 breathed at 284 then 223 kPa.

Almost all OTS in this series occurred at a treatment pressure of 243 kPa or more. There were only two cases of OTS during a 203 kPa table; one in the multiplace chamber and one in the monoplace. The OTS in the multiplace at 203 kPa occurred in a 28-year-old female who sustained a crush injury to her right hand for which she had received 1,300 mg of pethidine analgesia over the 30 h prior to HBOT. Pethidine's metabolite norpethidine is recognised

to cause seizures.[17] Norpethidine may have potentiated the CNS toxicity of O_2 in this patient, leading to the OTS early in the first O_2 period at a pressure at which OTS is cited to be uncommon.[18]

The patient with OTS in the monoplace chamber at 203 kPa was a 65-year-old man with previous radiotherapy post-laryngectomy, being treated with HBOT as prophylaxis to prevent mandibular osteoradionecrosis (ORN) post dental extraction. The treatment table was 10:120:06 (10 metres' sea water (msw) depth equivalent [203 kPa], 120 min of O_2 with no air break and 6 minute decompression). He had had 26 prior similar HBOT sessions without problems. At 108 min into the treatment, he had a grand-mal type seizure lasting approximately 3 min. No specific therapy was given aside from ceasing the O_2 supply to the monoplace chamber and immediately flushing it with air. There were no apparent predisposing factors to OTS and his finger-prick blood glucose was normal (4.8 mmol L^{-1}) upon surfacing the chamber. As his dental clearance wounds were now well healed, it was decided to stop his HBOT at this point.

Twelve OTS occurred during treatments at 243 kPa in 10 patients. HBOT administered at 243 kPa has been the routine treatment compression pressure in Fremantle Hospital HMU since February 1999; the change from 203 kPa to 243 kPa was to comply with the Marx and Wilford-Hall treatment protocols.[19,20] Of the 12 OTS at 243 kPa, only two occurred with the first HBOT. One patient (Case 12) had been medicated with acetazolamide and pethidine, both recognised as potential precipitating factors for OTS.[17,21] The other OTS occurring at 243 kPa during a first treatment was in a 14-year-old male with a crush injury to his right ankle (Case 16), being treated in the monoplace chamber on a 14:90:08 table (two 45-minute periods on oxygen with a 5-min air break via mask at 243 kPa and 8 min decompression). The OTS occurred at 77 min on oxygen. The chamber was flushed with air, the patient breathed air for 15 min then resumed oxygen for decompression. He had a further 14:90:08 HBO treatment the next day, then six 10:120:06 treatments, all without incident.

OTS TIMING

It has been suggested that the majority of seizures occur in the final third of the second O_2 period. In the present series, this was also the case (9/25 cases), followed by OTS occurring at the end of the first O_2 period (6/25 cases). The variation in timing of OTS both regarding the timing during a treatment and the HBOT session in which it occurred are again consistent with the observations of Donald who reported a wide variation in susceptibility between subjects as well as an enormous individual variation.[2] As such, there should be a heightened awareness of the increased likelihood of an OTS in the approach to an air break, with the understanding, however, that an OTS may occur earlier in the period of O_2 breathing.

METHOD OF O_2 DELIVERY

We did not observe a difference in OTS rate between multiplace and monoplace treatments. Almost all patients in this series had O_2 delivered via a head hood when compressed in a multiplace chamber, apart from a few, usually divers, who preferred using a mask. Mask delivery of O_2 was also used for patients in the monoplace chamber when at pressures greater than 243 kPa. The design of head hood used over the 20 years has remained constant, with the inflow and outflow tubing ports adjacent to each other at the front. Monoplace HBOT at 284 kPa has O_2 delivered via mask in an air-filled chamber, whereas at 243 kPa or less, the monoplace is compressed with 100% O_2 and air breaks are via mask. It was not possible to compare OTS rates between these various modalities of oxygen delivery in our series. OTS is a relatively rare event and hence extremely large numbers of patients and treatments would be required to show whether any one modality of treatment (mask, hood, monoplace and multiplace) was safer than any other. Also, most higher pressure and longer duration tables, such as USN TT6 and COMEX 30 treatments occurred in the multiplace chamber.

RECURRENT OTS

Only three of 21 patients had a recurrent OTS. Of these, one had a recurrence in the same HBOT session, one during the same course of HBOT (3 OTS in total) and one in two HBOT courses nine years apart. Continuation of HBOT occurred in 16 without further OTS, indicating that ongoing HBOT post OTS is not necessarily contraindicated. Patients who do have recurrent OTS should have a risk assessment as to the need for continuation of HBOT, a review of any factors, especially medications that may predispose them to OTS and consideration of modification to their treatment table to reduce their OTS risk. This could include increasing the frequency of air breaks, or reducing the treatment pressure or both. The use of benzodiazepines or other anticonvulsants may be considered; however, diazepam 4 mg IV did not prevent a recurrent OTS in the diver discussed above.

OTS TREATMENT

Eight of 11 patients with OTS prior to the year 2000 were treated with intravenous benzodiazepines (diazepam or midazolam) in addition to oxygen cessation. One patient (Case 5) required multiple doses of IV benzodiazepines to control post-seizure agitation. From the year 2000, our Unit's OTS protocol was amended such that benzodiazepines were not given routinely in response to OTS, but only for prolonged seizures or post seizure agitation, being required in none of these 12 patients.

With the recognition that HBOT may cause hypoglycaemia in patients with diabetes, whether insulin-dependent or non-insulin-dependent,[22,23] it became routine in our HMU

from 2003 to document a finger-prick blood glucose in such patients immediately before and after each HBOT session and if any hypoglycaemic symptoms or OTS occurred during HBOT. Only one patient with a seizure in the chamber had evidence of hypoglycaemia, and, as such, the remainder of OTS were attributed to and treated as hyperoxic seizures.

CO POISONING OTS

Both OTS in CO-poisoned patients occurred during their first HBOT. This is consistent with a recent review of the data by Sanders et al. who reported that 100% (18/18) of OTS occurred during the first HBOT for CO toxicity compared to 2/8 OTS (25%) during the first HBOT for non-CO related conditions.[4] Sanders et al. commented that they were unable to "*determine whether HBOT increases the risk of seizure in CO-poisoned patients or whether the risk of seizures is simply the result of the CO poisoning*".[4] The rate of OTS in CO patients has been reported variously from 0% to 4.7%.[24] Our series rate of 0.18% OTS (but 0.49% of first HBOT) compares favourably to the average in the reviewed medical literature: 1.45% (32 OTS/2,200 HBOT) for CO-poisoned patients versus 0.008% (8 OTS/106,158 treatments) for HBOT for indications other than CO poisoning. It is also very similar to the 0.2% reported by Wilkinson (which is not included in Sanders' analysis).[4,7] There was no significant difference in the OTS rate for the first versus follow-up HBOT for CO poisoning.

DYSBARISM OTS

Our overall rate of 0.28% for dysbarism treated at 284 kPa compares favourably to the range of 0.49% to 1.11% reported by others.[5–7] The rate of OTS for patients for their initial treatment for dysbarism in this series (4/721, 0.56%) was less than that reported by another Australian hyperbaric unit of 1.64% (7/427).[7] Of these, four occurred during USN TT6 and the other three during an 18:60:30 table. Both of these treatment tables have identical initial three 20-minute O_2 periods. This difference in OTS rates between the two hyperbaric units in the same country treating similar diving populations is unexplained. Interestingly, there were no cases of OTS in any of the iatrogenic or diving-related CAGE reported by the Adelaide unit. There were no cases of OTS documented in the 1,632 HBOT for dysbarism following their first compression despite the fact that the profile for the first two O_2 periods of a USN TT5 is identical to that of the USN TT6. This may be explained simply by the relatively low number of patients in this cohort.

The higher rates of OTS for the initial treatment of dysbarism and CO poisoned patients at 284 kPa compared to elective treatments at 243 kPa warrant that appropriate information regarding this is provided to such patients during the process of gaining informed consent for their HBOT. In addition, attendants and technicians should have a heightened awareness of the risk of OTS and the recognition of any prodromal O_2 toxicity symptoms with consequent cessation of O_2 in an attempt to avoid OTS in this group of patients.

ASSOCIATED RISK FACTORS

No patient with an OTS had a documented past history of seizures or a fever (temperature > 37.5^{O}C) during the HBOT session where the OTS occurred.

Conclusions

This study is the longest longitudinal study yet published of all OTS occurring in all patients for all treatment indications in a single hyperbaric unit.

It demonstrates similar rates of OTS for HBOT administered in Fremantle Hospital's Hyperbaric Medicine Unit to those described elsewhere.

The rate of OTS occurring at a treatment pressure of ≤ 203 kPa is significantly less than for pressures > 203 kPa.

The OTS rate at 243 kPa, our most common treatment pressure, was 12/20633 (0.06%) or 1 in 1,719 treatments.

The OTS rates for the treatment of dysbarism and CO are much higher than for routine HBOT and, as such, appropriate vigilance should be maintained and the relative risk explained to patients or their close relatives prior to compression for these indications.

Prodromal symptoms of cerebral O_2 toxicity are not witnessed prior to the onset of an OTS in a majority of patients.

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The effects of increased pressure, variation in inspired gases and the use of a mask during dry chamber dives on salivary cortisol in professional divers

Janne Tikkinen, Ari Hirvonen, Kai Parkkola and Martti A Siimes

Abstract

(Tikkinen J, Hirvonen A, Parkkola K, Siimes MA. The effects of increased pressure, variation in inspired gases and the use of a mask during dry chamber dives on salivary cortisol in professional divers. *Diving Hyperb Med*. 2011 December;41(4):211-215.)

Introduction: Stress activates the hypothalamic-pituitary-adrenal axis resulting in measurable changes in hormone levels in blood or saliva in humans. We aimed to find out if professional divers expressed any change in salivary cortisol levels during a simulated dive to 608 kPa (50 meters' sea water) in a hyperbaric chamber. Furthermore, we investigated the effect of wearing a mask or modifying the breathing gas during decompression.

Methods: We investigated 89 Navy and Coast Guard male divers. The divers were randomised into three groups for decompression by inspired gas and the use of a mask. The saliva samples were collected before and approximately 3 minutes after the hyperbaric test.

Results: Salivary cortisol levels decreased from a mean (SD) of 16.0 (8.1) nmol L^{-1} pre-dive to 10.3 (5.0) nmol L^{-1} post-dive ($P < 0.01$). Cortisol values did not relate to the anthropometric and physical fitness characteristics of the divers or to increased pressure, variation in inspired gases or the use of a mask. The individual variation in cortisol values was large.

Conclusions: These findings are in line with previous studies demonstrating large individual variations in salivary cortisol. Our findings suggest that professional divers are well adapted to these hyperbaric conditions. However, there continues to be a need to identify divers sensitive to stress caused by diving and the hyperbaric environment.

Key words

Stress, endocrinology, hyperbaric oxygen, equipment, military diving, occupational diving, hyperbaric research

Introduction

Stress activates the hypothalamic-pituitary-adrenal axis resulting in measurable changes in hormone levels in blood or saliva in humans. Cortisol level in saliva ranges from less than 2 nmol L^{-1} to more than 100 nmol L^{-1}.[1] According to reference ranges by Aardal and Holm, salivary cortisol varies from 3.5 to 27.0 nmol L^{-1} at 0800 h.[2] Further, cortisol levels are age-dependent and vary in circadian fashion after the age of one year. The cortisol levels are typically high in the morning upon waking, increase 50–60 % in the first 30–45 min after awakening then drop over the next few hours and finally decline more slowly to reach the lowest point at 2400 h.[3] Salivary cortisol values vary considerably both within and between individuals and, according to Casals et al., a change greater than 104% between two measurements is needed to be considered as significant.[4] Two- to three-fold elevations above baseline measurement of salivary cortisol were found in men preparing for skydiving.[5] Arithmetic calculations in a noisy environment doubled cortisol values, and the Trier Social Stress Test, which consists of delivering a speech and performing mathematical calculations in front of an audience, led to two- to four-fold increases.[6,7]

Similar salivary cortisol changes have been noted with pharmacological and psychological stimuli.[8] Psychological stressors have been reported to induce cortisol release only if an emotionally negative stressor is employed.[9] In addition, a correlation has been demonstrated between the state of anxiety and cortisol measures in phobic situations.[10] Physical fitness may be important to the cortisol response, since trained men had higher pre-stress cortisol values and a faster fall than untrained.[11] Recent data in soldiers and emergency rescue recruits demonstrated significantly higher cortisol secretion in those suffering from a phobia to a protective mask.[12]

Limited data based on small numbers of subjects have suggested that although divers may show evidence of a generalised hormonal stress response under some conditions, elevated air pressure itself may not induce this response. For example, Davis et al. found a strong correlation between raised plasma cortisol levels and open-water deep dives.[13] They considered it to be a response to psychological stress. Smith et al. demonstrated that cold-water immersion at 304 kPa elevated plasma cortisol and ACTH levels in sixteen divers.[14] In another study, serum concentrations of cortisol decreased in divers who were exposed to air and oxygen at 254 kPa.[15] Similarly, a Japanese study of six qualified divers who were exposed to heliox at 3.65 MPa showed no changes in salivary cortisol.[16] In another study, hyperoxia did not cause significant changes in cortisol values, whereas using a face mask resulted in a prolonged elevation in cortisol values compared to breathing chamber air.[17] Lower levels of physical fitness have been associated with more pronounced hormonal responses to diving pressure in five professional divers.[18]

In this study we aimed to find out if professional divers expressed changes in salivary cortisol levels during a simulated dry dive to 608 kPa (50 meters' sea water, msw) in a hyperbaric chamber. Furthermore, we investigated the effect of wearing a mask or modifying the breathing gas during decompression.

Methods

SUBJECTS

The study protocol was reviewed and approved by the Ethical Committee of the University Hospital of Helsinki and accepted by the Headquarters of the Finnish Defence Forces. One-hundred-and-five professional Navy and Coast Guard male divers undergoing their annual medical check-up gave their written informed consent to participate following a detailed explanation in writing of the study procedures.

Prior to their medical check-ups, the divers completed a questionnaire seeking information on their diving experience (year of basic training, hours of diving) and the result of the latest Cooper's 12-minute running test. Height and weight were recorded and body mass index (BMI) calculated. Proportional body fat (PBF) was calculated using the Body Impedance Analysis InBody 7200 (Biospace Co, South Korea). Maximal oxygen (O_2) uptake (expressed as ml min^{-1} kg^{-1}) was measured using a treadmill ergometer (Schiller CS-200, Schiller AG, Switzerland) and inspired and expired gas analysis (PowerCube Ergo, Ganshorn Medizin Elektronic GmbH, Germany). The maximum O_2 uptake had to be estimated in six divers instead of direct analysis of the breathing gases because of calibration difficulties.

DIVE PROTOCOL

We exposed two divers at a time to a pressure of 608 kPa (50 msw) in a dry multiplace chamber. The exposure was part of their annual medical check-up. The speed of compression was 9 ± 1 msw min^{-1} and the target pressure was reached in 6 ±1 min. The divers remained at depth for 3 minutes. Decompression was based on a depth of 51 msw and bottom time of 15 min (Finnish Navy, DCAP-FINN), with decompression stops at 9, 6 and 3 msw for 7, 2 and 8 min, respectively. The subjects breathed either the chamber air or from a mask and built-in-breathing-system (BIBS). The breathing gas in the BIBS was changed from air to O_2 for the divers in the O_2 group (see below) at 304 kPa on ascent (15 ±1 min after the onset of the test dive).

RANDOMISATION

We prepared 105 envelopes containing the study design, questionnaire, consent form and the research code forms. When the divers attended their annual medical check-up, each of them picked out one of the shuffled envelopes from the box. The research codes divided the divers initially into three groups for decompression:

- Group 1: the divers breathed chamber air throughout the dive (n = 22 analysed)
- Group 2: the divers breathed air through the mask and BIBS throughout the dive (n = 32 analysed)
- Group 3: the divers breathed air through the mask and BIBS during the dive but the breathing gas was switched to O_2 at 304 kPa during decompression (n = 35 analysed).

Of the 105 divers entered into the study, 89 had a complete set of pre- and post-dive cortisol and VO_2max measurements.

SALIVARY CORTISOL

The saliva samples were collected before and after the hyperbaric chamber test. The tests were performed between 0800 and 1100 h. The divers avoided vigorous physical exercise and intake of any food for two hours prior to the test.[19] To collect a saliva sample, the diver chewed a cotton swab for two min (Salivette, Sarstedt, Numbrecht, Germany). The swabs were collected directly into sampling tubes, which were stored frozen at -21^{O}C prior to analysis. The half-life of salivary cortisol is relatively long (58–113 min) and it peaks 20–30 min after the onset of a stressful stimulus.[19] Therefore, post-dive samples were taken at approximately 3 minutes after reaching the surface. An increase in salivary cortisol of more than 100% between the pre- and post-dive concentrations was considered as a significant change. For technical reasons, not all the cortisol samples could be analysed on five non-mask chamber air subjects.

The salivary cortisol levels were measured using a commercially available luminescence immunoassay (Cortisol Saliva LIA, IBL Immuno-Biological Laboratories, Hamburg, Germany). This assay is based on the competition principle and microtitre plate separation. Briefly, an unknown amount of cortisol present in the sample and a fixed amount of enzyme-labelled cortisol compete for the binding sites of the antibodies coated onto the wells. After 3 h incubation, the wells are washed to stop the competition reaction. Once the luminescent substrate solution is added, the relative luminescence units (RLUs) can be read within 10–40 min, the concentration of cortisol being inversely proportional to the measured luminescence. Measuring range of the method is 0.43–110 nmol L^{-1}. The coefficients of variation of intra- and inter-assay of the method are 5% and 8% respectively.

