Cold-water diving induces airway narrowing

Decompression sickness – the tip of the scuba injury pyramid

A baby puzzle, low visibility and nitrogen narcosis

Divers need to be reasonably fit for scuba diving

Don't faint while escaping from a submarine

Immersion pulmonary oedema can be fatal

An update on investigating diving fatalities

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Editorials

Aerobic demand and scuba diving: concerns about medical evaluation

Gerardo Bosco, Antonio Paoli and Enrico Camporesi

Scuba diving has become a popular recreational sport throughout the world. Although it is not a competitive sport, a certain level of physical fitness is recommended because of the physical characteristics of the underwater environment.[1] Immersion alone will increase cardiac preload leading to a rise in both cardiac output and blood pressure, counteracted by increased diuresis. Increased oxygen partial pressure and cold exposure during scuba diving will additionally increase afterload by vasoconstrictive effects and may induce bradyarryhthmias in combination with breath-holds.[2,3] Volumes of gas in the body cavities will be affected by changing pressure, and inert gas components of the breathing-gas mixture will dissolve in body tissues and blood with increasing alveolar inert-gas partial pressure.[4,5] During decompression, a free gas phase may form in supersaturated tissues, resulting in the generation of inert gas microbubbles that are eliminated by venous return to the lungs under normal circumstances.[4] It has been reported that more air bubbles were detected in divers when dives were performed in the open sea rather than in hyperbaric chambers.[6,7] Both dry and wet dives are associated with hyperoxia, increased density of breathing gas, and decompression stress, with possible formation of venous bubbles and enhancement of the inflammatory cascade.[4,8]

However, open-water dives are also associated with immersion, the mechanical load of the breathing apparatus, a high level of physical activity, and exposure to a cold environment. Immersion in cold water results in breathing colder and denser gas and may also, by inducing peripheral cutaneous vasoconstriction in conjunction with the immersion effect, potentiate central pooling of blood more than in dry dives.[1,3,5] Water immersion-induced changes in haemodynamic, neuroendocrine and autonomic activities have been reviewed previously.[1] Cardiovascular conditions may have an impact on these physiological changes, increasing the risk of suffering adverse events from scuba diving. Systemic hypertension may be aggravated by underwater exercise and immersion. Metabolic disorders are also of concern, since obesity is associated with both higher bubble grades in Doppler ultrasound detection after scuba dives when compared to normal subjects and with an increased risk of decompression illness.[4]

Thus, the diver's cardiovascular status is important in the assessment of fitness to dive, and some cardiovascular conditions, such as symptomatic coronary artery disease and heart rhythm disorders, should preclude scuba diving.[9] Any history of cardiac disease or abnormalities detected during routine physical examination should prompt further evaluation and specialist referral. Recreational scuba diving is usually performed without accurate medical examination. In 2003, the only countries requiring pre-diving medical examination were France, the UK and Australia (no longer the case in Australia), while it is still required for commercial diving.[9] Swimming in rough water and strong currents can induce fatigue, anxiety or panic in divers.

A question of fundamental importance is: what is a level of physical fitness needed to deal with the reasonable, expected and unexpected demands of a recreational dive? The paper by Buzzacott et al investigates this topic, confirming previous research that the mean aerobic need is about 7 METs (metabolic equivalents).[10–13] Nonetheless, we know that a US Navy diver must swim at least at 1.3 knots, which means 13 METs and a recreational scuba diver usually swims at 0.5 knots (5 METs) but during an emergency he could reach up to 1.0 knot (10 METs). Moreover, we have to take into account that an expert scuba diver has a better exercise efficiency compared to the non-expert, so these conclusions appear reasonable.[14]

Many unresolved questions remain open: for example, the reliability of the value of MET. A recent paper reported that the mean rate of resting oxygen consumption ($\dot{V}O_2$) in a sample of healthy men was 3.21 mL·kg^{-1}·min^{-1}, significantly lower than the standard resting MET value of 3.5 mL·kg^{-1}·min^{-1}.[15] Also, another prediction model which included body surface area and percent body fat as predictors demonstrated relatively poor predictive ability.[16] Moreover, the error in estimating resting $\dot{V}O_2$ from 1 MET increases with increasing adiposity but the 1-MET value also overestimates resting $\dot{V}O_2$ values in normal-weight persons. Therefore, the use of a more correct raw value in mL O$_2$·kg^{-1}·min^{-1} might be preferable in addition to having a wider safety margin.[16] Others have suggested that a peak capacity of 11 to 12 METS could be an appropriate goal for recreational divers.[17] Another issue to be considered is the experience of the diver.[9] Paradoxically a more expert diver needs a lower $\dot{V}O_{2max}$ than a non-expert.[18] As Buzzacott reports: "*Dwyer's methods included swimming at a fixed depth for four minutes while pushing a board and his gas collection took place during only the last minute of steady-rate exercise, whereas our study involved free-swimming recreational divers and our data were averaged over much longer and more variable periods.*"[10,19]

The role of oxygen demands, with or without exercise, in immersion is implicated in decompression physiology as well, particularly in processes defined as 'denucleation' and 'denitrogenation'.[20,21] The increased oxygen content rapidly diffuses into micronuclei in exchange for nitrogen,

which is then eliminated from the body via the lungs. The oxygen is then absorbed by the surrounding tissue to cause rapid decay of the micronuclei. This theory has been supported by studies that demonstrate significantly reduced decompression-induced bubble formation in animals pre-treated with hyperbaric oxygen (HBO), believed to be owing to the elimination of bubble nuclei.[23] HBO has been observed to eliminate most of the gas nuclei in decompressed animals, thus reducing the number and size of bubbles during decompression. Reduction in the inflammatory cascade in humans has also been reported.[8] A previous study showed that HBO pre-breathing significantly reduced decompression-induced bubble formation and platelet activation in simulated dives in an HBO chamber.[6] Recent studies demonstrated that pre-breathing normobaric and hyperbaric oxygen in open water also decreased venous gas emboli formation, with a prolonged protective effect and repercussions on platelet activation and intracellular calcium accumulation in lymphocytes.[7,23–26]

In our recently completed Tremiti Islands experiment to quantify underwater exercise variables, all subjects were asked to perform the same mild workload at a depth of 30 metres' sea water on an underwater bicycle ergometer at a pedalling rate of 25 rpm to ensure no difference of ventilation and gas exchange in all dives, guided by the Borg category ratio 0–10 scale at an intensity level of 3.[7,26–27] Basic activities associated with scuba diving, such as surface swimming or walking with heavy equipment, may be enough to allow the passage of venous gas emboli through intrapulmonary arterial-venous anastomoses.[28] Some of the differences observed between the aerobic exercise and a non-exercise control dive related to a decompression-induced inflammatory pattern, and provide additional insight into the potential protective benefits of exercise performed before a dive.[29] Further study is needed to understand the potential of these benefits. In our opinion, the preventive measures to reduce decompression complications of diving include the acceptance of safe diving procedures, particularly related to descent and ascent and the exclusion of individuals with specific medical conditions. A more specific and in-water activity-related medical examination might be desirable for recreational scuba divers.

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Just say NO to decompression bubbles: is there a real link between nitric oxide and bubble production or reduction in humans?

Costantino Balestra

Vascular gas emboli (VGE) start forming during the degassing of tissues in the decompression (ascent) phase of the dive when bubble precursors (micronuclei) are triggered to growth. The precise formation mechanism of micronuclei is still debated, with formation sites in facilitating regions with surfactants, hydrophobic surfaces or crevices.[1,2] However, significant inter-subject variability to VGE exists for the same diving exposure and VGE may even be reduced with a single pre-dive intervention.[3,4]   The precise link between VGE and endothelial dysfunction observed post dive remains unclear and a nitric oxide (NO) mechanism has been hypothesized.[5]

Subjects in good physical condition are at lesser risk of VGE and DCS observed post dive.[6]   More surprisingly, single pre-dive interventions or 'preconditioning' can influence the VGE observed post dive. Studies in rats have shown that a single bout of exercise 20 h pre dive can reduce post-dive VGE and mortality.[4]   In humans, the role of exercise has been debated and depending on its timing and intensity may increase or decrease bubbles.[3,7–10]

A NO-mediated change in the surface properties of the vascular endothelium favouring the elimination of gas micronuclei has been suggested to explain this protection against bubble formation.[11] NO synthase activity increases following 45 minutes of exercise and NO administration immediately before a dive reduces VGE.[5]   Nevertheless, bubble production is increased by NO blockade in sedentary but not in exercised rats, suggesting other biochemical pathways such as heat-sensitive proteins, antioxidant defenses or blood rheology may be involved.[12]

The first link between NO and DCS protection was shown by chance.[4]   In an experiment using explosive decompression of sedentary rats resulting in >80% mortality, some additional rats were needed to complete the experiment but only trained (treadmill-exercised) rats were available instead of sedentary ones. After the decompression, 80% of the trained rats survived. The explanation given for this observation was that the presence of NO in the trained rats resulted in fewer bubbles and less DCS.

However, a French study showed that human volunteers had fewer bubbles post decompression after a treadmill exercise compared to the same exercise (same $\dot{V}O_2$) after a cycle-ergometer stress test. If this was related to NO production, the number of bubbles should be more or less the same. There are some mechanical differences between the two forms of exercise, namely more impacts and vibrations during the treadmill test. It is hypothesized that micronuclei are reduced by a mechanical effect as shown by an experiment with vibration applied before diving, which reduced decompression bubbling.[13]

In conclusion, more investigations are needed to further

ascertain the link between NO and post-decompression VGE modulation. Such studies should be directed more on high-intensity training (less NO-related), since aerobic efforts have already been extensively studied in relation to the reduction of decompression stress, this will probably allow more understanding of the subtle mechanisms for DCS protection. The variable effect of oxygen on bubble decay, with transient increase of volume in some cases, also requires further investigation.[14–16]

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The Editor's offering

Two thoughtful editorials leave little room for comment from your Editor. Interestingly both relate to different aspects of physical fitness and its potential impact on diving safety – if you are fit, you may be at less risk of suffering decompression sickness and, secondly, you are more likely to be able to handle any unexpected, physically-demanding situation whilst diving. Being physically fit for diving is an entirely different thing to being medically 'fit to dive' and highlights yet again the difficulty facing diving physicians when assessing the latter concept in divers.

Many parents will be familiar with the Tupperware™ *Shape O Toy* for young children, in which differently-shaped objects are 'posted' through the matching holes in a plastic ball – it was a favourite of my children when they were 'toddlers'. Its use for testing performance, as in the following paper by van Wijk and Meintjes, is an example of thinking 'outside the square'. One trusts that this is not a reflection on the perceived mental age of military and commercial divers!

I have just returned from the SPUMS ASM in Bali. This was a highly successful meeting, with excellent presentations from all, especially Peter Wilmshurst, our Guest Speaker from the UK. Next year is in Palau – see you there!

Mike Davis

The front page photo was taken by Quentin Bennett in Fiordland, New Zealand. The Editor is examining the black coral *Antipathes fiordensis* with the perching snakestar *Astrobrachion constrictum*, found only on black coral. The sea really is green.

Original articles

Complex tactile performance in low visibility: the effect of nitrogen narcosis

Charles H van Wijk and Willem AJ Meintjes

Abstract

(Van Wijk CH, Meintjes WAJ. Complex tactile performance in low visibility: the effect of nitrogen narcosis. *Diving and Hyperbaric Medicine*. 2014 June;44(2):65-69.)

Background: In a task-environment where visibility has deteriorated, individuals rely heavily on tactile performance (perception and manipulation) to complete complex tasks. When this happens under hyperbaric conditions, factors like nitrogen narcosis could influence a person's ability to successfully complete such tasks.

Objective: To examine the effect of nitrogen narcosis on a complex neuropsychological task measuring tactile performance at a pressure of 608 kPa (6 atm abs), in the absence of visual access to the task.

Methods: In a prospective cross-over study, 139 commercial divers were tested in a dry chamber at 101.3 kPa and 608 kPa. They completed the Tupperware Neuropsychological Task (TNT) of tactile performance without visual access to the task, and completed questionnaires to provide psychological and biographical data, which included trait anxiety and transient mood states, as well as formal qualifications and technical proficiency.

Results: A significant decrement (9.5%, $P < 0.001$) in performance on the TNT at depth was found, irrespective of the sequence of testing. Generally, neither the psychological nor biographical variables showed any significant effect on tactile performance. Tactile performance on the surface was a good indicator of performance at depth.

Conclusion: These findings have practical implications for professional diving where conditions of low visibility during deeper diving occur. Recommendations are made towards managing potential impairments in tactile performance, such as pre-dive practical learning ('rehearsal') as an aid to successful completion of tasks.

Key words

Nitrogen narcosis, deep diving, performance, psychology, diving research

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Introduction

Inert gas narcosis is one of the challenges facing deep air divers, with the neuropsychological manifestations of nitrogen narcosis typically encountered from about 30 metres' sea water (msw).[1] Impairment of cognitive function attributed to nitrogen narcosis includes effects on concentration, choice reaction time, time estimation, visual scanning, memory and conceptual reasoning.[2–12] Dexterity appears the neuropsychological function least affected by mild nitrogen hyperbaric pressure.[3,5,11,13] Some studies have found no evidence that nitrogen narcosis at pressures within sport diving limits leads to significant psychomotor impairment on simple tasks.[3,11,13] Others have reported decreased manual dexterity at depth, which potentially could reduce a diver's ability to operate equipment and increase the risk for mistakes during emergencies. [2,4,6,11,14,15]

Apart from the general challenge of impaired neuropsychological functioning due to nitrogen narcosis at depth, commercial and military diving often takes place in low visibility. Divers thus may need to complete tasks that require the above neuropsychological functions but without the ability to see what they are doing. In a task-environment where visibility has deteriorated, individuals have to rely on tactile perception as a primary mode of data acquisition. To complete more complex tasks, they need to translate tactile information into three-dimensional mental images and solve problems mentally before translating them back into motor actions, with tactile sensation providing feedback. When these actions happen under hyperbaric conditions, factors like nitrogen narcosis could influence a person's ability to successfully complete such tasks.

A search of the available literature did not find any studies describing the effect of hyperbaric nitrogen on complex tactile performance. This study set out to examine: 1) the effect of hyperbaric nitrogen narcosis at a pressure of 608 kPa on tactile performance without visual access to the task, and 2) to investigate the influence of psychological and biographical factors on the above relationship.

Methods

STUDY DESIGN

The study employed a simple, prospective, cross-over design. Participants were randomly allocated to two subgroups. One group was studied first at 101.3 kPa, and then at 608 kPa (equivalent to 50 metres' sea water, msw), while the other group completed the two conditions in the reverse order. Ethics approval was obtained from the Stellenbosch University Health Research Ethics Committee (approval no: N11/06/176).

Table 1
Distribution of academic attainment, diving qualifications
and technical work experience (%)

Academic achievement:

Did not complete high school	9
High school certificate	56
Formal post-school vocational training	21
National diploma or university degree	14

Diving qualification:

Class IV	49
Class III	32
Class II	19

Formal technical work experience:

None	73
1 to 2 years	18
3 or more years	9

PARTICIPANTS

After written informed consent, a total of 139 commercial divers – as part of a larger study on the effects of nitrogen narcosis – completed the procedure in a dry pressure chamber, with participants breathing air and dressed in loose coveralls. The study included mainly younger divers (mean age 26.8 ± 5.9 years, range 18 to 44 years), mostly male (87%), and with little previous deep diving exposure. Their academic attainment, diving qualification, and formal technical work experience are presented in Table 1. The majority of the group had no formal technical qualifications (83.5%). Participants were also asked to self-rate their technical competence.

MEASURES

Tupperware Neuropsychological Task (TNT)

Tactile performance was measured using the Tupperware Neuropsychological Task (TNT). The TNT is based on the Tupperware™ *Shape O Toy*, which consists of a round ball that has 10 different shapes cut out of it, and 10 three-dimensional shapes that correspond to the cut-outs. The purpose is to fit the correct shapes into the cut-outs. To isolate any visual effects, the TNT was completed with the subject wearing a blacked-out facemask, without prior opportunity to see the ball or shapes. Participants' scores were the total number of shapes completed in 10 minutes.

The TNT incorporates the classical elements of performance sequencing, namely: 1) perception, 2) information processing and 3) motor response.[16] The TNT's association with the underlying constructs of tactile form perception, three-dimensional spatial perception and fine motor manipulation has been established earlier, and no significant correlations with age, education, anxiety or sex effects have been found.[17,18]

State-Trait Personality Inventory, Trait Anxiety (STPI)

Trait anxiety was measured with the STPI, Form Y.[19] The STPI is a self-administered questionnaire designed to measure dispositional anxiety in adults. The 10-item trait anxiety subscale was used in this study.[19]

Brunel Mood Scale (BRUMS)

Transient mood states (including state anxiety) were recorded with the BRUMS. It is a 24-item scale that measures six identifiable affective states through a self-report inventory. It taps the mood states of tension, depression, anger, vigour, fatigue and confusion.[20]

Biographical questionnaire (BQ)

A questionnaire recorded age, sex, formal education (academic, technical and diving qualifications) and technical proficiency (experience in formal technical work environments and self-graded technical competency). Age and education may affect the time to complete tactile performance tests,[21] and technical skills, through over-learning, may be particularly resistant to the effects of depth-induced narcosis.

STATISTICAL ANALYSIS

The effect of hyperbaric nitrogen at 608 kPa on TNT performance was analysed with a repeated measures ANOVA. The association of neuropsychological performance under hyperbaric conditions with psychological factors, age and self-rated technical competence was explored using correlational statistics. Its association with gender, qualifications and formal technical work experience was explored using Student's *t*-tests and one-way ANOVAs. Correlational statistics was also used to determine the interaction of surface performance and performance at depth.

Results

The STPI had a mean score of 16.4 ± 3.9, comparable to the scores in the manual. BRUMS scores represented the typical desired iceberg profile. There were no significant correlations with age, sex differences or differences between the two subgroups on either the STPI or the BRUMS. There was no violation of the assumption of sphericity. The TNT results are shown in Table 2 and Figure 1. Performance deteriorated significantly at depth compared to the surface, irrespective of the sequence of the dives (shallow/deep or deep/shallow), the mean decrement being 9.5%. Further, the order of the two pressure exposures did not show a statistically significant effect on TNT performance.

There was a significant correlation between TNT scores on the surface and at depth ($r = 0.71$, $P < 0.001$). Tactile performance on the surface appears a good predictor of

Table 2
Tupperware Neuropsychological Task (TNT); results from repeated measures ANOVA; ηp^2 (partial eta squared) indicates effect size

	Wilk's Lambda	F	df	P	η_p^2
TNT Condition	0.683	63.694	1,137	0.001	0.317
Condition*Sequence	0.993	0.983	1,137	0.323	0.007

TNT [mean total (SD)]	Surface		Depth		n
Group 1 Surface – Depth	8.69	(1.37)	7.99	(1.58)	70
Group 2 Depth – Surface	8.30	(1.51)	7.41	(1.89)	69

Figure 1
Mean (95% confidence interval, CI) performance on the Tupperware Neuropsychological Task (TNT) by 139 divers at pressures of 101.3 kPa and 608 kPa with different sequences for the two dives
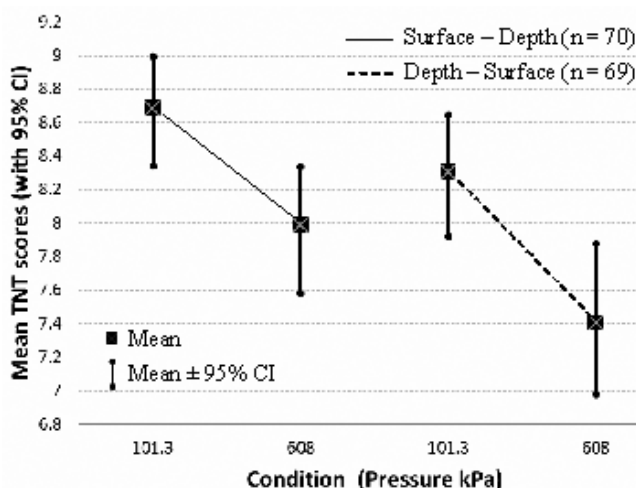
performance under hyperbaric pressure at 608 kPa.

No significant correlations between the STPI or BRUMS and performance at depth were found. Further, none of the biographical variables had any significant effect on performance.

Discussion

The decrement of performance on the TNT at depth was similar for all participants, independent of their psychological or biographical profile, indicating that any effects can be attributed to nitrogen narcosis alone. Since motor dexterity is considered less affected by narcosis than other aspects of neuropsychological performance, impairment of performance on the TNT could probably be attributed to the cognitive processing of tactile performance. The results obtained with the TNT are consistent with predictions from the slowed processing model.[22]

The TNT is a complex task, requiring tactile perception, mental translation and manipulation, planning and psychomotor execution, with feedback and then a repeat of the process. This task complexity makes it particularly susceptible to the cumulative effects of slowed information processing. In the light of the performance sequencing referred to earlier, the consequences of slower task completion would hold true for many underwater tasks in the commercial diving industry.

It has been demonstrated that elevated anxiety may have a significant effect on manual dexterity, quite separate from any narcotic effect.[14,15] Anxiety exacerbates the effects of narcosis, and especially the impairment of psychomotor functioning.[6,15,23,24] The anxiety effects reported in previous studies were not replicated here.[14,15,23] This may in part be due to both the sample composition and the diving environment.

Firstly, previous studies generally used sport divers, whereas the present study used commercial divers. It is hypothesised that more individuals with high anxiety would self-select out of commercial diving. There is support for this from the present sample, in the form of the small range of anxiety scores, clustered on the lower end of the scales of both trait and state anxiety measures (data not shown). Secondly, the study took place in a dry hyperbaric chamber, generally regarded as a low-threat environment considered not to have the anxiety-provoking effect of the open water.[27]

Environmental (open water, temperature) and individual (psychological and biographical) factors further influence the relationship between performance and narcosis. The effect of narcosis on performance is greater under wet than dry conditions.[23] Inside a hyperbaric chamber, the psychological stressors of ocean diving are absent, as well as the muscular strain and environmental conditions (buoyancy, temperature) encountered there.[26] Open water further elicits performance interference from the water effect, i.e., movement in a viscous medium and the buoyancy effects in a tractionless setting that affects the use of tools, as well as anxiety around potential dangers.[16]

Thus, results from protected environments cannot always be transferred directly to open-water conditions.[23] It is generally accepted that the greater effect of nitrogen narcosis in the open water, relative to hyperbaric chambers, can largely be attributed to increased anxiety.[14] It is recognised

that results from dry chambers cannot be transferred directly to open-water conditions.[23,26] Thus, the hyperbaric nitrogen effect found in this study would most likely be amplified in an open-water situation.

The most severe stress in diving is cold exposure. The relationship between cold-water exposure and performance on tasks involving tactile sensitivity, grip strength, and finger dexterity has been well established, with divers' tactile sensitivity decreasing linearly with decreased finger skin temperature at 40 msw.[16,27] While cold itself adversely affects performance, protective measures (e.g., gloves) limit tactile sensation and fine manual manipulation even further.[16]

Although there is no evidence available that age, sex, formal education or previous technical exposure play any significant role in moderating the effect of hyperbaric nitrogen, it can be hypothesised that formal exposure, through a process of over-learning of principles or practices, might protect against impaired performance in tasks requiring technical reasoning or manual dexterity. The absence of reported evidence that age, training or formal technical work play a role in moderating the effects of nitrogen narcosis was maintained in this study. As with previous studies, no gender effect of nitrogen narcosis on performance has been found, although women formed only a small proportion of the sample.[28] However, the variation within this sample on the psychological and biographical markers was very small, and it cannot be concluded that these variables would not influence performance in other samples.

Future studies could attempt to replicate the effects on tactile performance in open-water conditions, with the added considerations of muscular strain (and associated tactile sensations), buoyancy, water viscosity, and temperature and associated protective clothing. Further, future studies need to explore the role of tactile and psychomotor practice effects, particularly the optimal amount of rehearsal required to counter the effects of hyperbaric nitrogen. This needs to be done on land (where practice in reality will generally take place), and in shallow water, to explore the practice effects of not only the number of repetitions, but also of the environment.

Conclusions

A complex neuropsychological task requiring tactile input (the Tupperware Neuropsychological Task), performed without visual aid, was impaired by a mean of 9.5% at 608 kPa in a dry pressure chamber compared with 101.3 kPa. These findings have important practical implications for professional divers in both the commercial and military diving industries, where conditions of low visibility during deeper diving typically occur. To compensate for this, divers would benefit by firstly planning more time to complete complex tasks (especially in low visibility), and secondly by practicing those tasks prior to the actual deep dive, either

on the surface or in shallow water. In this regard, 'blind' performance on the surface was a good predictor of blind performance at depth. Pre-dive practical learning (rehearsal) as an aide to successful completion of that task may be helpful, especially with more complex tasks.

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The database of randomised controlled trials in hyperbaric medicine maintained by Michael Bennett and his colleagues at the Prince of Wales Hospital Diving and Hyperbaric Medicine Unit, Sydney is at:
<http://hboevidence.unsw.wikispaces.net/>

Assistance from interested physicians in preparing critical appraisals is welcomed, indeed needed, as there is a considerable backlog. Guidance on completing a CAT is provided. Contact Associate Professor Michael Bennett: <M.Bennett@unsw.edu.au>

Lung function after cold-water dives with a standard scuba regulator or full-face-mask during wintertime

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Abstract

(Uhlig F, Muth CM, Tetzlaff K, Koch A, Leberle R, Georgieff M, Winkler BE. Lung function after cold-water dives with a standard scuba -regulator or full-face-mask during wintertime. *Diving and Hyperbaric Medicine*. 2014 June;44(2):70-73.)

