Diving and Hyperbaric Medicine

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EUBS

Immersion pulmonary oedema reviewed

Occupational diving does not cause hearing loss

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PURPOSES OF THE SOCIETIES

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

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DIVING AND HYPERBARIC MEDICINE

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The Editor's offering

Most who read this column will be members of the European Underwater and Baromedical Society (EUBS) or the South Pacific Underwater Medical Society (SPUMS); two niche medical craft groups for whom *Diving and Hyperbaric Medicine* (DHM) represents their societal footprints in the world. The journal is a pivotal thread in the fabric of both societies, and it is appropriate as we transition into a new editorship in 2019 to reflect on its journey over the years, and where it is going in future.

DHM began as the SPUMS Newsletter in May 1971 with Carl Edmonds as the first editor. There followed a brief reign by Bob Thomas, and a long editorship by Douglas Walker between 1974 and 1990. The newsletter became SPUMS Journal around 1980, and after John Knight became editor in 1990, the format evolved into something resembling the modern content. John was responsible for the journal's first listing on a searchable database (EMBASE) in 2000. In 2002, Mike Davis became Editor. Whilst working to enhance the academic rigor of the journal's various processes, Mike presided over several pivotal projects that have seen DHM become an influential, modern medical publication.

First, in the mid-2000s, negotiations were entered into with EUBS regarding amalgamation of the *SPUMS Journal* with the *European Journal of Underwater and Hyperbaric Medicine* published by EUBS, largely through the editorial efforts of Peter Mueller. In 2007, *SPUMS Journal* was renamed *Diving and Hyperbaric Medicine*, with the first issue published jointly by EUBS and SPUMS in March 2008. Second, the challenging process of applying for Medline listing was successful in 2011. This truly marked the journal's coming of age as a serious scientific publication. Third, in 2018, with huge support from Nicky Telles, DHM's editorial assistant, the transition from print to a fully electronic format was achieved. Finally, also in 2018 and again with crucial support from Nicky, an application for our papers to be deposited electronically in PubMed Central was successful. From March 2017 forward, full text and pdf versions of our papers will be available with the click of a mouse to those performing literature searches (although the one-year members-only embargo remains).

We must acknowledge the extraordinary job Mike has done for our societies. DHM has undergone a remarkable surge in impact factor over recent years (from 0.607 in 2013 to 1.197 in 2017) and this undoubtedly reflects a consistent improvement in the quality and rigor of articles appearing in the journal. My apprenticing into the role of Editor over the past six months has given me some insight into the enormity of the job when done to the standard that Mike has set. I assumed the role of Editor in January 2019, with Mike looking over my shoulder for the first six months. I am also relieved our current European Editor, Lesley Blogg will continue to be another steadying influence and source of continuity. Although I am an anaesthesiologist and head of a university academic department, my main message about myself as incoming editor is that I am very much one of you: a diver, diving/hyperbaric physician and scientist. I am passionate about the field and determined to perpetuate the great work my predecessors in both societies have done in developing a fantastic journal.

With this in mind, I urge all our publishing members to give their own journal strong consideration when deciding where to submit manuscripts. DHM can only be as good as the science published therein and relies on a flow of quality submissions. DHM will, in turn, provide constructive and (as much as possible) timely review. With the recent gain in impact factor and deposition of papers in PubMed Central, DHM represents a very suitable home for high quality work in our field; all to the betterment of our societies' reputations. Parenthetically, over recent times I have tried to live up to this expectation by submitting much of my own work to DHM. I mention this to reassure members that when editorial staff submit to the journal, they have no role in management of the manuscripts whose review is strictly organized and adjudicated by non-conflicted others.

Further to maintaining the quality of the journal, I am committed to high quality review and constructive engagement with authors to ensure interesting work is published. My recent exposure to the inner workings of DHM has already identified typical stumbling blocks. Simple solutions to common problems include getting statistical help at the planning stages of a study, getting English as a second language help when preparing manuscripts and following the DHM instructions for authors (especially in relation to reference formatting – which is evolving in this electronic age). An increasing problem is the submission of observational human studies without ethics approval. This is claimed to be acceptable in some jurisdictions, and we have generally taken the view that approaches which are indisputably acceptable within an author's research environment are acceptable to DHM. But this is an evolving situation, and journals are becoming stricter. My advice is to engage with ethics committees on observational studies; they are usually approved with little difficulty.

I close my first editorial in DHM with a thank you to all members of SPUMS and EUBS, and particularly the societal presidents David Smart and Ole Hyldegaard, for trusting me with this important role. Irrespective of the preceding history, from 2008 the journal was (and remains) a joint publication with both societies (and their members) as equal partners going forward. Let's make our journal the best it can be.

Simon Mitchell

Key words
Editorials; Medical society; General interest

Front cover: Editorial Board member Associate Professor David Doolette diving at Northern Arch, Poor Knights Islands, New Zealand, December 2018. Photo: Simon Mitchell

Original articles

The impact of diving on hearing: a 10–25 year audit of New Zealand professional divers

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Key words
Audiology; Fitness to dive; Hearing loss; Medicals – diving; Occupational diving; Surveillance

Abstract
(Sames C, Gorman DF, Mitchell SJ, Zhou L. The impact of diving on hearing: a 10–25 year audit of New Zealand professional divers. Diving and Hyperbaric Medicine. 2019 March 31;49(1):2–8. doi: 10.28920/dhm49.1.2-8. PMID: 30856661.)
Introduction: Surveillance of professional divers' hearing is routinely undertaken on an annual basis despite lack of evidence of benefit to the diver. The aim of this study was to determine the magnitude and significance of changes in auditory function over a 10–25 year period of occupational diving with the intention of informing future health surveillance policy for professional divers.
Methods: All divers with adequate audiological records spanning at least 10 years were identified from the New Zealand occupational diver database. Changes in auditory function over time were compared with internationally accepted normative values. Any significant changes were tested for correlation with diving exposure, smoking history and body mass index.
Results: The audiological records of 227 professional divers were analysed for periods ranging from 10 to 25 years. Initial hearing was poorer than population norms, and deterioration over the observation period was less than that predicted by normative data. Changes in hearing were not related to diving exposure, or smoking history.
Conclusion: Audiological changes over 10 to 25 years of occupational diving were not found to be significantly different from age-related changes. Routine annual audiological testing of professional divers does not appear to be justifiable.

Introduction

Hearing loss is recognised as an important and preventable occupational injury. In most industries, exposure to excessive noise is the responsible mechanism, and where all other measures to reduce noise levels have been exhausted, employers are obligated to provide hearing protection and appropriate staff education. For working divers, however, hearing can be adversely affected by several mechanisms that are independent of noise exposure. These include: conductive loss due to middle ear barotrauma (MEBt), which impairs transduction of sound by the tympanic membrane and ossicular chain;[1] sensorineural deficit due to noise-induced hearing loss (NIHL); barotraumatic damage to the inner-ear structures[2,3] and inner ear decompression sickness (DCS).[4,5] Apart from these discrete barotraumatic and DCS events, doubt remains as to whether diving *per se* has a clinically significant negative impact on hearing over the long term. Controlling for the effects of increasing age and discrete injurious events remains a confounding factor for research in this area. The value of such research, for divers and employers, is that after identifying and either eliminating or minimising any preventable causes of hearing loss, including high-risk diving practices, they could have realistic, evidence-based, expectations about the impact of diving on hearing. The objective of the current study was to identify evidence of hearing loss that appears related to long-term occupational diving, with the intention of informing auditory surveillance policy for divers.

Reviews of diving-related hearing loss suggest that long-term changes are not clinically significant, and that, after correcting for age, any deterioration is likely due to noise exposure or trauma.[6,7] However, results of individual studies are variable, with some studies reporting significant hearing loss and a correlation with diving experience, and others reporting no such loss or correlation. For example, it was found that at most frequencies, divers had poorer hearing than age-matched otologically normal subjects at both the initial and final examination six years later.[8,9] Also, a significant correlation was found between hearing loss and both diving experience and smoking. Similarly, in a

Table 1
Characteristics of 227 occupational divers undergoing audiological testing over periods of between 10 and 25 years; *n* – number (mean or median); * – 2nd medical refers to data collected from each diver's most recent medical examination; number of dives refers to the year prior to the most recent audiometry

Characteristic	*n (% or range)*
Male	204 (90)
Female	23 (10)
Non-smoker	166 (73)
Smoker and ex-smoker	61 (27)
Dives/year (at 2nd medical*)	39 (median) (0–350)
Age (at 2nd medical*)	47 (median) (31–75)
BMI (at 2nd medical*)	27.1 ($kg \cdot m^{-2}$) (18.8–40.8)
Age change (yrs)	12 (median) (10–25)
Scientific	80 (35)
Commercial	45 (20)
Instructor	37 (17)
Construction	33 (14)
Aquaculture	15 (7)
Military/Police/Customs	8 (3)
Film	8 (3)
HBU attendant	1 (< 1)

prospective series of studies of professional divers over a twelve year period, although divers had better hearing than the general population at both initial and final examinations (in contrast to the above findings), minor reduction in hearing seemed related to diving exposure.[10–12] Similar results were reported in a five-year prospective study of Japanese fishery divers,[13] and in a cross-sectional study of Malaysian Navy divers whose hearing deteriorated at a faster rate than controls.[14] However, in a previous cross-sectional study, no differences were found between the hearing of a group of construction divers with a mean of 20 years' diving experience and a matched control group of workshop workers.[15] Another prospective study of professional divers over six years reported no correlation between hearing loss and diving frequency or history of middle ear barotrauma.[16] Other studies of professional divers have also found no significant difference in hearing between divers and control subjects or a relationship between hearing loss and diving experience.[17–19] Most studies of recreational divers have reported no significant hearing impairment compared with control subjects.[20–24] All this suggests that increased noise exposure, more likely to be encountered by professional divers, is the most plausible explanation for any finding of increased hearing loss in that group. A comparison of professional divers and offshore workers found that these divers were indeed more likely to suffer noise-induced hearing loss.[25]

As one of only two mandatory physical investigations routinely required of professional divers, the other being assessment of lung function, investigation of the evidence underlying the requirement for audiometry, repeated annually in most countries, is both apposite and overdue.

Method

This study was reviewed and authorised by the Waitemata District Health Board Research and Knowledge Centre and was deemed not to require full review by a Health and Disability Ethics Committee (reference no. RM13630). As part of their medical assessments, all divers signed consent for the use of their anonymised health data for research purposes.

The New Zealand occupational divers' database was audited for all divers with two hearing assessments separated by at least 10 years. We used the earliest hearing assessment available on our database as their baseline, but this was not invariably the first hearing assessment in the diver's career. To clarify, the duration of occupational diving between assessments was not necessarily equivalent to the total occupational diving experience of any diver. Qualifying divers' records were also audited for a history of middle (MEBt) or inner (IEBt) ear barotrauma, inner ear DCS, pre-existing hearing loss or tinnitus.

Initial and follow-up recordings of pure tone air conduction hearing thresholds, in decibels (dB), were collated for each ear for the frequencies of 500 Hz, 1, 2, 4, 6 and 8 kHz. For each of these recordings, a corresponding age-adjusted value was calculated by subtracting from the observed value, the median normal hearing threshold, derived from the appropriate ISO 7029:2017 prediction equation for otologically normal subjects, based on age and gender.[26] This model uses, as the reference zero level, the median of the 18-year old population. So, for example, the recorded thresholds for an 18-year old would require no adjustment. The changes in both recorded and adjusted values were calculated between the initial dataset and the paired dataset recorded after a period of 10–25 years of occupational diving. Correlations were sought between changes in hearing and duration of professional diving experience, intensity of

Figure 1
Age-adjusted, initial observed and predicted hearing thresholds of 227 divers (medians and 95% confidence limits); predicted values were derived from ISO Standard 7029[26]

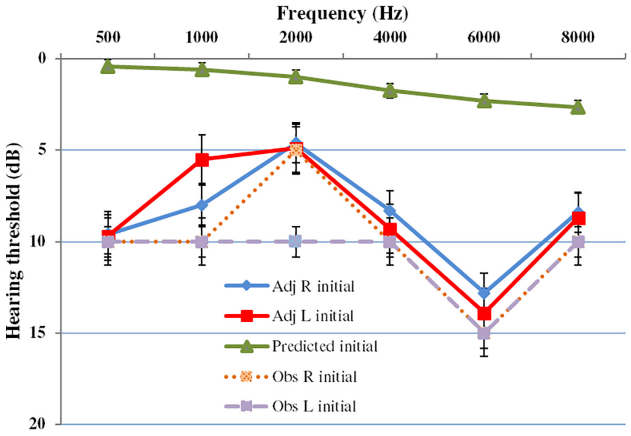


Figure 2
Age-adjusted, observed and predicted hearing thresholds of 227 divers after 10–25 years of diving (median and 95% confidence limits); predicted values were derived from ISO Standard 7029[26]

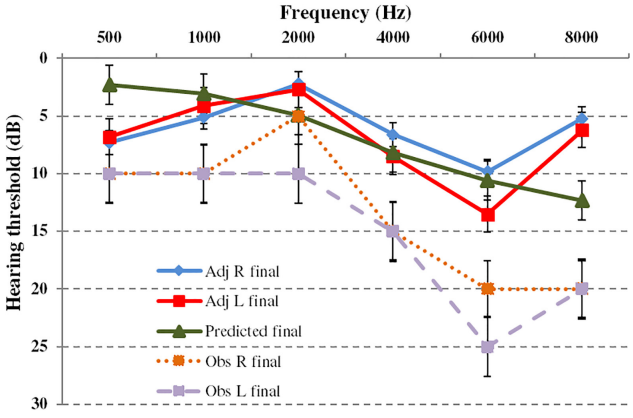


Figure 3
Degree of hearing loss at certain frequencies in 227 divers over 10–25 years

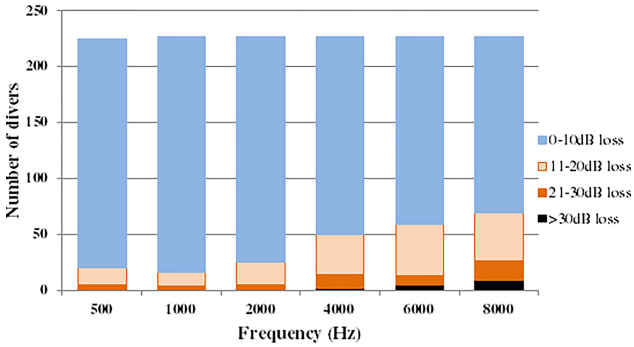
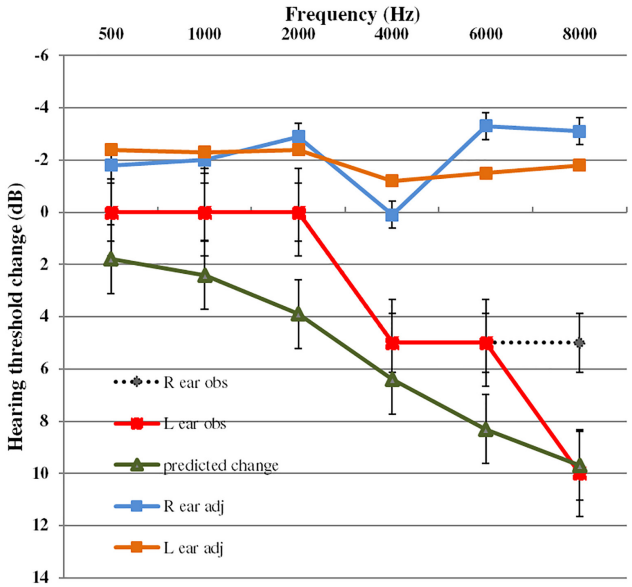


Figure 4
Change in observed and age-adjusted hearing thresholds over 10–25 years of diving compared with predicted change (medians and 95% confidence limits); predicted values were derived from ISO Standard 7029[26]



diving (as described below), smoking status (categorised as non-smokers, ex-smokers and current smokers) and body mass index (BMI).

Statistical analysis used SAS® v9.4 software (SAS Institute Inc., Cary, North Carolina, USA). Frequency and proportion (%) were used for describing categorical variables, such as gender, smoking status and type of diving. Median with minimum and maximum were used for describing the continuous variables including age (and change in age used to represent duration of diving experience), BMI and number of dives per year, as they did not follow a normal distribution. Median, and its distribution-free 95% confidence intervals, were used to present the study outcomes including observed, predicted and age/gender-adjusted values of hearing thresholds. Robust regression models (using the ROBUSTREG procedure, an alternative to least squares regression, that provides stable results in the presence of outliers, and limits their influence) and analysis of co-variance with general linear models were used in multiple regression analyses. A P-value of < 0.05 was considered to be statistically significant. Type 1 error was not adjusted for multiple comparisons, in order to allow for outliers and include all possible important information.

Results

Two-hundred and twenty-seven divers satisfied the entry criterion of having adequate records spanning periods of 10–25 years (median 12 years). Demographic data for the divers are presented in Table 1.

None of the divers had a recorded history of either IEBt or DCS, but two had a history of MEBt, and 44 (19.4%) had a record of either pre-existing hearing loss and/or chronic

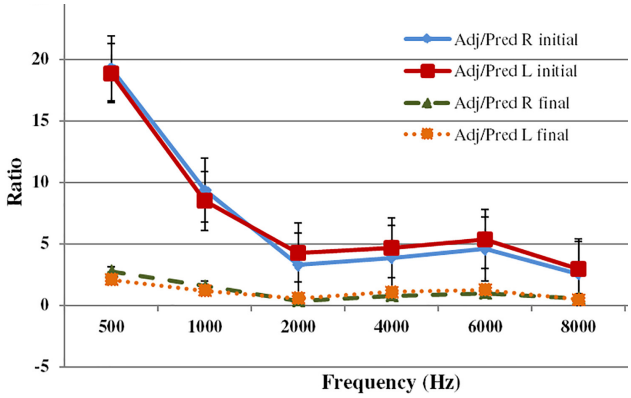
Table 2

Changes of the observed, age-gender-adjusted and predicted hearing threshold values of 227 occupational divers over 10–25 years of diving; * 25 percentile; ** 75 percentile; *** predicted values were derived from ISO Standard 7029[26]; all values are expressed in decibels (dB)

Frequency (Hz)	Side	Median	95% CI of median		Q1*	Q3**	Interquartile range
			Lower	Upper			
Change in observed values							
500	R	0	0	0	-5	5	10
500	L	0	0	0	-5	5	10
1000	R	0	0	0	-5	5	10
1000	L	0	0	0	-5	5	10
2000	R	0	0	0	-5	5	10
2000	L	0	0	0	-5	5	10
4000	R	5	5	5	0	15	15
4000	L	5	5	5	0	15	15
6000	R	5	5	10	-5	15	20
6000	L	5	5	10	0	15	15
8000	R	5	5	10	0	15	15
8000	L	10	5	10	0	20	20
Change in age/gender- adjusted values							
500	R	-1.8	-2.8	-1.2	-7.9	3.6	11.5
500	L	-2.4	-3.8	-1.4	-7.7	3.3	11.0
1000	R	-2.0	-3.6	-1.2	-7.0	3.2	10.3
1000	L	-2.3	-3.4	-1.6	-7.4	2.5	9.9
2000	R	-2.9	-3.9	-2.0	-8.0	2.4	10.4
2000	L	-2.4	-3.7	-1.7	-7.8	2.0	9.8
4000	R	0.1	-1.9	1.1	-7.3	5.3	12.5
4000	L	-1.2	-2.9	0.9	-8.5	6.1	14.7
6000	R	-3.3	-4.6	-1.1	-10.5	6.1	16.7
6000	L	-1.5	-3.1	0.4	-8.9	5.8	14.7
8000	R	-3.1	-5.3	-0.8	-10.0	7.5	17.4
8000	L	-1.9	-3.5	0.4	-9.8	8.7	18.5
Change in predicted values* **							
500	both	1.8	1.6	2.0	1.1	3.0	1.9
1000	both	2.4	2.1	2.6	1.4	3.9	2.5
2000	both	3.8	3.1	4.1	2.3	6.1	3.8
4000	both	6.3	5.5	6.7	3.8	9.6	5.8
6000	both	8.0	7.1	8.5	4.9	12.2	7.3
8000	both	9.4	8.2	10.0	5.7	14.4	8.7

tinnitus. Both initial and final hearing thresholds for the group were higher than normal values, meaning that sounds were detected at a higher sound intensity and indicating that hearing was slightly worse than predicted for age. However, changes over the recording period were smaller than predicted by the relevant ISO Standard.[26] Both age-adjusted and observed hearing thresholds for right and left ears were compared with predicted (normal) values for initial (Figure 1) and final recordings (Figure 2). The median values and 95% confidence limits of changes in observed and predicted thresholds are shown in Table 2, together with 25 and 75 percentiles and interquartile ranges. Despite

Figure 5
Ratio of age-adjusted and predicted* hearing thresholds of divers before and after 10–25 years of diving (medians and 95% confidence limits); * predicted values were derived from ISO Standard 7029[26]



more than half of the group showing a significant hearing reduction in at least one ear and at one frequency, more notable at the higher frequencies (Figure 3), median values for the group showed no change in the hearing thresholds at lower frequencies (500 Hz, 1 kHz, 2 kHz) in either ear, and only minor changes at the higher frequencies (4 kHz, 6 kHz, 8 kHz) that were less than predicted for increasing age (Figure 4).

The reduction, over time, in the difference between age-adjusted recordings and predicted thresholds is further demonstrated by comparison of the ratio of median age-adjusted observations and predicted thresholds at initial and subsequent testing after 10–25 years of occupational diving (Figure 5).

This reduction in difference (approaching the predicted values) of thresholds is significantly more pronounced at the low frequencies (500 Hz and 1000 Hz). Multiple regression analysis, using the models described above, found no significant correlation between hearing change and intensity of diving or smoking status, but at most frequencies there was a statistically significant association with BMI ($P < 0.05$ for multiple comparisons). No correlation was found between hearing change and duration of diving apart from at 4 kHz in the left ear ($P = 0.034$) and 8 kHz in the right ear ($P = 0.038$).

Discussion

Our data show that, for this sample of 227 professional divers, there was less deterioration in hearing after 10–25 years of professional diving than would be expected in the age-matched general population. However, we do not suggest that diving confers a degree of hearing protection, as most of the demonstrated changes are too small to be clinically relevant, and fall within the margin of error of many commonly used audiometers. Our finding of a correlation between hearing loss and BMI at most of the tested frequencies was unexpected and of unlikely clinical significance. Previous studies have shown an association between high BMI and increased risk of hearing loss in adolescents[27] and adult women,[28] but not in adult men.[29]

Valid reasons for testing divers' hearing include determination of fitness for work (i.e., communication issues), tracking of hearing loss with the aim of prevention of further damage, and documentation of existing damage for possible future compensation claims. But whether the results of such tests are usually acted upon, and/or have a role in the prevention of further deterioration of hearing is debatable. Abnormal results mean that damage is already done or may imply a pre-existing condition. They could certainly point to modifiable causes, but *post hoc* rationalisation is an unsound basis on which to mandate formal routine audiological examinations. For example, while abnormal results do not imply an unsafe environment, normal results do not imply an audiologically safe working environment, that should ideally be provided, regardless of test results, by adherence to all practicable safety measures.

Our results concur with the majority of previous studies and suggest that, while professional divers are always at increased risk of hearing damage due to a specific traumatic incident, they are at no greater risk of hearing loss than the general public in the absence of such an incident. Of particular note, in the past fifteen years, since the introduction in New Zealand of five-yearly rather than annual full medical evaluations, not a single diver has been found, on routine audiological testing, to have a hearing condition that has resulted in any restriction on their certification. Employers, and divers themselves, are responsible for minimising exposure to excessive noise and other potential causes of hearing damage, such as barotrauma and DCS.

Consequently, we believe that a reasonable approach to surveillance of divers' health in this regard would be to perform formal audiological testing on entry to the industry, as a screening test and baseline, followed by further testing only if clinically indicated (for example, after a barotraumatic or inner ear DCS event), and then final testing on exit from the industry.

LIMITATIONS

Firstly, we did not have an objective measure of actual diving exposure, and our first audiometric recordings did not invariably represent the beginning of that exposure. The number of years of occupational diving between assessments, although a blunt measure, was used as a surrogate for diving exposure. In addition, as mentioned above, the number of years of occupational diving used in this study is not necessarily representative of an individual diver's complete diving career, as many divers had already been diving for several years before the earliest of our

usable audiological records. We have reported the change in hearing over periods of occupational diving ranging from 10 to 25 years. However, the initial recordings represent the divers' hearing at various points in their diving careers. So, we cannot exclude the possibility that our initial recordings may have been influenced by existing damage which could, in turn, influence later changes. Divers with an initial history of MEBt, hearing loss or tinnitus were not excluded from this study, because they were still considered to be fit to dive, and including them produced a more complete record of the real-world situation for working divers. For the multiple regression analysis, diver occupational groups were stratified into 'high intensity' and 'low intensity' groups on the basis that the high intensity group, consisting of construction, commercial, and military divers, was more likely to be exposed to deeper and more exertional diving with greater likelihood of noise pollution from in-helmet communications or equipment, than the low intensity group. Again, we acknowledge that this classification may be subject to inaccuracies.

Another limitation of this study is the possibility that a selection or attrition bias (healthy worker effect), based on divers leaving the industry because of hearing problems, might have influenced our findings. The only way to resolve this question would be to compare the audiograms of all divers on entry to, and exit from the industry, a topic for ongoing study. Preliminary results of a study into health reasons for diver attrition (pending[30]) demonstrate no evidence of hearing loss being a reason for quitting diving.

As with all such audits, data gathered over many years and from many sources are subject to the vagaries of variable equipment quality and the technical competence of operators. We were limited to using pure tone air conduction data when a more complete data set would have included bone conduction and speech discrimination data.

Finally, we used the latest ISO Standard data set as the normative data for comparison. An appropriate alternative may have been to use a matched group with similar occupational noise exposure to divers, such as firefighters, a consideration for future study.

Conclusions

Audiological changes over 10–25 years of professional diving were not found to be significantly different from the changes expected due to ageing. Development of policies for health and safety surveillance of occupational divers should be guided by the best available evidence of benefit when determining the frequency and type of screening examinations required. The results of this study suggest that routine annual audiological testing of occupational divers is not justifiable.

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Eustachian tube balloon dilation in treatment of equalization problems of freediving spearfishermen

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Key words
ENT; Ear barotrauma; Diving; Treatment; Outcome

Abstract
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Background: Eustachian tube balloon dilation is a minimally invasive surgical procedure used to treat Eustachian tube dysfunction which is not responsive to conventional therapies.
Methods: In this cohort intervention series we report the results of balloon dilation in treating refractory equalization problems in 20 freediving spearfishermen; in 19 cases the problem was unilateral and in one case bilateral. All the patients had already received medical and insufflation therapy and four patients had also had nasal surgery. None of these treatments or procedures had achieved improvement. We used a 20 mm x 3 mm disposable balloon catheter inserted through a 70-degree guide catheter and inflated up to 12 ATM three times at three different depths of insertion within the Eustachian tube. Every inflation lasted 120 seconds.
Results: Fifteen out of 20 patients improved. Ten patients reported a complete resolution of equalization problems, five showed improvement with persistence of a slight equalization delay on the treated side. Five patients did not report any improvement. Two complications occurred: subcutaneous emphysema of the parotid region in one case; and a mild high frequency (4–8 KHz) sensorineural hearing loss in another patient.
Conclusion: Balloon dilation of the Eustachian tube is an effective therapy in the treatment of equalization problems with a good success rate.

Introduction

The Eustachian tube (ET) is a complex structure connecting the middle ear to the nasopharynx. It is approximately 44 mm in length and includes a medial cartilaginous portion (two thirds) and a lateral bony part (one third).[1] Normally the tube is collapsed at rest and it opens during swallowing thanks to the paratubal muscles, primarily tensor veli palatini and secondarily levator veli palatini.[2,3] This involuntary opening of the tube happens 1.4 times each minute for about 0.4 s and allows the middle ear pressure to be balanced with the ambient pressure.[4] It is also possible to voluntarily open the ET to counterbalance acute changes in ambient pressure by swallowing or yawning, and to increase or decrease nasopharyngeal pressure creating an air flow through the tube by maneuvers like Valsalva, Toynbee, Marcante-Odaglia, sniffing, etc.

ET dysfunction is defined by the presence of pressure dysregulation in the middle ear, with chronic dysfunction diagnosed when this is present for at least three months.[5] If the dysfunction is mild, symptoms like aural fullness, popping or pain occur. In more severe cases, other middle ear diseases may develop (e.g., otitis media with effusion, chronic suppurative otitis media, retraction pocket on the tympanic membrane (TM) and cholesteatoma). ET dysfunction during substantial barometric change, commonly seen with flying or diving,[5] can lead to barotrauma, which is damage to the middle or inner ear structures caused by ineffective equalization of middle ear pressure. There are many different tests for assessing ET function but evidence for their use is weak. There have also been also many attempts to develop objective tests but no single test has been found to be a reliable diagnostic tool.[1]

Tympanometry is the simplest and most widely available test. It uses the application of pressure in the external ear canal and records a chart called a tympanogram that displays TM compliance. Most commonly observed tympanogram types are the following: type A, considered normal; type B, most commonly considered expression of middle ear effusion; and type C, considered a manifestation of negative pressure in the middle ear usually due to ET dysfunction. The value of this test to diagnose ET dysfunction is poor

Table 1
Equalization problems (EP) score

Description	EP score
Perfect equalization	0
Equalization possible and effective but slight difference between the ears (no middle ear barotrauma reported even after many dives and/or many days of diving)	1
Equalization possible but ineffective (middle ear barotrauma after few dives)	2
Equalization not possible	3

since middle ear pressure varies considerably over time and, therefore, one single measure is of limited relevance.[6] When tympanometry is used to measure compliance changes after manoeuvres like Valsalva or Toynbee or after sniffing, sensitivity for ET dysfunction is reported to be higher.[7,8] The nine-step inflation/deflation test was found to be the most efficient test with the highest predictive value as a predictor of middle ear barotrauma, especially in scuba divers.[9] Other tests, such as tubomanometry and sonotubometry, although interesting, did not demonstrate optimal sensitivity and specificity and imaging techniques are not recommended as part of a routine assessment of ET function.[1]

Patient questionnaires offer an attractive method to assess the symptoms that are seen in ET dysfunction. The ETDQ-7 was developed by assessing the differences between the answers to a set of questions between cases with ET dysfunction and controls.[10] A German translation of ETDQ-7 showed very high sensitivity and specificity that outperformed any of the objective tests described.[11]

Therapy for ET dysfunction is not completely standardized yet. Currently, no medical therapies have shown efficacy[12] even though the use of antibiotics and/or steroids as first-line treatment is very common. Despite the widespread use of ventilation tubes (grommets), there are no studies published evaluating their efficacy.[12] LASER tuboplasty and balloon dilation of the nasopharyngeal entrance of the ET are two surgical procedures that are becoming increasingly popular, with many published series reporting good results and a low complication rate.[13–17] A comparison between the two techniques concluded that both procedures can improve symptoms of ET dysfunction but could not clarify if there is one that provides greater benefits.[18]

In this paper, the results are reported of balloon dilation in treating equalization problems of 20 freediving spearfishermen. To our knowledge, this is the first study conducted on this particular patient population.