STATISTICS

The data are presented as means (SD). The IBM SPPS 19 computer package was used for all statistical analyses. The Kolmogorov-Smirnov test was used to assess the normality of distribution of the cortisol values. Skewed values were normalised, and the t-tests were performed after logarithmic transformation (natural log). Relationships between pre-dive

or post-dive cortisol and various variables were assessed with Pearson correlation analysis. The association of the decompression gas and use of a mask with cortisol levels was determined by an analysis of variance (ANOVA). A pre-study power analysis was not performed.

Results

Of the 105 divers entered into the study, 89 had a complete set of pre- and post-dive cortisol and VO_2max measurements. Demographic data are provided in Table 1. As expected, the individual variation in salivary cortisol values was large. The pre-dive cortisol concentration ranged from 2.6 to 39.6 nmol L^{-1} with a mean (SD) of 16.0 (8.1) nmol L^{-1} and the post-dive cortisol ranged from 3.4 to 29.1 nmol L^{-1} with a mean of 10.3 (5.0) nmol L^{-1}. The decrease in cortisol over the dive was statistically significant ($P < 0.01$). Of the 89 divers, 15 experienced an increase of cortisol concentration during the experiments, only three of these exceeding a rise of more than 100%. On the other hand, 21 divers showed a decrease in cortisol level of more than 50%. The pre- and post-dive salivary cortisol values are plotted against each other in Figure 1.

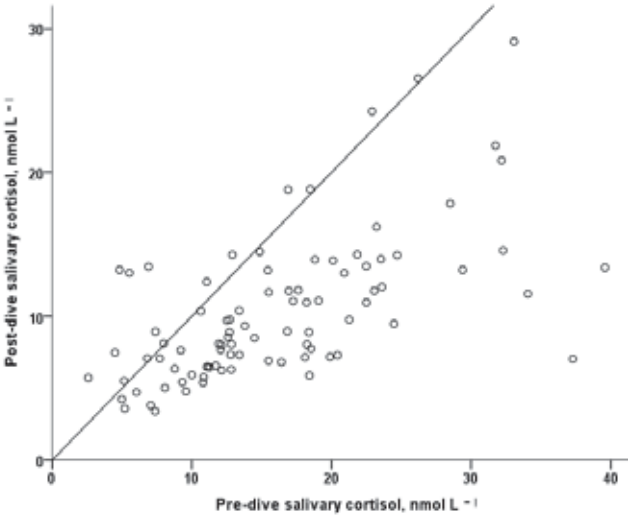
Using Pearson correlation coefficients, we found no statistically significant associations between the pre- or post-dive cortisol values and the age, weight, BMI, PBF, Cooper's 12 minutes running test, VO_2max or the diving experience of the divers. Neither were the relative or absolute changes in cortisol associated with the age, anthropometric or physical fitness parameters, or the diving experience of the divers.

The findings were similar in all divers within the three groups randomized for decompression gas (Figure 2). The mean pre-dive cortisol values decreased from 16.8 (8.4) nmol L^{-1} (range 2.6–34.0) to 10.7 (5.0) nmol L^{-1} (range 3.8–24.0) ($P < 0.01$) in the divers inhaling O_2 through mask and from 15.3 (8.0) nmol L^{-1} (range 5.0–39.6) to 9.6 (4.7) nmol L^{-1} (range 3.4–26.5) ($P < 0.01$) in the divers inhaling air through mask and from 15.9 ±7.8 nmol L^{-1} (range 4.5–33.1) to 10.8 (5.5) nmol L^{-1} (range 4.8–29.1) ($P < 0.01$) in the divers inhaling chamber air without mask. The ANOVA revealed no differences between the pre-dive and post-dive cortisol values in the three groups.

Table 1
Demographics of 89 professional divers studied

Parameter	Mean	SD	Range
Age (years)	36.0	(8.5)	20–50
Body weight (kg)	84.2	(8.2)	66–110
Body mass index (kg m^{-2})	25.9	(2.2)	21.7–30.9
Proportional body fat (%)	16.0	(5.1)	7.3–29.1
12-min run distance (m)	2900	(240)	2200–3550
VO_2max (ml min^{-1} kg^{-1})	54.8	(8.1)	34.4–69.4
Diving experience			
Years	12.0	(8.1)	1–26
Hours	521	(603)	14–3300

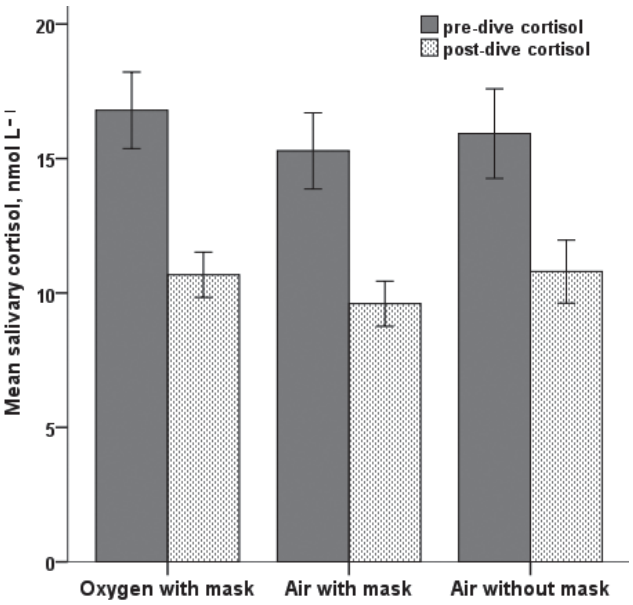
Figure 1
The pre- and post-dive salivary cortisol values (nmol L^{-1}) in 89 professional divers; the line of identity is drawn to separate those with increased/decreased values



Discussion

The study aimed to find out if professional divers expressed any change in salivary cortisol values as a marker of stress during a simulated dive to 608 kPa in a hyperbaric

Figure 2
The mean pre- and post-dive salivary cortisol values (± SEM) of the 89 divers divided into three groups according to the breathing gas used and its mode of delivery during decompression



chamber. We used salivary cortisol because the method is non-invasive and does not itself activate the hypothalamic-pituitary-adrenal axis. Salivary cortisol is often used as a marker for stress but has a large variation both biologically and for methodological reasons.[3,19] We aimed to reduce the variability by utilising the same commercial preparation and laboratory for collecting and analysing the saliva samples. Further, the samples were collected at the same time of the day and year. The divers also received the same instructions prior to their tests. The individual variation of cortisol values was large, but they were within the normal range in the majority of individuals and in line with previous studies.[2,12,18]

We observed no evidence that absolute values or relative changes in salivary cortisol levels correlated with the physical fitness of the divers as in a previous study.[11] However, these Navy and Coast Guard divers were a selected, homogeneous group with good to excellent physical fitness compared to the general population. This may explain the lack of an association.

Our findings of lower post-dive cortisol values are in line with the results of the previous study at 254 kPa.[15] Only a few of the 89 divers had an increment of more than 100 % in their salivary cortisol concentrations and in most cases the elevation, if present, was marginal (see Figure 1). On the other hand, we observed a decrease greater than 50% in 21 divers. The decrease might be in part due to normal circadian variation.[3] Nevertheless, it is known that anticipation of an emotionally stressful situation can induce cortisol secretion.[10] We speculate that, at least in some of the divers, anticipation of the forthcoming dry dive and the various tests may have been sufficient to stimulate an increase in cortisol release. Some of the pre-dive cortisol values were higher than the upper limit of the Aardal and Holm reference range for morning salivary cortisol.[2] Studies in athletes such as paragliders, tennis players, wrestlers, judo wrestlers and swimmers have demonstrated increases in pre-competition plasma or salivary cortisol concentrations.[20–24]

The use of a mask during the chamber test or the nature of the inhaled gas during decompression (air or O_2) did not have any influence on the cortisol levels. The subjects were professional divers who are accustomed to heavy masks which would normally be uncomfortable and cause stress. Our observation that hyperoxia was not related to the change in the cortisol values supports the findings of two previous studies.[15,16]

The main limitations of our study are the lack of a control group, utilisation of only one marker for stress and use of only single pre- and post-dive samples. However, we were unable to expand the study design since the experiments were performed in combination with the clinical medical check-ups of the divers during a working day.

Our earlier experiences with measuring subjective psychological stress responses by a self-reporting method (unpublished observations) in professional divers have been discouraging. Over the last 10 years, none of the divers reported any sensations of anxiety, nervousness, anger, palpitations, etc., during their annual dry chamber dives to 608 kPa. There continues to be a need to identify divers sensitive to stress caused by diving and the hyperbaric environment. In future studies, a combination of markers of activation of the hypothalamic-pituitary-adrenal axis and autonomic nervous system responses would be useful.

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Conflict of interest: none

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Review article

Predicting performance in competitive apnea diving. Part III: depth

Erika Schagatay

Abstract

(Schagatay E. Predicting performance in competitive apnea diving. Part III: depth. *Diving Hyperb Med*. 2011 December;41(4):216-228.)

Part I described the physiological factors defining the limits of static apnea, while Part II examined performance in dynamic distance swimming. This paper reviews the factors determining performance in the depth disciplines, where hydrostatic pressure is added to the stressors associated with apnea duration and physical work. Apneic duration is essential for performance in all disciplines, and is prolonged by any means that increases gas storage or tolerance to asphyxia, or that reduces metabolic rate. For underwater distance swimming, the main challenge is to restrict metabolism despite the work of swimming, and to redirect blood flow to allow the most vital functions. Here, work economy, local tissue energy and oxygen stores, anaerobic capacity of the muscles, and possibly technical improvements will be essential for further development. In the depth disciplines, direct pressure effects causing barotrauma, the narcotic effects of gases, decompression sickness (DCS) and possibly air embolism during ascent need to be taken into account, as does the risk of hypoxia when the dive cannot be rapidly interrupted before the surface is reached again. While in most deep divers apneic duration is not the main limitation thus far, greater depths may call for exceptionally long apneas and slower ascents to avoid DCS. Narcotic effects may also affect the ultimate depth limit, which elite divers predict to be around 156 metres' sea water. for constant weight with fins. To reach these depths, serious physiological challenges have to be met, technical developments are likely to be needed and safety procedures must be developed concomitantly.

Key words

Breath-hold diving, hypoxia, exercise, cardiovascular, respiratory, physiology, safety, review article

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Introduction

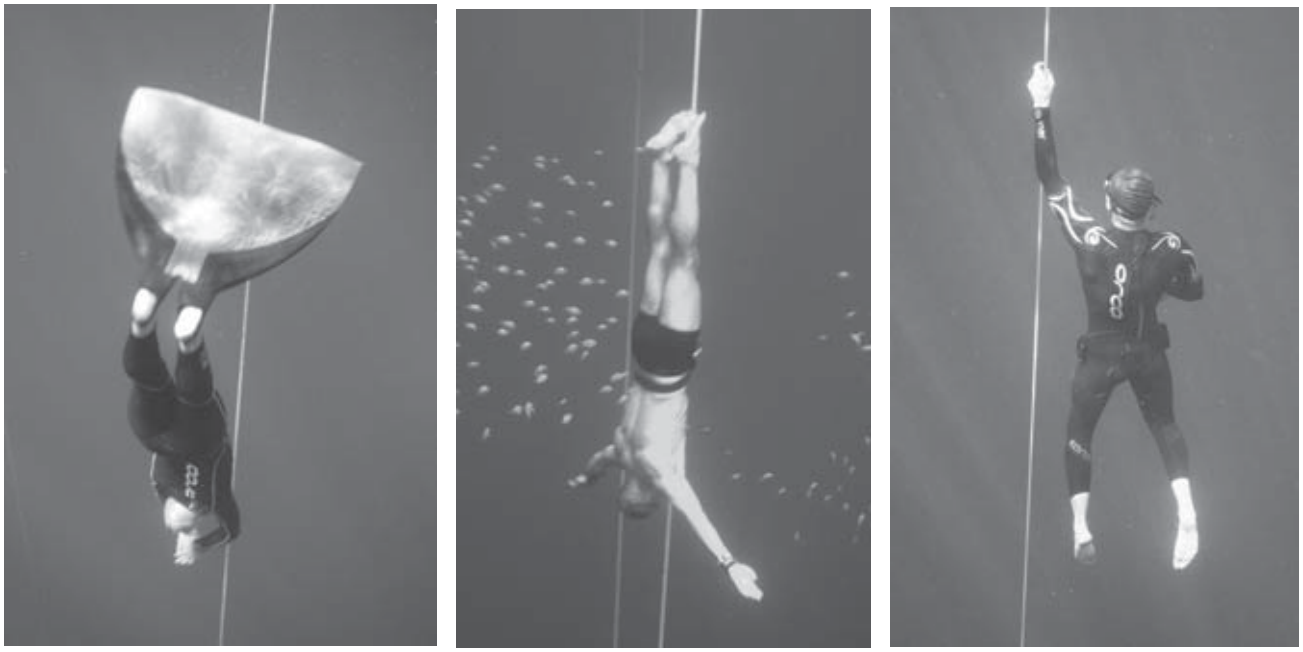
While my two preceding reviews have dealt with the human ability to make long apneas during rest in the competitive discipline of 'static apnea' (STA) and to produce long underwater swims in those of 'dynamic apnea' (DYN and DNF),[1,2] this final review of the pre-requisites for human competitive apnea diving performance will focus on deep diving. The factors associated with apnea duration and physical work are also essential for reaching great depths, but for a detailed review the reader should refer to the previous papers. Here the new factors added with increasing depth will be reviewed.

Several previous papers describing human apneic deep diving have focused on the medical problems and worst-case scenarios that may be associated with such activities.[3–7] However, the fact is that most deep dives by trained athletes are done without any harm to the divers, and the number of divers reaching a depth of over 100 metres' sea water (msw) depth on one breath in unassisted dives is now approaching two dozen, and in assisted dives is even higher. Most physicians and researchers understandably view deep apnea diving largely 'from the outside' and typically describe the threats that the divers are exposed to as events happening somewhat randomly and without the diver's previous awareness or preparedness. After years of study, I believe, on the contrary, that elite divers are well aware of the obstacles they have to overcome to accomplish such performance at an acceptable level of safety, and how to prepare their bodies and minds to sustain such extreme activities. It is correct that there are a series of important limitations to human apneic deep diving, and it can certainly be dangerous if the proper safety measures are not in place. But let us study the diving from the divers' viewpoint to see what can be done to limit risk, to overcome some of the physiological problems and to extend human depth limits. Much of this research is very recent and is only to be found in meeting abstracts and as unpublished observations.

Deep-diving disciplines

The regular competition disciplines of deep diving are 'constant weight with fins' (CWT), 'constant weight without fins' (CNF), in both of which the diver swims down and up without changing their ballast, and 'free immersion' (FIM) in which the diver pulls her/himself down and up along a vertical rope (Figure 1 a–c). There are also two assisted disciplines, which occur only as single events announced by a diver challenging the existing record. These are 'variable weight' (VWT), in which the diver is pulled down by ballast that is left at the bottom and then swims up, and 'no limit' (NLT) where a ballast is used on the way down and lifting bags pull the diver back to the surface. The focus of this

Figure 1
The three unassisted disciplines of deep diving
a. Constant weight with fins (CWT) b. Constant weight without fins (CNF) c. Free immersion (FIM)



review will be on the unassisted competition disciplines, with some examples from the assisted disciplines, mainly because these records can help predict the ultimate limits in unassisted deep diving. Contrary to popular belief, competitions in the three unassisted disciplines have not caused any fatalities; these are, without exception, connected to the assisted disciplines, mainly NLT diving. The male and female record depths in these five disciplines are shown in Table 1. The classic competition discipline is CWT, and we will now follow Annelie Pompe as she completes an 87 msw dive using a monofin, along a rope to which she is attached by a safety line (Figure 1a).

SWIMMING TO 87 msw ON ONE BREATH

"*I have finished three warm-up dives to 14 m on empty lungs and it felt fine. I take calm, deep breaths, exhaling as slowly as possible, and my heart rate falls to a nice low pace. I feel completely relaxed, there are no disturbing thoughts in my mind and I am ready to dive. I take a few last deep breaths, exhale even more slowly and completely, then make a last full inspiration. I lift my upper body over the surface and top my lungs by packing ten packs, and go.*

The first swim stroke is powerful to overcome the buoyancy and I descend rapidly, feeling the pressure rise, and equalize my ears repeatedly using Frenzel – with the back of my tongue, air pressing against the tight nose clip. I try to relax all muscles, except the legs during each kick with the monofin, and in the pauses between kicks also my legs. I equalize based on how my ears feel, using reversed packing to fill my mouth before each Frenzel. The pauses between kicks become longer and longer, I pass what I think is 50 msw, swimming very slowly, and then start free-falling.

I fall head down through the water in my favorite part of the dive in a relaxed but streamlined position. My heart rate falls even further. Feeling the water passing at a more and more rapid rate, I close my eyes, knowing my safety line will keep me close to the rope. The pressure on my chest is increasing and I decide it is time to stop the reversed packing, so I make one last big 'mouth-fill' and then keep my epiglottis closed. This air will be used for the last equalization; blood shift will protect my lungs, but I have to protect my ears. It becomes darker, and I feel the first respiratory contraction, but don't let it disturb my relaxation as I fall toward the bottom plate at 87 m. It becomes even darker and I know I'm almost there. My arms and legs are tingling, everything feels smaller in my head and, being somewhat 'narked', I have to focus to stay alert.