Introduction: Full-face-masks (FFM) prevent the diver's face from cold and can support nasal breathing underwater. The aim of the study was to evaluate the effect of the use of FFMs on lung function and wellbeing.

Methods: Twenty-one, healthy, non-asthmatic divers performed two cold-water dives ($4^{\circ}C$, 25 min, 10 metres' depth) – one with a FFM and the other with a standard scuba regulator (SSR). Spirometry was performed before and after each dive and well-being and cold sensation were assessed after the dives.

Results: Significant decreases in forced vital capacity (FVC), forced expiratory volume in one second (FEV_1) and mid-expiratory flow at 75% of FVC (MEF_{75}) occurred after both FFM and SSR dives. Changes in FVC and FEV_1 did not differ significantly between FFM and SSR dives. However, the mid-expiratory flows measured at 50% and 25% of FVC (MEF_{50} and MEF_{25}) were significantly lower 10 minutes after the FFM dive compared to 10 minutes after the SSR dive. The well-being and cold sensation of the divers were significantly improved with FFM dives compared to SSR dives.

Conclusions: Cold-water dives during wintertime can be associated with airway narrowing. During cold-water dives, the use of a FFM appears to reduce the cold sensation and enhance the well-being of the divers. However, a FFM does not appear to prevent airway narrowing in healthy, non-asthmatic subjects.

Key words

Cold, scuba diving, lung function, physiology, diving reflex, thermal problems (hypothermia and hyperthermia)

Introduction

Even though the majority of recreational diving is performed in warm water, a considerable number of scuba divers also conduct cold-water dives. Diving during wintertime and in cold water is associated with a heat loss from the body and the face. While the face of commercial divers is usually protected from cold stress by diving helmets or full-face-masks (FFM), standard scuba regulators (SSR) and diving masks are frequently used by recreational divers.

Apart from covering and protecting the entire face from cold water, a FFM offers the possibility of nasal breathing. Consequently, the ventilated air can be humidified and warmed in the nose and nasal pharynx before reaching the lungs. Nasal breathing as well as the use of face-masks has been reported to reduce the likelihood of airway narrowing in non-diving subjects with exercise-induced asthma.[1] Enforced mouth breathing can decrease the lung function in susceptible subjects.[2] A FFM provides some thermal insulation of the face and might, therefore, reduce both heat loss and reflexes induced by facial cooling.[3,4] Since the inhalation of cold, dry breathing air has been reported to foster airway narrowing, it may increase the risk of pulmonary barotrauma during the ascent.[5] The use of a FFM might, therefore, reduce the effects of cold-water dives on the respiratory system.

The aim of the present study was to evaluate changes in lung function after a cold-water dive during wintertime and the impact of the breathing system, i.e., use of a SSR or a FFM.

Methods

SUBJECTS

The study was approved by the Committee on Human Ethics of the University of Ulm, Germany (approval no. 290/10). Twenty-six subjects provided written, informed consent before participating. All subjects were certified adult professional rescue divers or diving instructors. Five divers were excluded from statistical analysis. One did not attempt the first dive because of feeling unwell. In another diver, the FFM regulator froze during his FFM dive and resulted in a free-flow (downstream) failure. Three divers were sick on the second weekend. Accordingly, 21 male divers completed both dives. The individual diving experience was 438, range 118–1,160 logged dives. All were non-smokers and none suffered from bronchial asthma or other chronic obstructive pulmonary disease. All divers successfully passed a medical fitness-to-dive examination prior to the study.

EXPERIMENTAL PROTOCOL

Height and body weight were measured and spirometry performed 45 and 15 minutes pre dive. The divers performed two dives in random order in a cross-over design: 11 divers performed the FFM dive first and the SSR second, and 10 divers in the reverse order. An interval of seven days between dives and the same time of day for diving performance were chosen in order to control for possible variations in circadian lung function. Prior to each dive a period of relative rest for 24 h and no diving for 72 h was required.

The subjects used the mask they were most experienced with, either the Interspiro MK II (Interspiro AB, Täby, Sweden) or the Dräger Panorama Nova Dive (Dräger Safety AG, Luebeck, Germany). The corresponding standard scuba regulators were used for the SSR dives. During diving in a cold lake in wintertime (25 min at a depth of approximately 10 metres), the divers were equipped with a dive computer (Scubapro Uwatec AG, Henggart, Switzerland) for the measurement of ambient water temperature and diving depth. During the FFM dives, the divers were instructed to breathe through the nose exclusively. Ascent and descent times were specified with 1 min for each. The divers were secured with a signalling line. In the case of FFM dives, a special phone line was used for verbal communication. The divers ascended 24 minutes after descent.

After surfacing, spirometry was performed 10, 20 and 30 minutes post dive. While being transferred to the measurement room, the divers continued to breathe via the FFM or SSR. The divers quantified their well-being during the dive according to a visual analogue scale, 0–10 (0 – very uncomfortable, 10 – very comfortable). The divers' sensation of cold was quantified in the same way (0 – not cold at all, 10 – extremely cold) after each dive.

SPIROMETRY

Spirometry was performed whilst standing, wearing a nose clip. The spirometer (Jaeger Master Scope Pneumotachograph, Viasys Healthcare GmbH, Wuerzburg Germany) was volume-calibrated before each spirometry and the following parameters were measured: forced vital capacity (FVC), forced expiratory volume in 1 s (FEV_1) and mid-expiratory flow at 75%, 50% and 25% of FVC (MEF_{75}, MEF_{50} and MEF_{25}). For each measurement, the best of three consecutive trials differing in FVC and FEV_1 by no more than 5% was selected for analysis. Baseline lung function was computed as the mean of the 45-min and 15-min pre-dive measurements and post-dive lung function was defined as the mean value measured 10, 20 and 30 min post dive.

STATISTICS

Microsoft Excel™ 2007 (Microsoft Inc, Redmond, Washington, USA) and SPSS 19 (IBM Inc., Armonk, NY, USA) were used for statistical analysis. All variables were tested for normal distribution using a Kolmogorov-Smirnov test. The pre-dive values of each spirometric parameter were assessed with an ANOVA. Pre- and post-dive lung function within each group and the absolute and relative changes between both groups were compared by paired Student's *t*-tests. A paired Student's *t*-test was also performed to compare the spirometric changes in subjects using the Interspiro MK II mask to those using the Dräger mask. The FEV_1 and FVC, heart rate, diving depth and ambient water temperature were analyzed by a two-factorial (breathing system and time of measurement) ANOVA for repeated measures and a Bonferroni adjusted post-hoc

analysis was performed. For mid-expiratory flows, a three-factorial ANOVA was performed (breathing system, time of measurement and MEF-type 75/50/25).

A relative decrease of at least 10% in FEV_1 was considered to be a clinically relevant degree of airways narrowing according to the ATS guidelines for exercise challenge testing.[6] Based on this criterion, both FFM and SSR dives were separated into two groups (bronchoconstriction after dive and no bronchoconstriction after dive). Differences between the bronchoconstriction and no-bronchoconstriction groups concerning dive specifics and anthropometric data were assessed by independent-samples Student's *t*-tests.

The wellbeing and cold-sensation between the SSR and FFM groups were compared by Wilcoxon signed rank tests. The interaction of bronchoconstriction and wellbeing or cold sensation was tested by a Mann-Whitney U test. Data are presented as mean ± standard deviation (range). Statistical significance was assumed with *P*-values ≤ 0.05.

Results

The mean age of the 21 subjects was 37.2 ± 9.7 (range 20–55) years, height 177 ± 7 (range 158–188) cm and weight 84.4 ± 14.4 (range 55–112) kg. The body mass index (BMI) was 26.6 ± 3.4 (range 20.8–32.4) kg·m^{-2} and body surface area 2.0 ± 0.2 (range 1.5–2.4) m^2.

The average depth reached during the dives was 9.7 ± 0.5 (range 8.9–10.5) metres in FFM dives and 9.7 ± 0.4 (range 8.9–10.5) metres in SSR dives. The mean ambient water temperature was 3.7 ± 0.5 (range 3.2–5.2) °C (air temperature 3.8 ± 2.5, range 0–7.5 °C) for the FFM dives, and 3.8 ± 0.7 (range 2.8–5.6) °C (air temperature 3.7 ± 2.6, range 0.2–8.2 °C) for the SSR dives.

Table 1 presents the pre- and post-dive values for pulmonary function parameters. There were no significant differences in the pre-dive values for any spirometric parameter. Both groups showed significant decreases in FVC, FEV_1 and MEF_{75} post-dive compared to pre-dive. Pre- and post-dive MEF_{50} and MEF_{25} were not significantly different. The pair-wise comparison of absolute and relative changes in lung function values did not reveal any significant differences between FFM and SSR dives. Furthermore, the spirometric changes in subjects using the Interspiro mask did not differ significantly from those using the Dräger mask.

The two-factorial ANOVA demonstrated significant time effects for post-dive FVC and FEV_1. The post-hoc analysis demonstrated a significant difference between 10 and 20 minutes post dive for FEV_1 but not for FVC and did not show a difference between FFM and SSR. The three-factorial ANOVA for post-dive mid-expiratory flows revealed significant effects for all three MEF values (MEF_{75}, MEF_{50} and MEF_{25}) and a significant interaction between MEF type and time, but no effect for time and type of breathing

Table 1

Pre- and post-dive spirometric values, mean (SD), and relative changes and significance levels for forced vital capacity (FVC), forced expiratory volume in one second (FEV_1) and mid-expiratory flows at 75, 50 and 25% of FVC (MEF_{75}, MEF_{50}, MEF_{25}) after dives breathing from either a full-face-mask or a standard scuba regulator; *P*-values are derived from paired-samples *t*-tests (pre-dive vs. post-dive)

	Pre-dive		Post-dive		Relative change (%)		*P*-value
Full-face-mask							
FVC (L)	6.08	(1.00)	5.85	(1.04)	-4.1	(3.4)	<0.01
FEV_1 (L)	4.77	(0.96)	4.56	(0.99)	-4.5	(4.0)	<0.01
MEF_{75} ($L \cdot s^{-1}$)	8.98	(2.43)	8.54	(2.60)	-5.1	(10.6)	0.017
MEF_{50} ($L \cdot s^{-1}$)	5.10	(1.78)	4.91	(1.91)	-4.4	(10.4)	0.144
MEF_{25} ($L \cdot s^{-1}$)	1.86	(0.88)	1.77	(0.77)	-3.0	(12.1)	0.121
Standard scuba regulator							
FVC (L)	6.08	(0.99)	5.83	(0.99)	-4.2	(3.2)	<0.01
FEV_1 (L)	4.79	(0.99)	4.61	(0.99)	-3.8	(2.9)	<0.01
MEF_{75} ($L \cdot s^{-1}$)	9.13	(2.41)	8.52	(2.43)	-6.9	(6.5)	<0.01
MEF_{50} ($L \cdot s^{-1}$)	5.30	(2.06)	5.12	(2.06)	-3.8	(7.6)	0.059
MEF_{25} ($L \cdot s^{-1}$)	1.91	(0.88)	1.88	(0.88)	-1.1	(9.4)	0.559

regulator. The post-hoc analysis did not show general differences between the breathing regulators but a significant difference between FFM and SSR in the MEF_{50} and MEF_{25} measured 10 minutes after the dive. Mean MEF_{50} and MEF_{25} were 4.91 and 1.77 $L \cdot s^{-1}$ 10 minutes after the FFM vs. 5.12 and 1.88 $L \cdot s^{-1}$ after the SSR dive.

Subjects with post-dive bronchoconstriction did not significantly differ from those without with respect to any demographic parameters. Wellbeing scores were significantly higher during and after FFM dives. Cold sensation was significantly more pronounced after SSR dives (6.4 ± 1.9, range 3–9 and 5.1 ± 2.0 range 2–9 respectively; *P* = 0.03) than after FFM dives. (7.3 ± 1.6, range 4–10 and 5.1 ± 1.5 range 3–8 respectively; *P* <0.01). There was no significant relation between bronchoconstriction and wellbeing after the dives (FFM dives *P* = 0.79; SSR dives *P* = 0.95) or bronchoconstriction and cold-sensation (FFM dives *P* = 0.27; SSR dives *P* = 0.61).

Concerning diving depth and ambient water temperature, two-factorial ANOVA did not show any effects for the time of measurement or for the breathing system used. Thus, comparable diving profiles and conditions can be assumed for both breathing systems.

Discussion

The present study revealed significant decreases in lung function after cold-water dives during winter time. The mean changes in expiratory flows and volumes were small and most likely not clinically relevant. Nevertheless, individual subjects demonstrated more pronounced respiratory effects and met the diagnostic criteria of the American Thoracic Society for exercise-induced bronchoconstriction.[6] The literature is inconsistent concerning lung function changes after scuba diving. On the one hand, previous studies have reported that breathing and diving at shallow depths does not have an impact on static and dynamic lung function

parameters after a single scuba dive.[7–8] On the other hand, scuba diving has been reported to be associated with a decrease in spirometric values directly after diving and as a long-term effect.[7,9–12] Breathing cold, dry breathing gas might trigger bronchoconstriction.[5,13–14] One study reported significant changes in FEV_1 during winter dives but not during summer dives.[15]

When using a FFM, the divers were instructed to breathe through the nose. Hence, the air entering the lung is likely to be warmer and more humid when using a FFM than when breathing via the mouth during SSR dives. This assumption is in line with previous studies in non-diving subjects with exercise-induced asthma in whom it was reported that nasal breathing and the use of face masks reduces airway narrowing and the likelihood of asthma attacks.[1] In contrast, enforced mouth breathing – as performed during SSR dives, can decrease lung function in susceptible subjects.[2]

Surprisingly, the use of a FFM resulted in a similar, possibly slightly more pronounced (MEF_{50} and MEF_{25} 10 minutes after the dive) airways narrowing than diving with a conventional SSR. It is possible that factors other than the humidity and temperature of the inspiratory gas during scuba diving may be responsible for the changes in expiratory flows and volumes measured after dives, for instance, intrapulmonary fluid redistribution due to immersion, inspiratory resistance from the regulators and the increased dead space of a FFM. However, most previous studies investigating the respiratory effects of cold, dry air were performed in susceptible subjects with exercise-induced asthma. In contrast to the non-smoking and non-asthmatic healthy subjects that participated in the present study, subjects susceptible to the cold and dryness stimuli might have benefitted from the use of a FFM. We tried to include a group of asthmatic scuba divers at an early stage of the study design but the conduct of these experiments was considered potentially dangerous to susceptible subjects because of a possible increased risk of pulmonary barotrauma. Based on the current findings in

healthy subjects, divers with pre-existing exercised-induced bronchoconstriction might indeed be at an increased risk for airway narrowing and injury in cold-water dives. The use of a FFM does not appear to reduce the adverse respiratory effects of cold-water diving observed in this study.

Hyperoxia has also been reported to foster bronchoconstriction.[16] However, the diving profiles were comparable and hyperoxia is unlikely to play a relevant role. Furthermore, ambient cold is also believed to contribute to airway narrowing but the ambient conditions of depth and water temperature were comparable during both dives. Hence, the thermal effects cannot sufficiently explain the decrease in expiratory flows and volumes after FFM dives in this study. The cold sensation of the divers was significantly lower and individual wellbeing was higher during FFM dives but did not reduce the spirometric responses.

Conclusions

Cold-water (3–5°C) scuba diving resulted in a decrease in expiratory flows and volumes that may be clinically relevant in individual subjects. The use of a FFM reduced the cold sensation and enhanced the wellbeing of the divers. However, FFM diving did not appear to prevent the airway narrowing observed after these cold-water dives. The use of a FFM is unlikely to reduce the risk of bronchoconstriction-associated pulmonary barotrauma in healthy subjects. Subjects with susceptible airways might potentially benefit from the use of a FFM because airway irritation by cold, dry air might play a more pronounced role in these subjects. However, asthmatic divers were not included in the present study for ethical considerations. Further studies are required to investigate the respiratory effects of cold-water diving, especially in subjects who might be more at risk for airway narrowing and, therefore, pulmonary barotrauma.

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Exercise intensity inferred from air consumption during recreational scuba diving

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Abstract

(Buzzacott P, Pollock NW, Rosenberg M. Exercise intensity inferred from air consumption during recreational scuba diving. *Diving and Hyperbaric Medicine*. 2014 June;44(2):74-78.)

Introduction: Episodic exercise is a risk factor for acute cardiac events and cardiac complications are increasingly recognized in fatalities during recreational scuba diving. What is not known is the exercise intensity involved in typical recreational diving.
Methods: This study used pre- to post-dive gas cylinder pressure drop to estimate air consumption and, from that, exercise intensity during recreational dives. Dive profiles were captured electronically and divers self-reported cylinder pressure changes, perceived workload, thermal status and any problems during dives. Mean surface air consumption (SAC) rate per kg body weight and mean exercise intensity (reported in metabolic equivalents, MET, multiples of assumed resting metabolic rate of 3.5 $mL \cdot kg^{-1} \cdot min^{-1}$) were then estimated. Data are reported as mean ± standard deviation.
Results: A total of 959 recreational air dives (20 ± 9 metres' sea water maximum depth; 50 ± 12 min underwater time) by 139 divers (42 ± 10 y age; 11 ± 10 y of diving; 12% smokers; 73% male) were monitored. Problems were reported with 129/959 dives: buoyancy (45%), equalization (38%), rapid ascent (10%), vertigo (5%) and other (2%). Assuming a 10% overestimate due to cylinder cooling and uncontrolled gas loss, the estimated exercise intensity associated with monitored dives was 5 ± 1 MET. Mean ± 2SD, or 7 MET, captures the effort associated with the vast majority of dives monitored.
Conclusion: Our estimates suggest that uncomplicated recreational dives require moderate-intensity energy expenditure to complete, with a 7-MET capacity generally adequate. Higher levels of aerobic fitness are still strongly recommended to ensure ample reserves. Further research is needed to quantify energetic demands of recreational diving during both typical and emergent events in both experienced and less experienced divers.

Key words

Aerobic capacity, oxygen consumption, physiology, exercise, fitness to dive, diving research

Introduction

Episodic exercise is a risk factor for acute cardiac events and cardiac complications are increasingly being recognized as a common contributing factor in fatalities occurring during recreational scuba diving activity.[1,2] A recent review of the medical assessment records of 200 professional divers found that 81% had at least one cardiovascular risk factor and 66% had an alterable risk factor.[3] While the presence of such risk factors may promote discussion of physical fitness for diving, such efforts are limited by the fact that the exercise intensity involved in a typical range of recreational diving is not well known.

Aerobic capacity ($\dot{V}O_{2\,max}$) is defined as the maximum amount of oxygen that can be consumed per unit time. Describing $\dot{V}O_{2\,max}$ per kilogram body mass ($mL \cdot kg^{-1} \cdot min^{-1}$) eliminates total body size as a confounder. The complicated units associated with $\dot{V}O_{2\,max}$ can be eliminated by converting weight-indexed $\dot{V}O_{2\,max}$ measures into dimensionless metabolic equivalents (MET), multiples of assumed resting metabolic rate (3.5 $mL \cdot kg^{-1} \cdot min^{-1}$). For example, a $\dot{V}O_{2\,max}$ of 35 $mL \cdot kg^{-1} \cdot min^{-1}$ would be divided by 3.5 $mL \cdot kg^{-1} \cdot min^{-1}$ to yield a dimensionless 10 MET capacity (10 MET_{max}).[4] A review of studies actually measuring aerobic capacity in divers found the mean in 14 varied groups of mainly male and mainly experienced divers ranged from 37–57 $mL \cdot kg^{-1} \cdot min^{-1}$ (10.6–16.3 MET).[4] Aerobic fitness was not known to be a problem in these groups, but it is also not

known what fraction of their fitness level was required for routine or exceptional diving conditions. Another point to appreciate is that land-based aerobic capacity may not translate directly to in-water capability. A comparison of maximal performance achieved during a treadmill test and during tethered finning on scuba found significantly lower $\dot{V}O_{2\,max}$ and ventilation volume while on scuba (32 vs 42 $mL \cdot kg^{-1} \cdot min^{-1}$ and 72 vs. 104 $L \cdot min^{-1}$, respectively).[5]

Recommendations for aerobic capacity to be maintained for recreational diving are typically based on expert opinion or consensus in the absence of comprehensive data. The proceedings of the 2010 Divers Alert Network Fatality Workshop concluded, "*It was generally agreed that the metabolic requirement for normal swimming in modest to benign diving conditions was around 4 MET, and a safety margin is gained by having the capacity to sustain a 6-MET exercise intensity.*"[6] This is similar to both the 5.7 mean MET (SEM ± 0.2) estimated from heart rate during exercising dives, and a 7-MET capacity posited by other authors to represent a desirable minimum capacity.[5,7,8]

Efforts to develop field assessment of metabolic demand have been limited, in no small part because of the potential confounders of experience, capability, equipment performance, psychological comfort and an array of environmental conditions. Dwyer investigated the relationship between heart rate and oxygen uptake ($\dot{V}O_2$) and minute ventilation ($\dot{V}_E$) among male divers while

performing three levels of exercise intensity in relatively controlled ocean dives at 203, 304 and 406 kPa.[9] The dives were conducted in temperate waters with the divers wearing wetsuits, weight belts, buoyancy compensators, dual-hose regulators, single 71.2 ft³ cylinders and a single model of relatively-high-torque fins. The subject-divers finned along marked circuits. Heart rate, ventilatory frequency, minute ventilation ($\dot{V}_E$) and oxygen consumption ($\dot{V}O_2$) were determined for the final minute of individual four minute exercise periods at different depths with multiple resistive loads. $\dot{V}_E$ was calculated from the cylinder pressure drop over a test period, effectively similar to how divers describe their air consumption operationally. Our goal was to see if we could use Dwyer's regression formulae with cylinder pressure drop information from recreational dives to estimate mean workloads.

Methods

Adult certified divers making recreational, open-water dives were recruited as previously described.[10,11] Briefly, dive businesses and dive clubs in Western Australia (WA) were invited to participate. A researcher met groups of recreational divers at popular dive sites around the WA coast. The study was approved by the Human Research Ethics Committee of the University of Western Australia (approval # RA/4/1/1664). Written informed consent was obtained.

Dive and diver information was collected using a modified Divers Alert Network (DAN) Project Dive Exploration (PDE) questionnaire and Sensus Ultra data-loggers (ReefNet, Mississauga, Ontario) were attached to the front of each diver's buoyancy compensator to capture pressure-time profiles. Depth to ± 0.01 metres' sea water (msw) resolution (0.3 msw accuracy) and water temperature (± 0.01°C and 0.8°C accuracy) were estimated every 10 seconds.[12] Diver data collected included sex, age, height, mass, certification level, current smoking status, number of years of diving and dive counts within the most recent year. Dive-specific data included dress, thermal comfort ('cold', 'pleasant' or 'warm'), perceived workload ('resting/light', 'moderate' or 'severe/exhausting'), starting and ending cylinder pressures and any problems occurring during dives. Any dives lacking one or more of the required variables (cylinder capacity, start and end pressures, or body mass) were excluded from analysis.

Mean depth was calculated by dividing the total of recorded depths (depth >1 msw) for each dive by the number of samples recorded. This then would include time for divers swimming back to the boat underwater but exclude time spent swimming at the surface. It was assumed that divers at the surface would have temporarily discontinued using scuba and breathed air from the atmosphere, but this could not be confirmed. Surface air consumption was calculated by dividing the gas volume used by the number of minutes spent underwater and the ambient pressure in bar at the mean depth.

Dwyer produced regression equations based on the correlation between $\dot{V}O_2$ and $\dot{V}_E$ at different test pressures. The formula for the 203 kPa trials ($r = 0.79$, standard deviation 0.411) was $\dot{V}O_2 = 0.0256\dot{V}_E + 1.070$. This formula was used in the current study to estimate $\dot{V}O_2$ since it was computed for a depth closest to the mean depth for the current dive series. Dwyer also calculated regression equations for 304 and 406 kPa exposures, but these were not applicable in the present study.

Data were imported into the Statistical Analysis System (SAS) version 9.3 (Cary, North Carolina) for analysis. The analysis was conducted in two phases. Firstly, to investigate the gas consumption rate (SAC) per kg body weight, likely associated variables were fitted to a linear regression model (PROC GLM). These included sex (SEX), age in years (AGE), body mass index (BMI) calculated by dividing each diver's weight by the square of their height (kg·m⁻²), certification status (CERT), current smoking status (SMK), number of dives within the previous year (NUM), time since first diving, in years (TIME) and perceived workload (WORK). Certification was classed as 'basic' for levels requiring fewer than 10 training dives, 'intermediate' for certification requiring 10–20 total training dives and 'leadership' for certifications requiring more than 20 total training dives. Data were stratified by organized group dive and the effect of this was accounted for by retaining a stratification variable (ORG) throughout the backwards elimination of non-significant variables. The initial model built was:

$$SAC_{ij} = \beta_0 + \beta_1 SEX_i + \beta_2 AGE_i + \beta_3 BMI_i + \beta_4 CERT_i + \beta_5 CERT2_i + \beta_6 SMK_i + \beta_7 NUM_i + \beta_8 TIME_i + \beta_9 WORK_{ij} + \beta_{10} WORK_{ij} + \beta_{11} ORG_j \qquad (1)$$

where β_0 = the intercept of the regression. Variables were associated with the diver (subscript i) and/or the group dive on which data were collected (subscript j). The regression equation correlation coefficient of the final model (r) was derived from the square-root of the coefficient of determination (R^2). In the second phase, $\dot{V}O_2$ and mean inferred exercise intensity were estimated using Dwyer's formula, as described previously. Descriptive anthropometric and dive data are reported as mean ± standard deviation (SD), dive certification is reported as percentage within each level and dive experience is reported as median and range, because of the non-normal distribution of years of experience and dives within the previous year. Significance for all statistical tests was accepted at $P < 0.05$.