Methods

From October 2012 until May 2015, balloon catheter dilation of the cartilaginous portion of the ET was offered to 20 freediving spearfishermen who complained of equalization problems (EP) during freediving and had experienced multiple episodes of middle ear barotrauma. All the patients were amateurs who used to dive at least twice a week, all year long and were symptomatic for at least for one year, but did not show any symptoms in their everyday life. Exclusion criteria were age less than 18 and more than 65 years, evidence of other diseases that could explain the complaint and the impossibility of follow up of the patient and/or to collect a detailed history.

All 20 patients had been treated medically (oral and/or intravenous antibiotics and steroids) already and received insufflation therapy (Politzer, catheter insufflation, Otovent®) without improvement. Four patients had also received surgical treatment; inferior turbinate reduction in two cases, septoplasty and inferior turbinate reduction in one case and a combined procedure including septoplasty, inferior turbinate reduction and endoscopic sinus surgery in the fourth case. In all four, surgery did not provide symptom relief, whilst the patient who had the combined surgery experienced worsening of the symptoms, with fullness and the presence of a Type C or B tympanogram even in daily life.

In 19 cases, the problem was unilateral (left side in 11 cases and right in eight) and in one bilateral case. Patients' ET function was scored using a four-stage system (0 – normal equalization, to 4 – equalization not possible, Table 1) called the *EP score*. A diagnosis of middle ear barotrauma was only accepted when made by a physician.

Before the intervention, all the patients were evaluated with a detailed interview, a complete ear, nose and throat (ENT) examination using a microscope to assess the TM and a fiberoptic flexible endoscope to assess the nasal cavities, nasopharynx and ET orifices. All patients also had a hearing test, tympanometry, an acoustic reflexes test and a tympanometric assessment of ET function using the nine-step inflation/deflation test.

At this preoperative assessment, all but two patients had normal hearing, type A tympanogram on tympanometry, normal acoustic reflexes and no pathology on clinical examination. One patient had bilateral conductive hearing loss from otosclerosis. The patient who had undergone the combined surgery and came to our attention for left-sided equalization problems had slight left conductive hearing loss, a B tympanogram on the left side, left TM retraction and inferior turbinate asymmetry (left > right) from the previous surgery. This patient also had a high frequency sensorineural hearing loss on the right side from a previous (three years before) inner ear barotrauma. Interestingly he did not report equalization problems on that side except for that particular

Table 2
Results of clinical assessment of 20 patients. TM = tympanic membrane; R = right; L = left

Patient	Affected side	Clinical assessment	Hearing test	Tympanometry and acoustic reflex test	Eustachian tube function test	EP score on affected side
1	Right	Nothing to report	Normal hearing	Bilateral type A Normal reflexes	Right dysfunction	2
2	Left	Nothing to report	Normal hearing	Bilateral type A Normal reflexes	Left dysfunction	2
3	Bilateral	Nothing to report	Normal hearing	Bilateral type A tympanogram	Bilateral normal	3 (bilateral)
4	Right	Nothing to report	Normal hearing	Bilateral type A Normal reflexes	Bilateral normal	2
5	Right	Nothing to report	Normal hearing	Bilateral type A Normal reflexes	Bilateral normal	2
6	Left	Nothing to report	Normal hearing	Bilateral type A Normal reflexes	Bilateral normal	2
7	Right	Nothing to report	Normal hearing	Bilateral type A Normal reflexes	Bilateral normal	2
8	Left	Nothing to report	Bilateral conductive loss (otosclerosis)	Bilateral type A Absent reflexes	Left dysfunction	2
9	Left	Nothing to report	Normal hearing	Bilateral type A Absent reflexes	Left dysfunction	2
10	Left	TM retraction L>R Inferior turbinate asymmetry L>R	Mild L conductive hearing loss R high frequency sensorineural loss.	Type C/B on L Type A on R	Left dysfunction	3
11	Right	Nothing to report	Normal hearing	Bilateral type A	Right dysfunction	2
12	Right	Nothing to report	Normal hearing	Bilateral type A	Right dysfunction	2
13	Left	Nothing to report	Normal hearing	Bilateral type A	Left dysfunction	2
14	Left	Nothing to report	Normal hearing	Bilateral type A	Bilateral normal	2
15	Right	Nothing to report	Normal hearing	Bilateral type A	Right dysfunction	2
16	Left	Nothing to report	Normal hearing	Bilateral type A	Bilateral normal	2
17	Left	Nothing to report	Normal hearing	Bilateral type A	Bilateral normal	2
18	Right	Nothing to report	Normal hearing	Bilateral type A	Bilateral normal	3
19	Left	Nothing to report	Normal hearing	Bilateral type A	Left dysfunction	2
20	Left	Nothing to report	Normal hearing	Bilateral type A	Left dysfunction	2

Figure 1
The guide catheter is directed toward the nasopharyngeal Eustachian tube orifice under endoscopic control. **a.** Nasopharynx. **b.** Eustachian tube orifice. **c.** Guide catheter

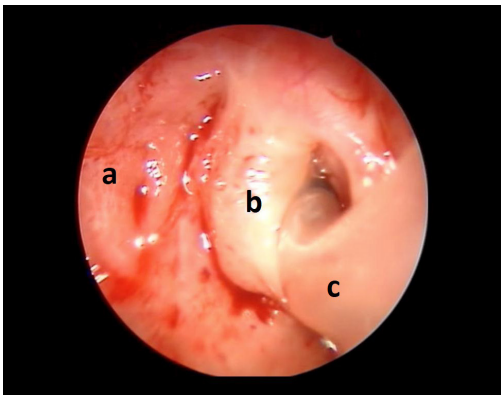


Figure 2
Balloon catheter inflated inside the Eustachian tube. **a.** Nasopharynx. **b.** Eustachian tube orifice. **c.** Guide catheter. **d.** Inflated balloon

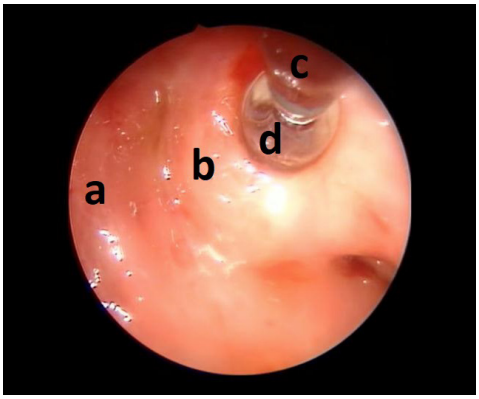
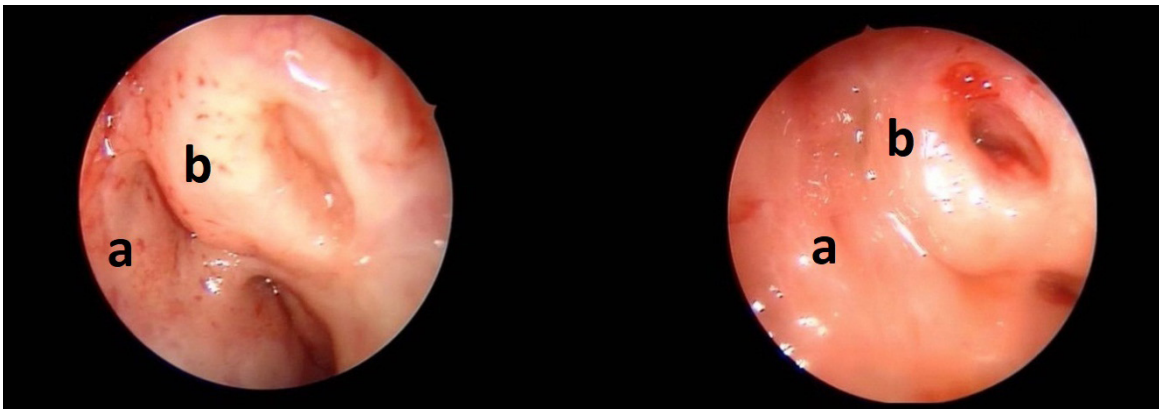


Figure 3
Eustachian tube appearance before (left) and after (right) the procedure. **a.** Nasopharynx. **b.** Eustachian tube orifice



episode. Tympanometric assessment of ET function revealed dysfunction on the affected side in 11 patients and normal function in nine (Table 2).

All patients were fully informed about the procedure and signed an informed consent approved by our hospital. We used a 20 mm x 3 mm disposable balloon catheter inserted through a 70-degree guide catheter (Spiggle & Theis, Overath, Germany). The procedures were carried out as follows:

- general anaesthesia induction;
- introduction of 4.0 mm diameter, 45 degree view endoscope in the contralateral nasal cavity and nasopharyngeal ET orifice visualization;
- guide catheter introduction in the ipsilateral nasal cavity;
- introduction of the balloon catheter in the ET (Figure 1);
- execution of three 12 ATM (balloon pressure) dilations at three different depths (balloon catheter completely inserted and no more visible at the orifice level; balloon catheter completely inserted but visible at the orifice level; balloon catheter partially inserted) of 120 seconds each (Figure 2);
- balloon catheter removal and final check (Figure 3).

Results

All the patients were discharged the day after surgery with an oral antibiotic and steroid therapy, mostly prednisone 25 mg daily and amoxicillin/clavulanic acid 875/125 mg twice daily for one week and re-examined after 7–10 days postoperatively. All were advised not to dive until after the postoperative examination but they were allowed to perform delicate Valsalva manoeuvres many times a day.

Eighteen patients had an uncomplicated postoperative recovery. One patient had subcutaneous emphysema of the parotid region on the first postoperative day following a Valsalva maneuver. The patient was temporarily forbidden from performing Valsalvas again and the emphysema resolved after four days without additional treatment. The patient who presented with a preoperative left B tympanogram and a right sensorineural hearing loss developed acute otitis media on the treated (left) side two

Table 3
Results of Eustachian tube balloon dilation in 20 freedivers

Case	Affected side	Pre-op EP score	Post-op EP score
1	Right	2	0
2	Left	2	0
3	Bilateral	3, 3	3, 3
4	Right	2	1
5	Right	2	1
6	Left	2	0
7	Right	2	2
8	Left	2	0
9	Left	2	2
10	Left	3	1
11	Right	2	0
12	Right	2	1
13	Left	2	0
14	Left	2	0
15	Right	2	1
16	Left	2	2
17	Left	2	0
18	Right	3	0
19	Left	2	2
20	Left	2	0

days after the procedure. This resolved within seven days with medical therapy but resulted in tinnitus and slight high frequencies (4–8 KHz) sensorineural hearing loss exactly the same as on the contralateral side. Neither prolonged intravenous and oral steroid therapy nor hyperbaric oxygen therapy were effective.

Fifteen out of 20 patients showed improvement or resolution of the equalization problems (Table 3). Ten patients reported complete resolution of the problem; nine patients went from an EP score of 2 to a score of zero and one from EP score 3 to a score of zero. Five patients reported an improvement with persistence of a slight equalization delay on the treated side, four going from EP 2 to 1 and one from 3 to 1 (the patient with preoperative B tympanogram and EP score 3 who suffered from postoperative acute otitis media). After the procedure he exhibited a type A tympanogram. Five patients did not report any improvement; four with a preoperative EP score of 2 and one preoperative score of 3 (Table 3).

After a follow-up period ranging from 19 to 50 months, the above reported results remained stable.

Discussion

In this paper the results are presented of ET balloon dilation in 20 freediving spearfishermen with equalization problems, of whom 18 had normal hearing and were completely asymptomatic in their everyday life. To our knowledge, this paper is the first to consider this particular population, since other authors have enrolled only patients with chronic symptoms and who were not subject to fast barometric pressure changes.

ET balloon dilation is a minimally invasive procedure used to treat ET dysfunction that is not responsive to conventional, non-invasive therapies. The procedure is carried out through the nasal cavities with endoscopic visualization of the nasopharyngeal ET orifice and introduction of a balloon catheter, which is inflated up to variable pressures of 5–12 ATM and left in place for a varing time; most surgeons performing each dilation for 30–120 seconds at 10–12 ATM balloon pressure.[19,20,21] A success rate of 70–87% and stable results up to three years have been reported.[17,20,22] The procedure is fast, easy and safe since the only complication, rarely reported, is subcutaneous emphysema that resolves spontaneously.[22,23] Extratubal structural damage or a patulous ET have not been reported in cadaveric or in clinical studies.[17,19,24,25]

No particular preoperative radiological examination is necessary, based on the evaluation of CT scan images of 1,000 patients (2,000 temporal bones).[26] No internal carotid artery anomalies or defects of the osseous portion of the ET were found.[26] However, a very detailed interview, clinical examination, hearing test, tympanometry and ET function tests are mandatory. Making a correct diagnosis of ET dysfunction is difficult because of the lack of a test with high levels of specificity and sensitivity.[1] For this reason diagnostic questionnaires have been developed with the ETDQ-7 being the most popular and reliable.[10,11] The ETDQ-7 was not a good tool for our study since it considers symptoms that are present in everyday life and not just during a specific activity like freediving. When administered to these 20 patients, it was inconclusive. Therefore, a new four-step scoring system was devised (the 'EP score') to categorize equalization difficulties during freediving.

Although LASER tuboplasty has shown good results, balloon dilation was chosen because of our familiarity with the routine use of balloons in endoscopic sinus surgery. Also, the formation of peritubal adhesions are sometimes reported after LASER tuboplasty,[13] but never after balloon dilation. Using a 20 x 3 mm balloon catheter inflated to 12 ATM to perform three consecutive dilations at different depths within the ET, 120 seconds for each dilation appears to be an adequate time to obtain a good dilation without risking mucosal ischaemia. The three dilations at different depths ensure the treatment of the cartilaginous portion of the ET for its entire length. The 75% success rate

(15 of 20 divers) is similar to that reported by other authors in treating patients with ET dysfunction and chronic symptoms.[20,22,25]

However, there were more complications in this series than was hoped for, with both a case of subcutaneous emphysema and one of acute otitis media which resulted in a high frequency sensorineural hearing loss and tinnitus on the treated side. In both cases the procedure was straightforward and no difficulties were noted during balloon insertion. The subcutaneous emphysema could be explained by a small unseen mucosal tear as already reported by other authors,[27] but it is not clear how the otitis media and the resulting hearing loss could occur. A barotrauma caused by a Valsalva manoeuvre performed by the patient in the postoperative period or by an inadvertent movement of the inflated balloon during the procedure could explain this. Indeed, this patient had a contralateral inner ear barotrauma three years before, which resulted in a sensorineural hearing loss identical to the one experienced on the treated side after the surgery. Why this patient developed inner ear damage is not clear since a CT scan performed after the surgery did not highlight any temporal bone anomaly. Also, although he suffered from multiple middle ear barotrauma on the affected side, there was never inner ear involvement. Even if a sensorineural hearing loss after balloon dilation is considered an exceptional event, it should always be kept in mind during the procedure and patients should be informed of this potential complication.

Conclusion

Although this study has some limitations, such as a relatively small number of patients and questionnaire-based results, balloon dilation of the Eustachian tube appears to be an effective therapy in the treatment of persistent equalization problems in divers. However, it is an invasive procedure that may have complications. This should be considered especially when treating patients who are, in the majority of cases, symptom-free in their everyday life. Therefore, ET balloon dilation should be reserved for patients who have failed to respond to alternative non-invasive therapies. It should be performed by experienced surgeons in the most delicate and atraumatic way.

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The effects of pressure *in vitro* on three methods of root canal obturation

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Key words
Barotrauma; Dental; Diving; Endodontics

Abstract
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Introduction: The seal of root canal fillings depends on the anatomy of the root canal system and on the properties of filling materials and their bond to the walls of the root canal. Alterations in pressure may influence these properties. The current study investigated and compared microleakage in a tapered single-cone method versus lateral and vertical condensation after diving simulation.
Methods: One hundred and thirty five extracted single-rooted teeth were used. Following instrumentation and irrigation to #30.06 Mtwo rotary system, obturations were performed in three groups of 45 teeth: Group 1, tapered single-cone with Endoseal MTA sealer; Group 2, lateral condensation with AH26 sealer; Group 3, vertical condensation with AH26 sealer. Then all specimens were divided into three groups and incubated at ambient room pressure (101.3 kPa), 203 kPa or 304 kPa for 120 minutes respectively 20 times over one month to simulate diving conditions. Microleakage quantitative analysis was recorded by using a 2% Methylene blue dye for 24 hours.
Results: The amounts of microleakage increased with increasing pressure in all obturation groups; however, the differences were not statistically significant ($P > 0.05$). At all three pressures, the least microleakage was recorded in Group 3, vertical condensation. Although the differences between vertical condensation, lateral condensation, and tapered single-cone methods were statistically significant ($P < 0.001$), the vertical condensation and lateral condensation groups did not differ from each other ($P > 0.05$).
Conclusions: Vertical condensation may be the best technique, based on sealing ability, for people who frequently experience pressure alterations.

Introduction

Appropriate nonsurgical endodontic treatment is associated with knowledge of root canal morphology, cleaning and shaping, and finally proper obturation using appropriate sealant, filling materials and obturation techniques.[1–4] In addition, with the growing number of scuba divers and aircrew members, dentists will increasingly encounter oral conditions relating to pressure changes and these require careful attention.[5–7] These phenomena are mainly related to Boyle's Law which states that at a constant temperature, the volume and pressure of an ideal gas are inversely proportional. Amongst these oral conditions, barodontalgia is the term used to describe toothache related to ambient pressure changes.[8–10] Odontocrexis is another term used to describe tooth or restoration structure destruction associated with pressure changes.[11] Dental barotrauma is a more general term describing the damage to the tooth structure resulting from pressure changes with or without pain. Dental barotrauma is a potential cause of incapacitation that could jeopardize the safety of scuba diving or flight.[12]

Pressure changes may lead to structural alterations and microleakage in applied dental materials.[13,14] There are a few studies that address other physical impacts of pressures in pulp chamber and root canals.[6,13] Previous studies reported trapped gas bubbles in tooth root canal and chamber after the completion of root canal treatment and also the existence of microleakage between the restoration and the walls of the pulp chamber.[15,16] Hence, changes in pressure may influence the strength and physical properties of dental materials. This could influence recommendations about when patients should not dive.

The current study investigated the effect of repeated pressure exposure on microleakage in three different obturation methods after simulation *in vitro* of typical recreational diving pressures.

Materials and methods

The teeth used were extracted from patients for orthodontic reasons after written informed consents were obtained. The study was approved by the Ethics Committee of AJA University of Medical Sciences, Tehran, Iran.

SAMPLE PREPARATION

Specimens used were 135 sound, single-rooted premolars, with less than 20 degrees root curvature, extracted over a six-month period and stored in chloramine-T 1% (Sigma-Aldrich, Nst. Louis, Mo, United States) at room temperature. All teeth were evaluated by a qualified endodontist via radiography to determine that the root canals and teeth were suitable for inclusion in the study. Collected teeth were randomly divided into three groups, 45 teeth in each group. Twenty-eight other teeth were excluded because of ribbon-shaped canal anatomy.

ENDODONTIC TREATMENT

A single endodontist (PS) with special training in the respective filling techniques performed all root canal preparations and obturations in a standardized manner in order to minimize procedural variations. In all groups, access cavity preparation was done by D+Z diamond burs 837 (D+Z, KalletalT, Diamant GmbH, Germany) and working length was determined by inserting a size 15 K-file (Dentsply Maillefer) into the root canal until it was visible at the apical foramen and subtracting 1 mm from that length. The preparation of root canals was executed with the Mtwo rotary system, starting with #15.05 and continued to #30.06 (VDW, Munich, Germany). Irrigation with 5 ml 2.5 % sodium hypochlorite was done between successive instruments. At the end of preparation, the canal was irrigated with distilled water. After drying the canal with sterile paper points, specimens were obturated by the following techniques;

Group 1: Teeth were obturated using the tapered single cone method. Sealing cement Endoseal (MTA Endoseal, Maruchi, South Korea) was inserted into the canal with its syringe. Then, the master cone (#30.06 tapering) was impregnated with sealing cement and positioned to the working length.

Group 2: Teeth were obturated using a cold lateral condensation technique and sealing cement AH 26 (AH 26, Dentsply Maillefer, Ballaigues, Switzerland) was prepared and inserted into the canal with a lentulo. Then, the master cone (#30.02 tapering) was impregnated with sealing cement and positioned to the working length. Accessory gutta-percha cones with spreader #25 were applied in this group .[17]

Group 3: Teeth were obturated with a vertical condensation method and sealing cement AH26. In this group, gutta-percha and pluger were utilized for obturation.[18]

Finally, all obturated teeth were radiographically evaluated by another, blinded endodontist (BF) and the specimens in each of the three groups were randomly divided into three subgroups for the pressure simulations (n = 15 in each subgroup). For setting of the root canal sealers, the access cavity was sealed with temporary restoration material (Cavisol, Golchai, Karaj, Iran) and the teeth were stored for 24 hours (h) at 37°C and humidity of 90%. Prior to pressure simulation, all the specimens were sectioned mesiodistally at the cementoenamel junction with a diamond disc (D+Z, KalletalT, Diamant GmbH, Germany).

PRESSURE SIMULATION

To simulate pressure changes typical of shallow water diving, a custom-made chamber with an external pressure gauge was used. The chamber was pressurised with air, and an air vacuum pump was used to decrease pressure. In each of the three groups, one subgroup was exposed 20 times over one month to a pressure of 203 kPa for 120 min; a second subgroup to 304 kPa and the third acted as a control group at ambient room pressure. In the control group, specimens were incubated in 90% humidity, 37°C for one month. The pressurised subgroups were kept in the same ambient conditions as the control group between compressions.

MICROLEAKAGE TEST

Except for 2 mm in the coronal part, the entire tooth surfaces were covered with two layers of nail polish. The root apices were sealed with sticky wax. The specimens were immersed in 2% methylene blue for 24 h then rinsed under running water to remove excessive dye. The teeth were subsequently sectioned buccolingually with a diamond disc (D+Z, KalletalT, Diamant GmbH, Germany). Two sections of each specimen were examined under the stereomicroscope at 16X magnification. In order to quantitatively assess microleakage, dye penetration was measured (millimetre) from orifice to apical constriction via stereomicroscopy (SZX9, Olympus, Japan) and OLYSIA Zoom soft imaging system GmbH software by an experienced oral and maxillofacial radiologist (BF) blinded to the pressure grouping.

STATISTICAL ANALYSIS

Sample size was based on a similar study,[19] due to the nature of the study in which rigorous inclusion criteria were required to make specimens as similar to each other as possible. Data analysis was carried out using SPSS software (SPSS version 18.0, SPSS, Chicago, IL, USA). A Kolmogorov-Smirnov test determined that the data were not distributed normally. Therefore, a Kruskal-Wallis H test was used as a nonparametric analysis to evaluate the statistical significance of all results and pair-wise comparisons ($P < 0.05$).

Table 1
Dye penetration (mm) after repeated exposure to three different pressures; mean (median, interquartile range); see text below for statistical differences between methods

Obturation method	Mean of dye penetration (mm)		
	Ambient room pressure	203 kPa	304 kPa
Single cone	8.0 (8.0, 6.5–9.2)	8.0 (8.0, 6.7–9.1)	8.2 (8.1, 7.9–9.2)
Lateral condensation	4.5 (4.0, 2.9–5.0)	4.8 (4.4, 3.2–5.2)	4.8 (4.7, 3.1–5.1)
Vertical condensation	3.9 (2.8, 1.9–3.8)	4.1 (3.6, 2.6–4.0)	4.1 (3.6, 2.8–4.1)

Results

AMBIENT ROOM PRESSURE

Stereomicroscopy photographs showed the least dye penetration with the vertical condensation method at all three pressures (Group 3). Mean of dye penetration for each group at each pressure are shown in Table 1. The difference between lateral and vertical condensation techniques was not statistically significant ($P = 1$). However, vertical and lateral condensations methods had significantly less dye penetration in comparison with the tapered single-cone method ($P = 0.001$).

203 kPa PRESSURE

Stereomicroscopy again showed the least dye penetration in the vertical condensation group. The difference between lateral and vertical condensation techniques was not statistically significant ($P = 1$). However, vertical and lateral condensations methods had significantly less dye penetration in comparison with tapered single-cone method ($P = 0.001$ and 0.01 respectively) (Table 1).

304 kPa PRESSURE

Stereomicroscopy showed the least dye penetration in vertical condensation group (Figure 1). The difference between lateral and vertical condensation techniques was not statistically significant ($P = 1$). However, both of them had significantly less dye penetration in comparison with tapered single-cone method ($P = 0.01$) (Table 1).

Discussion

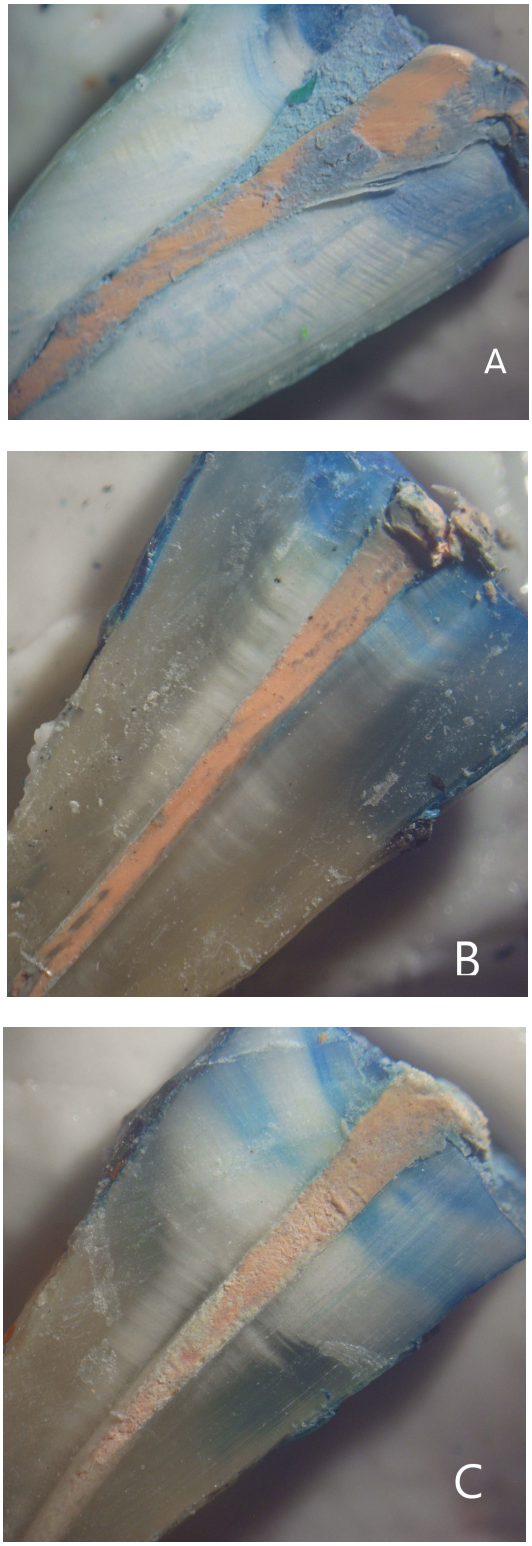
Simulated root canals in plastic blocks or extracted natural teeth have proved to be useful in systematically examining root fillings *in vitro*. In this context, natural teeth are superior to plastic blocks since they better reflect the dentin surface and the resulting mechanical properties at the interface between dentin and root filling material. Depending on the configuration of the root canal, natural teeth differ widely in the anatomy of the root canal system. In addition, root curvature plays an especially important role in the preparation and obturation of the root canal system. Therefore, in this study, only single-root extracted teeth with a root canal curvature of less than 20 degrees were used in order to facilitate comparisons.

Microorganisms present inside root canals may remain active in the dentinal tubules even after vigorous chemico-mechanical preparation. Thus, perfect apical sealing is desirable to prevent bacteria and their endotoxins from reaching the root apex. Coronal leakage is considered to be the common cause for endodontic failure and is influenced by many variables such as different filling techniques, chemical and physical properties of root canal filling materials and the presence or absence of a 'smear' layer. There are several assessment methods for microleakage such as dye penetration, dye extraction or dissolution, bacteria and toxin infiltration, air pressure, scanning electron microscopy, transmission electron microscopy and micro-computed tomography.[20] In the present study, methylene blue penetration was used as it is relatively inexpensive, safe and results in high penetration because the dye is a small molecule.[21]

In the current study, AH-26 and Endoseal MTA were used as sealers. For a sealer to penetrate the narrow root canal, its flowability should be good. The flowability of Endoseal MTA which is prepared from a powder, is not significantly different from the paste type AH-26.[22] The penetration of the sealer within dentinal tubules improves the sealing ability by increasing the contact surface between the filling material and dentin.[23] Penetration of Endoseal MTA to dentinal tubules is significantly lower than AH-26.[24] On the other hand, MTA has been shown to be an effective root apex filler and Endoseal MTA was better than other MTA products for root canal filling.[24,25]

A variety of systems are available for obturating root canals.[26] Gutta-percha is the most widely used filling material. It is

Figure 1
Stereomicroscopy of three sectioned teeth after repeated exposure to 304 kPa pressure (magnification x16); A) tapered single-cone method, B) lateral method and C) vertical condensation method; microleakage of methylene blue is obvious between gutta-percha and canal walls in tapered single-cone method but in other methods microleakage mostly occurs through dentinal tubules in the coronal region not covered by nail polish



suitable for both cold and warm obturation techniques. In the literature, studies comparing cold and warm methods of obturation in terms of leakage under standard conditions are not uniform. Cold lateral condensation is one of the most widely used obturation techniques. Compared with thermoplastic obturation techniques, cold lateral condensation is reported to have possible disadvantages such as inhomogeneity, an increased risk of canal fracture and poor adaptation to the canal walls.[27]

Regarding the microleakage test, this study showed statistically significant differences between the tapered single-cone method and other methods. Possible explanations are a potentially increased proportion of sealer in the root filling or a poor marginal seal under higher pressure levels. Contradictory results reported by different studies may be because of the variations in leakage evaluation techniques, test conditions, cavity design and dimensions, type of teeth and observation time. These contrasting results underline the obvious importance of standardizing testing parameters of leakage studies.[28]

Regarding the effects of pressure, no significant differences were seen between the control and pressure-cycled groups. Although after repeated pressure exposure there was a tendency towards increasing microleakage, these differences were not statistically significant. Although the pressure changes had no effect on the root canal filling irrespective of the method used, the use of vertical condensation appeared to have superior sealing ability compared to lateral condensation and the tapered single-cone method and, therefore, may be the most suitable method for use in divers and aviators. According to our literature review, this study is the first evaluation of various environmental pressures on microleakage after endodontic treatment.

