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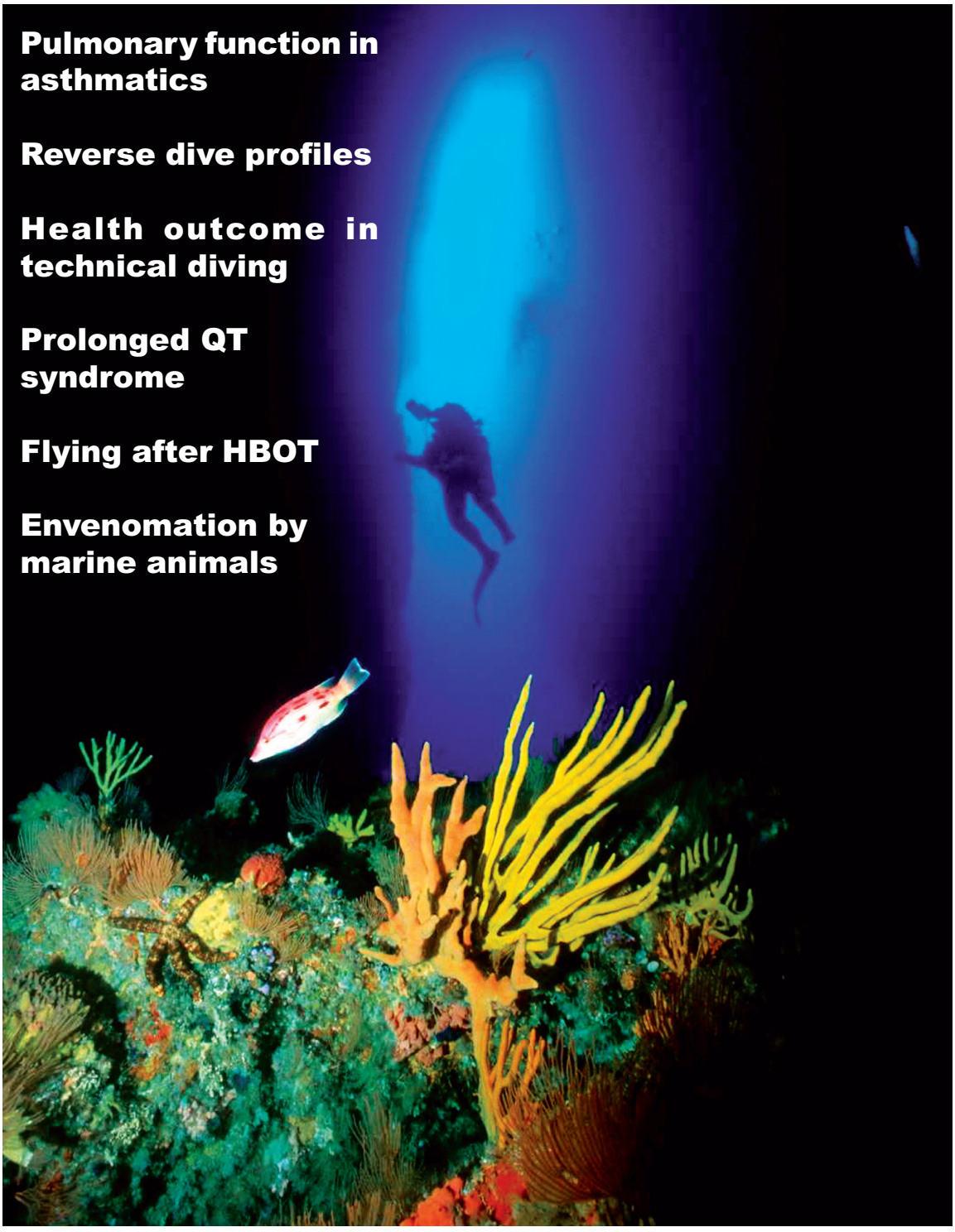

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PURPOSES OF THE SOCIETY

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
To convene members of the Society annually at a scientific conference

OFFICE HOLDERS

ADMINISTRATION

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Membership is open to all medical practitioners.
Associate membership is open to all those who are not medical practitioners but are interested in the aims of the Society, and/or those engaged in research in underwater medicine and related subjects.
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The Society's financial year is January to December, the same as the Journal year.
The 2005 subscription will be Full Members A$132.00 and Associate Members A$66.00, including GST.
There will be an additional surcharge of $8.00 for journal postage for all members living outside Australia.

The Editor's offering

Having something relevant and interesting to say in these editorials is a challenge, as those who have stood in these shoes before will know. Right-hand pages early in a journal are the most often read, so there is a fair chance that most readers peruse this page. It is, therefore, reassuring to receive from members appreciative comments about our efforts to provide interesting and stimulating content in the Journal. In this, of course, we are endebted to our authors whose efforts are greatly appreciated, though there may be times in dealing with the review process when they wonder about that! We are eager to hear from members about ways to improve the Journal; it is, after all, your journal. Now that its new name of *Diving and Hyperbaric Medicine (the Journal of the South Pacific Underwater Medicine Society)* has been ratified, the process of putting this change in place will commence. This is not necessarily a simple process as the impact on EMBASE and registration of the new name need to be considered and may take time.

Of all the issues in diving, asthma and 'fitness to dive' has been perhaps the most contentious. This journal has contributed regularly to the debate. The article by Gotshall et al provides new data on how individuals with bronchial hyperreactivity interact with breathing apparatus. Their findings of a significant fall in respiratory function when breathing from a scuba regulator even at normal ambient pressure, and especially during exercise, are highly relevant to this debate. Clearly, we also need to see what further effect increased pressure will have.

Our difficulty is turning such physiological data into something meaningful regarding risk. I believe there is little doubt that asthmatics are at greater risk, but as yet we have no means of quantifying this. Whether this risk is unacceptable in diving (an entirely prescriptive approach to 'fitness to dive'), or whether appropriate individual risk analysis and a mutual decision-making process is the right approach remains an open question, and one that has certainly polarised medical opinion in this and other Societies. The fact that approaches differ widely around the world indicates that there may be no one right answer, and that obtaining meaningful epidemiological data is fraught with difficulty.

Self-reporting has considerable potential for bias. However, the efforts of the Plymouth group to collect data from a large cohort of women divers over several years are starting to yield some interesting returns. Subjects recruited in this study were not aware that an analysis of reverse dive profiles (the reverse of the 'deepest dive first' rule for repetitive dives) was intended. Projects like this and DAN's Project Dive Exploration will enhance our knowledge of just what recreational divers do. This then allows sensible questions to be asked about what interventional strategies could be investigated to enhance diving safety.

Endpoints such as mortality and decompression illness are epidemiologically inadequate since their frequency in recreational diving is so low, as illustrated in this study. What is now needed is to combine such data gathering with approaches to health surveillance like that carried out by Doolette in several diving groups to look at subtler health-status markers. Doolette's report in this issue on a group of deep, technical cave divers illustrates this approach. This paper provides an interesting insight for diving physicians into the sort of activities undertaken by the growing community of technical divers. I have commented in a previous editorial that Doolette's approach requires validation by other groups, and this remains the case.

Sudden death in children and young adults is well recognised. The case report from South Australia illustrates this in a young-adult male diver with long QT syndrome. There were at least three young divers in the New Zealand database who died inexplicably, but were known to have some form of cardiac arrhythmia such as paroxysmal supraventricular tachycardia or Wolff-Parkinson-White syndrome (unpublished observations), and there are several more such cases in the Project Stickybeak database.[1] A prescriptive approach to prohibition from scuba diving is the only appropriate one for such individuals.

The Noumea ASM this year had many excellent presentations. Peter Fenner, a world authority, commences his series of articles from the meeting with a succinct review on marine envenomation. He makes the distinction between envenomations that place a concentrated dose of venom in a small area (e.g., blue-ringed octopus) and those where the venom is spread over a large area (e.g., jellyfish stings), and then goes on to summarise the major envenomations, their presentation and principles of management in a pithy, easy-to-read style. Chris Acott provides a review of flying after treatment for decompression illness and comes to the firm conclusion that a gap of four weeks is needed, based on a detailed, logical argument. This topic was also discussed at the workshop on decompression illness in remote locations that preceded the Undersea and Hyperbaric Medical Association's Sydney meeting in May.

We wish Society members a happy summer (winter) holiday and a safe and successful 2005 in all that you do. We trust that certain aspects of this issue of the Journal will not be taken too seriously; and don't drink and dive (or drive)!

Michael Davis

Reference

1 Walker D. *Report on Australian diving deaths 1972-1993*. Melbourne: JL Publications; 1998.

The photo on the front page was taken by Dr Simon Mitchell at Northern Arch, The Poor Knights Islands, New Zealand. It catches the Editor searching for good articles for the Journal amongst the nooks and crannies.

Original articles

Severity of exercise-induced bronchoconstriction during compressed-air breathing via scuba

Robert W Gotshall, Laurie J Fedorczak and Jeremy J Rasmussen

Key words
Diving, asthma, exercise, scuba, pulmonary function, research

Abstract

(Gotshall RW, Fedorczak LJ, Rasmussen JJ. Severity of exercise-induced bronchoconstriction during compressed-air breathing via scuba. *SPUMS J*. 2004; 34: 178-82.)
Introduction: Cool, dry air is a potent stimulus for bronchoconstriction in those individuals susceptible. Thus, diving with compressed air potentially increases the risk of airway narrowing in those with exercise-induced bronchoconstriction (EIB). However, this has not been investigated.
Objective: The purpose of this study was to determine the influence of breathing compressed air via self-contained underwater breathing apparatus (scuba) regulators while exercising on the severity of EIB.
Methods: Ten non-asthmatic controls and ten subjects with diagnosed EIB volunteered. Pre- and post-exercise pulmonary function was measured by spirometry. Subjects performed a treadmill run for five minutes at approximately 80% of their age-predicted maximal heart rate. On one occasion they breathed ambient air and on another they breathed compressed air via scuba regulators. Oral breathing was forced via the use of a nose clip. Forced expiratory volume in one second (FEV_1) was used to determine changes in pulmonary airway function.
Results: Both groups had normal pulmonary function prior to the exercise. Control subjects had no change in pulmonary function after exercise on either occasion. The EIB subjects demonstrated post-exercise reductions in FEV_1 of -15.1 $\pm$ 5.3% (mean $\pm$ SD) at 1 min, -15.1 $\pm$ 1.7% at 5 min, and -13.9 $\pm$ 1.4% at 15 min post exercise during ambient-air breathing. With compressed-air breathing the reductions in FEV_1 further increased to -27.0 $\pm$ 6.0% (P = 0.0002) at 1 min, -24.1 $\pm$ 2.4% (P = 0.0001) at 5 min, and to -20.5 $\pm$ 1.3% (P = 0.0001) at 15 min.
Conclusions: This study demonstrated that compressed-air breathing via scuba regulators increased the severity of EIB in EIB subjects. The results have implications for those individuals with EIB wishing to dive.

Introduction

Exercise-induced bronchoconstriction (EIB), or exercise-induced asthma (EIA), refers to airways narrowing associated with vigorous exercise. Up to 90% of those with chronic asthma and 10 to 50% of athletes demonstrate significant airways narrowing, EIB, triggered by six to eight minutes of strenuous exercise.[1,2] Post-exercise reductions of the forced expiratory volume in one second (FEV_1) vary depending on the severity of the EIB.[3] While the mechanism by which exercise triggers airways narrowing is debatable, all agree that breathing cold, dry air while exercising results in the most severe EIB.[4,5] Anderson et al recently published a diagnostic exercise protocol that uses cycling exercise while breathing cool, dry air from a compressed-air tank in order to optimally provoke the airways.[3]

Certain activities, including scuba diving, force the individual to breathe cold, dry air. Scuba (self-contained underwater breathing apparatus) diving requires breathing from a tank of compressed gas, thus producing inspired air characterised as cold and dry. For compressed-air diving, a standard air tank contains approximately 21% oxygen and 79% nitrogen. In the United States, most scuba tanks have pressures of 3000 psi, some up to 6000 psi. A first-stage regulator attached to the tank valve reduces the delivered pressure to approximately 150 psi. This pressure is delivered to the demand regulator incorporated into the scuba mouthpiece, delivering inspired gas at ambient pressure. The reduction in pressure at the first-stage regulator causes the gas to greatly expand (adiabatic expansion) which, in turn, reduces the temperature of the inspired gas as the gas flows through the valves: the Joule-Thompson effect. The higher the tank pressure is, the greater the pressure drop, and the larger the reduction in temperature of the delivered air. Temperature falls of 10° Celsius are common. In addition to being cool, the air delivered is very dry: approximately 0.1% relative humidity.[6]

Despite the fact that scuba divers breathe cold, dry air, there has been no investigation of the potential increase in severity of EIB in those breathing via scuba regulators and tanks. Because of the perceived increase in risk for diving in individuals with asthma and/or EIB, diving is generally contraindicated in those with these conditions.[7–10] Thus, the purpose of this study was to evaluate the potential effect

of compressed-air breathing via scuba on the severity of EIB. We hypothesised that those with EIB would demonstrate greater reductions in post-exercise pulmonary function while breathing compressed air via scuba than when breathing room air. In order to separate the single effect of breathing compressed air from the potential influence of other components of diving such as hyperbaria and/or hyperoxia on pulmonary function, all testing took place on land in the laboratory, as opposed to while diving.

Methods

This study was approved by the Colorado State University Human Research Committee. Each research subject completed a health and physical activity history questionnaire and signed an informed consent prior to participation. Twenty subjects volunteered for this study. Subjects were recruited by the investigators on the campus and in the surrounding community through posted announcements and word of mouth. Ten subjects were non-asthmatic (control group). Ten subjects had been diagnosed by their personal physician as having EIB at least one year prior to the study (EIB group). All EIB subjects were confirmed positive with EIB by a treadmill exercise test and demonstrated a minimum of a 10% decrease in FEV_1 post exercise. Control subjects were confirmed negative for EIB by the same exercise protocol and criteria. All subjects were non-smokers and recreationally active, exercising most days of the week for at least 30 minutes a day. EIB subjects were not on steroid medications, but used a physician-prescribed inhaler with a short-acting beta-2 adrenergic agonist, typically used prior to their normal exercise bout.

PROTOCOL

Resting pulmonary function was determined before exercise and again at 1, 5 and 15 min post exercise. Treadmill running was used as the exercise stimulus. Subjects completed two different treadmill tests, three to five days apart. Subjects were alternately assigned to the normal (ambient air) breathing or scuba breathing (compressed air) first, and then they switched for the second test day.

Exercise was performed on a treadmill for eight minutes.[11] Subjects chose a comfortable running speed that was maintained throughout. The treadmill grade was increased as required during the first three minutes of the exercise to increase subjects' heart rate to 85–90% of their age-predicted maximum.[11] The subjects then ran at this intensity for the remaining five minutes. For the normal-breathing test day, subjects wore a nose clip to force mouth breathing and inspired room air. Ambient conditions were 22.2° C and 50% relative humidity. For the scuba-breathing day, subjects wore a nose clip and breathed from a tank of compressed air (3000 psi) through a first-stage regulator (150 psi) via a demand second-stage regulator at the mouth (SCUBAPRO™ MK2 Plus first stage, SCUBAPRO™ R190 second stage; El Cajon, CA). Air delivered at the mouth by the scuba regulator averaged 18.1° C and 0% relative humidity. Heart rate was determined with a Polar™ heart-rate monitor, and oxygen saturation was monitored for safety with a pulse-oximeter on the ear (Ohmeda, Louisville, CO). All testing occurred on the campus of Colorado State University during the months of September and October. The testing site is at an altitude of 1,519 metres (4,984 feet).

MEASUREMENTS

Spirometry (Vmax 22™, SensorMedics, Loma Linda, CA) was performed using forced vital capacity (FVC). Subjects were required to perform three acceptable spirograms

Table 1
Subject characteristics and resting pulmonary function prior to each treatment (ambient air, compressed air) in control and EIB subjects. Values are mean ± standard deviation. Age-predicted normal pulmonary function values are in parentheses.[20] No significant differences between groups or treatments. Statistical P values for pulmonary function for corresponding ambient-air pulmonary function comparisons between control and EIB subjects were: 0.88, 0.08, and 0.98, respectively. (BMI – body mass index; FVC – forced vital capacity; PEF – peak expiratory flow; FEV_1 – forced expiratory volume in one second)

	Control			Exercise-induced bronchoconstriction		
Age	23.6 ± 1.0			24.4 ± 6.6		
Gender (m,f)	6,4			4,6		
Height (m)	1.76 ± 0.13			1.68 ± 0.07		
Weight (kg)	75.6 ± 18.3			70.4 ± 13.3		
BMI (kg/m^2)	24.2 ± 3.4			24.8 ± 3.4		
	Ambient air		**Compressed air**	**Ambient air**		**Compressed air**
FVC (l)	5.4 ± 1.6	(5.39)	5.4 ± 1.6	4.4 ± 0.6	(4.40)	4.1 ± 0.7
PEF ($l.sec^{-1}$)	9.7 ± 2.6		10.4 ± 3.8	7.8 ± 1.9		7.7 ± 1.4
FEV_1 (l)	4.55 ± 1.32	(4.52)	4.6 ± 1.35	3.76 ± 0.56	(3.79)	3.65 ± 0.84

Table 2
Post-exercise forced expiratory volume in one second (l) in control and EIB subjects. Values are mean ± standard deviation. No significant differences for control subjects. All compressed-air breathing via scuba values for the EIB group were significantly different to the corresponding ambient-air values: * P = 0.003, ** P = 0.023, *** P = 0.043

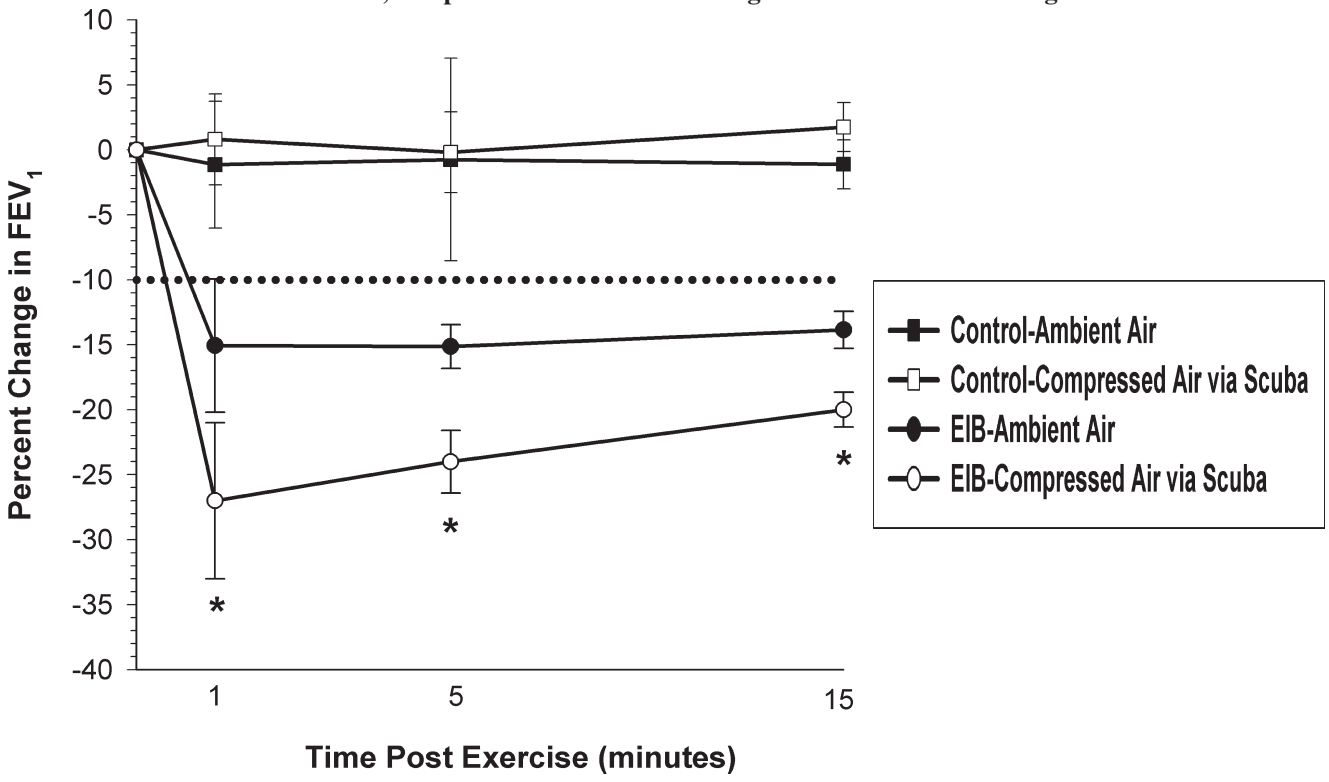
	Control		Exercise-induced bronchoconstriction	
	Ambient air	Compressed air	Ambient air	Compressed air
1 min	4.54 ± 1.43	4.66 ± 1.42	3.19 ± 0.43	2.66 ± 0.21*
5 min	4.53 ± 1.34	4.60 ± 1.36	3.19 ± 0.53	2.77 ± 0.38**
15 min	4.50 ± 1.29	4.69 ± 1.39	3.25 ± 0.49	2.92 ± 0.62***

according to the American Thoracic Society Standardization of Spirometry.[12] FVC, peak expiratory flow rate (PEF), and forced expiratory volume in one second (FEV_1) were the principal variables to monitor pulmonary function. All spirograms were measured and calculations were performed electronically by the Vmax 22™ Spirometer. Normal values for pulmonary function were predicted as per Crapo et al.[13] FEV_1 was used principally to reflect airway narrowing. A 10% or greater decrease in FEV_1 post exercise compared with pre-exercise values was indicative of EIB.[14]

DATA ANALYSIS

The sample size of ten was determined based on the standard deviation of the percentage change in FEV_1 pre to post exercise, and the ability to detect a difference of two percentage units between two means, with a power of 95% and P = 0.05.[15] Data are expressed as means and standard deviations. Post-exercise pulmonary function is expressed as a percentage change from pre-exercise pulmonary function values. Subject characteristics were compared using an unpaired *t*-test. Pulmonary function across the four conditions was analysed with a two-factor (treatment, group), repeated-measures (treatment) ANOVA. Post-exercise FEV_1 was analysed with a two-factor (treatment, time), repeated-measures (treatment, time) for each group. When a significant *F*-ratio occurred for a main effect, a Bonferroni's post-hoc test for multiple comparisons was performed. Significance for a two-tailed test was set *a priori* at P <0.05.

Figure 1
Percentage change in forced expiratory volume in one second (FEV_1) from pre to post exercise in non-asthmatic control subjects and exercise-induced asthmatic (EIB) subjects when breathing either ambient or compressed air via scuba. Dotted line indicates a 10% decrement in FEV_1, indicative of abnormal pulmonary function. *P <0.05, compressed-air scuba breathing vs. ambient-air breathing



Results

Subject characteristics are presented in Table 1. As shown, there were no significant differences in subject characteristics between the two groups. Baseline, pre-exercise pulmonary function was normal and did not differ between the two groups or between the two visits to the laboratory (Table 1). Post-exercise FEV_1 was not altered by compressed-air breathing via scuba in control subjects (Table 2). In contrast, for EIB subjects, all post-exercise values for FEV_1 were reduced further by compressed-air breathing via scuba (Table 2).

The percentage change (pre to post exercise) in FEV_1 was the principal diagnostic measure of EIB and is presented in Figure 1. Figure 1 indicates that non-asthmatic control subjects had no decrement in pulmonary function with regard to airways narrowing post exercise when breathing ambient air. For the three post-exercise time periods (1, 5, and 15 min) the change in FEV_1 was, respectively, -1.1 $\pm$ 4.8%, -0.75 $\pm$ 7.8%, and -1.12 $\pm$ 1.9%. Additionally, post-exercise FEV_1 was not affected by compressed-air breathing via scuba in control subjects: 0.85 $\pm$ 3.5% (P = 0.31), -0.19 $\pm$ 3.1% (P = 0.83), and 1.75 $\pm$ 1.9% (P = 0.15).

In contrast, subjects with EIB had marked reductions in FEV_1 post exercise while breathing ambient air, exceeding the diagnostic value of 10% reduction compared with pre exercise (Figure 1). This post-exercise reduction in FEV_1 was significantly exacerbated by compressed-air breathing via scuba. At 1 min post exercise, FEV_1 decreased from -15.1 $\pm$ 5.3% during ambient-air breathing to -27.0 $\pm$ 6.0% (P = 0.0002) with compressed-air breathing via scuba; at 5 min this change was from -15.1 $\pm$ 1.7% to -24.1 $\pm$ 2.4% (P = 0.0001); and at 15 min it was from -13.9 $\pm$ 1.4% to -20.5 $\pm$ 1.3% (P = 0.0001).

Discussion

This study shows for the first time that breathing compressed air through scuba regulators worsens post-exercise pulmonary function in those with EIB, while having no measurable effect on those without EIB. FEV_1 is an indicator of airway calibre and characteristically is decreased in those with EIB. Compressed-air breathing via scuba caused a significantly greater decrease in FEV_1 in those with EIB when compared with the same subjects breathing ambient air. Thus, severity of EIB was increased when exercising while breathing compressed air via scuba.

In general, exercise hyperpnoea while breathing cold, dry air is the most potent stimulus for airway obstruction or EIB.[16] For example, Mcfadden et al recently demonstrated that cold, dry air was a more potent stimulus for EIB than ambient air when ventilation was voluntarily increased in those susceptible.[5] In their study, voluntary isocapnic hyperpnoea of cold, dry air by EIB subjects exacerbated the percentage drop in post-exercise FEV_1, from a 16% reduction to approximately a 30% reduction in post-hyperpnoea FEV_1. Thus, in the present study, the cold, dry air from the compressed-air tank was likely the stimulus for the exacerbation of the EIB observed in these subjects.

Boutet et al suggested previously that scuba diving with a compressed-gas cylinder would be likely to exacerbate EIB in susceptible individuals.[17] The results of the present study support this hypothesis. They further proposed that divers with EIB would have airways narrowing associated with the hyperpnoea, and that potentially the airways narrowing/obstruction could be hazardous upon ascent. Upon ascent, gas volumes increase and complete expiration is required to prevent barotrauma. With airways obstruction, the danger of trapped gas and barotrauma would be greater. Recently, Leddy et al evaluated the possibility of air trapping and barotrauma in EIB subjects exercising while immersed in water.[18] They demonstrated a fall in FEV_1 and other lung volumes in their subjects, and some of the data suggested that airways narrowing may be increased due to the immersion. This potentially could lead to air trapping.

Current epidemiological data are insufficient to determine the risk of diving for asthmatics. There is a consensus, however, that asthmatic subjects with acute bronchoconstriction and individuals with exercise-induced bronchoconstriction should exercise caution before considering diving.[9,10] Asthmatic divers should consult their physician and may want to read Tetzlaff et al for a better review of the potential risks of diving.[9] The diver's level of understanding of the risks of diving contributes to making diving more safe. The more recent guidelines for asthmatics who wish to dive published by the British Thoracic Society state that "*asthmatic individuals who are currently well controlled and have normal pulmonary function tests may dive if they have a negative exercise test*."[19] Thus, the results of the present study emphasise the importance of this statement.

The sample size in the current study is small. However, this design was based on a power analysis, and the results are statistically supported. Subjects were pre-assigned to ambient or scuba as the first treatment. Though this was not a random design, for small sample studies a balanced assignment precludes the potential of a markedly unbalanced result of randomisation, with one treatment occurring first more frequently than the other. Furthermore, this study was conducted on land, so any influence of the hyperbaria associated with diving on these results has not been determined. Thus, some care must be used when generalising these results to the diving population.