Table 1
AIDA world records in the five depth disciplines as of October 2011 (depths in [ ] awaiting confirmation)

Discipline	Depth (msw)	
	Men	Women
Constant weight without fins	101	62
Constant weight with fins	124	[101]
Free immersion	121	[88]
Variable weight	142	126
No limit	214	160

I touch the bottom plate with my fingers, grab the rope and let my body fall down, and look around. I wish I could stay here longer, but know how far it is back to the surface. I make a single pull upwards on the line and make a first powerful stroke with my legs, now holding my arms close to my chest in order not to stretch the lungs too much. Up, up… I close my eyes again and tell myself my legs are strong and it is not so far left. I feel lactate building up in my legs, but can see it is getting lighter. I feel strong contractions but have to think positive and stay relaxed. I start swimming more slowly as my buoyancy increases, and there is my safety diver at 20 m. When floating the last few metres, I go through in my mind what to do when surfacing. A metre under the surface I exhale half of my lung volume, and as soon as I surface I take a breath. I make one – two – three fast breaths with some resistance on exhale (hook breathing), then the surface protocol: Mask off, OK sign and say "I am OK". I'm more than OK – I'm happy."

Characteristics of the deep-diving disciplines

When studying the 87 msw CWT dive described above, it becomes evident that there are great differences between diving to depth and the other disciplines. The three major differences are:

- the inability to interrupt the dive when at depth;
- the periodic work in depth disciplines;
- the pressure effects.

APNEA DURATION, ASCENT HYPOXIA AND SAFETY

Unlike in the pool disciplines, which are performed at or near the surface, in deep diving the diver cannot resume breathing until reaching the surface again, and must thus estimate the maximal effort at 'mid time' of the performance. In all disciplines, however, the attempted depth has to be pre-announced, and the diver will either reach this depth or turn early and incur penalty points. As with the pool disciplines, divers have to correctly perform the 'surface protocol' to show they are in full control at the end of the dive.[2] Should there be signs of hypoxia the diver is disqualified. Thus, divers cannot propose a dive that is more than they are confident of achieving without risking disqualification. As with the pool disciplines, there are safety divers, who closely follow the competitor during the last 20–30 m of the ascent, depending on depth, where there is a risk of ascent syncope.

Ascent syncope, often somewhat incorrectly termed 'shallow-water blackout',* is a potential risk associated with deep diving and a direct effect of the change in ambient and thereby gas partial pressures. When the diver descends, the air in the lungs is compressed in direct proportion to the ambient (hydrostatic) pressure (Boyle's law) and, as the partial pressure of oxygen (PO_2) in the lungs rises with the total gas pressure (Dalton's law), more O_2 can be transferred from the lungs to the blood (Henry's law). Thus, during the initial half of the dive, there is actually more O_2 available than during a breath hold at the surface. However, during the ascent, ambient (hydrostatic) pressure falls and, as part of the O_2 has now been used, PO_2 will be much lower than before. In fact, it may reach levels lower than the corresponding oxygen pressure in the blood, leading to the reverse transfer of O_2 from the blood back to the lungs.[8] This will rapidly cause a fall in brain oxygenation and potentially a loss of consciousness near the surface. This is because of the non-linearity of the O_2-haemoglobin dissociation curve, where at this point in the dive, a small decrease in PO_2 lead to a large fall in O_2 content of the blood. Thus, as this risk is directly associated with the fall in pressure near the surface, so is the risk for the diver. No such risk of hypoxic loss of consciousness is present at depth where oxygen pressure is higher than normal, as long as the diver is not delayed or prevented from surfacing. This is why it is sufficient to have safety divers present for the last part of the dive in order to prevent an accident should syncope occur during ascent.

The safety divers waiting at depth are also apneic divers, as the transportation to the surface must be very rapid, so that the diver can resume breathing. Divers breathing an air or other gas supply cannot rise to the surface at a sufficiently rapid rate because of a slower swimming speed, and risk decompression sickness (DCS) or arterial gas embolism (AGE) if they try. Using safety free-divers, an hypoxic syncope, if it occurs, happens within a controlled situation in which the athlete begins to breathe spontaneously within seconds of surfacing, or after a 'blow-tap-talk' procedure.[2] It seems that laryngospasm, i.e., automatic closure of the airways, prevents water from entering the lungs as long as the diver is submerged.[9] Although its function in drowning has been debated in the medical literature, it seems clear from all the events in competitive apnea, where unconscious divers brought to the surface start breathing spontaneously without signs of water aspiration, that laryngospasm is involved in protecting the airways in apnea divers (Schagatay E, personal observations and communications with divers).

Should the competitor have problems during earlier parts of the dive, other safety arrangements are present, the major one being a counter-ballast system. The diver is connected with a safety line to the vertical rope, and the rope can be pulled up with the diver attached should the diver not return to the surface at the announced time. Before diving, the diver states the expected dive duration, and the time keeper will feel the pull on the rope when the diver turns and heads for the surface. In major competitions, the diver is often monitored via sonar, and technical divers are standing by on the surface

Editor's footnote: The term '*shallow-water blackout'* is understood to have been coined during World War II by the Royal Navy to describe loss of consciousness (LoC) underwater in attack frogmen breathing oxygen from a closed circuit breathing apparatus. LoC in apnea divers should be termed either '*hypoxic syncope'* or '*ascent syncope'*.

ready to dive should there be any other problems. This has, however, never been necessary in AIDA competitions. Risk of hypoxic syncope is also evident directly after surfacing, due to the circulation time between lungs and brain, and the diver is, therefore, closely observed for 30 s. Divers often use 'hook breathing' to '*re-establish lung function*', likely counteracting hypoxia and pulmonary oedema.[2]

WORK ECONOMY AND THE FOUR PHASES OF A DEEP DIVE

Energy-efficient locomotion is essential for deep dives. As in the dive described above, muscles of the legs and lower abdomen are initially used for propulsion, but the diver will free-fall instead of swimming once negative buoyancy exceeds drag. The level of exertion involved is, therefore, not the same as in the pool disciplines of 'dynamic apnea', although the same muscles are used in these distance disciplines. In dynamic apnea, the work is quite constant, except during the turns against the pool wall. Also, in the most demanding depth discipline from an energetic standpoint, CNF, where the entire body works for propulsion using breaststroke, the total work required is less than in 'dynamic apnea without fins', because, after an initial phase of swimming, the diver reaches a phase of passive, free-falling descent. Also in FIM, involving only upper-body work, the diver free-falls instead of pulling the rope during part of the distance. Thus, despite the differences between the depth disciplines concerning levels of exertion and the muscles used for propulsion, all share a period of free-fall to minimise energy expenditure.

Therefore, diving to depth can be defined in four phases:

- an initial phase of *positive descent* when the diver works to overcome positive buoyancy;
- a second *free-fall* phase when the diver is sufficiently negatively buoyant;
- after the turn, a third phase of *negative ascent* with work to overcome negative buoyancy;
- a final *positive ascent* phase above the point of neutral buoyancy, when the diver may passively rise to the surface.

The same phases have been identified in diving seals and whales, whereby the deep-diving Weddell seal was found to reduce diving energy costs by up to 60%.[10]

The transition points between these phases vary between divers both in duration and depth depending on the individual diver's tissue density (fat/lean body mass ratio), lung volume, dive suit and weighting used. While body tissues will displace the same volume of water and have the same effect on buoyancy at depth, a high ratio of compressible air spaces in the body and dive suit to tissues will lead to a more rapid switch to negative buoyancy during descent (a lean subject with large lungs will thus fall faster compared to someone with low tissue density and small lungs. The first diver can stop swimming and rapidly gain speed in free-fall, while for the latter it may still pay to swim but with a lower stroke frequency. This may be yet another reason large lung volume is beneficial for human diving performance in addition to enhanced oxygen and carbon dioxide storage and increasing surface lung capacity to residual volume (SV/RV) ratio allowing greater compression (see below).[1,2,11]

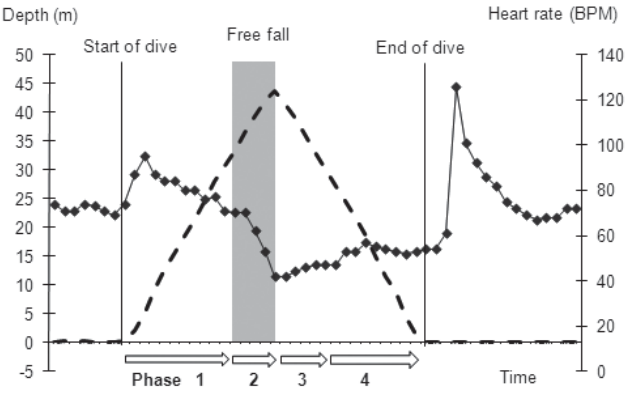
Anthropometric differences may be compensated for by the choice of suit and weighting, as some divers prefer to start to free-fall at an earlier point at the cost of having to work more on the way up, others preferring a deeper point of neutral buoyancy requiring greater initial effort. The passive part of the positive ascent phase may vary greatly in length, which may also reflect individual apneic ability and basal metabolic rate; some divers choose to continue swimming or pulling until surfacing. These transitions between working and resting phases during progressive hypoxia may in turn have effects on cardiovascular regulation.

THE DIVING RESPONSE AT DEPTH

The diving response is a priority system of circulatory responses to apnea that redistributes blood within the body, with a focus on maintaining the oxygen delivery in support of the most vital functions of heart, brain and working muscle.[9,12] The response involves vasoconstriction in organs tolerant of hypoxia and bradycardia, which conserves oxygen both during resting and working dives.[12–16] Its functions have been discussed in detail in the previous reviews.[1,2]

In the depth disciplines, the self-propelled diver must supply working muscle with sufficient energy from some source for the two working phases of the dive. The diver will initially be exposed to an increasing PO_2 caused by the air compression during descent. Thus, in phase 1 of the dive there will be sufficient oxygen and circulation will likely be kept in the working muscles as well as in heart and brain.[12] In phase 2, when the diver stops swimming, muscle circulation no longer needs to be prioritised and during free-fall, the diving

Figure 2
Heart rate (continuous line) during a training CWT dive by Annelie Pompe to 44 msw lasting 2 min; when free-falling starts, HR falls further



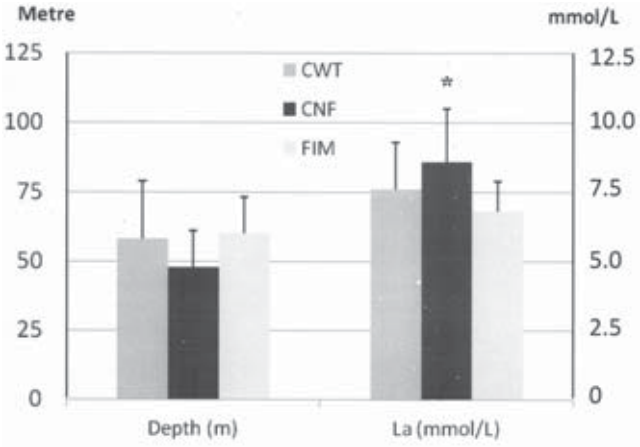
response can develop fully and conserve oxygen for heart and brain. In many divers, this transition between phases can be seen as an additional fall in heart rate (Figure 2). In itself, lung compression at depth may also enhance the diving response as will the colder water at depth, and by the end of the dive the developing asphyxia may contribute.[17–19] These factors all seem to act together to keep O_2 conservation at its maximum during deep diving.

During ascent in phase 3, despite the heavy work needed to overcome the negative buoyancy directly after the turn, heart rate remains low (Figure 2). Despite the urgent need of muscles for O_2, it appears blood flow is not shared between brain and working muscles once it has been redirected. Seals are known to rely on their unusually high levels of muscle myoglobin when this occurs,[20,21] but it is unknown if this factor is important in man. When there is conflict of interest, the brain always takes priority, and oxygen has to be conserved to sustain consciousness during the last part of the dive. Thus, the working muscles may have to rely completely on stored oxygen in the myoglobin and on anaerobic energy pathways, evident as the muscle soreness noted by Annelie in her description of her 87 msw dive, and classically interpreted as lactate accumulation, although its causes are complex and not fully understood.[20–23] In phase 4, the diver will often passively float to the surface and vasoconstriction can prevail.

In all animals relying on anaerobic glycolysis for energy production, the end products lactate and protons may cause metabolic acidosis.[24] It was observed in seals that, during a dive, lactate is sequestered and then released into the general circulation after breathing has been resumed, with a massive surge in the circulating blood on surfacing.[25] In exercise during breathing, where temporary and local limitations in blood flow cause lactate production, other regions may oxidize lactate, with only the net between production and use being detectable in the circulating blood.[26,27] However, the lactate produced during diving probably cannot be metabolised during the apnea, in part because of the massive peripheral vasoconstriction limiting transportation between tissues and in part to the over-all hypoxia; thus there may be no areas with sufficient oxygenation to oxidize lactate.

Therefore, high peaks of lactate can be seen directly after apneic dives, despite a fairly limited level of exertion.[2] To determine these levels and any differences between the depth disciplines, we measured capillary lactate 2–4 min after CWT, CNF and FIM dives during an international depth competition.[28] It seems that CNF, where the whole body is at work, represents the greatest hypoxic stress (Figure 3). However, compared to levels of around 10 mmol L^{-1} found in elite divers after dynamic disciplines,[2] the values after deep dives were lower. Even during static apnea at rest, there will be lactate accumulation, with levels of 5 mmol L^{-1} measured.[2] Reference values before dives were in the range 1–2.5 mmol L^{-1}, while normal resting levels are around 1 mmol L^{-1}. In

Figure 3
Mean (SD) depth and blood lactate concentration after CWT (n = 13), CNF (n = 10) and FIM (n = 10) competition dives
*** indicates $P < 0.05$ between CNF and FIM**



diving mammals, high baseline haemoglobin (Hb) as well as splenic contraction during diving will help buffer the blood and so will the human elevation of circulating Hb via splenic contraction.[29–31] In turtles, lactate can be stored in the shell and skeleton via the formation of calcium lactate, explaining how these air-breathing animals can survive anoxic periods of up to months.[32] Even in humans, asphyxia in critically ill patients has been found to be correlated with hypocalcaemia, suggesting a possible role of this mechanism in extreme conditions.[33]

REACHING MAXIMAL DEPTH – THE EFFECTS OF PRESSURE

While apneic duration and the ability to work efficiently with limited oxygen supplies can be limiting, in deep diving the ultimate challenges may be associated with the effects of increased hydrostatic pressure. The pressure will have direct effects on the air-filled spaces of the body; the increased hydrostatic pressure will cause these to be compressed with a risk of rupture if no equalization takes place, and there will be several effects of pressure changes on gas exchange between air and tissues, one being the risk of hypoxic syncope described above.

Training, preparation and equalization to avoid barotrauma

During increases in ambient pressure, the internal air spaces of the human body will shrink and eventually the negative pressure created may cause damage to surrounding tissues. During ascent, any air trapped in closed spaces will increase in volume and may also cause damage, although the latter case is more typical of compressed gas diving. Deep apnea dives must be preceded by training, and efficient equalization techniques must be employed to avoid barotrauma.

Figure 4
Lotta Ericson, professional free-diving instructor, performing 'reversed packing' and stretching, one of the many training exercises used for deep dive

The depth which can be reached without risk of barotrauma is set by the relation between the inspired lung volume at the surface (SV), and the diver's residual volume (RV). Classically, this surface volume was assumed to be the TLC reached by maximal inspiration.[34] However in elite divers, this is not the case. Divers use 'lung packing' to fill the lungs beyond total lung capacity (TLC) by using the oral cavity as a pump, which means SV can exceed TLC by several litres.[35–37] In addition, by employing long-term training methods involving lung packing and chest stretching, divers state they have increased their TLC by 2 L or more over time. Such training could partially explain why mean vital capacity was 7.3 L in divers, 1.8 L greater than that of a matched control group.[11]

A method called 'reversed packing' in which, after full expiration, the diver will suck air from the lungs can be employed to reduce the RV below normal levels by stretching the diaphragm and chest inwards (Figure 4).[38] Combined with other chest-stretching methods, these training regimes may enable divers to extend their 'lung-safe' depth considerably. Long-term lung training may include dives to moderate depths with empty lungs in order to mimic greater depths. For instance, pool dives to 5 m depth at RV are estimated to mimic dives with full lungs to approximately 90 msw.[39] However, this training may in some cases lead to capillary rupture and pulmonary oedema.[39]

As an example of the effects of such training, we can use a model diver with an original TLC of 4 L and a RV of 1 L. Using Boyle's law, we can calculate that this diver, after a normal TLC inspiration, will reach RV at a pressure of 405 kPa (30 msw). If instead the diver has a TLC of 5 L and an ability to pack an extra litre of air, the 'lung-safe' depth would be 50 msw (608 kPa). However, if the diver also manages to reduce RV by training to 0.5 L, this depth limit will now be 110 msw (1.21 MPa).