Conclusion

Within the limitations of this study based on the sealing ability of three assessed obturation methods, vertical condensation would seem to be the most suitable technique for use in divers and aviators. Typical diving pressure cycles demonstrated no significant adverse effects on sealing in endodontically treated teeth. However, further investigation is required applying new assessment methods and more highly sensitive detectors.

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A 20-year analysis of compressed gas diving-related deaths in Tasmania, Australia

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Key words
Diving deaths; Diving incidents; Incidents; Risk management; Root-cause analysis; Safety; Case reports

Abstract
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Introduction: This study reviews diving deaths that occurred in Tasmanian waters over a 20-year period.
Methods: Detailed analysis was undertaken of deaths that occurred from 01 January 1995 to 31 December 2014. The cases were collated from numerous sources. Utilising a chain of events analysis, factors were identified and assigned to predisposing factors, triggers, disabling agents, disabling injuries and cause of death. These were then scrutinised to ascertain regional variables, remediable factors and linkages which may benefit from targeted risk mitigation strategies.
Results: Seventeen deaths were identified across this 20-year period, which included one additional case not previously recorded. All were recreational divers and 15 were male. Five were hookah divers, 12 were scuba divers. Important predisposing factors identified included equipment (condition and maintenance), pre-existing health conditions, diver experience and training. These factors can now be used to promote public health messages for divers.
Conclusions: This 20-year study highlighted regional variations for Tasmanian deaths and presents opportunities for strategies to prevent diving deaths in the future. Of particular concern was the diving practice of 'hookah' diving, which has no governing regulations. The study highlighted the importance of applying a structured methodology such as chain of events analysis to scrutinise diving deaths.

Introduction

Diving-related fatalities in Australian waters have been collated and reported since the early 1970s, initially as *Project Stickybeak*,[1] and then continued by the Divers Alert Network Asia-Pacific (DAN AP) dive fatality reporting project, with the aim of identifying the causes of fatal dive incidents, and to reduce their incidence.[2,3] A series of reports identified the critical incidents and causative factors in the overall Australian experience but there have been no studies that accounted for regional variables.

It is likely that Tasmania has regional idiosyncrasies compared to the rest of Australia. For instance, a large percentage of Tasmania's recreational divers use 'hookah' surface-supply breathing equipment. Exact numbers of hookah divers are unknown but they account for up to 30% of divers annually requiring treatment for decompression illness (DCI) at the Royal Hobart Hospital (RHH).[4] It was thought possible that hookah divers may be over-represented in Tasmanian diving accidents and fatalities. Water temperature in Tasmanian waters is cooler than the rest of Australia.[5] Diving in cooler water requires greater thermal protection, the associated need to carry more weights, and the potential of increased exertion. These can create an increased risk of a thermal- or buoyancy- or cardiac-related injury, or immersion pulmonary oedema (IPE).[6]

In December 2015, Tasmania's population was 517,400 and, for its size, it has a relatively large recreational diving population.[7] In 2014–2015, there were 8,742 recreational rock lobster diving licences and 12,083 recreational abalone licences issued.[8] This implies a participation rate of 2.3% to 4.0% of the population undertaking underwater recreational fishing without including commercial divers or recreational divers who do not hold fishing licences.

Over the years 1995 to 2009 there were 154 deaths recorded by DAN AP in compressed gas divers in Australia (0.52 per million population per year).[9] Tasmania had 12 deaths in the same period, with a death rate of 1.68 per million population per year, indicating a potential regional anomaly.

Table 1
Key words/search phrases for electronic identification of Tasmanian diving death coronial reports

Period : 01 January 1995 to 31 December 2014
Diving
Tasmania
Scuba
Hookah
Fatality
Surface-supply breathing apparatus
Compressed gas
Cerebral arterial gas embolism (CAGE)
Decompression illness
Decompression sickness
Death
Mortality

There is merit in investigating diving deaths in a structured way, for instance, applying root cause analysis (RCA) to diving accidents to trace their aetiology.[10] This process has been refined further to include predisposing factors via a five-step analysis and structured template.[11] This could be applied to Tasmanian diving deaths to identify predisposing and causative factors that may assist future preventative strategies. Therefore, the aims of this study were:

- to identify all compressed gas diving deaths in Tasmania over a 20-year period and the events preceding each death;
- to identify and evaluate Tasmanian diving practices and risks, using the five-step, sequential chain of events analysis;
- to generate public health messages to inform divers and to assist future prevention strategies.

Methods

STUDY DESIGN

A retrospective consecutive case series analysed all diving-related deaths associated with the use of compressed gas in Tasmania between 01 January 1995 and 31 December 2014. The study was approved by The Tasmanian Health and Medical Human Research Ethics Committee (Ref No. H0014793).

IDENTIFICATION AND SELECTION OF SUBJECTS

For inclusion in the study, the deceased diver must have breathed compressed gas in the aquatic environment. There was no central database in Tasmania which had sufficient detail to identify all diving deaths, hence a multimodal approach was used to capture all Tasmanian diving deaths. Previous information from DAN AP suggested that the total study population would be no more than 20 subjects. Searches were undertaken from the following sources:

- coronial records;
- interviews with the RHH Hyperbaric staff and the Tasmanian State Director of Forensic Pathology;
- hyperbaric facility records from 1995;
- DAN AP records (including *Project Stickybeak*);
- newspaper reports from Tasmanian major newspapers;
- hand search of the *SPUMS Journal* and *Diving and Hyperbaric Medicine*.

Reports were searched by using key words/search phrases as detailed in Table 1.

STUDY VARIABLES AND MEASUREMENTS

The basic data sought for each of the deaths included age, sex, year of death, equipment used, type of diving (recreational or professional), level of training, experience, geographical location of incident dive, previous medical history including dive medical, incident dive trend and cause of death. All available records were examined. These included: Coroners' reports; autopsy and toxicology reports; police and witness statements, equipment reports and gas analyses and medical records.

Each diving death was evaluated using chain of events analysis (CEA).[11] The analysis sequentially identified predisposing factors, triggers, the disabling agents, disabling injury(ies) and cause of death.[11] Each death was independently analysed by each author. Working sequentially was intended to minimise bias and reduce variability when assessing the divers' likely actions prior to their death. The findings were then cross-referenced. Each step of the original diving root cause analysis categories were divided into a number of broad sub-categories, permitting a structured approach.[10,11] The additional fifth category, 'predisposing factors' was also divided into a number of subcategories Table 2.[11]

Trends in causation and, in particular, identifiable factors that may be preventable and remediable were looked for. These factors were used to guide recommendations for preventive strategies and public health campaigns. Using this study methodology, it was possible that a cause of death other than that stated by the Coroner might be identified. In this event, such cases were highlighted and discussed.

STATISTICS

Although the data were categorical in nature, the expected small numbers and potentially diverse causative factors were likely to allow only basic statistical analyses which included incidence, mean, standard deviation, medians and interquartile ranges, ratios and percentages. All data were

Table 2
Predisposing factors contributing to the diving accident[11]

Predisposing Factor	Description
Health-related factors	Contributing to the dive accident (e.g., cardiovascular disease, epilepsy, diabetes, mental or physical fatigue)
Organisational/Training/Experience/Skills-related	Diver's practical readiness to dive (e.g., suitability of training course design and conduct, the overall dive organisation by a dive operator, the level of skill and/or experience of the diver)
Planning-related	Decisions made prior to the dive
Poor communication or co-ordination	Between dive team members and/or operator
Absence of appropriate equipment; using obviously faulty equipment	Specifically contributed to the accident
Activity related risks	Higher risk in-water activity
Unsafe supervision	Lack of or inadequate supervision
Other	Other factors not in this list
Unknown or none	No cause identified; insufficient information

tabulated, and basic statistical calculations undertaken using Microsoft® Excel. Normality of data was assessed using the D'Agostino and Pearson omnibus normality test with GraphPad Prism 6 software (GraphPad Software inc. version 6.0e 2014, La Jolla CA). Death rate was expressed as a rate per million population. Populations were calculated as an average of the five yearly Australian Bureau of Statistics December census edition data (1996–2011).[7]

Results

Over the 20-year study period, 17 deaths were identified associated with compressed gas diving in Tasmania were identified. Age data were statistically not inconsistent with a normal distribution but, because of small study numbers, medians and interquartile ranges are documented. Fifteen divers were male, and two female; median age (all divers) 40 years (range 29–66, interquartile range 34–50). Most deaths (11 of 17) occurred during summer (all in South-East Tasmania), three in Autumn, two in Winter and one in Spring. All victims were recreational divers. Of the 17 deaths, five of the victims used hookah and 12 scuba equipment.

The Tasmanian death rate was calculated as 1.77 deaths per million population per year.[7] One of the deaths identified had not previously been recorded by Project Stickybeak or DAN AP. For two of the diving deaths more comprehensive documentation (not previously held by DAN, or Project Stickybeak), was identified that allowed full CEA. The locations of where deaths occurred shows a high occurrence rate within 100 km radius of Hobart (Figure 1). All were in the marine environment.

PREDISPOSING FACTORS (PF)

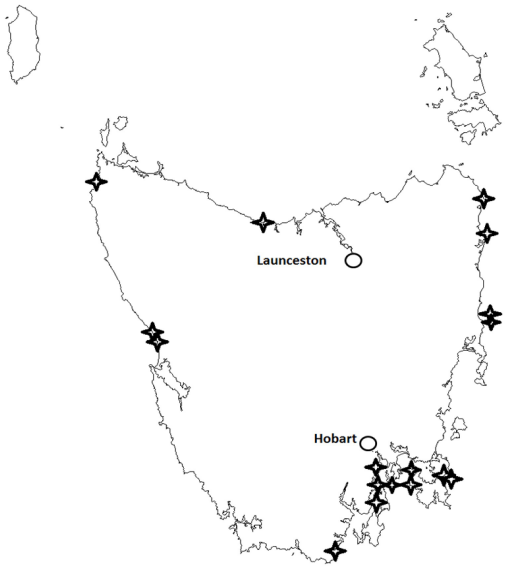
Tables 2 and 3 summarise 41 predisposing factors identified in the 17 divers. It was established that 15 of the 17 divers had two or more predisposing factors. In fact, one diver (CG-16) had five predisposing factors identified. Two divers had more than one equipment issue. Table 4 summarises the equipment factors which contributed to deaths.

Health conditions affected eight divers whose age ranged from 29 to 58 years, with a median of 46 years. Three of the divers had a recent significant health issue whilst two had a possible medical PF identified at autopsy. These included severe cramps for one diver, an aborted dive due to ear clearing problems and a previous likely severe episode of IPE. The other five cases all had significant cardiac disease and one also had concomitant respiratory disease. There was no evidence that any of the victims had undergone a recent dive medical.

Organisational/training/experience/skills-related factors predisposed to the deaths of eight victims while inexperience contributed to four. Two victims had returned to diving after a prolonged absence. The remaining two victims were inexperienced in the type of diving being undertaken (one being a night dive and the other in cold water).

Dive planning problems potentially contributed to seven fatalities. Four divers had planning problems affecting a safe buddy system. They either chose not to dive with a buddy or had formed a group of three divers. A further three dived in recognised poor conditions or were without adequate surface

Figure 1
Location of Tasmanian diving deaths between 1995 and 2015



rescue. The final three PF groups included the type of diving activity, i.e., poor visibility, kelp diving, strong currents ($n = 4$), poor communication ($n = 3$), and lack of supervision ($n = 3$). Poor communication led to poor diving plans that ultimately led to the divers' demise. Unsafe supervision led to delays in identifying problems, or difficulty with rescue, once in danger.

TRIGGER FACTORS (TF)

Table 3 summarises 26 TFs in 17 divers. One diver (Case 16) had three potential TFs, but it was unclear which was the most important. Five of the six equipment-related triggers involved the use of faulty hookah equipment. The sixth fatality was using incorrectly assembled scuba equipment. All hookah deaths involved equipment failure or inappropriate setup. The use of a Y-connector in the air hose distal to the hookah pump hose outlet resulted in air being diverted from one diver when the second one on the same system ascended. There was contamination of the air supply due to an incorrect inlet/ exhaust set up, whilst a T-piece dislodgement resulted in loss of the air supply. None of the hookah divers had an accessory air supply. Primary diver error involved either running out of air or losing a buddy. The buoyancy trigger involved inexperience with the use of a particular BCD. Two divers ran out of air whilst at depth, whilst in two cases it was not possible to clearly identify a trigger.

DISABLING AGENTS (DA)

Twenty-three DAs were identified (Table 3). Twelve divers had a single DA; four divers had two DAs and three DAs were identified in one case (Case 02). Problems with ascent were identified as the DA in five cases. The gas supply issues were either carbon monoxide contamination or loss of air. The buoyancy problems were attributable to difficulty with weights in three and problems with the BCD in one. Medical DAs were IPE in two and one with ear clearing difficulties. Environmental DAs were due to impact with rocks with subsequent head injuries.

DISABLING INJURIES (DI)

Twenty DIs were identified in 17 divers (Table 3). One victim (Case 02) had evidence of both asphyxia and cerebral arterial gas embolism (CAGE), creating a degree of uncertainty. Two cases of head trauma also had asphyxia – these divers (Case 09 and Case 14) were assigned two DIs.

CAUSE OF DEATH

The causes of death shown in Table 3 were those given by the Coroner. For 14 cases, the results of the CEA agreed with the Coroner's conclusions (10 drowning and 4 CAGE). However, after review in this study, the causes of death for three divers (Case 02, Case 06, Case 15) were considered to be different to the Coroner's determination.

Discussion

EPIDEMIOLOGY OF TASMANIA'S DIVING DEATHS

Tasmania had nearly four times the national average for diving deaths in the study period (1995–2009 Australian deaths = 0.46 per million population per annum). Although 'snapshot' surveys are available which estimate diving participation rates, there are no reliable national longitudinal participation data.[12] Hence, it is not possible to determine if Tasmania's higher death rate is due to higher participation or greater risk for the divers. The geographical and seasonal distribution of death mainly occurring in South-East Tasmania in summer is likely attributable to proximity to Hobart (highest population centre), warmer summer diving conditions, accessible sheltered waterways and coinciding with the open rock-lobster season.

The median age of divers (40 years), was similar to national data;[2,13–15] however, of the most recent six Tasmanian deaths, five were aged over 45 years (median 52 years, range 40–66). This may indicate participation risk from older divers who require greater medical input and surveillance. SPUMS recommends that regular medical checks for divers should commence at age 45, even in the absence of known health issues.[16] Of all Tasmanian deaths, only 12% were female, compared to 23% nationally.[2,13–15] Without knowing female participation rates, it is not possible to draw conclusions about prevalence by sex.

In previous studies, it was identified that lack of regional data was a limitation.[2] This series identified possible regional

Table 3

Predisposing factors identified in 17 diving deaths; trigger factors identified in 17 diving deaths (n = 26); disabling agents detected in 17 diving deaths (n = 23); disabling injuries identified in 17 diving deaths (n = 20); CAGE – cerebral arterial gas embolism; IPE – immersion pulmonary oedema

Case	Predisposing factor	Trigger factor	Disabling agent	Disabling injuries	Cause of death
01	Health/Planning	Environment/Exertion	Unknown	Asphyxia	Drowning
02	Health	Primary diver error/ Unknown	Ascent/Medical/ Buoyancy	Asphyxia/CAGE	Unclear–potential for drowning/CAGE
03	Poor communication or co-ordination/Absence of appropriate equipment/Obviously faulty equipment	Equipment	Ascent	CAGE	CAGE
04	Organisational/Training/Experience/Skills	Equipment	Gas supply	Other	Drowning
05	Organisational/Training/Experience/Skills	Equipment	Gas supply	Other	Drowning
06	Health	Environment/Exertion	Medical	Other Medical	Drowning/IPE
07	Health/Organisational/Training/Experience/Skills/Absence of appropriate equipment/Obviously faulty equipment	Environment/ Equipment	Unknown	Asphyxia	Drowning
08	Organisational/Training/Experience/Skills/Planning/Absence of appropriate equipment/Obviously faulty equipment	Primary diver error	Gas-supply/Ascent	CAGE	CAGE
09	Plannin/Poor communication or Co-ordination/Activity	Environment	Environment	Asphyxia/Trauma	Drowning
10	Organisational/Training/Experience/Skills/Unsafe supervision	Gas-supply/Primary diver error	Ascent	CAGE	CAGE
11	Organisational/Training/Experience/Skills/Planning/Absence of appropriate equipment or obviously faulty equipment/Activity	Environment/ Equipment	Buoyancy/Equipment	Asphyxia	Drowning
12	Planning/Absence of appropriate equipment; or obviously faulty equipment/Activity/Unsafe supervision	Environment	Gas supply/Equipment	Asphyxia	Drowning
13	Health/Absence of appropriate equipment or obviously faulty equipment/Activity	Equipment/Gas-supply	Ascent/Buoyancy	CAGE	CAGE
14	Health/Planning	Environment	Environment	Asphyxia/Trauma	Drowning
15	Organisational/Training/Experience/Skills	Environment	Medical	Cardiac	Drowning/IPE
16	Organisational/Training/Experience/Skills/Planning/Poor communication or co-ordination/Absence of appropriate equipment or obviously faulty equipment/Unsafe supervision	Buoyancy/Anxiety/ Stress/Primary diver error	Buoyancy	Asphyxia	Drowning
17	Health/Absence of appropriate equipment or obviously faulty equipment	Unknown	Unknown	Asphyxia	Drowning

Table 4
Equipment predisposing factors; hookah equipment trigger factors

Hookah	Other faults
Homemade hookah	No accessory safety air source
Previously compressor had other function	Incomplete assembly pre-dive – attributed to both hookah and scuba
Fuel leak, no inlet filter	Faulty or no buoyancy control device
Absent non-return valve in breathing line	Over-weighted
Made of non-stainless steel (significant corrosion)	Tight wetsuit
No suitable snorkel or intake extension	Not serviced

risk from diving in the colder waters around Tasmania. Two divers were from overseas and had not dived in cold water before. Feelings of claustrophobia and buoyancy control problems can result from wetsuit compression, exacerbated if a wetsuit is tight and/or thicker. This appeared relevant in one of the deaths. In addition, two victims were likely to have suffered from IPE, known to be exacerbated by cold water immersion.[6]

OCCUPATIONAL VERSUS RECREATIONAL DIVER DEATHS

Despite Tasmania's large population of active professional divers, there were no occupational diving deaths, which is surprising given that professional divers made up 41% of divers treated for decompression illness in Tasmania in 2010.[4] Australia-wide data between 2008 and 2011[2,13–15] revealed that commercial diving accounted for three deaths (out of 39 compressed gas diving deaths nationally) over this four-year period.[2,13–15] Occupational divers are subject to Federal Work Health and Safety (WHS) Legislation and Australian/New Zealand Standards. These govern diver operations and supervision, equipment maintenance and mandate yearly medical health risk assessment. Occupational divers are required by law to have training appropriate to the activity they are undertaking, to perform risk assessments, and work in teams that provide appropriate supervision. These are appropriate 'upstream' risk mitigation strategies to maintain diver safety. Tasmania appears to have a good safety record with professional diving, if absence of deaths is used as a measure of safety.

It must be acknowledged that better systems need to be implemented to improve Tasmania's recreational diving safety record. Hookah divers were over-represented in Tasmanian diving deaths (over a quarter of all deaths). Australia-wide data shows hookah divers constituted 10.3% of all compressed gas diving between 2008 and 2011.[2,13,14,15] Precise data on participation rates (hookah vs. scuba) are not available, but warrant further research.

CHAIN OF EVENTS ANALYSIS

Problems with equipment contributed to 10 out of 17 deaths. Faulty equipment was the major predisposing factor contributing to diving deaths. This had downstream effects whereby a PF identified with equipment became a trigger for equipment problems in six out of 17 deaths. For all hookah diving deaths, the compressors were in disrepair and/or had inappropriate or hazardous configurations which were apparent before divers entered the water. Key issues for hookah equipment were use of home-made apparatus, hazardous air intake and air-hose setups, disconnections, use of y-connectors in diver air hoses, absence of an accessory air supply and excessive weighting of divers. Hookah divers also dived without buoyancy compensators, preventing stabilisation at the surface in an emergency.

In Tasmania, recreational hookah equipment can be purchased and operated without any training and without regulation. The responsibility for maintenance rests solely with the owner, hence the capability of producing a home-made apparatus. The deaths in this report demonstrate a need for regulation around the use and maintenance of hookah apparatus, the training of divers who use hookah and the mandating of accessory air supplies. The authors have been unable to identify any training systems or courses for recreational hookah divers in Australia. This contrasts with widespread availability of scuba courses.

Recreational scuba training, equipment maintenance and scuba cylinder filling are usually undertaken by dive shops, who are governed by Australian Standards and Federal WHS legislation. Divers have relatively easy access to dive shops in Tasmania and, although some oversight of cylinders occurs when they are filled at commercial filling stations, the onus is still on the diver to have their regulators serviced regularly. The scuba equipment used by three of 12 divers for whom it was checked was found to have faults, had not been maintained or servicing was not documented. Where equipment was identified as a causative factor, incorrect assembly was contributory. With equipment problems featuring as a significant predisposing factor, the need for divers to ensure their scuba equipment is well maintained and properly assembled is an important safety message.

Health issues were the second most common PF contributing to diving deaths, present in almost half of the victims. Health issues were also identified as the DA for three victims, directly following the identified PF. Had these older divers received appropriate health risk assessment, their deaths

may have been avoided. This study has highlighted the need for medical risk assessment to be undertaken, particularly in older divers. We found no evidence that any divers with medical issues had received any medical risk assessment regarding diving. In Australia, there are no enforceable regulations which mandate any form of medical assessment of recreational scuba divers of any age.

Lack of experience relative to the diving conditions was identified as a PF in almost half of these deaths. It became apparent through chain of events analysis that inadequate (or relative) experience led directly to fatal outcomes. On that basis, it is reasonable to advocate that all recreational divers should have training that is appropriate to the dives being undertaken, and that refresher courses or at least skills assessment are mandatory when divers have had a prolonged period out of the water. At present, a scuba diving qualification is issued as a lifetime certification, irrespective of how frequently a diver enters the water.

Failures of the buddy system contributed to 14 of the 17 fatalities in this study. These were classified as planning-related (PF) and primary diver error (TF). These victims either dived alone or intentionally/accidently parted from their buddy. In fact, only three divers were with a buddy, which is an identical proportion to that reported for victims Australia-wide from 1972–2005.[3] The high number of dive fatalities occurring when the buddy system broke down highlights the importance of this system in contributing to safety. With an alert buddy, a distressed or impaired diver may be identified prior to becoming incapacitated and rescued swiftly. Whilst the diver may not always be saved, as seen in some of the cases in this series, it does afford the victim a better chance of survival.

The activity-related factors identified mainly related to poor choices when diving in hazardous sea conditions, leading to a chain of events which was ultimately fatal. Environmental factors included cold water immersion, dangerous sea conditions, current and waves and dangerous features such as rocks, caves, kelp or ropes (risking entanglement). Extra time and effort to observe and assess the diving environment prior to a dive is a worthwhile safety investment; choosing not to dive is an important option that may be overlooked. Risk assessment and dive planning are usually not taught until more advanced recreational courses.

As demonstrated in CEA by other authors, there are often multiple factors contributing to negative outcomes.[10] Many fatalities had multiple PFs that contributed to a diver's death. Such PFs can be addressed through public health messages, and education. Some may require strengthening of regulations and training, particularly relating to recreational hookah equipment and health surveillance.

Including PF in investigating diving deaths, analysing a chain of events rather than looking for a single root cause, added value to this study. Without assessing PF, important pre-immersion issues that contributed to diving deaths would be missed and important opportunities for risk mitigation would be lost. Analysis of linkages between PFs and TFs highlighted some areas for further research and possible prevention strategies. For example, linkages of diver health to impact from the environment are very important for safe diving in Tasmania. Campaigns to improve equipment maintenance and eliminating home-made equipment may save lives. In addition, a focus on diver training, particularly in the area of pre-dive decision making may help to reduce the incidence of fatalities. Emphasis on the advantages of the buddy system should be part of this training.

Investigating diving deaths presents many challenges, even with a systematic approach. For two cases (CG-02 and CG-17) it was difficult to ascertain or assign a trigger, despite reasonable information from the scene. Three of the cases (CG-01, CG-07 and CG-17) had an unknown DA owing to missing information, difficulty appropriating information or the fact that these deaths occurred unwitnessed. One fatality (CG-02) had three DAs assigned; it was difficult designating which agent was the most important in precipitating death. On some occasions, multiple possibilities need to be accepted when analysing diving deaths.

The identification of DIs was relatively straight-forward, well-conducted autopsies making it possible to assign these with reasonable accuracy. They again flowed logically into the cause of death as described by the Coroner. However, for cases CG-02, CG-06 and CG-15, it was considered after CEA that there was a likely alternative causation of death other than that given by the Coroner. For CG-02, despite extensive analysis of available information, it could not be determined whether the diver experienced a rapid ascent causing CAGE (which was never witnessed) or whether the diver drowned at depth. The Coroner's report stated: "*Without knowing the deceased's dive profile, it is extremely difficult to postulate the exact pathological sequence of events*". This diver did have a CT which led to the conclusion of CAGE as there was gas throughout the body. However, there was also evidence of drowning. Without clear history of a rapid ascent and off-gassing prior to a delayed post-mortem CT scan, the diagnosis of CAGE is less convincing.

In CG-06 and CG-15, IPE was considered a more plausible cause of death than the coronial finding.[17] IPE is difficult to diagnose at autopsy; the pathological findings being similar to those of drowning.[17] During CEA both divers demonstrated risks for IPE, including previous similar episodes and pre-existing medical issues. Both were over 50 years of age, placing them at a higher risk. In 30% of cases of IPE in scuba divers, there is a likelihood that this had occurred on previous dives.[17] Autopsy in both cases confirmed cardiac disease which may have increased susceptibility to IPE. The CEA highlights the benefits of having an expert prospectively involved when diving deaths are analysed.

LIMITATIONS

This was a retrospective series and it is recognised that data may be incomplete for individual cases and that some cases may have been missed. The study methodology was structured to limit such problems by utilising a model template that has been verified to allow a more effective and systematic collection and analysis of these data.

Data quality was variable; in particular deaths occurring up to the year 2000 had less comprehensive information. At times this was inevitable due to absence of witnesses, or the diver being separated from buddies.

Autopsy reports can sometimes have a degree of uncertainty, because of the difficulty in determining the presence of CAGE, delays in performing an autopsy or difficulties in identifying medical causes such as dysrhythmias or differentiating IPE from drowning.[14]

Even with five-step chain of events analysis, it remains possible that other important co-factors were overlooked.

The number of deaths (17 over a 20-year period) was low. Despite this, it proved possible to gather important information that has potential to improve safety for divers in Tasmania into the future.

Conclusions

Seventeen diving deaths were recorded in Tasmania over 20 years; all recreational divers (five on hookah and 12 on scuba). There were no diving deaths in the professional sector during this period. The key public health messages for divers include:

- Health – promotion of regular diver health reviews, especially for those with known medical conditions or > 45 years of age.
- Equipment condition and maintenance – recreational gear should be subject to similar standards to those for commercial divers.
- Hookah compressors – require a system of oversight for use in the recreational environment, including air testing certification. There is a need for a specific recreational hookah diver training course and mandating a functioning accessory air supply.
- Training – all divers should have regular training to ensure they maintain safe diving practices. Divers who have not dived for some time should undergo refresher training. The buddy system should be reinforced.
- Acclimatisation to the colder waters surrounding Tasmania has been identified as a key risk for visiting divers. These divers should receive initial higher-level supervision and support until their competence in colder water is confirmed.
- Choosing not to dive is an important option that may be overlooked by divers.

These data and key messages will be presented to the Tasmanian Government and to the Coroner's office, as well as presenting to local dive regulators and diving communities to ensure that this diving knowledge is dispersed to help reduce future fatalities.

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Conflicts of interest

David Smart is a member of the DHM Editorial Board, but took no part in the peer review or decision to publish processes.

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Review articles

Immersion pulmonary oedema: a cardiological perspective

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Key words
Cold; Exercise; Hypertension; Rebreathers; Renal artery stenosis; Takotsubu cardiomyopathy; Transpulmonary pressure; Case reports

Abstract

(Wilmshurst PT. Immersion pulmonary oedema: a cardiological perspective. Diving and Hyperbaric Medicine. 2019 March 31;49(1):30–40. doi: 10.28920/dhm49.1.30-40. PMID: 30856665.)
It is postulated that immersion pulmonary oedema (IPE) occurs because of combinations of factors that each increase the hydrostatic pressure gradient between the pulmonary capillaries and the alveoli. The factors, by definition, include the effects of immersion, particularly raised central blood volume and hence cardiac filling pressures. Breathing against a negative pressure is important but the magnitude of the effect depends on the relation of the diver's lung centroid to the source of the breathing gas and the breathing characteristics of diving equipment. Other factors are cold-induced vasoconstriction, exertion and emotional stress, but variations of the responses of individuals to these stimuli are important. Hypertension is the most frequent cardiovascular disease predisposing to IPE but other medical conditions are implicated in some patients.

Background

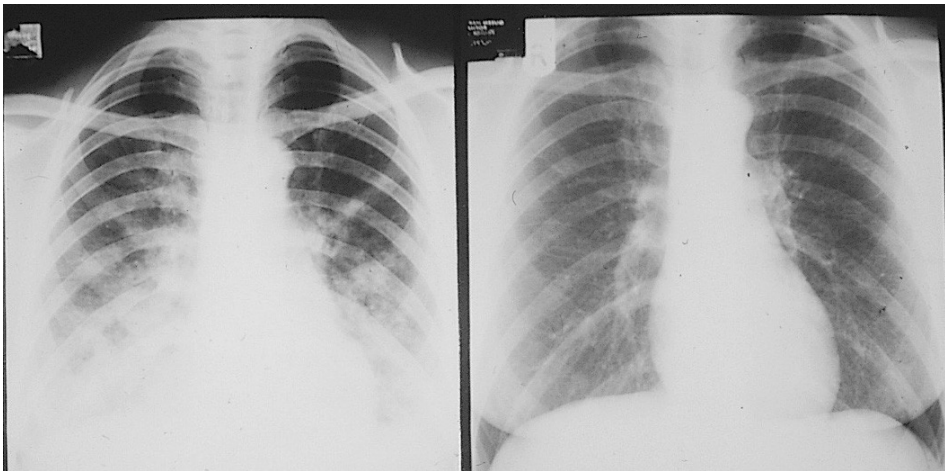
Immersion pulmonary oedema (IPE) occurs in divers and surface swimmers and onset is acute.[1] Acute pulmonary oedema can occur if the pulmonary capillary permeability is increased as a result of damage to the alveolar-capillary interface (non-cardiogenic pulmonary oedema).[1] Cardiogenic pulmonary oedema occurs without increased capillary permeability when the hydrostatic pressure gradient across the pulmonary capillary membrane exceeds the oncotic pressure – the osmotic pressure exerted by plasma proteins.[2] The term cardiogenic pulmonary oedema implies that the cause is within the heart, but that is not always so. If the plasma albumin concentration is low, so that the plasma oncotic pressure is also low, pulmonary oedema can occur when the hydrostatic pressure gradient across the pulmonary capillary membrane is within the normal range. The distinction between cardiogenic and non-cardiogenic pulmonary oedema is not always sharp. Cardiac disease with high pulmonary capillary pressures can cause fracture of capillaries to produce bloodstained oedema and haemoptysis, so the oedema is not a pure transudate. The evidence suggests that IPE is a form of cardiogenic (or hydrostatic) pulmonary oedema.