The scuba apparatus likely added some resistance to breathing. It is not known if ventilation during scuba breathing was impaired and carbon dioxide retention occurred, potentially affecting bronchoconstriction. There was the possibility that minute ventilation differed between the air and the scuba conditions as scuba breathing occurred via the scuba apparatus and air breathing did not. Though ventilation was not determined during exercise, both control

subjects and those with EIB received the same treatment. Subjects did not remark about increased resistance to breathing or dyspnoea with scuba apparatus. The scuba regulator itself was adjusted to reduce the pressure differential required to initiate flow from the tank. Thus, the probability is low that there was a marked influence on the EIB subjects imparted by breathing via the scuba apparatus during exercise.

The exercise intensity used in this study is generally higher than the typical recreational dive. Recreational divers generally operate at low exercise intensities in order to conserve air, prolonging the dive time. However, EIB severity is determined both by the intensity and the duration of the exercise.[20] Concern would arise if an increased intensity of exercise were required, such as in an emergency. Occupational divers may be working at exercise intensities that can induce EIB. Therefore, the results of this study are relevant to divers and support the consensus that those with EIB should not dive without careful medical and self-monitoring of their condition.

In conclusion, exercise while breathing compressed air via scuba regulators induced an additional decrement in pulmonary function in those with EIB. Non-asthmatics had no significant changes in pulmonary function. Because the study was conducted on land, any specific effects of diving, such as hyperbaria and hypoxia, were eliminated. These results indicate that individuals with EIB should carefully consider their condition, become very well informed, and obtain medical clearance prior to considering scuba diving.

Acknowledgements

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A prospective field study of reverse dive profiles in UK female recreational divers

Marguerite St Leger Dowse, Alex Gunby, Richard Moncad, Caroline Fife and Philip Bryson

Key words
Reverse dive profiles, decompression sickness, diving, women

Abstract

(St Leger Dowse M, Gunby A, Moncad R, Fife C, Bryson P. A prospective field study of reverse dive profiles in UK female recreational divers. *SPUMS J*. 2004; 30: 183-8.)
Everyday diving habits of a large group of female recreational divers were observed for up to three years. Reverse dive profiles (RDPs) were compared with the conclusions of the Smithsonian Institution Reverse Dive Profiles Workshop. Volunteers did not know RDPs would be scrutinised, making changes to their diving habits unlikely. 570 participants returned 'diaries'; 62% for three consecutive years, reporting 30,480 dive days (16,706 multiple-dive days). The majority of dive depths ranged from 15 m to 89 m. In 29.7% of the multiple-dive days the second dives were of greater depth than the first dives, with 0.25% outside the Smithsonian conclusions for RDPs (depth differentials between the first and second dive of the day were >12 m, and the second dive was deeper than the first and deeper than 40 m). Rates of self-assessed symptom data of possible decompression sickness (DCS) were analysed by RDP with no significant correlation found (minimum P = 0.18). Maximum depth ever dived and total dives logged at the start of the study (surrogates for diving experience) were both significantly correlated with percentage of RDPs (P <0.0001 and P = 0.0008). There were significantly fewer RDPs for one dive training organisation (P = 0.0005). This work suggests future studies should consider carefully the type and amount of data necessary to address these issues, with power calculations demonstrating 30,000 to 180,000 multiple-dive days from 1,000 to 6,000 women needed for any significant effect (at the 5% level) to show. More complex physiologically based studies are possibly required.

Introduction

Within the recreational diving industry the tradition that it is safest to make the deepest dive first has evolved from interpretations of decompression modelling and historical custom.[1] The question of whether reverse dive profiles (RDPs) incur a higher risk of decompression sickness (DCS) than non-reverse dive profiles is the subject of frequent debate.[2] In a review of the literature it was found that there was no theoretical or experimental evidence to indicate a repetitive dive must be shallower than the dive that precedes it.[3] The exception was direct ascent from deep repetitive dives that have been shown to produce a high incidence of DCS.

Theoretical predictions have tended to 'suggest' safe limits with regard to 'no-decompression' dives for forward and reverse dive profiles.[4] Anecdotal observations indicate that within the recreational diving industry it is widely believed the practice of RDPs makes for an increased risk of DCS, and so are not always consistent with scientific and diving medicine literatures.[5,6] When asked to substantiate this belief, however, educators cannot always evidence the argument. Additionally, educators within the industry acknowledge that the practice of RDPs does take place but have no means of quantifying the activity.

In the Smithsonian Reverse Dive Profiles Workshop it was observed that the use of the physiological model serves to draw attention to the complexities of the problem and generates the need for clearer thinking regarding the evaluation of the risks involved.[7] Current assessment of the risks therefore cannot be regarded as hard science, and there is a clear need for additional studies and the gathering of more field data.[4,8] The Workshop concluded, "*We find no reason for the diving communities to prohibit reverse dive profiles for no-decompression dives less than 40 msw (130 fsw) and depth differentials less than 12 msw (40 fsw)*."

We have prospectively observed the everyday diving habits of a large group of female recreational divers over a prolonged period of time. The primary goal of the study being to observe respondent-reported scuba-diving problems and the menstrual cycle. As part of this study the prevalence of RDPs was recorded and compared with the Workshop conclusions. Respondent-reported self-assessed symptoms of possible DCS were also examined. This paper communicates the information gathered.

Methods

From 1997 female recreational divers had volunteered to keep diving 'diaries' for up to three consecutive years as part of a project designed to observe ordinary diving habits, respondent-reported scuba-diving problems, and the menstrual cycle. Publicity for the project was generated via United Kingdom (UK) dive clubs, dive shows and press releases in the diving journals. Volunteers did not know

that reverse-dive-profile data would be scrutinised and therefore made no changes to their diving habits as a result. Volunteers recorded basic dive information (maximum depth, total dive time, and if a mandatory decompression stop was added). Respondents were also given the option to record any possible signs and symptoms of DCS from a fixed option list (lower limb or joint pain, upper limb or joint pain, dizziness/disorientation, visual disturbance, inappropriate fatigue/weakness, difficulty in speaking, skin itching, tingling, skin rash, chest pain or breathlessness, partial paralysis, loss of sensation/numbness, problems with thinking, memory or performance). They were also asked to record any further information not included in the fixed options they felt appropriate. Additionally volunteers were asked if they had reported the possible symptoms of DCS to a diving physician, whether diagnosis was confirmed, and if so to detail their subsequent treatment.

Social and demographic data were gathered (age, weight, smoking and alcohol consumption) together with diving histories (training grade and affiliation, years diving, number of dives and maximum depth ever dived at the start of the study, use and type of dive computers and tables). Respondents were also asked if, in their opinion, they used dive computers/tables exactly as written, conservatively, or if they "took risks" by shaving times and depths.

All participants initially completed the comprehensive background questionnaire. From then on they completed and returned update questionnaires with monthly 'diary' charts, returning them on a six-monthly basis. Questionnaires and specifically designed charts were developed in conjunction with a psychologist. Volunteers were offered no incentive to participate and were free to leave the project at any time.

Data were categorised into multiple-dive day data and by whether or not the second dive of the day was deeper than the first dive of the day. When more than two dives were performed in a day only the first and second dives of the day were used for statistical analysis. Data were also categorised by whether or not they were within the Smithsonian Reverse Dive Profiles Workshop conclusions. We did not restrict the definition to no-decompression dives since there was insufficient granularity in the data to do this. We used the following interpretation of dives outside of the conclusions of the Workshop: the second dive is deeper than the first dive and deeper than 40 metres seawater (msw), with the depth differential between first and second dives greater than 12 msw. We used 30 msw as a marker (second dive is deeper than the first dive and is deeper than 30 msw) when analysing the relationship between experience (at the start of the study) and percentage of respondents doing RDPs (during the study), and training organisations and RDPs (during the study).

Trained operators entered all data, with data quality checks carried out against hard copy. All data were recorded anonymously.

STATISTICAL ANALYSIS

The dive days outside of the Smithsonian Workshop conclusions were analysed, together with untreated, self-assessed symptom data of possible DCS. Self-assessed symptoms were considered only if occurring after the second dive. For each method of categorising the data, the rate of untreated, self-assessed symptoms per 1,000 dive days is given. The self-assessed symptom rates were compared with the symptom rates for dive days within the Workshop conclusions using z-tests.

Power calculations were performed to estimate the size of study sample needed to be able to detect possible differences in self-assessed symptom rates between RDP and non-RDP dives. The calculations assumed that the proportion of RDP dives and the self-assessed symptom rate for non-RDP dives would both be as observed in this study. The calculations were performed for the cases where the self-assessed symptom rate for RDP dives was 33% of the observed value and 125% of the observed value, thus giving a range of sample sizes.

We analysed the relationship between the proportion of dives that were RDPs and diving experience. The proportions of RDP dives were arcsin-transformed before applying linear regression. The explanatory variable of experience was surrogated in two ways: as the number of dives logged at start of study; and as maximum depth ever dived at start of study. A linear regression was performed for each of the two explanatory variables.

Table 1
Profiles of dive days with numbers of dive days, dives, self-assessed symptoms of possible DCS, and rates of symptoms per 1000 dive days

Profile of dive day	Dives days	Dives	Symptoms	Rates
Outside Workshop conclusions	41	82	2	48.78
Inside Workshop conclusions	4,928	9,856	19	3.86
Second dive shallower than first dive	11,737	23,474	46	3.92
Only 1 dive in day	13,774	13,774	54	3.92
Total	30,480	47,186	121	3.97

Chi-square tests were used to examine the relationship between RDP frequency and training organisation.

Results

A total of 570 women returned data for a minimum of six months, with 62% continuing to return data for three consecutive years. At the start of the study the age range was 14 to 63 years (mean 35, SE 0.37).

DIVING BACKGROUND AT START OF STUDY

Prior to the study the number of years of diving experience per person ranged from 1 to 34 years, with the number of dives reported per person ranging from 1 to 3,000 (mean 208, SE 4.14), and a collective experience recorded of 117,919 dives. Also prior to the study 49% had dived to 40 m and deeper (1.9% >70 m) during their diving career, and had already recorded more than 100 dives each.

Fifty-eight per cent of women dived all the year round, though not always evenly throughout the months. Addition of extra stops over those demanded by the tables and computers was reported by 67%. Few women (1%) admitted to taking risks, with 60% reporting they dived conservatively, and 37% using tables and computers exactly as written; 2% declined to give this information.

Table 2
Reported first and second dives from the 41 dive days outside Smithsonian Workshop conclusions

1st dive of day	2nd dive of day	1st dive of day	2nd dive of day
14	51	8	41
30	46	11	46
28	45	20	43
25	42	20	46
26	61	12	46
22	42	30	58
8	41	47	68
28	46	28	40
15	44	28	40
6	48	23	52
32	48	36	58
25	40	22	43
11	51	6	42
10	46	25	45
10	42	21	58
22	61	26	40
33	59	15	42
34	60	41	56
15	40	35	50
13	53	30	57
		17	55

Figure 1
Relationship between RDPs during the study and maximum depth (msw) ever dived at start of study

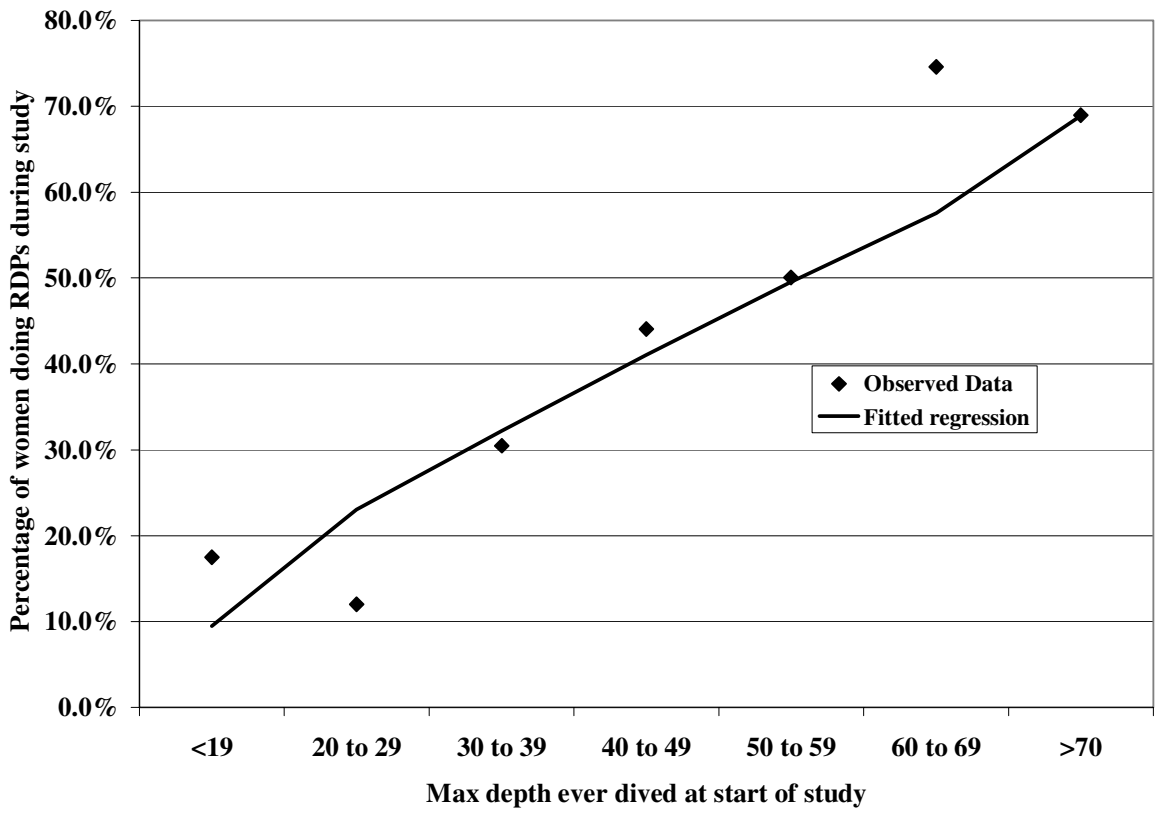
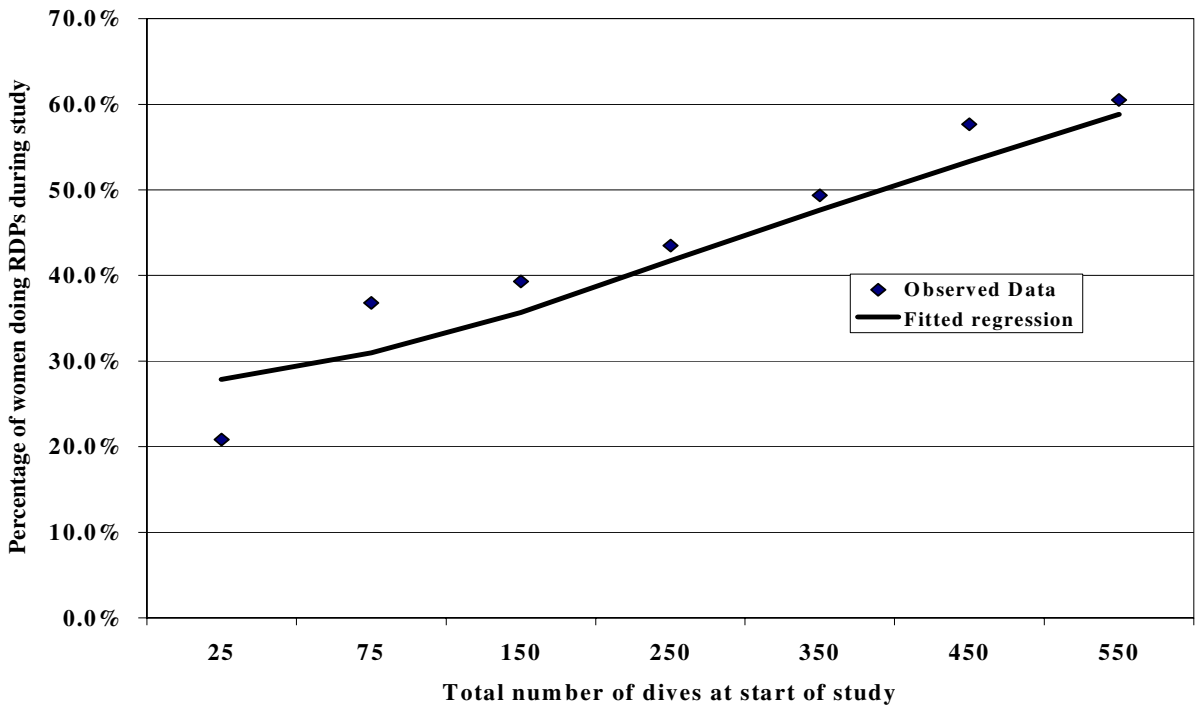


Figure 2
Relationship between RDPs during the study and total number of dives recorded at start of study



DIVES RECORDED DURING THE PERIOD OF THE STUDY

During the period of the study the women recorded a total of 50,261 dives (including third dives of the day) from 30,480 dive days. Also during the study 44% dived to 40 m or more, with a 174 m mixed gas dive being the deepest dive recorded; this depth was verified with the subject concerned. With the exception of this dive, taken overall, the deepest first, second and third dives of any day were 89 m, 68 m and 57 m respectively, performed by different respondents recorded on separate days. Eighty-four per cent of women added mandatory decompression stops to 31% of the dives. The 62% of women who had returned diaries for three consecutive years accounted for 83% of the dives and averaged 40 dives each per year.

REVERSE DIVE PROFILE DIVES

There were 16,706 multiple-dive days recorded (Table 1). Of these there were 4,969 days (29.7%) with second dives of the day to a depth greater than or equal to that of the first dive of the day. Only 41 (0.25%) dive days fell outside the Smithsonian conclusions. There were 3,074 multiple-dive days (18.40%) with three dives recorded.

Untreated, self-assessed symptom rates are far higher for the dive days outside our interpretation of the Smithsonian conclusions than all other dive-day profiles, but these differences are not statistically significant (minimum P = 0.18). Two women reported untreated, self-assessed symptoms of possible DCS outside the Smithsonian conclusions. The first woman reported itching, tingling, and "strange sensations in the left part of the body", with the first dive to 47 m and the second dive to 68 m. The second woman reported skin rash and itching right shoulder, with the first dive to 28 m followed by a dive to 45 m. First and second dives from the dive days outside the Workshop conclusions are shown in Table 2. There were no treated cases of DCS in this study.

REVERSE DIVE PROFILES, AND EXPERIENCE AT THE START OF THE STUDY

We used the maximum depth ever dived, and the total number of dives reported by each respondent at the start of the study as surrogates for experience. The percentage of respondents performing RDP dives during the study (as defined by the second dive being deeper than the first dive and being deeper than 30 m) is shown in Figures 1 and 2. The correlation between percentage of respondents doing RDP dives during the study and each surrogate for experience at the start of the study is significant (linear regression of arcsin percentage RDP dives against maximum depth has a P-value of <0.0001 and against number of dives has a P-value of 0.0008).

RDPs and training organisations

When RDPs (as defined by the second dive being deeper than the first dive and being deeper than 30 m) were analysed by training organisation, there were significantly

fewer RDP dives done by members of one particular organisation (P = 0.0005) when compared with all others.

Discussion

Our data give a unique insight into female-specific, everyday recreational diving, documenting more than 50,000 dives from 570 women over three years. The women were from a wide range of experience levels and from across different training organisations. Attrition was evenly distributed over the time of the study, with 10.7% in the second six months of the study falling to 8.2% at 30 months. In the first eighteen months of the study 12% of women changed address, with 8% in that group moving more than once, emphasising the problems of tracking a large group of the population over a prolonged period of time. Where possible, reasons for ceasing to participate were established, and these ranged from changes in marital status (7% married during the course of the study) to not wanting to commit further time to the study.

The women kept normal diving records for long periods of time, 62% for up to three years, and so it is reasonable to conclude that they did not conduct their diving in line with any preconceived criteria or restraints. The prevalence of RDP dives, in one form or another, has been demonstrated by these study data and therefore may lend support to anecdotal observations that RDPs are indeed regularly taking place. Around 30% of multiple-dive days within the study population involved some combination of RDP, even though only 0.25% of multiple-dive days fall outside our interpretation of the Smithsonian Reverse Dive Profiles Workshop conclusions (Table 1). Studies have shown women may dive with more caution than men,[9] and therefore it is likely that a greater number of RDPs are taking place amongst male recreational divers.

Previous studies have implied that there are a large number of divers who never report their symptoms of possible DCS to a physician, and our data might be considered to support those studies.[9–11] Untreated, self-assessed symptoms of possible DCS in this study were not reported to a physician. In addition to those described outside the Smithsonian conclusions (Table 1) other unreported symptoms of possible DCS within the Smithsonian conclusions included skin rash, visual disturbance, loss of sensation, and inappropriate fatigue. When analysing the symptom rates in relation to dive days outside the Smithsonian conclusions, no significant effect was discernable even though the observed rate is far higher than for other dive-day profiles. This can be explained by the tiny number of dive days seen outside the Smithsonian conclusions, which makes the estimated symptom rate for that category subject to a large standard error.

This study was not specifically designed to observe the incidence of RDP dives and any definitive association with possible signs and symptoms of DCS. If there truly is an underlying correlation, power calculations based on our study data indicate that we would need to observe in the order of 30,000 to 180,000 multiple-dive days (giving 60,000 to 400,000 dives) in order to detect a significant effect at the 5% level. This translates to between 1,000 and 6,000 women in total taking part in the study.

When attempting to analyse any correlation between RDPs and experience, we used the 30 m marker (second dive is deeper than first dive and deeper than 30 m) given that diving to depths over 30 m has been traditionally defined as 'deep' by some training organisations. Additionally, 30 m is the depth limit beyond which many dive operators will not take divers, particularly in holiday destinations. The diving practices of the study group outline a possible need for training agencies to promote a clearer policy message with regard to RDPs. The exact policies and type of reverse-dive-profile recommendations by training organisations are not always clear to instructors and the grass-roots diver. The significantly lower number of RDP dives performed by one particular training agency is evidence of this.

The use of dive computers, which has largely overtaken the use of traditional dive tables, could also be a contributory factor in clouding the issue and understanding of RDPs, with divers relying on the culture of dependence and trust in technology. Within the study group as a whole more than 50% used computers only or computers in conjunction with dive tables. In the group where the second dive of the day was greater than the first dive and also greater than 40 metres, 73% of women used computers. The clear link between performing RDP dives and the experience of the diver may, on the other hand, indicate that initially divers pay heed to advice given during training. Subsequently divers become either more empirical in their approach, or more distant from the training, and so develop their own ideas about what is and is not safe.

The concern surrounding the risks associated with RDP dives and DCS, which may or may not exist, is ongoing and generates controversy. It is beyond the scope of this study to analyse or discuss a definitive relationship between RDPs and DCS. Problems identified with field data and self-assessment symptomatology have been reported.[12] Although in our study the more aggressive RDP categories were associated with a higher rate of self-assessed symptoms of possible DCS per thousand dive days, these differences were not statistically significant. The availability to date of so few field data on which to draw any understanding has allowed much of the debate and recommendations of safe limits to be substantiated by statistical modelling, with some conclusions drawn from hyperbaric chamber records and limited field data.[8,13] Our study data provide a 'real-world' insight into the everyday diving practices of a large population of female divers over a long period of time, and should therefore be useful in formulating more definitive studies, and as an indicator to teams collecting field data in the future.

Conclusions

The question of whether RDPs are 'safe' is complex. Our data are not entirely conclusive despite the large numbers and the time scale involved. However, this work indicates to other bodies and organisations wanting to take this work further the problems associated with this type of study. For example, to answer this question many more participants would need to be studied over a longer period of time. Additionally, a study would need to take into account such factors as biological variables and the anomalies of self-assessed symptoms.[12] Any future study would need to consider carefully the type and amount of data required to show any possible significant effect. It may be that these questions cannot be answered by a field-study approach and will require a more complex physiologically based study.

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<www.hboevidence.com>

Decompression practice and health outcome during a technical diving project

David J Doolette

Key words
Technical diving, cave diving, safety, health surveillance, decompression, air, mixed gas, trimix, nitrox, oxygen

Abstract

(Doolette DJ. Decompression practice and health outcome during a technical diving project. *SPUMS J*. 2004; 34: 189-95. **Paper presented at the SPUMS ASM in Noumea, 2004**)
Technical divers use multiple helium/nitrogen/oxygen breathing-gas mixtures to reach depths greater than 40 metres seawater (500 kPa) using scuba. Self-assessment of health outcome by 9 divers using a validated decompression health survey followed a series of 200 technical dives to a maximum of 123 metres of fresh water. Decompression was planned using the ZH-L16 calculation procedure. Although the incidence of treated decompression sickness was only 0.1%-3.4% (95% confidence limits), high health survey scores, possibly marginal symptoms of decompression sickness, were associated with maximum diving depth greater than 90 metres.

Introduction

The practical range of 'recreational' diving is limited by the use of a single-cylinder, nitrogen/oxygen, open-circuit scuba to a maximum depth of 40 metres seawater (msw) (500 kPa) with minimal decompression. 'Technical' diving, although typically conducted for recreational purposes, is scuba diving beyond the practical range of recreational diving. Although technical diving encompasses many types of diving activities, technical diving in the format that is presently common arose from underwater cave exploration in Europe and USA in the 1980s.[1,2] Principally, technical divers use multiple helium/nitrogen/oxygen breathing-gas mixtures to reach depths greater than 40 msw and to accelerate decompression. With the advent of instructional programmes for this style of diving there is a growing 'mainstream' of technical divers conducting brief dives to 100 msw. A smaller core of technical divers is pioneering dives beyond this range.

A principal challenge for technical divers is uncertainty in the safety of decompression procedures. In the past only the military and commercial diving communities have had the resources for large-scale development and validation of decompression schedules, and even where resultant decompression schedules are available they are designed for diving procedures quite different to technical diving. Military and commercial deep-diving procedures include use of heliox mixtures, tethered divers, diving bells, and saturation techniques, whereas many technical dives are helium/nitrogen/oxygen trimix bounce scuba dives.

Technical diving decompression procedures are commonly wholly or partly based on the ZH-L16 decompression calculation method of Bühlmann.[3,4] Although not developed for technical diving there are several reasons for the popularity of this method. Unlike many decompression procedures a full description of the ZH-L16 (or its ZH-L12 predecessor) calculation method was freely available in the mainstream scientific literature at the outset of modern technical diving. The ZH-L16 is a decompression model rather than a set of schedules generated by a model and therefore can be used to calculate decompression requirements for helium/nitrogen/oxygen dives of any complexity. The ZH-L16 calculation method is simple and user-controllable implementations have been developed for microcomputers, palm tops, and mobile phones and it is commonly programmed into decompression computers carried by divers.

Although there are many anecdotes about the safety or otherwise of the ZH-L16 calculation method and its variants, mainly in Internet forums, there does not appear to be any formal evaluation of the ZH-L16 in the specific context of technical diving. This manuscript examines the decompression health outcome following technical dives using the ZH-L16 decompression calculation method during a technical diving project to explore and map an underwater cave called 'The Shaft'.