'Blood shift'

Another potential lung-protecting mechanism is the central pooling of blood in the thorax, called 'blood shift' which takes place during immersion, and increases progressively as the diver descends.[34,40,41] This redistribution of blood from the periphery to the pulmonary vessels compensates for the diminishing air volume and counteracts the risk of vessel rupture until vessel volume is maximally extended. Training with negative lung volumes may, in fact, increase the possible volume expansion of these vessels as suggested by the notion by divers that haemoptysis occurred only when they had not trained sufficiently (various divers, personal communications, 2010). Thus, specific long-term training methods, combined with warm-up dives, lung packing before descent, and the effects of blood shift may help explain why certain individuals can reach beyond 100 msw, and in extreme cases beyond 200 msw depth, without any detectable damage to the lungs or airways.

Preparation

Just as Annelie before her 87 msw dive, many, but not all, deep divers will use warm-up dives simulating greater depths by diving on either functional or residual lung volume. This may stretch the lung tissue, facilitate blood shift, and protect the lungs from sudden extreme negative pressure at maximal depth. As before performing the pool disciplines, breathe-up techniques may be used before deep dives to lower carbon dioxide storage in fast and slow tissues, and for the diver to reach complete relaxation.[1,2]

Upper airway equalization techniques

To many inexperienced divers, just being able to free-dive to 20 msw seems to present a series of challenges, as the pressure in the middle ear and sinuses generally has to be actively equalized. The most commonly used method is a Valsalva maneouvre, whereby attempted exhalation against a closed nose and mouth elevates airway pressure and forces air via the Eustachian tube into the middle ears. While this method can be used down to the diver's RV depth, after which a negative lung pressure makes it impossible, most trained divers instead use the Frenzel maneouvre. In Frenzel, the back of the tongue is used to elevate pressure in the oral cavity, pushing up against a closed nose. By using this technique, the lungs can be kept separated from the upper airways and pressure differences be allowed to develop.

When the diver descends beyond RV depth, air can still be drawn up into the oro-nasal cavity via reversed packing, the glottis closed, and the air used for equalization of ears and sinuses. However, the deep diver will eventually reach a

level beyond which it is extremely difficult to draw up more air from their lungs and at that point the diver takes the last '*mouth-fill*', after which the glottis must be tightly closed. All the muscles involved, most unfamiliar to the non-diver, can be actively controlled by the trained diver. This last '*mouth-fill'* must enable the diver to equalize all the way to maximal depth, or their eardrums may rupture. A last resort for the deepest divers is to use 'wet equalization', whereby the oro-nasal cavity (including the sinuses) and middle ear are flooded with water thus obviating the need to equalize the ears and sinuses and thereby avoiding aural and sinus barotrauma. This method appears to be used by some divers in extreme dives, but it is unclear if and by what methods the water is removed subsequently or whether there are other problems, such as vertigo when cold water enters the middle ear or infection related to the technique.

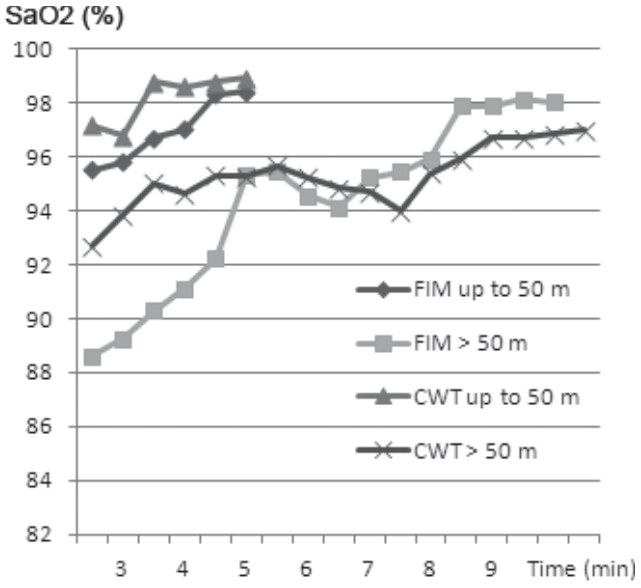
As the diving mask also has to be equalized to avoid eye and soft tissue squeeze, deep divers either use flexible masks with extremely low volumes or goggles filled with water, or avoid a mask entirely. Water-filled goggles offer some protection against abrasion and cold at the cost of good vision, but specialized goggles with lenses compensating for the loss of refractive power in water can also be used.

Despite long- and short-term preparation and excellent equalization skills, squeeze is the single most common problem associated with deep apnea diving. If not limited by other factors, each individual diver will eventually reach a depth where there is competition between ears and lungs for the last volume of air, beyond which damage may occur.

It has been shown that pulmonary oedema, detected as a delay of recovery of arterial oxygen saturation (S_aO_2) is quite common in deeper dives.[42] When we compared the recovery of S_aO_2 after deep CWT and FIM dives using pulse oximetry, we found evidence that not only depth but also the specific discipline may influence lung function. It was clear that, in dives beyond 50 msw, desaturation was greater and recovery slower after FIM dives, possibly because it involves upper body work (Figure 5).[43] This could explain why divers say they have to be careful not to move the chest excessively, as this may cause increased stress in the compressed lung. In the same study, involving a total of 56 dives, S_aO_2 recovery to 97% was found to be delayed beyond 10 min in nine cases (five CWT and four FIM) and cough with haemoptysis was found in three cases. Of these nine cases, eight had been to > 50 msw depth.[43]

This suggests that for divers to reach greater depths, their training and techniques for equalization need further development. However, the record depths reached in the no-limits category show that the lungs of some individuals may tolerate the immense effect of hydrostatic pressure of 2.3 MPa (214 msw), suggesting that the ultimate limiting factor might not be barotrauma. In addition to hypoxia and squeeze, there are other serious physiological challenges

Figure 5
CWT and FIM dives; recovery was the same for dives to ≤ 50 msw, but desaturation was more pronounced and recovery delayed after FIM dives to >50 msw ($P < 0.01$); mean depth for CWT was 53 msw and for FIM 52 msw (NS)



relating to depth, including nitrogen narcosis and DCS that have to be met.

Nitrogen narcosis

While efficient equalization techniques and training extend the tolerance to the effects of mechanical pressure and reduce the risk of barotrauma, the direct effects of hydrostatic pressure on gases cannot easily be avoided. The narcotic effect of nitrogen is fast enough to be evident in current apneic divers, whilst CNS oxygen toxicity may occur given enough partial pressure and time.[44] These effects may limit deep apneic air diving. Oxygen may also give rise to toxic damage to lung tissue, but this is extremely unlikely during the limited exposure time in apneic diving.

Nitrogen narcosis is evident from the anecdotes of many breath-hold divers. Describing her 'no limit' dive to 160 msw, Tanya Streeter reported an inability to perform well-rehearsed maneouvres at depth.[45] Other diving-related problems, including DCS, AGE, alternobaric vertigo, hypoxia and cold exposure could give rise to similar symptoms, but typical of the symptoms associated with narcosis is that they appear regularly and often at a certain depth, and disappear before or with dive termination, as seen by the following accounts (various divers, personal communications, 2009, 2010).

Diver account 1: One diver with a personal best beyond 90 msw, said that all dives beyond 75 msw (about 10 dives) involved some component of narcosis. This could be a lack

of focus as well as a sensation of numbness in the mouth and tingling in the fingers and toes. In some of the dives when he was inexperienced, this resulted in confusion and panic. The confusion was often related to hallucinations: an example was on the ascent from an 85 msw dive, when he believed that he passed the same reef over and over again, and felt he was not moving and was sure he was not going to reach the surface. Suddenly he surfaced without understanding how he had got there. He was now beginning to control these sensations at depth by focusing on something specific prior to the first signs of narcosis beginning.

Diver account 2: A diver with numerous dives beyond 100 msw and best performance beyond 120 msw, stated that narcosis is evident in all her dives beyond 50 msw. The way for her to proceed had been to learn to not give in to it but to cope with it, and focus on the tasks at hand. She has now found ways to perform well despite these problems.

Diver account 3: On his first FIM dive to 82 msw, the diver felt well at 75 msw but when he went on to 82 msw, he suddenly could not focus his mind and experienced blurred vision, making it difficult to see the rope. He found it impossible to think straight. He headed towards the surface with small pulls on the rope so as not to lose it. When he surfaced his mind was clear again. The next day he repeated a dive to the same depth without any problems.

While individual susceptibility and experiences seem to vary, narcosis is a common problem with deeper breath-hold dives and it seems important for divers to train in order to recognise the symptoms and sustain performance despite these effects. In a survey concerning lifetime experience of narcosis-like symptoms among 24 divers during a competition, we found that 12 had experienced symptoms that could be associated with narcosis, including dizziness and confusion (each seven cases), and these were all evident at depths greater than 40 msw (Schagatay et al, unpublished observations).

Decompression sickness

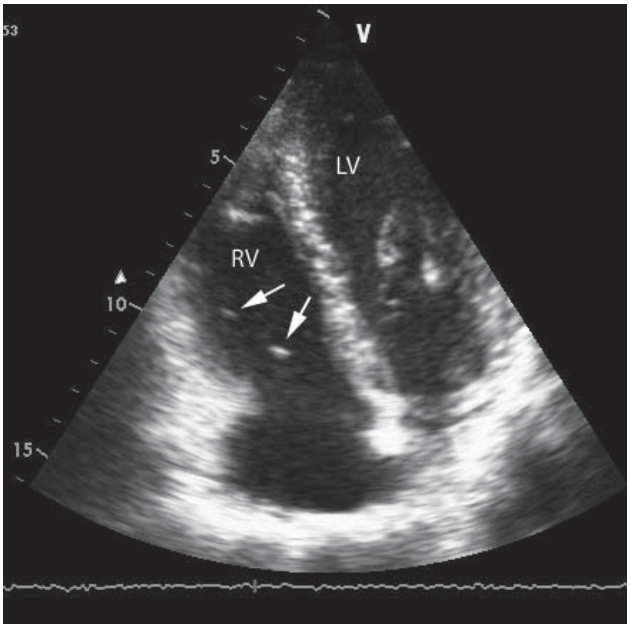
DCS, caused when nitrogen accumulates at depth then forms bubbles in supersaturated tissues during and after ascent, is one of the major limitations of diving breathing compressed gases, but it has long been debated whether breath-hold divers could develop DCS. Early reports of symptoms such as vertigo, nausea, paralysis and unconsciousness were suggestive of neurological DCS after repeated apneic diving in Tuamotu pearl divers.[46] That this could be the case was confirmed when Paulev showed on himself that repeated apnea dives to 20 m depth in a tank resulted in DCS symptoms, which were reversed by recompression in a hyperbaric chamber.[47] It is now generally accepted that repetitive dives even to moderate depths with short intervals may, over time, cause an excessive nitrogen load leading to DCS.[48–50] However, it is usually considered unlikely that competitive divers would be submersed for sufficient time and to sufficient depth to develop DCS, as single or few dives to depth are done in a day. Nevertheless, apnea divers are reaching greater depths, and thereby also increased durations, and DCS-like symptoms are reported after both training and competition, as seen from the following accounts (various, personal communications, 2009).

Diver account 1: An experienced diver was doing six to seven serial dives of 3–3.5 min duration with surface intervals of 3–4 min to 40–45 msw depth, when he suddenly became totally paralysed and unable to speak. His friends took him ashore, and he was taken by ambulance to a specialist hospital with a recompression chamber, but when they heard he had been free-diving, he was transferred to another hospital rather than being recompressed. After about 2 h, the situation spontaneously improved and, after some time, he was able to go home. There were no long-term effects.

Diver account 2: An experienced diver was doing a 'no limit' dive to 90 msw. When she turned on the air for the liftbag, it did not fill up. She tried twice more unsuccessfully and finally the bag filled and the sled started to move, but she had spent much time at depth. Five minutes after surfacing, she developed amnesia which lasted for about 30 min. During the amnesia, she had been "*talking funny*" according to friends who put her on O_2. For one month after this event, she was very weak and thinking and speaking slowly, but her symptoms improved and she could dive again.

We received many similar reports of possible DCS events among elite apnea divers in a 2009 survey (Schagatay E, Lodin-Sundström, unpublished observations). To our

Figure 6
Cardiac ultrasonic image from a diver with occasional bubbles (arrowed) after a competition dive to 70 msw



knowledge, no studies of bubble formation after competitive dives existed, so we also screened for bubbles in divers at two international diving competitions including all three depth disciplines. Mean depths for screened dives were respectively 58, 44 and 56 msw for CWT, CNF and FIM. Divers reported to the laboratory as soon as possible after dive termination, and screening of the right ventricle was done within 10–20 min after dives, using ultrasonic imaging for a single observation of 5–10 min. In about 100 dives, only two low (grade I on the Kisman-Masurel scale[51]) bubble scores were found, one after a CWT dive to 70 msw, the other after a FIM dive to 68 msw (Figure 6).[52] Sixteen of the dives were of depths of 70–90 msw. While bubble formation after deep apnea dives has hereby been confirmed, screening of a greater number of deeper dives over a longer post-dive period is needed to determine the risk for DCS.[53]

These reports and observations illustrate the need for increased awareness among apneic divers of how to prevent nitrogen accumulation and bubble formation, as well as increased awareness of these risks among physicians, so that appropriate treatment is made available. With increasing depths being reached, it becomes relevant to develop advice on free-diving profiles and surface intervals. The ratio between surface interval and diving time determines the nitrogen accumulation and when the ratio is 1 the exposure is roughly equivalent to 50% of the depth for a continuous dive.[54] Some divers, like the Ama and Bajau are capable of a 50/50 ratio over prolonged periods,[55] while competitive apnea divers, despite making fewer dives, reach depths great enough to cause nitrogen accumulation. Whilst the single breath per dive limits nitrogen uptake, the diving response could enhance uptake, by maintaining or increasing circulation to fast tissues such as the brain.

If warm-up dives are kept few and shallow, and spaced by sufficient time and possibly done with empty lungs, their contribution to nitrogen accumulation would be limited. An additional risk factor is that ascent rates are much more rapid in breath-hold diving than those recommended in standard decompression tables. Slowing ascents and including safety stops may help to reduce risk. Normal behaviour patterns may protect diving mammals from decompression injury, but even marine mammals may suffer from DCS in rare events, such as disturbances to their diving pattern from human underwater activity.[56]

The most advanced human divers can make slow ascents if allowed by apneic duration. In his record 'no limit' dive to 214 msw lasting 4 min 24 s, Herbert Nitsch reached target depth within 1 min 45 s, immediately turned and made a fast ascent to 60 msw within 54 s, after which he slowly ascended in 50 s to 10 msw, where he made a 30 s safety stop before slowly surfacing. After an accepted surface protocol, he returned to 10 msw on O_2; a standard method now used after deep competitive dives is for the diver to breathe O_2 at 6 msw depth for a period post-dive.

In deep-diving mammals, some species of which may reach depths of over 1,000 msw, pulmonary shunts and lung collapse may prevent nitrogen uptake and bubble formation on ascent.[50,57,58] For extreme depths in human apnea diving, similar effects may be present, but it is currently unknown when they occur and to what extent the mechanisms could be protective.[50,59] To prevent alveolar gas exchange and thereby be protective against nitrogen accumulation in humans, lung collapse would have to occur at a shallower depth than in most marine species, but modelling in divers after lung packing suggests this occurs at greater depths.[59] Diving with submaximal lung volume would allow collapse at a shallower depth, yet with the great human dependence on lung O_2 stores for apnea, it appears unlikely that this would be beneficial, at least in unassisted diving with great demands on energy for propulsion.

Arterial gas embolism

Another problem related to ascent from depth is AGE. AGE occurs as a direct effect of expansion of trapped air during ascent leading to tissue rupture and, when this occurs in lungs, bubbles may pass into the bloodstream to the left side of the heart.[60] This may lead to bubbles being transported to the brain, where they can block vessels leading to stroke-like symptoms. The development of AGE is increased with rapid ascent, as gas trapping is more likely to occur, especially in regions of the lung injured from pre-dive lung packing or squeeze during the descent in a deep dive. Despite different causes, the symptoms are similar and the treatment of AGE is the same as those for DCS, the most important actions being to give first-aid O_2 and to transport the diver to a recompression chamber.

Psychological requirements

Stress management, 'guts' and self-preservation

Stress management in order to achieve maximal relaxation and minimal metabolic rate is important in all disciplines of competitive apnea, and the mental capacity (stamina) to tolerate the extreme discomfort of an increasing urge to breathe and progressively enforced involuntary breathing movements ('contractions') for performance in static and dynamic apnea should not be neglected. However, in addition to these factors, a new psychological aspect will become crucial in deep diving; the way down is usually easy, but as you cannot start breathing before surfacing, it is an all-or-nothing event, and risk management is essential.