The precise hydrostatic capillary pressure equivalence of the oncotic pressure depends on the plasma concentrations of proteins, particularly albumin, but in normal individuals it is approximately 25 mmHg.[2] At rest, healthy individuals have a lower pulmonary capillary pressure (less than 12 mmHg) and there is net movement of water from the alveoli into the pulmonary capillaries. Even in normal individuals the difference between dry lungs and onset of pulmonary oedema formation may be an increase in pulmonary capillary pressure of only 15 or 20 mmHg.

An illustration of the delicacy of the balance between pulmonary oedema and dry lungs is the common appearance of a chest X-ray (CXR) of a patient with pulmonary oedema (Figure 1a). There is airspace opacification at the lung bases but clearer lung apices. That is because, in a patient who is upright, the pulmonary capillaries situated at the level of the left atrium will have a capillary pressure that mirrors left atrial pressure, but pulmonary capillaries that are 15 cm above the level of the left atrium will have a capillary pressure that is 15 cm water (H_2O) pressure (approximately 11 mmHg) less than left atrial pressure. Cardiogenic pulmonary oedema forms most rapidly when the pressure gradient across the pulmonary capillary membrane increases rapidly to a high level to cause 'flash' pulmonary oedema.[3]

In most situations, acute pulmonary oedema occurs because of a significant increase in left atrial pressure: plasma protein concentrations do not fall rapidly and usually intra-alveolar pressure changes are small. However, acute pulmonary oedema can occur when there is sudden extrathoracic airway obstruction, such as glottic obstruction following extubation after general anaesthesia, resulting in a patient exerting high negative inspiratory pressures – effectively sucking fluid from plasma into the alveoli.[4] Slower onset of pulmonary oedema is described in dogs when resistance to inspiration caused a 15–20 cm H_2O negative pressure during inspiration that was maintained for 20 to 120 minutes (min).[5] More chronic pulmonary oedema is described in man when there

Figure 1
The left chest X-ray is of the first diver recognised as having pulmonary oedema triggered by diving; the right chest X-ray is of the same diver four weeks later



were comparably negative airway pressures maintained for days.[6]

Cardiogenic pulmonary oedema is produced in the opposite ways to the therapeutic methods employed to clear it from the lungs, which are by reducing pulmonary capillary pressure (with diuretics and vasodilators) and by increasing intra-alveolar pressure, for example by use of continuous positive airway pressure (CPAP).[7]

Anyone, no matter how fit they are, can develop pulmonary oedema if the pressure gradient across their pulmonary capillary membrane is increased sufficiently for long enough. The data and clinical observations suggest that a number of factors that individually increase the pressure gradient across the pulmonary capillary membrane can, when acting in combination, produce a cumulative increase in the pressure gradient to cause IPE.

Immersion pulmonary oedema

In 1977, I was in the boat that picked up a pair of divers from my diving club in the United Kingdom (UK). They had aborted a dive to 20 m in water at 8°C after 5 min because one had become severely breathless for no apparent reason. She was cyanosed and coughing up blood and froth.[8] I examined her. She had a tachycardia, gallop rhythm and basal crepitations. Hospital investigations, including a CXR six hours after she left the water, confirmed pulmonary oedema (Figure 1a). By the time the CXR was taken she was already feeling better and she refused treatment. A repeat CXR four weeks later was normal (Figure 1b); an electrocardiogram (ECG), echocardiogram, exercise test and myocardial perfusion scan were also normal. She said that she had had six previous episodes. Years later she was found to have hypertension and subsequently she had an episode of high altitude pulmonary oedema.[9]

In the next two years I saw two other divers in my club who had IPE. Both described two episodes. One had been so hypoxic that he became unconscious for 10 min. All episodes occurred when diving in relatively cold UK conditions. Each of the three divers had dived in warmer waters without having any problems. These three divers were the subjects of the initial report of "*recurrent pulmonary oedema when scuba diving*".[10]

It appeared that there was an individual susceptibility to IPE, because some individuals were affected repeatedly when their dive buddies were unaffected. Also there were extrinsic factors because only immersion triggered their pulmonary oedema. The occurrence of IPE in three divers in a club with approximately 200 members suggested that this previously unrecognised condition might be common.

A presentation in 1984 included cases that we thought had died from "*heart failure when diving*" and concluded that "*it is probably a very serious and frequent cause of illness amongst divers…and an occasional cause of death*".[8]

VASCULAR RESPONSE TO COLD

Eleven divers out of a larger group of patients with IPE agreed to be subjects of a research study.[1] Their ages were 38 to 60 years (mean 45.6 years). They had no detectable cardiac disease and had good exercise capacity on land. They each had many years of diving before their first episodes, suggesting that susceptibility developed with time or age. Episodes occurred when diving in cooler British waters but some had dived in warmer waters without symptoms. The equipment they had used during the episodes included single- and twin-hose demand valves. Two also had pulmonary oedema when surface swimming. The vascular responses to cold (head and neck packed in towels soaked in ice-cold water) and to a raised partial pressure of oxygen

(67%) in the supine position in these eleven divers were compared with ten control divers. The stimuli induced pathological vasoconstriction in the divers that had a history of IPE compared with the controls. In a previous report, an initial group of IPE sufferers had increased venous tone and reduced venous compliance at rest and with vasoconstrictor stimuli.[11] (Comparable data were available for the whole group, but the *Lancet* requested that the paper be shortened and those data removed.)

An important observation was that nine of the eleven developed evidence of cardiac decompensation during the physiological stimuli out of the water, including one who had frank pulmonary oedema and required treatment with an intravenous vasodilator infusion.[1] None of the controls had evidence of cardiac failure. During follow up for an average of eight years the majority of the divers with IPE developed hypertension. It was postulated that increased vascular reactivity to cold in some individuals combined with the increased preload that occurs during immersion might be enough to trigger pulmonary oedema when immersed.[12] It is notable that hyper-reactivity during a cold pressor test in children is predictive of development of hypertension during follow up for 45 years.[13]

The observation in the Lancet paper that those who had IPE were at increased risk of having pulmonary oedema when out of the water was supported by the report of three individuals without evidence of heart disease who had pulmonary oedema when scuba diving but also had pulmonary oedema unrelated to immersion.[9] One had three episodes of pulmonary oedema precipitated by emotional stress. One had three episodes precipitated by sexual intercourse. The third, an exceptionally fit member of special forces, had an episode during strenuous exertion in a cold climate. Others have reported that hypertension is associated with IPE and with recurrent episodes of IPE.[14,15]

The increased preload caused by immersion combined with cold-induced vasoconstriction does not explain all cases of IPE. In a report of four individuals who had radiologically confirmed pulmonary oedema when scuba diving and/or swimming and another with probable IPE based on history without radiological or clinical confirmation, the vascular responses to cold were compared to six controls.[16] No differences were found between the groups and none developed evidence of cardiac decompensation during the experiment. The protocol used was similar but not identical to the protocol in the earlier study.[1,16]

With the exception of a woman aged 39 years, who had hypertension, the individuals reported were much younger (23–27 years)[16] than the age range (38–60 years) of subjects reported previously,[1] and none had hypertension. The woman aged 39 had five episodes of IPE (two were when diving in water colder than 6°C, and three were when swimming long distances in competition or training in water warmer than 18°C). The three younger individuals had one episode of IPE each. Two occurred when diving in cold water (less than 6°C). The other occurred during a competitive long-distance swim in warmer water (20.6°C). Recreational scuba diving and competitive open water swimming each involve immersion, but generally the latter involves considerably more exertion during immersion.

EXERCISE

Further evidence appeared that there is another younger group of patients who get IPE .[17,18] Eight out of 30 male military recruits (aged 18–19 years) developed pulmonary oedema during a 2.4 km swimming time trial in seawater at 23°C.[17] They swam in a supine position using fins. The recruits each drank five litres of water during the two hours before the swim. Two of the eight had recurrence of IPE when they repeated the swim without prior fluid loading. This suggests that when immersed some individuals are at increased risk of developing IPE when there is only one additional factor increasing the gradient across the pulmonary capillary membrane (i.e., exercise) whilst others needed a further additive factor (i.e., fluid loading).

The same group later reported a further 70 fit male military recruits age 18–19 who had IPE during a three-year period and 16 of these had recurrent episodes.[18] The episodes represented 1.8% of all swimming trials performed. The authors postulated factors predisposing to IPE in fit young men including increased pulmonary vascular pressures from cold and exercise plus "*during head-out immersion, negative pressure respiration due to the hydrostatic pressure differences between upper and lower airways produces transmural pulmonary hydrostatic forces that favor a fluid shift from pulmonary vasculature to the alveoli*." Obviously the pulmonary vascular pressure that is important in formation of pulmonary oedema is pulmonary capillary pressure. When pulmonary capillary pressure is elevated, pulmonary arterial pressure is often increased secondary to the backpressure transmitted from the left heart via the pulmonary capillaries. However, when pulmonary artery pressure is increased without an increase in pulmonary capillary pressure by pathology proximal to the pulmonary capillaries, such as in primary pulmonary hypertension or massive pulmonary embolism, pulmonary oedema does not occur. Others have also suggested a role of negative pressure breathing in aetiology.[19]

Support for the possibility of two subgroups of people having IPE comes from Duke University. In one study, 36 divers with IPE were reported and another 292 cases identified from the published literature.[20] In recreational divers and swimmers with IPE the mean age was 47.8 $\pm$ 11.3 years and, within military divers and swimmers, mean age was 23.3 $\pm$ 6.4 years. Hypertension was the most frequent risk factor documented, particularly in recreational divers and swimmers who had IPE.

The same group compared 10 subjects with a history of IPE and 20 controls. They had radial artery and pulmonary artery catheters inserted before exercising on a cycle ergometer while submerged in water at 20°C.[21] The IPE group had significantly greater pulmonary artery pressure, pulmonary capillary wedge pressure, systemic vascular resistance and pulmonary vascular resistance and significantly lower cardiac output than the control group. Following the first exercise test only the IPE group were given 50 mg sildenafil orally and after 150 min the measurements during the underwater exercise protocol were repeated. During the repeat exercise after sildenafil, the haemodynamic parameters in the IPE subjects tended to normalise towards the parameters in control subjects without sildenafil, and some parameters were no longer significantly different.

For risk of developing pulmonary oedema, the key measurement is pulmonary capillary pressure. In the controls, pulmonary capillary wedge pressure during exercise was 13.1 ± 5.0 mmHg and in the IPE subjects pre-sildenafil it was 18.9 ± 5.5 mmHg (difference $P = 0.03$). After sildenafil, pulmonary capillary pressure during exercise was 16.9 ± 6.2 mmHg, which was not significantly different from either the control subjects or the IPE subjects pre-sildenafil.

There is no mention of any subject developing IPE during the test, but the haemodynamic comparison of control and IPE subjects before sildenafil suggests that those who had a history of IPE have haemodynamic differences from controls, which are consistent with an increased risk of pulmonary oedema. As such, that comparison of control and IPE subjects was consist with the original report on IPE.[1] The authors acknowledged that performing the post-sildenafil exercise test after the baseline exercise test in the IPE subjects may have influenced the findings after sildenafil. They suggested that sildenafil might be useful in the prevention of IPE.

It has been found previously that nifedipine reduces the pathological vasoconstrictor responses to physiological stimuli in individuals with IPE.[22] Therefore, for more than 30 years, I have advised divers that refused to stop diving after an episode of IPE to take 5 mg nifedipine before diving. I am not aware of recurrence when they did so, but the numbers are small.

The high incidence of IPE in the swimming trials of male military recruits in Israel is supported by data from the Vansbro swimming race (Vansbrosimningen) in Swedish rivers.[23,24] In 2016, 69 of 13,878 (0.5%; 58 of the 69 patients were women) of participants had symptoms varying from coughing to fulminant pulmonary oedema.[23,24] Forty-six patients were treated with CPAP.

A survey in triathletes in the USA reported that 1.4% had experienced symptoms compatible with IPE.[25] In the survey risk factors for IPE were female gender and hypertension. Though IPE appears to be more common in women than in men, research studies have been performed predominantly or exclusively in men.

TAKOTSUBO CARDIOMYOPATHY

There are a small number of case reports of patients who had IPE and Takotsubo cardiomyopathy.[26,27] Takotsubo affects women more frequently than men and particularly affects middle-aged and post-menopausal women, who are the group who get IPE most commonly. Takotsubo cardiomyopathy presents with chest pain more frequently than dyspnoea, whereas the converse is the case with IPE. Many cases of IPE improve within hours of leaving the water, which would not be expected if the aetiology were Takotsubo cardiomyoapthy. Takotsubo cardiomyopathy produces dramatic electrocardiographic and echocardiographic changes, which resolve in days, weeks or months in survivors. These are not found in most people with IPE. Therefore it is possible that when Takotsubo cardiomyopathy and IPE are present in the same patient, the Takotsubo cardiomyopathy is secondary to the stress of having IPE.

Reversible myocardial dysfunction was reported in 15 of 54 consecutive divers with IPE.[14] Myocardial dysfunction was defined as an elevated level of troponin with electrocardiographic and/or echocardiographic abnormalities. The abnormalities in individual patients were described in the table in that paper, but in no case were they consistent with Takotsubo cardiomyopathy. Significantly more of the 15 with myocardial dysfunction were older than 50 years (11 of 15 vs. 13 of 39), hypertensive (nine of 15 vs. six of 39) and diabetic (three of 15 vs. none of 39). Because IPE causes hypoxaemia and because myocardial ischaemia can cause reversible myocardial dysfunction we do not know whether the myocardial dysfunction in these cases contributed to causation of IPE or was the result of IPE.

NEGATIVE PRESSURE BREATHING

There is increasing evidence that divers using closed circuit (CCR) and semi-closed circuit rebreathers are at increased risk of IPE compared to divers using open circuit (see discussion below of divers 10, 11 and 12). The first reported CCR diver was a 20-year-old combat diver using a CCR, who had done 17 previous dives with the equipment, but whether the counter-lung was back- or front-mounted is not stated.[28] The authors noted that "*the pressure difference between the lung centroid and the breathing bag of closed circuit scuba equipment when this is positioned higher than the diver's suprasternal notch, produce transmural pulmonary hydrostatic forces that favor a fluid shift from the pulmonary capillaries to the alveoli*."[28]

A further 11 mine clearance divers who had IPE when using rebreathers have been reported; ten of which were semi-closed circuit rebreathers worn on the back.[29]

The effects of positive and negative pressure breathing at rest and during exercise (i.e., four interventions each for 30 min in random order on separate occasions) were investigated in 16 male professional divers, mean age 34.4 ± 12.1 years.[30] It appears that none had a history of IPE. In the divers orientated prone, breathing with a positive pressure was achieved by attaching a rebreather anteriorly and for negative pressure breathing the rebreather was attached posteriorly. Lung comet score, a grading of extra-vascular lung water detected by ultrasound, was the measure of interstitial pulmonary oedema. The ultrasound comet score was zero following dives at rest regardless of breathing pressure. Following exercise while maintaining the heart rate at 110 bpm, the mean comet score was 4.2 with positive pressure breathing and 15.1 with negative pressure breathing.[30] This study suggests that IPE may occur in fit divers during exertion even if positive pressure breathing but negative pressure breathing increases the risk of IPE.

The same group also described a special forces trainee aged 26 years who had IPE with a front-mounted counter-lung rebreather.[31] The authors demonstrated that when in a head up position in the water with the automatic diluent valve adjusted as the diver had done, he was breathing against a negative pressure.

Swimmers and snorkellers are negative pressure breathers because their lung centroid is below the surface of the water. Their posture in the water will affect the degree of negativity. A scuba diver using a single hose demand valve will be positive pressure breathing if he descends head first and negative pressure breathing during ascent. If using a rebreather, the relative positions of the gas in the counter-lung and the diver's lung centroid will determine the pressure difference. The pressure difference may be dynamic. For example, as a diver in the prone position breathes from a back-mounted counter-lung, the reduction in volume may cause the negative pressure to increase towards end-inspiration for two reasons; 1) all counter-lungs have a degree of elastance and, therefore, the pressure in the counter-lung is slightly greater at the start of inspiration, and 2) during inspiration the volume of gas in the counter-lung will decrease and the distance between the lung centroid and the gas "*level*" in the counter-lung will increase (G Anthony, personal communication, 2018). In addition, resistance to breathing of the equipment may affect generation of negative intrathoracic pressures, particularly as gas density increases with depth.

TIGHT SUIT/EQUIPMENT

Compression from a wetsuit increases urine volume.[32] It is almost certainly the result of an increase in venous return and hence filling pressures. It is possible that a tight wetsuit would also increase pulmonary capillary pressure by the same mechanism.

Illustrative case reports

Therefore, it appears that the risk of experiencing IPE is determined by factors intrinsic to the diver or swimmer and extrinsic factors related to the conditions during immersion and the equipment used during a dive. Investigation and analysis of events may be required to determine causation. Some previously unreported cases have been selected as examples to provide insight into the mechanisms involved. Patients gave consent for publication of the description of their case reports.

Hypertension

Hypertension is the most frequent intrinsic factor found in divers who had IPE. Some were known to have hypertension before they had IPE. Many of those divers stop diving and remain well thereafter. The majority of divers that I see with IPE had it diving in cool UK waters, but as more UK diver travel abroad for diving holidays, a greater number are reporting IPE when immersed in warm waters.

CASE 1

A 67-year-old female had performed 240 dives breathing air on open circuit scuba: 153 in British waters wearing a drysuit and 87 in warm seas wearing a wetsuit. She had dyspnoea, cough and haemoptysis during three warm-water dives (depths 12–30 metres' sea water (msw) at ages 58, 64 and 67. CXR confirmed pulmonary oedema on the last occasion. Apart from hypertension, first found during pregnancy, her cardiac findings, ECG and echocardiogram were normal. A treadmill exercise test was normal except for an exaggerated blood pressure response. She stopped diving and remained well 11 years later.

Some people who get IPE develop hypertension later. Some also had high altitude pulmonary oedema, as occurred in the first case seen in 1977.[9]

CASE 2

A female, who had done 40 dives in warm waters during her vacations, had four episodes of IPE between the ages of 55 and 66 years. Three occurred when scuba diving (depths 14–30 m) breathing air or nitrox 32 on open circuit. One was when snorkelling on the surface. She also had an episode of high altitude pulmonary oedema after a rapid ascent to 14,500 feet in Ecuador. A subsequent ECG, echocardiogram, treadmill exercise test, urinary catecholamine excretion and renal ultrasound were normal. She has not dived for eight years but has continued to snorkel without recurrence of IPE. She subsequently developed hypertension.

Two men had renal artery stenosis (cases 3 and 4). Neither had pulmonary oedema out of the water, but it appears that in these individuals, who had activation of the renin-

angiotensin-aldosterone axis, the additional effects of immersion triggered pulmonary oedema.

CASE 3

A commercial diver had scuba dived since age 14 and done many thousands of uneventful dives. Hypertension was diagnosed at age 48. Between the ages of 49 and 51, he did 65 dives and had nine episodes of severe dyspnoea, sometimes expectorating bloodstained froth. The first episode was when snorkelling in tropical water. Eight episodes were when using open circuit scuba: one was in a heated training pool and six episodes were when diving with air as the breathing gas in tropical water wearing a wet suit. The depths were 6–40 msw. Another diver rescued him because of severe dyspnoea on one occasion.

The eighth episode was when diving to 15 msw in the English Channel, wearing a drysuit and breathing nitrox32. Water temperature was 8°C. He became severely breathless 25 min into the dive. He had bilateral pulmonary crepitations and CXR confirmed pulmonary oedema. Outpatient investigations showed persistent hypertension; left ventricular hypertrophy (LVH) and global impairment of left ventricular (LV) systolic function (ejection fraction 42%) on echocardiography; impaired renal function (glomerular filtration rate 63 $ml \cdot min^{-1}$); normal urinary catecholamine excretion; and a severe stenosis of the left renal artery consistent with fibromuscular dysplasia on a CT angiogram. Following balloon angioplasty of the renal artery stenosis his blood pressure was controlled very well on amlodipine 10 mg daily. He returned to diving and performed hundreds of commercial dives in both tropical and British waters during the next eight years without event.

At age 59 he had another episode of IPE after 30 min when at 16 m depth in 16°C water. (He had performed longer working dives in the two preceding days without problems.) Soon after he surfaced he became unconscious. His initial oxyhaemoglobin saturation in hospital was 79% on 100% oxygen. CXR again confirmed pulmonary oedema. ECG showed lateral T-wave inversion. Measurements of troponin were normal. He improved over two days, but after returning to the UK he had dyspnoea on exertion and nocturnal dyspnoea. Two months later his ECG was unchanged, and an echocardiogram showed LVH with moderate global impairment of LV function. A CT angiogram showed no recurrence of the renal artery stenosis. A myocardial perfusion scan showed no evidence of ischaemia but the LV was dilated with an ejection fraction of 33%. The current diagnosis is LV dysfunction secondary to hypertension. He will not dive again.

In case 4, in addition to renal artery stenosis there were other risk factors for IPE (fluid loading and a long duration dive using a rebreather).

CASE 4

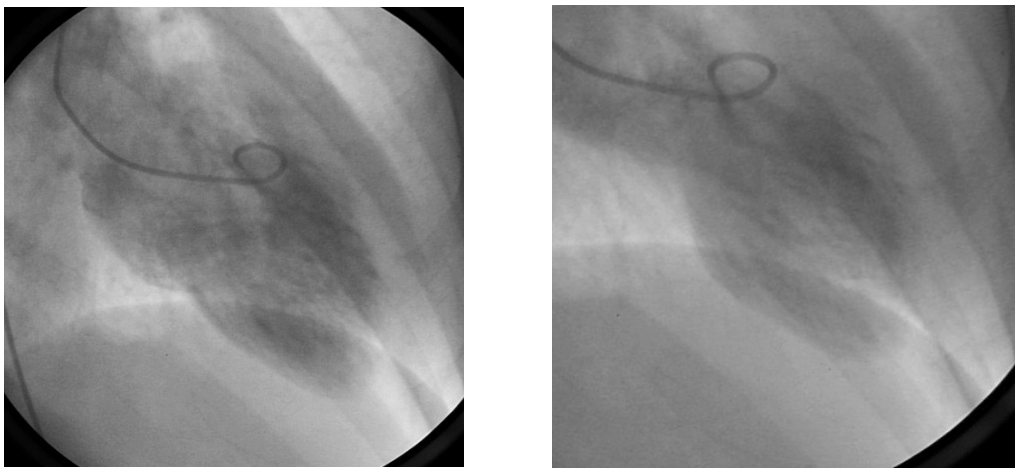
A 57-year-old male with mild hypertension had performed 240 scuba dives in the five years preceding an episode of pulmonary oedema. Most dives were on open circuit scuba, but 72 dives were with a rebreather (Evolution Plus, Ambient Pressure Diving Ltd, Cornwall, UK). For 15 of the dives using the rebreather, the breathing gas was trimix. During a holiday in the tropics, he performed two dives per day for three weeks. The breathing gas on some dives was air on open circuit and on others it was trimix using the CCR. In the few hours before the next deep trimix dive, he drank more than 3 L of fluid. The maximum depth was 89 msw and duration 81 min. He became progressively more breathless from 12 min into the dive with a cough during the ascent, but managed to follow his planned decompression schedule. However, he needed assistance from his dive buddy and about half a metre below the surface he lost consciousness. On regaining consciousness, he expectorated blood-stained froth but had no chest pain. CXR and CT chest showed pulmonary oedema. An ECG showed lateral T-wave inversion. Two weeks later a repeat ECG showed persistence of lateral T-wave inversion, but subsequently the T waves became normal. An echocardiogram showed mild aortic valve thickening without stenosis and good LV function. During a treadmill exercise test, he completed 12 min of the Bruce protocol without chest pain or ECG changes. Blood pressure rose to 190/100 on exercise. An MRI angiogram showed stenosis of the left renal artery. The kidneys and the adrenal glands were normal. The stenosed left renal artery was stented. Since returning to diving, he has performed 160 dives - using a CCR for 121 dives (deepest 101 msw and longest over 185 min) and using open circuit for 39 dives. without recurrence of symptoms. He now avoids over-hydration before dives.

Case 5 also suggested the possibility of excessive activity of the renin-angiotensin-aldosterone axis as a contributor to IPE in some cases.

CASE 5

A 47-year-old female had done 357 uneventful dives before having pulmonary oedema when diving to 30 msw, breathing air on open circuit scuba in British waters. It was the first day of her menstrual cycle. Her weight often increased 1.5 kg at the start of menstruation. Before this particular dive, she had peripheral oedema and felt bloated, which was not unusual at that stage of her cycle. She had cough and dyspnoea 21 min into the dive. Her consciousness level was impaired. CXR confirmed pulmonary oedema. Oxyhaemoglobin saturation was 84%. Cardiac enzymes and ECGs were normal. An echocardiogram four days later was normal. Lung function tests and treadmill exercise tests were normal. In the subsequent 10 years she has been well, with normal blood pressure and she has done about 50 uneventful shallow dives.

Figure 2
Left ventriculogram of Case 7 who had Takotsubo cardiomyopathy associated with immersion pulmonary oedema, showing the end diastolic frame (left panel) and end systolic frame



Case 5 had pulmonary oedema on the first day of menstruation when she was oedematous. Fluid retention in the late-luteal phase of the menstrual cycle is related to activation of the renin-angiotensin-aldosterone axis.[33] Pulmonary oedema at the time of menstruation in 14 consecutive menstrual cycles was reported in a woman with mitral stenosis and was attributed to fluid retention and increased blood volume.[34]

Other cardiac diseases

As stated before, hypertension is the most common cardiovascular finding in divers presenting with IPE. Most have preserved LV function, but some have LV systolic dysfunction on an echocardiogram and/or a cardiac MRI scan, but without significant coronary narrowing or evidence of myocardial infarction. In a far smaller number of cases, IPE is the presenting event in divers who have significant coronary artery disease, dilated cardiomyopathy or valvular disease.

CASE 6

A 56-year-old male had two episodes of pulmonary oedema during consecutive shallow dives using open circuit scuba in tropical water. His work involved heavy lifting but he had no cardiac symptoms in everyday life. He had dived for 10 years but only on his annual vacations in tropical water. He became unusually breathless during his first dive of a holiday, a gentle dive to 8 msw for 55 min, but he recovered quickly afterwards. The next day, 13 min into another 8 msw dive he had cough and severe dyspnoea. He had signs and the CXR appearances of pulmonary oedema. There was a loud mitral systolic murmur. Cardiac enzymes and an ECG were normal. An echocardiogram showed that the left ventricle was mildly dilated with prolapse of the posterior mitral valve leaflet causing moderate mitral regurgitation into a dilated left atrium. Cardiac catheterisation showed pulmonary artery pressure 62/24, mean 40 mmHg; mean pulmonary capillary wedge pressure 23 mmHg; left ventricular end diastolic pressure 29 mmHg; and aortic pressure 117/73 mmHg. Coronary angiography showed severe stenoses in the proximal left anterior descending and right coronary arteries. He had a mitral valve repair and two coronary artery bypass grafts. He has not dived again in eight years following surgery.

Takotsubo cardiomyopathy and emotional stress

Takotsubo cardiomyopathy has been described in association with IPE.[26,27] I have seen only one case.

CASE 7

A 59-year-old female had done over 600 dives in 27 years and developed pulmonary oedema during a dive to 29 msw for 33 min off the Canary Islands. She breathed air on open circuit scuba. She wore a wetsuit, but felt cold. She had dyspnoea without chest pain and expectorated bloodstained froth. CXR confirmed pulmonary oedema. Blood gases showed severe hypoxaemia. Her ECG showed ST elevation evolving to T-wave inversion in anterior and inferior leads and Troponin T was elevated. Two days later, a coronary arteriogram showed normal coronary arteries. The left ventriculogram was typical of Takotsubo cardiomyopathy (Figure 2). Four months later blood pressure was 180/80, but ECG, CXR and echocardiogram were normal. During a treadmill exercise test she completed 11 minutes of the Bruce protocol without abnormality. She did not dive in the subsequent four years and remained well, but has hypertension.

In this and other cases there is uncertainty whether the Takotsubo cardiomyopathy was the cause of IPE or the result of the stress of the events. Certainly, other divers

with stressful episodes precipitating IPE have no evidence of Takotsubo cardiomyopathy (Cases 8 and 9).

CASE 8

A 65-year-old male had performed over 1,000 dives before he had an episode of immersion pulmonary oedema during a highly stressful dive to 30 msw using open circuit scuba in the Mediterranean. Clinical features of IPE were confirmed by CXR and CT chest; ECG and troponin were normal. During a treadmill exercise test there was an exaggerated blood pressure response (maximun 230/95). Ambulatory blood pressure recording confirmed hypertension. An echocardiogram showed concentric LVH, confirmed on a cardiac MRI scan, but there was no evidence of ischaemia, infarction or fibrosis. Urinary catecholamine and cortisol excretion, plasma aldosterone and renin concentrations and a renal ultrasound were normal. In the six years since his episode of IPE he has continued diving without recurrence.

CASE 9

A 53-year-old female had performed more than 1,000 uneventful dives using open circuit scuba in 29 years. Most were in the UK. She then performed two uneventful dives one day in Scottish waters wearing a neoprene drysuit. The next day she felt congested and took a proprietary brand of pseudoephedrine 60 mg one hour before a 47 msw dive breathing air. She had not used pseudoephedrine for 10–15 years. It was dark and she became separated from her buddy. She felt stressed. During the ascent she started to cough, had chest tightness and felt breathless. On the surface she coughed up bloodstained froth. Oxyhaemoglobin saturation was 80%. CXR showed widespread pulmonary oedema. An ECG was normal apart from sinus tachycardia. Cardiac enzymes were normal. She was treated with CPAP. Blood pressure, an echocardiogram, a cardiac MRI and stress perfusion imaging were normal. She has not dived in three years since she had IPE and remains well.