Diving

The Shaft is a freshwater cenote near Allendale East in South Australia named for the shaft of sunlight that at times shines through the 1 m wide entrance and tracks across the debris cone below. A brief history of diving in this site has been published.[5] The site has been dived since the 1960s and the first mapping project, using air diving, was undertaken in the early 1980s. The diving since then has been limited to 40 metres fresh water depth (mfw; 494 kPa) at the request of the landowners. After obtaining permission for some preliminary exploratory deep technical dives in

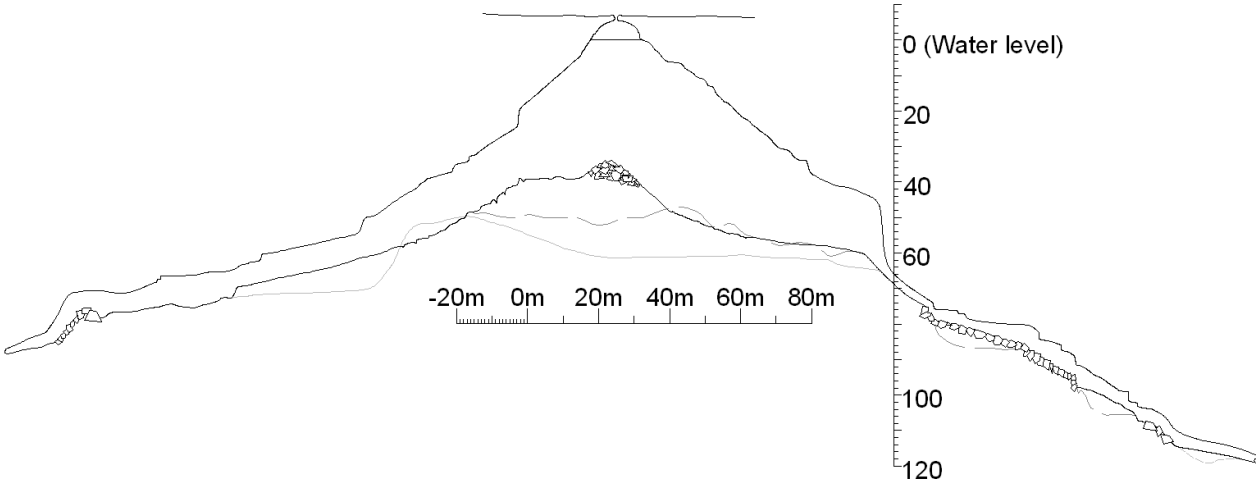
the early 1990s a group of divers obtained permission to undertake an extensive exploration and mapping project of The Shaft deeper than 40 mfw under the auspices of the Australian Speleological Federation. This project, which is the subject of the present report, was conducted between October 2000 and January 2004.

Aspects of the earlier map were verified but the majority of the mapping occurred at depths greater than 40 mfw. The survey used knotted line, compass, depth gauge, and clinometer. Survey lines knotted every 5 m were installed throughout the cave in locations chosen to capture the shape of the cave. Each change in direction of the line was designated a survey station and at each station divers recorded the depth of water, distance to walls, floor and roof, the bearing, and the distance to the next station. The inclination along a few steeply sloping line segments was also measured. On a subsequent dive, using the line survey as reference, the significant features of the cave at and between stations were sketched. Eleven divers conducted 225 air and trimix dives during the exploration and survey of this site. The resulting map is a 1:500 scale plan and extended elevation (Australia, The Shaft Cave [speleological map]. Payne T, cartographer. Adelaide, Australia: Australian Speleological Federation - Cave Diving Group; 2004. 1 sheet: black & white, 69x98cm, scale 1:500, ASF grade 33A. Located at: Cave Exploration Group of South Australia archive, Adelaide, Australia; map 1322). A miniature of the extended elevation is shown in Figure 1.

Typically, teams of two or three divers assigned specific mapping or exploration tasks dived once per day. Divers dressed in drysuits with passive insulation provided by air inflation and undergarments; water temperature was 15°C to 16°C. Primary lighting was provided by hand-held halogen or arc lamps with waist-mounted battery canisters. Depth and time were monitored using Uwatec AladinPro™ or Suunto Vyper™ decompression computers. The breathing-gas supply for the deepest portion of the dives was carried in twin 12 l or 15 l 23.2 MPa working pressure steel cylinders; these were back-mounted along with buoyancy wings. To provide redundant scuba, back-mounted cylinders were equipped with dual open-circuit regulators either independently or via a duel outlet manifold. If additional breathing gas and/or different breathing-gas mixtures were required to travel to the maximum distance or depth, these were breathed from separate cylinders with single, open-circuit regulators attached to the divers' left side. These cylinders would be detached and left on the cave floor (staged) either at a planned cylinder pressure or when the maximum operating depth (determined by P_IO_2 or P_IN_2) for that gas was reached. Staged cylinders were retrieved and breathed upon return. Gases breathed only during decompression were contained in separate cylinders left attached to shot-lines from the surface at the appropriate depths. Each cylinder was clearly labelled with its maximum operating depth.

Dives planned at less than 45 mfw were conducted entirely breathing air. For deeper dives air was breathed to approximately 45 mfw, then helium/nitrogen/oxygen trimix was breathed deeper than 45 mfw to reduce the work of breathing, reduce the level of nitrogen narcosis, and reduce the risk of oxygen toxicity. Trimix was produced by mixing helium and air for an equivalent air narcotic depth – [[D/10.33+1]*[1-FHe]-1]*10.33 – of 46 mfw (P_IN_2 = 436 kPa at the planned maximum actual depth, D), although mapping was generally conducted shallower. Trimix was produced by partial pressure mixing of helium and air, and nitrogen/oxygen mixtures by partial pressure mixing of oxygen and air in the cylinders used by the divers. The mixtures were analysed for oxygen content by galvanic and polarographic methods at the time of mixing and all breathing gases were analysed for oxygen content

Figure 1
Extended elevation of 'The Shaft' reduced from the original (Australia, The Shaft Cave [speleological map]. Payne T, cartographer. Adelaide, Australia: Australian Speleological Federation - Cave Diving Group; 2004. 1 sheet: black & white, 69x98cm, scale 1:500, ASF grade 33A. Located at: Cave Exploration Group of South Australia archive, Adelaide, Australia; map 1322) with permission of the cartographer.



immediately prior to use. The commonly used helium fractions were 20%, 30%, 40%, 50%, and 60% for maximum depths of 60, 70, 80, 100, and 120 mfw, resulting in equivalent air narcotic depths of 46, 46, 44, 45, and 42 mfw, respectively. For dives deeper than 100 mfw an intermediate helium-air mixture was breathed between 45 and 70 or 80 mfw. Decompression was accelerated by switching from trimix to air at either 45 mfw or 55 mfw, then to a 50% nitrogen-50% oxygen mixture at 21 mfw, then to 100% oxygen at 6 mfw.

Decompression calculations

Underlying the ZH-L16 calculation method is a mammillary model of helium and nitrogen tissue uptake and washout comprising 16 compartments in communication with a central pool equivalent to arterial blood or alveolar gas. For each compartment, different half-times describe the rate of exchange of nitrogen (4-635 minutes) and helium (1.5-240 minutes) with the central pool. The helium and nitrogen half-times are related by the inverse of the square root of the molecular weight of the two gases according to Graham's law for auto-diffusion of gases, implying diffusion of gas between the compartments and central pool. Decompression is controlled by the maximum of the set of minimum tolerated ambient pressures (P_{amb_tol}) for the compartments:

$$\max\left\{P_{amb\_tol_i} = \left(P_i - a_i\right) \times b_i\right\}$$

where, for the i^{th} compartment, P is the inert gas tension before ascent and a and b are constants. Various ZH-L16 implementations modify the set of a and b constants in a more or less *ad hoc* manner to modify the decompression in line with prevailing folklore; however, the present project used the unmodified ZH-L16 a and b constants as published.[4] Since this original publication a unique set of a and b constants for helium has been promulgated that allows a more accelerated decompression; this was not used.

Decompression calculations for trimix dives were performed using Excel (Version 9.0. Redmond, WA, USA: Microsoft Corp; 1999). Schedules that defined a multi-level bottom time and all decompression stops were generated for specific dives. The ascent always included one-minute stops at each stage bottle collection depth and a three-minute swim across the main chamber at 40 mfw in addition to the decompression stops dictated by the ZH-L16 model. Figure 2 shows an example schedule. Air-diving decompression was not structured but instead followed the prescription of diver-carried decompression computers. Most air dives were conducted using Uwatec AladinPro™ computers, which use an 8-compartment version of the ZHL model; a very few air dives were conducted using Suunto Vyper™ computers which use a 9-compartment ZHL model variant where half-times are reduced for gas washout and a and b constants are reduced for repetitive diving. Divers tended to extend air-dive decompression time beyond that prescribed by the decompression computer.

Methods

DATA COLLECTION

The study was conducted in accordance with the *National statement on ethical conduct in research involving humans* (Commonwealth of Australia. Canberra: AusInfo; 1999) and is an analysis of the records originally devised, maintained, and used by the author to audit all diving and decompression procedures during the mapping project. Subsequently, informed written consent was sought from each diver to use these data for scientific publication. Two divers refused to give consent for reasons unrelated to their health outcome. Exclusion of these data did not alter the conclusions from the unpublished audit of all diving during the project. The consenting nine divers include the author. The centrally maintained records included a workbook of all steps in the mixing and analysis for oxygen content of breathing gases, all decompression schedules used, and paper dive logs. Divers completed a decompression health survey to measure decompression-related health status before commencing on the project and then again following each dive.

The decompression health survey has been described in detail elsewhere.[6] It is an inventory of nine standardised items and responses covering five symptoms of decompression sickness (paraesthesia, rash, balance, fatigue, and pain), five health-status indicators (vitality, pain, physical functioning, role limitation, and health perception), and time of onset of symptoms, plus one free response, each item scored from 0 to 3. The resulting summed decompression health score (DHS) ranges from 0 (well) to 30 and can be analysed as interval data. DHS are correlated with diagnosed decompression sickness (DCS)[6] and following routine occupational air diving the DHS increases with increase in decompression stress in the absence of diagnosed DCS.[7] The validated format of the decompression health survey and scoring instructions are available from the author.

In addition to paper records those divers using decompression computers capable of recording depth/time profiles submitted these profiles. These devices record gauge pressure (as depth of water) at fixed intervals of 10 or 20 seconds. Dive profiles were compared with the corresponding decompression schedules. The difference in area of the allowed time at depths deeper than 40 mfw and the recorded depth/time profile was calculated (area between the solid and dashed line in Figure 2) and divided by the time allowed to give a safety margin index in mfw. The ascent to first decompression stop was determined by visual inspection of a plot of the depth/time profile and the ascent rate in $mfw.min^{-1}$ extracted. A decompression index was calculated in the following manner. First, the depth/time profile was reduced to a smaller number of depth/time nodes by manual selection of nodes during visual inspection and the fraction of helium and nitrogen in the breathing gas at

each node assigned. Then the uptake and washout of helium and nitrogen into each of the 16 compartments defined by the ZH-L16 model was tracked for the duration of the dive and the time on the surface up until the diver health survey was completed (^{t}DHS). The decompression index was calculated as the sum across compartments of the time integral of positive values only of supersaturation scaled by ambient pressure (P_{amb}):

$$\sum_{i=1}^{n=16}\left(\int_{0}^{t_{DHS}} \frac{r\left(P_i + 0.19 - P_{amb}\right)}{P_{amb}}\right)$$

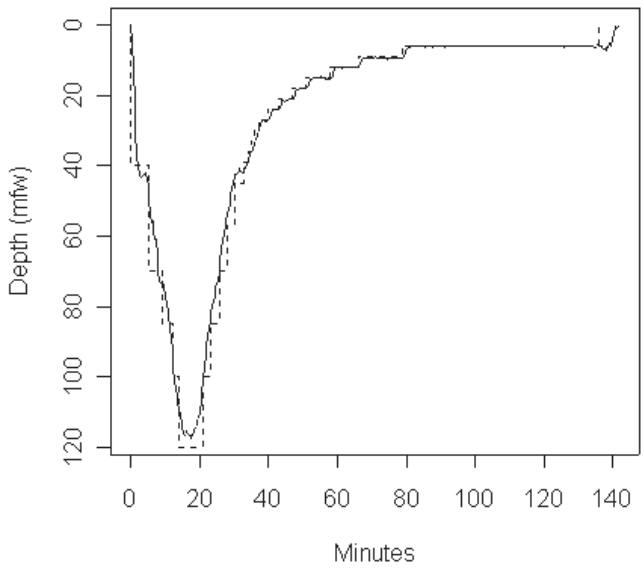
where 0.19 is the contribution of dissolved metabolic gases to the compartment and *r* is the ramp function. Although notionally an indicator of decompression severity, this decompression index is not validated against any measure of decompression stress. These calculations were implemented in GNU Fortran (g77 version GCC-2.95.2 for Mingw. The Free Software Foundation; 1999) and 'R' (R base package version 1.7.0. The R Development Core Team; 2003). For trimix dives, which were rigidly scheduled, if only one team member carried a recording device the other team members were assigned the same values of safety margin, ascent rate and decompression stress unless the dive plan or paper dive log indicated separation of the dive team at any point.

EVALUATION OF HEALTH OUTCOME

The DHS was used to measure decompression-related health status following technical diving and the contribution of the diving exposure was evaluated by linear regression. The full model investigated was of the form:

$DHS_{ij} = \beta_{0i} + e_i + \beta_1 DEPTH_{ij} + e_{ij}$

Figure 2
Dive profile recorded by a diver-carried computer (solid line) and the associated schedule (dashed line) of multi-level bottom times (deeper than 40 mfw) and decompression stops (shallower than 40 mfw)



which comprised the dependent variable DHS and fixed explanatory variable maximum depth (DEPTH in mfw). Bottom time was similar for all dives and total dive time was highly correlated with DEPTH so no indicator of dive duration could be usefully included in the model. In previous studies we have found that different subjects describe their normal health status differently,[7] and a preliminary analysis using factorial regression on divers suggested the same was true here. This is manifest as a different intercept for each diver (DHS at DEPTH = 0) in the linear model; to accommodate this the nine divers were considered a random sample from a population where the intercept (β_0) of the regression on the explanatory variables depends on the attendant. Subscript *i* denotes diver, subscript *j* denotes days, and *e* denotes error.

Parameters of the regression models were estimated by maximising the likelihood. The likelihood is the joint probability density function of the observed values of the dependent variable given the respective regression model. The full model was compared with the null model that includes only the intercept terms and where DHS only varies between divers. Significant difference ($p \leq 0.05$) between these nested models was evaluated by the likelihood ratio test, $2(LL_f - LL_r) \quad X^2_{f\text{-}r}$, where LL is the maximised log-likelihood of the model and f and r are the number of parameters in the full and null models respectively ($f > r$). For each model the data were examined for influential values (outliers with high leverage). Outliers were data with standardised residual more than two standard deviations from the mean. Leverage was taken as the diagonal of the hat matrix and values more than twice the mean were considered high.

There was insufficient number of full dive profiles collected to include safety margin, ascent rate, or decompression index in the linear modelling so only descriptive statistics of these variables are presented.

Decompression data were managed using an Access database (Version 9.0. Redmond, WA, USA: Microsoft Corp; 1999). All statistical calculations were performed using 'R' software base package (version 1.7.0. The R Development Core Team; 2003) and the non-linear mixed effect package (version 3.1-39. Pinheiro J, Bates D, DebRoy S, Sarkar D; 2003).

Results

Dive log or DHS was missing for seven dives; Table 1 shows the contribution of the nine divers to the remaining data set of 200 dives and their non-diving DHS from prior to commencing diving on the project. Divers dived once per day for a mean bottom time of 21 minutes (SD = 5, range 9-35), typically two days in succession (range 1-4 days). Dives ranged in depth from 35 mfw to 123 mfw, with total dive durations ranging from 18 to 179 minutes.

Table 1
Data summary for 9 divers for three types of dives; air, trimix <90 mfw depth and trimix >90mfw. DHS – decompression health survey score; median DHS (range); n – number of dives; NA – no data

Diver ID	Pre-diving	Air		Trimix <90 mfw		Trimix >90 mfw	
	DHS	DHS	n	DHS	n	DHS	n
1	2	0 (0-3)	5	0 (0-5)	20	5 (0-10)	9
2	0	0 (0-6)	6	0 (0-6)	24	1 (0-5)	10
3	7	5 (3-8)	11	5 (3-10)	22	7 (4-12)	7
4	1	2 (1-3)	7	2 (1-4)	15	3 (NA)	2
5	2	0 (0-2)	34	NA	0	NA	0
7	4	NA	0	2 (1-3)	4	7 (3-9)	3
9	1	2 (1-5)	6	NA	0	NA	0
10	1	0 (0-2)	5	NA	0	NA	0
11	5	3	1	3 (2-3)	6	NA	0

Depth/time profiles were available for 44 of the trimix dives and a sample profile along with the decompression schedule is shown in Figure 2. These trimix dives had a mean ascent rate of 5.5 mfw/min (SD = 1.2, range 3.4–7.6), a mean safety margin of 10.2 mfw (SD = 5.4, range 1.0–4.2), and a mean decompression index of 366 (SD = 186, range 47–796). Depth/time profiles were available for 6 of the air dives and showed a mean ascent rate of 5.2 mfw/min (SD = 2.2, range 3.6–8.6) and a mean decompression index of 1540 (SD = 276, range 1104–1956). These high values of the decompression index for air dives are a result of conducting only shallow decompression stops without breathing increased oxygen fraction.

Not accounting for individual variability, a DHS <6 can be considered acceptable for typical diving operations.[6] Figure 3 plots the maximum depth and total decompression time as open circles of all such uneventful dives. There were 12 health surveys that described both DHS = 6 (mean = 9, SD = 2, range 6–12) and occurrence of new symptoms during or following diving; these are plotted as filled circles on Figure 3. The common symptoms were pain (11/12), paraesthesia (9/12), and unusual fatigue (4/12). Pain was described as mild or moderate usually in the left elbow or shoulder and typically appeared during the 9- or 6-metre decompression stop and subsided before or shortly after surfacing. In two cases moderate bilateral joint pain occurred during decompression from 117 and 120 mfw and persisted following diving; these divers were diagnosed with DCS and successfully treated with hyperbaric oxygen.

The influence of maximum diving depth on DHS was examined by linear modelling of all 200 diving and 9 non-diving (DEPTH = 0) data and the results are shown in Table 2. In model 1 there was a significant, approximate unit increase in DHS for DEPTH for every 40 mfw increase in depth. The intercept (DHS at DEPTH = 0) was not significantly different from zero for the fixed component of the model; however, there was a significant inter-individual variation in intercept with a standard deviation of 1.3 (95% confidence interval 0.8, 2.2). This difference in how individuals differently score their normal non-diving health status is evident in Table 1. A strong linear relationship was not evident in a plot of DHS versus DEPTH (not shown), where it was obscured by the between-diver variability. It is evident from Figure 3 that poor health outcomes were associated with the deepest dives and by trial and error it was found that the DHS data were well described by factoring dive depth as =90 mfw or >90 mfw (model 1a). Model 1a had a larger log-likelihood than model 1 but these models are not nested and cannot be formally compared. Both model 1 and model 1a provided a significantly better fit to the data than the null model (model

Figure 3
Plot of maximum depth and total decompression time of air and trimix dives. Uneventful dives (DHS <6 or no new symptoms following diving) are plotted as open circles and dives where DHS =>6 and new symptoms occurred following diving are plotted as filled circles. Mean bottom time was 21 minutes.

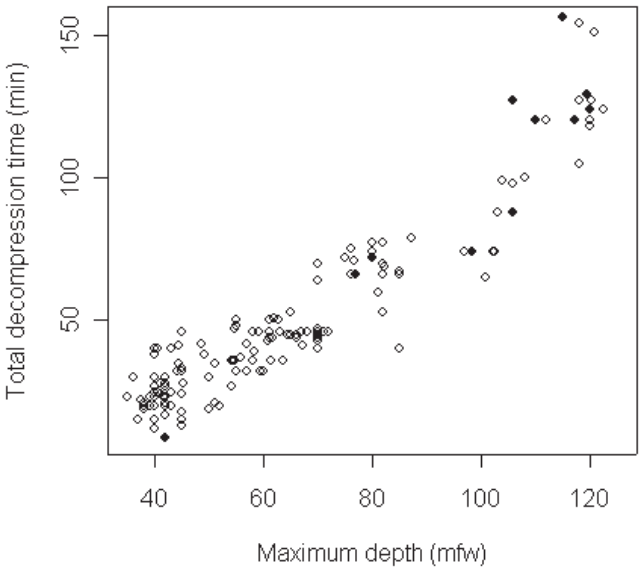


Table 2
Model comparisons (CI = confidence intervals; df = degrees of freedom; LL = maximised log-likelihood)

Model	Variables	Parameter estimate	Parameter (95% CI)	P	df	LL	Likelihood ratio test	Likelihood ratio ratio	Likelihood ratio P
1	intercept	0.7	(-0.4, 1.8)	0.1856	4	-427	1 vs 2	24	<0.0001
	DEPTH	0.026	(0.016, 0.036)	<0.0001					
1a	intercept	2.0	(1.0, 2.9)	0.0001	4	-417	1a vs 2	44	<0.0001
	DEPTH>90	2.4	(1.7, 3.1)	<0.0001					
2	intercept	2.3	(1.3, 3.2)	<0.0001	3	-439			

2) where only DHS varied between divers.

Discussion

Since technical diving is not regulated there are no records of the number, nature, or outcome of technical dives conducted worldwide with which to compare the present series. For the same reasons it is not possible to define typical technical diving. However, the training agencies provide technical diving training to a typical maximum depth of 100 msw, which could be considered the present status of mainstream technical diving. At the pioneering fringe, this author is aware of a handful of depth-record-setting scuba dives briefly reaching the vicinity of 300 msw, exploration dives with substantial bottom time in the vicinity of 180 and 260 msw, and two-hour bottom times in the vicinity of 70 msw. By these latter standards the present series of dives is not extreme.

To the author's knowledge this small series of dives is the only published analysis of health outcome following technical diving. Based on the two treated cases of DCS in the present series, the probability of DCS using the described protocols is between 0.1% and 3.4% (95% confidence limits). A zero risk of DCS is neither possible nor was expected and the measured risk of DCS seems reasonable. However, inclusion of all poor health scores indicates a probability of poor outcome of between 3.1% and 10.2% (95% confidence limits). Nine of these incidents occurred during 33 dives deeper than 90 mfw, indicating a probability of poor outcome during these deeper dives of between 13.3% and 45.5% (95% confidence limits).

Health outcome was measured by diver self-assessment using the decompression health survey. Extensive validation of the survey against medical assessment shows that high DHS correlates with diagnosed DCS.[6] Two of the poor health scores were confirmed as DCS by subsequent medical diagnosis. It seems likely that some of the remaining poor health scores were 'niggles' or marginal DCS. In occupational air diving the DHS correlates with calculated decompression stress in the absence of DCS.[7] No attempt was made to establish a similar correlation in the present series because there was an insufficient number of recorded dive profiles and no validated decompression-stress-calculation method for trimix dives. High DHS was associated with dives deeper than 90 mfw; although the data must be interpreted cautiously, it appears that the ZH-L16 decompression calculation method provided inadequate decompression for these deeper dives.

There was a trend towards a decreased safety margin with deeper dives; in order to minimise total decompression time, maximum use was made of limited scheduled bottom times to complete survey tasks. Nevertheless, all dives were conducted within limits prescribed by the ZH-L16 model. Inaccuracies in any decompression model as well as execution errors will accumulate with deeper diving: there is more opportunity for failure of a decompression schedule requiring 14 decompression stops than with five stops. Examination of depth/time profiles showed that decompression schedules were well executed. The data used to develop the ZH-L16 model include 211 simulated heliox dives up to 5.1 MPa (490 msw) but the majority of the development is based on dives to a maximum of 450 kPa (34 msw).[3,4] In the present series of dives, as is the case for much technical diving, the ZH-L16 model was used to plan dives that are quite different from the dives used to develop and test the model. It is not surprising that such extrapolation of the ZH-L16 model is not always successful.

During the last several years there appears to have been a shift in the technical diving community away from using the ZH-L16 model and towards using calculation procedures based on the varying permeability model of bubble formation and its derivatives.[8] These models calculate a theoretical number of bubbles formed during decompression and schedules are designed to limit this bubble formation. Implementations of the varying permeability model are now widely available, usually with the bubble model overlaying a ZH-L16 mammillary compartment structure. There are anecdotal reports of both the success and failure of decompression schedules based on these bubble models but there is apparently no published human testing or field validation.

Recreational diving has developed with existing, relevant decompression procedures, such as the standard air diving tables of various navies. More recently, diver-carried electronic decompression computers have been programmed with variants of the ZH-L16 model, which is well tested in the range of recreational diving. There has even been specific development of recreational no-stop decompression procedures. No such basis exists for technical-diving decompression procedures. Decompression safety remains a principal challenge for technical divers.

Acknowledgements

All the divers thank the Ashby family for their kind permission over many years to dive in 'The Shaft'. Thanks to Dr Tim Payne for producing Figure 1.

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SPUMS Annual Scientific Meeting 2005

23 April - 1 May

CocoPalm Resort and Spa

Dunikolhu Island, Baa Atoll

The Maldives

Theme: Evolving Diving Practices

Principal Guest Speaker: Michael A Lang

Marine Sciences Program and Scientific Diving Officer
Smithsonian Institution, USA

Conference Convenor:
Dr Cathy Meehan

McLeod Street Medical,
67 McLeod Street,
Cairns, Queensland 4870,
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The correct dates for the 2005 SPUMS ASM are as above. Australian delegates will arrive back into Australia on 01 May. In previous editions of the Journal preliminary dates were published which have been changed for improved airline connections and premium diving conditions. We apologise for any confusion this has caused.

Review articles

Venomous marine animals

Peter J Fenner

Key words
Marine animals, envenomation, injuries, toxins, first aid, review article

Abstract

(Fenner PJ. Venomous marine animals. *SPUMS J*. 2004; 34: 196-202. **Paper presented at the SPUMS ASM in Noumea, 2004**)
Marine animals cause human deaths and severe morbidity in many countries having tropical or sub-tropical waters. With travel to remote areas becoming more commonplace, general practitioners, and diving and tropical medicine specialists must routinely advise others of potential marine envenomation problems. Simple first aid and medical treatments are suggested. Although not all current recommended first-aid treatments have been proven, most have a sound basis and appear effective. The known world distribution of the main groups of venomous marine vertebrates is discussed and suggestions made for the awareness and prevention of such marine envenomation.

Introduction

Venomous marine animals are present worldwide but are mainly confined to the Indo-Pacific Region (Table 1). Their envenomation usually injects a large amount of venom into one area, with a consequent relatively slow absorption. This needs different first-aid principles to jellyfish envenomation, where extensive amounts of tentacle contact usually results in a significant amount of venom injected over a larger area with consequent rapid onset of symptoms.

The first-aid treatment of all marine envenomation can be divided into two main treatment groups (Figure 1).

1 Prevention of further envenomation
   - Pressure-immobilisation bandaging[1] – bites, etc
   - Vinegar – for box jellyfish stings only, e.g., *Chironex*, Irukandji (*Carukia barnesi*)[2,3]

2 Treatment of pain
   - Heat (hot water) for penetrating spiny fish injuries (fish barbs, etc)[4]
   - Cold packs or ice for jellyfish stings (any species)[5]

In addition, resuscitation in the field followed by advanced life support may be required as clinically indicated.

First-aid treatment of marine envenomation pain

HEAT TREATMENT: PENETRATING SPINE INJURIES

Heat is the best first-aid treatment to date for all penetrating spine injuries.[4] The envenomed limb should immediately be placed in hot water; about 43 ºC is the most that the majority of people can stand without causing scalding. It is essential the rescuer tests it first, as the victim may be in such pain that they cannot properly distinguish the apparent water temperature, and many severe scalds have been caused by not doing so.[4] In Europe, there have been so many victims scalded from this treatment, especially when treating Weever fish injuries, that their policy has changed to cold packs for penetrating injuries (personal communication, Dietrich Mebbs, Clinical Toxicology Course, Adelaide, 2003).