Results

A total of 1,032 recreational dive profiles were collected from 163 individual divers. SAC was estimated for 959 dives (93% of the 1,032 dives) made by 139 divers (85% of the 163 divers). A total of 73 dive records were excluded due to missing information. All dives were made with compressed air. Minimum water temperature during dives followed

Table 1

Diver demography and dive characteristics; mean (SD) or percent, as appropriate, with median and range for dive experience; BMI – body mass index; Certification = 'basic' if required training dives < 10, 'intermediate' if 10–20 and 'leadership' if > 20

Demography	Males (n = 102, 73%)		Females (n = 37, 27%)		Pooled (n = 139)	
Age (y)	43	(10)	39	(9)	42	(10)
Weight (kg)	88	(15)	67	(13)	82	(17)
Height (cm)	179	(7)	166	(8)	175	(9)
BMI (kg m^{-2})	27	(4)	24	(4)	27	(4)
Smokers (%)	11	(11)	5	(14)	16	(12)
Years of diving	10	(0–40)	6	(0–37)	9	(0–40)
Dives in previous year	25	(0–250)	42	(0–200)	39	(0–250)
Certification (% basic/ intermediate/leadership)	27/33/40		24/35/41		26/34/40	
Dive characteristics						
Maximum depth (msw)	20.7	(9.0)	20.3	(9.4)	20.5	(9.1)
Mean depth (msw)	10.7	(4.3)	10.9	(4.3)	10.8	(4.3)
Total dive time (min)	49	(12)	55	(12)	50	(12)

Table 2

Gas consumption and inferred exercise intensity by perceived workload; mean (SD); BMI – Body mass index; SAC – Surface-equivalent air consumption; $\dot{V}O_2$ – oxygen uptake

	Resting/Light (n = 683)		Moderate (n = 247)		Severe/exhausting (n = 9)		Pooled (n = 939)	
Age (y)	41	(9)	40	(8)	44	(10)	41	(8)
Weight (kg)	84	(16)	78	(17)	78	(16)	83	(17)
Height (cm)	176	(9)	172	(9)	174	(7)	175	(9)
BMI (kg·m^{-2})	27	(4)	26	(5)	26	(5)	27	(5)
Mean depth (msw)	10.6	(4.3)	11.0	(4.4)	10.4	(3.4)	10.7	(4.3)
SAC (VE) (L·min^{-1})	17.4	(5.4)	17.9	(5.5)	22.3	(6.2)	17.6	(5.5)
$\dot{V}O2$ (L·min^{-1})	1.52	(0.14)	1.53	(0.14)	1.64	(0.16)	1.52	(0.14)
SAC (L·kg^{-1}·min^{-1})	0.21	(0.07)	0.23	(0.07)	0.29	(0.06)	0.22	(0.07)
$\dot{V}O2$ (mL·kg^{-1}·min^{-1}) estimate	18.6	(3.6)	20.4	(4.4)	21.5	(3.1)	19.1	(3.9)
Exercise intensity (MET) estimate	5.3	(1.0)	5.8	(1.3)	6.2	(0.9)	5.5	(1.1)

geographic lines, from 15°C in the south of the state to 29°C in the north. Problems were reported with 129 of the 959 dives; buoyancy (45%), equalization (38%) rapid ascent (10%), vertigo (5%) and other (2%). Diver demography and characteristics of the dives are presented in Table 1.

There were 887 data (86%) that were complete for all nine variables in the regression model. A higher SAC was not significantly associated with either sex ($P = 0.25$) or smoking status ($P = 0.08$) but was significantly associated with older age ($P < 0.01$), lower BMI ($P < 0.01$), lower dive certification ($P < 0.01$), higher number of dives in the previous year ($P < 0.01$), fewer years of diving ($P < 0.01$) and higher perceived workload ($P = 0.01$). The final model is shown with the respective coefficients in equation 2 ($r = 0.52$). The dive stratification variable was retained but is not shown because it was not significant ($P = 0.46$) and had low estimated effect ($\beta = -0.00004$):

$$SAC_{ij} = -2.73 + 0.002AGE_i - 0.006BMI_i + 0.054CERT_i + 0.015CERT2_i + 0.0002NUM_i - 0.001TIME_i - 0.052WORK1_{ij} - 0.047WORK2_{ij} \quad (2)$$

Air consumption, inferred oxygen consumption and inferred exercise intensity categorized by subject-perceived workload are presented in Table 2. A trend was apparent for mean exercise intensity to increase with increasing perceived workload (Table 2). Overall ($n = 939$), mean inferred exercise intensity was 5.5 MET, approximately midway between the perceived workloads 'Resting/Light' (5.3 MET) and 'Moderate' (5.8 MET).

To explore the potential influence of thermal status, Table 3 presents air consumption, inferred oxygen consumption and inferred exercise intensity categorized by subject-perceived thermal comfort. Regardless of perceived thermal status, mean inferred exercise intensity for 'Cold', 'Pleasant' and 'Warm' were all also between 5 and 6 MET (Table 3).

Table 3
Gas consumption and inferred exercise intensity by perceived thermal comfort; mean (SD); BMI – body mass index;
SAC – Surface-equivalent air consumption; $\dot{V}O_2$ – oxygen uptake

	Cold ($n = 105$)		Pleasant ($n = 527$)		Warm ($n = 324$)		Pooled ($n = 956$)	
Age (y)	39	(9)	42	(9)	40	(7)	41	(8)
Mass (kg)	74	(16)	85	(16)	82	(17)	83	(16)
Height (cm)	170	(9)	175	(10)	176	(9)	175	(9)
BMI (kg·m^{-2})	25	(4)	28	(5)	26	(4)	27	(4)
Time (min)	51	(11)	50	(12)	51	(13)	50	(12)
Mean depth (msw)	11.4	(4.5)	11.0	(4.4)	10.3	(3.9)	10.8	(4.3)
Max depth (msw)	21.1	(8.8)	21.0	(9.0)	20.1	(9.2)	20.6	(9.1)
SAC ($\dot{V}_E$) (L·min^{-1}) estimate	16.0	(5.0)	17.5	(5.2)	18.5	(5.6)	17.7	(5.4)
$\dot{V}O_2$ (L·min^{-1}) estimate	1.48	(0.13)	1.52	(0.13)	1.54	(0.15)	1.52	(0.14)
SAC (L·kg^{-1}·min^{-1}) estimate	0.22	(0.07)	0.21	(0.07)	0.23	(0.07)	0.22	(0.07)
$\dot{V}O_2$ (mL·kg^{-1}·min^{-1}) estimate	20.8	(4.2)	18.5	(3.7)	19.4	(3.9)	19.0	(3.9)
Exercise intensity (MET) estimate	5.9	(1.2)	5.3	(1.1)	5.5	(1.2)	5.4	(1.1)

Discussion

We estimated oxygen consumption from gas cylinder pressure drop in 959 recreational dives conducted in temperate to tropical open water. Higher SAC was significantly associated with older age, lower BMI, lower dive certification, higher number of dives in the previous year, fewer years since first diving and greater perceived workload. That lower certification level was associated with higher gas consumption is in keeping with the assumption that additional training and experience improves diving skills and, thus, lowers gas consumption. It was also not surprising that a higher SAC was associated with newer divers (fewer years of experience). It was not expected that divers who had made more dives in the previous year would have a higher SAC. It is possible that this reflects a higher degree of difficulty associated with the dives conducted, but this cannot be confirmed by the current data.

The BMI data are most difficult to interpret. As a simple ratio of body mass to height, BMI establishes neither body composition nor physical fitness, but nevertheless, it is reasonable to accept that BMI is positively correlated with body fat. This does not help much for interpretation since the use of external weighting and thermal protection may confound commentary on potential differences in buoyancy and/or thermal protection that might be postulated based on BMI. Further research is required to determine the precise role BMI plays in the energetics of recreational diving.

Applying Dwyer's regression formula to this sample of recreational dives provided an estimate of aerobic effort. The accuracy is unconfirmed, but the fundamental pattern is as expected with the mean MET levels increasing across self-reported levels of perceived effort. Still, the validity and practical utility of the results are open to question given the relatively small absolute difference between dives with varying degrees of effort.

Several factors are most likely to lead to an overestimate for any computation based on tank pressure. First, the cooling of a cylinder upon immersion will reduce initial readings. It is common for cylinder pressures to drop 10–20 bar when cooled by immersion, equating to a 5–10% overestimation if the pre-immersion pressure is used as the reference. Second, gas may be lost during surface swimming prior to descent or at the end of the dive. For example, at a SAC of 20 L·min^{-1}, a diver breathing from his cylinder during a 10-minute surface swim would consume 200 litres, raising the exercise intensity estimate by 10%. Third, free flow from a regulator, or additional exhalation to clear a leaking mask could further contribute to non-respiratory losses. Fourth, hyperventilation, driven by anxiety or cold stress below the level of reportability by a diver, could also increase ventilation over the course of a dive. The cumulative effect of such factors could result in a significant overestimation of mean exercise intensity. Differences in self-reported thermal status in the current study did not correlate with differences in $\dot{V}O_2$ or MET estimates (Table 3), with mean inferred exercise intensity narrowly ranged between 5 and 6 MET for all three perceived thermal categories.

Given the uncertainty in our measures, it is prudent to assume a 10% overestimate in gas consumption in this study, reducing the overall mean estimated exercise intensity of 5.4 MET to about 5 MET. With a standard deviation of 1.1 MET, 7 MET (mean plus two standard deviations) would meet the demands of the vast majority of dives we monitored. To consider this as a threshold target for aerobic fitness necessary to meet the demands of uncomplicated recreational dives is consistent with previous recommendations.[4,6,8] Describing a 7-MET capacity as a possible lower-end aerobic fitness target, one review specified that this might be acceptable for divers with strong watermanship skills and comfort.[4] A 10-MET capacity has been recommended for less experienced divers, although balancing temporal with quantitative experience has yet to be defined satisfactorily.[4]

While we have focused on minimum capability targets, it should be remembered that higher levels of aerobic fitness should be encouraged to ensure that exceptional demands arising during any dive can be met.

There are several limitations to this study. Foremost is the rough estimate of tank pressure drop. The divers were not cautioned on the importance of reporting starting cylinder pressures after cooling was complete or to avoid breathing from the cylinder at the surface. Dwyer's methods included swimming at a fixed depth for four minutes while pushing a board and his gas collection took place during only the last minute of steady-rate exercise, whereas our study involved free-swimming recreational divers and our data were averaged over much longer and more variable periods.[9]

Conclusions

Based on estimated breathing gas consumption, a moderate energy expenditure of 7 MET is required to meet the normal demands of almost all uncomplicated recreational dives. Higher aerobic fitness levels are strongly encouraged to meet any emergent demands with ample aerobic reserves. Research into the aerobic demands of a range of recreational diving and for both experienced and inexperienced divers is currently absent and deserving of attention.

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Scuba diving injuries among Divers Alert Network members 2010–2011

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Abstract

(Ranapurwala SI, Bird N, Vaithiyanathan P, Denoble PJ. Scuba diving injuries among Divers Alert Network members 2010–2011. *Diving and Hyperbaric Medicine*. 2014 June;44(2):79-85.)

Background: Scuba diving injuries vary greatly in severity and prognosis. While decompression sickness (DCS) and arterial gas embolism can be tracked easily, other forms of diving injury remain unaccounted for.

Purpose: The purpose of this paper is to assess rates of overall self-reported scuba-diving-related injuries, self-reported DCS-like symptoms, and treated DCS and their association with diver certification level, diving experience and demographic factors.

Methods: We analyzed self-reported data from a Divers Alert Network membership health survey conducted during the summer of 2011. Poisson regression models with scaled deviance were used to model the relative rates of reported injuries. Models were adjusted for sex, age, body mass index (BMI) and average annual dives, based on the bias-variance tradeoff.

Results: The overall rate of diving-related injury was 3.02 per 100 dives, self-reported DCS symptoms was 1.55 per 1,000 dives and treated DCS was 5.72 per 100,000 dives. Diving-related injury and self-reported DCS symptom rates decreased for higher diver certification levels, increasing age, increasing number of average annual dives and for men; they increased for increasing BMI.

Conclusions: Diving injury rates may be higher than previously thought, indicating a greater burden on the diving community. Self-reported DCS-like symptoms are a small fraction of all dive-related injuries and those receiving treatment for DCS are an even smaller fraction. The small number of divers seeking treatment may suggest the mild nature and a tendency towards natural resolution for most injuries.

Key words

Injuries, decompression sickness, epidemiology, health survey, DAN – Divers Alert Network, recreational diving

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Introduction

Scuba diving is a popular recreational activity, which occurs in an unforgiving environment. It is associated with known hazards related to interaction with the marine environment, complex equipment, physical limitations and the human element. Injuries are not uncommon but fatalities are rare. There were about 2.5 million scuba divers in the United States in the year 2000.[1] The lower age limit of divers is 12 years, but there is no upper age limit. Data from the United States and Japan indicate that the scuba diving population is ageing.[2,3] The decline in physical capacity and encroachment of chronic diseases with increasing age may increase diving-associated injuries and fatality rates.

Diving injuries range from mild to fatal. They may be diving-specific like ear barotrauma, decompression sickness (DCS) and arterial gas embolism (AGE), or non-specific, like trauma, envenomation, etc. While a reporting system for diving injuries that provides reliable injury data for comparison with other sports does not exist[4], Divers Alert Network (DAN), the world's largest scuba divers' membership organization, monitors fatalities in the USA and Canada through media outlets and sometimes by requesting access to autopsy and accident reports. A separate ongoing study by DAN Asia Pacific (previously known as *Project Stickybeak*, which had reported Australian diving-related fatalities since the early 1970s) continues to report annually.[5]

The number of fatalities in North America varies between 80 and 90 deaths per year.[6] Based on the DAN AP reports (not including snorkelling deaths), the number of fatalities in Australia during 2000 to 2009 averaged 10 deaths per year.[5] An analysis of DAN insurance claims data estimates the rate of diving fatalities at 16.5 per 100,000 person-years.[7]

The literature on overall diving-related injuries is sparse.[8] DCS and AGE may be tracked through hyperbaric treatment records, while other injuries remain largely unaccounted for. In terms of cost from diving-related injuries, severe DCS, AGE, and fatalities cause the most loss to victims and their families and bear the highest costs of evacuation, treatments and/or repatriation. Other less severe diving-related injuries are usually less costly to treat; however, they are so much more common that the treatment costs and days of productive life lost may amount to a significant burden to the diving community. In August 2011, we conducted an online survey among DAN members that sought to investigate diving-related behaviour and the incidence of diving injuries. This paper explores the relationship between diver demographics, certification, and scuba-diving injuries.

Methods

A DAN membership health survey was administered online as a two-part cross-sectional survey. The survey comprises 20 sections and is too large to be reproduced here; a copy

may be obtained from the corresponding author upon request. One part comprised several modules from the Behavioral Risk Factor Surveillance System (BRFSS) investigating the health status of divers, which will be reported separately.[9] The other part was a survey about diving practices and injuries, a slightly modified version of a survey conducted among dive instructors in Sweden.[10] The original survey was validated, and has been described by the authors.[10] We adjusted this survey for the DAN population by adding options and additional questions applicable to a general diving population. Doing so, we went through multiple phases of critical review by dive medicine experts, instructors, dive medicine technicians and divers. It was piloted among 15 divers for face validation; that is, to see if the respondents understood the questions and answered them appropriately. We randomly selected 30,000 of a little over 150,000 DAN members and invited them to participate via e-mail. Only invited divers had access to the survey and they could respond only once. On-line consent was obtained, which informed the participants that they had to be 18 years of age or older and must have dived at least once during 2010/2011 to be able to participate; if ineligible, they were asked to exclude themselves. Unless they requested exclusion or completed the survey, invitees were reminded three times by weekly e-mails. There was no follow up and the survey was open for two months. No identifying information was collected. This study was approved by the Divers Alert Network (IRB # 001-11, 08 February 2011; re-approved 2012 and 2013).

This retrospective survey focused on participant diving exposures and diving-related injuries in the preceding twelve months over 2010/2011. It also included the number of hyperbaric treatment(s) received or advised for corroborating the diagnosis of DCS. However, some mild cases of DCS may not be identified and/or receive treatment. In an attempt to capture potentially undiagnosed DCS cases, we identified self-reported DCS-like symptoms irrespective of their treatment or referral status to a hyperbaric facility. Self-reported DCS-like symptoms included: loss of muscular strength (paralysis) after diving, skin rash or marbling up to several hours after diving and pain in joints or muscles after diving. The respondents reported their diving-related injuries and the number of dives cumulatively over one year; hence the estimated injury rates from these aggregated data are ecologic in nature.

STATISTICAL METHODS

Descriptive statistics of the collected variables are discussed using tables and frequencies. Body mass index (BMI) was calculated using the self-reported weight and height from the survey and was classified as normal (< 25 kg m^{-2}), overweight (25–30), and obese (> 30).

Injury rates were calculated for self-reported dive-related injuries, self-reported DCS-like symptoms, and treated

DCS using Poisson regression models with scaled deviance among divers with different diving certification levels (basic, advanced, instructor). These rates were adjusted for sex, average annual dives, BMI and age, based on change in estimate and bias-variance tradeoff.[11] Specifically, a covariate was retained in the model if the change in the estimated variance of the diving certification level coefficient was negative upon adjustment for that covariate or positive but smaller than the squared change.

Self-reported injury rates were expressed per 100 dives. We excluded seasickness from the rate calculations because high turbulence in the sea may lead to motion sickness in almost all occupants of a boat.[12] The self-reported DCS-like symptom rates were expressed per 1,000 dives and the rate of treated DCS was reported per 100,000 dives. Rates were compared for different diving certification levels (basic, advanced, and instructor), sex (female and male), 10-year increments in age, BMI categories (normal, overweight, and obese) and 20-dive increments of average annual dives.

Two-sided Student's t-tests were used to compare the age distribution of 'respondents' to that of 'invitees' and to compare the percentage of specific injuries between males and females. We used the Mantel-Haenszel chi-square test to compare the sex distribution of respondents to that of invitees. The SAS 9.3 statistical package (SAS Inc, Cary, NC) was used for descriptive statistics, t-tests, Mantel-Haenszel chi-square test, and Poisson regression analyses.

Results

Out of the invited 30,000 DAN members, 18.4% ($n = 5,514$) responded to the survey, and 16.2% ($n = 4,859$) submitted all 20 survey sections. The respondents who did not submit all the sections of the survey, maybe owing to internet failure or unwillingness to answer the survey any further, were considered self-withdrawn.

The median age of respondents was 52 (range 18–90) years. The mean BMI was 26.9 ± 4.51 kg·m^{-2} (21% obese, 42% overweight, and 37% normal) (Table 1). We compared the age and sex distribution among invitees to the demographic distribution of respondents. The invitee population comprised 74.1% males, and respondents 73.5% males ($P = 0.47$). The mean age of invitees was 48.1 years and that of respondents was 50.2 years, a mean age difference of 2.1 years ($P < 0.001$). This indicates that those who completed the survey were, on average, older than the DAN population.

DIVING PRACTICE

The majority of the divers had advanced level certifications (63%), followed by basic (20%) and instructor level certifications (17%). Respondents made a total of 174,912 dives in the preceding year, mostly performing between 1 and 100 dives (95.7%; median 20 dives, mean 37 dives,

Table 1
Characteristics of the participants by diver certification level; missing values in the table refer to the missing data for
diver certification level of the respondents

	Diver certification level									
	Basic		Advanced		Instructor		Missing		Total	
Variable										
Sex*	n	%	n	%	n	%	n	%	n	%
Male	573	65.0	2,080	73.9	655	82.0	80	71.4	3,388	73.5
Female	308	35.0	735	26.1	144	18.0	32	28.6	1,219	26.5
BMI†										
Normal	326	39.1	1,047	37.7	238	30.4	78	54.5	1,689	37.3
Overweight	333	40.0	1,178	42.4	352	45.0	46	32.2	1,909	42.1
Obese	174	20.9	551	19.8	192	24.6	19	13.3	936	20.6

* 252 participants did not provide sex information
† 325 participants did not provide height and/or weight information

Table 2
Self-reported diving-related injuries by participant's sex; * $P < 0.05$; male vs. female

	Males n (%)	Females n (%)	Missing n (%)	Total n (%)
Ear problems (other than hearing loss)*	733 (19.9)	371 (21.7)	76 (16.1)	1,180 (20.1)
Headache*	543 (14.7)	285 (16.7)	78 (16.6)	906 (15.4)
Sinus troubles	425 (11.5)	180 (10.5)	50 (10.6)	655 (11.2)
Puncture wound, laceration	375 (10.2)	110 (6.4)	43 (9.1)	528 (9.0)
Overexertion, low back pain, sprain/strain*	290 (7.9)	155 (9.1)	52 (11.0)	497 (8.5)
Mask squeeze	302 (8.2)	142 (8.3)	46 (9.8)	490 (8.4)
Pain in joints and/or muscles[1]	175 (4.7)	66 (3.9)	15 (3.2)	256 (4.4)
Itching after diving (< 20 min)	140 (3.8)	66 (3.9)	6 (1.3)	212 (3.6)
Allergic contact dermatitis*	86 (2.3)	103 (6.0)	14 (3.0)	203 (3.5)
Tooth pain	98 (2.7)	30 (1.8)	14 (3.0)	142 (2.4)
Hearing loss	84 (2.3)	36 (2.1)	16 (3.4)	136 (2.3)
Dizziness/giddiness	81 (2.2)	35 (2.0)	13 (2.8)	129 (2.2)
Skin rash/marbling (several h)[2]	79 (2.1)	40 (2.3)	10 (2.1)	129 (2.2)
Dyspnoea	71 (1.9)	21 (1.2)	8 (1.7)	100 (1.7)
Animal bite	55 (1.5)	31 (1.8)	10 (2.1)	96 (1.6)
Crushing injury/fracture*	64 (1.7)	8 (0.5)	6 (1.3)	78 (1.3)
Burns	37 (1.0)	15 (0.9)	4 (0.8)	56 (1.0)
Ankle sprain	31 (0.8)	13 (0.8)	5 (1.1)	49 (0.8)
Loss of muscular strength/ (paralysis)[3]	14 (0.4)	1 (0.1)	1 (0.2)	16 (0.3)
Unconsciousness	3 (0.1)	0 (0)	4 (0.8)	7 (0.1)
Total*	3,686	1,708	471	5,865
Decompression sickness (1+2+3)	192	73	17	282

[1], [2] and [3] – total self-reported DCS symptoms were computed by combining three symptoms: pain in joints and/or muscles, skin rash or
marbling (several hours), and loss of muscular strength (paralysis)

range 1–300 dives). Those who performed more than 100 dives per year were predominantly advanced- and instructor-level divers. The mean and median number of dives was similar for divers of all age groups except for 75 and over, who dived less. Respondents engaged in various types of diving: wreck dives (64.6%), night dives (65%), cave dives (11.4%), planned decompression dives (16.2%), rebreather dives (2.3%), nitrox dives (49.2%), ice dives (4.1%), cold water dives (41%), altitude dives (9.9%), and dives for repair work (8.6%). Over 60% of divers reported diving in low visibility and about 44% reported diving in strong currents.

INJURIES

A total of 5,865 diving-related injuries were reported by 1,580 (32.5%) respondents regardless of whether they

Table 3
Adjusted rate ratios (95% confidence interval) for all injuries, DCS-like symptoms and treated DCS, adjusted for sex, BMI, average annual dives, and diving certification; and overall rates per 100,000 dives for all injuries, DCS-like symptoms and DCS

Variable	Category	All injuries		DCS symptoms		Treated DCS	
Diver certification level	Advanced	0.78	(0.65–0.92)	0.79	(0.62–1.01)	–	
(reference = basic)	Instructor	0.68	(0.55–0.84)	0.81	(0.60–1.10)	–	
Sex (reference = female)	Male	0.67	(0.58–0.77)	0.65	(0.53–0.79)	2.56	(1.75–3.74)
Age	10-years increase	0.89	(0.85–0.94)	0.90	(0.84–0.97)	1.04	(0.95–1.14)
BMI	Overweight	1.10	(0.96–1.27)	2.12	(1.69–2.65)	–	
(reference = normal)	Obese	1.07	(0.91 –1.26)	1.97	(1.54 – 2.54)	–	
Average annual dives	20 dives increase	0.89	(0.87–0.92)	0.84	(0.80–0.88)	–	
Rate per 100,000 dives (95% CI)		3,024	(2,845–3,214)	155	(142–169)	5.72	(5.11–6.39)

*Adjusted for sex, BMI, average annual dives, and diving certification level

received treatment or not. Injuries requiring treatment were reported by 665 (13.7%) respondents; 38 (5.7%) of these respondents claimed insurance. Eleven divers (0.2%) received 18 recommendations for hyperbaric oxygen treatments and in 16 instances this advice was followed. Table 2 shows the proportion of each self-reported diving injury. Ear problems other than hearing loss were the most reported. DCS-like symptoms were reported by 282 respondents (5.8%).