Whilst in deep diving, the *negative ascent* after turning at depth is the most physically demanding part of the dive, the *free-fall* may, in this respect, be the most psychologically demanding. The diver must decide before the dive what depth to aim for, but determine during the easiest course of the dive how well the *negative ascent* will be managed; and decide if an early turn should be performed or not. This is a balance between risk taking and safety awareness. To

Table 2
Factors rated by divers as the main limiting factor per discipline of apnea: CWT – constant weight with fins; DYN – dynamic apnea distance swimming, and STA – static apnea

	Pressure effects	O_2/CO_2 storage	Relaxation	Training capacity	Equipment	Other	Total no. answers
CWT	8	5	-	1	-	1 (science)	15
DYN	-	10	-	2	2	1 (diet)	15
STA	-	9	6	-	-	1 (diet)	16

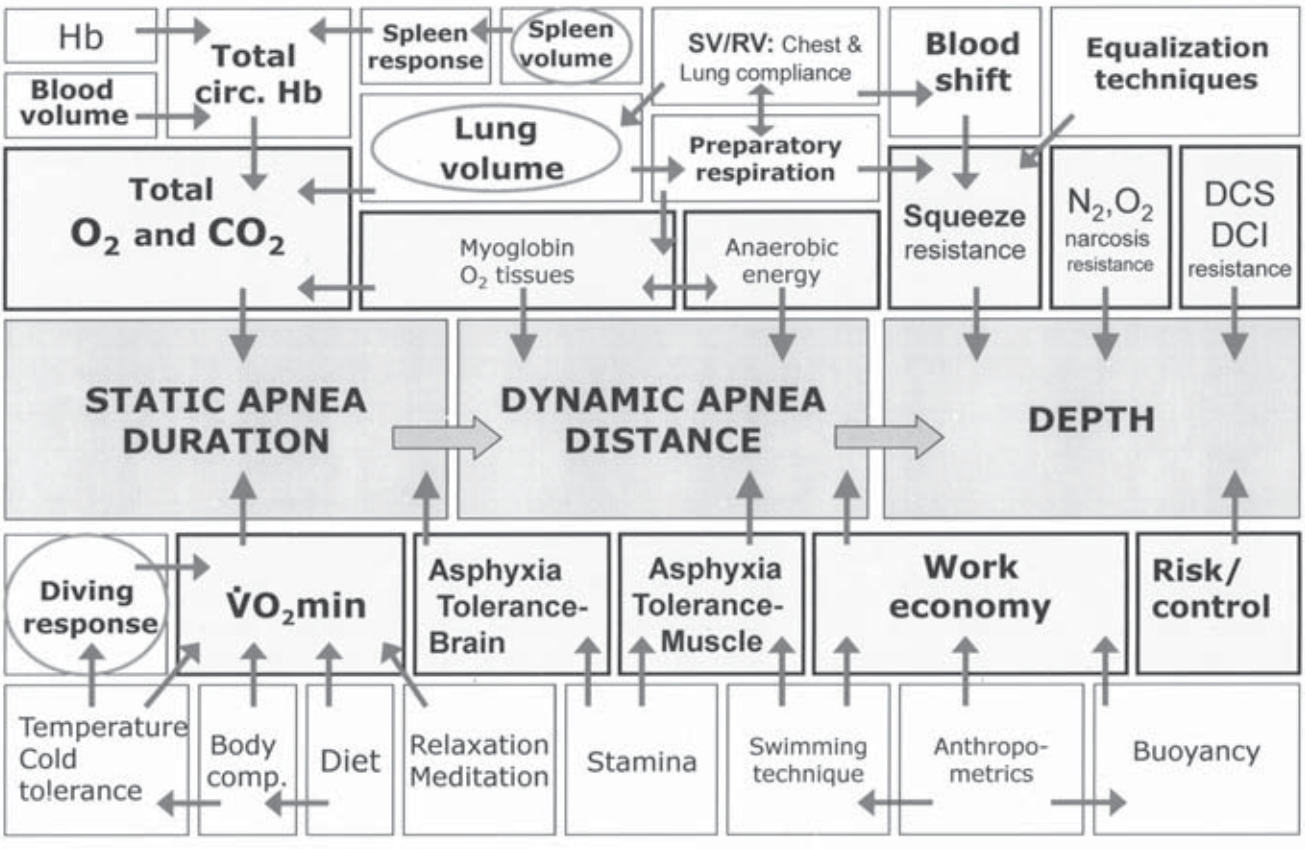
be deprived of the possibility to breathe during hard work, when the urge becomes overwhelming, is truly frightening. To make decisions about how to manage the last part of the dive and reach the surface without blacking out, before being at the midpoint is an unusual challenge. It is essential to keep this focus even under the influence of narcosis.

The commonly held view that these attempts are made by suicidal dare-devils is incorrect; breath-hold divers are generally highly concerned with safety and do not want to take unnecessary risks. Yet the balance between a safe and easy dive and an extremely uncomfortable event leading to a record has a component of 'pure guts' and determination to it. This is paralleled in very few sports except, perhaps, extreme skiing and climbing.

Where are the limits?

Predicting the limits in deep apneic diving is not easy, as a new set of factors related to depth are added to all the ones determining resting apnea duration and shallow diving working capacity. Essential in the development of records is to balance faster speed with reduction in energy cost; just faster speed will not help, as the energy requirements may increase and duration shorten. While apneic duration appears not to have been the major limitation thus far, greater depths may call for exceptionally long apneas and slower ascents with safety stops aiming at avoiding DCS. Thus, only descent speed may be increased; but that puts great pressure on the diver's equalization techniques and the body's resistance to barotrauma. Increases in body oxygen storage as well as

Figure 7
Factors influencing performance in the various apnea disciplines; factors affecting static apnea are transfered right to dynamic apnea, which then all combine with new factors added for depth (for explanations see text)



enhanced tolerance to asphyxia will allow deeper dives even at the same speed and metabolic rate.[1,2]

It is clear that individual athletes may be limited by different factors, but in order to get further, all eventually have to deal with the enhanced effects of hydrostatic pressure. Physiological features involved may include lung volume, where both TLC and the SV/RV ratio are important, blood shift and resistance to squeeze, haemoglobin concentration and total haemoglobin, splenic volume and maximal contraction, muscle myoglobin concentration, the diving response, work economy, cold tolerance, asphyxia tolerance, anaerobic capacity and minimum metabolic rate, special techniques for respiration, inspiration and equalization, and also psychological factors involving stress control, risk/caution and to some extent 'pure guts' (Figure 7). As in all sports, maximal performance will be determined by the maximum capacity of individuals, achieved by genetic predisposition, long-term training, and use of proper techniques for preparation and performance. The ultimate limits will be reached only if participation in the sport of apnea diving approaches that of more widespread sports.

Input from elite divers

Physiologists are historically known to be utterly wrong in predictions of maximal performance, partly because these are often based on laboratory-derived data from apnea experiments collected from non-divers or moderately trained divers. Even after several studies of the physiology of elite divers, predicting future performance is difficult, as new divers enter the field and training methods improve. My prediction in the first review of a likely maximum static apnea time of slightly over 11 minutes, based on current data from active divers, was redundant when a new record of 11 min 35 s was set the same year![1]

We asked elite apneic divers what they thought would set the limits of free-diving. Seventeen divers (10 males and seven females) participating in the 2008 apnea world championship were asked to predict the ultimate performance in the CWT, DYN and STA disciplines and to list the main limiting factor per discipline, without suggestions given as to what these factors might be. Their mean (SD) personal bests were 63 (15) msw in CWT, 136 (30) msw in DYN and for STA 6 min 4 s (13 s). Predicted ultimate limits in the three disciplines were for CWT 156 (13) msw depth, for DYN 323 (31) metres' distance and for STA a time of 14 min 54 s (3 min 17 s). Different limiting factors were stated for the different disciplines of competitive apnea (Table 2). In CWT, the most frequent 'number one limitation' stated was 'pressure effects' followed by five first ratings for gas storage (Schagatay E and Lodin-Sundström A, unpublished observations, 2008).

Based on physiological measurements and observations in elite divers, and the fact that the current record development does not seem to be levelling off, I would consider these predictions to be realistic, with the pressure effects related to nitrogen saturation most likely setting the ultimate limits for the depth disciplines.

Conclusions

Currently, deep diving problems are dominated by hypoxic syncope on surfacing and barotraumas which, even if not life-threatening, may stop many individual divers from reaching further. However, the 214 msw performance in the 'no limit' discipline suggests that certain individuals may tolerate nearly complete compression of the lungs and airways. As DCS and nitrogen narcosis cannot likely be avoided in man by diving on empty lungs allowing early lung collapse, because of human dependence on lung oxygen stores, I would hold it likely that the ultimate limits for deep diving will be set by these factors, when training techniques have been developed further to prevent barotraumas, hypoxic syncope and AGE. Further technical developments may also be needed, and safety arrangements must develop concomitantly with the increase in depth.

Acknowledgements

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Technical report

Suitability of the partially implantable active middle-ear amplifier Vibrant Soundbridge® to hyperbaric exposure

Christoph Klingmann, Angela Klingmann and Theodoros Skevas

Abstract

(Klingmann C, Klingmann A, Skevas T. Suitability of the partially implantable active middle-ear amplifier Vibrant Soundbridge® to hyperbaric exposure. *Diving Hyperb Med.* 2011 December;41(4):229-232.)

Introduction: Active middle-ear amplifiers represent a modern possibility to treat sensorineural, conductive and combined hearing loss. They can be in use in divers and patients who need hyperbaric oxygen therapy. Therefore, active middle-ear amplifiers have to be tested to determine whether or not they are prone to implosion or function loss in hyperbaric conditions.

Material and methods: We asked three of the companies registered by the German health authorities as manufacturers of active middle ear amplifiers to test their devices in hyperbaric conditions. Med-El agreed to support the study; Envoy stated that their devices were unable to withstand a pressure of 608 kPa; Otologics had no capacity to take part in this study. Twelve Vibrant Soundbridge® (Med-El) middle-ear amplifiers were tested in a water bath in a hyperbaric chamber. Four devices were pressurised to a maximum of 284 kPa, four devices to 405 kPa and four devices to 608 kPa, each for a maximum dive time of 78 minutes. The functions of the devices were tested in the laboratory by the manufacturer pre- and post-hyperbaric exposure.

Results: Visual inspections and laboratory function tests were normal in all 12 devices after hyperbaric exposure.

Discussion and conclusion: Hyperbaric exposure to more than one bar pressure difference can result in structural damage, implosion or loss of function of mechanical devices. The Vibrant Soundbridge® middle-ear amplifier tolerated a single hyperbaric exposure to pressures of up to 608 kPa for 78 minutes with no loss of performance.

Key words

Diving, implantable devices, hearing, equipment, pressure, barotrauma, performance

Introduction

Implantable, active middle-ear amplifiers represent an innovative option for the treatment of patients with sensorineural hearing loss, and, since the indication criteria were expanded, also for patients with mixed and pure conductive hearing loss.[1–5] Unlike conventional hearing aids that can be left behind when a patient goes into the water, active middle-ear amplifiers have parts fully or partially implanted into the patient's body.

The advantages of active middle-ear amplifiers are:

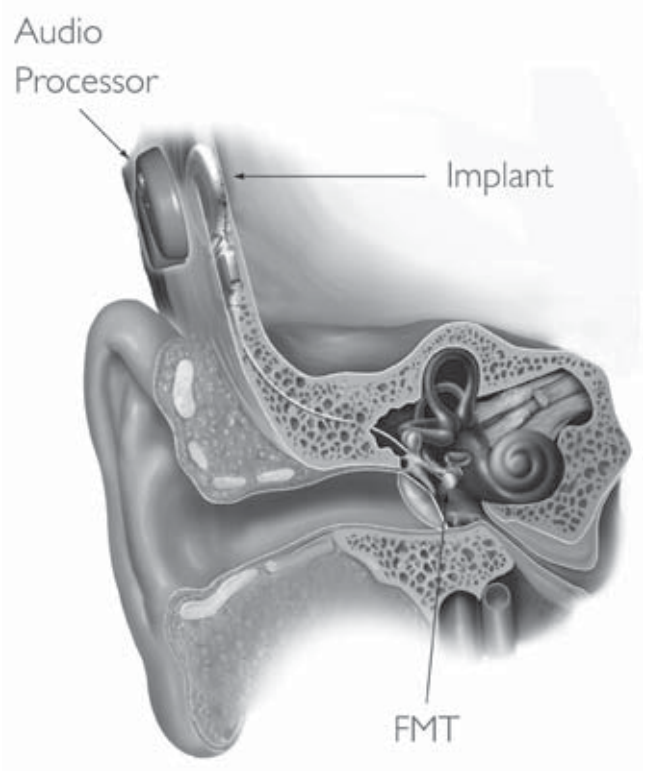
- better sound transmission into the inner ear through the direct connection to the ossicular chain or the round window membrane resulting in improved hearing, especially at high frequencies;
- increased sound transmission for patients with profound hearing loss;
- avoidance of recurrent external otitis which often occurs in patients with conventional hearing aids.

The basic principle of active middle-ear amplifiers is that they transfer the sound from the retrocochlearly implanted device through a lead that runs through the mastoid into the middle ear to a vibratory structure that activates the ossicular chain or the bone surrounding the inner ear (in the round window niche).

The products *Carina*® from Otologics, and *Esteem*® from Envoy are fully implantable devices with a microphone in the ear canal, whereas the *Vibrant Soundbridge*® (VSB) from Med-El is a partially implantable hearing system which needs an additional device on the patient's skin, kept in place by magnetic forces and which must not be worn in water. Therefore, it cannot be used underwater. The VSB uses a floating mass transducer (FMT) that is clipped to the incus or fixed in the round window niche to transmit the sound into the inner ear (Figure 1), whereas the *Carina* transmits the sound via a plunger on the incus body, while the *Esteem* transmits the sound through the stapes after the incus has been removed.

The implanted parts of the hearing devices are exposed to increased ambient pressure during diving. Since diving with compressed air involves an ambient pressure of up to 608 kPa (6 Ata) the implanted part of the active middle-ear device must be able to withstand this pressure without malfunctioning or endangering the diver through implosion-related trauma. Exposure to increased ambient pressures also occurs in situations other than diving. If it is necessary to administer hyperbaric oxygen therapy (HBOT) to a patient at some point in time after the implantation of an active middle-ear amplifier, there is a danger that the device will malfunction or that an implosion-related trauma in the hyperbaric chamber could occur.[6]

Figure 1
The Vibrant Soundbridge®, a partly implantable, active middle-ear amplifier. Only the implant and the floating mass transducer (FMT) were pressure tested as the audio processor is removed for diving or HBOT (image courtesy of Med-El)



The same problems exist for patients with cochlear implants (CI). There are patients who want to perform scuba diving after CI procedure, and the growing number of people with CIs increases the likelihood that such a patient will need HBOT at some point in time. For this reason, an American team exposed various cochlear implants to pressures up to 608 kPa.[7] The implantable parts of all the devices tested had no loss of function, leakages or implosion damage.[7]

The purpose of the current study was to expose all of the active middle-ear implants which were available on the German market in 2008 to a maximum pressure of 608 kPa.

Material and methods

The companies Envoy, Med-El and Otologics were approached to supply their devices for testing under hyperbaric conditions. Envoy advised that their *Esteem*® active middle-ear implant had already been tested up to a pressure of 608 kPa. This had led to loss of function in the implant, and for this reason they were not interested in supporting this study (Krey C, personal communication, 2006). Otologics stated that their *Carina*® implants had so far been tested up to a positive pressure of 203 kPa and to reduced pressures. This would indicate that diving up to 10 metres' sea water (msw) should be possible with this device. The package insert recommends that the patient discuss diving suitability with the surgeon. Otologics declined to have their devices tested up to an ambient pressure of 608 kPa (Teigland P, personal communication, 2007).

Med-El provided the implantable parts of 12 Vibrant Soundbridge® devices and financed the hyperbaric chamber exposure at the Heidelberg Hyperbaric Unit, Germany. The audio processor that is placed on the patient's skin was not tested as this would be removed before diving. Therefore, no function test in hyperbaric conditions was performed.

The devices were placed in a water bath and four were exposed to each of the pressure profiles:

- 284 kPa (18 msw), chosen as the maximum pressure used during HBOT;
- 404 kPa (30 msw), chosen as the pressure used for a COMEX 30 treatment table;
- 608 kPa (50 msw) chosen as the maximum pressure during air dives in the German military services.

The exposure time at the maximum depth was 60 minutes and the compression and decompression rates were both 50 kPa min^{-1}. These protocol were used to find a staged pressure tolerance of the devices in case the devices could not withstand the maximum test pressure. Pressure was measured with a diving computer, also placed in the water bath (Uwatec, Switzerland) and the hyperbaric chamber pressure measurement system (Haux Life Support, Karlsbad).

The implants were tested for normal function by the manufacturers before and after hyperbaric exposure using a Laser Doppler Vibrometer. Frequency response and signal quality were measured by means of harmonic distortion. The function tests were considered to be successful if the same criteria were fulfilled as those required for newly produced implants. Each device was also visually inspected for damage or distortion.

Results

All 12 Vibrant Soundbridge® devices were deemed fully functional prior to hyperbaric exposure. After completion of hyperbaric chamber exposure, all 12 implants exhibited no evidence of damage or distortion or loss of function.

Discussion

Implantable devices used while diving or in a hyperbaric chamber must be able to withstand increased ambient pressures. Trigano et al. examined cardiac pacemakers in vitro at pressures of 404 kPa and 606 kPa.[8] The pacemakers proved to be fully functional at both depths; however, in 15

out of the 29 tests there was damage to the housing after hyperbaric exposure at 606 kPa. Because of this, the authors recommend that patients with pacemakers dive only to a maximum of 304 kPa (20 msw).[8]

There are several theoretical risks under hyperbaric conditions following the implantation of an active middle-ear implant.

IMPLOSION

An implosion of the device in the mastoid could occur leading to pain, injury, cochleo-vestibular symptoms and even intracranial complications.

LOSS OF FUNCTION

So far, only the Med-El Vibrant Soundbridge® devices, with 12 devices tested at pressures up to 608 kPa have been shown to be completely functional after hyperbaric exposure.