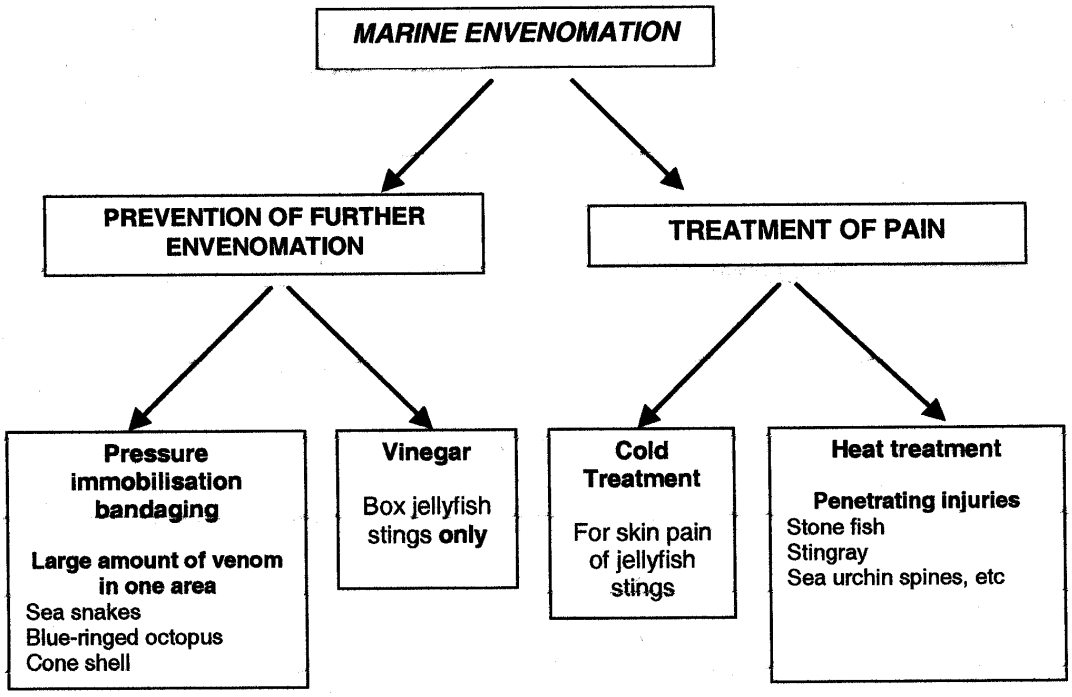
It has been postulated that the hot water denatures the venom; but a temperature of about 60 ºC is needed to achieve this in the species concerned. It is a little difficult and unwise to place a victim's appendage into water at around 60 ºC!

Hot-water treatment is very effective, but as soon as the water cools the pain will return. Consequently, it needs topping up regularly, testing each time this is done.[4] The author has known it used for up to four hours until someone arrived back for further treatment; and it still remained

Table 1
Geographical locations of human fatalities from marine animal envenomation

Blue-ringed octopus	Australia, Singapore
Cone shell	Australia, Fiji, India (Banda), New Caledonia, Japan (Okinawa), Vanuatu
Sea snake	Burma, Malaysia, India (Madras), Indonesia (Java), Japan (Okinawa), Oman, Vietnam
Stingray	California, Colombia, Fiji, New Zealand, Surinam, Texas
Stonefish	?Australia (Thursday Island), East Africa, Japan, Seychelles

Figure 1
First-aid treatment of marine-envenomation pain



effective. A source of hot water on a deserted island is usually obtainable from the engine on the boat that transported people there.

After reaching medical aid, the envenomated area must be assessed and, if warranted, surgically explored under good anaesthetic cover; a nerve block or general anaesthetic, not just local anaesthetic.[6] Often the author also treats victims routinely with antibiotics to cover marine organisms (see below). Finally tetanus prophylaxis must be up to date. The tetanus protocol has recently changed, with the usual course up until the age of 15 years, then just a single booster at 40 for life cover. Obviously, many clinical factors must be considered if further tetanus injections may be needed for any particular wound at any time.

The most common causes of penetrating spine injuries are described below.

Stonefish

There are two main types of stonefish – the estuarine and reef species. They are brown to sandstone in colour, blending perfectly with their surroundings and so are very difficult to see (Figure 2). The usual means of envenomation is when the unsuspecting victim steps on them, often whilst walking on rocks. The stonefish (*Synanceja sp.*) has 13 dorsal spines, which are defensive and not used for capture of food. Paired venom glands are attached to each spine, and when the spine is pushed down (e.g., pressure applied by a human foot) it penetrates the skin and acts like an injector, forcing venom into the wound.

ENVENOMATION

The envenomation is quite characteristic (Figure 3). The puncture mark often has a local bluish tinge around it and the severe local pain (usually on the foot) extends, causing severe limb pain and paraesthesiae. The victim is usually distraught with pain and in apparent shock, with low blood pressure, pale skin, nausea and dizziness.[7]

TREATMENT

The severe local pain usually responds to hot-water treatment as described above and/or local nerve block. Parenteral analgesia may be necessary and Commonwealth Serum Laboratories (CSL) antivenom is available and provides good pain relief given as soon as possible, but later use in intractable pain is still effective.

FATALITIES

Despite the stonefish's fearsome reputation, there have been no fatalities in Australia. One described as occurring on Thursday Island appears more like a stingray death when the actual evidence is considered.[8] World fatalities are both rare and poorly documented, and often cannot be corroborated. In the deaths mentioned in the literature occurring in East Africa, the reports that do exist are poor and first-hand accounts do not actually exist.[9] In Okinawa a fisherman supposedly picked up a stonefish and suddenly collapsed in the water: classified as a stonefish death but with a very unusual history of sudden onset. Perhaps the pain triggered a pre-existing cardiac condition?[4] Another

'unusual' death was in a diver who sat on one underwater. The injury was so painful that he made a rapid, uncontrolled ascent to the surface and died from a massive arterial gas embolism (personal communication, Williamson J, 2002).

Stingray

The stingray is a large, flat fish with flaps that enable it to swim gracefully through the water. It has a tail with at least one barb, and occasionally up to seven. The barb(s) face backwards in the normal state and are usually covered in a friable sheath of tissue. Stingrays are usually gentle creatures, known as the 'pussycat of the sea'. However, they have a bad habit of burying themselves in the sand and just resting there. Fishermen also have some very bad habits, such as walking through shallow water not looking where they tread.

ENVENOMATION

Treading on the flap of the stingray produces a reflex action where the tail whips forwards with great force, and the barbs can easily penetrate the integument of the victim, even through rubber or leather covering (boots, etc., Figure 4).

Pain is instant and severe. Jagged lacerations can occur and heavy bleeding has resulted in several fatalities.[10] The barbs can either break off and remain embedded in tissue, or stab the tissue and be totally withdrawn. As the barb sheath penetrates the tissue its surrounding integument is torn off, leaving a trail of venom and debris. This venomous tissue causes localised myolysis and inflammation with local or extended tissue necrosis. Penetration of abdomen and chest greatly increases morbidity and mortality.

TREATMENT

The whole tract should be excised, if at all possible, and the crater packed with an alginate-based wick to allow healing by secondary intention. These dressings are useful in toxin absorption and are left in place for as long as possible. They frequently fall out at about 8 to 10 days, or are then easily removed without pain or damage to the healing wound.[11] Tetanus immunisation is advised. Follow up to exclude secondary infection may be necessary as this often causes major problems.

FATALITIES

At least 17 fatalities from stingrays have been reported worldwide, including in New Zealand, Surinam, West Atlantic, Texas, Fiji, California, and Australia.[4] Although it was reported that a fresh-water species of stingray had caused multiple fatalities in Colombia,[12] this report has recently been questioned for its accuracy. Trunk wounds are responsible for most of the fatalities, but acute exsanguination has caused at least two deaths, and another occurred from tetanus complicating a lower-leg wound.

The two Australian deaths were bizarre. The first occurred in 1936 in St Kilda in the open swimming baths. A soldier dived into the water but suddenly surfaced in severe pain and quickly died. He had a stab wound in his chest that had penetrated his heart. Despite draining the whole of the baths and finding nothing, the cause was presumed to be a stingray.[13] The other occurred about ten years ago as a boat travelled across Mourilyan Harbour near Innisfail. A large stingray leaped out of the water. Its flaps hit the first person and the barbs hit a 12-year-old boy sitting in the middle of the boat, with one barb entering his knee and another the chest. In hospital, he had only some chest discomfort and was cardiologically stable with a normal chest X-ray. The chest wound was explored using local anaesthetic and a probe and the tract suggested the barb had glanced off a rib and been withdrawn as there was no trace of any part of it. After thorough local cleansing and injection of Betadine™ down the tract, he was sent home the next morning. Five days later he suddenly dropped dead at home.[6] The venom causes muscle necrosis, and his post-mortem examination revealed that the barb had gone directly into his heart before being withdrawn and that the retained venomous tissue caused progressive myocardial necrosis. Suddenly, this caused a small hole to develop in the cardiac muscle with its consequent catastrophic effect. Death was caused by cardiac tamponade with a huge haemothorax.[6]

Bullrout

These are common fish in fresh and brackish water where they are usually unseen and easily stepped on by their unsuspecting victims, in a way similar to stonefish.

Other fish with venomous spines

Lungfish, scorpion fish, lionfish (Figure 5) and Weever fish all have penetrating barbs that may cause local pain ranging from irritating to really severe and agonising. Lionfish do have a reputation in the Philippines for causing death, but again nothing has ever been proven and published in a reputable journal. However, the problem is that in the Philippines you die, you are buried and that's it; there are no good or reliable figures on mortality or morbidity on any deaths in non-metropolitan areas.[4]

Sea urchins – echinoderms

Sea-urchin-spine penetrations are common and are not very venomous but most unpleasant. Echinoderms have hundreds of sharp spikes, looking like 'spiky balls'. They are often present on rocks close to or in shallow water. When they are accidentally stepped on, thousands of little spikes break off in the victim's foot. Though they are not venomous, they are mentioned here as they fit into the heat-treatment framework.

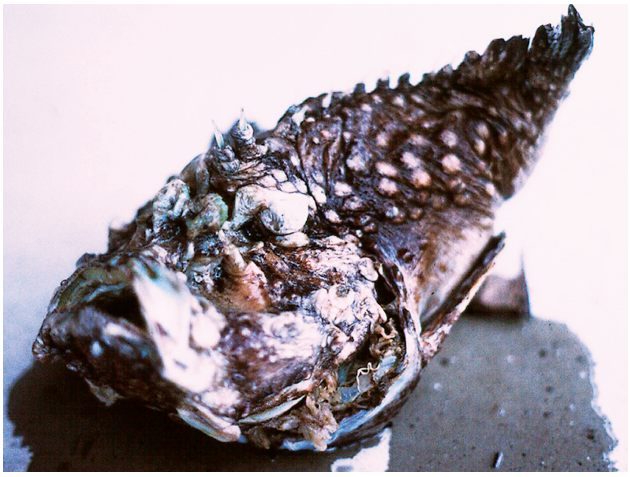


Figure 2. Stonefish

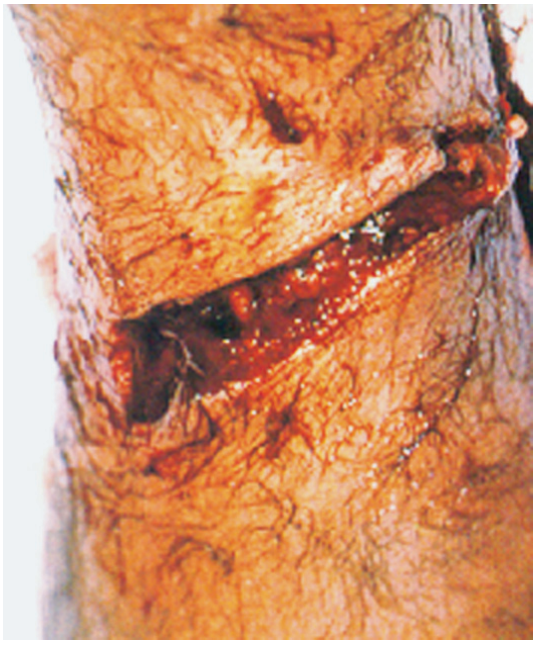


Figure 4. Stingray laceration

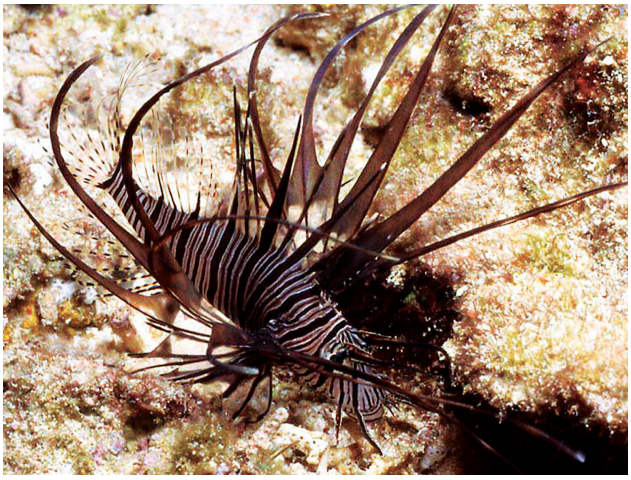


Figure 5. Lionfish

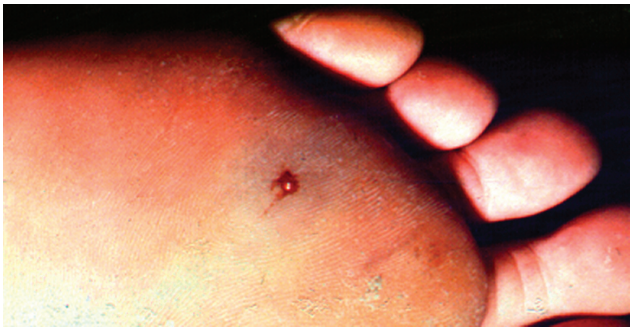


Figure 3. Stonefish injury

Figure 6. Crown of thorns starfish

Figure 7. Cone shell

Figure 8. Blue-ringed octopus

TREATMENT

There are bizarre stories of treatment (sounding more like torture), where the bottom of the foot is beaten with a stick to 'break up all the bits'. Theoretically, this should make it easier for the tissue to exude these smaller particles! Alternatively, soaking the foot in vinegar is said to help dissolve the spikes; it does not! In fact, there is no really effective, long-term treatment. Hot water relieves the pain during use and the only other treatment consists of picking out any spikes individually. This is very difficult as they break up, and this method is also painful. However, they need to just exude out individually. An X-ray should be taken to identify any deeper, broken-off fragments.

Crown of thorns starfish

Similar to the echinoderm, the crown of thorns starfish has sharp spines that can easily cause a penetrating injury (Figure 6). The spine tips are covered with a mild venom that may cause local irritation. Recurrent injury is seen in marine scientists and professional 'pest controllers' whilst injecting the animals with copper sulphate, and leads to sensitisation which may cause more severe or extensive local reactions.[4]

Pressure-immobilising bandages (PIB)

LARGE AMOUNT OF VENOM IN ONE AREA

When there is a large amount of venom in one area, Sutherland proved that, in Australian elapid snake envenomation, PIB worked very efficiently as a first-aid treatment by preventing spread of venom until medical aid could be reached.[1] The venom is not destroyed but with intravenous access, medication, colloids and antivenom, this effective, timed release of the venom means that it could be counteracted by the applied medical treatment.

It is in this context, without further medical experimentation that would be very difficult, that PIB has been recommended for envenomation by sea snake, cone shell, blue-ringed octopus and even, with great controversy, major envenomation by *Chironex fleckeri*.

There has always been a fight, especially amongst various first-aid groups and emergency-medicine doctors, as to where the bandage should start. The most current agreed practice, discussed by the Australian Resuscitation Council recently, is that you start at the tips – distally to proximally. However, the author's strongly held view is that there is real benefit from immediate pressure bandaging over the envenomation site followed by pressure bandaging from the distal tip of the envenomated limb(s) towards the body. Finally, and most importantly, pressure immobilisation should involve the whole limb, which also must be completely immobilised; both legs tied together with firm boarding between them, or a sling applied to the arm and it tied to the victim's side.

In summary, based on Sutherland's work on Australian elapids, PIB is used in the first aid for envenomation by:
- land snakes in Australia
- sea snakes (probably)
- cone shells
- blue-ringed octopus.

Cone shells

Cone shells are brightly coloured, triangular-shaped shells with a longitudinal fold, or 'split' running the length of the shell from the blunt shell top to the point (Figure 7). A proboscis, like a small, hollow, flexible tube, can emerge from anywhere along the slit, although frequently and usually from the tip of the cone. At the base of the proboscis is an area containing a number of barbs, called radicular teeth. These barbs are bathed in a potent venom. The proboscis acts similarly to a blowpipe and these tiny barbs can be fired a reasonable distance, possibly a metre or so. This barb envenomation leaves a bolus of venom in one area; making PIB the initial first-aid treatment of choice.

Human envenomation occurs in a few main ways. It most often occurs in shell collectors. In the one Australian fatality, a shell collector picked up a 15 cm *Conus geographicus*, and placed it in his pocket. Despite feeling a small prick he thought nothing of it, but developed breathing difficulty and died on the boat trip to the mainland.[14]

ENVENOMATION

Cone shells produce a rather unusual envenomation. It causes a localised, sharp pain, sometimes very mild but sometimes extremely painful.

There is usually very little reaction at the site. Careful illumination may reveal an embedded barb, but this is very rare. Local flushing of the skin or a small macular rash may be present at the envenomation site. Macular rashes can develop over the face and ears or become generalised and may be intensely pruritic and distressing. The skin pain and pruritus may continue for weeks.[4]

Envenomation by some of the fish-eating cones, particularly *Conus geographicus* and *Conus textile* can rapidly cause numbness and local swelling, marked nausea, incoordination, muscular weakness and difficulty breathing due to progressive weakness of diaphragm and intercostal muscles. This may very rapidly proceed to respiratory paralysis and death from asphyxia without immediate airway and ventilatory support.[4,15]

An unusual presentation reported by the author involved someone just walking along the beach, touching nothing, but feeling a sudden small stab in the leg. Minutes later he had the severe pruritic rash all around his face and ears

and developed trouble breathing. Despite his oxygen saturation dropping to 85% on room air and commencing oxygen in the ambulance, he was not admitted to hospital as the rash had gone and his breathing had improved! This presentation is unproven and atypical but can surely be attributed to cone-shell envenomation after all other possibilities had been exhausted.

TREATMENT

PIB and respiratory support are required. Initial expired-air resuscitation is followed by intubation and mechanical ventilation in hospital for four to six hours until spontaneous breathing returns.[4]

FATALITIES

There have been fifteen proven deaths, all of them from the Indo-Pacific region.[15] The Australian death occurred on Hayman Island in 1936.[14] Other deaths have occurred in Fiji, and Okinawa, Japan.[4]

Sea snakes

Sea snakes are common in all oceans except for the Atlantic. They are most common in tropical and sub-tropical zones of Australia. They are similar in appearance to land snakes except they have a flattened paddle-like tail that makes swimming possible. They are air breathers having no gills, and are inquisitive but not usually aggressive. However, there was one notable attack by a sea snake at Bondi Beach that caused severe envenomation problems.[16]

Sea snakes in Australia can be divided into two main groups:

- those with large mouths that rarely bite and even more rarely envenomate, i.e., they do not inject their venom
- those with small mouths and, although their venom is some of the most potent in the world, are almost unable to take a large enough bite to envenomate. However, even a scratch from a tooth covered in venom may produce a full envenomation and death.

ENVENOMATION

Most bites are 'dry', with less than 10% of sea snakes injecting venom. The bite is relatively painless, and, if venom is injected, is followed by symptoms that include drowsiness, nausea and vomiting, weakness, visual disturbances, breathing problems and muscle pains or stiffness. Severe myolysis may cause renal impairment.[4]

TREATMENT

Antivenom is available for sea-snake envenomation from CSL. Interestingly, when CSL were actually producing this antivenom they were injecting the sea-snake venom into the same horses that had been previously used for tiger-snake antivenom production! Thus, if sea-snake antivenom is unavailable, it is possible to use tiger-snake antivenom, which works just as effectively.

FATALITIES

Fatalities have occurred regularly worldwide.[15] The greatest problem is for fishermen. When they go out fishing, sea snakes get caught up in their nets and have to be removed. This is a common time for envenomation, and figures suggest there are probably about 100 to 150 deaths per year (personal communication, Warrell D, Oxford, 2003). As only about 3% of bites are thought to be fatal this means there may be some 35–40,000 sea-snake envenomations per year.

Blue-ringed octopus (*Hapalochlaena spp.*)

The blue-ringed octopus is generally yellow to brown in colour, enabling it to blend in with its background, which is usually a rocky area, e.g., it is often found in shallow inter-tidal rock pools (Figure 8). It has eight arms growing up to 15–20 cm in diameter with the tentacles extended. When frightened or in danger, or when 'angry', e.g., being prodded by young children with sticks (!), many small, electric-blue rings appear, making it look very attractive. This often results in children picking it up to play with it, thus getting envenomed out of water. It is found all around Australia and throughout the Indo-West Pacific.[4]

ENVENOMATION

Envenomation occurs after a painless bite from a beak that is under the body of the octopus. The venom is tetrodotoxin (as in 'fugu', the Japanese fish) and is produced in the salivary glands. These are connected to the beak by small ducts.[4] The bite is commonly painless and unnoticed, and not every bite results in symptoms of envenomation. However, deaths and near deaths are well documented.[17] The venom is a salivary toxin with a complex composition, including a tetrodotoxin and hapalotoxin of great biological interest.

The first sign of serious envenomation may occur within ten minutes, with the onset of generalised progressive muscle weakness, dysphasia, dysphagia, visual disturbances and respiratory difficulties.[14] Nausea and vomiting may occur, and collapse from muscle weakness is soon seen, with unconsciousness and death in the absence of effective resuscitation. The autonomic effects of the toxin may result in fixed, dilated pupils, suggesting death. However, the victim may be conscious and aware, although unable to breathe, and may be able to recall everything that is going on during resuscitation (personal communication, Williamson J, 1985). Death occurs from respiratory paralysis, unless resuscitation is commenced.[18]

TREATMENT

PIB should be applied, and assisted ventilation will be needed for four to six hours, or possibly up to 12 hours, after which spontaneous breathing usually recurs.

FATALITIES

There are two published deaths in Australia and one in Singapore.[18,19] Note that envenomation never occurs in the water, only when the creature is irritated when taken from its natural habitat.

Marine-wound infections

Marine wounds often become contaminated by a variety of organisms different to those usually cultured from 'normal' skin wounds. Organisms include *Vibrio*, *Altermonas*, *Mycobacteria spp*. and marine varieties of *Pseudomonas*. These organisms do not respond to the usual skin antibiotics such as flucloxacillin or erythromycin. The antibiotics of choice, if culture and sensitivity are not immediately available, are doxycycline (100 mg daily) or a third-generation cephalosporin.[20] When taking a culture from a marine wound, it is essential to state this on the pathology form. Laboratories will then culture these organisms on saline-based culture plates, as marine organisms will not grow on the usual culture media.

Conclusions

Envenomation from marine animals causes human deaths and severe morbidity in both tropical Australia and many countries in the world surrounded by tropical, or sub-tropical waters. With travel becoming commonplace, doctors involved in this area must routinely advise patients and colleagues about the hazards of marine envenomation, both in Australia and overseas, whenever they are consulted or become aware of such trips.

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Flying after recompression treatment for decompression illness: why wait four weeks?

Christopher J Acott

Key words
Flying (and diving), decompression illness, treatment, treatment sequelae, review article

Abstract

(Acott C. Flying after recompression treatment for decompression illness: why wait four weeks? *SPUMS J*. 2004; 34: 203-8. **Paper presented at the SPUMS ASM in Noumea, 2004**)
The risk of a relapse associated with flying following recompression treatment for decompression illness (DCI) is unknown. Recommendations regarding the safety of flying following treatment for DCI are varied and personal, and few are published. The author's view that a diver should wait four weeks is based on:

- a review of the current literature
- personal clinical observations
- case reports
- postulated pathophysiological mechanisms for recorded relapses such as reactivation of 'primed' leucocytes and endothelium by new bubble formation and growth of 'stabilised' bubbles
- the physics and physiology of airflight including hypobaric hypoxia, hypobaric hypovolaemia, gas cavity expansion in accordance with Boyle's law and a decrease in gravity.

This evidence is presented in the following review.

Introduction

Flying and altitude exposure following compressed-air diving (CAD) are associated with a risk of decompression illness (DCI) due to a lower barometric pressure. Various recommendations have been published concerning when it would be safe to fly following diving; however, few have been tested.[1–3] These recommendations have also been used as a guide concerning the minimal stay at 'ground level' before flying following recompression treatment for DCI. Similarly, these recommendations are varied and based on unsubstantiated opinion and bias; few are published. Some of these recommendations are listed in Table 1.

The objects of this paper are to review current literature, postulate relapse mechanisms and provide supportable advice that can be given to divers following treatment for DCI.

Environmental and physiological factors associated with airflight

CABIN PRESSURE: HYPOBARIC HYPOXIA

Modern commercial aircraft fly at altitudes between 9,000 and 12,000 metres above sea level (mASL) to keep fuel costs low and to avoid unfavourable air conditions. With ascent the atmospheric pressure drops so that at 12,000 mASL the pressure is 176 mmHg (23.38 kPa).[4] However, the pressure inside the cabin is maintained by drawing in external air and limiting its outflow. Most aircraft cabin structures can withstand a pressure differential of 430 mmHg (57.14 kPa), thus the pressure inside the cabin at 12,000 mASL would be 606 mmHg (80.53 kPa). This is the air pressure found at an altitude of 1,600 m, therefore, the 'cabin altitude' is said to be 1,600 m.[4] With the decrease in atmospheric and cabin pressure there is a corresponding decrease in the inspired partial pressure of oxygen (P_IO_2) in the air breathed, causing a hypobaric-induced hypoxia. Depressurisation to 'cabin altitude' takes about 20 minutes.[5,6] 'Unpressurised' aircraft flights in Australia are limited to 3,000 mASL (approximately 68 kPa).[5,6]

Table 1
Recommendations for flying after treatment for DCI (TT USN 6A – US Navy treatment table 6A)

Organisation	Suggested time interval
Australian Standard AS2299	7 days
Royal Adelaide Hospital	4–6 weeks
Townsville General Hospital	3 weeks
Royal Hobart Hospital	2 weeks
Prince of Wales Hospital	1–2 weeks
Fremantle Hospital	2 weeks
PADI	3 days
NAUI	1 day
DAN (unconfirmed)	3 days
US Navy	24 hours for Type 1
	48 hours for Type 2
	72 hours if symptoms persist
	72 hours after TT USN 6A

To prevent hypobaric hypoxia the aircraft cabin is usually pressurised to give an equivalent altitude of 2,440 mASL, at which the ambient pressure is 562.4 mmHg (74 kPa).[3] From the alveolar gas equation[7] the alveolar partial pressure of oxygen (P_AO_2) at a 'cabin altitude' between 2,000–2,500 mASL, excluding the saturated vapour pressure of water, would be between 63 and 68 mmHg (approximately 9.03 kPa) and because of the alveolar/arterial difference the arterial partial pressure of oxygen (P_aO_2) would be between 35 and 58 mmHg (4.78–7.70 kPa).

Hypoxia causes pulmonary vasoconstriction and mild hyperventilation, due to hypoxic stimulation of peripheral chemoreceptors, shifting the oxygen dissociation curve to the left and marginally increasing the P_IO_2 and hence P_AO_2 (the alveolar gas equation).[7]

The drop in cabin pressure will cause a 4% reduction in the amount of oxygen carried by the blood and has a minimal effect on healthy passengers. Even with a P_aO_2 of 56 mmHg the passenger lies on the flat part of the oxygen-haemoglobin dissociation curve.[7] However, this reduction may be critical for tissues with a marginal blood supply, particularly when the dissociation curve is shifted to the left.[6] A reduction in P_aO_2 will reduce the flux of nitrogen from air bubbles present due to a reduction in the 'oxygen window' effect, thus slowing bubble resolution.[6,8]

Acute hypoxia induced by reducing the P_IO_2 to 71 mmHg (9.43 kPa) promotes a rapid microvascular response characterised by increased leucocyte rolling and adherence to the venular endothelium, leucocyte emigration to the perivascular space, increased vascular permeability and proinflammatory features in the endovascular compartment. This appears to be a generalised response. After three weeks of acclimatisation to hypoxia this microvascular response resolves.[9,10] Although the induced hypoxia during flight is not as severe as this it is not known what degree of hypoxia (if any) in combination with other factors may activate a previously 'primed' or 'activated' endothelium.