This experienced diver had IPE after a stressful dive and after she had taken pseudoephedrine. Takotsubo-like myocardial dysfunction can be associated with phaeochromocytoma.[35] Pseudoephedrine has sympathomimetic properties, but she had no evidence of Takotsubo cardiomyopathy. She was not and is not hypertensive, but pseudephedrine causes vasoconstriction and can increase blood pressure.[36] It is possible that in a situation when there was increased preload from immersion and increased afterload from cold exposure, the combination of stress and pseudoephedrine acted synergistically to precipitate IPE.

IPE during use of a rebreather

A number of divers who had dived for years without problems had episodes of IPE soon after starting to use a rebreather, particularly when there was a back-mounted counter-lung. Case 10 is an example.

CASE 10

A 29-year-old male had a single episode of pulmonary oedema during his thirteenth dive using a CCR (Inspiration Vision, Ambient Pressure Diving Ltd, Cornwall, UK) and breathing nitrox. He had performed about 300 dives in cool British waters and about 100 dives in warmer seas using open circuit scuba without problems. He breathed air on about half the dives and nitrox on the others. The deepest dive was 44 msw. He did four uneventful training dives with the CCR before he went on a trip to the Orkney Isles. The episode of pulmonary oedema occurred on the ninth dive of the trip. The maximum depth of the dive was 38 msw. Cough and dyspnoea started about 30 min into the dive when at 25 msw. He expectorated bloodstained froth. He had bilateral pulmonary crepitations. Oxyhaemoglobin saturation was 94%. CXR confirmed pulmonary oedema. Subsequently his blood pressure, cardiovascular examination, an ECG, echocardiogram, treadmill exercise test, urinary catecholamine excretion and CT renal angiogram were all normal. He has stopped diving and has been well without cardiac problems in the subsequent seven years.

CASE 11

A scuba instructor was first seen at age 43 years. He is now age 50. Over 24 years he had performed more than 2,000 uneventful dives using open circuit scuba with breathing gases being air, nitrox and trimix (deepest 96 msw). He also used rebreathers for about 150–200 dives, breathing nitrox and trimix. The average duration of dives using the rebreather was approximately 90 min. When first seen he described five episodes of dyspnoea, wheeze, cough and expectorated bloodstained froth when diving using a CCR with a back mounted counter-lung (Ouroboros, VR Technology Ltd, Dorset, UK).

CXR confirmed pulmonary oedema on one occasion. All five episodes of pulmonary oedema occurred when he used the CCR in cool British waters. He did not have episodes when using the rebreather in warm water or when using open circuit in cold or warm water. Some IPE episodes occurred when breathing nitrox and others occurred when breathing trimix with depths of dives from 30–84 msw. The onset of episodes was consistently 25–30 min into the dive, but he had no symptoms on some longer dives (over 90 min duration). Blood pressure, cardiovascular findings, ECG and echocardiogram were normal. During a treadmill exercise test he completed 19 min of the Bruce protocol without difficulty. He also described occasional irregular palpitations.

He returned to diving predominantly using open circuit scuba but occasionally using the Ouroboros CCR. At age 45, he reported another episode of IPE when diving in cold water using his CCR. Then at age 46, he had pulmonary oedema using the Ouroboros in warm water in Florida when he had to swim hard against a current. At age 48 he had IPE with

expectoration of blood-stained froth when he snorkelled rapidly in a wet suit across a cold lake in the UK. He tried diving using other rebreathers, including five dives on the Sentinel CCR (VR Technology Ltd, Dorset, UK). At age 49 he performed two dives in one day in a lake in the UK wearing a dry suit and using the Sentinel. The first was for 32 min duration. He started to wheeze 58 min into the second dive and on surfacing expectorated pink froth. Subsequently he developed persistent atrial fibrillation requiring catheter ablation. His blood pressure remains normal.

Case 11 initially had IPE only when using a CCR in cold water, but later had IPE when using it on a dive in warm water when there was significant exertion and when snorkelling rapidly in cold water. Later he had IPE when using a different make of rebreather that also has a back mounted counter-lung. Subsequent onset of atrial fibrillation raises the possibility of LV diastolic dysfunction in the absence of hypertension. It appears that Case 11 had IPE only when there were at least two of three risk factors that increased the pressure gradient across the pulmonary capillaries, namely negative pressure breathing from a rebreather with a back mounted counter-lung, cold and/or exertion.

In some cases the onset of symptoms appears linked to an event during use of a rebreather that increased negative inspiratory pressure.

CASE 12

A 48-year-old male had performed more than 100 dives on open circuit with durations up to 80 min and a number of triathlons without problems. He performed eight training dives with a CCR with a back mounted counter-lung (RedBare, Vobster Marine Systems, Somerset, UK). His next dive was his first recreational dive with this CCR. He drank a litre of fluids in the four hours before the dive. Water temperature was 5°C. He wore a drysuit. After 40 min at 10 m he descended to practice manual injection of diluent but failed to inject the diluent quickly enough and tried to inhale from an under-filled counter-lung, exerting a forceful negative pressure as he did so. He immediately experienced difficulty breathing. After surfacing he expectorated blood streaked frothy sputum. He was found to have hypertension, but an ECG, an echocardiogram, CT coronary angiogram and renal function were normal.

In Case 12, a single inspiration with forceful negative pressure appears to have triggered the onset of IPE. This is analogous to attempted inspiration with laryngeal obstruction.

Medical conditions

Some medical conditions may also increase negative pressures during inspiration.

CASE 13

A 45-year-old female had four episodes of IPE in 26 dives using open circuit scuba. The first was on her third dive and was in the UK. She became breathless after 6 min at 7 msw. After surfacing she coughed up frothy sputum. Next, she was breathless after a surface swim to a dive site in Fiji and had to abort the dive almost immediately she started to descend. The last two episodes occurred on consecutive days in the Mediterranean. On each occasion she became breathless soon after the start of the dive and on surfacing coughed up bloodstained froth. Symptoms resolved in between one and three hours. Blood pressure, ECG and echocardiogram were normal. She has Ehlers Danlos syndrome with a history of joint dislocation and an abnormally mobile trachea such that she finds that she needs to sleep with her neck in a certain position to prevent her waking with choking at night. She uses CPAP for sleep apnoea. Lung function tests and a CT chest were normal. She has stopped diving.

The lack of rigidity of the airway in Ehlers Danlos syndrome can cause dynamic extrathoracic airway obstruction.[37–39] Dynamic extrathoracic airway obstruction causes the greatest limitation of flow during inspiration, when subatmospheric intraluminal pressure draws the obstructing lesion inward, reducing the diameter of the airway lumen.[40] It can cause pulmonary oedema unconnected with immersion.[41] It is possible that in case 13, dynamic extrathoracic airway obstruction from her Ehlers Danlos syndrome coupled with the transpulmonary pressure gradient during immersion acted in synergy to increase negative airway pressures during inspiration, which coupled with other effects of immersion, caused IPE.

Death from IPE

IPE can cause sufficient hypoxia to result in unconsciousness, so it is probable that it can cause death. Some deaths from IPE have been described in the literature.[8,42,43] In the UK, coroners have certified IPE as the cause of some diving deaths.

Making a diagnosis of IPE in a diver or swimmer who survives and is able to give a history of the events is much easier than making a diagnosis of IPE in someone who has died. At post mortem examination it may be difficult to distinguish pulmonary oedema fluid from inhaled fluid. The greater difficulty is that pulmonary oedema formation after death is very common and particularly so when resuscitation is attempted.[44] The reason is that when cardiac function ceases in a normally hydrated person, all intravascular pressures equalise at approximately 40 mmHg – not at zero. A pulmonary capillary pressure of 40 mmHg exceeds the plasma oncotic pressure and transudation occurs. During efficient cardiac massage forward flow occurs so more blood enters the pulmonary capillaries from the right heart to replace the plasma that has passed into the alveoli. In

addition, during chest compression, all cardiac chambers are compressed, so there is backpressure from the left atrium to pulmonary capillaries. Therefore, unless a person dies from haemorrhage or is severely hypovolaemic at the time of death, pulmonary oedema is the usual pulmonary finding at post mortem.[44]

Therefore, in order to diagnose IPE as the cause of death one needs to rely on other indirect evidence. It might be that other divers observe that the diver was breathing rapidly for no obvious reason or see a change to a secondary breathing gas supply when the primary gas supply is later found to be working satisfactorily. Occasionally there is photographic or video evidence of the events to suggest IPE.

Using such evidence, it seems probable that IPE is the cause of death in a significant number of UK divers who died. In particular, some very experienced UK scuba divers, who had performed thousands of dives over many years, appear to have died from IPE during their earliest dives, often training dives, using rebreathers.

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Diving and attention deficit hyperactivity disorder

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Abstract

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Attention deficit hyperactivity disorder (ADHD) is a psychiatric condition that affects attention, concentration, impulse control and awareness. Not only these symptoms, but also the medications used to treat ADHD (psychostimulants) pose a risk to both the diver and his or her buddy. This article presents guidelines for recreational diving in combination with ADHD and psychostimulants. These guidelines are based solely on 'expert' opinion and were adopted at a meeting of the Dutch Association for Diving Medicine in 2017.

Introduction

Scuba diving, with millions of participants across the world, is a generally safe sport, provided reasonable limits of depth/time/frequency are observed by a reasonably fit and well-trained diver.[1] To certify that a person is fit to be taught to dive ideally implies that he/she is physically fit, medically healthy and psychologically stable. A preventative diving medical examination is aimed at detecting any medical risks and issuing an appropriate recommendation. Psychiatric issues and psychopharmacology pose a risk to both the diver and his or her buddy. Little is known about how medicines and hyperbaric or diving conditions might interreact. Psychotropic medicines may increase susceptibility to nitrogen narcosis and cerebral oxygen toxicity; however, there is no experimental evidence for these claims.

The Divers Alert Network Europe, in discussing the lack of knowledge about the interaction between psychiatric disease and pharmacological agents used in its treatment, makes the general comment that "*stimulant medications used to treat attention deficit hyperactivity disorder (ADHD) show no clear risk; the greater risk is the loss of medication benefit if it is not taken in a timely fashion*".[2]

The following guidelines were endorsed by the Dutch Association of Diving Medicine at its annual meeting in 2017 and formulate advice for recreational diving in people with ADHD and taking psychostimulants.[3] The guidelines cannot be substantiated beyond the level of 'expert opinion' but are intended to support rational clinical practice.

Method

A systematic search of the literature was performed using PubMed up to November 2017. A screening literature search was used to identify all literature discussing scuba diving and any ADHD topics. Search terms included:
(Ritalin[tiab] concerta [tiab] or Dexedrine [tiab] or Strattera [tiab] or methylphenidate[tiab] or "*Methylphenidate*" [Mesh] or "*Dextroamphetamine*" [Mesh] or "*Atomoxetine hydrochloride*" [Mesh] or "*Attention deficit disorder with hyperactivity*" [Mesh] or "*ADHD*" [tiab] or "*ADD*"[tiab] or (attention [tiab] and deficit [tiab]) AND ("*Diving*" [Mesh] or Diving [tiab] or SCUBA [tiab] or divers [tiab]). The search resulted in identification of 29 titles, with only one relevant research article.[4]

In addition, handbooks on diving medicine and diving medical books which discussed medical examination, psychological and psychiatric areas of attention were screened to identify additional information.[5–9]

Applicability of the recommendations in these guidelines:

- This document relates to recreational sports diving. The recommendations given are not aimed at professional divers, including dive masters and instructors who dive recreationally.
- Under Dutch occupational law (ARBO-besluit, H6, Afd 5, Art 6.14) an instructor must be "*physically and mentally capable of recognizing and, if possible, preventing hazards*".
- Divemasters and instructors with psychiatric issues

Table 1
Clinical manifestations of ADHD

Core symptoms	
Attention problems	Easily distracted or bored Difficulty finishing tasks Switching from one activity to another Inability to distinguish between primary and secondary elements Poor planning, organization and decision-making Only able to read for a short while; only able to concentrate if the topic is of great personal interest Difficulty listening and allowing information to sink in Getting lost in the details or being overly precise Endless procrastination Difficulty with filling in forms, understanding instructions, remembering things Indecisiveness Forgetfulness Often loses things Is chaotic Temporary overconcentration or hyperfocus
Hyperactivity	Difficulty sitting still Preoccupied Always having to go and fetch something A feeling of internal restlessness Fidgeting Not able to relax in a calm way Rapid speech
Impulsiveness	Blurts things out Interrupts others Impatience Acts without considering the consequences (spends too much money, gambles, steals, impulsive eating binges, etc.) Starts and ends relationships or jobs impulsively
Mood swings and fits of rage	Short fused Rapidly changing moods in the event of setbacks, up to five times daily

are in principle unsuitable for carrying out diving instruction, but may be deemed suitable for personal recreational sports diving activities based on the current guidelines.

- These guidelines articulate recommendations which are not set in stone. The scientific rationale has limitations and deviation from the recommendations may be justifiable in individual cases.
- The key principle is that the individual diver is primarily responsible for his/her health and safety, as well as the health and safety of his/her buddy.
- The existence of other medical contraindications should be checked for each diver, including the indication for the use of the relevant substances.

Prevalence

ADHD is one of the most common psychiatric disorders in children, with a prevalence estimated to be 4–8%.[10] For a long time, ADHD was thought to be a developmental problem that would resolve automatically with age, but recent research shows that over two-thirds of adults who had childhood ADHD still had the disorder in adulthood.[11] Estimates of the prevalence of ADHD in adults varies from 2.5 to 5%; a meta-analysis reported an average of 2.5%.[12]

Neurodevelopmental disorders

The fifth edition of *Diagnostic and statistical manual of mental disorders* (DSM-5), a classification system of psychiatric disorders developed under the auspices of the American Psychiatric Association,[13] contains a new category of 'neurodevelopmental disorders'; a group of disorders that manifest during the course of an individual's development, often at a very young age. ADHD, amongst others, belong to this group. A diagnosis of ADHD can only be asserted if the symptoms are of such a nature that they cause severe impairment to social, educational or occupational functioning. It is also required to specify the severity level of the ADHD as either mild, moderate, or severe.

Core symptoms

The core symptoms of ADHD are attention or concentration disorders, hyperactivity or (inner) turmoil and impulsive behaviour (Table 1).[14] DSM-5 classifies hyperactivity and impulsiveness as a single symptom criterion; the other symptom criterion being inattentiveness. Attention problems generally become apparent with matters that require prolonged attention and are boring, complex and/or repetitive. Hyperfocus is a specific type of attention problem – an intense form of mental concentration in which attention is focused on a single subject or a task. When someone is hyperfocused, they are oblivious to external stimuli and have little or no concept of time.

Hyperactivity in children is often expressed in a more physical way, whereas adults are usually more 'mentally' preoccupied and complain of inner turmoil and chaos. Impulsiveness is expressed, amongst other ways, by acting before thinking, answering a question before it has been asked in full, starting and ending things like relationships and jobs impulsively. Sensation seeking is a specific form of impulsiveness, seeking excitement and sensation because of a need for new stimuli, variation, excitement and change, as well as making risky decisions.[15]

Comorbidity

Management of ADHD is complex because of the chronicity and the frequent comorbidity of other psychiatric disorders. Mood and anxiety disorders and substance abuse are frequent comorbidities, but also personality, autism spectrum, eating disorders and sleep problems are all reported in association with ADHD. In three-quarters of patients, ADHD is associated with one or more psychiatric disorders.[14]

ADHD and epilepsy

The relationship between ADHD and epilepsy deserves special attention. Children with epilepsy are at increased risk of ADHD.[16,17] Conversely, the relative risk of an epileptic seizure or epilepsy in children with ADHD is two to three times higher than in the general population.[18] Electroencephalographic epileptic abnormalities are also seen more frequently in children with ADHD.[19] There are thought to be different causes for an increased risk of epilepsy in individuals with ADHD, varying from a common genetic defect,[20] to a deficit of the central noradrenergic system.[21] A complicating factor is that all commonly used antiepileptic drugs have some effect on cognitive function, impacting attention and memory. However, the mechanism explaining ADHD/epilepsy comorbidity is still unclear.

Medication

The Dutch guideline "*Richtlijn ADHD bij volwassenen*" (ADHD in adults) recommends methylphenidate and dexamphetamine as the first-line medicinal treatment for ADHD in adults.[22] Both work through blockade of dopamine and noradrenaline reuptake into neurons; furthermore, dexamphetamine causes release of the catecholamines dopamine and noradrenaline. If the effect of these two medications is insufficient, or they cause too many side effects, atomoxetine is the second-line treatment, with less potent bupropion being the third choice. There can be reasons for starting with atomoxetine first, such as parent/child preference, addiction (risk), comorbidity concerns, tics and prior negative experiences with stimulants. Atomoxetine is a non-stimulant medication that works via noradrenergic reuptake inhibition. Bupropion is registered as an antidepressant, it is a weak noradrenergic and dopaminergic reuptake inhibitor.

Modafinil is advised if other treatments have been implemented properly and appear to be ineffective. Little is known about the clinical effectiveness of modafinil as a treatment option for adults with ADHD because there have been only a limited number of adequately controlled studies.[22]

Guanfacine has recently been registered in the Netherlands for treatment of ADHD in children and adolescents and, like clonidine, is an alpha-2 agonist (albeit a little more selective). Its mode of action in ADHD is still unknown, although it is hypothesized that it binds preferentially to postsynaptic alpha 2A receptors and thus stimulates the effect of noradrenaline postsynaptically.[22] Guanfacine and clonidine could be trialled first in adults with ADHD who have a cardiovascular risk (e.g., hypertension) and with tic disorders (stimulants can actually exacerbate tics). The effectiveness of clonidine for ADHD is not included in the guideline for adults because of a lack of scientific evidence and its pronounced side effects.[23]

SIDE EFFECTS OF ADHD MEDICATION[24]

Stimulants (methylphenidate/dexamphetamine)

The most commonly occurring side effects are insomnia, agitation, headache, dry mouth, reduced appetite and tachycardia. Less common side effects include dizziness, anxiety, depression, drowsiness, changes in blood pressure and heartbeat and palpitations. A troublesome problem is the rebound that occurs once the medication has left the system; a temporary increase in ADHD symptoms.

Atomoxetine

The most commonly occurring side effects are headache, drowsiness, reduced appetite, abdominal pain, nausea, vomiting, increased blood pressure and tachycardia. Less frequently mood swings, insomnia, agitation, anxiety, depression and low mood, dizziness, skin rash, fatigue and listlessness have all been reported.

Bupropion

Commonly occurring side effects (10–30%) are insomnia, dry mouth, headache and gastrointestinal symptoms (such as nausea, vomiting, abdominal pain and constipation, particularly at the start of treatment). Less commonly (1–10%) insomnia, dizziness, tinnitus and blurred vision are reported. Anxiety, nervousness and excitability generally occur at the start of treatment and tend to reduce with time. There is an increased risk of seizures with higher doses (450 mg).

Selective serotonin reuptake inhibitors (SSRIs) may cause bleeding complications, though the absolute risk appears to be clinically insignificant in most patients, being clinically relevant only for 'at-risk' patients, e.g., those with thrombocytopenia or platelet disorders, those with coagulopathy and/or in the throes of an acute intracerebral haemorrhage and patients on multiple antiplatelet drugs. Though bupropion is not an SSRI, there may be a risk attached to the combination of bupropion with non-steroidal anti-inflammatory drugs or warfarin.[25]

Modafinil

Headache is common, whilst rare side effects include gastrointestinal symptoms, dry mouth, symptoms of depression, dizziness, drowsiness, palpitations and blurred vision.

Guanfacine

The commonest side effects are somnolence, headache and fatigue. Reduced appetite, depression, anxiety, affective lability, insomnia, sedation, dizziness, lethargy, bradycardia and orthostatic hypotension have all been documented.

CARDIOVASCULAR EFFECTS

The cardiovascular effects of the use of ADHD medication in children and young adults are generally clinically insignificant.[26] However, a clinically relevant and continued increase in blood pressure can occur in individual cases, but extreme values are rare.[26] Thus, blood pressure and heart rate checks should form part of routine monitoring in all ADHD patients on medication.[27] Neither is there evidence of stimulants causing clinically significant electrocardiographic changes.[26] A USA study of over a million children and young adults between the ages of two and 24 years showed no significant increased risk of cardiovascular problems.[28] Two other studies also confirm that ADHD medication is not associated with an increased risk of serious cardiovascular problems.[29,30]

It may be concluded from these various studies that the cardiovascular risks of stimulants is very limited in adults.[23] There are reports of sudden cardiac death but these do not appear to exceed the frequency of such incidents in the normal paediatric population.[31] For safety's sake, a cardiac examination is recommended for children with a family history such as unexplained sudden death in the family before the age of 40 or death as a result of cardiac arrest during exercise and for children with preexisting cardiovascular conditions with potential cardiac symptoms such as syncope, extreme shortness of breath, or prolonged palpitations. It can be useful for the attending physician to refer children in these risk groups to a paediatrician or paediatric cardiologist.

SEIZURES

The *Summary of product characteristics* (SPC) is a specific document required within the European Commission before any medicinal or biocidal product is authorized for marketing. The SPC contains a warning that methylphenidate (and other amphetamines) "*should be used with caution in patients with epilepsy*".[23] Methylphenidate may lower the convulsive threshold in patients with a prior history of seizures, in patients with prior EEG abnormalities in the absence of seizures and rarely in patients without a history of convulsions and no EEG abnormalities. Given the likely fatal outcome of a seizure underwater, and considering the known increased risk of seizure with exposure to elevated oxygen partial pressures, this risk should be addressed in any assessment of diving risk.

The studies in which an increased frequency of seizures were found with methylphenidate suggest that this may occur particularly in patients with uncontrolled epilepsy[32,33] and in patients with abnormal epileptic discharges in the absence of a history of epilepsy.[34] There are few data about seizure risk in non-epileptic children treated with stimulants. A review of the recent literature on the treatment of ADHD with methylphenidate identified nine relevant references.[35] When results from these nine studies are summarized, 92% of 201 participants did not experience an increase in seizure frequency. From these data one may conclude that methylphenidate is relatively safe for use in children with comorbid ADHD and epilepsy. The advice the authors give is to make a careful risk/benefit analysis when considering treatment of ADHD with methylphenidate in children with epilepsy.

In a large cohort study, the risk of seizures in patients aged from six to 17 years treated with either atomoxetine or stimulants for the first time was compared retrospectively (13,398 and 13,322 patients respectively).[36] The risk of seizure was not significantly different between pediatric patients taking atomoxetine compared with those taking stimulants.[36] Higher doses of bupropion, in fact, lower the threshold, but this medication also has few side effects in doses of up to 450 mg.

Hyperbaric conditions

Hardly anything is known about the influence of hyperbaric conditions on ADHD and its treatment. Animal experimentation has shown that the blood-brain barrier becomes more permeable to medications under hyperbaric conditions.[37] There are indications that alcohol and psychotropic drugs increase the risk of nitrogen narcosis [38,39] and acute oxygen toxicity.[40,41]

Discussion

ADHD is a condition that, if untreated, is associated with symptoms that may present a risk to scuba diving, such as attention problems, reduced awareness and impulsiveness. The argument that concentration underwater is particularly good is often presented by the diver, the parents or the diving instructor during a psychiatric evaluation to establish if the diver is fit to dive or not. However, over-concentration or hyperfocus means concentration is so intense at times that awareness of the external world is lost. Concentration is determined by stimuli from the environment and the task that has to be done at a certain moment. People with ADHD are easily distracted by stimuli from the environment, with the result that they have great difficulty staying focused. During diving this can result in ignoring important tasks such as maintaining buddy contact, monitoring depth and gas supply. Also, the need for excitement can lead to poor or risky decision-making since risk prevention is of particular importance in scuba diving. Since comorbidities are common in ADHD, this poses an additional risk to diving, whilst the effects of medications and their side effects are unpredictable and/or may worsen nitrogen narcosis.

Severity of ADHD can vary from one person to another, but there is also situational variability (home, school, work, sport). Some children and adults are capable of suppressing their 'ADHD behaviour' effectively but it is harder for many children to suppress this behaviour.[42] If the severity of the condition (as specified in DSM-5) is mild, then ADHD does not need to present grounds for declaring the diver unfit for recreational diving.

Communication between the different areas of the brain is still far from optimal in adolescents. Rapid maturation of some regions of the brain, coupled with the slower maturation of others, explains many typical adolescent behaviours like acting impulsively, taking risks and mood swings. Executive functions including cognitive flexibility and working memory improve further during adolescence; in other words, young age is an additional risk factor. The sports diving physician could consider assessing a child with very mild symptoms, who is capable of controlling their behaviour effectively and where there are no complicating factors, as fit to dive with compressed air. When in doubt, the sports diving physician in the Netherlands is advised to seek a second opinion from a psychiatrist specialising in diving medicine.

The side effects of stimulants and atomoxetine, whilst diverse, are generally mild and well tolerated so may not in themselves be a contraindication to scuba diving. Rebound is incompatible with diving as it exacerbates ADHD symptoms. Depending on the medication used and the time it is taken, as a rule the medication will have left the system by late in the day, with a possible return of ADHD symptoms. For this reason, evening or night-time diving is not recommended. Somnolence, a frequent side effect of guanfacine, makes this agent less suited to diving with compressed air.

Medical recommendations for diving with ADHD

- Diving is allowable if symptoms are mild.
- No significant comorbidity is present.
- Individual has insight into and understanding of the condition.
- Age: The Dutch Association of Diving Medicine sets the age limit for responsible diving practice at 14 years, preferably 16 years. For divers with ADHD an age of 18 years and older is recommended, unless there are reasonable grounds for the attending physician (preferably in consultation with a psychiatrist specialising in diving medicine) to advise otherwise.
- Medication:
  i. No significant side effects.
  ii. Only a single psychotropic medicine: more than one psychotropic medicine will increase the risk of potentially dangerous side effects whilst diving and susceptibility to nitrogen narcosis.
  iii. Medication and therapy compliant.
  iv. Maximum diving depth 18–20 m: a maximum diving depth is advised to minimize the risk of DCI and the slight theoretical risk that some drugs might increase narcosis.
  v. No combination with psychostimulants and epilepsy.
- Dizziness and paraesthesiae are side-effects that could mimic DCI. Whilst not necessarily a contraindication to use, such symptoms should be considered during an evaluation for diving.
- Diving is not advised in the event of rebound.

Who carries out the examination?

The recommendation to the Dutch Association for Diving Medicine is that, with the use of these guidelines, certified sports diving physicians should be able to arrive at a well-considered opinion on whether a diver should be deemed fit or unfit for diving with compressed air. In case of doubt or a complicated underlying condition, a psychiatrist/sports diving physician may be asked to carry out a specialist examination.

Conclusions

ADHD is a condition which, if untreated, is associated with

symptoms such as attention problems, reduced awareness and impulsiveness that present a risk to scuba diving. Mild ADHD need not preclude diving with compressed air. Impulsivity, risky behavior and mood swings are typical adolescent behaviours which pose an extra risk for children with ADHD because communication between the different areas of the brain is still far from optimal.

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Technical report

The performance of 'temperature stick' carbon dioxide absorbent monitors in diving rebreathers

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Key words
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Abstract
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Introduction: Diving rebreathers use canisters containing soda lime to remove carbon dioxide (CO_2) from expired gas. Soda lime has a finite ability to absorb CO_2. Temperature sticks monitor the exothermic reaction between CO_2 and soda lime to predict remaining absorptive capacity. The accuracy of these predictions was investigated in two rebreathers that utilise temperature sticks.
Methods: Inspiration and rEvo rebreathers filled with new soda lime were immersed in water at 19°C and operated on mechanical circuits whose ventilation and CO_2-addition parameters simulated dives involving either moderate exercise (6 MET) throughout (mod-ex), or 90 minutes of 6 MET exercise followed by 2 MET exercise (low-ex) until breakthrough (inspired PCO_2 [P_iCO_2] = 1 kPa). Simulated dives were conducted at surface pressure (sea-level) (low-ex: Inspiration, n = 5; rEvo, n = 5; mod-ex: Inspiration, n = 7, rEvo, n = 5) and at 3–6 metres' sea water (msw) depth (mod-ex protocol only: Inspiration, n = 8; rEvo, n = 5).
Results: Operated at surface pressure, both rebreathers warned appropriately in four o five low-ex tests but failed to do so in the 12 mod-ex tests. At 3–6 msw depth, warnings preceded breakthrough in 11 of 13 mod-ex tests. The rEvo warned conservatively in all five tests (approximately 60 minutes prior). Inspiration warnings immediately preceded breakthrough in six of eight tests, but were marginally late in one test and 13 minutes late in another.
Conclusion: When operated at even shallow depth, temperature sticks provided timely warning of significant CO_2 breakthrough in the scenarios examined. They are much less accurate during simulated exercise at surface pressure.

Introduction

A closed circuit rebreather is a type of underwater breathing apparatus that recycles expired gas through a carbon dioxide (CO_2) absorbent and incorporates a gas addition system designed to maintain both a safe inspired pressure of oxygen (P_iO_2) and an appropriate mix of diluent gases. They are popular with so-called 'technical divers' and scientific divers performing deep and/or long dives because the recycling of expired breath markedly reduces use of expensive gases such as helium, and maintenance of a constant optimal P_iO_2 increases decompression efficiency.[1]

There are several forms of CO_2 absorbent, but the most commonly used is soda lime; a granular compound containing calcium hydroxide, water and sodium hydroxide. This is packed in a canister (often referred to as a 'scrubber') through which the exhaled gas is passed. Soda lime has a finite capacity for absorbing CO_2 and, if this capacity is exceeded, CO_2 will 'break through' the scrubber and its re-inhalation by the diver may lead to dangerous hypercapnia. Therefore, the soda lime must be replaced in a timely fashion. Rebreather manufacturers provide guidelines on scrubber canister duration, based on tests conducted under demanding conditions with high simulated CO_2 production

and low water temperature, which divers may consider to be conservative. Anecdotally, this often results in divers using soda lime for longer than recommended based on their previous experience and best guesses on expected duration.

In an attempt to bring some objectivity to determining safe duration of use of soda lime, several manufacturers have incorporated so-called 'temperature sticks' into the scrubber canister to monitor the exothermic reaction between CO_2 and soda lime. These devices are comprised of an array of thermistors that pass through the soda lime bed, and they apply proprietary algorithms to interpret the distal movement of the reaction as it progresses through the canister while proximal exhausted soda lime cools. Proximal in this context refers to the end of the scrubber canister where the exhaled gas enters. Two very popular rebreathers utilising temperature sticks are the Inspiration™ rebreather (Ambient Pressure Diving, Helston, Cornwall, UK), and the rEvo™ rebreather (rEvo Rebreathers, Brussels, Belgium).