INCREASED SUSCEPTIBILITY TO BAROTRAUMA

Drilling of the mastoid is necessary for the implantation of a middle-ear device, and the sound amplifying component must be coupled to the ossicular chain or placed in the round window niche. Mounting a plunger on the incus (company: Otologics) or a Floating Mass Transducer (FMT) on either the incus or at the round window niche (company: Med-El) increases the weight and inertia of the ossicular chain. However, it is unlikely that this increases the danger of barotrauma. If the ossicular chain is disrupted as with the *Envoy* devices, there is even an increased protection of the inner ear because pressure transfer from the auditory canal into the inner ear is excluded, resulting in a lowered risk for barotrauma to the inner ear. Unfortunately, these devices are not pressure-resistant according to the manufacturer.

All active middle-ear amplifiers are implanted via a 'canal-wall-up' mastoidectomy, which does not represent a contraindication for diving since the posterior outer-ear canal wall remains intact and the mastoid cavity and the labyrinth are separated from the ear canal and, therefore, protected from direct cold-water stimulation. 'Canal-wall-down' mastoidectomy (see below) is used when the posterior ear canal wall has to be removed and the mastoid cavity and the labyrinth are directly exposed to water when the patient submerges.

LOCAL SUPERSATURATION WITH LOCALISED DECOMPRESSION SICKNESS

During decompression and after a dive, nitrogen is released from the body tissues. Vann et al. determined that dives which complied with the permitted diving regulations led to gas bubble formation in breast implants and a consequent, minimal enlargement of the implant. Only diving which was not conducted in the manner of recreational diving (e.g., saturation diving) and subsequent direct altitude exposure at 10,000 metres led to significant changes in the volume of the implants.[9] Therefore, an increase in localised inert gas bubbles through supersaturation along the implanted electrode could occur. However, since active middle-ear implants and their components are contained in the middle ear and on the skull and not in the vulnerable sites of the inner ear, this danger would seem negligible.

PERIPHERAL VERTIGO FROM EXPOSURE OF THE LABYRINTH

Scar tissue can occur after a mastoidectomy, which can reduce ventilation in the mastoid. However, since the majority of patients who receive an implantable middle-ear amplifier have healthy middle ears, this seems very unlikely. Therefore, in patients with sensorineural hearing loss, when the posterior wall of the auditory canal is intact, an irritation of the labyrinth, which must be exposed while implanting the device, is unlikely and, therefore, can be disregarded. With mixed or pure conductive hearing loss, patients often have a history of 'canal-wall-down' mastoidectomy, which can represent a contraindication for diving because these patients have an increased risk for vertigo when cold water enters the mastoid. These patients need to have a cold-water provocation test (4^{o}Celsius) and, if vertigo occurs, they are not fit to dive.

Conclusions

Twelve Vibrant Soundbridge® middle-ear amplifier devices (Med-El) showed no changes in shape and no loss of function when subjected once to pressures up to 608 kPa. For this reason, the Austrian and German diving and hyperbaric medical societies recommend only this device for divers since alternatives are either not pressure-resistant or have only been tested to 203 kPa. The manufacturer should always be contacted to ensure no changes have been made to the device in the meantime. Further tests with other implants are necessary to decide whether these devices may be exposed to diving or HBOT.

Acknowledgements

The authors would like to thank Drs Anke Fabian and Johannes von Reumont from the Druckkammerzentrum Heidelberg for their support and provision of the hyperbaric chamber.

Conflict of interest: The study was supported with provision of active middle ear implants and reimbursement of expenses for the hyperbaric tests by Med-El.

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Critical appraisal

Does hyperbaric oxygen administration decrease side effects and improve quality of life after pelvic irradiation?

Bottom line

1. Hyperbaric oxygen improved symptoms and signs of radiation proctitis in women following pelvic irradiation for cervical cancer.
2. There was also an improvement in both proctoscopic and histological findings following HBOT.

Citations

1. Sidik S, Hardjodisastro D, Setiabudy R, Gondowiardjo S. Does hyperbaric oxygen administration decrease side effect and improve quality of life after pelvic radiation? *Acta Med Indones*. 2007;39:169-73.
2. Sidik S, Daldiyono H, Setiabudy R, Gondowiardjo S, Yoewona V. Oxygen hyperbaric therapy in patients with radiation proctitis. *The Indonesian Journal of Gastroenterology, Hepatology and Digestive Endoscopy*. 2007;8:1-4.

Three-part clinical question

For females with radiation proctitis after receiving radiotherapy for cervical cancer, does hyperbaric oxygen therapy result in improvement of quality of life and objective evidence of radiation proctitis?

Search terms

Hyperbaric oxygen therapy, radiation proctitis, quality of life

The study

Non-blinded, randomised controlled trial, intention to treat unclear.

The study patients

Adult females less than 55 years old with cervical cancer stages I–IIIB. All had received radiotherapy (25 x 2 Gy external beam with 2 x 85 Gy brachytherapy doses), and developed radiation proctitis (confirmed by proctosigmoidoscopy and biopsy) between 1 to 6 months post radiotherapy. Exclusion criteria were: pneumothorax, diabetes, malnutrition, other chronic disease, or depression. HBOT was delivered for a minimum of 18 treatments.

Outcomes reported in two separate papers:

Side effects and quality of life outcomes assessed using LENT SOMA and Karnofsky scores at baseline pre-intervention, then percentage change of scores were reported comparing post-intervention (HBOT or standard care) with baseline, and six months post-intervention with baseline.

Presence or absence of radiation proctitis on proctosigmoidoscopy and histopathology at 6 months after HBOT.

CONTROL GROUP (n = 33, 20 analysed) received standard care (not defined).

EXPERIMENTAL GROUP (n = 32, 26 analysed), received HBOT (not defined – see comments) for a minimum of 18 treatments.

The evidence

See Tables 1 and 2 (next page).

Comments

1 High drop-out rate due to long travel distances, intolerance of HBOT (three) and high loss to follow-up (seven controls) with 16 deaths (10 control, six HBOT) by 6 months.
2 75 patients eligible for study, 35 allocated to HBOT, and 40 to standard care. Three unable to comply with HBOT, seven lost from standard care (four due to travel distance, three dropped out).
3 Of the patients receiving HBOT (n = 32), six died of cervical cancer prior to 6-months follow up. In the control group (n = 33), 10 died, two moved away and one was lost to follow up.
4 No definition of 'standard therapy'. HBOT was probably between 203 and 304 kPa for 2 hours as discussed in the introduction.
5 Quality-of-life outcomes reported as percentage change from baseline, rather than raw scores.
6 Study reported in English, with evidence that this was not the first language of the authors.

Appraised by: D Smart, 20 August 2011
E-mail: *<dsmart@iinet.net.au>*

Source

<www.hboevidence.com>

Key words

Hyperbaric oxygen, cancer, radiotherapy, side effects, soft-tissue radionecrosis, outcome, critical appraisal

Table 1
Major outcomes at 6 months for radiation proctitis following at least 18 HBOT compared with standard care

Absence of radiation proctitis	Control group	Experimental group	Relative risk reduction (%)	Absolute risk reduction	*P* value	Number needed to treat
Reported:	9/20 = 0.45	20/26 = 0.77				
Intention to treat:	9/40 = 0.23	20/35 = 0.57	154	0.35	0.026	3
95% CI			61 to 247	0.14 to 0.56		2 to 7

Table 2
Major outcomes for quality of life following at least 18 HBOT compared to standard care (mean and SD)

Measure	Control group		HBOT group		Difference	95% CI	*P* value
	Mean	SD	Mean	SD			
LENT SOMA score at end of HBOT (% change)	44.1	28.2	0.7	30.2	43.4	-472.4 to 559.25	< 0.001
LENT SOMA score at 6 months (% change)	33.6	57.6	-19.7	69.4	53.3	-2448.4 to 2555.1	0.008
Karnofsky score at 6 months (% change)	15.3	14.7	2.5	16.1	12.8	-131.8 to 157.4	0.007

Book reviews

Decompression illness, a simple guide and practical advice on the recognition, management and prevention of DCI

John Lippmann

Paperback, 64 pages
Submariner Publications, 2011
ISBN: 9780975229064
Available from: <www.danasiapacific.org>
Price: DAN Members AUD8.50, others AUD10, plus p&p

John Lippmann is a well known author of books relating to dive injuries and incident management such as the *DAN emergency handbook*, *Deeper into diving* and *Advanced O_2 first aid*. He is also the founder, current Executive Director and Director of Training for DAN Asia Pacific.

The title of this book describes its contents well. By way of introduction, Lippmann reinforces the teaching that decompression illness (DCI) may present even when the diver may not technically 'deserve' the illness. He reminds us that staying within the 'safe' parameters of a decompression schedule, or within no-stop limits, may not keep us from getting a 'bend' and that when we are diving in remote locations we may be unpleasantly surprised at the lack of access to DCI-related care.

Following on from this thought-provoking reminder, the reader is given a brief refresher on the uptake and elimination of nitrogen in the body. The next chapter, the largest, defines DCI, its typical symptoms and signs and risk factors. These risk factors are divided into diver-related and post-dive-related. Some factors can be mitigated and Lippmann provides advice on these.

Patent foramen ovale (PFO) is a major risk that may require medical intervention and this is dealt with in Chapter 3. First aid for DCI is briefly discussed but oxygen provision is dealt with more fully in a later chapter. Photographs of the different styles of recompression chambers and typical treatment schedules serve to demystify what, for many, must be outside their previous experience (we hope!). Lippmann, drawing on his experience in diver rescue, sounds a warning regarding the quality, safety and accessibility of chambers in some locations. He dedicates the final chapter ("*The realities of diving accidents in remote places*") to these issues.

The use of a key points summary at the end of each chapter allows for quick reference, and case reports demonstrate real-world scenarios that reinforce the benefits of immediate recognition of and response to DCI events. The glossary is useful.

One of the stated objectives of this booklet is to answer frequently asked questions on DCI in a way that can be understood by most divers. On the question of risk factors, Lippmann alludes to the results of surveys, studies and

experiments, and the degree of certainty associated with current knowledge on whether or not they are indeed risk factors. He has chosen not to reference these studies. This keeps the text clear and uncluttered, though some may be left wanting to access these. However, for those interested, a short list of further reading is provided at the end.

This booklet is an excellent resource for all divers, as much for novices as for the more experienced diver, and will strengthen understanding and increase competence and confidence in the provision of care.

Rob Edward, New Zealand Fire Service, Wellington
Selina Davis, RN, Heart and Lung Unit, Wellington Hospital

Key words
Decompression illness, recreational diving, first aid, hyperbaric facilities, general interest, book reviews

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Wound care certification study guide

Jayesh Shah, Editor
Paul Sheffield, Caroline Fife, co-editors

Soft-cover, 320 pages, full colour
ISBN: 978-1-930536-61-6
Best Publishing Company, 2011
Palm Beach Gardens, FL 33410, USA
Available from: <www.bestpub.com>
Price: USD54.95

This guide offers much wisdom to the reader. It is easy to read, carry around and understand; it focuses on the key information considered important when studying wound care; and, if completing formal exams, provides essential examinable knowledge.

The sequence of chapters is logical, progressing in specifics as the reader's knowledge increases. The sample questions at the end of each chapter and room for note taking are common sense and consider the readers' needs. This multi-disciplinary guide provides essential information necessary for developing a comprehensive wound management programme for those involved in the management of chronic wounds. Seven subjects are covered: general knowledge, anatomy, diagnosis, physiology, therapeutics and finally psychosocial topics.

Starting the guide with a chapter on test-taking strategies is a sensible idea and assists the reader in focussing on what is required and how best to achieve this; after all, who doesn't suffer from exam nerves? Each chapter begins with a brief introduction and lists objectives expected to be achieved for the topic covered, with the subsequent information set out in a well-organised manner and key study points with not too much detailed information. The full tests and answers at the end of the guide, comprehension questions at the end of each chapter along with illustrations all add to this as a valuable aid to learning.

To complete the guide, a chapter on "*Dr Shah's wound management guidelines"* has included multiple guidelines on how to approach problems encountered with particular wounds, whilst always including a full health assessment. A practical guideline on nutritional assessment is also present. This appears to have good, simple-to-follow algorithms for many of the wound care issues faced today. Whilst not the complete answer on how to manage a wound, it gives direction and a plan to the approach to the patient and their wound.

As expected of any book on wound care, there are numerous colour diagrams and photographs, with only a few out-of-focus images used. As with previous book reviews on wound care, I question why authors and publishers persist in publishing poor quality images when it is possible, in this electronic era and with patient consent, to obtain good quality, near-professional images.

This reviewer considers this to be a well-organised guide to wound care and valuable, not only as a study guide, but as a tool for refreshing the trained practitioner's knowledge. Several of the nursing staff in my unit, all with post-graduate training in wound care, also found this guide well worth reading. With the added benefit of being able to self test and find areas where knowledge may be inadequate or lacking, this will prove an adaptable resource for those teaching wound care as well as those participating in this 'dark art'. This is an essential book for anyone teaching or involved in wound care or preparing for exams in this field and well worth a place, not only on the hyperbaric unit bookshelf, but in every wound care nurse's library.

Yvonne Denny
Charge Nurse Manager, Hyperbaric Medicine Unit, Christchurch Hospital, New Zealand

Key words
Nursing, wounds, book reviews

Continuing professional development

CME ACTIVITY 2011/4

Fluid balance in diving and diving-related injury

John A S Ross

Accreditation statement

Intended audience

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

Objectives

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

Faculty disclosure

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

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

Background reading

Practitioners are referred to the following references and reading; review articles are indicated. Many of the articles are applicable to more than just one section. A useful database to search is at: < http://rubicon-foundation.org/>

Effects of immersion and hypothermia

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Effects of intravascular bubbles and loss of capillary integrity

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Hypovolaemia and hypoperfusion

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31 Vann RD, Butler FK, Mitchell SJ, Moon RE. Decompression illness. *Lancet*. 2011;377(9760):153-64. REVIEW

Answers should be sent by e-mail to the nominated CPD co-ordinator. For EUBS members for this CPD issue this will be **Dr John Ross, E-mail:** <j.a.ross@abdn.ac.uk>.
For ANZCA DHM SIG members, this will be **Dr David Cooper, E-mail:** <david.cooper@dhhs.tas.gov.au>.
On submission of your answers, you will receive a set of correct answers with a brief explanation of why each response is correct or incorrect.

Successfully undertaking the activity will require a correct response rate of 80% or more. Each task will expire within 24 months of its publication to ensure that additional, more recent data has not superceded the activity.

Key words
MOPS (maintenance of professional standards), decompression sickness, cerebral arterial gas embolism, endothelium, circulation, coagulation, resuscitation

How to answer the questions
Please answer all responses (A to E/F) as True or False.

Question 1. During immersion in water while breathing air, loss of body fluid:

A. is preventable
B. is predominantly due to immersion diuresis at normal body temperature
C. can be greatly increased by a fall in body temperature
D. can lead to increased risk of decompression sickness
E. is immediately reversed on surfacing.

Question 2. Intravascular bubbles occurring during and after decompression illness:

A. can have no cardiovascular effects
B. may reduce endothelial function in the absence of decompression illness
C. occasionally cause loss of capillary integrity leading to acute hypovolaemia due to increased endothelial permeability
D. always lead to increased capillary permeability and loss of circulating plasma after diving
E. are thought potentially to cause life-threatening post-decompression cardiovascular shock.

Question 3. Clinically significant hypovolaemia after decompression:

A. does not need aggressive correction
B. may be disregarded by the victim
C. may lead to circulatory collapse
D. can be associated with post-traumatic coagulopathy
E. can be prevented by rapid recompression.

Question 4. Regarding the symptoms and signs of hypovolaemia:

A. the symptoms of hypovolaemia differ clearly from those of decompression illness
B. cutaneous decompression illness can be an indication of hypovolaemia
C. identification of a raised haematocrit is not an indicator of hypovolaemia in decompression illness
D. symptoms and signs are always obvious to an examining physician
E. peripheral vasoconstriction is a reliable sign of hypovolaemia in spinal cord decompression illness with paresis.

Question 5. Regarding treatment of hypovolaemia in decompression illness:

A. hypovolaemia in cases of decompression illness can be managed using ATLS guidelines
B. extravasation of plasma in decompression illness is reversed by recompression therapy
C. recompression treatment is unnecessary if all symptoms resolve after correction of hypovolaemia
D. administration of oral fluids can reliably treat dehydration or hypovolaemia identified after diving
E. correction of hypovolaemia with intravenous fluid is contraindicated in cases with increased cerebral or pulmonary capillary leak
F. hypotonic oral or intravenous fluids are to be avoided in the treatment of decompression illness.

Letter to the Editor

The death of buddy diving?

Dear Editor,

By focussing on the details of the Watson case, I believe Bryan Walpole has missed the thrust of my earlier letter.[1,2] I agree this was a complex case, which is why I deliberately avoided the murky specifics in order to consider the 'big-picture' ramifications of the judgement. My concerns relate to the potential consequences of the unintended interplay between unrelated developments in the medical and legal arenas. Taken together, I believe these developments threaten the very institution of buddy diving.

I have been unable to verify Dr Walpole's claim that the statute under which Mr Watson was convicted has not been used previously in a criminal trial. I must, however, refute his assertion that this legislation is some sort of idiosyncratic historical hangover or legal curiosity unique to Queensland. Although the original legislation pre-dates Australian federation, this statute has survived intact through 110 years of reviews and amendments to the Queensland Criminal Code. The application of this 19th century law to the Watson case now provides a direct, post-federation, 21st century relevance. Nor is Queensland alone in having such a statute on its books. Section 151 of the Criminal Code Act in Dr Walpole's home state of Tasmania states "*When a person undertakes to do any act, the omission to do which is or may be dangerous to human life or health, it is his duty to do that act*."[3] Similar statutes can also be found in the legislation of other Australian states and as far afield as New Zealand and Canada.[4–6] The phrasing of the relevant sections is, in many cases, almost identical to Queensland's, reflecting the common judicial heritage of these places.