CABIN PRESSURE: DECREASE IN GRAVITY

Gravity decreases with altitude. This decrease initially causes:

1 a decrease in venous and arterial pressures in the dependent capillary beds promoting intravascular fluid retention, hence decreasing blood pooling in the legs causing an increase in venous return, and hence thoracic and pulmonary blood volume;[7] and
2 a linear decrease in gas density with the decrease in atmospheric (cabin) pressure promoting, perhaps, better alveolar gas mixing; and
3 these combining to produce a more uniform distribution of ventilation and blood flow.

CABIN PRESSURE: HYPOBARIC HYPOVOLAEMIA AND OTHER CARDIOVASCULAR ALTERATIONS

Cabin air is exchanged every 3–4 minutes and because it is drawn in at altitude it has a very low water content. Engineering constraints do not allow for humidification systems in aircraft; hence, cabin atmosphere has a low relative humidity (15–25%) which will lead to water loss from the respiratory tract.[4] In addition, the increase in preload due to the increase in thoracic blood volume results in a diuresis. This induced diuresis and loss of respiratory fluid promotes hypovolaemia (called hypobaric hypovolaemia).

The combination of hypoxic pulmonary vasoconstriction and increase in pulmonary capillary blood volume results in pulmonary hypertension. This increases the work performed by the right heart and promotes peripheral, particularly ankle, oedema. The increased hypoxic vascular permeability may also contribute to this.[9]

Early in hypovolaemic shock the lumen of capillaries become narrower as a result of swelling of hypoxic endothelial cells and adhesion of activated polymorphonuclear leucocytes to the endothelium of the post-capillary venules. The interaction of leucocytes with the endothelium is followed by the release of vasoactive mediators and toxic oxygen species promoting macromolecular leakage, interstitial oedema and redistribution of tissue perfusion, hence compromising oxygen delivery.[11] Although the altitude-induced hypobaric hypovolaemia is not severe enough to initiate these mechanisms it may have an additive effect to endothelial reactions occurring because of hypobaric hypoxia or because the endothelium has been 'primed' due to bubbles.

CABIN PRESSURE: BOYLE'S LAW

Gas spaces will expand with a decrease in ambient pressure in accordance with Boyle's law and Pascal's Principle;[12] hence, any tissue bubbles present during altitude exposure will grow. At an ambient pressure of 74 kPa bubble volume will increase by about 35% (assuming a spherical bubble), which corresponds to an increase in radius of nearly 11%. If bubbles have been present for some time they will have become 'stabilised' with a coating of surfactant, haematological and immunological active substances.[6] These bubbles will be in equilibrium with the surrounding tissues and will consist of nitrogen, carbon dioxide, oxygen and water vapour. As the ambient pressure drops, these bubbles will enlarge and quantities of these gases will diffuse into the bubbles until equilibrium is again achieved. So the actual prediction of bubble/gas expansion by Boyle's law may initially be an underestimation.

A summary of the pressure changes with altitude and airflight and other relevant clinical information are listed in Table 2.

Table 2
Changes in pressure, temperature, gas space volume expansion and alveolar oxygen pressure (P_AO_2) breathing air, 30% oxygen and 100% oxygen (saturated water vapour pressure included) with airflight.[3–6] Table modified from Everest et al[13]

Altitude (mASL)	Pressure (kPa)	°C	Expansion %	P_AO_2 breathing Air (mmHg)	30% (mmHg)	100% (mmHg)	General comments
Sea level	101 (760 mmHg,)	15		100	178	710	Average temperature
304.9	97.4 (733 mmHg)	13	+3.6	96	169	680	Minimum altitude for helicopters
609.8	93.8 (706 mmHg)	11	+8.0	91	162	657	Altitude for helicopters above sea terrain
914.7	90.5 (681 mmHg)		+12.0	85	153	626	
1219.6	87.2 (656 mmHg)	7	+16.0	81	146	604	Cabin altitude for ambulance aircraft
2134.3	77.9 (586 mmHg)	1	+29.0	67	125	535	Cabin altitude for commercial aircraft
3049.0	69.5 (523 mmHg)	-5	+45.0	55	106	474	Hypoxic threshold, ceiling for helicopters
6098.0	46.4 (349 mmHg)	-25	+117.0	20	55	299	Upper cruise altitude for turbo prop aircraft
8200.0	37.5 (282 mmHg)		+170.0	<10	35	231	
12196.0	18.7 (141 mmHg)	-56	+439.0	-	-	94	Upper cruise altitude for commercial aircraft

Pathophysiology of decompression illness (DCI) and postulated mechanisms of a relapse following treatment

The pathophysiology of DCI involves bubble formation and either intra- or extra-vascular growth. These bubbles distort tissues, obstruct perfusion and interact with formed blood elements or proteins (stimulating platelets, denaturing lipoproteins, activating and aggregating leucocytes, activating complement and coagulation pathways, releasing cytokines, and causing capillary leakiness). Bubbles may also damage both the luminal surfactant layers and endothelial cells of blood vessels.[14]

Relapse following initial treatment is multifactorial:[15]
- persistence of gas bubbles
- new bubble growth
- tissue ischaemia and oedema
- reactivation of blood proteins, leucocytes and endothelium
- barotrauma on ascent
- a combination of some or all of these mechanisms.

These mechanisms may also be responsible for relapse with airflight. However, barotrauma on ascent is unlikely.

Divers Alert Network (DAN) data reported that 40% of divers were asymptomatic following one recompression treatment, with diminishing clinical benefit with successive treatments.[16] Only 67.7% of divers had complete relief of symptoms following six treatments (six days, assuming one treatment per day). This would indicate that following an initial US Navy treatment table 6 (Royal Navy table 62), 'stabilised' bubbles and their pathophysiological effects may still be present in some cases, but are responsive to recompression and hyperbaric oxygen therapy (HBOT).

Case reports: altitude exposure

There is a paucity of published case reports regarding a relapse with airflight following treatment for DCI. A series of four cases at the Royal Adelaide Hospital relapsed with symptoms on ascent to 300 mASL two days following treatment.[3] Allan showed recurrence of DCI symptoms two weeks post development in aviators.[17] Fury also reported recurrence after three days.[18] Millar reported nine cases of relapse following treatment with an altitude exposure (range 100 mASL to 'cabin' pressure). Six of these relapsed within seven days, while three did so 10 to 21 days post treatment (altitude exposure 400–1,000 mASL).[6]

Uguccioni et al reported on 126 divers some of whom had a return of or worsening of their symptoms during commercial flight.[19] Of the 74 who 'flew' within 72 hours of treatment, 20 had a return of symptoms. Seventeen of these did not have a full resolution of symptoms prior to flight. Of the 52 who 'flew' after 72 hours, 11 relapsed of whom five did not have full symptom resolution prior to the flight. These data show that, in divers still symptomatic prior to a flight, the risk of a worsening of symptoms decreases from 27% to 21% if they wait 72 hours.

In an unpublished series of 46 divers treated at the Townsville General Hospital, Australia (personal communication, Bishop E, 2004), 28 flew after treatment, of whom eight relapsed. No relapses occurred if divers waited 35 days before flying.

Recently a diver was treated at the Royal Adelaide Hospital who had relapsed twice following airflight. Each relapse was worse than the previous one. His initial symptoms were considered 'mild': paraesthesia in one arm and the face,

which was treated with surface oxygen. He waited three days before flying and relapsed following this brief flight. He was treated with standard recompression protocols and then waited five days on medical advice before his next flights, which included a flight from Canada to Australia. His final presentation was with paraplegia. The time frame over which his relapses occurred was three weeks.

Vann et al reported a DAN online survey that involved 121 divers, comparing relapse rates in divers who 'flew' and those who did not.[20] Online surveys have limitations, but the data showed that:

- those who relapsed 'flew' within 13 days
- those who 'flew' but did not relapse had a range of 1–28 days.

Case reports: persistence of bubbles

The effect of recompression treatment with HBOT on inert gas elimination and bubble dynamics remains poorly understood and understudied. There are data, however, which show that asymptomatic divers following treatment do still have circulating bubbles.[21]

Acott and Gorman published a case history of a diver who developed postoperative symptoms of decompression illness following a general anaesthetic involving the use of nitrous oxide.[22] The diver had his operation one week following a group of dives in which two dives were followed by symptoms of decompression illness that were 'treated successfully' with surface oxygen. He presented two weeks after his operation with symptoms suggestive of DCI. These had been present for the two weeks. His symptoms resolved with HBOT.

Hills and Le Messurier (reported by Butler[3]) followed up an asymptomatic abalone diver using X-rays that showed bubbles still present 22 days following a 'bends'-provoking dive. Gorman et al reported a series of asymptomatic divers following treatment for DCI who had persistent abnormalities detected by EEG, psychological tests and/or CT scan at four weeks following treatment.[23] The cause of these abnormalities was unknown but they may have been due to continuing presence of bubbles, and their continuing haematological activity, or residual nerve damage.

Personal clinical observations during follow up of treated divers over a decade have shown that divers do not 'feel 100%' until at least four weeks following treatment. However, it is not known whether this is due to persistent bubbles or their haematological and immunological effects.

Historically, there are data to show that bubbles exist for prolonged periods in excess of those predicted by mathematical models. Boycott et al showed the presence of bubbles in their experimental goats' spinal cords 15 and 27 days following decompression, and in blood six days after; the goats were asymptomatic prior to euthanasia.[24]

New bubble growth

There are data to suggest that bubble formation may occur during flight. Eckenhoff showed that venous bubbles were detected in 50% of subjects following decompression from saturation at only 3.5 msw depth.[25] This pressure difference is similar to that in exposure to an altitude of 74 kPa. Conkin et al demonstrated venous gas emboli in subjects subjected to 70 kPa or less.[26]

Discussion

Vann et al and others argue that altitude exposure relapses within five days following treatment may be an example of the natural history of DCI seen in those divers; that is, they would have relapsed regardless.[20] However, it is difficult to maintain this argument for times longer than this. Anecdotal data from case histories indicate that bubbles may remain in tissues much longer than predicted by mathematical and *in vitro* models of bubble dissolution.[3] The persistence of these bubbles is due to a surrounding 'semi rigid' haematological barrier, which provides a diffusion barrier. These 'stabilised' bubbles will change shape and size during airflight, distorting tissues with local pressure effects.[6]

Airflight may give rise to new bubble formation (see earlier) and animal data suggest that even a small number of 'silent bubbles' will cause endothelial damage and impairment.[27,28] The additive effect of the expansion of 'stabilised' bubbles with new bubble formation may further cause activation of haematological and immunological events during airflight resulting in an increase in inflammation and oedema. These haematological and immunological events may be 'primed' by earlier pathophysiological processes. It is unknown how long the vascular endothelium and leucocytes stay 'primed' following initial bubble stimulation (with DCI), but in the acute hypoxic model it is three weeks.[10]

In addition, the 'primed' endothelium may be reactivated by the physiological effects of 'hypobaric hypoxia and hypovolaemia' with airflight.

Proposed management to decrease risk with flying

ROLE OF INFLIGHT OXYGEN ADMINISTRATION

The postulated mechanism that hypoxia is mainly responsible for relapse during airflight is too simplistic. Oxygen via nasal prongs at 3 $l.min^{-1}$ will elevate the fraction of inspired oxygen (F_IO_2) to approximately 30%; therefore, at cabin altitude the P_IO_2 will be 154 mmHg (20.5 kPa). Although oxygen may be used to negate any hypoxic effect

during airflight, it will not diminish other physiological adjustments to the hypobaric environment, such as the decrease in gravity and the expansion of air-containing spaces, particularly bubbles. In Australia the aviation industry considers oxygen to be a hazardous material and it can be used on a commercial aircraft only on a prescription basis, which requires 48 hours' notice.[4]

PRE-FLIGHT DENITROGENATION WITH NORMOBARIC OR HYPERBARIC OXYGEN (HBO)

Pre-flight denitrogenation has been shown to decrease the incidence of altitude DCI in astronauts. Rice et al showed that 30 minutes of pre-flight normobaric oxygen breathing was effective.[29] However, earlier studies by Behnke and others show that denitrogenation may take up to 240 minutes.[30] In man, the initial elimination was measured at 50 $ml.min^{-1}$, decreasing to 0.1 $ml.min^{-1}$ at nine hours. Bornstein reported that 25–33% (200–300ml) of the total nitrogen was eliminated from a lean man in nine minutes.[31] Nitrogen elimination in animals was an exponential function plateauing at 240 minutes.[30] Any pre-flight denitrogenation, as practised by NASA and various military airforces, would require the diver to breathe oxygen while boarding the aircraft, which would prove to be impracticable in most circumstances.

Pre-flight HBO therapy would denitrogenate and oxygenate tissues but its effect on the endothelium and leucocytes is unknown even if they are 'primed' but not activated. Practicalities of the application of HBO, which treatment table one should use, and at what stage it should be applied before boarding the aircraft, would limit any usefulness. Currently, there are no data to suggest it would be useful.

Recommendations

The physiological changes associated with airflight following treatment may create an unpredictable DCI risk. However, if flight is unavoidable what can be done to reduce the risks of relapse?

- Explain to the patient (diver) that the risks associated with flight are unknown.
- Ensure the airline is aware that there may be a problem and has an adequate supply of 100% oxygen available.
- Maintain hydration before and during the flight.
- Consider the role of a prior hypobaric exposure before flight; at least the patient could be treated if any problems did occur.
- Check the flight path and note if there are any recompression facilities available en route should problems arise.
- Check if there is a hyperbaric facility at the patient's destination or where the nearest facility is to be found.
- Advise a review of the diver by a diving physician as soon as possible following the flight.
- Send a detailed clinical summary with the patient.

My recommendation is to wait four weeks before airflight following decompression illness because:

- there are no published case histories where a relapse has occurred after this time period;
- the pathophysiology associated with DCI may take three to four weeks to abate.

Conclusions

Case histories that show early relapse with or without airflight within five to seven days of treatment would indicate that airflight is not advisable for at least seven days post treatment. No relapses have been reported in divers who waited 35 days. Flight between seven and 35 days has a diminishing risk. However, this risk cannot, at present, be quantified.

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Case report

Prolonged QT syndrome: a probable cause of a drowning death in a recreational scuba diver

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Key words
Diving, scuba, death, prolonged QT syndrome, case reports

Abstract

(Acott CJ. Prolonged QT syndrome: a probable cause of a drowning death in a recreational scuba diver. *SPUMS J*. 2004; 34: 209-13. **Paper presented at the SPUMS ASM in Noumea, 2004**)
This case report concerns the death of a 17-year-old scuba diver that was probably due to prolonged QT syndrome (LQTS). LQTS is a cause of sudden death and may be responsible for drownings for which no obvious reason can be found from post mortem or the history of events. Some aspects of LQTS are reviewed and its potential contribution to diving fatalities considered. Consideration should be given to genetic screening of young victims for LQTS in cases of 'in water' sudden death.

Introduction

Unexplained loss of consciousness in young adolescent and adult swimmers and divers is uncommon but well documented anecdotally.[1] This case report concerns the death, which was probably associated with prolonged QT syndrome (LQTS), of a 17-year-old, male, recreational scuba diver. His death was one of a cluster of nine diving-related deaths recorded in South Australia in a twenty-month period (2001–2002).[2] Other deaths from this unfortunate series have been reported previously.[3]

Case report

The deceased, a newly qualified diver, and his father were diving for crayfish. The weather was warm, surface sea conditions calm, and underwater visibility was good with no current. The diving depth was to 7 metres for 30 minutes. The divers were separated for about a minute. The deceased's father found him lying but not wedged under a ledge; he was unresponsive to his father tugging on his legs. An absence of stirred sand causing decreased visibility indicated that there had not been a struggle or perhaps a grand mal convulsion. His rescue was uneventful. His regulator was out of his mouth when found and was not replaced during ascent. Cardiopulmonary resuscitation (CPR) was commenced once he had been retrieved to the boat (of the two rescuers only one had done a basic CPR course). He was taken to the local hospital and a medical retrieval team from the Royal Adelaide Hospital (RAH) was summoned. He was intubated and ventilated (Oxylog ventilator, 100% oxygen [F_iO_2 = 1.0] with a positive end expiratory pressure [PEEP] of 5 cm water) during transport.

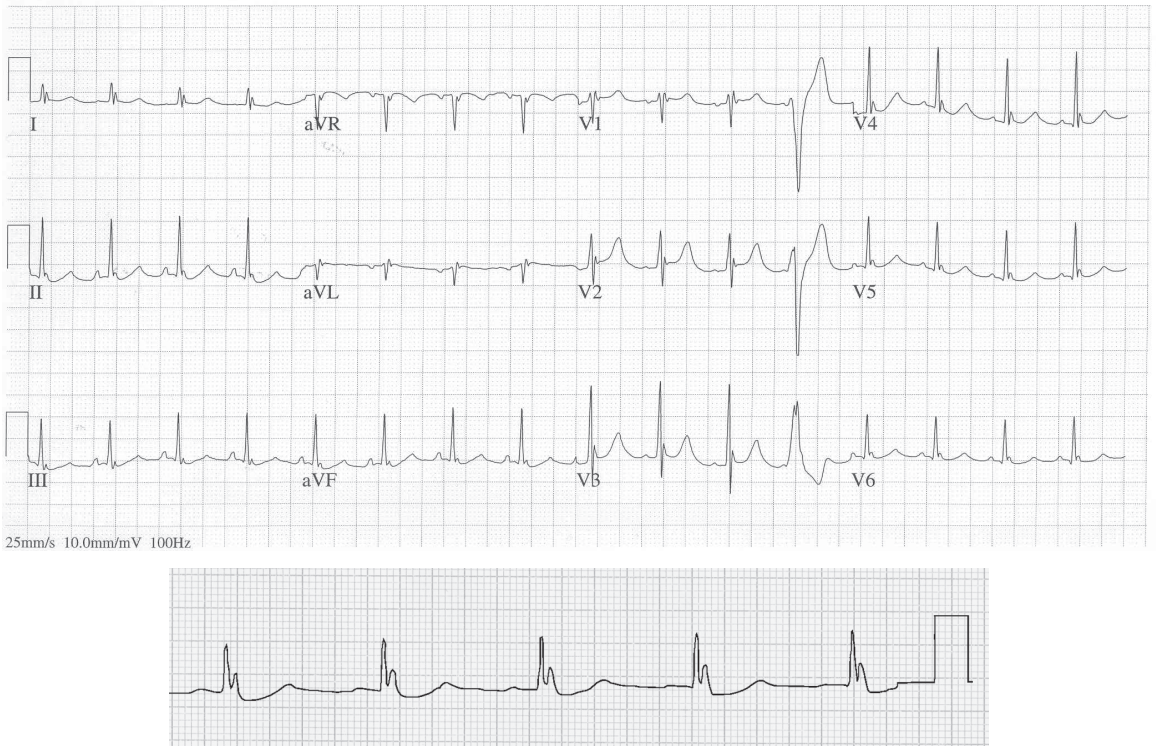
On arrival at the RAH, his Glasgow Coma Scale score was noted to be three, he was tachycardiac but in sinus rhythm, and his blood pressure was normal. He had not received any medication. He was normothermic on arrival.

His initial arterial blood gases showed an increased A-a gradient (F_iO_2 = 1.0), no hypoxaemia but a mixed acidosis, the transport ventilator proving to be inadequate against non-compliant oedematous lungs. Initial serum biochemistry was abnormal and he was haemoconcentrated (Table 1). A chest X-ray showed pulmonary oedema and

Table 1
Relevant investigations on admission

Arterial blood gas analysis	
F_iO_2	1.0; Oxylog Ventilator; 5 cm PEEP
pH	6.99
pO_2	95 mmHg
pCO_2	80 mmHg
HCO_3	18 mmol.l^{-1}
Biochemistry	
Na^+	157 mmol.l^{-1} (135–148)
K^+	4.8 mmol.l^{-1} (3.5–5.3)
Cl^-	119 mmol.l^{-1} (98–106)
Mg^{++}	3.12 mmol.l^{-1} (0.7–0.95)
Creatinine	0.133 mmol.l^{-1} (0.05–0.12)
Glucose	8.9 mmol.l^{-1} (3.9–6.2)
Anion gap	25.1 mmol.l^{-1} (>18)
CK	9753 µg.l^{-1} (0–270)
CKMb	358 µg.l^{-1} (0–7.0)
CKMb/CK	3.7% (<2.6)
Liver enzymes elevated; normal bilirubin	
Haemoglobin	186 gl^{-1}
Haematocrit	55
Platelet and white cell counts normal	

Figure 1
Twelve-lead electrocardiogram from 17-year-old diver. Also shown is a separate lead II rhythm strip in which the long QT interval is more apparent.



his initial ECG a prolonged QT interval at 483 msec at a pulse rate of 62 min^{-1} (despite the elevated serum Mg^{++}) with an intraventricular conduction defect (Figure 1). During his admission he had several episodes of spontaneous ventricular tachycardia, which spontaneously reverted. These episodes may have been a result of electrolyte abnormalities, cerebral oedema or LQTS. He did not require inotropic support during his resuscitation.

Within 12 hours of admission to the RAH he developed bloody osmotically induced diarrhoea. This is the result of gut ischaemia and is a poor prognostic sign. His pulmonary function improved; the F_iO_2 was decreased because the risk of decompression illness was thought to be negligible from his dive profile.[4] However, his cerebral oedema failed to improve and his intracranial hypertension resulted in 'coning', brain death occurring within 24 hours of admission. At post mortem no cause for his drowning was found. Genetic screening for LQTS was not performed. The cause of death was reported to be due to salt-water drowning, presumably due to an arrhythmia caused by LQTS.

Specific questioning of his family revealed a history of 'drop attacks' witnessed by his uncle but not by his parents. These were not reported to his general practitioner and were not investigated, and, therefore, it is unknown if they were syncopal attacks. He had had a diving medical prior to commencement of his diving course by his local general practitioner. His family have agreed to undergo genetic investigation performed by their local general practitioner; however, these results are not available to the author.

Discussion

Many of the laboratory findings reported in Table 1 are the combined result of salt-water aspiration and severe hypoxaemia. His elevated serum magnesium (Mg^{++}) level, haematocrit and sodium concentration reflect the fluid shifts that occur when large amounts of salt water are aspirated.[5] The decreased bicarbonate concentration and increased anion gap were due to metabolic acidosis, whilst the elevated creatine phosphokinase indicated muscle and myocardial ischaemia. Presumably his elevated liver enzymes indicated a degree of hepatic ischaemia (although at post mortem he was noted to have Wilson's disease). The cause of the victim's pulmonary oedema is obvious, but there may also have been a degree of myocardial failure due to ischaemia and an elevated Mg^{++}-induced myocardial depression.[4] However, inotropic support was not required during resuscitation.

LQTS has been implicated in cases of 'sudden death' in young adults and children and has been overlooked as a cause of drowning.[6–8] A study in Finland by Lunetta et al reported that LQTS was responsible for one out of 165 drownings and suggested that this may be one cause of 'dry drownings'.[8] Other cardiac causes for sudden death are listed in Table 2.

LQTS is an arrhythmogenic cardiovascular disorder resulting from mutations in the cardiac ion channels. Persons with LQTS have a disorder of cardiac electrical activity causing a delay of repolarisation. The clinical

Table 2
Some causes of sudden death in adults, age <35 years (modified from Hockings[7])

Cardiac
- Hypertrophic cardiomyopathy
- Dilated cardiomyopathy
- Ventricular pre-excitation syndromes (Wolff-Parkinson-White syndrome)
- Coronary artery anomalies (particularly the left coronary artery from the non-coronary sinus of the aortic root)
- Arrhythmogenic right ventricular dysplasia
- Long QT syndrome
- Myocarditis
- Aortic valve stenosis (associated with congenital bicuspid valve)
- Aortic dissection (Marfan's syndrome)
- Left ventricular hypertrophy
- Coronary artery disease – myocardial infarction
- Concussion of the heart (commotio cordis) due to an accidental precordial thump
- Brugada syndrome (idiopathic ST-segment elevation) – electrical familial disease caused by a genetic ion-channel defect

Other
- Drug-induced
- Intracranial events
- Asthma

features of LQTS result from an episodic ventricular tachyarrhythmia called 'torsades de pointes' (twisting of the points – the QRS axis). Traditionally it has been classified as either inherited – the Romano Ward syndrome – or acquired.[6,7] The inherited disorder is autosomal dominant; hence, if either parent has LQTS then the child has a 50% chance of inheriting the disorder. Synchronised ion movement into and out of cells (potassium, calcium and sodium) produces cardiac electrical activity. These ion channels are genetically encoded with at least six genotypes, and three genes specific for LQTS have now been isolated.[7,9] In addition, it is thought that the acquired variety may have a silent mutation of one of these six genotypes and will remain asymptomatic until exposed to certain conditions, for example hypokalaemia or drugs.[7]

LQTS occurs rarely, 1 in 20,000, and is characterised by sudden death, recurrent syncope and seizures. Indeed, some patients have been diagnosed as epileptic.[10] It can present at any age. Often the first manifestation is syncope or cardiac arrest precipitated by emotional or physical stress or loud noises.[6,7,10] Overall, the world data suggest that 40% of patients are asymptomatic at the time of diagnosis. Weintraub et al published a series of 23 Australian children with congenital LQTS.[11] The median age at time of referral was 10 years (range 4 days to 19 years); 14 (61%) had a family history of the syndrome, and 19 (82%) were symptomatic at the time of diagnosis. Syncope was the main presenting symptom (69%), then aborted sudden death (26%) and near drowning (5%).[11]

Table 3
Diagnostic criteria in LTQS (from Booker et al[6]). A score >4 is considered diagnostic

ECG	Points
QTc:	
>480 ms	3
460–470 ms	2
450 ms (males)	1
Torsades de pointes	2
T-wave alternans	1
Notched T wave in 3 leads	1
Low heart rate for age	0.5
Clinical history	
Syncope with stress	2
Syncope without stress	1
Congenital deafness	0.5
Family history	
Family member:	
with LQTS	1
unexplained sudden death <30 yrs	0.5

The QT interval varies with heart rate, and because of this variation the QT interval is corrected for rate and presented as QTc. A QTc interval greater than 440 msec is considered prolonged. Lead II is the best lead in which to calculate it. Diagnosing LQTS in patients is difficult – up to 12% of affected individuals may have a normal ECG.[7] However, a clinical scoring system is used (Table 3) and a score >4 is diagnostic of LQTS.[6] Using this scoring system the deceased had a score of 3–5 (2–3 for ECG findings and either 1 or 2 for syncopal episodes – it is unknown whether these were related to stress, see later).