The Inspiration rebreather control display notionally depicts the temperature profile in the soda lime bed as a bar that turns from clear to black as the scrubber heats up early in the dive, and then progressively (in six steps from proximal to distal) turns from black to clear as the reaction decreases. When the display has only one black step left, which has been designed to occur prior to a P_iCO_2 of 0.5 kPa, the diver receives a warning. The display bar is designed to become completely clear prior to a P_iCO_2 of 1 kPa, at which point the diver is advised to 'bail-out' off the rebreather and onto an open-circuit gas supply.

Soda lime in the rEvo is divided into two smaller separate canisters connected in series by a short conduit. Each canister has its own temperature stick. This configuration facilitates a cycling regimen between shorter dives whereby the proximal heavily used canister is discarded, the less consumed distal canister is moved into the proximal position and a new canister is placed in the distal position. The idea is to avoid discarding an entire canister containing a lot of unconsumed soda lime after a short dive. The temperature stick algorithm counts down a time (in minutes) to the point beyond which cycling (as above) is no longer considered appropriate. If the dive duration exceeds this cycling time threshold, then the two scrubbers are treated as one and the algorithm counts down a "*remaining scrubber time*" in minutes.

This presentation of information that is analogous to a CO_2 scrubber 'fuel gauge' inevitably invites the diver to interpret the data literally, and to base important decisions about conduct of the dive on the temperature stick. This requires that the temperature stick predictions of remaining scrubber life are reasonably accurate in the majority of plausible scenarios. Other than a reference to "*experimentally determined calibration*" in the patent describing the rEvo temperature stick[2] and an abstract alleging successful development of the same device,[3] no data could be found in the public domain describing the accuracy of these devices. Therefore, the ability of these rebreathers to predict CO_2 breakthrough was tested. The question in respect of both the Inspiration and rEvo devices was: would the temperature stick warn the diver prior to significant CO_2 breakthrough during simulated dives?

Methods

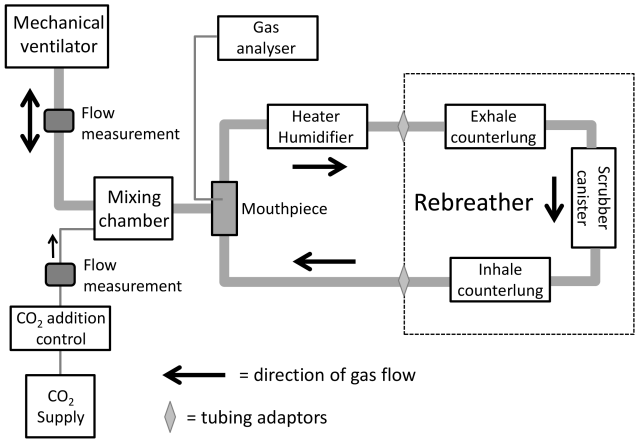
Those aspects of the protocol requiring human participation were approved by the University of Auckland Human Participation Ethics Committee (Reference 015280). This was a laboratory study in which an Evolution Plus™ (a rebreather model in the Inspiration range, henceforth referred to simply as the Inspiration) and a rEvo (standard model) rebreather were operated in a test circuit designed to simulate resting and exercising dives. Thus, in a preliminary phase of this study (described in more detail previously[4]) indicative values for respiratory minute ventilation (V_E), tidal volume (T_V), respiratory rate (RR), oxygen consumption (VO_2), and CO_2 production (VCO_2) were established in a working subject at the chosen exercise intensity.

A recent consensus on functional capacity for diving activity identified continuous exercise at 6 MET as a desirable and plausible target for sustained exercise output in a diver.[5] One MET [the approximate metabolic rate of an individual at rest] equals an assumed oxygen consumption of 3.5 $mL \cdot kg^{-1}$ body $weight \cdot minute^{-1}$ (min). Therefore, to establish the ventilation and CO_2 addition parameters for the benchtop tests our human participant exercised at 6 MET on an electronically braked cycle ergometer whilst breathing on the Inspiration rebreather in dry conditions. At steady state V_E was 44 $L \cdot min^{-1}$ (T_V = 2.0L, RR = 22 $breaths \cdot min^{-1}$) and VCO_2 was 2.0 $L \cdot min^{-1}$, actual temperature and pressure dry (ATPD).

SURFACE PRESSURE MECHANICAL TEST CIRCUIT

The initial studies were conducted at the University of Auckland, New Zealand. The ambient pressure for all New Zealand trials was at sea level (surface pressure), chosen of necessity because no pressure testing facility was available. In these studies, the inspiratory and expiratory hoses of the rebreather were attached to a test circuit (Figure 1). The test circuit was composed of 35 mm (internal diameter) smooth-bore respiratory tubing (MLA1015, AD Instruments, Dunedin, New Zealand) connected to a one-way respiratory valve (5710, Hans Rudolph, Shawnee, KS, USA) which simulated the rebreather mouthpiece. A port in the valve allowed continuous sampling of the inspired and expired gas for infrared analysis of inspired and end-tidal PCO_2 (ML206 Gas Analyser, AD Instruments, Dunedin, New Zealand). A clinical heater-humidifier (Fisher and Paykell Medical, Auckland, New Zealand) was incorporated into the exhale hose of the circuit to reproduce the heating and humidification of expired gas that would occur with a human breathing on the loop. The heating function was set to 34°C for all experiments.

Figure 1
Schematic layout of the surface pressure test circuit and monitoring equipment; (see text for explanation)



Breathing was simulated using a sinusoidal mechanical ventilator (17050-2 Lung Simulator, VacuMed, Ventura, CA, USA) with an inspiratory-expiratory ratio of 1:1. The T_V was set at 1.5 L and the RR at 30 breaths·min^{-1} for the 6 MET experiments. These parameters differed slightly from the derived human values described above (T_V 2.0 L, RR 22 breaths·min^{-1}) because the ventilator struggled with the work of moving gas around this circuit with a T_V of 2.0 L. Accurate ventilation was ensured through independent monitoring with a pneumotachograph (800 L, Hans Rudolph, Shawnee, KS, USA).

The ventilator was connected to the circuit one-way valve via a 4 L mixing chamber where the inspired and expired gas mixed with instrument grade CO_2 introduced at 2 L·min^{-1} ATPD using a precision flow pump (R-2 Flow Controller, AEI Technologies, Pittsburgh PA, USA) drawing from a Douglas bag reservoir. The CO_2 flow was also independently monitored to ensure accuracy using a flow transducer (MLT10L, AD Instruments, Dunedin, New Zealand).

Sofnolime 797™ (Molecular Products, Essex, UK) was used in both rebreathers for all experiments. All Sofnolime was newly purchased, in date, and stored in the manufacturer-supplied sealed containers before use. The Sofnolime was precisely weighed (2.64 kg for the Inspiration scrubber, and 1.35 kg for each of the two rEvo canisters) (GM-11, Wedderburn Scales, Auckland, New Zealand) prior to canister packing. Each new scrubber canister was packed approximately 15 min before the start of an experiment.

In all tests the rebreathers were immersed in water at room temperature (19°C), chosen as a matter of convenience. Although water temperature is known to affect scrubber duration, there are no data on how it may affect temperature stick performance, and any water temperature within the range frequented by divers is operationally relevant.

SURFACE PRESSURE TEST PROTOCOL

The circuit was tested for leaks by holding a positive pressure. The rebreather was switched on and the default surface PO_2 set point of 0.7 atmospheres (atm) was chosen for the Inspiration. The rEvo was operated with the oxygen addition system switched off because this unit has a constant mass flow oxygen addition system and with no actual oxygen consumption occurring this resulted in gas accumulation and over-pressure of the circuit. An easily exceeded surface PO_2 set point of 0.19 atm (19 kPa) was used to avoid constant hypoxia alarms. The diluent gas was air for all experiments. Ventilation of the circuit was initiated and, after appropriate operation was confirmed, a timed trial started with the continuous addition of CO_2 at 2.0 L·min^{-1} ATPD. Every 30 min the ventilation and CO_2 addition were briefly paused (approximately one min) to recalibrate the CO_2 flow and infrared sensors and to remove any excess moisture from the circuit hoses. These pauses did not elicit any alarms or obvious changes in the temperature stick display (Inspiration) or remaining scrubber time (rEvo).

For each rebreather we ran tests on two protocols. The first was designed to emulate the exercise and ventilation pattern of typical long dives where there would usually be moderate exercise initially followed by a long period of low exercise during decompression. Thus, the rebreathers ($n = 5$ for each model), each containing a newly packed soda lime scrubber, were run on 6 MET parameters (described above) for 90 min (half the Inspiration's expected scrubber life before breakthrough when operated at 6 MET),[4] followed by 2 MET parameters (ventilation 16.5 L·min^{-1} [Tv 1.5 L;, RR = 11 breasths·min^{-1}], VCO_2 = 0.67 L·min^{-1}) until the P_iCO_2 rose to 1 kPa; a P_iCO_2 that is considered dangerous,[6] and after which the rise in CO_2 is generally extremely rapid.

The second protocol was designed to emulate the less plausible scenario of continuous moderate exercise throughout a dive. Thus the rebreathers (n = 6 for the Inspiration and n = 5 for the rEvo) were run on the 6 MET parameters continuously until the P_iCO_2 rose to 1 kPa. Throughout the tests, the decay was noted of the six segments on the Inspiration temperature stick display and recorded the remaining scrubber time (at 10 min intervals) displayed by the rEvo. The primary endpoint in each test was whether the rebreather warned the diver (decay to one segment on the Inspiration and counting down to zero time remaining on the rEvo) prior to reaching breakthrough at 1 kPa.

HYPERBARIC TEST CIRCUIT

After some results of the surface pressure tests were found to be discordant with manufacturer tests conducted under pressure (Martin Parker, personal communication, December 2016), we elected to repeat the continuous moderate exercise tests in both rebreathers at elevated ambient pressure at the

Figure 2
The ANSTI underwater breathing apparatus test system. The pressure vessel is in the centre. The pressure control, ventilation and heater/cooler systems are on the right of the pressure vessel, and the monitoring system is on the left

Swedish Armed Forces Diving and Naval Medicine Centre at Karlskrona. The same scrubber and temperature stick units used in the surface pressure experiments (both rebreathers) were employed here. In these studies, the rebreather was connected to an ANSTI machine test circuit.[7] The ANSTI machine is a purpose-built underwater breathing apparatus test station (Figure 2) that allows mechanical ventilation with heated and humidified gas, and precise CO_2 addition to an immersed rebreather under pressure.

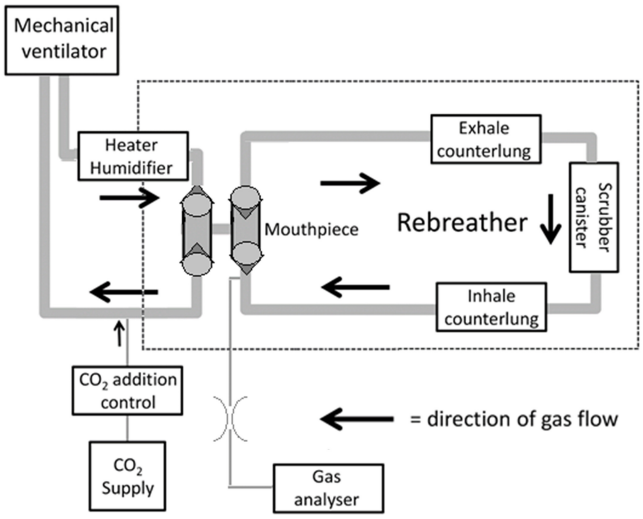
The laboratory environment was maintained at 20°C and 35–45% relative humidity. As in the surface pressure circuit, CO_2 was precisely introduced to the ANSTI machine ventilation system at 1.86 L·min^{-1} standard temperature and pressure dry (STPD) giving a volume of 2 L·min^{-1} at ATPD via a mass flow controller (Brooks Instrument 0-5 L·min^{-1} CO_2, Hatfield PA, USA) such that it entered the exhale hose of the rebreather loop as it would during use by a diver (Figure 3). Gas from the rebreather inhale hose was sampled at 250 mL·min^{-1} for continuous analysis in an infrared CO_2 analyser (Servomex 1440 D, Crowborough, UK). This sampled gas was replaced, and rebreather loop volume preserved during compression to elevated pressures, by allowing the rebreathers' automatic diluent addition valves to add air into the rebreather circuit.

The experiments were identical to the surface pressure tests with respect to rebreather configuration, ventilation parameters, expired gas heating and humidification, water temperature and soda lime management (see above). As in the surface pressure experiments throughout each test there was periodic two-point calibration of the inspired CO_2 analyser using reference gases, and independent calibration of the CO_2 inflow rate (DryCal Definer 220, Butler NJ, USA).

HYPERBARIC TEST PROTOCOL

The set up and oxygen management in each rebreather was as described for the surface pressure studies, except that the

Figure 3
Schematic layout of the ANSTI breathing test circuit and monitoring equipment; (see text for explanation)



rEvo would not accept a PO_2 set point of 0.19 atm at depth and the 0.7 atm (71 kPa) set point for the Inspiration was unacceptably high for safe operation of the ANSTI circuit. Therefore, a set point of 0.5 atm (50.6 kPa) was used for both rebreathers. The rEvo was run with the oxygen addition system switched off so that the constant oxygen flow would not disturb the measurements, and the hypoxia alarm was cancelled when it was active.

For each experiment the rebreather was secured in the ANSTI test chamber and immersed while being ventilated to check for leaks. The test chamber lid was then closed and the chamber pressurised to the chosen depth. Because the hyperbaric studies were being performed in response to the finding of suboptimal temperature stick performance at the surface (Figures 5 and 6), we ran the hyperbaric experiments at the shallowest depths that are nevertheless of undisputed relevance to divers during decompression (3 or 6 metres' sea water (msw)). Similarly, because the temperature sticks had performed well on the low exercise protocol but failed on the moderate exercise protocol at surface pressure, we only performed the hyperbaric studies in Sweden on the moderate exercise protocol.

Two Inspiration scrubber canisters were available (thus two different temperature sticks: stick A that had been used in the surface pressure experiments, and stick B, not previously used in our work). Two tests were run using each stick at 3 and 6 msw; a total of eight Inspiration tests. Five tests were run with the rEvo; three at 3 msw and two at 6 msw. Finally, in order to corroborate our previous finding of temperature stick failure during moderate exercise at surface pressure (sea level) one test was run with the Inspiration (stick A as previously used at surface pressure) immersed in the ANSTI machine but without pressurising the test chamber.

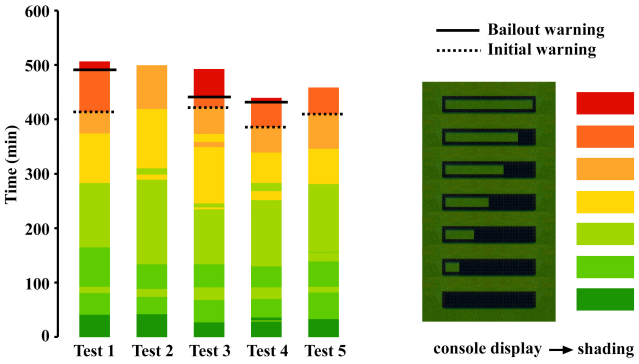
Table 1

The remaining scrubber time (RST) (minutes) displayed by the rEvo rebreather at the point of CO_2 breakthrough to a P_iCO_2 of 1 kPa in the low and high exercise tests conducted at surface pressure; a negative offset is the time elapsed between zero time remaining on the scrubber monitor and the actual time of breakthrough to P_iCO_2 = 1 kPa, and represents early warning; a positive offset is the time remaining on the scrubber monitor at the actual time of breakthrough to P_iCO_2 = 1 kPa, and represents a late warning; Zero offset means that the remaining time of the scrubber monitor fell to zero at exactly the same time as breakthrough to P_iCO_2 = 1 kPa

Condition	Low exercise tests						Moderate exercise tests				
Test number	1	2	3	4	5		1	2	3	4	5
RST at P_iCO_2 = 1 kPa	0	15	0	0	0		15	75	4	0	25
Offset (minutes)	-45	15	-57	-18	-63		15	75	4	0	25

Figure 4

Changes in the Inspiration temperature stick display over the course of each low exercise test conducted at surface pressure. Each bar represents a separate test; the top of the bar represents the time (y axis) of breakthrough to a P_iCO_2 of 1 kPa; the coloured shading represents the appearance of the temperature stick display according to the key. Note that the dark green segment at the base of each bar represents both the time taken for the stick display to become completely black signifying heat throughout the soda lime bed, and the time it remained completely black. The timing of both alarm conditions is shown (initial warning = dotted line occurring when one black segment remains, and bailout warning = solid line occurring when no black segments remain)



Temperature stick data from both rebreathers were recorded as described for the surface pressure studies.

THERMISTOR EVALUATION

After small but consistent differences were found in the performance of the two Inspiration temperature sticks (Figure 7), the readings obtained from the nine thermistors arrayed in each temperature stick were compared under carefully controlled temperature conditions. The two sticks were placed in a climate chamber (T-70/1000, CTS GmbH Hechingen, Germany), and the temperature reading of each thermistor noted after 30 minutes' stabilisation at 5ºC and 50ºC. Similarly, each stick was placed in a heated water bath and stabilised at a fixed temperature measured with a digital thermometer (Fluke 51, Fluke Corporation Everett, USA). The temperature reading of each thermistor was noted after five minutes' stabilisation.

Results

SURFACE PRESSURE TESTS

Both rebreather temperature sticks warned prior to significant breakthrough (P_iCO_2 = 1 kPa) in four of the five low-exercise tests conducted at surface pressure. The changes in the Inspiration temperature stick display over the course of each test are depicted in Figure 4. The time remaining on the rEvo scrubber monitor at the point of CO_2 breakthrough in each test is shown in Table 1.

In contrast, both rebreathers' temperature sticks failed to warn prior to significant CO_2 breakthrough in the moderate exercise tests conducted at surface pressure (Table 1 for the rEvo and Figure 5 for the Inspiration results, respectively). In testing of the rEvo, a lack of linearity was noted in the remaining scrubber time estimation which was over-estimated early in the test, then declined faster than real time later (Figure 6).

HYPERBARIC TESTS

Both rebreather temperature sticks performed substantially better on the constant moderate-exercise protocol when operated at pressure. There was no discernible difference in performance between 3 and 6 msw. The changes in the Inspiration temperature stick display over the course of eight tests are depicted in Figure 7.

Whereas the Inspiration temperature stick had failed to warn before breakthrough to P_iCO_2 = 1 kPa on any of six continuous moderate-exercise tests at atmospheric pressure, it warned before or soon after breakthrough in all the tests under pressure. However, there was a difference between the two sticks tested. The accuracy of Stick A in precisely predicting and defining breakthrough was remarkable. The P_iCO_2 data are not presented here, but in every test Stick A initially warned just prior to breakthrough to P_iCO_2

Figure 5

Changes in the Inspiration temperature stick display over the course of each moderate exercise test conducted at surface pressure; note the much shorter duration of each test in comparison with the low exercise tests in Figure 4; interpretation of the figure is otherwise as described as for Figure 4; none of the runs reached the alarm condition (1 black segment remaining) prior to P_iCO_2 = 1 kPa

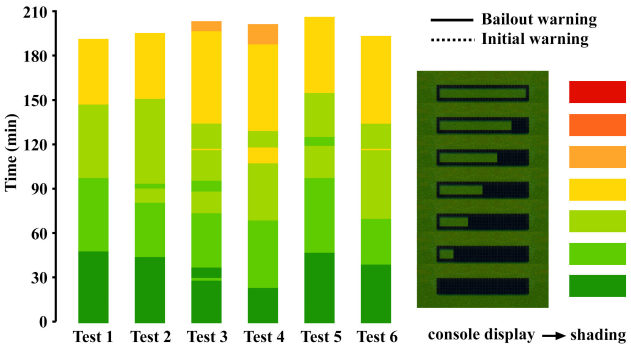
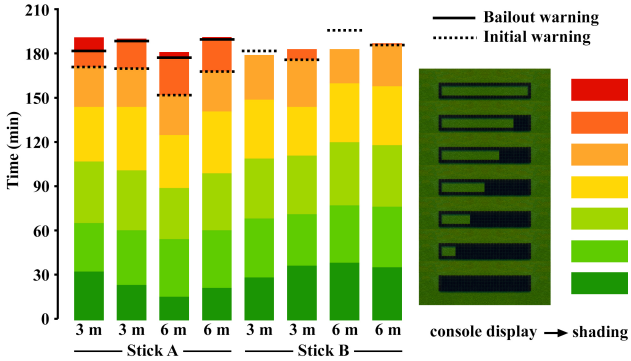


Figure 7

Changes in two Inspiration temperature stick displays (designated A and B) over the course of eight moderate exercise tests conducted at 3 and 6 msw as indicated; interpretation of the figure is otherwise as described for Figure 4

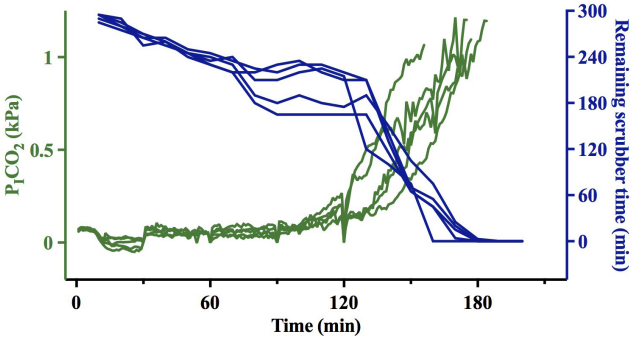


= 0.5 kPa, and then recommended bailout just prior to breakthrough to P_iCO_2 = 1 kPa. In contrast, Stick B gave warnings just prior to breakthrough to P_iCO_2 = 1 kPa in two tests, and 3 min after in one. The warning came 13 min after breakthrough in a fourth test (Figure 7). In contrast to the above results, in the single test performed using the Inspiration rebreather and Stick A in the ANSTI machine at surface pressure (data not shown) we recorded exactly the same failure to provide any warning prior to breakthrough to P_iCO_2 = 1 kPa as seen in the previous moderate-exercise tests at surface pressure.

The time remaining on the rEvo scrubber monitor at the point of CO_2 breakthrough in each test is shown in Table 2. Toward the end of several rEvo tests problems with moisture from the rebreather circuit entering the gas sampling line were experienced, and it was not possible to run every test through to a breakthrough of P_iCO_2 = 1 kPa. We did, however, get

Figure 6

Remaining scrubber time (blue lines) and $PiCO_2$ over the course of the five moderate exercise tests at surface pressure using the rEvo rebreather; time remaining predictions are non-linear



to P_iCO_2 = 0.5 kPa in all tests. We thus report 0.5 kPa as an alternative endpoint. In fact, our primary question was answered in the absence of continuing to a breakthrough of P_iCO_2 = 1 kPa because the remaining scrubber time had declined to zero prior to P_iCO_2 = 0.5 kPa in every test (see Table 2). As with the Inspiration, this result contrasted markedly with the rEvo temperature stick's failure to warn of breakthrough in four of five moderate-exercise tests conducted at surface pressure. We also noted that although there remained a minor tendency for the rEvo to report overly-optimistic remaining scrubber time estimations early in the dive, the decline in estimated time to zero was much more linear in the tests conducted under pressure (Figure 8).

The comparison of the temperature readings obtained from the nine thermistors on each of the two Inspiration temperature sticks (designated A and B respectively) in both the climate chamber and water bath evaluations are shown in Tables 3 and 4.

Discussion

Hypercapnia in diving may arise from either failure by the diver to ventilate adequately or from rebreathing of CO_2, or a combination of both.[8] The potential to rebreathe CO_2 is important in the use of rebreathers which rely on soda lime to remove CO_2 from the expired gas. Soda lime has a finite life and must be replaced in a timely fashion or expired CO_2 will break through the soda lime canister and be rebreathed. Temperature sticks represent an attempt to indirectly confirm CO_2 removal by measuring reactivity in the soda lime canister during a dive. This study evaluated the reliability of these devices in warning the diver prior to significant CO_2 breakthrough as soda lime became exhausted under two test conditions. The first simulated the work rate and respiratory parameters of a notional long decompression dive with moderate exercise early in the dive, followed by less activity during a long decompression when the soda lime would often be nearing the limits of its absorptive capacity. The second protocol involved moderate exercise throughout

Table 2
The remaining scrubber time (RST) (minutes) displayed by the rEvo rebreather at the point of CO_2 breakthrough in the moderate exercise tests conducted at 3 and 6 msw; a negative offset is the time elapsed between zero time remaining on the scrubber monitor and the actual time of breakthrough to P_iCO_2 specified and represents early warning

Depth (msw)	**3**				**6**	
Test number	1	2	3		4	5
RST at P_iCO_2 = 0.5 kPa	0	0	0		0	0
Offset (minutes)	-46	-36	-22		-40	-22
RST at P_iCO_2 = 1 kPa	–	0	–		0	–
Offset (minutes)	–	-60	–		-61	–

Table 3
Temperature readings from the nine individual thermistors (designated T0 – T8) on two Inspiration temperature sticks (designated A and B) recorded at 5 and 50ºC in a climate chamber

Thermistor number	**T0**	**T1**	**T2**	**T3**	**T4**	**T5**	**T6**	**T7**	**T8**
Stick A @ 5°C	4.5	6.6	5.0	5.5	5.5	5.0	5.5	6.0	6.0
Stick B @ 5°C	4.5	4.8	4.5	4.5	4.5	4.0	4.5	5.0	5.0
Stick A @ 50°C	49.0	50.9	49.0	49.0	49.0	48.0	48.5	48.5	48.0
Stick B @ 50°C	49.0	49.3	49.0	49.0	49.0	48.5	48.5	48.5	48.5

Table 4
Temperature readings from the nine individual thermistors (designated T0–T8) on two Inspiration temperature sticks (designated A and B) recorded at fixed temperatures in a water bath

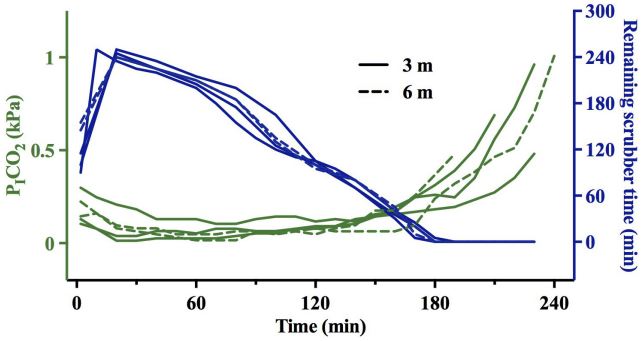
Thermistor number	**T0**	**T1**	**T2**	**T3**	**T4**	**T5**	**T6**	**T7**	**T8**
Stick A @ 32.5°C	32.5	34.4	32.5	33.0	33.0	32.5	33.0	33.0	32.5
Stick B @ 33.1°C	31.5	31.9	31.5	31.5	31.5	31.0	31.5	31.5	29.5

the life of the scrubber. It should be made clear that the latter is a less plausible real-world scenario than the former, but it was purposely chosen as a relevant scenario thought likely to provoke failure in temperature stick predictions. Based on these results, the following observations about temperature sticks are offered.

Firstly, there was a substantial improvement in accuracy when tests were conducted at even shallow depths compared to surface pressure. It is notable that, in the process of following up on the results of the surface pressure tests, the manufacturer of the Inspiration rebreather also found less accuracy when conducting an ANSTI machine test on the moderate-exercise protocol at surface pressure (Martin Parker, personal communication, July 2017). It seems clear that even small elevations of ambient pressure are an important requirement for accurate function of temperature sticks. The basis for this effect of depth was not established. An explanation is both beyond the scope of this work and inconsequential to answering the current research question. It could, however, form the basis for further research.

Secondly, based on the reasonably good performance of both rebreathers' temperature sticks during the low-exercise protocol even at surface pressure (appropriate warnings occurred prior to significant breakthrough in four of five tests in both rebreathers) together with the finding of markedly improved accuracy at shallow depths compared to surface pressure, it is predicted that both rebreathers tested will reliably provide warnings prior to significant CO_2 breakthrough in typical long decompression dives where the diver is at rest in shallow, temperate water toward the end of scrubber life. One can feel confident in this prediction for conditions conforming to those of the study tests, but it must be acknowledged that the scrubbers had not been exposed to typical dive depths early in each test and that variations

Figure 8
Remaining scrubber time (blue lines) and P_iCO_2 over the course of the five moderate exercise tests conducted under pressure using the rEvo rebreather; time remaining predictions are more linear than when the rebreather was operated at surface pressure (Figure 6)



in other conditions such as water temperature could affect temperature stick performance.

Thirdly, both rebreathers performed surprisingly well in the much more provocative continuous moderate-exercise protocol when tests were conducted at depth, though both exhibited different vulnerabilities.

There was a difference in performance between the two Inspiration temperature sticks with one (Stick A) providing precisely timed and accurate warnings before significant breakthrough on all four tests, and the other (Stick B) providing appropriate warnings on two occasions, a marginally late warning on one occasion, and a warning 13 min late on another (Figure 7). The comparison of temperature measurements in the thermistor arrays of the two sticks did reveal some subtle differences in accuracy (Tables 3 and 4) which might explain their different behaviour, but one cannot be certain about this. More detailed investigation, which would include consideration of the dynamic nature of the responses, is beyond the scope of this study.

The rEvo temperature stick provided warnings prior to significant breakthrough on all the moderate exercise tests, but these warnings came an hour before our experimental end-point of 1 kPa of inspired CO_2, and could perhaps be interpreted as too conservative. On the other hand, if the goal is to warn before a lower pressure of inspired CO_2 (such as 0.5 kPa)[6] then the decline in "*remaining scrubber time*" to zero seems substantially less premature (Figure 8) with negative offsets between 22 and 46 min (Table 2). There was also a small degree of non-linearity in the time remaining predictions, with optimistic predictions early in the simulated dive and a subsequent decline that was faster than real time. These observations on both temperature sticks must be interpreted within the context of the experiment in which they were made; that is, a sustained exercise test scenario that was considered likely to provoke failure and which is relatively less plausible in real-world technical decompression diving.

Fourthly, the failure of both temperature sticks during the moderate exercise protocol tests conducted at surface pressure is potentially relevant to surface swimming at the end of a dive while breathing on the rebreather loop. Although the consequences of a hypercapnic event at the surface are likely to be much less serious than one occurring at depth, divers should nevertheless be aware that a temperature stick may not provide accurate data during a vigorous surface swim conducted near the end of scrubber life.

An obvious limitation of this study is the relatively small number of tests with the various temperature sticks in the different conditions, and the limited range of conditions tested. There are other scenarios such as deeper depths, colder and warmer water temperatures, use of different gases, and different patterns of exercise and rest in which temperature stick performance could be evaluated and might be different. This work was challenging and time consuming, and the effect of any variation in conditions requires multiple confirmatory repetitions. Thirty-five tests are reported in this paper; and each test took four to eight hours to complete depending on whether it addressed moderate or lower exercise, respectively.