Even if this ruling's reach extended no further than the Queensland border its ramifications would be immense. Tourism statistics reveal that over 1.2 million visitors perform nearly 3.5 million dives/snorkels in Queensland each year.[7] An estimated 93% of international divers visiting Australia stopover in Queensland and 40% of domestic recreational diving holidays occur there.[8] This ruling, however, has implications potentially far beyond this single State. In the absence of local precedents, courts may examine precedents arising in other jurisdictions with which they share a common legal heritage. Rare cases may indeed make bad law but precedent is one of the cornerstones of our legal system.

The medical profession, through the revised SPUMS guidelines on recreational diving medical examinations, has now made explicit the level of support expected from dive buddies (e.g., to diabetic divers). The legal profession, through the Watson judgement, has demonstrated the potential consequences of failure to perform an act that a diver has undertaken to perform towards their buddy. The halcyon days of casually agreeing to act as someone's buddy are now gone. Serious consideration should be given to the personal consequences of undertaking this role. The potential to face a custodial sentence for criminally negligent manslaughter if a diver fails in his/her duty-of-care increases the pressure to save their buddy at all costs – or die in the attempt.

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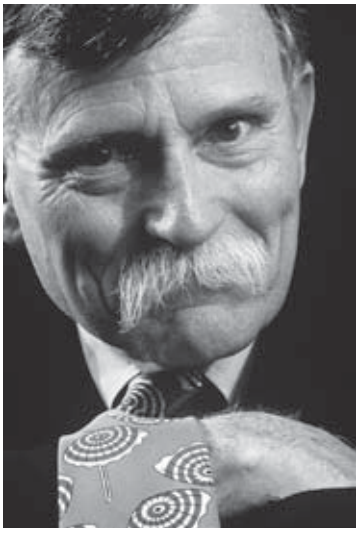
Key words
Medicals – diving, buddies, diving deaths, legal and insurance, letters (to the Editor)

Obituary

R W 'Billy Bob' Hamilton, 1930-2011



Bill Hamilton's daughter Lucy summed up her father in saying "*It's best said that Billy Bob was larger than life. And, by the way, know any other 81-year-olds who are still updating their résumé?*"

Bill Hamilton, PhD, had over four decades in diving, aerospace and environmental physiology, with a particular interest in decompression, breathing gases, and the effects of pressure. From 1964 to 1974, he was employed by Union Carbide as director of a leading environmental physiology and diving research laboratory which served as the research arm of Ocean Systems, Inc. From 1976, Bill was principal of his own consulting firm, Hamilton Research, Ltd. His work there included the development and assessment of decompression tables and operating and safety procedures for commercial, scientific, recreational, and military diving, for caisson work, and for aerospace and hyperbaric medicine. Amongst other roles, he has served as consultant to NASA, the Swedish Defense Research Agency, the German GKSS laboratory, the Japanese Navy, the Norwegian Underwater Institute and NOAA's Aquarius sea-floor habitat project, for which he developed operational and evacuation decompression tables. Further work with NOAA, on the *USS Monitor* project which led to the development of trimix tables, resulted in nitrox saturation-excursion procedures and NOAA's Repex procedures for repetitive excursion diving, which also include a serendipitous but now widely applicable algorithm for managing long-term exposure to oxygen.

He pioneered the study of neon as a breathing gas component, studying human performance and decompression aspects of this gas to 400 metres' sea water (msw). He advocated the use of neon as the ideal inert gas for long-duration space flight, and collaborated in neon's use offshore in a series of deep commercial dives, and in exploring its value when used with closed-circuit breathing systems. The hyperbaric chamber fire safety programme developed with colleagues in his lab produced the fundamental data now used as guidelines by the National Fire Protection Association and other standards organizations. His concept of the "*zone of no combustion*" led to the development of safe atmospheres and breathing gases for underwater habitat welding, and he worked offshore to help implement these ideas.

He led Ocean Systems' Access programme, which involved rapid compressions to 300 msw using nitrogen to mitigate the effects of the HPNS and excursions from saturation. He worked with Shell in Norway to develop an operational diving capability to 450 msw. He has been principal investigator in two cooperative repetitive saturation-excursion diving projects at the Chinese Underwater Technology Institute, Shanghai, Chisat I (1988) and Chisat II (1995); the latter was the first extensive study of excursions with trimix breathing mixtures from a nitrogen-based habitat environment.

With various colleagues, his work on decompression procedures for a wide range of gas mixes and pressure exposures has evolved into the comprehensive computer programme, DCAP, used widely today throughout the world. With Dave Kenyon, he was very recently completing a new and original dive computer, DCAP-X, which incorporates the DCAP computational programme for use in technical diving. He has made many other contributions to the development of recreational technical diving. At a time when many diving physicians were vehemently opposed to this new activity, Bill's approach was the opposite, to help make 'teckie' diving safer.

Bill was a past Chairman of the Board of Directors of the Divers Alert Network (DAN). He was also a charter member of the Undersea and Hyperbaric Medical Society, serving on its Board of Directors and several committees and as Editor of its newsletter, *Pressure*, and on the editorial board of *Undersea and Hyperbaric Medicine*. One of Bill's specialties was 'workshops', which he guided or participated in on a huge range of topics. In 1996, he was Principal Guest Speaker at the SPUMS Annual Scientific Meeting, which focused on technical diving for the first time.

Major Hamilton was a fighter pilot, serving in Alaska during the Korean War and in combat service in Viet Nam, where he earned the Distinguished Flying Cross, Air Medal, and other decorations. As Life Support Officer, he helped solve an equipment problem that had caused unsuccessful bailouts. He has been a principal investigator for both USAF and NASA in research on atmospheres for space flight.

Dr. Hamilton received many meritorious scientific and other awards in his time, as well as being the proud recipient of his dunking as 'Diver of the Year' by the Boston Sea Rovers. He has numerous scientific and technical publications, contributions to many books, reports, and workshops and a major article on Life Support in the Encyclopedia Britannica. His important contributions to operational manuals and training materials will be a dynamic legacy in the diving world for many years to come.

The thoughts and good wishes of both Societies are extended to Dr Hamilton's surviving family, as we all remember a man who contributed enormously to the fields of diving and aerospace research and operational safety. 'Billy Bob' was indeed larger than life.

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EUBS European Underwater and Baromedical Society

38th EUBS Annual Scientific Meeting 2012

First Announcement

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

11–12 September: ECHM Consensus Conference
Organisation of a clinical hyperbaric therapy centre and related health management issues
12–15 September: EUBS Annual Conference
16 September: DAN Divers Day

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

For the first time the EUBS Annual Meeting will be preceded by an ECHM Consensus Conference on the organisation of a clinical hyperbaric therapy centre and related health management issues. This offers the unique opportunity to participate in two highly significant events for the European hyperbaric community.

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

Conference main topics:

Pressure physiology and medicine
Diving physiology and medicine
Basic research in hyperbaric medicine
New frontiers of HBOT
Hyperbaric oxygenation fundamentals
Cost-benefit in HBOT
Nursing in hyperbaric medicine practice

Preliminary timetable:

February 2012: Second announcement
15 May: Deadline for submission of abstracts
15 June: Notification of accepted abstracts

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

Contact details:

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Fax: *+381-(0)11-2650-823*
E-mail: *<chm@scnet.rs>*

Minutes of the Executive Committee (ExCom) of EUBS Meeting, Gdansk Medical University, Gdansk, 26 August 2011

Opened: 1403 h

Present:
P Germonpré, C Balestra, J Schmutz, P Knessl, A Møllerløkken, JM Pontier, P Müller, P Bryson, N Bitterman, AO Brubakk, P Wooding
Invited: Mariana Sedlar (Serbia), Jacek Kot (Poland)

Minutes 2010:
Accepted by e-mail and published on the website and in DHM; no further comments.

Secretary General's report 2011:
An overview is given by J Kot, Secretary General of the EUBS 2011 Meeting.
Extra copies of the Book of Abstracts and Posters will be sent to the EUBS Library (managed by Dr Arvid Hope from NUTEC). An electronic copy should be sent to ExCom and also to the DHM European Editor.
ExCom congratulates the organisers for making a book of abstracts to include a blank page for every oral presentation, allowing the writing of personal notes.

Update of EUBS 2012:
Presented by Marian Sedlar and Miodrag Zaric from the local organising committee.
There are no major problems in the organisation. A poster and leaflets have been prepared for distribution to EUBS 2011 participants. There will be satellite events from ECHM, DAN, EBAss. The satellite conferences will be timed separately from the main EUBS ASM. The general progress is approved by ExCom. There is some concern that in allowing too many satellite meetings around the EUBS, the visibility of EUBS could be impaired. This should definitely be considered in the planning for future meetings.

Update EUBS 2013:
The history of this issue is summarised by Peter Germonpré. In 2009, ExCom proposed a joint meeting with SPUMS in a location between Europe and Australia. Réunion was felt suitable. Any diplomatic difficulties have now been cleared. SPUMS and SAUHMA have expressed major interest, so we are now working towards a three-society meeting. Formal accords will be requested from SPUMS and SAUHMA Executive Committees.
Actual state of affairs:
Location: either Réunion (preferred) or Mauritius;
150–200 participants to be expected
Main logistic problem: to find a conference venue within (ideally) one single hotel for 150 persons; organise diving for at least 100 persons.
Timing: best season for diving in Réunion is September
SPUMS: usually have their meeting in May
UHMS: June (must not conflict)
SAUHMA (South African Undersea and Hyperbaric Medical Association: are flexible in dates
Programme structure:
One week programme
Diving activities
Satellite meetings?
Timing:
Finalise location and dates by end 2011
Constitute scientific committee : EUBS + SPUMS + SAUHMA
Local organiser: will be welcomed to facilitate local contacts.
Scott Haldane Foundation (The Netherlands) prepared to help out with practical organisation, including hotel booking and diving. Flights to be arranged and booked by individuals.
ExCom will take a lead in arranging the scientific programme; see below.

Next meetings:
2014: no proposal received yet. Peter Knessl suggests the possibility of Geneva, as it is central in Europe and could be a Swiss organisation. He will follow this further.
2015: An official proposal was received from The Netherlands (Amsterdam AMC – Albert van den Brink and Rob van Hulst). They propose to organise EUBS in conjunction with the rally of big sailing ships taking place in Amsterdam at that time. ExCom agrees and will officially confirm.

Travel grant:
There has been only one application for a travel grant, which was cancelled because the applicant did not provide proof of studentship.

Zetterström Committee:
Committee members: J Schmutz, P Knessl (EUBS ExCom) M Hajek (local scientific committee). The selected poster was: M Cervaens, O Camacho, R Resende, F Marques, P Barata Coelho (Portugal). *HBOT treatment for the recovery of muscle injury induced in rats*.
The registration to the next EUBS will be paid by EUBS if the final paper is sent within 12 months to DHM for peer review and possible publication.

Ballots:
The internet-based voting system is generally accepted as easy and quick. The relatively low voting rate may be explained in part by the fact that there was only one option to vote for. Low response rates have also occurred with postal ballots.
Member-at-Large: one candidate (Fiona Sharp) is elected.
Peter Knessl is leaving the ExCom after his three-year term: many thanks are expressed.
Next year's election will need candidates for Member-at-

Large and Vice-President. ExCom members are tasked to identify suitable candidates.

Diving and Hyperbaric Medicine Journal (Peter Müller)

The Annual Report of the Editor-in-Chief, Mike Davis, was discussed.

The main goal for this year, indexation in MedLine, was achieved. Issues for the three years before indexation will be available in the PubMedCentral database (this needs a transformation into searchable XML format files). This will not directly aid to increase the Journal's Impact Factor, but at least all content will be visible and searchable.

The second goal, financial restructuring, is still for discussion.

The third goal, the dedicated website of the Journal, is still under construction.

The new advertising policy, which hopefully will bring financial support to the Journal, was published in the September issue.

Journal contents mainly decided on by the Editor-in-Chief. The European Editor is rarely consulted. Article reviewers are chosen by the Editor-in-Chief on the basis of the content of the article. One reviewer is chosen from the Editorial Board, the other one is external.

There have been inquiries from non-members of EUBS and SPUMS to receive DHM. The topic has been discussed by PG with the SPUMS President and possible solutions have been identified. This matter will officially be put forward to the SPUMS Committee.

Costs for attendance of Editors to Annual Scientific Meetings: Registration fees are classically waived by the Congress Organisers, and this should be continued. Travel costs will be incorporated in the annual journal budget, allowing attendance to both meetings, where the Editor-in-Chief and European Editor should coordinate between themselves to do this in the most economical way. This should be part of the global budget restructuring of the Journal.

Website (Peter Germonpré):

There have been no major changes to the website, which is kept up to date by Peter Germonpré.

People are more and more using electronic payment, PayPal is working well now. Some countries are not 'allowed' by the PayPal system (e.g., Egypt) so we will retain the possibility for members to pay cash at ASMs. Some technical problems have arisen, mostly due to security settings on users' computers. People can contact the webmaster or Tricia Wooding in case of problems, which should be quickly resolved.

Membership:

Combined membership of SPUMS/EUBS: A member asked if he could receive a reduced fee and not receive the Journal twice. This has been discussed before and there is still no definite answer to this. It will be discussed further and put forward to the SPUMS Committee.

Financial matters (Patricia Wooding):

The ONR sponsoring for EUBS 2011 (to be used for invited speakers, travel grants and abstract book) has been paid on time, in contrast to last year's major delays. It is uncertain if there will be a possibility for a grant next year, it will be applied for a.s.a.p.

This year the finances look good, with a break-even. The involvement of ExCom in the Annual Meeting organisation becomes possible; however, if we contribute financially to a meeting in a substantial way, we could 'impose' invited speakers. Peter Germonpré proposes that EUBS should receive a percentage of the registration fee in order to be able to help in financing quality speakers for the following year. Registration fees should, however, be kept low, and taking away part of the financial burden from the travel of invited speakers should ensure that this fee is not raised. Also, ExCom agrees that a percentage of exhibition income should be transferred to EUBS. 10–20% seems reasonable. This should be definitely implemented starting 2013. Another way to increase financial possibilities in connection with the Meeting could be to increase fees for Corporate Members and, in return, offer them exhibition space at the Meeting.

Mrs Anna Stillman from DDRC has audited the books of EUBS and found in them order. They will be available for a short time on the website in the 'members-only' section.

Other business:

ExCom tasks:

We have three Members-at-Large on the Committee. The one who is in his third year of activity should be a member of the congress scientific committee – this is going to be implemented in the next meetings (for the 2012 Meeting, A Marroni, P Germonpré, C Balestra are already on the Scientific Committee)

The Member-at-large in his second year should have a function for the website, maybe responsible for the Discussion Forum, which currently is never used.

The Member-at-Large in his first year should have an observational role only.

A letter was received from the Secretary of ICHM, Mike Bennett, inviting a representative of EUBS to take part in the Executive Committee of ICHM. The main (only) task of ICHM is to organise a three-yearly scientific meeting. This is done by the President of ICHM, who is thus elected every year based on the candidatures. Phil Bryson agrees to act as representative and will contact Mike Bennett. However, any member of ExCom should be entitled to replace Phil in case of his unavailability.

Any other business:

Tino Balestra informs that the Belgian Society for Diving and Hyperbaric Medicine (SBMHS-BVOOG) proposes to

create a grant in honour of Patrick Musimu, the Belgian apnea diver who died this summer during an immersion. Patrick was passionate about research into the physiology of extreme apnea diving. This grant will reward the best oral or poster presentation on breath-hold diving. The prize, similar to the Zetterström award, will consist of free registration to the following Annual Meeting of EUBS. A Prize Committee will be nominated annually and could well be the same as the one for the Zetterström Committee. ExCom agrees in principle to second this grant and organize the Prize Committee – this matter will be presented for vote to the General Assembly.
NTNU has sponsored a nice EUBS banner for Tricia Wooding, and is officially thanked for this. The banner will help her to be more visible during EUBS meetings.

Closed: 1632 h

J Schmutz
Honorary Secretary, EUBS

Minutes of the EUBS General Assembly Gdansk, 28 August 2011

Opened: 1252 h

Welcome:
The President, Peter Germonpré (PG), welcomes all the participants.

Minutes of GA 2010, Istanbul:
The minutes from the General Assembly 2010 are accepted by accolade.

Status of the 2011 Annual Scientific Meeting:
PG thanks the meeting organisers for a well-conducted meeting with excellent and professional technical staff. 269 persons attended the meeting, of whom 118 were members EUBS/UHMS/SPUMS, meaning that the proportion of EUBS members still is too small and more effort should be made in the Society to promote the meetings; 21 students or nurses and 22 accompanying persons; registrants came from all continents (38 different countries). There were 34 oral presentations, 4 invited lectures and 2 pro/con debates; 37 posters were submitted, of which 34 were presented. The pro/con debate system is approved by the members and will be pursued during the next meetings. Dr Kot thanks his staff for the work done. Patricia Woodings presents a small present to the members of the organising committee on behalf of the EUBS.