Specific triggers for arrhythmia in LQTS vary according to the genotype involved and are exercise, emotional stress, loud noises, rest, sleep, and neurosensorial stimulation.[6,7,10]

Table 4
Factors associated with diving that may trigger the prolonged QT syndrome

Swimming
Facial immersion
Drugs (Table 5)
Valsalva manoeuvre (particularly if fully β-blocked)
Adrenergic sympathetic stimulation:
    anxiety, physical and emotional stress
    hypoxia
Hypothermia
Sinus bradycardia
Hypokalaemia
Hypocarbia
Sudden auditory stimulation

Table 5
Drugs that need to be avoided in patients with the prolonged QT syndrome, are associated with torsades de pointes (TDP), or have been associated with a prolonged QT interval but lack substantial evidence for causing torsades de pointes[6,7,15]

Type of drug	Avoid in prolonged QT interval	Associated with TDP in clinical dose	Associated with TDP in higher dose	Possibly associated with prolonged QT interval
Anti-arrhythmic				
Class Ia		Disopyramide Quinidine Procainamide		
Class Ib			Mexilente	
Class Ic				Flecainide
Class III		Sotalol Amiodarone		
Antimalarials		Chloroquine Quinine Halofantrine		Mefloquine
Antihistamines				Terfenadine Astemizole
Prokinetic agents		Cisapride		
SSRIs			Fluoxetine Paroxetine Sertraline	
Antibiotics	Azithromycin Clarithromycin	Erythromycin	Trimethoprim Sulfamethoxazole Ampicillin	
Tricyclic antidepressants			All	
Antipsychotics		Thioridazine Haloperidol Chlorpromazine Pimozide	Lithium	
Bronchodilators	Salbutamol Most bronchodilators			
Adrenergic drugs	All except Dobutamine	Dobutamine		
Miscellaneous	Cocaine	Droperidol Methadone Some appetite suppressants	Chloral hydrate	Vasopressin

In a retrospective study by Ackerman et al of 35 cases of congenital LQTS, six (17%) had a personal history or family history of near drowning or drowning.[11] These investigators also concluded that swimming was a specific arrhythmogenic trigger for genetic-specific LQTS, but offered no explanation as to why. Another study of children with LQTS by Yoshinaga et al showed that face immersion in cold water induced T-wave alternans or a notched T wave.[12] Therefore, several factors associated with recreational diving may be arrhythmogenic triggers (Table 4). Certain drugs are associated with LQTS and torsades de pointes and are listed in Table 5. Diving medical practitioners need to be aware of these.

The treatment options of LQTS include high-dose β blockers, pacemaker, automatic implantable defibrillators (ICD) and specific gene therapy.[6,7,10] However, 25–35% of patients with LQTS on high-dose β-blocker therapy are

likely to have another syncopal attack within five years.[13] A recent case report showed that, even with an ICD, syncopal attacks while swimming are not prevented. This is not surprising because an ICD requires a short time to 'diagnose' the arrhythmia and deliver a counter shock if required, so there may be a brief syncopal episode. Hence, an implantable ICD is a contraindication to scuba diving and swimming. Besides the problems associated with an ICD, swimming appears to be a specific trigger for arrhythmias in LQTS; the reason for this is unknown but it could be due to intense sympathetic stimulation due to physical activity or a high level of anxiety. Swimming or scuba diving while on therapy should, therefore, be avoided.[1,12–14]

The deceased had a history of 'drop attacks', which may have been syncopal episodes. These episodes were not reported to his local general practitioner and hence not investigated. It is not known if his diving medical had included an ECG, nor is it certain that the significance of a prolonged QTc interval would have been recognised and acted upon; without the history of drop attacks he would have scored 3 at best (Table 4). However, a normal QTc interval on an ECG does not exclude LQTS. Up to 12% of these individuals have been reported to have a normal ECG.[7] The author believes the importance of an ECG is understated in diving medical assessment. In his recent practice, five cases of Wolff-Parkinson-White syndrome were diagnosed in 80 consecutive diving medicals performed over a two-year period. Any history of 'fainting episodes' should be investigated with a careful medical and family history, particularly any family history regarding sudden or unexplained deaths in family members less than 30 years old. If there is any doubt, echocardiography, 24-hour Holter monitoring, exercise testing, facial water immersion with ECG monitoring, and genetic screening should be performed.

This case illustrates five points:

- An elevated serum Mg^{++} is indicative of salt-water drowning or near drowning.[5]
- The clinical observation that bloody diarrhoea is a poor prognostic sign following cardiac arrest and resuscitation is widely recognised.
- Specific questioning regarding syncopal or 'drop' attacks must be included in a diving medical and the cause for any of these attacks must be determined before the risks associated with diving can be assessed.
- 'Bystander CPR', or 'just doing something', may be effective.
- All investigations of 'in-water sudden deaths' must include a thorough family and medical history and perhaps genetic screening for genetic disorders associated with 'sudden death' if the family and medical history warrant it.

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The world as it might be

Intermittent exposure to 152 – 405 kPa breathing hyperbaric air treats *Palinuridae spp.* vitamin deficiency

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Introduction

The vitamin *Palinuridae spp.* is available to the human population on a worldwide basis. Regarded by some learned authorities as a non-essential vitamin, difficulties with its harvesting and processing have led to deficiencies in many non-endemic areas. *Palinuridae spp.* deficiency has been recorded in the island State of Tasmania, because the vitamin is found in Tasmanian sea water. There are many methods of harvesting and producing the vitamin; however, drinking large quantities (of sea water) has not corrected the deficiency, and may result in significant morbidity (Mitchell S, Bennett M, Wilkinson D, personal communication, undated conference proceedings).

The null hypothesis is that *Palinuridae spp.* deficiency results in a reduction in quality of life. It is proposed that intermittent exposure to 152 to 405 kPa breathing hyperbaric air treats *Palinuridae spp.* deficiency and results in improvement in quality of life.

The aims of this research were:

- to investigate methods of correcting *Palinuridae spp.* deficiency by intermittent exposure to hyperbaric air;
- to examine the effects of correcting this deficiency using previously validated quality-of-life measurements.

Methods

A systematic search was made of all available sources of data pertaining to *Palinuridae spp*. Paediatric data were included in the search (Figure 1). Sources were personally inspected by the authors, who undertook a detailed examination. This included examination of hard-to-find literature, hidden in dark places (also known as 'grey literature') (Figure 2).

Figure 1. Paediatric search strategy

Figure 2. Dark place search

Results

Available data confirm that it was possible to correct *Palinuridae spp.* deficiency by intermittent exposure to 152 to 405 kPa hyperbaric air. Hyperbaric air proved to be a co-factor in correction of the vitamin deficiency.

Quality of life (QoL) as measured by width of teeth relative to nares, and curling up on outer edges of lips is apparent after correcting the vitamin deficiency. This is illustrated in Figures 3 and 4. An unexpected side-effect in individuals with severe *Palinuridae spp.* deficiency was the desire to eat the vitamin raw in the aquatic environment. This high-risk practice warrants further study (Figure 4).

Figure 3. Improved QoL

Figure 4. Severe *Palinuridae spp.* deficiency

Conclusions

Palinuridae spp. deficiency can be corrected by intermittent exposure to hyperbaric air at pressures of 152 to 405 kPa. Quality of life was improved by correcting the deficiency. A rare but potentially life-threatening side-effect identified by the study was the finding of individuals so deficient in the vitamin that they were prepared to consume the raw, unprocessed product. Further field research is planned to identify this subset with severe *Palinuridae spp.* deficiency.

A review of recent advances in technical drinking and the treatment of technical or extreme drinkers

Colin M Wilson, Martin DJ Sayer, A Gordon Murchison and John AS Ross

Abstract

(Wilson CM, Sayer MDJ, Murchison AG, Ross JAS. A review of recent advances in technical drinking and the treatment of technical or extreme drinkers. *SPUMS J*. 2004; 34: 215-7.)
The likelihood of contracting DCI or DCS (drink clearance illness or drink clearance sickness) is known to be higher in drinkers who use SCUBA (self-contained undiluted bottled alcohol). In recent years, there has been a growing body of drinkers who employ advanced drinking techniques in order to prolong drinking immersions without the concomitant increase of risk of DCI/DCS. Commonly termed 'technical drinking', this can take the forms of trimix, saturation drinking, use of re-drinkers, or extreme drinking. Recent advances in these areas of technical drinking are reviewed along with possible treatment protocols for drinkers who do suffer the consequences of DCI/DCS following technical drinking.

Introduction and background

For many years, an increasing proportion of recreational drinkers have suffered from the effects of DCI or DCS (drink clearance illness or drink clearance sickness). Commonly termed 'the staggers', the symptoms of DCI/DCS are often separated into type 1 (simple, pain only; cranial and/or wrist and elbow, right more commonly than left) or type 2 (serious, neurological) effects. The most noticeable of neurological signs and symptoms of type 2 are sweating, tremor, reduction in cerebral function, hallucinations and convulsions. Occasionally the victim presents with vestibular DCI/DCS, 'the staggers', which is characterised by dizziness, nystagmus, difficulty in walking and a tendency to fall over. These symptoms may be indicative of hepatopathic post-potency neurological syndrome (HPNS). Associated with type 1 and type 2 cases of DCI/DCS are problems of toxicity, narcosis, decompression, limb bends and embolism.

The increasing incidence of DCI/DCS has often been related to the wider use of SCUBA (self-contained undiluted bottled alcohol) drinking. A single SCUBA drinking event is typified by high, rapid rates of alcohol on-loading into the blood stream over periods of two to four hours, followed by a gradual reduction and finally a rapid cessation of uptake. This typically skewed drink profile, commonly-termed 'bounce drinking', very rarely produces incidences of DCI/DCS as long as the depth and duration of uptake is conducted around tables.

Incidences of DCI/DCS have been recorded where the depth and duration of single bounces are excessive (extended-range drinking), or where multiple bounce-drinking episodes occur over a number of days (multi-day drinking) or repeatedly within a 24- to 36-hour period (repetitive drinking). Often the victims end up on or under the tables and their drinking is typified by aggressive profiles.

Professional drinkers recognise that significant drink exposures can be managed within low but accepted rates of DCI/DCS in a variety of ways, e.g., for the drinker to take on mixtures of relatively volatile substances (RVS) over extended periods. This procedure usually involves the use of three RVS and has been termed 'trimix drinking'. Although trimix drinking permits extension of depths and durations of drinking sessions it requires higher levels of drinking discipline and training to be able to execute the changes in mixtures when the drinker is subjected to probable signs of narcosis.

The logical extension to trimix drinking is 'saturation drinking' where, in a properly managed environment, the drinker can continue to drink for periods of many days as long as the alcohol attains complete saturation of the bloodstream and is not permitted to drop in level. However, when the saturation drinker does have to surface this requires a well-managed and very slow withdrawal protocol. In all cases of deep or technical drinking, the drinker should always be 'drink fit'. This involves undertaking a number of preliminary drinks, sometimes referred to as 'work-up' drinks, whereby the drinker drinks to levels close to or exceeding the depths of the subsequent technical-drinking session. There is anecdotal evidence to suggest that fully worked-up drinkers are less prone to hits; less worked-up drinkers are often just prone.

In recent years, the recreational-drinking industry has started to adapt advanced drinking techniques that were first developed for professional drinkers. One example of this is the rapid development of the use of re-drinkers by recreational drinkers. Covert military drinkers have long used re-drinkers to disguise active drinking programmes. Originally developed during the Second World War, the design for the covertly drinking beer apparatus (CDBA) has recently been adapted for recreational use. Of more concern is the gradual development of 'extreme drinking', sometimes incorrectly termed 'free drinking', whereby drinkers descend rapidly to great depths using RVS of extremely high alcohol content.

This review summarises recent advances in a selection of forms of technical drinking. However, the rates of avoiding DCI/DCS are not 100% and technical drinkers may require advanced forms of treatment. Therefore in addition, this account details current practices in treatments for technical drinkers. A more extensive review is available from the authors on request to the e-mail address provided.

Trimix drinking

The mixing of forms and contents of RVS has long been known to prolong the depth and duration of the drinking excursion. However, improper or reverse profile mixing can cause problems. The UK Navy rule, derived in the 1960s, refers to simple two-RVS mixtures.

Beer and wine and you'll feel fine
Wine and beer and you'll feel queer

recognised the dangers of getting the mixture order wrong.

Trimix is a combination of three forms of RVS in varied proportions. The ranges found in typical trimix drinking regimes are given in Table 1. Trimix drinking requires prior training and a certain degree of discipline in order to be executed safely. Drinkers undertaking trimix drinking must adhere to alcohol toxicity dose (ATD) guidelines. The ATD value is a factor of percentage alcohol and time, and a general rule is that the higher the percentage the shorter time that can be spent drinking. However, ATD rules can be further complicated by 'bar pressure' that can result in ATD levels being exceeded. Many trimix drinkers, therefore, tend to 'solo' drink to avoid bar pressure but this can bring its own risks if excessive levels of narcosis are experienced. Having a buddy drinker present is recommended to ensure that mix changes occur smoothly. Often mix changes occur when the drinker moves between bars and in such cases a 'travel' mixture can aid prolonged movement times.

Saturation drinking

Although mixed drinking techniques allow for significant increases in depth and duration of drinking, the drinker still has to surface in order to reduce the threat of DCI/DCS. In the 1970s, professional drinkers in Scotland developed techniques that permitted prolonged submersion for many days by allowing the bloodstream to become saturated with RVS. The development of saturation drinking coincided with the discovery of large deposits of oil. The oil was initially recovered through adherence onto a range of organic transfer substrates that were submerged into the deep hot-oil reserves but was later also discovered in veins. Saturation drinking permitted the recovery of oil reserves for many years.

A typical saturation drinking event starts in the same way as most trimix drinking immersions (Table 1). Once fully immersed the drinker has to carefully enter the saturated state. This is achieved through continual intake of RVS but at levels that do not exceed permitted ATD rates (see trimix section). Typically a period of three hours of RVS intake, with at least two RVS 'breaks' every hour, is followed by a maximum of two hours with no RVS intake; any more than two hours and the drinker can come out of saturation.

Surfacing from a saturation drinking session must be managed with extreme care; the surfacing schedule is a routine 36 hours and 4 minutes irrespective of the time spent in saturation as the level of saturation will always be the same. This is effectively an inverse exponential relationship where alcohol intake is reduced rapidly at first but then there is a gradual decline in the rate of surfacing, during which RVS mixtures may need altering. At all times in the surfacing schedule, the drinker intersperses periods of RVS intake with drinking increasing volumes of inert solutions. The schedule is also dependent on maintaining hydration levels.

Re-drinkers

Re-drinker technology is based around the fact that the drinker's body does not use alcohol efficiently and so a proportion of the on-loaded alcohol will always be excreted in the urine. Re-drinkers are twin-hosed devices (the original CDBA design was single hosed, but pendulum drinking has been found to be less efficient than twin-hose drinking) that pass the drinker's urine through a filter (a canister containing soda and lime; sometimes referred to as sodalime) to leave only the alcoholic content of the solution (technically termed low alcohol gain expelled regularly, or LAGER) and then remix the filtered alcohol with a continuous inflow of high content alcohol. In the basic models of re-drinkers, the inflow mixture is pre-mixed and the flow set depending on the alcohol content of the mixture. More modern re-drinkers have separate alcohol and mixer bottles permitting the drinker to alter the alcohol content of the mixture depending on the drinking profile. One side-effect of re-drinkers is that the chemical reaction in the filters can warm the resultant fluid. This is not a

Table 1
Typical limit ranges for trimix drinking regimes. The order of uptake must be strictly adhered to

Order of RVS uptake	Common name	Alcohol (%)	Flow rates (ml.hr^{-1})	Duration of flow (hrs)
1	Hop extract	3.8 – 5.2	1500 – 2000	1.5 – 3.0
2	Grape extract	11.5 – 14.5	300 – 700	1.0 – 2.0
3	Grain distillate	40.0 – 46.5	50 –120	Until early hours

problem in countries where alcohol is consumed warm.

Extreme drinking

A very recent development in technical drinking is extreme drinking. This is based on obtaining the maximum depths of immersion in the quickest time. At the start of an excursion, extreme drinkers usually on-load rapidly and repetitively with relatively small volumes of hyper-alcoholic RVS. It is usual not to mix the form of RVS, but percentage content of the fluids usually has to exceed 50%. Up until the early 1970s the navy employed navy reality undermining mixtures (Navy RUM at 54.5%) in extreme drinking trials. The effects of extreme drinking are typified by the rapid onset of narcosis on descent and symptoms similar to DCI/DCS on ascent.

Extreme drinking has also been termed 'free drinking'. This can occur if the RVS are supplied by other drinkers and regular competitions used to be staged to determine the maximum depths that could be achieved with no means of support. There are extreme dangers of personal injury to the free drinker if they rely on other drinkers over prolonged periods of time and in some cases the rapid increase in bar pressure associated with free drinking can be extremely aggressive. Extreme periods of free drinking can result in a series of 'hits'. Many extreme drinkers actively dissociate themselves from the free-drinking faction and the inter-use of the terms extreme and free drinking is, in fact, incorrect.

Treatment protocols for technical drinkers

It is debatable whether the treatment of DCI/DCS in technical drinkers should vary from that for illness caused by standard drinking protocols. However, immediate recompression is known for its benefits and requires returning the sufferer to a depth of intoxication to relieve symptoms.

Whether the DCI is type 1 or type 2, the non-recompression treatment is administered using a drink-denial chamber (DDC). The chamber must be staffed with adequately trained and equipped personnel, and HDU (heavy drinking unit) or, preferably, ITU (intensive tequila unit) facilities must be available. Rehydration and the correction of hypovolaemia are important aspects of therapy. The DDC is not all it seems, as treatment is usually between two or three bars. During recovery an excellent time can be had in a good atmosphere, usually about eight tenths of the way between the second and third bar.

Discussion and summary

The present account outlines recent advances in technical drinking. Although there are obvious benefits in terms of depth and duration of immersion, technical drinking requires more discipline and planning than standard drinking, and can be time consuming if the drinker enters into saturation. The cumulative effects of bar pressure cannot be understated. Technical drinking can also be costly unless the individual is sponsored or is persuaded to undertake very dangerous forms of free drinking. Because the risks associated with these more advanced forms of drinking are higher, drinking with experienced buddies is advisable and many technical drinking events now insist on having medical personnel present.

Colin Wilson and his colleagues are (ir)responsible for the care or otherwise of divers at the Dunstaffnage Marine Laboratory and Hyperbaric Centre, near Oban, Scotland, whilst John Ross is a consultant in Environmental and Occupational Medicine at the University of Aberdeen, who should know better. They wish everyone a grand wee Hogmanay.
E-mail: *<colinwilson@tiscali.co.uk>*

The Poetry Doctor

John Parker

Twilight Diving

I've dived for many decades.
It used to be a game.
But now I'm getting older
Things are not the same.

For hours on end I'd free dive
To thirty metres' depth.
Now I float the surface
For I can hardly hold my breath.

I used to suck a twin set dry
That I threw over my head.
Now I hardly use one tank
That my buddy lifts instead.

I dived in rip and surf and swell.
I'd fin where I would go.
Now I only dive the calm
Or gently drift the flow.

I'd be first in and out the last.
To the deepest depth I'd drop.
Now I'm in last, first one out,
Including safety stop.

I might now seem shallow and slow
But at least I did survive.
It's nice to have no more to prove
I just enjoy to dive!

<www.thepoetrydoctor.com>

Opinion

South Australian diving-related deaths 2001–2: an alternative view with discussion of responsibilities of an 'expert witness'

Douglas Walker

Key words
Diving deaths, investigation, expert witness, theory-based advice

Abstract

(Walker D. South Australian diving-related deaths 2001–2: an alternative view with discussion of responsibilities of an 'expert witness'. *SPUMS J*. 2004; 34: 218-20.)
The reports by the State Coroner of South Australia on five diving-related deaths that occurred during 2001-2002 are discussed. Non-medical factors are proposed as having been critical to the course of these incidents. The difference between the role of an 'expert witness' and that of an 'advisor to the coroner' is discussed.

Introduction

There has been international legal, media and political interest in the findings of South Australia's State Coroner concerning the critical factors incriminated in several diving-related deaths that occurred in the period 2001–2002.[1] A recent medical report has provided a rare opportunity to observe the involvement of an 'expert witness' in advising a coroner on the interpretation of the facts presented and therefore in his reaching his formal findings and recommendations.[2] This paper reviews the information the Coroner has made available concerning these cases, summarising relevant issues briefly, and reaches a different conclusion from that of Acott.[2] Attention is drawn to the importance of those advising any coroner to be careful to avoid proposing as facts matters that are opinions, because of the consequences which may flow from the coroner's reliance on the advice.

Case reports

CASE 1

This diver had recently undertaken a refresher course. Although described by her son as being very experienced, the dive supervisor appointed a diving instructor to be her buddy. When she became aware her contents gauge showed it was appropriate for her to ascend her buddy persuaded her to make a 20.7-metre solo ascent while the buddy continued to hunt for crayfish. A cry for help was heard by the 'surface cover' but ignored. Clinically, this was a case of cerebral arterial gas embolism, though no autopsy evidence was found. The medical history, not divulged in a diveshop questionnaire, included a spontaneous pneumothorax with unilateral pleural ablation treatment, chest infections that were just possibly asthma and pneumonia but probably not, two occasions of seeking advice about equalising her ears, and pains following a gynaecological operation. It was put to the Coroner that vertigo during ascent and the pleural adhesions were critical. No supporting facts were presented. The critical factors were her belief that by her remaining-air status she needed to ascend, and being asked to do this solo.

CASE 2

An infrequent diver, this obese man was wearing a tight wetsuit and using borrowed equipment. There is no mention in the available records whether he appeared to be unfit. One of his two buddies accompanied him to the surface when he was becoming low on air, holding onto his vest to prevent a too rapid ascent although it was not rapid, then descended again to continue hunting for crayfish. When left at the surface he had a partially inflated buoyancy compensator (BCD) and had been advised to fin back to the dive boat, face up. He was later found floating, dead, with a drum-tight BCD. The autopsy showed the presence of air embolism, pleural adhesions, hypertensive cardiomegaly, and body mass index of 32 $kg.m^{-2}$. The pleural adhesions were ascribed to a past road traffic accident. It was suggested that he possibly suffered gastric reflux and was suffering from 'dive dehydration' causing blood thickening which interfered with oxygen uptake. The pleural adhesions were described as a reason for the pulmonary barotrauma. There is no evidence for these suppositions. His cardiovascular fitness was certainly suspect but there was no evidence presented concerning his apparent fitness. The buddy's action in restraining him during the ascent may have distracted him from correct breathing rhythm, while his tight wetsuit disadvantaged him further. The over-inflation of his BCD did not cause his air embolism and was probably a terminal event.

CASE 3

After an unhurried ascent with his buddy, this hookah diver began a surface swim to their dive boat. He made no call

for help before silently sinking to the sea floor. There is no information concerning his health history other than his son's comment that "*he had been diving most of his life and never had any problems*". However, he was obese, had a severely fatty liver, emphysematous bullae, and minor myocardial ischaemic changes. He was wearing a wetsuit so tight it required significant effort both to don and remove. His weight belt not only carried excessive weights, 20 kg, but was unditchable because it had shoulder straps that were under his BCD. He had not inflated his BCD. The cause of death was drowning, circumstances pointing to this having followed a sudden fatal cardiac arrhythmia. There is nothing to indicate his health this day was any different to that on his many previous dives. He must have been aware of his limited effort tolerance.

CASE 4

This woman was regarded by her buddy as "*a not overly confident diver due to being unfit and overweight, which restricted her mobility*". During the snorkel swim in calm water to the dive area she requested to make several rests. Believing herself to be underweighted, she took an additional weight from her buddy. The final weight she carried, 18 kg, was considered excessive for her. After a short period diving she indicated a need to surface to rest on some nearby rocks. The two divers were washed off the rocks by an unexpected wave and she was repeatedly submerged by following waves, losing the regulator from her mouth. She drowned despite the attempts of her buddy to save her. She failed to inflate her BCD or ditch her weight belt; lethal errors. The coroner was advised that oesophageal reflux may have occurred causing laryngeal spasm, or that she was sedated by codeine taken for back pain, although the toxicology tests showed no codeine. She was a thoroughly unfit person but died because she failed to respond correctly to the unexpected immersion, her inexperience and carrying of excess weights, which she did not ditch, being critical factors.

CASE 5

This diver had been advised by his cardiologist that he should not dive, a fact which he made known to his dive companions. This was to be a deep dive, 37 msw depth, to recover an anchor and a sunken dinghy. He stated his intent to make his dive short, in partial acceptance of the medical advice. When he became separated from the others on the sea floor by a silt out from disturbed sand, he failed to ascend. He met the next pair of divers and snatched the regulator from the first, who snatched his buddy's, who fortunately then used her octopus regulator. They then started a rapid ascent in daisy-chain formation. After about seven metres' ascent the regulator he had snatched was noticed hanging loose, now minus its rubber mouthpiece. It is not known whether he resumed use of his own regulator or made an out-of-air 30 metres' ascent. His original buddy decided to descend again to make a deco stop and there found his friend floating, dead. Although he was obese and had cardiomyopathy with a past history of cardiac failure, the critical factor was allowing himself to become low on air rather than ascending as would be indicated after separation. Wearing a tight wetsuit jacket was unhelpful.

Discussion

The Coroner correctly noted that in all five cases there were medical factors that would have been identifiable at a medical examination had a completely honest medical history been provided. However, it does not necessarily follow that these conditions were the critical factors that decided the fatal outcomes of these dives. It is suggested that it was the actions of the deceased, and their fellow divers in some cases, which were critical, as evidenced by their having safely dived on previous occasions with these health problems and by analysis of the dive details.[1,2] It is clear that in cases 3, 4 and 5 the divers were well aware of their adverse health factors, which did not require identification by a dive fitness medical, but chose to scuba dive. Diver 1 would have considered herself fit, while diver 2 was a large, obese man who would have been aware that his tight wetsuit was clearly unhelpful to him, but would not have considered himself at risk of sudden cardiac death. A medical check would most likely have resulted in a prescription of medication rather than imposition of a severe restriction to his activities. Unfortunately there is no information concerning his effort tolerance.

The role of an expert witness in a court of law may be as either an advocate for one participant's case, or as an impartial and dispassionate advisor to the court on agreed facts. In the former he can be questioned concerning his statements, in the latter there is no such safeguard. Therefore, it is important that an expert witness makes it clear when he is presenting a personal opinion rather than undisputed fact. It is clear from the published findings that the Coroner based his findings on the medical advice provided and gave less weight to the details of the incidents.

It is informative that Acott introduces his paper with the statement "*five of these deaths were associated with medical conditions that were incompatible with safe diving, such as a body mass index of greater that 30 kg.m^{-2}, cardiomyopathy, asthma, lung bullae, pleural adhesions, poor physical fitness, and controlled cardiac failure*".[2] Certainly, these conditions are adverse to physical activities such as scuba diving, but reference to the literature confirms that many active divers have adverse health conditions.[3]

A belief in the reliability of medical examinations may be muted by consideration of the case where at least two formal examinations and a (misread) chest X-ray in a military diver failed to diagnose a chronic pneumothorax.[4] He had continued active diving despite this condition. In the United Kingdom there is an appeal procedure available to those refused a medical certificate of fitness to dive (UK Sport Diving Medical Committee) and it is clear they have a more liberal attitude to such decisions despite being subject to

the same level of legal risk as in Australia if they are proved wrong.[5] It is probable that the advice given the Coroner concerning the influence of the health factors was weighted by his expert advisor's above-stated beliefs.

The Coroner, in apparent deference to medical advice, gave low significance to the factors of inexperience, tight wetsuits, inadequate training, failure to monitor air supply, incorrect weighting, failure to ditch weight belts, and the failure of an instructor buddy to follow correct procedure. The Coroner made no mention of these factors in his well-publicised recommendations on these cases, restricting himself to discussion of the medical findings.

Opinion

It is important when providing considered advice concerning the apparently critical factors in a diving-related fatality that there should be attention to all the details of the incident rather than too great a focus on factors that may be considered adverse but are not demonstrably involved in the case under discussion. It is salutary to remember a comment attributed to Samuel Johnson that

> "*it is incident to physicians, I am afraid, beyond all other men, to mistake subsequence for consequence.*"

Undoubtedly, this warning is applicable to far more than physicians but it is particularly apposite when providing evidence or opinion in a court of law.

It is proposed that in at least four of these fatalities the critical element lay in the actions of those involved rather than the medical conditions found to be present, and that the divers involved were not in ignorance of the medical factors recorded. In consequence of the expert advice he was given, the Coroner's recommendations concerning these cases were directed solely to medical factors of lesser significance than dive-management errors.

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Dr Acott replies:

Thank you for allowing me to address Dr Walker's criticisms, which appear, in part, to be based on a 'blinkered' view of events. Accident details available to the primary investigators are often overlooked or their significance dismissed by 'investigators from afar'.