INJURY RATES

The crude rate for all diving-related injuries was 3.02 per 100 dives (95% confidence intervals (CI) 2.85 to 3.21). The adjusted injury rates decreased with increasing age (Figure 1). The crude injury rate for males was 2.65 per 100 dives (95% CI 2.46 to 2.85) and for females was 4.30 per 100 dives (95% CI 3.87 to 4.79), giving a crude male-to-female rate ratio of 0.62 (95% CI 0.54 to 0.70). From Table 3, the adjusted injury rate ratio for males as compared to females was 0.67 (95% CI 0.58 to 0.77).

The overall crude self-reported DCS-like symptom rate was 1.55 per 1,000 dives (95% CI 1.42 to 1.69). Higher diver certification level was associated with a monotonic decrease in all dive-related injuries (Table 3). Compared to basic certification divers, the adjusted rate ratio of all dive-related injuries for instructor divers was 0.68 (95% CI 0.55 to 0.84), and for advanced divers was 0.78 (95% CI 0.65 to 0.92). We considered the diagnosis of DCS confirmed in the 11 respondents who received hyperbaric oxygen treatment (16 treatments). After taking into account multiple treatments for two divers, the DCS incidence rate was 5.72 per 100,000 dives (95% CI 5.11 to 6.39).

Table 3 also shows the comparison of rates between self-reported diving injuries, self-reported DCS-like symptoms, and treated DCS cases by using a common denominator of

Figure 1
Adjusted self-reported injury rates by age of the participants, adjusted for sex, BMI, average annual dives, and diving certification level
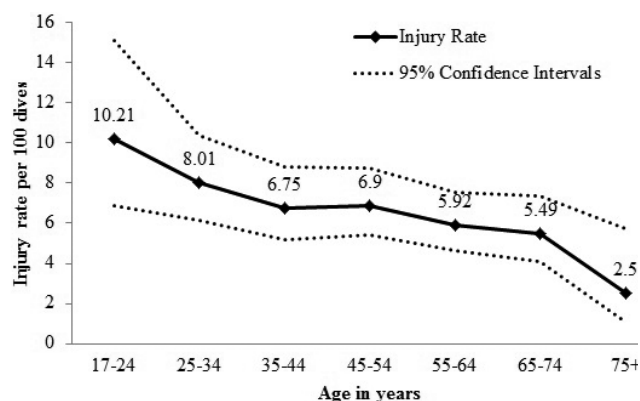
100,000 dives. Thirty-seven per cent of respondents reported diving-related injuries, compared to 4% reporting DCS-like symptoms, and 0.23% receiving hyperbaric treatment. This implies that the incidence rate of all diving-related injuries is almost 20 times greater than the incidence of DCS-like symptoms. Furthermore, the incidence rate of DCS-like symptoms was over 25 times greater than treated DCS cases.

We also compared the injury rates of those who logged their dives (84%) versus those who did not (16%). Divers who log their dives reported fewer injuries (mean 1.15 per person) than those who do not log their dives (mean injuries 1.54). Also, those who log their dives reported fewer dives (mean = 32) during the survey period compared to those who do not log their dives (mean = 47). Consequently, the crude rate ratio of injuries for those who log their dives versus those who do not is 1.09 (95% CI 0.95, 1.25). The adjusted rate ratio for those who log their dives versus those who do not is 1.10 (95% CI 0.95, 1.25).

Discussion

SELF-REPORTED DIVING INJURIES

The crude overall self-reported diving injury rate was 3.02 per 100 dives. The most common injuries included ear troubles, headaches, and sinus troubles. Headache, although not a specific symptom of a dive injury, may be observed in cases of DCS, AGE, sinus or ear barotrauma and carbon dioxide retention. Thus it was included in dive-related injuries but because of unknown context it was not accounted for as DCS-like symptoms.

The higher injury rates amongst 17–24 year-olds compared to older divers could be explained by older divers being more experienced and diving more conservatively. We have no clear explanation for the higher injury rate amongst women compared to men. The inverse relationship between certification level and injury rate could be expected as divers with higher certification levels should have learnt better skills. The only previous study to have reported on all diving-related injuries found much lower rates ratios – 0.56 per 100 dives in 1999 and 0.98 per 100 dives in 2000 – than we have reported in this study.[8] However, they obtained the injury information from dive operators rather than from divers, and the number of dives was calculated based on the number of tanks used.[8]

DECOMPRESSION SICKNESS

The crude incidence of self-reported DCS-like symptoms was 1.55 per 1000 dives. Men reported 35% fewer DCS-like symptoms than women, whereas, when considering treated-DCS rates in our survey, that in men was 2.56 times higher than in women. In the Swedish study, no such effect of sex on DCS symptoms was evident.[10] Men may have a tendency to perform more extreme dives, which lead to more severe cases that need treatment. This observation is supported by other studies which report higher incidence of treated DCS rates in men than in women.[2,13]

Insurance claims data for DCS suggest that the incidence of DCS claims decreases with increasing age, which corroborates our results.[2] This may be attributed to conservative diving habits in older divers; however, this explanation may be inadequate because older divers have higher rates of diving fatalities.[7]

The treated-DCS incidence rate based on our self-reported data was 5.72 per 100,000 dives. This is considerably less than the previously reported incidences of DCS (20 per 100,000 dives in 1999 and 46 per 100,000 dives in 2000) in Orkney, known for deep decompression diving, unlike most recreational diving.[8] These results are also lower than those from a prospective study conducted in Canada for 14 months between 1999 and 2000, in which the estimated decompression illness incidence was 9.57 per 100,000

dives.[14] Both the above-mentioned studies were prospective in nature, and were conducted more than a decade before our study.

Few studies have evaluated self-reported data for scuba -iving-related injuries. Other studies that used a questionnaire evaluated only DCS and did so based on confirmed cases.[13] Only one other study determined the incidence of self-reported symptoms of DCS to be 1.52 per 1,000 dives, for which the diver may or may not have sought medical help.[10] This is similar to our estimate of 1.55 per 1,000 dives. In both reports, skin DCS symptoms were reported more often, unlike reports of treated DCS which carry only a small percentage of skin DCS. Advanced training and age resulted in lower incidence rates of DCS symptoms in the Swedish study.[10] Similar effects of lowered DCS incidence for divers with higher certification levels have been noted by others.[15] The results of our survey are consistent with these data.

In our study, the overall self-reported DCS-like symptom rate was over 25 times higher than the rate of treated DCS. This suggests the presence of mild symptoms of DCS that resolved spontaneously. This is supported by a 1993 Norwegian study of commercial and sports divers in which about 19% of sports divers, 50% of commercial air divers, and 63% of saturation divers reported DCS-like symptoms; however, only 3% of sports divers, 13% of commercial air divers and 28% of saturation divers sought hyperbaric treatment for DCS.[16] Under-reporting of DCS symptoms may be a problem for outcome-based evaluations of diving practices and safety of decompression algorithms.

The incidence of treated DCS in survey-based studies varies between 5 and 30 cases per 100,000 dives.[13,17–19] Field studies reporting DCS incidence rates among smaller diving populations range from 10 to 20 cases per 100,000 dives.[20–22] Navy divers have a similar or higher incidence of DCS compared to recreational divers.[21–23] This variability is small and largely owing to the difference in study populations and their diving practices.

DIVING EXPERIENCE

Diving experience affects dive safety and risk of injury, but it is difficult to quantify. A diver who has dived for many years may be considered more experienced although, in most sports, experience is usually defined as the number of games played in a lifetime. Using lifetime dives would be a better metric to represent diving experience; however, both these metrics fail to account for the frequency of diving. A diver who dives five times a year for 20 years will be considered less experienced than a diver diving 50 dives a year for 2 years. Hence, annual dives may represent diving experience better than number of years diving or lifetime dives. In our analysis we found that an increase of 20 average annual dives was associated with 11% fewer diving injuries and 16% fewer DCS-like symptoms.

LIMITATIONS

We used a survey that was developed for diving instructors and made adjustments to it for a general diving population. The demographic data were collected using questions from the BRFSS.[9] Our survey used these two well-validated surveys with only minor modifications; however, it was not validated in multiple groups of divers. Instead, our survey underwent multiple reviews by diving experts, and we pilot-tested it among a small group of 15 divers for face validation.

Determining incidence rates of diving injuries and DCS remains a challenge because of differences in demographics and diving practices amongst various subgroups. DAN's membership population is composed of divers who are older and more active than the general scuba-diving population, but their diving practices reflect overall practice of that population. In addition, at present, DAN's membership may be the best-known, defined diver population for such a study. However, a longitudinal study would establish injury incidence more reliably. The findings from our study may not be generalizable, but they are indicative of similar trends in the general diving population.

The retrospective study design introduces recall bias and may affect the reliability of the study. There were 1,580 divers who reported symptoms consistent with diving injury, but only 665 sought medical help. There is a chance that the divers may have over-reported or under-reported the frequency of symptoms. It may be assumed that those who log their dives may have better recollection of post-dive symptoms than those who do not. However, there was no difference in the crude and adjusted injury-rate ratios between the two groups.

Ecological analyses limit our interpretation of results, which may be prone to ecological fallacy. By this we mean that we collected diver-level data (the number of dives and the number of symptoms), not dive-level data (depth, time underwater, breathing gas mixture, etc.), although the estimated injury rates are per-dive measures. Caution is advised in the interpretation of these estimates as dive outcomes may be affected by depth and time measures of a dive, which our study could not explore.

Conclusions

The incidence of overall dive-related injuries may be higher than previously thought, but most injuries appear to be mild. DCS-like symptoms make up only about 5% of all reported injuries. The incidence rate of the symptoms consistent with DCS (DCS-like symptoms) was 25 times higher than the incidence rate of DCS cases receiving medical evaluation. This suggests that the incidence of DCS may be higher than that which gets treated or recorded.

Rates of all diving-related injuries and DCS-like symptoms decrease with advancing age. Males report fewer diving injuries than females. Divers with advanced- and instructor-level certification report fewer injuries than basic-level divers.

Prospective cohort studies with better recording of dive exposures and better evaluation of associated outcomes, including all injuries, not only DCS and AGE, may be necessary to attain greater insight into the potential impact of diving injuries. Establishing reporting systems for all diving-related injuries and encouraging divers to report all symptoms as they occur may also yield better data. Injury-prevention strategies such as the use of pre-dive checklists, and encouraging divers to do refresher courses and to attain higher diving certification levels may target all diving-related injuries and yield better health outcomes for scuba divers.

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Physiological effects of rapid reduction in carbon dioxide partial pressure in submarine tower escape

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Abstract

(Loveman GAM, Seddon FM, Thacker JC, White MG, Jurd KM. Physiological effects of rapid reduction in carbon dioxide partial pressure in submarine tower escape. *Diving and Hyperbaric Medicine*. 2014 June;44(2):86-90.)

Introduction: The objective of this study was to determine whether adverse effects from a rapid drop in inspired carbon dioxide partial pressure (P_iCO_2) in the breathing gas could hinder or prevent submarine tower escape.

Methods: A total of 34 male volunteers, mean (SD) age 33.8 (7.5) years, completed the trial. They breathed air for five minutes then 5% CO_2/16% O_2, 79% N_2 ($5CO_2/16O_2$) for 60 minutes before switching to breathing 100% O_2 for 15 minutes and then returned to air breathing. Breathing gases were supplied from cylinders via scuba regulators and mouthpieces. Blood pressure, cerebral blood flow velocity, electrocardiogram and end-tidal CO_2 and end-tidal O_2 were monitored throughout. Subjects were asked at intervals to indicate symptom type and severity.

Results: Symptoms whilst breathing $5CO_2/16O_2$ included breathlessness and headache. Following the switch to 100% O_2 seven subjects reported mild to moderate faintness, which was associated with a significant drop in cerebral blood flow compared to those who did not feel faint ($P < 0.02$). No subject vomited or fainted following this breathing-gas switch.

Conclusions: This study shows that the risk of fainting, sudden collapse or vomiting on switching to 100% O_2 following acute exposures to hypercapnia at a P_iCO_2 of up to 5.0 kPa is less than 8%.

Key words

Hypercapnia, oxygen, cerebral blood flow, Doppler, physiology, submarine

Introduction

In a scenario where the crew of a UK Royal Navy (RN) submarine is unable to surface their vessel, they may attempt escape. The escape tower is an air-lock supplied with diving quality air. The submarine crew member will switch from breathing a possibly hypercapnic and hypoxic atmosphere in the distressed submarine (DISSUB) to a normocapnic (approximately 0.0395 kPa inspired carbon dioxide partial pressure, P_iCO_2) and normoxic atmosphere in the escape tower. Subsequent pressurisation during tower escape means that the escaper will also be exposed to a hyperoxic atmosphere, with the inspired oxygen partial pressure (P_iO_2) reaching as high as 380 kPa at the maximum permitted escape depth (180 metres' sea water, msw).

An early study reported that switching from breathing a hypercapnic gas (6% CO_2) to 100 kPa O_2 resulted in nausea and vomiting in three of six subjects.[1] The authors indicated that the work could have been better controlled. To our knowledge only one other study has examined the effect of this gas switch; using 7 kPa P_iCO_2, it was found that two of 12 subjects vomited shortly after the switch to oxygen breathing.[2] Cerebral hypoperfusion has been associated with nausea.[3] The reduction in cerebral blood flow associated with a rapid reduction in P_iCO_2 combined with a rapid elevation in P_iO_2 could induce nausea and vomiting, in addition to the risk of fainting. Vomiting during the pressurisation or ascent phase of submarine escape would likely result in pulmonary injury and possibly death. The term 'carbon dioxide-off' effect refers to any symptoms that might be experienced by an individual who has been exposed to a high level of CO_2 (a hypercapnic atmosphere) and then switches to breathing a normal (normocapnic) or reduced (hypocapnic) level.

Fainting can be provoked by anything that endangers cerebral perfusion.[4] The switch from breathing a hypercapnic gas in the DISSUB to a hyperoxic gas whilst stood in the submarine escape tower might lead to cerebral hypoperfusion, which could in turn result in fainting. A crew member who faints in the escape tower presents an additional obstacle for fellow crew members to negotiate and furthermore his airway could be compromised.

The purpose of the present study was to determine the risk to escapers with P_iCO_2 of approximately 5.0 kPa and P_iO_2 of approximately 16.0 kPa that might exist in the DISSUB when a switch is made to breathing 100 kPa O_2 (the maximum P_iO_2 that can be delivered under normobaric conditions and equivalent to that experienced in tower escape breathing air at approximately 40 msw tower depth).

Methods

The study was carried out at the QinetiQ Hyperbaric Medical Unit, St. Richard's Hospital, Chichester, UK and was conducted in accordance with the principles of the declaration of Helsinki.[5] An ethical protocol for the study was reviewed and approved by the QinetiQ Research Ethics Committee (approval number: SP774v2.3).

A power calculation (single-sample binomial test, two-tailed, power = 0.8 and $P = 0.05$) showed that 34 subjects would

need to complete a switch from a hypercapnic and hypoxic breathing gas to 100% O_2, without vomiting or fainting, to demonstrate the underlying risk to be less than 8%.

SUBJECTS

Volunteer subjects were requested to fast from 2000 h and to refrain from alcohol for 24 h prior to the morning of the test. They were asked to drink only clear liquids other than taking their usual caffeinated drink in the morning and not to consume any liquids for two hours prior to the test.

PROCEDURE

All tests were carried out at normobaric ambient pressure. A nose clip was worn throughout the test. Each subject sat at rest breathing air from a scuba mouthpiece for 5 min while baseline measurements were taken. The subject then breathed room air for a short period. The subject then commenced breathing a hypercapnic, hypoxic mixture for 60 min. The composition of the mixture was 5% CO_2, 16% O_2, 79% N_2, referred to here as $5CO_2/16O_2$.

A subjective symptoms questionnaire was administered each minute for the first 5 min of breathing $5CO_2/16O_2$, then after a further 5 min and then at 10 min intervals. The subject was required to rate their level of discomfort on a five-point scale – as none, mild, moderate, severe or intolerable – for four symptoms – nausea, breathlessness, faintness and headache.

At 50 min the subject was asked to stand. At 60 min the breathing gas was switched to 100% O_2 and the subjective symptoms questionnaire administered at 1 min intervals for 5 min, then at 2 min intervals. At 75 min the breathing gas was switched to air and the subject asked to sit. At 80 min the test was ended.

INSTRUMENTATION AND MEASUREMENTS

PCO_2 and PO_2 were measured continuously at the centre of the scuba mouthpiece (AMIS 2000 respiratory mass spectrometer, Innovision Denmark). Mean blood flow velocity in the middle cerebral artery ($MCAv_{mean}$) was measured continuously using transcranial Doppler (TCD) (TC-Pioneer EME/Nicolet Vascular), the probe being located at the temporal region above the zygomatic arch. Insonation of the MCA was adjusted to the angle resulting in the highest recorded blood velocity and best-quality Doppler signal.

At 1 min intervals for 5 min after changing breathing gas and 5 min intervals thereafter, mean arterial pressure (MAP) was measured with an automated sphygmomanometer (DINAMAP® Pro 1000, General Electric) at the brachial artery of the right arm. Electrocardiogram (ECG) was continuously displayed on two ECG monitors. The 3-lead monitor of the DINAMAP® Pro 1000 was used to allow display of the lead I signal and a 5-lead monitor (LifePulse10, HME Ltd.) was used to display the lead II signal.

TEST TERMINATION CRITERIA

The test would be terminated:
- at the subject's request;
- on a subjective questionnaire response of 'intolerable' to any aspect;
- on failure of any equipment used to monitor withdrawal variables;
- on recording end-tidal carbon dioxide ($ETCO_2$) > 8.5 kPa for more than five consecutive breaths;
- if the subject began to vomit;
- if the subject requested assistance as feeling severely faint or the subject fainted;
- on subjective signs of impending panic or
- if BP, measured by DINAMAP was greater than either a systolic of 180 or a diastolic of 110 mmHg, sustained for over 1 min.

STATISTICS

The relative percentage changes in respiratory rate, heart rate, MAP, $ETCO_2$ and $MCAv_{mean}$ were calculated for the minute pre-switch to the minute post-switch to 100% O_2. A boxplot was used to determine whether any of these data warranted further statistical analysis. Where this was the case, subject data were grouped according to symptoms and differences between groups tested using the unpaired, unequal variance t-test. Differences were considered significant if $P \leq 0.05$.

Results

SUBJECT DETAILS

A total of 39 male volunteers participated in the trial. The procedure was stopped in six subjects; three because they exceeded the upper BP limits, two whilst breathing $5CO_2/16O_2$ and one on 100% O_2 (the last subject's data were included in the analysis, however); two for increasing ventricular ectopics (not present on their pre-trial ECGs) whilst breathing $5CO_2/16O_2$ and one who was entraining room air around the mouthpiece during the test. The mean (SD) age of the 34 volunteers whose data were used was 33.8 (7.5) years; height 180.7 (5.7) cm; body mass 82.8 (9.1) kg.

SYMPTOMS

No subject vomited, fainted or was incapacitated on the switch to 100% O_2 breathing. Six subjects reported no symptoms throughout the test. Eleven subjects reported mild to moderate headache. Only three subjects reported a headache that developed after the switch to 100% O_2, as opposed to eight whose headache developed whilst breathing $5CO_2/16O_2$; three of these eight found their symptoms of headache resolved following the switch to 100% O_2.

Seven subjects reported mild to moderate faintness occurring only after the switch to breathing 100% O_2, while six reported

Table 1
95% confidence intervals on absolute values of physiological variables ($n = 34$); MAP – brachial BP was measured once per min, other signals were recorded continuously; [*] value taken over 1 min; [†] value taken over 5 min; $MCAv_{mean}$ – mean middle cerebral artery blood flow velocity

	Air baseline[*]	First min[*] $5CO_2/16O_2$	First 5 min[†] $5CO_2/16O_2$	After 30 min[*] $5CO_2/16O_2$	Final min[*] $5CO_2/16O_2$	First min[*] 100% O_2	First 5 min[†] 100% O_2	Final min[*] 100 % O_2	Final min air[*]
Respiratory rate (breaths·min^{-1})	8 ± 1.2	8.9 ± 1.2	9.1 ± 1.2	10.2 ± 1	12.7 ± 1.2	11.6 ± 1.4	9.8 ± 1.4	9.5 ± 2	8.5 ± 1
MAP (mmHg)	94 ± 2	99 ± 4	100 ± 4	97 ± 4	106 ± 4	102 ± 2	99 ± 2	98 ± 4	95 ± 2
Heart rate (beats·min^{-1})	66 ± 4	67 ± 4	67 ± 4	69 ± 4	82 ± 4	84 ± 6	82 ± 6	76 ± 4	65 ± 4
$MCAv_{mean}$ (cm·s^{-1})	52 ± 4	73 ± 6	77 ± 6	71 ± 6	72 ± 8	39 ± 4	38 ± 4	43 ± 6	45 ± 6
$ETCO_2$ (kPa)	5.2 ± 0.2	6.6 ± 0.2	6.9 ± 0.2	6.9 ± 0.2	6.8 ± 0.2	4.3 ± 0.2	4.1 ± 0.2	4.1 ± 0.4	4.4 ± 0.4

Figure 1
Percentage change in physiological variables taken over 1 min before and after switching to 100% O_2 ($n = 34$); heavy lines denote median values; box extents show interquartile range; whiskers denote data values within 1.5 times the interquartile range from upper/lower quartiles
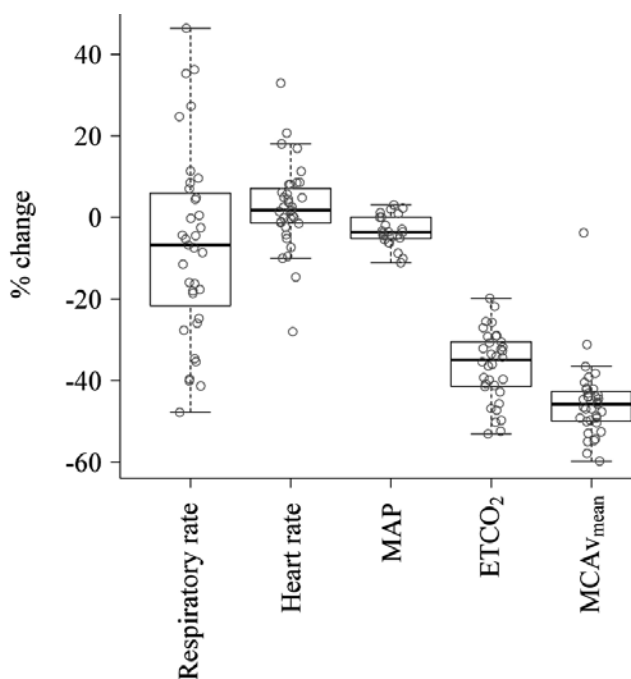
mild faintness whilst breathing $5CO_2/16O_2$. Faintness was the only symptom which developed in an appreciable number of subjects following the switch to 100% O_2. One subject reported mild nausea after the switch to 100% O_2 and one reported mild nausea whilst breathing $5CO_2/16O_2$. Eighteen subjects reported mild to severe breathlessness whilst breathing $5CO_2/16O_2$. Three of these continued to experience breathlessness on the switch to 100% O_2, one of whom rated it as moderate.

CHANGE IN PHYSIOLOGICAL VARIABLES ON SWITCH TO 100% OXYGEN

The values of physiological variables are summarised in Table 1. Figure 1 shows a boxplot of the percentage changes in respiratory rate, heart rate, MAP, $ETCO_2$ and $MCAv_{mean,}$ taken from 1 min pre-switch to 1 min post-switch to 100% O_2. There was a significant difference in the percentage drop in $MCAv_{mean}$ on the switch between subjects experiencing faintness following the switch ($n = 7$, mean reduction in $MCAv_{mean}$ 51%) compared to those who did not ($n = 27$, mean reduction in $MCAv_{mean}$ 44%) ($P < 0.02$, unpaired, unequal variance t-test). The boxplot demonstrates no significant change in the mean respiratory rate, heart rate or MAP.

Discussion

SYMPTOMS WHILST BREATHING $5CO_2/16O_2$

Increased cerebral blood flow and headache and possible nausea related to increased intracranial pressure were anticipated in subjects whilst breathing $5CO_2/16O_2$, the symptoms of headache in eight subjects and one reported case of mild nausea were in accord with the findings of an earlier study.[1] Cerebral blood flow increases in the order of 50% when breathing 5% CO_2.[6] At 2.5% CO_2, there is no effect; at 3.5% a significant effect has been reported and at 7% the effect is far greater than at 5%.[6,7] After 5 min of breathing $5CO_2/16O_2$ in the present study, the $MCAv_{mean}$ increased by 49%, in agreement with these previous studies.

Moderate hypertension was recorded in all subjects whilst breathing $5CO_2/16O_2$, which has also been reported by others investigating the effect of an increased P_iCO_2.[8]

EFFECTS OF THE SWITCH TO 100% OXYGEN

Nausea and vomiting
No subjects vomited. One reported mild nausea which

developed after the switch to 100% O_2. Since one subject reported mild nausea whilst breathing $5CO_2/16O_2$, there is little or no evidence of a difference in the apparent effects of breathing $5CO_2/16O_2$ and the switch to 100% O_2 in terms of inducing nausea.