It is germane to state that temperature sticks do not actually measure CO_2 and are not capable of detecting or predicting CO_2 rebreathing that occurs as a result of exhaled gas bypassing the scrubber bed, or abnormally channelling through it for some reason. Therefore, divers should adopt a holistic approach to appraisal of scrubber performance during diving and not consider temperature stick predictions to be immutably correct, especially in the face of symptoms that might suggest hypercapnia.

Conclusions

These data represent the first publicly reported demonstration that temperature sticks can reliably warn indirectly of CO_2 breakthrough before it occurs during simulation of a common rebreather diving scenario (resting decompression in 19°C temperate water). This was usually also true even during moderate exercise at shallow depths; conditions which, based on our tests at surface pressure, we incorrectly predicted would significantly confound temperature stick accuracy. However, despite this positive result, one cannot draw confident conclusions about temperature stick performance in conditions beyond those tested in this study. The possibility cannot be excluded that factors such as colder or warmer water, greater levels of exercise, greater pressures and different gases may change their accuracy.

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Conflicts of interest

Simon Mitchell and Neal Pollock are members of the Editorial Board of *Diving and Hyperbaric Medicine*, but had no input into the peer review or decision-to-publish processes.

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Case reports

Lion's mane jellyfish (*Cyanea capillata*) envenoming presenting as suspected decompression sickness

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Key words
Marine animals; Envenomation; Clinical toxicology; Diving incidents; Case reports

Abstract

(Trevett A, Sheehan C, Wilkinson A, Moss I. Lion's mane jellyfish (*Cyanea capillata*) envenoming presenting as suspected decompression sickness. Diving and Hyperbaric Medicine. 2019 March 31;49(1):57–60. doi: 10.28920/dhm49.1.57-60. PMID: 30856668.)

Lion's mane jellyfish stings are usually characterised by local inflammation, especially weals. Systemic symptoms are not widely described although there is a well known fictional description of a fatal reaction to envenoming. We describe five divers presenting with suspected decompression sickness, where the probable diagnosis was jellyfish envenoming.

Introduction

Lion's mane jellyfish (LMJ, *Cyanea capillata*) occur widely throughout boreal waters (Figure 1).[1] They are the largest jellyfish known and can have tentacles extending as much as 30 metres from the bell, which can be up to a metre across. The tentacles are covered with nematocysts which contain toxin. Lion's mane jellyfish have been identified with more than a thousand tentacles, each of which can have up to a quarter of a million nematocysts.[2]

In Orkney waters, it is relatively common, particularly in the summer months, for divers to come into contact with tentacles of LMJ either in the water or on shot lines. This can result in stings occurring around the regulator or in nematocyst deposition on dry suits which may result in toxin coming into contact with the diver's skin on de-kitting. In most cases the result of this contact is trivial although may result in local weals, erythema or swelling. Debate continues about the most effective local treatment for contact lesions with most convincing evidence suggesting local heat[3,4] whilst others recommend ice or baking soda.[5,6] Systemic symptoms are thought to be rare after envenoming although nausea, sweating, abdominal pain and muscular cramps have been described.[7] Autonomic neurotoxicity has been described as a feature of a number of jellyfish and marine venoms.[8] Anaphylaxis has been described following a second exposure.[9]

The only fatality we are aware of in association with LMJ envenoming is the fictional case described by Conan Doyle.[10] In this story, there is a description of a second near-fatal case of envenoming where pain, respiratory distress, sweating and cardiac symptoms are described, all features of Irukandji syndrome.[11] Lion's mane jellyfish are known to produce a variety of neuropeptide toxins but the effects of absorption of these toxins are largely unknown.

Decompression sickness (DCS) has protean manifestations. These include a variety of neurological symptoms and signs including numbness, weakness, paralysis, vestibular dysfunction, bladder dysfunction and confusion which may occur in isolation or in association with other features of DCS such as skin rashes, joint pain or constitutional symptoms. It is not infrequent that a presentation may be indistinct, especially in the recreational diving community where other pre-existing pathologies may make definitive diagnosis more difficult. It is appropriate that divers present for assessment in the event of symptoms potentially attributable to DCS and also appropriate that if DCS cannot be excluded, recompression treatment is undertaken.

There is considerable diving activity in the waters around Orkney, both commercial and recreational. The dive sites of Scapa Flow attract large numbers of recreational divers each year.[12] The Orkney hyperbaric unit manages on average between 15 and 20 cases of decompression illness (DCI) each year. Approximately a third of these cases have neurological symptoms either in isolation or alongside other symptoms or signs. We describe a series of divers who presented to us as cases of suspected decompression illness.

Figure 1
Lions mane jellyfish (*Cyanea capillata*); photo taken in the Orkney Islands by Penny Martin

Case series

CASE 1

A 27-year-old man undertook a 31-minute (min) dive to a maximum depth of 25 metres' sea water (msw) breathing nitrox25. During the dive he saw LMJ in the water and was aware of tentacles around his regulator with a tingling sensation around his lips. On surfacing he felt continued tingling around his lips and had visible local swelling. Ten minutes later he experienced pinprick sensations in all of his limbs which were fleeting and migratory. On de-kitting he felt irritation around his left wrist where he subsequently noticed a transient red patch. He was treated with oxygen (O_2) on the dive boat, noticing no difference in his symptoms. He presented to the diving medical team two and a half hours after surfacing. On examination, apart from swelling around his lips, there were no abnormal signs. He was treated with surface oxygen and an antihistamine and observed. His symptoms gradually subsided and he was discharged.

CASE 2

A 61-year-old man undertook a 35-min dive to a maximum depth of 34 msw breathing air. During a safety stop at 6 msw he noticed an intense pain across his top lip and left cheek. He was aware of jellyfish in the water around the shot line. On surfacing, 10–15 min later he noticed fleeting pinprick sensations from his neck to his toes. These symptoms persisted and the dive-boat skipper treated him with O_2, without any change in his symptoms. He was referred to the diving medical team. By the time he arrived some five hours after surfacing, his symptoms had substantially improved. Physical examination was unremarkable and no other treatment was required. On review the following morning his symptoms had completely resolved.

CASE 3

A 21-year-old man undertook a 36-min dive to a maximum depth of 27 msw, breathing nitrox27. This was the first dive of his holiday. He was aware of LMJ in the water and tentacles around his regulator as he was going down the shot line. During his dive he noticed a tingling sensation around his lips which persisted on surfacing. An hour or so later he noticed pinprick tingling in both arms, axillae, across his abdomen and in both legs. He felt light-headed and hot. Several hours later, with the pinprick sensations persisting, he consulted a doctor who was supervising his party. On advice, he presented to medical care six hours after surfacing. On arrival, he had slight swelling of his lower lip but no other abnormal signs. He was clearly anxious. After discussion, he was recompressed using a USN Treatment Table 6 (USN TT6). There was no apparent change in his symptoms during the treatment but when reviewed the following day, these had resolved completely and he felt well.

CASE 4

A 49-year-old man undertook a 49-min dive to a maximum depth of 38 msw, breathing nitrox27. It was the third dive of his trip. He was aware of LMJ in the water and of tentacles around his regulator. On surfacing he noticed tingling around his lips and had local swelling. Thirty minutes later, he developed tingling of his right big toe, the sole of his left foot, the palmar aspect of his right hand and his left hand. On arrival at the chamber he was anxious but not hyperventilating. Physical examination was unremarkable with no objective sensory loss and no focal neurological abnormalities. In view of persisting symptoms in both hands and feet, he was recompressed using a USN TT6. His symptoms resolved completely during the treatment and he remained asymptomatic on review the following morning.

CASE 5

A 28-year-old marine scientist presented to us two hours after completing a 50-min dive to a maximum depth of 36 msw, breathing nitrox25. She was stung on her face by a jellyfish during descent which she identified as a LMJ. She felt immediate pain around her top lip during the dive which worsened as her dive progressed. On surfacing she was aware of a flitting "*pins and needles*" sensations throughout her whole body. She was treated with 80% O_2 on the dive-boat and self-medicated with a 1 mg betamethasone tablet and topical 1% hydrocortisone cream to her face. At the time of presentation there was visible swelling of her upper lip but no other abnormal clinical signs. Her symptoms had already begun to resolve. She was observed on O_2 for a further hour by which time her systemic symptoms had subsided. No other treatment was necessary.

Discussion

The cases described above are from a large series of more than 550 divers presenting to the Orkney unit over the past 20 years. All of these divers presented with suspected DCI or were perceived at risk of developing DCS after uncontrolled ascents or missed decompression. Three-hundred and sixty-two of these divers had a final diagnosis of DCS of which 130 had symptoms or signs consistent with involvement of the neurological system. More than 80% of these divers developed neurological features within an hour of surfacing, including localised numbness, weakness, girdle pain, paralysis, vestibular dysfunction and urinary retention.

The five cases described in this series are distinct from the DCS cases in a number of features. In all cases, the neurological symptoms were flitting and widespread with pinpoint tingling sensations at multiple sites. Treatment with O2 on the dive-boat did not produce any identifiable change in symptoms. None of the five divers in this series had any obvious precipitating factors for DCS, such as an unusually significant nitrogen load, uncontrolled ascents or missed decompression. None of the cases had presentations or histories suggestive of shunt-related DCS, e.g., skin rashes, vestibular or spinal symptoms, history of migraine with visual aura or previous DCS. The absence of an identifiable precipitating event or predisposition does not exclude DCS, but it does make it less likely. In all five cases, the diver reported contact with jellyfish tentacles in the water. Whilst we cannot be completely confident that all were LMJ, all of the descriptions were consistent and these are the predominant jellyfish with long tentacles in Orkney waters and are commonly found around shot lines. In one case there was a definitive identification, in the other four cases LMJ were reported to have been in the water at the dive site. The presence of local symptoms at the site of contact also point to LMJ as the species implicated, there are no other toxic jellyfish commonly present in Orkney waters (Porter J, personal communication, 2018). It is not possible at present to identify toxin in serum samples but in the event of significant local reaction, skin scrapings or sticky tape tests may aid identification of nematocysts in future cases (Currie B, personal communication, 2018).

Despite the presence of systemic symptoms, none of these five cases were seriously unwell. We did not see features suggestive of the Irukandji syndrome which has been described in envenoming by a variety of carybdeid jellyfish.[13,14] It appears, however, that although less severe, there may be some shared symptoms of Irukandji-like envenoming from toxins of non-carybdeid jellyfish (Currie B, personal communication, 2018).[7] Two of our patients were recompressed because at the time of presentation it was impossible to definitively exclude DCS. There was an improvement in the symptoms of one of these divers during recompression, which could suggest that DCS was indeed the underlying diagnosis. However, we suspect that what we observed was the natural resolution of symptoms with time rather than a therapeutic effect of recompression (an un-extended USN TT6 lasts 4 hours 40 min). Symptoms in the other four divers in this series resolved within a similar time frame without recompression. It was salutary to observe that case 1 was a diver who normally carried adrenaline for self-treatment of anaphylaxis for a nut allergy. He did not usually carry adrenaline on the dive-boat during dive trips. Anaphylaxis has been described after a second LMJ sting and we advised this diver that in future he should carry his adrenaline with him on the boat. The mechanism of anaphylaxis was first described by using a protein extracted from the tentacles of a Portuguese man of war (*Physalia physalis*).[2]

We believe that these cases exhibited a mild syndrome of systemic envenoming with LMJ toxin and that it is likely that this was the explanation for the symptoms in all five divers. In addition to discomfort and inflammation at the contact site, all five had similar flitting neurological symptoms. It was appropriate that all five divers presented as possible cases of DCS but the symptoms were not typical of the presentations we usually see. It was impossible to definitively exclude DCS in two of these cases and, in similar cases where significant doubt exists, it will always remain safer to treat with recompression than not to do so. Notwithstanding this, where there are no obvious precipitating factors, a definite history of LMJ contact, atypical flitting symptoms and an absence of neurological signs, we believe it is reasonable to observe these patients on oxygen in the first instance rather than recompress them.

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Ethics statement

This was a retrospective case series of divers presenting over more than 10 years. It was not possible to contact some of the earlier cases in the series. Advice was sought from the Director of Public Health and the Caldicott Guardian for the National Health Service – Orkney. Given the time course that had elapsed and the absence of identifiable clinical information, it was considered that there were no significant confidentiality issues which would prevent publication.

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Massive portal venous gas embolism after scuba diving

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Key words
Decompression sickness; Liver; Gastrointestinal tract; Hyperbaric oxygen therapy

Abstract
(Siaffa R, Luciani M, Grandjean B, Coulange M. Massive portal venous gas embolism after scuba diving. Diving and Hyperbaric Medicine. 2019 March 31;49(1):61–63. doi: 10.28920/dhm49.1.61-63. PMID: 30856669.)
Introduction: Portal venous gas from a diving injury is an infrequent finding and only a few cases are described. We report a case of severe decompression sickness (DCS) associated with a massive amount of gas in the portal and mesenteric veins.
Case report: A 49-year-old man suffered from DCS after two deep dives on the same day. He presented with cutaneous, neurological and pulmonary symptoms associated with hypoxaemia. He had no abdominal pain. A computed tomography (CT) scan showed large quantities of hepatic and portal venous gas and excluded other explanations for its presence. All symptoms disappeared with hyperbaric oxygen therapy and there were no further complications.
Discussion and conclusion: The role of portal venous gas in DCS is not obvious. Isolated portal venous gas seems to cause no obvious harm. Medical imaging should be considered for differential diagnosis and to prevent some complications, especially in divers presenting with abdominal pain.

Introduction

Gas embolism is one of the main causes of diving injury and can happen after decompression sickness (DCS), in which large quantities of inert gas are released by the tissues. Gas embolism can also occur after lung and possibly hollow organ barotrauma during ascent, especially if the ascent is rapid. The condition may lead to severe neurological disability, and hyperbaric oxygen treatment (HBOT) is needed urgently. In this report, a diver with severe decompression sickness (DCS) exhibited massive and unusual gas formation in the liver and gastrointestinal tract.

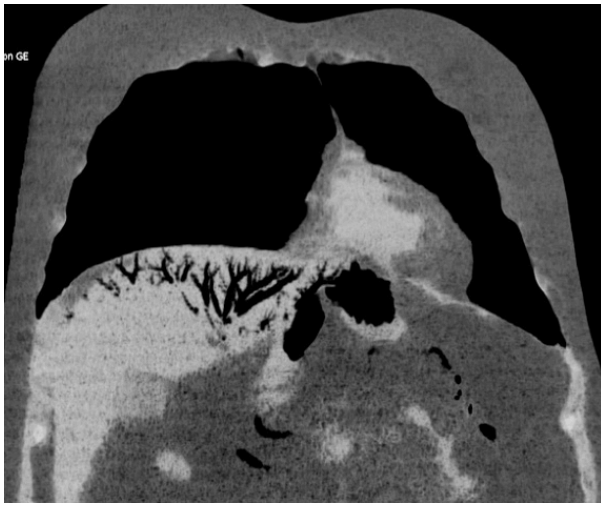
Case report

A 49-year-old, obese (body mass index 34 $kg \cdot m^{-2}$) man who was a moderately experienced diver (three years of diving, 35 dives in the past year) had a first dive to a maximum depth of 60 metres' sea water (msw) (15-minutes (min) bottom time, 52-min total dive time). After three and three-quarter hours resting, he made a second dive. He stayed one min at 40 msw (maximal depth) and 12 min at 25 msw, with a total dive time of 53 min. All decompression procedures were fully respected. He did not ingest a gaseous beverage or heavy meal before diving. Approximately 20 min after surfacing, he experienced asthenia, chest tightness, shortness of breath, vertigo, and mental confusion. He was treated immediately with oxygen (O_2) 15 $L \cdot min^{-1}$ via a mask and transferred to the hyperbaric centre at Ajaccio Hospital, Corsica. On admission, clinical examination revealed vertigo and a pruritic erythematous rash on the abdomen, evoking skin bends. O_2 saturation was 91% on air. Haemodynamic parameters and the rest of the clinical review were normal. Blood tests showed hypoxaemia (P_aO_2 = 55 mmHg), mild haemoconcentration (haematocrit 55%), and C reactive protein (CRP) was 30 $g \cdot L^{-1}$. Because of the initial dyspnoea and hypoxaemia, the patient underwent a computed tomography (CT) scan, which excluded pleural or pulmonary injury (i.e., pneumothorax, pleural effusion, pulmonary oedema, or infection). However, CT showed portal venous gas, looking like an intrahepatic 'dead tree'. There was also gas within mesenteric vessels. No other abnormality was seen on abdominal CT scan. An ENT consultation found that the vertigo was of a central origin.

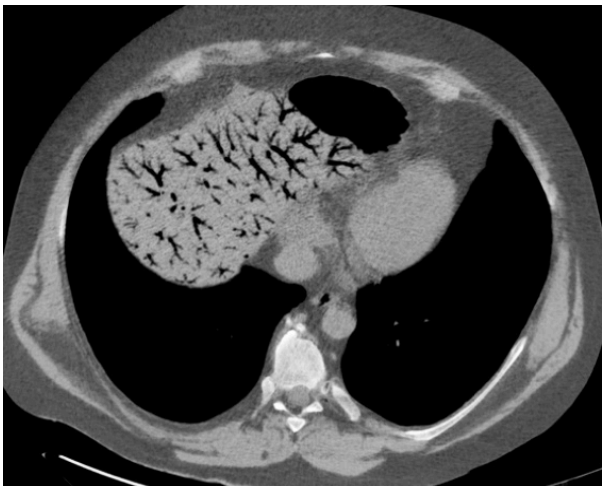
The patient underwent hyperbaric oxygen treatment (HBOT) using a six-hour table (405 kPa maximum pressure, nitrox 80/20, then nitrox 70/30 and finally 100% O_2), intravenous hydration and corticosteroids (40 mg methylprednisone thrice daily, then prednisone 20 mg thrice daily for seven days' total treatment). Four further HBOT (152 kPa, 90 min, 100% O_2) were given, with full recovery of all symptoms and signs of DCS. Transcranial Doppler demonstrated a right-to-left shunt which further investigation confirmed to be a persistent foramen ovale (PFO). He remained asymptomatic and was discharged on the fourth day post incident to return

Figure 1
Abdominal CT; (A) coronal reconstruction and (B,C) axial slides showing portal venous gas with a hepatic 'dead tree' appearance; gas is also present in mesenteric vessels (A, C)

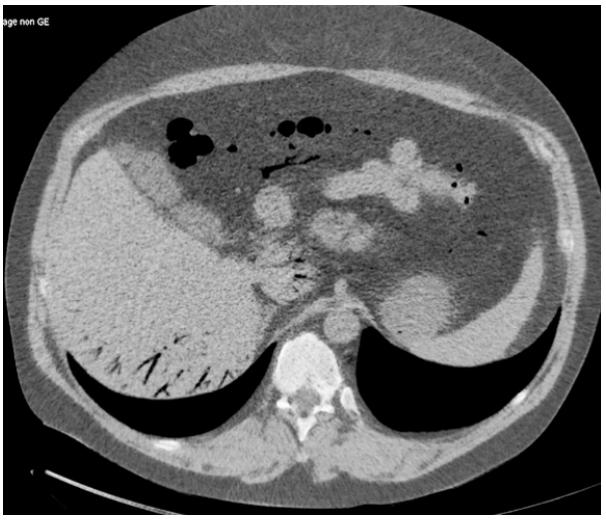
A



B



C



by sea to mainland France. Although now symptom-free, a further four HBOT were administered as an outpatient in Nice. Two months later the PFO was closed and he has returned to diving without any problems.

Discussion

Portal venous gas is a radiological diagnosis. It is defined as a radiolucency of vascular branches within 2 cm beneath the liver capsule. The condition is usually considered a rare and critical disease and may be caused by sepsis (e.g., appendicitis, cholecystitis, diverticulitis), stomach or bowel lining disease (e.g., mesenteric ischaemia, peptic ulcer, inflammatory bowel disease), or an excessive pressure inside hollow organs (e.g., barotrauma, blunt trauma, endoscopy).[1] However, the origin of portal venous gas is not found in 15% of cases. DCS is considered a rare cause of hepatic/portal venous gas, and digestive tract barotrauma (from stomach or bowel) is the primary differential diagnosis.[2] Its pathological significance is not clearly understood and remains controversial. One case has been reported of portal venous gas resulting from barotrauma after a rapid ascent.[3] This occurred after having a heavy meal and gaseous drink just before the dive, but the diver also had symptoms of DCS, and the off-gassing process could also explain the portal venous gas. This case is important because the patient developed a portal thrombosis. Therefore, the authors considered this risk requires further investigation in severe DCS. In another case of DCS exhibiting portal venous gas, abdominal pain was reported in addition to neurological and cutaneous symptoms.[4]

The decision to recompress was driven largely by the presence of neurological symptoms and rash, in addition to the radiological finding of apparently symptomatic portal gas. However, when it is isolated or asymptomatic, the evolution of portal venous gas may be a typically benign process and may be more common than presently appreciated. For example, portal venous gas was found in four of nine divers with DCS investigated with CT for unrelated symptoms such as dyspnoea.[5] Although other case reports have been published, the pathological significance of portal venous gas is not obvious. It seems that a poor prognosis is not related to isolated portal venous gas, but rather to associated severe DCS symptoms. Medical imaging, mainly CT, is essential for differential diagnosis.

We strongly recommend CT scanning when divers present with abdominal symptoms and there is doubt about the cause. CT scans are now easily available in many centres without important loss of time to HBOT. Since a small incremental delay to recompression is unlikely to adversely affect outcome in presentations of typical latency,[6] there seems little potential for harm in pursuing such investigations. A quick, non-invasive device, such as hepatic ultrasound and Doppler, might also be useful, further reducing the delay to recompression.

Conclusion

Portal venous gas may be present in cases of DCS but appears to be a radiological-clinical mismatch; that is, few or no symptoms despite positive CT scan images. Early CT helps in the differential diagnosis and should be performed when divers present with abdominal pain of uncertain cause. A cohort study would indicate the true incidence of portal venous gas after diving.

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The diver initially was lost to follow up but subsequently contacted by phone, at which time he gave verbal consent for publication of his case and radiology.

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Letter to the Editor

Gas micronuclei that underlie decompression bubbles and decompression sickness have not been identified

Gas micronuclei are gas-filled voids in liquids from which bubbles can form at low gas supersaturation. If water is depleted of gas micronuclei, high gas supersaturation is required for bubble formation. This high gas supersaturation is required in part to overcome the Laplace pressure at the point of transition from dissolved gas to a bubble of perhaps nanometer-scale radius. The sum of gas and vapour partial pressures inside a spherical bubble (P_{bub}) of radius r exceeds the ambient barometric pressure (P_{amb}) and is given by the Young-LaPlace equation:

$$P_{bub} = P_{amb} + \frac{2\gamma}{r} \quad (1)$$

for a bubble not in contact with a solid surface. The second term on the right-hand side is the Laplace pressure across the gas-liquid interface due to surface tension (γ). For instance, for a surface tension characteristic of blood of 0.056 $N \cdot m^{-1}$, *de novo* formation of a bubble of r = 10 nm requires gas supersaturation exceeding $2\gamma/r$ = 11.2 MPa.[1]

However, in humans, detectable venous gas bubbles follow decompression to sea level from as shallow as 138 kPa air saturation, implying gas supersaturation of only a few kPa are required for decompression bubble formation.[2] It is widely accepted that bubbles that form at such low gas supersaturation grow from pre-existing, micron-scale gas micronuclei. For such gas micronuclei to already exist prior to gas supersaturation they cannot simply be small bubbles because positive feedback of Laplace pressure causes a micron radius bubble to dissolve in a fraction of a second.[3] Theoretical candidates for gas micronuclei are bubbles coated in surfactants that counteract the Laplace pressure or crevices where gas voids assumes shapes that negate the Laplace pressure. However, to date, the nature of gas micronuclei that underly decompression-induced bubbles and decompression sickness have yet to be identified.

Consequently, I was intrigued that in two previous issues of *Diving and Hyperbaric Medicine* (2018 Volume 48, Issue 2, page 114 and Issue 3, page 197), letters from Ran Arieli to the Editor hypothesized a mechanism for decompression bubble formation in blood vessels and in the skin. Both letters stated "*It is known that nanobubbles form spontaneously when a smooth hydrophobic surface is submerged in water containing dissolved gas. We have shown that nanobubbles are the gas micronuclei underlying decompression bubbles and decompression sickness*". Surface nanobubbles have been extensively described in the physical chemistry literature, but the second sentence is supported by citation of an hypothesis article.[4] The latter is based on experimental work (referenced therein) in which sections of large blood vessels from sheep were incubated in saline and compressed to 1.013 MPa for 18 hours then rapidly decompressed to the surface, whereupon macroscopic bubbles were photographed forming on the luminal surface of the vessels. The authors speculate that the bubbles were forming from surface nanobubbles on the vessel lumen, but no experimental or analytical evidence was presented that surface nanobubbles were present on the vessel lumen or were the precursors of the observed macroscopic bubbles.

Surface nanobubbles form on atomically smooth, hard surfaces in gas supersaturated liquids and, imaged with atomic force microscopy, appear as spherical caps of gas. As far as I can determine, surface nanobubbles have not been reported on biological tissue surfaces. Surface nanobubbles typically have diameters less than 100 nanometers but have lifetimes that are orders of magnitude longer than would a bubble of similar dimensions. Surface nanobubbles do not grow into macroscopic bubbles when exposed to pressure waves sufficient to cause bubble formation from adventitious gas micronuclei elsewhere in the apparatus.[5,6] This is surely not the last word in this new and active field of research into nanoscopic gas species; however, based on current evidence one must treat with skepticism speculation that unobserved surface nanobubbles are the gas micronuclei from which bubbles form in humans with low gas supersaturation and which underlie decompression sickness.

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Key words
Letters (to the Editor); All other relevant keywords are in the title

Critical appraisal

Boussuges A, Ayme K, Chaumet G, Albier E, Borgnetta M, Gavarry O. Observational study of potential risk factors of immersion pulmonary oedema in healthy divers: exercise intensity is the main contributor. Sports Medicine Open. 2017;3:35. doi: 10.1186/s40798-017-0104-1.

Abstract

Background: The risk factors of pulmonary edema induced by diving in healthy subjects are not well known. The aim of the present study was to assess the parameters contributing to the increase in extravascular lung water after diving.
Methods: This study was carried out in a professional diving institute. All divers participating in the teaching program from June 2012 to June 2014 were included in the study. Extravascular lung water was assessed using the detection of ultrasound lung comets (ULC) by chest ultrasonography. Clinical parameters and dive profiles were recorded using a questionnaire and a dive computer.
Results: One-hundred [and] six divers were investigated after 263 dives. They used an open-circuit umbilical supplying compressed gas diving apparatus in 202 cases and a self-contained underwater breathing apparatus in 61 cases. A generalized linear mixed model analysis was performed which demonstrated that the dive induced a significant increase in ULC score (incidence rate ratio: 3.16). It also identified that the predictive variable of increased extravascular lung water after the dive was the exercise intensity at depth ($z = 3.99$, $P < 0.0001$). The other parameters studied such as the water temperature, dive profile, hyperoxic exposure, or anthropometric data were not associated with the increase in extravascular lung water after the dive.
Conclusions: In this study, the exercise intensity was the main contributor to the increase in extravascular lung water in healthy divers. To improve the prevention of immersion pulmonary edema, the exercise intensity experienced during the dive should thus be adapted to the aerobic fitness level of the divers.

This article was not referenced in the paper on immersion pulmonary oedema (IPE) published in this issue for the following reasons. Other research suggests that exertion is an important factor for producing IPE in fit individuals, but the claim in this paper to have demonstrated that exercise intensity is the main contributor to IPE is invalid owing to flaws in the study. Ideally such a claim should be based on research with a predefined primary end-point with all other variables being fixed. So, one would study a group of individuals performing a series of dives with different exercise intensities and in random order.

Instead of setting a primary aim of assessing the role of exercise intensity, the authors investigated a number of environmental and personal factors of which exercise intensity was just one. Analyses of many factors without adequate correction for multiple analyses can result in chance but spurious associations. In this study, there was no proper standardization of exercise and other dive parameters were not fixed. They studied 104 men and only two women, though IPE is more common in women; ages varied from 19 to 58 years. They performed a total of 263 dives, but the number of dives per diver varied from one to six. Some wore drysuits and others wetsuits. Some used scuba and others used surface supply. Maximum depths varied from 8 to 48 m and dive durations were between nine and 77 minutes (min). Gases breathed at decompression stops differed between dives. The duration of immersion varied from 15 to 113 min, so some were immersed at the surface for more than 30 minutes after surfacing. The lung ultrasounds used to assess extravascular lung water (lung comet score) were performed between five and 60 min after the end of the dives, which is presumably after getting out of the water rather than surfacing because no diver was out of the water within five min of surfacing. The paper refers to water temperature of dives, but it is not stated which water temperature they used (surface, bottom or average temperature).

There are also statistical concerns. The authors performed multiple complex statistical comparisons and found a relationship between exercise intensity and comet scores. At best, such analysis can be used to generate a hypothesis: it does not provide proof of causal association. Critically when considering causality, the way exercise intensity was assessed is subjective, based on the 15-point Borg Rating Scale of Perceived Exertion; objective exercise testing was not done. This scale depends on fitness level and will clearly be affected by anything that makes a subject breathless, such as increased extravascular lung water causing an increase in lung stiffness. Put simply, the way many people judge how much they are exerting themselves is how breathless they feel. So it is not surprising that there was a relation between perceived exertion (i.e., breathlessness) and lung comet score. It seems equally plausible that extravascular lung water caused breathlessness and hence perception of exercise intensity as opposed to high levels of exertion causing increase amounts of extravascular lung water.

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Key words
Critical appraisal; Immersion; Pulmonary oedema; Exercise

Reprint

Clinical Practice Guidelines Committee of The American Society of Colon and Rectal Surgeons. The American Society of Colon and Rectal Surgeons Clinical Practice Guidelines for the Treatment of Chronic Radiation Proctitis

Paquette IM[1], Vogel JD[2], Abbas MA[3], Feingold DL[4], Steele SR[5]

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[5] *Cleveland Clinic, Cleveland, Ohio*

Hyperbaric oxygen therapy is an effective treatment modality to reduce bleeding in patients with CRP. Grade of Recommendation: Strong recommendation based on moderate-quality evidence, 1B.