The role of an 'expert witness' in the legal process to either give an opinion or clarify a complex medical situation is clearly defined prior to the commencement of proceedings. Expert opinion can be debated at any stage. The coronial process is not to establish blame but to establish facts. The Coroner's conclusions and recommendations are based on evidence, expert opinion and information gathered during an inquiry. The Coroner's findings in the recent cluster of South Australian diving deaths followed this process. 'Expert witness' opinion is considered in reference to all the other evidence. I am sure that if Dr Walker had been present during the Inquiry he would have found this to be so.

In Case 1 the critical factor was not "*her belief she was required by her remaining air to ascend*"; there was no indication that she was anxious or about to panic. Is Dr Walker suggesting that a solo ascent caused her death? The Diving Incident Monitoring Study has shown that vertigo underwater can be nearly fatal. The combination of a Valsalva and continuing to ascend may have embolic consequences; however, vertigo was not considered critical as Dr Walker claims. The postulated cause(s) of death are clearly stated. The most significant factor in this report was the presence of two dive medical forms – one stating she suffered from 'lung problems' the other one that she did not. This was the basis for the Coroner's recommendation number 3.

In Case 2 it was not suggested by me (read the commentary) that he suffered from gastric reflux or 'dive dehydration'– nor did the Coroner make any reference to this. The postulated mechanism for death is clearly stated. There was no suggestion that an over-inflated BCD had caused an air embolism.

Cases 1 and 2, however, do highlight the problems associated with diving in 'threes' – having too (2) many buddies – someone is always 'solo'!

In Case 4 Dr Walker stated "*she was a thoroughly unfit person but died because she failed to respond correctly to the unexpected immersion, her inexperience and carrying excess weights, which she did not ditch, being critical*

factors." Dr Walker has made this observation (failure to ditch the weight belt) consistently in regard to diving deaths without any explanation of the context of divers' deaths.[1] Caruso has tried to address this problem in his analysis of DAN's database concerning diving deaths:[2] "*People are unable to make critical decisions while hypoxic*". Her buddy noted the deceased to be cyanosed when he reached her. There are many reasons for her becoming hypoxic in addition to her aspiration: obesity, tight wetsuit and immersion would all increase the A-a gradient and an increase in oxygen consumption would be expected due to excessive exercise in an unfit person. She was also the only case noted at post mortem to have food and gastric fluid throughout the laryngotracheobronchial tree. In addition, she was the only one noted to have suffered from gastro-oesophageal reflux (GOR), which can be (and usually is) made worse by immersion. Mendelson noted 60 years ago that aspiration of gastric contents, in particular food, is a terminal event.[3] There is no suggestion in the reports that codeine was involved in the death; it was part of her medical history. She died because immersion plus swallowing water made her GOR worse causing respiratory embarrassment, and because she attempted to do a surface swim of 200 metres that she was unfit to do.

In Case 5 Dr Walker states "*the critical factor was allowing himself to become low on air rather than ascending*". I suggest he re-reads what happened. The deceased, an experienced diver, depleted his air supply within minutes of immersion and grabbed another diver's second stage who in turn had to use the octopus regulator from a third diver. The deceased began to ascend pulling the others with him. He then disappeared. His post-mortem chest X-ray showed pulmonary oedema consistent with left ventricular failure and gas within the heart. The pathophysiological events are discussed in the article so I will not elaborate further.

Bitten tongues noted at post mortem can be and usually are indicative of a convulsion (personal communication, J Caruso, Pathologist, DAN, 2004). A convulsion following ascent would strongly indicate that the diver had suffered from an embolic event. Three of the deceased (Cases 1, 2 and 5) were noted to have a bitten tongue at post mortem. Areas of pulmonary compliance change are associated with embolic events.[4]

I would suggest that no person would consider himself or herself at risk of a sudden cardiac death. People dive with conditions that are considered by some to be contrary to safe practice and while they may dive uneventfully for a while it is possible that interplay of situational factors may result in a fatal outcome. Just a little more exertion on the dive, an episode of aspiration or a more rapid ascent than usual may be all that is required. Accidents are unpredictable and can be the products of unlikely coincidences or errors occurring at an inopportune time when there is no 'system flexibility'.[5] Even trivial errors can have catastrophic results if they occur at the wrong time. The 'system' can be regarded as the 'scene setters' or 'latent errors'.[6,7] Dr Walker's military diver illustrates the importance of Alnutt's 'system flexibility'. A cardiovascularly fit, young, military diver has a greater 'flexibility' compared with an obese, cardiovascularly compromised, middle-aged diver, and military diving differs enormously from recreational diving.

South Australia is unique in its process of investigating diving fatalities. The Coroner insists that all diving deaths are investigated by the police diving unit, that a diving medical representative from the Royal Adelaide Hospital's Diving Hyperbaric Medicine Unit is present at all post mortems and that this representative is familiar with all the known facts immediately prior to attending.

Having a database of over 1500 incidents in the Diving Incident Monitoring Study I am sure that I do not "*mistake subsequence for consequence*". In fact, I am a believer in *mortus vivos docet* (the injured teaches the uninjured). I suggest that Dr Walker re-acquaint himself with the physiology of obesity, hypoxia, immersion and the Frank Starling mechanism.

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Articles reprinted from other journals

Project Dive Exploration 2003

The report, *Decompression illness, diving fatalities and Project Dive Exploration*, DAN's annual review of recreational scuba-diving injuries and fatalities was reviewed in the previous edition of this journal.[1] Project Dive Exploration (PDE) adds 35 pages to this publication. This article attempts to summarise the key features of PDE.[1]

The introduction states that the physiology of decompression sickness (DCS) is still not well understood and PDE addresses this scientific need by creating a database of recreational dives. It is an observational research study that collects and analyses electronic pressure-time exposures from dive computers. It also collects the diver's demographic data, pre-existing medical data and 48-hour report on any medical outcome associated with the pressure exposure. Data have been collected since 1995 with 40% of dives reported in 2001 (the final year of collection analysed), taking the total of dives to 37,572. The major groups of dives were collected from recreational diving professionals in Mexico, live-aboard dive boats, 2001 DAN interns and dives made in Scapa Flow in Scotland.

Nearly 90% of divers contributed only one dive series. Over 90% of series were fewer than six days in duration and consisted of fewer than 19 dives. The median number of series was one, the median number of days – four, the median number of dives – seven, and the median number of dives per day was two. Seventy-five per cent of divers were between 20 and 49 years old with 18% over 50 and 8% under 20; 27% were women. In the groups of divers with higher dive qualifications, there were more men than women. Forty-five per cent of divers had five years of experience or less and 25% had ten years of experience or greater. Men made up a greater proportion of the experienced divers.

Seasonal allergy as a chronic health problem was reported by 13.5% of divers, and 12% had had orthopaedic problems. A surprising 7.5% reported diving with flu or acute infections, while 4% had chronic ear and sinus problems. A past history of asthma was reported by 3.5% of divers, whilst 1% had diabetes. Previous DCS had occurred in 1.4%. Nearly all dives occurred in the ocean from boats and 88% were on air.

Seventy per cent of dives were between 9 and 27 metres sea water (msw) depth with 8% less than 9 msw and 5% greater than 36 msw. More than 50% of divers went deeper than 27 msw and 21% deeper than 36 msw at least once during their dive series. The last day of diving tended to involve shallower depths than earlier days in the series. For dive planning, most divers used a dive computer, 17% followed a dive guide, and 2% consulted dive tables without using a computer or guide. A safety stop was reported in only 20% of all dives. Twelve per cent of dives involved decompression stops and these were mostly at Scapa Flow.

Eighteen per cent of divers flew in commercial airliners within 48 hours of their last dive, and 93% of cases flew at least 20 hours after their last dive. No DCS symptoms were reported by the 515 divers who reported being exposed to altitude after diving.

Of the large subsets of divers, day-boat dive professionals and cold-water wreck divers tended to be younger than live-aboard and shore or day-boat divers. The mean number of

Table 19
Reported problems during dive

Problems	% All	% Liveaboard	% Cold-Water Wreck	% Shore/Day-Boat	% Day-Boat Dive Professionals
Equalization	1.95	3.69	6.01	0.71	0.51
Rapid Ascent	0.84	2.70	4.39	0.08	0.32
Buoyancy	0.29	0.18	0.29	0.58	0.19
Vertigo	0.19			0.66	
Out of Air	0.05	0.03	0.04	0.11	0.06
Missed Deco Stop	0.04				0.10
Shared Air	0.03			0.03	0.04
Seasickness	0.03	0.10	0.17		
N Dives	15,385	3,928	2,413	3,796	5,248
% With Problems	4.14	3.59	13.22	2.58	1.70
Total	637	141	319	98	89

Table 20
Reported equipment problems

Equipment Problems	Frequency	% (of all)	% (of reported)
Mask	43	0.28	0.37
Fins	29	0.19	0.25
Weight Belt	33	0.21	0.28
BC	29	0.19	0.25
Thermal Protection	24	0.16	0.20
Computer	17	0.11	0.14
Depth Gauge	4	0.03	0.03
Pressure Gauge	2	0.01	0.02
Regulator/Breathing Apparatus	45	0.29	0.38
None	11,541	75.01	98.08
Subtotal	11,767	76.48	100
Not Reported	3,618	23.52	
Total	15,385	100	

years diving did not differ much among the four groups. The cold-water wreck divers and live-aboard groups tended to participate in PDE only once. The average depth of the Scapa Flow dives was deepest with half of the dives greater than 27 msw. Eleven groups did occasional deep dives to greater than 60 msw.

Problems occurred in more than 4% of dives. Some of these are detailed in Tables 19, 20, 23 and 24 reproduced here.

The incidence of DCS was twice as high in divers with dive master or instructor certification than divers with basic or advanced open water certification. However, the rates per 10,000 dives were nearly the same, suggesting that the incidence is largely explained by a larger numbers of dives per diver.

The report compares the DAN injury population, the fatality population and the PDE group. The average age of fatalities was 43 years, average age of injuries – 40, and PDE divers – 37. Females were injured in slighter greater proportion than males, but males contributed to 85% of fatalities. Fatalities had pre-existing medical conditions of high blood pressure, heart disease, diabetes and smoking more frequently than injuries or the whole PDE population. Students and technical divers were more likely to be fatalities than the PDE population. Diving in a lake, quarry, river or spring produced a higher proportion of fatalities than the percentage of these divers in the total diving population. Injured divers were much more likely to report difficulty maintaining buoyancy, with rapid ascents, running out of gas or missed decompression.

The sub-population of each group (injuries, fatalities and PDE) using mixed gas demonstrated that nitrox was not associated with an increased proportion of fatalities. Trimix or heliox featured in a higher percentage of fatalities than did mixed gas, but still constituted only 5% of the fatalities. Fatalities on trimix or heliox combined were more experienced than other mixed-gas or air divers, with a mean of 15 years since certification. Injured divers had the deepest maximum depths in their dive series regardless of what gas they used, compared with fatalities and the PDE population. Divers with injuries were more likely to have high oxygen partial pressures when diving using nitrox.

PDE states that it does not purport to be a representative sample of recreational diving and using PDE to make general statements about all divers is inappropriate. Representative subsets can be used as control groups for comparison studies. The data are beginning to show some tantalising trends for women, older divers, pre-existing medical conditions and experience. These may become clearer over the next few years. DAN are to be congratulated for PDE, which has enormous potential for increasing our knowledge of the true risks of recreational diving.

Table 23
Incidence of DCS in four groups of divers

Group	# Divers	# Dives	# DCS	Incidence/10,000 Dives	Incidence/100 Divers
Liveaboard	290	3,928	0	0	0.00
Cold-Water Wreck	233	2,413	9	37	3.86
Shore/Day-Boat	705	3,796	2	5	0.28
Day-Boat Dive Professionals	48	5,248	5	10	9.53

Table 24
Incidence of DCS by category of dive qualification

Group	# DCS Cases	Denominator		Incidence	
		# Dives	# Divers	Per 10,000 Dives	Per 100 Divers
Instructors & Divemasters	8	7,070	387	11	2.1
Open Water & Advanced Open Water	8	6,069	748	13	1.1
Total	16	13,139	1,135	12.2	1.4

Reference

1 Divers Alert Network. *Report on decompression illness, diving fatalities and Project Dive Exploration. The DAN annual review of recreational scuba diving injuries and fatalities, based on 2001 data, 2003 edition.* Durham, NC: Divers Alert Network; 2003.

Graham McGeoch, Hyperbaric Medicine Unit, Christchurch Hospital, Christchurch, New Zealand

Key words

Diving, decompression illness, accidents, equipment, DAN - Divers Alert Network, reprinted from, book review

Hyperbaric oxygen therapy for chronic wounds

Kranke P, Bennett M, Roeckl-Wiedmann I, Debus S

Synopsis

We found some evidence that people with diabetic foot ulcers are less likely to have a major amputation if they receive hyperbaric oxygen therapy. This is based on three randomised trials with a limited number of patients. Further research is needed.

Chronic wounds, often associated with diabetes, arterial or venous disease are common and have a high impact on the wellbeing of those affected. Hyperbaric oxygen therapy (HBOT) is a treatment designed to increase the supply of oxygen to wounds that are not responding to other measures to treat them. HBOT involves people breathing pure oxygen in a specially-designed chamber (such as that used for deep-sea divers suffering pressure problems after resurfacing).

The review of trials found that HBOT seems to reduce the number of major amputations in people with diabetes who have chronic foot ulcers, and may reduce the size of wounds caused by disease to the veins of the leg, but found no evidence to confirm or refute any effect on other wounds caused by lack of blood supply through the arteries or pressure ulcers.

Background

Chronic wounds are common and present a health problem with significant effect on quality of life. The wide range of therapeutic strategies for such wounds reflects the various pathologies that may cause tissue breakdown, including poor blood supply resulting in inadequate oxygenation of the wound bed. Hyperbaric oxygen therapy (HBOT) has been suggested to improve oxygen supply to wounds and therefore improve their healing.

Objectives

To assess the benefits and harms of adjunctive HBOT for treating chronic ulcers of the lower limb (diabetic foot ulcers, venous and arterial ulcers and pressure ulcers).

Search strategy

We searched the Cochrane Wounds Group Specialised Trial Register (searched 6 February 2003), CENTRAL (The Cochrane Library Issue 1, 2003), Medline (1966–2003), EMBASE (1974–2003), DORCTHIM (1996–2003), and reference lists of articles. Relevant journals were handsearched and researchers in the field were contacted.

Selection criteria

Randomised studies comparing the effect on chronic wound healing of therapeutic regimens which include HBOT with those that exclude HBOT (with or without sham therapy).

Data collection and analysis

Three reviewers independently evaluated the quality of the relevant trials using the validated Oxford Scale (Jadad 1996) and extracted the data from the included trials.

Main results

Five trials contributed to this review.

DIABETIC FOOT ULCER (4 TRIALS, 147 PATIENTS)

Pooled data of three trials with 118 patients showed a reduction in the risk of major amputation when adjunctive HBOT was used, compared to the alternative therapy (RR 0.31, 95% CI 0.13 to 0.71). Sensitivity analysis for the allocation of dropouts did not significantly alter that result. This analysis predicts that we would need to treat 4 individuals with HBOT in order to prevent 1 amputation in the short term (NNT 4, 95% CI 3 to 11). There was no statistically significant difference in minor amputation rate (pooled data of two trials with 48 patients). Healing rates were reported in one trial which showed a significant improvement in the chance of healing 1 year after therapy (RR for failure to heal with sham 2.3, 95% CI 1.1 to 4.7, P = 0.03), although no effect was determined immediately post HBOT, nor at 6 months. Further, the beneficial effect after 1 year was sensitive to allocation of dropouts.

VENOUS ULCER (1 TRIAL, 16 PATIENTS)

This trial reported data at six weeks (wound size reduction) and 18 weeks (wound size reduction and healing rate) and suggested a significant benefit of HBOT in terms of reduction in ulcer area only at 6 weeks (WMD 33%, 95% CI 19% to 47%, P <00001).

ARTERIAL AND PRESSURE ULCERS

No trials that satisfied inclusion criteria were located.

Reviewers' conclusions

In people with foot ulcers due to diabetes, HBOT significantly reduced the risk of major amputation and may improve the chance of healing at one year. The application of HBOT to these patients may be justified where HBOT facilities are available, however economic evaluations should be undertaken. In view of the modest number of patients, methodological shortcomings and poor reporting, this result should be interpreted cautiously however, and an appropriately powered trial of high methodological rigour is justified to verify this finding and further define those patients who can be expected to derive most benefit from HBOT. Regarding the effect of HBOT on chronic wounds associated with other pathologies, any benefit from HBOT will need to be examined in further, rigorous randomised trials. The routine management of such wounds with HBOT is not justified by the evidence in this review.

Implications for practice

There is some limited evidence that HBOT reduces the rate of major amputation in people who have chronic foot ulcers as a result of diabetes. Thus, the application of HBOT to these patients may be justified where HBOT facilities are available however an economic evaluation should be undertaken. Furthermore the small number of studies, the modest numbers of patients and the methodological and reporting inadequacies of the primary studies included in this review demand a cautious interpretation. To date no useful information regarding the efficacy or effectiveness of HBOT for chronic wounds with other underlying pathologies can be provided.

Implications for research

There is insufficient evidence to recommend the routine use of HBOT in the clinical treatment schedule for people with diabetes related foot ulcers. There is a strong case for further large randomised trials of high methodological rigour in order to define the true extent of benefit from the administration of HBOT. Specifically, more information is required on the subset of disease severity or classification most likely to benefit from this therapy, the time for which we can expect any benefits to persist, and the oxygen dose most appropriate.

Any future trials would need to consider in particular:

- Appropriate sample sizes with power to detect expected differences
- Careful definition and selection of target patients
- Appropriate oxygen dose per treatment session (pressure and time)
- Appropriate comparator therapy
- Use of an effective sham therapy
- Effective and explicit blinding of outcome assessors and surgeons
- Appropriate outcome measures including all those listed in this review
- Careful elucidation of any adverse effects
- The cost-utility of the therapy

There is a strong case for investigation of the effects of HBOT on chronic wounds due to venous disease, arterial disease and pressure damage, in large, rigorous randomised clinical trials. Future trials should consider the items and outcomes as stated above (diabetic foot ulcers).

This review should be cited as Kranke P, Bennett M, Roeckl-Wiedmann I, Debus S. Hyperbaric oxygen therapy for chronic wounds (Cochrane Review). In: *The Cochrane Library*, Issue 2, 2004. Chichester, UK: John Wiley & Sons, Ltd. Reproduced with kind permission of the authors.

Key words
Cochrane library, hyperbaric oxygen, treatment, chronic wounds, diabetes, reprinted from

SPUMS notices and news

South Pacific Underwater Medicine Society Diploma of Diving and Hyperbaric Medicine

Requirements for candidates

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 a medically qualified 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 Hyperbaric Medicine Unit.
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, and in a standard format, for approval by the Academic Board 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.

Additional information

The candidate must contact the Education Officer to advise of their intended candidacy, seek approval of their courses in Diving and Hyperbaric Medicine and training time in the intended Hyperbaric Medicine Unit, discuss the proposed subject matter of their research, and obtain instructions before submitting any written material or commencing a 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.

The Academic Board reserves the right to modify any of these requirements from time to time. The Academic Board consists of:
Dr Chris Acott, Education Officer, Professor Des Gorman and Associate Professor Mike Davis.
All enquiries should be addressed to the Education Officer:
Dr Chris Acott,
30 Park Avenue
Rosslyn Park
South Australia 5072
Australia
E-mail: <cacott@optusnet.com.au>

Key words
Qualifications, underwater medicine, hyperbaric oxygen, research

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Minutes of the SPUMS Committee meeting held at the ASM Noumea in May/June 2004 over several days

Opened: 2 June 2004
Present: Drs R Walker (President), G Williams (Immediate Past-President), C Meehan (Secretary), M Davis (Editor), A Patterson (Acting Treasurer), M Bennett, D Doolette (Education Officer), D Smart (ANZHMG Representative)

Apologies: Drs S Mitchell, D Walker (committee members)

1 Minutes of the previous meeting (9 March 2004)
Moved that the minutes be accepted as a true record, after minor corrections.
Proposed Dr R Walker, seconded Dr G Williams, carried.

2 Matters arising from the minutes
2.1 Improving our Internet cost effectiveness
Dr Walker will formalise a proposal.
2.2 Update from Editor, Dr M Davis
The list of SPUMS diplomates needs to be corrected. Dr D Doolette will give the list to Dr C Meehan for revision.
2.3 UHMS report
Dr M Bennett was congratulated for coordinating a very successful meeting in Sydney.
2.4 Resignation of Dr Doolette was accepted with sadness by the Committee. David was thanked for

all his hard work as SPUMS Education Officer. Dr Chris Acott has volunteered to fill the position and the Education Committee will be reviewed in one year.

2.5 Update from ANZMG, Dr D Smart
Various issues were discussed. Dr Smart will provide the Committee with a copy of the year's report.

2.6 Dr M Davis gave an update on the new University of Auckland PGDipMedSc – Diving and Hyperbaric Medicine course. It will commence 2nd semester 2004.

2.7 CME points – clarification of the position of various colleges is being sought. At present the conference is approved by RACGP, ANZCA and the College of Emergency Medicine. Yearly approval is necessary for these.

3 Annual Scientific Meetings

3.1 2004 ASM Noumea
An update was given by Dr G Williams. There was discussion regarding some complaints relating to the registration fee and the additional conference room fee for those not booking the Allways Travel Package (in which this fee was factored into the accommodation costs). It was decided that in future there should be clearer demarcation between the conference and the travel costs, and no disadvantage to those choosing to book independently. There was also some discussion regarding the cost of the cocktail party for those not registered for the meeting. Dr Robyn Walker will update the SPUMS conditions and requirements for the ASM travel arrangements.

3.2 2005 ASM will be at Coco Palm Resort and Spa, Maldives. Dr Meehan is the Convener. Michael Lang has been suggested as a guest speaker. Allways Dive Expeditions are the chosen travel agents. Every quote for the travel costs provided by the travel agent should clearly show the exchange rate used at the time. There should also be separation between the costs of travel and accommodation. No attendant will be disadvantaged if they choose to book independently. However, if the attendant wishes to dive with the SPUMS group, they will need to book the diving package through Allways Dive Expeditions. There is some advantage to SPUMS if attendants take advantage of the Allways package, as the FOCs generated belong to SPUMS and will be used to offset the travel costs of the Guest Speaker, Convener and Administrator.

3.3 Venues for future ASMs will be discussed at the face-to-face meeting in Sydney later this year.

4 Treasurer's report
Given by Dr Patterson.

5 Correspondence
Communications from Dr Turnbull. The issues regarding the diving and the conference fee for the ASM in Noumea have already been discussed.

6 Other busines
None

Closed: 4 June 2004

Minutes of the SPUMS committee telephone conference held on Sunday 12 September 2004

Opened: 0900 hr

Present: Drs R Walker (President), G Williams (Immediate Past-President), C Meehan (Secretary), M Davis (Editor), A Patterson (Treasurer), C Acott (Education Officer)

Apologies: Drs S Mitchell, D Walker (committee members), D Smart (ANZHMG Representative)

1 Minutes of the previous meeting (May 2004)
Moved that the minutes be accepted as a true record, after minor corrections.
Proposed Dr A Patterson, seconded Dr R Walker, carried.

2 Matters arising from the minutes

2.1 Improving our Internet cost effectiveness
A decision will be made on Tuesday when all the Committee has had a chance to look at the proposals and quotes provided by Dr R Walker.

2.2 Update from the Editor, Dr M Davis
A written report will be presented at the face-to-face meeting to be held in Sydney on 30 October. Dr Davis reports that only one presenter has provided him with a written paper of their presentation given at the Noumea ASM. This is an ongoing problem.

2.3 Update from Education Officer, Dr C Acott
Dr Acott reports that there a four more candidates interested in doing the SPUMS Diploma. Dr Acott will look up and check the list of SPUMS diplomates and the dates they received their diplomas. The list has errors at present.

3 Annual Scientific Meetings

3.1 2004 ASM Noumea final figures
The final figures are not yet available but will be presented at the face-to-face meeting in Sydney. There was discussion on how to streamline the registration process for future meetings. Although the registration form and fees go initially to Steve Goble, the Administrator, the Convener needs to be kept informed of the progress of bookings. Due to the reducing numbers attending the Conference, the road ahead also needs to be discussed.

3.2 2005 ASM, Coco Palm Resort and Spa, Maldives update
Mr Geoff Skinner from Allways Dive Expeditions and Dr Cathy Meehan recently returned from the site inspection. The venue is suitable and the resort organisers are anxious to have an idea of numbers as early as possible in order to facilitate smooth running of the Conference. In view of this, there will be an early bird registration to encourage delegates to book early. Final costings are not yet available from Allways Dive Expeditions, and so the Conference brochure cannot be finalised. There will therefore be a delay in the September journal being posted, as the brochure will go with this. There have been several changes in the quotes provided by Allways, partly as a result of exchange-rate variations with the US dollar, and also with changes in the SPUMS requirements from the travel agents. Clarification of the changes from the original quote provided in December 2003 has been requested.

3.3 Venues for future ASMs were briefly discussed. The Marshall Islands has been recommended, as has returning to PNG. This will be discussed further at the face-to-face meeting in Sydney.

4 Treasurer's report
An update of the Society's financial position was given by Dr A Patterson.

5 Correspondence

5.1 A letter of resignation from the position of Committee Member was received from Dr Michael Bennett. The Committee thanks Dr Bennett for all his hard work.

5.2 Letter Stephen Etheredge. The Committee decided that, as there seemed to be some confusion as to which night the Gala Dinner was to be held at the recent ASM in Noumea, a refund for this event would be given to Dr Etheredge. All attendees who missed the Gala Dinner will be written to and the cost of this refunded if they feel that they were misinformed as to the correct date.

5.3 Letter Tom Remar, re Australian diving regulations; no response required as letter not addressed to SPUMS.

5.4 Letter regarding International Federation of Baromedical Interest Groups. Dr R Walker has responded.

6 Other business

6.1 Proposal that Dr Christine Lee be coopted to fill the position on the Committee vacated by Dr M Bennett. Dr Lee will stand in as a Committee Member until the next AGM. She will be invited to attend the face-to-face committee meeting in Sydney to be held on 30 October.

6.2 Update from Public Officer regarding timing of the AGM. The AGM in future needs to be held prior to 30 May each year in order to comply with the regulations.

6.3 The face-to-face committee meeting will be held in Sydney on 30 October. The meeting will be held at Dr Robyn Walker's residence commencing at 1000 hr. There is suitable accommodation within walking distance and Dr Walker will book this.

Closed: 1030 hr

SPUMS Annual General Meeting 2005

Notice of Annual General Meeting of SPUMS to be held at Coco Palm Resort, Baa Atoll, Maldives at 1800 hr on Wednesday 27 April 2005

Agenda

Apologies:

Minutes of the previous meeting:

Unratified minutes of the previous meeting will be posted on the meeting notice board and appeared in the *SPUMS J*. 2004; 34: 159-64.

Matters arising from the minutes:

Annual reports:

President's Report.
Secretary's Report
Education Officer's Report
Presidents' Committee Report

Annual Financial Statement and Treasurer's Report:

Proposal regarding subscription fees for 2006:

Election of office bearers:

Nominations have been called for the positions of President, Secretary and three (3) Committee Members.

Appointment of the Auditor:

Business of which notice has been given:

Notice from the Secretary

There have been no objections to the motions passed at the 2004 AGM received by the Secretary within the required one calendar month of their posting in the last issue of the Journal. These motions, therefore, now come into effect.

Obituary

Ed Thalmann, MD

Dr Ed Thalmann, an Assistant Medical Director of Divers Alert Network (DAN) for nine years, died 24 July in his Durham, NC, home. He was 59.