Some studies have not shown any evidence of incapacitation when switching from breathing a hypercapnic gas to air. Exposure to CO_2 at a concentration of 7% has been used as a tool to investigate panic and fear.[9] Neither sudden collapse nor vomiting was reported, although headache was, on return to air breathing. In another study, subjects were exposed inside a chamber to a PCO_2 of 1.3–5.6 kPa for 5 days, coming out of the chamber once each day to breathe air for 30 min. The study did not report any adverse effects on the subjects of switching between hypercapnia and air.[10] In the studies where adverse effects were reported, the P_iCO_2 was higher.[1,2] It appears that a CO_2-off effect that causes vomiting when switching to 100% O_2 following acute (~ 1 h) exposures to hypercapnia may only become apparent when switching from a P_iCO_2 above 5.0 kPa and that the severity may rapidly increase with only slight further increases in P_iCO_2.

Headache
In the current study, only three subjects reported headache that developed after the switch to 100% O_2, with the majority of subjects that experienced headache (8 of 11) having symptoms developing whilst breathing $5CO_2/16O_2$. Thus the exposure to $5CO_2/16O_2$ was more likely to induce headache than the switch to 100% O_2. The resolution of symptoms in three subjects following this switch suggests that it was at least as likely to reduce as to provoke headache.

Faintness
Faintness (mostly mild) was the most frequent symptom reported following the switch from hypercapnia to breathing 100% O_2. This occurred in seven subjects where faintness was not reported prior to the switch. There is some controversy over whether administration of 100% O_2 can maintain cerebral oxygenation in spite of hypoperfusion. It has been argued that hyperoxic hyperventilation and hypocapnia could decrease cerebral blood flow in excess of the effect of the increased O_2 content of breathing gas and paradoxically diminish O_2 delivery to the brain.[11] However, other authors have presented evidence that any likely effect of hypoperfusion (such as inducing fainting) caused by breathing 100% O_2 would be offset by the increased blood O_2 tension.[12] A clear independent cerebral vasoconstrictive effect of hyperoxia across a wide range of arterial PCO_2 has been demonstrated in at least one study.[13] Therefore, the decrease in cerebral blood flow observed in the present study when subjects switched to breathing 100% O_2 is likely to have been caused by cerebral vasoconstriction due to hyperoxia and the associated hypocapnia.

Several studies using TCD to measure $MCAv_{mean}$ have demonstrated a drop in values in association with pre-syncope and syncope. Passive head-up tilt in healthy subjects reduces $MCAv_{mean}$ and cerebral O_2 saturation and pre-syncopal symptoms appear when there is a reduction of about 50% in $MCAv_{mean}$.[14–16] Similar percentage drops in $MCAv_{mean}$ associated with symptoms of faintness have been observed in the present study.

Signs of imminent syncope have been associated with reductions in $MCAv_{mean}$ of 62% and 68% induced by sudden cold water immersion.[17] $MCAv_{mean}$ has also been measured in one study after acute hypercapnia reversal.[18] Subjects rebreathed from a bag containing 5% carbon dioxide in O_2 up to an $ETCO_2$ of 10% or to the limit of tolerance. When rebreathing ceased, there was a rapid decline in $MCAv_{mean}$ within 42 s, followed by a further rapid decline to below baseline, $MCAv_{mean}$ falling by 31% in total.[18]

Another study found reductions in $MCAv_{mean}$ of 44% and 69% respectively and concluded this decrease to be more important as a predictive factor of syncope than the MAP.[19] This is in agreement with the present study where a mean percentage decrease in $MCAV_{mean}$ of 51% was associated with pre-syncopal symptoms (sensation of mild or moderate faintness) while decrease in MAP was not associated with the group of subjects who experienced faintness developing following the switch.

LIMITATIONS OF THE STUDY

Use of a demand valve (DV) regulator for the mouthpiece
Subjects who were inexperienced in the use of a DV made comment on the difficulty of breathing. It is known that breathing systems have an effect on the depth, flow and pattern of breathing.[20,21] The use of a DV regulator could be avoided in future trials by supplying the subjects' breathing gases from pre-filled Douglas bags.

Duration of the test and effects of raised pressure
It should be noted that survivors waiting in the DISSUB may be exposed to raised ambient pressure and wait for up to seven days before rescue or escape. Investigation of prolonged (chronic) exposure to hypercapnic gas at raised pressure and the effects that acid-base balance, buffering and compensation may have on the response to a switch from hypercapnia to hypocapnia and/or hyperoxia was outside the scope of the current study. Effects of a switch to air or 100% O_2 following prolonged exposure to raised PCO_2 and/or hyperbaric exposure remain as possible topics for future investigation.

Possible additional effect of Valsalva
The Valsalva manoeuvre is carried out during the compression phase of escape in order to equalise pressure across the tympanic membrane, preventing otic barotrauma. During Valsalva, the $MCAv_{mean}$ can drop by about 35% when supine, and by around 50% when standing.[22] Thus, Valsalva may partially compromise cerebral perfusion and this may be compounded by any CO_2-off effect during escape. This issue is currently under investigation.

Conclusions

On undergoing a switch from breathing $5CO_2/16O_2$ to breathing 100% O_2, a significant difference was observed in percentage drop in $MCAv_{mean}$ between subjects who had symptoms of faintness that developed after this switch and those who did not, suggesting that feeling faint is linked to the drop in cerebral perfusion. The risk of incapacitation owing to fainting, sudden collapse or vomiting on switching to 100% O_2 following acute exposures to hypercapnia at a P_iCO_2 of up to 5.0 kPa is less than 8%. The relative mildness of symptoms observed does not indicate that a change to current procedures is necessary. However, the limitations of the current study suggest that the possibility of worse symptoms in some DISSUB scenarios cannot be ruled out. Evidence from other studies suggests that the severity of symptoms will increase if P_iCO_2 rises above 5.0 kPa.[1,2]

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Review article

Diving fatality investigations: recent changes

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Abstract

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Modifications to the investigation procedures in diving fatalities have been incorporated into the data acquisition by diving accident investigators. The most germane proposal for investigators assessing diving fatalities is to delay the drawing of conclusions until all relevant diving information is known. This includes: the accumulation and integration of the pathological data; the access to dive computer information; re-enactments of diving incidents; post-mortem CT scans and the interpretation of intravascular and tissue gas detected. These are all discussed, with reference to the established literature and recent publications.

Key words
Diving deaths, investigations, autopsy, radiological imaging, review article

Background

The investigation of diving fatalities has changed markedly over the last few decades, more than many conventional pathologists and diving physicians have appreciated. The 19th-century caisson physicians who investigated the dysbaric causes of death demonstrated the value of autopsy diagnoses. Later, the diving pathologists were not so fortunate – having to cope with a delayed recovery of an often damaged body, as well as the supervening complications of drowning, marine animal trauma, decomposition, post-mortem decompression artefact (PMDA) and resuscitation effects. Drowning was the autopsy diagnosis in about 80% of cases, with about 10% of cases attributed to gas embolism.[1] A review of the techniques used to investigate diving fatalities, including the interpretation of autopsy findings, was indicated. A fully annotated description of the changes and the reasons for them has recently been published in the forensic pathology literature.[2] A summary is presented here for diving physicians.

When the first edition of *Diving and Subaquatic Medicine* was printed in 1976, the autopsy, which had been the gold standard for post-mortem diagnosis, was being complemented by assessments from the clinical and diving data.[3,4] The publication focused on clinical interpretations, the dive profile and equipment testing. A new approach to diving accident scenarios has since been introduced, influencing fatality assessments.[5] A similar approach has been adopted in recent years in the annual reports of Australian diving-related fatalities.[6]

The causes of diving deaths have been extensively reported since the advent of scuba diving as a popular sport.[1,4–7] Reports acknowledge the importance of early liaison between diving experts, technicians, diving clinicians and pathologists if inappropriate conclusions are to be avoided. If the autopsy is performed in isolation from other sources of the diving data, interpretations may be problematic.

The investigation of diving deaths has followed this documented scenario:
1. Recovery, handling and observations associated with the body and all related equipment;
2. Witness and other informed statements;
3. The formal autopsy for immersion victims (techniques for aberrant gas identification and some procedural modifications for autopsies on divers are recommended to maximize the value of the postmortem examination[2,7–10]);
4. Technical assessments of the functioning of the diving equipment;
5. Gas analysis by reputable laboratories;
6. Coronial or other enquiries.

Approaches differ from country to country. In the Australian and New Zealand context (unlike the medical examiners system in the USA), it is usually the coroner who receives all of the documents and reports. While the prudent forensic pathologist would try to access these reports and would invite the diving physician to attend the autopsy, this is often the exception rather than the rule.

This paper deals with recent modifications to these diving fatality investigations and encompasses some autopsy techniques and laboratory tests, post-mortem CT scanning (PMCT), interpretation of extraneous gas in the deceased, dive computer data and the re-enactment of diving incidents. Frequent modifications of diving equipment and diving techniques (closed and semi-closed rebreathers, technical diving, varying dive profiles and newer decompression algorithms, etc.) dictate the constant up-dating of test procedures and are thus beyond the scope of this paper.

Autopsy techniques

These are well documented in current texts.[7,9] Some of the techniques previously proposed for diving autopsies have slipped into disuse because of difficulty in performing them or problems with their interpretation.[9–15] These are mentioned later, together with more valuable developments such as: the accumulation and integration of other contributing data, the use of dive computer information, re-enactments of diving incidents, post-mortem CT scans (PMCT) and the interpretation of intravascular and tissue gas detected by these.[2] Possibly the most important proposal for the autopsy investigation is to delay the drawing of conclusions until all possible diving information is known.

Fluid in the trachea and main bronchi is often observed in (but not specific to) drowning, as it is present in some cardiac deaths. Frothy airway fluid is present in both drowning and scuba divers' pulmonary oedema (SDPE).[15,16] The formal autopsy, where the chest cavity is manually opened, more clearly demonstrates the hyper-expanded lungs of drowning and the overweighted lungs of all three disorders.

Middle ear haemorrhage is often described as evidence of drowning.[2,14] The presence of middle ear/sinus mucosal congestion or haemorrhage is a frequent observation in clinical diving medicine, with associated symptoms and radiological validation. Symptoms usually limit the conscious diver from descending further, but when the victim is unconscious and descending and there is still circulatory activity, such barotrauma is to be expected.[7] Thus, this observation may simply imply descent whilst unconscious, not drowning as such. This explanation of middle ear and sinus barotraumas of descent is a far more feasible one than an inexplicable indication of drowning per se.

Diatom identification from various parts of the body, the airways and the incriminated water environment, despite having some potential value, has serious limitations and is rarely undertaken. Species recognition only implies water aspiration whilst alive, not death from drowning. Similarly, strontium, chloride, haemoglobin and other biochemical analyses have not had widespread acceptance and usage.[15]

One biochemical investigation that may differentiate other causes of death from sea water drowning, is the elevation of vitreous sodium and chloride levels.[17] It is not known whether this can differentiate non-fatal sea-water aspiration from drowning. False negative and false positive results need to be quantified.

While serological and immunoglobulin assessments to identify injuries from venomous marine animals have theoretical value, their use has not reached the international acceptability that was once predicted. In the appropriate setting, morbid anatomy and histology of skin and tissue wounds, and microscopic identification of nematocysts, are still of value.

Conventional diving autopsy techniques allow for the demonstration and examination of gas in atypical sites, such as the cardiac chambers, vessels, pleura, peritoneum and in other tissues. This can be achieved by opening these sites underwater or by perfusing vessels with inserted gas traps.[7–9] These techniques are not always easy or reliable, but they allow for the aspiration of the gas and analysis of its composition, which may indicate its source. PMCT is now recommended for demonstration of gas, prior to a diving autopsy, which itself can cause gas artefacts.

Conducting the autopsy at depth in a hyperbaric environment, which is possible under certain saturation diving conditions, avoids the disruptive influence of PMDA (or post-mortem 'off-gassing'). Such an autopsy location would be logistically and technologically problematic for most pathologists, even though some may argue it should be the 'gold standard' in identifying gas-related diving diseases.[7] In most diving fatalities the diver is either already at the surface or is brought to the surface with the hope of resuscitation. Transferring the body back to depth is not only impractical but fails to eliminate this artefact.

Post-mortem computerized tomography (PMCT)

Post-mortem X-rays were of value but these have been superseded by PMCT and other imaging techniques. These are becoming more commonplace as an adjunct, or even replacement, to the formal autopsy for detecting the origin, site and volume of abnormal gas spaces, as well as other pathology of the respiratory tract. It is more reliable in detecting gas spaces, more sensitive, less invasive, less time consuming and less offensive to various cultural and ethnic groups than a formal autopsy.[2,12,14] PMCT is performed as soon as possible, preferably within hours of the incident, and should precede any formal invasive autopsy procedure.

Evidence of pulmonary interstitial oedema is seen with the drowning syndromes (aspiration, near-drowning and drowning), cardiac disease and SDPE. SDPE has now been identified as a cause of diving deaths, but with an unknown incidence and similar lung pathology to drowning.[16] On PMCT a ground-glass appearance is observed in all these diagnoses. High attenuation particles, indicating sand or other sediment, may be present on PMCT in any of the drowning or aspiration syndromes, in the airways or para-nasal sinuses.[14] Frequently haemorrhage or effusion is detected in the middle ear and para-nasal or mastoid sinuses with PMCT.[2,14] This is an indication of possible barotrauma of descent – a consequence of descent by an unconscious diver.

Extraneous gas in the diving autopsy

Perhaps the most valuable but controversial aspect of the PMCT is the observation and interpretation of extraneous gas spaces in the diver's body. The techniques previously embraced to demonstrate abnormal gas in the diving autopsy

were introduced because gases are influential in the causes of death from diving and hyperbaric exposures, especially with gas embolism induced by pulmonary barotrauma (PBt) and decompression sickness (DCS).

The interpretation of gas detected radiologically and with newer scanning techniques has been marred in controversy. Some authors have embraced the newer technologies with enthusiasm whilst others have denigrated them as being valueless or misleading. This quandary has been addressed recently and is clarified if one understands the aetiology of the gas.[2] It requires a knowledge of infrequently accessed literature and an understanding of the processes that cause gas formation. Unfortunately, the presence of gas from processes that are not related to the cause of death have complicated its interpretation. These include PMDA, putrefaction, trauma and resuscitation effects.

It is often concluded that gas embolism caused a diver's death despite the diver being in a situation where this development was impossible. In 12 out of 13 diving fatalities autopsied at the NSW Institute of Forensic Medicine, intravascular gas was detected.[13] In some, the history and autopsy findings were inconsistent with pre-morbid gas embolism.

Extraneous gas may be detected post mortem in many anatomical sites: pleural, peritoneal, gastric, hepatic, muscular and intravascular. Interpretation relies not only on the site but also the volume and composition of the gas.[2] The potential causes are as follows.

Post-mortem decompression artefact
Boycott, Damant and Haldane in 1908 warned that "*the presence of bubbles in vivo must be inferred from their discovery post-mortem with considerable caution. The supersaturation of the body may be such that the separation of the gas bubbles may take place after death.*"[18]

The bubbling is mainly from inert gas, previously breathed by the diver and then dissolved in the blood and tissues.[12,13] PMDA can develop if the diver dies at depth or soon after ascent, if his body still retained supersaturated, dissolved gas. From deep and/or prolonged dives, it can produce extensive surgical emphysema, be present in all tissues and replace blood from both venous and arterial vessels (gas angiograms) and both sides of the heart. A PMCT scan should include the thighs, where gas is easily seen in the intra-muscular fascial layers. There are few other explanations for this observation.

Well-controlled animal experiments, across different species validate and quantify the concept of PMDA or off-gassing.[19–21] Animals that die at sea level and are then exposed to pressure do not subsequently develop PMDA. Nor do those that die immediately after exposure to pressure. Only those who were exposed to pressure whilst still alive and thus had a functioning circulation are so affected after surfacing. The depths and durations in these animal

experiments were designed to parallel typical profiles of human compressed-air divers. There was a latent period of about an hour until the PMDA became evident, and then a progression of this effect over the subsequent 1 to 8+ hours. Another pertinent observation was the presence of small local areas of gas pockets adjacent to trauma from resuscitation and invasive procedures (see later).

Thus the animal experiments confirmed clinical experiences but were in disagreement with a popular belief that deep diving, in excess of 40 metres' sea water (msw), may be necessary for PMDA to develop. Excessive depths were not reached in most of the animal experiments described above, nor in human divers and caisson workers described by others.[22,23] Logically one can understand that, while a deep or decompression dive is not required to initiate this phenomenon, the amount, likelihood, extent and speed of development of PMDA is a consequence of both depth and duration of the hyperbaric exposure.

Decompression sickness
Although uncommon, death can occur from DCS, with gas bubbles developing within any tissue, owing to excessive exposure to pressure (depth) and too rapid an ascent. Histopathological signs include haemorrhage, necrosis and tissue reaction or inflammation around the tissue gas bubbles, differentiating it from PMDA; but histopathology is frequently not sought.[24,25] It is more evident in lipid tissues, including myelin sheaths of peripheral nerves, induced by the nitrogen breathing of compressed air divers.

Over a century ago DCS deaths were far more frequent and the pathology not usually complicated by resuscitation – which may cause local gas artefacts and re-distribution of intra-vascular gas. Hoff, reporting on autopsies performed on divers and caisson workers, observed that the less acute DCS cases tended to have gas in the right ventricle of the heart, whereas, in those who died very soon after decompression or from explosive decompression, gas was present in both the arterial and venous systems, with widespread gross distribution throughout many tissues.[22] This latter group is more likely to have complicated their DCS findings with the effects of PMDA and/or barotrauma.

Sir Leonard Hill, in his literature review, noted that Von Schrotter observed gas in the vascular system in 11 of his 18 well-described autopsies on DCS victims, whilst Keays described it in eight of his 12 victims.[23] Paul Bert showed in animal experiments that gas from decompression collected in the venous system and the right heart and also that the composition of this gas reflected tissue gas pressures. Hill described further autopsies of DCS in caisson workers who were exposed to pressures equivalent to 19–34 msw for over 3 hours, then had a very slow ascent (so PBt was an unlikely complication). In these cases, with typical DCS symptomatology preceding the death, the gas was often observed in the venous system at autopsy. It collected in the right heart in seven of the 10 cases. No arterial and left-heart

gas involvement was reported in the cases that died after a delay of some hours (when PMDA was unlikely).

Of relevance, but not specifically addressed by Hill, was the excessive volume and widespread extent of the gas in four workers who died of DCS within an hour of surfacing. In these cases there was also gross gas in the arterial system, the left heart, the viscera, subcutaneous tissues, thighs and even the cerebral ventricles. DCS cases that succumb very soon after ascent are vulnerable to supervening PMDA, obscuring many of the DCS features, but not all. Necrotic areas around obstructed vessels, lesions in myelin sheaths and skin manifestations may still be detected.

Gas embolism following pulmonary barotrauma
This is well documented in diving medical texts and is initially observed as air (nitrogen/oxygen) or gas bubbles in the systemic arterial system. It arises from lung rupture allowing inhaled gas to pass into the pulmonary veins, then the left heart and the arterial system. Because gas emboli are redistributed partly by buoyancy in the larger vessels, they tend to travel to the brain in the ascending diver and with the erect posture after surfacing. Some of the emboli may obstruct the smaller arterioles, or involve multiple generations of arterioles. Many, however, pass through to the venous system and thus to the right heart and pulmonary arteriolar filter. This occurs with continuation of life and circulation, including effective resuscitation efforts. The arterial bubbles may persist and obstruct, especially in small arteries such as the circle of Willis, and indicate the pathological diagnosis and the cause of death. Gas within the venous system does not invalidate this. The association of lung damage, pneumothoraces, pneumoperitoneum and mediastinal emphysema are strongly supportive of a PBt origin for the embolism, as is a history of rapid ascent followed by unconsciousness.

Resuscitation-induced gas (artefact)
Resuscitation efforts may admit small volumes of gas into the venous system or cause local subcutaneous emphysema over the affected sites.[21,26,27] This rarely simulates the large volumes seen with PMDA or even PBt. Knowledge of the resuscitation scenario and the usually small amounts of gas, as well as its location, should suffice to exclude this as a contributor to death in non-traumatised patients, but it may show up in the CT scans.

Invasive and traumatic events, including head injury, intravenous cannulation, endotracheal intubation, external cardiac compression, etc., can induce local gas artefacts that may be misinterpreted. Subcutaneous gas or surgical emphysema at the site of thoracic compression can be produced from resuscitation. Shiotami et al has quantified this using PMCTs, with 71% of non-traumatic CPR fatalities containing some cardiovascular gas and 7.5% with cerebral gas, compared to zero in non-CPR cases.[27] The vast majority of bubbles were grade 1 (< 5 mm diameter) and were in the right heart or systemic venous system. Because gas can be introduced during the autopsy, they recommended that the PMCT be performed first.[27]

Lung damage from resuscitation efforts and/or paradoxical embolism from arterio-venous anastomoses, such as a patent foramen ovale, may explain the small and uncommon intra-arterial gas bubbles seen in some cases. Resuscitation may thus occasionally redistribute intravenous gas, such as from DCS, into the arterial system. The use of oxygen during resuscitation may reduce the volume and number of gas bubbles detected. This is the basis of our current first-aid resolution of the bubbles induced in diving accidents (DCS, PBt) and continued resuscitation, even after death, could have a similar effect if circulation is maintained.

Putrefaction (decomposition)
This is well described in general medical texts. It is evident after about 24 hours if the body is not refrigerated, although the onset varies from 3 to 72 hours, depending on the environmental conditions and the gas volumes being detected.[7,8,13,27] Some recommend that the diver's protective clothing (usually a wetsuit) should be removed early, before the body is refrigerated, to more rapidly reduce the body temperature and thus delay decomposition.

Putrefaction causes a foul-smelling gas initially evident in the gastro-intestinal tract, the portal veins and liver. Hydrogen, carbon dioxide, hydrogen sulphide and methane may be present. Because divers who die underwater are exposed to environmental cooling influences, it is likely that putrefaction may be more delayed. It is this gas that causes many submerged divers to float to the surface a few days after death.

Drowning
In addition to aspirating fluid, drowning often results in the swallowing of air and water into the gastrointestinal tract, explaining the tendency of near-drowning victims to vomit.[7,14,15] Ascent may increase the volume of gas, according to Boyle's Law, distending the stomach. The composition of the gas (usually nitrogen and oxygen) is in approximately the same proportions as in the air or other gases being breathed. In some cases, individuals may regurgitate and aspirate stomach contents, but typically this is not a factor in the drowning process, although it is not uncommon during resuscitation.

Dive computer records

Often, the description of a fatal dive is vague, sanitised and inaccurate, especially from the diving companions and dive operators who may have a conflict of interest in the results of the investigation. Also, the deceased is often alone prior to or at the time of the incident, denying the investigator of relevant diving data. Over the last few decades, the use of dive computers has become ubiquitous. These accurately depict the details of the fatal dive. Depths, dive durations, ascent rates, the number of ascents, decompression staging

and decompression stress, dive profiles (reverse or forward), water temperature, gas pressures and gas consumption are all informative and accessible by downloading with suitable computer software. If the dive computer is gas-integrated, i.e., the changing breathing gas pressure is being recorded and integrated into the database, then, knowing the scuba tank size, the diver's gas consumption can be extrapolated for the various sectors of the dive profile. In addition, similar information, together with gas sensor data to record oxygen pressures, is accessible from rebreather sets.

This information allows an assessment of the likelihood of PBt, DCS, SDPE, panic, fatigue, aspiration, gas toxicity, cold effects, etc. As previous diving data is also stored in the dive computer, this may imply a predisposition to diving accidents. It may indicate the diver's experience, rapid ascents, inadequate decompression, deep diving, 'low-on-air' situations, etc. As well as the dive computer data from the deceased diver, that of his companions and rescuers may also be downloaded by an impartial, competent technician.

Re-enactment of the diving incident

This term does not refer to the laboratory testing of diving equipment. That is conducted routinely after diving fatalities, to ensure compliance with the manufacturers' or other's specifications (usually performed by technicians in a diving equipment laboratory). The re-enactment is a more recent and totally different concept used to demonstrate the functioning of the equipment under the conditions prevailing during the time of the fatality.

Laboratory testing of equipment will determine whether it meets certain performance criteria and that it can be used as intended. It does not imply that it did not contribute to the death. Thus, a diving regulator may be functional, producing a water-tight seal and adequate inspiratory gas flows under normal conditions, with an experienced diver breathing gently in an upright position; however, under different conditions, it may malfunction. Examples of such conditions are excessive air consumption from anxiety or extreme exertion, negative buoyancy or swimming against strong currents, at great depths or with the diver in a different spatial orientation. That information can only be elicited with a re-enactment, which can also detect hazards such as other equipment problems, potential entrapments, hazardous environments, technique difficulties and personal demands.