Awards and grants:
Fourteen posters were submitted for the **Zetterström Award**. The Zetterström Committee (Jörg Schmutz and Peter Knessl, both members of the Executive Committee of EUBS and Michal Hajek member of the local scientific committee) awarded the prize to a poster from Portugal:

M Cervaens, O Camacho, R Resende, F Marques, P Barata Coelho. *HBOT treatment for the recovery of muscle injury induced in rats*.

The Award (free registration for next EUBS meeting) is basically a research stimulus and is conditional upon submission to *Diving and Hyperbaric Medicine* (DHM) Journal for peer review.

There was no application for the travel grant this year, which is rather surprising because many students were attending the meeting. The EUBS will again offer travel grants for next year's meeting.

EUBS publications (journal and website):
Peter Müller (PM), European Editor of DHM, informs the GA that our journal has received an Institute of Scientific Information (ISI) Impact Factor of 0.356 for the year 2010. As a comparison the UHMS journal (UHM) has an Impact Factor (IF) of 0.694. Last year's numbers were respectively 0.491 for DHM and 1.046 for UHM. DHM is now indexed on PUBMED, which is very encouraging. The fall in the IF happened for all the journals in the field last year and must not be taken as too serious a setback. It is expected that with increased visibility now that PubMed indexing is achieved, the number of referenced articles, and thus the Impact Factor, will increase again.

Members are asked to visit the members' area of <www.EUBS.org> to check that their address data and e-mail are still correct in order to ease the administrative work of the secretary. Members should also visit the literature database offered by GTUeM and the list of ongoing research projects. The latest version of DHM is downloadable from here as well; however, this is a service to EUBS and SPUMS members only.

Financial report:
The financial report was prepared by our membership secretary, Patricia Wooding (PW) and projected to the GA. The audit was conducted by Anna Stillman from DDRC in Plymouth, who is thanked by ExCom for her voluntary efforts. The financial report is accepted by the General Assembly and will be accessible for a limited period of time on the society's website (members-only section). We have a rather good balance and are slowly recovering from the bad years.

Votes and elections:
The results of the voting ballot were as follows: Member-at-Large 2011, one candidate, Fiona Sharpe, who was accepted by the vast majority of voters, will replace Peter Knessl who has finished his three-year term.

Unfortunately only 103 out of 208 registered members have voted. One possible reason for this weak result could be the lack of motivation for the members because there was only one candidate. On the other hand, some technical problems were reported by members. A solution will be sought for these. The members are asked to make proposals for next year's elections. We need a new Vice-President and a new Member-at-Large. Peter Germonpré will finish his term next year and Tino Balestra will be the next President.

Next meeting:

The next meeting will be held in Belgrade, Serbia on 12–15 September. An ECHM Consensus Conference will take place at the same location on 11–12 September. A nice video presentation is shown. The 2013 meeting is not yet definitely settled. It will be either La Réunion or Mauritius. Both SPUMS and the South African Underwater and Hyperbaric Society (SAUHMA) are interested in joining. The challenge will be to find suitable hotels and diving logistics for all participants. 2014 is still open, possibly Geneva. Adel Taher has proposed Sharm el Sheikh again, but since the annual meeting was held in Egypt in 2007, it is too early. Furthermore, the political situation in Egypt remains unstable. Amsterdam has applied for 2015. There are no proposals after this date and the audience is asked for volunteers.

Phil Bryson (PB) has done a good job as Liaison Officer, and ExCom proposes to prolong his mandate; accepted by the membership. The International Congress on Hyperbaric Medicine <http://www.ichm.net> wants a liaison member, this is offered to Phil who accepts this task. PB will contact SPUMS to find a solution for members who are or want to be members of both societies at the same time. Both societies will have to discuss this problem and make a proposal.

ExCom will help the future organisers with the scientific programme. In order to increase the attractiveness of the meetings, ExCom will participate in the scientific committees of the meetings, select and pay guest speakers. For this purpose, EUBS will ask for a small percentage of the congress and exhibition fees. From the audience, it is suggested that the membership fee be increased as an alternative way to increase finances. PG indicates that this is not desirable, as the membership fee was increased considerably some years ago. Furthermore, an increase would mean that EUBS fees are higher compared to SPUMS. Another proposal is made to open the door for companies. PB answers that we already have Corporate Members; furthermore we have open the possibility to advertise in DHM.

Miscellaneous:

No other business from the floor.

PG informs the audience that the Belgian Society for Diving and Hyperbaric Medicine (SBMHS-BVOOG) proposes to create a grant in the name of Patrick Musimu, the Belgian apnea diver who died this summer during an immersion. Patrick was passionate about research into the physiology of extreme apnea diving. This grant will reward the best oral presentation or poster on breathhold diving at each EUBS ASM. The prize, similar to the Zetterström Award, will consist of a free registration to the next EUBS ASM. A prize committee will be nominated annually. There were no objections from the floor.

The NTNU (Norwegian Technical University) has graciously donated EUBS a nice roll-up banner for the Secretary to use during the meetings.

Closed: 1336 h

J Schmutz
Honorary Secretary, EUBS

SPUMS notices and news

South Pacific Underwater Medicine Society Diploma of Diving and Hyperbaric Medicine

Requirements for candidates (updated October 2008)

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

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

Additional information – prospective approval of projects is required

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

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

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

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

The Academic Board reserves the right to modify any of these requirements from time to time. As of October 2011, the SPUMS Academic Board consists of:
Associate Professor David Smart, Education Officer
Associate Professor Simon Mitchell
Associate Professor (retired) Mike Davis.

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

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

South Pacific Underwater Medicine Society

41st Annual Scientific Meeting 2012
Preliminary announcement

Dates: 20–27 May 2012
Venue: Madang Resort, Madang, Papua New Guinea

Theme:
What lies beneath: the pleasures and perils of our diving environment

Keynote speakers:
Associate Professor Jamie Seymour, James Cook University, Queensland,
"*the jelly dude*"
Richard Fitzpatrick, James Cook University, Queensland
"*the shark guy*"

Call for abstracts, conference information and registration forms

Abstracts:
Abstracts for presentation should be submitted before 31 March 2012 as a Word file of up to 250 words (excluding references – four only) and with one figure. Please forward to, e-mail: <cmeehan@mcleodstmed.com.au>

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*. 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 2012 Convenor
Dr Cathy Meehan
E-mail: <cmeehan@mcleodstmed.com.au>
Mobile: +61-(0)4-1778-3653

For further information copy the link below into your web browser:
<http://www.madangresort.com/convention_centre/spums-meeting-2012/>

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

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The

website is at
<www.spums.org.au>
Members are encouraged to log in and to keep their personal details up-to-date

Rear Admiral Robyn Walker AM, RAN, MBBS, Dip DHM

It is with the greatest of pleasure (and a significant measure of pride) that SPUMS can announce that long-term member and past President, Commodore Robyn Walker AM RAN is to be promoted to Rear Admiral in the Royal Australian Navy (RAN). In December, she will take on the dual roles of Surgeon-General of the Australian Defence Force as well as Commander – Joint Health Command, and, as the Navy's first female Admiral, will be the highest ranking woman to have ever served in the RAN.

Dr Walker qualified as a medical practitioner in 1982 and spent the next nine years working in the public health system in Queensland. A senior colleague from those days recalls her "*quiet persistence and humility as a young doctor*". Her final position in Queensland, before she joined the RAN in 1991, was as Director of Hyperbaric Medicine at Townsville General Hospital.

Much of the next decade was spent working full-time in diving and hyperbaric medicine, including submarine escape and rescue and, from 1996 to 2000, as Officer-In-Charge of the Submarine and Underwater Medicine Unit (SUMU) at HMAS PENGUIN in Sydney. In 2000, she was posted to Defence Headquarters in Canberra with promotion to Commander, then to Captain and to Commodore in 2005. Subsequent to leaving SUMU, her roles have included Staff Officer positions in Joint Operations Command, as well as a succession of senior positions in preventative health, development of health policy and delivery of health operations, with her most recent roles being Director-General – Health Capability and also Director-General – Naval Health Service. To each of these positions, Robyn devoted her considerable energies and clarity of thought, resulting in a military career which can only be described as remarkable. On 26 January 2010, Robyn was appointed as a Member in the Military Division of The Order of Australia for exceptional service as a medical officer in the Australian Defence Force.

As many of you will know, Robyn has contributed enormously to SPUMS as a committee member from 1995 to 1999, President from 1999 to 2005 and as Convenor of several Annual Scientific Meetings (ASMs). She was an outstanding President who overcame a number of major challenges in her time at the helm, including a serious financial crisis for the organisation. Robyn has published a number of papers in the *SPUMS Journal* and *Diving and Hyperbaric Medicine* over the years on a range of topics including decompression illness in RAN divers, women in diving, and submarine escape and rescue, and contributed to ASMs on many other topics. During her time in Queensland, she was awarded the SPUMS Diploma in diving and hyperbaric medicine. She is co-author with Carl Edmonds, Chris Lowry and John Pennefather of the widely acclaimed diving medicine textbook *Diving and Subaquatic Medicine*. Robyn also played a pivotal role in negotiations with EUBS

in 2007 to establish *Diving and Hyperbaric Medicine* as the joint publication for our two societies.

What none of this list of achievements identifies, however, is what a superb, supportive, yet unassuming colleague she has been for many of us over the years and what outstanding qualities of leadership she provides in any role she undertakes – attributes that the Australian Defence Force has clearly recognised.

As current SPUMS President Mike Bennett has pointed out, Robyn also has a lot to teach about underwater efficiency, being widely known in SPUMS for her economical gas consumption and graceful movement through the water column. This is not surprising, given her extensive diving experience.

On behalf of the Society, we would like to recognise this very significant achievement and extend our warmest congratulations to our colleague and friend on the occasion of her promotion. We wish her well in her upcoming role, and sincerely hope that she still manages to find the time for an occasional dive in her constantly busy schedule. It just goes to show that working at depth does not preclude rising to high places!

ANZCA Certificate in Diving and Hyperbaric Medicine

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

Eligibility criteria are:

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

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

3 **EITHER:**
   a Completion of the Diploma of the South Pacific Underwater Medicine Society, including six months' full-time equivalent experience in a hyperbaric unit and successful completion of a thesis or research project approved by the Assessor, SPUMS
   b **and** Completion of a further 12 months' full-time equivalent clinical experience in a 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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SPUMS Award, Hyperbaric Technicians and Nurses Association ASM 2011

The SPUMS Award for the best presentation by an HTNA member at their Annual Scientific Meeting 2011 in Sydney was given to Derelle A Young, BN,MN,PGCertNSc (Intensive Care), Clinical Nurse, Hyperbaric Medicine Unit, The Townsville Hospital, Queensland.

Her project was entitled:
Transcutaneous oximetry measurement: normal values for the upper limb

Co-authors were Denise F Blake BN, MD, FRCPC, FACEM, PG Dip Med Sci (DHM), Staff Specialist, Emergency Department, The Townsville Hospital.
and Lawrence H Brown MPH & TM, Senior Principal Research Officer, School of Public Health, Tropical Medicine and Rehabilitation Services, James Cook University, Townsville.

Advertising in *Diving and Hyperbaric Medicine*

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

Details of advertising rates and formatting requirements for publication may be obtained on request to this office at:
E-mail: *<editor@dhmjournal.com>*
Fax: *+64-(0)3-329-6810*

The Poetry Doctor

Hyperbaric stress

I'm just an oxygen molecule living under stress
Trying to make a living under pressurised duress.
In these days of unemployment and budgetary restrain
Where can I be useful for monetary gain?
Alas CO poisoning once common in the past
Is now rarely encountered thanks to natural gas.
Thank God for diabetics. They are truly heaven sent
For their slowly healing ulcers now pay the basic rent.
I keep my image sexy with the diver's DCI
As their dramatic presentations hold me high in the public eye.
But I need more for my pension, new chambers and new staff
So I pray there's more infections by a necrotising Staph.
Don't see me as cold hearted only thinking what I earn
But I love a skin flap failing or a crush or major burn.
I'd like to expand the business to autism and MS,
Fibromyalgia and psoriasis but they're considered in excess.
So I follow current research for promising inclusions
Like sensorineural deafness and retinal artery occlusions
Or jaw osteonecrosis from bisphosphonate.
They may not be numerous but they all remunerate.
Don't judge me too harshly if my ambition has exceeded,
I only want a living and to feel that I am needed.

John Parker
< drjohnparker@hotmail.com>

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

Dates: 20 February–02 March 2012
Venue: Prince of Wales Hospital, Sydney, Australia

Faculty includes Associate Professors Mike Bennett, Simon Mitchell and David Smart.
Course content includes:

- History of hyperbaric oxygen
- Physics and physiology of compression
- Pressure-related injuries (barotraumas, decompression illness)
- Accepted indications for hyperbaric oxygen therapy
- Wound assessment, including transcutaneous oximetry
- Gas toxicities, marine envenomation, drowning
- Practical sessions, including assessment of fitness to dive
- Visits to HMAS Penguin and the NSW Water Police

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

Contact for information:
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>

Royal Adelaide Hospital

Medical Officers Course
December 2012
Unit 1 3–7 December
Unit 2 10–14 December
Diving Medical Technician (DMT) – Full Course
May 2012
Unit 1 21–25 May
Unit 2 28 May–1 June (lecture week)
Unit 3 4–8 June
July/August 2012
Unit 1 30 July–3 August
Unit 2 6–10 August (lecture week)
Unit 3 13–17 August
DMT – Refresher Course
January 2012
Unit 1 9–13 January
Unit 2 16–20 January

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

Scott Haldane Foundation

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

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

Course details for 2012

24 February–03 March: In-depth Course - Decompression and HBO therapy (Sipadan/Mabu)
10–17 March: Malaysia Basic Course Diving Medicine for ENT and Pulmonary Specialists (Bonaire)
24 and 30 March: Basic Course Part I (Loosdrecht NL)
14, 20 and 21 April: Basic Course Part II (Amsterdam NL)
08–15 May: Basic Course Part II (Dahab, Egypt)
22 September: Refresher Course Diving Medical Examiner, (Amsterdam NL)
09–17 November: Basic Course Part I (Maldives)
16–24 November: 20th In-depth Course – Diving Medicine (topics to be confirmed) (Maldives)
23 November–01 December: 20th In-depth Course – Diving Medicine (topics to be confirmed) (Maldives)

For further information: <www.scotthaldane.nl>

Asian Hyperbaric & Diving Medical Association

8th Annual Scientific Meeting

Venue: Phuket, Thailand
Dates: 26–28 July 2012
Pre-conference DMAC-EDTC Level IIa top-up course 23–25 July 2012

For registration details and all enquiries please go to: <www.ahdma.org>

DIVING HISTORICAL SOCIETY AUSTRALIA, SE ASIA

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

Hyperbaric Oxygen, Karolinska

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

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

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

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

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

German Society for Diving and Hyperbaric Medicine (GTUeM)

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

Inter-university Diploma in Diving and Hyperbaric Medicine, France

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

Undersea and Hyperbaric Medical Society

45th Annual Scientific Meeting
Preliminary announcement

Dates: 20–23 June 2012
Venue: JW Marriott Desert Ridge Resort, Phoenix AZ

Contact: <www.uhms.org>

Instructions to authors

(Short version, updated December 2011)

Diving and Hyperbaric Medicine welcomes contributions (including letters to the Editor) on all aspects of diving and hyperbaric medicine. Manuscripts must be offered exclusively to *Diving and Hyperbaric Medicine*, unless clearly authenticated copyright exemption accompanies the manuscript. All manuscripts will be subject to peer review. Accepted contributions will also be subject to editing. An accompanying letter signed by all authors should be sent.
Contributions should be sent to:
The Editor, Diving and Hyperbaric Medicine,
C/o Hyperbaric Medicine Unit, Christchurch Hospital,
Private Bag 4710, Christchurch, New Zealand.
E-mail: *<editor@dhmjournal.com>*

Requirements for manuscripts
Documents should be submitted electronically. The preferred format is Microsoft® Office Word or rich text format (RTF). Paper submissions will not be accepted. All articles should include a title page, giving the title of the paper and the full names and qualifications of the authors, and the positions they held when doing the work being reported. Identify one author as correspondent, with their full postal address, telephone and fax numbers, and e-mail address supplied. The text should generally be subdivided into the following sections: a structured Abstract of no more than 250 words, Introduction, Methods, Results, Discussion, Conclusion(s), Acknowledgements and References. Acknowledgements should be brief. Legends for tables and figures should appear at the end of the text file after the references. Conflicts of interest and funding sources should be identified.

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

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

Illustrations, figures and tables must NOT be embedded in the wordprocessor document, only their position indicated, and each should be submitted as a separate file.
Tables should be presented either with tab-separated columns (preferred) or in table format. No gridlines, borders or shading are to be used.
Illustrations and figures should be submitted 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 printing is available only when it is essential and will be at the authors' expense. Indicate magnification for photomicrographs.

References
The Journal reference style is based closely on the the *International Committee of Medical Journal Editors (ICMJE) Uniform Requirements for Manuscripts*. Examples are given in detail at:
<http://www.nlm.nih.gov/bsd/uniform_requirements.html> (last updated August 2009). References must appear in the text as superscript numbers at the end of the sentence after the full stop.[1,2] Numbered them in order of quoting. Use Index Medicus abbreviations for journal names: <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(s) before being considered for publication.

Consent
Studies on human subjects must comply with the Helsinki Declaration of 1975 (revised 2000) and those using animals must comply with health and medical research council guidelines or their national 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 July 2011) may be found on the DHM Journal, EUBS and SPUMS websites and should be consulted before submission.

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)
+10-4500-9113 (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.

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

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