Dr Thalmann was regarded as one of the world's foremost authorities on diving decompression. At DAN he was the physician resource for DAN medics, consulting with dive medicine physicians who treated patients worldwide. He headed DAN's continuing education programmes for the medical staff and on-call doctors, and participated in numerous research efforts. While at DAN, Dr Thalmann also worked on the Duke University Medical Center medical staff, the Duke Anesthesiology Department and at the Center for Hyperbaric Medicine and Environmental Physiology. There, he participated in both patient care (dive accidents and clinical hyperbaric oxygen treatments) and in medical research programmes.

Michael D Curley, PhD, president and CEO of DAN, was a longtime colleague of Thalmann's, beginning in the US Navy. Curley said "*We mourn the passing of a truly prodigious talent, a wonderful diving physician and a fine man. During the past 25 years Ed Thalmann guided and inspired a generation of dedicated researchers in undersea medicine; his high standards of excellence and commitment to improving diving safety are gratefully acknowledged. Ed led by example, exhibiting courage by performing the same arduous experimental dives as his volunteers and risking the 'bends' and oxygen-induced seizures underwater. Whether developing new guidelines for decompression, diver thermal protection, underwater breathing apparatus or exposure to underwater sound, Ed always kept focused on improving diver safety. He leaves a great legacy of scientific excellence which we seek to emulate.*"

During a distinguished 22-year career in the US Navy, he helped develop operational procedures for diving, testing and evaluation of diving life-support equipment and medical research programmes. Projects included the development of new diving decompression tables (surface-supplied and saturation), measuring the performance of and writing specifications for diver thermal protective equipment, studying the effects of underwater exercise on diver performance and improving underwater breathing apparatus design and testing. He spearheaded two major revisions to the diving medicine chapter of the US Navy Diving Manual in 1985 and 1993. Dr Thalmann finished his tour in the Navy at the Naval Medical Research Institute in Bethesda, Md, as its head of diving medicine and physiology research. There, he was principal investigator for the Navy's decompression research programme.

Though only a few SPUMS members would have known Ed Thalmann, many will be familiar with his publications on diving medicine. With Richard D Vann, PhD, DAN Vice President of Research, he co-authored the decompression physiology chapter of the third edition of *The physiology and medicine of diving* and he wrote a section on decompression sickness for the *Handbook of physiology, adaptation of the environment*.

This is an edited version of the obituary in *Pressure*, reprinted with the kind permission of the Undersea and Hyperbaric Medical Society.

Book reviews

Textbook of hyperbaric medicine, 4th edition

K K Jain (ed)

550 pages, hardback
ISBN 0-88937-277-2
Hogrefe & Huber; 2004
Copies can be ordered online from Amazon.com: <www.amazon.com>
Also available direct from publisher: <www.hhpub.com>
Price: US$140.00

The arrival of the latest edition of K.K. Jain's *Textbook of Hyperbaric Medicine* renews the controversy about which is the 'best' general textbook in the field. This book has held an interesting place in the field of hyperbaric medicine since the first edition appeared in 1990, and that looks set to continue with this latest offering. Beloved of those who embrace the treatment of a wide range of neurological conditions, challenged by those who prefer a more evidence-based approach to practice, this edition is unlikely to persuade anyone to change sides in the ongoing debate about the 'proper' place of hyperbaric oxygen therapy (HBOT). This edition certainly loses nothing by comparison to the three previous ones. While much of this book is directly addressed to the hyperbaric physician, there is a wealth of information here for nursing and technical staff.

I have read the book in a final galley proof form, so may not have the right impression, but the book is likely to be of similar size to the preceding edition, although 140 pages shorter, and so is quite an imposing text, too big to balance on the chest in bed at night! The bibliography is unchanged in size (maintained at approximately 2000 references), but in his preface Professor Jain tells us that 200 new references have been included and 200 old ones discarded. There is a single new chapter devoted to the use of HBOT for the treatment of cerebral palsy.

The *Textbook of hyperbaric medicine* is described by the editor and principal author as "*a definite and inclusive source covering this body of knowledge* [hyperbaric medicine]." This is an accurate claim. The general tone of this book is both definite (no room for doubt) and inclusive (supports a wide range of indications). In my opinion, this is both a fascinating and a dangerous book. The scope is wide, yet despite the editor selecting a total of 19 specialists, much of the work remains that of Jain himself. He is sole author of 29 of 40 chapters, and the first author of a further three. In large part, therefore, we should take this book as Jain's view of the field rather than a widely collaborative work. If anything, this position has intensified since the previous edition. For example, Jain has taken sole authorship of the chapter on oxygen toxicity, although there is little new information and the text is largely unchanged since the third edition that was attributed to four authors. There is still no information for the clinician on the relative frequency of different clinical manifestations of cerebral oxygen toxicity.

The book is logically organised, well presented and the text reads well for the most part. Starting with a series of prefaces and forewords, the chapters lead us through history, physics and physiology, hypoxia and oxygen toxicity, equipment and clinical aspects. In general, the text maintains its focus, but the chapter on exercise under hyperbaric conditions seems a little out of place in what is essentially a clinical textbook.

Each chapter is presented in a two-column format and subdivided into sections by attractive single-column headers. I found the sequential development of most chapters quite logical and easy to follow, but the impression is a little staccato in the short chapters, (some only two to three pages long). At times there is a tendency to make statements unsupported by references.

Taking the chapter on decompression sickness (DCS) as an example relevant to the SPUMS membership, there is first a brief introduction, following which Professor Jain discusses pathophysiology (one reference from 1985), pulmonary changes (one reference from 1968) and bubble-induced CNS injury (eight references, none more recent than 1994). A significant proportion of the discussion in this section is actually about a vascular-embolic theory for the pathogenesis of multiple sclerosis (MS) proposed by Philip James and others in the 1980s, which is largely ignored by neurologists. Correct or otherwise, there is no acknowledgement that this theory is at best controversial. Neither does Professor Jain acknowledge the difficulties in terminology when discussing vascular bubble injury in the context of an autochthonous/venous disease such as DCS. I would commend the more specialist text of Bennett and Elliot for those wanting to bring themselves up to date in this area. The chapter continues through changes in the blood, dysbaric osteonecrosis, the role of free radicals and then into the clinical features. Interestingly, the only condition mentioned in the differential diagnosis is our old adversary, MS. This is not top of my usual list of possibilities. Several sections then follow covering management, including the use of SPECT brain imaging in persisting deficits to monitor the response to low-pressure 'tailing' treatments. Interestingly, we are informed that the US Navy has adopted this approach. Finally in the discussion of risk factors, many of us will be bemused by the exhortation to "*prohibit from diving*" any person over 20% in excess of ideal body weight.

Other chapters also contain unusual omissions and errors. For example, the chapter on carbon monoxide poisoning follows a good, detailed discussion of mechanisms, causes and clinical effects with a discussion of clinical trials which fails to even mention the controversial study of Scheinkestel

1999 (although it appears in an accompanying table). The chapter on wound healing, plastic surgery and dermatology refers to a Hart 1974 trial as "*a randomized, double-blind study of 191 burn patients*", although it is clear this trial enrolled only 16 subjects and was followed by a discussion of the authors' experience treating 191 cases.

The chapters on the treatment of a variety of neurological conditions are, not surprisingly, contentious given some of the authors involved (e.g., Philip James, Paul Harch and Richard Neubauer), and should be read by all who work in the field. They provide a great insight into another world of hyperbaric practice for those who work under more 'mainstream' constraints. There is quite a bit of unreferenced detail (e.g., Tables 17.3 and 17.6) while some of the chapters draw contradictory conclusions from the same evidence; Jain recommends 1.5 ATA (153 kPa) treatments for acute ischaemic stroke in Chapter 17, while Harch and Neubauer recommend pressures above 2.0 ATA (202 kPa) in Chapter 18.

I reserve my most strident criticism for the chapters on MS and cerebral palsy (CP). These chapters are misleading of the evidence and should not be used alone for any appraisal of the rectitude or otherwise of using HBOT for these conditions. The treatment of MS is justified by an unopposed championing of the vascular–embolic theory for the development of MS (see above), while there is a lengthy presentation of the highly biased (and unpublished) data presented from the '*Federation of multiple sclerosis treatment centers*'. This 'research' claims among other things, that 77% of people with walking difficulties are improved in this regard in response to the initial course of HBOT. This claim is wildly above even the most optimistic of the comparative data, and somewhat at odds with the Perrins, Neubauer and James' assertion that "*initial response is an unreliable guide*" to the ultimate success of HBOT in ameliorating progression.

The treatment of the clinical evidence concerning HBOT for CP is similarly misleading. In a discussion of the Collet 2001 study in Quebec for example, the virtues of 1.3 ATA air (131 kPa) 'treatments' are extolled as exerting "*a real effect on the partial pressure of blood gases and…increases the plasma oxygen tension*." They did not point out that the same result can be achieved with 27% oxygen at 1 ATA (101 kPa) with considerably less cost and a zero rate of barotrauma.

The authors of these chapters are, of course, entitled to their opinions. Buyers of this text should be aware, however, that the opinions expressed in some of this book are not those of the majority of their hyperbaric colleagues. Caveat emptor!

Physically, the book will be a treat. Handsome and well presented, the layout is straightforward and elegant with high-quality line drawings and tables. There are several colourful pages of SPECT scan images. The same clarity applies to the text, where the fonts used are limited in number and easy to read. The editors have avoided a busy style in favour of a simple approach that this reader finds conducive to comprehension.

To the credit of all concerned, there are relatively few typographical errors and the written style is generally clear and concise. The index is useful, if a little spare and idiosyncratic. For example 'Necrotising enterocolitis' yields two references: 'HBO therapy' in the gastroenterology chapter, and 'treated with HBO' in the paediatric chapter. The reason for the two sub-descriptors escapes me as they discuss the same subject.

In summary, the *Textbook of hyperbaric medicine* remains a significant text in the field. I thoroughly recommend it as a counterpoint reference for any hyperbaric physician because of the relatively marginal views expressed in some of the chapters. While the price tag is not inconsiderable, it is worth a working knowledge of how some of our colleagues think.

Michael Bennett
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Key words

Hyperbaric oxygen, oxygen, treatment, textbook, book reviews

Freedive!

Terry Maas and David Sipperly

152 pages, hardback
ISBN 1-9644966-1-5
Ventura, California: BlueWater Freedivers; 1998
Available from BlueWater Freedivers, 552 N Victoria Ave, Ventura, California 93003, USA.
Ph: (+1)-805-677-7462; Fax: (+1)-805-650-3014
Copies can be ordered online at <www.freedive.net>
Price US$47.00, postage and packing extra

My interest in reviewing this book was stimulated by a visit to New Zealand by Terry Maas earlier in 2004. Knowing of his considerable reputation in freediving circles and the quality of his underwater photography, which has appeared in many magazines, I felt this might be worth a look for Society members; I was not disappointed. Although publication was some years ago (1998), the book does achieve its goal to be a "*complete guide to breath-hold diving*".

The lack of emphasis on snorkel swimming techniques in the programmes of the major recreational-dive-training

agencies has been a concern for this reviewer for many years. Yet many scuba-diving fatalities and near misses are the result of divers getting into trouble on the surface simply because they do not have the skills to cope with surface fin swimming. In addition, a quarter to a third of all diving-related deaths in Australia and New Zealand are in freedivers (the term now preferred). Therefore, it is important that diving physicians and other health workers in the field, as well as diving instructors and dive masters, know something about freediving.

The book starts with a "*Warning - Disclaimer*", the first sentence of which states "*freediving is a potentially dangerous endeavour that can lead to permanent injury and death*." The tenor of this full-page warning embraces the measured sense of responsibility combined with enthusiasm and sheer love of this wonderful sport that is present throughout, from the pleasures of simple sightseeing to extreme-depth freediving.

In the introduction, the freediver is likened to the glider pilot, "*both enjoying an exhilarating world, silent of machinery.*" Since most of the ocean's colour and life are within 10 metres of the surface, there is little reason to go deeper, and this is a depth range that most people can master with relative ease. The short chapter on history, though it tends to focus particularly on the authors' own backyard, California, has some fascinating insights and some interesting photos of early home-made equipment. Of interest, the early freedivers tended to dive in exhalation rather than full inspiration, a technique that still has its devotees today.

There are two chapters on physiology. The mechanisms and dangers of 'apnoeic hypoxia', still incorrectly termed 'shallow-water blackout' in this book as so often elsewhere, are strongly emphasised, and the summary table on how to avoid it is excellent. There is a brief but clear account of barotrauma problems. Given the long list of acknowledgements to physicians and physiologists for these chapters, I was somewhat surprised at some of the statements by 'true-believers' that got through. Whilst the importance of proper breathing technique cannot be denied, the statement that 'chest breathing' "*leaves air trapped in the lower portions of the lungs, where it stagnates*" raised an eyebrow.

Many of the claims made here for breath-hold adaptation in human freedivers cannot be substantiated scientifically. There is, nevertheless, a widely held belief in freediving circles that this can be developed with training and that the trained diver can develop pre-dive manoeuvres that will reduce air consumption (presumably they mean oxygen) and narcosis. This is an area wide open for physiological research. There are some omissions here, such as the subtle effects of mild hypothermia, and the initial reflex respiratory response to sudden immersion in cold water. Most importantly, immersion diuresis is barely mentioned only right at the end, whereas it is of considerable importance to freedivers who spend any length of time in the water, especially in cooler climes.

Two chapters follow on equipment and diving techniques. There are lots of useful tips from choosing a mask to using a paddleboard. The so-called quick-release weight-belt buckle depicted on page 62 would have many safety-conscious divers squirming.

In practical terms, the best chapter in the book is the one on techniques. After a half-century of freediving, I thought I knew everything there was to know about diving skills, but this excellent piece of writing made it abundantly clear that I do not! These are skills and exercises that should be taught to every diver, and would undoubtedly enhance scuba-diving safety; time for our senior PADI and SSI members to take note. I have already incorporated some of Maas's tips into my own freediving preparation; who said you cannot teach an old dog new tricks!

The remaining five chapters deal with some of the activities available for the freediver. Not surprisingly given the authors' background, underwater photography figures large here. This is followed by underwater hockey ("*If you love the water, enjoy team play and are looking for a way to stay in excellent shape, then underwater hockey is your sport.*" Right on, Mr Maas!), use of monofins, game gathering and extreme-depth diving. The chapter on diving for depth records starts: "*We want to be clear: competitive deep freediving is a dangerous sport, and we don't condone it.*" As you can see, this is far from being a macho book about bravura and spearfishing. Indeed, only six pages are devoted to spearfishing, and three of these are full of photos.

The presentation is that of a coffee-table publication on shiny, high-quality paper with large-format, double-column text and frequent use of pull-out quotes. There are very few typesetting errors. Whilst this book is very well written, the most outstanding feature is the photography. It is full of stunning underwater images and the practical aspects are illustrated by good, clear diagrams and excellent photos demonstrating equipment and techniques.

There is an accompanying hour-long video, *The joy of freediving*, produced by Emmy-Award winner Laszlo Pal and Maas (available from <www.freedive.net>), which I have not seen but am now keen to do so. I look forward with enthusiasm to reviewing the next edition. This book is definitely one for your Christmas/New Year dive bag.

Michael Davis, Editor, SPUMS Journal

Key words

Freediving, breath-hold diving, snorkelling, general interest, book reviews

Bove and Davis' diving medicine, 4th edition

Alfred A Bove

648 pages, hardback
ISBN 0-7216-9424-1
Philadelphia: Saunders; 2004
Available from Best Publishing Company, P O Box 30100, Flagstaff, Arizona 86003-0100, USA.
Ph: (+1)-928-527-1055; Fax: (+1)-928-526-0370
E-mail: <divebooks@bestpub.com>
Copies can be ordered online at www.bestpub.com
Price US$79.00, postage and packing extra

There are currently three major English-language textbooks in diving medicine: this book, *Bennett and Elliott's Physiology and medicine of diving* (B & E) and Edmonds et al's *Diving and subaquatic medicine*. All are in their fourth or fifth edition. It is inevitable, therefore, that some comparisons need to be drawn between the three and that the question be asked why this subspecialty needs three such books; surely they overlap a great deal? Clearly, Saunders consider B & E and Bove and Davis to be complementary as they publish both. B & E is regarded as the main scientific reference text with a multitude of 'expert' contributors and in-depth reviews of a wide range of subjects. As such, it tends not to be particularly clinically orientated, but rather is solidly grounded in the basic and applied sciences. Edmonds et al is the opposite, written by a small group of Australian authors all of whom draw strongly on their time at the School of Underwater Medicine in Sydney, and is very much a clinical textbook combined with good summaries of the relevant background theory and physiology. Being an Australian textbook it is probably less regarded in the Northern Hemisphere, and especially the USA, than it deserves.

Bove and Davis lies somewhere between the two, having a large number of contributors (over 30) but with a greater clinical emphasis to it than B & E. Therefore, in the USA which is clearly its main market, it is likely to fill the same niche as Edmonds et al does in our neck of the woods. This text is new to the reviewer, who had not read any of the previous three editions.

There are 29 chapters, ranging from an opening gambit into history and exiting with a look at US Navy diving practices, four appendices and an extensive index. Nine of the chapters are written by the same authors as the equivalent, more detailed chapters in B & E, making for some strong similarities between the two books in these areas. For instance, the chapters by Francis and Mitchell on the pathophysiology of decompression sickness and that by Bennett on inert gas narcosis and the high-pressure nervous syndrome follow closely the content and format of their contributions in B & E. This works well in the first case, but is a disappointment in the latter, leading to a not very readable account. Other authors, though including much of the same content, have altered their text to provide more clinical emphasis. This is sometimes the result of the involvement of a different joint author, as in the case of the hypothermia chapter where the inclusion of Frank Golden produces a quite different presentation.

The chapter on physics, written by Larry Taylor, a scientist at the University of Michigan, deserves special attention because most doctors tend to skip the physics sections of these books, judging by their generally poor understanding of this area of the diving environment. Taylor's account is worth reading as it is well written with lots of examples to help. The difficulty, however, lies in the necessity to use both the English system (used in the USA) and the metric system (used pretty well everywhere else) of measurement to meet market needs, which will undoubtedly add to some readers' confusion. There is at least one error in the calculations and figure 2.2B has been incorrectly reproduced.

Highlights for me included Richard Vann's two excellent chapters on inert gas exchange and bubble formation, and the mechanisms and risks of decompression. Hard going, but if you want just one source to help come to grips with this difficult area then Vann's accounts are the ones to go for. Ed Flynn contributes a chapter, full of common sense and clearly based on years of experience, on the medical supervision of diving operations that should be mandatory reading for all diving physicians. There is some duplication of information here on ENT aspects when compared with the chapter by Hunter and Farmer, but the practical clinical approach adopted by Flynn will be particularly useful for many readers. An increasing number of women dive, but there are few data regarding issues of their physiology and health. Maida Taylor brings together a wide range of material in an interesting account of current knowledge, and this chapter indicates some clear lines for future research. How divers perform underwater is reviewed succinctly by Egstrom and Bachrach; it's not just about inert gas narcosis. Carl Edmonds' and Richard Moon's chapters maintain their usual high standard.

Ten chapters, about one third of the book, are devoted to specific clinical issues such as cardiovascular disease, diabetes and the neurologic consequences of diving, and are rounded off with reviews of the medical evaluation of recreational (Bove) and working (Elliott) divers. It is in this area that this text departs significantly from and is complementary to B & E. Amongst a number of interesting issues in the neurology chapter was a brief discussion of post-decompression illness chronic pain syndromes, of which the reviewer has seen several cases but about which there is little information in the literature.

So, what was disappointing? Nothing really, though Egstrom's other contribution, on diving equipment, seemed

somewhat dated and was supported by poor photos, often illustrating equipment of a decade ago. Three chapters by Neuman, one of the current editors of B & E, on pulmonary barotrauma, pulmonary disorders and near drowning are average, but readable. He provides no real insights into the controversy about asthma and diving. Some authors have managed to include papers from 2003 and even early 2004 in their discussions, whereas others appeared not to have made much effort to update their reference lists. Other minor omissions include inadequate discussion of the alternative descriptive system to the Type 1/Type 2 categorisation of decompression sickness and no reference in the chapter on carbon monoxide poisoning to the extensive research on mechanisms of poisoning by Gorman's group.

Presentation of the book is good as one would expect from a major publisher, with legible typefaces and few typographical errors (one on page 117 on an equivalent air depth calculation needs correcting). However, some of the photographs and X-ray images do not reproduce particularly well, and deserve better attention in the future, whereas diagrams and tables are generally clear. The use of units of measurement used only in the USA is irritating for any non-American. Each chapter is accompanied by an extensive reference list. The one quibble about references is that they are only partially in the Vancouver format and should be fully converted for the next edition. The index runs to nearly 50 pages, but contains some errors. For instance, a check of the various entries for 'Immersion' revealed that four of the 10 items listed were incorrect.

This textbook is worth having, as are B & E and Edmonds et al. Any hyperbaric unit should have all three, as should all physicians working in the diving field as a regular component of their professional activities. If I had to recommend only one for healthcare workers with only an occasional diving medical involvement or just an interest, and for educated divers wishing to understand more of their body's interaction with their watery environment, then my choice is firmly rooted in Australia and Edmonds et al's book; a hard thing for a Kiwi to admit!

Mike Davis, Editor, SPUMS Journal

Key words

Underwater medicine, textbook, book reviews

HTNA 13th Annual Scientific Meeting
Preliminary Notice

Dates: 18 - 21 August 2005
Venue: Novotel St Kilda, Melbourne
Host Institution: <htna@alfred.org.au>
Registration/Enquiries: <www.htna.com.au>
Phone: +61-(0)3-9276-2269
Fax: +61-(0)3-9276-3052

Diving-related fatalities document resource

All the coronial documents relating to diving fatalities in Australian waters up to and including 1998 have now been deposited by Dr Douglas Walker for safe keeping in the National Library of Australia, Canberra.

These documents have been the basis for the series of reports previously printed in this Journal as Project Stickybeak.

These documents will be available free of charge to bona fide researchers attending the library in person, subject to the stipulation that the researcher signs an agreement that no identifying details are to be made public.

Accession number for the collection is: MS ACC 03/38.

It is hoped that other researchers will similarly securely deposit documents relating to diving incidents when they have no further immediate need of them. Such documents can contain data of great value for subsequent research.

The
SPUMS
web site is at
http://www.SPUMS.org.au

UNDERSEA AND HYPERBARIC MEDICINE SOCIETY
38th Annual Scientific Meeting 2005
Preliminary Notice

Dates: 16-18 June
Pre-courses: 15 June
Venue: Flamingo Hotel, Las Vegas, Nevada
For more information contact: Lisa Wasdin, UHMS
Phone: +1-301-942-2980, extn 104
E-mail: <lisa@uhms.org>

HISTORICAL DIVING SOCIETY AUSTRALIA, SE ASIA

All enquiries to:
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Phone: +61-(0)8-558-2970
Fax: +61-(0)8-558-3490
E-mail: <bramsay@iaccess.com.au>

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For further information, including fees, please contact the Course Coordinator: Jessica Rorich
Phone: +64-(0)9-373-7599, extn 88489
Fax: +64-(0)9-308-2379
E-mail: <occmed@auckland.ac.nz>
Full information on courses and admission regulations is available in the University of Auckland Calendar or at <http://www.auckland.ac.nz>

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For more details and application forms go to <www.navyhyperbaric.co.nz>

The Australia and New Zealand College of Anaesthetists Special Interest Group in Diving and Hyperbaric Medicine
and
The Australia and New Zealand Hyperbaric Medicine Group

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Phone: +61-(0)2-9382-3880
Fax: +61-(0)2-9382-3882
E-mail: <janikg@sesahs.nsw.gov.au>

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Instructions to authors

(Revised March 2004)

The *SPUMS Journal* welcomes contributions (including letters to the Editor) on all aspects of diving and hyperbaric medicine. Manuscripts must be offered exclusively to the *SPUMS Journal*, unless clearly authenticated copyright exemption accompanies the manuscript. All manuscripts, including SPUMS Diploma theses, will be subject to peer review. Accepted contributions will be subject to editing.

Contributions should be sent to:
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C/o Office 137, 2nd Floor, Christchurch Hospital,
Private Bag 4710, Christchurch, New Zealand.
E-mail: <spumsj@cdhb.govt.nz>

Requirements for manuscripts
Documents should be submitted electronically on disk or as attachments to e-mail. The preferred format is Word 97 for Windows. If submitted as a paper version, two printed copies of all text, tables and illustrations should also be mailed. 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 be subdivided into the following sections: an Abstract of no more than 250 words, Introduction, Methods, Results, Discussion, Acknowledgements and References. Acknowledgments should be brief. References should be in the format shown below. Legends for tables and figures should appear at the end of the text file after the references.

Paper versions and electronic files should be double-spaced, using both upper and lower case, on one side only of A4 paper. Headings should conform to the current format in the *SPUMS Journal*. All pages should be numbered. Underlining should not be used. Measurements are to be in SI units (mm Hg are acceptable for blood pressure measurements) and normal ranges should be included.

The preferred length for original articles is 3,000 words or less. Inclusion of more than five authors requires justification as does more than 30 references per major article. Case reports should not exceed 1,500 words, with a maximum of 10 references. Abstracts are also required for all case reports and reviews. Letters to the Editor should not exceed 500 words (including references, which should be limited to five per letter). Legends for figures and tables should be less than 40 words in length.

Illustrations, figures and tables should not be embedded in the wordprocessor document, only their position indicated. All tables are to be in Word for Windows, tab-separated text rather than using the columns/tables option or other software and each saved as a separate file. They should be double-spaced on separate sheets of paper. No vertical or horizontal borders are to be used. Illustrations and figures should be separate documents in JPEG or GIFF format. Please note that our firewall has a maximum size of 5Mbytes for incoming files or messages with attachments.

Photographs should be glossy, black-and-white or colour. Slides should be converted to photographs before being sent. Colour reproduction is available only when it is essential for clinical purposes and may be at the authors' expense. Indicate magnification for photomicrographs.

Abbreviations should only be used in brackets after the complete expression, e.g., decompression illness (DCI) can thereafter be referred to as DCI.

References
The Journal reference style is the 'Vancouver' style *(Uniform requirements for manuscripts submitted to biomedical journals*, updated July 2003. Web site for details: <http://www.icmje.org/index.html>).
In this system references appear in the text as superscript numbers.[1,2] The references are numbered in order of quoting. Index Medicus abbreviations for journal names are to be used (<http://www.nlm.nih.gov/tsd/serials/lji.html>). Examples of the format for quoting journals and books 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.

There should be a space after the semi-colon and after the colon, and a full stop after the journal and the page numbers. Titles of quoted books and journals should be in italics. Accuracy of the references is the responsibility of authors.

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

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+64-9-445-8454 (International)
The toll-free number 0800-4-DES111 can only be used in New Zealand

The DES numbers are generously supported by DAN-SEAP

PROJECT STICKYBEAK

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 regards to identifying details, is utilised in reports and case reports on non-fatal cases. Such reports can be freely used by any interested person or organisation to increase diving safety through better awareness of critical factors.

Information may be sent (in confidence) to:
Dr D Walker
PO Box 120, Narrabeen, NSW 2101, Australia.

DIVING INCIDENT MONITORING STUDY (DIMS)

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

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

PROJECT PROTEUS

The aim of this investigation is to establish a database of divers who dive or have dived with any medical contraindications to diving. At present it is known that some asthmatics dive and that some insulin-dependent diabetics dive. What is not known is how many. How many with these conditions die is known. But how many dive safely with these conditions is not. Nor is the incidence of diving accidents in these groups known.
This project is under the direction of Dr Douglas Walker and Dr Mike Bennett. The investigation has been approved by the Ethics Committee of the Prince of Wales Hospital, Randwick, approval number 01/047.

If you are in such a group please make contact. All information will be treated as CONFIDENTIAL.
No identifying details will appear in any report derived from the database.

Write to: Project Proteus
PO Box 120, Narrabeen, NSW 2101, Australia.
E-mail: <diverhealth@hotmail.com>

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All opinions expressed are given in good faith and in all cases represent the views of the writer and are not necessarily representative of the policy of SPUMS.

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