The concept of re-enactment of the diving incident was introduced in 1967.[28] It followed the unexplained deaths of two divers using re-breathing equipment. It was designed for internal use by experts in the Royal Australian Navy in Australia, which was the primary organisation that investigated such accidents at that time. The concept, which has become more widespread and is now often employed by police divers, is designed for the situation where a death has occurred but where there is no convincing explanation for the fatality.[11,28] The purpose is to observe the presence

of adverse situations that had previously not been evident, and which may help to clarify the fatal incident and/or prevent future ones. It is carried out only after all the other dive investigations (including the autopsy) are complete and involves the following:[2]
- A detailed and accurate knowledge is required of the dive plan, dive profile, environmental conditions, buoyancy status, equipment used, and breathing gas pressures, composition and volume that existed at the time of the unexplained death.
- An accurate replication of the above is made by expert divers of a similar stature to the deceased, using the same or equivalent equipment and performing a similar dive in similar circumstances. Sample ports allowing for repeated gas sampling and analysis may be added when re-breathing equipment is involved.
- The divers need to have access to redundant emergency equipment to be used if necessary.
- Diving medical support, full resuscitation facilities and a rescue dive team must be available on site. It can be a hazardous exposure and attention must be paid to the ethical issues.
- Observer divers record the re-enactment using underwater video. Full documentation of the experiences and observations is made independently by each participant and this is compared to the video records.
- The fatal dive profile is replicated, but terminated prior to a catastrophic event.

If more than one potential scenario is present for the fatal dive, then more than one re-enactment may be required. In this event, any findings may not represent the actual situation existing at the time of the fatality, and should only be considered as possibilities to explore, not actualities.

A variety of observations may clarify the original assumptions and encompass demanding conditions, entrapment, water aspiration, disorientation, resistance to breathing, equipment inadequacy, gas toxicities (carbon dioxide, hyperoxia, hypoxia, narcosis), etc.

Conclusions

In most countries there are no analogous diving units to those that investigate aircraft accidents. Thus, the typical practice is for the investigation of the diving accident to be performed by police only moderately knowledgeable in the investigatory aspects of diving accidents, a local clinician who has little training in diving medicine, and a pathologist who is overworked and less then amenable to varying the standard conventional techniques. The result is often a mistaken diagnosis without an explanation of the causative sequence of events. There is thus a loss of valuable information and a failure to learn from the mistakes of the past.

A common error is for the diver fatality investigators to conclude a cause of death based on their own sphere of expertise before all the data are available. There needs to be a

close integration of data acquisition from all parties involved in the investigation. The pathologist needs to be aware of the specific requirements for a diving autopsy, as well as those required with aquatic/submersion fatalities.[2,7–9,29] The use of more sophisticated scanning techniques, their interpretation and the possible integration with the formal autopsy findings, conventional equipment testing and gas analysis, the dive computer data and, occasionally, re-enactment findings, requires a multi-disciplinary team approach.

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Case reports

Two fatal cases of immersion pulmonary oedema – using dive accident investigation to assist the forensic pathologist

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Abstract

(Smart DR, Sage M, Davis FM. Two fatal cases of immersion pulmonary oedema – using dive accident investigation to assist the forensic pathologist. *Diving and Hyperbaric Medicine*. 2014 June;44(2):97-100.)
Immersion pulmonary oedema (IPE) is being increasingly recognized in swimmers, snorkellers and scuba divers presenting with acute symptoms of respiratory distress following immersion, but fatal case reports are uncommon. We report two fatal cases of probable IPE in middle-aged women, one whilst snorkelling and the other associated with a scuba dive. In the snorkeller's case, an episode of exercise-related chest tightness and shortness of breath that occurred 10 months previously was investigated but this proved negative, and she was on no medications. However, at autopsy, moderate left ventricular hypertrophy was noted. The scuba diver had suffered several previous episodes of severe shortness of breath following dives, one being so severe it led to cyanosis and impaired consciousness. At inquest, the pathologist's diagnosis was given as drowning and IPE was not mentioned. Expert input from doctors trained in diving medicine should be compulsory in the investigation of diving deaths, and forensic pathologists should be properly trained in and have guidelines for the conduct of post-immersion and post-diving autopsies.

Key words

Introduction

Immersion pulmonary oedema (IPE) is being increasingly recognized in swimmers, snorkellers and scuba divers presenting with acute symptoms of respiratory distress following immersion, but fatal case reports are uncommon.[1–10] We report two fatal cases of probable IPE, one in a snorkeller and the other in a scuba diver, the latter case having similarities to one of the cases described by Edmonds et al.[1] These were referred to the coroner and, therefore, are in the public domain.

Case 1

A 56-year-old woman got into difficulties while snorkelling with dolphins and died the same day shortly after admission to hospital. She was described as a fit, healthy person on no medication. She was 1.68 m tall, weighing 71 kg (BMI 25.2 kg·m⁻²). About 10 months earlier she had had an episode of chest tightness and shortness of breath after strenuous exercise. She had a normal ECG at that time. Other investigations were negative and the episode resolved spontaneously over several days. More recently she had several shorter episodes of shortness of breath after strenuous exercise, but did not seek medical attention. She had some snorkelling experience, including three days on the Great Barrier Reef before travelling to New Zealand.

She was one of 14 snorkellers on a dolphin encounter trip, crewed by four staff. They were given a safety briefing, including a video, and provided with wetsuits. The victim completed a waiver form on which she did not list any medical problems. Water temperature was 15–16ºC and weather and sea conditions reasonable. They were instructed to go no further than 80 metres from the boat and occasionally, as they swam with the dolphin pod, the skipper moved the boat closer to the group. They had three swims, returning to the boat each time to move a short distance to where the dolphins were. It was during the third swim that the victim got into difficulty, the first two swims being apparently uneventful.

After about 10 minutes' interaction with the dolphins, a crewman noticed one snorkeller in distress with their fist raised (the prescribed signal for assistance). The vessel immediately drove towards her and the swimmer responded to calls with a thumbs-up signal. She then rolled over onto her back but had the snorkel in her mouth, and came up coughing and put her fist up again, along with the two people now with her, who assisted her back to the boat. She said "*I can't breathe*"; a small amount of froth was observed on her bottom lip. Her difficulty breathing worsened, and a friend helped her to remove the top of her wetsuit and sat her on the floor, raising her arms in the air and holding her head up. She was coughing up some foam from time to time. The swimmers were called back to the boat, an emergency call was made and the boat headed to shore. The maintenance crew and an ambulance were requested to meet them as the woman's condition had deteriorated. By the time the boat arrived at the jetty she was so weak she could no longer hold herself up.

The ambulance crew arrived as the vessel arrived, and they took oxygen, a defibrillator and their portable packs on board within a minute of the vessel mooring. The victim appeared to be in cardiac arrest, as she was deeply cyanosed and unresponsive. A rapid ABC assessment revealed an obstructed airway, with vomit and blood-stained sputum. The woman was turned on her side and her airway cleared with a finger sweep. The defibrillator showed "*sinus rhythm at about 80 beats per minute*", and a strong pulse was detected. An oropharyngeal airway was inserted after clearing her airway and she was placed on 100% oxygen. All the while, the victim continued to vomit and she had to be repeatedly rolled onto her side to clear her airway. She was placed on the stretcher, and started breathing for herself, at about eight breaths per minute, but "*quite distressed breaths*". She opened her eyes to command and became responsive and complained that she could not breathe.

On admission to the local hospital, the victim was breathing in a laboured fashion at about 40 breaths per minute, pulse rate 127 per min, blood pressure normal and temperature 35.4°C. Pulse oximetry was not possible because of peripheral shutdown. An intensive care retrieval team was requested from the regional tertiary-level hospital but this could not be despatched as they had been sent to a mining accident. Because of repeated airway compromise from vomiting and further deterioration, it was decided to intubate the victim. This proved difficult though eventually successful. Despite this, ventilation was difficult and she continued to deteriorate. Cardiac arrest occurred about 30 minutes later. Despite continued resuscitation for approximately 35 minutes, the victim died.

Post-mortem examination by an experienced forensic pathologist (MS) demonstrated congestive heart failure in the context of cardiomegaly and exertion. There was no coronary artery disease and no circumstances indicating drowning or near drowning. The heart weighed 443 g, 140–160 g greater than expected for her body height and weight. The larynx, trachea and major bronchi were internally unobstructed but contained frothy sputum. There was no macroscopic evidence of aspiration of vomitus. The left lung weighed 869 g and the right 954 g (expected normal weight for body size would be 250–300 g each). Trace amounts of alcohol but no other drugs were detected. Microscopy of the myocardium showed mild regular hypertrophy only with no old or new ischaemic injuries. Her cardiomegaly might in part be the result of a training effect and/or mild hypertension. The cause of death was given as "*immersion pulmonary* [o]*edema syndrome*".

Case 2

In 2002, a 51-year-old, fit female (height 171 cm, weight 71 kg, BMI 24.3 kg·m²) on no medications had done 20 dives over two years. She undertook a shore dive to a maximum depth of 11.9 metres in a sheltered bay (water temperature 17–18 °C). She experienced difficulty with breathing during the dive and over a 12-minute period made three ascents to the surface (accompanied by her buddy), from various depths, before finally returning to the surface. For the whole time, her regulator remained in her mouth. After the final ascent, she was noted to appear panicked and dyspnoeic, but mentally coherent and responding appropriately to her buddy. She was escorted to a mooring buoy to rest. Her respiratory condition deteriorated rapidly, with pink froth coming from her mouth, so she was towed to shore by her buddy, keeping her head above water. By arrival at the shore, she was unresponsive with no respirations or pulse. Basic life support (BLS) was commenced; ambulance officers assisted but the diver was unable to be resuscitated.

On examination all her diving gear was functioning normally except the diaphragm of the octopus regulator, which was not fitted correctly (this was not used at any stage). Calculations of air usage from her cylinder (11.4 L internal volume, 85 bar remaining) indicated she had breathed approximately 1,400 litres during her 12-minute dive.

Autopsy recorded pale pink, frothy fluid in the trachea, lower airways and bronchioles. There was no evidence of lung barotrauma, but hilar nodes showed histological features of sarcoidosis. There was minor mitral and tricuspid degeneration, normal coronary arteries, minor quantities of air in the brain, heart and liver consistent with post-mortem gas, and a degree of cerebral oedema. She had fractured ribs consistent with BLS. Post-mortem radiology was not performed. The cause of death (with a considerable degree of uncertainty) was given by the pathologist as drowning. However, witness reports stated that at no stage was the regulator out of her mouth underwater, and her head was always above water during rescue. These reports were confirmed by interview during the process of an independent investigation by one of the authors (DS).

Further information about her diving history was relevant, and obtained during the independent investigation. She had had three previous episodes of significant dyspnoea precipitated by scuba diving, one episode being so severe it led to cyanosis and impaired consciousness. She recovered fully from each of these episodes.

Discussion

For Case 1, it is not possible to completely rule out salt water aspiration; however, the post-mortem findings were consistent with congestive cardiac failure and cardiomegaly. In addition, despite continued emesis, there was no evidence of aspirated vomitus detected. The striking feature of this case is the rapidity of development of severe symptoms. This has recently been reported in other, non-fatal cases.[9]

On review of the records for Case 2, it was considered that this case was consistent with death due to scuba divers'

pulmonary oedema (SDPE – also known as immersion pulmonary oedema, IPE), and that the preceding non-fatal episodes were also consistent with SDPE. At the time of the coroner's hearing, a report was submitted to the coroner disputing the post-mortem conclusions. The dive buddy had indicated that at no stage was the regulator out of the diver's mouth, and ascents were controlled. Conditions on the day were calm, and the dive regulators were functioning normally, making salt water aspiration most unlikely. The 12-minute dive to 12 msw made pulmonary decompression illness unlikely.

SDPE appears to be under-reported and may not be recognised at postmortem because the pulmonary findings may be so similar to drowning.[1] IPE is an entity in which apparently fit adults develop sudden-onset pulmonary oedema while swimming, snorkelling or scuba diving, without any circumstantial evidence that this is part of a drowning or near-drowning event or necessarily related to any underlying cardiac disease.[1–10] There is an indication that older individuals with pre-existing hypertension or cardiovascular disease may be at higher risk than younger individuals. It is likely that Case 1 did have some degree of untreated hypertension.

The most common presentation is acute-onset coughing and shortness of breath while participating in the activity. The great majority of sufferers survive with symptoms resolving within 24 hours or less with or without supportive treatment.[3,4] Subsequent investigation of those affected often shows no or only minor underlying cardiac disease, which would be the alternative explanation for sudden onset pulmonary oedema. Initially there was a strong association seen between immersion in cold water and the onset of this condition, but it has been described in mild and even warm (swimming pool) conditions.[5,6] There has been a strong association with relatively extreme exertion (military diver training and triathlon sport) and prior overhydration, but cases have also been described in recreational settings.[1–10]

From a forensic pathology perspective, the appearance of the lungs cannot be reliably distinguished from classic drowning by autopsy findings alone, reinforcing the universal need to correlate the circumstances of death with the autopsy findings. In both these cases, the circumstances of death suggest drowning was unlikely, perhaps with slight uncertainty for Case 1 because of the brief episode of snorkel immersion. We believe that expert input from doctors trained in diving medicine should be compulsory during investigation of diving deaths, and forensic pathologists should be properly trained in and have guidelines for the conduct of post-immersion and post-diving autopsies.[11] Through a sequential analysis of events leading to diving fatalities a more comprehensive picture is constructed, which identifies causative triggers, disabling incidents and injuries and may be used for future prevention of accidents.[12] It may well be that IPE has been the mechanism of death

in immersion in other cases attributed to drowning without such eye-witness observation to provide the distinction. Immersion deaths of victims dying alone, or sufficiently removed from their companions for them to give any reliable account of the precise circumstances, could well represent overlooked IPE.

Similar mechanisms may also explain sudden-onset pulmonary oedema in 'stress situations' not involving water immersion in the absence of structural heart disease, and is possibly related to the otherwise well-documented, stress-induced Takotsubo cardiomyopathy.[13] Pre-existing high vascular resistance and exaggerated vascular response to cold stress in divers who developed pulmonary oedema has been reported, although this was not a consistent finding in subsequent reports.[14–16] In one series, over a quarter of individuals sustaining IPE may have had reversible myocardial dysfunction.[17] Current research at Duke University Medical Centre is seeking volunteers who have suffered IPE to study whether there may be a genetic disposition in a small proportion of the population who carry a number of gene markers that may be associated with IPE.

Another issue is controversial. Should scuba divers be advised they are safe to return to diving after a non-fatal episode of IPE? When evaluating diver risk, the worst possible consequence is a fatality. Certainly all should be evaluated for manifest or occult cardiovascular disease.[18] IPE is not a benign condition. It also appears to be idiosyncratic, not occurring with every dive or immersion. Case 2 is remarkably similar to other reported cases, with significant periods of time between IPE episodes.[1,8] Unfortunately Edmond's case developed fatal IPE even after extensive land-based investigations had detected no abnormality.[1] Until a full epidemiological study is performed, or markers of IPE risk are identified, we recommend extreme caution when evaluating individuals seeking to return to diving after an episode of IPE.

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Letters to the Editor

Treatment of decompression illness with heliox: the best of both worlds?

There are many ways to treat decompression illness (DCI) at increased pressure. In the last 20 years much has been published on the use of oxygen and helium/oxygen mixtures at different depths. The type of information ranges from impressive work on animals,[1] case reports, small series of case reports and, finally, mathematical work on the behavior of different inert gases in the hyperbaric physiological environment.

During a therapeutic compression, the use of a different inert gas from that which was breathed during the dive may facilitate bubble resolution. If the physical properties of the treatment gas are chosen in such a way that its rate of transport through the tissue is lower than that of the breathing gas, bubble shrinkage will be accelerated. Another factor to be considered is whether the exchange of gases in tissue and blood is limited by perfusion or diffusion. Finally, the solubility of the treatment gas in blood and fatty tissues, in relation to the breathing gas which resulted in bubble formation and DCI, should be taken into account.

The above-mentioned factors play a complex role in the choice of the treatment tables in relation to the different types of dives accomplished. Compressed air is used for professional dives to 50 metres' depth and, for shallower depths, nitrox can be used. Technical diving using trimix has become more widespread during the last decade, whereas saturation divers breathe different gases at different depths to prevent the effects of counter diffusion and the high-pressure nervous syndrome.

Almost all cases of DCI in humans diving to 50 metres with air or nitrox can be adequately treated at 283 kPa (equivalent to 18 msw), where 100% oxygen is both safe and effective. Serious neurological and vestibular DCI, with only partial improvement during initial compression at 283 kPa on oxygen, may benefit from treatment at 404 kPa with 50/50 heliox (Comex 30 treatment table, Cx30). Cases have been successfully treated with the heliox Cx30 or modifications thereof, or the US Navy 6A recompression table using 80/20 and 60/40 heliox instead of air.

Theoretically, the use of helium-oxygen during therapeutic recompression might be advantageous. However, experience with the use of deeper treatment tables with either helium or nitrogen as inert gas in a treatment mixture with oxygen, has not consistently shown an advantage of helium. Also, there is growing evidence that helium is biochemically not inert and has biological effects on organs and tissues.[2] In experimental research, helium reduces ischaemia-reperfusion damage in the brain.[3] Because this is one of the mechanisms in DCI, heliox mixtures could be advantageous and enhance the treatment of DCI. Four studies have described the possible role of helium in neuroprotection but only speculate on an underlying mechanism: antagonists of the NMDA receptor or removal of nitrogen from mitochondrial compartments have been suggested.[4–7]

Therefore, a systemic research programme is still needed, including animal models and large human trials, to define the benefit derived from the use of different treatment gases and depth profiles during recompression therapy.[8]

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Key words
Decompression illness, treatment, helium, neuroprotection, letters (to the Editor)

Vinegar and *Chironex fleckeri* stings

We read the in-vitro experiment on the effects of vinegar on *Chironex fleckeri* tentacles by Welfare et al in the March edition of *Diving and Hyperbaric Medicine* with great interest.[1] Their experiment demonstrates that the application of 4% acetic acid to segments of *Chironex* tentacles may promote "*further discharge of venom from already discharged nematocysts*" and that this may cause harm in the clinical setting. Although well designed, we have a number concerns about the paper and the conclusions presented following its release.

Firstly, the assertion that the use of vinegar is associated with an increase in pain appears to be anecdotal and we are aware of only one paper that reports this.[2] We believe that clarification of this adverse effect alone is worthy of an independent study.

Secondly, the method used by the authors to stimulate the nematocysts is worthy of discussion, and is the crux of the relevance of this paper to the clinical setting. This technique, originally described by Barnes, is used to extract venom for the production of antivenom, and involves the application of an electric current to sections of *Chironex* tentacle on a section of human amniotic membrane.[3] Although an extremely useful technique to collect venom for research purposes, it is unclear whether it correlates to what happens in vivo when someone is stung by a *Chironex* jellyfish.

The use of vinegar to inactivate undischarged nematocysts has been recommended by the Australian Resuscitation Council (ARC) following the work by Hartwick et al in 1980.[4] This is based on the premise that there are two populations of nematocysts following a jellyfish sting – discharged and undischarged. Vinegar unequivocally inactivates the latter, and has the potential to prevent the firing of a large proportion of nematocysts on the skin. Without inactivation, these nematocysts, estimated to be as high as 80%, could potentially fire and worsen envenomation.

The results seen by Welfare require a premise that there is a population of partially discharged nematocysts with residual venom available. In their in-vitro model, it appears that vinegar might cause complete discharge in these partially discharged nematocysts. Is this population of partially discharged nematocysts present in the clinical setting, and in what magnitude? Without clinical studies to further clarify this, we would not recommend the removal of vinegar from the management of jellyfish stings in tropical Australia.

Thirdly, it is unfortunate that other readily available liquids were not compared to vinegar using this model. The authors mention the use of hot water in the treatment of stings by *Physalia* species, and it would be interesting to see what effect this would have in the model used. Without a comparison group, it is unclear as to whether the demonstrated effect of vinegar would also occur with the application of other liquids. It is possible that hot water may also complete discharge of nematocysts, but without inactivating undischarged ones, in which case it would be worse than vinegar.

The paper by Welfare et al has certainly raised an interesting question, but further research, ideally clinically-based, needs to occur before vinegar should be removed from the management of jellyfish stings in tropical Australia.

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Reply:

We thank Drs Gibbs, Corkeron and Blake for their interest in our study.[1] We are delighted to respond to their comments. Firstly, the anecdote that vinegar increases pain and an unpublished case series (into analgesic requirements in

Irukandji envenomation) performed at Cairns Hospital concerned us that vinegar may not be the panacea it is thought to be and prompted the study. Interestingly this increased opiate requirement was for systemic pain and not for any pain at the sting site. Our initial suspicion was that the increased opiate requirement was driven by the lack of application of vinegar; however, our findings suggested otherwise; the use of vinegar on an envenomation increased opiate requirements and increased the length of stay at a medical facility.

Secondly, the relevance of our stimulated nematocysts model to clinical envenoming has been discussed previously with our in vivo pressure immobilisation bandages (PIB) experiment in 2000.[2,3] We would expect that by now there would be evidence to support this concern but, to date, we are unaware of any evidence to this effect. Whether this technique is an adequate simulation does not refute the evidence that discharged nematocysts still have residual venom, and that, when vinegar is applied, an average of 60% more venom is released.[1–3] It has been demonstrated previously that nematocysts have residual venom and that the volume of venom retained within may be equivalent to that which has already been discharged.[2,3] It has also been demonstrated that this venom can be expressed by pressure and we now add to this knowledge that this residual venom can also be expressed by application of vinegar. Similar to our conclusions with PIB, this has the potential to worsen an envenomation.

Thirdly, that vinegar effectively disables undischarged nematocysts is not disputed; however, we are unaware of any data that would support the quoted figure that 80% of nematocysts in contact with skin are undischarged. Consequently, the claim that vinegar protects the victim from these discharging, causing further envenomation, is speculative. It is, however, plausible that some nematocysts may not be in contact with skin, considering that *Chironex fleckeri* tentacles are ribbon-shaped and may adhere to the victim in a convoluted and contracted state. Without further manipulation these nematocysts are clinically irrelevant to further envenomation. We are unaware of any data that answers the question raised by the authors in relation to the population of discharged versus undischarged nematocysts in direct skin contact, where the relevance of vinegar does actually have a bearing.

Finally, vinegar is the one recognised first-aid treatment for tropical marine jellyfish stings. As such, this experiment was performed specifically to examine the effect of vinegar on residual venom held in discharged nematocysts. Further to this, the testing of other common liquids as suggested, which have already been shown to be ineffective in de-activating nematocysts is irrelevant to the experiment and the envenomed victim.[4]

We disagree with Gibbs, Corkeron and Blake. Without evidence as to its effectiveness or safety, vinegar was promoted and recommended to specifically reduce further envenomation. Instead we have now demonstrated that it has potential to worsen envenomation. This is not just an interesting finding, it is a genuine concern.

Like PIB, where the potential to cause harm has been demonstrated in the absence of effectiveness or safety, it would be prudent to acknowledge the risk in the use of vinegar and to judiciously express this risk in a measured recommendation for its continued use, rather than continuing to recommend its unfettered use. That modified recommendation should continue until the safety and efficacy of vinegar has been established fully by appropriate research. We recognise that vinegar has been introduced and accepted as a core first-aid treatment in marine stings at a time when the requirements for demonstrated safety or efficacy were not as stringent. We now provide a need to re-examine this.

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Critical appraisal

Weak evidence for benefit of hyperbaric oxygen in patients more than six months after stroke

Clinical bottom line:
There is some limited evidence that HBO improves disability more than six months after stroke.

Citation:
Efrati S, Fishlev G, Bechor Y, Volkov O, Bergan J, Kliakhandler K, et al. Hyperbaric oxygen induces late neuroplasticity in post stroke patients – randomized prospective trial. *PLoS ONE*. 8(1): e53716.doi:10.1371/journal.pone.0053716

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Three-part clinical question:
For patients who have suffered a stroke, does the addition of hyperbaric oxygen treatment (HBOT) to standard care improve disability outcome?

Search terms:
Stroke, neuroplasticity

The study:
Single-blinded, randomised controlled trial without intention to treat.

The study patients:
Adult patients at least six months after stroke and with at least one motor deficit.

Control group:
(*n* = 37; 29 analysed)
Usual physiotherapy for 8 weeks, then cross-over to HBOT.

HBOT group:
(*n* = 37; 30 analysed)
Standard care plus 40 sessions of HBOT at 203 kPa for 90 minutes daily for 8 weeks.

The evidence:
See Table 1.

Comments:
i   The unblinded study design may have contributed to positive bias from patient perception.
ii   Assessment of the clinical benefit of a mean improvement of 2.8 in NIHSS score requires specialist interpretation.
iii The outcomes are very short-term and may not persist.
iv   There are patient data missing from each graph in Figure 2 of the published paper.
v   The EQ-VAS mean improvement of 4.9 to 6.5 is extremely small on a scale of 1–100. Probably incorrectly reported and is actually a scale from 1 to 10.

Appraised by: Alan Bourke
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Key words
Central nervous system, neuroprotection, hyperbaric oxygen therapy, outcome, research, critical appraisal

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Table 1
Outcomes for patients post stroke (means and SD shown); HBOT – hyperbaric oxygen treatment

Outcome measure assessed	Control group		HBOT group		Difference	95% CI
National Institutes of Health Stroke Scale (0 = normal, 40 = dead) at 8 weeks	8.3	(4.3)	5.5	(3.6)	2.8	0.77 to 4.87
Activity of daily living	17.6	(9.5)	12.8	(7.3)	4.7	0.27 to 9.09
Quality of life (EQ-5D)	8.6	(1.7)	7.7	(1.3)	1.0	0.18 to 1.76
Quality of life (EQ-VAS)	5.3	(2.3)	6.5	(1.5)	1.3	0.2 to 2.2

Continuing professional development

Diving hazards

Ian Millar

Accreditation statement

INTENDED AUDIENCE

The intended audience consists of all physicians subscribing to *Diving and Hyperbaric Medicine* (DHM), including anaesthetists and other specialists who are members of the Australia and New Zealand College of Anaesthetists (ANZCA) Diving and Hyperbaric Medicine Special Interest Group (DHM SIG). However, all subscribers to DHM may apply to their respective CPD programme coordinator or specialty college for approval of participation.