Hyperbaric oxygen therapy (HBOT) has emerged as an effective treatment for nonhealing wounds from various etiologies including traumatic, postoperative, diabetic related, or radiation induced. The impact of HBOT on tissue healing is postulated through improving tissue oxygenation and possible angiogenic and antibacterial effects. Woo et al evaluated 18 patients with CRP. Of 13 patients with rectal bleeding, 4 patients had complete resolution and 3 had some improvement. Kitta et al from Japan reported the outcome of 4 patients with radiation proctitis after treatment of prostate cancer. Although patients had a significant reduction in degree of bleeding, 1 patient relapsed 3 months after completion of therapy, 1 continued to have minor rectal bleeding, and 1 continued to have persistent proctalgia with no rectal bleeding. Another study examined the outcome of 10 patients, including 3 men and 7 women, with CRP treated by HBOT. HBOT was well tolerated, and 9 of the 10 patients completed the full course of 40 treatments. During a median follow-up period of 25 months, rectal bleeding stopped in 4 patients and improved in 3 others. There was symptomatic improvement in bleeding, diarrhea, and rectal pain in the majority of patients. Only 2 of the 10 patients had no response. Similarly, Oscarsson et al conducted a prospective cohort study to assess the effectiveness of HBOT in patients with CRP. Thirty-nine patients (35 men and 4 women, mean age of 71 y) were evaluated. The mean number of treatment was 36 sessions. The symptoms of CRP were alleviated in 89% of the patients. A randomized, controlled, double-blind crossover trial was conducted by Clarke et al to assess the long-term effectiveness of HBOT in patients with refractory radiation proctitis. The patients were randomly assigned to HBOT (100% oxygen at 2.0 atmospheres, group 1) versus air (21% oxygen at 1.1 atmospheres, group 2). Patients in group 2 were subsequently crossed to group 1. Symptoms were assessed at 3 and 6 months and then at year 1 to 5. Of 150 patients enrolled in the study, 120 patients were evaluable. The amount of improvement was nearly twice as great in group 1, which had a greater portion of responders (88.9% vs 62.5%; $P = 0.009$). In group 2, the differences were abolished after crossover. Virginia Mason Medical Center reported its experience with HBOT for patients treated for prostate cancer with radiotherapy. Over a 5-year period, 27 patients received HBOT (average of 36 sessions; range, 29 to 60 sessions). Complete resolution of bleeding was noted in 48% of patients, and 28% reported fever bleeding episodes. Fecal urgency resolved in 50% of the patients. Of patients with rectal ulcers on endoscopy, complete resolution was noted in 21%, and 29% had some improvement. In this study, only 33% of the patients had no response. Although there is a clear benefit to HBOT in patients with CRP, it is an expensive therapy that requires specialized equipment and several weeks of treatment sessions; thus, it is not widely available.

Summary section on hyperbaric oxygen treatment reproduced from:
Paquette IM, Vogel JD, Abbas MA, Feingold DL, Steele SR. Clinical Practice Guidelines Committee of the American Society of Colon and Rectal Surgeons. The American Society of Colon and Rectal Surgeons Clinical Practice Guidelines for the treatment of chronic radiation proctitis. Dis Colon Rectum. 2018;61(10):1135–40. doi: 10.1097/DCR.0000000000001209. PMID: 30192320. [cited 2018 October 06]. Available from: https://journals.lww.com/dcrjournal/fulltext/2018/10000/The_American_Society_of_Colon_and_Rectal_Surgeons.4.aspx.

Key words
Soft-tissue radionecrosis; Reprinted from; Abstract

Notices and news

EUBS notices and news and all other society information is now to be found mainly on the society's website: www.eubs.org

EUBS Annual Scientific Meeting 2019

Dates: 09–12 September 2019
Venue: David Intercontinental Hotel Tel Aviv, Israel

Main theme
Hyperbaric medicine and the brain

Emerging indications related to hyperbaric oxygen and the brain will be covered in depth by the world's leading investigators. In addition, new physiological data related to the effect of hyperbaric oxygen on sports performance and mitochondrial function will be presented.

The conference will be jointly organised with the International Conference on Hyperbaric Oxygen and the Brain. The conference will be hosted by the Israeli Society for Hyperbaric and Diving Medicine.

This period has been chosen as it is after the summer break but just before the high holidays in Israel. Tel Aviv is an exciting hypermodern coastal city, with top-notch medical research and treatment facilities, but also bustling with beaches, restaurants and nightlife. Please register in time, as early registration for EUBS 2019 ends on 15 April 2019. Also, favourable airfares are dependent on early booking and hotel accommodation tends to be in high demand. Please submit your abstract too, as your input is an important part of the success of our annual meeting.

Every year, the EUBS ExCom supports young scientists by awarding Student Travel Grants and other awards. You can find information on the conditions and application process on https://eubs2019.com/eubs-awards/.

You can find all the information regarding the conference on the website www.eubs2019.com, or by visiting the EUBS website www.eubs.org.

EUBS Executive Committee

Every year, a new Executive Committee member needs to be elected – elections start well before our next General Assembly (during the EUBS Annual Scientific Meeting). Candidates will be presented by the Executive Committee by June 2019, and the voting, as usual, will be by internet ballot starting on 30 June. If you want to contribute and help our Society, please come forward and send your short CV to our secretary (secretary@eubs.org).

This year, we will need a new Member-at-Large. If you do not feel like presenting yourself, why not nominate someone else? Suggestions are welcome at the same e-mail address.

EUBS website

Please visit the EUBS website for the latest news and updates. The 'EUBS History' section (under the Menu item 'The Society') is still missing some information in the list of EUBS Meetings, Presidents and Members-at-Large. Please dig into your memories and help us complete this list.

By popular demand, EUBS members can now also download the complete Abstract Book of previous EUBS meetings from the members' area.

While on the EUBS website, make sure you take a look at our Corporate Members' webpage http://www.eubs.org/?page_id=91. On this page, logos and links are placed to those organizations, societies and companies that support EUBS financially. EUBS is grateful for their continued support and would suggest that if you contact any of them, please do so by clicking on the link on that page, so they will know that you did so through the EUBS website.

EUBS Address change

Please note that the administrative address for EUBS changed as from 01 January 2019. You can find the complete up-to-date address and bank information at: http://www.eubs.org/?page_id=163.

EUBS President's message

Ole Hyldegaard

New editor of *Diving and Hyperbaric Medicine*

We welcome our new Editor-In-Chief of the journal *Diving and Hyperbaric Medicine* (DHM), Professor Simon Mitchell, who has taken over from Associate Professor Mike Davis. Simon will be taking over the DHM journal at a time when we need to further professionalize the production and related administrative procedures for DHM. Having a fully-fledged electronic journal with online PubMed Central publications available for download puts higher demands on the overall workload and thereby also our annual DHM budget. The ExComs of both publishing Societies will have to address these issues more closely during 2019.

Having an esteemed journal such as ours is extremely important for the fields of both diving and clinical hyperbaric medicine, and I call upon all members to support a continuously positive development for our journal and encourage new potential colleagues to become members of the EUBS. Without a strong membership foundation our journal cannot exist and because the European region is large, we should hold a membership potential significantly larger than the current one. However, diversity between the nations of Europe exists, with different traditions of hyperbaric and diving medicine. Different national regulations and requirements for continuing medical education (CME) may also mean that the national diving and hyperbaric societies may be the first and more obvious choice for memberships locally, rather than the EUBS; my own country – Denmark – being no exception. As former presidents and EUBS ExComs before me have done, I encourage all members to seek every opportunity to encourage fellow divers and colleagues to become members of the EUBS. The need for us to build strong international cooperation and produce scientific data is greater than ever before as the situation described below clearly demonstrates.

Dark clouds build over clinical hyperbaric therapy in the UK … and may spill over to other regions of the European continent.

For some time, the National Health Service (NHS) in the UK has targeted clinical hyperbaric medicine as an easy, non-industrially (pharmaceutically) supported treatment option for demonstrating their determination to reduce public health care costs by choosing an approach of economically driven 'evidence-based medicine'. It seems as if any deviation from high level evidence is interpreted as similar or equal to 'no effect of therapy'. Only decompression illness or cerebral arterial gas embolism may be left as NHS publicly supported and endorsed diagnoses to be treated by hyperbaric therapy, which overall seems to me to be contradictory.

Recently we learned that the London Hyperbaric Centre is closing due to the cessation of NHS endorsement and funding. This grave situation in the UK may be different from the rest of Europe for many reasons. However, being a small community within the medical world, we should learn and listen to these lessons, and through international cooperation supported by the EUBS and SPUMS, centres involved in hyperbaric medicine should come together and exchange data, including prospective studies on treatment outcomes, using harmonized treatment protocols and data registries.

The recent example and initiative of Pieter Bothma (UK) to form a committee on the treatment of necrotizing soft tissue infections and use of HBOT in this context is a step on the road. Initiatives to form a joint clinical data registry within the Nordic countries, currently driven by Nicklas Oscarsson in Gothenburg, Sweden and Guro Vaagbø in Bergen, Norway, is another such example that could also be expanded further once the database becomes operational and validated. Areas such as wound care and HBOT effects on brain injuries, acute or chronic, will in a similar way benefit from joint, international research initiatives. The work of our colleagues in Israel and the upcoming conference and workshops of the EUBS 2019 meeting in Tel-Aviv will be a great platform to promote such initiatives. If we manage to seize the moment, there may be light at the end of tunnel after all. We need to take the initiative.

Key words
Medical society; General interest

The EUBS website is at <www.eubs.org>

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

The latest issues of *Diving and Hyperbaric Medicine* are via your society website login.

EUBS Affiliate Society agreements

For 2019, an agreement has been renewed with the following scientific societies in order to promote membership and contact among the hyperbaric and diving scientists and practitioners in Europe and (why not) worldwide. Members of these societies benefit from a 10% reduction on the EUBS membership fees, when providing proof of their membership of the 'other' society. Simply indicate the affiliate society on the EUBS membership application or renewal form.

Belgian Society for Diving and Hyperbaric Medicine
http://www.sbmhs-bvoog.be
Scott Haldane Foundation, The Netherlands
http://www.scotthaldane.org
Italian Society for Diving and Hyperbaric Medicine
http://www.simsi.it/
German Society for Diving and Underwater Medicine
http://www.gtuem.org
French Society for Diving and Hyperbaric Medicine
http://www.medsubhyp.com
Swiss Society for Underwater and Hyperbaric Medicine
http://www.suhms.org
Undersea and Hyperbaric Medical Society
http://www.uhms.org

We are pleased to announce that in exchange, EUBS members benefit from a substantial reduction to their UHMS membership – simply mention your EUBS membership when enrolling/renewing your UHMS membership. In addition, we are discussing a new agreement with the Danish Aviation and Diving Medical Society and invite other National Societies to contact us in order to further expand these affiliate agreements.

Obviously, members of SPUMS already automatically benefit from most of our EUBS membership benefits, such as the DHM Journal, a reduced registration fee for the EUBS Annuals Scientific Meetings and access to the GTÜM database of non-indexed scientific literature.

Hyperbaric oxygen lectures

Welcome to: http://www.hyperbaricoxygen.se/
This site offers publications and high-quality lectures from leading investigators in hyperbaric medicine. Please register to obtain a password via email. Once registered, watch online, or download to your iPhone, smart device or computer for later viewing.
For information contact: folke.lind@gmail.se

OXYNET Database to be updated

Since 2004, a public database of European Hyperbaric Chambers and Centres has been available online, started and initially maintained by the OXYNET Working Group of the COST B14 project of the European Commission, later by the European Committee for Hyperbaric Medicine (ECHM). The database can be accessed on www.oxynet.org.

However, over the past few years, the list and contact information of the OXYNET database has not been maintained regularly, and EUBS ExCom has proposed to take over this task and not only update the information but also to modernize the database and its functionality.

In order to do this, we can use all the help we can get. Please visit the OXYNET and verify the information that is listed for your own hyperbaric centre. Then, rather than using the online form to correct the information, send an e-mail to (oxynet@eubs.org) with the updated information. If you could collect information for more than one centre in your area or country, please do so.

Once the OXYNET database has been relocated and restructured, a direct link will be placed on the EUBS website, however, we will maintain the address www.oxynet.org as well.

The Science of Diving

Support EUBS by buying the PHYPODE book "*The science of diving*". Written for anyone with an interest in the latest research in diving physiology and pathology. The royalties from this book are being donated to the EUBS.
Available from: Morebooks https://www.morebooks.de/store/gb/book/the-science-of-diving/isbn/978-3-659-66233-1

20th International Congress on Hyperbaric Medicine 2020

Dates: 13–16 September 2020
Venue: Rio de Janeiro, Brazil
For preliminary information contact:
Dr Mariza D'Agostino Dias
Email: mariza@hiperbarico.com.br

German Society for Diving and Hyperbaric Medicine (GTÜM)

An overview of basic and refresher courses in diving and hyperbaric medicine, accredited by GTÜM according to EDTC/ECHM curricula, can be found on the website:
http://www.gtuem.org/212/Kurse_/_Termine/Kurse.html

Notices and news

SPUMS society information and news is to be found mainly on the society website: www.spums.org.au

Venue: Solomon Kitano Mendana Hotel, Honiara, Solomon Islands

Keynote Speaker
Associate Professor Nigel Stuart Jepson
Senior Staff Specialist and Director of the Cardiac Catheterization Laboratories of the Eastern Heart Clinic, Prince of Wales Public Hospital (POWH), Sydney, Australia

Scientific Convenors:
Clinical Professor David Smart and Professor Michael Bennett
Have an abstract or paper you would like to present? – Please email our Scientific Convener:
scientific.convener@spums.org.au

Event Convener
Dr Catherine Meehan cmeehan@mcleodstmed.com.au

Follow this link to get to the event webpage and to register for the event: http://www.cvent.com/d/2bqnpp
Check out the Facebook page: https://www.facebook.com/events/2112622009000126/

SPUMS 49th Annual Scientific Meeting

19–24 April 2020

Venue: Oceans Resort, Tutukaka, New Zealand

The scientific programme for 2020 is coming together and looking good - "*Medical Support of Diving in Remote Locations*", featuring our very own Richard Harris as Keynote Speaker. Add to that some of the best subtropical diving on the planet at the Poor Knights Islands Marine Reserve and this is an event not to be missed! Full details coming soon.
See you in Tutukaka in April 2020.

Guest Speaker: Richard Harris, Adelaide
Convenor: Greg van der Hulst
Scientific Convenors: Hanna van Waart and Xavier Vrijdag

SPUMS President's message

David Smart

I hope all SPUMS members had a safe and happy Christmas and New Year. By now, all members will have received membership renewal notices and possibly some reminders, if your first notice was not acted upon. Thank you for your continued support of SPUMS.

This edition of *Diving and Hyperbaric Medicine* marks the commencement of a new era, with Professor Simon Mitchell officially taking over as Editor. Both SPUMS and EUBS are indeed very lucky that Simon has been recruited to take on the Editor's role. I personally cannot think of anyone more qualified to lead our premier academic journal, following the outstanding contribution by Associate Professor Mike Davis from 2002–2018. A warm welcome, Simon.

2019 will be a year of consolidation following enormous changes over the preceding two years. With great assistance from our treasurers, Peter Smith (past) and Sarah Lockley (current), and Nicky Telles (web assistant), SPUMS has migrated its banking from St George to ANZ, and implemented a new website. The change of the journal from paper to electronic format has also required enormous effort by the editorial team but this has set DHM up for the future. I encourage all members of SPUMS and EUBS to get behind Simon and support our journal!

This year we move back to our usual format of regional scientific meetings. Dr Cathy Meehan is convener of this year's conference in the Solomon Islands – with keynote speaker Associate Professor Nigel Stuart Jepson. The theme is "*Old divers, bold divers but no old, bold divers*". Cardiovascular health risk assessment and diving. It promises to be a terrific programme, including diving and I invite all members to join us in Honiara in May 2019.

SPUMS Membership numbers have been static at just over 400 for a number of years. To grow and develop, we need more members. On behalf of the SPUMS Executive, I encourage all members to approach colleagues who have an interest in diving or hyperbaric medicine, to join our organisation. Growing our membership will be a key component of our strategic direction for 2019 and beyond. ExCom member Jen Coleman has kindly taken on this task.

Key words
Medical society; General interest

SPUMS Facebook page

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

ANZCA Report

Michael Bennett

2018 was a great year for SPUMS and DHM indeed – the year the Australia and New Zealand College of Anaesthetists (ANZCA) demonstrated their commitment to the field of diving and hyperbaric medicine unequivocally. The all new, entirely compliant qualification was officially established in August 2017, but 2018 was the year ANZCA followed through with the necessary infrastructure and governance systems lacking for the pre-existing certificate.

Many people within the college have worked hard to get us here and we (ANZHMG and SPUMS) owe a debt of gratitude to Lindy Roberts, Leona Wilson, Richard Waldron, Michael Jones, Moira Besterwich, Tamara Rowan and many others for their drive and commitment to what proved to be a complex process.

The college is a registered training organisation for the specific purpose of two classes of qualification - Fellowships and Diplomas. A 'certificate' has no official status and is not a qualification the college is registered to award. Hence the rather confusing new qualification: The ANZCA Diploma of Advanced Diving and Hyperbaric Medicine. We hope this qualification will be named in the next update of the Australian and NZ standards document (AS 4774.2) as the appropriate qualification for the medical director of a hyperbaric facility (publication imminent).

Transitional arrangements

By the time of publication, the opportunity for application via the three transition pathways for current 'specialist' practitioners will have closed. At my last calculation there are approximately 20 holders of the new ANZCA Diploma of Advanced DHM with a handful of further applications in the process. We have assessed one foreign medical graduate with an equivalent qualification and there are a couple of others who will be required to sit (and pass) the examination to complete their requirements.

Continuing professional development (CPD)

All practitioners will need to complete elements of DHM continuing education and ANZCA accepts the programmes from all other specialty bodies in Australia and New Zealand for this purpose. For ANZCA Fellows, the requirements for full CPD will continue unchanged. On advice from the college representatives, the DHM subcommittee for CPD, led by Susannah Sherlock, determined that because DHM is not a registered specialty, there is no mandated requirement for targeted CPD for specific DHM-related activities. This situation is the same for those holding membership of the Faculty of Pain Medicine, for example, so we are not alone in this. Each practitioner is responsible for their

own appropriate CPD activities, so the CPD subcommittee plans to develop some suitable activities to augment those currently available (e.g., the SPUMS ASM and this journal).

Anaesthetic DHM practitioners are advised to undertake the two emergency response activities relating to cardiac arrest and "*can't intubate can't oxygenate*" (CICO) as these are relevant to both areas of practice, however other emergency responses may be chosen.

The mandatory practice evaluation activities will be supported by minimally adapting existing templates to be more applicable to DHM. Additional wording will be inserted in the DHM Handbook to provide further advice both for ANZCA fellows and fellows from other colleges.

Accreditation of facilities for training

The process for accreditation of hyperbaric medicine units for training has been revised as part of the new Diploma. Existing training facilities have been accredited in the interim and the process is being finalised with the assistance of the appropriate college committees. Accreditation will be largely paper-based, assessed by members of the DHM SIG, with five-yearly review, and supplemented with opportunistic site visits, or as required.

The examinations committee

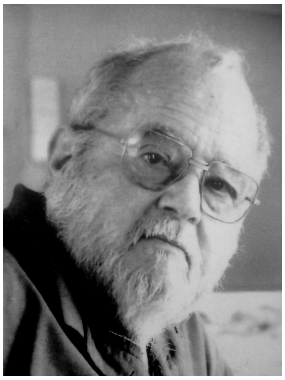
An examinations committee has been appointed and the first exam was held in Melbourne in July 2018 with a 100% pass rate recorded (1 of 1 candidate). Professor David Smart is chair of the committee and has a good team of examiners with him. The plans are to hold one examination each year. There will be two parts – written and oral – and all details are on the ANZCA website.

The special interest group (SIG)

The SIG continues to hold an annual meeting in conjunction with the ANZCA ASM. This meeting is very poorly attended, although it does give us an opportunity to officially present scientific papers in a dedicated setting and make our presence known in the schedule and proceedings. The SIG is currently considering a move to join the annual Hyperbaric Technicians and Nurses Association meeting where larger numbers of DHM practitioners are in attendance. The current chair of the SIG is Suzy Szekely. She has announced her intention to step down in the near future and anyone keen to contribute to the field in this way should contact her or Mike Bennett to discuss this (relatively non-onerous) job. Many thanks to Suzy for her years of service in this regard.

Key words
Education; MOPS (maintenance of professional standards; Hyperbaric medicine; General interest

Obituary



Long-time SPUMS member Rees Jones passed away on 06 September 2018 aged 83. Rees was a go-to expert for diving doctors throughout New Zealand on matters relating to his specialist field of pathology. He had a calm, collegial manner which made him unfailingly approachable to timid junior colleagues, including this Editor who learned a lot from him.

Rees was also a passionate diver, having started in the early 1950s and remaining active until recent times. Having a leg amputated did not deter him; he had an extra prosthetic leg made especially for diving which he wore beneath both dry and wetsuits. To go along with his medical expertise, Rees was also a knowledgeable marine naturalist, and a member of the Historical Diving Society. He dived widely throughout the New Zealand, Asia and Pacific regions. As he aged, he found other ways to remain engaged with the sport he loved. He and his late son Gareth operated the popular dive charter vessel *Pacific Hideaway* on many trips to the Poor Knights Islands and nearby sites. These included multiple successful technical diving expeditions to the famous bullion wreck *RMS Niagara*.

Rees was a major asset to the New Zealand diving medicine community, and one of diving's larger than life characters. He will be missed.

Simon Mitchell, with thanks to Keith Gordon

Key words
Obituary; General interest; Medical society

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: They must

1 be medically qualified, and remain a current financial member of the Society at least until they have completed all requirements of the Diploma;
2 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 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 submit a written proposal for research in a relevant area of underwater or hyperbaric medicine, in a standard format, for approval before commencing the research project;
5 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 (EO) 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 EO 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 and clinical research are acceptable. Case series reports may be acceptable if thoroughly documented, subject to quantitative analysis and if the subject is extensively researched 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, including case series, 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 proposal is approved prior to commencing research.

Projects will be deemed to have lapsed if:

- the project is inactive for a period of three years, or
- the candidate fails to renew SPUMS Membership in any year after their Diploma project is registered (but not completed).

For unforeseen delays where the project will exceed three years, candidates must explain to the EO by email why they wish their diploma project to remain active, and a three-year extension may be approved. If there are extenuating circumstances why a candidate is unable to maintain financial membership, then these must be advised by email to the EO for consideration by the SPUMS Executive. If a project has lapsed, and the candidate wishes to continue with their DipDHM, then they 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 January 2016, the SPUMS Academic Board consists of:

Dr David Wilkinson, Education Officer, Adelaide;
Professor Simon Mitchell, Auckand;
Dr Denise Blake, Townsville.

All enquiries and applications should be addressed to:
David Wilkinson
education@spums.org.au

Key words
Qualifications; Underwater medicine; Hyperbaric oxygen; Research; Medical society

Australian and New Zealand College of Anaesthetists Diving and Hyperbaric Medicine Special Interest Group

The new Diploma of Advanced Diving and Hyperbaric Medicine was launched on 31 July 2017. Those interested in training are directed to the ANZCA website http://www.anzca.edu.au/training/diving-and-hyperbaric-medicine.

Training

Documents to be found at this site are:

- Regulation 36, which provides for the conduct of training leading to the ANZCA Dip Adv DHM, and the continuing professional development requirements for diplomats and holders of the ANZCA Certificate of DHM;
- ANZCA Advanced DHM Curriculum which defines the required learning, teaching and assessment of the diploma training programme; and
- ANZCA Handbook for Advanced DHM Training which sets out in detail the requirements expected of trainees and accredited units for training.

Examination dates for 2019

Written paper	26 June 2019
Viva voce	31 July 2019

Accreditation

The ANZCA Handbook for Advanced DHM accreditation, which provides information for units seeking accreditation, is awaiting approval by Standards Australia and cannot yet be accessed online. Currently six units are accredited for DHM training and these can be found on the College website.

Transition to new qualification

Transitional arrangements for holders of the ANZCA Certificate in Diving and Hyperbaric Medicine and highly experienced practitioners of DHM seeking recognition of prior experience lapsed on 31 January 2019.

All enquiries should be submitted to dhm@anzca.edu.au.

Suzy Szekely, Chairperson, ANZCA DHM SIG
Suzy.Szekely@health.sa.gov.au

Royal Australian Navy Medical Officers' Underwater Medicine Course 2019

Dates: 14–25 October
Venue: HMAS Penguin, Sydney

The MOUM course seeks to provide medical practitioners with an understanding of the range of potential medical problems faced by divers. Emphasis is placed on the contra-indications to diving and on the diving medical assessment (including workshops covering key components), together with the pathophysiology, diagnosis and management of common diving-related illnesses. The course includes scenario-based simulations focusing on the management of diving emergencies.

Cost: AUD1,355 without accommodation
(tbc with accommodation and meals 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
E-mail: Rajeev.Karekar@defence.gov.au

Diving and Hyperbaric Medicine
is now on Facebook
Like us at:
https://www.facebook.com/divingandhyperbaricmedicine/

Undersea and Hyperbaric Medical Society Annual Scientific Meeting 2019

Dates: 27–29 June
Venue: Wyndham Grand Rio Mar, Puerto Rico

The UHMS offers an educational program at the meeting that is sure to have something for everyone and with three pre-courses on 26 June:
1. How to prepare for accreditation;
2. Hyperbaric oxygen safety – technical and procedural aspects;
3. Treatment of decompression illness in recreational diving.

Ideally located on the shores of the Atlantic Ocean, this 500-acre resort is the closest to El Yunque rain forest. In addition to its natural beauty, many activities for both adults and children are available.

https://www.uhms.org/meetings/annual-scientific-meeting/uhms-annual-scientific-meeting-information.html

Scott Haldane Foundation

As an institute dedicated to education in diving medicine, the Scott Haldane Foundation has organized more than 250 courses all over the world over the past 22 years, increasingly targeting an international audience with courses worldwide. The courses Medical Examiner of Diver (part I and II) and SHF in-depth courses, as modules of the level 2d 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 (ECB).

SHF Course Calendar 2019
18–25 May: Medical Examiner of Divers part 2, Caribbean
September: Dangerous Marine creatures (level 2d) NL
September: Refresher course 'Organization diving medical' NL
9–16 November: Medical Examiner of Divers part 1, Nosy be, Madagascar
16–23 November: 27th SHF In-depth course diving medicine (2d): Nosy be, Madagascar
23–30 November: 27th SHF In-depth course diving medicine (2d): Nosy be, Madagascar

On request: Internship different types of diving (DMP), NL
On request: Internship HBOT (DMP certification), NL/Belgium

The course calendar will be supplemented regularly.

For the latest information: www.scotthaldane.org

DIVING HISTORICAL SOCIETY AUSTRALIA, SE ASIA

P O Box 347, Dingley Village
Victoria, 3172, Australia
Email: hdsaustraliapacific@hotmail.com.au
Website: www.classicdiver.org

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Agenda 2019 of the international Capita Selecta Diving Medicine

Capita Selecta offers symposia on diving medicine presented by speakers of international renown to a multinational audience. The lectures are in English.

13–17 May: Marsa Alam, Egypt
Diving Medicine
Topics include fitness to dive, medication, remote management, diving with children, ageing, situational awareness, breath holding, fatalities, saturation diving.
Speakers include Adel Taher and others.

30 November: AMC Amsterdam
Physiology and medicine of the metabolic and inert gases in diving
Topics include O_2, CO_2, CO, He, N_2, Nitrox, He-mixtures, saturation diving, diagnosis, intoxications, high pressure nervous syndrome, treatment.
Speakers include J-C Le Péchon and Mattijn Buwalda

For programmes and registration:
www.capitaselectaduikgeneeskunde.nl.

Publications database of the German Diving and Hyperbaric Medical Society (GTÜM)

EUBS and SPUMS members are able to access the German Society's large database of publications in diving and hyperbaric medicine. EUBS members have had this access for many years. SPUMS members should log onto the SPUMS website, click on "*Resources*" then on "*GTÜM database*" in the pull-down menu. In the new window, click on the link provided and enter the user name and password listed on the page that appears in order to access the database.

Advertising in *Diving and Hyperbaric Medicine*

Companies and organisations within the diving, hyperbaric medicine and wound-care communities wishing to advertise their goods and services in *Diving and Hyperbaric Medicine* are welcome. The advertising policy of the parent societies appears on the journal website: www.dhmjournal.com

Details of advertising rates and formatting requirements are available on request from:
Email: editorialassist@dhmjournal.com

Diving and Hyperbaric Medicine: Instructions for Authors (summary)

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). It 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, members of the diving and hyperbaric industries, and divers. 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.

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Contributions should be submitted electronically by following the link:
http://www.manuscriptmanager.net/dhm
There is on-screen help on the platform to assist authors as they assemble their submission. In order to submit, the corresponding author needs to create an 'account' with a user name and password (keep a record of these for subsequent use). The process of uploading the files related to the submission is simple and well described in the on-screen help, provided the instructions are followed carefully. The submitting author must remain the same throughout the peer review process.

Types of articles

DHM welcomes contributions of the following types:

Original articles, Technical reports and Case series: up to 3,000 words is preferred, and no more than 30 references (excluded from word count). Longer articles will be considered. These articles should be subdivided into the following sections: an **Abstract** (subdivided into Introduction, Methods, Results and Conclusions) of no more than 250 words (excluded from word count), **Introduction, Methods, Results, Discussion, Conclusions, References, Acknowledgements**, **Funding** sources and any **Conflicts of interest**. **Legends / captions** for illustrations, figures and tables should be placed at the end of the text file.

Review Articles: up to 5,000 words is preferred and a maximum of 50 references (excluded from word count); include an informative **Abstract** of no more than 300 words (excluded from word count); structure of the article and abstract is at the author(s)' discretion.

Case reports, Short communications, Work in progress reports, etc: maximum 1,500 words, and 20 references (excluded from word count); include an informative **Abstract** (structure at author's discretion) of no more than 200 words (excluded from word count).

Educational and historical articles, Commentaries, Consensus and other meeting reports, etc., for occasional sections may vary in format and length, but should generally be a maximum of 2,000 words and 15 references (excluded from word count); include an informative **Abstract** of no more than 200 words (excluded from word count).

Letters to the Editor: maximum 600 words, plus one figure or table and five references.

Formatting of manuscripts

All submissions must comply with the requirements set out in the full instructions on the DHM website. Non-compliant manuscripts will be suspended whilst the authors correct their submission. Guidance on the general structure for the different types of articles is given above.

The following pdf files are available on the DHM website to assist authors in preparing their submission:

- Instructions for authors (full version)
- DHM Key words 2018
- DHM Mandatory Submission Form 2018
- Trial design analysis and presentation
- EASE participation and conflict of interest statement
- English as a second language
- Guideline to authorship in DHM 2015
- Helsinki Declaration revised 2013
- Is ethics approval needed?

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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 incidents. 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 e-mail: research@danasiapacific.org

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

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

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

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