This activity, published in association with DHM, is accredited by the ANZCA Continuing Professional Development Programme for members of the ANZCA DHM SIG under Learning Projects: Category 2 / Level 2: 2 credits per hour.

OBJECTIVES

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

FACULTY DISCLOSURE

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

DO I HAVE TO PAY?

All activities are free to subscribers.

Key words
Underwater diving, nitrogen narcosis, pulmonary function, cold, hypercapnia, aerobic capacity, deaths, MOPS (maintenance of professional standards)

Recommended background reading

Practitioners are referred to the following background references and reading.

This CME activity is primarily based upon the material published in this edition of *Diving and Hyperbaric Medicine* and more detail on various matters can be found in the references quoted.

International guidelines on the performance and interpretation of spirometry can be accessed from the website of the American Thoracic Society <http://www.thoracic.org/ statements/ (pulmonary function testing)>, whilst a more succinct handbook can be downloaded from the website of the National Asthma Council Australia: <http://www.nationalasthma.org.au>.

Wikipedia provides a useful overview of the MET under the term "Metabolic Equivalent"

How to answer the questions

Please answer all responses (A to E) as True or False. Answers should be posted by e-mail to the nominated CPD coordinator.
EUBS members should send their answers to Lesley Blogg:
E-mail: <lesley.blogg@eubs.org>.
ANZCA DHM SIG and other SPUMS members should send their answers to Neil Banham:
E-mail: <Neil.Banham@health.wa.gov.au>.
If you would like to discuss any aspects with the author, contact him at:
E-mail: <i.lmillar@alfred.org.au>.

On submission of your answers, you will receive a set of correct answers with a brief explanation of why each response is correct or incorrect. A correct response rate of 80% or more is required to successfully undertake the activity. Each task will expire within 24 months of its publication to ensure that additional, more recent data have not superseded the activity.

Question 1. Nitrogen narcosis

A. Nitrogen narcosis can impair cognitive functions such as concentration, visual scanning, estimation of time, choice reaction time, and conceptual reasoning. Mechanical tasks requiring dexterity are particularly badly affected.
B. Nitrogen narcosis only becomes measurable at depths deeper than 30 msw.
C. The Tupperware Neuropsychological Test (TNT) of tactile performance involves the selection and accurate fitting of the correct lids onto proprietary plastic containers of differing sizes.
D. The finding that all participants had difficulty performing the TNT in low visibility at 608kPa suggests that the impairment arises from nitrogen narcosis-related slowing of the cognitive processes required.
E. Pre-dive and shallow-dive "over-learning" of critical tasks is a sensible precaution prior to deeper dives breathing air.

Question 2. Cold-water diving and bronchoconstriction

A. Inhalation of very cold air commonly triggers bronchoconstriction, especially with exercise, in both asthmatic and non-asthmatic subjects.
B. Whereas scuba regulators deliver cold, dry air, use of a full-face-mask reliably provides the diver with warm, humidified air.
C. Contrary to expectations, Uhlig et al found that using a full-face-mask during dives in very cold water did not significantly moderate lung function changes.
D. Other factors that may induce lung function test changes include fluid redistribution due to immersion, cold-induced vasoconstriction or other mechanisms related to cold stress.
E. International guidelines for performing definitive spirometry require the subject to perform at least three "blows" with at least two tests having acceptable performance characteristics.

Question 3. Exercise intensity of scuba diving

A. The energy expenditure measure "MET" or "metabolic equivalent" is a reliable and repeatable measure as it quantifies the energy expenditure of any given activity as multiples of the basal metabolic rate.
B. Whilst the MET is quite widely used in epidemiology and physical fitness guidelines, $\dot{V}O_{2\ max}$ in mL·min^{-1}·kg^{-1} is preferred for the accurate testing of individuals, as it is based upon oxygen consumption per unit of time, rather than ergometer workload which is influenced by efficiency of technique and pulse rates, which provide only a very indirect estimate of cardiac output.
C. A diver's air consumption can provide a reliable and accurate measure of $\dot{V}O_2$, as the oxygen concentration is known and the pre- and post-dive cylinder pressure provides an accurate measure of air usage.
D. Scuba diving is an activity that has been demonstrated to demand greater oxygen uptake during maximal effort than that required at maximum effort conducted out of the water.
E. Despite the limitations of Buzzacott's study, it does provide overall support for 7 MET being sufficient for uncomplicated recreational scuba diving in a range of conditions.

Question 4. Carbon dioxide physiology and toxicity

A. At the surface, carbon dioxide concentrations must exceed 2.5% before cerebral blood flow starts to increase significantly.
B. For dives to 50 msw, breathing gas levels of carbon dioxide below 4000 ppm are, therefore, without risk.
C. Loveman et al's study created a physiological replica of the breathing gas environment that would be encountered by submariners at the time point of escape from a sunken submarine.
D. Oxygen breathing reliably reduces headache by causing cerebral vasoconstriction and an improvement in cerebral oxygenation.
E. With a sudden switch from hypercarbic breathing to hyperoxia, the sensations of faintness and nausea that can be experienced are probably pre-syncopal and associated with reduced cerebral blood flow.

Question 5. Risk of injury and death

A. Although the death and major injury rates for diving are much lower than those associated with many other sports, survey data suggest adverse events may be occurring during more than 10% of recreational dives, whilst symptoms possibly representing mild decompression illness may be 20–30 times more common than diagnosed and treated decompression illness.
B. Early post-mortem CT (PMCT) scanning is now recommended as an initial step in investigating diver deaths as it can reliably detect many relevant pathologies such as intravascular or tissue gas, haemorrhage into the sinuses or middle ear and lung changes associated with barotrauma, drowning or pulmonary oedema.
C. A finding of intravascular gas on PMCT confirms that arterial gas embolism was the most likely cause of death.
D. If CPR has been performed, any finding of intravascular gas cannot be attributed to diving as large quantities of air have often been forced into the cerebral vasculature and heart.
E. Post-mortem decompression artefact is a term used to describe tissue gas that has evolved in the body of a diver who died at depth or shortly after ascent, following dives that have dissolved inert gas in the diver. As for decompression illness risk, the quantity of gas is correlated with the depth and duration of the pre-mortem dive.

Book review

NOAA Diving Manual – Diving for Science and Technology, 5th Edition

Dinsmore DA, Bozanic JE, editors
Soft cover manual, or eBook format
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Available from: <www.bestpub.com>
Price: USD129.99

This is the eagerly-awaited fifth edition of the US National Oceanic and Atmospheric Administration (NOAA) Diving Manual. The fourth edition was published in 2001 and, although a revision to that edition was made in 2010, there is no doubt that significant advances have been made to how diving in support of science and technology is carried out 12 years on. This book is rightly considered to be one of the most useful diving reference manuals and weighing in at over 750 pages it contains a wealth of information. In undertaking this review, I was most interested to highlight where the main revisions have occurred.

The first thing you notice about the fifth edition is that it is now published in soft rather than hard cover (it is also published in eBook format). The structure of the manual is very similar to the fourth edition; with 20 chapters and 10 appendices, compared with 21 chapters and 10 appendices in the earlier version. Two chapters disappear: Diver and Support Personnel Training, and Underwater Support Systems; a third chapter, Diving from Seafloor Habitats, is now incorporated into the chapter on Saturation Diving. Three new Appendices come after: NOAA Air Diving Table, Rebreather Checklist, and Recognition of Diving Maladies. The deleted appendices are: Dive Planning, No-decompression Tables, and Saturation Treatment Tables.

In evaluating this new edition, I invariably compared the old chapters with the new. Some chapters, such as Diving Physics and Diving Physiology, show negligible alteration, as would be expected for subjects where there has been little fundamental change over the past 12 years. Similarly, the changes in the chapters on Polluted Water Diving, Diving in Special Conditions, Hyperbaric Chamber, Dive Planning, and Surface-Supplied Diving were relatively few. Other chapters, such as those on rebreathers (although it must be noted that at the time of publication, NOAA only recommends the use of a single model of closed-circuit rebreather), nitrox and mixed gases had received significant updates. The chapter on Diver Location and Recovery was new and up to date with the description of EPIRB and PLB technologies.

There is a huge amount of information contained within the manual, most of which is of high quality with many examples of significant update. I do not want to denigrate the overall value of this new edition but I do feel that this revision was an opportunity lost. Unfortunately, too many of the Chapters appear to have been revised in a less than standard way.

Including an initial chapter on the History of Diving seems to be traditional for this manual but you do wonder why. Yes, it is interesting from the contextual point of view but it is tackled in a cursory fashion to the point that it is probably worthless to the modern science-based reader. And it is not helped that there are some incredible mistakes in the chapter, the worst being Figure 1.18, which uses a photo of the modern day *Aqualung* Mistral regulator as an example of the Cousteau/Gagnon patent. An accurate history of scientific diving per se would probably have been a more inspiring introduction.

The chapter on Diving Equipment was also frustrating in that there is some update from the previous edition but this is limited and varied. Examples are: a superficial description of the various dry glove designs, whereas there have been significant advances in this area plus new methods of attachment (e.g., the *DUI* zip-seal approach); the lack of explanation of the different DIN fittings now in regular use (232 and 300 bar); and no reference to the new nitrox connection standards (M25/M26). Yes, it is agreed that some of these changes are probably more pertinent to the European sector but this manual is marketed to a world-wide audience and so should be catering for these variations. In the same manner, perhaps it is time to widen the variations of diving in different geographical regions to outside of continental USA. However, the inclusion of metric units as alternatives in many sections is a welcome addition to this edition.

In the section on depth gauges, there is no discussion about the problems of estimating depth caused by water density/salinity, and does anyone in modern scientific diving still use depth gauges based on capillary action or the Bourdon design? In the segment on dive computers there were some impressive statements, such as: "*multiple deep dives require special consideration*" but these are followed up with zero guidance on what form the special consideration should take or what the potential outcomes of that consideration would be. Also the section on videography and still photography is written in such general terms that you are left thinking that the author had no topical or detailed knowledge of the subject matter at all.

The chapter on Scientific Procedures requires significant updating and preferably from authors who are currently working in these areas. Much of the supporting literature is decidedly out of date and many modern mainstream approaches to ecological investigation, for example the use of multi-dimensional scaling or cluster analysis, are overlooked. I could also not find any reference to or

explanation of some of the fundamental experimental principles such as independent sampling. This section also contained some wonderfully divergent approaches to outlining practical approaches to underwater science. There is considerable text dedicated to the methodologies of writing on slates and waterproof paper underwater but relatively little on three-dimensional imaging or the use of lasers or PCs or digital callipers, etc., underwater.

Unfortunate though both incidents were, it is strange that the fatalities of a snorkeller in the Antarctic as a result of a leopard seal attack, and those of two US Coastguard divers in the Arctic were not mentioned in the sections on Hazardous Aquatic Animals and Diving under Ice. Both incidents involved scientists and have been published widely and they act to serve as illustrative examples to be learned from. Related to this, I was surprised that there was no mention of the threat of polar bear attacks in the Arctic. In the Dive Planning chapter, there was inference toward risk assessment but no detailed explanation of its near-universal application in scientific diving. Although mentioned in the supporting text, there is also no reference to the management of hypovolaemia in the summary section on the treatment of decompression sickness.

The References Appendix is particularly disappointing as it appears as if there has been little effort taken to optimise this section. A few new references have been added but rarely at the expense of some of the older ones. Although some historical references are interesting and may still be relevant, it is difficult to see what use it is to readers who are working in modern-day underwater science and technology to be referred to texts well over 20 years old. Referencing guides to underwater photography that are over 40 years old, for example Church (1971), will be of little

use to researchers based in the digital age. Similarly, one wonders why the editors are retaining references to the use of horse-collar BCs (Snyderman 1980 and 1981) or hard contact lenses (Simon and Bradley 1978, 1980, 1981). And there are many other examples where the references to the use of various diving equipment and techniques are very outdated (1970s and 1980s). Scientific techniques such as tagging, census methods, use of anaesthetics underwater, underwater archaeology etc., also suffer from the same indolent approach to the references, with most examples from between the 1950s and 1970s. Hopefully in a revision to this edition or the next edition, more time will be taken to make a fundamental modification of this section.

Irrespective of my negative comments, the manual remains an impressive piece of work and covers all areas of diving that could be employed in support of science and technology. It is difficult to see how any scientific diving unit cannot continue to have the most current version of this book on their shelves. The inclusion of new and updated chapters does make this new edition worthy of purchase. However, overall this was a very disappointing revision based on a seemingly apathetic editorial policy. With a new edition coming out no quicker than every 10–12 years, I do hope the editors of the next edition start now in planning their revision and take the time and invest the effort in making it truly up to date and pertinent for use at the time of publication and for the decade subsequent to that event.

Martin Sayer
UK National Facility for Scientific Diving

Key words
Scientific diving, operations – diving, textbook, book reviews

Reprinted with kind permission from Sayer MDJ. Book review: *Underwater Technology.* 2013;31:217-8. This review has been slightly re-edited from the original.

Advertising in *Diving and Hyperbaric Medicine*

Commercial advertising is welcomed within the pages of *Diving and Hyperbaric Medicine*. Companies and organisations within the diving, hyperbaric medicine and wound-care communities who might wish to advertise their equipment and services are welcome.

The advertising policy of the parent societies – EUBS and SPUMS – appears on the journal website: <www.dhmjournal.com>

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

Notices and news

EUBS news is on the website

EUBS notices and news, such as the minutes of the most recent meeting of the Executive Committee and the 2013 General Assembly held during the Tricontinental meeting on Réunion Island, can be found, along with all other EUBS information, on the society website: <www.eubs.org>.

In order to increase space for original research and educational articles and to minimise the rising costs of publishing *Diving and Hyperbaric Medicine*, the decision has been made to reduce the amount of society business appearing within these pages.

European Editor for *Diving and Hyperbaric Medicine*

EUBS announces with sadness that the European Editor, Peter Müller, will resign at the EUBS 2014 ASM next September. A search has been established to appoint a new European Editor as soon as possible, so that (s)he has time to overlap with Peter.

Candidates for the position should send a brief application and their full curriculum vitae, in electronic format, to the Honorary Secretary, Joerg Schmutz at:
E-mail: <joerg.schmutz@eubs.org>.

EUBS Member-at-Large Elections 2014

Each year, the EUBS needs to elect a Member-at-Large, to serve in the Executive Committee (ExCom) for a period of three years (2014-2017).

As of 01 June, the CVs of candidates will be available on the EUBS website. An electronic Ballot Sheet will be circulated to all regular members on 15 June. In case you have not received a ballot by then, please check your 'spam' e-mail folder or contact the EUBS Webmaster at <webmaster@eubs.org>.

The

website is at
<www.eubs.org>

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

41st EUBS Annual Scientific Meeting

Dates: 19-22 August, 2015
Venue: Amsterdam

For information see:
<www.eubs2015.org>

The 5th Arthur Bornstein Workshop Diving in Offshore Wind Farms

Date: 23 September 2014
Venue: Wiesbaden
Chairmen: W Sterk and W Welslau
A satellite meeting of the 40th EUBS ASM 2014

Topics:
- The current situation in offshore wind energy in Northern Europe and outlook for tomorrow (company manager or government official)
- A view from the bottom (diver)
- A view from topside (diving supervisor)
- Presentation of current regulations in Northern Europe
- Myths and facts about surface decompression (to be named)
- Mix-gas options (to be named)
- Saturation options (to be named)
- System solutions (dive company)
- The need for a joint action to improve offshore shallow divers' safety (discussion)

For information please contact: <drfaesecke@aol.com>

Third Announcement
Dates: 24–27 September 2014
Venue: Wiesbaden, Germany

The 40th EUBS Annual Scientific Meeting will be held in conjunction with the 2014 congress of the German Society for Diving and Hyperbaric Medicine (GTÜeM). The patrons of this event are GTÜM and the Compression Chamber Centre Rhein-Main-Taunus (HBO-RMT) in Wiesbaden/Germany.

Organising Committee
Peter Müller (Secretary General), Peter Germonpré (EUBS), Karin Hasmiller (EUBS/GTÜM),
Michael Kemmerer (EUBS/VDD/Wiesbaden), Dirk Michaelis (EUBS/GTÜM/Wiesbaden), Peter Freitag (HBO-RMT)

Scientific Committee
Costantino Balestra (EUBS), Lesley Blogg (EUBS), Bjorn Jüttner (EUBS/GTÜM), Claus-Martin Muth (EUBS/GTÜM),
Lars Perlik (Wiesbaden), Tim Piepho (GTÜM), Christian Weber (Frankfurt), Christian Werner (Mainz)

Main topics
- Invited lectures: marine biology; carbon monoxide toxicity; stem cells and HBOT
- Diving medicine: physiology; decompression theory; treatment
- HBO medicine: physiology; treatment; technical and safety aspects
- Special session on "*Physicians and critical care in hyperbaric chambers*"
- GTÜM session: guideline – treatment of diving accidents; checklist – fitness to dive

The meeting format will be the usual EUBS style, with invited keynote lectures,
presentations of free papers (oral and posters) and an industry exhibition.

Call for abstracts
Abstracts for oral and poster presentations should be submitted electronically via <www.eubs2014.org>.
The Organising Committee intends to publish all accepted abstracts in a conference book and encourages all authors
to submit full papers for consideration in *Diving and Hyperbaric Medicine*.

Preliminary timetable
Registration is open via the website: <www.eubs2014.org>
01 May: End of early-bird registration period
01 June: Extended deadline for submission of abstracts
15 July: Notification of accepted abstracts

A detailed programme will become available on the website: <**www.eubs2014.org**> after 01 July 2014.

Language: The official language for all scientific sessions and the International DAN Diver's Day will be English.
The language for the GTÜM session will be German.

Satellite meetings
23 September	European Code of Practice for Hyperbaric Medicine: authors' meeting
23 September	5th Arthur Bornstein Workshop on Diving in Offshore Wind Farms
25 September	EDTC Medical Subcommittee Luncheon Meeting (by invitation only)
27 September	Dilemmas in running a hyperbaric research trial (afternoon)
27 September	Exhibition "Rescue Day" in front of the townhall of Wiesbaden (all day)
27 September	Reception by the Mayor of the City of Wiesbaden (evening)

For further information and hotel bookings see: <www.eubs2014.org>
Conference Secretariat (Peter Freitag) Phone:+49-(0)611-847-27-170
Fax: +49-(0)611-847-27-179
E-mail: <info@eubs2014.org>

SPUMS   **Notices and news**

Education Officer's Report May 2014

SPUMS Diplomas

Congratulations to the following SPUMS members who have been awarded their Diploma of Diving and Hyperbaric Medicine since my last report:

Mirjam Nolting: *The effects of hyperbaric oxygen therapy on pro-inflammatory cytokines involved in glucose homeostasis*

Denise Blake: *Nitrogen narcosis in hyperbaric chamber attendants*

Ian Gawthrope: *The cardiac effects of HBOT at 243kPa in healthy subjects using in-chamber echocardiography*

Andrew Ng: *Incidence of middle ear barotrauma in staged versus linear chamber compression during HBOT.*

Sam Koch: *Intravenous infusions in hyperbaric chambers: effect of syringe plunger construction on syringe function*

The above projects demonstrate the diversity of research being undertaken by SPUMS members.

Guidelines for Dip DHM (revised)

The guidelines for the Dip DHM have been revised and are published in this issue of DHM. It is now a requirement that members who have registered a Dip DHM project must remain financial for the duration of their project. There is also a three-year limit on inactive projects, after which they will be deregistered. This does not mean candidates are limited to three years to complete their project. The project can remain active by informing the Education Officer, in writing, of your progress and desire to remain registered. If the Education Officer has not heard from candidates for three years, then the project will be assumed to have lapsed. The member will then need to re-register the original project (or a new one) to become active again.

Current activity

The SPUMS Diploma continues to attract interest. During my time as Education Officer, there have been 16 Dip DHM's awarded and there are currently 19 active projects. In addition, 32 projects registered since 2000 were not progressed and have been classified as lapsed. There have also been multiple inquiries from members that have not led to projects.

It appears at present that much of the research is hospital-based. A major proportion of SPUMS members are general

practitioners. It would be great to receive some research submissions from this group, covering topics in primary health care. Comprehensive literature reviews are also accepted for the Diploma project, provided the topic has not been covered recently in the literature.

Table 1 summarises currently active SPUMS projects. If your name does not appear on the list below and you regard your project as still active, please write to the Education Officer.

There have been more inquiries from countries other than Australia and New Zealand. The Dip DHM qualification has a significant clinical practice requirement. It is also recognised by the Australian Federal Government as an appropriate qualification in Diving and Hyperbaric Medicine and for working at a hyperbaric facility in Australia. This affords the qualification significant status in Australia. The Australian Medicare Benefits Schedule section T1.1 contains the following definition:[1]

T 1.1 Hyperbaric Oxygen Therapy

Hyperbaric Oxygen Therapy not covered by these items would attract benefits on an attendance basis. For the purposes of these items, a comprehensive hyperbaric medicine facility means a separate hospital area that, on a 24 hour basis:
(a) is equipped and staffed so that it is capable of providing to a patient:
(i) hyperbaric oxygen therapy at a treatment pressure of at least 2.8 atmospheric pressure absolute (180 kilopascal gauge pressure); and
(ii) mechanical ventilation and invasive cardiovascular monitoring within a monoplace or multiplace chamber for the duration of the hyperbaric treatment; and
(b) is under the direction of at least 1 medical practitioner who is rostered, and immediately available, to the facility during the facility's ordinary working hours if the practitioner:
(i) is a specialist with training in diving and hyperbaric medicine; or
(ii) holds a Diploma of Diving and Hyperbaric Medicine of the South Pacific Underwater Medicine Society; and
(c) is staffed by:
(i) at least 1 medical practitioner with training in diving and hyperbaric medicine who is present in the facility and immediately available at all times when patients are being treated at the facility; and
(ii) at least 1 registered nurse with specific training in hyperbaric patient care to the published standards of the Hyperbaric Technicians and Nurses Association, who is present during hyperbaric oxygen therapy; and
(d) has admission and discharge policies in operation.

Table 1
Currently active SPUMS projects.

Year	Name	Project Title
2011	I Lewis	Performance of the Baxter Infusor LV under hyperbaric conditions
2011	K Thistlewaite	The effectiveness of HBOT for healing chronic venous leg ulcers: a randomised, double blind, placebo-controlled trial
2011	A Pullen	A survey of illicit drug use amongst Western Australian recreational divers (project completed June 2013, but not finished all DipDHM requirements)
2011	A Tyson	Understanding risks of asthma and diving – how well informed are divers with asthma?
2011	J Bruce-Thompson	A comparison between air and nitrox gas for recreational scuba divers to 18 msw
2012	I Dey	A longitudinal study of lung function in hyperbaric attendants
2012	G Kay	Resolution of decompression illness (DCI) as a function of time delay in treatment: 10 years of DCI treatment at the Townsville Hospital
2012	P Naidoo	A comparison of the tissue oxygenation achieved using different delivery devices and flow rates: implications for managing decompression illness
2012	C Oltvolgyi	A review of subatmospheric decompression illness
2012	M Reid	Decompression of recompression chamber operators and medical attendants during submarine rescue
2012	S Todd	Lymphatic decompression sickness: a retrospective observational study.
2013	S Sherlock	Outcomes of HBOT for sudden sensorineural hearing loss
2013	M Gelsomino	Development and testing of a device to allow normalised pressurisation rates and minimal staff input during the use of intercostal underwater drains in the hyperbaric environment
2013	L Elliott	A literature review of the assessment and management of inner ear barotrauma in occupational divers and recommendations for returning to diving
2013	B Devaney	Evaluation of the function and accuracy of the Braun Perfusor Space Syringe Pump under hyperbaric conditions
2013	D Teubner	The effect ambient pressure (and therefore gas density) has on the relationship between intrathoracic pressure and peak expiratory flow rate
2013	R Franks	Evaluation of novel oxygen delivery methods using a snorkel
2013	S Szekely	Hyperbaric oxygen therapy and insulin resistance – the effect of one treatment
2013	M Thomsen	Bench study of two new devices to display and maintain endotracheal cuff pressure under hyperbaric conditions
2014	H Beevor	Analysis of the patterns of presentation, management and outcomes of patients with iatrogenic arterial gas embolism referred to the Alfred Hyperbaric Unit
2014	J Wallace	Development of a gold-standard process for assessing safety for entry of new equipment into the hyperbaric environment

It is regretted that because of the clinical practice requirements associated with the Diploma, SPUMS is unable to register overseas doctors, unless they obtain medical registration in Australia or New Zealand and undertake their clinical Hyperbaric/Diving Medicine practice component in either country. There is, however, flexibility to allow part-time practice, and a number of SPUMS members have accessed this option to achieve their Dip DHM. Some hyperbaric facilities in Australia also have accreditation with ACRRM and RACGP, permitting GP registrars to undertake 6 months of accredited training in diving and hyperbaric medicine. The Dip DHM is currently recognised by the ANZCA as

an appropriate entry point for the ANZCA Certificate in Diving and Hyperbaric Medicine. A review by ANZCA of the Certificate is currently being finalised. I fully support the ANZCA Certificate programme. Even though there is only a small number of doctors training in the programme, it is important in providing a vocational training route under the auspices of a Speciality College.

Diving medicine courses

Three two-week courses are currently approved for the SPUMS Diploma: The Prince of Wales Hospital Introductory Course in Diving and Hyperbaric Medicine, the HMAS Penguin Medical Officers Course in Underwater Medicine, and the Royal Adelaide basic and advanced courses in Diving and Hyperbaric Medicine.

Appointment of new Education Officer

I retire as SPUMS Education Officer, effective from the SPUMS AGM in Bali in May. It has been an honour to serve in the role over the past six years. I wish to sincerely thank the Academic Board for their support, the SPUMS Executive and the many SPUMS members who have voluntarily provided their time as reviewers of other member's scientific work. Dr David Wilkinson, Royal Adelaide Hospital, will likely take over as Education Officer.

References

1   Australian Government Department of Health. Medicare Benefits Schedule Book. January 2014. p 31, 85. ISBN: 978-1-74186-061-0. Available from: <https://www.health.gov.au/internet/mbsonline/publishing.nsf/Content/D19F9DADF74138ADCA257C3600004D2D/$File/201401-Cat3.pdf>

Associate Professor David Smart
E-mail: *<david.smart@dhhs.tas.gov.au>*

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

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SPUMS 44th ASM 2015

Preliminary Notice

Venue: Palau, Micronesia
Tentative dates: 17–23 May 2015
(dates subject to flight schedules)

Convenor: Dr Catherine Meehan, Cairns

For further information at this early stage:
E-mail: <cmeehan@mcleodstmed.com.au>

SPUMS news is on the website

SPUMS notices and news, such as the minutes of the September 2013 meeting of the Executive Committee and the 2013 Annual General Meeting held during the tricontinental meeting on Réunion Island, including the officers' and financial reports, can now be found, along with all other information about the Society, on the website <www.spums.org.au>. In order to increase space for original research and educational articles and to minimise the rising costs of publishing *Diving and Hyperbaric Medicine*, the decision has been made to reduce the amount of society business appearing within these pages.

The

SPUMS

website is at
<www.spums.org.au>

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

SPUMS and Facebook

Remember to 'like' SPUMS at:
<http://www.facebook.com/pages/SPUMS-South-Pacific-Underwater-Medicine-Society/221855494509119>

SPUMS Diploma in Diving and Hyperbaric Medicine

Requirements for candidates (May 2014)

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. (S)he must be medically qualified, and remain a current financial member of the Society at least until they have completed all requirements of the Diploma.
2. (S)he must supply evidence of satisfactory completion of an examined two-week full-time course in diving and hyperbaric medicine at an approved facility. The list of such approved facilities may be found on the SPUMS website.
3. (S)he 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. (S)he must submit a written proposal for research in a relevant area of underwater or hyperbaric medicine, in a standard format, for approval before commencing their research project.
5. (S)he must produce, to the satisfaction of the Academic Board, a written report on the approved research project, in the form of a scientific paper suitable for publication. Accompanying this report should be a request to be considered for the SPUMS Diploma and supporting documentation for 1–4 above.

In the absence of other documentation, it will be assumed that the paper is to be submitted for publication in *Diving and Hyperbaric Medicine*. As such, the structure of the paper needs to broadly comply with the 'Instructions to Authors' available on the SPUMS website <www.spums.org.au> or at <www.dhmjournal.com>.

The paper may be submitted to journals other than *Diving and Hyperbaric Medicine*; however, even if published in another journal, the completed paper must be submitted to the Education Officer for assessment as a diploma paper. If the paper has been accepted for publication or published in another journal, then evidence of this should be provided.

The diploma paper will be assessed, and changes may be requested, before it is regarded to be of the standard required for award of the Diploma. Once completed to the reviewers' satisfaction, papers not already submitted to, or accepted by, other journals should be forwarded to the Editor of *Diving and Hyperbaric Medicine* for consideration. At this point the Diploma will be awarded, provided all other requirements are satisfied. Diploma projects submitted to *Diving and Hyperbaric Medicine* for consideration of publication will be subject to the Journal's own peer review process.

Additional information – prospective approval of projects is required

The candidate must contact the Education Officer in writing (or email) to advise of their intended candidacy and to discuss the proposed topic of their research. A written research proposal must be submitted before commencement of the research project.

All research reports must clearly test a hypothesis. Original basic or clinical research is acceptable. Case series reports may be acceptable if thoroughly documented, subject to quantitative analysis and if 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 the research project and the written report will be primarily the work of the candidate, and that the candidate is the first author where there are more than one.

It is expected that all research will be conducted in accordance with the joint NHMRC/AVCC statement and guidelines on research practice, available at: <www.nhmrc.gov.au/_files_nhmrc/publications/attachments/r39.pdf>, or the equivalent requirement of the country in which the research is conducted. All research involving humans or animals must be accompanied by documentary evidence of approval by an appropriate research ethics committee. Human studies must comply with the Declaration of Helsinki (1975, revised 2013). Clinical trials commenced after 2011 must have been registered at a recognised trial registry site such as the Australia and New Zealand Clinical Trials Registry <http://www.anzctr.org.au/> and details of the registration provided in the accompanying letter. Studies using animals must comply with National Health and Medical Research Council Guidelines or their equivalent in the country in which the work was conducted.

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

As of 01 June 2014, projects will be deemed to have lapsed if
1. The project is inactive for a period of three years, or
2. The candidate fails to renew SPUMS Membership in any year after their Diploma project is registered (but not completed).

With respect to 1 above, for unforeseen delays where the project will exceed three years, candidates must advise the Education Officer in writing if they wish their diploma project to remain active, and an additional three-year extension will be granted.

With respect to 2 above, if there are extenuating circumstances that a candidate is unable to maintain financial membership, then these must be advised in writing to the Education Officer for consideration by the SPUMS Executive.

If a project has lapsed, then the candidate must submit a new application as per these guidelines.

The Academic Board reserves the right to modify any of these requirements from time to time.

As of June 2014, the SPUMS Academic Board consists of:
Dr David Wilkinson, Education Officer;
Associate Professor Simon Mitchell;
Associate Professor (retired) Mike Davis;
Dr Denise Blake.

All enquiries and applications should be addressed to:
David Wilkinson
Fax: *+61-(0)8-8232-4207*
E-mail: *<education@spums.org.au>*

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

Certificate in Diving and Hyperbaric Medicine of the Australian and New Zealand College of Anaesthetists

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

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

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

Capita Selecta Dive Research English Seminars 2014
University of Amsterdam, The Netherlands

06 September 2014: *Pulmonology and Diving*
Speakers: Pascal Constantin, diving and hyperbaric physician; Jacques Regnard, sport-diving and hyperbaric physician; Nico Schellart, diving physiologist and medical physicist
29 November 2014: *Breath-hold diving*
Speakers: Rik Roskens; Erika Schagatay, environmental physiologist; Jochen Schipke, medical physiologist and diving physician

For full information contact: <www.duikresearch.org>

British Hyperbaric Association Annual Meeting 2014

Dates: 07–08 November 2014
Venue: The East Riding Medical Education Centre
Hull Royal Infirmary, East Yorkshire

Day 1: Oxygen and the traumatised brain
Day 2: Diving physiology / diving medicine

Keynote speakers:
Brad Sutherland, University of Oxford, UK
Shia Efrati. Assaf Harofeh Medical Centre, Israel
Galan Rockwood, University of Minnesota, USA
Ole Hildegard, University Hospital Copenhagen, Denmark
David Doollete USN Experimental Diving Unit, USA
Martin Sayer, UK National Scientific Diving Facility, Oban

The call for abstracts is now open. Please make submissions of 300 works or less to: <gerardladen@aol.com>

Royal Adelaide Hospital Hyperbaric Medicine Unit Courses 2014

Medical Officers' Course
Part 1: 01– 05 December (Lectures)
Part 2: 08–12 December

DMT Full Courses
06–24 October

DMT Refresher Courses
22 Sept–03 Oct

All enquiries to:
Lorna Mirabelli, Course Administrator
Phone: +61-(0)8-8222-5116
Fax: +61-(0)8-8232-4207
E-mail: <Lorna.Mirabelli@health.sa.gov.au>

Royal Australian Navy Medical Officers' Underwater Medicine Course 2014

Dates: 06–17 October 2014
Venue: HMAS PENGUIN, Sydney

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

Costs: AUD1,355 (without accommodation)
AUD2,300 (approx. with accommodation at HMAS Penguin)

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

Diving and Hyperbaric Medicine Index of contents, Vol 43, 2013

The Index of contents, Volume 43, 2013, is on the journal website <www.dhmjournal.com> and also on the SPUMS and EUBS websites.

Scott Haldane Foundation

The Scott Haldane Foundation is dedicated to education in diving medicine, and has organized more than 150 courses over the past 19 years, both in the Netherlands and abroad. Below is a list of remaining courses for 2014.

The courses Medical Examiner of Diver (part I and II) and the modules of the Diving Medicine Physician course fully comply with the ECHM/EDTC curriculum for Level 1 and 2d respectively and are accredited by the European College of Baromedicine.

Remaining courses for 2014
19–20 September: 21st In-depth course Diving Medicine: Fitness to dive in normal and extreme conditions, Loosdrecht, The Netherlands
04 October: Refresher course. AMC, Amsterdam
08–15 November: Basic course (medical examination of divers) Part 1. Costa Rica
15–22 November: 22nd In-depth course Diving Medicine: case-based diving medicine. Costa Rica
22–29 November: 22nd In-depth course Diving Medicine: case-based diving medicine. Costa Rica

For further information: <www.scotthaldane.org>

German Society for Diving and Hyperbaric Medicine

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

DAN Europe

DAN Europe has a fresh, multilingual selection of recent news, articles and events featuring DAN and its staff.
Go to the website: <http://www.daneurope.org/web/guest/>

**DIVING HISTORICAL SOCIETY
AUSTRALIA, SE ASIA**

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

18th International Congress on Hyperbaric Medicine

03–06 December 2014
Buenos Aires, Argentina
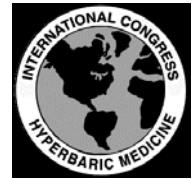
The ICHM is a worldwide organization for physicians and scientists interested in diving and hyperbaric medicine. The organization has minimal formal structure and is entirely dedicated to hosting an international scientific congress every three years.

ICHM Committee (2011–2014):
President: Prof Dr Jorge B Pisarello (Argentina)
Executive Director: Dr Alessandro Marroni (Italy)
Secretary: Assoc Prof Michael Bennett (Australia)

Registration: Online registration is now open
Website:
<http://ichm.drupalgardens.com/content/what-ichm-0>

Hyperbaric Oxygen, Karolinska

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

You need to register to obtain a password via e-mail. Once registered, watch the lectures online, or download them to your iPhone or computer for later viewing. We offer video lectures from:
• The 5th Karolinska PG course in clinical hyperbaric oxygen therapy, 07 May 2009.
• The European Committee for Hyperbaric Medicine *"Oxygen and infection"* Conference, 08–09 May 2009.
• The 17th International Congress on Hyperbaric Medicine, Cape Town, 17–18 March 2011.

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

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

Undersea and Hyperbaric Medicine Society Annual Scientific Meeting 2014

Dates: 19–21 June
Venue: Hyatt Regency St Louis at the Arch, St Louis, MS
For full information go to: <www.uhms.org>

Instructions to authors
(updated June 2014)

Diving and Hyperbaric Medicine (DHM) is the combined journal of the South Pacific Underwater Medicine Society (SPUMS) and the European Underwater and Baromedical Society (EUBS) and seeks to publish papers of high quality on all aspects of diving and hyperbaric medicine of interest to diving medical professionals, physicians of all specialties, and members of the diving and hyperbaric industries. Manuscripts must be offered exclusively to *Diving and Hyperbaric Medicine*, unless clearly authenticated copyright exemption accompanies the manuscript. All manuscripts will be subject to peer review. Accepted contributions will also be subject to editing.

Address: The Editor, Diving and Hyperbaric Medicine
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Fax: +64-(0)3-329-6810

Contributions should be submitted electronically to:
E-mail: <submissions@dhmjournal.com>

European Editor: <euroeditor@dhmjournal.com>

Editorial Assistant: <editorialassist@dhmjournal.com>

Requirements for manuscripts

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

Original Articles, **Technical Reports and case series:** up to 3,000 words is preferred, and 30 references (excluded from word count). These articles should be subdivided into the following sections: a structured **Abstract** of no more than 250 words, **Introduction**, **Methods**, **Results**, **Discussion**, **Conclusions**, **References** (excluded from word count). **Acknowledgements,** which should be brief, **Funding** sources and any **Conflicts of interest** should be listed after the references.

Review Articles: up to 5,000 words is preferred and 60 references (excluded from word count); include an **Abstract** of no more than 300 words (excluded from word count); structure of the article is at the author(s)' discretion.

Case Reports, **Short Communications** and **Work In Progress reports:** maximum 2,000 words, and 20 references (excluded from word count); include an **Abstract** of no more than 200 words (excluded from word count).

Educational articles, **commentaries** and **case reports** for 'The Diving Doctor's Diary', 'World as it is', 'Opinion'

or '**Historical**' occasional sections may vary in format and length, but should generally be a maximum of 3,000 words and 15 references (excluded from word count).

Letters to the Editor: (generally maximum 600 words, plus one figure or table and 5 references).

DHM follows as much as possible the ***Recommendations for the conduct, reporting, editing and publication of scholarly work in medical journals***. International Committee of Medical Journal Editors; December 2013. Available from: <http://www.icmje.org/icmje-recommendations.pdf>.

Authors are strongly encouraged to read this and other documents on the ICMJE website in preparing their submission. Authors should also consult guidelines for specific types of study (e.g., the CONSORT guidelines for the reporting of randomized controlled trials); see <http://equator-network.org>.

All submissions must comply with the requirements below. Manuscripts not complying with these instructions will be returned to the author for correction before consideration.

Inclusion of more than six authors in any one manuscript requires justification. Authors must have contributed significantly to the study (see http://www.dhmjournal.com/index.php/instructions-to-authors for more information).

Documents must be submitted electronically. Multiple or large files may be bundled as a Zip file and sent as an e-mail attachment or using internet services such as <https://www.wetransfer.com> or <www.yousendit.com>.

All articles should include a **Title Page**, giving the title of the paper and the full names of all authors (given names first, followed by the family/surname), their principal qualifications and affiliations at the time of doing the work being reported. One author must be identified as correspondent, with their full postal address, phone number and e-mail address supplied. If a different author to the principal (first) author, then full contact details for her/him are also required.

A **Covering Letter** signed by the principal (first) author must accompany all submissions. Authors should complete the proforma cover letter on the DHM website <http://www.dhmjournal.com/index.php/instructions-to-authors>.

A maximum of seven **Key Words** best describing the paper should be chosen from the list on the journal website <http://www.dhmjournal.com/files/Key_word_list_Jan_2014.pdf>. New key words, complementary with the NLM MeSH, <http://www.nlm.nih.gov/mesh/> will be used at the discretion of the Editor. Key words should be placed at the bottom of the title page.

Text: The preferred format is Microsoft Office Word or rich text format (RTF), with 1.5 line spacing, using both upper and lower case throughout. The preferred font is Times New Roman, font size 11 or 12. Headings should conform to the current format in DHM:

Section heading
SUBSECTION HEADING 1
Subsection heading 2

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

English spelling will be in accordance with the *Concise Oxford Dictionary*, 11th edition revised (or later). Oxford: Oxford University Press; 2006.

Measurements are to be in SI units (mmHg are acceptable for blood pressure measurements) and normal ranges should be included where appropriate. Authors are referred to the online BIPM brochure, International Bureau of Weights and Measures (2006), *The International System of Units* (SI), 8th ed, available at ISBN 9282222136 : <http://www.bipm.org/utils/common/pdf/si_brochure_8_en.pdf>, or Baron DN, McKenzie Clarke H, editors. *Units, symbols and abbreviations. A guide for biological and medical editors and authors*, 6th edition. London: Royal Society of Medicine; 2008. Atmospheric and gas partial pressures and blood gas values should be presented in kPa (ATA/bar/mmHg may be provided in parenthesis on the first occasion). The ambient pressure should be clearly identified whether it is given in absolute (a) or gauge (g) values. Water depths should be presented in metres' sea (or fresh) water (msw or mfw). Cylinder pressures and inspired gas pressures in a rebreather apparatus may be presented as 'bar'.

Abbreviations may be used once they have been shown in parenthesis after the complete expression. For example, decompression illness (DCI) can thereafter be referred to as DCI. This applies separately to the abstract and main text. Use generally accepted abbreviations rather than neologisms of your own invention.

References

The Journal reference style is based **exactly** on that of the *International Committee of Medical Journal Editors (ICMJE) Uniform requirements for manuscripts submitted to biomedical journals*. Examples of the formats for different types of references (journal articles, books, monographs, electronic material, etc) are given in detail on the website: <http://www.nlm.nih.gov/bsd/uniform_requirements.html> (last updated 20 August 2013).

Correct formatting and the accuracy of references in a submission are the responsibility of the author(s).

Additional requirements for DHM are:
References should be numbered consecutively in the order in which they are first mentioned in the text, tables or figures as superscript numbers, preferably at the end of the sentence **after** the full stop.[1,2]
References appearing in table or figure legends should continue the sequence of references in the main text of the article in accordance with the position of citing the table/figure in the text.
Use MEDLINE abbreviations for journal names. The *List of Journals Indexed for MEDLINE* publication ceased with the 2008 edition. The journals database: <http://www.ncbi.nlm.nih.gov/sites/entrez?Db=journals&Cmd=DetailsSearch&Term=currentlyindexed[All]>
can be used to obtain a list of currently indexed MEDLINE journal titles. Journals not indexed in MEDLINE should have the journal name written in full.
Abstracts from meeting proceedings should not be used as references unless absolutely essential, as these are generally not peer-reviewed material.
If using EndNote to prepare the references in the document, then the submitted text should have all EndNote field codes removed before submission (see EndNote website for advice on how to do this).
Verifying the accuracy of references against the original documents is the responsibility of authors.
Personal communications should appear as such in the text and not be included in the reference list (e.g., Other AN, personal communication, year).

'Long' and 'short' examples of a journal reference in the full ICMJE format are shown below:
    Wilson CM, Sayer MDJ. Transportation of divers with decompression illness on the west coast of Scotland. Diving and Hyperbaric Medicine. 2011 June;41(2):64-69.
If a journal carries continuous pagination throughout a volume (as many medical journals do) then the month and issue number should be omitted and the pagination reduced.

Therefore, the shortened ICMJE version used in DHM is:
    Wilson CM, Sayer MDJ. Transportation of divers with decompression illness on the west coast of Scotland. Diving Hyperb Med. 2011;41:64-9.
An example book reference is:
    Kindwall EP, Whelan HT, editors. Hyperbaric medicine practice, 3rd ed. Flagstaff, AZ: Best Publishing Company; 2008.
Examples of all other types of references are to be found on the uniform requirements website.

Illustrations, figures and tables

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

(Table 1)", etc. The approximate positions of tables and figures should be identified in the text.

No captions should appear within the body of the table or image, but should be placed in the legend. **Legends** should generally contain fewer than 40 words and must be listed on a separate page at the end of the main text file. Any definition of symbols used in the figures should appear within the white space of the figure to allow the figure to attain maximum size, or be submitted separately or be included in the legend rather than in the figure. Figures should be readable in black and white, with no unnecessary shading, grid lines or box lines. Both markers and lines should be unique to facilitate easy discrimination of the data being presented.

If any figures, images or tables are to be reproduced from previous publications, it is the responsibility of the author to obtain the necessary permissions.

Table data should be presented either as tab-spaced normal text or using table format, with tab-separated columns auto-formatted to fit content. No grid lines, borders or shading should be used.

Illustrations and X-rays should be submitted as separate electronic files in TIFF, high resolution JPEG or BMP format. Colour is available only at the author's request and will be at the author's expense (currently approximately AUD600 for a single A4 page). Therefore, authors need to convert figures and images to grayscale to ensure that contrast within the image is sufficient for clarity when printed. Any graphs or histograms created in Excel should be sent within their original Excel file, including the data table(s) from which they were produced. This allows the journal office to edit figures for maximum legibility when printed.

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

Scanned photographs should be submitted as TIFF, JPG or BMP files at a minimum resolution of 300 dpi. Magnification should be indicated for photomicrographs, and consideration given to the positioning of labels on diagnostic material as this can greatly influence the size of reproduction that can be achieved in the published article.

Consent and ethical approval

Studies on human subjects must comply with the Helsinki Declaration of 1975, as revised in 2013 (see <http://www.dhmjournal.com/index.php/instructions-to-authors> for a copy. Studies using animals must comply with National Health and Medical Research Council Guidelines or their equivalent in the country in which the work was conducted.

A statement affirming Ethics Committee (Institutional Review Board) approval (and the approval number) should be included in the text. A copy of that approval should be provided with the submission. Patient details must be removed and photographs made unrecognizable. Written informed consent should be indicated in the article.

Clinical trials commenced after 2011 must have been registered at a recognised trial registry site such as the Australia and New Zealand Clinical Trials Registry <http://www.anzctr.org.au/> or EudraCT in Europe <https://eudract.ema.europa.eu/> and details of the registration provided in the accompanying letter.

For individual case reports, patient consent to anonymous publication of images or their clinical details must have been obtained. Case series, where only limited, anonymous summary data are reported, do not require patient consent, but do require ethical approval.

English as a second language

Adequate English usage and grammar are prerequisites for acceptance of a paper. However, some editorial assistance may be provided to authors for whom English is not their native language. English language services can be accessed through the European Association of Science Editors (EASE) website <http://www.ease.org.uk/>. Alternatively, the journal office may be able to put you in touch with a commercial scientific ghost writer.

Copyright

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

Articles are embargoed for one year from the date of publication, after which they will be free to access. If authors wish their article to be free to access immediately upon publication, then a fee (determined by the publishers, EUBS and SPUMS) will be charged for its release.

SPUMS and EUBS Annual Scientific Meetings

DHM has published articles based on many of the presentations from SPUMS annual scientific meetings (ASM). Presenters, including the Guest Speaker(s),

are reminded that this is an explicit condition of their participation in the SPUMS meetings, but recognizing that not all presentations are suitable for publication in DHM. Speakers at EUBS meetings, both those giving keynote addresses and those presenting previously unpublished research are strongly encouraged to submit manuscripts to DHM. All such articles are subject to the above requirements of standards, presentation and peer review.

Zetterström Award

The author(s) of the scientific poster winning the Zetterström Award at each EUBS ASM explicitly agree(s) to submit an article based on their poster to DHM. This paper is subject to the above requirements of standards and presentation and will be subject to peer review.

Musimu Award

Recipients of the Musimu Award of the EUBS are strongly encouraged to publish their research in DHM.

SPUMS Diploma dissertations

It is the policy of SPUMS that diploma candidates are strongly encouraged to publish their dissertation in DHM. Most dissertations require editing for submission, and these *Instructions to Authors* should be used to guide this.

Synopses or summaries of master's or doctoral theses will also be considered in order to draw the diving and hyperbaric medical and scientific community's attention to the work of young researchers. Permission to reprint such material may be required from the host institution, and obtaining this is the author's responsibility.

Publication schedule

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

The review process typically takes about 8 weeks to complete, but can be longer. If additional reviews are needed, this will prolong the process. Papers are generally scheduled for publication in order of final acceptance. The Editor retains the right to delay publication in the interests of the Journal.

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

Reprints

Following publication, one complimentary copy of DHM will be sent to the corresponding author, if they are not a current member of SPUMS/EUBS. A PDF copy of articles will also be forwarded to the corresponding author. A limited number of additional print copies of the journal issue containing the article are available for purchase from the SPUMS Administrator, <admin@spums.org.au>.

Editor's note:

The *Instructions to Authors* as printed in this issue are available as a pdf file on the DHM website at:
<http://www.dhmjournal.com/index.php/instructions-to-authors>
They are also available on the EUBS and SPUMS websites.

These instructions have required further revision since the March 2014 version in order to comply with a new edition of the *Recommendations for the conduct, reporting, editing and publication of scholarly work in medical journals*, International Committee of Medical Journal Editors, published December 2013.

A shortened, single-page version of these instructions, as published in the past, will no longer appear in the Journal.

DIVER EMERGENCY SERVICES PHONE NUMBERS

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

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

ASIA
+10-4500-9113 (Korea)
+81-3-3812-4999 (Japan)

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

EUROPE
+39-6-4211-8685 (24-hour hotline)

UNITED KINGDOM
+44-7740-251-635

USA
+1-919-684-9111

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

DAN ASIA-PACIFIC DIVE ACCIDENT REPORTING PROJECT

This project is an ongoing investigation seeking to document all types and severities of diving-related accidents. All information is treated confidentially with regard to identifying details when utilised in reports on fatal and non-fatal cases. Such reports may be used by interested parties to increase diving safety through better awareness of critical factors. Information may be sent (in confidence unless otherwise agreed) to:

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

DAN Asia-Pacific NON-FATAL DIVING INCIDENTS REPORTING (NFDIR)

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

The NFDIR reporting form can be accessed on line at the DAN AP website:
<www.danasiapacific.org/main/accident/nfdir.php>

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